The relationships of prolonged standing induced low back pain development with lumbopelvic posture and movement patterns

By

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Author's Declaration

I hereby declare that I am the sole author of this thesis. This is a true copy of the thesis, including any required final revisions, as accepted by my examiners.

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Abstract

Over 80% of individuals will suffer from low back pain at least once in their lifetime. The cause within the population is not homogenous, leading to sub-classifications of non-specific low back pain. One such sub-classification is low back pain in response to prolonged standing. Over 50% of people who have never suffered a low back injury will develop transient low back pain when completing a prolonged standing occupational simulation where there is no option to sit. Many service and manufacturing tasks require prolonged standing and the introduction of sit-stand desks into the office workplace means that even more workers will be standing on the job. Many workplace health and safety societies recommend the use of standing aids to prevent the negative effects of prolonged standing; however, very few of these standing aids have been validated. Currently, it is difficult to provide advice for people who perform these jobs and get low back pain, as we still do not know enough about the origins of this pain. As a result, the purpose of this thesis was to investigate the relationships of prolonged standing induced low back pain development with lumbopelvic postures and movement patterns. The four specific questions asked were (1) Are movement patterns different between pain and non-pain developers (2) Do pain and non-pain developers have different lumbopelvic postures? (3) How do different foot positions alter lumbopelvic posture?, and (4) Can a standing aid that alters posture or movement patterns prevent low back pain development in standing?

Study #1: Previous research points to the lack of movement during prolonged standing as a pre-disposing factor to low back pain. Such movements could be at the level of the lumbar spine or at the foot-ground interface. The primary purpose of this *in vivo* study was to determine if there were differences in magnitude, region, and frequency of movement patterns between pain and non-pain developers. Thirty-two participants reported their low back pain development using a visual analogue scale over 2-hrs of prolonged standing. Time-varying lumbar spine kinematics were used to assess the magnitude and frequency of lumbar spine fidgets and shifts. Ground reaction forces were used to assess the magnitude and frequency of whole body weight transfers and anterior-posterior center of pressure movements.

Fourteen of 32 participants (43.75%) were categorized as pain developers. The first 15 minutes of standing distinguished the two pain groups, as non-pain developers performed a higher frequency of lumbar spine flexion/extension fidgets and large body weight transfers. Both of these differences may be pre-disposing factors for transient low back pain development, as they both occurred prior to pain developers reaching the 10 mm visual analog scale threshold for low back pain classification.

Study #2: The purpose of this study was to investigate differences in lumbar posture between 17 participants categorized as a pain or non-pain developers during level ground standing. A secondary purpose was to evaluate the influence of two standing aids (an elevated surface to act as a foot rest and declined sloped surface) on lumbopelvic posture. Four sagittal plane radiographs were taken– a normal standing position on level ground, when using an elevated foot rest and declined sloped surface, and maximum lumbar spine extension to act as a reference posture. Lumbosacral lordosis, total lumbar lordosis, and individual intervertebral joint angles were measured on each radiograph. On level ground, pain developers stood closer to their maximum lumbosacral lordosis and L5/S1 intervertebral joint maximum extension angles. The elevated surface was most effective at causing lumbosacral lordosis flexion, while the declined surface was more effective at inducing L1/L2 intervertebral joint flexion. The differences between the posture and the influence of standing aids point to postural characteristics as a factor influencing pain development.

Study #3: While it is common to assess postural characteristics that may predispose a person to low back pain, these measures do not capture valuable information on the intrinsic properties of the lumbar spine, such as stiffness. The purpose of this study was to assess the relationship between the *in vivo* lumbar spine lumped passive stiffness and the location of the neutral zone with the self-selected lumbar spine angle of pain and non-pain developers in four standing postures. Twenty-two participants with known pain group status stood in four postures for 5 minutes each: on level ground, while resting a foot on an elevated surface, with their feet staggered, and on a sloped decline. Median lumbar spine angle was calculated for each position. Participants were then placed in a near-frictionless jig and brought through

passive lumbar spine extension and flexion to characterize their passive stiffness curve and location of their neutral zone. Overall, pain developers stood with a lumbar spine angle that was further beyond their passive lumbar spine neutral zone than non-pain developers. Not all aids thought to be successful at reducing low back pain worked in a similar manner, as only the elevated surface brought the lumbar spine into flexion and closer to the passive neutral zone. As a result, pain developers may be standing in a position that puts higher mechanical loads on the passive tissues of the lumbar spine. While flexion was induced by the elevated surface, the lack of changes caused by the sloped surface signal that there may be other postural changes other than just altered lumbar spine angle that are important for reducing low back pain.

Study #4: While alternating standing position on a declined and inclined surface has proven successful at reducing low back pain during standing, the purpose of this study was to evaluate standing solely on a declining sloped surface to isolate the influence of the postural change alone. Seventeen participants performed two 75-minute prolonged standing occupational simulations in a random order – one on level ground and one a declining surface. Fifty-three percent of participants (9/17) were categorized as pain developers during the level ground standing condition; however, the average maximum pain scores of pain developers were 58% lower during sloped standing. All participants showed hip joint flexion, trunk-to-thigh angle flexion, and posterior translation of the trunk center of gravity towards the ankle joint when standing on the sloped surface compared to level ground. These postural changes could cause the muscles crossing the posterior aspect of the hip joint to increase their passive stiffness and assist with stabilizing the pelvis. This study stresses the importance of hip kinematics, not just lumbar spine posture alone, in reducing low back pain during prolonged standing.

General Conclusions: The differences in posture of the lower lumbar arc between pain groups, the influence of standing aids on posture, and lower self-reported low back pain reports with a change in posture point to postural differences between the pain groups as being responsible for prolonged standing induced low back pain development. A working hypothesis for pain development is that when standing

on level ground, pain developers stand with their lower lumbar arc closer to its end range of extension, placing additional mechanical load on the posterior elements of the lumbar spine resulting in transient pain. When mild flexion is induced, the mechanical load on these tissues would be decreased – a potential reason that low back pain is decreased as well. Future work should focus on lower lumbar arc and hip posture differences between the pain groups and alternative interventions that do not require a physical standing aid to reduce pain development. These include further assessment of the staggered standing position and the influence of an exercise intervention on posture and movement patterns.

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Dedication

To all the graduate students who are currently dealing with anxiety, depression, or any other combination of brain illnesses – let this thesis be an example of what can happen when you reach out for help, practice self-compassion, resist avoiding what makes you uncomfortable, and take care of yourself. The road is challenging, but it's worth it!

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1 Introduction

An overwhelming number of individuals will suffer from low back pain at least once in their lifetime. The lifetime prevalence of low back pain is up to 84% (Airaksinen et al., 2006) and the Pan American Health Organization cited low back pain as one of three occupational problems that should be under surveillance (Choi et al., 2001). The cause of low back pain within a population is not homogeneous, as many tissues in the lumbar spine can be responsible for pain development and 93% of primary contact clinicians think that non-specific low back pain is a heterogeneous condition (Kent and Keating, 2004). As a result, there have been attempts to develop sub-classifications of non-specific low back pain. One such sub-clinical population is individuals who develop transient low back pain during prolonged standing tasks (Nelson-Wong and Callaghan, 2010c), with up to 71% of individuals reporting low back pain development during 2-hours of prolonged standing (Marshall et al., 2011). Many occupations, such as cashiers and bank tellers (Seifert et al., 1997), involve prolonged standing and the prevalence of workers whose usual work occurs in a standing posture is approximately 58%, with one in six of these workers reporting having the option to sit during their task (Tissot et al., 2005). The introduction of sit-stand and standing desks in the workplace means that more workers will be standing throughout the day. To combat the potential pain development linked with prolonged standing, standing aids designed in an attempt to reduce pain development are recommended; however, their impact has only been assessed in a limited manner and the mechanism underlying any pain mitigation is unknown. As a result, an investigation of the mechanisms of transient low back pain development and the influence of standing posture during prolonged standing is warranted to further our knowledge on the relationship between low back pain and prolonged standing from occupational, clinical, and basic science points of view.

1.1 Occupational Prolonged Standing and Development of Low Back Pain

1.1.1 Evidence of an association between prolonged standing and low back pain development

Epidemiological and laboratory studies have been used to assess the relationship between occupational prolonged standing and low back pain. Currently, epidemiological studies have demonstrated mixed results, while transient low back pain development has been demonstrated in laboratory studies.

Epidemiological studies looking at the association between prolonged standing and low back pain query a person's typical standing duration either at work or throughout the day using a questionnaire and ask participants about their low back pain reports or follow up with participants to determine if they have reported a low back injury/pain within the year. Many studies have demonstrated an association between prolonged standing and low back pain (Andersen et al., 2007; Kopec et al., 2004; Macfarlane et al., 1997; Mendelek et al., 2011; Roelen et al., 2008; Tissot et al., 2009) while some have not (Engels et al., 1996; Harkness et al., 2003). Despite the positive findings of some studies, two systematic reviews have concluded that there is a lack of epidemiological proof of a causal relationship between low back pain and occupational prolonged standing (Bakker et al., 2009; Roffey et al., 2010). Two disadvantages to the systematic review study design in these two papers were that the definitions of standing are variable (Tissot et al., 2009) and they treat low back pain as homogenous (McGill, 2011). When the conditions of standing work were factored into study design, Tissot and colleagues (2009) found that of workers who report standing as part of their occupation, those who stand in a constrained posture showed a low back pain prevalence of 30.4% versus 17.4% when standing with the freedom to sit. Also, Andersen and colleagues (2007) found that standing for greater than 30 minutes per hour resulted in a hazard ratio of 2.1.

Laboratory simulations have been used to assess the self-reported reports of low back pain during occupational prolonged standing tasks (Marshall et al., 2011; Nelson-Wong et al., 2008b; Nelson-Wong and Callaghan, 2010c; Raftry and Marshall, 2012). Most of these studies have been conducted on asymptomatic individuals who report never having a low back injury or pain that has required them to

visit a clinical and/or miss work. Despite this, between 41 and 71% of these participants self-reported clinically relevant changes in the magnitude of low back pain during prolonged standing tasks after 15 to 45 minutes of standing. While these laboratory studies have provided many distinguishing characteristics of pain developers and non-pain developers, they have yet to conclude what the mechanisms and pathways for pain development are in these individuals. As a result, it is necessary to build off the knowledge of previous prolonged standing research to investigate these possible mechanisms.

1.1.2   Increased use of sit-stand and standing workstations

A sedentary lifestyle has been associated with increased risk of all-cause and CVD mortality (Dunstan et al., 2010), metabolic syndromes and their components (Gardiner et al., 2011), cardio-metabolic and inflammatory biomarkers (Healy et al., 2011), and in women, non-Hodgkin Lymphoid Neoplasms (Teras et al., 2012). In 2011, these studies became the sources for many news stories on a sedentary lifestyle and associated negative health outcomes. With headlines such as "Is your office chair killing you?" (McGinn, 2011) and "Prolonged bouts of sitting increase cancer risk" (Beck, 2011), the concept of a typical workstation, which involves a high volume of seated work, was under fire and has brought about a movement towards sit-stand or standing desks. For example, the use of standing biased workstations have demonstrated positive results in a classroom setting, where a significant increase in calorie expenditure was found in students who were in the classroom with stand biased desks, as compared to the seated control group (Benden et al., 2011).

A discussion point is whether less sitting automatically means more standing (Messing et al., 2013). First, along with the relationship between low back pain and prolonged standing, there are also negative health outcomes (Waters and Dick, 2014) such as cardiovascular (such increased carotid atherosclerosis (Krause et al., 2000) and varicose veins (Tuchsen et al., 2005)) and pregnancy issues (increased incidence of still births, spontaneous abortions (McDonald et al., 1988) , and pre-term deliveries(Mozurkewich et al., 2000). Second, despite changing a workstation that requires predominately sitting to standing, the worker may still remain in a stationary position for an extended period of time.

Since people can develop low back pain in standing, and a previous injury is not required for this to occur, many individuals may find that they begin to develop low back pain while standing at their desk. Until we understand the mechanisms of pain development in prolonged standing, it will be difficult to provide guidelines for the sitting, standing, and walking/alternate activities proportions during a typical workday (Messing et al., 2013). If standing is also seen as good and sitting as bad, some people who develop low back pain during standing may not want to sit down for fear of being interpreted as lazy, ignoring their health, and rely on other coping strategies to minimize their pain and discomfort (Messing et al., 2005).

1.1.3   Asymptomatic population

The most interesting phenomenon related to prolonged standing and low back pain is that it affects individuals who, to their own knowledge, have never suffered a back injury or pain that required a visit to a clinician or lost time at work/school. Out of a battery of clinical physical and psychological tests, pain development in standing was only predicted by an Active Hip Abduction Test (Nelson-Wong et al., 2009a).

The transient nature of this pain development can provide insights into possible pain mechanisms that predispose certain individuals to low back pain development during standing. Individuals become classified as pain developers between the 15-45 minute mark of a 2-hour prolonged standing trial (Marshall et al., 2011; Nelson-Wong and Callaghan, 2010c) and pain quickly dissipates once the standing has ceased. Transient pain development is due to mechanical stimulus of free nerve endings located in the lumbar tissues (Cavanaugh, 1995), while the persistence of pain after the mechanical stimulus is removed is due to inflammation (Cavanaugh, 1995) or chemical insult (Winkelstein and DeLeo, 2004) of the surrounding tissues causing nerves to fire at lower thresholds than they would typically respond. Lumbar spine passive tissue strain and interaction of the posterior elements of adjacent vertebrae may occur in standing when an individual stands beyond the limits of their lumbar spine passive neutral zone (Scannell and McGill, 2003), which can lead to firing of the nociceptive sensory units in the lumbar tissues,

providing a potential mechanism for low back pain development in certain individuals during prolonged standing.

If there is no evident back injury, people may disregard their low back pain or fatigue as simply part of their job and not important because it goes away once the workday is done (Messing et al., 2005). But what are the implications of 8 hours a day, 5 day a week of this pain development on future risks of chronic low back pain? Using a longitudinal study to follow prolonged standing pain and non-pain-developers over 3 years, Nelson-Wong and Callaghan (2014) found pain developers three times more likely to experience chronic low back pain over the first 24 months, seek medical care within three years of their participation, and report multiple episodes of chronic low back pain. While low back pain during standing is transient in nature, it appears to be a positive predictor for experiencing future chronic and recurrent low back pain (Nelson-Wong and Callaghan, 2014). The cumulative mechanical insult and strain to the tissues in the low back region may decrease their tolerance over time resulting in an injury or structural changes leading to chronic pain. As a result, it is important to stress that although people reporting acute pain development during standing have never suffered a low back injury, they could be characterized as a pre-clinical group for the development of chronic low back pain in the future.

1.2   Influence of altered muscle activation patterns of pain developers

While many characteristics that distinguish pain and non-pain developers have been examined, muscle activation patterns of the hip and trunk have been the most consistent differences between the pain groups. Non-pain developers typically present a reciprocal firing pattern of their right and left gluteus medius muscles (Nelson-Wong et al., 2008b), while pain developers exhibit co-activation of these muscles (Marshall et al., 2011; Nelson-Wong et al., 2008b) and higher levels of co-contraction (Nelson-Wong and Callaghan, 2010c). The higher levels of gluteus medius co-activation might be because pain developers may have poor motor control and insufficient stabilization of their trunk muscles (Nelson-Wong et al., 2008b). An exercise program that targeted stabilization of the hip and trunk muscles resulted in lower subject reports of low back pain in those previously categorized as pain developers during

standing, as well as less gluteus medius co-contraction in male pain developers (Nelson-Wong and Callaghan, 2010a).

Altered hip and trunk stabilization of pain developers might influence how pain developers control their movement while standing. In a typical standing posture (feet inline and parallel to each other), movement typically occurs in the frontal plane and the medial-lateral center of pressure is predominantly controlled by a load/unload strategy driven proximally by the hip abductors/adductors (Winter et al., 1993). Increasing activity of the right hip musculature will unload the left limb and the weight is automatically transferred to the right limb, and vice versa (Winter et al., 1993). This has been demonstrated in both quiet and prolonged standing (Nelson-Wong et al., 2008a; Winter et al., 1993). Due to altered hip muscle activation patterns, differences in the frontal plane movement patterns may exist between the pain groups. Differences in the amplitude, duration, and frequency of lumbar spine movements may also be exist between the pain groups due to the altered trunk muscle co-activation.

1.3   Use of standing aids in the workplace

The National Institute of Occupational Health and Safety, Canadian Center for Occupational Health and Safety, and the Occupational Safety and Health Administration recommend the use of standing aids as a low cost solution that alternates a worker's standing posture in an attempt to reduce low back pain development during prolonged standing tasks (Konz and Rys, 2003). Commonly recommended standing aids that alter foot position are a bar or platform to elevate one foot (Rys and Konz, 1989; Whistance et al., 1995) or a sloped surface (Nelson-Wong and Callaghan, 2010b). There are also recommendations for altered foot positions that do not require the use of standing aids that alternate foot position in standing in contrast to the typical feet inline and parallel (Zacharkow, 1988).

Despite recommendations, there is no accompanying guidance or standards for the use and design of standing aids. For example, there is no information on the height of the platform or bar rail and whether or not it is necessary to adjust it based on an individual's anthropometrics. With the introduction

of sit-stand and standing only desks into the workplace, there will likely be an increase in the use of standing aids. It would be advisable to be able to form a guideline for manufactures on how to design these aids and usage guidelines for health and safety practitioners so that they are effectively integrated into the workplace.

There are two benefits to studying the use of standing aids during prolonged standing tasks. First, we can establish the most effective standing aids for decreasing or eliminating transient low back pain during standing. Second, by examining how successful standing aids alter movement and lumbopelvic postures, hypotheses on the mechanisms and sources of transient low back pain development during level ground standing can be established and investigated. These changes in posture and foot position can be assessed during level ground standing and compared between pain and non-pain developers, or compared between level ground standing and when using a standing aid.

1.4 Implications of altered lumbar spine and pelvis posture on transient low back pain development

Individuals who develop transient low back pain during prolonged standing may have an altered spinal alignment compared to those who do not develop transient low back pain. For example, patients who stood in a state of segmental hyperextension were likely to have spinal stenosis (Roussouly et al., 2005). Since an exercise program that targeted trunk and hip musculature altered muscle activations of pain developers (Nelson-Wong and Callaghan, 2010a), lumbar and pelvic postures were also likely affected. This is supported by literature that suggests lumbar lordosis angle can be modified from hyperextension to a flexed posture similar to the sample's average posture using a specific exercise program (Scannell and McGill, 2003).

Individuals characterized as having a hyperlordotic lumbar spine have been shown to stand in a posture that was outside of their lumbar spine passive neutral zone (Scannell and McGill, 2003). Hyperextended postures can lead to contact between the spinous processes or bottoming out of the facets

that can result in impingement of soft tissue (Adams et al., 1988; Yang and King, 1984), increased compressive stress on the posterior annulus (Adams and Hutton, 1980), increased contact between the facet faces (Dunlop et al., 1984), reduction in the size of the intervertebral foramen (Fujiwara et al., 2001; Inufusa et al., 1996; Panjabi et al., 1983) and stretching of the facet joint capsule (Yang and King, 1984). With increased extension and compression, the amount of the compressive load carried by the facets is increased, which results in bony interaction and pivoting of the facet and pars interarticularis, and stretching of the highly innervated joint capsule (Yang and King, 1984). The facet joint capsule is highly innervated by group III and IV mechanosensitive units compared to surrounding tissue, which might serve a nociceptive function, suggesting that the facet joint capsule may be a source of low back pain (Yamashita et al., 1990).

While hyperextension may result in pain development, there may be benefits to mild flexion of the lumbar spine during prolonged standing in those who develop transient low back pain. The benefits of mild flexion are suggested by the impact of standing aids on lumbar spine and pelvis posture using a sloped surface (Nelson-Wong and Callaghan, 2010b) and elevating one leg onto a platform (Dolan et al., 1988; Whistance et al., 1995). With each of these standing aids, flexion of the lumbar spine is induced by changing lower limb posture. A collection of studies have shown that flexion of the lumbar spine results in reduced stress of the facet joints, reduced compressive stress on the posterior annulus, improved transport of disc metabolites, and increased compressive strength of the spine in mild flexion compared to typical upright or extended postures (Adams and Hutton, 1985).

1.5 Implications of altered movement patterns on transient low back pain development

The higher trunk and hip muscle co-contraction seen in pain developers (Marshall et al., 2011; Nelson-Wong et al., 2008b; Nelson-Wong and Callaghan, 2010c) could affect how a person moves while standing. There are three things to consider when discussing movement - location, size, and frequency of motions. A common movement while standing is shifting body weight between the right and left leg.

Lumbar spine movement has been assessed in both sitting and standing with pain developers utilizing less of their lumbar spine range of motion despite similar postures (Gallagher et al., 2014). When breaking down the types of movement during sitting, it has been hypothesized that large movements are a good indicator of discomfort while small quick movements can alleviate it (Vergara and Page, 2002).

The lack of movement by pain developers means that they may not have any relief from the potential pain mechanisms or mechanical loads seen in the posterior lordotic spine (Section 1.4). If two people have similar postures, but one person moves their lumbar spine more frequently than the second, the second person may be more susceptible to prolonged loading of the same tissues. The first person could potentially share the loading between many other tissues. Even voluntary movements distal from the lumbar spine, such as cyclically altering of lower limb position, can induce movement in the pelvis and lumbar spine due to the kinematic chain, preventing prolonged static loading of the same tissues.

1.6   General Themes/Research Questions

The global objective of this thesis was to investigate the relationships of prolonged standing induced low back pain development with lumbopelvic posture and movement patterns. The specific research questions addressed by this thesis are outlined below and in Figure 1.1. The specific hypotheses underlying each question and study are presented in their respective chapters.

Are movement patterns different between pain and non-pain developers during prolonged standing?

A typical standing posture is with the feet inline and parallel, and dictates a person's movement. Study #1 assessed the frequency and magnitude of body weight transfers, center of pressure, and lumbar spine movements. This was the first study to assess differences in the movement patterns of pain and non-pain developers during prolonged standing.

Do pain and non-pain developers have different lumbar spine and pelvic flexion/extension postures?

This question was answered across two studies. Using a radiographic examination, Study #2 assessed differences in lumbar spine and pelvis sagittal alignment during level ground standing between pain and non-pain developers. In this study, the lumbopelvic posture when standing on level ground was compared between pain and non-pain developers. Study #3 assessed where the lumbar spine functions with respect to the passive neutral zone as defined by passive flexion and extension trials using a frictionless jig. The lumbar spine posture when standing on level ground was compared to the passive neutral zone for pain and non-pain developers.. These two studies contributed new knowledge on how postural differences between the pain groups might result in transient low back pain development and provided a foundation for developing a hypothesis of the mechanisms for the development or absence of pain during prolonged standing.

What is the potential for different foot positions and standing aids to alter lumbar and pelvic flexion/extension posture?

Standing aids that alter foot position are recommended for occupations that require prolonged standing; however, little is known about why these standing aids are successful or how they impact the lumbar spine. The influence of standing aids on the posture and where the lumbar spine functions with respect to the lumbar passive stiffness curve defined *in vivo* using a frictionless jig were assessed in Studies 2 and 3, respectively. Assessing altered foot positions in this manner linked different postures with the potential to cause or reduce pain during standing and any potential negative effects to non-pain developers. Combined with question two, hypotheses on the pathways for pain development were expanded.

Can a standing aid that alters posture or movement patterns prevent low back pain development in standing?

The last study assessed the impact of standing aids in those who typically develop low back pain during level ground standing and tested the hypotheses of low back pain development established regarding

posture and movement from the first three studies and from previous literature. Participants performed

two sessions of prolonged standing – one on level ground and one using a standing aid. The results of this

study provided evidence for occupational implementation and tested hypotheses for pain development

established in studies 1-3.
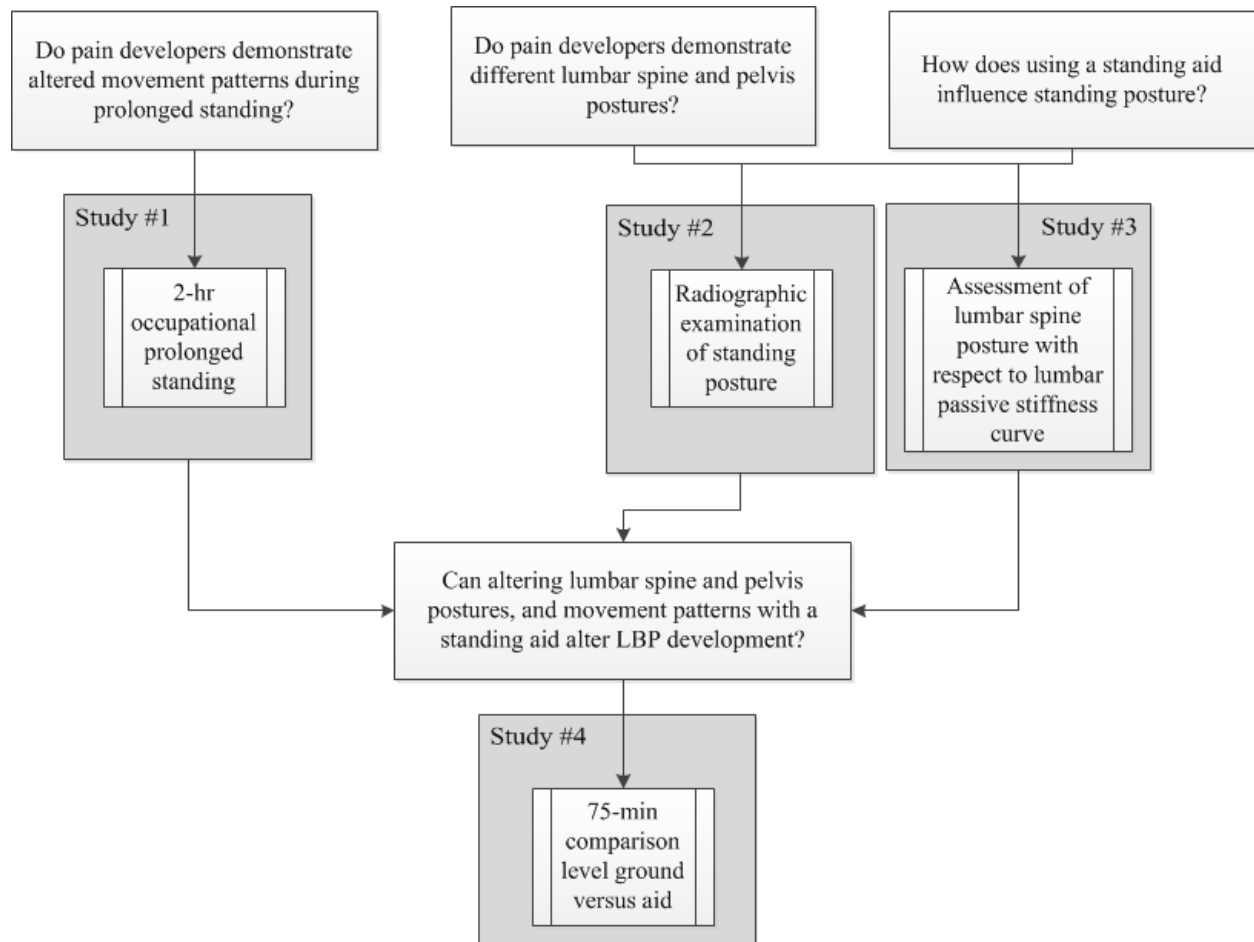
Figure 1.1 Schematic of the research questions and studies for this thesis. Note: LBP = low back pain.

2 Literature Review

2.1 Occupational Standing and Low Back Pain

For over 15 years, researchers have examined the link between occupational activities, such as standing, and low back pain through epidemiology and laboratory studies. Both of these designs provide complimentary, yet sometimes conflicting, evidence on the nature and relationship of low back pain development during prolonged standing.

2.1.1 Epidemiological Evidence and Systematic Reviews

Epidemiology studies that assessed the association between low back pain and occupational prolonged standing have provided mixed results. Many studies have established an association between prolonged standing and low back pain (Andersen et al., 2007; Kopec et al., 2004; Macfarlane et al., 1997; Mendelek et al., 2011; Roelen et al., 2008; Tissot et al., 2009). MacFarlane and colleagues (1997) found that females and males were 2.9 and 1.6 times more likely, respectively, to seek consultation for low back pain over a 12 month period when their baseline job descriptions involved standing and/or walking for longer than two hours per shift. Andersen and colleagues (2007) found that in a 2-year prospective study, standing for greater than 30 minutes per hour resulted in a hazard ratio of 2.1 (95% CI 1.3-3.3). Tissot and colleagues (2009) found that of workers who report standing as part of their occupation, those who stand in a constrained posture showed a low back pain prevalence of 30.4% versus 17.4% when standing with the freedom to sit.

Conversely, some studies do not demonstrate this relationship. Harkness and colleagues (2003) investigated newly employed workers from various occupational settings over a 2 year period and found that standing of any kind was not a predictor of new-onset low back pain. In nurses working in nursing homes in The Netherlands, Engel and Colleagues (1996) found that being hampered by standing did not demonstrate a significant association with low back pain in a multivariate logistic regression model.

A major issue with the epidemiology studies lies in their assessment and definitions of low back pain and prolonged standing (Table 2.1). First, each study assessed a person's low back pain and reports of occupational standing at different stages of the study and none directly assessed low back pain and duration of standing. Also, no study asked people directly if they develop low back pain when they stand. Second, questions emphasize events of low back pain that are of longer durations, assuming that prolonged standing will only cause lengthy, chronic, or more serious injuries. Due to the phrasing of these questions, low back pain that occurs during standing but is relieved shortly after the person stops standing may remain unreported or buried within the results of the study. Finally, the inconsistent results could be due to the lack of consistent definitions of standing since there may be variations in how a worker can move around and their freedom to change postures or sit down (Tissot et al., 2009).

While the results of these field studies have been mixed, recent systematic reviews conclude that there is a lack of epidemiological proof of a causal relationship between low back pain and occupational prolonged standing (Bakker et al., 2009; Roffey et al., 2010); however, there are limitations associated with these studies. First, basic science and experimental laboratory studies are excluded from the analysis (Kuijer et al., 2011; Takala, 2010) in order to assess actual occupational physical activities and actual low back pain rather than associated biomechanical or anatomical parameters that are related to the cause of pain (Dagenais et al., 2010), thus excluding laboratory studies that demonstrate short-term associations between mechanical exposures and outcome related variables to low back pain (Takala, 2010). These reviews also do not take into account the heterogeneous causes of low back pain, as they do not consider potential mechanisms and their relationship with different injuries or attempt to separate different low back injuries (McGill, 2011). Lastly, they do not parse out the potential influence of the quality of standing, such as the freedom to move around versus a constrained task, on low back pain development (Tissot et al., 2009).

Table 2.1 List of epidemiology studies that assessed the association between low back pain and prolonged standing.

Study	Low Back Pain Question	Assessment of Standing
Kopec and colleagues (2004)	"Have you been diagnosed by a health professional with back problems, excluding arthritis?" Note: Instructed to report long-term problems that lasted/expected to **last 6 months or longer**.	Asked to categorize working habits i.e. usually sit during the day and don't walk about very much; stand or walk about quite a lot during the day but don't have to carry or lift things often.
Harkness and colleagues (2003)	Asked participants, "Thinking back over the past month, have you had any ache or pain which lasted one day or longer?" If participants localized their pain between the 12th rib and gluteal folds lasting for **24 hours** or longer in the past month, they were categorized based on this into those with and without low back pain	Asked about their postures in their last work day (i.e. standing) and categorized by "Do not stand as part of job", < 15 min, 15 min - <2 hrs, >= 2 hrs.
Andersen and colleagues (2007)	"How much have you been bothered by pain during the past 12 months?"	Standing was categorized as > 30 minutes per hour or not
Mendelek and colleagues (2011)	Assess low back pain cause (accident at work, work excess, disease, other) and duration (intermittent or felt sometimes, acute lasting less than 3 months, sub-acute lasting between 3 to 6 months, chronic lasting more than 6 months)	Asked about standing hours/per day with categories (< 4 hours, 4-8 hours, > 8 hours)
Tissot and colleagues (2009)	"In the past twelve months if you had any significant pain in any of the following body sites which interfered with your activities?" low back pain that was significant was reported when it interfered with their usual activities "fairly often" or "all the time".	Standing was assessed based on (a) do you usually work in a standing posture and (b) if the participant usually stood at work they were asked "which best describes your posture most of the time: 1) standing in a fixed position with no possibility of moving around, 2) standing in a relatively fixed position, with possibility of making one or two steps 3) standing and moving around a little bit (one desk to another) 4) standing and moving around a little bit more (one office building to another) 5) standing with the possibility of sitting down whenever you want).
Roelen and colleagues (2008)	Do you have regular pain, or do you feel stiff at the small of your back? Are these symptoms possibly during or made worse by your work? Do these symptoms trouble you while working?	Determined according to worker's perception in last month, and asked "Are you troubled during work with prolonged standing (yes/no)?
MacFarlane and colleagues (1997)	Any ache or pain lasting longer than **24 hours**, in the area bordered at the top by the 12th rib and at the bottom by the gluteal fold. Information obtained on the presence of current (within past month) and previous low back pain.	Asked to report for current and previous jobs if they stand or walk for more than two hours total.
Engel and colleagues (1996)	Do you suffer regularly from back complaints? If yes, do you experience these complaints in (a) the lower back or (b) the upper back?	Are you hampered or have difficulty with adopted postures, such as standing?

2.1.2 Laboratory Assessments of Occupational Prolonged Standing and Transient Low Back Pain Development

Laboratory studies that categorize transient low back pain developers during prolonged standing using a 100 mm visual analogue scale score repeatedly during the standing task. Participants are classified as pain developers if they demonstrate a 10 mm increase in their self-reported rating of low back pain relative to the level reported at the start of the standing task.

Four published studies have assessed transient low back pain development during prolonged standing using the above protocol and have demonstrated distinct trends in pain reporting between pain and non-pain developers. The first study found that 65% of the participants were categorized as pain developers, and pain development occurred after about 15 to 30 minutes of standing (Nelson-Wong et al., 2008b). Nelson-Wong and Callaghan (2010c) found that 40% of participants were categorized as pain developers, with pain groups beginning to separate after 30 minutes. Marshall and colleagues (2011) reported that 71% of participants developed low back pain, with separation also occurring after 30 minutes. In the most recent study, Raftry and Marshall (2012) found that 50% of their participants developed low back pain during standing, with separation of the two groups after 15 minutes.

Currently, laboratory studies have provided the only knowledge regarding the development of transient low back pain during prolonged standing, which one could argue is a necessary and expected precursor to chronic pain and injury. Although these studies have not assessed low back pain development with workers directly in the field, they provided evidence that this phenomenon exists and allow researchers to investigate the mechanism behind transient low back pain development. By testing different hypotheses in the lab, we can conduct better informed field studies testing these hypotheses in the workplace.

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2.2 Indicators of transient low back pain development during prolonged standing

2.2.1 Clinical Predictors

Out of a battery of clinical tests used during a routine Physical Therapy examination, only an Active Hip Abduction (AHAbd) test, where a person performed active side-lying hip abduction (Nelson-Wong et al., 2009a), was shown to distinguish pain and non-pain developers (Nelson-Wong and Callaghan, 2010c). This test was designed to assess an individual's ability to maintain their lumbar spine and pelvic alignment while performing a task in an unstable position (Nelson-Wong et al., 2009a). Pain developers had more difficulty maintaining a neutral position of their pelvis and trunk, which may relate to the poor trunk control demonstrated by pain developers during level standing (Nelson-Wong et al., 2009a).

2.2.2 Anthropometrics, Demographics, Gender, and Psychosocial Assessments

Participant anthropometrics and demographics, such as height, weight, age, and BMI, have consistently been shown to be similar between pain and non-pain developers (Marshall et al., 2011; Nelson-Wong et al., 2008b; Nelson-Wong and Callaghan, 2010c; Raftry and Marshall, 2012). The percentage of males and females who develop low back pain during prolonged standing varies between studies. Raftry and Marshall (2012) found that for both males and females the percentage of pain developers was 50%. Nelson-Wong and Callaghan (2010c) found that 48% of females developed pain, while only 32% of males developed pain. Marshall and Colleagues (2011) found that 75% of males and 69% of females developed transient low back pain.

Questionnaires designed to provide a psychosocial assessment of attitudes toward injury, pain, and fear of movement did not demonstrate differences between groups or genders; and thus likely did not influence pain development (Nelson-Wong and Callaghan, 2010c).

2.2.3 Muscle Activation Patterns

Muscle activation patterns, rather than the magnitude of muscle activity (which does not exceed 3%MVC for trunk and hip muscles, independent of pain group (Nelson-Wong, 2009)), have been found to

distinguish pain and non-pain developers. Based on gluteus medius cross-correlation values, 74% of pain developers were categorized into the correct pain group (Nelson-Wong et al., 2008b). Gluteus medius co-activation was found in pain developers, while non-pain developers demonstrated a reciprocal firing of these muscles (Nelson-Wong et al., 2008b). In two follow up studies, gluteus medius co-contraction indexes (Nelson-Wong and Callaghan, 2010c) and cross-correlation values (Marshall et al., 2011) were higher in pain compared to non-pain developers. Pain developers also initially had higher levels of trunk flexor-extensor co-activation than non-pain developers; however, they did not use this strategy when their pain reports began to rise, whereas non-pain developers used this strategy during that same time period (Nelson-Wong and Callaghan, 2010c).

This co-activation response is not an adaptation to pain development, but is hypothesized to be a potential pre-disposing factor or symptom of pain development since it was evident at the start of the standing task (Nelson-Wong and Callaghan, 2010c). Errors to the neural control system (like from faulty or absent proprioceptive information) may cause undesirable amplitude and timing of muscle contractions (Panjabi, 1992a). The underlying cause of higher gluteus medius co-activation in pain developers might be poor motor control or the inability to use their trunk muscles for stabilization (Nelson-Wong et al., 2008b). The right gluteus medius was activated before the trunk muscles, demonstrating a bottom up strategy for control, signaling that hip muscle co-activation may compensate for the inability to properly control the trunk in these individuals (Nelson-Wong and Callaghan, 2010c).

2.2.4   Muscular Strength and Endurance

Although the magnitude of muscle contractions is low while standing, sustained low level contractions may be responsible for muscle fatigue. Pain developers have a shorter total gap length during two hours of prolonged standing for bilateral gluteus maximus and medius, which may be related to the elevated co-contraction in pain developers (Nelson-Wong and Callaghan, 2010c). Although this hypothesis for pain development is plausible over prolonged periods of time, pain development begins quickly after standing

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begins and separation occurs at around 15 to 45 minutes, which may be too early for this mechanism to be responsible pain.

To test the influence of muscle fatigue and strength on pain development, differences in strength and endurance between pain and non-pain developers, gender, and as a function of time have been compared using a variety of tests. In a trunk extensor endurance test, only a main effect of gender was found, with females holding the position longer than males (Nelson-Wong, 2009). Side-bridge endurance was lower for pain developers prior to prolonged standing; however, hip abduction strength between the two groups was not different (Marshall et al., 2011). Side-bridge endurance and hip abduction strength prior to prolonged standing were also associated with gluteus medius co-activation – the lower the strength or endurance, the greater the co-activation (Marshall et al., 2011).

2.2.5 Asymmetrical Standing Postures

Standing with a majority of body weight on one leg results in higher muscle activity on the load bearing side and a reduction of muscle activity of the unloaded leg, which in theory should provide periodic unloading and relaxation of the musculature of one limb at a time (Carlsöö, 1961). Since pain developers demonstrated bilateral gluteus medius co-activation, it was hypothesized previously that they would spend less time in an asymmetrical posture than non-pain developers who demonstrate reciprocal firing (Marshall et al., 2011; Nelson-Wong et al., 2008b) and a lower level of co-contraction (Nelson-Wong and Callaghan, 2010c). Participants spent less than 50% of the 2-hour standing protocol in an asymmetrical posture, which was defined for this study as within +/-15% of 50% BW (Gallagher et al., 2011). Female pain and non-pain developers demonstrated similar levels of asymmetry, whereas male pain developers spent under 10% of the first 30 minutes of standing in an asymmetric posture then stood at similar levels to non-pain developing males (between 20 and 30%) for the remaining 90 minutes (Gallagher et al., 2011).

In line with the theory that people move with higher frequency over time as way to combat discomfort and pain development, people demonstrate an increased frequency of their asymmetrical

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postures of shorter duration (Gallagher et al., 2011) over time, independent of pain group. The greatest change in frequency and duration of these postures occurred during the first 30 minutes of standing, which coincides with when the pain groups generally begin to separate (Gallagher et al., 2011). As a result, the authors concluded that bilateral co-contraction of the gluteus medius muscles did not prevent asymmetrical postures, but that the two groups may recruit different muscular strategies to perform the same task (Gallagher et al., 2011).

2.2.6 Low Back Loading

Of the kinetic variables assessed previously, only L4/L5 joint shear decreased over time during prolonged standing (Gregory and Callaghan, 2008). During a single leg stance, Nelson-Wong and Callaghan (2010) found that there was a general decrease in vertebral joint rotational stiffness at the L2/3 and L4/5 levels after two hours of prolonged standing during a 10 second single leg stance trial supported by the right leg, while no differences were noted for the left side or between the pain groups. As a result of this decreased lateral bend stiffness; the authors concluded that it might not be safe for individuals to perform tasks requiring side loading and balance (Nelson-Wong et al., 2010).

2.2.7 Physiological Changes

Near infrared spectroscopy (NIRS) (Callaghan et al., 2010), heart rate, and skin temperature (Gregory and Callaghan, 2008) have been assessed during prolonged standing, with only skin temperature over the right thoracic erector spinae showing a significant change that decreased over time (Gregory and Callaghan, 2008). Despite evidence of co-contraction in pain developers, the development of low back discomfort during prolonged standing is not linked with sustained muscle contractions that prevent the delivery of oxygen to postural muscles responsible for maintaining upright stance (Callaghan et al., 2010).

2.3 Other Characteristics of Prolonged Standing

2.3.1 Center of pressure

There is a proposed relationship between fatigue and postural changes of center of pressure – a high frequency of movements may provide momentary relief of pressure on the foot pad to restore blood flow and decrease fatigue (Duarte et al., 2000); however, whether the increased frequency of postural changes transfers to low back pain relief is unknown.

Gregory and Callaghan (2008) found that anterior-posterior center of pressure shifts in the first 15 minutes of standing was the only significant variable related to the center of pressure used in a regression model to predict low back discomfort during prolonged standing. It was possible for a person to move around too much in the first 15 minutes of standing, which might cause discomfort even if it was being done to prevent or reduce low back discomfort development over time (Gregory and Callaghan, 2008). This study only assessed shifts that exceeded +/- 2 standard deviations of the average center of pressure location, and thus, different types of movement patterns were not examined.

In an assessment of unconstrained standing, Duarte and Zatsiorsky (1999) characterized three types of center or pressure changes – shifts, fidgets, and drifts. Fidgets, defined as quick high amplitude movements of the center of pressure, are the most frequent postural change during prolonged standing, followed by shifts, then drifts (Duarte and Zatsiorsky, 1999). As mentioned above, it is expected that with pain and discomfort, more movement would occur; however, this may not be the case. Over 30 minutes of prolonged standing, patients with chronic low back pain demonstrated a decrease in the frequency of shifts and drift patterns in the AP direction exhibiting a more rigid standing strategy (Lafond et al., 2009).

The relationship between bilateral gluteus medius muscle activations and the medial-lateral center of pressure during prolonged standing has been assessed using a cross-correlation analysis (Nelson-Wong et al., 2008a; Nelson-Wong et al., 2009b). In an efficient postural control strategy, the medial-lateral center of pressure should move towards the left when the left gluteus medius muscle is activated, and vice

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versa for the right (Winter et al., 1993). Males and females demonstrated the load-unload mechanism through the hip abductors for control of the medial-lateral center of pressure (Nelson-Wong et al., 2008a). The strength of this correlation was higher in males than females, with males demonstrating a more dominant medial lateral postural control through the hip abductors compared to females (Nelson-Wong et al., 2008a).

The influence of altered co-activation patterns of the hip musculature in pain developers on the successful performance of the load/unload mechanism and medial-lateral center of pressure patterns is unknown. Since pain developers may lack the ability to properly control their medial lateral center of pressure via the hip load/unload mechanism because of elevated co-contraction, their response to prolonged standing may be similar to that of the elderly, who have smaller amplitudes of medial lateral shifts, which may be the result of lack of mobility in this population and reflects their inability to use the load/unload mechanism of postural control (Freitas et al., 2005).

2.3.2   Lumbar spine movement

There is limited literature assessing the movement of the lumbar spine during prolonged standing. In quiet standing, people vary their lumbar spine postures by approximately 10% of their range of motion (Callaghan and McGill, 2001). Over time, lumbar spine flexion increases and an increase in axial twist angle during the first 15 minutes was one of three variables linked with the prediction of low back discomfort development in standing (Gregory and Callaghan, 2008).

Gregory and Callaghan (2008) assessed lumbar spine movements defined as angle changes that are greater than +/- 2 standard deviations from the mean posture and Callaghan and McGill (2001) used an APDF to assess variation in posture; however, these methods do not parse out different types of movements, such as shifts versus fidgets. This has been addressed in a variety of ways in the sitting literature. Movements have previously been divided into macro- and micromovements (Vergara and Page, 2000; Vergara and Page, 2002). Macro-movements are global changes in posture, while micro-

movements are small movements that occur around a global posture that is adopted for several minutes (Vergara and Page, 2000). Another method is an adaptation of Duarte and Zatsiorsky's (1999) method for assessing shifts and fidgets of the center of pressure (Dunk and Callaghan, 2010).

In sitting, it is hypothesized that static postures provoke more pain, while small quick movements of the lumbar spine can alleviate it and help to reduce muscular strain (Vergara and Page, 2002). On the other hand, macromovments are a good indicator of discomfort in sitting – bigger and quicker movements may be made as a response to lumbar pain and discomfort (Vergara and Page, 2002). Dunk and Callaghan (2010) found that people who did not develop low back pain in sitting used a more static sitting strategy and smaller portion of their lumbar spine range of motion. People who developed low back pain demonstrated a wider range of postures over a larger range of motion (61.4 vs. 27.3% ROM), greater frequency of shifts over the last 45 minutes, and fidgets and shifts amplitudes 1.5-2.5 times larger than those found in control participants; therefore, those who developed low back pain may have moved around more frequently and at greater amplitudes in an attempt to decrease their discomfort even though this method was not successful (Dunk and Callaghan, 2010).

Amplitude and frequency of lumbar spine movements may also be related to the quality of proprioceptive information from the muscles surrounding the lumbar spine. In sitting, proprioceptive information from multifidus is crucial for successful repositioning of the pelvis in a seated lumbosacral task (Brumagne et al., 1999). The inability to use a trunk flexor-extensor co-activation strategy during the greatest increase in pain development (Nelson-Wong and Callaghan, 2010c) may also mean that pain developers will have greater lumbar spine movement throughout this time.

No attempt has been made to quantify the amplitude and frequencies of the lumbar spine movements separated into shifts and fidgets during prolonged standing and assess differences in lumbar spine movement patterns between pain and non-pain developers.

2.3.3   Weight Transfers

While frequency and duration of asymmetrical postures have been assessed previously (Gallagher et al., 2011), the amplitude of weight transfers have not been quantified. Elderly participants produce significantly fewer large amplitude weight transfers compared to young, healthy individuals, but demonstrated the same number of small magnitude weight transfers (Prado et al., 2011). Elderly participants might not be able to efficiently use the medial-lateral load/unload strategy during prolonged standing (Prado et al., 2011). With the influence of co-contraction in pain developers, it would be hypothesized that they would have a greater number of small versus large weight transfers compared to non-pain developers.

2.4   Characterizing standing posture

2.4.1   Sagittal alignment of the lumbar spine during prolonged standing

2.4.1.1 Is there a typical standing posture?

The goal of a standing intervention is to provide people with the proper postural alignment for completing a task with minimal discomfort; however, the definition of this posture is poorly defined (Claus et al., 2009) because there is large variation in sagittal alignment between individuals. Even in a standardized radiograph position, an assessment of 160 volunteers with no spinal deformities demonstrated a global lumbar lordosis angle mean of 61.4 degrees, with a range of 41.2 to 81.9 degrees (Roussouly et al., 2005). Sacral slope also varied from 20 to 65 degrees in standing (Roussouly et al., 2005). De Carvalho and colleagues (2010) found that lumbar lordosis angle demonstrated a mean of 63 degrees and standard deviation of 15 degrees and sacral inclination showed a mean of 43 degrees with a standard deviation of 10 degrees.

While lumbar lordosis and pelvis angles are highly variable, most of this variability occurs in the lower lumbar spine. The upper arc of the lumbar spine remains constant at around 20 degrees, while the lower arc (lumbosacral lordosis angle) is the most important component in determining lumbar lordosis

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angle (Roussouly et al., 2005). This was echoed by Mitchell and colleagues (2008), who found that a greater portion of the total lumbar range of motion was in the lower lumbar spine (58%) versus the upper lumbar spine (42%).

Studies have developed classifications systems for sagittal alignment of the lumbar spine and pelvis to deal with the large variability. Scannell and McGill (2003) assessed females in three sagittal posture categories - hypolordosis (greater than -8 degrees), hyperlordosis (less than -24 degrees), or the mean (17-19 degrees) assessed in the study (Scannell and McGill, 2003). Classification systems based on combined criteria of the lumbar spine and sacral postures have also been used (Roussouly et al., 2005). In this study, a majority of the people fell into the posture that the authors defined as the "balanced spine" (Roussouly et al., 2005).

2.4.1.2 Influence of the kinematic chain

The association between pelvic alignment and lumbar lordosis is an important determinant in sagittal posture (Roussouly et al., 2005) and the link between rotation of the pelvis and lumbar lordosis has been well established (Day et al., 1984; Levine and Whittle, 1996; Roussouly et al., 2005). Tilting the pelvis posteriorly results in a flattening of the lumbar curvature, while anterior tilting results in the opposite (Day et al., 1984; Levine and Whittle, 1996). Maximum pelvis tilt does not elicit maximum lumbar flexion and extension; however, alterations in trunk or lower limb posture can alter lumbar spine angles even more (Levine and Whittle, 1996). For example, without voluntary pelvic tilt, standing with one leg on a platform results in reduced lumbar curvature (Dolan et al., 1988; Whistance et al., 1995) and plantar or dorsiflexion of the foot causes changes in pelvis, trunk and lumbar lordosis posture (Nelson-Wong and Callaghan, 2010b).

2.4.2   Passive Stiffness of the lumbar spine during standing

Since morphological differences in the lumbar spine may dictate sagittal posture, assessment of an individual's lumbar spine posture during standing with respect to the lumbar spine passive neutral zone as

defined in vivo provides an alternate look at the influence of standing posture on the possibility for low back pain development.

2.4.2.1 Concept of the neutral zone

According to Panjabi (1992b), intervertebral range of motion can be split into two parts – the neutral zone and elastic zone. The neutral zone is the portion of the physiological range of motion in which spinal motion is produced with minimal resistance; it is a zone of high flexibility and laxity (Panjabi, 1992b). The elastic zone is measured from the end of the neutral zone up to the physiological limit; within this zone spinal motion is produced against significant internal resistance due to stiffening of ligaments, intervertebral disc, or bony contact and has a high stiffness (Panjabi, 1992b). The non-linear properties of ligaments result in a high amount of laxity around the neutral zone and a stiffening towards the end range of motion (Panjabi, 1992b).

The presence of a neutral zone has been defined *in vitro* (Goertzen et al., 2004; Oxland and Panjabi, 1992; Thompson et al., 2003) in the absence of muscle tone. *In vivo,* the neutral zone has been defined as the range where the lumbar spine would demonstrate the least amount of passive tissue stiffness (Scannell and McGill, 2003). Flexion (Beach et al., 2005; De Carvalho and Callaghan, 2011; McGill et al., 1994; Parkinson et al., 2004; Scannell and McGill, 2003), extension (McGill et al., 1994; Scannell and McGill, 2003), lateral bend (Gombatto et al., 2008; McGill et al., 1994), and axial twist (Drake and Callaghan, 2008; McGill et al., 1994) passive stiffness curves have been previously quantified *in vivo.*

By characterizing the neutral zone of the lumbar spine *in vivo,* the location of a person's typical lumbar spine standing posture with respect to their passive stiffness curve and the likelihood of passive tissue strain can be assessed. It is important for people to function within their lumbar spine neutral zone during activities of daily living, such as standing, in order to minimize and avoid this passive tissue strain (Scannell and McGill, 2003).

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2.4.3 The relationship between posture and low back pain

Many studies have attempted to establish a relationship between low back pain and lumbar lordosis and pelvic alignment by comparing those with low back pain to healthy controls. People categorized with significant low back pain demonstrated a smaller maximum extension angle than mild or no pain groups (Mitchell et al., 2008). The duration of pain (i.e. acute versus chronic) can also impact posture, such that people with chronic low back pain may adapt to their pain by changing their posture to balance their upper spine and maintain their upright stance, while people with acute pain may alter their posture in hopes to reduce the pain immediately (Christie et al., 1995).

Sagittal spinal alignment also affects regional trunk muscle activity. There is lower trunk extensor and internal oblique muscle activity in sway standing (where the pelvis translates anteriorly relative to the trunk), while muscle activity was higher during upright standing postures (O'Sullivan et al., 2002). A short lordosis angle elicits the highest muscle activity in a majority of the trunk muscles compared to a flat, long, or slumped lumbar lordosis posture (Claus et al., 2009). Claus and colleagues (2009) stated that if the objective of an efficient posture is balance of the spine with minimal stress on the articular and ligamentous system, then a flat lumbar and thoracolumbar posture should be recommended because it is midway between lordotic and slumped postures, and demonstrates the lowest muscle activity compared to a long or short lumbar lordotic posture in sitting.

Patients with spinal stenosis stand in a state of hyperextension, while those who have a "well balanced" spine reported minimal complaints (Roussouly et al., 2005). In a similar posture, the sway standing position is achieved through extension of the lower lumbar segments with very little motion of the upper lumbar segments (Mitchell et al., 2008). As mentioned above, these passive postures result in the inhibition of lumbopelvic stabilizing muscles (O'Sullivan et al., 2002), which can result in pain development due to higher strain on the passive tissues in the lower lumbar spine (Mitchell et al., 2008).

Aberrant postures alone may not be enough to define whether or not a person could develop low back pain. A person who demonstrates "poor" posture may do so as a consequence of their individual anatomy and as a result, the posture that they choose to stand in elicits the least amount of elastic strain (Scannell and McGill, 2003). As a complimentary measure to sagittal lumbopelvic alignment, passive lumbar spine stiffness provides insight into the potential for passive lumbar spine tissue strain. People categorized with a hyperlordotic lumbar spine stand beyond the extension limit of their neutral zone (Scannell and McGill, 2003) and might place unnecessary strain on the passive structures of their lumbar spine. Scannell and McGill (2003) had participants enter an exercise rehabilitation program designed to bring individuals who were hyper- or hypolordotic into postures closer to the average lumbar spine angles seen in their study population. The exercise training brought the lumbar spine posture of the previously hyperlordotic individuals to within the limits of the neutral zone during standing (Scannell and McGill, 2003).

2.5   Sources of pain development during prolonged standing

2.5.1   Transient vs. Persistent Pain

Evidence suggests that separate mechanisms cause transient versus persistent pain. Transient pain development is due to mechanical stimuli, while persistent pain is the result of inflammation.

A mechanical insult is required for pain to occur (Winkelstein and DeLeo, 2004) and lumbar pain development may be due to mechanical stimulus of the nociceptors located within the facet capsules, superficial annulus and dorsal root ganglion (Cavanaugh, 1995). Low levels of mechanical compression result in transient pain that disappears quickly, while higher levels of loading may initiate mechanisms that will eventually result in sustained or chronic pain (Hubbard et al., 2008). Stimulating the dorsal root ganglion can result in discharges after the stimulus has been ceased and this has been demonstrated to occur for ~25 minutes after the stimulus has been removed, whereas with stimulation of peripheral nerves, the pain will usually cease immediately after stimulus removal (Cavanaugh, 1995).

Since nerves usually stop firing once a stimulus is removed, mechanical insult alone does not explain the persistence of pain (Cavanaugh et al., 1997). The presence of inflammation or a chemical insult decreases the threshold that a nerve fires and as a result the nerve will fire with mechanical stimuli lower than its usual threshold (Cavanaugh, 1995), which can result in the ongoing background discharge of sensory nerves (Cavanaugh et al., 1997). In rats, a chemical insult on its own does not demonstrate any changes, which shows that a mechanical stimulus is still required for pain to occur (Winkelstein and DeLeo, 2004). The introduction of a chemical insult resulted in a lower mechanical strain to cause the pain response compared to the higher strains required when no chemical insult was used (Winkelstein and DeLeo, 2004).

2.5.2   Lumbar extension and possible sources of pain development

2.5.2.1 *Facet Joints, Facet Joint Capsule, and Posterior Elements*

Facet load is increased with extension or compressive loading and causes an interaction of the top of the inferior facet with the pars of the vertebrae below through a thin layer of soft tissue, resulting in load transmission from this "bottoming out" (Yang and King, 1984) and a greater chance of soft tissue impingement (Adams and Hutton, 1980). In two degrees of extension, the facet joints resist approximately 16% of the compressive load after creep and the facet joints of the three lowest lumbar levels carry increased loads compared to the facet joints of the upper lumbar joints (Adams and Hutton, 1980). Six degrees of extension and 4 mm of disc height loss produced the greatest amount of contact between the facet faces (Dunlop et al., 1984). With this amount of disc height loss, soft tissue can be caught between the pedicle and facets or within the facet faces, which can result in pain development (Dunlop et al., 1984). Excessive facet loads can cause the inferior facet to pivot about the pars interarticularis (Yang and King, 1984) and in full extension the inter-facet compressive forces can cause the inferior articular process to bend backward and upward by 2.24 +/- 2.48 degrees (Green et al., 1994)

Increased loading of the facets stretches the facet joint capsule (Yang and King, 1984), and with sustained loading, this could be a mechanism of pain development (Adams and Hutton, 1980). The facet

capsule is highly innervated with group III and IV mechanosensitive units, with more than any of the surrounding tissues and may serve a nociceptive function (Yamashita et al., 1990).

Finally, the posterior elements are the first tissues damaged in hyperextension (Adams et al., 1988). When the posterior elements are in contact they can carry compressive load and soft tissue, such as the interspinous ligament, can become trapped between the adjacent spinous processes resulting in pain development (Adams et al., 1988).

2.5.2.2 *Intervertebral Disc*

Intradiscal pressure can be relieved in extension; however, extension causes more compressive stress on the posterior annulus (Adams and Hutton, 1980). A greater percentage of fluid loss occurs with intact lumbar lordosis compared to a fully flexed spine, which can cause disc height loss leading to a reduction in the size of the intervertebral foramen and increased load bearing of the facets if the facet tips come into contact (Adams and Hutton, 1983; Adams and Hutton, 1986). Lumbar intervertebral discs are highly supplied by nociceptive nerve endings on the outer annulus (Bogduk et al., 1981); therefore, increased stress on the annulus may result in a pathway for pain development (Adams et al., 1996).

2.5.2.3 *Intervertebral Foramen and Spinal Canal*

The nerve roots exit the spinal canal through the intervertebral foramen. Two joints and other ligaments form the borders of the intervertebral foramen; therefore, the foramen dynamically changes its configurations with movement of the lumbar spine (Gilchrist et al., 2002). With extension, the intervertebral foramen becomes smaller, whereas it is larger size in flexion (Fujiwara et al., 2001; Harrison et al., 1999; Inufusa et al., 1996; Panjabi et al., 1983).

It is debated whether changes in intervertebral foramen size could result in pain development. Nerve roots typically occupy 30% of the available intervertebral foramen area, but as high as 50% has been reported (Gilchrist et al., 2002). Although changes in spinal canal and the intervertebral foramen can occur with changes in sagittal posture, these changes may be too small to cause pain development and

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slight extension is preferred to reduce mechanical stress and strains on the tissues of the CNS (Harrison et al., 1999). Panjabi and colleagues (1983) state that there is still adequate space remaining for the nerve root to maintain its integrity in extension; however, influence of ligamentum flavum or intervertebral disc bulging on the size of the intervertebral foramen was not assessed in this study.

Mechanical compression of nerve root results in nociceptive responses (Winkelstein et al., 2002) and compromised blood flow (Gilchrist et al., 2002; Harrison et al., 1999). In rats, higher levels of mechanical compression result in a higher pain response (Winkelstein et al., 2002). Repetitive flexion and extension in the presence of compression result in mechanical compression of a surrogate nerve root that was 44% (bur ranged as high as 70%) of the pressure threshold to elicit pain behavior as shown in rats, and 30% (but as high as 48%) of the pressure required to elicit the sustained pain response seven days after exposure to a 15-minute nerve root compression in rats (Drake and Callaghan, 2009).
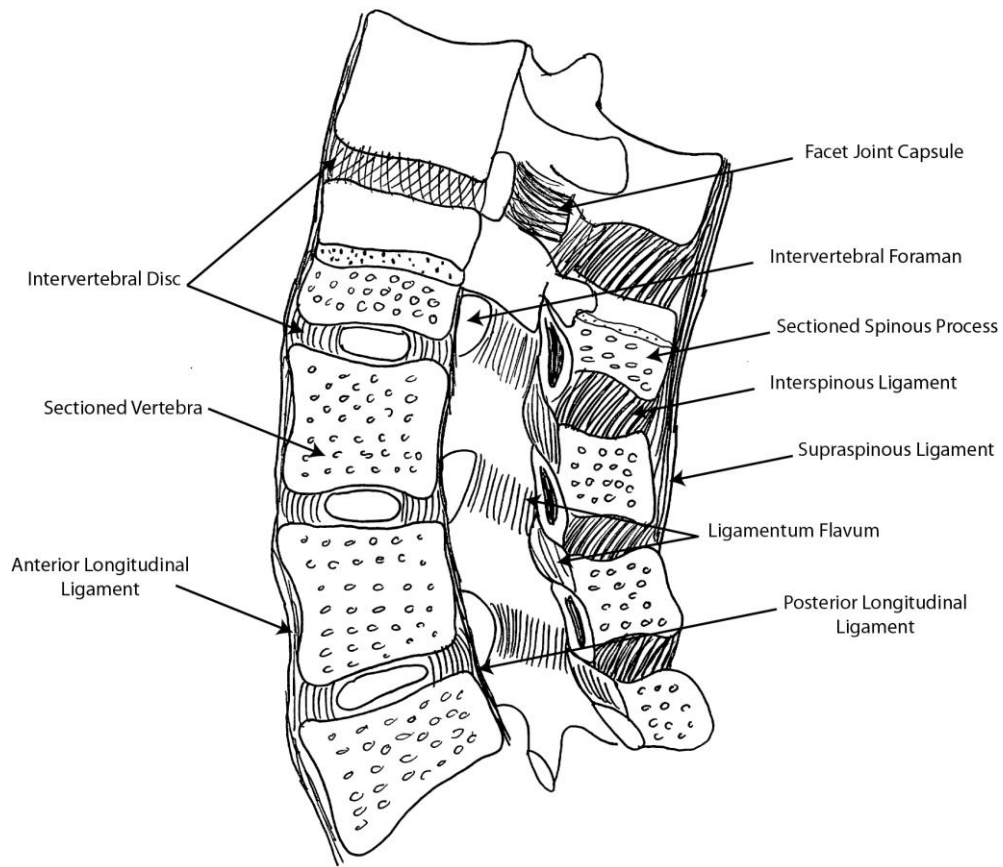
Figure 2.1 A partially sectioned sagittal view of the lumbar spine. Adapted from Adams et al. (2002).

2.5.3 Potential benefits and disadvantages of mild lumbar spine flexion while standing

In symptom free, normal population, it is mechanically and nutritionally advantageous to mildly flatten the lumbar spine while sitting and lifting heavy weights (Adams and Hutton, 1985). While no mention of its influence in standing, there are possible benefits to mild lumbar flexion in standing.

In mild flexion, the spinous processes separate, and the facet joints play less of a role in resisting compression (Adams and Hutton, 1980). The average peak pressure between the facet joints is lowest in flexion,(Dunlop et al., 1984); therefore, extra-articular impingement of the articular cartilage or soft tissue between the faces of the facets is less likely to occur. The highest compressive stress is transmitted through the anterior annulus and lowest through the posterior annulus in flexion (Adams and Hutton, 1985). The transport of disc metabolites and fluid exchange is improved via the thinning of the posterior annulus (Adams and Hutton, 1983). Flexion also results in an increase in size of the intervertebral foramen (Fujiwara et al., 2001; Inufusa et al., 1996; Panjabi et al., 1983), which decreases the likelihood for impingement of the nerves, blood vessels, and other soft tissues that are found within the foramen.

The disadvantages of flexed postures are that they can increase the compressive stress on the anterior annulus; however, this is the thickest and stiffness part of the annulus (Galante, 1967). Flexed postures can also result in increased hydrostatic pressure in the nucleus at low levels of loading; however, the compressive load in standing is only around 1000 N. Lastly, flexed postures are likely to involve higher extensor activity to maintain the posture. Dolan and colleagues (1988) compared lumbar spine angle and extensor muscle activity during a range of commonly adopted standing postures and found that that the adoption of these postures results in less lumbar lordosis; however, an increase in extensor muscle activity is noted, suggesting that people will adopt these postures despite the increased muscle activity.

2.6 Strategies to prevent transient low back pain during prolonged standing

2.6.1 Exercise Intervention

An exercise intervention designed to promote stabilization of the trunk and hip muscles has been proposed to prevent transient low back pain development during prolonged standing (Nelson-Wong and Callaghan, 2010a). To assess the influence of the exercise intervention on transient low back pain development a longitudinal study (Nelson-Wong and Callaghan, 2010a) was initiated when participants performed a two-hour occupational standing task and were grouped as either pain or non-pain developers. Within each pain group, participants were either assigned to the exercise intervention or control group and came back after four weeks to repeat the prolonged standing task. Pain developers assigned to the exercise group reported less low back pain visual analogue scale scores after the exercise intervention and were categorized as non-pain developers, whereas pain developers who did not partake in the intervention demonstrated no change in their visual analogue scale scores. For male pain developers enrolled in the exercise program, gluteus medius co-contraction index was less and total gap length was larger (Nelson-Wong and Callaghan, 2010a). It was concluded that an exercise program that emphasized trunk and hip stabilization had a positive impact on pain development reports, motor control and muscle activation patterns (Nelson-Wong and Callaghan, 2010a).

Previous work has also concentrated on the use of an exercise intervention to promote a more balanced sagittal posture in individuals who demonstrated hypo- or hyper-lordotic spines, with the goal of bringing these individuals more towards the mean lumbar lordosis angle found in the study population (Scannell and McGill, 2003). This exercise intervention had a positive effect on individuals with hyperlordosis – by the end of the intervention, these individuals stood with their lumbar spine postures similar to the mean of the study population and within their lumbar spine neutral zone (Scannell and McGill, 2003).

2.6.2 Sloped Platforms

Sloped standing platforms allow for variation in standing posture by placing a worker's feet in dorsi- or plantar flexion. Nelson-Wong and Callaghan (2010b) had both pain and non-pain developers stand on a sloped surface (16-degree slope) for two hours. Participants could stand in plantar or dorsiflexion and were allowed to alternate freely between the two positions. Using a sloped surface resulted in pain development scores that were 59.4% lower in those previously categorized as pain developers during level ground prolonged standing and their self-reported low back pain scores were now close to those reported by non-pain developers (Nelson-Wong and Callaghan, 2010b). During the prolonged trial, participants spent 72% of their time standing in the decline versus 28% in the incline position (Nelson-Wong and Callaghan, 2010b). After the study, 14 of the 16 participants said that if they worked in an occupational task that required prolonged standing, they would utilize the sloped surface (Nelson-Wong and Callaghan, 2010b).

Standing on a decline resulted in greater trunk flexion, posterior rotation of the pelvis (Nelson-Wong and Callaghan, 2010b), and flattening of the lumbar spine (Gallagher et al., Submitted). Standing on an incline surface resulted in the opposite, with greater trunk extension, anterior rotation of the pelvis (Nelson-Wong and Callaghan, 2010b), and extension of the lumbar curve (Gallagher et al. Submitted) compared to level ground standing.

Gluteus medius co-contraction was lower in pain developers while standing on a sloped surface versus level ground, while co-contraction is lower for non-pain developers, such that the two pain groups now had similar co-contraction levels during the sloped standing trial (Nelson-Wong and Callaghan, 2010b).

L5-S1 AP shear and L5-S1 moment were greater in the sloped conditions, and extension moment was significantly greater in the decline compared to the incline position (1.42 vs. 0.71 of the level

standing moment), but neither was different from level ground. Overall, the differences only amounted to 3-5% body weight and were likely not biologically significant (Nelson-Wong and Callaghan, 2010b).

The downside to using this standing aid is that it can be bulky, a potential tripping hazard, and would need to be long if the workstation is large and requires worker movement. Since participants were allowed to move freely between the two positions, it is unknown if the ability to vary posture over the two hours promoted lower pain development compared to the altered posture or muscle activation profiles.

2.6.3   Altered Foot Position

In the early to mid-1900s, many scientists recommended that people should shift their body weight in the anterior posterior direction, versus the typical medial-lateral weight transfer noted in bilateral stance (Zacharkow, 1988). In 1913, Mosher (as reiterated in (Zacharkow, 1988)) advocated the importance of learning a different way to transfer weight from between legs while standing. The instructions by Mosher (1913 & 1914) were for a person to place one foot a short step in front of the other and sway their entire trunk gently forward until their body weight rested on the ball of the forward foot (Zacharkow, 1988). To alternate the foot that bore the majority of the body weight, he or she would move the other foot forward to take the body weight. In the mid-1900s, Lee and Wagner (1949, as reiterated in (Zacharkow, 1988)) recommended that a person should "never stand with the weight on one foot unless it is a one-foot-forward position with the weight on the forward foot".

When a person stands with their feet in a 45-degree stance, with the feet parallel and one foot placed in front of the other, both the ankle and hip strategies contribute to the net center of pressure in the AP and ML directions (Winter et al., 1996). In the AP direction, the load/unload hip strategy accounts for 60% of the net AP center of pressure movement, while the ankle strategy accounts for 40% (Winter et al., 1996). Since pain developers exhibit higher gluteus medius co-contraction, this standing strategy might provide relief to the hip mechanism while standing. Weight transfers that are not solely controlled by a

hip strategy may now be successful due to the combined contribution of the hip and ankle musculature in performing these transfers.

Foot position also affects the center of pressure travel distance and its mean location in the AP or ML directions. For example, when a person places one foot 10 cm in front of the other, there is a slight increase in the total distance travelled by the center of pressure and this is significantly greater when the feet are placed apart by 30 cm (Kirby et al., 1987). Also, the ML and AP center of pressure moved towards the foot placed posterior, likely because the majority of an individual's weight was supported by the posterior leg (Kirby et al., 1987); however, this may be altered with instructions given to the participant on how to distribute body weight between the front and back foot.

2.6.4   Platforms/Bar Rails

The installation of a railing or platform for a person to elevate one foot on while standing is a commonly recommended to prolonged standing workers. Brass bar rails are a mainstay in most bars because "a happy customer will stick around" (Carson, 1995). NOISH, CCHOS, and OSHA all recommend the use of a standing rail or a footstool when a worker must stand for a prolonged period; however, there is little guidance provided to the worker on height requirements (standard or adjusted based on a person's height) or how to properly utilize the standing aid (switch between feet often versus one leg elevated constantly). Some literature recommends that workers use foot rails of a standard height, about 10 to 12.5 centimeters (Carson, 1995), but up to 20 centimeters (Canadian Center of Occupational Health and Safety (CCHOS), 2008).

Elevating one foot while standing is hypothesized to work by altering the proximal kinematic chain. Clinicians have suggested that raising one foot onto a footrest results in a standing posture that is stable, leaves the hands free, and prevents excessive spinal curvature (Whistance et al., 1995). Dolan and colleagues (1998) assessed extensor muscle activity and lumbar lordosis angle in this raised leg standing posture, as they believed that common postural habits were employed to reduce muscle activity and alter

the lumbar curvature. When people stood for brief periods with one leg elevated on a 20 cm platform, a 3-6 degree reduction in lumbar lordosis (flattening) was demonstrated; however, extensor muscle activity at the L1 and L5 levels was actually shown to increase in this posture (Dolan et al., 1988). Others claim that this posture relaxes the large trunk muscles on the ipsilateral side of the raised leg, leading to increased blood flow, which provides nutrients and removes waste products (Konz and Rys, 2003); however, this has never been studied.

One of the first studies to assess this standing aid in an occupational setting had people stand for 4 hours with their foot on a 10 cm platform for one minute every seven minutes (Rys and Konz, 1989). They found that comfort was generally greater in the platform condition; however, the greatest changes were seen for the neck and rear foot (Rys and Konz, 1989). An extension of this study assessed three raised foot standing conditions as compared to level ground – a 10 cm platform supporting one foot, a 10 cm platform angled at 15 degrees, and a 10 cm high 0.5 cm diameter bar rail - during two hours of prolonged standing (as described in Rys and Konz, 1994). Participants were instructed to use the standing aid as needed. The three standing aids were preferred over level ground standing, and the two platform conditions outperformed the rail condition; however, they were not used for an equal amount of time. The bar rail was used for 50% of the two hours, while the inclined platform was used for 75% and flat platform for 83% and participants switched their foot position on average every 90 seconds.

While the relationship between foot position and postural adaptations during standing has been established, the evidence of increased perceived comfort is not universal, despite their recommendation for use in the workplace (Whistance et al., 1995). This study found greater trunk flexion, more posterior tilt of the pelvis, less knee flexion in the supporting leg, and more plantar flexion in the supporting foot when using the rail and found no comfort changes between the footrest and other conditions; however, there was an increased discomfort in the thigh and hamstring of the supporting leg. The authors believe that increased trunk flexion was the reason that self-reported comfort ratings of the low back remained the same in the different conditions and may result in increased loading on the low back and call for frequent

changing of postures and not standing in the same position for a long period of time (Whistance et al.,

1995).

3   Study #1: Early movement of the lumbar spine and large body weight transfers are associated with prolonged standing induced low back pain

3.1   Introduction

In light of the recent research on the negative health impacts of a sedentary lifestyle (Dunstan et al., 2010; Gardiner et al., 2011; Healy et al., 2011; Teras et al., 2012), performing tasks while standing has become a targeted workplace intervention. Despite this, research has shown that standing also related to cardiovascular and musculoskeletal disorders (Waters and Dick, 2014). In both field (Andersen et al., 2007; Roelen et al., 2008; Tissot et al., 2009) and lab studies (Gallagher et al., 2014; Marshall et al., 2011; Nelson-Wong and Callaghan, 2010c), low back pain has been found in individuals required to perform prolonged static standing tasks. In a sample of individuals who have never had a back injury, at least 40% of participants report low back pain within 30-60 minutes of standing (Gallagher et al., 2014; Marshall et al., 2011; Nelson-Wong and Callaghan, 2010c). This pain is transient in nature as it goes away once the task is terminated. A worker who endures such pain daily could also put themselves at risk for future low back problems, as transient prolonged standing induced low back pain has been shown to be a predictor of future chronic low back pain leading to healthcare involvement (Nelson-Wong and Callaghan, 2014). A common recommendation is to incorporate movement to break up a prolonged sedentary task (Latouche et al., 2013); however, the definition of movement that could help people with transient prolonged standing induced low back pain is unclear since the underlying pain mechanism is unknown. Given the lack of research on movement patterns and low back pain while standing, this study assessed differences in movement patterns between low back pain and non-pain developers during prolonged standing.

Previous research points to a lack of movement during standing as a potential predisposing factor for low back pain development. Field studies have shown that standing for greater than 30 minutes predicts low back pain status (Andersen et al., 2007) and there is a higher prevalence of low back pain reports in those who stand in constrained areas with no freedom to sit compared to those who can stand

with freedom to sit (Tissot et al., 2009). Higher bilateral gluteus medius co-activation exists in pain developers during prolonged standing (Marshall et al., 2011; Nelson-Wong et al., 2008b; Nelson-Wong and Callaghan, 2010c) whereas non-pain developers demonstrated reciprocal firing of the right and left gluteus medius muscles (Nelson-Wong et al., 2008b). This co-activation occurs prior to pain development and has been proposed to cause PDs to stand in more static postures (Nelson-Wong and Callaghan, 2010c). We originally hypothesized that co-activation may prevent pain developers from transferring their weight between their legs; however, weight transfers from one leg to another become more frequent the longer a person stands, independent of pain group (Gallagher et al., 2011). Non-pain developers also utilize a greater range of their lumbar spine motion during both standing and sitting when working at a computer compared to pain developers, and the large shift from standing to sitting reduced the self-reported low back pain reports of pain developers (Gallagher et al., 2014).

In contrast to the lack of movement hypothesis, Gregory and Callaghan (2008) found that when monitoring the first 15 minutes of standing, a higher number of center of pressure shifts and the time muscles spent at rest were associated with higher low back discomfort ratings. They hypothesized that a person could (1) move around too much and causing pain, or (2) they were aware that they would develop low back pain when standing and tried to move around as a pre-emptive strategy (Gregory and Callaghan, 2008). If the lack of movement during prolonged standing is a pre-disposing factor to pain development, more information is required to provide advice to workers regarding the size, body region involved, and frequency of movements that they should perform.

During prolonged standing, we can assess body weight transfers and center of pressure movement at the feet – ground interface, or movement at the lumbar spine where the pain develops. In standing, loading and unloading of the lower limbs is a common movement pattern that should allow for relaxation of the muscles in the unloaded limb (Carlsöö, 1961). One study has assessed body weight transfers from one leg to another in pain developers (Gallagher et al., 2011); however, it did not separate out different

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magnitudes of movement between pain developers and non-pain developers. In order to quantify the types of movement occurring in standing, Duarte and Zatsiorsky (1999) defined three types of center of pressure patterns evident in unconstrained standing (1) fast and large displacement of center of pressure that returns to approximately the same location (fidget), (2) a fast displacement of the center of pressure from one location to another (shift), and (3) a slow continuous displacement (drift). In standing, fidgeting of the center of pressure is the most common pattern, followed by shifting (2 to 3 times less frequent) and drifting (6 times less frequent) (Duarte et al., 2000). Assessing weight transfer and center of pressure movements is important because the kinematic of the lower limb alters pelvis and lumbar spine posture (Dolan et al., 1988). Lumbar spine movements can be broken into different patterns based on the proposed categories proposed by Duarte and Zatsiorsky (1999). During sitting, large shifts in lumbar spine posture are a good indicator of low back discomfort and were larger and faster in the presence of pain, while small movements around a global posture maintained for several minutes are hypothesized to alleviate or prevent pain development by reducing muscular strain (Vergara and Page, 2002). Dunk and Callaghan (2010) have also applied the algorithm by Zatsiorsky and Duarte (1999) to lumbar spine angles during prolonged sitting. They found that those who had low back pain shifted more frequently and the amplitudes of the movements were 1.5 to 2.5 times greater in amplitude than those of controls participants. Only one study has assessed lumbar spine movement in standing; however, they did not separate out the size of the movement patterns (Gregory and Callaghan, 2008). As a result, this was the first study to take a comprehensive assessment of weight transfers, center of pressure movements, and the lumbar spine movements to assess the influence of size, body region, and frequency of movements on prolonged standing induced low back pain development.

The primary purpose of this study was to determine if there were differences in the magnitude, region, and frequency of movement patterns between prolonged standing pain developers and non-pain developers. It was hypothesized that non-pain developers would have a higher frequency and magnitude of movement patterns of their lumbar spine, especially the fidgets, and large weight transfers, yet similar

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frequency and size of weight small weight transfers and center of pressure movements compared to pain developers. Lastly, we hypothesized that there would be no influence of gender on any of the variables. To evaluate this, participants reported their low back pain development every 7.5 minutes during a 2-hour prolonged occupational standing simulation. Data from motion capture and two force platforms were continuously collected to provide time-varying kinematics of the lumbar spine and ground reaction forces, respectively. The motion capture data were used to assess the size and frequency of flexion-extension, lateral bend, and axial twist movements of the lumbar spine. The ground reaction forces and center of pressure data were used to assess the size and frequency of whole body weight transfers and anterior-posterior center of pressure movement, respectively.

3.2   Methods

3.2.1   Participants

Thirty-two participants (17 male; 15 female) between the ages of 18 and 35 were recruited from a University population. The exclusion criteria were any previous history of low back pain that required medical intervention or time off from work longer than three days, previous lumbar or hip surgery, employment in a task that required prolonged static standing during the past 12 months, and the inability to stand for at least two hours. The University of Waterloo Research Ethics Committee approved this study and all participants provided written informed consent prior to starting the study.

3.2.2   Pre-Collection Recordings

After filling out informed consent, the participant's height (via wall mounted tape measure) and mass (via a force platform) were recorded. Participants filled out questionnaires to assess their physical activity levels, pain attitudes, and fear avoidance beliefs. Since these tests were administered on an asymptomatic, rather than clinical population, a modified questionnaire (Nelson-Wong, 2009) containing questions from the Cognitive Risk Profile for Pain (Cook and Degood, 2006), Survey of Pain Attitudes – Brief (Tait and Chibnall, 1997), and Fear Avoidance Belief Questionnaire (Waddell et al., 1993) was used. This modified

questionnaire has been used previously to assess psychosocial differences between pain developers and non-pain developers (Nelson-Wong, 2009). These tests were conducted to ensure any differences between the pain groups were not associated with individual factors. The Minnesota Leisure-Time Physical Activities Questionnaire (Folsom et al., 1986) was used to determine if the activity levels between pain developers and non-PDs differed in the four weeks prior to data collection.

3.2.3   Instrumentation

3.2.3.1 General Lab Setup

The global coordinate system for the laboratory was set up according to ISB standards (Wu and Cavanagh, 1995). With respect to the position of the participant interacting with the workstation, +Y pointed axial, in a cranial direction and parallel with the field of gravity, +X pointed anterior-posterior in the direction that the participant faced, and +Z pointed perpendicular to X and Y towards the participant's right, medial-laterally with respect to the participant.

3.2.3.2 Visual Analogue Scales

A 100 mm visual analog scale was administered to assess a participant's current level of low back pain when each visual analog scale was taken. On a piece of paper, a horizontal line was anchored at the left with "No Pain" and the right with "Worst Pain Imaginable" and participants place one vertical line on the continuum to describe their pain. This has been used previously to identify pain developers during prolonged standing (Gallagher et al., 2014; Marshall et al., 2011; Nelson-Wong and Callaghan, 2010a). Participants filled out a visual analog scale when they entered the lab, immediately after instrumentation was complete, and throughout the prolonged standing task.

3.2.3.3 Force Platforms

Two force platforms (AMTI, Watertown, MA, USA) measured bilateral three dimensional ground reaction forces and moments. This setup allowed for separate analysis of the left and right feet. Each force

plate was sampled at 2048 Hz and collected using a 16-bit analog to digital conversion card with a range

of +/- 10 V.

3.2.3.4 Motion Capture

An Optotrak Certus motion capture system (Northern Digital Inc., Waterloo, ON, sampling at 32 Hz,

Registration RMS Error = 0.37 mm +/- 0.03 mm, RMS Alignment Error = 0.23 mm +/- 0.03 mm) was

used to track movement (Figure 3.1). Rigid bodies tracked segment movement, while anatomical

landmarks were tracked within each individual rigid body (Table 3.1). Rigid bodies were placed on the

participant's back to track gross trunk (level of T9), upper lumbar spine (L1/L2), and pelvis (sacrum)

movement. Movement of the feet was tracked using one rigid body per foot placed on the lateral aspect of

the heel. While the participant stood in an anatomical posture, a digitizing probe with a rigid body

containing four markers and a known location of a point at the end of the probe was placed on the

anatomical landmark of interest (Figure 3.1, Table 3.1) to capture the location of the tip with respect to

the rigid body of interest. This created a fixed relationship with the rigid body of interest and the digitized

anatomical landmark.
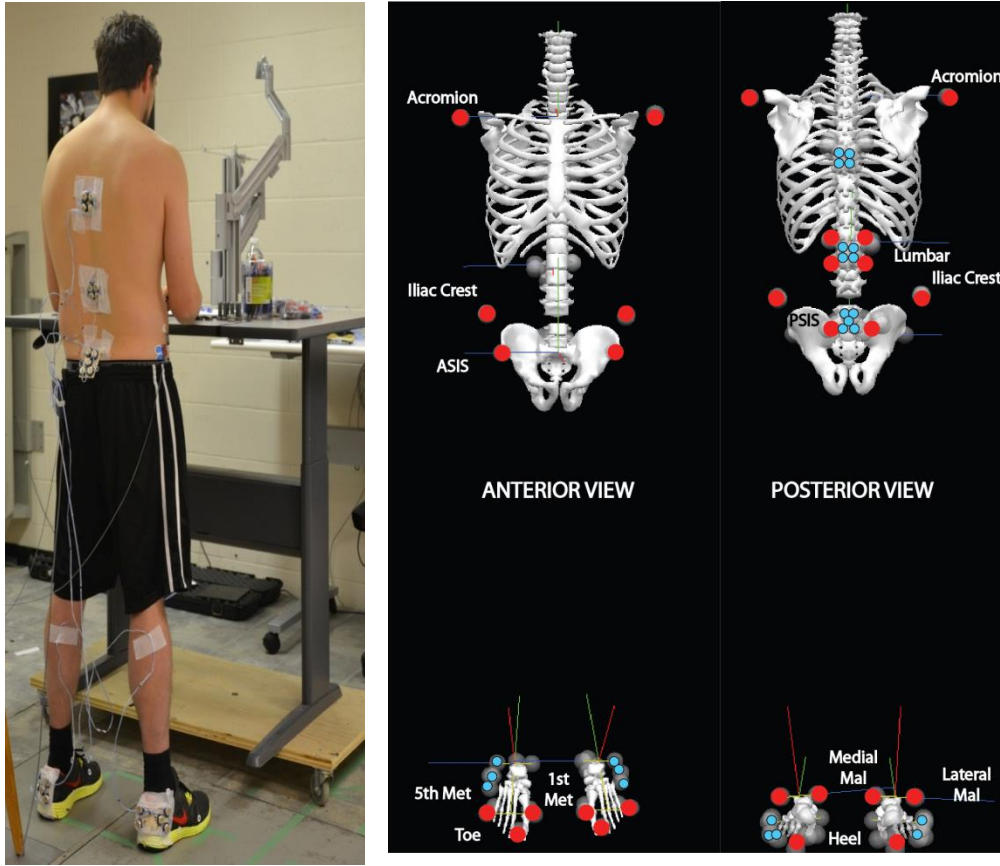
Figure 3.1 Left: Workstation set up. Participants worked at the table performing assembly and sorting tasks. Motion capture was used to track thoracic, lumbar spine, pelvis, and foot movements. There were two force platforms to get separate ground reaction forces and center of pressure for each foot. Right: Anatomical landmarks (red) digitized to define segments. The blue circles represent the markers making up the rigid bodies (they can also be visualized in the left figure). These landmarks were taken while the participant was in the anatomical position. For the remainder of the trial, the landmarks were tracked within the rigid body on their segment to create a fixed relationship between the landmark and rigid body.

Table 3.1. Description of the rigid bodies used in this study and the landmarks that were digitized within each of them.

Rigid body	Anatomical landmark for coordinate systems	Other landmarks/notes
Trunk – contained four markers and was placed at the level of T9	Left and Right Acromion Left and Right Iliac Crest	
Lumbar Spine – contained four markers and was placed at the level of L1/L2	Two points proximal to the rigid body Two points distal to the rigid body	Note: This was done to define the plane of the upper lumbar spine
Pelvis – contained five markers and was placed on the surface of the sacrum	Left and Right Anterior Superior Iliac Spine Left and Right Posterior Superior Iliac Spine	Note: Coordinate system defined a standard technical marker set (Wu et al., 2002)
Left and Right Foot – contained five markers and was placed on the lateral aspect of the heel	Medial and Lateral Malleoli Distal head of the first and fifth metatarsals	Toe: Marked at the distal end of the first toe Heel: Marked at the most posterior aspect of the back of the shoe

3.2.4   Experimental Protocol

Following instrumentation, a 10 second standing trial in anatomical position was captured. Participants provided a visual analogue scale score and performed maximum lumbar spine flexion, extension, and right/left lateral bend and axial twist trials to act as reference posture for the lumbar spine angles.

Participants then began the 2-hour occupational prolonged standing protocol. Participants stood in front of a table adjusted to 5-6 cm below their wrist when their elbows were placed at 90 degrees, which is the position recommended for light work (Kroemer and Grandjean, 1997). Before beginning the protocol, participants received the following instructions, "*Stand in your usual manner as if you were standing for an extended period of time. You cannot lean on the table surface with your upper extremities. Your left and right feet can move around within their respective grey surfaces (force plates); however, the*

two feet must never be in contact with the same one surface at the same time". They also filled out a visual analogue scale score just before the collection began.

In order to simulate an occupational environment, light assembly and sorting tasks were performed. For the assembly tasks, participants assembled and dissembled mechanical pens and nuts bolts and washers. For the sorting tasks, participants sorted cards and money into pre-determined piles. All tasks required participants to work within a primary reach zone – a depth of 25.4 cm from the table edge, width of 101.6 cm, and diameter of approximately 33 to 43 cm with respect to the shoulder joint (Cohen et al., 1997) on a table to eliminate any influence of long reaches on the outcome measures. The tasks were randomized into 15-minute blocks. Every 7.5 minutes, the participant recorded a visual analogue scale score, resulting in 17 scores over two hours.

3.2.5   Data Processing

3.2.5.1 Pain Group Classification

In total, 19 visual analogue scale scores were reported for each participant – one immediately when they enter the lab, one after instrumentation, one before entering the prolonged standing task, and sixteen (one every 7.5 minutes) during the prolonged standing task. To assess the relative increase in visual analogue scale score attributed to the prolonged standing task, visual analogue scale scores upon entering the lab were subtracted from all scores that followed. As a result, all participants began with an initial score of 0 mm.

Participants were categorized as pain developers or non-pain developers based on their reported visual analogue scale scores. A threshold of 10 mm with respect to baseline was used to categorize participants as pain developers based on protocol established previously in studies of prolonged standing (Gallagher et al., 2014; Marshall et al., 2011; Nelson-Wong et al., 2008b; Nelson-Wong and Callaghan, 2010c). Hagg and colleagues (2003) found that when using a visual analogue scale to track worsening of

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low back pain, the clinically important difference for patients to feel their low back pain symptoms worsening was 8 mm. As a result, 10 mm is a conservative threshold to use for categorizing participants as pain developers or non-pain developers.

3.2.5.2 Force Platforms

Voltage data from the two force platforms were imported into Visual3D (Version 4, C-Motion, Inc. Germantown, MS, USA). In order to account for excitation voltage and the amplifier gain setting, a scaling factor was used to account for these two values. A calibration matrix provided by the manufacturer was used to convert the signal from volts into Netwons for Fx, Fy, and Fz, and Newton-meters for Mx, My, Mz. The forces and moments were filtered using a low pass filter (Butterworth, 2nd order, dual pass) with an effective frequency cut-off of 10 Hz based on a residual analysis of the vertical ground reaction force of both places during the first and last 15 minutes of the prolonged standing trials.

3.2.5.3 Motion capture

Motion capture data were imported into Visual3D (Version 4, C-Motion, Inc. Germantown, MS, USA) for post-processing. If marker interpolation was required due to missing or occluded data, a third order polynomial was used to fill gaps of a maximum 16 frames (0.5 seconds). Once interpolated, the signal was low pass filtered (Butterworth, 2nd order, dual pass) with an effective cut-off frequency of 4 Hz, as determined using a residual analysis on one of the digitized foot markers of four different subjects, since the markers of the foot could have the most variable positioning during the prolonged standing trial depending on how a person moved their feet while standing. For the trunk, foot, and lumbar spine, coordinate systems were constructed using the digitized landmarks for each segment. The +y axis was defined be a line connecting the midpoints between the two proximal markers and two distal markers of the segment. The +x was perpendicular to the plane created by the four segment markers. The +z axis was then calculated as the vector perpendicular to the +x and +y planes. For each segment, +y pointing proximally, +x pointing anteriorly, and +z pointing laterally.

For the pelvis, a standard technical marker set was defined according to International Society of Biomechanics standards (Wu et al., 2002), such that the z axis points from the midpoint between the right and left anterior superior iliac spines to the right anterior superior iliac spine, the x-axis lies on a plane defined by the two anterior superior iliac spines, and midpoint of the two posterior superior iliac spines, pointing anteriorly. Finally, +y was orthogonal to +x and +z, and points proximally.

All angles were calculated using the joint coordinate system and rotation sequence of flexion-extension/lateral bend/axial twist. All angles were calculated using the distal segment with respect to the proximal segment. Flexion-extension occurred about the local z axis, lateral bend about the local x-axis and axial twist about the local y axis.

3.2.6   Outcome Measures

3.2.6.1 Lumbar spine movement patterns

A modified algorithm presented for center of pressure data (Duarte and Zatsiorsky, 1999) was run on the angles about the lumbar spine flexion-extension, lateral bend, and axial twist axes to detect the fidgets and shifts (Figure 3.2). The parameters (Table 3.2) were the same as used previously for the lumbar spine during prolonged sitting (Dunk and Callaghan, 2010). Each variable was calculated over a 15 minute block using a sliding moving window (Table 3.2, window length) and expressed as a frequency per 15 minutes.

To determine if a shift in the lumbar spine angle occurred, two sliding widows 15 seconds long and separated by five seconds were computed. A shift was recorded if any two consecutive windows satisfied Equation 3.1:

$$\left| \frac{\overline{x_{W1}} - \overline{x_{W2}}}{\sqrt{SD_{W1}^2 + SD_{W2}^2}} \right| \geq Shift\ threshold \qquad (3.1)$$

where $\overline{x_{w1}}$ and $\overline{x_{w2}}$ were the means of windows 1 and 2 and SD_{W1} and SD_{W2} were the standard deviations of the windows.

To determine if a fidget occurred. A 60 second moving window was computed. All peaks and valleys with an estimated width of less than or equal to 4 seconds (maximum fidget duration) were found. A fidget was recorded when any peak or valley satisfied Equations 3.2:

$$\left| \frac{x_f - \overline{x_W}}{SD_W} \right| \geq fidget\ threshold \qquad (3.2)$$

where x_f is the magnitude of the peak or valley of interest, $\overline{x_W}$ and SD_W are the mean and standard deviation of the moving window, respectively.

The amplitude of each fidget and shift (in degrees) was also quantified for comparison.

Table 3.2 . Parameters for the lumbar spine shift and fidget algorithm detection

Postural Change	Threshold	Window Length	Other
Shift	+/- 5 degrees	Two 15 second windows separated by the shift duration	Shift duration = 5 seconds
Fidget	+/- 3 SD	One 60 second moving window	Max fidget duration = 4 seconds
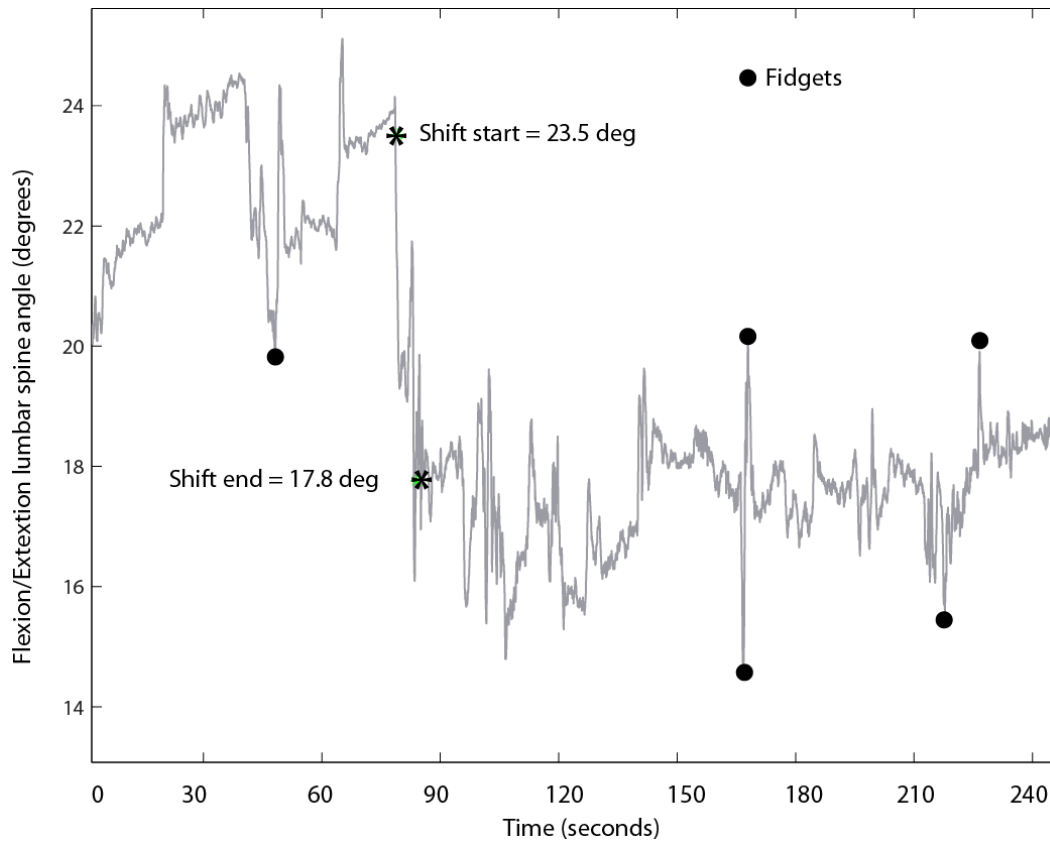
Figure 3.2 Example of flexion/extension lumbar spine fidgets and shifts. There was only one shift (asterisk) found over this 250 second block, and five fidgets (filled circles). Refer to Table 3-1 and text for additional algorithm details.

3.2.6.2 Anterior posterior center of pressure

The center of pressure from the left and right force platforms was calculated separately for every frame over the 2 hours of standing. Since participants were allowed to change their foot position during the protocol, it was important to express the location of the center of pressure with respect to the foot position. The location of the center of pressure was expressed as a percentage of foot length (distance between toe and heel landmarks) (Figure 3.3). Foot length remained constant for all the measurements. The anterior-posterior location was calculated by projecting the location of the center of pressure onto a vector connecting the toe and heel markers. The percentage of foot length was calculated by dividing the

length of this projection by the foot length, and multiplying by 100. A value of 0% would represent that

the center of pressure was at the heel and 100% would represent that the center of pressure was at the toe.
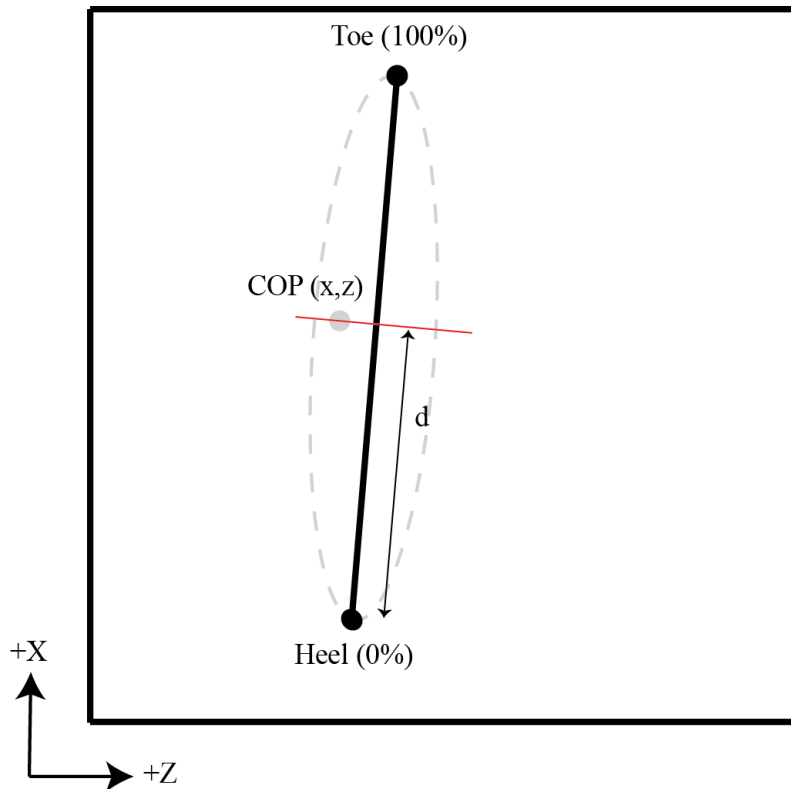
Figure 3.3. An example the anterior-posterior center of pressure location measurement using a top down view of the foot The large box surrounding the foot (dashed oval) represents the force platform. The distance of the center of pressure away from the heel was defined by the length of *d*.

The 10th and 90th percentiles of the anterior-posterior center of pressure movements were

tabulated using an amplitude probability distribution function (APDF). The 10th percentile represented

how close the center of pressure was to the heel and 90th percentile to the toe in each 15-minute block.

3.2.6.3 Body weight transfer algorithm

The vertical ground reaction force from each plate was used to determine when a weight transfer occurred

by normalizing the difference between the right and left ground reaction forces to the sum of both ground

reaction forces at each instance of time (Prado et al., 2011) using Equation 3.3

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$$FvRL(i) = \frac{1}{2}\left(\frac{FvR(i)-FvL(i)}{FvR(i)+FvL(i)}\right) \qquad (3.3)$$

where FvR, L are the vertical ground reaction forces of the right, left force plate (in Newtons),

respectively, and i is the frame number

The cumulative sum of positive and negative changes in the FvRL time series was calculated and

each frame continuously compared to a threshold. When this threshold is exceeded, the algorithm restarts

from zero and this is detected as a weight transfer. A threshold of +/- 0.3 BW was used to detect large

weight transfers. This means that a change in the FvRL time series was defined as a weight transfer if

more than 30% of one's body weight was transferred from one leg to another. We chose to use a threshold

of 30% because if a person was standing with their weight evenly distributed (50-50 split) and then

shifted onto one leg more than 30%. This would be an 80-20 split between the two legs, realistic of

asymmetrical stance. A person may then shift back to even distribution which would be 30% again. Since

pain developers may only perform weight transfers of smaller magnitudes due to hip abductor co-

contraction, weight transfers of amplitudes between 0.1 to 0.29 BW were also quantified. As a result,

each participant had two weight transfer frequencies per 15-minute block: one for the small (0.1-0.29

BW) and one for the large (>0.3 BW) (Figure 3.4).
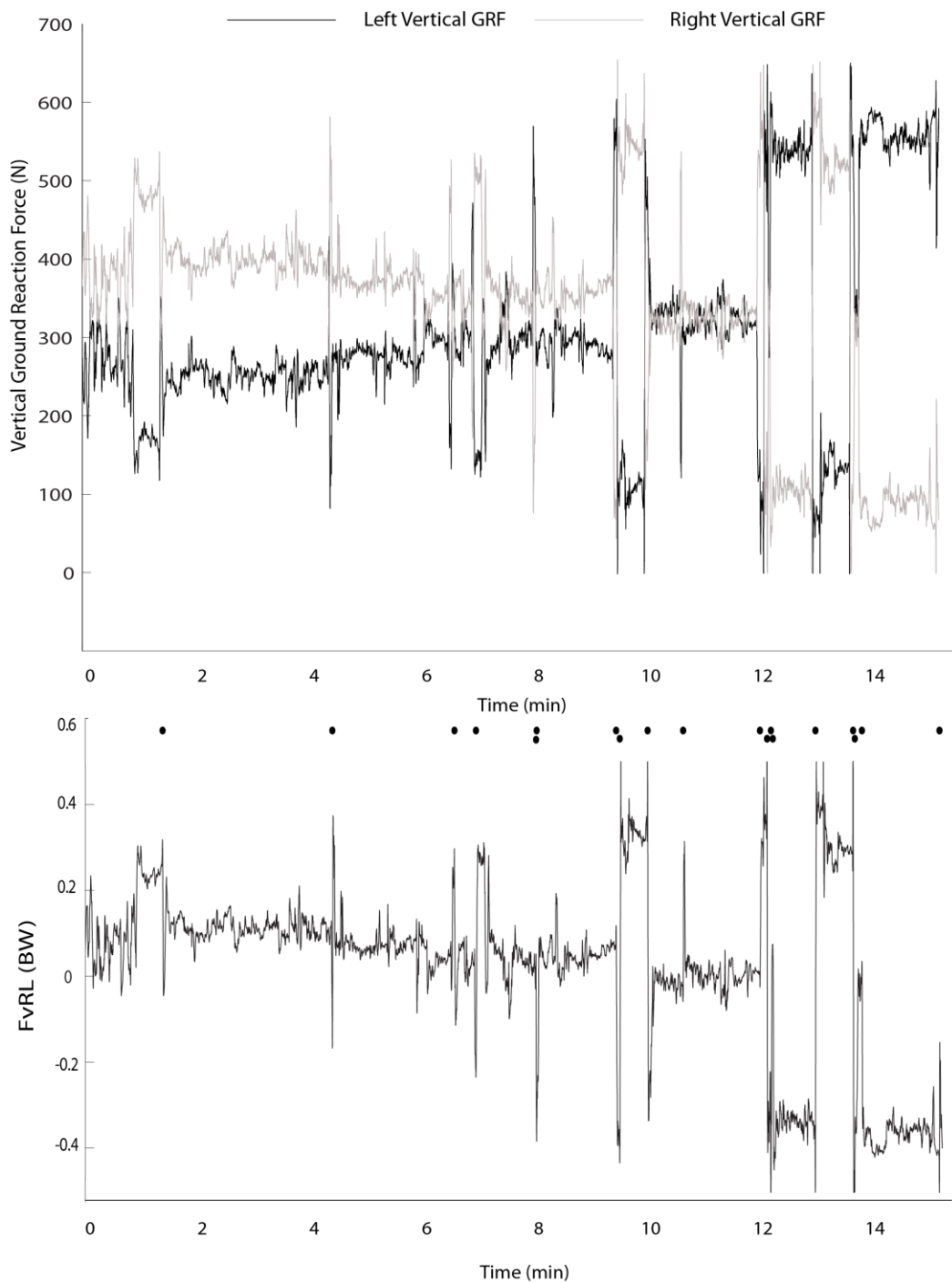
Figure 3.4 A 15 minute left and right ground reaction force tracing (top) and subsequent FvRL tracing. The filled circles represent where the weight transfers >30% BW occurred. There were 19 instances in this tracing. There were 51 weight transfers between 10 and 29 %BW (they were not placed on the graph for clarity purposes)

3.2.7 Statistics

Independent t-tests were run on the participant anthropometrics and questionnaire results to compare pain and non-pain developers. After assessing the time-varying data for each of the variables, it was concluded that if any differences between groups were to be found, they would be within the first 45 minutes of the standing protocol. As a result, the first 45 minutes for each variable was entered into a three-way general linear model with between factors of gender and pain group and a within factor of time. When data did not meet the sphericity assumption (not immediately passed due to time factor containing seven levels), Huynh-Felt-Lecourre Epsilon corrections were used and the *p*-value was adjusted accordingly. If there were no main effects or interactions including gender, the variable was collapsed and entered into a two-way ANOVA. For any significant main effects, Tukey post-hoc tests using the least square means were used. For any significant interactions, simple effects were used to assess the differences. The alpha level of significance was set at $p < 0.05$ for all tests. If a trend towards significance was found ($0.5 < p < 0.10$), the three 15 minute blocks were investigated using non-parametric Wilcoxon scores test, with the *p* value adjusted accordingly to account for multiple comparisons using a Bonferonni correction to 0.0167 (0.05/3).

3.3 Results

3.3.1 Pain Scores and Questionnaire items

Eighteen of the 32 participants (56.25%, Table 3.3) were categorized as non-pain developers, whereas the remaining 14 participants (43.75%) were categorized as pain developers. Of the non-pain developers, 11 were female (61.11%) and seven (38.89%) male. For the pain developers, four of 14 were female (28.57%) and 10 male (71.43%). On average, pain scores passed the 10 mm threshold at approximately 37.5 minutes into the 2-hr protocol (Figure 3.5), with males surpassing the 10 mm threshold on average at 30 minutes, and females at 60 minutes.

Table 3.3 Breakdown of male versus female pain developers and non-pain developers for Study #1

	Female (n)	Male (n)	Total (n)
Non-pain developers	11	7	18
Pain developers	4	10	14
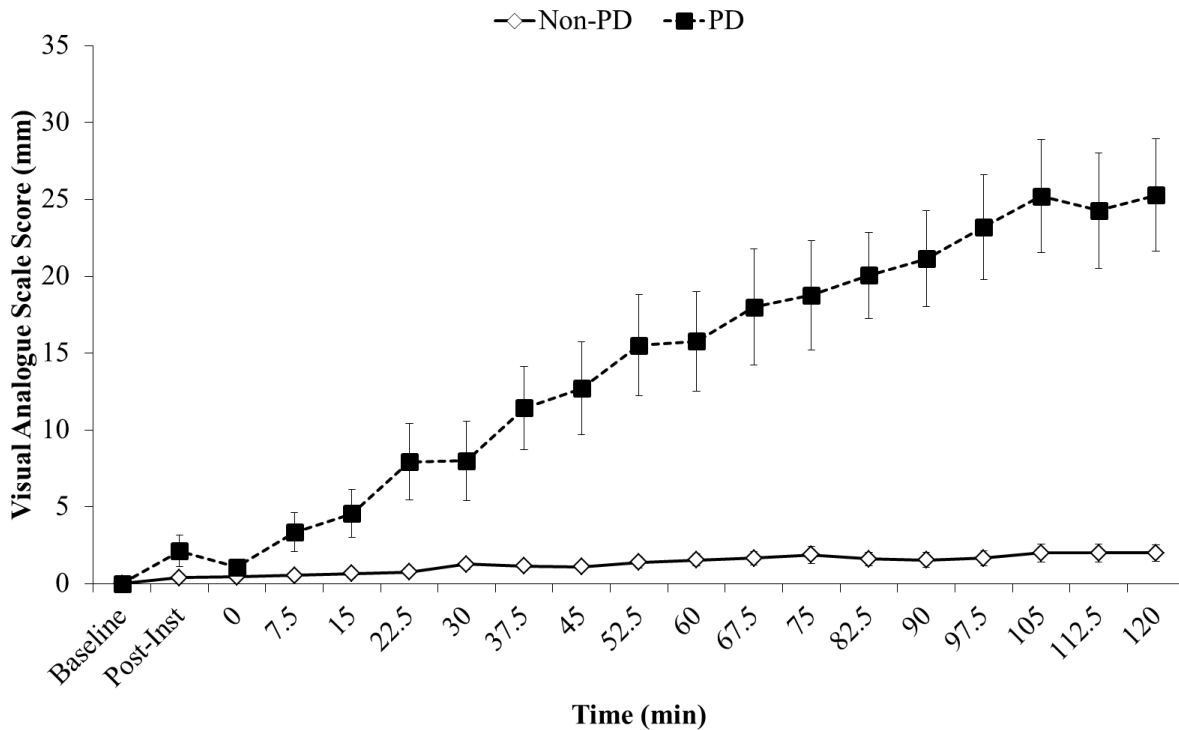
Figure 3.5 Visual analogue scale scores for low back pain over 2-hours of prolonged standing. Two distinct groups emerged during the protocol: those who surpassed the 10 mm threshold set for pain development, and those who did not. Pain development occurred at approximately 37.5-45 minutes. Note: mean and standard error bars. Standard error bars are present for the non-PD; however, are too small to be visible on the graph.

There were no significant differences between pain groups for age, body mass index, MPAQ score, and baseline visual analogue scale score (Table 3.4). The composite scores for each section of the modified CRPP, SOPA-b, and FABQ did not show any differences between pain groups or genders.

Table 3.4 Participant characteristics for Study #1. Note: PD = pain developers

		N	Mean	Std. Deviation	Independent t-test values
Age (years)	Non-PD	18	22.17	2.04	0.31
	PD	14	23.00	2.54	
Body mass index (kg/m2)	Non-PD	18	24.59	3.73	0.08
	PD	14	22.48	2.48	
Minnesota Physical Activity Questionnaire	Non-PD	17	18910.59	18379.06	0.95
	PD	14	18571.21	13622.02	
Baseline low back visual analogue scale (mm)	Non-PD	18	0.56	1.15	0.26
	PD	14	1.36	2.65	
Post Instrumentation low back visual analogue scale (mm)	Non-PD	18	0.60	0.86	0.056
	PD	14	3.36	5.9	

For the remaining variables, while gender was taken into account in running the statistical analyses, it was not a significant factor for any of the variables.

3.3.2 Lumbar spine movements

3.3.2.1 *Lumbar spine fidgets about the flexion-extension axis*

There was a significant difference between the frequency of fidgets for pain developers versus non-pain developers during the first 15 minutes (p=0.0091); however, not at the 30 and 45 minute time points (p=0.2466 and 0.5970, respectively). During the first 15 minutes, non-pain developers performed approximately 13 fidgets (+/- 4.2) then decreased to approximately 10 fidgets per 15 minutes after 30 minutes of standing. Pain developers began with 8 to 10 fidget movements during the first 15 minutes, and for the remainder of the protocol, all participants remained stable at 8-10 fidgets per 15 minutes (Figure 3.6).
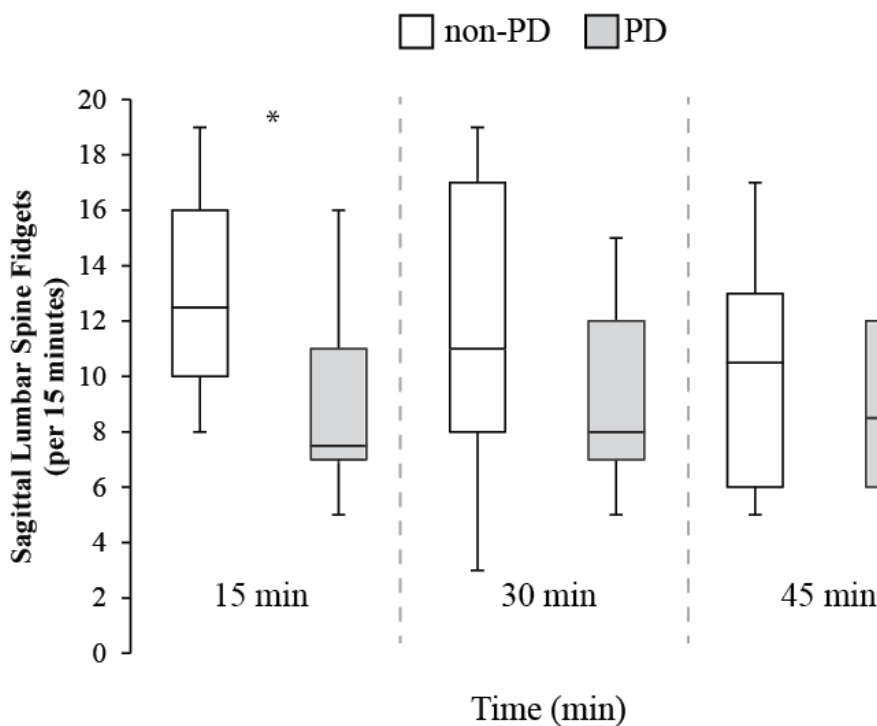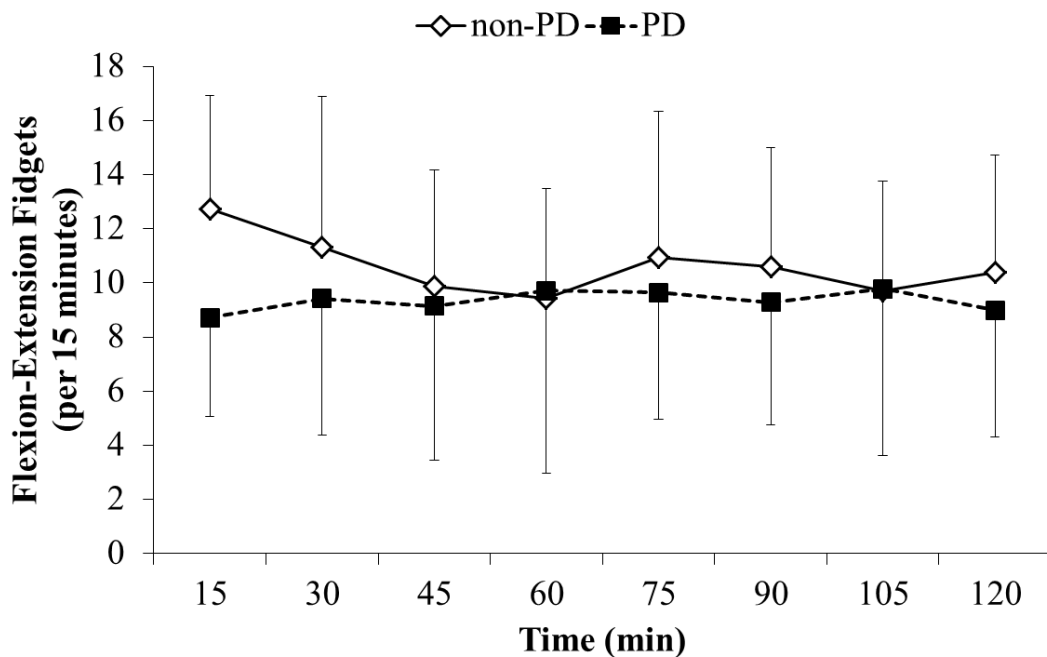
Figure 3.6 Top: Frequency of sagittal lumbar spine angle fidgets. Non-pain developers (non-PD) performed more sagittal lumbar spine fidgets than pain developers (PD) in the first 15 minutes of the trial. Bottom: Plot of medians and interquartile ranges to represent the non-parametric tests run on the initial 45 minutes of the standing trial to confirm that there was a significantly greater frequency of sagittal lumbar spine fidgets performed by the non-pain developers during the first 15 minutes of the prolonged standing protocol.

3.3.2.2 Lumbar spine shifts about the flexion-extension axis

A main effect of TIME was found for the frequency of lumbar spine shifts ($p=0.0003$, df=6, F=5.26). For all participants, there was an increased in the frequency of shifts over time (Figure 3.7).
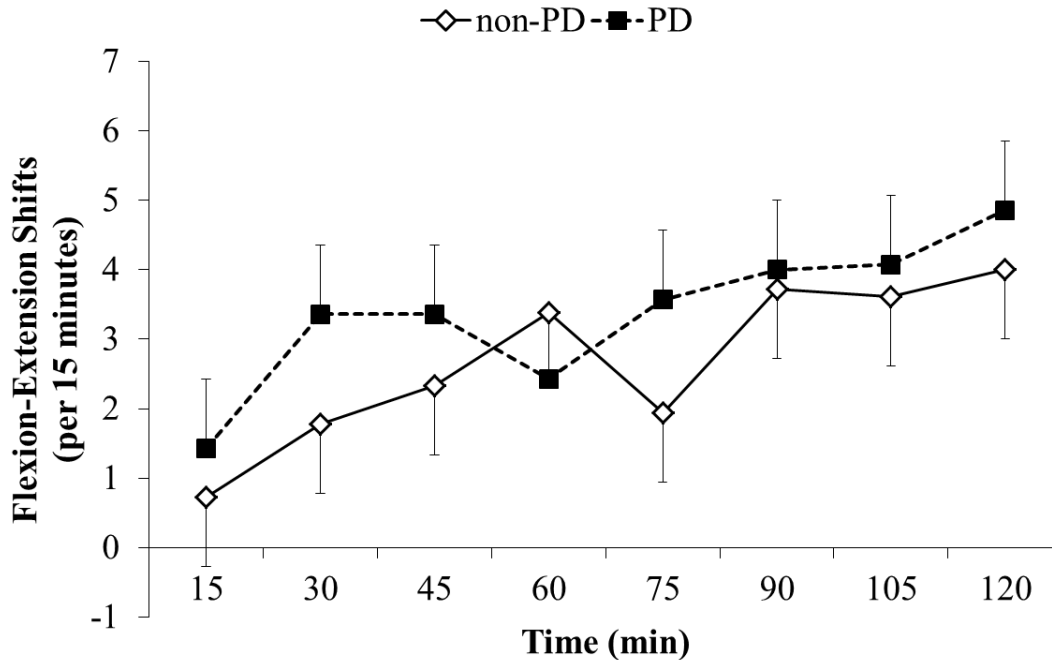
Figure 3.7 Lumbar spine flexion/extension shifts. For both pain groups there was an increase in the frequency of shifts over the entire protocol.

3.3.2.3 Summative movements

The difference in movement frequency (Figure 3.8) during the first 15 minutes was clearly defined by developing an approach to generate a combined metric of the movement patterns that involved taking the square root of the fidget and shift frequency sum of square using Equation 3.4:

$$total\ movement\ estimate\ (per\ 15\ minutes) = \sqrt{(\#\ fidgets)^2 + (\#\ shifts)^2} \qquad (3.4)$$

A significant difference between pain developers and non-pain developers was only found during the first 15 minutes of the standing trial ($p=0.0088$).
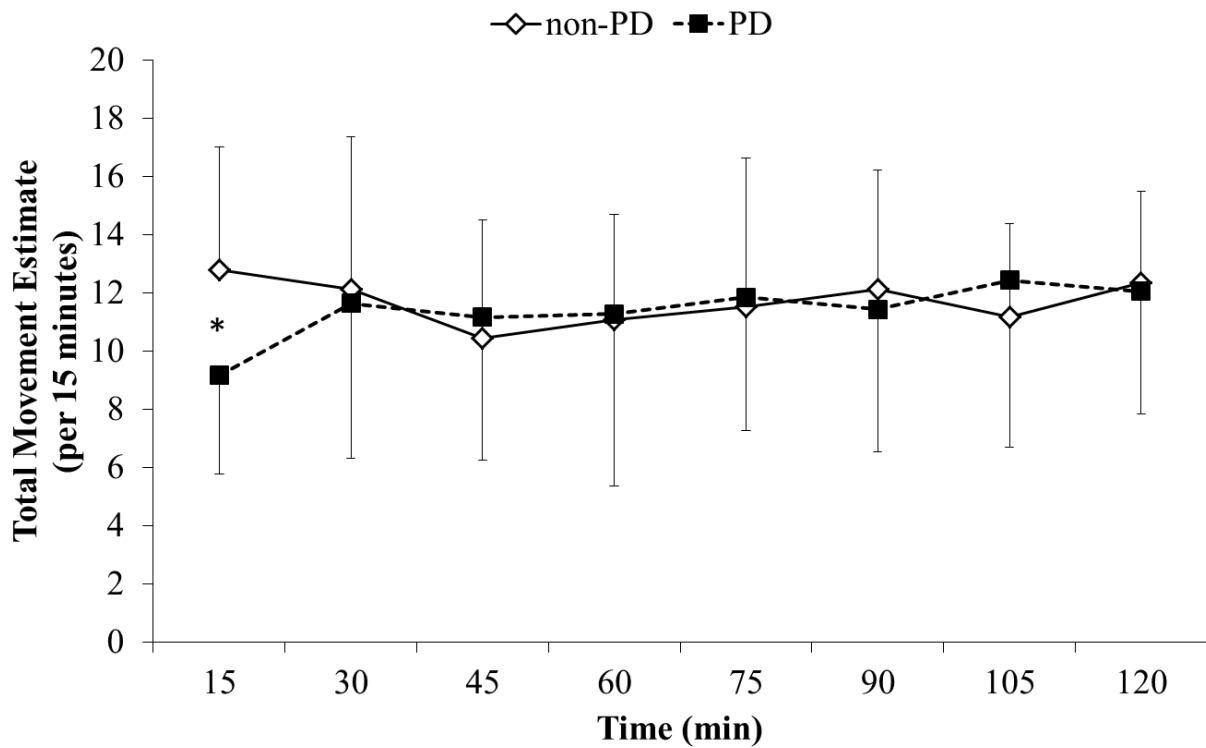
Figure 3.8 Total movement estimate that was calculated by taking the root sum of squares of the sagittal lumbar spine fidgets and shifts. The only difference between the pain groups was found during the first 15 minutes of the prolonged standing trial.

3.3.2.4 Lumbar spine movements about the lateral bend and axial twist axes

There were no main effects or interactions containing pain group for lateral bend and axial twist fidgets and shift frequency (Figure 3.9). A main effect of time was found for lateral bend fidgets ($p<0.0001$, df=6, F=5.68) and shifts ($p=0.0002$, df=6, F=5.43), and axial twist shifts ($p=0.0191$, df=6, F=3.51). For movements about the lateral bend axis, there was a decrease in fidgets and increase in shifts over time. For movements about the axial twist axis, there was an increase in shifts and a consistent number of fidgets over time.
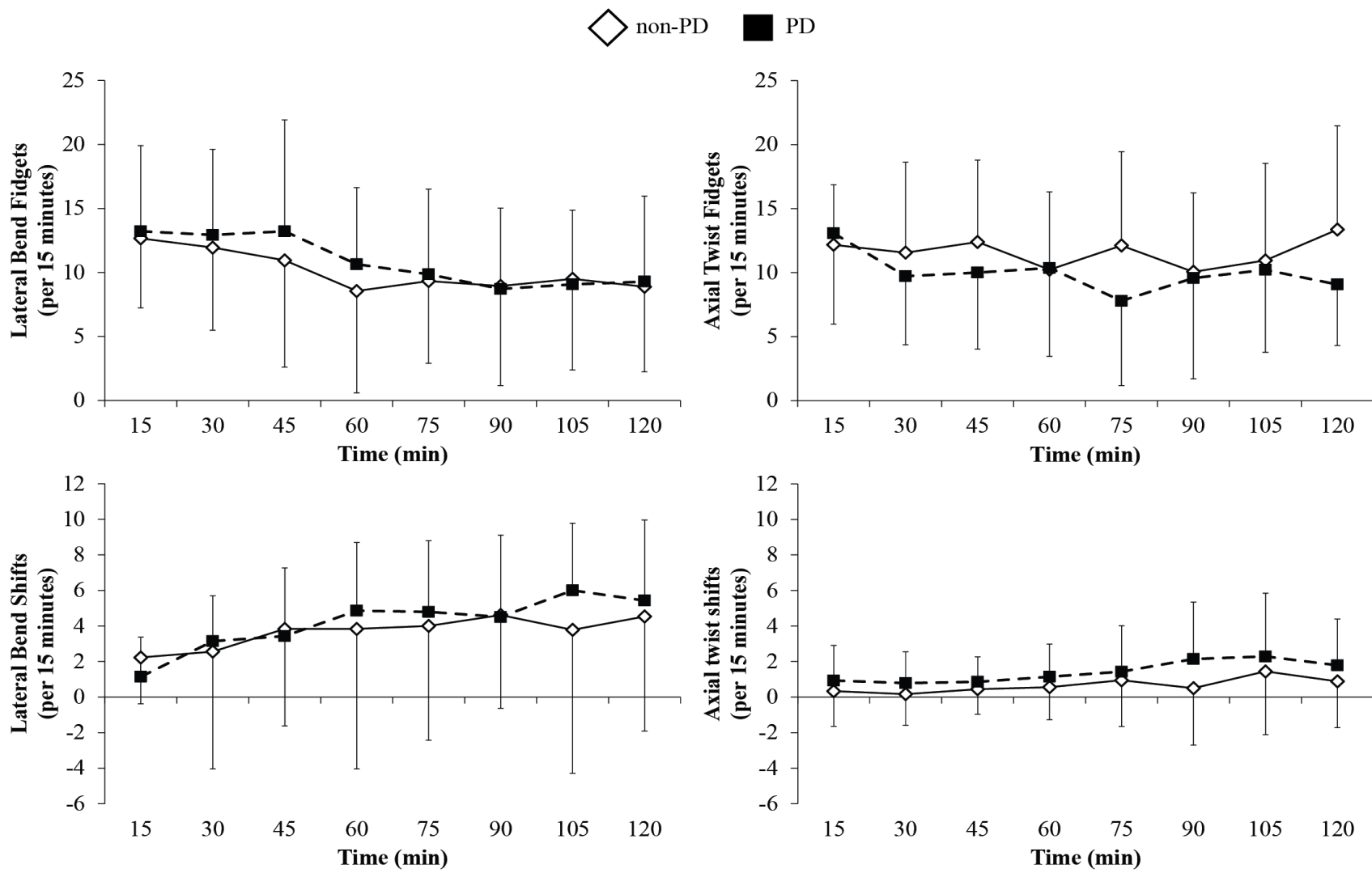
Figure 3.9 Fidgets (top) and shifts (bottom) about the lateral bend (left) and axial twist (right) lumbar spine axes. There was a decrease in lateral bend fidget frequency and increase in lateral bend and shift frequency over time.

3.3.2.5 Movement Magnitude

The magnitude of both the fidgets and shifts could not be evaluated with statistics because participants did not always perform a movement in each block (and therefore no magnitudes were present for that block). For fidgets, pain developers began with a magnitude of 3.5 (+/- 1.9) degrees, while non-pain developers began with magnitudes of 5 (+/- 3) degrees. For both groups, the shift magnitudes ranged from 6-8 degrees over the entire trial.

3.3.3   Adjacent segment movements

3.3.3.1 Body weight transfers

A main effect of TIME was found for the frequency of weight transfers (Figure 3.10) between 10 and 29% BW (p=0.0005, df=6, F=5.95) and those greater than or equal to 30% BW time (p<0.0001, df=2, F=17.52). All participants continuously increased the weight transfer frequency over the protocol duration. For the large body weight transfers, there was also a significant trend towards a main effect of pain group (p=0.0899, df = 1, F=3.08). An independent t-test performed on the first three 15 minute blocks of the large body weight transfers showed a significant difference between pain and non-pain developers only during the first 15 minutes (p=0.0018), with non-pain developers performing more large weight transfers (28.2 +/- 21.5 per 15 minutes) than pain developers (8.2 +/- 7.1 per 15 minutes).
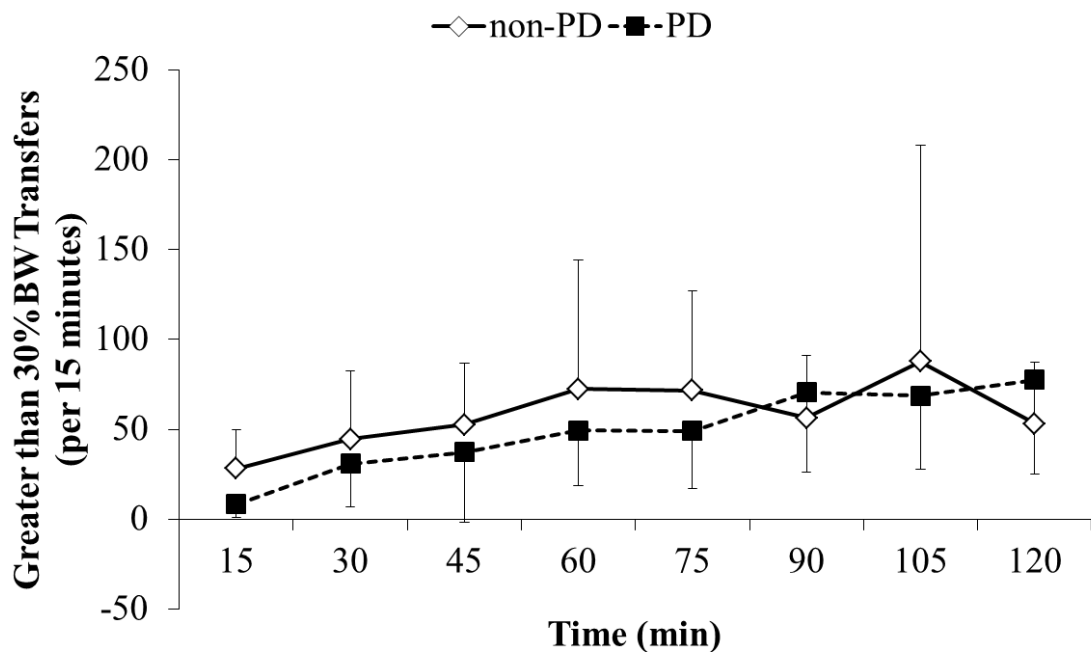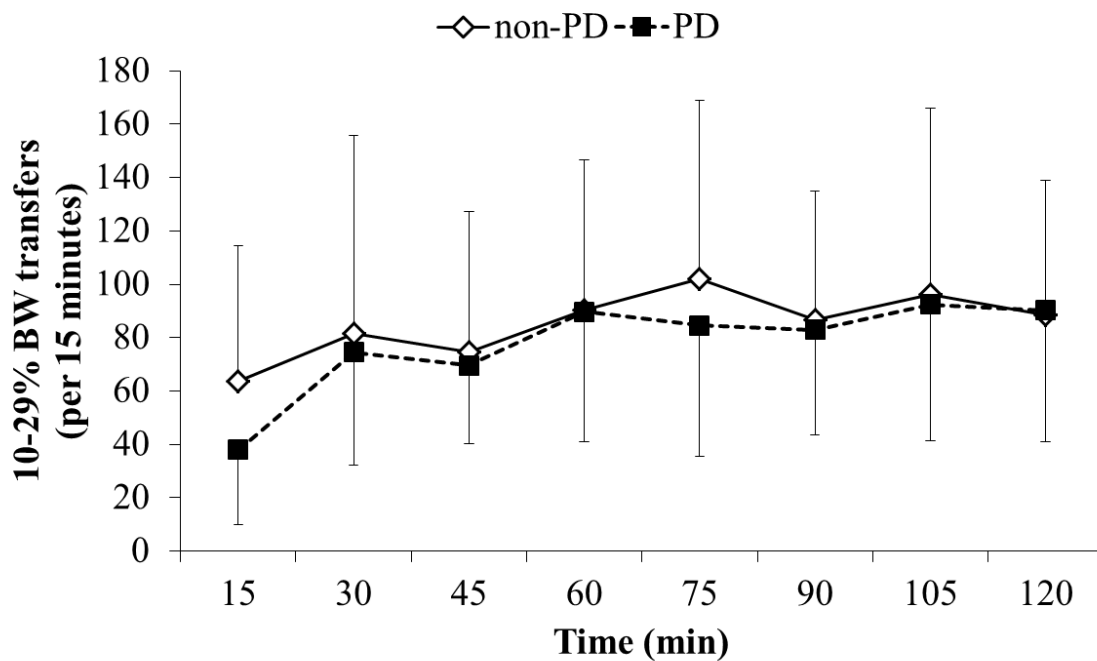
Figure 3.10 Frequency of body weight transfers that were between 10 and 29% (top) and greater than 30% body weight. For both variables, there was a main effect of time such that the frequency of the weight transfers increased over time. When only analyzing the first 45 minutes of standing, there was a trend towards a greater frequency of body weight transfers greater than 30%BW for non-pain developers (non-PD) versus pain developers (PD).

3.3.3.2 Anterior-posterior center of pressure movements

There were no main effects or interactions of center of pressure fidget and shift frequency. Both groups averaged between 10 and 12 anterior-posterior center of pressure fidgets and three to four shifts per each 15 minute block. The 10th percentile anterior-posterior center of pressure location was different between the two pain groups over time (PAIN*TIME interaction df=2, F=3.20, *p*=0.0484). A simple effects post hoc test for the interaction sliced by time found a significant difference between pain developers and non-pain developers (*p*=0.0001) during the first 15 minutes (Figure 3.11). The center of pressure was located closer to the heel for non-pain developers (37.1 +/- 4.6%) than pain developers (39.5 +/- 5.0). To put this into context if two participants had a foot length of 30 cm, the non-pain developers would move their center of pressure 0.77 cm closer to the back of the foot. All participants moved their center of pressure to at most approximately 50% of the foot length. The center of pressure location always remained in front of the ankle (25 +/- 4.4 % of the distance between the heel and toe location).
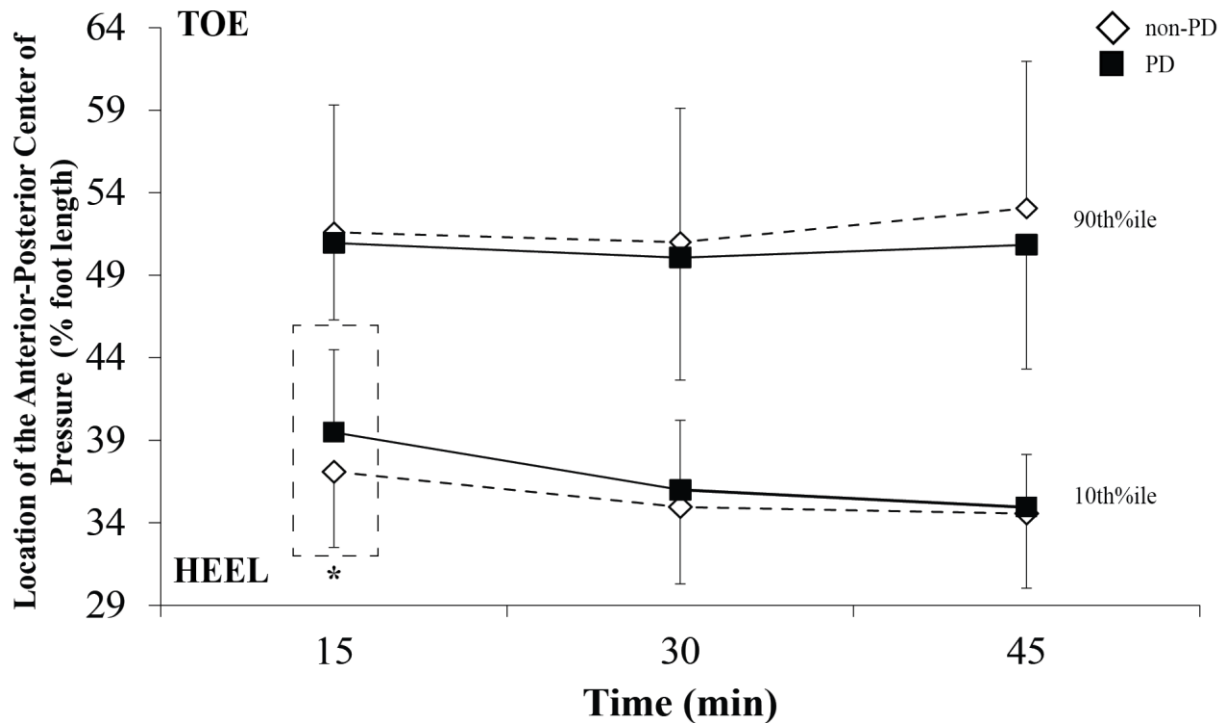
Figure 3.11 10th[th] and 90th percentile locations of the center of pressure (in percent) from the amplitude probability distribution function (APDF). A value of 0 would represent that the center of pressure was at the heel, and 100% at the toe. Thus, the tenth percentile represents how close the center of pressure moved to the heel, while the ninetieth represents how close it came to the toe location. These values are expressed as a percentage of foot length. The sole difference between pain groups was found for the tenth percentile during the first 15 minutes of standing (asterisk). Non-pain developers moved their center of pressure closer to their heel during this time period than pain developers did.

3.4   Discussion

The purpose of this study was to determine if movement patterns of the lumbar spine or at the foot-ground interface could were different between prolonged standing induced low back pain and non-pain developers. During the first 15 minutes of standing, non-pain developers performed more flexion/extension rotation fidgets of the lumbar spine, moved their center of pressure closer to their heel along the length of the foot compared to pain developers, and exhibited a more large body weight transfers between the feet. These differences all occurred prior to the pain developers reached the 10 mm

visual analogue scale threshold, indicating that movement differences between the pain groups are a pre-disposing factor to low back pain development in standing.

The importance of the initial 15 minutes of prolonged standing has been found in previous studies. Gluteus medius co-activation was greater in pain developers than non-pain developers during the first 15 minutes of prolonged standing (Nelson-Wong and Callaghan, 2010c). The first 15 minutes of standing was also found to be a good predictor of a participant's low back discomfort over two hours of standing (Gregory and Callaghan, 2008). The results of the current study add more evidence that pain developers demonstrate pre-disposing patterns of standing that potentially lead to low back pain development.

Fidgeting of the lumbar spine during standing could help prevent low back pain development in non-PDs by reducing static loading on the lumbar spine tissues during the initial 15 minutes. Fidgeting provides variability in loading, preventing sustained static loading of tissues such as the intervertebral disc and facet joint capsule that could lead to changes in posture of the intervertebral vertebrae in a way that would cause more extension to occur and lead to impingement of the facet joint capsule and tissues between the spinous processes (Adams and Hutton, 1985). Less fidgeting of the lumbar spine at the start of standing in pain developers could be due to the higher co-contraction or poor postural control of the trunk musculature. The use of an exercise intervention that concentrated on promoting stabilization of the hip and trunk was effective at decreasing low back pain reports during standing and reduced co-contraction (Nelson-Wong and Callaghan, 2010a). While movement patterns were not examined in that study, the reduction in co-contraction would likely reduce the stiffness and allow for greater ability to generate fidget movements in the lumbar spine.

Transferring body weight from one leg to another is very common when standing (Carlsöö, 1961) and people tend to increase their weight transfer frequency over time (Gallagher et al., 2011). As with the other movement variables, non-pain developers performed a higher frequency of large weight transfers

during the first 15 minutes of standing compared to pain developers, but a similar frequency of smaller transfers. This is different than previous results, which found that the frequency of body weight shifts increased over time (Gallagher et al., 2011); however, large and small body weight transfers were not separated. We hypothesized that a difference between the pain groups would exist because of the higher gluteus medius co-contraction in pain developers. This may explain why pain developers can perform smaller shifts in body weight, but not larger shifts. Bilateral co-contraction may cause stiffening of the hip joint, limiting the joint range of motion. Without this range of motion, pain developers may not have adequate range of motion to perform larger body weight transfers. Conversely, by not performing larger body weight transfers, pain developers rarely unloaded one of their legs, which would inhibit relaxation of the off-loaded gluteus medius muscle (Carlsöö, 1961) and could also explain the higher gluteus medius co-contraction.

Since many of the variables shown to differentiate pain developers and non-pain developers occur at the start of standing, it is important that whatever intervention the worker selects is utilized right when s/he begins to stand. This is especially important when interacting with a standing aid, such as lifting one foot onto an elevated surface. Based on our previous knowledge of interventions after pain development occurs (Gallagher et al., 2014), moving into this elevated foot position may not be sufficient for reducing low back pain if the pain is already present, especially when returning to a regular standing position. Also, while different postures may be effective at altering low back pain, they could also put other parts of the body in a poor posture. For example, using the elevated surface increases discomfort of the supporting leg (Whistance et al., 1995), thus, this posture is also not suitable for prolonged standing. This highlights the important of immediately interacting with any standing and calls for frequently changing postures so that one does not stand in the any position for a long period of time.

While there may be a relationship between movement patterns and low back pain development, it is not appropriate to say that there is causal relationship since the higher muscle co-contraction seen in pain developers is a potential confounding factor. As a result, the goal of altering movement patterns may

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not be the proper place to promote interventions; rather, addressing the muscle co-contraction would be a more appropriate spot for intervention. A secondary result of altering muscle co-contraction could be a change in lumbar spine movement patterns. More research needs to be conducted on the relationship between gluteus medius co-contraction and movement patterns of the lumbar spine and foot-ground interface to determine the most effective place to promote interventions.

A limitation to this study was that movement was assessed indirectly using the ground reaction forces and center of pressure tracings and not with the use of motion capture to track the lower limb kinematics. The ground reaction forces and center of pressure will show if movement occurred, but cannot isolate the kinematic mechanism for this movement. Since this was a static standing task where the participants were constrained where they could stand, little lower limb movement was required and thus motion capture was removed. Second, although we assessed non-sagittal plane movements, this study did not use a task that would have forced the participant to make deliberate axial twist or lateral bend movements. As a result, it is unknown how a task that required participants to twist about their lumbar spine or incorporated side reaches inducing lateral bend motions would affect low back pain development and whether movement in the other two planes would be just as effective as the movement in the sagittal plane.

3.5 Conclusions

More frequent movement of the lumbar spine and large body weight transfers during the first 15 minute of standing distinguished pain developers and non-pain developers. Despite this, it is unknown whether this initial lack of movement in the lumbar spine and large body weight transfers are responsible for pain development, as they may only be a result of poor trunk and hip muscle control. Future studies will look at worker interaction with standing aids at the start of a prolonged standing trial and the influence of successful exercise interventions that work to improve muscle activation patterns on the movement of pain developers during prolonged standing.

4 Study #2: Mild lumbar spine flexion during level ground standing may reduce prolonged standing induced low back pain development

4.1 Introduction

A recent culture shift in the office workplace has resulted in a movement towards standing and sit-to-stand workstations; however, not all workers may tolerate an increase in occupational standing. Shown in both field (Andersen et al., 2007; Roelen et al., 2008; Tissot et al., 2009) and lab studies (Gallagher et al., 2014; Marshall et al., 2011; Nelson-Wong and Callaghan, 2010c), prolonged standing induced low back pain development can occur in workers who have not previously had a low back injury or reported low back pain. The cumulative effects of this pain development result in prolonged standing induced pain developers to be three times more likely to seek medical help for a low back problem within three years (Nelson-Wong and Callaghan, 2014). Despite little scientific evidence on their effectiveness, many people who stand also use improvised or purchased standing aids aimed to reduce low back pain and these aids are commonly recommended by various occupational health and safety associations(Canadian Center of Occupational Health and Safety (CCHOS), 2008; Cohen et al., 1997; Occupational Safety & Health Administration (OSHA), 2012). The underlying mechanism for this pain development during standing is currently unknown; however, the recommendation of standing aid use in the workplace combined with the importance of movement (Gallagher et al., 2014) and posture (Sorenson et al., 2014) in standing point to a relationship between standing posture and low back pain development. As a result, the purpose of this study was to investigate the differences in lumbar spine posture between prolonged standing pain developers and non-pain developers and the subsequent influence of two lower limb posture changing standing aids on lumbopelvic posture.

It is difficult to define what a "typical" or "neutral" standing posture should be since sagittal spinal alignment shows significant variability from person to person even when taken in a standardized posture (Roussouly et al., 2005). A "natural" standing position has been defined as the posture a person would adopt during habitual unsupported standing (Mitchell et al., 2008). In a natural standing posture,

both the upper and lower portions of the lordotic curve are in an extended position (Mitchell et al., 2008). The upper arc of the lumbar lordosis is more consistent from person to person than the lower arc, which is most important in determining a person's global lordosis angle(Roussouly et al., 2005) and has been shown to have a greater range of motion than the upper arc of the lumbar spine (Mitchell et al., 2008). Efforts have been made to classify individuals based on the characteristics of their spine to determine what (if any) characteristics of a standing posture are associated with low back pain development (Mitchell et al., 2008; O'Sullivan et al., 2002; Roussouly et al., 2005). Patients with spinal stenosis have been classified to stand in a state of hyperextension, while participants with spine postures categorized as "well-balanced" had minimal low back pain complaints (Roussouly et al., 2005). Research on participants with and without low back pain show that people can stand in a "sway" posture, which is achieved through extension of the lower arc of the lumbar spine with very little motion of the upper arc(Mitchell et al., 2008) and the pelvis translating anteriorly to the trunk (O'Sullivan et al., 2002). Sway standing is a passive posture associated with an inhibition of the lumbopelvic stabilizing muscles and possibly resulting in greater strain on the passive tissues in the lower lumbar spine (O'Sullivan et al., 2002).

Two studies have characterized lumbopelvic posture of prolonged standing induced pain developers versus non-pain developers. Pain developers have been shown to stand with greater lumbar lordosis (extension) than non-pain developers during a prolonged standing task (Sorenson et al., 2014); however, the margin of safety with respect to maximum extension angle did not differ between the two groups (Gallagher et al., 2014). There was also a positive relationship between the lordosis angle and maximum self-reported rating of low back pain on a visual analogue scale –the more extended a person stood, the higher their maximum self-reported rating of low back pain (Sorenson et al., 2014). Pain developers also stand with more thoracic extension during prolonged standing than non-pain developers (Gallagher et al., 2014). The larger lordotic curve and thoracic extension point to a potential sway standing posture by PDs. Since there is greater variability of the lower lumbar lordosis arc (Roussouly et

al., 2005), it is possible that this is the region that differs between pain developers and non-pain developers during level ground standing but has not been specifically evaluated to date.

Two general categories of standing aids exist – ones that involve a change in floor surface but no change in lower limb posture (e.g. anti-fatigue matting) or ones that require the worker to change their lower limb posture (e.g. foot stool). For the second category, the theory is that by influencing the kinematic chain through postural changes of the lower limbs, you change the posture of the pelvis and lumbar spine. A movement that causes the pelvis to rotate will affect lumbar spine angle, such that tilting the pelvis posteriorly results in lumbar flexion, while anterior rotation results in lumbar extension (Day et al., 1984). Additional movement of the lower limbs, such as a smaller in the trunk to thigh angle, will also cause the pelvis to rotate posteriorly and flatten the lumbar spine (Keegan, 1953). A smaller lumbar lordosis lumbar lordosis (flexion) is associated with a smaller thigh to trunk angle (Bridger et al., 1989) and changes in pelvic tilt alone do not provide the maximum changes in lumbar spine angle, which are further altered by changing lower limb or trunk posture (Levine and Whittle, 1996).

Currently, the most recommended standing position for those who stand for long periods of time is to elevate one leg onto a surface or bar (Canadian Center of Occupational Health and Safety (CCHOS), 2008; Cohen et al., 1997; Occupational Safety & Health Administration (OSHA), 2012), and a second is using a sloped surface(Nelson-Wong and Callaghan, 2010b). The use of a bar rail or platform provides a stable standing posture that leaves the hands free and prevents excessive spinal curvature (Whistance et al., 1995). Elevating one leg onto a surface causes trunk (Whistance et al., 1995) and lumbar spine flexion (Dolan et al., 1988). Standing on sloped surfaces also causes changes in pelvic and lumbar spine angles. When standing in plantar flexion induced by a declining surface, there is a posterior rotation of the pelvis (Nelson-Wong and Callaghan, 2010b) and flattening of the lumbar spine (Gallagher et al., 2013), while standing in dorsiflexion associated with an inclined surface causes greater trunk extension, anterior rotation of the pelvis(Nelson-Wong and Callaghan, 2010b), and lumbar extension (Gallagher et al., 2013) compared to standing on a flat floor or surface. By examining the influence of standing aids on

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lumbopelvic posture, we can determine what could be helpful at reducing low back pain and also parse out the fundamental posture differences that may exist between pain developers and non-pain developers.

The primary purpose of this study was to examine the sagittal lumbopelvic postural differences between prolonged standing low back pain developers and non-pain developers and the subsequent influence of two standing aids that induce changes in lower limb posture. We hypothesized that pain developers would stand with a posture closer to their end range of motion in extension compared to non-pain developers. Due to the variability of the lower arc of the lordotic curve and the bottom up influence of the standing aids, we hypothesized that a majority of the differences between pain developers and non-pain developers would be found in the lower lumbar arc. We also hypothesized that the use of both standing aids would bring all participants into higher flexion. To evaluate this, participants whose pain categories were previously defined in a prolonged standing protocol had four sagittal plane radiographs taken to characterize their level ground extension, and standing aid influenced lumbar spine postures. Measurement of the lower lumbar arc, total lordosis, and individual intervertebral angles were compared between the three standing postures.

4.2   Methods

4.2.1   Participant information

Seventeen participants, nine pain developers and eight non-pain developers, participated in this study (Table 4.1). Each participant was recruited from a previous prolonged standing study (Section 3.2.1) to prevent unnecessary exposure to radiation without a-priori knowledge of their prolonged standing pain development status. In order to minimize health risks associated with elevated ionizing radiation exposure, participants were excluded if they have had a radiographic investigation within the past year (excluding dental x-rays) or were exposed to radiation in their occupation. A female participant was excluded if there was any chance that she might be pregnant. The University of Waterloo Research Ethics

Committee approved this study and all participants provided written informed consent prior to starting the study.

Table 4.1. Participant details and anthropometrics. Note: table values are mean (standard deviation)

	Age (years)	Height (m)	Mass (kg)
Non-pain developers			
Male (n=4)	22 (2.2)	1.82 (9.5)	88.9 (12.7)
Female (n=4)	21.5 (0.58)	1.68 (7)	62.0 (12.4)
Pain Developers			
Male (n=5)	22.8 (1.9)	1.82 (9.4)	77.6 (6.9)
Female (n=4)	22.3 (1.5)	1.65 (5.2)	56.3 (7.7)

4.2.2   Radiograph Instrumentation

Radiographs were taken with a diagnostic x-ray high voltage generator machine (DX-D 300 DR, AGFA Healthcare) by an experienced licensed technician. The central x-ray tube was directed perpendicular to the participant, 2.5 cm superior to the iliac crest, and slightly posterior to the mid-axillary line with the focal field distance set at 1.02 cm (Botranger, 2002). The collimation was set to include T12 superiorly, S3 inferiorly, and slightly lateral to include the greater trochanter. Technique factors were individually adjusted to the thickness of the trunk in the coronal plane of each participant and were kept consistent for each radiograph. For this study, the technique factors were 80 KVP (kilovolt peak) and 58 mA.s (milliampere-seconds) on average and yielded an average entrance dose of 704 mRem, which is under the Healing Arts Radiation Protection Act maximum dose limit of 2000 mRem for lateral lumbar films (Healing Arts Radiation Protection Act, 1990). The exposure dosage is then converted into an effective dose that takes into account the distribution of the radiation dose among radiosensitive organs in the body and is the sum of individual organ doses weighted according to the relative sensitivity of the organ to radiation induced somatic or genetic effects (Wall and Hart, 1997). The typical effective dose for a lateral lumbar radiograph is 30 mRem (Wall and Hart, 1997). When taking multiple x-rays, as done in this study, the effective dose of an examination is the sum of the effective doses for each radiograph (Wall and Hart, 1997); therefore, the effective dose for this radiographic examination was a maximum 180 mRem (1.8 mSv; maximum of six x-rays allowed as per ethical review). This is comparable to the natural background

radiation that a person is exposed to over six months (National Council on Radiation Protection and Measurements, 2009). The Radiological Society of North America and American College of Radiology (2011) describe the lifetime risk of cancer for this effective dose as very low. An effective dose of 10 mSv may be associated with an increase in the possibility of fatal cancer by 0.05% (US Food and Drug Administration, 2009).

4.2.3   Protocol

Radiographs of four different postures were taken for each subject (Figure 4.1):

1. Level ground standing to emulate the person's regular standing posture

2. Standing with one foot raised on a platform to represent the most likely posture when using this standing aid. The height of the platform adjusted so that a participant's thigh to trunk angle was 135 degrees. This posture is described as the physiological normal for the lumbar curve compared to erect standing where some curvature of the lumbar spine is still maintained (Keegan, 1953)

3. Standing in plantar flexion on a sloped surface of 16 degrees

4. Maximum lumbar spine extension: the participant was told to keep their knees locked and bend backwards about their lumbar spine without shifting their hips forward

The level ground standing position was always taken first in order to obtain a baseline posture for comparison. Radiographs of the standing aids were taken next and the order of the elevated and sloped surfaces were randomized for each participant. The last radiograph taken was always the maximum lumbar spine extension position.
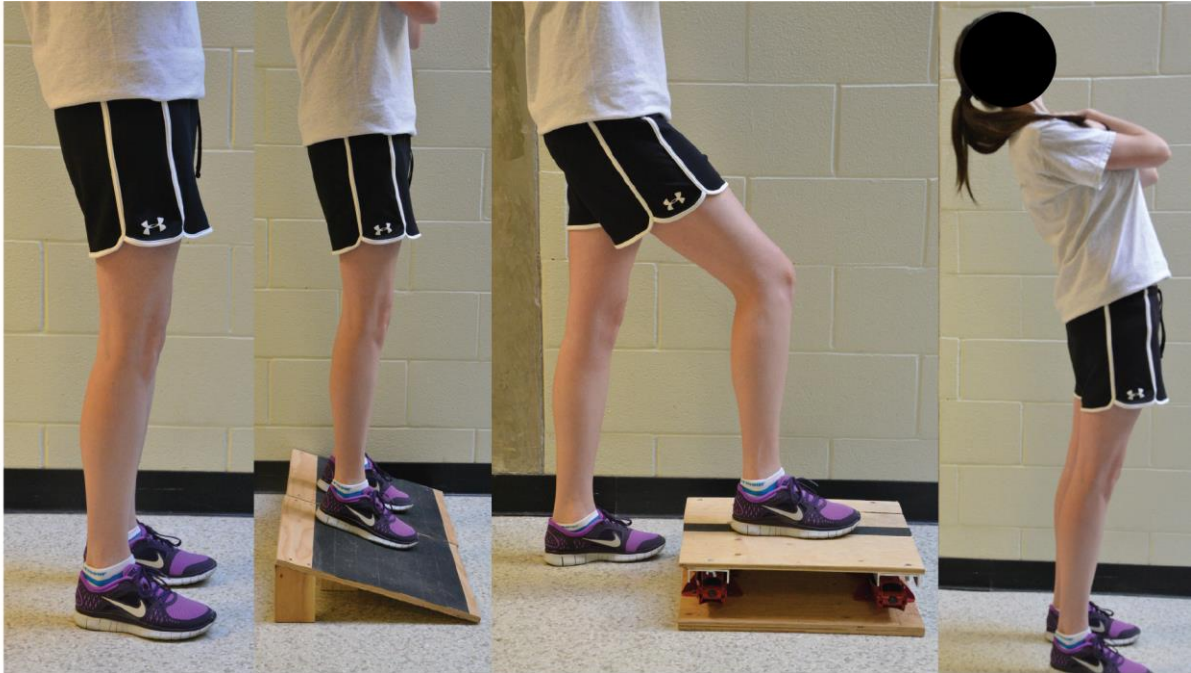
Figure 4.1. Example of the standing conditions (left to right) – level ground, sloped surface, one leg elevated, and maximum lumbar spine extension.

Since upper extremity position alters lumbar spine posture (Stagnara et al., 1982), arm position was standardized between all of the radiograph measurements. Participants stood with their arms replicating the posture required to perform a light assembly task and positioned their neck to gaze at their hands. To prevent superimposition of the diaphragm over the vertebral bodies of the upper lumbar spine, radiographs were captured on a participant's suspended expiration. For the maximum extension radiograph, the participant was asked to take the position and the radiograph was taken.

4.2.4 Lumbopelvic Angle Measurements

Radiographic measurements were completed using eFilm workstation software (v 2.0, Merge Healthcare, Milwaukee, WI) on a 52 cm tall by 32 cm wide screen. The vertical and horizontal pixel spacing was 0.139 mm. Sagittal measurements of lumbar lordosis, lumbosacral lordosis, and lumbar intervertebral disc angles were measured using the methods presented by Yochum & Rowe (1996) (Figure 4.2). *Lumbar lordosis angle* was taken by drawing a line parallel to the superior endplate of the first lumbar segment

and a second line drawn through the inferior endplate of the fifth lumbar segment. Two additional lines, one perpendicular to each of these lines, were drawn and the angle that these two perpendicular lines intersect was measured. For *lumbosacral lordosis angle* , first, the centers of the 3rd and 5th lumbar vertebral bodies were located using intersecting diagonal lines and a line was drawn to join the midpoint of these two vertebral bodies. The midpoint of the S1 vertebrae was located in the same manner and a line was drawn connecting the midpoints of L5 and the sacrum. The angle formed by these two lines was measured posteriorly. *L1/L2 and L5/S1 intervertebral joint angles* were measured by drawing two lines – one parallel to the inferior endplate of the superior vertebrae and the second parallel to the superior endplate of the inferiorly adjacent vertebrae. These lines were extended posteriorly until they intersect and the angle formed by these lines was measured. The experimenter was blinded to the participant's pain status and the order of the measurements was randomized for each participant. The magnitude of each angle measured for the maximum extension trial was subtracted from the corresponding measures in the level ground, elevated, and sloped standing conditions. This meant that all angles were expressed with respect to maximum extension.

To assess repeatability of the radiograph measures, the lumbosacral lordosis angle for level ground standing was measured on two occasions by one rater. This measurement was chosen because of previous research pointing to this angle being the region of greatest variability within a sample of the population (Roussouly et al., 2005). The reliability and repeatability of the lumbar lordosis angle measurement has been assessed previously in our research laboratory (De Carvalho et al., 2010). On average, two measurements of lumbar lordosis angle differed by 0.1 +/- 2.3 degrees and 0.5+/- 2.2 degrees for two separate raters, and there were no differences between the two measurements within or between raters.
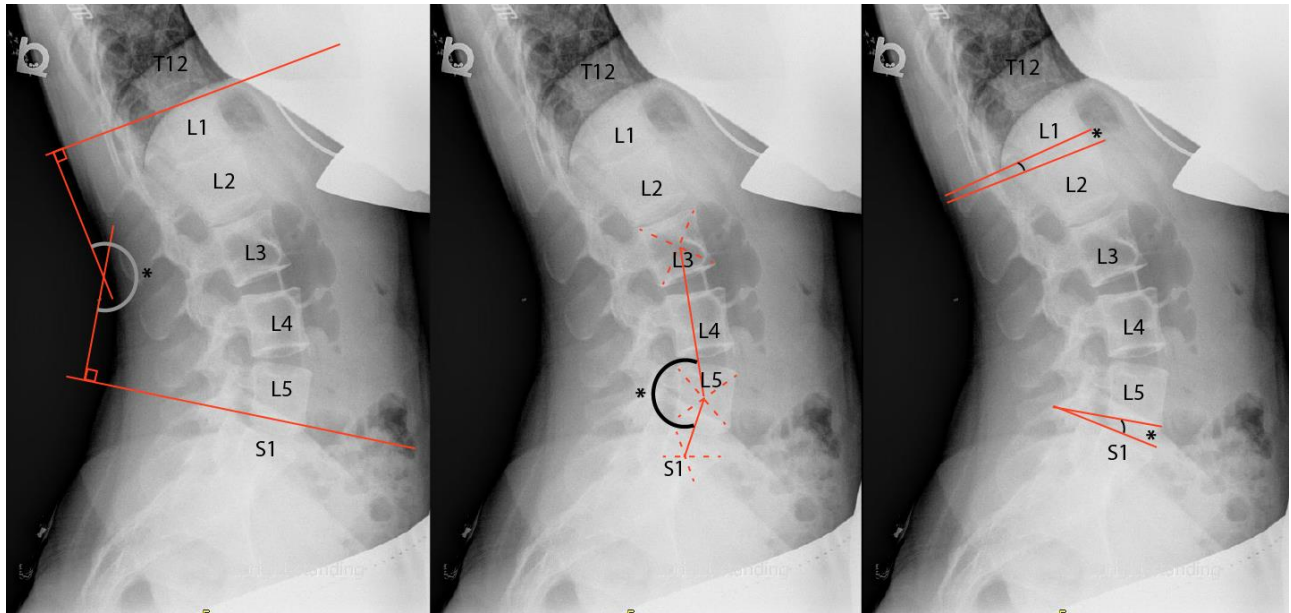
Figure 4.2. Radiograph measurements (from left to right): Lumbar lordosis, lumbosacral lordosis, intervertebral joint angle. Measurement protocol based on Yochum and Rowe (1996). Asterisks and arcs denote the reported angle.

4.2.5   Statistical Analysis

A two-way general linear model with one between factor of pain group (pain/non-pain developer) and one within factor of standing posture (level ground/elevated/sloped) was run on each angle measurement. A Cohen's f statistic (Ellis, 2010) was calculated from the effect and error Sum of Squares using Equation 4.1:

$$f = \sqrt{\frac{\frac{SS_{effect}}{SS_{effect}+SS_{error}}}{1-\frac{SS_{effect}}{SS_{effect}+SS_{error}}}} \tag{4.1}$$

where SS_{effect} is the Sum of Squares of the specific effect (main effect or interaction of interest) and SS_{error} is the Sum of Squares error term for the ANOVA.

If there was a trend towards a main effect of posture, a Wilcoxon signed rank sum test was performed between the results of level ground standing and the standing posture with the greatest angular

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difference from level ground. Unless otherwise noted, the level of significance for all tests was set at alpha=0.05.

A sample size analysis was also run on variables that approached significance for a main effect of pain group in order to determine how many participants would be required to obtain significance for level ground standing in a future study. This was done using the pooled standard deviation from the pain developers and non-pain developers groups during level ground standing, the means of each group, a power of 0.8, and alpha = 0.05.

To assess the repeatability of the lumbosacral lordosis angle measurement, a paired t-test was run between the first and second measurements in level ground standing. A correlation was run between the average of the two measurements and their difference to determine if there was a relationship between the mean and the difference. A Bland-Altman analysis (Bland and Altman, 1986) was also conducted to assess agreement and repeatability of the lumbosacral lordosis measurement (SAS v. 9.4, SAS Institutes Inc., Cary, NC) to provide an accurate representation of whether there may be clinically relevant discrepancies between two measurements. The measurements were taken approximately one year apart, without the rater having any practice measuring the angles; therefore, they represent a worst-case scenario for intra-rater error.

4.3 Results

4.3.1 Pain Group Differences

Exposure to ionizing radiation and the small sample size approved by the ethical review for this study impacted the ability to find significant main effects between the pain groups. The variable with the strongest main effect was lumbosacral lordosis angle (p=0.1318, df=1, F=2.54). Further investigation of the ANOVA produced a Cohen's f value of 0.41 (Table 4.2), signifying a large effect (f >0.40) and suggesting that although not significantly different, there may be a clinical meaning to the difference in

this measure. Pain developers had 2.9, 2.1, and 3.2 degrees more lumbosacral lordosis extension than non-ain developers in the level ground, sloped, and elevated conditions, respectively (Figure 4.3, top left).

A main effect of pain group for the L1/L2 intervertebral joint angle was not found (p=0.2464); however, evaluation of the ANOVA produced a Cohen's f of 0.29 (medium effect). Pain developers had 2, 2.1, and 2.6 degrees more L1/L2 intervertebral joint angle flexion than non-PDs, respectively (Figure 4.3, bottom left). Similarly, a main effect for pain group for the L5/S1 intervertebral joint angle was not found (p=0.2635), but evaluation of the ANOVA produced a Cohen's f of 0.31 (medium effect). Pain developers had 2.8, 1.5, and 1.9 degrees more L5/S1 intervertebral joint extension than non-pain developers (Figure 4.3, bottom right).

There were no significant differences in lumbar lordosis angle (p=0.9216). The greatest difference between lumbar lordosis occurred during the sloped condition, with pain developers having 2.7 degrees more lumbar lordosis extension than non-pain developers (Figure 4.3, top right).

4.3.2 Posture Differences

The introduction of standing aids had a significant impact on lumbar spine postures. A main effect of posture was found for lumbosacral lordosis angle (p=0.0003, F=10.59, df=2; Figure 4.3). There was a significant difference between elevated and level ground (p=0.0003) and sloped conditions (p=0.0133); however, no differences were found between sloped and level ground (p=0.3167). Participants stood with 3.4 and 4.2 degrees more flexion during the elevated condition (non-pain developers = 8.9 (4.6), pain developers= 6.8 (3.5)) than in the level ground condition (non-pain developers = 5.5(3.9); pain developers = 2.6(4.4)).

For lumbar lordosis angle, the main effect for posture was not significant (p=0.0634, df=2, F=3.03). Further investigation (Table 4.2) demonstrated a Cohen's f of 0.45 (large effect), signifying a potential clinical difference. A Wilcoxon signed rank sum test was run on the elevated position lordosis angle versus level ground and a significant difference was found (p=0.0421). The lumbar lordosis angle

(Figure 7) was more flexed during elevated (non-pain developers = 14.4(9.2); pain developers = 13.4(9.11)) compared to level ground standing (non-pain developers = 10.9(7.7); pain developers=11.5(8.2)). We only compared these two positions because both level ground and elevated conditions showed similar trends between the pain groups.

The L1/L2 intervertebral joint angle demonstrated a main effect of posture (p=0.0307, df=2, F=3.92). There was a significant difference between sloped and level (p=0.0252), but not the elevated (p=0.2139). Elevated and level ground standing were not different from each other (p=0.5539). The sloped condition produced the most L1/L2 intervertebral joint flexion (pain developers=5.5(5.2), non-pain developers=3(3.2)) compared to level ground and elevated standing

Table 4.2. Summary of ANOVA, effect size, 95% Confidence Intervals and required sample sizes for significance. Note: Asterisks for the Cohen's f are as follow: ***Large Effect (f>0.40), **Medium Effect (0.39>f>0.25), *Small Effect (0.24>f>0.1)[1]

	LSL	LL	L1/L2	L5/S1
***p* value**				
Pain	0.1318	0.9216	0.2464	0.2635
Posture	0.0003	0.0634	0.0307	0.5317
Pain*Posture	0.7901	0.2166	0.8406	0.8632
Cohen's *f*				
Pain	0.41***	0.026	0.29**	0.31**
Posture	0.84***	0.45***	0.51***	0.21*
Pain*Posture	0.12*	0.23*	0.11*	0.1*
95% CI for pain vs. non-pain developers in level ground standing	(-7.27,1.39)	(8.8,-7.6)	(-6.3,2.31)	(-1.7,7.33)
Sample size per group for pain group significances in level ground standing	29	n/a	39	31

Note: LSL = Lumbosacral lordosis angle; LL = lumbar lordosis angle; L1/L2 = L1/L2 intervertebral joint angle; L5/S1 = L5/S1 intervertebral joint angle. [1]Thresholds for Cohen's f statistic(Ellis, 2010)
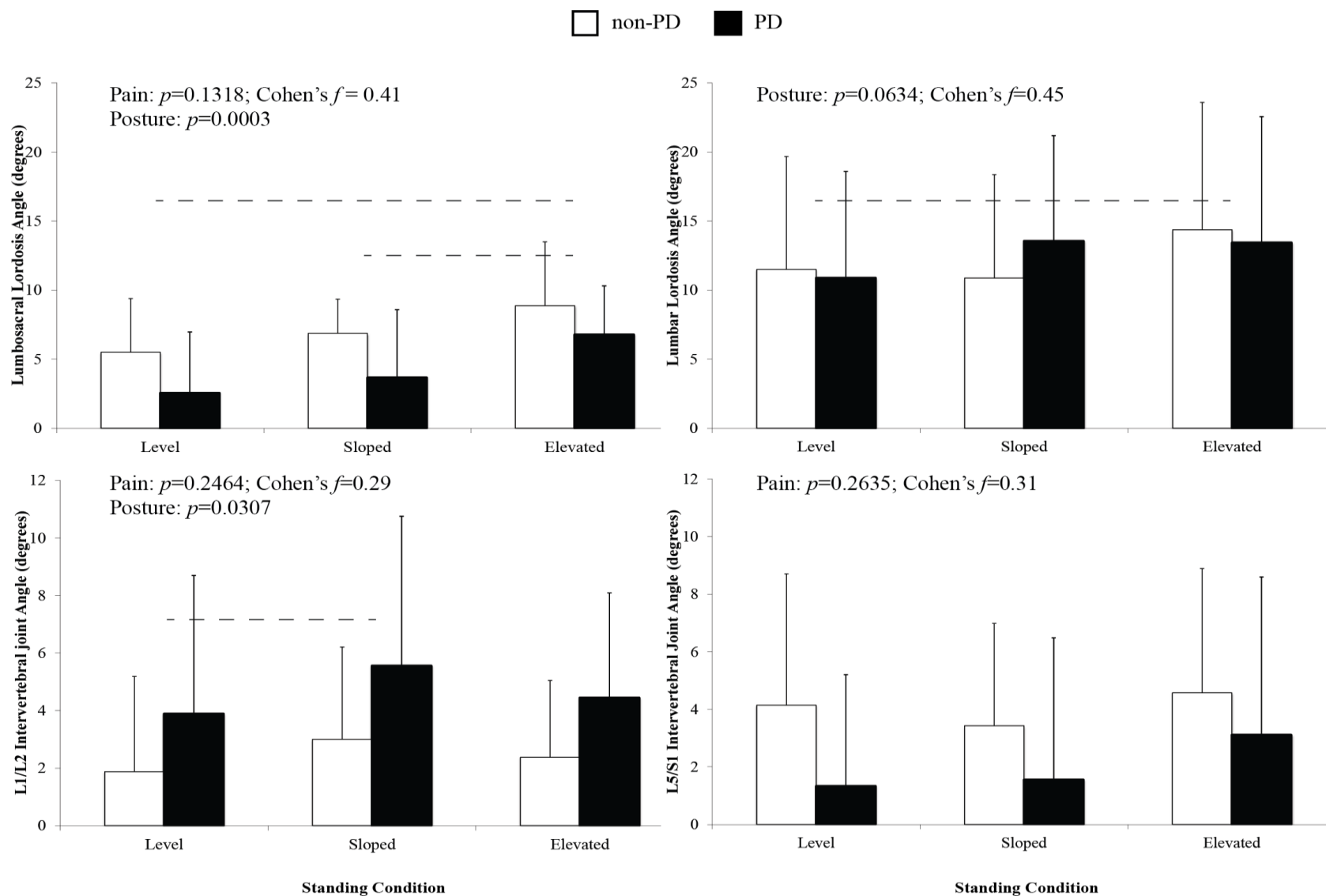
Figure 4.3. Measured internal angles from radiographs with statistic results. The dotted lines represent the significant differences between the two connected standing postures when there was a main effect of posture.

4.3.3 Repeatability of Lumbosacral Lordosis Measurement

There was no significant difference between the two lumbosacral lordosis measurements (t=1.67, df=16, $p = 0.1144$). On average, the two measurements made by the rater differed by 0.76 degrees +/- 1.89 degrees, with an absolute difference of 1.7 degrees +/- 1.05 degrees (Table 4.3). There was no correlation between the mean and the difference between the two measurements (Pearson's Correlation Coefficient = 0.13285; $p = 0.6112$); therefore, the lack of agreement was summarized by the "limits of agreement" (mean +/- two standard deviations). The upper and lower limits were 4.54 and -3.01 degrees, respectively. We can assume that 95% of the differences will lie between these two limits. It is accepted that there will be discrepancies in scores on two different occasions of up to 4.54 degrees (Figure 4.4).

Table 4.3. Comparison of two lumbosacral lordosis measurements taken on the level ground standing radiographs.

Participant	Measure 1 (deg)	Measure 2 (deg)
1	141	138
2	143	141
3	142	142
4	146	147
5	132	129
6	154	152
7	133	134
8	137	139
9	149	149
10	127	129
11	140	138
12	138	134
13	147	146
14	141	140
15	139	137
16	145	144
17	134	136
Mean (standard deviation) (deg)	140.5 (6.8)	139.7 (6.5)
Difference (deg)	0.76 (1.89)	
Absolute Difference (deg)	1.71 (1.05)	
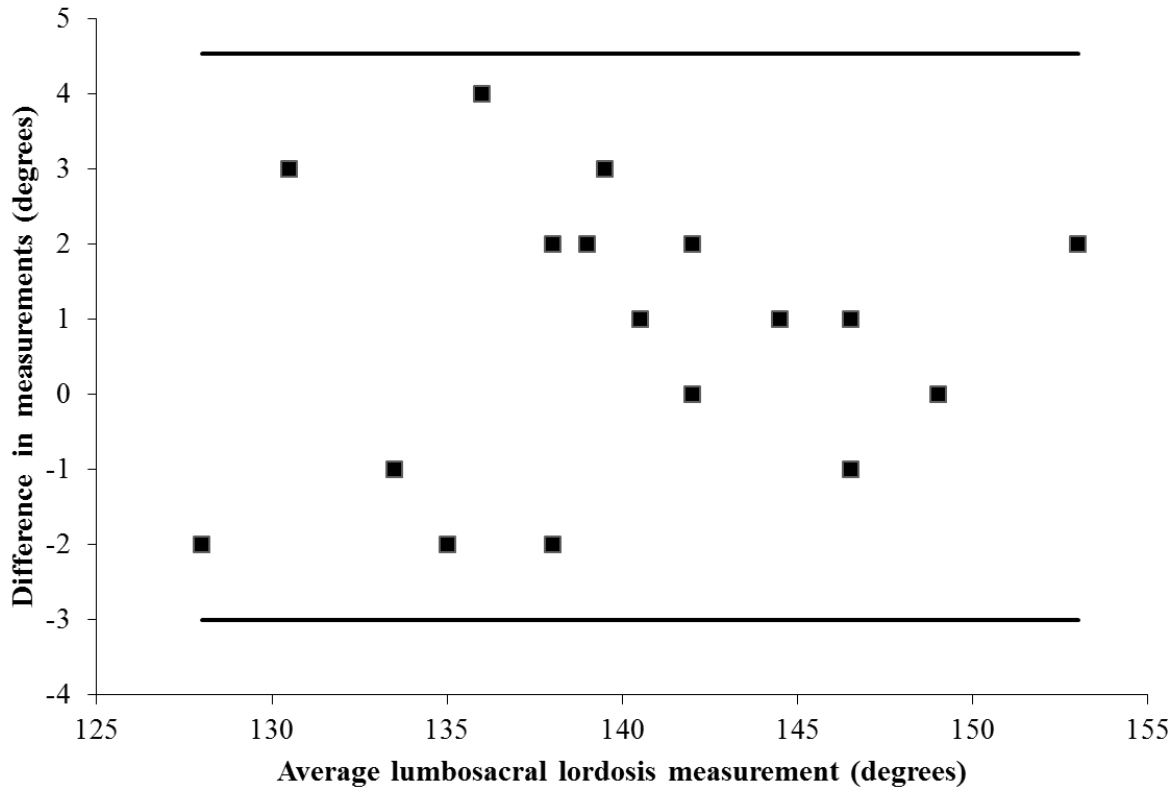
Figure 4.4. Bland-Altman analysis of two lumbosacral lordosis measures of 17 participants when standing on level ground. There was no correlation between the average lumbosacral lordosis measurement and the difference between the two measurements. The solid horizontal black lines represent the upper (4.54 degrees) and lower (-3.01 degrees) limits of agreement (mean +/- 2SD).

4.4   Discussion

The purpose of this study was to examine the sagittal lumbopelvic postural differences between low back pain and non-pain developers during prolonged standing. The lumbar spine posture of non-pain developers during level ground standing was characterized by greater lumbosacral lordosis and L5/S1 intervertebral joint flexion, as well as less L1/2 intervertebral joint flexion than pain developers. A second purpose was to evaluate the influence of standing aids that change lower limb posture on these same measures. Both the elevated and declining surfaces induced lumbosacral lordosis, lumbar lordosis, and L1/2 flexion. The elevated surface affected these variables more than the sloped surface. The sloped surface was more effective at altering the upper lumbar arc than the elevated surface.

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While lumbar lordosis angle was not significantly different between pain developers and non-pain developers, there was a difference between the upper and lower lumbar arcs. Our results follow the previous research showing the most variability between groups is in the lower lumbar lordosis angle (Mitchell et al., 2008; Roussouly et al., 2005), with pain developers standing closer to their maximum lumbosacral lordosis extension angle. The combination of this with previous evidence that pain developers stand with greater thoracic extension than non-pain developers (Gallagher et al., 2014) means that pain developers may stand with a sway back posture, potentially putting additional strain on the posterior tissues of the spine. Pain developers also demonstrated an altered muscle recruitment strategy during flexion-extension movements compared to non-pain developers (Nelson-Wong et al., 2012). When performing this movement, the non-pain developers group performed the typical muscle activation order – a bottom up strategy where extension of the trunk from flexion is dominated by hip movement. Pain developers demonstrated top-down (spine dominant) muscle recruitment, with the lumbar extensors being activated before the gluteal muscles, also seen in clinical low back pain populations (Nelson-Wong et al., 2012). Pain developers may be altering their posture from the top down, which is why there were differences in L1/2 posture, and non-pain developers from the hips resulting in an altered lower lumbar arc posture. The alterations of both these groups would then explain why no differences in lumbar lordosis angle were found.

Greater lumbar spine flexion, initiated by a change in lower limb position, was common to both standing aids hypothesized to lower low back pain during prolonged standing. The changes in postures were above what has been shown to be the biological variability of a level ground standing posture, which when measured using motion capture is approximately two degrees within a session (Dunk et al., 2005). It was fitting that LSL angle differs between the two groups since both standing aids work from the bottom up by changing foot position and affecting pelvis angle, and in turn lumbar angle. This aligns with previous work that has shown sitting (inducing lumbar flexion) for 15 minutes reduced self-reported low back pain reports in pain developers (Gallagher et al., 2014). Of the aids we tested, raising one leg onto a

platform so that the trunk to thigh angle was 135 degrees was most effective at changing lumbosacral angle, while the sloped platform used a different mechanism to change lumbar spine angle, such that it affected the L1/L2 intervertebral joint more than the elevated surface did compared to level ground standing.

This study also provides insight into potential mechanisms behind successful interventions. An exercise intervention that concentrated on promoting stabilization of hip and trunk proposed to reduce low back pain development during prolonged standing musculature showed less pain development in those previously diagnosed as pain developers (Nelson-Wong and Callaghan, 2010a). Unfortunately, lumbar spine angle was not assessed in that study; however, training aimed at changing lumbar spine angle in hyperlordotic individuals to lessen lumbar lordosis angle was successful at doing so (Scannell and McGill, 2003). While pain development was not assessed in that study, the training could have the potential to reduce passive strain on the lumbar spine (Scannell and McGill, 2003). It is possible that the training program used by Nelson-Wong and Callaghan (2010a) was also successful at changing the lumbar lordosis angle during level ground standing. Also, Gallagher and colleagues (2014) demonstrated that sitting for 15 minutes, which induced a large change in lumbar spine angle towards flexion, was also positive at reducing pain development. These studies taken collectively indicate that movement away from maximum extension angle was positive at reducing self-reported reports of low back pain during standing.

The results also show why differences in movement patterns of pain and non-pain developers would also be beneficial for reducing pain development. Two prior studies have shown that pain developers demonstrate less flexion/extension movement of their lumbar spine during standing (Gallagher et al., 2014). Cycling through changes in lower limb posture would in theory promote movement of the lumbar spine. This was accomplished when moving between dorsi and plantar flexion when using a sloped surface (Nelson-Wong and Callaghan, 2010b). While standing with one leg elevated could be effective, it can also induce discomfort in the supporting leg and requires frequent changes in posture to

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prevent standing in this position for a long period of time (Whistance et al., 1995). The combination of the altered posture from level ground standing and the initiation of movement could be the potential reason these postures reduce low back pain during standing.

Based on the characteristics of pain developers versus non-pain developers during level ground standing and potential ways to lower pain development by inducing flexion of the lumbar spine, extension aggravated tissues could be a cause of the pain development seen in prolonged standing. These postures may result in a mechanical insult or stimulus of the nociceptors located within the facet capsules (Adams and Hutton, 1980; Dunlop et al., 1984; Yang and King, 1984) , superficial annulus (Bogduk et al., 1981), dorsal root ganglion (Cavanaugh, 1995), or contact of adjacent spinous processes and impingement of tissues between (Adams et al., 1988), ultimately causing pain (Winkelstein and DeLeo, 2004). Low levels of mechanical compression result in transient pain that disappears quickly, while higher levels of loading may initiate mechanisms that will eventually result in sustained or chronic pain (Hubbard et al., 2008). Scannell and colleagues (2003) showed that people who demonstrated hyperlordosis had a lumbar spine angle that was beyond their measured neutral zone extension limit while they were standing and as a result were standing in a position described as elastic extension. Those who were controls or had hyperlordosis stood within their lumbar spine neutral zone. As a result, it is possible that pain developers stand in a position that puts excessive strain on their lumbar spine passive tissues, causing a mechanical insult that is leading to this transient pain. By using a standing aid, it may be possible to lessen the strain on the passive tissues by bringing pain developers into mild flexion during prolonged standing removing the joint positions possibly triggering the pain development.

A small sample size was collected for this study because of the associated exposure to ionizing radiation. This limited our ability to make conclusions between the pain and non-pain developers; however, the effect sizes for lumbosacral lordosis and the intervertebral joints strongly suggest that while the results were not significantly different, they may still be of clinical significance and are worth investigating further with larger sample sizes during *in vivo* data collections (see sample size calculations

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in Table 4.2). Secondly, radiographs only provide a "snapshot" of the participant in a posture that mimics their working posture from the prolonged standing study. Due to time restrictions at the imaging facility, participants could only stand for a few seconds before the image was taken. As a result we could not evaluate their posture after a longer period of time before the image was taken, limiting our ability to characterize the actual "working posture".

4.5   Conclusions

People who develop pain during prolonged standing have a sagittal lumbar spine angle that is characterized by lumbosacral lordosis and L5/S1 intervertebral joint angles that are closer to their maximum extension angles for each measurement. This could lead to aggravation of the posterior elements of the lumbar spine and could be the source of pain development during standing. Standing aids recommended for use during jobs that require prolonged standing affect sagittal posture by causing flexion of the lower arc of the lumbar spine, with the elevated standing aid being the most influential. This change, which is in the same area of the lumbar spine that differences are seen between pain and non-pain developers, may be the reason these aids are successful.

5 Study #3: Prolonged standing induced low back pain is linked to extended lumbar spine postures: A study assessing lumbar spine passive stiffness

5.1 Introduction

Over 40% of people who have never suffered a low back injury report clinical levels of low back pain during an occupational prolonged standing simulation (Gallagher et al., 2014; Marshall et al., 2011; Nelson-Wong and Callaghan, 2010c). Altering posture through sitting (Gallagher et al., 2014) or the use of standing aids (Nelson-Wong and Callaghan, 2010b) has successfully reduced prolonged standing induced low back pain; therefore, lumbar spine posture in standing may be an important predisposing factor for transient pain development. While it is common to assess postural characteristics that may predispose a person to low back pain using x-ray (Roussouly et al., 2005) and external measurements (Mitchell et al., 2008) often valuable information is overlooked regarding the intrinsic characteristics of lumbar spine, such as lumbar spine stiffness. Pain development during standing is a positive predictor for future chronic low back pain requiring medical care (Nelson-Wong and Callaghan, 2014) and the cumulative exposure of these passive tissues could be a potential mechanism for injury. Given the limited information provided by internal and external measurements of the lumbar spine, this study assessed the relationship between the *in vivo* lumbar spine lumped passive properties and the self-selected lumbar spine angle of prolonged standing induced low back pain developers and non-pain developers in four standing postures to determine the relationship between lumbar spine posture and passive stiffness on prolonged standing induced low back pain.

Passive intervertebral joint range of motion can be split into a neutral zone and elastic zone. The neutral zone is the portion of the physiological range of motion where spinal motion is produced with minimal resistance (Panjabi, 1992b). *In vivo,* the neutral zone is the range where the lumbar spine demonstrates the least amount of lumped passive tissue stiffness (Scannell and McGill, 2003). Aberrant neutral zone characteristics have been associated with injury (Panjabi, 1992b). The EZ is measured from the end of the neutral zone up to the physiological limit and is a region of high stiffness (Panjabi, 1992b).

In vitro replicated joint positions that mimic the lumbar curve during standing are associated with increased facet loading and contact, soft tissue impingement, and stretching of the facet joint capsule (Adams and Hutton, 1985; Dunlop et al., 1984; Yang and King, 1984) and account for the increased stiffness seen in the EZ. By characterizing the lumbar spine neutral zone *in vivo*, the location of a person's typical lumbar spine standing posture with respect to their passive stiffness curve and the likelihood of passive tissue involvement can be assessed. For example, people with hyperlordotic spines stood with a lumbar lordosis angle that was beyond their neutral zone extension limit, while people with hypolordosis stood within their lumbar spine neutral zone (Scannell and McGill, 2003). Standing with a lumbar curve beyond the extension neutral zone boundary could mean the person is in a posture that requires elevated muscular involvement to maintain posture and could promote a mechanical insult to the passive tissues, which is a mechanism that has previously been shown to trigger transient pain (Winkelstein and DeLeo, 2004).

A variety of interventions are recommended to help people tolerate prolonged standing. The most commonly recommended aid is placing one foot on an elevated surface (Canadian Center of Occupational Health and Safety (CCHOS), 2008; Cohen et al., 1997; Occupational Safety & Health Administration (OSHA), 2012). This position causes flattening of the lumbar spine (Dolan et al., 1988), with the greatest effect on the lower lumbar arc (Study #2, Section 4.3.2). The sloped platform has been shown to lower pain development by 59.4% in those categorized as pain developers during level ground standing (Nelson-Wong and Callaghan, 2010b). The explanation for how this is possible is mixed, as some studies have reported greater trunk flexion, posterior rotation of the pelvis (Nelson-Wong and Callaghan, 2010b), and flattening of the lumbar spine (Gallagher et al., 2013); while a recent radiographic investigation only found a difference in the L1/L2 intervertebral joint angle caused by standing on a declined work surface (Section 4). A second prolonged standing study found that hip flexion, not lumbar spine flexion, was induced by this standing aid (Study #4, Section 6.3.3). Lastly, scientists recommended that people should shift their body weight in the anterior-posterior direction by placing one foot a short step in front of the

other and sway their entire trunk gently forward until their body weight rested on the ball of the forward foot (Zacharkow, 1988). To change the weight bearing foot, you alternate the forward foot. The benefit of changing weight transfer to this anterior posterior pattern is that it may induce small flexion-extension movements of the lumbar spine, which have been shown to be greater in non-pain developers than pain developers during the first 15 minutes of prolonged standing (Study #1, Section 3.3.2).

The purpose of this study was to determine if the lumbar spine angle of prolonged standing pain developers during standing was at a location of the lumbar spine passive stiffness curve that could provide insight into the pain mechanism underlying low back pain development during standing. A secondary purpose was to determine if commonly recommended alternate foot intervention strategies move people into a posture that could positively change the location of the lumbar spine within their lumbar spine passive stiffness profile. We hypothesized that (1) the lumbar spine posture of non-pain developers will be located within the lumbar spine passive neutral zone, while the posture of pain developers will be located outside the extension limit of the neutral zone, and (2) the use of standing aids would move all participants into lumbar flexion, and for pain developers, to stand with their lumbar spine within their passive neutral zone. Participants previously classified as pain developers and non-pain developers stood in four postures for 5 minutes – level ground standing and three alternate foot placement positions. Following this, their lumbar spine passive stiffness curve was quantified using a near-frictionless jig and the location of the lumbar spine neutral zone was compared to the lumbar spine postures assumed in the four standing positions.

5.2   Methods

5.2.1   Participants

Thirty-one participants (13 male; 18 female) between the ages of 18-35 were recruited to participate in this study. All participants had previously participated in either a 75 (Section 6.2.1, Study #4) or 120-minute (Section 3.2.1, Study #1) simulated occupational prolonged standing simulation where they were

categorized as either pain developers or non-pain developers based on their self-reported visual analog scale scores. To qualify for these prolonged studies, participants could not have had any previous history of low back pain that required medical intervention or time off from work/school longer than three days related to low back pain, previous lumbar or hip surgery, employment in a task that required prolonged static standing during the past 12 months, and any self-declared inability to stand for at least 2-hrs. Before entering this study, participants were asked again if they fit any of the exclusion criteria to determine if their participation status had changed since they last participated, which could have impacted their pain group classification. The University of Waterloo Research Ethics Committee approved this study and all participants provided written informed consent prior to starting the study.

5.2.2   Instrumentation

Electromyography was used to track the muscle activity that occurred during the passive flexion and extension trials to ensure that these trials were indeed passive and not influenced by active muscular resistance. The skin above the bilateral thoracic erector spinae (5 cm lateral to ninth thoracic spinous process), lumbar erector spine (above and below the third lumbar spinous process), and internal obliques (1 cm media to anterior superior iliac spine beneath a line joining bilateral anterior superior iliac spines) were prepared for electrode application by shaving and abrasion of the skin with rubbing alcohol. Two disposable, pre-gelled silver-silver chloride electrodes (Blue Sensor, Ambu A/S, Denmark) were placed on each muscle with a 2-cm inter-electrode distance. A system reference electrode was placed over the seventh cervical vertebrae. Maximum voluntary contractions were collected from each muscle for normalization purposes. For the thoracic and lumbar erector spinae, participants lay prone on a table with their torso hanging off the edge of the table at the level of their anterior superior iliac spine. Participants crossed their arms over their chest, bent their torso towards the ground as a starting position and then extended their trunk to meet resistance applied by the experimenter. For the internal obliques, participants lay supine on a table in the sit up position and performed right and left rotation against resistance. A ten second rest trials were taken in both the prone and supine positions.

An Optotrak Certus motion capture system (Northern Digital Inc., Waterloo, ON, sampling at 32 Hz for standing, 128 Hz for passive jig) was used to track movement of infrared markers during data collection for both the standing trials and the passive spine stiffness trials. Three rigid bodies were used to track movement of the spine –at the level of T9 (thoracic spine), at the level of L1/L2 (upper lumbar spine), and the sacrum. Anatomical landmarks were captured while the participant stood in a neutral posture and a digitizing probe with a rigid body containing four markers and a known location of a point at the end of the probe with respect to the origin of the rigid body was placed on the anatomical landmark of interest and the location of the tip was captured in the global system and transformed into the rigid body of interest. For the remainder of the collection protocol this point had a fixed relationship to the origin of the rigid body.

A custom built frictionless jig (Figure 5.1) was used to measure lumbar spine passive stiffness. This jig allows for the moment-angle relationship of the lumbar spine to be isolated while the legs and pelvis are in a fixed position and the upper body is moved through flexion and extension. The jig is composed of three components:

1. Nylon ball bearings (1.2 cm diameter) evenly distributed over a Plexiglas surface (1.22 m x 1.83 m x 2.54 cm)

2. Upper body wooden cradle lined with Plexiglas on the inferior surface, which glides over the ball bearings and a lower body support that restricts motion at the hip and is vertically adjustable.

3. A force transducer placed in series with a cable to pull participants into flexion and extension, a metal rod fixed to the point of application of the applied force, and a parallel cable to ensure that applied forces are perpendicular to the thoracic harness.
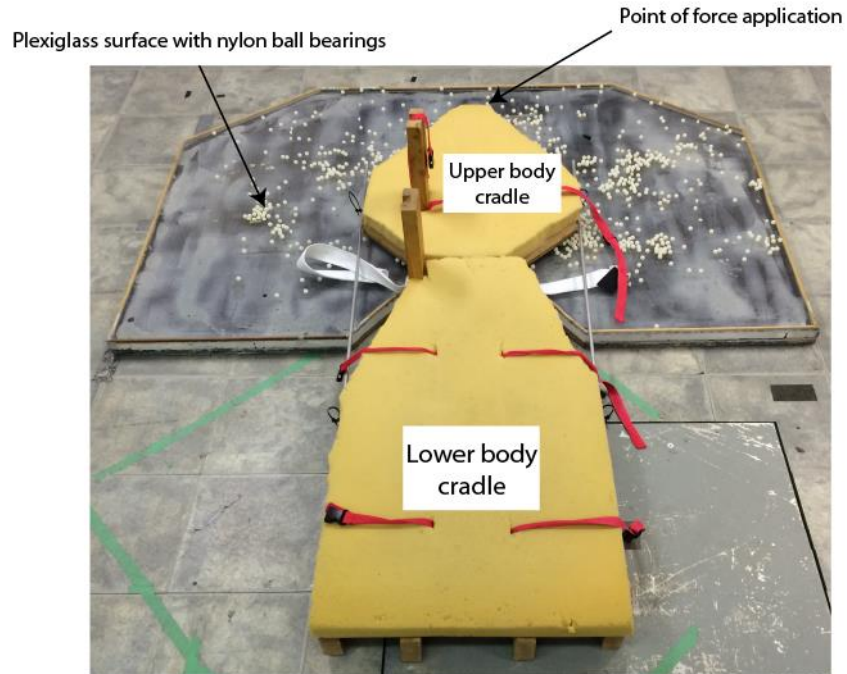
Figure 5.1. Frictionless jig used to assess passive stiffness of the lumbar spine. Initially, the upper and lower body cradles are secured together using rods on the sides of each cradle. The upper body cradle can be adjusted horizontally for each participant so that his or her lumbar spine is isolated between the upper and lower body cradle.

5.2.3 Standing Conditions and Workstation Set Up

Four standing positions were assessed in this study (Figure 5.2):

1. Standing with both feet on level ground.

2. Standing with one foot raised on a platform to represent the most likely posture when using this standing aid. The height of the platform adjusted so that a participant's thigh to trunk angle was 135 degrees. This posture is described as the physiological normal for the lumbar curve compared to erect standing where some curvature of the lumbar spine is still maintained (Keegan, 1953). Participants choose which leg they would prefer to stand on for the 5-minute trials.

3. Standing on a sloped surface with a 16-degree decline (toes down).

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4. Staggered Standing. The foot that was placed on the elevated surface was the same one that was the front foot during the staggered standing trials.
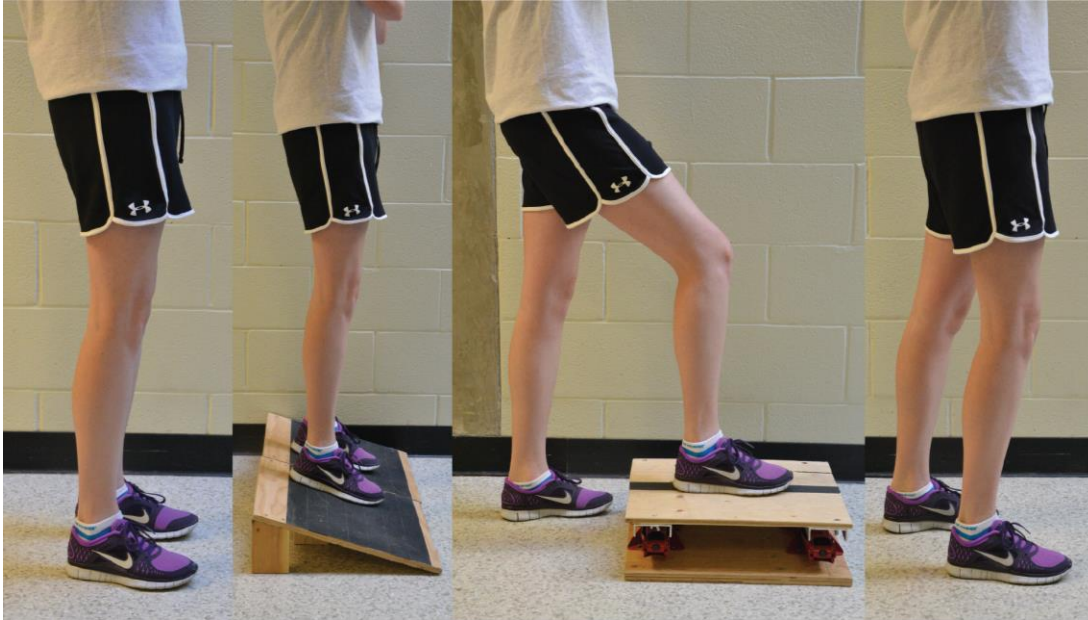
Figure 5.2. Four standing conditions tested in Study #3 (left to right): level ground, sloped, elevated, and staggered

Participants worked at a computer performing a standardized typing task while standing. The custom computer program had a window with a paragraph of text that they needed to duplicate by typing it into a second window below the source document. Participants stood in front of a table adjusted to 5-6 cm below their wrist when their elbows were placed at 90 degrees, which is the position recommended for light work (Kroemer and Grandjean, 1997). For the sloped surface, the height of the table needed to be higher to accommodate standing on the surface.

5.2.4 Experimental Protocol

5.2.4.1 Standing Protocol

Once instrumented, a 5-second standing trial with the participant in anatomical position was recorded to characterize the motion capture set up. All participants were exposed to level ground standing as their

first condition. The remaining three standing conditions were then randomized for each participant. The participant stood for 5-minutes and then sat for 1-minute between the trials to minimize low back pain development. After the entire standing protocol was completed, the participant sat for 5-minutes to prevent any carryover affects from the standing before being placed in the frictionless jig. The frictionless jig protocol was also done after the standing so that the passive flexion and extension postures did not induce any changes in a person's lumbar spine posture during standing.

5.2.4.2 *Frictionless Jig Protocol*

The participant laid on their right side with their anterior superior iliac spine aligned with a vertical column on the lower body support and shoulders with the vertical column on the upper body support. Straps secured the ankles, thighs, and pelvis to the lower body platform. A pillow was placed under the participant's head to keep the entire spine in horizontal alignment. The torso was strapped to the upper body cradle so that when the platform is moved with respect to the lower body platform, motion only occurred about the lumbar spine. To minimize trunk axial rotation and limit movement between the upper body and the cradle, the participant folded their arms around the vertical column.

Both lumbar spine extension and flexion were measured in this study (Figure 5.3), with extension always being measured first because it was more likely that participants would stand closer or beyond the extension limit of the neutral zone and this prevented important tissues involved in resisting lumbar spine extension from being stretched during passive flexion. Each passive trial began with the upper body cradle moved into a lumbar spine rotated position away from the intended movement direction. For the extension trials, the participant began in mild flexion, and vice versa for flexion trials. The trials were performed to a metronome to ensure that movement was performed at a constant speed. The participant was pulled into extension or flexion by the experimenter and the trial was stopped just before the experimenter felt the participant was at their maximum range of motion requiring an increase in applied force or if the participant reported that they could not move any further. Maximum range of motion was not assessed in this study to prevent unnecessary stretch of the lumbar spine passive tissues. Since the use

of the measurement approach assumes muscle activity is negligible in resisting the applied moment, a trial was confirmed to be passive if muscle activity of the erector spinae and abdominal wall remained less than 5% MVC. The muscle activity was checked immediately after each trial to ensure the collection of three successful trials in each direction. Three successful trials were collected for each participant and a maximum of four trials in each direction were collected to minimize passive stretching of the lumbar spine tissues.
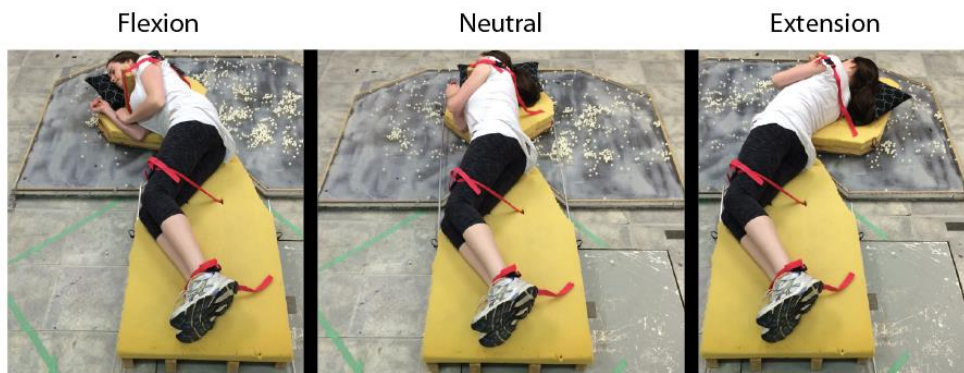
Figure 5.3. Example of frictionless jig positions. The neutral position (center) was used to secure the participant into the jig. Following this the two bars attaching the cradles together are removed so that the upper platform could move freely over the Plexiglas surface. The extension trials (right) were collected first, followed by the flexion trials (left). For each direction, the upper body cradle start position was moved in the opposite direction to provide a "buffer". For extension the participant's start position was in mild lumbar flexion compared to neutral and vice versa for flexion.

5.2.5 Data Analysis

Motion capture data were imported into Visual3D (Version 4, C-Motion, Inc. Germantown, MS, USA) for post-processing. If marker interpolation was required, a third order polynomial was used to fill gaps of a maximum 16 frames (0.5 seconds). Once interpolated, the signal was low pass filtered (Butterworth, 2nd order, dual pass) with an effective cut-off frequency of 4 Hz, as determined using a residual analysis (Study #1, Section 3.2.5.3). Coordinate systems were constructed using the landmarks for each segment with +y pointing proximally, +x pointing anteriorly, and +z pointing laterally in the same manner as for

Study #1 (Section 3.2.5.3). Lumbar spine angle was calculated using the joint coordinate system and flexion/extension-lateral bend-axial twist rotation sequence. The 50th percentile sagittal lumbar spine angle for each 5-minute standing condition was determined from an amplitude probability distribution function (APDF).

The first passive trial deemed acceptable in both flexion and extension was used to characterize the neutral zone. A moment-angle relationship (Figure 5.4, top row) was created by multiplying the force from the force transducer by a moment arm measured by placing an infrared marker at the point of force and the L4/L5 joint (level of the ASIS). The curve was fit with a fourth order polynomial and differentiated to provide a stiffness estimate (Figure 5.4, bottom row). Previous studies have used exponential (Parkinson et al., 2004) and sixth order polynomial (Beach et al., 2005; De Carvalho and Callaghan, 2011) to fit the passive curves; however, these studies did not characterize the neutral zone. Inspection of the resulting curves using these methods showed that they were very poor at representing the initial toe region of the curve, which was of main interest in this study. A fourth order polynomial has been used previously to separately fit flexion and extension moment-angle curves in *in vitro* work that has assessed neutral zone boundaries of individual intervertebral joints (Thompson et al., 2003). The boundary of the neutral zone was defined as the point where the slope of the curve was less than 0.1 Nm/degree and the change in the moment over the section was less than 7 Nm (Scannell and McGill, 2003). Flexion and extension boundaries were defined to give the neutral zone range in degrees.

The corresponding moments for the neutral zone boundaries and the lumbar spine angle during level ground standing were extracted for comparison. The median lumbar spine angles from the four standing trials were expressed with respect to the extension neutral zone boundary (in degrees) and also normalized to the length of the neutral zone to account for inter-participant variability in magnitude of the neutral zone.

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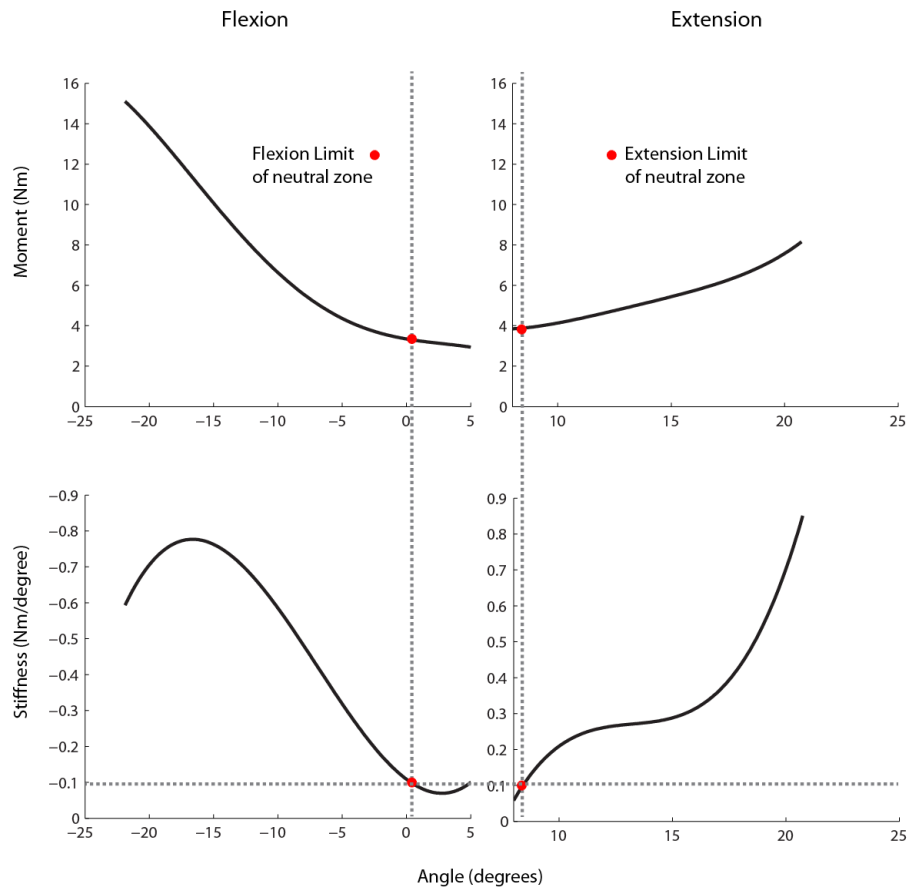
Figure 5.4. Sample data analysis for the neutral zone boundaries. The moment-angle curves (top row) were fit with a fourth order polynomial and differentiated to provide an estimate of lumbar spine passive stiffness (bottom row). Stiffness cut off of 0.1 Nm/degree was used to define the angle boundaries of the neutral zone in both flexion and extension. In the above example, the neutral zone boundaries for flexion and extension were 0.42 and 8.35 degrees, respectively (range = 7.93 degrees). The flexion and extension curves are not continuous on the x-axis (angle) because each direction was measured in a separate trial.

5.2.6   Statistical Analyses

To compare the lumbar spine posture in each standing condition with respect to the extension neutral zone

boundary, the raw relative angle and the ratio of the distance divided by the neutral zone length were

entered into a three-way general linear model with between factors of gender and pain group and within

factor of standing condition. The neutral zone length and corresponding neutral zone boundary moments

from the passive test were entered into a two-way general linear model with between factors of gender

and pain group. The corresponding moment to the lumbar spine angle during level ground standing was

compared using an independent t-test. For all tests, the alpha level of significance was set at alpha <0.05. A Cohen's *f* statistic (Ellis, 2010) was calculated from the effect and error Sum of Squares using Equation 4.1. If the Cohen's f statistic was medium or large (suggesting that despite lack of significance there may be a clinical meaning to the difference in this measure), the variable was further assessed using a one-tailed non-parametric Wilcoxon scores test, with the *p* value adjusted accordingly to account for multiple comparisons using a Bonferonni correction.

5.3   Results

This study began with 31 participants; however, nine were excluded from the passive jig analysis. For three participants the pelvis pulled away from the vertical column of the lower body cradle immediately at the start of the extension trials, one participant exceeded the size of the jig, three were not able to relax their muscles enough to meet the 5% MVC criteria, and two had kinematic errors that prevented further processing of the trials. In total, 22 participants were analyzed for this study – 11 non-pain developers (7 females, 4 males) and 12 pain developers (5 females, 7 males). Best-fit coefficients and root mean square error are reported in Table 5.1.

Table 5.1. Best fit coefficients, r^2, and root mean square error (RMSE, N m) for a fourth order polynomial (Moment = ax^4 + bx^3 + cx^2 + dx + e, where x = the corresponding angle in degrees) for flexion and extension.

	Flexion	Extension
a	-0.0007 (0.00187)	-0.0003 (0.00357)
b	0.0387 (0.1516)	0.0540 (0.248)
c	-1.13 (4.63)	-2.26 (7.60)
d	13.38 (62.06)	36.40 (108.6)
e	-64.3 (304.12)	-206.63 (594.7)
r^2	0.51 (0.22)	0.40 (0.15)
RMSE	1.65 (0.23)	1.76 (0.41)

For neutral zone length, there was a significant interaction between pain group and gender (p=0.0466, df=1, F=4.57). There was an opposite trend for what the pain groups demonstrated within each gender (Figure 5.5). For males, pain developers had a greater neutral zone length than non-pain

developers and vice versa for females. The neutral zone length for the female non-pain developers was similar to that of the male pain developers (between 10 and 11 degrees), while the male non-pain developers and female pain developers had similar neutral zone lengths (between 6 and 7 degrees).
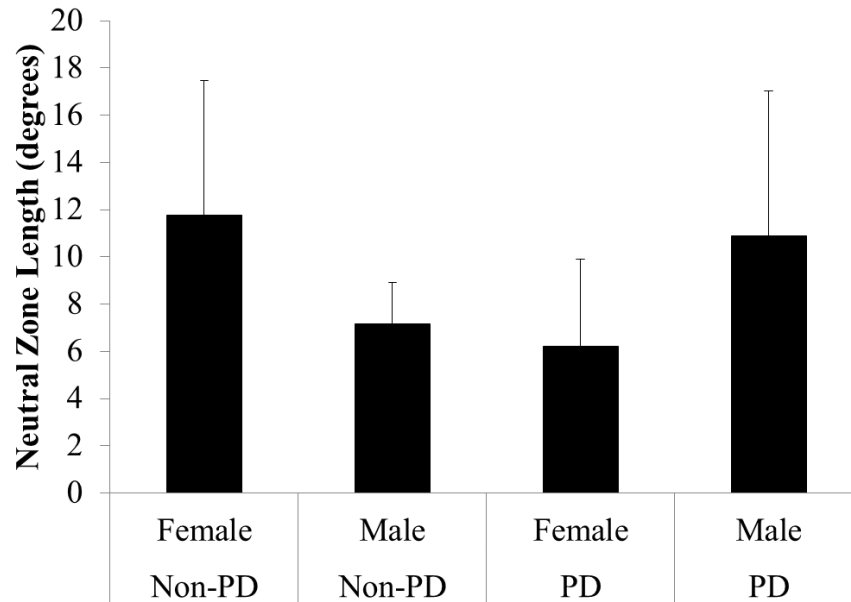
Figure 5.5. Neutral zone lengths separated by pain group and gender. There was a significant interaction between gender and pain group. For non-pain developers, the neutral zone length for females was greater than males, but the opposite trend was found for pain developers.

For the corresponding extension and flexion moments to the neutral zone boundaries (Figure 5.6), there were significant main effects of pain group (Ext: $p=0.0175$, df=1, F=6.85; Flex: not significant) and gender (Ext: $p=0.0002$, df=1, F=21.90; Flex: p=0.0021, df=1, F=12.85), but no interactions. For the extension moment, non-pain developers had a higher moment (2.82 +/- 1.3 Nm) corresponding to the location of their neutral zone boundary than pain developers (2.1 +/- 1.5 Nm). For both flexion and extension, males had a corresponding moment two times higher than females' corresponding moment.
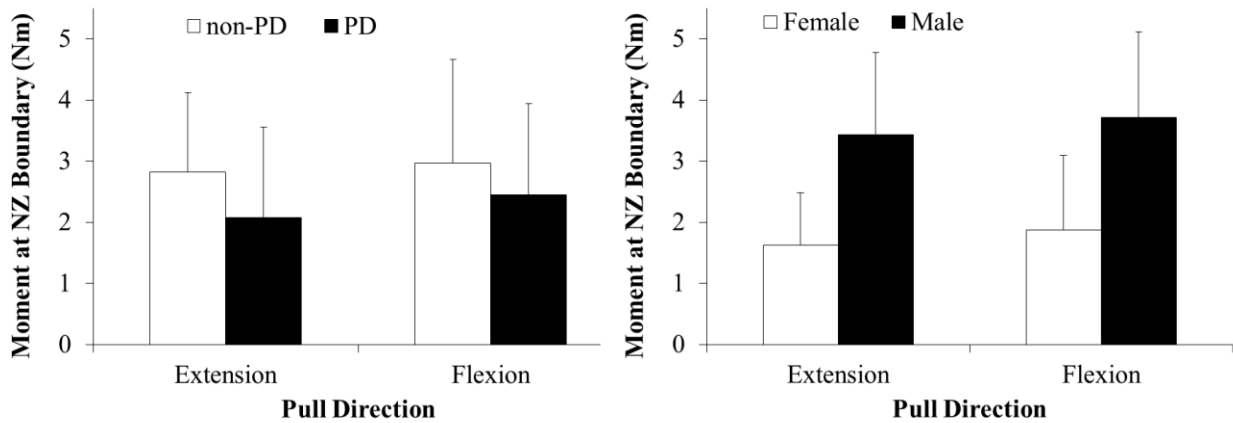
Figure 5.6. Corresponding moment to the flexion and extension neutral zone boundaries compared between pain groups (left) and gender (right). Between pain groups, the external moment was significantly greater for non-pain developers (non-PD) than pain developers (PD) for the extension limit, but not the flexion limit. Between genders, males had a higher corresponding moment than females for both the extension and flexion limits. Asterisks denote significant differences between the black and white bars.

There was a main effect of condition for the raw difference (Figure 5.7) between the standing positions and the extension neutral zone boundary ($p < 0.0001$, df=3, F=8.89), but no main effect of gender or pain group. For all pain groups, the use of the elevated surface resulted in more lumbar spine flexion ($p=<0.0013$), with non-pain developers standing on average right at the neutral zone extension limit and pain developers standing 2.2 degrees beyond the extension limit compared to level ground standing.
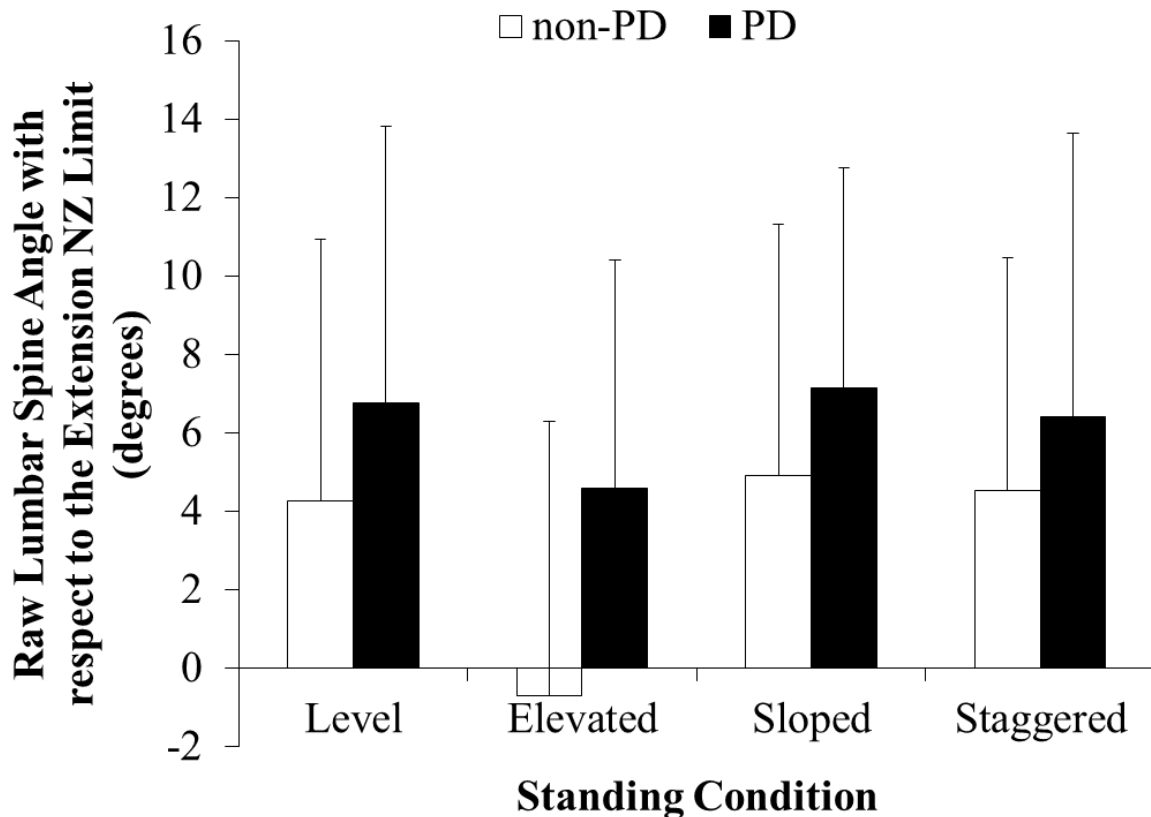
Figure 5.7. The distance (in degrees) between the lumbar spine angle in each standing condition and the calculated extension neutral zone limit. The zero value is the neutral zone extension limit with a positive number indicating that the postures were outside of the neutral zone. The smaller the value, the closer the lumbar spine angle was to the extension neutral zone limit, the larger the number the more extended and beyond the neutral zone extension limit. Only a main effect of condition was found such that the elevated surface brought the lumbar spine angle into more flexion compared to the other standing conditions.

When distance away from the NZ normalized to each individual's neutral zone length (Figure 5.8), the main effect of pain group was not significant (p=0.1089, df=1, F=2.82); however, the calculated Cohen's f statistic of 0.3752 indicated a medium effect. To be significant, future studies would need to collect a minimum of 13 participants for each pain group. There was no main effect of gender. To investigate this further, a one-tailed non-parametric paired t-test was used to assess the difference between pain developers and non-pain developers during the elevated condition because it was the condition with the greatest ratio difference (1.3 times the neutral zone length). A one-tailed test was used because it was hypothesized that non-pain developers stood closer to the neutral zone limit. There was a significant

difference between the two groups (p=0.0401, one-tailed t-test). Like the raw difference between the extension limit and lumbar angle, there was also main effect of condition (p=0.0156, df=3, F=3.74).
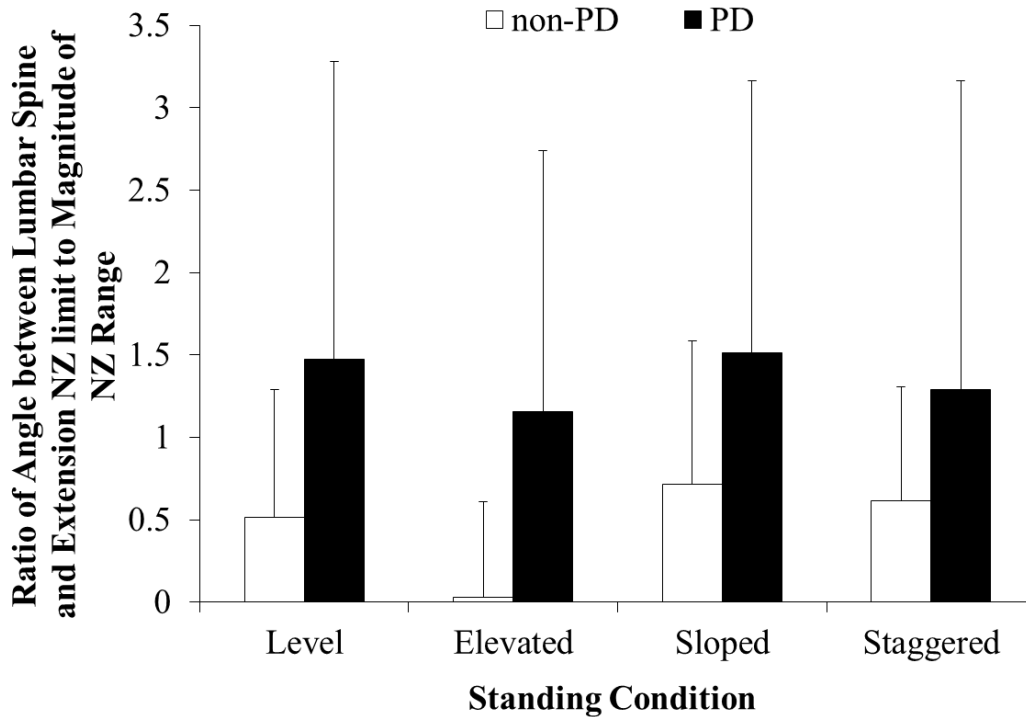
Figure 5.8. The ratio of the distance between the lumbar spine angle and the extension neutral zone (NZ) limit (from Figure 5.7) to the length of the neutral zone zone (distance/neutral zone length). A ratio of 1 means that the distance between the lumbar spine angle and the neutral zone limit is equal to the length of the neutral zone. A positive value means that the posture is outside of the neutral zone. In addition to a main effect of condition, there was a trend towards a main effect of pain group, suggesting that while on average, participants all stood outside of their passive lumbar spine neutral zone, non-pain developers stood closer to their extension neutral zone limit, or a more neutral posture, than pain developers.

There was no significant difference for the moment corresponding to the lumbar angle during level ground standing between pain and non-pain developers (p=0.8497). The average across all participants was 4.56 Nm +/- 3.47 Nm.

5.4 Discussion

The purpose of this study was to compare the lumbar spine angle during level ground an alternate standing postures to the location of the lumbar spine passive neutral zone. On average, participants stood with a lumbar spine angle outside of their passive neutral zone in extended lumbar spine postures; however, non-pain developers stood closer to their neutral zone extension limit when expressed as a function of neutral zone length. The use an elevated surface with a hip to thigh angle of 135 degrees changed the lumbar spine angle so that participants stood closer to their passive lumbar spine neutral zone.

Once normalized to neutral zone length, there was a trend towards non-pain developers standing closer to their neutral zone extension boundary than pain developers. The high variability in this data set likely prevented the significant findings; however, testing to see if the non-pain developers were closer to their neutral zone than pain developers in the elevated condition revealed significant differences. With non-pain developers standing closer to their neutral zone, two things are possible. First, they may minimize contact of the facet joints, facet joint capsule stretch, and tissue impingement between the adjacent facets and spinous processes, ultimately preventing a mechanical stimulus that could cause transient pain development (Winkelstein and DeLeo, 2004). Second, small movements within the neutral zone may transmit signal information to the central nervous system for proper functioning of the spinal stabilizing system (Panjabi, 1992b). Non-pain developers demonstrate more frequent fidgeting of their lumbar spine during standing (Study #1, Section 3.3.2; Study #4, Section 6.3.3), thus, such movements combined with a posture closer to this neutral zone may be positive for proper functioning of the lumbar spine in standing.

Since previous work has shown that participants who had hyperlordotic spines stood in a state of elastic strain compared to controls and those with hypolordotic spines (Scannell and McGill, 2003), we hypothesized that non-pain developers would stand within their neutral zone and pain developers in their extension elastic zone; however, this was not the case as all participants tended to be beyond the

extension limit of their neutral zone. The algorithm for determining the neutral zone in this study was more rigorously implemented than the Scannell and McGill (2003) study. Their study (Scanell and McGill, 2003) looked at the participant's moment-angle curves qualitatively first to segment the graphs and then calculated the stiffness post-hoc. The average stiffness of the neutral zone in that study was 0.13 Nm/degree (compared to 0.1 Nm/degree for our study), resulting in a neutral zone length of approximately 20 degrees (Scannell and McGill, 2003), 8 degrees greater than seen in our study.

It is interesting that despite standing varying distances beyond the side-lying neutral zone limit, the average moment from the passive moment-angle curve corresponding to the lumbar spine angle during level ground standing was the same. As a result, the average linear stiffness of the initial elastic zone region between the extension neutral zone limit and the level ground lumbar spine angle was greater in non-pain developers (0.44 Nm/degree) than pain developers (0.38 Nm/degree); however, it is unknown the physiological significance of this potential difference. This could correspond to greater laxity in the lumbar spine of pain developers, and may point to why they stand further beyond their neutral zone and with greater muscle co-contraction than non-pain developers. Due to higher passive stiffness, non-pain developers could also have a more robust lumbar spine, allowing their spine to withstand greater perturbations, as seen with more movement of the lumbar spine in standing (Study #1 – Chapter 3, Study #4 – Chapter 6, Gallagher et al., 2014). More research must be conducted to further assess the passive lumbar spine extension zone in order to confirm and expand on these results.

Of the three standing positions tested, only standing with one foot on an elevated surface affected lumbar spine angle compared to level ground standing. The elevated surface brought the lumbar spine angle closest to being within the neutral zone of any intervention through mild flexion of the lumbar spine. The interesting result is that while standing on a decline has positively affected low back pain scores during prolonged standing (Nelson-Wong and Callaghan, 2010b) it did alter lumbar spine posture compared to level ground. Standing on a decline surface induced hip and trunk-to-thigh flexion while the trunk, lumbar spine, and pelvis, functioned as one unit with no changes of the relative arrangement

between the segments (Study #4, Section 6.3.1). Therefore, only assessing lumbar spine angle may not provide all the information about why an aid is successful or the kinematic alterations that occur.

A limitation to this study was that quantification of the lumbar spine passive properties was done while horizontal. While lying horizontal, the neutral zone represents the passive characteristics of the lumbar spine void of compression due to body weight and muscle activity, thus, lumbar spine stiffness may be less than that seen in tasks involving high compressive loads (McGill et al., 1994). During *in vitro* testing of lumbar functional spinal units, the motion of the spinal units is stiffer when compressive pre-loads are applied (Gardner-Morse and Stokes, 2003; Janevic et al., 1991). Secondly, the passive characteristics describe the intact lumbar spine properties and do not separate out the lumbar spine passive tissues or the contributions of viscera, skin, or fat; however, the influence of these tissues is assumed to be small (McGill et al., 1994); however, these tissues will likely not influence the neutral zone location as they will be engaged close to the end range of motion, which was not of interest in this study. Finally, more valuable information may have been gained from isolating only the lower lumbar arc of the lumbar spine and not the entire lumbar spine, as this is the area shown to have the most difference between individuals (Roussouly et al., 2005) and the lumbosacral lordosis arc was shown to be different between pain developers and non-pain developers (Study #2, Section 4.3.1). However, using surface tracking of kinematics, separating the lumbar spine into these two segments would contain substantial errors in estimating the location and attempting to account for skin motion involved in the full range of postures involved

5.5 Conclusions

Pain developers stood with a lumbar spine angle further beyond their passive lumbar spine extension neutral zone limit than non-pain developers. This may put them in a posture that is more likely to sustain mechanical insults in the lumbar spine that result in transient pain development during prolonged standing. The results also point to the potential for a stiffer passive lumbar spine in non-pain developers; however, these results must be further expanded on in future work. Standing aids proposed to aid in

reducing low back pain demonstrated conflicting results - the elevated surface brought the lumbar spine angle into flexion compared to level ground standing, while the sloped surface did not. The difference in the lower lumbar lordosis angle between pain groups found in previous research suggests that future studies should concentrate on comparing the passive characteristics of the isolated lower lumbar spine between pain developers and non-pain developers.

6 Study #4: Standing on a declining sloped surface reduces transient prolonged standing induced low back pain development

6.1 Introduction

The increased use of sit-stand and standing workstations has produced products that help people tolerate longer bouts of standing to prevent potential lower limb and back pain commonly associated with standing (Waters and Dick, 2014). When it comes to preventing low back pain during standing, this is a large market audience, as greater than 40% of people who have never had a back injury report low back pain while standing (Gallagher et al., 2014; Marshall et al., 2011; Nelson-Wong and Callaghan, 2010c). The Canadian Center for Health and Occupational safety (2008), the Occupational Safety and Health Association (2012), and the National Institute of Occupational Safety and Health (1997) all recommend some sort of standing aid when prolonged or static standing is an ergonomic risk factor. Despite these recommendations, we know very little about how, why, or if these aids are successful at reducing low back pain. A working hypothesis is that when a device alters lower limb position it also alters pelvis and lumbar spine posture. A relevant example of this from sitting – decreasing the trunk to thigh angle causes the pelvis to rotate posteriorly and the lumbar spine to flatten (Keegan, 1953). While more subtle, the same mechanism is also found when changing lower limb position while standing (Dolan et al., 1988). Studying the influence of standing aids on prolonged standing induced low back pain provides insight into (1) the characteristics of successful interventions and (2) the fundamental difference between prolonged standing low back pain developers and non-pain developers. Thus, the purpose of this study was to assess the acute and prolonged kinematic differences between pain developers and non-pain developers when utilizing an intervention shown to be successful at reducing low back pain during standing – a sloped standing surface.

A sloped surface allows for workers to vary their posture by either placing their feet in dorsi- or plantar flexion (Figure 6.1). When using this aid during a 2-hour occupational standing simulation, self-

reported reports of low back pain development were 59.4% less in those previously categorized as pain

developers when standing on level ground and the scores were similar to the non-pain developers

(Nelson-Wong and Callaghan, 2010b). The success of this aid was linked back to the kinematic changes

induced by plantar and dorsiflexion of the feet. Standing on a decline resulted in greater trunk flexion,

posterior rotation of the pelvis (Nelson-Wong and Callaghan, 2010b), and flattening of the lumbar spine

(Gallagher et al., 2013). Standing on an incline surface resulted in the opposite changes, with greater

trunk extension, anterior rotation of the pelvis (Nelson-Wong and Callaghan, 2010b), and extension of the

lumbar curve(Gallagher et al., 2013) compared to level ground standing.

Figure 6.1. An example of a person standing on level ground (left) and on a 16-degree declining surface (right). To stand on an incline, the participant would face the other way so that they faced the taller side of the surface.

A limitation to the study by Nelson-Wong and Callaghan (2010b) was that participants could

move freely between standing on the decline and incline. Participants spent 72% of their time standing on

the decline, meaning that most of the time was spent in a slightly flexed posture compared to level ground standing. The ability to cycle between the two postures makes it difficult to parse out the influence of standing in a modified posture and the influence of the surface on lumbar spine posture induced by moving between decline and incline have on reducing low back pain while standing. Determining whether posture and/or movement are important factors for reducing low back pain will help to further design aids and also point to a potential fundamental difference between how pain developers and non-pain developers stand.

The purpose of this study was to evaluate the effectiveness of standing with the ankles continuously in plantar-flexion (which from here-in will be referred to as *standing on a decline or sloped surface*) on the development of prolonged standing-induced low back pain, as well as kinematics and movement variables. We hypothesized that the mild lumbar flexion induced by standing on a decline would help reduce or prevent low back pain. Secondly, we hypothesized that standing on a decline would induce postural changes of the hip angle, thigh-to-trunk angle, and location of the trunk center of gravity with respect to the ankle location. Lastly, we hypothesized that movement patterns would differ between the two groups. To evaluate these hypotheses, we used self-reported reports of low back pain and motion capture to compare pain development and kinematics between standing on level ground and a sloped surface over a 75 minute occupational simulation.

6.2 Methods

6.2.1 Participants

Seventeen participants (nine male, eight female) aged 18-35 were recruited to participate in this study (Table 6.1). Participants could not have had any previous lifetime history of low back pain that required medical intervention or time off work longer than three days, previous lumbar or hip surgery, employment in a job requiring prolonged static standing during the past 12 months, and the inability to

stand for at least two hours. The University of Waterloo Research Ethics Committee approved this study and all participants provided written informed consent prior to starting the study.

Table 6.1. Participant characteristics. Note: mean (standard deviation)

	Female (n=8)	Male (n=9)
Age (years)	22.6 (1.5)	23.3 (2.6)
Height (cm)	163.8 (4.8)	178.7 (4.5)
Mass (kg)	62.8 (6.6)	84.6 (12.7)

6.2.2 Instrumentation

An Optotrak Certus motion capture system (NDI Inc., Waterloo, ON, sampling at 32 Hz) was used to track movement of the infrared markers during data collection. Three rigid bodies were used to track movement of the spine – one at T9 (thoracic spine), L1/L2 (upper lumbar spine), and the sacrum. Movement of the feet and thighs were also tracked bilaterally with rigid bodies. Anatomical landmarks were captured while the participant stood in a neutral posture and a digitizing probe with a rigid body containing four markers and a known location of a point at the end of the probe was placed on the anatomical landmark of interest. The location of the tip was captured in the global system and transformed into the rigid body used for tracking the corresponding segment. For the remainder of the collection protocol this point was assumed to have a fixed or non-varying relationship to the origin of the rigid body.

A 100 mm visual analogue scale was used to assess low back pain development. A horizontal line was anchored at the left with "No Pain" and the right with "Worst Pain Imaginable" and participants were asked to place a vertical line to mark their CURRENT level of low back pain. This has been previously used to identify pain developers during prolonged standing (Gallagher et al., 2014; Marshall et al., 2011; Nelson-Wong and Callaghan, 2010a). Participants filled out a visual analogue scale when they entered the lab, immediately after instrumentation was complete, and during the standing protocol.

6.2.3 Standing Conditions

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Two standing conditions were tested in this study – level ground and sloped. During level ground standing, participants stood as they normally would on a flat surface. For the sloped surface, participants stood on a declining 16 degree angled wooden slope. For both conditions participants were told to: *"Stand as you normally would if you were required to work at a computer workstation. You can lift up each foot, change your foot position, and shift your weight back and forth. The two things you cannot do are support your body weight on the table and cross your feet".*

6.2.4   Workstation Setup

Participants worked at a computer performing a standardized typing task. The custom computer program had a window with a paragraph of text that they needed to duplicate by typing it into a second window below the source document. The table and monitor heights were separately adjusted for both standing conditions. Participants stood in front of a table adjusted to 5-6 cm below their wrist when their elbows were placed at 90 degrees, which is the position recommended for light work (Kroemer and Grandjean, 1997). The distance of the table from the participant was initially set up to be a fist width away from their trunk at the level of their abdomen.

6.2.5   Experimental Protocol

Participants came into the lab on two occasions separated by at least 48 hours. The order that the participants saw the conditions was randomized. Half the participants performed the level ground standing condition on Day 1, while the other half performed the sloped standing condition first. Participants filled out a baseline visual analogue scale score after completing informed consent and proceeded through the instrumentation process. A five second trial with the participant standing in anatomical position was recorded to characterize the motion capture set up, followed by a maximum active lumbar spine extension trial used as a reference joint angle. For the maximum extension trial, the participant was told to keep their knees locked and bend backwards about their lumbar spine without shifting their hips forward.

On the day that the participant stood on the sloped surface, two 60 second foot constrained standing trials were taken before starting the prolonged standing trial – one on level ground and one on the sloped surface. These standing trials assisted with comparing the acute postural changes between level ground and sloped standing. Stance width was standardized between the two standing conditions to control for foot position by creating an outline of a box using masking tape with dimension equal to the participant's foot length. The participant assumed a comfortable foot position within the box with the lateral border of the small toe positioned at the side of the box(Carpenter et al., 2001) and was told to, *"Stand as still as possible with your arms by your side and weight evenly distributed between your feet"*(Zok et al., 2008).

Once the pre-trials were taken, participants entered the 75 minute prolonged unconstrained standing trial. They took a visual analogue scale score at the start of the trial and then every 7.5 minutes.

6.2.6   Data Analysis

Twelve visual analogue scale scores were reported for each participant – one immediately when they entered the lab, one before entering the prolonged standing task, and 11 (one every 7.5 minutes) during the standing protocol. To assess the relative increase in visual analogue scale score attributed to the prolonged standing task, visual analogue scale scores upon entering the lab were subtracted from all visual analogue scale scores that followed. As a result, all participants began with an initial score of 0 mm.

Participants were categorized as pain developers based on their visual analogue scale scores reported *during the level ground standing condition*. As done previously in studies on prolonged standing (Gallagher et al., 2014; Marshall et al., 2011; Nelson-Wong et al., 2008b; Nelson-Wong and Callaghan, 2010c), a threshold of 10 mm with respect to baseline was used to categorize participants as pain developers. Hagg and colleagues (2003) found that when using a visual analogue scale to track worsening of low back pain, the clinical important difference for patients to feel their low back pain symptoms

worsening was 8 mm. As a result, 10 mm is a conservative threshold to use for categorizing participants as pain developers or non-pain developers.

Marker locations were brought into Visual3D (Version 4, C-Motion, Inc. Germantown, MS, USA) for post-processing. Segment locations were defined using the five second standing trial taken at the beginning of the experiment. If marker interpolation was required, a third order polynomial was used to fill gaps of a maximum 16 frames (0.5 seconds). Once interpolated, the signal was low pass filtered (Butterworth, 2nd order, dual pass) with an effective cut-off frequency of 4 Hz, as determined using a residual analysis (Study #1, Section 3.2.5.3). The orientation of each local coordinate system was defined with +y pointing proximally, +x pointing anteriorly, and +z pointing laterally to the right in the same manner as done for Study #1 (Study #1, Section 3.2.5.3). Joint angles were calculated using a flexion/extension-lateral bend-axial twist rotation sequence. All angles were expressed as the rotation (in degrees) from their measured maximum extension angle. Angles measured were trunk (trunk with respect to pelvis), lumbar spine (lumbar spine with respect to pelvis), hip angle (thigh with respect to pelvis), and trunk-to-thigh (thigh with respect to trunk). The anterior posterior distance between the location of the trunk center of gravity and the joint center of the right ankle and the anterior posterior distance between the trunk and pelvis center of gravity were also calculated.

For the constrained standing, means were taken for the two 60 second trials for comparison. For the prolonged standing trials, an amplitude probability distribution function (APDF) was used to extract the median posture/location (50th percentile) and range of movement (90th-10th percentiles) for each variable. In addition to this, lumbar spine fidgets, shown previously to differ between pain developers and non-pain developers, were defined as a fast, large displacement and return of the lumbar spine angle to approximately the same starting position. The parameters for the fidgets were the same as used previously (Dunk and Callaghan, 2010) – a change in threshold of +/- 3SD, window length of 60 seconds, and maximum duration of 4 seconds. Fidgets were calculated for the sagittal plane only.

6.2.7 Statistical Analyses

For constrained standing, outcome measures were entered into a three-way general linear model with between factors of gender and pain group (pain developers/non-pain developers) and within factor of standing condition (level/sloped). If there were no main effects or interactions including gender, the variable was collapsed and entered into the two-way ANOVA.

For prolonged standing, outcome measures were entered into a four way general linear model with between factors of gender and pain group and within factors of standing condition and time. When data did not meet the sphericity assumption (not immediately passed due to the time factor containing five levels), Huynh-Feldt-Lecourre Epsilon corrections were used and the p-value was adjusted accordingly. If there were no main effects or interactions including gender, the variable was collapsed and entered into the three-way ANOVA. For any significant main effects, Tukey post-hoc tests using the least square means were used. For any significant interactions, simple effects were used to assess differences. The alpha level of significance was set at $alpha < 0.05$ for all tests.

6.3 Results

6.3.1 Pain Group Categorization and Visual Analog Scores

Fifty-three percent of participants (9/17) were categorized as pain developers during the level ground trial. Of the nine, four were males and five females. There was a distinct separation of the visual analogue scale scores during the level ground session, with pain developers demonstrating an increase in pain scores over the 75 minutes (Figure 6.2). The average maximum score (Figure 6.3) for the pain developers during the control session was 16.2 (5.3) mm and 2.3 (3.4) mm for non-pain developers.

When standing on the sloped surface, the average maximum pain scores for pain developers was 58% less compared to their reports during level round standing (16.2 (5.3) mm for level ground versus 6.8 (5.8) mm for sloped). Of the nine participants categorized as pain developers during the level ground condition, only three of nine were still categorized as PDs during sloped standing. One pain developers

participant reported a higher visual analogue scale on the sloped surface, with one report up to 18 mm versus their level ground maximum of 10 mm. The other two participants reported scores that were 6 mm and 13 mm less to bring their maximum sloped standing visual analogue scale scores to 10 and 11 mm, respectively.
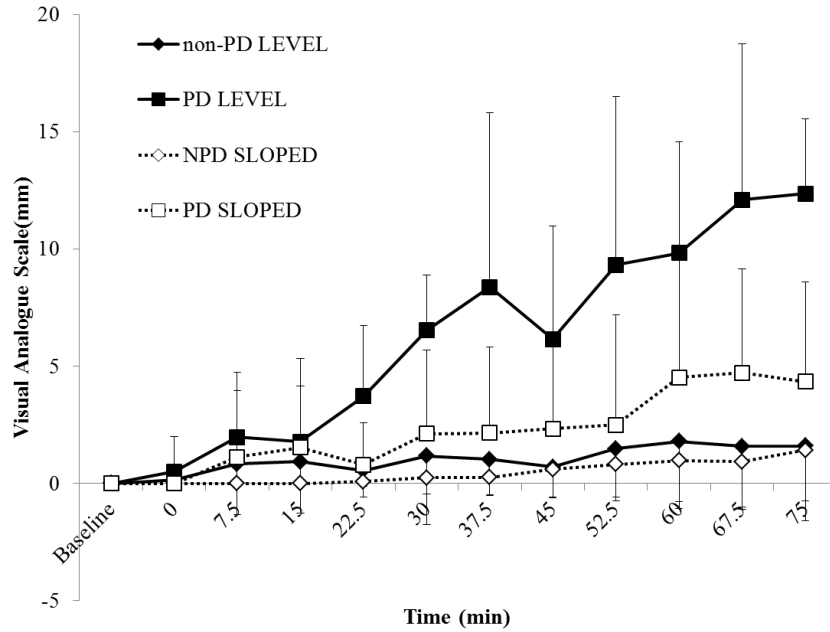
Figure 6.2. Visual analogue scale scores for pain (PD) and non-pain developers (non-PD) in the level ground and sloped standing conditions. Pain developers visual analogue scale scores were lower over the 75 minute trial during the sloped versus control condition.
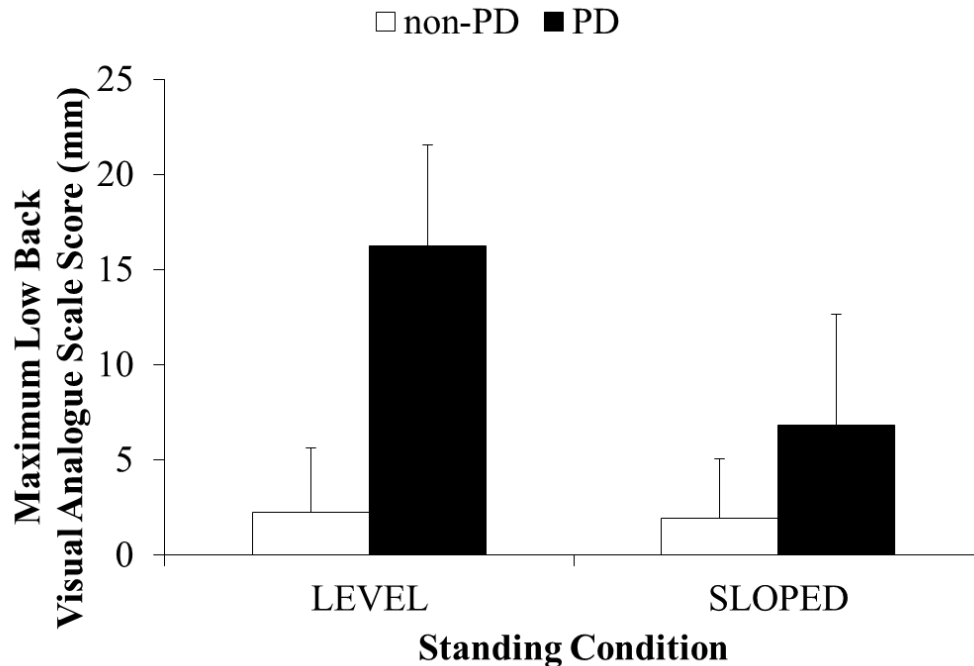
Figure 6.3. Maximum average visual analogue scale score for the low back region for pain (PD) and non-pain developers (non-PD). Pain developers demonstrated a 58% (~10 mm on average) drop in maximum visual analogue scale score during the sloped condition compared to the control condition.

In order to assess potential offsetting of other body regions, the visual analogue scale scores of the feet and calf regions were assessed. There were no changes in the visual analogue scale scores of the calf regions between level ground (8.6 mm +/- 10.3 mm) and the sloped surface conditions (8.1 mm +/- 9.8 mm) and only five of the 17 participants saw greater visual analogue scale scores in their calf region. There was an increase of 6 mm in the reported visual analogue scale scores for the foot region, with scores being higher on the sloped surface (16.0 mm +/- 14.3 mm) than on level ground (10.0 mm +/- 11.3 mm) and 11 of 17 participants saw an increase in their visual analogue scale scores for this region.

6.3.2   Foot Constrained Standing

There were no main effects or interaction containing pain group for all kinematic variables (Table 6.2). All participants stood approximately 15 (+/- 9) degrees away from maximum lumbar spine extension. The closest variable to demonstrate a main effect of pain group was trunk angle (p=0.1037). Pain developers

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demonstrated a trunk angle that was 4 degrees further away from the maximum trunk extension angle than non-pain developers.

The sloped surface induced hip flexion (p=0.0347), trunk-to-thigh flexion (p=0.0114), and moved the location of the trunk center of gravity posteriorly ($p<0.0001$) by 1.9 cm (level = 4.4 +/- 1.94 cm versus; sloped = 2.5 +/- 1.72 cm)).

Table 6.2. Kinematics from the foot constrained standing trials. Note: mean (standard deviation) in degrees (deg) or centimeters (cm). The asterisk (*) denotes the variables that showed a significant main effect of condition

	Level	**Sloped**
Trunk Angle (deg)	22.6 (10.6)	22.8 (10.6)
Lumbar Angle (deg)	15.5 (10.2)	15.4 (10.1)
Left Hip (deg)*	9.2 (10.5)	11.0 (11.2)
Right Hip (deg)*	9.6 (10.1)	11.3 (10.9)
Left Trunk-Thigh Angle (deg)*	31.1 (14.3)	33.0 (14.9)
Right Trunk-Thigh Angle (deg)*	31.5 (14.0)	33.3 (14.4)
Trunk center of gravity with respect to ankle Joint (cm, + = anterior)	4.43 (1.94)	2.55 (1.72)

6.3.3   Prolonged unconstrained Standing

For all participants, the lumbar spine was 2-3 degrees more flexed during the sloped condition (Figure 6.4); however, the standard deviation was large in comparison (10-12 degrees). There was a main effect of time ($p<0.0001$) such that there was a steady increase in lumbar spine flexion over the standing trials, but this was only by two degrees after 75 minutes.
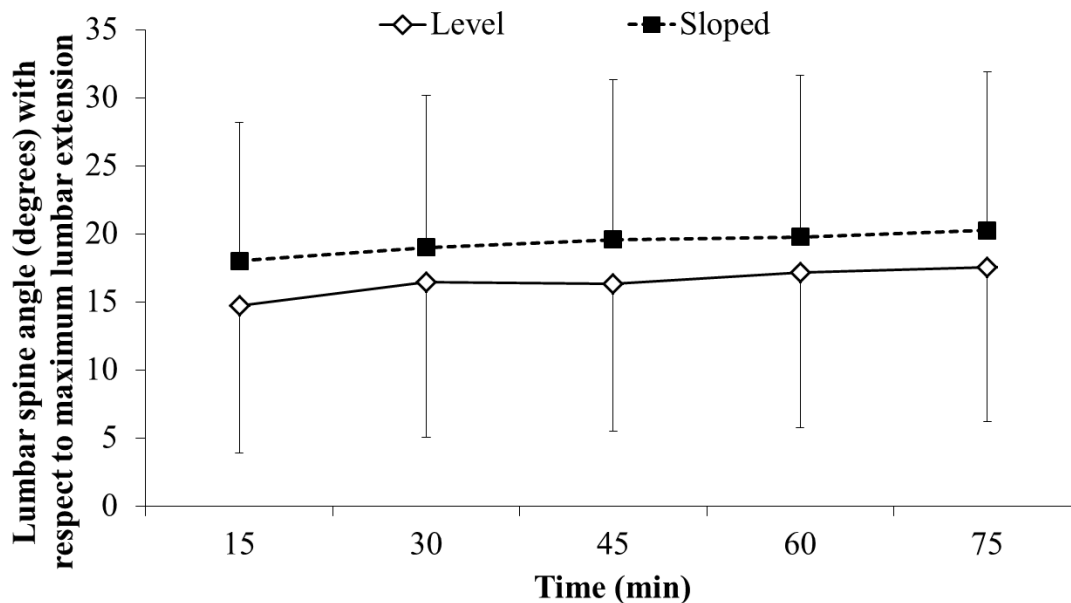
Figure 6.4. Lumbar spine angle compared between the control and sloped conditions. The greater the angle, the farther away the lumbar spine is from its maximum extension angle (0 degrees = maximum extension). There was a 2-3 degree difference in angle between during the two days; however, this was not significant. Over time, there was a steady trend towards greater flexion of the lumbar spine, but this was only by two degrees over the 75 minute trial.

Non-pain developers performed more lumbar spine fidgets in the sagittal plane than pain developers (p=0.0412), independent of the standing condition (Figure 6.5). Non-pain developers performed approximately 13 fidgets (+/-6.7) per 15 minutes during the level ground condition compared to 9 (+/- 6.8) by the pain developers. There was also a trend towards an interaction between standing condition and time (p=0.0598). During both conditions, participants performed approximately 14 fidgets during the first 15 minutes. After this period, fidget frequency remained constant for the sloped surface, but decreased to approximately 10 per 15 minutes during the level condition and remained there for the rest of the trial.
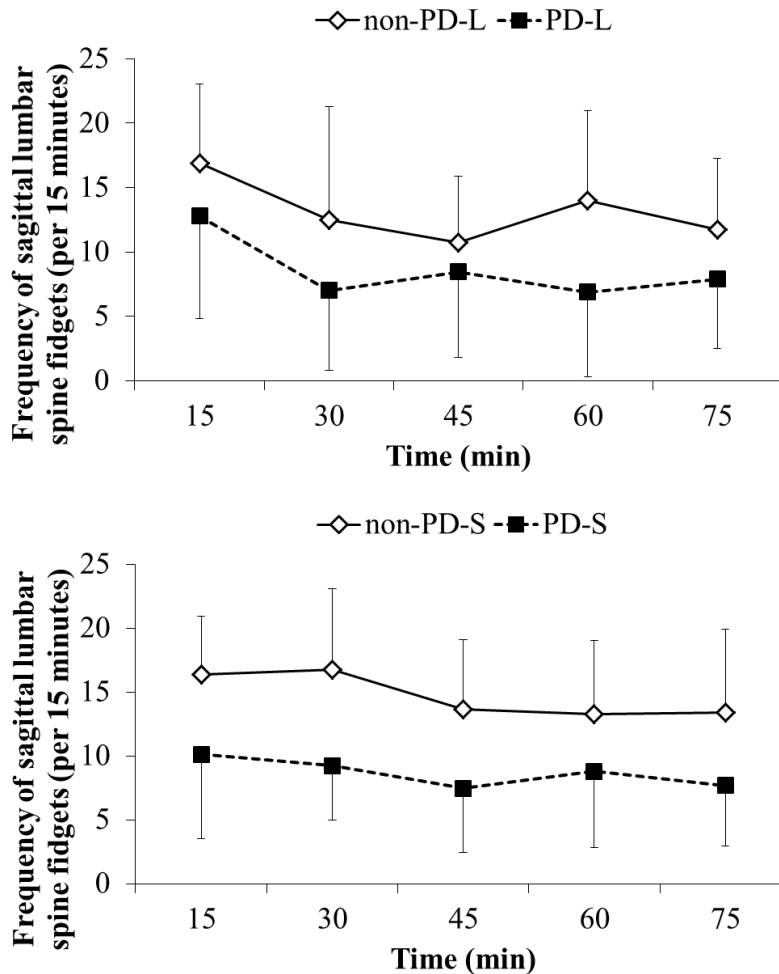
Figure 6.5. Frequency of sagittal lumbar spine fidgets (per 15 minutes) for pain (PD) and non-pain developers (non-PD) during level ground (top) and the sloped condition (bottom). During both conditions, non-pain developers had a higher frequency of fidgets compared to pain developers.

There was an interaction between pain group and standing condition (*p*=0.0217) for the median location of the trunk center of gravity location with respect to the right ankle (Figure 6.6, top row). During the level ground condition, the location of the trunk center of gravity in pain developers was on average 3.1 (+/- 1.5) cm in front of the right ankle, whereas it was 1.4 (+/- 1.8) cm for the NPD. During the sloped condition, the locations became similar between the two pain groups, with the trunk center of gravity moving posteriorly and vertically aligned with the right ankle joint (non-pain developers = 0.75 +/- 1.3 cm; pain developers = 0.83 +/- 1.3 cm).

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Movement of the trunk center of gravity with respect to the ankle joint varied between pain groups and over time (Figure 6.6, bottom row). There was a significant main effect of time ($p=0.0002$), and a trend towards an interaction between time and pain group ($p=0.0797$). Non-pain developers moved the location of their trunk center of gravity with respect to the right ankle about 3-4 cm over the standing trials; however, pain developers steadily increased their movement from 3-5 cm to almost 9 cm by the by the end of the standing trial.
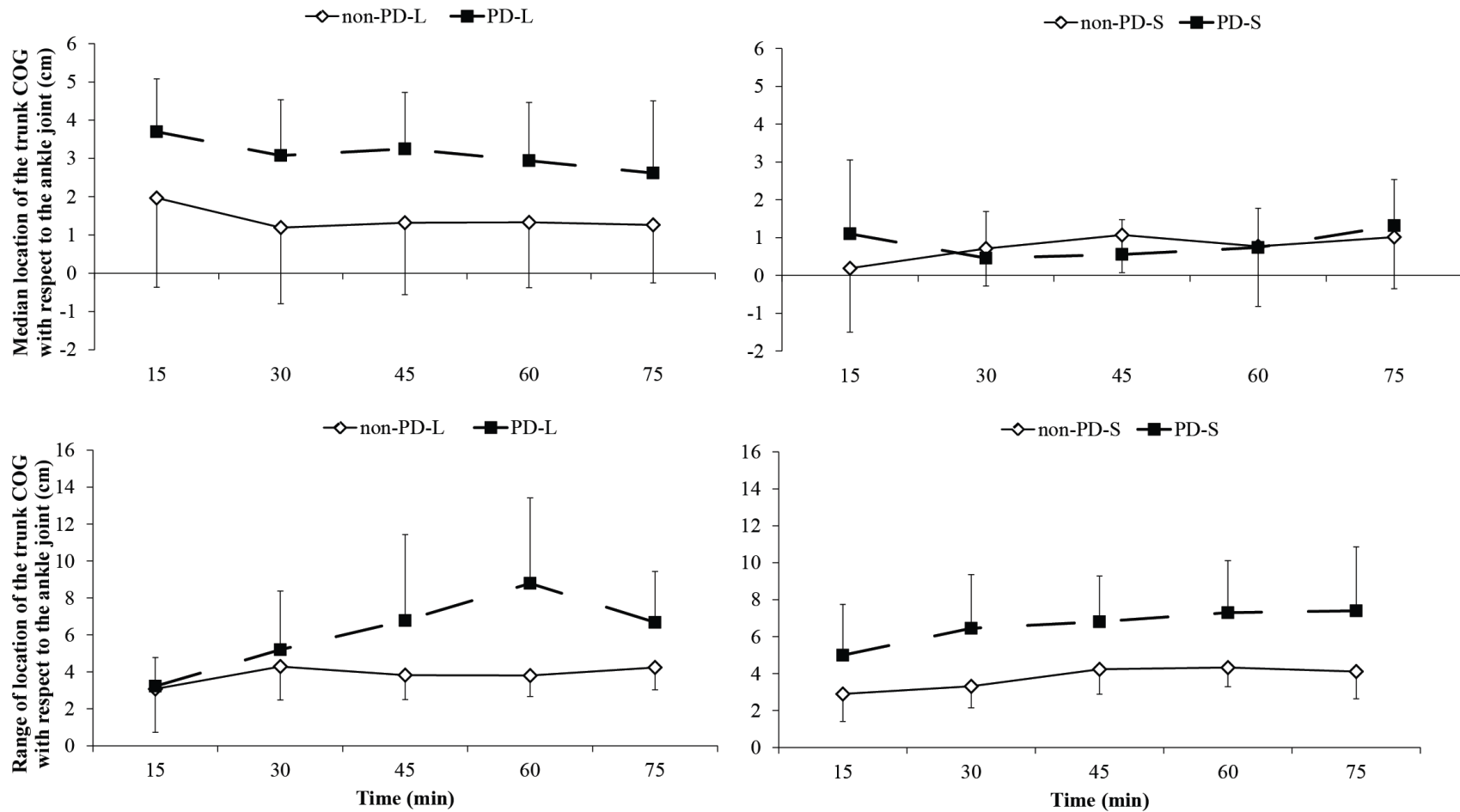
Figure 6.6. Median (top row) and range of movement (bottom row) of the trunk center of gravity with respect to the right ankle joint during the control condition (left side) and sloped condition (right side). For the top row, the closer the value is to zero, the closer the center of gravity was to the ankle joint. The trunk center of gravity was further in front of the ankle joint for pain developers during the control standing but moved closer to the ankle joint during the sloped standing. Pain developers tended to have a greater range of this variable compared to non-pain developers for both standing conditions.

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6.4 Discussion

Standing on a sloped surface with the ankles in plantar-flexion resulted in lower self-reported reports of low back pain development in those previously defined as pain developers when standing on level ground. While it was hypothesized that the main postural change between the two groups would be altered lumbar spine angle, this was not the case. The sloped surface had a greater effect on the hips, demonstrating greater flexion of the hip joints and the trunk-thigh angles, and posterior translation of the trunk center of gravity moving to become vertically aligned over the ankle joint. While non-pain developers demonstrated more movement of the lumbar spine during level ground standing, lumbar spine movement patterns were unchanged when standing on the sloped surface.

While previous work on a similar sloped surface found lumbar spine and trunk flexion induced by standing on a decline (Gallagher et al., 2013; Nelson-Wong and Callaghan, 2010b), this study did not show similar results. An internal measurement of lumbar spine angle using x-rays found that there was no difference in lumbar spine angle when standing on level ground versus a decline (Study #2, Section 4.3.2); therefore, it seems that it is not solely altering lumbar spine angle, but rather postural alterations facilitated through hip and thigh postural changes, that are important for this standing intervention's success.

The location of the trunk center of gravity with respect to the ankle joint during prolonged standing was the only variable that showed an interaction between pain group and standing condition. The trunk center of gravity was more posterior in non-pain developers than pain developers in level ground standing. During sloped standing, the trunk center of gravity moved posteriorly for all participants compared to level ground standing and was in line with the ankle joint. The difference during level ground standing would mean a greater moment arm with respect to the ankle for the force of the trunk due to gravity in pain developers; however, the implications of this with respect to low back pain development are unknown.

Sloped standing appears to prompt participants to perform a hip dominant correction. Flexing at the hips and moving the trunk and pelvis as a unit can accomplish this change accomplished without altering the moment induced by the trunk center of gravity at the L5/S1 joint. Flexion of the hips allows for a greater passive force contribution of posterior muscles that cross the hip joint since they are stretched to the right or up the increasing slope on their force-length curves, which increases the contribution of the passive properties to force production. This change could be why we see lower gluteus medius co-contraction when using this standing aid (Nelson-Wong and Callaghan, 2010b), since the higher passive contribution from the posterior hip muscles can assist in stabilizing the hip joint. While there were no obvious changes in pelvis angle, a slight posterior rotation of the pelvis induced by the sloped surface may also explain the larger L5/S1 anterior-posterior shear previously noted (Nelson-Wong and Callaghan, 2010b).

While a relationship between less movement of the lumbar spine and pain development during standing has been shown previously (Gallagher et al., 2014), the sloped surface did not induce altered movement patterns. As in previous work (Study #1, 3.3.2), non-pain developers fidgeted more than pain developers during the first 15 minutes; however, in this study that trend continued for the entire 75-minute trial. On the sloped surface, both pain developers and non-pain developers had a steady frequency of fidgeting over the entire trial; however, non-pain developers still fidgeted more than pain developers. This could point to a fundamental difference between the two groups in terms of how they move as the differences occur prior to pain development – non-pain developers can control their lumbar spine movement better than pain developers.

A limitation to this study was that the prolonged standing kinematics were measured over two days. The order of the conditions was randomized to prevent any learning effects; however, there might have been a small influence of between day error caused by land marking and marker placement. The strength of this experimental design was that it removed any fatigue or accumulated pain that would occur within a day. Second, we did not assess the center of pressure data for either collection day as accounting

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for the sloped surface may have cause larger error when determining the center of pressure location. To account for this in future studies, we plan to use either a pressure mat placed on top of the sloped surface or pressure insoles to remove any potential error. Lastly, we did not study intermittent use of the sloped surface, which is more likely to occur due to potential lower limb discomfort that could be caused by the posture changes, as is seen when placing one foot on an elevated surface (Whistance et al., 1995). While the purpose of the study was to alter posture without also inducing cyclic lumbar spine movement, future work will assess the relationship cyclic postures and prolonged standing induced low back pain.

When assessing the impact of an intervention over two separate days, the repeatability of pain group classification using the visual analogue scale scores must be discussed. In a previous study where participants performed two 2-hour prolonged standing trials separated by four weeks, 83% of the participants remained in their same pain group (Nelson-Wong and Callaghan, 2010d). In this study, all non-pain developers remained non-pain developers in the sloped standing condition. Only one pain developers had higher pain scores during the sloped condition and two remained pain developers but saw scores that were lower by 10 and 11 mm. Thus, 89% (8 of 9) pain developers reported lower pain scores during the sloped standing, thus we can say that the improvement was due to the sloped surface and not participants being improperly classified during the level ground condition.

6.5 Conclusions

Standing on a declined slope surface resulted in a lower of self-reported low back pain reports during prolonged standing. Compared to standing on level ground, the sloped surface resulted in larger hip flexion, trunk-to-thigh flexion, and smaller moment arm of the trunk center of gravity with respect to the ankle joint. This change in hip posture could result in higher passive stiffness produced by the muscles crossing posterior aspect of the hip joint to assist with stabilizing the pelvis without the use of the gluteus medius co-contraction. Future studies will continue concentrate on hip kinematics through changes in foot posture without the use of device and the influence of an exercise program that is positive for reducing prolonged standing induced low back pain on the hip joint kinematics.

7   General Discussion

7.1   Thesis Questions Revisited

The global objective of this thesis was to investigate the relationship between low back pain development, lumbopelvic posture, and movement patterns in prolonged standing induced pain developers using a non-pain developers sample as a comparison group. Below is a summary of the thesis findings with respect to the specific questions posed in Chapter #1 (Section 1.6).

Are movement patterns different between pain and non-pain developers during prolonged standing?

The studies in this thesis showed that movement patterns differed between the pain groups. In Study #1 (Chapter 3), non-pain developers fidgeted their lumbar spine more frequently about the flexion/extension axis and had more large body weight transfers than pain developers during the first 15 minutes of standing. After 15 minutes of standing the two groups exhibited similar movement patterns. Study #4 (Chapter 6) also showed that non-pain developers had more flexion/extension lumbar spine fidgets than pain developers; however, this was consistent over a 75-minute prolonged standing trial and was independent of the type of standing aid used (level ground versus sloped standing).

Do pain and non-pain developers have different lumbar spine and pelvic flexion/extension postures?

Studies #2 (Chapter 4) and #3 (Chapter 5) demonstrated that lumbopelvic postures about the flexion/extension axes differ between pain developers and non-pain developers. A radiographic imaging study (Chapter 4) showed that lumbosacral lordosis and L5/S1 intervertebral joint angle were more extended for pain developers than non-pain developers during level ground standing; however, there were no differences in gross (L1/S1) lumbar lordosis angle. Study #3 (Chapter 5) showed that there was a trend for non-pain developers to stand closer to their *in vivo* lumbar spine flexion/extension passive neutral zone. As a result, the lower lumbar arc is important at modulating pain development during prolonged

standing and the area that future studies should concentrate on when assessing differences between the pain groups.

What is the potential for different foot positions and standing aids to alter lumbar and pelvic flexion/extension posture?

For all participants, Study #2 (Chapter 4) showed that lumbosacral lordosis angle was brought into the greatest amount of flexion when standing with one foot on an elevated surface. The sloped surface brought the L1/L2 intervertebral joint into the greatest amount of flexion. Study #3 (Chapter 5) showed that the use of the elevated surface brought the lumbar spine angle into mild flexion and reduced the associated lumbar spine passive stiffness in both pain developers and non-pain developers when standing in this position. The sloped surface and staggered stance did not affect the relationship between the lumbar spine angle and neutral zone location. While it is possible to positively alter the lumbar spine posture so that it functions within its lumbar spine flexion/extension passive stiffness curve, not all standing interventions hypothesized to be successful work in the same manner.

Can a standing aid that alters posture or movement patterns prevent low back pain development in standing?

In study #4 (Chapter 6), low back pain development and kinematic variables were compared between level ground standing and standing on a sloped surface. Standing on a declining surface resulted pain scores that were 58% lower for pain developers compared to their reports during prolonged level ground standing. Sloped standing caused greater hip flexion, trunk-to-thigh flexion, and a smaller moment arm of the trunk center of gravity with respect to the ankle joint but no change in trunk angle or lumbar spine flexion/extension postures. As a result, flexion of the hips and not just the lumbar spine can assist with reducing prolonged standing induced low back pain development.

7.2 Major Contributions

Contribution #1: There is a relationship between posture and low back pain development during prolonged standing.

This thesis adds to the evidence that the pain development process begins prior to the reporting of pain development and any intervention that is proposed must address an individual's standing posture. Studies #2 (Chapter 4) and #3 (Chapter 5), both demonstrated postural differences between pain and non-pain developers. In Study #4 (Chapter 6), changing a person's standing posture for the entire protocol reduced pain development. Once a person is aware of their pain development, it is too late to make a modification, as even reducing the pain slightly through a short change in posture, such as a seated break, does not completely remove the pain when standing is resumed (Gallagher et al., 2014).

Contribution #2: The lower lumbar lordosis and hip angles are the regions of interest for further assessing low back pain development during prolonged standing.

Previous work has shown that lower lumbar arc has the greatest variability within a sample (Roussouly et al., 2005), so it is not surprising that this is where the difference in posture between pain developers and non-pain developers was found. In erect standing, the facet joints bear approximately 16% of the compressive load, whereas in a flexed posture they resist almost none (Adams and Hutton, 1980). Thus the more flexed lumbosacral lordosis angle means that non-pain developers may be supporting less of their compressive load with their facet joints in comparison to non-pain developers. This would prevent potential pain development impingement of the facet joint capsule (highly innervated by free nerve endings (Cavanaugh, 1995)) between the facet tip and lamina or pedicle when standing (Dunlop et al., 1984). This change in lower lumbar arc may also be related to the co-contraction of the gluteus medius muscles, as this could negatively modify the lumbosacral angle. While Studies #2 (Chapter 4) and #3 (Chapter 5) did not demonstrate many favourable kinematics changes when using the sloped surface, a majority of the kinematic changes in Study #4 (Chapter 6) were related to larger hip flexion induced by the sloped surface.

Contribution #3: While small movements of the lumbar spine were greater in non-pain developers, the direct link between movement patterns and pain development is still unknown.

While there were differences in movement patterns between pain groups, this study did not directly test the hypothesis that altering movement patterns would alter pain development. First, gluteus medius co-contraction is a confounding factor that may lead to this lack of movement found in Studies #1 (Chapter 3) and #4 (Chapter 6). While gluteus medius co-contraction is higher in pain developers (Marshall et al., 2011; Nelson-Wong and Callaghan, 2010c) and has been used to predict those who develop low back pain (Nelson-Wong et al., 2008b), the very low levels of muscle contraction are likely not directly responsible for the quickly developing low back pain during standing; however, they may be responsible for the lack of movement in pain developers. Study #1 (Chapter 3) showed that movement patterns were different between pain developers and non-pain developers during the first 15 minutes of standing; however, Study #4 (Chapter 6) showed lower self-reported visual analogue scale scores for low back pain when posture was altered but the lumbar spine fidgeting frequency was unchanged. This evidence supports the previous hypothesis that pain developers stood in a more frozen posture than non-pain developers; therefore, pain developers may not have the capabilities to perform this movement pattern. With the previous success of an exercise intervention on pain development (Nelson-Wong and Callaghan, 2010a), specifically targeting muscular control strategies through an exercise intervention may be more important than simply focusing on changes in movement patterns alone for reducing low back pain development during level ground standing.

Contribution #4: Successful standing aids for reducing low back pain alter the lower lumbar flexion/extension arc and hip kinematics about the flexion/extension axis.

The standing aids served two purposes in this study. The first purpose was to assess their influence on lumbopelvic posture and develop hypotheses regarding their success. The second purpose was to link back to how people stand when they are on level ground so that we can further pinpoint the mechanism of low back pain. Consistent to what was found on level ground, the standing aids in this thesis demonstrated the importance of lower lumbar flexion/extension arc and hip posture about the

flexion/extension axis during standing. Study #4 (Chapter 6) showed that a postural change caused by standing on a sloped surface still reduced low back pain development despite not altering lumbar spine flexion/extension fidgeting patterns. Mild lumbosacral lordosis flexion induced by placing one foot on the elevated surface may reduce stress on the facet joints, reduce compressive stress on the posterior annulus, and improve compressive strength of the spine compared to upright and extended postures (Adams and Hutton, 1985). Study #2 (Chapter 4) showed that the sloped surface did not modify the lower lumbar arc as much as the elevated surface and Study #3 (Chapter 5) showed that lumbar spine passive stiffness in this posture remained unchanged compared to level ground. Despite this, the sloped surface may modify hip angle enough that it caused enough flexion to reduce a mechanical insult to the posterior tissues.

7.3   Working Hypothesis of Prolonged Standing Induced low back pain development

Based on the findings from this thesis and previous work, the following working hypothesis on low back pain development during prolonged standing was established. On level ground, people prone to low back pain development stand with their lower lumbar arc closer to their end range of extension for this region. This was shown in Study #2 (Chapter 4), which found that pain developers stood with more lumbosacral lordosis and L5/S1 intervertebral joint extension. This places additional strain and mechanical insult on the posterior elements of the lumbar spine, causing transient pain development. Study #3 (Chapter 5) showed that pain developers stood in state of higher lumbar spine passive stiffness compared to non-pain developers. It may also prevent pain developers from performing small movements of their lumbar spine that are seen in non-pain developers (Studies #1 (Chapter 3) and #4 (Chapter 6)).

When mild flexion of the hips and lumbar spine is initiated through sitting or alterations of lower limb posture, mechanical insult on the posterior elements of the lumbar spine is lessened and low back pain decreases. Study #2 (Chapter 4) showed that when placing one foot on an elevated surface, all participants demonstrated lumbosacral lordosis flexion and Study #3 (Chapter 5) demonstrated that this

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standing aid brought participants into a posture that induced less lumbar spine passive stiffness. Study #4 (Chapter 6) showed that despite not inducing large changes in flexion of the lumbar spine or changes in lumbar spine flexion/extension movement patterns, a sloped surface that induced hip dominant flexion/extension rotation and posterior translation of the trunk center of gravity in the sagittal plane still reduced the self-reported low back pain reports in pain developers.

The origin of the flexion/extension lumbar spine, pelvis, and hip postural differences between the pain groups is unknown; however, there is evidence that the difference may be due to poor muscle activation patterns. Since an exercise intervention concentrating on hip and trunk stabilization has been shown to reduce low back pain reports (Nelson-Wong and Callaghan, 2010a), altering muscle activation patterns could promote mild flexion of the lumbar spine, posterior rotation of the pelvis, and altered hip angles. It could also allow people to better control their lumbar spine movement and posture while standing.

7.4   General Limitations

Separating out the contribution of movement and posture was a challenge in this thesis. Study #1 (Chapter 3) to compare movement and Studies #2 (Chapter 4) and #3 (Chapter 5) to assess posture were designed to look at movement and posture in as much of a separate way as possible, with Study #4 (Chapter 6) integrating their results. Initially, the elevated surface was going to be tested in Study #4 (Chapter 6), as it was the most commonly recommended aid in the literature; however, previous work shows that standing in this posture results in lower limb discomfort (Whistance et al., 1995) that was confirmed in pilot work. It was determined that it would be very challenging for the participants to stand in one position without cycling the leg that was on the elevated surface and the sloped surface was chosen for evaluation as the standing aid in study #4 (Chapter 6). Previous work demonstrated that when individuals were allowed to cycle between standing on an incline and decline, low back pain was less during prolonged standing (Nelson-Wong and Callaghan, 2010b). Participants stood for a majority of their time on the decline

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surface, so the logical next step in that study was to assess standing only in the decline position and see if the results were similar. Using this approach still confirmed the effectiveness of changing a person's standing posture for promoting lower low back pain reports despite movement patterns remaining unchanged between level ground and sloped surface standing.

The balance between acute and prolonged data collections to assess posture and movement was also a challenge. Study #4 (Chapter 6) showed that standing on the sloped surface showed slight lumbar spine flexion during the prolonged session but not between the two 60 second foot constrained standing trials. During the prolonged sessions, pain developers' trunk center of gravity was more anterior to the ankle joint compared to non-pain developers – a result not evident during constrained standing. The foot constrained standing studies (Studies #2 (Chapter 4) and #3 (Chapter 5)) from this thesis provided general information about posture; however, these are only snapshots in time and it has been clearly shown that both pain and non-pain developers move within a range during prolonged standing. During 3 minutes of standing, the lumbar spine moves through approximately 10% of total flexion range of motion(Callaghan and McGill, 2001). When working at a computer, non-pain developers move through a range of approximately 11 degrees and pain developers 4 degrees (Gallagher et al., 2014). It was especially hard to look at the staggered stance condition since slight differences in performing the task could change the results.

When assessing the posture over five minutes as in Study #3, a difficult decision was whether to allow people to shift the forward leg in the elevated and staggered stance conditions. On one-hand, cycling between elevated legs is more realistic. On the other hand, the goal of the study was to assess posture and if cyclic movement was induced it could have confounded the results. While the prolonged sessions provide a large amount of information on the pain results, movement patterns, and posture over time, they are lengthy to collect and thus limit the number of measures or conditions that can be collected. A major strength of this thesis is a combination between the prolonged and acute assessment of movement and posture; however, applying what has been learned in the acute studies to prolonged

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assessment, and vice versa needs to be continued in future research on this topic as current work highlights the potential errors that could be present if simple representative postures are used to evaluate the dynamics of standing work.

7.5 Future Directions

The results of this study provided many new and interesting questions regarding the relationship between postures, movement patterns, and prolonged standing-induced low back pain. The following are six questions that were built from my thesis results.

Question #1: If pain developers adapt a more flexed posture (either through sitting or the use of a standing aid) before pain development occurs, can pain development be reduced or eliminated? Given the link between standing posture and pain development, and the evidence that changing posture once pain development occurs is not effective at completely eliminating pain development (Gallagher et al., 2014), future studies should assess what cyclic changes in posture from typical level ground standing early in a trial do for pain development. An example of this would be still keeping a 3:1 standing to sitting ratio as seen in Gallagher et al.(2014), but keeping the duty cycle shorter such that the person stands for 15 minutes then sits for 5 minutes instead of 45 minutes standing 15 minutes sitting as has been assessed before. While this may not be the most convenient duty cycle for workers, it will provide insight into what early changes in posture can do for reducing low back pain development. Another important reason for pursuing this question is to provide advice for how people should interact with a standing aid that they cannot use for a prolonged period of time, such as the elevated surface.

Question #2: Are there different postures that people can stand in that reduce pain development without the use of a standing aid? In some work environments there may not be room for the aid or it may pose a tripping hazard. The results of this thesis with regards to posture and movement patterns can help to better define new standing positions that will be successful to reduce low back pain development.

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Question #3: Do standing aids that do not change lower limb posture, such as insoles or anti-fatigue matting, alter the movement and posture variables in a similar way to the aids mentioned in this study? Anti-fatigue matting is a very common recommendation for prolonged standing tasks, but their effectiveness in reducing low back pain through inducing measurable physiologic changes is debated. Using the results of this thesis, lumbar spine fidget movements and lumbopelvic posture can be assessed to see if these mats induce similar changes to bring the movement and posture characteristics of pain developers and non-pain developers closer together.

Question #4: If the movement patterns of pain developers are standardized to mimic non-pain developers during prolonged standing, is the pain development reduced in pain developers? While a change in posture was assessed in Study #4 (Chapter 6), it would be interesting to see if standardizing movement patterns so that all participants performed the same cyclic movements over a standing trial reduces low back pain development. An example of how to do this is to have all participants stand on level ground, but lift up one leg onto the elevated surface at fixed intervals and immediately place it back on the ground. In this manner, movement is being induced but the majority of time is spent in their typical standing posture.

Question #5: Can isolating the *in vivo* passive characteristics of the lower lumbar spine provide better information on the differences between pain developers and non-pain developers during standing? While Study#3 (Chapter 5) held the pelvis locked and had the entire lumbar spine suspended between the lower and upper body cradle of the near-frictionless jig, in hindsight and based on findings from the radiographic study (Chapter 4) it may not have been the best way to test differences between the pain groups since most differences were found in the lower lumbar spine. Keeping the trunk locked in one position and suspending only the lower lumbar vertebrae between the two cradles while having the lower limbs on the frictionless surface may provide better evidence of the basic passive characteristics between the pain groups and where people stand with respect to the passive neutral zone of this region.

Question #6: Does an exercise intervention that reduces low back pain development in standing change the posture and movement characteristics found different between the pain groups in this thesis? While an exercise intervention that promoted stabilization of the hip and trunk (Nelson-Wong and Callaghan, 2010a) has been shown to be successful, the mechanism for how this was effective at reducing low back pain is still unknown. By assessing these movement patterns and postural characteristics before and after an exercise intervention, we could get much more evidence on why it was successful and continue to determine more of the fundamental differences between pain developers and non-pain developers as well as guide future interventions and workplace practices for standing workers.

References

Adams, M. A., Dolan, P., Hutton, W. C., 1988. The lumbar spine in backward bending. Spine 13, 1019-1026.

Adams, M. A., Hutton, W. C., 1986. The effect of posture on diffusion into lumbar intervertebral discs. Journal of Anatomy 147, 121-134.

Adams, M. A., Hutton, W. C., 1985. The effect of posture on the lumbar spine. The Journal of Bone and Joint Surgery.British Volume 67, 625-629.

Adams, M. A., Hutton, W. C., 1983. The effect of posture on the fluid content of lumbar intervertebral discs. Spine 8, 665-671.

Adams, M. A., Hutton, W. C., 1980. The effect of posture on the role of the apophysial joints in resisting intervertebral compressive forces. The Journal of Bone and Joint Surgery.British Volume 62, 358-362.

Adams, M. A., McMillan, D. W., Green, T. P., Dolan, P., 1996. Sustained loading generates stress concentrations in lumbar intervertebral discs. Spine 21, 434-438.

Airaksinen, O., Brox, J. I., Cedraschi, C., Hildebrandt, J., Klaber-Moffett, J., Kovacs, F., Mannion, A. F., Reis, S., Staal, J. B., Ursin, H., Zanoli, G., COST B13 Working Group on Guidelines for Chronic Low Back Pain, 2006. Chapter 4. European guidelines for the management of chronic nonspecific low back pain. European Spine Journal 15 Suppl 2, S192-300.

Andersen, J. H., Haahr, J. P., Frost, P., 2007. Risk factors for more severe regional musculoskeletal symptoms: a two-year prospective study of a general working population. Arthritis and Rheumatism 56, 1355-1364.

Bakker, E. W., Verhagen, A. P., van Trijffel, E., Lucas, C., Koes, B. W., 2009. Spinal mechanical load as a risk factor for low back pain: a systematic review of prospective cohort studies. Spine 34, E281-93.

Beach, T. A. C., Parkinson, R. J., Stothart, J. P., Callaghan, J. P., 2005. Effects of prolonged sitting on the passive flexion stiffness of the in vivo lumbar spine. The Spine Journal 5, 145-154.

Beck, L., 2011. Prolonged bouts of sitting increase cancer risk. Globe and Mail.

Benden, M. E., Blake, J. J., Wendel, M. L., Huber, J. C.,Jr, 2011. The impact of stand-biased desks in classrooms on calorie expenditure in children. American Journal of Public Health 101, 1433-1436.

Bogduk, N., Tynan, W., Wilson, A. S., 1981. The nerve supply to the human lumbar intervertebral discs. Journal of Anatomy 132, 39-56.

Botranger, K. L., 2002. Botranger's Pocket Atlas: Handbook of Radiographic Position and Technique. 4th Edition.

Bridger, R. S., Wilkinson, D., van Houweninge, T., 1989. Hip joint mobility and spinal angles in standing and in different sitting postures. Human Factors 31, 229-241.

Brumagne, S., Lysens, R., Swinnen, S., Verschueren, S., 1999. Effect of paraspinal muscle vibration on position sense of the lumbosacral spine. Spine 24, 1328-1331.

Callaghan, J. P., Gregory, D. E., Durkin, J. L., 2010. Do NIRS measures relate to subjective low back discomfort during sedentary tasks? International Journal of Industrial Ergonomics 40, 165-170.

Callaghan, J. P., McGill, S. M., 2001. Low back joint loading and kinematics during standing and unsupported sitting. Ergonomics 44, 280-294.

Canadian Center of Occupational Health and Safety (CCHOS), 2008. Working in A standing Position. 2012.

Carlsöö, S., 1961. The static muscle load in different work positions: An electromyographic study. Ergonomics 4, 193-211.

Carpenter, M. G., Frank, J. S., Silcher, C. P., Peysar, G. W., 2001. The influence of postural threat on the control of upright stance. Experimental Brain Research 138, 210-218.

Carson, R., 1995. How to select a proper workbench. Occupational Health & Safety 64, 53-56.

Cavanaugh, J. M., 1995. Neural mechanisms of lumbar pain. Spine 20, 1804-1809.

Cavanaugh, J. M., Ozaktay, A. C., Yamashita, T., Avramov, A., Getchell, T. V., King, A. I., 1997. Mechanisms of low back pain: a neurophysiologic and neuroanatomic study. Clinical Orthopaedics and Related Research (335), 166-180.

Choi, B. C., Eijkemans, G. J., Tennassee, L. M., 2001. Prioritization of occupational sentinel health events for workplace health and hazard surveillance: the Pan American Health Organization experience. Journal of Occupational and Environmental Medicine 43, 147-157.

Christie, H. J., Kumar, S., Warren, S. A., 1995. Postural aberrations in low back pain. Archives of Physical Medicine and Rehabilitation 76, 218-224.

Claus, A., Hides, J. A., Moseley, G. L., Hodges, P. W., 2009. Different ways to balance the spine: subtle changes in sagittal spinal curves affect regional muscle activity. Spine 34, E208-14.

Cohen, A. L., Gjessing, C. C., Fine, L. J., Bernard, B. P., McGlothin, J. D., 1997. Elements of Ergonomics Programs: A primer based on workplace evaluations of musculoskeletal disorders. 97-177.

Cook, A. J., Degood, D. E., 2006. The cognitive risk profile for pain: development of a self-report inventory for identifying beliefs and attitudes that interfere with pain management. The Clinical Journal of Pain 22, 332-345.

Dagenais, S., Roffey, D. M., Wai, E. K., 2010. Reply: Lack of ''statistically significant'' association does not exclude causality. The Spine Journal 10, 944-945.

Day, J. W., Smidt, G. L., Lehmann, T., 1984. Effect of Pelvic Tilt on Standing Posture. Physical Therapy 64, 510-516.

De Carvalho, D. E., Callaghan, J. P., 2011. Passive stiffness changes in the lumbar spine and effect of gender during prolonged simulated driving. International Journal of Industrial Ergonomics 41, 617-624.

De Carvalho, D. E., Soave, D., Ross, K., Callaghan, J. P., 2010. Lumbar Spine and Pelvic Posture Between Standing and Sitting: A Radiologic Investigation Including Reliability and Repeatability of the Lumbar Lordosis Measure. Journal of Manipulative and Physiological Therapeutics 33, 48-55.

Dolan, P., Adams, M. A., Hutton, W. C., 1988. Commonly adopted postures and their effect on the lumbar spine. Spine 13, 197-201.

Drake, J. D. M., Callaghan, J. P., 2009. Intervertebral neural foramina deformation due to two types of repetitive combined loading. Clinical Biomechanics 24, 1-6.

Drake, J. D. M., Callaghan, J. P., 2008. Do flexion/extension postures affect the in vivo passive lumbar spine response to applied axial twist moments? Clinical Biomechanics 23, 510-519.

Duarte, M., Harvey, W., Zatsiorsky, V. M., 2000. Stabilographic analysis of unconstrained standing. Ergonomics 43, 1824-1839.

Duarte, M., Zatsiorsky, V. M., 1999. Patterns of center of presure migration during prolonged unconstrained standing. Motor Control 3, 12-27.

Dunk, N. M., Callaghan, J. P., 2010. Lumbar spine movement patterns during prolonged sitting differentiate low back pain developers from matched asymptomatic controls. Work 35, 3-14.

Dunk, N. M., Lalonde, J., Callaghan, J. P., 2005. Implications for the use of postural analysis as a clinical diagnostic tool: reliability of quantifying upright standing spinal postures from photographic images. Journal of Manipulative and Physiological Therapeutics 28, 386-392.

Dunlop, R. B., Adams, M. A., Hutton, W. C., 1984. Disc space narrowing and the lumbar facet joints. The Journal of Bone and Joint Surgery.British Volume 66, 706-710.

Dunstan, D. W., Barr, E. L., Healy, G. N., Salmon, J., Shaw, J. E., Balkau, B., Magliano, D. J., Cameron, A. J., Zimmet, P. Z., Owen, N., 2010. Television viewing time and mortality: the Australian Diabetes, Obesity and Lifestyle Study (AusDiab). Circulation 121, 384-391.

Ellis, P. D., 2010. The Essential Guide to Effect Sizes: Statistical Power, Meta-Analysis, and the Interpretation of Research Results. Cambridge University Press, Cambridge, UK, .

Engels, J. A., van der Gulden, J. W., Senden, T. F., van't Hof, B., 1996. Work related risk factors for musculoskeletal complaints in the nursing profession: results of a questionnaire survey. Occupational and Environmental Medicine 53, 636-641.

Folsom, A. R., Jacobs, D. R.,Jr, Caspersen, C. J., Gomez-Marin, O., Knudsen, J., 1986. Test-retest reliability of the Minnesota Leisure Time Physical Activity Questionnaire. Journal of Chronic Diseases 39, 505-511.

Freitas, S. M. S. F., Wieczorek, S. A., Marchetti, P. H., Duarte, M., 2005. Age-related changes in human postural control of prolonged standing. Gait & Posture 22, 322-330.

Fujiwara, A., An, H. S., Lim, T. H., Haughton, V. M., 2001. Morphologic changes in the lumbar intervertebral foramen due to flexion-extension, lateral bending, and axial rotation: an in vitro anatomic and biomechanical study. Spine 26, 876-882.

Galante, J. O., 1967. Tensile properties of the human lumbar annulus fibrosus. Acta Orthopaedica Scandinavica, Suppl 100:1-91.

Gallagher, K. M., Campbell, T., Callaghan, J. P., 2014. The influence of a seated break on prolonged standing induced low back pain development. Ergonomics, 1-8.

Gallagher, K. M., Nelson-Wong, E., Callaghan, J. P., 2011. Do individuals who develop transient low back pain exhibit different postural changes than non-pain developers during prolonged standing? Gait & Posture 34, 490-495.

Gallagher, K. M., Wong, A., Callaghan, J. P., 2013. Possible mechanisms for the reduction of low back pain associated with standing on a sloped surface. Gait & Posture 27, 313-318.

Gardiner, P. A., Healy, G. N., Eakin, E. G., Clark, B. K., Dunstan, D. W., Shaw, J. E., Zimmet, P. Z., Owen, N., 2011. Associations between television viewing time and overall sitting time with the metabolic syndrome in older men and women: the Australian Diabetes, Obesity and Lifestyle study. Journal of the American Geriatrics Society 59, 788-796.

Gardner-Morse, M. G., Stokes, I. A., 2003. Physiological axial compressive preloads increase motion segment stiffness, linearity and hysteresis in all six degrees of freedom for small displacements about the neutral posture. Journal of Orthopaedic Research 21, 547-552.

Gilchrist, R. V., Slipman, C. W., Bhagia, S. M., 2002. Anatomy of the intervertebral foramen. Pain Physician 5, 372-378.

Goertzen, D. J., Lane, C., Oxland, T. R., 2004. Neutral zone and range of motion in the spine are greater with stepwise loading than with a continuous loading protocol. An in vitro porcine investigation. Journal of Biomechanics 37, 257-261.

Gombatto, S. P., Norton, B. J., Scholtes, S. A., Van Dillen, L. R., 2008. Differences in symmetry of lumbar region passive tissue characteristics between people with and people without low back pain. Clinical Biomechanics 23, 986-995.

Green, T. P., Allvey, J. C., Adams, M. A., 1994. Spondylolysis. Bending of the inferior articular processes of lumbar vertebrae during simulated spinal movements. Spine 19, 2683-2691.

Gregory, D. E., Callaghan, J. P., 2008. Prolonged standing as a precursor for the development of low back discomfort: an investigation of possible mechanisms. Gait & Posture 28, 86-92.

Hagg, O., Fritzell, P., Nordwall, A., Swedish Lumbar Spine Study Group, 2003. The clinical importance of changes in outcome scores after treatment for chronic low back pain. European Spine Journal 12, 12-20.

Harkness, E. F., Macfarlane, G. J., Nahit, E. S., Silman, A. J., McBeth, J., 2003. Risk factors for new-onset low back pain amongst cohorts of newly employed workers. Rheumatology 42, 959-968.

Harrison, D. E., Cailliet, R., Harrison, D. D., Troyanovich, S. J., Harrison, S. O., 1999. A review of biomechanics of the central nervous system--Part I: spinal canal deformations resulting from changes in posture. Journal of Manipulative and Physiological Therapeutics 22, 227-234.

Healing Arts Radiation Protection Act, 1990. RRO 1990, Regulation 543 Xray Safety Code.

Healy, G. N., Matthews, C. E., Dunstan, D. W., Winkler, E. A., Owen, N., 2011. Sedentary time and cardio-metabolic biomarkers in US adults: NHANES 2003-06. European Heart Journal 32, 590-597.

Hubbard, R. D., Chen, Z., Winkelstein, B. A., 2008. Transient cervical nerve root compression modulates pain: load thresholds for allodynia and sustained changes in spinal neuropeptide expression. Journal of Biomechanics 41, 677-685.

Inufusa, A., An, H. S., Lim, T. H., Hasegawa, T., Haughton, V. M., Nowicki, B. H., 1996. Anatomic changes of the spinal canal and intervertebral foramen associated with flexion-extension movement. Spine 21, 2412-2420.

Janevic, J., Ashton-Miller, J. A., Schultz, A. B., 1991. Large compressive preloads decrease lumbar motion segment flexibility. Journal of Orthopaedic Research 9, 228-236.

Keegan, J. J., 1953. Alterations of the lumbar curve related to posture and seating. The Journal of Bone and Joint Surgery.American Volume 35-A, 589-603.

Kent, P., Keating, J., 2004. Do primary-care clinicians think that nonspecific low back pain is one condition? Spine 29, 1022-1031.

Kirby, R. L., Price, N. A., MacLeod, D. A., 1987. The influence of foot position on standing balance. Journal of Biomechanics 20, 423-427.

Konz, S. A., Rys, M. J., 2003. An ergonomics approach to standing aids. Occupational Ergonomics 3, 165-172.

Kopec, J. A., Sayre, E. C., Esdaile, J. M., 2004. Predictors of back pain in a general population cohort. Spine 29, 70-7; discussion 77-8.

Krause, N., Lynch, J. W., Kaplan, G. A., Cohen, R. D., Salonen, R., Salonen, J. T., 2000. Standing at work and progression of carotid atherosclerosis. Scandinavian Journal of Work, Environment & Health 26, 227-236.

Kroemer, K. H. E., Grandjean, E., 1997. Fitting the task to the human: A textbook of occupational ergonomics. CRC Press, .

Kuijer, P. P. F. M., Frings-Dresen, M. H. W., Gouttebarge, V., van Dieën, J. H., van der Beek, A. J., Burdorf, A., 2011. Low back pain: we cannot afford ignoring work. The Spine Journal 11, 164.

Lafond, D., Champagne, A., Descarreaux, M., Dubois, J., Prado, J. M., Duarte, M., 2009. Postural control during prolonged standing in persons with chronic low back pain. Gait & Posture 29, 421-427.

Latouche, C., Jowett, J. B., Carey, A. L., Bertovic, D. A., Owen, N., Dunstan, D. W., Kingwell, B. A., 2013. Effects of breaking up prolonged sitting on skeletal muscle gene expression. Journal of Applied Physiology 114, 453-460.

Levine, D., Whittle, M. W., 1996. The effects of pelvic movement on lumbar lordosis in the standing position. The Journal of Orthopaedic and Sports Physical Therapy 24, 130-135.

Macfarlane, G. J., Thomas, E., Papageorgiou, A. C., Croft, P. R., Jayson, M. I., Silman, A. J., 1997. Employment and physical work activities as predictors of future low back pain. Spine 22, 1143-1149.

Marshall, P. W. M., Patel, H., Callaghan, J. P., 2011. Gluteus medius strength, endurance, and co-activation in the development of low back pain during prolonged standing. Human Movement Science 30, 63-73.

Martin Bland, J., Altman, D., 1986. Statistical methods for assessing agreement between two methods of clinical measurement. The Lancet 327, 307-310.

McDonald, A. D., McDonald, J. C., Armstrong, B., Cherry, N. M., Cote, R., Lavoie, J., Nolin, A. D., Robert, D., 1988. Fetal death and work in pregnancy. British Journal of Industrial Medicine 45, 148-157.

McGill, S. M., Seguin, J., Bennett, G., 1994. Passive stiffness of the lumbar torso in flexion, extension, lateral bending, and axial rotation. Effect of belt wearing and breath holding. Spine 19, 696-704.

McGill, S. M., 2011. Letter to the Editor regarding: "Causal assessment of occupational lifting and low back pain: results of a systematic review" by Wai et al. The Spine Journal 11, 365.

McGinn, D., 2011. Is your office chair killing you? Globe and Mail.

Mendelek, F., Kheir, R. B., Caby, I., Thevenon, A., Pelayo, P., 2011. On the quantitative relationships between individual/occupational risk factors and low back pain prevalence using nonparametric approaches. Joint Bone Spine 78, 619-624.

Messing, K., Fortin, S., Rail, G., Randoin, M., 2005. Standing still: why North American workers are not insisting on seats despite known health benefits. International Journal of Health Services : Planning, Administration, Evaluation 35, 745-763.

Messing, K., Stock, S., Tissot, F., 2013. The Importance of Not Standing Too Long. Archives of Environmental & Occupational Health 68, 128-129.

Mitchell, T., O'Sullivan, P. B., Burnett, A. F., Straker, L., Smith, A., 2008. Regional differences in lumbar spinal posture and the influence of low back pain. BMC Musculoskeletal Disorders 9, 152.

Mozurkewich, E. L., Luke, B., Avni, M., Wolf, F. M., 2000. Working conditions and adverse pregnancy outcome: a meta-analysis. Obstetrics and Gynecology 95, 623-635.

National Council on Radiation Protection and Measurements, 2009. Ionizing radiation, exposure of population in United States. 160.

Nelson-Wong, E., 2009. Biomechanical predictors of functionally induced low back pain, acute response to prolonged standing exposure, and impact of stabilization-based clinical exercise intervention. PhD Thesis. University of Waterloo.

Nelson-Wong, E., Alex, B., Csepe, D., Lancaster, D., Callaghan, J. P., 2012. Altered muscle recruitment during extension from trunk flexion in low back pain developers. Clinical Biomechanics 27, 994-998.

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, E379-E383.

Nelson-Wong, E., Callaghan, J. P., 2010a. Changes in muscle activation patterns and subjective low back pain ratings during prolonged standing in response to an exercise intervention. Journal of Electromyography and Kinesiology 20, 1125-1133.

Nelson-Wong, E., Callaghan, J. P., 2010b. The impact of a sloped surface on low back pain during prolonged standing work: A biomechanical analysis. Applied Ergonomics 41, 787-795.

Nelson-Wong, E., Callaghan, J. P., 2010c. Is muscle co-activation a predisposing factor for low back pain development during standing? A multifactorial approach for early identification of at-risk individuals. Journal of Electromyography and Kinesiology 20, 256-263.

Nelson-Wong, E., Callaghan, J. P., 2010d. Repeatability of Clinical, Biomechanical, and Motor Control Profiles in People with and without Standing-Induced Low Back Pain. Rehabilitation Research and Practice 2010, 289278.

Nelson-Wong, E., Flynn, T., Callaghan, J. P., 2009a. Development of active hip abduction as a screening test for identifying occupational low back pain. The Journal of Orthopaedic and Sports Physical Therapy 39, 649-657.

Nelson-Wong, E., Gregory, D. E., Winter, D. A., Callaghan, J. P., 2008a. Gender differences in postural control strategies during prolonged standing. In North American Conference on Biomechanics (NACOB), Ann Arbor, MI.

Nelson-Wong, E., Gregory, D. E., Winter, D. A., Callaghan, J. P., 2008b. Gluteus medius muscle activation patterns as a predictor of low back pain during standing. Clinical Biomechanics 23, 545-553.

Nelson-Wong, E., Howarth, S., Winter, D. A., Callaghan, J. P., 2009b. Application of autocorrelation and cross-correlation analyses in human movement and rehabilitation research. The Journal of Orthopaedic and Sports Physical Therapy 39, 287-295.

Nelson-Wong, E., Howarth, S. J., Callaghan, J. P., 2010. Acute biomechanical responses to a prolonged standing exposure in a simulated occupational setting. Ergonomics 53, 1117-1128.

Occupational Safety & Health Administration (OSHA), 2012. Youth Worker Safety in Restaurants (eTool) - Prolonged Standing. 2012.

O'Sullivan, P. B., Grahamslaw, K. M., Kendell, M., Lapenskie, S. C., Moller, N. E., Richards, K. V., 2002. The effect of different standing and sitting postures on trunk muscle activity in a pain-free population. Spine 27, 1238-1244.

Oxland, T. R., Panjabi, M. M., 1992. The onset and progression of spinal injury: A demonstration of neutral zone sensitivity. Journal of Biomechanics 25, 1165-1172.

Panjabi, M. M., 1992a. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. Journal of Spinal Disorders 5, 383-9; discussion 397.

Panjabi, M. M., 1992b. The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis. Journal of Spinal Disorders 5, 390-6; discussion 397.

Panjabi, M. M., Takata, K., Goel, V. K., 1983. Kinematics of lumbar intervertebral foramen. Spine 8, 348-357.

Parkinson, R. J., Beach, T. A. C., Callaghan, J. P., 2004. The time-varying response of the in vivo lumbar spine to dynamic repetitive flexion. Clinical Biomechanics 19, 330-336.

Prado, J. M., Dinato, M. C. M., Duarte, M., 2011. Age-related difference on weight transfer during unconstrained standing. Gait & Posture 33, 93-97.

Radiological Society of North American and American College of Radiology, 2011. Patient Safety: Radiation Exposure in X-ray and CT Examinations. 2012.

Raftry, S. M., Marshall, P. W. M., 2012. Does a 'tight' hamstring predict low back pain reporting during prolonged standing? Journal of Electromyography and Kinesiology.

Roelen, C. A. M., Schreuder, K. J., Koopmans, P. C., Groothoff, J. W., 2008. Perceived job demands relate to self-reported health complaints. Occupational Medicine 58, 58-63.

Roffey, D. M., Wai, E. K., Bishop, P., Kwon, B. K., Dagenais, S., 2010. Causal assessment of occupational standing or walking and low back pain: results of a systematic review. The Spine Journal 10, 262-272.

Roussouly, P., Gollogly, S., Berthonnaud, E., Dimnet, J., 2005. Classification of the normal variation in the sagittal alignment of the human lumbar spine and pelvis in the standing position. Spine 30, 346-353.

Rys, M. J., Konz, S., 1989. Standing with one foot forward. Advances in Industrial Ergonomics and Safety I, 207-213.

Rys, M. J., Konz, S., 1994. Standing. Ergonomics 37, 676-687.

Scannell, J. P., McGill, S. M., 2003. Lumbar posture--should it, and can it, be modified? A study of passive tissue stiffness and lumbar position during activities of daily living. Physical Therapy 83, 907-917.

Seifert, A. M., Messing, K., Dumais, L., 1997. Star wars and strategic defense initiatives: work activity and health symptoms of unionized bank tellers during work reorganization. International Journal of Health Services : Planning, Administration, Evaluation 27, 455-477.

Sorenson, C. J., Norton, B. J., Callaghan, J. P., Van Dillen, L. R., 2014. Lumbar curvature angles in low back pain developers and non-pain developers. In Institute of Clinical and Translational Sciences Conference., Washington, D.C.

Stagnara, P., De Mauroy, J. C., Dran, G., Gonon, G. P., Costanzo, G., Dimnet, J., Pasquet, A., 1982. Reciprocal angulation of vertebral bodies in a sagittal plane: approach to references for the evaluation of kyphosis and lordosis. Spine 7, 335-342.

Tait, R. C., Chibnall, J. T., 1997. Development of a brief version of the Survey of Pain Attitudes. Pain 70, 229-235.

Takala, E., 2010. Lack of "statistically significant" association does not exclude causality. The Spine Journal 10, 944.

Teras, L. R., Gapstur, S. M., Ryan Diver, W., Birmann, B. M., Patel, A. V., 2012. Recreational physical activity, leisure sitting time and risk of non-hodgkin lymphoid neoplasms in the american cancer society cancer prevention Study-II cohort. International Journal of Cancer 131, 1912-1920.

Thompson, R. E., Barker, T. M., Pearcy, M. J., 2003. Defining the Neutral Zone of sheep intervertebral joints during dynamic motions: an in vitro study. Clinical Biomechanics 18, 89-98.

Tissot, F., Messing, K., Stock, S., 2009. Studying the relationship between low back pain and working postures among those who stand and those who sit most of the working day. Ergonomics 52, 1402-1418.

Tissot, F., Messing, K., Stock, S., 2005. Standing, sitting and associated working conditions in the Quebec population in 1998. Ergonomics 48, 249-269.

Tuchsen, F., Hannerz, H., Burr, H., Krause, N., 2005. Prolonged standing at work and hospitalisation due to varicose veins: a 12 year prospective study of the Danish population. Occupational and Environmental Medicine 62, 847-850.

US Food and Drug Administration, 2009. What are the Radiation Risks from CT? 2012.

Vergara, M., Page, A., 2002. Relationship between comfort and back posture and mobility in sitting-posture. Applied Ergonomics 33, 1-8.

Vergara, M., Page, A., 2000. System to measure the use of the backrest in sitting-posture office tasks. Applied Ergonomics 31, 247-254.

Waddell, G., Newton, M., Henderson, I., Somerville, D., Main, C. J., 1993. A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. Pain 52, 157-168.

Wall, B. F., Hart, D., 1997. Revised radiation doses for typical X-ray examinations. Report on a recent review of doses to patients from medical X-ray examinations in the UK by NRPB. National Radiological Protection Board. The British Journal of Radiology 70, 437-439.

Waters, T. R., Dick, R. B., 2014. Evidence of Health Risks Associated with Prolonged Standing at Work and Intervention Effectiveness. Rehabilitation Nursing.

Whistance, R. S., Adams, L. P., van Geems, B. A., Bridger, R. S., 1995. Postural adaptations to workbench modifications in standing workers. Ergonomics 38, 2485-2503.

Winkelstein, B. A., DeLeo, J. A., 2004. Mechanical thresholds for initiation and persistence of pain following nerve root injury: mechanical and chemical contributions at injury. Journal of Biomechanical Engineering 126, 258-263.

Winkelstein, B. A., Weinstein, J. N., DeLeo, J. A., 2002. The role of mechanical deformation in lumbar radiculopathy: an in vivo model. Spine 27, 27-33.

Winter, D. A., Prince, F., Stergiou, P., Powell, C., 1993. Medial-lateral and anterior-posterior motor responses associated with centre of pressure changes in quiet standing. Neuroscience Research Communications 12, 141-148.

Winter, D. A., Prince, F., Frank, J. S., Powell, C., Zabjek, K. F., 1996. Unified theory regarding A/P and M/L balance in quiet stance. Journal of Neurophysiology 75, 2334-2343.

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

Wu, G., Siegler, S., Allard, P., Kirtley, C., Leardini, A., Rosenbaum, D., Whittle, M., D'Lima, D. D., Cristofolini, L., Witte, H., Schmid, O., Stokes, I., 2002. ISB recommendation on definitions of joint coordinate system of various joints for the reporting of human joint motion—part I: ankle, hip, and spine. Journal of Biomechanics 35, 543-548.

Yamashita, T., Cavanaugh, J. M., el-Bohy, A. A., Getchell, T. V., King, A. I., 1990. Mechanosensitive afferent units in the lumbar facet joint. The Journal of Bone and Joint Surgery.American Volume 72, 865-870.

Yang, K. H., King, A. I., 1984. Mechanism of facet load transmission as a hypothesis for low-back pain. Spine 9, 557-565.

Yochum, T., Rowe, L., 1996. Essentials of Skeletal Radiology - Volume 1. Williams and Wilkins, Baltimore, MD, .

Zacharkow, D., 1988. Posture: sitting, standing, chair design, and exercise. Thomas, Springfield, Ill, USA, .

Zok, M., Mazza, C., Cappozzo, A., 2008. Should the instructions issued to the subject in traditional static posturography be standardised? Medical Engineering & Physics 30, 913-916.

Appendix A: Questions taken from psychosocial surveys.

Table A.1. Questions taken from the Cognitive Risk Profile for Pain (Cook and Degood, 2006)

Strongly Agree	Moderately Agree	Slightly Agree	Slightly Disagree	Moderately Disagree	Strongly Disagree	**Please Rate your level of agreement with the following statements**
1	2	3	4	5	6	Feeling angry can increase my pain
1	2	3	4	5	6	Pain can put me in a bad mood
1	2	3	4	5	6	Exercise can help to manage my pain
1	2	3	4	5	6	My life should be pain free
1	2	3	4	5	6	Worry can increase the pain I feel
1	2	3	4	5	6	My attitude and the way I think are an important part of how to manage my pain
1	2	3	4	5	6	Stress in my life can make my pain feel worse
1	2	3	4	5	6	Pain can make me feel depressed.

Table A.2 Questions from the Survey of Pain Attitudes – Brief (Tait and Chibnall, 1997)

Please rate your level of agreement with the following statements	Very Untrue	Somewhat Untrue	Neither True nor Untrue/ or Does Not Apply	Somewhat True	Very True
There are many times when I can influence the amount of pain I feel	0	1	2	3	4
When I hurt, I want my family to treat me better	0	1	2	3	4
Anxiety increases the pain I feel	0	1	2	3	4
When I am hurting, people should treat me with care and concern	0	1	2	3	4
It is the responsibility of my loved ones to help me when I feel pain	0	1	2	3	4
Exercise and movement are good for a pain problem	0	1	2	3	4
Just by concentrating or relaxing, I can 'take the edge' off my pain	0	1	2	3	4
Medicine is one of the best treatments for chronic pain	0	1	2	3	4
Depression increases the pain I feel	0	1	2	3	4
If I exercise, I could make my pain problem much worse	0	1	2	3	4
I believe that I can control how much pain I feel by changing my thoughts	0	1	2	3	4
Often I need more tender loving care than I am now	0	1	2	3	4

getting when I am in pain					
There is a strong connection between my emotions and my pain level	0	1	2	3	4

Table A.3 Questions from the Fear Avoidance Beliefs Questionnaire (Waddell et al., 1993)

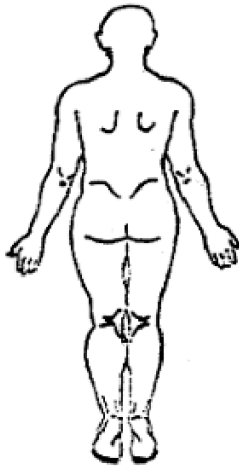| Please rate your level of agreement with the following statements	Completely Disagree	Moderately Disagree	Slightly Disagree	Unsure	Slightly Agree	Moderately Agree	Completely Agree
Physical activity might harm my back	0	1	2	3	4	5	6
I should not do physical activities that (might) make my pain worse	0	1	2	3	4	5	6
My work is too heavy for me	0	1	2	3	4	5	6
My work might harm my back	0	1	2	3	4	5	6

Appendix B: Visual Analogue Scale for Low Back Pain Development

VISUAL ANALOG SCALE FOR PAIN

Please place a mark on the line to indicate the CURRENT level of pain in your LOW BACK

No Pain   Worst Pain Imaginable

Please mark the location of your   Please check the words that you would use to describe your
pain on the body chart   CURRENT level of pain in your LOW BACK*

☐ throbbing   ☐ tiring-exhausting
☐ shooting   ☐ sickening
☐ stabbing   ☐ fearful
☐ sharp   ☐ cruel-punishing
☐ cramping
☐ gnawing
☐ hot-burning
☐ aching   *Melzack, 1987.
☐ heavy
☐ tender

BACK

Figure B.1. Sample visual analogue scale for rating of low back pain during prolonged standing. Note that when the participant took the scale, the horizontal line was drawn to 100 mm.