The Effects of Hip Abductor Fatigue on Low Back Pain Development during Prolonged Standing

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 final revisions, as accepted by my examiners.

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Daniel Viggiani

Abstract

Pain and muscle fatigue are two factors that are linked to musculoskeletal injuries. Not only do both factors interact with each other, but their interactions depend on the specific tasks used to induce pain or fatigue. The goal of this thesis was to explore the interaction between low back pain developed from prolonged standing and fatigue of the hip abductors muscle group. Specifically, how does perturbing those who do and do not develop pain in standing postures using muscle fatigue affect the muscular responses and postures adopted while standing? The hip abductors were chosen as they represent the muscle group with the greatest potential to influence other muscular or postural responses during standing with respect to musculoskeletal function and pain generation.

Forty young healthy participants (20 male, 20 female) were recruited to perform two sessions of standing work for two hours each. Participants performed a side-lying leg raising (hip abduction) exercise to fatigue prior to one of the standing sessions, the other session acted as a control. Surface electromyography (EMG) of six muscles bilaterally, motion capture of the trunk and lower limbs, and force plate data under each foot were measured continuously during each standing session. EMG data were also collected during fatiguing exercise trials. Self-reported pain and isometric hip abductor strength were assessed at discrete time points using 100 mm visual analog scales and uni-axial force transducers tethered to participant's legs respectively. Pain measures were taken at baseline and every 15 minutes during standing in each session. Strength measures were taken at time points 0 minutes, 1 minute, 2 minutes, 3 minutes, 5 minutes, 10 minutes, 15 minutes 30 minutes and every 15 minutes thereafter during standing and were normalized to a baseline strength measure occurring immediately after instrumentation. EMG data from the standing exposures were expressed as a percentage of maximal voluntary isometric

contraction then used to compute cocontraction indices of 10 muscle pairs and resting gap measures on all 12 muscles. EMG data from the exercise protocol were used to assess fatigue through decreases in mean power frequencies. Motion capture data were used to compute low frequency postural responses through averaging lumbar spine, pelvic tilt and bilateral hip angles in 15 minute blocks. Force plate data were used to quantify transient movements through centre of pressure motion and medial-lateral body weight transfers. Pain scores were used to classify participants into pain developers (PDs) and non-pain developers (NPDs) based on a 10 mm threshold in low back pain scores during the control standing session.

PDs (8 male, 8 female) reported decreases in low back pain while standing during the fatigue session compared to the control session (10.9 ± 11.7 mm reduction) while NPDs reported minimal changes in pain with fatigue (1.5 ± 3.9 mm increase). While there were decreases, PDs were still experiencing low back pain of intensities above the 10 mm threshold in the fatigue session. Also, male and female PDs reported different pain patterns during the fatigue session. A decrease in trunk (1033.9 ± 528.6 %MVIC reduction across R-LES/R-EXO, L-LES/L-EXO and R-LES/L-LES CCIs in first hour) and gluteal cocontraction indices (398.8 ± 792.4 %MVIC decrease in the 15 minute block) and an increase in the number of anterior-posterior centre of pressure fidgets (13.5 ± 25.4 increase in the 30 minute block) within the first hour of the fatigue session were associated with fatigue-related pain reductions in PDs of both genders. Female PDs had more posterior pelvic tilt with fatigue (3.9 ± 9.2° more posterior with fatigue from 30 to 90 minutes) that distinguished them from male PDs, whose pelvises were more anteriorly tilted with fatigue (7.0 ± 11.3° more anterior with fatigue from 15 to 120 minutes). Additional changes seen exclusively in PDs during the fatigue session, such as larger force residuals with time, lateral

migration of centre of pressures and increases in tensor fascia latae activity, indicate that hip abductor fatigue did not recover while standing for PDs.

The fatigue protocol resulted in reductions in mean power frequency in four to nine of the 12 muscles measured, and affected both pain groups similarly with respect to EMG frequency shifts and strength lost. Females had longer times to fatigue (F: 21.7 ± 12.5 minutes; M: 17.2 ± 7.1 minutes; $p = 0.0031$) and smaller force losses with fatigue than males (F: 7.2 ± 10.4 %Baseline; M: 12.8 ± 11.1 %Baseline; $p = 0.0500$), and NPDs had longer times to fatigue than PDs (NPDs: 20.1 ± 11.2 minutes; PDs: 18.7 ± 9.3 minutes; $p = 0.0106$).

This study provides evidence that the hip abductor musculature is likely one causative factor in the low back pain developed from standing, although it appears as though both static and dynamic postural responses are important in the low back pain pathway of prolonged standing. Also, this study provides further evidence that a two hour standing exposure can identify those likely to develop chronic low back pain in the future in that PDs had fatiguing characteristics of persons suffering from chronic low back pain, only without any pain present. Exercise protocols aimed at fatiguing a single hip abductor do not appear to be muscle-specific, and the muscles fatigued by gross motor exercise are different between participants and cannot be predicted based on low back pain development during standing.

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My participants are my heroes. They willingly stood half naked at least twice connected to giant science machines with their leg tied to a wall while enduring monotonous tasks that were designed to inflict pain. This study gained infamy around those who heard about it due to the "extreme unpleasantness" of the fatigue protocol, yet participants kept coming back. Upon explaining this project to people outside of academia, the first response was always: "who would do that?" But actually, this thesis would not have been possible without them.

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Section 1: INTRODUCTION

Pain and fatigue are two common factors associated with musculoskeletal injury (Ding et al., 2000; Kumar, 2001; Gregory et al., 2008b). The severity, duration and disability caused by pain or fatigue are moderated by a number of different factors which can have profound influences on the perception and actions taken as a result of that pain (Loeser and Melzack, 1999; Altas and Wager, 2012; Gore et al., 2012) or fatigue (Gandevia, 2001; Enoka and Duchateau, 2008). Another common feature to pain and fatigue is a task dependency. The mechanisms behind the causes of both phenomena will differ dependent on, for example, what movements exacerbate that pain (O'Sullivan, 2005; Fritz et al., 2007), the painful person's attitudes towards their treatment process (Finniss et al., 2010), the activity that resulted in fatigue (Yung et al., 2012), or the gender of the person being fatigued (Albert et al., 2006).

It has also been shown that pain and fatigue interact with each other. In some instances, inducing fatigue has been shown to simulate the responses of those with chronic pain (Janssens et al., 2010; Johanson et al., 2011). In other instances, those in pain will respond differently to fatigue than those without chronic pain (Kankaanpää et al., 1998; Cheung, 2012; Negahban et al., 2013, Sutherlin and Hart, 2015). Pain can reduce a person's resistance to fatigue (Biering-Sorensen, 1984; Ciubotariu et al., 2004, 2007), alter the muscles that become fatigued from a task (Kankaanpää et al., 1998), or slow the recovery from fatigue (Roy et al., 1990; Peach and McGill, 1998). What has been made clear is that the nature of the interaction between fatigue and pain is dependent on what specific types of pain and fatigue are coupled together, as well as the measures used to assess that interaction (Williams et al., 2010). For example, in the lower leg, intermittent plantar and dorsi flexion can reduce the severity of pain, if the pain was induced by repetitive

eccentric loading (Sakamoto et al., 2010), but that same exercise will exacerbate pain symptoms if the pain is induced by a hypertonic saline injection (Graven-Nielsen et al., 1997).

One of the major drawbacks of the existing studies focused on the pain-fatigue interaction is that both the types of fatiguing tasks (sustained, high level contractions or high velocity isokinetic contractions) and the varieties of pain induced (injection of noxious substances, noxious thermal stimuli) are not frequently encountered outside of laboratory settings. The purpose of this project was to determine the interaction of a specific, workplace relevant type of pain generating task (prolonged standing – Gregory and Callaghan, 2008; Nelson-Wong et al., 2008; Tissot et al., 2009; Nelson-Wong and Callaghan, 2010a, 2014) with fatigue of a muscle that would likely modulate the development of that pain.

The muscle group chosen for fatigue, the hip abductors, is one that is both relevant to the pain development associated with prolonged standing (Nelson-Wong et al., 2008; Nelson-Wong and Callaghan, 2010b) and the postural control of standing (Winter et al., 1996; Salavati et al., 2007; Bellew et al., 2009). It could be argued based on the scientific literature (reviewed in Section 2.1) that the lumbar erector spinae might have been a better choice to fatigue than the hip abductors prior to a prolonged standing protocol. The evidence behind this logic is that it is believed that those who develop pain from standing (pain developers) activate gluteus medius to compensate for inadequate trunk stability stemming from a "faulty" erector spinae activation pattern (Gregory et al., 2008a; Nelson-Wong and Callaghan, 2010a, 2010b). The gluteus medius response could be an indicator of other deficits in trunk neuromuscular control. Since fatigue of a muscle can simulate pain in some standing tasks (Janssens et al., 2010; Johanson et al., 2011), inducing lumbar erector spinae fatigue could make those who do not develop pain from standing (non-pain developers) respond like pain developers, potentially providing very strong evidence for the pain pathway in

standing exposures. However, gluteus medius fatigue has been chosen instead for a number of reasons. First, the observed bilateral gluteus medius cocontraction in pain developers may not be a response to "faulty" erector spinae activation, but rather the faulty erector spinae activation could be an outcome of the bilateral gluteus medius cocontraction. If true, fatiguing gluteus medius may cause pain developers to behave like non-pain developers, forcing the pain developers to use erector spinae over gluteus medius. Second, determining how unilateral fatigue influences the bilateral gluteus medius cocontraction response of pain developers can allow for novel insights into the purpose of that cocontraction response. For example, if unilateral fatigue results in increased activation of the fatigued side only, then a potential purpose of the cocontraction could be to generate a requisite level of force. Lastly, studies using a prolonged standing protocol to induce low back pain consistently identify gluteus medius muscle activity as a differentiating factor between pain developers and non-pain developers (Gregory and Callaghan, 2008; Nelson-Wong et al., 2008, 2010; Nelson-Wong and Callaghan, 2010a, 2010b, 2010c; Raftry and Marshall, 2012). In contrast, there are minimal (Nelson-Wong et al., 2008) or no (Gregory and Callaghan, 2008; Nelson-Wong and Callaghan, 2010a, 2010b, 2010c) differences in lumbar erector spinae activation patterns between the two groups. Therefore, gluteus medius fatigue is the logical starting point, although there is ample rationale to introduce lumbar erector spinae fatigue in later projects.

1.1 Questions and Hypotheses

Specific questions to be examined include:

- Are muscles, the hip abductors in particular, a source of pain in prolonged standing?
- Does a gluteus medius fatigue protocol act as an analgesic (reduce reported pain), a hyperalgesic (increase reported pain) or an allodynic (make a non-painful stimulus painful) stimulus?

- Does hip abductor fatigue simulate the muscular and postural responses of those with low back pain in standing in those without low back pain from standing?
- How does hip abductor fatigue recover, if at all while performing a low-level workplace simulated task (word processing or assembly-like tasks)?
- Will some individuals respond similarly between the fatigue and control sessions? How do those individuals differ from persons who do alter their responses between the fatigue and control sessions?

It is hypothesized that:

1. Hip abductor fatigue will remove the bilateral gluteus medius cocontraction response present in pain developers (Nelson-Wong and Callaghan, 2010b).
2. The fatigue protocol will act as a hyperalgesic stimulus, increasing the severity of pain experienced by pain developers but not non-pain developers (Dedering et al., 2004; Ciubotariu et al., 2007).
3. The fatigue protocol will also cause non-pain developers to change their postural responses so that their postural responses match the pain developer responses (Janssens et al., 2010; Johanson et al., 2011).
4. Pain developers will show greater hip abductor fatigue over the course of the control session and have a slower recovery of fatigue during the fatigue session than those who do not develop pain (Nelson-Wong and Callaghan, 2010b).
5. The persons whose pain reporting patterns differ between the control and fatigue session, termed fatigue sensitive, will be identifiable by either a muscle activation parameter or a postural variable. That indicator will be different between the fatigue sensitive pain developer and the fatigue sensitive non-pain developer.

Section 2: LITERATURE REVIEW

This literature review is divided into four major sections: studies inducing pain in persons without any current pain (2.1), studies observing differences between the responses of those with and without chronic pain (2.2), studies determining the effects of muscle fatigue (2.3), and studies that examine interactions between muscle fatigue and pain (2.4). Following these major sections, there is a brief discussion on the function of the hip abductors (2.5), the use of the words pain versus discomfort in the context of prolonged standing protocols (2.6) and a list of key messages from within the literature review (2.7).

2.1 Pain Induced in Asymptomatic Persons

By taking an otherwise healthy, pain free sample from the population and manipulating their level of perceived pain, there is more confidence than when observing individuals with existing pain that any results found are related to the induced pain development and not as a response of coping mechanisms or strategies adopted after pain initiation. In practice, this has been accomplished by either: a) applying a standard mechanical exposure and differentiating those who do and do not develop pain, or b) injecting painful (4 to 7%) and non-painful (0.5%) levels of saline solutions to the same participants across different testing sessions. The injection methods tend to result in higher magnitudes of reported pain (severities of 30 to 50 out of 100 on a visual analog scale; Graven-Nielsen et al., 1997; Schulte et al., 2004; Henchoz et al., 2013) than the mechanical exposures (15 to 30 out of 100; Gregory and Callaghan, 2008; Nelson-Wong et al., 2008; Marshall et al., 2011; Gallagher et al., 2014). For comparison, those with chronic back pain often report pain levels of at least 35 out 100 when not in remission (Hägg et al., 2003; Henchoz et al., 2013). The two different models often report different specific findings, but both agree that inducing pain alters muscle recruitment patterns.

2.1.1 Pain from Standard Mechanical Exposures

Research involving standard mechanical exposures have often used a two hour, quasi-static standing protocol to induce low back pain. Advantages of using this method are as follows. First, participants clearly differentiate into pain developing (PD) and non-pain developing (NPD) groups over the course of the two hour protocol (Gregory and Callaghan, 2008). Second, those who are in the PD group in one session reliably stay within the PD group for repeated sessions (Nelson-Wong and Callaghan, 2010a). Third, the PD group is thought to be fairly homogenous in that PDs show similar responses across multiple studies (Nelson-Wong et al., 2008; Nelson-Wong et al., 2010; Marshall et al., 2011; Gallagher et al., 2011; Gallagher et al., 2013; Gallagher et al., 2014). Fourth, an active hip abduction test can predict whether a person is an NPD or a PD with moderate sensitivity (0.35-0.40) and high specificity (0.85-0.92) (Nelson-Wong et al., 2009). Lastly, those who are classified into the PD are more likely to develop chronic low back pain in the future (odds ratios of 1.82 to 3.33), making this method of pain induction highly valid (Nelson-Wong and Callaghan, 2014). Some drawbacks of this model are that the division of NPD and PD groups are entirely based on subjective measures, within-subject designs are not feasible as a person cannot be both in the NPD and the PD group, and that the magnitude of pain developed in some individuals over the 2 hours is lower than the magnitude of pain in those actually suffering from chronic low back pain. Although widely used and valid for clinical pain populations (Loeser and Melzack, 1999; Burton and Waddell, 2001), fear avoidance behaviour questionnaires do not differentiate PD and NPD groups (Nelson-Wong and Callaghan, 2010b), suggesting that the PD group is asymptomatic both in actual pain reporting and psychosocial risk factors for pain development.

Gluteus medius activity is one of the recurring differences between the PD and NPD groups. Initially, Gregory and Callaghan (2008) found that a person with a greater number of gaps in gluteus medius activity during the first 15 minutes of standing would develop higher levels of low back pain at the end of two hours, implying that the PD group showed less gluteus medius activity. This finding is peculiar; gaps as a measurement tool were initially developed such that a person with a greater number of gaps would have reduced levels of pain (Veiersted et al., 1990). However, the majority of the work since then has been relatively unanimous in finding that those in the PD group generally have shorter gap lengths, and higher levels of gluteus medius activity (Nelson-Wong et al., 2008; Nelson-Wong and Callaghan, 2010b; Marshall et al., 2011). Gluteus medius bilateral cocontraction is commonly found in PDs, and is hypothesized as a compensatory mechanism in an attempt to increase trunk stability (Nelson-Wong and Callaghan, 2010b). The ability of the active hip abduction test, a test that requires trunk musculature involvement to prevent undesirable movements or rotations, to differentiate pain development groups is believed to be based on the PD group having some deficit of "trunk control in the frontal plane" (Nelson-Wong et al., 2009, p. 654). Also, PDs have a delayed thoracic and lumbar level erector spinae onset time in response to a suddenly applied load, compared to NPDs (Gregory et al., 2008a). That same study also reported that PDs have a greater likelihood to activate gluteus medius in response to flexion loads than NPDs (Gregory et al., 2008a). These findings suggest a tendency for PDs to use gluteus medius in situations where other muscles of the trunk would be more appropriate (Nelson-Wong and Callaghan, 2010b).

Perhaps the strongest evidence that an inability to achieve trunk stability is a major factor in pain development during prolonged standing comes from an exercise intervention study (Nelson-Wong and Callaghan, 2010a). By implementing a progressive, four week exercise

protocol aimed at increasing the ability to achieve trunk stability, those initially in the PD group had a reduction of reported low back pain of 15 to 20 out of 100, enough of a reduction for them to no longer be classified as pain developers (Nelson-Wong and Callaghan, 2010a). For comparison, the control PD group had a non-significant reduction in back pain of 3 to 10 out of 100 (Nelson-Wong and Callaghan, 2010a). The pain developers in the control group reported similar levels of physical activity to the exercise group over the four-week interim period, but reported no reduction in low back pain (Nelson-Wong and Callaghan, 2010a). In addition, the males in the PD exercise group had reductions in gluteus medius cocontraction and an increase in gluteus medius gap length after the exercise intervention (Nelson-Wong and Callaghan, 2010a). These findings indicate that the initial gluteus medius responses were no longer necessary; the exercise intervention provided the PD group with the capacity to stabilize the lumbar spine and pelvis using trunk musculature instead of involving gluteus medius.

PDs also show postural differences from NPDs during a two hour period of standing. In both standing and sitting, PDs tend to utilize a smaller range of motion in the lumbar spine than NPDs (Gallagher et al., 2014). Those in the PD group have also been reported to have greater mean angles in trunk axial twist (Gregory and Callaghan, 2008), more lumbar spine extension (Sorensen et al., 2015), and greater thoracic spine flexion (Gallagher et al., 2014) than those in the NPD group. Male PDs have shown different responses in weight-support strategies based off of force plate measures. Compared to male NPDs, the male PDs spent less time in asymmetric postures in the first 30 minutes of standing, were highly variable in the number of shifts in limb support beyond the first 30 minutes of standing, had smaller magnitudes in centre of pressure migration in the anterior/posterior direction, and had fewer centre of pressure migrations in the medial/lateral direction (Gallagher et al., 2011). In essence, the spine kinematics indicate that PDs tend to be

more static than NPDs, while the force plate measures indicate PDs are more dynamic than NPDs. Although it appears as though there is conflicting evidence, it is possible that the changes in weight support could have occurred without altering spine angles by using the changes in hip, knee and ankle to transfer loads.

Sloped surfaces have been found to positively affect PDs, albeit through a different mechanism than the exercise intervention. In standing on a sloped surface (self-selected as to inclined - toes upwards, or declined - toes downwards), PDs showed a decrease in reported low back pain and gluteus medius cocontraction (Nelson-Wong and Callaghan, 2010c). The magnitude of both changes were similar to that of the exercise intervention (Nelson-Wong and Callaghan, 2010c). Interestingly, the NPD increased the level of gluteus medius cocontraction on a sloped surface compared to level standing (Nelson-Wong and Callaghan, 2010c). It is thought that the changes brought about by standing on a sloped surface are purely related to changes in posture as measures of muscle thickness in the abdominal wall were not altered by either an inclined or declined surface compared to normal standing (Gallagher et al., 2013). However that study (Gallagher et al., 2013) did not quantify muscle activity, so their conclusion is still tentative.

2.1.2 Pain from Injection of Hypertonic Saline

While pain in prolonged standing takes two hours to develop, pain from hypertonic saline injections peaks within minutes, and dissipates within two hours (Graven-Nielsen et al., 1997; Ciubotariu et al., 2007). Advantages of using an injection model for studying pain include: a) the ability to compare findings within-subjects, b) the ability to choose the site and tissue of pain with relative ease and c) the pain induced is localized to the injection site and tissue. The major disadvantage of using hypertonic saline injections is that this type of pain functionally differs from chronic pain. Pain from hypertonic saline injections is not episodic in nature (it peaks then steadily

declines) and the source of pain is a direct, local stimulation of nociceptor afferents (Graven-Nielsen et al., 1997). In contrast, chronic pain is often episodic (Balagué et al., 2012) and the sources of chronic pain are thought to be multifactorial (Adams et al., 1999; Kumar, 2001; see Section 2.2 for more details). These discrepancies limit the validity of the findings from studies employing this pain induction model (Williams et al., 2010).

Regardless of whether the pain is induced in the calf (Graven-Nielsen et al., 1997; Hodges et al., 2008), the biceps brachii (Schulte et al., 2004), the flexor pollicis longus (Tucker et al., 2010) or the femoral-patellar fat pad (Tucker et al., 2010), the injected muscles and those surrounding the injected tissues are incapable of producing the same levels of maximal force as they could prior to the injection. There is often some increase in muscular activity in a related, non-synergistic muscle in response to the pain injections as well. Biceps brachii injection increased trapezius activity during elbow flexion (Schulte et al., 2004), injection into the gastrocnemius resulted in greater tibialis anterior activity during walking (Graven-Nielsen et al., 1997), and tibialis anterior injections increased gastrocnemius activity during walking (Graven-Nielsen et al., 1997).

There is also evidence that motor unit recruitment is altered in the presence of hypertonic saline injections. In painful conditions, participants were observed to recruit a larger number of motor units for the same level of submaximal isometric force compared to non-painful conditions (Tucker et al., 2010). When the level of force production was increased (but still submaximal), roughly half of the motor units recorded during the previous submaximal contraction were absent, even though the absolute number of motor units recruited were found to be the same between the low level pain and high level non-pain contractions (Tucker et al., 2010). This finding held true for both the lower limb (injection into the femoral-patellar fat pad) and the upper limb (injection into flexor pollicis longus). Furthermore, the motor units recruited during the painful conditions

are believed to fire at a slower rate than the non-painful conditions, despite no differences in surface electromyography (EMG) amplitude (Hodges et al., 2008). These findings lead to the conclusion that injections of hypertonic saline results in the central command centres recruiting motor units against the principle of orderly recruitment proposed by Henneman and colleagues (1965).

The combination of non-orderly motor unit recruitment and cocontraction patterns in pain from hypertonic saline injections suggests that this type of pain functionally differs not just from chronic pain, but also the pain induced from prolonged standing exposures (Williams et al., 2010).

2.1.3 Synopsis of Induced Pain Literature

Attempts have been made to fit the different muscular and postural responses from induced pain into an overarching theory. Prior to the publication of the majority of research discussed above, it was thought that painful muscles naturally respond by increasing their activity level, which then results in more pain. The earliest formalization of this "vicious cycle" concept of muscle pain was made by Travell and colleagues in 1942 based off of observations on surgical procedures (Travell et al., 1942). However clinical findings had begun to disagree with the idea that pain in muscles increases their activity, leading to the proposition of the pain-adaptation model (Lund et al., 1991). In this model, a painful muscle will decrease its activity level, and increase activity in "antagonistic" muscles through central drive (Lund et al., 1991). Although many of the saline injection studies support this model (Graven-Nielsen et al., 1997; Schulte et al., 2004; Ciubotariu et al., 2007), the findings in other saline injection studies show contradictory results (Hodges et al., 2008; Tucker et al., 2010). Also, studies of induced pain through thermal stimuli (Henchoz et al., 2013), and prolonged standing (Nelson-Wong et al., 2008; Nelson-Wong and Callaghan 2010b) give contradictory results to the predictions of the pain-adaptation model.

It has been found that evidence for both the vicious cycle and pain adaptation models exists (Sterlin et al., 2001; van Dieën et al., 2003a). The authors of one of these reviews suggest that both models are inadequate for describing the muscle responses associated with pain and that a third model, based on the maintenance of joint stability, could explain the findings from both the existing studies of induced pain (this was prior to the prolonged standing induced pain studies) and the studies observing clinical pain (van Dieën et al., 2003a). The results of the exercise intervention study on PD and NPD groups in standing align with this theory as well. Upon gaining the ability to stabilize the lumbar spine through trunk musculature, male PDs did not showcase the typical gluteus medius bilateral cocontraction response or develop clinically significant low back pain while standing (Nelson-Wong and Callaghan, 2010a). Also, PDs tend to use more of a gluteal-based muscle pattern in response to a sudden, unexpected trunk flexion load where NPDs use a more erector spinae-based approach (Gregory et al., 2008a). These results suggest that the muscle adaptations of PDs are attempts to use gluteus medius to bolster the stabilizing effects of the other trunk muscles. However it is still unclear as to whether the muscle activity patterns of the PD group are in response to the pain, or are the source of the low back pain in standing. Additionally, it has yet to be determined if the bilateral gluteus medius cocontraction is only an indicator of other aberrant motor patterns, or is in part responsible for those motor patterns.

2.2 Persons with Pre-existing Pain

Studies that compare persons with chronic low back pain to healthy, matched controls are generally highly valid, but cannot infer causality with much confidence as observations are usually cross-sectional. The inter-study findings are quite dissonant owing to, in part, the large heterogeneity in the chronic low back pain population (O'Sullivan, 2005). The treatment that is often successful in one patient can be exacerbating in another because of the different mechanisms of injury, different tissues affected by the injury, different genetic predispositions, different muscle activation patterns and different personality characteristics (Adams et al., 1999; Waddell and Burton, 2001; Balagué et al., 2012). A comprehensive longitudinal study found that the best predictor of low back pain in an occupational setting with fairly uniform exposure levels consisted of a combination of patient history, psychological testing and physical capabilities, but could only account for 12% of all back pain cases reported (Adams et al., 1999). The single largest predictor of low back pain is having a history of prior low back pain (Biering-Sorensen, 1984; Adams et al., 1999; Waddell and Burton, 2001; Balagué et al., 2012).

2.2.1 Non-Specific Low Back Pain

The inability to predict and treat low back pain is seen through the use of the term "non-specific low back pain", usually referring to back pain without an identifiable cause (Balagué et al., 2012). This term is not very useful as it gives little information about the nature of pain, however non-specific back pain encapsulates the vast majority of all reported chronic low back pain cases (Balagué et al., 2012). There is evidence that all of those with non-specific low pain share some risk factors, suggesting some common traits. These common traits include: inadequate rest time in occupational exposures (Veiersted et al., 1990; Waddell and Burton, 2001; Heneweer et al., 2009; Tissot et al., 2009; Gallagher and Heberger, 2013), low levels of physical activity

(Kumar, 2001; Waddell and Burton, 2001; Heneweer et al., 2009; Balagué et al., 2012), a greater likelihood of having negative social interactions and high levels of mental stress (Adams et al., 1999; Sterling et al., 2001; Waddell and Burton, 2001), an inability to reliably predict or identify tissue damage with medical imaging (van Tulder et al., 1997; Waddell and Burton, 2001; Balagué et al., 2012), non-responsiveness to "novelty treatments" such as back belts (Waddell and Burton, 2001; Balagué et al., 2012), and that drug treatments for pain tend to work no better than placebos (Finniss et al., 2010; Balagué et al., 2012). Also, those who tend to catastrophize their injuries are at an increased risk to become functionally disabled by their pain, lose time at work due to symptoms and are less likely to recover (Waddell and Burton, 2001; Eriksen et al., 2004; Atlas and Wager, 2012). It should be noted that all these traits are common to the majority of chronic pain sufferers regardless of pain location (Loeser and Melzack, 1999; Eriksen et al., 2004).

When patients are separated or sub-classified into groups based on what movements exacerbate their symptoms, the inter-study findings become more consonant. For those with flexion-related back pain, persons for whom spine flexion worsens pain, the differences separating patients from healthy controls are mainly seen in sitting, a posture of greater spine flexion. Specifically, these persons tend to naturally sit more towards their end range of motion (O'Sullivan et al., 2006; van Hoof et al., 2012), have less back extensor endurance (O'Sullivan et al., 2006), have a reduced ability to finely control the activation levels of their back extensors (O'Sullivan et al., 2013a; O'Sullivan et al., 2013b), have higher levels of trunk cocontraction (D'hooge et al., 2013), and have their pain symptoms alleviated in more lordotic postures (O'Keeffe et al., 2013; O'Sullivan et al., 2013a). Sufferers with flexion-related back pain also do not demonstrate the flexion-relaxation phenomenon - the reduction of trunk extensor activity near the mid to end ranges of spine flexion (Geisser et al., 2005).

The findings in those with flexion-related low back pain are consistent with the theory that damaged and painful tissues in the back cannot contribute to intervertebral joint stability during spine flexion (McGill et al., 2003; van Dieën et al., 2003b). Therefore, additional muscle activity is required in order to compensate for the failures of the damaged tissues (Cholewicki and McGill, 1996). For example, those with flexion-related back pain could have higher levels of back extensor activity and exhibit more cocontraction in sitting than healthy controls to increase the compressive force acting on the spine (D'hooge et al., 2013). Healthy controls are able to rely on ligamentous compression while sitting as the posterior ligaments become taut from the spine flexion (Solomonow, 2004). Since muscle activity is not required to compress the intervertebral joints, the back extensor musculature is relatively inactive while sitting, leading to the seated flexion-relaxation phenomenon (Callaghan and Dunk, 2002). Likewise, the postural compensations of those with flexion-related back pain tend to reduce the moment arms of the back extensors (McGill and Norman, 1986; Cholewicki and McGill, 1996), reducing the moment generating capacities of these muscle while resulting in greater amounts of compression for a given magnitude of moment (Cholewicki and McGill, 1996). Both these compensations tend to reduce to likelihood of joint instability where muscle activity attempts to accommodate the lack of passive contributions to stability (Reeves et al., 2007). Findings that when in pain, there is a tendency to alter the recruitment of muscle groups en masse rather than a specific muscle, also supports the concept that muscles need to maintain joint stability in the presence of pain signaled from passive tissue damage (Nelson-Wong et al., 2013; Falla et al., 2014).

Classification systems that are based on functional differences between sub groups with low back pain have higher success rates in patient outcomes than more generic treatments (Fairbank et al., 2011; Foster et al., 2011). Hall and colleagues (2009) propose an inclusive four

pattern classification, which differentiates the location and exacerbating factors of back pain. The flexion-related back pain group identified by O'Sullivan and colleagues (2006) corresponds to one of the four patterns in this sub-classification system. In comparing treatment groups using a generic and classification-based treatment system, patients who were sub grouped into these four patterns were 2.3 to 10.9 times more likely to be pain free after 18 months, and 2.0 to 4.1 times more likely to not require medications for pain relief (Hall et al., 2009). An alternative classification approach differentiates patients who have hypermobile backs (stabilization group) from those whose backs are excessively stiff and rigid (manipulation group), from those exhibiting pain with specific motion tasks (specific exercise group), from those with nerve impingement (traction group) (Fritz et al., 2007). Patients classified into these sub groupings also had better outcomes after a four month treatment period as evidenced by reductions in the Oswestry Disability Scores and further progression into treatment protocols (Brennan et al., 2006). Although portions of these different classification systems may not agree with each other, all operate on the rationale that the manner in which symptoms are moderated by orthopedic and mechanical testing should dictate diagnosis and treatments (Fairbank et al., 2011). It is therefore more effective to treat individuals with non-specific back pain on a case by case basis rather than using global algorithms (McGill, 2007).

Pain symptoms are often assessed by use of provocative clinical testing and self-reported levels of severity and disability (Deyo et al., 1998; Ostelo et al., 2008). Studies on the reliability of clinical testing often conclude that the tests used to diagnose and classify low back pain patients on their own have poor to moderate inter-rater reliability (McCarthy et al., 2007; Balagué et al., 2012; Simopoulos et al., 2012). However the inter-rater reliability in patient classification is quite high when multiple clinicians are trained by a common source (van Dillen et al., 1998; Paatelma

et al., 2009). Clinicians can also employ a battery of tests to compensate for the less than desirable sensitivity and specificity of a single test (van Dillen et al., 1998; Balagué et al., 2012).

In assessing patient outcomes, visual analog scales, numerical rating scales, the Roland disability questionnaire, Oswestry disability index, the Quebec back pain questionnaire and the short-form of the Medical Outcomes Study are all widely used and have fairly reliable inter-session reporting (Deyo et al., 1998; Ostelo et al., 2008). Visual analog scales are among the easiest to implement, and have strong support in favour of their ability to distinguish the severity of pain symptoms (Price et al., 1983; Kelly, 1998, 2001; Ostelo et al., 2008). Both the Oswestry disability index and the short-form questionnaire scores are capable of identifying those at risk for, and separating severity levels within those with chronic low back pain (Deyo et al., 1998; Adams et al., 1999; Ostelo et al., 2008). The level of success of a treatment for low back pain is thought to depend on six domains: reduction in pain symptoms, an increase in function, an increase in well-being, absence of physical disability, absence of social disability and an overall satisfaction with the care received (Deyo et al., 1998). The severity of the pain (often assessed by a visual analog scale or a numerical rating scale), is only one aspect. Function and well-being are assessed by the short-form questionnaire (Deyo et al., 1998), physical and mental disability are assessed by the Oswestry disability index and Roland disability questionnaire (Ostelo et al., 2008), and patient satisfaction is obtainable from the patient themselves.

2.2.2 Occupational Demands and Low Back Pain

The link between occupational demands and worker health was noted as early as 1700 by Bernardino Ramazzini, (in Felton, 1997). More contemporary publications have shown that occupational requirements are related (Kumar, 2001; Waddell and Burton, 2001; Lis et al., 2007; Tissot et al., 2009; Gallagher and Herberger, 2012) or are not related (Roffey et al., 2010a; Roffey

et al., 2010b; Wai et al., 2010) to the development of low back pain. There is a stark difference between these groups of publications; the publications reporting null findings often study occupations with relatively lower exposure levels than the publications that reported positive findings. For instance, sitting was not related to back pain in a review that examined occupations that did not involve prolonged periods (more than 2 hours continuously) of sitting (Roffey et al., 2010a). However, in a review examining occupations that contained both prolonged seated exposures (more than 4 hours) and intermittent seated exposures, those in occupations with prolonged seated exposures were more likely to develop back pain than those with only intermittent exposures (Lis et al., 2007). Likewise, a review (Roffey et al., 2010b) concluded standing at work was unrelated to back pain, but did not incorporate occupations with prolonged, static standing postures. A review that did conclude that standing and back pain were related incorporated and classified occupations with prolonged, static standing exposures (Tissot et al., 2009). In both cases, it is clear that sitting or standing are not inherently risk factors for developing low back pain, but become risk factors when maintained over long periods of time without ample opportunity for variation in posture (Callaghan and McGill, 2001).

Prolonged standing has been reported to pose different health detriments to the worker dependent on the occupation. Dentists who perform more procedures while standing have a greater risk of developing foot and knee pain (Kierklo et al., 2011), while poultry processing workers who stand for longer periods of time develop greater levels of foot and low back pain (van Dieën and Oude Vrielink, 1998). One variable that may account for these differences is the absolute exposure level. Studies using static standing to induce pain (see Section 2.1.1), have reported sooner onsets of foot and knee pain than that of back pain (Antle et al., 2013). A large scale study of Danish workers reported a dose-response effect where those who stood for a larger percentage of the

workday had a higher risk of developing low back pain, with a stronger association between those working more than 37 hours per week than in those working fewer hours per week (Xu et al., 1997). Differences in exposure levels can influence what symptoms are developed from standing, with longer exposures having a greater likelihood to result in lower back symptoms than shorter exposures.

2.2.3 Synopsis of Those with Pre-existing Conditions

In one respect, chronic low back pain is similar to cancer: it is most fruitful to study a specific type than the research area as a whole. There are over 200 different types of cancers, each one has its own separate symptoms, diagnostic criteria, prognoses and treatments. Likewise, if and when a cure is developed, it is not likely to be a single cure for all cancers, but rather a specific cure corresponding to a specific diagnosis of cancer. Since, like the cancer patient population, the population with chronic low back pain is highly diverse, the treatments and preventions for low back pain should therefore be approached in a similarly diverse manner. Different mechanical exposures in persons with different predispositions will lead to different disorders (Kumar, 2001); the major confounding factor is that all of those with low back pain have a common, dominating symptom. However, it is clear from the scientific literature that the underlying factors and preventative measures specific to many back pain cases are still unknown. In order to best complete these gaps in knowledge, samples of low back pain patients should be made as homogenous as possible in how the pain was developed, and whom developed that pain. The low back pain literature has begun to recognize the need to have homogenous samples of patients and steps have been taken to isolate these homogenous groups as best as possible by using functionally relevant classification systems (Fairbank et al., 2011; Foster et al., 2011).

2.3 What is Muscular Fatigue?

There are numerous definitions of muscular fatigue. Most definitions label fatigue as a decline in a muscle's force production that a) is not caused by injury, b) occurs following measureable muscle use, c) occurs in spite of increasing effort and d) is only temporary. Things that muscle fatigue is not, include: the ability to perform a task (Enoka and Duchateau, 2008), general weakness (González-Izal et al., 2012), and decreased mental alertness. Muscle fatigue is usually further divided into central fatigue - the ability of the nervous system to voluntarily activate the muscle, and peripheral fatigue - the muscle's capacity to generate force in response to stimulation (Gandevia, 2001). The specific cause of muscle fatigue is thought to be dependent on the task being performed as there are many potential contributors to the development and onset of fatigue (Allen et al., 2008; Enoka and Duchateau, 2008; Yung et al., 2012).

Fatigue is a fairly easy phenomenon to observe because a myriad of physical and measureable biological changes accompany decreases in external force. In fatigue compared to a resting state, there are elevated levels of calcium, hydrogen, inorganic phosphate, dihydrogen phosphate (H_2PO_4) and creatine ions (Miller et al., 1988; Westerblad et al., 2000; Allen et al., 2008), and decreased levels of phosphocreatine and potassium ions (Allen et al., 2008) inside the muscle. The neural stimulation of a fatigued muscle has a greater voltage amplitude, occurs at lower frequencies and travels to the muscle at slower conduction velocities than in resting muscles (Merton, 1953; Petrofsky, 1979; Bigland-Ritchie and Woods, 1984; Potvin and Norman, 1993; Dolan et al., 1995). There is also evidence that changes in cortical electrical activity (Gandevia, 2001), reductions in local proprioception (Boucher et al., 2012; Paillard, 2012), an earlier onset of the flexion relaxation phenomenon (in trunk muscle fatigue only - Descarreaux et al., 2010), a synchronization of action potentials (Bigland-Ritchie et al., 1981; De Luca et al., 1993), an

increased risk of injury (Gregory et al., 2008b), and broad scale recruitment of adjacent (Grondin and Potvin, 2009) and non-adjacent muscles (Gandevia, 2001) are all consequences of muscle fatigue. In addition, a model that simply assumes that non-fatigued fibres can produce force and fatigued fibres cannot results in very accurate gross muscle force predictions (Xia and Frey Law, 2008; Frey Law et al., 2012), even though it is clear that fatigued fibres *in-vivo* still have some force generating capacity (Bigland-Ritchie and Woods, 1984; Allen et al., 2008).

2.3.1 Cellular Changes with Fatigue

The changes in ion concentrations from fatigue are thought to result from either a) an inability of the circulatory system to meet the demands of the working muscles or b) an inability of the muscle fibre to regenerate the molecular inputs needed for its metabolic pathways (Allen et al., 2008). Generally, larger changes in metabolite concentrations occur in muscle fibres that fatigue more easily (Allen et al., 2008). Brief fatigue (less than 2 to 3 minutes) usually occurs from a failure of the anaerobic metabolic system to regenerate its molecular inputs and force is usually recovered relatively quickly (Skof and Strojnik, 2006a, 2008). Fatigue from longer exertions can possibly be caused by excessively high core body temperatures, muscle hypoxia, glycogen depletion, malfunctioning membrane voltage sensors, excessively high pulmonary blood pressure or an accumulation of inorganic phosphate (Gandevia, 2001; Allen et al., 2008). A specific source has not been linked to a specific method of fatigue, however any combination of these factors may play a role in a given fatiguing bout (Enoka and Duchateau, 2008).

Calcium ions are the chemical link between electrical excitation of a muscle and its force production (Tupling, 2004). Normally, calcium is stored within the sarcoplasmic reticulum, is released into the sarcoplasm upon electrical activation and returns into the sarcoplasmic reticulum upon relaxation. In a fatigued state, both calcium release from and reuptake into the sarcoplasmic

reticulum are impaired, resulting in lower force generation and slower relaxation times (Westerblad et al., 2000, Allen et al., 2008).

The increases in hydrogen ions will lower the pH within the muscle. The magnitude of the pH decrease that occurs *in-vivo* does not appear to alter the force levels attained, but does decrease the contraction velocity of the fibre (Nelson and Fitts, 2014). Inorganic phosphate is currently thought to play a much larger role than hydrogen, with evidence that the increases in concentration reduces the ability of cross-bridges to be strongly bound to each other, and reduces the sensitivity of the sarcomere to calcium ions (Westerblad et al., 2002; Allen and Trajanovska, 2012). Both of these changes would result in less force generated for a given level of neural activation. Repeated muscle fibre action potentials without adequate rest decreases the intracellular potassium concentration (Blalog and Fitts, 1996), which in turn reduces the conduction velocity and firing rate of the muscle fibre action potentials (Fortune and Lowry, 2009). Glycogen depletion not only limits the available "fuel" of the muscle, but also seems to limit calcium release from the sarcoplasmic reticulum; reducing the capacity to perform work and the efficiency with which that work is performed (Ørtenblad et al., 2011). The effects of increased H_2PO_4 are not known, however its concentration tracks accurately over time with both the decline in force from fatigue, and the subsequent recovery of force over time (Miller et al., 1988). In spite of all the molecular changes, concentrations of ATP remain constant throughout fatigue (Miller et al., 1988). It has been hypothesized that muscle fatigue may be an attempt to prevent the complete exhaustion of ATP (Allen et al., 2008).

2.3.2 Myoelectric Changes with Fatigue

EMG amplitudes, reported as a mean or peak absolute value, root mean square, or linear enveloped (absolute value and low pass filtered), are often found to be increased in fatigue during

sustained submaximal contractions (Merton, 1953; Petrofsky, 1979, Arendt-Nielsen et al., 1989). It was hypothesized that the increase in amplitude for a submaximal effort reflects either an increase in motor unit recruitment or motor unit firing rate to perform the same workload (Arendt-Nielsen et al., 1989; González-Izal et al., 2012). However, the signal amplitude has also been found to remain constant (Rosendal et al., 2004; Movahed et al., 2011) or decrease (Hostens and Ramen, 2005; Ciubotariu et al., 2007) in fatiguing scenarios utilizing constant, submaximal force outputs over time. Dimitrova and Dimitrov (2003) warned against the use of amplitude changes to detect fatigue as the action potential duration, propagation velocity, negative after potentials and its relative distance from the electrode can interact to alter the amplitude of recorded electrical activity. The filter cutoffs implemented can also affect the amplitude of the myoelectric signal. In comparing band pass filters with cutoff ranges of 20 to 500 Hz, 10 to 500 Hz and 1 to 500 Hz, it was found that implementing the 20 to 500 Hz band pass filter changed the polarity of the after potential from negative to positive (Dimitrova and Dimitrov, 2003). An unintentional consequence of this polarity shift in the 20 to 500 Hz band pass filter compared to the other cutoff ranges is that the summation of individual motor unit action potentials (the recorded EMG amplitude) would be altered (Dimitrova and Dimitrov, 2003). Since the magnitude of the after potential increases in greater levels of fatigue due to a slower restoration of the resting membrane potential (Blalog and Fitts, 1996), this change in polarity would have a more dramatic effect in higher levels of fatigue.

Contrary to the variability in amplitude changes with fatigue, the frequency or spectral content of recorded EMG has been consistently shown to shift to lower frequencies with fatigue. The mean (Arendt-Nielsen et al., 1989; Potvin and Norman, 1993), median (Kim et al., 1994; De Luca, 1997; Dedering et al., 2004) and centre values (Petrofsky, 1979) of the power spectrum have all been found to be reliable indicators of the frequency content of EMG within a testing session.

The shift to lower frequencies has been nearly universally attributed to the slower action potential conduction velocities as the fibres fatigue (Bigland-Ritchie et al., 1984; Gandevia, 2001; Dimitrova and Dimitrov, 2003; Allen et al., 2008; González-Izal et al., 2012). Other potential sources of the frequency shifts include either a selective drop out of larger diameter, more fatigable motor units (Seki and Narusawa, 1998), or a slower firing rate of motor unit action potentials (Milner-Brown et al., 1973; Milner-Brown and Miller, 1986). The inability to restore the intracellular potassium ion concentration would result in action potentials that both propagated more slowly and required a greater refractory period (Allen et al., 2008). The reduction in action potential amplitude with fatigue ("dropping out") is believed to originate with slowed calcium reuptake into the sarcoplasmic reticulum and the net potassium efflux (Westerblad et al., 2000; Tupling, 2004). Therefore, the reduction in frequency content of a muscle reflects the decreased capacity of the muscle to produce force when fatigued.

2.3.3 Low Level Fatigue

Low level fatigue is not the same thing as low frequency fatigue, although the two are not mutually exclusive and can often coincide (Allen et al., 2008). Low frequency fatigue refers to a selective reduction in force at low frequencies of electrical stimulation that is indicative of long lasting soreness and structural disruption of the muscle fibre (Allen et al., 2008). Low level fatigue refers to fatigue occurring from sustained exposure to a low level of voluntary muscle activation (de Looze et al., 2009).

There have been difficulties encountered in identifying low level fatigue as it requires longer times to develop and the magnitude of changes are smaller in comparison to traditional fatigue (fatigue from higher levels of muscle activity). Electromyographic (de Looze et al., 2009) and blood flow measures (van Dieën et al., 2009) differ from those reported in traditional fatigue,

and there is not a clearly established point at when low level fatigue is expected to occur (Hostens and Ramon, 2005; de Looze et al., 2009). As traditional EMG amplitude variables (e.g. mean or peak absolute values) are not sensitive enough to detect low level fatigue, alternative processing techniques have been developed to quantify this type of fatigue. Counting the number and duration of the gaps in EMG activity has been shown as one effective measure of low level fatigue, where a gap is defined as a drop below 0.5% maximum voluntary isometric contraction (MVIC) for at least 0.2 seconds (Veiersted et al., 1990). Amplitude probability distribution functions have also been successfully employed in using EMG to detect fatigue from these exposures, where if a person is found to spend less than 10% of the time during an 8 hour work shift at greater than 2% to 5 %MVIC, they are at risk for muscle injury due to fatigue (Jonsson, 1978). Thresholds for 50% (less than 10 to 14 %MVIC) and 90% (less than 50 to 70 %MVIC) of the work day also have been utilized to identify the occurrence of fatigue (Jonsson, 1978). Quantifying the frequency and the length of sustained low-level muscle activations (a contraction of at least 0.5 %MVIC for at least 1.6 seconds; a functional opposite of a gap) has also been shown as an effective means to determine fatigue occurring over prolonged exposures (Østensvik et al., 2009). Reductions in the mean frequency of EMG power spectra have been found to be a fairly reliable indicator of low level fatigue in addition to the aforementioned amplitude domain techniques (de Looze et al., 2009; van Dieën et al., 2009).

Low level fatigue has paradoxically been found to result in twitch potentiation (Johnson et al., 2013). The explanation put forth by the authors is that normally in repeated muscle use, the effects of fatigue (decline in force) and potentiation (increase in force) occur simultaneously. At moderate to high levels of activation however, the effects of potentiation are usually only seen in the early stages of use, while fatigue effects dominate later in use. However in low level fatigue,

the magnitude of force decrease is much smaller than in traditional cases of fatigue, re weighting the sum of potentiation and fatigue to result in an increase in force for a given level of activation (Johnson et al., 2013). Although the outcome of this study does not fit with the current definition of fatigue as force outputs increased with subjective reports of muscle weakness, the relatively large effect of potentiation on force development due to the very low muscle activation levels partially explain the greater sensitivity required to detect EMG amplitude changes with low level fatigue.

2.3.4 Recovery from Fatigue

To determine whether a person has recovered from muscle fatigue, studies will measure how long it takes for the metabolic, myoelectric or force indicators of fatigue to return to their pre-fatigue values. The specific measure used to track recovery is not consistent between studies, however there are fairly reliable differences in the relative rates of recovery between variables for the same fatiguing exposure (Lind, 1959; Petrofsky, 1979; Baker et al., 1993; Skof and Strojnik, 2006a, 2006b; Yung et al., 2012). In general, myoelectric indicators of fatigue, such as shifts in frequency content, return to resting levels sooner than metabolic indicators of fatigue, such as blood lactate or hydrogen ion concentrations (Petrofsky, 1979; Oksa et al., 2013). Recovery of force tends to occur in between spectral content and metabolite recovery (Petrofsky, 1979; Baker et al., 1993; Albert et al., 2006), however inorganic phosphate and H_2PO_4 concentrations within the muscle itself have been found to recover at the same rate as force (Miller et al., 1988). Self-perceived recovery (e.g. Borg scale ratings of perceived exertion) also differs from physiological measures of recovery. In longer fatiguing protocols (> 10 minutes to fatigue), participants often report that they “feel recovered” before maximal force capacity, blood plasma ion concentrations or EMG indicators return to resting values (Kumar et al., 2000; Leyk et al., 2006; Kimura et al.,

2007). The opposite has been found in one of the studies employing shorter fatigue protocols (Pereira and Goncalves, 2008). However self-reported ratings of perceived exertions are often only used to estimate how effective protocols were in producing fatigue in the absence of direct force measurements; other more objective measures are usually favoured in tracking fatigue recovery.

Despite these general tendencies in the recovery of variables relative to each other, there is still a considerable lack of consistency in how long of a recovery period is necessary. The decrease of EMG frequency content, one of the more reliable fatigue indicators, has been shown to require 30 seconds, (Pereira and Goncalves, 2008), 3 minutes (Petrofsky, 1979), 30 minutes (Jensen et al., 2000), 55 minutes (Kimura et al., 2007) or 48 hours (Pexioto et al., 2010) to recover back to resting levels for similar levels of voluntary exhaustion (rating of perceived exertions of 8-9.5 out of 10). Likewise, the amount of time required to recover voluntary, isometric force back to resting levels ranges from within minutes (Toubekis et al., 2008; Pereira and Goncalves, 2010), to at least an hour or two (Lind, 1959; Oksa et al., 2013) and in one instance, 48 hours of recovery was insufficient for force recovery in the wrist and finger flexors (Leyk et al., 2006). One potential explanation for these differences in recovery times for the same variables is that there are different causes of fatigue for the different tasks used to induce fatigue (Enoka and Duchateau, 2008; Yung et al., 2012).

In other cases, the time allotted for recovery to occur was insufficient for factors to return to baseline levels. Fifty five minutes of recovery following 2 hours of seated typing was deemed to be too short to alleviate trapezius and deltoid fatigue as conduction velocity and root mean square EMG amplitude measures had yet to return to baseline values (Kimura et al., 2007). Also, a thirty minute shoulder abduction fatigue protocol required longer than thirty minutes to allow for the recovery of maximal abduction force, mean power frequency and root mean square EMG

amplitude (Jensen et al., 2000). Tasks that take longer to induce fatigue appear to require longer recovery periods; fatigue recovers sooner in shorter, higher level exertions than in longer, lower level exertions (Soo et al., 2012). For a repetitive lifting protocol at roughly 33 %MVIC, the time required to recover was twice the time taken to fatigue the individual (Kumar et al., 2000). A fatigue modelling approach suggests that the time for a given muscle fibre to recover is five times the time taken to fatigue to fibre across activation levels (Xia and Frey Law, 2008). An example relating the time to fatigue to recovery times comes from a study (Baker et al., 1993) comparing a high-intensity short duration fatigue protocol (time to fatigue was ~ 2 minutes) and an intermittent, lower intensity fatigue protocol (time to fatigue was 15 to 20 minutes). There were similar changes in EMG and metabolic fatigue indicators immediately upon cessation of the two fatigue protocols. The recovery of those variables from the shorter protocol was adequately captured in a 15 minute recovery window, but the same recovery window was not long enough to track complete recovery from the long duration fatigue protocol (Baker et al., 1993).

The influence of variations in work load on the development and recovery of fatigue appears to be dependent on the absolute levels of work being performed. At lower levels of activity (~ 15 %MVIC), more variable work tends to reduce the level of muscle fatigue and the time required for recovery from fatigue, especially so if there are periods of rest within that activity (Yung et al., 2012; Yung and Wells, 2012). The amount of strength lost from an exposure of fixed length is proportionate to the work/rest ratios, and in equivalent work/rest ratios, those with a shorter total duty cycle result in less fatigue and faster recovery (Paquet and Nirmale, 2004). However at higher levels of activity (~ 70% maximal aerobic power - note that maximal aerobic power is not the same as MVIC), more variable work rates resulted in greater subjective and objective measures of muscle fatigue (Theurel and Lepers, 2008). In this instance, the variable

fatigue protocol involved no rest periods; the workloads alternated between 50%, 100%, 150% and 200% maximal aerobic capacity. This may be one potential explanation for the differences in variation and activity levels as rest periods are believed to play a major role in how workload variation prevents fatigue (Paquet and Nirmale, 2004; Yung et al., 2012).

Additionally, the presence of a mental task also reduced the time required to reach a given level of muscle fatigue, but not the recovery from that fatigue (Mehta and Agnew, 2012). It was hypothesized that the mental task would specifically induce additional central fatigue as changes in cortical activity have been noted in fatigued individuals (Gandevia, 2001).

In most cases, recovery from muscle fatigue is tracked while participants are at rest, termed passive recovery. Active recovery refers to performing a relatively lower level, non-zero exertion while recovering from fatigue. It was initially thought that active recovery was more efficient than passive recovery because active recovery would allow for a better washing out of metabolites from the fatigued muscle (Stamford et al., 1981). Studies have been fairly unanimous in that active recovery does quicken the recovery of blood lactate concentrations compared to passive recovery (Stamford et al., 1981; Hildebrandt et al., 1992; Toubekis et al., 2008). However, active recovery does not appear to help in recovering the losses of muscle force (Mika et al., 2007; Jougla et al., 2010). Studies comparing task performance following active and passive recovery from a common fatiguing protocol have found that active recovery does not alter (Toubekis et al., 2008) or worsens performance (Jougla et al., 2010) compared to passive recovery. Also, if the recovery activity uses different muscles than the fatiguing activity, active recovery will result in vasoconstriction around the fatigued muscles in an attempt to redirect blood flow towards the unfatigued muscles currently being used (Hildebrandt et al., 1992). The vasoconstriction about the fatigued muscles occurs even if the active recovery activity is perceived as “light and pleasant” by the participants (Hildebrandt

et al., 1992). This scenario effectively nullifies any theoretical benefits of employing an active recovery as its purpose is to increase blood flow to the fatigued muscle (Stamford et al., 1981).

The time varying course of fatigue recovery is exponential, with the majority of recovery occurring shortly after the fatiguing exertion has stopped (Lind, 1959; Dedering et al., 2004; Soo et al., 2012). Physiologically, the initial fast recovery corresponds to the replenishment of metabolic pathway inputs (e.g. creatine phosphate, glucose), restoration of potassium ion concentrations (which is linked to the recovery of EMG spectral content) and adequate reuptake of calcium into the sarcoplasmic reticulum (Allen et al., 2008; Fortune and Lowry, 2009). The slower, longer duration recovery of force, usually only present in longer fatigue protocols, corresponds in part to a reduced sensitivity to calcium induced from the high levels of inorganic phosphate (Westerblad et al., 2000; Allen and Trajanovska, 2012). Tissue damage may also be a source of longer lasting fatigue, but this is usually only a factor in fatigue from eccentric contractions (Iguchi and Shields, 2010; Sakamoto et al., 2010).

2.3.5 Fatigue and Stability

In a discussion involving characteristics of fatigue and pain development, the term stability can refer to one of three things. First, stability can refer to the resistance of a joint to become dislocated upon being perturbed, often labelled as joint stability. This reference is related to the ratio of compression to shear forces experienced in that joint, the level of muscular cocontraction about that joint and the stiffness of the structures spanning that joint (Cholewicki and McGill, 1996; McGill et al., 2003). Secondly, stability can refer to the kinematic variability of a repeated movement, often labelled as dynamic stability. This reference is related to whether the postures of a repeated motion tend to converge towards a common underlying motion pattern, or transiently shift between different patterns (Granata and Gottipatti, 2008). Lastly, stability can refer to whole

body balance responses to perturbation, which can be labelled whole body stability. This reference is related to the interactions between the centre of pressure and centre of mass in upright standing and sitting (Paillard, 2012).

For joint stability, studies comparing muscular responses to perturbations have often concluded that fatigue decreases joint stability based on higher levels of observed cocontraction post-fatigue (Psek and Cafarelli, 1993; Granata et al., 2001; Chappell et al., 2005; Herrmann et al., 2006; Ortiz et al., 2010; Kellis et al., 2011). This cocontraction response to fatigue has been found across different body segments. Trunk musculature cocontraction increases in preparation and in response to suddenly applied hand loads (Granata et al., 2001; Grondin and Potvin, 2009); quadriceps and hamstring cocontraction also increases just prior to and in the initial phases of landing from jumping (Ortiz et al., 2010; Kellis et al., 2011). An alternative conclusion for the increases in cocontraction is that muscle fatigue decreases the amount of stiffness produced for a given level of activation. Since muscles produce both force and stiffness upon contraction (Cholewicki and McGill, 1996), it is reasonable that the mechanisms which reduce a muscle's force upon contraction can also reduce its stiffness. There is one instance where a loss of muscle stiffness has been measured after a fatigue protocol. A study employing a swinging arm to knock participants anteriorly, measured more compliant erector spinae muscles in participants when they were fatigued; impacts of the same impulse had lower peak forces registered by the swinging arm post-fatigue (Herrmann et al., 2006). However, greater amounts of anterior shear forces in the knee have also been measured in jumping tasks upon fatigue (Chappell et al., 2005); suggesting that losses of stiffness, increases in destabilizing forces or a combination of both can act to decrease joint stability in a fatigued state.

For dynamic stability, it has been found that repetitive movements tend to become more variable upon fatigue (Granata and Gottipatti, 2008; Gates and Dingwell, 2011). In both repetitive lifting and sawing tasks, the standard deviations of joint angles as well as the maximum Lyapunov exponents during unrestricted motions with fixed end targets increased with fatigue (Granata and Gottipatti, 2008; Gates and Dingwell, 2011). The increasing kinematic variability has been hypothesized to be indicative of a loss of neuromuscular control as a result of muscle fatigue (Paillard, 2012). Also, studies on whole body stability and fatigue conclude that fatigue tends to reduce the participant's neuromuscular control, based on higher centre of pressure velocities and sway areas in fatigued states (Yaggie and Armstrong, 2004; Fox et al., 2008).

An earlier onset of the flexion relaxation phenomenon in trunk muscle fatigue provides further evidence that fatigue results in a loss of neuromuscular control (Descarreaux et al., 2010). It is believed that the myoelectric silence of the trunk extensors in mid to high levels of spine flexion, the flexion relaxation phenomenon, represents the transfer of load from the musculature to the ligaments (Callaghan and Dunk, 2002). The changes in the timing of the flexion relaxation phenomenon were related to the ratio of lumbar spine to hip flexion (Descarreaux et al., 2010). This deviation of relative spine and hip contributions during forward flexion could reflect that the increases in tissue compliance while fatigued are only partially accounted for by changes neuromuscular control (Boucher et al., 2012); more so in times when increased cocontraction is undesirable such as forward flexion (McGill et al., 2003).

For all three instances of "stability", muscle fatigue can increase the risk of injury. For joint stability, the loss of stiffness reduces the amount of shear force required to cause unwanted joint translations. Also, the loss of neuromuscular control can result in "instability slips", where an

inappropriate sequence of muscle activation can produce a weak spot in which an injury can occur from overloading (Cholewicki and McGill, 1992; McGill et al., 2003).

2.3.6 Personal Factors and Fatigue Development

Two personal factors associated with differences in fatigability are age and gender. Although not a universal finding (Senefeld et al., 2013), females tend to be more fatigue resistant than males (O'Brien and Potvin, 1997; Hunter and Enoka, 2001; Albert et al., 2006). This gender difference is thought to relate to either females having more fatigue resistant fibres or to females having slower changes in mean arterial pressure than males during the course of the fatiguing protocols (Hunter and Enoka, 2001). Additional support for the differences in blood pressure explaining gender differences come from findings that occluding blood flow during fatiguing contractions removed gender differences in endurance times (Enoka and Duchateau, 2008). Older adults are generally more fatigue resistant than younger adults in sustained isometric and lower velocity contractions (Enoka and Duchateau, 2008; Avin and Frey Law, 2011), however older adults are less fatigue resistant than younger adults in higher velocity contractions (Dalton et al., 2010). These findings are in line with observations that older adults can generate more muscular power at lower velocity contractions whereas younger adults generate maximal muscle power at higher velocity contractions (Dalton et al., 2014). These age differences have been attributed to a shifting towards slower contracting, more fatigue resistant muscle fibres with age (Monemi et al., 1999; Lee et al., 2006).

2.4 Fatigue and Pain

The presence of pain results in an earlier onset of muscle fatigue, and lengthens the time required for local fatigue recovery when induced via hypertonic saline injections (Ciubotariu et al., 2004, 2007) or occurring naturally from tissue damage (Kankaanpää et al., 1998; Dedering et al., 2004; Johanson et al., 2011). The earlier fatigue onsets have been reported if the muscle being fatigued is a non-painful synergist or antagonist to the painful muscle (Kankaanpää et al., 1998; Ciubotariu et al., 2004), but not if the fatigued muscle is unrelated to the painful muscle's function (Janssens et al., 2010). It has been suggested that the pain itself can limit force production rather than one of the previously mentioned fatigue mechanisms (Dedering et al., 2004).

Since there are differences in how those with and without pain fatigue, attempts have been made to identify those in pain based on muscle fatigue measures (Biering-Sorensen, 1984). Peach and McGill (1998) found that persons with low back pain had a higher initial median frequencies (the median frequency in an unfatigued state) and smaller median frequency slopes (rate of median frequency decline during a fatiguing contraction) in their lumbar level trunk extensors in a semi-sitting trunk extension fatigue task. These authors were able to successfully classify persons with and without low back pain based on these parameters with only one false label (Peach and McGill, 1998). Initial median frequencies and median frequency slopes in conjunction with rates of median frequency recovery of the lumber level trunk extensors have also been used to classify varsity level rowers with and without low back pain with similar accuracy (Roy et al., 1990; Klein et al., 1991). Contrary to Peach and McGill (1998), in rowers it was found that those with low back pain had larger median frequency slopes than those without any (Roy et al., 1990). Possibilities for these discrepancies include the populations used (athletic vs. non-athletic), the modeling procedures employed (discriminant analysis vs. logistic regression) and the fatiguing protocol (sustained to

voluntary exhaustion vs. a fixed duration; in all of the comparisons above, the methods used by Roy et al., 1990 and Klein et al., 1991 are listed first and the methods by Peach and McGill, 1998 are second). In both instances, the classification systems were more accurate when inputs from a larger range of muscles were employed. A third group found that those with and without low back pain had similar myoelectric and force fatigue indices of the trunk extensors, but those with low back pain had more fatigable hip extensors (Kankaanpää et al., 1998). An explanation for this finding is that deconditioned hip musculature can predispose an individual to developing low back pain because of the connection between the gluteal muscles and the lumbodorsal fascia (Kankaanpää et al., 1998). Additional support for this finding comes from the gluteus medius cocontraction response in those who develop low back pain from prolonged standing; the two hour standing protocols used in this sense can be thought of as a low level erector spinae fatigue protocol (Nelson-Wong et al., 2008; Nelson-Wong and Callaghan, 2010b; see Section 2.1.1).

Additionally, the presence of fatigue can introduce different responses in those with and without pain during functional tasks. Fatigue of the hip external rotators increases the severity of pain during step descent in those with patellofemoral pain (Cheung, 2012). It was also found that pain upon step descent while fatigued better correlated with functional disability than when unfatigued, providing evidence that a fatigued state can better differentiate the presence and severity of pain (Cheung, 2012). In an unfatigued state, those without low back pain are differentiated from those with low back pain during challenging balance tasks by those without low back pain being able to reweight proprioceptive feedback from different sources dependent on the environment (Janssens et al., 2010; Johanson et al., 2011). When either the intercostal inspiratory muscles (Janssens et al., 2010) or the trunk extensors (Johanson et al., 2011) were fatigued, those without low back pain were unable to reweight their proprioceptive feedback

whereas the low back pain group continued to respond as they did prior to fatigue. Also, the postural sway measures in those with low back pain were unaffected by fatigue of either the inspiratory or trunk extensor musculature (Janssens et al., 2010; Johanson et al., 2011), while those without low back pain did have greater postural sway when fatigued (Janssens et al., 2010). In both cases, the fatigued persons without low back pain responded similarly to unfatigued persons with low back pain. Similar findings have been reported in introducing leg muscle fatigue to those with and without patellofemoral pain: initial differences between the painful and non-painful groups were lessened upon fatiguing the knee extensors or hip abductors (Negahban et al., 2013). It appears as though in standing balance tasks, the effects of muscle fatigue can simulate chronic pain to an extent (Johanson et al., 2011).

Fatigue caused by eccentric exercise can produce pain for up to seven days following the eccentric fatigue protocol (Jones et al., 1989). Initially thought to be analogous to low frequency fatigue (Jones et al., 1989), recent evidence suggests that the delayed muscle soreness from eccentric fatigue is independent from the cellular mechanisms of low frequency fatigue (Iguchi and Shields, 2010). In contrast to exercise worsening pain symptoms in those with chronic of injection induced pain (Ciubotariu et al., 2004; Dedering et al., 2004), exercise, regardless of contraction type seems to alleviate the pain caused by eccentric fatigue (Sakamoto et al., 2010). This evidence suggests that the pain from eccentric exercise is not analogous to the pain from hypertonic saline injections or chronic pain.

2.5 Hip Abductor Function

Three muscles are often identified as hip abductors in humans: gluteus medius, gluteus minimus, and tensor fascia latae (Gottschalk et al., 1989). Of the three, gluteus medius has both the largest physiological cross-sectional area (Flack et al., 2014), and largest abduction moment arm (Dostal et al., 1986; Nemeth and Ohlsen, 1989), although the moment arm of tensor fascia latae is similar to that of gluteus medius. Although gluteus maximus shares an insertion into the iliotibial band with tensor fascia latae, its relative location with respect to the hip joint centre gives it a slight adduction moment arm (Dostal et al,. 1986; Flack et al., 2012).

Gluteus medius is believed to contain three distinct muscular compartments, anterior, middle and posterior, with evidence for distinct branches of the superior gluteal nerve supplying each compartment separately (Gottschalk et al., 1989; Flack et al., 2012). Based solely its origin, insertion and lines of action, gluteus medius can produce abduction and external rotation moments about the hip (Dostal et al., 1986). Indirectly, gluteus medius can act as a frontal plane stabilizer about the pelvis through preventing lateral tipping in single leg stance scenarios such as during gait or balance tasks (Gottschalk et al., 1989; Earl, 2005). Electrical responses of gluteus medius appear to show that maximal activity occurs in tasks that combine abduction with internal or external rotation, such as a side-lying clamshell exercise (Wilcox and Burden, 2013; Lee et al., 2014). It is not known whether this greater observed electrical response is a product of concentric (causing the motions) or stabilization (preventing displacement of the femoral head) requirements.

Tensor fascia latae is more suited than the gluteal musculature in producing hip motion in the frontal plane due to its lines of action and relatively longer fibres (Gottschalk et al., 1989; Flack et al., 2012; Flack et al., 2014). Examinations of tensor fascia late in-vivo have found that it is often co-active with gluteus medius in dynamic abduction tasks (Gottschalk et al., 1989). By

abducting the leg in an externally rotated position, tensor fascia latae can be preferentially activated over gluteus medius, however, gluteus medius activity is still present in this task (Lee et al., 2014).

Gluteus minimus is less studied than the other two hip abductors, as it can only be accessed in-vivo through indwelling electrodes. Its primary function is to produce compression and resist shear at the hip joint rather than motion about the hip (Gottschalk et al., 1989). Evidence for this is based off of its relatively small cross-sectional area (Flack et al., 2014), and its line of action is more closely aligned with the compressive axis of the hip joint than the other two muscles (Nemeth and Olsen, 1989), analogous to supraspinatus in the shoulder complex (Gottschalk et al., 1989). Gluteus minimus activity appears to match that of gluteus medius for side-lying abduction exercises, but not during single-leg stance tasks (Dieterich et al., 2015).

During standing, a hip abduction moment would result in the centre of pressure moving more laterally (Winter et al., 1996). As a result, the hip abductors are believed to be the primary muscular drivers for medial/lateral centre of pressure control in conjunction with the hip adductors (Winter et al., 1996). A brief burst of unilateral activity is required In order to effectively transfer the body mass in one direction. Bilateral activation of hip abductors, as seen in persons who develop low back pain during standing (Nelson-Wong et al., 2008; Nelson-Wong and Callaghan, 2010b), prevents this lateral transfer of body mass, resulting in less movement during prolonged standing (Gallagher, 2014). This lack of movement resulting from bilateral gluteus medius cocontraction could potentially be a cause of the low back pain developed during standing, though evidence thus far has been predominantly associative in nature (Section 2.1.1).

Attempts at introducing fatigue to the hip abductors have been performed using side-lying, leg raising exercises, often to a fixed height and cadence (Vuillerme et al., 2009; Geiser et al., 2010; Patrek et al., 2011; Sutherlin and Hart, 2015). These protocols have assumed that only the

ipsilateral hip abductors (on the side being raised) are affected by the exercise (Vuillerme et al., 2009; Geiser et al., 2010; McMullen et al., 2011), and assessment of the effects of fatigue have likewise, been tested using predominantly unilateral tasks such as single-leg drop jumps (McMullen et al., 2011; Patrek et al., 2011; Lee and Powers, 2013). There is little empirical evidence documenting how these fatigue protocols affect tensor fascia latae or gluteus minimus activity, as EMG data during these exercises has been confined to gluteus medius in these investigations. Based on the exercises used to produce muscle fatigue, the roles of the other two hip abductors and their relatively smaller sizes, tensor fascia latae and gluteus minimus should be similarly, if not exhibiting greater levels of fatigue than gluteus medius during these exercises (Flack et al., 2014).

2.6 On the Terminology of "Pain" versus "Discomfort"

"Pain" and "discomfort" are often used interchangeably to describe the same sense of physical unpleasantness associated with a physical exposure. Hamberg-van Reene and colleagues (2008) make the distinction that discomfort is a transient phenomenon whereas pain persists after the stimulus has been removed. Within this project, the regional, relative "pain" of participants will be measured; pain being in quotations as that is the word to be used on the visual analog scale given to participants. The nature of the protocol however is one where the inquiry of "pain" is made during the exposure itself, the "pain" was not present prior to the exposure and dissipates after the exposure has ended, implying that the proper term to be used should be "discomfort".

The rationale for this is three-fold. First, the word "pain" has been previously used in studies employing a two hour standing protocol to effectively differentiate groups (Nelson-Wong and Callaghan, 2010a, 2010b, 2010c; Gallagher et al., 2011; Raftry and Marshall, 2012). Second, there is evidence that those who develop "discomfort" from a prolonged standing protocol are more likely to develop persisting pain (Nelson-Wong and Callaghan, 2014). Third, there is also evidence that those who develop "discomfort" in one two hour exposure are very likely to continue to develop discomfort in future exposures (Nelson-Wong and Callaghan, 2010a). Both the repeatability and long-term outcomes of the "discomfort" developed during standing suggests an underlying mechanism, when given enough exposure, results in pain. Furthermore, the qualitative measures of "discomfort" are only being used to separate the participants into two groups based on a threshold value. Those above the threshold stand in such a way that it will lead to the development of low back pain. In essence, what is being referred to as pain is not the noxious sensation itself but rather an analog of the underlying mechanism responsible for this noxious sensation. Since it is not feasible for a person to quantify intrinsic capacities of a local area of their

musculoskeletal system, a term that can be easily understood will be quantified instead; pain. The need to differentiate pain from discomfort is therefore not necessary as, in this instance, they both appear to describe different levels of the same mechanism (Hamberg-van Reene et al., 2008).

2.7 Key Messages from the Literature Review

- Pain induction is preferred to using participants with chronic pain due to the large diversity of the chronic pain population and potential for interaction of fatigue with psychosocial variables (Sections 2.1 and 2.2).
- Prolonged standing is a reliable method of inducing pain in healthy, asymptomatic persons. It is preferred over hypertonic saline injections or noxious thermal stimuli as it is more valid to chronic pain development, especially pain development from occupational exposures (Sections 2.1 and 2.2.2).
- The pain specific to prolonged standing is linked to differences in trunk and gluteal muscle activation patterns, postural support strategies, or a combination of the two (Section 2.1.1). However it is not clear which variables, if any are responsible for causing the pain developed in prolonged standing (Section 2.1.3).

Therefore, the current project aims to determine what role, if any, gluteus medius plays in the pain developed during prolonged standing. This will be done by using muscle fatigue to perturb the natural pain developer response and measure any compensations.

- Muscle fatigue is an easily observable phenomenon with a strong physiological basis; changes in EMG frequency parameters correspond to changes in chemical concentrations (Sections 2.3.1 and 2.3.2). However in tracking the recovery from fatigue, the relative timings for non-force measures to return to rest are less reliable than force measures (Section 2.3.4).
- The development of fatigue is dependent on the type of exertion being performed (static, dynamic, eccentric, concentric etc.), the relative strength required for the exertion, and personal factors such as the age and gender of the individual (Section 2.3).

- Fatigue from lower level exertions are often difficult to quantify, and can produce responses that differ from fatigue caused by higher level exertions (Section 2.3.3).
- Recovery from fatigue is dependent on the time taken to fatigue, the amount of rest in the fatiguing task and the type of exertion. (Section 2.3.4).
- Muscle fatigue reduces the force and stiffness that the fatigued muscle can produce at a given level of activation, and reduces the level of neuromuscular control about the fatigued sites (Sections 2.3.1 and 2.3.5). Changes in joint, dynamic and whole body stability with muscle fatigue can all potentially increase the risk of injury (Section 2.3.5).
- Those with chronic pain fatigue more quickly than those without pain. Those without pain can respond like those with chronic pain when fatigued (Section 2.4).
- Fatiguing exercises targeting the hip abductors should affect gluteus medius, tensor fascia latae and gluteus minimus to similar extents, despite differences in individual hip abductor muscle function. Changes in hip abductor muscle activity while standing as a result of fatigue can influence low back pain development (Section 2.5).

The fatigue intervention may either increase or decrease the levels of pain reported in pain developers, and may also increase the reported pain in non-pain developers while standing. The recovery from a fatigue intervention has not been studied when participants perform workplace simulated tasks and may influence muscle activation patterns and postural support strategies in different manners dependent on the level of pain experienced. Although non-force measures (EMG or ion concentrations) can effectively identify the occurrence of muscle fatigue, force measures are more stable in tracking fatigue recovery. Any changes brought about by fatigue may also potentially increase the risk of injury during prolonged standing.

Section 3: METHODS

3.1 Overview of Study Design

Participants underwent two hour standing protocols on two separate occasions, once while unfatigued, and once immediately following dominant side hip abductor fatigue. Participants were classified as pain developers (PD) or non-pain developers (NPD) based on low back pain reporting during the unfatigued (control) session. Isometric hip abductor strength was assessed intermittently throughout the control and fatigue sessions to track fatigue development and recovery from fatigue respectively. Muscular responses were assessed using EMG and postural responses were assessed using a combination of force plates and an optoelectronic motion capture system.

3.2 Participants

Twenty males and twenty females between 18 and 35 years of age were recruited for this study. The age range was selected to reduce the likelihood of musculoskeletal pathologies, limit fatigue differences influenced by age (Enoka and Duchateau, 2008), and to align with prior studies on prolonged standing (Nelson-Wong et al., 2008; Gallagher et al., 2011). Additionally, all participants satisfied the following inclusion criteria: 1) no history of low back or hip pain requiring any medical intervention or time lost from work, 2) no prior hip or lumbar surgery, 3) no employment in an occupation requiring static standing during the previous 12 months and 4) able to stand unsupported for 2 hours. All participants gave their written informed consent prior to participating. Participant ages, heights, and masses, split by gender are reported in Table 3.1.

Table 3.1: Participant Anthropometry Split by Genders

	Age (years)	Height (m)	Mass (kg)
Males (n = 20)	23.7 (2.7)	1.82 (0.06)	85.0 (12.8)
Females (n = 20)	22.7 (3.0)	1.65 (0.06)	62.0 (9.2)

3.3 Instrumentation

3.3.1 EMG

Twelve channels of electromyography were collected from six muscles bilaterally: Thoracic Erector Spinae (TES), Lumbar Erector Spinae (LES), Gluteus Medius (GMD), Tensor Fascia Latae (TFL), External Oblique (EXO) and Internal Oblique (INO). Prior to electrode placement, the skin covering the muscle sites were shaved and cleaned with a light abrasive cloth (KimWipes, Kimberley-Clark Inc., Irving TX, USA) coated in an alcohol solution. Two disposable silver/silver-chloride electrodes (Blue Sensor, Medicotest Inc., Ølstykke, Denmark) were placed over the middle of the muscle belly, oriented parallel to the muscle's fibre direction with a 2 cm inter-electrode distance. A single reference electrode was placed on either the iliac crest, anterior superior iliac spine or rib cage, selecting the bony landmark with the least subcutaneous tissue covering for each participant. Specific electrode placements for each muscle are described in Table 3.2 and shown in Figure 3.1. EMG signals were differentially amplified from a bipolar electrode configuration using a common mode rejection ratio of 115 dB (at 60 Hz; input impedance of 10^{10} ohms), analog band-pass filtered from 10 to 500 Hz and gained by a factor of 500 to 5000 (AMT-8, Bortec, Calgary AB, Canada). The specific gain factor used was tailored to each individual muscle using sub-maximal test contractions and real-time visual feedback in order to best fill the input range of the A/D converter without clipping or otherwise distorting the signal (Winter, 2009). The gained signal was then sampled at 2048 Hz using a 16-bit A/D conversion card (+/- 3.5 volt input range).

Table 3.2: Electrode Locations for Electromyography

Muscle	Electrode Placement
Thoracic Erector Spinae	Oriented vertically 5 cm lateral to the T9 spinous process.
Lumbar Erector Spinae	Oriented vertically 5 cm lateral to the L3 spinous process.
Gluteus Medius	Oriented vertically at the point halfway between the line connecting the iliac crest with the posterior aspect of the greater trochanter.
Tensor Fascia Latae	Roughly one-sixth to one quarter of the way along the line connecting the anterior superior iliac spine and the lateral femoral condyle. The electrodes will be oriented slightly off of vertical (medio-superiorly).
External Oblique	Oriented medio-inferiorly just inferior to the rib cage along a line connecting the ipsilateral costal margin to the contralateral pubic tubercle.
Internal Oblique	Oriented horizontally just medial to the anterior superior iliac spine along the line connecting one anterior superior iliac spine to the other.

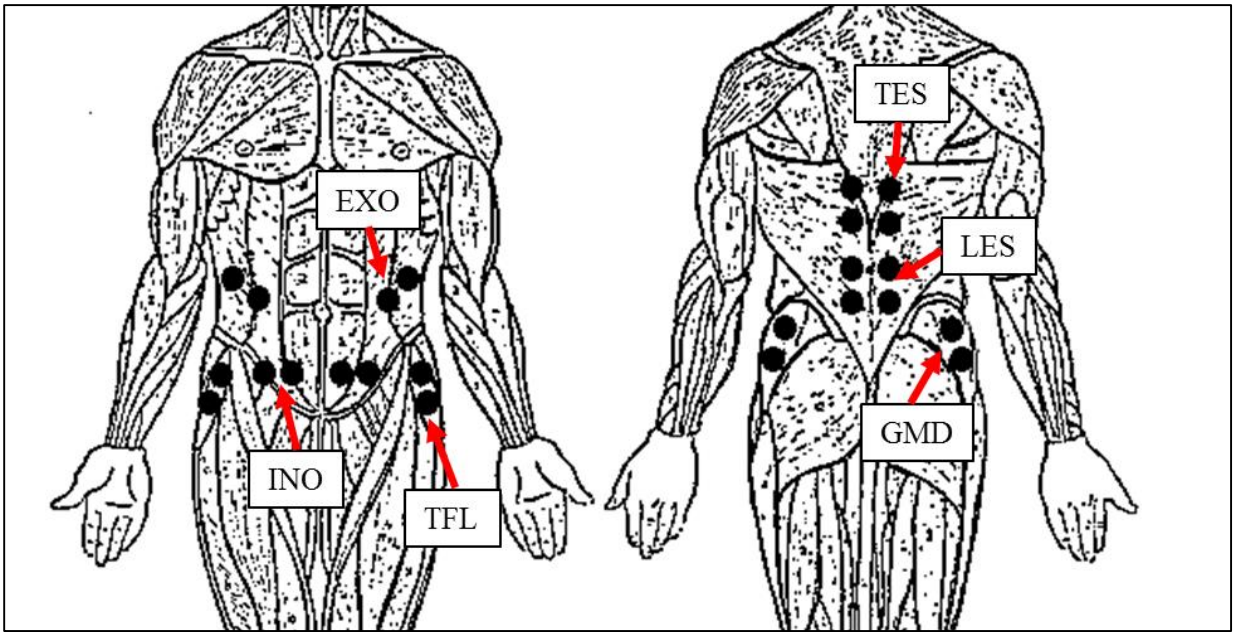

Figure 3.1: Schematic of Electrode Placements for Electromyography. Reference electrodes are not shown in this figure. See Table 3.2 for detailed land marking descriptions.

EMG signals were normalized to maximal voluntary isometric contractions (MVICs). For each contraction, participants were asked to ramp up to a maximal effort over three seconds while maintaining that maximal effort for an additional two seconds. Each MVIC was separated by at least two minutes of rest to prevent muscular fatigue, during which the signal was visually inspected for artifacts and that trial was discarded and re performed if any were present. MVICs for the left and right TES and LES were performed simultaneously with the participant lying prone on a table with their legs secured and their trunk suspended over the edge of the table (Dankaerts et al., 2004). With their arms crossed in front of their chest, participants were asked to extend their torso upwards against resistance provided by an experimenter (Dankaerts et al., 2004). MVICs for GMD were performed one side at a time. The participant lay on the side contralateral to the muscle being tested and were asked to abduct their leg against resistance provided by an examiner (Bolgla and Uhl, 2007). MVICs for TFL were performed in a similar position to the GMD MVIC, participants abducted with their leg externally rotated (instructed to point their toe upwards; McBeth et al., 2012). Since GMD and TFL perform similar functions and unilateral activation can be difficult for some participants, both MVICs from either side were considered in determining the maximum electrical activity for the hip abductors. The rationale for performing separate MVICs for the two muscles is based on evidence that externally rotating the leg activates GMD as an external rotator of the hip rather than an abductor, leaving TFL to act as the primary mover of abduction in this posture (Lee et al., 2014). MVICs for the left and right EXO and INO were performed simultaneously. Participants sat upright with their knees bent and feet flat on the table with their arms crossed against their chest and legs affixed to the table at the ankles. They performed three separate exertions in a 15 second window, cued and resisted by the experimenter. The first exertion was trunk flexion, followed by a rightward rotation of their torso, followed by a

leftward rotation of their torso (Ng et al., 2003; Dankaerts et al., 2004). Five second resting trials were collected with participants lying prone and supine on a table to allow for removal of resting bias in EMG activity.

3.3.2 Motion Capture

A twelve camera optoelectronic motion capture system (Optotrak Certus, Northern Digital Inc., Waterloo ON, Canada) was used to measure the 3D position of each participant's trunk, pelvis, thighs and feet. The capture volume of the experiment was calibrated prior to each participant's arrival using a 16-marker cube until the internal tracking system reported root mean square marker position errors between camera banks of less than 0.50 mm using dynamic and static calibration trials. The dynamic trial was used to define the capture volume of interest and the relative positions of the camera banks with respect to each other. The static trial was used to define the origin and axes of the global coordinate system. ISB conventions were followed (Wu and Cavanaugh, 1995), with the X-axis directed anteriorly, the Y-axis directed superiorly and the Z-axis directed laterally to the right. Immediately following the calibration and definition of the capture volume, the four corners of each force plate were defined in the motion capture's global coordinate system using a calibrated probe in order to align the force plates with the kinematic system and enable the mapping of centre of pressure locations onto the participant's foot (See Data Analysis, Section 3.5.3).

Four to six infrared-emitting diodes adhered to each of six rigid plastic cut-outs forming rigid marker clusters to track each segment of interest. Marker clusters were affixed to the participant's skin on the lateral edges or their left and right feet and thighs, as well as their L1 and S1 spinous processes using double sided carpet tape (Indoor Carpet Tape, Scotch, St. Paul MN, USA) and flexible surgical tape (Hypafix, BSN Medical, Hamburg, Germany) to prevent motion

of the marker clusters with respect to the participant's skin. For each marker cluster, bony landmarks were used to define the local coordinate system of the segment the cluster was to represent (Table 3.3). The feet were defined proximally by the medial and lateral malleoli and distally by the heads of the first and fifth metatarsal. Additionally, the calcaneal tuberosity was digitized for each foot to serve as a reference for the location of the centre of pressure (see Data Analysis, Section 3.5.3). The thighs were defined superiorly by the appropriate greater trochanter and inferiorly by the appropriate medial and lateral femoral condyles. The pelvis was defined superiorly by the left and right iliac crests and inferiorly by the left and right greater trochanters. The lumbar spine was defined superiorly by the most lateral portion of the left and right 12^{th} rib, and inferiorly by the left and right iliac crests. The location of these bony landmarks with respect to the local coordinate system of the appropriate rigid marker cluster were defined using the same calibrated probe that defined the force plate corners. The location of these landmarks were palpated by the same experimenter on each participant to limit inter- and intra-participant error.

Once instrumented, additional calibration procedures were performed. First a five second quiet stance trial was used to define the zero degree angle for each joint and construct a rigid link model for joint angle calculations (see Data Analysis, Section 3.5.2). A dynamic, functional joint trial for the left and right hips were then performed in order to increase the accuracy of the hip angle calculations (Sangeux et al., 2014). These trials consisted of the participant moving their leg through moderate ranges of flexion/extension, abduction/adduction, internal/external rotation and circumduction to allow for a prediction of the hip joint centre of rotation using a sphere-fitting technique (see Data Analysis, Section 3.5.2).

Table 3.3: Location of Marker Clusters and their Associated Digitized End-Points for Motion Capture

Segment Name	Cluster Location	Digitized Bony Landmarks
Lumbar Spine	L1 Spinous Process	• Most lateral portion of the left 12th Rib and right 12th Rib • Bilateral Iliac Crests
Pelvis	S1 Spinous Process	• Bilateral Iliac Crests • Bilateral Greater Trochanters
Left Thigh	Left Femoral Shaft	• Left Greater Trochanter • Medial and lateral Femoral Condyles of the left leg
Right Thigh	Right Femoral Shaft	• Right Greater Trochanter • Medial and lateral Femoral Condyles of the right leg
Left Foot	Postero-lateral aspect of the participant's left foot	• Left medial and lateral malleoli • Left 1st and 5th metatarsals of the left foot • Left Calcaneal Tuberosity
Right Foot	Postero-lateral aspect of the participant's right foot	• Right medial and lateral malleoli • Right 1st and 5th metatarsals • Right Calcaneal Tuberosity

3.3.3 Force Transducers

Two in-ground, strain gauge force plates (Left foot on an OR6-7 model - 50 cm by 50 cm surface, Right foot on a BP900900 model - 90 cm by 90 cm surface; both from Advanced Medical Technology Inc., Watertown, MA, USA) were used to measure postural shifts, fidgets and body weight transfers based on the methods of Duarte and Zatsiorsky (1999), Prado et al., (2011) and Gallagher (2014). The analog signal from each force plate was amplified (MSA-6 Miniamp, Advanced Medical Technology Inc., Watertown, MA, USA) then A/D converted using a 16-bit A/D card (+/- 10 volt input range) at 64 Hz. Calibration matrices provided by the manufacturer

were used to convert the voltage outputs into Newtons and Newton metres. Both force plates were turned on at least one hour prior to the start of the protocol to limit voltage errors linked with drift caused by internal temperature changes of the amplifiers and zeroed immediately prior to the start of the standing protocol in each session.

A linear variable displacement transducer (LVDT; MLP-150-CO, Transducer Technologies, Temecula, CA, USA) was used to measure hip abduction force in test contractions in five second windows while participants stood. Within each five second window, there was a two second buildup of force, a two second maximal hold, and a one second release window. The specific time points for these measurements are stated in Section 3.4.3: Data Collection. A 66.684 Newton assembly was hung from the LVDT to calibrate the sensor prior to each data collection using a linear transformation. The LVDT was tethered to a cuff placed around the participant's femoral condyles. The voltage outputs were amplified (Strain Gauge Conditioner 3270, Daytronic Corporation, Miamisburg, OH, USA), A/D converted using a 16-bit A/D card (+/- 10 volt input range) and sampled at 64 Hz.

3.3.4 Visual Analog Scales of Pain

Regional pain of participant's lower back, left gluteal and right gluteal region were assessed using visual analog scales (VAS). Each VAS was 100 mm long anchored at either end with "No Pain" (0 mm) and "Worst Pain Imaginable" (100 mm). Participants indicated their pain for each region by marking the location on the line that best represented their current state of pain. Scales of this size and anchoring have been previously used to study pain development during prolonged standing in order to differentiate pain developers from non-pain developers using a cut-off threshold of 10/100mm (Gregory and Callaghan, 2008; Nelson-Wong et al., 2008).

3.4 Experimental Protocol

Each participant partook in two separate laboratory testing sessions. These sessions occurred at the same time of day per participant to minimize circadian effects in spine posture (Adams et al., 1990) and fatigability (Mika et al., 2007). Sessions were separated by at least one week to allow for full recovery from fatigue or pain development (Leyk et al., 2006; Mika et al., 2007; Jougla et al., 2010; Yung et al., 2012).

3.4.1 Initial Documentation

Upon receiving consent to initiate testing, three tests were carried out prior to starting the first session: a leg dominance test, an active hip abduction test, and Ober's test. The leg dominance test consisted of asking the participant to kick a soccer ball, write their name in on the floor using a foot as an imaginary marker, and arrange five small scattered objects into a straight line (Chapman et al., 1987). Three additional tests (closed-eye single leg stance, a lateral reaching task, and a catch and throw task) were performed to blind the participant to the activities assessed during the test. When the leg dominance tests required objects to be manipulated, the objects were placed equidistant from either leg to avoid any biases in limb choice based on proximity (Chapman et al., 1987). The limb used for at least two of the three tasks was recorded as the dominant limb.

The active hip abduction test (Figure 3.2) has been previously used to screen healthy young adults to determine whether or not they are likely to develop pain over the course of a two hour standing exposure (Nelson-Wong et al., 2009). For this test, participants lay on their side in zero degrees of trunk, hip, knee and ankle flexion and actively raised and lowered one leg to its end range of motion. Participants were told to perform this motion in a smooth and controlled manner without allowing their trunk or pelvis to roll frontwards or backwards. Upon completion, participants were asked to rate the difficulty of performing the test on a scale from zero to five with zero indicating "no difficulty at all" and 5 indicating "cannot perform the task". The test was

repeated for both the left and right limbs, with the self-rated scores summed for both limbs. Additionally, the experimenter visually observed the motion and rated each side from zero to three based on postural cues with higher scores indicating greater loss of alignment. Specific cues for experimenter grading are shown in Table 3.4, with the worse score from the two sides representing the participant's grade. Those who have a combined self-rated score of greater than 4 out of 10 or are graded by the experimenter as a 2 or 3 were found to be more likely to develop low back pain during two hours of standing (Nelson-Wong et al., 2009).

Ober's test (Figure 3.3) is an orthopedic test designed to detect tightness within the iliotibial tract (Gajdosik et al., 2003; Herrington et al., 2006). Participants were asked to lie on their side with their top leg extended and bottom hip and knee flexed to approximately 90 degrees. The experimenter raised the participant's lower limb into slight abduction and then allow gravity to passively lower the limb into adduction while applying pressure to the pelvis to prevent pelvic rotation. A positive test was indicated by the upper leg remaining in abduction (above the horizontal) while under the influence of gravity when the participant is relaxed (Ferber et al., 2010). The state of the iliotibial tract is believed to influence the muscle activation levels of TFL (Earl et al., 2005), and therefore may also affect measures within the hip abduction contractions or the time and level of fatigue (see Sections 3.4.3 and 3.4.2 respectively).

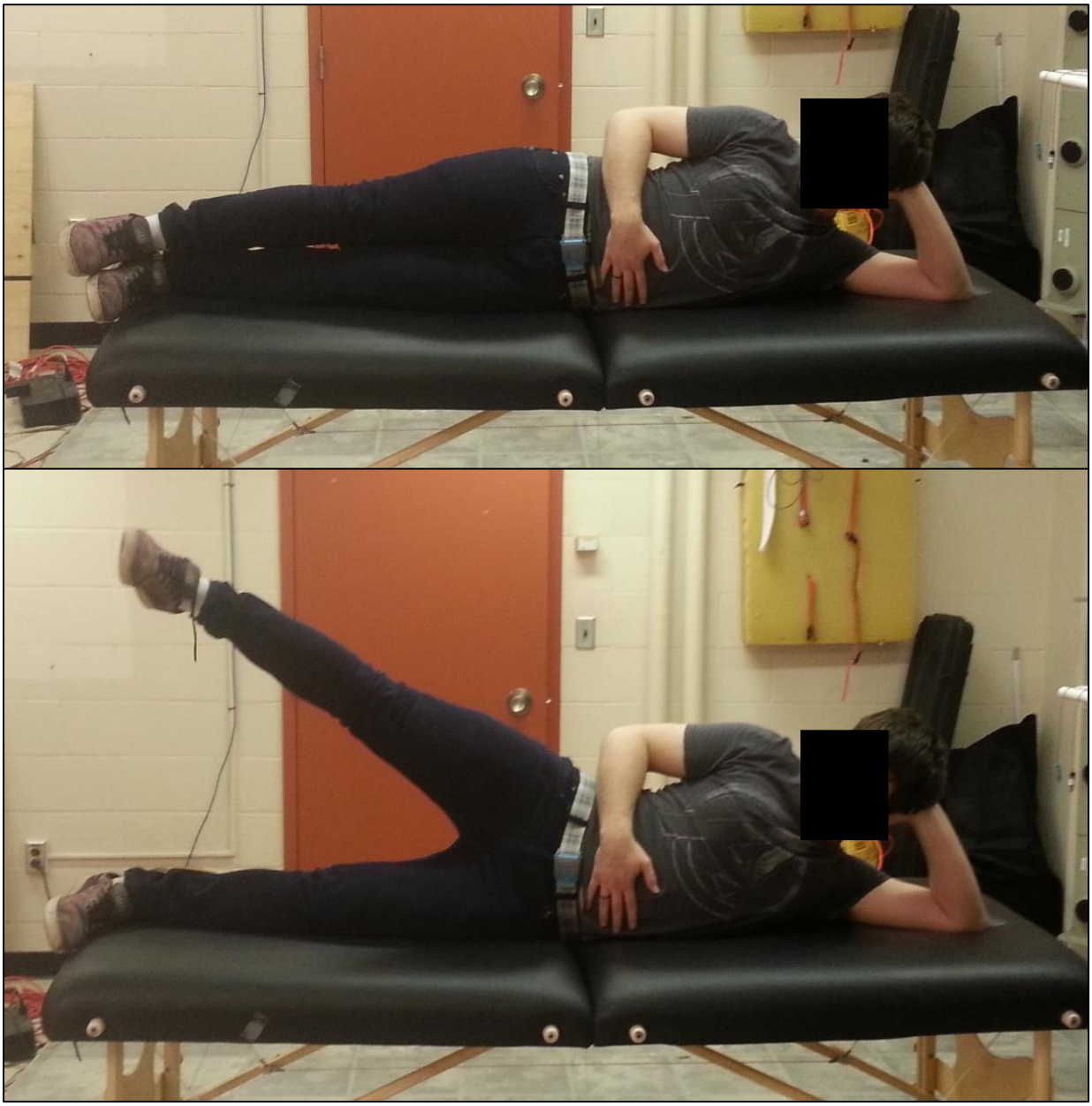

Figure 3.2: The Active Hip Abduction Test. Top panel shows the initial starting position, bottom panel shows the peak abduction angle this participant could achieve. The participant is instructed to perform the movement smoothly and with as little pelvic rolling as possible.

Table 3.4: Examiner Cues for Grading on the Active Hip Abduction Test. Adapted from Nelson-Wong et al., (2009). The participant in Figure 3.2 would receive a score of zero.

Examiner Score	Visual Cues
Zero (No loss of pelvis frontal plane)	• Movement is performed smoothly and with ease. • Lower extremities, pelvis, trunk and shoulder are aligned in the frontal plane throughout the movement.
One (Minimal loss of pelvis frontal plane)	• A slight "wobble" is apparent upon initiation of movement, but movement is otherwise smooth and controlled. • The motion requires noticeable effort or contains a "ratcheting" like pattern (staggers upwards/downwards)
Two (Moderate loss of pelvis frontal plane)	• There is a definite "wobble" or tipping of the pelvis. • The shoulders or trunk are rotated, the hips are flexed or the abducting limb is internally rotated. • Movement is rushed and the participant is unable to regain control of the limb during the motion.
Three (Severe loss of pelvis frontal plane)	• Similar patterns to a rating of two only with greater severity. • Movement is uncontrolled and requires the use of a hand or arm to maintain balance.

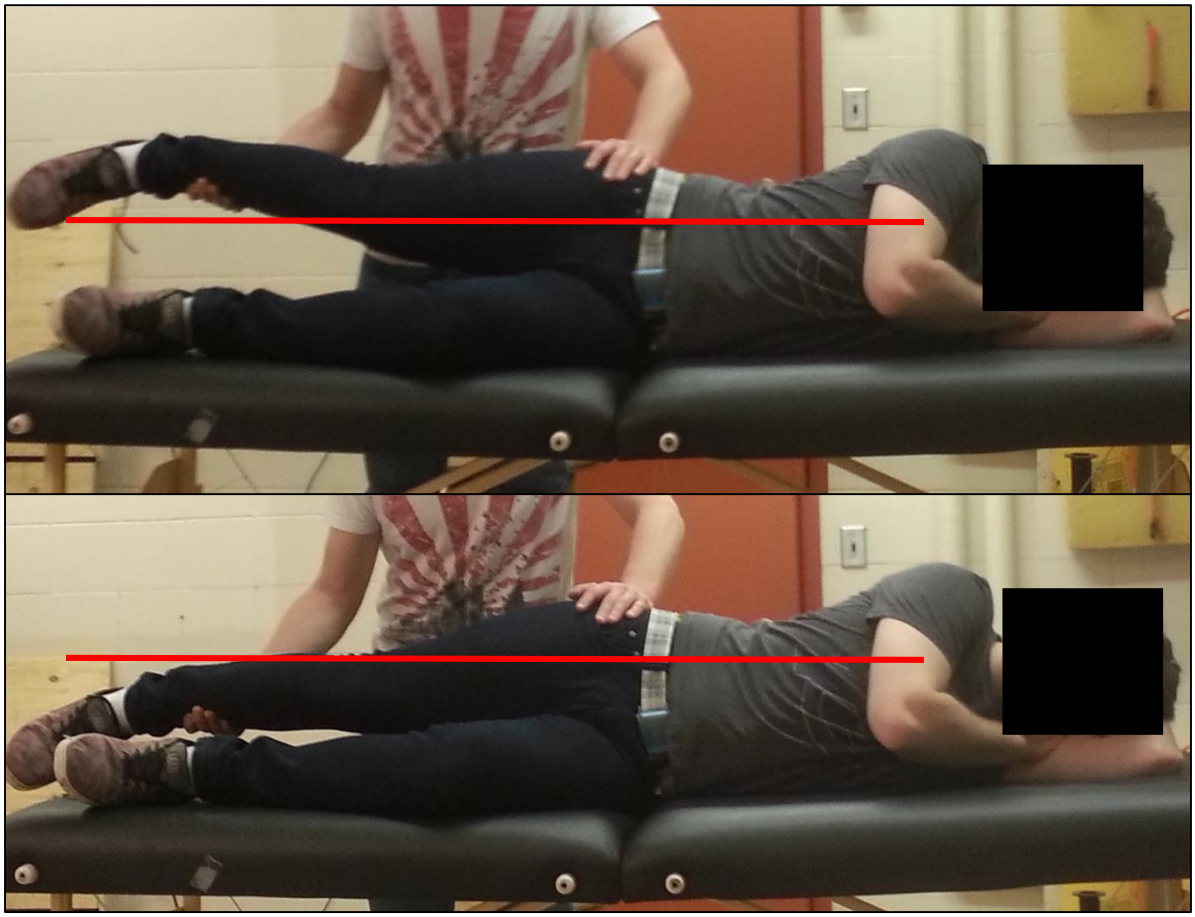

Figure 3.3: Ober's Test. The test involves the participant side lying with the ipsilateral leg flexed at the hip and knee and the contralateral leg supported by the examiner (top panel). While fixing the pelvis, the examiner lets the contralateral leg drop into adduction (bottom panel). A horizontal line is drawn over the pictures for reference.

3.4.2 Control and Fatigue Conditions

Upon EMG and motion capture instrumentation, participants either began the standing protocol or a fatigue protocol depending on the session (control or fatigue). The order of sessions was randomized for each participant. The fatigue protocol consisted of cyclical, side lying hip abduction similar to the active hip abduction test. Participants repeatedly abducted and lowered their dominant leg against gravity to 30 degrees, taking 1 second to ascend and another second to descend the limb back to rest. The requisite height corresponding to 30 degrees of abduction for each participant was set using a height-adjustable tensor band and a goniometer (Figure 3.4). The

limb coming into contact with the tensor band provided kinesthetic feedback to the participant that the required amount of abduction was performed. A 2:1 work to rest duty cycle was implemented with this task where participants performed ten seconds of cyclical abduction (5 cycles), followed by 5 seconds of rest. Positive, non-threatening verbal encouragement was given to participants while performing the fatiguing task and intensified as the protocol progressed. This protocol was continued until the participant was unable to successfully complete two consecutive duty cycles, or five duty cycles in total. Duty cycle failure criteria are listed in Table 3.5.

The fatigue protocol was designed in this manner in an attempt to induce fatigue that required as long of a recovery period as was reasonable without causing whole body fatigue. Longer times to fatigue result in longer times to recover force and electromyographic variables even when participants report similar levels of exhaustion (Baker et al., 1993). A timed, cyclical hip abduction fatigue protocol very similar to the one proposed was previously used in varsity-level student athletes and resulted in a mean time to fatigue of just under 3 minutes (Patrek et al., 2011). Implementing rest breaks would prolong the time to fatigue, the time to recover (Yung and Wells, 2012), and made the fatigue more relevant to the intermittent work patterns encountered in the workplace (Westerblad et al., 2000). Using stringent exhaustion criteria rather than voluntary criteria attempted to reduce the inherent variability in self-reported measures and obtain a requisite level of fatigue across participants.

EMG was collected for every eighth duty cycle (once every two minutes) during the fatigue protocol to track the rate of fatigue. All duty cycles following an unsuccessful duty cycle were also recorded to capture the end fatigue state. Upon completion of the fatigue protocol, a rating of perceived exertion (10 point Borg scale) score was taken in an effort to quantify exhaustion level.

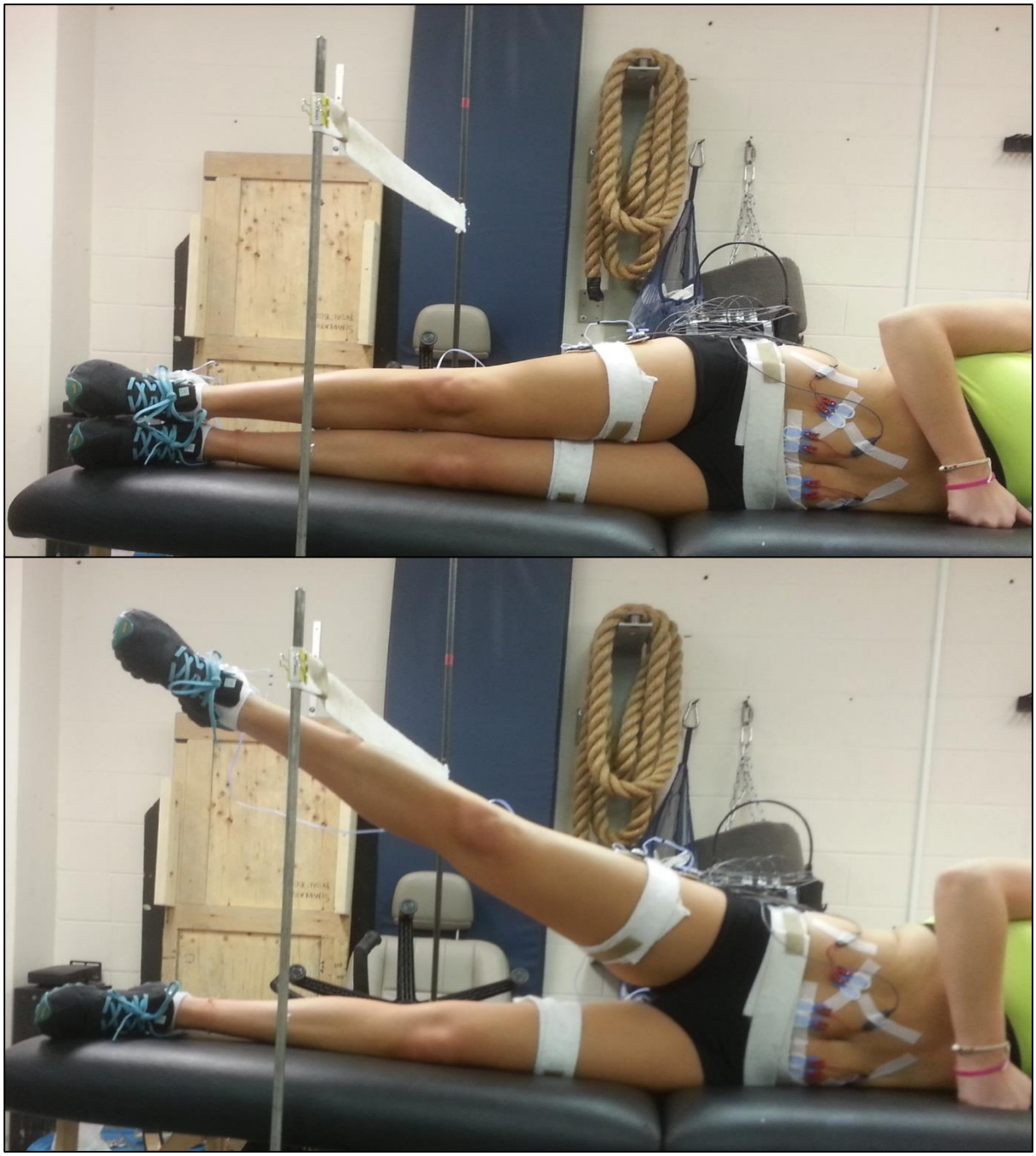

Figure 3.4: The Fatigue Protocol. While side-lying, participants moved their dominant leg to 30 degrees of abduction, marked by a tensor bandage strung between two metal columns. A metronome provided auditory cues as to when their leg should hit the tensor bandage and return to rest. Arm and hand positions were not constrained during this task.

Table 3.5: Criteria for Unsuccessful Duty Cycles within the Fatigue Protocol.
Compensations performed by participants during the fatigue protocol that would eventually terminate the protocol. Each participant was given 4 duty cycles to establish a rhythm before criteria were implemented. Participants were not explicitly told what compensations were being judged but were instructed that failure was on the basis of movement quality.

Compensation	Details
Unable to contact the tensor bandage	If the top leg did not reach 30 degrees of abduction for at least 3 of 5 repetitions, that duty cycle was considered unsuccessful.
Cycle time was inconsistent	There were two patterns in this compensation that could cause a duty cycle to be labelled as unsuccessful: 1. Time of contact was at least half a second after the auditory cue for at least 3 of 5 repetitions, often displayed as a slowing over time. 2. The durations of at least 3 of 5 repetitions were shorter than 1 second.
Lack of control of movement in sagittal plane	Consistently presenting at least two of the following patterns for at least 3 of 5 repetitions would label a duty cycle as unsuccessful: • Multiple changes in limb velocity upon ascent or descent not related to changing direction (ratcheting) • Having an excessively high limb velocity upon initiation of a cycle • Combining internal rotation of the limb with knee flexion to contact the tensor bandage without the requisite amount of hip abduction. • Muscular tremor while attempting to hold the abducted limb at the tensor bandage, often done in attempt to correct timing errors.
Lack of control of movement in frontal plane	Consistently presenting at least two of the following patterns for at least 3 of 5 repetitions would label a duty cycle as unsuccessful: • Frontal plane wobbling upon ascent or descent, wobble amplitude had to exceed the width of the abducting thigh • Pelvic tipping similar to the active hip abduction test • The point of contact with the tensor bandage was visually more anterior or posterior than the lower leg
Did not attempt 5 repetitions in a duty cycle	Any duty cycle without 5 distinct leg abductions was automatically considered unsuccessful.

3.4.3 Data Collection

Immediately after the fatigue protocol or instrumentation dependent on the session, participants began two hours of standing, during which they completed typing and sorting tasks at a workstation. The work surface height was initially set to 5 centimetres below the participant's pronated hands when their arms were positioned at their side and elbows flexed to 90 degrees. Slight adjustments from that starting point were made based on participant comfort prior to the start of the first task and fixed for the duration of the standing protocol. One task was performed for the first hour, the other for the second hour with the order of the two tasks randomized for each participant and reversed from the first to the second session. While standing, participants were given the following instructions:

- Stand naturally, focusing on the current task as if you were in a workplace setting
- You may move your feet around as you wish, however you must keep each foot within the boundaries its respective force plate.
- You may rest your hands and forearms on the workstation, however you are not allowed to rest your upper body or lean on the workstation.
- Please keep in mind that you are tethered to the wall.

Adherence to these instructions were determined visually by the experimenter, and participants were prompted when instructions were violated. EMG, motion capture, and force plate measurement systems were recording continuously throughout the two hours of standing with short breaks to allow for hip abduction contractions (see below).

Prior to and throughout the standing protocol, maximal isometric standing hip abduction contractions (ABCs) were performed to track changes in muscle strength. Participants were asked to brace against the workstation and abduct their dominant leg, ramping up to and holding a

maximum contraction over a five second period. Resistance was provided by a cuff and tether secured around their femoral condyles and tensioned such that resistance was applied in a hip abduction angle very close to quiet stance while minimally interfering with the standing tasks. During the first session, participants were given time to become familiar with the apparatus and determine a consistent bracing and activation strategy to produce maximal efforts. ABCs were performed a) immediately after instrumentation (to be used as a 100% measure for force and EMG variables and the 0 minute ABC during the control condition), b) immediately after the fatigue protocol on the fatigue session (0 minute ABC for the fatigue condition), and c) at times 1 minute, 2 minutes, 3 minutes, 5 minutes, 10 minutes, 15 minutes, 30 minutes and once every 15 minutes thereafter during the 2 hours of standing. The spacing of the ABCs attempts to best capture the exponential recovery of force and EMG variables (Lind, 1959; Soo et al., 2012). During the ABCs, the tension in the tether as measured by the LVDT (representing abduction strength) and muscle activity were recorded.

VAS scores were reported prior to instrumentation, at the zero minute time point (control: upon completion of instrumentation; fatigue: upon completion of fatigue protocol) and every 15 minutes thereafter while standing. The order of operations for the two data collection sessions are outlined in a flow chart (Figure 3.5). During times when a VAS score and an ABC were required at the same time, the VAS score was completed prior to the ABC. Time taken to complete the VAS and ABC measures was not deducted from the two hours allotted for the standing protocol in order to allow for an equal standing exposure length for all participants across sessions.

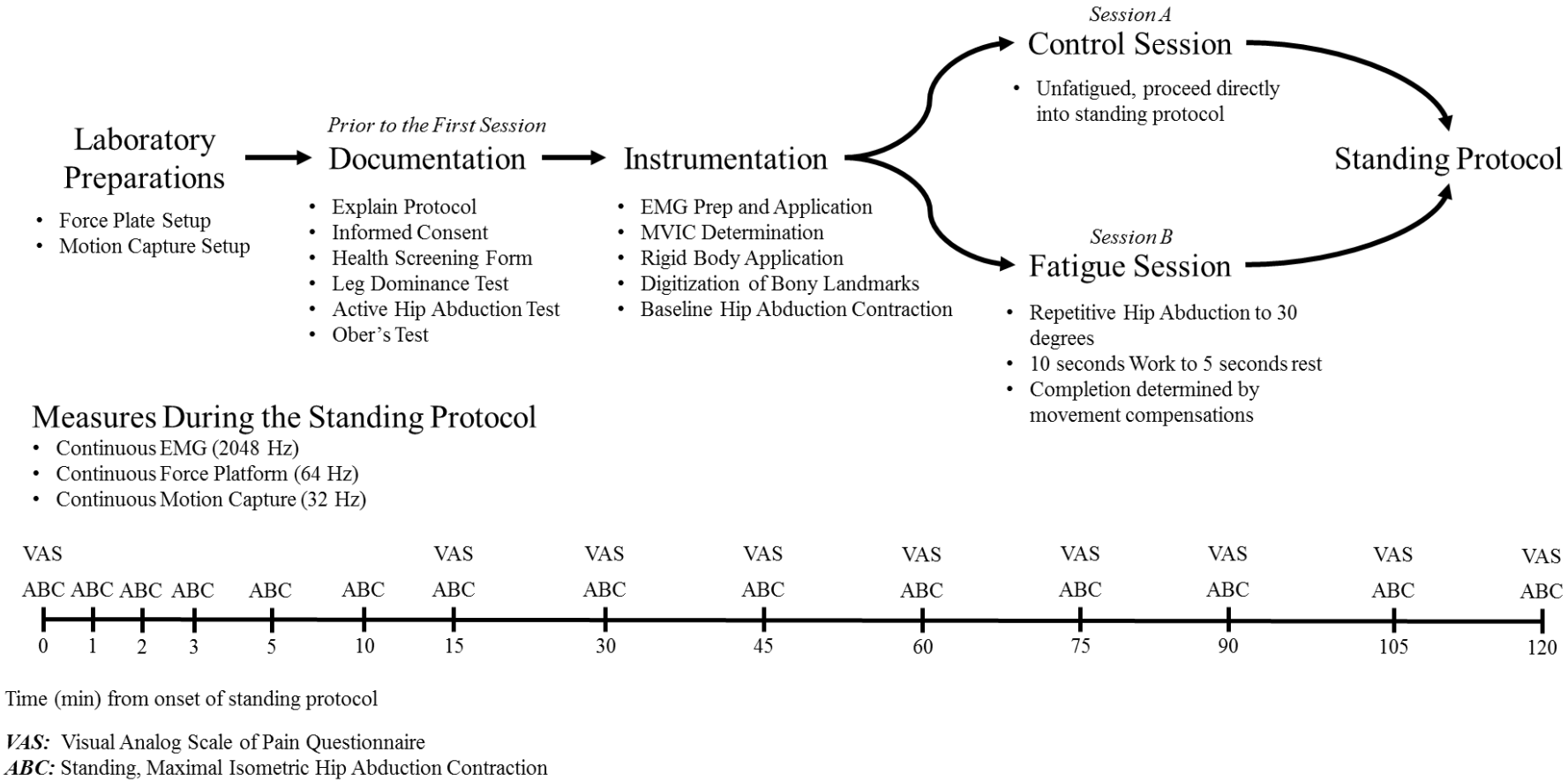

Figure 3.5: Outline of Experimental Protocol. The laboratory was prepared prior to participant arrival. Informed consent, leg dominance tests and orthopedic test were performed on the first session prior to instrumentation. The order of the control and fatigue sessions were randomized for all participants and completed on separate dates at least one week apart at the same time of day. Session duration ranged from three to four and a half hours dependent on participant familiarity with testing procedures, presence and duration of the fatigue protocol and the success of signal detection and software functionality.

3.5 Data Analysis

Unless otherwise specified, all data processing was performed using custom written MatLab scripts (version 2013b, The Mathworks Inc., Natick MA, USA).

3.5.1 EMG

EMG data were collected on both high activity (the ABCs) and low activity levels (the rest of the prolonged standing task), with different rationales for collecting muscle activity data in the ABCs and the prolonged standing tasks. Because of these differences in purpose, the data processing algorithms were different between the two portions to reflect their specific end goals.

The EMG signals during the ABCs were processed to track the level of muscle fatigue and subsequent recovery. Changes in the frequency content of the unprocessed EMG signal are a historically reliable index of muscle fatigue (Petrofsky, 1979; Bigland-Ritchie and Woods, 1984; Dolan et al., 1995; Champagne et al., 2008). Each ABC was divided into ten, 500 ms windows, a fast Fourier transform was performed on each window separately, the mean power frequency of the resulting frequency spectrum for each window was computed, and then the average of the ten windows was taken. This splitting and re-averaging procedure was done to allow for a more stable indicator of the mean power frequency and maintain the stationarity requirements of Fourier transforms (Cho and Kim, 2012). Mean power frequencies were computed for all recorded muscles, and the resulting mean power frequencies were expressed as a percentage of the baseline (pre standing or hip abductor fatigue) mean power frequency. Sixty Hz band stop (59 to 61 Hz cutoffs) and 30 Hz high pass Butterworth filters were implemented prior to the fast Fourier transform to remove contamination from electromagnetic hum (Mello et al., 2007) and heart rate activity (Drake and Callaghan, 2006) respectively.

The time varying EMG data from the two hours of prolonged standing was collected in order to document broader scale muscle activity patterns. Cocontraction indices (CCI) and gaps in muscle activity were utilized for this purpose. Both measures were performed on normalized, low pass filtered EMG signals with the resting activity, electromagnetic hum, and heart rate activity removed. The following steps were taken in the data processing chain:

1. Any bias in the signal was be removed (after the hardware band pass filter).
2. A high pass, 4^{th} order, zero lag Butterworth filter with a 30 Hz cutoff was applied to remove contamination from heart activity (Drake and Callaghan, 2006).
3. A 4^{th} order 60 Hz band stop Butterworth filter was applied to remove contamination from electromagnetic hum; the same filter used on the ABCs (Mello et al., 2007).
4. The uncontaminated data was full-wave rectified, then low pass filtered using a 4^{th} order, zero lag Butterworth filter with a 2.5 Hz cutoff (Brereton and McGill 1998) and one second (Howarth and Callaghan, 2009) of reflected padding points on either end to represent the activation level of each muscle.
5. The lower of the mean voltages from the two resting trials for each muscle were subtracted from the standing trials to account for resting electrical activity levels.
6. The rest-removed muscle activity was expressed as a percentage of the maximum activity recorded during the MVIC trial (processed using steps 1 to 5) for that muscle to allow comparisons between subjects.

The CCIs between muscle pairs were calculated on the normalized, linear enveloped EMG data in the same manner as Lewek et al., (2004) and Nelson-Wong et al., (2008) using Equation 3.1.

$$CCI = \sum_{i=1}^{N} \left[\left(\frac{EMG_{low\,(i)}}{EMG_{high(i)}} \right) \times \left(EMG_{high(i)} + EMG_{low\,(i)} \right) \right] \quad \textbf{(3.1)}$$

Where EMG_{high} and EMG_{low} refer to the muscle of the two with the higher and lower activation levels in the i^{th} frame over a period of N frames of data. EMG data were down sampled to 32 Hz prior to the calculation of CCI. The variable N was set as a one minute duration (32 frames per second for 60 seconds; 1920 frames). The data were reduced further by averaging the CCI in 15 minute blocks to align with the periods of VAS reporting, for 8 CCIs per muscle pair for a 120 minute standing protocol. Of the 66 unique combinations available, 10 muscle pairings were selected for analysis: four bilateral pairings (R-TES/L-TES, R-LES/L-LES, R-GMD/L-GMD and R-TFL/L-TFL), two anterior-posterior pairings (R-LES/R-EXO and L-LES/L-EXO), and four gluteal-lumbar pairings (R-GMD/R-LES, R-GMD/L-LES, L-GMD/R-LES, and L-GMD/L-LES). Appendix B contains more information on factors that contribute to a larger CCI.

A gaps analysis was also performed on the time varying normalized EMG signals. In prolonged, low-level exposures, those who show more "gaps" in their time varying EMG data - defined as an interval when the muscle is below 0.5 %MVIC for at least 200 ms - tended to report less muscle pain and fatigue (Veiersted et al., 1990). Also, the number of gaps in gluteus medius activity within the first fifteen minutes of standing has been used in conjunction with other variables to predict whether a person will develop pain over two hours of standing (Gregory and Callaghan, 2008). Processed EMG signals for each muscle were partitioned into 15 minute blocks, all time points when a muscle falls below 0.5 %MVIC was marked, and any continuous marked time of sufficient length (410 frames at 2048 Hz is 200.2 ms) was labelled as a gap. The total number of gaps, the mean gap duration and the cumulative time spent below 0.5 %MVIC was

computed on each 15 minute block. As with the CCIs, the intent in using 15 minute blocks was to have gap information align with VAS scores.

EMG data collected from the fatigue protocol were put through both the mean power frequency window (ABCs) and normalization to %MVIC (standing) processing algorithms. Normalized, low-pass filtered EMG data from the fatigue protocol was used to determine broad-scale activation levels during different stages of fatigue protocol completion. For each participant, trials from time points representing the start, 1/3 to completion. 2/3 to completion and end of the fatigue protocol were identified. Trials were required to have the peak dominant GMD activity of the first repetition occurring within the first 250 ms of the trial to be included. The peak activation of the dominant GMD was identified, and the sum of all twelve muscles at that frame in the trial was computed as an indicator of total muscle use during that duty cycle. The timing of that peak expressed as a percentage of that duty cycle was also recorded for each trial.

3.5.2 Motion Capture

Motion capture position data were used to construct a three dimensional rigid-link model using Visual 3D software (ver. 4, C-Motion Inc., Kingston ON, Canada). A thoracic spine, lumbar spine, pelvis segment, and bilateral thigh segments were constructed using the digitized bony landmarks (refer to Table 3.3) to define each segment's local coordinate system and a static upright stance trial to define a reference posture. All local coordinate systems followed ISB conventions for the trunk and lower limb (Wu and Cavanagh, 1995).

The raw time varying marker data from the standing protocol, partitioned into 15 minute blocks, were imported into Visual 3D. Marker positions were filtered using a 4^{th} order, zero lag Butterworth filter with a cutoff of 6 Hz and one second of reflected padding points within the rigid link software (Howarth and Callaghan, 2009). Participant and session specific rigid-link models

were applied to the filtered data. Data from the functional hip joint trials were then used to improve the accuracy of the hip joint centre estimations within each model using a built-in recursive sphere-fitting algorithm. The program uses the three-dimensional motion of the distal segment with respect to the proximal segment to find the tip of a cone representing the centre of rotation of the joint between those two segments. Although a similar algorithm could be used for the other joints, it is not possible for many participants to isolate movements at a single vertebral joint without causing motion at an adjacent joint. As the accuracy of sphere-fitting methods relies on movement only occurring at the joint of interest (Sangeux et al., 2014), attempts to perform functional joint trials at the various spine levels would likely result in erroneous calculations. The model was used to derive time-varying Euler angles for the 15 minute block. Left and right hip (thigh w.r.t. pelvis), lumbar spine (lumbar w.r.t. pelvis), and pelvic tilt (pelvis w.r.t. Global) angles were computed using a Z-X-Y rotation sequence corresponding to flexion, abduction/lateral bend, then axial/external rotation. This sequence was chosen as it is expected that the greatest amount of motion for each segment will occur in the sagittal, frontal then transverse planes in descending order. The mean angles and standard deviations were computed on each 15 minute block. Since motion capture data are only used for kinematic purposes, the inertial properties of the models were not computed.

The foot clusters were used to infer where the measured centre of pressures from each force plate fell with respect to the participant rather than angle calculations. Additional details of this computation appear in Section 3.5.3 below. The foot segments had their local coordinates derived outside of Visual 3D as it was not used in any angle computation. The local coordinate systems of the left and right feet were defined to best align with the global coordinate system, outlined in Table 3.6.

Table 3.6: Definition of Local Coordinate Systems for the Left and Right Feet. The orientations of the foot's axis systems are designed to align with the Optotrak global coordinate system as best as possible.

Axis	Definition
X axis	The line connecting the calcaneal tuberosity and the midpoint between the 1st and 5th metatarsal. Positive is directed anteriorly.
Temporary Axis	The line connecting the medial and lateral malleoli. Positive is directed to the right.
Y axis	The cross product between the X axis and the temporary axis. Positive is directed upwards (dorsally).
Z axis	The cross product between the X axis and Y axis. Positive is directed to the right for both the left and right feet.
Origin	The calcaneal tuberosity.

3.5.3 Force Transducers

The vertical components of the two force plates were used to quantify medial-lateral sway measures termed body weight transfers (Prado et al., 2011; Gallagher, 2014). A body weight transfer was defined as a continuous lateral movement of the torso involving transferring at least 30% of the participant's body weight from one leg to the other. These were quantified by combining the vertical ground reaction forces into a single measure, F_vRL, representing the level of asymmetry in body weight support between the legs (Equation 3.2).

$$F_vRL = \frac{F_vR - F_vL}{2 * (F_vR + F_vL)} \quad \textbf{(3.2)}$$

F_vR represents the vertical component of the ground reaction force from the right force plate, and F_vL represents that component from the left force plate. If a participant was evenly supporting their upper body with both legs, F_vRL would be 0, and can range from 0.5 (entirely supported by the right foot) to -0.5 (entirely supported by the left foot). The derivative of F_vRL was taken to identify times where F_vRL was continually increasing (moving rightward) or decreasing (moving leftward).

If a continuous change in F_vRL exceeded a threshold set at 0.3, representing a movement of 30% body weight, a body weight transfer was said to have occurred. A continuous transfer of more than 60% body weight was still counted as a single transfer.

Centre of pressure data was computed from both force plates, transformed into the global coordinate system, and fed into two data processing streams: 1) the computation of postural shifts and fidgets, and 2) the mapping of centre of pressure onto the foot.

The location of the centre of pressure on each force plate for the anterior/posterior and medial/lateral directions was determined using Equations 3.3 and 3.4 respectively.

$$CoP_{AP} = \frac{M_{ML}}{F_{Vert}} \quad \textbf{(3.3)}$$

$$CoP_{ML} = -\frac{M_{AP}}{F_{Vert}} \quad \textbf{(3.4)}$$

Where M refers to channels of moment data and F_{vert} refers to the vertical channel of force data, the moment subscripts AP and ML refer to the force plate axes oriented anterior/posterior and medial/lateral respectively. A force plate local coordinate system was then constructed using the digitized force plate corners and the centre of pressures were rotated from the force plate local system into the global coordinate system. Once in the global reference frame, the centre of pressures were re-expressed with relation to the global origin (positioned near on the postero-lateral corner of the left force plate) by computing the distance from the origin of the force plate - offset from top centre of the force plate by a fixed amount specified by the manufacturer - to the origin of the global coordinate system and adding that vector to the rotated centre of pressure.

For the first centre of pressure processing stream, global centre of pressure from each foot was combined into a single centre of pressure using Equation 3.5.

$$CoP_{combined} = \left(CoP_{left} * \frac{F_vL}{F_vL + F_vR}\right) + \left(CoP_{right} * \frac{F_vR}{F_vL + F_vR}\right) \quad \textbf{(3.5)}$$

F_vL and F_vR correspond to the vertical channel of force data from the left and right foot respectively, and the subscripts left and right refer to which force plate the centre of pressure data originated from. Only the anterior centre of pressures were combined and fed into the postural shift and fidget algorithm as body weight transfers already quantify medial/lateral weight support strategies and have been associated with low back pain development with standing (Gallagher, 2014).

Two postural support variables were calculated from the combined centre of pressure data: 1) shifts, quick "step-like" changes in percent body weight that do not return to their previous values and 2) fidgets, large changes in percent body weight that quickly return to their previous values (Duarte and Zatsiorsky, 1999). The number of shifts, fidgets and drifts were determined over the entire two hour standing exposure, then grouped together based on which 15 minute block they occurred in.

A single algorithm computed both variables. First, the entire two hour exposure was passed with two moving windows (W_1 and W_2) of equal size separated by a fixed distance. The mean and standard deviation of each window was calculated for each feasible point in time and used to determine the variable β in Equation 3.6.

$$\beta = \left| \frac{Mean_{W1} - Mean_{W2}}{\sqrt{{SD_{W1}}^2 + {SD_{W2}}^2}} \right| \quad \textbf{(3.6)}$$

When the value of β was greater than twice the denominator in Equation 3.2 (the geometric mean of the standard deviations of the two windows) then a shift was said to have occurred between W_1 and W_2 (Duarte and Zatsiorsky, 1999). The size of W_1 and W_2 was set at 15 seconds (960 frames)

and the distance between W_1 and W_2 was set at 5 seconds (320 frames). The exact duration of each shift was determined by reducing the distance between W_1 and W_2 until the value of β was less than the established threshold, at which point the then reduced inter-window distance was recorded as the shift duration. Given the size of the moving windows, no shifts were detected within the first and last 15 seconds of data collection; those time points were not feasible for this approach.

Once the number, temporal location and duration of all the shifts within the two hours of standing were determined, the data in between shifts was passed forward in order to compute fidgets. The reduced data set had the time points of all local maxima and minima identified with a half maximum width of less than four seconds. The mean and standard deviation within a 60 second window centered on each maximum and minimum was calculated and used to determine the variable γ in Equation 3.7.

$$\gamma = \left| \frac{Peak - Mean_{window}}{SD_{window}} \right| \quad \textbf{(3.7)}$$

Where *Peak* is the local maxima or minima that the window is centered about. If γ for a given peak is greater than three times the value of SD_{window}, then that peak represents a fidget (Duarte and Zatsiorsky, 1999). For peaks occurring within the first or last 30 seconds, the window for this calculation was arbitrarily set as the first 60 seconds or last 60 seconds respectively in order to not exclude fidgets from within these regions.

For the second data processing stream, the global centre of pressure data from both force plates were utilized in conjunction with the location of the foot markers to determine the location of the centre of pressure on each participant's foot. This was done by rotating the global centre of pressure of each force plate into the local coordinate system defined by Table 3.6 (Section 3.5.2) for each foot. Equation 3.8 represents the rotation and translation of the centre of pressure values in the global frame of reference into the foot's frame of reference.

$$[1 \quad f_X \quad f_Y \quad f_Z] = [1 \quad CoP_X \quad CoP_Y \quad CoP_Z] \cdot \begin{bmatrix} 1 & D_X & D_Y & D_Z \\ 0 & cos_{Xx} & cos_{Xy} & cos_{Xz} \\ 0 & cos_{Yx} & cos_{Yy} & cos_{Yz} \\ 0 & cos_{Zx} & cos_{Zy} & cos_{Zz} \end{bmatrix} \quad \textbf{(3.8)}$$

The x, y and z components of the centre of pressure in the global coordinate system are CoP_X, CoP_Y and CoP_Z respectively; the x, y and z components of the centre of pressure in the foot local system are f_X, f_Y and f_Z respectively. Within the 4 by 4 matrix of Equation 3.8, the capital letter subscripts (X, Y and Z) represent the axis of the Optotrak global coordinate system, and the lower case letter subscripts (x, y and z) represent the axis of the foot local coordinate system. D_x, D_y and D_z represent the x, y and z components of the vector connecting the origin of the global coordinate system with the origin of the foot (see Equation 3.9).

$$\vec{\boldsymbol{D}} = \overrightarrow{\boldsymbol{Origin}}_{foot} - \overrightarrow{\boldsymbol{Origin}}_{Global} \quad \textbf{(3.9)}$$

The mean and standard deviation of the anterior/posterior and medial/lateral centre of pressure location relative to the foot system were computed on each 15 minute block. Figure 3.6 depicts an approximate layout of the various reference systems to aid in visualizing the transformation sequences.

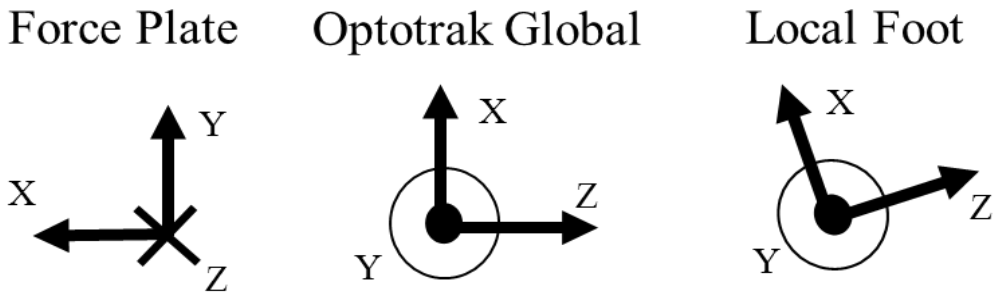

Figure 3.6: Relative Orientation of the Three Different Coordinate Systems. A top-down view of the force plate, Optotrak and local foot coordinate systems; anterior is towards the top of the figure. All centre of pressure data were transformed from the force plate to the global system prior to computations. Postural shifts and fidgets were performed on data combined from the left and right feet in the global system. To map the centre of pressure location onto each foot, the uncombined global centre of pressures were then transformed into each local foot system, and basic statistics were computed in 15 minute blocks.

To summarize, force plate data was used to accomplish three main goals. Firstly, dynamic weight support strategies in the medial/lateral direction were quantified using body weight transfers. Secondly, dynamic weight support strategies in the anterior/posterior direction were quantified using postural shifts and fidgets computed from a combined centre of pressure. Lastly, more static position and longer term movement patterns were quantified using centre of pressure locations mapped to the participant's feet.

LVDT data from each ABC was filtered using a 4^{th} order, zero lag low pass Butterworth filter at a 6 Hz cut-off. The maximum tension of the last two seconds (after the ramp period) from the filtered LVDT data of each ABC was selected as the force output of that five second contraction. The force values for each participant were expressed as a percentage of the baseline value (immediately post instrumentation value) within each session. The difference between the normalized force recordings on the control and fatigue sessions for each time point and participant was computed, termed the force residual. A higher force residual indicates greater levels of force losses relative to the control session.

3.5.4 Visual Analog Scales of Pain

All pain scores had the initial values from the respective regions and session removed from them. The baseline-removed pain from the low back region was used to separate participants into pain developers and non-pain developers using a threshold value of 10 mm out of 100 mm. The threshold was employed such that only one score exceeding the threshold over the entire two hour exposure of the control condition was used to separate pain and non-pain developers. The low back VAS scores were also used to separate whether or not a participant's pain development is sensitive to the fatigue session. Those who altered their pain reporting and changed their pain status based on the 10 mm pain threshold from the control to fatigue sessions were classified as being fatigue

sensitive, while those who did not were classified as fatigue insensitive. The four different possible categories based on these two dichotomies are shown in Table 3.7. For example, a participant reporting above 10/100 mm on the control but not the fatigue condition was classified as a fatigue sensitive pain developer.

Table 3.7: Classification of Participants Based on Pain Reporting Between the Control and Fatigue Conditions. A sensitivity in pain reporting to the effects of dominant side hip abductor fatigue has been added to the previous classification systems used in previous studies on pain development in prolonged standing (Gregory and Callaghan, 2008).

	Reports *Less than 10 mm* in Fatigue Condition	**Reports *10 mm or Greater* in Fatigue Condition**
Reports *Less than 10 mm* in Control Condition	Fatigue Insensitive, Non-Pain Developer (FI-NPD)	Fatigue Sensitive, Non-Pain Developer (FS-NPD)
Reports *10 mm or Greater* in Control Condition	Fatigue Sensitive, Pain Developer (FS-PD)	Fatigue Insensitive, Pain Developer (FI-PD)

A synopsis of all proposed data processing outcomes with a brief description of intent is included in Table 3.8.

Table 3.8: Summary of Data Processing for the Proposed Project.

Dependent Variable (#)		Source	Time Points	Rationale
EMG	*Cocontraction Indices (10)*	Standing Protocol (down sampled)	Computed every minute, averaged over 15 minute blocks.	Activation-sensitive measure of co activity between muscle pairs.
	Gaps Analysis (12)	Standing Protocol	Computed in 15 minute blocks.	Quantifiable measure in identifying muscles as a source of pain.
	Mean Power Frequencies (12)	Abduction Contractions and Fatigue Protocol	Every ABC and during the fatigue protocol (see text)	Estimating fatigue and recovery.
	Total Contraction Level at Peak GMD Activity	Fatigue Protocol	During the fatigue protocol (see text)	Determining differences in activation strategies during the fatigue protocol
Motion Capture	*Segment Angles (4)*	Standing Protocol	Basic statistics (mean, max, min, range) computed over 15 minute blocks.	Changes in spine and hip posture over discrete blocks
Force Plate	*Shifts* *Fidgets* *Body Weight Transfers*	Standing Protocol	Computed over an entire session, then separated into 15 minute blocks.	Patterns of lower limb loading and unloading.
Location of Centre of Pressure on the Foot		Standing Protocol	Basic statistics computed over 15 minute blocks.	Differences in force transmission up the leg.
LVDT Tension		Abduction Contractions	Every ABC (see text).	Estimating fatigue and recovery.
Visual Analog Scores (3)		Standing Protocol	Taken once every 15 minutes.	Quantifying musculoskeletal pain and separating pain development groups.

3.6 Statistics

Statistical processing was performed using SAS (ver. 9.4, The SAS Institute, Cary NC, USA). Computed dependent variables were input into general linear models. Each general linear model had between-subject factors of pain status (pain developer/non-pain developer) and gender as well as within-subject factors of session (control/fatigue) and time (point within the standing protocol – every 5 minutes for correlations, at ABC time points for LVDT tension and mean power frequency, and every 15 minutes for all other variables). For dependent variables only measured on one of the sessions (all variables exclusive to the fatigue protocol) or only occurring at one time point (active hip abduction test), those factors were not tested. For all procedures, an alpha level of 0.05 was used for determining significance. Each muscle and muscle pair was assumed to be independent of every other muscle for gap measures and cocontraction indices respectively. Additionally, Mauchly's test was performed on datasets to test if the assumption of sphericity was violated, using Huynh-Feldt adjusted p-values in those cases to determine significance. When relevant, post-hoc testing was performed using Tukey's Honest Significant Difference Tests.

Section 4: RESULTS

The results are split into three major parts identified by capitalized headings. First, results concerning the delineation of participant pain groups are presented (initial documentation and low back VAS scores), driving later analyses of data. Second, the results concerned with quantifying muscle fatigue in the fatigue session compared to the control session are presented (muscle activity during the fatigue protocol and ABCs and hip abductor force generation). Lastly, results concerning the standing protocols on both sessions are presented in the context of participant groups and fatigue levels (EMG, kinematic and force plate variables). Independent variables are capitalized and italicized when referred to in text. Numerical data in tables are presented as: means (standard deviations); error bars on figures show standard deviations unless specified in the caption.

4.1 DEFINING PARTICIPANT GROUPS

Sixteen (8 male, 8 female) of 40 participants were classified as PDs based on low back VAS pain scores during the control session. There were no differences in age ($p = 0.7108$), height ($p = 0.2093$), mass ($p = 0.4866$) or initial VAS scores ($p = 0.2526$) between pain groups (Table 4.1). There was a tendency of NPDs to have higher baseline strength values than PDs ($p = 0.0920$). As expected, males were taller ($p = 0.0226$), had more mass ($p = 0.0242$), and had higher baseline strength values ($p < 0.0001$) than females.

There were *GENDER*PAIN* interactions in VAS scores during standing driven by female PDs reporting higher levels of low back pain than male PDs: 1) in the low back in all standing time blocks, 2) in the right gluteal region from the 15 to 90 minute time blocks, and 3) in the left gluteal region from pre-standing to the 60 minute time block (See Table 4.2 for specific probabilities and values).

Table 4.1: Demographic and Baseline Pain Information on Pain and Non-Pain Developers.
A main effect of *GENDER* ($p < 0.05$) is indicated by an asterisk.

		Males		Females		Combined	
		PD	***NPD***	***PD***	***NPD***	***PD***	***NPD***
Age (years)		24.5 (3.6)	23.2 (1.7)	22.4 (1.7)	22.8 (3.6)	23.4 (3.0)	23.0 (2.8)
Height (m)*		1.83 (0.07)	1.82 (0.05)	1.67 (0.04)	1.64 (0.06)	1.75 (0.10)	1.73 (0.11)
Mass (kg)*		85.1 (16.2)	85.0 (10.0)	63.1 (6.0)	61.3 (10.8)	74.1 (16.4)	73.2 (15.8)
Baseline Strength (N)*	*Control*	293.4 (39.5)	327.0 (73.7)	192.0 (49.2)	230.4 (61.1)	242.7 (67.6)	278.7 (83.2)
	Fatigue	327.2 (62.6)	340.4 (93.7)	208.56 (56.1)	230.6 (55.4)	267.9 (84.0)	285.5 (94.6)
Control Baseline Pain (mm)	*Low Back*	0.8 (2.0)	1.2 (4.1)	0.0 (0.0)	0.0 (0.0)	0.4 (1.5)	0.6 (2.9)
	R-Gluteal	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	0.0 (0.1)	0.0 (0.0)	0.0 (0.1)
	L-Gluteal	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)
Fatigue Baseline Pain (mm)	*Low Back*	0.1 (0.3)	0.5 (1.4)	1.9 (4.8)	0.0 (0.0)	1.0 (3.5)	0.3 (1.0)
	R-Gluteal	0.1 (0.3)	0.1 (0.2)	0.3 (0.6)	0.0 (0.0)	0.2 (0.5)	0.0 (0.2)
	L-Gluteal	0.1 (0.3)	0.0 (0.1)	0.3 (0.6)	0.0 (0.0)	0.2 (0.5)	0.0 (0.1)

Dividing PDs by gender revealed different pain reporting patterns during the fatigue session (Figure 4.1). In the fatigue session, female PDs showed a curve of similar slope as during the control session, but with a lower y-intercept. Male PDs showed a curve of similar slope and intercept up to the second hour of standing, where pain scores during the fatigue session plateau rather than continually increase as in the control session. The different shapes of the curves suggested different responses to the fatigue interventions as a result of *GENDER*.

Table 4.2: Gender by Pain Status Interaction Effects of Low Back and Gluteal Pain. P-values indicated when significant. The Control and Fatigue sessions are combined to highlight gender by pain group differences. All values are in mm.

Pain Developers		**Pre-Stand**	**15 min**	**30 min**	**45 min**	**60 min**	**75 min**	**90 min**	**105 min**	**120 min**
Low Back	*Male*	0.5 (1.1)	**0.7 (1.3)**	**1.5 (1.8)**	**3.0 (2.0)**	**5.6 (4.2)**	**7.9 (4.0)**	**9.0 (3.2)**	**11.4 (5.2)**	**15.1 (12.6)**
	Female	0.3 (4.8)	**4.5 (6.0)**	**8.3 (9.9)**	**12.8 (9.6)**	**16.3 (12.7)**	**18.8 (14.0)**	**23.8 (14.3)**	**25.9 (17.8)**	**30.9 (20.6)**
	p-value	-	0.0416	0.0137	0.0001	0.0017	0.0041	0.0001	0.0016	0.0025
Right Gluteal	*Male*	**2.7 (4.1)**	**2.3 (2.3)**	**2.1 (1.8)**	**3.3 (3.5)**	**3.9 (3.6)**	**4.7 (4.7)**	**5.4 (4.6)**	8.5 (7.9)	9.8 (9.3)
	Female	**15.5 (24.9)**	**9.8 (11.1)**	**8.4 (7.9)**	**7.6 (8.2)**	**9.6 (8.5)**	**11.3 (9.4)**	**13.3 (11.0)**	11.9 (11.3)	15.7 (15.1)
	p-value	0.0295	0.0020	0.0006	0.0150	0.0032	0.0063	0.0022	-	-
Left Gluteal	*Male*	0.5 (1.1)	**1.2 (1.2)**	**1.6 (1.7)**	**2.7 (2.3)**	**2.9 (2.3)**	**6.3 (7.0)**	**5.8 (5.4)**	8.6 (7.7)	10.0 (9.8)
	Female	9.6 (18.0)	**5.7 (7.8)**	**5.7 (6.7)**	**6.6 (6.9)**	**9.3 (8.8)**	**9.8 (10.1)**	**10.7 (9.0)**	10.6 (10.0)	13.5 (13.2)
	p-value	-	0.0112	0.0110	0.0090	0.0003	0.0822	0.0192	-	-
Non-Pain Developers		**Pre-Stand**	**15 min**	**30 min**	**45 min**	**60 min**	**75 min**	**90 min**	**105 min**	**120 min**
Low Back	*Male*	0.6 (5.9)	-0.6 (2.3)	0.0 (3.5)	-0.1 (4.1)	-0.3 (3.3)	-0.2 (4.3)	0.4 (4.4)	-0.1 (4.3)	0.6 (4.8)
	Female	1.8 (8.5)	0.3 (1.8)	0.9 (2.3)	0.2 (1.1)	0.8 (2.3)	0.8 (3.1)	0.5 (1.3)	1.1 (1.6)	0.5 (1.4)
	p-value	-	-	-	-	-	-	-	-	-
Right Gluteal	*Male*	11.1 (22.5)	2.4 (8.6)	2.1 (4.5)	2.9 (7.2)	1.2 (3.6)	2.0 (7.6)	1.8 (5.0)	2.4 (6.5)	2.8 (8.1)
	Female	7.9 (16.3)	0.7 (2.7)	1.1 (3.3)	0.8 (1.8)	0.4 (1.4)	1.0 (2.2)	0.8 (1.9)	0.7 (1.8)	1.0 (2.7)
	p-value	-	-	-	-	-	-	-	-	-
Left Gluteal	*Male*	5.7 (17.2)	1.2 (4.3)	2.0 (2.9)	3.6 (6.8)	3.6 (5.4)	3.0 (4.8)	3.2 (5.6)	4.0 (6.2)	3.6 (7.9)
	Female	4.5 (15.2)	0.9 (2.6)	0.5 (0.8)	1.2 (1.4)	1.0 (2.0)	0.9 (2.0)	1.1 (2.2)	1.1 (1.9)	2.0 (3.1)
	p-value	-	-	-	-	-	-	-	-	-

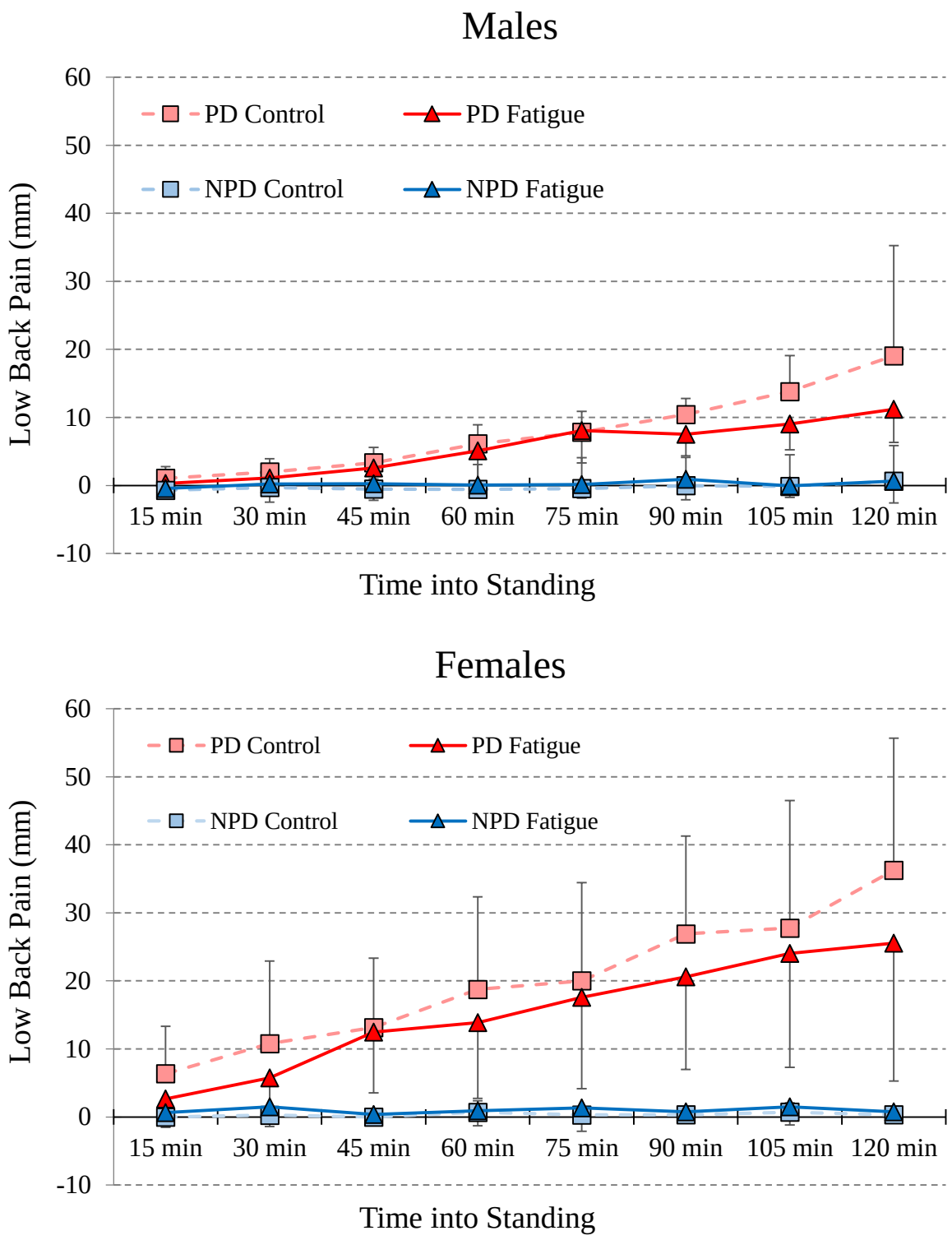

Figure 4.1: Low Back Pain VAS Scores. Pain groups were separated based on a 10 mm threshold. Male and female pain developers showed different pain reporting patterns on the fatigue session: males had similar levels to the control session during the first hour and plateaued after 75 minutes while females had lower scores on the fatigue session with no plateauing.

There was a significant main effect of *PAIN* on peak low back pain changes between sessions (Figure 4.2). PDs reported systematic pain reductions in the fatigue session (Male PD: 11.5 ± 15.7 mm; Female PD: 10.3 ± 5.1 mm) whereas NPDs reported minimal peak pain changes of varying polarity (Male NPD: -0.9 ± 4.4 mm; Female NPD: -2.1 ± 3.1 mm; $p < 0.0001$) between sessions. There were no *GENDER* ($p = 0.3585$) or *GENDER*PAIN* ($p = 0.9990$) effects on peak low back pain.

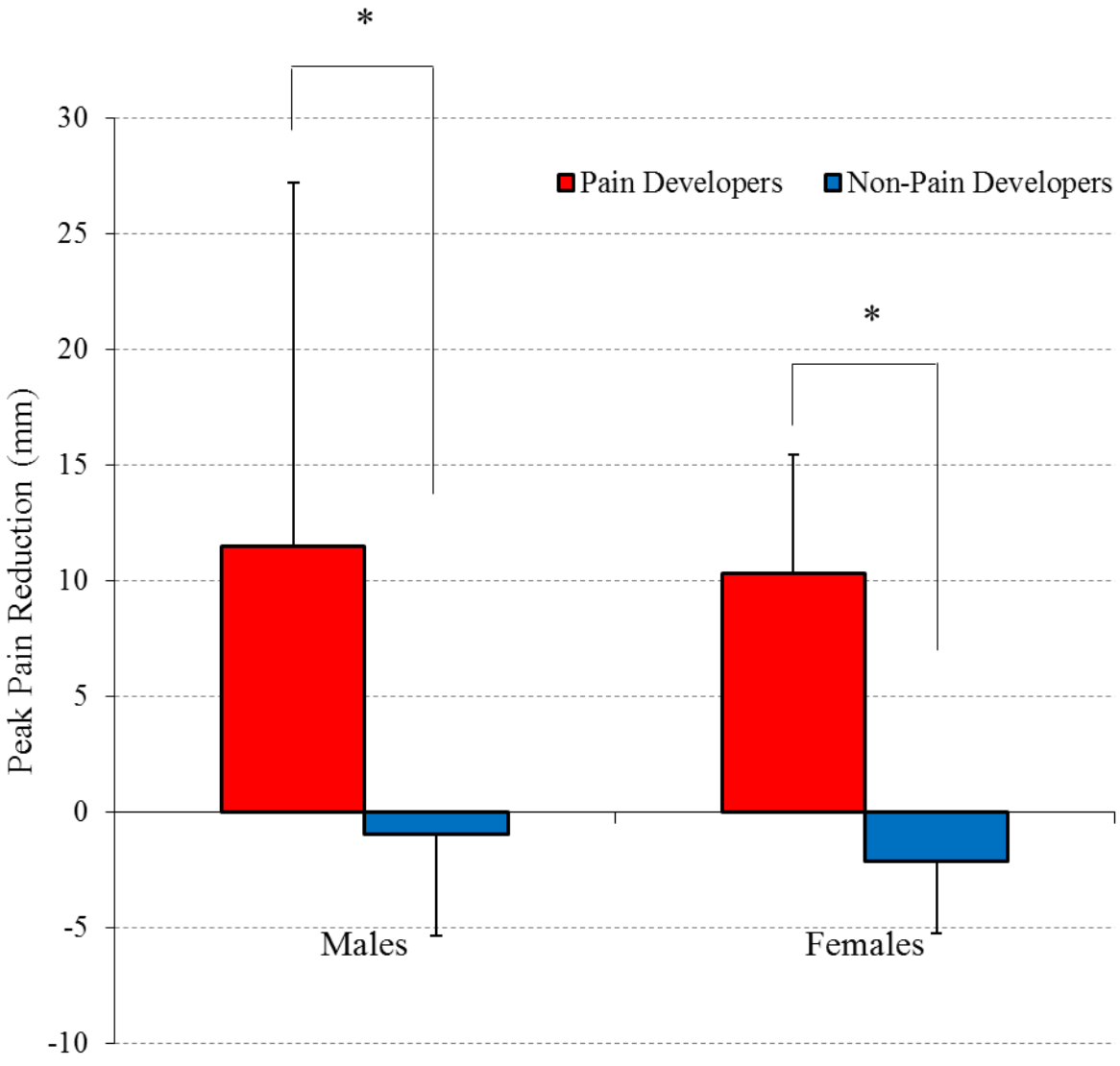

Figure 4.2: Peak Pain Reductions between Sessions. A positive value indicates a greater score during the Control session. A significant effect of *PAIN* ($p < 0.05$) is indicated by an asterisk.

There was a *GENDER*PAIN* interaction on active hip abduction test scores with female PDs (3.06 ± 1.01) having higher self-rated scores than female NPDs (0.88 ± 1.38; p = 0.0019). The experimenter rated scores had a similar trend in the same direction (Female PDs: 1.25 ± 0.66; Female NPDs: 0.75 ± 0.72; p = 0.1459). There was no such trend with males (Male PD: Self = 1.69 ± 1.20, Experimenter = 1.13 ± 0.60; Male NPD: Self = 1.79 ± 1.49, Experimenter = 1.33 ± 0.75; p > 0.5386). According to the criteria of Nelson-Wong et al. (2009), five of eight female and two of eight male pain developers would have been correctly identified on the basis of the active hip abduction test.

Five PDs (3 male, 2 female) and one female NPD were classified as being fatigue sensitive based on low back VAS scores between sessions, and all forty participants passed Ober's test. As a result of the skewed distribution of the proposed groups, effects of fatigue sensitivity and iliotibial band tightness were not investigated. The leg dominance test classified three participants as left leg dominant: one male PD and two female NPDs.

4.2 QUANTIFYING MUSCLE FATIGUE AND RECOVERY

4.2.1 Immediate Effects of the Fatigue Protocol

Ratings of perceived exertion upon completion of the fatigue protocol were similar across gender and pain groups (Male PDs: 8.8 ± 0.4 out of 10; Male NPDs: 9.1 ± 0.7; Female PDs: 9.3 ± 0.5; Female NPDs: 9.2 ± 1.1; p = 0.1260). Females had longer times to fatigue than males (Males: 17.2 ± 7.1 minutes; Females: 21.7 ± 12.5 minutes; p = 0.0031), and had smaller percent force losses immediately following fatigue based on differences between the 0 minute ABC in the control and fatigue session (Males: 12.8 ± 11.1 %Baseline; Females: 7.2 ± 10.4 %Baseline; p = 0.0500). Additionally, there was a main effect of *PAIN* with NPDs having longer times to fatigue than PDs (PDs: 18.7 ± 9.3 minutes; NPDs: 20.1 ± 11.2 minutes; p = 0.0106). In addition to the

failure criteria listed in Table 3.5, every participant exhibited three other compensations: 1) external rotation of the dominant limb during limb ascent, 2) slight knee flexion during limb ascent and descent and, 3) pushing down into the table with the non-dominant limb as observed by the table cushion deforming upon limb ascent.

EMG data collected during the fatigue protocol revealed uniform decreases in mean power frequencies for both GMDs and TFLs with no significant differences detected as a result of *GENDER* ($p > 0.1865$), *PAIN* ($p > 0.2591$) and *GENDER*PAIN* ($p > 0.1738$; Table 4.3). There appeared to be a trend of male NPDs having greater mean power frequency decreases in the non-dominant TFL than Male PDs ($p = 0.1738$) in response to the fatigue protocol. Mean power frequency data from a sample participant's hip abductors are shown in Figure 4.3.

Table 4.3: Mean Power Frequencies of Hip Abductor Muscles in the Final Duty Cycle of the Fatigue Protocol. Values are expressed as a percentage of the mean power frequency during the initial duty cycle. There were no pain or gender group differences ($p > 0.05$).

Muscle		Males		Females	
		PD	***NPD***	***PD***	***NPD***
Dominant	*GMD*	69.95 (15.55)	71.74 (12.11)	74.22 (18.12)	76.07 (13.45)
	TFL	78.45 (13.04)	75.46 (17.59)	83.00 (9.10)	79.74 (14.91)
Non-Dominant	*GMD*	86.98 (9.34)	81.68 (17.50)	77.35 (11.34)	78.18 (12.52)
	TFL	66.12 (23.44)	60.55 (19.20)	68.43 (18.82)	68.48 (12.95)

Muscles other than the hip abductors were affected sporadically by the fatigue protocol. Some participants only exhibited hip abductor fatigue while other participants had myoelectric evidence of muscle fatigue in up to five of the eight other muscles measured (Table 4.4). Muscles were determined to be fatigued as a result of the protocol if they were activated to at least 30 %MVIC (in order for a reliable mean power frequency measure; De Luca, 1997) and had a relative decrease in mean power frequency of at least 10% in the last measured duty cycle compared to the

first duty cycle (Chaffin, 1973). Both hip abductors for all participants satisfied the above metrics for being considered fatigued. The limited number of muscles measured is by no means comprehensive as any number of unmeasured muscles could have also satisfied this criteria for fatigue. The R-EXO (10 participants), R-LES (8 participants) and R-INO (6 participants) were the muscles most frequently found to also meet this criteria.

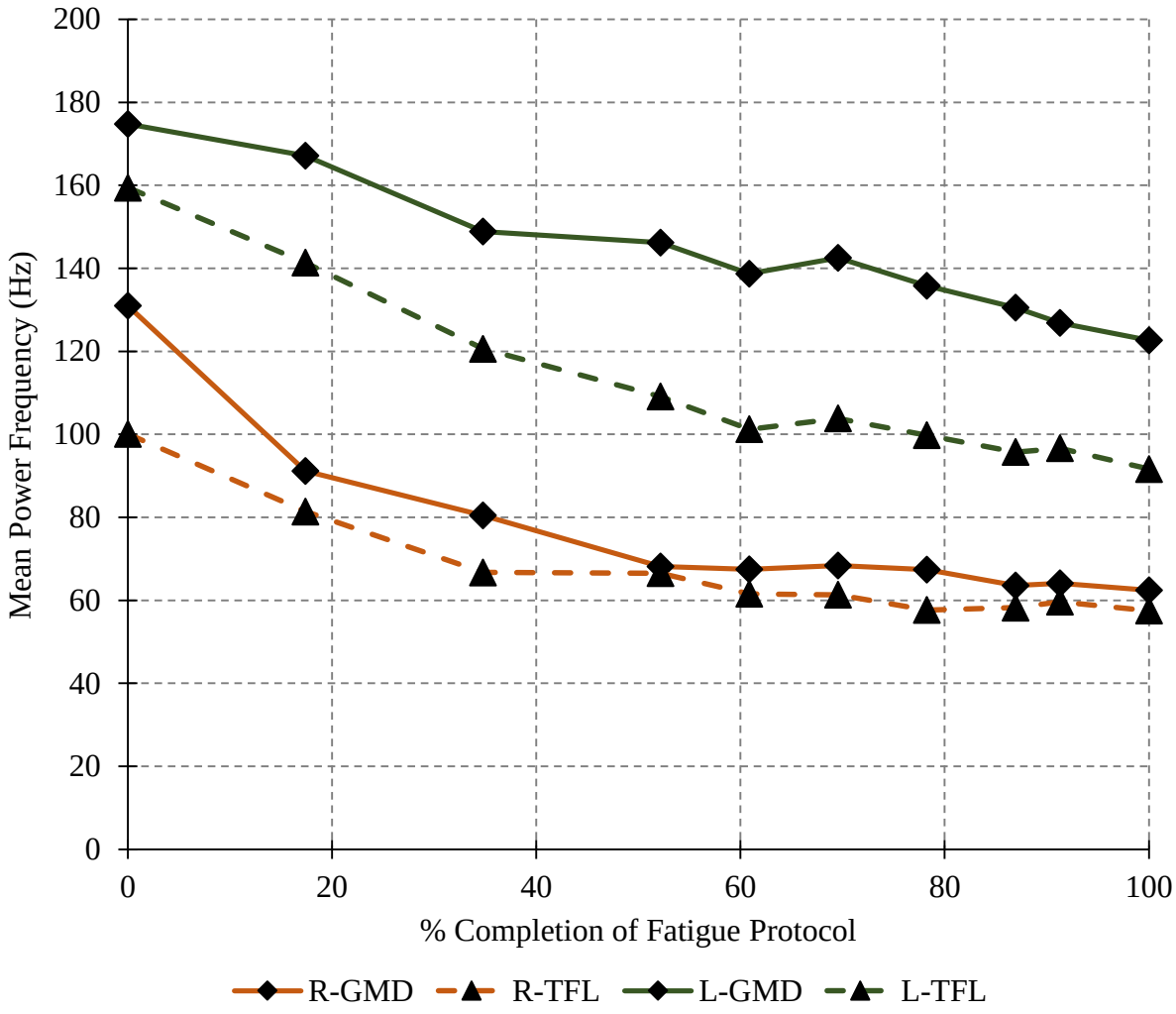

Figure 4.3: Decreases in Hip Abductor Mean Power Frequencies during the Fatigue Protocol. Sample data is from a right-leg dominant Male PD, the time to fatigue was 11 minutes and 30 seconds. Similar curves were observed for this participant's R-EXO and R-INO, however this was not consistent across genders or pain groups.

Table 4.4: Additional Muscles Fatigued as a Result of the Fatigue Protocol. Within each column, the number of fatigued muscles in addition to both GMDs and TFLs from the protocol experienced by each participant are presented. There were a total of eight other muscles measured.

Male PDs		Male NPDs		Female PDs		Female NPDs	
EG6	3	GI0	1	HP2	1	LP8	0
FX9	0	FZ1	0	FY7	2	FR3	0
CD9	0	MX0	2	MZ6	0	XY5	2
DD7	1	MY7	2	XP4	0	PC0	0
MP4	0	TB4	2	DX7	0	AD4	1
OP5	0	BW5	0	OK3	1	NA8	0
LY7	2	RT2	2	QA2	5	PM7	2
AJ5	1	WC3	0	EZ3	0	MS5	0
		CT6	0			SR0	0
		RV5	0			IQ6	1
		JP3	2			VG4	0
		KL2	0			EW1	0

There was a *GENDER*PAIN* interaction for total muscle activity at the instance of peak dominant GMD activation during the 1/3 to completion duty cycle where Female PDs had greater summed activity than Female NPDs ($p = 0.0261$; Figure 4.4). There was a non-significant trend in the same direction during the 2/3 to completion duty cycle ($p = 0.0829$). Additionally, male NPDs had greater total muscle activity at the instance of peak dominant GMD activation than female NPDs during the 2/3 to completion duty cycle ($p = 0.0277$). Although not significant, male NPDs tended to have higher levels of activity than male PDs, ($p = 0.1570$) contrary to females where the reverse was found (NPD < PD). There was a main effect of *TIME* where total muscle activity increased with percentage completion ($p < 0.0001$).

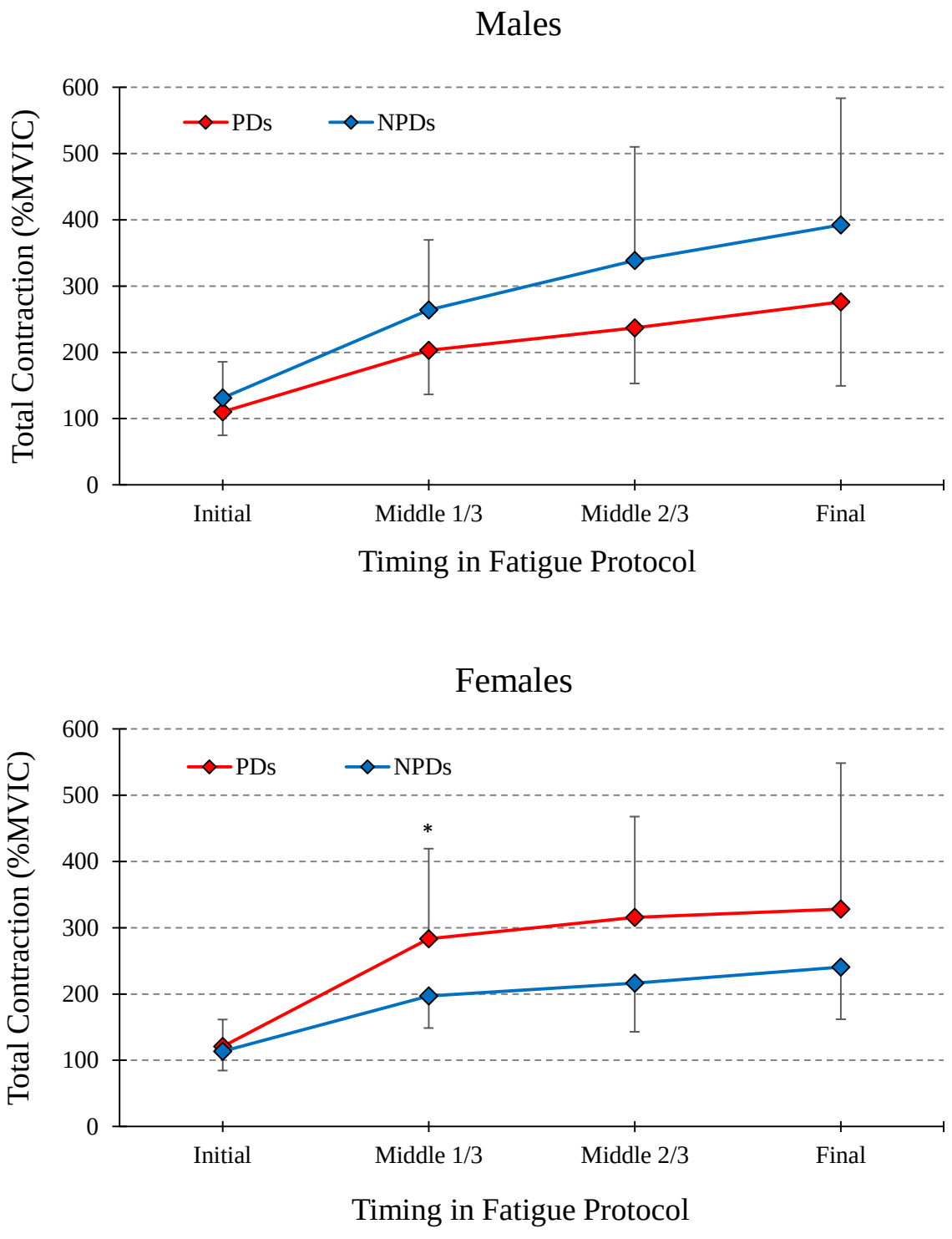

Figure 4.4: Total Contraction Levels at the Instance of Peak Dominant GMD Activity during the Fatigue Protocol. In males, NPDs tended to have greater activity levels than PDs, while in females, NPDs tended to have less activity than PDs. Significance ($p < 0.05$) is indicated by an asterisk.

4.2.2 Fatigue and Recovery during Standing

There was a *PAIN*SESSION* interaction for ABC force values during standing. Both NPDs and PDs had lower relative force in the fatigue session at time points 0 minutes to 3 minutes. After 3 minutes, NPDs relative force values were similar between sessions, however PDs relative force was lower in the fatigue session throughout the entire two hours, reaching significance at all ABC time-points except for at the 5 minute ($p = 0.0742$), 45 minutes ($p = 0.0743$) and 75 minute ABCs ($p = 0.0630$; Figures 4.5a and 4.5b).

The force residual (control session minus fatigue session) was different between pain groups at the 105 minute (PDs: 15.27 ± 20.14; NPDs: 1.42 ± 14.78; $p = 0.0216$) and 120 minute ABC time-points (PDs: 16.44 ± 20.86; NPDs: -1.43 ± 15.50; $p = 0.0044$) with PDs having a larger residual than NPDs (Figure 4.6).

There were *TIME*SESSION* interactions in mean power frequencies computed on the EMG data from the ABCs. During the fatigue session, the zero minute ABC (immediately post-fatigue) had a lower mean power frequency in the dominant GMD ($p = 0.0073$), dominant TFL ($p = 0.0055$) and non-dominant GMD ($p = 0.0376$) than all other time blocks in that session (Table 4.5). The non-dominant TFL did not share this pattern ($p = 0.6426$).

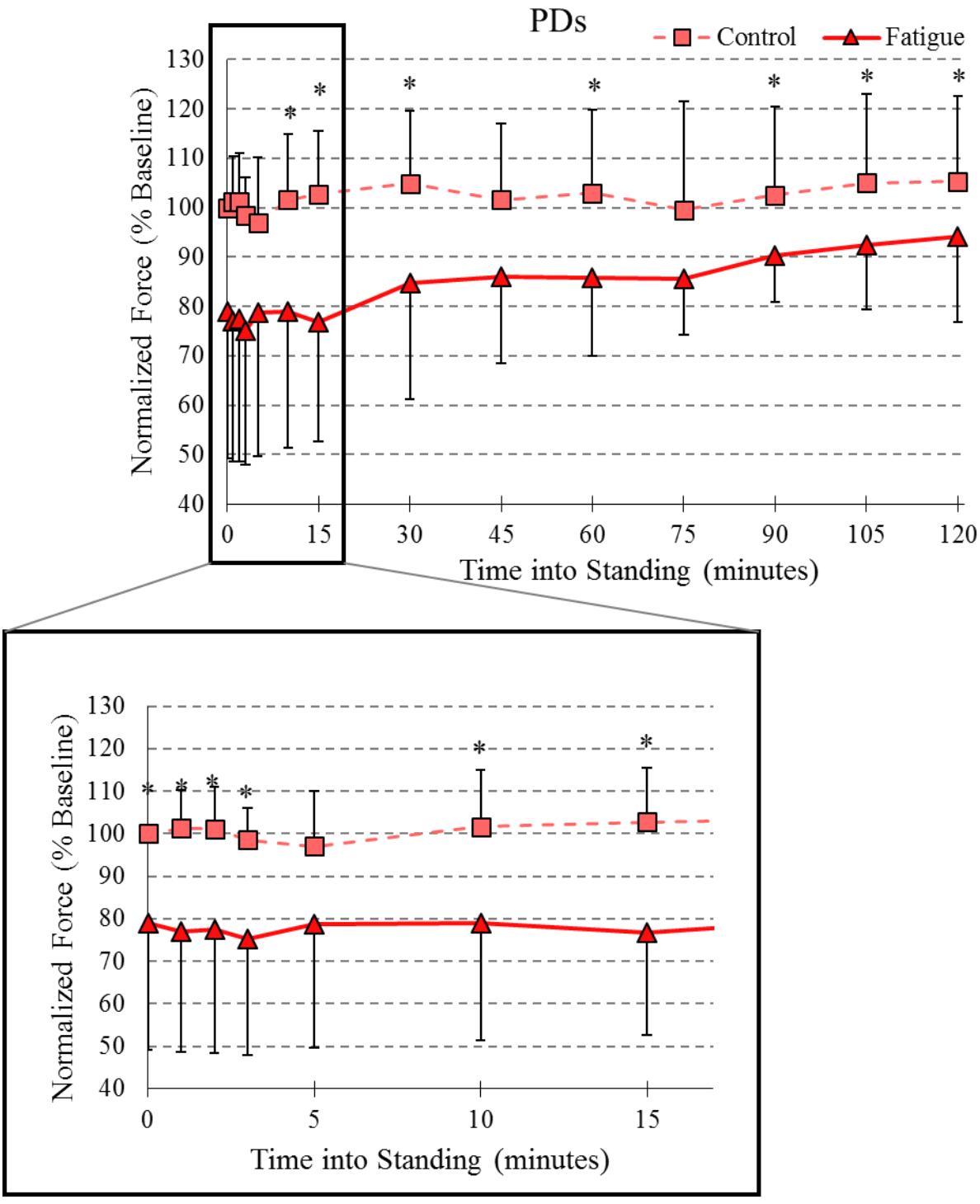

Figure 4.5a: Differences in Normalized Force in Pain Developers between Sessions. The upper panel shows the entire 2 hours on both sessions, the bottom panel is zoomed in on the first 15 minutes. Effects of *SESSION* ($p < 0.05$) are indicated by an asterisk.

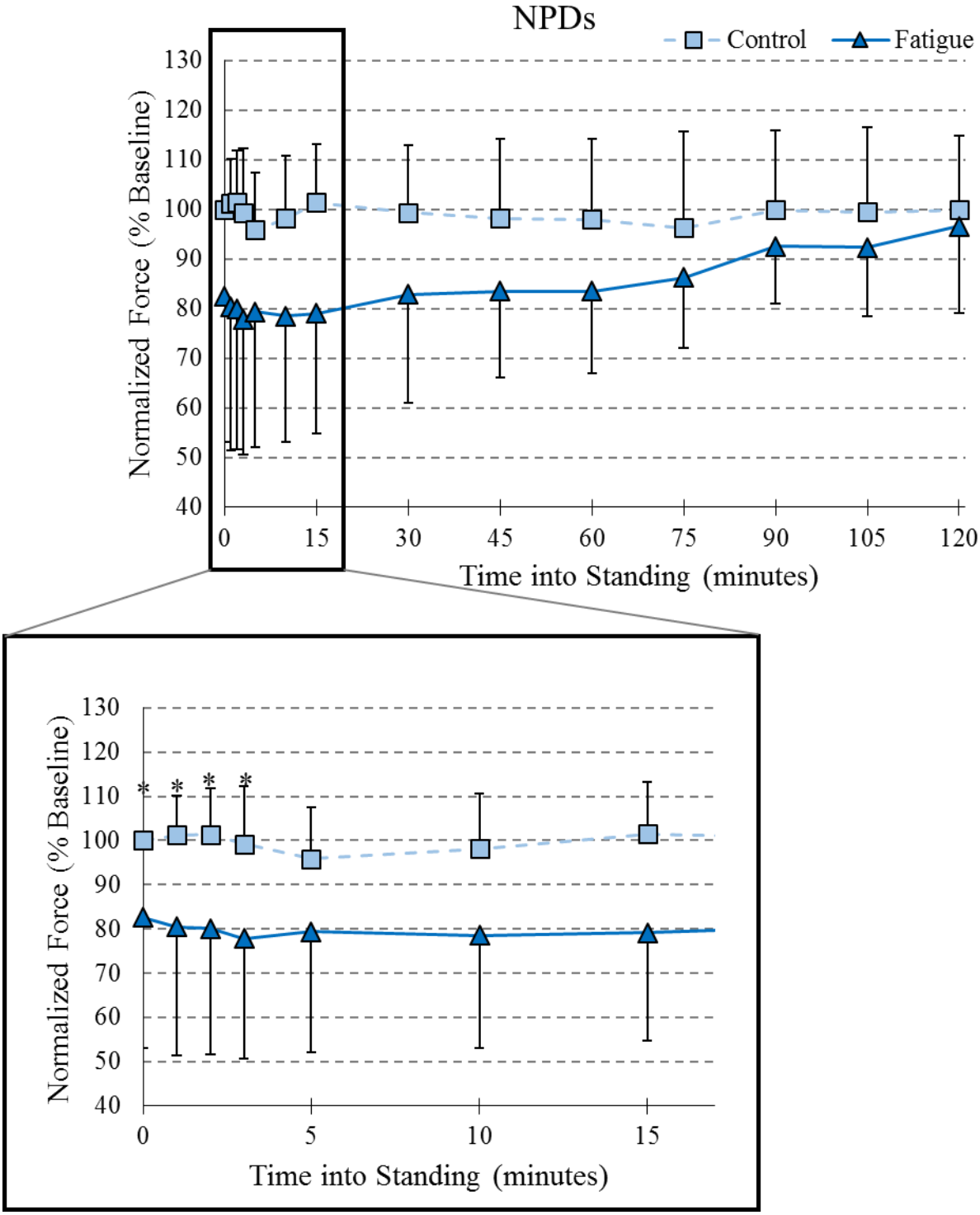

Figure 4.5b: Differences in Normalized Force in Non-Pain Developers between Sessions. The upper panel shows the entire 2 hours on both sessions, the bottom panel is zoomed in on the first 15 minutes. Effects of *SESSION* ($p < 0.05$) are indicated by an asterisk.

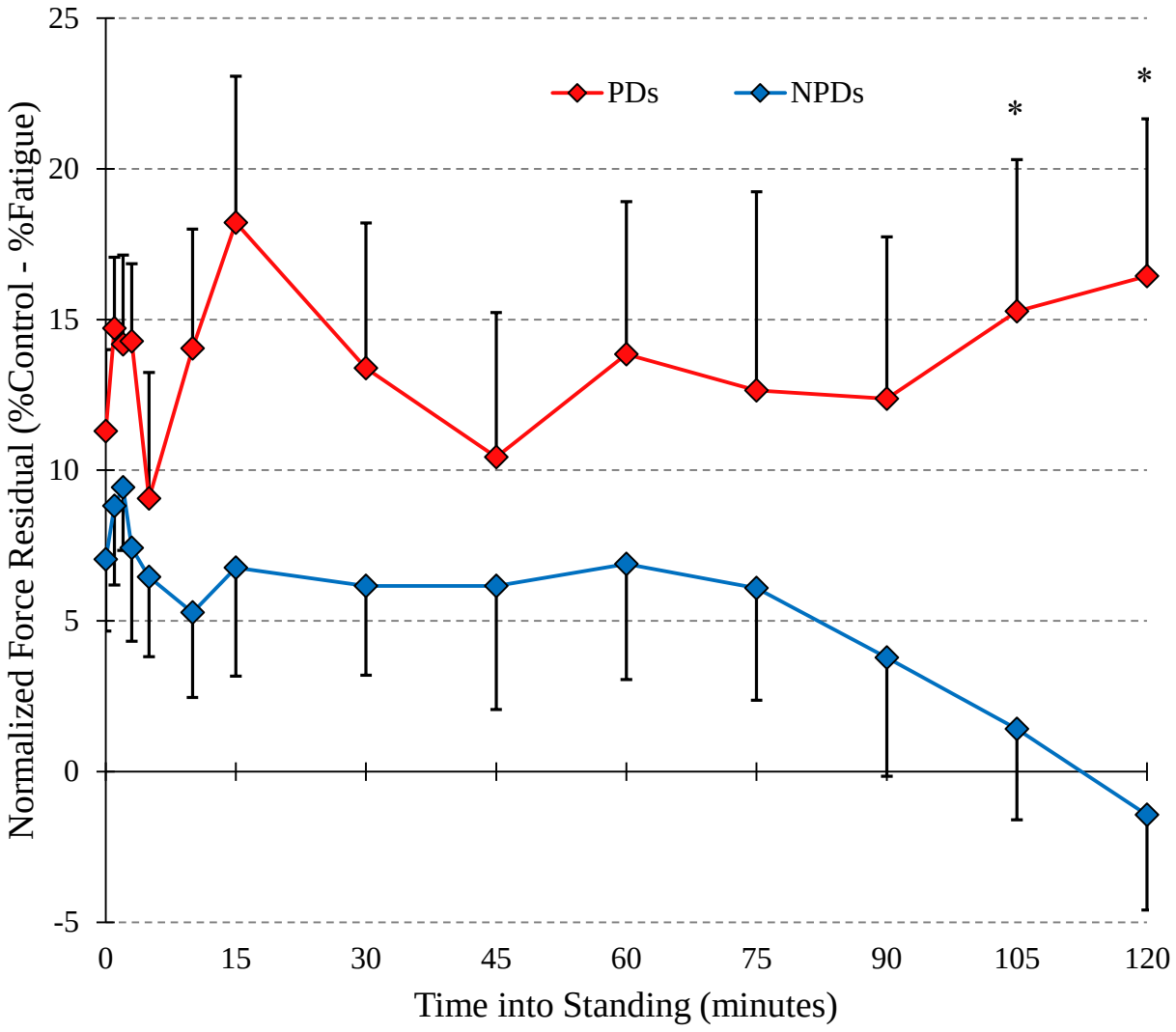

Figure 4.6: Normalized Force Residual in PDs and NPDs. A more positive number indicates greater force losses with fatigue. The error bars show standard errors rather than standard deviations. A main effect of *PAIN* ($p < 0.05$) is indicated by an asterisk.

Table 4.5: Initial Decreases in Mean Power Frequencies following the Fatigue Protocol. Mean power frequencies are expressed as a percentage of the initial ABC performed prior to the fatigue protocol. Only the zero minute (post-fatigue), 1 minute and 120 minute (final) ABCs during the fatigue session are shown to reduce clutter. ABCs at other points had similar values as the one and 120 minute ABCs. Significant *TIME*SESSION* interactions ($p < 0.05$) are indicated with asterisks.

Muscle		Post-Fatigue ABC	One Minute ABC	Final ABC
Dominant	***GMD****	87.9 (15.9)	95.6 (17.0)	102.0 (30.4)
	TFL*	90.2 (21.3)	97.6 (19.9)	99.4 (24.8)
Non-Dominant	***GMD****	93.7 (14.5)	100.8 (14.1)	101.0 (25.0)
	TFL	99.5 (20.3)	105.0 (21.0)	102.9 (22.1)

Gluteal VAS scores were higher in the fatigue session compared to the control session immediately following the fatigue protocol. This difference was found on both sides immediately prior to standing (for the right gluteal region, Control: 0.73 ± 2.40 mm; Fatigue: 18.22 ± 24.48 mm; $p < 0.0001$, For the left gluteal region, Control: 0.42 ± 1.20 mm; Fatigue: 9.93 ± 20.53 mm; $p = 0.0057$) and after 15 minutes of standing (for the right gluteal region, Control: 1.20 ± 3.04 mm; Fatigue: 5.51 ± 9.44 mm; $p = 0.0017$, For the left gluteal region, Control: 1.02 ± 2.02 mm; Fatigue: 3.01 ± 6.08 mm; $p = 0.0195$). The right gluteal VAS scores were also higher in the fatigue session then in the control session after 75 minutes of standing (Control: 2.76 ± 4.63 mm; Fatigue: 5.45 ± 8.63 mm; $p = 0.0289$). Reported values for gluteal pain immediately following the fatigue protocol spanned the full range of the VAS (0 to 100 mm), with six participants exceeding 50/100 mm (2 Male NPDs, 3 Female PDs, 1 Female NPD) and seven participants reporting 0/100 mm (3 Male PDs, 2 Male NPDs, 2 Female NPDs).

4.3 STANDING BEHAVIOURS AND PATTERNS

The large number of dependent variables in this project resulted in a similarly large number of findings. To ease with clarity, only those results linked to pain development or fatigue are discussed in text. A list of significant findings from this section can be found in Appendix A.

4.3.1 EMG Measures during Standing

There were *GENDER*PAIN*SESSION* interactions in cocontraction indices with differences between the control and fatigue session in male PDs occurring during the second hour of standing (Figure 4.7). For male PDs, R-GMD/R-LES CCI was greater in the control session in the 75 minute (Control: 5119.3 ± 6470.8 %MVIC; Fatigue: 1855.0 ± 1679.2 %MVIC; $p = 0.0161$), 90 minute (Control: 4639.3 ± 5936.0 %MVIC; Fatigue: 2061.8 ± 1861.9 %MVIC; $p = 0.0479$), 105 minute (Control: 4781.9 ± 5728.0 %MVIC; Fatigue: 1994.1 ± 1836.3 %MVIC; $p = 0.0386$), and 120 minute time blocks (Control: 4325.9 ± 5131.4 %MVIC; Fatigue: 1932.2 ± 2006.8 %MVIC; $p = 0.0105$). Also in male PDs, the mean R-LES/R-EXO CCI doubled in the control session in the 75 minute block and remained elevated for the last hour, an pattern that was absent from the fatigue session. This finding was not significant even though the magnitude of mean differences and standard deviations were similar to the R-GMD/R-LES CCI (at 75 minutes, Control: 4751.8 ± 6181.9 %MVIC; Fatigue: 2004.3 ± 1533.7 %MVIC; $p = 0.1230$). Closer inspection revealed that this jump was driven by one participant (FX9). Similar inspection of the R-GMD/R-LES cocontraction index showed that same participant (FX9) drove the large increase at the 75 minute mark, however in that instance, it did result in significance. The marked increases in both CCIs for FX9 appeared to have stemmed from an increase in mean R-LES activity after 75

minutes of standing (from 2.73 %MVIC to 6.06 %MVIC). A Fourier transform was unable to identify any changes in frequency content between the 60 and 75 minute time blocks. Table 4.6 expresses this group divergence in muscular responses. Removing FX9 from the analysis resulted in no *GENDER*PAIN*SESSION* interactions for either of the above CCIs ($p > 0.1283$; see Figure 4.7). Removing FX9's VAS responses from both the control and fatigue sessions did not alter the plateau response of male PDs during the fatigue session (Figure 4.1). FX9's data was removed from these two cocontraction indices (R-GMD/R-LES and R-LES/R-EXO) and gaps measures pertaining to the R-LES for all subsequent analysis (Male PD n = 7 for those measures).

Table 4.6: EMG Differences between Participant FX9 and Other Male PDs. Means from start to 60 minutes are grouped under 1st hour, and 75 to 120 minutes are grouped under 2nd hour.

Measure	FX9			Other Male PDs		
	1st Hour (%MVIC)	***2nd Hour (%MVIC)***	***Percent Increase***	***1st Hour (%MVIC)***	***2nd Hour (%MVIC)***	***Percent Increase***
R-GMD/R-LES CCI	1727.3	17659.5	922%	2753.7 (2723.7)	2867.6 (3322.7)	16% (61%)
R-LES/R-EXO CCI	3375.6	17561.3	420%	2532.4 (1579.1)	2188.3 (1596.3)	5% (80%)
R-LES Activity	2.73	6.06	173%	2.39 (1.15)	2.36 (2.12)	-16% (60%)

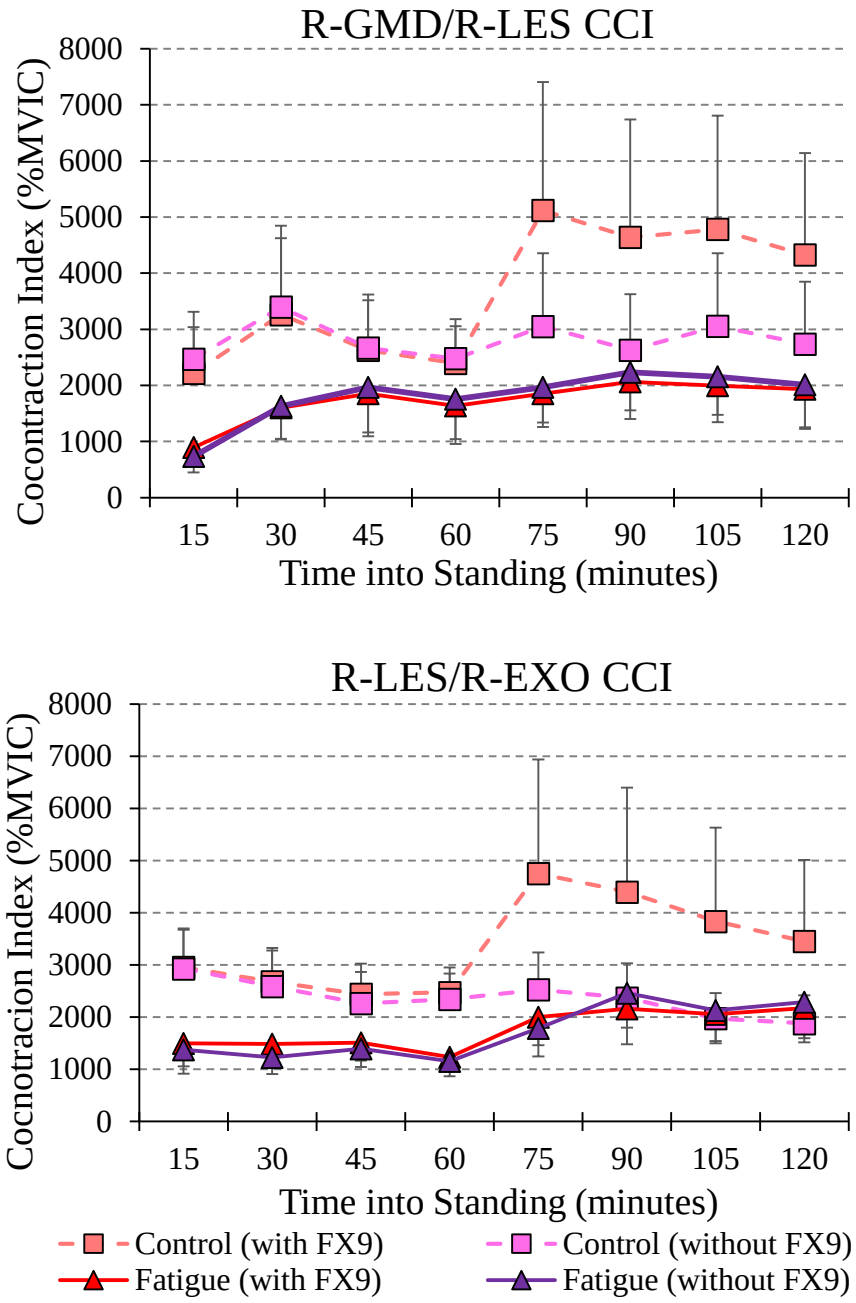

Figure 4.7: Influence of Participant FX9 on Lumbar Erector Spinae Cocontraction Indices. Initially, the R-GMD/R-LES and R-LES/R-EXO cocontraction indices had a unique response at the 75 minute mark of the control session for Male PDs. Removal of FX9 drastically altered the shape of the control session with minimal changes to the fatigue session. Means and standard errors are shown.

Changes in R-GMD/L-GMD cocontraction index were linked primarily to pain groupings. There was trend in R-GMD/L-GMD cocontraction index in the 15 minute time block where PDs had smaller CCIs in the fatigue session compared to the control session (PD control: 988.9 ± 1184.2 %MVIC; PD fatigue: 537.6 ± 731.7 %MVIC; p = 0.0888), but NPDs had larger CCIs in the fatigue session than the control session (NPD control: 604.7 ± 698.4 %MVIC; NPD fatigue: 907.0 ± 896.3 %MVIC; p = 0.0921; see Figure 4.8). Determining the difference between control and fatigue sessions for each participant (like the ABCs), revealed a main effect of *PAIN* where PDs had higher R-GMD/L-GMD residuals than NPDs in the 15 minute block (PD: 398.8 ± 792.4 %MVIC; NPD: -282.5 ± 966.6 %MVIC; p = 0.0331). There was also a *TIME*PAIN* interaction where the R-GMD/L-GMD cocontraction index was lower at 15 and 30 minutes than at 90 to 120 minutes, but only in PDs (Figure 4.8, left panel shows the pattern, Table 4.7 gives specific values). The differing responses of the two pain groups demonstrate an interaction in response to hip abductor muscle fatigue (Figure 4.8). PDs had an initial decrease in R-GMD/L-GMD CCI with fatigue that progressed to higher values than during the control session at later points in time, while NPDs shift to higher R-GMD/L-GMD CCIs with fatigue.

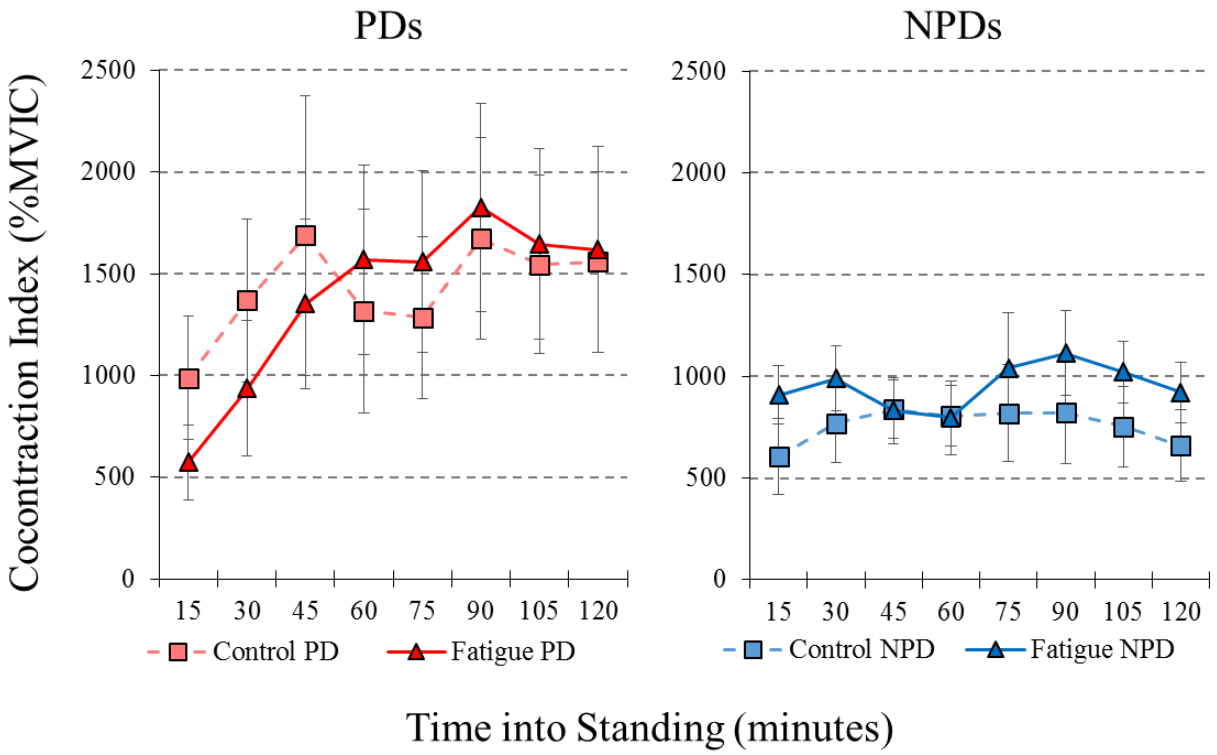

Figure 4.8: Differences in R-GMD/L-GMD Cocontraction Index in Pain and Non-Pain Developers between Control and Fatigue Sessions. Although not significant, PDs had consistently lower CCIs in the first 15 minutes of the fatigue session while NPDs had consistently higher CCIs in the first 15 minutes of the fatigue session, both are relative to the same time in the control session. Within PDs, there was an effect of *TIME* where earlier values (15 and 30 minutes) are smaller than later values (90 to 120 minutes; Table 4.7 below).

There were decreases in trunk cocontraction indices with fatigue in the first hour of the standing protocol (Figure 4.9). Compared to the control session, R-LES/R-EXO was smaller in the 15 minute block (Control: 2760.1 ± 2712.9 %MVIC; Fatigue: 1965.9 ± 2177.8 %MVIC; $p = 0.0421$), L-LES/L-EXO was smaller in the 45 minute block (Control: 3339.5 ± 3693.4 %MVIC; Fatigue: 1873.4 ± 1893.0 %MVIC; $p = 0.0285$), and R-LES/L-LES was smaller in the 30 to 60 minute blocks ($p < 0.0239$). These decreases with fatigue were consistent across *GENDER* and *PAIN* (for R-LES/R-EXO, $p > 0.2098$; for L-LES/L-EXO, $p > 0.6769$; for R-LES/L-LES, $p > 0.2805$).

Table 4.7: Time Effects of R-GMD/L-GMD Cocontraction Index in Pain Developers. The relevant time points are in bold in the grey shaded portion of the table, the CCIs at that point in time are to the right of or below each time. Probabilities within the lower right part refer to the intersection of the two time points.

PDs	**Later Time Points**	**90 Minutes**	**105 Minutes**	**120 Minutes**
Earlier Time Points	***R-GMD/L-GMD CCI (%MVIC)***	*1752.0 (1988.8)*	*1597.4 (1793.3)*	*1589.6 (1882.7)*
15 Minutes	*774.5 (999.0)*	***P = 0.0108***	***P = 0.0085***	***P = 0.0027***
30 Minutes	*1146.2 (1458.1)*	***P = 0.0286***	***P = 0.0289***	***P = 0.0092***

There was a *TIME*GENDER* effect in R-TFL/L-TFL CCI where females of both pain groups had smaller values in the first half hour of standing than the 120 minute block (15 minutes: 185.4 ± 192.2 %MVIC; 30 minutes: 221.9 ± 252.0 %MVIC; 120 minutes: 356.9 ± 399.3 %MVIC; 15 minutes < 120 minutes, p = 0.0086; 30 minutes < 120 minutes, p = 0.0402). The R-TFL/L-TFL CCI was unaffected by *SESSION* (p > 0.2007).

There was a *GENDER*PAIN* interaction in R-TES/L-TES CCI where male NPDs had higher cocontraction indices than male PDs in the 90 minute (Male PD: 2388.2 ± 2330.8 %MVIC; Male NPD: 4971.5 ± 3499.4 %MVIC; p = 0.0260) and 120 minute time blocks (Male PD: 2440.0 ± 1760.3 %MVIC; Male NPD: 4814.7 ± 3774.9 %MVIC; p = 0.0321). There was a non-significant trend of the same direction occurring in the 105 minute time block (Male PD: 2401.9 ± 1900.7 %MVIC; Male NPD: 5161.5 ± 4073.8 %MVIC; p = 0.0540). Another *GENDER*PAIN* interaction was found in R-LES/L-LES CCI where female PDs had higher CCIs than female NPDs in the 75 minute (Female PD: 4184.8 ± 3684.6 %MVIC; Female NPD: 2097.0 ± 2179.7 %MVIC; p =

0.0496), 90 minute (Female PD: 4680.6 ± 3635.3 %MVIC; Female NPD: 2278.3 ± 2200.2 %MVIC; p = 0.0273), 105 minute (Female PD: 4310.1 ± 3573.6 %MVIC; Female NPD: 2078.7 ± 2095.4 %MVIC; p = 0.0166) and 120 minute time block (Female PD: 4825.4 ± 3941.6 %MVIC; Female NPD: 1846.5 ± 1737.3; p = 0.0023). In both of the aforementioned *GENDER*PAIN* interactions, groups were initially similar but became more divergent over the standing protocol.

There were main effects of *PAIN* in the contralateral pairings of GMD and LES. In R-GMD/L-LES CCI, PDs had greater values than NPDs in the 45 minute (PDs: 1664.7 ± 1953.7 %MVIC; NPDs: 949.3 ± 952.3 %MVIC; p = 0.0431) and 60 minute blocks (PDs: 1644.9 ± 1797.8 %MVIC; NPDs: 840.5 ± 930.0 %MVIC; p = 0.0277). In L-GMD/R-LES CCI, the opposite was found in that NPDs had greater values than PDs in the 15 minute block (PDs: 2439.4 ± 2560.5 %MVIC; NPDs: 3202.6 ± 3280.4 %MVIC; p = 0.0166).

There was a *TIME*SESSION* interaction in L-GMD/L-LES CCI where in the control session, the 30 to 60 minute time blocks were higher than the 120 minute time block (30 minutes: 3670.1 ± 4025.1 %MVIC, p = 0.0183; 45 minutes: 3665.7 ± 4247.9 %MVIC, p = 0.0161; 60 minutes: 2914.4 ± 3057.4 %MVIC, p = 0.0423; 120 minutes: 1863.2 ± 2314.1 %MVIC). There was a non-significant trend in the same direction differentiating the 15 and 120 minute blocks in the control session (15 minutes: 3222.7 ± 3541.3 %MVIC, p = 0.0758). Despite L-GMD/L-LES decreasing over time in the control session but not doing so in the fatigue session, there were no *SESSION* differences in any pain or group (p > 0.1706).

Time-varying normalized EMG data is presented in Appendix B for the R-GMD/L-GMD, and R-LES/R-EXO CCIs in a sample PD and NPD to show patterns that differentiate pain groups.

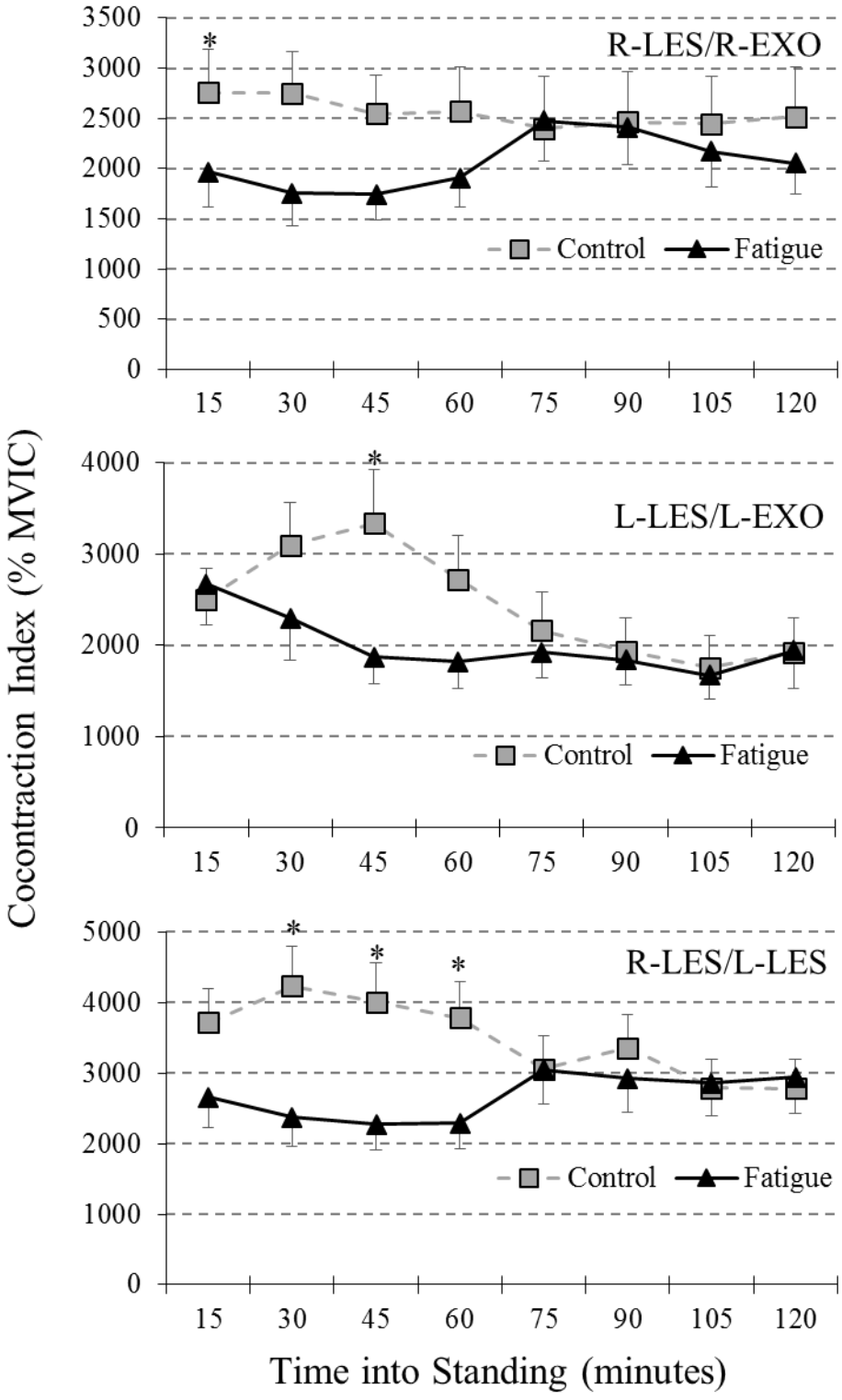

Figure 4.9: Trunk Cocontraction Indices Decreased in the First Hour of the Fatigue Session. PDs and NPDs of both genders are combined for all plots. Significant effects of *SESSION* ($p < 0.05$) are indicated by asterisks.

There were a number of differences in EMG gap measures in females. Female PDs had lower cumulative times in R-TFL in the fatigue session than in the control session for the entire two hours of standing (*GENDER*PAIN*SESSION*; $p < 0.0038$; Figure 4.10). Within NPDs, females also had lower R-TFL cumulative rest times than males throughout both sessions (*GENDER*PAIN;* $p < 0.0014$; Figure 4.10). Female PDs had decreases in R-LES cumulative times from the first 30 minutes to the last 15 minutes of the fatigue session (15 minutes: 346.4 ± 341.9 seconds; 30 minutes: 385.9 ± 330.7 seconds; 120 minutes: 120.7 ± 176.2 seconds; $p < 0.0342$) that were not present in the control session (15 minutes: 190.3 ± 266.1 seconds; 30 minutes: 143.1 ± 240.5; 120 minutes: 124.1 ± 159.6 seconds). Also in the fatigue session, Female PDs had more gaps in L-EXO activity than Female NPDs during the 15 minute time block (Female PD Fatigue: 179.0 ± 89.1 gaps; Female NPD Fatigue: 26.9 ± 39.5 gaps; $p = 0.0253$). Across sessions, Female PDs had fewer gaps in R-TFL than Female NPDs from the 45 to 75 minute time blocks (at 45 minutes, PD: 16.3 ± 16.2 gaps; NPD: 47.0 ± 44.5 gaps; $p = 0.0114$; at 60 minutes, PD: 20.6 ± 16.8 gaps; NPD: 52.3 ± 60.4 gaps; $p = 0.0156$; at 75 minutes, PD: 22.0 ± 18.0 gaps; NPD: 47.8 ± 41.2 gaps; $p = 0.0277$). Female PDs also had an increase in the number of L-TFL gaps in the 90 minute block compared to earlier time points across sessions (15 minutes: $p = 0.0403$, 30 minutes: $p = 0.0396$, 45 minutes: $p = 0.006$, 60 minutes: $p = 0.0603$, 75 minutes: $p = 0.0032$; Figure 4.11). The number of L-TFL gaps decreased back to initial levels in the 120 minute block ($p = 0.6421$). It should be noted that Female PDs had the largest proportion of left leg dominance (25% vs 8.3% in Male NPDs and 0% in both Male PDs and Female NPDs). Female NPDs had an increase in L-GMD cumulative times from the 15 minute to the 120 minute block (15 minutes: 198.4 ± 284.5 seconds; 120 minutes: 422.6 ± 293.5 seconds; $p = 0.0176$) and from the 30 minute to 120 minute

block (30 minutes: 224.4 ± 299.4 seconds; p = 0.0260); both of which were absent in Female PDs (15 minutes: 392.7 ± 342.1 seconds; 30 minutes: 367.5 ± 358.9 seconds; 120 minutes: 322.0 ± 348.4 seconds). Females in both pain groups had decreases from the 30 to 120 minute time blocks (30 minutes: 496.2 ± 326.6 seconds; 120 minutes: 328.1 ± 265.8 seconds; p = 0.0082) and from the 60 to 120 minute time blocks (60 minutes: 468.0 ± 303.0; p = 0.0451) in R-TES cumulative time during the fatigue session that was absent in the control session. Similarly in the control session, females from both pain groups had longer R-INO gap lengths in the 120 minute block than the 15 minute block (15 minutes: 7.22 ± 8.40 seconds; 120 minutes: 14.31 ± 26.02 seconds; p = 0.0154). A summary of findings from gaps computations in females is presented in Figure 4.12.

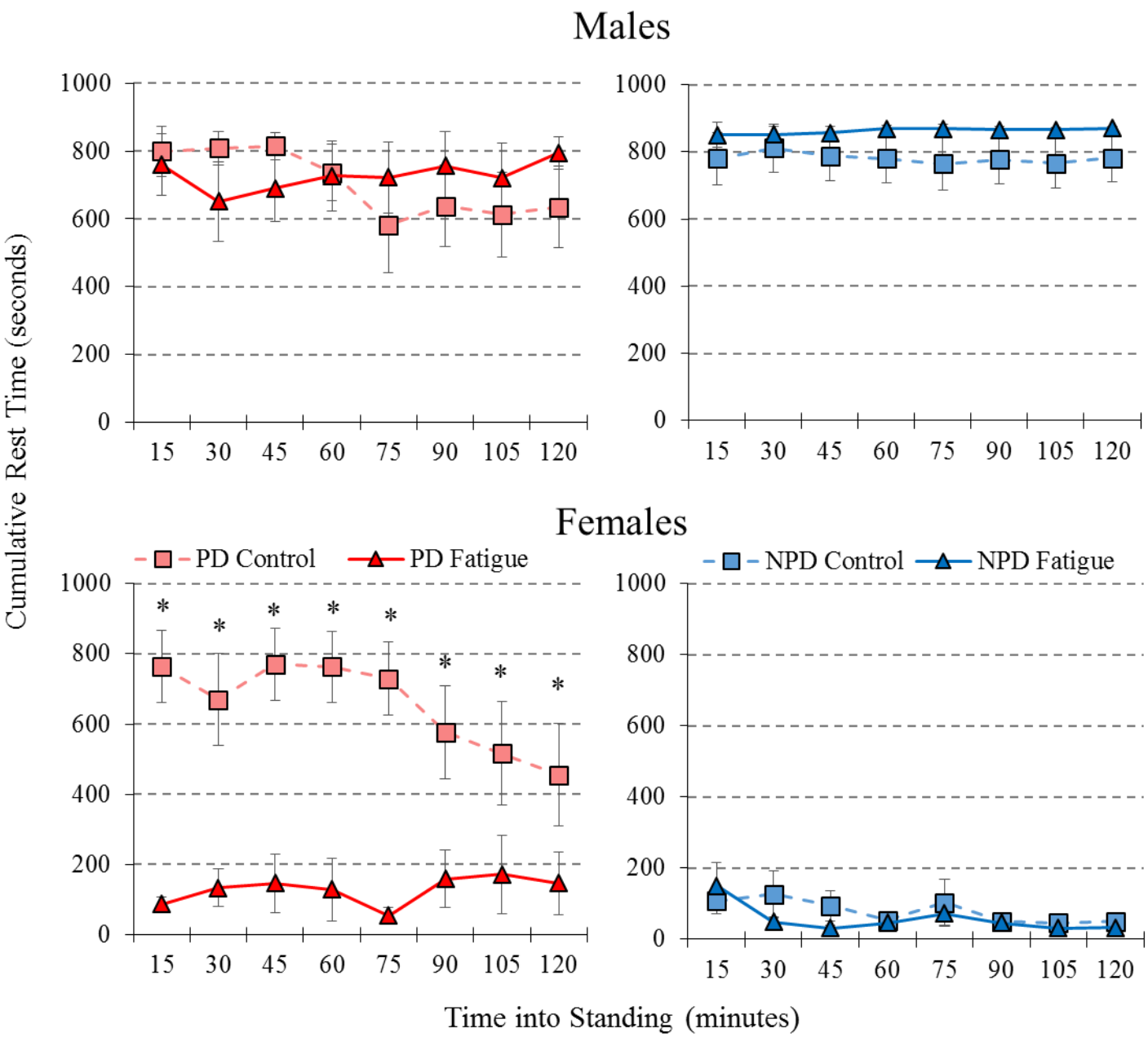

Figure 4.10: Gender Differences in R-TFL Cumulative Rest Time. The measure cumulative time refers the total length of time within a 15 minute window when R-TFL activity was below 0.5 %MVIC. In males (top panels), R-TFL was inactive most of the time, and consistently so between *PAIN* and *SESSION* groups. In females (bottom panels), PDs did not utilize their R-TFLs during the control session while NPDs did, however during the fatigue session, female PDs responded more like female NPDs, showing a larger decrease with fatigue. Significant effects of *SESSION* ($p < 0.05$) within female PDs are indicated with asterisks.

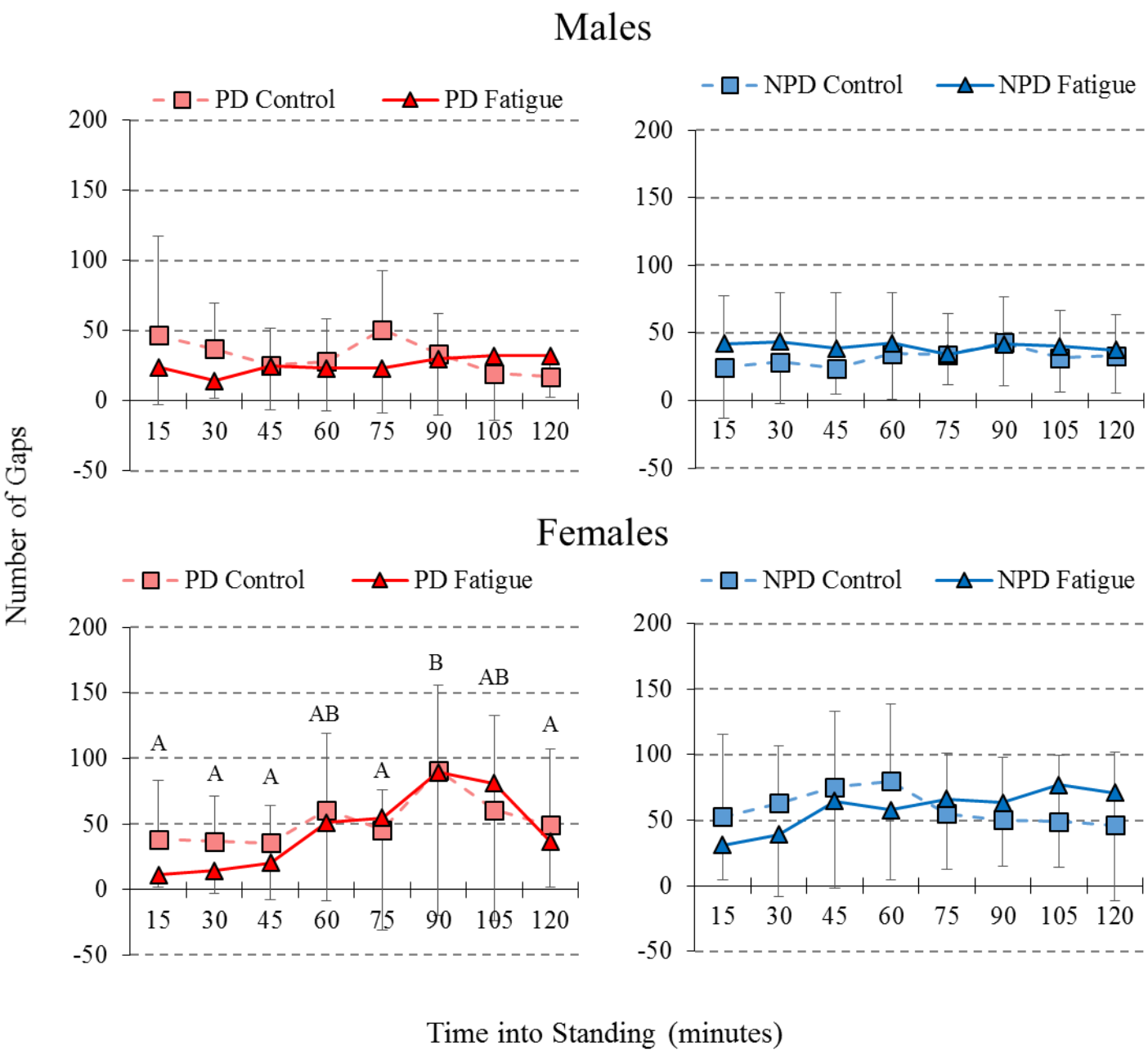

Figure 4.11: Gender Differences in the Number of L-TFL Gaps during Standing. As with R-TFL Cumulative Times (Figure 4.11), males responded similarly across *PAIN* and *SESSION* groups, whereas females had different time-varying responses. Letters indicate a *TIME*GENDER*PAIN* interaction ($p < 0.05$) where Female PDs had more L-TFL gaps in the 90 minute block than in most of the first 75 minutes or the last 15 minutes. The 105 minute block was not different from any other time point.

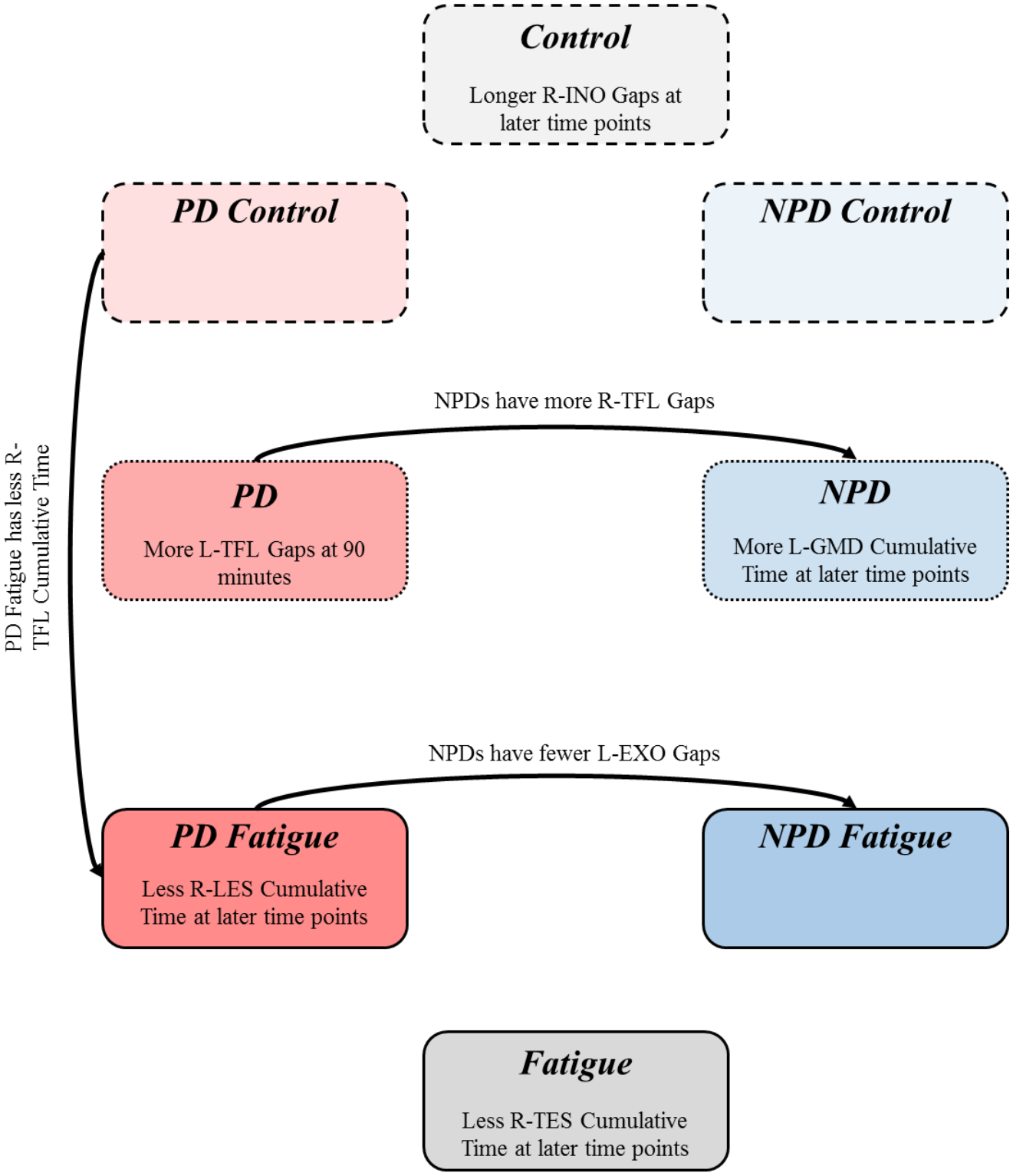

Figure 4.12: Changes in Gaps Variables for Females. *TIME* effects are placed within the appropriate box, other effects are indicated by arrows. See text and Figures 4.11 to 4.12 for specific values and probabilities.

There were a number of time effects specific to PDs. PDs had more R-INO gaps during the first hour and 15 minutes than in the last 15 minutes of the control session, reaching significance in the 45 to 75 minute blocks ($p < 0.0460$). This change over time was not present in the fatigue session (Figure 4.13). Across sessions, PDs had decreases over time in the number of gaps in R-EXO from the 30 to 120 minute blocks (30 minutes: 94.2 ± 91.4 gaps; 120 minutes: 65.2 ± 69.5 gaps; $p = 0.0403$), and from the 60 to 120 minute blocks (60 minutes: 86.2 ± 74.1 gaps; $p = 0.0272$). Also across sessions, the cumulative time that R-INO spent below 0.5 %MVIC decreased from the first hour and fifteen minutes to the last 45 minutes (Table 4.8).

Table 4.8: Changes in R-INO Cumulative Time in PDs over the Standing Protocol.
Italicized values in the Table are of PDs averaged over *GENDER* and *SESSION*. Probabilities for each pair of time points are placed at the intersection of that pair.

PDs	**Later Time Points**	**90 Minutes**	**105 Minutes**	**120 Minutes**
Earlier Time Points	***R-INO Cumulative Time (seconds)***	*295.0 (353.2)*	*327.5 (370.5)*	*305.4 (375.0)*
15 Minutes	*375.3 (347.1)*	***P = 0.0042***	***P = 0.0118***	***P = 0.0092***
30 Minutes	*411.4 (351.2)*	***P = 0.0092***	***P = 0.0173***	***P = 0.0130***
45 Minutes	*415.3 (341.0)*	***P = 0.0062***	***P = 0.0206***	***P = 0.0133***
60 Minutes	*434.0 (339.9)*	***P = 0.0003***	***P = 0.0071***	***P = 0.0045***
75 Minutes	*381.2 (371.8)*	***P = 0.0107***	P = 0.3266	P = 0.2352

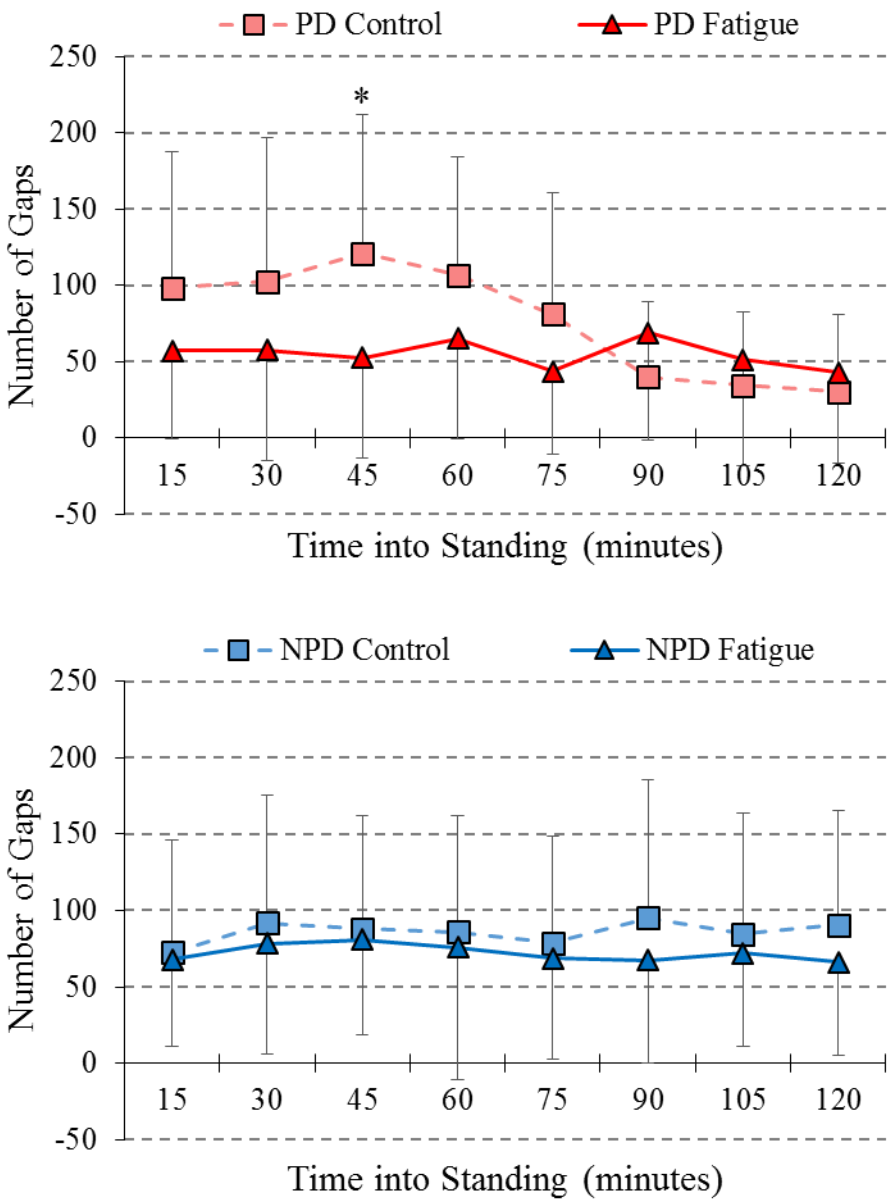

Figure 4.13: Differences in R-INO Gaps between Pain Groups Over Time. There was a *TIME*PAIN*SESSION* interaction where in PDs, the number of gaps in the control session decreased over time while they remained constant during the fatigue session. In this respect, the fatigue intervention caused PDs to behave more like NPDs while standing. Interestingly enough, the number of gaps in the 15 and 30 minute blocks of the PD control trial were not different from any other time point (15 minute: $p = 0.0639$; 30 minute: $p = 0.0652$) despite appearing graphically similar to the 60 minute block (60 minute: $p = 0.0460$).

Aside from the *GENDER* and *TIME* interactions, NPDs had more gaps than PDs in three of twelve muscles while standing. There were main effects of *PAIN* in R-LES during the 120 minute block (PD: 59.1 ± 60.8 gaps; NPD: 101.7 ± 77.5 gaps; p = 0.0139), in L-GMD during the 75 minute block (PD: 52.3 ± 51.2 gaps; NPD: 75.3 ± 71.5 gaps; p = 0.0380), and in L-TFL in the 30 minute (PD: 25.6 ± 28.5 gaps; NPD: 43.4 ± 41.8 gaps; p = 0.0439), and 45 minute blocks (PD: 26.5 ± 29.1 gaps; NPD: 50.5 ± 53.6 gaps; p = 0.0163).

There were also main effects of *SESSION* in the cumulative times of the thoracic level erector spinae. R-TES had longer cumulative times in the fatigue session (at 30 minutes, Control: 180.2 ± 247.5 seconds; Fatigue: 346.2 ± 320.5 seconds; p = 0.0165), however L-TES had longer cumulative times in the control session (at 120 minutes, Control: 441.5 ± 317.7 seconds; Fatigue: 282.9 ± 278.5 seconds; p = 0.0239).

4.3.2 Kinematic Measures during Standing

There were *GENDER*PAIN*SESSION* interactions in mean pelvic tilt angles in the sagittal plane such that the fatigue intervention elicited different responses in male and female PDs when compared both across gender and between pain groups (Figure 4.14). In males, PDs were more anteriorly tilted during the fatigue session throughout the 2 hours of standing, reaching significance in the 45 minute (control: 6.7 ± 7.7°; fatigue: -0.3 ± 5.1°; p = 0.0460), 75 minute (control: 7.8 ± 6.8°; fatigue: 0.5 ± 4.5°; p = 0.0401), 90 minute (control: 8.3 ± 6.9°; fatigue: 0.5 ± 5.1°; p = 0.0332), 105 minute (control: 8.6 ± 7.4°; fatigue: 0.9 ± 4.7°; p = 0.0285), and 120 minute block (control: 8.5 ± 7.9°; fatigue: 0.8 ± 5.0°; p = 0.0240). Male NPDs moved in the same direction with fatigue, however were less perturbed by the fatigue protocol and the two sessions were not significantly different from each other (mean difference ~ 3°; p > 0.2734). In females, PDs and NPDs shifted

in opposite directions, with female PDs standing in more posterior tilt than NPDs from the 30 to 90 minute blocks (30 minutes, Female PD: 8.9 ± 11.3°; Female NPD: -0.2 ± 6.9°; p = 0.0062, 45 minutes, Female PD: 8.8 ± 12.1°; Female NPD: 1.2 ± 5.1°; p = 0.0178, 60 minutes, Female PD: 9.5 ± 12.9°; Female NPD: 2.5 ± 5.7°; p = 0.0365, 75 minutes, Female PD: 10.5 ± 12.4°; Female NPD: 1.0 ± 5.4°; p = 0.0042, 90 minutes, Female PD: 9.7 ± 12.4°; Female NPD: 2.4 ± 6.4°; p = 0.0302). After 90 minutes, the two pain groups were no longer different from each other (p > 0.4655).

Similar to pelvic tilt, there were *GENDER*PAIN*SESSION* interactions in mean relative angles for the segments adjacent to the pelvis. Male PDs were standing in more lumbar extension and hip flexion during the fatigue session. In contrast, Female PDs were standing in more hip extension during the fatigue session. NPDs of both genders had similar lumbar spine and hip postures in the sagittal plane between sessions, but in both cases tended to shift in the direction of Male PDs rather than female PDs. Although female PDs were found to have similar lumbar postures between sessions, female PDs were more flexed than female NPDs in the fatigue sessions within the 30 minute (p = 0.0134), 45 minute (p = 0.0263), 60 minute (p = 0.0397), 75 minute (p = 0.0191) and 90 minute blocks (p = 0.0211; see Tables 4.9 and 4.10). Specific angles with probabilities of between session comparisons are presented in Tables 4.9 and 4.10, and contrasting changes with fatigue between male and female PDs are shown in Figure 4.15.

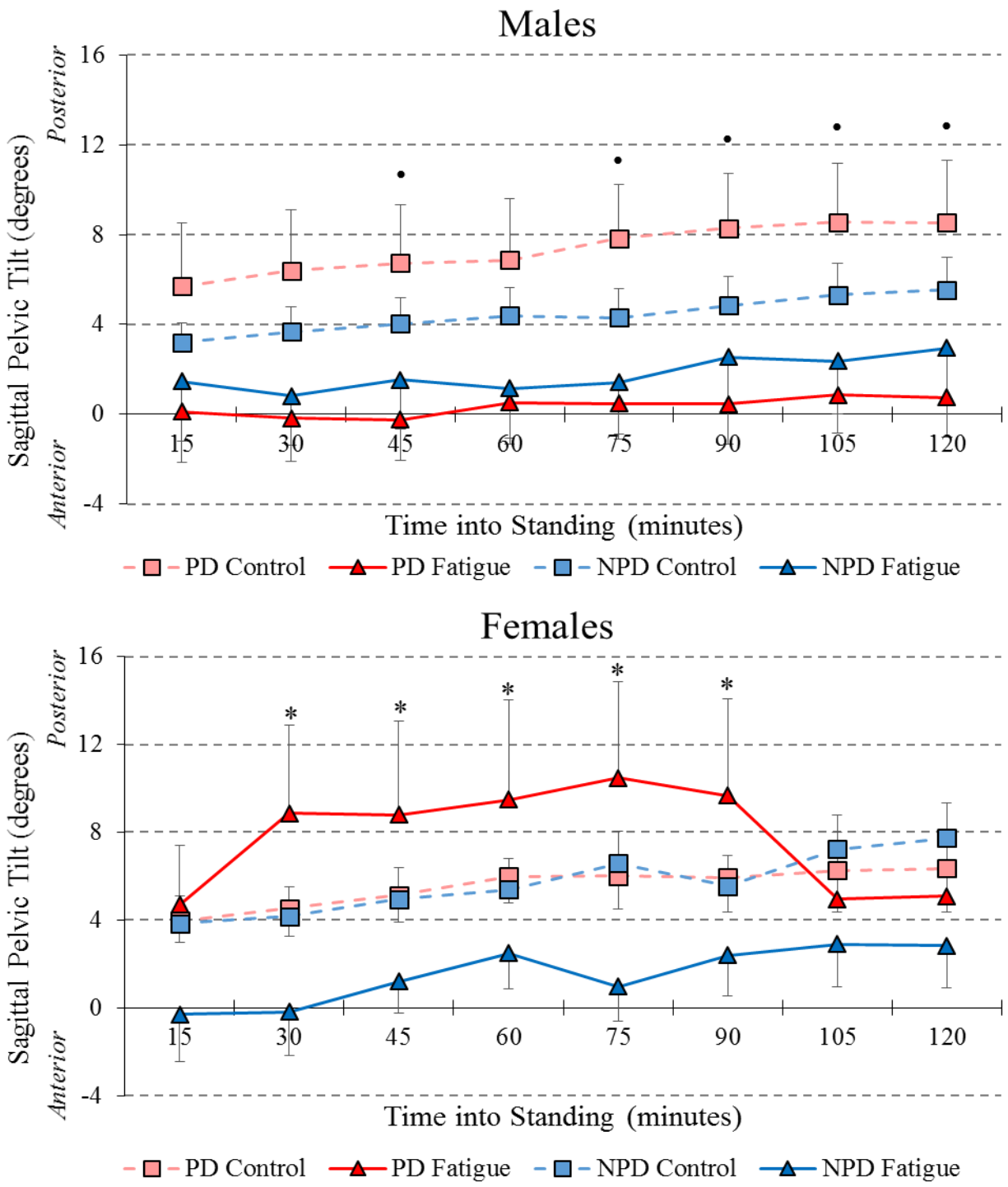

Figure 4.14: Changes in Mean Anterior/Posterior Pelvic Tilt Angles with Fatigue. In males (top panel) both pain groups moved into more anterior tilt with fatigue, PDs more so than NPDs. Significance between control and fatigue sessions in male PDs (red lines) are indicated with solid circles ($p < 0.05$). In females (bottom panel), PDs tilted more posteriorly with fatigue while NPDs tilted more anteriorly with fatigue. Significant differences between female PDs and NPDs within the fatigue sessions (solid lines) are indicated by asterisks ($p < 0.05$). Error bars show standard errors.

Table 4.9: Sagittal Lumbar and Hip Angles in PDs. Lumbar extension and hip flexion are positive. Differences between sessions are bolded.

Male PDs		**15 min**	**30 min**	**45 min**	**60 min**	**75 min**	**90 min**	**105 min**	**120 min**
L/P Angle (°)	Control	-2.3 (10.7)	-3.4 (10.9)	-4.5 (11.2)	-5.3 (11.2)	-6.2 (10.6)	-7.4 (10.6)	-7.6 (10.6)	-8.5 (11.3)
	Fatigue	4.9 (4.3)	4.9 (5.0)	4.0 (4.6)	3.5 (4.5)	3.2 (4.0)	2.7 (4.1)	2.7 (4.0)	2.3 (4.1)
	Probability	0.1628	0.0783	0.0789	0.0818	0.0589	***0.0391***	***0.0258***	***0.0227***
Right Hip Angle (°)	Control	-9.8 (9.3)	-10.8 (9.1)	-10.0 (9.0)	-10.3 (9.3)	-11.4 (8.3)	-11.2 (8.8)	-11.8 (8.7)	-11.5 (8.8)
	Fatigue	-4.7 (4.0)	-4.1 (5.4)	-3.1 (4.9)	-3.4 (5.7)	-3.8 (4.9)	-2.9 (5.6)	-3.0 (5.0)	-3.1 (4.9)
	Probability	0.1994	0.0915	0.0762	0.0771	***0.0428***	***0.0364***	***0.0292***	***0.0325***
Left Hip Angle (°)	Control	-10.6 (7.8)	-11.1 (7.3)	-10.7 (6.7)	-10.5 (7.2)	-12.7 (6.2)	-12.3 (6.9)	-12.9 (7.0)	-13.1 (7.4)
	Fatigue	-4.9 (4.6)	-3.7 (6.8)	-2.6 (6.1)	-4.0 (6.3)	-2.5 (5.7)	-2.8 (5.9)	-3.4 (5.8)	-3.0 (5.8)
	Probability	0.1252	***0.0438***	***0.0307***	0.0828	***0.0053***	***0.0138***	***0.0155***	***0.0076***
Female PDs		**15 min**	**30 min**	**45 min**	**60 min**	**75 min**	**90 min**	**105 min**	**120 min**
L/P Angle (°)	Control	3.0 (4.8)	1.9 (5.4)	1.7 (5.7)	-0.2 (5.5)	-0.2 (6.9)	-1.1 (7.7)	-0.3 (8.5)	-0.7 (8.9)
	Fatigue	0.3 (8.2)	-4.0 (12.1)	-5.0 (12.8)	-5.9 (12.6)	-7.2 (11.5)	-7.8 (11.2)	-3.6 (6.3)	-3.8 (4.3)
	Probability	0.5429	0.1780	0.1469	0.2447	0.1466	0.1533	0.4879	0.5235
Right Hip Angle (°)	Control	-4.4 (4.3)	-3.6 (5.1)	-2.9 (6.0)	-2.4 (6.9)	-2.9 (7.2)	-2.7 (6.6)	-2.2 (7.5)	-2.9 (7.7)
	Fatigue	-6.9 (4.1)	-8.2 (7.6)	-10.0 (7.4)	-10.0 (8.4)	-10.8 (7.5)	-9.4 (7.8)	-4.7 (8.4)	-4.2 (6.8)
	Probability	0.5284	0.2422	0.0670	0.0492	***0.0389***	0.0850	0.5392	0.7431
Left Hip Angle (°)	Control	-4.9 (5.5)	-3.9 (6.8)	-4.2 (6.6)	-4.0 (6.8)	-4.1 (7.9)	-3.9 (7.6)	-4.1 (8.1)	-3.9 (8.1)
	Fatigue	-8.4 (4.1)	-12.0 (7.3)	-11.6 (7.9)	-11.5 (8.5)	-12.2 (7.8)	-10.9 (8.1)	-5.8 (9.0)	-5.8 (6.9)
	Probability	0.3497	***0.0264***	***0.0450***	***0.0451***	***0.0258***	0.0671	0.6794	0.6190

Table 4.10: Sagittal Lumbar and Hip Angles in NPDs. Lumbar extension and hip flexion are positive. There were no significant differences found due to fatigue.

Male NPDs		**15 min**	**30 min**	**45 min**	**60 min**	**75 min**	**90 min**	**105 min**	**120 min**
L/P Angle (°)	Control	1.1 (5.1)	0.3 (5.3)	-0.5 (5.7)	-0.9 (6.2)	-0.9 (5.9)	-1.3 (6.1)	-1.8 (6.4)	-2.2 (6.9)
	Fatigue	3.2 (16.0)	3.7 (10.3)	3.0 (10.1)	3.3 (10.5)	2.4 (11.5)	2.1 (11.1)	1.1 (11.3)	0.2 (11.5)
	Probability	0.6156	0.3842	0.3820	0.3077	0.4181	0.3923	0.4420	0.5431
Right Hip Angle (°)	Control	-6.5 (4.5)	-6.1 (5.2)	-5.3 (6.8)	-5.9 (6.5)	-6.2 (6.3)	-6.3 (6.8)	-7.3 (6.3)	-7.5 (6.5)
	Fatigue	-5.6 (11.7)	-3.4 (7.7)	-3.8 (7.8)	-3.2 (6.2)	-3.3 (6.4)	-5.3 (6.1)	-4.7 (6.6)	-4.9 (6.8)
	Probability	0.7816	0.3949	0.6244	0.3912	0.3292	0.7606	0.4085	0.4117
Left Hip Angle (°)	Control	-7.9 (4.8)	-7.7 (5.7)	-7.9 (6.1)	-8.3 (6.4)	-8.2 (6.0)	-8.7 (6.1)	-9.1 (6.3)	-9.2 (6.2)
	Fatigue	-7.0 (11.9)	-5.6 (8.1)	-6.1 (8.6)	-5.6 (7.7)	-5.5 (7.8)	-7.0 (8.2)	-6.2 (8.1)	-7.3 (7.6)
	Probability	0.7514	0.4554	0.5356	0.3898	0.3518	0.5675	0.6794	0.5300
Female NPDs		**15 min**	**30 min**	**45 min**	**60 min**	**75 min**	**90 min**	**105 min**	**120 min**
L/P Angle (°)	Control	0.4 (9.9)	-0.5 (10.1)	-2.1 (11.0)	-3.1 (12.3)	-3.7 (11.7)	-3.0 (10.5)	-3.4 (9.8)	-3.8 (10.0)
	Fatigue	7.2 (9.3)	6.7 (8.0)	4.9 (7.5)	3.6 (7.8)	3.5 (7.7)	2.6 (8.2)	1.2 (7.8)	1.0 (8.5)
	Probability	0.1083	0.0621	0.0793	0.1031	0.0787	0.1637	0.2133	0.2089
Right Hip Angle (°)	Control	-5.7 (6.2)	-5.5 (7.8)	-5.9 (8.1)	-5.9 (7.6)	-6.9 (7.9)	-7.0 (8.6)	-7.4 (8.4)	-7.7 (8.5)
	Fatigue	-3.1 (9.2)	-2.7 (9.5)	-3.5 (6.8)	-3.8 (7.5)	-2.9 (6.6)	-4.1 (7.6)	-4.5 (7.9)	-4.9 (7.6)
	Probability	0.4126	0.3751	0.4434	0.4951	0.1850	0.3478	0.3639	0.366
Left Hip Angle (°)	Control	-7.8 (7.2)	-6.8 (7.1)	-6.9 (7.6)	-7.8 (7.7)	-8.3 (6.9)	-7.4 (7.3)	-8.0 (7.0)	-9.3 (7.5)
	Fatigue	-7.3 (4.6)	-6.4 (4.8)	-6.7 (4.9)	-7.9 (6.2)	-7.3 (5.0)	-7.8 (6.5)	-8.0 (6.7)	-8.2 (6.4)
	Probability	0.8578	0.8909	0.9325	0.9695	0.7256	0.9150	0.9874	0.7214

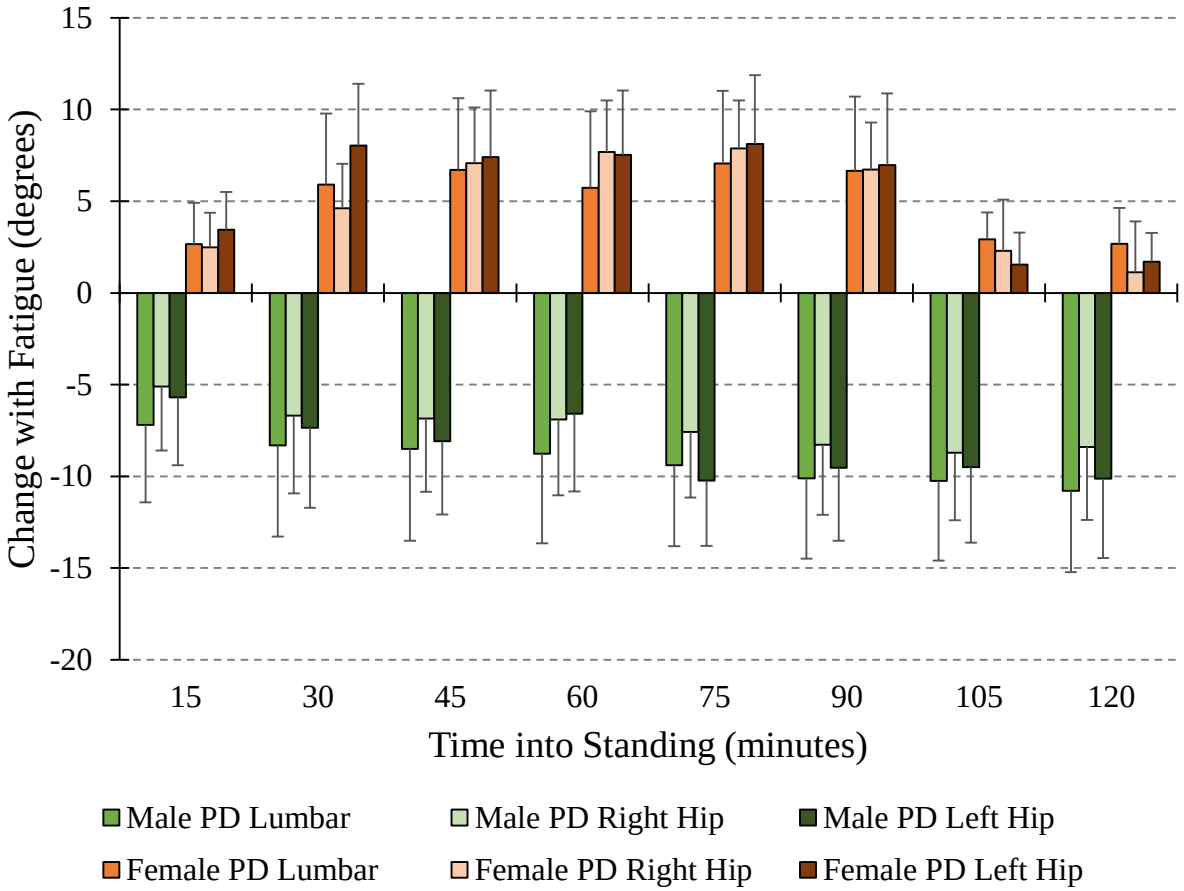

Figure 4.15: Changes in Lumbo-pelvic and Hip Angles with Fatigue in PDs. Males are in green and females are in orange. Positive values on this figure indicate a larger angle on the fatigue session – a shift into lumbar flexion or hip extension in the fatigue session compared to the control session. NPDs of both genders (not pictured) tended to follow the green bars (males), albeit with smaller magnitudes and larger variability.

In addition to the sagittal plane interactions, there were *GENDER*PAIN*SESSION* interactions in pelvic lateral tilt angles but with a different pattern (Figure 4.16). Male PDs and female NPDs had neutral pelvic postures that were similar to each other and similar between sessions (~ 0° of lateral pelvic tilt). Female PDs and male NPDs had similarly neutral pelvic postures during the control session, however were tilted to the right in the fatigue session (~ 3°). This pattern was seen more strongly in female PDs than male NPDs. Removing all left leg-

dominant persons strengthened this relationship in female PDs and had minimal changes in male NPDs. More specifically, during all time blocks, female PDs in the fatigue session were more rightward tilted than female PDs in the control session (from a mean difference of 3.5° and $p = 0.0482$ in 105 minute block, to a mean difference of 5.2° and $p = 0.0018$ in the 30 minute block) and female NPDs during the fatigue session (from a mean difference of 3.4° and $p = 0.0194$ n the 60 minute block, to a mean difference of 5.2° and $p = 0.0004$ in the 30 minute block). Male NPDs during the fatigue session were more rightward tilted than male PDs during the fatigue session from the 30 minute block to the 120 minute block (from a mean difference of 3.2° and $p = 0.0488$ in the 105 minute block, to a mean difference of 4.0° and $p = 0.0099$ in the 75 minute block) and male NPDs during the control session in the 120 minute block (Control: 0.6 ± 2.3°; Fatigue: 3.3 ± 4.9°; $p = 0.0486$).

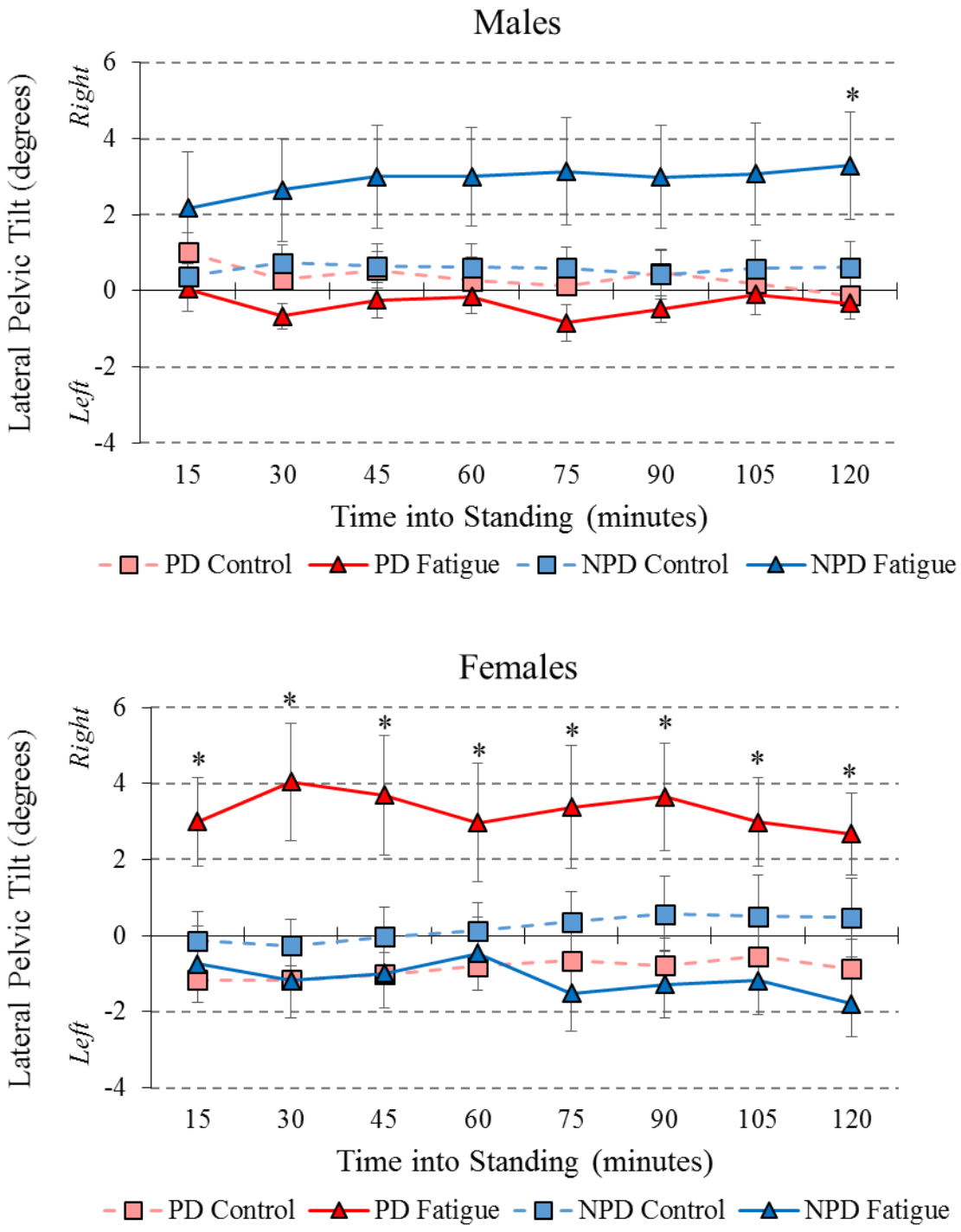

Figure 4.16: Changes in Mean Lateral Pelvic Tilt Angles with Fatigue. In males (upper panel), PDs were unaffected by fatigue while NPDs tilted their pelvises to the right (positive). In females (lower panel) PDs experienced a rightward tilt while NPDs were less affected (appears to be tilted slightly leftward, $p > 0.0942$). Differences between sessions are indicated with asterisks for male NPDs and female PDs. Error bars show standard errors.

Similar to the sagittal plane, these differences in lateral pelvic tilt translated into *GENDER*PAIN*SESSION* interactions at the hip (Figure 4.17). In males, NPDs stood in greater right hip abduction than PDs across sessions, reaching significance in the 30 minute (PD: -1.2 ± 3.3°; NPD: -5.2 ± 5.1°; p = 0.0189), 45 minute (PD: -1.6 ± 3.5°; NPD: -5.2 ± 6.2°; p = 0.0238), 60 minute (PD: -0.9 ± 2.8°; NPD: -5.2 ± 5.6°; p = 0.0050), 75 minute (PD: -0.6 ± 2.4°; NPD: -4.5 ± 5.6°; p = 0.0151) and 120 minute blocks (PD:-1.3 ± 3.0°; NPD: -4.4 ± 5.2°; p = 0.0481). In females, PDs stood in greater abduction than NPDs at the right hip and greater adduction at the left hip for most time blocks. In the right hip, pain differences in females occurred in the 30 minute (PD: -4.7 ± 3.7°; NPD: -1.0 ± 4.6°; p = 0.0029), 45 minute (PD: -4.8 ± 4.9°; NPD: -0.6 ± 3.8°; p = 0.0091), 75 minute (PD: -5.4 ± 5.2°; NPD: -0.9 ± 4.4°; p = 0.0105), 90 minute (PD: -5.7 ± 5.0°; NPD: -0.5 ± 4.2°; p = 0.0007), 105 minute (PD: -6.2 ± 5.3°; NPD: -0.3 ± 4.5°; p = 0.0008), and 120 minute blocks (PD: -5.3 ± 4.7°; NPD: -0.2 ± 4.7°; p = 0.0018). In the left hip, *PAIN* differences occurred in all blocks except the 60 minute block (from a mean difference of 3.2° and p = 0.0451 in the 15 minute block, to a mean difference of 4.4° and p = 0.0041 in the 90 minute block). Although no *GENDER*PAIN*SESSION* interactions were found by the initial general linear model (for the right hip: p > 0.1193), females appeared to have greater differences in right hip frontal plane angles between pain groups in the fatigue session than in the control session (Table 4.11).

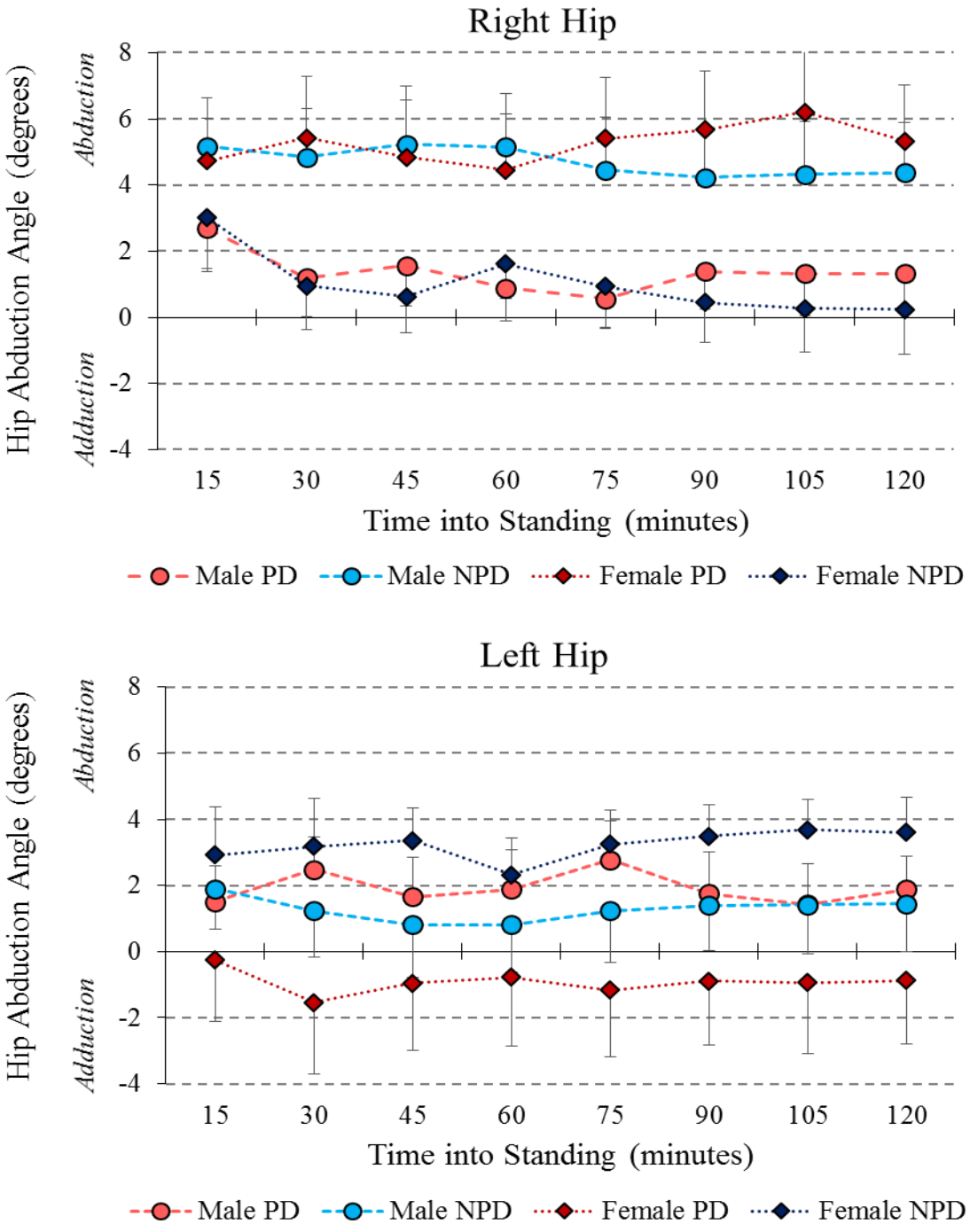

Figure 4.17: Differences in Right and Left Hip Abduction Angles between Pain Groups and Genders. Right hip abduction was computed as being negative based on ISB axis conventions and reported as such in-text and in tables, however it was inverted in this figure so that abduction was positive for both legs. In the right hip (top panel), Male PDs and Female NPDs remained fairly neutral while Male NPDs and Female PDs were more abducted. In the left hip (bottom panel), there were minimal differences between male PDs and NPD while females retained pain group differences (PDs less abducted) that match the differences in lateral pelvic tilt. Error bars show standard errors.

Table 4.11: Session Differences in Right Hip Abduction Angles in Female PDs and NPDs. Abduction is negative and adduction is positive. Significant effects of *PAIN* ($p < 0.05$) are bolded. Although statistical data did not indicate a *GENDER*PAIN*SESSION* interaction, pain differences in right hip abduction angles in females appeared to be accentuated in the fatigue session.

Time into Standing (minutes)		***15***	***30***	***45***	***60***	***75***	***90***	***105***	***120***
Both Sessions	*Female PD*	-4.7 (3.7)	-5.4 (5.3)	-4.8 (4.9)	-4.5 (4.8)	-5.4 (5.2)	-5.7 (5.0)	-6.2 (5.3)	-5.3 (4.7)
	Female NPD	-3.0 (5.3)	-1.0 (4.6)	-0.6 (3.8)	-1.6 (3.6)	-0.9 (4.4)	-0.5 (4.2)	-0.3 (4.5)	-0.2 (4.7)
	Probability	0.2693	***0.0029***	***0.0091***	0.0683	***0.0105***	***0.0007***	***0.0008***	***0.0018***
Control	*Female PD*	-3.4 (3.2)	-2.6 (3.0)	-2.9 (2.5)	-3.4 (3.5)	-4.1 (3.8)	-3.5 (4.4)	-5.0 (6.0)	-3.96 (5.26)
	Female NPD	-3.7 (3.7)	-1.2 (3.3)	-0.8 (3.2)	-1.8 (3.0)	-1.7 (3.6)	-0.7 (3.8)	-0.4 (4.7)	-1.1 (4.7)
Fatigue	*Female PD*	-6.0 (3.7)	-8.3 (5.5)	-6.8 (5.9)	-5.5 (5.5)	-6.8 (6.0)	-7.9 (4.6)	-7.3 (4.3)	-6.5 (3.9)
	Female NPD	-2.3 (6.4)	-0.7 (5.7)	-0.4 (4.3)	-1.4 (4.1)	-0.2 (5.0)	-0.2 (4.5)	-0.2 (4.3)	0.6 (4.5)

There were effects of *TIME* on lumbar and pelvic angles where participants in all conditions and groups tended towards more spine flexion and more posterior pelvic tilt in later time blocks. Changes in lumbar spine flexion were steady over the 2 hours with each incremental time block being different than the previous with the exception of moving from the 90 to 105 minute block (Table 4.12). While both PDs and NPDs had more posteriorly tilted pelvic postures over time, the timing of those changes differed between pain groups (Figure 4.18). In PDs, posterior tilting occurred mostly in the first hour, with the 75 minute block being the most posteriorly tilted block (at 15 minutes, PD: 3.6 ± 6.4°; at 75 minutes, PD: 6.2 ± 8.5°; $p = 0.0009$;

Table 4.13). In NPDs, there was a more gradual increase in posterior pelvic tilt, continuing up to the 120 minute block (at 15 minutes, NPD: 2.1 ± 8.0°; at 120 minutes, NPD: 4.8 ± 6.7°; p = 0.0004; Table 4.14).

Table 4.12: Changes in Average Lumbar Spine Angles over the Standing Protocol. Each time block is within the grey shaded area along the top and left hand side of the table, the mean angle (and standard deviation) associated with each time block is to the right or below each time block. Numbers at the intersection of each time point pairing are probabilities in comparing that pair. Flexion is negative and extension is positive.

All Participants	**Later Time Points (minutes)**	**30**	**45**	**60**	**75**	**90**	**105**	**120**
Earlier Time Points	***Lumbar Spine Angle (degrees)***	*1.5 (9.5)*	*0.4 (9.6)*	*-0.4 (10.0)*	*-0.8 (10.0)*	*-1.3 (9.8)*	*-1.3 (9.0)*	*-1.8 (9.3)*
15 Minutes	*2.4 (10.0)*	***0.044***	***0.0006***	***0.0001***	***0.0001***	***0.0001***	***0.0001***	***0.0001***
30 Minutes	*1.5 (9.5)*		***0.0001***	***0.0001***	***0.0001***	***0.0001***	***0.0002***	***0.0001***
45 Minutes	*0.4 (9.6)*			***0.0001***	***0.0001***	***0.0001***	***0.0156***	***0.0016***
60 Minutes	*-0.4 (10.0)*				***0.0107***	***0.0004***	0.2116	***0.0482***
75 Minutes	*-0.8 (10.0)*					***0.0088***	0.5501	0.1621
90 Minutes	*-1.3 (9.8)*						0.8334	0.5003
105 Minutes	*-1.3 (9.0)*							***0.0135***

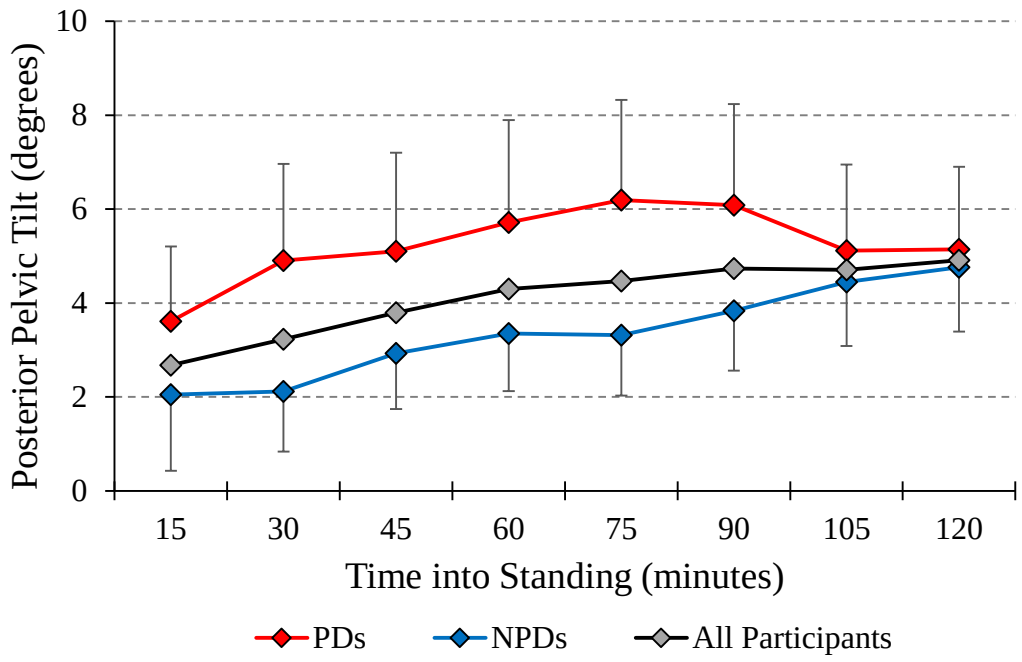

Figure 4.18: Changes in Posterior Pelvic Tilt over Time. While both pain groups tended to become more posteriorly tilted with time, PDs tended to increase earlier than NPDs. Posterior tilt is positive on this figure, error bars show standard errors.

Table 4.13: Changes in Posterior Pelvic Tilt in PDs over Time. Probabilities from post-hoc testing are at the intersection of time point pairs. Posterior pelvic tilt is positive.

PDs	**Later Time Points (minutes)**	**30**	**45**	**60**	**75**	**90**	**105**	**120**
Earlier Time Points	***Posterior Pelvic Tilt (degrees)***	*4.9 (8.3)*	*5.1 (8.4)*	*5.7 (8.7)*	*6.2 (8.5)*	*6.1 (8.6)*	*5.1 (7.3)*	*5.1 (7.0)*
15 Minutes	*3.6 (6.4)*	***0.037***	***0.0342***	***0.005***	***0.0009***	***0.0019***	0.1519	0.0884
30 Minutes	*4.9 (8.3)*		0.357	***0.0032***	***0.0001***	***0.0003***	0.8892	0.8560
45 Minutes	*5.1 (8.4)*			***0.0004***	***0.0001***	***0.0019***	0.9982	0.9811
60 Minutes	*5.7 (8.7)*				0.0753	0.2391	0.6488	0.6414
75 Minutes	*6.2 (8.5)*					0.5787	0.3928	0.3674
90 Minutes	*6.1 (8.6)*						0.4149	0.3887
105 Minutes	*5.1 (7.3)*							0.9077

Table 4.14: Changes in Posterior Pelvic Tilt in NPDs over Time. Probabilities from post-hoc testing are at the intersection of time point pairs. Posterior pelvic tilt is positive.

NPDs	**Later Time Points (minutes)**	**30**	**45**	**60**	**75**	**90**	**105**	**120**
Earlier Time Points	***Posterior Pelvic Tilt (degrees)***	*2.1 (6.3)*	*2.9 (5.8)*	*3.4 (6.0)*	*3.3 (6.3)*	*3.8 (6.2)*	*4.5 (6.7)*	*4.8 (6.7)*
15 Minutes	*2.1 (8.0)*	0.9249	0.2788	0.1059	0.1000	***0.0247***	***0.0017***	***0.0004***
30 Minutes	*2.1 (6.3)*		***0.0035***	***0.0038***	***0.004***	***0.0006***	***0.0001***	***0.0001***
45 Minutes	*2.9 (5.8)*			0.1100	0.2335	***0.0206***	***0.0005***	***0.0001***
60 Minutes	*3.4 (6.0)*				0.8697	0.0824	***0.0002***	***0.0001***
75 Minutes	*3.3 (6.3)*					***0.0186***	***0.0001***	***0.0001***
90 Minutes	*3.8 (6.2)*						***0.001***	***0.0004***
105 Minutes	*4.5 (6.7)*							0.0729

The left hip angle in became more externally rotated over time in all participants. The left hip angle was more internally rotated in the 15 minute block (4.0 ± 5.8°) than in the last hour (75 minutes: 5.5 ± 6.5°, p = 0.0002; 90 minutes: 5.4 ± 6.2°, p = 0.0008; 105 minutes: 5.3 ± 6.3°, p = 0.0014; 120 minutes: 5.2 ± 6.4°, p = 0.0015). There were also differences between the 30 minute and 75 minute blocks (30 minutes: 4.9 ± 6.4°; p = 0.0389). This pattern of increasing hip external rotation with time was not present in the right hip (p = 0.6275).

4.3.3 Force Plate Measures during Standing

Mean center of pressure values were more lateral in the fatigue session for pain developers (*PAIN*SESSION;* Figure 4.19). Positive numbers are to the right of the calcaneus, (more lateral for the right foot, more medial for the left foot). In the left foot, the two sessions were different from each other at the 120 minute block (for PDs, Control: -13.9 ± 8.6 mm; Fatigue: -23.4 ± 22.2

mm; p = 0.0477), and in the right foot, the two sessions were different from each other at the 45 minute (Control: 4.9 ± 12.1 mm; Fatigue: 14.7 ± 14.2 mm; p = 0.0026), 105 minute (Control: 7.9 ± 8.2 mm; Fatigue: 16.2 ± 16.0 mm; p = 0.0438), and 120 minute blocks (Control: 6.9 ± 13.3 mm; Fatigue: 16.9 ± 16.6 mm; p = 0.0024). There were also differences in right foot medial-lateral centre of pressure means with PDs being more medial than NPDs in the control session in the 15 minute (PD Control: 5.2 ± 10.2 mm; NPD Control: 12.6 ± 9.1 mm; p = 0.0073), 30 minute (PD Control: 7.6 ± 7.8 mm; NPD Control: 14.5 ± 8.1 mm; p = 0.0047), 45 minute (PD Control: 4.9 ± 12.1 mm; NPD Control: 14.6 ± 8.4 mm; p = 0.0013) and 120 minute block (PD Control: 6.9 ± 13.3 mm; NPD Control: 14.5 ± 8.4 mm; p = 0.0103), however this finding was confined to the right foot (left foot: p > 0.4225).

In the anterior-posterior direction, there was a *TIME*PAIN* interaction where PD's centre of pressures moved posterior over time. The means in the first hour were more anterior (larger) than the means in the second hour of standing for the left foot (Table 4.15). The right foot did not have this *TIME*PAIN* interaction, however there was a main effect of *TIME* where the centre of pressure was more anterior in the 15 minute block than in the 120 minute block (15 minute: 141.9 ± 17.9 mm; 120 minute: 133.7 ± 22.7 mm; p = 0.0490).

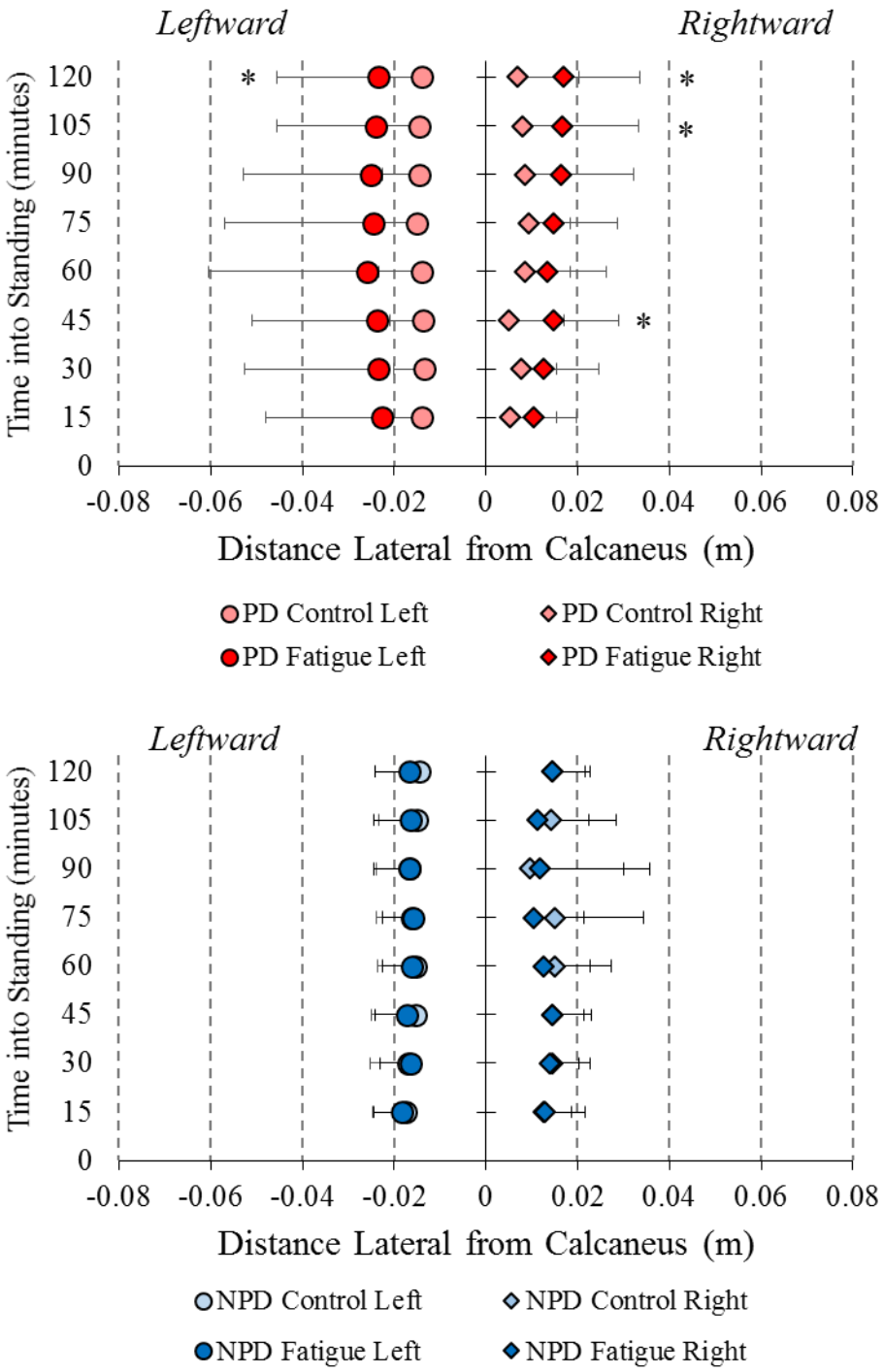

Figure 4.19: Lateral Centre of Pressure Shifts with Fatigue. Lateral distance from the calcaneus is on the x-axis and each 15 minute block is on the y-axis. Positive indicates right of the calcaneus and negative is left of the calcaneus. Circles refer to the left foot and diamonds refer to the right foot. Significant effects of *SESSION* ($p < 0.05$) are indicated by asterisks. In PDs (top panel), there was a shift lateral with fatigue whereas NPDs (bottom panel) did not changes between sessions.

Table 4.15: Posterior Movement of the Centre of Pressure over time in PDs. Time blocks are in the grey shaded areas, the associated means (and standard deviations) are below or to the right of each time block. Probabilities for each time pair are positioned at the intersection of that pair. Significant findings ($p < 0.05$) are bolded and italicized.

PDs	**Later Time Points**	**75 Minutes**	**90 Minutes**	**105 Minutes**	**120 Minutes**
Earlier Time Points	***Centre of pressure (mm anterior of calcaneus)***	*118.5 (16.5)*	*118.7 (13.0)*	*118.4 (14.2)*	*119.6 (15.1)*
15 Minutes	*128.1 (14.3)*	***P = 0.0089***	***P = 0.0002***	***P = 0.0022***	***P = 0.0036***
30 Minutes	*122.3 (12.5)*	P = 0.2377	***P = 0.0331***	P = 0.1002	P = 0.1644
45 Minutes	*121.9 (13.2)*	P = 0.1258	***P = 0.0037***	***P = 0.0232***	***P = 0.0443***
60 Minutes	*122.2 (14.8)*	P = 0.1115	***P = 0.0046***	***P = 0.0288***	***P = 0.0378***

There were additional *PAIN* differences ($p < 0.0488$) in anterior-posterior centre of pressure means. In the left foot, NPDs had more anterior centre of pressures than PDs during the 105 minute block (PD: 118.4 ± 14.2 mm; NPD: 122.9 ± 15.0 mm; $p = 0.0488$). In the right foot, female PDs had more anterior centre of pressures than female NPDs in the 105 minute time block (female PD: 138.6 ± 13.7 mm; female NPD: 126.6 ± 11.8 mm; $p = 0.0246$). Within NPDs, males had more anterior right foot centre of pressure means than females in the 30 minute (male NPD: 142.6 ± 18.6 mm; female NPD: 132.3 ± 17.8 mm; $p = 0.0074$), 75 minute (male NPD: 140.9 ± 19.2 mm; female NPD: 130.1 ± 19.0 mm; $p = 0.0437$), 90 minute (male NPD: 139.4 ± 14.4 mm; female NPD: 123.5 ± 14.2 mm; $p = 0.0471$) and 105 minute time blocks (male NPD: 137.9 ± 19.6 mm; female NPD: 126.6 ± 11.8 mm; $p = 0.0200$).

There was also a *GENDER*PAIN* interaction where female PDs had larger centre of pressure standard deviations than male PDs during standing. In the anterior-posterior direction,

female PDs had larger right foot centre of pressures standard deviations than male PDs in the 30 minute (male PD: 13.3 ± 6.8 mm; female PD: 19.6 ± 8.9 mm; $p = 0.0255$), 75 minute (male PD: 15.4 ± 5.8 mm; female PD: 23.3 ± 8.3 mm; $p = 0.0132$), 90 minute (male PD: 17.9 ± 5.9 mm; female PD: 25.4 ± 11.5 mm; $p = 0.0137$), and 120 minute time blocks (male PD: 19.1 ± 6.8 mm; female PD: 28.7 ± 10.6 mm; $p = 0.0064$). There were no differences between male and female PDs in left foot anterior-posterior centre of pressure standard deviations ($p > 0.1438$). In the medial-lateral direction, females had similarly larger left foot standard deviations in the 30 minute to 120 minute time blocks (range of differences from 2.7 mm and $p = 0.0127$ in the 30 minute block, to 6.0 mm and $p = 0.0001$ in the 105 minute block). In the right foot, this pattern was only found within the 45 minute time block (male PD: 4.1 ± 1.9 mm; female PD: 12.0 ± 13.3 mm; $p = 0.0014$).

Centre of pressure standard deviations tended to increase with time across sessions in both feet. In the anterior-posterior direction, the 120 minute time block (21.7 ± 7.0 mm) was larger than the 15 minute (15.5 ± 6.3 mm; $p < 0.0001$), 30 minute (18.4 ± 7.9 mm; $p < 0.0001$) and 75 minute time blocks (20.5 ± 7.3 mm; $p = 0.0345$) in the left foot for all groups. In the right foot, the 120 minute time block (23.3 ± 9.6 mm) had larger standard deviations than the first 75 minutes of standing (15 minute: 14.4 ± 7.7 mm, $p < 0.0001$; 30 minute: 16.6 ± 8.2 mm, $p < 0.0001$; 45 minute: 19.6 ± 9.1 mm, $p = 0.0001$; 60 minute: 20.1 ± 9.0 mm, $p = 0.0046$; 75 minute: 20.8 ± 8.9 mm, $p = 0.0027$). In the medial-lateral direction, the 45 to 120 minute time blocks had larger left foot centre of pressure standard deviations than the 15 and 30 minute time blocks (Table 4.16), but only in females. For the right foot, the 120 minute time block was larger than the 15 minute block in both genders (15 minute: 4.4 ± 7.3 mm; 120 minute: 7.2 ± 4.6 mm; $p = 0.0036$).

Table 4.16: Increases in Right Foot Medial-Lateral Centre of Pressure Standard Deviations over Time in Females. Time blocks are in the grey shaded areas, the associated means (and standard deviations) are below or to the right of each time block. Probabilities for each time pair are positioned at the intersection of that pair. Significant findings ($p < 0.05$) are bolded and italicized.

Females	**Later Time Points**	**45 Minutes**	**60 Minutes**	**75 Minutes**	**90 Minutes**	**105 Minutes**	**120 Minutes**
Earlier Time Points	***Right Foot M/L CoP Standard Deviations (mm)***	*7.6 (5.9)*	*7.7 (5.2)*	*8.0 (5.3)*	*8.3 (4.6)*	*8.7 (5.0)*	*8.0 (5.6)*
15 Minutes	*4.2 (2.8)*	***0.0057***	***0.0020***	***0.0025***	***0.0011***	***0.0001***	***0.0065***
30 Minutes	*5.7 (3.6)*	***0.0272***	***0.0243***	***0.0141***	***0.0095***	***0.0001***	0.0666

There were *GENDER*PAIN* group interactions for fidget sizes. In males, PDs had larger sized fidgets than NPDs in the 15 to 45 minute time blocks (15 minutes, male PD: 4.3 ± 0.5 SDs; male NPD: 4.0 ± 0.3 SDs; $p = 0.0242$; 30 minutes, male PD: 4.3 ± 0.8 SDs; male NPD: 3.9 ± 0.4 SDs; $p = 0.0168$; 45 minutes, male PD: 4.5 ± 0.6 SDs; male NPD: 3.9 ± 0.3 SDs; $p = 0.0011$), however NPDs had larger sized fidgets in the 60 minute time block (male PD: 3.9 ± 0.3 SDs; male NPD: 4.1 ± 0.7 SDs; $p = 0.0152$). There were also *PAIN*SESSION* interactions wherein the control session, PDs had larger fidgets than NPDs in the 45 minute time block (PD control: 4.5 ± 0.6 SDs; NPD control: 3.9 ± 0.4 SDs; $p = 0.0006$), yet NPDs had larger fidgets than PDs in the 105 minute block (PD control: 4.0 ± 0.3 SDs; NPD control: 4.5 ± 1.3 SDs; $p = 0.0320$). Additionally there was a *TIME*PAIN* interaction where PDs had smaller sized fidgets in the 60 minute time block (3.8 ± 0.3 SDs) than during all other time blocks except for the 105 minute block (for 105 minutes, $p = 0.2997$; for other time blocks, $p < 0.0351$; Figure 4.20).

The number of body weight transfers, shift number, shift size, and fidget size were not affected by *SESSION* (body weight transfer $p > 0.1438$; shift number $p > 0.4142$; shift size $p > 0.1857$; fidget size $p > 0.2426$). There was a trend of increasing the number of fidgets with fatigue in the first hour of standing (Figure 4.21), approaching significance in the 30 minute (Control: 38.9 ± 24.4 fidgets; Fatigue: 52.4 ± 33.4 fidgets; $p = 0.0519$) and 45 minute blocks (Control: 30.0 ± 19.5 fidgets; Fatigue: 40.1 ± 23.2 fidgets; $p = 0.0537$).

There were time effects for weight support strategy variables. There was a *TIME*GENDER*SESSION* interaction where females in the fatigue session had fewer body weight transfers in the 45 to 75 minute time blocks than in the 105 and 120 minute time blocks ($p < 0.0361$). There was also a main effect of *TIME* in the number of body weight transfers that appeared to be much more prevalent than the *TIME*GENDER*SESSION* interaction (Figure 4.23). When averaged across all conditions, there were fewer body weight transfers in the 15 minute block than all other time blocks ($p < 0.0021$), the 30 minute time block had fewer body weight transfers than every other time block following it ($p < 0.0179$), and the 45 minute time block had fewer body weight transfers than the 90 and 105 minute time blocks ($p < 0.0342$). The only observable difference between the female fatigue curve and the other participants is a reduction in body weight transfers in the 45 to 60 minute blocks (at 45 minutes, female fatigue: 14.3 ± 12.1 transfers, all others: 28.4 ± 44.1 transfers; at 60 minutes, female fatigue: 18.1 ± 19.4 transfers, all others: 30.8 ± 39.4 transfers). A comparison of these two time effects in body weight transfers are shown in Figure 4.22. NPDs had more fidgets in the 30 minute block than in the 120 minute block (30 minute: 46.8 ± 33.4 fidgets; 120 minute: 35.8 ± 23.1 fidgets; $p = 0.0278$). Across gender and pain groups, the 60 minute time block had more shifts than the 120 minute block (60 minutes: 6.0 ± 5.2 shifts; 120 minutes: 4.8 ± 5.1 shifts; $p = 0.0340$).

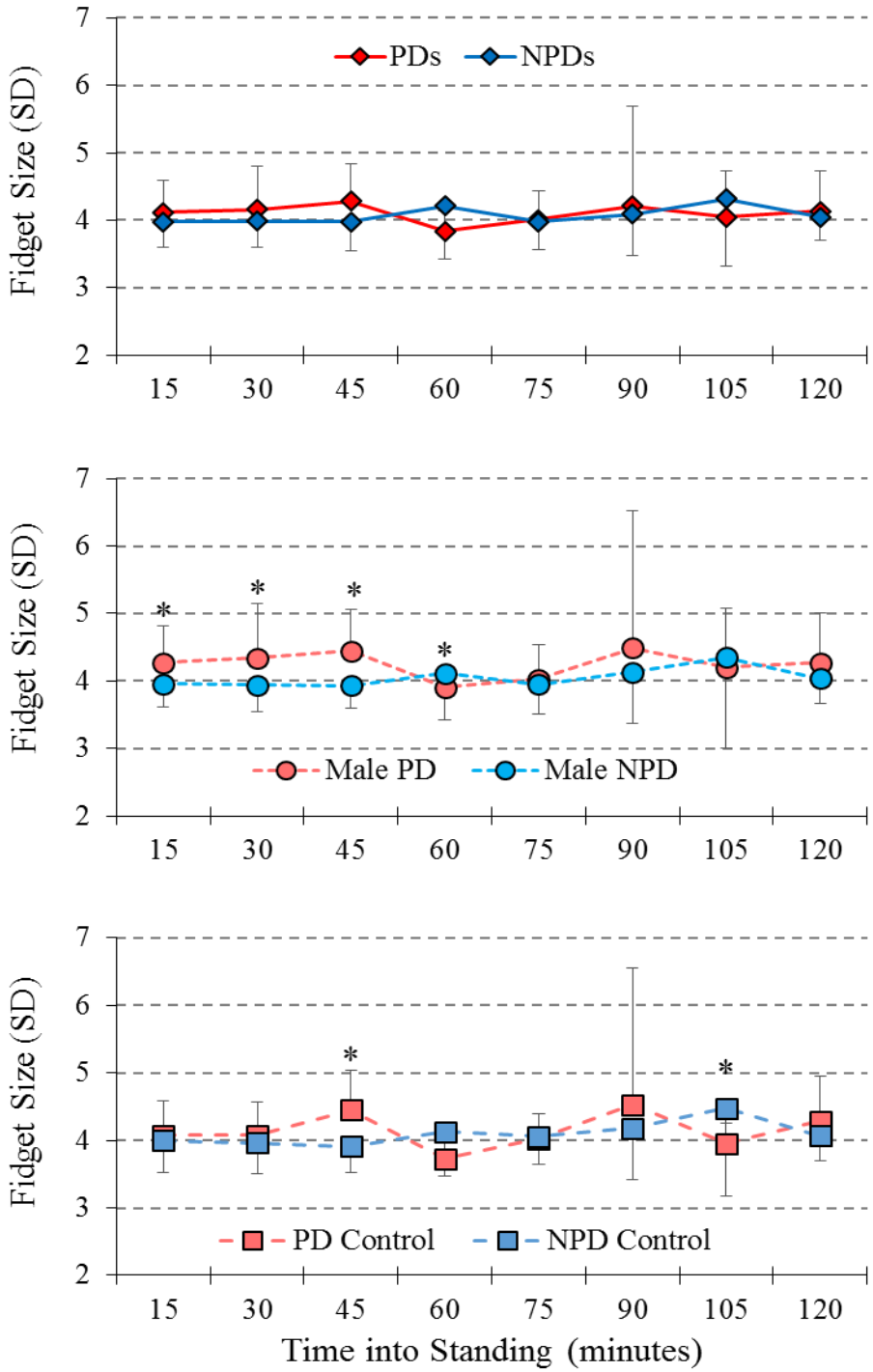

Figure 4.20: Pain Group Differences in Fidget Size. Significant effects of *PAIN* ($p < 0.05$) are indicated by asterisks. As a whole, PDs had smaller fidgets in the 60 minute time block compared to all others, NPDs did not show this decrease (top panel). In males (middle panel) PDs had larger fidgets earlier, until that drop at 60 minutes. In the control session (bottom panel), PDs and NPDs would alternate between having larger or smaller fidgets. The minimum fidget size is 2 standard deviations, a standard deviation refers to the anterior-posterior centre of pressure in a preceding window.

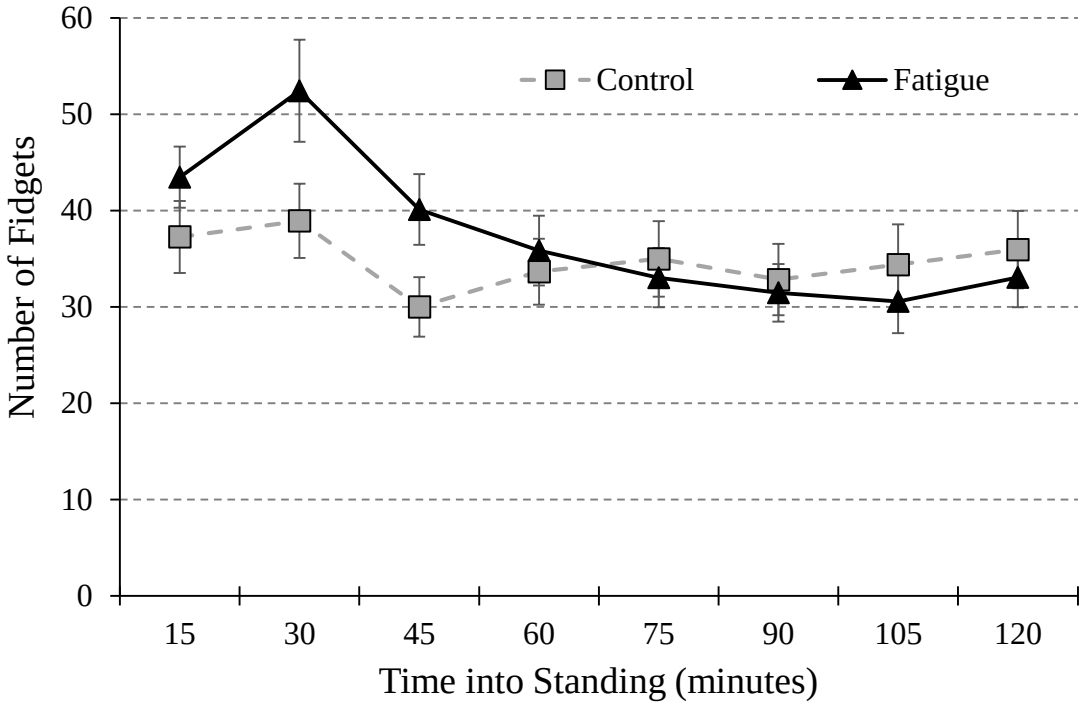

Figure 4.21: Transient Increase in Fidgets with Fatigue. There was a trend of participants having more fidgets during the fatigue session in the 30 and 45 minute blocks, however this difference was not significant ($p > 0.05$). Error bars show standard errors.

Additionally, females shifted more frequently than males in the 30 minute (males: 3.73 ± 3.75 shifts; females: 5.95 ± 5.46 shifts; $p = 0.0496$), 45 minute (males: 4.38 ± 4.22 shifts; females: 6.88 ± 6.03 shifts; $p = 0.0436$) and 90 minute time blocks (males: 4.00 ± 3.77 shifts; females: 6.43 ± 4.81 shifts; $p = 0.0221$).

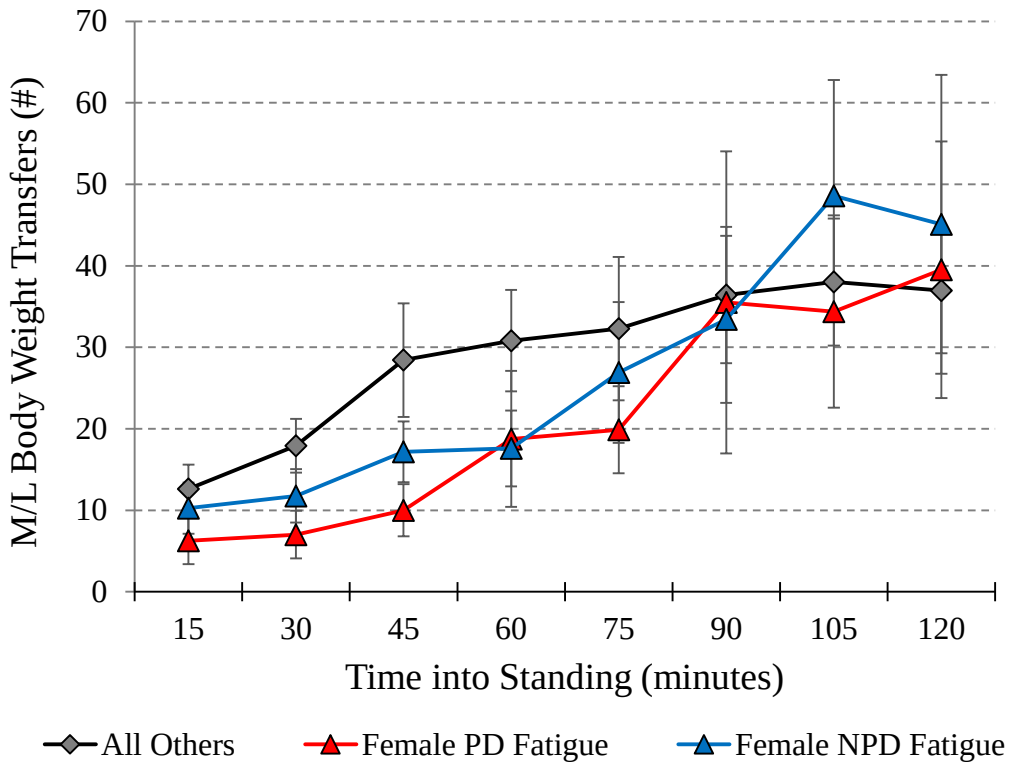

Figure 4.22: Changes in Medial-Lateral Body Weight Transfers over Time. Statistical testing indicated a time interaction specific to females of both pain groups in the fatigue session. However, there was a much strong main effect of time over all groups. It appears as though the differences between females in the fatigue session and all other groups occurs in the 45 minute to 60 minute time blocks. Error bars show standard errors.

Section 5: DISCUSSION

The hypotheses presented in the introduction are revisited in light of the results.

Hip abductor fatigue will remove the bilateral gluteus medius cocontraction response from pain developers. Partially accept. While there were early decreases in the bilateral gluteal CCI in PDs in response to the fatigue protocol, this drop in CCI was only transient, reverting to control session values after 45 minutes of standing. Interestingly enough, NPDs had increases in this same cocontraction index in response to fatigue. Cocontraction indices involving the external obliques and lumbar erector spinae were also found to decrease with hip abductor muscle fatigue.

The fatigue protocol will act as a hyperalgesic stimulus, increasing the severity of pain experienced by pain developers but not non-pain developers. Reject. Contrary to findings regarding coupling muscle fatigue with pain induced by hypertonic saline injections (Ciubotariu et al., 2007), PDs reported less low back pain during standing in the fatigue session, suggesting that the exercise performed here was an analgesic stimulus. This is further supported by NPDs reporting similar levels of low back pain between sessions.

The fatigue protocol will also cause non-pain developers to change their postural responses so that their postural responses match the pain developer responses. Reject. NPDs postural responses were generally unaffected by the fatigue protocol, providing evidence that hip abductor muscle fatigue does not simulate low back pain during standing. Rather PDs showed postural responses to the fatigue protocol with male and female PDs exhibiting different patterns.

Pain developers will show greater hip abductor fatigue over the course of the control session and have a slower recovery of fatigue during the fatigue session than does who do not develop pain. Partially accept. While PDs did not show any evidence of muscle fatigue during the control

session, they had a quicker onset of fatigue than NPDs and, unlike NPDs, were unable to recover from hip abductor fatigue while standing. Since standing prevented PDs from recovering from hip abductor fatigue, this suggests differential recruitment of the hip abductors during prolonged standing between pain groups.

The persons whose pain reporting patterns differ between the control and fatigue session, termed fatigue sensitive, will be identifiable by either a muscle activation parameter or a postural variable. That indicator will be different between the fatigue sensitive pain developer and the fatigue sensitive non-pain developer. Unable to comment. While fatigue sensitivity was not characterized due to its rare occurrence, this phenomenon was much more commonly observed in PDs than in NPDs.

The main topic of discussion based on the hypotheses concerns how hip abductor muscle fatigue reduced pain in PDs. Separate sections are devoted to determining the usefulness of the fatigue protocol and relations between cocontraction indices and muscle fatigue.

5.1 How Did Fatigue Reduce Low Back Pain in PDs?

Both male and female PDs had reductions in peak low back pain in the fatigue session, however there were gender differences in the patterns and magnitude of pain reporting during the fatigue session. There were also gender differences in muscular and postural variables during standing in the fatigue session. Both of these observations suggest differing mechanisms between genders in how the fatigue protocol had reduced low back pain. Potential variables responsible for pain changes with fatigue are shown in Table 5.1. All effects of *SESSION* regardless of their specificity to PDs were included with findings separated by gender.

Table 5.1: Variables that could Influence Pain Development with Hip Abductor Fatigue in Males and Females. Changes from control to fatigue are specified by arrows or descriptors where appropriate. See text below for explanations why variables were placed on their respective sides of the table. Variables that are italicized are those without conclusive evidence that changes would increase or decrease reported pain and are included in both sides of the table.

*These cocontraction indices were heavily influenced by a single participant and not representative of Male PDs as a whole.

	Changes with Fatigue that could *Reduce* Low Back Pain			**Changes with Fatigue that could *Increase* Low Back Pain**		
	EMG	Kinematic	Force Plate	EMG	Kinematic	Force Plate
Males	• ↓ R-GMD/R-LES* • ↓ R-LES/R-EXO* • ↓ R-TES/L-TES	• ↑ *Hip Flexion*	• N/A	• N/A	• ↓ Posterior Pelvic Tilt • ↓ Lumbar Flexion • ↑ *Hip Flexion*	• N/A
Females	• ↓ R-TFL/L-TFL • ↑ L-EXO Gaps • ↓ *R-TFL Cumulative* • ↓ *R-LES Cumulative*	• ↑ Posterior Pelvic Tilt • ↑ Lumbar Flexion • ↑ *Hip Abduction* • ↓ *Hip Flexion*	• N/A	• ↓ *R-TFL Cumulative* • ↓ *R-LES Cumulative*	• ↑ Lateral Pelvic Tilt • ↑ *Hip Abduction* • ↓ *Hip Flexion*	• Fewer Body Weight Transfers
Both	• ↓ R-GMD/L-GMD • ↓ R-LES/R-EXO • ↓ L-LES/L-EXO • ↓ R-LES/L-LES • ↓ L-GMD/L-LES • ↑ *R-TES Cumulative* • ↓ *L-TES Cumulative*	• N/A	• ↑ Fidget Frequency • ↑ *Lateral Movement of CoP*	• ↓ R-INO Gaps • ↑ *R-TES Cumulative* • ↓ *L-TES Cumulative*	• N/A	• ↑ *Lateral Movement of CoP*

5.1.1 Changes in Muscular Responses

The fatigue protocol reduced a number of cocontraction indices. Previous work has identified increases in bilateral gluteal (R-GMD/L-GMD) and ipsilateral trunk cocontraction (R-LES/R-EXO and L-LES/L-EXO) as being indicative of low back pain development during standing (Nelson-Wong et al., 2008; Nelson-Wong and Callaghan, 2010b; Marshall et al., 2011). Most reductions in cocontraction indices with fatigue in this study occurred within the first hour of standing (Figures 4.8 and 4.9; Section 4.3.1), starting with a reduction of R-GMD/L-GMD and R-LES/R-EXO in the first 15 minutes, and reductions in R-LES/L-LES and L-LES/L-EXO occurring over the next 45 minutes. This change with fatigue mimics the responses of NPDs identified by Nelson-Wong and Callaghan (2010b): initial decreases in cocontraction resulted in no pain development in NPDs, despite similar amounts of cocontraction between pain groups during the later times when PDs reported their onset and rapid rise of low back pain. Therefore, these early reductions in cocontraction indices could have reduced the eventual level of pain in PDs during the fatigue session.

Abdominal bracing through trunk cocontraction is often desired in order to prevent low back pain or injury by producing compression of and stiffness around the spinal column. There is evidence that the stiffness provided by trunk cocontraction is more beneficial in preventing injuries in dynamic tasks (e.g. lifting) than any detriment to vertebral endplates caused by higher joint compression as a result of that cocontraction (Granata and Marras, 2000). Reduced trunk cocontraction, as seen in the fatigue session, would reduce this buttressing effect of muscular stiffness (McGill and Norman, 1986; Granata and Marras, 1995, 2000), thereby reducing the capacity of the spine system to tolerate perturbations and resulting in an increased risk of injury (Oxland et al., 1991; Cholewicki and McGill, 1996). However in prolonged standing, the main

threat to musculoskeletal health comes in the static, prolonged nature of the task rather than more historic causes such as vertebral displacement or compressive spinal loading (van Dieën and Oude Vrielink, 1998; Callaghan and McGill, 2001). Levels of muscle activity during standing are below static level injury thresholds (Callaghan and McGill, 2001), and involve relatively neutral spine postures (Endo et al., 2012), which further emphasize that the risks of injury from prolonged standing are different from those in dynamic trunk tasks, repetitive or otherwise. It is the "prolonged" part of prolonged standing that makes it a method to induce pain (Callaghan and McGill, 2001; Wells et al., 2007), and increases the likelihood of musculoskeletal injury (Xu et al., 1997). Pain does not develop in a young healthy population if the standing exposure is only 1 minute in length, and the most effective method of pain relief for PDs is stopping them from standing. Muscular cocontraction facilitates rigidity about a joint, and in many cases this is desired (Oxland et al., 1991; Cholewicki and McGill, 1996; Granata and Marras, 2000; Kellis et al., 2011), however it is this very same rigidity that makes prolonged standing painful (Gregory and Callaghan, 2008; Gallagher et al., 2011), and associated with workplace injury (Xu et al., 1997; Tissot et al., 2009). The theory explaining why the bilateral gluteal cocontraction index is predictive of back pain in standing is that it prevents the lateral shifting of body weight between the feet (Nelson-Wong et al., 2008; Gallagher et al., 2011). Assuming that greater cocontraction of the trunk and hip facilitates holding static postures, having less cocontraction about the trunk and lower limb would be beneficial for preventing the development of low back pain. The observed increase in centre of pressure fidgets in the first 45 minutes of the fatigue session aligns with this theory (Figure 4.21).

A second viewpoint incorporates the activation-sensitive nature of the cocontraction index used in this study. Cocontraction decreases with the fatigue intervention could be a result of less

co-activity between muscle pairs, or lower activity level of either muscle in the summation windows (Lewek et al., 2004). Less cocontraction in the fatigue session could then indicate that one or both of the muscles in the pair investigated were less active during that session. The outlier case mentioned in the results (Section 4.3.1), was an example of this where an increase in R-LES activity in the last hour of the control session caused a massive increase in two cocontraction indices involving the right lumbar erector spinae. If a muscle's activity level decreased in the fatigue session, it could then be apparent through decreases in cocontraction indices involving that muscle. Reductions in cocontraction indices involving at least one lumbar spine stabilizer could indicate a greater contribution of passive tissues to intervertebral joint stability. Although a larger contribution of passive structures would indicate less tissue damage in a chronic pain population (Geisser et al., 2005), chronic pain is fundamentally different from the transient pain induced by standing (Loeser and Melzack, 1999), and this conclusion may not be applicable.

It is useful to bring up the outlier case for another reason. Had FX9's data not been identified as aberrant, the stark differences with fatigue in the two affected cocontraction indices could have provided an explanation for the male PD pain reporting response. The low back pain plateau in the Male PD's fatigue session started after 75 minutes on standing. It was during these same time blocks that the R-GMD/R-LES and R-LES/R-EXO cocontraction indices showed changes with fatigue, a change occurring exclusively in male PDs (Figure 4.7). Had this been the case, it could be concluded that male PD's pain development was more associated with low back and gluteal muscular responses than female PDs. This finding could also explain a discrepancy in Nelson-Wong and Callaghan (2010a). Although an exercise intervention targeting core stabilization techniques was effective at reducing low back pain in PDs of both genders, only male PDs showed any muscular changes during standing as a result of the training intervention. This

does not necessarily rule out that male PD's pain development is preferentially linked to muscle activity patterns during standing, but the pain reduction in male PDs observed in this study cannot conclusively be linked directly to the changes in R-GMD/R-LES and R-LES/R-EXO cocontraction brought about by hip abductor fatigue.

Gaps in muscle activity have previously been shown to be beneficial in separating those who do and do not develop pain from quasi-static exposures in that more gaps were found to indicate both less (Veiersted et al., 1990), or more pain development (Gregory and Callaghan, 2008). However, the positive outcomes of more frequent and longer gaps were based on identifying muscular sources of pain and have are often localized to the neck and shoulder region (Veiersted et al., 1990; Hagg and Astrom, 1997; Sandsjo et al., 2000; Westgaard et al., 2001). Also, this set of studies only included female participants (Veiersted et al., 1990; Hagg and Astrom, 1997; Sandsjo et al., 2000; Westgaard et al., 2001), which may account for the plethora of gap measure differences in this study that were only seen in females.

For prolonged standing, reductions in pain should be associated with more gaps in either hip abductor or postural support musculature. With more frequent and longer duration gaps in gluteus medius activity, there is a higher likelihood of lower activation levels in that muscle, and over long periods of time, would reduce the activation-sensitive cocontraction indices involving gluteus medius that predict low back pain in prolonged standing (Nelson-Wong and Callaghan, 2010b). In postural support muscles such as the erector spinae, having more gaps would also be beneficial as it would slow the progression of fatigue (Paquet and Nirmale, 2004; Østensvik et al., 2009; Yung et al., 2012). However, the two changes in gap frequency with fatigue occurred in the obliques, with L-INO having fewer gaps with fatigue (both genders; Figure 4.13), and R-EXO having more gaps with fatigue (females only; Section 4.3.1) Since there were changes in opposite

direction in muscles with similar roles in the tasks studied – providing stiffness about the spinal column (McGill et al., 2003), it is unlikely that there would be any net change in pain responses as a result of gap frequency.

The gap measures that most commonly changed with fatigue were cumulative rest times, measures that have not been linked to muscular pain or injury to the same extent of gap frequency or length. Cumulative rest time was computed to give an idea of muscle use over the course of a 15 minute block. In this study, the distributions of cumulative rest time measures were often bimodal, with one subset of participants within a pain or gender group having values around 900 seconds (the muscle was mostly inactive) and another subset having values less than 100 seconds (the muscle was mostly active). Changes in group means with fatigue were often based on the number of participants that moved from one subset to the other between sessions rather than uniform decreases across all participants. Three muscles were observed to have smaller cumulative rest times with fatigue, indicating more muscle use (L-TES, R-LES and R-TFL, the latter two only in females) while one muscle was observed to have a larger cumulative rest time (R-TES).

The decrease in R-TFL cumulative rest time in female PDs (Figure 4.10) is of note for two reasons. Firstly, it was the most consistent change in any gap variable with fatigue, with sessional differences between female PDs and female NPDs occurring in every time block of the standing protocol. Of greater note, this decrease in cumulative rest time could potentially indicate a compensation where R-TFL is more active to make up for the lack of force generating capacity in the R-GMD due to fatigue. If so, then this change in activity could contribute to the lack of force recovery in female PDs while standing (Gottschalk et al., 1989), female PD's lateral shift in centre of pressures (Winter et al., 1996; McMullen et al., 2011; Lee and Powers, 2013), or both. Interestingly, there was a similar magnitude difference in R-TFL cumulative rest times between

male and female NPDs regardless of session (Figure 4.10). Although not related to reductions in pain with fatigue, this may be an indicator of differences between genders where in contrast to females, males do not utilize TFL during standing as much as females. With respect to the thoracic and lumbar erectors, the lack of a unified direction of change in paraspinal cumulative rest times suggest that, like the gap frequency measures, they would have minimal net effects on pain development during standing.

Muscular changes with fatigue that were associated with a reduction in hip and trunk rigidity appeared to help reduce the low back pain experienced by PDs. This was observed through reductions in CCIs in muscle pairs involving GMD, LES and EXO. A reduction in R-TFL cumulative rest time in females may not have reduced low back pain, but did act as an indicator of muscle fatigue.

5.1.2 Changes in Spine Postures

Gender and pain group differences in spine and hip postures during the fatigue session appeared to be driven by the orientation of the pelvis. Female PDs had more posteriorly and laterally tilted pelvis orientations with fatigue (Figures 4.14 and 4.16), which led to greater flexion of the lumbar spine and extension at the hip (Table 4.9). Male PDs had more anteriorly tilted pelvic orientations with fatigue (Figure 4.14), which likewise led to greater lumbar spine extension and hip flexion (Table 4.9; Section 4.3.2). The magnitude of differences observed here ranged from 4 to 7 degrees, roughly two to three and a half times the repositioning error of the lumbar spine in healthy adults (Gill and Callaghan, 1998; Rausch Osthoff et al., 2015), indicating that these postural shifts were probably not due to chance. Standing aids implemented for the prevention of low back pain during standing, such as a sloped surface or a raised ledge, try to manipulate the pelvis such that it is more posteriorly tilted either though ankle plantar flexion (sloped surface) or

hip flexion (raised ledge). In either case, the interventions are thought to drive lumbar flexion while standing by indirectly manipulating the pelvis to rotate posteriorly (Day et al., 1984; Levine and Whittle, 1996).

More posterior pelvic tilt is believed to be desired for PDs because, as intended by the standing aids, it would move PDs into more lumbar spine flexion. An assessment of lumbar curvature in a relaxed (non-working) standing posture found that those with more lumbar lordosis developed more pain over 2 hours of standing (Sorensen et al., 2015). Although there is mixed evidence as to whether PDs stand in more lumbar extension than NPDs while performing workplace tasks (Gallagher, 2014; Gallagher et al., 2014; there were no pain group differences in the current study during the control session – Tables 4.9 and 4.10; Section 4.3.2), pain group differences were more apparent when focusing on the L5/S1 intervertebral joint where PDs were observed to stand in more L5/S1 extension than NPDs (Gallagher, 2014). Additionally, the pelvis-driven spine flexion achieved through sitting (Dunk et al., 2009) is thought to be a rationale behind the effectiveness that sitting breaks have in reducing pain from prolonged standing (Gallagher et al., 2014). Therefore, it is plausible that the posterior pelvic tilt seen in female PDs during the fatigue session was a factor in their reduced peak low back pain. Male PDs however, were more anteriorly tilted with fatigue which should increase spine extension (Levine and Whittle, 1996), and may have exacerbated existing pain development with fatigue (Sorensen et al., 2015). Therefore, these gender differences in pelvic postures with fatigue are a feasible source of the different pain patterns observed between male and female PDs.

Asymmetry in frontal plane pelvic postures has been associated with low back pain. Clinical asymmetries are often slight; with less than 5 degrees of lateral tilting or morphological differences being enough to differentiate healthy from pathological individuals (Al-Eisa et al.,

2004; Herrington, 2011). Initially investigated on the basis of leg-length discrepancies, null findings in these studies (Grundy and Roberts, 1984; Soukka et al., 1991) caused the focus to shift towards relating low back pain to the amount of morphological asymmetries in the pelvis. Persons with low back pain were found to have greater frontal plane pelvic asymmetry in standing, but similar leg lengths to healthy controls (Al-Eisa et al., 2004, 2006). Additionally, those with low back pain tend to have greater movement asymmetries in the frontal and transverse planes when performing primarily sagittal plane movements (Al-Eisa et al., 2006; Van Dillen et al., 2007; Kim et al., 2013). Whether or not frontal plane asymmetries in standing and forward flexion tasks can cause low back is still unknown due to the cross-sectional nature of these studies. Also of note, this work has been focused on patients with chronic pain and is not necessarily applicable to those with pain in standing, although a pain group identified in two of the studies (rotation with extension - Van Dillen et al., 2007; Kim et al., 2013) were similar to PDs in that they stood in more lumbar spine extension than a healthy control group (Sorensen et al., 2015). There appears to be evidence that the lateral pelvic tilt seen in female PDs in the fatigue session could have increased their low back pain based on evidence from chronic pain populations. In spite of the lateral tilt, female PDs reported less low back pain in the fatigue session compared to the control session. The measured lateral pelvic tilt in this study is likely more similar to leg-length discrepancies (a morphologically symmetric pelvis is tilted) than the pathological pain cases (a level pelvis is asymmetric in shape). It is highly unlikely that the fatigue protocol induced transient changes in pelvis geometry during the standing protocol as the time required for bone-related shape changes in response to a mechanical exposure is measured in weeks or months (Villemure and Stokes, 2009). Female PDs may still have the lateral asymmetries in sagittal motions similar to patients in Van Dillen et al., (2007) or Kim et al., (2013), or innate morphological asymmetries in their pelvises like the patients

in Al-Eisa et al., (2004, 2006) as neither were measured in this study. Regardless of outcome, it appears that the amount of pain induced via pelvic asymmetries from the fatigue protocol (Figure 4.16) is either negligible to the development of, or was washed out by other more potent moderators of low back pain in standing.

There are reported gender differences in naturally adopted pelvic and spine postures. In upright standing, females have a tendency to stand in more lumbar spine extension and anterior pelvic tilt than males (Norton et al., 2004; Vialle et al., 2005; Janssen et al., 2009). These differences appear to be based on the individual orientation of thoracic and lumbar vertebrae (Janssen et al., 2009), and does not appear to affect spine range or motion (Dvořák et al., 1995; Kienbacher et al., 2015). As it has been previously noted that PDs tend to stand in more lumbar spine extension than NPDs (Sorensen et al., 2015), there may be an interaction where female PDs naturally stand in more lumbar spine extension than male PDs. If a gender difference in sagittal postures within PDs does exist, it may have influenced the opposing postural compensations with hip abductor fatigue. Although there were no gender differences in either pain group with regards to any sagittal plane angle measured in the control session current study (Figure 4.14 and Tables 4.9 and 4.10), this may have been due to the constraints imposed by the typing and sorting tasks and does not rule out a PD-specific gender difference in spinal curvature.

Postural changes with fatigue were divergent between genders, and were the measures most likely responsible for gender differences in pain reporting during the fatigue session of those observed in this study. The posterior pelvic tilt and lumbar spine flexion seen in female PDs aligned with reductions in low back pain during the fatigue session. Although female PDs also exhibited lateral deviations in lumbo-pelvic postures during the fatigue session, these postural shifts did not appear to affect low back pain development during standing. The anterior pelvic tilt

and lumbar spine extension seen in male PDs had the potential to increase reported low back pain during standing, however this was not observed here. Perhaps the reductions in muscle cocontraction had a larger effect than these coincident postural shifts in males. This interaction between the changes in muscular rigidity and posture of the trunk and hip may have affected the time varying low back pain responses in male and females in different manners.

5.1.3 Changes in Force Plate Measures

There has been a paradoxical finding of persons with low back pain having both more movement and less movement during standing. The argument that more movement is beneficial takes a preventative stand point: having increased movement prior to the development of pain will prevent the development of low back pain. This can be seen in the success of sit-stand workstations (Davis and Kotowski, 2014; Karakolis and Callaghan, 2014), where having workers alternate between sitting and standing more frequently than traditional sitting or standing only workstations reduces worker pain levels in multiple body regions. The counter argument states that those in pain move more frequently because they are agitated by their pain; the presence of pain causes movement. Those with chronic low back pain have been observed to have larger centre of pressure excursions and greater postural sway than healthy controls when standing (Ruhe et al., 2011; Mazaheri et al., 2013), with differences exacerbated in more challenging scenarios (Mientjes and Frank, 1999). These two arguments are not mutually exclusive as they differentiate when the movement is supposed to occur with respect to the development of pain. Therefore, one would expect NPDs to move more frequently at the beginning of a standing exposure to prevent pain development and PDs to move more frequently near the end of a standing exposure in response to their pain.

However, empirical data on prolonged standing does not completely align with this theory. A regression approach identified more anterior-posterior centre of pressure motion in the first 15 minutes of standing as being predictive of higher levels of induced low back pain at the end of that standing exposure (Gregory and Callaghan, 2008). On the other hand, a separate study showed that NPDs had more small amplitude (10 to 29% body weight) medial-lateral weight transfers and lumbar flexion-extension fidgets than PDs in the first 15 minutes of standing (Gallagher, 2014). Findings from this study and prior work (Gallagher et al., 2011) found no pain group differences in larger amplitude medial-lateral body weight transfers (30% body weight or more) in the initial phases of prolonged standing, but both groups performed these transfers more frequently at later points in time (Gallagher et al., 2011). Although not quantified, NPDs from this study anecdotally reported foot and knee pain over the course of standing, which may have provoked their increases in body weight transfers with time. A similar time-varying body weight transfer compensation in PDs could have feasibly resulted from their back pain, as would be expected. However, there is evidence that the onset of foot pain precedes back pain in those employed in standing occupations (Antle et al., 2013; Antle and Cote, 2013), so PDs may have developed foot pain in addition to their back pain from standing. If so, it would be unclear as to whether the lower limb or back pain prompted the reactive increase in weight transfers in PDs.

The three main movement-based changes with the fatigue protocol in this study were that PDs had a lateral shift of centre of pressure in later parts of the fatigue session (Figure 4.19), females (both PDs and NPDs) had fewer body weight transfers in the 45 to 75 minute blocks (Figure 4.22), and that all participants tended to have more anterior-posterior centre of pressure fidgets in the first 45 minutes of standing (Figure 4.21; Section 4.3.3). Based on the above discussion, the early increase in fidgets should decrease eventual pain reporting, as there would be

less continuous time spent in a static posture (van Dieën and Oude Vrielink, 1998; Wells et al., 2007). The timing of the decrease in body weight transfers in females could potentially indicate that this change was reactive instead of preventative. Female PDs on average were still reporting clinically significant levels of pain (> 10/100 mm) in the fatigue session by the end of the 30 minute time block, but less pain than in the control session (Figure 4.1). Since females PDs were in less pain, they did not transfer their weight as much, but this does not account for the same transient drop seen in female NPDs, which was independent of back pain. The lateral shift of centre of pressures also occurred after low back pain was developed in the fatigue session and coincided with the timing of PD's ABC force losses in the fatigue session (the 105 and 120 minute time blocks; Figure 4.6). Introducing hip abductor fatigue into more challenging balance tasks such as lateral reaches, unstable support surfaces or unilateral jump landings tend to increase the variability in medial-lateral centre of pressure derived measures of whole body stability (Salavati et al., 2007; McMullen et al., 2011; Lee and Powers, 2013). In contrast, NPDs had a steady recovery of ABC force and did not show this lateral shift in centre of pressure. The timing of differences that were exclusive to PDs combined with the role of hip abductor musculature in medial-lateral centre of pressure control suggest that this change with fatigue is primarily an indicator of fatigue and likely not a factor directly influencing low back pain (Winter et al., 1996; Lee and Powers, 2013).

The increase in centre of pressure fidgets within the first hour of standing could have reduced low back pain in the fatigue session. It is likely that the reduction in body weight transfers and lateral shifts in centre of pressure were indicators of less pain and greater fatigue respectively rather than causal factors of pain reductions.

5.1.4 Potential Mechanism of Pain Reduction during the Fatigue Session

The major driver of low back pain reductions with hip abductor fatigue appears to be movement. Having less muscle cocontraction in the trunk and hip would allow for more motion during standing. This increase in motion would allow for more frequent off-loading of tissues, reducing any time-varying viscoelastic changes within structures of the low back that would result in pain development. This relationship of motion to pain can be moderated by the general sagittal plane posture adopted, where a more flexed lumbar spine and posterior pelvic tilt can allow for earlier, and more consistent reductions in low back pain over a 2 hour standing exposure as seen in female PDs. A representation of this relationship is shown in Figure 5.1.

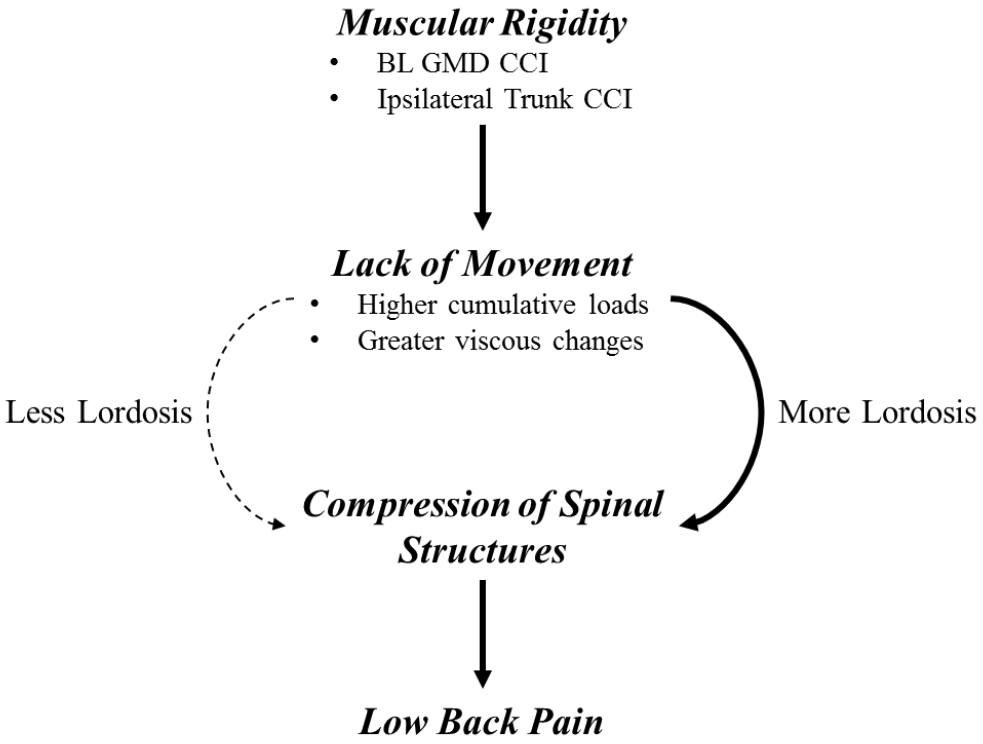

Figure 5.1: Potential Pathway of Low Back Pain Development during Prolonged Standing. Greater muscular cocontraction prevents movement, which results in static loading conditions that result in tissue damage and low back pain. Standing in greater lordosis (lumbar spine extension and anterior pelvic tilt) strengthens this relationship of static loading to pain.

5.2 Effectiveness of the Fatigue Protocol

The fatigue protocol was intended to produce unilateral hip abductor fatigue of relatively similar levels across participants, and have lasting effects during the standing tasks. The fatigue protocol did result in similar perceived levels of fatigue between gender and pain groups, but affected both measured hip abductors bilaterally (Figure 4.3), and up to five additional muscles in some participants (Table 4.4; Section 4.2.1). Females, while taking a longer time to reach the movement quality criteria for cessation of the exercise, had smaller initial force losses than males. NPDs had longer times to fatigue than PDs, again based on the movement quality criteria.

Gender differences in endurance times have been previously established, with females having either similar (Senefeld et al., 2013) or longer endurance times than males (Hunter and Enoka, 2001; Albert et al., 2006). Although this difference has been attributed to females having slower rises in mean arterial pressure than males with exercise (Hunter and Enoka, 2001), the differences seen in this study could also be attributed to males having more leg mass and having longer legs than females. With greater leg mass and having that mass further away from the hip joint centre of rotation, there would be a larger gravitational moment to overcome in order to abduct the leg to the same absolute angle. In this respect, the load was somewhat tailored to the individual in that stronger participants would have more leg mass. In spite of this, there was still a wide range of activation levels between participants, with some participants exceeding 90 %MVIC throughout the protocol in multiple muscles and others rarely surpassing 30 %MVIC in their hip abductors. However some of the variation in EMG amplitude could be a byproduct of the fatiguing exercise being a dynamic task (De Luca, 1997).

Both genders exhibited similar losses in mean power frequencies and similar ratings of perceived exertion following the fatigue protocol. As previously mentioned, differences in

endurance times were likely related to gender differences in blood pressure responses (Hunter and Enoka, 2003) or anthropometry. Both would have been possible to control for, anthropometry through selective participant recruitment and blood pressure through pressurized cuffs about the active muscles, though it would limit the validity of the findings to a non-representative population and would reduce the fatigue recovery times as the pressurized cuff technique also reduces endurance times (Hunter and Enoka, 2003). Even though there were gender differences in endurance times, those differences would be expected for similar levels of fatigue and that aspect likely did not influence the gender differences in pain development during the fatigue session.

The gender differences in relative force losses during the fatigue session may provide more insight into potential gender differences in the resulting muscle fatigue. The magnitudes of these gender differences were around 5% of baseline forces, roughly 10 to 15 Newtons. The existence of muscle fatigue is often defined through force production, whether directly or indirectly assessed. A result of which, is that when there are conflicting measures indicating whether fatigue has or has not happened, investigators often rely on internal or external force measurements to sort out discrepancies (Lind, 1959; Petrofsky, 1979; Miller et al., 1988; Baker et al., 1993; Kumar et al., 2000; Skof and Strojnik, 2006a, 2006b; Kimura et al., 2007). In this study, relying on force losses would indicate that males had greater levels of muscle fatigue than females. However, as indicated in Table 5.1, females had more measurable consequences of the fatigue protocol in both muscle activity and postural differences, which could imply that females were actually more fatigued than males. Also, subjective ratings of fatigue and decreases in mean power frequencies were not different between genders (Table 4.3). Prior work documenting force gains (potentiation) with a low intensity and long duration fatigue protocol further cloud the seemingly straight forward relationship between muscle fatigue and external force generation (Johnson et al., 2013).

Therefore, it is not clear as to whether one gender was more fatigued than the other; it may not even be appropriate to think of them as different intensities on the same scale. Since the other indicators of fatigue besides external force were similar between genders, it is likely that males and females were experiencing slight variations on the same type of fatigue. Although possible, gender differences in pain patterns during standing cannot be directly attributed to males and females experiencing differences in fatigue.

The large decreases in mean power frequency and smaller decreases in strength for both genders indicate that muscle fatigue was likely more related to the cellular mechanisms responsible for reduced action potential velocity – net potassium efflux and slowed calcium reuptake (Tupling, 2004; Allen et al., 2008). Other muscle fatigue characteristics that result in force decreases not related to decreases in EMG frequency content (e.g insufficient metabolic inputs, accumulation of inorganic phosphate), although not measured, did not appear to be as prominent based on the relatively small force losses compared to other hip abductor fatigue protocols (McMullen et al., 2011; Patrek et al., 2011). These differences in fatigue indicators could indicate a different mechanism of muscle fatigue in the current study compared to these other shorter duration fatigue protocols.

Contrary to gender findings where the stronger male participants had shorter endurance times than females, PDs had both shorter endurance times and a tendency to have less hip abductor strength than NPDs. Persons with chronic pain or whom experience pain from hypertonic saline injections have also been reported to produce less muscle force (Dedering et al., 2004) and have earlier onsets of muscle fatigue (Ciubotariu et al., 2004; Johanson et al., 2011). However PDs were not in pain during the fatigue protocol, it was only later during standing that pain was developed. As a result, it was not the presence of pain that limited PDs muscle force generating capacities or

times to fatigue. This provides further evidence that PDs are a subclinical pain population (Nelson-Wong and Callaghan, 2014), as PDs have fatiguing characteristics of those with chronic pain, only without having developed chronic pain. Alternatively, it may not be the pain that limits muscle force or increases fatigability in those with chronic pain, but rather those characteristics that that are related to poorer fatigue resistance could predispose a person to developing chronic pain (Biering-Sorensen, 1984; O'Sullivan et al., 2006). It is also feasible that a third variable, such as subclinical tissue damage, leaves persons with a greater susceptibility to developing both muscle fatigue (Venditti and Di Meo, 1997; Choi and Widrick, 2009) and chronic pain (Loeser and Melzack, 1999).

PDs were determined to have slower recovery from fatigue than NPDs based on the normalized force residual (Figure 4.6); participant-specific %Baseline force in the control session minus the fatigue session. Inspection of Figure 4.5 reveals the control session strength for PDs slightly increased with time into standing, while control session strength for NPDs remained constant over the two hours. If recovery from fatigue was defined as the time block when the force value in the fatigue session was no longer different from 100 %Baseline force in the fatigue session, it is likely that there would have been fewer if any fatigue recovery differences with respect to pain grouping. The main rationale for comparing the fatigue session ABCs to the control session ABCs was to account for participants with non-level ABC force values during the control session. Within both pain groups, individuals exhibited patterns of increasing, decreasing, or steady strengths during the control session, and it was deemed to be more appropriate to account for these natural strength changes (or lack of changes) over time than to assume a steady level of strength over a 2 hour standing exposure. Additionally, in PDs, the differences between the control and fatigue points as shown in Figure 4.5a in the 120 minute block was approximately 10

%Baseline, while the mean residual averaged across PDs was approximately 16 %Baseline. Accounting for these individual differences in control session strengths was able to better differentiate hip abductor strength recovery patterns between PDs and NPDs.

PDs having shorter times to fatigue could relate to the ability of the active hip abduction test to identify PDs (Nelson-Wong et al., 2009). Since poorer movement quality in side-lying hip abduction would result in both a higher score on the active hip abduction test and a shorter time to fatigue, the two activities may be assessing similar underlying features. The rationale behind the success of the hip abduction test is that those who develop pain while standing are unable to effectively stabilize their trunk, whether during the test or in standing (Nelson-Wong et al., 2009). Thus there is a compensation in the hip abductors while standing so that they are activated bilaterally to assist the oblique and paraspinal muscles as stabilizers (Nelson-Wong et al., 2008; Nelson-Wong and Callaghan, 2010b), rather than sequentially activated to facilitate weight transfer between the limbs (Winter et al., 1996). This can also be seen in PDs lack of motion early into standing (Gallagher, 2014). The differences in endurance times could then be due to an inability to adequately utilize trunk stabilizers during the fatigue protocol, and require additional muscles to perform the requisite hip abduction. PDs recruiting greater abdominal musculature than NPDs in response to suddenly applied loads (Gregory et al., 2008a), and female PDs having greater total muscle activity at the time of peak GMD activity from this study are additional indicators of this insufficiency.

Initially, termination of the fatigue protocol was based only the leg raising height and timing criteria (Table 3.5 in Methods, Section 3.4.2). However, pilot testing revealed that participants began showing the same compensations that were deemed as “poor movements” in the active hip abduction test during the fatiguing exercise (Table 3.5 in Methods, Section 3.4.1).

The compensations in Table 3.5 under the "Lack of control of movement in the sagittal plane" and "Lack of control … frontal plane" headings, were then added in as criteria for an unsuccessful duty cycle, provided that the compensations were systematic. Since the active hip abduction test compensations are thought to be indicative of a lack of trunk stabilization in PDs (Nelson-Wong et al., 2009), PDs may have had shorter endurance times because of an inability to "abdominally brace" (McGill et al., 2003) as effectively or for as long as NPDs. If so, PDs may not have had more fatigable hip abductors, but rather more fatigable trunk musculature or poorer trunk muscular coordination than NPDs. Training abdominal muscle activity has been shown to be beneficial for PDs during standing (Nelson-Wong and Callaghan, 2010a), also giving evidence of pain group differences in trunk muscle use or capacity. Regardless, PDs and NPDs had similar force losses, reductions in hip abductor mean power frequencies bilaterally, ratings of perceived effort, and similar numbers of additional muscles affected by the fatigue protocol (Section 4.2.2). Although there do appear to be pain group differences in trunk muscle recruitment strategies or capacities, these only appeared to affect endurance times and not the level or quality of muscle fatigue resulting from the fatigue protocol.

Other studies employing hip abductor fatigue protocols have not reported whether the contralateral side or other non-synergistic muscles were affected to the same extent as in the current study. Studies on muscle fatigue are often performed on index finger abduction (Milner-Brown and Miller, 1986; Seki and Narusawa, 1998), thumb adduction (Miller et al., 1988), ankle dorsi flexion (Baker et al., 1993; Ciubotariu et al., 2007), or isolated muscle fibers (Balog and Fitts, 1996) to prevent such "contamination" from other muscles. A number of other studies using hip abductor fatiguing exercises have assumed unilateral fatigue without measuring EMG activity of any hip abductors (Salavati et al., 2007; Bellew et al., 2009; Vuillerme et al., 2009; Geiser et

al., 2010; Lee and Powers, 2013). Contrary to these assumptions, there was strong evidence of bilateral hip abductor fatigue from the current protocol though reductions in mean power frequencies from both GMDs and TFLs. From a mechanical perspective, an abduction moment generated at the contralateral (bottom) hip would be required to prevent the pelvis from laterally tilting upwards with the upper leg. As a result, it was noted that participants were visibly pushing into the examination table with their contralateral limb in conjunction with raising the ipsilateral limb during the fatigue protocol. This pushing down with the other limb to "scissor" the top limb up to the target height and prevent an upward pelvis tilt was likely the source of fatigue of the contralateral GMD and TFL. This tendency was noted upon its first occurrence (observed in every participant), and not reported in other studies employing similar side-lying leg raising protocols. An unintentional consequence of having the hip abductors affected bilaterally by the fatigue protocol may have rendered the need to assess leg dominance redundant for the purposes of performing the exercise.

With regards to non-synergists, one study reported a side-lying, eccentric hip abduction fatigue protocol did not alter ipsilateral vastus lateralis activity (McMullen et al., 2011), but did not quantify EMG of the contralateral limb or any trunk musculature. Another study on hip abductor fatigue with EMG of multiple muscle sites found a side-lying, intermittent, isometric fatigue protocol may have also fatigued gluteus maximus and lumbar erector spinae EMG selectively in those with chronic low back pain (Sutherlin and Hart, 2015). It was thought that the additional muscle activity in that protocol reflected low back pain patients having inappropriate muscle recruitment for the task, or insufficient hip abduction strength compared to controls. However that study (Sutherlin and Hart, 2015) did not report EMG frequency content of those muscles, their findings were based off of changes in activation levels during their fatiguing

exercise with respect to their control group. The non-synergists found to be fatigued by this protocol were predominantly oblique muscles on the ipsilateral side of dominant limb (R-EXO and R-INO). Of the three left-leg dominant participants, only one of them had one additional muscle fatigued by the exercise, the muscle in question was their L-EXO. This suggests that while side-lying hip abduction requires stabilizing cocontraction of the trunk, there is greater demand on the ipsilateral, anterior musculature than either posterior or contralateral musculature.

Referring back to Section 2.7, muscle fatigue was employed to "perturb the natural pain developer response while standing", seen through differences in muscle activity and postural strategies. It was thought that the most effective perturbation would be observed by targeting a muscle group implicated in the pain development in standing, and have other muscular compensations derive from having only that muscle group fatigued. In practice, the fatigue protocol resulted in quantifiable fatigue in four to nine of the muscles measured, with inter-participant differences in affected muscles that were not systematic with respect to gender or pain groups. In spite of these differences in the number of muscles fatigued, there were consistent changes in muscular and postural responses during standing that were able to be associated with a reduction in PD's low back pain (Section 5.1). The one possible exception would be a gender difference in posterior pelvic tilt during standing that may have related to differences in force lost following the fatigue protocol. Also, fatigue effects were sustained sufficiently into the standing period as evidenced through ABC measures. NPDs showed signs of fatigue recovery as early as 5 minutes into standing their fatigue session with force residuals (control minus fatigue force) dropping below zero within 2 hours. Meanwhile, PDs did not show any recovery from fatigue during standing, as session differences in ABC forces increased with time into standing. Differences in fatigue recovery between pain groups could be related to some of the observed

differences in standing behaviours such as lateral centre of pressure movement or smaller cumulative times in R-TFL muscle activity. This fatigue protocol, using a 2:1 work/rest ratio and movement quality cessation criteria, was adequate for the purpose of this study, and can be utilized in future work where a longer recovery time is desired.

5.3 Why Was There Less Cocontraction with Fatigue?

Contrary to the current study (Figures 4.8 and 4.9; Section 4.3.1), other reports of muscle fatigue have almost universally reported increases in muscular cocontraction with muscle fatigue (Psek and Cafarelli, 1993; Granata et al., 2001; Chappell et al., 2005; Herrmann et al., 2006; Grondin and Potvin, 2009; Ortiz et al., 2010; Kellis et al., 2011; Cashaback and Cluff, 2015). The purpose of increasing cocontraction about a joint in response to fatigue has been theorized to stem from a need increase joint stability with muscle fatigue related to resulting force or stiffness losses (Granata et al., 2001; Chappell et al., 2005; Cashaback and Cluff, 2015). In all of these cases, the muscle pairs examined were muscles with opposing actions about a joint (e.g. flexor/extensor) that were specifically targeted for fatigue, in tasks that directly required the use of those muscles. The current study does not satisfy these criteria.

The cocontraction indices reported in this study were mostly computed on muscle pairs that a) run parallel but produce the same direction of moment about a joint (e.g. R-TES/L-TES), b) were on lateral edges of opposing limbs (R-GMD/L-GMD), or c) did not act about the same joint (R-LES/R-GMD). Since the majority of muscle pairings would not provide stiffness about a joint, increased cocontraction with fatigue would not be expected with these pairings. Since only two of ten pairs could be considered to have opposing actions about a joint (R-LES/R-EXO and L-LES/L-EXO), these pairs should be the only ones that would co-activate with the goal of increasing joint stiffness. However, the decreases in these cocontraction indices occurring with

fatigue contraindicate the need for these muscle pairs to act as stabilizers during prolonged standing. This could be because in the standing tasks performed here, the intervertebral joints were sufficiently stable through the levels of muscle activity required to keep the torso upright (Woodhull-McNeal, 1986; Snijders et al., 1995; O'Sullivan et al., 2002). The values of the trunk cocontraction indices were often much larger (3000 to 4000 %MVIC; Figure 4.9) than the other cocontraction indices (500 to 2000 %MVIC; e.g. Figure 4.8; text in Section 4.3.1), likely attributable to the tonic activity in those muscles were larger than the muscles in other pairings in standing (Woodhull-McNeal, 1986; Snijders et al., 1995). Alternatively, these erector spinae-oblique pairs may not share the "common drive" found in muscles about the knee that exhibit greater cocontraction with fatigue in attempts to increase joint stability (Psek and Cafarelli, 1993; Mullany et al., 2002).

The major agonists of prolonged standing, if such terminology is useful or correct, could be considered the erector spinae muscles: they are tasked with maintaining an upright posture while standing (Dolan et al., 1995). The muscle targeted for fatigue was not one of the erector spinae muscles, but rather the gluteus medius and tensor fascia latae. This dissonance between the joint about which the fatigue exercise occurred and the joints that the "active" musculature acts about could be another reason why cocontraction was found to decrease with fatigue rather than increase. Perhaps the current fatigue protocol resulted in an increased GMD-adductor magnus cocontraction index during standing, as cocontraction of those muscles would produce compression and stiffness about the hip joint. If the current study were to be repeated using erector spinae fatigue in place of hip abductor fatigue (as suggested in the Introduction), then there may be increases in cocontraction indices that better fit as agonist-antagonist pairs than those measured here such as lumbar erector spinae and rectus abdominus.

The goal of computing cocontraction indices in this work was not to determine whether joints would be considered stable while standing, but rather 1) to quantify gross trunk and hip rigidity, 2) to get a sense of muscles pairs that tended to activate concurrently, and 3) relate changes with fatigue to previously established pain group differences during standing (Nelson-Wong et al., 2008). Since these elevated cocontraction indices indicate greater pain development during standing (Nelson-Wong et al., 2008; Nelson-Wong and Callaghan, 2010b), it would be expected that a decrease in pain levels would be matched by decreases in cocontraction indices, as were observed here. Had the purpose of this study been to assess hip joint stability in response to a hip abduction fatigue protocol, muscle pairs whose cocontraction reflects increasing resistance to joint displacement would have been selected.

5.4 Limitations

This study had a large number of dependent variables (65 dependent variables with 8 to 30 repeated measures) without any statistical corrections beyond violations of sphericity. Therefore, the likelihood of encountering Type I errors was quite high. A Bonferroni correction was advised against due to the large number of repeated measures, however even if implemented, that would not have addressed the number of total measures. Consistency in findings, as well as changes linked to a specific theory were focused upon during the discussion to try and combat this scenario.

There are some inherent limitations in quantifying pain intensities. While visual analog scales have been previously validated for determining levels of perceived pain (Price et al., 1983), participants have unique perceptions of the relative intensity of their pain, with large inter-participant variability in these measures. There was also no indicator of the quality of pain experienced by participants, just the intensity. However, employing a pain/no pain threshold to form sub groups has been done previously (Nelson-Wong et al., 2008; Gallagher et al., 2011;

Marshall et al., 2011) and found to be reliable within a participant (Nelson-Wong and Callaghan, 2010a). As mentioned in Section 2.5, pain measures were surrogates for determining how participants stood; participants stood in a fashion where they developed back pain, or they stood such that did not develop back pain. Furthermore, pain was said to have decreased with fatigue in PDs by using within subject comparisons, eliminating potential errors from between-subject variability from this finding. Aside from determining gross pattern differences between male and female PDs, the between session comparisons were also the only findings with regards to pain scores that were not based on a pain/no pain threshold.

It is possible that some participants could have used their gluteal pain as an anchor for the intensity of their low back pain (Herr et al., 2004; Kemp et al., 2012). In the control session, pain steadily increases over time; participants have no knowledge of what a given point on the scale should feel like prior to standing other than the anchor descriptions at either end. It is possible that the fatigue protocol gave participants a third anchor, which for most participants, was positioned above the 10 mm threshold. There is a possibility that PDs could have experienced the same absolute level of low back pain between sessions, yet reported different values because of the presence of that third anchor provided by the pain of the fatiguing exercise (Herr et al., 2004). In other words, the intensity of their low back pain may have been lessened with fatigue simply because they were "recalibrated" to the pain of the fatigue protocol immediately prior to standing. Since pain reporting increases over time in prolonged standing, the time between peak pain reporting from standing and this initial exercise-related pain was roughly two hours. It is unlikely that participants would have been able to recall the precise location of their scores immediately following the fatigue protocol at the end of the standing protocol. Also, the type of pain resulting from intensive exercise is likely different than that developed during a prolonged standing

exposure, which may have made the two noxious experiences less relatable than if the quality of pain was similar.

Aerobic exercises have been found to be analgesic stimuli in healthy participants in response to chemically or thermally induced pain (Koltyn, 2000; Hoffman et al., 2004; Ellingson et al., 2014). This phenomenon, termed exercise induced hyposensitivity, is thought to derive from an increasing of pain thresholds around active musculature following extensive muscle use (Koltyn, 2000). It is possible that the fatigue protocol, just by nature of being an exercise, may have reduced pain reporting regardless of changes in muscular or postural responses while standing. This was unlikely because the only session effects on gluteal pain reporting, the site of greatest potential pain reductions based on the fatiguing exercise (Koltyn, 2000), were increases rather than expected decreases with fatigue had exercise induced hyposensitivity been a factor. Also, the changes in pain sensitivity appear to be limited to the first 10 minutes following exercise (Hoffman et al., 2004), while the largest differences in low back pain development occurred after 2 hours of standing. While there may have been changes in sensitivity contributing to reductions in low back pain with fatigue, it is likely that changes in muscle activity patterns and postural compensations had a greater influence on low back pain reductions due to similar gluteal pain reporting between sessions from both pain groups and the timing of low back pain differences between sessions.

The fatigue protocol was administered while participants were encumbered with instrumentation required for the standing protocol, and may have affected performance during the fatiguing exercise. This was necessary to minimize the time between the end of the fatigue protocol and the initiation of standing, as the time required for instrumentation was around 90 minutes. If participant instrumentation occurred after the fatigue protocol, many findings relevant to

differences in low back pain development during standing would not have been observed such as the reductions in cocontraction indices or increases in centre of pressure fidgets. Participants were motivated by the experimenter during the fatigue protocol, and while some participants did have hesitations during the procedural explanation, participants were often too focused on the exercise at hand to notice the instrumentation by the end of the fatigue protocol. Wires were checked prior to recordings to ensure they would not impede participant range of motion during the study. The total mass of the instrumentation was 2.07 kg, almost half of which (0.9 kg) consisted of battery packs for the EMG recording system which were resting beside the participant during the fatiguing exercise. The mass of the objects on the leg being abducted during the fatigue protocol and the ABCs was 0.24 kg, most of which (0.18 kg) was proximal to the knee. Other fatigue protocols targeting the hip abductors have used additional masses of one to three kilograms placed around the ankles to increase the relative workload (Bellew et al., 2009; Vuillerme et al., 2009). The much smaller mass of current instrumentation was likely negligible due to the limited moment produced about the hip relative to the mass of the participant's leg. If there were any effects, they were likely similar across participants.

The fatigue protocol was only administered to most participants once, therefore it is not known how reliable the cessation criteria were in determining endurance times. Five participants, three of whom were included in the data presented here, performed the fatigue protocol on two separate occasions. Of those three included in the current dataset (EG6, RT2 and OK3), only one participant performed the protocol to completion both times. That participant (EG6), had endurance times of 27.5 and 30.0 minutes for their first and second attempts. Only the first attempt was used in this study as the second attempt was part of a separate pilot project. Participants RT2 and OK3 had their first attempt of the fatigue protocol interrupted prior to completion by a

technical error and a fire alarm respectively. In both instances, those participants had endurance times during the second attempted session that exceeded their truncated initial attempts by at least 5 minutes. The second attempts of their fatigue protocols, collected at least a week after the initial attempt, were included for analysis. The two participants not included in this dataset who performed the fatigue protocol twice, as well as three others who performed the fatigue protocol once were used in refining cessation criteria and as training for the experimenter in judging duty cycles as successful or unsuccessful.

For the ABCs, hip abductor strength was assessed while standing using isometric exertions, while the fatiguing exercise was a dynamic task performed while side-lying. These differences in contraction type and participant orientation may have resulted in the ABCs inaccurately quantifying fatigue. However, the current setup was preferred to a fatigue assessment simulating the fatiguing exercise for a number of reasons. Firstly, the continuous standing would have been interrupted at each ABC by moving from upright standing to a side-lying posture, which would alter the pain developed during standing (Gallagher et al., 2014). Secondly, the time required to ensure participant setup in a device capable of assessing dynamic strength would have added a substantial amount of time to the standing protocol. This could be in the form of moving the participant into the device, which would be unwanted due to the interruption of standing, or by moving the device to the participant at each time point, as the bulk of these devices would interfere with force plate and kinematic measures if left in place while participants stood. Also, the fatiguing exercise while timed, was not isokinetic like dynamic strength assessment devices are, and any conclusions drawn from an isokinetic, dynamic device would face similar limitations with regards to contraction type as faced by the current setup. Using a brief isometric exertion to assess fatigue

while standing allowed for a testing procedure that minimally disrupted the standing protocol while providing a suitably accurate representation of hip abduction strength.

The distance of the cuff from the hip joint centre of rotation was kept constant within a session and based off the same bony landmarks for each participant, but was not quantified. Had that moment arm been known, strength could have been expressed in terms of moments rather than forces, to account for differences in leg lengths. The cuff height was marked on each participant upon completing the baseline ABC and was checked to have remained at the original height prior to each subsequent ABC. Also, all between session comparisons were normalized to the baseline ABC in the respective session in order to account for any variation in cuff height, absolute strength, or motor recruitment strategy between sessions.

Participants were instructed to use the same gross motor strategy while performing each ABC throughout the collection, and were given as much time as was needed to devise a method that they could reproduce comfortably. However, participants may have not recalled their original technique upon returning for their second visit, or may have made compensations over the course of the two hour standing protocol. An ABC was repeated if a compensation was detected either visually by the experimenter or by the participant based on proprioceptive feedback. However there was likely variability between participants as to what they deemed to be a compensation; when there was any uncertainty, the ABC was repeated.

Participant productivity in the two tasks was not monitored. In typing and manual materials handling, working at a relatively faster pace has been found to increase local discomfort in the hands, however the discomfort in the low back and gluteal regions were more influenced by posture than productivity (Ulin et al., 1993; Gerard et al., 2002). It is unlikely that productivity would have influenced postural or pain related variables. Since the muscles measured for EMG

were not related to the tasks performed, but rather the control of standing, any task or productivity effects should have minimal influence on EMG variables. The order of tasks was randomized for each participant and was reversed for the participants second session. As a result, any potential task effects should have been washed out by the randomization procedure.

Section 6: CONCLUSIONS & FUTURE DIRECTIONS

Conclusions that can be drawn from this work are follows:

1. ***Hip abductor muscle activity does play a causative role, but is likely not solely responsible for the low back pain developed in prolonged standing.*** Evidence for this conclusion is as follows. Hip abductor muscle fatigue reduced low back pain development, but did not remove pain from PDs. Gender differences in pain reporting were primarily linked to pelvic postural differences, indicating that sagittal plane pelvic tilt can affect pain development during standing. Increases in movement early into prolonged standing exposures also appears to be beneficial for the reduction of low back pain developed from standing.
2. ***Hip abductor muscle fatigue does not simulate pain in NPDs, but rather simulates the pain-free response in PDs.*** NPDs did not develop pain over the course of the fatigue session and showed minimal changes with fatigue corresponding to PD standing responses. PDs were more influenced by the fatigue protocol in measures that were beneficial or neutral to reductions in low back pain. Fatigue of a different muscle group may simulate pain responses or result in pain development in NPDs.
3. ***Hip abductor fatiguing characteristics may be related to the development of chronic low back pain.*** PDs, while not experiencing chronic pain, exhibited fatiguing characteristics of those with chronic low back pain. This suggests that either PDs are more likely to develop low back pain in the future or that the same characteristics that limit fatigue resistance are also involved in the development of chronic low back pain.
4. ***Prolonged standing is not an inherently fatiguing task for PDs or NPDs, but those who develop low back pain do not recover from hip abductor fatigue while standing.*** ABC measures during the control session indicated that neither group lost hip abductor strength

during the control session. Both groups had similar force losses immediately following the fatigue protocol, NPDs were able to return to control session values by the end of the standing protocol, but PDs did not. A lateral shift in centre of pressure and increased right tensor fascia late activity persisting throughout the fatigue session also indicate that PDs were still exhibiting hip abductor fatigue upon standing for two hours.

5. ***Introducing rest breaks into a fatiguing exercise not only prolonged the endurance and recovery times, but may have altered the mechanism of muscle fatigue.*** The fatigue indicators associated with the longer endurance times in this study reflect that reductions in action potential conduction velocity were the main drivers behind the current fatigue protocol. Shorter duration fatigue protocols resulted in larger relative force decreases with minimal decreases in EMG frequency content, suggesting that non-electrical factors relating to muscle force generation were the main contributors to muscle fatigue.
6. ***Side-lying leg raising exercises can affect hip abductor musculature bilaterally, and can also fatigue trunk stabilizing musculature.*** There were similar mean power frequency decreases in the dominant and non-dominant limbs for all participants, regardless of leg dominance. The upper limb works to elevate the limb directly, while the lower limb pushes down into the surface being laid upon to aid in leg abduction. The movement compensations during the fatigue protocol matching those from the active hip abduction test and decreases in mean power frequencies in ipsilateral oblique muscles indicate that side-lying hip abduction exercises can fatigue trunk stabilization muscles in addition to the hip abductors.

It would be incorrect to assume on the basis of this work that it is beneficial for persons to begin prolonged standing exposures in a fatigued state to reduce low back pain development. What

should be taken away is that the postural and muscular compensations that were a consequence of this fatigue protocol - increased movement within the first hour of standing - were responsible for the observed low back pain reductions rather than fatigue itself. Therefore, a preventative approach would involve frequent movements during standing, especially within the first hour of an exposure.

Future directions to be considered:

- Were gender differences with fatigue in PDs truly related to gender, or indicative of another factor that was related to gender differences such as hip abductor strength, leg length or force lost from fatigue?
- Do those who develop pain during standing have similar fatigue recovery characteristics to those who do not develop pain from standing when the recovery task is not standing (sitting, lying supine or prone)?
- Is there a common thread linking fatigability and chronic pain development? Does one directly influence the other or does a third variable cause both?
- Does introducing erector spinae fatigue change pain reporting or fatigue recovery characteristics in those who do and do not develop pain during standing?
- Are those who are more sensitive to pain changes with fatigue during standing at a lower risk of injury based on muscular or postural differences during prolonged standing?

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Appendix A: List of Significant Findings

These tables document which variables were found to be significantly different ($p < 0.05$) between one or more levels of independent variables (Session, Time, Gender and Pain Group). Interaction effects or main effects are indicated by the column labeled "Group", indicating which sub-group of participants exhibited the listed changes. Main effects are indicated by "All". Probabilities are given as a range when multiple time points are indicated unless the lower bounds were below 0.0001, in which case the maximum bound is given.

Effects of the Fatigue Intervention

Directionality goes from the control to the fatigue session. For example a decrease indicates the values was greater during the control session (it decreased from control to fatigue).

EMG Changes with Fatigue

Cocontraction Indices

Finding	Time Points	Group	Probability
Decrease in R-LES/R-EXO	15 Minutes	All	0.0421
Decrease in L-LES/L-EXO	45 Minutes	All	0.0390
Decrease in R-LES/L-LES	30 to 60 Minutes	All	0.0044 to 0.0239
Decrease in R-GMD/R-LES	75 to 120 Minutes	Male PDs	0.0105 to 0.0479

Gaps Analysis

Finding	Measure	Time Points	Group	Probability
Increase in R-TES	Cumulative Time	30 Minutes	All	0.0165
Decrease in R-TFL	Cumulative Time	15 to 120 Minutes	Female PDs	< 0.0038
Decrease in L-TES	Cumulative Time	120 Minutes	All	0.0239

Force Plate Measure Changes with Fatigue

Finding	Foot	Time Points	Group	Probability
More Lateral CoP Mean	Left	120 Minutes	PDs	0.0356
More Lateral CoP Mean	Right	45, 105, and 120 Minutes	PDs	0.0024 to 0.0438

Kinematic Changes with Fatigue

Angle	Direction of Finding	Time Points	Group	Probability
Pelvic Tilt Mean	Anterior	105 to 120 Minutes	All	0.0098 to 0.0125
Pelvic Tilt Mean	Anterior	45, 75 to 120 Minutes	Male PDs	0.0240 to 0.0460
Pelvic Tilt Mean	Right Lateral	15 to 120 Minutes	Female PDs	0.0018 to 0.0482
Pelvic Tilt Mean	Right Lateral	120 Minutes	Male NPDs	0.0486
Lumbar Angle	Extension	90 to 120 Minutes	Male PDs	0.0227 to 0.0391
Lumbar Angle	Flexion	30 to 90 Minutes	Female PDs	0.0134 to 0.0397
Right Hip Angle	Flexion	75 to 120 Minutes	Male PDs	0.0292 to 0.0428
Right Hip Angle	Extension	75 Minutes	Female PDs	0.0389
Left Hip Angle	Flexion	30 to 120 Minutes	Male PDs	0.0053 to 0.0438
Left Hip Angle	Extension	30 to 75 Minutes	Female PDs	0.0258 to 0.0451

Effects of Time

Each table contains a header labelled “What times are different”. Numbers separated by a vertical bar within this column are different from each other. Directionality goes from the times (minutes into standing) on the left of the bar towards the times on the right of the bar. For example, if a variable at 15 minutes was larger than that same variable at 120 minutes, the finding would “Decrease” from “15 | 120”.

EMG Changes with Time

Cocontraction Indices

Finding	What Times are Different	Group	Probability
Increase in R-GMD/L-GMD	15 and 30 | 90 to 120	PDs	0.0027 to 0.0289
Decrease in L-LES/L-EXO	45 | 120	All	0.0390
Increase in R-TFL/L-TFL	15 and 30 | 120	All	0.0086 to 0.0402
Increase in R-TES/L-TES	75 | 120	PDs	0.0070
Decrease in R-TES/L-TES	75 | 120	NPDs	0.0471
Decrease in L-GMD/L-LES	30 to 60 | 120	Control	0.0161 to 0.0483
Increase in R-GMD/L-LES	15 | 120	All	0.0072
Decrease in L-GMD/R-LES	15 to 90 | 120	NPDs	0.0041 to 0.0425
Decrease in L-GMD/R-LES	15 to 60 | 75 to 120	Control	< 0.0071

Gaps Analysis

Finding	Measure	What Times are Different	Group	Probability
Increase in R-TFL	Number of Gaps	15 to 75 | 120	All	0.0002 to 0.0389
Decrease in R-EXO	Number of Gaps	30 and 60 | 120	PD	0.0272 to 0.0403
Decrease in R-INO	Number of Gaps	45 to 75 | 120	PD Control	0.0031 to 0.0460
Increase in L-TFL	Number of Gaps	15 to 75 and 120 | 90 and 105	Female PDs	0.0032 to 0.0403
Increase in L-INO	Number of Gaps	30 and 60 | 120	All	0.0116 to 0.0464
Decrease in R-TFL	Gap Length	15 | 120	All	0.0492
Increase in R-INO	Gap Length	15 | 120	Female Control	0.0154
Increase in R-TES	Cumulative Time	30 and 60 | 120	Control	0.0082 to 0.0451
Decrease in R-TES	Cumulative Time	30 and 60 | 120	Female Fatigue	0.0257 to 0.0481
Decrease in R-LES	Cumulative Time	15 to 45 | 120	Female PD Fatigue	0.0067 to 0.0342
Decrease in R-GMD	Cumulative Time	15 to 30 | 120	All	0.0203 to 0.0429
Decrease in R-TFL	Cumulative Time	15, 45 and 60 | 120	PD Control	0.0046 to 0.0225
Decrease in R-INO	Cumulative Time	15 to 75 | 90 to 120	PDs	0.0003 to 0.0206
Increase in L-LES	Cumulative Time	30 | 120	Control	0.0238
Decrease in L-LES	Cumulative Time	105 | 120	Control	0.0325
Increase in L-GMD	Cumulative Time	15 to 30 | 120	Female NPDs	0.0176 to 0.0260
Increase in L-TFL	Cumulative Time	15 to 60 | 120	All	0.0038 to 0.0500
Decrease in L-EXO	Cumulative Time	15 | 120	All	0.0201
Increase in L-INO	Cumulative Time	15 to 60 | 120	All	0.0116 to 0.0493

Force Plate Measure Changes with Time

Finding	Foot	What Times are Different	Group	Probability
Decrease Shift Counts	Both	60 | 120	All	0.0340
Decrease Fidget Counts	Both	30 | 120	NPDs	0.0278
Decrease Fidget Sizes	Both	60 | 15 to 45, 75 to 90, 120	PDs	0.0002 to 0.0351
Increase Body Weight Transfers	Both	45 to 75 | 105 to 120	Female Fatigue	0.0034 to 0.0361
Increase Body Weight Transfers	Both	15 to 30 | 45 to 120	All	< 0.0179
More Posterior CoP Mean	Left	15 to 60 | 120	PDs	0.0036 to 0.0443
More Posterior CoP Mean	Right	15 | 120	All	0.0001
More Lateral CoP Mean	Left	15 | 120	NPDs	0.0435
More Lateral CoP Mean	Right	45 | 120	PDs	0.0351
Increase in AP CoP SD	Left	15 to 30, 75 | 120	All	< 0.0345
Increase in AP CoP SD	Right	15 to 75 | 120	All	< 0.0046
Increase in ML CoP SD	Left	15 to 30 | 45 to 120	Females	< 0.0272
Increase in ML CoP SD	Right	15 | 120	All	0.0036

Kinematic Changes with Time

Angle	Direction of Finding	What Times are Different	Group	Probability
Pelvic Tilt Mean	Posterior	15 to 30 | 120	All	0.0003 to 0.0057
Pelvic Tilt Mean	Posterior	30 | 75 to 120	PDs	0.0165 to 0.0489
Pelvic Tilt Mean	Posterior	75 to 90 | 120	NPDs	0.0117 to 0.0476
Pelvic Tilt SD	Lateral (Increase)	30 to 45 | 120	All	< 0.0009
Lumbar Angle*	Flexion	15 to 105 | 30 to 120	All	< 0.0482
Right Hip Angle	Abduction	60 | 120	NPDs	0.0210
Right Hip Angle	Extension	30 to 75 | 120	NPDs	0.0016 to 0.0132
Left Hip Angle	Internal Rotation	15 | 120	All	0.0438
Left Hip Angle	Extension	30, 45, 75, 105 | 120	NPDs	0.0096 to 0.0389
Left Hip Angle	Extension	30 and 60 | 120	Control	0.0213 to 0.0328

*See Table 4.12 for more details

Effects of Gender

Directionality is indicated with the column labelled "Finding": Males = M; Females = F.

EMG Gender Differences

Cocontraction Indices

Muscle Pairing	Finding	Time Points	Group	Probability
R-GMD/L-GMD	M > F	60 to 75 Minutes	All	0.0137 to 0.0256
R-LES/R-EXO	M > F	15 to 75 Minutes	All	0.0148 to 0.0314
R-LES/R-EXO	M > F	45 to 105 Minutes	Control	0.0046 to 0.0238

Gaps Analysis

Muscle	Finding	Measure	Time Points	Group	Probability
R-TES	F > M	Gap Length	15 to 30 Minutes	All	0.0043 to 0.0201
R-EXO	M > F	Gap Length	90 to 105 Minutes	All	0.0043 to 0.0372
L-GMD	M > F	Gap Length	90 Minutes	All	0.0187
L-TFL	M > F	Gap Length	75 Minutes	All	0.0471
R-TES	F > M	Cumulative Time	15 Minutes	PDs	0.0489
R-GMD	F > M	Cumulative Time	30 and 90 Minutes	All	0.0203 to 0.0429

Force Plate Measure Gender Differences

Measure	Finding	Foot	Time Points	Group	Probability
Shift Counts	F > M	Both	30, 45 and 90 Minutes	All	0.0027 to 0.0392
A/P CoP Mean	M more anterior than F	Right	30 and 75 to 105 Minutes	NPDs	0.0074 to 0.0471
A/P CoP SD	F > M	Right	30, 75, 90 and 120 Minutes	PDs	0.0064 to 0.0255
M/L CoP SD	F > M	Left	30 to 120 Minutes	PDs	< 0.0311

Kinematic Gender Differences

Angle	Finding and Direction	Time Points	Groups	Probability
Pelvic Tilt Mean	M more right tilted than F	15 to 120 Minutes	NPDs	0.0003 to 0.0255
Right Hip Angle	M more abducted than F	30, 60 to 120 Minutes	PDs	0.0096 to 0.0109
Right Hip Angle	F more abducted than M	30 to 120 Minutes	NPDs	0.0018 to 0.0137
Left Hip Angle	M more externally rotated than F	30 Minutes	All	0.0145

Effects of Pain Group

Like Gender, directionality is indicated within the "Finding" column: Pain developer = PD, non-pain developer = NPD.

EMG Pain Group Differences

Cocontraction Indices

Muscle Pairing	Finding	Time Points	Group	Probability
R-GMD/L-GMD	PD > NPD	60, 90 to 120 Minutes	All	0.0125 to 0.0495
R-TES/L-TES	NPD > PD	90 and 120 Minutes	Males	0.0260 to 0.0321
R-LES/L-LES	PD > NPD	75 to 120 Minutes	Females	0.0023 to 0.0496
R-GMD/R-LES*	PD > NPD	120 Minutes	All	0.0347
R-GMD/R-LES*	PD > NPD	30, 60 to 120 Minutes	Male Control	0.0038 to 0.0359
R-GMD/L-LES	PD > NPD	45 to 60 Minutes	All	0.0277 to 0.0431
L-GMD/R-LES	NPD > PD	15 Minutes	All	0.0166

*These findings were no longer different upon removing FX9's data

Gaps Analysis

Muscle	Finding	Measure	Time Points	Group	Probability
R-LES	NPD > PD	Number of Gaps	120 Minutes	All	0.0139
R-TFL	NPD > PD	Number of Gaps	45 to 75 Minutes	Females	0.0114 to 0.0277
L-GMD	NPD > PD	Number of Gaps	75 Minutes	All	0.0380
L-TFL	NPD > PD	Number of Gaps	30 to 45 Minutes	All	0.0163 to 0.0439
L-EXO	PD > NPD	Cumulative Time	15, 30 and 75 Minutes	All	0.0096 to 0.0482

Force Plate Pain Group Differences

Measure	Finding	Foot	Time Points	Group	Probability
Fidget Size	PD > NPD	Both	15 to 45 Minutes	Males	0.0011 to 0.0242
Fidget Size	NPD > PD	Both	60 Minutes	Males	0.0152
Fidget Size	PD > NPD	Both	45 Minutes	Control	0.0006
Fidget Size	NPD > PD	Both	105 Minutes	Control	0.0320
A/P CoP Mean	NPD > PD	Left	105 Minutes	All	0.0488
A/P CoP Mean	PD > NPD	Right	105 Minutes	Females	0.0246
M/L CoP Mean	PD more medial than NPD	Right	15 to 45 and 120 Minutes	Control	0.0013 to 0.0103
M/L CoP SD	NPD > PD	Left	30 Minutes	Males	0.0282

Kinematic Pain Group Differences

Angle	Finding and Direction	Time Points	Groups	Probability
Pelvic Tilt Mean	NPD more right tilted than PD	30 to 120 Minutes	Male Fatigue	0.0099 to 0.0448
Pelvic Tilt Mean	PD more right tilted than NPD	15 to 120 Minutes	Female Fatigue	0.0004 to 0.0194
Pelvic Tilt Mean	PD more posteriorly tilted than NPD	30 to 90 Minutes	Female Fatigue	0.0042 to 0.0365
Lumbar Angle	NPD more extended than PD	30 to 90 Minutes	Female Fatigue	0.0134 to 0.0397
Right Hip Angle	PD more abducted then NPD	30, 45, and 75 to 120 Minutes	Females	0.0007 to 0.0091
Right Hip Angle	NPD more abducted than PD	30 to 75 and 120 Minutes	Males	0.0005 to 0.0481
Left Hip Angle	NPD more abducted than PD	15 to 45 and 75 to 120 Minutes	Females	0.0041 to 0.0451

Appendix B: EMG Cocontraction Patterns of Pain and Non-Pain Developers

Embedded within the EMG cocontraction index calculation (Equation 3.1, repeated below), there are two terms that are combined number reported. The first term is a "common area" term, the magnitude of the smaller of the two muscle activations divided by the larger one. This term becomes larger if the two activation levels are closer to each other and can range from 0 to 1. The second term is a "scaling" term, the sum of the two muscle activations. This term increases linearly with the absolute magnitude of muscle activity and ranges from 0 to 100. As a result, there are two methods to obtain a larger cocontraction index, firstly through matching levels of muscle activity, and secondly by increasing the total amount of muscle activity.

$$CCI = \sum_{i=1}^{N} \left[\left(\frac{EMG_{low\,(i)}}{EMG_{high(i)}} \right) \times \left(EMG_{high(i)} + EMG_{low\,(i)} \right) \right]$$

The original purpose in using this equation was to separate out persons who had similarly sized "common area" terms based on having a larger "scaling" term, as other cocontraction index calculations have avoided the inclusion of such an activation scaling term. In the work of Nelson-Wong and colleagues (2008, 2010a,b,c), PDs were identified based on having non-zero activity levels in gluteus medius bilaterally, while NPDs would frequently have at least one side of gluteus medius activity at or around rest. Sample time-varying plots of muscle activity for a gluteal (R-GMD/L-GMD) and trunk (R-LES/R-EXO and L-LES/L-EXO) cocontraction index calculation are provided here (Figures B.1 to B.4) to elucidate how different factors in the time-varying EMG signal contribute to PDs having larger cocontraction indices than NPDs.

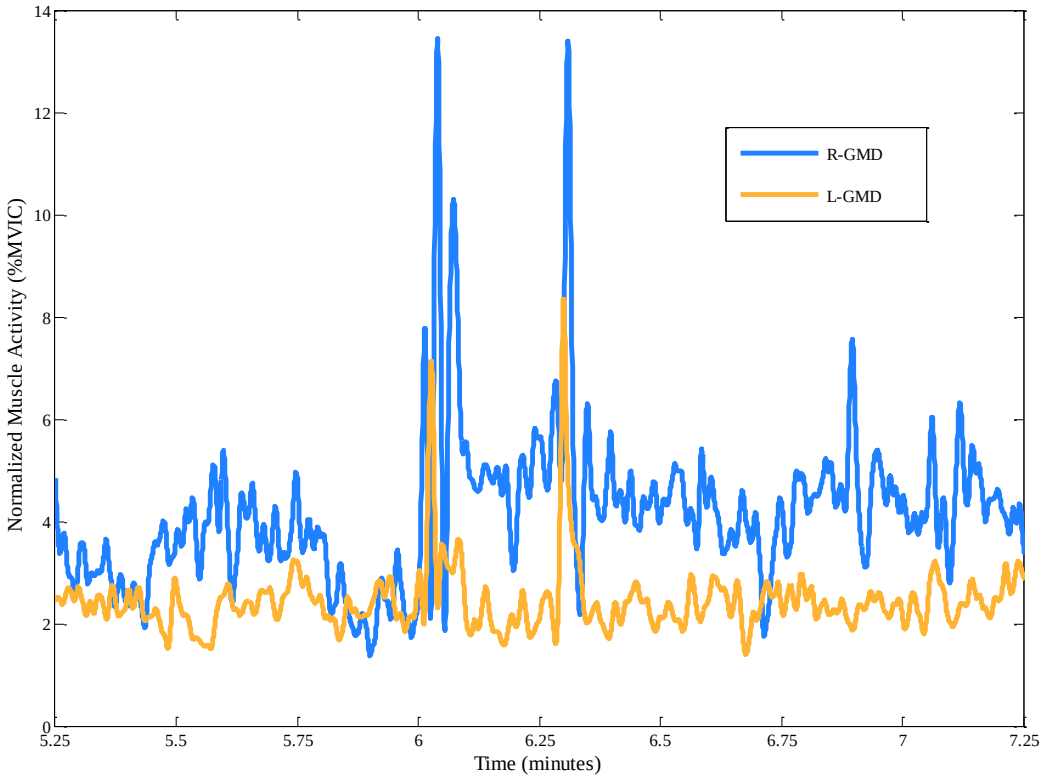

Figure B.1: PD Bilateral Gluteal Cocontraction Response. Data is taken from MP4 Control and the computed CCI from this window was 4214.5 %MVIC. Both gluteus medius' have non-zero levels of activity, and brief bursts of activity from both muscles temporally align with each other. This increases both the common area and scaling terms of the CCI equation, resulting in the large output.

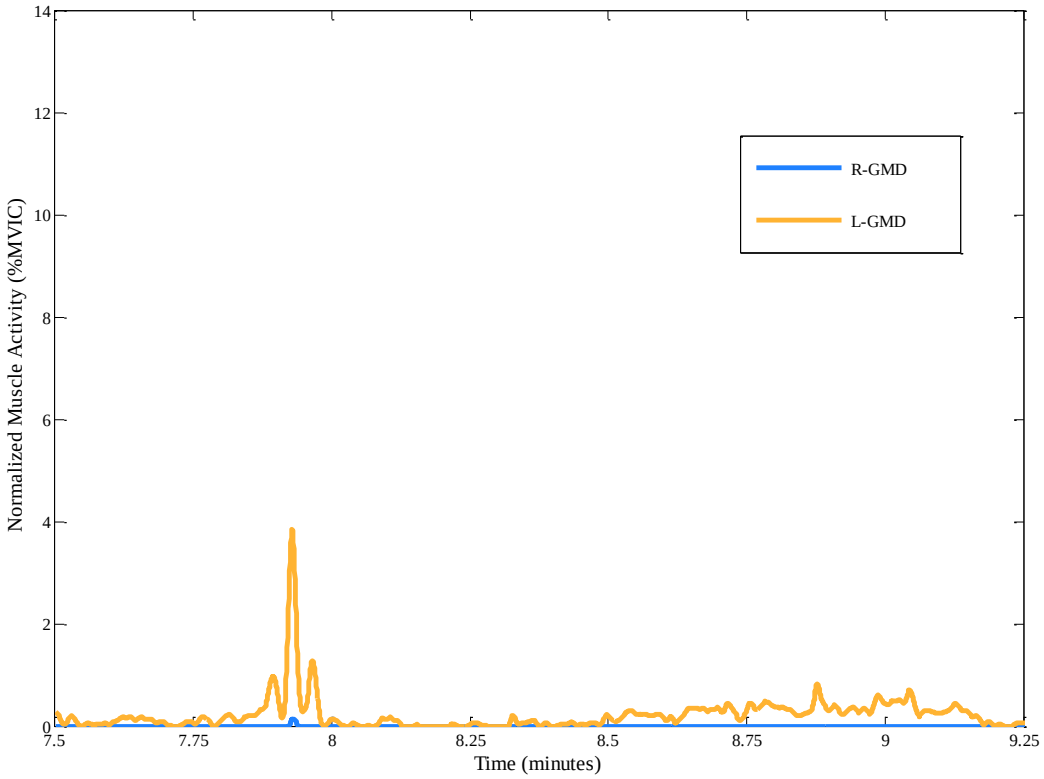

Figure B.2: NPD Bilateral Gluteal Cocontraction Response. Data is taken from VG4 Control and resulted in a CCI of 407.9 %MVIC. Both muscles are at lower activity levels during standing than in the PD example in Figure B.1. Also, when one side exhibits a small burst of activity, the other side exhibits myoelectric silence.

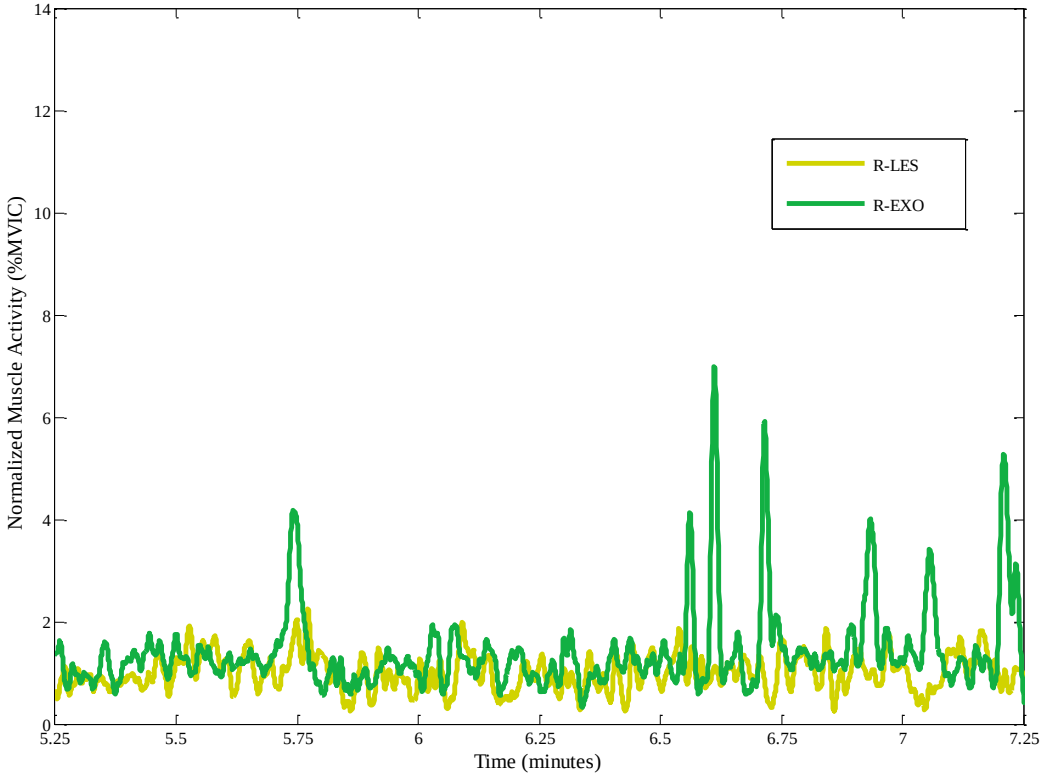

Figure B.3: PD Ipsilateral Trunk Cocontraction Response. Data taken from MP4 Control and resulted in a CCI of 2445.8 %MVIC. Like the gluteal cocontraction response (Figure B.1), both muscles show constant, non-zero activity levels during standing (higher common area). However unlike the gluteal response, bursting in tandem is not a common occurrence.

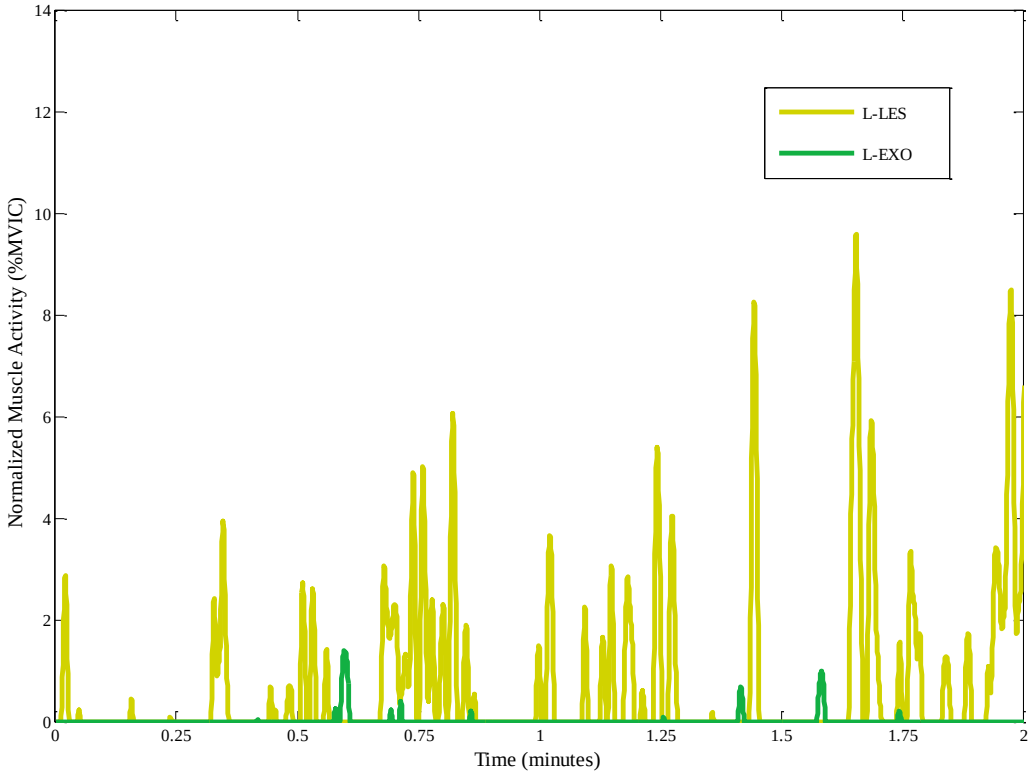

Figure B.4: NPD Ipsilateral Trunk Cocontraction Response. Data taken from VG4 Control and resulted in a CCI of 127.8 %MVIC. Although muscle activity levels in this example are at higher levels than the PD example, there is almost no common overlap between tracings, resulting in a common area term of zero for these instances.