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Danielle Marie Stewart

Under the supervision of

Dr. Diane Gregory

Submitted to the Department of Kinesiology and Physical Education, in fulfillment of the requirements for the degree of Master of Science in Kinesiology

Wilfrid Laurier University

June 2015

Abstract

Introduction: The flexion-relaxation phenomenon (FRP) represents myoelectric silencing of the erector spinae musculature during peak trunk flexion. In individuals with chronic low back pain (LBP), the FRP response is absent. Acute LBP development is commonly induced by prolonged static postures such as standing and tends to be temporary in nature. If FRP is present in people who develop acute LBP, it is plausible that intermittent peak trunk flexion may reduce discomfort development by inducing periods of muscle rest experienced during FRP. The purpose of this study is two-fold: 1) to determine if transient trunk flexion mitigates pain development induced by prolonged standing; and 2) to determine if FRP occurs and changes over time due to prolonged standing.

Methods: Sixteen participants free of LBP in the previous 12 months were recruited to complete two separate periods (one week apart; randomized) of a 2-hour standing protocol. On one of the days participants stood statically for the full 2 hours (control day); on the other day participants were instructed to bend forward to full spine flexion for 5 seconds every 15 minutes to illicit FRP (flexion day). During the full spine flexion periods, erector spinae and gluteus medius muscle activation was recorded using surface electromyography (EMG). Lumbar flexion angles were recorded to determine the degree of flexion when FRP occurred. During flexion days, participants reported low back discomfort prior to and following each flexion trial using a 100mm visual analogue scale (VAS). On the control day, participants reported discomfort every 15 minutes, again using the VAS. Based on control day VAS scores, a cut-off of 8mm of discomfort was used to categorize participants as low back discomfort developers (LBD developers) or non-low back discomfort developers (non-LBD developers). These two groups

were then statistically compared using two-way mixed model ANOVAs to determine any differences in the FRP response between discomfort and condition.

Results: Twelve of the sixteen participants were classified as LBD developers (16.19mm VAS (SE 1.78)) and four participants were considered non-LBD developers (1.64mm VAS (SE 0.4)). FRP was present in all participants regardless of discomfort development. No notable differences were seen in the FPR response over time, though LBD developers experienced significantly greater muscle activation during FRP than non-LBD developers.

The transient flexion trials significantly decreased the level of low back discomfort (LBD) after 75 minutes of standing (p=0.01) when VAS scores during control days were compared to those recorded immediately after flexion; LBD decreased by approximately 10mm of discomfort on the VAS after flexion. Further, in an exit survey, 81% of participants reported that flexing forward was beneficial and helped to alleviate any discomfort development that occurred during the two hour standing protocol.

An additional and unexpected finding of this study was the presence of FRP not only in the erector spinae but also in the gluteus medius.

Discussion and Conclusion: People who developed acute LBD during prolonged standing continued to experience erector spinae muscle FRP. The FRP response in acute LBD developers seems to more closely resemble healthy populations than chronic LBP populations, for which it has been shown to be absent. However, the increased muscle activation during FRP seen in LBD developers suggests that they may be considered a sub-clinical population prone to developing chronic LBP. An interesting and novel finding of this work is the occurrence of the FRP in the gluteus medius muscle. Previous research has identified that co- activation of the gluteus medius is likely a predisposing factor to LBP development. It

is possible that during the flexion trials, FRP of the erector spinae and gluteus medius muscle mitigated LBP development. This study has shown that transient trunk flexion significantly reduces LBD experienced during prolonged standing.

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To my parents Larry and Mary-Ann Stewart, I thank them for their unconditional love, support and patience. They have instilled in me a sense of humility and a passion for education, for that I am forever thankful. To my brothers and built-in best friends - I thank Lee Stewart for teaching me to never give up no matter how difficult life gets, and Todd Stewart for teaching me to always push my limits, seek out new adventures and new ways of thinking.

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person who has endured all of my long rants and supported me without question every step of the way. I would not have wanted to complete this degree without him by my side. I cannot wait to see what other adventures we take on together.

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List of Abbreviations

ANOVA Analysis of Variance

CoP Centre of Pressure

EMG Electromyography

EO External Oblique

FRP Flexion Relaxation Phenomenon

FRR Flexion Relaxation Ratio

GM Gluteus Medius

LBD Low Back Discomfort

LBP Low Back Pain

LBD developers Low Back Discomfort Developers

LES Lumbar Erector Spinae

MVC Maximum Voluntary Contraction

Non-LBD developers Non-Low Back Discomfort Developers

RPD Rating of Perceived Discomfort

ROM Range of Motion

SE Standard Error

VAS Visual Analog Scale

1.0 Introduction

Low back pain (LBP) is a common, chronic and recurrent condition that affects approximately eighty-five percent of Canadians (Cassidy, Cote, Carroll, & Kristman, 2005; Cassidy, Carroll & Cote, 1998) with the highest incidence of LBP occurring between the ages of 30-50 years old (Kopec, Sayre, & Esdaile, 2004; Waxman, Tennant, & Helliwell, 2000; Hoy, Brooks, Blyth, & Buchbinder, 2010). The specific cause of LBP is unclear in 85-90% of cases (Deyo & Weinstein, 2001; Diamond, & Borenstein, 2006; Ehrlich, 2003), though various non-neutral postures such as static flexion (Kumar, 2001), axial twisting (Shan et al., 2013) and exposure to whole body vibration (Hoogendoorn et al., 2000) have been shown to influence LBP. Repetitive motions (Sbriccoli et al., 2004) and cumulative loading (Seidler et al., 2001) also increase the chance of developing LBP and often lead to chronic pain. In today's society, sedentary lifestyles are becoming increasingly prevalent. LBP is prominent and strongly associated with static postures related to a sedentary lifestyle such as sitting (Janwantanakul, Pensri, Moolkay, & Jiamjarasrangsi, 2011; Beach, Parkinson, Stothart & Callaghan 2005; Howarth, Glisic, Lee, & Beach, 2013) and standing (Mohseni-Bandpei et al., 2011; Xu, Bach, & Orhede, 1997; Cook, Branch, Baranowiski, & Hutton, 1993; Zander, King, & Ezenwa, 2004).

Occupations such as assembly-line workers, bank tellers, grocery store cashiers, teachers, surgeons and casino dealers, along with various other professions, require workers to stand for extended periods of time, increasing the incidence of LBP. Despite minimal compressive loading of the low back during prolonged standing (Nachemson, 1981; Gregory, 2005), LBP persists. It is hypothesized that individual differences in how a person stands impacts the severity of LBP development during a prolonged period of standing (Gregory & Callaghan, 2008). Further, neuromuscular activity varies between acute LBP developers and non-pain developers during prolonged standing. Greater co-contractions of the gluteus medius (Nelson-Wong, Gregory, Winter, & Callaghan 2008) and trunk

musculature (Nelson-Wong & Callaghan, 2010) are found in people who develop acute LBP in comparison to non-pain counterparts. Alterations in muscle recruitment strategies are also found between these two cohorts (Nelson-Wong, Csepe, Lancaster, & Callaghan, 2012). Additionally, postural control strategies and changes in a person's centre of pressure (CoP) during standing reflect differences in people who experience LBP and those who do not (Lafond et al., 2009; Gregory & Callaghan, 2008; Gallagher, Nelson-Wong, & Callaghan, 2011). Knowledge of neuromuscular and postural control strategies may be helpful in early identification of at-risk LBP populations.

Interestingly, acute pain developers, after prolonged standing, report the desire to flex forward in an attempt to alleviate their pain (Gregory & Callaghan, 2008) despite known findings that flexion can cause LBP (McGill & Brown, 1993; Solomonow et al., 2003; Kumar, 2001). In 1951, Floyd and Silver coined the term "flexion-relaxation phenomenon" (FRP), whereby the erector spinae musculature quiets while in peak flexion. Thus, desired reports to flex forward after prolonged standing may in fact reduce LBP through the onset of FRP, inducing periods of muscle rest.

It is important to develop methods that alleviate and potentially prevent LBP in order to increase quality of life and the productivity of workers in occupations that demand prolonged standing postures. Therefore, the purpose of this study was to determine if transient trunk flexion would mitigate LBP development induced by prolonged standing in previously asymptomatic individuals. It was expected that the FRP would occur in acute LBP populations, and that FRP would provide transient muscle rests throughout two hours of prolonged standing.

2.0 Review of the Literature

2.1 Standing Induced Low Back Pain: The Impact of Ergonomic Mats and Insoles

Previous examinations of prolonged standing are vast (Cook et al., 1993; Gallagher et al., 2011; Gallagher, Campbell & Callaghan, 2014; Gregory, Sperling & Callaghan, 2006; Gregory, Brown, & Callaghan 2008; Gregory & Callaghan, 2008; Mika, Clark, & Oleksy, 2013; Nelson-Wong et al., 2012; Nelson-Wong and Callaghan, 2010; Nelson-Wong et al., 2008; Orlando & King, 2004; Pascal, Voigt, & Arendt-Nielsen, 1998). Many of these studies have examined the degree of pain experienced during prolonged standing and how it can be modulated by extraneous factors such as standing surfaces and footwear.

Ergonomic improvements in occupational settings aim to reduce risk factors for pain and increase worker productivity. The implementation of ergonomic mats and insoles have been reported to be beneficial in reducing discomfort and fatigue of the low back and lower leg muscles after prolonged standing (Pascal et al., 1998; Orlando & King, 2004; King, 2002). The purpose of an ergonomic floor mat is to induce sway and increase muscle activation of the lower extremity, promoting blood flow and minimizing the effects of fatigue (King, 2002). King (2002) examined the effect of four different standing conditions: a hard floor, an ergonomic floor mat, wearing shoe insoles and the combination of shoe insoles while standing on an ergonomic floor mat. The author compared floor conditions to perceptions of general fatigue and discomfort to the leg and back musculature over one week of eighthour shifts. The study found that insoles and the combination of insoles with floor mats provided the greatest pain relief whereas hard flooring conditions induced the most amount of pain. Likewise, Pascal et al. (1998) identified that soft surfaces prevented swelling of the lower leg associated with decreased fatigue and pain development. In general, ergonomic mats and insoles are beneficial tools to help alleviate LBP induced by standing. However, further understanding of the mechanisms behind standing-

induced LBP is crucial for ergonomists to properly develop appropriate and more advanced ergonomic tools to relieve LBP.

2.2 Neuromuscular Response Patterns to Low Back Pain

It has been debated within the literature whether impaired motor control of the lumbar spine predisposes people to pain, or if the pain causes impairment of motor control. The pain-spasm-pain model explains that pain will cause increased muscle activity that will in turn increase pain, supporting the latter notion that motor control impairments are likely adaptive to pain rather than causative (van Dieen, Selen, & Cholewicki, 2003). However, variations in neuromuscular response patterns such as muscle co-activation, trunk muscle reflex response latencies, trunk extensor activation, gaps in muscle activation and muscle recruitment patterns have been found to occur prior to the onset of LBP in previously non-symptomatic individuals, and are thought to be a risk factor of LBP development (Cholewicki et al., 2005; Gregory et al., 2008; Nelson-Wong & Callaghan, 2010; Nelson-Wong et al., 2008). This would support the notion that impaired motor control of the lumbar spine would predispose people to develop LBP. Differentiating between whether or not neuromuscular response differences are causal or adaptive in LBP holds significant weight in the diagnosis, treatment and prevention of LBP.

Cholewicki and colleagues in 2005 examined athletes' responses to suddenly applied loads prior to, and after the occurrence of low back injuries. The authors found that individuals who sustained an injury within the 2-3 year study period displayed approximately 14 millisecond longer delays in trunk muscle reflex latencies than non-injured controls. Interestingly, injured athletes, in comparison to healthy athletes, exhibited these different motor control patterns even prior to their injury. Therefore, trunk muscle reflex response latencies may be considered a risk factor in developing low back injuries and/or LBP rather than an adaptive mechanism.

In a study by Gregory et al. (2008), an involuntary trunk flexion perturbation in response to a suddenly applied load prior to 2 hours of standing consistently activated the trunk extensors in acute pain developers, with 100% firing probability (i.e. all muscles fired during each perturbation) even prior to the development of LBP. In comparison, non-LBP developers activated fewer trunk extensors and less consistently with a lower firing probability (78%). Trunk extensor musculature is therefore activated more consistently for LBP developers than non-pain developers (Gregory et al., 2008). Noting the delayed muscle responses of injured athletes in Cholewicki et al., 2005, Gregory et al. 2008 hypothesized that the higher activation of back extensors in acute pain developers may actually be an attempt to stabilize the spine to prevent injury.

Other neuromuscular differences exist in people who develop LBP as a result of prolonged standing such as increased number of gaps, or short periods of muscle quieting (Gregory & Callaghan, 2008) and co-activation of gluteus medius muscle is known to be approximately three times higher in pain developers as compared to non pain developers within the first 30 minutes of standing (Nelson-Wong and Callaghan, 2010; Nelson-Wong et al., 2008). The gluteus medius co-activation in LBP developers is believed to compensate for an inability to effectively utilize core musculature to maintain postural stability. The onset of muscle co-activation prior to pain development is suggested to be a predisposing factor in LBP development (Nelson-Wong and Callaghan, 2010; Nelson-Wong et al., 2008).

LBP developers demonstrate different muscle recruitment strategies compared to non-LBP developers (Nelson-Wong et al., 2012). In Nelson-Wong and colleagues' 2012 study, the researchers classified forty-three participants into two categories, acute pain developers and non-pain developers, after two hours of standing. Continuous electromyography (EMG) and hip and trunk range of motion were collected during standing trunk flexion and extension prior to standing. Results indicated that

acute pain developers utilized an atypical control pattern, activating a spine-dominant strategy during trunk extension, meaning that the lumbar muscles were active prior to gluteal muscles. In non-pain developer's muscles are activated in a caudal-to-cephalic order during extension, meaning that gluteal muscles were active prior to lumbar muscle activation. Their results demonstrate that LBP developers utilize different muscle recruitment strategies during prolonged standing in comparison to healthy matched individuals. Alterations in motor control patterns seem to occur prior to the onset of pain and may predispose individuals to develop LBP, appearing to be more of a causative factor than an adaptive factor of LBP. The alterations in muscle recruitment strategies may be beneficial for early intervention strategies in individuals who are predisposed to LBP induced by standing (Nelson-Wong et al., 2012).

One method to examine the neuromuscular response patterns is to quantify periods of muscle activation and muscle quiescence, or gaps, through the use of EMG. When muscle activity drops below 0.5% max voluntary contraction (MVC) for longer than 0.2 seconds, a muscle gap has occurred (Veiersted, et al., 1990) (Figure 1). Muscle quiescence conserves energy and decreases fatigue during demanding tasks.

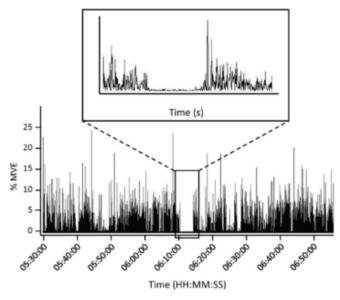


Figure 1: An example of a muscle gap (Harwood et al., 2011)

Harwood et al., (2011) demonstrated age- and sex-related differences in upper and lower body EMG gaps during various tasks, noting that older adults and females exhibited fewer gaps when compared to young adults and males. Using previous studies as a basis (Lynch et al., 1999; and Trappe et al., 2003), Harwood et al. (2011) proposed that differences in gap activity between males and females were due to reduced strength, muscle quality per cross sectional area and reduced muscle mass within females. Though Harwood et al., (2011) did not consider anthropometrics to standardize their data they concluded that physiological differences in musculature would necessitate higher amplitudes and longer durations of muscle activity in females than males, thus reduced gaps would be essential for females to execute tasks to the same degree as males.

The relationship between gap analysis and its influence on pain has revealed contradictory findings within the literature. Gregory and Callaghan (2008) found an increased number of gaps in the gluteus medius to be associated with the onset of LBP, suggesting that higher muscle activation might support the spine to a greater degree, decreasing LBP. However, Veirested et al., (1990) found that workers with preexisting complaints of pain to the trapezius muscle had higher levels of static muscular activity and fewer EMG gaps than workers who did not complain of pain, identifying that higher muscle activation was concomitant with pain. Muscle gaps may therefore provide brief periods of relaxation, or microbreaks for the muscle.

2.3 Postural Control Responses to Low Back Pain

Aspects of postural control such as CoP shifts, fidgets and drifts help researchers to characterize differences in postural control in individuals with LBP and healthy matched counter parts. Shifts occur when the CoP is displaced in a stepwise fashion. Fidgets occur when CoP moves away and then returns

to the same position and drifting identifies slow consistent displacement of the average CoP, as seen in Figure 2 (Duarte & Zatsiorsky, 1999).

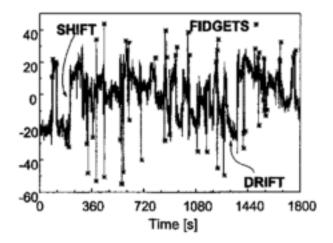


Figure 2: Example of CoP shift, drift and fidget in the A/P direction (Duarte & Zatsiorsky, 1999). Y axis shows location of CoP.

Using the aforementioned aspects of postural control, Lafond et al., (2009) found that chronic LBP patients displayed fewer shifts in CoP and less postural sway in comparison to healthy matched counterparts during a thirty minute standing protocol. The lack of mobility adopted by chronic LBP populations is thought to be an attempt to maintain spine stability (van Dieen et al., 2003).

Postural control strategies seem to differ between chronic LBP sufferers and acute back pain developers. Unlike Lafond et al., (2009), CoP A/P shift increases have been reported in acute pain developers (Gregory & Callaghan, 2008). Generally, acute pain developers seem to relate more closely to healthy populations than to chronic LBP populations (Gallagher et al., 2011) with greater postural sway in acute LBP developers and healthy counter parts than chronic LBP populations (Gregory & Callaghan, 2008; Lafond et al., 2009). Thus, it is likely that once a person develops LBP, their postural control system becomes altered. As pain progresses to become chronic, different strategies are utilized

that reduce postural sway (Lafond et al., 2009), potentially due to stiffer, more ridged muscles that may negatively affect mobility and adaptability to ones environment. Guarding ones motion to prevent sway may be another potential explanation for the different strategies used between chronic and acute LBP populations. If a person with chronic LBP were to adopt a more rigid posture they would likely prevent or guard against motion that may instigate pain development.

2.4 Flexion Tasks and Their Impact on Low Back Pain

To perform trunk flexion and extension motions from an erect posture, the pelvis and lumbar spine must work in synergy. During the initial phase of flexion from standing, the lumbar spine dominates approximately 58% of the motion; in the final stage of flexion the hip becomes dominant. In contrast, during the initial extension phase, 22% of the motion is hip dominant while the lumbar spine and hip equally dominate the remaining extension motion (Pal, Milosavljevic, Sole & Johnson, 2007).

In 1988, Dolan, Adams and Hutton studied thirteen commonly adopted postures and their effect on the lumbar spine. The authors found that majority of these postures put the spine into a flexed position, reducing lumbar lordosis in comparison to erect standing, concluding that individuals tend to gravitate towards postures that result in a flexed spine. Studies have further examined the impact of spinal flexion to alleviate/prevent LBP. Nelson-Wong and Callaghan in 2010 found that standing on a sloped surface (eQ Almond, Alberta, Canada) decreased reports of LBP during standing by 59.4%. Standing on the decline, when the toes are pointed downward, has been shown to decrease discomfort during standing by flattening the lumbar spine through posterior rotation of the pelvis and flexion of the lumbar spine and trunk (Gallagher et al., 2013). In contrast, standing on the incline, when the toes are point upward, acts in the opposite manner inducing anterior rotation of the pelvis and extension of the lumbar spine (Gallagher et al., 2013). Interestingly, Nelson-Wong and Callaghan (2010) found that

participants elected to spend 72% of their time standing in the declined position on the eQ Almond which induced lumbar flexion. Standing in transient trunk flexion induced from a sloped surface is therefore thought to be a promising means of reducing LBP (Nelson-Wong & Callaghan, 2010; Gallagher et al., 2013).

Alleviation of LBP through the use of transient trunk flexion has also been studied in the form of seated rest breaks during prolonged standing (Gallagher et al., 2014). These researchers evaluated the effect of a 15-minute seated break amidst two 45-minute standing periods. As acute pain developers transitioned into lumbar flexion during sitting, subjective reports of LBP decreased. Upon returning to standing, pain increased. By placing the spine in a flexed position, the seated breaks were able to effectively decrease pain development.

Trunk flexion angles are also altered when high-heeled shoes are worn (Franklin et al., 1995). As heel height increases during standing, trunk flexion angles decrease and extension of the spine increases, likely compensating for anterior displacement of the center of mass, while muscular activity of the lower leg and trunk increases contributing to muscle fatigue and pain (Lee, Jeong, & Freivalds, 2001; Franklin et al., 1995; Mika et al., 2013). Ultimately, over extension of the spine caused by excessively high-heeled shoes may cause musculoskeletal pain to the low back and leg musculature. Ko and Lee (2013) found that low-heeled shoes (0.5cm) and high-heeled shoes (9 cm) significantly altered the pressure distribution and CoP displacement of the foot. However, mid-heeled shoes (4cm) displaced the CoP minimally and did not significantly alter pressure distribution under the foot. In the mid-heeled shoe, balance and postural control was well maintained. Therefore, a medium heeled shoe, similar to the sloped surface examined by Nelson-Wong & Callaghan (2010) may reduce pain development by flattening the spine into a flexed posture to reduce over extension and maintaining CoP displacement and pressure distribution under the foot.

In summary, flexion of the spine has been shown to reduce LBP development during standing. The use of sloped surfaces (Nelson-Wong & Callaghan, 2010; Gallagher et al., 2013), seated rest breaks (Gallagher et al., 2014) and mid-heeled shoes (Ko & lee, 2013) have been shown to be effective at reducing LBP possibly by inducing flexion of the spine.

2.5 Flexion-Relaxation Phenomenon

In healthy individuals during peak lumbar flexion, the back extensors are silenced from their posture-supporting role. This is known as the flexion-relaxation phenomenon (FRP) (Figure 3). It is presumed that passive tissues (the intervertebral disc and posterior ligaments) take over for the trunk extensor musculature to support the increased moment about the spine during peak flexion. While silencing of the extensor muscles occurs, small amounts of force are still generated elastically through passive stretching (McGill & Kippers, 1994). Research has demonstrated that the quadratus lumborum and deep erector spinae muscles increase activity during FRP (Andersson et al. 1996). Thus, elastic responses of superficial erector spinae muscles and active responses of the deep musculature may support the spine at peak flexion when electrical silencing of the superficial erector spinae muscles occurs.

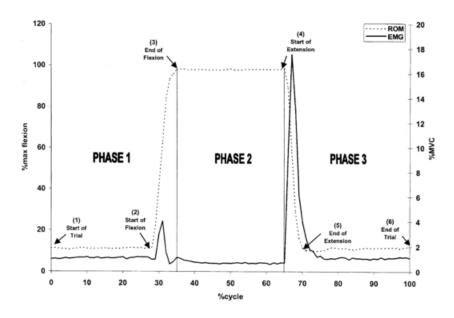


Figure 3: Lumbar flexion and EMG identifying FRP and the flexion extension cycle (Callaghan & Dunk, 2002). Note: ROM is defined as range of motion.

Activation of muscles other than the erector spinae have also been documented during the FRP. In addition to the lumbar paraspinal muscles, Olson, Solomonow, and Li (2006) measured the activity of the rectus abdominis, external obliques, semimembranosus and biceps femoris muscles during standing flexion. At peak flexion, myoelectric silencing of the paraspinal muscles and hamstring muscles was present, however the rectus abdominis and external obliques were active. During the extension phase, the onset of the semimembranosus and biceps femoris muscles occurred earlier than the reactivation of the paraspinal muscles (Olson et al., 2006). Though the abdominal muscles do not fully support the load of the upper body during the FRP, the abdominal activation found in Olson et al., (2006) indicates that they would aid in stabilization of the spine.

FRP is modulated by a multitude of factors. One way this may occur is by increasing erector spinae demand. Previous studies have shown that when a load is held anterior to the body, the FRP response is delayed. Specifically, delayed FRP onset and cessation angles in the lumbar and thoracic

region (Descarreaux et al., 2010) and cervical region (Pialasse et al., 2010) have been observed. Howarth and Mastragostino (2013) applied a load on the shoulders during flexion trials using a custom made vest, and found that as the mass increased, a greater degree of flexion was needed to initiate FRP. Together these studies identify that higher activation of the back extensor musculature is required to maintain stability of the spine at peak flexion when loaded.

The FRP is also different in standing versus seated positions specifically in terms of the onset and cessation angles of the FRP (Howarth et al., 2013; Olson et al., 2006; Callaghan & Dunk, 2002). Callaghan and Dunk (2002) found an average standing FRP onset angle (myoelectric silencing) to occur at 84% max lumbar flexion in standing posture, whereas the FRP onset angle in seated posture was much smaller, 46% max lumbar flexion. After extension had been initiated, the FRP cessation angle (myoelectric re-activation) during standing occurred at 94% of maximum flexion, while seated flexion produced an average FRP cessation angle of 52% of maximum flexion. FRP therefore occurs in seated flexion, though at much lower lumbar flexion angle than standing FRP. Callaghan and Dunk (2002) also noted that a slumped seated position induced FRP in the thoracic erector spinae muscles more consistently than in the lumbar erector spinae (LES) muscles.

Fatigue of the LES muscles further mitigates the effects of FRP such that myoelectric silencing occurs earlier after fatigue-inducing tasks and is accompanied by a later cessation of myoelectric silencing during extension post fatigue (Descarreaux et al., 2008). Thus in a state of fatigue, the erector spinae muscles are unable to adequately stabilize the spine, transferring the load to the passive tissues earlier in trunk flexion, increasing the duration of the FRP response. Interestingly, as the speed of flexion increases, the FRP response post fatigue occurs less frequently (Mathieu & Fortin 2000). Therefore the speed of motion when paired with a fatiguing task will act in the opposite manner and eliminate the FRP response.

Solomonow and colleagues in 2003 also found that induced tissue creep altered the FRP response. Three intervals of peak lumbar flexion and extension were performed before and after ten minutes of static flexion to induce creep. The authors found that creep increased the average FRP onset angle (approximately 46° of trunk flexion prior to creep versus approximately 50° post creep), causing a shorter period of erector spinae silencing during flexion. The authors also found that the erector spinae muscles were active earlier during the extension phases, after creep development potentially to assist in maintaining spine stability.

The change in FRP over time has been briefly reviewed in both sitting and standing postures. Howarth et al., (2013) found that one-hour of sitting increased the spinal flexion onset angle of FRP, indicating that creep may have developed in the viscoelastic structures of the spine. However, Nelson-Wong et al., (2010) found no significant changes in resting muscle activation of FRP post two hours of standing. To further understand changes in FRP over time, additional research is required.

In individuals with chronic LBP, myoelectric silencing of the FRP response is absent (Figure 4) (Colloca & Hinrichs, 2005; Neblett et al., 2003; Mannion et al. 2001; Kaigle et al., 1998; Shirado et al., 1995). To examine the change in FRP in clinical LBP populations, Kaigle et al. (1998) simultaneously quantified erector spinae muscle activation patterns, kinematic behaviour and FRP in seven people with chronic LBP and six asymptomatic controls. Chronic LBP individuals experienced only a 13% silencing of the trunk musculature as compared to 78% silencing shown by healthy controls. Kaigle et al. (1998) proposed that persistent myoelectric activity in chronic LBP individuals restricts intervertebral motion as a means to stabilize their injured or diseased spine. Likewise, Shirado et al. (1995) found that FRP occurred in 25 healthy controls, the average FRP onset angle was $81.6 \pm 5.1^{\circ}$ of trunk flexion and $40.8 \pm 8.2^{\circ}$ hip flexion. Myoelectric silencing continued until $62.2 \pm 6.7^{\circ}$ and $32.5 \pm 6.2^{\circ}$ of trunk and hip flexion during extension, respectively. In contrast, not one of 20 chronic LBP patients examined

experienced FRP at any point during the study (Shirado et al., 1995).

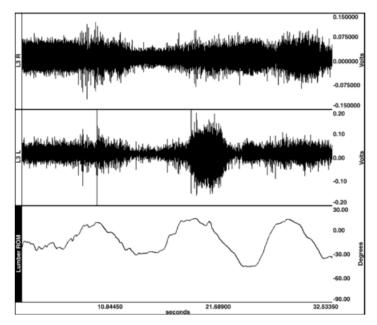


Figure 4: The top graph shows the absence of FRP in a person with clinical LBP. The middle graph shows FRP occurring in the second flexor motion in a person free of chronic LBP. The bottom graph shows the range of motion during three flexion-extension trials (Colloca & Hinrichs, 2005). Note that in the bottom graph, a more negative value is increased flexion.

The FRP has been found to reappear in chronic LBP populations after rehabilitation period, allowing shorter activation periods during peak flexion and increasing bouts of muscle relaxation.

Neblett et al., (2003) followed 54 chronic LBP sufferers over seven weeks of rehabilitation. Prior to the intervention only 30% of the chronic LBP population showed FRP. Post intervention, nearly 95% of the population who completed the program showed FRP.

Differing from chronic LBP patients, FRP has been shown to occur in acute LBP participants. Horn and Bishop (2013) utilized a delayed onset muscle soreness protocol (strenuous back extension task) to induce acute LBP in 51 non-symptomatic individuals. Muscle activity of the LES muscles and measures of FRP were tested prior to and post delayed onset muscle soreness protocol. The presence of acute pain did not significantly alter myoelectric silencing during trunk flexion and extension. These findings suggest that the delayed onset muscle soreness protocol is not enough to eliminate FRP as seen

in chronic LBP populations. During trunk flexion in acute LBP, the muscles continue to silence similar to non-LBP population therefore resulting in the passive structures (ligaments) of the spine to accommodate for the increased spinal loading during peak flexion.

2.6 The Importance of Work Breaks

Providing workers with appropriate rest breaks during any prolonged static posture is an important preventative mechanism to reduce LBP. Sbriccoli et al., (2004) identified that seven hours of recovery after a 1:1 work-rest schedule was not enough time to alleviate creep development in the viscoelastic structures of the spine, which can propagate injury. In 1998, van Dieen and Vrielink studied the effect of four different work-rest ratios used by poultry inspectors: 30-30 minutes, 30-15 minutes, 60-15 minutes and 45-15 minutes. The researchers found that frequent short breaks and periods of dynamic activity reduced viscoelastic deformation of the spine during prolonged standing to a greater extent than the 60-15 ratio. More recent research has expanded the concept of frequent short breaks, proving that scheduled microbreaks, lasting approximately 30 seconds, interspersed between periods of 15-20 minutes of seated desk work decreased muscular discomfort and increased worker productivity to a greater extent than longer duration, less frequent rest breaks (Mclean et al., 2001; Balci & Aghazadeh, 2003). It is possible that the low back muscle silencing that occurs during the FRP may aid in the alleviation/prevention of LBP by acting as a microbreak.

2.7 Concluding Remarks

In summary, prolonged standing is a known risk factor for LBP (Cook, Branch, Baranowiski, & Hutton, 1993; Xu, Bach, & Orhede, 1997; Zander, King, & Ezenwa, 2004). Ergonomic aids such as sloped surfaces, ergonomic mats, and proper insoles can be used to alleviate and potentially prevent

LBP. Interestingly, some of these interventions put the spine into flexion and therefore it is possible that small bouts of flexion may alleviate pain. Silencing of the erector spinae muscles during the FRP may act as a short microbreak for the low back musculature, reducing LBP development. Engaging FRP may be a simple method that workers can utilize to relieve and possibly prevent LBP.

2.8 Purpose and Hypotheses

2.8.1 Over-Arching Purpose

The purpose of this study was to determine if trunk flexion would mitigate low back discomfort development induced by prolonged standing in previously asymptomatic individuals.

2.8.1.1 Hypothesis

Intermittent, short duration periods of flexion would cause the flexion relaxation phenomenon (FRP), which would induce rest, in turn decreasing low back discomfort (LBD) development.

2.8.1.2 Specific Aims and Hypotheses

Aim #1) To determine if FRP occurs during prolonged standing in those who develop low back discomfort and those who do not.

a. It was expected that some participants would develop acute LBD and be considered discomfort developers (LBD developers) and that some participants would not develop LBD and be considered non-discomfort developers (non-LBD developers) and that FRP would occur in both LBD developers and non-LBD developers.

Aim #2) To determine if FRP changes over time during prolonged standing.

b. It was hypothesized that muscle quiescence would occur at a greater point of trunk flexion over the 2-hour standing period.

- c. Additionally, it was hypothesized that muscle activity during FRP would increase over time.
- d. It was hypothesized that no differences would exist between males and females during FRP.
- e. It was also hypothesized that the CoP excursion would move anteriorly during flexion and would not change over time.

Aim #3) To determine if FRP affects discomfort development.

f. It was hypothesized that FRP would provide transient muscle rests throughout two hours of prolonged standing such that the level of LBD reported would be lower at the end of two hours of standing in comparison to that at the end of a two-hour control day (standing with no flexion periods).

3.0 Methodology

3.1 General Overview of the Research Experiment

Each individual participating in the study was required to stand for two hours on two separate occasions, one week apart. One day was considered the control protocol during which participants were asked to stand for two hours within a confined workspace (approximately 0.25 square meters). On a separate day, the experimental protocol, two hours of standing (within the same confined workspace) was divided into eight 15-minute blocks. At the start of the protocol, and after every 15 minutes, the participants were instructed to bend forward to maximum flexion and then return to upright standing, for a total of nine flexion trials (Figure 5). Each flexion trial was 25 seconds consisting of five phases: (1) 5 seconds neutral standing, (2) 5 second flexion phase, (3) maintain full flexion for 5 seconds, (4) 5 second extension phase, and (5) 5 seconds standing neutral (Howarth et al., 2013; Howarth & Mastragostino, 2013). Timing the motion with a metronome set at 60 beats per minute controlled for the cadence of movement during the flexion tasks as demonstrated in previous studies (Callaghan & Dunk, 2002; Howarth et al., 2013). During the flexion phase, participants were instructed to bend forward as far as they could comfortably, let their arms and upper body hang freely and allow their back to relax fully. These flexion trials were used to induce the FRP previously described. Participants were given two to three practice trials before initiating the experimental protocol to ensure participants understood the procedure. The order of the control protocol and experimental protocol were randomly assigned to each participant prior to commencing the study. Data collections for both protocols were conducted at a similar time of day for each participant to avoid any diurnal effects.

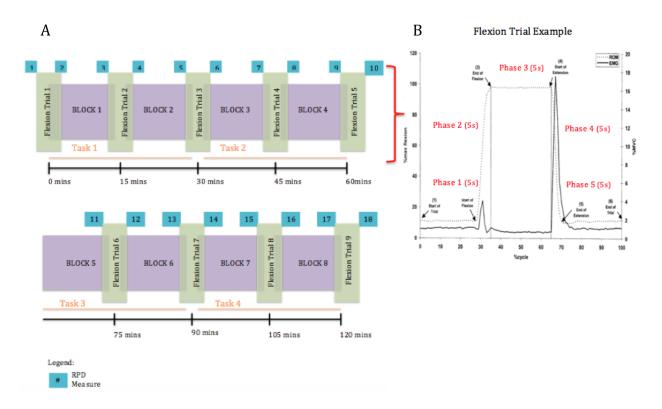


Figure 5: Depiction of experimental protocol (A). Eight standing blocks were separated with nine flexion tasks; prior to and after each flexion trial, an RPD scale was filled out. For every two standing blocks (30minuts) participants performed one of four fine motor control tasks. Flexion trial example (B) with each of the five-flexion phases identified (adapted from Dunk & Callaghan, 2002).

While standing, participants performed various fine motor tasks: pen assembly, computer work, currency sorting, and card dealing. Each task was completed for 30 minutes; in the case of the experimental protocol, one flexion trial occurred at each 15-minute mark. The tasks were completed in the same order as listed above for every participant. A standing workstation was adjusted to fit to the height of each participant such that the top of the desk was slightly lower than the participants elbow, when flexed at 90 degrees.

Participants were not permitted to use anti-fatigue mats or foot rests but were asked to wear athletic shoes as per previous work (Nelson-Wong 2012; Nelson-Wong 2010; Gregory & Callaghan 2008). Participants were able to slightly adjust their posture within the confined space and rest their

forearms on the workstation without supporting their body weight.

During the flexion trials, muscle activation from two back extensor muscles was collected (refer to 3.3.1) using surface electromyography (EMG), as well as lumbar spine flexion, and CoP data in the anterior/posterior direction. Prior to and following each flexion trial, each participant completed a rating of perceived discomfort scale (RPD) for the following body regions: upper back, lower back, feet and overall (refer to 3.3.4).

Upon completion of both the experimental protocol and the control protocol, participants were asked to complete an exit survey to discuss any pain/discomfort they may have experienced, if they felt flexion provided any relief to discomfort, if they believe there were enough flexion periods and any other positions they would adopt to alleviate any pain development. Participants were compensated \$25.00 for participating in the study.

3.2 Participants

A sample population of young adults between 18-30 years of age were tested for this study. Eight males and eight females were recruited to perform the study, totalling sixteen (Table 1). Participants were recruited using posters displayed across campus. Each participant was given a screening questionnaire (Appendix A) to determine if they met the criteria to partake in the study. The questionnaire included general information about demographics and previous history of LBP. Participants were excluded from the study if they had experienced LBP in the past 12 months that required them to visit a doctor and/or take time off of work. Participants were also asked to refrain from the use of non-steroidal anti-inflammatory drugs for the relief of pain 48 hours prior to each collection day.

On collection day 1, each participant was asked to sign a formal consent form detailing the

protocol and any benefits or risks that the participants may experience during the protocol. Wilfrid Laurier University Ethics Board reviewed and approved the study prior to data collection.

Table 1: Descriptive statistics of participants who completed the study; mean (SE).

	n	Height (cm)	(SE)	Weight (Kg)	(SE)	Age (yrs.)	Activity Level (Days per week of moderate to vigorous activity)
Male	8	176.20	1.17	74.32	3.32	22.75	4.5
Female	8	171.13	1.73	66.10	2.22	22.88	3.5
Total	16	173.67	1.20	70.21	3.30	22.81	4

3.3 Instrumentation and Data Processing

3.3.1 Electromyography

Two pairs of Ag-AgCl electrodes (Ambu Blue Sensor, Denmark) were placed on prepared skin (shaved if necessary and cleaned with 70% isopropyl rubbing alcohol) over the following muscles: lumbar erector spinae (LES) and gluteus medius (GM). Electrodes were placed 3cm lateral to L3 spinous process for LES (McGill, Norman, & Cholewicki, 1996), and 15cm inferior and 5cm posterior to each iliac crest for GM (Gregory & Callaghan, 2008). Last, one electrode was placed on the left anterior superior iliac spine as a reference electrode.

3.3.1.1 Maximum Voluntary Contractions and Baseline Measures

Following electrode placement, maximum voluntary contractions (MVC) were collected to normalize the EMG data. Before MVCs were obtained, lead connections were checked to ensure

appropriate readings using an oscilloscope. If necessary the signals were gained up or down to maximize signal resolution. For the LES MVC, a back extension was performed against resistance. Participants' lower body was secured to a physiotherapy table with his/her torso hanging off of the table. The participant was instructed to lift his/her torso to horizontal and extend against resistance applied by the researcher. To obtain a GM MVC, participants were instructed to lie on their side opposite to the leg performing the MVC and abduct their leg against resistance. Each MVC was performed for 3-5 seconds during which verbal encouragement was given. Following MVCs, participants were instructed to lie quietly, prone, on the physiotherapy table while a 5 second rest trial was collected.

3.3.1.2 Data Processing

All EMG data were bandpass filtered from 10 to 1000Hz, amplified (Bortec, Calgary, Alberta) and sampled at 2048Hz. Raw EMG data were full-wave rectified and dual-pass filtered using a fourth-order Butterworth filter with a low-pass cut off of 2.5Hz to create a linear envelope. The linear enveloped data were further normalized to the MVC performed by each muscle and down sampled to 32Hz to coordinate with kinematic data.

3.3.2 Kinematics

To capture lumbar flexion angles during FRP onset and cessation, an electromagnetic motion capture system was used (Liberty, Polhemus, Colchester, Vermont). Following MVCs, motion sensors were placed on the body at the L5/S1 (lumbosacral) joint, T12/L1 joint and C7/T1 joint. The angle between the T12/L1 and L5/S1 markers represented the lumbar spine flexion posture. Prior to the start of the standing protocol, each participant performed an upright standing and full flexion-extension trial to determine the maximum spinal range of motion (ROM) in the lumbar spine.

3.3.2.1 Data Processing

Motion data were sampled at 32 Hz, dual-pass filtered with a low-pass cut-off of 6Hz. Data was then normalized to the maximum trunk flexion reached in the ROM trial. Kinematic and EMG data were synchronized.

3.3.3 Centre of Pressure (CoP)

CoP excursion in the anterior-posterior direction ($CoP_{A/P}$) was determined during each flexion trial during the experimental protocol. $CoP_{A/P}$ was calculated using Equation 1 below; moment (M_y) and force (F_z) data were collected from the force plate (sampled at 2048 Hz and dual-pass filtered with a low-pass cut-off of 6Hz) and converted from volts to Newtons (N) and Newton-metres (Nm), respectively. The absolute maximum was then subtracted from the absolute minimum to determine the CoP excursion and expressed in centimetres (cm).

Equation 1:
$$CoP_{A/P} = \frac{M_y}{F_z}$$

3.3.4 Ratings of Discomfort

The visual analog scale (VAS) has been shown to be reliable (Bijur, Silver & Gallagher, 2001) and have good construct validity (Summers, 2001). Therefore, a 100mm VAS was used to measure perceptions of pain at the start and end of the protocol and before and after each flexion trial, for a total of 18 measures during the experimental protocol (Figure 5). The same VAS scale was used to measure discomfort at the start, end and every 15 minutes during the protocol of the control day, for a total of 9 measures. Participants were asked to mark their discomfort by drawing a vertical line on a 100mm scale, where 0 represented "no discomfort" and 100 represented "worst discomfort imaginable". Additionally, participants were asked to describe the

discomfort as soreness, numbness, sharp, dull, localized, painful or distributed, as seen in de Looze et al., (2003). Please see the Ratings of Perceived Discomfort Scale (RPD) in Appendix B. Discomfort ratings were determined by measuring the distance from the origin to the marking, to the nearest millimeter for each body area (upper back, lower back and legs and overall). Participants who ranked LBP greater than or equal to 8mm at any point during the standing protocol were considered to be pain developers. This cutoff was chosen because 8mm is the minimum clinically important difference for an increase in VAS scores in the low back (Hägg et al., 2003).

3.3.5 Flexion Relaxation Phenomenon

FRP was determined through visual inspection, threshold analysis and by using the flexion relaxation ratio (FRR) (Nelson-Wong et al., 2010). For each of the nine-flexion trials, relaxation of the right erector spinae musculature was identified initially through visual inspection of the linear enveloped EMG data (Howarth et al., 2013; Howarth & Mastragostino, 2013). This visual inspection was not used to define FRP but rather to determine a relaxation zone. The average magnitude of the EMG signal during relaxation was then calculated over 2 seconds of data. Since kinematic data and EMG data were collected simultaneously, the lumbar spine angle at the point of FRP onset and cessation was determined using an amplitude threshold that was two times the average EMG relaxation value over 2 seconds of data (Howarth et al., 2013; Howarth & Mastragostino, 2013; Jin, Nig, & Mirka, 2012) (Figure 6). For each participant, the first flexion trial was used to determine a threshold, which was subsequently used to calculate FRP for all preceding flexion trials. This constant threshold was used to determine if any changes had occurred to the FRP over time. Variables of interest included: lumbar spine angle at onset and cessation of FRP, maximum flexion angle, average muscle activation during FRP, peak activation during the flexion phase of the trial, peak activation during extension from flexion, FRP duration (based

on threshold), and average muscle activation during quiet standing. Based on previous literature (Nelson-Wong et al., 2012; Nelson-Wong et al., 2010; Dankaerts et al., 2006), which found no differences between the left and right LES during FRP, only the right side of the body was analyzed.

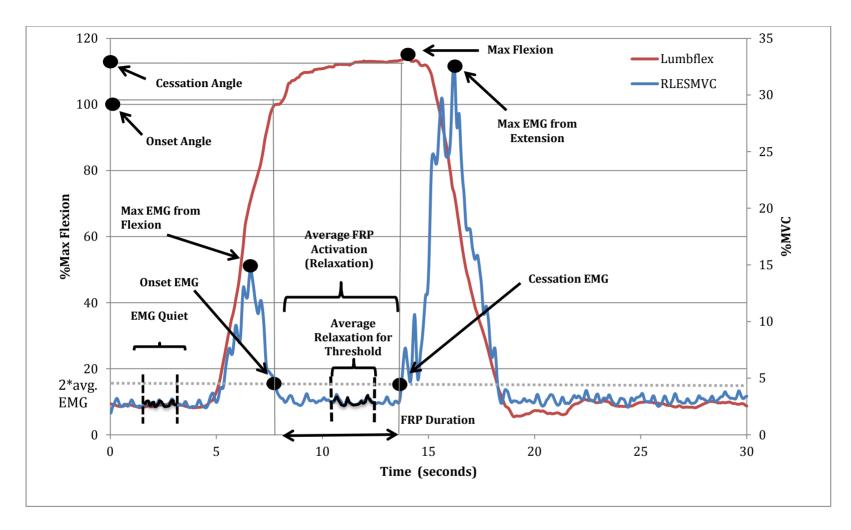


Figure 6: FRP analysis. Linear enveloped EMG data from the lumbar erector spinae (solid blue line) and the kinematic data during flexion (solid red line). Variables include lumbar spine onset and cessation flexion angle, maximum flexion angle, average muscle activation during FRP, peak activation during flexion, peak activation during extension from flexion, FRP duration, and average muscle activation during quiet standing. Threshold identified (dashed grey line) calculated by 2*(Avg. FRP activation over 2 seconds) as identified by the vertical black dashed lines.

The ratio (termed flexion relaxation ration; FRR) between average muscle activation during upright standing and during FRP was used to further assess if FRP occurred (Equation 2) (Nelson-Wong et al., 2010; Dankaerts et al., 2006). Trials were considered to have reached FRP if the FRR was equal to or greater than 0.75 for the LES and 0.50 for the GM. This criterion was determined using previous literature (Nelson-Wong, 2009) to determine average upright standing muscle activation of the LES and GM (~3% and 2% MVC, respectively) and by visually inspecting the current data set to determine muscle activation during FPR (found to be ~4% MVC for each muscle). As such, 112 trials from the LES data set and 93 trials from the GM data set were considered to have reached FRP from the original 144 trials. Any trials that did not reach FRP were individually removed from the data set as opposed to removing the entire participant data set.

Equation 2:
$$FRR = \frac{avg.\ muscle\ activation\ during\ upright\ standing}{avg.\ muscle\ avtivation\ during\ FRP relaxation}$$

3.4 Statistical Analysis

Using SAS statistical software, a two-way, mixed model, analysis of variance (ANOVA) was conducted to determine if differences existed in FRP and CoP over time during the experimental (flexion) day. In each case, independent variables included blocks of time (repeated measure) and sex.

A second two-way, mixed model ANOVA was conducted to determine if differences existed in ratings of perceived discomfort during the control day, where blocks of time (repeated measure) and sex were the independent variables.

A third two-way, mixed model ANOVA was conducted to determine if differences existed in ratings of perceived discomfort by condition (control day, discomfort rating just prior to flexion trial (pre-flexion), discomfort rating just after flexion trial (post-flexion)) and by sex.

A fourth two-way mixed model ANOVA was used to determine if a difference existed between the FRP variables and LBD developers versus non-LBD developers, where independent variables included blocks of time and discomfort classification.

Finally, a Chi-square analysis was used to further assess if the occurrence of FRP affected LBD.

An alpha level of 0.05 was considered significant.

Any significant main effects of time, sex or discomfort (for the ANOVAs) were further examined using Duncan post hoc comparisons.

4.0 Results

4.1 FRP

4.1.1 Lumbar Erector Spinae FRP

A total of 144 (9 trials x 16 participants) flexion trials were collected from 8 males and 8 females. Of those 144 trials, FRP occurred in the LES 78% (112/144) of the time, with an average of 92.4% muscle silencing in the LES. All sixteen participants achieved FRP in LES at least once during the experimental protocol. Average and standard error values are displayed in Table 2 for all FRP variables (significant p-values are listed in Appendix G, Table G1).

Table 2: LES FRP variable averages (SE) of trials considered to have reached FRP (112 trials).

FRP Variable	Average	(SE)
Threshold Value (%MVC)	3.39	(0.16)
Onset FRP (%Max. Flexion)	93.2	(1.55)
Cessation FRP (%Max.	103.36	(1.63)
Flexion)		
Peak Flexion Angle	104.12	(1.64)
(%Max. Flexion)		
FRP Duration (sec.)	6.95	(0.15)
Average Muscle Activation	2.10	(0.08)
During FRP (%MVC)		
Peak Activation During	12.25	(0.36)
Flexion (%MVC)		
Peak Activation During	27.49	(0.66)
Extension for Flexion		
(%MVC)		
Average Muscle Activation	2.18	(0.09)
During Quiet Standing		
(%MVC)		
FRR	1.05	(0.03)

4.1.1.1 Effect of Time

A significant effect of time for lumbar angle at FRP onset (p=0.007) was identified (Figure 7) where trials 4 (45mins) and 8 (105mins) displayed significantly larger flexion angles. The average FRP duration also significantly differed over time (p=0.02). At trials 1 (0mins) and 3 (30mins), the total length of time that FRP occurred (Figure 8: FRP duration) was greater compared to all other trials.

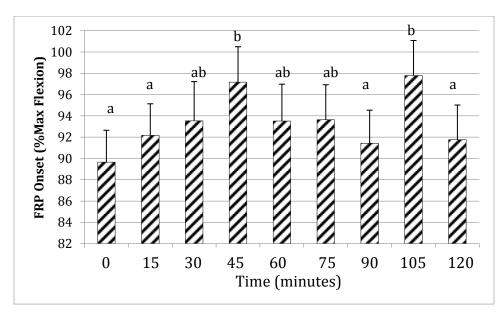


Figure 7: Effect of time for FRP onset (LES FRP). Different letters represent significant differences determined via post-hoc analysis.

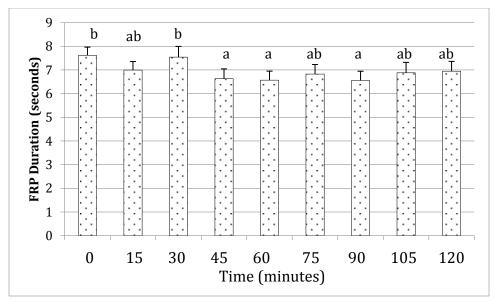


Figure 8: Effect of trial for average (SE) LES FRP duration. Different letters represent significant differences determined via post-hoc analysis.

4.1.1.2 Effect of Sex

A significant effect of sex (p=0.012) for peak LES activity during the extension phase of FRP was identified such that females reached a higher activation during the extension phase (30.49%MVC; SE 0.73) compared to males (23.55%MVC; SE 0.95), refer to Figure 9. There was no main effect of sex for average LES activation during quiet standing, though there was a trend towards significance (p=0.068).

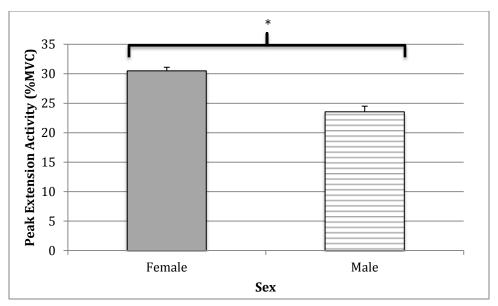


Figure 9: Effect of sex for peak LES activity during extension phase of FRP. Average EMG activity and SE displayed.

4.1.1.3 Interaction Between Time and Sex

Significant interactions between time and sex were identified for average LES activation during FRP (p=0.0018), average LES activation during quiet standing (p=0.036) and peak LES activity during the extension phase of flexion (p=0.0005). In general, females tended to exhibit higher LES activation during FRP, higher activation during quiet standing, and higher peak activation during extension from flexion. However, this difference was not consistent over time, as seen in Figures 10-12. Interestingly, males experienced greater relaxation than females (as seen by the lower EMG values in Figure 10). Though there was no main effect of sex for activation during FRP, trends towards significance for both sex (p=0.06) and time (p=0.069) were noted (refer to Appendix G, Table G1).

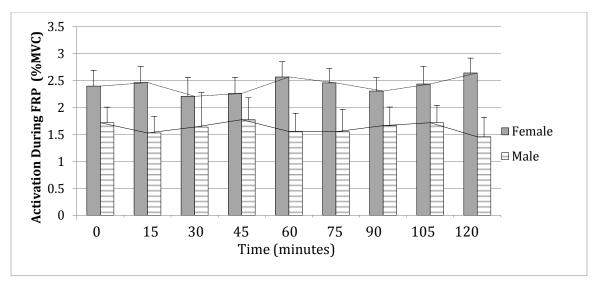


Figure 10: Interaction between time and sex for LES activation during FRP.

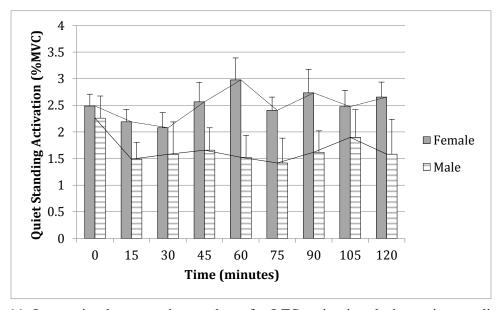


Figure 11: Interaction between time and sex for LES activation during quiet standing.

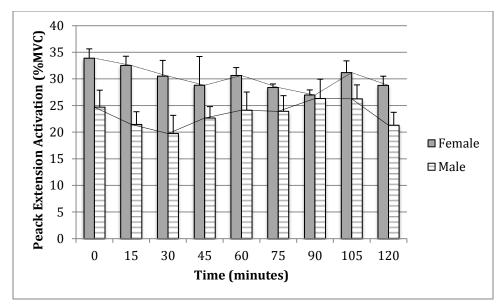


Figure 12: Interaction between time and sex for peak LES activation during extension phase of trial.

4.1.2 Gluteus Medius FRP

Similar to LES, a total of 144 FRP trials were collected (9 trials x 16 participants). Of those 144 trials, FRP occurred in the gluteus medius 65% (94/144) of the time. Thirteen of the sixteen participants achieved FRP in the gluteus medius. Average and standard error values are displayed in Table 3 for all variables and p-values marking significance of each variable in a 2-way repeated measures ANOVA of sex by time are displayed in Appendix G, Table G2.

Table 3: GM FRP variable averages (SE) of trials considered to have reached FRP.

FRP Variable	Average	(SE)
Threshold Value (%MVC)	6.72	(0.28)
Onset FRP (%Max.	86	(2.47)
Flexion)		
Cessation FRP (%Max.	98.38	(2.06)
Flexion)		
Peak Flexion Angle	100	(2.05)
(%Max. Flexion)		
FRP Duration (sec.)	7.18	(0.17)
Average Muscle Activation	3.94	(0.19)
During FRP (%MVC)		
Peak Activation During	15.75	(1.12)
Flexion (%MVC)		
Peak Activation During	19.81	(1.24)
Extension for Flexion		
(%MVC)		
Average Muscle Activation	3.58	(0.19)
During Quiet Standing		
(%MVC)		
FRR	0.95	(0.03)

4.1.2.1 Effect of Time

A significant effect of time for average GM activation during FRP (%MVC) was identified (p<0.001). At trial 9 (120mins), GM activation during FRP was higher than all other trials (4.62% MVC; SE 0.81), as seen in Figure 13. Average GM activation during quite standing also significantly differed as time progressed (p<0.001). At trials 8 (105mins) and 9 (120mins), activation during quiet standing was found to be higher (4.08%MVC, SE 0.88; 4.05%MVC, SE 0.84 respectively) than all other trials, as seen in Figure 14.

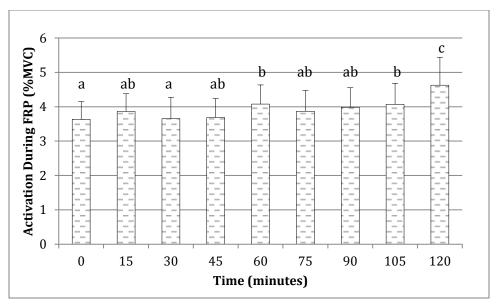


Figure 13: Effect of time for average (SE) GM activation during FRP. Different letters represent significant differences determined via post-hoc analysis.

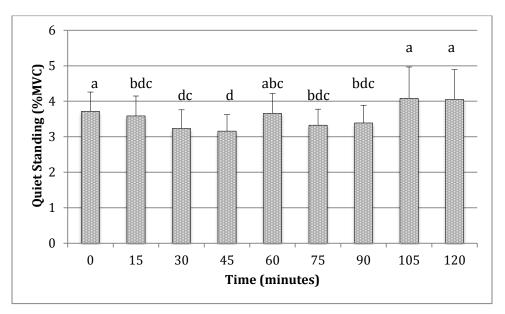


Figure 14: Effect of time for average (SE) GM activation during quiet standing. Different letters represent significant differences determined via post-hoc analysis.

4.1.2.2 Effect of Sex

No significant results were identified for the effect of sex as seen in Appendix G, Table

4.1.2.3 Interaction Between Time and Sex

Significant interactions between time and sex were identified for cessation angle of FRP (p=0.029), for average GM muscle activation during FRP (p=0.006), and average GM activity during quiet standing (p <0.0001). Typically, FRP ended at a larger flexion angle in females than males. Regardless, cessation angle was relatively consistent, with some variability over time (Figure 15). Similar to LES FRP activation, males experienced greater relaxation in the GM than females with the exception of trial 8 (105mins) (Figure 16). Finally, males tended to experienced more variable GM activity during quiet standing, compared to more consistent activity in females (Figure 17).

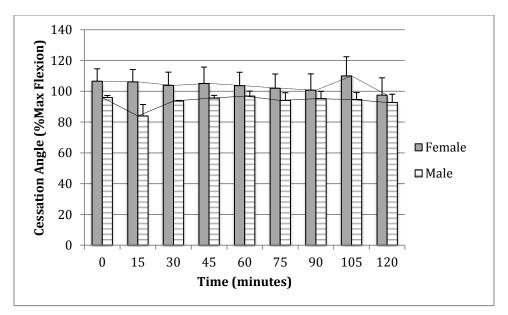


Figure 15: Interaction between time and sex for average (SE) lumbar flexion at GM FRP cessation.

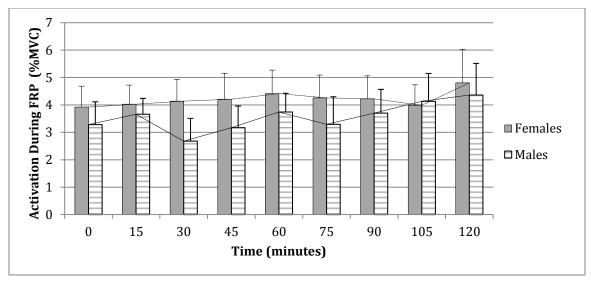


Figure 16: Interaction between time and sex for average (SE) GM activation during FRP.

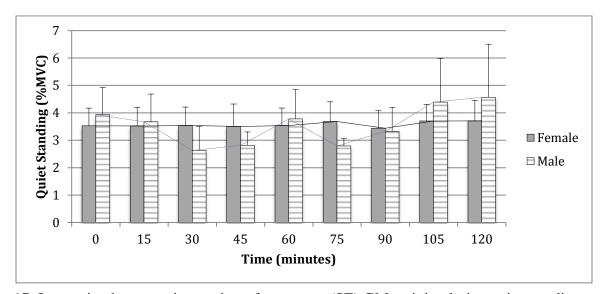


Figure 17: Interaction between time and sex for average (SE) GM activity during quiet standing.

4.1.4 Centre of Pressure Analysis

No significant differences were found between males and females (p=0.992), over time (p=0.360) or in the interaction between sex and time (p=0.962). The overall average CoP excursion was 8.53cm (SE 0.23).

4.2 Ratings of Perceived Discomfort

4.2.1 Low Back Discomfort Questionnaire

Though all participants were free of previous history of serious LBP within the last 12 months, 44% of participants had previously experienced acute low back pain due to prolonged standing exposure. Those who experienced pain due to standing reported onset of pain as early as 20 minutes after standing to 4 hours after standing. Eighty-eight percent of the participants had previous work experience that required prolonged standing; jobs included: cashiers, sales workers, teachers, coaches, city workers, waitresses, labour workers and factory workers. Of the 16 participants, 69% reported standing for more than 15 minutes a day, 25% reported standing for more than 15 minutes twice a day and 6% reported standing for more than 15 minutes multiple times a day.

4.2.2 Rating of Perceived Discomfort During the Control Protocol

4.2.2.1 Effect of Time

Lower-back (p<0.0001) discomfort ratings significantly increased as time progressed, most notably at 75 minutes as seen in Figure 18 (see Appendix G and H for all other body regions). Figure 19 identifies maximum discomfort ratings over two hours of prolonged standing. Ratings ≥8mm were considered LBD developers.

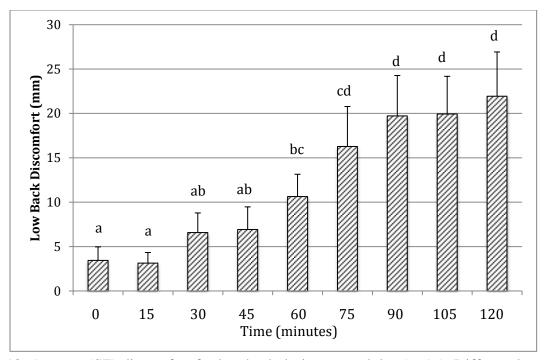


Figure 18: Average (SE) discomfort for low back during control day (n=16). Different letters represent significant differences in discomfort ratings determined via post-hoc analysis.

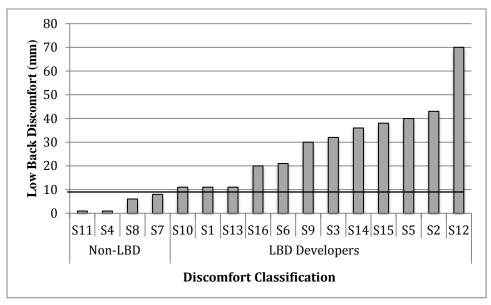


Figure 19: Subjects maximum low back discomfort ratings over two hours of prolonged standing. Ratings ≥8mm were considered LBD developers.

4.2.2.2 Effect of Sex

Females experienced greater LBD than males, although this did not quite reach significance (p=0.06). Refer to Appendix H, Figure H4 for main effect of sex for all other body regions.

4.3 The Effect of Flexion on Low Back Discomfort

4.3.1 Low Back Discomfort Developers Vs. Non-Low Back Discomfort Developers

Of the sixteen participants, twelve participants were considered low back discomfort developers (LBD developers) based on control day low back VAS scores that exceeded 8mm at any time point during the two hours of standing. The average VAS score for non-low-back discomfort developers (non-LBD developers) was 1.64mm (SE 0.4) and 16.19mm (SE 1.78) for LBD developers over the two-hour control protocol.

As the overarching purpose was to determine the effect of intermittent, short duration flexion periods on LBD development, only the 12 individuals who developed LBD during the control day were included in the following analysis.

4.3.2 Effect of Condition (control versus pre-flexion versus post-flexion)

Ratings of perceived low back discomfort were examined between three conditions (control day, pre-flexion score and post-flexion score) and across time. For the effect of condition, near significance was observed (p = 0.055). Although not significant, a general trend of higher discomfort scores during the control day followed by pre-flexion then post-flexion was noted, as seen in Figure 20.

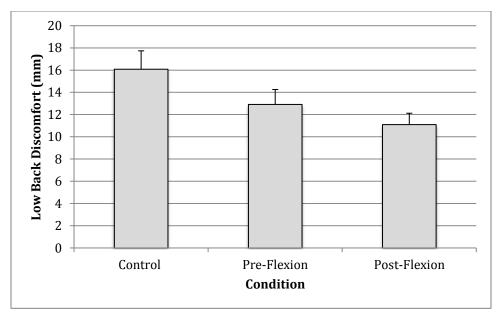


Figure 20: Effect of condition on low back discomfort for LBD developers individuals. Average (SE) discomfort scores collapsed across time.

4.3.3 Effect of Time

A significant effect of time was identified (p<0.0001), collapsed across condition, with increasing LBD scores over time (Figure 21).

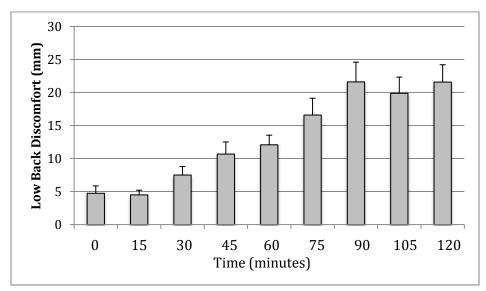


Figure 21: Effect of time for LBD developers. Average (SE) discomfort scores collapsed across condition.

4.3.1.3 Interaction Between Condition and Time

Of particular interest was a significant interaction between condition and time (p<0.0001). While the pre and post-flexion trials generally resulted in less discomfort than the control condition (Figure 22), post hoc analysis revealed that post-flexion discomfort scores became significantly different from control at 75 minutes into the two-hour standing protocol and pre-flexion discomfort scores after the full 120 minutes (Figure 22). Interestingly, no significant changes in low back discomfort were identified between pre and post flexion.

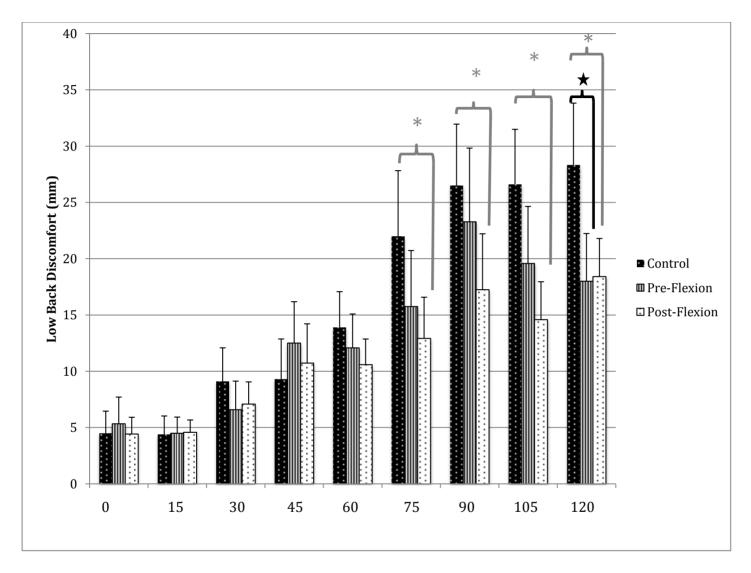


Figure 22: Interaction between condition and time for low back pain developers. Significant differences found at 75 (p=0.01), 90 (p=0.02), 105 (p=0.01) and 120 (p=0.04) minutes between control and post-flexion (represented by the grey asterisks). At 120 minutes, pre-flexion discomfort scores became significantly different from control sores (p=0.01), as represented by the black star. Average and standard error displayed.

4.4 Does FRP Influence LDB Scores?

4.4.1 FRP Variables and LBD Developers vs. Non-LBD Developers

4.4.1.1 LES FRP: LBD Developers vs. Non-LBD Developers

Reintroducing the four non-LBD developers, the following section reviews the differences found between non-LBD developers and LBD developers in FRP for the LES.

Average and standard errors for each of the FRP variables of interest are presented in Table 4 for the LES and categorized by non-LBD developers or LBD developers. Level of significance for each FRP variable compared by discomfort across time in the LES muscle can be found in Appendix G, Table G3.

Table 4: Non-LBD developers vs. LBD developers – LES FRP variable averages (SE)

FRP Variable	Non-LBD Developers	LBD Developers
	Average (SE)	Average (SE)
Onset FRP (%Max. Flexion)	91.72 (3.51)	93.66 (1.74)
Cessation FRP (%Max.	99.04 (2.9)	104.71 (1.95)
Flexion)		
Peak Flexion Angle	100.06 (2.91)	105.39 (1.95)
(%Max. Flexion)		
FRP Duration (sec.)	7.15 (0.23)	6.89 (0.18)
Average Muscle Activation	1.15 (0.05)	2.38 (0.08)
During FRP (%MVC)		
Peak Activation During	8.13 (0.27)	13.53 (0.37)
Flexion (%MVC)		
Peak Activation During	21.40 (1.23)	29.58 (0.59)
Extension for Flexion		
(%MVC)		
Average Muscle Activation	1.13 (0.09)	2.51 (0.096)
During Quiet Standing		
(%MVC)		
FRR	0.98 (1.07)	1.07(0.04)

4.4.1.1.1 Effect of Discomfort

A significant main effect of discomfort was identified for LES peak activation during flexion (p=0.004), LES peak activation during extension from flexion (p=0.009) and LES activity during quiet standing (p=0.004). In all cases, LBD developers experienced greater activity of the LES than non-LBD developers (refer to Figures 23-25).

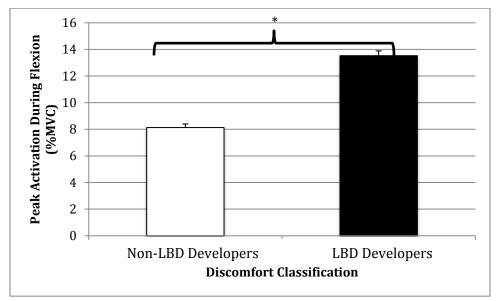


Figure 23: Main effect of discomfort for LES peak activation during flexion (average and SE displayed; asterisk represents $p \le 0.05$).

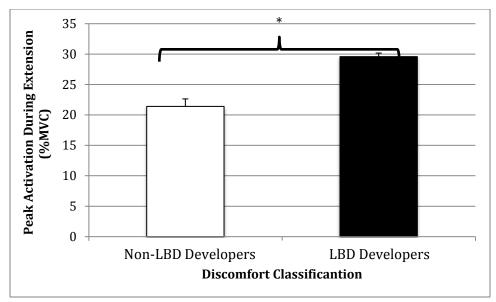


Figure 24: Main effect of discomfort for LES peak activation during extension from flexion (average and SE displayed; asterisk represents $p \le 0.05$).

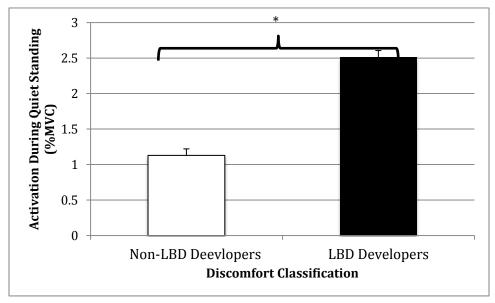


Figure 25: Main effect of discomfort for LES activity during quiet standing (average and SE displayed; asterisk represents $p \le 0.05$).

4.4.1.1.2 Effect of Time

Please refer to section 4.1.1.1 to see the effect of time on FRP.

4.4.1.1.3 Interaction Between Discomfort and Time

A significant interaction between discomfort and time was detected for LES FRP activation (p=0.0004). At all time points LBD developers experienced less amount of relaxation, as seen by the higher EMG values in Figure 26, than non-LBD developers. This was least pronounced at time 1 (0mins).

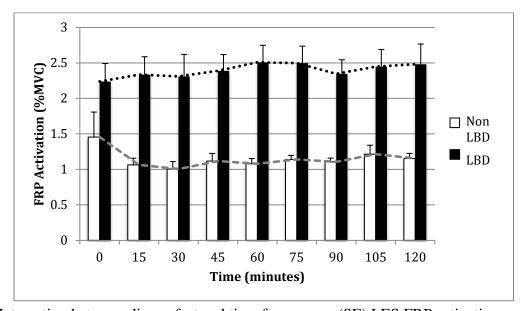


Figure 26: Interaction between discomfort and time for average (SE) LES FRP activation.

4.4.1.2 GM FRP: LBD Developers vs. Non-LBD Developers

Again, once the four non-LBD developers were reintroduced the differences between FRP responses in the non-LBD developers and LBD developers of the GM were analyzed.

Average and standard errors for each of the FRP variables of interest are presented in Table 5 for the GM and categorized by non-LBD developers or LBD developers and p-values for each FRP variable in the GM muscle can be found in Appendix G, Table G4.

Table 5: Non-LBD developers vs. LBD developers GM FRP variable averages (SE)

	Non-LBD	LBD
FRP Variable	Developers	Developers
	Average (SE)	Average (SE)
Onset FRP (%Max. Flexion)	81.32 (3.01)	86.91 (2.76)
Cessation FRP (%Max. Flexion)	89.75 (1.96)	100.04 (2.24)
Peak Flexion Angle	90.81 (1.95)	101.75 (2.22)
(%Max. Flexion)		
FRP Duration (sec.)	7.57 (0.29)	7.11 (0.19)
Average Muscle Activation	2.43 (0.4)	4.23 (0.2)
During FRP (%MVC)		
Peak Activation During Flexion	6.15 (0.59)	16.65 (1.07)
(%MVC)		
Peak Activation During Extension	10.07 (0.83)	21.68 (1.36)
for Flexion (%MVC)		
Average Muscle Activation	2.33 (0.24)	3.82 (0.21)
During Quiet Standing (%MVC)		
FRR	1.12 (0.09)	0.92 (0.04)

4.4.1.2.1 Effect of Discomfort

No significant main effects of discomfort were found for the GM.

4.4.1.2.2 *Effect of Time*

Please refer to section 4.1.2.1 to see the effect of time on FRP in GM.

4.4.1.2.3 Interaction Between Discomfort and Time

Significant interactions were found between discomfort and time in the GM for FRP activation (p <0.0001), GM peak activation during flexion (p=0.03) and GM activation during quiet standing EMG (p=0.033). In all cases LBD developers experienced greater GM muscle activity than non-LBD developers (refer to Figures 27-29).

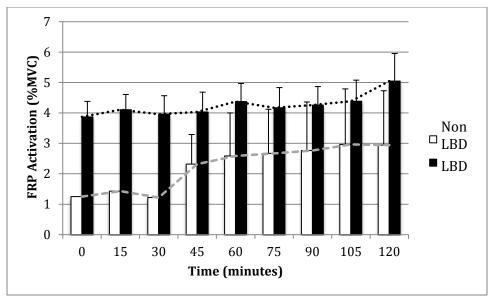


Figure 27: Interaction between discomfort and time for average (SE) for GM FRP activation. (Only one trial reached FRP in the GM for first three time points, error bars not displayed).

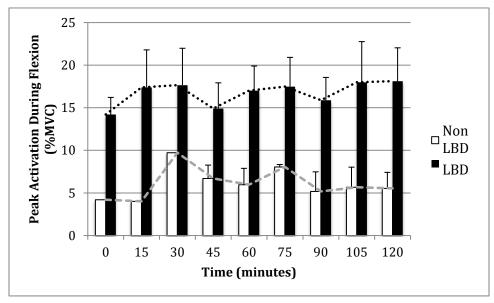


Figure 28: Interaction between discomfort and time for average (SE) GM peak activation during flexion. (Only one trial reached FRP in the GM for first three time points, error bars not displayed).

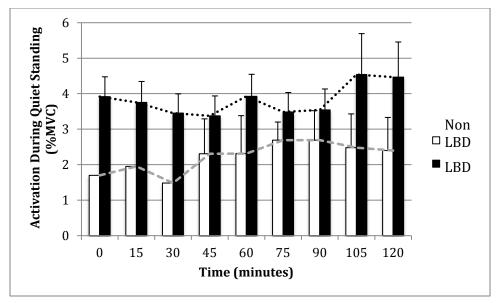


Figure 29: Interaction between discomfort and time for average (SE) GM activation during quiet standing. (Only one trial reached FRP in the GM for first three time points, error bars not displayed).

4.4.2 Occurrence of FRP and LBD

To determine if flexion, and particularly FRP during flexion, significantly influenced low back discomfort development, a chi-square was conducted on the number of occurrences of FRP (yes/no) and the occurrence of LBD (yes/no) on a trial-by-trial basis. This analysis was performed for both the lumbar erector spinae and gluteus medius muscles. No significance was found for either lumbar erector spinae (p= 0.408) or gluteus medius (p=0.855) (Table 6).

Table 6: Distribution of Chi-Squares

	Lumbar Ere	ector Spinae	Gluteus Medius		
	LBD No	LBD Yes	LBD No	LBD Yes	
FRP No	20	14	33	18	
FRP Yes	75	35	62	31	

4.5 Exit Survey

Eighty-one percent (13/16) of participants believed that flexing alleviated discomfort. When asked if participants were given a long enough period of time to flex forward (i.e. 5 seconds in full flexion), 87.5% agreed (14/16). Sixty-nine percent (11/16) of the participants felt they were given enough flexion opportunities throughout the two-hour protocol and when given the opportunity to express which postures participants would adopt if future discomfort induced by standing were to occur, 56% (9/16) reported flexing their back would be preferred. "Seated rest break" was the most popular posture, with 13 of the 16 participants preferring it (Table 7).

Table 7: Postures reported to alleviate discomfort

Posture to be Adopted	People who would adopt posture (%)
Seated rest break	81.25
Crouching down to the ground	62.5
Extending your back	62.5
Flexing your back	56.25
One leg raised on a leg rest	37.5
Bending to the left and right	31.25
Twisting to the left and right	31.25
Other	Standing with one foot forward Shifting weight Leaning Moving Laying down with legs up at 90°

5.0 Discussion

5.1. Revisiting the Purpose and Hypothesis

The purpose of this study was to determine if trunk flexion would mitigate low back discomfort (LBD) development induced by prolonged standing in previously asymptomatic individuals.

5.1.1 Hypothesis

Intermittent, short duration periods of flexion would cause the flexion relaxation phenomenon (FRP), which would induce rest, in turn decreasing LBD development.

5.2 The Effect of Short Periods of Flexion on Low Back Discomfort Developed During Prolonged Standing

Similar to previous literature (Nelson-Wong et al., 2012; Gallagher et al., 2011; Nelson-Wong & Callaghan 2010; Nelson-Wong et al., 2008; Gregory & Callaghan 2008; Gregory et al., 2006), the control day protocol induced LBD in 75% of the participants. Over time, LBD scores significantly increased with a marked difference noted at 75 minutes, similarly found by Gregory and Callaghan (2008). The control day discomfort ratings were then used to divide participants into the two groups: LBD developers and non-LBD developers. A cutoff of 8mm of LBD during the control day delineated significant discomfort.

Interestingly, for participants who experienced LBD, intermittent short duration trunk flexion was found to alleviate discomfort (an average of 10mm less on a 100mm VAS scale) after 75 minutes of standing. Further, the effectiveness of these flexion periods also seemed to coincide with the onset of significant discomfort during the control day (75 minutes). At every time point beyond 75 minutes of standing, a significant difference was identified between the

control day LBD ratings and post-flexion ratings. As time progressed to the 120-minute time point, a significant difference was found between control day LBD and both pre- and post-flexion LBD ratings. It is likely that small micro breaks induced through flexion allowed muscle rest to occur, as these flexion periods were found to induce the flexion relaxation phenomenon (FRP). Therefore, for those who developed LBD, flexion seemed to be beneficial at alleviating discomfort. However, because no significant differences were found at any point during the protocol between pre and post-flexion discomfort scores, a single bout of flexion did not appear to be enough to significantly alleviate discomfort. Rather, the cumulative effect of multiple short duration trunk flexions, inducing micro breaks over time, did significantly reduce discomfort.

5.3 The Occurrence of FRP During Flexion Throughout Prolonged Standing

As expected, twelve participants were classified as LBD developers and four participants were considered to be non-LBD developers. The FRP response occurred in both LBD developers and non-LBD developers, more closely mirroring healthy populations rather than chronic LBP populations where the FRP response is known to be absent (Colloca & Hinrichs, 2005; Neblett et al., 2003; Mannion et al. 2001; Kaigle et al., 1998; Shirado et al., 1995). In the current study, participants experienced an average of 92.4% muscle silencing in the LES. These findings are similar to that of healthy controls from Kaigle et al., (1998) who experienced 78% silencing of the trunk musculature. Echoing previous research (Horn & Bishop 2013; Nelson Wong et al., 2010), results from the current study indicate that the FRP response does occur in individuals who acutely develop low back discomfort.

5.4 The Relationship Between FRP Occurrence and Low Back Discomfort Development

Although all participants experienced FRP, interestingly, LBD developers experienced higher muscle activation than non-LBD developers at multiple phases of FRP. Notably, higher muscle activation was found during both the flexion and extension phases of the flexion trials for the LES, as well as during quiet standing. However most importantly, activation levels of both LES and GM were higher during the relaxation phase of FRP. Higher muscle activation during two hours of standing is typical in people who experience LBP (Nelson-Wong & Callaghan, 2010; Gregory & Callaghan 2008; Gregory et al., 2008; Nelson-Wong et al., 2008; Veirested et al., 1990), mirroring findings in the current study.

As previously stated, due to the occurrence of FRP, the overall population in the current study seems to resemble a healthy population. However, the increased muscle activation during FRP in LBD developers may actually indicate a slow progression from a healthy population to a more chronic LBP population in which muscle activity during FRP persists and no relaxation occurs rendering the FRP absent. LBD developers may actually represent a sub-clinical group that may be at risk of developing chronic LBP later in life. Such speculations need to be further tested.

Surprisingly even at 0 minutes, before the standing commenced, LBD developers experienced higher levels of muscle activation at all points (activation during quiet standings, at peak flexion, during FRP and at peak extension) compared to non-LBD developers. It is likely that higher muscle activation is a predisposing or causative factor of LBD development, rather than an effect of pain development or prolonged standing. It is probable that LBD developers experience different muscle recruitment patterns than non-LBD developers, which may necessitate the need for additional rest breaks.

A commonly accepted theory "The Cinderella Theory" may help to explain the different motor unit recruitment patterns of LBD developers. The Cinderella Theory postulates that low threshold motor units in type I muscle fibres are continually active during long duration exertions and are inefficient at recruiting additional motor units upon overuse (Thorn et al., 2002; Kitahara et al., 2000; Hagg, 1991). It seems reasonable to believe that LBD developers may be subject to the Cinderella hypothesis or a similar effect, comprising more inefficient type 1 muscle fibres that are active for a longer period of time and unable to solicit support from additional motor units, thus fatiguing the muscles and inducing a greater amount of pain. Nelson-Wong and colleagues in 2008 also suggested that higher co-activation of the GM should be considered a casual effect of discomfort development, supporting the current findings.

Finally regardless of FRP, the simple act of flexion has been shown to relieve compression on the apophyseal joints of the vertebrae (Adams & Hutton, 1980) and increase transport of disc metabolites (Adams & Hutton, 1986). Most commonly adopted postures put the spine in some degree of flexion reducing lumbar lordosis (Dolan et al., 1988); these findings may provide additional rational for the decrease in discomfort associated with flexion in the current study.

5.4.1 Did FRP Induce Rest?

The increased muscle activation found in LBD developers as compared to non-LBD developers suggests that LBD developers experienced less muscle relaxation, which could be the most likely explanation for the higher level of discomfort reported. It is plausible that the greater myoelectric silencing seen in both the LES and GM in the non-LBD developer group provided a transient period of rest for the LES and GM thereby potentially decreasing muscle fatigue and subsequent LBD development. Co-activation of the left and right GM muscles is a prominent

finding in LBD developers (Nelson-Wong 2t al., 2008); the opportunity for rest induced through FRP in the GM may be even more beneficial for LBD-developers for this reason. It is likely that the transient rest initiated through FRP relieved the muscles of their moment supporting roles, providing muscle relaxation.

5.5 Notable Findings in FRP

Similar to previous research (Nelson-Wong et al., 2010), the current study found minimal changes to the FRP response in the LES after prolonged exposure to standing. Paralleling previous research, the current study's results are within the previously documented ranges of FRP onset. In the current study, average FRP onset occurred at 93.2% of maximum flexion. Previous literature has documented average FRP onset to have occured between 79.93% max flexion and 92.7 ± 5.8% max flexion (Schinkel-Ivy et al., 2014; Howarth et al., 2013; Callaghan & Dunk, 2002).

5.5.1 Changes in FRP Over Time

5.5.1.1 Change in LES FRP Over Time

Limited information exists in the literature concerning the FRP response in the LES over time. Of the research that is available, contradictory findings exist. In seated FRP, one-hour of sitting has shown to increase FRP onset angle (Howarth et al., 2013). However, following two hours of standing, Nelson- Wong et al., (2010) found no significant changes in the flexion relaxation ratio. In the current study, cessation angle, max flexion, peak EMG activation during flexion, and the FRR all remained consistent over 2 hours of standing, with no main effect of time or interaction between sex and time. Although main effects of time were found for the onset

angle for FRP and the quieting duration indicating a change over two hours, it is unlikely that these findings had any biological significance as no clear patterns emerged (i.e. gradual increase or decrease over the two hours). The patterns observed were more random in nature, unlikely to hold any significant importance. As such the hypothesis that muscle quiescence would occur at a greater point of flexion over time was rejected; although muscle quiescence was affected by time, no pattern was observed.

5.5.1.2 Changes in GM FRP Over Time

The FRP response has been shown to occur in the lumbar (Schinkel-Ivy et al., 2014; Howarth et al., 2013; Descarreaux et al., 2008; Callaghan & Dunk, 2002) and thoracic erector spinae muscles (Dickey et al., 2003; Callaghan & Dunk, 2002), gluteus maximus (Nelson-Wong et al., 2012), and hamstrings (Olson et al., 2006). To the authors knowledge this is the first time FRP in the gluteus medius has been reported. Similar to FRP in the LES, little changes to FRP in the GM occurred over time. GM FRP onset angle, quieting duration, max flexion angle, peak activation during flexion, peak activation during extension from flexion and FRR all remained consistent with no main effects of time or interactions between sex and time observed over two hours of standing. However, average GM activation during FRP, activation during quiet standing and FRP cessation angle each presented statistically significant interactions between sex and time. Similar to the LES, it is unlikely that there are any biologically significant changes over time, as no clear patterns emerged.

5.5.1.3 Changes in CoP Over Time

The effect of time also proved insignificant when considering CoP excursion. As hypothesized, CoP moved anteriorly during FRP and no significant changes were found over time.

5.5.3 Sex Differences in the FRP Response

Though a significant main effect of sex in the LES at activation during extension was found in addition to significant interactions between sex and time in the LES and GM, few consistent patterns emerged. For this reason, though statistically significant, sex differences were not believed to be of great importance. However, males did experience lower LES and GM muscle activation overall.

5.6 Ergonomic Applicability

Determining the effectiveness of trunk flexion in workstation design is of primary importance. A 10mm (using a 100mm VAS scale) average decrease in discomfort scores was identified from control days to flexion days. Ultimately, participants perceived that short, intermittent periods of trunk flexion alleviated discomfort during prolonged standing. By placing the spine in a flexed position seated breaks (Gallagher et al., 2014) and standing on sloped surfaces (Nelson-Wong & Callaghan, 2010; Gallagher et al., 2013) have been shown to effectively decrease pain development. Likewise, purposeful flexion of the spine during standing, as seen in the current study, significantly alleviated LBD induced from standing. Intermittent transient trunk flexion during prolonged standing is a quick, easy-to-perform and cost-effective mechanism to potentially mitigate LBD. Further, the cumulative effect of multiple flexion trials has the ability to prevent discomfort from worsening. However, it is apparent that trunk flexion may be more beneficial for some individuals than others. If a person does not experience LBD during prolonged standing, trunk flexion does not seem to significantly benefit

the person. Even though the majority of the non-LBD developers rated their discomfort lower post-flexion, these participants' discomfort scores were not considered to be at clinical levels, rendering flexion as a potential intervention unnecessary for those individuals. Therefore, for people who are prone to experiencing discomfort, transient trunk flexion can be used in workplaces as a means to reduce low back discomfort induced by prolonged standing.

5.7 Considerations and Future Directions

5.7.1 Considerations

There are a number of points to consider for the current study. First, because of the amount of data collected, it was not feasible to analyze the muscles bilaterally. Therefore only the right side of the body was analyzed. It is possible that muscle asymmetry exists between the left side and right side of the body; such differences would not have been identified in the current analysis. However, previous work (Nelson- Wong et al., 2012; Nelson-Wong et al., 2010; Dankaerts et al., 2006) found no differences between the left and right side musculature in the FRP response.

The study was also limited in that muscle activation, spine posture and CoP was not monitored during the two hours of standing, only during the flexion trials. Therefore, changes during the actual periods of standing could not be determined; this however has been extensively examined (Gallagher et al., 2014; Mika et al., 2013; Nelson-Wong et al., 2012; Gallagher et al., 2011; Nelson-Wong and Callaghan, 2010; Lafond et al., 2009; Gregory et al., 2008; Gregory & Callaghan, 2008; Nelson-Wong et al., 2008; Gregory et al., 2006; Orlando & King, 2004; Pascal, van Dieen et al., 2003; Duarte & Zatsiorsky, 1999; Voigt, & Arendt-Nielsen, 1998) and was not

within the scope of the current project. Because a great amount of research has examined the FRP response, numerous procedures have been documented to analyze FRP data. In this study, visual inspection, a threshold and a ratio where used to confidently identify FRP (Schinkel-Ivy et al., 2013). When determining FRP threshold analysis, FRP has previously been calculated by creating a new threshold for each trial. In the current study a participant-specific base level threshold of activation during FRP (relaxation) was determined based on the first FRP trial of each participant. That threshold was then used in all subsequent FRP trials to determine if any trial-to-trial changes had occurred in the FRP response. Both trial-to-trial and baseline thresholds were examined statistically and no notable differences were observed between the two thresholds (Refer to Appendix G, Table G5). The use of the baseline threshold is unique to the author and additional research should follow to further examine the trial-to-trial variation within participants.

Additionally, though the minimum clinically important difference for an increase in VAS scores in the low back is 8mm (Hägg et al., 2003), it should be acknowledged that 8mm is a conservative cut-off to use when identifying non-LBD developers and LBD developers. Before choosing the 8mm cut-off a threshold of 10mm was used (which moved three individuals from the LBD-developer group to the non-LBD developer group) to classify the participants, the same statistical analysis was run and no significant differences were found between the two VAS thresholds. Ultimately, the 8mm VAS cut-off was chosen because it best depicted the current data set.

In regards to digital filtering, it is important to note that all digital filters are limited in their ability to completely remove 100% of the power at the chosen cut-off frequency. Therefore some power would have remained in the signal even above the cut-off frequency.

Finally, a preliminary questionnaire to examine participants' perceptions of pain development (i.e. attitudes towards pain development, injury or disability) would have been beneficial to determine if such perceptions influenced discomfort responses.

5.7.2 Future Directions

While this study has sought to answer a number of questions, many more questions have emerged. Future research should compare numerous spine flexion postures such as full flexion, crouching down to the ground, one leg raises and various degrees of seated postures to determine which best alleviates discomfort.

As more research continues to examine standing FRP over time, it would be interesting to explore the possibility of creating an intrapersonal threshold for each participant that reflects changes in FRP over time. Using the average activation during FRP across all trials to determine an appropriate threshold, rather than using the first trial as seen in the current study, may better depict changes over time.

5.7.2.1 Recommendations

The FRP response can be induced through short duration, intermittent trunk flexion during prolonged standing to alleviate acute LBD due to muscular discomfort. More specifically, maintaining forward trunk flexion for 5 seconds every 15 minutes can decrease the development of standing-induced discomfort. For people who suffer from intervertebral disc herniations or any other spine disorders, trunk flexion is not recommended. Additionally, chronic LBP populations are not advised to stand for prolonged periods of time, and therefore trunk flexion would have little value for such a population. Similarly, flexion may not be useful for people who do not experience LBD induced from standing.

6.0 Conclusion

The current study examined FRP during intermittent, short duration trunk flexion as a mechanism to alleviate LBD induced through two hours of prolonged standing. FRP occurred in both non-LBD developers and LBD developers, with little changes occurring over time. For people who experienced LBD, ratings of discomfort significantly decreased over time when FRP occurred. It is thought that transient muscle rest induced through FRP is responsible for the decreased discomfort. For those who did not experienced discomfort (less than 8mm on the VAS), LES activation during FRP was significantly less than those who experienced discomfort, suggesting that greater muscle rest occurred in non-LBD developers. Flexion was not effective at mitigating discomfort after a singular bout of flexion, however the cumulative effect of intermittent, short duration trunk flexions over time significantly reduced discomfort, most notably after 75 minutes of prolonged standing. Therefore, transient lumbar flexion of the spine seems to effectively mitigate LBD induced by prolonged standing.

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APPENDIX A

A1: General Questionnaire

1.	What is your gender (please circle)? Male Female
2.	What is your height (feet/inches)?
3.	What is your weight (pounds)?
4.	What is your age?
5.	How often do you participate in moderate-to-vigorous physical activity per week, lasting 30 minutes or more? (Please circle one). a. 1 day/week b. 2-4 days/week c. 5 days/week d. 6-7 days/week
6.	How often do you stand for longer than 15 minutes at a time? a. Once weekly b. 2+ weekly c. Daily d. Multiple times a day
7.	Do you tend to experience lower back pain after you stand for a prolonged period of time? (Please circle one). Yes No a. If you answered "yes" please describe how long you stand before the pain occurs.
	If you were to experience low back pain after standing for a prolonged period of time, would you use any of the following methods to alleviate the discomfort? (Please circle all that apply). a. Seated rest break b. One leg raised on a leg rest c. Twisting to the left and right d. Bending to the left and right e. Flexing your back f. Extending your back g. Crouching down to the ground h. Other (please describe)
9.	Do you have a history of chronic or clinical Low Back Pain (LBP) (please circle one)?
	Yes No

APPENDIX B

B1: Ratings of Perceived Discomfort (VAS)

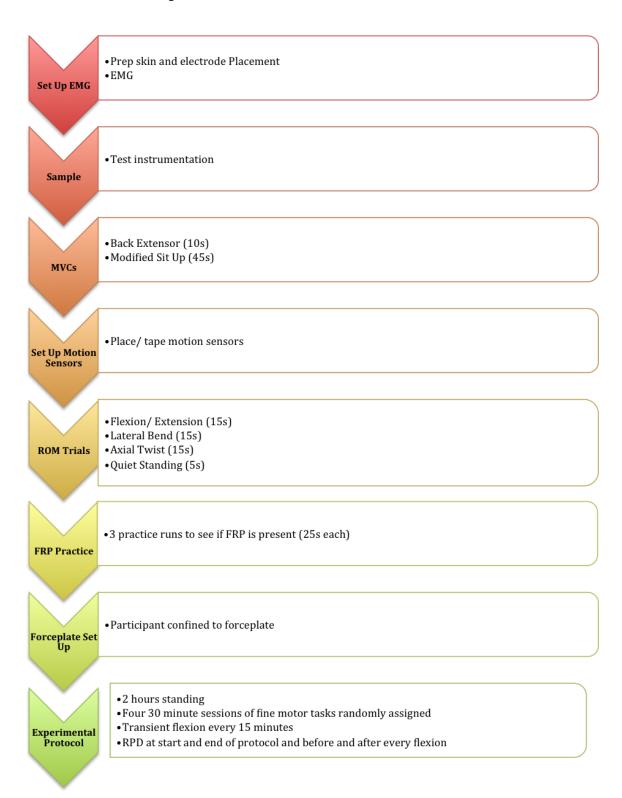
RATING OF PERCEIVED DISCOMFORT SCALE

Part A: Please make a mark on the line that corresponds to the level of discomfort you feel, reflecting your CURRENT state of discomfort in each of the following areas:

$\mathcal{S}_{\mathcal{L}}$	No Discomfort At Al	I Worst Discomfort Imaginable
Norl	Head-Neck	
[#\\K\	Shoulders	
1/4/3	Upper Back	
)<(><	Lower Back	
$\backslash M /$	Legs-Feet	
B		
BACK		
No D	iscomfort At All	Worst Discomfort Imaginable
	be the level of your current BA	rresponding to the descriptive words CK DISCOMFORT. You may include
Tiredness	Dull	
Soreness*	Localized	
Numbness	* Distributed	prophysical foreign per a foreign per una comprehensi a foreign per a foreign per a foreign per a foreign per p
Sharp	Painful	

APPENDIX C

C1: Outline of the Experimental Protocol



APPENDIX D

h. Other (please describe)

D1: Exit Survey						
Participant of	code:					
Date of coll	ection:	 -				
10. Would	•	der the pro	otocol to be disc	comforting?	Please circ	ele one.
11. Do yo	u believe fl	exing forv	ward relieved ar	ny discomfor	t that you r	nay have
experi	enced durii	ng the prot	tocol? Please ci	rcle one.	Yes	No
12. Do yo	u believe y	ou were g	iven a long eno	ugh period of	f time to flo	ex forward? Please
circle	one.	Yes	No			
13. Do yo	13. Do you believe there were enough flexion periods during the protocol? Please circle			ocol? Please circle		
one.		Yes	No	•		
14. If you	experience	low back	discomfort afte	er standing fo	r a prolon	ged period of time,
•	-		ollowing metho	_		
	all that app	-	C			`
	Seated res	=				
b. One leg raised on a leg rest						
c. Twisting to the left and right						
	Bending t		_			
	Flexing y		411G 11G11t			
f.	Extending		k			
	_	-				
g.	g. Crouching down to the ground					

APPENDIX E

Apparatus and Set-up

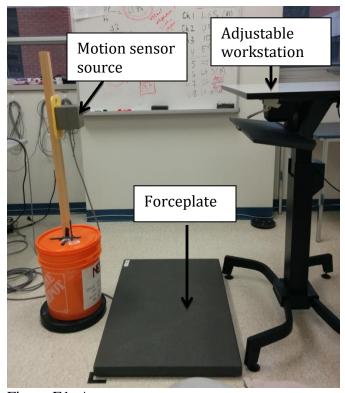


Figure E1: Apparatus

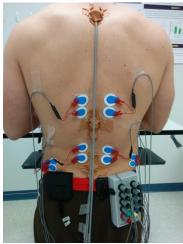


Figure E2: Electrode and motion sensor set up

APPENDIX F

MCVs & ROM



Figure F1: Back MVC- Participant extended their back until horizontal and parallel to the ground. The research assistant then resisted a maximum back extension.

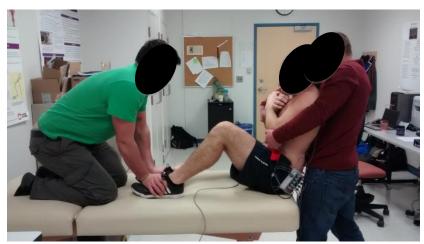


Figure F2: External Oblique MVC- Resisted sit up, twists and lateral bends.



Figure F3: Gluteus Medius MVC. Participants lay on one side, lifted leg while research assistant resisted leg abduction. Participants performed the MVC for right and left legs.



Figure F4: ROM back extension

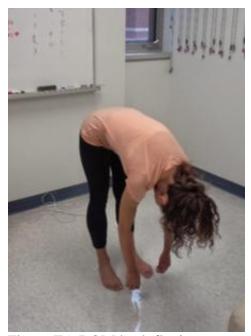


Figure F5: ROM back flexion



Figure F6: Quiet standing

APPENDIX G

FRP

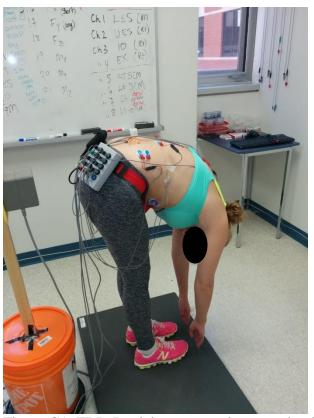


Figure G1: FRP. Participants were instructed to bend as far as they could forward letting their head and arms dangle freely.

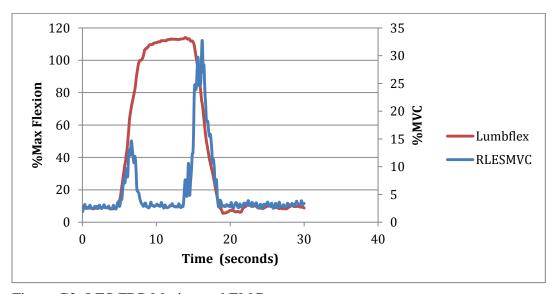


Figure G2: LES FRP Motion and EMG

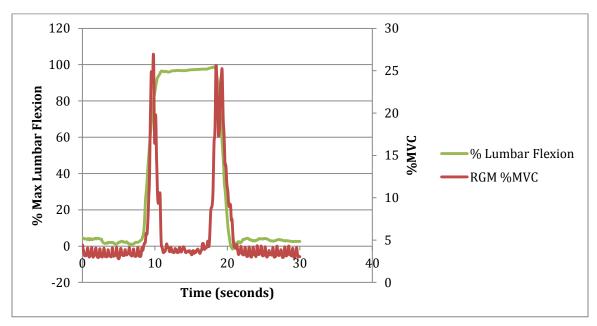


Figure G3: GM FRP Motion and EMG

Table G1: P-values for LES FRP variables of interest. Asterisk represents p <0.05.

FRP variable	Sex	Time	Sex x Time
Onset FRP (%Max. Flexion)	0.884	0.007*	0.869
Cessation FRP (%Max. Flexion)	0.480	0.115	0.401
FRP Duration (sec)	0.887	0.02*	0.388
Average Muscle Activation During FRP (%MVC)	0.06	0.069	0.0018*
Peak Flexion Angle (%Max. Flexion)	0.522	0.139	0.340
Peak Activation During Flexion (%MVC)	0.093	0.457	0.253
Peak Activation During Extension from Flexion (%MVC)	0.012*	0.088	0.0005*
Average Muscle Activation During Quiet Standing (%MVC)	0.068	0.073	0.036*
FRR	0.763	0.118	0.142

Table G2: P-values for GM FRP variables of interest. Asterisks represents p <0.05.

FRP variable	Sex	Time	Sex x Time
Onset FRP (%Max.	0.571	0.549	0.876
Flexion)			
Cessation FRP	0.265	0.233	0.029*
(%Max. Flexion)			
FRP Duration (sec)	0.987	0.132	0.567
Average Muscle	0.58	< 0.0001*	0.006*
Activation During			
FRP (%MVC)			
Peak Flexion Angle	0.368	0.551	0.194
(%Max. Flexion)			
Peak Activation	0.334	0.869	0.526
During Flexion			
(%MVC)			
Peak Activation	0.296	0.098	0.38
During Extension			
from Flexion			
(%MVC)			
Average Muscle	0.988	< 0.0001*	<0.0001*
Activation During			
Quiet Standing			
(%MVC)			
FRR	0.239	0.384	0.967

Table G3: P-values for LES FRP variables of interest, discomfort by time. Asterisk

represents p < 0.05.

represents p <0.03.			
FRP variable	Discomfort	Time	Discomfort x Time
Onset FRP (%Max.	0.842	0.004*	0.255
Flexion)			
Cessation FRP	0.585	0.099	0.187
(%Max. Flexion)			
FRP Duration (sec)	0.750	0.03*	0.921
Average Muscle	0.007*	0.179	0.0004*
Activation During			
FRP (%MVC)			
Peak Flexion Angle	0.609	0.124	0.191
(%Max. Flexion)			
Peak Activation	0.004*	0.492	0.610
During Flexion			
(%MVC)			
Peak Activation	0.009*	0.178	0.164
During Extension			
from Flexion			
(%MVC)			
Average Muscle	0.004*	0.083	0.079
Activation During			
Quiet Standing			
(%MVC)			
FRR	0.469	0.135	0.332

Table G4: P-values for GM FRP variables of interest, discomfort by time. Asterisks represent p < 0.05.

FRP variable	Discomfort	Time	Discomfort x Time
Onset FRP (%Max.	0.753	0.559	0.944
Flexion)			
Cessation FRP	0.475	0.370	0.910
(%Max. Flexion)			
FRP Duration (sec)	0.667	0.160	0.944
Average Muscle	0.232	< 0.0001*	<0.0001*
Activation During			
FRP (%MVC)			
Peak Flexion Angle	0.469	0.627	0.879
(%Max. Flexion)			
Peak Activation	0.364	0.814	0.03*
During Flexion			
(%MVC)			
Peak Activation	0.099	0.151	1.00
During Extension			
from Flexion			
(%MVC)			
Average Muscle	0.327	0.0004*	0.033*
Activation During			
Quiet Standing			
(%MVC)			
FRR	0.424	0.292	0.189

Table G5: Differences between baseline FRP threshold and current FRP threshold. Averages for the FRP variables that pertain to threshold calculations are identified below. Variables at time point 9 (120 minutes) are depicted, as the largest difference would occur between time point 1 (0 minutes) which was used for baseline threshold, and time point 9.

	FRP Variable	Baseline Threshold	Current Threshold
	Threshold Value	3.25	4.09
	(%MVC)		
	Onset FRP (%Max.	89.19	88.72
Time Point 9 (120	Flexion)		
minutes)	Cessation FRP	99.69	99.67
	(%Max. Flexion)		
	FRP Duration (sec.)	6.9	7.14
	Average Muscle	2.14	2.18
	Activation During		
	FRP (%MVC)		

Table G6: The number of times each participant reached FRP (out of nine) across the two hours of standing.

Participant	Number of Times FRP was Achieved
1	9
2	5
3	8
4	9
5	3
6	3
7	7
8	9
9	9
10	8
11	1
12	7
13	9
14	7
15	9
16	9

APPENDIX H

Discomfort Ratings for Additional Body Areas

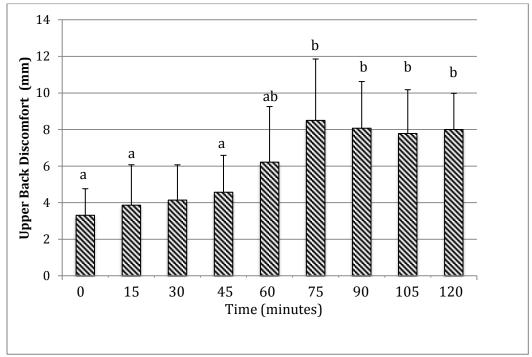


Figure H1: Main effect of time, average (SE) discomfort for upper-back during control day (p= 0.0006). Different letters represent significant differences in discomfort ratings determined via post-hoc analysis.

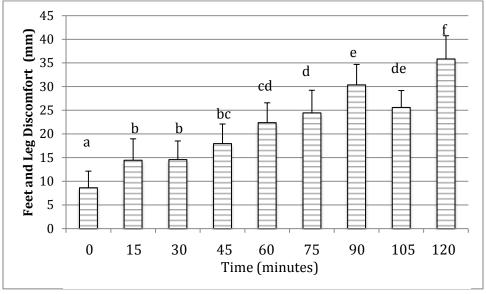


Figure H2: Main effect of time, average (SE) discomfort for feet and legs during control day (p<0.0001). Different letters represent significant differences in discomfort ratings determined via post-hoc analysis.

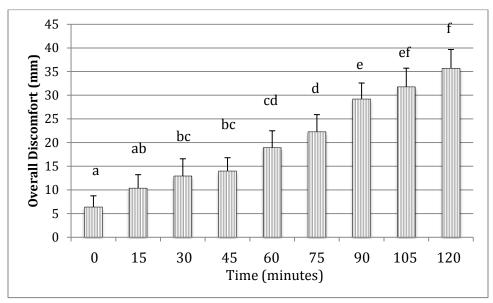


Figure H3: Main effect of time, average (SE) discomfort for overall body during control day (p<0.0001). Different letters represent significant differences in discomfort ratings determined via post-hoc analysis.

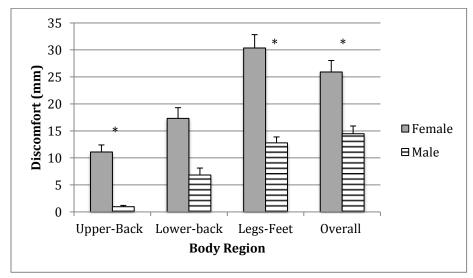


Figure H4: Main effect of sex, average (SE) discomfort ratings for each body region during control day. Lower back discomfort (p=0.06) upper back discomfort (p<0.01), legs and feet discomfort (p=0.02) and overall discomfort (p<0.0001). Asterisk represents significant differences.

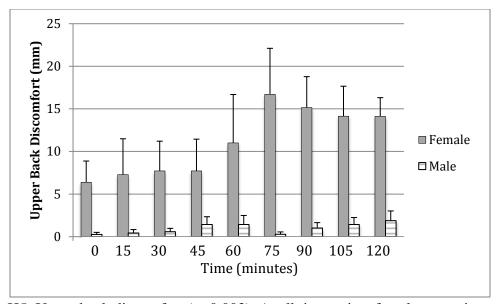


Figure H5: Upper back discomfort (p=0.003). At all time points females experienced significantly greater discomfort than males, but most notably in 75mins (16.7mm, SE 5.39; 0.29mm, SE 0.27 respectively).