An Investigation of the Flexion Relaxation Ratio in Adults with a Self-Reported History of Low Back Pain and Transient Sitting-Induced Pain.

Written by © Sarah Mackey. A Thesis submitted to the School of Graduate Studies in partial fulfillment on the requirements for the degree of MSc. in Medicine (Clinical Epidemiology)

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Abstract

Background: Transient, sitting-induced back pain is commonly reported in response to prolonged sitting. Since pain may confound spine function outcomes, this investigation aimed to determine whether the flexion relaxation ratio (FRR) was affected by sitting-induced pain development or clinical history.

Methods: 47 participants, aged 18-69, with (n = 24) and without a history of non-specific low back pain (n = 23), were exposed to 1-hour of sitting. Perceived pain ratings were taken throughout the trial. Surface electromyography was collected bilaterally over the lumbar erector spinae, and lumbar angle was measured by accelerometers. Participants performed a maximal trunk flexion trial immediately pre/post and the FRR was calculated.

Results: There were no significant interactions or differences in the FRR between time (pre/post), clinical history (+/-ve low back pain), or pain development status (pain developer/non-pain developer).

Conclusions: 1-hour of sitting-induced pain and a clinical history of low back pain does not appear to induce changes in the motor control of a forward bending task, and in turn, the magnitude of the FRR. Therefore, neither sitting-induced pain or history of low back pain are likely to confound this outcome measure for sitting exposures less than 1-hour.

General Summary

Sitting-induced low back pain (LBP) is often reported by the working population. It has been shown that this pain is not an aggravation of a previous history of LBP as it is reported equally amongst individuals with and without a history of LBP. The confounding effects of this pain are often not considered amongst studies that measure spine function outcomes after sitting interventions. The purpose of this thesis was to determine whether sitting-induced pain development (pain developers/non-pain developers) and a self-reported history of LBP (+ve history /-ve history), differentially affects an outcome of spine function, the flexion relaxation ratio (FRR). The results of this study indicated that for one-hour of sitting, the FRR is not differently altered based on a history of LBP and pain development status. Consequently, studies that used a sitting-intervention of one-hour and did not control for sitting-induced LBP may not have confounded results.

Land Acknowledgement

We respectfully acknowledge the territory in which we gather as the ancestral homelands of the Beothuk, and the island of Newfoundland as the ancestral homelands of the Mi'kmaq and Beothuk. We would also like to recognize the Inuit of Nunatsiavut and NunatuKavut and the Innu of Nitassinan, and their ancestors, as the original people of Labrador. We strive for respectful relationships with all the peoples of this province as we search for collective healing and true reconciliation and honour this beautiful land together.

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List of Abbreviations

EMG: Electromyography

FRP: Flexion relaxation phenomenon

FRR: Flexion relaxation ratio

LES: Lumbar erector spinae

LLES: Left lumbar erector spinae

L1: First lumbar vertebrae

MVC: Maximum voluntary contraction

NPD: Non-pain developers

NSLBP: Non-specific low back pain

PD: Pain developers

RLES: Right lumbar erector spinae

ROM: Range of motion

sEMG: Surface electromyography

SIP: Sitting-induced pain

S2: Second sacral vertebrae

VAS: Visual analog scale

+veHx: History of low back pain

-veHx: No history of low back pain

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Chapter 1: Introduction

General Introduction

Back pain is a debilitating, common condition that up to 80% of the population is estimated to experience at least once in their lifetime (Rubin, 2007). Occupational populations have been reported to have especially high rates of back pain as they are less likely to take time off work and seek treatment (Al-Otaibi, 2015). The indirect economic burden of low back pain due to missed work and reduced work capacity is estimated to be as high as 28.2 million dollars in the United States alone (Dagenais et al., 2008). Both the economic and the physical burdens of back pain on the work force are causes for further investigation into the mechanism/cause and possible treatments.

The etiology of back pain is predominantly unknown and is characterized by both acute and chronic durations. Back pain can be caused by specific structural abnormalities of the spine such as injury through fracture or misalignment, known as specific back pain. However, non-specific causes can be just as common and can present with the same symptoms. Chronic non-specific back pain can be especially difficult to treat as the specific mechanism is unknown and cannot be fixed through targeted therapy such as surgery. This type of back pain is commonly experienced by individuals in the work force, as workers have been reported to spend on average, 10 hours per day seated (McCrady & Levine, 2009). Sitting time has been implicated to be a risk factor for low back pain (Jackson et al., 2016).

In occupational populations specifically, the shift to prolonged seated deskwork amongst workplaces has been speculated to be a risk factor in the development of low back pain. The relationship between prolonged sitting and low back pain is unclear as it is not known whether the relationship is of a causal nature or purely association. Regardless, prolonged sitting, which is extremely prevalent amongst occupational populations, is associated with various health risk such as all-cause mortality (Benatti & Ried-Larsen, 2015), some cancers (Rangul et al., 2018), and type II diabetes (Åsvold et al., 2017).

Sitting-induced pain

In addition to the above health risks, it has been commonly reported that prolonged sitting can cause bouts of back pain for some individuals (De Carvalho et al., 2020; Greene et al., 2019). Anecdotally, this posture-induced low back pain has been reported to be predominantly transient and has been said to subside quickly after standing. Sitting-induced back pain is different from chronic non-specific low back pain as it has been found to have no relation to a previous experience of low back pain (Greene et al., 2019). Consequently, this sitting-induced pain is not believed to be an aggravation of a previous clinical history of low back pain. A study by Greene et al. (2019) reported that there was an equal reporting of sitting-induced back pain between individuals with and without a history of low back pain. This indicates that regardless of clinical history, sitting may cause back pain in individuals who are considered otherwise 'healthy'.

This sitting-induced pain is important to consider as it may affect some aspects of spine function. Commonly, amongst studies that measure some aspect of spine function before

and after an intervention of prolonged sitting, the confounding effects of sitting-induced pain are not considered. Although patients that report experiencing NSLBP are typically excluded from participating in these investigations, the included 'healthy' participants may develop pain while seated, regardless of the exclusion of participants with a previous history. This may confound the results of the study as alterations in motor control can be present amongst those who experience pain.

One of the confounding effects of pain during sitting is alterations in muscle activation. Commonly, individuals who are experiencing pain, exhibit greater levels of muscle activation and less relaxation compared to healthy individuals (Dankaerts et al., 2017; Watson, Booker, Main, et al., 1997). A study by Kaigle et al., determined that individuals with back pain were only able to relax their muscles by 13%, compared to healthy individuals that were able to reduce their muscle activation by 78% (Kaigle et al., 1998). This constant, heightened muscle activation is commonly referred to as 'guarding.' It is an alteration in motor control to effectively increase the control of the spine, as the spine is considered destabilized while experiencing back pain (Ahern et al., 1988). Guarding ensures that the muscles do not relax and lose stability, to prevent further injury to spinal tissues.

An aspect of spine function that may be altered with the changes in muscle activation with pain is the flexion relaxation phenomenon (FRP). The FRP is a biomechanical phenomenon that occurs at end-range of trunk flexion, where the low back muscles 'turn off'. It is speculated that the low back muscles turn off when the passive tissues of the

spine are able to withstand the load of the torso, allowing the muscles to turn off (Floyd & Silver, 1955). However, amongst individuals experiencing low back pain, the muscles do not relax and turn off due to guarding. Consequently, the FRP is commonly absent amongst individuals who experience low back pain**.** Shirado and colleagues reported that all participants enrolled in their study that were experiencing an episode of chronic low back did not exhibit the FRP compared to all their participants without low back pain that did exhibit the phenomenon (Shirado et al., 1995). Logically, it is of interest to investigate whether the FRP, specifically using the flexion relaxation ratio, is altered due to the presence of sitting-induced low back pain.

Due to the predominate absence of the FRP amongst individuals with low back pain, the flexion relaxation ratio (FRR) has been commonly used to quantify the FRP amongst this population. The FRR is the ratio of muscle activation during the bending movement of trunk flexion to the muscle activation at full flexion. Since this ratio is sensitive to changes in muscle activation, it is of interest to determine whether this outcome of spine function is different for individuals based on whether they experience sitting-induced pain. Additionally, it is of interest to determine whether a history of low back pain also has an effect on the FRR. Consequently, the purpose of this investigation is to determine whether an outcome of spine function, specifically the flexion relaxation ratio, is different for subgroups of participants according to their history of low back pain and/or status as a pain developer in response to a 1-hour exposure of seated deskwork (i.e., PD-Hx+, PD-HX-, NPD-Hx+, NPD-Hx-).

This manuscript is divided into five chapters. The first chapter consists of the general introduction, purpose, and research questions. The second chapter contains the literature review. The third chapter is the manuscript, submitted to the Journal of Electromyography and Kinesiology, titled "An Investigation of the Flexion Relaxation Ratio in Adults with History of Clinical Low Back Pain and Transient Sitting-Induced Pain." The fourth chapter contains a general discussion on the outcomes and implications of the investigation, and the fifth chapter is the conclusion.

Purpose:

The primary purpose of this investigation is to assess the effect of pain development during sitting and self-reported low back pain have on an outcome of spine function, the flexion relaxation ratio.

Research Questions:

General question: Do individuals with a history of non-specific low back pain and/or transient sitting-induced low back pain exhibit different flexion relaxation ratios?

Specific questions:

(1) Do individuals who have experienced at least one episode of chronic non-specific back pain in the past six months exhibit different flexion relaxation ratios compared to back-healthy individuals?

(2) Do individuals who develop transient sitting-induced low back pain from 1-hour of prolonged sitting exhibit different flexion relaxation ratios compared to back-healthy individuals?

Hypotheses:

(1) **Null Hypothesis ($H_0 = 0$):** There is no difference in FRR between individuals with a history of low back pain (+veHx) and those without a history of self-reported LBP (-veHx).

(2) **Null Hypothesis ($H_0 = 0$):** There is no difference in FRR between individuals showing a sitting-induced pain response (PD) and those that do not develop pain (NPD)

(3) **Alternative Hypothesis ($H_0 \neq 0$):** There is a significant difference in the FRR between PD and NPD and/or between individuals with a history of low back pain and without a history of low back pain.

Chapter 2: Literature Review

2.1 Overview of Low back pain

2.1.1 Low back pain epidemiology

Low back pain is extremely prevalent globally as it is estimated that the cumulative lifetime prevalence of low back pain is 13.8% and the mean lifetime prevalence is 38.9% (Deyo & Yuh-Jane, 1987; Hoy et al., 2012). It has been suggested that up to 80% of the population will experience some form of low back pain in their lifetime (McPhee & Papadakis, 2009). This condition affects individuals of all ages, ethnicities, and occupations. Globally, it is the leading cause of years-lived with disability (Wu et al., 2020). The resulting disability can result in a variety of consequences for the sufferer such as lost time from work, financial concerns, or even social/societal exclusion. In recent years, there has been a steady increase in low back pain prevalence, proposed to be due to the increase in global population, specifically the aging portion (Hoy et al., 2010).

2.1.2 What is low back pain?

Low back pain is defined as the experience of pain between the inferior rib and the gluteal sulcus, lasting for three months minimum (Dionne et al., 2008). Low back pain is not classified as a disease; it is a symptoms of, in most cases, an unknown origin (Hartvigsen et al., 2018). This pain can be chronic, acute, or repetitive in nature. The source of this

pain is usually unable to be pinpointed as it can have a variety of origins or interactions: biophysical, psychological, or social. Consequently, most low back pain is classified as non-specific low back pain. However, specific causes of low back pain may also be diagnosed (<15% of low back pain cases) and can be the result of many conditions such as disc herniation, vertebrae fracture, or tumours (Airaksinen et al., 2006). Diagnostic tests and imaging can be used to further investigate the origin of the pain but since many complicated structures are involved, any common structural abnormality could be incorrectly proposed to be the cause. Low back pain symptoms have been found to not be correlated with imaging findings (Lateef & Patel, 2009) so imaging has been commonly discouraged for diagnostic purposes. Currently, most clinical guidelines do not recommend routine imaging for cases of suspected non-specific back pain (Chou et al., 2011). However, it is still common practice amongst many general practitioners to refer patients with a complaint of low back pain for imaging for various reasons that include a lack of awareness and/or adherence to clinical guidelines to patient expectations (D. De Carvalho et al., 2021).

The interpretation of pain is largely subjective and involves an interplay of many factors: genetic susceptibility, mood, psychological state, pain processing, previous experiences of pain, and more (Sanzarello et al., 2016). Consequently, it is very difficult, potentially impossible, to pinpoint the origin/cause of back pain and requires a broad perspective; not solely focused on anatomical or structural abnormalities. Further, the experience of pain can alter the way the central nervous system processes the pain which can further

complicate the investigation and treatment of low back pain. This is called central sensitization.

2.1.3 Pathophysiology of low back pain

The central nervous system (CNS) receives afferent feedback continuously from various peripheral origins. One of the common types of signals the CNS integrates are pain signals, also known as nociceptive signals, that originate from specific nociceptive receptors. The CNS can augment or tone down signals, especially of nociceptive origin, depending on the frequency. If the noxious input is maintained, sensitization (both peripheral and central) may ensue to convert acute pain into chronic pain (Allegri et al., 2016). It is speculated that one of the origins of chronic pain may be an impairment in conditioned pain modulation (Mlekusch et al., 2016). This has consequences for individuals experiencing chronic pain, especially in the case of low back pain, as Mlekusch and Neziri (2016) determined that low back pain patients may be at risk of widespread central hypersensitivity due to impaired conditioned pain modulation.

In a study conducted by Giesecke et al., after exposing patients with chronic non-specific low back pain (CNLBP) and healthy controls to the same level of pressure on the thumbnail, the individuals with CNLBP reported experiencing significantly more pain than the controls (Giesecke et al., 2004). It is speculated that this hypersensitivity is caused by a prolonged noxious input, leading to even healthy tissues generating a noxious signal, rather than solely the acute pain stimuli (Sanzarello et al., 2016). In patients with

central sensitization, the noxious signal sent from the periphery is not proportionate to the experience of pain (Nijs et al., 2015). Consequently, a slight change in posture or small movement, can cause a heightened experience of pain that would not occur normally. For example, periods of sitting could become excessively painful for individuals with CNLBP, as the posture may aggravate their back pain or they may be sensitive to pain during sitting when they otherwise would not be.

2.2 Prolonged sitting

On average, individuals in the working force sit for 10-hours each day: 110 minutes more than during leisure days (McCrady & Levine, 2009). As seated desk work has become a standard mode of working amongst developed countries, workers spend less time active and more time in a reclined, sedentary posture. This time spent sedentary, defined as any seated or reclined behaviour that expends less than 1.5 METs of energy, can become harmful overtime to workers who sit for long periods as it increases their risk of all-cause mortality (Benatti & Ried-Larsen, 2015) and various health issues such as hypertension (Dempsey et al., 2018)**,** cardiovascular disease (Katzmarzyk et al., 2009), certain cancers (Rangul et al., 2018) and type II diabetes (Åsvold et al., 2017).

Large amounts of sedentary time can also expose workers to increased risk of experiencing back pain as working individuals are approximately 2.5 times more likely to experience backpain compared to non-working individuals (Jackson et al., 2016)**.** Sitting

has been commonly linked to low back pain (LBP) (Hartvigsen et al., 2000). However, it is not known whether sitting and LBP is related through a causal relationship or if sitting merely exacerbates the effects of LBP. De Carvalho et al. (2020) determined that even individuals who did not have history of low back pain developed sitting-induced pain; indicating that sitting pain may not be due to an aggravation of a previous history of back pain and may potentially be a risk factor for low back pain. Some studies have found that time spent seated is positively associated with back pain severity (Gupta et al., 2015; Omokhodion & Sanya, 2003). The literature on this topic is not consistent however, with studies also finding that sitting time is not associated with low back pain severity. Nonetheless, additional studies with large, heterogenous populations are needed in this area to assess the nature of the relationship between sitting and back pain. Until then, much work is needed to develop prevention strategies for individuals who must sit for extended periods for work.

2.2.1 Sitting interventions and is it enough?

Given the possible complications of prolonged sitting, various interventions have been suggested to reduce time spent seated, especially in occupational environments. Ergonomic engineering and administrative interventions such as sit-to-stand desks, walking breaks, computer-prompted standing messages, etc. have been proposed to reduce time spent seated at work. However, a review conducted by the Cochrane Collaboration concluded that the current research indicates that sit-to-stand desks are the

only intervention that can consistently decrease time spent seated, although the evidence quality was low. Other interventions such as walking strategies and computer prompts showed varying results (Shrestha et al., 2018). This indicates that more research and interventions are needed to find more conclusive, consistent methods of breaking up time spent seated to reduce the associated health risks.

Prolonged sub-maximal trunk flexion has a negative impact on a variety of biomechanical factors in the low back. It has been shown that prolonged sitting can induce spine height loss by compressing the intervertebral discs and increasing intradiscal pressure. This can result in an outward flux of disc fluid which decreases the height of the discs and the overall height of the spine (Pape et al., 2018). Loss of disk height can impact the health of the intervertebral disc cells, as well as alter the mechanics of the joints of the segment. Prolonged sitting has also been linked to an increase in passive stiffness of the lumbar spine (Beach et al., 2005). Stiffness changes can in turn negatively affect the functional stability of the spine and may lead to possible injury.

2.1.2 Sitting-induced pain

In both laboratory and field settings, those who sit for prolonged periods report experiencing transient, sitting-induced low back pain. This pain, often referred to as sitting-induced pain, can be experienced in as little as 1-hour of uninterrupted sitting (De Carvalho et al., 2020). Anecdotally, it has been reported that this pain quickly subsides

upon standing or interruption of the seated posture. Although it may be suggested that sitting is an aggravating posture for those who already suffer from low back pain, sitting-induced pain is approximately equally reported amongst healthy and clinical groups (Greene et al., 2019). These groups do not have an identical response however, pain developers tend to move more frequently with greater amplitude, report greater magnitudes of pain, and exhibit greater levels of co-contraction (Dunk & Callaghan, 2010; Greene et al., 2019; Schinkel-Ivy et al., 2013a). As a result, individuals with no previous history of low back pain may be at equal risk to develop sitting-induced pain as it may or may not be related to clinical history.

Often, studies that measure some aspect of spine function before and after periods of sitting do not consider the confounding effects of transient sitting-induced pain. To avoid these effects, individuals who experience low back pain are usually excluded from participating. However, an otherwise healthy population still may develop pain during periods of sitting. Consequently, it is critical to explore this transient posture-induced pain, to identify what effects it can have on outcomes of spine function.

The transient low back pain that is experienced by a large portion of individuals when seated can alter mechanisms of the spine, therefore negatively affecting its functionality. A factor of spine function that is altered when experiencing low back pain is muscle activation (Ahern et al., 1988). An alteration in muscle activation become problematic over time as this can cause dysfunction in the normal functioning of the spine. As a result of low back pain, it is commonly believed that the stability of the spine is reduced;

resulting in the need for additional input through motor control (Panjabi, 1992). To overcome the alteration in spine stability, it has been suggested that the muscles of the spine exhibit a behaviour known as ‘guarding’. Guarding is classified as the persistent activation of the muscles of the spine to ensure that stability is upheld (Ahern et al., 1988). This pain behaviour is notable because the constant, maintained activation of the spine muscles can create further pain and dysfunction in the spine. This is shown as individuals who are suffering from low back pain typically have greater magnitudes of muscle activation, which can lead to further injury and exacerbation of symptoms (Dankaerts et al., 2017; Watson, Booker, Main, et al., 1997). An aspect of spine function that may be altered due to changes in muscle activation is the flexion relaxation phenomenon.

2.3 Flexion relaxation and the flexion relaxation phenomenon

Flexion-relaxation is a biomechanical phenomenon that originates in the muscles of the low back during trunk flexion. At the onset of the movement, there is a burst of muscle activation amongst the paraspinal muscles to initiate the flexion movement. Near maximum flexion, at 46° to 50° of flexion of the lumbar spine, the muscle activity of the paraspinal muscles of the low back, ceases or dramatically decreases (Figure 1) (Solomonow, Baratta, Banks, Freudenberger, & Zhou, 2003). This point during the range of motion of the spine where the muscles effectively ‘turn off’ is the onset of flexion relaxation phenomenon (FRP). Floyd and Silver first identified this phenomenon in 1955

and proposed that during flexion, the intervertebral ligaments increasingly gain tension and are eventually able to support the load of the trunk near the end-range of flexion (Floyd & Silver, 1955). Their interpretation of the mechanics of the phenomenon was correct and has been further expanded to believe that all passive tissues of the spine (various ligaments, tendons, etc.) receive the transfer of the weight of the torso from the active tissues (lumbar extensor muscles), to allow the muscles to relax. It is speculated that the quiescence of the erector spinae muscles is modified by the activation of the stretch receptors of the spinal tissues during flexion, to reflexively inhibit erector spinae activation (Solomonow & Barrata (2003), Shultz, Kippers, Solomonow (1999), Solomonow (2003)).

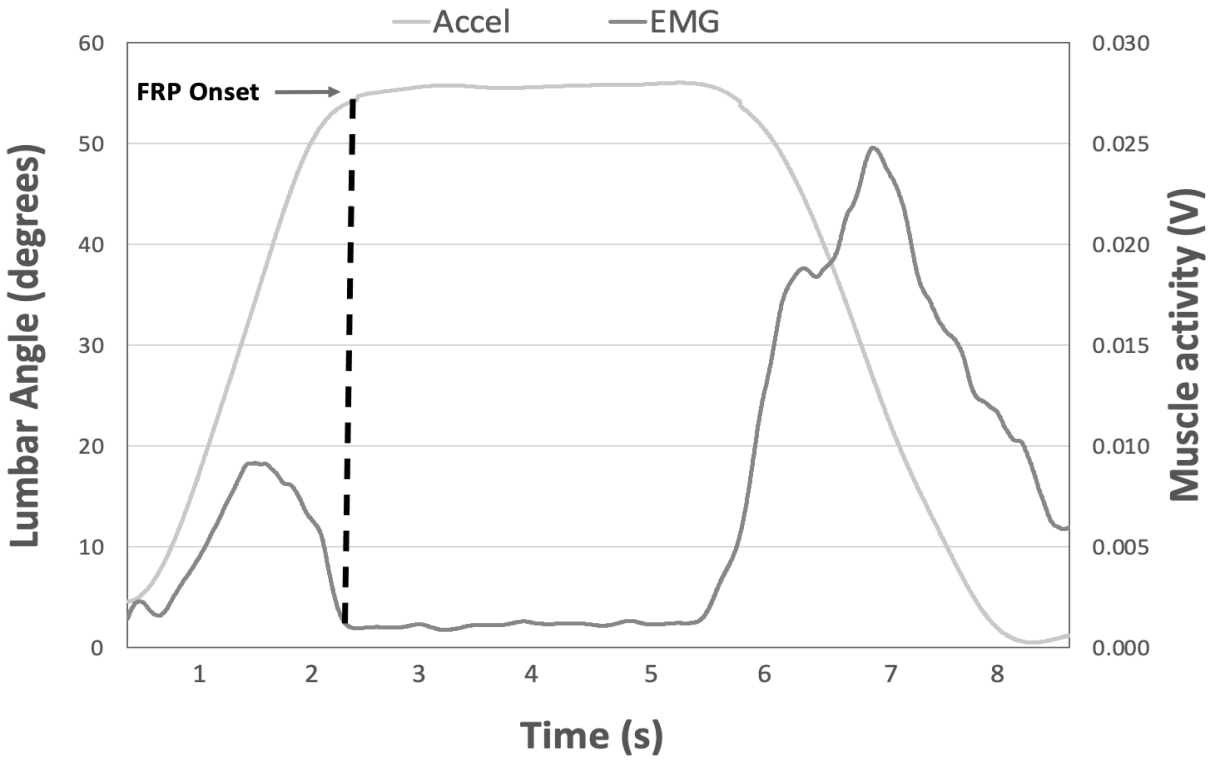

Figure 1: Muscle activation (dark grey) and lumbar flexion angle (light grey) depicting the FRP onset. Representative data collected from one participant in this thesis study.

The onset of the FRP, usually measured in degrees (°), has been suggested to be modulated by various factors such as: clinical status of low back pain (Floyd & Silver, 1955), loading rate (Sarti et al., 2001), and magnitude of load (Schultz et al., 1985). The FRP has also been shown to be altered after exposure to stretching of the tissues in the form of torso flexion. Prolonged spinal flexion stretches the tissues of the spine, temporarily decreasing the stiffness of the ligaments and muscles. If this posture is maintained for a prolonged period of time, the viscoelastic passive spinal tissues stretch under the constant stress created by the upper body and become laxer as a result (Abboud et al., 2016). This lengthening and viscoelastic relaxation of the tissues is referred to as 'creep'. One of the most prevalent changes induced by creep is the increase in range of motion (ROM) after prolonged flexion. The altered ROM is typically present until the tissues regain their typical stiffness, after the prolonged stretching has ceased.

Prolonged spinal flexion can cause many functional issues including a reduction in disc height and a delay in normal spine muscle reflexes. Prolonged flexion without intermittent breaks can result in the dehydration of the discs as the compression prohibits the discs from receiving fluid that is necessary for normal spine mechanics. Consequently, the shortened discs can cause altered mechanics amongst the joints of the spine, possibly resulting in pain or functional consequences. Lengthening of the spinal tissues can also delay the normal muscle reflexes that are initiated near the end range of

motion. Secondly, with more length, the spine joints can move past their normal range of maximum flexion (Solomonow, 2012). In this state, a sudden movement that requires quick response from back muscles to prevent excessive spine motion, may result in an injury. Since the FRP occurs at a point where the tension of the tissues of the back can passively counteract the moment of force of the upper body, it makes sense that longer tissues will increase the range of motion where this point occurs. Reflecting this, the angle at which the FRP occurs has been shown to increase in response to sitting (Howarth et al., 2013).

2.4 FRP and Low Back Pain

The FRP has been consistently measured in individuals who do not suffer from low back pain and has become an indicator of proper mechanical function of the spine. It has been reported that more than 90% of individuals without low back symptoms exhibit the FRP (Shirado et al., 1995). However, Floyd and Silver (1955) first identified that individuals who have 'spine pathology', predominantly NSLBP, lacked the FRP. In most cases, individuals with NSLBP do not exhibit the FRP because the muscles of the low back do not relax near the end range of flexion. Kaigle et al. (1998) measured both healthy individuals and those with NSLBP and found that healthy participants reduced their muscle activation by 78% at the onset of the FRP. However, the clinical participants only had a decrease in muscle activation of 13% (Kaigle et al., 1998).

Although NSLBP patients typically have a restricted ROM, Triano and Shultz noted that it is not the sole cause of the lack of FRP (Triano & Schultz, 1987). One of the proposed theories for the lack of flexion-relaxation amongst clinical groups is the fear-avoidance theory. It is believed that due to the experience of pain, the sufferers' normal behaviours change to avoid experiencing additional pain and/or aggravating their current level of pain. This can involve exhibiting altered movement patterns and experiencing a heightened perception of pain. Changes in motor control then ensue; commonly involving prolonged muscle activation during flexion. The lack of muscular relaxation during flexion causes the absence of the FRP. The absence of flexion relaxation amongst clinical participants is not permanent, however and can be regained through rehabilitative treatment (Neblett et al., 2010). Mayer et al. (2009) determined that after undergoing rehabilitative treatment, 74% of participants exhibited the FRP compared to 31% pre-treatment.

2.5 Flexion Relaxation Ratio (FRR)

Since the FRP is predominantly absent amongst individuals with NSLBP, the flexion relaxation ratio (FRR) has frequently been used to quantify flexion-relaxation amongst individuals with NSLBP. There are many ratios that have been used to quantify the flexion-relaxation phenomenon, with varying methodology. For example, some studies choose to use surface electromyography (sEMG) or RMS EMG in the calculation and in each case, either maximum or mean EMG. The most common FRR that is used to

measure flexion relaxation amongst this clinical group is the ratio of sEMG during the flexion movement divided by the sEMG during full flexion (Alschuler et al., 2009; Mak et al., 2010; Mcgorry & Lin, 2012; Neblett et al., 2003; Watson, Booker, Main, et al., 1997). However, Schinkel-Ivy (2013) reported that six ratios are commonly used for this purpose (Table 1) (Schinkel-Ivy, Nairn, & Drake, 2013). Ratios 1-2 and 4-6 were found to have the greatest sensitivities. Consequently, not all FRRs may be used interchangeably; only Ratios 1, 2, 4 and 6 can be considered to be equivalent. In a study by Alschuler et al. (2009), the authors confirmed that only ratios 1,2,4 and 6 are interchangeable because they were the only ratios that greatly correlated with each other. They also speculated that the ratios that use the re-extension movement in the calculation may be the most sensitive because muscle activation is typically of greater magnitude in re-extension compared to flexion (Alschuler et al., 2009).

Table 1: The commonly used flexion relaxation ratios identified in the literature.

Ratios	Investigations
(1) $sEMG_{flexion}$ / $sEMG_{full\ flexion}$	Watson, Booker, & Main (1997) Xia et al. (2017)
(2) $sEMG_{extension}$ / $sEMG_{full\ flexion}$	Marshall & Murphy (2008) Pool-Goudzwaard et al. (2018) Shin & Yoo (2014)
(3) $RMS\ EMG_{upright}$ / $RMS\ EMG_{slumped}$	Mak et al. (2010)
(4) $RMS\ EMG_{flexion}$ / $RMS\ EMG_{full\ flexion}$	Bicalho et al. (2010) Grześkowiak et al. (2019) Marshall & Murphy (2006) Pagé et al. (2015) Pouretezad et al. (2018) Ritvanen et al. (2007)
(5) $RMS\ EMG_{extension}$ / $RMS\ EMG_{full\ flexion}$	Horn & Bishop (2013) Kim et al. (2013) Moore et al. (2015)
(6) $RMS\ EMG_{full\ flexion}$ / $RMS\ EMG_{flexion}$	Owens et al. (2011)

The FRR has been reported to have great specificity, sensitivity, and test-retest reliability for discriminating between individuals with and without low back pain (Watson, Booker, Main, et al., 1997; Wei et al., 2019). NSLBP patients have smaller FRRs because they typically do not exhibit relaxation during the full flexion phase. In contrast, healthy

individuals typically have higher ratios because their muscle activation typically drops dramatically during full flexion. In a study conducted by Watson and Booker (1997), they reported that their clinical group had FRRs of 3.39 (3.6) and 2.78 (2.8) respectively for the left and right sides, compared to their healthy controls of 13.98 (11.0) and 12.56 (8.5). The changes in FRR amongst clinical populations are not permanent, however, and can be regained upon rehabilitation (Neblett et al., 2003).

2.6 FRR and Sitting-induced pain

Since changes in muscle activation are typically seen amongst individuals who are experiencing low back pain, it would be expected that the FRR would be altered amongst individuals who experience sitting-induced pain. Similar to clinical groups, participants who develop sitting-induced pain, or 'pain developers', would be hypothesized to have lower FRRs since they commonly undergo guarding, a lack of muscular relaxation.

Horn and Bishop conducted an investigation in 2013 to assess whether an acute-induced pain - delayed onset muscle soreness - altered the FRR amongst healthy participants (Horn & Bishop, 2013). They determined that although their participants were induced with acute pain, there were no differences in the FRR between the pain group and control. The results from this study indicate that acutely induced pain may not be sufficient to cause neuromuscular changes. Instead, a prolonged, chronic experience of pain may be necessary for pain modulation to occur and the FRR to be reduced, as a result.

Since sitting-induced pain is an acute form of pain, the FRR may not be different between pain developers and non-pain developers. However, it is of interest to investigate the relationship between sitting-induced pain and an aspect of spine function, such as the FRR, to determine whether sitting-induced pain can cause alterations of outcomes of spine function. Since this sitting-induced pain is not typically recognized and controlled for amongst studies that have a sitting intervention, it is of interest to determine whether it can be a confounding factor on outcomes of spine function, like the FRR. This leads to the purpose of our study, to assess whether sitting-induced pain should be considered a confounding factor in studies that use a sitting-intervention and measure an outcome of spine function pre- and post-sitting.

Co-Authorship Statement

The concept and design of this study was developed by Sarah Mackey, Diana De Carvalho and Samuel Howarth. Data collection was conducted by Sarah Mackey with assistance from Mona Frey. Processing routines were written and edited by Diana De Carvalho, Sarah Mackey, and Mona Frey. Data processing and analysis was conducted by Sarah Mackey with assistance from Mona Frey. Interpretation of results was conducted by Sarah Mackey with discussion and consultation with Diana De Carvalho and Samuel Howarth. The manuscript was written by Sarah Mackey and edited by Diana De Carvalho and Samuel Howarth. The manuscript is formatted to the guidelines of the Journal of Electromyography and Kinesiology.

Chapter 3: Manuscript

An Investigation of the Flexion Relaxation Ratio in Adults with and without a History of Self-Reported Low Back Pain and Transient Sitting-Induced Pain.

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1. Introduction

On average, individuals in the workforce sit for 10-hours each day: 110 minutes more than during leisure days (McCrady & Levine, 2009). Changes to spine function have been reported following exposure to continuous seated durations as short as 1-hour. While commonly thought to be an aggravating posture for those currently experiencing low back pain (LBP), prolonged sitting exposures have also been shown to induce transient, but clinically relevant, levels of LBP in otherwise healthy individuals. This phenomenon has also been observed for exposures as low as one hour (De Carvalho et al., 2020; Greene et al., 2019). Most investigations that measure biomechanical outcome measures before and after seating exposures do not consider the potential for this temporary pain response to affect their desired outcomes even when using back-healthy populations. Consequently, it is important to explore whether sitting-induced pain (SIP) alters spine function before and after exposures to sitting.

Frequent, prolonged trunk flexion can cause detrimental effects to spine function if the exposure is not interrupted by posture breaks. Maintained flexion of the spine can alter spinal mechanics over time. For example, sustained flexion compresses the intervertebral discs (Pape et al., 2018), impeding the normal exchange of fluid necessary for disc health and leading to reduced disc height. Which in turn changes the way the facet joints of the spine interact and function (Billy, 2014). Sitting can also induce viscoelastic changes in the tissues of the low back where prolonged stretching of the tissues leads to a reduction of stiffness and a variety of alterations in spine mechanics

(McGill & Brown, 1992). Decreased tissue stiffness, also known as viscoelastic creep, can alter spine functionality in many ways including delayed muscle reflexes of the spine, potentially leaving the spine vulnerable to injury in the case of sudden perturbation (Sanchez-Zuriaga et al., 2010). Fundamentally, these are related to potential adaptations to neuromuscular control induced by sustaining a flexed spine posture.

One indicator of spine function that could be altered by exposures to spine flexion and/or back pain is the flexion-relaxation phenomenon (FRP). Flexion-relaxation is a biomechanical phenomenon that occurs during a certain range of trunk flexion, where the paraspinal muscles of the back effectively "turn off" or relax at a specific point near the end range of flexion. It is speculated that this occurs at the point where the passive tissues of the low back and pelvis become able to withstand the total weight of the trunk (Mcgill & Kippers, 1994). This event takes the load off the muscles of the low back and allows the muscles to effectively "turn off" (Li et al., 2019). The point at which these muscles turn off, has been found to be delayed with sustained sub-maximal and maximal spine flexion (Callaghan & Dunk, 2002; McGill & Brown, 1992; Solomonow et al., 2003). Howarth et al. reported that as little as 1-hour of sitting can delay the onset of the FRP by 1.3 ± 1.5 degrees amongst participants with no previous LBP (Howarth et al., 2013); however, these authors did not monitor LBP development throughout the seated deskwork exposures. This is relevant because the FRP is typically absent or diminished in those with non-specific low back pain (NSLBP) (Ahern et al., 1990; Kaigle et al., 1998; Kim et al., 2013; Neblett et al., 2003; Shirado et al., 1995).

The flexion-relaxation ratio (FRR), derived from the electromyographic recordings of the spine extensors during a dynamic forward bending trial, has become a common measurement to differentiate between people with and without NSLBP. One of the most common methods for calculating the FRR is dividing the maximum surface electromyography (sEMG) value during the flexing phase by the sEMG during full flexion. NSLBP patients typically experience greater levels of muscle activation during full static flexion, resulting in a smaller FRR for clinical groups (Neblett et al., 2003). Guarding has been proposed to be one of the mechanisms that result in the absence of relaxation amongst patients with NSLBP (Colloca & Hinrichs, 2005). The FRR has been found to have great specificity, and sensitivity for distinguishing patients with NSLBP from healthy controls (Wei et al., 2019). Watson et al. reported that 90% of clinical participants in their investigation were able to be correctly classified from their FRR (Watson, Booker, Main, et al., 1997).

The presence or development of LBP might have a confounding effect on the magnitude of the FRR. Specifically, the altered muscle activation that is commonly experienced with LBP and that may be transiently present during sitting exposures have the potential to alter the FRR post-sitting. Thus, the purpose of this investigation was to determine whether outcomes related to spine function, specifically the FRR, are differentially altered according to history of LBP and/or pain development from a 1-hour seated exposure. It is hypothesized that the FRR ratio will be different between groups based on a history of LBP and pain development, after 1-hour of prolonged sitting.

2. Methods

2.1 Participants

47 participants (21 males, 26 females) were recruited via research posters in the greater St. John's area, Newfoundland and Labrador, Canada. The inclusion criteria for this study were adults between the ages of 18-69 years old, either with or without a history of NSLBP in the past six months. Participants were not eligible if they had any specific cause(s) of back pain such as a history of spinal tumors, surgery, fracture, infection, or inflammatory arthritis (i.e., rheumatoid arthritis or ankylosing spondylitis). Participants were also unable to participate if they were experiencing any back pain at the time of data collection. Individuals who were unable to sit for 1 hour were also ineligible to participate. This project received ethics approval from the Health Research Ethics Board of Newfoundland (#20170082). All participants read the information letter, had an opportunity to ask questions about the experimental protocol, and gave written consent prior to data collection.

2.2 Instrumentation

2.2.1 Electromyography

sEMG data were obtained from the right and left lumbar erector spinae (RLES/LLES). Areas of skin approximately 50mm lateral to either side of the L1 spinous process, representing the location for RLES and LLES sEMG recordings, were lightly shaved and cleaned with rubbing alcohol prior to electrode application. A pair of disposable electrodes (Ag-AgCl, Blue Sensor, Medicotest Inc., Ølstykke, Denmark) were

placed with a 20mm interelectrode distance, parallel to muscle fibres, within each of the areas for RLES and LLES sEMG recordings.

Raw sEMG data were bandpass filtered from 10-1000 Hz, differentially amplified (Desktop DTS, Noraxon, Pheonix, AZ, USA: CMRR > 100 dB, input impedance > 100 MΩ), and collected at a sample rate of 1500 Hz synchronously with accelerometer data.

2.2.2 Accelerometers

Two tri-axial accelerometers (ADXL335, Analog Devices, Norwood, MA, USA) were placed on the skin over the spinous processes of L1 and S2. Surface palpation using established landmarking techniques was used to locate and mark these vertebrae prior to instrumentation (Ambulkar et al., 2017; Snider et al., 2011). The accelerometers were then adhered to the back over the desired vertebrae using double-sided and fabric medical tape in the +y down orientation. Prior to the scheduled data collection each day, the sensors were calibrated relative to gravity (+1g/-1g). The analog accelerometer voltages were digitally sampled at a rate of 1500 Hz using a 16-bit analog-to-digital conversion board (Optotrak Data Acquistion Unit III, Northern Digital Inc., Waterloo, ON, Canada).

2.2.3 Perceived Pain

A custom software program (The Mathworks Inc., Natick, MA, USA) was used to obtain measures of each participant's perceived pain. The program prompted participants to slide a bar along a 100 mm line with anchors of 0 (no pain) to 100 (worst pain ever).

2.3 Study Procedure

Once consent was obtained, the participant was asked to complete a screening questionnaire to confirm study inclusion. Desk and chair heights were adjusted so that the sagittal plane angle of the participants' elbows, hips and knees were at approximately 90° with feet flat on the floor and the height of the computer monitor was comfortable. Next, the program that was used to obtain ratings of perceived pain was introduced and a baseline pain rating was collected. Finally, the participant was instructed on how to use the typing program that was used during the 1-hour sitting trial.

Electrodes for recording sEMG data were then applied to the participant and a 5-second trial of resting sEMG data was obtained where the participant laid prone, relaxed, on a manual therapy plinth. The participant was then asked to move forward on the table so that the anterior superior iliac spines of their pelvis were lined up with the edge of the table. The participant's pelvis and calves were strapped to the plinth using ratchet straps with foam coverings to support the participant's lower body. A series of three 10-second maximum voluntary contraction (MVC) trials were completed. For these exertions, the participant attempted to actively extend their spine against matching resistance applied manually by an investigator so that the participant's torso remained parallel to the floor.

Accelerometers were applied following the MVC trials and four static posture trials (upright standing, maximum standing flexion, extension, and maximum seated

flexion) were collected to calibrate the accelerometers to the participant's range of motion. To standardize these postures, the participants were read a script that instructed them on how to achieve these postures, such as bending through the back and not hip hinging. A 5-second recording was collected in each of the postures.

Next, accelerometer and sEMG data were obtained as the participant completed a single 15-second trial of dynamic full forward flexion for the FRR calculation. To do so, the participant's full flexion movement was recorded: flexing forward, resting at maximum flexion, then returning to normal standing. Verbal cues for pacing were given by a research team member about when to start flexing and extending, but the pace of these movements was not controlled (e.g., by metronome). After the flexion trial, the participant was instructed to sit at the ergonomically adjusted workstation, complete a baseline pain rating and then begin the 1-hour sitting trial. During the sitting trial, the participant was asked to type a standardized script and rate their pain using the VAS every 7.5 minutes. A single trial of dynamic full forward flexion was performed upon completion of the 1-hour sitting exposure.

2.4 Data processing and analysis

2.4.1 Sitting-induced Pain Status

Individuals displaying a change in baseline pain ratings of at least 10/100mm were classified as a PD and those below the threshold were classified as a NPD (Nelson-Wong et al., 2008).

2.4.2 EMG and Accelerometer Signal Processing

All raw sEMG data were processed with custom Matlab code. First, DC bias was removed from the raw sEMG signal before high-pass filtering with a dual-pass of a 2nd order Butterworth filter with a cut-off frequency of 30 Hz to remove electrocardiogram contamination (Drake and Callaghan, 2006). The resulting sEMG signals were full wave rectified and smoothed using a dual pass of a low-pass 2nd order Butterworth filter with an effective cut off frequency of 2.5 Hz to create a linear envelope (Brereton & McGill, 1998). The minimum sEMG signals obtained from each of the RLES and LLES during the prone-lying resting trial were used to remove background electrical signal from sEMG data obtained during the full forward flexion trials. Finally, the processed sEMG data from the full forward flexion trials were normalized to the maximum signal obtained from each muscle across the three MVC trials.

Raw accelerometer data were also processed by custom Matlab software that calibrated the sensors with respect to gravity and converted the voltages to accelerations, calculated the absolute inclinations of each sensor, smoothed the data using a dual-pass of a low-pass 2nd order Butterworth filter with a cut-off frequency of 1 Hz and then calculated the relative lumbar and pelvic angles. The time-varying lumbar flexion/extension angle was calculated for the dynamic forward bending trials as the difference between the accelerometers positioned at L1 and S2. Lumbar spine angular velocity in the sagittal plane was determined as the first derivative of the time-varying flexion/extension angles.

2.4.3 FRR calculation

Processed sEMG and spine kinematic data from the dynamic forward flexion trials were imported to a spreadsheet (MS Excel, version 16.49). An average sEMG signal for the lumbar erector spinae (LES) was calculated from the right (RLES) and left (LLES) processed time-varying signals, and used for the FRR calculation. The flexing and fully flexed phases of these trials were visually identified using the lumbar spine angular velocity data. The FRR was calculated as the ratio of the maximum sEMG during the forward flexing phase to the average signal magnitude over a 1-second window at the fully flexed posture (Neblett et al., 2013; Schinkel-Ivy et al., 2013b). The 1-second window at the fully flexed posture was defined by the 500ms before and after the visually identified point of minimum velocity.

A separate analysis was undertaken to evaluate the interrater reliability of the FRR calculation process. A random subset of 20 trials was selected from the pre-sitting trials, and a second independent rater calculated the FRR using the above described method.

2.5 Statistics

All statistical analyses were performed using SPSS (SPSS Inc., Chicago, USA). Group averages and standard deviations for demographic data and the FRR were determined as descriptive measures. Dependent measures used for statistical analysis were the pre/post FRR values from the dynamic forward flexion trials completed before and after the 1-

hour sitting exposure. A 1-factor between-subjects analysis of variance (ANOVA) was conducted for the post-FRR measures with a factor of pain group (PD/NPD). Additionally, another 1-factor ANOVA was completed with pre-FRR measures and self-reported history of NSLBP (+veHx/-veHx). Independent sample t-tests were performed as post-hoc comparisons for a statistically significant interaction effect and effect sizes. Statistical significance was taken at $p < 0.05$.

Interrater reliability for the FRR calculation process was assessed by an intraclass correlation coefficient ($ICC_{2,1}$) (Shrout & Fleiss, 1979) between the 20 double-rated trials. The 95% confidence interval limits for the ICC were also determined. Strength of reliability was interpreted using the following criteria: less than 0.50 was considered poor, between 0.50 and 0.75 moderate, between 0.75 and 0.90 good, and above 0.90 excellent (Koo & Li, 2016).

3. Results

3.1 Participants

47 participants completed this study, with an average age, height, and mass of 25.9±11.4years, 1.69±0.11m, and 75.3±15.3kg. The breakdown of participants as either PDs or NPDs, NSLBP history, and sex are presented in Table 1. Forty-nine percent of the participants self-identified as having a history of NSLBP and forty percent were classified as PDs.

Self-reported History	No history of back pain (N=24)				History of back pain (N=23)			
Pain Group	NPD (N=15)		PD (N=9)		NPD (N=13)		PD (N=10)	
Sex	F	M	F	M	F	M	F	M
n	7	8	5	4	9	4	5	5
Age in years (SD)	28.86 (14.08)	20.63 (0.70)	22.80 (4.21)	23.75 (2.05)	27.22 (13.81)	20.50 (0.87)	31.80 (13.17)	31.20 (14.58)
Height in m (SD)	163.03 (4.66)	177.11 (6.90)	164.08 (3.82)	178.44 (6.75)	163.57 (4.44)	173.57 (2.80)	167.12 (2.43)	164.93 (26.02)
Mass in kg (SD)	68.10 (15.36)	76.07 (5.29)	65.95 (13.34)	81.60 (6.76)	71.19 (19.30)	82.71 (7.51)	76.18 (12.57)	88.63 (15.69)
Peak Pain Reported in mm (SD)	1.90 (3.05)	3.00 (2.87)	20.86 (4.97)	29.58 (13.22)	0.04 (8.48)	1.63 (5.44)	23.31 (13.22)	26.03 (10.92)

Table 1: Participant Characteristics.

3.2 Flexion Relaxation Ratio (FRR)

Interrater reliability for the FRR calculation process was excellent (ICC 0.970, 95% CI [0.924-0.988]). There was no significant difference (p = 0.492) in the FRRs pre- and post-sitting. There was no significant interaction between pain groups and history of NSLBP for the pre/post FRR (p = 0.214). Additionally, there were no statistically significant main effects for pain groups (PDs 7.707 ± 6.873, 95% CI -4.617, 10.797; NPDs 5.175 ± 3.551, 95% CI 3.860, 6.490; p = 0.105, η^2 = 0.057) or the self-reported history groups (+veHx

6.817 ± 6.372, 95% CI -6.072, 19.033, -veHx 7.136 ± 5.180, 95% CI -2.989, 16.455; p = 0.851, η^2 = 0.001) (Figure 1 and 2).

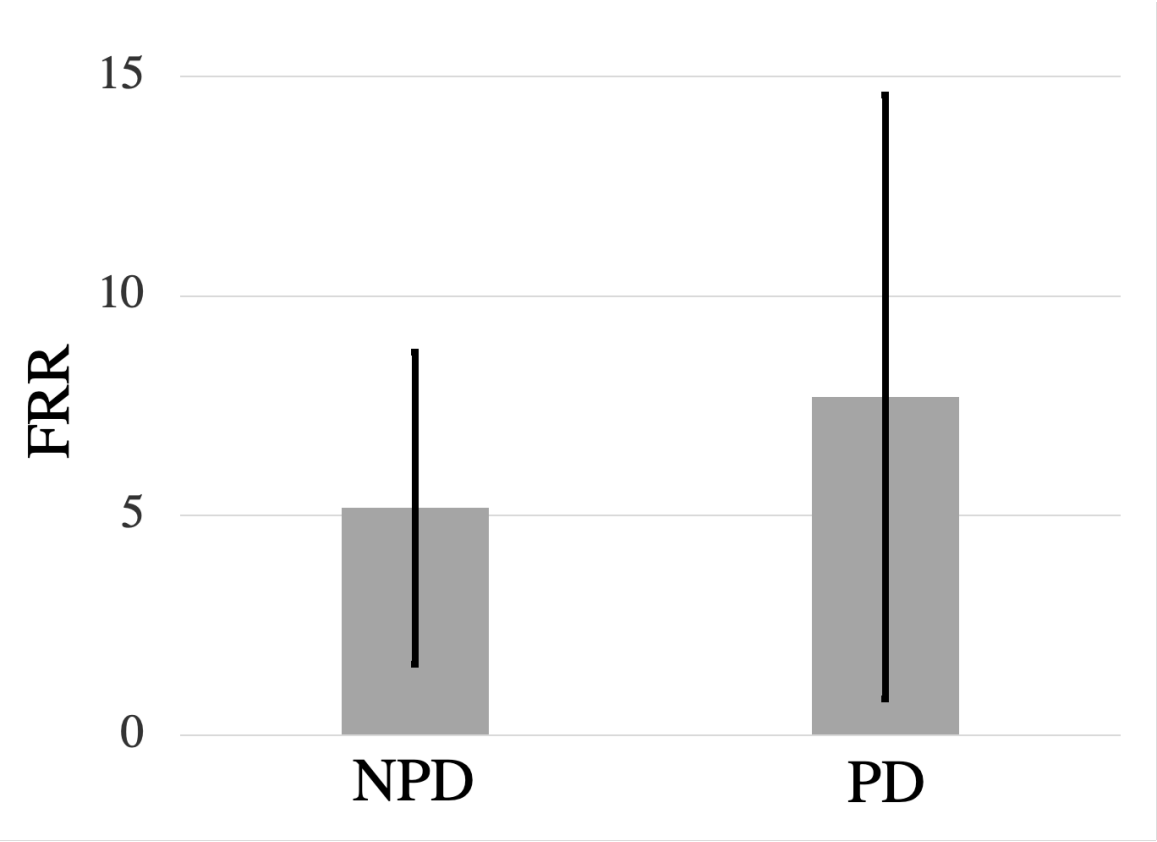

Figure 1: The average FRR for participants who developed clinically relevant levels of transient pain during the 1-hour exposure to sitting (PD) and those who do not (NPD).

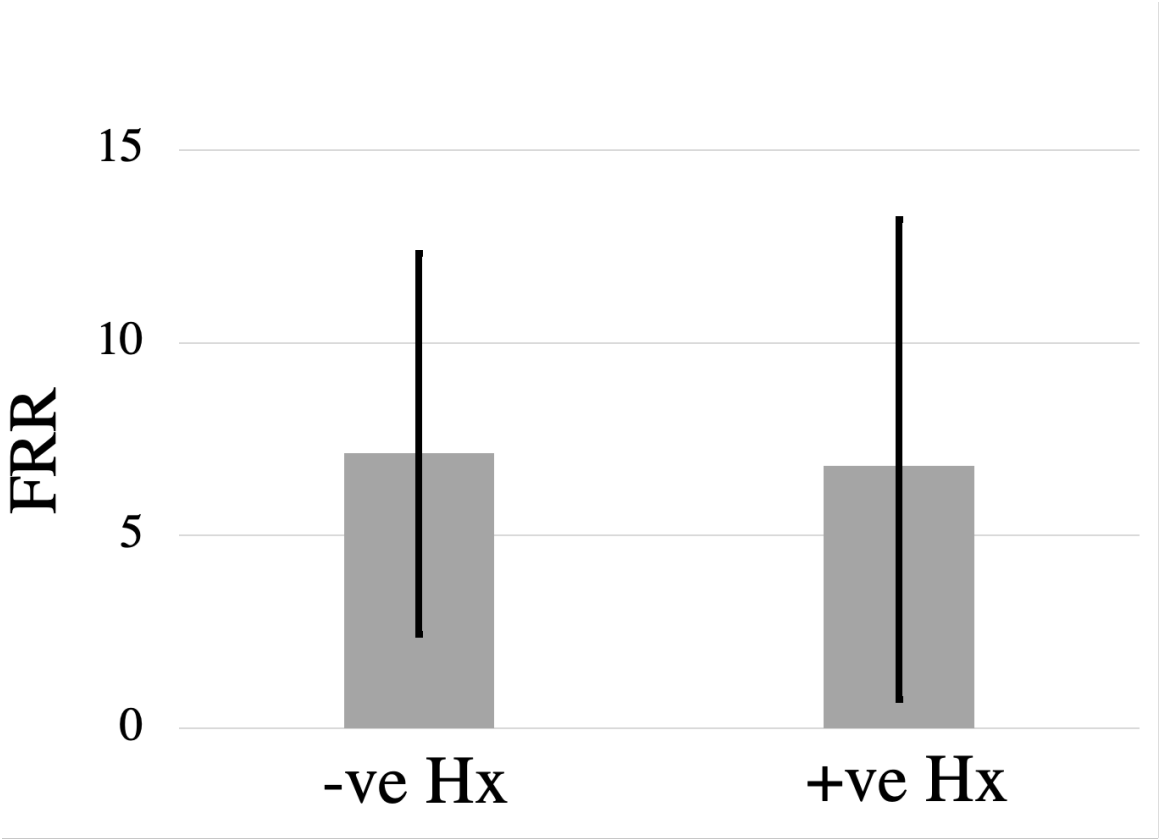

Figure 2: The average FRR for participants with a self-reported history of LBP (+veHx) and without a history of LBP (-veHx).

4. Discussion

Previous work has reported changes in spine mechanics and neuromuscular function following seated exposures. Our study aimed to determine whether individuals with and without a self-reported history of NSLBP and SIP developer status demonstrate different magnitudes of change in FRR following 1-hour of seated deskwork. Interestingly, there were no changes in the FRR following sitting. This suggests that neither short, seated exposures (1-hour or less) nor the presence of transient sitting-induced LBP influences the FRR.

To our knowledge, no studies have been published that explored the FRR and SIP and only a single study has investigated the relationship between the FRR and acute pain induced in the form of delayed onset muscle soreness (Horn & Bishop, 2013). These authors found that the induction of acute pain in the low back did not affect the FRR. They proposed that pain of a muscular origin may not affect the FRR as significantly as pain that originates in other tissues (ligaments or fascia). Since little is known about the origin of SIP, it is possible that it originates in the muscle or has a partial origin in the musculature.

Another possible explanation for the lack of change in the FRR between PDs and NPDs is the relatively short duration of the pain induction. The short duration of noxious stimulus may not have been of sufficient duration to induce pain modulation through neural mechanisms, such as central sensitization, since the pain was transiently induced over only 1-hour of sitting. Consequently, no differences in muscle activation would be seen between the PDs and the NPDs, and the FRRs would be similar. Participants may also not have been in pain during the FRR trial as it was measured while standing. Currently, it is not known how long transient posture-induced pain lasts; however, anecdotal reports from our research group suggest it dissipates quickly upon standing. Although efforts were made to minimize the time between the end of the sitting trial and the FRR trial, it is possible that the pain may have dissipated in that short time or with movement. Consequently, when the FRR was measured, the participants may not have

been experiencing the changes in muscle activation that are associated with pain modulation. The FRR may not be a useful measure in this population as it either is not changed with sitting or quickly returns to normal upon standing.

It was also determined that a self-reported history of NSLBP did not significantly change the FRR. Although previous studies have consistently seen greater FRRs for -veHx participants compared to +veHx participants, our study did not find a statistically significant difference in the FRR between self-reported clinical histories. A potential reason for these findings might have been that the +veHx group were not currently experiencing pain, as they merely had a history of experiencing an episode of NSLBP. Participants who were currently experiencing moderate levels of pain, reported to be greater than 5/10, were excluded. Consequently, the results of this investigation indicate that participants with a self-reported history might need to be in pain to have alterations in the FRR, and not merely have a history of NSLBP. The FRR has been shown to return to magnitudes that are consistent with healthy groups following rehabilitative treatment (Neblett et al., 2003). Therefore, individuals who are not currently experiencing NSLBP would exhibit regular flexion relaxation and no differences would be expected.

The magnitude of the FRRs for the +veHx group is consistent with the literature as Watson et al. reported the average FRR of the LES muscles at $L_{1/2}$ to be 3.09 amongst the +veHx group; compared to our +veHx group average of 6.8177 ± 6.372. However, the average FRR for the -veHx group was lower than expected at 7.136 ± 5.180, with Watson

reporting an -veHx group average of 13.27. Potential reasons for the difference is the small sample size for the healthy group in the Watson study (20 participants in the healthy group compared to 70 in the clinical group) and their control of pace for flexion-extension trials. It is more likely that the smaller group sample size explains these differences, given that the Watson mean is captured with the 95% confidence interval of our mean.

Since the interaction term between clinical history and pain development status was insignificant, it can be surmised that the transient SIP may not have confounding effects on the FRR, after 1 hour of sitting. The results of this study suggest that the SIP would not confound the results of studies that measure an outcome of spine function after sitting. Additional studies should be completed to ensure that this lack of effect is consistent for other variables of spine function, for example low back reflexes, and for postural exposures of longer durations.

The strengths of this study include the use of spine velocity during the calculation of FRR to confirm that the participant was not moving when the minimum muscle activation was observed, the calculation and high magnitude of the interrater reliability of the calculation of the FRRs to ensure the subjectivity of the FRR calculation was minimal, and the detailed inclusion/exclusion criteria. Limitations of this investigation include recall bias that may have occurred when the participants were asked to self-report their history of LBP and the lack of standardization of foot placement and flexion pace

during the flexion-relaxation trials. Additionally, the observed effect sizes for this investigation were small, so replication of this work with a larger sample size would be warranted.

In conclusion, this study did not find a difference in FRR across time or between SIP groups and self-reported clinical history of NSLBP. Consequently, a combination of SIP development and a clinical history of LBP should not have confounding effects on the FRR in short (1-hour) exposures to sitting. The findings of this investigation suggest that with a 1-hour sitting intervention, clinical history and SIP development do not have to be tightly controlled as it may not have confounding effects on the FRR.

Chapter 4: Summary and Future Directions

4.1 Summary

The goal of this investigation was to assess whether the FRR was differentially influenced based on subgroups of self-reported history and status of pain development, after 1-hour of seated deskwork. The rationale was to assess whether developing pain while seated had confounding effects on spine function outcomes, measured after prolonged sitting. It was determined that the FRR was not significantly different between participants with and without a self-reported clinical history of low back pain. Furthermore, the FRR was not significantly different between individuals who developed pain while seated and those who did not. Consequently, studies that did not control for sitting-induced pain during a 1-hour sitting intervention may not have altered FRRs due to the confounding effects of this low back pain. However, durations longer than one hour and other spine function outcomes may have the potential to induce motor control alterations but further research in this area is needed to answer these questions.

4.2 Limitations and Future Directions

Although all efforts were made to minimize the limitations of this investigation, naturally, there were some factors that could have influenced the results of this study. One of the biggest limitations of this investigation was the small, homogeneous, sample size. Participants in this investigation were mainly recruited from a university population and were predominantly young, healthy, and of Caucasian ethnicity. All efforts were made to advertise to possible participants outside the university population, such as social media

postings and the encouragement of word-of-mouth recruitment. However, this recruitment strategy did not result in many participants from outside of the university population. Future studies should explore whether a more broad, representative population has the same response based on grouping of clinical history and pain development, after a 1-hour exposure to sitting. Another possible limitation of this investigation is the reliance on self-reported history of low back pain. This could prompt some participants to be incorrectly classified based on their clinical history of low back pain. Additional investigations could use case-review or reference medical records to confirm the timing and self-report of a history of low back pain. Flexion rate was not standardized for this investigation, meaning some participants might have not completely used their muscles to achieve full flexion and instead, let their torso fall forward into flexion without solely using their back muscles to control the movement. Consequently, more relaxation would be seen amongst these individuals and a lower FRR as a result. The use of a metronome in future studies could ensure that the descent and ascent into flexion is controlled. Additionally, foot position was not standardized between participants. Again, possibly leading to some variation in the flexion task between participants.

This investigation sought to determine whether the spine function outcome, the FRR, was differentially influenced based on clinical history of LBP and SIP development, after an exposure to 1-hour of prolonged sitting. We presume that 1-hour of prolonged sitting was not of sufficient duration to induce pain modulation and consequent alterations in muscle activation. Future studies should investigate whether longer durations of SIP, has the

same result. Finally, our study excluded participants that were currently experiencing low back pain self-reported to be greater than 5/10. Therefore, participants that reported having a history of low back pain were not currently experiencing an acute episode of LBP. Future investigations should assess whether a current episode of LBP and not a mere history of LBP, alters the FRR.

Chapter 5: Conclusion

Sitting-induced low back pain is commonly reported amongst workers who spend much of their workday seated. This posture-induced low back pain, however, does not seem to be an aggravation of a previous history of back pain as it is reported equally amongst workers with and without a history of back pain. This pain may have confounding effects for research studies that measure an outcome of spine function before and after sitting, like the FRR. Consequently, the purpose of our study was to determine whether a history of low back pain and/or sitting-induced pain differentially alters the FRR. We found that the FRR was not significantly different between individuals with and without a history of low back pain, and between individuals who develop pain during 1-hour of sitting and those who do not. Our findings indicate that self-reported clinical history and sitting-induced pain development may not have to be tightly controlled when measuring spine function outcomes before and after a 1-hour exposure to sitting. Consequently, the results from this study indicate that sitting-induced pain should not have had confounding effects on the results of previous studies that measured spine function preceding and following a 1-hour sitting-exposure. However, future investigations should assess whether longer durations of pain-induction change spine function outcomes,. include patients that are currently experiencing an acute episode of LBP, and utilize a larger sample size.

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Appendix A

Appendix A. Copy of current ethics certificate

HREB - Approval of Ethics Renewal 568570

administrator@hrea.ca

Sent: Monday, April 04, 2022 12:55 PM
To: De Carvalho Diana(Principal Investigator) [ddecarvalho@mun.ca]
Cc: Hreaadministrator

Researcher Portal File #: 20170082

Dear Dr. Diana De Carvalho:

This e-mail serves as notification that your ethics renewal for study HREB # 2016.126 – An investigation of transient low back pain development in response to prolonged sitting using a healthy and clinical population: a pilot study. – has been **approved**. Please log in to the Researcher Portal to view the approved event.

Ethics approval for this project has been granted for a period of twelve months effective from April 28, 2022 to April 28 2023.

Please note, it is the responsibility of the Principal Investigator (PI) to ensure that the Ethics Renewal form is submitted prior to the renewal date each year. Though the Research Ethics Office makes every effort to remind the PI of this responsibility, the PI may not receive a reminder. The Ethics Renewal form can be found on the Researcher Portal as an "Event".

The ethics renewal [will be reported] to the Health Research Ethics Board at their meeting dated April 7 2022.

Thank you,

Research Ethics Office

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Office Hours: 8:30 a.m. – 4:30 p.m. (NL TIME) Monday-Friday