The Effect of Prolonged Sitting on Neuromuscular and Biomechanical Responses of the Low Back in Healthy Individuals

by © Ryan Greene

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Abstract

Background: Sub-maximally flexed spine postures have the potential to elicit creep (lengthening) in the posterior passive tissues of the spine leading to a delay in the normal muscle reflexes of the spine. This scenario could result in a low back injury when a sudden perturbation is experienced following a prolonged period of sitting.

Methods: 17 men and 23 women were recruited to examine the effect sitting in an office chair had on the reflex onset times of muscles in the low back. Surface EMG of the low back, and lumbar spine and pelvic angles were collected continuously through all trials. Muscle reflexes were elicited immediately before and after exposure to 2 hours of sitting, and onset times were compared.

Results: Low back muscle reflexes were non-significantly longer after sitting for two hours (72.89 ms ± 38.72) as compared to pre-sitting latencies (60.00 ms ± 27.77). No significant interactions or main effects of pain groups or sex were found for reflex times.

Conclusion: Sitting for two-hours in an office chair does not appear to affect the ability of the low back muscles to respond to a sudden perturbation. This conclusion holds for males and females as well as those who develop transient sitting-induced pain. Future work should examine if longer periods of sitting and/or different chair conditions and spine postures induce delayed reflexes.

Keywords: Prolonged sitting, Low back, Muscle Reflex, Quick release, Low Back pain.

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List of Abbreviations

A/D	Analogue to digital
CM	Centimetres
EMG	Electromyography
GPAQ	Global physical activity questionnaire
HZ	Hertz
L1	First lumbar vertebra
L4	Fourth lumbar vertebra
LBP	Low back pain
LLS	Left lumbar erector spinae
LML	Left multifidus
LTS	Left thoracic erector spinae
MM	Millimetres
MS	Milliseconds
MVC	Maximum voluntary contraction
NPD	Non- pain developer
PD	Pain developer
RLS	Right lumbar erector spinae

RML	Right multifidus
ROM	Range of motion
RTS	Right thoracic erector spinae
S1	First sacral vertebra
S2	Second sacral vertebra
SD	Standard deviation
T9	Ninth thoracic vertebra
T12	Twelfth thoracic vertebra
V	Volts

Introduction

Low back pain (LBP) is one of the most common occupationally relevant musculoskeletal disorders (Kumar, 2001), affecting between 70-85% of individuals at some point in their life (Andersson, 1999). Low back pain represents 25% of all workplace injuries (Yang et al., 2016) and 40% of workplace-associated costs (Reeves et al., 2005). In today's workforce, back pain is the second-highest cause of pain resulting in lost productive time (Stewart et al., 2003). With modern technology resulting in many jobs being completed in seated postures, prolonged sitting is the most common posture in today's workforce in developed countries around the world (Clemes et al., 2014). This has great consequences, since sedentary behaviour is responsible for 9% of premature global mortality, secondary to diseases associated with inactivity, namely type 2 diabetes, coronary heart disease and all-cause mortality (Lee et al., 2012).

Associated with the high prevalence of LBP, further consequences include the large costs associated with people who suffer from low back pain. It is estimated that indirect and direct costs of low back pain can range in the billions, approximated as $100-200 billion in the United States (Katz, 2006) and $11 billion in the United Kingdom (Maniadakis and Alastair, 2000). Indirect costs associated with time missed from work contribute significantly to back pain-related expenses: estimated to be 75% of total costs (Katz, 2006). Further, an estimated 5.28 hours of lost productive time per week can be attributed to back pain in contrast to individuals who suffer common pain conditions such as headache (3.51 hours/week) and arthritis (5.19 hours/week) (Stewart et al., 2003).

Research has shown that biomechanics, forces and postures experienced by the body, play a significant role in the generation of musculoskeletal injury (Adams and Dolan, 1996). For flexed postures of the spine like those in sitting, it is believed that passive tissue deformation of the lumbar spine leads to inflammation and delayed muscle responses (Hendershot et al., 2011). This prolonged flexed posture could also lead to less stiffness in the lumbar spine (due to this passive tissue deformation), as muscle reflexes are not able to adequately respond to an event acting on the back. Biomechanically, a lack of stiffness puts tissue at an increased risk of becoming injured under load. Further, less stiffness can result in a delay of the normal muscle activation that would occur reflexively during typical activities to protect excessive joint range of motion (ROM). Delaying these reflexes would arguably place those joints at greater risk of injury. In the literature, alterations in reflex response timing alone has been shown to differentiate developers of low back pain in 80% of individuals, with pain developers showing longer muscle reflex latencies than their healthy controls (Reeves et al., 2005).

Work by Boudreau et al. (2011) found that individuals with low back pain exposed to a sudden perturbation (unexpected changes in balance) experienced longer onset muscle reflex times. Similar findings were also found by Sanchez-Zuriaga et al. (2010), where muscle reflex onset was delayed significantly from 60 ± 12 ms to 96 ± 26 ms. Muscle reflex response time is important since delayed onset times could lead to potential injuries of the spine. This is because muscle reflexes act to limit excessive flexion of spine joints during normal movements (Sanchez-Zuriaga et al., 2010).

Muscle reflexes are initiated at the level of the spinal cord. Specifically, alpha motor neurons can control contraction of musculoskeletal muscles through a reflex event resulting from a muscle stretch, which excites muscle spindles in afferent axons. From here, this signal is relayed to alpha motor neurons at the level of the spinal cord causing an efferent volley to the muscle, causing activation (Hill et al., 2008). The role of muscle reflexes is to turn the appropriate muscles on so that the body can maintain balance and co-ordination. They also protect peripheral joints from moving beyond the physiological and para-physiological ranges where tissue disruption can occur (McGill and Brown, 1992). Controlling the spine is done through co-activation of agonistic and antagonistic trunk muscles, and this increased activation results in a stiffer lumbar spine that is more stable (Cholewicki and McGill, 1996). Both expected and unexpected challenges to balance will elicit muscular reflexes; however, the response in expected events is muted due to input from the central nervous system as it anticipates the challenge (Shahvarpour et al., 2014). Changes in muscle length will delay normal pre-event muscle activation (Avela et al., 1999). In the spine, alterations to muscle reflexes is theorized to lead to reduced stability and increased risk of injury as impaired motor control is unable to limit spinal motion to within healthy ranges. Passive tissue creep, the viscoelastic material property that results in the stretching of tissues when exposed to constant load, has been shown to occur in the spine when exposed to prolonged flexion: maximum spine flexion angles increase upwards of five degrees over a twenty-minute exposure and takes longer than double this time to return to baseline (McGill and Brown, 1992). Prior work examining maximum spine flexion has also shown that tissue creep can cause a decrease in muscle reflex response of the lower back (Solomonow et al., 2003). This increased flexibility

is hypothesized to contribute to increased injury risk. If the range of flexion motion goes past the normal physiological limits because a delayed muscle reflex has not stopped this motion earlier, structures in the spine such as ligaments, joint capsules, muscles and tendons could experience pathological loading profiles (Adams et al., 1987). While numerous studies have looked at the reflex response to sustained maximal spine flexion, to date, no work has been done exploring the effect sub-maximum spine flexion has on the normal muscle reflexes of the back. This is important since sub-maximal flexion is involved in many occupationally relevant postures including sitting in an office chair. Determining the effect of sub-maximal flexion on low back muscle reflexes would be needed to establish safety guidelines for individuals who sit for prolonged periods of time at work.

Literature Review

Back pain: Neuromuscular and Biomechanical Factors

There are many biomechanical and neuromuscular factors which can contribute to low back pain development. Back pain is complex, as there are several ways for back pain to be elicited, whether through the spine itself, injury to muscles, tendons or ligaments of the low back. Injuries can also happen transiently, or through overloading the lumbar spine or from impact. Aside from biomechanics and neuromuscular factors that can lead to back pain, biopsychosocial factors can also contribute to worsening or prolonging low back pain (LBP), such as a fear of pain, depression, a tendency to catastrophize pain or even a person's work environment (Deyo, 2015).

The primary functions of the spine are to allow movement between body parts, carry loads and to protect the spinal cord (Panjabi, 1992). The anatomy of the lumbar spine consists of a vertebral body, which resists most of the vertical compressive forces of the body acting on it (Adams, 2004). Excessive loading of these discs is what can cause tensile failure of the pulposus (Adams et al., 1996). Vertebra are separated by intervertebral discs which is comprised of the nucleus pulposus (which act like hydraulic shocks in the back) and help distribute the load of compressive forces. Intervertebral ligaments span adjacent vertebrae and help limit range of motion of the spine (Adams, 2004). To resist horizontal forces, aphophyseal joints have cartilage-covered surfaces and protect lumbar discs from shear and torsion forces (Adams, 2004).

Biomechanical factors such as forces and postures acting on the body contribute significantly to generating musculoskeletal injuries (Adams and Dolan, 1996). Primarily, compressive forces acting vertically on the spine are a likely culprit to pain development. When under compression, vertebral bodies are the most likely to fail before intervertebral discs (Adams, 2004). Discs can also degenerate to smaller compressive forces over time due to repetitive loading, which can lead to micro-fractures (Adams, 2004). Damage to these vertebral bodies decompresses adjacent discs which can lead to degeneration of these discs (Adams et al., 2000).

Other biomechanical factors like loading on the spine, or bending or twisting past normal range of motion can also generate injury of the low back (Cholewicki et al., 2005). Adams and Dolan (1996) have shown that the risk of bending injury to the lumbar discs and ligaments is significantly impacted by loading rate, loading history and how large the load applied is. Specifically, they showed that fast bending movements increased peak bending by 10-15% of a participant's normal range of motion. When a person surpasses their normal range of motion, this is when they are at an increased risk of hyperflexion injuries to tissues such as muscles and ligaments (Adams et al., 1987). While compression and bending on their own can cause damage to the spine, many daily tasks involve both bending and compression. In these situations (such as retrieving something from the floor), this puts a person at risk of disc prolapse when either the compression or bending loads exceed an individual's normal limits (Adams, 2004).

Prolonged postures have also been shown to elicit tissue creep (McGill and Brown, 1992). Work by McGill and Brown (1992) examined how prolonged flexion can cause tissue creep to occur in the low back; tissue creep is a phenomenon where sustained load on a material will cause it to deform, with the load being insufficient to actually cause the tissue to fail. When a person develops tissue creep from a sustained posture (such as prolonged flexion), their peak flexion can increase, depending on the length of time the posture is applied. As mentioned earlier, when a person is able to go past their normal range of motion, this is when a person is more susceptible to hyperflexion injuries to the discs or ligaments (McGill and Brown, 1992).

Related to neuromuscular function, back discomfort and injury can also occur. Muscles are important for maintaining stiffness of the low back, as they help prevent the low back from surpassing its normal range of motion (McGill and Brown, 1992). When a person surpasses this range of motion, they are at risk of developing injuries to soft tissue such as sprains and other hyperflexion injuries (McGill and Brown, 1992). So, while surpassing your normal range of motion is more of a biomechanically-related event, the function of the low back's muscles help prevents this from happening by providing stiffness. However, if muscles stretch due to tissue creep as well, this will actually lead to inflammation and delayed muscle responses (Hendershot et al., 2011). Inflammation alone of muscle tissue can directly cause pain in the low back, and delayed muscle reflexes can actually prevent adequate stiffness of the back which is needed to protect the back's range of motion. Increased muscle activity on the low back can also contribute to increased discomfort as increased muscle recruitment of trunk musculature can increase loading on the spine (Gregory et al., 2008) which can occur transiently, or from increased recruitment of muscles due to some event. Gregory et al. (2008)

also noted however that increased recruitment of back muscles also acts as a protective mechanism from potential over-loading of the low back due to increased stiffness.

Simply ageing, and degeneration of discs over time is a contributing component to back pain (Adams, 2004). As people get older, tissue begins to become less hydrated than it previously was. Due to this dehydration of the discs, the low back's ability to return to normal after loading is reduced (Adams, 2004). Furthermore, ageing affects collagen fibres in the back, causing them to be thicker, and the tissue to become stiffer, and therefore poorer at being able to absorb sudden loading (Adams, 2004). In a study by Brinjikji et al. (2015), it was shown that the prevalence of disc degeneration in asymptomatic individuals increased from 37% in 20-year olds, to 96% in those who were 80; disc bulge prevalence and disc protrusion prevalence also increased in older individuals.

Injury Mechanics (how does motor control of a joint help prevent injury?)

As detailed above, there are many ways that low back pain can be elicited, whether through overloading of the spine, degeneration of discs, or inflammation. Motor control of the low back is important for protecting an individual from surpassing their normal range of motion, at which point they become at risk of injury. While the previous section has addressed hyperflexion injuries, it is specifically motor control of the low back which protects this. There has been an increased focus on motor control in patients with LBP in recent years, as opposed to exploring only those models that focus on excessive loading or the signs and symptoms (O'Sullivan, 2005). Spine stability is integral, as surpassing the spine's range of motion puts

an individual at risk of developing a hyperflexion injury (McGill and Brown, 1992). Furthermore, the role of muscles is important for maintaining spinal stability, as without muscles, the spine is inherently unstable (Hodges and Moseley, 2003). These muscles when stiffened help prevent the back from surpassing its normal range of motion (Sanchez-Zuriaga et al., 2010). However, proper control of these muscles is determined by the central nervous system reacting appropriately: determining the level of stability of the spine, and preparing itself for anticipated movements, and by being able to adequately react to unexpected movements (Hodges and Moseley, 2003). These strategies can include the timing and number of muscles recruited, co-contractions of agonist and antagonistic muscles, as well as the magnitude of muscle contraction. Altered muscle recruitment strategies can also affect the magnitude and direction of loading on the low back, which in turn affects stability of the spine (Marras et al., 2001; Reeves et al., 2005b). Very little work has been done to determine whether any of these strategies are affected by prolonged flexion of the back; therefore, as a first step, this thesis will focus solely on muscle reflex onset times.

Currently, it is well established that those with existing LBP have movement and motor control impairments as secondary effects of LBP (O'Sullivan, 2005). However, it still remains unclear if people who have LBP exhibit altered motor control due to existing pain, or if motor control changes are what lead to pain (Hodges and Moseley, 2003). When exposed to inflammation, motor control can be limited as a protective mechanism (O'Sullivan, 2005). However, there is increasing evidence that this adaptive mechanism to reduce range of motion and motor control of the back results in improper loading of the back and may induce more

pain (O'Sullivan, 2005). Those who have chronic LBP also exhibit increased muscular contraction over those who do not (O'Sullivan, 2005) and even transient pain developers have been found to have increased muscle activity compared to healthy individuals (Gregory et al., 2008). This increased contraction can contribute to increased discomfort due to tissue strain, as well as reduced movement (for both flexion and extension) due to excessive rigidity (O'Sullivan, 2005). These altered muscular patterns from healthy individuals can even be observed in individuals who were in remission from low back pain (Hodges and Moseley, 2003).

In a study by Marras et al. (2001), it was found that when exposed to spine loading, patients with low back pain experienced 26% greater, as well as 75% greater lateral shear forces as compared to healthy controls. This shows that individuals who suffer from low back pain not only exhibit altered muscular control for dealing with compressive forces, but that they are also more negatively impacted by trying to maintain loads (such as their own body weight when bending, or even holding a heavy object). Marras et al. (2001) also found that during free-dynamic lifting exercises, individuals with LBP were found to compensate for discomfort by reducing trunk movement and motion. To compound this reduced motion, during the same exercise, individuals with LBP also showed the same greater spinal compression, and muscle activity (Marras et al., 2001).

Conversely, a study by van Dieën et al. (2003) showed that this altered muscle recruitment pattern in patients with LBP increased spine stability. Specifically, those with LBP exhibited increased electromyography (EMG) amplitudes (van Dieën et al., 2003), meaning that there was greater recruitment of low back muscles that consequently would translate into greater stiffness/stability of the spine. As mentioned before however, this increase in muscle activity may also contribute to discomfort, due to increased stiffness in the back. While this seems to contradict the Marras et al. (2001) study, this may not necessarily be so; the van Dieën et al. (2003) study focused strictly on muscle activation, as well as muscle co-contractions and trunk angles, meaning that the observation of increased spine compressive forces would not have been detected in this study.

How people sustain an injury, and how they are more likely to get one in the future

As can be seen in the previous sections, there are many mechanisms which can contribute to both injury, and re-injury, for low back pain. LBP is associated with not just a high prevalence rate between 70-85% (Andersson, 1999), but also high one-year recurrence rates, estimated at 33% (Hartvigsen et al., 2018). These mechanisms which contribute to initial injury of the back can also compound the risk for future injury, due to mechanical reduction in the backs ability to respond to compressive forces for example. In the case of injury causing damage to the vertebral bodies, this can lead to degeneration of surrounding discs (Adams et al., 2000). This degeneration of the discs then reduces the low back's tolerance for sustaining a load in the future (McGill, 1997).

As mentioned earlier regarding spinal control, the existence of an initial injury may also contribute to mal-adaptation of motor control mechanisms for the low back, which can contribute to future injuries (O'Sullivan, 2005). Compounding poorer motor control for those with LBP, they also exhibit higher compressive forces on the spine during loading than healthy counter parts (Marras et al., 2001), and are slower doing so (meaning they also experience these forces for a longer period of time). This increased loading (and increased length of time via loading) on an individual means that a person with LBP will have a lower failure tolerance to a load, and are at an increased risk of another LBP episode due to having a lower failure tolerance threshold than if they were healthy.

Psycho-social issues can also contribute to potential re-injury. This can be due to increased guarding mechanisms and reduced movement due to the fear of pain, and this fear can be reaffirmed by relatives and caregivers (O'Sullivan, 2005). After an acute episode of LBP, this mal-adaptation and fear of movement can lead to increased nociceptor sensation and can lead to a chronic pain state (O'Sullivan, 2005). Without movement, there will also be reduced movement of the nucleus pulposus in the spine, which is necessary for hydration and helping the back bear compressive forces (Adams, 2004). Without movement and hydration of the discs, this can increase the risk of future injury as well. Furthermore, this poor control of joint movement can then lead to microtrauma and pain (Hodges and Moseley, 2003), where this microtrauma can then continue to increase the risk of re-injury due to the back's reduced ability to sustain loads or react to sudden perturbations.

Muscle Activity and Reflexes

When a muscle contracts, an electrical signal is presented (the action potential) which exhibits an amplitude and frequency over time, with the nervous system controlling this signal for relaxation and contraction (Raez et al., 2006). Studying the muscle activity signal is made possible through electromyography. Via surface or intramuscular locations, the EMG sensor measures the summation of action potentials as they travel through the muscle (Raez et al., 2006). An action potential being the depolarization along the neuronal membrane when the resting voltage changes from polarized to depolarized due to an influx of ions via sodium/potassium ion channels (Raez et al., 2006). As the action potential propagates, voltage-gated sodium channels open, resulting in the influx of sodium ions to depolarize the membrane further along the axon (Kress and Mennerick, 2008). As the sodium ion channels open, potassium ion channels close (Kress and Mennerick, 2008). For muscle fibres, EMGs are measuring the motor unit action potential (Raez et al., 2006). A motor unit action potential is the summation of all action potentials from a motor unit, which includes motor neurons and all the muscle fibers each motor neuron innervates, where a varying number of active motor neurons increases or decreases the amount of tension on the muscle (Hill et al., 2008). The more muscle fibres recruited, the stronger the motor unit action potential is and the larger the amplitude that EMG will record.

The magnitude of muscle activity exhibited between individuals will be different due to a number of factors such as the level of subcutaneous fat, fat distribution, muscle size and electrode placement. Therefore, the voltages recorded by EMG are often normalized to an

individual's maximum voluntary contraction level in order to allow accurate comparison between research participants (Lehman and McGill, 1999). To do this, participants' muscle activity is recorded at complete rest (considered 0% maximum voluntary contraction), and then participants perform maximum voluntary contractions to determine muscle activity at its maximum effort (100% maximum voluntary contraction). Values to be normalized are divided by this maximum voltage value and then multiplied by 100 to give a percentage.

Muscle control can be either voluntary or reflexive in nature, both relying on different mechanisms. The flexion withdrawal reflex involves sensory neurons in skin, muscles and joints which cause excitatory synaptic contacts in the central nervous system, which then turns on motor neurons to cause muscles to contract (Hill et al., 2008). The reflex circuit is relatively short, as its goal is to be protective from an unexpected event (such as when you step on a sharp object without knowing), and directly bypasses having to send signals to the brain in order for the body to respond quickly to the offending event (Hill et al., 2008). While the muscle reflex is initially isolated in the central nervous system in order to respond quickly to the potential hazard, stimuli are still sent to the brain via interneurons so that you are aware of the event happening, and can also feel the sensation of that event (such as pain) and can make a voluntary contraction to further react to what happened (Hill et al., 2008). This way, the reflexive mechanisms of the central nervous system avoid potential damage to the body by reacting quickly, and locally, before the brain is even aware that something is wrong.

When muscle spindles are stretched, the contraction of this muscle in response to the stretching is known as a stretch reflex (Walker et al., 1990). When stretching of these muscle spindles occurs, the mechanical stimulus activates a voltage gated ion channel which leads to an action potential that is carried by the Ia afferent fibres to the dorsal horn of the spinal cord. The Ia afferent synapses directly with the nucleus of an alpha motoneuron which then relays this action potential directly back to the same muscle to cause a contraction (Palmieri et al., 2004). Since there is only one synapse that occurs in this pathway it is known as a mono-synaptic reflex and it occurs quite quickly with a typical range of 6.5-19 ms in paraspinal muscles (Skotte et al., 2005). To control the sensitivity of the stretch reflex, gamma motoneurons are controlled by the cerebellum to either tighten or relax the muscle fibres within the muscle spindle (Walker et al., 1990). This stretching of the muscle spindles is what differentiates the stretch reflex from the Hoffmann reflex, as that reflex bypasses the muscle spindles directly and is initiated by electrical stimuli instead (Palmieri et al., 2004). While muscle spindles detect change in the muscle length, the golgi tendon organ senses changes in tension in the muscle in a similar manner (Moore, 1984) with the exception that the action potential is carried by Ib fibres to the dorsal horn of the spinal cord which then act upon interneurons and inhibit antagonist muscles (Moore, 1984). Stretch reflexes, in combination with the golgi tendon reflex, regulate proper length and tension of the muscle which facilitates limb posture (Walker et al., 1990).

Muscle reflexes are integral to protecting a person quickly from potential injury. The quicker muscles are able to respond to a particular event (such as putting your hand on a stove

accidentally), the sooner you are able to avoid further harm. To accurately calculate muscle reflex latencies, two methods are primarily used. One method, developed by Staude and Wolf (1999), uses an algorithm and a likelihood ratio to calculate the onset of a muscle turning on. The second method uses the baseline activity of the muscle being recorded for a period of time leading up to the onset of the muscle being activated. The muscle is then considered to have turned on when the %MVC surpasses the mean plus a predetermined standard deviation (Hodges and Bui, 1996). In the literature, the baseline time used to calculate this average, as well as the mean plus standard deviation used, vary study to study.

The method used for each study in the literature also varies greatly. For example, Cholewicki et al. (2005) used the onset detection algorithm developed by Staude and Wolf (1999), Miller et al. (2010) used the mean muscle activity between 500 to 250 ms prior to their perturbation, and muscle activation was considered when muscle activity surpassed that mean plus two standard deviations of the mean. Gregory et al. (2008) took the average baseline activity 50 ms leading to the perturbation, and onset was determined by muscle activity surpassing this mean plus three standard deviations of the collected baseline. Shahvarpour et al. (2014) used both the likelihood ratio developed by Staude and Wolf (1999), as well as the standard deviation method. Specifically, they used their baseline plus two standard deviations from this mean (Shahvarpour et al., 2014). In their discussion, Shahvarpour et al. (2014) noted that both the likelihood ratio and the standard deviation methods yielded comparable results. Liebetrau et al. (2013) took five perturbation trials, and calculated onset time as the median of the trials. The standard deviation method was also used here, using baseline activity 300ms leading to

the perturbation as the mean, and then using the mean plus four standard deviations as the threshold to determine muscle reflex onset time (Liebetrau et al., 2013).

Hodges and Bui (1996) in their comparison of different standard deviation methods found that multiple standard deviations provided accurate determination of EMG onset. However, error is still likely to be observed regardless of standard deviation used. In their work, it was found that a lower standard deviation (such as 1 SD) resulted in type I error, where the muscle may be considered active when it actually is not due to such a low threshold being needed to be met for the muscle to be considered on (Hodges and Bui, 1996). Conversely, a larger standard deviation (such as 3 SD) would be more prone to type II error, where there is a failure to determine when actual EMG onset occurs due to a higher threshold being needed to be achieved (Hodges and Bui, 1996). Ultimately, Hodges and Bui (1996) observed that there were insignificant differences in muscle reflex onset times when comparing different standard deviations to determine muscle onset latencies.

Alternatively, few studies analyzed reflex latencies by visual inspection. Sanchez-Zuriaga et al. (2010), for example, determined the exact time point the perturbation occurred and the start of the reflex activity. Specifically, the muscle was considered to have turned on when the amplitude for muscle activity began increasing after the perturbation. This method was less frequently utilized as compared to the standard deviation method, or through using an algorithm and likelihood ratio (Abboud et al., 2017).

This difference in methodologies was observed in a systematic-review by Abboud et al. (2017); many studies utilize the standard deviation method, but utilize varying standard deviations, with two standard deviations being the most used (Abboud et al., 2017). Furthermore, Abboud et al. (2017) also cite reporting differences in baseline activity ranging from 50 ms-3 s prior to the quick release. These differences in standard deviations should be noted, as a low standard deviation (such as 1.4) may estimate a reflex to be faster than if a larger standard deviation threshold had to be passed (such as four).

To quantify as a muscle reflex, and not a voluntary response, only muscle onset latencies between the range of 15 ms to 150 ms are considered reflexive (Cholewicki et al., 2005). This range is utilized as it is assumed that muscle reflexes cannot occur in less than 15 ms after the perturbation or quick-release, and that any muscle onset time longer than 150 ms is considered a voluntary response, rather than reflexive (Cholewicki et al., 2005). According to Abboud et al. (2017), there is also inconsistency with the maximum latency allowed to be considered a reflexive or voluntary response, with researchers using a threshold of 120 ms (Granata et al., 2005) to 300 ms (Radebold et al., 2001). Consequently, these differences may mean a reflex in one study is considered a voluntary response in another.

As can be seen in this section, there are several differences amongst studies with actually quantifying muscle reflex times in the low back. While differences exist, it seems that

differing standard deviations used will not ultimately cause a difference in reported muscle reflex latencies (Hodges and Bui, 1996). Furthermore, it was observed that there is little difference in reported muscle reflex times when using both the likelihood ratio method developed by Staude and Wolf (1999) or when using the standard deviation as reported by Hodges and Bui (1996) (Shahvarpour et al., 2014). Therefore, it seems that either method is appropriate for quantifying muscle reflex times.

Quick release and eliciting reflexes (what can change reflex timing? Injuries or continued postures for too long?)

As detailed earlier, muscle activity, and particularly muscle stiffness, is important for protecting the low back from surpassing its normal range of motion, where surpassing this range of motion risks injury to the individual. When exposed to an expected perturbation, the back is able to increase muscle activity in an effort to reduce the effect of the perturbation by offering increased stiffness (McGill and Brown, 2009). However, when exposed to sudden perturbations, even pre-loading back muscles were not enough to reduce the need for a reflex response (Shahvarpour et al., 2014). In the event of an unexpected perturbation, muscle reflexes act to try to prevent the low back from surpassing this normal range of motion. However, if the reflex is not quick enough, the individual may surpass their range of motion, and is at risk of a hyperflexion injury or bending injury (Sanchez-Zuriaga et al., 2010).

There are a number of factors that might affect the timing of muscle reflexes. Previous literature has shown that individuals who develop LBP exhibit delayed muscle reflex times when exposed to a sudden perturbation as compared to those who do not develop LBP

(Cholewicki et al., 2005). There is the potential that reflexes can be affected by the pain experience, by increasing baseline muscle activity or potentially through interneuron modulation at the level of the spinal cord.

Literature has shown that individuals with LBP have longer muscle reflex times compared to those without back pain (Radebold et al., 2000; Radebold et al., 2001; Cholewicki et al., 2002; Cholewicki et al., 2005; Reeves et al., 2005). However, it is unclear why pain developers (PD) exhibit longer muscle reflex times than non-pain developers (NPDs) (Gregory et al., 2008). During a prolonged standing trial with a quick-release pre and post the exposure, Gregory et al. (2008) showed that while there was no difference in pre and post standing reflex times, muscle recruitment differed between PDs and NPDs. Specifically, PDs recruit more muscles in the event of a sudden perturbation. These muscle recruitment strategies employed by PDs as compared to NPDs could be a double-edged sword, considering that the recruitment of more muscles may help prevent risk of injury in the event of a sudden perturbation. Consequently, this increased recruitment of muscles may cause excess loading on the spine, leading to discomfort. While differential muscle recruitment was observed in this study, there was no significant difference in pre and post standing muscle reflex latencies.

A study by Liebetrau et al. (2013) recruited 34 women, including 17 clinical low back pain patients, and 17 healthy controls. For their study, they observed an insignificant delay in muscle reflex onset times for the clinical low back pain group in erector spinae, and a

significant delay in the external obliques and the internal obliques (Liebetrau et al., 2013). Liebetrau et al. (2013) also found that in conjunction with delayed muscle reflexes, clinical low back pain patients also exhibited lower reflex amplitudes, meaning that not only were LBP patients not as able to respond to a sudden perturbation, but they also responded less intensely compared to healthy controls.

Work by Cholewicki et al. (2005) focused on PDs and NPDs differently, in that they recruited muscle reflex times from 303 college athletes, and did a 2- to 3-year follow-up to see which participants developed a low back injury. Cholewicki et al. (2005) found that athletes who developed a low back injury during the time since the initial muscle reflex test reacted 14 ms slower than those who did not sustain an injury during the follow-up period. Furthermore, it was found that having a history of a low back injury increased the odds of a future episode of a low back injury by 2.8 times (Cholewicki et al., 2005).

Similarly, to Cholewicki et al. (2005), Reeves et al. (2005) were able to use the difference in muscle reflex times when exposed to a sudden perturbation to identify separate clinical low back pain groups, and healthy controls with an over 80% success rate. Reeves et al. (2005) observed that during a quick-release for flexion, healthy controls reacted 20 ms quicker than clinical LBP patients.

Lastly, in regard to differences in muscle latencies for those who suffer from LBP and those who do not, a systematic review by Abboud et al. (2017) found that there was an increased erector spinae latency in chronic LBP patients, as compared to healthy controls; however, there was high methodological heterogeneity amongst studies, making other results related to spinal stability inconclusive (such as tissue creep, muscle fatigue and pre-perturbation muscle activity). Furthermore, it was observed that in studies which utilized only healthy populations, and induced LBP in some form, the induced LBP participants exhibited similar muscle reflex latencies to their healthy controls (Abboud et al., 2017).

Previous work has already shown that prolonged full flexion can lead to tissue creep (McGill and Brown, 1992). Creep, the deformation of tissues under sustained load, would lead to tissues that have greater length and consequently allow larger joint ranges of motion before setting off muscle spindles and the stretch reflex. Indeed, creep-induced delays to muscle reflexes have been identified in response to sustained maximum flexion (Sanchez-Zuriaga et al., 2010). Following this, it would be logical that prolonged sitting postures may also contribute to delayed muscle reflexes if enough tissue creep was induced. To date, the study of sub-maximal flexion and its effect on delayed muscle reflexes has not been thoroughly investigated.

Currently, little work has examined the effect of prolonged postures, such as sitting, on the low back's ability to respond to a quick-release. However, plenty of work has already

examined the effect of full flexion on the low back's ability to react to a sudden perturbation, and particularly, how tissue creep impacts these reflex responses. To date, literature shows that periods of prolonged flexion, as well as tissue creep will significantly delay muscle reflexes when exposed to a sudden perturbation (Sanchez-Zuriaga et al., 2010).

Sanchez-Zuriaga et al. (2010) studied the effect of sitting in a slumped posture for one hour on low back muscle reflexes. To elicit tissue creep, they had participants sit in a chair at 70% of their range of motion for the hour, and then tested muscle reflexes prior to and after inducing creep. They observed that under the influence of tissue creep, participants reacted significantly slower after the exposure, with latency increasing from 60 ms (±12) to 96 ms (±26) (Sanchez-Zuriaga et al., 2010).

Differences in muscle reflex times between sexes have been observed in the literature, although insignificantly so (Miller et al., 2010). In a study by Miller et al. (2010), they observed that while women exhibited muscle reflex latencies 18.7% shorter than males, the difference was insignificant. This study by Miller et al. (2010) recruited 10 men and 10 women athletes for their study, and induced flexion perturbation on the participants. Many studies (Cholewicki et al., 2005; Gregory et al., 2008; Sanchez-Zuriaga et al., 2010) also feature recruitment of male and female participants, but do not address potential differences in reflex times between men and women. For Gregory et al. (2008) and Sanchez-Zuriaga et al (2010) this may be due to small sample sizes recruited for each study.

Work by Kastelic et al. (2018) has focused on occupationally relevant muscle reflexes in both expected and unexpected perturbations. This study used 17 actual office workers, and collected their muscle reflex times both before working, and after. They found that muscle reflexes were 10-20 ms longer after working, but the difference was insignificant (for all muscles but the external oblique), and that when exposed to an expected perturbation, muscle reflexes were quicker after working (Kastelic et al., 2018). Muscle reflexes in unexpected perturbations were much longer than when the perturbation was expected (Kastelic et al., 2018). This study is interesting to note; however, it does not state what kind of roles each participant had in this working environment, other than that prolonged sitting was expected to be part of their daily tasks. The author suspected having a small sample size contributed to a high variance in reflex times, and that a larger sample size may have allowed them to determine if the difference in reflex times for the unexpected perturbation may have become significant for muscles other than the external obliques (Kastelic et al., 2018). This work shows that there is a vested interest in determining the role of prolonged occupational postures on low back muscle reflexes, however current studies have not been able to accurately determine the true role these prolonged postures may play.

It is clear that muscle reflexes play an integral role in preventing hyperflexion or bending injuries when exposed to a sudden perturbation. Based on the literature, it is currently unknown how prolonged postures such as sitting will impact the low back's ability to respond to a quick-release. Currently, previous work has shown an obvious difference in

neuromuscular responses between clinical LBP patients and healthy controls, and that muscle reflex times can even be a predictor to those who are more likely to sustain an injury in the future. Conversely, other work has also looked at the effect of tissue creep on low back muscle reflexes, showing that creep negatively impacts the low back's ability to respond to a sudden perturbation. Therefore, there is a gap in the current literature examining how these prolonged, sub-maximal postures will impact the low back's muscle reflex times when exposed to a sudden perturbation. Theoretically, if a participant's posture is flexed enough during sitting, and for a long enough period of time, he or she will be at risk of developing an injury if the low back muscles are not able to adequately stiffen the back in time.

Purpose

The purpose of this study is to determine the impact prolonged sitting in an office chair, a posture that involves sub-maximal spine flexion, has on biomechanical and neuromuscular variables in healthy individuals. Specifically, the reflex onset times of three bilateral low back muscles will be compared before and after a two-hour exposure to office chair sitting. Secondary objectives will examine if this response is different between sexs or pain groups, and will also examine potential differences between sex and pain groups average EMG and spine angles as collected during the prolonged typing trial.

Hypothesis statements

Primary

Null Hypothesis: There will be no difference between low back muscle reflex onset times measured before and after sitting for 2 hours.

Alternative Hypothesis: Increased latencies in muscle reflex onset times will be identified after sitting for 2 hours compared to baseline measures collected immediately prior to the sitting trial.

Secondary

Null Hypothesis: There will be no difference in biomechanical or neuromuscular features (spine posture, muscle activity, perceived pain rating, spine flexibility and muscle reflex onset time) between pain developers and non-pain developers or for men and women while sitting.

Alternative Hypothesis: Women will exhibit different neuromuscular and biomechanical properties (spine posture, muscle activity, perceived pain rating, spine flexibility and muscle reflex onset time) than men and pain developers will exhibit different neuromuscular and biomechanical properties than non-pain developers.

Methods

Participants

Forty healthy participants (20 men and 20 women) were recruited for the study from the St. John's campus of Memorial University with posters (Appendix 3), email notifications

(Appendix 4) and in-class recruitment presentations (verbal text, Appendix 4). All recruitment strategies provided an opportunity for interested participants to direct questions to the primary investigator and/or to schedule a convenient time to take part in the research study. Recruitment methods were directed at all members of the campus community: students, staff and faculty. Ethics approval from the Health Research Ethics Authority of Newfoundland and Labrador was received on September 13th, 2017 (HREA # 2017.199).

The sample size was estimated based on previous sitting and biomechanics research of the low back using mean muscle reflex onset time, pre-creep of 62.5 ms (SD 13.5 ms) and 103 ms (SD 29.5 ms) post-creep (Sanchez-Zuriaga et al., 2010). That study elicited tissue creep by having the participant sit in a chair causing them to be at 70% of their maximum flexion for one hour. Using an alpha of 0.05, and a power of 0.80, and calculating for a 1-way ANOVA with 2 sided-equality for 10 pairwise comparisons (Equation 1). The sample size calculated (8) was then rounded up to 10, and doubled to 20 to account for sub-group analyses of men and women. To account for subgroups when comparing muscle reflex onset timing pre-and post-sitting, we then doubled the sample size again to a total of forty (20 men and 20 women) to ensure there was enough power to compare groups.

Equation 1: Sample size equation for a 1-way ANOVA (pairwise, 2-sided equality) where η= sample size, σ =standard deviation, α = type 1 error, τ = number of comparisons to be made and β = type II error (and 1-β = power).

$$n = 2\left(\sigma\frac{\frac{Z_{1-\alpha}}{2\tau} + Z_{1-\beta}}{\mu_A - \mu_B}\right)$$

Inclusion and Exclusion Criteria

Inclusion criteria were individuals who self-reported not having experienced an episode of low back pain within the last 6 months, aged between 18-69, with the ability to visit the laboratory in the Health Sciences Centre and having four hours of time to complete the study. For the purposes of this study, an episode of back pain was defined as pain in the region bordered by the twelfth ribs and inferior gluteal folds of the buttocks (Krismer et al., 2007) that required treatment or resulted in time missed from work or school. Exclusion criteria were individuals who self-reported having a history of back pain within the past six months, patients with a known history of inflammatory arthritis, spinal deformity, and scoliosis or spine surgery, or marking above 0 mm on a 100 mm Visual Analog Perceived low back pain scale.

Inclusion/exclusion criteria were confirmed by having potential participants complete a health history checklist (Appendix 1). No participants were excluded based on the above criteria.

Instrumentation

Questionnaires

Participants were asked to fill out three questionnaires throughout the course of the study. The first two included a health history checklist (Appendix 1) and the Global Physical Activity Questionnaire (Appendix 2). The Global Physical Activity Questionnaire involved collecting data about a participants physical activity within the previous week of taking part in the study. These were completed after the informed consent process was completed, and before the participant was introduced to the computer workstation. Throughout the prolonged sitting trial, participants were asked to fill out perceived ratings of pain using a 100 mm visual analogue scale with anchors of 0 (no pain) and 100 (worst pain ever) for the different regions of the neck, back, buttocks and thighs. These measures were collected every seven and a half minutes throughout the prolonged typing trial.

Surface electromyography

Six channels of surface electromyography (EMG) were used to continuously collect data from two disposable electrodes (Ag-AgCl, blue sensor, Medicotest Inc., Ølstykke, Denmark) per channel. Following a standardized skin preparation procedure that involved cleaning, lightly shaving and wiping the skin with alcohol, electrodes were affixed bilaterally over the thoracic (5 cm lateral to the T9 process, Callaghan et al., 1998) and lumbar erector spinae (5 cm lateral to the L1 spinous process, Danneels et al., 2001), and lumbar multifidus (2 cm away from the L4 vertebra, Stokes et al., 2003) (Figure 1). For a diagram of low back muscles, please refer to

Netter (2006) pages 174-176. Raw EMG signals were band pass filtered from 10-1000 Hz, differentially amplified (Desktop DTS, Noraxon, Phoenix, AZ: CMRR > 100 dB, input impedance>100 MΩ) and were collected at a sampling rate of 2048 Hz with a 16-bit A/D converter (-2/+2 V range; Optotrak Data Acquisition System, 3D Investigator, Northern Digital Inc., Waterloo, ON, Canada).

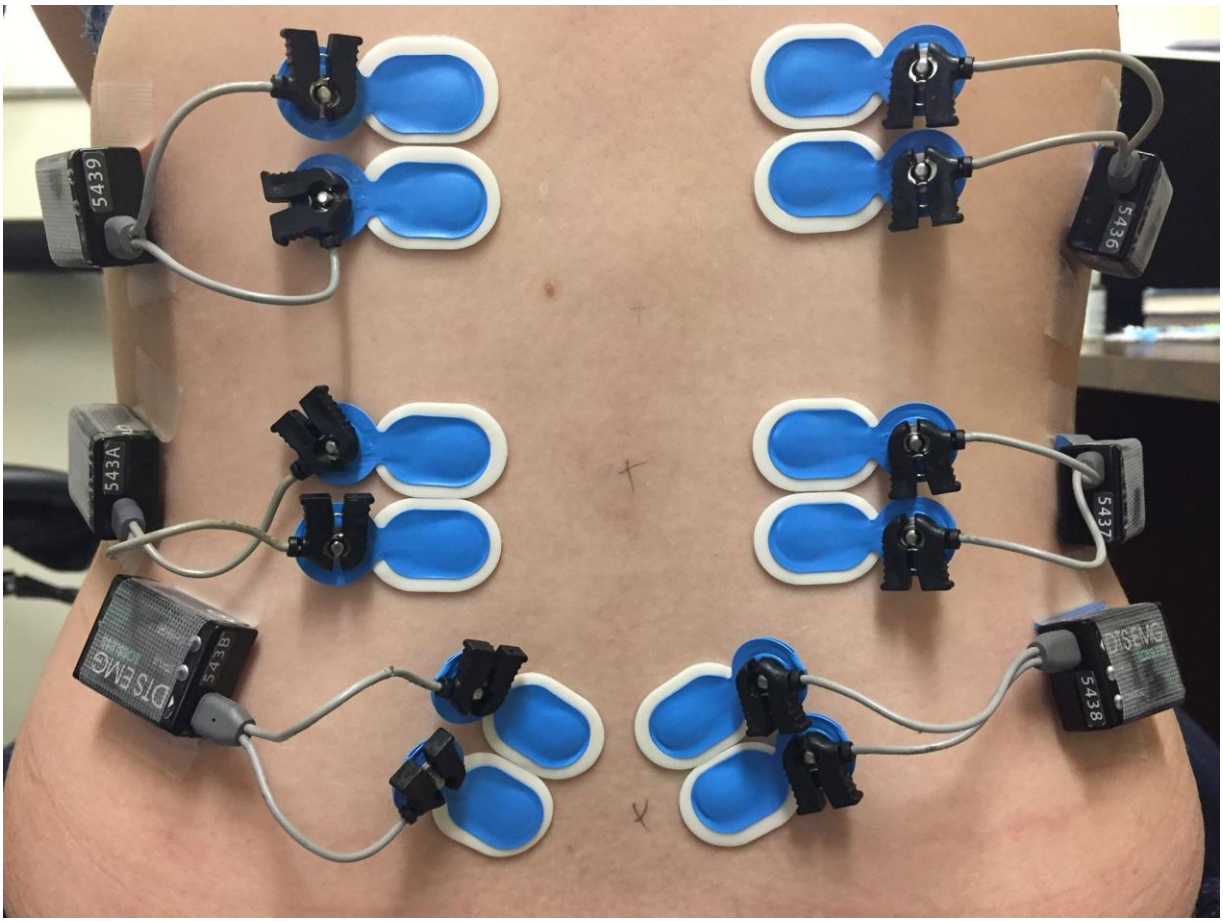

Figure 1: Example of the surface electrodes set up for the thoracic erector spinae (top), lumbar erector spinae (middle) and lumbar multifidus (bottom) muscles.

Tri-axial Accelerometers

Two tri-axial accelerometers (ADXL335, Analog Devices, Town, State, Country) were fixed to the skin over the spinous processes of the first lumbar vertebra (L1) and the sacrum (S2) in the + y down and + z anterior orientation using double sided tape and medical fabric tape. Tri-axial accelerometer data were A/D converted using a 16-bit board at a sampling frequency of 1024 Hz for the quick release trials, and 256 Hz for the prolonged typing trial (Optotrak Data Acquisition System, 3D Investigator, Northern Digital Inc., Waterloo, ON, Canada). These signals were used to calculate lumbar spine and pelvic angles.

Custom Jig for Quick Release Protocol

A custom-made jig, fabricated by the Department of Technical Services (Faculty of Medicine, Memorial University), was used to elicit low back muscle reflex responses in this study. The plywood jig, depicted in Figure 2 and Figure 3, consisted of a bench angled 20º towards the ground. A pole, secured to the jig base with a height-adjustable solenoid release mechanism, was used to release the harness tether. This tether, a galvanized wire cable, was attached to a torso harness fitted to each participant. The participant was instructed to sit upright in the jig and relax, allowing the weight of their torso to be held by the attached cable. This resulted in each participant sitting upright, at an approximate angle of 20º, until the quick release mechanism was initiated. A lap belt was fastened across the participants' thighs to ensure they were stable and secure on the jig.

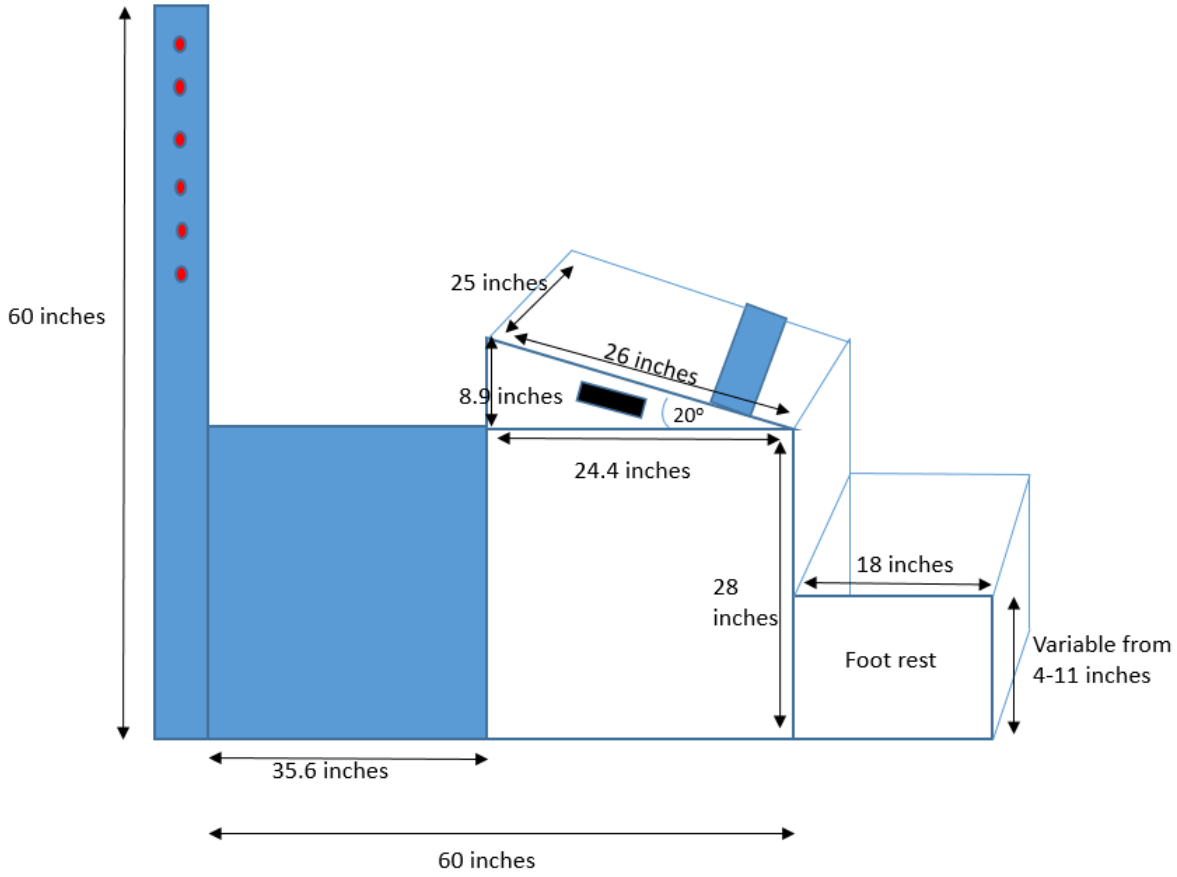

Figure 2: Schematic of the custom-made jig that was used to elicit muscle reflex responses from participants using gravity.

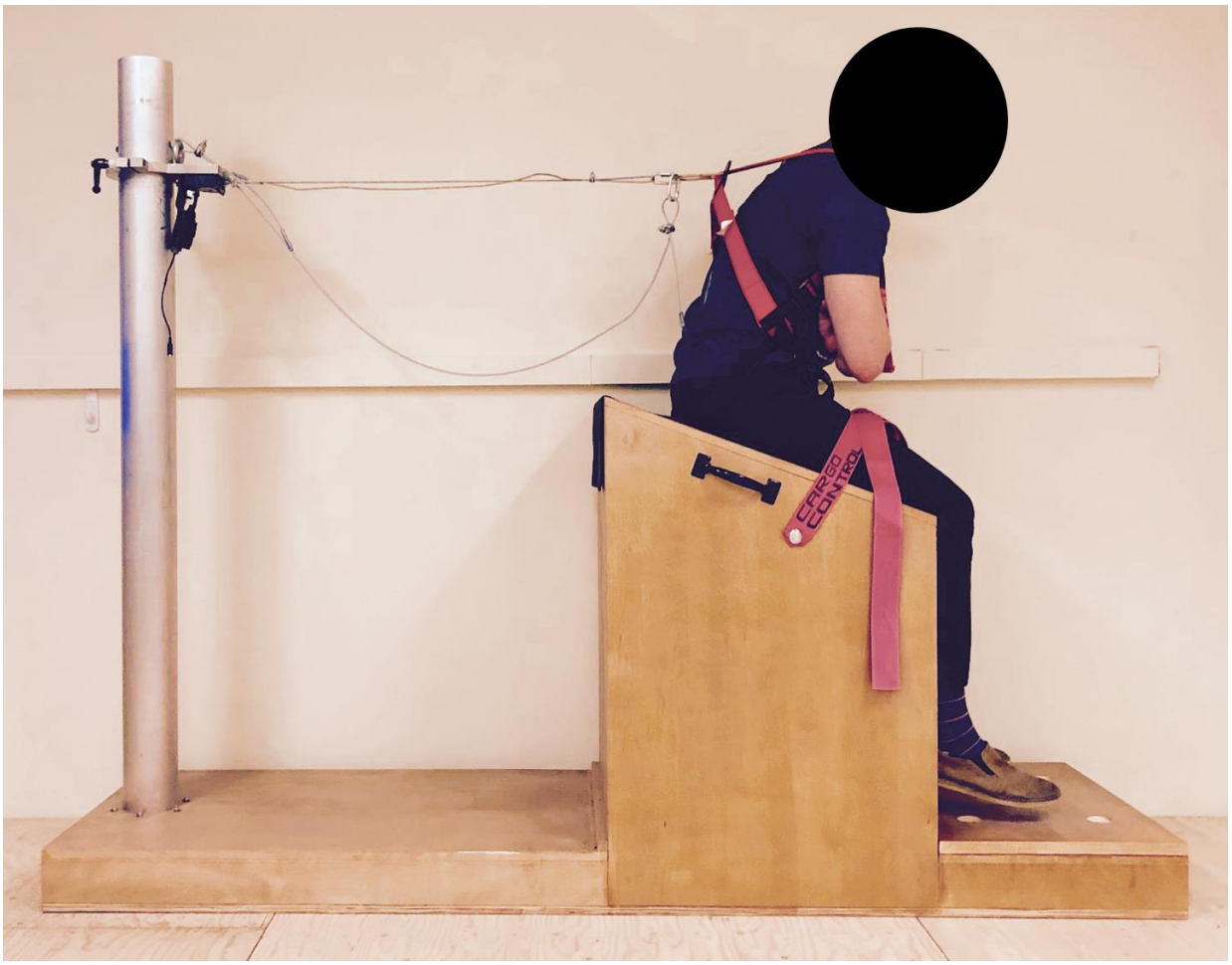

Figure 3: Picture of the custom-made jig, with a participant sitting in position for the quick release.

Perceived Pain Response

A digital visual analogue scale (Figure 4) was used to measure perceived ratings of pain using custom software (Matlab version 2017, The MathWorks, Natick, MA, USA). During the typing trial, participants were asked to rate their back pain by sliding a bar along a 100 mm scale (continuous from 0-100) for each of the nine regions of the body including the neck, upper back (1, 2), low back (3, 4), glutes (5, 6) and thighs (7, 8). Anchors on the scale were 0

mm (no pain) and 100 mm for (worst pain imaginable). All sliders reset to zero as soon as the save button was selected so participants were not able to see their last score.

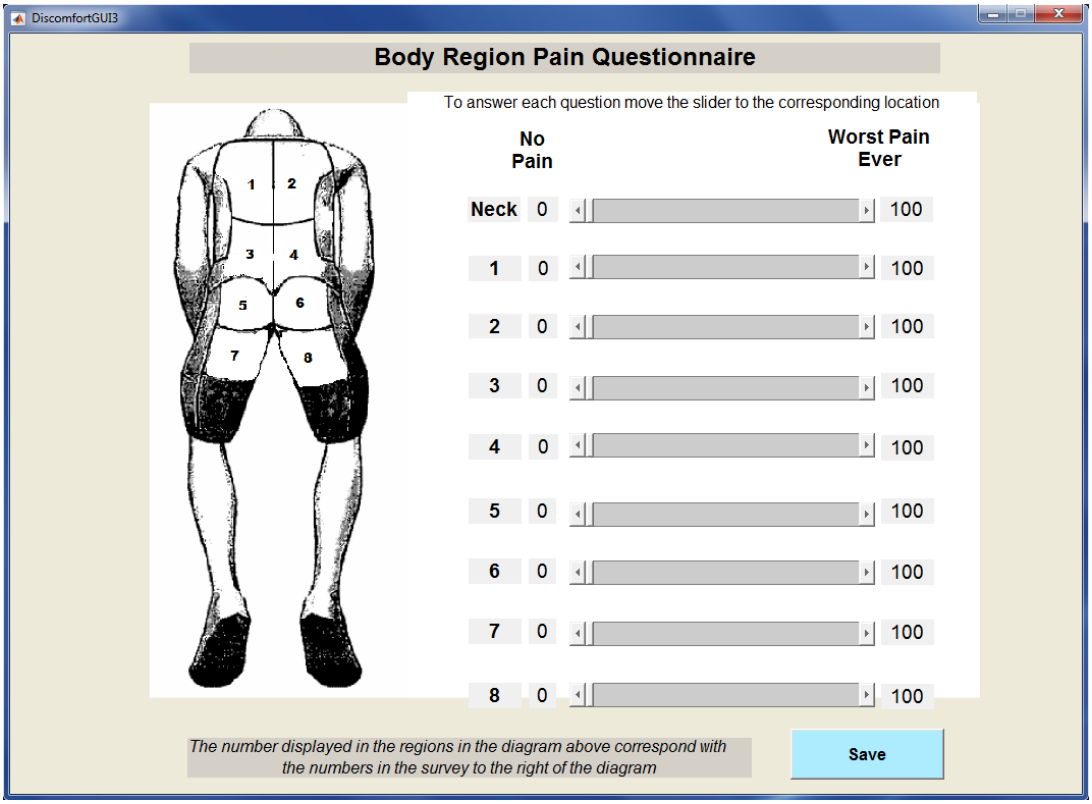

Figure 4: Image of custom written VAS pain rating program. Each number corresponds to the region of the back, neck, buttock and thighs as depicted on the schematic. Participants were instructed to rate their perceived pain for each of these regions by sliding each bar along the line from 0 to 100.

Experimental Workstation

During the two-hour exposure to prolonged sitting, participants completed a standardized typing task. Only one task was chosen as it has been shown that office tasks (i.e., reading, typing and mousing) result in significantly different seated postures (van Dieën et al., 2001).

The workstation included a desktop computer, monitor, keyboard and mouse as seen in Figure 5. The computer monitor, chair height and desk height were adjusted to each participant's anthropometrics according to current ergonomic recommendations (Canadian Standards Association, 2000). The backrest of the office chair was removed in order to minimize interaction with instrumentation fitted to the participant's back. The typing task completed throughout the prolonged sitting trial required all participants copy the same script into a text box during the two-hour typing trial, as seen in Figure 6.

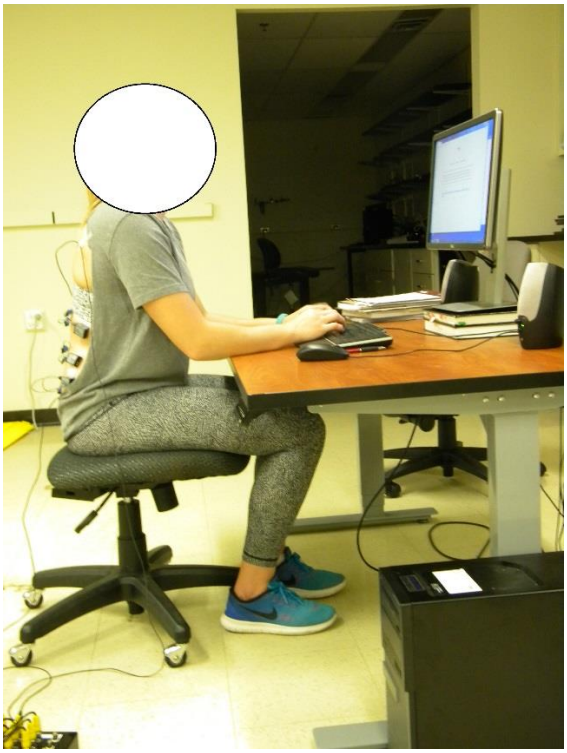

Figure 5: Workstation participants used for the typing trial. Workstation included the computer with typing and pain rating software. Both the desk and chair were height adjustable.

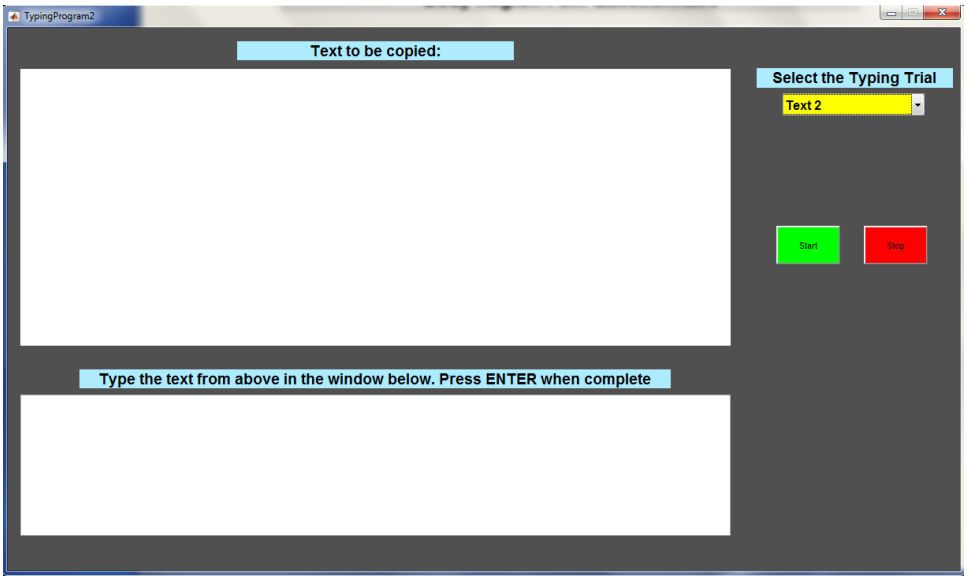

Figure 6: Typing program that was used for the standardized work task during the sitting trial. This work was intended to mimic a typical office task of report writing.

Data Collection Procedure

Instrumentation

When arriving at the laboratory (HSC Room 5315), participants completed the informed consent process (Appendices 5 and 6) with the principal investigator. Next, participants were asked to complete two questionnaires, the Global Physical Activity Questionnaire and a health status checklist to confirm study inclusion. Then, the participant was seated at the experimental workstation and the computer desk and office chair were adjusted to their body size by the researcher according to standard ergonomic recommendations (Canadian Standards Association, 2000).

Next, the participant was instructed to lie prone on a manual therapy plinth such that surface anatomy landmarks of T12, T9, L1, L4 and S1 spinous processes could be located and marked with a washable pen. These landmarks were used for the placement of surface EMG electrodes and the two accelerometers as previously described on pages 29 and 31.

Calibration Trials

In order to allow the comparison of EMG signals between study participants, all channels were normalized to a percent of maximum voluntary isometric contraction. To normalize muscle activity signals between 0 (rest) and 100% (maximum voluntary effort) one 5-second trial with the participant lying face down on a manual therapy plinth at rest and three trials (10 s) with the participant exerting maximum voluntary contractions of their back muscles were completed. To collect maximum exertions, participants were positioned lying prone at the edge of a manual therapy plinth such that their upper torso was off the table (supported between trials by a stool) and their legs were supported firmly by a research assistant. For each trial, they were instructed to extend their back against the manual resistance of a second research assistant as much as possible to get maximum effort from their back muscles. For this maximum effort, the research assistant ensured the participant remained parallel with the floor so as to not over-extend the back (Figure 77).

To again facilitate comparison between participants, low back and pelvic angles were normalized to the end range of flexion motion. Four 5-second posture calibration trials were

collected for these calculations: upright standing, maximum spine flexion in standing (the participant was instructed to bend forward as far as they could as if to touch their toes without bending their knees or hips), maximum spine extension (arching the back as far as possible), and maximum flexion while sitting (Figure 8).

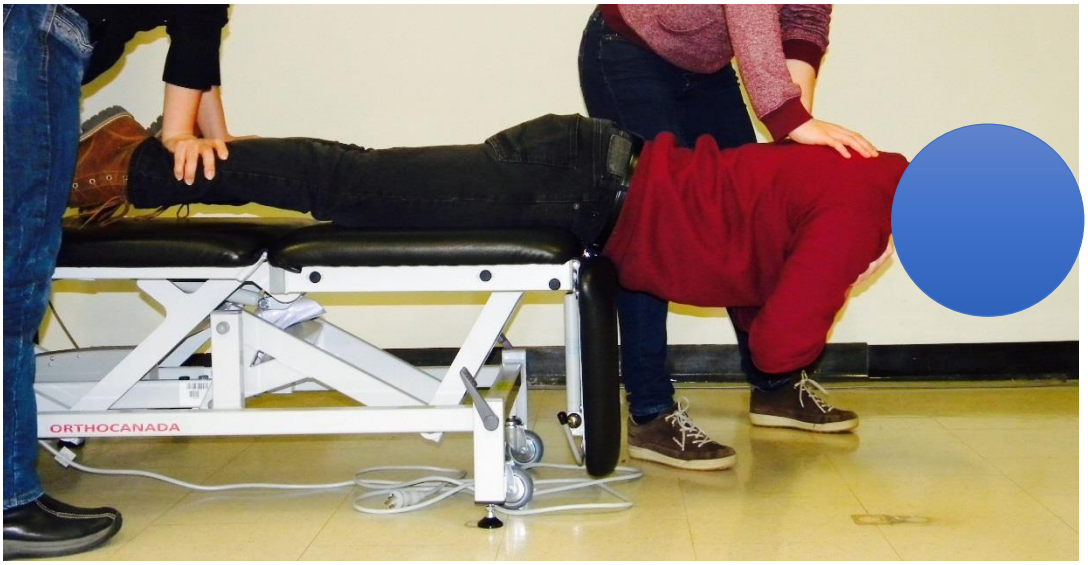

Figure 7: Positioning for a maximum voluntary contraction, with research team members restraining the participant's legs so they were secure, and their back so the participant was not able to extend past parallel with the manual therapy plinth.

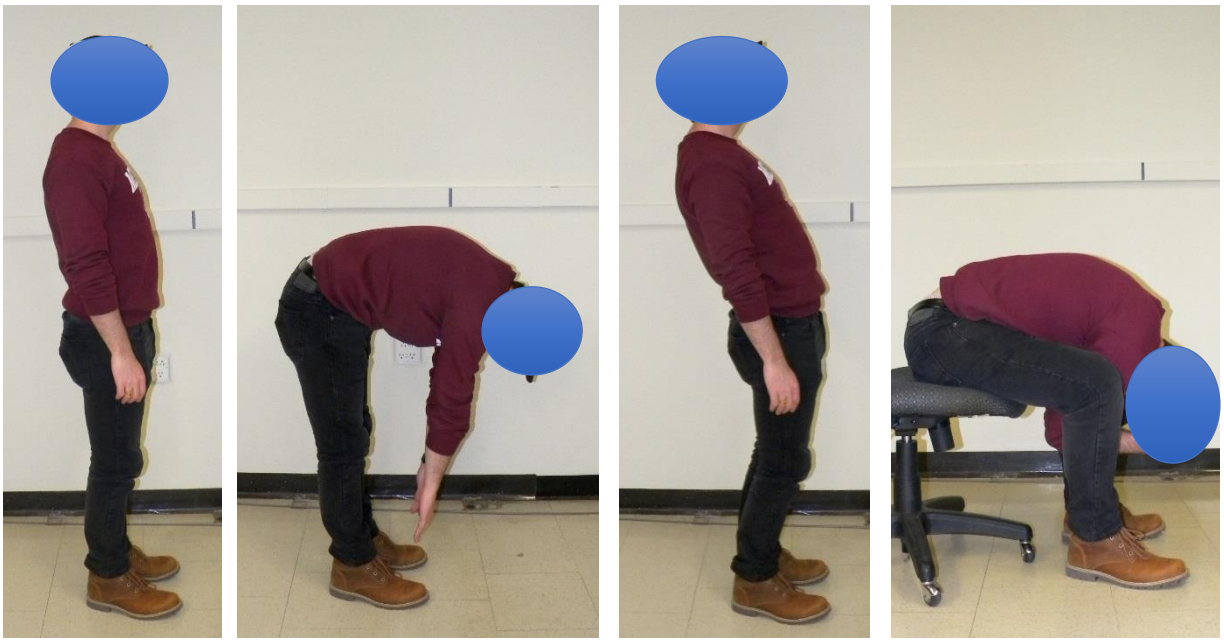

Figure 8: Postures used to normalize spine angles amongst participants. From left to right, the first posture involves the participant standing upright with their back straight. The second posture is maximum frontal flexion, while the next posture is maximum extension. The last stance involves forward flexion while seated, with the participant bending into their knees.

Quick Release Protocol

After all calibration trials were complete, the participant was introduced to the custom-made muscle reflex jig. A safety harness was fitted to the participant's torso and they were seated in the custom jig with the lap belt secured across their thighs. Then the participant was asked to cross their arms over their chest and the harness cable was set such that the participant was oriented perpendicularly to the seat and the cable held the weight of their upper body securely so they could relax. A one-minute adjustment period was provided at this time to ensure that the participants relaxed as much as possible. During this period, the principle investigator

monitored the participant's posture and muscle activity signals to confirm relaxation. At the start of the quick release trial, the experimenter started the data collection. At a time, unknown to the participant, within one-minute of the start of the trial, the harness was released via electrical trigger. While the time the release mechanism was initiated varied from release to release, the time it took for the release mechanism itself to act on the participant remained the same. During the one-minute time period where the participant was released, synchronized EMG and accelerometer data were collected continuously (Figure 8). Specifically, quiet baseline data was collected for 50 milliseconds (Brown and McGill, 2009; Gregory et al., 2008) followed by the release of the tether and 150 milliseconds of post-event data. This release temporarily caused the upper body of the participant to fall forward due to gravity. This unexpected movement elicited a normal postural balance response. This response involves a reflex where the back muscles turn on to cause extension of the torso in order to stop the torso from falling forward. Therefore, the entire quick release protocol involved one minute to allow the participant to become comfortable on the jig, while the researcher further coached the participant on their posture and ensured their muscle activity was low enough that a reflex could occur. Specifically, coaching entailed asking the participant to sit in the chair in a slumped posture so that their low back muscle activity was as low as possible, and so that they were "dead weight" in the harness. This coaching also involved a detailed explanation of the release protocol, so the participant knew what to expect from the release. The researcher would assist the participant in adjusting their posture and would also answer any questions they had during this time. Then, after verbal agreement from the participant, the one-minute period began where the participant could be released at any point within that window of time. After this release, the participant was assisted out of the harness and moved to the typing

station as quickly as possible for the two-hour typing trial. After the typing trial, the participant was moved back to the jig for a second quick-release trial. Following this, the participant was assisted to stand beside the jig for one more trial of maximum spine flexion. The reflex jig system included a safety that would engage if the participant did not balance themselves within approximately 10 degrees of rotation. Consequently, there was no chance that the participant could fall from the jig.

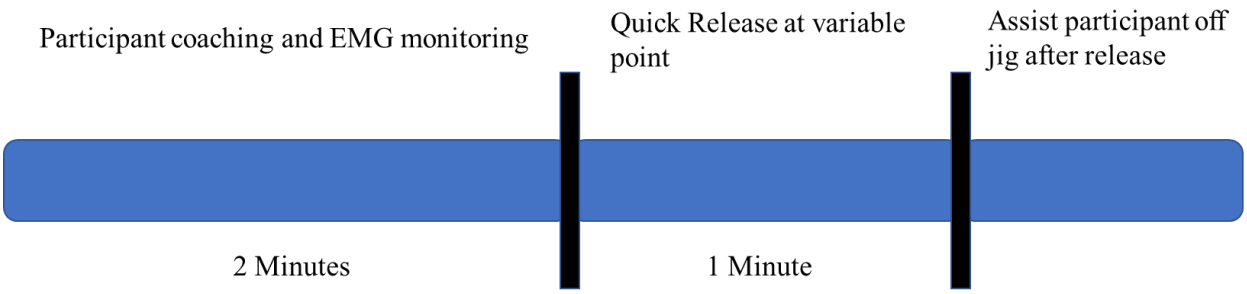

Figure 9: Quick-release protocol, involving an initial two-minute coaching session for how participants would sit in the jig, with concurrent EMG monitoring by the primary investigator. After confirmation from the participant, the one-minute period where the quick release would occur variably began. After the quick release, participants were assisted off the jig by the research team.

Prolonged Sitting Trial

The participant was brought back to the workstation that had been ergonomically adjusted at start of the study and they were asked to complete a baseline rating of perceived pain. The 2-hour typing trial was started with the participant instructed to complete a standardized typing task (copying a report). Repeated measures of perceived pain rating were collected every 7.5

minutes. Synchronized accelerometer and EMG signals were collected continuously throughout the trial.

Post-Sitting Trial Measures

Immediately after the typing trial was completed, the participant completed a second muscle reflex trial. The participant was moved back to the jig and the harness re-attached. The participant was asked to complete one last maximum spine range of motion while standing (5 second trial). Following this, the participant was de-instrumented and received a lab t-shirt as a thank you for their time.

Data Analysis and Interpretation

EMG Signal Processing

EMG signals were processed using custom software (Matlab 2017, The Mathworks Inc., Natick, Massachusetts, USA) written by Dr. De Carvalho. Signals were processed as such: bias removal and high pass filtering at 30 Hz to remove heart rate contamination (Drake and Callaghan, 2006). Next, data was full wave rectified and a linear envelope was used through low-pass filtering with a second order Butterworth filter (cut-off frequency of 2.5 Hz), subtraction of resting EMG levels, and then normalization to the maximum voluntary contraction (MVC) obtained for each muscle group. During the sitting trial, average and peak

EMG values were calculated for each muscle group. For quick-release data processing, data were down sampled to 1024 Hz after filtering so that data would match with accelerometer data. Similarly, after filtering, EMG data from the prolonged trials were down sampled to 256 Hz. During the reflex trials, average EMG data during the 50 ms prior to release had to be below 5 %MVC in order to be considered as effectively off (and so as to permit the calculation of the muscle reflex onset time). Muscle activity during the prolonged trial was used to determine average muscle activity of the right side thoracic erector spinae (RTS), right side lumbar erector spinae (RLS), right side multifidus (RML), left side thoracic erector spinae (LTS), left side lumbar erector spinae (LLS) and left side multifidus (LML) during this two-hour period.

Determination of Muscle Reflex Onset Timing

Muscle reflex onset timing was determined by taking the average linear envelope EMG signal during a 50 ms period that preceded the release of the participant (Gregory et al, 2008) as determined by visual inspection of acceleration of the participant. The timing of the release was identified by the first spike of acceleration, detected on the Y-axis of the accelerometer mounted at the L1 spinous process on the participant (Figure 9). A muscle was considered to be active when the EMG amplitude became equal to or greater than three standard deviations of the baseline mean. This time point when the three standard deviation threshold was passed was then used to determine the muscle reflex latency period: the period of time between the participant being released and when their muscles first responded to the event. The first

muscle to respond reflexively to the release was used as the onset time for the trial. Since muscle reflexes occur in very specific periods of time (too quick can be considered anticipatory and too long would be voluntary), the muscle reflex latency was measured as the earliest onset event within a range of 15 ms to 150 ms after the release (Cholewicki et al., 2005). Trials where all muscles responded after 150 ms were to be considered to have not reacted reflexively to the perturbation and a maximum latency of 150 ms was assigned (Cholewicki et al., 2005).

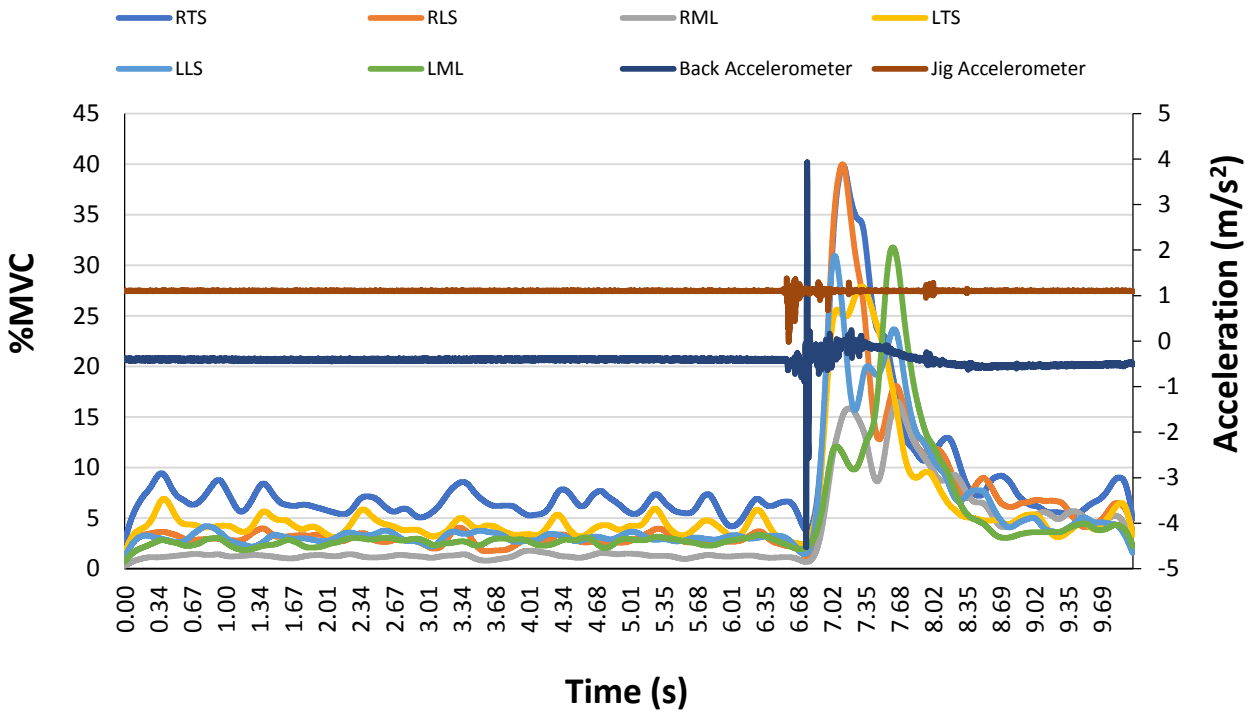

Figure 10: Representative data of a muscle reflex. The muscle reflex onset time was determined to be the first time point after the accelerometer detected movement and muscle activation surpassed ±3 standard deviations of the mean rectified EMG recorded over 50 ms prior to the release of the participant.

Accelerometer Signal Processing

Custom software (Matlab 2017, The Mathworks Inc., Natick, Massachusetts, USA), written by Dr. De Carvalho, was used to calculate lumbar spine and pelvic angles from the raw accelerometer data. All accelerometer data was collected at 1024 Hz. This process included: calibrating the sensors with respect to gravity (+1g, and -1g for X, Y and Z axis, where g=-9.81m/s^2), converting raw voltages to accelerations, calculating absolute inclinations relative to the position of each accelerometer to the other, smoothing the data with a dual-pass 2^{nd} order Butterworth filter (cut-off frequency 1 Hz) and calculating the relative spine angles from the absolute inclinations. These angles were normalized to a percentage of maximum low back flexion using the range of motion calibration trials. For the quick-release trials, acceleration values (m/s^2) were used in order to determine the moment the quick release was elicited via the electronic trigger, as well as when the participant actually began falling forward on the jig. In tandem with EMG activity during the quick-release, this allowed the researcher to determine the exact moment perturbation occurred for the participant through visual inspection. For the quick-release data, there was no down-sampling of the data from 1024 Hz. Processed data for the prolonged sitting trials were down sampled from 1024 Hz to 256 Hz after the filter was applied; pelvic and lumbar angles were then calculated, based on the position of each accelerometer relative to the other. Prolonged accelerometer data was then also normalized based on the calibration trials within each participant's range of motion. Average normalized lumbar and pelvic angles from the whole two-hour typing trial were used to examine if there were any differences in average postures during prolonged sitting between PDs and NPDs and between sexes.

Pain Rating Data

Pain rating data were saved by the program as the distance (to the nearest mm) from 0 to the position of the sliding bar moved by the participant at each time point. Baseline pain ratings from the start of the sitting trial were subtracted from each subsequent data point such that the dataset represented changes in pain that occurred through the sitting protocol itself. Participants who scored a pain rating change of 10 mm or more at any point during the trial were classified as a "pain developer" (PD), and those with changes of 9 mm or less were classified as a "non-pain developer" (NPD) (Gregory et al., 2008). This enabled the primary investigator to address the secondary objective, whether reflex onset timing was different depending on whether or not clinically relevant levels of transient pain were experienced throughout the sitting trial.

Statistical Analysis

A three-way ANOVA was performed comparing muscle reflex onset times, with the fixed factors of pre- and post-sitting, pain group and sex. Statistical comparison of low back posture variables (such as average normalized low back and pelvic angles) and muscle activity variables (average and peak EMG values and muscle reflex data) were used to determine the difference in biomechanical and neuromuscular outcomes after the participant sat for two hours. For the secondary objectives, pain ratings among participants (pain developers vs. non-pain developers) were used to look at biomechanical and neuromuscular differences between

those who developed pain while sitting compared to those who did not. Secondary statistics were done though a two-way ANOVA comparing the outcome variables of average EMG, posture, and qualitative data (GPAQ) when looking at the fixed factors of pain groups and sex. Significance was taken at the $p \leq 0.05$ level. SPSS Statistics version 23 (SPSS Inc., Chicago, USA) was used for statistical analysis. To determine effect sizes partial eta squared (η^2) was calculated where 0.01 is considered small, 0.06 medium and 0.14 considered large effects (Cohen, 1988).

Results

Setup time for the first quick-release procedure took an average of 3 minutes and 33.6 seconds (± 2 minutes and 15 seconds) before the sitting trial and took an average of 1 minute and 56.4 seconds after the sitting trial (± 25.8 seconds). Transferring the participant from the typing station back to the jig following the sitting trial took an average of 6.5 seconds (±3.79). Out of 80 quick-releases, the safety was engaged twice.

Forty participants were collected for the study, with 23 of them being women, and 17 men (Table 1). Average EMG data for one participant was consistently above 5%MVC over the 50 ms prior to release. This meant that it was not possible to calculate the reflex onset timing for this individual; consequently, they were removed from the analysis.

Table 1: Participant characteristics for the study population separated by sex.

	N	Age (years)	Height (cm)	Weight (kg)
Males	16.00	25.00 (+/- 6.26)	178.69 (+/- 7.65)	77.56 (+/- 14.16)
Females	23.00	23.05 (+/- 4.34)	168.20 (+/- 6.61)	62.87 (+/- 8.83)

Anthropometrics

Thirty-five participants provided demographic and anthropometric information for this study. The average age for men was 25.0 (± 6.26) and for women was 23.05 (± 4.34). Average height and weight for men was 178.69 cm (± 7.65) and 77.56 kg (± 14.16) respectively. For women, average height was 168.20 cm (± 6.61) and average weight was 62.87 kg (± 8.83).

Following classification of pain groups, 20 individuals were identified as NPDs (17 women and 3 men) and 19 were identified as PDs (6 women and 13 men). In general, participants reported a maximum pain rating of 15.49 mm (±17.61) and an average pain rating of 2.37 mm (± 3.68) during the two-hour typing trial. NPDs specifically had an average max pain rating of 4.02 mm (± 2.74), and an average pain rating of 0.46 mm (± 0.41) during the two-hour trial. PDs, on the other hand, had an average max pain rating of 27.56 mm (± 18.56), and an average pain rating of 4.39 mm (± 4.44) during the typing trial. Pain groups were found to be significantly different when comparing average max pain rating between PDs and NPDs ($p=0.000$). There was no significant effect of sex on max pain rating, whether when comparing

an interaction between sex and pain groups (p=0.157) or when comparing sex to max pain rating (p=0.365).

Muscle Reflexes

Pre-sitting reflex times for male NPDs had an average reflex time of 56.64 ms (± 7.37) prior to sitting, and a reflex time of 78.78 ms (± 45.95) after the exposure. Male PDs, on the other hand, had a pre-sitting reflex average time of 62.58 ms (± 33.60) and a post-sitting reflex time of 74.14 ms (± 31.05). Male PDs only had a latency increase of 11.56 ms, whereas male NPDs had a latency increase of 22.14 ms after sitting for two-hours. Meanwhile female NPDs had a pre-sitting reflex time of 57.39 ms (± 25.22) and a post sitting reflex time of 67.50 ms (±42.71) and female PDs had a pre-sitting reflex time of 63.48 ms (± 32.16) and a post sitting latency of 82.52 ms (± 46.51). Female NPDs reacted 10.11 ms slower after sitting for two hours and female PDs react 19.04 ms slower after the exposure (Figure 10).

There were no significant interactions between pain group, sex and time (pre/post sitting) for muscle reflexes (p=0.621). There were also no significant main effects for pain group (p=0.967), or sex (p=0.908) with pre/post sitting muscle reflex latencies.

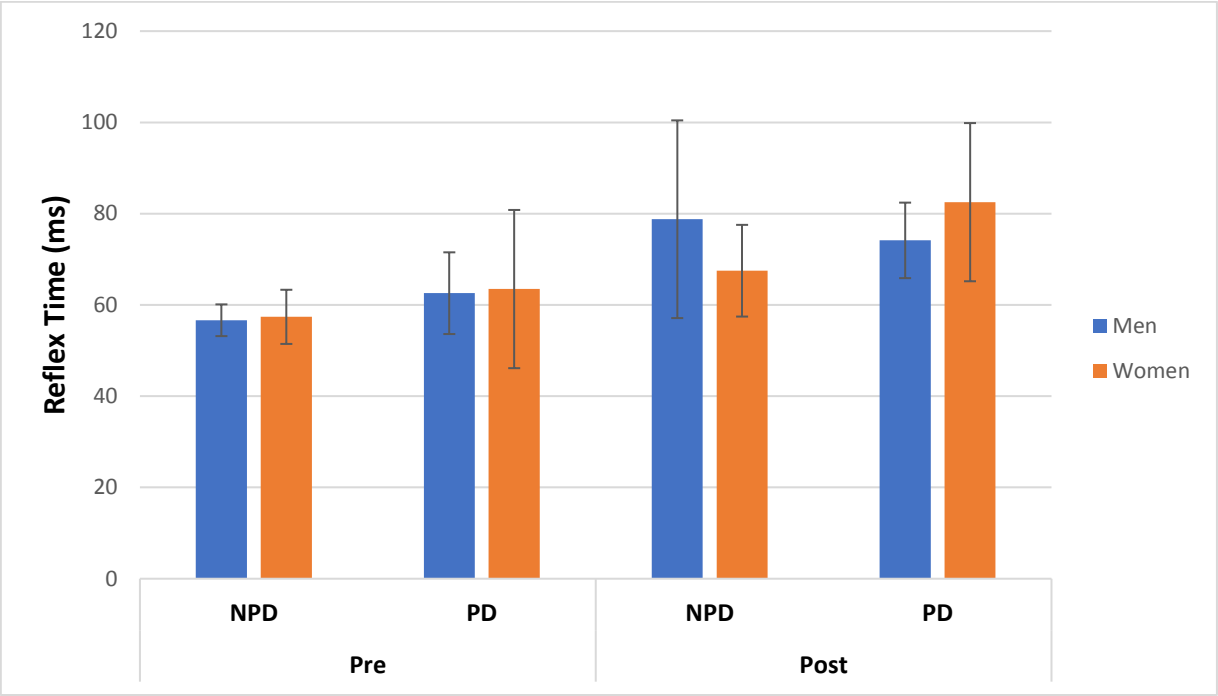

Figure 11: Average muscle reflex times pre- and post-sitting, based on pain groups and sex with error bars showing standard deviation.

Since main effects of sex and pain group were non-significant, these factors were taken out of the model and muscle reflex times before and after the sitting exposure were compared with a 1-way ANOVA for time. For all participants, the average muscle reflex times prior to the exposure were 60.00 ms (± 27.77 ms), while post sitting average reflex times were 12.89 ms longer with an average of 72.89 ms (± 38.72 ms) (Figure 11). This represents an average increase of 21.48% in muscle latency after sitting. The collapsed model found no statistical difference in muscle reflex onset time for the main effect of time (pre/post sitting) ($p=0.114$, $\eta=0.035$).

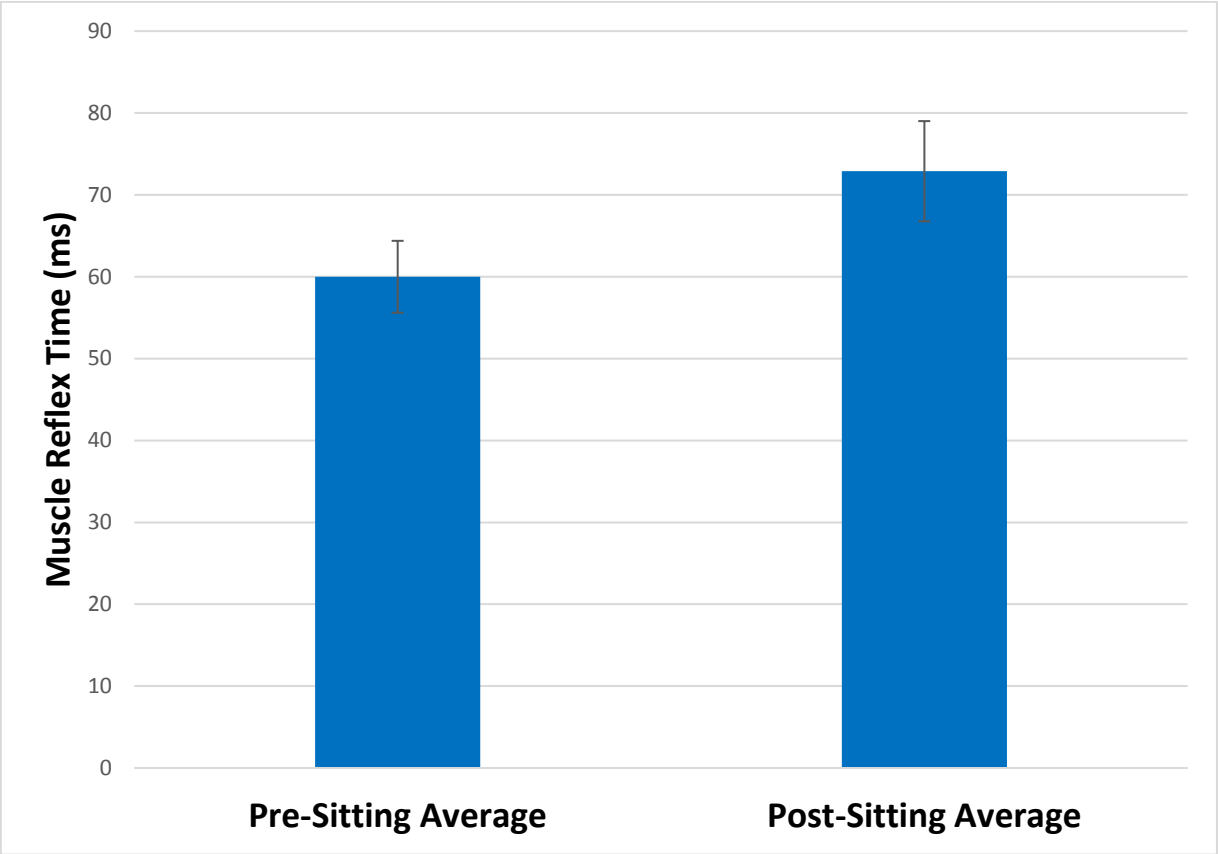

Figure 12: Average muscle reflex times for all participants prior to sitting, and after sitting for two hours with error bars showing standard deviation. No statistical difference was found for time (p=0.114).

Prolonged Sitting Variables

Spine and Pelvic Angles

During the two-hour typing trial, the average normalized lumbar angle for participants was 56.34 % RoM (± 31.68) and normalized pelvic angle was 6.45 % RoM (± 50.89). There was no significant interaction between sex and pain groups for normalized pelvic angle (p= 0.983),

or normalized lumbar angle (p=0.335). Significance was not observed for lumbar or pelvic angles for the main effect of pain groups on normalized pelvic angle (p=0.862) or normalized lumbar angle (p=0.233). There was also no significance for the main effect of sex on normalized pelvic angle (p=0.718) or normalized lumbar angle (p=0.138).

Muscle Activity

Average muscle activity during the two-hour typing trial ranged from 3.36 %MVC to 5.88 % MVC. Specifically, average muscle activity for the RTS was 5.88 %MVC (± 3.15), RLS was 4.00 % MVC (± 2.51), RML was 3.43 % MVC (±1.80), LTS was 3.93 % MVC (± 2.11), LLS was 3.36 % MVC (± 2.03), and LML was 3.56 % MVC (± 2.64). There were no significant interactions between pain groups and sex for muscle activity levels during the prolonged sitting trials (p-values included in Table 2). Similarly, there were no significant main effects for pain group or sex for any of the muscles studied (Table 2), except for sex and RTS, where women had an average muscle activity of 6.86 %MVC (± 3.41), and men had an average of 4.41 %MVC (± 2.07) (p=0.018).

Table 2: P-values for muscle activity during the prolonged sitting trial when examining the interaction of pain groups and sex, as well as the main effects of pain group and sex on their own.

Variables	Pain Group (PG)				Sex (S)				S*PG			
	df	F	p-value	η	df	F	p-value	η	df	F	p-value	η
RTS	1	1.911	0.18	0.077	1	6.445	0.018	0.219	1	0.097	0.759	0.004
RLS	1	1.054	0.315	0.044	1	0.882	0.357	0.037	1	0.143	0.709	0.006
RML	1	0.674	0.42	0.028	1	2.306	0.142	0.091	1	0.11	0.743	0.005
LTS	1	0	0.998	0	1	1.649	0.212	0.067	1	0.03	0.865	0.001
LLS	1	1.541	0.227	0.063	1	1.779	0.195	0.072	1	1.014	0.324	0.042
LML	1	0.005	0.945	0	1	0.447	0.51	0.019	1	0.541	0.47	0.023

During the quick-release, 52.14% of muscles activated in response to the event before sitting, and after the exposure, 48.29% of muscles responded in the second quick-release. In the remaining 47.86% and 51.71% for the respective releases, either noise (due to the harness hitting the sensor during the release), or the muscle already being activated at over 5% MVC prevented it from being included in the analysis.

Global Physical Activity Questionnaire

For pain groups, NPDs had an average GPAQ score of 2585.56 (±2468.80) and PDs had a GPAQ score of 3185.26 (±2186.26) (p=0.755). Men had an average GPAQ score of 3468.75 (±2294.35) and women had an average score of 2455.24 (±2287.85) (p=0.406). There were no significant interactions between pain group or sex for physical activity (p=0.461). Similarly, there were no significant main effects for pain group (p=0.755) or sex (p=0.406).

Secondary Analysis/ Incidental Findings

During data analysis, an interesting trend was observed, where 22 people had delayed muscle reflexes after sitting for two hours, and 17 participants had a quicker muscle reflex latency. Therefore, a secondary analysis was attempted where groups were identified based on whether individuals responded more quickly, or more slowly in the second quick release, as compared to the pre-sitting quick-release time. When looking at muscle reflex times for the delayed muscle group, there was a pre-sitting average of 48.38 ms (± 17.46) and a post sitting time of 90.86 ms (± 20.28). The group that had faster muscle reflexes had a pre-sitting muscle reflex

time of 75.02 ms (± 26.73) and a post-sitting reflex time of 49.63 ms (± 23.39) (Figure 12). For the group which was slower for post-sitting muscle reflexes, 11 were NPDs (11 PDs) and 12 were women (10 men). Of the 17 who responded more quickly, 9 were NPDs (8 PDs), and 11 were women (6 men). Individuals who responded more slowly, significantly did so after sitting for two-hours (p=0.000), as did those who responded more quickly (p=0.007).

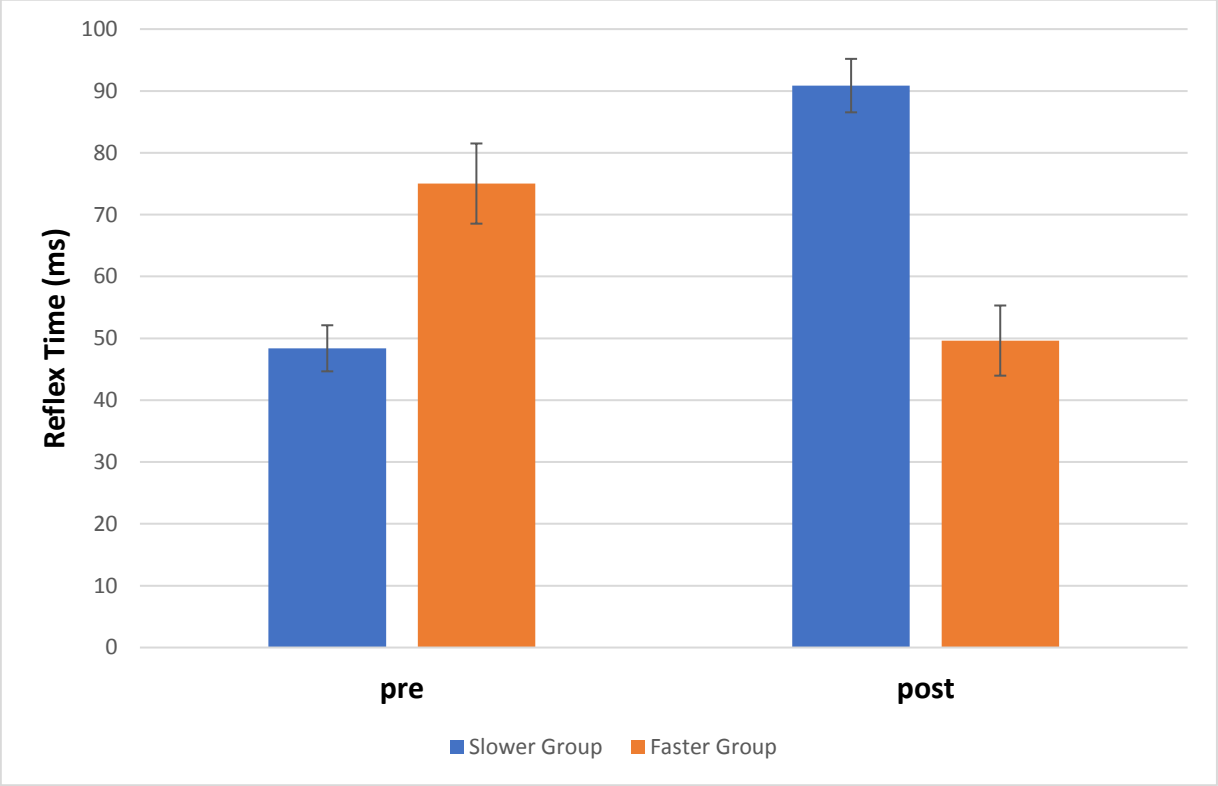

Figure 13: Average muscle reflex times pre- and post-sitting for two hours, based on participants which either responded more slowly, or more quickly after the exposure. Error bars represent standard deviation.

Spine angles were also examined to see if they helped explain differences in those who responded faster or slower after the exposure. Average normalized lumbar angle during sitting for those who responded more slowly during the second quick release was 56.21% RoM

(±32.77) and average normalized pelvic angle was -8.85% RoM (±44.93). For those who responded more quickly, average normalized lumbar angle was 56.50% RoM (±31.63) and average normalized pelvic angle was 23.95% RoM (±53.19). Neither normalized lumbar ($p=0.897$) or normalized pelvic angles ($p=0.090$) were significantly different between these speed groups.

Individuals who responded more slowly after sitting for two-hours were 174.66 cm (± 8.99), while participants who responded more quickly were shorter at 168.68 cm (± 7.21) (Figure 13). When examining demographic information for the groups based on whether they responded more quickly or more slowly after sitting for two-hours, it was found that there was a significant main effect of height with response time ($p=0.044$), specifically where taller participants responded more slowly after sitting. There were no significant differences for reflex time for age ($p=0.470$) or weight ($p=0.241$).

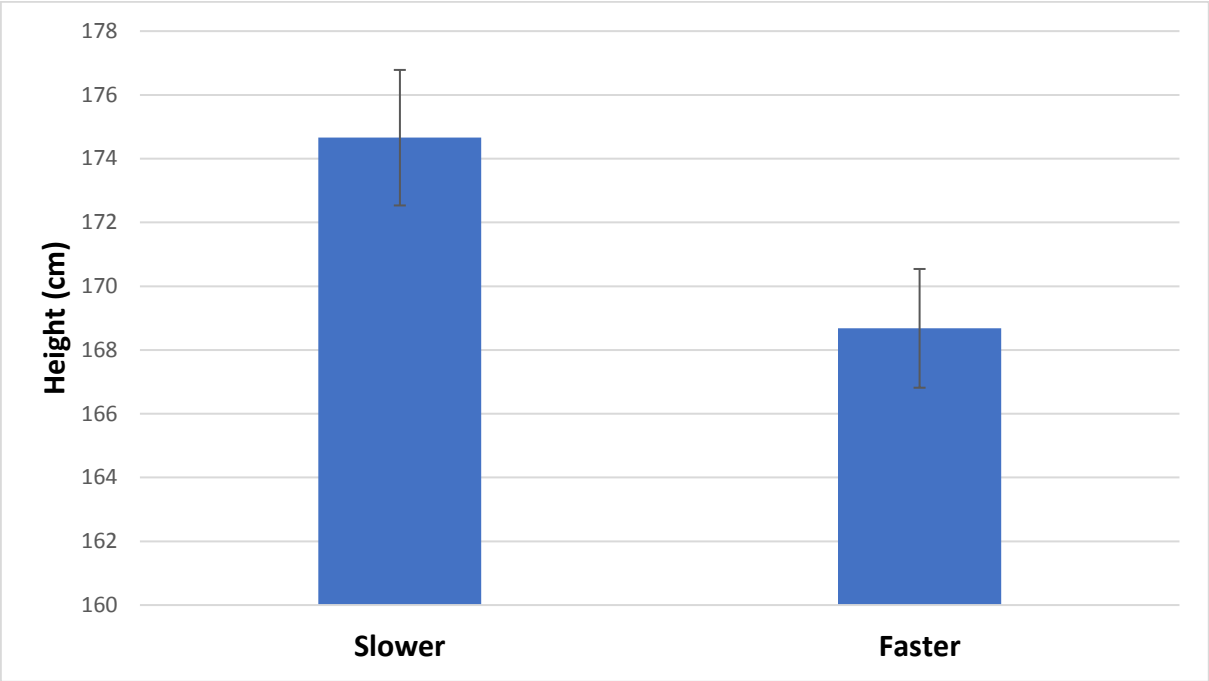

Figure 14: Average height based on whether or not the participant responded more slowly or more quickly after sitting for two-hours, with error bars representing standard deviation.

Discussion

This study investigated whether sitting for two hours had an effect on muscle reflex times of the low back when exposed to a sudden perturbation. Results show that muscle reflex times appear to increase an average of 12.89 ms (±42.12) following prolonged sitting; however, this difference was not statistically different. Therefore, we cannot reject the null hypothesis that prolonged sitting will not delay low back muscle reflexes. Regarding our secondary hypothesis, it was found that only average maximum perceived pain rating significantly differed between PDs and NPDs, with PDs having a higher maximum pain rating (p=0.000). Other biomechanical (normalized lumbar and pelvic angles) and neuromuscular differences (average muscle activity per muscle) between pain groups, or sex during prolonged sitting

were not observed. This study also found that there were no sex differences in muscle reflex times. The analysis looked at reflex times between men and women before prolonged sitting, after the exposure, and also for an interaction between sex and reflex times pre/post sitting. Once again, no significant differences were found. In line with other work, women exhibited faster muscle reflex times as compared to men (Miller et al., 2010), and this difference does not significantly contribute to differences in pain development between men and women. Therefore, our secondary hypotheses must also be rejected.

In this study, participants sat with approximately 60% of their total spine flexion range. This is lower than the 70% of flexion that was observed to induce muscle reflex delays in the paper by Sanchez-Zurriaga et al. (2010). Thus, there is the potential that differences in muscle reflex timing were not observed due to the lower amount of flexion which may have not induced enough tissue deformation to lead to reflex delays or that the sitting exposure was not long enough. Further, there is a chance that our sample size was just too small to reach statistical significance. Practically, we did observe a 21.48% increase in muscle reflex time following the sitting exposure, which may be functionally relevant. This is further supported by the small effect size for this outcome measure ($\eta = 0.035$). Future work can now build off the data in this thesis to determine an appropriate sample size based on the variability we have observed.

Quick-release methods and quantification of muscle reflex times vary greatly study to study, however the muscle reflex times found in this study still agree with those published previously. Miller et al. (2010) used only healthy participants and two standard deviations to

calculate muscle onset and they elicited reflexes in 94% of participants and found that women had an average reflex time of 48.8 ms (±3.0), and males had an average reflex time of 60.0 ms (±3.2). Gregory et al. (2008) used three standard deviations for determining muscle onset timing, and reported ranges of muscle reflexes from 86 (±18) to 95 (±20) ms for thoracic and lumbar erector spinae. These findings are similar to our results, as the average muscle reflex time prior to sitting was 60.00 ms (± 27.77 ms) and the average post-sitting muscle reflex time was 72.89 ms (± 38.72 ms). We are unable to compare our pre and post exposure reflex times with Gregory et al. (2008) as they did not provide pre- and post-reflex times for the erector spinae. For muscle reflexes related to back pain specifically, Radebold et al. (2000) used a standard deviation of 1.4 times the mean to detect muscles turning on, and found that healthy controls had an average muscle reflex time of 53 (±10) ms while LBP patients had an average reflex time of 59 (±14) ms. Another study by Radebold et al. (2001) also looked at the relationship between participants with LBP and their healthy controls, and found significantly larger reflex times of 80 (±20) ms for those with LBP, and found that healthy controls had an average of 63 (±9) ms. In regard to PD/NPD groups, the study for this thesis observed those who were NPDs had an average muscle latency of 57.28 ms (±23.27) and PDs had a latency of 62.86 ms (±32.25) prior to sitting. After the exposure, NPDs had an average muscle reflex time of 69.19 ms (±42.13) and PDs had an average reflex time of 76.79 ms (±35.49). The Gregory et al. (2008) study is the most similar to this thesis, in that a two-hour exposure was used, as well as using three standard deviations to calculate muscle onset. Furthermore, similar to Gregory et al. (2008), this study found large standard deviations in reflex times whereas other studies observed smaller standard deviations. While Radebold et al. (2000) observed quicker reflex times than the study presented in this thesis, a lower standard deviation method

could contribute to this. However, reflex times have been shown to not differ statistically based on standard deviation method used (Hodges and Bui, 1996) so the reflex times can still be compared.

Based on the literature, it was expected that PDs would exhibit delayed muscle reflex times as compared to their NPD counterparts. Cholewicki et al. (2005), in a population of athletes, showed that those who developed back injuries had a muscle reflex response, which was 14 ms longer than their healthy counterparts. This thesis study included male participants of similar anthropometrics (height and mass) and female participants of similar height and approximately 10 kg lighter on average, also observed that PDs exhibited delayed muscle reflex onset times compared to NPDs both pre- and post-sitting, but with smaller differences in reflex times than what was observed by Cholewicki. Pre-sitting, PDs reacted 5.58 ms slower to the jig release, and after sitting for two-hours they reacted 7.60 ms longer. While the difference exists between PDs and NPDs, this study identified transient low back pain developers over the course of the two-hour sitting period as opposed to using a follow-up session with participants to identify those who developed an injury later, which was used in the Cholewicki study. This result found by Cholewicki could be explored in the future by repeating this protocol and following the participants for a period of time in to determine if those individuals that exhibit longer latencies become PDs.

We found that transient pain developers do not have a significantly different muscle reflex latency compared to those who did not develop pain. This result is consistent with other studies. Larivière et al. (2010) used a clinical LBP population and healthy controls and found

there to be no difference in muscle reflex latencies between these two groups, and used a sample size similar to our study. A different study using only a healthy population by Gregory et al. (2008) found that in prolonged standing, there was no significant difference in muscle reflex times pre- and post- a two-hour standing trial, nor were there significant differences between PDs and NPDs. This shows that there seem to be neuromuscular differences between those who have developed clinical LBP, and those who only develop pain transiently during prolonged sitting.

Work by Gregory et al. (2008) has shown that individuals who develop LBP during prolonged standing undergo greater muscle recruitment in response to a sudden, unexpected perturbation. While the study in this thesis also examined which muscles successfully activated during each quick-release, the analysis was limited by the inability to calculate the onset times for certain muscles. Specifically, noise, most likely the release harness interfering with EMG sensors and/or %MVC for some muscles being above 5% before the release, caused many muscles to not be counted as "turning on". While only one participant was removed from analysis due to no muscles having activity below 5% MVC, all other participants had at least one muscle which was below 5% MVC for their baseline muscle activity to calculate the muscle reflex onset time. In the study by Gregory et al. muscle responses were found in 95-100% of trials for participants with LBP, and in 73-86% of those without. Greater recruitment of low back muscles during an unexpected perturbation could lead to increased discomfort, and could be a factor in the development of LBP. While it would have been interesting to see if a similar observation was found in prolonged sitting for this study, having noise so frequently in the signal, as well as muscles already being activated prior to release, makes this comparison

difficult, as our study underreported the number of muscles which actually responded to the sudden perturbation. Furthermore, Gregory et al. (2008) used prolonged standing as the exposure, and muscle reflexes were elicited by dropping a 6.78 kg weight in a box to set the participant off balance. Therefore, these differences may contribute to why our results differ when it comes to muscle recruitment during a sudden perturbation. Future work could explore muscle recruitment during a quick-release after prolonged sitting further, to see if muscles respond similarly (or differently) after prolonged sitting as they did in prolonged standing with Gregory et al. (2008).

There were no significant differences in normalized joint angles for participants when examining their reflex time. While it may have been expected that those with long latencies would have exhibited a more flexed posture during sitting, due to increased passive tissue deformation of the lumbar spine, this was not observed. PDs and NPDs also had no significant differences in posture during the sitting trial, and there were no differences between sexes either. This lack of differences in posture and pain groups, as well as muscle reflex times, could be due to the length of the study not being long enough to fully elicit tissue creep. Other work has shown that tissue creep can occur by exposing an individual to sitting at 70% of their range of motion for an hour (Sanchez-Zuriaga et al., 2010). Perhaps the reduced amount of flexion during the two-hour sitting trial may not have been of a large enough magnitude to affect a statistically significant difference in muscle reflex onset timing. Further, the experience of transient pain, sex participant characteristics, and physical activity parameters also did not appear to effect muscle reflex onset times. Based on these results, it appears that both sitting posture and demographics will not pre-dispose an individual to have delayed

muscle reflexes when exposed to a sudden perturbation. Furthermore, other than PDs having a higher average maximum pain rating than NPDs, demographics, sitting posture, and muscle activity all did not differ amongst pain groups or sex during the actual sitting trial itself, with the exception of RTS activity being significantly different between men and women during the prolonged typing trial.. While there was a significant difference between men and women when it came to average muscle activity, with women having an average muscle activity of 6.86 %MVC (± 3.41) and men had an average of 4.41 %MVC (± 2.07), this significance does not mean much functionally. All participants had quite low levels of muscle activity (below 10% of maximum effort) and a difference of 2% MVC is not thought to make a practical difference. Further, greater muscle activity is typically thought to be associated with perceived discomfort; however, women were much more likely to be in the NPD group than the PD group.

All other variables collected during the study were insignificant when comparing muscle reflex times. Demographic information for participants was relatively homogenous, as the age range only went from 19-39 (only three participants were over 30), with an average age of 23.88, even though participants could be recruited up to age 69 (as limited by the GPAQ). Height in particular was also fairly similar amongst participants, with weight being more varied. However, when recruiting from a university population, this low average age is to be expected. Joint angles also did not significantly contribute to the delayed muscle reflexes. As sitting is a form of sub-maximal flexion over an extended period of time, it was expected that those who sat in a more flexed position would exhibit more tissue creep, and this may cause delayed muscle reflex times. However, this was not observed in a 2-hour exposure to

prolonged sitting. A longer intervention, or perhaps grouping participants into two groups, one sitting more in extension, and another more flexed, could determine if prolonged sitting is able to elicit enough tissue creep to also cause delayed muscle reflexes.

During analysis, it was found that 43.59% of participants (17) actually had a faster muscle reflex time in the perturbation post-sitting. Furthermore, the makeup of characteristics for those who did respond faster in the second release was fairly similar. Of the 17 that responded faster in the second release, nine were NPDs, and 11 were women.

Height may have acted as an influencing factor as to whether a person reacted slower or faster during the reflex tests. It was observed that people who were more likely to respond more quickly in the second quick-release were 5.98 cm shorter than those who responded slower. However, this result might not be practical as when people who were considered tall (people over 172cm) were compared to those considered short (under 172cm), both exhibited delayed muscle reflexes after sitting for 2-hours. Shorter people responded 8.38 ms slower after sitting for 2-hours, while taller people responded 17.27 ms slower. As was mentioned earlier as well, high variance in muscle reflex times contributed to why this delay in muscle reflexes was not significant. So, even when grouping participants by height, reflex times still had standard deviations in the range to 25 to 40 ms. This would infer that even with a significant difference in reflex times based on height, the result may not best describe why some participants respond more quickly or why some respond slower after prolonged sitting. Unfortunately, it seems that a variable which this study was not able to capture could be the contributing factor as to why people respond more or less quickly after sitting for two-hours as no other variable

significantly contributed to the determination of these groups. Furthermore, no studies have noted this observation.

Muscle reflex timing of the low back has many potential factors which may lead to delayed muscle reflexes. As was established earlier, these delayed reflexes may pre-dispose an individual to a soft tissue injury in the event of a sudden, unexpected perturbation (such as falling). So far, the literature has focused on populations which already have low back pain, or those who will develop pain in the future, with little focus on the effect prolonged, static postures have on low back reflexes. While Gregory et al. (2008) explored this in prolonged standing, this thesis is the first study to examine the effect of prolonged sitting on low back muscle reflex times in a young, healthy population. Based on what was observed, a two-hour exposure to sitting appears to increase the latency of spine muscle reflexes, but not significantly so. Reinforcing previous work, it does seem that transient PDs exhibit altered neuromuscular recruitment when exposed to a sudden perturbation, potentially identifying their future status as a chronic back pain developer, however this study does not provide enough information to discern this, plus there is no follow-up to see if PDs become future low back pain patients.

While our work has shown that a two-hour period of prolonged sitting appears to be safe in terms of altered reflex times for the low back, future work should investigate longer periods of time. Evidence suggests that workers in developed countries sit for almost three quarters of their workday (Clemes et al., 2014); therefore, exposures to these longer durations may result in significant differences in reflex times. Further, a longitudinal design would also provide the

ability to see if delayed muscle reflexes after sitting is associated with future cases of clinical low back pain. Clearly, something at the neuromuscular level is being affected by prolonged, static postures; however, what influences delayed muscle reflexes remains unknown. Delayed muscle reflexes are already an indicator for future development of LBP, and with this study identifying two distinct groups of people (those who respond more or less quickly after sitting), something that this study was not able to capture must be influencing why some people react more or less quickly than others.

Strengths and Limitations

The large sample size of this study is one if its greatest strengths. Many studies examining muscle reflex times have sample sizes below 20 participants, with only a handful of studies having a sample size similar, or greater to what this one entailed. This sample size allowed analysis of a greater number of variables and sub-grouping for the reflex test, such as normalized lumbar and pelvic angles, sex, pain group and pre/post sitting reflex times. Furthermore, with this sample size of 40 recruited participants, there was a quick-release success rate of 97.5% (39/40) for being able to calculate muscle reflex times.

This was also the first study to focus on prolonged sitting and muscle reflexes. The design of the jig allowed for a very subtle reflex event, whereas most other studies actively push or use a weight to pull a participant in a direction forward or backwards. Compared to some studies, this ensures that no portion of the perturbation may be impacted by another part of the body which isn't the low back. This study also made an effort to reduce potential bias based on the

method used to calculate muscle reflex times, as the standard deviation chosen by studies varies greatly from 1.4-3 SD of the mean. This study used the more conservative 3SD method, but made sure to check different size standard deviations (1.5-3 SD in increments of 0.5) to make sure differences in methodology did not vary greatly. Furthermore, only one person calculated all muscle reflexes, reducing bias had multiple people calculated them.

The posture of the participant being slumped in the harness during the quick release prevented the use of abdominal EMG. Some participants also struggled to get fully relaxed in the harness, even after extensive coaching prior to the quick release, preventing many cases of muscles already being activated prior to the release. A biofeedback device in front of the quick-release mechanism for the participant could have assisted in helping participants reduce their muscle activity and adjust their posture accordingly. Practice quick-releases may have helped reduce variability in muscle reflex times, and a lack of participants being familiar with the response prior to taking part may have influenced why some people responded more quickly in the post-sitting release. However, coaching for each release and due to the measurement obtained being a reflex which is not a conscious decision should have minimized this potential error in the first release.

The use of a harness which did not potentially interfere with the EMG also prevented adequate analysis of which muscles turned on during the quick release. Furthermore, with the jig, the design of the seated portion being angled twenty degrees towards the ground prevents any

kind of alternate reflex test, such as a sudden perturbation towards extension. However, this study was not concerned with that outcome, so it was less of an issue. Cable length from the jig to the participant also varied for each participant depending on their height. This variance in height and cable length led to different lengths of time between when the cable was released manually, to when the participant actually began falling forwards. This may have impacted reflexes, as the sound of the mechanical release may have resulted in the participant anticipating the event and firing their muscles in advance of a reflex. While the jig was designed to accommodate very tall and very short people, this cable length still prevented there from being a precise consistent amount of time between release and the participant falling. The angle of the jig also caused issues with some reflexes, as participants with larger abdominal mass would have their stomach resting on their knees prior to and during the release, preventing them from being able to fall very far and the response seemed more "muted" than thinner participants. While reflexes were still found in these individuals, it was still more challenging to elicit these reflexes, and skewed the demographic we were able to recruit from. Furthermore, this skewed younger demographic may have impacted the results, as a younger, healthy population may not exhibit reflex differences, as compared to an older demographic.

This study is also limited in that there will be no follow-up with participants to see if they develop chronic LBP in the future. Cholewicki et al. (2005) found that delayed muscle reflexes increased the risk of future low back injuries, but this study will not be able to measure long-term outcomes. Another limitation is how the sub-grouping of variables was

distributed. While there was a near even split for pain groups, there were a disproportionate number of women in the study, compared to men. To further compound this difference, when sub-grouped into PDs and NPDs, only three out of twenty NPDs were men, and most of the PDs were also men as a result.

The seated exposure we used in this study did not involve a backrest. Therefore, the results of this study may not be generalizable to a typical office setting. In a study examining task and seated posture, van Dieën et al. (2001) found that in typing tasks the backrest is minimally used by occupants; therefore, the limitation imparted to our data may have been mitigated by the fact that our task only involved typing.

Lastly, the length of the sitting exposure may not have been long enough to observe tissue creep, or to have an impact on low back muscle reflexes.

Future Directions

Future work could look at the contribution of the abdominal muscles during an unexpected perturbation, as Gregory et al. (2008) found that pain developers had increased usage of their abdominal muscles during the quick release. A follow-up study with participants may also provide further evidence towards delayed muscle reflexes being a contributing factor towards developing a low back injury. A larger sample size, with a more even ratio of men to women may also help the distribution of men and women in pain groups. A longer sitting exposure could also be used, as a longer exposure may be needed to see if some people continue to react

just as quickly after sitting for several hours or not. This longer exposure could contribute to reducing the high variability in reflex times, as there was much more variability in latencies after the exposure, as compared to before sitting.

Another study could examine the effect of different chairs, or more flexed postures during prolonged sitting on muscle reflex times. Theoretically, more flexed postures will contribute to more tissue creep, which could increase muscle reflexes much more quickly than the chair used in this study. For example, prolonged sitting in a simulated car seat, or having participants more slumped during the exposure period. In future studies of this nature, a greater effort should be focused on ensuring no noise can contaminate the signal during the release, as it appears increased muscle recruitment for the reflex could be one of the contributing factors to delayed reflexes, as well as discomfort.

Another direction for this type of research could examine interventions which may preserve muscle reflex times in those who react more slowly after prolonged sitting. For example, sit/stand work stations, or periodic walking. If future studies use this design for another prolonged sitting study, the work could further examine the influence of height on muscle reflexes during sitting. While the result was significant in this study, high variability in the data makes it difficult to accept this result with the data that was collected. However, a clearer definition and cut-off point for “tall” people and “short” people when entering the study may

help determine if this result was actually substantial, and could help explain which people are at a greater risk for back injuries after prolonged sitting.

Lastly, recruiting participants with chronic LBP, and healthy controls would help determine if prolonged sitting is even more detrimental to those with chronic pain or not. Studies could also look for a greater age range, as this study had a very narrow scope for recruited participants' ages, even though we attempted to recruit participants up to age 69.

Impact of Work

With this study, it can be seen that sitting for two hours will not significantly reduce the ability of back muscles to respond to a sudden perturbation. This is also the first piece of evidence confirming a two-hour exposure to sitting does not have an impact on these reflexes. While prolonged sitting can elicit back pain in some people, this study shows muscle reflexes are not the contributing factor to this pain, or injury. As this is the first study exploring prolonged office chair sitting, future work can build off of the framework of this study and can contribute to establishing ergonomic guidelines on prolonged sitting.

This study was also the first to report the distinction between two unique groups of people when responding to a sudden perturbation. By identifying that some people are able to conserve their reflex latencies after prolonged sitting, while others develop significantly longer reflex times after prolonged sitting, it is clear that something at the neuromuscular level is

occurring to cause this. However, it is unclear if it is something biomechanical, genetic, or some other pre-disposing factor which could be causing this.

Conclusion

Prolonged sitting for a two-hour session does not impact the ability of the low back to respond to a sudden perturbation. In line with previous work on muscle reflexes, women tend to have quicker muscle reflex times than men, however insignificantly so, much like how NPDs tend to have faster reflex times as compared to PDs. Biomechanical and neuromuscular parameters during prolonged sitting also do not differ between pain groups or between sexes. While it was expected that transient PDs would exhibit delayed muscle reflex times as compared to NPDs, a difference was observed, but not significantly so. Pain groups and sex were also difficult to compare, as the distribution of men and women in pain groups was skewed more to one sex in each group. While this study used a large sample size compared to others in the literature, future work studying muscle reflexes should utilize a larger sample size, and attempt to have a more even ratio or men and women when recruited. A larger sample size could help reduce the variability in reflex times, and could help evenly distribute sex representation in pain groups.

Furthermore, there were no differences observed between sexes or pain groups when it came to neuromuscular or biomechanical function during the prolonged typing trial. Distinct pain groups (PDs and NPDs) were identified during the typing trial, with PDs having a significantly higher average maximum pain rating as compared to NPDs. While pain groups were nearly separated into even groups, this aligned with previous work where transient pain developers were observed in approximately half of the recruited participants.

During data analysis, it was observed that participants fell into two distinct groups, those who respond more quickly, and those who responded more slowly after the typing trial to the quick-release. Future work can determine if this was merely a coincidence, or if there is something else contributing to why some people are more likely to have delayed or quicker muscle reflexes after sitting. A longer exposure which more closely mimics an occupational sitting (such as sitting for three- or four-hour periods) should also be examined. As a longer exposure may have a greater impact on muscle reflexes, causing an increased risk of predisposing a person to injury due to their back not being able to adequately stiffen itself in time when exposed to a sudden perturbation.

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Appendix 1

Health History Questionnaire, used as a screening tool to exclude participants from the study who identify as having had a back injury. Questionnaire includes the individuals self reported health history, family history, and current subjective amount of pain.

Health Screening Form:

STUDY: The Effect of Prolonged Sitting on Neuromuscular and Biomechanical Responses of the Low Back in Healthy Individuals

Subject Code: _____

This questionnaire asks some questions about your health status. This information is used to guide us with your entry into the study as well as provide health data that will help us learn more about sitting-induced back pain.

Exclusion criteria to participating in this study include:

1 A history of back injury (such as a fracture or disc herniation), infection (such as osteomyelitis), arthritis (ie. osteoarthritis, rheumatoid arthritis or psoriatic arthritis) or spine surgery.

Past Relavent Health History (please check all that apply)

- [ ] Back Injury (soft tissue), please specify: _____
- [ ] Back Injury (fracture), please specific: _____
- [ ] Low Back Pain
- [ ] Disc Herniation
- [ ] Disc Bulge
- [ ] Vertebral End Plate Fracture
- [ ] Scoliosis, known severity: _____
- [ ] Spondylolisthesis
- [ ] Pars Defect
- [ ] Scheuermann's Disease
- [ ] Transitional Vertebrae
- [ ] Congential Vertebral Abnormality
- [ ] Arthritis
- [ ] Cancer
- [ ] Leg Pain
- [ ] Surgeries, please specify: _____

Recent Health History (*within the past six months, including date of injury/ pain*, please check all that apply):

- [ ] Back Injury (soft tissue), please specify: _____
- [ ] Back Injury (fracture), please specify: _____
- [ ] Low Back Pain
- [ ] Disc Herniation
- [ ] Disc Bulge
- [ ] Leg Pain

At This Moment, Rate the Level of Pain You Feel in Your Low Back (mark a vertical dash along the line)

no pain ***worst pain***
0 **100**

_____

Family History of Low Back Pain

Does anyone in your family besides yourself have a history of low back pain? (circle one):
YES NO Don't Know
If yes, how many family members are affected? (circle one):
1-3 4-7 8-10 11 or more Don't Know
Are those affected on one side of the family or both? (circle one)
mother's side only father's side only both sides Don't Know

Appendix 2

Global Physical Activity Questionnaire

GPAQ

Physical Activity

Next I am going to ask you about the time you spend doing different types of physical activity in a typical week. Please answer these questions even if you do not consider yourself to be a physically active person.

Think first about the time you spend doing work. Think of work as the things that you have to do such as paid or unpaid work, study/training, household chores, harvesting food/crops, fishing or hunting for food, seeking employment. *[Insert other examples if needed].* In answering the following questions 'vigorous-intensity activities' are activities that require hard physical effort and cause large increases in breathing or heart rate, 'moderate-intensity activities' are activities that require moderate physical effort and cause small increases in breathing or heart rate.

	Questions	Response	Code
Activity at work			
1	Does your work involve vigorous-intensity activity that causes large increases in breathing or heart rate like *[carrying or lifting heavy loads, digging or construction work]* for at least 10 minutes continuously? *[INSERT EXAMPLES] (USE SHOWCARD)*	Yes 1 No 2 *If No, go to P 4*	P1
2	In a typical week, on how many days do you do vigorous-intensity activities as part of your work?	Number of days ⌊_⌋	P2
3	How much time do you spend doing vigorous-intensity activities at work on a typical day?	Hours : minutes ⌊_⌋⌊_⌋ : ⌊_⌋⌊_⌋ hrs mins	P3 (a-b)
4	Does your work involve moderate-intensity activity that causes small increases in breathing or heart rate such as brisk walking *[or carrying light loads]* for at least 10 minutes continuously? *[INSERT EXAMPLES] (USE SHOWCARD)*	Yes 1 No 2 *If No, go to P 7*	P4
5	In a typical week, on how many days do you do moderate-intensity activities as part of your work?	Number of days ⌊_⌋	P5
6	How much time do you spend doing moderate-intensity activities at work on a typical day?	Hours : minutes ⌊_⌋⌊_⌋ : ⌊_⌋⌊_⌋ hrs mins	P6 (a-b)
Travel to and from places			
The next questions exclude the physical activities at work that you have already mentioned. Now I would like to ask you about the usual way you travel to and from places. For example to work, for shopping, to market, to place of worship. [insert other examples if needed]			
7	Do you walk or use a bicycle (*pedal cycle*) for at least 10 minutes continuously to get to and from places?	Yes 1 No 2 *If No, go to P 10*	P7
8	In a typical week, on how many days do you walk or bicycle for at least 10 minutes continuously to get to and from places?	Number of days ⌊_⌋	P8
9	How much time do you spend walking or bicycling for travel on a typical day?	Hours : minutes ⌊_⌋⌊_⌋ : ⌊_⌋⌊_⌋ hrs mins	P9 (a-b)
Recreational activities			
The next questions exclude the work and transport activities that you have already mentioned. Now I would like to ask you about sports, fitness and recreational activities (leisure), [insert relevant terms].			
10	Do you do any vigorous-intensity sports, fitness or recreational (*leisure*) activities that cause large increases in breathing or heart rate like [*running or football,]* for at least 10 minutes continuously? *[INSERT EXAMPLES] (USE SHOWCARD)*	Yes 1 No 2 *If No, go to P 13*	P10
11	In a typical week, on how many days do you do vigorous-intensity sports, fitness or recreational (*leisure*) activities?	Number of days ⌊_⌋	P11
12	How much time do you spend doing vigorous-intensity sports, fitness or recreational activities on a typical day?	Hours : minutes ⌊_⌋⌊_⌋ : ⌊_⌋⌊_⌋ hrs mins	P12 (a-b)

Continued on next page

GPAQ, Continued

Physical Activity (recreational activities) contd.			
Questions		**Response**	**Code**
13	Do you do any moderate-intensity sports, fitness or recreational (*leisure*) activities that causes a small increase in breathing or heart rate such as brisk walking,(*cycling, swimming, volleyball*)for at least 10 minutes continuously? *[INSERT EXAMPLES] (USE SHOWCARD)*	Yes 1 No 2 *If No, go to P16*	P13
14	In a typical week, on how many days do you do moderate-intensity sports, fitness or recreational (*leisure*) activities?	Number of days ⌊_⌋	P14
15	How much time do you spend doing moderate-intensity sports, fitness or recreational (*leisure*) activities on a typical day?	Hours : minutes ⌊_⌋⌊_⌋ : ⌊_⌋⌊_⌋ hrs mins	P15 (a-b)
Sedentary behaviour			
The following question is about sitting or reclining at work, at home, getting to and from places, or with friends including time spent [sitting at a desk, sitting with friends, travelling in car, bus, train, reading, playing cards or watching television], but do not include time spent sleeping. *[INSERT EXAMPLES] (USE SHOWCARD)*			
16	How much time do you usually spend sitting or reclining on a typical day?	Hours : minutes ⌊_⌋⌊_⌋ : ⌊_⌋⌊_⌋ hrs min s	P16 (a-b)

Appendix 3

Recruitment poster for the study

Participants Needed For a Biomechanics Research Study

Inclusion Criteria:

- Those who have not recently experienced an episode of low back pain (20 participants).
- 4 hours of available time to participate in the study
- Must be between the ages of 18-69

Contact Info:

Ryan Greene
rdg068@mun.ca
munbiomechanics@gmail.com
709-777-7334

What's Involved??

- Complete Questionnaires (Health History Form, and activity level).
- Have sensors attached to your back and torso
- Complete sensor calibration tasks (such as range of motion and maximum muscle effort)
- Test muscle reflex response
- Type for 2 hours at a computer workstation
- While you sit, measures of muscle activity, perceived discomfort, back posture and motion capture will be collected.
- You will receive a $10 remuneration as a thank you for your time and offered free parking during the study.

MEMORIAL UNIVERSITY
Faculty of Medicine/Discipline of Medicine

Biomechanics Study
rdg068@mun.ca
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Appendix 4

In-class recruitment script and email script.

Hello,

My name is Ryan Greene. I am a Masters student in the Discipline of Medicine at Memorial University of Newfoundland working under the supervision of Dr. Diana De Carvalho. I am leading a study examining the effects of prolonged sitting and muscle reflex onset time; as well as the neuromuscular and biomechanical differences between those who develop pain while sitting and those who do not.

Participants needed:

Forty participants, 20 men and 20 women, with no history of back pain will be recruited from the local population.

Time commitment for this study:

Participation will involve a laboratory study that will take approximately 4 hours.

What the study involves:

Upon arrival at the laboratory, participants will be asked to fill out two questionnaires (a health history checklist and The Global Physical Activity Questionnaire). Afterwards you will be equipped with EMG and electrodes, accelerometers, and infrared sensors for motion capture. You will then be asked to sit in a custom-made jig, angled at 20 degrees towards the ground while being strapped in and a harness will be attached to your torso. The release of the harness will allow us to measure your muscle reflex response. This will be done before and after sitting at a computer workstation working on a standardized typing activity. During this

sitting trial we will ask you to complete perceived ratings of low back pain on a scale and posture, and back muscle activity will be recorded continuously.

Implications of the study:

This research may lead to a better understanding of the biomechanical and neuromuscular impacts from prolonged sitting in healthy individuals, such as muscle reflex onset time after being exposed to sitting. This study will also examine the neuromuscular and biomechanical differences between those who develop back pain while sitting and those that do not. By better understanding sitting and its impact on the back, we can develop targeted intervention and prevention strategies to prevent the possibility of an injury.

Contact information:

If you are interested in taking part in the study, or would like more information, please contact me directly at rdg068@mun.ca, munbiomechanics@gmail.com or 709-777-7334.

This study has been reviewed and received ethics approval from the provincial Health Research Ethics Board.

Cheers,

Ryan Greene

Appendix 5

Informed consent letter.

Faculty of Medicine/Discipline of Medicine

Consent to Take Part in Research

TITLE: The Effect of Prolonged Sitting on Neuromuscular and Biomechanical Responses of the Lower Back in Healthy Individuals

INVESTIGATOR(S):

Principal Investigator: Ryan Greene

MSc Student (Clinical Epidemiology)

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Faculty of Medicine, Discipline of Medicine

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You have been invited to take part in a research study. Taking part in this study is voluntary. It is up to you to decide whether to be in the study or not. You can decide not to take part in the study. If you decide to take part, you are free to leave at any time.

Before you decide, you need to understand what the study is for, what risks you might take and what benefits you might receive. This consent form explains the study.

Please read this carefully. Take as much time as you like. If you like, take it home to think about for a while. Mark anything you do not understand, or want explained better. After you have read it, please ask questions about anything that is not clear.

The researchers will:

- discuss the study with you
- answer your questions
- keep confidential any information which could identify you personally
- be available during the study to deal with problems and answer questions

1. Introduction/Background:

Low back pain is a common disorder, affecting many people at some point in their life. Many people find that sitting for long periods of time uncomfortable. This may be related to how the back is bent in sitting. This posture stretches muscles and ligaments in the back and can lead to pain and delayed muscle response. A lack of stiffness in the back puts tissue at an increased risk of becoming injured because of this muscle delay. Studies have shown that a delayed muscle response occurs when the back is held at end range bending. Currently, no research has been done to see if lower amounts of back bending, as in chair sitting, delay muscle response time.

2. Purpose of study:

To determine the impact a two-hour exposure to office chair sitting has on biomechanical and neuromuscular variables (muscle reflex onset time, and spine stiffness) in healthy individuals. Secondly, we will be looking to see if biomechanical and neuromuscular differences (seated

posture, muscle activity, perceived pain rating, spine stiffness and muscle reflex onset time) occur between those who do and do not develop short-lived back pain while sitting.

3. Description of the study procedures:

For this study you will be asked to wear comfortable gym-type clothing (pants are fine).

Completing Questionnaires:

- At the start of the study you will be asked to complete two questionnaires. This will take you about 10 minutes.

	Questionnaires	**What is asked?**
1	Health history form	Your health history and current back pain level
2	Global Physical Activity Questionnaire	How active you are

Next, some sensors will be attached to your back to record how hard your muscles are working and how much your back is moving. You will be asked to wear a shirt with no back in it so that sensors can be easily attached.

Sensors that will be attached to you:

- To record how hard your muscles are working, sensors will be attached to the skin of your upper, mid and lower back with tape. We will need to clean the skin with alcohol to remove any oils, dirt or dead skin cells before the sensors are put on. Any hair located where electrodes will be attached will need to be shaved so the signal does not get distorted.

- To record the shape of your back in different positions, two sensors will be attached to the skin over the bones of your mid and lower back using tape.

- To record the position of your body in space, clusters of motion capture sensors will be attached to the head (via Velcro headband), thorax, upper arms and forearms, and the sacrum.

Getting ready for the experiment

- There are a number of factors that affect recordings of muscle activity from muscle sensors. In order to compare your muscle activity with other people in the study we need to know two things. What your muscle activity is <u>when you are relaxed</u> and <u>when you are using them as much as you can</u>.

- To do this we need to collect 4 short trials of data: 3 hardest effort trials and 1 quiet trial.

    - For the quiet trial you will lie down on your stomach and relax as much as possible. We will then take a five second recording of your muscle activity.

    - For the hardest effort trials you will lie down on your stomach so that only your hips and legs are on the height-adjustable table. An assistant will support your legs so you are secure. You will be low enough that you can reach the floor

with your hands to hold up your upper body until the trial begins. During each hardest effort trial you will try to push your back up towards the ceiling as hard as you can. While you are doing this, an assistant will push down on your shoulders with just enough pressure to keep you in line with the floor so you don't over arch your back. Each hardest effort trial will be 5 seconds. There will be a rest break of 2 minutes in between each trial to make sure you do not get tired.

- In order to compare your body postures to others in the study, we need to know four things about your normal movement. We will therefore ask you to do the following:
    - Stand
    - Bend forward as much as possible
    - Bend backwards as much as possible
    - Bend forward as much as possible while you are sitting.

- In order to create a computerized model of your body position within our laboratory, we need to take a few short calibration trials:
    - Standing still with your arms held out to your sides in a "T" pose.
    - Rolling your shoulders around in circles
    - Bending your elbows
    - Rolling your head around in circles

Experiment

- To determine muscle reflex onset time we will be measuring how long it takes your back muscles to turn on with a quick balance trial. We will use gravity to cause you to loose your balance momentarily, automatically turning on your low back muscles stop the motion.
- To do this, you will be seated in a custom made apparatus with a seat angled forward 20 degrees and a footrest for your feet. There are handles by the seat pan for you to hold on to and a lab belt will be used to secure your lower body. We will attach a custom-made harness to your torso. This harness is attached to a cable that is secured

on the wall. At an unknown time, the cable will be released and your upper body will move forward briefly with gravity (for a maximum of 10 degrees rotation). This will cause a normal reflex that turns your back muscles on quickly to balance yourself. If, for any reason, you are unable to balance yourself, a safety cable will engage automatically after you have rotated 10 degrees forward which will stop the motion.

- You will then be seated at the experiment workstation. This includes a standard office chair and desk with a computer, monitor and keyboard. The height of the chair, monitor and keyboard will be changed to fit you according to standard ergonomic guidelines.

- You will then be shown the typing program you will use for the experiment. This program includes a large text box that you will type in. Once the experiment starts, paragraphs of a report will appear above. You will be asked to copy this text into the text box. When you have typed in the paragraph that is shown you will click "enter" and a new paragraph to record will appear. You will continue copying out the text throughout the entire study, working at your own pace.

- Once you are set at your workstation, just before the study starts, you will be asked to complete another pain rating scale. You will also be asked to repeat this scale every 7.5 minutes throughout the study.

- During the sitting study you can move around in your seat, but you will not be able to stand up for two hours.

- After sitting for two hours, we will ask you to perform another trial of maximum low back flexion and a second balance trial using the custom jig.

- At the end of the sitting study, the sensors will be removed from your back and you will be free to go.

Photographs

- If you sign the consent for dissemination of photographs form, one picture of your back with the equipment attached will be taken while you are sat in the chair. The purpose of this is to allow the researcher to see how the equipment is attached to your back at a future date. Pictures are also helpful to have for the purposes of publications and conference presentations. This picture will be taken using a laboratory camera, and the data will be stored on secure computers along with the rest of the study data.

4. Length of time:

You will need to make a one-time visit to the laboratory for this study. It is estimated that this visit will take up to 4 hours of your time.

5. Possible risks and discomforts:

- There is always a risk of feeling sore when sitting for a long time. However, the risks in this study are not greater than those associated with everyday sitting tasks. If you do feel sore after the sitting study you will likely feel better as soon as you get up and move around.

- Turning your muscles on as much as you can is of similar intensity to moving heavy objects around at home or a workout at the gym. You may feel sore after because of this, but you will feel better within three days.

- Some people may get a mild skin irritation/redness from the tape used to attach the sensors to their skin. If this happens the symptoms typically fades within 2-3 days. If you have skin sensitivities, or have had reactions to tape in the past, please inform the research team.

- There is potential that you may be surprised or uncomfortable with the experience of being off balance for a brief period of time when undergoing the muscle reflex trial.

6. Benefits:

It is not known whether this study will benefit you.

7. Liability statement:

Signing this form gives us your consent to be in this study. It tells us that you understand the information about the research study. When you sign this form, you do not give up your legal rights. Researchers or agencies involved in this research study still have their legal and professional responsibilities.

8. What about my privacy and confidentiality?

Protecting your privacy is an important part of this study. Every effort to protect your privacy will be made. However it cannot be guaranteed. For example we may be required by law to allow access to research records.

When you sign this consent form you give us permission to

- Collect information from you
- Share information with the people conducting the study
- Share information with the people responsible for protecting your safety

Access to records

The members of the research team will see study records that identify you by name. Other people may need to look at the study records that identify you by name. This might include the research ethics board. You may ask to see the list of these people. They can look at your records only when supervised by a member of the research team.

Use of your study information

The research team will collect and use only the information they need for this research study.

This information will include your

- age
- sex
- medical conditions
- information from study questionnaires

- muscle activity during sitting
- back bending during sitting
- pain levels during sitting
- pressure information regarding how you sit on the chair
- calf circumference before and after sitting

Your name and contact information will be kept secure by the research team in Newfoundland and Labrador. It will not be shared with others without your permission. Your name will not appear in any report or article published as a result of this study.

Information collected for this study will be kept for twenty-five years and then will be destroyed. The reason it is kept for this length of time is so that data can potentially be re-analyzed if any new technology is developed that allows further processing.

If you decide to withdraw from the study, the information collected up to that time will be destroyed.

Information collected and used by the research team will be stored in a locked room (HSC 5315). Dr. Diana De Carvalho is the person responsible for keeping it secure.

Your access to records
You may ask the researcher to see the information that has been collected about you**.**

9. Questions or problems:

If you have any questions about taking part in this study, you can meet with the

investigator who is in charge of the study. That person is:

Ryan Greene (709) 777-7334

Or you can talk to someone who is not involved with the study at all, but can advise you on your rights as a participant in a research study. This person can be reached through:

Ethics Office at 709-777-6974
Email at info@hrea.ca

This study has been reviewed and given ethics approval by the Newfoundland and Labrador Health Research Ethics Board.

After signing this consent you will be given a copy.

Signature Page

Study title: Effect of an "active" office chair on spine biomechanics and perceived pain during prolonged sitting

Name of principal investigator: Ryan Greene

To be filled out and signed by the participant:

	Please check as appropriate:
I have read the consent and information sheet.	Yes { } No { }
I have had the opportunity to ask questions/to discuss this study.	Yes { } No { }
I have received satisfactory answers to all of my questions.	Yes { } No { }
I have received enough information about the study.	Yes { } No { }
I have spoken to Mr. Greene and he has answered my questions	Yes { } No { }
I understand that I am free to withdraw from the study	Yes { } No { }

- at any time
- without having to give a reason
- without affecting me in any way

I understand that it is my choice to be in the study and that I may not benefit. Yes { } No { }

I understand how my privacy is protected and my records kept confidential Yes { } No { }

I agree to take part in this study. Yes { } No { }

_____ _____ _____

Signature of participant Name printed Year

Month Day

To be signed by the investigator or person obtaining consent

I have explained this study to the best of my ability. I invited questions and gave answers. I believe that the participant fully understands what is involved in being in the study, any potential risks of the study and that he or she has freely chosen to be in the study.

_____ _____ _____

Signature of investigator Name printed Year Month Day

Telephone number: _____

Appendix 5: Consent form to participate in research.

Signature Page – Dissemination of Photographs

Study title: The Effect of Prolonged Sitting on Neuromuscular and Biomechanical Responses of the Lower Back in Healthy Individuals

Name of principal investigator: Ryan Greene

To be filled out and signed by the participant:

	Please check as appropriate:
I agree to be photographed	Yes { } No { }
I understand that my face, and any identifying characteristics (e.g. tattoos) will be blurred.	Yes { } No { }
I agree to having these blurred photographs used for teaching and publication (conference presentations and journal publications).	Yes { } No { }

_____ _____ _____

Signature of participant Name printed Year

Month Day

To be signed by the investigator or person obtaining consent

I have explained this study to the best of my ability. I invited questions and gave answers. I believe that the participant fully understands what is involved in being in the study, any potential risks of the study and that he or she has freely chosen to be in the study.

_____ _____ _____

Signature of investigator Name printed Year Month Day

Telephone number: _____

Appendix 6: Consent form, obtaining consent to take a photo of the participant for the study for possible publication use, or for a reference when looking at the participants EMG and accelerometer setup.