THE EFFECT OF PROLONGED SITTING ON TRUNK MUSCLE AND KINEMATICS RESPONSES TO SUDDENLY APPLIED LOADS

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ABSTRACT

Prolonged sitting facilitates individuals to adopt detrimental postures that may lead to the development of low back discomfort. Interestingly, subjecting individuals to unexpected trunk perturbations can reveal altered trunk muscle activation, kinematics, and kinetics. In this research participants were exposed to two-1hr prolonged sitting exposures. Prior to, between, and post sitting exposures a 6.78 kg load was unexpectedly applied to the participants' hands to perturb them. Trunk muscle electromyography, trunk kinematics, and centre of pressure (of the feet) were recorded during these unexpected perturbations. Participants who developed low back discomfort displayed increased muscle activation and longer muscle latency times in response to the unexpected perturbations. Likewise, these participants also displayed decrease centre of pressure and trunk angle displacement. Therefore, the responses to the unexpected perturbations displayed innate neuromuscular actions of the trunk that appear to increase the likelihood of an individual developing low back discomfort during prolonged sitting.

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1.0 INTRODUCTION

Low back pain (LBP) is a major concern in the sedentary work environment. Prolonged sitting exposures have been shown to elicit pain in office chair users during work-related tasks on a computer (Gerr *et al.*, 2002), and contribute to low back musculoskeletal disorders (MSDs) in the workplace (Vos *et al.*, 2017). A prolonged sitting posture, which is a sedentary position held by an individual for a length greater than 30 minutes was shown to induce musculoskeletal pain (Andersen *et al.*, 2007). In most of these environments it is quite unavoidable to be in a seated posture for a prolonged period of time. It has been acknowledged in biomechanics and ergonomics research, that these postures result in prolonged deep flexion of the trunk and altered neuromechanical responses of trunk musculature that contribute to the development or occurrence of low back pain and musculoskeletal disorders of the low back. Specifically, these sustained postures lead to impaired spinal stability.

Spine stability is often described as a complex mechanism involving three essential components: spinal muscles, passive spinal tissues and neuromuscular control (Panjabi, 1992). These seated postures result in greater lumbar spine flexion and low static loading (De Carvalho *et al.*, 2015). Therefore, seated postures held for prolonged periods cause detrimental neuro-mechanical alterations to the body such as: creep of the passive elements and latent trunk muscle responses and a lack of neuromuscular control of the trunk (Panjabi, 1992). The postural changes of the spine as a consequence of prolonged sitting can cause a decrease in passive stability due to creep as well as decrease active stability through fatigued muscle and/or latent muscle responses. Thus, the neuromuscular system compensates by trying to increase the activation of remaining spinal components, both the passive and active components. Unfortunately, this may lead to high co-contraction of trunk musculature. The simultaneous contraction of two or more muscles

around a joint is defined as co-contraction (Schinkel-Ivy *et al.,* 2013). Moreover, it has been hypothesized that changes in muscle recruitment patterns, like co-contraction, act as compensation for spinal instability resulting from passive elements laxity or reduced neuromuscular control. Co-contraction initially predisposes individuals to LBP and tries to further to alleviate the pain and reduce spine instability by trying to increase trunk stiffness and thus the cycle perpetuates. Spinal instability involving deformation of passive spinal tissues has been associated with the development or occurrence of low back pain (LBP) and disorders.

When subjected to unexpected external perturbations of the trunk, muscle activation is delayed, leaving the spine with reduced stability for few milliseconds (Abboud *et al,,* 2017). Moreover, spinal stability is refered to as the active muscles surrounding the spine, passive spinal tissues, and neuromuscular control working in cohesion with one another (Panjabi, 1992). Therefore, the spinal response to perturbations is controlled by the overall stiffness of the passive spine, muscular activation and neural control. In concert with prolonged sitting, these instabilities and responsorial actions of the trunk in response to perturbations may be exposed. For example, low back extensor muscles are required to generate more active forces in weight holding after static flexion to compensate for the reduced contribution of creep deformed tissues in maintaining spinal stability. The degraded force generating capacity of the fatigued muscles can be a significant risk factor for low back pain.

Prolonged sitting exposures have been shown to affect muscle activity and negatively change postural responses of trunk and lower limb musculature during sitting and increase the risk of developing MSDs in individuals exposed to prolonged sitting. During prolonged sitting, the absence of postural adjustments is strongly associated with increased risk of developing lower back pain because of the passive tissue loading of the trunk (McGill, 1992). While seated, postural

changes that occur are greater lumbar spine flexion (Callaghan, 2016), and low static loading which are associated with the development of lower back discomfort (LBD) (Nairn et al., 2013). Furthermore, seated exposures place the lower back passive tissues into significant flexion leading to greater tissue creep in these tissues and subsequent decrease in spinal stability and stiffness (Rogers and Granata, 2006). In mechanical engineering, creep is defined as is the tendency of a solid material to move slowly or deform under the influence of persistent mechanical stresses. In biomechanics, it is the mechanical stress of prolonged static stretching of passive tissues that deform and cause the degradation of their viscoelastic properties. The presence of creep has detrimental tendencies, creep of passive spinal tissues due to prolonged sitting can produces more laxity in the lumbar spine and impair spinal stability. Previous in vivo and in vitro sitting research has shown large postural changes (Callaghan, 2016) and tissue loading adaptations (McGill, 1992) occur in prolonged sitting, which is an indicator of pain. There are many underlying mechanisms such as postural changes and muscle activity adaptations associated with prolonged sitting which has been shown to increase the risk of injury in prolonged sitting exposures. For example, individuals subjected to prolonged sitting produce increased reflex amplitude and longer muscle latency times in trunk musculature (Abboud *et al.,* 2017).

While prolonged sitting is a contributor to LBP and MSDs of the lower back, another major risk factor is a suddenly applied loads to the trunk. Peak muscle forces in the trunk are much greater in sudden loading than in static conditions, and even more so when sudden loading is unexpected (Marras *et al.,* 1987). In unexpected sudden loading conditions, muscle activation is delayed and thus leaves the spine with impaired stability for milliseconds. Unfortunately, even this fractional amount of time is enough to elicit the onset of LBP. Sudden applied loads to the trunk can be threatening to the health of the lower back if unexpected. In concert with prolonged sitting

exposures, perturbations can pose an even greater risk to individuals. Those that experience LBP have negative muscular responses to sudden trunk perturbations, specifically longer muscle activation reaction times (Cholewicki *et al.,* 2002). Moreover, it has been hypothesized individuals who develop LBP display lack of neuromuscular control of trunk musculature to compensate for decreased spinal stability (van Dieen *et al.,* 2003) The combination of LBP and prolonged sitting exposures lead to negative neuromuscular adaptions to the trunk as compensation to decreased spinal stability.

Prolonged sitting exposures held for prolonged periods causes injurious neuro-mechanical alterations to the body such as: creep of the passive elements and latent spinal muscle responses. Sustained lumbar flexion in concert with neuro-mechanical changes opens the susceptibility of the occurrence and development of LBP. Creep and latent spinal muscle responses interfere with spinal stability. Spinal instability has been investigated as a risk factor for LBP and injury. Borghuis *et al.* (2010) defined trunk stability as the ability of the neuromuscular system to maintain or resume an upright position of the trunk in the presence of disturbances. The spinal response to perturbations is controlled by the overall stiffness of the passive spine, muscular activation and neural control (Panjabi, 1992). Likewise, this research will examine if there is a relationship between prolonged sitting exposures and detrimental muscular and kinematic responses to sudden trunk perturbations.

2.0 RESEARCH QUESTIONS AND HYPOTHESIS

This research was designed to examine the variability in trunk muscle activation, kinematics and kinetics to suddenly applied loads post prolonged sitting exposures. The primary purpose of this research was to advance our knowledge of the electromyographic, kinematic and kinetic

responses of the trunk musculature, with a specific focus on the lumbar erector spinae (LES) prior to, during and post prolonged sitting. Moreover, another purpose of this research was to examine differences in muscular, kinematic and kinetic responses to sudden loading between pain developers and non-pain developers as well as between sex. A secondary purpose to this research was to evaluate the anticipatory and responsorial actions of postural muscles in situations of sudden loading of the hands.

It is vital to have a more thorough understanding of the stabilizing mechanisms of the trunk musculature and their function in neuromuscular control in response to unexpected perturbations. General guidelines and recommendations in alike ecological situations will become possible with the unearthing of these response mechanisms. Methods to address this purpose will include the trunk muscle activity, trunk spine angles, limb angles, center of pressure (COP) and applied vertical ground reaction forces (VGRF) pre, during and post prolonged sitting exposures. The research will also examine sex difference in perturbation response, maximal muscle activation, muscle activation onset times, COP displacement, and trunk angular displacement in response to suddenly applied loads. The specific questions and related hypotheses that will be addressed are:

1. Will prolonged seated exposures lead to detrimental muscular and kinematics responses to a suddenly applied load?
    - **Ha:** Prolonged sitting will cause an increase amount of erector spinae muscle activation and decreased amount trunk flexion. Gregory *et al.,* 2008, reported the probability of activation in response to unexpected perturbations for left LES and right TES significantly increased. Therefore, with the increase amount of erector spinae activity in response to unexpected loading, trunk flexion will be minimized.

2. Will pain developers experience impaired muscle activation and kinematics responses compared to non-pain developers?
    - **Hb:** Pain developers will experience increase amount of muscle activation from the erector spinae and rectus abdominis. Previous literature has found that patients with acute LBP, have an increase in muscle activation of the erector spinae and rectus abdominis muscles (Lariviere *et al.,* 2010; Jones *et al.,* 2012).
3. Will muscle activation, kinematics and kinetic responses be different between sex?
    - **Hc:** Females will experience increase amount of muscle activation from the trunk muscles in response to be unexpected perturbations. Females adopt more altered postures while seated than males, therefore this may lead to different responsorial actions of the trunk (Dunk and Callaghan, 2005).
4. Will muscle onset times and COP displacement be negatively altered post prolonged sitting exposure?
    - **Hd:** Muscle onset times will increase post prolonged sitting for the trunk musculature and COP displacement will also decrease. Reeves *et al.,* 2005, found a significant increase in muscle latency time of the erector spinae and EO. Similarly, it has been found that with unexpected perturbations COP displacement decreases (Grondin and Potvin, 2009).

2.1 RESEARCH CONTRIBUTION

A vast amount of research in the perturbation and prolonged sitting fields involve the assessment of different environmental conditions with minute cohesion between both fields. Prolonged sitting is underrepresented in the perturbations field. However, the current research aims to ameliorate

this deficit and bridge the gap between unexpected perturbations and prolonged sitting effects on trunk muscle activations, kinematics and kinetics. Specifically, outlining the detrimental effects of prolonged sitting on the trunk responses to unexpected perturbations. These results will give insight on potential guidelines and recommendations to sedentary work environments.

3.0 METHODS

3.1 OVERVIEW OF PROTOCOL

Participants completed a prolonged sitting protocol lasting approximately 2 hours. The experiment involved the participants sitting for two 1-hour blocks. Prior to, in between, and following each 1-hour seated exposure, participants completed two blind unexpected suddenly applied loads while standing on a force platform. Electromyography (EMG), kinematic analysis, force, centre of pressure and visual analog scale (VAS) tool were collected to quantify muscle activation, whole-body kinematics, centre of pressure distribution, and self-reported pain measures respectively. All procedures were approved by York University's Office of Research Ethics, certification #: e2017 – 397, and participants were asked to review and sign the informed consent form prior to the collection of any data.

3.2 PARTICIPANTS

A total of thirty participants (15 females and 15 males) between 18 and 30 years of age were recruited to participate in the study. The mean ± SD age, body mass, and height were 23.2 yrs ± 2.9, 70.4kg ± 12.9, and 1.71m ± (0.08), respectively. Additionally, anthropometric data, body mass index (BMI), bioelectrical impedance analysis (BIA), resting heart rate (RHR) and waist circumference (mean ± SD) for male and female participants are listed in Table 1. Participants

were required to have had no history of LBP or injury within the previous 12 months that caused them to miss school, work, and/or seek healthcare; and to had have no upper or lower extremity pain and/or injury. Participants were asked to wear a t-shirt/tank-top/sports bra and shorts/tights (to facilitate electrode and motion tracking sensor placements).

Table 1. Mean (SD) anthropometric data for all university aged participants, including females and males independently. Including body mass index (BMI), bioelectric impedance amplitude (BIA), and resting heart rate (RHR).

Anthropometrics	**Females** n = 15	**Males** n = 15	**All** n = 30
Age (years)	22.7 (2.6)	23.7 (3.2)	23.2 (2.9)
Height (m)	1.68 (0.1)	1.75 (0.1)	1.71 (0.1)
Weight (kg)	62.2 (9.8)	78.6 (10.7)	70.4 (12.9)
BMI (kg/m^2)	22.0 (2.3)	25.7 (2.8)	23.8 (3.1)
BIA (%)	19.6 (3.2)	16.6 (5.6)	16.6 (5.6)
RHR (b/min)	68.3 (8.2)	67.3 (7.2)	67.8 (7.6)
Shoe Length (cm)	26.6 (1.8)	29.4 (1.8)	27.9 (2.2)
Shoulder Height (cm)	139.6 (6.5)	143.9 (7.4)	141.7 (7.1)
Shoulder Width (cm)	30.2 (4.5)	33.6 (2.7)	31.9 (3.9)
Arm Span (cm)	163.6 (8.8)	163.6 (8.8)	167.6 (9.9)
Hip Height (cm)	90.7 (5.4)	91.7 (4.6)	91.2 (4.9)
Hip Width (cm)	25.8 (2.5)	26.5 (2.7)	26.2 (2.7)
Knee Height (cm)	48.7 (2.4)	51.1 (3.5)	49.9 (3.1)
Ankle Height (cm)	9.2 (1.1)	9.9 (1.3)	9.6 (1.3)
Sole Height (cm)	3.2 (0.8)	2.8 (1.1)	3.0 (0.9)

3.3 EQUIPMENT AND MEASURES

Instruments used in this research to quantify anthropometric measurements, vertical ground reaction force, centre of pressure, muscle activation, kinematics and psychophysical data are detailed in the sections below.

3.3.1 SEATED TASK

Individuals were required to sit for two 1-hour exposures in both a static and dynamic chair (Liberty Task™ , Humanscale®, CO, USA), in a varying order per participant (balanced design ½ of each male and female participant groups sat in the static first; ½ sat in the dynamic first). During the seated exposures, participants cycled through three customized tasks set at 5 minutes per task and thus 15 minutes per task set. Four cycles of all three tasks (4 x 15 mins) were completed in random order per 1-hour prolonged sitting exposure. These tasks involved a typical office computer workstation, that included a desktop monitor, standard desk, office chair (static or dynamic), keyboard, mouse, and tray to mimic a typical office environment.

Transient pain is the term used to describe the development of a clinically meaningful level of pain in asymptomatic participants in response to a given exposure (Gallagher *et al.*, 2011; Nelson-Wong & Callaghan, 2014). These participants will not experience a low back injury with this bout of transient pain but have been shown to be at a greater risk of developing future low back pain (Nelson-Wong & Callaghan, 2014). A 100mm VAS (with end points labeled “no pain” and “worst pain imaginable”) was used to quantify participants’ self-reported discomfort in their low back during this study. An initial baseline VAS was taken and removed from each subsequent VAS recorded; time points PRE, MID, and POST the sitting exposure.

Furthermore, time point MID normalized VAS score was calculated by subtracting VAS score PRE by VAS score MID. Likewise, time point POST normalized VAS score was calculated by subtracting VAS score MID by VAS score POST. If a participant VAS score increased 10mm or more relative to baseline they were classified as pain developers (PDs), whereas they were classified non-pain developers (NPDs) if the changes were less than 10mm.

3.3.2 MUSCLE ACTIVATION

Muscle activation was collected from eight trunk muscles bilaterally using surface EMG, and with disposable pre-gelled (Ag-AgCl surface) electrode pairs (Ambu® Blue Sensor N, Ambu A/S Denmark) placed over the target muscle bellies at a 2cm centre-to-centre inter-electrode distance. The electrodes were placed parallel to muscle fiber orientation over the bulk of the muscle (see Table 2 for electrode placements). The muscles collected bilaterally included: rectus abdominis (RA), external oblique (EO), internal oblique (IO), upper-thoracic erector spinae (UTES), lower-thoracic erector spinae (LTES), lumber erector spinae (LES), latissimus dorsi (LT), and gluteus medius (GM). Electrode placement are shown in Table 2. The raw EMG data were sampled at 2400Hz (frequency response 10-1000Hz, common mode rejection 115 dB at 60Hz, input impedance 10 GΩ) using two AMT-8 EMG Measurement Systems (Bortec Biomedical Ltd., AB, Canada) with two Optotrak Data Acquisition Units (Northern Digital Inc., ON, Canada). Maximum voluntary contraction (MVC) trials were collected to permit normalization of the EMG data. Participants completed two repeats of each MVC, with a five-minute rest in between repeats. To elicit the MVC for the trunk flexors: RA, EO, and IO, participants were seated on an athletic therapy table with both knees flexed at 90˚ and performed

a modified sit-up against manual resistance provided by the investigator. The participants were instructed to flex the trunk forward, lateral bend the trunk to the right and left, and axial twist the trunk to the right and left (McGill, 1992). For the trunk extensors: UTES, LTES and LES, participants laid prone on the athletic therapy table with their trunk cantilevered over the edge at the anterior-superior iliac spine (ASIS) level of the pelvis. Participants raised their trunks parallel to the ground and exerted maximally against the resistance applied manually by the researchers, which was applied approximately at the level of the scapulae of the participant (McGill, 1992). For LT MVC trials, individuals were instructed to sit on the athletic therapy table with both arms abducted to 90°, externally rotated, and elbows flexed to 90° (upper arm parallel to the floor, forearm perpendicular to the floor). Next, manual resistance was applied upward by the researcher to each of the participants' elbows while the participant maximally resisted by contracting their LT bilaterally (Dark *et al.*, 2007). Lastly, for the GM muscles participants performed a side lying leg raise for each leg, one at a time, against manually applied resistance (Nelson-Wong, 2009).

Table 2. Electrode placement description for muscles collected. α McGill (1991); ɳ Mirka & Marras (1993); β Zipp (1982)

Location	*Placement Description (Over muscle belly)*
Rectus Abdominis (RA)	~ 2cm above umbilicus, 3 cm lateral to the midline of the abdomen [α]
External Obliques (EO)	~ 15cm lateral to umbilicus at an angle of 45° [ɳ]
Internal Obliques (IO)	~ Superior to inguinal ligament [ɳ]
Latissimus Dorsi (LD)	~ Most lateral portion of muscle at the T9 level [α]
Upper- Thoracic Erector Spinae (TES)	~ 2.5cm lateral to T4 spinous process or over the largest section of the erector spinae muscle at T4 [α, β]
Lower-Thoracic Erector Spinae (LTES)	~ 4 cm lateral to T9 spinous process or over the largest section of the erector spinae muscle at T9 [α, β]
Lumbar Erector Spinae (LES)	~ 4cm lateral from the midline at L3 [α]
Gluteus Medius (GM)	~ 2.5 cm distal to the midpoint of the iliac crest [β]

3.3.3 KINEMATICS

Participant whole-body kinematics were collected using the Xsens Awinda wireless motion tracker system (Xsens TM Technologies B.V. CA, USA), with a 60 Hz sampling rate. This motion capture system is comprised 17 wireless inertial measurement units (IMU) that are affixed to the participants: ten on the upper-body and seven lower body. The placements were as follows: the head (headband), sternum (upper-body suit), right and left shoulders (upper-body suit), right and left upper arm, right and left forearm, right and left hand (hand gloves), pelvis, right and left upper leg, right and left lower leg, and right and left foot (Figure 1). All 17 trackers are required to operate the Xsens Awinda system.

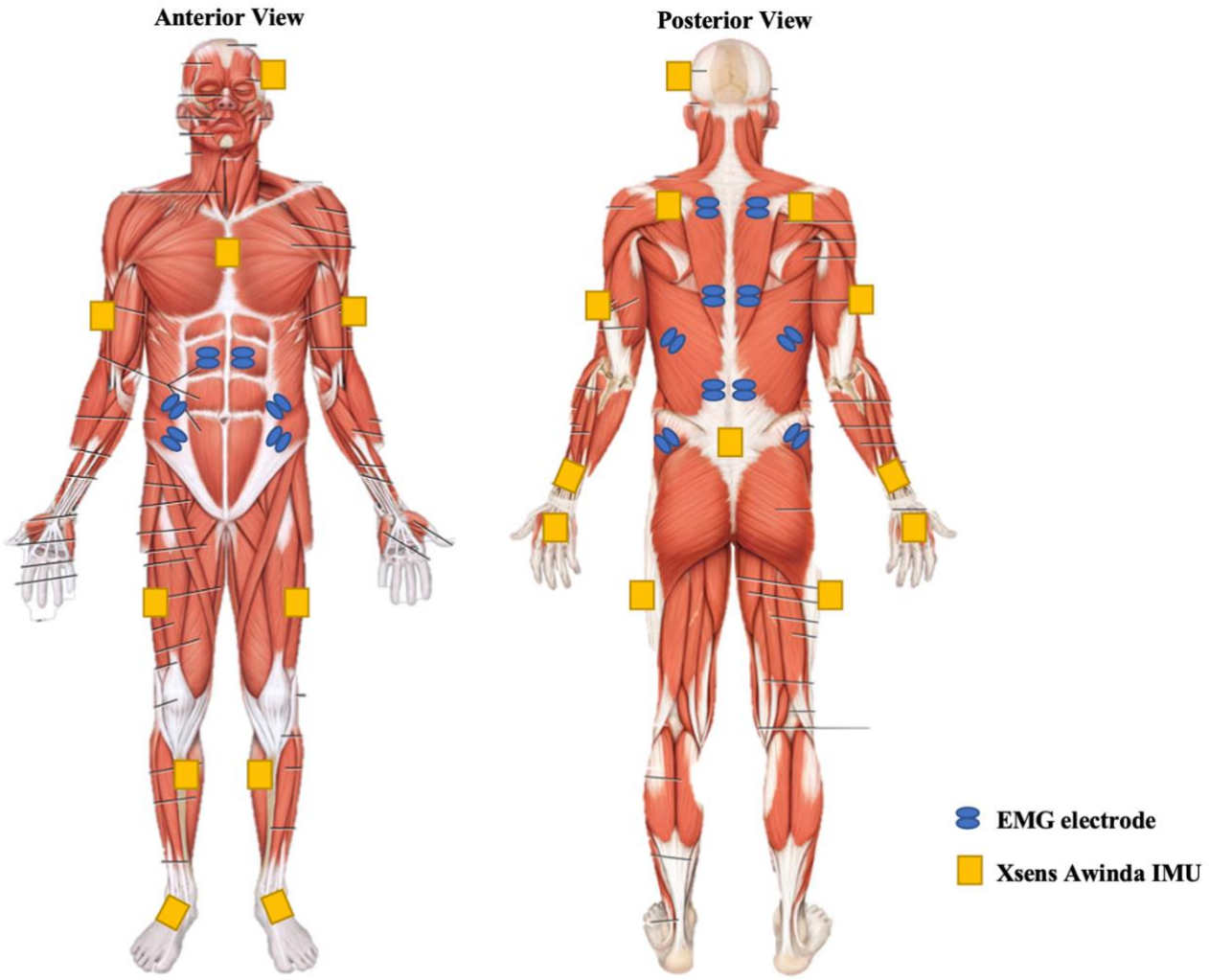

Figure 1. Diagram of Xsens Awinda IMU placement and electrode placement. The following image is modified from In*troduction to the human body (10th edition)* by Gerard J. Tortora and Bryan Derrickson, Publisher: Wiley.

3.3.4 PERTURBATION TASK

Prior to, between, and following the prolonged seated exposure, participants were required to stand on a force platform (Advanced Mechanical Technologies Inc. or AMTI, Model OR6-6-2000, Newton, MA, USA) to measure COP_{AP} and VGRF. The force platform data were digitally sampled at 1024 Hz. Participants stood on the force platforms prior to the prolonged sitting, where foot placement was marked for repeatability in subsequent trials. Participant's selected

their preferred foot placement within the dimensions of the force platform. Perturbations were executed at three time points; PRE (0 hours prolonged sitting), MID (1 hours prolonged sitting), POST (2 hours prolonged sitting). At each of the three time points, each individual was subjected to two unexpected suddenly applied loads within 30 seconds (6 total for the experiment). For each perturbation, the participants were unable to see the investigator apply the sudden load and were unaware of the time at which the load would be applied (Gregory *et al.,* 2008). Each of the two loads were randomly dropped within a 15s window (i.e. two unexpected suddenly applied loads within 30 seconds). The perturbations were manually applied by the examiner by dropping a mass of 6.78 kg from a height of approximately 2 cm. (Gregory *et al.,* 2008). To ensure repeatability, participants were required to hold the fixed sized container with an elbow angle of 90º and with elbows tucked to their trunk. Visual inspection of participant elbow positioning was confirmed by investigators prior to each unexpected suddenly applied load.

3.4 DATA ANALYSIS

3.4.1 MUSCLE ACTIVATION

Prior to analyses, electrocardiogram (ECG) contamination was removed by applying a high-pass filter using a dual-pass Butterworth filter with a cut-off frequency of 30Hz (Drake & Callaghan, 2006). EMG signals were normalized to the highest 20-millisecond moving average value between both MVC trials. Next, EMG signals were full-wave rectified and low pass filtered using a 2nd-order Butterworth filter, with a cut-off of 2.5 Hz (Gregory *et al.*, 2008). $Base_{EMG}$ (% MVC) was determined by averaging muscle activation 50ms prior to sudden load drop. To determine EMG response (%MVC), $Base_{EMG}$ was subtracted from the maximum value 375ms following the sudden load drop. The length of the windows used in this study were based on the

findings from Hodges and Bui (1996). In addition, this study assessed several methods of determining muscle on-set times and found several to be accurate, from which times were selected for this research. Moreover, for muscle latency time analysis, rectified EMG signals were low pass 4th-order Butterworth filtered with a 50 Hz cut-off. EMG signals were determined to be active if they exceeded $Base_{EMG}$ plus three standard deviations of the $Base_{EMG}$. Signals were considered a reflexive response if they occurred between 20ms to 160ms, and values not meeting this condition were considered a controlled response (Cholewicki *et al.*, 2005). Also, to determine the occurrence of muscle onset the activity of each muscle was calculated for each perturbation exposure (Gregory *et al.*, 2008).

Probability of activation (ProbON) was calculated for each muscle (bilaterally) collected. ProbON was calculated by determining if the muscle on-set was considered active for each perturbation trial, divided by the total number of perturbation trials (six total) (Gregory *et al.*, 2008). For example, if the RA was considered to be active three out of six perturbation trials, the ProbOn would be 0.5 for the RA. The number of trunk flexors activated (FlexON) was composed of left and right RA, EO, IO (six total). FlexON was determined by calculating the percentage of trunk flexors activated per unexpected suddenly applied load (Gregory *et al.*, 2008). For example, if three out of the six trunk flexors were met the conditions of muscle onset activity, the FlexON would be 0.5. Similarly, ExtON was determined by calculating the percentage of trunk extensors: left and right UTES, LTES and LES.

3.4.2 KINEMATICS

Participant kinematic data were processed using a programmed code in MATLAB (v.R2018b.9.5.0.9, Mathworks TM Inc., MA, USA) that calculated relative and absolute angles

for thoracic (data defined from points T1 to T12); lumbar (data defined from points L1 to L5); hip (right and left); elbows (right and left); and knees (right and left) in the sagittal plane. Peak angular displacement was determined for the upper-thoracic, lower-thoracic, lumbar, T8/Head, Pelvis/T8. It is important to note that the T8/Head joint corresponds to the rotation of the upper thoracic spine in the flexion/extension axis with respect to T8. Moreover, Pelvis/T8 corresponds to the rotation of the lumber spine in the flexion/extension axis with respect to T8 and pelvis. The hip, knee, and elbow relative angles were also calculated to determine participants maintained consistent postures for each perturbation. In addition, baseline angles were determined by calculating the trunk angle for each segment 50ms prior to sudden load drop. Peak trunk angle response was determined by subtracting baseline angle from the maximum trunk angle value within 400ms of the applied perturbation. The kinematic data were low-pass filtered using a dual-pass, fourth-order Butterworth filter with a cut-off frequency of 2.5Hz (Schinkel-Ivy *et al.,* 2014; Winter, 2005).

3.4.3 PERTURBATION TASK

Force and moment signals obtained from the force platform were dual-pass filtered with a 2nd-order Butterworth filter with an effective cut-off of 10 Hz (Carpenter et al., 2001). COP_{AP} displacement (with respect to the centre of the force platform) in the anterior–posterior direction was determined using "Mx divided by Fz", where Mx was the moment about the medial–lateral axis and Fz is the vertical ground reaction force. A decrease in COP_{AP} displacement would be considered posterior movement towards the global origin of the force platform. The VGRF was used to approximate the onset of loading. The main measures for the force platform data were divided into baseline and peak response. The baseline was defined as the average between 50ms

prior to load drop and the peak COP_{AP} displacement was calculated as the difference between the COP_{AP} location averaged over 50 ms prior to the perturbation and the maximum COP_{AP} location (within 450 ms post-perturbation).

3.4.4 STATISTICAL ANALYSIS

Three-way mixed analysis of variance (ANOVA), with time point (PRE, MID and POST) to the prolonged sitting exposure, transient pain development or LBD (yes/no), and sex (male/female) were used to test the response measures. Appropriate testing was conducted to ensure the assumptions of the ANOVA technique were not violated in the statistical analysis of these data. To complete an ANOVA, assumptions are required to be met. A main assumption is that the outcome measures approximately conform to normality. A log transform was used to adjust data to normality. Statistically significant effects were found when data were conformed to normality. Furthermore, a number of variables between displayed p-values of less than 0.05, and thus will be presented and discussed. If there was a significant finding, a Scheffe's post-hoc was used to identify which pairs of means of measures were significant.

4.0 RESULTS

Only five (one female, four males) of the 30 participants did not develop substantial LBD during the prolonged sitting exposure. For statistical purposes, these individuals were considered NPDs and the individuals who developed LBD were considered PDs. The normalized VAS scores (relative to baseline VAS score at time point PRE) of participants at the end of each one-hour prolonged sitting exposure are shown in Figure 2. These findings indicate that 25 out of 30 participants developed LBD as a result of one-hour of prolonged sitting.

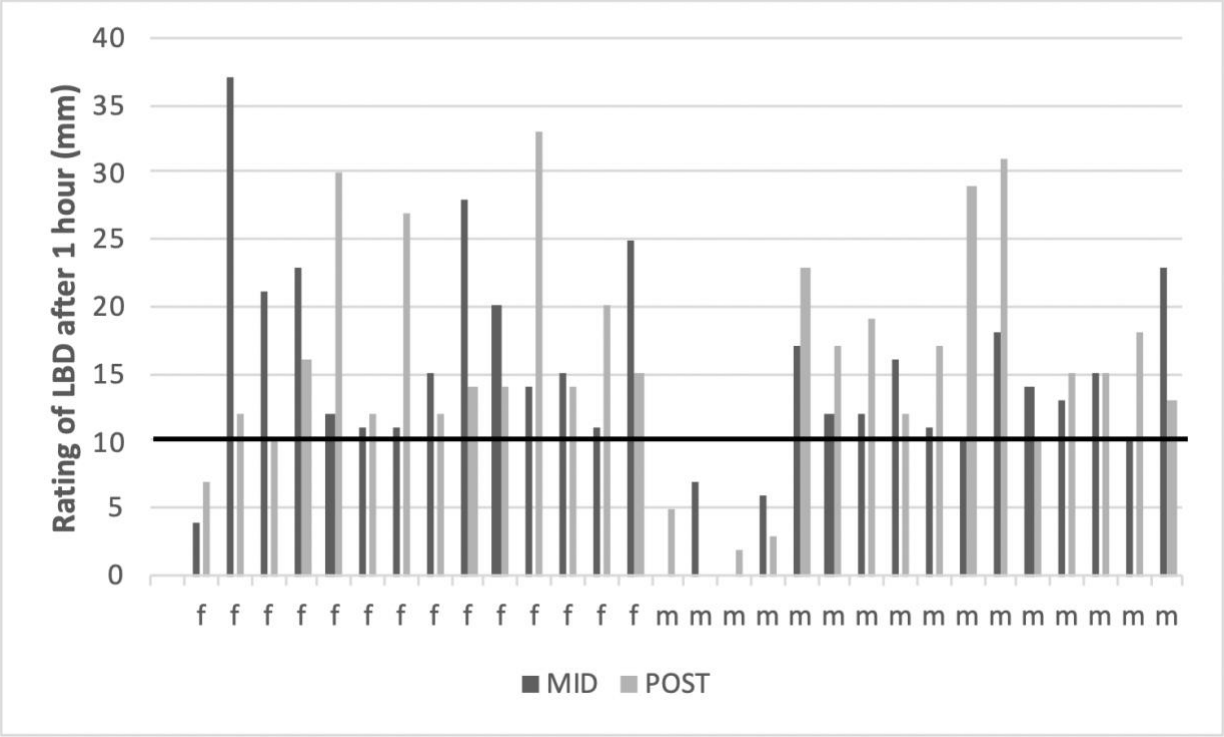

Figure 2. Relative ratings of perceived low back discomfort using 100mm VAS after each 1hr of sitting. When participants ratings were greater than 10mm they were classified as a PD (horizontal line), and when less as NPD. Females and males are labeled 'f' and 'm', respectively.

4.1 THE EFFECT OF TIME

The following findings are significant interactions with an effect of time. Baseline COP_{AP}, COP_{AP} displacement, EMG response, muscle latency time, probability of activation and T8/Head angle displacement were all effected due to the prolonged sitting exposure. Baseline COP_{AP} significantly changed ($p = 0.03$) throughout the prolonged sitting exposure. The Baseline COP_{AP} decreased from 23.72 mm ± 33.78 to 11.50 mm ± 21.84 as time elapsed, becoming more posterior with each hour of prolonged sitting exposure. COP_{AP} displacement significantly changed ($p = 0.048$) following the 1-hour sitting period from PRE to POST and MID to POST prolonged standing respectively. COP_{AP} decreased from 79.78 mm ± 21.69 to 67.24 mm ± 26.24

from PRE to POST prolonged sitting exposure (Figure 3). These results indicate prior to perturbations, participants stood more posteriorly on the force platform as prolonged sitting elapsed and in response to unexpected perturbations, participants remained more upright.

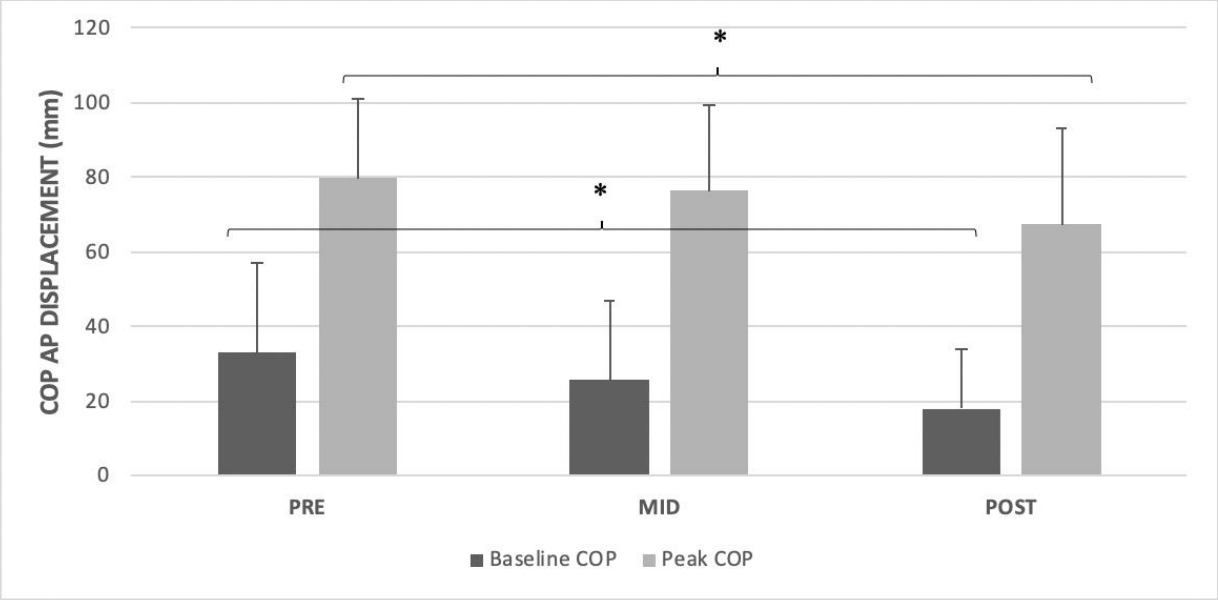

Figure 3. The average Baseline COP_{AP} and average COP_{AP} displacement in response to perturbations before, between, and following the prolonged sitting exposure. Baseline COP_{AP} and average COP_{AP} displacement were shown to decrease at time point MID and subsequently time point POST. Asterisk indicates $p < 0.05$.

For the left EO there was a significant effect of prolonged sitting on average EMG response ($p = 0.0372$). Average EMG response of the right UTES ($p = 0.041$) was differed PRE, MID and POST prolonged sitting exposure. The right UTES average EMG response increased from 14.48 %MVC ± 27.78 at time point PRE to 40.98 %MVC ± 16.89 at time point POST. Therefore, participants increased the muscle activation of the right UTES in response to unexpected perturbations as the prolonged sitting protocol elapsed.

There was a significant effect of prolonged sitting on muscle latency time for the left EO ($p = 0.084$). Average muscle latency time increased from 27.64ms ± 19.91 at time point PRE 39.5ms ± 29.55 at time point POST. These results indicate, for the left EO, participants displayed

increasingly delayed activation surpassing the calculated threshold in response to unexpected perturbation throughout the prolonged sitting exposure.

Regarding ProbON, there was a significant effect of time on the right EO, right UTES, left UTES, and left LES ($p = 0.00044$, $p = 0.023$, $p = 0.023$, and $p = 0.002$ respectively). As the prolonged sitting exposure elapsed, each muscle displayed increased probability of activation from: 0.62 ± 0.19 to 0.92 ± 0.18 (right EO), 0.56 ± 0.3 to 0.82 ± 0.24 (right UTES), 0.54 ± 0.43 to 0.88 ± 0.25 (left UTES), and 0.56 ± 0.39 to 0.86 ± 0.32 (left LES) respectively (Figure 4). Thus, as the participants completed the prolonged sitting exposure, the upper back was more active in response to the subsequent unexpected perturbations. Muscle latencies and ProbON for each muscle averaged across all participants are shown in Table 3.

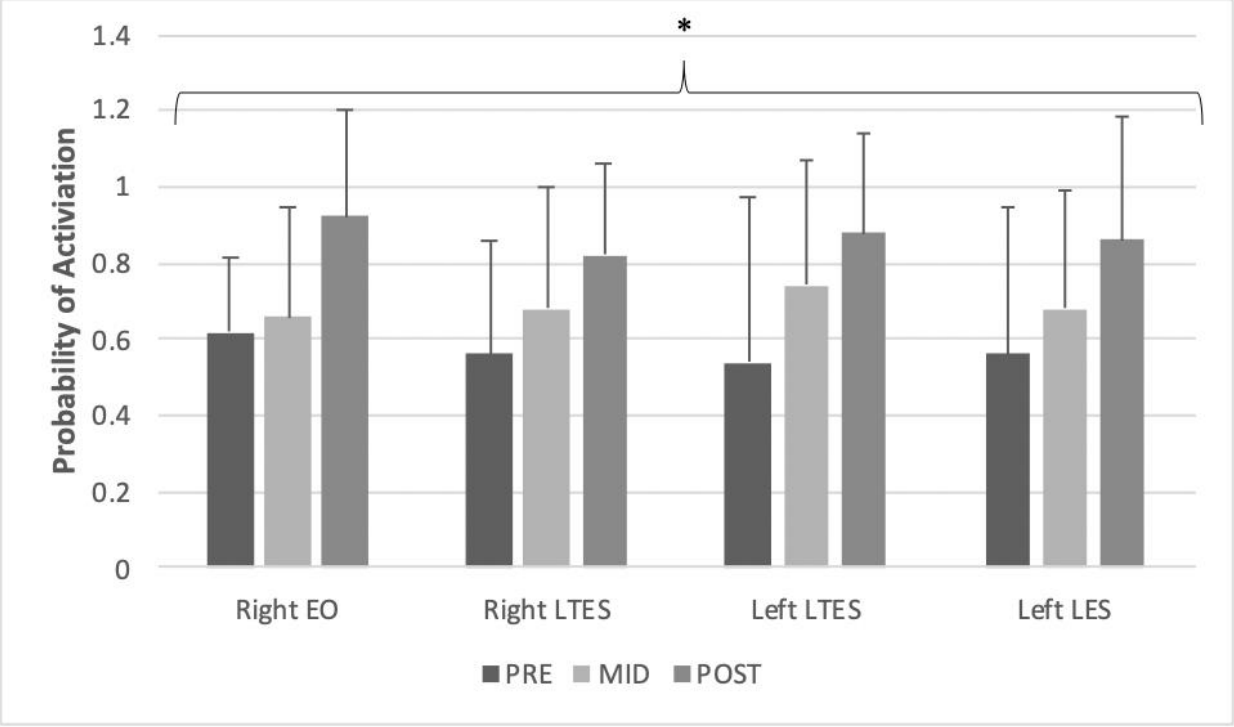

Figure 4. The average probability of activation for right EO, right LTES, left LTES and left LES throughout the prolonged sitting exposure increased in response to unexpected perturbations at each time point. Asterisk indicates $p < 0.05$.

Table 3. Average (standard deviation) probability of activation (ProbON) (out of six) and muscle latency (ms) across all participants

Muscle	*Probability of Activation*		*Muscle Latency (ms)*	
Right				
RA	0.7	(0.33)	88	(26)
EO	0.78	(0.29)	91	(25)
IO	0.63	(0.35)	87	(28)
UTES	0.7	(0.34)	85	(23)
LTES	0.67	(0.31)	86	(24)
LES	0.72	(0.32)	92	(24)
LT	0.74	(0.31)	88	(19)
GM	0.66	(0.31)	80	(20)
Left				
RA	0.75	(0.32)	96	(39)
EO	0.73	(0.34)	88	(23)
IO	0.78	(0.32)	89	(23)
UTES	0.75	(0.31)	84	(22)
LTES	0.72	(0.37)	87	(23)
LES	0.79	(0.33)	90	(25)
LT	0.7	(0.35)	86	(24)
GM	0.67	(0.36)	84	(21)

Lastly, there was a significant effect of time on average peak trunk angle from T8/HEAD ($p = 0.0151$). The average peak trunk angles of T8/HEAD decreased from $26.8^{o} \pm 12.1$ to $19.32^{o} \pm 10.56$ subsequently from time point PRE to time point POST (Figure 5). These results suggest that participants exhibited less angular displacement in the flexion/extension axis in response to unexpected perturbations with increased time spent sitting. Again, after two hours of sitting, participants displayed and increased EMG response, muscle latency time, probability of activation, and decreased T8/Head displacement, baseline COP_{AP}, and COP_{AP} displacement.

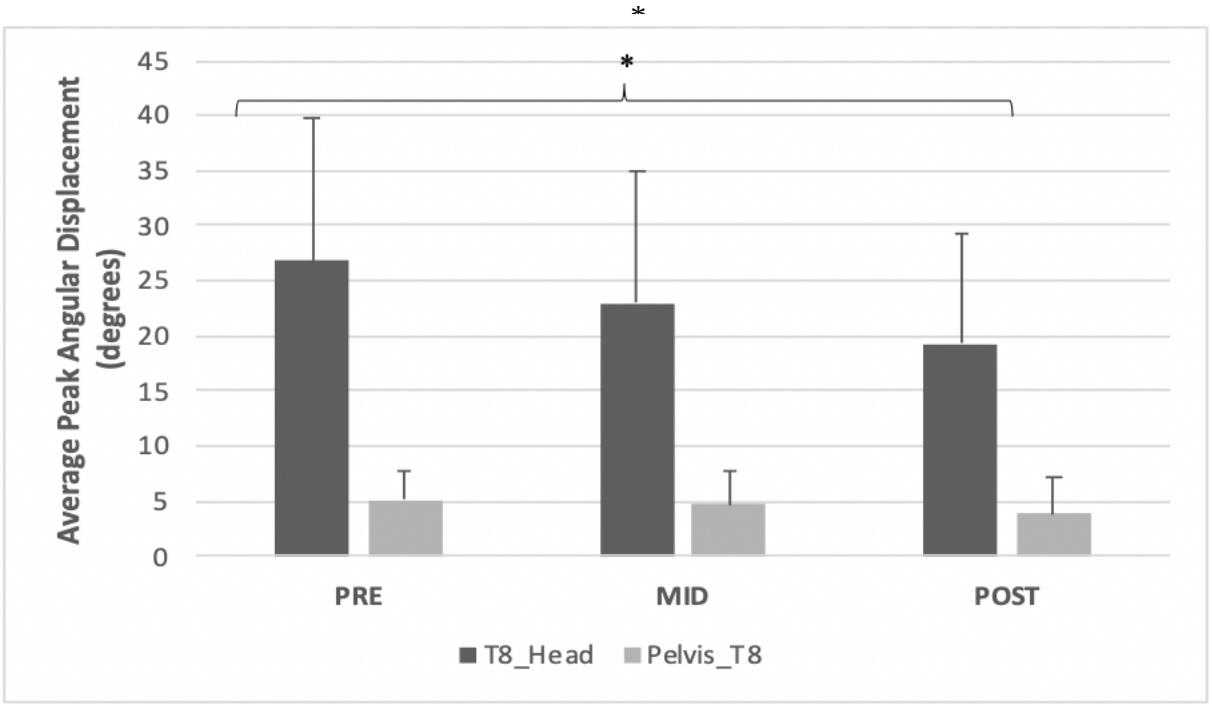

Figure 5. The average peak angular displacement of T8/Head decreased at time point MID and further decreased at time point POST whereas the Pelvis/T8 did not significantly decrease over the course of the prolonged sitting exposure. Asterisk indicates $p < 0.05$.

4.2 EFFECT OF LBD

The next presented results are measures involving a significant effect with the development LBD in participants. There was a significant effect of LBD on average EMG response for the left EO at time point MID ($p = 0.034$), where PDs displayed a greater average EMG response (44.63 %MVC ± 32.81) compared to NPDs (16.32 %MVC ± 19.29). For muscle latency time, there was a significant effect of LBD on the right LES ($p = 0.0304$). PDs displayed longer muscle latency times (40.88ms ± 33.72) of the right LES as compared to the NPDs (21.83ms ± 14.28). Furthermore, for the right UTES there was a significant effect of LBD ($p = 0.034$). NPDs displayed quicker muscle latency times (28.15ms ± 18.97) versus the PDs who displayed slow muscle latency times (41.26ms ± 29.17). There was a main effect of LBD on muscle latency time to right EO for females ($p = 0.018$). Moreover, Female PDs (33.47ms ± 13.30) displayed a

longer muscle latency time for the right EO versus female NPDs (24.97ms ± 16.71). The effect of LBD on EMG response and muscle latency time indicate that as a result of the development of LBD, PDs exhibited increase muscle activation of the left EO and right UTES. Therefore, the development of LBD suggests a delayed activation of the right EO, right LES, and right UTES in response to unexpected perturbations.

4.3 THE INTERACTION OF SEX AND TIME

The next set of results describe the interaction of sex and time that occurs on measures in this research. At time point PRE, female participants (13.53 %MVC ± 9.07) had significantly larger $Base_{EMG}$ for right LTES ($p = 0.017$) than males (2.64 %MVC ± 1.99), whereas male participants (5.62 %MVC ± 4.95) exhibited a greater magnitude of $Base_{EMG}$ for left RA ($p = 0.032$) than females (3.28 %MVC ± 2.77) at the same time point. Prior to unexpected loading and prolonged sitting females had increase baseline activation for the right LTES where at the same time point males exhibited increase left RA baseline activation.

These results show the effect of the interaction of sex and time on muscle latency time and probability of activation. At time point POST, there were significant effects of sex on the average muscle latency time of the right EO ($p = 0.041$). Male PDs (39.15ms ± 32.18) exhibited a greater length of average muscle latency time for the right EO when compared to female PDs (15.59ms ± 8.00). There was also a significant effect of sex on ProbON for the right LES ($p = 0.008$), where females (0.72 ± 0.33) activated the right LES are lesser number of trials as compared to males (0.85 ± 0.28). These findings suggest, after the two-hour prolonged sitting exposure male PDs took longer than females to activate the right EO in response to unexpected

perturbations. Also, these findings indicate that males utilized the activation of the right LES in response to unexpected perturbations throughout the prolonged sitting.

Lastly, there were sex differences observed in the kinematic data. There was a significant two-way interaction of sex and time on average peak trunk angle response of Pelvis/T8 ($p = 0.048$). Males ($6.26^{o} \pm 2.71$ to $10.23^{o} \pm 2.01$) had a greater magnitude of average peak trunk angle change compared to females ($3.98^{o} \pm 2.07$ to $5.78^{o} \pm 4.71$) between time point PRE to time point POST (but not PRE to MID). Therefore, in response unexpected perturbations after two-hours of prolonged sitting, males had increased lumbar spine displacement versus females.

4.4 INTERACTION OF LBD AND TIME

This next section of results details the interaction of LBD and time on $Base_{EMG}$ and ExtON. $Base_{EMG}$ had a significant interaction between the LBD and time factors for the right LTES ($p = 0.035$). $Base_{EMG}$ increased in magnitude at each time point for PDs (2.47 %MVC ± 2.27, 2.91 %MVC ± 2.52, 3.41 %MVC ± 2.014 respectively) while $Base_{EMG}$ decreased in magnitude at each time point for NPDs (1.45 %MVC ± 0.61, 1.32 %MVC ± 0.67, 1.19 %MVC ± 1.00). There was a significant main effect of LBD on left RA ($p < 0.005$) $Base_{EMG}$ for females at time point MID, where female NPDs (3.17 %MVC) exhibited a lesser magnitude in left RA $Base_{EMG}$ versus female PDs (4.47 %MVC ± 1.61). These results indicate that the participants that developed LBD throughout the prolonged sitting developed increased baseline activation of the right LTES and specifically at time point MID, sustained higher baseline activation of the left RA.

For ExtON, there was a significant two-way interaction of LBD and time ($p < 0.001$). Specifically, there was an effect of LBD on male and female participants at time point MID,

female and male PDs (4.50. ± 1.50 and 4.78 ± 0.51 respectively) activated greater number of trunk extensor versus female and male NPDs (1.50, 3.63 ± 0.6) as illustrated in Figure 6. However, there were no significant differences between ExtON for the other time points. Therefore, PDs were required to increase the recruitment of trunk extensors in order to resist the force of the unexpected perturbations after 1hr of prolonged sitting relative to NPDs.

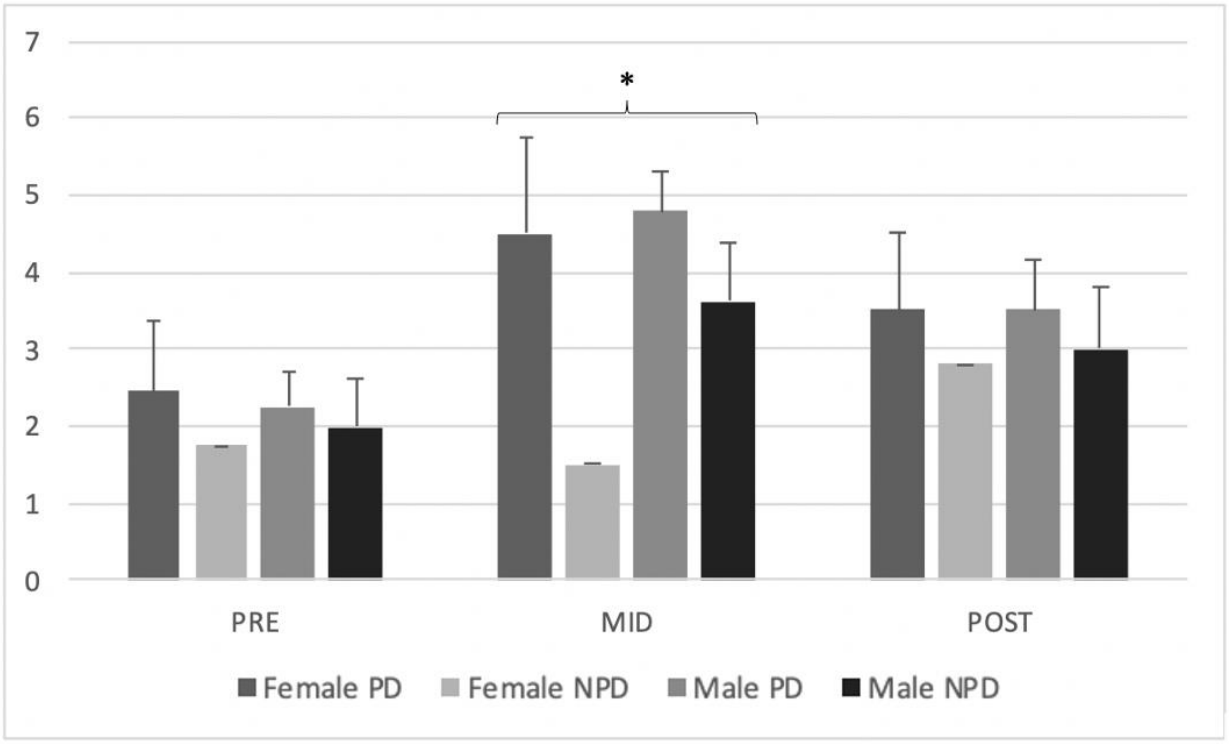

Figure 6. Graphical representation of the average number of responsive trunk extensors for female and male participants between PD's and NPD's. At time point MID, female PD's and male PD's had a significant greater number of responsive trunk extensors compared to female and male NPD's. Asterisk indicates $p < 0.05$.

4.5 INTERACTION OF SEX, LBD AND TIME

The following section of result address the effect of three-way interaction of sex, LBD and time on the outcome measures in this research. There were no statistically significant three-way interactions for Baseline and peak COP_{AP}, $Base_{EMG}$, baseline angular displacement, or muscle latency. A significant three-way interaction between sex, LBD, and time was observed for the left RA ($p < 0.001$), left IO ($p < 0.001$), left LTES ($p < 0.001$), and right IO ($p < 0.001$) EMG response. For the self-reported pain ratings, there was a significant effect of sex for PDs at time point POST, where the female PDs (111.01 %MVC ± 12.00) developed a greater magnitude of left RA activation at time point POST compared to female NPDs (44.91 %MVC). These findings indicate that at time point POST, female PDs had greater left RA activation that female NPDs and male PDs (Figure 7).

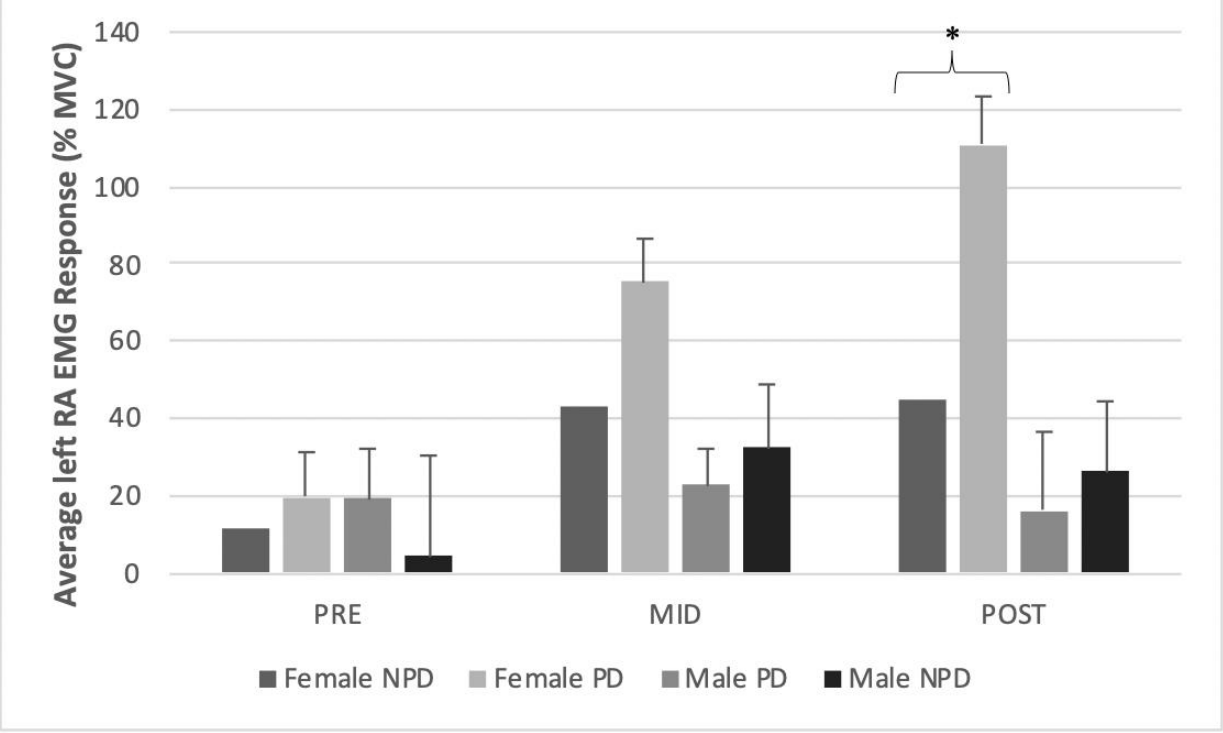

Figure 7. The average left RA EMG response between male PDs and NPDs and female PDs and NPDs throughout the prolonged sitting exposure. At time point POST, the average left RA EMG response is significantly different between PDs and NPDs and sex in response to unexpected perturbations. Asterisk indicates $p < 0.05$.

The left IO exhibited significant two-way interaction of sex and LBD at time point MID ($p < 0.001$). The female NPDs (16.23 %MVC) developed a greater magnitude of activation compared to male NPDs (11.1 %MVC ± 12.6), and female PDs (106.44 %MVC ± 23.33) demonstrated a greater magnitude versus female NPDs (16.23 %MVC). There was a significant two-way interaction of sex and LBD at time point PRE for the left LTES ($p < 0.001$). Similarly, to left IO and left RA, female NPDs (5.35 %MVC) exhibited a lesser magnitude of activation for the left LTES compared to female PDs (8.81 %MVC ± 7.33). For PDs, the mean left LTES activation was significantly different between female (28.40 %MVC ± 15.08) and male participants (14.45 %MVC ± 13.27).

At time point MID there was a significant two-way interaction of LBD and sex for the right IO ($p = 0.014$), where female PDs (33.71 %MVC ± 23.25) displayed greater muscle activation versus female NPDs (26.2 %MVC), and female NPDs (26.2 %MVC) developed greater magnitude of activation compared to male NPDs (14.15 %MVC ± 5.06). These results show that in response to unexpected perturbations, PDs and female were required increase activation of the aforementioned muscles after one hour and two hours of sitting.

There were significant 3-way interactions for muscle latency time, average peak trunk angle, and FlexON in this study. For muscle latency time, there was a significant three-way interaction for the right LES ($p < 0.001$). There was an effect of sex and time point MID as well as an effect of LBD on females at time point MID. Female PDs (39.42ms ± 33.16) displayed longer muscle latency times versus female NPDs (20.4ms). Similarly, female PDs (39.42ms ± 33.16) exhibited shorter muscle latency times as compared to male PDs (49.07ms ± 38.44) Additionally, there was a significant three-way interaction for the average peak trunk angle

response of T8/C7 segment ($p = 0.009$). At the time point MID, female PDs ($7.18^{o} \pm 5.65$) exhibited a decrease magnitude of average peak trunk angle displacement of T8/C7 segment compared to female NPDs (24.9^{o}), male PDs ($14.47^{o} \pm 14.43$). For FlexON, there was also a significant three-way interaction ($p = 0.001$), where female PDs (4.55 ± 0.61, 5.00 ± 1) activated a greater number of trunk flexors at time points MID and POST compared to female NPDs (3, 1.5) in response to unexpected perturbations (Figure 8). These data implied that after 1-hour and 2-hours prolonged sitting, female PDs performed less trunk flexion at T8/C7, did not experience delayed muscle activation, and activated greater number of trunk flexors in response to unexpected perturbations relative to female NPDs and males.

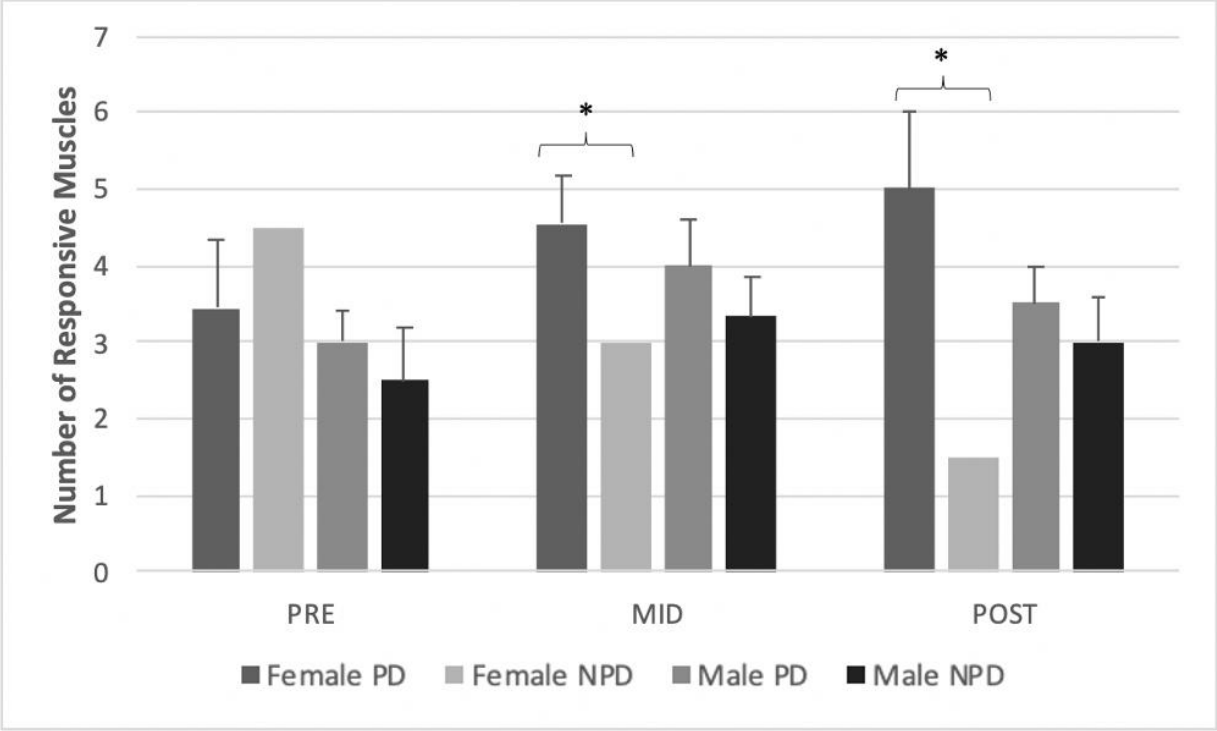

Figure 8. Average number of responsive trunk flexors for female participants between PDs and NPDs in response to perturbation throughout the prolonged sitting exposure. At time point MID and POST, female PDs activated a greater number of trunk flexors compared to female NPDs. Asterisk indicates $p < 0.05$.

5.0 HYPOTHESES REVISISTED

Hypothesis 1: Prolonged sitting will cause an increase amount of erector spinae muscle activation and decreased trunk flexion in response to unexpected perturbations.

Decision: Rejected the null hypothesis and accepted alternate hypothesis.

Participants experienced increased amount of right UTES EMG response ($p = 0.041$) throughout the prolonged sitting protocol. Furthermore, for female participants and PDs the LTES ($p = 0.715e\text{-}11$) displayed increased activation in response to perturbations and the prolonged sitting protocol. The average T8/Head displacement ($p = 0.015$) significantly decreased throughout the prolonged sitting protocol in response to unexpected perturbations. It is likely that individuals experienced increased erector spinae EMG response due to development of LBD, which may have subsequently led to increase T8/Head displacement in response to unexpected perturbations (Lariviere, *et al.*, 2010).

Hypothesis 2: Pain developers will experience increase amount of muscle activation from the torso muscles in response to unexpected perturbations.

Decision: Rejected the null hypothesis and accepted alternate hypothesis.

Pain developers experience greater EMG response versus non-pain developers in response to perturbations and prolonged sitting. Specifically, for the left RA, left IO, right IO, left EO, right UTES, and left LTES. Moreover, the increased activation seen in PDs may have occurred due to the impaired spinal stability associated with the development of LBD (Abboud *et al.*, 2017). Therefore, it appears that the trunk musculature tried to compensate by increasing activation of the trunk musculature.

Hypothesis 3: Females will experience increase amount of muscle activation of the trunk musculature in response to unexpected perturbations.

Decision: Rejected the null hypothesis and accepted alternate hypothesis.

Interestingly, both female PDs and NPDs displayed greater EMG response in the left RA, left IO, left LTES, and right IO compared to male counterparts in response to unexpected perturbations and prolonged sitting. The increased EMG response may the result of the different trunk postures while sitting between sex (Dunk and Callaghan, 2005).

Hypothesis 4: Muscle onset times will increase post prolonged sitting for the torso musculature and COP displacement will decrease.

Decision: Rejected the null hypothesis and accepted alternate hypothesis.

Upon development of LBD, pain developers displayed increased muscle latency times in the right EO, left EO, right LTES, right LES. Likewise, the COP displacement decreased throughout the prolonged sitting exposure and in response to unexpected perturbations. The decrease in COP could have occurred by potential development of erector spinae fatigue (Grondin and Potvin, 2009). In patient with acute LBP, Reevers *et al.,* 2005 found that individuals displayed greater muscle latency times versus control participants.

6.0 DISCUSSION

In this study, the majority of participants (83.33%) developed LBD over the two-hour period during the prolonged sitting exposure. As previously mentioned, prolonged sitting exposures result in greater lumbar flexion and low static loading associated with the development of LBD (Callaghan and McGill, 2001). These changes in posture of the lumber spine can lead to the development of creep, fatigue, and pain. Despite there being several pathological mechanisms to develop LBD, prolonged sitting exposure appears to consistently contribute greatly to LBD development in office workers. With LBD development, creep and fatigue responses induced by the ‘action’ of sitting may help explain the effect prolonged sitting exposure observed on the

responsorial actions of the trunk musculature. Prolonged sitting caused baseline and peak COP_{AP} displacement to decrease throughout the entire protocol. Additionally, the prolonged sitting had caused increased EMG response of left EO and of the right UTES in response to unexpected perturbations. Conversely, it has been found that RA, EO and IO reflex amplitude were not influenced by muscle fatigue (Granata *et al.,* 2004), and creep did not significantly decrease the reflex amplitude of the trunk musculature to unexpected suddenly applied loads (Granata *et al.,* 2005). However, the development of LBD has shown to increase erector spinae activity in response to perturbation (Lariviere *et al.,* 2010). Correspondingly, one hour after the prolonged sitting exposure and subsequently the onset of LBD it was found that male and females displayed increased probability of activation of trunk extensors (ExtON) in response to perturbations compared to prior to the exposure. Also, in patients who developed acute LBD, the EO response to perturbations increased in amplitude (Jones *et al.,* 2012). Along with Jones *et al,* (2012) it was found in the current research that LBD had caused an increase in reflex amplitude of the left EO. Finally, as the prolonged sitting exposure elapsed the probability of activation of the right EO, right UTES, left UTES and left LES increased. In the current study, the effect of LBD was not significant but these data suggested that there was a functional increase in activation above some threshold to influence responses, but not enough to detect significant increase in reflex amplitude with the current calculations. It was also hypothesized, that a similar mechanism of increasing activation of trunk musculature to increase stiffness may have been used to protect the lumber spine in response to the neuromechanical detriments of prolonged sitting in response to unexpected perturbations.

Previous research found that PDs are predisposed to develop increase co-contraction of the trunk musculature over time and co-contraction was significantly correlated to LBD

(Schinkel-Ivy *et al.,* 2013). The increased levels of co-contraction act to ameliorate the deficits of the passive and active elements of the trunk in response to prolonged sitting, However, it is likely that the co-contraction impairs spinal stability and contributes to the development of LBD. Based on the current study and previous research, it is theorized that the co-contraction may reinforce the poor neuromuscular responses of the trunk to prolonged sitting predisposing individuals to LBD trying to increase stiffness of the spine trying to alleviate the discomfort. Thus, there was an increased likelihood that the development of creep and fatigue of the trunk may not have been the primary mechanisms responsible for the increased activation of the trunk musculature in response to perturbations, but rather it may be the development of LBD.

Granata *et al.* (2001) found, that following a fatiguing task there was a significant increase of baseline electromyographic activity of erector spinae muscles. Likewise, it has been discovered there was higher RA EMG activity following erector spinae fatigue (Granata *et al.,* 2004). Interestingly, in the current study, the average $Base_{EMG}$ of right LTES and left RA increased in magnitude for the PDs. In research conducted by Jacobs *et al.* (2011), it was believed that the increased baseline muscle activation in PDs was to circumvent any further angular trunk displacement. This would explain the decrease average peak angular displacement of T8/Head that was seen as prolonged sitting exposure elapsed. Therefore, it can be theorized that there may have been a presence of erector spinae fatigue in individuals who developed LBD. Future investigations should include measures of fatigue to determine if fatigue is related to the development of LBD.

In the current study, the left EO muscle latency time increased with the development of LBD subsequently with prolonged sitting time. Muscle latency time of the external oblique has been shown to increase with the development of LBD (Reeves *et al.*, 2005). This research study

along with previous research illustrate that a prolonged sitting exposure increases the likelihood of developing LBD, and those who develop LBD demonstrated injurious muscular and kinematic responses to unexpected suddenly applied loads. Previous research has also shown that prolonged sitting induces low static flexion. A consequence of low static flexion for a prolonged period of time prompts the development of creep in the passive tissue of the lumbar spine (Shin *et al.,* 2009). This creep generation can be developed through fatigue of the lower spinal erectors due to passive stretching of these muscles (Shin *et al.,* 2009). In return, erector spinae muscles are required to generate increased amount of activation in weight holding after static flexion exposures in order to maintain spinal stability and posture to compensate for the muted contribution of the passive tissue that have experienced deformation (Shin *et al.,* 2009). The greater amount of muscle activation of the trunk musculature in response to unexpected sudden loads is detrimental by increasing the magnitude of forces acting on the spine. However, there is a trade-off between stiffness and stability and thus the trunk musculature may be increasing contributions to elicit a safer response to the unexpected sudden loading. It was hypothesized, the development of fatigue throughout the prolonged sitting exposure is possible due to the onset of creep that occurs due to the postures adapted while seated (Abboud *et al.,* 2016). As time elapsed throughout the prolonged sitting exposure in the current study, the left RA and right LTES displayed an increase baseline muscle activation. An increase in baseline muscle activation in trunk musculature in response to unexpected suddenly applied loads is characteristic of fatigue and would merit the responses observed in the current study (Granata *et al.,* 2001).

Several significant interactions were found between sex and the outcome measures. Briefly, females exhibited lower magnitudes of $Base_{EMG}$, shorter muscle latency times, greater EMG response and increased trunk angle flexion in response to perturbations. Perhaps, female

participants exhibited greater activation and subsequently increased trunk flexion in response to the perturbations, due to the magnitude of load relative to the bodyweight and anthropometrics between sex. Females on average were shorter and weighed less than the male participants. Likewise, the magnitude of the load relative to BMI was smaller for females compared to males. However, while some changes observed may seem like neuromechanical detriments altered by prolonged sitting and LBD development, the other measures impacted by these mechanisms did not exhibit the same trends. There are sex-based differences in sitting postures between males and females, where females sit with less lumbar and trunk flexion (Dunk and Callaghan, 2005). Therefore, the decreased spinal flexion during sitting may lead to less creep development and less erector spinae fatigue. Further, if these mechanisms were responsible for the negative display of responsorial actions of the trunk musculature, this would account for the less sensitive neuromechanical responses seen in females on key outcome measures in this study.

There was an external flexor moment to the trunk musculature with suddenly applied load into the hands of the body. As expected, there would be an increase in magnitude of muscle activation of the erector spinae in response to the applied load. The responses indicated increased activation of the erector spinae but also the trunk flexors. In the current study, the trunk flexors became activated in concert with the trunk extensors in response to sudden loading. Once again considering spinal stability, the results would signify that the response to unexpected sudden loading requires an increase in stiffness to maintain spinal stability by trunk muscle co-contraction. However, it is likely co-contraction that perpetuates the cycle that may be contributing to the development of LBD by reinforcing negative neuromuscular control of trunk tissues. Therefore, spinal stability can be compromised by prolonged sitting exposures, which may be further aggravated by unexpected external perturbations leading to potential LBP/LBI.

7.0 LIMITATIONS

As with any study, there are a few limitations to the current work that should be considered when interpreting or applying the findings. First, visual analog scales are subjective, and thus have the possibility of a misleading interpretation of results (e.g. people do not perceive pain the same way). However, based on verbal communication with participants throughout the prolonged sitting exposure, the investigators are confident that individuals who had a final rating of 10mm or less did not experience or develop LBP. Second, EMG data during the unexpected suddenly applied loads were subjected to unavoidable movement artifact. In an effort to minimize any impact of movement artifacts, MVC data and EMG data recorded during the responses to the perturbation were visually inspected and confirmed by the primary investigator. Third, the magnitude of load used was relatively lower for males as compared to females based on bodyweight but was relatively low overall. The use of a constant low load was selected for convenience to conduct the study and was similar to previous research (Gregory *et al.,* 2008). Further, the low load magnitude was selected was to minimize the risk of injury, as it was suspected participants would have at least some trunk muscle fatigue and/or creep in from the prolonged sitting exposure which was expected to compromise coordination and strength (Parnianpour et al., 1988). Lastly, individuals completed two one-hour prolonged sitting exposure blocks separated by a 3-minute walking break versus completing one continuous two-hour sitting exposure. Previous literature has shown that participants completing a 3-minute walking break, reduced pressure mapping kinetics, and self-reported pain ratings back to baseline levels for the subsequent sitting trials (De Carvalho *et al.*, 2015). As the sudden loading study was part of a larger data collection, the walking break was unavoidable. However, the impact of

the walking break was considered minimal because it occurred after the perturbation trials at time point MID. Also, participants began the second one-hour exposure of prolonged sitting immediately post walking break, therefore it is unliekly that the walking directly effected any measures in this study.

8.0 POTENTIAL APPLICATION

The responses observed in the current research could be used to identify key determinants of LBD or the likelihood of developing LBP following a period of prolonged sitting in response to unexpected perturbations with further study. Immediately, this research may be applicable to many environments where individuals lift post prolonged sitting and may be subjected to unexpected perturbances. For example, individuals in sedentary office environments may be subjected to lift boxes of similar dimensions and weight to the one use in the study. For example, nurses in hospitals may be subjected routinely to such situations. They may need to help lift patients and/or equipment immediately post prolonged sitting. Similarly, truck drivers and emergency services also experience prolonged sitting followed by bouts of lifting, where they may not be able to control what they are lifting (weight may shift unexpectedly). Therefore, the results of this research may aid in the general recommendations for these workers to minimize the occurrence of LBD/LBP. Such recommendations could be for individuals to refrain from lifting a load greater than 6.78 kg after prolonged sitting, unless they are able to walk for more than 3 minutes or complete other activity first.

9.0 CONCLUSION

The purpose of this research was to evaluate the effect of prolonged sitting on trunk muscle activation, kinematics and kinetics in response to unexpected perturbations. The prolong sitting exposure produced LBD in a 83.33% of individuals in the current study. This exposure alone caused increased trunk musculature activation and subsequently trunk stiffness, displayed through kinetic and kinematic alterations in response to the unexpected sudden loading. In addition to the sitting exposure, the response of individuals who developed LBD elicited a substantial increase in trunk muscle activation and muscle latency time. Further, the greater magnitude of spinal load (through the increase in trunk muscle activation) increased spinal stability though this was also potentially the mechanism that led to the progression of LBD (and perhaps in workers that sit habitually for longer durations, eventually LBI). However, it is also possible that reflexive activation of the trunk musculature is a safety mechanism, and by increasing stiffness and thus spinal stability could prevent the onset of LBI. Overall, the muscular, kinematic and kinetic responses observed in this study may signify key characteristic measures that can increase the likelihood of developing LBD and eventually LBI in individuals who experience prolong sitting exposures.

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