Models of stability, stiffness, pain catastrophizing, fear of movement and changes in these outcome measures due to a capsaicin experimental neck pain protocol.

by

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ABSTRACT

MODELS OF STABILITY, STIFFNESS, PAIN CATASTROPHYZING, FEAR OF MOVEMENT AND CHANGES IN THESE OUTCOME MEASURES DUE TO A CAPSAICIN EXPERIMENTAL NECK PAIN PROTOCOL.

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The purpose of our investigation was to determine the changes in upper back and lower back local dynamic stability (LDS) and lumbar rotational stiffness after experiencing neck pain, and to examine the models used to measure LDS and stiffness as well as the Pain Catastrophizing Scale (PCS) and Tampa Scale of Kinesiophobia (TSK). We found significant changes immediately after capsaicin/heat induced neck pain, in upper back LDS but not lower back LDS or lumbar rotational stiffness. Contrary to previous evidence LDS and rotational stiffness did not relate with one another. PCS and TSK were moderately correlated with LDS but weakly negatively correlated with stiffness. PCS scores significantly changed between baseline and experiencing pain. This suggests that lower back stability may not be influenced by pain similar to our experimental protocol and provides further avenues for continued research of the models we use to determine stability, catastrophizing and fear of motion.
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LIST OF SYMBOLS, ABBREVIATIONS OR NOMENCLATURE

EMG: Electromyography
EO: External oblique
IO: Internal oblique
LDS: Local Dynamic Stability
LD: Latissimus dorsi
LES: Lumbar erector spinae
MVC: Maximum Voluntary Contraction
PCS: Pain Catastrophizing Scale
RA: Rectus abdominus
S1: First sacral vertebra
T3: Third thoracic vertebra
T12: Twelfth thoracic vertebra
TES: Thoracic erector spinae
TSK: Tampa Scale of Kinesiophobia
VAS: Visual Analogue Scale
$\lambda_{max}$: Maximum Lyapunov Exponent
1 Introduction

Low back pain affects 80-85% of people over their lifetimes (WHO, 2003), yet it is most commonly diagnosed as non-specific (Koes et al., 2006), as we have yet to understand many of the mechanisms and causes of low back injury and pain. Since the isolated ligamentous lumbar spine buckles with a compressive load of 88N (Crisco et al., 1992), spine stability has been conceptualized to be a critical component of spine health. It is hypothesized that disruption in the function of the passive, active or control (neurological) systems of the spine can lead to instability which can cause injury or pain (Panjabi, 1992). A transient loss in a vertebral segment’s stability can conceivably cause unexpected and uncontrolled displacements that could irritate nociceptors or free nerve endings of surrounding tissues or cause buckling (Cholewicki and McGill, 1996). In response, the active and control systems can try to counteract these displacements, but at the risk of overloading or injuring muscles (Cholewicki and McGill, 1996) and over time causing cumulative overload of the spine itself. Current evidence strongly suggests that the possibility of such an instability causing injury and pain has biological plausibility (Preuss and Fung, 2005). However, confirming this is difficult as numerous variables and their interactions regarding the three different systems (passive, active and control), many of which are unknown, need to be accounted for and quantified. Thus, to investigate spine stability the current best available approach is to use models based on well accepted evidence and sub-models.
Since these models can only create a construct by which to estimate stability, comparing different tasks, populations and interventions can help better understand what variables may influence spine stability and hence risk of injury and pain. Lower back pain patients have been studied to determine differences compared to healthy controls in order to understand the etiology and consequences of lower back pain. The caveat to this approach is that lower back pain is heterogeneous such that the cause of pain can vary and cannot be reliably diagnosed. It is reasonable that different pathologies would affect spine stability in different ways. Hence a homogenous sample would be ideal. This can be accomplished by inducing pain in healthy participants, using experimental models. Recent research has used a topical capsaicin/heat pain sensitization model, to experimentally induce lower back pain, and shown that it can decrease lumbar rotational stiffness (directly related to mechanical stability) as well as lower back local dynamic stability (LDS) (Ross et al., 2015). Conversely it has yet to be established if pain elsewhere in the body, specifically other regions of the spine, can affect the stability of the lower back.
2 Literature Review

2.1 Modelling Spine Stability:

The laws of mechanics have been applied to modelling spine stability as early as 1989 (Bergmark, 1989). This model focused on the lumbar spine during upright standing. Although the effect of surrounding muscles was considered, a very simplified approach was used to determine the force and stiffness of these muscles. This approach did not account for individual differences in muscle activity but instead used fixed values to solve the equations of equilibrium. It was not until 1996, that Cholewicki and McGill developed an EMG assisted optimization model to determine muscle forces, to account for biological variability, as each individual will have a different muscle recruitment strategy. This model was anatomically detailed and used equations of elastic potential energy to estimate static mechanical stability of the lumbar spine during slow (‘quasi-static’) 3D motion. Under this static definition, when perturbed the system would be stable when the second derivative of its potential energy, which is the sum of the passive and active elastic energy (stiffness) minus the external work performed on the system, is a positive number (i.e. at a minimum). In this case the system will always be stable if the active stiffness, provided by the muscles, is increased by the neuromuscular system, to counteract the work performed on the system, regardless of the task. This method of modelling ignores kinetic energy and dampening of viscoelastic tissues, but uses slow and smooth movements to keep acceleration low, in order to justify the use of a stability concept that only adheres to situations of static equilibrium.
For a dynamic task, when the system is perturbed, it is not only important to resist a perturbation by increasing active stiffness, but also modulate muscle activation in such a way that the intended trajectory of motion is maintained (Granata and England, 2006; Reeves et al. 2007). In this case, similar to the static system, if stiffness is insufficient at any given instant to counteract external work, the system will be unstable. In addition, too much muscle activation or muscle activation at an inappropriate time, in response to a perturbation can alter the trajectory of motion which could result in an unstable system. In contrast, in a dynamic system, the system may initially deviate its position and trajectory after being perturbed. However, it will remain stable if it returns to its trajectory as time progresses without exceeding the physiological range of the joint. Understanding the time dependent changes due to the neuromuscular control of the system, hence becomes important when defining the spine as a dynamic system to estimate spine stability during motion (Granata and England, 2006; Reeves et al. 2007).

Granata and England (2006), postulated that dynamic stability of the spine can be estimated by the time-dependent behaviour of kinematic variance during a repetitive task. This was done by using the maximum Lyapunov exponent ($\lambda_{\text{max}}$) as a simplified method of estimating Lyapunov exponents for the whole kinematic time-series of a repetitive task (Rosenstein et al., 1993). Termed as local dynamic stability (LDS), $\lambda_{\text{max}}$ can be used to estimate whether the kinematic variance expands or contracts over time (behaviour of kinematic variance). It is assumed that variance in kinematics arises due to biomechanical disturbances in motion that are controlled by the neuromuscular
system (control and active systems) in an attempt to return the system to an equilibrium along its trajectory. A system is stable if the behaviour of kinematic variance contracts, as this is presumably the neuromuscular system controlling the motion towards the intended trajectory. However, the implications of kinematic variance have yet to be fully understood. In the past, variance in kinematic trajectories was postulated as being a problem, but recent evidence suggests it can benefit the motor control system (Latash, 2012).

To account for the activity of the neuromuscular system, an EMG driven biomechanical model that calculates the muscular contribution to lumbar spine rotational stiffness, a first approximation of mechanical stability, has been used alongside the estimation of LDS of the lower back using $\lambda_{\text{max}}$ (Graham and Brown, 2012; Beaudette et al., 2014; Ross et al., 2015). This model uses EMG estimated force and kinematic dependent muscle length to estimate the instantaneous stiffness of 58 muscle lines of action in an anatomically detailed model (Brown and McGill, 2010). Since the neuromuscular system is primarily responsible for counteracting instability caused by any perturbation, this allows insight into how the system is responding to provide spine stability. Previous evidence suggests that there is a strong correlation between lower back LDS and the muscular contribution to lumbar spine rotational stiffness with three different increments in load, and weaker correlations with three increments in rate of movement (Graham and Brown, 2012). Ross et al. (2015) were able to find a similar strong correlation between baseline, experimental pain and recovery trials. However these correlations were made using only three trials, each
differentiated by a significant intervention. It could be possible that these outcome measures react similarly to the interventions tested rather than being directly related to one another. To better understand how LDS and rotational stiffness correlate, multiple baseline trials (without intervention) might give insight into how closely these two outcome measures are related. Conducting a test of multiple baselines would also help us understand the biological variance in these measures to determine what a meaningful change in these outcome measures is.

2.2 Capsaicin/Heat Sensitization model:

Topical Capsaicin cream is derived from the active ingredient in hot chili peppers and acts on unmyelinated C fibers, myelinated A sensory neurons and polymodal nociceptors which are sensitive to noxious pressure, heat and chemical irritants (Winter et al., 1995). It can be used along with heat as a non-invasive experimental pain sensitization model that can cause stable and long lasting areas of primary and secondary hyperalgesia with minimal skin damage (Petersen and Rowbotham, 1999). Hyperalgesia is the increased response to a painful stimulus and usually occurs in the area of primary hyperalgesia. Allodynia occurs in the area of secondary hyperalgesia and is the increased response to a non-painful stimulus. The capsaicin/heat sensitization model has been used to study pain mechanisms and test various medications, as capsaicin can mimic the clinical pattern of neuropathic pain (Lötsch et al, 2015).
Topical application can also cause an unpleasant burning sensation that can be considered painful and lower heat pain thresholds (Roberts et al., 2011; Shenoy et al., 2011). This can hence be used not only to create sensitization but also a painful experience. To create a homogenous sample of lower back pain participants, Ross et al. (2015) applied capsaicin followed by heat, to induce pain in the lower back region, quantified by visual analogue scale (VAS). To compare our findings, this thesis used the same protocol but applied to the neck region.

2.3 Pain Catastrophizing and Fear of Movement:

Psychological factors can play a role in pain perception and thus can influence the mechanics of an individual in response to the perception of pain. The Pain Catastrophizing Scale (PCS) and Tampa Scale of Kinesiophobia (TSK) have been used in the clinic and research, to identify those who catastrophize about pain and those who are fearful of movement.

The PCS is a valid and reliable, thirteen item questionnaire with three sub-dimensions (rumination, magnification and helplessness) (Sullivan et al., 1995). Each item is scored on a five point scale (0-4) from ‘not at all’ to ‘all the time’. Rumination describes worry and inability to inhibit pain related thoughts. Magnification is the magnification of unpleasantness of painful events and expectations of negative outcomes. Helplessness describes the inability to deal with painful situations. Pain catastrophizing is strongly associated and a predictor of severity of pain experience, pain and illness behaviour, quality of life and disability (Lamé et al., 2005; Sullivan et al., 1995).
PCS scores had a negative correlation with lumbar spine rotational stiffness and lower back LDS during baseline, capsaicin and heat applied to the lower back, and recovery trials (i.e. higher PCS scores were correlated to lower stiffness and LDS) (Ross et al., 2015). A later investigation showed that PCS scores were able to differentiate between those who increased their LDS from those who decreased their LDS in response to a hypertonic saline injection (Ross et al., 2017).

The TSK is a 17 item questionnaire rated on a four point scale (1-4) from 'strongly agree' to 'strongly disagree'. It has been previously tested and shown good validity and reliability (Vlaeyen et al. 1995). The fear of movement has been associated and predictive of physical behaviour and disability (Gheldof et al., 2006; Leeuw et al., 2007). Furthermore, catastrophizing a pain experience can lead to and influence fear of movement (Vlaeyen and Linton, 2000). TSK scores have been associated with increased trunk stiffness in those with recurring lower back pain (Karayannis et al., 2013). Ross et al. (2015) found a negative correlation between TSK scores and lateral bend rotational stiffness but did not find any correlations between TSK scores and LDS. Ross et al. (2017) found that the TSK scores were higher for those who increased LDS in response to injected hypertonic saline, but PCS scores were a stronger predictor of LDS than TSK scores.

The majority of the available evidence has treated catastrophizing as a trait (dispositional) variable rather than a state (situational) variable (Quartana et al., 2009). Those who catastrophize report more intense pain (Sullivan et al., 2001; Sullivan et al., 2002; Edwards et al., 2004).
2006). It is plausible that pain experience and current pain state can influence catastrophizing and hence fear of movement. Asymptomatic individuals score lower than chronic pain patients, but the influence of pain state on psychological constructs such as PCS scores needs further investigation (Sullivan et al., 2001). Mixed results have been reported on the changes in PCS scores after experiencing various experimental pain protocols (Quartana et al., 2009). These studies asked participants to answer the PCS based on what they felt during the experimental pain protocol. However, pain catastrophizing may require a stimulation above a certain pain intensity threshold which may not be appropriately achieved by experimental pain protocols (Edwards et al., 2008). In addition, the differences between scores at baseline versus post experimental pain may be confounded by the actual pain experience (Quartana et al., 2009). While still controlling pain stimulus by using experimental pain, if participants were instructed to imagine a single painful experience while answering the PCS and TSK, rather than focusing on what they currently felt, it may give better insight as to how their current state influences their catastrophizing thoughts or fear of movement while trying to attenuate the influence of the specific pain experience and rather focus on the changes in cognitive processes. Comparing changes in scores between baseline and experimentally induced pain, while focusing on a single imagined pain experience, may give insight into how these constructs are influenced by the temporality of an individual’s current pain state.
3 Objectives and Hypotheses:

The primary purpose of this investigation was to quantify upper and lower back LDS and the muscular contribution to lumbar spine stiffness after an experimental neck pain protocol. The experimental neck pain protocol was hypothesized to reduce stiffness and LDS values compared to baseline, as in Ross et al. (2015).

The secondary objective was to better understand the models we use to estimate spine stability. This included analyzing the variance in LDS and lumbar spine rotational stiffness during repeated bouts of flexion/extension and understanding the correlation between LDS and lumbar spine rotational stiffness during repeated baseline trials. LDS and stiffness were hypothesized to not be significantly different amongst repeated bouts of flexion/extension. Also, LDS and stiffness were hypothesized to correlate as in previous investigations.

The tertiary objective was to analyze the effect of recent pain experience on pain catastrophizing and fear of movement. Since catastrophizing can lead to fear of movement and chronic pain patients have a higher PCS score than healthy individuals, PCS and TSK scores were hypothesized to be higher after experiencing the experimental pain protocol.
4 Methods

4.1 Participants:

Twenty one healthy males (mean ± SD age 26 ± 4.7 years; mass 83.6 ± 25.1 kg; height 178.7 ± 5.1 cm) were recruited. Exclusion criteria were any lower back or neck pain or injury within the last year, or allergies to adhesives or gels. A health screening questionnaire was completed by each participant that included questions about previous pain, injuries, musculoskeletal disorders and physical activity. These matters were additionally verbally discussed with participants. All procedures were approved by the University of Guelph Research Ethics Board.

4.2 Equipment and Calibration:

Skin overlaying the abdominal and back muscles of interest was cleaned with alcohol and shaved if necessary. Surface EMG electrodes (Ambu Blue Sensor, Medicotest Inc., Olstykke, Denmark) were applied bilaterally to the skin overlaying the latissimus dorsi (LD), thoracic erector spinae (TES), lumbar erector spinae (LES), external oblique (EO), internal oblique (IO) and rectus abdominus (RA)(Figure 1). The LD electrodes were placed inferior and lateral to the inferior angle of the scapula in the direction of the LD muscle fibre orientation. TES and LES electrodes were placed on the bulk of the muscle adjacent to the ninth thoracic vertebrae and the third lumbar vertebrae. EO electrodes were placed midway between the iliac crest and the twelfth rib, on the lateral aspect of the torso at an inferior angle to match the orientation of the
muscle fibres. IO electrodes were placed inferior to the inguinal ligament to match the orientation of its fibres at this location. RA electrodes were placed on lowest section of the RA over the muscle bulk. Ground electrodes were placed over the acromion. Three maximal voluntary contractions (MVCs) for each of these muscle groups were performed isometrically against the resistance of the researcher.

![Figure 1. Equipment set up showing placement of EMG electrodes and electromagnetic sensors.](image)

For the LD muscle MVC the upper arm was abducted and elbow flexed, both at 90 degrees, while the participant maintained a flexed hip to allow for their torso to be
approximately parallel to the floor. The opposite arm was placed on a Roman Chair for support, as the participant attempted to extend the abducted arm against the resistance of the researcher. For the back muscles (TES and LES), the participants lay prone on the Roman Chair with arms across their chest and a neutral spine. An s contraction was performed by attempting to extend their back against the resistance of the researcher. These MVCs were performed over a span of 10 seconds with a gradual increase to maximum followed by a gradual decrease in contraction effort. For the abdominal muscles (RA, IO, EO) the participants lay supine on the Roman Chair with their arms across their chest and their upper body supported by the researcher. Participants then attempted to flex their spine, laterally bend their spine in each direction and twist towards each side, against the resistance provided by the researcher. Each contraction was performed consecutively over a span of 10 seconds before the participant rested.

Electromagnetic sensors (Polhemus Liberty, Polhemus, Colchester, VT, USA) were affixed with double sided tape to the skin overlaying the first sacral vertebra (S1), twelfth thoracic vertebra (T12), third thoracic vertebra (T3), and the back of the skull using a headband. Participants then performed a quiet standing trial, maximum spine flexion/extension trial, and a static 5 second hold with the hip flexed and trunk angle at approximately 45° holding a 10 kg weight. The maximum flexion/extension and 45° trunk flexion trials were used to account for variability between individuals when using the rotational stiffness model.
4.3 Protocol:

Upon arrival, participants completed a consent form, general health questionnaire asking them about previous and current pain and injury, the PCS and TSK. After the equipment setup, a total of 8 rounds of 38 repetitions of continuous spine flexion/extension were performed at a pace of 4 beats per flexion/extension cycle paced by a metronome set to 60 beats per minute (0.25 Hz). Collection of data started after the first 3 cycles (35 flexion/extensions were recorded). Participants were required to repeatedly touch a target at chest height and a target at knee height with arms extended. Hips were constrained using an adjustable table and a strap around the pelvis. Before and after each flexion/extension trial, participants completed a 10 cm VAS indicating how much pain they were experiencing, in their neck and whole body, from no pain to the worst pain imaginable. Participants rested for at least 5 minutes between each trial except between trial 4 and 5 (B4 and P1, Fig.2) where they rested on a massage bench for a minimum of 15 minutes while the pain protocol was applied. Feet position, the adjustable table height (hip constraint) and target heights were kept consistent between each trial for each participant.

After the first 4 rounds of flexion/extension trials (baseline trials: B1-B4, Fig.2), the participant lay prone on a massage table and the neck region between the participant’s hairline and C7 was preheated with hot, moist towels heated with 40° C water for 5 minutes. Towels were replaced every 30 seconds. Next, topical capsaicin (0.075%; Zostrix, Medicis, Toronto, ON, Canada) was applied to the neck region
between the participant’s hairline and C7 (superior and inferior borders) and the sternocleidomastoid muscles (lateral borders). The cream was rubbed in and allowed to soak for 5 minutes. After this period a moist towel, heated with 40° C water, and a hot gel pack, was applied for 5 minutes (rekindling). The participant completed a VAS before heat was applied and just before the end of the rekindling period. Participants then performed another 4 rounds of flexion/extension trials.

After trial 5 (P1 Fig.2) the participants completed the PCS and TSK a second time. In order to provide some control of the participant’s thoughts, when answering the PCS and TSK, they were instructed to imagine if they were in pain and how they would react when in pain. After the pain protocol they were also instructed to imagine being in pain instead of being specific to any pain that they may have felt with the capsaicin. On this second occasion, an additional instruction was to pretend as though they were seeing these scales for the first time and to avoid trying to remember their previous answers. Approximately 2 hours elapsed between the administrations of the scales.

After trial 6 (P2, Fig.2) participants were asked the following questions:

- What did the pain protocol on your neck feel like?
- What has been your worst pain experience? How does it compare to what you have experienced with the experimental cream protocol on a scale of 1 to 10 with 10 being your worst pain experience?
- How would you compare and contrast what you just experienced with the experimental protocol versus your worst pain experience?
Figure 2. Pictorial representation of protocol. B1, B2 B3 and B4 refer to the first four baseline trials. P1, P2, P3 and P4 refer to the four trials after the pain protocol. VAS was conducted before and after B1, B2, B3, B4, P1, P2, P3 and P4. VAS was also conducted during the experimental Pain Protocol, after the application of cream only and after the application of the hot gel pack. PCS and TSK were completed by the participant on arrival to the lab as well as after P1.

4.4 Data Analysis:

Direction cosines of the electromagnetic sensors were collected at 32 Hz and were used to calculate relative 3D Cardan angles between sensors using a flexion-extension, lateral bend and axial twist sequence for the lower back (T12 to S1), upper back (T3 to T12) and neck (back of the skull to T3). However, the sensor attached to the back of the skull routinely exceeded the validated range of the sensor (i.e. traveled too far from the electromagnetic source) and therefore these data were not considered reliable and the neck data will not be reported. Data were low-pass filtered using a 10 Hz dual-pass effective fourth order Butterworth filter and standing bias was removed by subtracting angles calculated from the quiet standing trial.
Local dynamic stability (LDS) for both the upper back and lower back was estimated using the maximum finite-cycle Lyapunov exponent ($\lambda_{\text{max}}$). To do so, the first 5 cycles of the calculated 3D angles were removed and the 30 remaining cycles were time normalized to 3840 frames, while maintaining the temporal variability, as the length of the time series can influence the $\lambda_{\text{max}}$ (Bruijn et al., 2009). Angles were shifted to positive values and the Euclidean Norm was calculated to account for kinematic fluctuations in all three dimensions (Beaudette et al., 2016). A state space with six dimensions (selected by a global false nearest neighbours analysis (Kennel et al., 1992)) was reconstructed using the following method of delays:

$$Y(t) = [N(t), N(t + Td), N(t + 2Td), ..., N(t + (n - 1)Td)]$$

(1)

where $Y(t)$ is the n-dimensional state space, $N(t)$ is the original Euclidean norm data, $Td$ is the constant time delay set to 13%, and $n$ is the number of reconstructed dimensions. Maximum finite-cycle Lyapunov exponents were then calculated by analyzing the exponential rate of divergence of neighboring trajectories in the reconstructed state space, using the equation from Rosenstein et al. (1993):

$$y(i) = \frac{1}{\Delta t} \{\ln d(j)\}$$

(2)

where $\{\ln d(j)\}$ represents the average logarithmic divergence ($d(j)$), for all pairs of nearest neighbours ($j$), throughout a certain number of time delays ($\Delta t$). $\lambda_{\text{max}}$ was estimated using the slope of the linear line of best fit of the average logarithmic rate of
divergence, for approximately 0-0.5 movement cycles. Note that a lower $\lambda_{\text{max}}$ indicates a higher LDS.

EMG was collected at 2048 Hz. Bias was removed from the signal which was then low-pass filtered using a 500 Hz dual-pass effective fourth order Butterworth filter. EMG was then full-wave rectified and low-pass filtered, using a 2.5 Hz second order Butterworth filter, to create a linear envelope. This was then normalized to the participant’s respective linear enveloped maximum MVC. The normalized linear enveloped EMG was down-sampled to 32 Hz.

Both lumbar spine angles and normalized linear enveloped EMG were used to determine the muscular contributions to lumbar spine rotational stiffness, using an anatomically detailed EMG-driven model representing 58 muscle lines of action crossing the L4/L5 joint (Brown and McGill, 2010). Muscle force was estimated using:

$$F_m = NEMG_m \times PCSA_m \times \sigma_m \times l_m \times v_m \times G$$  \hspace{1cm} (3)

where $F_m$ is the force produced by muscle (m) about its line of action in Newtons, $NEMG_m$ is the normalized EMG signal for muscle (%MVC), $PCSA_m$ is the physiological cross-sectional area of muscle (cm$^2$), $\sigma_m$ is the stress generated by muscle set at 35 N/cm$^2$, $l_m$ is the length coefficient of muscle m (unitless), $v_m$ is the velocity coefficient of muscle (unitless), and $G$ is the participant specific calibration gain (unitless). Muscle force-length and force-velocity coefficients were adapted from McGill and Norman (1986). To accommodate for inter-participant variance, a participant-specific gain factor ($G$) was calculated by matching the L4/L5 moment calculated using 3DSSPP (Centre for
Ergonomics, University of Michigan, Ann Arbor, MI) during the static 45° loaded trunk flexion trial and the moment estimated by the EMG-driven model. This moment was calculated using a digital photograph of the participant during the trunk flexion showing the whole body in a sagittal plane view. Each participant’s height and mass were entered into 3DSSPP along with hand load (10 kg). Joint angles were measured using a protractor and additionally entered into 3DSSPP.

The muscular contributions to lumbar spine rotational stiffness were estimated as per Potvin and Brown (2005):

$$ S_z = \sum_{m=1}^{58} F_m \left[ \frac{A_x B_x + A_y B_y - r_y^2}{l} + \frac{q r_y^2}{L} \right]_m $$

(4)

where $S_y$ is the rotational stiffness about the flexion/extension axis of the L4/L5 joint, $F_m$ is muscle force (N), $l$ is the 3D length of the muscle vector that crosses L4/L5, $L$ is the full 3D length of the muscle, $r_y$ is the moment arm of the muscle force vector about the flexion/extension axis, $A_x$ and $A_y$ are the origin coordinates with respect to the L4/L5 joint at (0,0,0)m, $B_x$ and $B_y$ are the initial deflection or insertion (without deflection points) coordinates with respect to L4/L5, and $q$ is the stiffness gain relating muscle force and length to stiffness (value of 10 used). A $q$ of 10 was used based on previous modeling work that has estimated a range from approximately 0.5–50, with a mean of approximately 10 (Cholewicki and McGill, 1995; Crisco and Panjabi, 1991). Rotational
stiffness was also calculated about lateral bend and axial twist axes by appropriate substituting of coordinates. Rotational stiffness values were separated into the same 30 individual cycles as used for the Lyapunov analysis and time normalized to 101 samples per cycle (0-100% cycle). Maximum, mean, and minimum values were extracted for each cycle and then averaged across cycles within each trial for statistical analysis.

4.5 Statistics:

To analyze the variance between repeated baseline trials, a one-way repeated measures ANOVA was conducted. The independent variable was the four baseline trials (B1, B2, B3 and B4). The dependent variable was either the upper back or lower back maximum finite-cycle Lyapunov exponent ($\lambda_{\text{max}}$), maximum, mean or minimum rotational stiffness about each axis (flexion/extension, lateral bend, axial twist). Significance was considered if $p<0.05$ ($\alpha=0.05$) and Tukey HSD comparisons were conducted if any significance was found.

To analyze the effect of the experimental pain protocol one-way repeated measures ANOVAs were conducted on the average of the last three baseline trials (average of B2, B3 and B4), to account for the variability in $\lambda_{\text{max}}$ during baseline performance, compared to each of the four trials after the experimental pain protocol (P1, P2, P3 and P4). The decision to average the last three baseline trials was made based on the results of the statistical test described in the previous paragraph (specific results will be reported in the Results section (section 5). Here the independent variable was condition (Baseline, P1, P2, P3 and P4) and the dependent variable was $\lambda_{\text{max}}$ or
maximum, mean or minimum rotational stiffness about each axis (flexion/extension, lateral bend, axial twist) or VAS scores taken before each trial (α=0.05).

To analyze the relationship between stiffness and lower back $\lambda_{\text{max}}$ during the flexion/extension trials, Pearson’s correlations were calculated between $\lambda_{\text{max}}$ and the maximum, mean, and minimum, flexion-extension, lateral bend, and axial twist rotational stiffness for each participant. These correlation values were then averaged across all participants. Pearson’s correlations were also calculated for only the first four baseline trials for each participant and averaged across all participants.

To analyze the relationship between PCS and TSK questionnaire scores, VAS scores and stability (LDS and rotational stiffness), Pearson’s correlations were calculated between pairs of each of these variables. B1 and P1 values for LDS and rotational stiffness for all participants were correlated with PCS and TSK questionnaire scores before and after the pain protocol as well as VAS scores taken just before P1. PCS and TSK scores both pre and post pain protocol were also compared using Pearson’s correlations with VAS scores.

To compare the effects of experiencing the pain protocol on the scores of the PCS and TSK, pairwise t-tests were conducted to assess the changes in pain catastrophizing and fear of movement. The independent variable was the condition (baseline or post pain) and the dependent variable was either the score on the PCS, PCS subscales (Rumination, Magnification and Helplessness) or TSK (α=0.05).
5 Results

One participant was excluded from the analysis of VAS, rotational stiffness and LDS as they showed signs of fatigue during the protocol.

5.1 Baseline Trial Comparison:

No significant differences in $\lambda_{\text{max}}$ for the upper or lower back were found between the comparison of baseline trials (B1, B2, B3 and B4). There were significant differences in rotational stiffness such that maximum flexion/extension ($p<0.0001$) and maximum lateral bend stiffness ($p=0.0008$) were significantly higher during B4 than B1 and B2. Maximum flexion/extension stiffness was also significantly higher during B3 than B1. Mean flexion/extension stiffness was significantly higher ($p<0.0001$) during B4 and B3 than in B1. Minimum flexion/extension ($p=0.0026$) and mean lateral bend stiffness ($p=0.0170$) were significantly higher in B4 compared to B1.
Figure 3. Mean (+SEM) LDS of the upper back for the four baseline trials (B1, B2, B3 and B4). No significant differences were found.

Figure 4. Mean (+SEM) LDS of the lower back for the four baseline trials (B1, B2, B3 and B4). No significant differences were found.
Figure 5. Participant mean (+SEM) flexion/extension (FE) maximum, mean and minimum rotational stiffness for the four baseline trials (B1, B2, B3 and B4). Trials that were significantly (p < 0.05) higher than B1 are indicated by “*” and trials that were significantly (p < 0.05) higher than B2 are indicated by “#”.
Figure 6. Participant mean (+SEM) lateral bend (LB) maximum, mean and minimum rotational stiffness for the four baseline trials (B1, B2, B3 and B4). Trials that were significantly (p < 0.05) higher than B1 are indicated by “*” and trials that were significantly (p < 0.05) higher than B2 are indicated by “#”. 
Figure 7. Participant mean (+SEM) axial twist (AT) maximum, mean and minimum rotational stiffness for the four baseline trials (B1, B2, B3 and B4). No significant differences were found.

5.2 Effect of Experimental Neck Pain on Stability:

Because there were a number of statistically significant differences between the first baseline (B1) trial and later (B3 and B4) baseline trials, we decided to use the average of the last three baselines (B2-B4) as the comparison to the trials following the experimental pain protocol (P1-P4). Neck VAS scores were significantly (p<0.0001) higher during P1 and P2 compared to all the other trials and P3 was higher than P4 and baseline (Figure 8).
Figure 8. Mean (+SEM) VAS for neck pain, from no pain (0 cm) to worst pain imaginable (10 cm), taken before each flexion/extension trial for Baseline (average of B2-B4), and before the four trials after the experimental pain protocol (P1, P2, P3, P4). P1 and P2 were significantly (p < 0.05) higher than all other trials denoted by “*” and P3 was significantly (p < 0.05) higher than Baseline and P4 denoted by “#”. Note that Baseline values were zero since none of the participants had any neck pain prior to the experimental pain protocol.

Upper back $\lambda_{\text{max}}$ was significantly (p=0.0078) higher (indicating reduced LDS) in the first post pain trial (P1) compared to baseline and the third post pain trial (P3; 15 minutes post pain protocol) (Fig. 9). Lower back $\lambda_{\text{max}}$ was significantly (p=0.0278) lower in the third post pain trial (P3) than in the first post pain trial (P1) (Fig. 10). There were no significant differences amongst the baseline or any of the post pain trials for any of the rotational stiffness variables (Figs. 11, 12, 13). Mean flexion/extension stiffness was
lower during baseline than the pain trials and neared statistical significance (p=0.0730) (Fig. 11). Similarly maximum axial twist stiffness was lower during P1 than baseline or the other pain trials and neared statistical significance (p=0.0580) (Fig. 13).

Figure 9. Mean (+SEM) LDS of the upper back for average (B2-B4) baseline trial and four post pain protocol trials. P1 was significantly higher than baseline (average of B2, B3 and B4) and significantly higher than P3 (p = 0.0078).
Figure 10. Mean (+SEM) LDS of the lower back for the average (B2-B4) baseline trial and each of the four post pain protocol trials (P1, P2, P3 and P4). P1 was significantly higher than P3 (p = 0.0278).
Figure 11. Participant mean (+SEM) flexion/extension (FE) maximum, mean and minimum rotational stiffness for the average baseline (B2-B4) and the four trials post pain protocol (P1, P2, P3 and P4). Mean FE stiffness was significantly higher in P4 than baseline.
Figure 12. Participant mean (+SEM) lateral bend (LB) maximum, mean and minimum rotational stiffness for the average baseline (B2-B4) and the four trials post pain protocol (P1, P2, P3 and P4). No statistically significant differences were found.
Figure 13. Participant mean (±SEM) axial twist (AT) maximum, mean and minimum rotational stiffness for the average baseline (B2-B4) and the four trials post pain protocol (P1, P2, P3 and P4). Max AT stiffness was significantly higher during P3 than P1.

5.3 Relationship between Stability and Pain Questionnaires:

Weak (defined as r values between 0.20 and 0.39) to moderate (defined as r values between 0.40 and 0.59) correlations were found between lower back LDS and PCS scores as well as lower back LDS and TSK (Table1). Rotational stiffness was also weakly correlated with PCS and TSK scores (Table1).
Table 1. \( r \)-values for the correlations between \( \lambda_{\text{max}} \) values (inversely proportional to LDS) and questionnaire scores, and flexion/extension (FE) lateral bend (LB) and axial twist (AT) stiffness and questionnaire scores.

<table>
<thead>
<tr>
<th></th>
<th>PCS Pre</th>
<th>PCS Post</th>
<th>TSK Pre</th>
<th>TSK Post</th>
<th>VAS P1 Pre</th>
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<td></td>
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<td></td>
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<tr>
<td><strong>Lower Back ( \lambda_{\text{max}} )</strong></td>
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<tr>
<td>B1</td>
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</tr>
<tr>
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<td></td>
<td></td>
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</tr>
<tr>
<td>P1</td>
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</tr>
<tr>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B1</td>
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<td>0.10</td>
<td>0.14</td>
<td>0.17</td>
</tr>
<tr>
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<td></td>
<td></td>
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</tr>
<tr>
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<td>-0.09</td>
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<tr>
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<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td>Max FE</td>
<td>Mean FE</td>
<td>Min FE</td>
<td>Max LB</td>
<td>Mean LB</td>
</tr>
<tr>
<td>------------------</td>
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</tr>
<tr>
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<tr>
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</tr>
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<td>0.00</td>
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</tr>
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<tr>
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<tr>
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<tr>
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<td>Mean FE</td>
<td>Min FE</td>
<td>Max LB</td>
<td>Mean LB</td>
<td>Min LB</td>
</tr>
<tr>
<td>------------</td>
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<td>--------</td>
</tr>
<tr>
<td>Mean FE</td>
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<td>-0.17</td>
<td>-0.22</td>
<td>-0.16</td>
<td>-0.13</td>
</tr>
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</tr>
<tr>
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</tr>
<tr>
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<td>-0.22</td>
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<td>-0.08</td>
<td>-0.11</td>
<td>-0.05</td>
</tr>
</tbody>
</table>

### 5.4 Relationship between LDS and Rotational Stiffness:

Only very weak (defined as r values between 0.00 and 0.19) correlations were found between any of the rotational stiffness variables and lower back $\lambda_{\text{max}}$ (Table.2).
Table 2. $r$-values for the correlations between lower back $\lambda_{\text{max}}$ values (inversely proportional to LDS) and each of the rotational stiffness.

<table>
<thead>
<tr>
<th></th>
<th>MAX</th>
<th>MEAN</th>
<th>MIN</th>
</tr>
</thead>
<tbody>
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<td><strong>Flexion/Extension</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>All 8 trials</td>
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<td>0.02</td>
<td>-0.03</td>
</tr>
<tr>
<td>Baseline trials</td>
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<td>-0.16</td>
<td>-0.16</td>
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<tr>
<td><strong>Lateral Bend</strong></td>
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<tr>
<td>All 8 trials</td>
<td>0.07</td>
<td>0.16</td>
<td>0.08</td>
</tr>
<tr>
<td>Baseline trials</td>
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<td>0.02</td>
<td>-0.05</td>
</tr>
<tr>
<td><strong>Axial Twist</strong></td>
<td></td>
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</tr>
</tbody>
</table>
5.5 Effect of Pain State on PCS and TSK:

PCS scores were significantly (p=0.0172) higher after experiencing the pain protocol compared to baseline (Fig. 14). TSK were higher as well, but were not significantly (p=0.2375) different (Fig. 14). Among the subscales of the PCS, rumination was significantly (p=0.0183) higher after experiencing the pain protocol. Helplessness was higher after experiencing the pain protocol, but this did not reach statistical significance (p=0.0557). Magnification showed no significant difference. PCS and TSK scores showed very weak correlations with VAS scores (all were 0.15 > r > 0.03).
Figure 14. Mean (+SEM) PCS and TSK scores for baseline and post experimental pain protocol.

PCS scores were significantly higher post pain protocol (p = 0.0172).

6 Discussion

6.1 Objectives and Hypotheses Revisited:

The primary purpose of this investigation was to quantify LDS and the muscular contribution to lumbar spine rotational stiffness after an experimental neck pain protocol. The experimental neck pain protocol was hypothesized to reduce LDS and rotational stiffness values compared to baseline, as in Ross et al. (2015). The current investigation found that upper back LDS decreased after the experimental pain protocol and significantly increased in the third post pain trial (P3). Lower back LDS was only
significantly higher during P3 than in the first post pain trial (P1). There were no significant differences amongst the baseline or any of the post pain trials for any of the rotational stiffness variables. Hence our hypothesis was confirmed for the upper back regarding LDS but not the lower back regarding LDS or rotational stiffness.

The secondary objective was to better understand the models used to estimate spine stability. This included analyzing the variance in LDS and muscular rotational stiffness during repeated bouts of flexion/extensions and understanding the correlation between LDS and lumbar spine rotational stiffness during repeated baseline trials. LDS and stiffness were hypothesized to not be significantly different amongst repeated bouts of flexion/extension. We found only small non-significant differences in upper and lower back LDS amongst the four baseline trials. Maximum and mean flexion/extension and lateral bend rotational stiffness increased with each successive trial and reached statistical significance when comparing B4 to B1. Maximum flexion/extension and lateral bend stiffness were also significantly higher in B4 than B2. Maximum and mean flexion/extension stiffness were significantly higher in B3 compared to B1 and minimum flexion/extension stiffness was significantly higher in B4 than B1. Hence our hypothesis was confirmed that there was no significant difference in LDS amongst repeated baseline trials. However contrary to our hypothesis, flexion/extension and lateral bend rotational stiffness increased with successive bouts of baseline trials. It was also hypothesized that LDS and stiffness would correlate as in previous investigations. This investigation found almost no correlation between LDS and rotational stiffness. Thus our hypothesis was rejected.
The tertiary objective was to analyze the effect of recent pain experience on pain catastrophizing and fear of movement. PCS and TSK scores were hypothesized to be higher after experiencing the experimental pain protocol. This was confirmed for PCS but not for TSK.

6.2 Effect of Experimental Neck Pain on Stability:

The joints of the body are interlinked through anatomical structure and hence function. They work in a coordinated fashion to perform movement. The function of each joint can directly influence the demand and hence the function of the joints adjacent to it. For example, surgical fusions that provide strong mechanical restriction to individual vertebral segments are associated with degenerative changes in adjacent vertebral segments (Mannion et al., 2014). Even in the case where minimal mechanical restriction is applied to the lower back, using a liquid bandage overlaying the skin to cue a change in motion at a single vertebral segment, can increase the motion required at adjacent segments (Beaudette et al., 2018). Further, restriction of the whole lower back, using an orthopedic brace (Wu et al., 2014) or using tactile feedback to cue an individual to restrict lumbar motion (Pinto et al., 2018), can influence the motion at adjacent joints such as the hips and knees. Restricting joints that are more distal, such as the ankle, can influence lumbar spine mechanics, as it can influence the entire kinetic chain (Beach et al., 2014). Thus the mechanics of the lower back can influence the joints adjacent to it, and vice versa. Neck pain is the fourth leading cause of disability, ranked
behind lower back pain which is the first (Murray et al., 2013). The incidence of neck pain is strongly associated with lower back pain and those with pain in one of these regions (neck or low back) are fifteen more times likely to report pain in the other region (Fernández-de-las-Peñas, 2011). Since the causes of lower back pain are still yet to be fully understood, understanding how the adjacent joint systems such as the neck can influence the risk of injury at the lower back can be of importance in exploring possible explanations for certain cases of nonspecific lower back pain.

Unfortunately the sensor placed on the head of the participants frequently exceeded the range of the electromagnetic field and cannot give us reliable data about the kinematics of the neck region. We observed that some participants moved their neck less after experiencing the pain protocol, but did not want to draw their attention to how they were moving by asking them about their own perception of their motion, as we wanted to capture their natural reaction. When asked what the pain protocol felt like, one participant noted that he was moving his neck less compared to the baseline trials. Those with neck pain can have reduced range of motion (Stenneberg et al., 2017) that is usually associated with further negative outcomes (Snodgrass et al., 2014). Those with chronic neck pain have also been found to move their neck with lower velocities and acceleration than healthy individuals (Tsang et al., 2013) and increased muscle coactivation (Tsang et al., 2014). This suggests that neck pain could alter neuromuscular control of the neck and hence possibly alter kinematic variance or LDS.

Immediately after the pain protocol the LDS of the upper back was lower than baseline and then increased as the participant felt less pain in the subsequent trials.
This would suggest that the region of the upper back responded to the pain in the neck by expanding its kinematic variance through the trial directly after the pain protocol (P1), compared to the baseline value. This divergence in kinematic variance could suggest that the upper back was not able to adjust its trajectory behaviour and became more unstable by increasing its chances of exceeding physiological limits (buckling). However all of our participants were still able to continue the experimental task (flexion/extension to touch targets) and none of them reported any buckling or discomfort. Our results also show that LDS doesn’t change significantly amongst baseline trials which suggests that a statistically significant change in LDS in response to the pain may be biologically relevant (section 6.3.1). However, it is still unclear as to how large of a significant change is associated with risk of injury or pain. An alternate way to interpret these changes is that the upper back may have had to expand its kinematic variance to adjust to any alterations in the neck in order to keep the whole spine within a margin of kinematic variance to prevent exceeding physiological limits (prevent buckling) and maintain trajectory (maintain dynamic stability). It appears as though most of the adjustment needed to respond to alterations at the neck were sufficiently compensated at the upper back as the lower back LDS did decrease, but was not statistically significant.

Interestingly, as reported pain (VAS) decreased, both upper and lower back LDS significantly increased during P3 compared to P1. It may be possible that as pain decreases, the neuromuscular system attempts to readjust and return LDS to the baseline value but overshoots its adjustment. This suggests that it may take a few
attempts (trials) for the neuromuscular system to respond and change the behavior of its kinematic variance (LDS). This concept of adjustment between each trial may suggest a learning effect that is further explored in section 6.3.1.

The psychological perception of pain can influence the effect of pain on an individual's mechanics. In a previous study, those with higher PCS scores had lower LDS during baseline, capsaicin experimental lower back pain, and a recovery trial after the pain had subsided (Ross et al., 2015). They also had lower rotational stiffness during the pain and recovery trials. These authors also found that those with higher TSK scores had lower lateral bend rotational stiffness. Using the same capsaicin experimental pain protocol on the neck, we found weak to moderate negative correlations between lower back $\lambda_{\text{max}}$ and both the PCS and TSK, indicating that those with higher scores had higher LDS (opposite of the Ross et al., 2015 study). We also found weak negative correlations between rotational stiffness and PCS and TSK suggesting those with higher scores had lower stiffness (similar to Ross et al., 2015). These findings are surprising as previous evidence (Graham and Brown, 2012; Ross et al., 2015) has shown LDS and rotational stiffness to be strongly correlated (see section 6.3.2 for further discussion). Ross et al., (2015) reported average VAS scores just below 4.0 cm before the flexion/extension task, after applying capsaicin and heat to the lower back. The mean VAS score for our sample population was just above 4.0 cm (Figure 8). Since the same protocol was used and participants reported similar intensities of pain, the difference in the correlations between LDS and rotational stiffness with the PCS and TSK, for the pain trial, may be due to differences in the location of pain. Specifically,
differences in lower back LDS and rotational stiffness may be due to the fact that we applied the pain to the neck instead of directly to the lower back, as in Ross et al (2015). If LDS and rotational stiffness of the neck were measured it may have shown correlations with the PCS and TSK but this would need further investigation. The differences between our study and Ross et al. (2015), in terms of the correlations between LDS and rotational stiffness with the PCS and TSK, for the baseline trial, may be due to the differences in population samples. Unfortunately we could not compare our PCS and TSK scores with those from Ross et al. (2015) as they were not reported. This would have given us insight into the differences between our samples. Ross et al., (2017) used hypertonic saline injections, instead of capsaicin, to induce acute pain in the lower back, and also found differing results compared to Ross et al., (2015). This suggests that type of pain should also be considered.

Overall we did not observe any significant changes to the lower back when we induced neck pain via the capsaicin experimental pain protocol. This suggest that neck pain may not influence the mechanics of the lower back. However, this may be specific to the type of pain as it is possible that more intense pain may show different results. Intense pain may increase catastrophizing and hence response, but the required threshold of intensity may simply not have been achieved by our experimental protocol (Edwards et al., 2008). When asked how they felt the experimental pain protocol compared to their worst pain experience, participants in the current study noted that they were not as threatened or fearful of the experimental pain since they knew its cause and that it would pass. A different intensity or type of pain at the neck may
produce different results. In addition, the lower back can be more sensitive to innocuous thermal stimuli when compared to other body parts (Tracy et al., 2016) which suggests a higher pain intensity at the neck may be required to match the same experimental protocol applied to the lower back. Another possible reason for differences between our findings and Ross et al. (2015) may be the repeated baselines performed before the experimental pain protocol in our study, as it may have provided practice and learning that could have helped the participants react to our experimental neck pain protocol (see section 6.2.1).

6.3 Analysis of Stability Models:

The concept of stability is complex and quantifying stability can be even more complex. Understanding the intricacies of the models used to quantify stability is important in understanding the implications of the empirical evidence created using these models.

6.3.1 Repeated Baseline trials:

Previous research measuring LDS has only examined the difference between a single baseline trial and an intervention. Since the change in behavior of kinematic variance, described by LDS, does not relate to a specific biological feature, it is important to understand the variance that can occur in a baseline trial, to understand the biological significance of a change in LDS. We found that
baseline $\lambda_{\text{max}}$ values slightly vary and show a general decrease (indicating greater LDS) from the first to the fourth baseline trial, but these changes are not statistically significant. However, the relationship between a statistically significant change and a biologically relevant change in this variable is unclear. Therefore, the magnitude of a biologically or clinically meaningful change in LDS (i.e. one that corresponds to a risk of injury or pain) needs to be further investigated.

The experimental task (repeated trunk flexion/extensions) can be considered a novel task as it is not performed as part of common activities of daily living or work. If so, the general increase in LDS for both upper and lower back may be indicative of a learning effect. As the participants become accustomed to the experimental task, they are able to constrain their kinematic variance slightly, to possibly improve performance of the task and minimize energy expenditure or improve precision of kinematic trajectories while touching the given targets. This could also influence the effects of the intervention that is tested, as this learning effect may better equip the participant to adapt or respond to a perturbation. If the task has been practiced or learned, an intervention such as experimental neck pain may not perturb LDS to the same extent as it would for a novel task. Future work should examine if this is truly the case.

Flexion/extension and lateral bend rotational stiffness increased with each successive baseline trial. Since EMG amplitude can increase with fatigue, the increase in stiffness may be indicative of fatigue as the participant had to perform
38 repetitions of a novel task. However, this unlikely as each participant had a minimum of 5 minutes of rest between each trial. In addition there was a minimum of 15 minutes of rest while performing the experimental pain protocol, but rotational stiffness continued to increase after the pain protocol. When questioned about fatigue only one participant said that they did perceive some fatigue and this was clear when examining their kinematic data. This participant was excluded from further analyses of LDS and rotational stiffness data. Some participants noted that they felt slightly fatigued, specifically in their lower back, after the first trial but subsequent trials were not fatiguing. An alternative explanation may be that the increased rotational stiffness may be due to increased input from the neuromuscular system (increased EMG) to control the spine which would support the hypothesis that there was a learning effect with each trial. The perception of decreased fatigue after the first trial supports the hypothesis that participants may have improved efficiency by constraining their kinematic variance to decrease energy expenditure. However, this would need to be further investigated by examining the individual EMG channels to determine what specific changes led to the increase in rotational stiffness.

6.3.2 Correlation between LDS and Rotational Stiffness:

Graham and Brown (2012) reported strong correlations between LDS of the lower back and rotational stiffness across three trials, where each trial was conducted with a different load being lifted, and weaker correlations when three trials were compared in which the rate of movement differed. Ross et al. (2015)
later showed similar correlations between three trials, each under a different experimental condition (baseline, experimental lower back pain and recovery). However, each trial was conducted under a different experimental condition, hence it is possible that both these models (LDS and rotational stiffness) merely change similarly in response to the specific interventions tested. It is possible that these models may not correlate to each other with regard to the more subtle variance that occurs across repeated trials of the same condition.

Our data show that LDS and rotational stiffness may be complimentary to each other when experimental conditions are controlled and kept constant (see section 6.3.1). However when correlating the four baseline trials, for each participant, there is clearly very little correlation between the two models. This suggests that subtle variation in both these models are not directly related to each other and thus changes in one cannot be considered indicative of similar changes in the other. This is vital in applying empirical evidence obtained from these models, as it suggests that both models would be ideally used together, as they likely provide different and complimentary information, to be able to make conclusions on both static and dynamic stability as well as both the neuromuscular system and its kinematic output.

6.4 Effect of Pain State on PCS and TSK:

Understanding the intricacies of the psychological constructs of catastrophizing and fear or movement, measured by the PCS and TSK respectively, can help improve the
way these questionnaires are used and the inferences drawn from them. We found that after experiencing the experimental pain protocol that PCS increased significantly and TSK increased as well but this was not statistically significant. This suggests that these constructs aren’t static over time (not solely a trait variable) and can change with an individual’s pain state. When administering these questionnaires it is important to consider the state of pain the individual is experiencing. A score that may be considered normal, may change, after or while experiencing pain, to a score that would identify the individual as a catastrophizer or as kinesiophobic. In our sample, only one individual would have been considered a catastrophizer based on his baseline PCS score and another participant would have been considered a catastrophizer based on his post pain PCS score compared to normative data from Sullivan (2009). Our results show very weak correlations with VAS suggesting that the PCS and TSK did not predict how the participants would respond to the experimental pain protocol. This may be because in our sample our participants had relatively low PCS scores compared to normative data (Sullivan, 2009). However, compared to previous evidence ours is unique as we instructed participants to focus on an imagined pain experience on both occasions (baseline and after the pain protocol) rather than on their current pain state. It appears that current pain state was still able to influence their PCS and TSK scores regardless of instruction. This may suggest a subconscious role of the current pain condition. Although the experimental pain may not have been intense enough to create catastrophizing or fearful thoughts, our instruction to imagine a painful experience and specifically to answer the questions while not considering the specific experience from
the pain protocol, should have negated the influence of the specific characteristics of the experimental pain protocol. Rather it may have allowed us to test how responses to the PCS and TSK change at a subconscious level when in a painful state.

Rumination was significantly higher after the pain protocol and helplessness was higher but not statistically significant. Other investigations of state pain have also showed that when comparing baseline and pain scores, rumination and helplessness are the categories that change the most. However, these sub scales may need further investigation along with the PCS scale in order to truly understand how they are affected by the current state (Quartana et al., 2009). Rumination is defined as perseveratively thinking about negative content that can cause emotional discomfort and can exist as a trait or state psychological feature that can intensify the pain experience (Sansone and Sansone, 2012). The increase in rumination suggests that experiencing the experimental pain response may likely increase perseveratively thinking to increase the PCS score.

6.5 Limitations and Future Directions:

This investigation is not without limitations. A major limitation is the lack of data from the sensor placed on the head as it would give us a more clear understanding of the interconnectedness amongst the regions of the spine. We also used an experimental task where we restricted motion of the pelvis. A natural task without restriction may produce different results as all joints of the system would be able to move freely and adapt to any perturbation. Future research should focus on evaluating mechanical
changes in the neck region when experimental neck pain is applied to the neck to understand what these changes are and how they may apply in a real world scenario.

We have investigated some of the intricacies of the models used in our study but most of these models are limited by what we can measure and inferences from them are limited to what we understand about the human body. Since we provide evidence that LDS does not significantly change during baseline trials and learning of the experimental task, future work should determine what a significant change means on a biological level. Similarly to confirm the inference that a learning effect takes place between repeated baseline trials, our future goal is to examine the changes in individual channels of EMG. In addition this may improve our understanding of why rotational stiffness increased with each trial.

Our participant sample was limited to mostly those who do not catastrophize nor are kinesiophobic. It is possible that greater or different changes may be observed in those who have higher than normative PCS and TSK scores. Future work can study these populations to better understand how these psychological constructs relate to motion.
7 Conclusion

The purpose of our investigation was to determine the changes in upper back and lower back LDS and lumbar rotational stiffness after experiencing neck pain, and to examine the models used to measure LDS and stiffness as well as psychological constructs that relate to pain. We found the upper back decreased its LDS in response to neck pain and any accompanied mechanical changes that may have occurred at the neck. Limited adaptation was required at the lower back which resulted in no statistically significant changes in lower back LDS or rotational stiffness. We also found that flexion/extension and lateral bend rotational stiffness increased across repeated baseline trials, together with a non-statistically significant increase in LDS. This suggests that there may be a learning effect that takes place with each trial that might have helped participants respond to the experimental neck pain protocol. Contrary to previous evidence, LDS and rotational stiffness did not correlate to one another and may have merely adapted in a similar way to the experimental conditions provided in previous research. Finally, we found that PCS scores changed after experiencing the pain protocol even when the instruction was to focus on an imagined pain experience rather the specific pain of the protocol, which suggests a subconscious effect of pain state on PCS scores.
REFERENCES


