Accepted Manuscript

PAIN CATASTROPHIZING MODERATES CHANGES IN SPINAL CONTROL IN RESPONSE TO NOXIOUSLY INDUCED LOW BACK PAIN

Gwyneth B. Ross, Peter J. Sheahan, Brian Mahoney, Brendon J. Gurd, Paul W. Hodges, Ryan B. Graham

PII: S0021-9290(17)30209-9
DOI: http://dx.doi.org/10.1016/j.jbiomech.2017.04.010
Reference: BM 8191

To appear in: *Journal of Biomechanics*

Accepted Date: 10 April 2017

Please cite this article as: G.B. Ross, P.J. Sheahan, B. Mahoney, B.J. Gurd, P.W. Hodges, R.B. Graham, PAIN CATASTROPHIZING MODERATES CHANGES IN SPINAL CONTROL IN RESPONSE TO NOXIOUSLY INDUCED LOW BACK PAIN, *Journal of Biomechanics* (2017), doi: http://dx.doi.org/10.1016/j.jbiomech.2017.04.010

PAIN CATASTROPHIZING MODERATES CHANGES IN SPINAL CONTROL IN RESPONSE TO NOXIOUSLY INDUCED LOW BACK PAIN

Original Article

Gwyneth B. Ross[1,2], Peter J. Sheahan[2], Brian Mahoney[3], Brendon J. Gurd[2], Paul W. Hodges[4], and Ryan B. Graham[1,2*]

[1]School of Human Kinetics, Faculty of Health Sciences, University of Ottawa, Ottawa, Ontario, Canada

[2]School of Kinesiology and Health Studies, Faculty of Arts & Science, Queen's University, Kingston, Ontario, Canada

[3]School of Medicine, Faculty of Health Sciences, Queen's University, Kingston, Ontario, Canada

[4]School of Health and Rehabilitation Sciences, Faculty of Health and Behavioural Sciences, The University of Queensland, Brisbane, Queensland, Australia

Submitted to: Journal of Biomechanics

March 23rd, 2017

Keywords: *biomechanics, Lyapunov exponents, psychosocial, kinematics, local dynamic stability*

***** Corresponding Author:
Ryan B. Graham, PhD
Assistant Professor
School of Human Kinetics
Faculty of Health Sciences
University of Ottawa
125 University Private
Ottawa, ON K1N 6N5
E-mail: rgraham@uottawa.ca
Phone: +1 613 562 5800 x 1025
Fax: +1 613 562 5497

ABSTRACT

It is generally accepted that spine control and stability are relevant for the prevention and rehabilitation of low back pain (LBP). However, there are conflicting results in the literature, in regards to how these variables are modified in the presence of LBP. The aims of the present work were twofold: 1) to use noxious stimulation to induce LBP in healthy individuals to assess the direct effects of pain on control (quantified by the time-dependent behavior of kinematic variance), and 2) to assess whether the relationship between pain and control is moderated by psychological features (i.e. pain catastrophizing (PC) and kinesiophobia). Participants completed three conditions (baseline, pain, recovery) during a task involving completion of 35 cycles of a repetitive unloaded spine flexion/extension movement. The neuromuscular control of spine movements was assessed during each condition using maximum finite-time Lyapunov exponents (λ_{max}). Nociceptive stimulus involved injection of hypertonic saline into the interspinous ligament, eliciting pain that was greater than baseline or recovery ($p<0.001$). Although there was no overall main effect of the nociceptive stimulation (i.e. pain) on λ_{max} when the whole group was included in the statistical model ($p=0.564$), when data were considered separately for those with high and low PC, two distinct and well established responses to the pain were observed. Specifically, those with high PC tightened their control (i.e. stabilized), whereas those with low PC loosened their control (i.e. destabilized). This study provides evidence that individuals' beliefs and attitudes towards pain is related to individual-specific motor behaviors, and suggests that future research studying spine control/stability and LBP should account for these variables.

INTRODUCTION

It is generally accepted that spine control and stability are relevant for the prevention and rehabilitation of low back pain (LBP) (Cholewicki and McGill, 1996; Reeves et al., 2007; van Dieën et al., 2003), a major global public health concern (Global Burden of Disease Study 2013 Collaborators, 2015; Lee et al., 2015; Murray and Lopez, 2013). Histochemical, structural, and neuromuscular changes such as decreases in muscle cross-sectional area and fibre density (Demoulin et al., 2007a; Demoulin et al., 2007b), increased muscle fatigability (Demoulin et al., 2007a), abolition of the flexion-relaxation phenomenon (Demoulin et al., 2007a), reduced proprioception (Willigenburg et al., 2013) and kinesthesia (Ebenbichler et al., 2001), increased repositioning error and reduced precision control (Willigenburg et al., 2013), altered trunk muscle activation profiles (Larivière et al., 2000; van Dieën et al., 2003), and impaired local dynamic stability (Asgari et al., 2015; Graham et al., 2014) have been found in those with LBP.

There are major disadvantages to investigating patients with clinical LBP (Zedka et al., 1999). First, the heterogeneous nature of LBP compromises assessment of neuromuscular control and spine (in)stability as the relationship between motor control and pain may not be uniform (Brown et al., 2002; Demoulin et al., 2007b). Second, normative data are rarely available for comparison; there are few opportunities to collect pain-free and painful data from the same individuals, and data from other individuals provides an insensitive comparison because of the inherent variability in movement across individuals (Zedka et al., 1999).

To eliminate confounders, researchers have provided nociceptive stimuli to assess the effect of transient LBP on biomechanics. Methods include: comparing motor changes in those who develop LBP with standing/exercise (pain developers) versus those who do not (Miller et al., 2013; Nelson-wong et al., 2008), and induction of pain in pain-free individuals via noxious

heat (Dubois et al., 2011), electrical stimulation, hypertonic saline injection (Tsao et al., 2010; Zedka et al., 1999), or topical capsaicin cream (Dancey et al., 2014; Hung et al., 2014; Ross et al., 2015). It has been observed that thermal- (Dubois et al., 2011) and saline-induced (Hodges et al., 2003) LBP alter movement and muscle recruitment patterns. The first study to directly assess the effects on spine stability found that injection of hypertonic saline into the longissimus muscle increased mechanical stability over a small range of motion at slow movement speed, but without stereotypical between-subject changes in muscle activity patterns (Hodges et al., 2013).

Despite the benefits, there remains individual variation in responses to experimental pain (Hodges et al., 2013). Psychological factors are thought to moderate motor responses to pain and contribute to this variation, but results are conflicting. High kinesiophobia was associated with higher stiffness responses to global perturbations in patients with recurring episodes of clinical LBP (Karayannis et al., 2013). Conversely, during repetitive full range-of-motion spine flexion and extension movements, lower local dynamic spine stability (quantified using the non-linear time-dependent behavior of kinematic variance about the target movement trajectory (Granata and England, 2006; Granata and Gottipati, 2008)) and mechanical spine rotational stiffness (quantified using an EMG-driven spine model (Brown and McGill, 2010; Potvin and Brown, 2005)) were found in those with high pain and high pain catastrophizing in response to acute capsaicin-induced LBP (Ross et al., 2015). The differences might be explained by the methods to quantify control and stability/stiffness, the nature of the LBP, or the nature of the task.

The purposes of the present work were: 1) to use noxious stimulation to induce LBP in healthy individuals to assess the direct effects of nociceptive input on spine control, and 2) to assess whether the relationship between pain and spine control is moderated by psychological measures. Based on previous research, it was hypothesized that control strategies would change

in the presence of pain (Hodges et al., 2013; Ross et al., 2015) and that there would be a relationship between changes in control and pain catastrophizing (Ross et al., 2015).

MATERIALS AND METHODS

Participants

Sixteen healthy participants (8M, 8F), with no history of chronic LBP, were recruited for this investigation (Table 1). Prior to data collection, each participant read and signed an informed consent document that outlined experimental protocols. The Health Sciences Research Ethics Board at Queen's University approved the study (File No: 6011429).

(Table 1 approximately here)

Psychological measures

Participants completed two questionnaires related to psychological aspects of pain: 1) Tampa Scale for Kinesiophobia (TSK) (Kori et al., 1990), and 2) The Pain Catastrophizing Scale (PCS) (Sullivan et al., 1995). Both questionnaires are valid and reliable (Sullivan et al., 1995; Swinkels-Meewisse et al., 2003), and have been shown to relate to measures of stiffness/stability in previous studies (Karayannis et al., 2013; Ross et al., 2015).

Kinematic measures

Participants were outfitted with two 3-D electromagnetic sensors (trakSTAR, Ascension Technology Corporation, Shelburne, VT, USA), placed on the T_{12} and S_1 spinous processes. Sensors were attached using tape. Data were collected at 240 Hz using custom LabView software (National Instruments, Austin, TX, USA).

Procedure

After completion of the questionnaires, an anesthesiologist marked the participant's interspinous ligament between L_4 and L_5 using landmarks identified with a portable ultrasound

device (Vivid i, General Electric Healthcare, Little Chalfont, United Kingdom). The participants then completed the experimental task for three trials (baseline, injection/pain, and recovery). The experimental task required participants to complete 35 cycles of a repetitive spine flexion/extension movement with the position of the pelvis constrained by a belt to a board (Graham et al., 2014). Participants moved between an upright and a flexed position with movement range guided by pressing target buttons placed in front of the participant in the midline at arms' length at shoulder height to mark the upright position, and a second target located 50 cm anterior to the knee in the sagittal midline to mark the end of flexion range (Graham et al., 2014; Granata and England, 2006; Granata and Gottipati, 2008; Ross et al., 2015).

Upon completion of the baseline trial, the anesthesiologist injected 0.2 mL of hypertonic saline (5% NaCl) solution into the L_4/L_5 interspinous ligament of the participant in sitting (Fig 1) (Tsao et al., 2010). The expected pain responses were confirmed in pilot testing (see Supplementary Material) (Tsao et al., 2010). Immediately after the injection, participants were laid down on their side for one minute in case of fainting. Participants then stood up and completed the "injection/pain" trial. Participants then sat for one hour to allow for the pain levels to return to baseline and then completed the "recovery" trial. Immediately before and after each trial, as well as after the injection, participants rated their pain on a 100-mm visual analog scale (VAS) (Scott and Huskisson, 1976).

(Figure 1 approximately here)

Data Processing and Analysis

All data were processed using custom Matlab code (The Mathworks, Natick, MA, USA). Lumbar spine angles were calculated using 3-D Euler rotation matrices recorded from the T_{12} sensor with respect to the S_1 sensor (Graham et al., 2014; Ross et al., 2015), and the first five cycles were excluded to allow individuals to reach a steady state of movement (Graham et al., 2012a, 2012b; Graham and Brown, 2012). Data were not filtered due to problems associated with filtering nonlinear signals (Bruijn et al., 2009a; Dingwell and Marin, 2006; Kantz and Schreiber, 2004; Mees and Judd, 1993).

The neuromuscular control of spine movements (i.e. local dynamic stability) was determined using the maximum finite-time Lyapunov exponent, λ_{max}. The angular data from each of the trials were time normalized to 28,000 points (30 cycles*240 Hz*4 s/cycle) to account for the effects of time series length on λ_{max} (Bruijn et al., 2009a). Analyses were performed only on the Root-Mean-Square (RMS) of the three spine angles, which has also been referred to as the Euclidean norm (Gates and Dingwell, 2009; Granata and England, 2006). The RMS of the three spine angles was positively shifted upwards so they did not cross zero based on pilot work that revealed this to be the best method to maintain the original individual characteristics of each angular displacement (Beaudette et al., 2016). Data were then delay embedded to improve state space reconstruction as per the following equation:

$$Y(t) = [r(t), r(t + T_d), r(t + 2T_d), \ldots r(t + (n-1)T_d)], \quad (1)$$

where *Y(t)* is the *n*-dimensional state-space, *r(t)* is the original RMS time series data, *n* is the number of reconstruction dimensions, and T_d is a constant time delay (Abarbanel et al., 1993). A 6-D state space was chosen based on previous research (Kennel et al., 1992) and a time delay of 10% of mean period was used (Graham et al., 2014; Granata and England, 2006). Nearest

neighbors were then located, and the exponential rate of divergence between the neighbors was tracked over the course of one cycle. λ_{max} was then determined as the slope of the linear best-fit line calculated by:

$$y(i) = \frac{1}{\Delta t} < \ln d_j(i) > , \quad (2)$$

where $< \ln d_j(i) >$ represents the average logarithm of divergence, dj(i), for all pairs of nearest neighbors, j, throughout a certain number of time steps (iΔt) (Rosenstein et al., 1993). The slope was calculated from 0 to 480 samples (approximately 0–0.5 cycles) (Bruijn et al., 2009b). There is a negative relationship between λ_{max} and control/stability, where a larger λ_{max} indicates faster kinematic divergence, and thus less control/stability (Dingwell and Cusumano, 2000).

Statistical Analysis

All statistical analyses were performed using SPSS 23.0 (IBM Corporation, Armonk, NY, USA). Differences in VAS pain responses between the experimental conditions (baseline vs. injection/pain vs. recovery) and between times (pre- vs. post-trial) was assessed with a two-way repeated-measures (R-M) analysis of variance (ANOVA). Differences in control (maximum finite-time Lyapunov exponents; λ_{max}) between experimental conditions were assessed with a one-way R-M ANOVA. To investigate any potential moderating effects of pain psychology (PCS and TSK scores) and/or demographics (age, height, weight, and sex) on control responses to pain, these variables were added as covariates into the R-M ANOVA. Post-hoc tests (with Sidak corrections) examining significant differences between experimental pain conditions were undertaken when the main effect was significant at $p<0.05$. Last, binary logistic regression was applied to assess the predictive effects of these same variables on determining whether a participant would tighten (“stabilizer”) or loosen (“destabilizer”) their control in response to the

injection/pain. A 10-fold cross-validation was repeated five times to ensure that the results were robust and the model was valid. This was complemented by comparing each variable between stabilizers and destabilizers using one-way ANOVA.

RESULTS

As expected, there was a significant main effect of condition (baseline vs. injection/pain vs. recovery) on the VAS pain responses ($p<0.001$). Injection caused significantly higher levels of pain than either the baseline or recovery conditions ($p<0.001$), which were statistically similar ($p=0.943$) (Fig 2). There was also a significant ($p<0.001$) interaction between condition and time (pre- vs. post-trial), where discomfort increased throughout the 35 cycles during the baseline and recovery trials but decreased during the injection/pain trial (Fig 2A). At baseline, all participants but one had extremely low levels of discomfort (less than 10/100); one participant reported a baseline VAS score of 23/100 (Fig 2B). Furthermore, almost all participants experienced increased discomfort during the baseline and recovery trials, whereas the pre/post-injection VAS responses were more variable (Fig 2B).

(Figure 2 approximately here)

When the whole group was included in the statistical analysis without any covariates, control (λ_{max}) was not different between conditions (main effect: $p=0.564$) (Fig 3). However, after adding PCS scores as a covariate into the ANOVA, the main effect of condition on control became significant ($p= 0.044$), and there was a significant condition x PCS score interaction ($p= 0.048$). No other covariates significantly moderated the effect of condition on control ($p>0.05$).

(Figure 3 approximately here)

Because of the significant moderating effect of pain catastrophizing on the effect of condition on control, we further explored our data. We discovered that there were two distinct/categorical responses to the injection/pain based on the movement trajectories; individuals who tightened control during pain (lower λ_{max} – "stabilizers") (n=6) and those who

loosened control during pain (higher λ_{max} – "destabilizers") (n=10). In both groups, the modified movement strategies returned to their baseline strategy after pain recovered (Fig 3). To further explore this finding, we completed binary logistic regressions using PCS scores (cut score was equal to 0.5, which equaled a PCS >17) to predict the categorical outcome in response to pain (0= "stabilize", 1= "destabilize"). This analysis successfully predicted group membership in 87.5% of cases (5/6 stabilizers, and 9/10 destabilizers) (Table 2), each of the five times. As a follow-up, we compared PCS and TSK between the two responses using one-way ANOVAs. This analysis found PCS (p=0.004) and TSK (p=0.049) scores were significantly higher in the stabilizing group compared to the destabilizing group (Table 3); participants who stabilized have higher PCS and TSK. Neither demographics (i.e. age, height, weight, sex) nor pain intensity (i.e. VAS scores) was significantly different between stabilizers and destabilizers.

(Table 2 & 3 approximately here)

DISCUSSION

The purposes of this work were: 1) to use noxious stimulation to induce LBP in healthy individuals to assess the effects of noxious pain on control, and 2) to assess whether the relationship between pain and control is moderated by psychological features. It was hypothesized that control strategies would change in the presence of pain. It was also hypothesized that there would be a relationship between changes in control and pain catastrophizing.

There was no overall significant difference in spinal control (i.e. λ_{max}) during the injection/pain trial compared to baseline and recovery; therefore, our hypothesis was rejected. However, after further exploring the data, it was found that there were two different reactions to the pain. Compared to baseline/recovery, stabilizers (n =6) had a significant tightening of control (decreased λ_{max}) while the destabilizers (n = 10) had a significant loosening of control (increased λ_{max}). There were no significant differences between VAS scores between the stabilizers and destabilizers, suggesting that individual changes in control strategies were independent of pain experienced but rather related to how threatening they perceived their experienced pain.

In a recent study examining the effects of noxiously induced pain and gait speed on local dynamic stability, there were differential group reactions to either stabilize or destabilize in response to the pain based on gait speed (van den Hoorn et al., 2015). At 0.94 m/s, gait was less stable during LBP when compared to no pain. Conversely, it was found that when walking at 1.67 m/s, gait stability was significantly improved during LBP when compared to no calf pain or no pain (van den Hoorn et al., 2015). The opposite effects of LBP on stability between speeds were suggested to be a protective strategy at higher speeds. This links to the current work where

those with higher pain catastrophizing (PC) scores tended to tighten their control (i.e. stabilize), likely as a protective mechanism for the spine.

Contrary to our previous work (Ross et al., 2015), which showed that higher PC was related to higher levels of experienced pain and reduced control and spine rotational stiffness, the current work showed that PC differentially moderates the change in control in response to the pain (i.e. there were both stabilizers and destabilizers). One potential reason for the differences observed between studies is that capsaicin and the injection may have had different effects on proprioception. If proprioception is poor, there will be more variable movement, leading to a decrease in spinal stability. Another potential reason for the different effects may be due to the different type of pain experienced by the capsaicin and the hypertonic saline injection. Participants described the pain experienced by the capsaicin as a superficial burning sensation, whereas participants described the pain experienced by the saline to be a deep aching pain with some experiencing radiating pain in the legs. A further difference is that pain got worse with movement during the capsaicin trial (similar to the baseline and recovery trials here); however, in the present work pain was relieved with movement in many individuals. Since the pain experienced by the capsaicin was superficial and not alleviated by movement, it could have been more distracting; thus leading to more variable movement and loosened control.

Lastly, in our previous work (Ross et al., 2015), we showed that there was lower control and spine rotational stiffness across all trials (baseline, in pain/capsaicin, and recovery) in individuals with a higher PCS score. However, in the current study, control was the same for both the stabilizers and the destabilizers for the baseline and recovery trial. Differences were only detected in the injection/pain trial and were independent of pain levels. This is an important finding, as it appears to show that individuals' beliefs and attitudes towards pain can lead them to

perceive a given amount of pain as more threatening, thus leading to individual-specific motor behaviors. Moreover, previous research has found that individuals have individualized motor responses to stabilize the spine (Hodges et al., 2013), which agrees with the results observed here. Higher long-term local dynamic stability in LBP patients was also observed in recent work (Asgari et al., 2015).

The stabilizers in the current work had significantly higher PCS and TSK scores than the destabilizers, suggesting that stabilizers have a greater tendency to catastrophize about pain (i.e. have a greater negative orientation of pain) and are more fearful of (re)injury. The fear-avoidance model suggests kinesiophobia and PC are a spectrum with confrontation and fear-avoidance on either extreme and individuals fall somewhere along the continuum based on their fear of pain (Lethem et al., 1983; Rose et al., 1992). Previous research has found that greater PC and kinesiophobia is associated with lower performance (Vlaeyen et al., 1995) independent of pain (Crombez et al., 1999, 1998) and greater trunk stiffness (Karayannis et al., 2013). Using linear regression, the TSK was previously found to be the best predictor of performance (Crombez et al., 1999). In the present work, although both the TSK and PCS scores were significantly higher for the stabilizers than the destabilizers, the PCS was a better predictor of stabilizing than the TSK. The reason the PCS was a better predictor than the TSK could be because the participants were all healthy individuals with no previous history of LBP, whereas the previous studies used individuals with chronic LBP. In our previous study, where healthy individuals were induced with LBP via capsaicin, the PCS was significantly correlated with spine control across all conditions, whereas the TSK was not (Ross et al., 2015).

There are several possible interpretations of the two different responses to noxious stimulus observed in this study. From one perspective, the adaptations could be considered to be

purposeful adapatations to deal with the noxious input. From this perspective, tightened control may represent a protective strategy to reduce movement of the part and reduce the potential for error (Hodges et al., 2013), and loosened control may represent an alternative startegy to increase variation to enable the search for a new less painful solution (Moseley and Hodges, 2006). This interpretation appears to align with the relationship with psychological features with the high catastrophizing group selecting the protective stabilizing solution and the low catastrophising group selecting the destabilizing solution. Although this is logical and intuitive, the data could be explained by alternative mechainsms. For instance, the detsabilizing adaptation may be explained by interference with movement control by nociceptive input; secondary to compromised proprioceptive input from muscle (Matre et al., 2002) or inhibition of muscle contraction (Tsao et al., 2008). Stabilizing adaptation may represent augmented muscle activity according to the vicious cycle theory (Roland, 1986). These latter mechanisms are more difficult to reconcile with the psychological profiles but cannot be excluded with the current data.

The results of this study should be interpreted with consideration of several methodological limitations. First, it is important to acknowledge that pain in response to an acute noxious input differs in several respects to clinical pain. Participants expected that experimental pain will be transient, with recovery within a short time frame. Thus, the threat value of this pain will be lower than clinical pain with an unceratin time course. This means that our data will likely underestimate the impact of catastrophising on motor adaptation. Although hypertonic saline injection mimics some features typical of musculoskelatal pain (e.g. deep ache), unlike many clinical conditions, it tends to reduce with movement/muscle contraction (Tsao et al., 2010). Further, unlike clinical pain, the participants have not had the opportunity to live with the pain and only the immediate response to the noxious input can be assessed. Despite these, and

other differences, in many contexts experimental pain has been shown to induce similar motor adaptation to many of those observed in clinical conditions including LBP (Arendt-Nielsen et al., 1995; Hodges et al., 2003). The potential limitation of the experimental pain model needs to be weighed against the benefit of direct comparision of LBP and spinal control without confounding variables typically seen in the LBP population.

Second, a limitation of this study is the use of an electromagnetic tracking unit. Data from electromagnetic tracking units can be distorted due to the interfence of magnetic materials with the electromagnetic field. However, Ascension uses DC magnetic field technology which overcomes many of the meticallic distortion problems of older magnetic technologies, such as AC electromagnetic systems (Anisfield, 2000). In addition, we minimized the liklihood of magentic distortion by placing the source directly next to the participant while they were performing all trials. In addition, the source did not move relative to the particpant between trials or particiapnts. In addition, an error in the placement of the the sensors could affect the results. This error was minimized by using ultrasound to locate the T_{12} and S_1 spinous processes. This risk of error was further minimized by having the subjects wear the sensors throughout the entire duration of the study (even during the rest sessions) and the repeated measures design of the study.

Third, similar to earlier studies (Hodges et al., 2013; Ross et al., 2015), we studied the response of the trunk with the pelvis fixed. The purpose of this method was to restrict the motion to the trunk and minimize the contribution of the lower limbs. Although this renders the task less natural and alters the lifting technique commonly used in the field, it has the benefit of limiting further sources of inter-individual variation, and enables interpretation of the strategy specifcally implemented for trunk control. Future work should explore if similar trends for the same

variables examined in this study are found with an unconstrained pelvis. Fourth, although the sample size is adequate for this nature of study, it is small for logistic regression. However, we were able to repeatedly detect siginficant differences and relationships which indicates that the effects were sufficiently robust to be detected with the sample size. In future, this study should be repeated with a larger sample size to confirm the accuracy of the model. Lastly, although pain returned to baseline levels, based on VAS scores, other control mechanisms may not have returned to baseline and had an effect on spinal control. However, there were no significant differences in λ_{max} between baseline and recovery trials, suggesting that the effects of the injection had fully subsided.

The results of this study might help explain why different responses are observed in various studies looking at the effect of different pain modalities and groups. Even amongst healthy individuals, who subjectively report the same amount of pain, the group may be heterogeneous with respect to pain cognitions with some individuals who catastrophise about pain (negative orientation toward pain) and others who do not. This heterogeneity of pain catastrophizing may lead to different selected motor behaviors (i.e. tighten versus loosen control). Therefore, these results suggest that PC and kinesiophobia should be considered when studying motor behaviors (e.g. spine control and stability) in response to pain (induced or chronic).

ACKNOWLEDGEMENTS

This study was funded by the Natural Sciences and Engineering Research Council of Canada (NSERC) [B. Gurd (402635-2011), R. Graham (RGPIN-2014-05560)].

363

364 **CONFLICT OF INTEREST**

365 The authors have no conflicts of interest to declare.

366 **REFERENCES**

367 Abarbanel, H.D.I., Brown, R., Sidorowich, J.J., Tsimring, L.S., 1993. The analysis of observed
368 chaotic data in physical systems. Rev. Mod. Phys. 65, 1331–1392.

369 Anisfield, N., 2000. Ascension technology puts spotlight on DC field magnetic motion tracking.
370 HP Chron. 7, 3.

371 Arendt-Nielsen, L., Graven-Nielsen, T., Svarrer, H., Svensson, P., 1995. The influence of low
372 back pain on muscle activity and coordination during gait: a clinical and experimental
373 study. Pain 64, 231–240.

374 Asgari, M., Sanjari, M.A., Mokhtarinia, H.R., Moeini Sedeh, S., Khalaf, K., Parnianpour, M.,
375 2015. The effects of movement speed on kinematic variability and dynamic stability of the
376 trunk in healthy individuals and low back pain patients. Clin. Biomech. 30, 682–688.

377 Beaudette, S.M., Howarth, S.J., Graham, R.B., Brown, S.H.M., 2016. On the use of a Euclidean
378 norm function for the estimation of local dynamic stability from 3D kinematics using time-
379 delayed Lyapunov analyses. Med. Eng. Phys. 38, 1139–1145.

380 Brown, M.D., Holmes, D.C., Heiner, A.D., 2002. Measurement of cadaver lumbar spine motion
381 segment stiffness. Spine (Phila. Pa. 1976). 27, 918–922.

382 Brown, S.H.M., McGill, S.M., 2010. The relationship between trunk muscle activation and trunk
383 stiffness: examining a non-constant stiffness gain. Comput. Methods Biomech. Biomed.
384 Engin. 13, 829–835.

385 Bruijn, S.M., van Dieën, J.H., Meijer, O.G., Beek, P.J., 2009a. Statistical precision and
386 sensitivity of measures of dynamic gait stability. J. Neurosci. Methods 178, 327–333.

387 Bruijn, S.M., van Dieën, J.H., Meijer, O.G., Beek, P.J., 2009b. Is slow waking more stable? J.
388 Biomech. 42, 1506–1512.

389 Cholewicki, J., McGill, S.M., 1996. Mechanical stability of the in vivo lumbar spine:
390 implications for injury and chronic low back pain. Clin. Biomech. 11, 1–15.

391 Crombez, G., Vervaet, L., Lysens, R., Baeyens, F., Eelen, P., 1998. Avoidance and confrontation
392 of painful, back-straining movements in chronic back pain patients. Behav. Modifcation 22,
393 62–77.

394 Crombez, G., Vlaeyen, J.W.S., Heuts, P.H.T.G., Lysens, R., 1999. Pain-related fear is more
395 disabling than pain itself : evidence on the role of pain-related fear in chronic back pain
396 disability 80, 329–339.

397 Dancey, E., Murphy, B., Srbely, J.Z., Yielder, P., 2014. The effect of experimental pain on motor
398 training performance and sensorimotor integration. Exp. Brain Res. 232, 2879–2889.

399 Demoulin, C., Crielaard, J.-M., Vanderthommen, M., 2007a. Spinal muscle evaluation in healthy
400 individuals and low-back-pain patients: a literature review. Jt. Bone Spine 74, 9–13.

401 Demoulin, C., Distrée, V., Tomasella, M., Crielaard, J.-M., Vanderthommen, M., 2007b. Lumbar
402 functional instability: a critical appraisal of the literature. Ann. Réadaptation Médecine
403 Phys. 50, 677–684.

404 Dingwell, J.B., Cusumano, J.P., 2000. Nonlinear time series analysis of normal and pathological

405 human walking. Chaos 10, 848–863.

406 Dingwell, J.B., Marin, L.C., 2006. Kinematic variability and local dynamic stability of upper
407 body motions when walking at different speeds. J. Biomech. 39, 444–452.

408 Dubois, J., Piché, M., Cantin, V., Descarreaux, M., 2011. Effect of experimental low back pain
409 on neuromuscular control of the trunk in healthy volunteers and patients with chronic low
410 back pain. J. Electromyogr. Kinesiol. 21, 774–781.

411 Ebenbichler, G.R., Oddsson, L.I.E., Kollmitzer, J., Erim, Z., 2001. Sensory-motor control of the
412 lower back: implications for rehabilitation. Med. Sci. Sport. Exerc. 33, 1889–1898.

413 Gates, D.H., Dingwell, J.B., 2009. Comparison of different state space definitions for local
414 dynamic stability analyses. J. Biomech. 42, 1345–1349.

415 Global Burden of Disease Study 2013 Collaborators, 2015. Global, regional, and national
416 incidence, prevalence, and years lived with disability for 301 acute and chronic diseases and
417 injuries in 188 countries, 1990-2013: a systematic analysis for the Global Burden of Disease
418 Study 2013. Lancet (London, England) 6736, 1990–2013.

419 Graham, R.B., Brown, S.H.M., 2012. A direct comparison of spine rotational stiffness and
420 dynamic spine stability during repetitive lifting tasks. J. Biomech. 45, 1593–1600.

421 Graham, R.B., Oikawa, L.Y., Ross, G.B., 2014. Comparing the local dynamic stability of trunk
422 movements between varsity athletes with and without non-specific low back pain. J.
423 Biomech. 47, 1459–1464.

424 Graham, R.B., Sadler, E.M., Stevenson, J.M., 2012a. Local dynamic stability of trunk
425 movements during the repetitive lifting of loads. Hum. Mov. Sci. 31, 592–603.

426 Graham, R.B., Sheppard, P.S., Almosnino, S., Stevenson, J.M., 2012b. Dynamic spinal stability
427 and kinematic variability across automotive manufacturing work shifts and days. Int. J. Ind.
428 Ergon. 42, 428–434.

429 Granata, K.P., England, S.A., 2006. Stability of dynamic trunk movement. Spine (Phila. Pa.
430 1976). 31, E271–E276.

431 Granata, K.P., Gottipati, P., 2008. Fatigue influences the dynamic stability of the torso.
432 Ergonomics 51, 1258–1271.

433 Hodges, P.W., Coppieters, M.W., MacDonald, D., Cholewicki, J., 2013. New insight into motor
434 adaptation to pain revealed by a combination of modelling and empirical approaches. Eur. J.
435 Pain 17, 1138–46.

436 Hodges, P.W., Moseley, G.L., Gabrielsson, A., Gandevia, S.C., 2003. Experimental muscle pain
437 changes feedforward postural responses of the trunk muscles 262–271.

438 Hung, L., Maracle, E., Srbely, J., Brown, S., 2014. Acute experimentally induced neck pain does
439 not affect fatigability of the peripheral biceps brachii muscle. Motor Control 18, 395–404.

440 Kantz, H., Schreiber, T., 2003. Nonlinear Time Series Analysis, 2nd ed, Nonlinear Time Series
441 Analysis. Cambridge University Press, Cambridge, UK.

442 Karayannis, N. V, Smeets, R.J.E.M., van den Hoorn, W., Hodges, P.W., 2013. Fear of movement
443 is related to trunk stiffness in low back pain. PLoS One 8, e67779.

444 Kennel, M.B., Brown, R., Abarbanel, H.D.I., 1992. Determining embedding dimension for

445 phase-space reconstruction using a geometrical construction. Phys. Rev. A 45, 3403–3411.

446 Kori, S., Miller, R., Todd, D., 1990. Kinesiophobia: a new view of chronic pain behavior. Pain
447 Manag. 3, 35–43.

448 Larivière, C., Gagnon, D., Loisel, P., 2000. The comparison of trunk muscles EMG activation
449 between subjects with and without chronic low back pain during flexion-extension and
450 lateral bending tasks. J. Electromyogr. Kinesiol. 10, 79–91.

451 Lee, H., McAuley, J.H., Hübscher, M., Allen, H.G., Kamper, S.J., Moseley, G.L., 2015.
452 Tweeting back: predicting new cases of back pain with mass social media data. J. Am. Med.
453 Informatics Assoc. In Press, ocv168.

454 Lethem, J., Slade, P.D., Troup, J.D.G., Bentley, G., 1983. Outline of a fear-avoidance model of
455 exaggerated pain perception. I. Behav. Res. Ther. 21, 401–408.

456 Matre, D., Arendt-nielsen, L., Knardahl, S., 2002. Effects of localization and intensity of
457 experimental muscle pain on ankle joint proprioception. Eur. J. pain 6, 245–260.

458 Mees, A.I., Judd, K., 1993. Dangers of geometric filtering. Phys. D Nonlinear Phenom. 68, 427–
459 436.

460 Miller, E.M., Bazrgari, B., Nussbaum, M.A., Madigan, M.L., 2013. Effects of exercise-induced
461 low back pain on intrinsic trunk stiffness and paraspinal muscle reflexes. J. Biomech. 46,
462 801–805.

463 Moseley, G.L., Hodges, P.W., 2006. Reduced variability of postural strategy prevents
464 normalization of motor changes induced by back pain : A risk factor for chronic trouble ?
465 Behav. Neurosci. 120, 474–476.

466 Murray, C.J.L., Lopez, A.D., 2013. Measuring the global burden of disease. N. Engl. J. Med.
467 369, 448–457.

468 Nelson-wong, E., Gregory, D.E., Winter, D.A., Callaghan, J.P., 2008. Gluteus medius muscle
469 activation patterns as a predictor of low back pain during standing 23, 545–553.

470 Potvin, J.R., Brown, S.H.M., 2005. An equation to calculate individual muscle contributions to
471 joint stability. J. Biomech. 38, 973–980.

472 Reeves, N.P., Narendra, K.S., Cholewicki, J., 2007. Spine stability: The six blind men and the
473 elephant. Clin. Biomech. 22, 266–274.

474 Roland, M.O., 1986. Review Paper A critical review of the evidence for a pain-spasm-pain cycle
475 in spinal disorders was palpated. Clin. Biomech. 1, 102–109.

476 Rose, M.J., Klenerman, L., Atchison, L., Slade, P.D., 1992. An application of the fear avoidance
477 model to three chronic pain problems. Behav. Res. Ther. 30, 359–65.

478 Rosenstein, M.T., Collins, J.J., De Luca, C.J., 1993. A practical method for calculating largest
479 Lyapunov exponents from small data sets. Phys. D Nonlinear Phenom. 65, 117–134.

480 Ross, G.B., Mavor, M.P., Brown, S.H.M., Graham, R.B., 2015. The effects of experimentally
481 induced low back pain on spine rotational stiffness and local dynamic stability. Ann.
482 Biomed. Eng. 43, 2120–2130.

483 Scott, J., Huskisson, E.C., 1976. Graphic representation of pain. Pain 2, 175–184.

484 Sullivan, M.J.L., Bishop, S.R., Pivik, J., 1995. The Pain Catastrophizing Scale : Development

485 and validation 7, 524–532.

486 Swinkels-Meewisse, E.J.C.M., Swinkels, R.A.H.M., Verbeek, A.L.M., Vlaeyen, J.W.S.,
487 Oostendorp, R.A.B., 2003. Psychometric properties of the Tampa Scale for kinesiophobia
488 and the fear-avoidance beliefs questionnaire in acute low back pain. Man. Ther. 8, 29–36.

489 Tsao, H., Galea, M.P., Hodges, P.W., 2008. Concurrent excitation of the opposite motor cortex
490 during transcranial magnetic stimulation to activate the abdominal muscles. J. Neurosci.
491 Methods 171, 132–139.

492 Tsao, H., Tucker, K.J., Coppieters, M.W., Hodges, P.W., 2010. Experimentally induced low
493 back pain from hypertonic saline injections into lumbar interspinous ligament and erector
494 spinae muscle. Pain 150, 167–172.

495 van den Hoorn, W., Hug, F., Hodges, P.W., Bruijn, S.M., Dieën, J.H. Van, 2015. Effects of
496 noxious stimulation to the back or calf muscles on gait stability. J. Biomech. 48, 4109–
497 4115.

498 van Dieën, J.H., Cholewicki, J., Radebold, A., 2003. Trunk muscle recruitment patterns in
499 patients with low back pain enhance the stability of the lumbar spine. Spine (Phila. Pa.
500 1976). 28, 834–841.

501 Vlaeyen, J.W.S., Kole-Snijders, A.M.J., Boeren, R.G.B., van Eek, H., 1995. Fear of
502 movement/(re)injury in chronic low back pain and its relation to behavioral performance.
503 Pain 62, 363–372.

504 Willigenburg, N.W., Kingma, I., Hoozemans, M.J.M., van Dieën, J.H., 2013. Precision control
505 of trunk movement in low back pain patients. Hum. Mov. Sci. 32, 228–239.

506 Zedka, M., Prochazka, a, Knight, B., Gillard, D., Gauthier, M., 1999. Voluntary and reflex
507 control of human back muscles during induced pain. J. Physiol. 520 Pt 2, 591–604.

508

Figure Captions

Figure 1 – Injection of hypertonic saline into the L_4/L_5 interspinous ligament with ultrasound imaging.

Figure 2 – A) Mean visual analog scale (VAS) pain responses directly prior-to (pre) and after (post) the baseline (B), injection/pain (I), and recovery (R) trials. B) Individual participant VAS pain responses for these same variables. Red lines show individual responses across each trial, whereas black lines link these responses to the mean response during each trial. For A and B, the red dotted line refers to the average VAS pain response immediately after the injection across all participants.

Figure 3 – Mean maximum finite-time Lyapunov exponents (λ_{max}). Individual responses for baseline, injection/pain, and recovery trials. Destabilizing responses are highlighted in black and stabilizing responses are highlighted in red.

Demographic	Male	Female	All
Age (yrs)	20.75 (2.43)	21 (2.61)	20.88 (2.44)
Height (cm)	176.69 (8.90)	162.88 (3.09)	169.78 (9.61)
Mass (kg)	74.38 (11.15)	62.56 (9.02)	68.57 (11.54)
PCS /52	15.37 (10.27)	15.75 (8.96)	15.56 (9.31)
TSK /68	34.13 (6.66)	33.50 (4.14)	33.81 (5.37)

PCS = The Pain Catastrophizing Scale, TSK = Tampa Scale for Kinesiophobia

523 Table 1. Participant mean (standard deviation) demographics and kinesiophobia scores.

524 Table 2. Binary logistic regression variables, when a cut value of .500 was used.[a]
525

		B	S.E.	Wald
Step 1	PCS	-.321	.170	3.570
	Constant	5.649	2.837	3.964

526 B = coefficients, S.E. = standard error.

527 [a]Note that 87.5% of participants could be correctly classified using only PCS scores (5/6
528 stabilizers = 83.3%, 9/10 destabilizers = 90%).

529 Table 3. Comparison of mean (standard deviation) demographics, pain catastrophizing (PCS)
530 and kinesiophobia (TSK) scores, maximum finite-time Lyapunov exponents (λ_{max}), and VAS
531 pain scores between stabilizers and destabilizers.
532

	Stabilizer	Destabilizer	p-value
n	6	10	-
Age	21.17 (2.22)	20.70 (2.67)	0.725
Height	172.08 (9.52)	168.4 (9.89)	0.477
Weight	72.83 (10.19)	65.85 (12.00)	0.255
Baseline λ_{max}	2.09 (.301)	2.01 (.218)	0.526
Injection λ_{max}	1.87 (.327)	2.24 (.159)	0.008*
Recovery λ_{max}	2.06 (.401)	2.12 (.152)	0.644
PCS	23.50 (9.05)	10.80 (5.63)	0.004*
TSK	37.17 (4.26)	31.80 (5.10)	0.049*
VAS Pre-Baseline	2.33 (3.14)	4.60 (6.82)	0.460
VAS Post-Baseline	20.33 (15.21)	15.10 (13.68)	0.489
VAS Pre-Injection	42.50 (15.66)	48.90 (20.27)	0.519
VAS Post-Injection	35.83 (22.58)	34.90 (19.56)	0.932
VAS Pre-Recovery	2.50 (2.43)	4.40 (6.24)	0.492
VAS Post-Recovery	20.00 (20.31)	12.20 (6.93)	0.277

533 * = significant difference between groups at $p < 0.05$.