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Trunk stiffness decreases and trunk damping increases with experimental low back pain

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ABSTRACT

Movement adaptations to low back pain (LBP) are believed to protect the painful area. Increased trunk stiffness and decreased trunk damping have been shown in people with recurrent LBP. However, no study has examined these properties using external force perturbations to the trunk during acute LBP when protective adaptations might be expected to have most relevance.

Adaptations to an acute painful stimulus via unilateral injection of hypertonic saline into the right longissimus muscle were assessed using a trunk force perturbation paradigm and a mass-spring-damper model to describe effective trunk dynamical properties. Equal weights (15% body weight) were connected to the front and back of the trunk via a cable. Either one was dropped at random to perturb the trunk. Effective trunk dynamical properties were estimated in fourteen males (mean (standard deviation) age 25 (6) years) assuming that trunk movement can be modelled as a second order linear system. Effective trunk dynamical properties were compared before, during and after the experimentally induced painful period.

Estimates of effective trunk stiffness (K) decreased and damping (B) increased during pain compared to both before ([mean contrast, 95% CI] K: −403 [−651 to −155] Nm^{-1}, B: 28 [9–50] Nms^{-1}) and after (K: −324 [−58 to −591] Nm^{-1}, B: 20 [4–33] Nms^{-1}) the experimentally induced painful period. We interpret our results to show that, when challenged by a step force perturbation, a healthy system adapts to noxious input by controlling trunk velocity rather than trunk displacement, in contrast to observations during remission from recurrent clinical LBP.

1. Introduction

Many movement and muscle activation strategies adopted during low back pain (LBP) are considered protective with the goal to limit provocation of pain or damage/re-injury to the spine and surrounding tissues (Hodges and Tucker, 2011; van Dieën et al., 2003ab). These adaptations can alter the dynamical properties of the trunk including stiffness and damping (Freddolini et al., 2014; Gildea et al., 2015; Hodges et al., 2009; van den Hoorn et al., 2012). For instance, people with chronic LBP showed greater trunk muscle activity, which corresponded with greater trunk stiffness compared to individuals without LBP (van Dieën et al., 2003a, b). Greater trunk stiffness was also observed in individuals with sub-acute LBP compared to individuals without LBP (Freddolini et al., 2014). Further, individuals with recurring episodes of LBP, who were in remission at the time of testing (i.e., pain-free), displayed higher trunk stiffness (Hodges et al., 2009) but lower trunk damping (Gildea et al., 2015; Hodges et al., 2009) than pain-free controls. No study has examined these properties during acute LBP when protective adaptations might be expected to have most relevance.

Adaptations to pain depend on the task (Hodges and Tucker, 2011). Current pain adaptation theory (Hodges and Tucker, 2011; Lund et al., 1991) suggest that a common protective adaptation would be to limit movement during pain. With consideration of a force perturbation to the trunk, such adaptation to pain would be expected to increase trunk stiffness. In this context, trunk damping might decrease as has been observed in several studies of recurrent LBP (Gildea et al., 2015; Hodges et al., 2009).

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Despite the notion that increased trunk stiffness and decreased damping during LBP are adaptive mechanisms that can protect the affected area, no study has measured these properties together during acute LBP. Clarification of whether this strategy is employed in response to the onset of back pain/injury (i.e., when the spine/trunk might be expected to derive greatest potential benefit from protection) is critical. This is difficult to assess because it is not possible to measure these properties before and after onset of clinical pain/injury, and there is considerable variation in the manifestations of clinical acute LBP (e.g., variation in local/tissue, central nervous system, and psychological changes), which are likely to influence trunk dynamics (Karayannis et al., 2013). One way to gain insight into the impact of pain on trunk dynamics without these issues is to compare the dynamics before and during the induction of experimental pain in healthy individuals. Using a step force-input response method established previously (Hodges et al., 2009), this study aimed to test whether trunk stiffness is increased and trunk damping is decreased in response to experimental pain induced in the lower back. To limit the invasiveness of the experimental pain protocol, we opted for unilateral injection of hypertonic saline into the right longissimus muscle. We acknowledge that unilateral experimental pain might cause movement adaptations that are not captured by the sagittal nature of the force perturbations to the trunk.

2. Methods

2.1. Participants

Seventeen male participants volunteered for the study. They were recruited from the local University community and by word of mouth. Data for three participants were excluded from further analysis due to technical issues with the equipment. Of the remaining 14 participants, the mean (SD) age was 25 (4) years, with a height and weight of 176 (7) cm and 72 (11) kg, respectively. Exclusion criteria included a history of LBP that limited function or required medical/health intervention, major pain/injury in other body regions, or any major neurological or respiratory conditions. Ethical clearance was obtained by The Institutional Medical Research Ethics Committee. All participants provided informed consent and procedures were conducted in accordance with the Declaration of Helsinki. Some data from concurrent experiments involving these participants have been reported previously (Hodges et al., 2013).

2.2. Procedure

For a separate experiment, participants were prepared for surface electromyographic recordings of trunk muscles [for more detail see (Hodges et al., 2013)] and indwelling fine wire electrodes were inserted into the deep and superficial multifidus muscles at the level of the fourth lumbar vertebrae and transversus abdominus muscle.

Participants sat in a semi-seated upright position on a slanted surface such that their body weight was supported through their buttocks (~50%) and knees (Fig. 1). As shown in Fig. 1, the pelvis was fixed by padded supports at the back and front of the pelvis at the level of the posterior and anterior superior iliac spines, respectively (Cholewicki et al., 2000; Hodges et al., 2009). A harness made of two padded aluminium profiles was tightly fitted (using bolts with wing nuts) around the thorax. Weights (15% of body weight) were connected at the front and back via steel cables that were aligned horizontally, at the approximate level of the trunk's centre of mass (T9). Cables passed through low-friction pulleys to weights attached by an electromagnet (GMXH065X20A01, Magnet Schultz Ltd., Surrey, UK). As equal weights were connected to each side, minimal trunk muscle activity was required to maintain upright posture and participants could move backwards and forwards with minimal resistance. Strain gauges (GK 2126, Gedge Systems, Melbourne, Australia) positioned between each cable and electromagnet were used to measure the force acting on the trunk.

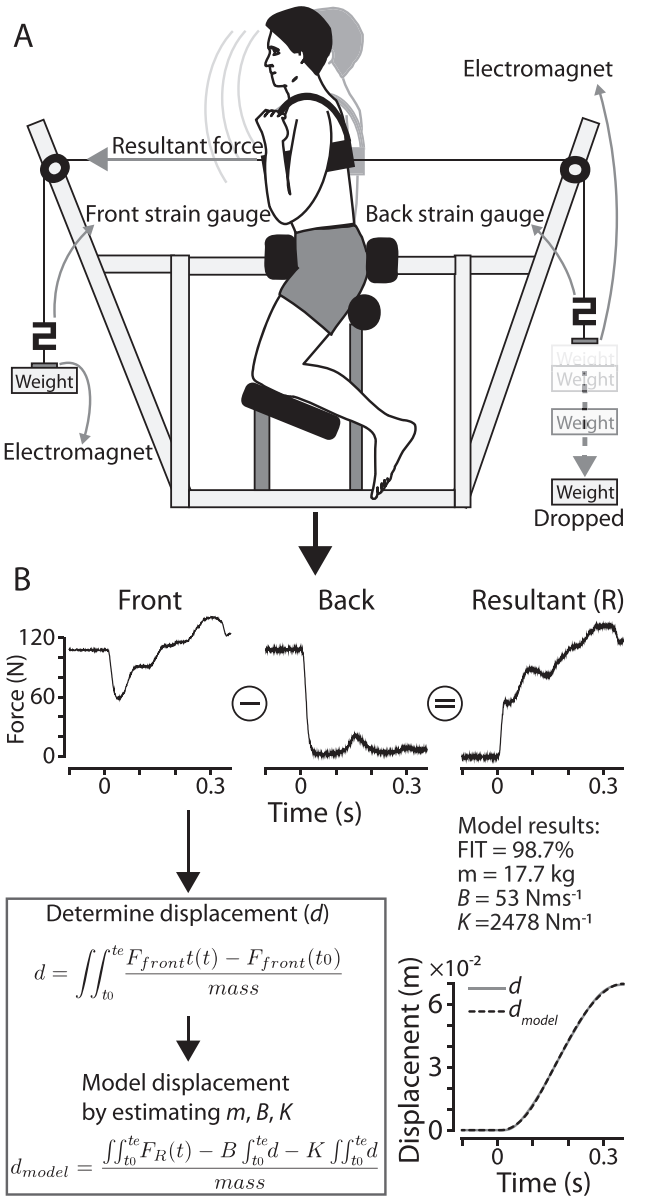

Fig. 1. Description of force-input response method. Participants sat in a semi-upright position with their pelvis fixated (A) (Figure adapted from Hodges et al., 2009). At random intervals, either the front or back weight was dropped. The participant did not know when or which weight was going to be dropped. A data processing example of a back-weight drop is provided (B). In this case, trunk displacement was determined from the front strain gauge. Linear second order model parameters *m* (mass), *B* (damping), *K* (stiffness) were estimated by minimising the error between the measured and modelled trunk displacement from the onset of the weight drop (*t*0) till maximum displacement of the trunk (*te*).

Participants were instructed to sit upright in a relaxed manner. At a random time, a weight from either the front or back was released by disengaging the respective electromagnet, resulting in a backward or forward perturbation to the trunk, respectively. Participants were instructed to return to an upright position after the perturbation. The weight was dropped 10 times in either

direction (front/back), in random order, and reconnected after ~5 s. This procedure was performed under three conditions: (1) before pain, (2) during pain, and (3) after pain.

Pain was induced by unilateral injection of a 1.5 mL bolus of hypertonic saline (5% concentration) into the right longissimus muscle, ~5 cm lateral to the fourth lumbar spinous process (Fig. 2). Participants reported their pain intensity using an 11-point numeric rating scale (NRS) anchored with 'no pain' at 0 and 'worst pain imaginable' at 10. Trials in the pain condition commenced when reported pain reached 4/10, ~1 min after the saline injection. The *after pain* condition was performed ~10 min after participants reported 0 on the NRS.

Strain gauge data were amplified (WT127, APCS, Seven Hills, Australia) and then digitized with 16-bit precision at 2000 samples/s with a Power 1401 Data acquisition system, using Signal software (Cambridge Electronic Design Ltd., Cambridge, UK).

2.3. Data analysis

Strain gauge data were exported to Matlab (Mathworks, Natick, MA, USA) then transformed to Newtons using calibration values. These data were used to determine force and trunk kinematics. It was assumed that trunk kinematics in response to the force perturbation followed a linear second order system (Eq. (1)).

$$F(t) = m\ddot{x}(t) + B\dot{x}(t) + Kx(t) \quad (1)$$

where m is the effective trunk mass, B is effective trunk damping and K is effective trunk stiffness. $F(t)$ is the resultant force on the trunk, determined by subtracting the back from the front force, and $\ddot{x}(t)$, $\dot{x}(t)$, and $x(t)$ are the horizontal trunk acceleration, velocity, and displacement over time from the onset of the weight drop until the maximum displacement of the trunk. Trunk acceleration was derived from the strain gauge connected to the weight that was not released by dividing measured force by the connected mass (mass + mass of the electromagnet). Trunk velocity and displacement were derived by numerically integrating acceleration once, and twice over time, respectively (Fig. 1). Estimation of mass was allowed to vary as trunk mass that actually moves in response to the weight drop could change between conditions, i.e., estimated mass reflects the effective trunk mass (Hodges et al., 2009). A least squares procedure (lsqnonlin.m trust-region-reflective algorithm using the function described in Eq. (2)) was used to estimate the m, B, and K for each individual weight drop. The initial values of m, B, and K were set to 30 kg, 500 Nsm^{-1}, and 2000 Nm^{-1}, and no other boundaries were set. To increase the robustness of this procedure, data on the left and right side of Eq. (1) were integrated twice over time (Eq. (2)) (Tsuji et al., 1995).

$$\iint_{t0}^{te} F(t)dt^2 = mx(t) + B\int_{to}^{te} x(t)dt + K\iint_{t0}^{te} x(t)dt^2 \quad (2)$$

Where $t0$ reflects the onset of the weight drop, and te reflects the maximum trunk displacement time points. Modelled and actual trunk displacement were compared using percentage FIT (Eq. (3)).

$$FIT = 100\left(\frac{\|x - \hat{x}\|}{\|x - \bar{x}\|}\right) \quad (3)$$

where x and $\hat{x}$ are the observed and modelled trunk displacement, respectively, and $\bar{x}$ reflects the mean of the observed trunk displacement. For each participant, the average of the mass, B, and K estimates with a model FIT better than 85% were determined across all 10 back or 10 front weight drops at each experimental condition.

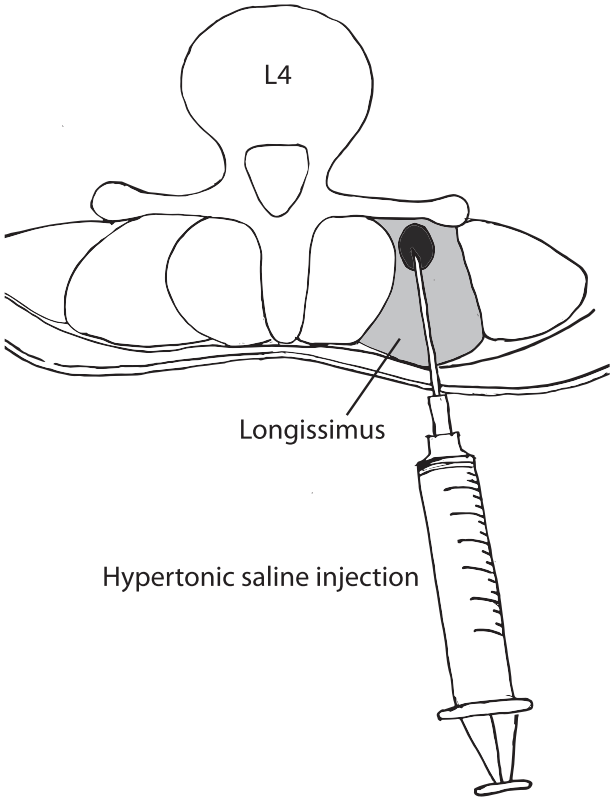

Fig. 2. Experimental pain. Pain was induced by injection of a 1.5 mL bolus of hypertonic saline (5% concentration) into the right longissimus muscle, Figure adapted from Hodges et al. (2013).

2.4. Statistical analysis

Trunk parameters (m, B, K, max displacement and duration) were compared between *conditions* (before, during, and after pain) and perturbation *directions* (forward and backward) using linear mixed models. Condition and direction and their interaction were entered as fixed effects and the intercepts of participants were entered as random effects into the model. The distribution of the standardised residuals was assessed using Shapiro Wilkinson test of normality for each model. If model residuals were not normally distributed, data were transformed (i.e., duration values were inverted, damping values were log transformed). Residuals of final models were normally distributed (all, $P > 0.080$). Standard error of estimates was determined robustly (Colin Cameron, Miller, 2015), and P-values were obtained using the maximum likelihood method. Non-significant interactions were removed from the model. Coefficients and their 95% confidence intervals (CI) comparing pain and after pain conditions versus the before pain condition, and after pain versus the pain condition, were extracted from the model using Bonferroni correction for multiple comparisons. Corrected P-values are reported. Statistics were performed using Stata (version 14, StataCorp LP, College Station, TX, USA). Significance was set at $P < 0.05$.

3. Results

As no significant Condition × Direction interactions were found for any outcome parameters ($P > 0.88$), all interactions were removed from the linear mixed models. Hence, although some differences between directions were identified, all effects of condition were independent of the direction of the perturbation.

3.1. Pain intensity

Participants reported a peak pain intensity of 6.1 (2.7) out of 10 (NRS) after injection of hypertonic saline into longissimus. All trials during the pain condition were completed before pain fell below 4 out of 10.

3.2. Trunk stiffness and damping modelled as a linear second order model

Overall mean (SD) model FIT was 97% (1.5%). One trial was removed due to a FIT value below 85%. Effective trunk mass (15.0 kg, 95% CI: 13.6–16.5) was not significantly affected by condition (P = 0.054), but its estimate was on average 3.2 kg greater during forward compared to backward perturbation (P < 0.001).

3.3. Effect of experimental pain on trunk stiffness and damping

Trunk stiffness was affected by condition (main effect; P < 0.001) but was not significantly different for direction (main effect; P = 0.092). Stiffness was lower during pain than both the before-pain (contrast (95% CI): −403 [−651 to −155] Nm^{-1}, post hoc: P < 0.001) and after pain (contrast (95% CI): −324 [−58 to −591] Nm^{-1}, post hoc: P = 0.011, Fig. 3) conditions. Trunk damping was also affected by condition (main effect; P < 0.001) but not different between directions (main effect; P = 0.360). In contrast to stiffness, damping was higher during pain than both the before-pain (contrast (95% CI): 28 [9–50] Nms^{-1}, post hoc: P = 0.001) and after pain (contrast (95% CI): 20 [4–33] Nms^{-1}, post hoc: P = 0.012, Fig. 3) conditions. No differences were found in stiffness or damping estimates between the before-pain and after pain conditions (Damping, contrast (95% CI): 8 [−15 to 38] Nsm^{-1}, post hoc: P = 1; Stiffness, contrast (95% CI) −79 [−518 to 359] Nm^{-1}, post hoc: P = 1).

3.4. Effect of experimental pain on trunk displacement and duration of displacement

Trunk displacement was affected by condition (main effect; P < 0.001) and direction (main effect; P = 0.001, forward > backward). Displacement was greater during pain compared to both before-pain (contrast (95% CI): 0.006 [0.002–0.010] m, post hoc: P < 0.001) and after pain (contrast (95% CI): 0.005 [0.001–0.008] m, post hoc: P = 0.011, Fig. 3). Trunk displacement

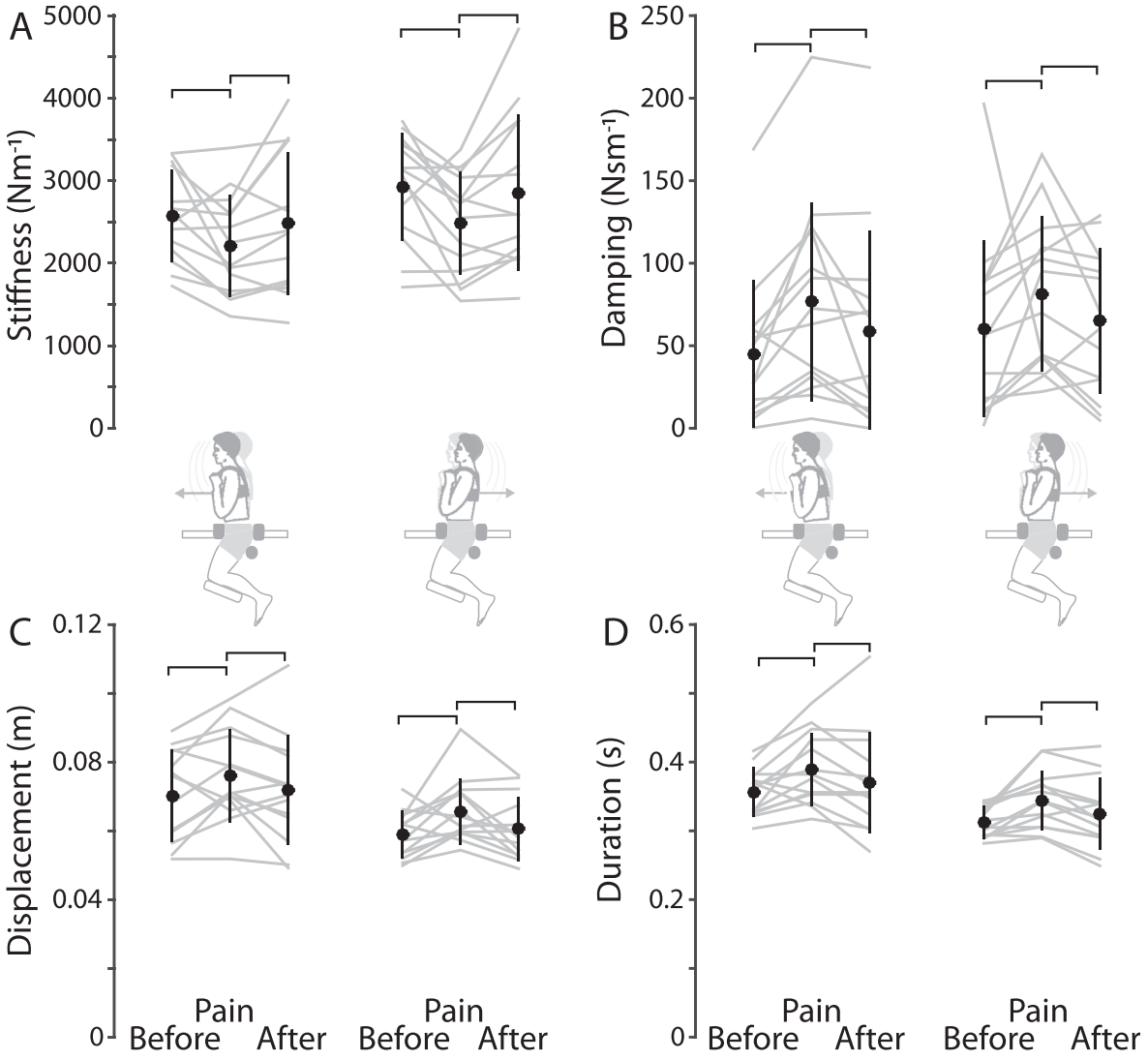

Fig. 3. Results of second order linear model fit. Findings are shown for trunk stiffness (A), damping (B), displacement (C), and duration (D) in response to a back or front weight drop (shown by pictograms in between the top and bottom row) before, during, and after pain conditions. Square brackets indicate significant difference between conditions. Group mean (black circles), the standard error bars (black), and individual data are shown (light grey).

after pain was not significantly different from before-pain (contrast (95% CI): −0.005 [−0.008 to −0.001 m, post hoc: $P = 1$).

The duration of trunk displacement was affected by condition (main effect; $P = 0.001$) and direction (main effect: $P < 0.001$, forward > backward). Duration was greater during pain compared to both before-pain (contrast (95% CI): 0.026 [0.013–0.040] s, post hoc: $P < 0.001$) and after pain (contrast (95% CI): 0.020 [0.007–0.033] s, post hoc: $P = 0.001$, Fig. 3). Duration of the trunk displacement after pain was not significantly different from before-pain (contrast (95% CI): 0.005 [−0.016 to 0.030] s, post hoc: $P = 1$).

4. Discussion

The main aim of this study was to assess the effect of acute experimental pain on trunk damping and stiffness that was estimated in response to a weight drop perturbation to the trunk. In contrast to our hypothesis, results show that during the transient exposure to acute experimental pain in individuals with no history of LBP, effective trunk stiffness did not increase as a protective response to limit movement. Rather, the adaptation resulted in further and longer duration of trunk displacements in response to the same perturbation due to *decreased* effective stiffness and *increased* effective damping. Although decreased stiffness and increased movement amplitude during experimental pain might be interpreted simply to contradict the predicted *protective* outcome from the adaptation, decreasing stiffness and increasing damping may have other benefits related to movement control.

4.1. Comparison with studies that used the same paradigm

Other studies (Gildea et al., 2015; Hodges et al., 2009; Karayannis et al., 2013) used similar methodology to assess trunk dynamics in response to a force perturbation in the current study. However, direct comparison of the stiffness and damping values with those obtained by Gildea et al. (2015) is limited because trunk dynamical parameters were estimated over a fixed time window length (0.329 s) in that study. Effective trunk stiffness estimates of the current study were higher than those obtained by Hodges et al. (2009) and Karayannis et al. (2013). There are several possible explanations for this. First, the tested population differed between studies. Here we tested only males, and effective stiffness is higher in males than females (Griffioen and van Dieën, 2020; Miller et al., 2012; Vazirian et al., 2016). Second, there are some differences in experimental setup. Participants in the present study also had electrodes in situ for fine-wire muscle electromyographic recordings (Hodges et al., 2013), and Karayannis et al. (2013) generated trunk perturbations by removal of a smaller load (7.5% of body weight), perhaps requiring less stiffness to resist the perturbation. Third, pain anticipation can induce changes in motor control (Moseley et al., 2004). Both fear of pain (Karayannis et al., 2013) and negative pain believes (Griffioen, 2020) have been associated with higher trunk stiffness. However, we did not measure fear of pain or pain beliefs in the current study. Considering the methodological differences between the studies and the repeated-measures design, we believe the impact of experimental LBP on trunk dynamics are both qualitatively and quantitatively valid.

4.2. Qualitative comparison with studies that used force perturbations

Direct comparison of estimated effective trunk stiffness and damping values is challenging due to differences in experimental methods (Bazrgari et al., 2012). We therefore compared our findings qualitatively with other studies (Table 1). In contrast with the current study, with clinical LBP, some studies observed higher stiffness (Freddolini et al., 2014; Griffioen, 2020; Hodges et al., 2009) and lower damping (Gildea et al., 2015; Hodges et al., 2009). However, other studies reported no difference between people with and without clinical LBP (Ludvig et al., 2019; Wong et al., 2013) or observed higher effective trunk damping in clinical LBP than pain-free controls (Moreno Catala et al., 2018), similar to the effect of experimental LBP in the current study. Variation in findings in clinical LBP might highlight the heterogeneous nature of clinical LBP and/or differences in experimental paradigms between studies. However, due to the similarities in methods with the studies of Hodges et al. (2009) and Gildea et al. (2015), these provide the most valid comparison and underpin the conclusion that acute noxious input induces an adaptation that contrasts that observed in more clinical persistent LBP presentations.

4.3. Clinical pain versus experimental pain adaptation

Clinical LBP might compel selection of a different adaptation strategy from that available to individuals with no history of clinical LBP subjected to experimental pain, such as those studied here. The adaptation to clinical LBP could be influenced by changes in osteoligamentous structures from injury (Panjabi, 1992), impaired proprioception (Brumagne et al., 2000), changes in spinal muscle morphology (Hides et al., 1994; Mannion et al., 2000; Shahidi et al., 2020; Teichtahl et al., 2015) that can alter force production capability, altered trunk muscle function (Hodges and Richardson, 1996; MacDonald et al., 2009), higher intrinsic stiffness observed in some individuals with clinical LBP (Gombatto et al., 2008; Latimer et al., 1996) and/or fear of pain (Karayannis et al., 2013), or negative pain beliefs (Griffioen, 2020), both shown to increase trunk stiffness.

Increased stiffness might be achieved simply, using co-activation of trunk flexor and extensor muscles (Gardner-Morse and Stokes, 1998; Lee et al., 2006) and is an effective strategy for limiting movement amplitude in response to some perturbations (Gardner-Morse and Stokes, 1998; Reeves et al., 2007). This strategy places less demand on active control, removing the need for accurate feedback and finely coordinated muscle responses (Andersen et al., 2004; Essendrop et al., 2002; Granata and Marras, 2000; Krajcarski et al., 1999; Stokes et al., 2000; Vera-Garcia et al., 2006). Force generated by increased trunk stiffness (if muscles are pre-activated prior to perturbation) is not delayed because force is generated via instantaneous intrinsic muscle and joint properties rather than depending on the delayed reflex response and voluntary pathways (Reeves et al., 2007). However, trunk muscle co-contraction increases spinal compression loads (Gardner-Morse and Stokes, 1998; Mizrahi, 2015; Vera-Garcia et al., 2006) and muscle fatigue (Gardner-Morse and Stokes, 1998; van Dieën et al., 2009) with potential negative impacts on long-term tissue health (Vera-Garcia et al., 2006).

Increased trunk damping may benefit trunk stability in the dynamic sense. Among possible benefits of increased damping, it would smooth movements at higher frequencies, thus limiting rapid movement changes and potentially reduce peak forces (Mizrahi, 2015). The resultant system would be less underdamped, taking longer to reach the new equilibrium state but with fewer movement oscillations requiring potentially smaller muscle forces. This concurs with the suggestion that optimal control of the spine requires not only force response based on position feedback (stiffness), but also force response based on velocity feedback (damping) (Reeves and Cholewicki, 2009).

The main disadvantage of the strategy of decreased trunk stiffness and increased damping is that it resulted in greater trunk displacement as observed during pain compared to both before- and after pain conditions. Protection via limiting trunk displacement might be an important factor for trunk control that is preferred in individuals with clinical LBP, but this was not the case in healthy

Table 1
Qualitative comparison of effective stiffness and damping values of studies with and without low back pain.

Clinical LBP studies	LBP population	Current LBP	LBP duration (weeks)	Tested posture	Perturbation method	LBP versus Controls	
						B	*K*
Moreno Catala et al. (2018)	Non-specific LBP	Yes	>12	Semi-seated	Quick release	Higher	NS
Freddolini et al. (2014)	Non-specific LBP	Yes	>6	Seated	Unstable seat perturbation	NS	Higher
Gildea et al. (2015)	Non-specific LBP	No	<24–676	Semi-seated	Weight drop	Lower	NS
Griffioen (2020)	Non-specific LBP	Yes	>12	Semi-seated	Force perturbations	NS	Higher
Hodges et al. (2009)	Non-specific LBP	No	Recurrent (pain free)	Semi-seated	Weight drop	Lower	Higher
Ludvig et al. (2019)	Non-specific LBP	Yes	>4	Standing	Force perturbations	NS	NS
Miller et al. (2013)	Prior to exercise induced LBP	Minimal	>24	Seated	Force perturbations	Set to 0–	Higher
Wong et al. (2013)	Non-specific LBP	Yes	Not reported	Prone	Mechanical indenter	NA	NS
Experimental LBP studies						Pain versus no pain	
Current study	No history of LBP	Yes (experimental LBP)	NA	Semi-seated	Weight drop	Higher	Lower
Hodges et al. (2013)	No history of LBP	yes (experimental LBP)	NA	Semi-seated	Slow movements about neutral	NA	Higher
Wong et al. (2016)	LBP-free in last year	Yes (experimental pain)	Na	Prone	Mechanical indenter	NA	Higher

Note that in Miller et al. (2013) the control group increased stiffness to similar levels to those of people with exercise-induced LBP after exercise, whereas people with exercise-induced LBP did not alter effective trunk stiffness after exercise. Hodges et al. (2013) and the current study investigated the same participants, but trunk stiffness was estimated using modelling informed by electromyographic recordings. Abbreviations: *B*; effective trunk damping, *K*; effective trunk stiffness, NS; not significant, na; not applicable.

individuals during pain that was induced experimentally in a back muscle.

Participants of the current study were also tested during the same session using a paradigm that estimates spine stiffness using a trunk muscle activation driven model during slow movements (quasi static) about the neutral sitting position (Hodges et al., 2013). In contrast to the current findings, experimental LBP increased trunk stiffness compared to non-pain conditions. Increased stiffness with experimental pain was also observed by Wong et al (2016) using a device that measures force and displacement of the spine while lying prone (Table 1). Contrasting observations within our cohort imply that adaptation to pain is task specific (Hodges et al., 2013). However, in both Hodges et al. (2013) and Wong et al. (2016) stiffness was assessed in quasi-static condition and increasing damping might not be a suitable strategy because velocity of the assessed motions was minimal, and control of displacement via increased stiffness seems to be the only viable option.

4.4. Methodological considerations

Some methodological limitations require consideration. First, the model used to predict trunk displacement assumes that trunk stiffness and damping do not change over time, yet this is a simplification of actual trunk control. Despite this simplification, the second order linear model predicted trunk displacement accurately. Second, trunk dynamics were calculated based on the maximum trunk displacement and the time taken to reach this displacement. Longer and further displacements in the pain versus non-pain conditions might have influenced the estimation of trunk dynamical parameters (Bazrgari et al., 2012). To assess for possible bias, we re-modelled trunk displacement using a fixed displacement duration (i.e., the amount of time allowed for the trunk to displace) of 300 ms. The results did not modify our original findings. Third, trunk dynamics in relation to certain force frequency inputs are challenging to quantify using the trunk perturbation paradigm in this study. Variation in the frequency of force perturbations will influence trunk dynamical properties and current findings should be viewed as limited to our perturbation paradigm. Fourth, our lumped-parameter model cannot separate passive and active components of control and our effective stiffness and damping estimates include both components over the duration of the response to the perturbations. Last, the experimental pain paradigm (intramuscular injections of hypertonic saline) was not aimed to replicate clinical LBP. Clinical pain is complex and can originate from various sources (e.g., muscle, joint, ligaments, etc.) due to different factors (e.g., mechanical loading, inflammation, injury), and underlying mechanisms differ substantially depending on whether the pain is acute or persistent/chronic. Experimental pain evaluates the response to a short-term nociceptive event, and findings may not be extrapolated to clinical pain conditions (Edens and Gil, 1995). Instead, experimental pain can provide valuable information about neuromuscular responses and/or adaptations to nociceptive events in the absence of tissue damage, in a carefully controlled manner (Graven-Nielsen 2006). Further studies of individuals with clinical acute LBP are needed for comparisons to investigate whether adaptations are similar to those induced with experimental LBP, or to those who experience recurrent (acute) LBP episodes.

4.5. Conclusion

To conclude, when acute experimental nociceptive input was induced by injection of hypertonic saline unilaterally into the trunk extensor muscle, estimates of trunk stiffness decreased and damping increased compared to both pre- and post-experimental pain conditions using a weight drop paradigm. We interpret our results to show that, when challenged by a step force perturbation, a healthy system adapts to noxious input by controlling trunk velocity and that this contrasts the observation of control of trunk displacement via increased stiffness that has been observed during remission from recurrent clinical LBP.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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References

Andersen, T.B., Essendrop, M., Schibye, B., 2004. Movement of the upper body and muscle activity patterns following a rapidly applied load: the influence of pre-load alterations. Eur. J. Appl. Physiol. 91, 488–492.
Bazrgari, B., Nussbaum, M.A., Madigan, M.L., 2012. Estimation of trunk mechanical properties using system identification: effects of experimental setup and modelling assumptions. Comput. Methods Biomech. Biomed. Eng. 15, 1001–1009.
Brumagne, S., Cordo, P., Lysens, R., Verschueren, S., Swinnen, S., 2000. The role of paraspinal muscle spindles in lumbosacral position sense in individuals with and without low back pain. Spine 25, 989.
Cholewicki, J., Simons, A.P., Radebold, A., 2000. Effects of external trunk loads on lumbar spine stability. J. Biomech. 33, 1377–1385.
Colin Cameron, A., Miller, D.L., 2015. A practitioner's guide to cluster-robust inference. J. Hum. Resour. 50, 317–372.
Edens, J.L., Gil, K.M., 1995. Experimental induction of pain: Utility in the study of clinical pain. Behav. Ther. 26, 197–216.
Essendrop, M., Andersen, T.B., Schibye, B., 2002. Increase in spinal stability obtained at levels of intra-abdominal pressure and back muscle activity realistic to work situations. Appl. Ergon. 33, 471–476.
Freddolini, M., Strike, S., Lee, R.Y.W., 2014. Stiffness properties of the trunk in people with low back pain. Hum. Mov. Sci. 36, 70–79.
Gardner-Morse, M.G., Stokes, I.A.F., 1998. The effects of abdominal muscle coactivation on lumbar spine stability. Spine 23, 86–91.
Gildea, J.E., Hoorn, W.V.D., Hides, J.A., Hodges, P.W., 2015. Trunk dynamics are impaired in ballet dancers with back pain but improve with imagery. Med. Sci. Sports Exerc. 47, 1665–1671.
Gombatto, S.P., Norton, B.J., Scholtes, S.A., Dillen, L.R.V., 2008. Differences in symmetry of lumbar region passive tissue characteristics between people with and people without low back pain. Clin. Biomech. (Bristol, Avon) 23, 986–995.
Granata, K.P., Marras, W.S., 2000. Cost-Benefit of Muscle Cocontraction in Protecting Against Spinal Instability. Spine 25, 1398–1404.
Graven-Nielsen, T., 2006. Fundamentals of muscle pain, referred pain, and deep tissue hyperalgesia. Scand. J. Rheumatol. 35, 1–43.
Griffioen, M., Drunen, P.V., Maaswinkel, E., Perez, R.S.G.M., Happee, R., Dieën, J.H.V., 2020. Identification of intrinsic and reflexive contributions to trunk stabilization in patients with low back pain: a case-control study. Eur. Spine J.: Off. Publ. Eur. Spine Soc., Eur. Spinal Deform. Soc Eur. Sect. Cervical Spine Res. Soc., 1–9
Griffioen, M., van Dieën, J.H., 2020. Effects of age and sex on trunk motor control. J. Biomech. 102, 109607.
Hides, J.A., Stokes, M.J., Saide, M., Jull, G.A., Cooper, D.H., 1994. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. Spine 19, 165–172.
Hodges, P., van den Hoorn, W., Dawson, A., Cholewicki, J., 2009. Changes in the mechanical properties of the trunk in low back pain may be associated with recurrence. J. Biomech. 42, 61–66.
Hodges, P.W., Coppieters, M.W., MacDonald, D., Cholewicki, J., 2013. New insight into motor adaptation to pain revealed by a combination of modelling and empirical approaches: New insight into motor adaptation to pain. Eur. J. Pain 17, 1138–1146.
Hodges, P.W., Richardson, C.A., 1996. Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. Spine (Phila Pa 1976) 21, 2640–2650.
Hodges, P.W., Tucker, K.J., 2011. Moving differently in pain: a new theory to explain the adaptation to pain. Pain 152, S90–S98.
Karayannis, N.V., Smeets, R.J., van den Hoorn, W., Hodges, P.W., 2013. Fear of movement is related to trunk stiffness in low back pain. PLoS ONE 8, e67779.
Krajcarski, S.R., Potvin, J.R., Chiang, J., 1999. The in vivo dynamic response of the spine to perturbations causing rapid flexion: effects of pre-load and step input magnitude. Clin. Biomech. 14, 54–62.
Latimer, J., Lee, M., Adams, R., Moran, C.M., 1996. An investigation of the relationship between low back pain and lumbar posteroanterior stiffness. J. Manipulative Physiol. Ther. 19, 587–591.
Lee, P.J., Rogers, E.L., Granata, K.P., 2006. Active trunk stiffness increases with co-contraction. J. Electromyogr. Kinesiol. 16, 51–57.
Ludvig, D., Preuss, R., Lariviere, C., 2019. The effect of extensible and non-extensible lumbar belts on trunk muscle activity and lumbar stiffness in subjects with and without low-back pain. Clin. Biomech. (Bristol, Avon) 67, 45–51.
Lund, J.P., Donga, R., Widmer, C.G., Stohler, C.S., 1991. The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. Can. J. Physiol. Pharmacol. 69, 683–694.
MacDonald, D., Moseley, L.G., Hodges, P.W., 2009. Why do some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. Pain 142, 183–188.
Mannion, A.F., Käser, L., Weber, E., Rhyner, A., Dvorak, J., Müntener, M., 2000. Influence of age and duration of symptoms on fibre type distribution and size of the back muscles in chronic low back pain patients. Eur. Spine J. 9, 273–281.
Miller, E.M., Bazrgari, B., Nussbaum, M.A., Madigan, M.L., 2012. Effects of gender, preload, and trunk angle on intrinsic trunk stiffness. J. Musculoskelet. Res. 15.
Miller, E.M., Bazrgari, B., Nussbaum, M.A., Madigan, M.L., 2013. Effects of exercise-induced low back pain on intrinsic trunk stiffness and paraspinal muscle reflexes. J. Biomech. 46, 801–805.
Mizrahi, J., 2015. Mechanical impedance and its relations to motor control, limb dynamics, and motion biomechanics. J. Med. Biol. Eng. 35, 1–20.
Moreno Catala, M., Schroll, A., Laube, G., Arampatzis, A., 2018. Muscle strength and neuromuscular control in low-back pain: elite athletes versus general population. Front. Neurosci. 12, 436.
Moseley, G.L., Nicholas, M.K., Hodges, P.W., 2004. Does anticipation of back pain predispose to back trouble?. Brain 127, 2339–2347.
Panjabi, M.M., 1992. The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis. J. Spinal Disord. 5, 390–396. discussion 397.
Reeves, N.P., Cholewicki, J., 2009. Expanding our view of the spine system. Eur. Spine J.: Off. Publ. Eur. Spine Soc., Eur. Spinal Deform. Soc Eur. Sect. Cervical Spine Res. Soc. 19, 331–332.
Reeves, N.P., Narendra, K.S., Cholewicki, J., 2007. Spine stability: The six blind men and the elephant. Clin. Biomech. 22, 266–274.
Shahidi, B., Fisch, K.M., Gibbons, M.C., Ward, S.R., 2020. Increased fibrogenic gene expression in multifidus muscles of patients with chronic versus acute lumbar spine pathology. Spine 45, E189–E195.
Stokes, I.A.F., Gardner-Morse, M.G., Henry, S.M., Badger, G.J., 2000. Decrease in trunk muscular response to perturbation with preactivation of lumbar spinal musculature. Spine 25, 1957–1964.
Teichtahl, A.J., Urquhart, D.M., Wang, Y., Wluka, A.E., Wijethilake, P., O'Sullivan, R., Cicuttini, F.M., 2015. Fat infiltration of paraspinal muscles is associated with low back pain, disability, and structural abnormalities in community-based adults. Spine J. 15, 1593–1601.
Tsuji, T., Morasso, P.G., Goto, K., Ito, K., 1995. Human hand impedance characteristics during maintained posture. Biol. Cybern. 72, 475–485.
van den Hoorn, W., Bruijn, S.M., Meijer, O.G., Hodges, P.W., van Dieën, J.H., 2012. Mechanical coupling between transverse plane pelvis and thorax rotations during gait is higher in people with low back pain. J. Biomech. 45, 342–347.
van Dieën, J., Cholewicki, J., Radebold, A., 2003a. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. Spine 28, 834–841.
van Dieën, J.H., Putten, E.P.W.-V.D., Kingma, I., Looze, M.P.D., 2009. Low-level activity of the trunk extensor muscles causes electromyographic manifestations of fatigue in absence of decreased oxygenation. J. Electromyogr. Kinesiol. 19, 398–406.
van Dieën, J.H., Selen, L.P.J., Cholewicki, J., 2003b. Trunk muscle activation in low-back pain patients, an analysis of the literature. J. Electromyogr. Kinesiol. 13, 333–351.
Vazirian, M., Shojaei, I., Tromp, R.L., Nussbaum, M.A., Bazrgari, B., 2016. Age-related differences in trunk intrinsic stiffness. J. Biomech. 49, 926–932.
Vera-Garcia, F.J., Brown, S.H.M., Gray, J.R., McGill, S.M., 2006. Effects of different levels of torso coactivation on trunk muscular and kinematic responses to posteriorly applied sudden loads. Clin. Biomech. 21, 443–455.
Wong, A.Y., Kawchuk, G., Parent, E., Prasad, N., 2013. Within- and between-day reliability of spinal stiffness measurements obtained using a computer controlled mechanical indenter in individuals with and without low back pain. Man Ther. 18, 395–402.
Wong, A.Y.L., Parent, E.C., Prasad, N., Huang, C., Chan, K.M., Kawchuk, G.N., 2016. Does experimental low back pain change posteroanterior lumbar spinal stiffness and trunk muscle activity? A randomized crossover study. Clin. Biomech. 34, 45–52.