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Altered trunk muscle coordination during rapid trunk flexion in people in remission of recurrent low back pain

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ABSTRACT

People with a history of low back pain (LBP) are at high risk to encounter additional LBP episodes. During LBP remission, altered trunk muscle control has been suggested to negatively impact spinal health. As sudden LBP onset is commonly reported during trunk flexion, the aim of the current study is to investigate whether dynamic trunk muscle recruitment is altered in LBP remission. Eleven people in remission of recurrent LBP and 14 pain free controls performed cued trunk flexion during a loaded and unloaded condition. Electromyographic activity was recorded from paraspinal (lumbar and thoracic erector spinae, latissimus dorsi, deep and superficial multifidus) and abdominal muscles (obliquus internus, externus and rectus abdominis) with surface and fine-wire electrodes. LBP participants exhibited higher levels of co-contraction of flexor/extensor muscles, lower agonistic abdominal and higher antagonistic paraspinal muscle activity than controls, both when data were analyzed in grouped and individual muscle behavior. A sub-analysis in people with unilateral LBP ($n = 6$) pointed to opposing changes in deep and superficial multifidus in relation to the pain side. These results suggest that dynamic trunk muscle control is modified during LBP remission, and might possibly increase spinal load and result in earlier muscle fatigue due to intensified muscle usage. These negative consequences for spinal health could possibly contribute to recurrence of LBP.

1. Introduction

Low back pain (LBP) is commonly reported to start during self-initiated sudden movements of the lumbar spine at work, home and sports (Omino and Hayashi, 1992; Troup et al., 1981). Such onset of LBP during trunk movement could be mediated by inadequate trunk muscle control (Cholewicki et al., 2002), such as those that have been causally linked to spinal injury and pain in other tasks (Cholewicki et al., 2005).

Voluntary trunk movements require finely-tuned coordination of the spinal muscles in order to provide optimal control of dynamic intervertebral, spinal and postural stability while concurrently perform the intended movement trajectory (Reeves et al., 2007). In this context, dynamic motor control involves an interplay between feedback and feedforward control mechanisms to modulate muscle activity to control changing internal and external spine forces during lumbar movement (Panjabi, 1992). Although muscle activation contributes throughout the entire range of movement, the support is essential around the mid-range where passive structures contribute negligibly to spinal stiffness (neutral zone) (Cholewicki et al., 1997; Panjabi, 1992). When acceleration increases (Granata and England, 2006) and predictability decreases, the potential for error enlarges. Inability of the motor system to meet the task demands may lead to spinal injury and pain (Cholewicki et al., 2005; Panjabi, 2003).

People with a history of LBP have an elevated risk of encountering additional LBP episodes (Cholewicki et al., 2005; Von Korff, 1994). Changes in motor control that persist during remission from symptoms of LBP have been proposed to contribute to this increased risk (Hodges and Richardson, 1996; Cholewicki et al., 2005; Hodges and Tucker, 2011). Impaired muscle coordination during functional trunk movements may maintain this cycle, but such tasks have received limited attention.

Most research during LBP remission has focused on changes in muscle recruitment in association with perturbations of neutral spine positions, including arm movements (Macdonald et al., 2009) and external trunk loading (predictable (Macdonald et al., 2010; Magnusson et al., 1996) or unpredictable (Cholewicki et al., 2002; Macdonald et al., 2010; Magnusson et al., 1996; Radebold et al., 2000, 2001)). In these tasks, muscle activity is automatically adjusted to overcome the spinal perturbation and restore/maintain the spinal orientation, but people with a history

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of LBP often have delayed and decreased activity of deeper muscles (Macdonald et al., 2009, 2010), and increased co-contraction of superficial muscles (Cholewicki et al., 2002; Radebold et al., 2000, 2001). During movement of the trunk itself there is also the requirement to maintain spine integrity, but in this case, as the spine is moving through a trajectory. We hypothesized that, as for the spinal control in association with neutral spine perturbations, the motor control strategy to perform voluntary trunk movements would be adapted in people with a history of LBP. This could have potential consequences for stability of the trajectory and integrity of the spine.

Although studies in remission of LBP often focus either on deep (DM) and superficial (SM) fibers of multifidus (Macdonald et al., 2009, 2010) or the more superficial muscles (Cholewicki et al., 2002; Magnusson et al., 1996; Radebold et al., 2000, 2001), a thorough investigation of voluntary movement requires simultaneous investigation of multiple muscles.

This study aimed to investigate trunk muscle recruitment in people in remission from recurrent LBP and a healthy control group during rapid, loaded and unloaded, voluntary trunk flexion. Muscle recruitment was analyzed from several complementary perspectives, in order to identify dysfunctional recruitment patterns in grouped muscle behavior (including co-contraction, and agonistic/antagonistic muscle behavior), as well as muscle-specific dysfunctions in individual muscle behavior in people with a history of recurrent LBP.

2. Methods

2.1. Participants

Eleven individuals with a history of recurrent, non-specific LBP and 14 healthy individuals with no history of LBP were recruited (Table 1). Inclusion criteria for the LBP group (Table 2) were a recurrent pattern of LBP (at least two episodes) that interfered with activities of daily living and/or required treatment. Participants with LBP were tested during symptom remission. Individuals were excluded if they had: pain elsewhere in the body; spinal deformities; spine surgery; participated in lumbar muscle training in the past year; or any major neurological, circulatory, respiratory or orthopedic condition. The study was approved by the Institutional Medical Research Ethics Committee and conformed to the Declaration of Helsinki. All participants provided signed informed consent.

2.2. Electromyography

EMG activity was recorded bilaterally from DM and SM at L4 using bipolar intramuscular fine-wire electrodes (Teflon-coated stainless steel wires, 75 μm with 1 mm of Teflon removed, tips bent back 1 and 3 mm to form hooks). Electrodes were threaded into a hypodermic needle (SM: 0.6 × 32 mm, DM: 0.65 × 70 mm) and inserted using real-time ultrasound guidance (Macdonald et al., 2009). Pairs of surface electrodes (disposable Ag/AgCl, 10 mm diameter, 20 mm inter-electrode distance, Ambu-Blue Sensor N, Malaysia), were placed bilaterally over: lumbar erector spinae (LES) at L4, thoracic erector spinae (TES) at T9, latissimus dorsi (LAT) at T9 (Marras et al., 1995), obliquus externus abdominis (OE) inferior to rib angle, obliquus internus abdominis (OI) medial to anterior superior iliac spine, and rectus abdominis (RA) adjacent to the umbilicus (Ng et al., 1998). A ground electrode was placed over the right iliac crest. Skin preparation included abrasion and alcohol cleaning. EMG data were amplified 2000 times, band pass filtered between 30 and 1000 Hz and sampled at 2000 Hz (surface EMG) and 3000 Hz (fine-wire EMG) using Spike2 software and Power 1401 data acquisition system (Cambridge Electronic Design, Cambridge, UK). The 30 Hz lower cut on the EMG filter was selected as it achieved the optimal balance between removal of unwanted movement artifact (if present) and the minor loss of energy from the lower frequency components of the EMG data.

Table 1
Participant demographics (mean [SD]).

Variable	LBP group	Control group	*p*-Value
n	11	14	–
Male:female	7:4	6:8	0.302
Age (years)	30 (9)	25 (6)	0.185
Body weight (kg)	78 (16)	61 (12)	0.007*
Body length (m)	177 (11)	167 (11)	0.022*
BMI (kg/m^2)	24.5 (2.5)	22.0 (2.9)	0.033*

SD – standard deviation. LBP – low back pain. BMI – body mass index.
* $P < 0.05$.

Table 2
Pain characteristics for LBP group (mean [SD]).

Variable	
Time since first onset of LBP (months)	98 (76)
Frequency of episodes (per year)	8 (7)
Time since last episode (days)	92 (122)
Duration of an episode (days)	9 (9)
Pain intensity during episode (0 = no pain at all, 10 = worst pain that can be imagined)	5 (2)
Disability during episode (0 = not disabled at all, 10 = totally disabled)	3 (2)

SD – standard deviation. LBP – low back pain.

2.3. Procedure

Trunk muscle control was evaluated during rapid voluntary trunk flexion from a semi-seated position with their pelvis fixed. A foam pad was placed in front of the participant to the range of trunk flexion (~20°) (Fig. 1). Participants moved forward as fast as possible immediately upon hearing an auditory cue and return. The task was performed without and with load (15% body weight) applied through a pulley system attached to the front of a harness to pre-activate the back muscles. Practice trials were allowed for familiarization. Ten proper repetitions of each condition were completed, separated by 5–7 s. Trunk acceleration and displacement were respectively measured with an Inertial Measurement Unit (SEN-10010, ±3 g, 300 mV/g sensitivity, Sparkfun, Colorado, USA) and linear wire potentiometer (full stroke 1000 mm, linearity ±0.15%, repeatability ±0.05%, Hontko, Taiwan) sampling at 2000 Hz.

Maximum voluntary isometric contractions (MVICs) were recorded (Cholewicki et al., 1997). Three repetitions each of resisted trunk extension (DM, SM, LES and TES), left and right trunk rotation (OE and OI), sit up (RA) and shoulder adduction (LAT) were completed with verbal encouragement, separated by 60 s rest.

2.4. Data analysis

Data were exported to Matlab (v7, Mathworks, USA) for further EMG amplitude analysis. EMG data were divided into 16 epochs based on the onset, peak and end of flexion identified from individual linear potentiometer data, to account for inter-individual variability in movement velocity: Epoch 1–50 ms before auditive cue (baseline EMG, B); Epochs 2–6 – five 100 ms epochs for 500 ms before flexion onset (preparatory phase, P1–P5); Epochs 7–11 – five equal duration epochs from onset to peak flexion (flexion phase, F1–F5); Epochs 12–16 – five equal duration epochs from peak to end of flexion (re-extension phase, R1–R5). The duration of the flexion movement was 1.14 ± 0.16 s for LBP patients and

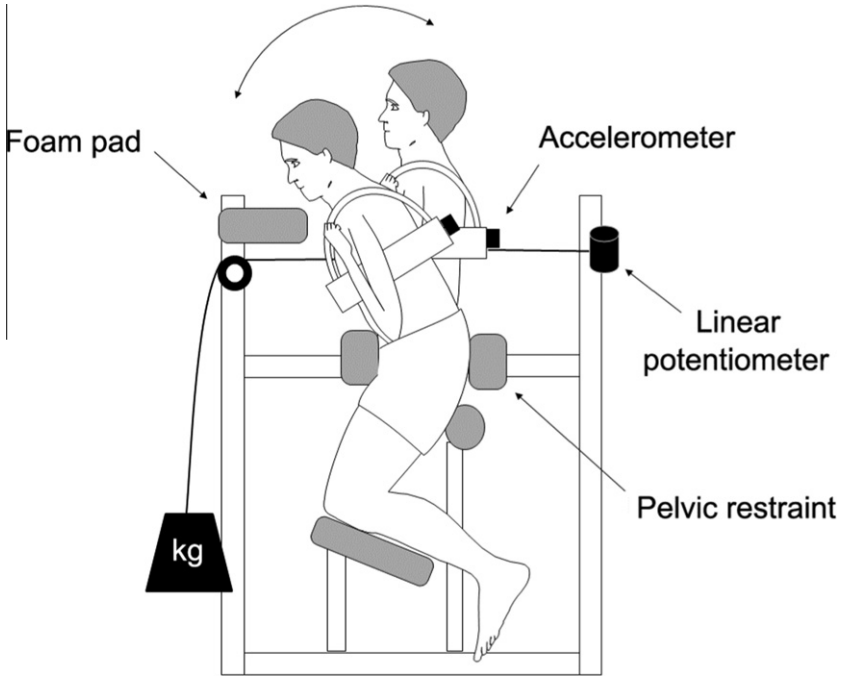

Fig. 1. Experimental set-up showing task position, pelvic restraint and position at the beginning (upright) and end of the flexion movement.

1.22 s ± 0.18 s for controls (each epoch was 10% of this duration) and did not differ between groups ($F = 2.15$; $p = 0.149$). For each epoch, root mean square (RMS) EMG amplitude was calculated and normalized to peak (averaged over 3 s) RMS–EMG during MVICs. In order to consider the validity of normalization of the EMG data to that recorded during MVIC efforts, the raw EMG amplitude during the MVIC trials was compared between groups. No difference was found for any muscle (main effect Group: $F = 0.376$; $p = 0.546$; interaction effect between Group and Muscle: $F = 0.540$; $p = 0.803$). This implies that there was no systematic difference in activity during MVIC efforts which implies that participants with a history of LBP performed true maximal efforts.

EMG activation patterns were analyzed via a multiscale approach, in order to provide a complementary view on several levels of trunk muscle control within a clinically relevant perspective of muscle dysfunction. First, a binary index of co-contraction was calculated. A value of "1" was assigned if the sum of MVIC normalized EMG amplitude of the flexors (OE, OI and RA) exceeded 30% and the sum of the extensors (DM, SM, LES, TES and LAT) exceeded 50%. A value of "0" was assigned if the amplitude of either sum was less. This construct accounts for individual variation in recruitment strategy by using the sum of the muscles rather than the requirement for each individual muscle to exceed 10% MVIC, as recent work has highlighted the individual specific nature of adaptation to trunk muscle coordination in people with low back pain (Hodges et al., 2006). The threshold has been empirically determined, based on the MVIC normalized EMG of each of the muscles generally exceeded 10% during the flexion phase. Second, MVIC normalized EMG activity of the three flexor and five extensor muscles was summed to reflect net agonist and antagonist activity, respectively. Although neither measure accounts for differences in moment arm or muscle mass, we argue they provide an estimate of tendency to co-contract flexor and extensor trunk muscles and an estimate of net activity of each muscle group. Third, the MVIC normalized EMG amplitude was analyzed for each muscle individually.

Trunk movement was analyzed as peak acceleration and movement duration (onset to end flexion) identified from accelerometer data, and maximum trunk displacement from potentiometer data.

2.5. Statistical analyses

Analyses were performed using Statistica v7 (Statsoft, USA). Differences between groups for demographic characteristics and movement parameters were investigated with independent samples *t*-tests and chi-square test. EMG data were analyzed in several ways with repeated-measures analyses of variance (ANOVA) using General Linear Models (GLMs). First, the co-contraction index and summed EMG amplitudes (agonists and antagonists) were compared between groups, loading condition and epochs. Second, EMG data for each muscle (average of left and right sides) were compared between groups, muscles, loading conditions and epochs. Third, an exploratory analysis was conducted on a subgroup of participants with a history of unilateral LBP ($n = 6$) to investigate whether EMG of individual muscles differed with respect to the side of previous pain during performance of a symmetrical task. Such side specific differences have been observed with respect to unilateral recurrent LBP (Macdonald et al., 2010). EMG data were compared between groups (previously painful and non-painful side vs. average of sides from the control group [$n = 14$]), muscles, conditions and epochs.

3. Results

3.1. Co-contraction index

LBP participants had a greater propensity to co-contract flexor and extensor muscles than controls ($F = 5.92$; $p = 0.019$), and this did not differ between loading conditions or epochs (Fig. 2).

3.2. Summed agonist and antagonist EMG

An interaction effect between epoch and group ($F = 3.32$; $p < 0.001$) showed that summed EMG of abdominal/flexor muscles was lower for the LBP participants than controls during the flexion phase (F1–F2) (post hoc: $P < 0.005$) (Fig. 3A). There was no difference between loading conditions or groups during other epochs.

For the paraspinal/extensor muscles, an interaction effect between epoch and group ($F = 2.70$; $p < 0.001$) showed that summed EMG was higher in the LBP group than controls during flexion (F2–F5) and re-extension phases (R5) (post hoc: $P < 0.05$) (Fig. 3B). As expected, paraspinal muscle activity was greater in the loaded than unloaded condition during epochs in each phase (B, P1–P4, F5, R1–R4) for both groups (interaction between epoch and condition: $F = 2.94$; $p < 0.001$; post hoc $P < 0.05$).

3.3. Individual muscle EMG

Analysis of individual muscles showed a significant interaction between muscle, epoch and group ($F = 2.44$; $p < 0.001$). Post hoc analysis ($P < 0.05$) (Table 3) (Fig. 4) revealed DM, SM, LES and TES EMG were higher in LBP participants than controls for several epochs during flexion, and other epochs. LAT and OI EMG did not differ between groups for any epoch, but OE and RA EMG was lower in the LBP group than controls at the beginning of flexion. An interaction between muscle, epoch and condition ($F = 1.86$; $p < 0.001$) showed that DM, SM, LES and LAT EMG was greater in the loaded condition during some epochs (Online supplementary Table) (post hoc $P < 0.05$), but this was similar for both participant groups.

3.4. Exploratory analysis of painful vs. non-painful side

For the six participants with a history of unilateral LBP there was a significant interaction for Muscle * Epoch * Group for the previously painful ($F = 2.11$; $p < 0.001$) and non-painful sides ($F = 1.78$; $p < 0.001$). Post hoc analyses ($P < 0.05$) revealed that although LES, TES, LAT, OE, OI, and RA EMG on the previously painful and non-painful side generally responded similarly for both groups, the DM and SM response differed (Table 3). On the painful side, DM EMG was lower and SM EMG higher, whereas on the non-painful side, DM EMG was higher and SM EMG lower than controls (Fig. 5).

3.5. Trunk movement

As LBP participants had lower abdominal and greater paraspinal EMG, differences in movement were considered. Although the duration of movement ($F = 2.15$; $p = 0.149$; LBP = 1.14 ± 0.16 s; control = 1.22 ± 0.18 s) and maximum displacement ($F = 1.46$; $p = 0.234$; LBP = 133 ± 23 mm; control = 143 ± 31 mm) did not differ between groups, peak acceleration was less for the LBP group ($F = 6.14$; $p = 0.017$; LBP = 0.66 ± 0.11 m s^{-2}; control = 0.76 ± 0.15 m s^{-2}).

3.6. Adjustment of EMG analyses for differences in peak acceleration

GLM repeated measures analysis were repeated with inclusion of peak acceleration as covariate. Results were generally identical to those without this component, although some differences in interaction effects were identified (Table 4).

4. Discussion

Consistent with our hypothesis, participants with a history of recurring episodes of LBP exhibited a range of changes in trunk muscle recruitment in a dynamic voluntary trunk movement, despite the absence of pain. The differences in motor control from control participants were multifaceted and included a greater propensity for people with recurring LBP to co-contract flexor and extensor trunk muscles throughout the task, with a concurrent lesser agonist flexor muscle activity and greater extensor muscle activity than controls during trunk flexion. Generally, similar observations were made for individual muscle behavior, but our exploratory analysis of people with a history of unilateral LBP revealed differential, opposite changes in DM and SM related to the pain side.

Within a broad perspective, the binary index of co-contraction indicated a greater likelihood for people with a history of LBP than controls to activate flexors and extensors concurrently. Trunk muscle co-contraction is commonly observed in LBP (Cholewicki et al., 2002; Radebold et al., 2000; van Dieen et al., 2003a) and has been proposed a strategy to stiffen the spine to increase the safety margin for protection from perturbation (Cholewicki et al., 1997). Co-contraction depends on spinal control demands (Granata and Orishimo, 2001; van Dieen et al., 2003b) and these may be increased because of reduced passive spine stability, distorted proprioceptive input or reduced trunk muscle force with spinal injury/pain (Panjabi, 2003; van Dieen et al., 2003b). As suggested for numerous motor control changes with recurrent LBP (D'Hooge et al., in press; Macdonald et al., 2009, 2010; Radebold et al., 2000), increased co-contraction may reflect a suboptimal muscle recruitment strategy (Granata and Marras, 1995).

At the grouped muscle level, reduced agonist muscle activity and increased antagonist activity during flexion, and reduced peak trunk acceleration appears consistent with the predictions of the "pain adaptation" theory (Lund et al., 1991), predicting stereotypical changes in muscle recruitment to reduce a painful movement's amplitude and velocity.

Considering individual muscle behavior, activity of paraspinal muscles was increased in the LBP participants, similar as observed in the grouped paraspinal behavior. Antagonist LES activity has been commonly studied during trunk flexion. Although some

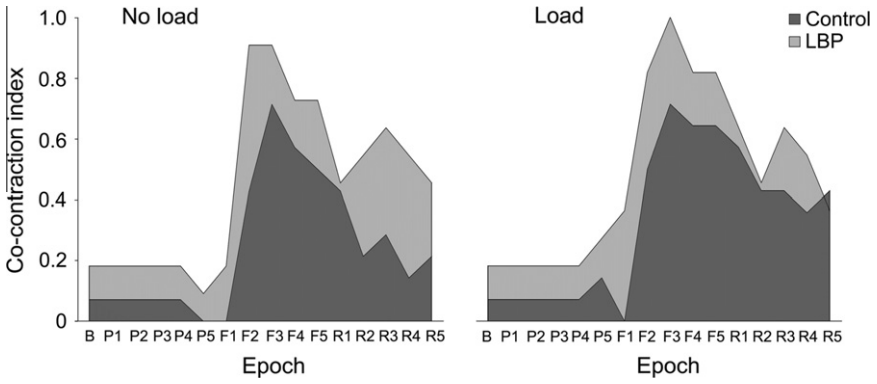

Fig. 2. *Co-contraction index.* Group data for co-contraction index (displaying proportion of participants with co-contraction of paraspinal and abdominal muscles) are shown for each epoch for trials with and without load in people with (LBP) and without (control) a history of low back pain.

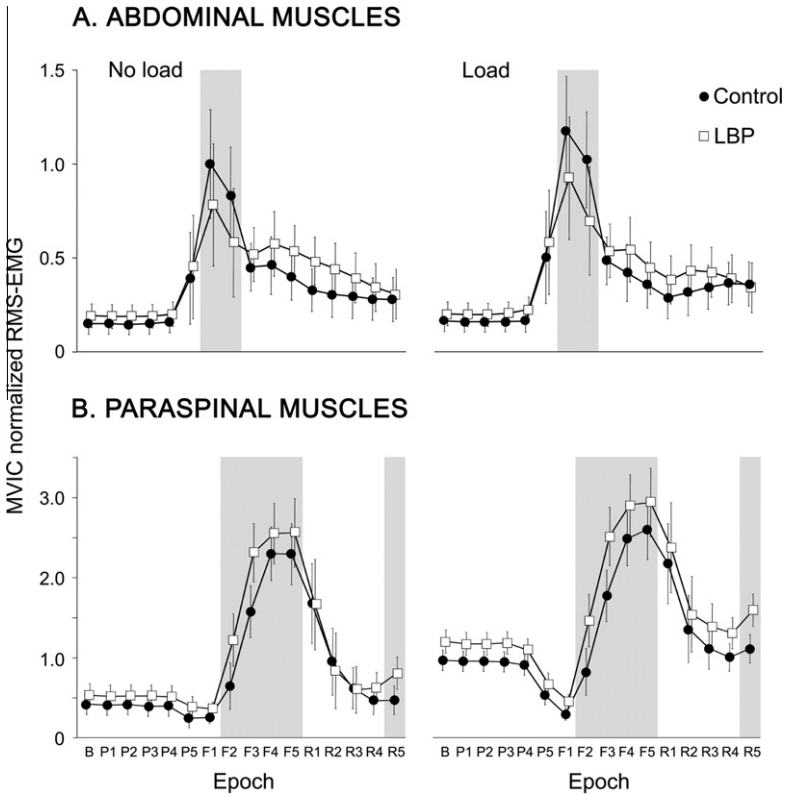

Fig. 3. *Summed EMG activity of the abdominal* (*A*) *and paraspinal* (*B*) *muscles*. Data for each epoch are shown for trials with and without load in people with (LBP) and without (control) a history of low back pain. Grey boxes indicate epochs with significant differences between LBP and control group ($P < 0.05$). Error bars represent 95% confidence intervals.

Table 3
Statistical comparison of RMS–EMG amplitude between groups and epochs for individual muscles.

Muscle	Post hoc Group * Epoch		
	Average of sides	Previously painful side	Previously non-painful side
	n: LBP = 11 CON = 14	*n*: LBP = 6 CON = 14	
DM	LBP > CON B, P2–P3, F2–F5, R5	CON > LBP F3–F5, R1–R2	LBP > CON B, P1–P5, F2–F5, R1, R3–R5
SM	LBP > CON F2–F3, R5	LBP > CON P3–P5, F2–F5, R1, R3–R5	CON > LBP F4–F5
LES	LBP > CON F2–F3	NS	NS
TES	LBP > CON F2–F5, R1, R5	LBP > CON F2–F5, R1, R5	LBP > CON F3–F5, R1
LAT	NS	NS	NS
OE	CON > LBP F1–F2	CON > LBP F1–F2	CON > LBP F1
OI	NS	LBP > CON R1–R2	LBP > CON R1
RA	CON > LBP F1	NS	CON > LBP F1

CON – control group; LBP – low back pain group; B – indicates significant epoch ($P < 0.05$) during baseline; P1–P5 – indicates significant epochs ($P < 0.05$) during the preparatory phase; F1–F5 – indicates significant epochs ($P < 0.05$) during the flexion phase; R1–R5 – indicates significant epochs ($P < 0.05$) during the re-extension phase of the flexion movement; DM – deep fibers of multifidus; SM – superficial fibers of multifidus; LES – lumbar erector spinae; TES – thoracic erector spinae; LAT – latissimus dorsi; OE – obliquus externus abdominis; OI – obliquus internus abdominis; RA – rectus abdominis; NS – no significant difference between LBP and control group.

report no changes with LBP (Lariviere, 2000; Sherman, 1985; Shirado et al., 1995), others described increased (Sihvonen et al., 1991) or decreased (Arena et al., 1989; Watson et al., 1997) LES EMG. Higher TES EMG has been reported with loaded movements (Lariviere, 2000). Differences between studies may be explained by variables of the flexion task such as movement speed (Granata and Orishimo, 2001); standing vs. semi-seated position (Radebold et al., 2000); small vs. full movement range (Shirado et al., 1995); EMG electrode location; and participant differences e.g. LBP diagnosis (Arena et al., 1989). Even when adjusting for differences in

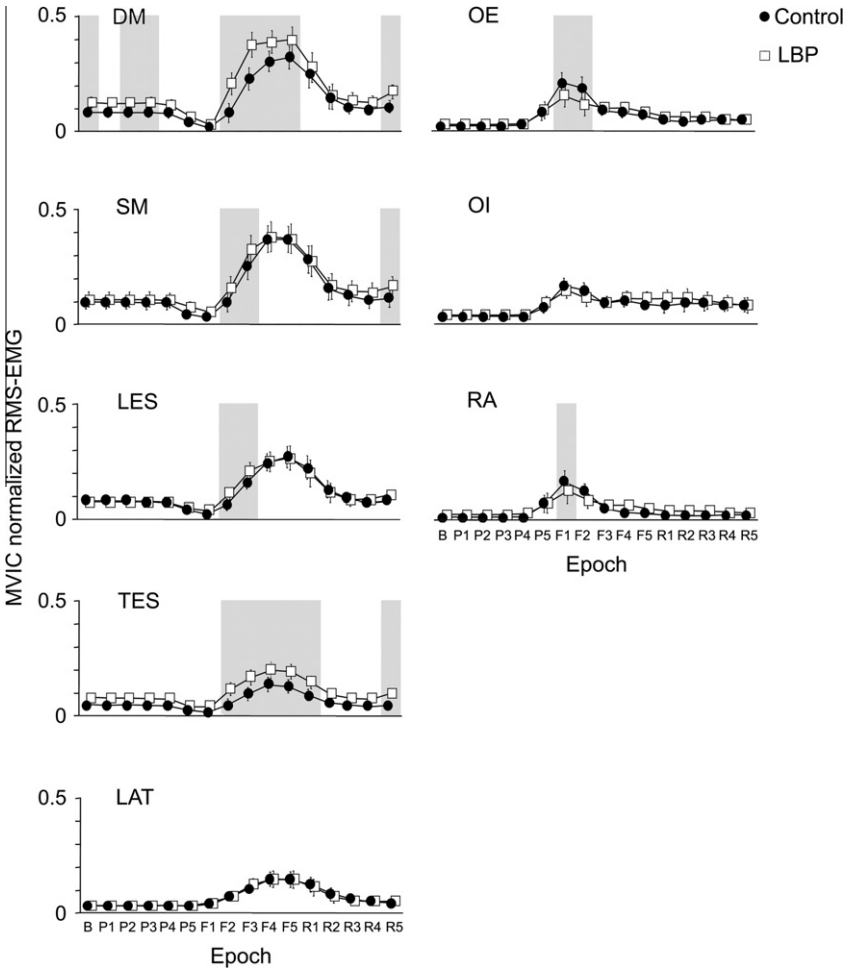

Fig. 4. *EMG activity of individual muscles.* Data (averaged for loads) are shown for deep (DM) and superficial (SM) multifidus, lumbar (LES) and thoracic (TES) erector spinae, latissimus dorsi (LAT), obliquus externus (OE) and internus abdominis (OI), and rectus abdominis (RA) in people with (LBP) and without (Control) low back pain. Grey boxes indicate epochs that differ between groups. Error bars – 95% confidence intervals.

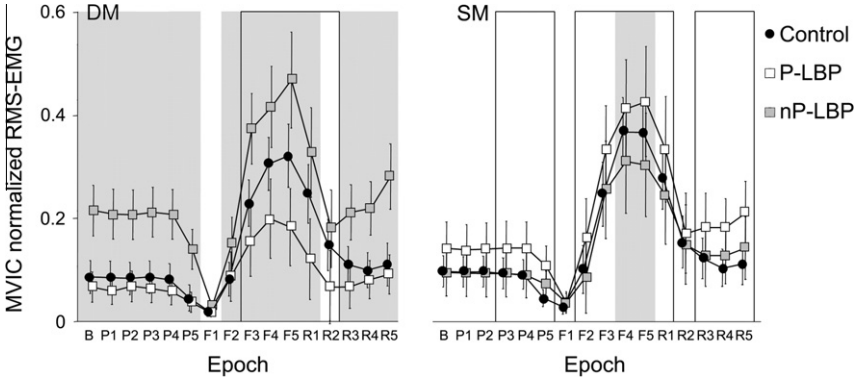

Fig. 5. EMG activity of deep (DM) and superficial (SM) multifidus with respect to side of pain. Data (averaged for loads) are shown for the previously painful (P-LBP) and non-painful side (nP-LBP) and Controls. White and grey boxes indicate epochs with differences between P-LBP or nP-LBP, and control data, respectively. Error bars – 95% confidence intervals.

peak trunk acceleration, differences remained in the present study. This corresponds with observations of Zedka et al. (1999) of reduced movement velocity during experimental pain, with persistent changes in LES EMG when matching velocity to pre-pain trials.

However, all individual muscles were not affected uniformly: no changes were detected for OI and LAT, and DM and SM were differentially affected in relation to the pain-side for the unilateral subgroup. EMG recorded with surface electrodes over OI reflects caudal regions of both OI and transversus abdominis muscles, which have lesser flexion moment arms compared to flexion torque-generating RA and OE, but also contribute to lumbopelvic stability (Bergmark, 1989). More complex functions of these muscles and inability to differentiate between them could explain the less predictable outcome. Also, differential changes were observed in

Table 4
Adjustment of statistical analysis with inclusion of covariate 'peak acceleration'.

Test	Significant factors	*p*-Value	Post hoc
Co-contraction index	Group	0.015*	LBP > CON
Agonists (sum of abdominals)	Epoch * Group	0.006*	CON > LBP F1–F2
Antagonists (sum of paraspinals)	Group	<0.001*	LBP > CON
Individual muscles	Epoch * Group	0.004*	LBP > CON F3–F4, R5

CON – control group; LBP – low back pain group; F1–F5 – indicates significant epochs ($P < 0.05$) during the flexion phase; R1–R5 – indicates significant epochs ($P < 0.05$) during the re-extension phase of the flexion movement.
* $P < 0.05$.

our exploratory analysis of unilateral pain. Diminished DM and augmented SM EMG on the previously painful side agree with previous reports of compromised deep and augmented superficial multifidus activity (Macdonald et al., 2009, 2010). Reduced activity of deeper spinal muscles in some participants, has been considered to imply less optimal control of intervertebral stability (Macdonald et al., 2006). Contrasting changes on the non-painful side (augmented DM, diminished SM) may reflect redistribution of activity between muscles to maintain symmetrical task output. These findings suggest that opposite and potentially compensatory changes in multifidus muscle activation can present during remission of unilateral recurrent LBP, and may be missed when both sides are analyzed together.

Non-uniform changes in individual muscle behavior compared to grouped muscle behavior implies more complex changes rather than uniform inhibition and facilitation of motoneuron pools of opposing muscle groups, as discussed in more recent theories of motor adaptation to pain (Hodges and Tucker, 2011). Our results encourage the differentiation between complementary levels of trunk muscle control, as different levels might reveal differential changes, which enhances the insight in trunk muscle dysfunction.

In general, participants with recurrent LBP exerted more co-contraction/paraspinal muscle activity than controls, both with and without load. Thus, LBP participants retained potential to modulate muscle activity, although at higher levels, in contrast to a report where addition of load did mask group differences (Silfies et al., 2005). This implies that people with recurring LBP exert a strategy normally used for higher loads, which implies a protective solution to reduce risk of further injury/pain (Hodges, 2011). Possible consequences of the altered, higher-load recruitment strategies during remission of LBP are potentially problematic as they could lead to earlier fatigue for a certain repetitive trunk movement task and compromise spinal stability, leaving it susceptible to pain/injury (Mannion, 1999). Furthermore, increased co-contraction and paraspinal muscle activity augments load on spinal structures, which although beneficial in the short term, may enhance the risk for injury in the long term (Gardner-Morse and Stokes, 1998; Granata and Marras, 1995).

Several methodological issues require consideration. First, technical issues meant accelerometer-data were not available for one participant in each group and reduced the power in the additional statistical analyses. Nevertheless, statistical effects with and without peak acceleration as a covariate were similar, except for loss of some details in interaction effects. Second, EMG was recorded during a dynamic task which could have introduced movement artifact in the EMG signal. The potential for movement artifact in the EMG recordings was minimized through careful fixation of the EMG-recording material during data collection, and selection of optimal band-pass filter settings. All data was visually inspected and any trial with movement artifact was excluded from analysis. Third, weight/height/BMI were higher for LBP participants, which could have contributed to lower peak acceleration in the LBP group. However, these characteristics did not appear significant covariates ($P > 0.05$) in the analysis of group differences in peak acceleration. Weight and BMI might influence EMG amplitude values measured with surface electrodes via differences in subcutaneous tissue thickness, but the use of EMG data in a normalized manner should have accounted for this. A final possible limitation is that the binary index of co-contraction used here provides a simple estimate of co-activation of the antagonist muscle groups and does not consider a range of biomechanical aspects that would permit detailed analysis of the mechanical aspects of the co-activation (e.g. muscle mass, moment arm). It has been used in this study to provide a simple measure of the simultaneous contraction between agonists (flexors) and antagonists (extensors), and (together with the other analyses) provides insight into complementary parameters of muscle control.

In conclusion, muscle coordination during rapid trunk flexion was altered in people in remission from recurrent LBP on several complementary levels of muscle control, both on grouped and individual muscle level. Although observations were generally consistent with predictions of the pain adaptation theory, and imply enhanced spine protection, several features imply more complex mechanisms than uniform inhibition/facilitation. These findings have potential repercussions for functional trunk movements performed in daily life and as such provide a potential pathophysiological mechanism for increased likelihood of subsequent episodes of LBP.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jelekin.2012.09.003.

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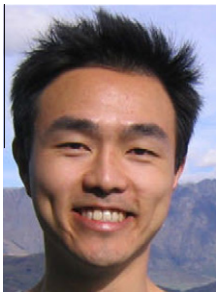

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