Why do some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain

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ABSTRACT

Approximately thirty-four percent of people who experience acute low back pain (LBP) will have recurrent episodes. It remains unclear why some people experience recurrences and others do not, but one possible cause is a loss of normal control of the back muscles. We investigated whether the control of the short and long fibres of the deep back muscles was different in people with recurrent unilateral LBP from healthy participants. Recurrent unilateral LBP patients, who were symptom free during testing, and a group of healthy volunteers, participated. Intramuscular and surface electrodes recorded the electromyographic activity (EMG) of the short and long fibres of the lumbar multifidus and the shoulder muscle, deltoid, during a postural perturbation associated with a rapid arm movement. EMG onsets of the short and long fibres, relative to that of deltoid, were compared between groups, muscles, and sides. In association with a postural perturbation, short fibre EMG onset occurred later in participants with recurrent unilateral LBP than in healthy participants ($p = 0.022$). The short fibres were active earlier than long fibres on both sides in the healthy participants ($p < 0.001$) and on the non-painful side in the LBP group ($p = 0.045$), but not on the previously painful side in the LBP group. Activity of deep back muscles is different in people with a recurrent unilateral LBP, despite the resolution of symptoms. Because deep back muscle activity is critical for normal spinal control, the current results provide the first evidence of a candidate mechanism for recurrent episodes.

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1. Introduction

Low back pain (LBP) is a common and disabling health problem with 60–90% of people experiencing an episode in their lifetime [9]. Furthermore, despite receiving intervention, and becoming pain-free, ~34% will experience an additional episode of LBP requiring further intervention [47]. This patient population, and those with persistent LBP, account for ~80% of the costs related to the management of LBP [46,47]. Regardless of an initial return to function and a recovery, some people continue to have recurrent episodic LBP. Unfortunately, we do not know why LBP becomes recurrent in some people and not in others.

A likely contributor to the recurrence of LBP is a change in control of the back muscles. The lumbar back muscles contribute to the control of spinal motion and stability [3,7,31,38,48], and are critical for spinal health [39,40]. Furthermore, changes in lumbar back muscle control have been observed in people with chronic LBP [23,26,30,41–44] and in people with LBP with sciatica

[28]. For example, the deep back muscles are less active in people with chronic LBP than in healthy controls while returning to standing from full trunk flexion [30]. However, the superficial back muscles are more active in people with LBP than in healthy controls during trunk movements [44], including an absence of the normal back muscle relaxation at full trunk flexion (the so-called 'flexion–relaxation phenomenon') [42]. Such changes in the back muscle activity are thought to reflect a change in the control strategy adopted by the nervous system. On the one hand, this adaptation appears to compromise the control of the spine. The reduction in deep back muscle activity would be expected to reduce the control, or 'fine tuning', of segmental motion that has been shown in biomechanical models and the experimentally injured lumbar spine [22,48]. On the other hand, the increased activity of superficial back muscles may serve to limit tensile forces and motion of injured/painful structures in the back [45].

One question that remains unanswered is: 'What happens to the control of the deep back muscles during remission from symptoms in people with recurrent LBP?'. This question is important because if the change in deep back muscle activity persists during remission, it could alter the control of segmental motion, and could be a factor in the recurrence of LBP. Although changes in numerous

muscles have been implicated in LBP, those in the back muscles are amongst the most consistent.

We answered this question using an established paradigm [2,4,8] that interrogates the control of the back muscles during a rapid voluntary arm movement. Arm movement provides a predictable spinal perturbation, and the associated back muscle activation reflects the preparatory postural strategy. Based on the histochemical [49] and morphological [12] changes in the back muscles of people with LBP, we hypothesized that changes in motor control would be more pronounced in the short fibres of the deep back muscles than in the long fibres, and that these changes would be greater on the previously painful side than on the asymptomatic side.

2. Methods

2.1. Participants

Fifteen people with recurrent (multiple episodes of LBP separated by periods of remission) unilateral LBP (7 male, mean (SD) age = 27 (7) years, height = 172 (8) cm and weight = 71 (14) kg) and 19 control subjects (9 male, age = 26 (5) years, height = 173 (9) cm and weight = 67 (11) kg) with no history of back pain participated in the study. To be considered to have recurrent LBP, patients were to have experienced an initial episode of unilateral LBP (symptoms from T12 to the upper buttock) at least 3 months prior with subsequent bouts of back pain, severe enough to limit their normal activity and make them seek treatment (medication, physiotherapy, chiropractic, etc.), separated by the periods of pain-free remission. Each participant was examined physically by an experienced clinician to verify an asymmetrical pattern of movement and/or symptom reproduction that supported the patient's report of unilateral LBP. All participants were pain-free at the time of testing.

Participants in the control group were to have had no LBP in the 2 years prior to participation in the study and no history of LBP that required intervention or limited functional abilities prior to that period. People who had any previous spinal surgery, major spinal deformities, respiratory or neurological conditions, or any orthopaedic condition which would have limited their ability to complete the experimental tasks were excluded from both groups.

Written informed consent was obtained. All procedures were approved by the Institutional Research Ethics Committee and conducted in accordance with the Declaration of Helsinki.

2.2. Electromyography

We used a well established method [32] to record electromyographic activity (EMG) of the deep and superficial back muscles on both sides of the spine, at the L5 spinal level. Two Teflon-coated 75 μm stainless steel wires were inserted, via a hypodermic needle (0.7×50 mm or 0.6×38 mm), under ultrasound guidance (5-MHz linear array transducer, Synergy CFM; Diasonics, Haifa, Israel).

For insertion of the intramuscular electrodes, participants were positioned in either supported sitting or side lying. The L5 vertebral lamina and the target muscles were clearly identified. The electrode that recorded short muscle fibre EMG was inserted ∼30 mm lateral to the midline and directed anteromedially until the tip of the needle reached the most medial aspect of the lamina of L5 (Fig. 1A). The electrode that recorded long muscle fibre EMG was inserted ∼40 mm lateral to the midline and directed anteromedially until the tip of the needle was visualized in the superficial back muscle (Fig. 1B). After the removal of the needle, gentle traction of the wires under ultrasound visualization confirmed the position of each electrode. Participants reported only mild transient
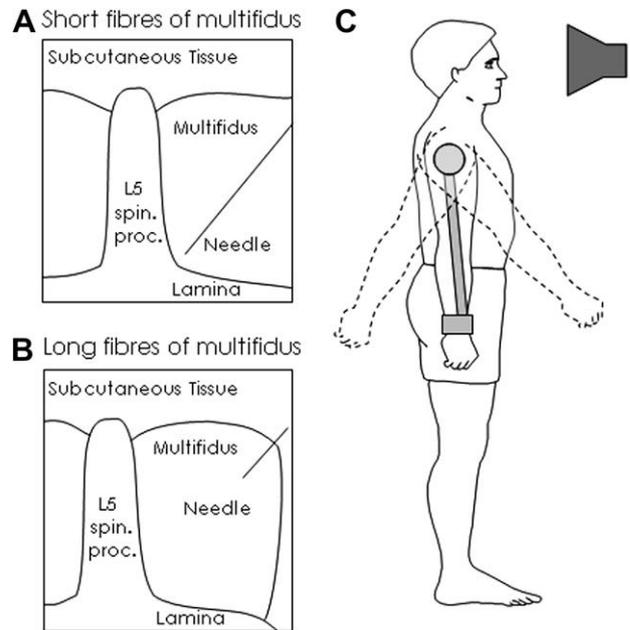
Fig. 1. Experimental Method: Line drawings depict the electrode placement into the (A) short and (B) long fibres of the lumbar multifidus adjacent to the lamina of L5. (C) Subjects performed a choice reaction time task (rapid shoulder flexion or extension) in response to an auditory cue.

discomfort during the insertions and if any significant discomfort was reported after the removal of the needle, the electrode was removed and a new electrode was inserted. This was done to ensure that participants were pain-free during the experiment.

Pairs of surface electrodes (Ag/AgCl discs, 10 mm and 5 mm in diameter, with an interelectrode distance of ∼20 mm) were placed over the anterior deltoid muscle and posterior deltoid muscles of both arms. The ground electrode was placed over the right iliac crest. EMG data were amplified 2000 times, band pass filtered between 30 Hz and 1 kHz, and sampled at 2 kHz using a Power 1401 and Signal software (Cambridge Electronic Design, Cambridge, United Kingdom). Data were exported for the analysis with Matlab 6.5 (Mathworks, Natic, MA, USA).

2.3. Upper limb movements

Angular displacement of the right and left arms was measured using a potentiometer attached to a lightweight bar, which was strapped to the arm at wrist level so as not to restrict the movement [20]. The axis of rotation of the bar was aligned with the estimated axis of rotation for flexion and extension of the glenohumeral joint. Movement data were recorded to confirm that the required arm movement distance was achieved.

2.4. Procedure

Participants stood relaxed with their arms by their sides. They were instructed to rapidly flex or extend at the shoulder to ∼45°, with their elbow straight, in response to an auditory cue (Fig. 1C). Tones of a different pitch were used to cue for flexion and extension. Subjects were instructed to react as quickly as possible to the cue by rapidly moving their arm in the correct direction. In between trials, subjects were told "relax, and wait for the cue". The auditory cue occurred randomly, between 0 and 1.25 s, after the investigator activated a manual trigger. Several practice trials were performed prior to data collection to ensure that the reaction time was consistent, and that the correct movement of
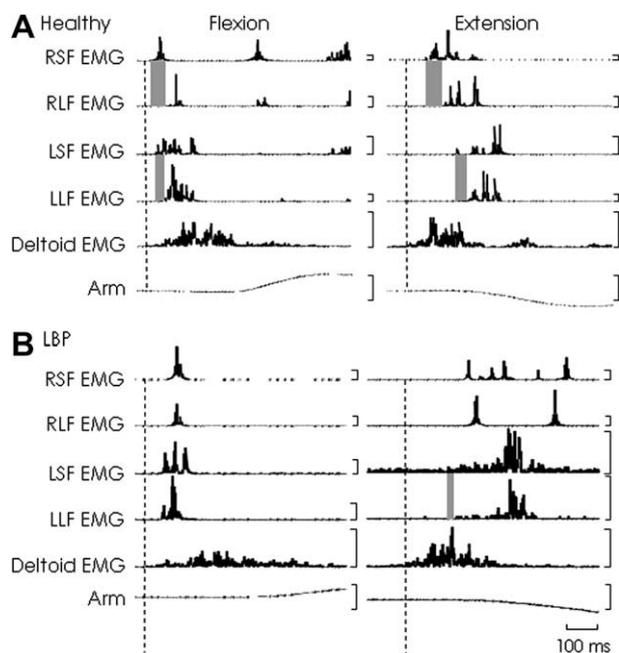
Fig. 2. Rectified electromyographic (EMG) data from a representative (A) healthy and (B) low back pain (LBP) participant (right-sided LBP) during arm flexion and extension. EMG data from the right and left short fibres of multifidus (RSF, LSF), right and left long fibres of multifidus (RLF, LLF), left anterior deltoid (during flexion trial) and left posterior deltoid (during extension trial) are presented. The dashed vertical line represents the onset of deltoid EMG. The grey boxes indicate the differences between the EMG onset of the short and long fibres of the lumbar multifidus. (*Note.* The EMG calibration for the control group – 100 μV except LDM 1 mV; EMG calibration for the LBP group – 100 μV except LDM 25 μV; angle calibration – 50°.)

the arm was performed in response to each auditory cue. The direction of movement was randomized and counterbalanced such that fifteen repetitions were performed in each direction for each arm.

2.5. Data analysis

The onset of EMG for each muscle was visually identified as the point at which activity increased above the baseline. EMG onset

Fig. 3. The mean latency of short (SF) and long fibres (LF) of the lumbar multifidus EMG onsets relative to the onset of activity in the deltoid muscle. Data for the healthy (open symbols) and LBP participants (filled symbols) during shoulder flexion (circles) and extension (squares) are shown. Error bars represent 95% confidence intervals. The dashed vertical line represents the onset of deltoid EMG (*$p < 0.05$).

detection was aided by the ability to visualize single motor units and therefore to detect the recruitment of new motor units as an indicator of the onset of EMG activity. Recordings were displayed in a random order, without reference to other muscles or events. The investigator was blind to the identity of the muscle being evaluated. There was no difference in the different intramuscular recordings that could alert the investigator to the identity of the muscle. This method of EMG analysis is reliable and valid [17,32].

The EMG onset of the short and long fibres of the lumbar multifidus, relative to that of deltoid, were used for analysis. Trials were excluded if the onset of back muscle activity occurred either 100 ms before or 200 ms after that of deltoid, because activations outside of those times are unlikely to be related to the arm movement [1]. Any data point greater than 2 SD from the mean was considered an outlier, and was removed from the analysis. Approximately 7% of trials were excluded.
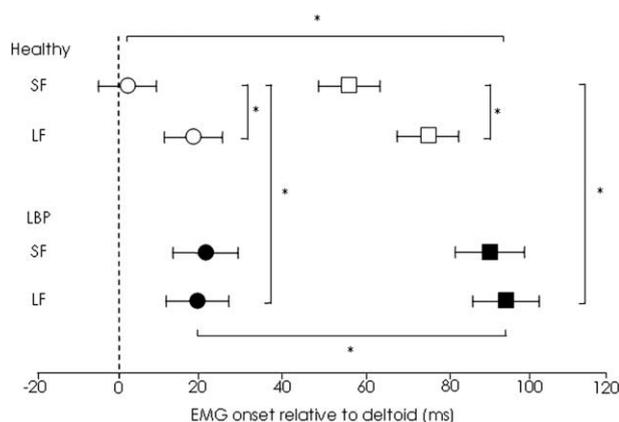
2.6. Statistical analysis

The onsets of the short and long muscle fibres, relative to that of deltoid, were compared between Groups (independent variable; patients and controls), Muscles (independent variable; short and long), Direction (repeated measures factor; flexion and extension) and Side (repeated measures factor; ipsilateral and contralateral to the moving arm). An initial multivariate analysis of variance (MANOVA) with an additional factor of Arm (left and right) showed no main effect ($p = 0.097$), so data from the left and right arms were pooled. There was also no main effect for Side (left and right) ($p = 0.674$) in the control group, so these data were also pooled in the main analysis.

To test the hypothesis that muscle activity is different between patients in remission and controls, we undertook a 2×2 MANOVA with two repeated measures factors: Side (ipsilateral and contralateral to the moving arm) and Direction (flexion and extension), and two independent variables: Muscle (short and long) and Group (patient and control). To test the hypothesis that the differences in muscle activity would be greater on the previously painful side than on the non-painful side in the patient group, we undertook a MANOVA with Pain Side (previously painful and non-painful) and Muscle (short and long) as independent variables and with Direction (flexion and extension) as a repeated measures factor. Post hoc testing was undertaken with the Duncan's multiple-range test. Significance was set at $\alpha = 0.05$.

3. Results

3.1. Short and long muscle fibre EMG during arm movements

When participants in the control group rapidly flexed or extended their arm, the onset of EMG was earlier for the short muscle fibres than it was for the long muscle fibres (Interaction: Group - Muscle, $p = 0.022$; post hoc, $p = 0.0008$) (Figs. 2 and 3 and Table 1). When participants in the patient group performed the

Table 1
Analysis of variance for comparison between LBP and healthy participants.

	D of F	F values	P values
Group	1	8.9	0.003
Muscle	1	6.7	0.011
Direction	3	146.9	0.000
Group × Muscle	1	5.4	0.022
Direction × Group	3	3.7	0.011
Direction × Muscle	3	0.4	0.757
Direction × Group × Muscle	3	0.1	0.984

D of F – degrees of freedom.

Table 2
Analysis of variance for comparison between pain sides in the LBP participants.

	D of F	F values	P values
Pain	1	0.5	0.476
Muscle	1	1.4	0.244
Direction	3	38.9	0.000
Pain × Muscle	1	4.4	0.045
Direction × Pain	3	2.4	0.076
Direction × Muscle	3	1.0	0.419
Direction × Pain × Muscle	3	1.6	0.200

D of F – degrees of freedom.

same task, there was no difference in the onset of the short and long muscle fibres with either flexion or extension (Interaction: Group × Muscle, $p = 0.022$; post hoc, $p = 0.850$) (Figs. 2 and 3 and Table 1). In both groups, the onset of short and long muscle fibres EMG was earlier with flexion of the arm than with extension (main effect: Direction, $p < 0.0001$) (Figs. 2 and 3 and Table 1).

3.2. Comparison of short and long muscle fibre EMG between groups

The onset of short muscle fibre EMG in the patient group was later than it was in the control group, for both shoulder flexion and extension (Interaction: Group × Muscle $p = 0.022$; post hoc, $p = 0.0005$) (Figs. 2 and 3 and Table 1). There was no difference in the onset of the long muscle fibres between the groups (post hoc: $p = 0.661$) (Figs. 2 and 3).

3.3. Difference between previously painful and non-painful side in patient group

The short and long muscle fibres were active earlier with arm flexion, and later with arm extension, on both the previously painful and non-painful sides (main effect: Direction, $p < 0.0001$, Table 2). On the non-painful side, the onset of short muscle fibre EMG was earlier than the onset of long muscle fibre EMG (Interaction: Pain × Muscle, $p = 0.045$, post hoc: $p = 0.040$, Table 2), regardless of the direction of the arm movement (Pain × Muscle × Direction: $p = 0.200$) (Fig. 4 and Table 2). However, there was no significant difference between the onset of short and long muscle fibre EMG on the previously painful side (post hoc: $p = 0.528$) (Fig. 4). That is, the earlier onset of short muscle fibre activity, compared to long,

Fig. 4. Comparison of the mean short (SF) and long fibres (LF) of the lumbar multifidus EMG onsets relative to deltoid collapsed across directions between the painful (black circles) and non-painful sides (white circles) in people with recurrent unilateral LBP. Error bars represent 95% confidence intervals. The dashed vertical line represents the onset of deltoid EMG (*$p < 0.05$).

observed in the control subjects and on the non-painful side in the patient group, was not found on the patient's previously painful side.

4. Discussion

We investigated the control of the short and long fibres of a deep back muscle in people with recurrent unilateral LBP who were pain-free at the time of testing, and a group of healthy participants, by recording intramuscular EMG during a postural task. We hypothesized that changes in motor control would primarily affect the short fibres of the deep back muscle, and would be greater on the previously painful side than on the non-painful side. The results support both hypotheses. The EMG onset of the short fibres of the deep back muscle relative to the arm muscle, deltoid, occurred later in people with recurrent LBP than in healthy participants. The observed delay was greater on the previously painful side than on the non-painful side. Furthermore, the timing of the muscle activity (short fibres active before long fibres) that was observed in the healthy participants and on the non-painful side in the patient group was not present on the previously painful side. These changes in the control of the deep back muscles are consistent with the findings of morphological studies [12,49] that suggest that the changes in the back muscles in people with LBP are more profound in the short fibres and on the side of symptoms.

A change in the control of the deep back muscles is important, because it is likely to have consequences for spinal function. Control of movement and stability has been argued to be critical for healthy function of the spine [39,40]. The deep back muscles investigated in this study have been reported to contribute up to 2/3 of the control of lumbar intersegmental motion [48], which suggests that impaired control of these muscles is likely to compromise spinal function. The persistence of these changes during remission of LBP, as identified here, implies persistent altered loading on spinal structures during remission, which may be a cause of recurrent episodes. Longitudinal studies are required to confirm this.

Delayed activation of the deep back muscles, and failure of the short fibres to activate prior to the long fibres, could be mediated by changes at any level of the motor system – from the motoneurone to the motor planning. At the spinal level, there is some evidence that pain can inhibit extensor motoneurone activity in animals [25]. However, it is not clear whether this occurs in humans, or whether it persists after the resolution of symptoms. At the cortical level, experimentally induced muscle pain has been shown to reduce the excitability of the primary motor cortex [27]. Furthermore, the recent data from an animal study [13] suggest that spinal injury specifically reduces excitability of cortico-motor inputs to the short fibres of the lumbar multifidus. However, it is unclear whether this change in excitability underlies the changes in multifidus activation observed here.

Alternatively, changes in timing of muscle activation, such as those observed here, may reflect changes in motor planning [16,33]. Such changes in planning could be due to inaccurate [29] or ignored [6] sensory information from the spine, or to a change in strategy adopted by the nervous system. It has been argued that people with LBP may prioritize patterns of muscle activation in an attempt to avoid pain provocation [18,45]. Several authors have hypothesized that increased load either due to increased paraspinal muscle activity (including strategies to protect the spine [18]or due to physical activity [5] or sustained postures [10]) may lead to persistence or recurrence of symptoms. However, the patients in this study adopted patterns of muscle activation that were accompanied by reduced activity of the deep back muscles, that could have the cost of decreased 'fine tuning' of segmental motion [22] and may be related to recurrence. Consistent with
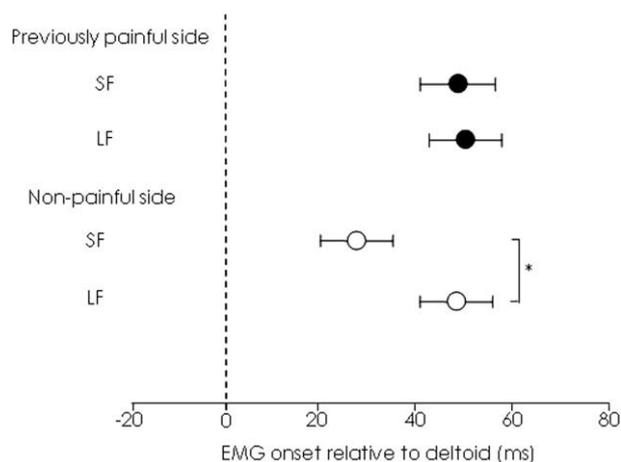
other work [45], it is possible that the activity of the other large trunk muscles may have been increased in our participants as a component of an alternative protective strategy. It is important to consider that a person's beliefs about their back pain [34] and their expectations of future episodes [35] can also influence the type of motor strategy they use to control the back muscles. That is, deep back and abdominal muscle EMG is delayed when healthy participants anticipate experimentally induced LBP [35]. Furthermore, those changes did not spontaneously resolve in some people, which were related to unhelpful beliefs about back pain [34]. The current finding corroborates that work and suggests that an alternative postural strategy, and subsequently changes in control of the deep back muscles, could be adopted by some individuals following the initial bout of LBP that remain despite the resolution of symptoms [19]. Such a change in strategy may indicate ongoing problems as alterations in control of the back muscles lead to changes in joint loading and the dynamic properties of the spine [15]. However, further work is needed to determine whether these factors underlie the changes in multifidus activation observed here.

Interpretation of the current findings requires consideration of several limitations. Whether insertion of intramuscular electrodes has an effect on the activity of the paraspinal muscles has not been unequivocally established. Although others have argued that insertion of electrodes does not change coordination [21], this has not been evaluated in people with LBP. Performance of a choice reaction task during stressful conditions can affect the pattern of back muscle activation [36], but we endeavoured to minimise stress, participants did not appear stressed, and they were given positive feedback about their performance. Although it is clear that the study was adequately powered to detect a difference in short fibres of the lumbar multifidus, we may not have had sufficient numbers to be confident that the long fibres did not differ between the groups. However, this does not affect the main conclusion of our paper that people in remission from LBP have persistent changes in back muscle activation. Although important, these limitations do not compromise the main findings of the study.

This study has clear clinical implications. First, it is clear that resolution of back pain does not imply a return to normal control of the deep back muscles. This finding corroborates data that show that reduced cross-sectional area of the deep back muscles remains in some patients following an acute episode of LBP despite the resolution of symptoms [12]. Notably, a clinical trial suggested that therapeutic exercise designed to improve the control of the deep trunk muscles in people with acute/subacute LBP can both restore the symmetry of the cross-sectional area of the back muscles and reduce recurrence [11]. Furthermore, a similar therapeutic exercise programme reduced pain and improved functional measures in patients with chronic low back pain and a radiologic diagnosis of spondylolysis or spondylolisthesis [37]. Second, it is clear that spinal dysfunction is associated with these changes in muscle morphology and control. Using an animal model, rapid segmental atrophy of the lumbar multifidus has been observed following experimentally induced injury to the lumbar intervertebral disc [14]. However, it remains to be determined if changes in morphology and control contribute to persistence or recurrence of pain. Third, the current findings also suggest that the sole foci on symptoms and functional performance as outcome measures following an acute episode of LBP need to be reconsidered. Perhaps a clinically viable measure of back muscle control is required as an outcome measure of recovery following an acute episode of LBP. Preliminary investigation suggests that high resolution ultrasound imaging may be useful in this regard [24], but further work is required.

In summary, the current experiment presents evidence that even though they are pain-free and thus between episodes of LBP, recurrent unilateral LBP patients do not control their back muscles in the same way as their healthy counterparts. These findings raise the possibility that this abnormal pattern of muscle control, in the absence of pain, may leave the spine vulnerable to (re)injury and hence predispose to recurrent episodes. Finally, this finding implies that pain and functional performance should not be the only outcome measures of interest after an acute episode of LBP.

Conflicts of interest

There are no conflicts of interest associated with this study.

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