The effect of experimental low back pain on lumbar muscle activity in people with a history of clinical low back pain - a muscle functional MRI study

Running head: Experimental LBP during remission of recurrent LBP

Danneels Lieven, Cagnie Barbara[1], D'hooge Roseline[1], De Deene Yves[2], Crombez Geert[3], Vanderstraeten Guy[1,4], Parlevliet Thierry[5], Van Oosterwijck Jessica[1]

[1] Department of Rehabilitation Sciences and Physiotherapy, Ghent University, Campus Heymans, De Pintelaan 185, B-9000 Ghent, Belgium

[2] Department of Radiotherapy and Experimental Cancer Research, Ghent University Hospital, De Pintelaan 185, B-9000 Ghent, Belgium

[3] Department of Experimental Clinical and Health Psychology, Ghent University, Henri Dunantlaan 2, B-9000 Ghent, Belgium

[4] Department of Physical and Rehabilitation Medicine, Ghent University Hospital, De Pintelaan 185, B-9000 Ghent, Belgium

[5] Department of Physical Medicine and Orthopedic Surgery, Ghent University Hospital, De Pintelaan 185, B-9000 Ghent, Belgium

Correspondence and reprint requests to Prof. Lieven Danneels, Ghent University Hospital, Dept. of Rehabilitation Sciences and Physical Therapy, De Pintelaan 185, 1B3, B-9000 Ghent, Belgium. Tel: +32 9 332 26 32, Fax: +32 9 332 38 11, E-mail: Lieven.Danneels@Ugent.be

ABSTRACT

In people with a history of low back pain (LBP), structural and functional alterations have been observed at several peripheral and central levels of the sensorimotor pathway. These existing alterations might interact with the way the sensorimotor system responds to pain. We examined this assumption by evaluating the lumbar motor responses to experimental nociceptive input of 15 participants during remission of unilateral recurrent LBP. Quantitative T2-images (muscle functional MRI) were taken bilaterally of multifidus, erector spinae and psoas at several segmental levels (L3 upper, L4 upper and lower endplate) and during several conditions: 1) at rest, 2) upon trunk-extension exercise without pain, and 3) upon trunk-extension exercise with experimental induced pain at the clinical pain-side (1.5ml intramuscular hypertonic saline injections in erector spinae). Following experimental pain induction, muscle activity levels similarly reduced for all 3 muscles, on both painful and non-painful sides, and at multiple segmental levels ($p=0.038$). Pain intensity and localization from experimental LBP were similar as during recalled clinical LBP episodes. In conclusion, unilateral and unisegmental experimental LBP exerts a generalized and widespread decrease in lumbar muscle activity during remission of recurrent LBP. This muscle response, is consistent with previous observed patterns in healthy people subjected to the same experimental pain paradigm. It is striking that similar inhibitory patterns in response to pain could be observed, despite the presence of pre-existing alterations in the lumbar musculature during remission of recurrent LBP. These results suggest that motor output can modify along the course of recurrent LBP.

Key words: recurrent low back pain; experimental muscle pain; muscle functional magnetic resonance imaging; lumbar paraspinal muscles; muscle recruitment

INTRODUCTION

Low back pain (LBP) is related to substantial reorganization of motor control strategies which are assumed to protect from further injury or pain (Hodges et al. 2003, 2011). It is believed that these motor alterations can persist after resolution of a LBP episode (Hides et al. 1996; Hodges et al. 2011; Macdonald et al. 2009). Long-term persistence of altered recruitment strategies has been hypothesized to have negative consequences for spinal health through suboptimal load sharing, reduced spinal movement and/or reduced variability in muscle recruitment strategies (Hodges et al. 2011). Therefore, further insight in the causal role of LBP in relation to lumbar muscle dysfunction is important to administer appropriate rehabilitation and prevent recurrence of LBP.

Experimental pain models have been applied to study the causal effect of peripheral nociception on motor output (Graven-Nielsen et al. 2000, 2006). Previous studies have demonstrated altered muscle behavior during experimental LBP in healthy people (Arendt-Nielsen et al. 1996; Dickx et al. 2008, 2010; Hodges, et al. 2003; Kiesel et al. 2008; Zedka et al. 1999), and its effects were also shown to be comparable to findings observed in clinical LBP (Graven-Nielsen 2006). However, changes in motor output in relation to clinical LBP not only depend upon peripheral nociceptive stimuli, but are the net resultant of a complex interaction at multiple levels along the sensory, central and motor nervous system (Hodges et al. 2003, 2011).

People with a history of clinical recurrent LBP have demonstrated several structural and functional alterations which are situated at multiple peripheral and central levels along the sensorimotor pathway. Compared to healthy controls, divergences in motor output during a variety of lumbar tasks (D'Hooge et al. 2013; Jones et al. 2012; Macdonald et al. 2009, 2010

2011) and in lumbar muscle structure (D'Hooge et al. 2012; Hides et al. 1996) were present subsequent to resolution of LBP. In addition, the cortical representation of specific lumbar muscles appeared to be reorganized (Tsao et al. 2011), and changes at the proprioceptive level (Brumagne et al. 2000) have been described, during remission of LBP. Applying an experimental pain paradigm during remission of clinical LBP offers the possibility to investigate whether and how existing alterations related to clinical LBP interact with muscle behavior in response to acute pain.

To determine if people who have had clinical pain before respond to acute pain in the same manner as healthy people, an established experimental low-back-pain paradigm will be replicated in a participant sample with a history of clinical low back pain. Previously, lumbar muscle activity has been investigated using muscle functional Magnetic Resonance Imaging (mfMRI) in healthy people with and without experimental induced LBP (Dickx et al. 2008). MfMRI is an innovative, post-exercise, evaluation method to assess the amount of metabolic muscle activity by quantifying shifts in T2-relaxation times of muscle water upon exercise (Cagnie et al. 2011; Meyer and Prior 2000). Published results in healthy people showed that muscle activity during trunk-extension significantly decreased in multifidus (MF), erector spinae (ES) and psoas (PS) at both body sides and multiple segmental levels, in response to unilateral and unisegmental experimental pain (Dickx et al. 2008). The same study set-up, has been used to demonstrate pre-existing dysfunctions in people in remission of recurrent LBP. Specifically, this population showed increased MF activity during trunk-extension on both body sides and at multiple levels compared to healthy controls, while no changes were evident in ES or PS activity (D'Hooge et al. 2013).

Therefore, the aim of the current study was to investigate lumbar motor responses to experimental nociceptive input in people with a pre-existing condition of the sensorimotor system due to a previous clinical history of recurrent LBP.

MATERIALS AND METHODS

Participants

Fifteen people (6 males, 9 females) with a history of unilateral, non-specific, recurrent LBP and aged between 20 and 55 years were recruited via advertisement from the local community and university setting. Volunteers were included when having at least 2 previous LBP episodes that interfered with daily functioning and/or required treatment (first onset LBP at least 6 months before) of which at least 2 episodes took place in the past 12 months (Stanton et al. 2010). An episode was defined as pain lasting for minimum 24 hours, preceded and followed by at least 1 month without LBP (de Vet et al. 2002). Testing was scheduled at least 1 month after resolution of the last LBP episode. The characteristics of participants their LBP history including duration since first onset of LBP (months), frequency of episodes per year, mean duration of an episode (days), mean duration of the last experienced episode (days), pain intensity (pain NRS 0-100), and disability during episodes (disability NRS 0-100), and time since last episode (days) were determined using a custom-designed questionnaire and the results are reported in Table 1.

Exclusion criteria were central, bilateral or side-variable localization of LBP; specific LBP; participation in lumbar motor control training in the previous year; spine surgery; spinal deformities; task-limiting medical conditions or contra-indications for MRI (ferromagnetic/electronic implants that could be moved/affected by a magnetic field e.g. pacemaker, aneurysm clip, etc.; claustrophobia; (possible) pregnancy).

All participants were informed of the study procedures, approved by the local Ethics Committee, and provided written informed consent. The findings from this study sample have not been published previously.

General experimental design

MRI-images were obtained under 3 consecutive conditions (Dickx et al. 2008): 1) at rest (T2-rest) after 30min of supine lying, 2) immediately following exercise without pain (T2-exercise), and 3) immediately following exercise performed with experimental pain (T2-exercise+pain). Between the second and third condition, participants rested supine for 60min to regain the resting metabolic state of the muscles (Cagnie et al. 2011).

Exercise protocol

Ten consecutive repetitions of a low-load, static-dynamic trunk extension were performed. Participants were positioned prone on a variable angle chair in 45° of trunk flexion, with their hands placed on the ipsilateral shoulders. One repetition consisted of extending the trunk in line with the legs to a horizontal position (2sec), maintain the trunk horizontally (5sec), and then lowering the trunk again (2sec) to the starting position. The exercise load was individually adjusted to 40% of 1-RM (one repetition maximum). Because the calculated weight of the exercise load was lower than the weight of the trunk, the body was assisted via a load-pulley system. Details of the exercise protocol and methods for calculating the individual exercise load are identical as described in previous studies (D'Hooge et al. 2013; Dickx et al. 2008, 2010). The individual 1-RM was indirectly determined, as described in those studies, on a separate day which took place at least 7 days prior to the experiment.

Muscle functional MRI

MfMRI has been validated and proven complementary to surface-electromyography (EMG) for assessing the amount of lumbar muscle activity during trunk-extension (Dickx et al. 2010). A 3-Tesla MRI-scanner (Magnetom Trio-Tim, Syngo MR VB13 software, SIEMENS AG®, Erlangen Germany) was used for imaging. Participants laid supine, with a foam wedge supporting the legs and ensuring a neutral spinal curvature. A flexible 6-element body-matrix coil, centered on L4 ventrally, was combined with the standard phased-array spine coil dorsally as a receiver-coil combination.

Three axial slices were planned from a sagittal localizing sequence with respect to vertebral inclination along the upper endplate of L3 and L4, and the lower endplate of L4 (Figure 1A). The lumbar MF, ES and PS were visualized.

T2-weighted images were acquired with a spin-echo multi-contrast sequence (SE_MC) with the following parameters: repetition time (TR) 1000ms, echo train of 16 echoes ranging from 10.1 to 161.6ms with steps of 10.10ms, acquisition matrix 256*176mm^2, field of view (FOV) 340mm, voxel size 1.3*1.3*5.0mm^3, scan-time 5min52s.

Experimental pain

Acute experimental LBP was induced by injecting a bolus of 1.5ml of hypertonic saline (5% NaCl) in the lumbar ES (4cm lateral from the L4 spinous process, at a depth of 2.5cm) (Dickx et al. 2008) of that side of the body in which participants had reported their natural unilateral clinical recurrent LBP to occur. Thirty seconds after pain induction, participants verbally rated the pain intensity induced by the injection of hypertonic saline using a pain numeric rating scale (NRS). Scores from this scale ranged from 0 (no pain) to 100 (worst possible

pain). If the subject reported a score below 40/100, an additional bolus of 0.5ml was injected. During the exercise, pain intensity was monitored by asking participant an NRS rate 1) before the 1st repetition, 2) after the 5th repetition and 3) after the 10th repetition of trunk extension. Upon completion of the experiment, pain localization was indicated on a pain diagram.

Psychological exercise measures

To not influence participants their pain experience they were informed that the injection of hypertonic saline would induce pain, but no information was given regarding the expected severity or localization of the induced pain. As participants had performed the trunk extension exercises during the pre-screening, in order to determine their individual 1-RM, they were familiar with these exercises which were repeated on the day of the experiments. Nonetheless, before each exercise condition, fear of exercise performance was rated on a NRS from 0 (not fearful at all) to 100 (extremely fearful). Similarly, fear of needle/injection and fear of experimental pain were rated prior to the saline injection (Dickx et al. 2008). After each exercise condition, experienced pain intensity during exercise (NRS, 0-100) and perceived exertion (RPE) (Borg-scale, 15-20) (Borg 1982) were rated. Additionally, participant rated the perceived similarity between experimental LBP and natural clinical LBP on a NRS from -100 (not similar at all) to +100 (completely identical) with 0 representing similar.

Data analysis

Images were analyzed using ImageJ (v. 1.41o, Java-based version of the public domain NIH Image Software, USA; Research Services Branch). For each of the 3 conditions and segmental levels, a quantitative T2-map was calculated using the MRI analysis T2-calculator, with a T2-value (ms) assigned to each voxel. The first of 16 echoes was excluded for reasons

of better curve fitting (De Deene et al. 2000). Regions of interests (ROI's) were traced on the T2-maps along the muscular borders of MF, ES and PS bilaterally (Figure 1B), excluding visual fat, blood vessels or connective tissue. For each ROI, the mean T2-value was calculated. Image processing was performed blinded to condition and pain-side. Then, T2-shifts were calculated as the difference between T2-exercise (with and without pain) and T2-rest.

Statistical analysis

Analyses were performed using SPSS (v19, IBM Statistics). Descriptive statistics (means and standard deviation [SD]) were calculated for the participants' characteristics and T2-values. Paired samples t-tests were used to compare fear, RPE and pain intensity between the exercise condition with and without pain, and between pain intensity experienced from experimental pain and pain intensity recalled from natural recurrent LBP episodes.

A general linear model (GLM) with repeated measures was used to examine T2-results. To investigate which muscles were activated during the trunk-extension exercise, the difference between the T2-rest and T2-exercise was tested for each muscle separately (because of interaction effect for 'condition*muscle': $p=0.004$) with within-subject factors 'condition' (T2-rest, T2-exercise), 'level' (L3 upper, L4 upper, L4 lower) and 'side' (painful side, non-painful side). To investigate the effect of experimental LBP on T2-shift, within-subjects factors were 'condition' (T2-shift exercise, T2-shift exercise+pain), 'muscle' (MF, ES, PS), 'level' (L3 upper, L4 upper, L4 lower) and 'side' (painful side, non-painful side).

Moreover, pearson correlation coefficients were calculated to investigate whether the decrease in muscle activity (delta T2-shift) in the pain condition correlated with increased fear (delta fear of exercise performance) or with changes in pain intensity (delta pain intensity).

Post-hoc comparisons were made when required and were adjusted using Bonferroni-correction. Statistical significance was accepted at $\alpha = 0.05$.

RESULTS

Mean T2-values in rest, exercise-without-pain and exercise-with-pain condition are presented in Table 2.

Effect of trunk-extension on T2-values

T2-values were significantly higher in the exercise condition (without pain) compared to the resting condition for MF ($p<0.001$) and ES ($p=0.003$), but not for PS ($p=0.281$) (Figure 2). There were no differences in T2-values between the previously painful and non-painful side (main effect 'side': MF $p=0.541$; ES $p=0.466$; PS $p=0.738$). There were no interaction effects for condition with 'level' or 'side' ($p>0.05$).

Effect of experimental LBP on T2-shift

T2-shift was significantly lower in the exercise-with-pain compared to the exercise-without-pain condition for all muscles (main effect 'condition' $p=0.038$) (Figure 3). For both conditions, T2-shift was significantly higher in MF compared to ES ($p=0.041$) and compared to PS ($p=0.002$), but was not significantly different between ES and PS ($p=0.244$) (main effect 'muscle' $p=0.001$) (Figure 3). No main effects for 'level' ($p=0.638$) or 'side' ($p=0.525$), and no interaction effects for condition with 'level' or 'side' were found ($p>0.05$).

Psychological exercise measures

Following saline injection, mean NRS pain intensity was 57±18 before the 1st repetition, 56±22 after the 5th repetition, and 54±23 after the 10th repetition of trunk extension. Total pain intensity experienced from experimental LBP during performance of the exercise (NRS=52/100) was not different from self-reported pain intensity recalled from recurrent LBP episodes (NRS= 57/100) (p=0.391).

Scores for fear of performance of the exercise, experienced pain and RPE (Table 3), were significantly higher in the exercise-with-pain versus the exercise-without-pain condition.

Upon completion of the experiment pain diagrams were used to localize the experienced pain elicited through pain induction. Interpretation of these diagrams revealed that 9 people reported focal unilateral paraspinal pain as a consequence of the experimental pain induction, from which 6 reported to have local pain during their natural episodes. The other 6 participants reported referred pain in the gluteal region, groin or posterior thigh (not below the knee), all of these were among the 9 persons who experienced referred pain during their natural episodes. None of the participants reported a more expanded region of pain.

The amount of inhibition in muscle activity was not correlated to the magnitude of pain intensity (r=0.103, p=0.749). A trend towards significance (r=0.533, r^2=0.284, p=0.074) indicated a weak association with muscle inhibition and fear of pain (delta NRS for fear of exercise performance: mean=-31, range=-90 to 0).

DISCUSSION

This study investigated the effect of experimental nociception on lumbar muscle activity during trunk-extension in people in remission of clinical recurrent LBP. During the experimental pain condition, muscle activity significantly decreased for all 3 evaluated

muscles (MF, ES and PS), equally at the painful and non-painful side at all 3 segmental levels.

This inhibitory response pattern was consistent with previously published results in healthy controls which were obtained with an identical study set-up (Dickx et al. 2008). Similarly, another study in healthy subjects reported decreased ES EMG activity during standing trunk re-extension following experimental pain (Zedka et al. 1999). Studies evaluating ES EMG activity during trunk extension in people with clinical (not experimental) LBP reported a decrease (Shirado et al. 1995; Watson et al. 1997), others an increase (Descarreaux et al. 2007) or no difference (Lariviere 2000) compared to healthy controls. Apparently, comparing changes in lumbar muscle activity between clinical LBP and healthy controls yielded more variable results versus comparing muscle activity with and without experimental LBP. This might be consistent with the proposition that alterations in motor output in clinical LBP do not solely depend on muscular nociceptive mechanisms or other possible sources of spinal nociception (e.g. disc, ligament, zygapophyseal joints, nerve root, etc.) (Deyo and Weinstein 2001), but also on other existing alterations along the sensorimotor system in relation to clinical LBP.

It has been postulated previously that pain yields a generalized, widespread effect, affecting recruitment of several muscles, sides and segmental levels (Ciubotariu et al. 2004; Dickx et al. 2008, 2010). In the present study, activity was reduced in all 3 measured muscles despite administration of pain took place in ES only and synergistic activation of MF and ES but not PS occurs during trunk-extension. Nevertheless, concurrent inhibition of all 3 muscles might be attributed to the fact that deep stabilizing muscles are more likely to be affected by pain compared to superficial torque-generating muscles (Hodges et al. 2003). Analogous to MF

and lumbar ES, evidence exists for the role of PS as a spinal stabilizer because of its segmental connections (Hansen et al. 2006). These alterations in motor output in response to pain have been postulated as an adaptive strategy, ultimately aiming to avoid further pain or injury (Hodges et al. 2011). In addition, the trend towards a weak association between inhibition of muscle activity and the increase in fear for exercise-performance during the pain condition, might support the contemporary idea that unfavorable pain-related cognitions can be involved in altering muscle recruitment patterns (Moseley and Hodges 2006).

Previously, several adaptations in motor output have been reported during remission of recurrent LBP (D'Hooge et al. 2013; Jones et al. 2012; Macdonald et al. 2009, 2010, 2011). A qualitative comparison of the systematic reduction in muscle activity following experimental LBP in this study, with the previously published pattern of pre-existing alterations during trunk-extension in remission of unilateral recurrent LBP (D'Hooge et al. 2013), demonstrates contrasting findings. During LBP remission, participants exhibited higher MF activity compared to healthy controls on both sides and segmental levels, without alterations for ES or PS (D'Hooge et al. 2013). Since different muscles are affected to a different extent and in opposite directions, the opposing patterns suggest that experimental LBP exerts a distinctive effect on lumbar muscle activity, which is observed over and above the existing alterations in lumbar muscle behavior during remission of recurrent LBP. Several factors might contribute to the opposing muscle activity patterns. A key feature of LBP remission is the absence of pain. Analogue to the restoration of recruitment strategies to a pre-pain state after experimental LBP (Moseley and Hodges 2005), it could be hypothesized that the inhibitory effects of nociception might have equally disappeared after resolution of clinical LBP. In addition to pain, injury-related mechanisms have been reported in relation to localized and

285 selective changes in MF structure in acute clinical LBP (Hides et al. 1994) and following an
286 experimental lumbar injury procedure in pigs (Hodges et al. 2006). In order to maintain spinal
287 functioning during LBP remission, lumbar muscle behavior might be compensating for
288 structural spinal deficits (e.g. increased activity in MF) (Panjabi 2003).
289
290 The current study was unique in administering experimental LBP at the site of previous
291 clinical LBP, instead of in healthy controls. In this way, muscle recruitment was investigated
292 intra-individually with and without pain, while accounting for the individuals' sensorimotor
293 pathway and biopsychosocial background, which had been relevantly influenced by a history
294 of LBP. The novelty of the current results is situated in that the results from a healthy control
295 group were replicated in a clinical population. It is striking that, in people with a history of
296 LBP the motor pattern in response to pain was similar as in healthy people, despite having a
297 pre-existing condition of the sensorimotor system. This pattern resemblance might indicate
298 that acute pain exerts a stereotypical, inhibitory effect on motor output. As such, these results
299 bring us a step forward towards our understanding of sensorimotor adaptions in relation to
300 pain, as motor responses to pain were studied in a more representative, clinical study sample.
301
302 With regard to pain measures, experimental pain intensity and psychometric scores of fear
303 were of similar order compared to those previously reported in healthy controls (Arendt-
304 Nielsen et al. 1996; Dickx et al. 2008, 2010; Hodges et al. 2003; Kiesel et al. 2008).
305 Experimental pain intensity and localization were comparable to their usual clinical LBP. In
306 contrast, people with chronic widespread pain reported enlarged areas of referred pain and
307 hyperalgesia in response to experimental pain as a result of central sensitization (Graven-
308 Nielsen and Arendt-Nielsen 2008). Although the experimentally induced LBP was not

perceived as completely identical to natural clinical LBP, the similarity between experimental and clinical pain was perceived within the positive range of the spectrum (NRS= +36 on scale from -100 to +100). Taken together, this is to our knowledge the first intra-individual evidence (cf. pain intensity and localization, perceived similarity) adding to the presumption that intramuscular injection of hypertonic saline can closely mimic clinical pain characteristics of acute LBP (Graven-Nielsen 2006). Nevertheless, recalling the intensity, distribution and type of LBP may not be evident for each participant. Furthermore, fundamental differences are situated within the perception of experimental compared to clinical LBP, since the experimental nociceptive stimulus is known not to be damaging and is controlled over a limited time-course (Graven-Nielsen 2006). These factors may reduce the affective-emotional component of pain.

The results should be viewed within the scope of the methodology. MfMRI depicts muscle activity post-exercise, hence other aspects of motor control, e.g. timing, cannot be considered. Also, imaging focused on 3 deep lumbar muscles. Since muscle activity decreased in these measured muscles, it is not known if redistribution of activity to other, superficial muscles occurred or if exercise performance altered during pain. Despite the lack of biomechanical data, movement velocity and range were controlled in a standardized way.

Furthermore, the exercise conditions were performed in a fixed order (first without pain, subsequently with pain) which introduces the possibility that sequential effects from the first exercise bout might compromise the second bout. Several arguments however might indicate that the effect of remaining fatigue would be minimal. The exercise was performed at low-load intensity (confer RPE score between very light and fairly light). The resting period in between the exercise bouts was prolonged to 60min, since it was not known if the standard

guidelines regarding recovery periods for T2-shifts on mfMRI (30-45min) (Cagnie et al. 2011) would equally apply to participants with musculoskeletal pain. Further, given that trunk-extension does not increase T2-values to an equal extent in the 3 measured muscles (T2-shift MF>ES>PS, Figure 3), it appears unlikely that T2-shift was homogeneously reduced in all 3 muscles in the experimental pain condition (confer no interaction effect muscle*condition p=0.336), if exercising muscles would not have recovered yet. Future studies could incorporate repeated baseline T2-rest measures in between the 2 exercise conditions to confirm that T2-shifts has recovered.

In addition, the current study did not control for possible mechanical effects from the injection. In healthy people, the effects of injections with isotonic saline in the lumbar region have been shown to be marginal compared to hypertonic saline (Hodges et al. 2003). Future research could confirm whether this holds in participants with a history of clinical LBP.

The study was conducted on a small number of participants because of the invasive character of the injections of hypertonic saline. The recruited numbers were in line with previous studies in this population (Macdonald et al. 2009, 2010, 2011) and previous studies using mfMRI (Dickx et al. 2008, 2010) and experimental pain inductions (Dickx et al. 2008, 2009). Nevertheless, due to the small sample size, caution is warranted towards extrapolation of the findings.

Finally, inclusion of a healthy control group would have allowed to directly compare the response to experimental pain and not only in a qualitative manner (recruitment patterns) with previous research, but also in a quantitative manner between participants with and without a history of clinical LBP.

The current findings might have some implications and perspective for further research. For now, it is assumed that adaptations fail to resolve following a LBP episode, resulting in ongoing alterations in muscle behavior during remission of LBP (Hides et al. 1996; Hodges et al. 2011; Macdonald et al. 2009). Since the current study shows immediate changes in muscle activity in response to pain in people with a history of recurrent LBP, opposite to the patterns observed during remission (=without pain), this might suggest that motor output can modify along the course of LBP. This encourages the need for further research to unravel the longitudinal course of muscle recruitment and the involved pathophysiological mechanisms during and after episodes of recurrent LBP.

In conclusion, administration of experimental LBP in people with a history of recurrent LBP effected a generalized, widespread inhibitory response in lumbar muscle activity during trunk extension. This response was consistent with previously established inhibitory patterns in healthy controls in response to acute pain, and appeared despite and in addition to the presence of pre-existing dysfunctions during remission of recurrent LBP. The response was opposite to the existing pattern of increased MF activity, which has been shown previously during remission of recurrent LBP. These results might suggest a potential pathophysiological role for pain in the modification of motor alterations along the course of recurrent LBP.

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DISCLOSURES

There are no conflicts of interests that may arise as a result of the research presented in this manuscript.

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FIGURE CAPTIONS

FIGURE 1 : Illustration of (A) sagittal localizer MRI scan of the lumbar spine indicating axial slice positioning and (B) T2-weighted axial MRI image at the level of L4 upper endplate demonstrating regions of interest bilaterally for multifidus, erector spinae and psoas.

FIGURE 2 : T2-values (in milliseconds, mean + SD; adjusted means for ‘side’ and ‘level’) in the resting (T2-rest) and the exercise condition without pain (T2-exercise) for multifidus, erector spinae and psoas.
Legends: * = $p < 0.05$

FIGURE 3 : T2-shifts (in milliseconds, mean + SD; adjusted means for ‘side and ‘level’) for the exercise in the non-pain (T2-shift Ex) and in the pain (T2-shift Ex+pain) condition for multifidus, erector spinae and psoas.
Legends: * = $p < 0.05$

519 TABLE CAPTIONS

520 TABLE 1 : Means ± SD for demographic and recurrent LBP characteristics of study
521 population

522 Legends: LBP = Low Back Pain; NRS = Numeric Rating Scale

523

524 TABLE 2 : Means ± SD of T2 values (in milliseconds) in the resting condition (T2-rest), in
525 the exercise condition without pain (T2-exercise) and in the exercise condition with pain (T2-
526 exercise+pain) for each muscle (multifidus, erector spinae, psoas), level (L3 upper, L4 upper,
527 L4 lower endplate) and side (painful, non-painful)

528

529 TABLE 3 : Means ± SD for psychometric exercise measures

530 Legends: LBP = Low Back Pain; NRS = Numeric Rating Scale; * = $p < 0.05$ between
531 exercise condition with and without pain