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

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Submitted 27 February 2015; accepted in final form 27 November 2015

LOW BACK PAIN (LBP) IS RELATED to substantial reorganization of motor control strategies, which are assumed to protect from further injury or pain (Hodges and Moseley 2003; Hodges and Tucker 2011). It is believed that these motor alterations can persist after resolution of a LBP episode (Hides et al. 1996; Hodges and Tucker 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 and Tucker 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; Graven-Nielsen 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 on peripheral nociceptive stimuli but are the net resultant of a complex interaction at multiple levels along the sensory, central, and motor nervous system (Hodges and Moseley 2003; Hodges and Tucker 2011).

People with a history of clinical recurrent LBP have demonstrated several structural and functional alterations that are situated at multiple peripheral and central levels along the sensorimotor pathway. Compared with 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 (fMRI) in healthy people with and without experimental
induced LBP (Dickx et al. 2008). MiMRI is an innovative, postexercise, 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 setup has been used to demonstrate preexisting 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 with 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 preexisting 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, nonspecific, recurrent LBP and aged between 20 and 55 yr were recruited via advertisement from the local community and university setting. Volunteers were included when having at least two previous LBP episodes that interfered with daily functioning and/or required treatment (first onset LBP at least 6 mo before) of which at least two episodes took place in the past 12 mo (Stanton et al. 2010). An episode was defined as pain lasting for minimum 24 h, preceded and followed by at least 1 mo without LBP (de Vet et al. 2002). Testing was scheduled at least 1 mo 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 numeric rating scale (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; and 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; and (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.** MRIs were obtained under three consecutive conditions (Dickx et al. 2008): 1) at rest (T2-rest) after 30 min 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 60 min 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 (2 s), maintaining the trunk horizontally (5 s), and then lowering the trunk again (2 s) to the starting position. The exercise load was individually adjusted to 40% of one-repetition maximum (1-RM). 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 before the experiment.

**Muscle functional MRI.** Muscle functional (mf)MRI 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, Erlangen, Germany) was used for imaging. Participants laid supine, with a foam wedge supporting the legs and ensuring a neutral spinal curvature. A flexible six-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 (Fig. 1A). The lumbar MF, ES, and PS were visualized.

T2-weighted images were acquired with a spin-echo multicontrast sequence (SE_MC) with the following parameters: repetition time (TR): 1,000 ms; echo train: 16 echoes ranging from 10.1 to 161.6 ms with steps of 10.10 ms; acquisition matrix: 256 × 176 mm²; field of view: 340 mm; voxel size: 1.3 × 1.3 × 5.0 mm³; and scan time: 5 min 52 s.

**Experimental pain.** Acute experimental LBP was induced by injecting a bolus of 1.5 ml of hypertonic saline (5% NaCl) in the lumbar ES (4 cm lateral from the L4 spinous process, at a depth of 2.5 cm) (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 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 0.5 ml was injected. During the exercise, pain intensity was monitored by asking participant an NRS rate (TR: 1,000 ms; echo train: 16 echoes ranging from 10.1 to 161.6 ms with steps of 10.10 ms; acquisition matrix: 256 × 176 mm²; field of view: 340 mm; voxel size: 1.3 × 1.3 × 5.0 mm³; and scan time: 5 min 52 s).

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 prescreening, 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 before 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 National Institutes of Health Image Software, 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 (ROIs) were traced on the T2 maps along the muscular borders of MF, ES, and PS bilaterally (Fig. 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 analyses.** Analyses were performed using SPSS (v19, IBM Statistics). Descriptive statistics (means ± 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 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, nonpainful side). To investigate the effect of experimental LBP on T2 shift, within-subject 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, nonpainful 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 α = 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 with the resting condition for MF (P < 0.001) and ES (P = 0.003) but not for PS (P = 0.281; Fig. 2). There were no differences in T2 values between the previously painful and nonpainful 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 with the exercise-without-pain condition for all muscles (main effect muscle).

**Table 2.** T2 values in the resting condition, exercise condition, and exercise condition with pain

<table>
<thead>
<tr>
<th>Muscle/Level/Side</th>
<th>T2-Rest</th>
<th>T2-Exercise</th>
<th>T2-Exercise + Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Multifidus</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L3 upper Pain</td>
<td>47.45 ± 4.30</td>
<td>51.91 ± 4.94</td>
<td>51.19 ± 6.35</td>
</tr>
<tr>
<td>Nonpain</td>
<td>47.46 ± 5.02</td>
<td>51.78 ± 3.47</td>
<td>50.82 ± 4.78</td>
</tr>
<tr>
<td>L4 upper Pain</td>
<td>44.77 ± 3.16</td>
<td>47.93 ± 4.32</td>
<td>46.45 ± 4.75</td>
</tr>
<tr>
<td>Nonpain</td>
<td>43.38 ± 2.33</td>
<td>47.04 ± 3.41</td>
<td>46.52 ± 4.13</td>
</tr>
<tr>
<td>L4 lower Pain</td>
<td>42.81 ± 2.07</td>
<td>45.52 ± 2.52</td>
<td>44.42 ± 3.17</td>
</tr>
<tr>
<td>Nonpain</td>
<td>42.59 ± 2.68</td>
<td>45.80 ± 2.80</td>
<td>44.76 ± 3.67</td>
</tr>
<tr>
<td><strong>Erector spinae</strong></td>
<td></td>
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</tr>
<tr>
<td>L3 upper Pain</td>
<td>43.04 ± 3.61</td>
<td>45.85 ± 2.96</td>
<td>45.36 ± 3.30</td>
</tr>
<tr>
<td>Nonpain</td>
<td>44.98 ± 3.14</td>
<td>47.78 ± 5.81</td>
<td>46.57 ± 5.24</td>
</tr>
<tr>
<td>L4 upper Pain</td>
<td>44.89 ± 3.63</td>
<td>46.73 ± 2.17</td>
<td>45.18 ± 3.92</td>
</tr>
<tr>
<td>Nonpain</td>
<td>44.75 ± 3.53</td>
<td>46.82 ± 4.42</td>
<td>46.47 ± 4.66</td>
</tr>
<tr>
<td>L4 lower Pain</td>
<td>47.86 ± 5.96</td>
<td>50.69 ± 6.28</td>
<td>48.40 ± 5.67</td>
</tr>
<tr>
<td>Nonpain</td>
<td>48.60 ± 5.37</td>
<td>51.55 ± 5.15</td>
<td>49.48 ± 6.02</td>
</tr>
<tr>
<td><strong>Psoas</strong></td>
<td></td>
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<tr>
<td>L3 upper Pain</td>
<td>40.84 ± 3.45</td>
<td>41.57 ± 2.49</td>
<td>40.07 ± 3.76</td>
</tr>
<tr>
<td>Nonpain</td>
<td>41.07 ± 3.15</td>
<td>42.14 ± 2.96</td>
<td>41.23 ± 3.95</td>
</tr>
<tr>
<td>L4 upper Pain</td>
<td>40.03 ± 2.64</td>
<td>40.86 ± 2.73</td>
<td>40.29 ± 2.97</td>
</tr>
<tr>
<td>Nonpain</td>
<td>40.58 ± 3.71</td>
<td>40.80 ± 3.04</td>
<td>40.81 ± 3.84</td>
</tr>
<tr>
<td>L4 lower Pain</td>
<td>39.60 ± 3.17</td>
<td>40.47 ± 3.06</td>
<td>39.71 ± 3.13</td>
</tr>
<tr>
<td>Nonpain</td>
<td>38.82 ± 2.95</td>
<td>40.35 ± 2.47</td>
<td>40.23 ± 3.56</td>
</tr>
</tbody>
</table>

Means ± SD of T2 values (in ms) in the resting condition (T2-rest), in the exercise condition without pain (T2-exercise), and in the exercise condition with pain (T2-exercise + pain) for each muscle (multifidus, erector spinae, psoas), level (L3 upper, L4 upper, L4 lower endplate), and side (painful, nonpainful).
was significantly higher in MF compared with ES (P = 0.002) and compared with PS (P = 0.074) but not significantly different between ES and PS (P = 0.244; main effect “muscle” P = 0.001; Fig. 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 vs. 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 nine people reported focal unilateral paraspinal pain as a consequence of the experimental pain induction, from which six reported to have local pain during their natural episodes. The other six participants reported referred pain in the gluteal region, groin, or posterior thigh (not below the knee), and all of these were among the nine 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, r² = 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 three evaluated muscles (MF, ES, and PS), equally at the painful and nonpainful side at all three segmental levels.

This inhibitory response pattern was consistent with previously published results in healthy controls that were obtained with an identical study setup (Dickx et al. 2008). Similarly, another study in healthy subjects reported decreased ES EMG activity during standing trunk reextension 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 with healthy controls. Apparently, comparing changes in lumbar muscle activity between clinical LBP and healthy controls yielded more variable results vs. 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 three 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 three muscles might be attributed to the fact that deep stabilizing muscles are more likely to be affected by pain compared with superficial torque-generating muscles (Hodges and Moseley 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 and Tucker 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. 2010). This inhibitory response pattern was consistent with previously published results in healthy controls that were obtained with an identical study setup (Dickx et al. 2008). Similarly, another study in healthy subjects reported decreased ES EMG activity during standing trunk reextension 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 with healthy controls. Apparently, comparing changes in lumbar muscle activity between clinical LBP and healthy controls yielded more variable results vs. 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.
A qualitative comparison of the systematic reduction in muscle activity following experimental LBP in this study, with the previously published pattern of preexisting 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 with 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. Analogous to the restoration of recruitment strategies to a prepain state after experimental LBP (Moseley and Hodges 2005), it could be hypothesized that the inhibitory effects of noception might have equally disappeared after resolution of clinical LBP. In addition to pain, injury-related mechanisms have been reported in relation to localized and selective changes in MF structure in acute clinical LBP (Hides et al. 1994) and following an experimental lumbar injury procedure in pigs (Hodges et al. 2006). To maintain spinal functioning during LBP remission, lumbar muscle behavior might be compensating for structural spinal deficits (e.g., increased activity in MF) (Panjabi 2003).

The current study was unique in administering experimental LBP at the site of previous clinical LBP, instead of in healthy controls. In this way, muscle recruitment was investigated intraindividually with and without pain, while accounting for the individuals’ sensorimotor pathway and biopsychosocial background, which had been relevantly influenced by a history of LBP. The novelty of the current results is situated in that the results from a healthy control group were replicated in a more representative, clinical study sample. In this way, muscle recruitment was investigated in a more representative, clinical study sample.
(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 two 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 with 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 direct comparison with 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 and Tucker 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 affected 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 preexisting 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.

ACKNOWLEDGMENTS

We acknowledge and thank Dr. Nele Dickx, Eline Renard, and Laaranne Verschueren for assisting in data collection.

GRANTS

R. D’hoore is funded by a PhD Fellowship from the Special Research Fund from Ghent University. B. Cagnie is a postdoctoral fellow of the Fund for Scientific Research (Research Foundation Flanders, FWO–Belgium). J. Van Oosterwijck is a Postdoctoral Fellow funded by the Special Research Fund of Ghent University.

REFERENCES


DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the author(s).

AUTHOR CONTRIBUTIONS


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Oosterwijck is a Postdoctoral Fellow funded by the Special Research Fund of Ghent University.


