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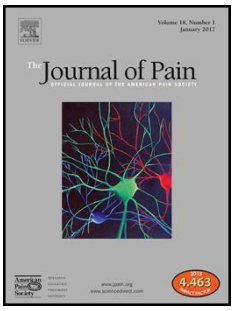


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# EXPERIMENTAL LOW BACK PAIN DECREASED TRUNK MUSCLE ACTIVITY IN CURRENTLY ASYMPTOMATIC RECURRENT LOW BACK PAIN PATIENTS DURING STEP TASKS

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**Highlights**

- Impact of experimental LBP studied in pain-free recurrent LBP patients and controls
- Higher baseline trunk muscle activity in patients than controls during step tasks
- Higher experimental pain intensity in patients than controls
- Pain-induced movement strategy changes in patients may increase LBP recurrence risk

**ABSTRACT**

Low back pain (LBP) patients demonstrate reorganized trunk muscle activity but if similar changes are manifest in recurrent LBP patients (R-LBP) during asymptomatic periods remains unknown. In 26 healthy and 27 currently asymptomatic R-LBP participants electromyographic activity (EMG) was recorded from trunk and gluteal muscles during series of stepping up and down on a step bench before and during experimentally intramuscular induced unilateral and bilateral LBP. Pain intensity was assessed by numeric rating scale (NRS) scores. Root-mean-square EMG (RMS-EMG) normalized to maximal voluntary contraction EMG and pain-evoked differences from baseline (Delta-RMS-EMG) were analyzed. Step task duration was calculated from foot sensors. R-LBP compared with controls showed higher baseline RMS-EMG and NRS scores of experimental pain ($P<0.05$). In both groups, bilateral compared with unilateral experimental NRS scores were higher ($P<0.001$) and patients compared with controls reported higher NRS scores during both pain conditions ($P<0.04$). In patients, unilateral pain decreased Delta-RMS-EMG in m. iliocostalis and bilateral pain decreased Delta-RMS-EMG in all back and gluteal muscles during step tasks ($P<0.05$) compared with controls. In controls, bilateral versus unilateral experimental pain induced increased step task duration and trunk RMS-EMG while both pain conditions decreased step task duration and trunk RMS-EMG in R-LBP patients compared with controls ($P<0.05$).

**PERSPECTIVES**

Task duration and trunk muscle activity increased in controls and decreased in R-LBP patients during experimental muscle LBP. These results indicate protective strategies in controls during acute pain while R-LBP patients showed higher pain intensity and altered strategies that may be caused by the higher pain intensity, but the long-term consequence remains unknown.

**KEYWORDS**

Lumbar spine, pain induction, motor control, recurrent low back pain, sensitization



## INTRODUCTION

Low back pain (LBP) is the primary musculoskeletal cause of disability globally[34]. The majority of LBP patients suffer from recurrent symptoms (R-LBP)[60]. R-LBP is defined as individual episodes of LBP after minimum 1 month without preceding pain[59] and has been suggested to predispose to persistent LBP from multiple factors[47]. Although the underlying mechanisms in transition from recurrent to persistent LBP generally remain unknown[5], changes in the sensory[73] and motor systems[33] in persistent LBP patients compared with healthy controls have been proposed to play an important role[44]. In patients with persistent LBP compared with asymptomatic controls, increased experimental pain intensity and decreased pressure pain thresholds in the extremities[53] indicate enhanced nervous system excitability[73]. These changes generally are established from long-term or repetitive nociceptive inputs that may influence pain perception and disability[61]. However, increased cold and mechanical pain sensitivity in the back and remote anatomical regions in acute LBP patients compared with pain-free controls[62] indicate that the somatosensory

system may be sensitized without long-term nociceptive input, but it is unclear if sensitization is manifest in currently asymptomatic R-LBP patients.

Furthermore, acute experimental pain changes the trunk muscle activity during gait[69] and after standing surface perturbation[30,41]. However, observations of alterations in the trunk muscle activity in persistent LBP patients compared with healthy controls during e.g. gait[3,40] and stance[49] served as basis for different hypotheses about the role of motor control and function in development of LBP. Hip[13,38] and trunk muscle dysfunction[33] and instability mechanisms[31] therefore have been suggested as drivers of inappropriate motor strategies resulting in pain. In R-LBP patients, however, alterations in the trunk muscle activity during different tasks[9,10,36] have been observed during remission of pain. These relatively permanent changes in the motor system may elevate the risk of additional LBP incidences[44].

Investigation of pain-evoked trunk muscle activity changes during functional motor tasks may contribute to further knowledge about the role of trunk muscles in LBP, and gait therefore has been studied extensively[20]. Stair ascent and descent requires higher force in the lower extremity and back muscles[45] than overground gait and may challenge the temporal control of the body during movement[22]. However, the impact of acute or persistent LBP on the trunk muscle activity during stepping up and down is unexplored. During overground gait the trunk muscle activity in persistent LBP patients compared with controls is generally increased and the task velocity lower[39] which has been interpreted as protective strategies[68,70].

Experimental pain in healthy participants has been used extensively to study underlying mechanisms in sensorimotor alterations[4]. Higher impact of experimental pain on the sensory system is often seen in patients compared with healthy participants[53] and since pain induction in R-LBP patients during remission of LBP mirrored recalled clinical pain[15], this model may effectively

replicate clinical LBP in an experimental setting. Most experimental pain studies are based on unilateral pain induction in contrast to patients that often report bilateral and high intensity pain after longer-lasting LBP[12]. A recent study showed increased LBP intensity and areas in healthy participants after bilateral compared with unilateral experimental LBP and correlation of pain intensity and the pain-evoked trunk muscle activity changes after surface perturbations[41].

The aim of this study was to compare effects of unilateral and bilateral experimental LBP on the trunk muscle activity and task duration during step tasks in healthy participants and R-LBP participants during remission of pain. In R-LBP patients compared with controls it was hypothesized that experimental LBP resulted in (1) longer step duration, (2) higher pain intensity and (3) increased trunk muscle activity and (4) that the underlying changes in hypothesis 1-3 was more expressed for bilateral compared with unilateral experimental LBP.

## MATERIALS AND METHODS

### *Participants*

Mild to moderate disabled currently asymptomatic R-LBP patients and healthy control participants with no previous self-reported history of LBP were recruited from University College Northern Denmark, Aalborg University, and Aalborg University Hospital by posters and e-mail groups. Participants aged 18 to 50 years were included and exclusion criteria were (i) pregnancy, (ii) present or previous self-reported psychological disease, and (iii) present pain or unable to make full trunk flexion and extension from standing position without reporting pain. Additionally, R-LBP participants were excluded if they had radiologic verified malignancy, osteoarthritis or previous fractures in or related to the lumbar spine. The age range of participants was based on excluding adolescents and elderly[54] subjects. The study was approved by the local ethics committee (N-

20140006) and conducted in accordance with the Helsinki Declaration. Informed consent was obtained from each participant prior to the study.

*Summary of experimental protocol*

The participants completed the Oswestry Disability Index version 1 (ODI)[7] which is considered a reliable and valid estimation of disability in LBP patients and were asked whether they suffered from any disability, surgery or pain[17]. Electromyography was recorded during maximal voluntary contraction values of relevant trunk muscles. Subsequently, three consecutive sessions of 10 steps up (ascent steps) and 10 steps down (descent steps) on a step bench were performed during electromyographic recordings of trunk muscles (1) at baseline, and in random order during (2) unilateral experimental LBP, and (3) bilateral experimental LBP. The three sessions were recorded with a minimum of 15 min break between sessions or until a pain free period of minimum 5 minutes.

*Experimental low back pain*

Experimental LBP was induced by intramuscular injections of hypertonic saline. While the participants were seated on a chair in a relaxed position, the injection site at L2 level (see below) was cleaned with alcohol and sterile hypertonic (1.0 ml, 5.8%) or isotonic (non-painful control, 1.0 ml, 0.9%)[23] saline was injected perpendicular to the skin surface with a 25G × 28 mm needle. The participants were informed about the procedure but blinded to the type of injection. The Th12 segment was identified by palpation of the ribs and counted down to L2. After this, L2 was verified by palpation of L4 at the line between the iliac crest bilaterally[8] and L2 was estimated by counting

upwards. The most bulky part of m. longissimus at the L2 level was then palpated (typically 3-5cm from the midline) and marked bilaterally as injection sites.

Saline was injected during two conditions: (1) Unilateral pain with one hypertonic saline injection in the dominant side immediately followed by an injection of isotonic saline in the contralateral side and (2) Bilateral pain with one injection of hypertonic saline in the dominant side immediately followed by an injection of hypertonic saline in the contralateral side. After completing the two injections, the participants were assisted to a standing position in front of the step bench to begin the step series. During the step tasks the participants were asked to rate the pain intensity on a verbal numeric rating scale (NRS), defined by numbers from 0 ('no pain') to 10 ('maximum pain') after each of the 10 ascent and descent steps.

*Step task trials*

Each step task session consisted of series of 10 ascent followed by descent steps on a step bench (height 30 cm x width 90 cm x depth 35 cm) at self-selected speed before and after induction of experimental LBP. The participants were required to complete ascent steps by stepping onto the bench with the dominant foot leading, step up and stand with both feet on the bench. The descent steps were then completed by stepping off the bench with the non-dominant foot leading. The participants were instructed to stand still for approximately 2 seconds between each of the ascending and descending steps and before turning towards the bench for the next step after completing the descending steps. Between each series of 10 ascent followed by descent steps the participants were seated on a chair for 3 minutes or a pain-free period of minimum 3 minutes.

The step task phases were recorded and extracted from four wireless footswitches (10 mm diameter, Noraxon FSR, Noraxon, USA) mounted bilaterally to the plantar surface of (i) the center

of the heel, and the most prominent spots on (ii) meta tarsal bone 1, (iii) meta tarsal bone 5 and (iv) hallux. The duration was defined as intermediate periods between foot contacts; during ascent steps between dominant toe off and non-dominant initial foot contact and during descent steps between non-dominant toe off and dominant initial foot ground contact (Fig. 1.A). The ascent and descent phases[74] were automatically identified from the footswitch data exported to Matlab® 2014 (Mathworks Inc.) Subsequently, the phases were visually confirmed, and excluded if onset or offset time was ambiguous. The duration of intermediate periods was averaged across the 10 steps. Additionally, the difference from baseline to post pain duration was calculated (Delta-time) and expressed as percentage of baseline, since the velocity was self-selected.

*Electromyography of trunk muscles*

Surface electromyography (EMG) signals were recorded from 3 back, 3 abdominal, and 2 gluteal muscles from the dominant side of the trunk by self-adhesive dual surface electrodes (4x2.2 cm, 10 mm diameter adhesives, with fixed inter-electrode distance of 1.75 cm, Noraxon USA). After the skin was shaved and cleaned with alcohol, electrodes were placed on the skin over the abdominal muscles according to previous recommendations[29]: m. obliquus internus (along the horizontal line between left and right anterior superior iliac spine, medial from inguinal ligament[1]), m. rectus abdominis (3 to 4 cm lateral to and at the level just above the navel[52]), and m. obliquus externus (along the line from most inferior point of costal margin to opposite pubic tubercle,cranial electrodes were placed directly below most inferior point of costal margin [2]). Likewise, electrodes were attached to the skin over the back muscles after confirmation of the anatomical landmarks by palpation during submaximal contraction: m. iliocostalis (approximately one finger width medial from a line from posterior superior iliac spine to lowest point of lower rib

at L2 level, m. longissimus (approximately 2 fingers width lateral from L1 spinal process), and the erector spinae muscle overlying m. multifidus at the L4 level (*m. multifidus*, 1 cm medial and parallel to a line between posterior superior iliac spine, and first palpable spinous process from the L4 level). Finally, electrodes were attached to m. gluteus maximus (approximately middle of the line between the sacral vertebrae and the greater trochanter of femur), and m. gluteus medius (approximately middle of the line from the highest point of iliac crest to the greater trochanter of femur).

EMG was recorded during maximal voluntary contractions (MVC) in standing positions with external manual resistance after 3 submaximal training trials for each muscle or muscle group. The lumbar extensor muscles were tested through spinal extension from about 30 degrees standing trunk flexion position[65]. The abdominal muscles were tested through standing trunk flexion from a 20 degrees trunk flexion position with and without left and right-sided rotation[16]. The gluteal muscles were tested during standing in slight forward trunk flexion position[56] while holding a firm grip in a bench with both hands.

The EMG signals were filtered with a 4$^{th}$ order Butterworth band-pass filter (10–500 Hz) and sampled at 1500 Hz with a gain of 500 by a wireless transmission system with 16 bit analogue-to-digital resolution (DTS, Noraxon USA). The EMG signals were exported to Matlab® 2014 (Mathworks Inc.) for offline analysis. EMG data were full-wave rectified, smoothed with a 100 ms moving average window and mean root-mean-square (RMS-EMG) values were derived for each of the ascending and descending phases.

The EMG data from MVC recordings were full-wave rectified, smoothed with a 200 ms moving average window and the maximum EMG amplitude of each muscle (max-EMG) was identified as maximal peak EMG values over a 500 ms window with the greatest average EMG

amplitude within the MVC test[72]. The RMS-EMG of ascending and descending phases was expressed as percentage of the max-MVC of the individual muscle[11] for analysis of the muscle activity between the groups at baseline. RMS-EMG after pain-induction was extracted for the individual ascending and descending phases during the experimental pain sessions and expressed as percentage of the baseline RMS-EMG values (Delta-RMS-EMG; baseline defined as 100%) for each phase.

*Statistics*

The sample size estimations were conducted a priori in GPower 3.1.9.2 based on results from previous studies of pain-evoked differences in trunk muscle EMG[41,42]. The variance of Delta-RMS-EMG was set for 0.1 and the level of significance was set at $p<0.05$. With a statistical power of 80%, a sample of 22 participants in each group were required and 26 healthy and 27 currently asymptomatic R-LBP participants were included.

Statistical analyses were performed in SPSS®23.0 (IBM) and statistical significance was accepted at $P < 0.05$. Data are presented as mean and standard error of the mean (SEM). Participant characteristics were compared between groups by independent t-tests. Pain NRS scores, step phase duration, and RMS-EMG data were normally distributed as assessed by Shapiro-Wilk's test of normality. All data were analyzed with mixed model analysis of variance (ANOVA). Significant results were post-hoc tested by independent t-tests for comparison between groups and dependent t-tests for comparison between conditions. Subsequently, the P-values were Bonferroni adjusted to correct for multiple comparisons.

The NRS scores were analyzed with a four-way ANOVA with *group* (Control and R-LBP) as between and *condition* (unilateral and bilateral pain), *step tasks* (ascent and descent) and *steps* (1-

10) as within factor. Step phase duration at baseline was analyzed with two-way ANOVA with *group* as between and *phases* (ascending and descending) as within factors. Baseline RMS-EMG during the ascent and descent step tasks were analyzed with two-way ANOVA with *group* as between and *muscle* (m. obliquus internus, m. rectus abdominis, m. obliquus externus, m. iliocostalis, m. longissimus, m. gluteus maximus and m. gluteus medius) as within factors.

The difference between baseline and pain conditions for the step phase duration (Delta-time) was analyzed with three-way ANOVA with *group* as between and *phases* and *condition* as within factors. Pearson's correlation coefficient (r) was used to correlate Delta-time between the two pain conditions and correlate Delta-time with pain NRS scores for each of the two pain conditions. Finally, Delta-RMS-EMG during each of the ascent and descent tasks was analyzed with three-way ANOVA with *group* as between and *muscle* and *condition* as within factors.



**RESULTS**

*Participant characteristics*

The study included 26 healthy participants [16 females; age 23.6 ± 4.4 years; body mass index (BMI) 23.8 ± 2.5 kg/m$^2$; no disability (ODI score 0.87 ± 1.69)] and 27 patients suffering from recurrent mild to moderate recurrent low back pain with no present pain [12 females; age 27.4 ± 9.9; BMI 21.9 ± 3.2 kg/m$^2$ and low to moderate disability (mean ODI score 32.2 ± 7.7)]. There were no significant differences between the two groups in age (P>0.09) and BMI (P>0.23). No participants had present or previous self-reported psychological disease, lower extremity dysfunction, gait limitations, or present LBP or other musculoskeletal pain at the beginning of the experiment. Participants in the R-LBP group suffered from recurrent non-specific LBP with a minimum of two

annual episodes of LBP during the last three years with pain intensity ranging from 3 to 6 on a NRS. In 2 R-LBP participants, the first experimental pain session resulted in longer lasting soreness that was still present when the second experimental pain session was scheduled. One of the asymptomatic participants additionally felt uncomfortable after the first experimental pain session and all three participants were excluded from analyses (R-LBP: n=25; controls: n=25).

*Experimental low back pain intensity*

An interaction between group, condition, task and steps was found for the pain NRS scores (Table 1; ANOVA: $F(9,432) = 5.93$, $P < 0.001$). Post-hoc tests showed no differences between pain NRS scores during ascent and descent tasks but pain NRS scores were higher in the R-LBP compared with the control group during both tasks during unilateral (Bonferroni: $P < 0.02$) and bilateral (Bonferroni: $P < 0.04$) pain. In both groups, NRS scores additionally were higher during bilateral compared with unilateral pain (Bonferroni: $P < 0.001$).

*Step phase duration and correlation with pain NRS scores*

The baseline step phase duration in the control group was 1536.6 ± 31.4 ms during ascent and 1454.4 ± 24.4 ms during descent and in the R-LBP group 1591.1 ± 18.4 ms during ascent and 1465.5 ± 14.6 ms during descent; a two-way ANOVA showed no interaction between groups and phases (ANOVA: $F(5,240) = 1.58$, $P = 0.21$). A three-way ANOVA of pain-induced changes in the step phase duration from baseline values (Delta-time) showed an interaction between groups, phases and conditions (Fig. 1; ANOVA: $F(1,48) = 39.0$, $P < 0.001$). Post-hoc analyses showed that both unilateral and bilateral pain reduced the ascent step phase duration compared with baseline (Delta-time) in the R-LBP group in comparison with the control group (Bonferroni: $P < 0.001$).

During bilateral compared with unilateral pain Delta-time decreased in the R-LBP group (Bonferroni: $P < 0.05$) and increased in the control group (Bonferroni: $P<0.03$).

Delta-time showed low correlation with NRS scores during unilateral ($r=-0.17$, $P=0.30$) and bilateral ($r=-0.01$, $P=0.23$) pain but high correlation was present in Delta-time between unilateral and bilateral pain ($r=0.73$, $P<0.001$).

*Baseline muscle activity during step tasks*

At baseline, an interaction between muscles and groups was observed for the ascent (Fig. 2A; ANOVA: $F(7,336) = 2.81$, $P < 0.03$) and descent (Fig. 2B; ANOVA: $F(7,336) = 23.27$, $P < 0.04$) step tasks. Post-hoc analyses showed higher baseline RMS-EMG muscle activity in all back and abdominal muscles in the R-LBP compared with the control group during ascent (Bonferroni: $P < 0.02$) and descent (Bonferroni: $P < 0.04$) step tasks.

*Muscle activity during painful step tasks*

A three-way ANOVA of Delta-RMS-EMG showed an interaction between groups, condition, and muscles for the ascent (Fig. 3; ANOVA: $F(7,336) = 6.05$, $P < 0.01$) and descent (ANOVA: $F(7,336) = 6.82$, $P < 0.01$) step tasks. Post-hoc analyses showed that during unilateral pain Delta-RMS-EMG was lower in m. iliocostalis in the R-LBP compared with the control group during ascent (Bonferroni: $P < 0.01$) and descent (Bonferroni: $P < 0.03$) step tasks. During bilateral pain, Delta-RMS-EMG was lower in the R-LBP compared with the control group in m. iliocostalis, m. longissimus, m. obl. ext. abdominis, m. gluteus medius, and m. gluteus maximus during both motor tasks (Bonferroni: $P < 0.04$) and lower in m. multifidus during ascent step tasks (Bonferroni: $P < 0.01$).

In the control group, bilateral compared with unilateral experimental pain resulted in higher Delta-RMS-EMG in m. iliocostalis, m. longissimus and m. obliquus externus during both motor tasks (Bonferroni: $P < 0.05$) and higher Delta-RMS-EMG in m. gluteus medius during ascent step tasks (Bonferroni: $P < 0.03$). In the R-LBP group, bilateral compared with unilateral experimental pain during both step phases resulted in higher Delta-RMS-EMG in m. rectus abdominis (Bonferroni: $P < 0.03$) and lower Delta-RMS-EMG in m. iliocostalis, m. longissimus, m. multifidus, m. obliquus externus, m. gluteus medius and m. gluteus maximus (Bonferroni: $P < 0.01$).

**DISCUSSION**

This study is the first to investigate the effects of LBP provocation on trunk and gluteal muscle activity during step tasks between healthy controls and participants with episodic LBP but currently asymptomatic during assessment. In line with hypothesis 2, experimental pain resulted in higher pain intensity in the R-LBP compared with the control group. Contrary to expectations (hypothesis 3), experimental pain decreased the back muscle activity during step tasks and, interestingly, the rectus abdominis activity increased during bilateral pain in the R-LBP compared with the control group. In the control group, the back and obliquus externus abdominis muscle activity increased during bilateral pain compared with baseline. These differences, together with pain-evoked decreased step duration in the R-LBP compared with the control group, indicate changed movement strategies. These findings imply that previous LBP incidences may have a longer lasting effect on the nervous system resulting in higher sensorimotor impact of bilateral experimental LBP in the patient group although they were characterized by low to moderate disability level and were currently asymptomatic during the baseline examination.

*Experimental low back pain model and sensory implication*

Injection of hypertonic saline is among the most studied experimental pain models[24]. Although experimental pain induction in healthy participants is accepted to replicate clinical pain, the aggravated sensorimotor impact of acute pain in R-LBP patients compared with controls supported the integration of experimental pain models in LBP patients to study the impact of R-LBP. Recently, a group of unilateral R-LBP patients during remission of pain furthermore described unilateral experimental LBP as a recall of clinical pain symptom characteristics[15]. In persistent LBP patients compared with subacute LBP patients, higher prevalence of bilateral pain and higher pain intensity is evident[12]. These findings have been linked to long-lasting LBP conditions resulting in hyperalgesia[53] and structural changes in the deep trunk muscles[21]. However, the increased response to acute pain in R-LBP patients during remission of pain in the current study showed that R-LBP can also induce durable spinal and supra-spinal level changes[25] that may facilitate the effect of acute pain. Furthermore, higher pain intensity in the controls during bilateral compared with unilateral pain is in line with previous observations. High pain intensity and increased referred pain areas have been observed in the trapezius[19], masseter[64] and longissimus[41,42] muscles during bilateral experimental pain, probably caused by summation from converging inputs from nociceptors bilaterally[27]. The present study is, however, the first demonstration of spatial summation from bilateral hypertonic saline-induced pain in R-LBP patients during currently asymptomatic periods. Higher pain intensity during both pain conditions in the R-LBP group in comparison with the control group, additionally pointed towards interaction effects of summation mechanisms and facilitated central mechanisms.

*Changes in step phase duration during pain*

No differences were observed in the baseline step phase duration between the groups in accordance with a previous study[43]. No previous studies reported pain-evoked staircase or step task duration. Unilateral and bilateral pain, however, prolonged the duration in the control group and decreased the duration in the R-LBP group during ascent steps in this study. Slower self-selected velocity during overground gait in persistent LBP patients compared with controls and further decreased velocity in patients with referred leg pain and higher pain intensity[58] is suggested to indicate protective mechanisms. Pain-evoked increased duration in the control group resembles clinical observations from where it has been hypothesized that patients attempt to reduce pain by increasing the local stability by changed muscle activity[45]. During stair step tasks compared with over-ground and level walking, the gait cycle duration and trunk stability demands, however, are higher, particularly during the more biomechanically demanding ascent step task[55]. Faster duration in the R-LBP group during ascent steps therefore may reflect a pain-evoked impairment of the musculoskeletal system to maintain trunk stability[22]. This is important, particularly during vertical ambulation where high back muscle activity is required to stabilize the trunk[74] and to control the lifting of the upper body, the head and the upper extremities. However, during fast sagittal trunk movements less variable movement strategies in LBP patients, compared with controls, suggest an alternative strategy to decrease the local trunk stability demands[6]. Faster step phase duration in the R-LBP group may indicate an attempt to reduce the time spent in phases with high trunk muscle activity requirements. This may serve as a short-term pain-evoked protective mechanism, but the present findings do not show correlation between the pain intensity and task duration changes. However, experimental pain intensity is variable between participants and high correlation in Delta-time between the two pain conditions may indicate

individual consistent impact of acute pain independent from the pain protocol. Decreased task duration results in reduced motor variation[50] and reduced local stability[22] and although evidence is limited, such changes are hypothesized to increase the risk of persistent LBP[33].

*Pain evoked muscle activity changes during step tasks*

Increased activity in all trunk muscles during the pain-free baseline step tasks in the R-LBP compared with the control group indicated generalized protective movement strategies. Force development may be attenuated by clinical[51] and experimental[26] pain and the normalization of baseline muscle activity to MVC is challenged in LBP patients[14] and may have biased the findings. However, the R-LBP participants were currently asymptomatic during the baseline recordings and the observed increased trunk muscle activity is consistent with findings in persistent LBP patients. Concurrently increased abdominal and back muscle activity is hypothesized to unload the spine and increase spinal stability[70]. Biomechanical modelling, however, showed that spinal stabilization required low intensity antagonistic contraction[63] and increased trunk muscle activity in persistent LBP patients during treadmill walking recently was interpreted as a muscle relaxation problem[70]. Similar mechanism may have caused the increased trunk activity during experimental pain in the current study.

The control group demonstrated lower baseline muscle activity compared with the R-LBP group and both experimental pain conditions increased the activity in all muscles during the step tasks. Aggravated trunk muscle activity induced by acute pain may indicate adaptive movement strategies to avoid redundant movements in line with observations of rigid movement patterns during gait in persistent LBP patients[40]. This was further supported by increased muscle activity in the iliocostalis, longissimus and obliquus externus abdominis muscles in the controls during

bilateral compared with unilateral pain. In conjunction with increased step duration during ascent, these observations supported that the control group performed the step tasks through adaptive strategies[68]. This is in line with observations in persistent LBP patients[70]. However, the impact of unilateral pain on the trunk muscle activity was in contrast with the hypothesis and recent observations of muscle activity adaptations after surface perturbation in healthy controls during experimental pain[41]. These differences may be attributed the different motor task characteristics. Surface perturbation results in reactive muscle activity in a short time window[36] while step tasks challenge the proactive motor planning[46] and the use of a high step bench challenge the trunk stability during the entire task[37].

In contrast with the control group, R-LBP patients were affected predominantly after bilateral pain induction where the activity decreased in all back and gluteal muscles during both tasks, except of m. multifidus during descent steps. These observations were in contrast with the hypothesized increased trunk muscle activity in the R-LBP group that was based on assumptions about adaptation of pain-related alternative movement strategies to avoid motion of the lumbar spine[66]. The rectus abdominis muscle activity, however, increased in both groups, but significantly higher activity was observed in the R-LBP group during bilateral compared with unilateral pain. Contraction of the abdominal muscles is involved in increased abdominal pressure[63] that is suggested to increase stiffness[32] and unload the spine. Modelling[63] and clinical[48] findings supported, however, that the rectus abdominis muscle is not involved in abdominal pressure development. Furthermore, reduced low back muscle activity and concurrently high rectus abdominis activity may increase the pelvic stability through an alternative strategy resulting in posterior rotation of the pelvis[35].

The present findings support a pain-evoked guarding strategy in the controls, particularly during bilateral pain in line with results from persistent LBP patients[70]. This may indicate that high intensity pain perception results in protection of the trunk by generally increased trunk muscle activity whereas changed strategies in R-LBP patients indicated an alternative attempt to protect the spine[71] or reduce the activity in the pain-induced back muscles. Decreased activity in these important muscles during demanding step tasks requiring a high amount of potential energy propulsion and absorption[57] may increase the mechanical load and play a role in recurrence of LBP[44]. Furthermore, increased sensory impact of acute pain indicated facilitated central pain mechanisms in the R-LBP patients, but it remains unknown if these changes reflected central changes in the motor planning related to recurrence of pain[67] or short-term adaptive peripheral changes related to acute pain. Nonetheless, the observed changes in strategies during step tasks may increase the load of the lumbar structures, although the long-term consequences remain unknown.

*Limitations*

Methodologically, the EMG recordings may be influenced by crosstalk between the erector spinae muscles[18] that could result in registration of higher signals by crosstalk between activity from adjacent muscles. Interpretation of the pain intensity data from the present study could be qualified by e.g. electronic pain VAS[28] and the use of pain NRS scores could be limited in detecting differences between groups or conditions[8].

*Conclusion*

During series of ascent and descent step tasks, unilateral and bilateral experimental LBP increased the pain intensity and decreased the step task duration and trunk and gluteal muscle activity in a group of recurrent LBP patients during remission of pain compared with a healthy control group. These results support that alterations in pain perception and motor control during experimental acute pain are manifest in currently asymptomatic recurrent LBP patients. The impact of pain on the movement strategies in patients may play a role in the recurrence of low back pain.

**Conflict of interest statement**

The authors declare no conflict of interest regarding the contents of this paper.

**Author contributions**

Lars Henrik Larsen was in charge of the study set-up, planning of the data collection, data analyses and drafting of the paper. Rogerio Pessoto Hirata and Thomas Graven-Nielsen participated in planning of the data collection, data analyses and development of the final version of the manuscript. All authors discussed the results and approved the final manuscript.

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**Figure 1.** Gait phase definition and pain-evoked task-duration changes. (A) Ascent and descent phases were defined from the gait pattern for calculation of pain-induced changes in duration (Delta-time) during (B) ascent and (C) descent step tasks. During ascent step tasks Delta-time decreased in the R-LBP compared with the control group during both pain conditions (*, Bonferroni: P<0.001) and bilateral compared with unilateral pain decreased Delta-time in the R-LBP (*, Bonferroni: P<0.05) and increased Delta-time in the control group (*, Bonferroni: P<0.03).

**Figure 2.** Baseline RMS-EMG muscle activity (mean + SEM, N=25) normalized to MVC in the individual muscles across ascending and descending gait phases, respectively. Baseline RMS-EMG was higher in the R-LBP group in all back and abdominal muscles during (A) ascent (*, Bonferroni: P < 0.02) and (B) descent (*, Bonferroni: P < 0.04) step tasks. ILI=m. iliocostalis, LON=m. longissimus, MUL=m. multifidus, RAB=m. rectus abdominis, OEX=m. obliquus externus, OIN=m. obliquus internus, GME=m. gluteus medius and GMA=m. gluteus maximus.

**Figure 3.** Pain-evoked Delta-RMS-EMG (mean +/- SEM, N=25) of trunk and gluteal muscles during step tasks. Bilateral compared with unilateral pain increased Delta-RMS-EMG in m. rectus abdominis (*, Bonferroni: P<0.02) and decreased Delta-RMS-EMG in the back, obliquus externus and gluteus muscles (*, Bonferroni: P<0.001). R-LBP compared with controls decreased Delta-RMS-EMG in m. iliocostalis and m. obliquus externus during both tasks (#, Bonferroni: P<0.05) during unilateral pain and decreased Delta-RMS-EMG during bilateral pain (#, Bonferroni: P<0.02).

**Table 1.** Mean (± SEM) pain numerical rating scale (NRS) scores during ascent and descent step tasks after induction of unilateral and bilateral pain in the control (N=25) and the R-LBP (N=25) group. NRS scores were significantly higher in R-LBP compared with the control group (*, P<0.05) and during bilateral compared with unilateral pain condition (#, P<0.05).

| CONDITION | UNILATERAL PAIN | | BILATERAL PAIN | |
|---|---|---|---|---|
| TASK | ASCENT | DESCENT | ASCENT | DESCENT |
| Control | 2.10 (±0.19) | 2.08 (±0.20) | 4.68 (±0.26)$^{\#}$ | 4.61 (±0.28)$^{\#}$ |
| R-LBP | 3.59 (±0.21)* | 3.57 (±0.27)* | 6.04 (±0.38)*,$^{\#}$ | 5.99 (±0.43)*,$^{\#}$ |

Figure 1

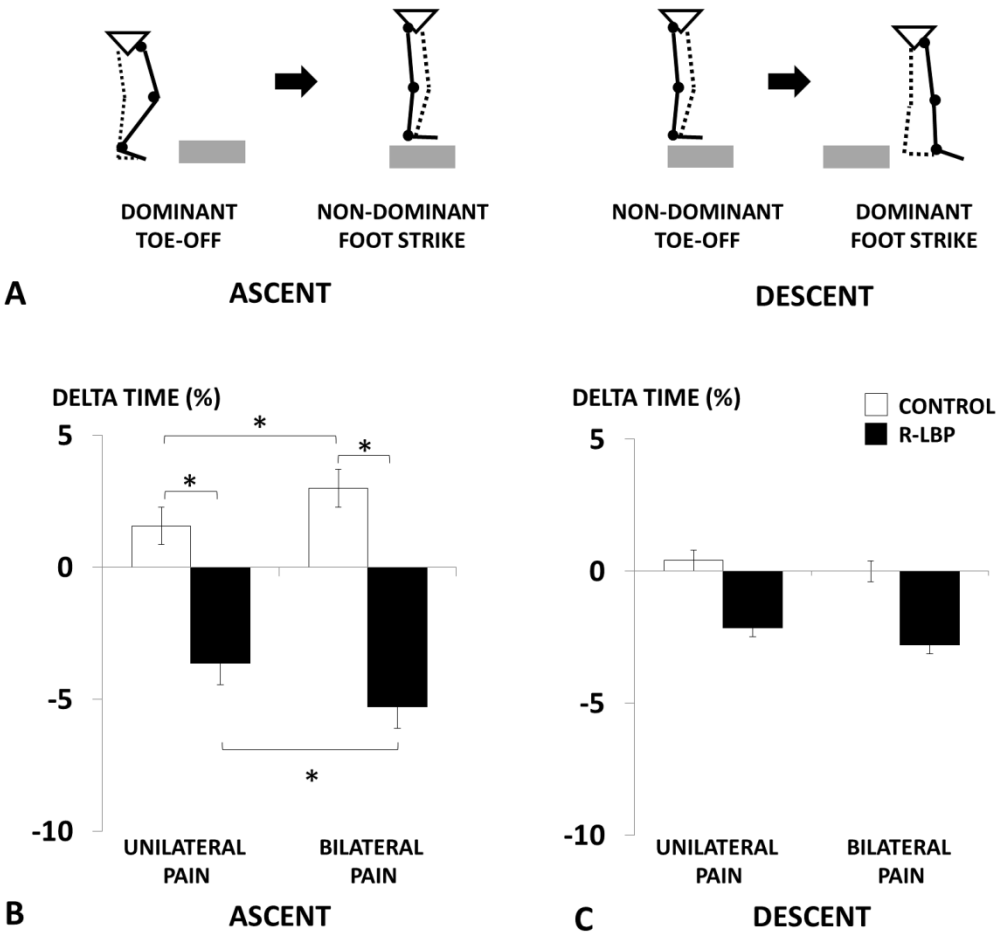

Figure 2.

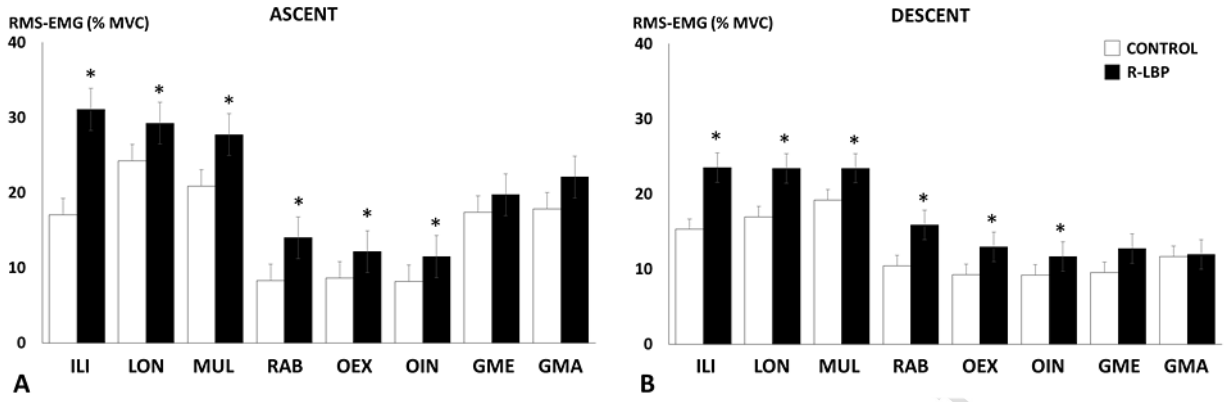


Figure 3.