3-21-2016

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Jo Armour Smith
Chapman University, josmith@chapman.edu

Kornelia Kulig
University of Southern California

Recommended Citation

Smith JA, Kulig K. Altered multifidus recruitment during walking in young asymptomatic individuals with a history of low back pain. *J Orthop Sports Phys Ther*. 2016;46(5):365-374. doi: 10.2519/jospt.2016.6230

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Altered multifidus recruitment during walking in young asymptomatic individuals with a history of low back pain.

Jo Armour Smith PT, PhD[1, 2]*, Kornelia Kulig[2] PT, PhD
[1]Department of Physical Therapy, Crean College of Health and Behavioral Sciences, Chapman University
[2]Division of Biokinesiology and Physical Therapy, University of Southern California

*Author for correspondence
Jo Armour Smith
Department of Physical Therapy
Harry and Diane Rinker Health Science Campus
Chapman University
9401 Jeronimo Road
Irvine, CA, 92618.

josmith@chapman.edu

This study was approved by the University of Southern California Health Sciences Campus Institutional Review Board

This study was funded in part by awards from the Orthopaedic Section of the APTA, the Foundation for Physical Therapy and the American Society of Biomechanics.

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Financial Disclosure and Conflict of Interest. The authors affirm that we have no financial affiliation (including research funding) or involvement with any commercial organization that has a direct financial interest in any matter included in this manuscript. We also affirm that we have no other conflict of interest (i.e., personal associations or involvement as a director, officer, or expert witness).

ABSTRACT

STUDY DESIGN: Cross sectional, laboratory study

BACKGROUND: Individuals with low back pain have impaired activation of multifidus during postural adjustments and increased activity of the erector spinae musculature during walking. However, it is unclear if these alterations in muscle activity are evident during locomotion in individuals with a history of low back pain when they are between symptomatic episodes.

OBJECTIVES: To compare paraspinal muscle activity in young healthy individuals and young individuals with a history of low back pain during walking turns

METHODS: 14 asymptomatic individuals with a history of low back pain and 14 controls performed 90° walking turns at both self-selected and fast speed. The duration and amplitude of activity in the deep fibers of multifidus and the lumbar and thoracic longissimus were quantified using intramuscular electromyography.

RESULTS: There was a significant speed by group interaction for the duration of multifidus activity ($p = .013$). Duration of activity increased from the self-selected to the fast locomotor speed in the controls, but decreased in the individuals with a history of low back pain ($p = .003$). Self-selected speed was the same in both groups ($p = .719$). There was a trend towards a significant association between group and the direction of change in the duration of deep multifidus activity ($\chi^2 = .058$). Duration of thoracic longissimus activity and amplitude of

multifidus and thoracic longissimus activity increased similarly in both groups from the self-selected to faster speed.

CONCLUSION: Even between symptomatic episodes, young individuals with a history of low back pain demonstrated altered recruitment of the deep fibers of lumbar multifidus in response to changing locomotor speed during walking turns.

Key Words: paraspinal muscles, locomotion, walking turns, recurrent back pain

Background

Despite substantial research, and escalating health care costs over the past few decades, the mechanisms underlying the transition from acute to persistent LBP are still not well understood or effectively managed[1,2]. The majority of back pain research to date has focused on individuals who experience chronic, largely unremitting pain (chronic LBP)[3-5]. However, there is increasing recognition that there is a distinct sub-group of individuals with persistent LBP who experience an episodic or recurrent pattern of symptoms[6]. In these individuals, successive episodes of LBP become longer and more likely to require absence from work and medical intervention over time[7]. In the absence of clear precipitating events or significant patho-anatomical dysfunction, it is often unclear why these individuals experience recurrences of their back pain following periods of time when they are entirely symptom-free. However, persistent and maladaptive alterations in dynamic trunk postural control may contribute to this recurrence[6,8-10]. In order to understand the development and persistent of both recurrent and chronic LBP, and to identify appropriate interventions, it is vital to clarify if changes in trunk postural control are an adaptive response to concurrent symptoms or if they reflect a persistent and maladaptive change in motor control. This can be ascertained by investigating individuals with recurrent LBP during the periods of time when they are asymptomatic[7,11].

Research investigating postural adjustments in the trunk has already demonstrated altered amplitude and timing of activity in the paraspinal muscles in both persons with chronic LBP and asymptomatic individuals with a history of recurrent LBP [8,12-14]. The paraspinal muscle group comprises the muscles adjacent to the spinal column. In the lumbar region the paraspinals can be subdivided into the erector spinae (iliocostalis

lumborum and longissimus thoracis pars lumborum, hereafter termed “lumbar longissimus”) and the transversospinales (of which the multifidus is the major component).[15] The lumbar multifidus is commonly further subdivided into the deep portion of the muscle, with fibers that extend across only two vertebral segments, and superficial portion of the muscle, with fibers that cross up to five vertebrae[16-18]. Similarly, in the thoracic region the paraspinals comprise the erector spine (spinalis, iliocostalis thoracis and longissimus thoracis pars thoracis, hereafter termed “thoracic longissimus”) and the transversospinales. Changes in paraspinal control in individuals with low back pain include delayed and decreased activation in the deep fibers of the lumbar multifidus muscle[8] and task- and subject-dependent modifications in the timing and amplitude of activity of the lumbar and thoracic erector spinae[12-14]. Impaired dynamic trunk postural control is also evident in symptomatic individuals with LBP during locomotion. Studies of treadmill walking utilizing surface electromyography have demonstrated increased duration and amplitude of activity in the erector spinae during locomotion in persons with chronic LBP[3-5,19-21]. To date it is unclear if these same changes in erector spinae function during walking are evident in individuals with a history of recurrent LBP during periods of time when they are asymptomatic. It is also unclear if, there are impairments in the recruitment of the deep fibers of the lumbar multifidus during walking in individuals with LBP.

Research also suggests that the normal increase in paraspinal activity in response to increasing locomotor speed is not affected by LBP[4,19]. However, as existing studies investigating paraspinal activity in individuals with LBP have used surface electromyography[19,4,20] they have not been able to differentiate between the muscles

comprising the paraspinal group[22]. Therefore it is not known if the relative contribution of the individual muscles to this increase in activity is the same, or whether individuals with LBP have altered distribution of activity across the paraspinal group. This problem can be overcome by utilizing fine-wire intramuscular EMG electrodes[23,24]. In the lower limbs, modulation of muscle activity in response to increasing locomotor speed encompasses both shifts in timing and changes in amplitude, and the pattern of these modulations is muscle specific[25]. Therefore, investigating temporal and spatial adaptations to increasing locomotor speed may help to elucidate functional differences in control of the paraspinals in individuals with a history of low back pain. Postural demand in the trunk during locomotion is also greater during functional locomotor perturbations such as walking turns, particularly in the upper trunk[26,27]. Thus, walking turns may provide an excellent paradigm for differentiating between activity in the lumbar and thoracic regions of the paraspinals in healthy individuals and those with a history of back pain.

The primary purpose of this study was to compare postural activity in the individual muscles of the paraspinal group during walking turns made at varying speeds in healthy young individuals and asymptomatic young individuals with a history of LBP. We hypothesized that individuals with a history of LBP would demonstrate reduced activity in the deep fibers of multifidus compared with healthy controls but greater activity in the lumbar and thoracic fibers of longissimus.

Methods

Participants

Twenty-nine young adults between the ages of 22 and 31 years participated in the study (17 women, Table 1). Participants were recruited via word of mouth and study flyers. Control participants (CTRL) were individually matched to participants with recurrent LBP (RLBP) by sex, age (± five years), height and weight (± 10 %), and typical activity level in metabolic equivalents (METS, ± 15 %; Table.1). One female participant with a history of recurrent LBP did not complete the data collection due to a transient vasovagal reaction to intramuscular EMG insertion. Therefore only the remaining fourteen participants with a history of recurrent LBP were matched to control participants. A priori power analyses of preliminary data collected in our laboratory indicated that a minimum sample size of ten per group would be adequate to determine a statistically significant difference between groups for duration of muscle activity at a power of $\beta = 0.8$ and statistical significance of $\alpha = 0.05$ and an effect size of 1.06. The Institutional Review Board of the University of Southern California approved the procedures in the study. Participants gave written informed consent after a full explanation of the study procedures.

Participants were eligible for inclusion in the RLBP group if they; 1) were between 18 and 40 years of age; 2) had a history of more than one year of recurrent episodes of LBP; 3) had primarily unilateral pain localized to the area between the twelfth rib and the gluteal fold; 4) reported at least two pain episodes of at least 24 hours' duration in the preceding year[6]; 5) had pain episodes that were severe enough to limit function; and 6) were in symptom remission at the time of the data collection (defined as a score of less than 0.5/10 cm on a visual analogue scale for current pain at the start of the data collection). Participants were excluded if they had contraindications to intramuscular

EMG, history of low back surgery, spinal stenosis, scoliosis, malignancy, spinal infection, or lumbar radiculopathy, or musculoskeletal injury affecting locomotion. Prior to instrumentation, all potential participants for both groups were screened by a physical therapist. This testing included a neurological screen (lower limb myotomes, dermatomes and reflexes), straight leg raise test and Thomas test, hip and spinal active range of motion in all planes and documentation of any symptoms produced or aberrant motions during these tests.

Participants with a history of RLBP also completed several questionnaires to assess potential psychosocial influences on LBP and motor behavior[28]. Fear avoidance beliefs were quantified using the Fear Avoidance Beliefs Questionnaire (FABQ[29]). Self-efficacy was quantified using the Low Back Activity Confidence Scale (LoBACS[30]). Disability due to LBP was quantified using the modified Oswestry Disability Index (ODI[31]). In addition, all participants completed visual analogue scales for current pain at the beginning of the data collection and for pain experienced during the walking turns at the end of the data collection (VAS)[32].

Instrumentation

Fine-wire intramuscular electrodes were inserted into the deep fibers of the lumbar multifidus at L4, the lumbar longissimus at L4 (LES), and the thoracic longissimus at T10 (TES) using real-time ultrasound imaging (custom-made, 50 μm gauge nickel chromium alloy wires, nylon insulation, tips bent back 5mm and 3mm with the distal 2mm of wire exposed, 25 gauge hypodermic needles, 8 MHz linear transducer, Sonoline Antares™, Siemens Medical Solutions Inc, USA; Figure 1). Electrodes were inserted into the symptomatic side in participants with a history of recurrent LBP and the

same side for the matched healthy participant. Depth of insertion was subject-specific and based on ultrasound visualization of the tip of the needle in the muscle. The correct electrode placement was confirmed by observing the contraction induced by light electrical stimulation using ultrasound imaging[33]. We have previously demonstrated that this methodology is associated with minimal pain or change in locomotor kinematics in both healthy individuals and individuals with a history of LBP[34].

The electrodes were connected to wireless differential preamplifiers. Wireless force-sensitive resistor foot switches were also attached bilaterally to participants' shoes under the lateral heel and the first metatarsophalangeal joint (TeleMyo DTS Telemetry, Noraxon USA Inc, Scottsdale, USA, baseline noise < 1µV RMS, cMR>100dB, system gain for all channels x 400). EMG and foot switch data were transmitted via a wireless transmitter, digitally sampled at 3000 Hz at 16 bit resolution and synchronized using photoelectric triggers (Qualisys Track Manager v2.6, Qualisys AB, Gothenburg, Sweden). As part of a broader study, participants were also instrumented with a full-body marker set for three-dimensional motion capture.

Experimental task

Participants performed multiple laps of a walking circuit that required both straight walking and a series of 90° turns (Figure 2a). Participants walked first at a relaxed, self-selected speed (SELF), and then at a controlled average speed of at 1.5 m/s ± 5 % (FAST). Average locomotor speed was quantified using the total time taken to complete the circuit. In each lap of the circuit, participants performed an ipsilateral turn by stepping into an outlined area with the foot ipsilateral to the turn direction and turning briskly 90° (Figure 2b). All participants spontaneously utilized a pivot strategy to

complete the turn, with the change in direction being accomplished by a pivot on the stance foot[35]. For consistency, all participants turned contralateral to the side of their EMG instrumentation (contralateral to the symptomatic side in the RLBP group and to the matched side in the CTRL group). Therefore, the stance phase of the turn occurred on the limb contralateral to the side of the electrodes. Although preliminary data indicated minimal differences in EMG variables between turn directions, turns contralateral to the instrumentation were selected in order to maximize erector spinae activity at initial contact. Prior to data collection, participants practiced the walking circuit until they were consistently able to turn with the correct foot in the correct area without altering stride length or changing cadence.

Data processing

15 trials were analyzed for each participant at each speed. The first 15 clean trials were selected for analysis for all individuals. Trials were excluded if the participant performed the turn incorrectly. Timing of locomotor events was determined using the foot switches and all data were analyzed across the stride cycle of the turn, from the initial contact of the limb ipsilateral to the turning direction to the next initial contact of the same limb. EMG data were processed in MATLAB® (MathWorks, MA, USA). After removal of the DC offset, the EMG signals were band-pass filtered (40 Hz – 1500 Hz, digital zero-phase Butterworth filter) and full-wave rectified.

Data analysis

The onset and offset of muscle activity during each turn was calculated using the integrated profile or iEMG method[36,37]. This technique has been validated in experimental data for the trunk musculature and in signals with artificially simulated

noise[36]. It results in fewer errors than standard threshold detection protocols when determining postural trunk muscle activity as it is not dependent upon baseline activity or the rate of signal increase[36].

The amplitude of each signal was first continuously integrated across the stride cycle and normalized so that the final value was 1. The time of the stride cycle for each individual trial was also normalized to 1. The integrated signal was then subtracted from a reference line with a slope of 1, that reflects the hypothetical condition where the muscle activity remains constant across the time-series of the trial[37]. The local maxima and minima of the deviations of the actual integrated signal from the reference line was then used to determine the timing of onset or offset[38]. The algorithm was implemented with a visual check of the detected onset and offset events superimposed over the rectified/band-passed signal to ensure appropriate determination[36,39,40]. The duration of the muscle burst occurring between each onset and offset event was calculated, and the sum of the duration of all bursts across each stride cycle, stance phase and swing phase was calculated and expressed as a percentage of the total duration of the stride cycle, stance phase and swing phase for that trial. The average amplitude of activity in each muscle was also calculated across the stride cycle and within the stance and swing phases individually for each turn at each speed. The stance phase and swing phase values were then amplitude normalized for each participant to the average value across the stride cycle during turns performed at the self-selected speed.

The within-day standard error of the measurement (SEM) of the EMG variables was also calculated. The SEM is an index of measurement error, expressed in the measurement units. Changes in any variable that exceed the SEM can be interpreted as

being larger than the measurement error.[41] Four healthy individuals performed two blocks of 15 turning trials at the faster speed. The two blocks of trials were separated by a period of approximately 15 minutes during which they performed a different sub-maximal motor task. Intra-class correlation coefficients (ICC [3,15]) were calculated for duration of activity and amplitude of activity between the two blocks of trials and the SEM was calculated using the following equation, where s is the standard deviation:

$$\text{SEM} = s\sqrt{1 - ICC}$$ [42]

Statistical analysis

Self-selected average locomotor speed and VAS for pain during the walking turns were compared between groups using paired t-tests. Parametric analysis is appropriate for VAS pain data as the VAS for pain has been demonstrated to have the properties of a ratio scale. [43] Individual mixed-design ANOVA was conducted to assess the main effect of speed (within-subjects factor, SELF and FAST speeds) and group (between subjects factor, CTRL and RLBP groups) and the interaction effect between speed and group for the average duration of the turn stride cycle, and the duration and average amplitude of muscle activity across the stride cycle of the turn and within the stance and swing phases for each muscle. Similarly, mixed-design ANOVA was conducted to assess the main effect of speed and group and the interaction effect between speed and group for the average normalized amplitude of muscle activity within the stance and swing phases for each muscle. Post-hoc comparisons were made using t-tests with a Bonferroni correction (adjusted level of significance = .01). Effect sizes for post-hoc comparisons were calculated using Cohen's *d*, with .8 indicating a large effect size, .5 a medium effect size and .2 a small effect size. Chi square analysis was used to investigate the association

between group and the frequency of increase or decrease in each variable. All statistical analyses were performed using PASW Statistics (Version 18, IBM Corp., Armonk, NY).

Results

Median ± interquartile range FABQ score (physical activity subscale) in the RLBP group was 12.50 ± 6.75. Median ± interquartile range LoBACS score in the RLBP group was 88 ± 12.83, which is higher than previously reported values in a LBP population[3,44]. Median ± interquartile range ODI score was 18.0 ± 15.0 % indicating minimal disability. At baseline, average ± standard deviation current pain was 0.12 ± 0.24 cm in the participants with a history of recurrent LBP and 0 cm in all of the controls. One individual who reported pain of less than 0.5 during the subjective screening procedures completed a VAS that was measured as 0.8 at the commencement of the data collection (after the physical examination). The decision was made to include his data as this value is well below the minimal detectable change for the VAS. During the locomotor trials participants reported low levels of discomfort associated with the intramuscular EMG electrodes (RLBP 0.50± 0.70 cm, CTRL 0.45± 0.70 cm, p = .779). Reliability was excellent, with ICC values exceeding 0.85 for all variables except the duration of activity in the thoracic longissimus.

Self-selected locomotor speed and locomotor events

All participants were able to complete the walking turns at the self-selected and faster speeds. Self-selected locomotor speed was the same in both groups and was slower than the fast speed in all participants except one individual in the CTRL group (average SELF speed, CTRL group = 1.22 ± 0.13 m/s, RLBP = 1.23 ± 0.10 m/s, p = .719). The

speed at which the turn was executed increased at the faster speed, with a significant decrease in the duration of the stride cycle of the turn ($F(1, 26) = 102.274$, $p = < .0001$; SELF average duration 1.16 ± 0.09 s, FAST average duration 1.02 ± 0.06 s). There was no effect of group or speed by group interaction for locomotor speed or turn duration.

Overview of paraspinal activity during walking turns

Exemplar EMG data and an overview of paraspinal activity are provided in Figure 3.

Duration of activity

Total duration of activity in each muscle during stance and swing phase at each speed is shown in Figure 4a and Figure 5. There was a significant speed by group interaction for the duration of deep multifidus activity ($F(1, 26) = 7.186$, $p = .013$, Figure 4a), but no main effect of speed or group ($F(1,26) = .006$, $p = .938$; $F(1, 26) = .021$, $p = .886$ respectively). Post-hoc comparisons indicated that was a trend towards a significant decrease in duration from self-selected to fast speed in the RLBP group ($p = .04$, Cohen's $d = 0.23$) and that the average duration of activity across the stride cycle increased in the CTRL participants but decreased in the RLBP participants (average change from SELF to FAST, CTRL + 0 .84 ± 1.87 %, RLBP -0.79 ± 1.30 %, $p = .003$, $d = 1.01$). This difference exceeded the SEM (0.56 % of stride cycle). Analyses of stance and swing phase individually indicated that this interaction effect was significant during swing phase (swing phase speed by group interaction $F(1, 26) = 4.861$, $p = .037$), but not during stance ($F(1,26) = 2.467$, $p = .128$) Eight of the individuals in the CTRL group demonstrated an increase in duration of activity compared with only three individuals in

the RLBP group, resulting in a trend towards a significant association between group and change in duration of deep multifidus activity (χ^2 = .058, Figure 4b).

There was no main effect of speed or group, or interaction of speed by group for lumbar longissimus across the stride cycle of the turn (Figure 5). Although the duration of lumbar longissimus activity increased in both groups during the swing phase of the turn at the faster speed (main effect of speed F (1, 26) = 14.109, p = .001), the change in the duration lumbar longissimus in response to increasing speed did not exceed the SEM for that muscle (1.51 %).

Duration of thoracic longissimus activity significantly increased at the faster speed in both groups (F (1, 26) = 6.09, p = .020, Figure 5) and the extent of this increase exceeded the SEM (0.75 %). Individual analyses of stance and swing phases indicated that the significant increase in duration of activity primarily occurred during swing phase (F (1, 26) = 12.542, p = .002). However, there was no main effect of group or group by speed interaction.

Amplitude of activity

The normalized amplitude of activity in the deep fibers of multifidus increased significantly from the self-selected to the fast speed. This change was evident during the stance phase (F (1, 26) = 9.67, p = .005) and within the swing phase (F (1, 26) = 16.36, p < .0001), but was not significantly different between groups (Figure 6). The extent of the increase in multifidus activity exceeded the SEM (0.001 mV). Normalized amplitude of activity in the lumbar longissimus and thoracic longissimus also significantly increased during stance and swing phases (LES stance F (1, 26) = 8.317, p = .008, swing F (1, 26) = 21.035, p =<.001;TES stance F (1,26) = 10.567, p = .003, swing F (1, 26) = 21.358, p

309 =<.000,Figure 6), but this change did not exceed the SEM in either case (LES = .27mV;
310 TES = .09 mV).

311

312 **Discussion**

313 This research demonstrates altered activation of the deep fibers of multifidus
314 during a locomotor task in people with recurrent LBP. In contrast with healthy
315 individuals, a majority of participants with a history of recurrent LBP responded to
316 increasing mechanical demand by reducing the duration of activity of the deep fibers of
317 multifidus. Impaired timing of the anticipatory activity of the deep multifidus muscle and
318 reduced amplitude of deep multifidus activity has previously been demonstrated in
319 asymptomatic individuals with recurrent LBP during standing postural perturbations and
320 voluntary trunk flexion[8,45]. Taken together, the results from the present and previous
321 studies suggest that changes in recruitment of the deep fibers of multifidus persist
322 between painful episodes in individuals with a history of LBP. The differences between
323 groups in this present study were small. However, it is striking that they were still evident
324 in a majority of young, asymptomatic individuals with a history of LBP, who had
325 minimal disability, low levels of fear avoidance and high self-efficacy. Additionally, it is
326 important to note that walking turns are a sub-maximal task for the paraspinal
327 musculature, with levels of muscle activity less than 20 % of maximum voluntary
328 contraction (Armour Smith & Kulig, unpublished data) and that walking is rarely a pain-
329 producing activity in individuals with LBP[3,46,47]. Therefore, it is likely that these
330 differences would be more pronounced during more demanding tasks. As there are
331 changes in the morphology and fatigability of the deep multifidus muscle in persons with

LBP[33,48,490] further research is needed to determine if this altered strategy is adaptive to compensate for altered morphology in the multifidus muscle or if it is a maladaptive consequence of pain. However, as changes in multifidus recruitment during anticipatory postural adjustments occur in response to anticipated experimental pain in healthy individuals, in the absence of any injury or muscle impairment, we propose that they represent a maladaptive postural control response.

This study did not find significant differences in the duration or amplitude of activity in the lumbar or thoracic longissimus in asymptomatic persons with a history of recurrent LBP in comparison with controls. This is in contrast to studies demonstrating increased erector spinae muscle activity in symptomatic individuals with chronic LBP[4,5]. Investigations of acute experimental LBP have also indicated increased amplitude of erector spinae activity during walking[20,50]. Taken together, the results from this present study and earlier work suggest that changes in postural trunk control during walking may form a continuum. Significant adaptations in superficial paraspinal muscle activity may be evident both acutely and persistently in response to concurrent pain but may not persist between symptomatic episodes during sub-maximal locomotor tasks. Clinically, this study adds valuable information regarding the timing of the development of the control changes that occur in association with LBP and how these changes are associated with symptoms. This is important to assist in effective sub-grouping of individuals with low back pain for the purposes of treatment and research and for determining when interventions targeting these impairments may be warranted.

All individuals in this study were able to complete the walking circuit at the faster, controlled speed. Interestingly, the asymptomatic individuals with a history of

recurrent LBP in this study did not have significantly different self-selected locomotor speed than the healthy individuals. This is in contrast with studies investigating steady-state locomotor speed in symptomatic individuals with chronic LBP that have consistently reported slower locomotion in the affected group[3,19,51]. This may be due to a number of factors. Unlike previous studies, the individuals with LBP in the present study were asymptomatic at the time of the data collection. Additionally, participants in this study were in their mid-twenties, whereas those in existing studies are at least a decade older[3,51]. However, they actually reported a longer duration of symptoms than either of the previously cited studies, suggesting that deficits in locomotor speed may be more related to current pain intensity than duration of symptoms.

On the whole, the activity of the paraspinal muscles during walking turns is consistent with previous studies investigating steady-state treadmill locomotion in healthy individuals[52,53]. Paraspinal activity during locomotion occurs at initial contact and during the double support phases of the locomotor cycle[4,54-58] and controls spinal flexion and side bending[54-58]. To our knowledge, the only study previously investigating trunk muscle activity during turning reported continuous activity of the erector spinae during 180° turns. The authors hypothesized that this activity helped to decelerate forward momentum and balance the trunk over the limb during the turn[49]. The more phasic activity evident in this present research is likely due to the turns in this study being both anticipated and of smaller amplitude. Observing the modulation in the activity in each muscle in response to increasing speed highlighted functional differentiation within the paraspinal group. The deep fibers of lumbar multifidus exhibited the most pronounced changes in response to greater mechanical demand, with an increase in both duration and

amplitude of activity at the faster speed. This is likely a reflection of the unique functional role of these fibers. The very small moment arm of the deepest fascicles of multifidus relative to the segmental axis of rotation in the sagittal plane suggests that the primary function of this portion of multifidus is control of spinal segmental motion via inter-segmental compression, rather than generation of torque[59,60]. As locomotor speed increases, ground reaction forces and, therefore, segmental shear forces increase[57]. The deep fibers of multifidus are ideally suited to control these segmental forces without generating large multi-segmental torques. In contrast, activity in lumbar longissimus was relatively unaffected by speed, while thoracic longissimus exhibited increased duration of activity only. More prolonged thoracic activity may be necessary to decelerate motion of the trunk on the pelvis at initial contact at the faster speed[55].

It is important to note that further research is necessary to clarify the relationship between altered paraspinal muscle activation in individuals with LBP and altered kinematic postural control strategies, in order to determine the mechanical consequences of changes in muscle activation. Additionally, although the integrated profile method of EMG activity onset/offset detection is the most appropriate analysis technique for postural trunk muscle data, like all EMG detection methods it is subject to the characteristics of the EMG signal and the task and must be utilized with careful visual checking to avoid anomalous results.

In both groups, increases in walking speed were associated with significant increases in duration of activity in the thoracic longissimus and amplitude of activity in the deep multifidus. However, this study demonstrated for the first time that even between symptomatic episodes, some young individuals with a history of recurrent LBP

demonstrate selectively altered modulation of the duration of deep multifidus activity in response to changing locomotor demands.

Key Points

Findings:

In comparison with healthy adults, young asymptomatic individuals with a history of recurrent low back pain demonstrated altered patterns of recruitment of the deep fibers of the lumbar multifidus muscle when increasing speed during walking turns.

Implications:

This study provides evidence of persistent alteration in the recruitment of lumbar multifidus muscle, even between symptomatic episodes of low back pain, and may help with the further development of targeted treatment approaches for individuals with low back pain.

Caution:

The individuals with a history of low back pain in this study were young and minimally disabled. The results may be different in an older or more disabled subject pool. Additionally, causality in the relationship between altered multifidus recruitment and recurrent low back pain cannot be determined by this study.

References

1. Freburger, JK. *et al.* The rising prevalence of chronic low back pain. *Arch Intern Med* 2009; 169:251–258.
2. Pengel L, Herbert RD, Maher, C. G. & Refshauge, K. M. Acute low back pain: systematic review of its prognosis. *BMJ* 2003; August: 1-5.
3. Lamoth, C. J. C., Meijer, O. G., Daffertshofer, A., Wuisman, P. I. J. M. & Beek, P. J. Effects of chronic low back pain on trunk coordination and back muscle activity during walking: changes in motor control. *Eur Spine J* 2005; **15:** 23–40.
4. van der Hulst, M. *et al.* Back muscle activation patterns in chronic low back pain during walking: a 'guarding' hypothesis. *Clin J Pain* 2010; 26: 30–37.
5. Vogt, L., Pfeifer, K. & Banzer, W. Neuromuscular control of walking with chronic low-back pain. *Man Ther* 2003; 8: 21–28.
6. Stanton, T. R., Latimer, J., Maher, C. G. & Hancock, M. J. How do we define the condition 'recurrent low back pain'? A systematic review. *Eur Spine J* 2009; 19: 533–539.
7. Wasiak, R., Kim, J. & Pransky, G. Work disability and costs caused by recurrence of low back pain: longer and more costly than in first episodes. *Spine* 2006; 31: 219–225.
8. MacDonald, D., Moseley, G. L. & Hodges, P. W. Why do some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. *Pain* 2009; 142: 183–188.
9. Seay, J. F., Van Emmerik, R. E. A. & Hamill, J. Influence of low back pain status on pelvis-trunk coordination during walking and running. *Spine* 2011; 36: E1070–E1079.
10. Seay, J. F., Van Emmerik, R. E. A. & Hamill, J. Low back pain status affects pelvis-trunk coordination and variability during walking and running. *Clin Biomech* 2011; 26: 572–578.
11. Henry, S. M., Hitt, J. R., Jones, S. L. & Bunn, J. Y. Decreased limits of stability in response to postural perturbations in subjects with low back pain. *Clin Biomech* 2006; 21: 881–892.
12. Jones, S. L., Hitt, J. R., DeSarno, M. J. & Henry, S. M. Individuals with non-specific low back pain in an active episode demonstrate temporally altered torque responses and direction-specific enhanced muscle activity following unexpected balance perturbations. *Exp Brain Res* 2012; 221: 413–426.
13. Radebold, A., Cholewicki, J., Panjabi, M. M. & Patel, T. C. Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. *Spine* 2000; 25: 947–954.
14. Silfies, S. P., Mehta, R., Smith, S. S. & Karduna, A. R. Differences in feedforward trunk muscle activity in subgroups of patients with mechanical low back pain. *Arch Phys Med Rehabil* 2009; 90: 1159–1169.
15. Macintosh, J. & Bogduk, N. 1987 Volvo Award in Basic Science: The Morphology of the Lumbar Erector Spinae. *Spine* 1987; 12: 658–666.
16. MacDonald, D. A., Lorimer Moseley, G. & Hodges, P. W. The lumbar multifidus: Does the evidence support clinical beliefs? *Man Ther* 2006; 11: 254–263.

17. Macintosh, J., Valencia, F., Bogduk, N. & Munro, R. The morphology of the human lumbar multifidus. *Clin Biomech* 1986*:* 196–204.
18. MacDonald, D., Moseley, G. L. & Hodges, P. W. People with recurrent low back pain respond differently to trunk loading despite remission from symptoms. *Spine* 2010; 35: 818–824.
19. Lamoth, C. J. C., Daffertshofer, A., Meijer, O. G. & Beek, P. J. How do persons with chronic low back pain speed up and slow down? Trunk–pelvis coordination and lumbar erector spinae activity during gait. *Gait Posture* 2006; 23: 230–239.
20. Arendt-Nielsen, L., Graven-Nielsen, T., Svarrer, H. & Svensson, P. The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. *Pain* 1996; 64: 231–240.
21. van der Hulst, M. *et al.* Back muscle activation patterns in chronic low back pain during walking: a 'guarding' hypothesis. *Clin J Pain* 2010; 26: 30–37.
22. Stokes, I., Henry, S. & Single, R. Surface EMG electrodes do not accurately record from lumbar multifidus muscles. *Clin Biomech* 2003; 18: 9–13.
23. Merletti, R. & Farina, D. Analysis of intramuscular electromyogram signals. *Philos Trans A Math Phys Eng Sci* 2009; 367: 357–368.
24. Solomonow, M. *et al.* Surface and wire EMG crosstalk in neighbouring muscles. *J Electromyogr Kinesiol* 1994; 4: 131–142.
25. Hof, A. L., Elzinga, H., Grimmius, W. & Halbertsma, J. P. K. Speed dependence of averaged EMG profiles in walking. *Gait Posture* 2002; 16: 78–86.
26. Akram, S. B., Frank, J. S. & Fraser, J. Effect of walking velocity on segment coordination during pre-planned turns in healthy older adults. *Gait Posture* 2010; 32: 211–214.
27. Taylor, M. J. D., Dabnichki, P. & Strike, S. C. A three-dimensional biomechanical comparison between turning strategies during the stance phase of walking. *Hum Movt Sci* 2005; 24: 558–573.
28. Hill, J. C. & Fritz, J. M. Psychosocial influences on low back pain, disability, and response to treatment. *Phys Ther* 2011; 91: 712–721.
29. George, S. Z., Valencia, C. & Beneciuk, J. M. A psychometric investigation of fear-avoidance model measures in patients with chronic low back pain. *J Orthop Sports Phys Ther* 2010; 40: 197–205.
30. Yamada, K. A. *et al.* The Low Back Activity Confidence Scale (LoBACS): preliminary validity and reliability. *Phys Ther* 2011; 91: 1592–1603.
31. Fritz, J. M. & Irrgang, J. J. A comparison of a modified Oswestry low back pain disability questionnaire and the Quebec back pain disability scale. *Phys Ther* 2001; 81: 776–788.
32. Carlsson, A. M. Assessment of chronic pain. I. Aspects of the reliability and validity of the visual analogue scale. *Pain* 1983; 16: 87–101.
33. Beneck, G. J., Baker, L. L. & Kulig, K. Spectral analysis of EMG using intramuscular electrodes reveals non-linear fatigability characteristics in persons with chronic low back pain. *J Electromyogr Kinesiol* 2013; 23: 70–77.
34. Smith, J. A. & Kulig, K. Does insertion of intramuscular electromyographic electrodes alter motor behavior during locomotion? *J Electromyogr Kinesiol* 2015; 25: 431–437.
35. Taylor, M. J. D., Strike, S. C. & Dabnichki, P. Strategies used for unconstrained

direction change during walking. *Percept Mot Skills* 2006; 102, 576–588.
36. Allison, G. T. Trunk muscle onset detection technique for EMG signals with ECG artefact. *J Electromyogr Kinesiol* 2003; 13: 209–216
37. Santello, M. & McDonagh, M. J. The control of timing and amplitude of EMG activity in landing movements in humans. *Exp. Physiol.* 1998; 83: 857–874.
38. Allison, G. T. & Henry, S. M. The influence of fatigue on trunk muscle responses to sudden arm movements, a pilot study. *Clin Biomech* 2002; 17: 414–417.
39. Cowan, S., Bennell, K. & Hodges, P. The test-retest reliability of the onset of concentric and eccentric vastus medialis obliquus and vastus lateralis electromyographic activity in a stair stepping task. *Phys Ther Sport* 2000; 1: 129–136.
40. Hodges, P. W. & Bui, B. H. A comparison of computer-based methods for the determination of onset of muscle contraction using electromyography. *Electroencephalogr Clin Neurophysiol* 1996; 101: 511–519.
41. Kiesel, K. B. *et al.* A comparison of select trunk muscle thickness change between subjects with low back pain classified in the treatment-based classification system and asymptomatic controls. *J Orthop Sports Phys Ther* 2007; 37:596–607.
42. King, M. T. A point of minimal important difference (MID): a critique of terminology and methods. *Expert Review of Pharmacoeconomics & Outcomes Research* 2011; 11: 171–184.
43. Price, D. D., McGrath, P. A., Rafii, A. & Buckingham, B. The validation of visual analogue scales as ratio scale measures for chronic and experimental pain. *Pain* 1983; 17: 45–56.
44. Yamada, K. A. *et al.* The Low Back Activity Confidence Scale (LoBACS): Preliminary Validity and Reliability. *Phys Ther* 2011; 91:1592–1603.
45. D'hooge, R. *et al.* Altered trunk muscle coordination during rapid trunk flexion in people in remission of recurrent low back pain. *J Electromyogr Kinesiol* 2013; 23: 173–181.
46. Lamoth, C., Beek, P. & Meijer, O. Pelvis-thorax coordination in the transverse plane during gait. *Gait Posture* 2002; 16: 101–114.
47. Taylor, N. F., Evans, O. M. & Goldie, P. A. The effect of walking faster on people with acute low back pain. *Eur Spine J* 2003; 12: 166–172.
48. Hides, J. A., Stokes, M. J., Saide, M., Jull, G. A. & Cooper, D. H. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. *Spine* 1994; 19: 165–172.
49. Beneck, G. J. & Kulig, K. Multifidus atrophy is localized and bilateral in active persons with chronic unilateral low back pain. *Arch Phys Med Rehabil* 2012; 93: 300–306.
50. Lamoth, C. J. C. *et al.* Effects of experimentally induced pain and fear of pain on trunk coordination and back muscle activity during walking. *Clin Biomech* 2004; 19: 551–563.
51. Lee, C. E., Simmonds, M. J., Etnyre, B. R. & Morris, G. S. Influence of pain distribution on gait characteristics in patients with low back pain: part 1: vertical ground reaction force. *Spine* 2007; 32: 1329–1336.
52. Thorstensson, A., Carlson, H., Zomlefer, M. R. & Nilsson, J. Lumbar back muscle activity in relation to trunk movements during locomotion in man. *Acta Physiol.*

Scand. 1982; 116: 13–20.
53. Saunders, S. W., Rath, D. & Hodges, P. W. Postural and respiratory activation of the trunk muscles changes with mode and speed of locomotion. *Gait Posture* 2004; 20: 280–290.
54. White, S. G. & McNair, P. J. Abdominal and erector spinae muscle activity during gait: the use of cluster analysis to identify patterns of activity. *Clin Biomech* 2002; 17: 177–184.
55. Saunders, S. W., Schache, A., Rath, D. & Hodges, P. W. Changes in three dimensional lumbo-pelvic kinematics and trunk muscle activity with speed and mode of locomotion. *Clin Biomech* 2005; 20: 784–793.
56. Thorstensson, A., Carlson, H., Zomlefer, M. R. & Nilsson, J. Lumbar back muscle activity in relation to trunk movements during locomotion in man. *Acta Physiol. Scand* 1982; 116:13–20.
57. Callaghan, J. P., Patla, A. E. & McGill, S. M. Low back three-dimensional joint forces, kinematics, and kinetics during walking. *Clin Biomech* 1999; 14; 203–216.
58. Anders, C. *et al.* Trunk muscle activation patterns during walking at different speeds. *J Electromyogr Kinesiol* 2007; 17: 245–252.
59. Ward, S. R. *et al.* Architectural Analysis and Intraoperative Measurements Demonstrate the Unique Design of the Multifidus Muscle for Lumbar Spine Stability. *J Bone Joint Surg Am* 2009; 91: 176-185.
60. Bogduk, N. *et al.* A universal model of the lumbar back muscles in the upright position. *Spine* 1992; 17; 897–913.

588 **TABLE 1**. Participant demographics (median ± inter-quartile range)

	CTRL N=14*	RLBP N=14*	p
Age (years)	24.5 ± 1.75	26.5 ± 4.75	.068
Height (m)	1.73 ± 0.05	1.73 ± 0.09	.664
Mass (kg)	66.68 ± 14.97	67.70 ± 23.42	.152
PAS score (MET-time)	47.60 ± 5.00	48.20 ± 7.55	.470

589 CTRL = control group; RLBP = recurrent low back pain group; *both groups

590 comprised 8 females, 6 males

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FIGURE 1A & B. Frontal plane schematic of the deep fibers of the lumbar multifidus, lumbar longissimus and thoracic longissimus muscles; **FIGURE 1C.** Axial ultrasound images (transverse plane projections) showing location of electrode placements at L4 (deep multifidus and lumbar longissimus) and T10 (thoracic longissimus), SP = spinous process. The locations of asterisks on figure 1 (a) and (b) correspond to the level of electrode insertions shown also as asterisks in figure 1 (c). Note that all insertions were made on the same side, but are shown here on different sides for clarity.

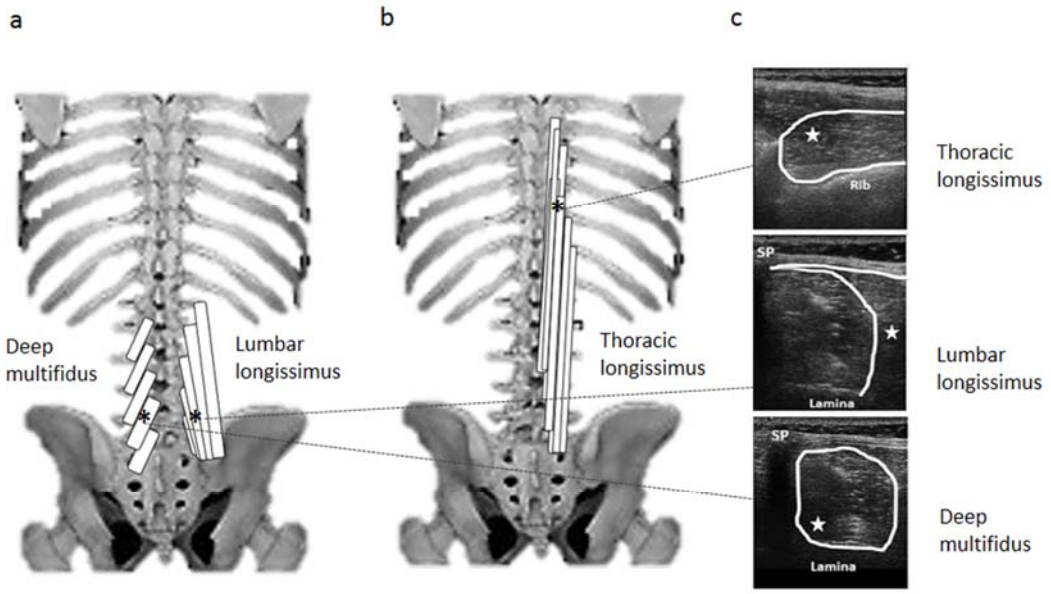


FIGURE 2A. Walking circuit, set up for participant instrumented on the left side, with turning area for the ipsilateral pivot turn indicated; **FIGURE 2B**. Stride cycle of an ipsilateral pivot turn, commencing with the initial contact of the foot ipsilateral to the turn direction. Participant instrumented on the left side and therefore turning towards the right.

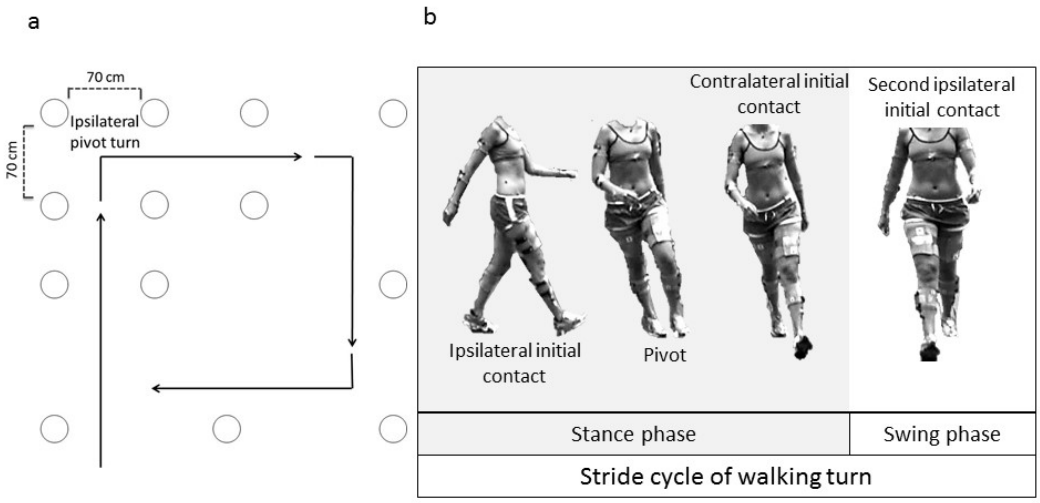
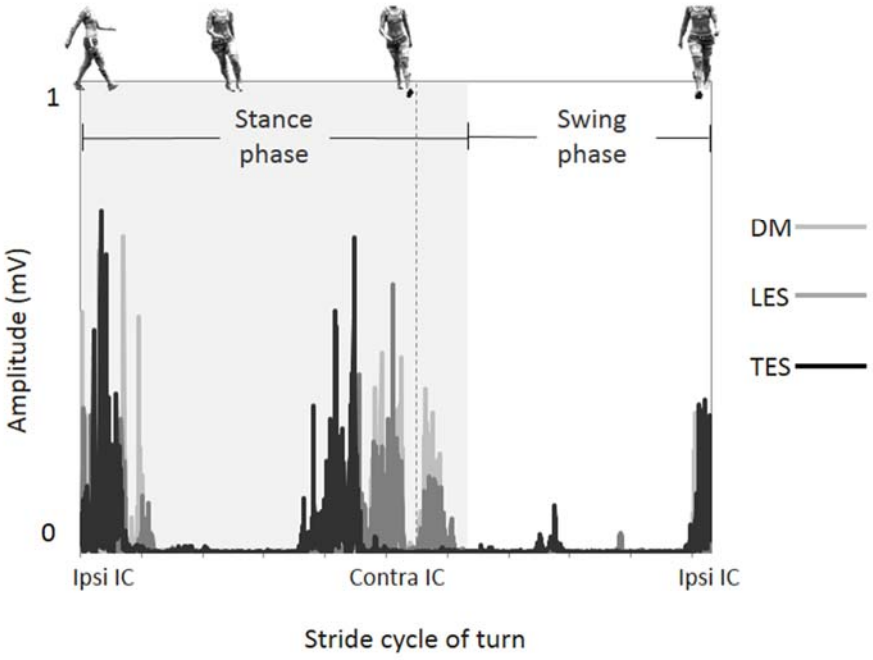
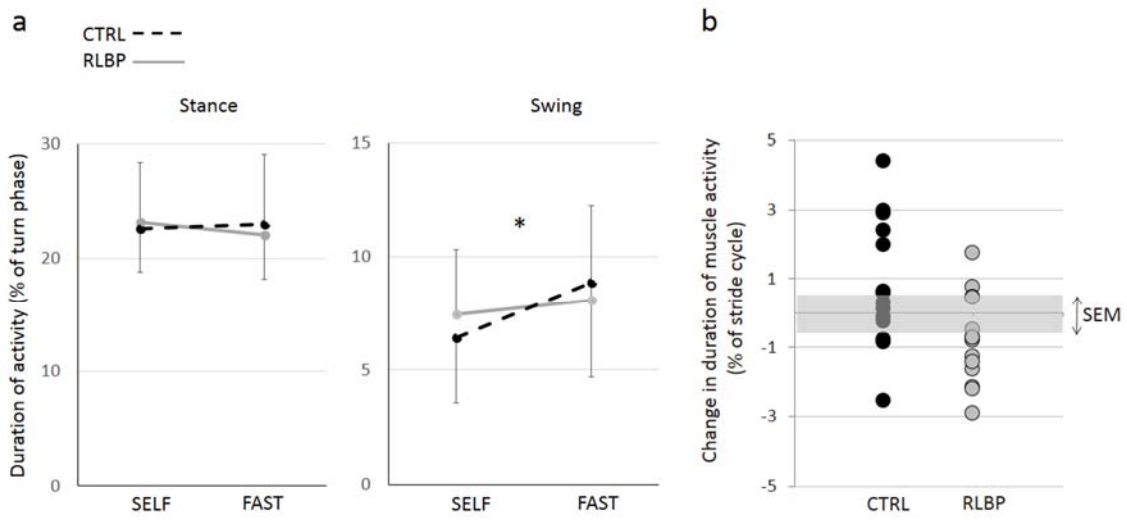


FIGURE 3. Exemplar EMG signals from one representative participant demonstrating primary patterns of muscle activity (individual trial, bandpass filtered and rectified signal, Ipsi IC = initial contact of the limb ipsilateral to the turning direction, Contra IC and dashed line = initial contact of the limb contralateral to the turning direction, DM = deep fibers of multifidus, LES = lumbar longissimus, TES = thoracic longissimus). Nineteen of the 28 participants exhibited this clear primary pattern of biphasic bursts of activity in all three muscles, beginning just prior to ipsilateral and contralateral initial contact. All 28 participants demonstrated this pattern of activity in the deep multifidus, and all participants except one also had activity at ipsilateral and contralateral initial contact in the lumbar longissimus. Six participants had a more unilateral pattern of TES activation, evident by a lack of activity at initial contact of the foot on the same side as the EMG instrumentation.





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631 **FIGURE 4A**. Duration of multifidus activity as a percentage of stance and swing phases
632 at the self-selected speed (SELF) and fast speed (FAST) in healthy participants (CTRL)
633 and individuals with a history of recurrent low back pain (RLBP). Error bars indicated
634 standard deviation. * Indicates significant interaction between speed and group. **FIGURE**
635 **4B**. Individual change in the duration of deep multifidus activity from the self-selected to
636 the fast walking speed across the stride cycle. The standard error of the measurement
637 (SEM) is outlined in gray. Nine of the fourteen individuals in the back pain group had a
638 decrease in the duration of deep multifidus activity that exceeded the SEM (the
639 measurement error) compared with only three individuals in the control group.

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FIGURE 5. Average duration of lumbar longissimus and thoracic longissimus activity during stance and swing phases at the self-selected speed (SELF) and fast speed (FAST) in healthy participants (CTRL) and individuals with a history of recurrent low back pain (RLBP). * Indicates significant main effect of speed, but magnitude of change was smaller than the SEM. ** Indicates significant main effect of speed, with an extent of change that was larger than the SEM.

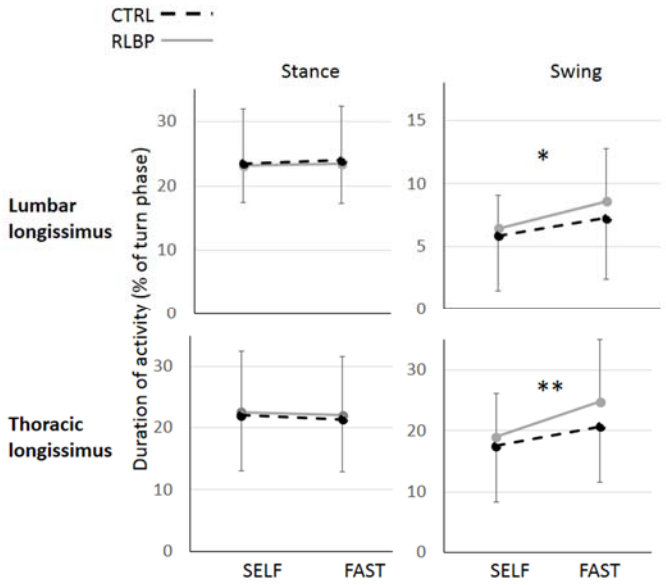

FIGURE 6 Average normalized amplitude of activity during stance and swing phases at the self-selected speed (SELF) and fast speed (FAST) in healthy participants (CTRL) and individuals with a history of recurrent low back pain (RLBP). Top - deep fibers of multifidus; middle - lumbar longissimus; bottom - thoracic longissimus. * Indicates significant main effect of speed, but magnitude of change was smaller than the SEM. ** Indicates significant main effect of speed, with an extent of change that was larger than the SEM.