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

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1 ABSTRACT

2

3 STUDY DESIGN: Cross sectional, laboratory study

4

5 BACKGROUND: Individuals with low back pain have impaired activation of multifidus during
6 postural adjustments and increased activity of the erector spinae musculature during walking.

7 However, it is unclear if these alterations in muscle activity are evident during locomotion in
8 individuals with a history of low back pain when they are between symptomatic episodes.

9

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

12

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

17

18 RESULTS: There was a significant speed by group interaction for the duration of multifidus
19 activity ($p = .013$). Duration of activity increased from the self-selected to the fast locomotor
20 speed in the controls, but decreased in the individuals with a history of low back pain ($p = .003$).

21 Self-selected speed was the same in both groups ($p = .719$). There was a trend towards a

22 significant association between group and the direction of change in the duration of deep

23 multifidus activity ($\chi^2 = .058$). Duration of thoracic longissimus activity and amplitude of

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

26

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

30

31

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

33

34 **Background**

35 Despite substantial research, and escalating health care costs over the past few
36 decades, the mechanisms underlying the transition from acute to persistent LBP are still
37 not well understood or effectively managed^{1,2}. The majority of back pain research to date
38 has focused on individuals who experience chronic, largely unremitting pain (chronic
39 LBP)³⁻⁵. However, there is increasing recognition that there is a distinct sub-group of
40 individuals with persistent LBP who experience an episodic or recurrent pattern of
41 symptoms⁶. In these individuals, successive episodes of LBP become longer and more
42 likely to require absence from work and medical intervention over time⁷. In the absence
43 of clear precipitating events or significant patho-anatomical dysfunction, it is often
44 unclear why these individuals experience recurrences of their back pain following periods
45 of time when they are entirely symptom-free. However, persistent and maladaptive
46 alterations in dynamic trunk postural control may contribute to this recurrence^{6,8-10}. In
47 order to understand the development and persistence of both recurrent and chronic LBP,
48 and to identify appropriate interventions, it is vital to clarify if changes in trunk postural
49 control are an adaptive response to concurrent symptoms or if they reflect a persistent and
50 maladaptive change in motor control. This can be ascertained by investigating individuals
51 with recurrent LBP during the periods of time when they are asymptomatic^{7,11}.

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

57 lumborum and longissimus thoracis pars lumborum, hereafter termed “lumbar
58 longissimus”) and the transversospinales (of which the multifidus is the major
59 component).¹⁵ The lumbar multifidus is commonly further subdivided into the deep
60 portion of the muscle, with fibers that extend across only two vertebral segments, and
61 superficial portion of the muscle, with fibers that cross up to five vertebrae¹⁶⁻¹⁸. Similarly,
62 in the thoracic region the paraspinals comprise the erector spine (spinalis, iliocostalis
63 thoracis and longissimus thoracis pars thoracis, hereafter termed “thoracic longissimus”)
64 and the transversospinales. Changes in paraspinal control in individuals with low back
65 pain include delayed and decreased activation in the deep fibers of the lumbar multifidus
66 muscle⁸ and task- and subject-dependent modifications in the timing and amplitude of
67 activity of the lumbar and thoracic erector spinae¹²⁻¹⁴. Impaired dynamic trunk postural
68 control is also evident in symptomatic individuals with LBP during locomotion. Studies
69 of treadmill walking utilizing surface electromyography have demonstrated increased
70 duration and amplitude of activity in the erector spinae during locomotion in persons with
71 chronic LBP^{3-5,19-21}. To date it is unclear if these same changes in erector spinae function
72 during walking are evident in individuals with a history of recurrent LBP during periods
73 of time when they are asymptomatic. It is also unclear if, there are impairments in the
74 recruitment of the deep fibers of the lumbar multifidus during walking in individuals with
75 LBP.

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

80 comprising the paraspinal group²². Therefore it is not known if the relative contribution
81 of the individual muscles to this increase in activity is the same, or whether individuals
82 with LBP have altered distribution of activity across the paraspinal group. This problem
83 can be overcome by utilizing fine-wire intramuscular EMG electrodes^{23,24}. In the lower
84 limbs, modulation of muscle activity in response to increasing locomotor speed
85 encompasses both shifts in timing and changes in amplitude, and the pattern of these
86 modulations is muscle specific²⁵. Therefore, investigating temporal and spatial
87 adaptations to increasing locomotor speed may help to elucidate functional differences in
88 control of the paraspinals in individuals with a history of low back pain. Postural demand
89 in the trunk during locomotion is also greater during functional locomotor perturbations
90 such as walking turns, particularly in the upper trunk^{26,27}. Thus, walking turns may
91 provide an excellent paradigm for differentiating between activity in the lumbar and
92 thoracic regions of the paraspinals in healthy individuals and those with a history of back
93 pain.

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

100

101 **Methods**

102 **Participants**

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

118 Participants were eligible for inclusion in the RLBP group if they; 1) were
119 between 18 and 40 years of age; 2) had a history of more than one year of recurrent
120 episodes of LBP; 3) had primarily unilateral pain localized to the area between the
121 twelfth rib and the gluteal fold; 4) reported at least two pain episodes of at least 24 hours'
122 duration in the preceding year⁶; 5) had pain episodes that were severe enough to limit
123 function; and 6) were in symptom remission at the time of the data collection (defined as
124 a score of less than 0.5/10 cm on a visual analogue scale for current pain at the start of the
125 data collection). Participants were excluded if they had contraindications to intramuscular

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

133 Participants with a history of RLBP also completed several questionnaires to
134 assess potential psychosocial influences on LBP and motor behavior²⁸. Fear avoidance
135 beliefs were quantified using the Fear Avoidance Beliefs Questionnaire (FABQ²⁹). Self-
136 efficacy was quantified using the Low Back Activity Confidence Scale (LoBACS³⁰).
137 Disability due to LBP was quantified using the modified Oswestry Disability Index
138 (ODI³¹). In addition, all participants completed visual analogue scales for current pain at
139 the beginning of the data collection and for pain experienced during the walking turns at
140 the end of the data collection (VAS)³².

141 **Instrumentation**

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

149 same side for the matched healthy participant. Depth of insertion was subject-specific and
150 based on ultrasound visualization of the tip of the needle in the muscle. The correct
151 electrode placement was confirmed by observing the contraction induced by light
152 electrical stimulation using ultrasound imaging³³. We have previously demonstrated that
153 this methodology is associated with minimal pain or change in locomotor kinematics in
154 both healthy individuals and individuals with a history of LBP³⁴.

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

164 **Experimental task**

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

172 complete the turn, with the change in direction being accomplished by a pivot on the
173 stance foot³⁵. For consistency, all participants turned contralateral to the side of their
174 EMG instrumentation (contralateral to the symptomatic side in the RLBP group and to
175 the matched side in the CTRL group). Therefore, the stance phase of the turn occurred on
176 the limb contralateral to the side of the electrodes. Although preliminary data indicated
177 minimal differences in EMG variables between turn directions, turns contralateral to the
178 instrumentation were selected in order to maximize erector spinae activity at initial
179 contact. Prior to data collection, participants practiced the walking circuit until they were
180 consistently able to turn with the correct foot in the correct area without altering stride
181 length or changing cadence.

182 **Data processing**

183 15 trials were analyzed for each participant at each speed. The first 15 clean trials
184 were selected for analysis for all individuals. Trials were excluded if the participant
185 performed the turn incorrectly. Timing of locomotor events was determined using the
186 foot switches and all data were analyzed across the stride cycle of the turn, from the
187 initial contact of the limb ipsilateral to the turning direction to the next initial contact of
188 the same limb. EMG data were processed in MATLAB[®] (MathWorks, MA, USA). After
189 removal of the DC offset, the EMG signals were band-pass filtered (40 Hz – 1500 Hz,
190 digital zero-phase Butterworth filter) and full-wave rectified.

191 **Data analysis**

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

195 noise³⁶. It results in fewer errors than standard threshold detection protocols when
196 determining postural trunk muscle activity as it is not dependent upon baseline activity or
197 the rate of signal increase³⁶.

198 The amplitude of each signal was first continuously integrated across the stride
199 cycle and normalized so that the final value was 1. The time of the stride cycle for each
200 individual trial was also normalized to 1. The integrated signal was then subtracted from
201 a reference line with a slope of 1, that reflects the hypothetical condition where the
202 muscle activity remains constant across the time-series of the trial³⁷. The local maxima
203 and minima of the deviations of the actual integrated signal from the reference line was
204 then used to determine the timing of onset or offset³⁸. The algorithm was implemented
205 with a visual check of the detected onset and offset events superimposed over the
206 rectified/band-passed signal to ensure appropriate determination^{36,39,40}. The duration of
207 the muscle burst occurring between each onset and offset event was calculated, and the
208 sum of the duration of all bursts across each stride cycle, stance phase and swing phase
209 was calculated and expressed as a percentage of the total duration of the stride cycle,
210 stance phase and swing phase for that trial. The average amplitude of activity in each
211 muscle was also calculated across the stride cycle and within the stance and swing phases
212 individually for each turn at each speed. The stance phase and swing phase values were
213 then amplitude normalized for each participant to the average value across the stride
214 cycle during turns performed at the self-selected speed.

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

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

224
$$SEM = s\sqrt{1 - ICC}^{42}$$

225 **Statistical analysis**

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

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

243

244 **Results**

245 Median \pm interquartile range FABQ score (physical activity subscale) in the
246 RLBP group was 12.50 ± 6.75 . Median \pm interquartile range LoBACS score in the RLBP
247 group was 88 ± 12.83 , which is higher than previously reported values in a LBP
248 population^{3,44}. Median \pm interquartile range ODI score was 18.0 ± 15.0 % indicating
249 minimal disability. At baseline, average \pm standard deviation current pain was $0.12 \pm$
250 0.24 cm in the participants with a history of recurrent LBP and 0 cm in all of the controls.

251 One individual who reported pain of less than 0.5 during the subjective screening
252 procedures completed a VAS that was measured as 0.8 at the commencement of the data
253 collection (after the physical examination). The decision was made to include his data as
254 this value is well below the minimal detectable change for the VAS. During the
255 locomotor trials participants reported low levels of discomfort associated with the
256 intramuscular EMG electrodes (RLBP 0.50 ± 0.70 cm, CTRL 0.45 ± 0.70 cm, $p = .779$).
257 Reliability was excellent, with ICC values exceeding 0.85 for all variables except the
258 duration of activity in the thoracic longissimus.

259 **Self-selected locomotor speed and locomotor events**

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

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

268 **Overview of paraspinal activity during walking turns**

269 Exemplar EMG data and an overview of paraspinal activity are provided in Figure
270 3.

271 **Duration of activity**

272 Total duration of activity in each muscle during stance and swing phase at each
273 speed is shown in Figure 4a and Figure 5. There was a significant speed by group
274 interaction for the duration of deep multifidus activity ($F(1, 26) = 7.186, p = .013$, Figure
275 4a), but no main effect of speed or group ($F(1, 26) = .006, p = .938$; $F(1, 26) = .021, p =$
276 $.886$ respectively). Post-hoc comparisons indicated that there was a trend towards a significant
277 decrease in duration from self-selected to fast speed in the RLBP group ($p = .04$, Cohen's
278 $d = 0.23$) and that the average duration of activity across the stride cycle increased in the
279 CTRL participants but decreased in the RLBP participants (average change from SELF to
280 FAST, CTRL $+0.84 \pm 1.87\%$, RLBP $-0.79 \pm 1.30\%$, $p = .003, d = 1.01$). This
281 difference exceeded the SEM (0.56 % of stride cycle). Analyses of stance and swing
282 phase individually indicated that this interaction effect was significant during swing
283 phase (swing phase speed by group interaction $F(1, 26) = 4.861, p = .037$), but not
284 during stance ($F(1, 26) = 2.467, p = .128$) Eight of the individuals in the CTRL group
285 demonstrated an increase in duration of activity compared with only three individuals in

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

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

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

300 **Amplitude of activity**

301 The normalized amplitude of activity in the deep fibers of multifidus increased
302 significantly from the self-selected to the fast speed. This change was evident during the
303 stance phase ($F(1, 26) = 9.67$, $p = .005$) and within the swing phase ($F(1, 26) = 16.36$, p
304 $< .0001$), but was not significantly different between groups (Figure 6). The extent of the
305 increase in multifidus activity exceeded the SEM (0.001 mV). Normalized amplitude of
306 activity in the lumbar longissimus and thoracic longissimus also significantly increased
307 during stance and swing phases (LES stance $F(1, 26) = 8.317$, $p = .008$, swing $F(1, 26) =$
308 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

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

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

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

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

365 On the whole, the activity of the paraspinal muscles during walking turns is
366 consistent with previous studies investigating steady-state treadmill locomotion in
367 healthy individuals^{52,53}. Paraspinal activity during locomotion occurs at initial contact and
368 during the double support phases of the locomotor cycle^{4,54-58} and controls spinal flexion
369 and side bending⁵⁴⁻⁵⁸. To our knowledge, the only study previously investigating trunk
370 muscle activity during turning reported continuous activity of the erector spinae during
371 180° turns. The authors hypothesized that this activity helped to decelerate forward
372 momentum and balance the trunk over the limb during the turn⁴⁹. The more phasic
373 activity evident in this present research is likely due to the turns in this study being both
374 anticipated and of smaller amplitude. Observing the modulation in the activity in each
375 muscle in response to increasing speed highlighted functional differentiation within the
376 paraspinal group. The deep fibers of lumbar multifidus exhibited the most pronounced
377 changes in response to greater mechanical demand, with an increase in both duration and

378 amplitude of activity at the faster speed. This is likely a reflection of the unique
379 functional role of these fibers. The very small moment arm of the deepest fascicles of
380 multifidus relative to the segmental axis of rotation in the sagittal plane suggests that the
381 primary function of this portion of multifidus is control of spinal segmental motion via
382 inter-segmental compression, rather than generation of torque^{59,60}. As locomotor speed
383 increases, ground reaction forces and, therefore, segmental shear forces increase⁵⁷. The
384 deep fibers of multifidus are ideally suited to control these segmental forces without
385 generating large multi-segmental torques. In contrast, activity in lumbar longissimus was
386 relatively unaffected by speed, while thoracic longissimus exhibited increased duration of
387 activity only. More prolonged thoracic activity may be necessary to decelerate motion of
388 the trunk on the pelvis at initial contact at the faster speed⁵⁵.

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

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

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

403

404 **Key Points**

405

406 **Findings:**

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

410

411 **Implications:**

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

416

417 **Caution:**

418 The individuals with a history of low back pain in this study were young and minimally
419 disabled. The results may be different in an older or more disabled subject pool.

420 Additionally, causality in the relationship between altered multifidus recruitment and

421 recurrent low back pain cannot be determined by this study.

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

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

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

590 comprised 8 females, 6 males

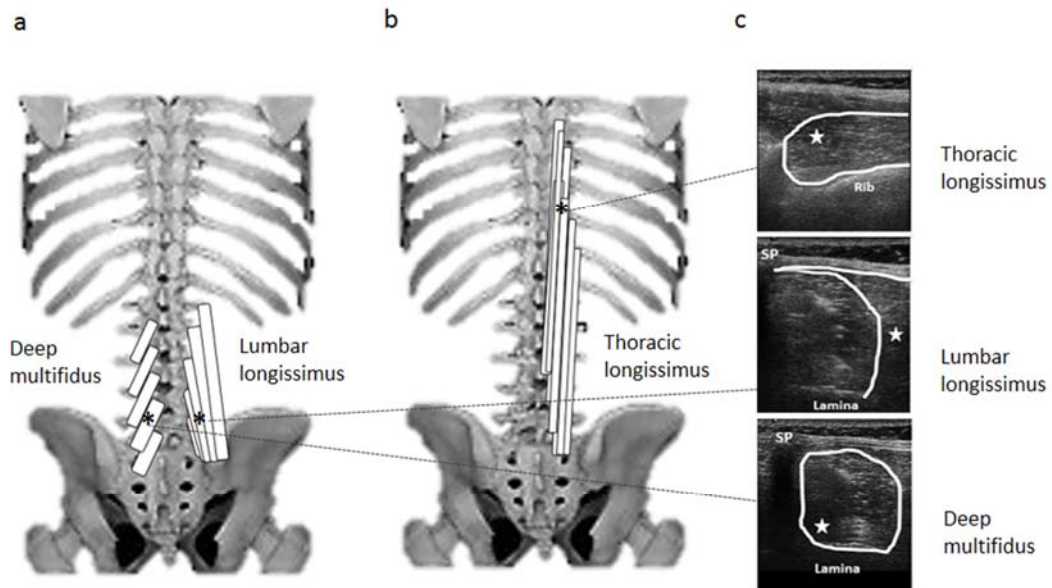
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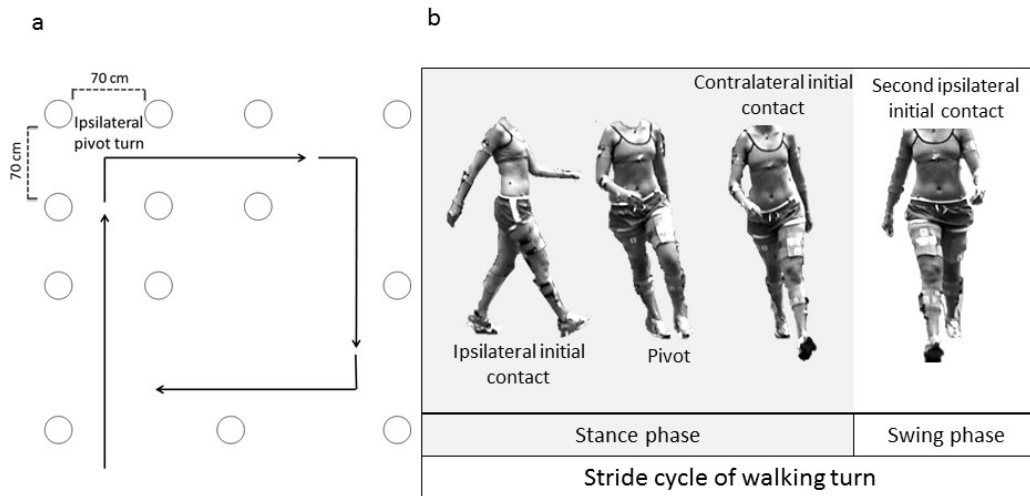
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595 **FIGURE 1A & B.** Frontal plane schematic of the deep fibers of the lumbar multifidus,
596 lumbar longissimus and thoracic longissimus muscles; **FIGURE 1C.** Axial ultrasound
597 images (transverse plane projections) showing location of electrode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



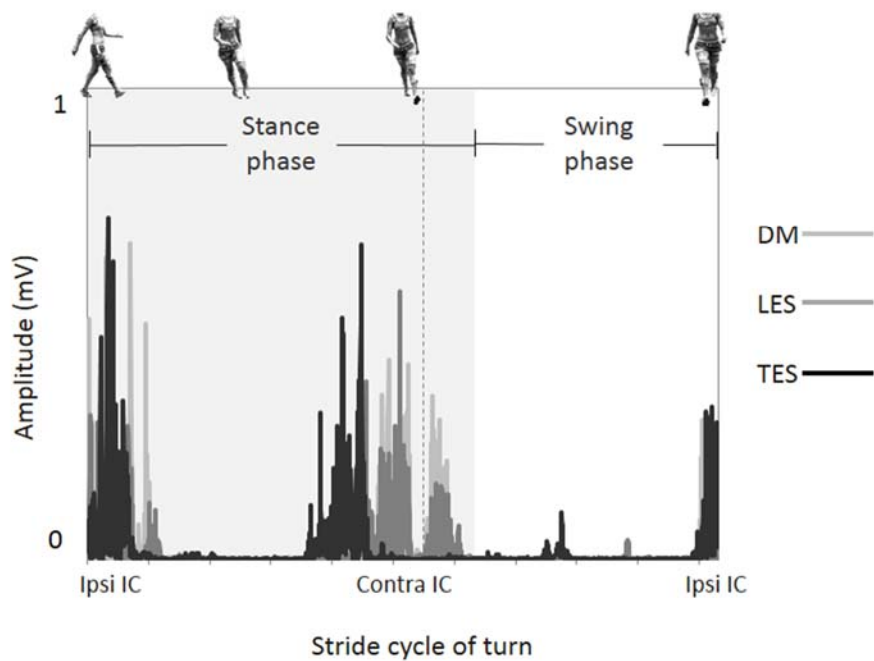
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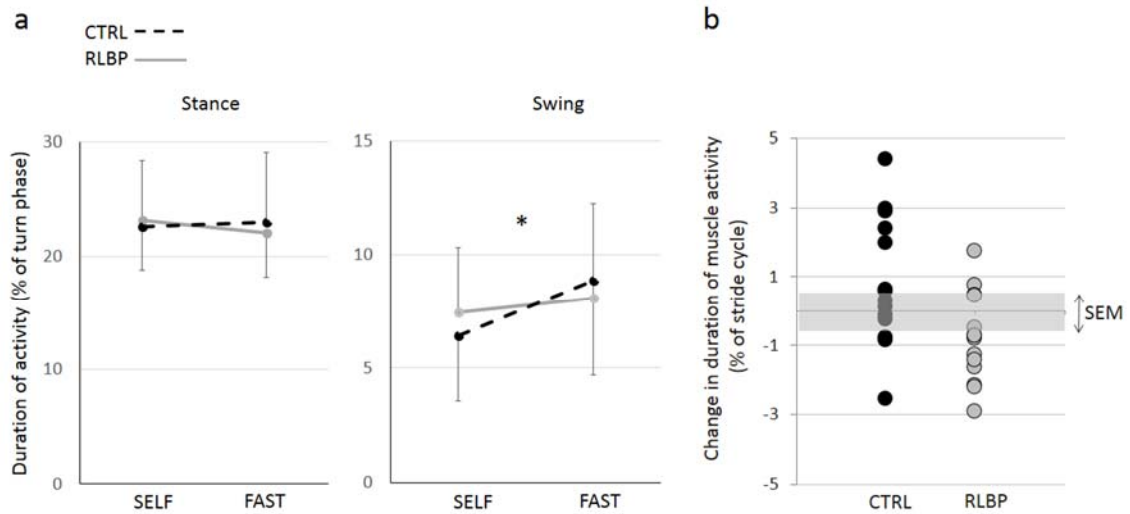
607 **FIGURE 2A.** Walking circuit, set up for participant instrumented on the left side, with
 608 turning area for the ipsilateral pivot turn indicated; **FIGURE 2B.** Stride cycle of an
 609 ipsilateral pivot turn, commencing with the initial contact of the foot ipsilateral to the turn
 610 direction. Participant instrumented on the left side and therefore turning towards the right.
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617 **FIGURE 3.** Exemplar EMG signals from one representative participant demonstrating
618 primary patterns of muscle activity (individual trial, bandpass filtered and rectified signal,
619 Ipsi IC = initial contact of the limb ipsilateral to the turning direction, Contra IC and
620 dashed line = initial contact of the limb contralateral to the turning direction, DM = deep
621 fibers of multifidus, LES = lumbar longissimus, TES = thoracic longissimus). Nineteen
622 of the 28 participants exhibited this clear primary pattern of biphasic bursts of activity in
623 all three muscles, beginning just prior to ipsilateral and contralateral initial contact. All
624 28 participants demonstrated this pattern of activity in the deep multifidus, and all
625 participants except one also had activity at ipsilateral and contralateral initial contact in
626 the lumbar longissimus. Six participants had a more unilateral pattern of TES activation,
627 evident by a lack of activity at initial contact of the foot on the same side as the EMG
628 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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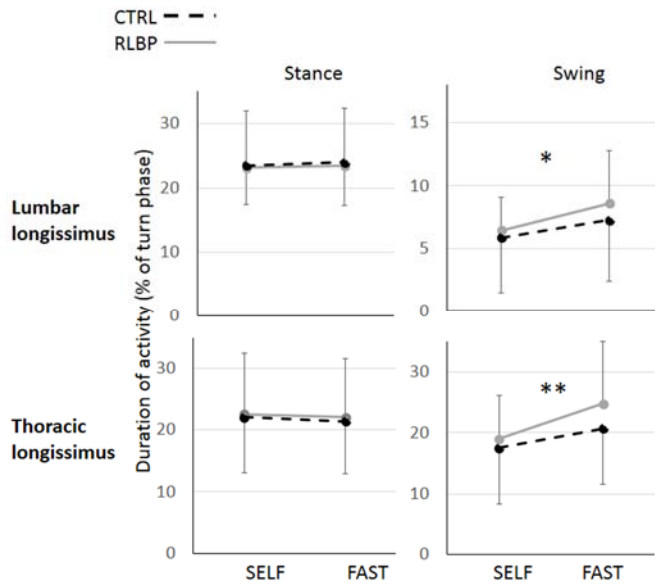
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645 **FIGURE 5.** Average duration of lumbar longissimus and thoracic longissimus activity
646 during stance and swing phases at the self-selected speed (SELF) and fast speed (FAST)
647 in healthy participants (CTRL) and individuals with a history of recurrent low back pain
648 (RLBP). * Indicates significant main effect of speed, but magnitude of change was
649 smaller than the SEM. ** Indicates significant main effect of speed, with an extent of
650 change that was larger than the SEM.



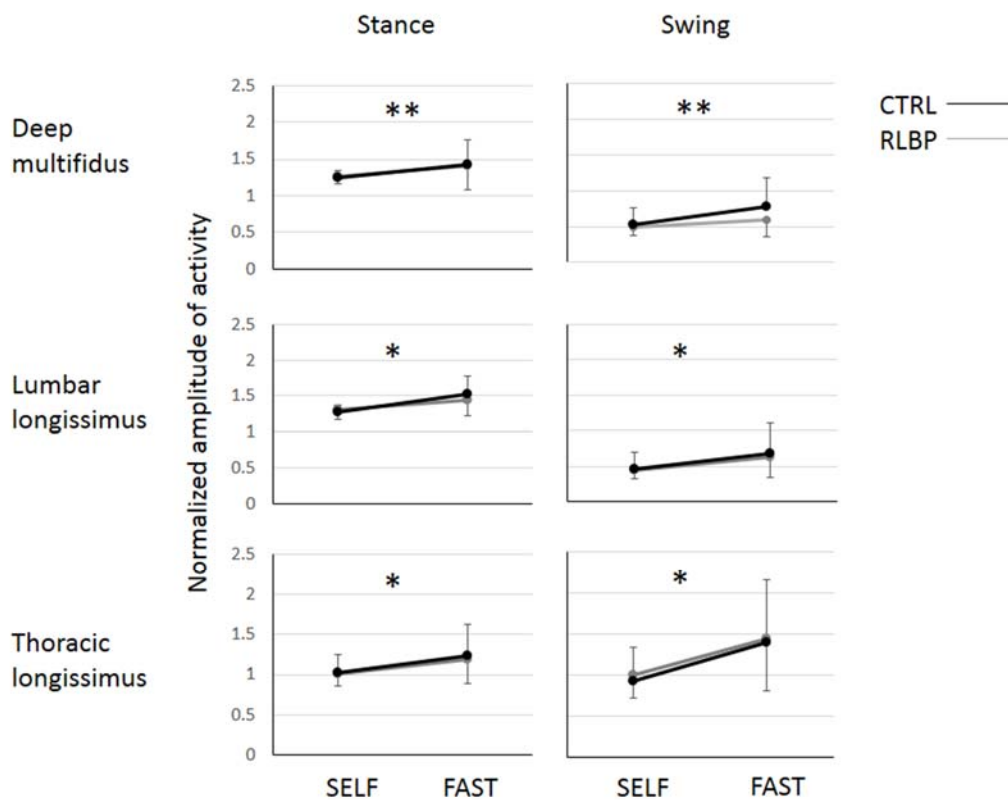
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655 **FIGURE 6** Average normalized amplitude of activity during stance and swing phases at
656 the self-selected speed (SELF) and fast speed (FAST) in healthy participants (CTRL) and
657 individuals with a history of recurrent low back pain (RLBP).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



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