



## Original research

# Rowers with a recent history of low back pain engage different regions of the lumbar erector spinae during rowing

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## ARTICLE INFO

## Article history:

Received 5 October 2018

Received in revised form 13 June 2019

Accepted 16 July 2019

Available online 19 July 2019

## Keywords:

Rowing

Low back pain

High-density surface electromyography

Erector spinae

## ABSTRACT

**Objectives:** Despite the high prevalence of low back pain (LBP) in rowers, there are few studies investigating changes in lumbar muscle activation in rowers with a recent history of LBP. Such knowledge is relevant to understand potential mechanisms contributing to the maintenance and recurrence of LBP in rowers. For the first time, we evaluate the spatial distribution of erector spinae (ES) activity in rowers with and without a recent history of LBP, using a novel application of high-density surface electromyography (HDEMG).

**Design:** Cross-sectional study.

**Methods:** Asymptomatic rowers ( $N=10$ ) and rowers with a recent history of LBP ( $N=8$ ) performed  $7 \times 4$ -min exercise bouts (rowing ergometer) until volitional exhaustion. HDEMG signals were acquired bilaterally over the lumbar ES and the root mean square (RMS) amplitude and entropy were analyzed. In addition, the y-axis coordinate of the barycentre (RMS-map) was used to assess changes in ES spatial activation.

**Results:** As the load increased, rowers with LBP showed higher amplitude ( $p < 0.01$ ) and less complexity (entropy) of the HDEMG signals ( $p < 0.001$ ). In addition, rowers with LBP showed opposite displacements of the barycentre, specifically showing a caudal shift of muscle activity at high intensities ( $p < 0.001$ ).

**Conclusions:** Both the magnitude of activation and distribution of ES activity were altered in rowers with a recent history of LBP. The lower complexity of signals together with the caudal displacements of the barycentre suggest an inefficient recruitment of the ES as the load progressed. Modification of the rowing technique in conjunction with feedback from HDEMG might prove useful in future studies.

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## Practical implications

- During an incremental rowing test, rowers with a recent history of low back pain show increased activation of the lumbar erector spinae.
- This increase in activation was accompanied by recruitment of mechanically inefficient portions of the lumbar erector spinae (lower regions of the lumbar erector spinae) at high loads.
- Rowers with a history of low back pain show more homogeneous activation of the lumbar erector spinae compared to controls,

which likely relates to preferential recruitment of a smaller portion of the muscle as the load progresses.

## 1. Introduction

Rowing is a sport with a high incidence of low back pain (LBP), with annual rates of occurrence between 32% and 51%.<sup>1,2</sup> Rowing is defined by cyclical lumbar flexion through the 'drive' phase of the stroke when the rower applies work and the hips, knees and trunk move from full flexion to relative extension. The 'recovery' phase of the stroke is when no work is applied and the rower moves from relative extension back to full flexion to another work cycle; in a boat, this is when the oars are out of the water. It has been recognized that cyclical lumbar flexion, particularly when combined with fatigue, may alter joint mechanics and loading patterns of the

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lumbar spine, possibly leading to risk of tissue failure and injury.<sup>3</sup> In the latest review of Wilson et al., it was discussed that the volume of training on an ergometer is one of the most important risk factors for LBP.<sup>1</sup> Studies have shown that flexion of the lumbar spine is increased by approximately 10% at the end of an incremental test in a rowing ergometer compared to that of the rowing boat.<sup>4</sup> High-intensity rowing on an ergometer also increases L5/S1 joint loading,<sup>5</sup> probably due to increased fatigability of the lower limb muscles,<sup>6</sup> showing that athletes likely involve the lower lumbar spine to compensate for the overall decrease in stroke force. These changes are also observed during submaximal rowing, as lumbar spinal motion increased during the course of a 60-min steady-state ergometer trial.<sup>7</sup> It is expected that these changes in lumbar spine kinematics and kinetics are accompanied by different activation patterns of the lumbar muscles. However, there is a paucity of studies quantifying lumbar muscle activation patterns in rowers with a recent history of LBP, and the studies that have been conducted, have applied classic bipolar surface electromyography (EMG),<sup>8,9</sup> which is known to have large variability and low reliability.<sup>10</sup> High-density EMG (HDEMG), using grids of tens of electrodes, increases the reliability and sensitivity of amplitude estimates.<sup>11–13</sup> In addition, HDEMG measures the spatial distribution of muscle activity and can identify relative adaptations in the intensity of activity within regions of a muscle.<sup>14,15</sup> Changes in homogeneity/heterogeneity of HDEMG have been previously used to evaluate changes in muscle behaviour in LBP<sup>14</sup>; nevertheless, this technique has never been used in rowers or during a rowing task. Here we apply this methodology to assess the topographical distribution of lumbar erector spinae (ES) activity of rowers with and without a recent history of LBP during an incremental rowing test. It was hypothesized that the rowers with a recent history of LBP would show altered activity within regions of the lumbar ES revealing a sub-optimal pattern of ES muscle use.

## 2. Methods

**Study design and participants.** A cross-sectional study was conducted on 18 rowers (junior, senior, and veteran) recruited via print and social media advertisements. All participants were rowing at a competitive level (club or international level) for at least one year and training a minimum of 7 sessions per week. Participants with a recent history of LBP ( $n=8$ , 5 male/3 female, age 32.1 (17.6) years, BMI = 24 (3), % body fat = 19.0 (6.8) %, years rowing = 15 (13.3)) experienced rowing-related symptoms within the last 6 months [Oswestry Disability Index of 21 (2) %] but not during the 6 weeks prior to the study. Rowers free of LBP ( $n=10$ , 8 male/2 female, age 27.0 (14.3) years, BMI = 23 (1), % body fat = 16.3 (4.9) %, years rowing = 12.1 (13.5)) for >12 months served as controls. The study was conducted in accordance with the declaration of Helsinki and the Ethics Committee of Trinity College Dublin Faculty of Health Sciences approved the study. Participants provided informed written consent and a pre-participation screening tool was applied to confirm that the participants were fit to test.

**Testing protocol.** Following the application of testing equipment (HDEMG electrodes, electro-goniometer and heart rate monitor; see below for details), the rowers performed a 5-min warm up on a Concept2 (Model D, USA) rowing ergometer at a power output equivalent to the first stage of the subsequent test. Participants then performed the 7 × 4-min incremental rowing test, devised by the Australian Institute of Sport (see Appendix A). Starting workload and incremental load were determined by participant's previous best 2000 m time. Thus, the rowers started at a load ranging between 140 and 200 W and an incremental increase of 15–45 W. Stage duration was 4-min with 1-min recovery, during which blood lactate was measured from the earlobe via Lactate Pro

2 monitor (COSMED, Italy). Lactate threshold was quantified using the V-Slope method.<sup>16</sup>

**Electromyography (EMG) and kinematics data acquisition.** During the test, both HDEMG signals and knee motion were recorded continuously. Surface HDEMG signals were detected with semi-disposable adhesive grids of electrodes (OT Bioelettronica, Italy). Each grid consisted of 13 rows and 5 columns of electrodes (1-mm diameter, 8-mm inter-electrode distance in both directions). Following skin preparation, the electrode grids were placed bilaterally over the lumbar ES, 2 cm lateral to the lumbar spinous processes, starting at the level of L5, extending approximately to the level of L3.<sup>17,18</sup> The electrode grid covers the iliocostalis lumborum pars lumborum and the iliocostalis lumborum pars thoracis, with the muscular portions of the longissimus being too medial or too cranial to be covered by the electrode grid.<sup>18</sup>

Conductive paste was inserted into each cavity of the grid to provide electrode-skin contact. A reference electrode was placed over the 7th cervical vertebra. EMG signals were amplified (400-channel HDEMG amplifier, OT Bioelettronica, Torino, Italy; –3 dB bandwidth 10–500 Hz) by a factor of 150, sampled at 2048 Hz, and converted to digital form by a 16-bit AD converter. HDEMG signals were recorded in monopolar mode (64 channels), however, these signals were then re-referenced offline to form 59 bipolar derivations, as the difference between adjacent electrodes in the direction of the muscle fibres.

Pairs of bipolar electrodes (15 cm inter-electrode distance, Spes Medica, Italy) were mounted over the rectus abdominis (RA) and external oblique (EO) muscles bilaterally according to guidelines.<sup>19</sup> This was done with the aim of assessing co-activation (formula: right EO + right RA + left EO + left RA RMS: right ES + left ES RMS × 100) during the drive phase. Sagittal motion of the rowing task was measured with a twin axis SG150B electrogoniometer placed over the knee joint (Biometrics Ltd., UK) with one axis (sagittal plane) used for analysis. The goniometer was attached to the knee joint since movement in the lumbar region is typically small<sup>4</sup> and does not allow to accurately distinguish the different phases of rowing (drive and recovery). The goniometer signal was acquired by the EMG amplifier (OT Bioelettronica, Italy) and sampled at 2048 Hz to ensure synchronization of the data.

**EMG and kinematic data analysis.** EMG signals were firstly band-pass filtered (20–350 Hz, second order, zero lag Butterworth). Root mean square (RMS) values were computed from each bipolar recording (59 bipolar EMG signals) obtained from the grid (12 longitudinal bipolar recordings in each column except the far left and right, which had 11 electrode pairs).<sup>14</sup> For graphical representation, the 59 values were interpolated by a factor of 8, but only the original values were used for data processing and statistical analysis. To characterize the spatial distribution of muscle activity, the following variables were extracted from the 59 bipolar signals: RMS (averaged over the 59 signals), modified entropy (measure of uniformity that can be used to indicate the degree of homogeneity in muscle activation).<sup>15,20</sup> A reduction in entropy values means that signals are becoming more heterogeneous) and the y-axis coordinate of the barycentre of the RMS map (cranial-caudal direction).<sup>14</sup> Values of RMS, modified entropy and y-axis coordinate of the barycentre were calculated during two different epochs during the drive phase, therefore, EMG signals were analyzed from full flexion to 33% of extension (epoch 1) and 33% of extension to 66% of extension (epoch 2). These epochs were selected because they represent the phases of the drive where the ES is mostly active (epoch 1 represents the beginning of ES activation while epoch 2 represents the period of peak force production).<sup>21</sup> Co-activation between the flexors and extensors was quantified on the first two epochs and in a third epoch [66% of extension to full extension (epoch 3)] as presented previously.<sup>21</sup> The windows where these phases were calculated was adjusted to kinematic changes (i.e. if the participants

increased stroke rate, the windows still represent full flexion to 33% of extension and 33% of extension to 66% of extension) and were not fixed in time as done in previous studies.<sup>21,22</sup> This was necessary as we instructed participants to perform the incremental test as naturally as possible (with their preferred stroke rate) in order to mimic a competitive scenario.

Extracted EMG values were averaged for the full duration of each incremental step (7 values of RMS, y-axis barycentre and entropy); however, only the first five steps were considered for further analysis, since in some cases large movement artefacts and/or sweating during the last two steps affected signal quality. To allow comparisons between rowers with and without a recent history of LBP, the RMS values were expressed as a percentage relative to the initial value, which was the average value of RMS in the first minute of the first exercise step of each epoch. This method of normalization was employed due to two reasons; first, individuals with a history of low back pain may not be able to activate their muscles maximally and therefore normalization against a submaximal contraction is preferable.<sup>23</sup> Second, this method has shown to be the most sensitive to measure changes in muscle activation during incremental exercise (cycling).<sup>24</sup>

All EMG variables extracted from the right and left electrode grids were compared within each group before the EMG variables were compared between groups. This was necessary to determine the most appropriate side of comparison between groups (e.g. LBP right vs. control right or LBP painful side, e.g. left vs. control left).

**Statistical analysis.** The normality of the data was tested using the Shapiro–Wilk’s test. The assumption of sphericity was checked by the Mauchly test, and in case of violation, the Greenhouse–Geisser correction was applied. Homogeneity of variances was checked with a Levene test. Data was analyzed descriptively via means and standard deviations (SD). Anthropometrics, lactate and power output between groups were compared by independent *t*-tests. Potential side differences in EMG variables were examined within the group with a recent history of LBP using 3-way repeated measures analysis of variance (ANOVA) with factors side (left, right), load (15%, 30%, 45%, 60%, 75% peak power output) and epoch (one and two). Statistical comparison for EMG variables between groups (RMS, entropy, y-axis barycentre) was performed using 4-way ANOVA with factors side, load, epoch and group. The degree of co-activation between the lumbar ES and abdominal muscles was evaluated using 3-way ANOVA with factors of group, epoch and load. Student–Newman–Keuls test was used for pairwise comparisons when ANOVA was significant. Finally, the partial eta-squared ( $\eta^2$ ) for ANOVA was used to examine the effect size of the differences in EMG parameters between groups. A  $\eta^2$  less than 0.06 was classified as “small”, 0.07–0.14 as “moderate”, and greater than 0.14 as “large”. Alpha level was set at 5%.

### 3. Results

There were no differences in anthropometrics or rowing profiles between groups ( $p > 0.27$  in all cases). Moreover, there were no group differences in the intensity where the lactate threshold was identified (control = 3.20 (0.46) w/kg vs. LBP = 2.92 (0.50) w/kg,  $p = 0.49$ ) and peak power output (control = 3.94 (0.76) w/kg vs. LBP = 3.96 (0.83) w/kg,  $p = 0.97$ ).

**Evaluation of side to side differences.** In the group with a recent history of LBP, 4 participants had a history of pain on the right, 3 on the left and one central, however, there were no side differences for any of the EMG variables in either group ( $p > 0.13$  in all cases). Therefore, for all further analyses the EMG variables were assessed between groups according to the same side (e.g. right ES LBP vs. right ES control).

**Y-axis barycentre.** Asymptomatic rowers showed cranial displacements of the barycentre at high loads, while rowers with a recent history of LBP showed a caudal shift of the barycentre throughout the rowing trial. Fig. 1 presents representative topographical maps of the EMG RMS value recorded from the ES for a rower in the control group and a rower with a recent history of LBP throughout the incremental test (5 steps, epoch 2). These representative results were confirmed for the group of participants (Fig. 2) as there was a significant interaction between group and load ( $F = 5.49$ ,  $p = 0.001$ ,  $\eta^2 = 0.26$ ).

**Average EMG amplitude.** The rowers with a recent history of LBP showed higher ES EMG amplitude compared to the rowers without LBP during epoch 2, while the asymptomatic rowers maintained their level of activation relatively constant on both sides (right, left) in both epochs (Fig. 3a) (3-way interaction between epoch, load and group;  $F = 2.81$ ,  $p = 0.032$ ,  $\eta^2 = 0.15$ ).

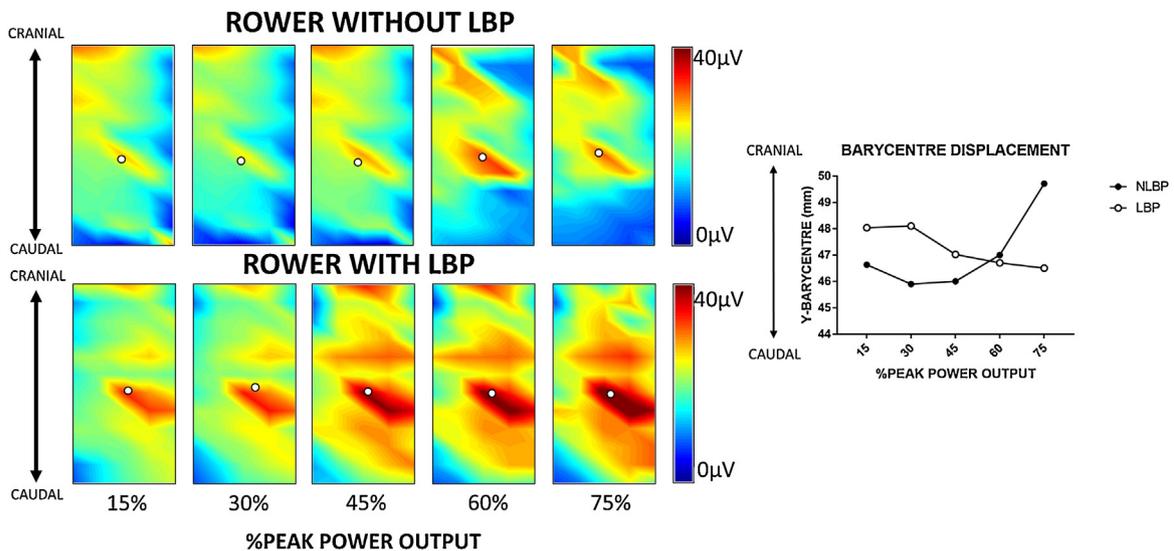
**Modified entropy.** The asymptomatic rowers showed increased heterogeneity of their EMG signals as the load progressed, in comparison to the rowers with a recent history of LBP (group  $\times$  load interaction,  $F = 3.66$ ,  $p = 0.01$ ,  $\eta^2 = 0.19$ ) (Fig. 3b). The right ES was the side showing the largest reduction in entropy for the control group (group, muscle side and load interaction,  $F = 2.81$ ,  $p = 0.033$ ,  $\eta^2 = 0.15$ ).

**Co-activation.** There were no differences in the level of co-activation between groups during the drive phase in all epochs ( $F = 1.68$ ,  $p = 0.109$ ,  $\eta^2 = 0.095$ ) (see supplementary figure).

### 4. Discussion

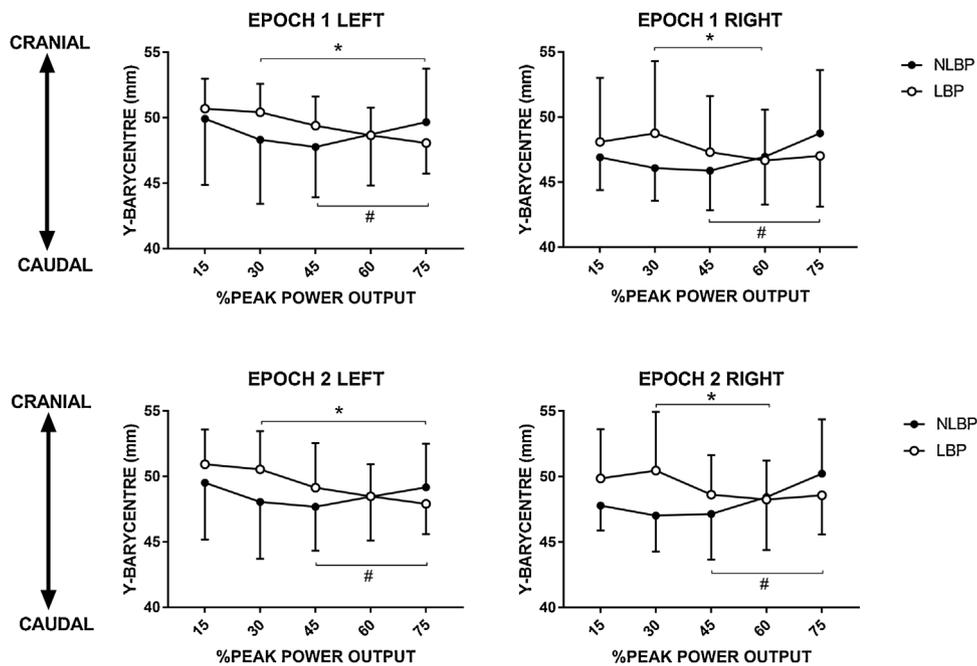
This study is the first to demonstrate that both the level and distribution of activation of the lumbar ES muscle is altered in rowers with a recent history of LBP. This, in addition to the lower complexity of the EMG signals, suggests an inefficient recruitment of the ES muscle in rowers with a recent history of LBP, which likely has significant implications for the perpetuation of LBP.

**Spatial changes in the distribution of lumbar ES activity.** Previous studies using classic bipolar EMG techniques have evaluated lumbar ES activation in healthy rowers,<sup>3,25–27</sup> but no previous study has investigated the distribution of activity within the lumbar ES during rowing. During incremental exercise, healthy rowers show no significant displacement of ES activity up to loads ~45% of the peak power output. Activation patterns change at higher loads as rowers show a cranial shift in ES activity (Figs. 2 and 3). These findings can be interpreted according to the results obtained from studies evaluating lumbar kinetics and kinematics. Previous studies investigating asymptomatic rowers show increased levels of lumbar flexion over the course of a 2000 m race simulation,<sup>22</sup> incremental rowing test<sup>4</sup> and prolonged steady-state rowing.<sup>3,7,21,22</sup> It is very likely that increased fatigability of lower limb muscles influenced spinal motion. Accordingly, Buckeridge et al.,<sup>5</sup> found that shear and compressive forces on the L5/S1 segment increases at higher loads, while peak knee extensor moments decreased. Taken together, these observations suggest that it is not entirely possible to avoid the increased movement of the lumbar spine at high rowing intensities; therefore, it would be necessary to transfer movement to higher lumbar areas, which would place the ES in greater mechanical advantage, thus potentially protecting the spine from compressive and shear forces. These observations are important considering our findings in symptomatic rowers, since they showed a consistent caudal displacement of ES activity as the load progressed. This likely placed the ES in a mechanical disadvantage, possibly increasing load on lower lumbar areas. Accordingly, in one of the few studies that evaluated rowers with LBP, Ng et al.,<sup>28</sup> showed that rowers with LBP have less excursion of the upper lumbar spine during the drive phase, maintaining a flexed posture of



**Fig. 1.** Representative topographical maps (interpolation by a factor 8) of the EMG root mean square (RMS) value recorded from the right lumbar erector spinae (ES) for a rower without a recent history of low back pain (NLBP, up) and with a recent history of LBP (LBP, down) during the rowing incremental test (15%, 30%, 45%, 60% and 75% peak power output). HDEMG maps were extracted from the second epoch (peak force phase). The white circle in the middle of each map represents the barycentre (centre of activity). The full displacement of the barycentre across the task for both rowers can be seen on the right side of the figure. For the NLBP rower, the barycentre started to shift cranially from 45% peak power output while for the LBP rower the barycentre displaced caudally from 30% peak power output.

### Y-AXIS BARYCENTRE

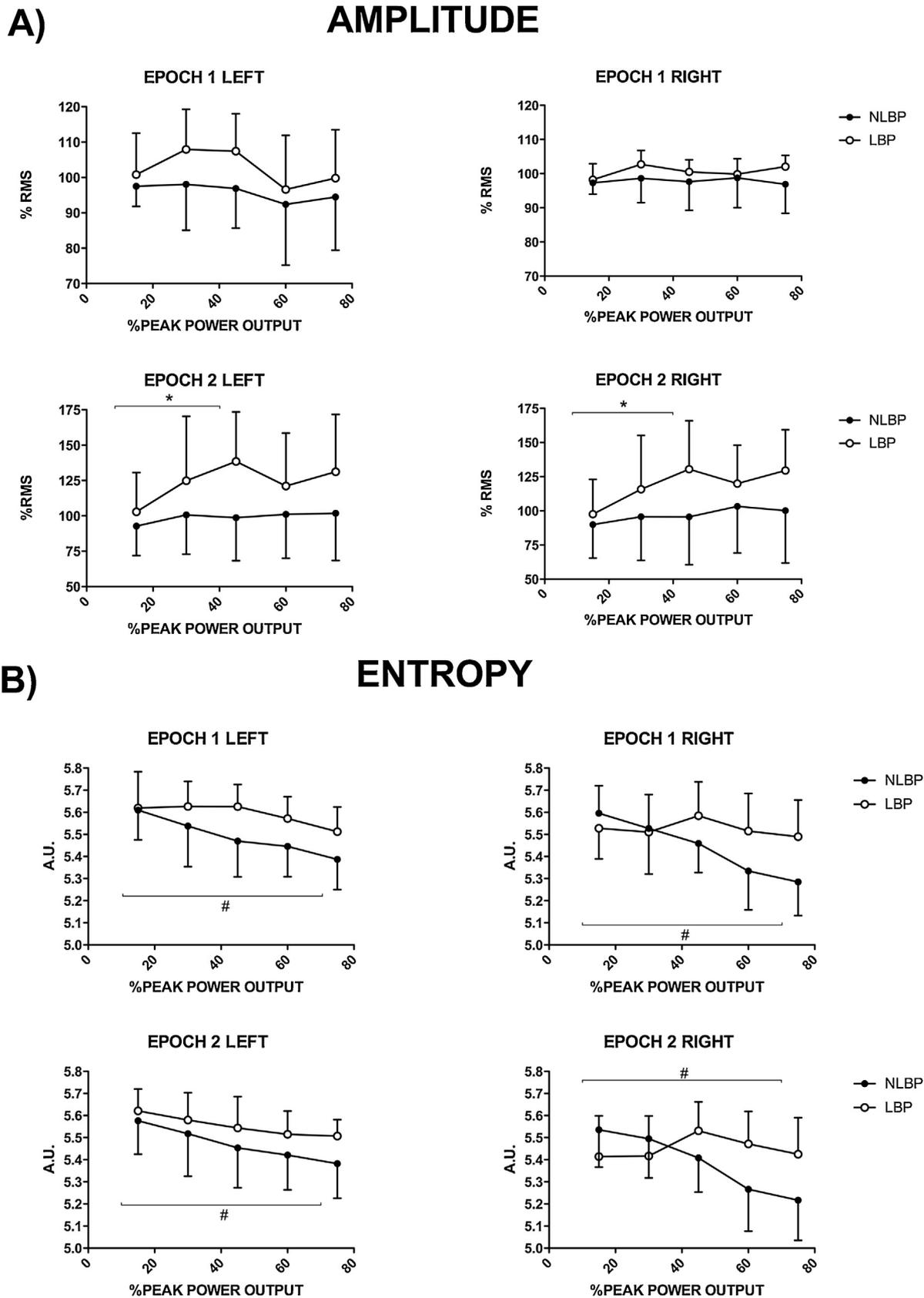


**Fig. 2.** Displacement of the barycentre of the left and right erector spinae (ES) at five steps (15%, 30%, 45%, 60%, 75% peak power output) of the incremental test in the two different epochs. Rowers without a recent history of low back pain (NLBP, black circles); rowers with a recent history of low back pain (LBP, white circles). \*, significant caudal displacements of the barycentre in the LBP group ( $p < 0.001$ ). #, significant cranial displacements of the barycentre in the NLBP group ( $p < 0.001$ ).

the lower lumbar spine while rowing at high intensities. Although ES activity was not assessed in that study, it is very likely that these rowers would also show a caudal displacement of lumbar ES activity as the rowing trial progressed, given the differences in lumbar movement observed.

*Changes in the amplitude of ES activity.* Previous research using bipolar EMG showed that asymptomatic rowers do not increase their level of ES activation as workload increases.<sup>21,22,25,29</sup> Our findings are in agreement, as the asymptomatic rowers maintained their

activation levels throughout the trial. In contrast, the rowers with a recent history of LBP showed higher ES activity compared to the control participants during the epoch where the ES was most active (2nd epoch, Fig. 3a). Recent research using HDEMG in people with chronic non-specific LBP has also shown increased levels of ES activation relative to asymptomatic people.<sup>14</sup> The authors suggested that the increased amplitude could be due to an increased excitatory drive to painful muscles. However, in the present study, none of the rowers experienced pain during the trial. Therefore,



**Fig. 3.** Root mean square (RMS, A) and entropy (B) values of the left and right erector spinae (ES) at five steps (15%, 30%, 45%, 60%, 75% peak power output) of the incremental test in the first and second epochs. Rowers without a recent history of low back pain (NLBP, black circles); rowers with a recent history of low back pain (LBP, white circles). \*, significant increase in RMS amplitude in the LBP group ( $p < 0.001$ ). #, significant decrease in entropy in the NLBP group ( $p < 0.001$ ). A.U., arbitrary units.

these changes are most likely attributed to an altered rowing pattern requiring higher activation of the more distal regions of the ES, compensating for increased flexion.<sup>28</sup> This observation can be confirmed by the co-activation data since there was no difference in the level of co-activation between groups, suggesting that the increased activation of the ES in the LBP group was not due to increased activation of the antagonists but rather due to changes in lumbar kinematics (increased lumbar flexion).

*Changes in the uniformity of muscle activity.* An increase in the heterogeneity of HDEMG signals (lower entropy) is thought to be related to altered spatial reorganization of muscle activity potentially with the aim of reducing muscle fibre overload during fatiguing contractions.<sup>15,30</sup> Therefore, while the barycentre provides an estimate of where the average activity of the muscle activity is positioned in space (centre of mass of the HDEMG map), entropy provides an estimate of homogeneity between signals of the whole 2D HDEMG map.

As predicted, rowers with a recent history of LBP showed higher values of entropy compared to the asymptomatic rowers as the load progressed (Fig. 3b). As appreciated in Fig. 1, the rower with a recent history of LBP increases ES activity (RMS amplitude) during the trial but the regions that were activated are maintained (with a higher increase in amplitude in caudal regions of the map as shown in the barycentre results). On the contrary, the rower without a recent history of LBP shows a clear difference in homogeneity, as regions that were active during the beginning of the trial (caudal regions at 15%, 30% and 45% of peak power output) become less active at 60% and 75% of peak power output.

These results reinforce our findings that rowers with a recent history of LBP fail to recruit their lumbar ES muscle efficiently. They shift activity towards lower lumbar regions, but also maintain motor output by a more homogenous activation of the ES, thereby activating regions which are at greater mechanical disadvantage.

*Implications.* Optimizing the rowing technique is essential to avoid the development or maintenance of LBP. One of the most important factors to correct is lumbo-pelvic positioning during the stroke phase.<sup>1,4,7,31</sup> Neutral positioning of the pelvis allows efficient movement of the hips, decreasing both flexion of the lumbar spine and the activation of the lumbar muscles. The development of new HDEMG methods allowing visualization of ES spatial changes in real time could prove a useful biofeedback tool in helping rowers to reduce the activity of their lower lumbar ES and facilitate more efficient lumbo-pelvic motion.

*Limitations.* There are some methodological considerations which warrant attention. Firstly, the study was conducted on a relatively small and heterogeneous sample of elite rowers. It was not possible to assess differences between rowers with different levels of experience. Future investigations should aim to evaluate differences between rowers with different levels of expertise since it could be expected that more refined use of the ES would be present in internationally competitive rowers versus those with less experience. Secondly, a Concept II stationary ergometer was used for all trials. Differences in stroke mechanics have previously been reported comparing stationary and dynamic ergometers.<sup>4,32–34</sup> However, no differences in ES activity were observed comparing rowing ergometer designs to each other<sup>27</sup> or to on-water sculling.<sup>25</sup> Nonetheless, it is possible that results may differ when performed on a dynamic ergometer (i.e. Rowperfect) or on-water. Thirdly, due to the complexity of recording HDEMG from the participants under such demanding conditions, it was not possible to quantify lumbar kinematics. Finally, it is important to mention that the participants in this study did not experience pain during the rowing trial. Longitudinal studies, specifically evaluating muscle behaviour whilst rowing during periods with and without pain should be conducted in order to better understand the relationship between LBP symptoms and an altered spatial distribution of muscle activity

in rowers. Furthermore, prospective studies examining risk of LBP should assess EMG longitudinally to examine if specific recruitment patterns can predict LBP onset or response to rehabilitation.

## 5. Conclusion

Both the magnitude of activation and the distribution of ES activity were altered in rowers with a recent history of LBP compared to back pain-free rowers. The lower complexity of signals together with the caudal displacement of the centre of activity at high work intensities suggests an inefficient recruitment of the ES as the load progressed. Modification of the rowing technique in conjunction with feedback from HDEMG might prove useful in future studies.

## Funding and financial support

This research did not receive any specific grant from funding agencies in the public, commercial or non-for-profit sectors.

## Ethical approval

Trinity College Dublin, Faculty of Health Sciences ethics committee approved the study (reference number of the project: 161107).

## Disclosure statement

The authors report no potential conflicts of interest

## Acknowledgements

This research did not receive any specific grant from funding agencies in the public, commercial or non-for-profit sectors.

## Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at <https://doi.org/10.1016/j.jsams.2019.07.007>.

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