# Spinal kinematics of adolescent male rowers with back pain in comparison to matched controls during ergometer rowing

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## ABSTRACT

There is a high prevalence of low back pain (LBP) in adolescent male rowers. In this study, regional lumbar spinal kinematics and self-reported LBP intensity were compared between 10 adolescent rowers with moderate levels of LBP relating to rowing with 10 reporting no history of LBP during a 15-minute ergometer trial using an electromagnetic tracking system. Adolescent male rowers with LBP reported increasing pain intensity during ergometer rowing. No significant differences were detected in mean upper or lower lumbar angles between rowers with and without LBP. However, compared to rowers without pain, rowers with pain had: 1) relatively less excursion of the upper lumbar spine into extension over the drive phase, 2) relatively less excursion of the lower lumbar spine into extension over time, 3) greater variability in upper and lower lumbar angles over the 15-minute ergometer trial, 4) positioned their upper lumbar spine closer to end range flexion for a greater proportion of the drive phase, and 5) showed increased time in sustained flexion loading in the upper lumbar spine. Differences in regional lumbar kinematics exist between adolescent male rowers with and without LBP, which may have injury implication and intervention strategies.

***Keywords***: *athletes, spinal pain, sports, biomechanics*

**Word Count:** 4330

## INTRODUCTION

The World Rowing Federation has identified that Low Back Pain (LBP) is a common condition experienced by rowers of all ages.[1] Amateur adolescent rowers aged between 14 to 16 years have been shown to have a high lifetime prevalence of LBP, with reported rates of 94% in males and 65% in female rowers.[2]

Mechanical loading factors such as long on-water rowing time in training sessions, repetitive lifting of the rowing shell, and ergometer rowing have been associated with LBP in rowers.[3-5] More specifically, there is a growing body of evidence suggesting that specific patterns of spinal kinematics during ergometer rowing may be particularly provocative of LBP in rowers.[3,6,7] In support of this, studies have identified that some rowers present with large magnitude of lumbar spine flexion during ergometer rowing reflecting a potential mechanism for LBP.[3,6,7] This relationship has not yet been specifically investigated in an amateur adolescent population. Understanding LBP mechanisms adolescent sporting populations such as rowing is important, as this is the age where most rowers take up the sport and they appear to be particularly susceptible to LBP. Further, LBP in adolescence is a known predictor of LBP in adulthood.[8]

It has been suggested that the repetitive nature of lumbar flexion during rowing may increase lumbar excursion during rowing,[7,9-11] and that this has been linked to back pain.[12,13] Further, end-range flexion may also be associated with back pain, [14-16] as it has been proposed that position of the lumbar spine relative to the end of range, where passive structures of the spine are close to being maximally loaded or stretched, may increase the risk of tissue strain and pain. [17,18] Previous research has identified end-range spinal flexion in sitting to be related to LBP in both sporting [15,19] and non-sporting populations supporting a pain / postural relationship.[16,20]

Several studies have reported spinal kinematics during rowing using healthy pain free populations and speculated a link with spinal movement and LBP.[7,9,10] These reports have shown that rowers frequently posture their spine at the end-range of spinal flexion with the magnitude of lumbar flexion increasing over time of the rowing task, which may increase the potential for back pain.[7,9-11] However, these investigations did not consider two separate lumbar regions (upper and lower), which is now recognized as a more appropriate method of quantifying lumbar regional kinematics, as individuals are shown to control their upper and lower lumbar spine differently during functional tasks [14,20,21]. At present, there is a paucity of literature that has examined regional spinal movement during rowing and to our knowledge no studies have investigated rowers with LBP. This is despite a demonstrated relationship between LBP and differences in regional lumbar kinematics in non-rowing populations.[15,19]

Therefore, the aims of this study were to; investigate whether there is an increase in LBP intensity in rowers with LBP, and to investigate differences in lumbar kinematics between rowers with and without LBP, during a 15-minute rowing ergometer trial. Specifically, we hypothesized that

1. Pain intensity levels for rowers with LBP would increase over the course of a 15-minute rowing ergometer trial.
2. Rowers with LBP would posture their upper and lower lumbar spine in a greater degree of flexion than rowers without LBP during the drive phase of ergometer rowing. Further, the LBP group would demonstrate greater increases in flexion over the 15 minutes period compared to the non-LBP group.
3. Rowers with LBP would spend a greater proportion of the drive phase of the rowing stroke with their upper and lower lumbar spine near end range flexion than rowers without LBP. Further, this difference would become greater over 15 minutes of rowing.

## METHODS

Twenty adolescent male rowers, aged between 14 to 19 years, with (n=10) and without (n=10) LBP participated in this study (Table 1). A power calculation prior to participant recruitment suggested that 10 participants in each group would provide 80% power to detect a group difference of 10 degrees (assuming a standard deviation of 10 in both groups, repeated measures for 3 phases over 1,7 and 15$^{th}$ minute, and a within-subject correlation of 0.6). Participants were included if they performed rowing training for a school-rowing club or a community rowing club at least three times per week as well as competing in rowing regattas. Participants were defined as having LBP if their self-reported LBP was located between the levels of the 1$^{st}$ and 5$^{th}$ lumbar vertebrae (i.e. $L_1$ – $L_5$) and if this pain was provoked by rowing with an intensity greater than 3cm (out of 10cm) as indicated by a visual analogue scale (VAS) within 30 minutes of rowing training. The characteristics of the participants including; age, height, mass, body mass index (BMI), self reported level of pain during participant recruitment (VAS) and their self reported disability score was collected from the Roland Morris Disability Questionnaire[22] and Patient Specific Functional Scale[23] are presented in Table 1. Participants in the no pain group had no history of LBP. Rowers were excluded from this study if there was a presence of specific causes of LBP such as inflammatory diseases, radicular pain or neurological signs to the lower limbs, or they had reported any lower limb musculoskeletal injury in the six weeks preceding data collection. Further participants were excluded if they received any rowing specific postural training during previous rehabilitation of their LBP, as this may influence their spinal kinematics during rowing, which this study was investigating. Permission to conduct the study was granted by the Institutional Human Research Ethics Committee and all subjects and their parents/guardians (where necessary) provided written informed consent/assent.

Three dimensional regional lumbar angles were collected using the 3-Space Fastrak™ electromagnetic tracking system at 25 Hz (Polhemus Navigation Science Division, Kaiser Aerospace, Vermont). The Fastrak™ system has been used in previous rowing studies,[24-26] and has been reported to be valid and reliable in measuring joint angles in the sagittal plane, reporting average errors of 0.4$^o$ using a wooden model positioned on a modified rowing ergometer.[27] Three of the device's sensors were secured on the participant's skin overlying the spinous processes of S2, L3 and T12 using double sided tape and Fixomull® such that the lower lumbar angle (LLA) and the upper lumbar angle (ULA) could be derived (Figure 1).[20,24,26] A rotary encoder was connected to the flywheel of the rowing ergometer to determine the stroke length and stroke rate. Prior to every data collection trial, stroke length was calibrated with the voltage on the rotary encoder and then synchronised with the Fastrak™ using a customized Labview software program (Version 8.6.1, National Instruments, Texas, USA). This stroke length was used to determine the start and the end of the drive phases, stroke length is shortest at the beginning of the drive phase (*catch*), and longest at the end of the drive phase (*finish*). Ergometer rowing was chosen for this study as it has been suggested as an aggravating factor to LBP in rowers[3,4,12,28], and this has the advantage of controlling extrinsic factors such as wind and water condition during data collection. [3]

INSERT FIGURE 1 ABOUT HERE

Participants' maximum slouch angles were determined in static sitting with participants instructed to place their feet flat on the ground; shoulders' width apart with their knees bent to 90$^o$; and their arms crossed in front of the chest. They were then instructed to 'slouch as far down as possible'. They were required to hold these positions for five seconds, and this process was repeated three times with a 30 second rest period between each trial. The maximum Lower Lumbar Angle (LLA) and Upper Lumbar Angle (ULA) were then calculated and used to represent maximum slouch. This protocol was used in a previous study by the authors.[24]

Prior to ergometer testing participants completed a 5-10 minute warm up involving sub-maximal ergometer rowing. Participants rowed for 15 minutes at a stroke rate of 22 strokes per minute with a rating of perceived exertion of 17/20. This protocol was designed after consultation between the research team and coaches as this was deemed to be common training practices in the adolescent rowing population. Kinematic data was collected during the last 15 seconds of the 1st minute (start), 7th minute (middle) and 15th minute (end). The 15-second period equated to three to five full-completed strokes. During the ergometer trial, the Numeric Pain Rating Scale (NPRS), which is an 11-point scale (0-10) to collect self-reported pain intensity,[29] were collected verbally at the beginning of every minute of the ergometer trial and also at the end of the 15-minute ergometer trial. Participants were advised to cease the ergometer trial if their level of pain during testing exceeded their level of pain during their usual rowing training or competition.

A customized LabVIEW program (Version 8.6.1, National Instruments, Texas, USA) converted outputs derived from the 3-Space Fastrak™ during the first three completed strokes to flexion and extension angles (angles in the sagittal plane) via matrix algebra procedures as described elsewhere.[30] From these procedures, LLA and ULA were derived[24-26] as shown in Figure 1. For the derived angles, $0^{o}$ of the LLA is reflected by L3 marker being parallel to the S2 sensor and positive values indicated flexion (anterior rotation of the L3 sensor over the S2 sensor) while negative values indicated extension (posterior rotation of the L3 sensor over the S2 sensor). Similarly, $0^{o}$ of the ULA is reflected by the T12 marker being parallel to L3 sensor, where positive values indicated flexion (anterior rotation of the T12 sensor over the L3 sensor) and negative values indicated extension (posterior rotation of the T12 sensor over the L3 sensor. Consistent with previous research, only sagittal plane angles and data from the drive phase were analysed,[7,9,11] given that the drive phase is known to be when the spinal load is greatest.[13] All data in the drive phase were time normalized, with 0% defined as the *catch* and 100% defined as *finish*. Near end-range flexion was defined as above 80% of the maximum slouch angle during the static sitting test.[19]

Independent t-tests were used to determine whether age, height, body mass and BMI differed between no pain and pain groups. A linear two level mixed-effects model was used to evaluate the change in NPRS scores reported at baseline and each minute over the 15 minutes of rowing to assess the relationship between rowing and LBP intensity over time.

Flexion angle measures taken at percentiles of the drive phase from three completed stokes were averaged to produce a single flexion angle (for both ULA and LLA) for the early (0,10 and 20$^{th}$ percentile), mid (30-70$^{th}$ percentile) and late (80,90 and 100$^{th}$ percentile) drive phase, at the end of the 1$^{st}$, 7$^{th}$ and 15$^{th}$ minute of rowing. A linear three level mixed-effects model was used to determine differences between pain and no pain groups, using the 9 repeated measures over drive phase (early/mid/late) nested in minutes (1,7 and 15). Differences in flexion angle across phase and minute were examined and estimates of group difference adjusted for these factors. To examine if the difference in flexion angles between pain and no pain groups became larger over the 15 minutes of rowing, a groupXminute interaction term was evaluated. To examine if the difference in flexion angles between pain and no pain groups were different over the early, mid and late drive phase, a groupXphase interaction term was evaluated.

To evaluate the proportion of drive phase near end range flexion, angular measures (for both ULA and LLA) were sampled at 25Hz for three completed strokes collected during the last 15 seconds of the 1$^{st}$, 7$^{th}$ and 15$^{th}$ minute of the ergometer rowing. These values were expressed as a percentage of maximum slouch sitting angle, and the proportion of drive phase measures for which this value exceed 80% was calculated then averaged over the three strokes at the 1st, 7th and 15th minute. A linear two-level mixed-effects model was used to determine differences between pain and no pain groups, using the 3 repeated measures over the 1st, 7th and 15th minute. Differences in proportion of drive phase near end range flexion across minute were examined and the estimate of group difference adjusted for minute. To examine if the difference in proportion of drive phase near end range flexion between pain and no pain groups became larger over the 15 minutes of rowing, a groupXminute interaction term was evaluated. The non-parametric ranks-based Mann-Whitney test was also performed on these measures to test for group difference at the 1st, 7th and 15th minute separately to confirm findings were robust to misspecification of the linear mixed models.

Models were estimated with and without adjustment for height, weight and age to check for confounding as there was evidence these factors differed between pain and no pain groups. An absence of confounding was assumed if potential confounders were non-significant in models at $\alpha>0.1$; in this case coefficients were estimated without adjustment for these factors. Additionally, although not an a priori objective, a post-hoc comparison of error variances between pain groups in the mixed-effects models was conducted as plotting of the raw data displayed suggested more within-subject variability in data from those subjects with pain (see Results section).

## RESULTS

The demographics of the participants showed that rowers with pain were significantly taller and heavier than rowers with no pain but no differences were found in the age and BMI between the two groups (Table 1). There were no statistically significant differences in the maximum slouch angles during the static sitting trial between groups, rowers in the pain group postured their LLA at 3.2$^{o}$ (17.5 $^{o}$) compared to 3.7$^{o}$ (7.8 $^{o}$) in the no pain group (95%CI: -13.2$^{o}$ to 12.3$^{o}$, p=0.942) and their ULA at 4.6$^{o}$ (8.1 $^{o}$) compared to 2.6$^{o}$ (11.1$^{o}$) in the no pain group (95%CI: -7.2$^{o}$ to 11.1$^{o}$, p=0.656).

Numeric Pain Rating Scale scores increased significantly over the 15 minutes of rowing from 1.7 (95%CI: 1.0 to 2.3) at baseline to 7.8 at the 15$^{th}$ minute (95%CI: 7.10 to 8.42), with the rate of increase estimated to be 0.41 per minute (95%CI: 0.38 to 0.44, p<.001) in rowers with LBP (Figure 2). All rowers in the no pain group reported 0 NPRS at each minute of the ergometer trial.

INSERT TABLE 1 AND FIGURE 2 ABOUT HERE

No significant differences were observed in the mean LLA between groups (Table 2). Adjustment for height, weight and age revealed no confounding of group differences and results are presented unadjusted for these factors to maximise precision of estimates. The LLA for each subject for the early, mid and late drive phase over the 1$^{st}$, 7$^{th}$ and 15$^{th}$ minute separately for each pain group are presented (Figure 3). Significant main effect for phase (p<.001) and no evidence of interaction between pain group and phase (p=.821), with flexion decreasing from early, mid to late phase similarly in both groups (Table 3). A significant main effect for the pain group was not detected (p=.688), although an interaction between minute and pain group was detected (p=.012), with the pain group displaying more extension (adjusted for phase) in the 15$^{th}$ minute compared to the 1$^{st}$ minute, whereas the no pain group displayed similar LLA at all three time points (Table 3). Examination of the raw data plotted suggested more within-subject variability in changes over minute in the pain group, with relatively large changes occurring in both directions, compared to a consistent pattern of no change in the no pain group (Figure 3). Therefore, this was formally tested by comparing the variance of the error terms in the mixed effects model. These were significantly different, with the standard deviation for the pain group being greater [10.6$^{o}$ (95%CI: 9.4$^{o}$ to 12.8$^{o}$)] than the no pain group [4.0$^{o}$ (95%CI: 3.4$^{o}$ to 4.7$^{o}$)], indicating significantly greater within-subject variability in the pain group data.

INSERT FIGURE 3 ABOUT HERE

INSERT TABLE 2 AND 3 ABOUT HERE

No significant differences were observed in the mean ULA between groups. Analysis using linear mixed effects model identified no effect for minute ($p=.526$) and no group by minute interaction ($p=.774$). The means and standard deviations for ULA by phase, minute and pain/no pain group are presented (Table 2). Adjustment for height, weight and age revealed no confounding of group differences and results are presented unadjusted for these factors to maximise precision of estimates. Raw data for ULA for each subject over the early, mid and late drive phase by 1$^{st}$, 7$^{th}$ and 15$^{th}$ minute, separately for each pain group are presented (Figure 4). Although there was evidence that groups differed by phase ($p<.001$), the estimated group difference was not statistically significant at any phase (table 3). There was a significant interaction between phase and group, meaning the degree of change over phase was estimated to differ by group, with a pattern of significantly more extension over early, mid and late phase evident in both groups (Table 3), but to a significantly lesser extent in the pain group. Raw data plotted in Figure 4 suggests more within-subject variability in changes over phase in the pain group, with less consistent pattern of increasing extension over the drive phase compared to the consistent pattern seen in the no pain group. This was formally tested by comparing the variance of the error terms in the mixed effects model. These were significantly different, with the standard deviation for the pain group being greater ($4.9^{o}$ (95%CI: $4.0^{o}$ to $6.0^{o}$) than the no pain group ($2.8^{o}$ (95%CI:$2.4^{o}$ to $3.3^{o}$)), indicating significantly greater within-subject variability in the pain group data.

INSERT FIGURE 4 ABOUT HERE

No statistically significant differences were observed in the LLA in the proportion of drive phase in near or end of range flexion between groups. The raw means and standard deviations for the proportion of drive phase near end range LLA flexion by minute and pain/no pain groups (Table 4). This data are presented graphically for each subject over $1^{st}$, $7^{th}$ and $15^{th}$ minute, separately for each group (Figure 5A). Analysis using a linear mixed effects model detected evidence of an association between a lesser proportion of drive phase spent in flexion with increasing age and (weight-adjusted) height (Table 5). No effect for minute (p=.872) and no group by minute interaction was observed (p=.284). The pain group was estimated to spend less time of drive phase in near end range when compared to no pain group, adjusted for minute, age, height and weight (-.27, 95%CI: -.59 to .04, p=.087, Table 3) but this difference was not statistically significant. The raw data plotted displays suggest greater degree of variability in the proportion of drive phase near end range LLA flexion in the pain group (Figure 5A), with less consistent patterns over time in the pain group. Again, this was formally tested by comparing the variance of the error terms in the mixed effects model. These were significantly different, with the standard deviation for the pain group being greater (.31 (95%CI: .23 to .42) than the no pain group (.06 (95%CI:.04 to .08), indicating significantly greater within-subject variability in the pain group data. Nonparametric analysis of this data also did not detect a difference in proportion of drive phase in near end range LLA in the pain group at the $1^{st}$, $7^{th}$ or $15^{th}$ minute (Mann Whitney test, p= .341, .272 and .702 respectively).

INSERT FIGURE 5A AND 5B ABOUT HERE

INSERT TABLE 4 ABOUT HERE

Rowers in the LBP group were found to spend a significantly greater proportion of the drive phase near the end of range of ULA flexion compared to the no-LBP group. The raw means and standard deviations for the proportion of drive phase near end range ULA flexion by minute and pain/no pain groups (Table 5). This data is presented graphically for each subject over $1^{st}$, $7^{th}$ and $15^{th}$ minute, separately for each pain group (Figure 4B). Analysis using a linear mixed effects model detected no evidence of an association between a lesser proportion of drive phase spent in ULA flexion with increasing age (β=.00, 95%CI: -.06 to .06, p=.974) and (weight-adjusted) height (β=-.01, 95%CI: -.02 to .01, p=.144), unlike results for LLA, and models were estimated unadjusted for these factors. No effect for minute (p=.548) and no group by minute interaction were observed (p=.226). The pain group was estimated to spend a greater proportion of the drive phase in near end range ULA than the no pain group (.19, 95%CI: .03 to .35, p=.021, Table 3). The raw data suggests a greater degree of within-subject variability generally in the proportion of drive phase near end range for ULA flexion versus LLA, with more inconsistent patterns over time in both groups for ULA than those for LLA (Figure 5B). The standard deviation of the residuals for the pain group (.29 (95%CI: .21 to .39) were comparable to the no pain group (.19 (95%CI: .14 to .26). Nonparametric analysis of this data confirmed a significantly greater proportion of drive phase in near end range ULA in the pain group at the 7th minute (Mann Whitney test, p=.002) but not the 1st (p=.160) or 15th minute (p=.650).

INSERT TABLE 5 ABOUT HERE

## DISCUSSION

The results of this study demonstrate that 15 minutes of ergometer rowing results in increasing intensity of LBP over time in male adolescent rowers with rowing reporting related LBP (Figure 2). Although no significant differences were detected in the mean LLA and ULA between rowers with and without LBP, rowers with pain did demonstrate less ULA excursion and ULA into extension compared to rowers without pain over time.

This increase in pain intensity may reflect a temporal summation of pain, where a repetitive stimulus on pain sensitive structures may cause a gradual increase of pain sensation.[31,32] A similar pattern of pain summation has been reported previously in cyclists with LBP during a 2-hour cycling trial.[19] There is debate regarding the underlying mechanism for this phenomena, with some researchers suggesting that it reflects inhibitory and facilitatory mechanisms in the central nervous system,[33] whilst other authors suggest provocative movement behaviours may result in repeated stress on sensitized tissues with a resultant summation of pain.[14,15] In reality a combination of both of these factors may interplay.

On average, rowers in the pain group maintained their ULA in flexion throughout the drive phase [early ($9.1^{o}$) mid ($5.7^{o}$) and late ($1.0^{o}$)] compared to rowers without pain who moved into more extension in the late phase [early ($10.5^{o}$) mid($6.4^{o}$) late($-3.0^{o}$)]. In addition, rowers with LBP postured their ULA within 80% of end range flexion for a greater proportion of the drive phase than rowers without LBP (mean diff .19, p=0.021). The increased proportion of drive phase spent in flexion by the rowers with LBP in this study is consistent with our hypothesis and may be reflective of a flexion loading strain mechanism for low back pain.[34] Previous studies have reported that both adolescent and adults with LBP provoked by lumbar 'flexion' movements and postures have a tendency to posture their spines closer to end range flexion during sitting [16,35,36]. Similarly, cyclists with LBP have been identified to maintain either lower lumbar spine in a more flexed position [15] or cycle closer to end range of flexion in the lower lumbar spine.[19] It may be that inability to maintain the lumbar spine away from end of range leaves the spine more vulnerable to flexion loading strain in sports where the lumbar spine is exposed to cyclical or sustained loading.

It was hypothesized that adolescent male rowers with LBP would posture their LLA and ULA in more flexion than rowers without LBP during the drive phase of ergometer rowing, and this difference would increase over 15 minutes of rowing. Although no differences in the mean LLA and ULA were detected overall or within the early, mid and late phase or 1st, 7th and 15th minute, on examination of the raw data it was noted that rowers with LBP had greater within-subject variability in LLA and ULAs compared to rowers without LBP. This is a preliminary finding that was not an a priori aim of the study and therefore further investigation is warranted. The within-subject variability in spinal kinematics in individuals with LBP is not a new concept, with higher variability in spinal movement during functional tasks reported in adults with chronic LBP compared to no-LBP. [37,38] This may be due to altered peripheral and central sensory processing of the nervous system, resulting in poorer spinal position sense in adolescents and adults with LBP [39,40], with a tendency to either under or over shoot a neutral sitting posture during a lumbar spine reposition test, a mechanism proposed to increase end range strain. Holt and associates (2003) have also reported variations in spinal kinematics in athletes with and without LBP over a 60-minute ergometer trial,[11] but no direct comparisons were made between the participants with and without a history of LBP.

We acknowledge the following potential limitations of this study. 1) The large variation reported in the kinematics of the pain group participants may explain the lack of significant differences detected in the mean LLA and ULA between the LBP and the no-LBP group. 2) A subjective indicator of rowing effort (RPE) was used in the study rather than an objective measurement of subjects' effort throughout the trial such as power output as it was commonly used in this age group to measure work rate in this group of rowers. Although differences in work rate will exist, the authors feel that this would be minimal as stroke rate was standardised between groups and unlikely to invalidate comparisons between groups. 3) In light of the current finding regarding variability, the analysis of a larger number of strokes and statistical procedures could be considered to evaluate spinal kinematics of rowers with LBP. 4) It is also acknowledged that assessing end range slouch position in the LBP subjects could have been influenced by the presence of pain, although there was no report of discomfort or observable movement guarding during this aspect of the testing. Further, no differences were detected in the maximum slouch angles between groups. Cross sectional studies do not give clear insight to causation requiring the need for future longitudinal studies in order to determine whether kinematic differences precede or follow low back pain in male adolescent rowers.

In conclusion, rowers with LBP positioned their upper lumbar spine nearer end range flexion for a greater proportion of the drive phase and demonstrated greater individual variation in spinal movement than rowers without LBP. These findings may have implications for coaching practices and targeted interventions to improve consistency in rowing technique and avoid prolonged end of range spinal loading so as to minimize the potential for end range sensitization of spinal structures.

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## TABLES

**TABLE 1** – Mean and standard deviation of characteristics in each group and the mean, standard error and p-value of differences between the no pain and pain group.

| Characteristic | No Pain (n=10) | Pain (n=10) | Mean | 95% CI | P value |
|---|---|---|---|---|---|
| **Age (years)** | 17.2 (1.4) | 16.0 (1.2) | 1.2 | -0.1, 2.4 | .074 |
| **Height (m)** | 1.85 (0.08) | 1.70 (0.09) | 0.15 | -0.2, -0.1 | <.001 |
| **Weight (kg)** | 78.2 (12.9) | 66.8 (10.8) | 11.5 | -22.9, 0.0 | .050 |
| **BMI (kg/m$^2$)** | 23.1 (3.4) | 22.8 (3.8) | 0.3 | -2.7, 3.4 | 0.818 |
| **VAS (/10)** | 0 (0) | 4.6 (1.1) | | | |
| **PSFS (/30)** | n/a | 17 (6.1) | | | |
| **RMDQ (/22)** | n/a | 3.5 (2.1) | | | |

BMI = Body Mass Index; VAS = Visual Analogue Scale; PSFS = Patient Specific; Scale; RMDQ = Roland Morris Disability Questionnaire.

**TABLE 2** - Mean and standard deviation of the lower and upper lumbar angles for drive phases over 1$^{st}$ 7$^{th}$ and 15$^{th}$ minute, for Pain and No Pain group.

| | No Pain | | | Pain | | |
|---|---|---|---|---|---|---|
| Minute | Early Phase | Mid Phase | Late Phase | Early Phase | Mid Phase | Late Phase |
| Lower lumbar angle ($^{o}$) | | | | | | |
| 1 | 8.8 (6.7) | 3.7 (7.4) | -4.2 (11.1) | 9.3 (16.2) | 7.7 (10.0) | 3.5 (11.5) |
| 7 | 8.7 (7.0) | 2.9 (7.5) | -2.8 (9.8) | 11.5 (9.6) | 7.6 (9.6) | 1.9 (10.8) |
| 15 | 8.8 (7.4) | 2.9 (8.3) | -3.0 (11.1) | 6.9 (21.4) | -1.1 (18.1) | -8.2 (21.9) |
| Upper lumbar angle ($^{o}$) | | | | | | |
| 1 | 8.6 (7.1) | 5.4 (8.0) | -4.8 (7.7) | 8.2 (7.2) | 5.4 (7.6) | 1.2 (9.3) |
| 7 | 11.2 (6.1) | 6.6 (6.7) | -2.4 (8.1) | 9.4 (8.4) | 6.3 (11.2) | 1.2 (14.0) |
| 15 | 11.8 (6.3) | 7.1 (6.6) | -1.8 (8.2) | 9.8 (10.1) | 5.5 (14.7) | 0.6 (17.1) |

**TABLE 3** - Mixed model coefficients for lower and upper lumbar angle.

| | | Marginal means ($^0$) | β coefficient($^0$) (i.e. contrast) | 95% CI | p-value |
|---|---|---|---|---|---|
| **Lower lumbar angle** | | | | | |
| Group (Pain – No Pain) | | | | | |
| At Minute 1: | NP | 2.8 | | | |
| | P | 6.8 | 4.1 | -3.8 to 12.0 | .313 |
| At Minute 7 | NP | 3.0 | | | |
| | P | 7.0 | 4.0 | -3.9 to 12.0 | .318 |
| At Minute 15 | NP | 2.9 | | | |
| | P | -0.8 | -3.7 | -11.6 to 4.2 | .358 |
| Phase (ref to Early Phase) | | | | | |
| | Early | 9.4 | | | |
| | Mid | 3.9 | -5.5 | -7.4 to -3.6 | <.001 |
| | Late | -2.5 | -11.9 | -13.8 to -10.0 | <.001 |
| Minute (ref to Minute 1) | | | | | |
| No Pain Group | Min 1 | 2.8 | | | |
| | Min 7 | 3.0 | 0.2 | -1.8 to 2.2 | .857 |
| | Min 15 | 2.9 | 0.1 | -1.9 to 2.1 | .903 |
| Pain Group | Min 1 | 6.8 | | | |
| | Min 7 | 7.0 | 0.1 | -5.4 to 5.7 | .961 |
| | Min 15 | -0.8 | -7.7 | -13.2 to -2.1 | .007 |
| **Upper lumbar angle** | | | | | |
| Group (Pain – No Pain) | | | | | |
| At Phase 1 | NP | 10.5 | | | |
| | P | 9.1 | -1.4 | -8.0 to 5.2 | .682 |
| At Phase 2 | NP | 6.4 | | | |
| | P | 5.7 | -0.6 | -7.2 to 6.0 | .849 |
| At Phase 3 | NP | -3.0 | | | |
| | P | 1.0 | 4.0 | -2.6 to 10.6 | .233 |
| Phase (ref to Early 1) | | | | | |
| No Pain Group | | | | | |
| | Early | 10.5 | | | |
| | Mid | 6.4 | -4.2 | -5.6 to -2.7 | <.001 |
| | Late | -3.0 | -13.5 | -15.0 to -12.1 | <.001 |
| Pain Group | | | | | |
| | Early (1) | 9.1 | | | |
| | Mid (2) | 5.7 | -3.4 | -5.9 to -1.0 | .007 |
| | Late (3) | 1.0 | -8.1 | -10.6 to -5.7 | <.001 |
| Minute (ref to Minute 1) | | | | | |
| | Min 1 | 3.9 | | | |
| | Min 7 | 5.4 | 1.5 | -1.6 to 4.6 | .358 |
| | Min 15 | 5.6 | 1.6 | -1.5 to 4.7 | .302 |

**TABLE 4** - Percentage of drive phase in greater than 80% of flexion range for Lower and upper angle, for Pain and No Pain group.

| | Lower Lumbar Angle (%) | | Upper Lumbar Angle (%) | |
|---|---|---|---|---|
| Minute | No Pain | Pain | No Pain | Pain |
| 1 | 0.56 (0.34) | 0.69 (0.36) | 0.45 (0.33) | 0.68 (0.36) |
| 7 | 0.58 (0.34) | 0.62 (0.38) | 0.48 (0.17) | 0.77 (0.17) |
| 15 | 0.58 (0.34) | 0.49 (0.46) | 0.48 (0.16) | 0.52 (0.38) |

**TABLE 5** - Mixed model results for proportion of drive phase in >80% lower and upper lumbar end range flexion.

| | | Marginal means $(^0)$ | β coefficient $(^0)$ (contrast) | 95% CI | p-value |
|---|---|---|---|---|---|
| **Lower Lumbar Angle** | | | | | |
| Group (Pain – No Pain) | | | | | |
| | NP | .72 | | | |
| | P | .45 | -.27 | -.59 to .04 | .087 |
| Minute (ref to Minute 1) | | | | | |
| | Min 1 | .58 | | | |
| | Min 7 | .59 | .01 | -.04 to .06 | .647 |
| | Min 15 | .59 | .01 | -.04 to .06 | .657 |
| Covariates | | | | | |
| Age (yrs) | 16.6[a] | .59 | -.10[b] | -.20 to -.01 | .036 |
| Height (cm) | 177.6[a] | .59 | -.02[b] | -.04 to -.00 | .030 |
| Weight (Kg) | 72.5[a] | .59 | .01[b] | .00 to .02 | .080 |
| **Upper Lumbar Angle** | | | | | |
| Group (Pain – No Pain) | | | | | |
| | NP | .47 | | | |
| | P | .66 | .19 | .03 to .35 | .021 |
| Minute (ref to Minute 1) | | | | | |
| | Min 1 | .56 | | | |
| | Min 7 | .60 | .04 | -.09 to .19 | .509 |
| | Min 15 | .53 | -.03 | -.17 to .11 | .668 |

[a]mean of covariate in the sample

[b]β coefficient represents the expected change in proportion of drive phase spent in >80% end range flexion with each increase of one unit in the covariate

## Figure Caption

**FIGURE 1** – Regional lumbar kinematics (ULA – Upper Lumbar Angle; LLA – Lower Lumbar Angle)

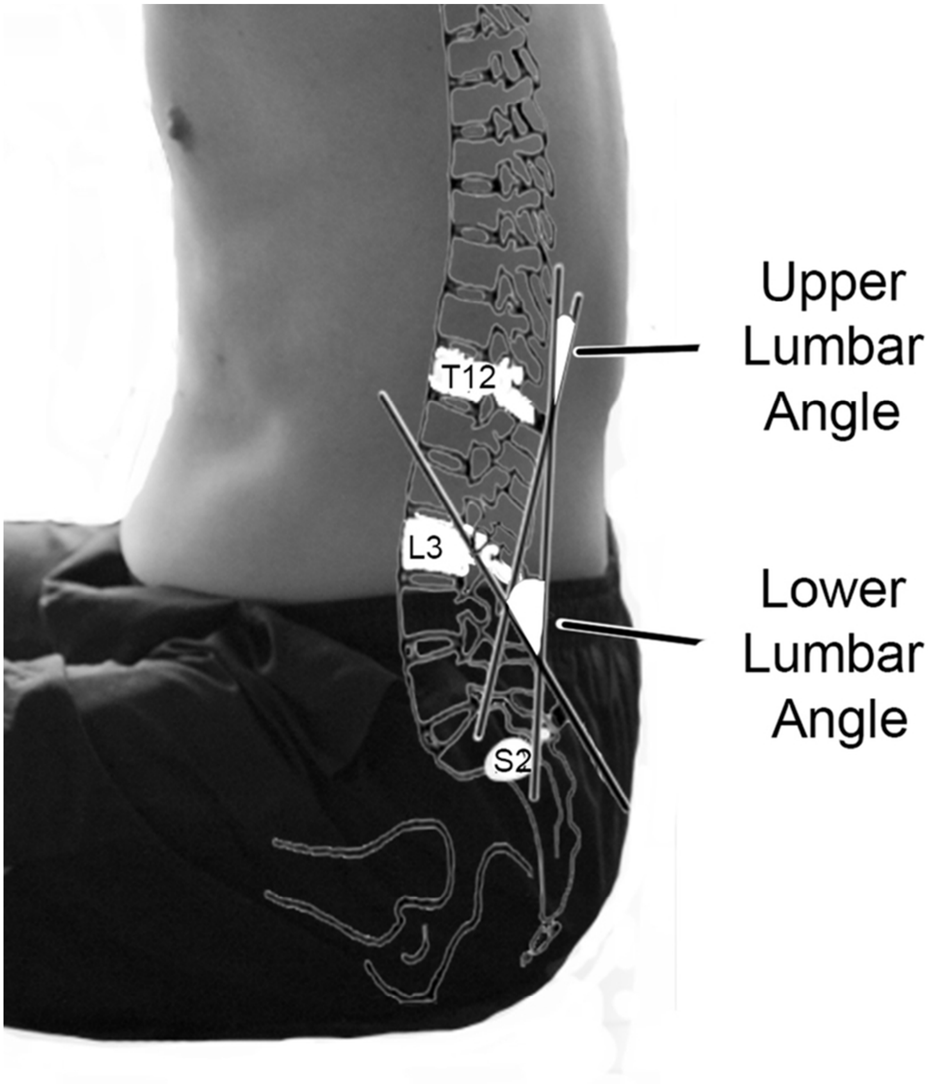
**FIGURE 2** – Group mean and standard deviation of low back pain intensity scores (measured by Numeric Pain Regional Scale) during the 15-minute rowing ergometer trial.

**FIGURE 3** – Lower lumbar angle for each subject over the $1^{st}$, $7^{th}$ and $15^{th}$ minute, for the early, mid and late drive phase separately, in pain and no pain groups separately.

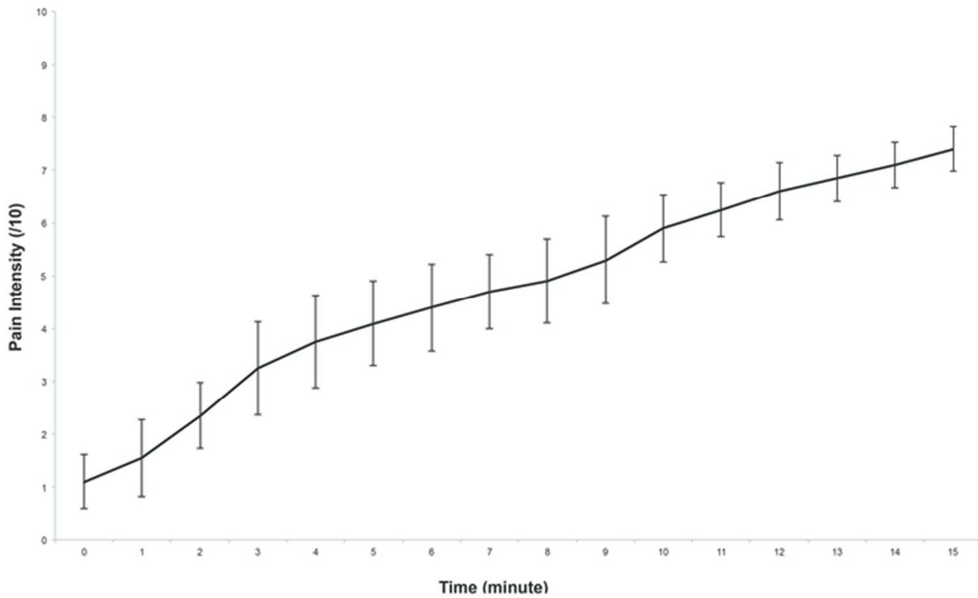
**FIGURE 4** – Upper lumbar angle for each subject over the drive phase separately for $1^{st}$, $7^{th}$ and $15^{th}$ minute, in pain and no pain groups separately.

**FIGURE 5A:** Proportion of drive phase lower lumbar angle in greater than 80% flexion over 1st, 7th and 15th minute, in pain and no pain groups separately

**FIGURE 5B:** Proportion of drive phase upper lumbar angle in greater than 80% flexion over 1st, 7th and 15th minute, in pain and no pain groups separately




Regional lumbar kinematics (ULA – Upper Lumbar Angle; LLA – Lower Lumbar Angle)
71x77mm (300 x 300 DPI)




Group mean and standard deviation of low back pain intensity scores (measured by Numeric Pain Regional Scale) during the 15-minute rowing ergometer trial.
60x36mm (300 x 300 DPI)

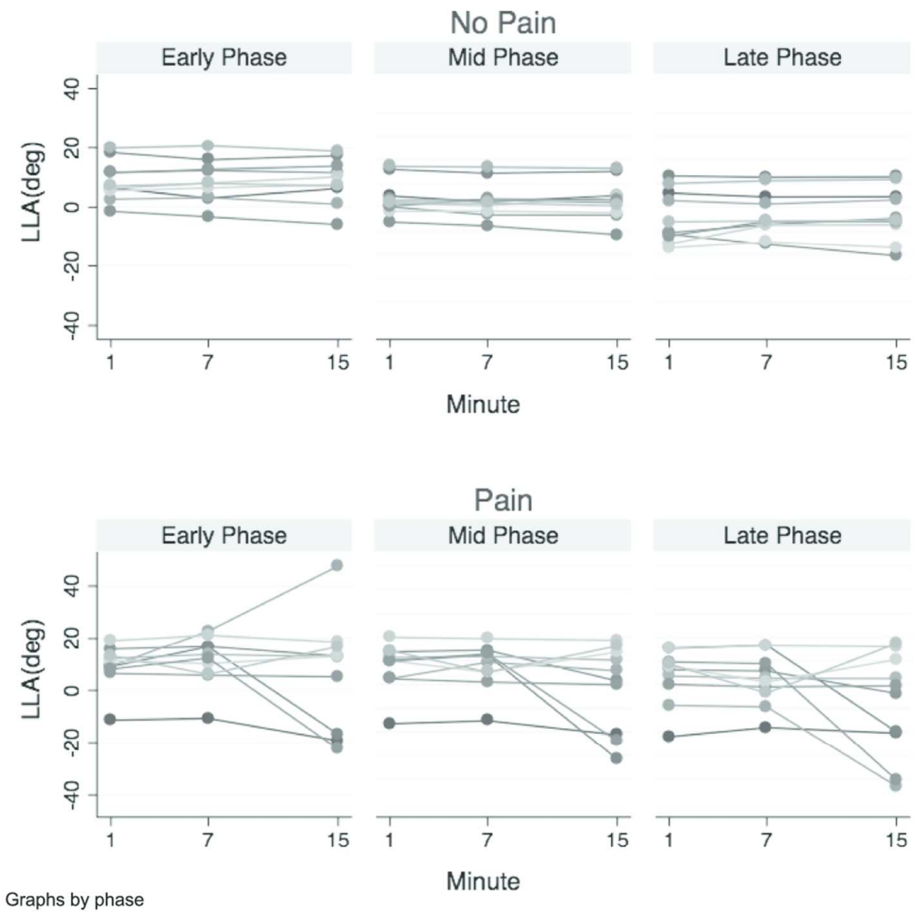



FIGURE 3 – Lower lumbar angle for each subject over the 1st, 7th and 15th minute, for the early, mid and late drive phase separately, in pain and no pain groups separately.
99x99mm (300 x 300 DPI)

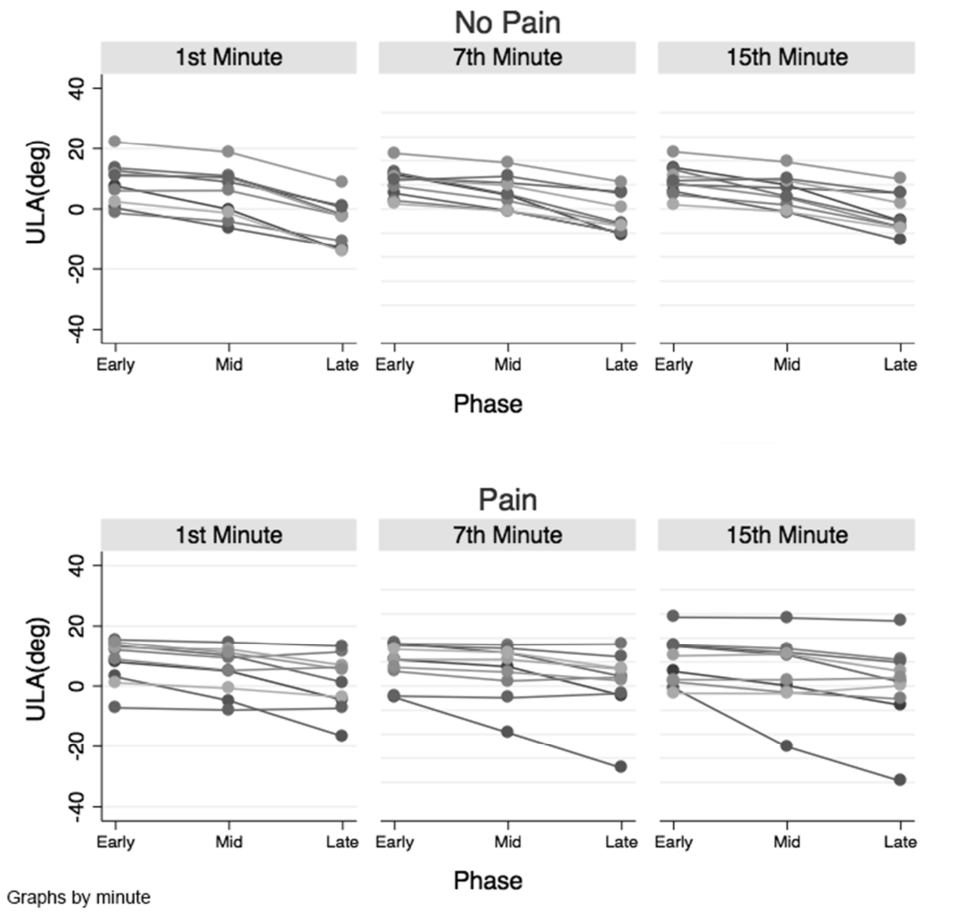



FIGURE 4 – Upper lumbar angle for each subject over the drive phase separately for 1st, 7th and 15th minute, in pain and no pain groups separately.
270x270mm (72 x 72 DPI)

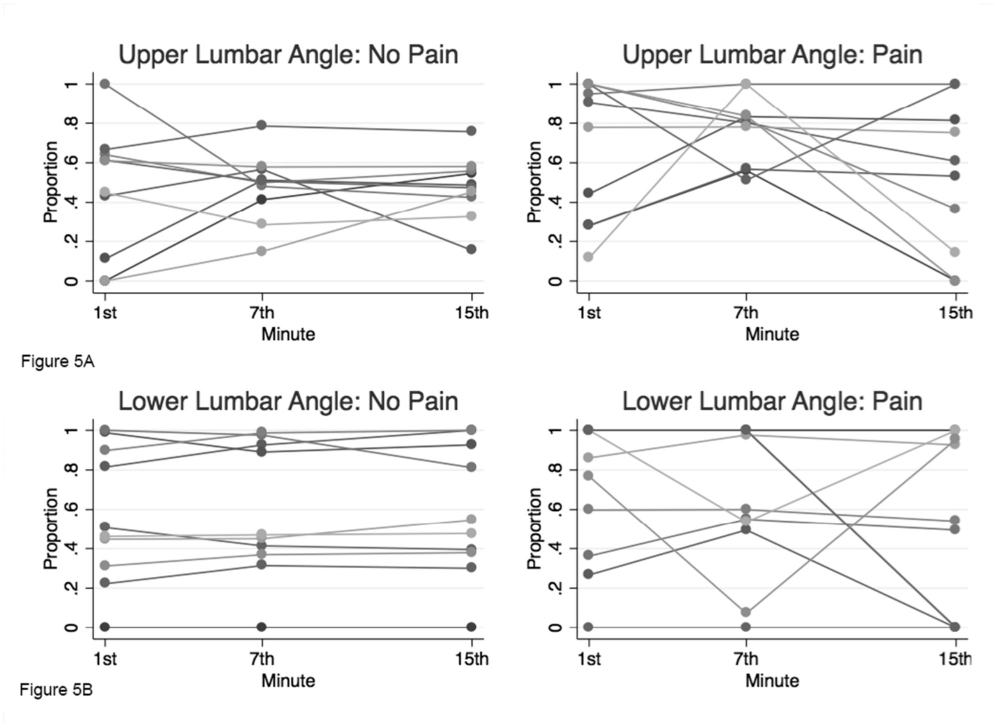



FIGURE 5A: Proportion of drive phase lower lumbar angle in greater than 80% flexion over 1st, 7th and 15th minute, in pain and no pain groups separately

FIGURE 5B: Proportion of drive phase upper lumbar angle in greater than 80% flexion over 1st, 7th and 15th minute, in pain and no pain groups separately

372x270mm (72 x 72 DPI)