Recruitment of Discrete Regions of the Psoas Major and Quadratus Lumborum Muscles Is Changed in Specific Sitting Postures in Individuals With Recurrent Low Back Pain

**STUDY DESIGN:** Cross-sectional controlled laboratory study.

**OBJECTIVES:** To investigate potential changes in the function of discrete regions of the psoas major (PM) and quadratus lumborum (QL) with changes in spinal curvatures and hip positions in sitting, in people with recurrent low back pain (LBP).

**BACKGROUND:** Although the PM and QL contribute to control of spinal curvature in sitting, whether activity of these muscles is changed in individuals with LBP is unknown.

**METHODS:** Ten volunteers with recurrent LBP (pain free at the time of testing) and 9 pain-free individuals in a comparison group participated. Participants with LBP were grouped into those with high and low erector spinae (ES) electromyographic (EMG) signal amplitudes, recorded when sitting with a lumbar lordosis. Data were recorded as participants assumed 3 sitting postures. Fine-wire electrodes were inserted with ultrasound guidance into fascicles of the PM arising from the transverse process and vertebral body, and the anterior and posterior layers of the QL.

**RESULTS:** When data from those with recurrent LBP were analyzed as 1 group, PM and QL EMG signal amplitudes did not differ between groups in any of the sitting postures. However, when subgrouped, those with low ES EMG had greater EMG signal amplitude of the PM vertebral body and QL posterior layer in flat posture and greater EMG signal amplitude of the QL posterior layer in short lordotic posture, compared to those in the pain-free group. For the group with high ES EMG, the PM transverse process and PM vertebral body EMG was less than that of the other LBP group in short lordotic posture.

**CONCLUSION:** The findings suggest a redistribution of activity between muscles that have a potential extensor moment in individuals with LBP. The modification of EMG of discrete fascicles of the PM and QL was related to changes in ES EMG signal amplitude recorded in sitting. J Orthop Sports Phys Ther 2013;43(11):833-840. Epub 10 October 2013. doi:10.2519/jospt.2013.4840

**KEY WORDS:** fine-wire electromyography, lumbar spine, postural control

Sitting may contribute to development and maintenance of low back pain (LBP), which may be related to changes in trunk muscle control. Although the psoas major (PM) and quadratus lumborum (QL) contribute to control of spinal curvature in upright sitting, it is not known whether the activity of these muscles changes in individuals with recurrent LBP. Opposing interpretations of compromised or augmented muscular activity have been debated, but there are no directly recorded data of PM and QL activity in sitting, and no previous work, to our knowledge, has considered the complex anatomy or the potential for changes in activation of discrete regions of the PM and QL.

Sitting commonly exacerbates LBP and difficulty adopting neutral,
midrange lumbar lordosis postures has been observed in individuals with LBP. Some individuals prefer to sit in a more kyphotic or lordotic lumbar posture compared to pain-free individuals, and these postures are related to differences in the level of muscle activity as measured with electromyographic (EMG) signal. Erector spinae (ES) EMG signal amplitude is reduced when healthy individuals adopt lumbar flexion/kyphosis postures and in people with LBP who use this posture naturally. In contrast, ES EMG is increased in extended/lordotic postures in healthy individuals and in patients who prefer this posture. Earlier studies of muscle activation levels in different postures. Additional insight can be gained from observation of EMG patterns required to adopt similar positions between groups. Studies of EMG in individuals with LBP when sitting have been limited to the superficial abdominal and paraspinal muscles. Yet activity of the deeply situated PM and QL muscles may also change. Disagreement on whether PM and QL activity is compromised or augmented has led to contrasting assumptions that clinical interventions should increase or reduce activity. Resolution of this debate has been compromised by a lack of direct EMG recordings and a failure to consider the potential for unique changes in separate LBP subgroups and different regions of these multifascicular muscles. Recent work has reported greater activity of PM fascicles arising from the transverse processes (PM-t) when sitting in a midrange lumbar lordosis compared to a flat-back posture, yet similar activity of the anterior fascicles from the vertebral body (PM-v) in these postures. QL EMG signal amplitude did not differ between sitting postures, but the activity level of the posterior QL fascicles (QL-p) was greatest during trunk extension and lateral flexion, whereas the activity of the anterior QL fascicles (QL-a) was greatest during trunk lateral flexion.

Different activation levels of distinct regions of the PM and QL, combined with anatomical/biomechanical data of these muscles, underpin several hypotheses regarding their activation levels with changes in spinal curvature in sitting in individuals with LBP. We hypothesized that PM and QL activity levels would differ between LBP subgroups that are defined on the basis of trunk ES EMG level when sitting. Specifically, individuals with recurrent LBP and low lumbar ES EMG signal amplitude (LB₃₋₄ESlow) may either reduce activity of the more posteriorly situated PM-t and QL-p, or increase PM and QL EMG as a generalized spinal-stiffening strategy. This study aimed to test these hypotheses.

Methods
Participants
Ten volunteers (mean ± SD age, 23 ± 4 years; height, 171 ± 11 cm; weight, 67 ± 12 kg; 6 male) with recurring episodes of LBP (multiple episodes of LBP separated by remissions) participated. Participants were pain free at the time of testing. Data from these 10 individuals with recurrent LBP were compared with previously published data from 9 healthy controls (mean ± SD age, 23 ± 3 years; height, 169 ± 5 cm; weight, 62 ± 8 kg; 7 male) with no history of LBP. Participants with recurrent LBP were included if they had their initial LBP episode (symptoms between T12 and gluteal fold) more than 12 months prior to the study, with subsequent LBP episodes severe enough to limit activities of daily living and/or cause time off from work and/or sports. Participants were excluded from both groups if they reported any major circulatory, cardiopulmonary, orthopaedic, or neurological conditions, or if they had back surgery or any surgery with an abdominal incision. Informed consent was obtained from all participants, and the rights of the participants were protected. All procedures were approved by the Institutional Medical Research Ethics Committee at The University of Queensland and were conducted in accordance with the Declaration of Helsinki.

Electromyography
A myoelectric signal was recorded from the PM and QL with bipolar, intramuscular fine-wire electrodes (Teflon-coated, 75-µm stainless steel wire, 1 mm of Teflon removed, threaded into a hypodermic needle [0.70 × 150 mm for PM and 0.65 × 70 mm for QL], bent back 1 and 2 mm to form hooks). Electrodes were inserted into the right PM-t, PM-v, QL-a, and QL-p at the level of the L3-4 interspinous space, with ultrasound guidance at 5 to 10 MHz (LOGIQ9; GE Healthcare, Waukesha, WI) (Figure 1). Anatomy of the different regions of each muscle was...
confirmed from preliminary investigation of cadavers and ultrasound examination of healthy volunteers. The middle layer of the QL was not examined due to its variable anatomy. Pairs of surface electrodes (Ag/AgCl discs, 10-mm diameter, 20-mm interelectrode distance; Noraxon USA Inc, Scottsdale, AZ) were placed over the right ES, 20 mm lateral to the L4 spinous process, and over the right obliquus externus abdominis (OE) and obliquus internus abdominis-transversus abdominis (OI/TrA) at standard landmarks. A ground electrode was placed over the right caudal rib cage.

EMG data were amplified 2000 times, band-pass filtered between 10 Hz and 1.5 kHz, and sampled at 3 kHz using a TeleMyo telemetered EMG system (Noraxon). EMG data were downsampled to 2 kHz using a Power1401-3 data-acquisition system with Signal software (Cambridge Electronic Design Ltd, Cambridge, UK).

**Procedure**

**Sitting Postures** Myoelectric signal was recorded while participants maintained 3 sitting postures on a flat-surfaced stool, with the height adjusted to each individual’s popliteal crease. Pictures of slump (kyphotic curves at thoracolumbar and lumbar regions), flat (minimal curve at thoracolumbar and lumbar regions), or short lordotic (lordotic lumbar and flat/kyphotic thoracolumbar curve) postures were shown to participants, along with verbal instruction and manual guidance to adopt each posture (FIGURE 2A). For the short lordotic posture, participants were taught to sit toward the front of their ischia using a subtle forward sacral tilt. Nine 20-second trials (3 repetitions of each posture in random order) were recorded, with a 60-second rest between trials to minimize fatigue. Participants maintained quiet breathing throughout.

Kinematic data of spinal curvature at 3 spinal regions were collected using a Vicon 3-D motion analysis system (OMG plc, Oxford, UK) with 8 cameras and commercial tracking software (Vicon Nexus; OMG plc). Data were collected at 100 Hz, and the system was calibrated to record from a volume of 5.6 m³. Only

![FIGURE 2](image-url)
calibrations with average residual errors of less than 1.0 mm in all cameras were accepted. Five reflective markers were adhered with double-sided tape to the skin over the spinous processes of T1, T5, T10, L3, and S2.

Maximal Voluntary Contraction Tasks
Maximal voluntary contractions of each muscle were performed with strong verbal encouragement for three 3-second contractions against manual resistance (60-second rest between trials) for EMG normalization. Three isometric tasks were performed for the PM in supine: trunk flexion with knees flexed and lumbar spine in contact with the support, right hip flexion at 30° with straight knees, and flexion of the right hip at 90°. For the QL, participants performed isometric trunk lateral flexion. For the ES, participants extended their trunk isometrically in prone. For the OE and OI/TrA, the participant was in a supine crook-lying position with the arms at 90° of shoulder flexion, and manual resistance was applied to the forearms and knees against an isometric trunk rotation to the right (OE) and left (OI/TrA).

Subgrouping of Participants With Recurrent LBP
The 10 participants with recurrent LBP were divided into 2 groups, based on the median value of ES EMG signal amplitude (4.17%) recorded in short lordotic sitting posture: those with high (LBPEShigh) and low (LBPESlow) activity of this muscle. This was not the activity recorded in natural sitting posture but the activity when adopting a specific spinal curvature.

Data Processing
Kinematic data of spinal position were exported and downsampled to 50 Hz. Thoracic, thoracolumbar, and lumbar angles in the sagittal plane were calculated as the angle between segments defined by 2 markers (FIGURE 2A) and averaged over the 5-second duration of the recording. The thoracic angle was measured between the T1-5 and T5-10 segments, the thoracolumbar angle between the T5-10 and T10-L3 segments, and the lumbar angle between the T10-L3 and L3-S2 segments. This method has been used previously to show differences in thoracic, thoracolumbar, and lumbar angles between different sitting postures.9 Kyphotic angles are positive. The angle between the L3-S2 segment and the horizontal approximated the hip angle.

EMG data were analyzed using MATLAB Version 7.5 (The MathWorks, Inc, Natick, MA). Data were high-pass filtered at 30 Hz (fourth-order Butterworth) to minimize movement artifacts. Electrocardiogram signal was removed using a modified turning-point filter.17 Root-mean-square EMG amplitude was calculated for 5 seconds and normalized to the greatest signal amplitude recorded across maximal voluntary contraction trials.

Statistical Analysis
Statistical analysis was performed using STATISTICA Version 8 (StatSoft, Inc, Tulsa, OK). The 3 spinal and hip angles were compared between the 3 postures and 2 groups (controls and LBP) or 3 groups (controls, LBPESlow, LBPEShigh) using a mixed-model analysis of variance (ANOVA). EMG signal amplitude for each muscle was compared between postures and groups using ANOVA models. Another separate analysis using ANOVA models was conducted in the controls, who were subgrouped into those with low and high ES EMG using the same criterion of median ES EMG amplitude used in the LBP group. This analysis compared the EMG amplitude of each muscle between 2 control groups and postures. Post hoc testing was undertaken with Duncan multiple-range tests, and significance was set at P < .05. Partial eta-squared was calculated to estimate the effect size. A small effect was defined as a partial eta-square of 0.01 or greater and less than 0.06, a medium effect as values of 0.06 or greater and less than 0.14, and a large effect as values of 0.14 or greater.10

RESULTS
Participants (n = 19) successfully adopted the target sitting postures. This was confirmed by differences in spinal angles between postures (interaction for angle by posture, P < .001; partial η² < 0.06). Spine angles in each posture did not differ between groups (interaction for group by angle by posture, P = .29; partial η² = 0.10) (FIGURE 2B), which indicates that our instruction was successful.

When participants with recurrent LBP were compared as a group against controls, there was no difference in PM and QL EMG signal amplitude in any sitting posture (group, P = .11; partial η² = 0.02; interactions for all, P > .26; partial η² < 0.06). However, when participants were subgrouped on the basis of ES EMG signal amplitude recorded in the short lordotic posture, those in the LBPESlow group had greater PM-v and QL-p EMG in the flat posture and greater QL-p EMG in the short lordotic posture than controls and LBPEShigh participants (interaction for group by muscle by posture, P < .01; partial η² = 0.19; post hoc for all, P < .05) (FIGURE 3A). In contrast, participants in the LBPEShigh group had less PM-t and PM-v EMG than LBPESlow participants in the short lordotic posture (post hoc for all, P < .05) (FIGURE 3A). In slump, all muscles were minimally active in all groups (post hoc for all, P > .23). ES, OE, and OI/TrA EMG signal amplitude did not differ between groups, whether or not they were subgrouped (post hoc, P > .13) (FIGURE 3B).

When control participants were subgrouped using the same criteria of median ES EMG signal amplitude used for the LBP group, there was no difference between those with low (n = 7) or high (n = 2) ES EMG for any posture (group, P = .98; partial η² < 0.001; interactions for all, P > .2; partial η² < 0.06). However, this analysis was limited by the infrequent use of higher EMG in the control group (n = 2), which calls into question the validity of this analysis. This strategy appears to be a feature of the LBPESlow subgroup and not common among controls.
DISCUSSION

When participants with recurrent LBP were considered as a single group, they could not be distinguished from controls on the basis of PM or QL EMG signal amplitude in sitting. However, when subgrouped by ES activation level in the short lordotic posture, those with low ES EMG signal amplitude had greater activity of the more posterior regions of the PM and QL than controls, whereas those with high ES signal amplitude used less activity of the PM than those in the LBP_{ESlow} group. This observation suggests a redistribution of activity between muscles with a potential extensor moment in specific subgroups of patients with recurrent LBP.

Changes in Activity of Regions of the PM and QL in the LBP_{ESlow} Group

The LBP_{ESlow} group was identified on the basis of less ES EMG signal amplitude when sitting in the short lordotic posture. Thus, we hypothesized that increased activity of other muscles with an extensor moment would be required to maintain the posture. Consistent with this hypothesis, activity of the QL-p was greater in the short lordotic sitting posture in the LBP_{ESlow} group than in the controls, but EMG of the PM-t (the extensor portion) was similar between groups. Whether greater activity of the QL-p compensates for reduced ES activity or is the cause of the decreased ES EMG is unclear. The flat-sitting posture requires less lumbar lordosis (ie, less extensor torque) compared to the short lordotic posture. In flat sitting, the LBP_{ESlow} participants, compared to controls, had greater EMG amplitude of the PM-v (which is more active in trunk flexion than extension)\(^3\) and QL-p (which is more active in extension than flexion).\(^4\) This implies a concurrent increase in trunk flexor and extensor torque, respectively, consistent with the objective to maintain the flat lumbar curvature in the sagittal plane. Unlike the QL-p EMG, the QL-a EMG was not different between groups. This may reflect

![Figure 3. EMG signal amplitudes (normalized to MVC) for (A) PM and QL and (B) ES, OE, and OI/Tra are shown across 3 different sitting postures: slump, flat, and short lordotic postures. Standard deviations are shown. *Participants with LBP_{ESlow} differed from the other 2 groups for the specific muscle (P<.05). †Difference between certain groups for the specific muscle (P<.05). Abbreviations: EMG, electromyographic; ES, erector spinae; LBP, low back pain; LBP_{ESlow}, participants with recurrent LBP with high activity of ES in short lordotic sitting; LBP_{ESlow}, participants with recurrent LBP with low activity of ES in short lordotic sitting; MVC, maximal voluntary contraction; OE, obliquus externus abdominis; OI/Tra, obliquus internus abdominis-transversus abdominis; PM, psoas major; PM-t, psoas major transverse process; PM-v, psoas major vertebral body; QL, quadratus lumborum; QL-a, quadratus lumbarum anterior; QL-p, quadratus lumbarum posterior.](chart)
the fact that this muscle has no spine attachment (QL-a runs from the 12th rib to the iliac crest) and has greater function in lateral flexion than in extension.\(^{26}\)

Evidence of increased activation of the PM-v and QL-p in the LBP\(_{\text{EShigh}}\) group while maintaining specific sitting postures parallels the clinical assumption of muscular overactivity in individuals with LBP. Although activity is not directly related to muscle-length changes, this augmented activity may underpin a tendency for these muscles to be shorter in those with LBP\(^{14,15,38}\) and is consistent with the observed greater asymmetry of the QL, with marked hypertrophy on the dominant-hand side, in cricket fast bowlers with LBP compared to those who were pain free.\(^{16,38}\)

A greater muscular activation level appears consistent with the goal to maintain upright posture and may have both positive and negative implications for spine control. On the one hand, increased activity of these deeply situated muscles may not be optimal, as these muscles have relatively small moment arms and, therefore, activation to keep the spine upright would be associated with substantial compressive forces applied to the spine,\(^{26}\) particularly from increased activity of the PM\(_v\) and QL. Thus, a shift toward a strategy biased toward these muscles may have consequences in the long term for spinal health as a result of sustained loading. However, a contrasting view is that the additional compression would enhance the stability of the segment\(^{27}\) and may be advantageous to increase the safety margin for control of the spine segments, at least in the short term. Future work is required to study the potential short- and long-term effects of this strategy.

**Reduced Activity of the PM in the LBP\(_{\text{EShigh}}\) Group**

Contrary to our hypothesis, PM and QL EMG was similar between the LBP\(_{\text{EShigh}}\) and control groups. However, PM-t and PM-v activity was less in the LBP\(_{\text{EShigh}}\) group than in the LBP\(_{\text{ESlow}}\) group (which had similar PM-t and PM-v EMG to controls) in the short lordotic sitting posture. This finding suggests that increased activity of the ES is sufficient to control lumbar lordosis in sitting, with less demand for activity of the extensor region of the PM (PM-t) in the LBP\(_{\text{EShigh}}\) group.

Recent data of the PM in sitting suggest greater changes in activity with subtle changes in spinal curvature than for the QL, ES, OE, and OI/TrA, and that PM-t has a combined potential to extend/lordose the lumbar spine and flex the hip in healthy individuals.\(^{26}\) The potential mechanical advantage of PM-t and PM-v assisting in the control of spine movement in these postures implies that a redistribution of activity in the LBP\(_{\text{EShigh}}\) group to ES alone may not be optimal for control of lumbar lordosis in sitting. High ES activity in participants with recurrent LBP may reflect a strategy of protective guarding or splitting of the spine, consistent with the predictions of earlier theoretical studies.\(^{3,21,24,25}\)

Compromised morphology and behavior of the PM in LBP have also been observed in other studies. The cross-sectional area of the PM is decreased ipsilateral to the pain in those with radicular pain and disc herniation.\(^{4,5}\) Such compromised morphology of the PM is consistent with compromised activity of other deep trunk muscles in LBP, such as delayed\(^{16,20}\) and reduced\(^{22}\) activity of the TrA and multifidus\(^ {24}\) muscles.

**Methodological Considerations**

This study investigated the function of discrete regions of the PM and QL at a single lumbar level (L3-4). As previous work highlights different functions of the PM at different levels,\(^ {4}\) these findings may not fully represent changes in PM activation with pain at the other lumbar levels. Although the LBP group was divided on the basis of ES activity in short lordotic posture, ES EMG between the LBP\(_{\text{ESlow}}\) and LBP\(_{\text{EShigh}}\) groups did not differ when all postures were included in the ANOVA. Inclusion of the statistical analysis of other sitting postures (which compromised the ability to identify a main effect for group when a difference was not apparent in all postures [similar activity between groups in slump]) and the small number of participants (which limited the power to detect a significant interaction between group and posture compared to a single comparison between 2 groups in 1 posture) likely explain the lack of difference between groups. The number of participants was relatively small due to the invasive nature of the experiment, but was sufficient to show significant differences (\(P<0.05\)) and a large effect size (partial \(\eta^2 = 0.19\)), according to the Cohen definition,\(^ {30}\) between subgroups when investigating the relationship between sitting postures and activity of trunk muscles.

**Clinical Implications**

The present findings show that there is redistribution of activity between muscles that have a potential trunk extensor moment in a specific subgroup of individuals with recurrent LBP, consistent with the predictions of recent theories of motor adaptation to pain.\(^ {21}\) The modification of activity of discrete fascicles of the PM and QL is reasonable based on changes in ES activity in sitting. The findings also suggest that the different strategies were adopted by different groups of patients with recurrent LBP to maintain the same specific sitting posture. This observation extends those of previous studies\(^ {12,38}\) that have shown different activation levels of the trunk muscles of people with LBP when they sat in different postures. Few clinical interventions consider different anatomical regions of the PM and QL. Thus, clinical interventions designed to reduce activity of the PM and QL by myofascial stretching, manipulation,\(^ {22}\) treatment of trigger points,\(^ {15,38}\) or the injection of botulinum toxin A\(^ {47}\) or contrasting clinical interventions with the intent to strengthen these muscles\(^ {27,28}\) may be appropriate for specific groups of patients and most effective when directed at specific anatomical regions of the muscles. Further investigation is required to examine the effectiveness of interventions...
targeting control of specific regions of the PM and QL.

CONCLUSION

Our findings show a redistribution of activity between muscles that have a potential extensor moment in a subgroup of individuals with recurrent LBP in specific sitting postures. Moreover, such redistribution was not uniform across all of the individuals with recurrent LBP who were tested, but changes in regional activity of the PM and QL were predictable based on ES activity in sitting.

KEY POINTS

FINDINGS: There is a redistribution of activity between muscles (including discrete regions of the PM and QL) that have a potential extensor moment in a specific subgroup of individuals with recurrent LBP during sitting in different postures.

IMPLICATIONS: Different strategies were adopted by different groups of people with LBP to maintain the same specific sitting posture. Such differential strategies involving unique modification of activity of discrete regions of the PM and QL could be predicted based on changes in ES activity in sitting.

CAUTION: The present study recorded PM EMG at a single lumbar level (L3-4). Caution is required when extrapolating these findings to muscle fascicles with more caudal and cranial origins.

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REFERENCES


