Title: Individuals with impaired lumbopelvic control demonstrate lumbar multifidus muscle activation deficit using ultrasound imaging in conjunction with electrical stimulation: A cross-sectional study.

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Abstract

Objective: To determine lumbar multifidus muscle (LM) activation deficits in individuals with impaired lumbopelvic control (iLPC) based on musculoskeletal ultrasound (MSKUS) in conjunction with electrical stimulation approach, and the correlation between back extension force and LM activation.

Design: A cross-sectional study design.

Setting: A university laboratory.

Participants: Fifty participants (25 iLPC and 25 NoLBP) were recruited from the university physical therapy clinic and surrounding areas.

Main Outcome Measures: The MSKUS was used to measure LM thickness at rest, maximum voluntary isometric contraction (MVIC), and electrical stimulation combined with MVIC, while a hand-held dynamometer was used to record force during MVIC and electrical stimulation combined with MVIC. These data were used to derive LM activation (LM_{ACT}) and percentage force generation ($Force_{GEN}$).

Results: The iLPC group had significantly lower LM_{ACT} (17%) than the NoLBP group ($P<0.05$). No significant difference was seen in $Force_{GEN}$ between the NoLBP and iLPC groups ($P>0.05$). No significant correlation was seen between LM_{ACT} and $Force_{GEN}$ ($P>0.05$).

Conclusion: The findings support the utility of our protocol to determine LM activation deficits. The lower LM activation in iLPC group suggests that individuals with iLPC were unable to fully recruit the motor units available in LM. Force generation measurements may not be an appropriate approach to determine such deficits in LM.

Key words: Impaired lumbopelvic control; Recurrent low back pain; Lumbar multifidus muscle; Ultrasound imaging; Neuromuscular electrical stimulation; Muscle activation deficit

List of abbreviation

COMB	Maximum voluntary isometric contraction combined with neuromuscular electrical stimulation
ICC	Intraclass correlation coefficient
iLPC	Impaired lumbopelvic control
IQR	Inter-quartile range
LBP	Low back pain
LM	Lumbar multifidus muscle
MDD_{95}	95% confidence minimal detectable difference
MSKUS	Musculoskeletal ultrasound
MVIC	Maximum voluntary isometric contraction
NMES	Neu

romuscular electrical stimulation

NoLBP	Individuals without low back pain
rLBP	Recurrent low back pain

INTRODUCTION

Mechanical low back pain (LBP) is one of the most common musculoskeletal conditions worldwide.[1] Although clinical practice guideline recommends several interventions to improve pain and disability, the recurrence rate is still high.[1,2] This could be because mechanical LBP has different underlying impairments.[2-6]

Clinical lumbar instability has been identified as a subgroup of LBP.[7,8] This subgroup demonstrates impaired lumbopelvic control (iLPC) represented by observed aberrant movement patterns during functional movements.[7-9] These repeated aberrant movements result in excessive tissue stress and microtrauma which could further cause episodes of LBP.[5] Furthermore, evidence indicates the existence of these aberrant movements in individuals with both a history of LBP and chronic LBP,[4,9-11] indicating that iLPC may be responsible for recurrent LBP (rLBP).[4,9-11]

One possible cause that could be responsible for such iLPC is arthrogenic muscle inhibition of the lumbar multifidus muscles (LM).[12,13] This reflex inhibition may reduce the ability of LM to generate sufficient force to stabilize the lumbar spine; thereby, increasing the shear forces on the lumbar spine resulting in tissue stress and microtrauma.[5,12,13] It has been reported that the LM provides up to two-thirds of the force generation needed to stabilize the lumbar spine.[14] In addition, the LM does not spontaneously recover after an episode of LBP, suggesting the existence of continued deficits in LM activation.[12,13,15-17] Although decreases in LM activity in individuals with rLBP have been reported using surface and intramuscular electromyography,[18,19] no study has previously investigated the amount of such LM activation deficits.

Force production with neuromuscular electrical stimulation (NMES) is one of the most common techniques used to quantify muscle activation deficits.[20-22] Previous studies have reported that when participants were asked to perform maximum voluntary isometric contractions (MVIC)

and NMES was then applied to superimpose MVIC, this offers a better representation of the maximum force from all motor units available in the muscle, which can be used to determine the amount of muscle activation deficit.[20-22] However, this technique was used to measure limb muscle activation, which can selectively investigate the muscle of interest. Therefore, this technique might not be suitable to measure the LM deficits as the back extension force is generated from all the back muscles, and not just LM. Accordingly, the overall force generated may not be correlated with the LM activation.

To overcome the challenge of force production using the NMES technique, musculoskeletal ultrasound (MSKUS) can be used in conjunction with the NMES to specifically investigate LM activation deficit.[23] Several studies and our pilot study demonstrated association between LM thickness measured by the MSKUS and LM activity using surface electromyography suggesting the validity of the MSKUS to measure LM activation.[24-27] Therefore, the MSKUS could be used to fulfill the gap in the knowledge regarding the degree of LM activation deficit.

The objectives of this study were to determine the existence of LM activation deficit in individuals with iLPC, and the correlation between back extension force and LM activation. We hypothesized that the iLPC group would demonstrate lower LM activation compared with individuals with no LBP (NoLBP), and that there would be no correlation between back extension force and LM activation.

METHODS

Participants

Fifty participants aged between 18 and 40 years (25 iLPC and 25 NoLBP) were recruited to the study. The inclusion criteria for iLPC were; at least two episodes of LBP that interfered with activities of daily living or required treatment, but were pain free at the time of data collection,

presence of aberrant movement during active standing trunk flexion, and an average passive straight leg raise of greater than 91 degrees. These inclusion criteria are supported by previous literature that demonstrated that an age of less than 40 years, presence of aberrant movement, and a passive straight leg raise greater than 91 degrees were predictors for iLPC.[7,28,29] Aberrant movements were defined as a painful arc in flexion or on return, Gower sign "thigh climbing", instability catch or reversal of lumbopelvic rhythm.[17] Participants with iLPC were selected during remission of pain to ensure that LM activation deficit was as a result of unrecovered LM, not just the response to the pain. The inclusion criteria for NoLBP were; no previous episodes of LBP, absence of aberrant movement, and a passive straight leg raise of less than 91 degrees. The participants were excluded if they had clinical signs of systemic disease, definitive neurologic signs including pain, previous spinal surgery, severe spinal stenosis and/or inflammatory joint disease, and BMI greater than 30 kg/m^2. An initial pilot study demonstrated that the iLPC group had lower LM activation than NoLBP with moderate effect size (Cohen's $d = 0.73$). We used this effect size with a one-tailed level of confidence with an alpha of 5% with a power of 80% to calculate the sample size. This determined that fifty participants, 25 in each group, were required for this study. Data were collected between June 2019 and March 2020.

Instruments and measures

A musculoskeletal ultrasound (MSKUS; model CX50, Philips, NV, USA) with a broadband linear array transducer (12-3 MHz) was used to measure bilateral LM thickness at the L4-5 facet joints (2 cm lateral to the lower half of the L4 spinous process).[23] Gain, frequency, depth, and focus were adjusted by the researcher for each participant. Several studies support the validity and reliability of the MSKUS to measure LM thickness in both individuals with and without LBP.[23-27] In addition, a hand-held dynamometer (Power Track II Jtech Commander, USA)

was used to measure back extension force, which was positioned and attached using straps at the thoracic spine (T3). Studies supported the validity and reliability of the hand-held dynamometer,[30,31] and the pilot data demonstrated an excellent test-retest reliability of back extension force measurements ($ICC_{3,2} = 0.94$).

Procedure

This cross-sectional study was designed to explore the existence of LM activation deficits in individuals with iLPC, and the correlation between back extension force and LM activation. The study protocol was approved by the Mahidol University institutional review board (MU-CIRB 2018/215.0712). All participants provided written informed consent prior to data collection. Demographic data from both groups and LBP behavior information from the iLPC group were collected.

The researcher who performed the data collection was blinded to the group allocation. The researcher received 3 hours of training by a MSKUS-certified expert and 50 hours of practice prior to data collection. The participants were asked to position themselves in prone lying on a treatment table, and the thorax (T3 level) and pelvis (S2 level) were then securely fastened to the table. A hand-held dynamometer was placed at the thorax to measure back extension force. Neuromuscular electrical stimulation (NMES; model Sonopuls 490 combination therapy, Enraf-Nonius BV, Netherlands) was used to deliver an electrical current to superimpose the MVIC. To measure right LM activation, a pair of adhesive electrodes (5cmX5cm) were attached at 3 cm lateral to the L3 and L5 spinous process levels on the right side, while another pair of electrodes were placed on the left side thus creating two diagonal lines intersecting at the right side LM (Fig 1A). Then, the ultrasound transducer was positioned in the longitudinal view at zero inclination on the L4-5 facet joint on the right side (Fig 1B).

The participant was asked to put both their hands behind their neck and relax in the prone position, while the researcher took two recordings of the LM thickness at rest (Fig 1B). The participant was then asked to perform 2 repetitions of a back extension MVIC against the hand-held dynamometer for 5 seconds with 1-minute rest between repetitions (Fig 1C; NMES: off). The instruction was "Please lift your head, trunk, and upper extremities using your lower back with maximum effort against the hand-held dynamometer attached on the strap while both ultrasound and force data were simultaneously collected". One practice trial was provided to ensure that the participant extended from the lower back and performed the task correctly according to the protocol. The MVIC value represents the participant's ability to recruit motor units in the muscle. Then, the NMES was applied to recruit additional motor units during MVIC. The NMES was set at interferential mode (6000 Hz, beat frequency 20-50 Hz, scanning effect).[32-34] The researcher increased NMES intensity until the point of maximum tolerance. The participant was asked to perform another 2 repetitions of a 5-second MVIC with NMES (Fig 1C; NMES: on), while LM thickness and peak force were concurrently collected. The LM thickness values from resting, MVIC and combined MVIC with NMES (Fig 1D) were then used to calculate the percentage of muscle activation, a value of less than 100 indicating a muscle activation deficit.[20-22] A one-minute rest period was provided between repetitions to prevent muscle fatigue. For the measurements combining MVIC with NMES, the LM thickness was recorded from a 5-second video file as the frequency was modulated between 20-50 Hz using the scanning technique. The entire process was performed to measure left LM activation, while the NMES electrodes were re-attached to create two diagonal lines intersecting over the LM on the left side. The overall protocol was approximately 15 minutes.

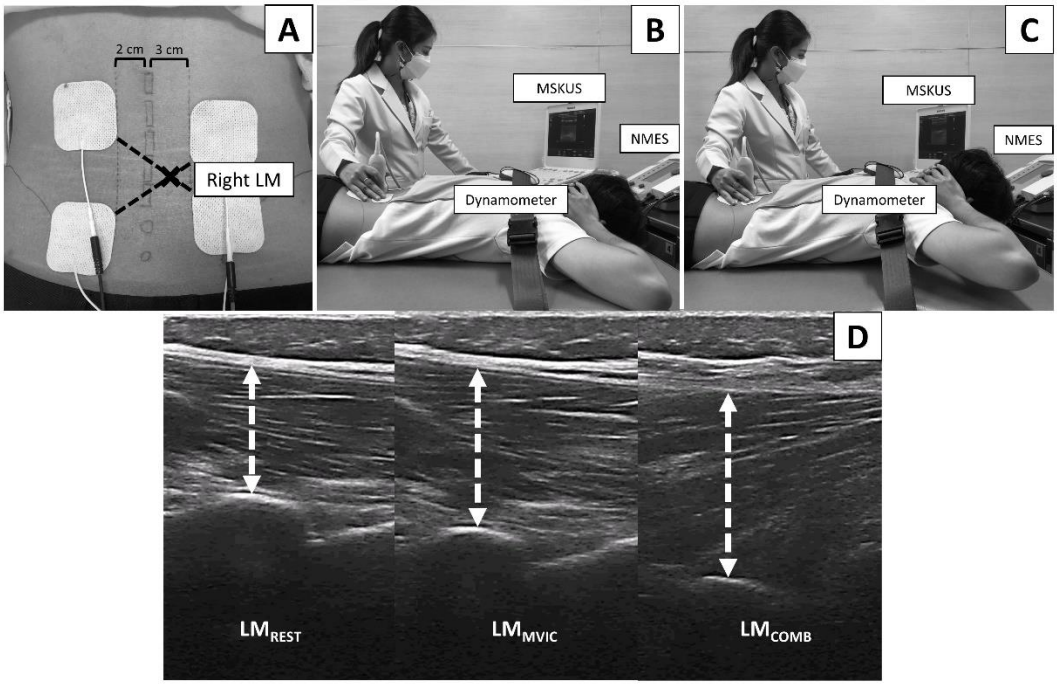

Fig 1. (A) Electrode placement for neuromuscular electrical stimulation (NMES) to activate right lumbar multifidus muscle (LM). (B) Example of participant's setup to measure LM thickness during resting (LM_{REST}) and (C) during maximum voluntary isometric contraction (LM_{MVIC}) and combined NMES with MVIC (LM_{COMB}). (D) LM thickness during rest, MVIC, and combined NMES with MVIC.

Data reduction

Data analysis was performed using a custom Matlab program (version R2014a, The MatWorks Inc, MA, USA). The researcher blinded to the group allocation measured the LM thickness from the resting and MVIC images (LM_{REST} and LM_{MVIC}), respectively. For the video files, the researcher selected the frames taken during the relaxation period and measured the LM thickness, then selected a frame showing maximum LM thickness during combined MVIC with NMES to measure LM thickness (LM_{COMB}). Our reliability analysis was based on all presented

data and showed excellent intra- and inter-rater reliability ($ICC_{3,1}$ = 0.97 and $ICC_{2,2}$ = 0.95, respectively) for LM thickness measurement, and the 95% confidence interval of the minimal detectable difference (MDD_{95}) was 0.08 cm. The averaged values from LM_{REST}, LM_{MVIC}, and LM_{COMB} across the two repetitions were used to calculate the percentage of LM activation (LM_{ACT}) using the following formula.[23]

$$LM_{ACT} = \left(\frac{LM_{MVIC} - LM_{REST}}{LM_{COMB} - LM_{REST}}\right) X\ 100$$

A previous study has demonstrated that no significant side-to-side differences in LM morphology exist, even in the case of unilateral LBP.[35] In addition, our preliminary data analysis demonstrated no significant difference between right and left LM_{ACT} in the NoLBP group, the ipsilateral and contralateral sides in individuals presenting with unilateral pain, or between the right and left sides in individuals presenting with bilateral pain, therefore left and right averaged LM_{ACT} data were used for statistical analysis.

The back-extension force data were normalized to the participants body weight, and the averaged values across the two repetitions for MVIC and COMB ($Force_{MVIC}$ and $Force_{COMB}$), were taken respectively. These were then used to calculate the percentage force generation ($Force_{GEN}$) based on the formula below. The percentage force generation was used to compare between groups and determine the correlation with LM_{ACT}. Our pilot data demonstrated excellent test-retest reliability of the force measurements normalized to body weight during MVIC and COMB ($ICC_{3,1}$ = 0.95 and 0.97), respectively, with MDD_{95} of 7.4 and 6.6% of body weight, respectively.

$$Force_{GEN} = \left(\frac{Force_{MVIC}}{Force_{COMB}}\right) X\ 100$$

Statistical analysis

Statistical analysis was performed using SPSS version 21 (IBM Corp., NY, USA) in conjunction with MDD_{95}. The distribution of the data was tested using Shapiro-Wilk tests, and the LM thickness data were found to be normally distributed; therefore, a two-way mixed ANOVA was used to determine the interaction between group and condition with planned post-hoc comparisons between-group at rest, MVIC, and COMB, as well as within-group tests between rest and MVIC, and between MVIC and COMB for each group. However, LM_{ACT} was found to be not normally distributed; thus, a Mann-Whitney U test was performed to compare the LM activation between groups. In addition, the normalized force data and percentage force generation were also not normally distributed; therefore, Mann-Whitney U tests were performed to compare between groups for MVIC and COMB conditions for the percentage force generation, and Wilcoxon tests were used to compare within group differences between the MVIC and COMB data. Finally, a Spearman's Rank correlation was used to determine the relationship between LM activation and percentage force generation.

RESULTS

Demographic data are presented in Table 1. The two-way mixed ANOVA showed no significant interactions between condition and group for the LM thickness, as well as no significant main effect of group. However, a significant main effect was seen for condition ($P<0.05$). The planned post-hoc comparisons (Table 2) revealed significant differences ($P<0.05$) in LM thickness between LM_{REST} and LM_{MVIC}, as well as LM_{MVIC} and LM_{COMB} with differences greater than MDD_{95} (0.08 cm). LM_{REST} did not show a significant difference between groups ($P>0.05$).

Table 1. Demographic data

Parameter	NoLBP	iLPC
Age (years)	22.2±2.3	22.8±2.6
BMI (kg/m^2)	22.0±1.6	21.4±2.3
Sex (%female)	60	60
Duration (months)	N/A	36.9±28.3
Frequency per year (number of episodes)	N/A	11.2±13.5
Time since last episode (days)	N/A	24.1±24.2
Duration for last episode (days)	N/A	2.8±2.8
Pain at last episode (0=no pain, 10=intolerable pain)	N/A	4.5±1.4
Disability at last episode (0=no disability, 10=total disability)	N/A	3.2±1.7

NoLBP = no history of low back pain in lifetime, iLPC = impaired lumbopelvic control, BMI = body mass index.

Table 2. Planned post-hoc pairwise comparisons between groups (mean±SD) for lumbar multifidus muscle thickness at rest (LM_{REST}), maximum voluntary isometric contraction (LM_{MVIC}), and combined MVIC with neuromuscular electrical stimulation (LM_{COMB}), as well as percentage lumbar multifidus muscle activation (LM_{ACT}) between groups (median (IQR))

Parameter	NoLBP	iLPC	Between-group mean/median diff (NoLBP vs iLPC)
LM_{REST} (cm)	2.64±0.46	2.58±0.55	0.06
LM_{MVIC} (cm)	3.39±0.56	3.28±0.64	0.11†
LM_{COMB} (cm)	3.51±0.57	3.55±0.63	0.04
Within-group mean diff (LM_{REST} vs LM_{MVIC})	0.76*†	0.70*†	
Within-group mean diff (LM_{MVIC} vs LM_{COMB})	0.12*†	0.27*†	
LM_{ACT} (%activation)	91.6 (82.7,93.8)	75.9 (64.0,83.3)	15.7*

NoLBP = no history of low back pain group, iLPC = impaired lumbopelvic control group, SD = standard deviation, IQR = interquartile range

* = significant difference (P<0.05)

† = exceeded 95% confidence minimal detectable difference

For LM_{ACT}, the results demonstrated that the iLPC group had significantly lower values than the NoLBP group (P<0.05); with a median = 75.9, interquartile range; IQR = 64.0 and 83.3 for the iLPC group, and median = 91.6, IQR = 82.7 and 93.8 for the NoLBP group, respectively.

The non-parametric Mann-Whitney U and Wilcoxon tests did not show any significant differences (P>0.05) in normalized force generation between-group and within-group comparisons (Table 3), respectively, and the median differences did not exceed MDD_{95}. In addition, no significant difference was seen in $Force_{GEN}$ between the NoLBP and iLPC groups; with a median = 101.2, IQR = 88.4 and 109.4 for the NoLBP group, and median = 95.7, IQR = 87.5 and 102.8 for the iLPC group. In addition, the Spearman's Rank correlation coefficient demonstrated no significant correlation between LM_{ACT} and $Force_{GEN}$ (rho=0.18, P>0.05).

Table 3. Comparisons between groups (median (IQR)) for normalized force generation at maximum voluntary isometric contraction (F_{MVIC}), and combined MVIC with peripheral electrical stimulation (F_{COMB}), as well as the percentage force generation (F_{GEN}) between groups (median (IQR))

Parameter	NoLBP	iLPC	Between-group median diff (NoLBP vs iLPC)
F_{MVIC} (%body weight)	20.5 (17.1, 26.9)	24.0 (17.4, 25.9)	3.5
F_{COMB} (%body weight)	20.7 (16.0, 26.2)	23.6 (16.9, 28.3)	2.9
Within-group median diff (F_{MVIC} vs F_{COMB})	0.2	-0.4	
F_{GEN} (%force generation)	101.2 (88.4, 109.4)	95.7 (87.5, 102.8)	5.5

NoLBP = no history of low back pain group, iLPC = impaired lumbopelvic control group, IQR = interquartile range

DISCUSSION

The significant findings from LM thickness for within-group comparisons (LM_{REST} vs LM_{MVIC}, and LM_{MVIC} vs LM_{COMB}) exceeded the MDD_{95}, this suggests a true change between resting and MVIC, and NMES concurrently with MVIC which was shown to elicit a greater LM activation.[20-22] These findings support the utility of our protocol to determine LM activation. Although there were no significant differences in LM thickness between groups in any condition, a within-group difference demonstrated the pattern in which the NoLBP group showed a greater

increased LM thickness from rest to MVIC than the iLPC group, while the iLPC group had a greater LM thickness change from MVIC to COMB than the NoLBP group. These findings suggest that the NoLBP group could increase LM activation volitionally, while the iLPC group required the NMES to facilitate LM activation.

The main findings from the LM activation supports our hypothesis in which individuals with iLPC had lower LM activation than those without LBP. This indicates the perseverance of LM activation deficit in individuals with iLPC.[12,13,15-17] This lower LM activation in iLPC group suggests that individuals with iLPC were unable to fully recruit all motor units available in the LM. This muscle activation deficit in the LM could be as a result of the persistence of a reflex inhibition mechanism.[12,13] This reflex inhibition mechanism initially occurs after injury to the tissue in the lumbar region,[12,13] resulting in abnormal afferent signals from damaged tissues which in turn reduce the motor commands to the LM.[12,13] This mechanism inhibits the individual's ability to fully recruit all motor units available in the LM.[12,13] As a result, the LM might not be able to provide sufficient force to stabilize the lumbar spine, thereby making the individual susceptible to re-injury of the lumbar spine.[5-8,12]

Fatty infiltration or muscle atrophy could be another potential factor that reduces the contractility of the LM resulting in decreased LM activation in individuals with iLPC,[15,36-40] which in turn can reduce lumbar stability. In addition, the LM activation deficit may also be related to changes in corticospinal mapping of the LM.[41] This change at the cortical level may compromise the ability to control and refine LM contractions,[41] which may be represented as LM activation deficits in our study. One study reported that NMES can send ascending activation to the sensorimotor cortex in the brain during electrical stimulation.[42] This mechanism has been shown to enhance the motor unit recruitment during combined MVIC with NMES.[42]

No study has previously used MSKUS in conjunction with the NMES to determine the LM activation. Therefore, we do not have results from other studies to directly compare the LM activation with ours. The finding in percentage LM activation is consistent with previous studies using surface electromyography in which they have found that patients with chronic/recurrent LBP had lower LM activation when compared with healthy individuals.[18,19] This can be considered as a confirmatory study for this point. Although using EMG amplitude can be interpreted as muscle activation deficit,[18,19] this approach failed to determine the extent of this deficit. Our approach uses the superimposition technique in which the NMES passively helps the individual to recruit more motor units in the LM.[10,13,29] Therefore, the percentage LM activation derived from our approach could be used to indirectly determine the extent of any deficit.[20-22] In this study, individuals with iLPC had approximately 17% less LM activation when compared to individuals with NoLBP.

Although not significant the difference in LM thickness during MVIC exceeded the MDD_{95}, this may be as a result of an underpowered statistical analysis. Our sample size calculation was based on a large effect size from our pilot study which may have inflated the sample size. Another potential explanation could be that LM fibers have transformed to type II fibers (fast-twitch fibers) which have larger diameter causing greater thickness change during cross-bridge.[39,42] Studies using MRI and muscle biopsy demonstrated a lower proportion of type I fibers and a greater proportion of type II fibers in patients with chronic LBP.[39,42] This transformation could be a coping strategy to compensate for insufficient force from type I fibers. Although this strategy is beneficial to stabilize the lumbar spine, muscle fiber type II is more susceptible to muscle fatigue.[12] This mechanism might be responsible for recurrence of LBP symptoms. However, our study did not have the data to confirm our interpretation based on muscle fiber types. Future studies to investigate the LM muscle type in individuals with iLPC is required.

As we expected, the results did not show a significantly lower force generation in the iLPC group when compared with the NoLBP group. These non-significant results could be resulted from the weakness of the approach using normalized force with NMES superimposition. Our results did not show significant increase in normalized force during combined MVIC with NMES comparing with MVIC in both groups even though we found a significant increase in LM thickness.

In addition, no correlation between LM activation and percentage force generation could be another potential explanation for non-significant difference in force generation. Based on basic anatomy, the LM has a short lever arm spanning only 1-2 vertebral segments.[12,14] Therefore, the LM might not generate greater force even though the thickness change was significant. In addition, the LM is primarily responsible for providing lumbar stability, rather than generating a force to move the spine into extension.[12,14] Another potential explanation is that our force measurement does not offer a representation of the LM function.[23] The force can be viewed as an output from all back-extensor muscles, and not specifically from LM.

Our approach described in this study was reliable, and our findings support the validity for LM activation measurement in which our approach had the ability to differentiate the amount of LM activation between individuals with iLPC and no LBP. Our findings suggest the clinical application to evaluate LBP patients in an iLPC subgroup. This approach could be used in future studies to determine the ability of therapeutic exercises to restore LM activation.

Study limitations

Some limitations should be taken into consideration in this study. Firstly, we used an iLPC subgroup of LBP, which limits the generalizability of these results to the wider LBP population. Secondly, we did not control the characteristics (pain intensity, disability level, etc.) of the participants with iLPC. These characteristics may affect our outcome measures, although these are

similar to those reported in the literature. Participants in this study did not have current pain, therefore, we were unable to determine the role of pain during LM activation. In addition, we did not collect fear avoidance behavior which may be associated with the memory of pain during muscle contractions. Therefore, future studies should take pain and the memory of pain into consideration. Another limitation is that we have assumed that the LM thickness can be used to represent the LM motor unit activation. The LM thickness data can be confounded by the LM physiological changes (e.g. fatty infiltration, muscle fiber type, etc.). We used a symmetrical task for LM activation, however this might not be the best task to evaluate LM activation, and several studies have reported using contralateral arm elevation against a force to activate LM function. Therefore, future studies should consider using asymmetrical tasks when measuring LM thickness as suggested by Sweeney et al.[38] In addition, this study did not include an investigation of the motor unit behavior. Recent studies have shown the utility of decomposition EMG to directly measure the motor unit behavior which may yield more information on any possible activation deficits in the LM.[43]

CONCLUSION

This is the first study using the NMES in conjunction with MSKUS and force measurement to investigate LM activation and back extensor force generation in individuals with iLPC and NoLBP and determine the correlation between LM activation and force generation. We found that the iLPC group had lower LM activation than the NoLBP group indicating the perseverance of an LM activation deficit. However, we did not find a difference in force generation between the groups, nor a correlation between LM activation and force generation. The lack of a significant correlation could be due to the fact that the force generation is an output from all back extensor

muscles, and not specifically from LM. These findings suggest that force generation measurements may not be an appropriate approach to determine LM activation.

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