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Original article

Functional and morphological lumbar multifidus characteristics in subgroups with low back pain in primary care

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ABSTRACT

Background: Since the contribution of the lumbar multifidus(LM) is not well understood in relation to non-specific low back pain(LBP), this may limit physiotherapists in choosing the most appropriate treatment strategy.
Objectives: This study aims to compare clinical characteristics, in terms of LM function and morphology, between subacute and chronic LBP patients from a large clinical practice cohort compared to healthy controls.
Design: Multicenter case control study.
Method: Subacute and chronic LBP patients and healthy controls between 18 and 65 years of age were included. Several clinical tests were performed: primary outcomes were the LM thickness from ultrasound measurements, trunk range of motion(ROM) from 3D kinematic tests, and median frequency and root mean square values of LM by electromyography measurements. The secondary outcomes Numeric Rating Scale for Pain(NRS) and the Oswestry Disability Index(ODI) were administered. Comparisons between groups were made with ANOVA, p-values<0.05, with Tukey's HSD post-hoc test were considered significant.
Results: A total of 161 participants were included, 50 healthy controls, 59 chronic LBP patients, and 52 subacute LBP patients. Trunk ROM and LM thickness were significantly larger in healthy controls compared to all LBP patients($p < 0.01$). A lower LM thickness was found between subacute and chronic LBP patients although not significant($p = 0.11$–0.97). All between-group comparisons showed no statistically significant differences in electromyography outcomes ($p = 0.10$–0.32). NRS showed no significant differences between LBP subgroups($p = 0.21$). Chronic LBP patients showed a significant higher ODI score compared to subacute LBP patients($p = 0.03$).
Conclusions: Trunk ROM and LM thickness show differences between LBP patients and healthy controls.

1. Introduction

Low back pain is a common problem in developed countries, with a reported life-time prevalence up to 84% (Airaksinen et al., 2006). Low back pain results in significant levels of disability and restrictions in daily activities including the inability to work (Kuijer et al., 2006). Worldwide, low back pain has the highest ranking in the years lived with disability index (Vos et al., 2012). Approximately 85% is classified as multifactorial or '-non-specific low back pain-' (LBP) (Steele et al., 2014) and most are firstly seen in primary care (Foster Nadine, 2018).

In clinical practice guidelines, recommendations for LBP treatment are based on self-management, physical and psychological therapies (Foster Nadine, 2018). In addition, the routine use of imaging, functional and physical measurements is not recommended (Foster Nadine,

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2018). On the other hand, reliable relevant differences have been identified between subgroups of acute and chronic LBP patients and healthy controls in physical aspects in the lumbar spine by the use of imaging and investigation (e.g. spine range of motion, muscle function and morphology). (Anders et al., 2005; Kiesel et al., 2007a; Hebert et al., 2015; Larivière et al., 2002; Goubert et al., 2016; Williams et al., 2013).

One of the most frequently studied muscles in LBP patients is the lumbar multifidus (LM). The LM is one of the muscles that contributes to the stability of the lumbar spine (MacDonald et al., 2009; Kiesel et al., 2008; Beneck and Kulig, 2012). In a subgroup of patients, LBP may be associated with dysfunction in active stabilization of the lumbar spine responsible for the transition of acute to chronic LBP, however, it is unclear how to identify this subgroup (Rosatelli Alessandro, 2008). Clinical studies concluded that stabilization is more effective than functional training in acute LBP patients (Hides et al., 2008; Belavy et al., 2010; MacDonald et al., 2006), especially, since stabilization therapy would prevent a decrease in LM cross-sectional area (i.e. atrophy) after prolonged bed rest (Belavy et al., 2010). This reduction in LM-diameter has also been observed in patients with chronic LBP (MacDonald et al., 2009; Hides et al., 1996; Danneels et al., 2002), however, it remains unclear if it was cause or result of the chronic LBP. Studies that found associations between LM dysfunction and LBP were mainly performed in a laboratory setting in small homogeneous populations, which complicates the generalizability of the results into clinical practice (Williams et al., 2013; Kiesel et al., 2012; Anders et al., 2007).

In order to develop better treatment approaches, it is necessary to know which of these LM muscle parameters (function and morphology) are relevant for routine care and if they are applicable for subacute and/or chronic LBP patients. Therefore, the need for studies that investigate the contribution of changes in LM function and/or morphology in acute, subacute and chronic subgroups of patients with LBP in real world situations with larger sample sizes is high. Knowledge from these studies can contribute to the identification of clinically relevant subgroups that need specific treatment, thereby increasing the efficacy of LBP treatments (Hebert et al., 2008; Hides et al., 2017).

The aim of the present study is to compare clinical characteristics, in terms of low back function and LM morphology, between subacute and chronic LBP patients from a large clinical practice cohort with healthy controls.

2. Method

2.1. Design

A multicenter cross-sectional case control design is used. The current study is registered at the Dutch Trial Register (NTR6331). The study was approved by the Medical Ethics Committee Twente, Enschede in the Netherlands (09-03-2017, NL60064.044.16).

2.2. Participants

Patients were recruited from the Spine Network in the Twente region in the Netherlands between March 2017 until May 2018. About 120 physiotherapists participate in this network. LBP (non-specific low back pain) patients between 18 and 65 years of age were included. Exclusion criteria were the presence of possible serious pathology that required referral to medical specialists, lumbosacral radicular syndrome, pregnancy, previous back surgery, current psychiatric diagnosis, insufficient knowledge of the Dutch language or a body mass index (BMI) > 30 (Nordander et al., 2003). Patients were stratified into subacute LBP (sLBP <3 months duration) or chronic LBP (cLBP >3 months duration) (Steenstra et al., 2017; Frank et al., 1996). Healthy controls between 18 and 65 years of age, with no history of LBP (in the previous 6 months) (Yodchaisarn et al., 2018; Griffin et al., 2012) were recruited through social networks in the Eastern part of the Netherlands. Exclusion criteria for these healthy controls were similar to those for LBP patients. Participants were informed about the purpose and protocol of the study before they were asked to sign informed consent. Priori we could not determine an estimated effect size, therefore it was chosen to use a generic calculation for sample size following the procedures of Bridges and Holler (2007), leading to 50 participants per group (Bridges and Holler, 2007).

2.3. Procedure

Patients, who were seen in primary care by one of the 120 therapists for the first time, were invited by one of these therapists to participate in the study. If they were willing to participate and met the inclusion criteria, they were provided with an information letter and informed consent form. When LBP patients signed informed consent, they were forwarded to one of the four physiotherapy practices where physiotherapists were trained for this study protocol (referred to as diagnostic centers). The physiotherapist received 20 h of training to perform the clinical tests with the use of the technologies ultrasound, 3D sensor and surface EMG (sEMG). Patients received regular treatment from their therapist after inclusion and study measurements. The healthy controls were recruited via open source and signed informed consent before participation. Healthy controls were assessed according to the inclusion and exclusion criteria at one of four diagnostic centers. All involved physiotherapists at the diagnostic centers were qualified to make ultrasound images, and they were trained in the protocol in four half-day sessions. The procedure is shown in a flowchart in the result section. All the measurements were administered in both LBP patient groups and the healthy control group (HCG). Prior to testing, after verbal instructions, the participant practiced every test once to validate the protocol. Then the examiner gave the starting signal for the movement which was recorded.

2.4. Measurements

The test protocol consisted of LM muscle function (sEMG) and morphology tests (ultrasound), and low back function tests administrated by means of 3D kinematic.

2.5. Primary outcomes

2.5.1. Surface electromyography

The sEMG protocol was executed with the sEMG system Mobita® 32-channel and analyzed with the software TMSi Polybench software (Twente Medical Systems International B.V., Oldenzaal, the Netherlands) to assess muscular electrical LM activity. The Mobita® 32-channel is validated by Askamp and van Putten (Askamp and van Putten, 1872). Surface electrodes were attached to the skin after the skin was shaved and cleaned (alcohol 70%) and were used to measure bipolar sEMG (Ag/AgCL Kendall H124SG ECG electrodes (24 mm), MedCat B.V at Klazienaveen in The Netherlands). Pairs of surface electrodes were attached to the skin at LM muscle parallel to the muscle fibres, according to the Seniam method (Hermens et al., 2000). A ground electrode was placed over the ilium. The electrodes were bilaterally attached to the skin of the participants (Hermens et al., 2000). The muscle activity of LM was measured during the Biering Sorensen test to assess isometric endurance as measure of LM muscle function (Demoulin et al., 2006). During this test, the participant lays in a prone position with only the lower body strapped on the bench with bands. The participant had to maintain a horizontal position without the support of the upper body from the bench for 60 s for practical reasons to keep the test program short. Also, patients with high risk of complaints were previously determined to endure the test shorter than 58 s (Alaranta et al., 1995). Therefore, a maximum of 60 s for this test was applied.

sEMG signals were recorded with a sample frequency of 2000 Hz and pre-processed with a high pass filter of 20 Hz. The data were exported for analysis with Matlab (version R2018a). First, a bandpass filter (2nd order Butterworth filter) with edges 20 and 500 Hz was applied to the raw data. Second, the signal was rectified by calculating the absolute value of each data point. Finally, results of first 5 s and the last 5 s of the test were calculated for the final data analysis. The median frequency was calculated to indicate fatigue and the average root mean square (RMS) was calculated to indicate the intensity/level of contraction by using the sEMG data (Plamondon et al., 2004; Roy et al., 1989; De Luca, 1997). For median frequency and RMS, the delta was calculated by subtracting the first 5 s of the Biering Sorensen test from the last 5 s.

2.6. *Ultrasound*

Ultrasound was used to assess LM morphology. The following ultrasound equipment was used in the diagnostic centers: Terason SMART3200T (Terason, a Division of Teratech Corporation in Burlington at USA), Philips CX30 (Philips Medical Systems Nederland B.V. at Best in the Netherlands), ALPINION ecube 7 (Alpinion Medical Systems at Seoul in Korea), Mindray M7 (Shenzhen Mindray Bio-Medical Electronics Co., Ltd. at Nanshan in China) and the Echomaster 200 (TELEMED UAB at Vilnius in Lithuania)). Separate images of the left and right LM were obtained in two conditions (in rest and submaximal contraction; four images in total per participant) (Kiesel et al., 2007b). The participants lay in prone position with a pillow beneath their abdomen (lower side of the pillow positioned to anterior superior iliac spine) to minimize lumbar lordosis. The left and right contralateral arm lift test was performed to achieve a standardized submaximal contraction of the LM (Kiesel et al., 2007b). The participant was asked to hold each position for 15 s (Kiesel et al., 2007a; Hebert et al., 2015).

The examiner palpated caudally to identify the superior iliac posterior spine (SIPS), L5 and S1 spinal levels. First, the probe was placed with gel longitudinally along the spine to identify the spinous process of L5 and S1. Second, the probe was turned horizontally to the spine at L5-S1 level. Third, the probe was moved laterally and stopped when SIPS was identified as anatomical landmark. Fourth, the probe was turned over in the transversal plane to create an angle (between probe and low back) that resulted in an optimal image of LM at the level L5-S1 with the anatomical landmarks SIPS and lamina. LM thickness (cm) was measured in the area between the lamina of the vertebrae to the superficial border of the LM (Richardson, 1999; Hosseinifar et al., 2015), see Fig. 1A. Thickness of LM (cm) was measured by the software program on the ultrasound equipment with the on-screen cursor (Fig. 1).

2.7. *3D kinematics*

The Microgate Gyko (ProCare B.V, Groningen the Netherlands) with the Gyko RePower software (Microgate, Bolzano-Bozen Italy) was used to measure the range of motion (ROM) in degrees (°) of the lumbar spine. The Gyko is an inertial measurement tool and was secured with an elastic belt. The elastic belt was placed on the bare trunk around the back and abdomen of a participant with the middle of the Gyko at the thoracolumbar junction at the back (Th12-L1), see Fig. 2. The ROM was measured during the following tests: trunk flexion and extension, and left and right lateral flexion. During these tests, the participant was asked to stand upright in a relaxed position, with feet at shoulder-width apart, knees bent in standard position of 10° flexion, and arms hanging relaxed by the side (Hamersma et al., 2019). Maximal trunk flexion and trunk extension were performed. For flexion, participants were instructed to bend their spine as far as possible and not their knees. For extension, participants extended their spine as far as possible, while keeping their hip in a neutral standing position (hip and pelvic movement were minimalized) (Hamersma et al., 2019). Furthermore, maximal lateral flexion left and right were performed. The Gyko has shown good reliability and concurrent validity for the measurement of ROM (Hamersma et al., 2019).

2.8. *Secondary outcomes*

Secondary outcomes were personal characteristics, body mass index, pain intensity with a numeric rating scale for pain (NRS) and the Oswestry Disability Index (ODI) (Hjermstad et al., 2011; van Hooff et al., 2015). The NRS is an 11-point rating scale for pain in which 0 is no pain and 10 is worst pain imaginable. Participants were asked to rate their average pain at the current day. The interpretation of NRS is as follows: 0 means no pain and 10 means maximum pain. The ODI is a disease-specific measure for patients with LBP and it ranges from 0 to 100. The ODI has five categories in end scores: 0–20% minimal limitations; 21–40% moderate limitations; 41–60% obvious limitations; 61–80% most limitation; 81–100% bedridden patients.

2.9. *Statistical analysis*

All analyses were performed between both LBP groups and the HCG. Five missing ultrasound data points were imputed by using the Monte Carlo Markov chain (MCMC) method, with a linear regression model. Five imputed datasets were created. Normality of the data was visually inspected using histograms. Potential confounding variables (gender,

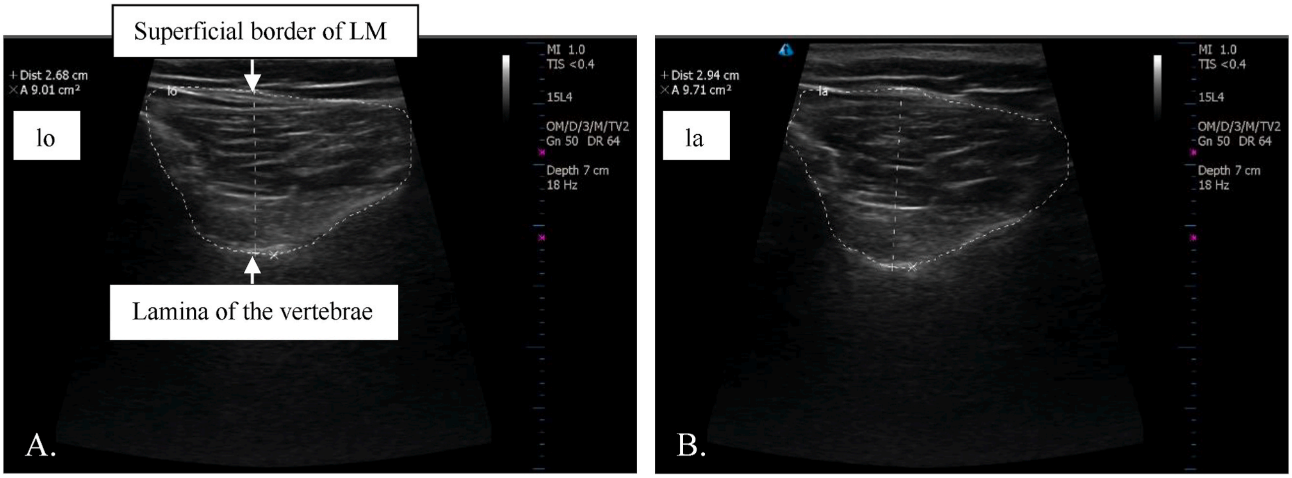

Fig. 1. Example of ultrasound images where the thickness of the left lumbar multifidus (LM) is calculated at L5-S1, while lying in prone position. A. Left lumbar multifidus in relaxed condition, which is marked with “lo”. B. Left lumbar multifidus in submaximal contraction, which is marked with “la”. The thickness in centimeters is presented next to “+Dist.” at the left side in the black column of Figure A and B.

Fig. 2. Placement of Gyko at thoracolumbar junction.

weight and age) were analyzed with linear regression for their relationship with the primary outcomes. Parametric analyses were performed with One-way ANOVA and pairwise comparisons with Tukey's HSD post-hoc test. The independent Samples *t*-test, was performed for the questionnaire data (NRS for pain and ODI) to compare differences between sLBP and cLBP group. An alpha of 0.05 was used for all tests, except from the multiple post-hoc comparisons using IBM SPSS Statistics 24.

3. Results

3.1. Participants

A total of 178 participants were referred to the diagnostic centers. Of these, 17 did not meet the inclusion criteria, because of BMI >30 (n = 14), age > 65 year (n = 1), actual psychiatric diagnosis (n = 1), or unknown duration of LBP (n = 1). Of the 161 finally included participants, 50 were healthy controls, 59 had cLBP and 52 had sLBP (see Fig. 3).

Overall, HCG had a statistically significant lower body weight in kilograms (HCG: 72.5 ± 10.6; sLBP: 81.3 ± 11.7; cLBP: 78.4 ± 12.1) compared to both LBP groups. Between the LBP groups, there was no statistically significant difference in pain intensity, however, the cLBP group had statistically significant higher disability scores compared to the sLBP group (ODI score: sLBP: 16 ± 12; cLBP: 22 ± 15; p = 0.03). Statistically significant differences between groups were found for all participant characteristic outcomes, except for body height. Analyses of the relation of potential confounders with primary outcomes showed little impact of gender, age and weight on the primary outcomes in all groups. The participant's characteristics for all participants are shown in Table 1.

3.2. Trunk ROM and LM thickness

The trunk ROM and LM thickness were significantly larger in all directions and conditions in HCG compared to all participants with LBP, except for ROM in lateral flexion right and LM thickness in relaxed condition right. The largest significant differences in LM thickness were found between HCG and cLBP. Reduction in LM thickness was observed between groups; the LM thickness was lower in sLBP and cLBP compared to HCG, and the LM thickness was lower in cLBP compared to sLBP. But Post Hoc Tests revealed that this reduction was only significant between the HCG and both LBP groups. Table 2 shows the trunk ROM (°) and LM thickness (cm) data.

3.3. Surface EMG

sEMG results from the Biering Sorensen test were presented in Table 3 (median frequencies and RMS values). The analysis was completed for 130 participants because 21 participants did not reach 60 s (because of pain) and of the other 10 participants, the sEMG data was not completed. The missing data per group was 1/50 participant in the HCG group, 13/52 in the sLBP group, and 17/59 in the cLBP group. There were no statistically significant baseline differences between groups (p = 0.10–0.32) (Table 3). Also, no statistically significant differences were found in the delta results of median frequency and RMS between all groups. The % change data showed no any substantial differences compared to the delta values in each variable of the median frequency and RMS. Notably, the standard deviations are high in the RMS data and the delta data of the median frequency in all groups, which means that the data is very heterogeneous.

4. Discussion

The current study aimed to compare clinical characteristics in terms of low back function and LM morphology, between subacute LBP patients, chronic LBP patients and healthy controls.

LBP patients had a significantly less trunk ROM and LM thickness compared with healthy controls. There is a trend in lower LM thickness between subacute and chronic LBP patients, however, this was non-significant. Patients with cLBP experienced larger functional impairments (higher score on the ODI questionnaire) than the patients with sLBP. Furthermore, no significant differences were found between the three groups in the sEMG data (median frequency and RMS values).

Overall in this study, both *LBP groups* show less ROM in different trunk movements than healthy controls. In detail, *LBP patients* had 15° less in ROM compared with healthy controls in trunk flexion. In trunk extension and trunk lateral flexion, *LBP patients* had 5° in ROM less compared to healthy controls. Mazzone et al. (2016) support our results about LBP patients who displayed reduction in lumbar motion than healthy controls during trunk extension (Mazzone et al., 2016). In our results no differences were found in the intensity of pain and in trunk movements between subacute LBP and chronic LBP patients.

The results of the LM thickness measures show that healthy controls had a significantly thicker LM muscle (approximately 1 cm ≈ 30%) compared with *LBP patients* at the level of L5-S1, except for LM thickness in relaxed condition right. This condition showed the same trend as the others that are were statistically significant between healthy controls and *LBP patients*, however with a slightly smaller difference leading to non-significant differences. The literature confirms that LBP patients

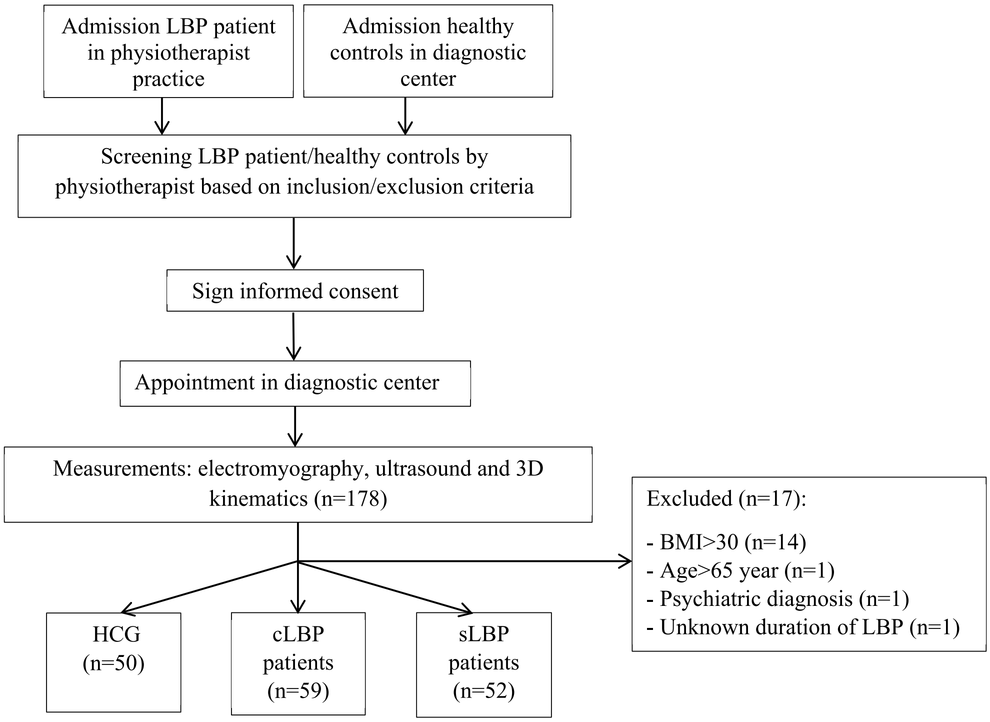

Fig. 3. Flowchart of the study procedure. Included participants are divided into healthy controls group (HCG), chronic non-specific low back pain (cLBP) group and subacute non-specific low back pain (sLBP) group.

Table 1
Participants characteristics (n = 161).

	HCG (n = 50)	sLBP (n = 52)	cLBP(n = 59)	all (n = 161)	p-value	Group comparison		Post Hoc p-value.
Gender (n) [a]					**0.03**	HCG	sLBP	0.05[d]
Male	19 (38%)	32 (62%)	24 (41%)	75 (47%)			cLBP	0.78[d]
Female	31 (62%)	20 (38%)	35 (59%)	86 (53%)		sLBP	cLBP	0.06[d]
Age (years) [b]					**0.04**	HCG	sLBP	0.08
Mean (SD)	38 (17)	44 (13)	44 (12)	42 (14)			cLBP	0.07
						sLBP	cLBP	1.00
Weight (kg) [b]					**<0.01**	HCG	sLBP	**<0.01**
Mean (SD)	72.5 (10.6)	81.3 (11.7)	78.4 (12.1)	77.5 (12.0)			cLBP	**0.02**
						sLBP	cLBP	0.39
Height (cm) [b]					0.14	HCG	sLBP	n.a.
Mean (SD)	176.2 (9.3)	179.4 (9.8)	176.3 (9.6)	177.3 (9.6)			cLBP	n.a.
						sLBP	cLBP	n.a.
BMI (kg/m^2) [b]					**<0.01**	HCG	sLBP	**<0.01**
Mean (SD)	23.3 (2.1)	25.2 (2.6)	25.2 (3.1)	24.6 (2.8)			cLBP	**<0.01**
						sLBP	cLBP	1.00
NRS [c]	–	n = 46	n = 54	n = 100	0.21	HCG	sLBP	n.a.
Mean (SD)		5 (2)	6 (2)	5 (2)			cLBP	n.a.
						sLBP	cLBP	n.a.
ODI [c]	–	n = 48	n = 54	n = 102	**0.03**	HCG	sLBP	n.a.
Mean (SD)		16 (12)	22 (15)	18 (14)			cLBP	n.a.
						sLBP	cLBP	n.a.

Abbreviations: HCG = healthy controls; sLBP = subacute non-specific low back pain patients; cLBP = chronic non-specific low back pain patients; kg = kilogram; cm = centimeters; BMI = body mass index; m^2 = square meter; NRS = Numeric Rating Scale for pain; ODI = Oswestry Disability Index; SD = Standard Deviation; n.a. = not applicable.

[a] Chi^2 test.

[b] .One-way ANOVA, Post Hoc Test Tukey HSD.

[c] Independent Samples *t*-test, Sig. (2-tailed).

[d] Statistical significant with Holm-Bonferroni correction.

(most literature included cLBP patients) suffer from LM atrophy or less LM thickness compared to healthy people in both conditions (rest and submaximal contraction) (Kader et al., 2000; Danneels et al., 2000; Lee et al., 2006; Djordjevic et al., 2014). An explanation for our results could be that, with LBP, there is disuse of LM within 12 weeks, which, consequently, leads to a lower LM thickness. In our results, the largest decrease in LM thickness was seen in the first 12 weeks of having LBP (differences between healthy controls and subacute LBP patients). This phenomenon of developing atrophy of LM muscle in the first period of a LBP is supported by other studies (Hides et al., 1994; Hodges et al., 2006). After 12 weeks of having LBP, a lower LM thickness was shown in the results between sLBP and cLBP patients. This is supported by earlier work, that concluded that compromised structure of low back muscles could plausibly increase the risk for further LBP (Hodges and Danneels,

Table 2

Results of trunk ROM and LM thickness in different groups (n = 161).

		HCG	sLBP	cLBP	p-value[a]	Group comparisons		Post Hoc p-value[a]
ROM – Trunk flexion (°)	Mean (SD)	103.7 (16.6)	88.2 (20.9)	90.9 (20.3)	<0.01	HCG	sLBP	**<0.01**
							cLBP	**<0.01**
						sLBP	cLBP	0.75
ROM – Trunk extension (°)	Mean (SD)	31.8 (10.5)	25.7 (9.7)	24.2 (9.2)	<0.01	HCG	sLBP	**<0.01**
							cLBP	**<0.01**
						sLBP	cLBP	0.70
ROM – Lateral flexion R. (°)	Mean (SD)	25.0 (5.0)	22.3 (6.0)	21.0 (8.4)	<0.01	HCG	sLBP	0.10
							cLBP	**<0.01**
						sLBP	cLBP	0.57
ROM – Lateral flexion L. (°)	Mean (SD)	26.8 (7.3)	21.7 (6.9)	22.0 (8.5)	<0.01	HCG	sLBP	**<0.01**
							cLBP	**<0.01**
						sLBP	cLBP	0.97
LM thickness – Relax L. (cm)	Mean (SD)	3.0 (1.0)	2.4 (0.9)	2.1 (0.7)	<0.01	HCG	sLBP	**0.01**
							cLBP	**<0.01**
						sLBP	cLBP	0.13
LM thickness –Contr. L. (cm)	Mean (SD)	3.4 (1.0)	2.7 (0.9)	2.4 (0.9)	<0.01	HCG	sLBP	**<0.01**
							cLBP	**<0.01**
						sLBP	cLBP	0.19
LM thickness –Relax R. (cm)	Mean (SD)	2.8 (0.9)	2.4 (0.9)	2.2 (0.9)	<0.01	HCG	sLBP	0.11
							cLBP	**<0.01**
						sLBP	cLBP	0.34
LM thickness –Contr. R. (cm)	Mean (SD)	3.2 (0.9)	2.6 (1.0)	2.4 (0.9)	<0.01	HCG	sLBP	**0.02**
							cLBP	**<0.01**
						sLBP	cLBP	0.49

Abbreviations: HCG = healthy controls; sLBP = subacute non-specific low back pain patients; cLBP = chronic non-specific low back pain patients; ROM = Range of Motion; LM = lumbar multifidus; relax = relax condition; contr. = submaximal contraction condition; R. = Right; L. = Left; SD = Standard Deviation; cm = centimeters; ° = degrees.

[a] One-way ANOVA, with df = 2, Post Hoc Test Tukey HSD.

Table 3

Results of EMG during Biering Sorensen Test in median frequency (Hz) and Root Mean Square (μV).

Muscle		Median Frequency (Hz)					Root mean square (μV)				
		HCG n = 49	sLBP n = 39	cLBP n = 42	All n = 130	p-value[a]	HCG n = 49	sLBP n = 39	cLBP n = 42	All n = 130	p-value[a]
		First 5 s									
LM – L	Mean (SD)	103 (18)	98(20)	102 (19)	101 (19)	0.32	74(39)	63 (33)	62(45)	66 (40)	0.27
LM – R	Mean (SD)	103 (20)	96(17)	103 (19)	101(19)	0.17	79 (40)	62 (30)	62 (54)	68 (43)	0.10
		Delta (last 5 s – first 5 s)									
LM – L	Mean (SD)	−16 (10)	−15 (10)	−15 (10)	−15 (10)	0.88	−2 (10)	−1 (13)	−1 (8)	−1 (11)	0.93
LM – R	Mean (SD)	−15 (12)	−15(9)	−15 (11)	−15 (11)	0.98	−2 (14)	−1 (12)	0 (10)	−1 (12)	0.78
		% change[b]									
LM – L	%	−16	−15	−15	−15		−3	−2	−2	−2	
LM – R	%	−15	−16	−15	−15		−3	−2	0	−1	

Abbreviations: HCG = healthy controls; sLBP = subacute non-specific low back pain patients; cLBP = chronic non-specific low back pain patients; LM = lumbar multifidus; R. = Right; L. = Left; SD = Standard Deviation.

[a] One-Way ANOVA with df = 2.

[b] % change ((Mean last 5 s – Mean first 5 s)/Mean first 5 s) * 100%.

2019). Whether the differences between *LBP patients* and healthy controls that were found are clinically significant is unclear, LM atrophy can also be developed by a range of biological and/or psychosocial influences (Beneck and Kulig, 2012; Hodges and Danneels, 2019; Hodges and Moseley, 2003).

In our sEMG measurements, median frequency and RMS data was used as an indicator of muscle fatigue and level of muscle activation in LM respectively (Plamondon et al., 2004). Between all groups, no differences were found in muscle fatigue and muscle activity during the Biering Sorensen test. An explanation for this could be that this test asks for a static contraction, a combined effort of low back extensors, less than a form of spine stability in which LM plays a major role (MacDonald et al., 2009). LM activity may have been compensated by activation of surrounding musculature in less-affected regions (Le Cara et al., 2014).

Interestingly, sEMG measurements show no differences between healthy controls and *LBP patients*, but statistically significant differences between these groups are found in trunk ROM and LM thickness. Our sEMG variables showed large standard deviations, indicating large inter subject heterogeneity, making it hard to show differences. From this cross-sectional study, it cannot be determined that decreased function and morphology are causal to our result of LBP, however, with these large cohorts, trends were observed that LM thickness is lower in sLBP patients compare to healthy controls and that sLBP patients have lower LM thickness compared to cLBP patients.

4.1. *Clinical implications*

This research is performed in a clinical setting, which means that most testers were physiotherapists and performed the tests in their physiotherapy practice. All tests were clinical tests, which are often used in physiotherapy practice. Therefore, this research design improves the generalization of our results to other clinical practices. For example, the trunk ROM measured by the Gyko is an application that can be useful in clinical practices. Thereby, no other study showed data with statistical analysis of comparisons between subacute LBP, chronic LBP patient groups and healthy controls on LM morphology and low back function in this clinical setting and with this number of participants. These results may be a first step to development of new clinical prediction rules which are based on function and morphology, and are thereby less subjective compared to other rule based algorithms.

4.2. Limitations

There were significant differences between the groups (healthy controls, subacute LBP and chronic LBP patients) in participants' characteristics at many variables. For example, the ratio of male/female, the sLBP group had more male participants (62%), compared with the healthy controls (38% male) and cLBP group (41% male). Healthy controls had significantly lower body weight compared with *LBP patients*. Our pre-analysis shows that there was limited impact of confounding variables as gender, age and weight. However, LM thickness right in relaxed condition in healthy controls had a R^2 of 0.15 with weight. Healthy controls had a statistically significant lower body weight, but a higher LM thickness in all conditions compared with *LBP patients* (except for in relaxed condition right), therefore we might assume that a possible bias would lead to an underestimation of the true differences between healthy controls and patients. The healthy controls had no history of LBP in the previous 6 months, which is an arbitrary limit we made. However, some literature proved that even if the back pain resolves 9–12 months before recruitment then there still may be morphological changes in the muscle (Hides et al., 1996). A weakness of the study could be that the exact duration of LBP is unknown, only more or less than 12 weeks of pain. Therefore the results of the sLBP group (0–12 weeks LBP) are difficult to interpret. A recommendation for further research is to measure the exact duration of pain in weeks in such a patient group, as far as this is possible.

Our protocol of the Biering Sorensen test had a maximum duration of 60 s, because of practical reasons to minimize the duration and charge of our test protocol to the participants. If there was chosen for a maximum duration as long as possible until the participant stopped the Biering Sorensen test, maybe more and/or larger differences would have been identified in muscle fatigue and muscle activity between HCG and *LBP patients* and between subacute LBP and chronic LBP patients (Demoulin et al., 2006). On the other hand, to compare sEMG data with different durations, a correction for different time durations has to be made which goes with other limitations.

5. Conclusion

Trunk ROM and LM thickness show differences between LBP patients and healthy controls. LBP patients and healthy controls. LM function, expressed in sEMG values as RMS and median frequency, presented no differences between LBP patients and healthy controls. Pain intensity showed no significant differences between subacute and chronic LBP patients. Chronic LBP patients showed a significant higher disability score compared to subacute LBP patients.

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References

Airaksinen, O., Brox, J.I., Cedraschi, C., et al., 2006. Chapter 4. European guidelines for the management of chronic nonspecific low back pain. Eur. Spine J. : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society 15 (Suppl. 2), S192–S300.

Alaranta, H., Luoto, S., Heliövaara, M., Hurri, H., 1995. Static back endurance and the risk of low-back pain. Clin. Biomech. 10 (6), 323–324.

Anders, C., Scholle, H.C., Wagner, H., Puta, C., Grassme, R., Petrovitch, A., 2005. Trunk muscle co-ordination during gait: relationship between muscle function and acute low back pain. Pathophysiology 12 (4), 243–247.

Anders, C., Brose, G., Hofmann, G.O., Scholle, H.C., 2007. Gender specific activation patterns of trunk muscles during whole body tilt. Eur. J. Appl. Physiol. 101 (2), 195–205.

Askamp J, van Putten MJ. Mobile EEG in Epilepsy. (1872-7697 (Electronic)).

Belavy, D.L., Armbrecht, G., Gast, U., Richardson, C.A., Hides, J.A., Felsenberg, D., 2010. Countermeasures against lumbar spine deconditioning in prolonged bed rest: resistive exercise with and without whole body vibration. J. Appl. Physiol. 109 (6), 1801–1811.

Beneck, G.J., Kulig, K., 2012. Multifidus atrophy is localized and bilateral in active persons with chronic unilateral low back pain. Arch. Phys. Med. Rehabil. 93 (2), 300–306.

Bridges, A.J., Holler, K.A., 2007. How many is enough? Determining optimal sample sizes for normative studies in pediatric neuropsychology. Child Neuropsychol. : a journal on normal and abnormal development in childhood and adolescence 13 (6), 528–538.

Le Cara, E.C., Marcus, R.L., Dempsey, A.R., Hoffman, M.D., Hebert, J.J., 2014. Morphology versus function: the relationship between lumbar multifidus intramuscular adipose tissue and muscle function among patients with low back pain. Arch. Phys. Med. Rehabil. 95 (10), 1846–1852.

Danneels, L.A., Vanderstraeten, G.G., Cambier, D.C., Witvrouw, E.E., De Cuyper, H.J., 2000. CT imaging of trunk muscles in chronic low back pain patients and healthy control subjects. Eur. Spine J. 9 (4), 266–272.

Danneels, L.A., Coorevits, P.L., Cools, A.M., et al., 2002. Differences in electromyographic activity in the multifidus muscle and the iliocostalis lumborum between healthy subjects and patients with sub-acute and chronic low back pain. Eur. Spine J. 11 (1), 13–19.

Demoulin, C., Vanderthommen, M., Duysens, C., Crielaard, J.M., 2006. Spinal muscle evaluation using the Sorensen test: a critical appraisal of the literature. Joint Bone Spine 73 (1), 43–50.

Djordjevic, O., Djordjevic, A., Konstantinovic, L., 2014. Interrater and intrarater reliability of transverse abdominal and lumbar multifidus muscle thickness in subjects with and without low back pain. J. Orthop. Sports Phys. Ther. 44 (12), 979–988.

Foster Nadine, E.N., 2018. Prevention and treatment of low back pain: evidence, challenges, and promising directions. Lancet, The. 391 (10137), 2368–2383.

Frank, J.W., Brooker, A.S., DeMaio, S.E., et al., 1996. Disability resulting from occupational low back pain. Part II: what do we know about secondary prevention? A review of the scientific evidence on prevention after disability begins. Spine 21 (24), 2918–2929.

Goubert, D., Oosterwijck, J.V., Meeus, M., Danneels, L., 2016. Structural changes of lumbar muscles in non-specific low back pain: a systematic review. Pain Physician 19 (7), E985–E1000.

Griffin, D.W., Harmon, D.C., Kennedy, N.M., 2012. Do patients with chronic low back pain have an altered level and/or pattern of physical activity compared to healthy individuals? A systematic review of the literature. Physiotherapy 98 (1), 13–23.

Hamersma, D.T., Hofste, A., Rijken, N.H.M., Roe Of Rohe, M., Oosterveld, F.G.J., Soer, R., 2019. Reliability and Validity of the Microgate Gyko for Measuring Range of Motion of the Low Back. Musculoskeletal science & practice, p. 102091.

Hebert, J., Koppenhaver, S., Fritz, J., Parent, E., 2008. Clinical prediction for success of interventions for managing low back pain. Clin. Sports Med. 27 (3), 463–479.

Hebert, J.J., Koppenhaver, S.L., Teyhen, D.S., Walker, B.F., Fritz, J.M., 2015. The evaluation of lumbar multifidus muscle function via palpation: reliability and validity of a new clinical test. Spine J. 15 (6), 1196–1202.

Hermens, H.J., Freriks, B., Disselhorst-Klug, C., Rau, G., 2000. Development of recommendations for SEMG sensors and sensor placement procedures. J. Electromyogr. Kinesiol. 10 (5), 361–374.

Hides, J.A., Stokes, M.J., Saide, M., Jull, G.A., Cooper, D.H., 1994. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. Spine 19 (2), 165–172.

Hides, J.A., Richardson, C.A., Jull, G.A., 1996. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. Spine 21 (23), 2763–2769.

Hides, J., Stanton, W., McMahon, S., Sims, K., Richardson, C., 2008. Effect of stabilization training on multifidus muscle cross-sectional area among young elite cricketers with low back pain. J. Orthop. Sports Phys. Ther. 38 (3), 101–108.

Hides, J.A., Walsh, J.C., Smith, M.M.F., Mendis, M.D., 2017. Self-managed exercises, fitness and strength training, and multifidus muscle size in elite footballers. J. Athl. Train. 52 (7), 649–655.

Hjermstad, M.J., Fayers, Pm Fau, Haugen, D.F., Haugen, Df Fau, Caraceni, A., et al., 2011. Studies Comparing Numerical Rating Scales, Verbal Rating Scales, and Visual Analogue Scales for Assessment of Pain Intensity in Adults: a Systematic Literature Review (1873-6513 (Electronic)).

Hodges, P.W., Danneels, L., 2019. Changes in structure and function of the back muscles in low back pain: different time points, observations, and mechanisms. J. Orthop. Sports Phys. Ther. 49 (6), 464–476.

Hodges, P.W., Moseley, G.L., 2003. Pain and motor control of the lumbopelvic region: effect and possible mechanisms. J. Electromyogr. Kinesiol. 13 (4), 361–370.

Hodges, P., Holm, A.K., Hansson, T., Holm, S., 2006. Rapid atrophy of the lumbar multifidus follows experimental disc or nerve root injury. Spine 31 (25), 2926–2933.

van Hooff, M.L., Spruit, M Fau, Fairbank, J.C.T., Fairbank, Jc Fau, van Limbeek, J., van Limbeek, J Fau, Jacobs, W.C.H., Jacobs, W.C., 2015. The Oswestry Disability Index (Version 2.1a): Validation of a Dutch Language Version (1528-1159 (Electronic)).

Hosseinifar, M., Akbari, A., Ghiasi, F., 2015. Intra-rater reliability of rehabilitative ultrasound imaging for multifidus muscles thickness and cross section area in healthy subjects. Global J. Health Sci. 7 (6), 354–361.

Kader, D.F., Wardlaw, D., Smith, F.W., 2000. Correlation between the MRI changes in the lumbar multifidus muscles and leg pain. Clin. Radiol. 55 (2), 145–149.

Kiesel, K.B., Uhl, T.L., Underwood, F.B., Rodd, D.W., Nitz, A.J., 2007a. Measurement of lumbar multifidus muscle contraction with rehabilitative ultrasound imaging. Man. Ther. 12 (2), 161–166.

Kiesel, K.B., Uhl, T.L., Underwood, F.B., Rodd, D.W., Nitz, A.J., 2007b. Measurement of lumbar multifidus muscle contraction with rehabilitative ultrasound imaging. Man. Ther. 12 (2), 161–166.

Kiesel, K.B., Uhl, T., Underwood, F.B., Nitz, A.J., 2008. Rehabilitative ultrasound measurement of select trunk muscle activation during induced pain. Man. Ther. 13 (2), 132–138.

Kiesel, K.B., Butler, R.J., Duckworth, A., et al., 2012. Experimentally induced pain alters the EMG activity of the lumbar multifidus in asymptomatic subjects. Man. Ther. 17 (3), 236–240.

Kuijer, W., Brouwer, S., Preuper, H.R.S., Groothoff, J.W., Geertzen, J.H.B., Dijkstra, P.U., 2006. Work status and chronic low back pain: exploring the international classification of functioning, disability and health. Disabil. Rehabil. 28 (6), 379–388.

Larivière, C., Gagnon, D., Loisel, P., 2002. A biomechanical comparison of lifting techniques between subjects with and without chronic low back pain during freestyle lifting and lowering tasks. Clin. BioMech. 17 (2), 89–98.

Lee, S.W., Chan, C.K.M., Lam, T.S., et al., 2006. Relationship between low back pain and lumbar multifidus size at different postures. Spine 31 (19), 2258–2262.

De Luca, C.J., 1997. The use of surface electromyography in biomechanics. J. Appl. Biomech. 13 (2), 135–163.

MacDonald, D.A., Lorimer Moseley, G., Hodges, P.W., 2006. The lumbar multifidus: does the evidence support clinical beliefs? Man. Ther. 11 (4), 254–263.

MacDonald, D., Moseley, G.L., Hodges, P.W., 2009. Why do some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. Pain 142 (3), 183–188.

Mazzone, B., Wood, R., Gombatto, S., 2016. Spine kinematics during prone extension in people with and without low back pain and among classification-specific low back pain subgroups. J. Orthop. Sports Phys. Ther. 46 (7), 571–579.

Nordander, C., Willner, J., Hansson, G.A., et al., 2003. Influence of the subcutaneous fat layer, as measured by ultrasound, skinfold calipers and BMI, on the EMG amplitude. Eur. J. Appl. Physiol. 89 (6), 514–519.

Plamondon, A., Trimble, K., Larivière, C., Desjardins, P., 2004. Back muscle fatigue during intermittent prone back extension exercise. Scand. J. Med. Sci. Sports 14 (4), 221–230.

Richardson, C., 1999. Therapeutic Exercise for Spinal Segmental Stabilization in Low Back Pain : Scientific Basis and Clinical Approach. Churchill Livingstone, Edinburgh; New York.

Rosatelli Alessandro, L.A., 2008. Three-dimensional study of the musculotendinous architecture of lumbar multifidus and its functional implications. Clin. Anat. 21 (6), 539–546.

Roy, S.H., De Luca, C.J., Casavant, D.A., 1989. Lumbar muscle fatigue and chronic lower back pain. Spine 14 (9), 992–1001.

Steele, J., Bruce-Low, S., Smith, D., Jessop, D., Osborne, N., 2014. Lumbar kinematic variability during gait in chronic low back pain and associations with pain, disability and isolated lumbar extension strength. Clin. Biomech. 29 (10), 1131–1138.

Steenstra, I.A., Munhall, C., Irvin, E., et al., 2017. Systematic review of prognostic factors for return to work in workers with sub acute and chronic low back pain. J. Occup. Rehabil. 27 (3), 369–381.

Vos, T., Flaxman, A.D., Naghavi, M., et al., 2012. Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 380 (9859), 2163–2196.

Williams, J.M., Haq, I., Lee, R.Y., 2013. An investigation into the onset, pattern, and effects of pain relief on lumbar extensor electromyography in people with acute and chronic low back pain. J. Manip. Physiol. Ther. 36 (2), 91–100.

Yodchaisarn, W., Puntumetakul, R., Emasithi, A., Boucaut, R., Chatchawan, U., 2018. Altered postural sway during quiet standing in women with clinical lumbar instability. J. Phys. Ther. Sci. 30 (8), 1099–1102.