Ultrasonography of multifidus muscle morphology and function in ice hockey players with and without low back pain

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Structured Abstract:

Objectives: To examine the relationship between lumbar multifidus (LM) morphology, function, echo-intensity (EI) and body composition among a group of university level ice hockey players with and without low back pain (LBP).

Design: Cross-sectional study

Setting: University Research Centre

Participants: Thirty-two hockey players (18 females, 14 males) participated in this study.

Main Outcome Measures: Resting LM cross-sectional area (CSA) was assessed bilaterally at the L5 level in prone and standing using ultrasound imaging. The LM thickness at rest and during contraction was evaluated in addition to LM EI. Body composition measures were acquired using dual-energy X-ray absorptiometry (DEXA) and LBP history was acquired using **a** self-reported questionnaire.

Results: LM muscle CSA was significantly associated with body composition measurements. LM EI was strongly associated with total % body fat and significantly greater in females. Resting LM muscle CSA and thickness (prone) was significantly smaller in players with LBP 4-weeks prior. LM side-to-side asymmetry (standing) was also significantly greater in players with LBP 3-months prior.

Conclusion: The results provide new insights with regards to LM morphology and activation in ice hockey players and revealed specific deficits in LM morphology in athletes with LBP. LM morphology was strongly associated with body composition measurements.

Key words: multifidus muscle; low back pain; ice hockey; ultrasound imaging; dual-energy X-ray absorptiometry

Introduction

Elite-level ice hockey players are exposed to high intensity training periods with high loading on the spine, pelvic region and lower limbs. Low back pain (LBP) is common in ice hockey players with reported prevalence varying between 55% and 89%,[3,12,48] and a 12-month incidence as high as 85%.[12] Spine abnormalities including disc signal reduction, disc height reduction, disc bulging, disc protrusion, disc extrusion and vertebral body endplate degenerative changes (e.g. modic changes) have been found to be common in hockey players.[3] LBP is often associated with sport-specific mechanical loads and injury patterns, especially in contact and combat sports, and athletes participating in sports with high loads on the spine were reported to have a higher incidence of LBP as compared to other athletes and non-athletes.[3,12,57] The most thoroughly investigated risk factors for the development of LBP in athletes are spinal load, anthropometrics, age, sex and previous history of LBP.[57] However, the influence of these factors on LBP in athletes remains uncertain. Importantly, LBP has been reported to affect trunk and lower limb kinematics, which may negatively impact sport performance.[38]

Biomechanical studies have demonstrated the critical role of the lumbar multifidus (LM) muscle to provide arthrokinetic control of the vertebral segment and spine stabilization,[25,64] as well as proprioception of the lumbar spine.[6] Imaging studies of both athletic and non-athletic populations with LBP have reported paraspinal muscle degenerative changes and functional deficits including LM muscle atrophy (decreased cross-sectional area, CSA),[4,11,19,22] LM CSA asymmetry (atrophy ipsilateral to the painful/pathological side),[4,28,43,44] increased fatty infiltration, [13,37,47,50] as well as increased or decreased muscular activity.[30,58,59] Elite cricketers with LBP were

reported to have localized LM muscle atrophy and side-to-side asymmetry.[22] Similar findings have also been reported in professional ballet dancers.[17] Decreased size and increased side-to-side asymmetry of the LM muscle have been found to be important predictors of lower-limb injuries in elite Australian Football League (AFL) players.[21] A recent study also suggested that runners with LBP have a decreased ability to contract the LM muscle.[7] However, other investigations reported no such LM morphological changes or functional deficits in elite athletes with LBP.[34,49,52] Interestingly, to date very few ultrasound-imaging studies have assessed LM muscle function in more functional positions, such as standing. Previous reports from non-athletic populations have reported an increase in LM CSA from prone lying to upright standing,[32,55] suggesting that a more accurate assessment of LM function may be performed while standing, when LM is contracted in a stabilizing role.[55]

Preseason-screening assessment of LM muscle characteristics using ultrasound imaging may be useful for the identification/prevention of athletes at risk of injuries and to monitor rehabilitation.[19,22] LM muscle CSA, thickness during submaximal contraction and at rest, and echo intensity (EI) in different postures can be conveniently measured with ultrasound to assess muscle size, function and quality, respectively. EI is measured using the ultrasound brightness scale, via a gray scale analysis, and may be used as an indicator of muscle quality by estimating intramuscular fat and connective tissue.[2,42] An increase in intramuscular fatty infiltration has been reported in people with chronic LBP,[27,37,47] and such change in muscle quality is hypothesized to increase the risk of injury and decrease overall muscle functionality.[23,31] Previous studies also reported that muscle EI is correlated with muscle strength and power.[8,16,35,36]

Evidence suggests that age, physical activity level and body composition (e.g. body mass index, BMI) influence paraspinal muscle morphology and quality.[10,14,15,47] BMI is the most commonly used variable to adjust for inter-subject anthropometric and body composition differences in this field. However, this measure remains a poor indicator of body composition especially in athletic populations, due to its inability to differentiate between muscle mass and fat mass. Dual-energy X-ray absorptiometry (DEXA) is the most accurate method to assess body composition. However, to our knowledge, there are no studies that have used this technology to examine the relationship between LM muscle characteristics and body composition. While body composition is recognized to influence muscle size and quality, the relationship between accurate measures of body composition, LM muscle characteristics and function in athletes with and without LBP deserves further attention.

Given the high incidence of LBP in ice hockey and evidence from previous related imaging studies, it seems imperative to examine LM muscle characteristics in this group of athletes. Therefore, the primary aim of this study was to examine the relationship between LM muscle morphology, function (prone and standing), EI and body composition in a cohort of male and female university level ice hockey players. A secondary aim was to compare LM muscle characteristics in university level ice hockey players with and without LBP. We hypothesized that players with LBP would demonstrate reduced LM muscle size and function (contraction) and greater side-to-side asymmetry.

Methods

Participants

Thirty-two ice hockey players (18 females, 14 males) from the Concordia University varsity team volunteered to participate in this study. Despite the different athletic demands related to different playing positions (e.g. forward, defense, goalie), all available players were invited to participate to maximize the sample size. The exclusion criteria were: previous history of severe trauma or spinal fracture, previous spinal surgery, observable spinal abnormalities and pregnancy. The study was approved by the Research Ethical Committee of the Institution and the Central Ethics Committee of Health and Social Services from the Ministry of Quebec. All players provided informed consent acknowledging that their data would be used for research purpose.

Procedures

During the preseason, each player participated in one testing session lasting approximately 30 minutes. All participants completed a self-administered questionnaire to collect information regarding players' demographics and history of LBP. LBP was defined as pain localized between T12 and the gluteal fold. Players were asked to answer "yes" or "no" to the presence of LBP during the past 4-weeks (pre-season) or 3-months (off-season) prior to the assessment. Players who answered "yes" to the presence of LBP completed a Visual Analogue Scale (VAS) to assess average LBP intensity, and were also asked about pain location (e.g. centered, right side, left side) and pain duration (in months) for both time points.

Ultrasound

Ultrasound B-mode images of LM muscle were acquired using a LOGIQ e ultrasound machine (GE Heathcare, Milwaukee,WI) with a 5-MHz curvilinear transducer. The imaging parameters were kept consistent in all acquisitions (frequency: 5MHz, gain: 60, depth: 8.0cm). Previous studies have established the reliability and validity of ultrasound imaging to assess LM muscle size and thickness, and determined that this imaging technique was repeatable, reliable and valid when performed by trained assessors. [29,51]

Prone lying measurements

To assess LM muscle size, participants were placed in a prone position, on a therapy table, with a pillow under their abdomen to minimize lumbar lordosis and instructed to relax the paraspinal musculature. The spinous process of L5 was palpated and marked on the skin with a pen prior to imaging. Acoustic coupling gel was applied to the skin and the ultrasound transducer was placed longitudinally along the midline of the lumbar spine to confirm the location L5. The transducer was then rotated and placed transversally over the L5 spinous process for imaging. Bilateral transverse images of LM muscle at L5 were obtained, with the exception of larger muscles, where the left and right sides were imaged separately (Figure 1). Three images were captured for the right and left LM muscle. This L5 level was selected based on a previous study reporting that decreased LM CSA and increased side-to-side asymmetry at this level was a predictor of LBP and lower-limb injuries in elite AFL players.[21]
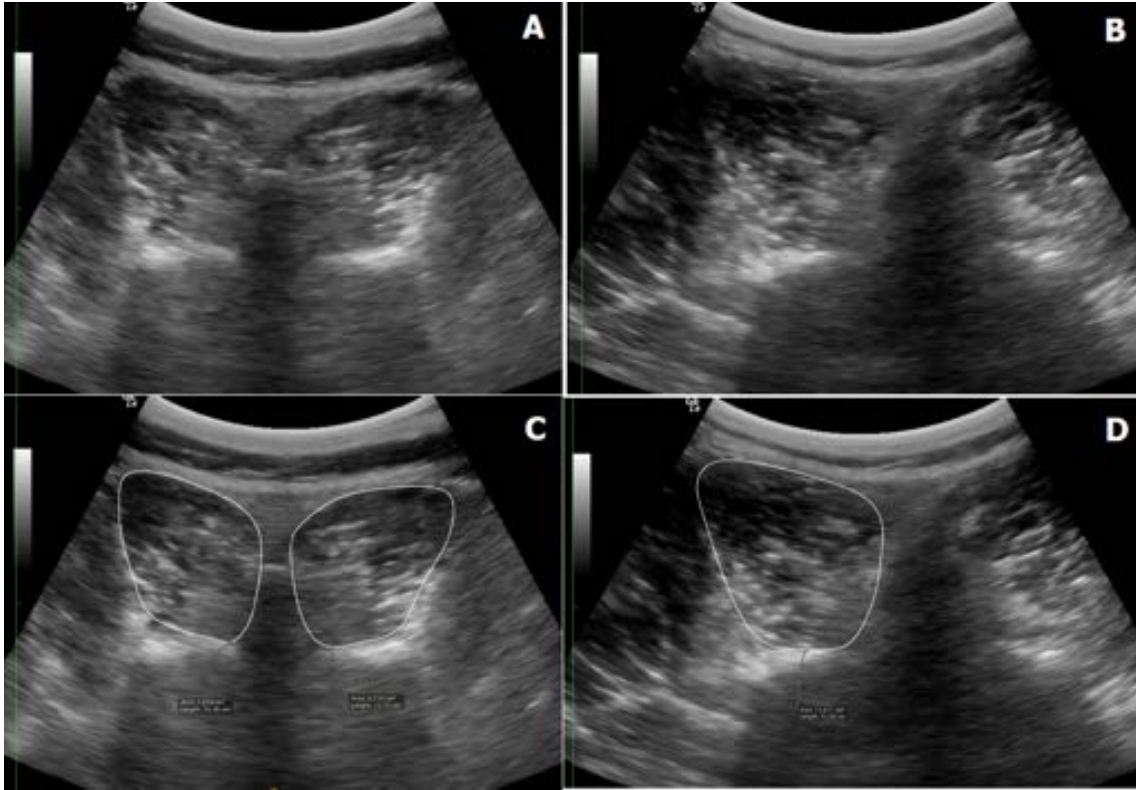
FIGURE 1. A) Bilateral transverse image at L5 vertebral level showing the right and left multifidus and shadow of spinous process of a female hockey player, B) Left side image at L5 vertebral level showing left multifidus muscle and shadow of spinous process of a male hockey player. Due to the larger musculature, the right and left multifidus were imaged separately. C) and D) images are showing the multifidus muscle cross-sectional area (CSA) measurements for the same players presented in images A) and B).

Muscle function was then assessed by obtaining thickness measurements of LM muscle at rest and during contraction (Figure 2). LM muscle was imaged bilaterally, in the parasagittal section, allowing for the visualization of the L5/S1 zygapophyseal joints. Participants were instructed to relax while three images were captured bilaterally. Participants were then instructed to perform a contralateral arm lift to induce a submaximal contraction [26,29,51] Each participant was given a handheld weight[26] [based on subject body weight: 1) <68.2kg = 0.68kg weight, 2) 68.2-90.9kg=0.9kg weight, 3) >90.9kg=1.36kg weight] and instructed to raise the loaded arm 5 cm off the examination table with the shoulder in 120° of abduction and elbow 90° of flexion. The handed weight was designed to load the LM muscle to approximately 30% of maximal voluntary

isometric contraction. Participants were instructed to hold their breath at the end of normal exhalation (minimize the effect of respiration on thickness measurement) and maintain the contraction for 3 seconds. Each player had a practice trial, followed by 3 contralateral arm lifts on each side.
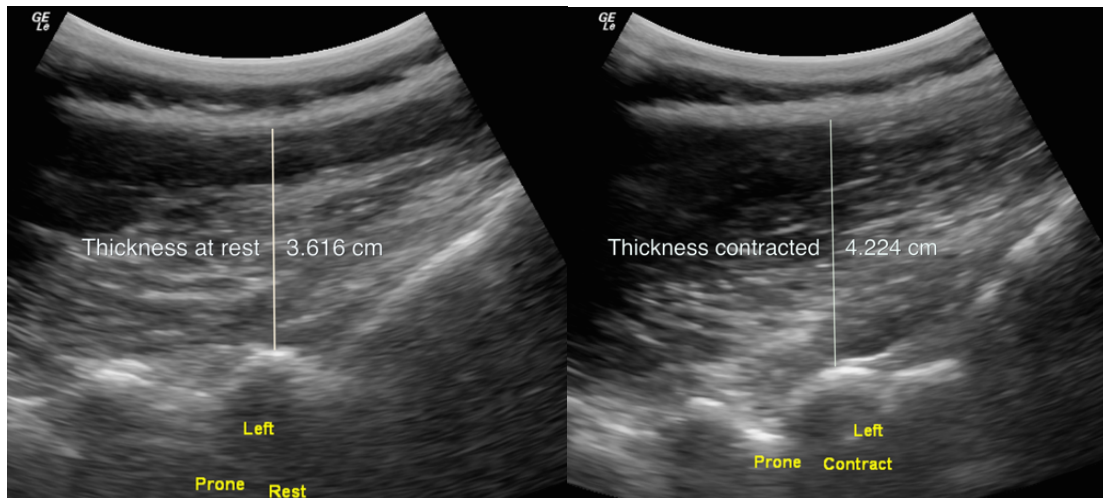

FIGURE 2. Multifidus thickness muscle measurements at rest (left) and during contraction (right) in the prone position at L5-S1 (e.g. left side male hockey player).

Standing measurements

Players were asked to stand barefoot on the floor with their arms relaxed on each side. In order to achieve a habitual standing posture, participants were instructed to march on **the** spot for a few seconds and remain **at** the position where their feet landed. The same procedure as described above was conducted to obtain LM size and thickness measurements at rest. To contract the LM muscle, each participant was asked to perform a contralateral arm lift, with the shoulder placed in 90° of flexion, elbow in complete extension and the wrist in **a** neutral position (palm facing down),[55] while holding the previously determined hand weight and **asked to** maintain the contraction for 3 seconds. Each player had a practice trial, followed by 3 contralateral arm lifts on each side.

Imaging assessment

Ultrasound images were stored and analyzed offline. LM muscle CSA and thickness measurements were conducted using OsiriX imaging software (OsiriXLiteVersion 9.0, Geneva, Switzerland). LM CSA measurements were obtained by tracing the muscle borders on both sides. The relative % asymmetry in CSA between the right and left side was calculated using the following formula: [(larger side – smaller side)/larger side x 100]. LM muscle thickness was assessed using linear measurements from the tip of the L5/S1 zygapophyseal joint to the inside edge of the superior muscle border, at rest and during contraction in both positions (e.g. prone and standing). Each measurement was repeated 3 times (on 3 different images) on each side, and the average value was used in the analyses. LM muscle function and contractile ability in the prone and standing position was calculated as a percent change using the following formula: [(thickness contraction – thickness rest)/thickness rest)x100]. LM muscle EI was measured using grayscale analysis imaging (ImageJ, National Institute of health, USA, Version 1.49) using the standard histogram function of pixels expressed as value between 0 (black) and 255 (white).[2] Enhanced EI is indicative of a greater amount of intramuscular fat and connective tissue. Prior to EI measurements, each image was calibrated by measuring the number of pixels within a known distance of 1 cm. EI was determined by tracing a region of interest (ROI) representing the LM muscle CSA (in the prone position only), avoiding the inclusion of bone or surrounding fascia. The average value of 3 EI measurements (on 3 different images) on each side was used in the analyses. All measurements were obtained by an experienced athletic therapist researcher (MF), with extensive experience in spine imaging analysis, and was also trained by a senior musculoskeletal ultrasound radiologist (MB) prior to the beginning of this study. At the time of imaging assessment, the rater was blinded to players' characteristics and LBP history. The intra-

class correlation coefficients ($ICC_{3,1}$) ranged between 0.96-0.99 with standard error of measurement (SEM) of 0.04-0.14 cm^2 for all prone measurements, and 0.96-0.98 with SEM of 0.06-0.25 for all standing measurements indicating a high level of reliability. The reliability for the LM EI measurement was 0.99 with a SEM of 1.97.

DEXA

Each player had a full body DEXA scan (Lunear Prodigy Advance, GE) performed by a certified medical imaging technologist (SF). Prior to imaging, all participants were asked to remove any metal and were required to wear loose fitting clothing, to avoid interference with the scan. Age, height, weight and ethnicity were entered in the computer software prior to imaging. Participants were asked to lie down supine in the center of the scanner with their arms slightly away from the body, thumbs pointing upwards, with their legs slightly apart and toes pointing upwards. Total lean mass, total bone mass, total fat mass and total percent body fat were **obtained**.

Statistical Analysis

Means and standard deviations were calculated for participants' characteristics, LM measurements of interest and body composition measurements. Analysis of variance (ANOVA) was used initially to assess the difference between LM muscle characteristics between male and female players. Linear regression models and spearman correlation were used to assess the relationship between LM muscle measurements of interest and body composition measurements. Analysis of covariance (ANCOVA) was used to examine the difference between LM muscle measurements between players with and without LBP. Separate analyses were performed for the presence of LBP at 4-weeks and 3-months prior. The variables "weight" and "height" were

entered as covariates in the analyses. All analyses were performed with STATA (version 12.0, StataCorp, LP, College Station, Texas).

Results:

Players' characteristics are presented in Table 1. The mean ± SD age, height, and weight was 21.4±1.4 years, 173.8±9.1 cm, and 76.0±12kg, respectively. The average number of years playing hockey was 10.7±4.1, including 1 to 5 years at the university level.

Table 1. Players' characteristics ((mean±SD) or n)

	All (n=32)	Female (n=18)	Male (n=14)
Age (yr)	21.4±1.4	21.3±1.8	21.6±0.8
Height (cm)	173.8±9.1	167.7±5.6	181.8±6.2
Weight (kg)	76.0±12.0	67.7± 7.8	86.7±6.8
Total lean mass (kg)	57.3±11.5	48.3±5.5	68.8±48.9
Total bone mass (kg)	3.2± 5.9	2.83±2.9	3.7±5.1
Total Fat mass (kg)	16.1±4.4	17.1± 4.5	14.7±4.1
Total body fat %	22.2±6.1	25.9±4.8	17.5±4.1
BMI	25.0±2.2	24.0±2.0	26.2±1.6
Dominant leg (n)			
Right	23	13	10
Left	5	4	1
Either	3	0	3
Position (n)			
Defense	11	5	6
Forward	15	11	4
Centre	2	0	2
Goalie	4	2	2
Hockey competitive level (yr)	10.7±4.1	9.1±3.9	12.4±3.8
Hockey university level (yr)	2.3±1.1	2.5±1.2	1.9±0.9
LBP 4-weeks prior (n)	13	6	7
LBP 3-months prior (n)	13	5	8
LBP last competitive year (n)	6	3	3
LBP location 4-weeks prior (n)			
Centered	7	3	4
Bilateral	4	2	2
Unilateral	2	1	1
LBP location 3-months prior (n)*			
Centered	6	3	3

Bilateral	4	1	3
Unilateral	1	1	1
VAS LBP (0-10) 4-weeks prior	4.0±1.4	3.8±1.7	4.1±1.2
VAS LBP (0-10) 3-months prior	4.3±1.7	4.2±2.5	4.3±1.3

kg=Kilograms, LBP=low back pain, VAS=visual analogue scale
  *= One missing data from male group

*LM muscle characteristics and body composition*

LM muscle CSA, side-to-side asymmetry (right vs. left), EI, thickness at rest and during contraction, % thickness change during contraction in prone and standing for female and male players are presented in Table 2. LM CSA in the prone position was significantly larger in male compared to female players (p=0.03). EI was significantly greater in female athletes compared to male athletes (p<0.001, rho=0.63). There was no significant difference between male and female players for LM CSA side-to-side asymmetry, or thickness measurements at rest or during contraction. LM CSA was significantly associated with height (rho=0.58, p<0.001; rho=0.55, p=0.001), weight (rho=0.53, p=0.002; rho=0.45, p=0.008), total bone mass (rho=0.48, p=0.005; rho=0.47, p=0.007) and total lean body mass (rho=0.49, p=0.004; rho=0.49, p=0.004) in prone and standing, respectively. BMI was not correlated with LM CSA in prone or standing. LM muscle EI was strongly associated with total percentage body fat (rho=0.76, p<0.001, Figure 3), total lean mass (rho=-0.60, p<0.001) and total fat mass (rho=0.56, p=0.001). BMI was not associated with LM muscle EI (rho=-0.33, p=0.06). LM muscle EI was not associated with function (e.g. % thickness change during contraction).

Table 2. Multifidus muscle measurements (mean (SD)) of interest in prone and standing for the right and left side.

	Female		Male	
	Right	*Left*	*Right*	*Left*
PRONE				
CSA (cm^2)*	**8.96 (1.18)**	**9.01 (1.23)**	**9.93 (1.53)**	**10.07 (1.50)**
CSA asymmetry (%)	3.19 (3.08)		4.58 (3.17)	
CSA EI**	**74.24 (14.86)**	**71.23 (17.50)**	**51.23 (14.64)**	**50.94 (13.66)**
Thickness (cm)				
Rest	2.96 (0.35)	3.04 (0.41)	3.02 (0.48)	3.10 (0.52)
Contracted	3.36 (0.35)	3.42 (0.39)	3.52 (0.57)	3.61 (0.61)
% change	14.07 (6.49)	13.02 (6.67)	16.60 (10.19)	16.88 (7.14)
STANDING				
CSA (cm^2)	10.38 (1.34)	10.48 (1.39)	11.29 (1.65)	11.49 (1.78)
CSA asymmetry (%)	3.30 (2.73)		3.75 (2.86)	
Thickness (cm)				
Rest	3.44 (0.39)	3.46 (0.42)	3.55 (0.58)	3.55 (0.64)
Contracted	3.52 (0.37)	3.55 (0.45)	3.69 (0.61)	3.74 (0.67)
% change	2.59 (4.43)	2.70 (4.97)	4.22 (4.36)	5.57 (5.33)

CSA=Cross-sectional area; EI=echo-intensity; %change= %change from thickness at rest to contacted
* $P<0.05$; ** $P<0.001$
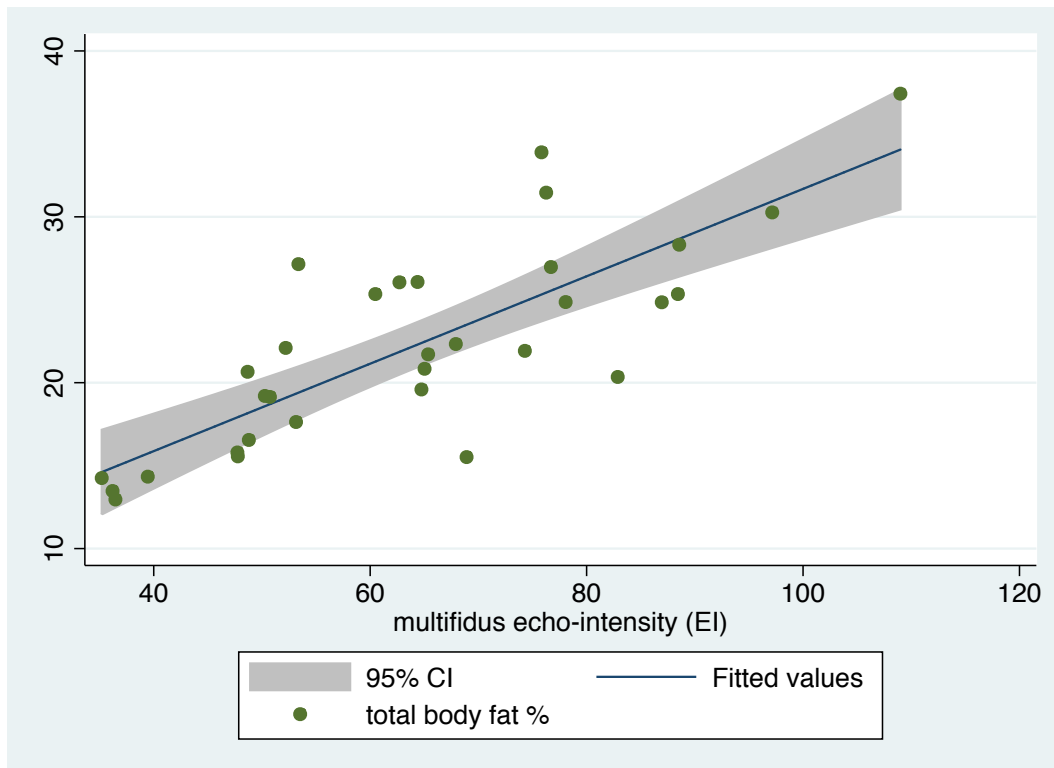
FIGURE 3. Correlation between multifidus muscle echo-intensity (EI) and total body fat percentage acquired with DEXA.

LBP Comparisons

LM CSA in the prone position was significantly smaller in players reporting the presence of LBP 4-weeks prior to measurement (F=9.62, p=0.004) (Table 3). Similarly, LM thickness at rest was significantly smaller in players with LBP 4-weeks prior (F=4.62, p=0.04). LM CSA side-to-side asymmetry in the standing position was also significantly greater in players who reported LBP 3-months prior (F=4.67, p=0.03) (Table 4). There were no significant differences for LM EI or % thickness change in prone or standing between players reporting LBP 4-weeks or 3-months prior to measurement.

Table 3. Adjusted means[a] (mean (SE)) of multifidus muscle measurements in prone and standing for players with and without LBP in past 4-weeks.

	No LBP (n=19)	LBP past 4-week (n=13)
PRONE		
CSA (cm^2)	**9.42 (0.23)**	**8.94 (0.29)**
CSA asymmetry (%)	3.92 (0.76)	3.61 (0.92)
CSA_EI[b]	61.67 (2.44)	65.60 (2.97)
Thickness (cm)		
Rest	**3.15 (0.09)**	**2.83 (0.11)**
Contracted	3.58 (0.10)	3.29 (0.13)
% change	13.85 (1.59)	16.82 (1.93)
STANDING		
CSA (cm^2)	10.78 (0.32)	10.95 (0.39)
CSA asymmetry (%)	3.38 (0.65)	3.67 (0.79)
Thickness (cm)		
Rest	3.61 (0.11)	3.32 (0.13)
Contracted	3.72 (0.11)	3.46 (0.14)
% change	3.32 (0.86)	4.02 (1.05)

LBP=low back pain; CSA=Cross-sectional area; EI=echo-intensity;
%change= %change from thickness at rest to contacted
[a] Adjusted means for weight and height
[b] Adjusted means for weight, height and total body fat %
bold = *P*<0.05

Table 4. Adjusted means[a] (mean (SE)) of multifidus muscle measurements in prone and standing for players with and without LBP in the past 3-months.

	No LBP (n=19)	LBP past 3-month (n=13)
PRONE		
CSA (cm^2)	9.49 (0.26)	9.16 (0.32)
CSA asymmetry (%)	4.35 (0.75)	2.99 (0.92)
CSA_EI[b]	62.65 (2.58)	64.17 (3.19)
Thickness (cm)		
Rest	3.09 (0.9)	2.93 (0.12)
Contracted	3.52 (0.11)	3.38 (0.14)
% change	14.29 (1.64)	16.19 (2.01)
STANDING		
CSA (cm^2)	10.81 (0.33)	10.92 (0.40)
CSA asymmetry (%)	**2.63 (0.61)**	**4.77 (0.75)**
Thickness (cm)		
Rest	3.54 (0.12)	3.41 (0.14)
Contracted	3.68 (0.12)	3.51 (0.15)
% change	4.04 (0.87)	2.94 (1.07)

LBP=low back pain; CSA=Cross-sectional area; EI=echo-intensity; %change= %change from thickness at rest to contacted
[a] Adjusted means for weight and height
[b] Adjusted means for weight, height and total body fat %
bold = $P<0.05$

Discussion

LM muscle characteristics

In accordance with previous reports, the results of the current study showed that LM muscle CSA in prone position was significantly larger in male players compared to female players.[53,54] However, our findings suggest a hypertrophy of the LM muscle at the L5 level, as the resting LM CSA of the hockey players was greater than previously published normative ultrasound data on non-athletic healthy subjects (females=~6.0cm^2, males=~7.5cm^2) of slightly greater age (females=32.76±6.53, males=31.88±6.53),[63] and comparable to elite weightlifters (females=8.65±0.32, males=10.95±0.31cm^2) of similar age (21.49±0.59 years).[49] Indeed, the core muscles are targeted in nearly every aspect of playing hockey. LM activation is required to stabilize the spine and upper body during skating, battling, changing direction and checking.[56]

Strong LM and core muscles activation is also critical while shooting to generate force and rotational power.[46] Furthermore, hockey players spend most of the game with their hips, knees and spine flexed. Holding a forward flexed position (in comparison to an upright position) significantly increases the LM muscular demand, thereby leading to an eccentric contraction.[32] As such, the LM hypertrophy observed in our female and male athletes is likely a response/adaptation to the specific physical demands of the sport.

Despite the asymmetrical nature of hockey, our findings showed no significant side-to-side asymmetry in LM muscle CSA in male or female players. In fact, LM side-to-side asymmetry in the prone position was <5%, which corroborates with previous reports in healthy normal subjects[20,54,63] and athletes.[22,49] Furthermore, the level of LM CSA side-to-side asymmetry when assessed in the standing position was similar to prone lying and remained <5%. As the LM muscle is contracted in a stabilizing role while standing, the CSA significantly increased from the prone lying to standing position. Accordingly, the % thickness change in standing was significantly smaller than the prone position, a finding that is consistent with a previous study in young healthy subjects (mean age of 31.8 years).[55] We are unaware of previous work that has assessed LM characteristics in the standing position in athletes. Though, LM % thickness change (contraction) in prone position was in accordance with values previously reported in elite athletes.[22]

Association between LM characteristics and body composition

As expected, EI was significantly greater in female players as compared to male players, demonstrating that females naturally a higher level of LM fatty infiltration/connective tissue due

to a greater percentage of body fat.[9,27,37,47] LM muscle EI was not associated with function (e.g. % thickness change). Given the intuitive perspective that increased fatty infiltration would have negative effects on muscle function, this finding was unexpected but in accordance with a previous study.[31] Other measures of LM muscle function (e.g. strength, electrical muscular activity, endurance) may have shown stronger associations with LM fatty infiltration, as previous research has reported increased intra-muscular fatty infiltration to be associated with decreased thigh muscle power and performance.[8,61,62] One should keep in mind the unique nature of the LM muscle, as it has been clinically observed and previously reported that LM fatty infiltration deposit is not homogeneous and mostly occurs in the deepest portion of the muscle.[1,23] Furthermore, differences in muscle activation between the deepest and superficial muscle layers have also been observed. [33]

LM muscle CSA was strongly dependent on hockey players' weight, height, total lean muscle mass and total bone mass. BMI was not correlated with LM muscle CSA, although it is the most commonly used variable to adjust for between-sex and between-subject variability in the field. One should take into account that BMI is often a poor measure of body composition in athletes, due to the inability to differentiate between muscle and fat mass. While previous studies of non-athletic subjects have reported a positive association between BMI and paraspinal muscle fat content,[13,24,41] others reported no such relationship.[9,27] LM muscle EI was strongly correlated with total percentage body fat, total lean mass and total fat mass, confirming that the influence of body composition on measurements on LM muscle quality (composition) cannot be ignored. Two previous studies using bioimpedance also reported a correlation between paraspinal muscle fatty infiltration and general body fat.[9,45] However, the accuracy of bioimpedance compared to

DEXA for percentage body fat measurement substantially decreases as weight-status increases,[60] thus our correlation estimates should be more accurate.

LM muscle characteristics and LBP

In our sample of hockey players, 41% experienced some level of LBP during the preseason. When assessing LM muscle characteristics according to LBP history, our results revealed that LM CSA was significantly smaller in players who reported LBP 4-weeks prior to measurement. Similarly, resting LM CSA in the prone position was also significantly smaller in players with recent LBP (previous 4-weeks). These findings corroborate with previous studies in athletes, where professional ballet dancers [17] and soccer players [19] with LBP also showed deficits in resting LM CSA compared to athletes with no LBP. Other studies, [34,49,52] however, reported no such association suggesting that some athletic populations may behave differently with regards to LM muscle size and LBP, possibly due to competing influences including specialized movements and specific training effects.[52] Our findings, along with others,[17,22] also indicates that players reporting the presence of LBP in the previous 3-months had greater LM side-to-side asymmetry, when assessed in the standing position. Such asymmetry, however, was not observed when players were measured in the prone position. This discordance between findings in prone and standing positions suggest that LM muscle likely behave differently while contracted in a stabilizing role (e.g. standing), which may be a more accurate position for the assessment of LM function.[55] Further studies assessing the association between LM muscle characteristics in functional positions (such as standing) and LBP are needed to confirm and expand our findings.

Similar to previous studies,[19,55] our results with regards to LM % thickness change showed that players with and without LBP were equivalently able to contract the LM muscle in the prone and standing positions. However, there was a trend for players with a history of LBP to have a greater LM contraction (e.g. higher % thickness change), a finding that has also been reported in previous studies in athletic[19] and non-athletic populations.[55] Subjects with LBP have been found to develop movement and motor control impairments, which may be manifested as a lack of segmental control of the neutral zone.[39] Thus, increased LM muscular activation may be developed as a compensatory mechanism.[30] While subjects with LBP have also been reported to have more LM fatty infiltration,[13,37,46,50] this was not the case for our hockey players as LM muscle EI was comparable between players with and without LBP. This finding, however, is consistent with previous studies that have compared people with LBP to healthy age- and activity-matched subjects and found no association between paraspinal muscle fat content and LBP.[5,11] Furthermore, previous studies also showed no association between LBP and fatty infiltration in young adults.[37,40] As the mean age of our hockey players was 21.4±1.4 years, the young age likely explains the lack of fatty infiltration. In addition, the mean VAS score of players reporting the presence of LBP varied between 4.0±1.4 and 4.3±1.7, suggesting a relatively low level of pain and disability.

Ice hockey players require strong, symmetrical and highly reactive core musculature in order to properly transmit force generated through the kinetic chain. Given that both smaller LM and greater LM side-to-side asymmetry were associated with the presence of LBP, altered trunk and/or lower limb movements may be responsible for the LM muscle changes observed in hockey players with LBP in this study. While specific stabilization exercises were effective to

restore LM muscle CSA and decreased LBP symptoms in a group of elite cricketers,[22] additional intervention trials assessing the effects of such exercise programme in different athletic populations with LBP are needed.

A limitation of this study is the relatively small sample size, though comparable to previous studies conducted with elite athletes. This might have affected some of the analyses. Additional investigations with more hockey teams are needed to confirm our results. Our study had a sufficient number of asymptomatic players, which allowed for a representative comparison between players with and without LBP. Although the number of players reporting LBP 4-weeks prior and 3-months prior were equal (n=13), the players included in each LBP group (e.g. 4-weeks and 3-months) differed slightly.

Conclusion:

This study provides new insights on LM muscle morphology and activation during movement in prone and standing positions in hockey players, and their associations with body composition and LBP. Our results suggest a hypertrophy of the LM muscle, which is likely due to the high demands of the sport on the back musculature. DEXA body composition measurements were significantly associated with LM morphology, suggesting that the influence of body composition on LM muscle quality cannot be ignored. While LM muscle function (e.g. contraction) was not associated with EI or LBP, hockey players with LBP showed specific deficits in resting LM CSA and thickness in the prone position, as well as greater LM side-to-side asymmetry in standing when compared to players without LBP. Preseason screening assessment of the LM muscle may be useful in injury prevention programs, which could help decrease the high prevalence of LBP

in this athletic population. Additional studies in athletes are needed to confirm these results, and allow for the investigation of further aspects of LM muscle function and neuromuscular motor control.

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