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Myofascial force transmission between the latissimus dorsi and gluteus maximus muscles: An in vivo experiment

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ABSTRACT

There are extensive connections between the latissimus dorsi (LD) and gluteus maximus (GMax) muscles and the thoracolumbar fascia (TLF), which suggests a possible pathway for myofascial force transmission. The present study was designed to provide empirical evidence of myofascial force transmission from LD to contralateral GMax through TFL in vivo. To accomplish this goal, we evaluated whether active or passive tensioning of the LD results in increased passive tension of the contralateral GMax, indexed by changes in the hip resting position (RP) or passive stiffness. The hip RP was defined as the angular position in which the passive joint torque equals zero, and passive hip stiffness was calculated as the change in passive torque per change in joint angle. Thirty-seven subjects underwent an assessment of their passive hip torque against medial rotation by means of an isokinetic dynamometer. These measures were carried out under three test conditions: (1) control, (2) passive LD tensioning and (3) active LD tensioning. Electromyography was used to monitor the activity of the hip muscles and the LD under all conditions. Repeated measures analyses of variance demonstrated that passive LD tensioning shifted the hip RP towards lateral rotation ($p=0.009$) but did not change the passive hip stiffness ($p>0.05$). Active LD tensioning shifted the hip RP towards lateral rotation ($p<0.001$) and increased the passive hip stiffness ($p\leq 0.004$). The results demonstrated that manipulation of the LD tension modified the passive hip variables, providing evidence of myofascial force transmission in vivo.

1. Introduction

Studies indicate that the tension produced by a particular muscle is not entirely transmitted to its tendons but can also be transmitted to connective tissues within and surrounding the muscle (endomysium, perimysium, epimysium) and to non-muscular connective tissues (fascia, neurovascular tract) (Huijing, 2009; Purslow, 2010; Smeulders and Kreulen, 2007; Yucesoy, 2010). This force transmission through connective tissue has been termed myofascial force transmission and can be classified as intramuscular (between muscle fibers), intermuscular (between muscles) and extramuscular (between muscle and adjacent non-muscular tissues) (Huijing, 1999). Thus, myofascial pathways allow tension produced by a muscle to propagate outside its boundaries and to potentially affect body structures non-adjacent to it.

The extensive connections of the superficial lamina of the thoracolumbar fascia (TLF) to latissimus dorsi (LD) and gluteus maximus (GMax) muscles (Barker and Briggs, 1999; Bogduk and Macintosh, 1984) suggest the occurrence of extramuscular myofascial force transmission between these structures. Evidence for this form of myofascial force transmission has been provided by studies in cadavers, which have demonstrated that tension in the LD or GMax significantly displaces the TLF, even on its contralateral side (Barker et al., 2004; Vleeming et al., 1995). In these studies, LD and GMax tensioning occurred by placing direct traction on these muscles in situ. These studies suggest that the LD and GMax could act as a functionally coupled unit.

Some authors have questioned the existence of myofascial force transmission in response to the physiological movement of joints (Herbert et al., 2008; Maas and Sandercock, 2008). Because the mechanical properties of tissues are modified in cadavers (Wilke et al., 1996), it is possible that the TLF traction that results from muscle stretching or contraction promotes different outcomes in live subjects. Therefore, in vivo investigations are

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necessary to demonstrate myofascial force transmission via the TLF under physiological conditions.

One way to demonstrate that force is transmitted via TLF in vivo is to investigate whether LD tensioning increases tension on the contralateral GMax. If tensioning of the LD changes the tension on the contralateral GMax, variables related to the net passive torque of the hip joint, in the transverse plane, are expected to change. The net passive torque of a joint is determined by the opposing passive tension produced by all elastic tissues crossing that joint (Souza et al., 2009; Winters et al., 1988). This configuration creates an equilibrium position, corresponding to the joint's resting position (RP), in which the opposing torques are equal in magnitude and offset each other (i.e. where the net passive torque equals zero). Thus, increasing the tension in one of the opposing tissues shifts the joint's RP towards these tissues (Souza et al., 2009; Wu et al., 2012). In addition, this tension also increases the joint passive stiffness (i.e. the change in net passive torque per change in joint angle) (Ocarino et al., 2008; Souza et al., 2009). Therefore, we hypothesize that increases in GMax tension due to LD tensioning would shift the hip joint RP towards lateral rotation and increase the passive hip stiffness. To provide evidence for force transmission via TFL under physiological conditions, we evaluated whether stretching or contraction of the LD muscle changes RP and passive stiffness of the contralateral hip in healthy subjects. A prestressed two-spring model was used to give formal expression to the hypothesis of the study (Souza et al., 2009).

2. Methods

This study was carried out with 42 volunteers. However, five of them were excluded due to their inability to keep their hip muscles relaxed during the passive test. Data from 37 individuals (15 men and 22 women), with a mean age of 24.92 ± 3.21 years, a mean body mass of 64.43 ± 11.02 kg and a mean height of 1.69 ± 0.09 m, were analyzed. The inclusion criteria were a pain-free passive range of motion of 25° of medial rotation and 25° of lateral rotation at the hip and the absence of any musculoskeletal injuries in the last six months. This study was approved by the Research Ethics Committee of the Universidade Federal de Minas Gerais.

2.1. Prestressed two-spring model

A prestressed two-spring model was used to represent the influence of hip tissue's passive tension on the net passive joint torque (Souza et al., 2009). This model is generic for synovial joints and allowed qualitative directional predictions about the changes in joint RP and passive stiffness in response to tension increases in the elastic structures surrounding the hip joint. Since opposing elastic structures of a joint are simultaneously under passive tension (passive co-tension), they can be represented by two non-linear springs imparting opposite forces on a massless body (Fig. 1A) (Souza et al., 2009). The sum of these opposite forces (model's net force) represents the net passive joint torque (Fig. 1B). The position of the model's body represents the joint angular position. The body equilibrium position in which the springs opposite forces are equal and offset each other represents the joint's RP. Finally, the change of the net force per change in position (model's stiffness) represents the joint's passive stiffness.

This model was implemented in a Matlab routine (The Mathworks Inc.) to simulate the effects of tension increases in Spring 1 on the model's RP and stiffness (Fig. 2). These effects constituted predictions on changes in the RP and passive stiffness of the hip in a condition in which only the tension of the GMax is increased. The simulation showed that an arbitrary increase in Spring 1 tension led to a shift in the model's RP towards this spring. This manipulation also led to an increase in the model stiffness, in body positions before and beyond the new RP. According to our hypothesis, the model's predictions supports that GMax tensioning (due to LD tensioning) would cause a shift of hip RP towards lateral rotation and an increase in passive hip stiffness, in joint positions before and beyond the new hip RP.

2.2. Procedures

After signing the informed consent form, the subjects underwent electromyographic (EMG) and dynamometric assessments. All tests were performed in the dominant upper limb and the contralateral lower limb.

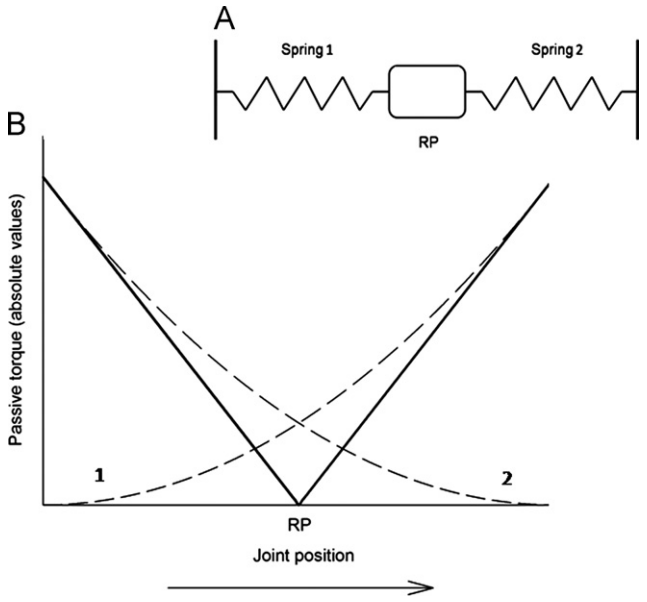

Fig. 1. Prestressed two-spring model. (A) Schematic representation of the model, in which two non-linear springs are attached to opposite sides of a massless body. The position of the body represents the joint angular position, and the springs represent a set of elastic structures that produce torques in opposite directions at the joint. Both springs are with a length greater than their slack lengths (prestressed system), and thus, the body is under the simultaneous action of both springs. In this figure, the body is at its resting position (RP), i.e. an equilibrium position in which the forces produced by opposing springs are equal and cancel each other. (B) Graphical representation of the absolute values of passive torque produced by joint opposing structures at each joint position. These curves were constructed from simulations performed on a routine developed in Matlab. If the joint moves in a specific direction (see arrow), the structures represented by the spring 1 are stretched and produce an increasing resistance torque (curve 1). In turn, the structures represented by the spring 2 are being shortened during this movement and produce a decreasing torque that contributes to the body displacement (curve 2). Thus, the passive torque that is measured during the joint movement is a net passive torque (solid curve), which is influenced by the opposite torques produced by the structures surrounding the joint. Note that the point in which the curves 1 and 2 cross each other represents joint RP, i.e. position in which the net passive torque equals zero.

2.2.1. Evaluation of the maximal voluntary isometric contraction

Three maximal voluntary isometric contractions (MVICs) of shoulder adduction associated with scapular depression were performed using a sphygmomanometer (Becton Dickinson, Juiz de Fora, Brazil) inflated to 20 mmHg and positioned between the arm and trunk, 15 cm distal to the acromion. During the MVICs, the EMG signal of the LD was recorded (Mega Electronics, Kuopio, Finland). The greatest LD EMG activity that was registered during the three MVICs was used to normalize the LD signal obtained during active tensioning condition.

2.2.2. Evaluation of passive torque

The passive hip torque during medial rotation was measured by an isokinetic dynamometer in passive mode (speed of 5°/s) (Biodex Medical Systems, Shirley, USA). The participant was placed in prone position, and the tibial tuberosity was aligned with the rotational axis of the dynamometer (Fig. 3). The equipment's attachment moved the participant's hip from 25° of lateral rotation to 25° of medial rotation. Initially, 15 repetitions of this movement were performed to reduce the effects of tissue viscosity. Afterwards, three repetitions were carried out at each of three separate test conditions (Fig. 3): (1) control: the participant remained with his/her upper limb resting along his/her side; (2) passive LD tensioning: the participant remained with his/her shoulder passively positioned at 120° of flexion and the scapula passively elevated through a cable attached to the wrist; (3) active LD tensioning: the participant performed shoulder adduction and scapular depression, actively pressing the sphygmomanometer that was inflated to 20 mmHg. The volunteer applied a pressure equivalent to 25% of the peak pressure recorded during the MVICs. Variations of 10% above or below this pressure were allowed. The order of conditions was random. To register the torque generated by the equipment's hip attachment mass, one repetition was performed without the participant.

During the passive torque evaluation, the EMG activity of the hip muscles (GMax, gluteus medius, biceps femoris, tensor fascia lata and adductor magnus) was monitored to ensure that these muscles were relaxed under all conditions.

The resting EMG of the LD was monitored during LD control and passive tensioning. The EMG data were collected at a frequency of 1000 Hz and band-pass filtered with cut-off frequencies of 30 and 500 Hz. After each repetition, these data were immediately processed, which allowed the signal obtained under each experimental condition to be compared with the signal obtained when the participant was resting. Any repetitions during which the EMG signal was equal to or greater than two standard deviations beyond the signal of the muscle during relaxation were rejected (Lamontagne et al., 1997). Three passive repetitions were obtained for each test condition.

The intraclass correlation coefficient was determined to investigate between-repetitions reliability of the passive torque measured ($\alpha = 0.05$). The standard error of measurement was calculated to establish between-repetitions variability. These values are described in Table 1.

2.3. Data reduction

The gravitational torques produced by the shank and foot mass of the participants during the tests were calculated based on anthropometric data (Winter, 1990). These torques and the one generated by the dynamometer attachment mass were subtracted from the total torque measured by the dynamometer. The absolute values of torque were plotted for each joint angle, and this torque-angle curve was used to obtain the following dependent variables: hip RP, stiffness before the RP and stiffness beyond the RP.

The RP was defined as the position where the passive hip torque equals zero. For each individual, the mean of three RP values was calculated for each test condition. The passive stiffness was calculated as the mean slope of the torque-angle curve over a range of 15° before and 15° beyond the RP. In nine participants, LD tensioning considerably shifted the RP towards lateral rotation, and therefore, they did not have the required 15° before the RP for analysis. In these cases, the smallest range available before the RP was used in the analysis of those individuals under all conditions. This approach ensured that any changes in stiffness were due to the manipulations performed in this study and not to differences in the length of the analyzed part on the torque-angle curve. For each individual, the mean of three stiffness values before and beyond the RP was calculated for each test condition.

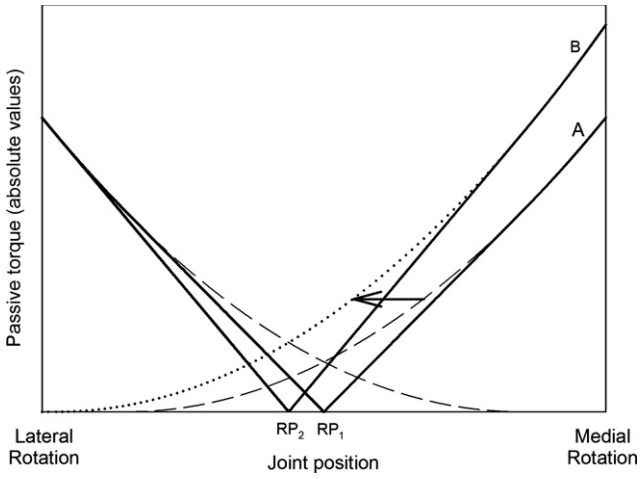

Fig. 2. Possible changes in the resting position and passive stiffness due to tensioning of tissues that resist medial hip rotation. These curves were constructed from simulations performed on a routine developed in Matlab. The dashed curves represent the torques produced by opposing elastic structures of the hip during medial rotation, and the solid curve (A) shows the net passive torque. The point where the dashed curves cross each other represents the joint resting position (RP_1). The RP_1 represents the position in which the torques against lateral and medial rotations are equal. An increase in the gluteus maximus tension shifts the dashed curve which represents the resistance torque to medial rotation into a position of greater lateral rotation (see arrow). After this displacement occurs, the curves of the opposing passive torques cross at a new point (RP_2). The new resting position (RP_2) is shifted to a position of greater lateral rotation. Note that the slope of the net curve (B) is greater than the slope of the net curve (A) at positions both before and beyond the resting position (i.e. the global stiffness of the curve B is greater than the curve A).

2.4. Statistical analysis

Repeated measures analyses of variance (ANOVA), with one factor (LD tensioning) and three levels (test conditions), were used to verify the effect that LD tensioning had on each dependent variable. Pre-planned contrasts were used to compare the passive and active tensioning conditions to the control condition. The significance level was set at 0.05.

3. Results

Table 1 shows the means and standard deviations for the dependent variables. The mean intensity of LD activation during active tensioning was $29.13 \pm 18.08\%$ of the MVIC. The ANOVAs demonstrated a significant main effect for the hip RP and passive stiffness before and beyond the RP ($p \leq 0.001$). Contrast analyses showed that passive LD tensioning modified the RP by shifting it into a position of greater lateral rotation ($p = 0.009$), but it did not change the stiffness, either before or beyond the RP ($p > 0.05$). Active LD tensioning shifted the RP into a position of greater lateral rotation ($p < 0.001$) and increased the stiffness before ($p = 0.004$) and beyond ($p < 0.001$) the RP. The torque-angle curve from one volunteer is depicted in Fig. 4.

4. Discussion

The results showing that forces were transmitted from the LD to the GMax support the existence of myofascial force transmission in vivo. Specifically, the LD stretching or contraction shifted the hip RP towards greater lateral rotation, and the LD contraction increased globally the passive hip stiffness. Myofascial force transmission was supported by the likely tension propagation between muscle and connective tissues, occurring from the more micro to the more macroscopic scale. Tension produced by LD sarcomeres was transmitted to the endomysium by connections between (1) the sarcomeres and basement membrane of muscle fibers through cytoskeleton, and (2) the basement membrane and endomysium through protein complexes (Huijing et al., 2011;

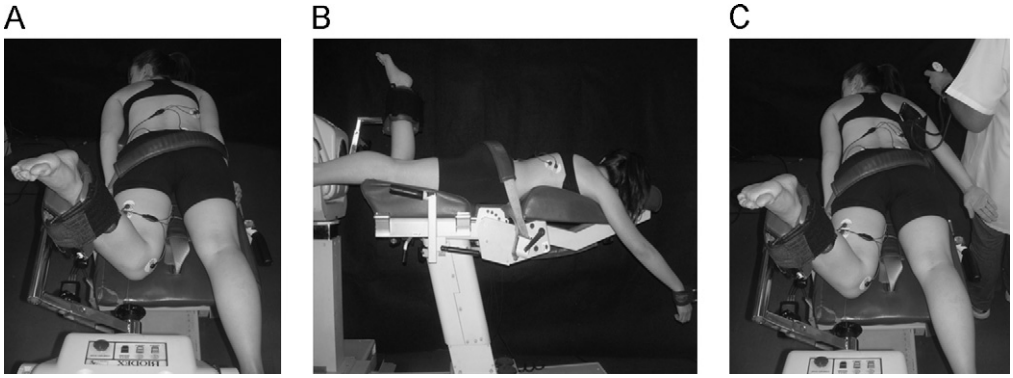

Fig. 3. Test conditions: (A) control, (B) passive latissimus dorsi tensioning and (C) active latissimus dorsi tensioning.

Table 1
Means, standard deviations (SD), intraclass correlation coefficients ($ICC_{3,1}$) and standard errors of measurement (SEM) of the dependent variables: hip resting position (RP), stiffness before the hip RP and stiffness beyond the hip RP.

	Conditions of latissimus dorsi tensioning								
	Control			Passive LD tensioning			Active LD tensioning		
Dependent variables	Mean ± SD	ICC	SEM	Mean ± SD	ICC	SEM	Mean ± SD	ICC	SEM
Hip RP	−5.60 ± 3.34	0.99	0.33	−6.37 ± 3.30*	0.99	0.33	−8.35 ± 4.21*	0.96	0.84
Stiffness before the hip RP	7.56 ± 1.97	0.91	0.59	7.58 ± 1.80	0.93	0.48	8.69 ± 3.22*	0.95	0.72
Stiffness beyond the hip RP	8.10 ± 2.44	0.99	0.24	8.07 ± 2.40	0.96	0.48	9.12 ± 2.74*	0.97	0.47

Negative values of RP indicate positions of lateral hip rotation.
Means, SDs and SEMs of the RPs are shown in degrees (°).
Means, SDs and SEMs of the values for stiffness before and beyond the RP are shown in units of Newton-meter per radian (Nm/rad).
* Significantly different from control condition ($p \leq 0.05$).

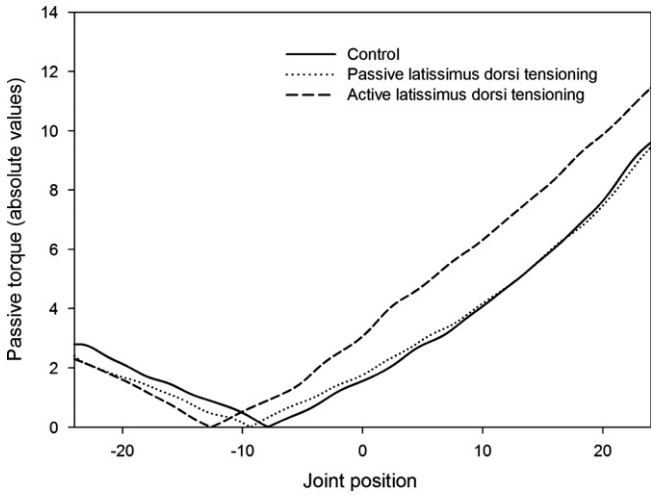

Fig. 4. Curves of net passive torque (absolute values) for each hip joint position. These curves show actual values of the passive torque measured during one repetition of hip medial rotation in each test condition and represent a typical behavior observed with LD tensioning.

Monti et al., 1999; Street and Ramsey, 1965). Because of the continuity between endomysium, perimysium and epimysium (Purslow, 2010), part of the force produced by the LD contraction was transmitted through the connective tissue in parallel to the muscle. Similarly, the LD stretching increased the tension of the muscle's connective tissue network. The forces that reached the epimysium due to LD contraction or stretching were transmitted via the connective tissue continuity to the TLF and GMax, pulling them upwards. The functional relationship between the GMax and the hip joint was responsible for the observed effects after LD tensioning.

The results of the present study support the findings of Vleeming et al. (1995) and Barker et al. (2004), which have demonstrated force transmission from the LD to the TLF in cadavers. Although force transmission from the LD to the GMax had not been investigated previously, in vivo studies have demonstrated myofascial force transmission between the gastrocnemius and soleus muscles (Bojsen-Moller et al., 2010; Huijing et al., 2011), and between the flexor carpi ulnaris and other wrist flexors (Bruin et al., 2011). Thus, the results of the present study reinforce earlier findings of in vivo myofascial force transmission.

Our results challenge the argument provided by some authors that forces generated by muscles during physiological movements are unable to act on adjacent muscles (Herbert et al., 2008; Maas and Sandercock, 2008). Maas and Sandercock (2008) proposed that myofascial force transmission between the soleus muscle of cats and its synergists occur only in situations in which the tissues underwent non-physiological lengthening. However, myofascial force transmission to the hip joint does occur during physiological lengthening or contraction of the LD muscle. The results also challenge the assumption that only an insignificant amount of LD force could act on the lumbopelvic region (Bogduk et al., 1998). The biomechanical model on which this assumption was formulated considered only myotendinous force propagation, and consequently, the possibility of myofascial force transmission between the LD and TLF was disregarded. The different pathways for force transmission considered may explain the discrepancies with our results. However, Bogduk et al. (1998) stated that LD action would not affect the lumbopelvic region even if the model used had considered the force propagation through the TLF. Our results demonstrated that disregarding the myofascial force transmission underestimates the capacity of the LD act on the hip and lumbopelvic region.

As predicted, passive and active LD tensioning shifted the hip RP towards lateral rotation. This finding indicated that changes in GMax tension, as a consequence of LD tensioning, were sufficient to increase the resistance torque at each hip joint position. Note that the LD EMG level produced during active tensioning (approximately 30% of the MVIC) was lower than the LD EMG level that is observed during certain activities, such as the overhead football throw (65–72% of the MVIC) (Kelly et al., 2002) and push-up exercises (117–130% of the MVIC) (Youdas et al., 2010). The higher levels of LD tensioning observed during these activities when compared to the levels observed in the present study would likely result in a stronger pull of the GMax and a more pronounced effects on the hip than we reported.

Passive LD tensioning did not affect the hip stiffness. This result is possible only if the opposing torques acting at joint were perfectly linear (Souza et al., 2009; Winters et al., 1988). However, the passive tension-length curves of soft tissues have a non-linear pattern (Fung, 1967; Taylor et al., 1990). Thus, the lack of significant changes in hip stiffness may be explained by the existence of relatively linear portions along the passive tension-length curves of soft tissues (Fung, 1967; Taylor et al., 1990). Small increases in GMax tension would cause small shifts in the curve produced by this muscle, within a range of joint positions where the opposing torques are relatively linear. Consequently, there would be no significant effects on hip stiffness during the passive condition, as was observed in the present study.

According to traditional biomechanical models (Panjabi, 1992,2003), which do not assume that the joint's antagonistic structures simultaneously produce torques in opposite directions, the increase in GMax tension would increase passive hip stiffness solely at positions beyond the RP, suggesting the presence of increased resistance against medial hip rotation, only. However,

the stiffness increase in both before and beyond RP supports our use of a prestressed model, in which the torque registered during hip medial rotation represented a net curve originated from torques produced simultaneously by hip antagonistic structures. Thus, our results indicated that the structures surrounding the hip are, in fact, prestressed.

Despite the consistency in the responses to LD tensioning, an analysis of individual results from the volunteers revealed a relatively large variability in the magnitude of these responses. The greatest shift in the hip RP was 10.83°. In contrast, some participants demonstrated shifts of less than 1°, suggesting that individual characteristics may influence the degree of myofascial force transmission. Studies have indicated that the degree of myofascial force transmission between the extensor digitorum longus and extensor hallucis longus muscles of rats is determined by the level of stiffness of the connective tissue surrounding these muscles (Huijing and Baan, 2003; Yucesoy et al., 2005). Future investigations should verify if the magnitude of force transmission between the LD and GMax is associated with the magnitude of the TLF stiffness.

The present study demonstrated that the hip RP and passive stiffness can be influenced by the stretching and contraction of an anatomically distant muscle. Although it was not possible to chart the exact pathway of force transmission, the anatomic connections between the LD and GMax (Barker and Briggs, 1999) suggest that the TLF was likely responsible for most of the force transmission. The confirmation that forces can be transmitted along connective tissue and under physiological conditions challenges the traditional assumption that muscular force is transmitted to bones, only, through a myotendinous junction (Huijing et al., 2011). Consequently, biomechanical models that do not account for myofascial force transmission may misinterpret the true degree of biomechanical complexity.

Conflict of interest statement

The authors affirm that there is no conflict of interest regarding the publication of this manuscript. We had no financial or personal relationships with people or organizations that could inappropriately influence or bias our work.

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