Available online at www.sciencedirect.com
ScienceDirect
Journal of Electromyography and Kinesiology 17 (2007) 698–707
JOURNAL OF ELECTROMYOGRAPHY AND KINESIOLOGY
www.elsevier.com/locate/jelekin

Myofascial force transmission between antagonistic rat lower limb muscles: Effects of single muscle or muscle group lengthening

Hanneke J.M. Meijer [a,*], Josina M. Rijkelijkhuizen [a], Peter A. Huijing [a,b]

[a] *Instituut voor Fundamentele en Klinische Bewegingswetenschappen, Faculteit Bewegingswetenschappen, Vrije Universiteit, van der Boechorststraat 9, 1081 BT, Amsterdam, The Netherlands*

[b] *Integrated Biomechanical Engineering for Restoration of Human Function, Instituut voor Biomedische Technologie, Faculteit Constructieve Wetenschappen, Universiteit Twente, Enschede, The Netherlands*

Abstract

Effects of lengthening of the whole group of anterior crural muscles (tibialis anterior and extensor hallucis longus muscles (TA + EHL) and extensor digitorum longus (EDL)) on myofascial interaction between synergistic EDL and TA + EHL muscles, and on myofascial force transmission between anterior crural and antagonistic peroneal muscles, were investigated. All muscles were either passive or maximally active. Peroneal muscles were kept at a constant muscle tendon complex length. Either EDL or all anterior crural muscles were lengthened so that effects of lengthening of TA + EHL could be analyzed. For both lengthening conditions, a significant difference in proximally and distally measured EDL passive and active forces, indicative of epimuscular myofascial force transmission, was present. However, added lengthening of TA + EHL significantly affected the magnitude of the active and passive load exerted on EDL. For the active condition, the direction of the epimuscular load on EDL was affected; at all muscle lengths a proximally directed load was exerted on EDL, which decreased at higher muscle lengths. Lengthening of anterior crural muscles caused a 26% decrease in peroneal active force.

Extramuscular myofascial connections are thought to be the major contributor to the EDL proximo-distal active force difference. For antagonistic peroneal complex, the added distal lengthening of a synergistic muscle increases the effects of extramuscular myofascial force transmission.

Keywords: Myofascial force transmission; Extensor digitorum longus muscle; Tibialis anterior muscle; Peroneal muscles; Connective tissue; Antagonistic muscles

1. Introduction

In the last decade, studies have shown that morphologically defined muscles are not independent actuators, but are capable of mechanical interaction via their connective tissue structures (Huijing and Baan, 2001a; Huijing et al., 1998; Maas et al., 2001; Smeulders et al., 2002; Yucesoy et al., 2003c). The concept of myofascial force transmission is based on the ability to transmit forces between muscles fibres and fascial connective tissues. Chains of molecules, including, trans-sarcolemmal molecules enable forces, generated by sarcomeres within a muscle fibre, to be transmitted via the basal lamina onto the surrounding endomysium and from there onto other intramuscular connective tissue structures (Street, 1983; Tidball, 1991; Trotter and Purslow, 1992). The intramuscular stromata of adjacent muscles are continuous. Via those connections, muscle force can be transmitted from a muscle, without passing, and thus parallel to, the muscle tendon. Consequently, sarcomeres are not only loaded by forces via the myotendinous pathways and sarcomeres in series, but also by forces transmitted by myofascial connections. Note that if the force is transmitted from muscle via its epimysium, force transmission is referred

* Corresponding author. Tel.: +31 20 5988252; fax: +31 20 5988529.
E-mail address: h.meijer@fbw.vu.nl (H.J.M. Meijer).

1050-6411/$ - see front matter © 2007 Elsevier Ltd. All rights reserved.
doi:10.1016/j.jelekin.2007.02.006

to as epimuscular myofascial force transmission (Huijing and Baan, 2001a; Huijing et al., 1998). For a muscle within its natural context of connective tissue, it has been shown that force measured at the origin of a muscle is not necessarily equal to the force exerted at its insertion. With the transmission of forces via epimuscular myofascial connections, additional loads act on the muscle. A difference in forces measured at the muscles' origin and insertion is an unequivocal indication for net epimuscular myofascial force transmission (Huijing and Baan, 2001b). As epimuscular myofascial force transmission is mediated by connective tissues, the fraction of force transmitted myofascially has been found to depend on muscle length as well as the muscle's position relative to its surrounding structures (Huijing and Baan, 2003; Maas et al., 2003a; Maas et al., 2003b).

So far, experimental studies in which only the muscle-tendon complex length of one muscle was changed have demonstrated the importance of myofascial force transmission between synergistic muscles (Maas et al., 2001; Maas et al., 2005) as well as antagonistic muscles (Huijing, 2002; Huijing and Jaspers, 2005). Although this unequivocally shows the mechanical interaction between muscles via myofascial pathways, such experimental conditions differ from the *in vivo* conditions. Besides for pathological conditions such as cramp or cerebral palsy, see other articles within this issue (Huijing, 2007; Malaiya et al., 2007; Smeulders and Kreulen, 2007; Yucesoy and Huijing, 2007) *in vivo* movement involves simultaneous lengthening of a group of synergistic muscles. Such movement will yield big changes in relative position between antagonistic muscles. Since relative position of muscles is an important determinant of myofascial force transmission (Maas et al., 2004), effects on transmission between antagonistic muscles may be expected. The aim of this paper is twofold: (1) to investigate the effect of lengthening of the whole anterior crural muscle group and test whether the added lengthening of tibialis anterior and extensor hallucis longus muscles (TA + EHL) alters myofascial interaction between synergistic extensor digitorum longus (EDL) and TA + EHL muscles, compared to myofascial force transmission for exclusive lengthening of EDL, and (2) to asses the effects of added lengthening of TA + EHL on the myofascial force transmission between anterior crural and antagonistic peroneal muscles.

2. Methods

Surgical and experimental procedures were in agreement with the guidelines and regulations concerning animal welfare and experimentation set forth by Dutch law, and approved by the Committee on Ethics of Animal Experimentation at the Vrije Universiteit. Immediately after all experiments, animals were killed using an overdose of urethane solution, and double-sided pneumothorax was performed.

2.1. Surgical procedures

Male Wistar rats (for lengthening of EDL $n = 7$, for added lengthening of TA + EHL, $n = 6$, with mean body masses of 303.4 (S.D. 11.80) and 302.6 gr. (S.D. 16.2)) were anaesthetized using intraperitoneally injected urethane solution (1.5 g kg^{-1} body mass, 12.5% urethane solution). Extra doses were given if necessary (maximally 1.5 ml). During surgery and data collection, the animals were placed on a heated water pad of approximately 37 °C to prevent hypothermia. The skin and the biceps femoris muscle of the left hind limb were removed, exposing the anterior crural compartment which confines extensor digitorum longus (EDL), extensor hallucis longus (EHL) and tibialis anterior (TA) muscles (Fig. 1). Connective tissue near the muscle bellies within the anterior crural compartment was left intact. Only limited distal fasciotomy was performed to severe the retinaculae (i.e. the transverse crural ligament and the crural cruciate ligament), and subsequently dissect the distal tendons of EDL, EHL and TA. For the added lengthening of TA + EHL, the original position of TA + EHL relative to the EDL in the reference position (i.e. corresponding to a knee angle of 110° and ankle angle of 180° plantar flexion) was preserved by aligning the markers on the distal TA, EHL and EDL tendons. In both experiments, the four distal tendons of EDL were tied together at the reference position using polyester yarn. After uniting, these tendons were severed distally to the knot. The distal tendons of TA and EHL muscles as well as the distal tendons of peroneal muscles were tied together and subsequently severed, and will be referred to as TA + EHL and peroneal complex, respectively. The position of the proximal EDL tendon in the reference position (knee angle of 110°) was marked with a small pin on the epicondylus lateralis of the femur, before cutting a small piece of the bone with the proximal attachment of the EDL muscle. All tendons were connected to metal rods using 100% polyester yarn. The sciatic nerve was dissected free from the upper limb muscles and severed as proximally as possible. The foot was firmly fixed to a plastic plate.

2.2. Experimental set-up

The rat was placed on a heated platform (37 °C) to prevent hypothermia, with the femur clamped to ensure a knee angle of 110°. The foot, attached to the plate, was firmly fixed into a rigid frame with the ankle in extreme plantar flexion (180°), with the metal rods passing over and under the foot (Fig. 2). All tendons were connected to force transducers (BLH Electronics Inc., Canton MA, compliance 16.2 μm N^{-1}, mounted on single-axis micropositioners) by the metal rods, which were aligned with the muscles' line of pull. The sciatic nerve was placed on a pair of silver electrodes and prevented from dehydration by covering it with paper tissue saturated with isotonic saline and which was covered by a thin piece of latex.

2.3. Experimental conditions

Ambient temperature (22 °C ± 0.5) and air humidity (70 ± 2%) were kept constant by a computer-controlled air-conditioning system (Holland Heating, Waalwijk, the Netherlands). Muscle and tendon tissue was further prevented from dehydration by regularly irrigation with isotonic saline. The peroneal complex, deep flexors and triceps surae were kept at a constant muscle tendon complex length. The peroneal complex was set at such a length as to exert an initial force of 5 N. For lengthening of only EDL, TA + EHL was kept at a constant length, set to exert a force of 3 N. The proximal EDL position was set to correspond to the marker on the femur, and was subsequently placed at a length which was 2 mm shorter than the original marker position. In the

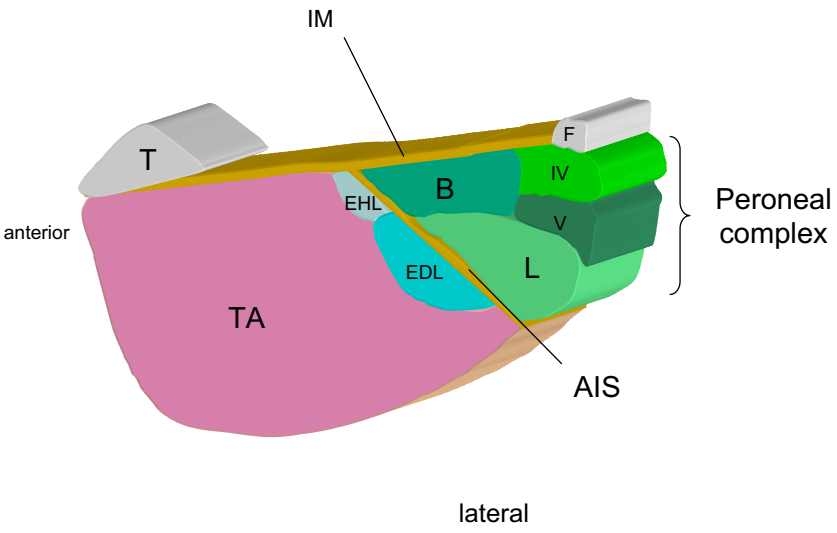


Fig. 1. Schematic representation of a cross-section of the anterior crural and peroneal compartment in the rat lower limb, at approximately the middle of the EDL muscle belly. The anterior crural muscle group consists of tibialis anterior muscle (TA) enveloping extensor digitorum longus (EDL) and extensor hallucis longus (EHL) muscles. The anterior crural compartment is bordered medially by the interosseal membrane (IM) spanning the distance between the tibia (T) and (f) fibula. The peroneal muscle group, separated from the anterior crural compartment by the anterior intermuscular septum (AIS), consists of the peroneus longus (L), peroneus brevis (B), peroneus quarti (IV) and peroneus quinti (V) muscles.

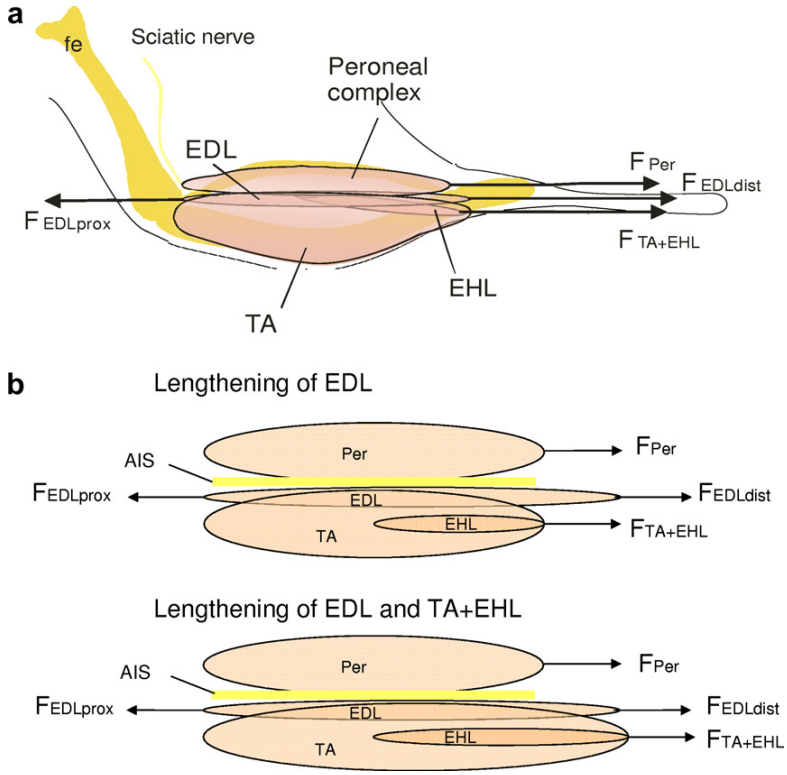


Fig. 2. Schematic representation of the experimental setup and lengthening conditions. (a) Lateral view of the rat lower limb after removal of the skin and biceps femoris muscle. The origin of extensor digitorum longus muscle (EDL) at the lateral condyle of the femur (fe) and the insertions of tibialis anterior (TA), extensor hallucis longus (EHL), EDL and peroneal muscles (Per) on the foot are severed and connected to force transducers (represented by arrows). Both femur and foot were fixed within a rigid frame in the reference position which corresponds to a knee angle of 110° and the ankle at 180° plantar flexion. (b) The two imposed lengthening conditions. Arrows indicate force transducers. For lengthening of EDL exclusively (upper panel), extensor digitorum longus muscle was lengthened distally by 1 mm increments, while the peroneal complex and tibialis anterior and extensor hallucis longus muscles were kept at a constant length. For synergist lengthening (lower panel), EDL and TA + EHL muscles were lengthened distally by 1 mm increments, while the peroneal complex is left at a constant muscle tendon complex length. The peroneal complex is separated from the anterior crural muscles by the anterior intermuscular septum (AIS).

experiments in which TA + EHL was lengthened as well, the marker at the distal EDL tendon was aligned with the marker on the distal TA + EHL tendon. This relative position was maintained during the experiment by moving both distal force transducers equal distances.

Prior to excitation, all muscles were brought to their desired length and position passively by moving the force transducers. Before acquiring data, EDL, and in the added lengthening of TA + EHL experiment also TA + EHL, were preconditioned by isometric contractions at alternating high and low lengths, until forces at low length were reproducible (i.e. effects of previous activity at high length (Huijing and Baan, 2001b) are removed). For the length–force curve, isometric contractions were performed at different EDL and TA + EHL lengths. Both TA + EHL and EDL muscles were lengthened distally with 1 mm increments. Two twitches were evoked, followed by a tetanic contraction at 100 Hz. All muscles studied were activated by stimulation of the sciatic nerve (with sureal and tibial branches denervated for the EDL lengthening experiment) with a constant current (3 mA) and a stimulation frequency of 100 Hz (pulse width 0.1 ms). Timing of stimulation of the nerve and A/D conversion (12-bit A/D converter, sampling frequency 1000 Hz) were controlled by a special purpose microcomputer. After each contraction, the muscles were allowed to recover near active slack length for 2 min. Passive isometric force was measured prior to the tetanic contraction and total force was measured during the tetanic plateau of the muscle force.

3. Data treatment

Passive muscle force was fitted using an exponential curve

$$y = e^{(ax+b)},$$

where y represents passive muscle force, x represents muscle-tendon complex length and 'a' and 'b' are fitting constants. Active EDL muscle force (F_{ma}) was estimated by subtracting the calculated passive force (F_{mp}) using the fitted function, from total force (F_m) for the appropriate muscle length. Active EDL length–force data were then fitted with a stepwise polynomial regression procedure (see statistics). The polynomial

$$y = b_0 + b_1x + b_2x^2 + \ldots .b_nx^n,$$

where y represents active muscle force, x represents active muscle force length and b_0 through b_n are fitting constants. Using the selected polynomials, mean and standard errors of active muscle force were calculated for given EDL lengths. Optimum muscle length was defined for each individual curve as the active muscle length at which the fitted active force curve showed maximum force (F_{mao}). In order to estimate more accurately the distal active slack length, data of muscle length and active muscle force ($F_{ma} < 0.3 \times F_{mao}$) were selected. The data points were then extrapolated with a fitted curve;

$$y = b_0 + b_1x + b_2^{\exp(x)}.$$

Active slack length was determined by solving this equation.

4. Statistics

In the active muscle force fitting procedure, the curve fitting starts with a first order polynomial and the power was increased up to maximally a sixth order, as long as this yields a significant improvement to the description of the length-active force data, as determined by one-way analysis of variance (ANOVA)(Neter et al., 1990). Two-way ANOVA's for repeated measurements were used to test for the effects of EDL muscle-tendon complex length and location of force measurement on EDL forces, to test for effects of EDL muscle-tendon complex length and lengthening condition (lengthening of EDL or synergistic lengthening of TA + EHL and EDL) on distal and proximal EDL forces, the proximo-distal force differences (for the interval $-7 \leqslant \Delta l_{m+t} \leqslant 3$) and peroneal active forces. One-way ANOVA's were used to test for the effect of EDL muscle-tendon complex length on TA + EHL as well as peroneal forces, and to test for differences between distally and proximally assessed optimum muscle lengths. Independent samples T-tests were performed to test for differences in active slack length and distal and proximal optimum muscle lengths between EDL lengthening and the added lengthening of TA + EHL. If significant effects were found, post hoc tests were performed using the Bonferroni procedure for multiple pair wise comparisons to locate differences. Main and interaction effects as well as differences were considered significant at $P < 0.05$. Values are plotted mean + SE, and muscle length is expressed as a deviation of distal EDL optimum muscle length.

5. Results

5.1. Effects of EDL lengthening and lengthening of synergists on length–force characteristics of EDL forces

5.1.1. Distal EDL forces

For distal EDL active forces (Fig. 3a), ANOVA showed significant effects of EDL length and lengthening condition, as well as interaction. At the ascending limb of the length–force curve, EDL distal active force during EDL lengthening is higher than for the added lengthening of TA + EHL. Optimal forces for both conditions are equal (2.4 N), and are similar for lengths over optimum muscle length. The length range of distal EDL active force exertion for the added lengthening of TA + EHL is smaller than for lengthening of EDL exclusively, as active slack length differed significantly (−8.3 mm and −10.7 mm for EDL + TA + EHL and EDL, respectively).

For distal EDL passive forces, ANOVA also showed significant effects of muscle-tendon complex length and lengthening condition, as well as interaction. Below optimum muscle length, lengthening of synergistic muscles resulted in a lower distal passive force than lengthening of EDL. Passive forces increased exponentially with muscle-tendon complex length. Note that for both conditions,

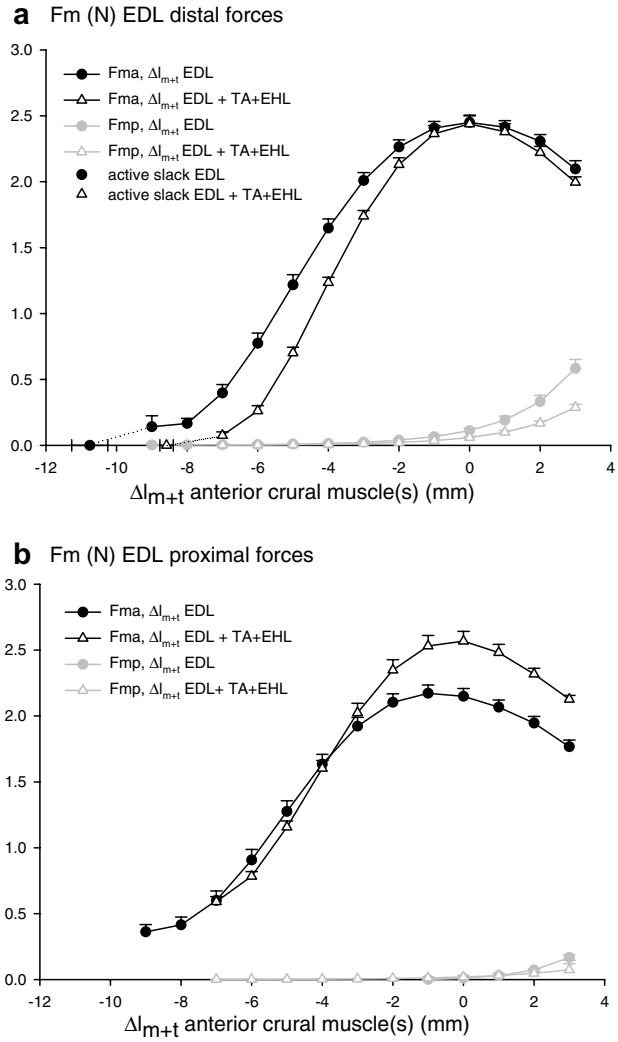


Fig. 3. Proximal and distal active and passive EDL forces. (a) Length–force characteristics of distally measured EDL forces as a function of distal EDL muscle tendon complex length or muscle tendon complex length of EDL + TA + EHL. (b) Length–force characteristics of proximally measured forces of EDL as a function of distal EDL muscle tendon complex length or muscle tendon complex length of EDL + TA + EHL. (All values are shown as means + SE, $n = 7$ for lengthening of EDL, $n = 6$ for the added lengthening of TA + EHL).

distal passive force starts to increase from well below distal optimum muscle length.

It is concluded that the added lengthening of synergistic TA + EHL causes distal EDL length–force characteristics to be different from those during distal lengthening of EDL exclusively.

5.1.2. Proximal EDL forces

ANOVA showed significant effects of muscle-tendon complex length of EDL and lengthening condition on EDL proximal active forces (Fig. 3b), as well as an interaction between these factors. ANOVA also showed a significant difference in proximal optimum muscle lengths for these conditions. At low muscle lengths ($\Delta l_{m+t} \leqslant -4$), proximal active forces do not differ for EDL and synergistic muscles. At higher muscle lengths ($-3 \leqslant \Delta l_{m+t}$), proximal active force for lengthening of synergistic muscles is higher than proximal active force measured during lengthening of EDL exclusively. Proximal passive forces increased significantly and exponentially as a function of EDL muscle length, but passive forces did not differ significantly for the two conditions.

In conclusion, also for proximal active forces, the added lengthening of TA + EHL alters the EDL active length–force characteristics compared to lengthening of EDL only.

5.1.3. Effects of the added lengthening of TA + EHL on proximo-distal EDL force differences

Differences between proximally and distally exerted EDL forces were found for both lengthening of EDL exclusively and lengthening of synergistic muscles (Fig. 4). Such differences indicate that EDL is loaded also by myofascial loads. Note that a negative value for this difference indicates that EDL is loaded myofascially in distal direction. The additional load is resisted by EDL active force and in this case the load is integrated into the force exerted at the proximal tendon. Conversely, a positive proximo-distal active force difference represents myofascial loading of EDL in proximal direction and integration of that load in distal EDL active force.

ANOVA showed effects of muscle-tendon complex length and location of lengthening for active and passive EDL force differences, but no interaction was found for active force. The effects of added lengthening of TA + EHL shift down the whole curve describing prox-

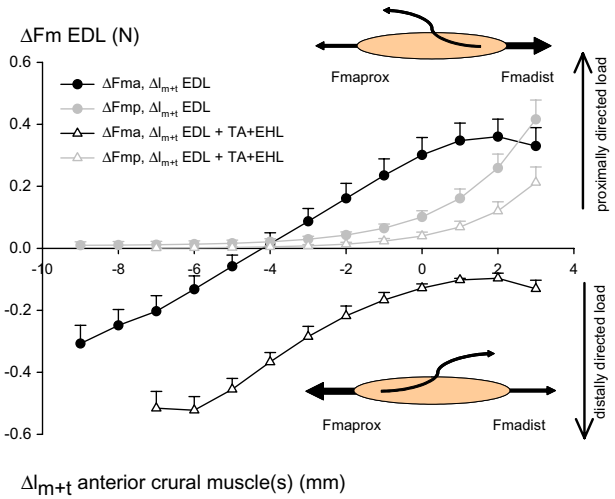


Fig. 4. EDL proximo-distal force differences in the two lengthening conditions. Active (ΔF_{ma}) and passive (ΔF_{mp}) force differences (F_{dist}–F_{prox}) are shown as means + SE ($n = 7$ for lengthening of EDL, $n = 6$ for the added lengthening of TA + EHL). Icons indicate direction of the net epimuscular load for positive and negative force differences. Note that an effect of added lengthening of TA + EHL is to remove the change in direction of the epimuscular load.

imo-distal active force difference as a function of length (Fig. 4). As a consequence, the reversal of the direction of loading seen for lengthening of EDL exclusively, is removed as an effect of added TA + EHL lengthening. In this case EDL is loaded myofascially in distal direction at all lengths. At higher lengths, a smaller myofascial load is imposed than at lower lengths (Fig. 4).

In the passive condition, EDL is always loaded myofascially in proximal direction. The added lengthening of TA + EHL does not shift the whole curve, but lowers the magnitude of the load at higher lengths (Fig. 4).

It is concluded that proximo-distal differences in EDL force exists also when a group of synergistic muscles are lengthened. However, the synergistic distal lengthening of TA + EHL and EDL alters the magnitude and direction of myofascial force transmission, especially at high muscle lengths.

5.1.4. TA + EHL forces for the two lengthening conditions

Fig. 5 shows the TA + EHL active and passive length force curve for as a function of the length the anterior crural muscles.

Distal lengthening of EDL exclusively caused a significant decrease in TA + EHL active force (Fig. 5, filled circles), despite the fact that TA + EHL was kept at a constant muscle-tendon complex length. TA + EHL active force decreased by 0.32 N (i.e. by 13%). Part of this decrease in TA + EHL active force should be ascribed to history effects, as control contractions after determining the EDL length–force curve indicate a force decrease of 5.7%. At the set length of TA + EHL, a relatively low muscle length, passive force is negligible, regardless of EDL length.

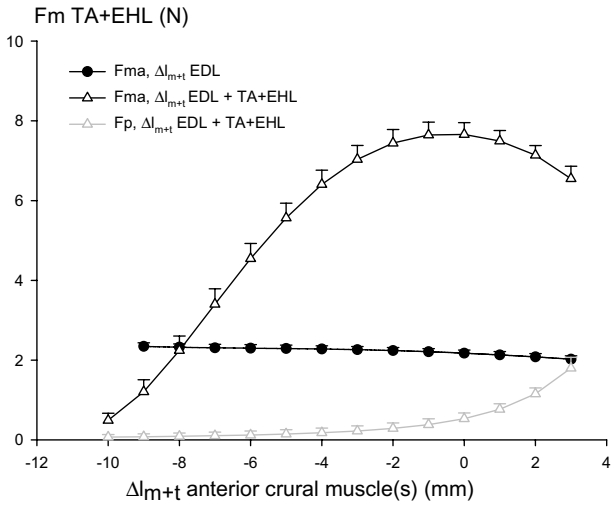


Fig. 5. TA + EHL active and passive forces as a function of EDL + -TA + EHL lengthening. Lengthening was obtained by moving the distal force transducers. As a reference, the decreasing active TA + EHL force with this complex at constant length is shown also. All values are shown as means + SE, $n = 7$ for lengthening of EDL, $n = 6$ for the added lengthening of TA + EHL.

5.1.5. Effects of lengthening of synergistic anterior crural muscles on peroneal force

During distal lengthening of EDL exclusively, peroneal active force (Fig. 6a, filled circles) decreased significantly ($P < 0.05$) despite the fact that peroneal muscles were kept at a constant length. Active force decreased by a relative small amount (maximally 0.25 N or 6%, Fig. 6b, filled circles).

Also after the added lengthening of TA + EHL, Bonferroni post-hoc tests showed a significant effect of EDL + -TA + EHL muscle-tendon complex length on peroneal active force (Fig. 6a, open triangles); peroneal active force decreased significantly, despite the fact that peroneal muscles were kept at constant length. Peroneal active force

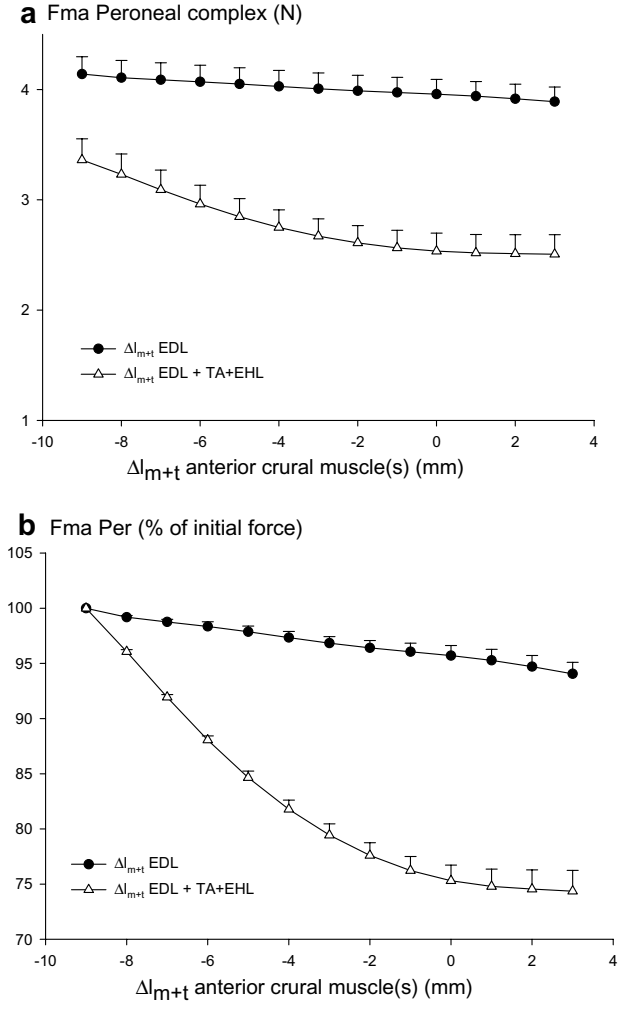


Fig. 6. Effects of anterior crural lengthening condition on antagonistic peroneal forces. (a) Peroneal active force as a function of muscle-tendon complex length of either EDL exclusively or EDL + TA + EHL. (b) Normalized peroneal active force, expressed as percentage of the initial force as a function of muscle-tendon complex length of either EDL exclusively or EDL + TA + EHL. All values are shown as means + SE, $n = 7$ for lengthening of EDL, $n = 6$ for the added lengthening of TA + EHL.

decreased from 3.36 N at $\Delta l_{m+t} = -9$ with 0.87 N to 2.48 N at $\Delta l_{m+t} = 3$. Expressed as a percentage of the initial active force level (Fig. 6b, open triangles), peroneal active force decreases with 26% as EDL + TA + EHL are lengthened by 12 mm.

The above results show that the added distal lengthening of a synergistic muscle has significant effects on active forces exerted by the antagonistic peroneal complex: force decrease in antagonistic peroneal muscles is much enhanced as a consequence of the added lengthening of TA + EHL.

6. Discussion

6.1. Epimuscular myofascial force transmission

6.1.1. Effects of added lengthening of TA + EHL on the EDL proximo-distal force difference

Distal lengthening of the extensor digitorum longus muscle, as well as distal lengthening of synergistic EDL + -TA + EHL within an intact anterior crural compartment results in length-dependent differences in active and passive forces exerted at the proximal and distal EDL tendons (Fig. 3). Such proximo-distal force differences are indicative of epimuscular myofascial force transmission (Huijing and Baan, 2001a; Huijing et al., 1998). The present results are in agreement with previous studies on maximally activated rat extensor digitorum longus muscle within an intact anterior crural compartment (Huijing and Baan, 2003; Maas et al., 2001; Maas et al., 2003a). However, the present study is a novel demonstration of the effects of length of a whole muscle group of synergistic muscles on epimuscular myofascial force transmission between synergistic muscles, as well as antagonistic muscles and the comparison of effects of length of a single muscle. For other new studies involving muscle group lengthening and antagonistic myofascial force transmission see elsewhere within this issue (Huijing, 2007; Rijkelijkhuizen et al., 2007).

Both the magnitude as well as the direction of the net epimuscular force on EDL is affected after the added lengthening of TA + EHL (Fig. 4). Whereas for lengthening of EDL exclusively the proximo-distal active force difference reverses from a negative value at low muscle lengths to a positive value at high muscle lengths. The added lengthening of TA + EHL shifts the whole curve downwards, resulting in a negative active force difference that decreases with increasing lengths. According to our sign definitions, for the added lengthening of TA + EHL a net epimuscular load in distal direction is now exerted on active EDL at all muscle lengths. After lengthening of EDL exclusively, this distally directed load is only found at lower lengths: the sign of the proximo-distal active force difference reverses at higher muscle lengths. This is ascribed to changes in relative position between TA + EHL and EDL occurring during EDL lengthening and concomitant length and stiffness changes in their intermuscular connections. It should be noted that, after the added lengthening of TA + EHL, such changes in relative position between TA + EHL and EDL are absent and as a result, a reversal in the direction of the epimuscular force is prevented.

Note that, in contrast to the EDL active force differences after added lengthening of TA + EHL, a positive passive proximo-distal force difference for EDL is found at high muscle lengths. This indicates that for the passive state of the muscles, the net epimuscular load on EDL is exerted in proximal direction, but that it changes into a net distally directed epimuscular force upon activation of the muscles.

The dominance of proximal active EDL force at low muscle tendon complex lengths for both lengthening conditions shows that even at low EDL forces, epimuscular connections are sufficiently stiff to allow myofascial loading of the proximal EDL segment. Such 'prestrained' epimuscular connections ((Yucesoy et al., 2005), see also (Yucesoy and Huijing, 2007)) were found for EDL at a reference length (i.e. a length corresponding to a knee angle of 100° and an ankle angle of 90°), as well as for proximal lengthening of EDL. It is concluded that myofascial force transmission between EDL and TA + EHL is present for lengthening of EDL as well as for the added lengthening of TA + EHL. However, magnitude and direction of the net epimuscular force have changed as a result of the added lengthening of TA + EHL.

6.1.2. Effects anterior crural muscle group length on peroneal active force

After distal lengthening of EDL exclusively, as well as after the added lengthening of TA + EHL, peroneal active force is always decreased significantly, despite the fact that this muscle group is kept at a constant muscle-tendon complex length. For lengthening of EDL exclusively, a decrease in peroneal active force was also reported by Huijing and Huijing & Jaspers in preliminary reports (2002, 2005). In the present work, after the added lengthening of TA + EHL, the decrease in peroneal active force is almost 25%. Note that EDL and TA + EHL are located within the anterior crural compartment and separated from the peroneal compartment by the anterior intermuscular septum (Fig. 1). The only connections between these muscle groups are myofascial ones, and therefore, the effect of lengthening anterior crural muscles on the peroneal active force is mediated by extramuscular myofascial force transmission, i.e. force transmission via non-muscular structures. For lengthening of EDL, the decrease in peroneal force is accompanied by an increase in distal EDL active force, which suggests that part of the EDL active force exerted at the distal tendon originates from the peroneal muscles. For the added lengthening of TA + EHL, it is hypothesized that the force exerted at the epimuscular connections between peroneal and TA + EHL muscles is increased, and therefore, more force will be transmitted from peroneal muscles onto TA + EHL muscles via this path. As a consequence, the force transmitted by epimuscular connections between peroneal muscles and the EDL is smaller, as this

pathway is then comparatively less stiff. At higher muscle lengths, the added lengthening of TA + EHL reduces the epimuscular force transmission onto EDL (Fig. 4).

For the conditions of our present study, the added lengthening of TA + EHL significantly increases myofascially transmitted forces between antagonists. Therefore, for interaction between antagonists, muscle size (e.g. cross sectional area) is one determinant of the magnitude of epimuscular myofascial force transmission (Yucesoy et al., 2005).

6.2. Pathways of myofascial force transmission

Epimuscular myofascial force transmission is mediated by connective tissue structures surrounding the muscles, and is therefore susceptible to changes in muscle relative position as well as muscle length (Huijing and Baan, 2003). Potential epimuscular connections are (1) direct connections between the intramuscular stromata of two adjacent muscles. Force transmitted via such pathways is referred to as intermuscular myofascial force transmission (Huijing and Baan, 2001a). Within the anterior crural compartment, intermuscular myofascial force transmission is known to occur at the EDL + TA + EHL muscle-belly interface (Maas et al., 2001; Maas et al., 2004). A recent study by Maas et al. (2005) reported that after compartmental fasciotomy and subsequent blunt dissection, distal lengthening of TA + EHL resulted in a significant EDL proximo-distal active force difference, despite the fact that EDL was kept at a constant length. These results suggest that conditions exist in which the contribution of intermuscular myofascial connections to the proximo-distal force difference is small. In our current study, changes in relative position of intermuscular myofascial connections between EDL and TA + EHL are absent, and the added lengthening of TA + EHL creates different conditions for these intermuscular connections, compared to exclusive lengthening of EDL. Therefore, it is hypothesized that the contribution of the intermuscular connections to the proximo-distal EDL active force difference during added lengthening of TA + EHL is small.

When force is transmitted between the muscle and (2) connections between the intramuscular stromata and extramuscular connective tissue structures, such as connective tissues reinforcing nerves and blood vessels, intermuscular septa, the interosseal membrane, compartmental fascia and epitendinous tissues (Rijkelijkhuizen et al., 2005), we refer to it as extramuscular myofascial force transmission (Huijing and Baan, 2001b). Previous studies (Huijing and Baan, 2001b; Maas et al., 2003a; Rijkelijkhuizen et al., 2005; Yucesoy et al., 2003a) have shown that the most likely candidate for extramuscular myofascial force transmission in the rat lower limb is the neurovascular tract, a collagen reinforced complex supporting the nerves as well as blood and lymph vessels, see also discussion and images elsewhere in the present journal issue (Huijing, 2007). The neurovascular tract passes between the peroneal compartment and the anterior crural compartment via a fenestration in the anterior intermuscular septum (Huijing and Baan, 2001a) and branches within the anterior crural compartment (Maas et al., 2001). Note that the rather stiff connective fasciae are anchored to epimysia and compartmental walls and allow forces to be transmitted (Yucesoy et al., 2003b). In addition to the neurovascular tract, an indirect extramuscular linkage of anterior crural muscles with peroneal muscles exists because of the connections of both the anterior crural muscles' and peroneal muscles' intramuscular stromata to the anterior intermuscular septum (Huijing, 2002).

For distal lengthening of EDL exclusively, it is hypothesized that the EDL branch of the neurovascular tract lengthens and consequently increases in stiffness, thereby exerting a proximally directed load on to EDL particularly at low lengths. This load is integrated in the force exerted at the distal EDL tendon and results in a higher distal than proximal EDL active force. The added lengthening of TA + EHL is expected to increase the total loading of the neurovascular tract, thereby increasing the stiffness of the extramuscular pathway between TA + EHL and peroneal muscles. More force (maximally 26%, Fig. 6b) is then transmitted from the peroneal muscles via the neurovascular tract onto the anterior crural compartment. However, such an increased proximal load exerted on EDL fails to explain a (albeit decreasing) negative EDL active force difference for the added lengthening of TA + EHL. Note that the proximo-distal force differences represent the net epimuscular load exerted on EDL. This means that a net negative EDL proximo-distal active force difference can exist in the simultaneous presence of a high proximally directed as well as a slightly higher distally directed load. One likely myofascial structure to impose such a distally directed load is the fasciae covering the distal segment of the TA muscle belly and tendons (Fig. 7). These fasciae, consisting of general fascia and the epimysia of biceps femoris muscle, are attached medially onto the tibial crest and run laterally over the TA muscle belly and its epimysium, and continue towards and are connected to the antagonistic compartment walls. At low TA lengths, these distal fasciae are lengthened and exert a distally directed load on TA. As EDL is connected to TA + EHL via epimuscular myofascial connections, a fraction of this distally directed load is also exerted onto EDL, resulting in a negative active force difference. Lengthening of TA + EHL shortens the distal fasciae and decreases the distal load exerted on EDL. For passive muscles at any given length, this distal load is smaller than during the active state, as the displacement of the active muscle belly lengthens the distal fasciae and increases their stiffness. Therefore, it is possible for a net proximally directed myofascial load to be exerted onto passive EDL, while a net distally directed load is exerted upon activation.

It is concluded that different pathways of extramuscular myofascial force transmission are major determinants of the EDL proximo-distal active force difference. Both the neurovascular tract and the distal fasciae are hypothesized

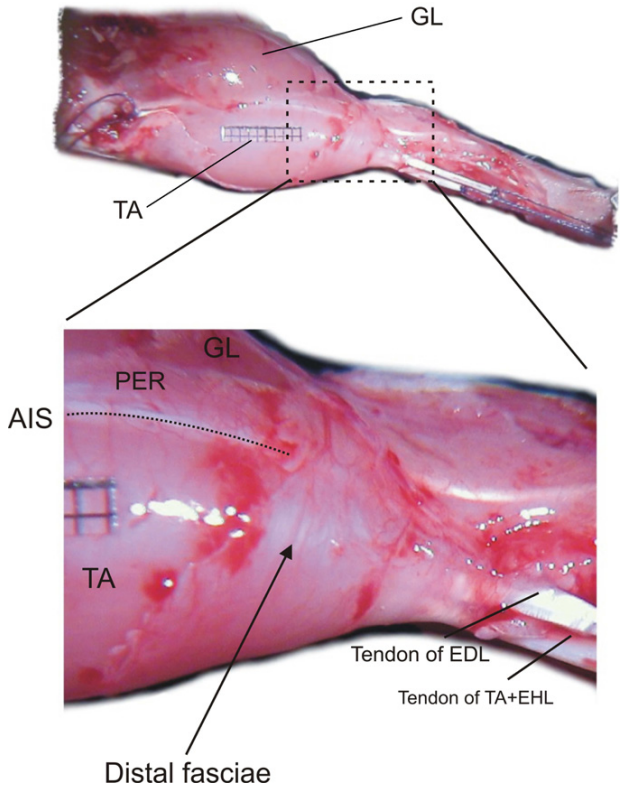


Fig. 7. Lateral view of the rat lower leg after removal of the skin and biceps femoris muscle showing the distal fasciae. The distal EDL and TA + EHL tendons as well as the proximal origin of the EDL are cut from their attachment and connected to Kevlar threats. Note that the distal fasciae run from the tibial crest over the TA muscle belly towards the peroneal and triceps surae compartments. Via these fasciae, it is thought that a distally directed force is exerted onto the anterior crural muscles. TA indicates the tibialis anterior muscle, GL indicates the lateral gastrocnemius muscle, PER indicates the peroneal muscles, and AIS indicates the anterior intermuscular septum. Scale bar indicates mm.

to be of major importance for extramuscular force transmission between the anterior crural compartment and antagonistic peroneal muscles.

6.3. Effects of myofascial force transmission on sarcomere length distributions

It has been argued that for a muscle with epimuscular myofascial connections, distributions of sarcomere lengths within the muscle fibres are altered (Maas et al., 2003a; Yucesoy et al., 2003a). Myofascially transmitted forces are not exerted equally on all muscle fibres within the muscle and as a consequence, sarcomeres at different locations within the muscle are allowed to shorten to a different degree. This leads to distributions in mean fibre sarcomere lengths, i.e. parallel distribution in sarcomere lengths (Huijing, 1996). Finite element modelling of a muscle with intact myofascial connections supports such concept (Yucesoy et al., 2003a; Yucesoy et al., 2002). A decreased distribution of mean fibre sarcomere lengths is hypothesized to explain the differences in the active length ranges between lengthening of EDL and the added lengthening of TA + EHL (Fig. 3). The fact that a major difference in EDL active length range is found only for distal force may be explained by the fact that distal lengthening of EDL in the higher length range has location specific effects: (1) It enhances the change in relative position of the distal half of the EDL muscle more than of its proximal half, (2) It enhances the change in relative position of the distal segments of EDL more than of the proximal segments of the same EDL fibres. These factors combined have consequences for the magnitude of the parallel distribution of EDL sarcomere lengths (i.e. distribution of mean fibre sarcomere lengths) of the distal fibre segments. On the added lengthening of TA + EHL, the epimuscular load on EDL is distributed more evenly particularly in the distal muscle fibre segments, because the effects of intermuscular relative position are decreased or removed. Simultaneously, also the extramuscular load on TA + EHL is increased which tends to unload EDL.

Note that the added lengthening of TA + EHL causes distal EDL passive force to be higher than proximal passive force, indicating that the passive distal sarcomeres are at higher length than their proximal counter parts. However, this is reversed upon activation of the muscle, as proximal EDL active force is higher than distal active force. A reversal of sarcomere length distribution can only occur when, upon activation, the more proximal sarcomeres within muscle fibres are exposed to higher loading than distal ones. Such enhanced distal loading of TA does occur via the distal fasciae (Fig. 7). As the muscle is activated the muscle belly will shorten from its passive condition, because of elastic effects on the distal tendon, and the distal compartmental fasciae are stretched. It is hypothesized that this distal load is, at least in part, myofascially transmitted onto EDL.

It is concluded that simultaneous lengthening of a group of synergistic muscles increases extramuscular myofascial force transmission between antagonistic muscles. Intermuscular myofascial force transmission between synergists is altered by different muscle relative positions within the compartment. Such major effects should be taken into account when studying muscles within an *in vivo* context.

References

Huijing PA. Important experimental factors for skeletal muscle modelling: non-linear changes of muscle length force characteristics as a function of degree of activity. Eur J Morphol 1996;34(1):47–54.

Huijing PA. Intra-, extra-, and intermuscular myofascial force transmission of synergists and antagonists: effects of muscle length as well as relative position. J Mechan Med Biol 2002;2(3&4):405–19.

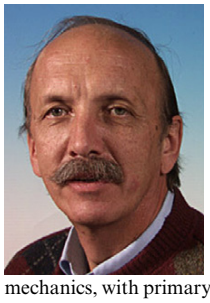
Huijing PA. Epimuscular myofascial force transmission between antagonistic and synergistic muscles can explain movement limitation in spastic paresis. J Electromyogr Kinesiol 2007;17(6):708–24.

Huijing PA, Baan GC. Myofascial force transmission causes interaction between adjacent muscles and connective tissue: effects of blunt dissection and compartmental fasciotomy on length force characteristics of rat extensor digitorum longus muscle. Arch Physiol Biochem 2001a;109(2):97–109.

Huijing PA, Baan GC. Extramuscular myofascial force transmission within the rat anterior tibial compartment: proximo-distal differences in muscle force. Acta Physiol Scand 2001b;173(3):297–311.
Huijing PA, Baan GC. Myofascial force transmission: muscle relative position and length determine agonist and synergist muscle force. J Appl Physiol 2003;94(3):1092–107.
Huijing PA, Jaspers RT. Adaptation of muscle size and myofascial force transmission: a review and some new experimental results. Scand J Med Sci Spor 2005;15(6):349–80.
Huijing PA, Baan GC, Rebel GT. Non-myotendinous force transmission in rat extensor digitorum longus muscle. J Exp Biol 1998;201(5):682–91.
Maas H, Baan GC, Huijing PA. Intermuscular interaction via myofascial force transmission: effects of tibialis anterior and extensor hallucis longus length on force transmission from rat extensor digitorum longus muscle. J Biomech 2001;34(7):927–40.
Maas H, Baan GC, Huijing PA, Yucesoy CA, Koopman BH, Grootenboer HJ. The relative position of EDL muscle affects the length of sarcomeres within muscle fibers: experimental results and finite-element modeling. J Biomechan Eng 2003a;125(5):745–53.
Maas H, Yucesoy CA, Baan GC, Huijing PA. Implications of muscle relative position as a co-determinant of isometric muscle force: a review and some experimental results. J Mechan Med Biol 2003b;3(2):145–68.
Maas H, Baan GC, Huijing PA. Muscle force is determined also by muscle relative position: isolated effects. J Biomechan 2004;37(1):99–110.
Maas H, Meijer HJM, Huijing P. Intermuscular interaction between synergists in rat originates from both intermuscular and extramuscular myofascial force transmission. Cells Tissues Organs 2005;181(1):38–50.
Malaiya R, McNee AE, Fry ELC, Gough M, Shortland AP. The morphology of the medial gastrocnemius in typically developing children and children wiht spastic hemiplegic cerebral palsy. J Electromyogr Kines 2007;17(6):657–63.
Neter J, Wasserman W, Kutner ME. 1990. Applied linear statistic models: regression, analysis of variance and experimental design Homewood, IL:Irwin.
Rijkelijkhuizen JM, Baan GC, de Haan A, de Ruiter CJ, Huijing PA. Extramuscular myofascial force transmission for in situ rat medial gastrocnemius and plantaris muscles in progressive stages of dissection. J Exp Biol 2005;208(Pt 1):129–40.
Rijkelijkhuizen JM, Baan GC, Huijing PA. Myofascial force transmission between antagonistic muscles located in opposite compartments of the rat hindlimb. J Electromyogr Kines 2007;17(6):690–7.
Smeulders MJ, Kreulen M. Myofascial force transmission and tendon transfer for patients suffering from spastic paresis: a review and some new observations. J Electromyogr Kines 2007;17(6):644–56.
Smeulders MJ, Kreulen M, Hage JJ, Baan GC, Huijing PA. Progressive surgical dissection for tendon transposition affects length–force characteristics of rat flexor carpi ulnaris muscle. J Orthopaed Res 2002;20(4):863–8.
Street SF. Lateral transmission of tension in frog myofibers: a myofibrillar network and transverse cytoskeletal connections are possible transmitters. J Cell Physiol 1983;114(3):346–64.
Tidball JG. Force transmission across muscle cell membranes. J Biomech 1991;24(Suppl. 1):43–52.
Trotter JA, Purslow PP. Functional morphology of the endomysium in series fibered muscles. J Morphol 1992;212(2):109–22.
Yucesoy CA, Huijing PA. Substantial effects of epimuscular myofascial force transmission on muscular mechanics have major implications on spastic muscle and remedial surgery. J Electromyogr Kines 2007;17(6):664–79.
Yucesoy CA, Koopman BH, Huijing PA, Grootenboer HJ. Three-dimensional finite element modeling of skeletal muscle using a two-domain approach: linked fiber-matrix mesh model. J Biomechan 2002;35(9):1253–62.
Yucesoy CA, Koopman BH, Baan GC, Grootenboer HJ, Huijing PA. Effects of inter- and extramuscular myofascial force transmission on adjacent synergistic muscles: assessment by experiments and finite-element modeling. J Biomechan 2003a;36(12):1797–811.
Yucesoy CA, Koopman BHFJM, Baan GC, Grootenboer HJ, Huijing PA. Extramuscular myofascial force transmission: experiments and finite element modeling. Arch Physiol Biochem 2003b;111(4):377–88.
Yucesoy CA, Koopman BHFJM, Baan GC, Grootenboer HJ, Huijing PA. Inter- and Extramuscular Myofascial Force Transmission Function as a Major Determinant of Muscle Force and Length Range of Force Exertion. Dunedin, New Zealand: International Society of Biomechanics XIXth Congress; 2003c.
Yucesoy CA, Baan GC, Koopman BHFJM, Grootenboer HJ, Huijing PA. Pre-strained epimuscular connections cause muscular myofascial force transmission to affect properties of synergistic EHL and EDL muscles of the rat. J Biomechan Eng 2005;127(5):819–28.

Josina M. Rijkelijkhuizen was granted in 1999 the degree of Doctorandus in Human Movement Sciences (i.e. Drs, which is equivalent to having passed the preliminary oral examinations of a Ph.D. program) at the Vrije Universiteit in Amsterdam, the Netherlands. After working in the field of occupational health for a year, she returned to the same Faculty to work on her PhD within the Institute for "Fundamentele en Klinische Bewegingswetenschappen". In 2005, she was granted her PhD, for work on muscle physiology. Currently, she is doing epidemiological research with respect to diabetes at the Institute for Research in Extramural Medicine at the Vrije Universiteit Medical Center in Amsterdam. Her research interests include muscle physiology and diabetes (with a special interest in beta cell function and cardiovascular risk factors).

Hanneke J.M. Meijer graduated in 2003 with the degree of Doctorandus (i.e. Drs, which is equivalent to having passed the preliminary oral examinations of a Ph.D. program) in Evolutionary Anatomy from the University of Leiden, the Netherlands. She is presently pursuing a Ph.D. in Human Movement Sciences, within the Institute for "Fundamentele en Klinische Bewegingswetenschappen" at the Vrije Universiteit in Amsterdam the Netherlands, with work in muscle mechanics. For this she investigates experimentally the physiological relevance of mechanical interaction between muscles and connective tissues via myofascial force transmission. Her research interests include skeletal muscle functioning, functional morphology and vertebrate paleontology.



Peter A. Huijing holds a degree in Physical Education from the Academy of Physical Education in Amsterdam the Netherlands, as well as a PhD. in Physiology from the University of Minnesota, Minneapolis, USA. He has a dual appointment at the Faculteit Bewegings- wetenschappen of the Vrije Universiteit, Amsterdam and the department of Biomechanical Engineering at Twente University at Enschede, The Netherlands.
He has worked for more than 30 years at the interface of muscle physiology, anatomy and biomechanics, with primary research interests in fundamental aspects of form–function relation of muscles, force transmission from muscle as well as processes of adaptation ranging from the molecular level to whole muscle. Biomechanical modeling has always played an important role in addition to his experimental work. In recent years his work has been applied particularly within the fields of surgery of the locomotor system and rehabilitation. He is the 2007 laureate of the Muybridge Award of the International Society of Biomechanics for career achievements in biomechanics.