



HAL
open science

Between-muscle differences in coactivation assessed using elastography

Simon Avrillon, François Hug, Gaël Guilhem

► **To cite this version:**

Simon Avrillon, François Hug, Gaël Guilhem. Between-muscle differences in coactivation assessed using elastography. *Journal of Electromyography and Kinesiology*, 2018, 43, pp.88-94. 10.1016/j.jelekin.2018.09.007 . hal-03329517

HAL Id: hal-03329517

<https://nantes-universite.hal.science/hal-03329517>

Submitted on 1 Sep 2021

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

1 **Between-muscle differences in coactivation assessed using elastography**

2 Type of Article: ORIGINAL ARTICLE

3 Simon AVRILLON¹, François HUG^{2,3}, Gaël GUILHEM^{1*}

4
5 ¹ French Institute of Sport (INSEP), Research Department, Laboratory Sport, Expertise and
6 Performance (EA 7370) Paris, France

7 ² University of Nantes, Faculty of Sport Sciences, Laboratory Movement, Interactions,
8 Performance (EA 4334), Nantes, France

9 ³ Institut Universitaire de France (IUF), Paris, France

10 11 ***Correspondence and reprints:**

12 Gaël Guilhem, PhD
13 Institut National du Sport, de l'Expertise et de la Performance
14 Département de la Recherche
15 Laboratoire Sport, Expertise et Performance (EA 7370)
16 11, avenue du Tremblay
17 75012 Paris, France
18 Tel: +33 (0)1 41 74 43 36
19 Fax: +33 (0)1 41 75 45 35
20 e-mail: gael.guilhem@insep.fr

21
22 **Running title:** Thigh muscle coactivation

23 **Acknowledgments:** S. Avrillon is supported by a scholarship funded by the French Ministry
24 of Research. F. Hug is supported by a fellowship from the Institut Universitaire de France
25 (IUF). This study was partly supported by a grant from the Région Pays de la Loire (QUETE
26 project, no. 2015-09035).

27 **Conflict of interest:** no conflicts of interest, financial or otherwise, are declared by the
28 authors.

29 **Keywords:** Hamstrings; Quadriceps; Muscle coactivity; Shear wave elastography; Surface
30 electromyography

1 **Abstract (192 words)**

2 **Purpose:** This study aimed to assess muscle coactivation in quadriceps and hamstring muscles using
3 ultrasound shear wave elastography.

4 **Methods:** During maximal voluntary isometric contractions (MVC), both myoelectrical activity and
5 shear modulus of antagonist muscles were measured (i.e., *rectus femoris*, *vastus lateralis* and *vastus*
6 *medialis* during knee flexions and *semitendinosus*, *semimembranosus* and *biceps femoris long head*
7 during knee extensions). To account for changes induced by inevitable joint rotation during MVC, the
8 shear modulus values were compared to those measured at the same knee angle during a passive cycle.
9 The difference between these values was considered as coactivation.

10 **Results:** Myoelectrical activity was detected in all antagonist muscles ($8.0 \pm 4.9\%$ of maximal EMG
11 RMS). Significant differences were observed between shear modulus values measured during MVC
12 and those measured at the matched knee angle for all muscles (range: 2.7-4.8 kPa; all $p < 0.011$)
13 except for *semitendinosus* ($+1.7 \pm 5.0$ kPa; $p = 0.16$) and *semimembranosus* ($+1.2 \pm 5.6$ kPa; $p = 0.39$).
14 The magnitude of coactivation varied greatly among individuals.

15 **Conclusions:** Although non-negligible myoelectrical activity was observed in all muscles,
16 coactivation assessed using elastography was considered as negligible in both the *semitendinosus* and
17 *semimembranosus*. Between-muscle and between-participants differences warrant further investigation.

18

19

1 **Introduction**

2 Coactivation is defined as the activation of an antagonist muscle that accompanies the
3 contraction of an agonist muscle (De Luca and Mambrito, 1987, Enoka, 2015). It is mediated by the
4 inhibition of Ia-interneurons which decreases reciprocal inhibition of motor neurons innervating
5 antagonist muscles and by the modulation of recurrent inhibition of antagonist muscles (Crone and
6 Nielsen, 1989, Nielsen, 2016). Coactivation is thought to increase the stability of the joint through
7 regulation of muscle stiffness (Reeves et al., 2008, Sartori et al., 2015). Numerous studies have
8 estimated the mechanical effect of coactivation through the relationship between myoelectrical activity
9 measured using electromyography (EMG) and joint torque (Kellis, 1998). Substantial antagonist
10 contraction has been reported for quadriceps and hamstring muscles during maximal concentric
11 (hamstrings: $\approx 23.7\%$ of maximal voluntary contraction [MVC] (Aagaard et al., 2000, Baratta et al.,
12 1988), eccentric (quadriceps: $\approx 8.5\%$ of MVC (Aagaard et al., 2000) and isometric contractions
13 (hamstrings: $\approx 23\%$ of MVC; quadriceps: $\approx 5\%$ of MVC (Macaluso et al., 2002).

14 An important assumption made by the studies that quantified coactivation using surface EMG
15 is that the recorded myoelectrical activity accurately reflects muscle activation. However, the
16 myoelectric signal detected over the targeted muscle may originate, at least in part, from nearby
17 muscles. This phenomenon is referred to as “crosstalk” (Farina et al., 2016, Winter et al., 1994). For
18 example, although a significant surface EMG signal was measured in the *soleus* muscle during ankle
19 dorsiflexion, leading to the conclusion that co-activation was occurring, no activation was detected
20 through fine-wire electrodes (Etnyre and Abraham, 1988). The distance from the recording electrodes
21 to muscle fibers, which is related to the thickness of the subcutaneous tissues, may influence the
22 amount of crosstalk (Latash, 2018). For example, even though Wu et al. (2017) reported a greater
23 amplitude of the surface EMG signal of the antagonist *biceps femoris* in males than females, this
24 difference disappeared when accounting for the difference in adipose tissue thickness. Together, these
25 results suggest that the mechanical effect of coactivation assessed using surface EMG might be
26 overestimated.

27 Shear wave elastography (SWE) has been proposed as a possible alternative to identify the
28 contribution of antagonist muscle (Raiteri et al., 2016). This technique provides an accurate estimation

1 of muscle stiffness during passive stretching and submaximal contractions, with the changes in muscle
2 stiffness being closely related to the changes in muscle force (Brandenburg et al., 2014, Chernak et al.,
3 2013, Hug et al., 2015). Taking advantage of this technique, Raiteri et al. (Raiteri et al., 2016)
4 observed no significant changes in stiffness of the antagonist *gastrocnemius lateralis* muscle during
5 maximal isometric dorsiflexion, despite substantial muscle activation ($19.1 \pm 12.9\%$ of maximal EMG
6 amplitude). This discrepancy between EMG results, which suggest the existence of a large
7 coactivation, and elastography results, which suggest the absence of active coactivation, needs to be
8 confirmed in other muscle groups.

9 Significant differences in coactivation may exist between joints and muscles (Frey-Law and
10 Avin, 2013). For example, coactivation of knee flexors assessed using EMG reached coactivation
11 levels of up to $35 \pm 14\%$ of MVC (Beltman et al., 2003) whereas antagonist muscle activity of $19 \pm$
12 13% were observed for plantar flexors (Raiteri et al., 2016). These differences may result from EMG
13 recording conditions [e.g. the distance from the electrodes to the source and the resistance of tissues
14 (Latash, 2018)], the nature of the task (Lavoie et al., 1997, Petersen et al., 1999) or variations in reflex
15 pathways (Yavuz et al., 2018). This can result in different coactivation between muscle groups
16 crossing the same joint, as reported for the knee joint where reciprocal inhibition is higher for
17 hamstring than quadriceps muscles (Hamm and Alexander, 2010).

18 This study aimed to assess muscle coactivation in quadriceps and hamstring muscles. As
19 proposed by Raiteri et al. (2016), we took advantage of ultrasound shear wave elastography to
20 estimate the shear modulus (an index of stiffness) of the antagonist muscles. Because muscle shear
21 modulus is strongly related to both active (Bouillard et al., 2012) and passive force (Hug et al., 2015,
22 Maisetti et al., 2012), this approach allowed us to dissociate the active and passive components of
23 coactivation (Raiteri et al., 2016). We compared these results to those obtained with a more classical
24 assessment of coactivation using the measurement of EMG amplitude. We hypothesized that i)
25 coactivation would be larger when assessed using EMG compared to elastography and ii) lower
26 coactivation level would be observed for hamstrings than quadriceps.

27

28 **Methods**

1 *Participants*

2 Eighteen healthy volunteers (age: 24 ± 3 yr.; height: 1.78 ± 0.09 m; body mass: 65 ± 11 kg; 9
3 females and 9 males), with no recent history of musculoskeletal injury participated in this experiment.
4 Participants were informed of the procedure before providing written consent. The study was approved
5 by the ethics committee of Paris III (ref no. 3418) and the French Health Agency (IRB no. 2016-
6 A00715-46). All procedures conformed to the standards of the Declaration of Helsinki.

7
8 *Data collection*

9 The experiment involved isometric contractions and passive cycles performed on an isokinetic
10 dynamometer (Con-trex, CMV AG, Dübendorf, Switzerland) which assessed the torque produced by
11 the participant. Torque signal was digitized by a 12-bit analog-to-digital converter (DT9804; Data
12 Translation, Marlboro, MA, USA) at 1000 Hz, corrected for gravity and low-pass filtered at 20 Hz in
13 the forward and reverse directions using a third order zero phase Butterworth filter.

14 During each test, the three-dimensional position of the leg and thigh was collected by a seven-
15 camera optoelectronic motion capture system (Vicon Motion System Ltd, Oxford, UK). Briefly, each
16 camera produced infrared light, reflected by the markers and then captured by these same cameras.
17 The collection of the marker's position by at least two cameras enables the calculation of its 3D
18 position by triangulation. Four reflective markers were attached to the lateral malleolus, the tibial head,
19 the lateral femoral epicondyle and the greater trochanter. Models of the leg and thigh segment were
20 created with the manufacturer software to calculate the knee angle. Marker position was sampled at
21 100 Hz and low-pass filtered at 20 Hz using a third order zero phase Butterworth filter.

22 Myoelectrical activity was recorded using wireless electrodes (Zerowire, Aurion, Italy) placed
23 over the *rectus femoris* (RF), *vastus lateralis* (VL), *vastus medialis* (VM), *semitendinosus* (ST),
24 *semimembranosus* (SM), and *biceps femoris long head* (BF) of the tested leg. B-mode ultrasound
25 (Aixplorer, Supersonic Imagine, Aix-en-Provence, France) was used to accurately place the electrodes
26 longitudinally on the muscle fascicle's alignment (for each muscle except RF), away from the borders
27 of the neighboring muscles. Due to the complex architecture of the RF, the electrodes were placed in
28 the shortening direction of this muscle. Before electrode application, the skin was shaved and cleaned

1 with alcohol. Ag/AgCl electrodes (Blue sensor N-00-S, Ambu, Copenhagen, Denmark) were attached
2 to the skin with an inter-electrode distance of 20 mm (centre-to-centre) following SENIAM
3 recommendations (Hermens et al., 2000). Raw EMG signals were pre-amplified (input impedance: 20
4 M Ω ; common-mode-rejection ratio: 90 dB; signal-to-noise-ratio: >50 dB; gain: 1000) and sampled at
5 2000 Hz (Zerowire, Aurion, Milan, Italy).

6 An Aixplorer ultrasound scanner (version 6; Supersonic Imagine), coupled with a linear
7 transducer array (4–15 MHz, SuperLinear 15-4, Vermon, Tours, France) was used in shear wave
8 elastography mode (musculoskeletal preset). In short, the linear transducer array produces a focused
9 ultrasound beam. Each pushing beam generates a mechanical perturbation that results in the
10 propagation of shear waves. An ultrasound imaging sequence is then performed to acquire successive
11 raw radio-frequency data at a very high frame rate (up to 2000 Hz). One-dimensional cross-correlation
12 of successive radio-frequency signals is used to determine the shear wave velocity (V_s) along the
13 principal axis of the probe. This propagation velocity is directly related to the shear modulus of the
14 tissue, that is, the faster the shear wave propagation, the higher the shear modulus.

$$15 \quad \mu = \rho V_s^2$$

16 where μ is the shear modulus of the tissue, ρ is the density of the tissue (1000 kg.m⁻³ for
17 muscles) and V_s is the shear wave velocity. A 2-dimensional map of shear modulus is provided at one
18 sample per second. The optimal ultrasound probe location and orientation was determined such that
19 several fascicles could be observed for all muscles except for the RF, for which the ultrasound probe
20 was aligned with the shortening direction of the muscle (Hug et al., 2014). These locations were
21 marked on the skin using a waterproof marker so that the transducer location remained constant for all
22 measurements. Previous studies from our group (Lacourpaille et al., 2012, Morales-Artacho et al.,
23 2017) and others (Umegaki et al., 2015) have reported a good to excellent inter-session reliability of
24 the shear modulus measurements for quadriceps (ICC: 0.74-0.87; CV: 4.7-5.6%) and hamstring
25 muscles (ICC: 0.86-0.99; CV: 3.5-11.6%). Because there is a strong linear relationship between
26 muscle shear modulus and muscle force, we considered changes in shear modulus as an index of
27 changes in muscle force (see Hug et al. (2015) for a detailed review). A transistor-transistor logic

1 pulse originating from each system and recorded on the acquisition card was used to synchronize
2 elastography, mechanical, motion capture and EMG data.

3

4 *Experimental setup*

5 Participants were positioned supine with the right leg attached to an isokinetic dynamometer
6 arm (Con-trex, CMV AG, Dübendorf, Switzerland). The right hip and knee joints were flexed at 90°
7 (0° = anatomical position and full extension for the hip and the knee, respectively). Non-compliant
8 straps were used to secure participants position and minimize changes in hip angle throughout the
9 contractions. This setup ensured a stable positioning of the ultrasound probe over the hamstring and
10 quadriceps muscles during maximal isometric knee extension and flexion, respectively (Fig. 1).

11

12 *Experimental tasks*

13 After a specific warm-up in isometric condition, participants performed nine maximal
14 isometric knee extensions and flexions such that three measurements of shear modulus and EMG per
15 antagonist muscle were obtained. Each contraction was maintained 3 s separated by 3 min of rest.
16 Instead of achieving MVC torque as fast as possible, participants were asked to progressively increase
17 their torque up to MVC over 6 s to ensure constant alignment and pressure of the ultrasound probe
18 over the muscle belly. The order of the tasks (i.e. knee flexion and extension) and muscles recorded
19 was randomized.

20 Passive shear modulus was assessed twice for each muscle. Flexor and extensor muscles were
21 passively stretched before measurements through five slow ($10^{\circ} \cdot s^{-1}$) loading/unloading cycles for
22 conditioning purposes. Because subtle knee rotations occur during maximal isometric contractions,
23 passive shear modulus was assessed in each thigh muscle during passive cycles performed at $1^{\circ} \cdot s^{-1}$
24 throughout a range of motion that encompasses this knee rotation (i.e., from 100° to 80° of knee angle).
25 The order of tested muscles was randomized.

26

27 *Data analysis*

1 All data were processed using Matlab (Mathworks, R2015a, Natick, MA) custom-written
2 scripts. Elastography recordings of each trial (passive cycle and MVC) were exported in video format
3 and sequenced into images. Image processing converted the colored map into shear modulus values
4 (Fig. 1A). The region of interest was inspected to exclude non-muscular structures and artifacts
5 (saturated values and void areas).

6 All EMG signals were first band-pass-filtered in the forward and reverse directions (10-500
7 Hz, third-order zero phase Butterworth filter). A 100-ms sliding window with a 1-ms step was applied
8 to EMG signals to calculate the root-mean square (RMS) amplitude. The maximal EMG RMS
9 amplitude (EMG RMS_{max}) was the maximal value achieved during MVC when each muscle acted as
10 an agonist. The EMG RMS of antagonist muscle was then normalized to this maximal EMG RMS
11 value.

12 The 3-s torque plateau reached during each maximal isometric contraction was used for
13 further analysis. The antagonist muscle shear modulus, EMG RMS and corresponding knee angle (Fig.
14 1A) were considered at the peak torque produced by agonist muscles. The shear modulus measured at
15 the matched knee angle during passive cycle was determined from the passive shear modulus–knee
16 angle relationship (Fig. 1B). This value was then subtracted from the shear modulus measured during
17 maximal contraction to determine the shear modulus representative of active force produced by
18 antagonist muscles only. EMG amplitude was continuously monitored to ensure that this relationship
19 accounted for passive muscle force only. Note that no passive cycles were excluded given that EMG
20 RMS was systematically lower than 3% of EMG RMS_{max}.

21 22 *Statistics*

23 All statistical analyses were conducted using Statistica version 7.1 software (StatSoft, Tulsa,
24 OK). Normality testing (Kolmogorov-Smirnov) was consistently passed, and values are therefore
25 reported as mean \pm SD. For each muscle group, we ran a repeated-measures analysis of variance to
26 compare EMG RMS among synergist muscles (within-subject factors: muscle). The significance level
27 was set at $P < 0.05$. When required, *post hoc* analyses were performed using Bonferroni tests.

1 Due to technical limitations of the scanner in measuring high shear modulus values (Hug et al.,
2 2015), measurements cannot be normalized to the value measured during MVC; in turn, between-
3 muscle comparison of shear modulus values is meaningless. Therefore, paired t-tests were used to
4 compare the shear modulus value measured during MVC and during the passive cycle at a matched
5 knee angle to test whether active coactivation was present. The absence of a significant difference
6 indicated that the change in shear modulus during MVC was only due to passive knee rotation and
7 therefore active coactivation was negligible. The significance level was set at $P < 0.033$ using the
8 Benjamini and Hochberg procedure (Benjamini and Hochberg, 1995) to limit the false discovery rate
9 induced by multiple comparisons (n=6).

10

1 **Results**

2 EMG activity measured while muscles acted as antagonists ranged from 6.7 ± 4.2 % to $9.4 \pm$
3 3.9 %. No differences were found between muscles (main effect of muscle: $p = 0.84$ and $p = 0.69$ for
4 Quadriceps and Hamstrings, respectively; Fig 2).

5 When considering the quadriceps during maximal knee flexion, shear modulus reached from
6 7.0 ± 2.7 kPa for RF to 12.4 ± 5.5 kPa for VL (Fig. 3A). Knee angle was $96.4 \pm 3.2^\circ$ at peak isometric
7 torque achieved during MVC, leading to a mean passive shear modulus of 6.1 ± 2.0 kPa (Fig. 3A).
8 Significant differences were observed between the passive shear modulus measured at matched angle
9 and the value measured during maximal knee flexion for RF (2.8 ± 2.8 kPa: $p < 0.001$), VL (4.3 ± 5.3
10 kPa: $p = 0.003$) and VM (3.3 ± 4.9 kPa; $p = 0.011$).

11 When considering the hamstrings during maximal knee extension, the shear modulus reached
12 from 9.0 ± 3.8 kPa for ST to 20.4 ± 7.1 kPa for BF (Fig. 3C). Knee angle measured during knee
13 extension MVC was on average $83.8 \pm 2.7^\circ$. The passive shear modulus measured at the
14 corresponding angle reached 12.5 ± 6.8 kPa (Fig. 3C). There was a significant difference between
15 the passive shear modulus assessed at matched angle and that measured during maximal knee
16 extension for BF (4.8 ± 6.9 kPa, $p = 0.009$). In contrast, no differences were observed for ST (1.7 ± 5.0
17 kPa; $p = 0.16$) and SM (1.2 ± 5.6 kPa; $p = 0.39$). Note that a large variability was observed between
18 participants (Fig. 3D).

19

1 **Discussion**

2 Taking advantage of shear wave elastography, this study aimed to assess the coactivation of
3 quadriceps and hamstring muscles during maximal isometric contractions. Despite EMG data
4 suggested a substantial and similar level of coactivation among antagonist muscles, elastography data
5 provided evidence of between-muscle differences in coactivation. Specifically, all quadriceps muscles
6 were consistently coactivated during knee flexions. Inversely, only BF muscle exhibited substantial
7 coactivation when the hamstrings acted as antagonists. For ST and SM, no differences were observed
8 between the shear modulus measured during MVC and that measured at rest at the corresponding knee
9 angle. Because this difference is attributable to the active force produced by the muscle, it provides
10 evidence that active coactivation is negligible for ST and SM. Our findings also show that the
11 magnitude of coactivation differed between individuals, with potential functional consequences on
12 joint stability.

13 Muscle activation measured in the present study using surface EMG is comparable to that
14 reported by previous work (quadriceps: 4.5-11.9% of maximal EMG amplitude (Krishnan and
15 Williams, 2010, Macaluso et al., 2002); hamstring: 5.3-12.7 % (Kellis and Katis, 2008, Krishnan and
16 Williams, 2010)). However, Macaluso et al. (2002) observed higher values of coactivation in
17 hamstring muscles (i.e., 23-41%). Although the origin of these differences is unclear, subcutaneous
18 adipose tissue has been proposed as greatly influencing the amount of crosstalk from agonist muscles
19 (Wu et al., 2017). Variations in adipose tissue thickness among muscles and participants might thereby
20 affect the level of coactivation inferred from surface EMG. Here, we used elastography, which offers
21 two main advantages: i) it provides a more direct estimation of muscle force than EMG; and ii) it is
22 insensitive to crosstalk (Hug et al., 2015).

23 We considered co-activation as present when the shear modulus of the antagonist muscles
24 measured during MVC and that measured during passive condition at a matched joint angle were
25 significantly different (Raiteri et al., 2016). Using this approach, significant coactivation was observed
26 for all quadriceps muscles (Fig. 3A). This result concurs with previous observations inferred from
27 EMG data during maximal knee flexions (Krishnan and Williams, 2009, Saito et al., 2013). The
28 presence of a quadriceps active force may be a prerequisite for maintaining joint stability during

1 maximal knee flexion (Baratta et al., 1988, Frey-Law and Avin, 2013, Kellis, 1998). More precisely,
2 the contraction of thigh muscles has been reported as inducing a shear force on passive structures
3 crossing the knee joint (i.e. anterior and posterior cruciate ligaments; (Frey-Law and Avin, 2013,
4 Kellis, 1998)). Previous data demonstrated the ability of antagonist muscles to compensate for such
5 internal loading forces that may induce joint instability (Solomonow et al., 1987). In addition,
6 although the present maximal contractions were performed at the same angular position, they resulted
7 in slight changes in joint angle ($\sim 6^\circ$). These small variations shorten the moment arm (Visser et al.,
8 1990). Thus, quadriceps coactivation could contribute to counteracting the decrease of antagonist
9 muscle mechanical advantage (Baratta et al., 1988).

10 When considering the hamstrings, a significant difference was observed between the shear
11 modulus measured during MVC and that measured at matched knee angle for BF but not for SM and
12 ST muscles (Fig. 3C). This absence of antagonist active shear modulus despite substantial
13 myoelectrical activity is in line with previous observations that coactivation of plantarflexors is
14 negligible during maximal isometric dorsiflexion (Raiteri et al., 2015, Raiteri et al., 2016). The present
15 study provides further evidence that the EMG technique may overestimate coactivation during
16 isometric contractions likely because of the presence of crosstalk.

17 Even though EMG data suggested that quadriceps and hamstring muscles were coactivated
18 during maximal isometric contractions, the elastography technique showed different coactivation
19 strategies for quadriceps and hamstring muscles. As coactivation of antagonist muscles is mediated by
20 reflex responses, these differences could originate from specific reciprocal inhibition (Bayoumi and
21 Ashby, 1989). Using electrical stimulation, Bayoumi and Ashby (1989) have demonstrated stronger
22 reciprocal inhibition from knee extensors to flexors while the opposite was not true. Our findings
23 could thus reflect larger reciprocal inhibition from quadriceps that may translate into substantial lower
24 coactivation of hamstring muscles during maximal isometric knee extension.

25 Our results also demonstrate differences in coactivation between hamstring muscles with only
26 BF being actively coactivated. Although previous studies reported significant coactivation levels for
27 all hamstrings muscles, they also reported a larger EMG amplitude in lateral (BF) than medial (SM
28 and ST) hamstring muscles (Aagaard et al., 2000, Krishnan et al., 2011). Such differences in

1 coactivation strategies may contribute to improve accuracy in tasks that require more precision, e.g.
2 during upper arm movements (Gribble et al., 2003). An additional functional advantage derived from
3 this strategy is that it may protect the passive structures of the knee joint by restricting internal tibial
4 rotation and the associated shear force (Aagaard et al., 2000).

5 Inspection of individual shear modulus values reveals substantial variability between
6 participants. For example, active shear modulus calculated in participant 3 was very close to 0 kPa,
7 while it was much higher in participant 14 (7.6 kPa, 9.6 kPa and 5.1 kPa for ST, SM and BF,
8 respectively; Fig. 3D). Although isometric contractions performed in the present study could be
9 considered as a constrained motor task, muscular redundancy still exists, leading to different possible
10 force-sharing strategies to achieve the motor task. Each individual may therefore use a different
11 combination of muscles over time. The interindividual variability could thus reflect the existence of
12 individual-specific force-sharing strategies to ensure dynamic stability of motor performance (Latash,
13 2018).

14 The causes of such differences are unclear. Additional force from antagonist muscles may
15 result from an inadequate passive force from joint stabilizers (e.g., ligaments crossing the knee). For
16 example, Solomonow et al. (1987) provided evidence that antagonist muscles (i.e., hamstrings) played
17 a substantial role as a stabilizer in individuals who had a deficient anterior cruciate ligament.
18 Alternatively, these differences could result from different force-generating capacities (i.e.,
19 physiological cross-sectional area [PCSA], specific tension and moment arm) among individuals. For
20 example, a muscle with a larger PCSA may need lower activation to produce sufficient antagonist
21 torque to stabilize the joint. Thus, despite the differences in shear modulus between individuals or
22 muscles, the variation in antagonist muscle torque may be lower, if not lacking, due to differences in
23 force-generating capacity. Further studies including separate groups of individuals who exhibit clear
24 differences in coactivation strategies could more directly investigate this hypothesis.

25 The use of shear wave elastography as an index of muscle force during submaximal
26 contractions require substantial considerations. Because the current ultrasound shear wave
27 elastography technique cannot accurately measure the shear modulus of very stiff tissues, we were not
28 able to normalize the shear modulus values to that recorded during MVC (Hug et al., 2015). Such a

1 normalization procedure is a prerequisite to quantify the mechanical effect of each muscle in the
2 production of antagonist joint torque. In the absence of normalization, our results only provide
3 information about the changes in muscle force between MVC and the passive condition (while the
4 muscle acts as an antagonist, where an absence of change can be directly interpreted as an absence of
5 active coactivation as for ST and SM muscles.

6 **Conclusion**

7 This study provides new insights into the coactivation strategy involved in quadriceps and
8 hamstring muscles during maximal contractions. We first observed substantial and similar myoelectric
9 activity for all muscles. However, the use of elastography revealed negligible coactivation in both the
10 *semitendinosus* and *semimembranosus*. In the latter case, the antagonist torque is mostly the result of
11 an increase in passive force. This suggests that the level of coactivation varies between muscles and
12 that surface EMG cannot detect such differences, likely due to crosstalk.

13

14 **Acknowledgments**

15 S. Avrillon was supported by a scholarship funded by the French Ministry of Research. F. Hug was
16 supported by a fellowship from the Institut Universitaire de France (IUF). The authors thank all the
17 participants for their involvement in the experiment. No conflicts of interest, financial or otherwise,
18 are declared by the authors.

19

1 **References:**

- 2 Aagaard P, Simonsen EB, Andersen JL, Magnusson SP, Bojsen-Moller F, Dyhre-Poulsen P.
3 Antagonist muscle coactivation during isokinetic knee extension. *Scan J Med Sci Sports*.
4 2000;10:58-67.
- 5 Baratta R, Solomonow M, Zhou BH, Letson D, Chuinard R, D'Ambrosia R. Muscular
6 coactivation. The role of the antagonist musculature in maintaining knee stability. *Am J*
7 *Sports Med*. 1988;16:113-22.
- 8 Bayoumi A, Ashby P. Projections of group Ia afferents to motoneurons of thigh muscles in
9 man. *Exp Brain Res*. 1989;76:223-8.
- 10 Beltman JG, Sargeant AJ, Ball D, Maganaris CN, de Haan A. Effect of antagonist muscle
11 fatigue on knee extension torque. *Pflugers Arch*. 2003;446:735-41.
- 12 Benjamini Y, Hochberg Y. Controlling the False Discovery Rate: A Practical and Powerful
13 Approach to Multiple Testing. *J R Statist Soc B*. 1995;57:289-300.
- 14 Bouillard K, Hug F, Guevel A, Nordez A. Shear elastic modulus can be used to estimate an
15 index of individual muscle force during a submaximal isometric fatiguing contraction. *J Appl*
16 *Physiol* (1985). 2012;113:1353-61.
- 17 Brandenburg JE, Eby SF, Song P, Zhao H, Brault JS, Chen S, et al. Ultrasound elastography:
18 the new frontier in direct measurement of muscle stiffness. *Arch Phys Med Rehabil*.
19 2014;95:2207-19.
- 20 Chernak LA, DeWall RJ, Lee KS, Thelen DG. Length and activation dependent variations in
21 muscle shear wave speed. *Physiol Meas*. 2013;34:713-21.
- 22 Crone C, Nielsen J. Spinal mechanisms in man contributing to reciprocal inhibition during
23 voluntary dorsiflexion of the foot. *J Physiol*. 1989;416:255-72.
- 24 De Luca CJ, Mambrito B. Voluntary control of motor units in human antagonist muscles:
25 coactivation and reciprocal activation. *J Neurophysiol*. 1987;58:525-42.
- 26 Enoka RM. Neural control of movement. *Neuromechanics of Human Movement 5th Edition:*
27 *Human Kinetics*; 2015. p. 255-313.
- 28 Etnyre BR, Abraham LD. Antagonist muscle activity during stretching: a paradox re-assessed.
29 *Med Sci Sports Exerc*. 1988;20:285-9.
- 30 Farina D, Stegeman DF, Merletti R. Biophysics of the Generation of EMG Signals. In:
31 Merletti R, Farina D, editors. *Surface Electromyography : Physiology, Engineering, and*
32 *Applications: John Wiley & Sons, Inc.*; 2016. p. 1-24.
- 33 Frey-Law LA, Avin KG. Muscle coactivation: a generalized or localized motor control
34 strategy? *Muscle Nerve*. 2013;48:578-85.
- 35 Gribble PL, Mullin LI, Cothros N, Mattar A. Role of cocontraction in arm movement
36 accuracy. *J Neurophysiol*. 2003;89:2396-405.
- 37 Hamm K, Alexander CM. Challenging presumptions: is reciprocal inhibition truly reciprocal?
38 A study of reciprocal inhibition between knee extensors and flexors in humans. *Man Ther*.
39 2010;15:388-93.
- 40 Hermens HJ, Freriks B, Disselhorst-Klug C, Rau G. Development of recommendations for
41 SEMG sensors and sensor placement procedures. *J Electromyogr Kinesiol*. 2000;10:361-74.

- 1 Hug F, Hodges PW, van den Hoorn W, Tucker K. Between-muscle differences in the
2 adaptation to experimental pain. *J Appl Physiol* (1985). 2014;117:1132-40.
- 3 Hug F, Tucker K, Gennisson JL, Tanter M, Nordez A. Elastography for Muscle
4 Biomechanics: Toward the Estimation of Individual Muscle Force. *Exerc Sport Sci Rev.*
5 2015;43:125-33.
- 6 Kellis E. Quantification of quadriceps and hamstring antagonist activity. *Sports Med.*
7 1998;25:37-62.
- 8 Kellis E, Katis A. Hamstring antagonist moment estimation using clinically applicable
9 models: Muscle dependency and synergy effects. *J Electromyogr Kinesiol.* 2008;18:144-53.
- 10 Krishnan C, Allen EJ, Williams GN. Effect of knee position on quadriceps muscle force
11 steadiness and activation strategies. *Muscle Nerve.* 2011;43:563-73.
- 12 Krishnan C, Williams GN. Sex differences in quadriceps and hamstrings EMG-moment
13 relationships. *Med Sci Sports Exerc.* 2009;41:1652-60.
- 14 Krishnan C, Williams GN. Error associated with antagonist muscle activity in isometric knee
15 strength testing. *Eur J Appl Physiol.* 2010;109:527-36.
- 16 Lacourpaille L, Hug F, Bouillard K, Hogrel JY, Nordez A. Supersonic shear imaging provides
17 a reliable measurement of resting muscle shear elastic modulus. *Physiol Meas.* 2012;33:N19-
18 28.
- 19 Latash ML. Muscle Co-activation: Definitions, Mechanisms, and Functions. *J Neurophysiol.*
20 2018.
- 21 Lavoie BA, Devanne H, Capaday C. Differential control of reciprocal inhibition during
22 walking versus postural and voluntary motor tasks in humans. *J Neurophysiol.* 1997;78:429-
23 38.
- 24 Macaluso A, Nimmo MA, Foster JE, Cockburn M, McMillan NC, De Vito G. Contractile
25 muscle volume and agonist-antagonist coactivation account for differences in torque between
26 young and older women. *Muscle Nerve.* 2002;25:858-63.
- 27 Maisetti O, Hug F, Bouillard K, Nordez A. Characterization of passive elastic properties of
28 the human medial gastrocnemius muscle belly using supersonic shear imaging. *J Biomech.*
29 2012;45:978-84.
- 30 Morales-Artacho AJ, Lacourpaille L, Guilhem G. Effects of warm-up on hamstring muscles
31 stiffness: Cycling vs foam rolling. *Scan J Med Sci Sports.* 2017.
- 32 Nielsen JB. Human Spinal Motor Control. *Annu Rev Neurosci.* 2016;39:81-101.
- 33 Petersen N, Morita H, Nielsen J. Modulation of reciprocal inhibition between ankle extensors
34 and flexors during walking in man. *J Physiol.* 1999;520 Pt 2:605-19.
- 35 Raiteri BJ, Cresswell AG, Lichtwark GA. Ultrasound reveals negligible cocontraction during
36 isometric plantar flexion and dorsiflexion despite the presence of antagonist
37 electromyographic activity. *J Appl Physiol* (1985). 2015;118:1193-9.
- 38 Raiteri BJ, Hug F, Cresswell AG, Lichtwark GA. Quantification of muscle co-contraction
39 using supersonic shear wave imaging. *J Biomech.* 2016;49:493-5.
- 40 Reeves NP, Cholewicki J, Milner T, Lee AS. Trunk antagonist co-activation is associated
41 with impaired neuromuscular performance. *Exp Brain Res.* 2008;188:457-63.

- 1 Saito A, Watanabe K, Akima H. The highest antagonistic coactivation of the vastus
2 intermedius muscle among quadriceps femoris muscles during isometric knee flexion. *J*
3 *Electromyogr Kinesiol.* 2013;23:831-7.
- 4 Sartori M, Maculan M, Pizzolato C, Reggiani M, Farina D. Modeling and simulating the
5 neuromuscular mechanisms regulating ankle and knee joint stiffness during human
6 locomotion. *J Neurophysiol.* 2015;114:2509-27.
- 7 Solomonow M, Baratta R, Zhou BH, Shoji H, Bose W, Beck C, et al. The synergistic action
8 of the anterior cruciate ligament and thigh muscles in maintaining joint stability. *Am J Sports*
9 *Med.* 1987;15:207-13.
- 10 Umegaki H, Ikezoe T, Nakamura M, Nishishita S, Kobayashi T, Fujita K, et al. Acute effects
11 of static stretching on the hamstrings using shear elastic modulus determined by ultrasound
12 shear wave elastography: Differences in flexibility between hamstring muscle components.
13 *Man Ther.* 2015;20:610-3.
- 14 Visser JJ, Hoogkamer JE, Bobbert MF, Huijing PA. Length and moment arm of human leg
15 muscles as a function of knee and hip-joint angles. *Eur J Appl Physiol Occup Physiol.*
16 1990;61:453-60.
- 17 Winter DA, Fuglevand AJ, Archer SE. Crosstalk in surface electromyography: Theoretical
18 and practical estimates. *J Electromyogr Kinesiol.* 1994;4:15-26.
- 19 Wu R, Delahunt E, Ditroilo M, Lowery M, De Vito G. Effect of Knee Joint Angle and
20 Contraction Intensity on Hamstrings Coactivation. *Med Sci Sports Exerc.* 2017;Publish
21 Ahead of Print.
- 22 Yavuz US, Negro F, Diedrichs R, Farina D. Reciprocal inhibition between motor neurons of
23 the tibialis anterior and triceps surae in humans. *J Neurophysiol.* 2018;119:1699-706.
- 24

1 **Figure captions**

2 Fig. 1: Illustration of the signal processing employed to determine the level of *the biceps femoris long*
3 *head* coactivation during maximal knee extension. A. Typical raw data of antagonist shear modulus
4 collected on *biceps femoris* muscle and variation of knee angle during maximal knee extension. B.
5 Typical raw data of shear modulus and variation of knee angle during a passive cycle. Quantification
6 of the active part of shear modulus determined by subtracting the amount of passive shear modulus to
7 MVC shear modulus at knee match-angle and direction.

8

9 Fig. 2: Coactivation level assessed with EMG. Mean \pm SD values are presented for quadriceps (*rectus*
10 *femoris* [RF], *vastus lateralis* [VL], *vastus medialis* [VM]) (A) and hamstrings (*semitendinosus* [ST],
11 *semimembranosus* [SM] and *biceps femoris* [BF]) (B).

12

13 Fig. 3: Muscle shear modulus during maximal voluntary contraction (MVC), passive cycle and the
14 subsequent active part, with significant differences between conditions for quadriceps [*rectus femoris*
15 (RF), *vastus lateralis* (VL), *vastus medialis* (VM)] (A) and *biceps femoris* (BF) while no differences
16 were observed for *semitendinosus* (ST), *semimembranosus* (SM) (C). Individual values of the active
17 part of shear modulus are presented for quadriceps (B) and hamstring muscles (D).

18

19

20