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Running title: Injury-induced changes in muscle coordination

ABSTRACT (250 words)

Background: Hamstring strain injuries (HSI) involve tissue disruption and pain, which can trigger long-term adaptations of muscle coordination. However, little is known about the effect of previous HSI on muscle coordination, and in particular, after the completion of rehabilitation and in the absence of symptoms. This study aimed to determine if elite athletes with a prior unilateral HSI have bilateral differences in coordination between the hamstring muscle heads after returning to sport.

Methods: Seventeen athletes with a unilateral history of biceps femoris injury participated in the experiment. Surface electromyography (EMG) was recorded from three hamstring muscles (biceps femoris [BF], semimembranosus [SM], and semitendinosus [ST]) during submaximal isometric torque-matched tasks. The product of normalized electromyographic amplitude with functional cross-sectional area (PCSA) and moment arm was considered as an index of muscle torque for each of the three hamstring heads.

Results: The contribution of the injured muscle to total knee flexor torque was lower compared to the uninjured limb (-10.8±27.5%; P=0.038). This reduced contribution of BF was compensated by a higher contribution of the SM muscle in the injured limb (+17.2±27.4%; P=0.007). These changes resulted from a decreased contribution of PCSA from the injured muscle (BF), and an increased contribution of activation from an uninjured synergist muscle (SM).

Conclusions: Bilateral differences in coordination were observed in previously injured athletes despite the completion of rehabilitation. Whether these bilateral differences in hamstring coordination could constitute an intrinsic risk factor that contributes to the high rate of hamstring injury recurrence remains to be investigated.

NEW & NOTEWORTHY:

We used an experimental approach combining the assessment of muscle activation, physiological-cross sectional area and moment arm to estimate force-sharing strategies among hamstring muscles during isometric knee flexions in atheletes with an history of hamstring injury. We observed a lower contribution of the injured biceps femoris to the total kne flexor torque in the injured limb than in the contralateral limb. This decreased contribution was mainly due to a selective atrophy of the injured biceps femoris muscle and was compensated by an increased activation of the semimembranosus muscle.

KEYWORDS

Hamstring injury; Torque-sharing strategies; Atrophy; Muscle activation; Muscle coordination

1. INTRODUCTION

A hamstring strain injury is a leading cause of unavailability for training and competition in numerous sports (27). Due to their high incidence and reinjury rate (29), prevention is a main challenge for both coaches and clinicians. Most of these strain injuries involve tissue disruption in the *biceps femoris* (BF) muscle (11). These lesions are associated with pain and functional losses due to mechanical alterations. In addition, changes in muscle activation and neuromuscular inhibition may occur (13, 34).

Some theories propose that movement is modified in the presence of pain in order to unload the painful/injured tissue (20, 26). Although unloading the injured muscle seems logical during the acute phase of hamstring strain injury (34), it is unclear whether this adaptation persists after rehabilitation when pain has resolved. It is important to address this question as previous work suggested that altered coordination strategies might have an immediate benefit for the system, but that the persistence of these changes might have negative long-term consequences and increase reinjury risk (10, 20).

Muscle coordination relates to the distribution of force among individual muscles to produce a given motor task (22). As such, the study of muscle coordination requires the consideration of individual muscle force rather than muscle activation alone (22), especially within the context of muscle injury where both muscle activation and muscle force-generating capacity are likely to be altered. During isometric contractions, individual muscle force can be estimated from information on both activation and physiological cross-section area (PCSA). This approach considers that a difference in force-generating capacity between synergist muscles is mainly attributable to their difference in PCSA. This is reasonable when considering submaximal isometric knee flexions during which neither the force-length relationship nor the specific tension is expected to vary greatly between the hamstring muscle heads, because of their similar action on both the knee and hip joints (39) and their similar

fiber-type content (14). Using this approach, Avrillon et al. (2) reported large interindividual variability in muscle coordination strategies between the hamstring muscles, which is in some cases detrimental for motor performance (i.e., the higher the activation variability, the lower the time to exhaustion).

Although previous studies reported an alteration in either muscle force-generating capacity or activation after hamstring strain injury, none of the studies considered these parameters together, making it complicated to infer changes in muscle coordination. Silder et al. (37) reported a selective decrease in volume of the injured muscle (BF in most of the participants) six months after injury. Although this result might suggest a reduced contribution of this injured muscle to joint torque, muscle activation was not assessed. Schuermans et al. (35, 36) reported a larger contribution of BF and SM muscles compared to ST up to two years after an injury, but did not consider muscle volume or PCSA. In addition, the fragmented information in these studies regarding injury localization made interpretation of the observed adaptations difficult.

Here, we assessed muscle coordination in elite athletes with a hamstring injury in the previous seven months that have returned to sport. We tested the hypothesis that the contribution of the injured muscle to submaximal knee flexion tasks will be reduced in the injured limb compared to the non-injured limb. This reduced contribution would be a combination of both a smaller volume and a lower activation of the injured muscle compared to uninjured muscles.

2. METHODS

2.1. Participants

Seventeen elite male sprinters and long jumpers volunteered for the study (age: 26.3±5.5 yr., height:1.79±0.05 m, body mass: 74.4±8.1 kg). They had a history of injury to the *biceps femoris* long head (BFlh). Note that the *semitendinosus* (ST) was also involved in the injuries

of three athletes (Table 1). All athletes were free from lower limb pain at the date of the experiment and were able to perform maximal knee flexions. All participants were informed regarding the nature, aims and risks associated with the experiments before they gave their written consent to participate. Experimental procedures were approved by the local ethical committee (reference no. 3418, RCB no. 2016-A00715-46) and conformed to the Declaration of Helsinki.

2.2. Injury history

All participants had a unilateral strain injury of the BFlh within the past seven months (Table 1). The average delay between injury occurrence and testing was 98.2±53.3 days (range 22-198 days). We defined a hamstring injury as an acute pain in the posterior thigh that occurred during a sprint and resulted in the immediate termination of the training session or competition. Each injured athlete underwent an MRI (n = 9) or an ultrasound (n = 8) exam performed by a radiologist within the week following injury. Athletes met inclusion criteria when the precise localization and the grade of the injury was confirmed by the exam. Their injuries caused training activities to stop for 32.5±17.5 days (range 14-70 days). All athletes completed a supervised rehabilitation protocol provided by a qualified physiotherapist. In the absence of standardization, the rehabilitation program could slightly differ in content and periodization. At the time of testing the participants were allowed to return to their regular sport activities (included sprinting) by the clinical staff, had recovered to their pre-injury peak knee flexor torque level, and were free of any lower limb pain.

2.3. Protocol

Participants attended three sessions in a randomized order: i) a MRI session to estimate both muscle volume and muscle moment arm, ii) an ultrasound session to estimate fascicle length

and pennation angle, and iii) an experimental session during which muscle activation was assessed using surface EMG. Specifically, participants sat on an isokinetic dynamometer (Con-Trex, CMV AG, Dübendorf, Switzerland) with non-compliant straps placed around the chest, the pelvis and the thigh. The hip and the knee were flexed at 90° and 45°, respectively (0° = neutral position for the hip and full extension for the knee). Knee angle was chosen as it corresponds to the peak knee flexor torque angle, i.e., the optimal angle (23). The torque signal from the isokinetic dynamometer was recorded and digitized by a USB data acquisition module (DT9804; Data Translation, Marlboro, MA, USA) at 1000 Hz. Torque was corrected for gravity and low-pass filtered at 20 Hz using a third-order Butterworth filter. Visual feedback of the exerted torque signal was displayed on a screen placed in front of the participants.

2.4. Estimation of muscle activation

2.4.1. Experimental tasks

After a standardized warm-up (ten isometric knee flexions at 50% of peak torque and five isometric knee flexions at 80% of peak torque), participants performed three MVC of the knee flexors for 3 to 5 s with 120-s rest in between. The maximal value obtained from a moving average window of 300 ms was considered as the peak knee flexor torque. Then, participants performed three 10-s contractions at both 20% and 50% of MVC peak torque (30-s rest in between). This protocol was performed for each leg in a randomized order with 5 min rest in between.

2.4.2. Surface electromyography

Myoelectric activity was recorded bilaterally through surface electrodes placed over the ST, SM, and BF. The participants were seated on a customized piece of foam with a free space

beneath each muscle to ensure that there was no contact between the electrodes and the seat. We used B-mode ultrasound (v10, Aixplorer, Supersonic Imagine, Aix-en-Provence, France) to determine the appropriate placement of electrodes on each muscle, longitudinally with respect to the muscle fascicle's alignment and away from the borders of neighboring muscles. As the superficial part of the BF short head is close to the popliteal fossa, it was not possible to investigate this muscle. We therefore followed the SENIAM recommendations for electrode placement on BF and considered the recorded myoelectrical activity originating from this pair of electrodes as being representative of both the short and long head. The skin was shaved and cleaned with alcohol and a pair of Ag/AgCl electrodes (recording zone area: 520 mm², Blue sensor N-00-S, Ambu, Copenhagen, Denmark) was attached to the skin with an inter-electrode distance of 20 mm (centre-to-centre). Raw EMG signals were pre-amplified (input impedance: 20 MM, CMRR: 90 db; gain: 1000), band-pass filtered (10-500 Hz, third order Butterworth filter) and sampled at 2000 Hz (Zerowire, Aurion, Milan, Italy). EMG and mechanical data were synchronized using a transistor-transistor-logic pulse recorded by a 12-bit analog to digital converter (DT9804, Data Translation, Marlboro, USA).

2.4.3. Data processing

All mechanical and EMG data were analyzed using MATLAB custom-written scripts (R2017a, The Mathworks, Nathick, MA, USA). The Root Mean Square (RMS) of the EMG signal was calculated over a moving time window of 300 ms and the maximal value achieved over the three trials was considered as the maximal activation level (EMG RMS_{max}). During the submaximal isometric knee flexion tasks, the EMG RMS amplitude was calculated over 5 s at the period corresponding to the lowest standard deviation of the torque signal. For each trial, this value was normalized to that measured during the MVC task. The ratio of activation

between the hamstring muscles was calculated as the normalized EMG RMS of the considered muscle divided by the sum of normalized EMG RMS values of all three muscles:

$$Activation\ ratio\ (muscle) = \frac{\%\ EMG\ RMS\ (muscle)}{\%\ EMG\ RMS_{BF} + EMG\ RMS_{SM} + EMG\ RMS_{ST}} \times 100$$

2.5. Estimation of muscle torque-generating capacity

2.5.1. Magnetic resonance imaging (MRI)

Participants were positioned supine in the MRI scanner (MRI; 1.5 T, Intera Achieva, Philips, Amsterdam, Netherlands), with their knees flexed at 45°. Flexible surface coils (SENSE, Philips, Amsterdam, Netherlands) were strapped to the medial and lateral sides of the knee. Moment arm was measured using a volumetric sequence (3D T1 fast field echo, 5.17 min, FOV 250×179 mm, TR/TE = 24/11.5 ms, voxel size: 1×1×2 mm, flip angle: 50°) that imaged the region comprised between the middle of the femur to the middle of the tibia. For each muscle, the knee flexor moment arm was defined as the shortest distance between the rotation center of the knee joint and the muscle line of action using a protocol described previously (2). In short, the 3D coordinates of the lateral and medial femoral epicondyles were determined, and the center of the joint was calculated as the midpoint between these two points. Then, the distal part of the hamstring muscle-tendon unit (ST, SM, BF) was outlined and the centroid of the axial slices was calculated to determine a line passing through. Then, the moment arm was considered as the shortest distance between the rotation center of the joint and the musculotendon path. Note that we considered one common moment arm for both BFsh and BFlh, as their distal tendon cannot be consistently distinguished with sufficient accuracy (41).

Muscle volume was estimated using a second MRI scan performed in a supine position, lying with hips and knees fully extended. Considering that muscles are isovolumetric, joint position did not affect muscle volume. A spine coil (15 elements, SENSE, Philips) was placed under

213 the pelvis and lower limbs to perform a volumetric sequence (3D T1 turbo fast field echo,
214 13.10 min, FOV 360 mm × 220 mm, TR/TE = 14/6.9 ms, voxel size: 0.8 × 0.8 × 2 mm, flip
215 angle: 20°). Slice thickness was 2 mm without an inter-slice gap. Contiguous MR images
216 were acquired from the iliac crest to half of the tibia to obtain images from the hamstring
217 heads (ST, SM, BFlh and BFsh) between their proximal and distal insertions. MR images of
218 the ST, SM, BFlh and BFsh were then segmented manually (Mimics, Materialise, Leuven,
219 Belgium; Fig. 1B) to calculate muscle volume (Fig. 1C).
220
221 ***2.5.2. B-mode extended field of view ultrasound***
222 Ultrasound panoramic mode (Aixplorer V10, Supersonic Imagine) was used to estimate
223 muscle fascicle length. This technique uses an algorithm that fits a series of images, allowing
224 the entire fascicles to be scanned within one continuous scan. This approach does not require
225 extrapolating the non-visible part of the fascicle (1), resulting in a more reliable assessment of
226 muscle fascicle length compared to single B-mode images (30). Participants were lying prone
227 with the hip and the knee flexed at 90° and 45°, respectively (0° = neutral position for the hip
228 and full extension for the knee). An ultrasound transducer (2–10 MHz, SL10-2, Supersonic
229 Imagine, Aix-en-Provence, France) was placed over the muscle to acquire transverse images
230 along the midline to determine the musculotendon path. Then, longitudinal scans progressed
231 along this midline in the fascicle line of action at an approximate scan speed of 2 $cm.s^{-1}$. The
232 total scan time was 10 to 15 s, and the scan was repeated for each muscle until two images
233 with visible fascicles were obtained (Fig. 1A). A segmented line (with a spline fit) was used
234 to model the fascicle and measure its length (ImageJ v1.48, National Institutes of Health,
235 Bethesda, MD, USA). One or two fascicles were measured for the BFsh, while one fascicle
236 was measured distally, medially, and proximally for the SM and BFlh. The pennation angle

was measured as the angle between the deep aponeurosis and the fascicle. The three values were averaged to obtain a representative value for the entire muscle.

2.5.3. Calculation of PCSA

The functional PCSA of each muscle was calculated as follows(32):

$$PCSA = \frac{Muscle\ volume}{Fascicle\ length} \times cosine(Pennation\ angle)$$

with PCSA in cm^2, muscle volume in cm^3, fascicle length in cm and pennation angle in rad. Because ST muscle and fascicles have the same line of action (16), its PCSA was considered as the anatomical cross-sectional area measured using MRI. The ratio of PCSA was calculated as the PCSA of the considered muscle divided by the sum of the PCSA of all hamstring muscles.

2.6. Estimation of an index of muscle torque

We considered PCSA, EMG amplitude, and moment arm to assess the difference in torque produced by the hamstring heads. An index of muscle torque was calculated as follows:

$$Index\ of\ muscle\ torque = PCSA \times moment\ arm \times normalized\ RMS\ EMG$$

where the index of muscle torque is expressed in arbitrary units (AU), PCSA in cm^2, moment arm in m and normalized RMS EMG in percentage of RMS EMG_{max}. The torque ratio was calculated as the index of torque of the considered muscle divided by the sum of the index of torque of all three muscles.

2.7. Statistics

Statistical analyses were performed using Statistica (v8, Statsoft, Tulsa, OK, USA). Distributions consistently passed the Kolmogorov–Smirnov normality test, and all data are reported as mean±SD. MVC peak torque was compared between the uninjured and injured

limb using a Student paired t-test. The effect of previous injury on RMS EMG values was tested using a repeated-measures three-way ANOVA (within-subject factors: intensity [20% and 50% MVC], limb [uninjured, injured] and muscle [ST, SM, BF]). The effect of a previous injury on muscle volume and PCSA was assessed using repeated-measures two-way ANOVAs (within-subject factors: limb [uninjured, injured] and muscle [ST, SM, BF]). When the sphericity assumption in repeated measures ANOVAs was violated (Mauchly's test), a Geisser-Greenhouse correction was used. When appropriate, post-hoc analyses were performed using the Bonferroni test. To address the main aim of the study, we compared muscle activation, PCSA and torque ratios (BF/Hams, SM/Hams, ST/Hams) between limbs using separated Student paired t-tests as the independence principle of the ANOVA was not respected. The level of significance was set at $P<0.05$.

3. RESULTS

Torque data

Peak MVC torque did not significantly differ between limbs (164.3±37.8 Nm and 171.3±28.5 Nm for the injured and uninjured limb, respectively; $P=0.20$). In turn, submaximal torque targets were similar between limbs at both 20% of MVC (32.9±7.6 Nm and 34.3±5.7 Nm for the injured and uninjured limb, respectively) and 50% of MVC (82.1±18.9 Nm and 85.7±14.2 Nm for the injured and uninjured limb, respectively).

Muscle activation

A main effect of intensity ($P<0.001$) was observed on muscle activation, with a mean hamstring activation of 14.8±7.0 % at 20% MVC and 38.3±13.3 % at 50% MVC (data for each individual muscle are detailed in Table 2). There was neither a main effect of limb ($P=0.85$) nor a main effect of muscle ($P=0.48$) on muscle activation. In addition, there was

no significant interactions between intensity and limb (P=0.39), intensity and muscle (P=0.41), limb and muscle (P=0.14) and intensity, limb and muscle (P=0.95).

The activation ratios measured during the isometric contraction performed at 20% MVC are depicted in Fig. 2A. We observed a higher SM/Hams ratio for the injured limb (38.2±11.0 %) compared to the uninjured limb (34.3±10.8 %, P=0.018). No between-limb differences were observed for BF/Hams (P=0.10) and ST/Hams (P=0.91). At 50% MVC, all ratios were comprised between 30.3±7.6 % (BF/Hams of the injured limb) and 35.4 ±6.9 % (ST/Hams of the injured limb; Fig. 2B), with no significant between-limb differences [BF/Hams (P=0.27), SM/Hams (P=0.12), and ST/Hams (P=0.90)].

Force-generating capacity

Although we observed a significant main effect of muscle (P<0.001) on volume, there was neither a main effect of limb (P=0.20) nor an interaction between limb and muscle (P=0.08). BF volume was significantly larger than SM (P<0.001) and ST (P<0.001), with no differences between SM and ST (P=0.34).

Regarding PCSA, we found a significant main effect of muscle (P<0.001), with no effect of limb (P=0.38). There was a significant interaction between limb and muscle (P=0.032). Regardless of the limb, PCSA was smaller for ST compared to both BF (P<0.001 and P<0.001 on injured and uninjured limbs, repsectively) and SM (P<0.001 and P<0.001 on injured and uninjured limbs, repsectively). In addition, BF exhibited larger PCSA than SM (P=0.031 and P<0.001 on injured and uninjured limb, respectively). Note that we ran the same analysis including BFlh and BFsh heads, and we did not observe a significant interaction between limb and muscle (P=0.063).

The BF/Hams ratio for PCSA was -3.0±6.2 % lower in the injured limb than in the uninjured limb (P=0.045). This difference was observed in 12 out of 17 (71%) of the participants as

reflected by the individual data (Fig. 3). Inversely, there was a trend, albeit non significant, for the SM/Hams ratio to be higher in the injured side compared to the uninjured side (P=0.083). No between-limb differences were observed for ST/Hams (P=0.661).

Bilateral differences in muscle coordination

When ANOVA was applied on the index of muscle torque, we observed a significant main effect of intensity (P<0.001) and muscle (P=0.005), a significant interaction between intensity and muscle (P=0.016), and a significant interaction between limb and muscle (P=0.022). There was neither a main effect of limb (P=0.88) nor an interaction between intensity and limb (P=0.57). For the sake of clarity, we report only the statistics associated with the interaction between muscle and limb, which relates to the main aim of this study. Regardless of the limb, ST produced a lower torque than both SM (P=0.006 for both limbs) and BF (P<0.001 for both limbs). The torque produced by BF was higher than that produced by SM in the uninjured limb (P=0.038), while no difference was observed between these two muscles in the injured limb (P=1.00).

We considered muscle coordination as the distribution of torque among the three heads of the hamstring muscles. The contribution of BF torque over the total hamstring torque (BF/Hams) was lower in the injured than in the uninjured limb at 20% MVC (-10.8 ± 27.5%; P=0.038; Fig. 4A). Inversely, the contribution of SM (SM/Hams) was higher in the injured than in the uninjured limb (+17.2 ± 27.4%; P=0.007; Fig. 4A). No between-limb differences were observed for ST/Hams. Notably, 13 participants (76%) presented a lower BF/Hams ratio associated with a higher SM/Hams ratio in the injured than in the uninjured limb. At 50% of MVC, only SM/Hams was higher in the injured compared to injured limb (+12.5 ± 21.3%; P=0.035). No significative differences were observed for BF/Hams (P=0.13) and ST/Hams (P=0.92; Fig. 4B).

4. DISCUSSION

This study aimed to determine whether coordination between hamstring muscles differs between an injured and uninjured limb in elite athletes with a history of unilateral hamstring strain injury. Our experimental approach considered muscle activation measured during an isometric task, muscle PCSA, and muscle moment arm to estimate an index of torque for each muscle. Our results highlight different coordination strategies between limbs, with a lower contribution of the injured muscle (BF) to total knee flexion torque compared to the uninjured limb. This reduced contribution of BF was compensated by a higher contribution of the SM muscle in the injured limb. These changes observed in the injured limb resulted from changes in activation of SM and/or the muscle force-generating capacity of BF muscle. These specific adaptations were observed after the completion of rehabilitation and when the participants no longer reported pain and were able to sprint. These results have clinical relevance as they provide evidence that substantial bilateral differences in hamstring coordination persist at the return to regular training. According to pain and injury adaptation theories, these changes may have long-term negative consequences.

Methodological considerations

Some methodological considerations should be kept in mind when interpreting the present data. First, muscle activation was assessed using surface EMG in a bipolar configuration. In order to minimize crosstalk, we used B-mode ultrasound to ensure similar electrode locations between participants, away from the border of neighboring muscles and aligned with the fascicle line of action. In a recent study, we showed that this procedure provides reliable measurements of activation between days (2). The normalization procedure is also crucial to accurately compare activation level between muscles and participants. Using the twitch

360 interpolation method, previous studies have reported that young healthy participants are able
361 to achieve near-complete activation of their hamstrings (e.g., 98.4±0.9% in Kirk et al. (24)).
362 Also, we found similar MVC torque values between legs in this study. We can therefore
363 reasonably assume that the hamstring muscles of both legs were fully activated during the
364 maximal isometric contractions.
365 Second, although we considered two important mechanical factors (i.e., PCSA and moment
366 arm), which influence torque-generating capacity during submaximal isometric contractions,
367 we did not consider specific tension or the individual muscle force–length relationship.
368 However, to date, there is no experimental technique available to accurately measure these
369 mechanical factors for the hamstrings. In addition, specific tension varies only marginally
370 between muscles with similar fiber type composition (14), especially at low contraction
371 intensity during which type I fibers are preferentially recruited. Given that hamstring muscles
372 share a similar function (39) and that the force-length properties of human skeletal muscles
373 may reflect the requirements imposed by daily activities (19), we considered each muscle as
374 acting at a comparable length relative to their optimal length.
375 Third, as our experimental tasks involved isometric contractions, our results cannot be
376 extrapolated to dynamic tasks. Of note, accurate estimation of force during dynamic tasks
377 remains challenging, if not impossible. Although musculoskeletal modeling may provide an
378 estimation of individual muscle forces during dynamic tasks, use of such modeling is limited
379 within the context of muscle injury. This is because most of the models make an *a priori*
380 assumption that muscles forces are optimally redistributed after injury (34), which is not
381 necessarily true. Recent evidence demonstrates that adaptations in muscle coordination are
382 not predictable as they do not follow any optimization rules or a stereotypical response (10,
383 20, 31). Changes in muscle coordination can occur on the uninjured limb even after a
384 unilateral alteration of force-generating capacity, mostly because of changes in motor control

385 in both limbs (5, 6). Such a cross-sectional design therefore precludes the possibility of
386 considering coordination of the contralateral limb as a 'pre-injury' status and in turn prevents
387 us from making any conclusions regarding a causal association between injury and muscle
388 coordination observed in the injured limb. With these considerations in mind, we interpreted
389 the differences in the hamstring coordination as between-limb differences rather than post-
390 injury adaptations.
391
392 **Bilateral differences in muscle activation**
393 Theories about motor adaptation to pain and injury have proposed that movement is altered in
394 order to decrease the threat of further pain or reinjury (20). The only way for the central
395 nervous system to adapt movement is to alter muscle activation. Our results did not show
396 significant differences in the activation of the injured (BF) muscle compared to the uninjured
397 limb (Table 2). Previous research has also suggested that BF activation is reduced or
398 unchanged following injury (28, 38). Such results were obtained during eccentric maximal
399 contractions that involve a specific neural control more prone to elicit neuromuscular
400 inhibition at both the supraspinal and spinal levels compared to concentric or isometric tasks
401 (12). Alternatively, these discrepancies may reflect that injury may not only alter the
402 activation of the injured muscle but also the relative contribution of other muscle synergists,
403 as a compensatory mechanism against neuromuscular inhibition (9). Here, we focused on the
404 muscle activation ratio to estimate the contribution of each muscle head to total hamstring
405 activation. Given that hamstring muscles have redundant contributions to knee flexor torque,
406 submaximal isometric contractions could be achieved using multiple combinations of
407 muscles. We observed an increased contribution of the activation of an uninjured synergist
408 muscle (SM), which is likely compensating for a decreased contribution in activation from the
409 injured muscle (BF), albeit non-significant (bilateral difference in BF/Hams: P=0.10). Of

note, a lower BF/Hams ratio was observed in the injured limb in 11 out of 17 participants. Changes in the ratios of muscle activation have also been observed during a Nordic hamstring exercise performed by previously injured athletes(4). Specifically, they found a greater contribution of the BF in total hamstring activation during the late phase of the Nordic hamstring, which is not consistent with our results. However, it is difficult to interpret these changes regarding the injury because the injured muscle was not specified. The Nordic hamstring is an eccentric-biased (i.e., with a specific neural control) bilateral near-maximal task, which offers less degree of freedom to change muscle activation.

The differences in activation ratios among hamstring muscles can be discussed within the context of current motor control theories. The optimal feedback control theory suggests that the activation strategies adopted by the central nervous system aim to minimize a cost and/or maximize a benefit (40). In the context of pain and injury, unloading the injured muscle, as suggested by previous studies (28, 38), can be considered as a benefit. This unloading was compensated with an increased SM activation, which seems to be an efficient strategy. Indeed, the metabolic cost associated with force generation is related to the activated volume of muscle to generate a given force. Given that muscle force is generally proportional to the cross-sectional area of activated fibers, longer-fibered muscles require a larger activated volume to generate a given force (3). This means that the SM may have a lower ATP consumption per unit of force generated compared to the ST. Therefore, differences in SM/Hams activation ratios may result from an optimization process initiated by the central nervous system at the time of injury (10, 20). Alternatively, each individual might use ‘motor habits’, i.e., a set of valid distributions of activations to perform the task without necessarily minimizing cost (25, 31). In the context of muscle injury, the distribution of activations might result from a rescaling of the original muscle activity, which is not reoptimized despite the deficit in force-generating capacity observed in the injured muscle (31). This could explain

why some participants (6 out of 17) did not exhibit any change in BF/Hams activation ratios. At 50% of MVC, activation ratios were not different between legs, likely because a higher activation of the hamstring muscles is required to perform the task (8, 21). During such tasks, fewer degrees of freedom are available to modify the activation distribution while maintaining the goal of the task.

Coupling between muscle activation and PCSA differences

Despite a similar PCSA for the whole hamstring group between limbs, we found that the BF/Hams ratio of PCSA was lower in the injured limb than in the uninjured limb (P=0.045). In other words, the previously injured muscle accounted for a lower proportion of the total hamstring PCSA. An opposite trend (albeit non-significant) was observed for SM (P=0.08). The observed reduction in PCSA seems more likely attributable to a reduction in the volume of BF as reflected by the similar relative differences in both parameters and the lack of changes in pennation angle (Table 3). This is constant with previous findings of selective atrophy of the BFlh up to 23 months after injury (37) or at 6 months after the return to play (33). Note that the BF was the injured muscle in most of the participants (72 to 85%) in the later studies (33, 37).

Due to its cross-sectional design, the present study cannot determine whether the observed bilateral differences in both muscle activation and PCSA distribution is a contributing factor or result from injury. For example, it is possible that a prolonged reduction in activation might result in the atrophy of the injured muscle, even after a rehabilitation program. Subsequently, the volume of the non-activated part of the muscle might decrease due to insufficient mechanical stimuli, resulting in atrophy of the whole muscle. Alternatively, these differences in activation and PCSA between the legs could have been present before the injury. However, asymmetry in hamstring volume has not been reported for active people (2) or sprinters with

460 no previous injury (17). Moreover, we observed similar hamstring activation ratios across
461 legs during submaximal isometric knee flexion in healthy controls (2). Thus, between-limb
462 differences in activation and PCSA have only been reported in previously-injured athletes,
463 which suggest that the injury might be the cause of such alterations (33, 37). Further
464 prospective investigations are needed to test this assumption.
465
466 **Individual hamstring coordination and their functional consequences**
467 Our results provide strong evidence of different force-sharing strategies in an injured versus
468 an uninjured limb. Adaptations in muscle coordination after a hamstring injury have been
469 suggested, using indirect measures such as functional MRI (35, 36) and surface EMG (4, 9).
470 Here the index of muscle torque provided a more direct assessment of muscle coordination
471 than activation alone (22). At 20% of MVC, we found the BF/Hams torque ratio to be lower
472 (-10.8±20.7%) and the SM/Hams torque ratio higher (+17.2±27.4%) in the injured than in the
473 non-injured limb. A large majority of participants adopted this strategy (13 out 17
474 participants). Although the origin of such differences remains unknown, it might have
475 functional consequences. A force deficit in the injured muscle could decrease its capacity to
476 sustain high mechanical loading, and in turn increase its susceptibility to damage (27). Data
477 from animal models have also shown that the greater the force produced by a muscle, the
478 higher its energy absorption before failure and injury (15).
479 Therefore, strengthening the injured muscle could be a primary target of rehabilitation
480 programs to adjust toward a balanced contribution of hamstring heads to total torque in order
481 to reduce the risk of reinjury. Crossley et al. (7) have shown that muscle coordination could
482 be durably changed in patients suffering from patellofemoral pain using an appropriate
483 rehabilitation program. In addition, recent studies demonstrated muscle- and regional-specific
484 activations within hamstring in response to various strengthening exercises (18). For instance,

hip extension or flywheel curl exercises could be proposed to selectively activate and strengthen BF muscle. Whether the chronic effects elicited by such individualized training could participate to level the contribution of the pre-injured muscle to total hamstring torque remains to be investigated. These research questions open promising perspectives for well-trained athletes (as those included in the present study) particularly exposed to the detrimental effects of hamstring strain injuries.

5. CONCLUSION

Previously injured athletes have bilateral differences in hamstring coordination. During submaximal knee flexions performed at 20% of MVC, the injured BF muscle contributed less to the total knee flexor torque than the same muscle in the uninjured limb; and this was compensated by a larger contribution of the SM muscle, also observed at 50% of MVC. These changes in muscle coordination were attributed to changes in muscle force-generating capacity and/or activation. These bilateral differences in hamstring coordination raises the question of its long-term impact on hamstring morphology and mechanics. Further studies are required to determine whether these adaptations to initial injury could constitute an intrinsic risk factor that contributes to the high rate of hamstring injury recurrence.

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No conflicts of interest, financial or otherwise, are declared by the authors. Authors declare that they have no conflicts of interest relevant to the content of this original research article.

TABLES:

Table 1- Demographics and injury characteristics of study participants. *The grade refers to the classification of the Munich consensus statement. Injury-to-test time represents the number of days between the injury occurrence and the experiment. BFlh: Biceps femoris long head. ST: Semitendinosus*

Participant	Age (yr)	Height (m)	Body mass (kg)	Injury site (side)	Grade	Rehabilitation duration (days)	Injury-to-test time (days)
1	25	1.78	65	BFlh (Right)	2	21	41
2	25	1.78	74	BFlh (Left)	3	42	141
3	24	1.78	68	BFlh (Right)	2	21	40
4	26	1.85	89	BFlh (Right)	2	14	62
5	38	1.84	70	BFlh/ST (Left)	2	21	69
6	33	1.84	77	BFlh (Right)	2	28	57
7	33	1.89	90	BFlh (Right)	2	21	113
8	24	1.84	86	BFlh (Left)	2	14	82
9	33	1.72	70	BFlh (Right)	2	56	183
10	27	1.79	73	BFlh (Right)	2	35	102
11	20	1.78	72	BFlh (Right)	2	14	94
12	31	1.70	69	BFlh/ST (Right)	3	42	78
13	23	1,80	69	BFlh (Left)	2	42	113
14	23	1,84	83	BFlh (Left)	2	28	83
15	18	1,73	65	BFlh (Right)	2	63	198
16	21	1,75	68	BFlh (Right)	3	70	192
17	23	1,78	78	BFlh/ST (Right)	2	21	22

Table 2. Normalized EMG RMS measured in injured and uninjured limb during submaximal isometric knee flexions performed at 20% and 50% of the peak torque produced during maximal voluntary contraction (MVC). *BF, biceps femoris; SM, semimembranosus; ST, semitendinosus.*

	INJURED LIMB			**UNINJURED LIMB**		
	BF (% max)	SM (% max)	ST (% max)	BF (% max)	SM (% max)	ST (% max)
20% MVC	13.0±6.0	18.0±8.2	14.8±7.2	14.1±7.6	15.4±8.0	13.5±4.4
50% MVC	34.7±12.8	39.9±14.5	39.6±9.5	37.7 ±15.6	38.5±17.7	39.5±9.1

Table 3 – Muscle architecture*. Fascicle length, pennation angle, muscle volume, physiological cross-sectional area (PCSA) and moment arm for injured and uninjured limb. Statistics are only reported for muscle × limb interaction.*
[a] Indicates a significant difference with BF, [b] indicates a significant difference with SM and [c] indicates a significant difference with ST.

	INJURED LIMB				UNINJURED LIMB			
	BFsh	**BFlh**	**SM**	**ST**	**BFsh**	**BFlh**	**SM**	**ST**
FL (CM)	11.9±1.7	11.9±1.0	9.8±1.1		11.9±1.1	11.8±1.7	9.9±1.3	
PA (°)	14.1±3.0	9.4 ±1.0	11.6±2.1		13.3±2.5	10.1±1.6	11.4 ±2.0	
	BF		**SM**	**ST**	**BF**		**SM**	**ST**
VOLUME (CM^3)	433.3±75.0		333.2±78.8	355.8±89.9	439.8±73.2		320.8±68.1	346.3±83.1
PCSA (CM^2)	35.9±7.4 [bc]		33.7±8.4 [ac]	19.2±4.8 [ab]	36.9 ±7.7 [bc]		32.2±7.3 [ac]	18.9±5.2 [bc]
MOMENT ARM (CM)	5.0±0.3		4.9 ±0.5	5.8±0.6	4.9±0.4		4.8±0.5	5.8±0.7

FIGURES

Figure 1 – Individual example of muscle architecture measurements. *A. Panoramic ultrasound image of the biceps femoris long head (BFlh) muscle. This image was used to calculate BFlh fascicle length. The yellow arrows indicate a fascicle. B. Individual example of MRI slice where each muscle was segmented. BFsh, biceps femoris short head; BFlh, biceps femoris long head; SM, semimembranosus; ST, semitendinosus. The volumes of all slices were then summed to obtain muscle volume (SM on panel C.)*

Figure 2 – Ratios of activation for hamstring muscles for the uninjured (black scatters) and injured (white scatters) limb. *The ratios of EMG RMS were estimated during submaximal isometric knee flexions performed at 20% and 50% of the peak torque produced during maximal voluntary contraction (MVC). * Indicates a significant difference between limb (P<0.05).*

Figure 3 – Ratios of physiological cross-sectional area (PCSA) for the uninjured (black scatters) and injured (white scatters) limb. ** Indicates a significant difference between limbs (P>0.05).*

Figure 4 – Torque ratios for the uninjured (black scatters) and injured (white scatters) limb. *The ratios of torque were estimated during submaximal isometric knee flexions performed at 20% and 50% of the peak torque produced during maximal voluntary contraction (MVC).*
** Indicates a significant difference between limbs (P<0.05).*

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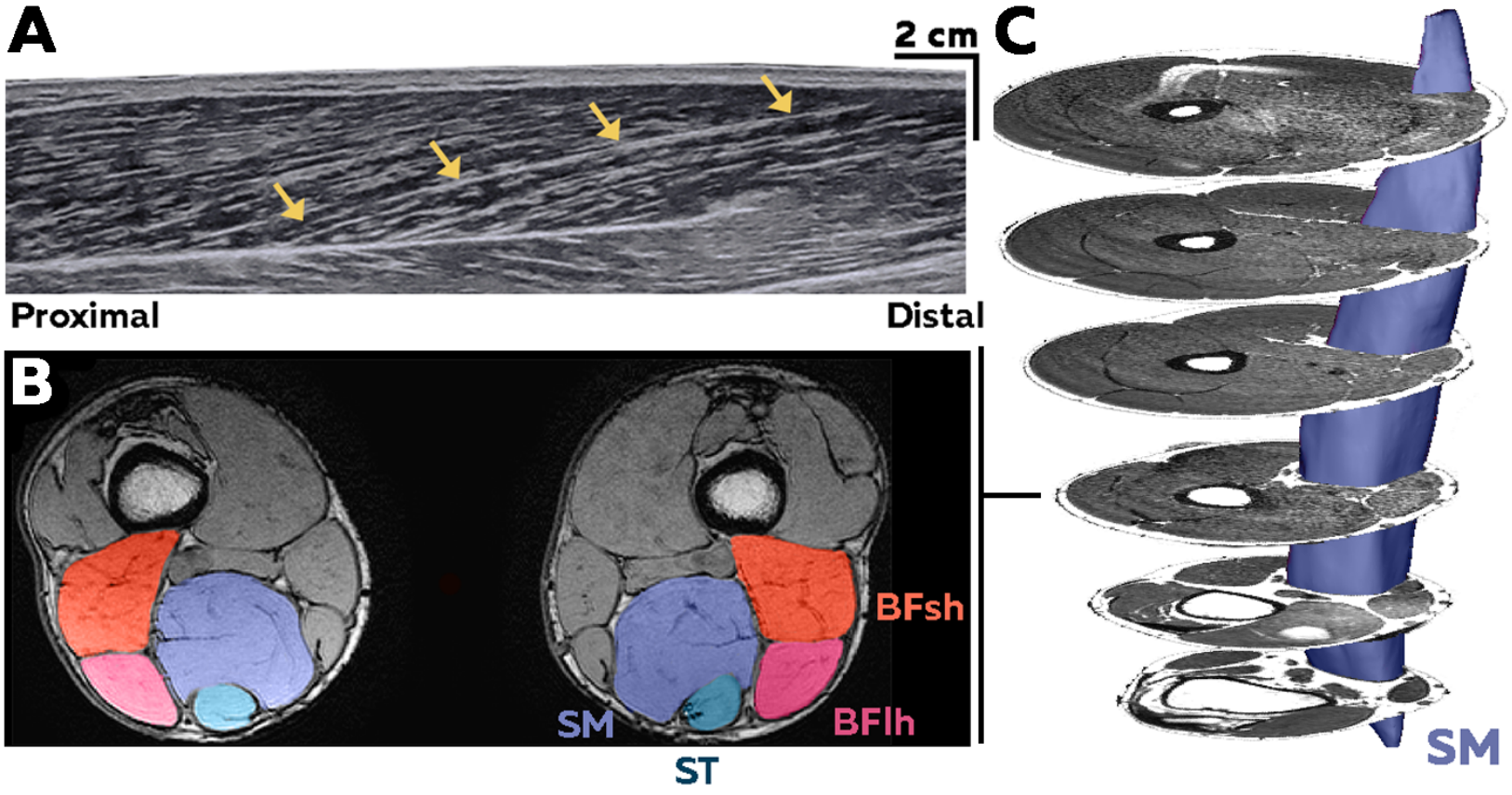
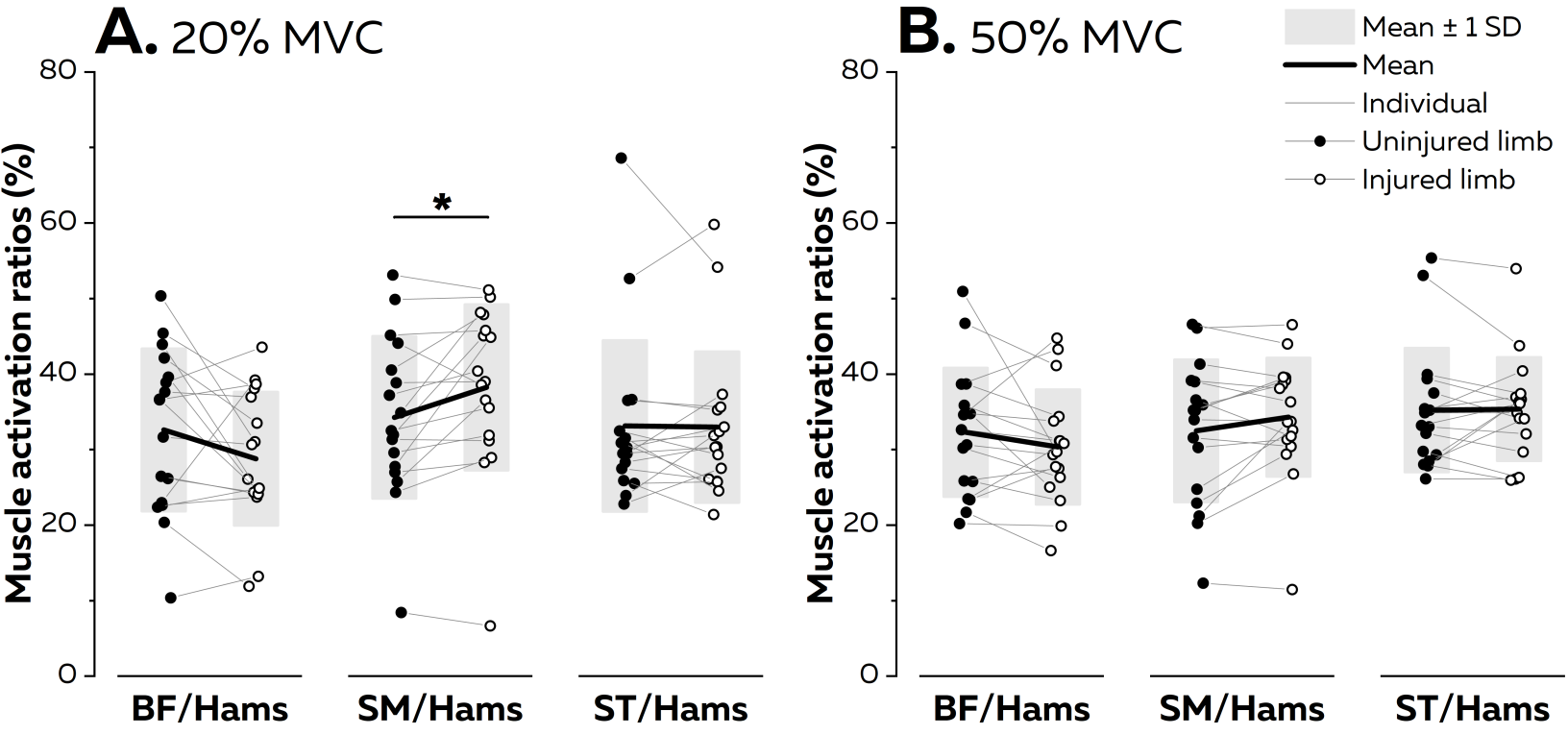
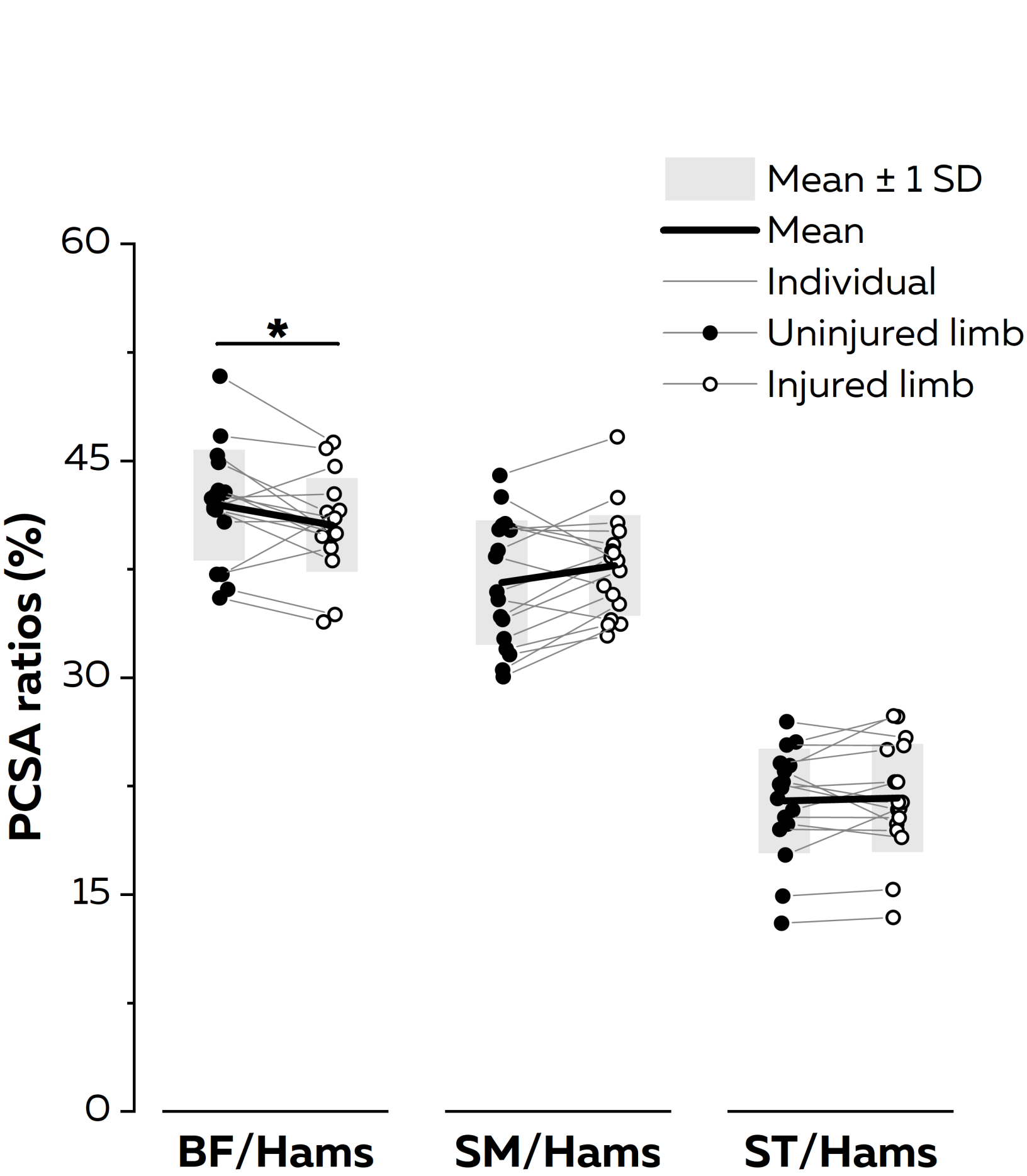
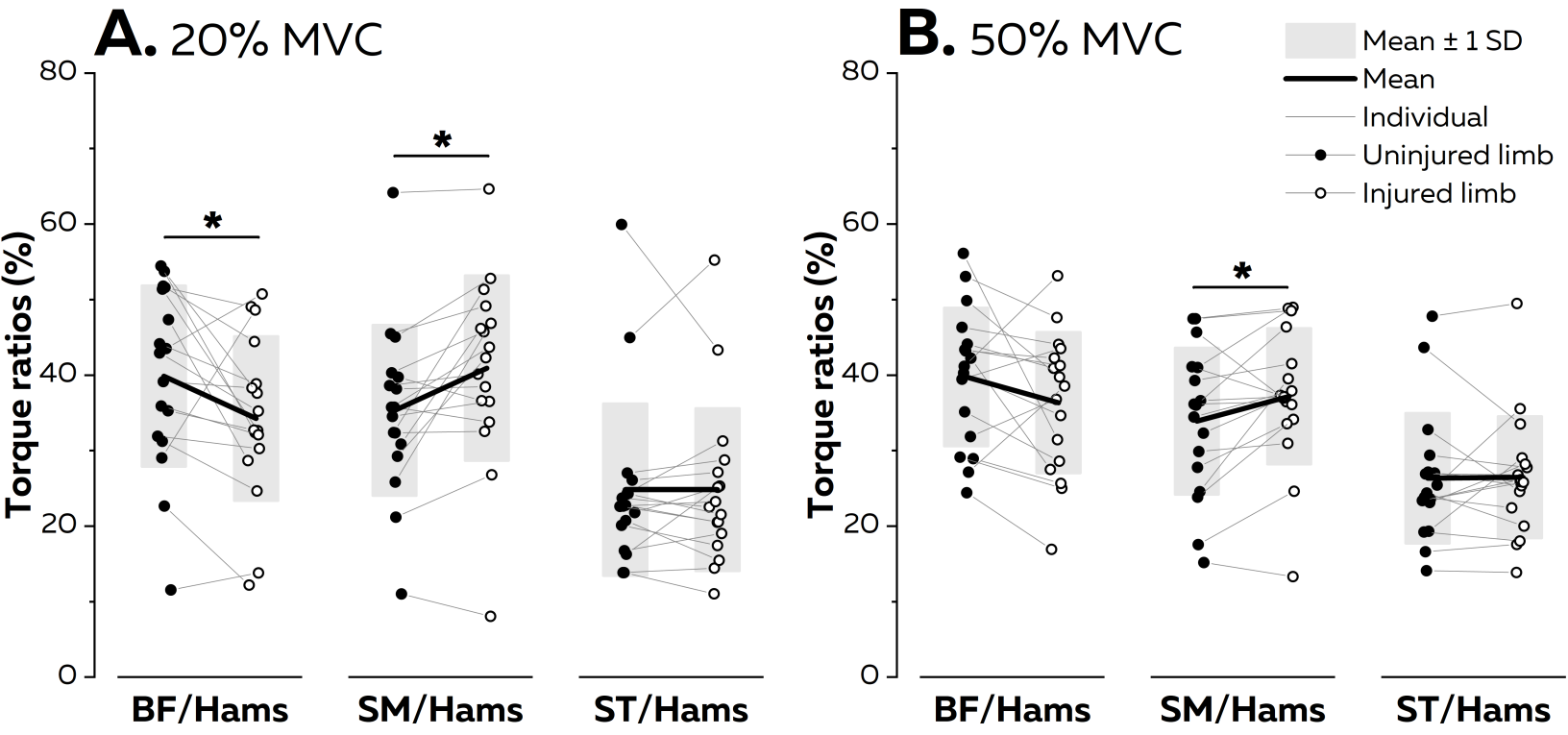

A
2 cm
Proximal
Distal
B
BFsh
SM
BFlh
ST
C
SM


A. 20% MVC
B. 50% MVC
Mean ± 1 SD
Mean
Individual
Uninjured limb
Injured limb
Muscle activation ratios (%)
80
60
40
20
0
*
BF/Hams
SM/Hams
ST/Hams


Mean ± 1 SD
Mean
Individual
Uninjured limb
Injured limb
PCSA ratios (%)
60
45
30
15
0
*
BF/Hams
SM/Hams
ST/Hams


A. 20% MVC
B. 50% MVC
Mean ± 1 SD
Mean
Individual
Uninjured limb
Injured limb
Torque ratios (%)
0
20
40
60
80
*
BF/Hams
SM/Hams
ST/Hams