J Appl Physiol 128: 688–697, 2020.
First published February 6, 2020; doi:10.1152/japplphysiol.00411.2019.

RESEARCH ARTICLE

Bilateral differences in hamstring coordination in previously injured elite athletes

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Submitted 12 June 2019; accepted in final form 3 February 2020

Avrillon S, Hug F, Guilhem G. Bilateral differences in hamstring coordination in previously injured elite athletes. *J Appl Physiol* 128: 688–697, 2020. First published February 6, 2020; doi:10.1152/japplphysiol.00411.2019.—Hamstring strain injuries (HSIs) involve tissue disruption and pain, which can trigger long-term adaptations of muscle coordination. However, little is known about the effect of previous HSIs 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. Seventeen athletes with a unilateral history of biceps femoris (BF) injury participated in the experiment. Surface electromyography was recorded from three hamstring muscles [BF, semimembranosus (SM), and semitendinosus] during submaximal isometric torque-matched tasks at 20% and 50% of maximal voluntary contraction. The product of normalized electromyographic amplitude with functional physiological cross-sectional area (PCSA) and moment arm was considered as an index of individual muscle torque. The contribution of the injured muscle to total knee flexion torque was lower in the injured than the uninjured limb ($-5.6 \pm 10.2\%$, $P = 0.038$). This reduced contribution of BF was compensated by a higher contribution of the SM muscle in the injured limb ($+5.6 \pm 7.5\%$, $P = 0.007$). These changes resulted from a lower contribution of PCSA from the injured muscle (BF) and a larger contribution of activation from an uninjured synergist muscle (SM). In conclusion, 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. We tested athletes with a history of hamstring injury. We observed a lower contribution of the injured biceps femoris to the total knee flexor torque in the injured limb than in the contralateral limb. This decreased contribution was mainly due to selective atrophy of the injured biceps femoris muscle and was compensated by an increased activation of the semimembranosus muscle.

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atrophy; hamstring injury; muscle activation; muscle coordination; torque-sharing strategies

INTRODUCTION

A hamstring strain injury is a leading cause of unavailability for training and competition in numerous sports (35). Because of their high incidence and reinjury rate (37), prevention is a main challenge for both coaches and clinicians. Most of these strain injuries involve tissue disruption in the biceps femoris long head (BFlh) muscle (16). These lesions are associated with pain and functional losses due to mechanical alterations. In addition, changes in muscle activation and neuromuscular inhibition may occur (18, 41).

Some theories propose that movement is modified in the presence of pain to unload the painful/injured tissue (25, 30). Although the unloading of the injured muscle seems logical during the acute phase of hamstring strain injury (41), 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 (25, 50).

Muscle coordination relates to the distribution of force among individual muscles to produce a given motor task (27). As such, the study of muscle coordination requires the consideration of individual muscle force rather than muscle activation alone (27), 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 (46) and their similar fiber-type content (19). With the use of this approach, Avrillon et al. (2) reported large interindividual variability in muscle coordination strategies between the ham-

string muscles, with specific effects on motor performance, i.e., the higher the difference in activation between synergist muscles, the lower the endurance time.

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. (44) reported a selective decrease in volume of the injured muscle (BF in most of the participants), 6 mo 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. (42, 43) reported a larger contribution of BF and semimembranosus (SM) muscles compared with semitendinosus (ST), up to 2 yr after an injury, but did not consider muscle volume or PCSA. In addition, these studies provide fragmented information about the injury localization, making it difficult to interpret the observed adaptations.

Here, we assessed muscle coordination in elite athletes, with a hamstring injury in the previous 7 mo, who have returned to sport. We tested the hypothesis that the contribution of the injured muscle to submaximal knee flexion torque will be reduced in the injured limb compared with the noninjured limb. This reduced contribution would be a combination of both a smaller volume and a lower activation of the injured muscle compared with uninjured muscles.

METHODS

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). 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.

Injury History

All participants had a unilateral strain injury of the biceps femoris long head (BFlh) within the past 7 mo. Note that the semitendinosus (ST) was also involved in the injury of three athletes (Table 1). The average delay between injury occurrence and testing was 98.2 ± 53.3 days (range 22–198 days). None of the participants had a history of hamstring injury in the contralateral leg within the previous 2 yr, and four participants had a history of grade I injury in the contralateral leg, 3–7 yr before testing.

We defined a hamstring injury as an acute pain in the posterior thigh that occurred during a running sprint and resulted in the immediate termination of the training session or competition. Each injured athlete underwent magnetic resonance imaging (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 were 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. Prior to testing, elite athletes attended an inclusion visit (i.e., medical exam conducted by a physician), during which they were asked to report their pain experience during maximal tasks. None of them reported pain.

Protocol

Participants attended three sessions within the same week in a randomized order: *1*) an MRI session to estimate both muscle volume and muscle moment arm, *2*) an ultrasound session to estimate fascicle length and pennation angle, and *3*) an experimental session during which muscle activation was assessed using surface electromyography (EMG). Specifically, participants sat on an isokinetic dynamometer (Con-Trex; CMV AG, Dübendorf, Switzerland) with noncompliant straps placed around the chest, pelvis, and thigh. The hip and knee were flexed at 90° and 45°, respectively (0° = neutral position for the hip and full extension for the knee). The torque signal from the isokinetic dynamometer was recorded and digitized by a USB data acquisition module (DT9804; Data Translation, Marlboro, MA) at 1,000 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.

Estimation of Muscle Activation

Experimental tasks. After a standardized warmup (10 isometric knee flexions at 50% of peak torque and 5 isometric knee flexions at

Table 1. *Demographics and injury characteristics of study participants*

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

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.

80% of peak torque), participants performed three maximal voluntary contractions (MVCs) of the knee flexors for 3–5 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.

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 (BFsh) is close to the popliteal fossa, it was not possible to investigate this muscle. To our knowledge, only a few studies considered the activation of each of the two heads of the biceps femoris during maximal knee flexions using intramuscular recordings (13, 33, 34). Whereas intramuscular electrodes are suitable to measure EMG activity in a small muscle region, this method cannot be used to provide information about the activation of a whole muscle (3). Consequently, we followed the Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles (SENIAM) recommendations, and we placed the electrodes in the middle of the biceps femoris muscle belly. Taking advantage of the lack of selectivity of classical bipolar EMG recordings (3), we considered that the EMG amplitude reflected the activation of both heads.

The skin was shaved and cleaned with alcohol, and a pair of Ag/AgCl electrodes (recording zone area: 520 mm^2, BlueSensor N-00-S; Ambu, Copenhagen, Denmark) was attached to the skin with an interelectrode distance of 20 mm (center to center). Raw EMG signals were preamplified (gain: 1,000), band-pass filtered (10–500 Hz, third-order Butterworth filter), and sampled at 2,000 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, Marlborough, MA).

Data processing. All mechanical and EMG data were analyzed using MATLAB custom-written scripts (R2017a; The Mathworks, Natick, MA). 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. 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.

Estimation of Muscle Torque-Generating Capacity

Magnetic resonance imaging. Participants were positioned supine in the MRI scanner (MRI; 1.5 T, Intera Achieva; Philips, Amsterdam, The Netherlands), with their knees flexed at 45°. Flexible surface coils (SENSE; Philips) were strapped to the medial and lateral sides of the knee. Moment arm was measured using a volumetric sequence [three dimensional (3-D) T1 fast-field echo, 5.17 min, field of view 250 × 179 mm, repetition time/echo time = 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 3-D 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 (51).

Muscle volume was estimated using a second MRI scan performed in a supine position, lying with hips and knees fully extended. With the consideration that muscles are isovolumetric, joint position did not affect muscle volume. A spine coil (15 elements, SENSE; Philips) was placed under the pelvis and lower limbs to perform a volumetric sequence (3-D T1 turbo fast-field echo, 13.10 min, field of view 360 × 220 mm, repetition time/echo time = 14/6.9 ms, voxel size: 0.8 × 0.8 × 2 mm, flip angle: 20°). Slice thickness was 2 mm without an interslice gap. Contiguous MR images were acquired from the iliac crest to one-half of the tibia to obtain images from the hamstring heads (ST, SM, BFlh, and BFsh) between their proximal and distal insertions. MR images of the ST, SM, BFlh, and BFsh were then segmented manually (Mimics; Materialise, Leuven, Belgium; Fig. 1*B*) to calculate muscle volume (Fig. 1*C*).

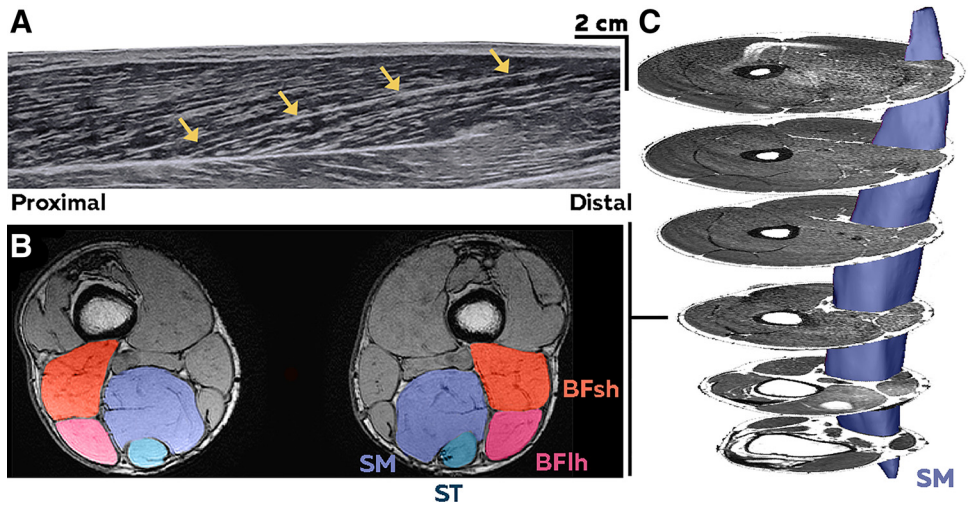

Fig. 1. Individual example of muscle architecture measurements. *A*: panoramic ultrasound image of the biceps femoris long head (BFlh) muscle. This image was used to calculate BFlh fascicle length. The yellow arrows indicate a fascicle. *B*: individual example of MRI slice where each muscle was segmented. BFsh, biceps femoris short head; SM, semimembranosus; ST, semitendinosus. Contiguous MR images were acquired from the iliac crest to one-half of the tibia to obtain images from the hamstring heads between their proximal and distal insertions. *C*: MR images of the ST, SM, BFlh, and BFsh were then segmented manually to calculate muscle volume, i.e., the sum of the volume of all slices (SM in *C*). In this figure, 6 of the 125 segmented slices are depicted.

B-Mode-extended field-of-view ultrasound. Ultrasound panoramic mode (Aixplorer V10; Supersonic Imagine) was used to estimate muscle fascicle length. This technique uses an algorithm that fits a series of images, allowing the entire fascicles to be scanned within one continuous scan. This approach does not require extrapolating the nonvisible part of the fascicle (1), resulting in a more reliable assessment of muscle fascicle length compared with single B-mode images (38). Participants were lying prone with the hip and the knee flexed at 90° and 45°, respectively (0° = neutral position for the hip and full extension for the knee). An ultrasound transducer (2–10 MHz, SL10–2; Supersonic Imagine, Aix-en-Provence, France) was placed over the muscle to acquire transverse images along the midline to determine the musculotendon path. Then, longitudinal scans progressed along this midline in the fascicle line of action at an approximate scan speed of 2 cm/s. The total scan time was 10–15 s, and the scan was repeated for each muscle until two images with visible fascicles were obtained (Fig. 1*A*). A segmented line (with a spline fit) was used to model the fascicle and measure its length (ImageJ, v1.48; National Institutes of Health, Bethesda, MD). One or two fascicles were measured for the BFsh, whereas one fascicle 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.

Calculation of PCSA. The functional PCSA of each muscle was calculated as follows (39):

$$\text{PCSA} = \frac{\text{Muscle volume}}{\text{Fascicle length}} \times \cos\left(\text{Pennation angle}\right),$$

with PCSA in squared centimeters, muscle volume in cubic centimeters, fascicle length in centimeters, and pennation angle in radians. Because ST muscle and fascicles have the same line of action (21), their 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.

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:

$$\text{Index of muscle torque} = \text{PCSA} \times \text{moment arm} \times \text{normalized RMS EMG},$$

where the index of muscle torque is expressed in arbitrary units, PCSA in squared centimeters, moment arm in centimeters, and normalized RMS EMG in percentage of maximal activation level. 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.

Statistics

Statistical analyses were performed using Statistica (v8; Statsoft, Tulsa, OK). Distributions consistently passed the Kolmogorov-Smirnov normality test, and all data are reported as means ± SD. MVC peak torque was compared between the uninjured and injured limb using a Student's 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 Greenhouse-Geisser correction was used. When appropriate, Bonferroni post hoc analyses were performed. To address the main aim of the study, we compared muscle activation, PCSA, and torque ratios [BF, SM, and ST torque over the total hamstring torque (BF/Hams, SM/Hams, ST/Hams, respectively)] between limbs using separated Student's paired *t* tests, as the independence principle of the ANOVA was not respected. The level of significance was set at $P < 0.05$.

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 normalized EMG amplitude, 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 estimated during the isometric contraction performed at 20% MVC are depicted in Fig. 2*A*. We observed a significantly higher SM/Hams ratio for the injured limb compared with the uninjured limb {+4.0 ± 6.3% [95% confidence interval (CI95%): +0.8%, +7.2%], $P = 0.018$, $d = 0.37$}. No between-limb differences were observed for BF/Hams ($P = 0.10$, $d = -0.39$) and ST/Hams ($P = 0.91$, $d = -0.02$). 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. 2*B*), with no significant

Table 2. *Normalized EMG RMS measured in injured and uninjured limbs during submaximal isometric knee flexions performed at 20% and 50% of the peak torque produced during maximal voluntary contraction*

	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

BF, biceps femoris; EMG, electromyography; Max, maximal; MVC, maximal voluntary contraction; RMS, root mean square; SM, semimembranosus; ST, semitendinosus.

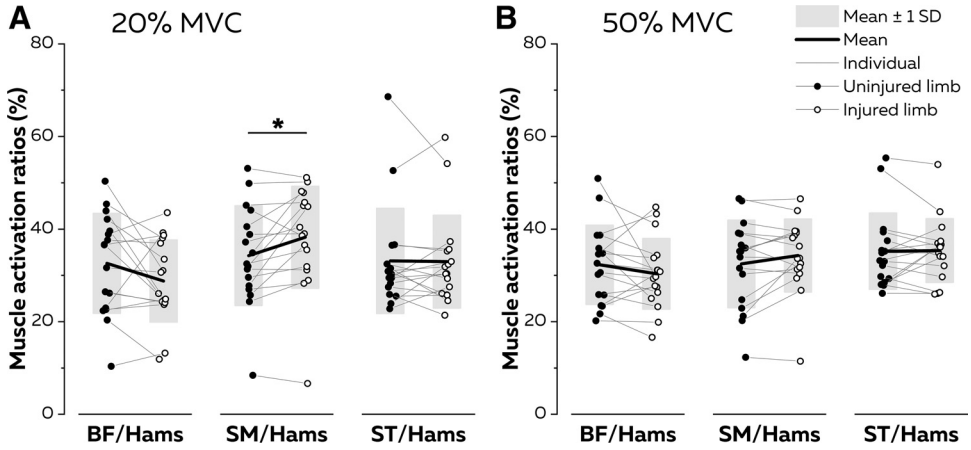

Fig. 2. Ratios of activation for hamstring muscles [biceps femoris (BF), semimembranosus (SM), and semitendinosus (ST) activation over the total hamstring activation (BF/Hams, SM/Hams, ST/Hams, respectively)] for the uninjured (black scatters) and injured (white scatters) limb. The ratios of electromyography root mean square were estimated during submaximal isometric knee flexions performed at 20% (*A*) and 50% (*B*) of the peak torque produced during maximal voluntary contraction (MVC). *$P < 0.05$, significant difference between limb.

between-limb differences [BF/Hams ($P = 0.27$, $d = -0.24$), SM/Hams ($P = 0.12$, $d = 0.21$), and ST/Hams ($P = 0.90$, $d = 0.02$)].

Physiological Cross-Sectional Area

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 ($P < 0.001$, $d = 1.51$) and ST ($P < 0.001$, $d = 1.08$), with no significant differences between SM and ST ($P = 0.34$, $d = -0.3$).

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, ST PCSA was smaller than BF PCSA (injured limb: $P < 0.001$, $d = -2.69$; uninjured limb: $P < 0.001$, $d = -2.74$). ST PCSA was also smaller than SM PCSA (injured limb: $P < 0.001$, $d = -2.13$; uninjured limb: $P < 0.001$, $d = -2.09$). In addition, BF exhibited larger PCSA than SM ($P = 0.031$, $d = 0.27$ and $P < 0.001$, $d = 0.62$ 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 ($P = 0.063$). No between-limb differences were observed for either BFsh PCSA (-0.2 ± 2.0 cm^2, $d = -0.05$) or BFlh PCSA (-0.8 ± 2.6 cm^2, $d = -0.16$).

The BF/Hams ratio for PCSA was lower in the injured limb than in the uninjured limb [$-1.4 \pm 2.6\%$ (CI95%: -2.7%, 0.0%), $P = 0.045$, $d = -0.38$]. This difference was observed in 12 of 17 (71%) of the participants, as reflected by the individual data (Fig. 3). The SM/Hams ratio of the injured side was not different for that of the uninjured side ($P = 0.083$, $d = 0.29$). No between-limb differences were observed for ST/Hams either ($P = 0.661$, $d = 0.05$).

Bilateral Differences in Muscle Coordination

When ANOVA was performed 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 (in arbitrary units; -15.6 ± 18.4, $P = 0.006$, $d = -1.02$ and -10.1 ± 19.0, $P = 0.006$, $d = -0.61$ on injured and uninjured limbs, respectively) and BF (-12.4 ± 17.2, $P < 0.001$, $d = -0.77$ and -18.5 ± 19.7, $P < 0.001$, $d = -0.87$ on injured and uninjured limbs, respectively). The torque produced by BF was higher than that produced by SM in the uninjured limb ($+8.4 \pm 26.6$, $P = 0.038$, $d = 0.37$), whereas no difference was observed between these two muscles in the injured limb ($P = 1.00$, $d = -0.17$).

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 [$-5.6 \pm 10.2\%$ (CI95%: -10.9%, -0.3%), $P = 0.038$, $d = -0.49$; Fig. 4*A*]. Inversely, the contribution of SM

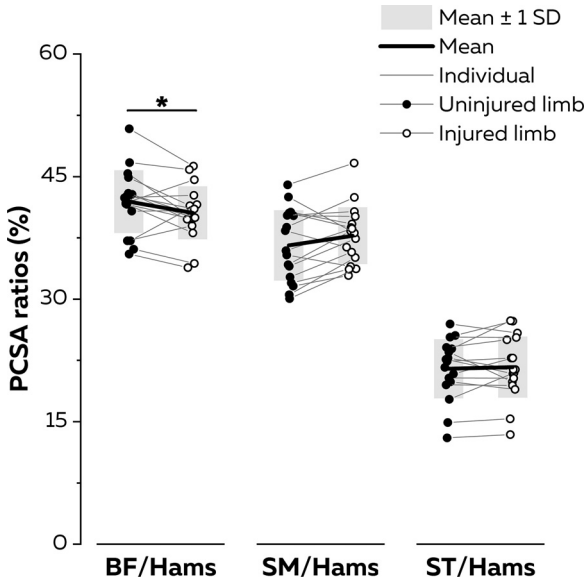

Fig. 3. Ratios of physiological cross-sectional area (PCSA) for the uninjured (black scatters) and injured (white scatters) limb. *$P > 0.05$, significant difference between limbs. BF/Hams, SM/Hams, ST/Hams, biceps femoris (BF), semimembranosus (SM), and semitendinosus (ST) PCSA over the total hamstring PCSA (BF/Hams, SM/Hams, ST/Hams, respectively.

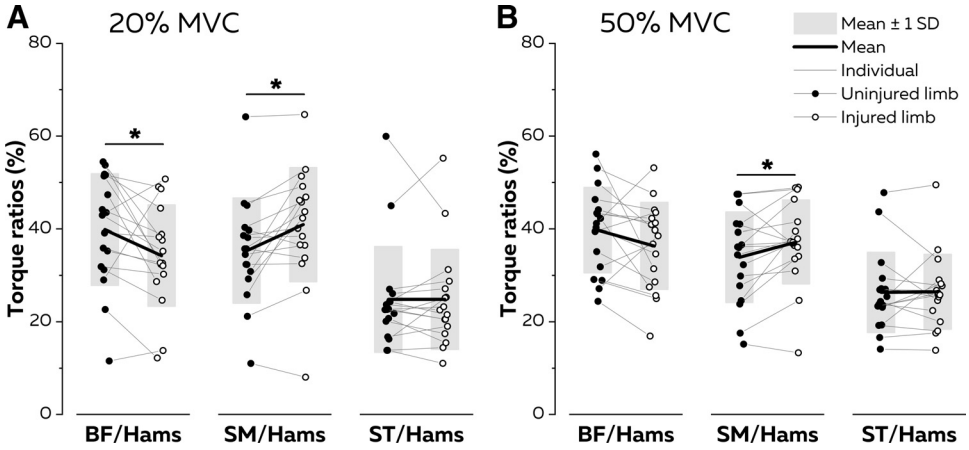

Fig. 4. Torque ratios for the uninjured (black scatters) and injured (white scatters) limb. The ratios of torque were estimated during submaximal isometric knee flexions performed at 20% (*A*) and 50% (*B*) of the peak torque produced during maximal voluntary contraction (MVC). *$P < 0.05$, significant difference between limbs. BF/Hams, SM/Hams, ST/Hams, biceps femoris (BF), semimembranosus (SM), and semitendinosus (ST) torque over the total hamstring torque, respectively.

(SM/Hams) was higher in the injured than in the uninjured limb [+5.6 ± 7.5% (CI95%: +1.7%, +9.5%), $P = 0.007$, $d = 0.47$; Fig. 4*A*]. 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 with the uninjured limb [+3.3 ± 5.9% (CI95%: +0.3%, +6.3%), $P = 0.035$, $d = 0.35$]. No significant differences were observed for BF/Hams ($P = 0.13$, $d = -0.37$) and ST/Hams ($P = 0.92$, $d = 0.02$; Fig. 4*B*).

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 with 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 the BF muscle. These specific adaptations were observed after the completion of rehabilitation when the participants no longer reported pain and were able to sprint. Together, these results 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. To minimize crosstalk, we used B-mode ultrasound to ensure similar electrode locations among 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. With the use of the twitch interpolation method, previous studies have reported that young, healthy participants are able to achieve near-complete activation of their hamstrings [98.4 ± 0.9%; Kirk et al. (28)]. As we found similar MVC torque values between legs in this study, we assumed that the hamstring muscles of both legs were fully activated during the maximal isometric contractions.

Second, there was a disparity in injury severity and time since the injury among elite athletes that likely influenced the neural and muscular contributions to the observed changes in muscle coordination. However, there was no significant correlation between injury severity or time since the injury and any of our main outcomes, i.e., hamstring muscle activation, PCSA, and peak knee flexor torque (r values ranged from −0.47 to 0.33; all P values > 0.057).

Third, although we considered two important mechanical factors (i.e., PCSA and moment arm), which influence torque-generating capacity during submaximal isometric contractions, we did not consider specific tension or the individual muscle force-length relationship. However, to date, there is no experimental technique available to accurately measure these mechanical factors for the hamstrings. In addition, specific tension varies only marginally between muscles with similar fiber-type composition (20), especially at low contraction intensity, during which type I fibers are preferentially recruited. Given that hamstring muscles share a similar function (46) and that the force-length properties of human skeletal muscles may reflect the requirements imposed by daily activities (23), we considered each muscle as acting at a comparable length relative to its optimal length.

Fourth, as our experimental tasks involved isometric contractions, our results cannot be extrapolated to dynamic tasks. Of note, accurate estimation of individual muscle force during dynamic tasks remains challenging. Although musculoskeletal modeling may provide an estimation of muscle forces during dynamic tasks, this approach presents some limitations when applied to the context of muscle injury. This is because most of the models make an a priori assumption that muscle forces are optimally redistributed (41), which is not necessarily true after injury (15, 25).

Finally, changes in muscle coordination can occur on the uninjured limb even after a unilateral alteration of force-generating capacity, mostly because of changes in motor control in both limbs (9, 10). Such a cross-sectional design therefore precludes the possibility of considering coordination of the contralateral limb as a "preinjury" status and in turn, prevents strong conclusions regarding a causal association between injury and muscle coordination observed in the injured limb. With these considerations in mind, we interpreted the differences in the hamstring coordination as between-limb differences rather than post-injury adaptations.

Bilateral Differences in Muscle Activation

In line with a recent meta-analysis (31), our results showed that previously injured elite athletes did not exhibit a significant lower isometric strength in the injured limb compared with the contralateral limb. Therefore, participants produced similar submaximal torque levels with their injured and uninjured limb during the experimental tasks.

Theories about motor adaptation to pain and injury have proposed that movement is altered to decrease the threat of further pain or reinjury (24). The only way for the central nervous system to adapt movement is to alter muscle activation. We did not find any significant difference in the activation of the injured (BF) muscle compared with the uninjured limb (Table 2). Previous research has suggested that BF activation is reduced or unchanged following injury (36, 45). Such results were obtained during eccentric maximal contractions that involve a specific neural control, more prone to elicit neuromuscular inhibition at both the supraspinal and spinal levels compared with concentric or isometric tasks (17). Here, we focused on the muscle activation ratio to estimate the contribution of each muscle head to total hamstring activation. Given that hamstring muscles have redundant contributions to knee flexor torque, submaximal isometric contractions could be achieved using multiple force-sharing strategies. We observed an increased contribution of the activation of an uninjured synergist muscle (SM), which likely compensates for a decreased contribution of the injured muscle (BF), albeit nonsignificant (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 (5, 7). Specifically, this latter study reports a greater contribution of the BF in total hamstring activation in previously injured athletes 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. In addition, Nordic hamstring is an eccentric-biased (i.e., with a specific neural control), bilateral, near-maximal task, which offers fewer degrees 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 (49). In the context of pain and injury, an unloading of the injured muscle can be considered as a benefit (36, 45). Here, we observed a larger contribution of SM activation in the injured limb, which seems to be an efficient strategy to compensate for an unloading of the injured BF. Indeed, the metabolic cost associated with force generation is related to the active muscle volume. Given that muscle force is generally proportional to the cross-sectional area of active fibers, longer-fibered muscles require a larger active volume to generate a given force (4). This means that the SM may have a lower ATP consumption per unit of force generated compared with 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 (25, 50). Alternatively, each individual might use "motor habits," i.e., a set of valid distributions of activations to perform the task without necessarily minimizing cost (15, 29). In the context of muscle injury, the distribution of activations might result from a rescaling of the pre-injury muscle activity, which is not reoptimized despite the deficit in force-generating capacity observed in the injured muscle (15). This could explain why some participants (6 of 17) did not exhibit any change in BF/Hams activation ratios.

Of note, 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 (12, 26). 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 observed a lower BF/Hams ratio of PCSA 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. This observed reduction in PCSA seems more likely attributable to a reduction in the volume of BF (Table 3). This is consistent with

Table 3. *Muscle architecture*

	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	
Volume, cm^3	161.0 ± 38.8	272.4 ± 51.7	333.2 ± 78.8	355.8 ± 89.9	159.9 ± 39.9	279.9 ± 51.4	320.8 ± 68.1	346.3 ± 83.1
PCSA, cm^2	13.3 ± 3.9	22.6 ± 4.4	33.7 ± 8.4	19.2 ± 4.8	13.5 ± 4.5	23.3 ± 5.0	32.2 ± 7.3	18.9 ± 5.2
	BF		SM	ST	BF		SM	ST
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

Fascicle length (FL), pennation angle (PA), muscle volume, physiological cross-sectional area (PCSA), and moment arm for injured and uninjured limb. BF, biceps femoris; BFlh, BF long head; BFsh, BF short head; SM, semimembranosus; ST, semitendinosus.

previous findings of selective atrophy of the BFlh, up to 23 mo after injury (44) or at 6 mo after the return to play (40). Importantly, BF was the injured muscle in most of the participants in the later studies (72–85%) (40, 44). These studies also reported a concomitant change in BFsh volume. In the present study, the BFsh PCSA was not different between limbs. However, we found a trend (albeit nonsignificant; $P = 0.08$) of a higher SM/Hams ratio of PCSA in the injured limb than in the uninjured limb. This is in line with Messer et al. (32) who found an increased SM volume in athletes who had an anterior cruciate ligament reconstruction involving the ST muscle. These results suggest that SM hypertrophy may occur as a compensatory mechanism that would counteract the BF atrophy in the injured limb. Note that in contrast to previous findings where static images with linear extrapolation technique were used (47, 48), we showed no between-limb difference in pennation angle and fascicle length for any of the investigated muscles. This discrepancy may be explained by the fact that we used extended field-of-view ultrasound measurements.

Because of its cross-sectional design, the present study cannot determine whether the observed bilateral differences in both muscle activation and PCSA distribution are 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. 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 no previous injury (22). Moreover, we observed similar hamstring activation ratios across legs during submaximal isometric knee flexion in healthy controls (2). Thus between-limb differences in activation and PCSA have only been reported in previously injured athletes, which suggests that the injury might be the cause of such alterations (40, 44). Further prospective investigations are needed to test this assumption.

Individual Hamstring Coordination and Their Functional Consequences

Our results provide strong evidence of different force-sharing strategies in an injured versus an uninjured limb. Adaptations in muscle coordination after a hamstring injury have been suggested, using indirect measures, such as functional MRI (42, 43) and surface EMG (5, 14). Here, the index of muscle torque provided a more direct assessment of muscle coordination than activation alone (27). At 20% of MVC, we found a lower BF/Hams torque ratio ($-5.6 \pm 10.2\%$) and a higher SM/Hams torque ($+5.6 \pm 7.5\%$) in the injured than in the noninjured limb. A large majority of participants adopted this strategy (13 out 17 participants). Although the origin of such differences remains unknown, it is likely to have functional consequences. Thus the strengthening of the injured muscle could be a primary target of rehabilitation programs to adjust toward a balanced contribution of hamstring heads to total torque to reduce the risk of reinjury. Crossley et al. (11) have shown that muscle coordination could be durably changed in patients suffering from patellofemoral pain using an appropriate rehabilitation program. In addition, recent studies demonstrated muscle- and regional-specific activation within the hamstring in response to various strengthening exercises (6, 8). For instance, Bourne et al. (6) showed that hip-extension exercises elicited greater BFlh hypertrophy than Nordic hamstring. Whether the chronic effects elicited by such individualized training could contribute 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.

Conclusions

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 raise 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.

ACKNOWLEDGMENTS

The authors thank Jérôme Renoux and Michel Daoud Crema for assistance with MRI data collection.

GRANTS

The Laboratory Sport, Expertise and Performance is a partner of the French-speaking network ReFORM, recognized as a Research Centre for the Prevention of Injury and Illness and the Protection of Athletes by the Olympic Committee (IOC). As a member of the IOC Medical Research Network, ReFORM has received funding from the IOC to establish long-term research program on the prevention of injuries and illnesses in sport for the protection of athlete health. S. Avrillon was supported by a scholarship funded by the French Ministry of Research. F. Hug was supported by a fellowship from the Institut Universitaire de France (IUF).

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

S.A., F.H., and G.G. conceived and designed research; S.A. performed experiments; S.A., F.H., and G.G. analyzed data; S.A., F.H., and G.G. interpreted results of experiments; S.A. prepared figures; S.A., F.H., and G.G. drafted manuscript; S.A., F.H., and G.G. edited and revised manuscript; S.A., F.H., and G.G. approved final version of manuscript.

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