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1 **Bilateral differences in hamstring coordination in previously injured elite**
2 **athletes**

3

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5

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25

26 **Running title:** Injury-induced changes in muscle coordination

27

28 **ABSTRACT (250 words)**

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

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

41 **Results:** The contribution of the injured muscle to total knee flexor torque was lower
42 compared to the uninjured limb ($-10.8 \pm 27.5\%$; $P=0.038$). This reduced contribution of BF
43 was compensated by a higher contribution of the SM muscle in the injured limb
44 ($+17.2 \pm 27.4\%$; $P=0.007$). These changes resulted from a decreased contribution of PCSA
45 from the injured muscle (BF), and an increased contribution of activation from an uninjured
46 synergist muscle (SM).

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

51

52

53 **NEW & NOTEWORTHY:**

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

61

62 **KEYWORDS**

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

65 **1. INTRODUCTION**

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

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

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

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

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

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

109

110 **2. METHODS**

111 **2.1. Participants**

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

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

121

122 **2.2. Injury history**

123 All participants had a unilateral strain injury of the BFlh within the past seven months (Table
124 1). The average delay between injury occurrence and testing was 98.2 ± 53.3 days (range 22-
125 198 days). We defined a hamstring injury as an acute pain in the posterior thigh that occurred
126 during a sprint and resulted in the immediate termination of the training session or
127 competition. Each injured athlete underwent an MRI ($n = 9$) or an ultrasound ($n = 8$) exam
128 performed by a radiologist within the week following injury. Athletes met inclusion criteria
129 when the precise localization and the grade of the injury was confirmed by the exam. Their
130 injuries caused training activities to stop for 32.5 ± 17.5 days (range 14-70 days). All athletes
131 completed a supervised rehabilitation protocol provided by a qualified physiotherapist. In the
132 absence of standardization, the rehabilitation program could slightly differ in content and
133 periodization. At the time of testing the participants were allowed to return to their regular
134 sport activities (included sprinting) by the clinical staff, had recovered to their pre-injury peak
135 knee flexor torque level, and were free of any lower limb pain.

136

137 **2.3. Protocol**

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

140 and pennation angle, and iii) an experimental session during which muscle activation was
141 assessed using surface EMG. Specifically, participants sat on an isokinetic dynamometer
142 (Con-Trex, CMV AG, Dübendorf, Switzerland) with non-compliant straps placed around the
143 chest, the pelvis and the thigh. The hip and the knee were flexed at 90° and 45°, respectively
144 (0° = neutral position for the hip and full extension for the knee). Knee angle was chosen as it
145 corresponds to the peak knee flexor torque angle, i.e., the optimal angle (23). The torque
146 signal from the isokinetic dynamometer was recorded and digitized by a USB data acquisition
147 module (DT9804; Data Translation, Marlboro, MA, USA) at 1000 Hz. Torque was corrected
148 for gravity and low-pass filtered at 20 Hz using a third-order Butterworth filter. Visual
149 feedback of the exerted torque signal was displayed on a screen placed in front of the
150 participants.

151

152 **2.4. Estimation of muscle activation**

153 **2.4.1. *Experimental tasks***

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

161

162 **2.4.2. *Surface electromyography***

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

165 beneath each muscle to ensure that there was no contact between the electrodes and the seat.
166 We used B-mode ultrasound (v10, Aixplorer, Supersonic Imagine, Aix-en-Provence, France)
167 to determine the appropriate placement of electrodes on each muscle, longitudinally with
168 respect to the muscle fascicle's alignment and away from the borders of neighboring muscles.
169 As the superficial part of the BF short head is close to the popliteal fossa, it was not possible
170 to investigate this muscle. We therefore followed the SENIAM recommendations for
171 electrode placement on BF and considered the recorded myoelectrical activity originating
172 from this pair of electrodes as being representative of both the short and long head. The skin
173 was shaved and cleaned with alcohol and a pair of Ag/AgCl electrodes (recording zone area:
174 520 mm², Blue sensor N-00-S, Ambu, Copenhagen, Denmark) was attached to the skin with
175 an inter-electrode distance of 20 mm (centre-to-centre). Raw EMG signals were pre-amplified
176 (input impedance: 20 MM, CMRR: 90 db; gain: 1000), band-pass filtered (10-500 Hz, third
177 order Butterworth filter) and sampled at 2000 Hz (Zerowire, Aurion, Milan, Italy). EMG and
178 mechanical data were synchronized using a transistor-transistor-logic pulse recorded by a 12-
179 bit analog to digital converter (DT9804, Data Translation, Marlboro, USA).

180

181 ***2.4.3. Data processing***

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

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

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

191

192 **2.5. Estimation of muscle torque-generating capacity**

193 **2.5.1. *Magnetic resonance imaging (MRI)***

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

210 Muscle volume was estimated using a second MRI scan performed in a supine position, lying
211 with hips and knees fully extended. Considering that muscles are isovolumetric, joint position
212 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, BF_{lh} and BF_{sh}) between their proximal and distal insertions. MR images of
218 the ST, SM, BF_{lh} and BF_{sh} 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 BF_{sh}, while one fascicle
236 was measured distally, medially, and proximally for the SM and BF_{lh}. The pennation angle

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

239

240 **2.5.3. Calculation of PCSA**

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

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

242 with PCSA in cm², muscle volume in cm³, fascicle length in cm and pennation angle in rad.

243 Because ST muscle and fascicles have the same line of action (16), its PCSA was considered

244 as the anatomical cross-sectional area measured using MRI. The ratio of PCSA was calculated

245 as the PCSA of the considered muscle divided by the sum of the PCSA of all hamstring

246 muscles.

247

248 **2.6. Estimation of an index of muscle torque**

249 We considered PCSA, EMG amplitude, and moment arm to assess the difference in torque

250 produced by the hamstring heads. An index of muscle torque was calculated as follows:

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

251 where the index of muscle torque is expressed in arbitrary units (AU), PCSA in cm², moment

252 arm in m and normalized RMS EMG in percentage of RMS EMG_{max}. The torque ratio was

253 calculated as the index of torque of the considered muscle divided by the sum of the index of

254 torque of all three muscles.

255

256 **2.7. Statistics**

257 Statistical analyses were performed using Statistica (v8, Statsoft, Tulsa, OK, USA).

258 Distributions consistently passed the Kolmogorov–Smirnov normality test, and all data are

259 reported as mean±SD. MVC peak torque was compared between the uninjured and injured

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

271

272 **3. RESULTS**

273 **Torque data**

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

279

280 **Muscle activation**

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

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

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

294

295 **Force-generating capacity**

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

300 Regarding PCSA, we found a significant main effect of muscle ($P<0.001$), with no effect of
301 limb ($P=0.38$). There was a significant interaction between limb and muscle ($P=0.032$).
302 Regardless of the limb, PCSA was smaller for ST compared to both BF ($P<0.001$ and
303 $P<0.001$ on injured and uninjured limbs, respectively) and SM ($P<0.001$ and $P<0.001$ on
304 injured and uninjured limbs, respectively). In addition, BF exhibited larger PCSA than SM
305 ($P=0.031$ and $P<0.001$ on injured and uninjured limb, respectively). Note that we ran the
306 same analysis including BF_{lh} and BF_{sh} heads, and we did not observe a significant
307 interaction between limb and muscle ($P=0.063$).

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

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

313

314 **Bilateral differences in muscle coordination**

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

325 We considered muscle coordination as the distribution of torque among the three heads of the
326 hamstring muscles. The contribution of BF torque over the total hamstring torque (BF/Hams)
327 was lower in the injured than in the uninjured limb at 20% MVC ($-10.8 \pm 27.5\%$; $P=0.038$;
328 Fig. 4A). Inversely, the contribution of SM (SM/Hams) was higher in the injured than in the
329 uninjured limb ($+17.2 \pm 27.4\%$; $P=0.007$; Fig. 4A). No between-limb differences were
330 observed for ST/Hams. Notably, 13 participants (76%) presented a lower BF/Hams ratio
331 associated with a higher SM/Hams ratio in the injured than in the uninjured limb. At 50% of
332 MVC, only SM/Hams was higher in the injured compared to injured limb ($+12.5 \pm 21.3\%$;
333 $P=0.035$). No significant differences were observed for BF/Hams ($P=0.13$) and ST/Hams
334 ($P=0.92$; Fig. 4B).

335

336 **4. DISCUSSION**

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

351

352 **Methodological considerations**

353 Some methodological considerations should be kept in mind when interpreting the present
354 data. First, muscle activation was assessed using surface EMG in a bipolar configuration. In
355 order to minimize crosstalk, we used B-mode ultrasound to ensure similar electrode locations
356 between participants, away from the border of neighboring muscles and aligned with the
357 fascicle line of action. In a recent study, we showed that this procedure provides reliable
358 measurements of activation between days (2). The normalization procedure is also crucial to
359 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., $98.4 \pm 0.9\%$ 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

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

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

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

440

441 **Coupling between muscle activation and PCSA differences**

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

452 Due to its cross-sectional design, the present study cannot determine whether the observed
453 bilateral differences in both muscle activation and PCSA distribution is a contributing factor
454 or result from injury. For example, it is possible that a prolonged reduction in activation might
455 result in the atrophy of the injured muscle, even after a rehabilitation program. Subsequently,
456 the volume of the non-activated part of the muscle might decrease due to insufficient
457 mechanical stimuli, resulting in atrophy of the whole muscle. Alternatively, these differences
458 in activation and PCSA between the legs could have been present before the injury. However,
459 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 ($-10.8 \pm 20.7\%$) and the SM/Hams torque ratio higher ($+17.2 \pm 27.4\%$) in the injured than in the
473 non-injured limb. A large majority of participants adopted this strategy (13 out of 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,

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

491

492 **5. CONCLUSION**

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

502

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509

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513

514

515 **TABLES:**

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

Partici pant	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

520

521

522 *Table 2. Normalized EMG RMS measured in injured and uninjured limb during*
 523 *submaximal isometric knee flexions performed at 20% and 50% of the peak torque*
 524 *produced during maximal voluntary contraction (MVC). BF, biceps femoris; SM,*
 525 *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

526
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 528

529 **Table 3 – Muscle architecture.** Fascicle length, pennation angle, muscle volume,
 530 physiological cross-sectional area (PCSA) and moment arm for injured and uninjured limb.
 531 Statistics are only reported for muscle × limb interaction.
 532 ^a Indicates a significant difference with BF, ^b indicates a significant difference with SM and ^c
 533 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 (CM³)		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 (CM²)		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

534
 535

536 **FIGURES**

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

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

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

551 **Figure 4 – Torque ratios for the uninjured (black scatters) and injured (white scatters)**
552 **limb.** The ratios of torque were estimated during submaximal isometric knee flexions
553 performed at 20% and 50% of the peak torque produced during maximal voluntary
554 contraction (MVC).

555 * Indicates a significant difference between limbs ($P < 0.05$).

556

557 **REFERENCES**

- 558 1. **Adkins AN, Franks PW, Murray WM.** Demonstration of extended field-of-view
559 ultrasound's potential to increase the pool of muscles for which in vivo fascicle length is
560 measurable. *J Biomech* 63: 179–185, 2017.
- 561 2. **Avrillon S, Guilhem G, Barthelemy A, Hug F.** Coordination of hamstrings is
562 individual specific and is related to motor performance. *J Appl Physiol (1985)* 125:
563 1069–1079, 2018.
- 564 3. **Biewener AA.** Locomotion as an emergent property of muscle contractile dynamics. *J*
565 *Exp Biol* 219: 285–94, 2016.
- 566 4. **Blandford L, Theis N, Charvet I, Mahaffey R.** Is neuromuscular inhibition detectable
567 in elite footballers during the Nordic hamstring exercise? *Clin Biomech (Bristol, Avon)*
568 58: 39–43, 2018.
- 569 5. **Brochner Nielsen NP, Hug F, Guevel A, Fohanno V, Lardy J, Dorel S.** Motor
570 adaptations to unilateral quadriceps fatigue during a bilateral pedaling task. *Scan J Med*
571 *Sci Sports* 27: 1724–1738, 2017.
- 572 6. **Carroll TJ, Herbert RD, Munn J, Lee M, Gandevia SC.** Contralateral effects of
573 unilateral strength training: evidence and possible mechanisms. *J Appl Physiol (1985)*
574 101: 1514–22, 2006.
- 575 7. **Crossley K, Bennell K, Green S, Cowan S, McConnell J.** Physical therapy for
576 patellofemoral pain: a randomized, double-blinded, placebo-controlled trial. *Am J Sports*
577 *Med* 30: 857–65, 2002.
- 578 8. **Crouzier M, Lacourpaille L, Nordez A, Tucker K, Hug F.** Neuromechanical coupling
579 within the human triceps surae and its consequence on individual force-sharing
580 strategies. *J Exp Biol* 221, 2018.
- 581 9. **Daly C, McCarthy Persson U, Twycross-Lewis R, Woledge RC, Morrissey D.** The
582 biomechanics of running in athletes with previous hamstring injury: A case-control
583 study. *Scan J Med Sci Sports* 26: 413–20, 2016.
- 584 10. **van Dieen JH, Flor H, Hodges PW.** Low-Back Pain Patients Learn to Adapt Motor
585 Behavior With Adverse Secondary Consequences. *Exerc Sport Sci Rev* 45: 223–229,
586 2017.
- 587 11. **Dimmick S, Linklater JM.** Imaging of Acute Hamstring Muscle Strain Injuries. *Semin*
588 *Musculoskelet Radiol* 21: 415–432, 2017.
- 589 12. **Duchateau J, Enoka RM.** Neural control of lengthening contractions. *J Exp Biol* 219:
590 197–204, 2016.
- 591 13. **Fyfe JJ, Opar DA, Williams MD, Shield AJ.** The role of neuromuscular inhibition in
592 hamstring strain injury recurrence. *J Electromyogr Kinesiol* 23: 523–30, 2013.
- 593 14. **Garrett WE, Califf JC, Bassett FH.** Histochemical correlates of hamstring injuries. *Am*
594 *J Sports Med* 12: 98–103, 1984.

- 595 15. **Garrett WE, Safran MR, Seaber AV, Glisson RR, Ribbeck BM.** Biomechanical
596 comparison of stimulated and nonstimulated skeletal muscle pulled to failure. *Am J*
597 *Sports Med* 15: 448–54, 1987.
- 598 16. **Haberfehlner H, Maas H, Harlaar J, Becher JG, Buizer AI, Jaspers RT.** Freehand
599 three-dimensional ultrasound to assess semitendinosus muscle morphology. *J Anat* 229:
600 591–9, 2016.
- 601 17. **Handsfield GG, Knaus KR, Fiorentino NM, Meyer CH, Hart JM, Blemker SS.**
602 Adding muscle where you need it: non-uniform hypertrophy patterns in elite sprinters.
603 *Scan J Med Sci Sports* 27: 1050–1060, 2017.
- 604 18. **Hegy A, Csala D, Peter A, Finni T, Cronin NJ.** High-density electromyography
605 activity in various hamstring exercises. *Scan J Med Sci Sports* 29: 34–43, 2019.
- 606 19. **Herzog W, Guimaraes AC, Anton MG, Carter-Erdman KA.** Moment-length
607 relations of rectus femoris muscles of speed skaters/cyclists and runners. *Med Sci Sports*
608 *Exerc* 23: 1289–96, 1991.
- 609 20. **Hodges PW, Tucker K.** Moving differently in pain: a new theory to explain the
610 adaptation to pain. *Pain* 152: S90-8, 2011.
- 611 21. **Hug F, Goupille C, Baum D, Raiteri BJ, Hodges PW, Tucker K.** Nature of the
612 coupling between neural drive and force-generating capacity in the human quadriceps
613 muscle. *Proc Biol Sci* 282, 2015.
- 614 22. **Hug F, Tucker K.** Muscle Coordination and the Development of Musculoskeletal
615 Disorders. *Exerc Sport Sci Rev* 45: 201–208, 2017.
- 616 23. **Kilgallon M, Donnelly AE, Shafat A.** Progressive resistance training temporarily alters
617 hamstring torque-angle relationship. *Scan J Med Sci Sports* 17: 18–24, 2007.
- 618 24. **Kirk EA, Gilmore KJ, Rice CL.** Neuromuscular changes of the aged human
619 hamstrings. *J Neurophysiol* 120: 480–488, 2018.
- 620 25. **Loeb GE.** Optimal isn't good enough. *Biol Cybern* 106: 757–65, 2012.
- 621 26. **Lund JP, Donga R, Widmer CG, Stohler CS.** The pain-adaptation model: a discussion
622 of the relationship between chronic musculoskeletal pain and motor activity. *Can J*
623 *Physiol Pharmacol* 69: 683–94, 1991.
- 624 27. **Opar DA, Williams MD, Shield AJ.** Hamstring strain injuries: factors that lead to
625 injury and re-injury. *Sports Med* 42: 209–26, 2012.
- 626 28. **Opar DA, Williams MD, Timmins RG, Dear NM, Shield AJ.** Knee flexor strength
627 and bicep femoris electromyographical activity is lower in previously strained
628 hamstrings. *J Electromyogr Kinesiol* 23: 696–703, 2013.
- 629 29. **Orchard JW, Seward H, Orchard JJ.** Results of 2 decades of injury surveillance and
630 public release of data in the Australian Football League. *Am J Sports Med* 41: 734–41,
631 2013.

- 632 30. **Pimenta R, Blazevich AJ, Freitas SR.** Biceps Femoris Long-Head Architecture
633 Assessed Using Different Sonographic Techniques. *Med Sci Sports Exerc* 50: 2584–
634 2594, 2018.
- 635 31. **de Rugy A, Loeb GE, Carroll TJ.** Muscle coordination is habitual rather than optimal.
636 *J Neurosci* 32: 7384–91, 2012.
- 637 32. **Sacks RD, Roy RR.** Architecture of the hind limb muscles of cats: functional
638 significance. *J Morphol* 173: 185–95, 1982.
- 639 33. **Sanfilippo JL, Silder A, Sherry MA, Tuite MJ, Heiderscheit BC.** Hamstring strength
640 and morphology progression after return to sport from injury. *Med Sci Sports Exerc* 45:
641 448–54, 2013.
- 642 34. **Schache AG, Kim HJ, Morgan DL, Pandy MG.** Hamstring muscle forces prior to and
643 immediately following an acute sprinting-related muscle strain injury. *Gait & Posture*
644 32: 136–40, 2010.
- 645 35. **Schuermans J, Van Tiggelen D, Danneels L, Witvrouw E.** Biceps femoris and
646 semitendinosus--teammates or competitors? New insights into hamstring injury
647 mechanisms in male football players: a muscle functional MRI study. *Br J Sports Med*
648 48: 1599–606, 2014.
- 649 36. **Schuermans J, Van Tiggelen D, Danneels L, Witvrouw E.** Susceptibility to
650 Hamstring Injuries in Soccer: A Prospective Study Using Muscle Functional Magnetic
651 Resonance Imaging. *Am J Sports Med* 44: 1276–85, 2016.
- 652 37. **Silder A, Heiderscheit BC, Thelen DG, Enright T, Tuite MJ.** MR observations of
653 long-term musculotendon remodeling following a hamstring strain injury. *Skeletal*
654 *Radiol* 37: 1101–9, 2008.
- 655 38. **Sole G, Milosavljevic S, Nicholson HD, Sullivan SJ.** Selective strength loss and
656 decreased muscle activity in hamstring injury. *J Orthop Sports Phys Ther* 41: 354–63,
657 2011.
- 658 39. **Stepien K, Smigielski R, Mouton C, Cizek B, Engelhardt M, Seil R.** Anatomy of
659 proximal attachment, course, and innervation of hamstring muscles: a pictorial essay.
660 *Knee Surg Sports Traumatol Arthrosc* 27: 673–684, 2019.
- 661 40. **Todorov E, Jordan MI.** Optimal feedback control as a theory of motor coordination.
662 *Nature Neurosci* 5: 1226–35, 2002.
- 663 41. **Woodley SJ, Mercer SR.** Hamstring muscles: architecture and innervation. *Cells,*
664 *Tissues, Organs* 179: 125–41, 2005.
- 665







