

1 **TITLE**

2 Rate of torque and EMG development during anticipated eccentric contraction is lower in previously  
3 strained hamstrings.

4 **Authors**

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15 **ABSTRACT**

16 **Background:** Hamstring strain injuries are prevalent in sport and re-injury rates have been high for  
17 many years. Whilst much focus has centred on the impact of previous hamstring strain injury on  
18 maximal eccentric strength, high rates of torque development is also of interest, given the important  
19 role of the hamstrings during the terminal swing phase of running. The impact of prior strain injury  
20 on myoelectrical activity of the hamstrings during tasks requiring high rates of torque development  
21 has received little attention. **Purpose:** To determine if recreational athletes with a history of  
22 unilateral hamstring strain injury, who have returned to training and competition, will exhibit lower  
23 levels of myoelectrical activity during eccentric contraction, rate of torque development and impulse  
24 30, 50 and 100ms after the onset of myoelectrical activity or torque development in the previously  
25 injured limb compared to the uninjured limb. **Study design:** Case-control study **Methods:** Twenty-six  
26 recreational athletes were recruited. Of these, 13 athletes had a history of unilateral hamstring  
27 strain injury (all confined to biceps femoris long head) and 13 had no history of hamstring strain  
28 injury. Following familiarisation, all athletes undertook isokinetic dynamometry testing and surface  
29 electromyography assessment of the biceps femoris long head and medial hamstrings during  
30 eccentric contractions at  $-60^{\circ}$  and  $-180^{\circ}.s^{-1}$ . **Results:** In the injured limb of the injured group,  
31 compared to the contralateral uninjured limb rate of torque development and impulse was lower  
32 during  $-60^{\circ}.s^{-1}$  eccentric contractions at 50 (RTD, injured limb =  $312.27 \pm 191.78Nm.s^{-1}$  vs. uninjured  
33 limb =  $518.54 \pm 172.81Nm.s^{-1}$ ,  $p=0.008$ ; IMP, injured limb =  $0.73 \pm 0.30 Nm.s$  vs. uninjured limb =  
34  $0.97 \pm 0.23 Nm.s$ ,  $p=0.005$ ) and 100ms (RTD, injured limb =  $280.03 \pm 131.42Nm.s^{-1}$  vs. uninjured limb  
35 =  $460.54.54 \pm 152.94Nm.s^{-1}$ ,  $p=0.001$ ; IMP, injured limb =  $2.15 \pm 0.89 Nm.s$  vs. uninjured limb =  $3.07 \pm$   
36  $0.63 Nm.s$ ,  $p<0.001$ ) after the onset of contraction. Biceps femoris long head muscle activation was  
37 lower at 100ms at both contraction speeds ( $-60^{\circ}.s^{-1}$ , normalised iEMG activity (x1000), injured limb =  
38  $26.25 \pm 10.11$  vs. uninjured limb  $33.57 \pm 8.29$ ,  $p=0.009$ ;  $-180^{\circ}.s^{-1}$ , normalised iEMG activity (x1000),  
39 injured limb =  $31.16 \pm 10.01$  vs. uninjured limb  $39.64 \pm 8.36$ ,  $p=0.009$ ). Medial hamstring activation  
40 did not differ between limbs in the injured group. Comparisons in the uninjured group showed no

41 significant between limbs difference for any variables. **Conclusion:** Previously injured hamstrings  
42 displayed lower rate of torque development and impulse during slow maximal eccentric contraction  
43 compared to the contralateral uninjured limb. Lower myoelectrical activity was confined to the  
44 biceps femoris long head. Regardless of whether these deficits are the cause of or the result of  
45 injury, these findings could have important implications for hamstring strain injury and re-injury.  
46 Particularly, given the importance of high levels of muscle activity to bring about specific muscular  
47 adaptations, lower levels of myoelectrical activity may limit the adaptive response to rehabilitation  
48 interventions and suggest greater attention be given to neural function of the knee flexors following  
49 hamstring strain injury.

50 **Key terms:** strain injury, neuromuscular function, surface electromyography.

51 **What is known about the subject?:** Previous hamstring strain injury results in a greater decline in  
52 eccentric knee flexor strength compared to concentric strength in athletes who have been  
53 rehabilitated sufficiently to return to training and competitive match play. It has also been reported  
54 that this eccentric specific weakness following injury is associated with a reduction in voluntary  
55 activation. However as the primary injurious activity type for hamstring strain injury is during the  
56 terminal swing phase of high speed running, the ability of the hamstrings to development eccentric  
57 torque rapidly is of interest. Whether previous hamstring strain injury impacts upon myoelectrical  
58 activity of rapid eccentric contraction remains to be seen.

59 **What this study adds to the existing knowledge:** To our knowledge this is the first study to report  
60 lower rate of torque development and impulse in previously injured hamstrings up to and including  
61 the first 100ms of an anticipated eccentric contraction. With respect to the neural factors associated  
62 with this torque development, myoelectrical activity of biceps femoris long head during slow  
63 maximal eccentric muscle contraction was lower 100ms after the onset of myoelectrical activity in  
64 the previously injured leg. As all hamstring strain injuries examined in this study were confined to

65 the biceps femoris long head, the decline in myoelectrical activity suggests a potentially muscle  
66 specific response to injury.

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## 84 INTRODUCTION

85 Muscle strain injuries are problematic for elite, sub-elite and recreational level athletes participating  
86 in running based sports.<sup>7, 13, 35, 48</sup> Of all muscle strain injuries in sport, hamstring strain injuries (HSIs)  
87 are the most prevalent.<sup>7, 13, 35, 48</sup> HSIs result in considerable lost time from training and absence from  
88 competition, decrements in athlete performance and, in team sports settings, a financial burden for  
89 the club or organisation.<sup>34</sup> One of the most prominent consequences of HSIs that is yet to be  
90 resolved is the high rates of reinjury, an issue of great importance considering previous HSI is  
91 consistently identified as the primary risk factor for future injury.<sup>3</sup> Whilst the existence of this injury-  
92 reinjury cycle is acknowledged,<sup>9</sup> success in reducing reinjury rates in one sport has been largely  
93 attributed to increased rehabilitation time,<sup>35</sup> more so than due to a greater understanding of the  
94 maladaptations associated with previous injury or improved rehabilitation practices.

95 Scant attention has been given to the potential for unattended neural maladaptations associated  
96 with a previous insult to increase the likelihood of future HSI. Recent work has reported lower levels  
97 of myoelectrical activity in the previously injured hamstring during maximal voluntary eccentric  
98 contractions tested at the movement speed of  $-60^0.s^{-1}$ .<sup>40</sup> That study was the first to provide empirical  
99 evidence that lower myoelectrical activity in a previously injured hamstring during maximal eccentric  
100 contractions exists. However many other aspects of neural function are yet to be examined.

101 Myoelectrical activity during rapid force generation is one such avenue of further investigation. Such  
102 work is warranted given one of the primary roles of the hamstring muscle group is rapid  
103 deceleration of the advancing thigh during the terminal swing phase of high speed running.<sup>41</sup>

104 Optimal hamstring function during this portion of the running cycle is important as terminal swing is  
105 considered by some to be most injurious phase of gait as it combines moderate muscle strains and  
106 high force eccentric contraction.<sup>23, 37</sup> As such, high rates of torque development (RTD)

107 ( $\Delta\text{torque}/\Delta\text{time}$ ) and early contractile impulse (IMP) (the area under the time vs. torque curve)

108 during eccentric contractions are important characteristics of hamstring function because the

109 limited time available for deceleration ( $\sim 100\text{ms}^{30}$ ) prevents the development of maximal torque.<sup>43</sup>  
110 Undoubtedly musculotendinous properties, such as muscle size, relative area of fast-twitch fibers,  
111 myosin heavy chain isoform composition and tendon stiffness partly impact on RTD,<sup>5, 20, 22</sup> however,  
112 the magnitude of myoelectrical activity also contributes. Specifically, the amount of myoelectrical  
113 activity during the early phase of the contraction has a positive relationship with RTD.<sup>1-2</sup> Whether  
114 the initial magnitude of myoelectrical activity is less in a previously injured hamstring and whether  
115 this result in lower initial eccentric RTD and IMP is, however, yet to be examined.

116 Measures of RTD, IMP and concurrent myoelectrical activity have been obtained largely during  
117 isometric contractions. The information obtained may be limited given the importance of eccentric  
118 strength in the aetiology of HSIs. Therefore assessment of these variables during eccentric  
119 contraction may be considered better suited. Yet, the potential to do so is somewhat limited mainly  
120 due to the lag between the onset of torque development and the movement of the isokinetic  
121 dynamometer lever arm, which we have observed in our lab to be in excess of 100ms. To some  
122 extent this issue can be overcome through the use of an anticipated eccentric contraction whereby  
123 the participant performs an isokinetic eccentric action, however given the short time frame over  
124 which RTD, IMP and myoelectrical activity is analysed the actual contraction is quasi-isometric.  
125 Nevertheless, the intention to perform an eccentric action has been shown to result in greater  
126 movement related cortical potential compared to concentric actions.<sup>14</sup> This suggests that the  
127 execution of motor activity is modulated according to the contraction type to be performed.<sup>14</sup>  
128 Indeed contraction mode specific neural control has been evidenced previously via surface  
129 electromyography (sEMG) with these anticipated eccentric contractions<sup>17</sup> suggesting that  
130 contraction mode specific information about myoelectrical activity can be determined with such an  
131 experimental design. Therefore the purpose of the current study was to examine if a previously  
132 injured hamstring displayed lower RTD, IMP and concurrent early myoelectrical activity from the  
133 biceps femoris long head (BF) and medial hamstrings (MH) during anticipated slow and fast eccentric  
134 actions in comparison to the contralateral uninjured hamstring. Myoelectrical activity was recorded

135 from both BF and MH to determine if alterations in myoelectrical activity were confined to the  
136 previously injured hamstring muscle. A control group was also examined to demonstrate that limb  
137 dominance did not influence RTD, IMP or hamstring myoelectrical activity.

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## 155 MATERIALS AND METHODS

### 156 Participants

157 Recreational level male athletes (n=26) were recruited to participate in the study. All participated in  
158 running based sports such as Australian football, soccer, sprinting and touch rugby. Of these, 13  
159 athletes ( $26.6 \pm 5.8$  years;  $1.8 \pm 0.04$ m;  $83.2 \pm 14.3$ kg) had sustained at least one grade II HSI within  
160 the last 36 months and another 13 athletes ( $25.9 \pm 3.4$  years;  $1.8 \pm 0.05$ m;  $82.8 \pm 7.5$  kg) had no  
161 history of HSIs. All participants were free of any other lower limb injury, were fully recovered from  
162 their previous HSIs and active in their chosen sport at the time of testing. For all athletes limb  
163 dominance was defined as the preferred kicking leg. All testing procedures were approved by the  
164 Queensland University of Technology Human Research Ethics Committee. Participants gave  
165 informed written consent prior to testing after having all procedures explained to them.

### 166 Injury questionnaire

167 Following recruitment, participants completed an injury questionnaire with their chosen practitioner  
168 (i.e. physiotherapist) who had previously diagnosed and treated all the athletes hamstring strain  
169 injuries. As per previous investigations<sup>40</sup> the notes taken from clinical examination were used to  
170 detail the: date of injury and return to pre-injured levels of training and competition; severity (grade  
171 I, II or III)<sup>4</sup>; location with respect to limb dominance and specific hamstring muscle (BF or MH)  
172 injured; and rehabilitation details of all previous HSIs. Athletes were considered to be successfully  
173 rehabilitated when they returned to pre-injury levels of training and were available for match  
174 selection or competition.<sup>15</sup>

### 175 EMG recording

176 Myoelectrical activity was measured via sEMG from the MH and BF through the use of circular  
177 bipolar pre-gelled Ag/AgCl sEMG electrodes (10mm diameter, 25mm inter-electrode distance). After  
178 preparation of the skin via shaving, abrasion and sterilisation, electrodes were placed on the



179 posterior thigh half way between the ischial tuberosity and tibial epicondyles, as per SENAIM  
180 guidelines.<sup>26</sup> Muscle bellies were identified via palpation during forceful isometric knee flexion and  
181 correct placement was confirmed by observing sEMG activity during active internal and external  
182 rotation of the flexed knee.

### 183 **Isokinetic dynamometry**

184 Assessment of knee flexor RTD was performed on a Biodex Systems 3 Dynamometer (Biodex Medical  
185 Systems, Shirley, NY). Participants were seated on a custom pad, placed on top of the original seat,  
186 which contained two holes at the level of the posterior mid thigh to minimise movement artefact  
187 from sEMG electrodes on the dynamometer seat. The hips were flexed at 85° from neutral with the  
188 lateral epicondyle of the femur carefully aligned with the fulcrum of the dynamometer. The tested  
189 leg was attached to the lever of the dynamometer via a Velcro strap and padded restraints were  
190 fastened across the trunk, hips and mid thigh of the tested leg to isolate movement to the knee  
191 joint. The range of motion was set at 5°-90° of knee flexion (0°=full knee extension; knee joint angle  
192 at start position=90°) and correction for limb weight was performed throughout the range of  
193 motion.

194 Three sets of four submaximal concentric contractions of the knee extensors and flexors were  
195 performed at +240<sup>0</sup>.s<sup>-1</sup> as a warm-up to prepare the participant for maximal effort in the following  
196 sets. Eccentric testing for both legs consisted of three sets of three consecutive eccentric maximum  
197 voluntary contractions (MVC) of the knee flexors at speeds of -60<sup>0</sup>.s<sup>-1</sup> and -180<sup>0</sup>.s<sup>-1</sup> with 30 seconds  
198 rest between sets. The leg and speed testing orders were randomised and athletes were informed of  
199 the testing speed prior to each set. Athletes were instructed to remain relaxed prior to contraction  
200 to allow a stable baseline measurement of torque and sEMG to be obtained. Athletes were  
201 instructed to push their heel back as quickly as they could towards their gluteus when given the  
202 signal to contract and were encouraged verbally by the investigators to ensure maximal effort. The

203 signal to contract was delivered verbally by the investigators. All athletes were required to attend at  
204 least one familiarisation session and one testing session with  $\geq$  seven days between each session.

## 205 **Data analysis**

206 For each movement speed the three contractions with the highest peak torque were used for  
207 further analysis. Dynamometer torque and lever position data were transferred to a personal  
208 computer at 1 kHz and stored for later analysis. RTD was determined as the mean of the average  
209 slope of the torque-time trace ( $\Delta\text{torque}/\Delta\text{time}$ ) for the three selected repetitions from the onset of  
210 contraction through until 30, 50 and 100ms of the contraction. Onset of contraction was defined as  
211 when torque deviated 4Nm from the baseline level of torque at rest (Figure 1).<sup>42</sup> IMP was calculated  
212 as the area under the torque-time trace across the same time periods.

213 Surface EMG data was sampled simultaneously with dynamometer data at 1kHz through a 16-bit  
214 PowerLab26T AD recording unit with in-built anti-aliasing filter (ADInstruments, New South Wales,  
215 Australia) (amplification = 1000; common mode rejection ratio = 110 dB; Input impedance = 100 M $\Omega$ ;  
216 fixed gain) and stored for later analysis where it was fourth order Butterworth filtered between 20-  
217 500Hz (24dB roll off) using MATLAB (MathWorks, Natick, Massachusetts) and then full wave rectified  
218 using the root-mean-square method. For each contraction, sEMG data for MH and BF was  
219 normalised to the maximum magnitude of the rectified sEMG signal for that contraction, for each  
220 muscle respectively. Myoelectrical activity was defined as the area under the rectified sEMG-time  
221 trace, commonly referred to as integrated EMG (iEMG), and was measured across 30, 50 and 100ms  
222 after the onset of myoelectrical activity. All myoelectrical data is expressed as normalised iEMG  
223 multiplied by a factor of 1000. Onset of myoelectrical activity was determined by smoothing the  
224 rectified EMG signal (100 point moving average) and then identifying when the smoothed rectified  
225 signal rose above 10% of the maximum signal for the final time.<sup>36</sup> The identification of onset was  
226 then confirmed by visual examination of the raw and rectified (unsmoothed) sEMG signal at the

227 same time point. All analysis was performed using LabCart 7.3 (ADInstruments, New South Wales,  
228 Australia).

### 229 **Statistical analysis**

230 Data was analysed using JMP version 9.0 Pro Statistical Discovery Software (SAS Inc). Aligned with  
231 the study's primary objectives, comparisons were made for each dependent variable (RTD, IMP and  
232 BF and MH myoelectrical activity) between the injured and uninjured limbs in the injured group.  
233 Comparisons between dominant and non-dominant limbs in the uninjured group were also made to  
234 determine any influence of limb dominance. The use of ANOVA models was deemed not valid since  
235 analysis of means for variances (ANOMV) used to test homogeneity of variance of dependent  
236 variables across groups<sup>46, 47</sup> indicated that this assumption was not satisfied ( $p < 0.05$ ). As such,  
237 dependent variables were compared using two tailed paired t tests for both groups. Bonferroni  
238 corrections were performed to account for three comparisons made for each dependent variable  
239 across the velocities used, with significance adjusted to  $p < 0.0167$ . To assess the magnitudes of the  
240 differences Cohen's d was also used to report effect size (ES).

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## 249 RESULTS

### 250 Participants

251 The two groups were similar with respect to age, height and body mass (Injured group,  $26.6 \pm 5.8$   
252 years;  $1.8 \pm 0.04\text{m}$ ;  $83.2 \pm 14.3\text{kg}$ ; Uninjured group,  $25.9 \pm 3.4$  years;  $1.8 \pm 0.05\text{m}$ ;  $82.8 \pm 7.5$  kg). All  
253 athletes from the injured group had suffered at least one grade II HSI in the last 36 months. The total  
254 number of HSIs sustained by each athlete in the injured group ranged between one and four  
255 (median = 2) in the same 36 month period. All injuries were confined to the BF. Median time since  
256 most recent HSI was 3.9 months (range = 1.0 – 18.2), with median time taken to return to pre-  
257 injured levels of competition being 4 weeks (range = 2 - 6). All athletes from the injured group  
258 reported standard rehabilitation progression (i.e. Ref 24) guided by their physiotherapist, with all but  
259 one of the injured athletes reporting some eccentric conditioning as part of their late phase  
260 rehabilitation program.

### 261 RTD and IMP

262 RTD and IMP was significantly lower in the previously injured knee flexor for  $-60^{\circ} \cdot \text{s}^{-1}$  anticipated  
263 eccentric contractions at 50 (RTD, injured limb =  $312.27 \pm 191.78\text{Nm} \cdot \text{s}^{-1}$  vs. uninjured limb =  $518.54 \pm$   
264  $172.81\text{Nm} \cdot \text{s}^{-1}$ ,  $p=0.008$ ,  $ES=1.12$ ; IMP, injured limb =  $0.73 \pm 0.30$  Nm.s vs. uninjured limb =  $0.97 \pm$   
265  $0.23$  Nm.s,  $p=0.005$ ,  $ES=0.87$ ) and 100ms (RTD, injured limb =  $280.03 \pm 131.42\text{Nm} \cdot \text{s}^{-1}$  vs. uninjured  
266 limb =  $460.54 \pm 152.94\text{Nm} \cdot \text{s}^{-1}$ ,  $p=0.001$ ,  $ES=1.27$ ; IMP, injured limb =  $2.15 \pm 0.89$  Nm.s vs.  
267 uninjured limb =  $3.07 \pm 0.63$  Nm.s,  $p<0.001$ ,  $ES=1.20$ ) after the onset of contraction (Figure 2,  
268 Supplementary Table 1). There was no significant difference for RTD or IMP during anticipated  
269 eccentric contractions at  $-180^{\circ} \cdot \text{s}^{-1}$  at any time point (Figure 2, Supplementary Table 1). There were  
270 no between limb differences for either variable in the uninjured group (Figure 3, Supplementary  
271 Table 1).

### 272 Integrated EMG

273 With respect to myoelectrical activity of BF, normalised iEMG was lower at 100ms at both  
274 contraction speeds between limbs in the injured group ( $-60^0.s^{-1}$ , injured limb =  $26.25 \pm 10.11$  vs.  
275 uninjured limb  $33.57 \pm 8.29$ ,  $p=0.009$ ,  $ES=0.80$ ;  $-180^0.s^{-1}$ , injured limb =  $31.16 \pm 10.01$  vs. uninjured  
276 limb  $39.64 \pm 8.36$ ,  $p=0.009$ ,  $ES=0.92$ ) (Figure 4, Supplementary Table 2), but there were no significant  
277 differences between limbs in the uninjured group (Figure 5). No differences existed with respect to  
278 MH iEMG in either group (Figure 4 & 5, Supplementary Table 2).

## 279 **DISCUSSION**

280 The hamstring muscle group is the most commonly strained muscle in running based sports.<sup>7, 13, 35, 48</sup>  
281 This is purportedly due to the demands of high speed running and specifically the need for rapid  
282 deceleration of the flexing hip and extending knee during terminal swing.<sup>23, 37</sup> As such the ability of  
283 the biarticular hamstrings to generate eccentric force rapidly is a key feature of hamstring function.  
284 The current study examined whether athletes with a prior unilateral HSI history displayed lower  
285 levels of RTD, IMP and myoelectrical activity in the previously injured hamstring compared to the  
286 contralateral uninjured hamstring for brief periods following the onset of anticipated eccentric  
287 contractions. The novel findings from this study are that recreational athletes with a history of HSIs  
288 confined to the BF exhibited i) lesser RTD and IMP 50 and 100 ms after the onset of an anticipated  
289 eccentric contractions at  $-60^0.s^{-1}$ ; ii) lesser BF myoelectrical activity at 100 ms after the onset of  
290 myoelectrical activity in anticipation of eccentric contractions at  $-60^0.s^{-1}$  and  $-180^0.s^{-1}$  in the  
291 previously injured limb compared to the uninjured limb. Of further importance was that  
292 myoelectrical activity of the MH was not different between limbs in the injured group. There were  
293 also no differences found between dominant and non-dominant limb for torque or myoelectrical  
294 activity in the control group, indicating no influence of limb dominance.

295 This is, to our knowledge, the first study to examine RTD, IMP and concurrent myoelectrical activity  
296 in previously injured hamstrings, which makes comparisons to previous work difficult. One previous  
297 study has examined the impact of a simulated handball game on isometric knee flexor function and

298 this study reported higher baseline RFD relative to bodyweight (6.92 – 9.27Nm/s/kg) compared to  
299 the uninjured limbs ( 4.82 – 5.41Nm/s/kg) in the current study.<sup>42</sup> The divergent RFD findings may be  
300 explained by the methodological differences such as athlete expertise (recreational active vs elite  
301 handball players), different knee joint angles used to assess RFD (90° vs 70° of knee flexion) and the  
302 use of anticipated eccentric contraction as opposed to isometric rate of force development in  
303 previous work .

304 The finding that a previous strain injury to BF results in a lesser ability to generate torque quickly in  
305 anticipation of an eccentric contraction may have important consequences for recurrent HSI risk and  
306 current rehabilitation practices. This is because the time frame in which the knee flexors have to  
307 decelerate the flexing hip and extending knee joints during terminal swing is limited (~100ms<sup>30</sup>). As  
308 such the rapid development of eccentric torque is paramount to minimise the risk of  
309 overlengthening of the hamstrings. If, as was observed in the current cohort, previously injured  
310 limbs display lower knee flexor RTD and IMP and lower BF myoelectrical activity up to 100ms  
311 following the onset of contraction it might be expected to increase the work required of the  
312 hamstrings at terminal swing to slow the forward moving shank due to poor deceleration during  
313 early swing. Furthermore, a lesser ability to produce a decelerating force for a brief period following  
314 the onset of contraction would likely increase the work required of the hamstrings at longer muscle  
315 lengths and the impact of this may be two fold. Firstly, the increase in work may induce the onset of  
316 fatigue earlier in the BF, which is the primary knee flexor at long muscle lengths.<sup>33</sup> Given fatigue  
317 reduces the amount of energy that can be absorbed by a lengthening muscle<sup>29</sup> this may increase the  
318 potential for strain induced muscle failure. Secondly, unpublished observations from our lab suggest  
319 that athletes with a previous HSI to BF display lower BF myoelectrical activity during eccentric  
320 contractions at long lengths. If there are extra demands placed on the BF at terminal swing due to  
321 poor RTD and IMP, but due to restricted myoelectrical activity at this muscle length the muscle  
322 cannot meet these demands then this has the potential to increase the likelihood for hamstring  
323 overlengthening. Such overlengthening can be problematic as it may increase the risk of the

324 hamstrings exceeding their mechanical limits<sup>34</sup> or accumulating microscopic muscle damage<sup>6</sup> and  
325 this increases the potential for injury/reinjury.

326 The observations that RTD and IMP were lower in anticipation of a slow, but not fast, eccentric  
327 contraction is intriguing given that the myoelectrical activity of the previously injured BF was lower  
328 in anticipation of both speeds of eccentric contraction. Whilst RTD was not lower in the previously  
329 injured limb compared to the contralateral uninjured limb at any time point at  $-180^{\circ}.s^{-1}$  there was a  
330 medium effect size at 100ms following the onset of contraction ( $p=0.064$ , Cohen's  $d$   $ES=0.57$ ) and a  
331 larger sample may have revealed a significant difference. However this finding might also be  
332 indicative of alterations in coordination of the knee flexor muscles in anticipation of a fast eccentric  
333 contraction. Altered coordination may be driven by the intent to protect the previously injured BF in  
334 anticipation of a high speed eccentric action. In the case of this study other knee flexors, not  
335 examined, might be recruited more heavily thus increasing their contribution to knee flexion torque  
336 generation, with the most suited candidate being the uniarticular biceps femoris short head. Indirect  
337 evidence supports this change in contribution to knee flexion torque, given that a previously injured  
338 leg displays compensatory hypertrophy of this muscle,<sup>39</sup> which would be suggestive of an increased  
339 volume of work during habitual activities. Moreover, BF atrophy has been found,<sup>39</sup> as a possible  
340 consequence of reduced activation and disuse following HSI. Whether such a reorganisation of  
341 muscle activity exists is, however, yet to be explored and should be an area for future examination.

342 If significant neuromuscular inhibition of BF exists its benefits are most likely to be confined to the  
343 early phase of recovery and rehabilitation. A novel framework proposed previously hypothesises  
344 that pain associated with HSI results in prolonged neural deficits which compromise the  
345 rehabilitation process.<sup>34</sup> This framework focuses largely on chronic reductions in voluntary activation  
346 of the previously injured hamstrings during eccentric contractions and the impact of such a  
347 neurological deficit on muscular adaptations (for a thorough discussion of this see Ref 34). However,  
348 reductions in early neural drive of the previously injured BF in response to strain injury may present

349 another problematic maladaptation associated with previous HSI. Acute restriction of early neural  
350 drive following injury presumably constitutes a strategy to unload the damaged tissue and reduce  
351 pain in the acute recovery period.<sup>34</sup> However chronic reductions in early neural drive would be  
352 expected to compromise the rehabilitation process, given the need for high levels of activation to  
353 bring about muscular adaptations.<sup>34</sup> The reduction in early myoelectrical activity of BF, combined  
354 with the restriction of myoelectrical activity of BF during maximal eccentric contraction (unpublished  
355 observations from our lab), might be expected to reduce the stimulus the previously injured BF is  
356 exposed to, resulting in limited muscle hypertrophy and sarcomerogenesis. Decrements in these two  
357 factors would be expected to reduce strength and reduce the optimum length of the hamstring  
358 muscle group, respectively, and both have been implicated in HSI aetiology.<sup>6,10</sup> Whilst much work  
359 has been done on the contractile and structural<sup>16,27</sup> implications of strain injury, neural  
360 maladaptation and associated changes have been largely neglected and should be the focus of  
361 future investigations.

362 If lower BF myoelectrical activity is in response to HSI, the underpinning mechanism responsible is  
363 of interest. At present most studies have examined the impact of resistance training on neural  
364 factors that influence RTD. These studies all have focused on mechanisms to explain improved RTD  
365 including: increased neural drive; increased motor unit discharge rates; increased motor unit  
366 synchronisation; and earlier recruitment of motor units.<sup>11,21,38,44</sup> Whether all of these adaptations  
367 occur 'in reverse' following HSI remains to be seen, however the current study found that lower  
368 myoelectrical activity occurred in the previously injured BF. Yet, as the stimulus for neural  
369 maladaptation to HSI is hypothesised to be due to pain<sup>34</sup> (as opposed to heavily load or explosive  
370 resistance exercise<sup>19</sup>) the altered function of the nervous system may differ markedly. HSIs induce  
371 acute<sup>45</sup> and chronic<sup>9,28</sup> pain particularly in athletes with recurrent strain injuries. Acute muscle pain  
372 is known to result in short term neural responses resulting in reduced strength, agonistic activation  
373 and muscle endurance, increased antagonistic activity and altered coordination patterns during  
374 static and dynamic motor tasks.<sup>12,18-19,25</sup> This muscular pain also has the potential to alter central



375 nervous function at both the spinal and supraspinal level, resulting in increased pain sensitivity and  
376 an expanded neuron population of the painful muscle in the dorsal horn of the spinal cord.<sup>31</sup> Pain  
377 has the potential to modulate descending neural pathways<sup>32</sup> and by extension the ability to fully  
378 activate the motor neuron pool. This maladaptation of neural function might therefore be expected  
379 to result in a restriction of myoelectrical activity during the onset of contraction and may be  
380 specifically confined to the muscle responsible for the noxious stimulus.

381 There are some limitations associated with the current work. Firstly, as discussed earlier, the  
382 statistical power of the current study was too low to detect small to moderate effect sizes (Cohen's  $d$   
383 = 0.2-0.8). A larger sample size might have revealed significant differences between dependent  
384 variables that were not identified in the current study. As such a larger sample, also considering the  
385 inclusion of female athletes, should be a consideration for future investigations; notwithstanding the  
386 difficulty in recruiting athletes for the INJ group. The retrospective nature of these findings do not  
387 allow for the determination of whether lower levels of RTD, IMP and concurrent early myoelectrical  
388 activity of BF are the cause of, or the result of HSI. Potentially the lesser myoelectrical activity in the  
389 previously injured BF could indicate incomplete rehabilitation, whereby the deficits could be  
390 ameliorated with further intervention; a permanent lessening of myoelectrical activity in response to  
391 injury; or a deficit that was present prior to injury. Regardless of the responsible mechanisms, all  
392 athletes were deemed sufficiently rehabilitated to return to play, however the deficits in RTD, IMP  
393 and myoelectrical activity might suggest that rehabilitation was in fact incomplete. Future work  
394 should investigate whether lower myoelectrical activity, particularly of BF, is a risk factor for future  
395 HSI and explore what interventions are successful at restoring myoelectrical activity following HSI.  
396 Furthermore, we were unable to control the rehabilitation programmes of the current cohort,  
397 however all reported largely conventional rehabilitation progression guided by a physiotherapist.  
398 We were also limited because current methodologies do not allow for the performance of eccentric  
399 isokinetic knee flexion in such brief time periods as examined in the current study. As such the  
400 muscle action performed during the assessed time periods was quasi-isometric. Regardless the

401 intention to perform an eccentric muscle action results in different cortical<sup>14</sup> and sEMG<sup>17</sup> activity  
402 compared to concentric contractions even when performing quasi-isometric contraction.<sup>17</sup> This  
403 suggests that information about contraction mode specific myoelectrical activity can be derived  
404 from quasi-isometric contractions with the intent of performing an eccentric action. Finally, the use  
405 of isokinetic dynamometry at speeds of -60 and -180<sup>0</sup>.s<sup>-1</sup> to assess eccentric neuromuscular function  
406 is not wholly reflective of the demands placed on the hamstrings during injurious activities such as  
407 running and kicking, where greater angular velocities are experienced. The impact of previous HSI on  
408 neuromuscular function during these tasks should be examined further. Nevertheless, isokinetic  
409 testing combined with sEMG allows for the determination of RTD, IMP and myoelectrical activity  
410 whilst controlling for different movement velocities, a variable which was found to influence RTD  
411 and IMP in the current study.

412 In conclusion, we have shown for the first time, to our knowledge, that a previously strained  
413 hamstring, which has been rehabilitated sufficiently to return to training and competition, displays  
414 lower levels of RTD and IMP in anticipation of a slow maximal eccentric contraction compared to the  
415 contralateral uninjured limb. Furthermore, lower early myoelectrical activity was observed in the  
416 injured BF compared to the contralateral uninjured BF in anticipation of fast and slow maximal  
417 eccentric contraction. Regardless of whether these deficits are a response to or the result of muscle  
418 strain injury they could have important implications for current preventative and rehabilitation  
419 practices. Particularly, given the importance of high levels of muscle activity to bring about specific  
420 muscular adaptations, lower levels of myoelectrical activity may limit the adaptive response to  
421 rehabilitation interventions. This would be expected to limit the effectiveness of rehabilitation  
422 exercises and suggests that consideration be given to deficits in myoelectrical activity following HSI.  
423 A greater appreciation for impaired neural function following HSI might be expected to improve  
424 rehabilitation outcomes.

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542 **FIGURE CAPTIONS**

543 **Figure 1:** Representative torque-time trace. Prior to the onset of contraction baseline levels of  
544 torque were determined. Onset of contraction was defined as when knee flexor torque deviated by  
545 4.0Nm from baseline. Rate of torque development was determined as the average change in torque  
546 over time ( $\Delta\text{torque}/\Delta\text{time}$ ) at 30, 50, 100ms from onset of contraction development.

547 **Figure 2:** Comparisons between the uninjured and injured limbs of previously injured athletes of  
548 knee flexor rate of torque development (A.  $-60^\circ.s^{-1}$  and B.  $-180^\circ.s^{-1}$ ) and impulse (C.  $-60^\circ.s^{-1}$  and D.  $-180^\circ.s^{-1}$ ) at 30, 50 and 100ms from the onset of torque development. Error bars indicate standard  
549 deviation. \* $p < 0.0167$  uninjured vs injured limbs.

551 **Figure 3:** Comparisons between the dominant and non-dominant limbs of uninjured athletes of knee  
552 flexor rate of torque development (A.  $-60^\circ.s^{-1}$  and B.  $-180^\circ.s^{-1}$ ) and impulse (C.  $-60^\circ.s^{-1}$  and D.  $-180^\circ.s^{-1}$ ) at 30, 50 and 100ms from the onset of torque development. Error bars indicate standard  
553 deviation.

555 **Figure 4:** Comparisons between the uninjured and injured limbs of previously injured athletes of  
556 integrated electromyography (iEMG) from the biceps femoris long head (A.  $-60^\circ.s^{-1}$  and B.  $-180^\circ.s^{-1}$ )  
557 and medial hamstrings (C.  $-60^\circ.s^{-1}$  and D.  $-180^\circ.s^{-1}$ ) at 30, 50 and 100ms from the onset of  
558 electromyographical activity. Error bars indicate standard deviation. \* $p < 0.0167$  uninjured vs injured  
559 limbs.

560 **Figure 5:** Comparisons between the dominant and non-dominant limbs of uninjured athletes of  
561 integrated electromyography (iEMG) from the biceps femoris long head (A.  $-60^\circ.s^{-1}$  and B.  $-180^\circ.s^{-1}$ )  
562 and medial hamstrings (C.  $-60^\circ.s^{-1}$  and D.  $-180^\circ.s^{-1}$ ) at 30, 50 and 100ms from the onset of  
563 electromyographical activity. Error bars indicate standard deviation.

564