1 **TITLE**

2 Rate of torque and EMG development during anticipated eccentric contraction is lower in previously

3 strained hamstrings.

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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 and $-180^0.s^{-1}$. **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 $-60^0.s^{-1}$ eccentric contractions at 50 (RTD, injured limb = 312.27 ± 191.78Nm.s^{-1} vs. uninjured

33   limb = 518.54 ± 172.81Nm.s^{-1}, p=0.008; IMP, injured limb = 0.73 ± 0.30 Nm.s vs. uninjured limb =

34   0.97 ± 0.23 Nm.s, p=0.005) and 100ms (RTD, injured limb = 280.03 ± 131.42Nm.s^{-1} vs. uninjured limb

35   = 460.54.54 ± 152.94Nm.s^{-1},p=0.001; IMP, injured limb = 2.15 ± 0.89 Nm.s vs. uninjured limb = 3.07 ±

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 ($-60^0.s^{-1}$, normalised iEMG activity (x1000), injured limb =

38   26.25 ± 10.11 vs. uninjured limb 33.57 ± 8.29, p=0.009; $-180^0.s^{-1}$, normalised iEMG activity (x1000),

39   injured limb = 31.16 ± 10.01 vs. uninjured limb 39.64 ± 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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INTRODUCTION

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

Scant attention has been given to the potential for unattended neural maladaptations associated with a previous insult to increase the likelihood of future HSI. Recent work has reported lower levels of myoelectrical activity in the previously injured hamstring during maximal voluntary eccentric contractions tested at the movement speed of $-60^0.s^{-1}$.[40] That study was the first to provide empirical evidence that lower myoelectrical activity in a previously injured hamstring during maximal eccentric contractions exists. However many other aspects of neural function are yet to be examined. Myoelectrical activity during rapid force generation is one such avenue of further investigation. Such work is warranted given one of the primary roles of the hamstring muscle group is rapid deceleration of the advancing thigh during the terminal swing phase of high speed running.[41] Optimal hamstring function during this portion of the running cycle is important as terminal swing is considered by some to be most injurious phase of gait as it combines moderate muscle strains and high force eccentric contraction.[23, 37] As such, high rates of torque development (RTD) (Δtorque/Δtime) and early contractile impulse (IMP) (the area under the time vs. torque curve) during eccentric contractions are important characteristics of hamstring function because the

109   limited time available for deceleration (~100ms[30]) prevents the development of maximal torque.[43]

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,[5, 20, 22] 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.[1-2] 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.[14] This suggests that the

127   execution of motor activity is modulated according to the contraction type to be performed.[14]

128   Indeed contraction mode specific neural control has been evidenced previously via surface

129   electromyography (sEMG) with these anticipated eccentric contractions[17] 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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MATERIALS AND METHODS

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 ± 5.8 years; 1.8 ± 0.04m; 83.2 ± 14.3kg) had sustained at least one grade II HSI within

160   the last 36 months and another 13 athletes (25.9 ± 3.4 years; 1.8 ± 0.05m; 82.8 ± 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[40] 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)[4]; 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.[15]

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.[26] 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.

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 $+240^0.s-1$ 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 $-60^0.s^{-1}$ and $-180^0.s^{-1}$ 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 glueteus 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 ≥ 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 (Δtorque/Δtime) 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).[42] 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 MΩ;

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.[36] 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[46, 47] 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 ± 5.8

252 years; 1.8 ± 0.04m; 83.2 ± 14.3kg; Uninjured group, 25.9 ± 3.4 years; 1.8 ± 0.05m; 82.8 ± 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 $-60^0.s^{-1}$ anticipated

263 eccentric contractions at 50 (RTD, injured limb = 312.27 ± 191.78Nm.s^{-1} vs. uninjured limb = 518.54 ±

264 172.81Nm.s^{-1}, p=0.008, ES=1.12; IMP, injured limb = 0.73 ± 0.30 Nm.s vs. uninjured limb = 0.97 ±

265 0.23 Nm.s, p=0.005, ES=0.87) and 100ms (RTD, injured limb = 280.03 ± 131.42Nm.s^{-1} vs. uninjured

266 limb = 460.54.54 ± 152.94Nm.s^{-1},p=0.001, ES=1.27; IMP, injured limb = 2.15 ± 0.89 Nm.s vs.

267 uninjured limb = 3.07 ± 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 $-180^0.s^{-1}$ 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 ± 10.11 vs.

275   uninjured limb 33.57 ± 8.29, p=0.009, ES=0.80; $-180^0.s^{-1}$, injured limb = 31.16 ± 10.01 vs. uninjured

276   limb 39.64 ± 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. [7, 13, 35, 48]

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. [23, 37] 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.[42] 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[30]). 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.[33] Given fatigue

317 reduces the amount of energy that can be absorbed by a lengthening muscle[29] 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[34] or accumulating microscopic muscle damage[6] 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 $-180^0.s^{-1}$ 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,[39] which would be suggestive of an increased

339   volume of work during habitual activities. Moreover, BF atrophy has been found,[39] 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.[34] 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.[34] 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.[34] 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.[6, 10] Whilst much work

359   has been done on the contractile and structural[16, 27] 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.[11, 21, 38, 44] 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[34] (as opposed to heavily load or explosive

370   resistance exercise[19]) the altered function of the nervous system may differ markedly. HSIs induce

371   acute[45] and chronic[9, 28] 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.[12, 18-19, 25] 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.[31] Pain

377 has the potential to modulate descending neural pathways[32] 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[14] and sEMG[17] activity

402   compared to concentric contractions even when performing quasi-isometric contraction.[17] 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 $-180^0.s^{-1}$ 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 (Δtorque/Δtime) 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. $-60^{o}.s^{-1}$ and B. $-180^{o}.s^{-1}$) and impulse (C. $-60^{o}.s^{-1}$ and D. -

549    $180^{o}.s^{-1}$) at 30, 50 and 100ms from the onset of torque development. Error bars indicate standard

550   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. $-60^{o}.s^{-1}$ and B. $-180^{o}.s^{-1}$) and impulse (C. $-60^{o}.s^{-1}$ and D. $-180^{o}.s^{-1}$

553    $^{-1}$) at 30, 50 and 100ms from the onset of torque development. Error bars indicate standard

554   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. $-60^{o}.s^{-1}$ and B. $-180^{o}.s^{-1}$)

557   and medial hamstrings (C. $-60^{o}.s^{-1}$ and D. $-180^{o}.s^{-1}$) at 30, 50 and 100ms from the onset of

558   electromyographical activity. Error bars indicate standard deviation. *$p<0.0167$ uninjured vs injured
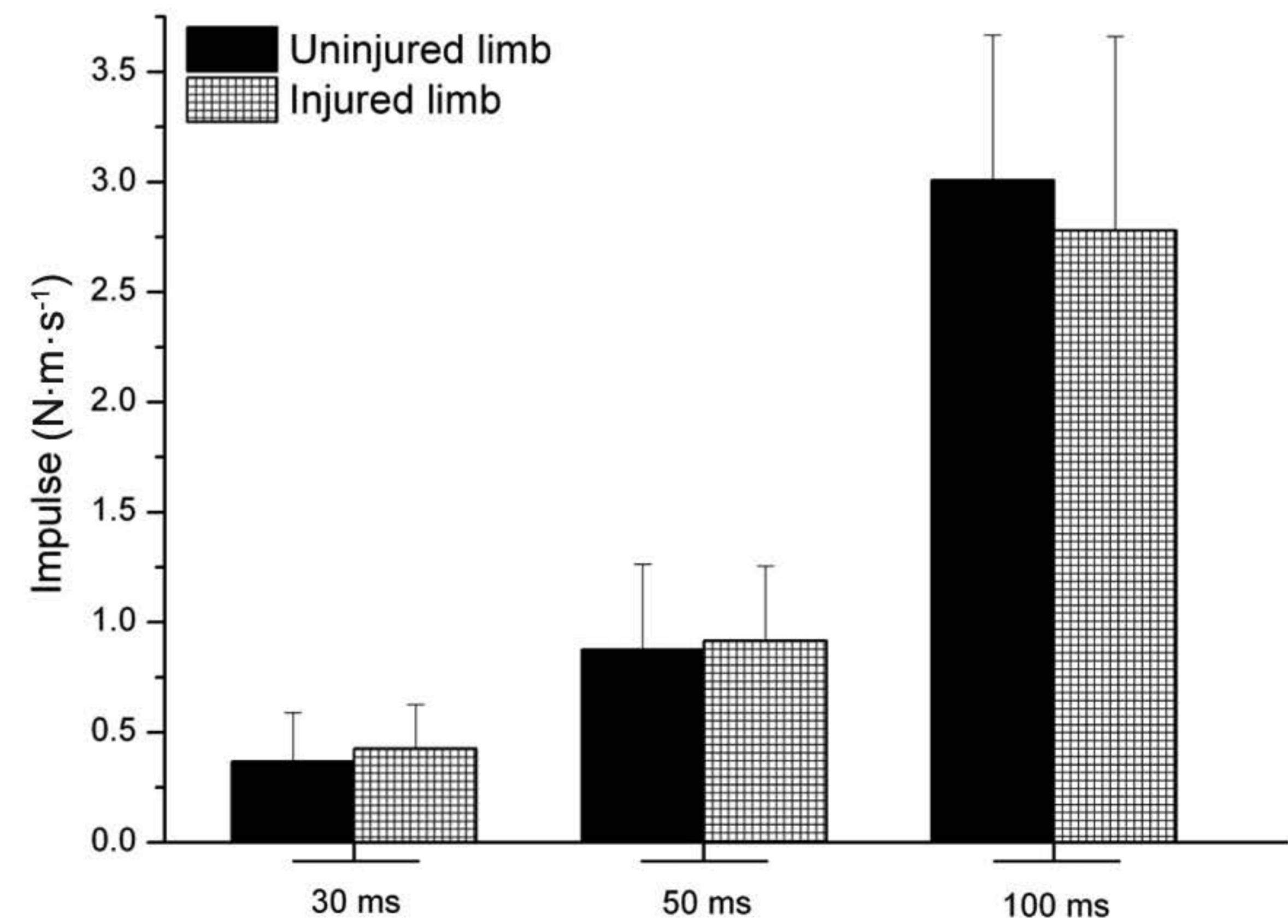
559   limbs.

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. $-60^{o}.s^{-1}$ and B. $-180^{o}.s^{-1}$)

562   and medial hamstrings (C. $-60^{o}.s^{-1}$ and D. $-180^{o}.s^{-1}$) at 30, 50 and 100ms from the onset of

563   electromyographical activity. Error bars indicate standard deviation.

564