TITLE

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

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ABSTRACT

Background: Hamstring strain injuries are prevalent in sport and re-injury rates have been high for many years. Whilst much focus has centred on the impact of previous hamstring strain injury on maximal eccentric strength, high rates of torque development is also of interest, given the important role of the hamstrings during the terminal swing phase of running. The impact of prior strain injury on myoelectrical activity of the hamstrings during tasks requiring high rates of torque development has received little attention. **Purpose:** To determine if recreational athletes with a history of unilateral hamstring strain injury, who have returned to training and competition, will exhibit lower levels of myoelectrical activity during eccentric contraction, rate of torque development and impulse 30, 50 and 100ms after the onset of myoelectrical activity or torque development in the previously injured limb compared to the uninjured limb. **Study design:** Case-control study **Methods:** Twenty-six recreational athletes were recruited. Of these, 13 athletes had a history of unilateral hamstring strain injury (all confined to biceps femoris long head) and 13 had no history of hamstring strain injury. Following familiarisation, all athletes undertook isokinetic dynamometry testing and surface electromyography assessment of the biceps femoris long head and medial hamstrings during eccentric contractions at -60 and -$180^{0}.s^{-1}$. **Results:** In the injured limb of the injured group, compared to the contralateral uninjured limb rate of torque development and impulse was lower during -$60^{0}.s^{-1}$ eccentric contractions at 50 (RTD, injured limb = 312.27 ± 191.78$Nm.s^{-1}$ vs. uninjured limb = 518.54 ± 172.81$Nm.s^{-1}$, p=0.008; IMP, injured limb = 0.73 ± 0.30 Nm.s vs. uninjured limb = 0.97 ± 0.23 Nm.s, p=0.005) and 100ms (RTD, injured limb = 280.03 ± 131.42$Nm.s^{-1}$ vs. uninjured limb = 460.54.54 ± 152.94$Nm.s^{-1}$,p=0.001; IMP, injured limb = 2.15 ± 0.89 Nm.s vs. uninjured limb = 3.07 ± 0.63 Nm.s, p<0.001) after the onset of contraction. Biceps femoris long head muscle activation was lower at 100ms at both contraction speeds (-$60^{0}.s^{-1}$, normalised iEMG activity (x1000), injured limb = 26.25 ± 10.11 vs. uninjured limb 33.57 ± 8.29, p=0.009; -$180^{0}.s^{-1}$, normalised iEMG activity (x1000), injured limb = 31.16 ± 10.01 vs. uninjured limb 39.64 ± 8.36, p=0.009). Medial hamstring activation did not differ between limbs in the injured group. Comparisons in the uninjured group showed no

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

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

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

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

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

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

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

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

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

MATERIALS AND METHODS

Participants

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

Injury questionnaire

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

EMG recording

Myoelectrical activity was measured via sEMG from the MH and BF through the use of circular bipolar pre-gelled Ag/AgCl sEMG electrodes (10mm diameter, 25mm inter-electrode distance). After 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.

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 $+240^0$.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

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

Data analysis

For each movement speed the three contractions with the highest peak torque were used for further analysis. Dynamometer torque and lever position data were transferred to a personal computer at 1 kHz and stored for later analysis. RTD was determined as the mean of the average slope of the torque-time trace (Δtorque/Δtime) for the three selected repetitions from the onset of contraction through until 30, 50 and 100ms of the contraction. Onset of contraction was defined as when torque deviated 4Nm from the baseline level of torque at rest (Figure 1).[42] IMP was calculated as the area under the torque-time trace across the same time periods.

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

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

Statistical analysis

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

RESULTS

Participants

The two groups were similar with respect to age, height and body mass (Injured group, 26.6 ± 5.8 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 athletes from the injured group had suffered at least one grade II HSI in the last 36 months. The total number of HSIs sustained by each athlete in the injured group ranged between one and four (median = 2) in the same 36 month period. All injuries were confined to the BF. Median time since most recent HSI was 3.9 months (range = 1.0 – 18.2), with median time taken to return to pre-injured levels of competition being 4 weeks (range = 2 - 6). All athletes from the injured group reported standard rehabilitation progression (i.e. Ref 24) guided by their physiotherapist, with all but one of the injured athletes reporting some eccentric conditioning as part of their late phase rehabilitation program.

RTD and IMP

RTD and IMP was significantly lower in the previously injured knee flexor for $-60^{0}.s^{-1}$ anticipated eccentric contractions at 50 (RTD, injured limb = 312.27 ± 191.78$Nm.s^{-1}$ vs. uninjured limb = 518.54 ± 172.81$Nm.s^{-1}$, p=0.008, ES=1.12; IMP, injured limb = 0.73 ± 0.30 Nm.s vs. uninjured limb = 0.97 ± 0.23 Nm.s, p=0.005, ES=0.87) and 100ms (RTD, injured limb = 280.03 ± 131.42$Nm.s^{-1}$ vs. uninjured limb = 460.54.54 ± 152.94$Nm.s^{-1}$,p=0.001, ES=1.27; IMP, injured limb = 2.15 ± 0.89 Nm.s vs. uninjured limb = 3.07 ± 0.63 Nm.s, p<0.001, ES=1.20) after the onset of contraction (Figure 2, Supplementary Table 1). There was no significant difference for RTD or IMP during anticipated eccentric contractions at $-180^{0}.s^{-1}$ at any time point (Figure 2, Supplementary Table 1). There were no between limb differences for either variable in the uninjured group (Figure 3, Supplementary Table 1).

Integrated EMG

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

DISCUSSION

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

This is, to our knowledge, the first study to examine RTD, IMP and concurrent myoelectrical activity in previously injured hamstrings, which makes comparisons to previous work difficult. One previous 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^{o} vs 70^{o} 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

hamstrings exceeding their mechanical limits[34] or accumulating microscopic muscle damage[6] and this increases the potential for injury/reinjury.

The observations that RTD and IMP were lower in anticipation of a slow, but not fast, eccentric contraction is intriguing given that the myoelectrical activity of the previously injured BF was lower in anticipation of both speeds of eccentric contraction. Whilst RTD was not lower in the previously injured limb compared to the contralateral uninjured limb at any time point at $-180^{0}.s^{-1}$ there was a medium effect size at 100ms following the onset of contraction ($p=0.064$, Cohen's d ES=0.57) and a larger sample may have revealed a significant difference . However this finding might also be indicative of alterations in coordination of the knee flexor muscles in anticipation of a fast eccentric contraction. Altered coordination may be driven by the intent to protect the previously injured BF in anticipation of a high speed eccentric action. In the case of this study other knee flexors, not examined, might be recruited more heavily thus increasing their contribution to knee flexion torque generation, with the most suited candidate being the uniarticular biceps femoris short head. Indirect evidence supports this change in contribution to knee flexion torque, given that a previously injured leg displays compensatory hypertrophy of this muscle,[39] which would be suggestive of an increased volume of work during habitual activities. Moreover, BF atrophy has been found,[39] as a possible consequence of reduced activation and disuse following HSI. Whether such a reorganisation of muscle activity exists is, however, yet to be explored and should be an area for future examination.

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

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

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

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

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

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

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

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FIGURE CAPTIONS

Figure 1: Representative torque-time trace. Prior to the onset of contraction baseline levels of torque were determined. Onset of contraction was defined as when knee flexor torque deviated by 4.0Nm from baseline. Rate of torque development was determined as the average change in torque over time (Δtorque/Δtime) at 30, 50, 100ms from onset of contraction development.

Figure 2: Comparisons between the uninjured and injured limbs of previously injured athletes of 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. $-180^{o}.s^{-1}$) at 30, 50 and 100ms from the onset of torque development. Error bars indicate standard deviation. *p<0.0167 uninjured vs injured limbs.

Figure 3: Comparisons between the dominant and non-dominant limbs of uninjured athletes of 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. $-180^{o}.s^{-1}$) at 30, 50 and 100ms from the onset of torque development. Error bars indicate standard deviation.

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

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