Title:

Reduced biceps femoris myoelectrical activity influences eccentric knee flexor weakness after repeat sprint running

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Hamstring EMG and weakness post running

Abstract

The aim of this study was to determine whether declines in knee flexor strength following overground repeat sprints were related to changes in hamstrings myoelectrical activity. Seventeen recreationally active males completed maximal isokinetic concentric and eccentric knee flexor strength assessments at $180^{0}.s^{-1}$ before and after repeat sprint running. Myoelectrical activity of the biceps femoris (BF) and medial hamstrings (MH) was measured during all isokinetic contractions. Repeated measures mixed model (Fixed factors = time [pre- and post- repeat sprint] and leg [dominant and non-dominant], random factor = participants) design was fitted with the restricted maximal likelihood method. Repeat sprint running resulted in significant declines in eccentric, and concentric, knee flexor strength (eccentric = 25 ± 34 Nm, 15% $p<0.001$; concentric 11 Nm± 22 Nm, 10% $p = 0.001$). Eccentric BF myoelectrical activity was significantly reduced (10%; $p= 0.033$). Concentric BF and all MH myoelectrical activity were not altered. The declines in maximal eccentric torque were associated with the change in eccentric biceps femoris myoelectrical activity ($p = 0.013$). Following repeat sprint running there were preferential declines in the myoelectrical activity of the BF, which explained declines in eccentric knee flexor strength.

Key terms: Fatigue; isokinetic dynamometry; hamstring injury; eccentric; repeat sprint; surface electromyography

Introduction

Hamstring strain injuries (HSIs) are the predominant injury type in many sports (Brooks, Fuller, Kemp, & Reddin, 2006; Ekstrand, Hagglund, & Walden, 2011; Orchard, Seward, & Orchard, 2013), with a number of risk factors identified (Opar, Williams, & Shield, 2012). Despite increased attention being placed on developing better prevention programs (Arnason, Andersen, Holme, Engebretsen, & Bahr, 2008; Askling, Tengvar, & Thorstensson, 2013), HSIs still occur and reoccur frequently (Brooks et al., 2006; Orchard et al., 2013). The combination of high incidence and recurrence rates (Opar et al., 2012), significant cost in terms of financial and lost time from training and competition (Hickey, Shield, Williams, & Opar, 2013; Orchard et al., 2013; Woods et al., 2004), as well as compromised performance levels upon return from injury (Verrall, Kalairajah, Slavotinek, & Spriggins, 2006), all make the management of HSI particularly challenging for both clinicians and athletes.

HSIs occur most commonly during high speed running (Askling, Tengvar, Saartok, & Thorstensson, 2007; Brooks et al., 2006) and often involve the long head of the biceps femoris (BF) (Koulouris, Connell, Brukner, & Schneider-Kolsky, 2007). It has been proposed that the terminal swing phase, where the hip is flexed and the knee is extending rapidly, is when the hamstrings are most vulnerable to injury (Schache, Dorn, Blanch, Brown, & Pandy, 2011; Thelen et al., 2005). The high levels of force required from the hamstrings to decelerate these movements via eccentric contractions, coupled with the increasing muscle strain, are proposed mechanisms for HSI (Opar et al., 2012; Schache et al., 2011; Thelen et al., 2005). Furthermore, fatigue has also been implicated in HSI aetiology, with prolonged match time resulting in an increase in HSI incidence (Brooks et al., 2006; Ekstrand et al., 2011). Previous research has reported that a soccer-specific running protocol results in preferential declines in eccentric knee flexor strength with minimal changes in concentric strength (Greig, 2008; Small, McNaughton, Greig, & Lovell, 2010). This suggests that prolonged intermittent running may increase the likelihood of HSI due to a reduction in eccentric hamstring strength, which is a noted risk factor for injury (Croisier, Ganteaume, Binet, Genty, & Ferret, 2008; Sugiura, Saito, Sakuraba, Sakuma, & Suzuki, 2008). However, the mechanism responsible for this contraction-mode-specific

decline in strength following prolonged intermittent running remains unknown. One possible explanation is a decline in activation of the hamstring muscles. Determination of whether reductions in the myoelectrical activity of the medial hamstrings (MH) and BF muscles explain the decline in eccentric knee flexor torque after running would inform whether reduced activation is somewhat responsible for this loss of strength. It is also unknown whether the changes in strength observed after prolonged bouts of intermittent running (Greig, 2008; Small et al., 2010) also occur after relatively short duration, high-intensity efforts. Given the importance of repeat sprint running in elite sport and reports of high-speed running being particularly injurious (Schache et al., 2011; Thelen et al., 2005), hamstring function following repeat sprint running requires examination.

No previous study has investigated the effects of a repeat sprint running protocol on knee flexor strength and hamstring myoelectrical activity. Hence, the aim of this study was to determine if reductions in knee flexor strength occur following repeat sprint running. Furthermore, we aimed to determine if these reductions in strength were associated with changes in hamstring myoelectrical activity. A thorough understanding of how hamstring activation patterns are altered as a consequence of high speed running is important; as such information could be used to develop better interventions for protecting the hamstrings against injury.

Methods

Seventeen recreationally active males (mean age of 23.3 ± 2.6 years; height 1.81 ± 0.06m; body mass 80.2 ±7.5kg) were recruited. None of the participants had a history of any lower limb injury in the past 36 months. Each participant provided written informed consent prior to undertaking their first session and approval for the study was obtained by the University Human Research Ethics Committee.

The exercise testing session consisted of three sets of six 20-metre maximal overground (grass surface) sprints with a 10-metre acceleration distance and 15-metres for deceleration. Rest periods of 90 and 240 seconds were employed between repetitions and sets, respectively. The protocol was based on elite European soccer data that shows the mean total sprint distance, in a competitive game, to be between 237 and 345 metres (Andrzejewski, Chmura, Pluta, Strzelczyk, & Kasprzak, 2012). Sprint performance was measured using dual-beamed, laser timing gates (Model WL250-P132, Sick Optex, Japan). Prior to and after completing the sprinting protocol, participants undertook a maximal isokinetic dynamometry strength test of the knee flexors. Post run testing occurred within 15 minutes after the sprinting session.

Participants completed two sessions of testing on a Biodex® System 3 isokinetic dynamometer (Shirley, NY). All tests were conducted on both legs (dominant or non-dominant) and testing order was randomised. Limb dominance was defined as the leg most often used for kicking a ball. A familiarisation session was completed on average 7 ± 1days prior to the testing session. Participants were seated on the dynamometer with a hip angle that was approximately 85° from full extension and were restrained by straps around the tested thigh, waist and chest to prevent compensatory movements. All seating variables (e.g. seat height, pad position, etc) were recorded to ensure the replication of the participants' positions. Gravity correction for limb weight was also conducted and range of motion was set between 5° and 90° of knee flexion (full extension = 0°) with the starting position for each contraction being 90° of knee flexion. Concentric contractions of the knee flexors were conducted from 5° to 90°, with eccentric efforts being from 90° to 5°. Prior to performing

maximal efforts, participants undertook a warm-up consisting of three sets of four concentric knee extension and flexion contractions at an angular velocity of $240°\cdot s^{-1}$. The intensity of these contractions increased each set until the final set at this velocity was performed at a maximal level. The test protocol began one minute following the final warm-up set and consisted of three sets of three concentric and eccentric MVCs of knee flexion at $180°\cdot s^{-1}$ with 30s rest between sets. The testing speed was chosen based on previous research which has investigated the effect of fatigue on knee flexor strength (Greig, 2008). All participants were verbally encouraged by the investigators to ensure maximal effort for all contractions. The testing order of contraction modes was randomised across the participant pool. To determine the impact of the isokinetic testing protocol on knee flexor strength a small pilot study (n = 5) was conducted. The participants completed three sets of three concentric and eccentric knee flexor MVCs at $180°\cdot s^{-1}$and rested passively for 2 hours. Following the rest period, the same testing protocol was repeated. Concentric and eccentric strength was not altered following the testing protocol (concentric $180°\cdot s^{-1}$: 7.7Nm; 95%CI = -29.1 to 13.7; p = 0.188; *d* = 0.51,eccentric $180°\cdot s^{-1}$: 5.6Nm; 95%CI = -31.4 to 20.1Nm; p = 0.289;*d* = 0.17).

Bipolar pre-gelled Ag/AgCl surface electromyography (sEMG) electrodes (10mm diameter, 25mm inter-electrode distance) were used to record myoelectrical activity from the MH and BF. After preparation of the skin, electrodes were placed on the posterior thigh half way between the ischial tuberosity and tibial epicondyles with electrodes oriented parallel to the line between these two land marks, as per SENIAM guidelines (Hermens, Freriks, Disselhorst-Klug, & Rau, 2000). The reference electrode was placed on the ipsilateral fibula head. Muscle bellies were identified via palpation during forceful isometric knee flexion and correct placement was confirmed by observing sEMG activity during active internal and external rotation of the flexed knee to assess cross talk between MH and BF. Once confirmed, an outline was then traced around the electrodes to ensure correct positioning following the sprinting protocol. As the participants completed the isokinetic testing in a seated position, custom made foam padding was used to minimise any movement artefact that may be caused from contact with the dynamometer chair.

Dynamometer torque and lever position data were transferred to computer at 1kHz and stored for later analysis. Average peak torques for concentric and eccentric knee flexion were defined as the means of the six highest torque values for each contraction mode. sEMG was sampled simultaneously with the dynamometer data at 1kHz through a 16-bit PowerLab26T AD recording unit (ADInstruments, New South Wales, Australia) (amplification = 1000; common mode rejection ratio = 110dB) and was filtered utilising a Bessell filter with a frequency bandwidth of 10 to 500Hz and then rectified using the root-mean-square method. At each contraction mode EMG data were averaged across a knee joint ROM between 15^{o}-35^{o} (full knee extension = 0°). The angle of peak torque was between 18.2° to 33.6° concentrically and 15.2° to 32.3° during eccentric contractions. sEMG data at all velocities were then normalised as a quotient of the average EMG signal during concentric knee flexion at $180°\cdot s^{-1}$ obtained in the pre-test(Aagaard et al., 2000).

All data were entered into JMP version 10.01 Pro Statistical Discovery Software (SAS Inc) and analysed using a mixed model repeated measures design fitted with the restricted maximum likelihood (REML) method. The analysis was a two tiered approach. The first tier was based on the hypothesis that changes in knee flexor strength and myoelectrical activity following repeat sprint running would be specific to contraction mode. Repeated measures mixed model (Fixed factors = time [pre- and post- repeat sprint] andleg [dominant and nondominant], random factor = participants) design was used.

Once it was established that changes in knee flexor strength and myoelectrical activity occurred, the second tier of linear analysis was employed. This was based on the premise that knee flexor torque is explained partly by combined MH and BF activity and therefore the changes in knee flexor torque at a given velocity could be related to the changes in myoelectrical activity of the hamstrings. The response variable modelled was the change in knee flexor torque (pre- minus post-running test) and the fixed factors were change in MH myoelectrical activity, change in BF myoelectrical activity and leg (dominant and non-dominant), with participants as random factors. This analysis was performed separately for the concentric and eccentric contraction modes. Any differences were considered significant with a p value <0.05. Furthermore, Cohen *d* effect sizes were calculated between tests (pre

and post running) with the levels of effect size being deemed small (d = 0.20), medium (d = 0.50) or large (d = 0.80) as recommended by Cohen (Cohen, 1988).

Results

Peak eccentric knee flexor torque declined by 15% (25.7 Nm; 95%CI = 18.0 to 33.2 Nm, p< 0.001; d = 0.63) while peak concentric torque declined by 10% (11.1 Nm; 95%CI = 6.3 to 16.0 Nm; p = 0.001; d = 0.48) after the sprint protocol (Fig.1). No significant difference between legs (eccentric p = 0.073; concentric p = 0.105) or leg by time interactions (eccentric p = 0.610; concentric p = 0.999) were observed.Normalised BF myoelectrical activity during the eccentric knee flexor contractions was significantly reduced by 10% (0.068; 95% CI = 0.005 to 0.131; p= 0.035; d = 0.33; Fig. 2) after the repeat sprints. In addition, normalised BF myoelectrical activity of the dominant leg in eccentric actions was greater than that of the non-dominant leg (11%; 0.076; 95% CI= 0.013 to 0.139; p = 0.020; d = 0.37) , however no time by leg interaction was observed (p = 0.724). Furthermore, BF myoelectrical activity during the concentric contractions displayed no significant change after sprint running (3%; 0.024; 95% CI=-0.094 to 0.045; p= 0.483; d = 0.12; Fig. 2) and no significant effects for leg (p = 0.903) or time by leg interaction (p = 0.903). Furthermore, normalised MH myoelectrical activity did not change after sprinting for either contraction mode (eccentric: 6%; 0.037; 95% CI= -0.04 to 0.113; p= 0.0.342; d = 0.16; Fig. 2, concentric: 6%; 0.062; 95% CI= -0.025 to 0.148; p= 0.089; d = 0.23; Fig. 2). Additionally, no significant main effects for normalised MH myoelectrical activity were found in leg (concentric p = 0.694; eccentric p = 0.417) or the time by leg interactions (concentric p = 0.694; eccentric p = 0.722).

Changes in hamstring myolectrical activity were able to explain changes in eccentric knee flexor torque following repeat sprint running (whole model R^2 = 0.69, p < 0.001) (see Figure 3 for an exemplar which illustrates the relationship during the eccentric contraction for both the changes in strength and BF myoelectrical activity). More specifically, it was change in the BF myoelectrical activity that was related to the decrease in knee flexor torque (p = 0.013) while no effects for the changes in MH myoelectrical activity (p = 0.372), or leg (p = 0.486) were found. For the concentric

contractions no significant effects were observed (MH myoelectrical activity $p = 0.984$; BF myoelectrical activity $p = 0.355$; leg $p = 0.973$).

Discussion

The main objective of the present study was to examine the impact of a repeat sprint running protocol on isokinetic knee flexor strength and hamstrings myoelectrical activity. The main finding was a reduction in eccentric knee flexor strength that was related to a reduction in BF myoelectrical activity. By contrast, changes in MH myoelectrical activity were statistically insignificant and therefore not related to the changes in strength.

Previous work investigating declines in knee flexor strength following running has primarily used intermittent running protocols designed to mimic the physiological demands of soccer (Greig, 2008; Rahnama, Reilly, Lees, & Graham-Smith, 2003; Small et al., 2010). These protocols have resulted in declines of between 17-18% in eccentric strength and 5-15% in concentric strength. These results are similar to those from the current study, where 15% and 10% declines were observed in eccentric and concentric torque respectively. No previous studies have determined whether or not such strength declines are related to changes in hamstrings myoelectrical activity.

HSIs commonly occur during high-speed running (Askling et al., 2007; Woods et al., 2004) and more often involve the BF than the medial hamstrings (Koulouris et al., 2007; Opar et al., 2012). There is also a tendency for most of these injuries to occur towards the end of each half in soccer (Woods et al., 2004) and rugby union matches (Brooks et al., 2006) and this suggests a role for fatigue in HSI aetiology. It is possible that the decline in eccentric knee flexor strength following repeat sprint running might increase injury susceptibility (Croisier et al., 2008; Sugiura et al., 2008) and while muscular metabolic changes undoubtedly explain a significant portion of muscle weakness after this sort of exercise (Bishop & Edge, 2006; Bishop, Lawrence, & Spencer, 2003; Davies, Eston, Fulford, Rowlands, & Jones, 2011), our observations suggest that reduced BF activation explains at least part of this strength loss. This finding may at least partially explain why BF is the primary hamstring head involved in HSI (Koulouris et al., 2007; Opar et al., 2012). *In-situ* animal experiments have found that

sub-maximal activation of a lengthening muscle reduces the amount of energy it can absorb before stretch induced failure occurs (Mair, Seaber, Glisson, & Garrett, 1996). Similar *in-situ* observations have also been reported in pre-fatigued muscle under lengthening conditions (Garrett, Safran, Seaber, Glisson, & Ribbeck, 1987). Additionally, of all the hamstring muscles, the BF undergoes the greatest amount of musculotendinous strain during high speed running (Schache et al., 2011; Thelen et al., 2005). As the level of muscle damage that occurs following eccentric contractions is a function of the strain within the musculotendinous unit (Lieber & Friden, 1993), it is thought that the extent of damage after repeat sprint running would also be augmented. Furthermore, as the terminal swing phase of running requires a high force eccentric contraction (Schache et al., 2011; Thelen et al., 2005), a reduced capacity to absorb energy might be expected to increase the chance of strain injury to BF.

We can only speculate as to why the myoelectrical activity of the BF muscle declined while the MH remained unaffected. There are a number of reports of structural and functional differences between the hamstring muscle heads (Woodley & Mercer, 2005) and these may play a role in determining muscle-specific responses to sprint running. For example, the BF experiences larger peak strains during the terminal swing phase of running than the MH (Thelen et al., 2005) and these may predispose the former to greater muscle damage (Garrett et al., 1987). Additionally, exercise induced muscle damage has been shown to result in significant reductions of voluntary activation (Endoh, Nakajima, Sakamoto, & Komiyama, 2005; Skurvydas, Brazaitis, Kamandulis, & Sipaviciene, 2010) and EMG (Beck, Kasishke, Stock, & DeFreitas, 2012). Furthermore such damage may increase afferent feedback which may act to reduce the myoelectrical activity of the BF in an attempt to minimise exposure to the damaging stimulus (Marqueste et al., 2004). Such a response might be perceived to have short term benefits (e.g. limit the amount of strain during eccentric contractions) but would most likely be counterproductive from the perspective of strain injury avoidance given the potential impact of reduced myoelectrical activity on the energy absorption capabilities of muscle (Mair et al., 1996). Also, the shorter fascicles of the BF (Woodley & Mercer, 2005) may potentially explain why this muscle is particularly prone to injury during high speed running. Eccentrically induced hamstring muscle damage has also been shown to alter position sense of the knee joint

(Paschalis et al., 2008), although the impact of damage on other aspects of neural control remains largely unexamined.

Some limitations exist within the present study. First and foremost, not all of the muscles which contribute to the production of knee flexor torque had their myoelectrical activity assessed before and after repeat sprint running, which means that the impact of this exercise on the activity of sartorious, gastrocnemius and gracilis could not be determined. Nevertheless, the hamstrings constitute the majority of the muscular cross sectional area crossing the posterior aspect of the knee joint (Woodley & Mercer, 2005) and would be expected to have the greatest influence on knee flexor strength (Lieber & Ward, 2011). Secondly, the isokinetic movement velocity utilised in the present study is much lower than the knee joint angular velocities noted during the terminal swing phase of sprint running. Current dynamometers do not allow assessments of torque at speeds above 300-500°·s^{-1}. We chose to limit our movement velocity to 180°·s^{-1} to allow comparisons with previous literature (Greig, 2008) and because we have found torque generation at faster speeds to be less reliable. It should also be acknowledged that knee extension velocities slow from maximum angular velocities of approximately 1000-1200°·s^{-1} (Schache et al., 2011; Thelen et al., 2005) to zero during the swing phase of sprinting and there is no reason to believe that testing at lower velocities is less indicative of eccentric muscle function than higher speeds in this range.

Whilst there is some error previously reported with isokinetic testing, we have found a high level of reproducibility. Our laboratory has previously examined the test-retest reliability using the exact protocol and Biodex® System 3 isokinetic dynamometer of the current study. We obtained intraclass correlations (ICCs) and typical error as a coefficient of variation (%TE) for peak knee flexor torque under both concentric 180°·s^{-1} (ICC= 0.93; TE% = 4.5%) and eccentric 180°·s^{-1} (ICC = 0.82; TE%= 6.0%) conditions. These data are similar to values of test-retest reliability of maximal knee flexor torque (ICC = 0.97 and 0.96) previously reported within the literature (Feiring, Ellenbecker, & Derscheid, 1990; Tsiros, Grimshaw, Shield, & Buckley, 2011) utilising the Biodex® isokinetic dynamometer.

We must acknowledge that the decline in eccentric strength observed here may also be due to other factors not measured within this study. Previous investigations have shown an altered muscle coordination pattern, as well as an augmented agonist-antagonist co-activation sequence to be partly responsible for these reductions in strength when fatigued (Psek & Cafarelli, 1993; Rodacki, Fowler, & Bennett, 2001). Additionally, the assessment of hamstring myoelectrical activity is not completely representative of the voluntary activation capacity within the muscle. The twitch interpolation technique is considered the most accurate way of determining muscle activation during voluntary contractions (Shield & Zhou, 2004), however, the use of this technique within the hamstrings is yet to be reported in the literature. Finally, it should be acknowledged that electromyography is not without limitations as it is influenced not only by factors related to the extent of muscle activation (motor unit recruitment and firing rates) but also by the degree of motor unit synchrony (Yao, Fuglevand, & Enoka, 2000) Nevertheless, observations of lower levels of activation in eccentric than concentric maximal contractions are supported by studies employing superimposed electrical stimulation(Amiridis et al., 1996; Beltman, Sargeant, van Mechelen, & de Haan, 2004; Westing, Cresswell, & Thorstensson, 1991), so it seems likely that the current measures of myoelectrical activity are reflective of muscle activation.

Conclusion

In conclusion, this study found that following repeat sprint running there was a decline in eccentric knee flexor strength that was related to a significant decline in the myoelectrical activity of the BF. Declines in BF myoelectrical activity following repeat sprint running and its role in the aetiology of HSIs still require further attention.

Perspectives

This study demonstrated a significantly lowered eccentric knee flexor strength following repeat sprint running. This is of interest as eccentric weakness and prolonged game time are risk factors within the aetiology of HSIs (Croisier et al., 2008; Sugiura et al., 2008; Woods et al., 2004). Furthermore, the

303 eccentric myoelectrical activity of the BF was also significantly reduced and this decrease in
304 myoelectrical activity was responsible for the reduction in eccentric knee flexor strength. As the BF is
305 the most frequently injured of the hamstring muscles (Koulouris et al., 2007)alterations in its
306 neuromuscular function following running are cause for further work to better understand the
307 relationship between fatigue and HSI risk.
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Conflict of interest

The authors report no conflicts of interest.

315 **Figure 1: Knee flexor peak torque at twodifferent isokinetic contraction modes before (Pre) and**
316 **after (Post) a sprinting session. Error bars illustrate the standard deviation. * p <0.05 pre vs**
317 **post.**

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Figure 2: Knee flexor normalised EMG in concentric and eccentric actions before (Pre) and after (Post) a sprinting session for both the A) medial hamstrings and B) biceps femoris. Error bars illustrate the standard deviation. * p <0.05 pre vs post.

Figure 3: Exemplar from a single participant. Comparison of knee flexor torque and normalised biceps femoris EMG activity before (Pre) and after (Post) sprinting session at A) concentric B) eccentric. Note for the whole group data, that only at eccentric $180^{0}.s^{-1}$ could the decline in torque be explained by the decline in biceps femoris EMG activity.

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