Is neuromuscular inhibition detectable in elite footballers during the Nordic hamstring exercise?

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Abstract

Background: The presence of neuromuscular inhibition following injury may explain the high incidence of biceps femoris injury recurrence in elite (soccer) footballers. This phenomenon may be detectable in elite players during the Nordic hamstring exercise. Thus, the first purpose of this study was to assess biceps femoris muscle activation during this exercise in players with hamstring injury history. Additionally, following injury, observed increases in synergistic muscle activation may represent a protective mechanism to the presence of neuromuscular inhibition. Thus, the second purpose was to identify if the relative contributions of biceps femoris, and its synergists reflected a post-injury pattern of activation suggestive of these potentially compensatory neural mechanisms.

Methods: Ten elite players with a history of hamstring injury and ten elite players without a history of hamstring injury, completed six repetitions of the Nordic hamstring exercise. During each trial, biceps femoris, semitendinosus and gluteus maximus muscle activations were collected at 90-30º and 30-0º of knee flexion.

Findings: Biceps femoris activation was significantly higher at 90-30º of knee flexion compared to 30-0º ($P < 0.001$) but did not differ between the groups. In players with a history of injury, muscle activation ratios for the biceps femoris/semitendinosus ($P = 0.001$) and biceps femoris/gluteus maximus ($P = 0.023$) were significantly greater at 30-0º of knee flexion than in the control group.

Interpretation: Neuromuscular inhibition of the biceps femoris was not detected during the exercise within elite footballers, yet the relative contributions of biceps femoris and its synergists appear to change following injury.

Keywords: *Hamstring injury recurrence, activation ratios*

1. Introduction

Hamstring strain injury is reportedly high within professional (soccer) football[1] despite extensive investigation seeking to address the incidence and recurrence of injury.[2,3,4] Although working synergistically at the hip and knee, the individual hamstring muscles differ not only in architecture and morphology,[5] but also in their susceptibility to injury. The majority of hamstring strain injuries may primarily occur during the terminal swing phase of sprinting[6] where peak activation of biceps femoris (BF) muscle and peak muscle elongation occur synchronously to decelerate the knee and hip.[7] These high activation levels and rapid lengthening demands may partially explain why the BF muscle is more susceptible to injury compared to the other hamstring muscles.[1]

Strategies to reduce hamstring strain injury have been primarily aimed at matching the lengthening and loading characteristics of the swing phase in sprinting to enhance knee flexor force production during eccentric contractions.[8,9] One such strategy associated with successfully reducing hamstring strain injury occurrence in football is the Nordic Hamstring Exercise (NHE).[10,11] Petersen et al. (2011) reported the NHE to be an effective strategy to reduce initial hamstring injury in football players.[11] However, in players with a previous hamstring strain injury, the NHE's protective effect proved less successful in preventing subsequent injury. One explanation for this difference may be neuromuscular inhibition following an initial hamstring strain injury[12] whereby reductions in muscle activation occur during eccentric contractions.[13,14,15] For example, acute reductions in eccentric muscle activation were present in the BF muscle during the final 30º prior to full knee extension of a seated leg curl exercise, in participants who had previously had a

hamstring strain injury.[14,15] This reduction in acute activation during eccentric exercise may offer some explanation as to why the NHE is less effective in improving the incidence of hamstring strain injury in players with a history of injury. However, before this assumption can be made, it is important to understand whether the reduced BF activation accompanying the long muscle lengths associated with the eccentric phase of the seated leg curl, is also evident at the shorter muscles lengths characteristic of the NHE. Although prior investigation has identified previously injured hamstrings may differ in their response to the NHE,[16] suggestive of the presence of neuromuscular inhibition, acute activation deficits have not been observed at these muscle lengths nor in an elite football population. Such a finding may offer some explanation to the divergence of injury rates between players experiencing recurrence of injury compared to an initial injury following the use of the NHE. Therefore, the first purpose of this study was to compare BF muscle activation at two discrete epochs of knee excursion (90-30º and 30-0º of knee flexion) during the NHE in players who had suffered a previous hamstring strain injury.

Previous research has suggested that reduced muscle activation in players with previous hamstring strain injury may be accompanied by changes in the relative contribution of other muscle synergists.[17] For example, in the presence of reduced BF muscle activation following injury, the recruitment of the gluteus maximus (GM) has been shown to be greater in comparison to controls during the terminal swing phase of sprinting.[17] This increased GM muscle activation may serve to reduce eccentric activity within the BF muscle,[18] potentially representing a compensatory mechanism to the presence of neuromuscular inhibition following injury. Indeed, footballers demonstrating higher GM

muscle activation levels during sprinting sustained fewer hamstring injuries in the competitive season following testing.[19] Changes in the relative contribution of muscle activation following injury may also be apparent between the hamstrings muscles.[20] Within injury-free individuals, the relative contribution of the hamstrings during the NHE has been reported through the use of activation ratios, identifying a bias in contribution towards the semitendinosus (ST).[2] Following injury, reduced activation of the BF muscle is likely to reveal activation ratios illustrating a shift towards greater relative GM and ST contribution compared to players with no history of injury. Such a finding would highlight that the NHE elicits a different pattern of muscle recruitment following injury; an observation likely to impact training programme design for those seeking to limit injury recurrence. Therefore, the second purpose of the study was to compare activation ratios of the BF and ST, and the BF and GM at 90-30º and 30-0º of knee flexion during the NHE in players with a history of hamstring strain injury and those without.

2. Methods

2.1 Participants

Twenty (mean age 18.7 y SD 1.08 y; mean stature, 1.82 m SD 0.07 m; mean body mass 76.4 kg SD 7.89 kg; elite youth (academy/U23 squad) male, outfield footballers, regularly exposed to the NHE, were recruited to participate. Participants were currently healthy (clear health questionnaire), available for selection, and absent of anterior cruciate ligament reconstruction. Based on club physician's data, 10 players (age 18.9 y SD 1.3 y; stature 1.83 m SD 0.07 m; body mass 77.4 kg SD 6.8 kg) met inclusion criteria (experiencing hamstring strain injury within the last 12 months leading to absence from

training or selection availability) to be placed in the hamstring strain injury group. Additionally, 10 players (age 18.4 y SD 0.8 y; stature 1.80 m SD 0.06 m; body mass 75.4 kg SD 9.0 kg) formed a matched-pairs control group identified as never experiencing a previous hamstring injury. Pairs were matched by limb dominance (preferred kicking leg) and body mass index (BMI Z-score) (P = 0.436). The University's Research Ethics committee approved all procedures, and signed informed consent were obtained from each participant and, where relevant, their parents prior to the study's commencement.

2.2 Experimental setup

The study followed a cross sectional design. Participants from both groups performed six repetitions of the NHE with minimal periods of rest between each descent. To standardise velocity of movement, participants were instructed to attempt to execute each repetition of the NHE with a constant knee extension velocity performed to a strictly monitored six second count.[21] During each repetition, joint position and muscle activity of the BF, GM and ST muscles were synchronously recorded. To prepare the skin for electromyography (EMG), hair and skin cells were removed by shaving, abrading and wiping the skin with alcohol. Two bipolar surface electrodes (DE– 2.3 MA; Delsys Inc., Boston, MA, USA) were placed 10 mm apart on the muscle belly of the BF, the GM and the ST in accordance with SENIAM guidelines[22] of the previously injured limb and the corresponding limb of the matched pair individual. Sensors were secured with tape to minimise motion artefact. A ground electrode (20 mm contact diameter) was fixed to the olecranon process of the right arm. A single axis electro-goniometer (S700; Measureand Inc., Fredericton, NB, Canada) was secured to each participant's right knee during standing (0º flexion) ensuring

the device's axis of rotation was positioned over the lateral femoral epicondyle. The proximal arm of the electro-goniometer was attached on the lateral aspect of the thigh, aligned with the lateral midline of the femur (employing the greater trochanter as a reference). The lateral aspect of the shank served as an attachment for the device's distal arm with the lateral malleolus acting as a reference point. Kinematic data were collected synchronously with EMG through a 16 bit, eight-channel telemetry system (Delsys Myomonitor IV, Delsys Inc., Boston, MA, USA) sampled at 1000 Hz.

2.3 Procedures

In order to normalise muscle activation during the NHE, maximal activation was required for each muscle of interest. For this purpose, a maximal voluntary contraction of the BF and ST was performed with the participant lying prone, with a knee flexion angle of 45º and a hip angle of 0º.[23] The lower leg was fixed in position and each participant completed three, five second maximal contractions whilst muscle activation was recorded. With the knee fixed at 90º flexion and the hip at 0º,[24] three further maximal contractions were performed for five seconds, to determine the maximal activation of the GM muscle.

From a high-kneeling start position with the ankles secured by a partner, each participant then performed six NHE repetitions. Strong verbal encouragement was provided throughout. Participants were instructed to resist the forward fall through the engagement of the hamstrings whilst adhering to the specified exercise tempo and maintaining a lumbo-pelvic neutral alignment until contacting the floor on completion of each repetition.

2.4 Data Processing and Analysis

All EMG data were processed by full wave rectification and filtered using a fourth order zero-lag Butterworth filter with a cut-off frequency of four Hz. Maximal EMG amplitudes were defined as the average of 150 ms before and after peak amplitude. An average of three maximal voluntary contractions was used for normalisation of the muscle activity for the GM, BF and ST during the NHE trials. Peak muscle activity during the NHE trials were identified and averaged across repetitions two to five. Average NHE EMG amplitudes were expressed as a percentage of maximal muscle activity for the BF, ST and GM.

Activation values for all muscles of interest were calculated at two epochs of knee angle excursion: 90-30º of knee flexion and 30-0º of knee flexion during the descent phase of the NHE. Initiation and termination of each repetition were determined from threshold values, set as two standard deviations above baseline. For initiation, baseline muscle activity at 90º was averaged to derive the threshold value, and for termination, an average of peak activation at approximately 0º determined baseline (termination) (Figure 1). Peak EMG values were calculated for each repetition at 90-30º and 30-0º using custom written analysis software (R, Version 3.2.1, The R Foundation for Statistical Computing Platform, Vienna, Austria). Peak normalised muscle activations were combined to derive the activation ratios at both 90-30º and 30-0º epochs: BF/ST and BF/GM. Ratios greater than 1.0 indicated a greater contribution from BF compared to the ST and GM, respectively.

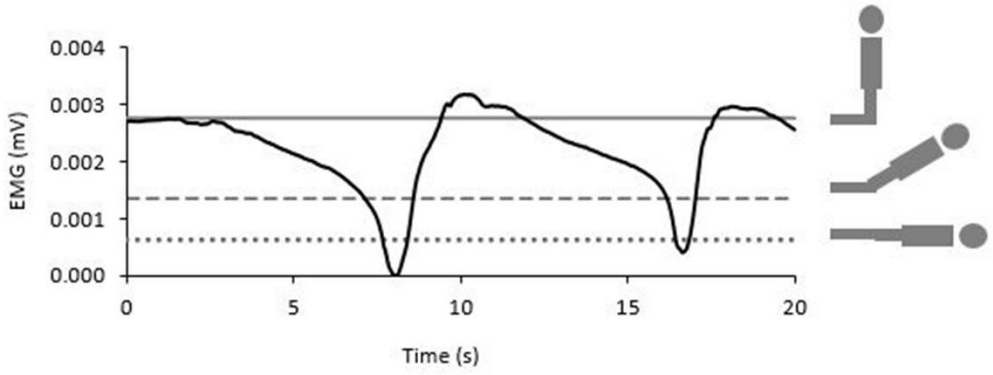

Figure 1 Two NHE repetitions with thresholds corresponding to the initiation of the exercise at 90º knee flexion (solid grey line), 30º (dash line) and 0º (dotted lined).

2.5 Statistical Analysis

To match individuals each player's BMI was calculated and expressed as an age-specific BMI Z-score[25] (Cole, Freeman, & Preece, 1995) and compared between groups using an independent t-test. To address purpose 1, a mixed 2 x 2 ANOVA was performed to assess differences in BF muscle activation at both 90-30º and 30-0º epochs between injury free players and those with a history of previous injury. To address purpose 2, a 2 x 3 mixed design ANOVA was used to assess differences in BF/ST and BF/GM ratios at 90-30º and 30-0º epochs between injury free players and those with a history of previous injury. In case of significance, post hoc tests were performed to determine the separate effects of injury history and angle of knee flexion on BF activation and BF/ST and BF/GM ratios, through the use of MANOVA. All statistical tests were performed using SPSS software

(version 22, SPSS Inc., IBM, Armonk, New York, USA). The level of significance was set at $P < 0.05$. To assess the magnitudes of the differences, partial eta squared was calculated to report effect size (η^2, small = 0.01, moderate = 0.06, large = 0.14).

3. Results

3.1 Biceps femoris muscle activation at 90-30º and 30-0º epochs in previously injured and players without injury history

Bicep femoris muscle activation in the 90-30º epoch was significantly greater compared to the 30-0º epoch ($F = 20.92$, $P < 0.001$, $\eta^2 = 0.54$) (Figure 2). There was no significant effect of injury history on BF muscle activation ($F = 0.62$, $P = 0.44$, $\eta^2 = 0.03$) and no significant interaction of angle of knee flexion or injury history on BF activation ($F = 0.002$, $P = 0.96$, $\eta^2 > 0.01$).

3.2 Activation ratios at 90-30º and 30-0º epochs in previously injured and players without injury history

A significant interaction effect was observed between angle of knee flexion and injury history on BF/ST ($F = 6.83$, $P = 0.018$, $\eta^2 = 0.275$) and BF/GM activation ratios ($F = 11.12$, $P = 0.004$, $\eta^2 = 0.38$) (Figure 3). There were no significant differences between the injury-free players and those with a history of injury for the BF/ST ratio ($F = 2.09$, $P = 0.17$, $\eta^2 = 0.10$), and the BF/GM ratio at the 90-30º epoch ($F = 0.22$, $P = 0.65$, $\eta^2 = 0.01$). However, at 30-0º epoch, players with a history of injury had significantly greater activation ratios

for both the BF/ST, (F = 16.48, *P* = 0.001, η^2 = 0.48), and BF/GM (F = 6.16, *P* = 0.02, η^2 = 0.255) (Figure 4).

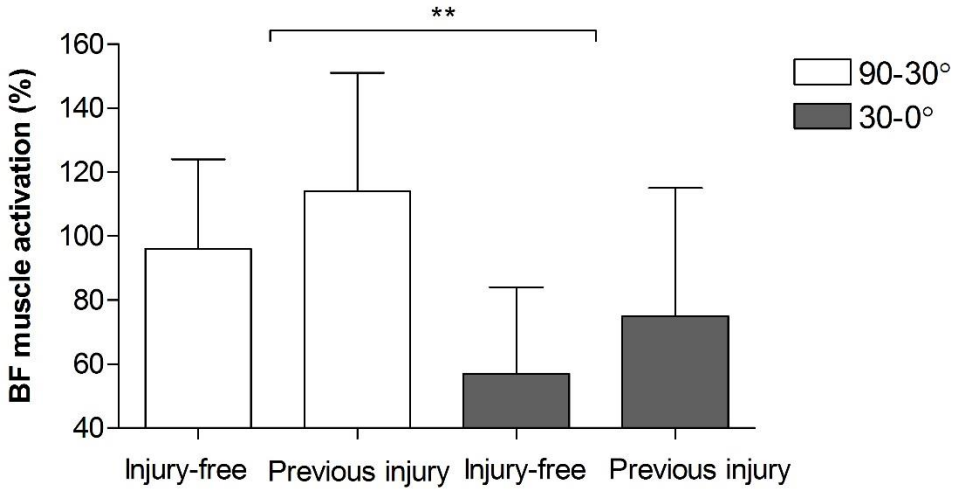

*Figure 2 BF activation at 90-30º and 30-0º of knee flexion. Values are means and SD. **P < 0.01*

*Figure 3 Figure 3. Interactions between injury history and knee flexion angle with respect to activation ratios. Values are means and SD. *P < 0.05*

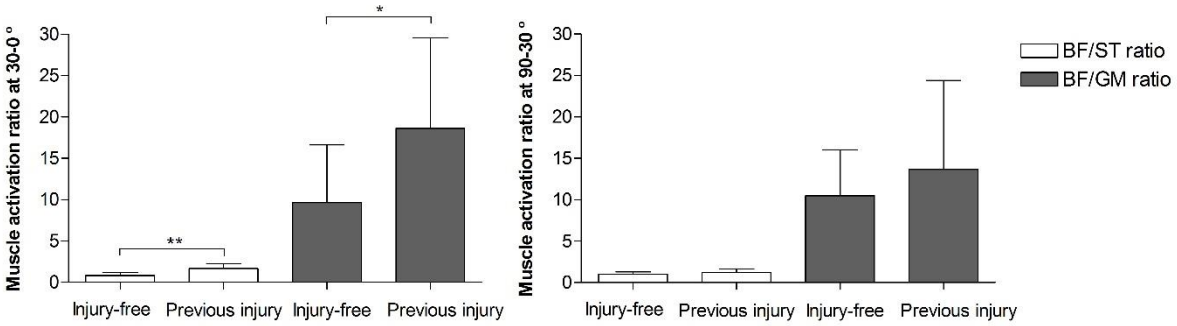

*Figure 4 Figure 4. Activation ratios at 30-0º (left) and 90-30º (right) of knee flexion. Values are means and SD. *P < 0.05, **P < 0.01*

4. Discussion

Previous hamstring strain injury results in changes to muscle morphology,[26, 27] but the effect of injury on neural function is less well reported.[14, 15] The purpose of this study was to determine if elite footballers with a history of hamstring strain injury, displayed differences in neural function in the BF, ST and GM, during the NHE, compared to those with no history of hamstring injury. The results show that 1) BF muscle activation was significantly higher when the knee was in a greater degree of knee flexion (90-30º) compared to more extended knee positions (30-0º), but this was not different between groups 2) BF/ST and BF/GM ratios at more extended knee positions (30-0º) were significantly greater in those with a previous history of hamstring strain injury, indicating a differing relative contribution of the BF muscle and its synergists during the NHE following hamstring strain injury.

Previous research has shown that the NHE is effective at reducing the chance of injury occurrence,[10,11] but is less effective at preventing recurrence in players with a history of hamstring strain injury.[11] This may be explained by previous findings reporting that

eccentric BF muscle activation is reduced at long muscle lengths, as seen during performance of a seated leg curl exercise by individuals with previous hamstring injury history.[14,15]

We postulated that the NHE may not be an effective exercise to prevent recurrence of hamstring injury, due to reduced levels of BF muscle activation as suggested by previous investigation.[14,15,16] However, our finding that BF muscle activation was not different between groups during the NHE does not support this concept. These results are consistent with a number of previous investigations assessing torque[15, 28] but different from Opar et al. (2013) who also assessed neural hamstring function.[14] In their study, an additional 85º of hip flexion was imposed using a seated leg curl, which may exacerbate activation deficits compared to compared to 0º hip flexion used in this study. Additionally, Daly et al. (2015) showed reduced activation in the BF during the terminal swing phase of sprinting, which may suggest the smaller amplitudes of elongation of the BF muscle during the NHE, compared to that imposed by the combined eccentric demands of hip flexion and knee extension of terminal swing,[17] may be insufficient to reveal the presence of neuromuscular inhibition. With respect to the difference in results of the present study and Bourne et al. (2016), population characteristics (recreationally active compared to elite footballers) and training intervention (six repetitions compared to six sets of ten repetitions) suggest the effects of neuromuscular inhibition may also be sensitive to the presence of fatigue.[16] Additionally, the previously mentioned study lacked a control group and muscle activity was not measured acutely but was inferred from imaging performed after the training protocol.[16] The findings of the present study therefore raise important

questions about the efficacy of the NHE to detect acute activation deficits of the BF muscle in elite footballers.

Despite no differences in BF activation between groups, higher levels of BF muscle activation were found at the more flexed (90-30º epoch) compared to the 30-0º epoch (Figure 2). These results agree with Iga et al. (2012) and Monajati et al. (2017) who demonstrated maximal muscle activity as occurring between 90-30º and 60-40º of knee flexion, respectively. [29,30] Our findings further support the effectiveness of the NHE to elicit high levels of muscle activation (96-114%) during the exercise's first 60º of knee excursion, which falls to and moderate levels of activation (57-75%) during the terminal 30º at long muscle lengths.[31]

Previous research suggests that in the presence of reduced BF activation following injury, changes in the relative contribution of other muscle synergists may represent a compensatory mechanism against neuromuscular inhibition.[17] Our findings are consistent with the presence of altered relative contribution of muscle synergists post-injury however, as no previous investigation has considered the activation of both the BF and the GM muscles during the NHE, direct comparison with other studies is not possible. During the terminal swing phase of sprinting, Daly et al. (2015) found that previously injured elite level field sport players had greater magnitudes of GM activity accompanying reduced BF activity.[17] In comparison to the NHE, this phase of the sprint cycle requires the BF to perform negative work at longer muscle lengths, offering partial explanation for the difference in results. If reduced BF activity is consistently accompanied by an increase

in GM activation, greater amplitudes of hamstring muscle length than imposed during the NHE may be required for this to be observed.

Within the present study, the BF/GM activation ratios suggest a lower contribution from the GM for the previously injured players during the exercise's terminal 30º (Figure 4). It would then appear reduced GM activation, a recognised risk factor for hamstring strain injury within footballers[19] is detectable during the NHE. Additionally, at this more extended knee position during the NHE, authors suggest the BF to be primarily resisting an anterior pelvic tilt (relative hip flexion) moment as opposed to knee extension,[30] and therefore acting as a synergist to the hip extensors including the GM.[32] The activation ratios reported for the previously injured players may represent a greater neuromuscular demand placed upon the BF to attenuate the anterior pelvic tilt moment. Questions therefore arise as to whether this pattern of activation may have been detectable during the NHE prior to injury, highlighting a limitation of this cross-sectional study.

Our results also showed a bias towards greater BF activation at more extended knee joint positions in those with previous hamstring strain injury (Figure 4). In non-elite sport populations, the NHE is reported to elicit a BF/ST activation ratio of 0.8 (SD 0.1), which is consistent with the 0.8 (SD 0.4) ratio found in this study for the injury free players[2]. Interestingly, a much greater ratio of 1.68 (SD 0.55) was observed for the players with a history of hamstring strain injury, suggesting a shift towards greater BF activation during lengthening[20] and illustrative of a fall in ST contribution. Therefore, with respect to purpose 2 of the study, the presence of an injury history appears to reduce the relative

contributions of both the GM and the ST compared to the BF during the NHE. This is an important finding as it may alter the training related adaptations the NHE provokes for previously injured players compared to those without injury history. Authors suggest the NHE may confer its protective effects through this greater emphasis on the ST, identified as the primary mediators of the demands of terminal swing.[33] Therefore, a reduction in ST activation, may limit the effectiveness of the NHE as an injury reduction intervention. These findings do supply support for literature championing a more holistic approach to hamstring injury reduction[34,35] through the targeting of a range of synergistic muscles, with a range of exercises during the rehabilitation process rather than just focussing on the most commonly injured muscles. It is also important to identify that although all previously injured players had experienced a hamstring strain injury in the 12 months preceding data collection, there cannot be absolute clarity on which hamstring muscle was affected. Although much less common, players may have experienced strains of the ST as opposed to the BF, offering a contrasting explanation to these results. Alternatively, the reductions observed in BF synergist's activation may have been present prior to injury, leading to a greater neuromuscular demand upon this muscle, apparent as the increased activation still detectable in the post-injury state.

Taken together, the results show that neuromuscular inhibition of the BF was not detectable during the NHE at either the 90-30º or 30-0º epochs of knee flexion in elite level footballers with previous hamstring strain injury. This finding may suggest that the NHE is not able to detect the purported re-injury risk factor for an elite football population. In the absence of detectable neuromuscular inhibition, the differences in both BF/ST and

BF/GM activation ratios at more extended knee joint positions identify the complex interactions between the hamstrings and their synergists following injury. The ratios suggest the BF may be exposed to greater neuromuscular demand following injury and the protective effects of the NHE hypothesised to be conferred through the ST bias, may not be elicited in the post injury state. The study also shows for the first time that reduced GM activity, recognised as a risk factor for injury during sprinting, appears to be present in previously injured elite level footballers and is detectable during the NHE. The study fails to assess other synergists at the hip and knee, which may also affect the activation deficits of the ST and the GM for previously injured players.

5. Conclusion

To conclude, the NHE did not detect muscle activation deficits of the BF muscle associated with the injury recurrence risk factor of neuromuscular inhibition. Yet, differences in activation ratios identified at more extended knee joint positions within previously injured elite-level footballers suggest activity of this commonly injured muscle's synergists are reduced. These knee joint angle specific reductions may potentially impede the NHE's protective effects but also highlight the need to consider altered synergistic interaction both within and external to the hamstrings following injury. The highlighted synergistic muscles may require specific intervention strategies during the return to play process, which suggests a divergent approach is required when seeking to reduce incidence of injury recurrence compared to initial injury events.

Declarations of interest
None

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