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

3
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29 **Abstract**

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

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

44 *Findings:* Biceps femoris activation was significantly higher at 90-30° of knee flexion
45 compared to 30-0° ($P < 0.001$) but did not differ between the groups. In players with a
46 history of injury, muscle activation ratios for the biceps femoris/semitendinosus ($P =$
47 0.001) and biceps femoris/gluteus maximus ($P = 0.023$) were significantly greater at 30-
48 0° of knee flexion than in the control group.

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

52 Keywords: *Hamstring injury recurrence, activation ratios*

53

54 **1. Introduction**

55 Hamstring strain injury is reportedly high within professional (soccer) football¹ despite
56 extensive investigation seeking to address the incidence and recurrence of injury.^{2,3,4}
57 Although working synergistically at the hip and knee, the individual hamstring muscles
58 differ not only in architecture and morphology,⁵ but also in their susceptibility to injury.
59 The majority of hamstring strain injuries may primarily occur during the terminal swing
60 phase of sprinting⁶ where peak activation of biceps femoris (BF) muscle and peak muscle
61 elongation occur synchronously to decelerate the knee and hip.⁷ These high activation
62 levels and rapid lengthening demands may partially explain why the BF muscle is more
63 susceptible to injury compared to the other hamstring muscles.¹

64

65 Strategies to reduce hamstring strain injury have been primarily aimed at matching the
66 lengthening and loading characteristics of the swing phase in sprinting to enhance knee
67 flexor force production during eccentric contractions.^{8,9} One such strategy associated with
68 successfully reducing hamstring strain injury occurrence in football is the Nordic
69 Hamstring Exercise (NHE).^{10,11} Petersen et al. (2011) reported the NHE to be an effective
70 strategy to reduce initial hamstring injury in football players.¹¹ However, in players with a
71 previous hamstring strain injury, the NHE's protective effect proved less successful in
72 preventing subsequent injury. One explanation for this difference may be neuromuscular
73 inhibition following an initial hamstring strain injury¹² whereby reductions in muscle
74 activation occur during eccentric contractions.^{13,14,15} For example, acute reductions in
75 eccentric muscle activation were present in the BF muscle during the final 30° prior to full
76 knee extension of a seated leg curl exercise, in participants who had previously had a

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

91

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

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

113

114 **2. Methods**

115 *2.1 Participants*

116 Twenty (mean age 18.7 y SD 1.08 y; mean stature, 1.82 m SD 0.07 m; mean body mass
117 76.4 kg SD 7.89 kg; elite youth (academy/U23 squad) male, outfield footballers, regularly
118 exposed to the NHE, were recruited to participate. Participants were currently healthy
119 (clear health questionnaire), available for selection, and absent of anterior cruciate
120 ligament reconstruction. Based on club physician's data, 10 players (age 18.9 y SD 1.3
121 y; stature 1.83 m SD 0.07 m; body mass 77.4 kg SD 6.8 kg) met inclusion criteria
122 (experiencing hamstring strain injury within the last 12 months leading to absence from

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

130

131 *2.2 Experimental setup*

132 The study followed a cross sectional design. Participants from both groups performed six
133 repetitions of the NHE with minimal periods of rest between each descent. To standardise
134 velocity of movement, participants were instructed to attempt to execute each repetition
135 of the NHE with a constant knee extension velocity performed to a strictly monitored six
136 second count.²¹ During each repetition, joint position and muscle activity of the BF, GM
137 and ST muscles were synchronously recorded. To prepare the skin for electromyography
138 (EMG), hair and skin cells were removed by shaving, abrading and wiping the skin with
139 alcohol. Two bipolar surface electrodes (DE- 2.3 MA; Delsys Inc., Boston, MA, USA)
140 were placed 10 mm apart on the muscle belly of the BF, the GM and the ST in accordance
141 with SENIAM guidelines²² of the previously injured limb and the corresponding limb of the
142 matched pair individual. Sensors were secured with tape to minimise motion artefact. A
143 ground electrode (20 mm contact diameter) was fixed to the olecranon process of the
144 right arm. A single axis electro-goniometer (S700; Measureand Inc., Fredericton, NB,
145 Canada) was secured to each participant's right knee during standing (0° flexion) ensuring

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

153

154 *2.3 Procedures*

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

162

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

168

169 *2.4 Data Processing and Analysis*

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

177
178 Activation values for all muscles of interest were calculated at two epochs of knee angle
179 excursion: 90-30° of knee flexion and 30-0° of knee flexion during the descent phase of
180 the NHE. Initiation and termination of each repetition were determined from threshold
181 values, set as two standard deviations above baseline. For initiation, baseline muscle
182 activity at 90° was averaged to derive the threshold value, and for termination, an average
183 of peak activation at approximately 0° determined baseline (termination) (Figure 1). Peak
184 EMG values were calculated for each repetition at 90-30° and 30-0° using custom written
185 analysis software (R, Version 3.2.1, The R Foundation for Statistical Computing Platform,
186 Vienna, Austria). Peak normalised muscle activations were combined to derive the
187 activation ratios at both 90-30° and 30-0° epochs: BF/ST and BF/GM. Ratios greater than
188 1.0 indicated a greater contribution from BF compared to the ST and GM, respectively.

189
190
191 Insert Figure 1

191 *2.5 Statistical Analysis*

192 To match individuals each player's BMI was calculated and expressed as an age-specific
193 BMI Z-score²⁵ (Cole, Freeman, & Preece, 1995) and compared between groups using an
194 independent t-test. To address purpose 1, a mixed 2 x 2 ANOVA was performed to assess
195 differences in BF muscle activation at both 90-30° and 30-0° epochs between injury free
196 players and those with a history of previous injury. To address purpose 2, a 2 x 3 mixed
197 design ANOVA was used to assess differences in BF/ST and BF/GM ratios at 90-30° and
198 30-0° epochs between injury free players and those with a history of previous injury. In
199 case of significance, post hoc tests were performed to determine the separate effects of
200 injury history and angle of knee flexion on BF activation and BF/ST and BF/GM ratios,
201 through the use of MANOVA. All statistical tests were performed using SPSS software
202 (version 22, SPSS Inc., IBM, Armonk, New York, USA). The level of significance was set
203 at $P < 0.05$. To assess the magnitudes of the differences, partial eta squared was
204 calculated to report effect size (η^2 , small = 0.01, moderate = 0.06, large = 0.14).

205

206 **3. Results**

207 *3.1 Biceps femoris muscle activation at 90-30° and 30-0° epochs in previously injured* 208 *and players without injury history*

209

210 Bicep femoris muscle activation in the 90-30° epoch was significantly greater compared
211 to the 30-0° epoch ($F = 20.92$, $P < 0.001$, $\eta^2 = 0.54$) (Figure 2). There was no significant
212 effect of injury history on BF muscle activation ($F = 0.62$, $P = 0.44$, $\eta^2 = 0.03$) and no
213 significant interaction of angle of knee flexion or injury history on BF activation ($F = 0.002$,
214 $P = 0.96$, $\eta^2 > 0.01$).

215

216 *3.2 Activation ratios at 90-30° and 30-0° epochs in previously injured and players without*
217 *injury history*

218

219 A significant interaction effect was observed between angle of knee flexion and injury
220 history on BF/ST ($F = 6.83$, $P = 0.018$, $\eta^2 = 0.275$) and BF/GM activation ratios ($F = 11.12$,
221 $P = 0.004$, $\eta^2 = 0.38$) (Figure 3). There were no significant differences between the injury-
222 free players and those with a history of injury for the BF/ST ratio ($F = 2.09$, $P = 0.17$, $\eta^2 =$
223 0.10), and the BF/GM ratio at the 90-30° epoch ($F = 0.22$, $P = 0.65$, $\eta^2 = 0.01$). However,
224 at 30-0° epoch, players with a history of injury had significantly greater activation ratios
225 for both the BF/ST, ($F = 16.48$, $P = 0.001$, $\eta^2 = 0.48$), and BF/GM ($F = 6.16$, $P = 0.02$, η^2
226 $= 0.255$) (Figure 4).

227 Insert Figure 2

228 Insert Figure 3

229 Insert Figure 4.

230

231 **4. Discussion**

232 Previous hamstring strain injury results in changes to muscle morphology,^{26, 27} but the
233 effect of injury on neural function is less well reported.^{14, 15} The purpose of this study was
234 to determine if elite footballers with a history of hamstring strain injury, displayed
235 differences in neural function in the BF, ST and GM, during the NHE, compared to those
236 with no history of hamstring injury. The results show that 1) BF muscle activation was
237 significantly higher when the knee was in a greater degree of knee flexion (90-30°)
238 compared to more extended knee positions (30-0°), but this was not different between

239 groups 2) BF/ST and BF/GM ratios at more extended knee positions (30-0°) were
240 significantly greater in those with a previous history of hamstring strain injury, indicating
241 a differing relative contribution of the BF muscle and its synergists during the NHE
242 following hamstring strain injury.

243

244 Previous research has shown that the NHE is effective at reducing the chance of injury
245 occurrence,^{10,11} but is less effective at preventing recurrence in players with a history of
246 hamstring strain injury.¹¹ This may be explained by previous findings reporting that
247 eccentric BF muscle activation is reduced at long muscle lengths, as seen during
248 performance of a seated leg curl exercise by individuals with previous hamstring injury
249 history.^{14,15}

250

251 We postulated that the NHE may not be an effective exercise to prevent recurrence of
252 hamstring injury, due to reduced levels of BF muscle activation as suggested by previous
253 investigation.^{14,15,16} However, our finding that BF muscle activation was not different
254 between groups during the NHE does not support this concept. These results are
255 consistent with a number of previous investigations assessing torque^{15, 28} but different
256 from Opar et al. (2013) who also assessed neural hamstring function.¹⁴ In their study, an
257 additional 85° of hip flexion was imposed using a seated leg curl, which may exacerbate
258 activation deficits compared to compared to 0° hip flexion used in this study. Additionally,
259 Daly et al. (2015) showed reduced activation in the BF during the terminal swing phase
260 of sprinting, which may suggest the smaller amplitudes of elongation of the BF muscle
261 during the NHE, compared to that imposed by the combined eccentric demands of hip

262 flexion and knee extension of terminal swing,¹⁷ may be insufficient to reveal the presence
263 of neuromuscular inhibition. With respect to the difference in results of the present study
264 and Bourne et al. (2016), population characteristics (recreationally active compared to
265 elite footballers) and training intervention (six repetitions compared to six sets of ten
266 repetitions) suggest the effects of neuromuscular inhibition may also be sensitive to the
267 presence of fatigue.¹⁶ Additionally, the previously mentioned study lacked a control group
268 and muscle activity was not measured acutely but was inferred from imaging performed
269 after the training protocol.¹⁶ The findings of the present study therefore raise important
270 questions about the efficacy of the NHE to detect acute activation deficits of the BF
271 muscle in elite footballers.

272
273 Despite no differences in BF activation between groups, higher levels of BF muscle
274 activation were found at the more flexed (90-30° epoch) compared to the 30-0° epoch
275 (Figure 2). These results agree with Iga et al. (2012) and Monajati et al. (2017) who
276 demonstrated maximal muscle activity as occurring between 90-30° and 60-40° of knee
277 flexion, respectively.^{29,30} Our findings further support the effectiveness of the NHE to elicit
278 high levels of muscle activation (96-114%) during the exercise's first 60° of knee
279 excursion, which falls to and moderate levels of activation (57-75%) during the terminal
280 30° at long muscle lengths.³¹

281
282 Previous research suggests that in the presence of reduced BF activation following injury,
283 changes in the relative contribution of other muscle synergists may represent a
284 compensatory mechanism against neuromuscular inhibition.¹⁷ Our findings are consistent

285 with the presence of altered relative contribution of muscle synergists post-injury
286 however, as no previous investigation has considered the activation of both the BF and
287 the GM muscles during the NHE, direct comparison with other studies is not possible.
288 During the terminal swing phase of sprinting, Daly et al. (2015) found that previously
289 injured elite level field sport players had greater magnitudes of GM activity accompanying
290 reduced BF activity.¹⁷ In comparison to the NHE, this phase of the sprint cycle requires
291 the BF to perform negative work at longer muscle lengths, offering partial explanation for
292 the difference in results. If reduced BF activity is consistently accompanied by an increase
293 in GM activation, greater amplitudes of hamstring muscle length than imposed during the
294 NHE may be required for this to be observed.

295

296 Within the present study, the BF/GM activation ratios suggest a lower contribution from
297 the GM for the previously injured players during the exercise's terminal 30° (Figure 4). It
298 would then appear reduced GM activation, a recognised risk factor for hamstring strain
299 injury within footballers¹⁹ is detectable during the NHE. Additionally, at this more extended
300 knee position during the NHE, authors suggest the BF to be primarily resisting an anterior
301 pelvic tilt (relative hip flexion) moment as opposed to knee extension,³⁰ and therefore
302 acting as a synergist to the hip extensors including the GM.³² The activation ratios
303 reported for the previously injured players may represent a greater neuromuscular
304 demand placed upon the BF to attenuate the anterior pelvic tilt moment. Questions
305 therefore arise as to whether this pattern of activation may have been detectable during
306 the NHE prior to injury, highlighting a limitation of this cross-sectional study.

307

308 Our results also showed a bias towards greater BF activation at more extended knee joint
309 positions in those with previous hamstring strain injury (Figure 4). In non-elite sport
310 populations, the NHE is reported to elicit a BF/ST activation ratio of 0.8 (SD 0.1), which
311 is consistent with the 0.8 (SD 0.4) ratio found in this study for the injury free players².
312 Interestingly, a much greater ratio of 1.68 (SD 0.55) was observed for the players with a
313 history of hamstring strain injury, suggesting a shift towards greater BF activation during
314 lengthening²⁰ and illustrative of a fall in ST contribution. Therefore, with respect to
315 purpose 2 of the study, the presence of an injury history appears to reduce the relative
316 contributions of both the GM and the ST compared to the BF during the NHE. This is an
317 important finding as it may alter the training related adaptations the NHE provokes for
318 previously injured players compared to those without injury history. Authors suggest the
319 NHE may confer its protective effects through this greater emphasis on the ST, identified
320 as the primary mediators of the demands of terminal swing.³³ Therefore, a reduction in
321 ST activation, may limit the effectiveness of the NHE as an injury reduction intervention.
322 These findings do supply support for literature championing a more holistic approach to
323 hamstring injury reduction^{34,35} through the targeting of a range of synergistic muscles,
324 with a range of exercises during the rehabilitation process rather than just focussing on
325 the most commonly injured muscles. It is also important to identify that although all
326 previously injured players had experienced a hamstring strain injury in the 12 months
327 preceding data collection, there cannot be absolute clarity on which hamstring muscle
328 was affected. Although much less common, players may have experienced strains of the
329 ST as opposed to the BF, offering a contrasting explanation to these results. Alternatively,
330 the reductions observed in BF synergist's activation may have been present prior to injury,

331 leading to a greater neuromuscular demand upon this muscle, apparent as the increased
332 activation still detectable in the post-injury state.

333

334 Taken together, the results show that neuromuscular inhibition of the BF was not
335 detectable during the NHE at either the 90-30° or 30-0° epochs of knee flexion in elite
336 level footballers with previous hamstring strain injury. This finding may suggest that the
337 NHE is not able to detect the purported re-injury risk factor for an elite football population.
338 In the absence of detectable neuromuscular inhibition, the differences in both BF/ST and
339 BF/GM activation ratios at more extended knee joint positions identify the complex
340 interactions between the hamstrings and their synergists following injury. The ratios
341 suggest the BF may be exposed to greater neuromuscular demand following injury and
342 the protective effects of the NHE hypothesised to be conferred through the ST bias, may
343 not be elicited in the post injury state. The study also shows for the first time that reduced
344 GM activity, recognised as a risk factor for injury during sprinting, appears to be present
345 in previously injured elite level footballers and is detectable during the NHE. The study
346 fails to assess other synergists at the hip and knee, which may also affect the activation
347 deficits of the ST and the GM for previously injured players.

348

349 **5. Conclusion**

350 To conclude, the NHE did not detect muscle activation deficits of the BF muscle
351 associated with the injury recurrence risk factor of neuromuscular inhibition. Yet,
352 differences in activation ratios identified at more extended knee joint positions within
353 previously injured elite-level footballers suggest activity of this commonly injured muscle's

354 synergists are reduced. These knee joint angle specific reductions may potentially impede
355 the NHE's protective effects but also highlight the need to consider altered synergistic
356 interaction both within and external to the hamstrings following injury. The highlighted
357 synergistic muscles may require specific intervention strategies during the return to play
358 process, which suggests a divergent approach is required when seeking to reduce
359 incidence of injury recurrence compared to initial injury events.

360

361 **Declarations of interest**

362 None

363

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366

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371

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