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Razor hamstring curl and Nordic hamstring exercise architectural adaptations: impact of exercise selection and intensity

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ABSTRACT

Objectives: To investigate knee flexor strength and biceps femoris long head (BFlh) architectural adaptations following two different Nordic hamstring exercise (NHE) interventions and one razor hamstring curl (RHC) intervention. **Methods:** Thirty recreationally-active males performed a total of 128 reps of $\text{NHE}_{\text{bodyweight}}$ (n=10), $\text{NHE}_{\text{weighted}}$ (n=10) or $\text{RHC}_{\text{weighted}}$ training (n=10) across six weeks. Following the intervention, participants avoided any eccentric training for four weeks (detraining period). Strength results during the NHE and RHC were recorded pre and post intervention, as well as following detraining. Architectural characteristics of the BFlh were assessed weekly throughout the intervention and detraining periods. **Results:** For the $\text{NHE}_{\text{weighted}}$ group, NHE strength increased (+81N, p=0.044, d=0.90) and BFlh fascicles lengthened (+1.57cm, p<0.001, d=1.41) after six weeks of training. After one week of detraining, BFlh fascicle lengths shortened, with the largest reductions seen in the $\text{NHE}_{\text{weighted}}$ group (-0.96cm, p=0.021, d=-0.90). Comparatively, BFlh fascicle length and NHE strength responses were moderate in the $\text{NHE}_{\text{bodyweight}}$ group and negligible in the $\text{RHC}_{\text{weighted}}$ group. The greatest RHC strength changes (+82N, p=0.038, d=1.15) were seen in the $\text{RHC}_{\text{weighted}}$ group. **Conclusions:** $\text{NHE}_{\text{weighted}}$ interventions induce large BFlh fascicle lengthening responses and these adaptations decay after just one week of detraining. $\text{NHE}_{\text{bodyweight}}$ training has a moderate impact on BFlh architecture while the $\text{RHC}_{\text{weighted}}$ group has the least. Weighted NHE and RHC training promoted exercise-specific increases in strength. These findings suggest that exercise selection and intensity should be considered when prescribing exercises aiming to increase eccentric strength and BFlh fascicle length.

KEY WORDS: fascicle length; eccentric training; muscle architecture; ultrasound.

INTRODUCTION

Hamstring strain injuries (HSIs) are prevalent across running-based sports.[1,2] The most commonly injured of these muscles is the biceps femoris long head (BFlh), which accounts for approximately 84% of all occurrences.[3] HSIs significantly burden athletes and their associated organisations, costing clubs on average USD $191,614 in the 2012 Australian Football League season.[4] However, despite significant research efforts, HSI rates continue to increase.[5]

There are a range of factors that increase the likelihood of an HSI occurring. Non-modifiable factors, including a history of HSI[6] and increasing age[7], have been proposed previously. However, modifiable factors, which have the potential to be altered through targeted interventions, are arguably of most interest to researchers and practitioners. These modifiable factors include, but are not limited to, eccentric knee flexor weakness[8] and short BFlh fascicles.[9] In elite Australian football, athletes with low eccentric knee flexor strength (<256N) at the start of pre-season were 2.7 times more likely to suffer a HSI in the subsequent season compared to their stronger counterparts.[8] Further, elite soccer players with short BFlh fascicles (<10.56cm) were 4.1 times more likely to suffer a HSI compared to those with longer fascicles.[9] Consequently, interventions which promote favourable adaptations in these variables may have implications for mitigating the risk of HSIs.[10]

Interventions employing the Nordic hamstring exercise (NHE) have resulted in significant increases in eccentric knee flexor strength and BFlh fascicle lengths, when supramaximal intensity of the exercise is ensured with the addition of extra weight beyond bodyweight.[11,12] To compensate for the lower training intensity, bodyweight NHE interventions, the most common prescription of this exercise, are typically undertaken with a high training volume (up to 30 reps per sessions/90 reps per week).[13] However, it is unknown if the addition of weight during lower volume NHE interventions promotes greater increases in knee flexor strength and BFlh fascicle length compared to bodyweight NHE interventions alone. Furthermore, following NHE training interventions, exposure to subsequent periods of detraining lasting two and four weeks can result in BFlh fascicles returning to baseline lengths.[11] However, the weekly alterations of BFlh fascicle length during a period of detraining following an NHE intervention remain unknown.

Despite the success of the NHE in reducing HSI risk[14,15] and promoting favourable adaptations,[11,12] protocols implementing this exercise still exhibit poor compliance rates in elite European soccer.[16] As such, other alternatives, including the razor hamstring curl (RHC), have been investigated.[17] This exercise has risen in popularity within the strength and conditioning community. Yet to date, its efficacy and impact on hamstring adaptations that follow a period of training are unestablished. Therefore, it is of interest to understand what adaptations occur after a period of training utilising the RHC.

The primary purpose of this study is to investigate the training-induced adaptations to BFlh architecture and knee flexor strength following six weeks of NHE training with ($\mathrm{NHE}_{\mathrm{weighted}}$) or without additional weight ($\mathrm{NHE}_{\mathrm{bodyweight}}$). Secondary to this, we aim to explore the same adaptations following $\mathrm{NHE}_{\mathrm{weighted}}$ or RHC training with additional weight ($\mathrm{RHC}_{\mathrm{weighted}}$). Finally, we aimed to determine the impact of four weeks of detraining on any training-induced adaptations to BFlh architecture or knee flexor strength across the three groups. It is hypothesised that 1) only $\mathrm{NHE}_{\mathrm{weighted}}$ training will result in lengthening of BFlh fascicles, 2) only $\mathrm{NHE}_{\mathrm{weighted}}$ and $\mathrm{RHC}_{\mathrm{weighted}}$ training will result in increases in knee flexor strength and 3) training-induced BFlh fascicle length adaptations will only be impacted by a subsequent detraining period in those who completed $\mathrm{NHE}_{\mathrm{weighted}}$ training.

METHODS

Thirty recreationally-active males (age, 24±4years; stature, 181±6cm; body mass, 78±11kg) were recruited for this randomised, stratified intervention trial. Participants were excluded if they had any history of injury to the lower limbs (including the hamstrings), wrist or back in the past 18 months. Prior to commencement, participants provided written informed consent. This study was approved by the Australian Catholic University Human Research Ethics Committee (Approval number: 2016–220E) and all testing and training was performed at the Australian Catholic University, Fitzroy, Victoria, Australia.

Participants undertook an initial familiarisation session which included having their baseline BFlh architecture assessed, as well as having their testing and training weights during the NHE or RHC determined. Following their familiarisation session (median = 6, range = 4 to 10 days), participants underwent pre intervention testing of their maximal knee flexor strength during the NHE and RHC. Participants were grouped according to their BFlh fascicle lengths (determined during familiarisation session), and stratified, at random, into one of three training groups; $\mathrm{NHE}_{\mathrm{bodyweight}}$ (n=10), $\mathrm{NHE}_{\mathrm{weighted}}$ (n=10) or $\mathrm{RHC}_{\mathrm{weighted}}$ (n=10). All participants then began their first session of the six-week training intervention after their strength assessment. There was no control group in this study as previous research across a similar timeframe saw no changes in control participants [12,18].

After their final training session (median = 7, range = 5 to 10 days), all participants underwent a post intervention assessment of their BFlh architecture and maximal knee flexor strength during the NHE and RHC. All participants then underwent a four-week detraining period, where they had their BFlh architecture assessed at weekly intervals. At the completion of this detraining period, participants underwent BFlh architecture and strength assessments. Across the course of the study, participants were instructed to maintain their habitual levels of physical activity and were specifically instructed to refrain from any other resistance training involving the hamstrings.

An illustration of the NHE and RHC can be found in Figure 1.

Prior to every training and testing session, participants rated their posterior thigh soreness with the aid of a visual analogue scale (0=no soreness, 10=unbearable soreness). Participants completed a six-week intervention performing $NHE_{bodyweight}$, $NHE_{weighted}$ or $RHC_{weighted}$ training. During the first two weeks, participants completed two sessions per week of four sets of six repetitions. For the final four weeks, participants performed a single session per week consisting of two sets of four repetitions. This protocol has previously been shown to result in favourable BFlh architectural adaptation during an $NHE_{weighted}$ intervention.[11] All training was performed on a custom-made device with published reliability on knee flexor force (Intra-class correlation (ICC) = 0.83 – 0.90; Typical error (TE) = 22 to 28N).[19] Training involved participants kneeling on a padded board with their ankles attached to straps above their lateral malleoli. The NHE involved participants starting in this kneeling position, lowering their torso with the hips remaining extended and arms across their chest. Participants were instructed to lower their torso to a prone position as slowly as possible. Once in this position, they were encouraged to continue resisting the fall until they touched the ground below with either the weight held ($NHE_{weighted}$/$RHC_{weighted}$) or their hands ($NHE_{bodyweight}$).

The RHC began with participants in a kneeling position, with their buttocks immediately superior to their ankles and their knees and hips fully flexed. Participants were then instructed to simultaneously extend at both the hip and knee, maintaining a consistent distance between their head and the mat, with the aim of reaching a near prone position (full hip and knee extension). Participants were asked to continue resisting the movement until their hand or the weight held touched the ground below. When either the NHE or RHC was performed with additional weight, participants were instructed to hold the weight centred over the xiphoid process.

If participants in the $NHE_{weighted}$ or $RHC_{weighted}$ groups were observed to have had enough control of the movement over the last 20° during the NHE, or were able to accomplish near full hip and knee extension during the RHC, additional weight was added in 2.5kg increments to ensure that the intensity of the exercise was still supramaximal (NHE; range: 5.0 to 27.5kg; RHC; range: 2.5 to 27.5kg). To ensure maximal effort during training, verbal encouragement was provided throughout. Finally, a two-minute rest period was allowed between each training set.

Knee flexor strength testing of the NHE and RHC was completed pre and post intervention, as well as post detraining on the same device.[19] Exercise order was also randomised to avoid testing order bias.

All participants completed one set of three repetitions of the NHE and RHC with bodyweight only, even if they were capable of completing weighted efforts. This allowed for comparisons for those participants who were unable to complete weighted efforts pre testing. Additional efforts of the NHE or RHC were performed with additional weight, only if participants could control the last 20° of the NHE or could reach full hip and knee extension during the RHC. The progression of additional weight continued until maximal force output plateaued, or if participants were unable to control the final stages of the exercise, akin to a one-repetition maximum test. Dominant and non-dominant limb force output (N) was recorded for all tests. Results were then collated into NHE and RHC strength, with measures being the peak force of each limb, irrespective of whether it was a bodyweight or weighted effort. The same testing protocol was applied during post intervention and post detraining assessments.[11]

Two-dimensional images of BFlh architecture were collected using B-mode ultrasonography (GE Healthcare Vivid-i, Wauwatosa; frequency, 12MHz; depth, 8cm; field of view, 14x47mm). Following five minutes of inactivity, and before any training or testing, participants laid prone with their hips and knees neutral and fully extended. The scanning site was the midway point between the ischial tuberosity and the popliteal crease, in line with the BFlh.[11,12,18] Once the scanning site was determined, the linear array probe (coated with conductive gel) was placed longitudinally to the muscle's axis. The probe was placed upon the skin with as minimal pressure as possible, as this may compromise measurement accuracy.[20]

Offline analyses (MicroDicom, Version 0.7.8, Bulgaria) of each image were completed to determine the architectural characteristics of interest, with the assessor blinded to participant identifiers. In line with previous research,[21] various points were digitised to estimate muscle thickness and aponeurosis angle. A fascicle of interest was determined, and the angle which it inserted into the intermediate aponeurosis was recorded as the pennation angle. As the entire fascicle was not visible in the probe's field of view, its length, reported in absolute terms (cm), was estimated through the following validated equation:[22]

$$FL = \sin(AA + 90°) \times MT \div \sin(180° - (AA + 180° - PA))$$

Where FL = fascicle length, MT = muscle thickness, AA = anatomical angle and PA = pennation angle

These methods have been used previously by our research group[9,11,18] and all scans were completed by the same researcher (R.G.T) with published reliability in fascicle length measures (ICC = 0.97 – 0.98; TE = 0.22 to 0.32cm).[23]

All statistical analyses were performed using JMP V.11.01 Pro Statistical Discovery Software (SAS Inc., Cary, North Carolina, USA). Normal distribution of the data was tested using Shapiro-Wilk's analyses. Repeated measures linear mixed models fitted with the restricted maximum likelihood (REML) method were used to assess changes in BFlh architecture (fascicle length, muscle thickness, pennation angle), knee flexor strength (NHE strength, RHC strength) and perceived soreness measurements across the duration of the study for each group ($NHE_{bodyweight}$, $NHE_{weighted}$, or $RHC_{weighted}$). For BFlh fascicle length, pennation angle and muscle thickness, the within-group variable was time (Day 0 [pre intervention], day 7, day 14, day 21, day 28, day 35, day 42 [post intervention], day 49, day 56, day 63, day 70 [post detraining]), with participant as the random factor. Similar analyses were completed to determine strength changes in each test (NHE strength, RHC strength), with the within-group variable being time (Day 0 [pre intervention], day 42 [post intervention], day 70 [post detraining]). As there were no significant differences between limbs for BFlh architecture and knee flexor strength ($p>0.05$), an average of the two limbs was used for all analyses. For perceived soreness, the within-group variable was also time (Day 0 [pre intervention], day 7, day 14, day 21, day 28, day 35, day 42 [post intervention], day 49, day 56, day 63, day 70 [post detraining]). Where significant main or interaction effects of architecture and strength variables were detected, post hoc t tests with Tukey's HSD were applied to determine where any differences occurred. Mean differences of all measurements were reported with their 95% confidence intervals (CI). Significance for all analyses was set at $p<0.05$. Where appropriate, Cohen's d effect sizes, classified as small ($d=0.20$), medium ($d=50$), and large ($d=0.80$), were also reported.[24]

Based on estimated fascicle length changes following the six-week intervention, G*Power version 3.1.9.2 was used *a-priori* to calculate sample size.[25] The effect size was derived from the most conservative effect size following a training intervention available in the literature.[18] This study reported a 16% increase in BFlh fascicle length following six weeks of training ($d=2.5$).[18] The effect size for the current study was conservatively determined as approximately half of this previously reported effect. Also, as a cross-reference to confirm these estimates, similar studies have utilised groups of 10[11,12,26] Therefore, a sample size of 9 per group, increased to 10 accounting for potential drop-outs, was determined as sufficient using the following inputs:

- Power ($1 – \beta$ err probability) = 0.80
- $\alpha = 0.05$
- Effect size = 1.2
- Anticipated drop-out rate (10%)

RESULTS

There were no significant differences (p>0.05) in participant age, height or weight between the $\text{NHE}_{\text{bodyweight}}$ (age, 24±4years; stature, 178±6cm; body mass, 77±12kg), $\text{NHE}_{\text{weighted}}$ (age, 24±4years; stature, 181±5cm; body mass, 78±11kg) or $\text{RHC}_{\text{weighted}}$ (age, 23±3years; stature, 183±6cm; body mass, 79±12kg) groups. The $\text{NHE}_{\text{weighted}}$ and $\text{RHC}_{\text{weighted}}$ groups completed 99% of all training sessions (71 of 72 sessions) respectively, whereas the $\text{NHE}_{\text{bodyweight}}$ group completed 97% of all training sessions (70 of 72 sessions).

A summary of the BFlh architectural adaptations following the training and detraining periods can be found in Figure 2 and Table 1.

A significant group x time interaction was observed for fascicle length (p<0.001). The greatest lengthening in fascicles was found when the pre- and post- intervention measures for $\text{NHE}_{\text{weighted}}$ (mean difference 1.57cm; 95% CI, 0.78 to 2.36cm, p<0.001, d=1.41) were compared. Changes were less for $\text{NHE}_{\text{bodyweight}}$ (p=0.087, d=0.72) with practically no change in fascicle length observed following the 6 weeks of $\text{RHC}_{\text{weighted}}$ training (p=0.995, d=0.00).

After one week of detraining, fascicle lengths shortened from post- intervention lengths in the $\text{NHE}_{\text{weighted}}$ group the most (0.96cm, 95% CI, -1.77 to -0.14cm, p=0.021, d=-0.86) followed by the $\text{NHE}_{\text{bodyweight}}$ to a lesser extent (p=0.136, d=0.63) and with a trivial change for the $\text{RHC}_{\text{weighted}}$ group (p=0.958, d=0.02). After two weeks of detraining, fascicle lengths in the $\text{NHE}_{\text{weighted}}$ (mean difference: -1.44cm; 95% CI, -2.28 to 0.60cm, p=0.001, d=-1.30) and $\text{NHE}_{\text{bodyweight}}$ (mean difference: -0.80cm; 95% CI, -1.47 to -0.14cm, p=0.019, d=-1.03) groups continued to shorten compared to the end of the intervention. Whereas the $\text{RHC}_{\text{weighted}}$ group saw no changes across the same period (mean difference: 0.02cm; 95% CI, -0.65 to 0.69cm, p=0.951, d=0.03). By the end of the four-week detraining period, BFlh fascicle lengths remained shortened in the $\text{NHE}_{\text{bodyweight}}$ (mean difference: -0.93cm; 95% CI, -1.58 to -0.28cm, p=0.005, d=-1.20) and $\text{NHE}_{\text{weighted}}$ groups (mean difference: -1.77cm; 95% CI, -2.56 to -0.97cm, p<0.001, d=-1.59) compared to post intervention. However, the $\text{RHC}_{\text{weighted}}$ group saw no change (mean difference: 0.28cm, 95% CI, -0.39 to 0.96cm, p=0.405, d=0.39).

A significant main effect for time was detected for BFlh pennation angle (p<0.001) however no significant interaction between group and time was found (p=0.532). Therefore, no Tukey’s HSD post hoc analyses were undertaken. Effect size comparisons showed that after the $\text{NHE}_{\text{weighted}}$ intervention, BFlh pennation angles decreased by 1.5° (d=1.09). However, there were no changes across this time-period in the $\text{NHE}_{\text{bodyweight}}$ (d=0.09) or $\text{RHC}_{\text{weighted}}$ (d=0.05) groups. After two weeks of detraining, there was an increase in BFlh pennation angle in the $\text{NHE}_{\text{weighted}}$ group when compared to post intervention values (d=0.97). There was also an increase in the $\text{NHE}_{\text{bodyweight}}$ group, yet to a lesser extent (d=0.70). Whereas the $\text{RHC}_{\text{weighted}}$ group saw no changes after two weeks of detraining (d=0.15). Pennation angles continued to increase in the NHE$_{\text{weighted}}$ group throughout detraining, with results at the end of the detraining being 1.7° greater than post intervention (d=1.22). A similar increase of 1.2° was found in the NHE$_{\text{bodyweight}}$ group (d=0.88) with the RHC$_{\text{weighted}}$ group remaining unchanged (d=0.03).

Muscle thickness remained stable in all groups over the study period (p=0.334 to 0.996, d=0.00 to 0.43).

A summary of the knee flexor strength adaptations following the training and detraining periods can be found in Figure 3 and Table 2.

A significant interaction of group x time was found for NHE strength (p=0.007). The NHE$_{\text{weighted}}$ training intervention increased NHE strength by 82N (95% CI, 2 to 161N; p=0.044, d=0.90), while the NHE$_{\text{bodyweight}}$ (mean difference: 67N; 95% CI, -23 to 158N, p=0.137, d=0.75) and RHC$_{\text{weighted}}$ groups (mean difference: 65N; 95% CI, -8 to 138N, p=0.080, d=0.72) saw smaller (non-significant) NHE strength changes. After the detraining period, the NHE strength training gains obtained were sustained and trivial to small changes were observed across the groups (p range=0.407 to 0.901, d range=0.05 to 0.30).

A significant interaction of group x time was found for RHC strength (p=0.048). Post hoc analyses showed an increase in peak razor strength after the RHC$_{\text{weighted}}$ training intervention (mean difference: 82N; 95% CI, 5 to 158N, p=0.038, d=1.15). Smaller RHC strength increases after the six-week intervention were found in the NHE$_{\text{bodyweight}}$ (mean difference: 23N; 95% CI, -66 to 112N, p=0.596, d=0.33) and NHE$_{\text{weighted}}$ (mean difference: 46N; 95% CI, -19 to 111N, p=0.157, d=0.65) groups. After the detraining period, the RHC strength gains obtained as a result of training were maintained in the RHC$_{\text{weighted}}$ group (p=0.066, d=1.01), but not in either of the NHE groups (p=0.712 to 0.965, d=0.02 to 0.48).

A summary of perceived posterior thigh soreness (0 - 10) values for each group can be found in figure 1D.

A significant main effect for perceived posterior thigh soreness was detected by time (p<0.001). Perceived soreness levels peaked after one week of training for all groups (mean difference: 3.0; 95% CI, 2.2 to 3.7, p<0.001, d=1.41 to 2.31) It remained elevated compared to baseline at week 2 (mean difference: 1.8; 95% CI, 1.0 to 2.5, p<0.001, d=0.73 to 1.47), returning to baseline levels at week three to six (p=0.064 to 1.00, d=0.06 to 0.67).

DISCUSSION

This study aimed to determine BFlh architectural and knee flexor strength responses to six weeks of NHE training with and without additional weight, as well as RHC training with added weight. This study also aimed to determine how these architectural and strength variables were impacted by a subsequent four-week detraining period. The novel findings of this study are: 1) $\text{NHE}_{\text{weighted}}$ interventions stimulate more BFlh fascicle lengthening than $\text{NHE}_{\text{bodyweight}}$ and $\text{RHC}_{\text{weighted}}$ interventions, 2) $\text{RHC}_{\text{weighted}}$ interventions promote increases specifically in RHC strength and 3) following one week of detraining, there was a reduction in BFlh fascicle length which was most pronounced in the $\text{NHE}_{\text{weighed}}$ group.

To the authors' knowledge, this is the first study to have investigated the impact of 1) both $\text{NHE}_{\text{weighted}}$ and $\text{NHE}_{\text{bodyweight}}$ training and 2) $\text{RHC}_{\text{weighted}}$ training on BFlh architectural and strength variables. One previous study has examined the architectural and strength impacts of NHE weighted training at a low and high volume,[11] while another has compared the architectural and strength adaptations that follow this form of training to weighted hip extensions.[12] Studies have also investigated the effects of bodyweight NHE training on BFlh architecture, with mixed results.[26,27] Further, to the authors' knowledge, this is the first study to have investigated the impact of detraining following an NHE intervention on BFlh architecture at weekly intervals. Previously, the shortest time-frame observed was two weeks.[11]

Fascicle lengthening and increases in eccentric strength have been highlighted as potential mechanisms responsible for the reduced HSI risk that results from eccentric training. In a cohort of elite Australian soccer players, those whose fascicle lengths were below 10.56cm saw their HSI risk increase four-fold.[9] Additionally, elite Australian footballers whose pre-season bodyweight NHE eccentric strength results were less than 256N were nearly three times more likely to sustain a HSI compared to their stronger counterparts.[8] Only participants in the NHE weighted group of our study saw their BFlh fascicles lengthen after the intervention (+16%), while both the $\text{NHE}_{\text{weighted}}$ and $\text{RHC}_{\text{weighted}}$ groups increased in NHE and RHC knee flexor strength by 18 and 17%, respectively. It should be noted that previous research infers that greater levels of NHE strength reduces HSI risk, however whether greater RHC strength confers the same benefits is not yet known.

In large randomised control trials, bodyweight NHE interventions have reduced HSI risk by 70% compared to control groups.[15] Despite this evidence however, HSI incidence rates continue to increase.[5] Potentially, insufficient compliance to NHE interventions could at least partially explain this continued rise in HSI incidence. For example, only 16 of a total of 150 club seasons (11%) were fully compliant to an evidence-based NHE program across 50 elite European soccer teams.[16] It is possible that NHE training volume may impact compliance rates, given that athletes are already heavily involved in significant amounts of other specified training.[15] High volume NHE interventions, with (up to 100 repetitions per week) and without (up to 90 repetitions per week) additional weight, have shown significant increases in BFlh fascicle length.[11,28] However, recent findings have shown that low volume (8 repetitions per week) weighted NHE interventions, that follow an initial two-week higher volume period (48 repetitions per week), are just as advantageous as high volume training in promoting BFlh fascicle lengthening.[11] In the current study, undergoing bodyweight NHE training with the same low volume, after an initial two-week higher volume period, resulted in no significant BFlh fascicle lengthening. However, with the addition of weight, BFlh fascicles significantly lengthened (Figure 2A and Table 1). This current evidence suggests that low volume NHE training, following an initial high volume period, can result in advantageous BFlh fascicle lengthening. However, the intensity of the NHE requires a high intensity (i.e additional weight beyond bodyweight) to induce these beneficial architectural adaptations using a low volume protocol.

This study also investigated BFlh architectural alterations following an $\mathrm{RHC}_{\mathrm{weighted}}$ intervention, with previous research into this exercise only outlining the surface electromyography (sEMG) profile during its shortening phase (starting in an extended position, flexing at the hip and knee joints to finish with both at 90°).[17] In the current study, $\mathrm{RHC}_{\mathrm{weighted}}$ training had the least impact of the three interventions, and there was no evidence of BFlh fascicle length change after six weeks of training, even when only the extension phase was performed (Figure 2A and Table 1). Potentially, the BFlh may not be preferentially recruited throughout the RHC given its bi-articular nature. Further to this, the opposing action that the BFlh possesses about the knee joint during the RHC may also negatively impact architectural adaptation. In supporting this, magnetic resonance imaging has revealed limited contributions from the BFlh during a squat, another movement where this muscle possesses an opposing action at the knee.[29] Consequently, in-series strain within the BFlh would be reduced during RHC training, impacting any stimulus for fascicle lengthening. However, it is possible that higher volumes of RHC training may be required to promote fascicle lengthening, and this should be explored in future investigations.

This study revealed that the positive responses of BFlh fascicle lengthening following $\mathrm{NHE}_{\mathrm{weighted}}$ interventions can rapidly (one week) decay when the stimulus is withdrawn (Figure 2A and Table 1). Additionally, this study showed that this decay continues from weeks two to four of detraining, in both the weighted and bodyweight NHE groups (Figure 2A and Table 1), which is in line with previous research investigating two weeks[11] and four weeks[11,28] of detraining after weighted and bodyweight NHE training. Importantly, these findings should help to guide prescription of NHE training interventions, highlighting the need to consider the acute reduction in fascicle length after only one week without a weighted NHE training stimulus.

The current study is the first to have investigated knee flexor strength following $\text{NHE}_{\text{bodyweight,}}$ $\text{NHE}_{\text{weighted}}$ and $\text{RHC}_{\text{weighted}}$ interventions. The addition of weight to NHE training resulted in an increase in NHE strength (Figure 3 and table 2), which is consistent with previous literature.[11,12] However, bodyweight NHE training resulted in no changes in NHE strength (Figure 3 and Table 2) with varied findings seen previously.[26,27] Additionally, there was exercise-specific strength adaptation after this study's intervention. Participants in the $\text{RHC}_{\text{weighted}}$ group increased in RHC strength but not NHE strength, while the reverse occurred for those in the $\text{NHE}_{\text{weighted}}$ group (Figure 3 and Table 2). These data suggest that both weighted NHE and RHC training regimes are capable of increasing knee flexor strength during their respective exercises.

There are limitations of this study which should be considered. Firstly, all participants were recreationally-active males so it is unknown whether these interventions would result in similar adaptations if the population were more highly trained athletes. Nevertheless, $\text{NHE}_{\text{bodyweight}}$ training interventions have seen significant reductions in HSI incidence in semi-professional athletes.[15] Secondly, the transducer field of view utilised in this study could not reveal a whole fascicle. Therefore, architectural analyses involved estimation of fascicle length via an equation.[22] However, this equation has been validated when compared against cadaveric measurements.[30] Thirdly, architectural assessment was only performed on the BFlh muscle and none of the other three hamstrings. However as the BFlh is the most commonly injured of the hamstrings, understanding the adaptations that occur within this muscle after a specific intervention may provide useful insight for injury prevention processes.[3] Lastly, ultrasonography operator reliability may have impacted results. However, the assessor (R.G.T) in the current study has proven reliability and has published extensively on the topic.[9,11,12]

CONCLUSION

Low volume NHE training, after an initial high volume period, requires the addition of weight to promote increases in BFlh fascicle length. However, these architectural adaptations are reversed after just one week of detraining. Additionally, extra weight is required to induce exercise-specific knee flexor strength adaptations after low volume NHE and RHC interventions. Finally, the RHC as a preventative exercise for HSI may have limitations, given it induces negligible BFlh architectural change or increases in NHE strength. Moreover, these findings have the potential to help guide HSI prevention and rehabilitation prescription by adding to the hamstring training adaptation evidence-base. However, future research is required to clarify if more volume is needed for RHC architectural adaptations to be observed.

PERSPECTIVE

The current study suggests that lower volume NHE training can result in favourable adaptations of both

BFlh architecture (notably fascicle length) and NHE strength but requires additional resistance to do so. As previous research has identified a relationship between these two factors and a reduction in HSI risk, these findings may guide the prescription of this exercise to fit in with the busy schedules often seen in elite sport. This study also found that the beneficial BFlh architectural adaptation that is accrued via NHE training is reversed after just one week of detraining. Therefore, a weekly prescription of this exercise in-season should be considered. Finally, this study found that weighted RHC training results in an increase in RHC strength. However, as there is no evidence to suggest that improving RHC strength may offset the risk of future HSI, it remains to be seen if the implementation of this exercise has its place in injury prevention practices.

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COMPETING INTERESTS

A co-author of this paper, David Opar, is listed as a co-inventor on a patent filed for a field test of eccentric knee flexor strength (PCT/AU2012/001041.2012), whilst also being a minority shareholder in a company (Vald Performance) that commercialises the device. David Opar is also the Chair of the Vald Performance Research Committee (a role which is unpaid). Morgan Williams is a member of the Vald Performance Research Committee. Christopher Pollard, Ryan Timmins and Matthew Bourne declare that they have no conflicts of interest related to this review. No other competing interests are declared.

CONTRIBUTORS

CWP was primarily responsible for recruitment, statistical analysis, data collection and manuscript writing. RGT performed all data collection and analyses of architecture. DAO, MDW, MNB, and RGT were responsible for the study’s design. MDW, RGT, and DAO were involved in statistical analysis. DAO, RGT, MNB, and MDW assisted in manuscript writing.

ETHICAL APPROVAL

This study was approved by the Australian Catholic University Human Research Ethics Committee (Approval number: 2016–220E) and all testing and training was performed at the Australian Catholic University, Fitzroy, Victoria, Australia.

DATA SHARING STATEMENT

The authors of this study are happy to provide de-identified data to other authors upon request.

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Figure 1: A) The Nordic hamstring exercise and B) the razor hamstring curl, progressed from left to right. Bodyweight efforts were performed with no weight held.

Figure 2. Absolute change in biceps femoris long head A) fascicle length, B) pennation angle, C) muscle thickness and D) posterior thigh perceived soreness (0 = no soreness, 10 = unbearable soreness) after Nordic hamstring exercise interventions with ($NHE_{weighted}$) and without ($NHE_{bodyweight}$) additional weight, as well as weighted razor hamstring curl ($RHC_{weighted}$) interventions. All architecture values are presented as an average of dominant and non-dominant limbs (mean ± SD). Perceived soreness values are presented as mean ± SD with week 1 and 2 values utilising the highest score of the two sessions for the week. All architecture values are compared to pre (Day 0) and post (Day 42) intervention. Perceived soreness values are compared to Day 0 (Pre intervention). #=p<0.05 vs. Day 0 (Pre intervention), $^{\#\#}$=p<0.001 vs. Day 0 (Pre intervention), *=p<0.05 vs. Day 42 (Post intervention), **= p<0.001 vs. Day 42 (Post intervention).

Figure 3. Change in peak A) Nordic hamstring exercise (NHE) strength and B) razor hamstring curl (RHC) strength (N) after Nordic hamstring exercise interventions with ($NHE_{weighted}$) and without ($NHE_{bodyweight}$) additional weight, as well as weighted razor hamstring curl ($RHC_{weighted}$) interventions. All values are in comparison to Day 0 (pre intervention) and are presented as mean ± SD. NHE = Nordic hamstring exercise, RHC = razor hamstring curl. $^{\#}$=p<0.05 vs. Day 0 (pre intervention).

Table 1. The effect of Nordic hamstring exercise (NHE) interventions performed with bodyweight ($NHE_{bodyweight}$) and additional weight ($NHE_{weighted}$), as well as weighted razor hamstring curl ($RHC_{weighted}$) interventions, on biceps femoris long head architectural characteristics. All data presented as mean ± SD of dominant and non-dominant limb. Effect sizes (*d*) are presented with comparisons to pre intervention (Day 0) values.

#=p<0.05 vs. Day 0 (Pre intervention), $^{\#\#}$=p<0.001 vs. Day 0 (Pre intervention), *=p<0.05 vs. Day 42 (Post intervention), **= p<0.001 vs. Day 42 (Post intervention).

Table 2. The effect of Nordic hamstring interventions performed with bodyweight ($NHE_{bodyweight}$) and additional weight ($NHE_{weighted}$), as well as weighted razor hamstring curl interventions ($RHC_{weighted}$), on measures of knee flexor force (N). All data presented as mean ± SD of dominant and non-dominant limb. Effect sizes (*d*) are presented with comparisons to Day 0 (Pre intervention). $^{\#}=p<0.05$ vs. Day 0 (Pre intervention).