EFFECT OF FLEXIBILITY TRAINING ON SYMPTOMS OF EXERCISE-INDUCED MUSCLE DAMAGE: A PRELIMINARY STUDY

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Exercise-induced muscle damage (EIMD) is characterized by loss of strength, increase in muscle stiffness, swelling, and soreness. The aim of this study was to assess the effects of flexibility training of the hamstring muscle group on symptoms of EIMD. Fourteen males (mean ± standard deviation: age = 20.6 ± 0.8 years; mass = 77.3 ± 10.4 kg; height = 1.77 ± 0.05 m) were randomly assigned to a 5-week flexibility (proprioceptive neuromuscular facilitation [PNF]) training or control group. Pre- and post-measures of peak static muscle force at knee joint angles representing decreasing muscle lengths of the hamstrings (25 deg, 30 deg, 40 deg, 80 deg) and flexibility were taken. Flexibility increased significantly following PNF flexibility training (pre-test = 19.4 ± 6.2 cm, post-test = 26.6 ± 6.9 cm), whereas it stayed constant in the control group (pre-test = 16.0 ± 9.3 cm, post-test = 16.7 ± 7.6 cm). Both groups underwent a damaging exercise protocol incorporating six sets of 10 isokinetic eccentric contractions of the hamstrings. Measurements of isometric strength, flexibility, and perceived soreness were recorded prior to and 1, 24, 48 and 72 hours after damage. There was a tendency for the PNF group to recover from strength loss earlier at longer muscle lengths (25 deg, $p = 0.06$; 30 deg, $p = 0.05$), but not at shorter muscle lengths. There was no evidence of a protective effect of PNF training on flexibility and soreness. In conclusion, an increase in flexibility of the hamstring muscle group led to some protection from strength loss at long muscle lengths following EIMD.

Keywords: hamstrings, muscle length, proprioceptive neuromuscular facilitation, strength retention

Introduction

Exercise-induced muscle damage (EIMD) is characterized by loss of isometric and dynamic strength (Clarkson et al. 1992; Cleak & Eston 1992), increase in muscle stiffness (Clarkson et al. 1992), swelling (Clarkson et al. 1992), muscle soreness (Newham et al. 1998; Jones et al. 1987), and decrements in rapid, dynamic muscle function, such as jumping, rebounding, and sprinting activities (Twist & Eston 2005; Bryne et al. 2004; Byrne & Eston 2002). McHugh et al. (1999) observed that greater hamstring muscle stiffness was associated with more severe symptoms of damage. Passive hamstring muscle stiffness is correlated with flexibility assessed by the straight-leg-raise range of motion (McHugh et al. 1998) and the sit-and-reach test (Magnusson et al. 1997). Therefore, flexibility and stiffness of muscle could be important factors in EIMD.

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Eccentric contractions lead to greater symptoms of EIMD when performed at long muscle lengths (Bryne et al. 2001; Rowlands et al. 2001; Child et al. 1998; Saxton & Donnelly 1996). When the muscle is functioning at relatively long lengths, corresponding to the plateau or descending limb of the muscle force–joint angle curve, Morgan's (1990) popping sarcomere theory predicts a greater disruption to sarcomeres. The theory proposes that during eccentric contractions, there are always some sarcomeres on the descending limb of the length–tension curve due to the non-uniformity of sarcomere length with eccentric contraction. When exercising at longer compared to shorter muscle lengths, a greater number of sarcomeres would be operating on their descending limb, leading to an increase in the number of sarcomeres that lengthen to the point of no myofilament overlap. Subsequent failure to re-interdigitate leads to disrupted sarcomeres and EIMD (Talbot & Morgan 1996).

There is evidence that the optimal angle for force production in more compliant and flexible muscle occurs at a longer muscle length than in the relatively stiff muscle. Two separate studies have observed that the passive flexibility of the quadriceps muscle is significantly greater in boys compared to men and that the muscle force–joint angle curve of the knee extensors in boys is shifted slightly to the right of the corresponding adult curve (Marginson et al. 2005; Marginson & Eston 2001). The greater flexibility and compliance of children's muscle may explain why peak muscle force occurs at a higher joint angle (longer muscle length) in children than adults (Marginson et al. 2005; Marginson & Eston 2001) and why boys suffer less severe symptoms of EIMD than men (Marginson et al. 2005).

Increased flexibility may be related to a greater number of sarcomeres in series within the muscle (Friden 1984). A greater number of sarcomeres in series would decrease the length of individual sarcomeres at any given muscle length, leading to the optimal angle for force production occurring at longer muscle lengths. Therefore, at any given muscle length, less severe symptoms of EIMD should occur because fewer sarcomeres would be operating on their descending limb.

In addition, McHugh et al. (1999) hypothesized that more severe symptoms of EIMD in participants with less compliant muscle may be explained by greater tendon–aponeurosis stiffness. The stiffness of the tendon–aponeurosis complex would prevent it from absorbing strain imposed by active lengthening, thereby transferring the strain to the muscle fibers and resulting in myofibrillar strain and greater EIMD.

McHugh et al. (1999) demonstrated that people with greater passive muscle stiffness suffered more severe symptoms of EIMD. However, this was a cross-sectional study. It has not been demonstrated whether actually increasing flexibility results in decreased susceptibility to symptoms of EIMD. Proprioceptive neuromuscular facilitation (PNF) training is an effective method of increasing chronic flexibility (Rowlands et al. 2003). Therefore, the aim of this study was to assess the effect of PNF flexibility training of the hamstring muscle group on the symptoms of EIMD following a damaging eccentric protocol. It was hypothesized that participants undergoing PNF training would experience less severe symptoms of EIMD following a damaging eccentric exercise protocol of the hamstrings.

Methods

Fourteen young active males from the University of Wales, Bangor, volunteered to participate in this study (mean ± standard deviation: age = 20.6 ± 0.8 years; mass = 77.3 ± 10.4 kg; height = 1.77 ± 0.05 m). Institutional ethics approval was granted and all participants provided written informed consent. Participants were randomly allocated to control ($n = 7$) and treatment groups ($n = 7$).

General procedures

Following familiarization procedures, which took place 2–3 days before, baseline measures of flexibility and isometric knee flexion strength at 25 deg, 30 deg, 40 deg, and 80 deg of knee flexion (0 deg = full knee extension anatomically) were recorded in all participants. Prior to these measures, all participants underwent a standardized 5-minute warm up and 5-minute static stretch. The treatment group underwent a 5-week flexibility PNF training program. Training took place twice per week. Previous research has shown that a 3-week program was sufficient to increase flexibility associated with the hamstrings (Rowlands et al. 2003). Both groups subsequently underwent an exercise protocol designed to damage the hamstring musculature. Measures of flexibility, isometric strength, and soreness were taken prior to and 1, 24, 48 and 72 hours following the exercise.

PNF training program

The contract, relax, agonist contract PNF training method was used to elicit an increase in flexibility and compliance of the hamstring muscle group (Rowlands et al. 2003). The participant lay supine on a stable horizontal surface, with both legs fully extended. An assistant helped the participant flex one leg at the hip as far as possible, while maintaining a fully extended knee position. A second assistant held the contralateral leg in the original supine position. The participant performed a maximal voluntary isometric contraction of the antagonist muscle group (hamstrings) for 6 seconds by pushing against the shoulder of the first assistant. The participant then contracted the agonist muscle (quadriceps) and, with assistance, moved their leg to a new end point which was held for 5 seconds. The procedure was carried out a further two times. The whole protocol was repeated for the contralateral leg to minimize the likelihood of any muscle imbalances occurring that may potentially contribute to future injuries. Training took place twice weekly for 5 weeks.

EIMD protocol

The damaging exercise protocol used six sets of 10 isokinetic eccentric contractions of the hamstrings (60 deg s^{-1}, between 80 deg and 10 deg of knee flexion) of the right leg on an isokinetic dynamometer (Kin Com, 500 H, Chattecx, Chattanooga, TN, USA) with the participant in the sitting position. Each set was separated by a 1-minute rest period. Participants were requested to contract the hamstrings maximally to resist knee extension on each repetition and were given verbal encouragement and real-time visual feedback by means of a computerized visual display.

Measures

Isometric strength

The isokinetic dynamometer was used to assess the isometric strength of the hamstrings in the sitting position. The knee joint angles tested during the study were 25 deg (long muscle length), 30 deg, 40 deg and 80 deg (short muscle length) of knee flexion, where 0 deg represents full anatomic extension of the knee. The axis of rotation of the dynamometer was aligned with the lateral femoral condyle. The input arm (containing the force transducer) was attached on a distal point of the tibia with the cuff fastened slightly above the malleolus. To restrict movement of the pelvis, the chest and thigh were secured to the chair with strapping. The vertical, horizontal, and length positions of the lever arm for each participant were recorded. After a warm-up of two submaximal contractions at the assigned joint angles, the participants were asked to perform two sets of maximal 3-second voluntary isometric contractions of the hamstrings at each of the four joint angles. The participants performed the contractions consecutively with a 3-minute recovery period between each joint angle contraction and a 5-minute recovery period between the first and second set of contractions. Verbal encouragement and real-time visual feedback by means of a computerized visual display were provided throughout testing. The mean peak force at each joint angle of contraction was then recorded as the performance measure.

Flexibility

Passive flexibility and compliance associated with the hamstring musculature was measured with an adapted sit-and-reach test. The hip and knee of the undamaged leg were flexed so that the foot rested toward the groin in order to minimize its interference on performance scores. The participants leaned forward slowly and held the position of greatest stretch for 2 seconds. The highest score from three attempts was recorded. A score of 15 cm was equivalent to touching the toes.

Perceived soreness

In the standing position, participants flexed the knee of the damaged leg to 90 deg, with a 1-kg ankle weight attached just above the malleoli in order to apply tension to the hamstrings. Participants indicated their perceived level of soreness on a scale ranging from "*no soreness*" at one end of a continuum to "*the worst soreness ever*" at the other end. On the reverse side, there was a continuous numerical scale ranging from 0 to 10 that allowed the participants' response to be quantified. This scale has been used previously to quantify soreness in EIMD research by the authors (Twist & Eston 2005; Kendall & Eston 2002; Marginson & Eston 2002).

Statistical analysis

Descriptive statistics were calculated for all variables. The effect of the PNF training on flexibility was analyzed using a two-factor (group × time) analysis of variance (ANOVA) with repeated measures on time. Strength data after the damaging exercise protocol were analyzed as a percentage of the baseline score at the respective angle in order to account for potential inter-individual and group differences. A series of two-factor (group × time) ANOVAs were used to explore possible group differences in symptoms of muscle damage following the damaging eccentric exercise protocol. The symptoms examined were: isometric strength at 25 deg, 30 deg, 40 deg and 80 deg of knee flexion; flexibility; and soreness.

Where appropriate, the Greenhouse Geisser correction was used to account for violation of the assumption of sphericity in the ANOVA analyses. *Post hoc* Tukey tests, modified for mixed model ANOVAs (Stevens 1996), were used to follow up significant results. Alpha was set at 0.05. All statistical analyses were carried out using SPSS version 9.0 (SPSS Inc., Chicago, IL, USA).

Results

As anticipated, there was a significant time by group interaction for flexibility ($F_{1,12} = 22.8$, $p < 0.001$). Flexibility increased significantly following PNF flexibility training (pre-test = 19.4 ± 6.2 cm, post-test = 26.6 ± 6.9 cm), whereas it stayed constant in the control group (pre-test = 16.0 ± 9.3 cm, post-test = 16.7 ± 7.6 cm).

Prior to EIMD, strength and flexibility did not differ between groups (strength: $F_{1,12} = 0.1$, $p = 0.826$; flexibility: $t_{12} = 0.8$, $p = 0.435$). Following EIMD, all symptoms showed a main effect for time, which confirmed that the damaging protocol was effective (Figures 1–6). There were borderline interactions of group × time on strength at 25 deg (Figure 1, $F_{4,48} = 2.5$, $p = 0.058$) and 30 deg (Figure 2, $F_{2.3,28.0} = 3.2$, $p = 0.051$), which showed a tendency for the PNF group to recover from strength loss earlier at long muscle lengths, but not at shorter muscle lengths (Figure 3, 40 deg, $F_{4,48} = 1.2$, $p = 0.332$; Figure 4, 80 deg, $F_{4,48} = 1.4$, $p = 0.263$). No interactions were evident for flexibility (Figure 5) or soreness (Figure 6). The PNF trained group were more

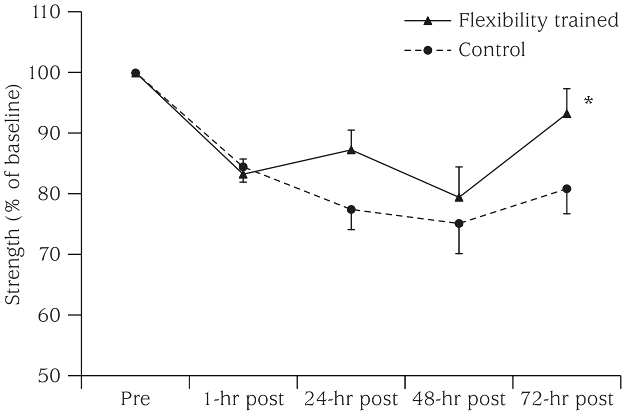

Fig. 1 Strength decrement in hamstrings at 25 deg of full extension (long muscle length) following exercise-induced muscle damage. *Trend for the flexibility-trained group to recover faster ($p = 0.058$). Main effect for time ($p < 0.001$).

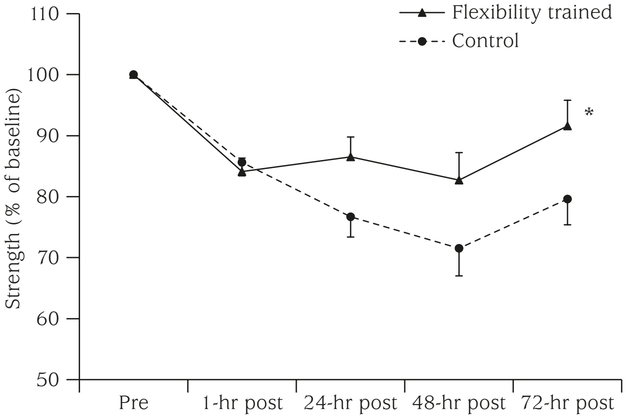

Fig. 2 Strength decrement in hamstrings at 30 deg of full extension following exercise-induced muscle damage. *Trend for the flexibility-trained group to recover faster ($p = 0.051$). Main effect for time ($p < 0.001$).

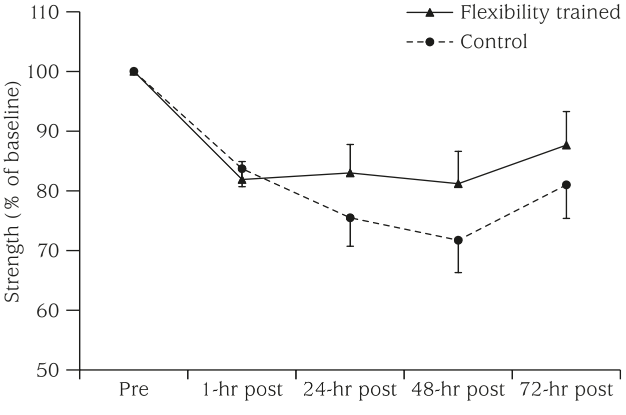

Fig. 3 Strength decrement in hamstrings at 40 deg of full extension following exercise-induced muscle damage. Main effect for time ($p < 0.001$).

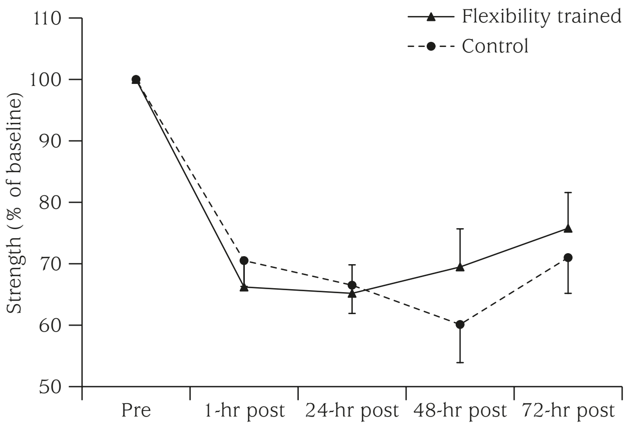

Fig. 4 Strength decrement in hamstrings at 80 deg of full extension (short muscle length) following exercise-induced muscle damage. Main effect for time ($p < 0.001$).

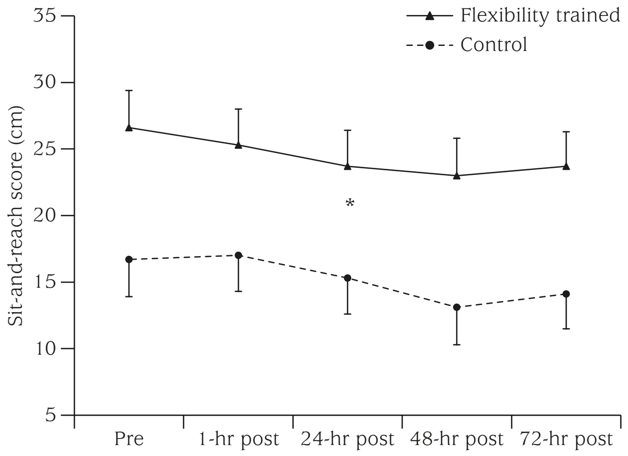

Fig. 5 Flexibility (sit-and-reach score) following exercise-induced muscle damage. *Main effect for group ($p < 0.05$). Main effect for time ($p < 0.001$).

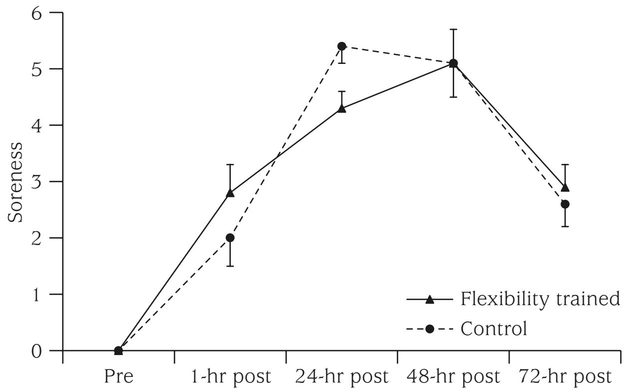

Fig. 6 Perceived soreness following exercise-induced muscle damage. Main effect for time ($p < 0.001$).

flexible at baseline than the control group ($F_{1,12} = 6.1$, $p = 0.03$) and this difference was maintained throughout recovery (Figure 5).

Discussion

The PNF training program led to increased flexibility associated with the hamstring muscle group, as indicated by an increased sit-and-reach score. There was a trend for better maintenance of strength at long muscle lengths following EIMD in the flexibility-trained group, but there was no evidence that increased flexibility offered protection from strength loss at short muscle lengths, soreness, or stiffness.

If, as hypothesized, the PNF training had resulted in the addition of sarcomeres in series, it is logical that the greatest protection would be evident at longer muscle lengths (25 deg and 30 deg). This is consistent with the strong, but not statistically significant, trend demonstrated in the current study. The increased number of sarcomeres in series would decrease the length of individual sarcomeres. Therefore, at long muscle length, fewer sarcomeres would be on the descending limb of the muscle force–joint angle curve, and more would be functioning nearer their optimal length than in the control group. Consequently, less damage would be expected to occur (Morgan 1990).

The relative lack of a protective effect of PNF training in the current study contrasts with the decreased susceptibility of participants with compliant hamstrings to symptoms of EIMD in an earlier cross-sectional study (McHugh et al. 1999). There are several possible explanations for the apparently greater protective effect of compliance or flexibility in the earlier study compared with the current study.

The damaging protocols differed across studies. In the earlier study, participants performed six sets of 10 submaximal (60% maximal voluntary contraction) eccentric contractions of the hamstrings at 2.6 rad s^{-1} (approximately 115 deg s^{-1}). The current study involved the same number of contractions but they were maximal (60 deg s^{-1}). Maximal contractions potentially lead to more damage (Warren et al. 1993), although the degree of damage appears to be more related to the exercising muscle length than the amount of force (Lieber &

Friden 1993). The severity of the symptoms in the studies supports the hypothesis that the damage induced in the earlier study was less severe. Examination of the results of the earlier study shows that symptoms were apparent in the stiffer subjects with relatively mild or no symptoms present in the normal and compliant groups (McHugh et al. 1999). Furthermore, there appeared to be an increase in strength above baseline in the compliant group by 3 days post-damage. In the current study, symptoms of damage were clearly evident in both the PNF trained and control groups.

Although one group was flexibility trained in the current study, and this led to a significant increase in flexibility and compliance of the hamstring musculature, there was some overlap in the performance scores between groups. Additionally, the control group could not be described as being "stiff". Their scores on the sit-and-reach test ranged from 7 to 26 cm, whereas the scores for the trained group ranged from 17 to 36 cm (15 cm being equivalent to touching toes). In contrast, McHugh et al. (1999) compared the top tertile of participants (compliant) to the bottom tertile (stiff) so the groups in their study were clearly distinct. Unfortunately, sex may have intruded as a confounding factor in their study as six of the seven compliant participants were female and six of the seven stiff participants were male. ANCOVA had been used in an attempt to control for this possibility.

The use of young active male participants in the current study with a relatively more severe damaging exercise protocol may have made it more difficult to demonstrate a protective effect of flexibility training. However, one of the strengths of this study is the use of a flexibility training protocol with random allocation of individuals to training or control. This allows the determination of cause and effect, which is not possible with a cross-sectional design. Further research should use this approach to compare the protective effects of flexibility training on participants with low and average baseline flexibility.

In conclusion, this study has demonstrated that an increase in the flexibility of the hamstring muscle group led to some protection from strength loss at long muscle lengths following EIMD. However, no protection from soreness, stiffness, or strength loss at short muscle lengths was evident. Further research should examine potential protective effects of flexibility training on symptoms of EIMD on people differing in baseline flexibility.

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