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Title: The role of neuromuscular inhibition in hamstring strain injury recurrence.

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ABSTRACT

Hamstring strain injuries are amongst the most common and problematic injuries in a wide range of sports that involve high speed running. The comparatively high rate of hamstring injury recurrence is arguably the most concerning aspect of these injuries. A number of modifiable and non-modifiable risk factors are proposed to predispose athletes to hamstring strains. Potentially, the persistence of risk factors and the development of maladaptations following injury may explain injury recurrence. Here, the role of neuromuscular inhibition following injury is discussed as a potential mechanism for several maladaptations associated with hamstring re-injury. These maladaptations include eccentric hamstring weakness, selective hamstring atrophy and shifts in the knee flexor torque-joint angle relationship. Current evidence indicates that athletes return to competition after hamstring injury having developed maladaptations that predispose them to further injury. When rehabilitating athletes to return to competition following hamstring strain injury, the role of neuromuscular inhibition in re-injury should be considered.

MAIN TEXT

1. INTRODUCTION

Hamstring strains are amongst the most common and problematic injuries in a wide range of sports that involve high speed running. They are the primary injury in Australian football [Gabbe et al., 2002; Orchard & Seward, 2002; Orchard et al., 2009, 2010; Seward et al., 1993], soccer [Ekstrand et al., 1983; Ekstrand et al., 2010; Hawkins et al., 2001; Woods et al., 2002; Woods et al., 2004] and the sprint events in track & field [Drezner et al., 2005; Sugiura et al., 2008; Yeung et al., 2009], while also accounting for a high proportion of lost playing time in cricket [Orchard et al., 2003; Orchard, James et al., 2002; Stretch, 2003], rugby union [Brooks et al., 2005a, 2005b, 2005c, 2006], Gaelic football [Newell et al., 2006; O'Sullivan et al., 2008], American football [Elliott et al., 2011; Feeley et al., 2008; Meeuwisse et al., 2000] and hurling [Murphy et al., 2010].

High rates of injury recurrence are arguably the most troublesome aspect of hamstring strains because recurring injuries often result in substantially more lost playing time than the original insults [Brooks et al., 2006; Ekstrand et al., 2011; Koulouris et al., 2007] and because the hamstrings remain at elevated risk of injury recurrence for longer than other strained muscles [Orchard & Best, 2002]. There is also evidence for a significant rate of recurrence across consecutive seasons [Carling et al., 2011; Hagglund et al., 2006; Verrall et al., 2006].

Hamstring strain recurrence rates of 16-60% have been reported in American football [Elliott et al., 2011; Heiser et al., 1984], rugby union [Brooks et al., 2006], soccer [Ekstrand et al., 2011], the sprint events in track and field [Drezner et al., 2005; Yeung et al., 2009] and in Australian football [Orchard et al., 2010]. A long-term program of compulsory injury reporting in the Australian Football League (AFL) indicates that same-season hamstring re-injury rates have averaged 23% in the last 10 seasons [Orchard et al., 2010]. However, this figure almost certainly underestimates true recurrence rates as it does not take into account lost pre-season training time.

Statistics on first and recurrent hamstring strain injuries indicate that injury prevention and rehabilitation practices are not as effective as athletes and coaches would like them to be. We

argue that the current understanding of injury risk factors is inadequate and that neuromuscular inhibition of the hamstrings, previously ignored in literature and underemphasised in rehabilitation programs, may at least partially explain high hamstring injury recurrence rates. The objectives of this review article are to i) discuss factors relating to hamstring strain injury recurrence and ii) discuss the potential role of neuromuscular inhibition in these recurrences.

2. WHAT FACTORS EXPLAIN THE HIGH RATES OF HAMSTRING INJURY RECURRENCE?

2.1 The persistence of risk factors

A number of modifiable and non-modifiable risk factors are proposed to predispose athletes to hamstring strains [Copeland et al., 2009; Croisier, 2004a; Devlin, 2000; Opar et al., 2012]. Modifiable factors include hamstring weakness [Croisier et al., 2008; Orchard et al., 1997; Sugiura et al., 2008; Yeung et al., 2009], poor flexibility [Bradley et al., 2007; Witvrouw et al., 2003] and fatigue [Brooks et al., 2006; Ekstrand et al., 2011; Woods et al., 2004], while the non-modifiable factors include age [Arnason et al., 2004; Gabbe et al., 2006c; Hagglund et al., 2006; Orchard, 2001; Woods et al., 2004], ethnicity [Brooks et al., 2006; Verrall et al., 2001; Woods et al., 2004] and a history of hamstring injury [Arnason et al., 2004; Gabbe et al., 2006a; Hagglund et al., 2006; Orchard, 2001; Verrall et al., 2001]. Injury recurrence could potentially be explained by the persistence of one or more risk factors after rehabilitation and the return to sport [Croisier, 2004a]. However, a history of hamstring injury is frequently reported to be a stronger predictor of future injury than other risk factors [Arnason et al., 2004; Hagglund et al., 2006; Orchard, 2001; Verrall et al., 2001], and this observation is consistent with the possibility that hamstring strain injury causes maladaptations which act as significant contributors to re-injury if not adequately addressed during rehabilitation. The following sections will outline some of these potential maladaptations and their possible association with hamstring strain injury recurrence.

2.2 Scar tissue

Scar tissue formation at the site of muscle rupture plays an important role in early healing [Järvinen et al., 2005; Kääriäinen et al., 2000]. The inelastic scar, however, may persist for months or become permanent [Kääriäinen et al., 2000; Silder et al., 2010a] and thereby increase the strain experienced by in-series muscle fascicles after the return to training and competition [Silder et al., 2010a]. Over-extension of fascicles in-series with the scar may then lead to re-injury. This possible role of scar tissue in hamstring re-injury is further discussed below.

2.3 Reduced flexibility

Anyone who has suffered a muscle strain injury will have experienced the acute negative impact on flexibility [Malliaropoulos et al., 2004]. Sometimes reductions in flexibility become chronic [Jönhagen et al., 1994] and some have proposed that long-lasting reductions in flexibility increase the risk of re-injury [Malliaropoulos et al., 2004]. However, there is mixed evidence for flexibility as a risk factor for strain injury. The majority of prospective studies have found no association between hamstring flexibility and increased injury risk [Arnason et al., 2004; Bennell et al., 1999; Gabbe et al., 2006a; Gabbe et al., 2005; Orchard et al., 1997; Yeung et al., 2009], although some have reported higher injury rates in athletes with poorer flexibility [Bradley et al., 2007; Henderson et al., 2009; Witvrouw et al., 2003]. It is important to consider, however, that most existing studies are too small to identify anything other than very strong associations between risk factors and injury rates. For example, Bahr & Holme [2003] suggest that prospective studies require 20-50 injured subjects to identify moderate to strong associations between risk factors and injury rates, whilst 200 injury cases may be required to detect small to moderate associations between these parameters. Furthermore, flexibility development may be more important in recurrent than first time injury and very large scale prospective studies, with many previously injured participants, would be required to adequately assess this possibility [Bahr & Holme, 2003].

The results of intervention studies [Arnason et al., 2008; Sherry & Best, 2004] and one randomised controlled trial [van Mechelen et al., 1993] also question the benefits of enhanced flexibility in reducing hamstring injuries. A non-randomised intervention involving contract-relax hamstring stretches in elite Scandinavian soccer teams failed to reduce injury rates which were statistically indistinguishable from those of teams that declined to follow the program [Arnason et al., 2008]. A limitation of this study, however, is that the stretching practices of the non-participating teams were not known or controlled. In a randomised comparison of two rehabilitation programs, Sherry and Best [2004] compared a progressive agility and trunk stabilisation approach to one that involved isolated strengthening and stretching of injured muscles. The strengthening and stretching was particularly ineffective as it resulted in significantly more injuries in the one year follow-up period than the alternative program [Sherry & Best, 2004].

2.4 Muscle weakness

Weakly activated or fatigued animal muscles absorb less energy before experimentally induced failure than more strongly activated or unfatigued muscles [Garrett et al., 1987]. Weak hamstrings may therefore absorb insufficient energy during the presumably injurious terminal swing phase of running [Heiderscheit et al., 2005; Schache et al., 2009] and become injured as a consequence.

Comparisons of eccentric knee flexor strength between previously injured and uninjured limbs or between previously injured and uninjured athletes have revealed significant deficits in the previously injured hamstrings [Croisier, 2004a, 2004b; Croisier et al., 2002; Croisier & Crielaard, 2000; Dauty et al., 2003; Jönhagen et al., 1994; Lee et al., 2009]. A degree of strength deficit may have preceded some injuries [Croisier et al., 2008; Sugiura et al., 2008; Yeung et al., 2009], however, it seems likely that between-leg strength asymmetries increase after hamstring strains as prospective studies have identified deficits of ~4.5% in eccentric hamstring strength to be associated with future injury [Sugiura et al., 2008], whilst much

larger deficits (22 – 24%) have been noted following injury [Croisier et al., 2002]. If weakness is a consequence of injury, it appears to be long lasting, as some of these studies involved isokinetic strength testing months to years after the insult and well after the return to competition [Croisier & Crielaard, 2000; Croisier et al., 2002, Dauty et al., 2003; Jönhagen et al., 1994; Lee et al., 2009]. Interestingly, eccentric deficits typically exceed declines in concentric strength (see Figure 1), which are often small or absent [Croisier & Crielaard, 2000; Croisier et al., 2002, Lee et al., 2009]. This contraction-mode specific weakness may explain why some studies employing concentric tests of hamstring function have reported no chronic strength deficits after injury [Brockett et al., 2004; Paton et al., 1989; Silder et al., 2010b; Worrell et al., 1991].

The predominantly eccentric weakness exhibited by previously injured athletes is strongly suggestive of heightened neuromuscular inhibition which is, in our view, the only established mechanism that can explain observations of eccentric strength being lower than or equal to concentric strength as reported by Croisier and Crielaard [2000].

INSERT FIGURE 1 HERE

Two prospective studies suggest that a low eccentric hamstring to concentric quadriceps torque ratio (the functional H:Q ratio) predisposes athletes to elevated risk of hamstring strain [Croisier et al., 2008; Sugiura et al., 2008], although Bennell and colleagues [1998] found no such association. Others have reported that a low concentric [Orchard et al., 1997; Yeung et al., 2009] or isometric [Yamamoto, 1993] H:Q ratio predicts hamstring injury.

Methodological differences between these prospective studies, including injury definitions, subject populations, testing protocols and/or other unknown factors may have contributed to these discrepant findings. Nevertheless, of these studies Croisier and colleagues [2008] was the largest (n = 462) and most convincing. Soccer players with hamstring strength deficits, including limb-to-limb asymmetries or low H:Q ratios were particularly prone to hamstring

strain injury. Interestingly, a relatively small number of players were identified as having concentric ‘strength disorders’ while eccentric measures of strength and particularly the functional H:Q quadriceps ratio detected many more players with deficiencies [Croisier et al., 2008]. Finally, strength training interventions, involving follow-up tests until the low functional H:Q ratios were normalised (i.e., strength of the normalised leg corresponded to a less than 5% deficit through bilateral comparison and the concentric H:Q and functional H:Q ratios were more than 0.57-0.55 and 0.98-1.05, respectively), reduced injury rates significantly in comparison with players whose deficits were not treated or retested [Croisier et al., 2008].

Three large intervention studies employing the Nordic hamstring curl also provide evidence for the protective role of eccentric hamstring conditioning in soccer [Arnason et al., 2008; Petersen et al., 2011] and rugby union [Brooks et al., 2006]. The most recent of these recruited 942 Danish male soccer players in a cluster-randomised controlled trial [Petersen et al., 2011]. Approximately half of the participants followed a program of Nordic hamstring curls which were performed 24 times in the 10 week pre-season before being continued once each week in the competitive season [Petersen et al., 2011]. Hamstring injury rates for the intervention group were approximately one third of those in control teams that maintained their normal conditioning practices. Segregation of injuries into first time and recurrent strains revealed that previously injured players who employed the Nordic curl were approximately six times less likely to suffer a recurrence than previously injured players from control teams [Petersen et al., 2011]. This finding strongly supports the benefits of eccentric hamstring training in rehabilitation from hamstring strain.

It is tempting to interpret the aforementioned studies as evidence for the protective role of improved eccentric hamstring strength. However, it is also conceivable that the benefits may be at least partly explained by shifts in knee flexor force-length relationship [Brockett et al.,

2001] or by connective tissue or cytoskeleton changes induced by eccentric conditioning [McHugh, 2003].

Others have shown no benefits of Nordic curls on hamstring injury rates in Australian football [Gabbe et al., 2006b] and soccer [Engebretsen et al., 2008], however, these studies reported extremely low compliance rates. Less than 50% of participants in Gabbe and colleagues' [2006b] study did two of five planned exercise sessions. This study also employed the Nordic curl only five times in 12 weeks [Gabbe et al., 2006b], which is not consistent with accepted strength training frequencies [Ratamess et al., 2009] or previous studies that have reported gains in eccentric strength [Mjølsnes et al., 2004] and injury prevention benefits [Arnason et al., 2008; Petersen et al., 2011] after more frequent use of this exercise. A strength training program based around isometric contractions, prone leg curls performed with ankle weights and Thera-band resisted hip extensions has also proven ineffective in rehabilitation from hamstring injury in comparison with a progressive agility and trunk stability program [Sherry & Best, 2004]. Based on other more recent studies, it seems possible that a lack of progression to more intense eccentric strength training might at least partially explain these poor results.

While discussing the effects of eccentric hamstring weakness on strain injury risk, it is important to recognise that running has been shown to reduce eccentric knee flexor strength more than concentric knee flexor or knee extensor strength [Greig, 2008; Koller et al., 2006; Oliveira Ade et al., 2009; Small et al., 2008, 2009]. This produces a reduction in the functional hamstrings to quadriceps ratio that may increase hamstring injury risk in athletes who have completely adequate functional ratios and levels of eccentric knee flexor strength in an unfatigued state.

2.5 Selective hamstring atrophy

A majority of hamstring strains incurred during running affect the long head of the biceps femoris [Askling et al., 2007; Koulouris et al., 2007; Silder et al., 2008; Verrall et al., 2003; Woods et al., 2004]. This muscle may be predisposed to strain injury because it reaches slightly longer relative lengths than the medial hamstrings during the terminal swing phase of running gait [Thelen et al., 2005].

Silder and colleagues [2008] have shown that recreational athletes with prior strains to the long head of biceps femoris displayed reduced muscle volume of that portion of the hamstrings 5-23 months after injury, despite having returned to competition. Many of the athletes in Silder and colleagues' [2008] study also exhibited concomitant hypertrophy of the short head of the biceps femoris, which suggests that atrophy of the long head is not a consequence of reduced knee flexor loading. Again, these observations are strongly suggestive of chronic inhibition of the biceps femoris long head activation following strain injury to that muscle.

2.6 Shifts in the torque-joint angle relationship

The torque-joint angle relationships of human skeletal muscles have been reported to adapt, in a contraction-mode specific manner, to concentric and eccentric training programs [Kilgallon et al., 2007]. Concentric hamstring training drives the knee flexor torque-joint angle relationship towards shorter muscle lengths [Kilgallon et al., 2007], while eccentric training has the opposite effect [Kilgallon et al., 2007] while also increasing fascicle lengths [Potier et al., 2009]. Presumably, shifts in human torque-joint angle curves are at least partly driven by changes in the number of in-series sarcomeres as observed in rat vastii muscles after as little as a week of daily uphill (predominantly concentric) or downhill (predominantly eccentric) running [Lynn et al., 1994; Lynn et al., 1998]. Shifts in angles of peak torque in human studies also occur rapidly and have been noted after as little as three weeks or seven training sessions [Kilgallon et al., 2007].

Torque-joint angle relationships derived from slow concentric [Brockett et al., 2004; Brughelli et al., 2009] or eccentric [Sole et al., 2011] knee flexion dynamometry reveal that previously injured knee flexors generate their peak torques at shorter muscle lengths than uninjured contralateral knee flexors [Brockett et al., 2004; Proske et al., 2004]. For example, Brockett and colleagues [2004] reported a 12° shift in the knee angle of peak flexion torque in previously injured limbs compared to the contralateral uninjured limbs. Again, there is no conclusive proof that these unusual angles of peak torque were not evident prior to the initial hamstring injury, however, the uninjured limbs of previously injured athletes demonstrated angles of peak torque that were indistinguishable from those found in athletes who had never suffered a strain injury [Brockett et al., 2004]. If one assumes a level of limb-to-limb symmetry prior to injury, this is consistent with the possibility that the difference in optimal angle is at least partly due to injury.

A shortened optimal length results in muscles operating to a greater extent on the descending limb of their force-length relationships and this may predispose muscles to greater microscopic damage and post-exercise weakness as a consequence of the powerful active lengthening that occurs during running [Brockett et al., 2004; Morgan, 1990; Proske et al., 2004]. An accumulation of such damage, which might occur as a consequence of numerous consecutive sessions of high-speed running, is proposed to result in macroscopic muscle strain [Brockett et al., 2004; Morgan, 1990]. At present, only a single small-scale (n = 44) prospective investigation has been carried out to test this hypothesis and it did not find any elevated risk in sprinters with greater angles of peak torque [Yeung et al., 2009]. Again, however, we must acknowledge that even moderately strong associations between angle of peak torque and injury risk may not have been detected by such a small study (Bahr & Holme, 2003).

Brockett and colleagues [2004] proposed that the shift in the torque-joint angle relationship after hamstring strain is mediated by a reduction in the number of in-series sarcomeres within muscle fascicles, possibly as a consequence of both the healing process and rehabilitation practices. In relation to the healing process, the formation of scar tissue was proposed to take the place of some in-series sarcomeres along the length of previously injured fascicles, thereby shifting optimal torque to shorter muscle lengths [Brockett et al., 2004].

Contemporary hamstring rehabilitation practices, which initially minimise muscle lengths and eccentric loads while emphasising isometric and concentric force generation [Drezner, 2003; Heiderscheit et al., 2010], may also permit a reduction in in-series sarcomeres and leave the muscle prone to post-exercise weakness and damage when it is eventually exposed to forceful active lengthening [Gleeson et al., 2003; Whitehead et al., 1998].

Like the loss of eccentric strength, the altered angle of peak knee flexion torque following hamstring strains appears to persist for months to years after injury (Brockett et al., 2004; Lee et al., 2009). However, there is currently no convincing explanation as to why something as highly adaptable as the torque-joint angle relationship does not respond to the demands placed on it by the return to full training and competition. While scar tissue has been proposed to contribute to shifts in the torque-joint angle relationship after hamstring injury, muscle fibres in-series with the scar appear to experience greater than normal strain [Silder et al., 2010a] and, if not causing re-injury first, this should act as a powerful stimulus for a compensatory sarcomerogenesis that would drive the optimal torque back towards longer muscle lengths.

Recent work by Sole and colleagues [2011] suggests an additional mechanism by which the angle of peak torque might shift to shorter lengths after strain injury. These authors showed that previously injured hamstrings were less completely activated during maximal eccentric actions at long muscle lengths than muscles of uninjured athletes [Sole et al., 2011]. This would effectively spare the recovering hamstring muscles from exposure to high forces at

long lengths, which could potentially reduce the stimulus for sarcomerogenesis otherwise provided in late rehabilitation. As a consequence, the reduced ability to activate the previously injured hamstrings at long lengths may be a neural mechanism for a shift in the torque-joint angle relationship and sustained neuromuscular inhibition may also limit the muscular adaptation that would otherwise shift this relationship back towards its optimal state.

2.7 Is rehabilitation sabotaged by neuromuscular inhibition?

The following section discusses the putative role of neuromuscular inhibition in hamstring strain injury recurrence [Opar et al., 2012]. A conceptual framework for the purported relationship between neuromuscular inhibition and several maladaptations associated with hamstring strain injury recurrence is shown in Figure 2.

INSERT FIGURE 2 HERE

The early stages of conventional hamstring rehabilitation are characterised by the avoidance of excessive muscle stretch, initially because stretch may exacerbate scar formation [Heiderscheidt et al., 2010; Järvinen et al., 2005; Kääriäinen et al., 2000]. As a consequence of these practices and the additional effect of a sudden reduction in physical activity, the long heads of the hamstrings might be expected to exhibit both a reduction in in-series sarcomeres and significant atrophy [Williams et al., 1978] if the strain is sufficiently severe (grade II or higher) [Drezner, 2003]. Progressively more intense running and strength training in late rehabilitation would be expected to increase the previously injured muscle's exposure to forceful eccentric actions at relatively long muscle lengths [Chumanov et al., 2007, 2011; Heiderscheidt et al., 2005; Schache et al., 2009; Simonsen et al., 1985; Thelen et al., 2005; Wood, 1987]. As a consequence, late rehabilitation might be thought sufficient to return hamstring fascicles to their pre-injury lengths and muscle bellies to their original size. However, if there were lingering neuromuscular inhibition it would reduce the activation of

the previously injured muscle, particularly during eccentric actions and at longer muscle lengths [Sole et al., 2011]. This would be expected to limit muscle hypertrophy as eccentric actions are powerful stimuli for muscle growth [Roig et al., 2009], particularly when performed at long muscle lengths [Butterfield et al., 2006]. Neuromuscular inhibition, which has the potential to be long-lasting after painful injury, may therefore explain the persistent atrophy that has been observed in the previously injured biceps femoris long head long after injury and despite the return to full training [Silder et al., 2008].

The possibility that neuromuscular inhibition of the hamstrings contributes to injury recurrence has, to our knowledge, not been previously proposed. Hamstring injuries are acutely painful and this pain may become chronic, particularly in athletes with recurrent strains [Brughelli et al., 2009; Croisier et al., 2002]. Joint and muscle pain are both known to induce acute neural responses, which can reduce strength, reduce agonist activation, increase antagonist activity, reduce muscle endurance and alter coordination patterns during static and dynamic motor tasks [Diederichsen et al., 2009; Graven-Nielsen et al., 2008, 2010; Henriksen et al., 2007]. Reduced voluntary activation of injured muscles presumably serves the purpose of reducing tissue loading soon after injury. However, pain of muscular origin also has the potential to cause chronic adaptations within the central nervous system at both spinal and supraspinal levels [Graven-Nielsen et al., 2010; Mense, 2003]. Some of these changes could potentially limit the capacity to voluntarily activate injured muscles.

Long-lasting deficits in maximal voluntary activation of surrounding skeletal muscles have been observed after a range of painful joint injuries, including isolated anterior cruciate ligament (ACL) rupture [Urbach et al., 2002; Urbach et al., 2001; Urbach et al., 1999], traumatic knee injuries involving ACL rupture, meniscectomy, tibial plateau or femoral condyle fractures [Hurley et al., 1994] and ankle fractures [Behm et al., 1997]. Voluntary activation deficits induced by arthrogenic inhibition are difficult to overcome and do not always respond even to intense rehabilitation [Hurley et al., 1994; Rice et al., 2010]. While

typically diminished, these deficits may also persist after joint pain has abated [Hodges et al., 2010].

3. CONCLUSION

The persistence of risk factors and the development of maladaptations following hamstring strain injury may explain injury recurrence. Several post-hamstring injury maladaptations have been identified in the literature, which, if not specifically addressed during rehabilitation, potentially contribute to injury recurrence. It is proposed that pain-driven neuromuscular inhibition of hamstring voluntary activation occurs following hamstring strain injury, and that this inhibition has a detrimental effect on hamstring recovery by limiting hamstring exposure to eccentric stimuli at long muscle lengths during rehabilitative exercise. This limited exposure to eccentric stimuli could potentially produce several maladaptations observed following hamstring injury, including chronic eccentric hamstring weakness, selective hamstring atrophy and shifts in the torque joint-angle relationship. There is evidence consistent with the possibility that current rehabilitation practices do not adequately address the need to increase voluntary muscle activation at long muscle lengths, possibly for fear of causing re-injury or simply because these practices deal with only the acute stages of recovery. Voluntary activation deficits may require more attention in late rehabilitation and eccentric exercise seems appropriate for this purpose because of its well-recognised positive effects on voluntary muscle activation [Hortobagyi et al., 1996; Hortobagyi et al., 1997; Pensini et al., 2002]. Further work is needed to clarify the potential role contribution of neuromuscular inhibition to hamstring injury recurrence and to examine the efficacy of various rehabilitation protocols on hamstring voluntary activation following injury.

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6. TABLES

No tables included in the main text.

7. CAPTIONS TO ILLUSTRATIONS

Figure 1. Unpublished observation from our laboratory comparing knee flexion torque-velocity relationship of a participant's previously injured hamstrings to the contralateral uninjured hamstrings.

Figure 2. Conceptual framework for the putative role of neuromuscular inhibition following hamstring strain injury in the development of several maladaptations associated with increased re-injury risk. * = Particularly at long muscle lengths, # = biceps femoris (BF) specific.

8. ILLUSTRATIONS

Figure 1:

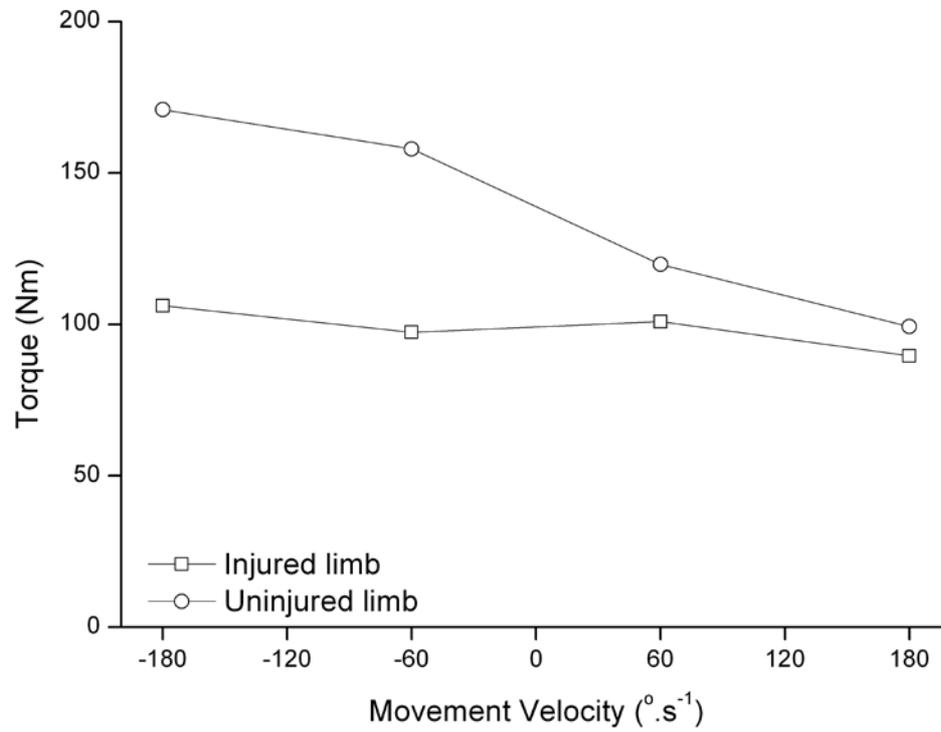


Figure 2:

