A. Title page

Hamstring strain injuries: Factors that lead to injury and re-injury.

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E. Figure Captions

Figure 1 – Injury incidence in the Australian Football League over 13 years. An injury is defined as “any physical or medical condition that prevents a player from participating in a regular season (home and away) match”.[7] HSI, hamstring strain injury; OP, osteitis pubis. Figure adapted from Orchard & Seward.[7]

Figure 2 – The active length-tension curve adapted from Gordon, Huxley & Julian.[63] The descending limb of the active length-tension curve, where sarcomere force declines as length increases, occurs when there is a reduction in the overlap of myosin cross-bridges and actin filaments. Morgan has proposed this is a region of instability where nonuniform lengthening of sarcomeres in-series leads to differences in maximal sarcomere force.[61]

Figure 3 – Unpublished concentric knee flexor torque–joint angle relationships from a single elite male athlete tested at 60°/second in our laboratory. Angle of peak torque is indicated by the downward arrows. 0º indicates full knee extension, 100º indicates 100 degrees of knee flexion. The previously injured hamstring produces its peak torque at shorter muscle lengths (greater angle of peak torque), and hence operates to a greater extent along the descending limb of the length-tension curve.

F. Abstract

Abstract

Hamstring strain injuries (HSI) are common in a number of sports and incidence rates have not declined in recent times. Additionally, the high rate of recurrent injuries suggests our current understanding of HSI and re-injury risk is incomplete. Whilst the multifactoral nature of HSI is agreed upon by many, often individual risk factors and/or causes of injury are examined in isolation. This review aims to bring together the causes, risk factors and interventions associated with HSI to better understand why HSI are so prevalent. Running is often identified as the primary activity type for HSI and given the high eccentric forces and moderate muscle strain placed on the hamstrings during running these factors are considered to be part of the aetiology of HSI. However the exact causes of HSI remain unknown and whilst eccentric contraction and muscle strain purportedly play a role, accumulated muscle damage and/or a single injurious event may also contribute. Potentially all of these factors interact to varying degrees depending on the injurious activity type (i.e. running, kicking). Furthermore anatomical factors such as the biarticular organisation, the dual innervations of biceps femoris (BF), fibre type distribution, muscle architecture and the degree of anterior pelvic tilt have all been implicated. Each of these variables impact upon HSI risk via a number of different mechanisms, including increasing hamstring muscle strain and altering the susceptibility of the hamstrings to muscle damage. Reported risk factors for HSI include age, previous injury, ethnicity, strength imbalances, flexibility and fatigue. Of these little is known, definitively, about why previous injury increases the risk of future HSI. Nevertheless, interventions put in place to reduce HSI incidence by addressing modifiable risk factors have focussed primarily on increasing eccentric strength, correcting strength imbalances and improving flexibility. The response to these intervention programs has been mixed with varied levels of success reported. A conceptual framework is presented suggesting that neuromuscular inhibition following HSI may impede the rehabilitation process and subsequently lead to maladaptation of hamstring muscle structure and function

including preferentially eccentric weakness, atrophy of the previously injured muscles and alterations in the angle of peak knee flexor torque. This remains an area for future research and practitioners need to remain aware of the multifactoral nature of HSI if injury rates are to decline.

G. Text pages

1. Introduction

Hamstring strain injuries (HSI) are the most prevalent non-contact injury in Australian football, [1-7] American football,[8] rugby union,[9-12] soccer, [13-17] and sprinting. [18-19] HSI are characterised by acute pain in the posterior thigh with disruption of the hamstring muscle fibres. [20] HSI range in severity from minor microscopic tearing and some loss of function (grade one) through to a full rupture of the muscle with complete loss of function (grade three). [21] The biceps femoris (BF) is the most commonly injured of the hamstring muscles[22-24] with the muscle-tendon junction and adjacent muscle fibres being the most common sites of disruption.[22, 25]

In many cases, HSI cause considerable time lost from training and competition[7, 9, 15, 26] which results in financial loss[27] and diminished athletic performance.[28] Injury has been suggested to have cost in excess of £74.4m in English premier and football league clubs during the 1999-2000 season.[27] Similar estimates, made by the authors, for elite Australian football teams indicate that HSI cost approximately AUD$1.5m in the 2009 season, which represents 1.2% of the salary cap in the Australian Football League. Furthermore, player performance has been found to be significantly reduced following return from HSI in elite Australian footballers.[28]

Epidemiological data obtained from Australian football, rugby union and soccer across a number of years indicates that rates of HSI have not declined in recent decades (Figure 1).[1-2, 9, 13, 15, 17, 29] This is particularly worrying when taking into account that HSI has for a long time been a well documented problem which has received considerable attention in the literature. Moreover, other injuries, such as ankle sprains in soccer[30] and posterior cruciate ligament injuries in Australian football,[31] have shown reduced incidence rates following the implementation of relatively effective preventative measures. The lack of decline in HSI rates

highlights that current practices aimed at preventing them requires further scientific investigation. In particular, whilst a number of risk factors for HSI have been identified, the potential role of the nervous system in strain injury aetiology has been largely overlooked. Furthermore, whilst it is commonly accepted that the aetiology of HSI is complex and multifactorial in nature[5, 9, 16] these factors are often considered in isolation. This review examines the causative and risk factors purportedly associated with HSI, taking an integrated approach to further understand how these factors may interact and also considers the impact of intervention programs on these variables. Assimilating this information we also propose a novel hypothesis as to how previous injury may lead to maladaptation of hamstring structure and function.

2. Literature search

The articles selected for review were obtained via searches of MEDLINE and SPORTDiscus between 1966 and April 2011. The following keywords were searched in combination: 'hamstring', 'knee flexor', 'muscle strain', 'injury', 'mechanism', 'risk factors' and 'prevention'. From the abstracts returned, articles were included for review if they related to hamstring injury incidence, causation, risk factor analysis or prevention. Full text copies of selected articles were then sourced and the reference lists of these articles were hand searched to identify other potential articles.

3. Hamstring strain injury incidence and recurrence rates

In track and field one group has reported that HSI account for 26.0% of all injuries sustained, with most occurring in sprinting events.[19] In comparison, observations from Australian football and soccer indicate that HSI are responsible for 13-15%[1-2, 4] and 12-14%[13-14, 16] of all injuries respectively. These figures are comparable to reports from American football training camps (12%)[8] and rugby union training (15%).[12] HSI are also the single largest

cause of lost playing time in Australian football[7] and are the predominant injury type responsible for prolonged absence (> 28 days) from training and playing in soccer.[13]

When compared to earlier epidemiology data from Australian football,[2] rugby union[2] and soccer,[17] recent observations indicate that the incidence of HSI in sport has trended upwards over the past two decades. Further, data from the Australian Football League Annual Injury Report displays an increasing trend in the incidence of HSI over the past seven competitive seasons whilst other major injuries, including other prevalent lower limb muscle strains, have remained largely stagnant (Figure 1).[7]

In addition to high incidence rates and significant time lost, HSI also exhibits a very high rate of recurrence.[2, 7, 9, 15, 32-35] Over 13 seasons of observation, 27% of all HSI in the Australian Football League are recurrences of previous injuries, however recent evidence suggests this is trending downwards arguably because of a more conservative approach in return to play strategies rather than improved rehabilitation practices.[7] Similarly high rates of HSI recurrence have also been reported in American football (32%),[32] rugby union (21%)[9] and soccer (16%).[36]

4. Hamstring function during running and potential for strain injury

Although kicking, tackling, cutting and slow-speed stretching can result in HSI,[9, 15, 17, 24, 37] running accounts for the majority of HSI in soccer[15] and rugby union,[9] which suggests the demands of running give the greatest insight into the causes of HSI.

Studies of running biomechanics have found the hamstrings are active for the entire gait cycle with peaks in activation during the terminal swing and early stance phases.[38-39] During the terminal swing phase the hamstrings are required to contract forcefully whilst lengthening to decelerate the extending knee and flexing hip.[38, 40-43] It is also in terminal swing that the

hamstrings reach their maximum length.[38, 43] Of the three biarticular hamstring muscles, biceps femoris long head (BF_L) undergoes the greatest stretch, reaching almost 110% of the length in upright standing during terminal swing whilst semimembranosus (SM) and semitendinosus (ST) reach 107.5% and 108.2% respectively.[43] In contrast, the maximum torques for hip extension and knee flexion are found to occur during ground contact in overground sprinting.[44] During this phase the hamstrings are acting primarily concentrically to extend the hip,[40] however it has been reported that an eccentric contraction of the hamstrings occurs during the late stance phase of overground sprinting.[38]

The presence of a high force eccentric contraction during the stance[38] and swing[38, 41-43, 45] phases likely contributes to the high rates of HSI during maximal speed running. The terminal swing phase is considered the most hazardous as the hamstring muscle-tendon units are at their longest length of the gait cycle and are most heavily activated.[38, 41-43, 45] This suspicion has been supported by two independent serendipitous observations of acute HSI during biomechanical studies of running, the timing of which was consistent with the insult occurring in terminal swing.[46-47] Whilst the stance phase is another possible period of susceptibility to HSI, due to high hip extension and knee flexion torque,[44, 48] it involves much shorter hamstring lengths compared to terminal swing.[38, 41-43]

5. Causes of hamstring strain injuries

In addition to strain injuries the hamstrings are also affected by tendinopathies[9] and back related injuries that referred pain to the posterior thigh.[20] These injuries display varying aetiological characteristics and as such the causes of these injuries vary considerably. For the purposes of this chapter the focus will be on the cause of HSI during running.

There is some debate as to whether muscle strain or the magnitude of eccentric force is the causative factor in muscle strain injuries. Observations from *in-situ* animal models suggest

that the magnitude of muscle strain is the primary determining factor in the occurrence of strain injury.[49-51] Many investigators have also suggested that *in-vivo* muscle strain injuries are associated with high force eccentric contractions,[38, 41-42, 51-58] where the lengthening demands placed on the muscle exceed the mechanical limits of the tissue.[41] It remains to be seen if both high eccentric force and high muscle strain are necessary conditions for a strain injury or whether each on their own is sufficient to bring about strain injury. Biomechanical observations suggest that eccentric contraction is a necessary condition for a HSI during running[46-47] and this claim is strengthened by the lack of strain injuries in concentrically-biased sports such as swimming and cycling.[59-60] An argument for muscle strain being a necessary condition is less clear given that HSI have been reported for both high (i.e. kicking)[9, 15] and low (i.e. sprinting)[9, 15, 19, 25] strain tasks. Potentially an interrelationship exists between eccentric force and muscle strain that dictates whether a muscle is injured. For example, strain injury may be avoided in tasks that involve high levels of strain if the level of eccentric force is low and the same may be true for high eccentric force/low strain activities.

There is also some uncertainty as to whether HSI most typically occur as a result of accumulated microscopic muscle damage,[61] or as a result of a single event that exceeds the mechanical limits of the muscle.[62] It seems feasible, however, that both may contribute. For example, the accumulation of microscopic damage may leave the muscle tissue in a vulnerable state and more susceptible to injury in the event of a single traumatic event such as bending to pick up or catch a ball.

Whilst the potential role of accumulated muscle damage in muscle strain injury aetiology is not disputed, debate continues as to the physiological process responsible for damage. Morgan[61] first proposed the accumulated damage theory when he postulated that microscopic damage caused to individual sarcomeres following eccentric exercise was as a result of preferential lengthening of weaker sarcomeres. This theory suggests that during

eccentric contractions there is non-uniform lengthening of adjacent sarcomeres when muscles are operating on the descending limb of the length-tension curve (Figure 2).[61] This difference in sarcomere length impacts upon force creating capabilities of sarcomeres as per the properties of the length-tension curve, which indicates that sarcomeres extended past their optimum length display a reduction in force generating capacity (Figure 2).[63] This results in weaker sarcomeres (i.e. sarcomeres longer than optimal length) lengthening uncontrollably during eccentric contractions and eventually being excessively stretched so that passive structures take up most of the tension due to the reduction in actin-myosin overlap.[61] The consequential damage to individual sarcomeres as a result of this uncontrolled lengthening was termed 'sarcomere popping' and was proposed to be the first step towards macroscopic muscle damage such as muscle strain injury.[61]

Morgan's hypothesis[61] is not, however, universally accepted. It has been criticised because it is based upon single myofibril stretch studies performed *in-vitro* and *in-situ* which involve fibre strains not considered to be within the physiological range.[64] Butterfield[64] also argues that the expectation of unstable sarcomere lengthening on the descending limb of the length-tension curve is flawed given that the length-tension curve is determined under isometric conditions, whilst muscle lengthening occurs during dynamic eccentric contraction. Indeed, evidence exists of inherent stability of the length-tension curve during lengthening contraction[51, 65] which is thought to be attributable to the physiological characteristics of titin.[64] Further evidence[66] also argues against the assertion that sarcomeres at a longer length will lengthen uncontrollably when exposed to eccentric contraction. This still remains an area of great controversy.

Our current understanding of HSI suggests that high levels of eccentric force[38, 41-42, 46-47, 51-58] and muscle strain[49-51] are implicated in the aetiology of strain injury, however it is not clear whether accumulated microscopic muscle damage[61] or the presence of a single injurious

event[62] are most typically responsible for injury. Potentially any one of these factors may be the primary cause of HSI depending on the injurious activity type. For example, muscle strain may be the predominant mechanism in kicking HSI whereas forceful eccentric contractions may be the major mechanism in running HSI.

6. Anatomical factors that predispose hamstrings to strain injury

The predominately biarticular nature of the hamstrings allows for simultaneous extension at the hip and flexion at the knee during concentric contraction and significant lengthening during concurrent hip flexion and knee extension as seen in running[40] and kicking.[67] Such lengthening demands are thought to predispose the hamstrings to strain injury as the lengthening may exceed the mechanical limits of the muscle[41] or lead to the accumulation of microscopic muscle damage.[61, 68]

The two heads of the BF muscle are innervated by different nerve branches; BF_L by the tibial portion of the sciatic nerve and the BF short head (BF_S) by the common peroneal branch of the sciatic nerve, and it has been suggested that this dual innervation is a possible explanation for HSI because of the potential for uncoordinated contraction of the two heads of BF.[15, 32] This, however, remains unsubstantiated and is yet to be the focus of scientific investigation.

Another commonly held belief is that the hamstring muscles possess a high number of type II fibres[69] and this would be expected to increase the risk of strain injury given that fast glycolytic fibres have shown a greater propensity for muscle damage following eccentric contraction in animal models.[70] However, whilst early histochemical analysis suggested that the hamstrings consisted predominately of type II muscle fibres (58%)[69] a more recent study reported that only 51% of fibres in the BF_L were classified as fast twitch.[71] This discrepancy may be due in some part to the difference in the ages of the study participants. Subjects

from the study by Garrett and colleagues[69] ranged in age from 37-76 years whereas the hamstrings utilised in the study by Dahmane et al.[71] better reflected the ages seen in elite sport (17-40 years). Whilst fibre type distribution may be one factor that impacts upon the strain injury risk of muscles, it's role in HSI may have been overstated previously given the fact that the vastus lateralis has been shown to have a greater proportion of type II muscle fibres[72] compared to BF_L[71], yet the hamstrings are more commonly injured than the quadriceps.[7, 11-13, 73] In this example the differing lengthening demands of the muscles may have a greater influence over the propensity for strain injury than fibre type distribution.

Variations in muscle architecture may also explain high rates of muscle-specific HSI. For example, BF_S possesses much longer fascicles but a much smaller physiological cross sectional area compared to BF_L[74] and this variation of architecture may predispose the BF, particularly the long head, to high rates of strain injury. Longer fascicles allow for greater muscle extensibility[64] and reduce the risk of over lengthening during eccentric contraction.[68] However BF_L, which undergoes the greatest lengthening of all the hamstrings during sprinting,[43] has shorter fascicles compared to BF_S and this may predispose the BF_L to repetitive over lengthening and accumulated muscle damage.[61, 68] Consideration must be given to the fact that the available hamstring architecture data from this cadaveric study[74] has been performed on muscles from donors aged 68-88 years and the architectural characteristics of these muscles may differ markedly from younger, athletic populations.

The degree of anterior pelvic tilt may also impact upon HSI risk given that the common origin for the long hamstrings, the ischial tuberosity,[75] is found on the posterior aspect of the pelvis. As a result, excessive anterior pelvic tilt will place the hamstring muscle group at longer lengths[76] and some have proposed that this may increase the risk of strain injury.[15, 77]

Whilst some commonly held beliefs relating to HSI risk, such as the importance of fibre type distribution, may now be questioned, the importance of structure still remains crucial to hamstring muscle function. As such, the anatomy of the hamstrings most likely contributes to its high propensity to injury, however each of the aforementioned anatomical variables may increase the risk of injury via discrete mechanisms. An understanding of each of these anatomical factors must also be intepreted with an understanding of the causes of HSI presented in Chapter 5.

7. Risk factors for hamstring strain injury

A number of unalterable and alterable risk factors have been proposed for HSI, including, but not limited to, increasing age,[6, 15-16, 20, 73, 78-80] previous injury,[6, 20, 73, 79-80] ethnicity,[9, 15, 20] strength imbalances,[5, 32, 81-88] extremes of flexibility[77, 89-93] and fatigue.[32, 50, 94-95] This chapter details those prospective studies which have identified unalterable and alterable factors that elevate the risk of an athlete sustaining a HSI. In addition, both intervention studies and randomised controlled trials (RCT) aimed at preventing HSI are examined to provide a thorough understanding of the alterable causative factors responsible for HSI.

7.1 Unalterable risk factors

7.1.1 Age

Increasing age has been identified by a number of investigators as an independent risk factor for HSI in Australian footballers[6, 20, 78-79] and soccer players.[15-16, 73, 80] Australian footballers older than 23[79] or 24 years[6] and soccer players older than 23 years[15] are at an elevated risk of HSI, with the odds ratios as high as 4.4 (95%CI: 1.6 -12.5) for the older athlete.[6] Furthermore, each year of age has been reported to increase the risk of sustaining a HSI by as much as 1.3 (95%CI: 1.1-1.5) fold in Australian footballers[20] and by 1.8 (95%CI: 1.2-2.7) fold in soccer players.[16] Importantly, all studies which report age as a significant risk factor have utilised regression or multivariate analysis to conclude that increasing age

increases the risk of sustaining a HSI independently of confounding variables such as previous injury.[6, 15-16, 20, 73, 78-80]

One attempt to identify age related changes that lead to an increased risk of HSI in Australian football identified increased body weight and reduced hip flexor flexibility as predictors of HSI in athletes aged 25 years or older.[78] Despite achieving significance, the increase in risk was moderate with risk ratios of 1.07 (95%CI: 1.0-1.2) and 1.15 (95%CI: 1.0-1.3) respectively.[78] Other suggestions are that decreases in muscle mass and strength due to ageing could partially explain the increased risk of HSI in the older athlete,[6] however evidence to support this hypothesis[96-97] comes from cross sectional studies that included non-elite, non-athletic cohorts of significantly greater age ranges than are observed in elite sport. It is, in our view, particularly unlikely that athletes aged 24-30 are weaker or have less muscle mass than their 18-23 year old counterparts. Other hypotheses are age-related changes to muscle structure[6] and entrapment of L5/S1 nerve root due to hypertrophy of the lumbosacral ligament,[98] however more evidence is required to test these hypotheses.

Despite the consistent identification of age as a risk for HSI, no convincing explanation has been given as to why athletes older than 24 years are at significantly greater risk than younger athletes. Ideally long term longitudinal studies are required to determine the physiological changes that occur across an athlete's career to further elucidate the relationship between increasing age and increased HSI risk.

7.1.2 Previous injury

A number of studies have indicated that Australian footballers with previous HSI are at an elevated risk of sustaining a future HSI.[6, 20, 79] HSI from the previous season was also a significant risk factor for hamstring injury in elite professional soccer players[80] and has been reported to increase the risk of future injury as much as 11.6 (95%CI: 3.5-39.0) fold.[73]

Following a HSI the primary goal must be to identify the predisposing factor responsible for the injury, which then should be a target for rehabilitation and/or intervention.[34] If this predisposing factor is not ameliorated the athlete will remain at an elevated risk of future HSI despite sufficient convalescence. Additionally, a number of suggested post-HSI maladaptations are thought to contribute to the increased risk of future injury. These maladaptations include the formation of non-functional scar tissue[34] that is associated with an alteration in muscle tissue lengthening mechanics,[99] reduced flexibility,[90-91, 93] persistent reductions in eccentric strength,[54, 82, 91, 100] long term atrophy of the injured muscle,[101] alterations in the angle of peak knee flexor torque[68] and alterations in lower limb biomechanics.[20] Given the retrospective nature of these observations[34, 54, 68, 82, 90, 91, 93, 99, 100, 101] it is difficult to ascertain if these traits are the cause of or the result of previous injury, however it is accepted that modifications (or maladaptations) do occur following HSI.[34] From the available literature, persistent reductions in eccentric strength,[54, 82, 91, 100] the alterations in the angle of peak knee flexor torque[61, 68, 87] and reduced flexibility[90-91, 93] have been examined most extensively in the literature and will be discussed in the following sections. The emerging evidence relating to the impact of scar tissue on muscle tissue lengthening mechanics,[99] however, is also worthy of further discussion. Findings from Silder and colleagues[99] suggest that previous hamstring injury at the muscle-tendon junction results in a proliferation of scar tissue in this region and ultimately leads to adjacent muscle fibres experiencing greater strain during eccentric contraction. Such an adaptation to muscle tissue lengthening mechanics following injury would imply a greater risk of re-injury given the association between higher levels of muscle fibre strain and susceptibility to muscle damage.[55]

The high rate of recurrence and the elevated risk associated with previous injury highlights the importance of preventing first-time HSI and avoiding the vicious injury-reinjury cycle.[34]

Furthermore, whilst previous injury has been identified as elevating the risk of future injury, much work still needs to be done to determine what maladaptations are responsible for this increased risk.

7.1.3 Ethnicity

Three independent studies have identified Aboriginal[20] and black African or Caribbean[9, 15] ethnicity as risk factors for HSI, however only one study reported the risk to be significantly increased (Odds ratio (OR)=11.2; 95%CI: 2.1-62.5).[20] Both high proportions of type II fibres[70, 102] and excessive anterior pelvic tilt[15, 77] have been suggested as factors in the incidence of HSI in these populations, however, these are not substantiated and more objective evidence is required to determine how ethnicity impacts upon HSI risk.

7.2 Alterable risk factors

7.2.1 Strength imbalances

Strength imbalances of the hamstring muscle group have long been suggested as causes of HSI.[81] For the purposes of this review a strength imbalance can include any of the following; knee flexor weakness, bilateral knee flexor strength asymmetry and low ratios of knee flexor to knee extensor strength, otherwise known as hamstring to quadriceps or H:Q ratios.

7.2.1.1 Strength

Experimental data from animal models has shown that fully stimulated muscles are able to withstand greater amounts of stress before stretch-induced failure compared with partially activated muscles.[49] The authors postulated that stronger muscles would provide greater protection from strain injury and that muscle weakness may be a risk factor for muscle strain injury,[49] however the evidence linking hamstring weakness to HSI in humans is mixed.[5, 84, 86] Whilst one prospective study has found that subsequently injured Australian footballers demonstrated lower peak concentric hamstring torque in preseason isokinetic testing,[84] this finding was not replicated in a larger but otherwise similar study a year later.[5] Prospective data on sprinters supports the findings of Orchard and colleagues[84] as isometric knee

flexion strength relative to body weight was significantly lower in subsequently injured limbs.[86]

7.2.1.2 Bilateral asymmetry

Testing to assess unilateral hamstring strength allows for the determination of a weaker limb, if one exists. It has been proposed that a significantly weaker hamstring on one leg compared to the contralateral leg, termed hamstring bilateral asymmetry, may predispose the weaker hamstring to an elevated risk of injury.[103] The use of a between leg comparison of strength may be a more meaningful marker of weakness for individuals than a comparison with a group average or standardised score.

Early studies suggested that between leg hamstring strength asymmetry of greater than 10% was a predictor of hamstring injury in American footballers and track and field athletes.[32, 81] Later, elite Australian footballers with a bilateral asymmetry of 8% or more were found to have an increased risk of HSI[84] whilst soccer players with an asymmetry of more than 15% were at an increased risk.[83] It should be noted, however, that some authors have found no predictive power of bilateral strength imbalances.[5, 87]

Whilst some disagreement exists in the literature to date, a number of studies have identified that bilateral hamstring asymmetry leads to an increased risk of sustaining a HSI in a number of athletic cohorts.[32, 81, 83-85] Further exploration of imbalances between the hamstrings and other muscles of the hip joint is warranted as this may impact upon hamstring loading particularly during the terminal swing phase of running. Any alterations in running biomechanics associated with hamstring strength asymmetry should also be explored to determine if hamstring loading is affected as a result of imbalance.

7.2.1.3 Hamstring:Quadriceps strength ratio

A lower H:Q ratio suggests a relatively poor capacity for the hamstrings to act as 'brakes' at the flexing hip and extending knee joints during the terminal swing phase of running. Thus forceful contraction of the quadriceps, as occurs during the early swing phase of gait, has the potential to produce angular momentum at the knee joint that exceeds the mechanical limits of the hamstring.[104] Initial research[32, 81, 84] focused on comparisons of concentric strength imbalances across the knee joint ($H:Q_{conv}$) but has been criticised as it neglects the functional role of the hamstrings during the terminal swing phase of gait, that of a forceful eccentric contraction.[38, 41, 43, 45] More recently the comparison of eccentric hamstring to concentric quadriceps strength, known as a functional strength ratio ($H:Q_{func}$), has been suggested[104] and popularised.[5, 82-83, 85, 87]

One of the earliest studies to examine the relationship between $H:Q_{conv}$ ratios and future injury risk found that American footballers with a $H:Q_{conv}$ ratio of less than 0.50 were at an elevated risk of HSI.[32, 81] A later small-scale study in Australian footballers found that a $H:Q_{conv}$ of less than 0.61 put an individual at a substantially increased risk of HSI[84] whilst a larger study performed only a year later was unable to find an association between $H:Q_{conv}$ or $H:Q_{func}$ ratio and future HSI in Australian footballers.[5] These studies employed athletes at different levels of expertise and professionalism and employed different methodologies, all of which make comparison of the findings difficult. With respect to sprinters, prospective observations found that neither $H:Q_{conv}$ or $H:Q_{funct}$ displayed any significant differences between athletes who did or did not suffer a HSI.[87] Whilst Cox regression analysis did determine that a $H:Q_{conv}$ ratio below 0.60 lead to an increase in the risk of sustaining a HSI by 17.4 (95%CI: 1.3-231.4) fold[87] the sample size of the injured group (*n*=8) should have precluded use of this statistical method. Other prospective observations have found that pre-season $H:Q_{funct}$[85] and an isometric $H:Q_{conv}$[86] were significantly lower in the subsequently injured limbs of sprinters.

Many of these studies are limited due to their small sample sizes which makes detecting small associations between H:Q and HSI risk difficult.[105] The most powerful study to have examined the association between H:Q ratio and HSI (*n*=462) found that uncorrected strength imbalances in soccer players, which included a $H:Q_{conv}$ below 0.45-0.47 (exact cut-off depends on dynamometer brand used) and a $H:Q_{funct}$ below 0.80-0.89 were associated with a significantly greater frequency of HSI compared to athletes without strength imbalances.[83] Furthermore, the correction of strength imbalances, including normalising H:Q ratios, led to a significant reduction in HSI frequency compared to athletes with uncorrected imbalances (see Chapter 7.3.2).[83] These findings provide the strongest evidence available that sufficient H:Q ratios protect athletes from future HSI.

7.2.1.4 Angle of peak knee flexion torque

Athletes with a greater knee angle at peak concentric knee flexion torque (those who produce peak knee flexor torque at shorter muscle lengths) are proposed to be at greater risk of HSI.[68] The hamstrings in these individuals would be expected to work on the descending limb of the length-tension relationship across a greater range of motion, leaving them more prone to damage.[106]

Athletes with a history of unilateral hamstring injury display peak knee flexion torque at a greater degree of knee flexion on their injured limb compared to the uninjured limb (Figure 3),[68] however, it is not known if this is the cause of or the result of previous injury given the retrospective nature of these observations. In an attempt to determine a relationship between the angle of peak torque and future HSI occurrence, a recent prospective study in elite and sub-elite Japanese sprinters was performed.[87] This investigation found no association between the angle of peak knee flexor torque and subsequent HSI during the competitive season.[87] Currently the evidence pertaining to the usefulness of the angle of peak knee flexor torque to predict previous or future HSI is too sparse to draw any firm inferences and more work in this area is required.

7.2.2 Flexibility

Flexibility training has traditionally been proposed as a key component of injury prevention in athletes despite a lack of convincing prospective scientific evidence.[95, 107] It is proposed that greater flexibility may reduce the risk of strain injury due to a greater ability of the passive components of the muscle-tendon unit to absorb energy as a result of greater compliance[107-108] although this point is disputed in the literature.[109]

Prospective studies in both American[81] and Australian footballers[6, 84, 108] have found no relationship between the hamstring flexibility from the sit-and-reach or toe-touch test and future HSI risk. In contrast to popular belief, Australian footballers with a history of HSI, who displayed greater sit-and-reach flexibility were actually more likely to sustain a recurrent HSI.[6] Furthermore, poor hamstring flexibility, as assessed via an active or passive knee extension test or a straight leg raise, did not increase the risk of HSI in Australian footballers,[110] soccer players[73] or sprinters.[87] In contrast, some studies have reported relationships between flexibility and hamstring injury.[16, 89, 92] A study in elite soccer players found that hamstring flexibility of less than 90 degrees in a passive straight leg raise correlated significantly with future HSI.[92] Further studies also identified reduced hamstring flexibility as a significant independent risk factor for HSI in elite soccer players.[16, 89]

Whilst the weight of evidence suggests that there is no protective benefit of greater hamstring flexibility on HSI risk, methodological flaws exist with the measurement techniques employed. Foreman and colleagues[111] suggest that no gold standard measurement for flexibility has been established and that tests of hamstring length such as the sit and reach, straight leg-raise and toe touch test can be inaccurate if they do not allow for stabilisation at the hip and lumbar spine.

Future studies should employ more objective measures of flexibility such as the method used by Arnason and colleagues[73] which involves a tension meter to determine the limits of range of motion. This is as opposed to the subjective assessment of the end of range of motion by the investigator or subject which may display good levels of inter- and intra-tester reliability but may suffer with respect to ecological validity. Even if such subjective measurements are reproducible there is no means of determining whether a subject has been stretched to their maximal range of motion. The use of a more objective approach would be expected to improve the ecological validity of clinical flexibility tests given that a set level of passive tension is defined as the end of range for all subjects.

7.2.3 Fatigue

Fatigue and its associated performance decrements have often been suggested as causative factors for injury.[32, 50, 95] Indeed, studies of injury incidence have shown that HSI occur at a greater rate in the latter stages of competitive matches and training.[9, 13, 15, 112-113]

The effect of fatigue on muscle lengthening properties was initially examined in a laboratory setting. In these experiments, muscles that were pre-fatigued via electrical stimulation absorbed less energy before failure when compared with unfatigued muscles.[50] Fatigued and control muscles still failed at the same length, indicating that a fatigued muscle may be more likely to suffer a strain injury due to a reduced capacity to resist over-lengthening.[50]

With respect to human muscle function, one group has shown that fatigue of the hamstrings induced by repeated dynamic efforts leads to an increase in the amount of knee extension observed during the terminal swing phase of running.[114] This increase in knee extension would be expected to lead to a greater strain on the hamstrings during the terminal swing phase of gait,[43] however, it was matched by a reduction in hip flexion.[114] These alterations in knee and hip joint positions suggest that fatigue from dynamic exercise may lead to alterations in proprioception, a phenomenon which has been reported in response to other

experimental models of knee flexor fatigue.[115] In these trials, isokinetic exercise which induced a 30% reduction in knee flexor MVC force resulted in a reduction in proprioceptive ability, whereby hamstring length was under-estimated in a fatigued state.[115] This could lead to the perception of normal hamstring muscle lengths during running whilst in reality repeated over-lengthening of the hamstrings is occurring. Such deficits in proprioception when fatigued may elevate the risk of HSI given the assertions made by Morgan[61] that continual over-lengthening would lead to microscopic muscle damage that may accumulate to become macroscopic damage (i.e. strain injury).

More recent work has also shown that intermittent running designed to mimic the demands of competitive soccer significantly reduces eccentric hamstring torque with little or no impact on concentric knee flexion or extension strength.[116-117] In our own unpublished work we have found marked variability in the loss of eccentric hamstring strength. Those who exhibit greater levels of preferential eccentric hamstring fatigue would be expected to be at a greater risk of a HSI with prolonged activity given the link between eccentric weakness and HSI risk.[83, 85]

Other potential factors linking fatigue with elevated risk of muscle strain injuries, such as altered technique, reductions in concentration and other intrinsic physiological changes such as reduced coordination of muscle recruitment patterns, have been suggested[94] but are yet to be rigorously tested.

7.3 Addressing risk factors to reduce the risk of hamstring strain injuries

Intervention studies and RCTs are important in determining if reported risk factors are indeed causative factors in injury aetiology. These study designs can determine whether interventions intended to improve purported causative factors result in reductions in the risk

of sustaining a HSI. In fact risk factors cannot be considered causative unless there is a reduction in the risk of sustaining a HSI following an intervention aimed at ameliorating them.

7.3.1 Eccentric strength training

7.3.1.1 Nordic hamstring exercise

Two RCTs[118, 119] and one intervention study[57] have examined the benefits of Nordic hamstring exercise (NHE) on HSI rates. The NHE is a body weight exercise that requires athletes to begin in a kneeling position and to gradually lower their upper bodies towards the ground by extending at the knee while contracting the knee flexors eccentrically to slow the descent. During the exercise the athlete's ankles are typically held down by a partner.[120] The NHE has been shown to increase eccentric hamstring torque[120] and shift the T-JA curve of the hamstrings to longer muscle lengths[121] and both are suggested mechanisms by which NHE may reduce HSI rates.

The implementation of NHEs failed to reduce rates of HSI in cohorts of amateur Australian footballers[119] and professional soccer players,[118] however compliance with both intervention programs was extremely low. Gabbe and colleagues[119] reported that approximately half of all participants allocated to their intervention group did not complete the second training session and that fewer than 10% completed the five planned sessions. Engebretsen and colleagues[118] also reported that only 21% of players performed 20 or more of 30 planned sessions of NHEs. Furthermore, the use of extremely high volume and low frequency (once per two to three weeks) hamstring training in one of these interventions[119] was inconsistent with conventional conditioning practices.[120]

In contrast, elite soccer teams who chose to implement the NHE as part of their pre-season and in-season conditioning programs displayed a 65% reduction in HSI compared to teams that did not.[57] Furthermore, the teams that utilised the intervention displayed significantly lower rates and severity of HSI compared with previous seasons.[57] This study was,

however, limited by a non-randomised approach as individual teams decided if they were to participate in the intervention. Interestingly the implementation of NHC did not reduce the rate of HSI recurrence.[57]

7.3.1.2 Flywheel training

Training on a flywheel ergometer,[122] which is designed to augment the amount of eccentric torque required during the performance of a lying leg curl, has been reported to increase eccentric hamstring strength and reduce HSI rates.[58] A small scale RCT performed on two elite soccer teams (*n*=30 players) found that flywheel hamstring training in the pre-season significantly reduced the number of HSI compared to the control group. However, the control group displayed a remarkably high rate of HSI incidence (66%)[58] and this potentially diminishes the significance of these findings.[123]

7.3.1.3 Considerations for exercise selection

At present the literature pertaining to the benefits of eccentric strength training on reducing HSI incidence is inconclusive.[123] Whilst a number of factors, including a lack of compliance to eccentric strength training interventions,[118-119] may contribute to this, exercise selection may also be a factor. The SM and ST reportedly exhibit greater activation levels at shorter muscle lengths, whereas the BF_L is most powerfully activated at longer lengths during isokinetic knee flexion.[124]

Magnetic resonance imaging has recently revealed that the BF_L and SM muscles were significantly less active than the ST and gracilis during a heavily loaded eccentric leg curl, which mimics the knee range of motion and hamstring lengths experienced in the NHE and flywheel training.[125] It is therefore possible that these exercises may be sub-optimal in bringing about adaptation in the BF_L, the muscle most frequently injured.[22-24] Exercises that

better target the BF_L such as the stiff-legged deadlift[126] may prove more effective in hamstring injury prevention than those that have so far been employed in RCTs.

7.3.2 Strength imbalance correction

A large scale cohort study (*n*=462) of isokinetic hamstring strength in elite soccer players found that correction of strength deficits (either concentric or eccentric asymmetries or low H:Q ratios) lead to similar HSI rates compared to athletes without strength deficits.[83] Participants who had strength deficits but did not undergo isokinetic rehabilitation or who did undergo isokinetic rehabilitation but did not perform post-intervention testing showed significantly higher rates of HSI.[83] This study is of great significance as it employed one of the largest sample sizes of any HSI prevention study and suggests that a reduction in the risk of HSI can be achieved via the detection and subsequent correction of isokinetic strength deficits.

7.3.3 Flexibility training

An intervention study performed on elite soccer players found that a prescribed contract-relax flexibility training protocol performed during the warm up did not reduce the rate of HSI compared with teams that did not incorporate flexibility training.[57] Similarly a RCT involving recreational level runners, who completed a 16-week unsupervised intervention consisting of warm-up and cool-down procedures and stretching showed no difference in the rate of HSI compared to a control group.[127] Consideration must, however, be given to the potential that the intervention may have been inadequate to increase flexibility due to the brief duration of stretching exercises (10 seconds).[123] These finding are not totally unexpected given the lack of evidence for poor flexibility being a risk factor for HSI (see Chapter 7.2.2). However further work needs to be performed, with greater control over other confounding variables such as aerobic and eccentric hamstring conditioning to fully elucidate the effect of flexibility training on HSI rates.

8. Hamstring strain recurrences and neuromuscular inhibition

Whilst there is an extensive list of risk factors for HSI which have been examined through a number of different methodological designs, epidemiological data suggest that first-time and recurrent HSI rates in sport are not in decline.[1-2, 7, 9, 13, 15, 29] This suggests that our current understanding of what increases the risk of a future HSI has not accounted for all contributing factors or that we are unable to resolve previously identified factors effectively. Despite previous HSI being consistently identified as one of the primary risk factors for a future HSI[6, 20, 73, 79-80] maladaptation associated with HSI, particularly nervous system function, has been largely overlooked. Potentially, a number of reported maladaptations associated with prior HSI may be explained by a common neurological mechanism in response to previous injury.

Weakness after painful musculoskeletal injury is typically mediated by both muscular and neural adaptations. For example, following traumatic knee injuries involving ACL ruptures maximal voluntary activation of the quadriceps is significantly reduced, even years after the injury occurred[128-129] and despite restoration of knee stability.[129] In the case of HSI, however, little attention has been paid to the possibility that prolonged deficits in activation contribute to the high injury recurrence rate. This is surprising given that the torque-velocity relationships of previously injured hamstrings are characteristic of heightened neuromuscular inhibition in the sense that they show greater deficits in eccentric than concentric strength.[54, 91, 100] Prolonged neuromuscular inhibition at long muscle lengths after HSI could potentially account for observations of preferentially eccentric weakness,[54, 91, 100] persistent atrophy of the previously injured muscles[101] and alterations in the angle of peak knee flexor torque,[68] all of which are purported risk factors for HSI and have been observed in athletes following ‘successful’ rehabilitation and the return to full competition and training.

A reduction in the capacity of the nervous system to activate injured muscles presumably constitutes a strategy to unload damaged tissues and thereby reduce pain in the acute recovery period. As the greatest pain after hamstring strain is typically felt at longer muscle lengths, it is not surprising that there is now evidence for a length-specific reduction in hamstring activation.[130] Inhibition, particularly during eccentric actions and at longer muscle lengths, may also impede the rehabilitation process by limiting adaptations within the previously injured muscle(s).

The early and middle stages of treatment for HSI are characterised by the avoidance of excessive stretching to prevent further scar formation and submaximal exercises performed through a limited range of motion and with hip joint movements restrained primarily to the frontal plane.[131] Thus, by the time athletes are in the late stages of rehabilitation, their hamstring muscles might be expected to have shed in-series sarcomeres[132] and to have atrophied considerably. Having fewer in-series sarcomeres would be expected to shift the peak of the knee flexor torque-joint angle curve to shorter muscle lengths and create even greater weakness at longer lengths than atrophy alone.[68] Such hamstring function is detrimental as running requires strength at relatively long muscle lengths to decelerate hip flexion and knee extension during terminal swing.[41-43]

The return to running at progressively faster speeds and the use of more intense strengthening exercises later in rehabilitation should increase exposure to forceful eccentric actions at relatively long muscle lengths[38, 40-43] and might therefore be expected to return muscles to their original size and fascicles to their pre-injury lengths.[133] However, any lingering neuromuscular inhibition would spare the previously injured hamstring muscle(s) from significant activation during eccentric actions at long length and would therefore limit or prevent hypertrophy and sarcomerogenisis. Evidence of persistent atrophy in the previously injured BF_L with simultaneous compensatory hypertrophy of the uninjured BF_S in recreational

level athletes, 5-23 months after HSI and after a full return to training and competition[101] is consistent with the hypothesis of prolonged muscle-specific inhibition.

Additional investigation is required to confirm whether previously injured athletes display significantly greater levels of neuromuscular inhibition within the previously injured leg compared to their contralateral uninjured limb and whether inhibition is confined specifically to the injured muscle. Ultimately to identify neuromuscular inhibition as a causative factor in recurrent HSI, prospective studies and RCTs need to be performed to determine if inhibition following HSI results in an increased risk of re-injury and whether ameliorating this neurological deficit reduces the incidence of recurrent HSI. Techniques such as surface electromyography,[124, 130, 134-136] twitch interpolation[137-138] and electrical stimulation[135, 139-140] have been used previously to assess voluntary muscle activation and all should be considered for future work in this area. Further work also needs to be carried out to rigorously determine the full extent of physiological maladaptation associated with altered neural function following HSI.

H. Conclusion

9. Conclusion

Hamstring strain injuries (HSI) remain the predominate injury in a number of sports despite concerted efforts to expand scientific knowledge. Additionally, HSI have shown a high rate of recurrence and the capacity to impact negatively on individual and team performance and financial viability of elite sports clubs. Whilst it is widely acknowledged that the causes of HSI are multifactoral, the interaction between these factors is often overlooked. This review has integrated the role of the hamstrings in running, the specifics of hamstring anatomy and reported risk factors and interventions for HSI to better understand the causes of this injury.

Sports medicine practitioners and sports injury researchers alike need to appreciate the complex nature of HSI and understand that no one single approach can be considered the gold standard for HSI prevention or rehabilitation. For example, a focus solely on markers of performance (i.e. eccentric strength, flexibility) may neglect the important role that correct running technique may have on injury avoidance. The biomechanical demands of running, the anatomical organisation of the hamstrings and a range of unalterable and alterable risk factors, such as age, previous injury, ethnicity, strength imbalances, flexibility and fatigue have all been linked to HSI. All of these factors need to be considered, as does the interaction between these factors and the impact of reported interventions, by practitioners looking to prevent HSI. Furthermore, understanding of the exact causes of HSI remains elusive but muscle strain, high force eccentric contraction, accumulated muscle damage and/or a single injurious event may all potentially play a role and all should be considering when developing HSI preventative strategies.

Further to this, more work needs to be carried out in the area of assessing maladaptation associated with previous HSI. Whilst it is commonly known that previous HSI is the primary risk factor for future injury very little is known about the maladaptations associated with a

previous insult. Understanding only that previous injury elevates the risk of injury without an understanding as to why, gives little insight into how HSI should be successfully rehabilitated. We propose a novel integrated framework of how previous injury may lead to persistent neuromuscular inhibition which could conceivably result in a cascade of maladaptations that elevate the risk of future HSI. This area should be a focus of future research given the high levels of HSI recurrence for a number of years in many sports.

I. Footnotes

None

J. Reference list

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K. Tables

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