The biomechanical determinants of sports related groin pain in athletes.

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Thesis submission for Doctor of Philosophy

The biomechanical determinants of sports related groin pain in athletes.

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Submitted in partial fulfilment of the requirements of the Degrees of Doctor of Philosophy

Statement of originality

I, Paulina Maria Kloskowska, confirm that the research included within this thesis is my own work or that where it has been carried out in collaboration with, or supported by others, that this is duly acknowledged below and my contribution indicated. Previously published material is also acknowledged below.

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Collaborators

- Saracens Rugby Football Club medical team (in particular, Mr Joe Collins, the Head of Medical Team)
- Reading Football Club medical tem (in particular Mr Jonathan Alty)
- Queen Mary University of London, Sports and Exercise Medicine academic team, including Dr Christian Barton, Dr Peter Malliaras, Mrs Claire Small, Dr Richard Twycross Lewis.
- Queen Mary University of London, Sports and Exercise Medicine intercalated BSc students: Mr Oliver Rabie, Ms Charlotte Hillary, Ms Laura Middleton, Mr Waleed Moussa and Mr Alexander Osborne

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Abstract

Introduction

Sports related groin pain (SRGP) is common, debilitating and often recurrent. Rehabilitation that addresses strength and flexibility deficits has only moderate effects. Recurrence of SRGP remains high, suggesting that deficits remain after apparently successful rehabilitation. The aims of this thesis were to inform best practice by (i) systematically reviewing the literature on biomechanical factors associated with SRGP (ii) investigating muscle activation and movement patterns associated with SRGP in both professional and amateur athletes; (iii) investigating muscle activation and movement patterns immediately after groin injury alongside their response to standard rehabilitation.

Methods

A systematic review with meta-analysis was completed. 84 athletes from four sports (56 professional and 28 amateur) were recruited and clinically assessed. Hip joint kinematics and surface electromyography of gluteus medius (GM) and adductor longus (AL) muscles were measured while performing selected manoeuvres. A further 5 athletes had serial measures during traditional rehabilitation from acute injury.

Results

The review found strong evidence for decreased adductor flexibility as a risk factor; and decreased adductor strength and external rotation range of movement being associated with SRGP. The GM:AL ratio in injured professionals was increased due to reduced AL activation, a decreased GM:AL ratio was found in amateurs due to a decrease of GM activation. In injured professionals hip kinematic change matched the sEMG findings (increased abduction), whereas no consistent pattern was observed in amateurs. Longitudinal study participants

improved clinically after groin injury, but the muscle activation and movement patterns did not alter.

Conclusion

These studies identified clear muscle activation differences that extend existing the literature while the kinematic changes are novel. Further, participation level and sports-specific subgroups had not previously been identified but are clearly evident. Published guidelines require amendment, while clinical innovation that addresses sub-group specific biomechanical factors in rehabilitation programmes may inform prevention, improve outcome and certainly warrant further research.

Table of contents

Glossary

2D / 3D	Two dimensional / Three dimensional
Add	Adduction
Ag / AgCl	Silver / Silver-chloride
AL	Adductor longus muscle
ANOVA	Analysis of variance
ASIS	Anterior superior iliac spine
ASLR	Active straight leg raise
CI	Confidence interval
CODA	Cartesian Optoelectronic Dynamic Anthropometer
df	Degrees of freedom
FABER	Flexion-abduction-external rotation test
FADIR	Flexion-adduction-internal rotation test
FAI	Femoroacetabular impingement
GM	Gluteus medius muscle
HD-EMG	High density array electromyography
HPL	Human Performance Laboratory
HQS	High quality study
ICC	Intraclass correlation coefficient
MFAP	Muscle fibre action potential
MFCV	Muscle fibre conduction velocity
mocap	Motion capture system
MRI	Magnetic resonance imaging
MU	Muscle unit
MUAP	Muscle unit action potential
MVC	Maximal voluntary contraction
Pec	Pectoralis muscle
RF	Rectus femoris muscle
ROM	Range of movement
SD	Standard deviation
SE	Standard error
sEMG	Surface electromyography
SENIAM	Surface EMG for Non-Invasive Assessment of Muscles
SHF	Standing hip flexion
SLS	Single leg squat
SMD	Standard mean difference
SRGP	Sports related groin pain
TrA	Transversus abdominis muscle
UEFA	The Union of European Football Associations
VAS	Visual analogue scale

Figures

Tables

Dedication

I dedicate this work to my parents and my sister, who have supported and encouraged me throughout the way, and gave me this wonderful opportunity for my development. I would not have achieved it without them.

This work is also dedicated to Professor Roger Woledge (1938–2015) who showed me the beauty of research, and was a constant source of inspiration, wisdom and support.

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Chapter 1: Introduction

Thesis overview

The Introductory chapter of the thesis provides the background to the problem of sports related groin pain (SRGP) and clarifies the rationale for subsequent experimental studies. The chapter presents the epidemiology of groin injuries, emphasising how common and troublesome these are in sports medicine. The second section focusses on biomechanical loading through the pelvis, which may be partly responsible for the susceptibility of multi-directional athletes to injuries in the area. I then summarise the most common pathologies in the groin region. An additional comprehensive section on the anatomy and pathology of the groin area can be found in Appendix 1. Further, the chapter describes the diagnostic terms as well as the diagnostic process in athletes with SRGP. The last part of the chapter gives an overview of treatment strategies. Overall, the introduction argues there is a lack of clarity about the potential importance of biomechanical factors such as muscle activation and movement patterns in the presentation of SRGP – therefore outlining the research space.

Chapter 2 is a systematic review which provides a detailed synthesis of published retrospective and prospective biomechanical factors associated with SRGP. As well as synthesising a substantial body of knowledge, this review further defines the research gap in the field and strengthens the rationale for the experimental work in the thesis. Simultaneously, the review is also an original piece of work, which separates, synthesises and analyses clinically applicable biomechanical variables and provides a clear, novel and useful message for clinicians.

Chapter 3 of the thesis details overt aims and hypotheses, in the form of null hypotheses. The aims are then addressed by separate chapters of the thesis. The alternative hypotheses are included in the introduction of each separate chapter.

Chapter 4 is a methods chapter which serves two purposes. Firstly, it describes my decision-making process when choosing the optimal data collection, data processing or analysis method by outlining the pros and cons of the most popular solutions and, in consequence, the rationale of the final choice. Secondly, this chapter clearly describes the chosen data collection, processing and analysis methods using worked example by explaining the step-by-step data processing methods and presenting graphic representations where relevant.

Chapter 5: This study addresses one of the aims of the thesis which is to establish in my hands, and unusually in this body of literature, the reliability of pelvic girdle surface electromyography and hip joint kinematic measurement during standing hip flexion (SHF) and single leg squat (SLS) manoeuvres.

Chapter 6; This observational study presents cross-sectional retrospective differences in gluteus medius versus adductor longus muscle activation ratios measured by surface electromyography, and hip joint kinematics measured by the CodaMotion capture system between healthy athletes and those suffering from SRGP. Measured groups are from four different sports disciplines and in two sports include both amateur and professional cohorts. The study procedure enabled us to explore associations between the movement and muscle activation patterns and SRGP. Separate analysis of the sports groups allowed for excluding potential bias of different biomechanical characteristics that may be associated with different sports and levels of participation. The results of the study showed clear biomechanical alterations between the healthy and injured athletes; therefore the level of play and sporting discipline, which may be likely to play a big role in the mechanism of injury, needs to be controlled when investigating the biomechanics of the athlete with injury.

Chapter 7: This longitudinal study presents the results of a repeated-measures study which investigated muscle activation and movement patterns during the time-course of rehabilitation from acute injury. This study was completed in order to discover whether the

biomechanical imbalances discovered in previous, observational study, have a primary or secondary character, and whether they may be improved by the current rehabilitation programmes.

The Last chapter of the thesis, chapter 8, discusses the study results in the context of other literature published in the field. It also considers the main study limitations, future directions and final conclusions.

Background

Sports related groin pain (SRGP) is a common entity, particularly in contact sports requiring repetitive high-speed kicking, twisting, pivoting and side-to-side movements (Lovell, 1995, Slavotinek et al., 2005); such as football, rugby and hockey. It is often associated with high recurrence and prolonged time away from sport (Weir et al., 2009), and together with hamstring injuries is responsible for the longest time away from playing sport (Orchard and Seward, 2011). The poor treatment outcomes and high recurrence rate make SRGP a key area for detailed study in order to deepen understanding of the pathophysiology and ultimately improve management.

The difficulties in diagnosis and treatment of SRGP result partly from a lack of consensus amongst researchers and clinicians in classification of the functional anatomy of the area and the large range of diagnostic terms used (Weir et al., 2015, Bradshaw et al., 2008). Patients suffering from SRGP are often 'diagnosed' with osteitis pubis, adductor tendinopathy, sportsman's hernia, Gilmore's groin or iliopsoas-, rectus abdominis- and adductor-related muscular disorders. Various underlying tissue pathologies are likely to coexist (Holmich, 2007) and there is a lack of clinical or imaging tests with high levels of sensitivity or specificity (Weir et al., 2015). SRGP was operationally defined in my thesis at the outset based on carefully considered pragmatic criteria, designed to exclude hip joint pathology and include over-

lapping soft tissue derangement and dysfunction diagnoses in the relevant anatomical area. No single published model was judged satisfactory at the time. The robustness of our approach can be inferred from the close agreement to an identical basket of pathologies defined at the recent Doha agreement (Weir et al., 2015).

SRGP usually has an insidious onset, but might also commence as an acute groin strain, which then becomes chronic (Fricker et al., 1991, Renstrom and Peterson, 1980). SRGP is a challenge to diagnose and, consequently, manage due to the complexity of pelvic girdle anatomy with multiple inter-dependent structures (Falvey et al., 2009) and the complex loading associated with the central-lateral load distribution from the spine to pelvis and hips (Dalstra and Huiskes, 1995, Dalstra et al., 1993). Simple length, strength, range and palpation tests seemed inadequate to fully elucidate diverse athlete presentations. Further, I felt there was a strong argument – particularly from a rehabilitation perspective and in view of the overalpping pathology - for *potentially* classifying patients by movement pattern rather than tissue diagnosis (Sahrmann, 2001). For this to be valid and for innovative treatments to be subsequently determined, there needed to be studies determining whether common movement patterns exist at all - and if so, of what nature, and in whom.

An international consensus on the taxonomy of groin injuries (Doha agreement) has been published very recently (Weir et al., 2015). It provided a useful tool for both clinicians and researchers, as it established terminological agreement and should enable groin pain research to move forward (Delahunt et al., 2015). Additionally, a number of recommendations regarding further research in the groin area have been identified, including specific recommendations considering epidemiology, risk factors, clinical examination, outcome measures, the role of imaging, and treatment of groin injuries (Weir et al., 2015, Delahunt et al., 2015) – all of which are covered in this introduction.

What is not present in the consensus, are consideration of the roles adverse movement patterns or muscle activation imbalance may play in SRGP occurrence and maintenance. This reflects a major gap in the literature, rather than being an error in the consensus state ment. What is perhaps more surprising is that consideration of sub-groups – and especially the sub-groups of elite vs non-elite athletes – is also not strongly represented in either the literature or the consensus. My thesis was positioned to address exactly those gaps, extending the promise of previous smaller-scale investigations of SRGP and recurrent hamstring injury by our research group, that have shown findings worthy of further more detailed exploration (Daly et al., 2015, Morrissey et al., 2012a). A central theme of my thesis was therefore exploration of whether biomechanical factors such as kinematically described movement patterns and electromyographically measured muscle imbalance might be relevant to SRGP presentation and management; and whether sports and participation-level specific groups may be identified using such methods.

Epidemiology

Epidemiology in football

The incidence and prevalence of groin pathologies have been reported in a variety of sports. One of the sports disciplines most commonly mentioned in relation to groin injuries is football. A prospective, high quality UEFA study investigated epidemiology of hip and groin injuries in 28 professional teams over 7 seasons (Werner et al., 2009). The prevalence for those types of injuries in football was reported to be between 12% and 16%. This is consistent with other authors reporting groin pathologies to account for 11% - 16% of all football injuries annually (Hagglund et al., 2006, Ekstrand and Gillquist, 1983, Hagglund et al., 2009, Hawkins and Fuller, 1999). The study of Ekstrand (Ekstrand and Hilding, 1999) on two professional football divisions (176 players) found groin injuries constitute 8% of all football injuries in 1995.

Epidemiology in rugby

Epidemiological report on injuries in professional English rugby union (Brooks et al., 2005a, Brooks et al., 2005b) has investigated the incidence and severity of rugby players during matches and training. The incidence of groin pathologies was 3.29 and 0.1 injuries per 1000 player-hours during training and matches, respectively. Groin injuries were also responsible for 101 (training) and 25 (match) absent days, and in consequence were ranked the fourth most severe injury among rugby players. O'Connor (O'Connor, 2004) reported a very high, 23% risk of sustaining groin injury in a prospective study on professional rugby players over a 2-year period. This rate is consistent with data from Gibbs (Gibbs, 1993).

Epidemiology in hockey

Groin injuries are also recognized as a major cause of morbidity in professional hockey players (Irshad et al., 2001). Emery et al. (Emery et al., 1999a) reported an incidence of 20 groin or abdominal injuries per 100 players annually and an increasing trend of these injuries incidence (increasing rate of 1.32 (95% confidence interval -0.58, 3.21) injuries/100 players/year). A study on sub-elite Swedish hockey players over four seasons, published by Pettersson (Pettersson and Lorentzon, 1993), found groin strains were the third most common injury, accounting for 8% of all injuries. Another epidemiological study by Agel et al. found hip and pelvis pathologies most common injury area during training among Collegiate Ice Hockey male players over a 16 years period (Agel et al., 2007).

Epidemiology in Australian Football league

The 2010 Injury Report in Australian Football, published by Orchard in 2011 (Orchard and Seward, 2011) (Table 1), shows that groin injury incidence failed to decrease between 2001 and 2010. Prevalence rate of this type of injuries, described as missed games per club per season, was the second highest value after hamstring strains. Groin injuries also had the

highest (recurrent groin injuries constituted 20% of all reported groin injuries) recurrence rate from all reported injuries.

Groin injuries	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	10yrA
Incidence	3.5	3.8	2.9	3.1	2.9	3.3	4.1	3.2	3.3	4.1	3.5
Prevalence	13.6	15.7	13.7	13.3	11.2	14	18	12.4	11.7	15.3	13.6
Severity	3.9	4.1	4.8	4.4	3.9	4.3	4.4	3.9	3.5	3.7	3.9
Recurrence rate	20%	23%	20%	24%	23%	28%	38%	23%	19%	20%	20%

Table 1: Key indicators for groin injuries over past ten years in Australian football (Orchard and Seward, 2011)

Epidemiology in other sports

Groin injuries were also reported in other sport disciplines. In Australian Cricket team they were the 5th most common injury (7% of all injuries in players per year) over two seasons (Orchard et al., 2002a). Swimmers, (mainly breaststroke discipline) also have a high incidence of groin injuries. Out of 296 surveyed breaststroke swimmers, 42.7% had missed at least one day of training due to groin problems (Grote, 2004).

Risk factors in groin pathologies

High incidence, prevalence and morbidity of SRGP has led to considerable interest among researchers to identify risk factors for this entity in order to better understand the mechanisms and facilitate prevention strategies. One systematic review (Hrysomallis, 2009) investigated the adductor muscle features associated with future SRGP and found low to moderate evidence of decreased adductor muscles strength and flexibility associated with increased risk for subsequent SRGP.

Two recently published systematic reviews identified factors consistently emerging from the literature as the risk factors for SRGP: previous adductor injury, decreased adductor strength and reduced sports-specific training (Maffey and Emery, 2007, Whittaker et al., 2015).

Previous adductor or abdominal injury was reported as a risk factor for groin strains in a number of separate studies (Emery and Meeuwisse, 2001, Engebretsen et al., 2010, Arnason, 2004, Gabbe et al., 2010). A high quality, prospective study of professional footballers (Arnason, 2004) reported that previous groin injury makes a player 7 times more susceptible to injury compared to uninjured player. These results are consistent with increased risk profile following exposure to musculo-skeletal injury in general (Hagglund et al., 2006, Maffey and Emery, 2007). Age was found to be another important risk factor in groin injuries (Arnason, 2004).

Emery et al. (Emery and Meeuwisse, 2001) reported much higher risk of groin injury associated with decreased number of sport-specific training hours. This might be related to other finding of Arnason et al. (Arnason, 2004), who reported, that injured players had significantly higher body fat compared to healthy cohort.

It is therefore important to note, that apart from factors more universally defining the risk factors for muscular injuries such as age and decreased number of training hours, there is also some evidence of simple biomechanical changes that precede pain onset. Those measures, as well as the more sophisticated so under-researched biomechanical investigations therefore warrant further study to improve our understanidng and management of SRGP.

Biomechanics of load distribution through the pelvis

The location between stable pelvis and mobile hips makes the groin area potentially very sensitive to biomechanical load or muscle activation imbalances transferred between the torso and lower limbs, regarding in particular the loading distribution.

Additionally to a specific 'sandwich-like' bony structure of the pelvis, with the extra strong cortical shell and relatively soft layer between external core, muscles have been reported to significantly influence loading in the area. The pelvic stress forces were reported to be

significantly decreased when applying muscle forces to the bony pelvic model (Dalstra and Huiskes, 1995). Specifically, the muscular force input in the purely bony model of the pelvis was reported to have a largely stabilising effect on the hip joint, compensating the hip joint reaction forces. This may suggest, that any weakness of any muscle attaching to the pelvis (particularly to pubic symphysis where the largest stresses occur) might increase the loading and lead to pathologies.

Sophisticated biomechanics of the pelvis and the stress distribution dependency of muscular support reported by Dalstra might suggest a particular susceptibility to pathologies in this area. Any muscular imbalance regarding increased or decreased forces acting on the pelvic-hip area may easily modify the stresses in pelvic region (particularly pubic bone) and lead to pathologies.

This is one of the reasons why the design of SRGP treatment strategies provides such a challenge for clinicians (Falvey et al., 2009). The simple model of pelvic and hip loading presented above becomes much more complex during dynamic, unilateral tasks, such as walking and jogging; even more in movements such as kicking, pivoting, cutting, side-to-side running or changing directions (Marshall et al., 2015), which are thought to increase the susceptibility for SRGP. In order to manage those high biomechanical demands, a high number of structures need to co-operate in an optimally balanced manner (Dalstra and Huiskes, 1995).

SRGP is often suggested to be an effect of overuse and overload of, initially, one single structure (Pizzari et al., 2008, Lynch and Renstrom, 1999, Marshall et al., 2015). Following this initial pathology, other structures may be exposed to relatively higher demands, leading to further overload, injury, pain or other mal-adaptations (Mueller and Maluf, 2002, Bussey, 2010, Renstrom and Peterson, 1980). This mechanism then results in multi-factorial, multi-structural symptoms, with a lack of clearly defined pathology within one structure and

biomechanical adaptive alterations regarding muscle features and movement patterns (Bussey, 2010).

A comprehensive and clinically applicable model of pelvis and hip loading was presented by Meyers et al. (Meyers et al., 2007), who focused on the pubic symphysis as a central point of the groin region and highlights the importance of the functional and biomechanical interactions between the abdominal and adductor muscles. The authors also provided insight into the biomechanical requirements of the pubic and groin area by presenting the directions of the forces acting on the pelvis and pubic symphysis (Figure 1, Figure 2).

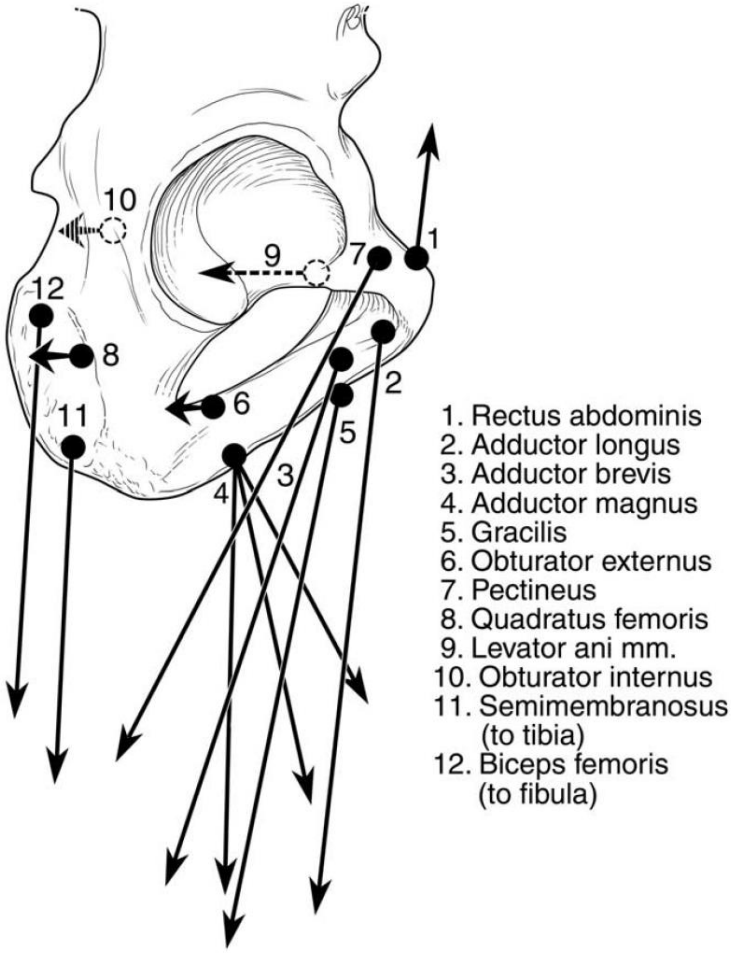


Figure 1: Medial view of the pelvis depicting the direction of forces acting on the pelvis and influencing pelvic tilt (Meyers et al., 2007).

According to Meyers, the initial cause of pain and pathology in the groin may be due to the multiple muscular insertions to pubic symphysis, which may likely provide imbalances forces applied to the pelvic and groin area.

Additionally to presenting an anatomical, biomechanical and functional approach, Meyers also provided some diagnostic advice. Using the pubic symphysis as the reference point, he

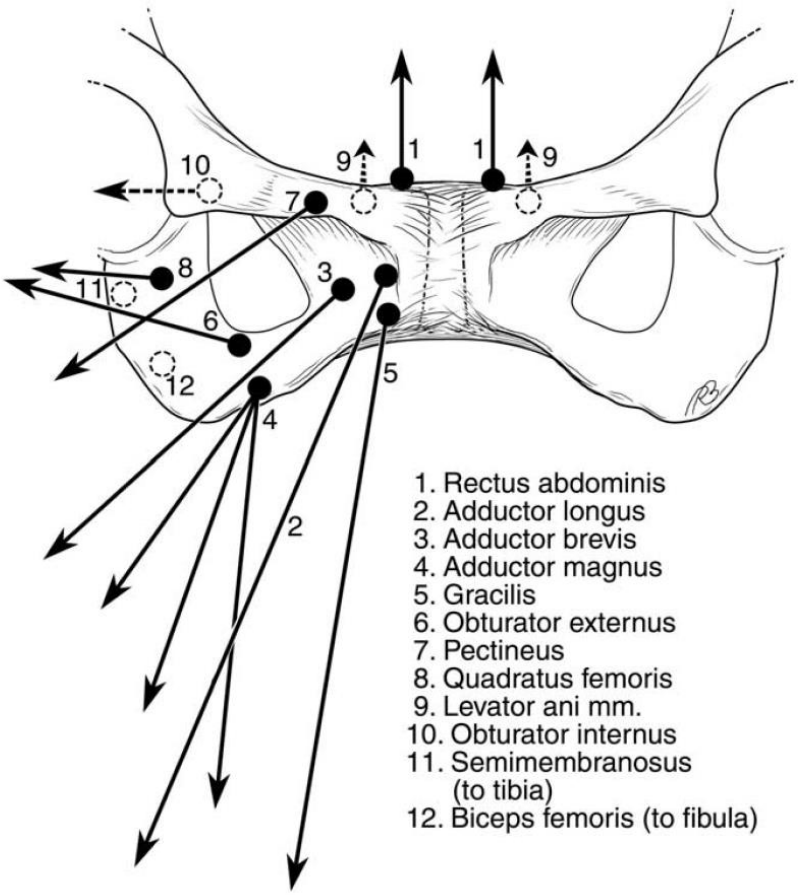


Figure 2: Anterior view of the pubic ramus with schematic depiction of the many forces acting on the pubic joint (Meyers et al., 2007).

distinguished clinical entities below as well as above the central point, which leads to focusing more on abdominal and "upper groin" area rather than adductors. This approach provides a description of pathologies occurring in abdominal area and is therefore an important overview of entities commonly confused with hernias. This review provides a very comprehensive

historical, anatomical, functional and biomechanical overview of SRGP but may be somewhat out-dated. The previously presented model by Falvey et al. (Falvey et al., 2009) includes all of those key elements, providing an additional clinical and diagnostic layer which I deemed to be a the optimal approach at the research design stage. Optimal but not ideal, as none of the published models had been subject to robust validation, nor were they based on a body of biomechanical literature on SRGP – thus indicating a clear research space for my studies.

Terminology in groin pain

There had been an on-going debate and lack of agreement among researchers and clinicians regarding the diagnosis and terminology of symptoms in the pelvis and groin areas. A number of terms had been used throughout (for example: osteitis pubis, Gilmore's groin, hockey's groin), with slightly different diagnostic criteria. This led to general confusion and misunderstanding, as well as preventing groin pain focused research moving the field forward due to the lack of similar criteria for included injured participants. However, a very recently published Doha agreement on the terminology and diagnosis in athletic groin pain provides clear and structured guidelines, which will allow the research in the area to move forward.

The published agreement was an effect of the meeting of the international group of experts in the groin pain research and treatment held in Doha, Qatar. Prior to the meeting, the organising committee has sent out two case studies of athletes presenting with groin symptoms to the group of 23 experts internationally in order to receive from them a preferred term for the diagnosis. The feedback from these studies served as the basis for one-day discussion regarding the terminology and diagnosis of groin symptoms. The effect of this discussion was then published as a consensus statement.

Groin pain in athletes was a preferred term to name the pain in the groin area, which was related to sports. This was then divided into three main sub-groups: (i) defined clinical entities

for groin pain (adductor-, iliopsoas-, inguinal- and pubic- related pathologies); (ii) hip joint related pathologies; (iii) other causes of groin pain (for example nerve entrapment or referred pain).

Throughout this thesis I am using the term sports-related groin pain (SRGP), which is consistent with the first sub-group defined by the group of experts, and includes all pathologies mentioned in the publication, and excludes the other causes of groin pain. At the time of designing the research protocol for the studies included in the thesis (2011) there was still little agreement of the terminology and diagnosis, therefore based on the available research at that time I chose to use self-selected inclusion and exclusion criteria. These, however, proved to be consistent with the Doha agreement.

A detailed overview of most common pathologies causing the groin pain in athletes can be found in Appendix 1 (p. 264).

Diagnosis

Physical assessment

SRGP is often associated with high recurrence and prolonged time away from sport (Weir et al., 2009) therefore early and accurate diagnosis is essential to prevent chronicity.

Groin pain gradually arising from musculo-skeletal origin is often suggested to be an effect of overuse and overload (Pizzari et al., 2008, Lynch and Renstrom, 1999). Moreover, there are often multiple diagnoses (Holmich, 2007, Nam and Brody, 2008, Caudill et al., 2008) and the pathologies may arise from poor biomechanics, pelvis instability and muscle imbalances in the groin area (Harris and Murray, 1974, Mandelbaum and Mora, 2005). Detecting functional abnormalities of muscles and bones in the area is considered crucial to effectively treat groin symptoms (Pizzari et al., 2008).

Diagnostic procedure should therefore include both the physical examination of the movement quality assessment, which would help to discover potential biomechanical imbalances; and diagnostic assessment of certain structures, which might already be pathologically altered as a consequence of poor biomechanics. The Doha agreement (Weir et al., 2015) provides useful guidelines regarding the diagnostic process of SRGP. Some good diagnostic recommendations were also given by Falvey et al. (Falvey et al., 2009) along the definition of the groin triangle.

The assessment techniques of groin symptoms (adductor muscles strength and flexibility, iliopsoas muscle strength and flexibility) include pain reproduction and reveal potential biomechanical imbalances and strength and flexibility deficits. They were reported to be reliable (Holmich et al., 2004, Malliaras et al., 2009) and are widely used in the diagnostic process of groin pathologies.

Most common causes of athletic groin pain are adductor-, iliopsoas- and abdominal related (Holmich, 2007). Reliable assessment of adductor, iliopsoas and abdominal muscles should include palpation, strength and flexibility measurements (Holmich et al., 2004, Malliaras et al., 2009, Weir et al., 2015). In the strength and flexibility measurements, both pain reproduction and side-to-side asymmetries are assessed.

Pain during palpation of adductor muscle insertion to pubic bone and 2-4 cm distally (6-8 on "pubic clock") allows detecting the abnormalities such as adductor tendinopathy, ethensopathy and musclulo-tendinous junction pathologies (Falvey et al., 2009). Bilateral adductor strength (in 0°, 30° and 45° of hip flexion (squeeze test)) and flexibility (lying supine (Holmich et al., 2004) or bent knee fall out test (Malliaras et al., 2009)) measurements detects muscle pathologies when reproducing pain. According to the Doha agreement, the pain or tenderness on palpation of the adductor muscles and during resisted adduction are the diagnostic criteria for adductor-related groin pain (Weir et al., 2015).

Reproduced pain during palpation of the iliopsoas muscle (superiorly to inguinal ligament), strength and flexibility testing (modified Thomas' test) suggests iliopsoas muscle pathology (Falvey et al., 2009, Holmich et al., 2004). Asymmetry in strength and flexibility measurements, similarly to adductor muscles, might detect biomechanical imbalances, which should be addressed in rehabilitation. Pain on palpation and during strength or flexibility testing are the diagnostic criteria for iliopsoas-related groin pain according to the Doha agreement (Weir et al., 2015).

Pubic-related pathologies can be suspected with the pain reproduction on the palpation on the pubic bone and adjacent bones (Weir et al., 2015). No particular stress or resistance test was identified to further diagnose the pubic-related pathology.

Abdominal musculature assessment should also include both pain reproduction during palpation, strength and flexibility assessment. Pain on palpation directed at "12" on the "pubic clock" is consistent with ethensopathy or tendinopathy of rectus abdominis muscle (Falvey et al., 2009). This is supported by painful abdominal functional tests (resisted sit-ups) (Falvey et al., 2009, Holmich et al., 2004). Weakness detected during this test implies potential muscular imbalances in pelvic and pubic symphysis areas.

Femoro-acetabular joint and acetabular labrum pathologies are commonly associated with groin symptoms (Narvani et al., 2003). Hip pathologies, particularly labral tears, still present a diagnostic challenge (Wenger et al., 2004, Martin et al., 2006). Falvey et al. advises using the impingement test to detect these groups of entities. This test (as well as the commonly used FABER test) was reported to be specific but only moderately sensitive compared to MRI scans (Troelsen et al., 2009). Therefore, MRI scans are still the "gold standard" in diagnostic process of hip and labrum pathologies (Chan et al., 2005, Freedman et al., 2006, Troelsen et al., 2009).

According to Lovell (Lovell, 1995), sportsman's hernia (referred to as "hernia without herniation") is the most common clinical entity responsible for groin symptoms among 189

athletes with groin pain. Falvey describes it as “incipitent hernia” and, following other authors, proposes reproduction of pain during resisted “torsion” of the trunk or as a result of palpation on superficial inguinal region (Gilmore, 1998, Kumar, 2004). However, many authors refer to “sport’s hernia” as a diagnosis of exclusion (Atkins et al., 2010), so further investigations to potentially exclude this entity from the differential diagnoses is necessary.

Imaging

When groin symptoms present atypically, do not respond to rehabilitation as expected or there is a suspicion of underlying serious pathology, imaging techniques give the opportunity to make the diagnostic process shorter and less frustrating (Albers et al., 2001).

Magnetic resonance imaging (MRI) is a commonly reported technique used in diagnosing pathologies associated with SRGP. Many authors report a high percentage of pathologies discovered by using MRI (Albers et al., 2001, Ekberg et al., 1996, Johnston et al., 2005, Kunduracioglu et al., 2007, Lawande et al., 2007, Zoga, 2009). It is frequently used to establish the diagnosis of pubic symphysis pathologies, hernia or muscular ethesiopathies. It also facilitates exclusion of other potential hip pathologies as labral tears (with contrast enhancement) and femoro-acetabular impingement.

Ultrasonographic investigations may also be of use in the diagnostic process for SRGP. They have been used previously for the diagnosis of groin pain, but mainly in non-athletic populations (Truong et al., 1993, Deitch and Soncrant, 1981). Few studies advocate the use of ultrasonography in SRGP, primarily to exclude the true hernias from the differential diagnoses (Davies et al., 2010, Orchard et al., 1998).

Other reported imaging techniques include plain film radiography and herniography (Ekberg et al., 1997, Ekstrand and Hilding, 1999). Both are reported to be reliable, but they are useful in only a limited number of cases. Similarly to ultrasonography, they are mainly used to exclude

the morphological or traumatic hip joint pathologies (radiography) or true hernias (herniography).

Functional assessment

Although the Doha agreement provided a very useful tool of the definition and diagnoses of groin injury, the mechanisms and inter-relation of different structures associated with pain is still not well understood (Weir et al., 2015). It is agreed, that the symptoms presenting in the groin area may have various aetiologies and are usually caused by a number of underlying structures (Falvey et al., 2009, Weir et al., 2015, Holmich, 2007). The complexity of the biomechanical demands of the area as well as multi-factorial cause of SRGP provides an argument for classifying patients by movement pattern rather than tissue diagnosis (Sahrmann, 2001), which is not mentioned in the Doha agreement nor present strongly in the literature.

Similar problems were identified in relation to other pathologies, such as shoulder pain and lower back pain (Roussel et al., 2013, Roussel et al., 2009, Worsley et al., 2013, Mottram et al., 2009), where identifying a discrete symptomatic structure is often challenging. In these cases, the rehabilitation strategy focusing on a movement pattern rather than being pathology may be a suitable way forward (Worsley et al., 2013, Mottram et al., 2009, Roussel et al., 2013). The argument is that identifying adverse movement patterns and muscle activation imbalances in SRGP, leads directly to relevant rehabilitation decision-making and may ultimately improve rehabilitation outcomes and reduce recurrence.

In groin pain area, this is supported by a variety of previous research on muscular function in SRGP, the vast majority of which used very vague inclusion criteria of the participants (Arnason et al., 2004, Cowan et al., 2004a, Crow et al., 2010, Jansen et al., 2010, Malliaras et al., 2009, Mens et al., 2006). The majority of those studies included participants suffering from groin symptoms for at least 4-6 weeks and presenting with pain reproduction during palpation of

the groin and popular tests such as the 'squeeze' test. All studies show strong results, which justify the more general and descriptive, rather than tissue specific strategy in athletes with SRGP. A comprehensive overview of all studies published on the biomechanical factors associated with SRGP and further considerations on the inclusion criteria and terminology used in those studies are presented in Chapter 2: Systematic review (p. 45).

Treatment

Progressive rehabilitation

An active, exercise based therapy (optimally combined with a manual treatment) has been reported to be most effective in SRGP (Weir et al., 2011b, Jansen et al., 2008, Machotka et al., 2009). However, high re-currency and symptoms that persist despite treatment suggest that there are underlying, not yet recognised factors that contribute to SRGP and prevent a consistently full recovery. Commonly agreed, multi-structural nature of SRGP (Delahunt et al., 2015, Weir et al., 2015, Falvey et al., 2009) makes a successful recovery challenging, but there is agreement that a multi-focused therapy needs to be applied.

Acute groin injuries commonly affect adductor muscles and tend to heal quickly. However, significant percentage of these injuries might turn into chronic condition, making them one of the major risk factors for subsequent SRGP (Arnason, 2004). The chronic SRGP is mainly treated by physiotherapy regardless of problems with diagnosis (Jansen et al., 2008, Machotka et al., 2009).

Active physical treatment focusing on stretching and strengthening of hip and pelvic stability muscles is reported to be effective (Hölmich et al., 1999, Rodriguez et al., 2001). These findings are more positive than in a recently published high quality randomized controlled trial comparing the outcomes of exercise (adductors, abdominal and stability exercises) and multi-modal (heating, manual therapy and stretching) therapy in 54 athletes (Weir et al., 2011b).

Although a greater percentage of athletes came back to sports after exercise therapy (55%) than after multi-modal therapy (50%), the multi-modal group returned to sporting activity quicker (after 12.8 weeks) than the exercise group (17.3 weeks). Outcomes of physical therapy were also reported to have far less positive outcomes than operative treatment in randomized clinical trial by Paajanen et al. Compared to 90% of participants who underwent surgical treatment, only 27% of those treated conservatively have returned to sport within 3 months since the commence of the treatment (Paajanen et al., 2011). However, the MRI or herniography confirmation of true hernia or severe pubic symphysis pathology was one of the inclusion criteria for study participants, with the SRGP subgroup not clearly extracted.

Injection therapy

Studies investigating the outcomes of steroid injections in groin pain treatment report 100% return to sport in symptomatic athletes, but they lack the control group to compare the outcomes with (Holt et al., 1995). The injections were usually used in groin pain diagnosed as "osteitis pubis" (Holt et al., 1995, O'Connell et al., 2002). This entity was reported to be a self-treating pathology, healing naturally over a period of time (Lynch and Renstrom, 1999), which may provide a large bias for this study. Prolotherapy (12.5% dextrose and 0.5% lidocaine injected in tender region) was reported to have good outcomes in a case study/series? (Topol et al., 2005), but the level of evidence is low.

Surgery

Surgical intervention is usually considered when conservative treatment is unsuccessful (Jansen et al., 2008). The outcomes, therefore, are only reported for a narrow population not responding to physiotherapy. The interventions vary depending on the final diagnosis. When sport's hernia (referred to as a functional deficiency of the abdominal wall without true hernia) was suspected, Bassisni hernia repair or mesh repair is undertaken (Smedberg et al., 1985,

Ingoldby, 1997, Taylor et al., 1991, Hackney, 1993). Reported results are excellent, but the quality of the studies is low to moderate.

In adductor-related problems, adductor tenotomy was reported to have positive results by two studies with low level of evidence (Martens et al., 1987, Akermark and Johansson, 1992). When positive outcomes from the above techniques are not realised, neurotomy of the ilio-inguinal or obturator nerve can be applied (Bradshaw et al., 1997, Polglase et al., 1991).

Conservative management tends to be the first option (Jansen et al., 2008) after a period of rest and NSAIDs intake. Steroid injections are sometimes given simultaneously.

Deficits in knowledge about treatment

Although a number of simple biomechanical deficits in athletes with SRGP have been described, not all of them have been addressed in evaluated rehabilitation programmes. Additionally, no recommendations about dysfunctional movement patterns or muscle imbalance have been described. Strengthening of the adductor, abductor and abdominal muscles tend to be the primary elements of the published treatment strategies, but little attention is given to muscular balance or movement patterns in either static or dynamic conditions. The paucity of research investigating these patterns has also been recognised by the Doha agreement, which highlights specific of movement analysis in SRGP as one of the area for further research (Weir et al., 2015).

A strong association between the movement and pathology has been found in other multi-structural entities such as shoulder and back pain (Mottram et al., 2009, Roussel et al., 2009). Some of the simple biomechanical measures, such as strength and flexibility deficits, have also been found in association with SRGP. They are systematically reviewed in Chapter 2, which summarises current knowledge about the biomechanical signatures of SRGP in athletes. These

simple measures indicate that there may be some benefit in exploring more complex biomechanical mechanisms and the deficit of a body of research in this area was noted.

Therefore, additionally to already described biomechanical deficits in athletes with SRGP, investigating the kinematics and muscle activation patterns in athletes with SRGP seemed to be a natural step forward. However, in order to consider altering treatment guidelines, further exploration is first required to determine whether such deficits exist. These represent significant gaps in the literature, which the thesis addresses.

The aims of this thesis are therefore to improve our understanding of the biomechanical characteristics of SRGP in athletes by exploring associated movement and muscle activation patterns. Additionally, it was decided to explore how these might differ between sporting groups and participation levels, in order to inform prevention and rehabilitation planning.

Chapter 2: Systematic review

Movement and muscular function in sports related groin pain: a systematic review with meta-analysis.

Chapter overview

This chapter reviews the published evidence on the movement and muscular function associated with sports related groin pain (SRGP) – prospectively and retrospectively. By summarising these associations it also identifies a research gap and thus provides further rationale for the experimental chapters of this thesis. This summary shows that there are clear associations between simple biomechanical measures, such as strength and flexibility, and SRGP - which are consistent between studies despite no clear diagnoses. However very little attention has been given to more sophisticated biomechanical measures, potentially a barrier to designing more successful rehabilitation programmes and improving outcomes.

Abstract

Background: Sports related groin pain (SRGP) is a common entity in rotational sports such as football, rugby and hockey, accounting for 12%-18% of injuries each year, with high recurrence rates and often prolonged time away from sport.

Objective: This systematic review synthesizes movement and muscle function findings to better understand deficits and guide rehabilitation.

Study selection: Prospective and retrospective cross-sectional studies investigating muscle strength, flexibility, cross-sectional area, electromyographic activation onset and magnitude in patients with SRGP were included.

Search methods: Four databases (Medline, Web of Science, Ebsco and EMBASE) were searched in June 2014. Studies were critiqued using a modified version of the Downs and Black Quality Index, and meta-analysis performed.

Results: Seventeen studies (14 high quality, 3 low quality; 8 prospective and 9 retrospective) were identified. *Prospective findings*: *Moderate evidence* indicated decreased hip abduction flexibility as a risk factor for SRGP. *Limited or very limited evidence* suggested that decreased hip adduction strength during isokinetic testing at ~119 °/s was a risk factor for SRGP, but no associations were found at ~30°/s or ~210°/s, or with peak torque angle. Decreased hip abductor strength in angular velocity ~30 °/s but not in ~119°/s and ~210 °/s was found as a risk factor for SRGP. No relationships were found with hip internal or external rotation range of movement, nor isokinetic knee extension strength. Decreased isokinetic knee flexion strength also was a potential risk factor for SRGP, at speed ~60°/s.

Retrospective findings: There was *strong evidence* of decreased hip adductor muscle strength during a squeeze test at 45°, and decreased total hip external range of movement (sum of both legs) being associated with SRGP. There was strong evidence of no relationship to abductor muscle strength nor unilateral hip internal and external rotation range of movement. *Moderate* evidence suggested that increased abduction flexibility and no change in total hip internal range of movement (sum of both legs) were retrospectively associated with SRGP. *Limited or very limited* evidence (significant findings only) indicated decreased hip adductor muscle strength during 0° and 30° squeeze test and during eccentric hip adduction test, but decrease in isometric adductors to abductors strength ratio at speed 120.32°/s; decreased abductors to adductors activation ratio in early phase in moving leg as well as in all three phases in weight-bearing leg during SHF; increased hip flexors strength during isokinetic and decrease in transversus abdominis muscle resting thickness associated with SRGP.

Conclusions: There were a number of significant movement and muscle function associations observed in athletes both prior to and following the onset of SRGP. The strength of findings was hampered by the lack of consistent terminology and diagnostic criteria, with there being clear guides for future research. Nonetheless, these findings should be considered in rehabilitation and prevention planning.

Introduction

Sports-related groin pain (SRGP) is a common clinical entity, accounting for 12% - 16% of all sports injuries (Werner et al., 2009, Ekstrand and Hilding, 1999). It is particularly prevalent in sports involving rotation and cutting movements, such as football, rugby and hockey (Orchard et al., 1998). It is often associated with prolonged time away from playing (Holmich et al., 1999, Weir et al., 2010) and therefore considered a significant problem in professional sport.

The difficulties in diagnosis and treatment of SRGP are partly caused by the lack of consensus amongst researchers and clinicians in classification of the functional anatomy of the area and the large range of diagnostic terms used (Weir et al., 2015, Bradshaw et al., 2008). Patients suffering from SRGP are often 'diagnosed' with osteitis pubis, adductor tendinopathy, sportsman's hernia, Gilmore's groin as well as iliopsoas-, rectus abdominis- and adductor-related muscular disorders. Various underlying tissue pathologies are likely to coexist (Holmich, 2007) and there is a lack of clinical or imaging tests with high levels of sensitivity or specificity. Further, there is a strong argument – particularly from a rehabilitation perspective - for classifying patients by movement pattern rather than tissue diagnosis (Sahrmann, 2001). There have been a number of studies examining movement and muscle function factors causally or associatively linked to SRGP, but little synthesis of this data. Our review will therefore include all sub-diagnoses of groin pain, gathered under the umbrella term of SRGP. Further, I will consider movement and muscle function factors for specific tissue diagnoses where these are clear, but also across the SRGP group in order to identify common biomechanical patterns.

Two systematic reviews (Machotka et al., 2009, Jansen et al., 2008) have been published on the effectiveness of conservative therapy in SRGP identyfying a paucity of high quality research in this area. Both reviews indicate that regardless of the underlying initial pathology of the groin pain, active rehabilitation including flexibility, stretching and strengthening exercises of

the pelvic girdle and hip muscles are critical components in effective management. Studies supporting active rehabilitation for SRGP tend to focus on hip adductor and abdominal muscle strengthening (Hölmich et al., 1999, Weir et al., 2010). However, the sports-specificity of these elements is limited. Moreover, the recurrence rate of groin symptoms is still relatively high, suggesting that current rehabilitation strategies may not fully address deficits in the neuromuscular system. This systematic review and meta-analysis will synthesise evidence related to movement and muscle function deficits in athletes with SRGP, with the aim of providing a useful guide for clinicians and researchers developing and evaluating rehabilitation and prevention programs. The hypothesis of this study was that there are clear and consistent biomechanical patterns in the athletes with SRGP emerging from previously published studies, giving evidence for either associations drawn from the retrospective studies, or risk factors from the prospective studies.

Methodology

Inclusion and Exclusion criteria

Prospective and retrospective cross-sectional (i.e. case-control) studies investigating movement and muscle function variables associated with chronic groin pain published in English from database inception to June 2014 were included. Groin pain diagnostic labels included 'adduction-related groin pain', 'osteitis pubis', 'pubialgia', 'pubalgia', 'sports hernia' or 'adductor tendinopathy'. Only participants whose groin pain was associated with playing sports were included. Biomechanical terms included strength, flexibility (range of motion), muscle activation magnitude and timing, muscle size and cross-sectional area. Measurement techniques included magnetic resonance imaging (MRI), ultrasound, electromyography, dynamometer or physical examination.

Single case studies, cadaver studies, studies on healthy participants only and studies without a control group were excluded. Studies including participants diagnosed with true hernias; and hip joint, thoracic or lumbar spine pathology were excluded from the review.

Search strategy and review process

A reproducible search strategy was created by three reviewers (PK, CB and DM). The search terms combined muscle features or measurement tools ("strength" OR "flexibility" OR "cross-section*" OR "onset" OR "activation" OR "range of motion" OR "ROM" OR "EMG" OR "electromyograph*" OR "ultrasound*" OR "dynamometer" OR "MRI" OR "magnetic resonance imaging" OR "ultrasonograph*" OR "US") and diagnostic terms ("groin pain" OR "chronic groin pain" OR "osteitis pubis" OR "pubialgia" OR "pubalgia" OR "adductor pain" or "adductor tendin*" OR "adductor tendon*" OR "adductor* strain" OR ""adductor*" injur*"). MEDLINE, Web of Knowledge, EMBASE and EBSCOHost databases were searched, using keywords wherever possible.

Retrieved studies were entered into Endnote (Thomson, California, USA) and duplicates deleted. Titles and abstracts were screened against the inclusion and exclusion criteria by two independent reviewers (PK and CS). Where necessary, abstracts and full texts were obtained to make a final decision. A third reviewer (CB) was available to reach consensus if there were any conflicts. The reference lists of included studies were searched and citation tracking performed via Google Scholar for additional relevant studies.

Quality assessment and study analysis

A modified version of the Downs and Black Quality Index was applied by two independent reviewers (PK and CS) to assess the quality of included studies. A third reviewer was available to resolve differences (DM). Irrelevant questions referring to intervention trials were excluded from the questionnaire. Fifteen relevant questions built up a modified version of Downs and

Black Quality Index, with a maximum score of 16 points (Barton et al. 2012). Papers were considered as high quality studies (HQS) when scored above 10 (inclusive) points and low quality studies (LQS) when scored below 10 points, following Barton et al (Barton et al., 2012).

Data extraction and analysis

Characteristics of the study participants (number, type and level of sport, age, height and weight), diagnosis of the symptomatic patients, task (if relevant), muscle and/or muscle group, diagnostic tool, and results of symptomatic and control group were extracted from the selected articles (**Error! Reference source not found.**). Means and standard deviations (SD) were extracted in order to enable calculation of standard mean differences (SMDs). Where the presentation of the data was not adequate to calculate SMDs, corresponding authors were contacted by email in an attempt to obtain the data. In one case (Ibrahim et al., 2007), where the SD was not published, it was calculated by the authors of this review as the paper included individual participant values for variables measured. Where possible, data was pooled for common measurement features of given muscle groups in order to establish the levels of evidence. If results were not presented nor obtained from authors in a format allowing data pooling, it was omitted in meta-analysis. If only one study investigated given muscle characteristics, SMD was calculated from the result presented in this study. This analysis is more stringent than statistics commonly used in individual studies (such as t-test). It might, therefore, show different results to those reported previously within a specific study.

If the results a study were provided for both legs/both sides of the body, only data from right *or* dominants side of the body were further calculated in order to maintain the data independence, as described or presented in previous studies (Menz, Neal et al., 2014).

In studies reporting results from isokinetic measurements, originally reported radians per second ($rad*s^{-1}$) were converted to degrees per second (°/s) accurately in abstract, and approximately in results section, in order to facilitate the delivery of the clinical implications.

Definitions for 'levels of evidence' were guided by recommendations made by van Tulder et al (van Tulder et al., 2003):

Strong evidence was defined as pooled results derived from three or more studies, including a minimum of two high quality studies (HQS), which are statistically homogenous ($p > 0.05$). *Moderate* evidence was defined as statistically significant pooled results derived from multiple studies, including at least one HQS, which are statistically heterogeneous ($p < 0.05$); or from multiple low quality studies (LQS), which are statistically homogenous ($p > 0.05$). *Limited* evidence was defined as results from multiple LQS, which are statistically heterogeneous ($p < 0.05$); or from one HQS. *Very limited* evidence was defined as results from one LQS. *Conflicting* evidence was defined as pooled results insignificant and derived from multiple studies regardless of quality, of which some show statistical significance individually, which are statistically heterogeneous ($p < 0.05$, that is, inconsistent).

Results

Seventeen studies were included in the final yield. The search results from each database are shown on Figure 3. Reference list screening of included studies identified two additional studies (Arnason et al., 2004, Thorborg et al., 2014) to the initial 15 included studies.

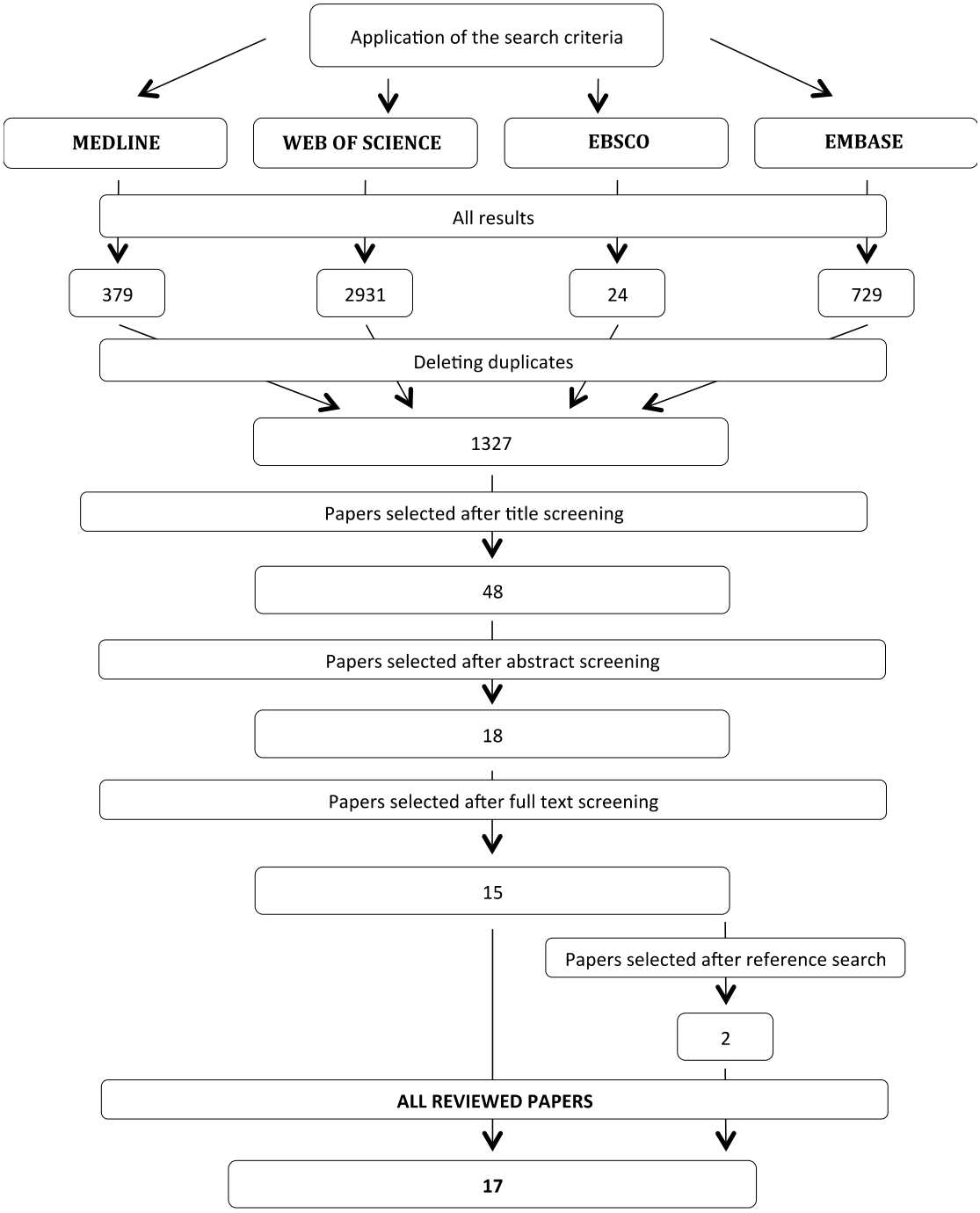


Figure 3: Flowchart showing studies inclusion and exclusion process for the review.

Paper	Type of study	Diagnosis	N (SRGP:C)	Type of sport	Level of sport	Age	Weight	Height
Arnason et al, 2004	Prospective cohort study	Groin strain	17:281	Icelandic football	Elite league and first division	SRGP:25.1(1.2);C:24.0(0.2)	SRGP:79.1(1.2); C:76.4(0.4)	SRGP:183.0(1.4); C:180.5 (0.4)
Cowan et al, 2004	Retrospective, case-control study	Long standing groin pain	10:12	Australian football	Elite or sub-elite	SRGP:26(7); C:25(6)	SRGP:78.1(8.4); C:76.8(11.3)	SRGP:180.7(7);C:176.5(7.9)
Crow et al, 2010	Prospective study	Groin injury	12:12	Australian football	Elite	16-18	N/R	N/R
Emery et al, 2001	Prospective cohort study	Groin strain injury	204:1088	Canadian National Hockey League	Professional	N/R	N/R	N/R
Engebretsen et al, 2010	Prospective cohort study	Groin injury	51:457	Football (soccer)	Amateur	N/R	N/R	N/R
Ibrahim et al, 2007	Prospective study	Adductor strain	8:79	Australian football	Professional	N/R	N/R	N/R
Jansen et al, 2010	Retrospective, case-control study	Adduction related groin pain	42:23	Various	Amateur	R SRGP:24.8 (6.9);L SRGP:28.2(10.4);C:23.9(4.7)	R SRGP:80(9.2); L SRGP:76.4(11.8);C:78.9(6.8)	R SRGP:184.4(6.8);L SRGP:181.4(6.5); C:183.7(6.7)
Malliaras et al, 2010	Retrospective, case-control study	Groin pain	10:19	Australian Rules football and soccer	Elite	SRGP:17.3(0.8); C:17.1(1.6)	SRGP: 78.5 (7.0); C: 77.1 (5.4)	SRGP: 184.4 (6.7); C: 183.9 (7.8)
Mens et al, 2006	Retrospective, case-control study	Adduction related groin pain	44:44	Various	Amateur	SRGP: 31.3 (28.1–34.6); C: 32.2 (30.0–35.4)	SRGP: 79.4 (76.3–82.5); C: 82.4 (79.5–85.3)	N/R
Mohammad et al, 2014	Retrospective, case-control study	Osteitis pubis	20:20	Football (soccer)	N/R	SRGP:19.94(3.51); C:20.78(3.35)	SRGP:70.91(7.26); C:71.33(7.35)	SRGP:176.16(4.93); C:176(4.15)

Morrissey et al, 2012	Retrospective, case-control study	Chronic groin pain	09:09	Football code	Amateur	SRGP:24(3); C:25(2)	SRGP: 81 (4); C:82(3)	SRGP:1.8(0.1); C:1.8(0.1)
Nevin et al, 2013	Retrospective, case-control study	Long standing groin pain	18:18	Gaelic football	Club-level	SRGP:23.89(3.18); C:23.83(3.55)	SRGP:80.28(9.77); C:72.28(10.3)	SRGP:1.79(0.06); C:1.80(0.06)
O'Connor et al, 2004	Prospective study	Groin injury	21:72	Australian Rugby	Professional	SRGP: 22.2 (2.9)*; C: 20.2 (4.5)*	SRGP: 90.5 (9.5)*; C: 84.7 (10.2)*	SRGP: 1.80 (0.13); C: 1.78 (0.06)
Thorborg et al, 2014	Cross-sectional study	Adductor related groin pain	21:16	Football (soccer)	Elite and sub-elite	SRGP: 24.5 (2.5); C: 22.9 (2.4)	SRGP: 74.6 (6.4); C: 78.6 (6.3)	SRGP: 179.8 (5.9); C: 179.8 (5.0)
Tyler et al, 2001	Prospective study	Adductor strain	08:37	Ice hockey	Professional	N/R	N/R	N/R
Verrall et al, 2005	Retrospective, case-control study	Chronic groin injury	47:42:00	Australian Rules football and soccer	Professional	N/R	N/R	N/R
Verrall et al, 2007	Prospective cohort study	Chronic groin injury	04:25	Australian Rules football	Professional	SRGP:22.75(1.70); C:21.16(0.63)	SRGP:72.50(3.28); C:84.92(1.99)	SRGP:175.50(2.33); C:177.36(6.82)

Table 2: Participants characteristics in reviewed studies; SRGP – sports related groin pain, C- controls, * indicate a significant difference between groin pain and control participant

Quality assessment and data analysis

The details of the modified Downs & Black scale results are shown in Table 3. The scores for the studies included in the review ranged between 8 and 15, with an average of 11. Of 17 included studies, 14 were HQS and 3 were LQS.

Where possible, the results of reviewed studies were pooled for analysis using Review Manager 5.2. Outcome values from a few papers were not reported and not obtainable despite contacting corresponding authors (Cowan et al., 2004a, Crow et al., 2010, Jansen et al., 2010, Tyler et al., 2001, Mohammad et al., 2014).

D&B criterion PAPER	(1)	(2)	(3)	(5)	(6)	(7)	(10)	(11)	(12)	(15)	(16)	(18)	(20)	(21)	(25)	TOTAL	Study quality
Thorborg *et al.*	1	1	1	2	1	1	1	1	0	1	1	1	1	1	1	**15**	**HQ**
Arnason *et al.*	1	1	1	2	1	1	1	1	0	0	1	1	1	1	1	**14**	**HQ**
Cowan *et al.*	1	1	1	2	1	1	1	0	0	0	1	1	1	1	1	**13**	**HQ**
Mens *et al.*	1	1	1	1	1	1	1	0	0	1	1	1	1	1	1	**13**	**HQ**
Engebretsen *et al*.	1	1	1	0	1	1	1	1	1	1	1	1	0	1	0	**12**	**HQ**
Malliaras *et al.*	1	1	1	1	1	1	1	0	0	0	1	1	1	1	1	**12**	**HQ**
O'Connor *et al.*	0	1	1	2	1	1	1	0	0	0	1	1	1	1	1	**12**	**HQ**
Crow *et al.*	1	1	1	0	1	1	1	0	0	0	1	1	1	1	0	**10**	**HQ**
Emery *et al.*	1	1	1	0	1	1	0	1	0	0	1	1	1	1	0	**10**	**HQ**
Ibrahim *et al.*	1	1	1	0	1	0	1	1	1	0	1	0	1	1	0	**10**	**HQ**
Jansen *et al.*	1	1	1	1	1	1	1	0	0	0	1	1	1	0	0	**10**	**HQ**
Morrissey *et al.*	1	1	1	1	1	0	0	0	0	0	1	1	1	1	1	**10**	**HQ**
Tyler *et al.*	1	1	1	1	1	0	1	0	0	0	1	1	1	1	0	**10**	**HQ**
Verral *et al.*	1	1	1	0	1	1	1	0	0	0	1	1	1	1	0	**10**	**HQ**
Nevin *et al.*	1	1	1	1	0	1	0	0	0	0	1	1	1	0	1	**9**	**LQ**
Verral *et al.*	1	1	0	1	1	1	1	0	0	0	1	1	0	1	0	**9**	**LQ**
Mohammad *et al.*	1	1	0	1	0	1	1	0	0	0	1	1	0	0	1	**8**	**LQ**

Table 3: Results of the quality assessment using a modified *Downs & Black* scale *(Downs and Black, 1998).* (1) Clear aim/ hypothesis; (2) clear outcome measures; (3) clear participant characteristics; (5) clear principal confounders; (6) clear study findings; (7) estimates of random variability provided; (10) probability values provided; (11) invited participants representative to entire population; (12) participants prepared to participate representative to entire population; (15) attempt to blind outcome measures; (16) no data-dredging; (18) appropriate statistical tests; (20) valid and accurate outcome measures; (21) appropriate case-control matching; (25) adequate adjustment for confounding variables.

Diagnostic nomenclature

Reviewed studies used a variety of diagnostic terms including groin pain (Malliaras et al., 2009), chronic groin pain (Morrissey et al., 2012a), long standing groin pain (Cowan et al., 2004a, Nevin and Delahunt, 2013), adductor related groin pain (Thorborg et al., 2014), adduction related groin pain (Jansen et al., 2010, Mens et al., 2006), groin strain (Arnason et al., 2004), groin injury (Crow et al., 2010, Engebretsen et al., 2010, O'Connor, 2004), chronic groin injury (Verrall et al., 2005a, Verrall et al., 2007a), adductor strain (Ibrahim et al., 2007, Tyler et al., 2001), groin or abdominal strain injury (Emery and Meeuwisse, 2001) and osteitis pubis (Mohammad et al., 2014) (Table 2).

Hip adductor muscle characteristics

Adductor muscle strength

Prospectively, four HQS (Crow et al., 2010, Engebretsen et al., 2010, O'Connor, 2004, Tyler et al., 2001) reported a significant decrease of adductor muscle strength as a risk factor for SRGP, whilst one HQS reported adductor muscle strength was not associated with risk of SRGP (Emery and Meeuwisse, 2001). Three of those studies measured adductor strength preseason (Engebretsen et al., 2010, Tyler et al., 2001, Emery and Meeuwisse, 2001). One study performed measurements weekly within season (Crow et al., 2010), and reported a significant decrease of adductor strength no sooner than two weeks pre-injury. Only one HQS (O'Connor, 2004) presented adequate data to complete calculation of SMDs, which indicated limited evidence of decreased adductor muscle strength during isokinetic test in angular velocity of 2.08 $rad*s^{-1}$ (~119°/s) (-0.51, -1.00 to -0.02) as a risk factor for SRGP, but not in angular velocities of 0.52 $rad*s^{-1}$ (~30°/s) (-0.33, -0.81 to 0.16) and 3.66 $rad*s^{-1}$ (~210°/s) (-0.18, -0.67 to 0.30) (Figure 4a). No indication was provided regarding when these measurements were taken.

Retrospectively, there was strong evidence emerging from three HQS (Jansen et al., 2010, Malliaras et al., 2009, Mens et al., 2006) and one LQS (Nevin and Delahunt, 2013) of existing association between adductor muscle weakness during squeeze test in 45° hip flexion and SRGP (-1.00, -1.31 to -0.70) (Figure 4b). There was limited evidence from single HQSs of decreased adductor muscle strength during squeeze test in 0° (-1.04, -1.86 to -0.22) and 30° (-0.83, -1.63 to -0.03) of hip flexion (Malliaras et al., 2009)(Figure 4b); and during eccentric adduction strength test (Thorborg et al., 2014) (-1.37, -2.10 to -0.64, Figure 4b) associated with SRGP. Limited evidence emerged from one HQS of no difference in adductor muscle strength during isometric adduction strength test (Thorborg et al., 2014) associated with SRGP (Figure 4b); very limited evidence emerged from one LQS indicates adductor muscle strength during isokinetic measurements in angular velocity 2.1 rad*s^{-1} (~120°/s) is not associated with SRGP (Mohammad et al., 2014) (Figure 4b).

Abduction flexibility

Prospectively, three HQS (Arnason et al., 2004, Tyler et al., 2001, Emery, 2012) reported no change in abduction flexibility preceding the onset of SRGP. Two studies presented adequate data to complete the meta-analysis (Arnason et al., 2004, Tyler et al., 2001), providing moderate evidence that abduction flexibility is not a risk factor for SRGP development (SMD -0.36, CI from -0.80 to 0.09, Figure 4c).

Retrospectively, there was moderate evidence emerging from two HQS (Malliaras et al., 2009, Thorborg et al., 2014) on an existing association between increased abduction flexibility during bent knee fall out test and SRGP (0.87, 0.35 to 1.40, Figure 4d). Limited evidence emerged from one HQS (Thorborg et al., 2014)of no change in abduction flexibility during unilateral test in 0° of hip flexion and SRGP (Figure 4d).

Adductor muscle peak torque angle

Prospectively, there was limited evidence from one HQS (O'Connor, 2004) that adductor muscle peak torque angle change in angular velocity of 3.66 rad*s^{-1} (~210°/s) is not a risk factor for SRGP development (Figure 4e).

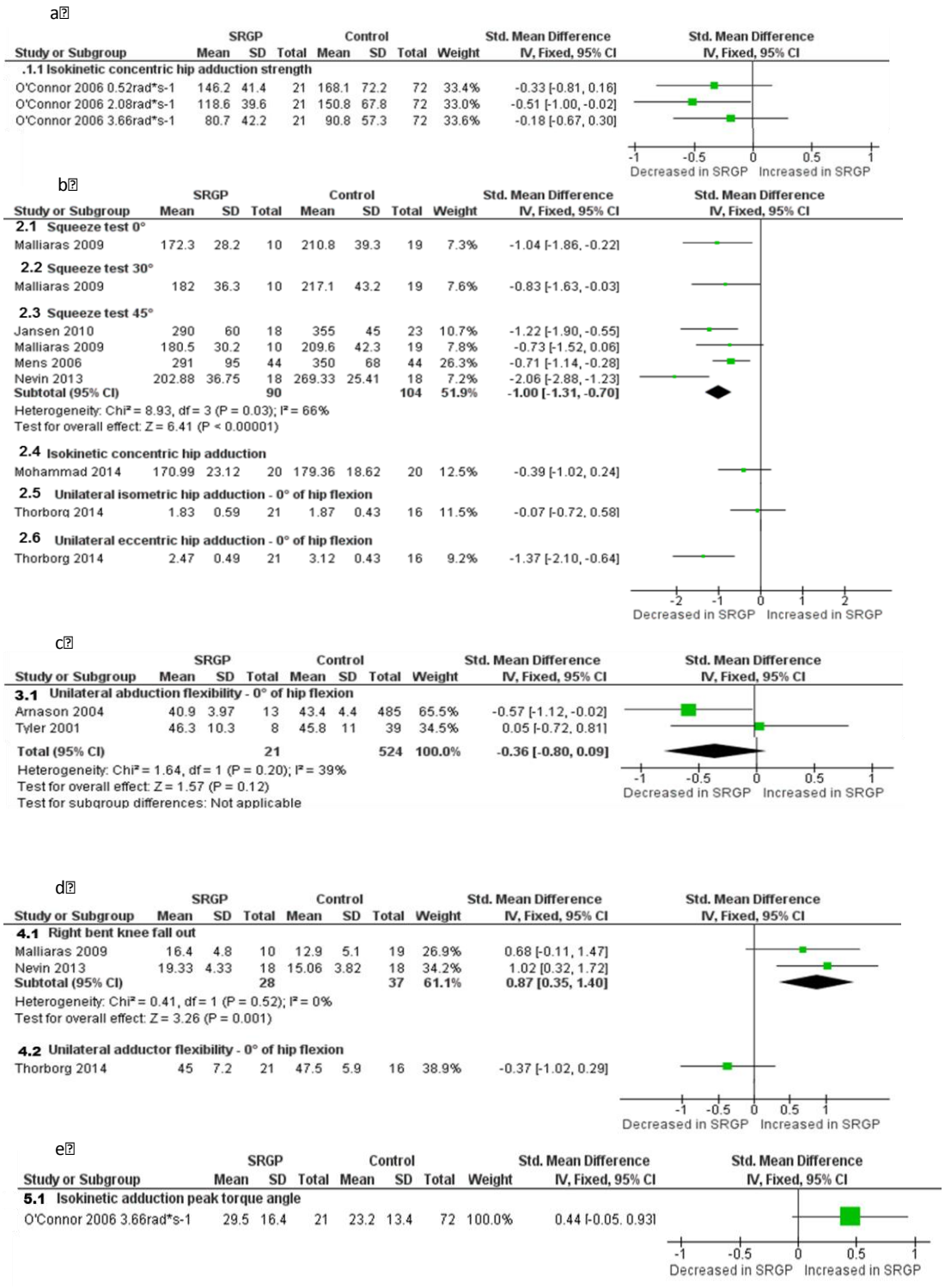
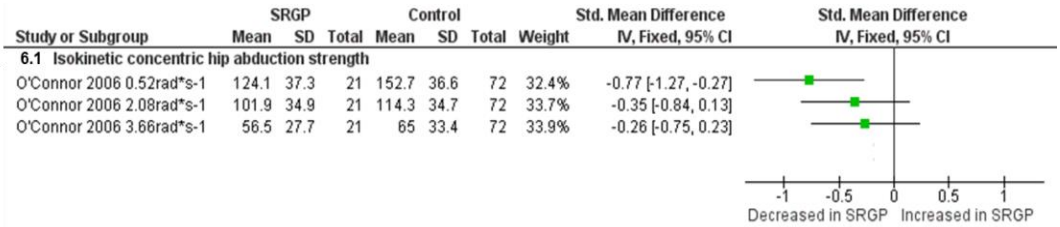
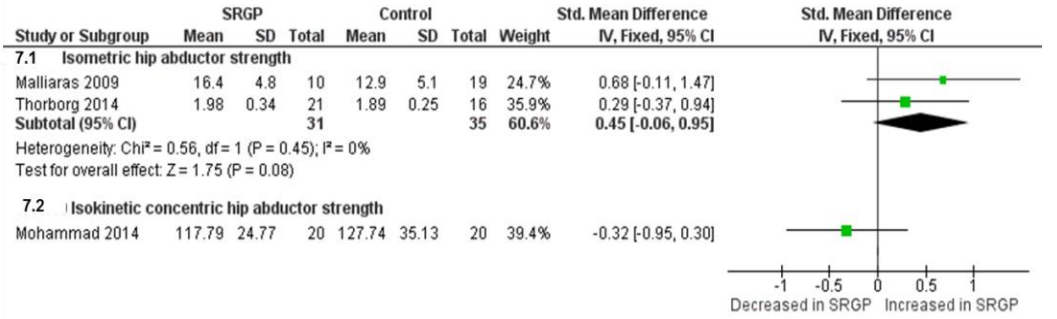


Figure 4: Forest plot detailing the analysis of the movement and muscular functions in the coronal plane relating to hip adductor muscles: a – hip adductor muscle strength prospective results, b – hip adductor muscle strength retrospective results, c – hip abduction flexibility prospective results, d – hip abduction flexibility retrospective results, e – hip adduction peak torque angle retrospective results.

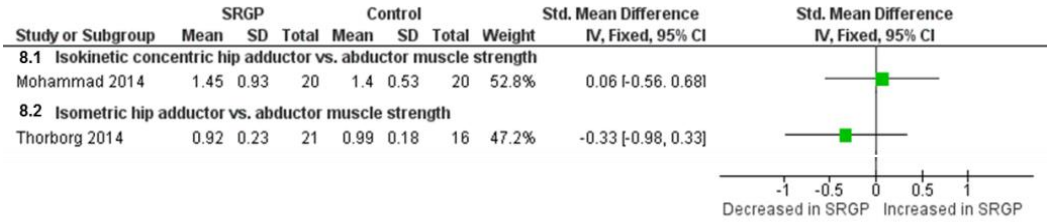
a



b



c



d

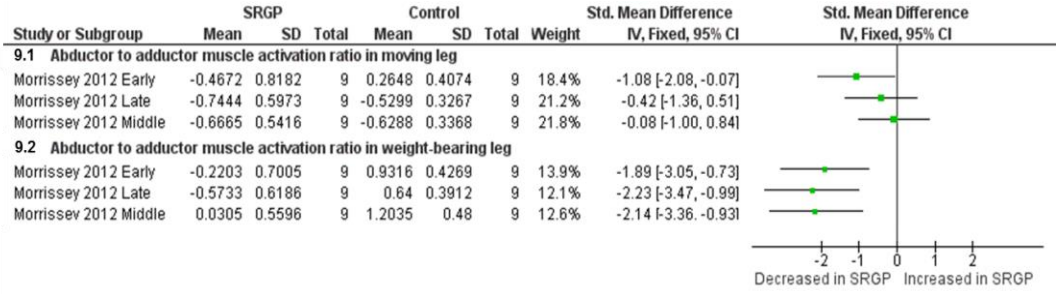


Figure 5: Forest plot detailing the analysis of movement and muscular functions in the coronal plane related to hip abductor muscles and relationship between the hip adductor versus abductor muscles: a – hip abductor muscle strength prospective results, b – hip abductor muscle strength retrospective results, c – hip adductor to abductor strength ratio retrospective results, d – hip abductor to adductor muscle activation ratio retrospective results.

Hip abductor muscle characteristics

Abductor muscle strength

Prospectively, there was limited evidence from one HQS (O'Connor, 2004) of a decrease in abductor muscle strength during isokinetic test in angular velocity of 0.52 rad*s^{-1} (~30°/s) (-0.77, -1.27 to -0.27) as a risk factor for SRGP development, but not in angular velocities of 2.08 rad*s^{-1} (~119°/s) and 3.66 rad*s^{-1} (~210°/s) (Figure 5a).

Retrospectively, there was strong evidence emerging from two HQS (Malliaras et al., 2009, Thorborg et al., 2014) of no change in abductor muscle strength during isometric unilateral measurements; and very limited evidence emerging from one LQS (Mohammad et al., 2014) of no difference in abductor muscle strength during isokinetic measurements in angular velocity 2.1 rad*s^{-1} (~120°/s), associated with SRGP (Figure 5b).

Relation between hip adductor and abductor muscles

Muscle strength ratios

Prospectively, one HQS (Tyler et al., 2001) reported decreased adductor to abductor muscle strength ratio as a risk factor for SRGP, but the format of data presentation was not adequate to complete the calculation of the SMD.

Retrospectively, there was limited evidence emerging from one HQS (Thorborg et al., 2014) and very limited evidence emerging from one LQS (Mohammad et al., 2014) of no change in isometric or isokinetic (in angular velocity 2.1 rad*s^{-1} (~120°/s)) adductor to abductor muscle strength ratio associated with SRGP (Figure 5c).

Abductor to adductor muscle activation ratio

Retrospectively, one HQS (Morrissey et al., 2012a) provided limited evidence of decreased GM:AL muscle activation ratio associated with SRGP in the moving leg during early (-1.08, -2.08

to -0.07), but not during middle or late phases of standing hip flexion movement (SHF) (Figure 5d). The same study provided limited evidence of decreased GM:AL muscle activation ratio associated with SRGP in weight-bearing leg during early (-1.89, -3.05 to -0.73), middle (-2.14, -3.36 to -0.93) and late (-2.23, -3.47 to -0.99) phases of SHF (Figure 5d).

Hip flexor muscle characteristics

Hip flexor muscle strength

Retrospectively, there was very limited evidence provided by one LQS (Mohammad et al., 2014) of increased hip flexor muscle strength during isokinetic test in angular velocity 2.1 rad*s^{-1} (~120°/s) associated with SRGP (1.72, 0.99 to 2.46); and limited evidence emerging from one HQS (Thorborg et al., 2014) of no change in hip flexor strength during isometric and eccentric strength test associated with SRGP (Figure 6a).

Hip extension flexibility

Prospectively, there was limited evidence provided by one HQS (Arnason et al., 2004) of no association between hip extension flexibility and risk of SRGP development (Figure 6b).

Retrospectively, there was limited evidence from one HQS (Thorborg et al., 2014) of no association between hip extension flexibility and SRGP (-0.19, -0.84 to 0.46, Figure 6c).

Hip extensor muscle characteristics

Hip extensor muscle strength

Retrospectively, there was very limited evidence emerging from one LQS (Mohammad et al., 2014) of no association between hip extensor muscle strength during isokinetic test in angular velocity 2.1 rad*s^{-1} (~120°/s) and SRGP (0.22, -0.40 to 0.84, Figure 6d).

Hip flexor to extensor muscle ratio

Retrospectively, there was very limited evidence emerging from one LQS (Mohammad et al., 2014) of no association between hip flexor to hip extensor muscle strength ratio during isokinetic test in angular velocity 2.1 rad*s^{-1} (~120°/s) associated and SRGP (0.15, -0.47 to 0.77, Figure 6e).

a

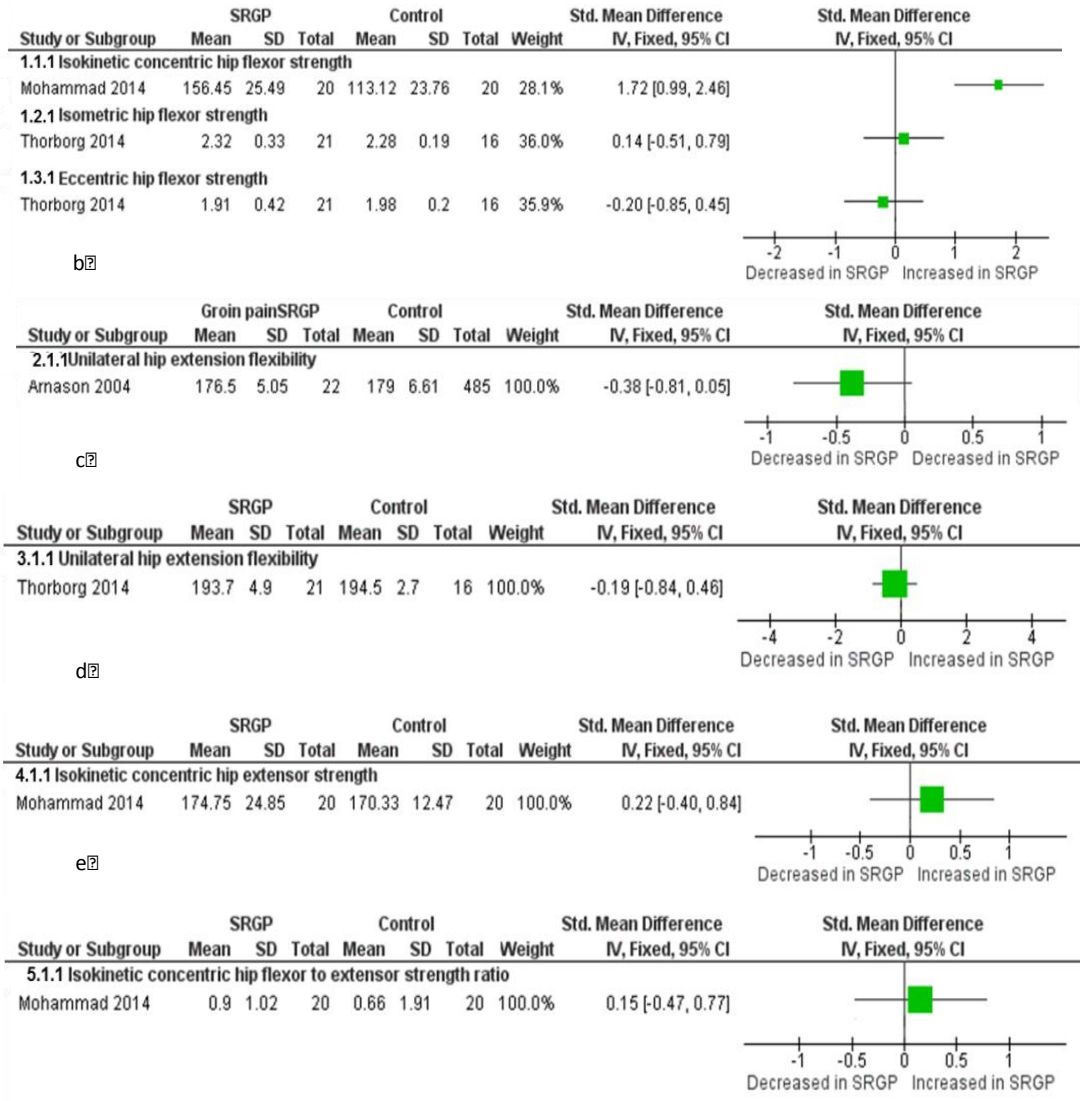


Figure 6: Forest plot detailing the analysis of movement and muscular functions in the sagittal plane: a – hip flexor muscle strength retrospective results, b – hip flexor muscle flexibility prospective results, c – hip flexor muscle flexibility retrospective results, d – hip extensor muscle strength retrospective results, e – hip flexor to extensor muscle strength ratio retrospective results.

Hip rotation range of movement

Prospectively, there was very limited evidence from one LQS (Verrall et al., 2007a) that hip internal and external rotation range of movement is not a risk factor for SRGP development (Figure 7a and c).

Retrospectively, there was strong evidence emerging from two HQS (Malliaras et al., 2009, Thorborg et al., 2014) and one LQS (Nevin and Delahunt, 2013) on no difference in unilateral hip internal rotation range of movement; and strong evidence emerging from two HQS (Malliaras et al., 2009, Verrall et al., 2005a) of no difference in bilateral hip total internal rotation range of movement (sum of both legs), associated with SRGP (Figure 7b). There was moderate evidence emerging from one HQS (Malliaras et al., 2009) and one LQS (Nevin and Delahunt, 2013) of no difference in unilateral hip external rotation range of movement; but strong evidence emerging from two HQS of decreased bilateral total hip external rotation range of movement (sum of both legs) associated with SRGP (-0.43, -0.80 to -0.05, Figure 7d).

Knee muscle characteristics

Prospectively, there was limited evidence from one HQS (O'Connor, 2004) that knee flexor muscle isokinetic strength measured with isokinetic measurements in angular velocity 1.04 rad*s^{-1} (~60°/s) is not a risk factor for SRGP (Figure 7e). The same study provided limited evidence of decreased concentric knee extensor muscle strength measured with isokinetic measurements in angular velocity 1.04 rad*s^{-1} (~60°/s) is not a risk factor for SRGP as a risk factor for SRGP (-0.51, -1.00 to -0.01, Figure 7f).

Abdominal muscle characteristics

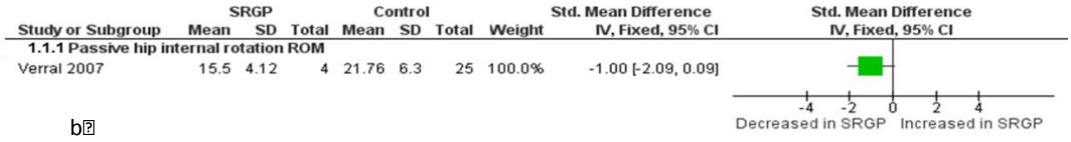
Retrospectively, there was limited evidence from one HQS (Jansen et al., 2010) of a decrease of transversus abdominis (TrA) muscle thickness at rest in participants with right-sided (-0.80, -1.32

to -0.28, Figure 7g) and left-sided SRGP symptoms (-1.05, -1.58 to -0.51, Figure 7g). One HQS (Cowan et al., 2004a) reported a delay in TrA activation onset during active straight leg raise task associated with SRGP, but adequate data was not available to complete SMD calculations.

One study (Jansen et al., 2010) additionally reported no change in TrA thickness during active straight leg raise (ASLR) and bilateral isometric adduction test; and internal and external oblique muscle thickness at rest, ASLR or bilateral isometric adduction associated with SRGP, but adequate data was not available to complete SMD calculations.

One study (Cowan et al., 2004b) reported no change in internal oblique and rectus femoris muscle activation onset timing during ASLR associated with SRGP, but adequate data was not available to complete SMD calculations.

a



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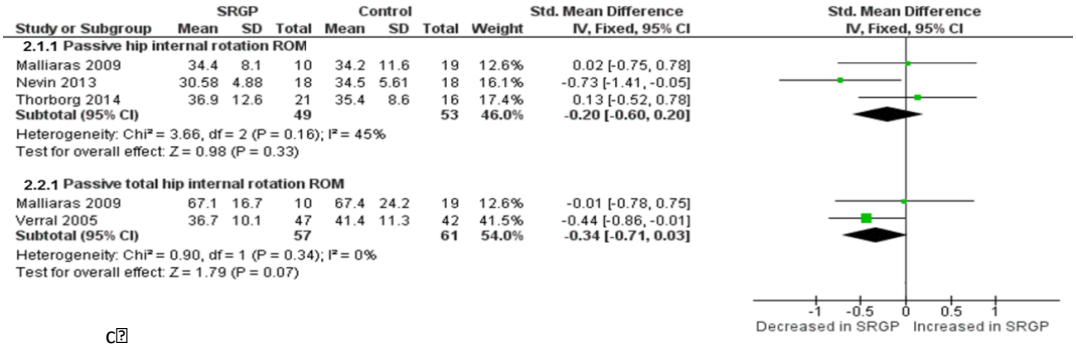

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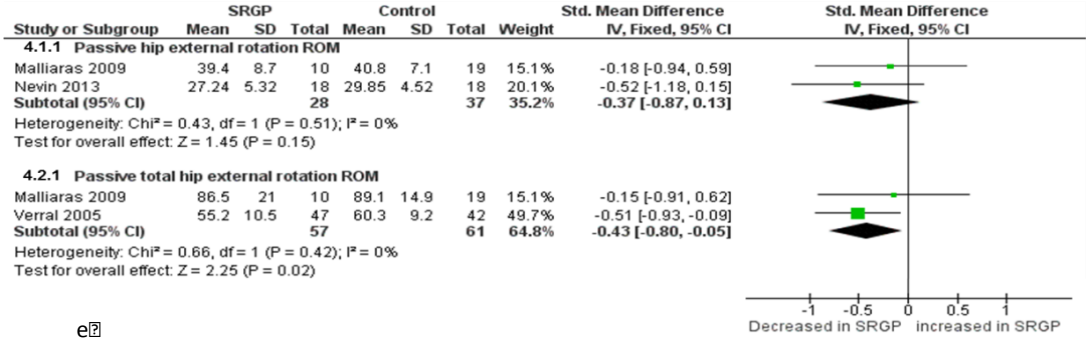
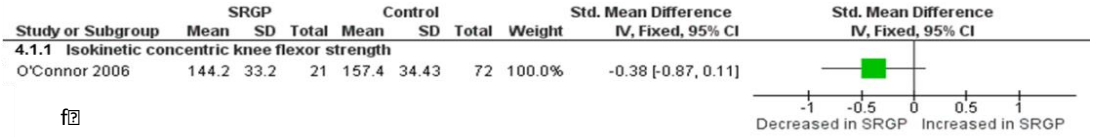
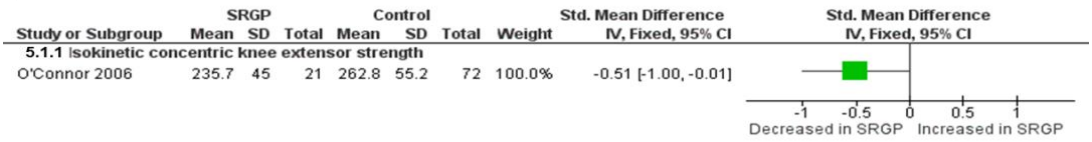


e



f



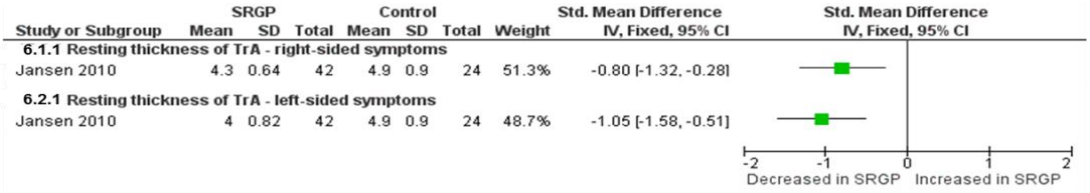


Figure 7: Forest plot detailing the analysis of other movement and muscular functions: a - hip internal rotation range of movement prospective results, b – hip internal rotation range of movement retrospective results, c – hip external rotation range of movement prospective results, d – hip external rotation range of movement retrospective results, e – knee flexor muscle strength prospective results, f – knee extensor muscle strength prospective results, g – transversus abdominis muscle thickness retrospective results.

Muscle	Feature	Pro/Retro	Studies missing in pooled results	Studies included in pooled results	Included studies quality	Specific criterion	Pooled result/Calculated SMD	Evidence
Adductor	Strength	Pro	Emery, 2001	O'Connor, 2004	HQ	Isokinetic hip adduction in 0.52 rad * s-1	No change	Limited
			Engebretsen, 2010	O'Connor, 2004	HQ	Isokinetic hip adduction in 2.08 rad * s-1	Decrease in SRGP	Limited
			Crow, 2010					
			Tyler, 2001	O'Connor, 2004	HQ	Isokinetic hip adduction in 3.66 rad * s-1	No change	Limited
		Retro		Malliaras, 2009	HQ	Squeeze test 0°	Decrease in SRGP	Limited
				Malliaras, 2009	HQ	Squeeze test 30°	Decrease in SRGP	Limited
				Jansen, 2010	HQ	Squeeze test 45°	Decrease in SRGP	Strong
				Malliaras, 2009	HQ			
				Mens, 2006	HQ			
				Nevin, 2014	LQ			
				Mohammad, 2014	LQ	Isokinetic concentric hip adduction	No change	Very limited
				Thorborg, 2014	HQ	Isometric hip adduction	No change	Limited
				Thorborg, 2014	HQ	Eccentric hip adduction	Decrease in SRGP	Limited
	Flexibility	Pro	Emery, 2001	Arnason, 2004	HQ	Unilateral abduction flexibility test	No change	Moderate - not homogenous
				Tyler, 2001	HQ			
		Retro		Malliaras, 2009	HQ	Right bent knee fall out	Increase in SRGP	Moderate
				Nevin, 2014	LQ			
				Thorborg, 2014	HQ	Unilateral abduction flexibility test	No change	Limited
	Peak torque angle	Pro		O'Connor, 2004	HQ	Peak torque angle	No change	Limited
Abductors	Strength	Pro		O'Connor, 2004	HQ	Isokinetic hip abduction in 0.52 rad * s-1	Decrease in SRGP	Limited
				O'Connor, 2004	HQ	Isokinetic hip abduction in 2.08 rad * s-1	No change	Limited
				O'Connor, 2004	HQ	Isokinetic hip abduction in 3.66 rad * s-1	No change	Limited
		Retro		Malliaras, 2009	HQ	Isometric hip abduction	No change	Strong
				Thorborg, 2014	HQ			
				Mohammad, 2014	LQ	Isokinetic concentric hip abduction	No change	Very limited
Relationship between abductor and adductor muscles	Strength	Retro		Mohammad, 2014	LQ	Isokinetic concentric hip adductor vs. abductor strength	No change	Very limited
				Tyler, 2001	HQ	Isometric hip adductor vs. abductor strength	Decrease in SRGP	Limited

	Activation	Retro		Morrissey, 2012	HQ	Moving leg - early phase of SHF	Decrease in SRGP	Limited
				Morrissey, 2012	HQ	Moving leg - middle phase of SHF	No change	Limited
				Morrissey, 2012	HQ	Moving leg - late phase of SHF	No change	Limited
				Morrissey, 2012	HQ	Weight-bearing leg - early phase of SHF	Decrease in SRGP	Limited
				Morrissey, 2012	HQ	Weight-bearing leg - middle phase of SHF	Decrease in SRGP	Limited
				Morrissey, 2012	HQ	Weight-bearing leg - late phase of SHF	Decrease in SRGP	Limited
Hip flexors	Strength	Retro		Mohammad, 2014	LQ	Isokinetic concentric hip flexion	Increase in SRGP	Very limited
				Thorborg, 2014	HQ	Isometric hip flexion	No change	Limited
				Thorborg, 2014	HQ	Eccentric hip flexion	No change	Limited
	Flexibility	Pro		Arnason, 2004	HQ	Modified Thomas's test	No change	Limited
		Retro		Thorborg, 2014	HQ	Modified Thomas's test	No change	Limited
Hip extensors	Strength	Retro		Mohammad, 2014	LQ	Isokinetic concentric hip extension	No change	Very limited
Relationship between flexor and extensor muscles	Strength	Retro		Mohammad, 2014	LQ	Isokinetic concentric hip flexion vs extension	No change	Very limited
Hip rotation ROM	Hip internal rotation	Pro	Ibrahim, 2007	Verral, 2007	LQ	Passive hip internal rotation test	No change	Very limited
		Retro		Nevin, 2014	LQ	Passive hip internal rotation test	No change	Strong
				Thorborg, 2014	HQ			
				Malliaras, 2009	HQ			
				Malliaras, 2009	HQ	Passive total hip internal rotation (sum of both legs)	No change	Moderate
				Verral, 2005	HQ			
	Hip external rotation	Pro	Ibrahim, 2007	Verral, 2007	LQ	Passive hip external rotation test	No change	Very limited
		Retro		Nevin, 2014	LQ	Passive hip external rotation test	No change	Strong
				Malliaras, 2009	HQ			
				Malliaras, 2009	HQ	Passive total hip external rotation test (sum of both legs)	Decrease in SRGP	Strong
				Verral, 2005	HQ			
Knee extensor	Strength	Pro		O'Connor, 2004	HQ	Isokinetic knee extension	No change	Limited
Knee flexor	Strength	Pro		O'Connor, 2004	HQ	Isokinetic knee flexion	Decrease in SRGP	Limited
Transversus abdominis	Thickness	Retro		Jansen, 2010	HQ	Resting thickness - right-sided symptoms	Decrease in SRGP	Limited
						Resting thickness - left-sided symptoms	Decrease in SRGP	Limited

Table 4: Table summarising all studies included in this systematic review, findings and levels of evidence.

Discussion

This systematic review and meta-analysis synthesised 17 studies, including 8 prospective and 9 retrospective, which investigated changes in movement and muscle function in professional and amateur athletes with SRGP. Overall, there was conclusive evidence that measurable differences in movement and muscle function factors exist in athletes with SRGP – some of which may precede and increase the risk of developing injury (Table 4). The findings should be considered by clinicians when designing rehabilitation and screening programmes (Table 5).

There were some strong findings emerging from the evidence synthesis. The most notable, supported by strong or moderate evidence (Table 4), were retrospective associations between existing SRGP and: adductor muscle weakness, increased abduction flexibility (bent knee fall out) and decreased internal and external rotation range of movement. These results should be particularly considered when designing rehabilitation programmes for athletes with established SRGP. Prospectively, a paucity of evidence and data is available to complete meta-analysis, but limited evidence indicates reduced hip adduction strength may be a risk factor for SRGP development. Additionally, it is worth noting that numerous studies also reported hip abductor strength deficits as a risk factor for SRGP development, but could not be included in the meta-analysis due to a lack of reported data and response requesting additional data from corresponding authors. Nonetheless, hip abduction strength deficits should be particularly considered in screening programmes.

Methodological considerations of included studies

There have been numerous attempts to introduce a common classification system for diagnosing SRGP (Holmich, 2007, Mens et al., 2006, Falvey et al., 2009), which I have not added to but have instead combined pragmatically in order to enable review. All but one study (Mohammad et al., 2014) provided clear diagnostic criteria. There was heterogeneity of SRGP

definitions, with 11 subtly different diagnostic criteria being identified. This may limit the strength of the review, but the similarities between classifications mean I am confident our review is sufficiently robust with each study using similar inclusion criteria regardless of diagnostic term. For example, both Morrissey et al. (2012) and Malliaras et al. (2009) use an anatomical location of pain analysis alongside resisted movement tests and passive joint stress tests to differentially diagnose adductor tendinopathy with respect to hip joint pathology. They differ in that Malliaras et al. (2009) additionally assessed the symptoms during functional task such as agility drills, but these differences are relatively minor. Very similar inclusion criteria, based mainly on the palpatory pain of the adductor muscle, tendon or insertion area, and reproduction of symptoms during resisted hip adduction, are presented by Cowan et al. (2004), Jansen et al. (2010), Morrissey et al. (2012) and Thorborg et al. (2014). Interestingly, the diagnostic term is different in all studies: long standing groin pain (Cowan et al., 2004a), adduction related groin pain (Jansen et al., 2010), chronic groin pain (Morrissey et al., 2012a) and adductor related groin pain (Thorborg et al., 2014). There is no question that initial recent attempts to establish international consensus on groin pain nomenclature should reduce confusion and lack of agreement regarding this issue. Potentially, future pathophysiological validity studies would help move clinical practice and research forward by enabling more robust result collation via shared nomenclature.

Measurement protocols for each specific movement and muscle function variable also varied across the included studies. For example, for measurement of adductor muscle strength, three studies used hand-held dynamometers (Jansen et al., 2010, Mens et al., 2006, Thorborg et al., 2014), two used sphygmomanometers (Nevin and Delahunt, 2013, Malliaras et al., 2009) and one used an isokinetic dynamometer (Mohammad et al., 2014). Additionally, one study using a hand-held dynamometer used it in two contraction types: isometric and eccentric (Thorborg et al., 2014). Further research is needed about the validity of each measure and consensus about

optimal methods would again improve both research synthesis and clinical translation. Additionally, variation in outcome measures and methodology across included studies limited the potential for data pooling.

Although I included only studies investigating movement and muscle function factors in athletic populations, this included varied sports disciplines and participation levels. This is both a strength and a potential weakness of our synthesis, as data pooling in such heterogeneous groups entails combining results from cohorts who have different sports specific training and participation volume. While these factors are highly likely to influence injury risk and presentation profile, it was nonetheless judged that the pooling conducted was valuable to strengthen the review findings considering the paucity of research currently available for each group. This may need to be re-considered once the volume of work is sufficient at different sporting levels and in different disciplines.

Interpreting the results of prospective studies was complicated by a lack of methodological clarity in manuscripts; for example testing dominant or non-dominant limb, moving or not moving, left or right, and inured or uninjured (Arnason et al., 2004, Emery and Meeuwisse, 2001, Engebretsen et al., 2010, Ibrahim et al., 2007, Tyler et al., 2001). The most accessible approaches (O'Connor, 2004, Verrall et al., 2007a) clearly measured and compared dominant and non-dominant sides. Additionally, only some retrospective studies were clear about the side of measurements (Cowan et al., 2004a, Jansen et al., 2010, Morrissey et al., 2012a, Nevin and Delahunt, 2013, Thorborg et al., 2014, Verrall et al., 2005a). Given that unilateral symptoms can reflect bilateral biomechanical dysfunction, it would be our recommendation that future work examines movement on both sides, under any and all conditions assessed – and analyses data with reference to both symptom and dominance. In this review, however, I chose to analyse the data from dominant *or* right leg only, in order to maintain the consistency of the analysis despite different ways of presenting the data by individual authors.

Very few retrospective studies attempted to blind the measurement assessor (Engebretsen et al., 2010, Malliaras et al., 2009, Thorborg et al., 2014, Verrall et al., 2007a) and only one study reported detailed sample size and power calculations (Nevin and Delahunt, 2013).

Five studies (Cowan et al., 2004a, Engebretsen et al., 2010, Morrissey et al., 2012a, Tyler et al., 2001, Verrall et al., 2005a) did not report the reliability of the measurements in the assessors' hands. Addressing these methodological limitations in future research is needed to improve confidence in findings and subsequently 'levels of evidence' which can be concluded.

Surprisingly, some studies (Crow et al., 2010, Emery and Meeuwisse, 2001, Engebretsen et al., 2010, Ibrahim et al., 2007, Tyler et al., 2001, Verrall et al., 2005a) did not provide basic anthropometric data such as age, height and weight, which limits the external applicability of findings and can be critical confounding factors, or co-variates, in biomechanical research. In particular, factors such as strength and muscle activation may clearly depend on the individual athlete's fitness and muscle morphology. In order to avoid a potentially significant source of bias, all studies investigating biomechanical factors should accurately measure these factors and include them in analysis.

Coronal plane muscle activation and strength

Adductor muscles

There is common agreement that the main muscles affected by SRGP are the hip adductors (Holmich, 2007, Crow et al., 2010), an assertion confirmed by eleven studies reporting decreased adduction strength associated with groin pain symptoms. Overall there is strong evidence of an association between adductor muscle weakness and SRGP. Meta-analysis results showed strong evidence of adductor muscle weakness after the SRGP onset, but only when measured by squeeze test in 45° of hip flexion. This may indicate the importance of testing the groin symptoms using this particular test, which seems most sensitive to detect

strength deficits in athletes with SRGP. There was limited evidence of decreased adduction strength prior to SRGP onset. It is important to note that there were four other prospective studies (Crow et al., 2010, Emery and Meeuwisse, 2001, Engebretsen et al., 2010, Tyler et al., 2001) reporting adductor muscle weakness prior to the onset of SRGP, but presentation of the data in those studies did not allow for data pooling. Adductor muscle weakness in the pre-season was associated with SRGP onset indicating that strengthening of this muscle group may be a key component of prevention. Crow et al. (2010) reported decreased adductor muscle strength two weeks prior to SRGP onset, but no earlier, suggesting a potential neuro-inhibitory mechanism for altered adductor motor output immediately before or at the time of pain onset for some athletes rather than long-standing weakness. Clinicians should consider implementing prevention strategies based on adductor strength screening findings.

Six studies investigated the association between abduction flexibility and SRGP (Arnason et al., 2004, Emery and Meeuwisse, 2001, Malliaras et al., 2009, Nevin and Delahunt, 2013, Thorborg et al., 2014, Tyler et al., 2001) and only one retrospective LQS reported a significant association (Nevin and Delahunt, 2013). However, *pooled* results show moderate evidence that abduction flexibility was not changed before, but increased after SRGP onset, measured with the bent knee fall out test.

The reason for such changes is not clear. There may be a relationship between optimal hip abductor flexibility and SRGP, with too much flexibility being problematic. It is worth noting, however, that the flexibility increase was noted only during the bent knee fall out test, which is a combination of abduction and external rotation flexibility test. It is possible that this flexibility increases following pain onset, perhaps with rehabilitation or due to reduced participation and therefore reduced stress. This may remove the impact of compensations for adductor weakness prior to symptom onset. This could be questioned, as some athletes may have joint factors that explain restriction, which could also change, on this timescale due to

reduced joint loading. Further, there may be an interaction between joint load, increased flexibility and sports participation volume. Further research is needed to elucidate the relationship these factors, with such work having the potential to clarify aetiology.

Abductor muscles

There is a commonly held belief that SRGP might be at least partly due to muscle imbalance in the pelvic girdle area and, consequently, sub-optimal loading on groin structures (Morrissey et al., 2012a, Renstrom and Peterson, 1980). There is an association between decreased hip abduction strength and SRGP observed in prospective, but not retrospective studies (Malliaras et al., 2009, Thorborg et al., 2014, O'Connor, 2004, Mohammad et al., 2014). It is plausible that there is a weakness of hip abductors preceding SRGP onset, which disappears following pain onset or subsequent rehabilitation. This rehabilitation may be particularly important for gluteus medius muscle which is thought to have a primary stabilising function (Grimaldi, 2011), and should be considered in future research.

Relationship between abductor and adductor muscles

A prospective study by Tyler et al. (Tyler et al., 2001) reports a significant decrease in adduction in relation to abduction strength associated with SRGP in professional (ice hockey) players, while Morrissey et al. (Morrissey et al., 2012a) found a decrease in GM:AL activation in amateur footballers. The relationship between muscle strength and activation is not linear (Kamen and Gabriel, 2010b). Therefore, although seemingly contradictory, if the abductor muscles are weaker they may need to increase activity to achieve their function of pelvic girdle stability. Additionally, GM activity were measured during a standing hip flexion movement (a functional task), whereas strength measurements were obtained using a maximal voluntary contraction break test and isolated hip abduction task (Tyler et al., 2001). These measures clearly investigate different aspects of the strength construct in a functional versus non-

functional task. Research designs that include muscle activation in functionally relevant tasks and strength measures are needed to broaden our understanding of how different aspects muscle function can be affected in SRGP.

Horizontal plane hip movement

Strong evidence of a decrease in hip total external rotation range of movement after the SRGP onset was the only significant finding in horizontal plane hip movements. It is not clear whether this ROM limitation have muscular or articular origin, and there might be a number of reasons why it exists. For example, hip rotation restriction may follow increased hip joint loading due to muscle imbalance around the hip (e.g. reduced abductor strength). Decreased ROM in athletes may also be related to underlying hip joint injury, which may be asymptomatic. Limitation of rotation ROM is clearly an area that requires further research in athletes with SRGP, as a clear distinction needs to be made between articular and muscular movement restrictions.

Other muscle function and architecture features

A decrease in TrA thickness and delayed onset during movement was found to be associated with SRGP. Cowan's high quality study reported delayed TrA activation in relation to the 'prime mover' in a straight leg raise manoeuvre (Cowan et al., 2004b, Jansen et al., 2010), while Jansen's group reported reduced relaxed cross sectional area. These findings suggest that muscle dysfunction in SRGP is not limited to hip muscles and TrA function may be an important prevention and rehabilitation consideration in some affected athletes.

Clinical variable assessed		Finding	Clinical takeaway	
Parameter	**Feature**	**Headline result**	**Include in screening [prospective findings]**	**Include in rehabilitation [retrospective findings]**
Adductor	**Strength**	Decreased in SRGP	✓	✓✓✓
	Flexibility	Increased in SRGP		✓✓
Abductor	**Strength**	Decreased in SRGP	✓	✓
Relationship between abductors and adductors	**Strength**	Decreased in SRGP		✓
	Activation	Decreased in SRGP		✓
Hip flexor	**Strength**	Increased in SRGP		✓
Hip rotation ROM	**Hip external rotation**	Decreased in SRGP		✓✓✓
Knee flexor	**Strength**	Decreased in SRGP	✓	
Transversus abdominis	**Thickness**	Decreased in SRGP		✓

Table 5: Table summarising the clinical implications emerging from this review; ✓✓✓ indicates strong evidence, ✓✓ indicates moderate evidence, ✓ indicates limited or very limited evidence; SRGP – sports related groin pain.

Clinical implications and future directions

In this section, I summarised the muscular and movement alterations associated with SRGP that could be considered during development of rehabilitation and prevention programmes. The strongest prospective risk factor from this review was reduced hip adductor strength, which should be considered for inclusion in pre-season screening programs. There is some indication for more regular screening of adductor strength in some environments (e.g. elite sport) given it may precede pain onset by 2 weeks in some individuals who go onto develop SRGP (Crow et al., 2010), although further studies in elite and other athletic populations are needed to confirm this finding. Recommendations for adductor muscle strength measurement and treatment strategies are well described. They include squeeze and unilateral resisted adduction tests to establish any potential strength deficits; and various exercises of graduated difficulty to restore them, such as squeezing the ball between knees in the early phase of rehabilitation and moving to long lever (ball between the feet) and open kinetic chain strengthening exercises using resistance devices as rehabilitation progresses (Weir et al., 2011b, Holmich et al., 2010). Other factors preceded groin pain onset but the evidence was limited. These included decreased hip abductor muscle strength, and decreased knee extensor strength, indicating screening for and addressing identified deficits may reduce the incidence of SRGP. The most effective interventions for addressing hip and knee muscle function deficits and whether they decrease the incidence of groin pain warrant further investigation. Restriction in hip external rotation range of movement, in athletes with SRGP, may be critical due to the requirement for sufficient range of hip movement for adequate load absorption during change of direction activities (L'Hermette et al., 2006). Clinicians should identify whether the underlying cause of possible deficits in hip rotation ROM is articular or muscular. If muscular restriction is present, specific techniques including stretching, soft tissue work as well as using the entire range of movement in sports-specific tasks during the end phase of

rehabilitation should be considered. Articular restriction may be less likely to change with these interventions, and end range loading may even provoke symptoms (Ratzlaff et al., 2013). This may partly explain why addressing flexibility specifically (e.g. stretching, soft tissue techniques) is less of a feature of current groin rehabilitation and prevention programs than adductor and other muscle strengthening (Holmich et al., 1999, Weir et al., 2010, Weir et al., 2011b).

This review has shown that despite a lack of clear SRGP treatment guidelines, there is in fact a paucity of studies investigating the biomechanical patterns in SRGP. Studies summarised in the chapter mainly focus on local, simple biomechanical measures such as strength, flexibility and range of movement. It is important to note, that presented studies seem to show consistent and similar results, clearly indicating that specific characteristics of the athlete with SRGP exist. However, this review has also highlighted that there are very few studies that have investigated more sophisticated biomechanical measures, such as muscle activation or kinematic imbalances. Additionally, only one study has measures those deficits during functional movement tests, which seem very relevant given the relation between SRGP and specific movements that predispose certain sports groups to become injured.

The assessment and treatment options for potential pelvic movement control deficits are not well established and certainly require further investigation. We recommend careful clinical assessment of functional movements such as standing hip flexion (Morrissey et al., 2012a) or single leg squat which reflect common movements in sports possessing a high incidence of SRGP and load the pelvis in a relevant fashion. These functional tasks are also relatively easily controlled, compared to cutting manoeuvres, and therefore have the potential to reveal characteristic biomechanical signatures of SRGP.

Conclusions

Our review identified a range of movement and muscle function features that can be prospectively identified in a range of athletes who subsequently develop SRGP and should be considered in screening programmes (Table 5). These findings provide clear clinical guidance that should be implemented in prevention and rehabilitation of athletes with SRGP.

Mainly hip adductors and knee flexor strength deficits should be screened and addressed as they may be risk factors for SRGP.

Further, this review identified both muscle function features and range of movement considerations, clearly shown by retrospective studies that should be considered in rehabilitation programmes (Table 5). In particular, adductor muscle weakness and increased abduction flexibility, hip total external rotation deficits, imbalances between adductor and abductor muscles, increased hip flexor strength and transversus abdominis muscle thickness should be addressed in rehabilitation programmes. The lack of consistency about various classification issues, alongside methodological heterogeneity also need to be addressed in order to optimally move the evidence base forward.

It is worth noting, that despite the agreement of a multi-directional and multi-structural nature of SRGP, only one study investigated more sophisticated and more holistic signatures of SRGP. Further research should therefore focus not on exact diagnosis of the tissues, but on more general outcome measures, which may be applicable clinically –thus muscle activation and movement patters seem to be a relevant target for investigation.

Finally, the literature is notable for the near complete lack of research on SRGP-related movement pattern differences during functional movements and also for comparison between sporting participation levels. These aspects will be addressed further in the forthcoming chapters.

Chapter 3: Aims and hypotheses

Chapter overview

This chapter summarises the main aims of the thesis by outlining null hypotheses for each study. It summarises the aim of the whole thesis, as well as for the separate chapters – alternative hypotheses being provided separate in each subsequent chapter.

Overall aim

The overarching aim of this thesis was to explore biomechanical factors associated with SRGP in order to determine whether they should be considered in improved rehabilitation and prevention paradigms, and if so, in what ways they may be beneficial. Allied to this primary research question were subsidiary questions exploring whether there was evidence of sports- and participation-level specificity.

Null hypothesis 1 – Reliability study

Coronal plane muscle activation measured with surface electromyography, and hip joint kinematics measured with 3D motion capture system, as well as the methods of data analysis during standing hip flexion and single leg squat movement manoeuvres would not be reliable between testing occasions.

Null hypothesis 2 – Observational study

There would be no consistent coronal plane muscle activation and movement pattern differences present when comparing athletes with sports related groin pain to well-matched, healthy controls, regardless of discipline and level of sport.

Null hypothesis 3 – Longitudinal study

Coronal plane muscle activation and hip joint kinematic patterns in athletes after an acute groin injury would not be altered by rehabilitation irrespective of the clinical signs of recovery.

Chapter 3: Methods

Chapter overview

The development of the methodology for the studies was based on the paper published previously by our group (Morrissey et al., 2012a). However, many aspects of the inclusion and exclusion criteria, the data collection and data analysis processes were further developed during my PhD.

The methodology for each study included in this thesis (observational and longitudinal) differed in details, regarding mainly the inclusion and exclusion criteria of the participants and the number of testing occasions. Those differences between studies are clearly described throughout this chapter. However, the main methodological consideration regarding the data collection, processing and analysis remain similar and consistent for all studies and are described in details.

The first part of the chapter ('Research protocol overview') provides a concise description of the data collection, data processing and data analysis overview. This particular part of the chapter aims at describing the generic data collection process subsequently implemented in the thesis, and provides only limited details regarding the decision-making of the given collection, processing or analysis methods for clarity. Those details are thoroughly discussed further in the chapter.

The second part ('Participants') describes the aspects associated with the study participants: ethical approval and potential ethical issues associated with the studies; the recruitment process and issues associated with it; the participant inclusion and exclusion criteria, and the rules of defining the dominance of participants' leg.

The third part of the chapter ('Measurement method') firstly describes the surface electromyography (sEMG) as a method of collecting the muscle activation data. This part includes the general considerations regarding different types of the EMG, introduces the chosen method and discusses its advantages and disadvantages. Further, it describes the reliability of the method and factors that may influence it as well as discusses. This part also describes the methodology of collecting and processing the kinetic and kinematic data.

The fourth part of the chapter describes the stages of analysis of the collected data. It describes the movement manoeuvres chosen to collect the data and the method of dividing the movements into stages in order to enable the statistical comparison of the data.

The last part of the Methods chapter presents a detailed analysis of the data processing techniques. A graphical presentation of a worked example of a sample data on each stage of data processing, alongside the justification of choosing certain processing techniques, facilitates the understanding the rationale of the chosen methods.

Research protocol overview

All studies presented in the thesis had ethical clearance, and all participants signed an informed consent before the data collections process.

All participants filled in study questionnaires and underwent a clinical examination in order to be included in the study as an injured or control participant. Injured participants of the longitudinal study underwent more detailed clinical examination and VAS scores were recorded during each testing occasion.

Surface electromyography (sEMG) electrodes and CodaMotion infra-red markers were placed on the pelvic, hips and lower limbs of each participant. After that, the participant was asked to

perform two movement manoeuvres: standing hip flexion (SHF) and single leg squat (SLS), each manoeuvre being performed three times on each leg.

Kinematic data was filtered, and sEMG data was rectified, smoothed and filtered before analysis. There were four outcome measures: gluteus medius versus adductor longus muscle activation magnitude ratio, and hip joint rotations in three planes: coronal, sagittal and horizontal; calculated separately for the injured and uninjured players of each sports discipline.

Movement manoeuvres were divided into exclusive, clinically relevant, phases prior to statistical comparison, with SHF being divided into three phases while SLS was divided into seven phases.

Each variable was averaged within each phase for each leg for each participant, considered separately if it was moving or in stance, then group comparisons between the injured and uninjured athletes were performed.

Participants

Ethics

All studies presented in the thesis were approved by the Queen Mary University of London Ethics of Research Committee. The ethical application for approval including the Participant Information Sheet and Informed Consents are enclosed in Appendix 5 for the observational study (p. 288) and in Appendix 7 for the longitudinal study (p. 308); the letters of ethical approval are enclosed in Appendix 4 for the observational study (p. 287) and in Appendix 6 for the longitudinal study (p. 307). NHS ethical approval was not necessary for the studies finally included in the thesis due to the specificity of investigated cohort as I aimed in recruiting amateur and professional athletes suffering from symptoms associated with their sports discipline rather than patients recruited via NHS.

A few ethical issues needed to be carefully considered and addressed before applying for the ethical approval for the studies.

Firstly, I ensured that all potential study participants take part in the study voluntarily and that they are aware that they may withdraw from the study at any point, without giving any reason for such decision, with no consequences. I have emphasised it verbally several times before the potential participants signed the informed consent, such statement was also included in the Participant Information Sheet, which the potential participants were encouraged and given time to read before signing the consent.

Second potential ethical issue of the study was the participants' anonymity, which was solved by applying the coding system of the study participants. Outcomes of the clinical examination, description of symptoms and collected data were recorded and stored using Participants' codes. Only the informed consents were signed by the participants with their names. The description of the coding system, which provided the link between the participants' personal details and their coding number, was stored in a locked cabinet based in a locked PhD students office and locked Laboratory. I was the only person who had the keys to the cabinet; only a limited number of Centre for Sports and Exercise Medicine staff members own the keys to the Laboratory and to the PhD students' office.

The preparation of the participant for the data collection process required uncovering certain areas of his body: upper thighs, groin, buttocks, lower back and lower abdomen; lower back, lower abdomen and upper thighs also had to be uncovered throughout the data collection process. This may have potentially caused participant's discomfort. Firstly, the participant had to undergo a clinical examination consisting of palpation and specific clinical tests focusing on their hip, groin and abdominal area. Then, the sEMG electrodes were placed on above mentioned parts of participant's body in order to obtain the muscle activation data; further,

CodaMotion markers were placed on participant's body. Finally, the entire data collection process had to be performed with the participant's abdomen and lower limbs uncovered in order to maximisee the visibility of the CodaMotion markers.

The nature and necessity of those procedures were emphasised in writing in Information Sheet, understanding and agreement were confirmed prior to signing the informed consent. In order to provide a professional clinical approach to the participants, all procedures were performed by me - a Senior Physiotherapist, a member of Chartered Society of Physiotherapy and the Health and Care Professions Council; I have also obtained a Disclosure and Barring check prior to the data collection. However, if agreed with the study participant, the clinical examination, electrode placement and data collection process were performed by 4th year medical students under my close supervision.

In order to minimise participants' discomfort during their preparation for the data collection process, the clinical measurements and electrodes placement on participant's body were always performed in a presence of at least two people behind the screen or in a separate ro om in the Human Performance Laboratory (HPL), which was locked throughout this process with the windows fully covered.

For the data collection event, all participants were asked to bring their own shorts to the HPL. If they failed to do so, they were provided with a suitable pair.

Another ethical consideration was associated with the necessity of provoking pain during clinical examination when checking participants' eligibility for the study. In order to ensure that the pain or discomfort during is minimal, the examination was performed in a careful and delicate way by me or a 4th year medical students under my close supervision.

Recruitment

The recruitment process of the professional and amateur athletes was one of the biggest challenges in the data collection process. No study included in this thesis was externally or internally funded, so the potential participant had to agree to partake in the study with no refund for their time, travel and effort.

The potential participants were contacted via private contacts, friends and colleagues as well as using the contact details found in the web. Participants to be included in control group were recruited in similar ways and were closely matched with the SRGP athletes in order to avoid the bias arising from confounding factors (weight, height, age, position played, but also training type, access to physiotherapy and personal training service or the level and frequency of play).

Careful matching of the control and symptomatic participants was one of the priorities in all included studies. Age, weight and height are typical sources of potential bias in biomechanical measurements, as the differences of these anthropometrical features between control and symptomatic groups may affect the sEMG values.

The athlete's position played on the pitch, among other mentioned factors, was treated with particular care, as in some sports the athletes playing in different positions may perform different movements. For example in rugby, while forwards perform repetitive twisting, cutting and pivoting and are frequently exposed to high loading in potentially very traumatic situations, backs perform additional kicking alongside running and changing directions.

Matching of the symptomatic athletes and controls was a relatively difficult task due to simultaneous recruitment process of both groups. In consequence, the mean anthropometrical values of the SRGP athletes had to be closely monitored along the recruitment and data collection process, which allowed the recruited healthy controls to present similar mean values

in height, weight and age. Fortunately, given a high interest of healthy athletes in participation in the studies, it was possible to select the participants to match the desired values.

The sample size calculation for each separate study was based on previous study by Morrissey et al (2012). Morrissey et al. recruited nine injured and nine healthy athletes, which proved to be a number large enough to observe significant differences in gluteus medius to adductor longus muscle activation ratio in standing hip flexion movement.

Based on the sample size estimation equation (Kadam and Bhalerao, 2010, Kirby et al., 2002):

where $Z\alpha$ is a constant number depending on the acceptance of the Type I error and whether the effect is one-sided or two-sided, according to the table presented below:

α-error	**5%**	**1%**	**0.10%**
2-sided	1.96	2.5758	3.2905
1-sided	1.65	2.33	

$Z_{1-\beta}$ is constant depending on the accepted power of the study as shown in the table below

Power	**80%**	**85%**	**90%**	**95%**
Value	0.8416	1.0364	1.2816	1.6449

σ is the standard deviation, which is estimated for the study, but may be retrieved from previous similar study;

Δ is a difference in effect between the injured and control groups.

Based on the formerly mentioned study by Morrissey et al. (Morrissey et al., 2012a), I chose the values for the formula:

$Z\alpha$ - 2.5758

$Z_{1-\beta}$ - 1.2816

σ – there were twelve different standard deviation values to be obtained from this study as there were two groups of participants (injured and control); the measurements were taken during the moving and weight-bearing phase of standing hip flexion movement (SHF); and SHF was further divided into three movement phases: early, middle and late. All of the values are listed below, sorted from the lowest to the highest value:

0.3267
0.3368
0.3912
0.4074
0.4269
0.48
0.5416
0.5596
0.5973
0.6186
0.7005
0.8182

I decided to use the median of all of those values for further calculations of the sample size for my study, which was 0.5108.

Δ also needed to be calculated, with six different values (three phases of movement during moving and weigh-bearing conditions), presented below, sorted from the lowest to the highest value:

276.435%
40.47934%
5.995547%
123.6475%
189.5781%
97.46572%

In this case I again decided to use the median of those values, which was 110.5566 (Δ = 1.105566).

Using all of the above values, the sample size calculations are presented below:

Above calculations suggest that recruiting eight participants in each study group would be an optimal number in order to reach the statistical significance of the results. However, very high differences between injured and control groups in the study which served as a base for the calculations (Morrissey et al., 2012a) may be associated with other factors.

One of the most important potential sources of bias was the amateur level sporting population recruited for Morrissey's study. This may mean that the participants have a higher variability of the training regime, general fitness and access to professional medical advice, and, in consequence, the reactions to pain may be emphasised. I aimed in recruiting mainly the professional athletes, who were potentially more equal in regards to those potential sources of bias; their general fitness and training regime is likely to be similar, professional medical help and advice is easily accessible. They professionals may also potentially show less change in muscle activation and movement patterns in the presence of pain, due to their better fitness levels. Therefore I decided to aim in a higher number of participants in studies focusing on

professional athletes, with the estimated number of ten in each group in each study (symptomatic and asymptomatic).

Inclusion and exclusion criteria

The definition of clear, evidence based and clinically relevant inclusion and exclusion criteria for study participants was one of the priorities before the data collection process. At the time of the data collection, there was a lack of international consensus among researchers regarding underlying pathology of groin symptoms, as well as its various diagnoses and terminology. A common agreement, however, exists that the nature of groin pain is multi-structural and multi-factorial.

In this thesis, an umbrella term 'sports-related groin pain' (SRGP) is proposed and used throughout. It includes the soft-tissue, but not hip joint diagnoses causing groin pain in athletes, which may be defined differently by the aforementioned classification systems. My hypothesis was that the multi-factorial and often misdiagnosed nature of groin symptoms would cause similar movement strategies in injured participants, which were associated with the symptoms rather than with diagnosis per se. This term, although defined prior to the Doha agreement (Weir et al., 2015), is consistent with one of the diagnostic and terminology subgroups specified there.

The athlete had to be over 18 in order to participate in *observational* and *longitudinal* study. 18 was selected as a minimum age in observational and longitudinal study firstly to ensure the participants were capable of making independent decisions whether to voluntarily take part in the study; secondly to ensure their physical maturity and avoid bias related to developmental imbalances.

In order to be included in the *observational* study, a potential participant must have been experiencing sports related groin pain (SRGP) for a minimum of four weeks. This time frame

was decided to be long enough to consider the symptoms as 'chronic'; it was also previously used in other studies (Mens et al., 2006, Morrissey et al., 2012a) and proved to be a sufficient period to demonstrate the significant biomechanical differences between the symptomatic and asymptomatic participants. Moreover, from a physiological perspective, four weeks after the symptoms onset is beyond the acute inflammation phase and into the intensive rehabilitation phase of injury, to which the study results are most relevant. Additionally, four weeks of pain is a long period of time in professional and amateur sports. Non-participation in training and game sessions may significantly decrease the levels of general and sports-specific fitness; it may also have serious consequences for the performance of the whole team, as well as may increase the players' risk of other injuries (Arnason et al., 2004).

In the *longitudinal* study, groin symptoms must have started as an effect of acute sports-related incident. The potential participant must have been able to visit the Human Performance Laboratory to collect the first set of data a maximum of five days after the injury. This time frame was chosen to enable retrieval of information from the activation of the injured muscle as early as possible in the healing process. Additionally, the participant must have been available for at least two further data collection appointments, which were scheduled in two or three week intervals. These multiple, longitudinal measurements were planned to start early in the healing process in order to capture the biomechanical information from acutely injured muscles and discover the natural biomechanical adaptations of the muscle tissue during the healing process.

Further inclusion criteria defined the clinical diagnosis process in order to assess potential participants' eligibility for *observational* and *longitudinal* studies.

For *observational* study, groin pain experienced by the potential participant must have decreased or prevented him from taking part in a game and/or training; for *longitudinal* study,

groin injury was defined as an acute incident, which resulted in groin symptoms and occurred in sports situations.

In *observational* and *longitudinal* studies, tenderness and/or pain as a result of palpating the adductor musculature, adductor tendons, or their insertion to the pubic rami, were the next inclusion criteria. Reproduction of this pain while palpating these areas is one of the most common diagnostic criteria for SRGP, widely used by a number of researchers (Holmich, 2007). Palpation may additionally be useful in excluding hip joint related pathologies, as palpating groin musculature is not likely to reproduce hip-related groin pain.

The reproduction of symptoms during a number of tests were the further inclusion criteria in all performed studies; specifically, the unilateral adductor muscle static resisted adduction test (lying supine); passive flexibility testing in the same position; unilateral iliopsoas muscle strength and flexibility testing; as well as squeeze tests in 0°, 45°, 90° of hip flexion. All of these tests were previously mentioned as reliable and valid (Malliaras et al., 2009, Thorborg et al., 2011); the squeeze test in particular is most commonly mentioned in association with diagnostics of SRGP (Delahunt et al., 2011a, Delahunt et al., 2011b).

The exclusion criteria in both *observational* and *longitudinal* studies were based mainly on the participant's positive response to hip joint specific clinical tests. Hip joint pathologies commonly manifest as pain in the groin area (Anderson et al., 2012, Banerjee and McLean, 2011), therefore carefully chosen clinical tests (flexion-adduction-internal rotation test (FADIR); flexion-abduction-external rotation test (FABER); and the grind test) were used to ensure that groin symptoms have muscular or biomechanical rather than hip joint origin. These tests are hip joint specific, sensitive, reliable and valid (Martin and Sekiya, 2008, Groh and Herrera, 2009). Additionally, significant lower back or posterior pelvic pain during the

physical examination excluded the potential participant from the study as this might indicate groin symptoms originating from the lumbar spine or sacro-iliac joints.

Potential participant in both *observational* and *longitudinal* studies was also excluded if he/she had any previous groin or abdominal symptoms, injury or surgery. Exclusion of those participants aimed in ensuring that there are no other but biomechanical and functional factors from most recent injury affecting the measurements. All inclusion and exclusion criteria are synthesised in Table 6 the inclusion and exclusion criteria for the injured participants in the observational study are enclosed in Appendix 8 (p. 329), for the longitudinal study in Appendices 9 (first testing occasion, p. 333) and 10 (subsequent testing occasions, p. 337). The inclusion and exclusion forms for the healthy control participants in both studie s are enclosed in Appendix 11 (p. 339).

<table>
<tr><th colspan="2">Inclusion criteria</th><th colspan="2">Exclusion criteria</th></tr>
<tr><td>Observational</td><td>Longitudinal</td><td>Observational</td><td>Longitudinal</td></tr>
<tr><td colspan="2"></td><td colspan="2">Positive response to any of the hip joint and SIJ clinical tests: quadrant test, grind test FADIR, FABER</td></tr>
<tr><td>>4 weeks of SRGP symptoms</td><td><5 days after the acute injury</td><td colspan="2">Other previous hip/groin/abdominal injury or procedure</td></tr>
<tr><td>Symptoms prevent from sporting activities</td><td>Acute injury occurring in the sports situation</td><td colspan="2"></td></tr>
<tr><td colspan="2">>18 years old</td><td colspan="2"></td></tr>
<tr><td colspan="2">Tenderness and/or pain when palpating the adductor muscles, tendons or insertion to pubic rami</td><td colspan="2"></td></tr>
<tr><td colspan="2">Pain reproduction during at least two of clinical tests: unilateral hip adduction, unilateral passive hip flexibility test, squeeze test (0°, 45°, 90° of hip flexion)
Additionally (but not necessarily), pain reproduction during other clinical tests: modified Thomas' test, active hip flexion against resistance</td><td colspan="2"></td></tr>
</table>

Table 6: Inclusion and exclusion criteria for the observational, and longitudinal studies.

Dominance

Athletes often perform voluntary movements in a certain way, involving the preferred side of the body. This is particularly relevant in repetitive, sports-specific tasks, such as kicking the ball. In consequence, the biomechanical patterns of muscle activation and movement may differ between dominant and non-dominant limb. Defining the leg dominance is therefore an important stage in data analysis when investigating the association between unilateral symptoms (such as SRGP) and biomechanical measurements (such as kinematics, kinetics and sEMG magnitude). Despite a common agreement regarding the importance of the effect of leg dominance on biomechanical measurements, this feature is often overlooked in sports medicine research and in clinical settings (Jessica Velotta, 2011).

All athletes included in presented studies were participating in sports disciplines requiring repetitive, sports-specific movements in training and game. A large number of those movements are one sided and asymmetrical, such as pushing off while starting to sprint, pushing off another player (rugby), kicking (football and rugby), twisting the whole body in particular direction and swinging the stick (field hockey) or throwing the Frisbee. It was likely that included participants performed those movements in a specific way, choosing the preferred side of the body more frequently than the other one. Therefore it was particularly important to determine the leg dominance of all the athletes included in the studies, and to include those data in further analysis.

There is a number of ways to define the leg dominance: by the hand preference (BARBER et al., 1990), preference to kick the ball (HB. Greenberger, 1995, Morrissey et al., 2012a, Malliaras et al., 2009, Petschnig et al., 1998, Brophy et al., 2010), preference to jump (John A. Nyland, 1994), or preference of a weight-bearing leg when kicking a ball (John A. Nyland, 1997). Dominance and preference of right or left leg may also depend on a task. The right leg was hypothesised to be more commonly chosen in movement (mobility) task, whereas left tends to be chosen during stability tasks (Gentry and Gabbard, 1995, Spry S, 1993).

In this thesis, the leg dominance of all participating athletes was defined as preferred kicking leg. The pitfall of this approach is that not all of the included athletes perform repetitive kicking movement in their primary sports disciplines (hockey and Frisbee). Therefore the movement of kicking may not be as intuitive in those sports, and may therefore have restricted application in defining the leg dominance.

However, studies investigating preference to kick a ball as a definition of leg dominance, included healthy participant with no specification of preferable sports discipline, and still obtained valid results (Jessica Velotta, 2011). Therefore, in order to keep a maximum

consistency in methodology, I decided to use this method to determine the leg dominance in all study participants.

Measurement methods

The primary aim of the thesis was to investigate the muscle activation and movement patterns associated with SRGP. In order to meet this aim, I chose surface electromyography (sEMG) to collect the muscle activation data, and the optical motion capture system to collect kinematic data from the study participant. Additionally, kinetic data from the force plates were also used to define the movement phases in the data processing and analysis. The rationale of the choice of these measurement methods are presented below, alongside the pros and cons of the alternative methods and reasoning behind the chosen data processing and analysis process.

EMG measurements

Surface electromyography measurements (sEMG) have been widely used in clinical and research settings to measure muscle activity and function (Luca, 1997). Classically, biomechanical researchers has recorded and analysed muscle activation during cyclic movements such as gait in order to establish normative muscle activity values and help to facilitate ambulation in patients with walking difficulties (Frigo and Crenna, 2009). The EMG has also been increasingly used to measure the pathological mechanisms in order to establish the movement patterns in chronic pain presentations (Szpala et al., 2014, Van Damme et al., 2014) or to guide rehabilitation after injury (Morrissey et al., 2012a). There are clear clinical implications emerging from the results of using the EMG in developing the treatment strategies for diverse groups of participants. Thus, using the EMG in clinical research enables us to better understand muscle activation patterns, altered recruitment strategies and their

association with pain, or risk factors for injury (Sole et al., 2012, Amiri-Khorasani and Kellis, 2013, Brophy et al., 2007).

There are three main electromyography methods, which are used to record muscle activity: surface, fine wire electromyography and – more recently – high density surface arrays. Those methods have various applications (Figure 8) as well as different advantages and disadvantages.

Surface electromyography (sEMG) is suitable for obtaining information about large areas of superficial muscle. It is widely used in biofeedback studies (Lyons et al., 2003, Yoo et al., 2014) and in a very broad field of biomechanics, rehabilitation and sports. The sEMG is a relatively easy to use, non-invasive technique, with a large variety of applications. It is used to study normative muscle activation values in athletic and non-athletic population in gait or sports-specific tasks; it helps to establish the movement efficiency (mainly in athletes); it guides the rehabilitation strategy by providing information of muscle activation in various exercises and movement tasks (Delmore et al., 2014, Boudreau et al., 2009); facilitates the diagnosis of muscle dysfunction (Disselhorst-Klug et al., 2009, Chendeb et al., 2004) as well as muscle damage (Felici et al., 1997, Merletti and Parker, 2004). The summary of the advantages and disadvantages of this method is presented in Table 7.

High density arrays (HD-EMG) can be used to collect detailed samples of muscle activity from the surface, thus yielding information about, for example, individual action potentials. HD-EMG is mainly used to investigate the details of the muscle activation strategies by collecting information about motor unit action potentials (MUAPs).

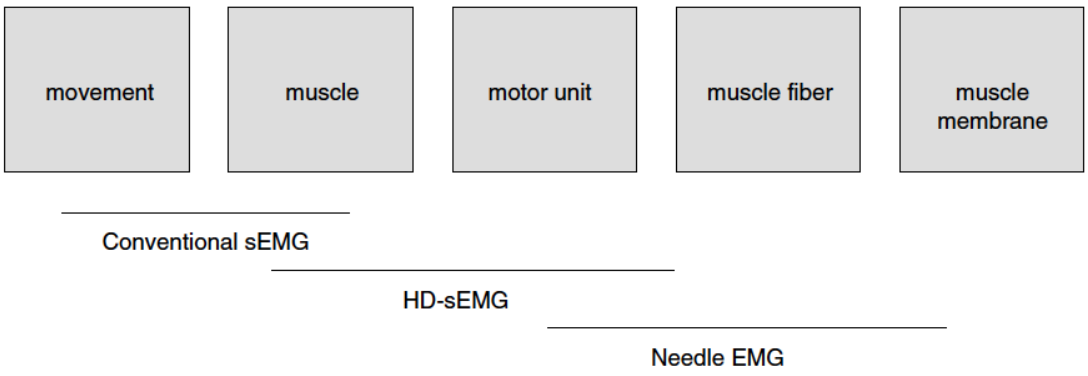


Figure 8: The overview of different EMG techniques. For movement studies, conventional sEMG is usually used; recently developed HD-sEMG with multiple electrodes allows to measure the muscular activity down to the level of the muscle unit; the needle EMG is used as a tool to obtain information from the muscle unit (Drost et al., 2006).

Indwelling electromyography (fine wire or needle) is the most invasive technique, but suitable to record the neuro-activity from smaller areas, down to a muscle fibre level. It is therefore extensively used in investigating the firing characteristics of motor units (MUs) (Hermens et al., 1992, De Luca et al., 2014), motor neuron excitability as well as clinically in establishing the neurological and neuromuscular diagnoses. Despite the invasiveness, indwelling (usually fine wire) EMG also finds its use in biomechanical research, as it gives the opportunity to investigate deeper muscles, not accessible with the sEMG (Jansen et al., 2010).

For the purpose of this study, sEMG was chosen to record muscle activation during movement because the muscles to be investigated were large and located superficially; it is non-invasive; has got an easy set up procedure and is relatively quick to learn (Hermens et al., 2000). Standard rounded Ag-AgCl passive disposable bipolar electrodes were used with an electrolyte gel built in.

The choice of using the sEMG as a main measurement tool in this study was associated with a few limitations (Kamen and Gabriel, 2010a).

The sEMG is an optimal device to measure the motor output from large and superficial muscles. Although such muscles were mainly the focus of my interest, few smaller and deeper muscles (such as internal oblique and ilio-psoas) were also relevant to my studies and collecting data from those was impossible with sEMG. I considered using additional types of EMG (such as indwelling fine wire EMG) before the data collection process, but adding another measurement tool would make the protocol too long and impractical. High invasiveness of fine wire EMG might additionally discourage some of the potential participants to take part in the studies.

The sEMG measurements have some standard limitations, which might have affected our measurements. Those limiting factors can not be fully controlled but can be minimised, albeit always being present in sEMG measurements.

One of the biggest potential sources of bias is misplacement of the surface electrodes on the body of the participant. In order to minimise this bias, I underwent an extensive training before the data collection and followed all available guidelines. My anatomical and physiological knowledge associated with my occupation facilitated the identification of desired muscles and the optimal electrodes location on the muscle. However, the individual's anatomical and physiological differences such as the location of innervation zones might have compromised the reliability and validity of the measurements.

Another potential limitation of the sEMG measurement is cross-talk. Cross-talk can be defined as any electrical activity recorded by the electrode that is not representing the activation of the desired muscle. It may originate from other muscles, when the surface electrodes are placed too close to them; it may also appear as an electric signal from the muscle of interest, which is not representing the magnitude of muscle activation. This can occur when the muscle electrical tripole (depolarized current sink and two current sources), which is a target signal for

sEMG, is covered by stronger dipole originating near tendon areas (where there is no ions to be delivered to depolarized zone) with significantly different properties. This dipole, also called a terminal phase of MUAP, is a high-frequency signal, which is reduced by distance to a less, extends than tripole. Subcutaneous fat, by increasing the distance between the signal source and receiver is therefore a natural source of bias.

There are limited ways to minimize the risk of cross-talk. Bipolar electrodes need to be placed close to each other to improve selectivity (10-20mm). However, placing them too close to each other may lead to salt bridge formation between electrolyte gel areas between two electrodes. This would reduce the difference between tw electrodes and significantly decrease recorded signal. In order to minimise the cross-talk risk during the data collection process, I followed closely all of the guidelines keeping the recommended distance between the electrodes.

Pros	Cons	How mitigated
Easy to use with a lot of guidelines of good practice available	No possibility to collect data from muscles located deeper	Such muscles were excluded from data collection process
Non-invasive	Potential bias due to the electrode misplacement	Extensive training completed prior to data collection
Relatively short time of participant set-up	Potential presence of cross-talk	Closely following available guidelines

Table 7: Summary of pros and cons of surface electromyography

Muscle neurophysiology is complex and there is a number of intrinsic and extrinsic factors potentially affecting the EMG signal (Luca, 1997). Therefore strict guidelines exist regarding

the sEMG data acquisition, handling and analysis. Extensive studies, which investigate the neurophysiology of muscle fibers and types of EMG instrumentation, serve as a base for those guidelines (Hermens et al., 1992, Hermens et al., 2000). The main educational role, although limited to sEMG, has been mostly held by SENIAM (Surface EMG for Non-Invasive Assessment of Muscles), which is an initiative in the Biomedical Health and Research Program (BIOMED II) of the European Union. SENIAM's two key objectives are to (i) provide a space and opportunity for researchers and clinicians working with sEMG to share experience on various aspects of using the sEMG; and (ii) to develop recommendations according to existing evidence regarding the use of sEMG. Another source of up-to-date knowledge about EMG is ISEK (The International Society of Electromyography and Kinesiology) together with its conference proceedings; and the Journal of Electromyography and Kinesiology.

In all studies included in this thesis I closely followed SENIAM guidelines in the sEMG data collection, acquisition and analysis. Following those guidelines, the electrodes used in these studies were placed on the surface of specially prepared skin. The skin area chosen for electrode placement was firstly shaved (if necessary) to ensure the optimal skin-electrode contact, then cleaned with alcohol wipes to discard any electric charges from the surface of the skin. Owing to a number of other skin preparation techniques reported in literature, I decided to additionally abrade the skin with a gentle sand paper. 'Sensor (electrode) location' refers to the centre of two bipolar electrodes located on a muscle. SENIAM provides the recommendations for electrode location for 30 muscles, defined as a point on the line between two anatomical landmarks. In individual muscles the sensors are recommended to be placed between the most distal motor endplate zone and the distal tendon (longitudinally) and within a maximal distance from the muscle edge or subdivisions (transversely); the bipolar electrodes need to be placed with the respect of the direction of muscle fibres (SENIAM.ORG, 2015).

Where SENIAM guidelines were not available (external oblique, adductor longus and adductor magnus muscles) other guidelines were followed (Lyons et al., 1983, Cram, 2011). It is worth noting, that although the data was collected from 12 muscles in all participants, this thesis focuses on the activation of 4 muscles only: gluteus medius and adductor longus muscle, bilaterally. Surface electromyography Ag-AgCl electrodes were placed bilaterally on external oblique (Figure 9a), gluteus medius (Figure 9b), adductor longus (Figure 9c), gluteus maximus (Figure 9d), rectus femoris (Figure 9e) and biceps femoris (Figure 9f) muscles.

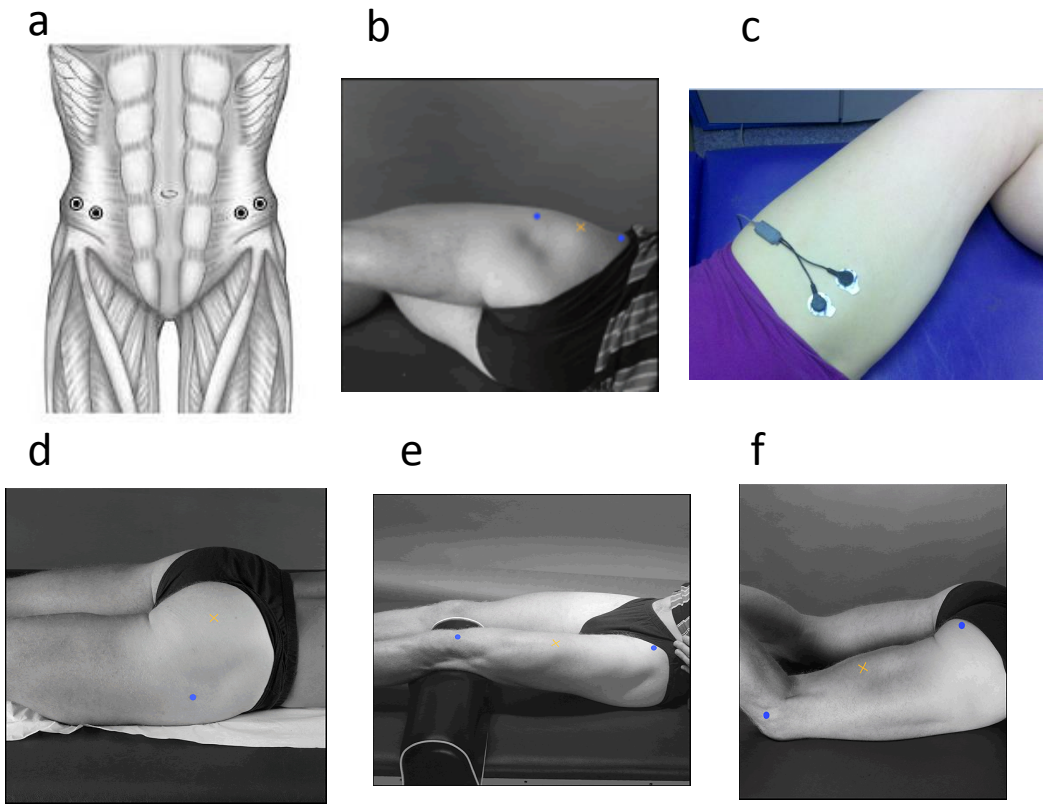


Figure 9: Surface electrodes placement location; a – external oblique muscle; b – gluteus medius muscle; c – adductor longus muscle; d – gluteus maximus muscle; e – rectus femoris muscle; f – biceps femoris muscle.

Reliability and validity of the sEMG measurements are reported to be high (Kollmitzer et al., 1999b). They may be, however, affected by the misplacement of the electrodes on the participant's body, which makes them user-dependent (Mathur et al., 2005). Thus the reliability and validity of the sEMG measurements may be compromised by a lack of anatomical or physiological knowledge, limited training time or no theoretical skills of the sEMG user (Kamen and Gabriel, 2010a).

Before collecting any data I completed extensive training in using the sEMG and gained essential knowledge to limit the risk of compormised sEMG measurement reliability or validity. A large number of practical sessions using sEMG device under close supervision improved my skills and provided confidence and independence in data collection process. Additionally, I have completed a five-year full time education, which led to obtaining BSc and MSc degrees in Physiotherapy, and have been working clinically since then. My education, qualification and clinical experience enabled me to gain and establish the anatomical and physiological knowledge, which likely minimised potential human error in my sEMG measurements.

Before collecting data from injured participants I had performed a reliability study, which reports a high to excellent reliability of performed research protocol. The details of those studies are described in Chapter 2: Reliability study (p. 140).

There are a number of limitations of sEMG measurements and there are limited methods to control them. However, sEMG measurements are generally reported to be reliable in biomechanical research (Kollmitzer et al., 1999a).

There are a number of studies reporting high sEMG reliability in the dynamic movements of upper (Reinold et al., 2004) and lower limb (Ng et al., 2008); in cyclic movements such as gait (Bogey et al., 2003) and more one-off sports specific movements (Ortiz et al., Amiri-Khorasani and Kellis, 2013). However, the reliability for the sEMG it is highly dependent on the specific

user and situation, for the studies included in the thesis reported in Chapter 5: Reliability study (p. 140).

The sEMG is commonly used to detect the motor output from muscles during intentional movement tasks of varied complexity. The movement of the participant, however, creates a number of limitations, which might potentially affect the reliability and validity of the measurements.

The most general issue is related to participants' comfort while performing movement tasks with a large number of wires around their body. In present studies, additionally to sEMG electrodes and their wires, CodaMotion markers were attached to participants' lower limbs and pelvis areas. The participants were given adequate time to habituate to moving with all the wires on; however, the quantity of markers and electrodes might have affected their movement.

The electrodes placed on the surface of the skin are supposed to detect and record the motor output of the muscle lying much deeper (Kamen and Gabriel, 2010a). The process of palpating the muscle, finding the recommended location for the electrode and attaching it there is performed with the participant lying down and relaxed (Hermens et al., 2000). If he or she performs an investigated movement during data collection process, associated contraction of the muscle will cause its displacement in relation to the skin and the initial proper electrode placement may lose its reliability.

Additionally, potential perspiration or electrodes rubbing against each other or against the skin may cause their displacement on the skin surface. This may further limit the reliability of measurements (Kamen and Gabriel, 2010a). This issue, however, was addressed by putting a large amount of tape over the electrodes in order to secure their position.

Other limitations of sEMG motor output, related to the movement, are associated with the physiological determinants of muscle electrical signal. The surface electrodes detect the motor unit action potentials (MUAPs), which is the sum of individual muscle fibres action potentials (MFAPs). MFAPs, which then determine the final recorded sEMG signal, might be affected by several factors. One of the MFAPs' characteristics, potentially affecting the sEMG recording, is the muscle fibre conduction velocity (MFCV). MFCV depends on factors such as muscle fibre diameter, temperature or intracellular pH. Muscle fibre's diameter, and consequently also MFCV, decreases with muscle stretch and increases with muscle contraction (Kamen and Gabriel, 2010a). MFCV might further increase with the increase of the temperature when performing the movement. But when the task is challenging, the MFCV might decrease due to decreased intracellular pH when muscle fatigues. All of those factors might potentially affect the MFCV and, consequently, the motor output detected by electrodes.

However, the movement tasks used in studies included in this thesis were rather static, not allowing the temperature to rise too much and not enabling the muscles to fatigue. Moreover, the temperature in the laboratory was kept similar during the data collection, which might have further minimised its potential effect on sEMG recordings. Nevertheless, those limitations exist and should be acknowledged while analysing collected sEMG data.

sEMG data normalisation

The procedure of normalisation of the sEMG signal generally means presentation of the raw sEMG data as a relative value by dividing it by another sEMG value. The aim of this is to decrease the between-subject variability and make the sEMG signal comparable between participants. Normalisation is proposed by a number of authors as a standard procedure before further analysis of the sEMG signal (Luca, 1997, Cram, 2011, Burden, 2010). Non-normalised sEMG should not be analysed between subjects or between different

measurement occasions in one subject due to the specificity of muscle physiology and different methods of generating force.

The motor output is a measure of the electrical activity of all individual muscle fibres located close enough to the surface electrodes to be recorded; this generates force. Different muscles and different muscle fibre types have different strategies to increase force. Moreover, in order to increase the force output, at low force levels the quantity of active motor units increases, while at higher force levels it is rather the frequency of motor units that is responsible for increased force.

The choice of normalisation method is critical in further interpretation of obtained data (Burden, 2010) and should be strongly dependent on the aim of the individual study. Therefore, the decision whether to normalise the sEMG data at all should also be consciously made. Considering the aims of individual studies and the desired outcome measures, standard normalization procedures may decrease the reliability of the sEMG data or simply not add any value, thus making the procedure useless.

There are a few common ways to normalize the EMG data and there is no agreement among researchers, which is the best normalizing procedure (Lai et al., 2009, Kamen and Gabriel, 2010a). Most frequently, the sEMG signal collected during a task is divided by other sEMG values from the same muscle. The most common procedure is to obtain the sEMG value from a maximal voluntary contraction (with isometric contraction being the most common and most reliable type) (MVC) and then dividing the obtained sEMG value from the investigated task by isometric MVC. A very similar procedure is also commonly used with various percentages of MVC (50%, 60% or 80%), which is then called sub-MVC. This method seems to be a reliable way of potentially obtaining a very clean sEMG signal, as all of the artifacts present in the desired motor output also appear in the value to which it is normalized. In consequence,

dividing two sEMG signals with exactly the same noise will naturally filter out this noise. However, MVC and sub-MVC are strongly dependent on the effort that the individual puts into the forceful muscle contraction. It was discovered that the term 'maximal voluntary contraction' is in fact misleading as much higher activation outputs are achieved when performing high-velocity muscle actions (Ball and Scurr, 2013). Moreover, there is no consensus regarding the type of manoeuvre used in order to achieve the maximal neural activation of the muscle.

Other values, that obtained sEMG signal might be normalized to, are peak or mean activation value when performing investigated movement task (Lai et al., 2009). This method was previously used in investigating the sEMG values in cyclic movements such as gait (Allison et al., 1993). It was reported to reduce the intra-subject and inter-subject variability, thus completing its normalization role. However, it might reduce the meaning of some real biological inter-subject differences, such as strength. Also, same movement task might be of graded difficulty among individuals. In consequence, different people with various strength levels would use different level of muscle activation to perform the same task. Normalising the sEMG signal obtained during this task to the mean of muscle activation, while performing it, might therefore disregard the strength differences.

Moreover, the movement strategies and their reproducibility in movement tasks are not well known. Therefore, muscle activation patterns used differently in individuals, or in different repetitions might alter the relation to the reference sEMG value.

None of normalization procedures described above were considered relevant to our study. Normalisation to 100% or 50% of MVC would be both non-ethical and non-reliable, as participants would have been asked to maximally contract potentially injured muscle. Not only would they not be able to perform a maximal contraction, but also such effort might

additionally increase their symptoms and deteriorate their condition, which would be highly unethical.

Normalising the sEMG to a peak or mean level of muscle activation during analysed movement task seems to be a convenient and relevant way in our study. However, because one of the aims of the study is to discover the muscle activation patterns, it would be wrong to assume that the patterns are not affected by the injury by including them as one of the inputs (or independent variables) in analysis. Moreover, peak or mean sEMG value used for normalisation would be obtained from healthy muscle in healthy participant and injured muscle in injured participant. Using the same value in healthy and injured participants would not allow obtaining a comparable sEMG value, as it would include pathology in injured participants.

Therefore, owing to the specific aim of this study, I chose another way to process sEMG data in our study. It is important to note that it was not the main aim of this study to discover the muscle activation magnitude in various groups of participants. Instead, I aimed to explore the movement patterns and relationship between the activation (and its consequences on the biomechanical balance) of two muscles (adductor longus and gluteus medius), and potential differences between groups. Firstly, the sEMG data was time-normalised by dividing analysed movement task into phases and averaging sEMG data for each recorded muscle within those phases. Time averaging is a common technique for analysing sEMG data by providing a standard and reliable value against which the data are measured (Burden et al., 2003a, Mathiassen et al., 1995). It thus allows reliable quantification of muscular motor output in chosen time phase (van der Hulst et al., 2010b, van der Hulst et al., 2010a, van der Hulst et al., 2010c).

Secondly, I used intra-subject muscle ratios as a primary outcome measure within participant, which was then averaged within group (such as symptomatic or asymptomatic group). Because the subcutaneous fat layer has a comparable depth within a given participant, using intra-subject ratios would minimise fat's potentially large effect on the sEMG signal. Moreover, by using muscle ratios I was able to explore the muscle activation patterns by analysing the relative relationship between two muscles rather than two separate values, which was our aim. This approach was previously used and published in several papers (Morrissey et al., 2012a, van der Hulst et al., 2010a, van der Hulst et al., 2010c, Ferguson et al., 2004, Reeves et al., 2006, Daly et al., 2015). Due to the aim of exploring movement patterns in specific athletic sub-groups in my study, it was even more important to ensure that the measurement values were comparable between groups such as amateurs and professionals, therefore analysing muscle ratios within defined movement phases was, in our opinion, the best, most relevant and accurate way of normalizing our sEMG data.

However, if the primary measure of two muscle sEMG activation ratios was showing significant differences between groups, I used the secondary measure of individual muscle activation magnitude within a given time phase in order to indicate the reason for an observed ratio difference. I did not analyse the exact quantity of the sEMG activation magnitude in separate muscles, but rather interpreted the trend (increase or decrease of the activation magnitude), which affected the ratio measures.

Normalisation of the sEMG is a process that aims in reducing the inter-subject variability in order to make various groups of participants, different muscles and different measurements occasions comparable (Luca, 1997, Cram, 2011). Although I made a potentially controversial decision not to normalize our sEMG data in the most common way, significant between-groups differences, with very small P values, found in our study make a strong argument in favour of our method of proceeding.

Muscle activation measured by sEMG is a useful representation of muscle function, but can't be proportionately related to muscle force (Nishihara and Isho, 2012). Although some research reported the linear relationship between sEMG signal and muscle force output, it was only found in specific conditions, specific muscles and during isometric contractions (Lippold, 1952). In a majority of studies, the muscle activation was found to increase with the increase of the force (Madeleine et al., 2001, Solomonow et al., 1990), but this relationship was not linear (Bilodeau et al., 2003, Gregor et al., 2002, Karlsson and Gerdle, 2001, Onishi et al., 2000).

In majority of biomechanical and kinesiological research, where the aim of study is to measure a real movement, treating the sEMG signal as a representation of force is incorrect for a number of reasons.

Firstly, all of the factors mentioned previously, that may affect the sEMG signal, make it impossible to directly relate it to force; specifically the surface electrode location, change of the muscle and muscle fibres length or dislocation of the surface electrode in relation to the muscle during movement (Gerdle et al., 2000, Gerdle et al., 1997, Wretling et al., 1987). Secondly, the size and shape of the surface electrodes determine its data collection from particular muscle areas only (Nishihara and Isho, 2012). Therefore only a certain number of muscle fibres and motor units are placed under and its activity recorded by the electrodes, which prevents from recording the whole muscle activity and, consequently, the number of motor units. If all of the motor units covered by the electrodes are active and muscle force is still increasing it might mean other areas of muscle are being activated, which is impossible to measure (Nishihara and Isho, 2012).

It might also mean that in order to increase force output, the frequency of already active motor units rather than activating new motor units occurs. This is also impossible to detect by

sEMG, which can only record the quantity rather than the quality of muscle fibres depolarization.

Additionally, the force directed in certain direction is frequently produced by more than one muscle (Nishihara and Isho, 2012). The presence and level of this synergy is not possible to control, therefore activation measurements from the muscle of interest can't be expected to be responsible for the entire generated force (Nishihara and Isho, 2012).

Kinematic measurements

Despite a clear link between the movement patterns and other multi-structural pathologies (such as shoulder or lower back pain), and clear muscle strength, flexibility and range of movement imbalances reported in SRGP athletes (Chapter 2: Systematic review, p. 45), the association between movement and pathology has not been investigated in that group. Interestingly, the movement patterns in association with SRGP are highlighted as one of the areas with the need to research on by the Doha agreement. I decided to collect the kinematic along the electromyographic data in order to discover how the muscle activation imbalances affect the movement of the hip joint in the injured athletes. Given that the muscle activation patterns are not easily measurable in clinical environment, I aimed at exploring associations, which may be observable and quantifiable by a clinician. Additionally, because the link between muscle activation and force is not linear, measuring hip joint rotations gave me the opportunity to investigate the link between the muscle electric output and hip joint movement.

3D kinematic and kinetic data present a good opportunity to quantify and analyse multi-segment movement. In my studies, I used the kinematic data in order to discover the signatures of SRGP in the movement patterns of injured participants.

Despite few concerns and limitations associated with the reliability of 3D kinematic measurements, one systematic review provided evidence for the reliability of the 3D kinematic measurements (McGinley et al., 2009).

'Motion capture' (mocap) refers to a method of measuring and quantifying human or animal movement so that it is presented in digital form that can be further analysed (Gabai and Primo, 2011, Scott Dyer, 1995). Mocap is very widely used in arts, performance, animation, in research in the areas of psychology, orthopaedics, neurological disorders, social relations and sports medicine as well as in clinical settings (McGinley et al., 2009).

Typical motion capture system includes a set of devices tracking the movement and software that determines the animation of the image based on calculations. The exact technical solutions to achieve the desired output vary between systems. Kinematic data can be collected by one of many available motion capture systems. Three main types are mechanical, magnetic and optical; each of these systems have certain advantages and disadvantages (Scott Dyer, 1995).

Mechanical mocaps are based on an exo-skeleton, which is worn by the object and is follows and tracks the movement of the object; the sensors in each joint recognise and track the rotations (Vlasic et al., 2007). This system is insensitive to any interference from light or magnetic field, which makes it seemingly straightforward to use. However, mechanical systems have got a number of limitations, which excludes them from biomechanical research with clinical implications. Firstly, they do not have any awareness of the ground level, which makes movements such as jumping impossible; secondly, the distal data of lower limbs tend to lose their accuracy; thirdly, in typical systems it is impossible to determine the object's orientation during data collection – the displacements is only calculated based on the amount of rotation that was detected by the mechanical frames (Vlasic et al., 2007).

Magnetic mocaps use wired sensors to measure the magnetic field created by the source (Scott Dyer, 1995). They typically include one or more control units, and all sensors and the source are wired to this unit. The sensors are attached to the tracked object, and the source (magnet) is usually placed centrally. These systems have high measurement accuracy and relatively low level of signal interruption. However, they are very sensitive to metal objects near the data collection area; the range of these devices is narrower than in optical systems and the sampling rate is too small for typical sports movements (Scott Dyer, 1995), which suggests their limited application in biomechanical measurements .

In optical mocaps the markers are placed on the object and then the optical signal of their location in transmitted to the receivers – cameras, which are then attached to the computer that manages the data collection process. The markers attached to the object may operate either in a passive or active way. In passive systems the markers are covered with a retro-reflective material, which allows them to reflect the LED light emitted from the cameras.

In active systems, the markers themselves are emitting light, which is then captured by the cameras. Regardless of the type of the markers, the optical signal is received by each camera, which then generated the 2D coordinates for each marker. This information is further transferred into the computer and the software calculates the 3D coordinates of each marker (Bodenheimer et al., 1997).

The optical mocaps are widely used in biomechanical research (Lebel et al., 2013, Laudner et al., 2014, Morrissey et al., 2012a) owing to: their high accuracy; high sampling rate which allows detecting a subtle movement or displacement; and typically wireless marker types which does not restrict the object's movement during data collection. Those systems, however, are usually more expensive, require a specially designated and prepared space in

order to operate and are sensitive to light and occlusion, which decreases the data quality and accuracy (Bodenheimer et al., 1997).

The quantification of observed movement in this study was enabled by using the Cartesian Optoelectronic Dynamic Anthropometer (CODA) motion system (Codamotion Cx1 sensor units, Charnwood Dynamics, Rothely, Leicestershire). To collect the kinematic data, active, infra-red markers were put on participant's body according to validated protocols (Monaghan et al., 2007). The markers attached to the surface of participant's skin on strictly specified anatomical landmarks served as a base to calculate joints centres: pelvis, hips, knees and ankles. Data for our study were collected only for pelvis and lower limbs, as only those body parts were relevant for the study aims.

The signal from the markers attached to participant's body is recorded by four cameras and then computed into 3D stick-figure displayed on the screen. During the data collection process the information from all of the markers was received in three planes and the displacement recorded in on all three axes. Further calculation according to standard CodaMotion protocols enabled to calculate the rotation of each joint bilaterally in all three planes.

The main source of bias in the kinematic measurements is misplacement of the infra-red markers on the participant's body. In order to accurately calculate the joint rotation centres, participant's anatomical landmarks must have been identified without error. As with the sEMG, extensive training period, a large amount of reliability data as well as my experience and occupation minimised the risk of the collected data being of poor quality.

In my studies, the pelvis and lower limbs kinematic data were obtained by the infra-red CodaMotion system markers sampling at 200Hz attached by the double sided tape to participant's bony anatomical landmarks according to modified Helen-Hayes protocol following standard protocol (Monaghan et al., 2007).

Kinematic data processing

The infra-red markers placed on each participant's anterior and posterior superior iliac spines, as well as the marker wands placed on the thighs allowed to calculate the internal hip joint centre of rotations in three planes, according to the local coordinate system. This process is automatic within the CodaMotion software, once the markers are appropriately named in the data collection set-up file. In order to calculate the joint rotations, the software must first determine the rigid segments, between which the rotations would further occur. In case of the hip joint, these segments are the pelvis and thigh.

Those segments and the points of segments definition (anterior and superior iliac spines, as well as femoral epicondyle) were defined following the International Society of Biomechanics (ISB) guidelines (Wu et al., 2002), which recommend using easily palpable anatomical landmarks as the frame for the definition of the hip joint centre. The rotations in the centre of the hip joint, as well as the rotations around the axes are also recommended, and presented on Figure 10, in this case – a right hip joint.

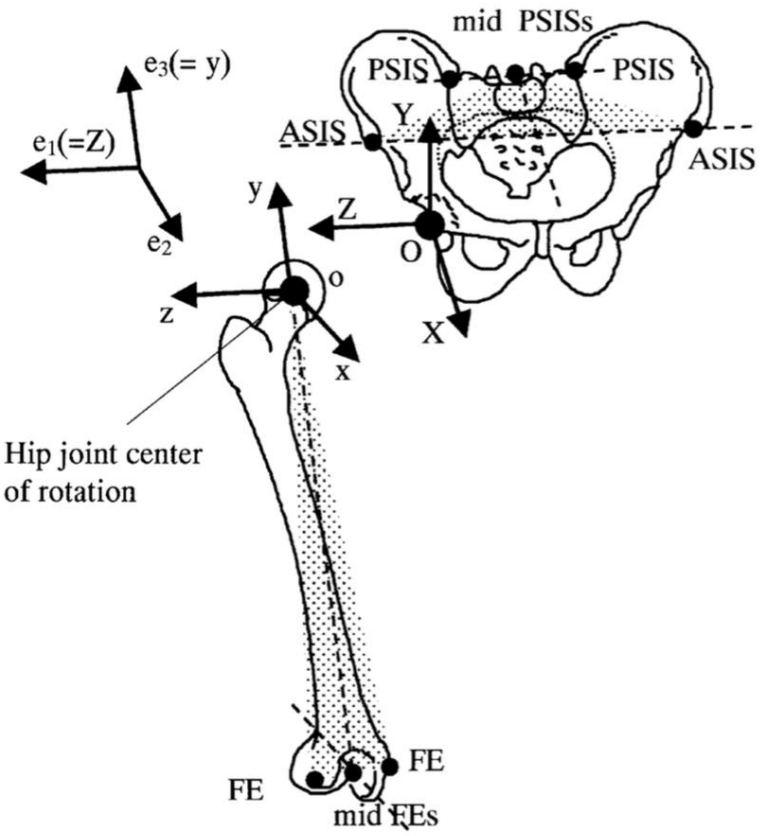


Figure 10: Graphical representation of the pelvis coordination system (XYZ), femur coordination system (xyz) and the right hip joint coordinate system (Wu et al., 2002).

Pelvis coordinate system (XYZ)

O: The origin coincident with the right hip centre of rotation

Z: This axis is made by the line, which is parallel to the line between both ASISs, and directed to the right

X: This axis is defined by the line lying parallel to the line lying in the plane defined by both ASISs and the midpoint between two PSISs, directed anteriorly

Y: This axis is defined by the line perpendicular to both Z and X axis, directed cranially.

Femoral coordinate system (xyz)

o: The origin coincident with the right hip joint centre of rotation, which is coincident with O in the neutral configuration

y: This axis is defined by the line originating int eh midpoint between the medial and lateral femoral epicondyle, and the origin, directed cranially.

z: This axis is defined by the line perpendicular to the y-axis, lying in the plane defined by the origin and two femoral epicondyles, and is directed to the right.

x: This axis is perpendicular to y- and z-axes, directed anteriorly (Cappozzo et al., 1995).

The hip joint rotation centres for each individual were based on adding the individually calculated offset to the line between the left and right ASIS reference point.

Kinetic measurements

Force platforms have been increasingly used in biomechanical research as a direct representation of vertical, latero-medial and fore-aft components of ground reaction forces during stance and movement (Cross, 1999, Bobbert and Schamhardt, 1990). Combining the kinetic output with additional kinematic data, using the link-segment models of human body, it is possible to calculate the joint reaction forces (Bobbert and Schamhardt, 1990).

Most generally, the force plate can be described as a metal platform including one or more sensors (strain-gauge transducer or piezoelectric transducer), which provides the electrical signal proportional to the force acting on a platform.

In my research, all of the participants performed all of the movements on the Kistler type 9281B force plates (Kistler Instruments Corporation, Winterthur, Switzerland). They are equipped in four built-in piezoelectric 3-component force sensors, and form a very rigid aluminium 'sandwich' constructions allowing for the measurements of a wide spectrum of movement frequency. I aimed in measuring the sEMG and kinematic data in various phases of selected movement tasks, some of them having very subtle force displacement signatures (for

example, change from bilateral to unilateral stance). Thus, high sensitivity of the force plates was necessary to detect and define the movement phases for further analysis.

Movement manoeuvres

I collected the electromyographic, kinematic and kinetic data from participants performing specific movement tasks. They were carefully chosen to be of a graded difficulty and provide a loading challenging for the pelvic and groin areas.

Prior to the data collection the participants were instructed how to perform each movement task, they were also given time to practice them; this procedure was performed in in order to allow the participants to familiarise themselves movement having CodaMotion markers and sEMG electrodes attached to their body. The collective flowchart of the data collection process is presented on Figure 11.

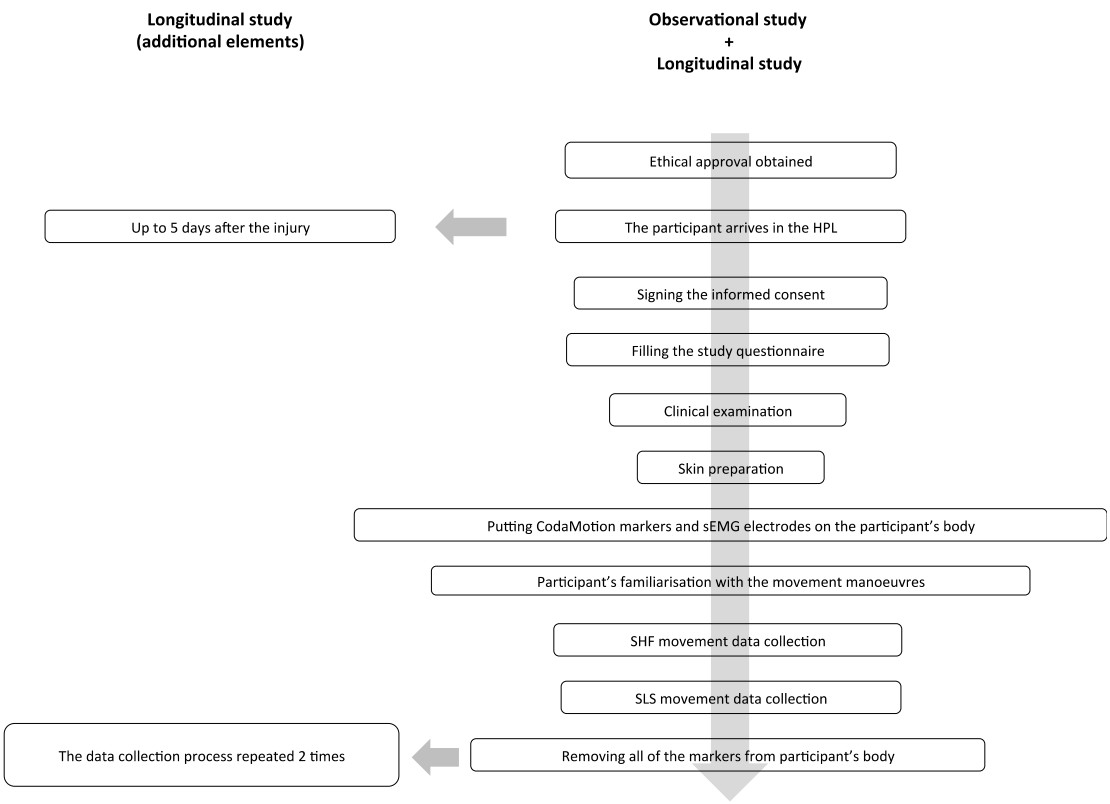


Figure 11: Flowchart representing the data collection process and the differences between the observational and longitudinal studies included in the thesis.

Standing hip flexion (SHF) was chosen as one of the test manoeuvres, with data for both moving and stance legs being collected and analysed. This task, analogous to kicking and locomotion, has been the subject of previous similar study (Hungerford et al., 2003). Participants were instructed to flex their hip to 90° within one second, then to maintain 90° of hip flexion for two seconds before returning to bilateral stance (Figure 12).

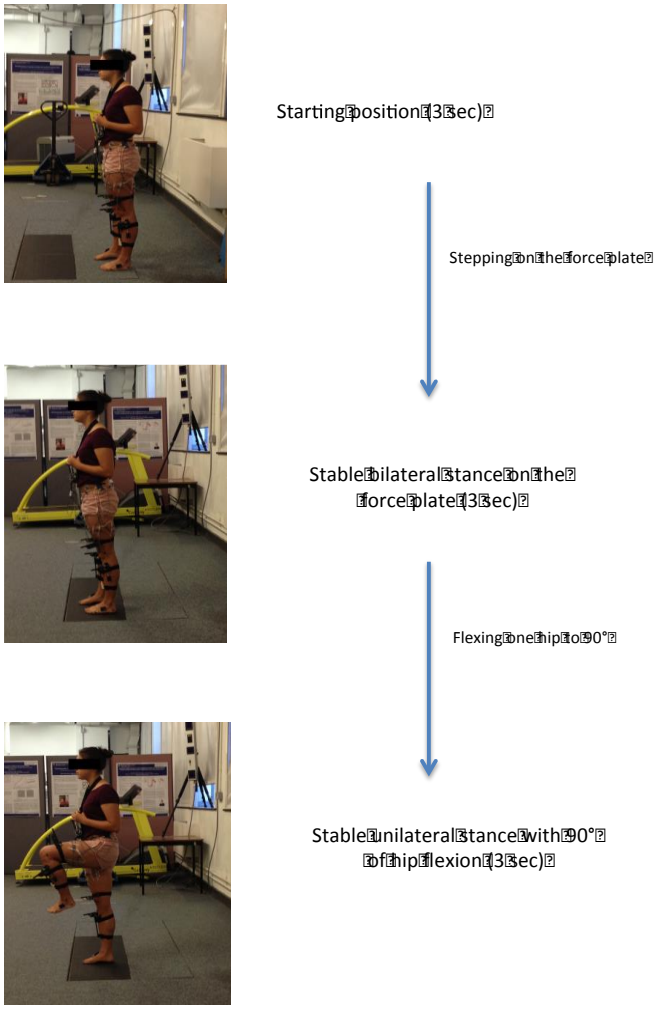


Figure 12: Stages of the standing hip flexion manoeuvre (SHF).

Single leg squat (SLS) was chosen as the second manoeuvre as being challenging for pelvic girdle stability and control (Hungerford et al., 2003, Hungerford et al., 2004), analogous to sports-specific movements common for investigated cohort and considered to be a progressive challenge comparing to SHF (Boudreau et al., 2009). Moreover, SLS is a widely used clinical test commonly used to assess the pelvic girdle and hip function (Boudreau et al., 2009, Crossley et al., 2011).

During this task, the participant was asked to flex the knee of the non weight-bearing limb to 45 degrees. They were permitted to position the non weight-bearing hip in whatever position they chose. They were then asked to squat on the weight-bearing/supporting limb as low as possible and then return to an upright single leg stance position (Figure 13).

Other manoeuvres, such as rapid direction change, cutting, pivoting, side-to-side steps or kicking were considered in order to add an element of sport specific tasks. They were, however, finally excluded from data collection procedure as were not validated and repetitive enough or were unable to be performed properly in laboratory environment. Moreover, majority of them also reproduced participants' symptoms. Therefore including them in data collection process was not only unethical, but might also considerably affect the measurements as presence of pain might change the way participants moved in order to avoid pain.

The order of testing the right and left legs in all movements was randomised by participant's preference. The process of dividing the movement manoeuvres into phases, as well as the detailed criteria of each movement phase, are presented in the next part of this chapter.

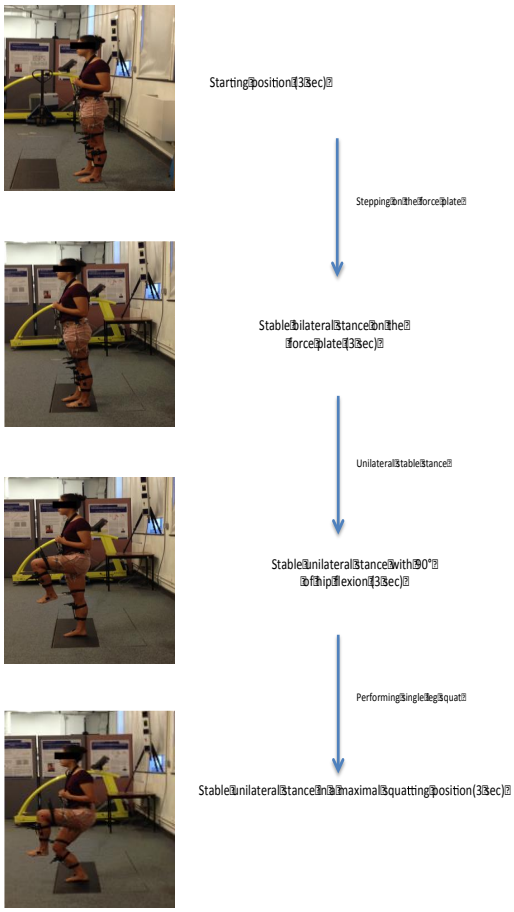


Figure 13: Stages of the single leg squat manoeuvre (SLS).

Dividing the movements into phases

Movement tasks were divided into phases using the kinetic and kinematic outputs by visual examination of the data using the custom made MatLab program (Figure 14).

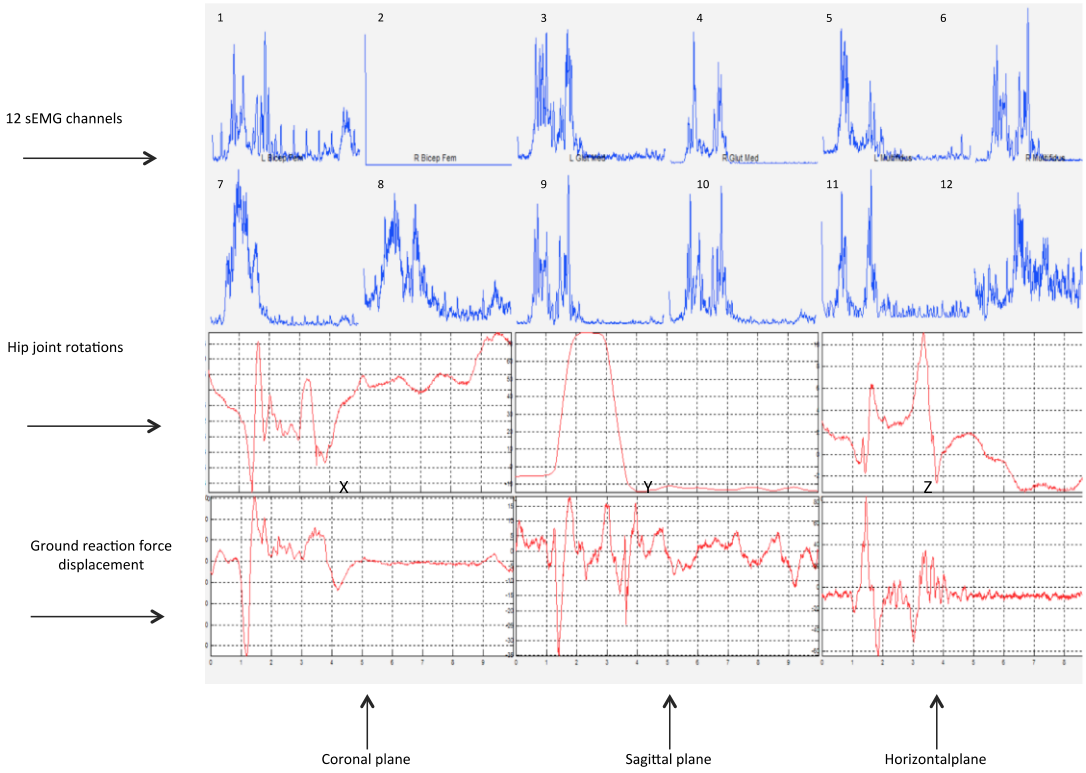


Figure 14: The interface of a custom-made MatLab program used to define the phases of movements. Top two rows (in blue) represent the rectified, filtered and smoothed sEMG signal obtained from all 12 channels; the top red row represents the hip joint rotation in x (coronal), y (sagittal) and x (horizontal) planes.

The kinetic data obtained from the force plates were necessary to identify the load displacement when performing the movement. In both movement tasks (SHF and SLS), the first defined phase included a change from bilateral to unilateral stance. This moment was only detectable from the kinetic data, as the movement itself was too subtle to be noticed using the kinematic output. The definition of subsequent SHF and SLS movement phases was completed by the visual examination of the kinematic and original CodaMotion data.

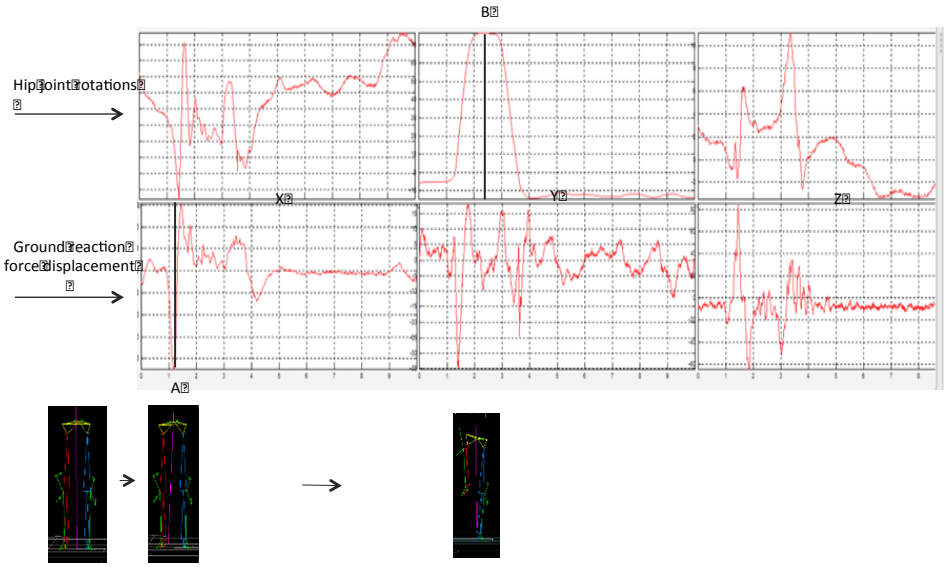


Figure 15: The process of defining the phases of SHF movement in the custom MatLab program. Top row represents the hip joint rotations; the bottom row represents the ground reaction force displacement in three planes: coronal (x), sagittal (y) and horizontal (z). A: the initial lateral force displacement when changing from bilateral to unilateral stance, B: maximal hip stable hip flexion.

The custom made MatLab program written to define the movement phases in investigated tasks allowed to display the graphs representing any lower limb joint rotation (pelvis, hip, knee and ankle) next to kinetic output from the force plate, plotted against time in all three planes (coronal, sagittal and horizontal) (Figure 14). The original, non-processed data from CodaMotion software in a form of stick figure were displayed simultaneously on another monitor in order to ensure that joint rotations displayed on graph represent the expected movement signatures in each participant (Figure 15).

This data processing and analysis approach requires a manual definition of each movement phase, for each participant in every data collection event. It is therefore very time consuming, and relatively sensitive to human error. I made some attempts to entirely automate the process of defining the phases of movements, but those solutions failed.

Depending on the movement task, the number and definition of the movement phases varied. SHF was divided into three phases: early, middle and late. The early phase was selected to represent the start of transition from bilateral to unilateral stance, which is a moment of increased demand on the pelvic girdle. It was defined as 50 ms before and 50 ms after initial lateral push on the force plate and initial abduction of the hip of the moving leg. The exact moment of this lateral force shift was defined using the kinetic output from the force plate as it was clearly represented by a positive or negative peak (depending whether the pushing leg was right or left, as the orientation of the laboratory co-ordinate system was constant) in the X-axis of the force plate output. This peak defined the exact moment of push-off, with addition of 50 ms before and 50 ms after used to define an early phase, being performed in the MatLab program. The period prior to activation was designed to partially account for electro-mechanical delay.

The end phase of SHF was selected to evaluate the biomechanical features of stable unilateral stance. It was defined as 50ms before and 50 ms after stable standing with 90° of hip flexion, defined mainly using the kinematic output of the moving hip in sagittal plane. The exact moment of stable stance was defined manually, when the participant achieved a stable standing hip flexion position. In order to measure a representatrvie period of the whole end phase of the movement, 50ms before and 50 ms were added after the defined stable stance moment.

The middle phase of SHF included the actual movement of flexing the hip joint, which represented the dynamic ability to adjust muscular output throughout the range of movement. It was defined as starting at the moment of the completion of the early phase of the movement (so 50 ms after defined initial lateral push represented by the lateral peak on the force plate output) and finishing at the commencement of the end phase, so 50 ms before the defined stable stance with one hip flexed.

In SLS seven phases were defined based on kinematic data - four movement phases and three stationary phases (Table 8). The choice of these phases enabled comparison of kinematic and muscle activation data at clinically relevant phases of the SLS task (e.g. deep knee flexion). These phases were divided into two categories: four movement (M) phases and three stable (S) phases (Table 8). The order of the phases was as follows: M1 -> M2 -> S1 -> M3 -> S2 -> M4 -> S3. The Movement I phase (M1) was defined in the same way as the early phase in SHF – firstly the initial lateral shift on the force plate was defined (lateral peak on the X-axis of the force plate output), then 50 ms were added before and after that lateral shift (Table 8). The Movement II phase (M2) occurs between the end of the M1 phase and beginning of S1 phase – so until the participants stand still on one leg (Table 8).

Stance I phase (S1) was defined as 50 ms before and 50 ms after the stable stance on one leg, with the other leg held in the front or at the back depending on individual preference. The exact moment of the stable stance was defined using the sagittal plane (rotations around the X axis) of the lifted hip – the moment of stability in sagittal plane was marked, then 50 ms were added both before and after the moment of stable stance but the MatLab program automatically (Table 8).

The Movement III phase (M3) was defined as the actual squatting down movement starting at the end of the Stance I phase and ending at the beginning of Stance II phase, including the movement of squatting down, which typically lasted 3 seconds (Table 8).

Stance II phase (S2) was defined as the moment of stable squatting position (Table 8). Firstly, the moment of stable squat was defined using three different out puts in order to determine the exact moment of a stable squat position: visual raw data from CodaMotion capture system, hip sagittal plane kinematic data (rotation around the X axis) and knee sagittal plane

kinematic data (rotation around the X axis). Then, 50 ms were added before and after the defined moment, constituting the S2 phase.

The Movement IV phase occurred when the participant was returning from the squatting position back to the stable one leg stance, between the end of the S2 phase and beginning of Stance III phase (S3) (Table 8).

Last phase of the SLS, the Stance III phase (S3), was defined similarly to S1 phase - the only difference was that S1 phase occurred before performing the squat, and S3 phase occurred after the squat.

Phase number	Phase characteristics	Phase code	Phase description
1	Movement phases	M1	Lateral shift of the load - initiation of change from bilateral to unilateral stance
2		M2	Change from bilateral to unilateral stance
3		M3	Squatting down
4		M4	Squatting up
5	Stable phases	S1	Stable unilateral stance prior to performing squat
6		S2	Stable stance in unilateral maximal squat position
7		S3	Stable unilateral stance after to performing squat

Table 8: Table showing the division of the SLS movement to seven phases

Data analysis process

There were four main outcome measures in the experimental studies of this thesis: the ratio of the magnitude of the gluteus medius to adductor longus muscle activation, and hip joint rotation values in coronal, sagittal and horizontal plane. In the longitudinal study, an additional outcome measure was the VAS scores for the clinical tests during each testing occasion.

sEMG	Kinematic
GM:AL activation ratio	Hip coronal Hip sagittal Hip horizontal

Table 9: Table summarising four main outcome measures in the experimental studies of the thesis; sEMG – surface electromyography; GM – gluteus medius muscle; AL – adductor longus muscle.

All four main outcome measures were firstly calculated for each individual participant within each defined movement phase in SHF and SLS as the mean of three repetitions, for each leg separately, for moving and weight-bearing leg in SHF and only the moving leg in SLS. Then, the group means were calculated in a similar fashion, each outcome measure being averaged for the relevant subgroups in each movement phase for each movement.

The collective flowchart of the data analysis process is presented on Figure 16.

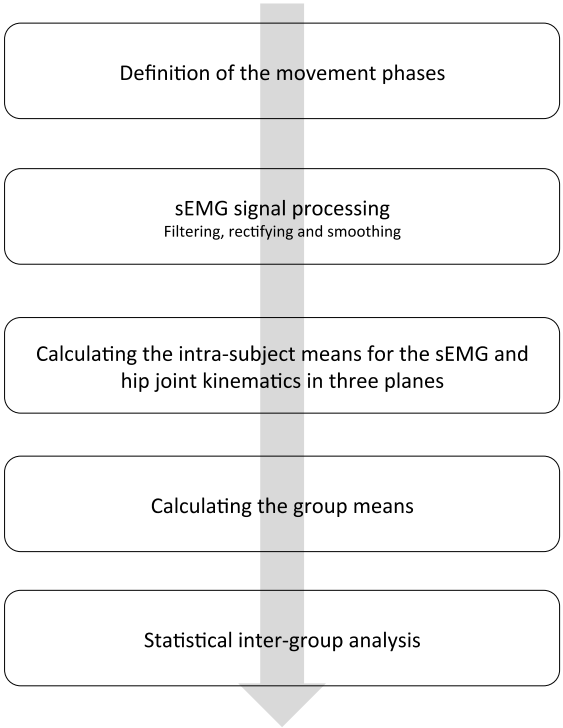


Figure 16: Flowchart presenting the stages of the data analysis process.

The details of the statistical analysis are presented in each chapter separately.

Step-by-step data processing

In this section we present the data processing and analysis to give a step by step representation of the process of getting from raw data to a format ready for statistical analysis.

The electromyographic, kinematic and kinetic data were collected with the CodaMotion software and all data were synchronised to give a time synchronised output, with the potential offset of any data source negated. The sEMG data presented here is in fact a short sample of 1.2 sec of the whole sEMG collected signal, which was cropped to better represent the effect of processing tools on the signal.

sEMG data processing

The sEMG provides a lot of valuable information about muscle function. However, a raw sEMG signal is a form of data associated with significant limitations, and can't be further compared between participants, or within one participant between different occasions. Moreover, the sensitivity of sEMG during data collection process enables a large amount of noise to be recorded along the desired, biological signal. Therefore, the sEMG signal needs to undergo certain procedures in order to be further analysed.

In order to present the effect of data processing techniques on the sEMG signal, the output was extracted for one participant, the example signal during one repetition of SHF from the adductor longus muscle being shown in Figure 17.

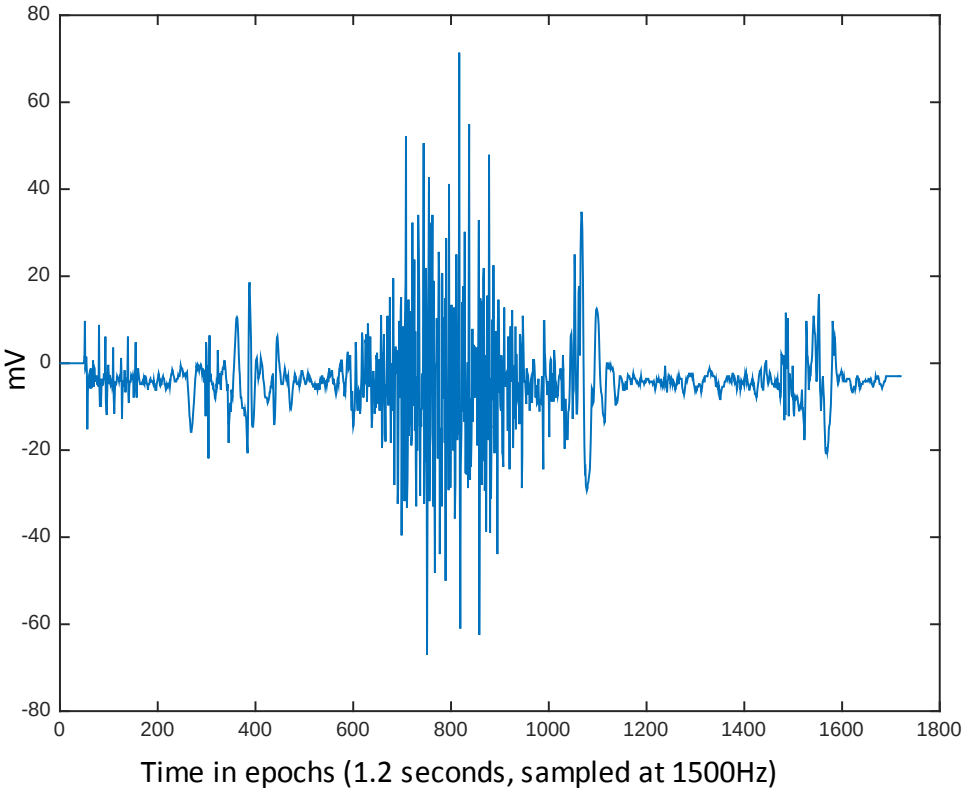


Figure 17: Graphical representation of a sample raw sEMG signal (please note that this signal has been analogue filtered in the amplifier attached to the sEMG electrode, but has not been digitally filtered), in this case of the left adductor longus muscle (AL) during the left standing hip flexion task (SHF). mV – millivolts.

Then, each individual raw sEMG signal output is further processed using filtering, rectifying and smoothing techniques applied by a custom made MatLab programme (Appendix 13, p. 358) –.

Filtering

The first level of the sEMG signal processing is data filtering: analogue and digital. Analogue filtering occurs during the data collection process in the amplifier attached to the electrode (Kamen and Gabriel, 2010a). The main function of the amplifier is to distinguish the desired muscular motor output from electrical noise, which is present in the environment and easily

transferred through the human body. Two bipolar electrodes placed close to each other are organized to record a potential difference between the two. They detect the entire electrical signal transferred through the area to which they are attached. This signal includes not only the biological, desired muscular motor output, but also any other biological or environmenatal signal. The detected sEMG is a summation of the noise, neural and muscle fibre membrane depolarisation. Therefore it is not detected at the same time by both electrodes. This phenomenon allows for the differentiation of the desired EMG signal from the electromagnetic noise, which is detected simultaneously by both electrodes. The amplifier therefore acts as a first stage filter by recognizing the desired, biological signal recorded by the bipolar electrodes and strengthening it; at the same time the amplifier decreases the amplitude of the noise. In this study, built-in sEMG amplifiers enabled us to complete the first stage of the data filtering.

The second stage of the sEMG filtering occurs during digital (software) data filtering (Kamen and Gabriel, 2010a). In this study, after visual examination of the data and identifying the potential sources of noise, I used a band pass filter with 500 Hz low-pass and 10 Hz high-pass cut-off frequencies. Although in healthy muscles the activation frequency does not decrease below 20 Hz, injured muscles might potentially generate lower frequency output (Kamen and Gabriel, 2010a). I therefore decided to allow an extra wide margin (10 Hz) while collecting data from injured muscles. Moreover, a 10 Hz high-pass filter is recommended by the International Society of Electromyography and Kinesiology in order to successfully remove the noise associated with wires and the movement of electrodes.

Additionally, to remove the electrical noise, a narrow notch filter (50 Hz) was used to filter out any 50 Hz electrical radiation signal resulting from mains electrical interference, if it was detected during plotting the frequency spectrum of each individual data.

In the presented example, the sEMG signal was band-pass filtered as a standard procedure (high-pass filter: 10 Hz, low-pass filter: 500 Hz) and notch filtered if the frequency analysis showed the noise at the 50 Hz (notch filter: 50 Hz) (Figure 18 in red).

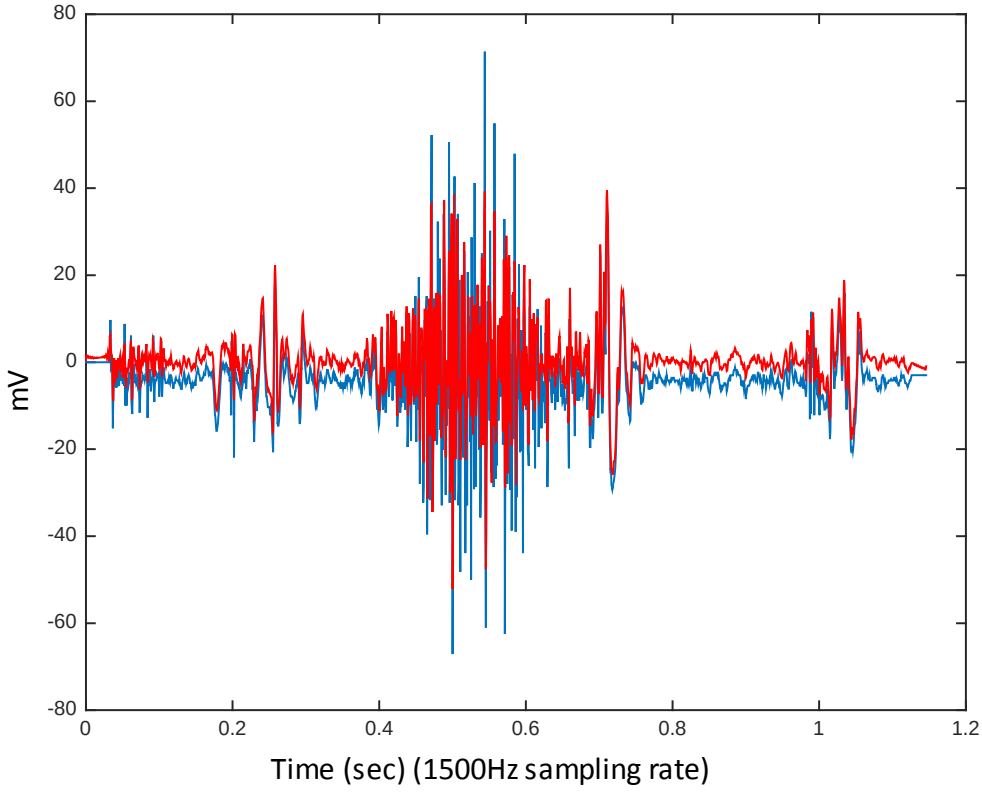


Figure 18: Graphical representation of the sEMG signal before (blue) and after (red) applying a band-pass and notch filter; mV – millivolt. A 2mv offset has been applied for visualisation purposes.

Rectification

Next, each individual sEMG signal was rectified, a data processing procedure which leaves only positive sEMG values for further analysis. The raw sEMG signal represents the difference between two electrodes in the level of de- and re-polarisation of the membrane of muscle fibres. Consequently, the raw sEMG always has corresponding positive and negative values. When smoothing, (averaging in specified time window), the sEMG recordings woud likely give

an average value of approximately zero. To address this issue, a half-wave (cutting off all negative values and leaving only positive values) or full-wave (using the absolute values of each data point) rectification is recommended. In our study, following available guidelines, I chose a full-wave approach to rectifying the sEMG signal.

The rectified signal is presented on Figure 19 in yellow, please note that the raw sEMG signal has been presented with 2 mV offset for visualisation purposes.

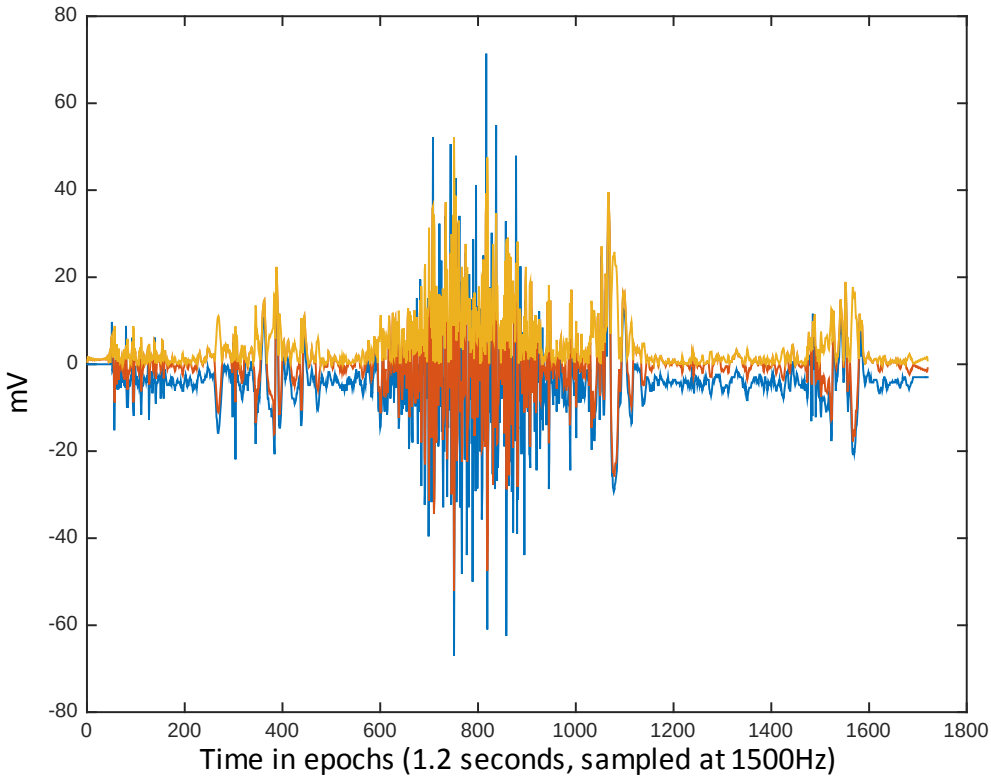


Figure 19: Graphical representation of the raw sEMG signal (blue), filtered sEMG signal (red) and a filtered signal after rectification procedure (yellow); mV – millivolt.

Smoothing

After rectification, the sEMG signal is smoothed. The technique of smoothing (or averaging) the sEMG signal is used to enable the quantification of muscle activation over time. This is measured in millivolts and is performed by averaging the sEMG signal in a specified moving time window. Common window lengths are between 100-200ms, in our study I used a 200ms averaging window. The smoothed sEMG signal is presented on Figure 20 in purple.

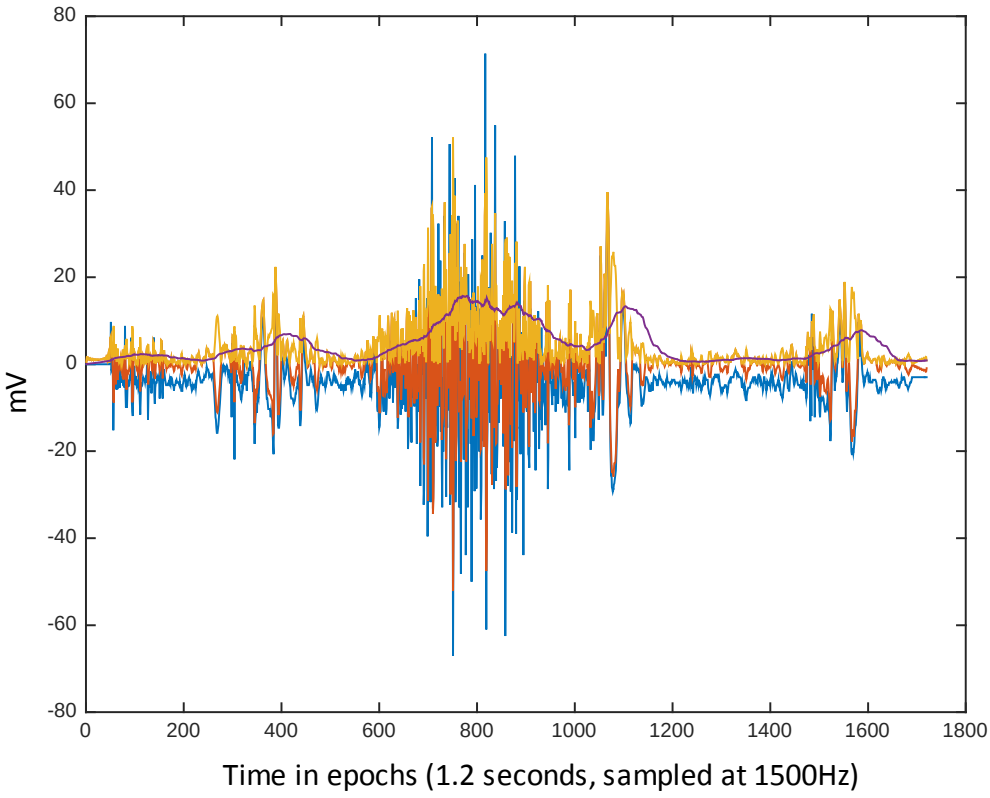


Figure 20: Graphical representation of the raw sEMG signal (blue), filtered sEMG signal (red), rectified sEMG signal (yellow) and smoothed sEMG signal (purple); mV – millivolt.

Log transforming

After these procedures, each separate sEMG signal was log transformed, which enabled us to limit the influence of the larger, more superficial, fast-twitch muscle fibres therefore yielding a composite sEMG with a quasi-linear measure of muscle activation (Robertson, 2004). This best served the purpose of this study and enabled us to limit the effect of outliers in the statistical analysis.

After these processing procedures, each individual data recording for each muscle was divided into phases, according to both visual kinematic data examination in the CodaMotion software interface, and either kinetic or kinematic data. This was achieved by close examination of both outputs and defining the time of the occurring events. Each phase of each movement has its own specific events that define the beginning and end of the phase, which are described in detailed on page 126.

For example, the early phase of both SHF and SLS movement is the beginning of load shift from bilateral to unilateral stance, defined as an initial lateral load shift on the force plate. Firstly, the approximate time of this event was defined by the visual data examination of the stick-figure on the interface of the CodaMotion software, with a special attention given to the Z-axis output on the stick-figure and the force plates (Figure 21).

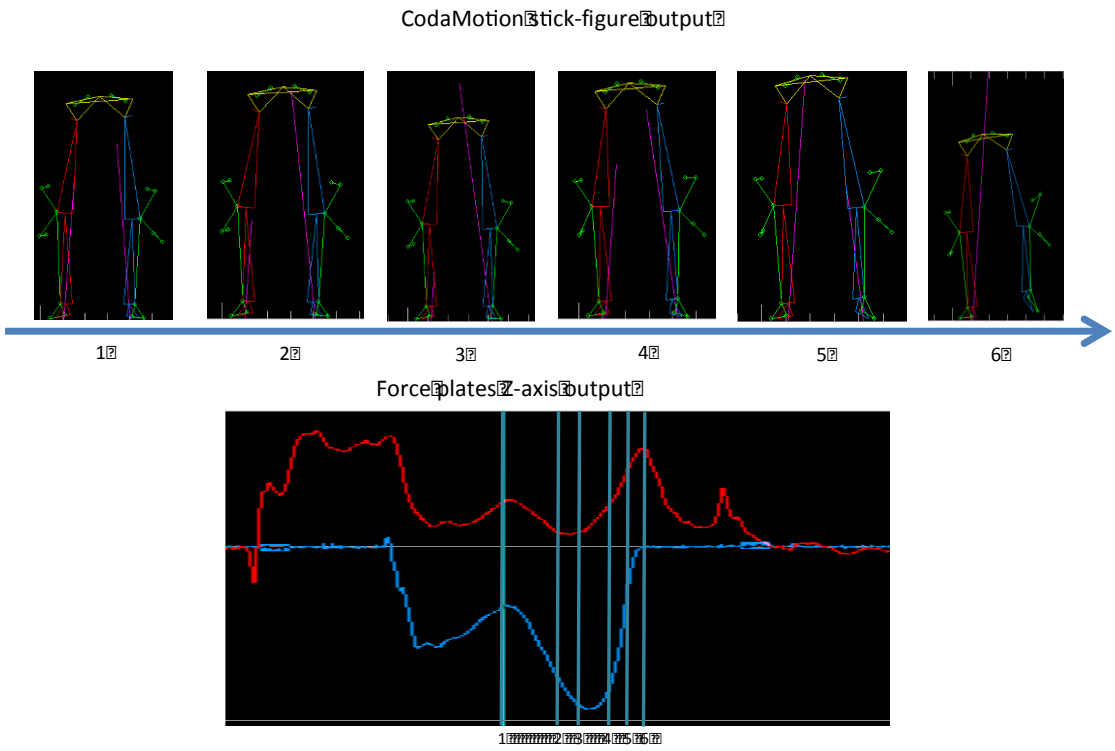


Figure 21: A graphical representation of the relevant outputs of the CodaMotion software interface; top row – stages of the change form a bilateral to unilateral stance characterising the early phase of both SHF and SLS movement; bottom picture – Z-axis output from both force plates. The numbers of the phase in the top row correspond to the numbers shown on the force plate output.

In this data sample of the left standing hip flexion movement (Figure 21), the participant is establishing his bilateral position on a force plate (1), then he pushes off by his left leg (2 and 3), after that he starts lifting his left leg (4) and starts offloading the left force plate. Then he proceeds to lift left leg (5), which leads to complete off-load of the force plate (6). In this part of the data, the push-off phase (so the early phase in SHF and Moving I phase in SLS) is defined between time phases (3) and (4). In order to establish the exact moment of the lateral push,

the output from the force plate in all three planes was transferred into a MatLab program for the detailed inspection of this movement signature (Figure 22).

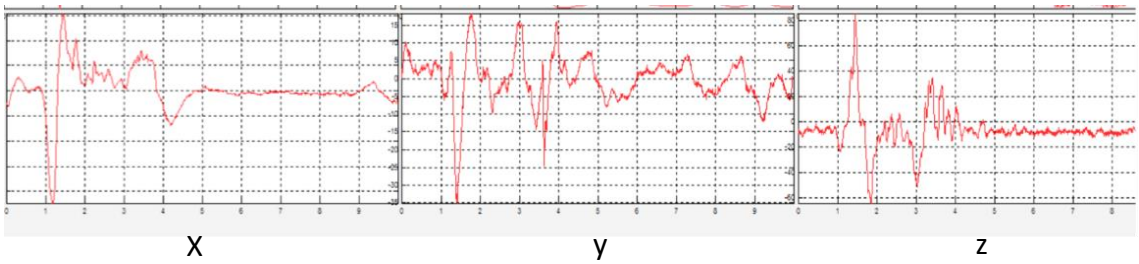


Figure 22: A relevant output from the interface of the custom made MatLab program, showing the X, Y and Z axes outputs from a force plate.

In particular, the X-axis output from the force plate is then examined closely in the approximate time window, which was established previously by the visual data examination in the CodaMotion software interface, as the approximate time of the occurrence of this movement; in this case between 1 and 1.2. on the presented example, a clear negative *peak* in the X-axis, indicating the maximal lateral push (in this case left leg push-off indicated a negative direction of the lateral push) is visible, and marked manually, as presented on Figure 23. The peak rather than first deflection was chosen as this was less susceptible to artefact such as that due to normal postural away.

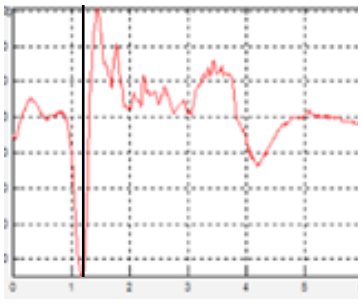


Figure 23: A part of the interface of the custom made Matlab program showing the X-axis output of the force plate; black line indicates a moment of lateral push on the force plate, which indicated the moment of the change from the bilateral to unilateral stance.

In order to define the phase, 50 ms is then retracted and 50 ms was added by the MatLab programme to the marked point in time to ensure a time period reflective of movement onset. This timeframe is then transferred to all the other data outputs – separate sEMG outputs from the gluteus medius and adductor longus muscles, and the hip joint coronal, sagittal and horizontal plane, in both standing and moving legs in SHF and only the moving side in SLS.

In a similar manner, other phases of SHF and SLS movements were defined based on their specific characteristics (p. 126).

Kinematic data

After the definition of the relevant movement phases for each movement, the event times of the defined phases were further applied to the kinematic outcome measures: hip joint rotations in coronal, sagittal and horizontal plane. The kinematic data were collected at a sampling frequency of 200 Hz.

The kinematic data received from the markers are highly susceptible to noise. One of the main source of this noise is a very short period of time when the CodaMotions cameras are acquiring the data in order to capture the exact marker position and prevent from 'smearing' of the data during fast movements. This period of the actual data acquisition is not dependent of the sampling frequency. The noise that may affect the real signal is a high-frequency noise mainly arising from the photo-detector current noise in the cameras or the sub-optimal room lightning. It is usually rather small in x- and z-axes. However, this noise may become more significant in the y-axis, which is derived by the triangulation calculation of the two outer CodaMotion cameras.

Additionally, there is high risk of low frequency noise associated with the fact that the markers are put on the participant's skin, which may provide some movement fluctuations.

The higher frequency components of the final noise can be filtered out (smoothed) by a low pass filter. In this study I used 20 Hz low pass filter to smooth out the high frequency noise. This frequency is recommended by the software provider as optimal in order to remove noise but prevent data loss (Contents, 2004). This process was implemented automatically within the CodaMotion software, therefore graphical representation of the effect of filtering and smoothing of the data was not possible to present.

Chapter 5: Reliability study

Chapter overview

This chapter addresses the inter-rater reliability of the data collection and analysis method, which is further used in the experimental chapters of the thesis. 24 healthy participants took part in the study, performing standing hip flexion and single leg squat, on two occasions with a one-week interval.

Introduction

Reliability of the measurements, defined as repeatability and stability of the test results as well as a minimal measurement error over time or occasions, is essential in sports medicine research (Atkinson and Nevill, 1998, Downing, 2004). If the measurement method is not repeatable, the results lose their meaning and need to be interpreted with caution, or not interpreted at all (Downing, 2004). Moreover, good reliability is an essential part of the validity of the results (Downing, 2003, Portney and Watkins, 2000).

Surface electromyography (sEMG) and kinematic measurements in biomechanical research are typically reported to have moderate to high reliability (Kollmitzer et al., 1999b, McGinley et al., 2009) according to the criteria established by Portney and Watkins (Portney and Watkins, 2000). sEMG measurements of the pelvis and hip musculature during concentric and eccentric movement and rehabilitation exercises were reported to have an ICC of between 0.5 – 0.95 (Claiborne et al., 2009, Bolgla and Uhl, 2007) whereas a systematic review of 3D kinematic measurement reliability reported that among 23 reviewed studies the reported error (standard deviation or standard error) of the measurements was less than 5 degrees with only a few exceptions (McGinley et al., 2009). However, the classic application of 3D kinematic measurements is for gait analysis, with good reliability within a given laboratory but not as

good between laboratories. There is a paucity of research determining the reliability of kinematic measurements in other functional manoeuvres (Goodwin et al., 1999).

Reliability of the results obtained by both sEMG and kinematic measurement devices has a considerably high user dependency due to a large number of extrinsic factors potentially affecting it (Monaghan et al., 2007). As mentioned previously in the Chapter 3: Methods (p. 85), sEMG signals will be significantly altered depending on the electrode location on the muscle and, for example, due to picking up signals from other muscles (cross-talk), or poor alignment of the electrode with the muscle fibres. Additionally, the quality of sEMG measurements might be compromised due to improper skin preparation prior to the electrode placement that is a lack of shaving, rubbing and sanitising. Because my testing procedure involved movement, there was also potential for the electrodes to be partially dislodged, which could also significantly alter the sEMG output. Movement artefact was an additional concern. Therefore lack of reproducible and careful process of securing the electrodes may also be a factor affecting sEMG measurement reliability.

In 3D kinematic measurements the critical extrinsic factors potentially affecting the results were the laboratory alignment and placement of the infra-red markers on participant's body (Monaghan et al., 2007). Poor alignment of the CodaMotion cameras and, consequently, noisy definition of the X, Y and Z axes in the laboratory would affect the levels of joint rotations defined for a given participant and reduce the validity of between-subject comparisons. Inaccurate placement of the infrared markers on a participant's anatomical landmarks, which serve as the base of the joint centre calculation, would mean the measurements are not only unreliable, but also naturally not valid.

Considering such a high number of potential user-dependent errors that might affect the reliability of the measurements, a study was performed to establish the reliability of the

measurement method applied in the studies included in the thesis – the alternative hypothesis was that the method of measurement and data analysis is reliable. The specific aims of this study were to establish the reliability of the sEMG and kinematic measurements and analysis method when performing standing hip flexion (SHF) and single leg squat (SLS) manoeuvres in order to inform our analysis of study findings in pathological groups. As further applied in other studies included in the thesis, the analysis focused on the gluteus medius to adductor longus muscle activation ratio (GM:AL) and the hip joint kinematics in sagittal, coronal and horizontal planes.

Methodology

Participants

The study was approved by the Queen Mary University of London Ethics of Research Committee and all study participants gave verbal consent to take part in the study after they read the patient information sheet and their question were answered. A convenience sample was recruited, 21-24 years of age, performing various sports at an amateur level (football, rugby, hockey, running, weightlifting, dancing, mixed martial arts, cricket, squash, tennis). Testing took place over an 18-month period, across the data collection phase of the observational study.

sEMG electrodes and CodaMotion markers placement

All study participants were invited to the Human Performance Laboratory (HPL) for the data collection process. They were asked to bring their own pair of shorts uncovering their legs. They were then asked to lie down on the plinth placed in a locked office or behind the screens in HPL and certain areas of their body (abdominal area, groin, legs and buttocks) were then uncovered. Then the participants were asked to perform standardised movements and

isometric muscle contractions in order to define the location for sEMG electrode placement. These areas were then shaved, lightly abraded and alcohol-wiped in order to optimise the electrodes' capability to record the electrical signal. sEMG electrodes, to which the disposable sensors equipped with ultrasound gel were attached, were then placed on previously prepared areas and secured with tape in order to prevent dislodging during subsequent dynamic data collection.

In a standing position, the CodaMotion markers were placed on the anatomical landmarks of participant's pelvis and lower limbs according to a modified Helen-Hayes protocol (Monaghan et al., 2007), and secured with tape.

Data collection protocol

All participants attended the HPL for the data collection process twice, with between seven and ten days between visits. This timeframe was chosen in order to minimise the measurement bias associated with an altered muscle signal due to change of training load or strategy.

The participant was asked to perform two different movement tasks; each task was performed three times with each lower limb. Before the data collection, the participant was given clear instructions regarding each task and a suitable amount of time to familiarise themself with each task.

Firstly, the participant was asked to perform a standing hip flexion manoeuvre. He/she was instructed to step on the force plate for three seconds, then lift a chosen leg up to achieve the hip and knee 90° flexion, hold it for three seconds, then put it back down and step back from the force plate.

The second task was the single leg squat manoeuvre. The participant was asked to step on a force plate, lift a chosen leg up for three seconds and perform a comfortable single leg squat

on the supporting leg. He was instructed to stay in this position for three seconds, after which he was asked to rise from the squat, put the elevated leg back on the force plate and step off.

Data analysis

Electromyographic, kinematic and kinetic data were further analysed using a set of custom made MatLab programmes (version 2008 - 2015, The Mathworks, Natick, MA, USA) and SPSS statistical program (IBM Corp. Released 2013. IBM SPSS Statistics for Mac, Version 22.0. Armonk, NY: IBM Corp).

Dividing the SHF and SLS movements into phases

Following the usual methodology developed for the studies included in the thesis, the SHF was divided into three phases: early, middle and late. The early phase was initially defined as 50 ms before and 50 ms after the initial lateral push of the lifted leg. The late phase was defined as 50 ms before and 50 ms after the stable one leg stance with the other leg flexed in the hip and knee to 90°. The middle phase was defined as occurring between the early and late phase, which include the actual lifting movement of the leg.

The SLS movement was divided into seven phases: four movement phases and three stable phases (Table 10).

Phase number	Phase characteristics	Phase code	Phase description
1	Movement phases	M1	Lateral shift of the load - initiation of change from bilateral to unilateral stance
2		M2	Change from bilateral to unilateral stance
3		M3	Squatting down
4		M4	Squatting up
5	Stable phases	S1	Stable unilateral stance prior to performing squat
6		S2	Stable stance in unilateral maximal squat position
7		S3	Stable unilateral stance after to performing squat

Table 10: Table showing the division of the SLS movement to seven phases

Statistical data analysis

Firstly, the quality all of the individual raw sEMG data were manually checked with parti cular attention to the data within previously defined phases. If the data quality was poor within any of the analysed phases, the record was deleted. If the data quality was poor beyond these phases, but acceptable within, the record was accepted. This was necessary as there were periods during the data collection phase when the sEMG collection unit developed intermittent faults. The 50Hz filter was available in case of the presence of commonly occurring electrical noise and applied if necessary. Then all of the finally approved sEMG data were 12Hz high-pass and 400Hz low-pass filtered, smoothed and rectified. The means were then calculated for each phase in each individual for each leg, across three repetitions, for both testing occasions separately.

ICCs values with the 95% CI were then calculated in a two way mixed model (with random effects for the study participants but fixed effect for the rater) to establish an inter-rater reliability of the electromyograhic and kinematic data collection and analysis method (de Vet et al., 2006, Shrout and Fleiss, 1979). Further, one-way ANOVA and t-tests were performed to

establish the overall agreement between two data series. To interpret the reliability values, standard criteria were referred to (Portney and Watkins, 1993)(Table 11).

Value range	Description
< 0.59	Poor
0.60 – 0.74	Moderate
0.75 – 0.89	Good
≥0.90	Excellent

Table 11: The interpretation of ICC values (Portney and Watkins, 2000).

Results

Twenty-eight participants took part in the reliability study. Owing to technical difficulties resulting in poor data quality, the measurements from twenty-four participants were finally analysed.

Standing hip flexion

sEMG

The overall reliability of the method of the GM:AL electromyography data collection and analysis of the SHF manoeuvre was moderate (ICC=0.71, CI=0.63 – 0.78, $F = 2.23$, $p = 0.14$). Separate analysis showed a higher reliability of the right SHF manoeuvre (ICC=0.8, CI = 0.7 – 0.8, $F = 15$, $p = 0.62$) than left SHF manoeuvre (ICC = 0.6, CI = 0.41 – 0.73, $F = 2.68$, $p = 0.11$).

When analysing separate phases, the highest (good) reliability of the measurements was achieved in the early phase of SHF (ICC = 0.83, CI= 0.73 – 0.83, $F = 0.43$, $p = 0.51$), moderate reliability was obtained in the middle (ICC = 0.67, CI = 0.48 – 0.8, $F = 1.41$, $p = 0.24$) and poor in the late (ICC = 0.58, CI = 0.32 – 0.74, $F = 0.59$, $p = 0.45$) phases of SHF movement.

Poor reliability was achieved in moving (ICC = 0.46, CI = 0.2 – 0.63, F = 0.84, p = 0.36) and moderate in stance (ICC = 0.6, CI = 0.44 – 0.7, F = 0.05, p = 0.95) phases of SHF, when analysed separately. A summary of the results is presented in Table 12.

Phases of SHF	Measured side	L SHF (ICC)	R SHF (ICC)	Combined L SHF (ICC)	Combined R SHF (ICC)	Combined L+R SHF (ICC)
early	Moving	0.54	0.83	0.6	0.8	0.83
	Stance	0.71	0.88			
middle	Moving	0.58	0.63			0.67
	Stance	0.63	0.72			
late	Moving	0.61	0.72			0.58
	Stance	0.32	0.51			

Overall	0.71
Moving side L+R SHF	0.55
Stance side L+R SHF	0.6

Table 12: Table summarizing the ICC values measured with the 95% of the confidence intervals for the measurements and the data analysis methods of the gluteus medius to adductor longus muscle magnitude activation ratio during three phases of the SHF manoeuvre, measured on the moving and stance side. SHF= standing hip flexion; L = left, R = right; GM:AL = gluteus medius to adductor longus muscle activation magnitude ratio; ICC = intraclass correlation coefficient; CI = confidence interval.

Kinematics

The overall reliability of the kinematic measurements of the hip joint rotations in coronal, sagittal and horizontal plane in SHF was excellent (ICC = 0.97, CI = 0.97 – 0.98, F = 1.63, p =0.2) with a similar reliability of the right and left leg manoeuvres (ICC = 0.97, CI = 0.97 – 0.98, F = 1.4, p = 0.24 and ICC = 0.97, CI = 0.97 – 0.98, F = 0.39, p = 0.53 respectively).

The analysis of the separate planes showed excellent reliability in the sagittal plane (ICC = 0.99, CI = 0.98 – 0.99, F = 0.6, p = 0.8) followed by the coronal plane (ICC = 0.91, CI = 0.89 – 0.93, F = 3.09, p = 0.8), but poor reliability in the horizontal (ICC = 0.48, CI = 0.33 – 0.6, F = 1.08, p = 0.3) plane.

The analysis of separate SHF movement phases showed an excellent reliability in the early phase of SHF (ICC = 0.93, CI = 0.91 – 0.95, F = 0.04, p = 0.84), middle (ICC = 0.97, CI = 0.97 – 0.98, F = 1.31, p = 0.25), and late (ICC = 0.98, CI = 0.98 – 0.99, F = 0.96, p = 0.33).

Both moving and stance measured sides showed excellent reliability, with the moving leg higher than the stance leg (ICC = 0.98, CI = 0.98 – 0.99, F = 2.72, p = 0.1 and ICC = 0.91, CI = 0.89 – 0.92, F = 0.001, p = 0.98 respectively).

The ICC values for separate planes in each movement phase are presented in Table 13.

Phases of SHF	Measured side	Plane	L SHF (ICC)	R SHF (ICC)	L+R SHF (ICC)	L+R SHF (ICC)
early	mov	cor	0.84	0.81		
		sag	0.88	0.84	0.66	
		hor	0.27	0.34		
	st	cor	0.61	0.9		0.93
		sag	0.85	0.92	0.66	
		hor	0.29	0.41		
middle	mov	cor	0.82	0.82		
		sag	0.85	0.62	0.64	
		hor	0.52	0.2		
	st	cor	0.67	0.8		0.97
		sag	0.9	0.88	0.66	
		hor	0.28	0.45		
late	mov	cor	0.74	0.59		
		sag	0.83	0.7	0.67	
		hor	0.68	0.47		
	st	cor	0.86	0.8		0.98
		sag	0.89	0.87	0.72	
		hor	0.38	0.53		

Overall	0.97
Coronal plane L+R SHF	0.91
Sagittal plane L+R SHF	0.99
Horizontal plane L+R SHF	0.48
Moving side L+R SHF	0.98
Stance side L+R SHF	0.91

Table 13: Table summarizing the ICC values measured with the 95% of the confidence intervals for the measurements and the data analysis methods of the hip joint kinematics in all three planes during three phases of the SHF manoeuvre, measured on the moving and stance side. SHF= standing hip flexion; L = left; R = right; ICC = intraclass correlation coefficient; CI = confidence interval; mov = moving side; st = stance side; cor = coronal plane; sag = sagittal plane; hor = horizontal plane.

Single leg squat

sEMG

The overall reliability of the SLS manoeuvre across all phases was moderate (ICC = 0.72, CI = 0.67 – 0.764, F = 0.002, p = 0.96). The reliability of the right SLS was higher than the left SLS (ICC = 0.78, CI = 0.72 – 0.83, F = 5.37, p = 0.21 and ICC = 0.66, CI = 0.57 – 0.73, F = 3.47, p = 0.64 respectively).

When analysing the separate phases, the highest reliability was achieved in the moving 2 phase (ICC = 0.83, CI = 0.73 – 0.89, F = 0.29, p = 0.59) followed by the stance 1 (ICC = 0.78, CI = 0.65 – 0.86, F = 0.63, p = 0.43), moving 1 (ICC = 0.75, CI = 0.61 – 0.84, F = 0.01, p = 0.91), moving 3 (ICC = 0.67, CI = 0.47 – 0.79, F = 0.29, p = 0.87), stance 2 (ICC = 0.66, CI = 0.47 – 0.79, F = 0.54, p = 0.47), stance 3 (ICC = 0.66, CI = 0.44 – 0.78, F = 0.05, p = 0.82) and moving 4 (ICC = 0.65, CI = 0.43 – 0.77, F = 0.09, p = 0.72) .

When analysing the stance and movement phases, higher reliability was achieved in the moving phases (ICC = 0.73, CI = 0.67 – 0. 77, F = 0.009, p = 0.92) than the stance phases (ICC = 0.7, CI = 0.61 – 0.77, F = 0.005, p = 94).

The summary of these results is presented in Table 14.

Phase of SLS	L SLS (ICC)	R SLS (ICC)	L SLS (ICC)	R SLS (ICC)	L+R SLS (ICC)	L+R SLS (ICC)
Mov 1	0.72	0.78				0.75
Mov 2	0.82	0.84			0.73	0.83
Mov 3	0.65	0.73				0.67
Mov 4	0.54	0.76	0.66	0.78		0.65
St 1	0.78	0.78				0.78
St 2	0.68	0.54			0.7	0.66
St 3	0.54	0.76				0.66
Overall		0.72				

Table 14: Table summarizing the ICC values measured with the 95% of the confidence intervals for the measurements and the data analysis methods of the gluteus medius to adductor longus muscle magnitude activation ratio during seven phases of the SLS manoeuvre, measured on the moving side. SLS = single leg squat; L = left; R = right; ICC = intraclass correlation coefficient; CI = confidence interval; mov1 = phase moving 1; mov2 = phase moving 2; mov3 = phase moving 3; st1 = phase stance 1; st2 = phase stance 2; st3= phase stance 3.

Kinematics

The overall reliability of the hip joint kinematic measurement and analysis method was excellent (ICC = 0.94, CI = 0.93 – 0.95, F = 1.52, p = 0.22). The reliability of the right SLS manoeuvre was higher than the left SLS manoeuvre (ICC = 0.93, CI = 0.92 – 0.94, F = 0.86, p = 0.35 and ICC = 0.9, CI = 0.71 – 0.93, F = 0.54, p = 0. 23 respectively).

Analysis of the separate planes showed a good reliability in coronal and sagittal planes (ICC = 0.87, CI = 0.83 – 0.9, F = 4.39, p = 0.04 and ICC = 0.86, CI = 0.82 – 0.89, F = 1.26, p = 0.26 respectively), but poor reliability in the horizontal plane of SLS (ICC = 0.38, CI = 0.22 – 0.51, F = 9.46, p = 0.002).

When analysed collectively, both moving and stance phases achieved excellent reliability (ICC = 0.93, CI = 0.91 – 0.94, F = 1.91, p = 0.17 and ICC = 0.94, CI = 0.92 – 0.95, F = 0.82, p = 0.37, respectively)

The analysis of the separate movement phases showed excellent reliability in the moving 2 (ICC = 0.92, CI = 0.88 - 0.94, F = 0.33, p = 0.57), moving 3 (ICC = 0.96, CI = 0.94 - 0.97, F = 0.07, p = 0.8), moving 4 (ICC = 0.91, CI = 0.86 - 0.95, F = 4.16, p = 0.44), stance 1 (ICC = 0.95, CI = 0.93 - 0.97, F = 0.002, p = 0.97) and stance 2 phases (ICC = 0.92, CI = 0.89 - 0.95, F = 0.000, p = 0.99); and moderate reliability in the moving 1 (ICC = 0.88, CI = 0.82 - 0.92, F = 0.7, p = 0.79) and stance 3 (ICC = 0.9, CI = 0.85 - 0.92, F = 0.76, p = 0.65) phases. A summary of these results is presented in Table 15.

Phase of SLS	Plane	L SLS (ICC)	R SLS (ICC)	L SLS (ICC)	R SLS (ICC)	L+R SLS (ICC)	L+R SLS (ICC)	L+R SLS (ICC)
	cor	0.85	0.6			0.72		
mov 1	sag	0.54	0.88			0.71	0.88	
	hor	0.54	0.93			0.74		
	cor	0.64	0.81			0.73		
mov 2	sag	0.36	0.88			0.62	0.92	
	hor	0.5	0.43			0.47		
	cor	0.59	0.82			0.7		
mov 3	sag	0.78	0.7			0.74	0.96	0.93
	hor	0.69	0.18			0.43		
	cor	0.22	0.8			0.51		
mov 4	sag	0.29	0.87			0.58	0.91	
	hor	0.49	0.33			0.41		
	cor	0.56	0.82	0.96	0.93	0.69		
st 1	sag	0.59	0.84			0.72	0.95	
	hor	0.48	0.49			0.48		
	cor	0.43	0.88			0.66		
st 2	sag	0.53	0.68			0.61	0.92	
	hor	0.51	0.33			0.42		0.94
	cor	0.32	0.8			0.56		
st 3	sag	0.4	0.77			0.58	0.9	
	hor	0.59	0.53			0.56		

Overall	0.94
Coronal plane SL+SR	0.87
Sagittal plane SL+SR	0.86
Horizontal plane SL+SR	0.38

Table 15: Table summarizing the ICC values measured with the 95% of the confidence intervals for the measurements and the data analysis methods of the hip joint kinematics in all three planes during seven phases of the SLS manoeuvre, measured on the moving side. SLS = single leg squat; L = left; R = right; ICC = intraclass correlation coefficient; CI = confidence interval; mov1 = phase moving 1; mov2 = phase moving 2; mov3 = phase moving 3; st1 = phase stance 1; st2 = phase stance 2; st 3= phase stance 3; cor = coronal plane; sag = sagittal plane; hor = horizontal plane.

Discussion

The methods of data collection and - in particular – analysis, implemented in the thesis are relatively complex and include multiple stages of data collection and processing. In order to establish the reliability of the data collection and analysis methods, the reliability of the final result was analysed, rather than the individual stages of data processing. The overall reliability of the measurements and the analysis method was moderate to high, in agreement with previous research (Kollmitzer et al., 1999b, McGinley et al., 2009).

There are many methods of assessing the reliability in the biomechanical and rehabilitation research, and little consensus has been achieved regarding which method should be used (Rankin and Stokes, 1998). It has been agreed that reliability measurements should in general represent the true variability of the observations (Riddle et al., 1989), but this has not led to researchers' achieving methodological agreement. Each reliability analysis method is associated with some advantages and disadvantages, and although some general guidelines exist regarding the methods used in a given field, the final choice of the method needs to be carefully chosen by the researcher, based on the aims of a particular study.

In my study I chose to analyse reliability using the ICC, performing multiple measurement on separate groups of data (such as one outcome measure during a particular movement phase) as well as analysing reliability throughout the whole movement. Therefore, the natural limitation of this analysis is the lack of limits of agreement or equivalent, which may have been achieved by employing an approach such as Bland and Altman's analysis plot (Atkinson and Nevill, 1998). Additionally, the ICC is just a one point representation of the reliability based on a given sample, which also limits its clinical interpretation (Rankin and Stokes, 1998). Therefore some authors suggest complimenting these measurements with the Bland and Altman 95% limits of agreement test (Bland and Altman, 1986).

There were several reasons why I chose to accept the results of the performed ICC measurements. Firstly, this test is certainly a better option for assessing the systematic difference between measurements than for example Person's correlation, which do esn't allow to relate the findings further to the individuals. ICC is actually influenced by the magnitude of between-subjects variation, which makes it more clinically applicable.

Secondly, although some authors encourage to compliments ICC with Bland and Altman 95% limits of agreement test (Bland and Altman, 1986), these measurements are also suggested for assessment of the reliability of the measurement method rather than the intra-rater reliability of the scientist performing measurements with a selected method (Costa-Santos et al., 2011, Bland and Altman, 1986). The aim of my study was to establish my reliability of using and analysing the method, which was previously used and validated. Therefore, adding the Bland and Altman results may not have been appropriate in this particular case (Costa-Santos et al., 2011).

Further, considering the amount of data that was analysed in my study, the division of both movements into a number of phases and both legs being measure in each condition, there were a very high number of outcome measures in this study. Adding further analysis would be rather a confusing than a clarifying factor, and might have decreased the understanding of the overall reliability of the measurements.

The overall reliability of the method of collecting and analysing the electromyographic data implemented in the thesis was moderate in both movement manoeuvres. This is in agreement with previous studies measuring the reliability of sEMG measurements in controlled movements (Zech et al., 2008, Rainoldi et al., 2001, Heinonen et al., 1994).

The movements implemented in this study, however, were not as fully controlled as would be the case with static, isometric contractions (Heinonen et al., 1994). Both SHF and SLS

manoeuvres are complex tasks, used clinically to assess the many aspects of the function of the hip and pelvis areas (Boudreau et al., 2009, Crossley et al., 2011, Marshall et al., 2015). One study measured the reliability of leg muscle electromyography during vertical jumping and implemented a similar method of dividing the movement into phases based on the kinematic events (Goodwin et al., 1999). The reliability was reported to be poor to moderate depending on the measured muscles. A satisfactory reliability was achieved in the rectus femoris and vastus medialis muscles (ICC = 0.88 and ICC = 0.7 respectively), but poor reliability (ICC < 0.25) was reported for the biceps femoris and gastrocnemius muscles. The hip and pelvis musculature was not measured in that study but is likely to be subject to similar concerns.

Compared to the vertical jumping, the reliability of the GM:AL muscle activation ratio reported in my study was considerably higher for both manoeuvres with an ICC of 0.72 for SHF and also 0.72 for the SLS. The reason for a better reliability of my study may be another level of data normalisation implemented in the electromyographic data analysis – muscle activation magnitude ratio rather than measuring only one muscle, which was the case during the vertical jump. The method of muscle ratio analysis was chosen in order to avoid the standard way of electromyographic data normalisation (such as normalising the sEMG signal to the maximal voluntary contraction or to the peak, or mean activation during the performed movement), which could not be implemented in the experimental studies including potentially injured muscles (van der Hulst et al., 2010a, van der Hulst et al., 2010b, Morrissey et al., 2012a).

Additionally, one of the aims of the thesis was to determine the overall muscle activation patterns and the functional characteristics of the symptomatic study participants rather than establishing the definitive level of muscle activation or obtaining very discreet activation information such as motor unit action potential (MUAP) or muscle fibre level data. The method of the agonist-antagonist muscle ratio seemed therefore optimal to achieve the study aims.

Relatively high reliability reported in this study may suggest a robustness of this method for the functional assessment of the muscle activation, which may be implemented in the clinical settings. However, an additional analysis of separate GM and AL muscle activation was performed only in the right SHF movement, and the reliability of both separate muscles was similar to the ratio (GM: ICC = 0.67, CI = 0.51 – 0.78, F = 0.24, p = 0.62; AL: ICC = 0.75, CI = 0.63 – 0.83, F = 0.95, p = 0.33).

Additionally, compared to the vertical jump movement, both SHF and SLS manoeuvres are more static, which may increase the reliability of the measurements. Interestingly, in the SHF (where both the moving and stance side were measured and analysed) a higher reliability was achieved in the moving than stance phase. Moreover, when analysing the phases of movement, the highest reliability was achieved in the early phase, during the initial lateral shift of the loading from the bilateral to unilateral stance, and the lowest – during the stable one leg stance. In SLS, where only the moving leg was analysed a separate analysis was performed to establish the reliability of the moving and stance phases of the movement, and the moving phases again achieved a higher reliability than the stance phases. A generally higher reliability of the moving than stance phases in SHF and SLS may be due to a higher muscle activation magnitude during movement than stance phases. A relatively low muscular activation during stance may be similar to the static muscle electrical output, which was also recorded and analysed. Nevertheless, both stance and movement phases achieved a similar and moderate reliability.

The separate ICCs for GM:AL ratio in each movement phase in both SHF and SLS were moderate and acceptable. However, the ICC on the stance side of the late phase during SHF was very low (0.322). This is a surprising result and clearly differs from the reliability levels of all of the other single measurements. The reason for such discrepancy is not clear. It may be due to the learning bias as the SHF manoeuvre, although apparently easy, presented a

challenge for study participants during the late phase to maintain stable unilateral stance. During the second testing occasion the study participants may have already expected and learned how to achieve an optimal level of stability when standing still with one knee lifted, which has affected the reliability of this particular phase. It may also be that there are alternative muscular co-ordination strategies the body can adopt to maintain the position such as is the case with scapular movement (Worsley et al., 2013).

Interestingly, the reliability of the manoeuvres performed on the right and left side, the reliability was consistently higher in the right compared to the left, in both SHF and SLS manoeuvres. I found no studies specifically investigating the reliability of the right compared to left side in the biomechanical literature, but it may be hypothesised that following the general population the majority of the participants were right side dominant (Lansky et al., 1988). Thus, performing the movement on their dominant and preferred side may have been more reproducible and therefore resulted in higher reliability values.

Kinematics data collection and analysis

The overall reliability of the method of 3D hip joint kinematic data collection and analysis during SHF and SLS manoeuvres was excellent, with SHF achieving a higher reliability compared to SLS.

Although 3D kinematic measurements are widely used in clinical research, two published studies have reported a poor to moderate reliability of 3D kinematic measurement *between* laboratories (Noonan et al., 2003, Gorton Iii et al., 2009), which may question the clinical applications of such measurements. A recent systematic review on the reliability of 3D kinematic measurements used in gait analysis (McGinley et al., 2009) has reported that although some errors and bias do exist in these measurements, they may not affect the clinical applications of 3D gait analysis as such. Although the review focuses specifically on 3D gait

analysis, the typical method of the assessment of the kinematic measurements in reviewed studies is based on the division of the gait pattern into phases (Baker, 2006) – which may be comparable to the method implemented in my study. Interestingly, one of the results of the review shows is that hip joint kinematic measurements tend to show a lower reliability compared to pelvis, knee and foot.

One study, not included in the review, shows a high reliability of the 3D gait analysis kinematic measures in the participants with the hip osteoarthritis (Laroche et al., 2011), which may be associated with the experimental studies included in the thesis due to similarity of the coronal plane kinematic impairment between hip osteoarthritis and groin pain.

The analysis of the reliability of the hip joint movement in the separate planes showed a good to excellent reliability in the coronal and sagittal plane (slightly higher in sagittal in SHF). However, a consistently lower reliability level was shown in the horizontal plane in both movements, which supports the results of the previous studies (McGinley et al., 2009). This clear discrepancy between planes may be associated with the hip joint functional anatomy and, consequently, the function of the muscles acting on the hip joint. Whereas the coronal and sagittal plane movements are mostly achieved with a large and well-defined muscle groups regardless of the hip position (such as ilio-psoas, adductor and gluteal muscle groups), the movements in the horizontal plane are often controlled by the same muscle, as their accessory function (adductor magnus, gracilis and biceps femoris). Moreover, some of the major hip stability muscles such as gluteus medius, act as both internal and external rotators depending on the part of the muscle (Gottschalk et al., 1989). Therefore, the general control and stability in the hip joint in horizontal plane may be worse than other planes, and therefore reliability compromised. This may mean that in the experimental studies including in the thesis, the results obtained from the hip rotations in the horizontal plane should be treated

with caution. In addition, the degree of movement in the horizontal plane is small in these movements, and noise will therefore be relatively high.

A previously mentioned review on the reliability of the 3D gait analysis kinematics (McGinley et al., 2009) emphasises the difficulty of drawing the definite conclusions due to study diversity. However, one of the recommendations of this review is that the acceptability of the reliability level of such measurements is highly dependent of the 'proposed use'. In the experimental studies included in the thesis, the kinematic characteristics of the hip joint in the symptomatic participants were the secondary finding, analysed in the context of the coronal plane muscle activation alterations. Therefore, despite a few cases of low reliability levels in the hip joint kinematic measurements, I decided to accept and implement this method of kinematic data and analysis.

Conclusions

Overall, the reliability of the GM:AL sEMG data collection and analysis was moderate, with better reliability for the right leg manoeuvres. The hip joint kinematic data collection and analysis was moderate with some exceptions (e.g. hip joint horizontal plane kinematics). Some measurement errors were present in the proposed method, but they don't compromise its application and allow the aims of the experimental studies to be met with confidence.

Chapter 6: Observational studies, combined as a cross sectional report

Chapter overview

This chapter summarises the results of the electromyographic and hip joint kinematic measurements of 84 amateur and professional athletes of various sports disciplines, while performing a standing hip flexion and single leg squat tasks.

Introduction

Sports related groin pain (SRGP) is common and recurrent. It is associated with a prolonged time away from sports and may be career ending. Athletes particularly susceptible to SRGP are those participating in high speed rotation-related sporting disciplines requiring repetitive kicking, pivoting, cutting or changing direction such as football, rugby and hockey (Brooks et al., 2005a, Brooks et al., 2005b, Ekstrand and Hilding, 1999, Emery et al., 1999a, Holmich et al., 2010, Werner et al., 2009, Hagglund et al., 2006, Ekstrand and Gillquist, 1983, Hagglund et al., 2009, Hawkins and Fuller, 1999, Holmich et al., 2013, Gibbs, 1993, O'Connor, 2004, Garraway et al., 2000, Emery et al., 1999b).

The diagnosis and treatment of SRGP is challenging (Werner et al., 2009), with ill-defined and multi-structural pathology (Holmich, 2007) and often non-specific symptoms (Ekberg et al., 1988, Falvey et al., 2009). Similar problems have been identified with other pathologies, such as shoulder pain and lower back pain, which present a huge challenge when identifying a distinct structure responsible for the symptoms. In these cases, the rehabilitation strategy focusing on a movement pattern rather than dictated by pathology seems to be the optimal way forward (Worsley et al., 2013, Mottram et al., 2009, Roussel et al., 2013). This presents an

argument that identifying the patterns of movement and muscle activation imbalances in SRGP may improve rehabilitation outcomes and reduce recurrence.

An international agreement meeting held in Doha, Qatar published a report stating that while there is no gold standard diagnostic process for groin pain in athletes, there are three categories for SRGP - 'defined clinical entities', 'hip-related pathology' and 'other conditions' (Weir et al., 2015). Identification of study participants in regards to one of these three groups has been recommended (Weir et al., 2015). The defined clinical entities comprise of adductor-, iliopsoas-, inguinal- and pubic-related groin pain. These entities can co-exist. Further, a set of recommendations for minimum standards of reporting (Delahunt et al., 2015) was published from the same set of meetings. These recommendations have been useful in analysing our findings and presenting our research.

There are a number of reported factors associated with SRGP (Arnason et al., 2004, Cowan et al., 2004a, Crow et al., 2010, Emery and Meeuwisse, 2001, Engebretsen et al., 2010, Ibrahim et al., 2007, Jansen et al., 2010, Malliaras et al., 2009, Mens et al., 2006, Mohammad et al., 2014, Morrissey et al., 2012a, Nevin and Delahunt, 2013, O'Connor, 2004, Thorborg et al., 2014, Tyler et al., 2001, Verrall et al., 2005a, Verrall et al., 2007a). The imbalances in the muscle features and range of movement in the SRGP athletes have been recognised in the Doha agreement and the necessity of including those imbalances in designing the prevention programs has been highlighted. However, little attention has been given to the necessity of recognising the biomechanical signatures associated with SRGP, such as movement pattern differences between injured and uninjured athletes. Nor has any consideration been given to the participation level of the athlete and sports-specificity, in either the published literature or consensus documents.

The large number of reported muscular changes in both static and dynamic tests of athletes with SRGP is very likely to alter the way they move (Suzuki et al., 2001, Worsley et al., 2013). These alterations in movement patterns may be the cause of further damage and lack of successful recovery (Worsley et al., 2013). One study has measured muscle activation magnitude with surface electromyography and found that gluteus medius to adductor longus ratio was significantly decreased in amateur footballers with SRGP compared with matched controls (Morrissey et al., 2012a) which suggests there may be biomechanical imbalances in the coronal plane.

Interestingly, although authors investigating biomechanical associations between SRGP and biomechanics use different diagnostic and inclusion/exclusion criteria in their studies, their findings are similar (Crow et al., 2010, Malliaras et al., 2009, Mens et al., 2006, Nevin and Delahunt, 2013, Thorborg et al., 2014, Verrall et al., 2005a). Some authors are very stringent with included SRGP group, (Thorborg et al., 2011, Holmich, 2007) whereas other define 'groin pain' very broadly and use rather limited inclusion criteria (Arnason et al., 2004). I chose the latter method, with our focus being broad categorisation rather than tissue-specific diagnosis. I appreciated this would yield a more heterogenous group, making any biomechanical patterns less likely to emerge yet also making any revealed patterns more robust and therefore of wider clinical relevance. Nonetheless, our criteria ensured that all subjects had adductor-related groin pain, alongside possible other defined clinical entities such as pubic-related or iliopsoas-related groin pain (Weir et al., 2015).

It is widely recognised that the athletes participating in certain sports disciplines are more susceptible to SRGP than others. A common approach associates this increased risk with repetitive kicking, twisting, cutting and pivoting manoeuvres. However, the mechanisms of SRGP development and the movements that may cause it are still not well understood. In the presence of a number of muscular and range of movement changes shown in association with

SRGP, it is a natural step forward to further explore the biomechanical characteristics of movement and muscle activation associated with SRGP. Further, although twisting, cutting and pivoting manoeuvres are common in all sports with a high incidence of SRGP, the requirements of each sport vary. Additionally, the level of sports and consequently the training load also varies among athletes suffering from SRGP, which suggests that there is an underlying biomechanical deficit, which potentially differs between different athletic groups, that is still under-researched. A lack of success in SRGP treatment among both amateur and professional athletes may mean that there are underlying factors such as alterations in movement patterns and muscle activation, which are under-researched, and may require more attention.

The aim of this study was to characterise the biomechanical patterns in athletes with SRGP participating in various multi-directional sports at professional and amateur levels, with comparison to closely matched controls in order to better understand sports-specific presentations and guide rehabilitation. The alternative hypothesis was that injured would and uninjured subjects would differ in a systematic way. Secondary hypothesis was that the SRGP athletes participating in professional sport would show similar movement and muscle activation patterns compared to well-matched controls, and that these patterns would differ depending on the level and sports discipline played.

The impact of the work is potentially considerable, with there being very little in the literature measuring movement patterns nor comparing participation levels as shown by an absence of recommendations or summary argument in recent consensus statements and an absence of such studies in the systematic review.

Methods

Ethical approval was obtained from Queen Mary Ethics of Research Committee and all of the study participants granted signed informed consent prior to the data collection process. Participants were recruited from local sports clubs, by snowball recruiting and or by contacting sports clubs directly. Control participants were recruited from the same or very similar sources as symptomatic subjects in order to closely match activity levels and training. For example, an injured professional midfielder from one club would ideally be tested alongside an uninjured player midfielder from the same club.

Symptomatic participants were included in the study if they were >18 years old, had unilateral SRGP for at least 4 weeks and if their main pain symptoms were reproduced by the palpation of adductor muscle insertion to the pubic bone and unilateral adductor muscle static resisted adduction test (lying supine), and/or the following tests: abduction passive flexibility testing (lying supine), unilateral iliopsoas muscle strength and flexibility testing, squeeze test in 0°, 45°, 90° (Delahunt et al., 2011a, Delahunt et al., 2011b, Malliaras, Hogan, 2009). Participants were excluded if they tested positive with hip joint tests (passive internal rotation, FABER, quadrant test), had a history of groin or abdominal surgery or true hernia, or had significant lower back pain during clinical examination. Additional exclusion criterion for both symptomatic and asymptomatic participants was a history of previous groin, adductor or abdominal symptoms or incidents. The dominant leg was defined as preferred kicking leg; weight and height were measured using calibrated stadiometer and scales (Seca 761, 217 stadiometer, Seca Scales and Measuring Systems, Birmingham, UK).

Surface electromyography Ag-AgCl round, 1 cm diameter electrodes (sEMG, Noraxon Telemyo 2400T, Scottsdale, Arizona, USA, sampling frequency 1500Hz) were placed within 1.2 cm centre-distance from each other and secured with tape on participants' gluteus medius (GM)

and adductor longus (AL) muscles bilaterally, after standard skin preparation (shaving, rubbing, sterilising). Infra-red active motion analysis system markers sampling at 200Hz (Codamotion Cx1 sensor units, Charnwood Dynamics, Rothley, Leicestershire) were placed on lower limb anatomical landmarks according to a modified Helen-Hayes protocol (Monaghan et al., 2007).

Standing hip flexion (SHF) and single leg squat (SLS) were chosen as the test manoeuvres, with data for both moving and stance legs in SHF and only the stance leg in SLS being collected and analysed. Data were collected whilst participants stood on a force platform (Kistler type 9281B, Kistler Instruments Corporation, Winterthur, Switzerland, sampling frequency 500Hz). The movements were divided into phases: SHF into three and SLS into seven.

The electromyographic data were rectified, smoothed and filtered. The mean electromyography values were computed in each phase for each participant, then GM:AL ratios were analysed individually and group means were then calculated. If the GM:AL ratio showed significant differences within groups, individual muscle sEMG values were further analysed.

The kinematic segmental rotations were defined using the same temporal windows. All data were processed using MatLab (version 2012a, The Mathworks, Natick, MA, USA). Data were collected from both legs in all participants, but for further analysis data from both limbs in control groups, and mainly from the symptomatic side in SRGP participants were used.

Analysis of variance was used with either muscle activation magnitude ratio or kinematic hip joint rotations in one of three planes as the dependent variable. Injury status, level of sport, movement stage and whether the measured limb was in stance or moving were entered as independent factors. Interactions between level of sport (professionals or amateurs) and injury status (injured or uninjured) and phase of movement (early, middle or late) were specified and tested with a post hoc Bonferroni test.

Further analyses were performed in order to determine (i) the differences between the dominant and non-dominant legs of the healthy controls in each group; and (ii) the differences between the healthy controls of the professional football and amateur football subgroups.

Results

Participants

Eighty four athletes participated in the study, thirty nine injured players and forty five well-matched, healthy control players: twenty professional footballers (ten injured and ten uninjured), nineteen amateur footballers (nine injured and ten uninjured), sixteen professional rugby players (eight injured and eight uninjured), fourteen Ultimate Frisbee players (seven injured and seven uninjured) and fifteen Field Hockey players (five injured and ten uninjured) (Table 16).

	Amateur football			Pro Fb			Rugby			Frisbee			Hockey		
	Inj	**Con**	**p**	**Inj**	**Con**	**p**	**Inj**	**Con**	**p**	**Inj**	**Con**	**p**	**Inj**	**Con**	**p**
N	9	10		10	10		8	8		7	7		5	10	
Height	180 (10)	180 (10)	0.52	180.6 (9.7)	178.2 (13.4)	0.17	179.9 (5.0)	182.63 (7.1)	0.39	182.79 (8.1	174.5 (6.97)	0.06	175.8 (9.0)	174.8 (10.8)	0.9
Weight	81 (4)	82 (3)	0.77	76.83 (24.6)	74.6 (17.5)	0.24	86.1 (7.9)	96.38 (15.2)	0.11	77.43 (12.0	71.29 (5.59)	0.24	73.3 (10.4)	76.4 (15.1)	0.77
Age	24 (3)	25 (2)	0.67	20.09 (4.3)	19.7 (2.74)	0.22	20.25 (2.4)	22.6 (3.4)	0.13	26.29 (3.59	24.57 (1.99)	0.29	24 (3.1)	22.8 (2.3)	0.52
Injured leg (Dom:Non)		7:2			2:8			7:1			7:0			2:3	

Table 16: Study participants' characteristics. Am Fb = amateur footballers; Pro Fb = professional footballers, Rb = rugby, Inj = injured, Con = controls, Dom = dominant leg injured, Non = non-dominant leg injured.

A majority of the injured athletes injured their dominant leg (twenty five out of thirty nine). Within the subgroups, the dominant leg was injured in majority of the symptomatic participants in the amateur footballers (seven out of nine), professional rugby players (seven out of eight) and Ultimate Frisbee players (seven out if seven). The non-dominant leg was injured in the majority of the professional footballers (eight out of ten). Field Hockey players did not show any association between the injury and leg dominance (two players were injured on the dominant side, three players on the non-dominant side). The characteristics of the study participants are presented in Table 16.

Standing hip flexion

Either leg in healthy participants versus injured leg in SRGP participants

sEMG

Professional footballers

Professional footballers with SRGP had significantly increased GM:AL ratios compared to well-matched controls while standing on the symptomatic leg in all three phases of the SHF movement (early $p = 0.00011$, middle $p = 0.00000093$ and late phase $p = 0.001$) (Table 17A, Figure 24). Analysis of the individual muscles showed no difference between injured and healthy participants in GM activation, but a significant decrease in AL activation in all three phases of movement (early: $p = 0.00035$, middle: $p = 0.0000012$, late: $p = 0.0017$) in injured compared to healthy participants.

While moving the injured leg, the GM:AL ratio in the injured professional footballers was significantly increased compared to controls in all three phases of movement (early: $p = 0.0023$, middle: $p = 0.0014$ and late: $p = 0.002$) (Table 17B, Figure 24). Analysis of the individual muscles showed that the injured professional footballers had an increased GM activation only in the late phase of SHF ($p = 0.0002$); and decreased AL activation in the early ($p = 0.013$) and middle ($p = 0.0021$) phases of SHF compared to healthy controls.

A: Summary measurements of professional footballers during standing hip flexion; describing the stance, injured leg with respe ct to the mean of the uninjured control group legs.

Movement phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)
Surface EMG									
GM:AL	0.06 (0.3)	2.09 (0.28)	<0.01*↑	-0.37(0.29)	2.1 (0.31)	<0.01*↑	0.32 (0.25)	2.32 (0.29)	<0.01*↑
Comments	Ratio difference due to a decrease of AL activation			Ratio difference due to a decrease of AL activation			Ratio difference due to a decrease of AL activation		
Kinematics									
Sagittal hip (Flex +)	2.5 (1.18)	6.08 (2)	0.17	-5.53 (0.85)	-4.96 (1.1)	0.68	-2.21 (0.87)	0.91 (1.52)	0.08**↑
Coronal hip (Add +)	0.56 (0.7)	-1.37 (1.06)	0.14	-3.42 (0.51)	-2.67 (1.1)	0.54	-2.250(0.69)	-5.9 (0.81)	0.024*↓
Horizontal hip (IR +)	-8.89 (1.97)	-9.17 (3.37)	0.93	-3.96 (0.66)	-2.93 (1.27)	0.41	-12.05 (2.07)	-11.78 (2.9)	0.94

B: Summary measurements of professional footballers during standing hip flexion; describing the moving, injured leg with respe ct to the mean of the uninjured control group legs.

Movement phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
Measured leg	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)
Surface EMG									
GM:AL	0.18 (0.29)	1.58 (034)	<0.01*↑	-0.82 (0.23)	0.2 (0.21)	<0.01*↑	-0.07 (0.21)	0.18(0.16)	<0.01*↑
Comments	Ratio difference due to a decrease of AL activation			Ratio difference due to a decrease of AL activation			Ratio difference due to an increase of GM activation		
Kinematics									
Sagittal hip (Flex +)	4.17 (1.43)	5.77 (1.77)	0.47	71.1 (1.34)	70.54 (2.39)	0.84	75.27 (1.27)	76.97 (1.86)	0.45
Coronal hip (Add +)	-0.07 (0.59)	-2.9 (1.02)	0.02*↓	1.41 (0.86)	2.59 (1.77)	0.55	0.72 (0.98)	-0.82 (1.47)	0.39
Horizontal hip (IR +)	-7.89 (1.43)	-6.95 (2.51)	0.75	7.17 (1.01)	5.7 (1.36)	0.39	-1.51 (1.17)	-3.64(2.45)	0.44

Table 17: Results from comparing surface electromyography and kinematic data between the injured leg of the injured professional foot ballers to the mean of both legs in the control professional footballers during standing hip flexion when the leg is weight bearing (stance) (A) and moving (B). Annotations: * = p < 0.05; ** = p < 0.1 (trend); sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in injured players; ↓ = decreased in injured players.

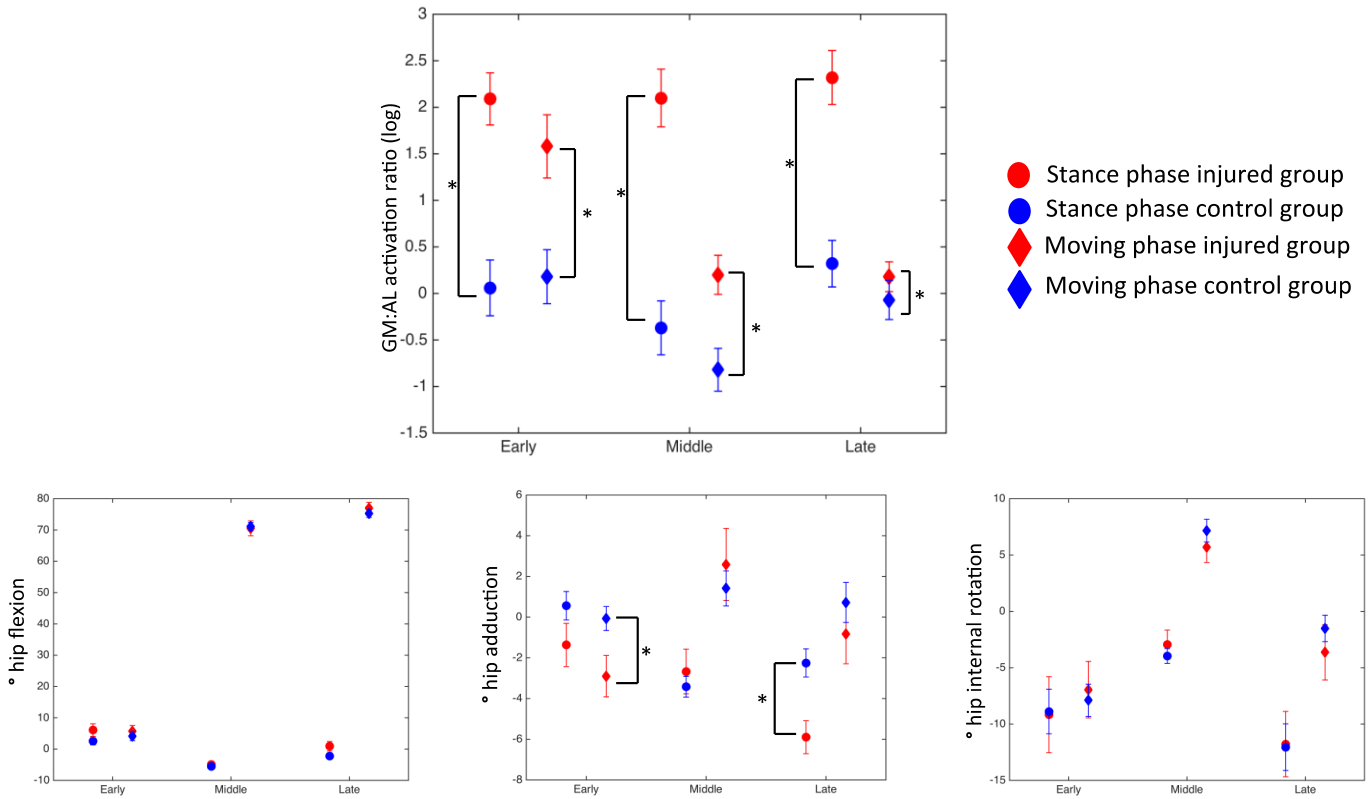


Figure 24: Graphical representation of the results comparing surface electromyography and kinematic data between the injured leg of the injured professional footballers to the mean of both legs in the control professional footballers during standing hip flexion when the leg is weight bearing (stance) (dots) and moving (diamonds). GM=gluteus medius; AL= adductor longus.

A: Summary measurements of amateur footballers during standing hip flexion; describing the stance, injured leg with respect to the mean of the uninjured control group legs.

Movement Phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
Surface EMG									
GM:AL	0.93 (0.12)	-0.22 (0.28)	<0.01*↓	0.64(0.11)	-0.57 (0.2)	<0.01*↓	1.2 (0.13)	0.03 (0.22)	<0.01*↓
Comments	Ratio difference due to a decrease of GM activation			Ratio difference due to a decrease of GM activation			Ratio difference due to a decrease of GM activation		
Kinematics									
Sagittal hip	5.97 (1.23)	7.21 (1.34)	0.06**↑	-5.63 (0.58)	-4.96 (2.97)	0.82	0.37 (1.15)	8.81 (4.05)	0.03*↑
Coronal hip (Add +)	2.32 (0.63)	0.42 (0.6)	0.08**↓	-5.07 (0.41)	-4.08 (0.79)	0.27	-2.82 (0.7)	-3.23 (0.73)	0.71
Horizontal hip (IR +)	-1.57 (1.28)	2.59 (1.93)	<0.01*↑	-4.03 (0.43)	-9.55 (4.3)	0.2	-5.41 (1.37)	2.59 (4.43)	<0.01*↑

B: Summary measurements of amateur footballers during standing hip flexion; describing the moving, injured leg with respect to the mean of the uninjured control group legs.

Movement Phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
Measured leg	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)
Surface EMG									
GM:AL	0.26 (0.11)	-0.47 (0.25)	<0.01*↓	-0.53 (0.09)	-0.74 (0.18)	0.29	-0.63 (0.9)	-0.67 (016)	0.85
Comments	Ratio difference due to a decrease of GM activation								
Kinematics									
Sagittal hip (Flex +)	9.29 (1.6)	10.81 (1.52)	0.49	56.38 (2.28)	48.12 (4.16)	0.08**↓	66.2 (1.7)	57.45 (4.46)	0.07**↓
Coronal hip (Add +)	-4.35 (0.66)	-3.59 (0.59)	0.39	2.37 (0.85)	3.23 (1.31)	0.58	-2.12 (0.83)	-0.47 (1.38)	0.3
Horizontal hip (IR +)	-4.29 (1.58)	3.08 (2.07)	<0.01*↑	8.42 (1.29)	5.24 (1.13)	0.07	3.8 (1.62)	7.97 (2.42)	0.16

Table 18: Results from comparing surface electromyography and kinematic data between the injured leg of the injured amateur footballers to the mean of both legs in the control amateur footballers during standing hip flexion when the leg is weight bearing (stance) (A) and moving (B). Annotations: * = p < 0.05; ** = p < 0.1 (trend); sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in injured players; ↓ = decreased in injured players.

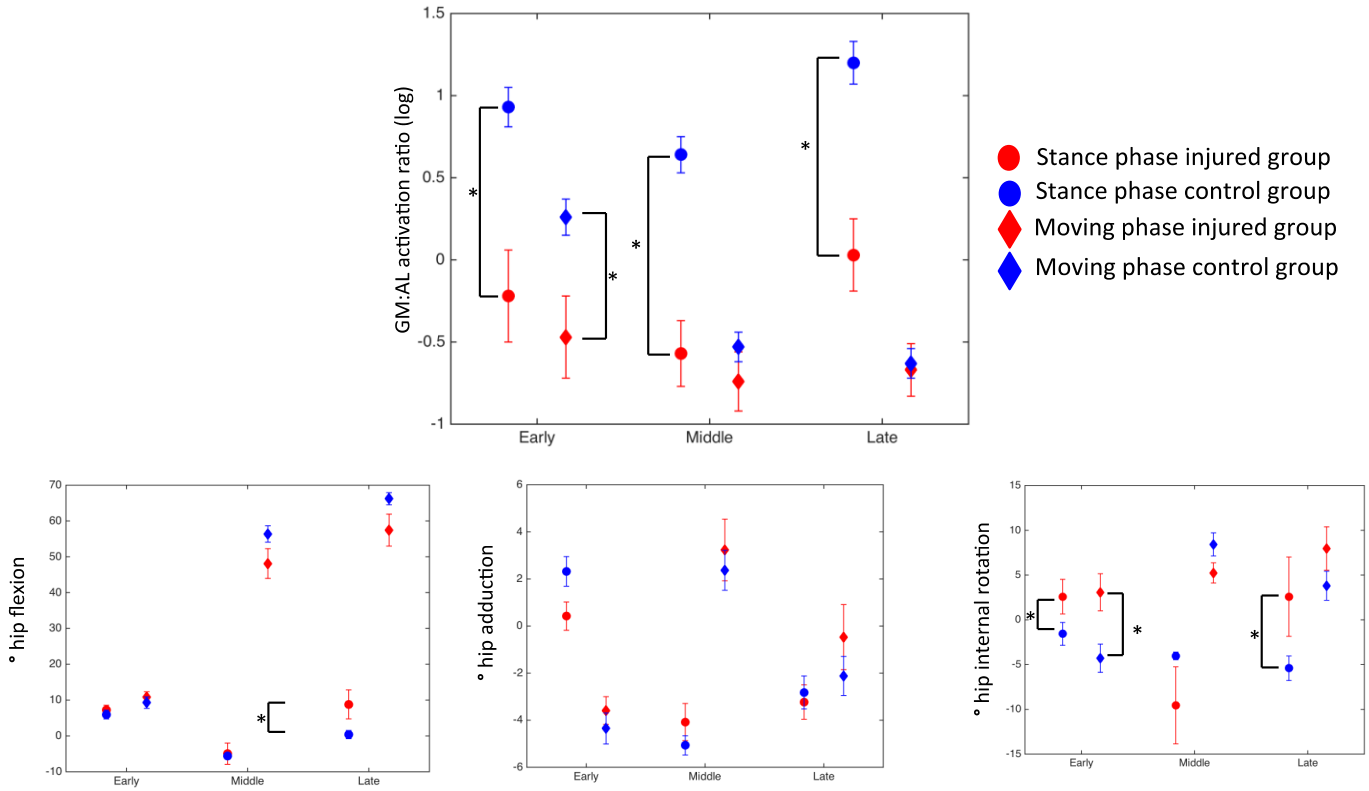


Figure 25: Graphical representation of the results comparing surface electromyography and kinematic data between the injured leg of the injured amateur footballers to the mean of both legs in the control amateur footballers during standing hip flexion when the leg is weight bearing (stance) (dots) and moving (diamonds). GM=gluteus medius; AL= adductor longus.

Amateur football players

While standing on the symptomatic leg, symptomatic amateur players had a significant decrease in GM:AL in all phases of the SHF task (early $p = 0.0000048$, middle: $p = 0.0000001$ and late: $p = 0.00000021$) compared to the control group (Table 18A, Figure 25). Analysis of the individual muscles showed a decreased activation of GM in all three phases of movement (early $p = 0.0000061$, middle: $p = 0.00000052$ and late: $p = 0.00000000042$); and increased activation of AL in the early ($p = 0.03$) and middle ($p = 0.043$) phases of SHF.

When moving the injured leg, these players showed a significantly decreased GM:AL compared to control group in the early phase of the movement ($p = 0.0078$) (Table 18B, Figure 25). Analysis of the individual muscles showed that injured players had a significant decrease in GM activation compared to controls in this phase of movement ($p = 0.0000092$).

Professional rugby players

When standing on the symptomatic leg, there was a significant increase of the GM:AL activation ratio in the middle phase of SHF in the injured players compared to healthy controls ($p = 0.0043$) (Table 19A, Figure 26). Further analysis did not show any significant differences between the symptomatic and asymptomatic rugby players when analysing separate muscles.

When measuring the moving leg, the injured players demonstrated an increase in GM:AL activation ratio in the early phase of SHF ($p = 0.011$) compared to controls (Table 19B, Figure 26) with analysis of the individual legs showing that AL demonstrated a decreased activation compared to healthy controls in the same phase of movement ($p = 0.014$).

A: Summary measurements of professional rugby players during standing hip flexion; describing the stance, injured leg with respect to the mean of the uninjured control group legs.

Movement Phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
Surface EMG									
GM:AL	1.01 (0.15)	1.34 (0.18)	0.17	0.94 (0.2)	1.65 (0.14)	<0.01*↑	1.82 (0.17)	2.09 (0.16)	0.26
Comments									
Kinematics									
Sagittal hip (Flex +)	23.17 (0.65)	18.96 (1.67)	<0.05*↓	-5.1 (0.97)	-7.77 (1.03)	0.07**↓	15.72 (1.29)	9.41 (2.57)	<0.05*↓
Coronal hip (Add +)	-1.56 (0.15)	-4.71 (0.73)	<0.05*↓	-7.45 (0.7)	-6.43 (0.63)	0.28	-9.88 (1.14)	-10.68 (0.43)	0.57
Horizontal hip (IR +)	-5.54 (1.85)	-17.82 (1.44)	<0.01*↓	-2.21 (0.64)	-5.54 (0.7)	<0.01*↓	-10.12 (1.85)	-23.74 (1.25)	<0.01*↓

B: Summary measurements of professional rugby players during standing hip flexion; describing the moving, injured leg with respect to the mean of the uninjured control group legs.

Movement Phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
Surface EMG									
GM:AL	0.4 (0.21)	1.13 (0.18)	<0.05*↑	-0.99 (0.15)	-1.11 (0.15)	0.59	-1.22 (0.11)	-1.2 (0.15)	0.92
Comments	Ratio increase due to a decrease of AL activation								
Kinematics									
Sagittal hip (Flex +)	20.29 (0.83)	18.2 (2.93)	0.25	63.3 (1.52)	70.78 (1.62)	<0.01*↑	82.53 (1.34)	86.98 (2.93)	0.18
Coronal hip (Add +)	-5.12 (0.75)	0.46 (01.2)	<0.01*↑	4.82 (1.98)	5.98 (1.49)	0.64	-0.38 (2.2)	8.62 (1.29)	<0.01*↑
Horizontal hip (IR +)	-12.25 (1.74)	-1.89 (2.02)	<0.01*↑	4.89 (1.51)	2.29 (2.09)	0.32	-5.73 (3.25)	-4.49 (4.3)	0.82

Table 19: Results from comparing surface electromyography and kinematic data between the injured leg of the injured professional rugby players to the mean of both legs in the control professional rugby players during standing hip flexion when the leg is weight bearing (stance) (A) and moving (B). Annotations: * = p < 0.05; ** = p < 0.1 (trend); sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in injured players; ↓ = decreased in injured players.

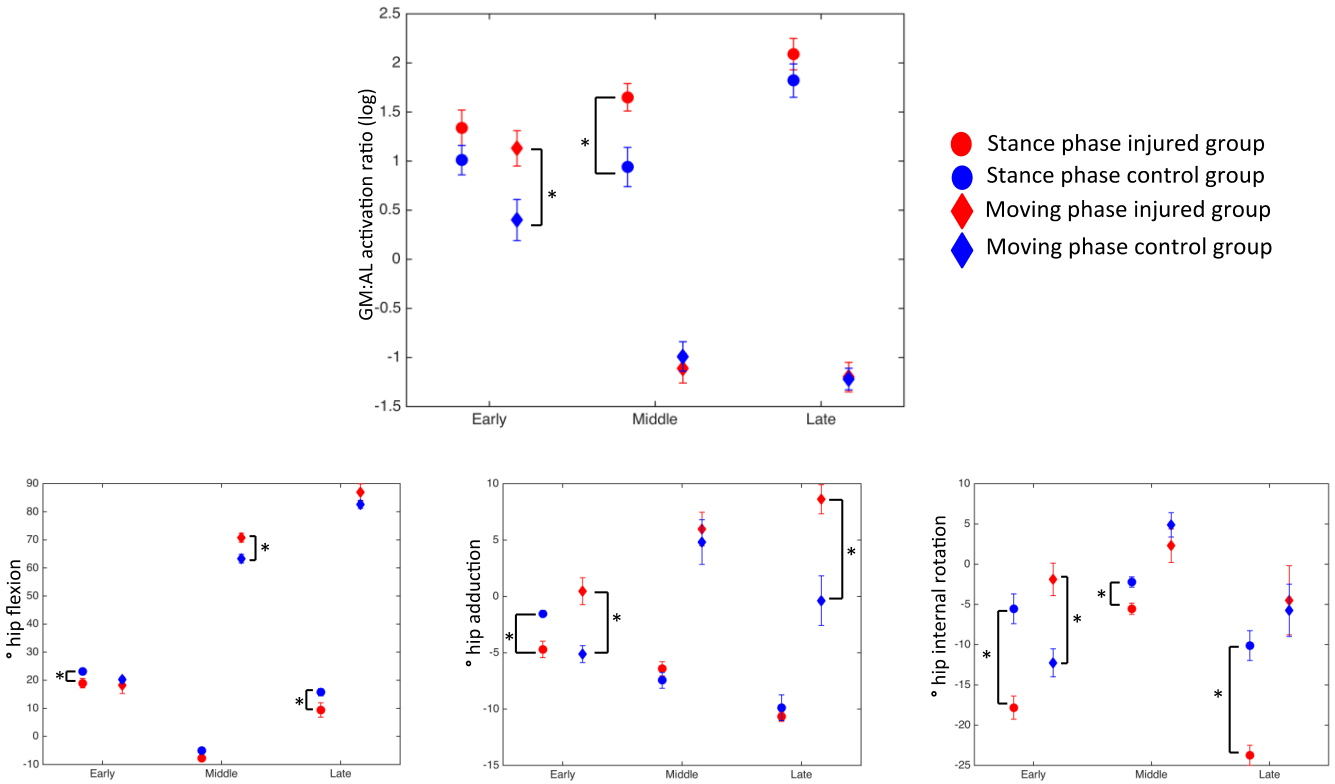


Figure 26: Graphical representation of the results comparing surface electromyography and kinematic data between the injured leg of the injured professional rugby players to the mean of both legs in the control professional rugby players during standing hip flexion when the leg is weight bearing (stance) (dots) and moving (diamonds). GM=gluteus medius; AL= adductor longus.

Ultimate Frisbee players

When measuring the SHF movement, there were no significant differences between injured and healthy Ultimate Frisbee players when the leg was moving or in stance (Table 20, Figure 27).

Field hockey players

When measuring the stance leg during the SHF, the injured field hockey players demonstrated a decreased GM:AL activation ratio in all three phases of movement (early: $p = 0.0064$, middle: $p = 0.017$, late: $p = 0.01$) compared to the healthy controls (Table 21A, Figure 28). The analysis of the individual muscles showed that the injured players had a significant decrease of GM activation in all three phases of SHF (early: $p = 0.00025$, middle: $p = 0.0014$, late: $p = 0.0021$) compared to the healthy controls.

When measuring the moving leg, the injured field hockey players showed a decreased GM:AL activation ration in all three phases of SHF (early: $p = 0.04$, middle: $p = 0.0024$, late: $p = 0.0061$) compared to the healthy controls (Table 21B, Figure 28). In the analysis of the individual muscles the injured players demonstrated a significantly decreased GM activation in all three phases of SHF (early: $p = 0.0021$, middle: $p = 0.0009$, late: $p = 0.0044$) and a significant decrease of the AL activation in the early phase of SHF ($p = 0.031$) compared to the healthy controls.

A: Summary measurements of Ultimate Frisbee players during standing hip flexion; describing the stance, injured leg with respect to the mean of the uninjured control group legs.

Movement Phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)
Surface EMG									
GM:AL	0.42 (0.23)	1.47 (0.6)	0.11	0.52 (0.25)	1.52 (0.5)	0.08**↑	1.56 (0.26)	2.57 (0.43)	0.05**↑
Comments									
Kinematics									
Sagittal hip (Flex +)	25.66 (0.79)	17.12 (3.64)	<0.05*↓	-4.4 (1.05)	-8.12 (1.45)	<0.05*↓	21.12 (1.33)	5.14 (4.35)	<0.01*↓
Coronal hip (Add +)	0.73 (0.89)	-4.44 (0.85)	<0.01*↓	-8.15 (1.61)	-6.71 (1.48)	0.52	-10.94 (1.51)	-10.87 (2.5)	0.98
Horizontal hip (IR +)	-2.05 (1.91)	-9.02 (2.54)	<0.05*↓	-3.37 (1.03)	-5.74 (1.18)	0.14	-5.55 (3.03)	-14.55 (4.13)	0.09**↓

B: Summary measurements of Ultimate Frisbee players during standing hip flexion; describing the moving, injured leg with respect to the mean of the uninjured control group legs.

Movement Phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)
Surface EMG									
GM:AL	0.61 (0.6)	0.57 (1.49)	0.36	-0.48 (0.28)	-1.25 (0.77)	0.35	0.05 (0.86)	-0.2 (1.57)	0.29
Comments									
Kinematics									
Sagittal hip (Flex +)	23.62 (0.8)	18.19 (3.6)	0.15	69.26 (1.93)	69.91 (2.08)	0.82	93.19 (2.19)	83.98 (4.9)	0.1
Coronal hip (Add +)	-1.82 (0.86)	2.01 (0.98)	<0.01*↑	0.56 (1.88)	-0.48 (2.79)	0.76	3.03 (1.63)	1.27 (4.05)	0.69
Horizontal hip (IR +)	-2.36 (1.98)	-3.85 (3.15)	0.69	3.65 (2.4)	-2.59 (2.58)	0.09**↓	0.95 (3.89)	-7.69 (3.14)	0.1

Table 20: Results from comparing surface electromyography and kinematic data between the injured leg of the injured Ultimate Frisbee players to the mean of both legs in the control Ultimate Frisbee players during standing hip flexion when the leg is weight bearing (stance) (A) and moving (B). Annotations: * = p < 0.05; ** = p < 0.1 (trend); sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in injured players; ↓ = decreased in injured players.

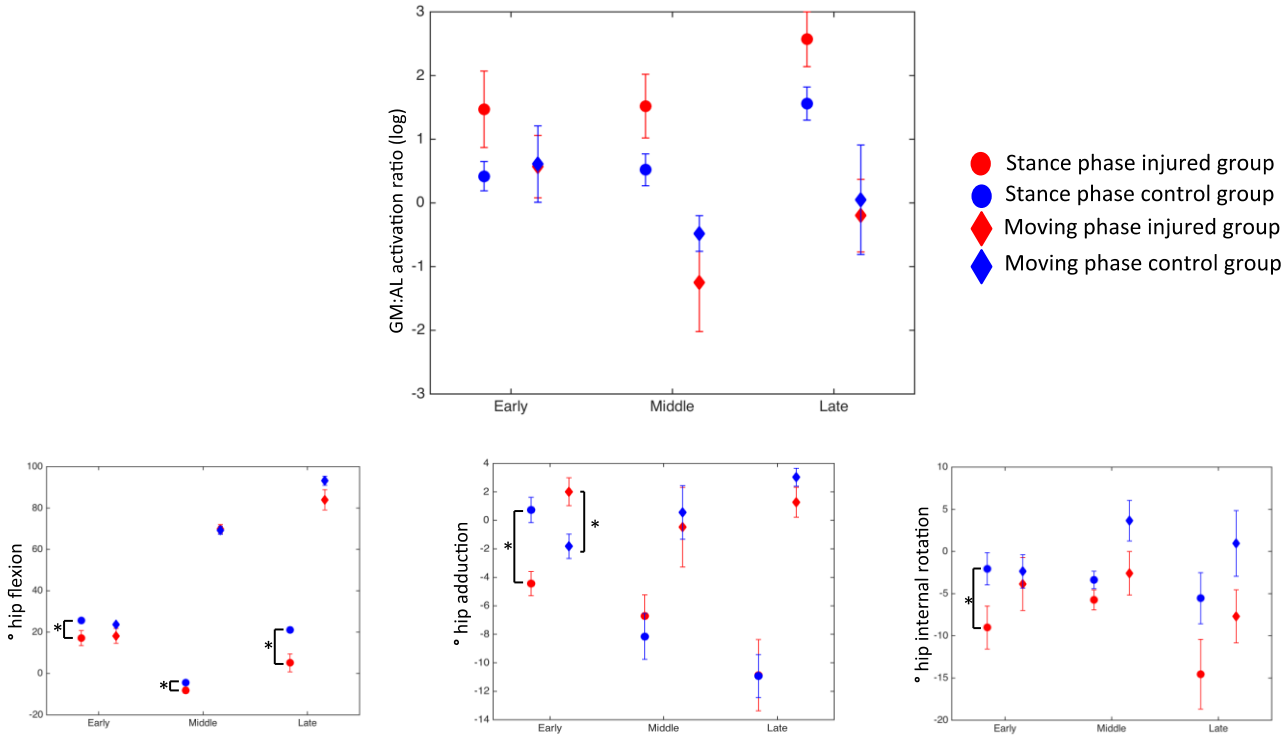


Figure 27: aphical representation of the results comparing surface electromyography and kinematic data between the injured leg of the injured Ultimate Frisbee players to the mean of both legs in the control Ultimate Frisbee players during standing hip flexion when the leg is weight bearing (stance) (dots) and moving (diamonds). GM=gluteus medius; AL= adductor longus.

A: Summary measurements of field hockey players during standing hip flexion; describing the stance, injured leg with respect to the mean of the uninjured control group legs.

Movement Phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)
Surface EMG									
GM:AL	1.76 (0.22)	-0.54 (0.78)	<0.01*↓	0.65 (0.23)	-1.6 (0.89)	<0.05*↓	1.91 (0.16)	-0.37 (0.84)	<0.01*↓
Comments	Significant decrease of GM activation			Significant decrease of GM activation			Significant decrease of GM activation		
Kinematics									
Sagittal hip (Flex +)	21.48 (1.24)	18.36 (1.66)	0.14	-8.54 (0.93)	-2.17 (8.5)	0.46	13.79 (1.86)	18.96 (8.54)	0.56
Coronal hip (Add +)	2.46 (0.84)	0.71 (0.57)	0.09**↓	-10.94 (1.07)	-9.29 (3.19)	0.63	-8.71 (1.17)	-9.16 (2.71)	0.88
Horizontal hip (IR +)	-4.37 (1.67)	-9.01 (1.99)	0.08**↓	-4.19 (0.82)	-5.96 (1.34)	0.27	-9.69 (1.54)	-15.77 (2.47)	<0.05*↓

B: Summary measurements of field hockey players during standing hip flexion; describing the moving, injured leg with respect to the mean of the uninjured control group legs.

Movement Phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)
Surface EMG									
GM:AL	0.82 (0.36)	-0.98 (0.77)	<0.05*↓	-0.18 (0.25)	-2.61 (-0.72)	<0.01*↓	0.47 (0.28)	-2 (0.81)	<0.01*↓
Comments	Significant decrease of GM activation			Significant decrease of GM activation			Significant decrease of GM activation		
Kinematics									
Sagittal hip (Flex +)	22.92 (1.44)	17. 97 (1.65)	<0.05*↓	61.36 (1.68)	57.62 (8.51)	0.67	85.41 (1.36)	78.85 (7.12)	0.37
Coronal hip (Add +)	-2 (0.84)	-1.33 (0.78)	0.56	2.45 (1.12)	-0.16 (3.41)	0.47	1.5 (1.48)	-1.12 (3.59)	0.5
Horizontal hip (IR +)	-8.85 (1.64)	-15.74 (1.2)	<0.01*↓	-1.18 (1.7)	4.97 (2.19)	<0.05*↑	-8.42 (2.59)	-9.08 (2.82)	0.86

Table 21: Results from comparing surface electromyography and kinematic data between the injured leg of the injured field hockey players to the mean of both legs in the control field hockey players during standing hip flexion when the leg is weight bearing (stance) (A) and moving (B). Annotations: * = p < 0.05; ** = p < 0.1 (trend); sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in injured players; ↓ = decreased in injured players.

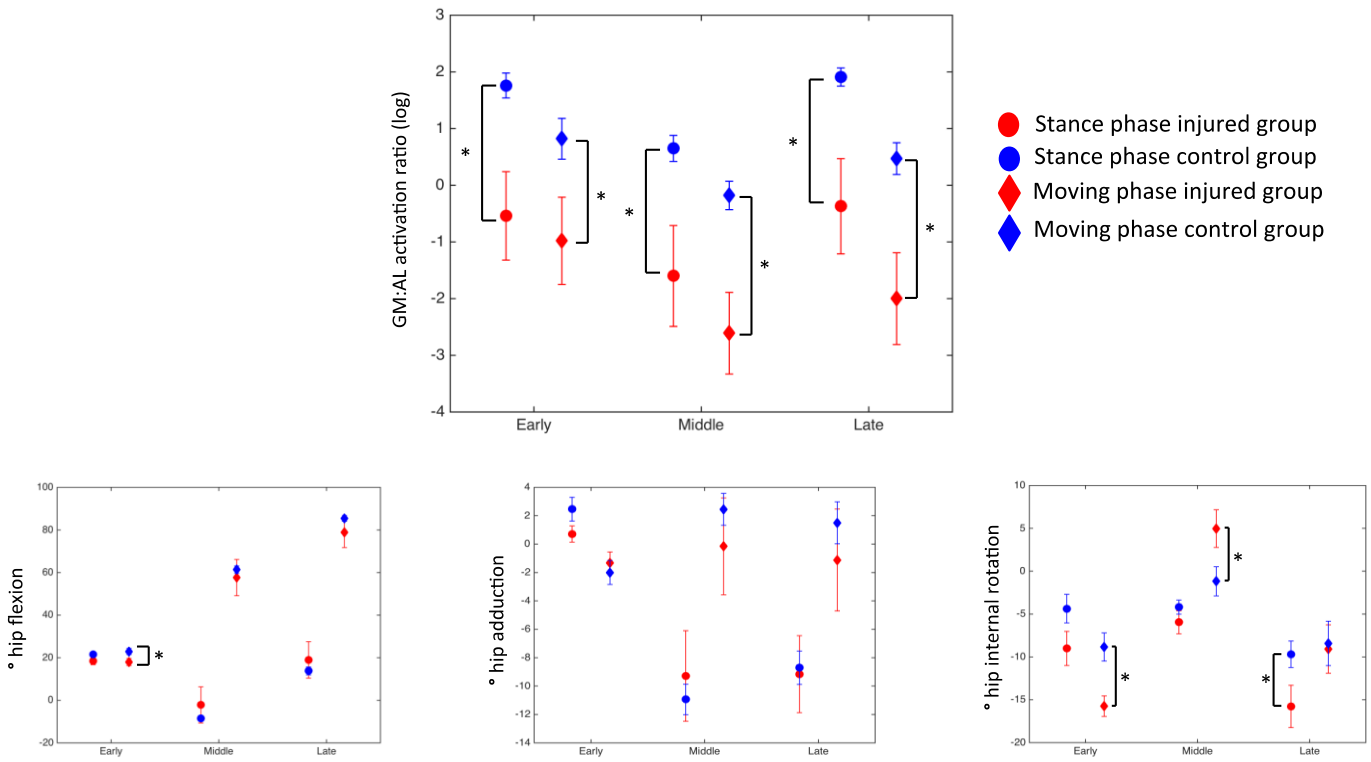


Figure 28: Graphical representation of the results comparing surface electromyography and kinematic data between the injured leg of the injured field hockey players to the mean of both legs in the control field hockey players during standing hip flexion when the leg is weight bearing (stance) (dots) and moving (diamonds). GM=gluteus medius; AL= adductor longus.

Kinematics

Professional football players

When measuring the stance leg during the SHF, the injured professional football players were more abducted in the late phase of SHF ($p = 0.024$) compared to the healthy controls (Table 17A, Figure 24).

When measuring the moving leg, the injured players were more abducted in the early phase of SHF ($p = 0.018$) compared to the controls (Table 17B, Figure 24).

Amateur football players

In the stance leg, the injured amateur football players were more flexed in the late phase ($p = 0.028$), and more internally rotated in the early ($p = 0.000035$) and late ($p = 0.00036$) phases of SHF compared to the healthy controls (Table 18A, Figure 25).

When measuring the moving leg, the injured players demonstrated an increased internal rotation in the early phase of SHF ($p = 0.0053$) compared to healthy controls (Table 18B, Figure 25).

Professional rugby players

When measuring the stance leg, the injured professional rugby players demonstrated a decreased hip flexion in the early ($p = 0.022$) and late ($p = 0.036$) phases of SHF; increased hip abduction in the early phase ($p = 0.022$) and increased hip external rotation in the early ($p = 0.00000019$) and late ($p = 0.0002$) phases compared to healthy controls (Table 19A, Figure 26).

When measuring the moving leg, the injured players demonstrated more flexion in the middle ($p = 0.031$) and late ($p = 0.027$) phases of SHF; increased hip abduction in the early ($p = 0.0016$) and late ($p = 0.000022$) phases of SHF; and increased hip internal rotation in the early ($p = 0.00009$) phase of SHF, compared to the healthy controls (Table 19B, Figure 26).

Ultimate Frisbee players

When measuring the stance leg the injured Ultimate Frisbee players demonstrated a decreased hip flexion in all three phases of SHF (early: $p = 0.026$, middle: $p = 0.048$, late: $p = 0.0017$); and increased abduction ($p = 0.0001$) and external rotation ($p = 0.033$) in the early phase of SHF, compared to healthy controls (Table 20A, Figure 27).

When measuring the moving leg, the injured players showed an increased hip adduction in the early phase of SHF ($p = 0.0047$) compared to the healthy controls (Table 20B, Figure 27).

Field hockey players

In the stance leg, the injured Field Hockey players demonstrated an increased hip external rotation in the late phase of SHF ($p = 0.044$) compared to the healthy controls (Table 21A, Figure 28).

When measuring the moving leg, the injured players showed less hip flexion in the early phase of SHF ($p = 0.028$), and increased hip external rotation in the early ($p = 0.0013$), but increased hip internal rotation in the middle ($p = 0033$) phase of SHF, compared to the healthy controls (Table 21B, Figure 28).

Single leg squat

sEMG

Professional football players

The symptomatic professional football players had a significantly increased GM:AL ratio in all seven phases of SLS (moving I: $p = 0.0001$, moving II: $p = 0.0000051$, moving III: $p = 0.0023$, moving IV: $p = 0.00065$, stance I: $p = 0.000021$, stance II: $p = 0.0068$, stance III: $p = 0.00019$), compared to the healthy controls (Table 22, Figure 29). Analysis of the individual muscles

demonstrated an increase in GM activation in the moving I ($p = 0.014$) and stance II ($p = 0.024$) phases of SLS; and a decrease in AL activation in all of the moving phases (moving I: $p = 0.0015$, moving II: $p = 0.000015$, moving III: $p = 0.0043$, moving IV: $p = 0.00014$) and stance III ($p = 0.0000019$) phase of SLS; in the injured players compared to the healthy controls.

	Phase 1: Moving I			
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	
sEMG GM:AL	0.014 (0.26)	1.774 (0.34)	<0.01*↑	
Comments	GM activation increase and AL decrease			
Sagittal hip	9.42(1.6)	6.68 (1.9)	0.27	NB Flex +
Coronal hip	3.76 (0.76)	-0.92 (1.04)	<0.01*↓	NB Add +
Horizontal hip	-8.43 (1.6)	-4.17 (2.65)	0.17	NB IR +

	Phase 2: Moving II			Phase 3: Stance I		
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
sEMG GM:AL	-0.34 (0.25)	1.9 (0.37)	<0.01*↑	-0.5 (0.24)	1.47 (0.36)	<0.01*↑
Comments	Ratio increase due to decrease of AL activation			Ratio increase due to decrease of AL activation		
Sagittal hip	16.32 (1.42)	8.67 (1.5)	<0.01*↓	25.41 (2.22)	16.14 (2.18)	<0.01*↓
Coronal hip	0.51 (0.42)	1.5 (0.74)	0.25	4.31 (0.86)	0.09 (0.96)	<0.01*↓
Horizontal hip	-1.73 (0.5)	-0.53 (0.66)	0.15	-10.17(1.58)	-5.01 (2.56)	0.09

	Phase 4: Moving III			Phase 5: Stance II		
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
sEMG GM:AL	-0.47 (0.2)	0.82 (0.36)	<0.01*↑	-0.26 (0.23)	0.9 (0.35)	<0.01*↑
Comments	Ratio increase due to decrease of AL activation			Ratio increase due to increase of GM activation		
Sagittal hip	42.81 (2.76)	41.61 (2.34)	0.74	67.55 (2.21)	57.49 (2.99)	<0.01*↓
Coronal hip	12.06 (0.75)	14.09 (1.4)	0.21	16.56 (1.11)	13.61 (2.08)	0.21
Horizontal hip	-0.9 (1.06)	1.4 (0.9)	0.09	-10.11 (1.75)	-5.31 (2.52)	0.12

	Phase 6: Moving IV			Phase 7: Stance III		
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
sEMG GM:AL	-0.31 (0.24)	1.08 (0.3)	<0.01*↑	-0.25 (0.34)	1.71 (0.37)	<0.01*↑
Comments	Ratio increase due to decrease of AL activation			Ratio increase due to decrease of AL activation		
Sagittal hip	-50.55 (2.17)	-44.66 (3.1)	0.13	16.95 (1.33)	13.43 (1.62)	0.98
Coronal hip	-15.05 (0.9)	-19.07 (1.36)	<0.05*↓	0.36 (0.85)	-4.84 (2.62)	0.11
Horizontal hip	1.01 (0.95)	-2.8 (0.88)	<0.01*↓	-9.63 (1.43)	-4.84 (2.62)	0.11

Table 22: Results from comparing surface electromyography and kinematic data between the injured leg of the injured professional footballers to the mean of both legs in the control professional footballers during single leg squat when the leg is moving. Annotations: * = $p < 0.05$; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in injured players; ↓ = decreased in injured players.

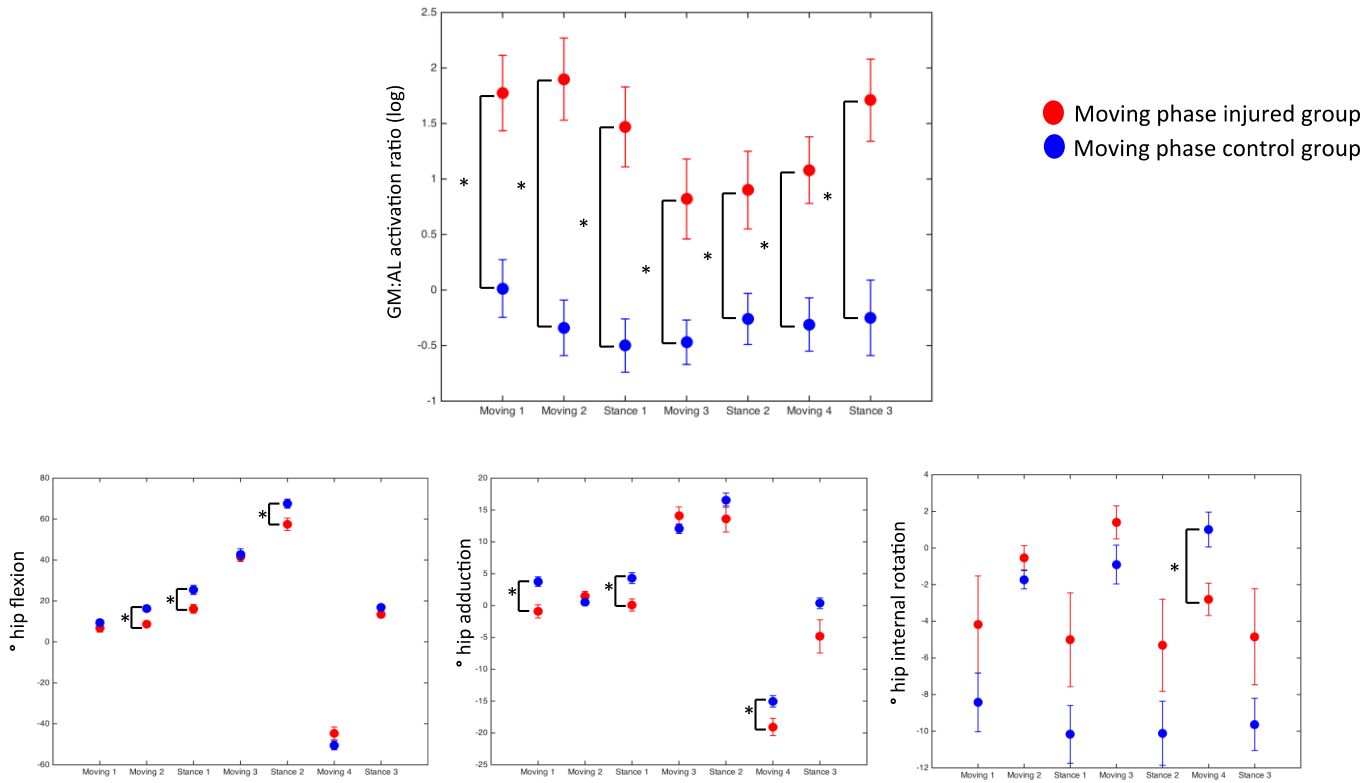


Figure 29: Graphical representation of the results comparing surface electromyography and kinematic data between the injured leg of the injured professional football players to the mean of both legs in the control professional football players during single leg squat when the leg is moving (dots). GM=gluteus medius; AL= adductor longus.

Amateur football players

The injured amateur football players demonstrated a decrease in GM:AL ratio in the moving III ($p = 0.00002$) and moving IV ($p = 0.00046$), as well as stance II ($p = 0.000015$) phases of SLS, compared to the healthy controls. The analysis of the individual muscles in these phases showed a decrease in GM activation in the moving III ($p = 0.0022$) and moving IV ($p = 0.0000028$) phases of SLS in the injured players compared to the healthy controls (Table 23, Figure 30).

Professional rugby players

The injured professional rugby players demonstrated an increase in GM:AL ratio only in stance II phase of SLS ($p = 0.017$) compared to the healthy controls (Table 24, Figure 31). The analysis of individual muscle activation showed a significant decrease of AL activation in the same phase ($p = 0.0003$).

		Phase: Moving I				
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**			
sEMG GM:AL	0.97 (0.16)	1.64 (0.4)	0.13			
Comments						
Sagittal hip	13.45(1.07)	16.78(2.9)	0.28	NB Flex +		
Coronal hip	3.19(1.07)	16.47(1.85)	0.02*↑	NB Add +		
Horizontal hip	-2.94(1.63)	-3.74(0.78)	0.66	NB IR +		
		Phase: Moving II			**Phase: Stance I**	
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
sEMG GM:AL	0.7(0.15)	0.73(0.23)	0.9	0.78(0.13)	0.96(0.15)	0.37
Comments						
Sagittal hip	26.47(1.95)	23.56(5.74)	0.63	40.08(2.13)	40.03(4.91)	0.99
Coronal hip	0.2(0.55)	1.85(2.64)	0.54	3.43(0.98)	5.28(2.4)	0.48
Horizontal hip	-2.41(0.61)	0.95(2.06)	0.12	-5.51(1.84)	-2.79(2.15)	0.34
		Phase: Moving III			**Phase: Stance II**	
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
sEMG GM:AL	0.76(0.1)	0.13(0.09)	<0.01*↓	0.76(0.11)	-0.35(0.21)	<0.01*↓
Comments						
Sagittal hip	24.54(1.84)	25.28(2.71)	0.82	64(1.71)	65(3.14)	0.78
Coronal hip	5.37(0.76)	6.33(1.69)	0.61	8.6(0.97)	11.6(3.26)	0.38
Horizontal hip	0.03(0.77)	2.54(1.52)	0.15	-5.27(1.67)	-0.25(1.19)	<0.05*↑
		Phase: Moving IV			**Phase: Stance III**	
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
sEMG GM:AL	0.92(0.1)	0.24(0.15)	<0.01*↓	0.82(0.17)	1.37(0.3)	0.12
Comments						
Sagittal hip	-48.48(2.4)	-47.25(4.27)	0.8	15.86(18.7)	1.41(0.38)	0.34
Coronal hip	-8.7(0.86)	-11.43(3.64)	0.47	-0.07(1.03)	0.17(0.84)	0.86
Horizontal hip	0.03(1.13)	-2.62(1.07)	0.09	-4.92(1.7)	-2.87(0.49)	0.25

Table 23: Results from comparing surface electromyography and kinematic data between the injured leg of the injured amateur footballers to the mean of both legs in the control amateur footballers during single leg squat when the leg is moving. Annotations: * = p < 0.05; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in injured players; ↓ = decreased in injured players.

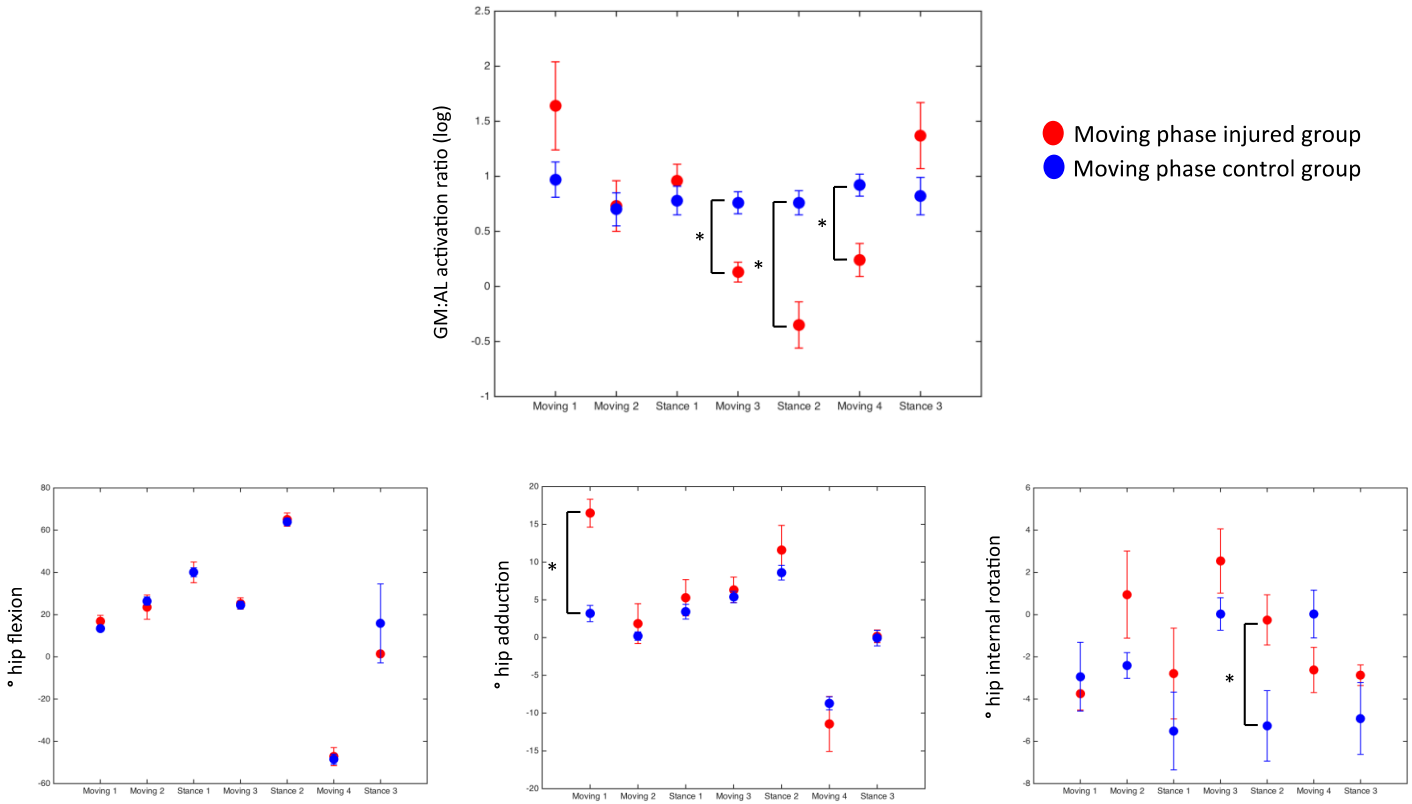


Figure 30: Graphical representation of the results comparing surface electromyography and kinematic data between the injured leg of the injured amateur football players to the mean of both legs in the control amateur football players during single leg squat when the leg is moving (dots). GM=gluteus medius; AL= adductor longus.

Ultimate Frisbee players

There were no significant differences in the GM:AL ratio between the injured and healthy Ultimate Frisbee players in any of the SLS movement phases (Table 25, Figure 32).

Field Hockey players

The injured Field Hockey players demonstrated a decrease in GM:AL ratio in all of the SLS movement phases (moving I: $p = 0.00015$, moving II: $p = 0.00036$, moving III: $p = 0.00022$, moving IV: $p = 0.001$, stance I: $p = 0.00044$, stance II: $p = 0.00011$, stance III: $p = 0.00026$) compared to healthy controls. The analysis of the individual muscles showed a decrease in GM activation in all of the SLS movement phases (moving I: $p = 0.00015$, moving II: $p = 0.000029$, moving III: $p = 0.000014$, moving IV: $p = 0.00012$, stance I: $p = 0.00005$, stance II: $p = 0.000033$, stance III: $p = 0.0000064$) in injured players compared to the controls (Table 26, Figure 33).

Phase 1: Moving I						
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**			
sEMG GM:AL	1.14(0.19)	0.99(0.24)	0.62			
Comments						
Sagittal hip	23.45(0.52)	22.94(1.75)	0.78	NB Flex +		
Coronal hip	-1.19(0.59)	3.68(0.93)	<0.01*↑	NB Add +		
Horizontal hip	-7.74(1.41)	1.35(3.04)	<0.01*↑	NB IR +		
Phase 2: Moving II				**Phase 3: Stance I**		
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
sEMG GM:AL	1.07(0.21)	0.94(0.3)	0.73	0.28(0.21)	0.9(0.26)	0.26
Comments						
Sagittal hip	1.63(0.57)	-0.57(0.79)	<0.05*↓	25.19(0.73)	23.87(2.38)	0.6
Coronal hip	-1.63(0.48)	0.39(0.58)	<0.01*↑	-2.86(0.82)	5.22(0.76)	<0.01*↑
Horizontal hip	-1.26(0.4)	-1.11(0.82)	0.88	-8.85(1.41)	1.51(2.78)	<0.01*↑
Phase 4: Moving III				**Phase 5: Stance II**		
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
sEMG GM:AL	0.25(0.16)	0.37(0.19)	0.65	0.18(0.18)	0.82(0.19)	<0.05*↑
Comments				Ratio increase due to a decrease of AL activation		
Sagittal hip	47.73(2.6)	42.32(5.09)	0.35	71.77(2.91)	67.27(5.18)	0.45
Coronal hip	14.52(1.79)	8.84(1.46)	<0.05*↓	12.16(2.68)	13.9(1.12)	0.56
Horizontal hip	0.3(2.44)	-2.58(1.92)	0.36	-0.53(2.57)	-3.14(1.52)	0.39
Phase 6: Moving IV				**Phase 7: Stance III**		
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
sEMG GM:AL	1.11(0.21)	1.39(0.17)	0.22	1.47(0.22)	1.45(0.23)	0.97
Comments						
Sagittal hip	-47.83(3.37)	-43.38(4.43)	0.43	25.71(0.94)	22.4(2.41)	0.21
Coronal hip	-13.08(1.96)	-7.99(1.48)	<0.05*↑	-3.87(1.17)	4.03(2.38)	<0.05*↑
Horizontal hip	-0.66(2.52)	1.87(2.16)	0.45	-8.25(1.44)	0.97(2.97)	<0.01*↑

Table 24: Results from comparing surface electromyography and kinematic data between the injured leg of the injured professional rugby players to the mean of both legs in the control professional rugby players during single leg squat when the leg is moving. Annotations: * = p < 0.05; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in injured players; ↓ = decreased in injured players.

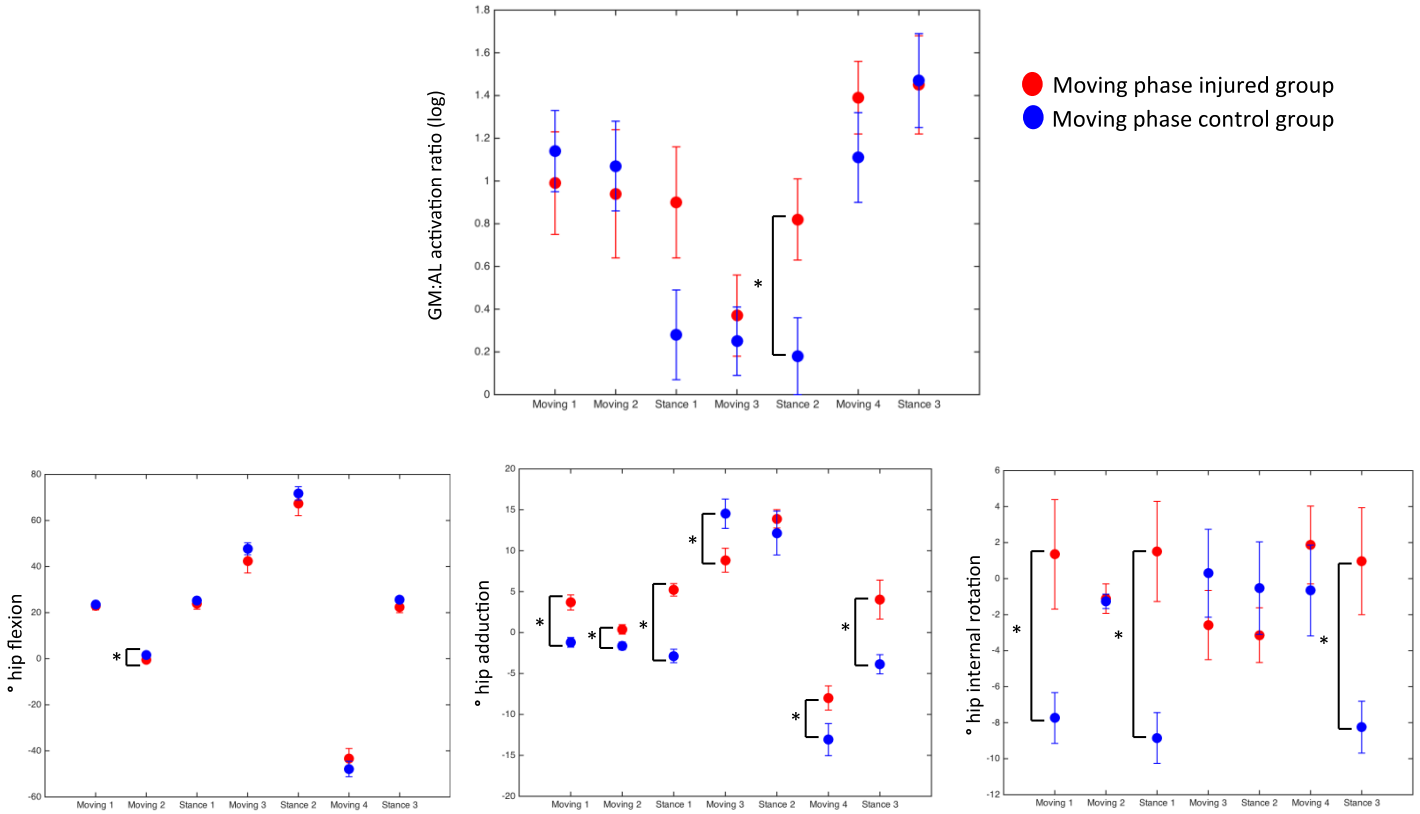


Figure 31: Graphical representation of the results comparing surface electromyography and kinematic data between the injured leg of the injured professional rugby players to the mean of both legs in the control professional rugby players during single leg squat when the leg is moving (dots). GM=gluteus medius; AL= adductor longus.

	Phase: Moving I					
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**			
sEMG GM:AL	1.26(0.28)	0.78(0.55)	0.44			
Comments						
Sagittal hip	28.48(1.04)	28.36(3.58)	0.97	NB Flex +		
Coronal hip	1.67(0.8)	5.16(0.74)	<0.01*↑	NB Add +		
Horizontal hip	-1.63(2.01)	1.99(2.31)	0.24	NB IR +		
	Phase: Moving II				**Phase: Stance I**	
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
sEMG GM:AL	1.46(0.33)	0.52(0.48)	0.11	1.65(0.31)	0.96(0.51)	0.26
Comments						
Sagittal hip	4.57(2.16)	3.14(1.13)	0.56	33.19(1.79)	33.33(3.92)	0.97
Coronal hip	-1.87(0.49)	-0.25(1.06)	0.17	0.07(0.92)	5.19(0.89)	<0.01*↑
Horizontal hip	1.14(0.86)	-0.9 (0.72)	0.07**↓	-0.81(2.12)	1.7(2.2)	0.42
	Phase: Moving III				**Phase: Stance II**	
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
sEMG GM:AL	0.43(0.24)	-0.11(0.54)	0.37	0.48(0.28)	0.21(0.6)	0.69
Comments						
Sagittal hip	40.61(2.78)	36.29(5.5)	0.49	71.22(2.86)	74.37(2.71)	0.43
Coronal hip	8.74(1.87)	14.96(1.8)	<0.05*↑	10.19(1.93)	19.01(1.87)	<0.01*↑
Horizontal hip	2.53(1.52)	0.75(1.58)	0.42	2.58(1.94)	4.14(2.34)	0.61
	Phase: Moving IV				**Phase: Stance III**	
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
sEMG GM:AL	1.07(0.21)	0.55(0.57)	0.39	1.23(0.29)	0.62(0.53)	0.32
Comments						
Sagittal hip	-0.35(2.79)	-44.17(2.68)	<0.05*↓	35.12(1.2)	29.04(2.75)	<0.05*↓
Coronal hip	-10.39(2.05)	-16.8(1.69)	<0.05*↓	-0.67(0.98)	1.7(1.33)	0.16
Horizontal hip	-1.42(1.2)	-2.39(1.26)	0.58	-1.33(2.08)	0.28(2.35)	0.61

Table 25: Results from comparing surface electromyography and kinematic data between the injured leg of the injured Ultimate Frisbee players to the mean of both legs in the control Ultimate Frisbee players during single leg squat when the leg is moving. Annotations: * = p < 0.05; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in injured players; ↓ = decreased in injured players.

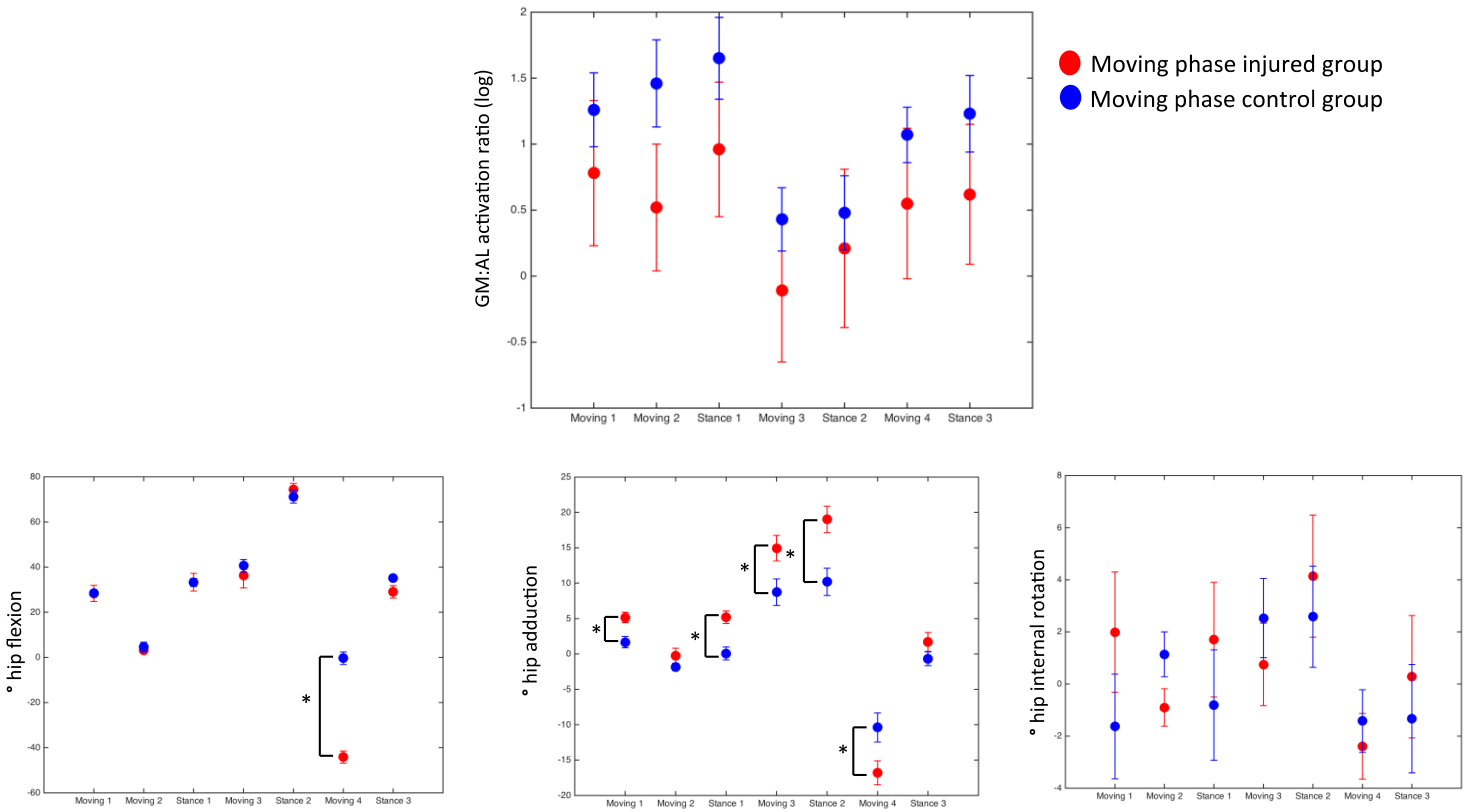


Figure 32: Graphical representation of the results comparing surface electromyography and kinematic data between the injured leg of the injured Untimate Frisbee players to the mean of both legs in the control Untimate Frisbee players during single leg squat when the leg is moving (dots). GM=gluteus medius; AL= adductor longus.

	Phase: Moving I					
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**			
sEMG GM:AL	2.76(0.52)	-1.8(0.9)	<0.01*↓			
Comments	Significant decrease of GM activation					
Sagittal hip	18.36(1.31)	18.46(1.4)	0.96	NB Flex +		
Coronal hip	1.14(0.78)	2.83(1.13)	0.23	NB Add +		
Horizontal hip	-3.78(3.09)	-12.09(0.87)	<0.05*↓	NB IR +		
	Phase: Moving II			**Phase: Stance I**		
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
sEMG GM:AL	2.16(0.37)	-2.03(0.97)	<0.01*↓	2.24(0.31)	-1.35(0.85)	<0.01*↓
Comments	Significant decrease of GM activation			Significant decrease of GM activation		
Sagittal hip	0.53(1.06)	0.93(0.77)	0.76	19.45(1.8)	19.39(0.99)	0.98
Coronal hip	-4.65(1.08)	-3.51(1.39)	0.52	-3.48(0.78)	-0.68(1.98)	0.2
Horizontal hip	-3.64(1.43)	-2.42(0.97)	0.49	-8.07(2.35)	-14.51(1.25)	<0.05*↓
	Phase: Moving III			**Phase: Stance II**		
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
sEMG GM:AL	1.07(0.28)	-2.86(0.89)	<0.01*↓	1.14(0.31)	-2.97(0.87)	<0.01*↓
Comments						
Sagittal hip	43.72(1.44)	57.76(4.34)	<0.01*↑	63.47(1.39)	77.18(5.62)	<0.05*↑
Coronal hip	12.81(1.91)	11.82(2.62)	0.76	8.19(1.59)	15.15(2.45)	<0.05*↑
Horizontal hip	6.38(1.43)	3.3(0.96)	0.09**↓	-0.21(2.9)	-10.6(2.36)	<0.05*↓
	Phase: Moving IV			**Phase: Stance III**		
Measured leg	**Uninjured**	**Injured**	**Statistic (p)**	**Uninjured**	**Injured**	**Statistic (p)**
sEMG GM:AL	1.67(0.24)	-2.38(1.08)	<0.01*↓	2.24(0.29)	-1.95(0.96)	<0.01*↓
Comments						
Sagittal hip	-40.04(0.95)	-62.48(6.08)	<0.01*↑	22.24(1.52)	17.67(2.25)	0.1
Coronal hip	-12.75(1.94)	-15.55(2.92)	0.43	-3.89(1.09)	-2.39(1.35)	0.39
Horizontal hip	-7.74(1.82)	-7.07(1.41)	0.77	-10.2(2.45)	-16.17(1.04)	<0.05*↑

Table 26: Results from comparing surface electromyography and kinematic data between the injured leg of the injured field hockey players to the mean of both legs in the control field hockey players during single leg squat when the leg is moving. Annotations: * = p < 0.05; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in injured players; ↓ = decreased in injured players.

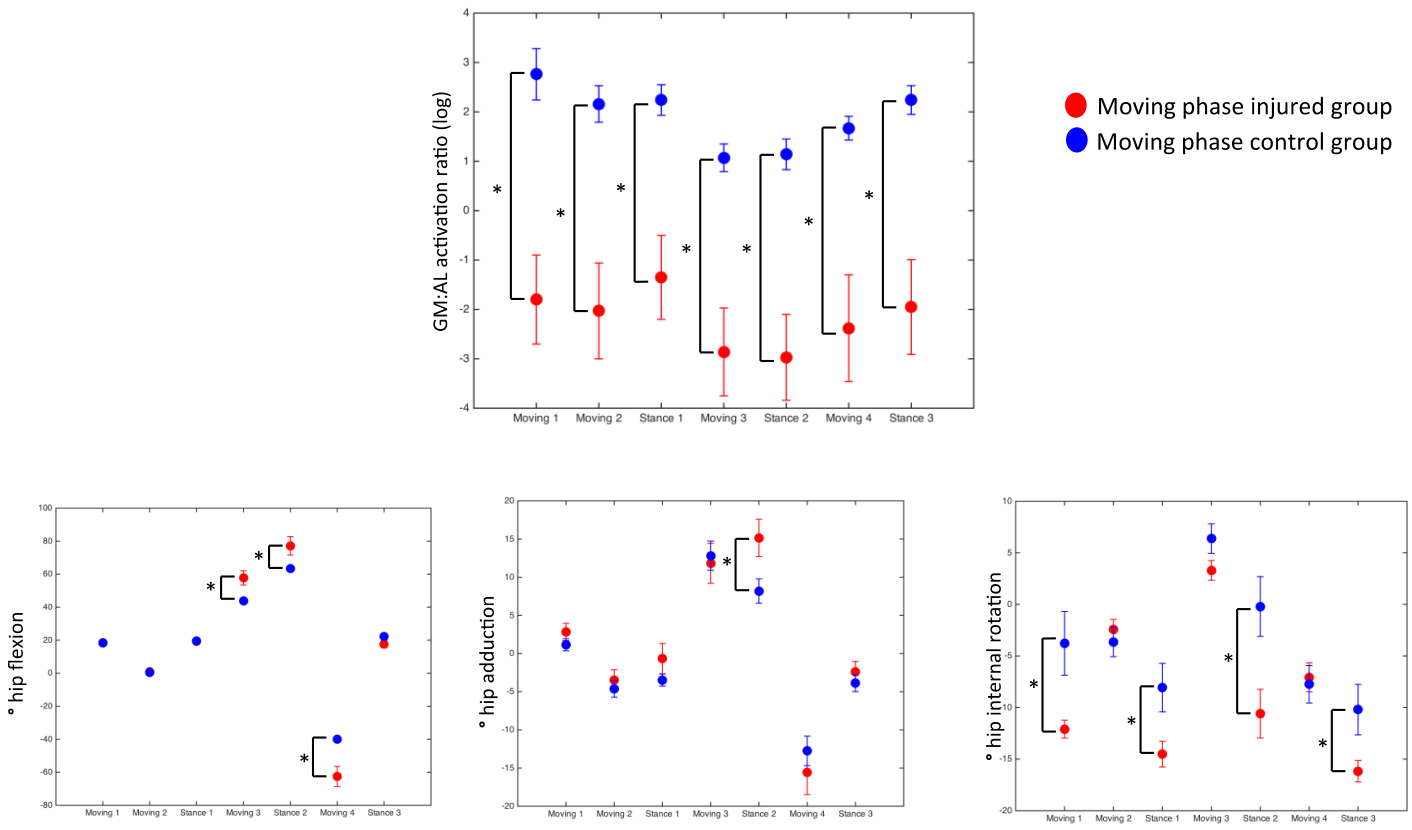


Figure 33: Graphical representation of the results comparing surface electromyography and kinematic data between the injured leg of the injured field hockey players to the mean of both legs in the control field hockey players during single leg squat when the leg is moving (dots). GM=gluteus medius; AL= adductor longus.

Kinematics

Professional football players

The injured professional football players demonstrated less hip flexion in the moving II ($p = 0.00042$), stance I ($p = 0.0041$) and stance II ($p = 0.0088$) phases of SLS; and more hip abduction in the moving I ($p = 0.00054$) and moving IV ($p = 0.017$), as well as stance I ($p = 0.0017$) and stance III ($p = 0.0019$) phases of SLS; compared to the healthy controls (Table 22, Figure 29).

Amateur football players

The only observed kinematic difference between the injured and healthy amateur football players was an increased hip internal rotation in the stance II phase of SLS in symptomatic athletes ($p = 0.017$) (Table 23, Figure 30).

Professional rugby players

The injured professional rugby players demonstrated increased hip adduction in moving I ($p = 0.000067$) and moving II ($p = 0.0011$), as well as stance I ($p = 0.0000000000000042$) and stance III (0.000000000036) phases of SLS; as well as significantly increased hip internal rotation in the moving I ($p = 0.00026$), moving III ($p = 0.00035$) and stance I ($p = 0.0000098$) and stance III ($p = 0.000000000015$) phases of SLS; compared to the healthy controls (Table 24, Figure 31).

Ultimate Frisbee players

The injured Ultimate Frisbee players demonstrated less hip flexion in the stance III phase of movement ($p = 0.048$); and increased hip joint adduction in the moving I ($p = 0.023$), moving III ($p = 0.021$), stance I ($p = 0.0002$) and stance II ($p = 0.002$), but increased abduction in the moving IV ($p = 0.02$) phases of SLS; compared to the healthy controls (Table 25, Figure 32).

Field Hockey players

The injured Field Hockey players demonstrated less hip flexion in the moving III ($p = 0.0058$) and moving IV ($p = 0.0013$), but more hip flexion in the stance II ($p = 0.027$) phases of SLS; more hip adduction in the stance II phase of the SLS ($p = 0.026$); and more hip external rotation in the movement I ($p = 0.015$) and all of the stance (stance I: $p = 0.023$, stance II: $p = 0.011$, stance III: $p = 0.033$) phases of SLS: compared to the healthy controls (Table 26, Figure 33).

Further analyses

Dominance data (sEMG and kinematics)

The analysis of potential dominance bias shows that there are some, but small differences between the dominant and non-dominant legs of the healthy controls in each subgroup; but these are smaller than the effects of the injury. All comparisons between the dominant and non-dominant legs of the healthy controls in each subgroup are enclosed in Appendix 2 (p. 264).

Overall professional vs. amateur footballers (sEMG) comparison at baseline and change from baseline

The mean of both legs of the healthy amateur footballers showed a significantly increased sEMG GM:AL ratio compared to the mean of both legs in healthy professional footballers in all stance phases, but not in the moving phases of SHF (Table 27), and in all phases of SLS (Table 28).

The difference between the injured and non-injured players, in professional and amateur subgroup, when comparing the mean of all of the phases in SHF, but moving and stance leg

separately, and mean of all phases of SLS; between the mean of both legs of the healthy controls and injured leg in the injured athletes is presented in Figure 34.

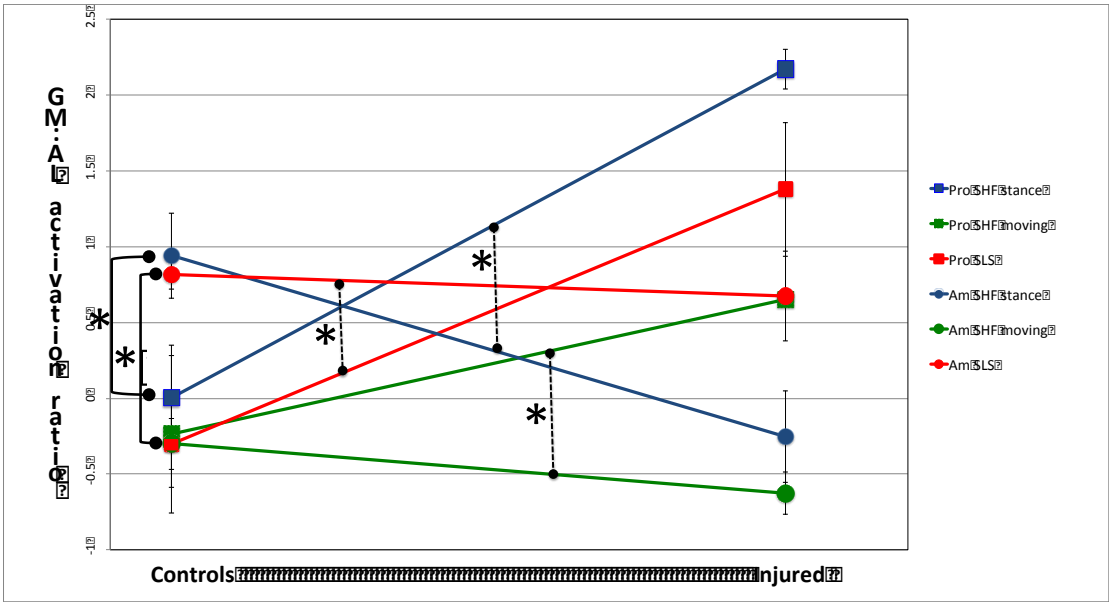


Figure 34: This graph shows the overall change in GM:AL activation ratio with data combined across movement phases in order to compare the professional and amateur footballers' similarity between control groups; alongside injured subjects' direction and degree of difference. Graph representing the mean of both legs in the healthy controls (Controls) and injured leg of the injured players (Injured), in all of the SHF movement phases collectively, but separately when the leg in stance and moving; and in all of the phases of SLS collectively; in the professional and amateur footballers. Pro – professional footballers; Am – amateur footballers; SHF – standing hip flexion movement; SLS – single leg squat movement. * represents significant difference between the control participants in the professional and amateur subgroups ($p < 0.01$).

A: Summary measurements of the control participants of professional vs amateur footballers (mean of both legs) during standing hip flexion; describing the stance legs.

Movement phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
	Professionals	Amateurs	Statistic (p)	Professionals	Amateurs	Statistic (p)	Professionals	Amateurs	Statistic (p)
Surface EMG									
GM:AL	0.06 (0.3)	0.93 (0.12)	<0.01*↑	-0.37(0.29)	0.64(0.11)	<0.01*↑	0.32 (0.25)	1.2 (0.13)	<0.01*↑
Comments									
Kinematics									
Sagittal hip (Flex +)	2.5 (1.18)	5.97 (1.23)	0.59	-5.53 (0.85)	-5.63 (0.58)	0.92	-2.21 (0.87)	0.37 (1.15)	0.8
Coronal hip (Add +)	0.56 (0.7)	2.32 (0.63)	0.06**↑	-3.42 (0.51)	-5.07 (0.41)	<0.05*↓	-2.250(0.69)	-2.82 (0.7)	0.56
Horizontal hip (IR +)	-8.89 (1.97)	-1.57 (1.28)	<0.01*↑	-3.96 (0.66)	-4.03 (0.43)	0.93	-12.05 (2.07)	-5.41 (1.37)	<0.01*↑

B: Summary measurements of the control participants of professional vs amateur footballers (mean of both legs during standing hip flexion; describing the moving legs.

Movement phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
Measured leg	**Professionals**	**Amateurs**	**Statistic (p)**	**Professionals**	**Amateurs**	**Statistic (p)**	**Professionals**	**Amateurs**	**Statistic (p)**
Surface EMG									
GM:AL	0.18 (0.29)	0.26 (0.11)	0.78	-0.82 (0.23)	-0.53 (0.09)	0.24	-0.07 (0.21)	-0.63 (0.9)	0.85
Comments									
Kinematics									
Sagittal hip (Flex +)	4.17 (1.43)	9.29 (1.6)	0.018	71.1 (1.34)	56.38 (2.28)	<0.01*↓	75.27 (1.27)	66.2 (1.7)	<0.01*↓
Coronal hip (Add +)	-0.07 (0.59)	-4.35 (0.66)	<0.01*↓	1.41 (0.86)	2.37 (0.85)	0.43	0.72 (0.98)	-2.12 (0.83)	<0.01*↓
Horizontal hip (IR +)	-7.89 (1.43)	-4.29 (1.58)	0.094**↑	7.17 (1.01)	8.42 (1.29)	0.45	-1.51 (1.17)	3.8 (1.62)	<0.01*↑

Table 27: Results from comparing surface electromyography and kinematic data between the mean of both legs of the healthy professional football players to the mean of both legs in the healthy amateur football players during standing hip flexion when the leg is weight bearing (stance) (A) and moving (B). Annotations: * = p < 0.05; ** = p < 0.1 (trend); sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in amateur players; ↓ = decreased in amateur players.

Phase 1: Moving I						
Measured leg	**Professionals**	**Amateurs**	**Statistic (p)**			
sEMG GM:AL	0.014 (0.26)	0.97 (0.16)	<0.01*↑			
Comments						
Sagittal hip	9.42(1.6)	13.45(1.07)	<0.05*↑	NB Flex +		
Coronal hip	3.76 (0.76)	3.19(1.07)	0.64	NB Add +		
Horizontal hip	-8.43 (1.6)	-2.94(1.63)	<0.05*↑	NB IR +		
	Phase 2: Moving II			**Phase 3: Stance I**		
Measured leg	**Professionals**	**Amateurs**	**Statistic (p)**	**Professionals**	**Amateurs**	**Statistic (p)**
sEMG GM:AL	-0.34 (0.25)	0.7(0.15)	<0.01*↑	-0.5 (0.24)	0.78(0.13)	<0.01*↑
Comments						
Sagittal hip	16.32 (1.42)	26.47(1.95)	<0.01*↑	25.41 (2.22)	40.08(2.13)	<0.01*↑
Coronal hip	0.51 (0.42)	0.2(0.55)	0.65	4.31 (0.86)	3.43(0.98)	0.5
Horizontal hip	-1.73 (0.5)	-2.41(0.61)	0.39	-10.17(1.58)	-5.51(1.84)	0.058
	Phase 4: Moving III			**Phase 5: Stance II**		
Measured leg	**Professionals**	**Amateurs**	**Statistic (p)**	**Professionals**	**Amateurs**	**Statistic (p)**
sEMG GM:AL	-0.47 (0.2)	0.76(0.1)	<0.01*↑	-0.26 (0.23)	0.76(0.11)	<0.01*↑
Comments						
Sagittal hip	42.81 (2.76)	24.54(1.84)	<0.01*↓	67.55 (2.21)	64(1.71)	0.25
Coronal hip	12.06 (0.75)	5.37(0.76)	<0.01*↓	16.56 (1.11)	8.6(0.97)	<0.01*↓
Horizontal hip	-0.9 (1.06)	0.03(0.77)	0.48	-10.11 (1.75)	-5.27(1.67)	0.048
	Phase 6: Moving IV			**Phase 7: Stance III**		
Measured leg	**Professionals**	**Amateurs**	**Statistic (p)**	**Professionals**	**Amateurs**	**Statistic (p)**
sEMG GM:AL	-0.31 (0.24)	0.92(0.1)	<0.01*↑	-0.25 (0.34)	0.82(0.17)	<0.01*↑
Comments						
Sagittal hip	-50.55 (2.17)	-48.48(2.4)	0.52	16.95 (1.33)	15.86(18.7)	0.63
Coronal hip	-15.05 (0.9)	-8.7(0.86)	<0.01*↑	0.36 (0.85)	-0.07(1.03)	0.75
Horizontal hip	1.01 (0.95)	0.03(1.13)	0.51	-9.63 (1.43)	-4.92(1.7)	<0.05*↑

Table 28: Results from comparing surface electromyography and kinematic data between the mean of both legs of the healthy professional football players to the mean of both legs in the healthy amateur football players during single leg squat when the leg is moving. Annotations: * = p < 0.05; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in amateur players; ↓ = decreased in amateur players.

Discussion

The aim of this study was to provide insight into pre- and re-habilitation strategies among multidirection athletes by exploring the muscle activation and kinematic patterns associated with SRGP. This was achieved by investigating relevant biomechanical patterns in injured athletes participating in a variety of amateur and professional sports during a standing hip flexion (SHF) and single leg squat (SLS) tasks.

The precursor question that needs to be answered is whether there are activation and movement pattern differences at all? This question was clearly answered as a range of significant biomechanical imbalances in the pelvic girdle were found in nearly all of the groups of athletes with SRGP, compared with well-matched controls, when performing movements challenging for the pelvis and hip areas. Typically, movement pattern differences were matched with relative muscle activation differences. Specifically, there was a marked difference in the GM:AL muscle activation ratio as well as significantly altered hip joint kinematics in coronal, sagittal and horizontal planes in both movement manoeuvres. There were clear differences between sports, and between participation levels within sports.

The professional footballers suffering from SRGP showed a consistent pattern of increased GM:AL ratio as a result of the increased GM activation and/or decreased AL activation in both movement manoeuvres, compared to healthy controls. This pattern was also observed in the professional rugby players, although less consistently.

In the professional footballer subgroup, the importance of gluteal activation and strengthening in the injury prevention strategies is recognised among healthcare professionals (Stolen et al., 2005, Lehnhard et al., 1996, Crow et al., 2012, Smith et al., 2014). Optimally strong and functional gluteal musculature in this cohort may be better suited to cope with any additional (over)loads (Caia et al., 2013, Lago-Penas et al., 2014, Stolen et al., 2005), possibly resulting in

the absence of observed activation deficiencies in GM. Moreover, increased loads on the groin area may result in the pain, dysfunction and potential inhibition of the AL as reported in rats (Ohira et al., 2011). Adductor muscle strengthening prevention programs implemented in the professional footballers were reported to reduce the incidence of SRGP in this cohort (Holmich et al., 2010). Thus, despite a commonly recognised dysfunction of the hip adductor muscles before and after the onset of SRGP (Crow et al., 2010, Emery and Meeuwisse, 2001, Engebretsen et al., 2010, Nevin and Delahunt, 2013), the decrease of AL activation in the professionals with SRGP suggests that more adductor-focused training should be implemented in the prevention and rehabilitation programmes in professional football. A study by Serner et al. (Serner et al., 2013) measured the activation of adductor musculature in six adductor exercises with graduated difficulty, which provides a good resource for the clinicians and should be considered in SRGP prevention and treatment programmes.

In rugby players, the pattern of hip joint movement in the presence of little muscle activation imbalances may indicate that the injured players are still able to fully activate their muscles on the optimal, 'healthy' level. However, in order to achieve that, they may be choosing various kinematic strategies – hence the non-uniform differences between injured and uninjured players in the hip kinematics. A lack of adequate 'kicking-specific' training in rugby has been recognised (Quarrie and Hopkins, 2015), and their focus on 'stability' over 'mobility' in game environment may indicate that open-chain manoeuvres (such as SHF) are relatively untrained and difficult to stabilise.

A similar trend of the GM:AL ratio decrease in injured players of both 'amateur' subgroups (amateur footballers and field hockey players) was a result of the GM activation decrease, with AL activation not being significantly altered. The sEMG pattern of change is opposite to this observed in the professional footballers and there may be a few explanations for this finding.

Firstly, the access to healthcare professionals' services may have biased the findings. Gluteal muscle hypertrophy as compared to the general population has been reported among professional footballers (Sanchis-Moysi et al., 2011) and their strong, well-trained and highly functional GM may be less likely to lose its properties as a response to an overload or increased movement demand. In the 'amateur' subgroup the decrease of GM activation affecting the GM:AL ratio may be the result of this muscle being sub-functional prior to the onset of symptoms, and the increased demands and/or overload may trigger a further loss of function. Alternatively, the loss of GM function may be the reason for SRGP in the amateurs as the weakness of this muscle leads to increased load on the hip joint (Fetto et al., 2002, Presswood et al., 2008). Although the joint itself may not have been affected in this group (as the players testing positive in hip joint tests were excluded in this study), lack of optimal function of GM may have cause the initial hip and pelvic imbalance in the coronal plane (Homan et al., 2013) and led to SRGP.

Different physiological characteristics of professionals associated with regular high-intensity training may also explain the different mechanisms of coping with overload and/or injury between the 'amateur' and 'professional' groups. The professionals receive a financial reward for being a part of the team, therefore a majority of their time is spent on training and optimising their performance (Stolen et al., 2005); classically they don't have any other time consuming occupation. Amateurs, however, have full-time jobs and their training time is limited. The difference in physiology between two groups may mean that the amateurs are more sensitive to any overload or imbalance occurring in their pelvic area and that the muscles react quicker with a decrease of function, activation or strength in these players. In the professionals, pelvic girdle musculature may cope well with the initial overload, but further excessive loading may potentially lead to muscle inhibition, as recently reported in hamstring injuries (Fyfe et al., 2013). Alternatively, the adductor activation deficit may be an effect of

pain in the groin region, which doesn't affect other musculature in these players (such as gluteal muscles).

Interestingly, in both subgroups of athletes: 'professionals' (professional footballers) and 'amateurs' (amateur footballers and field hockey players) the hip joint movement pattern during SHF was consistent with the muscle activation and ratio changes. The 'professionals', with an increase of the GM:AL ratio being mainly the result of the decrease of AL activation, were more abducted in several SHF phases. The amateurs, presenting a decreased GM:AL ratio due to a decrease of GM activation, tended to show more internal rotation, which is often associated with a GM dysfunction (Dai et al., 2014, Homan et al., 2013, Powers, 2010, Lack et al., 2014).

The lack of clear kinematic differences in injured compared to healthy amateur footballers in SLS is a surprising finding. SLS as a clinical test is more demanding than SHF, although the demands on pelvic control were reported to be similar in both movements (Boudreau et al., 2009). It was therefore expected that the biomechanical imbalances (both muscle activation and kinematic) demonstrated during SHF will be also present in SLS, potentially even to a greater extent.

In the field hockey players the hip joint kinematic imbalances were present in all planes, in the horizontal plane presenting a consistent pattern of increased external rotation throughout the movement. These findings are rather surprising, as in the presence of clear GM dysfunction manifesting as a decrease of its activation, hip joint kinematics of the injured players was not altered in an expected way; instead, they demonstrated increased hip external rotation (Crossley et al., 2011, Grimaldi, 2011).

Kawalek and colleagues (Kawalek and Garsztka, 2013) performed the analysis of the muscle flexibility in the field hockey players and found a shortened iliopsoas muscle in 100% of tested

participants. Iliopsoas muscle is the main hip flexor (Andersson et al., 1995), and in this cohort I only identified increased hip flexion in the phase of holding the squat in the lowest position i.e. the maximal knee flexion. However, iliopsoas is also an external rotator of the hip in the flexed position (Rajendran, 1989), which may explain the pattern of external rotation observed in the injured hockey players. Moreover, SRGP as a result of the iliopsoas muscle dysfunction is one of the most commonly recognised diagnostic sub-groups according to the Doha agreement (Weir et al., 2015). In this study, participants were not diagnosed according to the primary driver of their pain, therefore it may be that the iliopsoas muscle was the main cause of SRGP in injured field hockey players, which may have biased the results. It is, however, worth noting that despite the unexpected kinematic patterns presented by the injured players, the coronal plane muscle activation imbalance was still present in those players and that their GM was clearly underactive.

Interestingly, the injured Ultimate Frisbee players, in the absence of the GM:AL imbalances, showed a consistently increased hip abduction when measuring the stance leg in SHF. As with the professional rugby players, they may still be able to activate their muscles on a 'healthy' level despite the injury, but the force output may be smaller, particularly in the adductor muscle – which leads to increased abduction. This consistency is observed in all three phases of SHF but only in the stance leg, with no imbalances when the leg is moving. This pattern may suggest similar mechanisms of injury in this group and the professional footballers, which may be associated with the tendency to injure their weight-bearing leg regardless of whether this leg is dominant or non-dominant. Clear kinematic imbalances in the injured Ultimate Frisbee players in the stance, but not the moving leg, may therefore indicate that the leg being an actual stabiliser during turning, twisting and cutting manoeuvres is the dominant leg, and the one most commonly injured. It, however, raises a question whether the definition of the leg dominance by the preference to kick a ball is appropriate in this cohort.

The differences of the results between the subgroups may also be caused by the different injury mechanisms. Amateur players, with a lack of easy access to healthcare and strength and conditioning professionals, may not recognise the importance of targeted strengthening of certain muscle groups to prevent injuries caused by the imbalances in the pelvic area (Grimaldi, 2011). These differences in pre-habilitation and prevention strategies between the professional and amateur players may influence the target of biomechanical changes associated with groin injuries (Meister et al., 2011, Zheng et al., 2008).

Moreover, different injury mechanisms may be to some extend demonstrated by the different tendency in dominant/non-dominant leg injury pattern discovered in this study. When analysing separate subgroups, in three out of five (amateur footballers, professional rugby players and ultimate Frisbee players) the injured athletes were symptomatic on their dominant side and only one subgroup of injured athletes (professional footballers) have injured their non-dominant side. The field hockey players didn't show any tendency in injuring the dominant/non-dominant leg, but a low number of injured participants might have influenced the results.

It was surprising that only professional footballers (and not professional rugby players) showed a different pattern from all other groups and injured mainly their non-dominant side. Among all of the sub-groups measured in this study, the professional footballers potentially perform the largest number of kicking movements in the training and game (Lees and Rahnama, 2013, Barfield, 1998). Thus, increased susceptibility to injure the weight-bearing limb in professional footballers may potentially indicate that these players' training is focused on the open chain movements, such as kicking but less on the weight-bearing and stability exercises (Stolen et al., 2005). This may lead to the professional footballers lacking in optimal control and stability in the weight-bearing limb, which then is more sensitive to any biomechanical imbalances and therefore prone to pain and injuries. Additionally, a high amount of the dynamic movements

during competition and training of professional footballers (such as high number of repetitive kicking) (Stolen et al., 2005) may increase the demands for the weight-bearing leg to provide stability and control for the whole body, which then lead to overload and injury (Terje et al., 2015). The results of this study present clear clinical implications when designing the prevention strategies in this group of athletes. Firstly, the focus on adductor muscles should be emphasised, based on the exercises involving a high adductor activation (Serner et al., 2013). Secondly, everyday training should implement more weight-bearing and stability activities additionally to highly repetitive kicking movements. For example, more emphasis on every kind of twisting, turning and pivoting manoeuvres, potentially with additional weight in order to increase the challenge, may be useful.

The tendency of injuring the dominant (kicking) leg in amateur footballers, professional rugby and Field hockey players may indicate that the mechanism of injury is similar in these three groups of athletes, but different to professional footballers. A similar biomechanical pattern in these groups shown in this study may strengthen this suggestion. It may be that the amateur footballers and professional rugby players overload their limb in the repetitive open kinetic chain movements (such as kicking), which is not the focus or priority of their training (le Gall et al., 2010, Padulo et al., 2013). The kicking movement itself is a very demanding task for the balance of the antagonist coronal plane hip and pelvis musculature with a marked eccentric phase of adductor muscles work when slowing down the limb after the kick (Barfield, 1998). In the absence of specific training to perform this movement in a safe and optimal way, the coronal plane hip and pelvis musculature may become overloaded causing injury and pain. In order to prevent SRGP in these groups of athletes, gluteal strengthening, particularly during open chain exercises may be advised. Additionally, kicking-specific training with an optimal pelvic and hip musculature balance may be useful in SRGP prevention.

A different mechanism of injury might have occurred in the Ultimate Frisbee players, who don't perform the kicking movement at all. The dominant leg in this group of players was also defined as the preferred kicking leg, which may have been irrelevant to the athletes who don't kick the ball in game and training environment. However, this way of defining leg dominance was reported to be valid for athletes in various sports disciplines (Jessica Velotta, 2011) and was performed in order to maintain the consistency of the inclusion criteria in this study. The tendency of injuring the dominant leg in the Ultimate Frisbee players may be related to the fact that in the absence of kicking movement in this sport, the athletes have the opportunity to choose the preferred limb to perform the most challenging movements, such as twisting, turning, cutting and pivoting (Reynolds and Halsmer, 2006) or that it takes a particular stabilisation role for the dominant arm to throw the Frisbee. This may mean that although their dominant leg is the one most commonly injured (as in the amateur footballers and professional rugby players), the mechanism of injury is actually similar to the professional footballers, being associated with the increased demands on the weight-bearing leg when performing highly challenging manoeuvres.

An additional analysis was performed to compare the healthy controls of the professional and amateur footballers (Table 27 and Table 28). These two groups were selected from all others as were most comparable, and level-specificity is likely to be the only different factor in these players. Interestingly, there is a significant difference between the healthy control professionals and amateur in GM:AL activation ratio, clearly limited to the weight-bearing (closed kinetic chain) situation, that is, only in the stance leg in SHF (Table 27), and SLS (Table 28). This finding suggests that the professional and amateur players are in fact different cohorts, and strengthens previously stated hypothesis of different injury mechanisms to the moving or weight-bearing leg. The level-specificity, potentially even more than sport-specificity, may be therefore a major overlooked factor in SRGP rehabilitation, as established

conservative management guidelines are generic and do not differentiate the level of play. Critically, the difference in coronal plane muscle activation between control groups is the opposite difference to that measured we see in injured groups (Figure 34).

It is worth noting that a separate analysis of the differences between the dominant and non-dominant leg of the healthy control participants was also performed in order to explore whether the inter-group differences may be confounded by differences between dominant and non-dominant legs. Although some significant effects of leg dominance were found in the healthy cohort, I decided not to include this data in the main analysis. Firstly, the potential effects of dominance were not affecting the results, and obscured the main analysis simply by quantity of results; and secondly, almost all of the study participants have injured their dominant leg, which makes the dominance analysis somewhat spurious. The method of analysing and establishing the dominance bias is presented in Table 29 on the professional and amateur footballers: in professionals, the GM:AL ratio is increased in the dominant compared to non-dominant leg in healthy controls. They have mainly injured their non-dominant leg, which means that the increase of the sEMG ratio in their injured leg is a true finding, not biased by the dominance data. In healthy amateurs, the GM:AL ratio in the dominant leg is increased when measuring the stance, but decreased when measuring the moving leg. This cohort has mainly injured their dominant leg, which means that the decrease of the sEMG ratio is a true finding in all cases, except the early phase of the moving leg, which may have been a dominance bias.

	SHF											
	Pro (injured leg: non-dominant)						Am (injured leg: dominant)					
	Stance			Moving			Stance			Moving		
	E	M	L	E	M	L	E	M	L	E	M	L
Dominant leg	↑	↑					↑	↑		↓	↓	↓
Injured leg	↑	↑	↑	↑	↑	↑	↓	↓	↓	↓		

Table 29: The example of the potential dominance bias analysis shown on the professional and amateur footballers. SHF – standing hip flexion movement; Pro – professional footballers; Am – amateur footballers; E – early phase; M – middle phase; L – late phase; highlight shows a potential dominance bias.

The results of all comparisons between the dominant and non-dominant legs for all participants groups during SHF and SLS are included in Appendix 2 (p. 264).

Limitations

Although the link between injuring the dominant or non-dominant leg within the subgroups is clear in this research, it should be treated with caution as the relatively low number of participants prevents such epidemiological conclusions. However, further research focusing on the mechanisms of SRGP is required in order to fully understand its aetiology and design optimal prevention strategies for every athlete.

In this study the participants were not specifically diagnosed as having adductor-, iliopsoas-, abdominal- or inguinal-related SRGP. Instead I have diagnosed them as suffering from sports-related groin pain, which included all of those sub-categories. Therefore it may be that in some groups of tested athletes a particular structure being a primary driver of pain was dominating, providing some bias to the results.

There are some commonly recognised limitations associated with surface electromyographic measurements, which also apply in this study. These include the misplacement of the electrodes on the skin; inadequate preparation of participants' skin; unusual location of the motor plates and innervation zones within an individual; presence of the sEMG signal artefacts

and other (Kamen and Gabriel, 2010a). Except for those limitations that could not be controlled, effort was made decrease the risk of sEMG bias, including extensive theoretical and practical training..

The injured professional rugby players, although they were closely matched with the controls, were not controlled regarding the position played. This may have biased the results given the very different player characteristics depending on position.

A relatively low number of participants in the field hockey subgroup may bias the results as the minimum sample size was estimated at seven.

Future research

The results of this study show that injured athletes have clear muscle activation and kinematic imbalances in the coronal plane. No comprehensive study of other biomechanical imbalances in neither other muscles nor other planes has been reported; therefore researchers in the area should investigate other muscle activation and kinematic signatures in athletic groups and sub-groups with SRGP.

Although there is a clear link between pain, muscle activation and movement patterns in SRGP, the causality of these associations is still not established. A prospective, longitudinal study measuring reported biomechanical characteristics before and after the pain onset, and potentially after the completed rehabilitation course, would help to understand the mechanism of SRGP and provide a powerful clinical tool for the SRGP prevention programmes.

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Conclusions

There are clear coronal muscle activation and kinematic differences between injured and healthy groups of professional footballers, amateur footballers, professional rugby and field hockey players. Minimal biomechanical imbalances were found in the injured Ultimate Frisbee players compared to the controls.

In the majority of groups, in both SHF and SLS tasks, the hip joint movement patterns in the injured players were consistent with the muscle activation differences; most strongly in the SHF task. The muscle activation and movement patterns are different in the professional footballers with SRGP are different from the other groups of injured athletes. Moreover, they tended to injure their non-dominant leg, opposite to the rest of the groups. This may suggest that the mechanism of the injury in this group of athletes is different from others and that the healthcare professionals providing services to those athletes should be particularly cautious regarding a careful assessment and rehabilitation in this cohort.

The cross sectional nature of my work has enabled clarity to emerge that there are, previously unidentified, sports and participation level specific movement patterns and muscle activation patterns and therefore a good case for revisiting rehabilitation recommendations.

Chapter 7: Longitudinal study

Chapter overview

This chapter summarises the electromyographic and kinematic results of the study on five amateur rugby players after an acute groin injury measured three times – immediately after injury, mid-rehabilitation phase and after recovery. It correlates the results of each measurement, and additionally compares them to the cohort of five healthy amateur rugby players.

Introduction

Sports related groin pain (SRGP) is a chronic and debilitating condition in professional and amateur athletes participating in sports requiring repetitive kicking, twisting and pivoting (Thorborg et al., 2010, Holmich et al., 2011, Serner et al., 2015). The aetiology of this condition is not clear, but the relation between the acute groin injury and chronic SRGP has been longrecognised (Renstrom and Peterson, 1980). Acute groin injury was reported to typically occur during quick acceleration and sudden direction changes (Estwanik et al., 1990) as well as powerful overstretch of the lower limb in the directions of abduction and external rotation (Merrfield and Cowan, 1973, Smodlaka, 1980). Although previous groin injury has been recognised as one of the main risk factors for subsequent pathology (Arnason, 2004, Hagglund et al., 2009, Engebretsen et al., 2010, Steffen et al., 2008), there is a paucity of research on acute groin injuries (Serner et al., 2015) which have been reported to account for 39% of all groin injuries (Holmich et al., 2014).

Instead, the majority of studies investigating the diagnoses, mechanisms and treatment for groin pathologies focus on the chronic condition – SRGP (Morrissey et al., 2012a, Malliaras et

al., 2009, Mens et al., 1999), or do not specify the inclusion/exclusion criteria clearly enough to reliably differentiate between acute and chronic groin pathologies.

The consensus regarding the necessity of rest and adequate, early conservative treatment in acute groin injuries has been established by both clinicians and researchers (Jansen et al., 2008, Machotka et al., 2009, Serner et al., 2015).

There have been a few attempts to design pre-habilitation and rehabilitation programmes in order to decrease the incidence of groin injuries or optimise recovery (Holmich et al., 2010, Weir et al., 2011b, Weir et al., 2009). These programmes have focussed mostly on strengthening and stability of certain muscle groups (mainly hip adductors and flexors, as well as abdominals) and some of the interventions have been reported to be more successful than others. However, no study proposes an intervention including movement pattern retraining as well as relative muscle activation balance in the pelvic girdle areas, which was reported to be impaired in athletes with SRGP (Morrissey et al., 2012a).

Despite such common views and growing understanding of the treatment requirements, chronic SRGP is still a major and common problem in amateur and professional sports (Weir et al., 2015, Delahunt et al., 2015).

A number of biomechanical signatures of the athletes with chronic SRGP were discovered (Morrissey et al., 2012a, Malliaras et al., 2009, Arnason et al., 2004, Cowan et al., 2004b, Crow et al., 2010, Engebretsen et al., 2010, Emery and Meeuwisse, 2001, Emery et al., 1999a, Jansen et al., 2010, Mens et al., 2006, Mohammad et al., 2014, Nevin and Delahunt, 2013), mostly by observational studies. However, no longitudinal measurements have studied which biomechanical deficiencies remain after acute groin injury, a major risk factor for subsequent SRGP. Moreover, little attention is given to the kinematics and movement patterns of injured athletes.

As is the case in other multi-structural pathologies, such as patella-femoral pain syndrome, lower back and shoulder pain (Roussel et al., 2009, Mottram et al., 2009, Roussel et al., 2013, Worsley et al., 2013), optimising the movement patterns may be a key to successful rehabilitation, and may represent a way forward in groin pain (Morrissey et al., 2012a).

The aim of this study was to recognise the biomechanical deficiencies as a consequence of the acute muscle injury, to discover potential imbalances remaining after acute groin injury and optimise rehabilitation programmes, repeated electromyographic and kinematic measurements were performed immediately after groin injury, and throughout the rehabilitation process. Performed measurements enabled description of the muscle activation and hip joint movement patterns during the course of rehabilitation until the athletes were recovered according to established clinical measurements (Holmich et al., 2004). Additionally, the measurement of injured participants during the first, second and third occasion were compared with the results obtained from the healthy, well-matched control participants. The alternative hypothesis was the results of the outcome measures would differ significantly between the injured and uninjured participants, both at the beginning and at the end of the rehabilitation process; and that there would be no difference in the electromyographic and hip joint kinematic measurements between the first and the last testing occasion in the symptomatic players.

Methods

Queen Mary University Ethics of Research Committee approval was obtained and participants signed informed consent. Amateur rugby players were recruited from local and university teams through friends, family and contact details found on the web. The healthy control participants attended the Human Performance Laboratory on only one occasion; the injured participants attended the Laboratory on three occasions: up to five days after injury, four

weeks after initial injury and between eight and twelve weeks after initial injury, when the participant was functionally asymptomatic (Holmich et al., 1999). During every visit, they underwent a clinical examination; firstly to screen the potential participants against the inclusion criteria during the first visit; and secondly to assess the clinical outcomes indicating participants' recovery or otherwise. The clinical examination was divided into two parts: palpation and specific diagnostic tests. Each test was scored by participants according to their pain levels from 0-10 on the visual analogue scale (VAS). The dominant and injured limbs were established and clinically assessed.

After standard skin preparation, surface electrodes and CodaMotion markers were placed on participants' lower limb and pelvis areas. Then the participants were asked to perform two movements: standing hip flexion (SHF) and single leg squat (SLS) manoeuvres, for three repetitions of each leg. The SHF and SLS manoeuvres were then divided into three and seven phases, respectively.

A repeated measures ANOVA and paired t-tests were performed on the results of the maximal VAS scores obtained in each testing occasions – collectively, as well as separately for palpation and testing sections.

The sEMG signal from the gluteus medius (GM) and adductor longus (AL) muscles was then filtered, rectified and smoothed, GM:AL ratio and its logarithmic scale was then calculated for each participant, during each occasion separately, using custom made MatLab programmes (version 2012a, The Mathworks, Natick, MA, USA). Regarding the kinematic data, the hip joint rotation values were calculated as a mean of three repetitions for each leg separately, in all three planes, during each testing occasion. In SHF the injured leg was analysed when it was both stance and moving, in SLS the injured leg was analysed only when it was stance (weight-bearing). The details of this study methodology can be found in Chapter 3: Methods (p. 85).

Statistical analysis

Repeated measures two-way ANOVA was performed, with the testing occasion and the movement phases as the independent factors, and the sEMG or hip joint rotation values in each plane as the dependent factors. Where the Mauchly's test for spheri city violated the assumption of sphericity, Greenhouse-Geisser correction was used and the p values of significance as well as the degrees of freedom were reported according to the correction. Additionally, one-way ANOVA was performed for each movement phase separately, with a testing occasion as the independent factor and the sEMG or hip joint rotation values in each plane as the dependent factors.

A three-way mixed-model ANOVA analysis was performed in order to compare the results obtained from the three testing occasions, from the injured athletes with the right leg of the uninjured athletes; with the testing occasion, the movement phases and the injury status (injured or control) as the independent factors, and the sEMG or hip joint rotation values as the dependent factors. Because the healthy controls were tested once only, the values obtained from one testing occasion were multiplied and treated as obtained during all three testing occasion. As this approach might have biased the results, I have additio nally performed separate t-tests between the results obtained from the healthy controls and results obtained from injured participants during each testing occasion separately.

I recognised that the statistical analysis needed to be regarded as tentative due to the low number of participants. However, I made a conscious decision to statistically analyse this study as a longitudinal case-control study rather than case series, for reasons further explained in discussion section of this chapter.

Results

Five injured male amateur rugby players were recruited to this study between January 2013 and May 2014, all of them completed three testing occasions. The characteristics of the participants are presented in Table 30.

The outcomes of the clinical examination VAS scores during each testing occasion are presented in Table 31.

	Participants' characteristics		
	Inj	**Con**	**p**
N	5	5	
Height	1.81	1.83	0.68
Weight	84.2	81.6	0.72
Age	21	21.8	0.54
Injured leg (Dom:Non)		5:0	

Table 30: Characteristics of study participants. Inj – injured players; Con – controls. Dom – dominant leg injured; Non – non-dominant leg injured.

Clinical examination	Palpation												Clinical tests																				
													Adductor tests															Iliopsoas tests					
	Add tendon			Add insertion to pubic bone			Pubic symphysis			Iliopsoas			Adduction against resistance			Squeeze test 0°			Squeeze test 45°			Squeeze test 90°			Passive hip abduction (stretch)			Active hip flexion against resistance			Passive hip extension (stretch)		
Testing occasion	**1**	**2**	**3**	**1**	**2**	**3**	**1**	**2**	**3**	**1**	**2**	**3**	**1**	**2**	**3**	**1**	**2**	**3**	**1**	**2**	**3**	**1**	**2**	**3**	**1**	**2**	**3**	**1**	**2**	**3**	**1**	**2**	**3**
Patient 1	0	0	0	3*	0**	0***	0	0	0	0	0	0	0	0	0	0	2	0	0	0	0	3	3	0	4	2	0	0	0	0	5*	4**	0***
Patient 2	3	4	0	5*	5**	2***	0	0	0	0	0	0	5	2	0	5	0	2***	0	0	0	6*	6**	0	5	0	1	3	0	0	0	0	0
Patient 3	0	0	0	0	1	0	0	0	0	4*	3**	0***	0	0	0	2	2	0	2	0	0	0	0	0	3	4**	0	0	2	5***	0	4	0
Patient 4	0	0	0	0	0	0	0*	1**	0***	0	0	0	4	0	0	0	0	0	0	0	0	4*	1**	0***	0	0	0	3	1	0	0	1	0
Patient 5	5*	0	0	0	0	0	4	1**	0***	1	0	0	0	0	0	5*	2**	0***	4	0	0	0	2	0	0	0	0	3	0	0	0	0	0

Table 31: The results of the clinical examination of all five participants during three testing occasions in visual analogue scale (VAS); Add – adductor muscle. * indicates the maximal VAS score during the first measurement in both palpation and clinical tests; ** indicates the maximal VAS score during the second measurement in both palpation and clinical tests; * indicates the maximal VAS score during the last measurements in both palpation and clinical tests; underlined number represents the maximal VAS score overall.**

Vas scores

Repeated measures ANOVA showed a significant effect of the testing occasion on the maximal VAS scores when measured collectively and measuring the palpation and clinical tests separately (Table 32).

Paired t-test showed a significant difference between the first and the last measurement, both when analysing the palpation and clinical tests collectively ($p = 0.019$) and separately (palpation: $p = 0.01$; clinical tests: $p = 0.036$) (Figure 35). The results of analysis between other occasions are presented in Table 33.

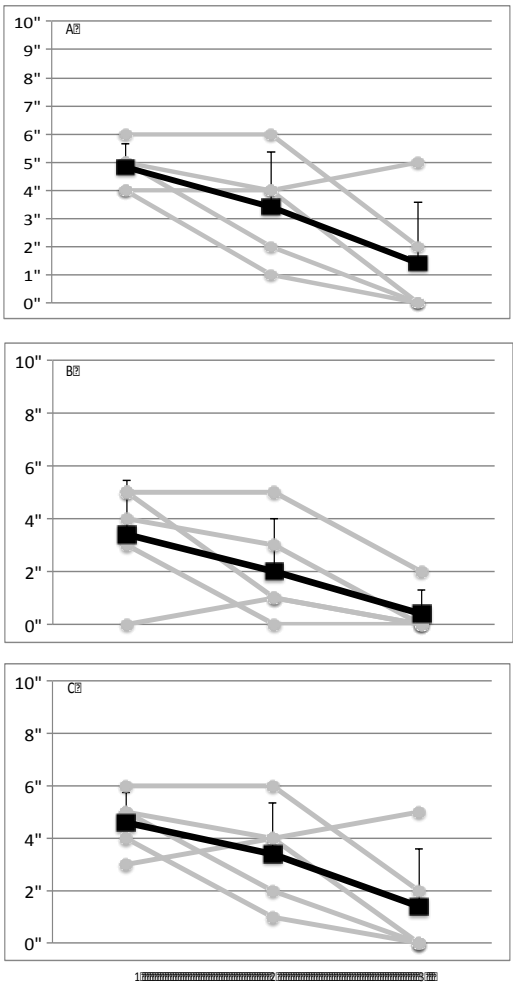

Figure 35: Graphic representation of the maximal visual analogue scale (VAS) scores obtained from each participant during each clinical examination as well as the means of the scores, analysed collectively (palpation and clinical tests) (graph A) as well as divided into palpation (graph B) and clinical test (graph C) separately. The X axis represents the first (1), second (2) and third (3) testing occasion, the Y axis shows the VAS scores. The grey lines represent each participant separately, the black line represents the mean of all participants, error bars represent the positive standard deviation.

ANOVA		Max pain all in VAS	Max pain palpation in VAS	Max pain clinical tests in VAS
Mauchly's test of sphericity	Significance	0.61	0.699	0.502
	Chi square	0.988	0.716	1.38
G-G?		No	No	No
F		6.687	7.042	4.78
df		2,8	2,8	2.8
p		0.02	0.017	0.043

Table 32: A summary of the results obtained from the repeated measures ANOVA comparisons with the testing occasion as the independent factor and the maximal VAS scores as the dependent factors in given comparison; G-G - Greenhouse-Geisser correction used; df – degrees of freedom; highlighted cells indicate the significant findings.

Paired t-test between occasions	Max pain collective in VAS (p value)	Max pain palpation in VAS (p value)	Max pain clinical tests in VAS (p value)
1/3	0.02	0.01	0.04
1/2	0.05	0.04	0.1
2/3	0.05	0.05	0.05

Table 33: The results of paired t-tests between the maximal scores obtained in visual analogue scale (VAS) during palpation and clinical tests analysed together (collective) and separately; 1/3 – comparing first and third occasion; 1/2 - comparing the first and second occasion; 2/3 -comparing the second and third occasion; Max pain – maximal obtained VAS scores. Highlighted cells indicate the significant findings.

sEMG results

SHF

No significant effect was found in both two-way repeated measures ANOVA, with a testing occasion and phase of movement as independent factors and the GM:AL activation ratio as the dependent factor; as well as analysing each phase separately by one-way ANOVA with the testing occasion as an independent factor and the GM:AL activation ratio as the dependent factor. The results of each comparison are presented in Table 34.

When comparing the injured participants with the healthy controls, no significant interaction was found in the three-way mixed ANOVA in SHF when the injured leg was in stance ($F = 0.185$, $p = 0.945$) or moving ($F = 0.91$, $p = 0.969$), as well as in the SLS ($F = 0.402$, $p = 0.95$) movement manoeuvre. No significant difference between the injured and uninjured participants was found when performing independent t-tests between two groups (Table 34).

Analysis combination		L SHF Two-way ANOVA	L SHF early	L SHF middle	L SHF late	R SHF Two-way ANOVA	R SHF early	R SHF middle	R SHF late	R SLS Two-way ANOVA	R SLS moving 1	R SLS moving 2	R SLS moving 3	R SLS moving 4	R SLS stance 1	R SLS stance 2	R SLS stance 3
sEMG																	
Maulchy's test of sphericity	Significance	0.22	0.7	0.84	0.35	0.262	0.51	0.86	0.88	-	0.76	0.64	0.52	0.99	0.95	0.98	0.77
	Chi square	0.67	0.71	0.34	2.13	12.854	1.34	0.3	0.25	-	0.54	0.89	1.3	0.02	0.11	0.04	0.54
G-G?		No	No	No	No	No	No	No	No	No	No	No	No	No	No	No	No
F		0.19	1.1	1.93	0.1	0.97	0.35	1.17	0.71	0.4	0.44	1.34	0.3	0.56	1.3	0.21	0.31
df		4, 16	2, 8	2, 8	2, 8	4, 16	2, 8	2, 8	2, 8	12, 48	2, 8	2, 8	2, 8	2, 8	2, 8	2, 8	2, 8
p		0.94	0.38	0.21	0.91	0.45	0.72	0.36	0.52	0.96	0.66	0.32	0.75	0.59	0.32	0.81	0.74
Kinematics																	
Coronal plane																	
Maulchy's test of sphericity	Significance	0.01	<0.01	0.01	0	0.1	0.1	0.17	0.72	-	0.48	0.12	0.18	0.88	0.03	0.39	0.88
	Chi square	24.57	16.32	8.93	14.92	4.66	17	3.51	0.65	-	1.47	4.33	3.43	0.25	6.87	1.89	0.25
G-G?		Yes ε=0.282	Yes ε=0.50	Yes ε=0.513	Yes ε=0.502	No	No	No	No	-	No	No	No	No	Yes ε=0.527	No	No
F		0.55	4.21	6.48	4.54	0.62	0.57	0.54	0.21	1.58	1.45	2.92	1.82	2.19	2.1	2.61	2.19
df		1.13, 4.52	1.002, 4.009	1.062, 4.105	1.003, 4.014	4, 16	4, 16	2, 8	2, 8	12,48	2, 8	2, 8	2, 8	2, 8	1.053, 4.213	2, 8	2, 8
p		0.71	0.11	0.62	0.1	0.51	0.69	0.6	0.82	0.13	0.29	0.11	0.22	0.17	0.22	0.13	0.17
Sagittal plane																	
Maulchy's test of sphericity	Significance	0.23	0.2	0.2	0.63	0.051	0.02	0.25	0.77	-	0.74	0.98	0.65	1	0.93	0.15	1
	Chi square	13.46	3.26	3.26	0.93	19.37	8.37	2.78	0.53	-	0.6	0.04	0.88	0.01	0.14	3.79	0.01
G-G?		No	No	No	No	No	Yes ε=0.516	No	No	No	No	No	No	No	No	No	No
F		0.7	0.83	1.02	0.99	0.68	0.39	0.75	0.95	0.73	4.27	3.29	1.45	1.25	1.75	0.5	1.25
df		4, 16	2, 8	2, 8	2, 8	4, 16	1.032, 4.127	2, 8	2, 8	12, 48	2, 8	2, 8	2, 8	2, 8	2, 8	2, 8	2, 8
p		0.61	0.47	0.4	0.41	0.62	0.57	0.93	0.43	0.71	0.55	0.09	0.29	0.34	0.24	0.63	0.34
Horizontal plane																	
Maulchy's test of sphericity	Significance	<0.01	0.35	0.52	0.92	<0.05	0.09	0.14	-	<0.01	0.6	0.44	0.08	0.71	0.52	0.05	0.63
	Chi square	26.42	2.09	1.31	0.18	22.17	4.82	3.99	-	19.85	1.02	1.64	4.97	0.69	1.3	6.18	0.92

G-G?	Yes ε=0.363	No	No	No	Yes ε=0.322	No	No	No	No	No	No	No	No	No	Yes ε=0.534	No
F	2.11	0.11	0.3	0.49	0.3	0.08	0.56	0.02	0.38	0.28	0.47	0.14	0.09	0.26	0.07	0.15
df	1.45, 5.81	2, 8	2, 8	2, 8	1.29, 5.15	2, 8	2, 8	2, 8	12, 48	2, 8	2, 8	2, 8	2, 8	2, 8	1.068, 4.272	2, 8
p	0.2	0.89	0.75	0.63	0.67	0.93	0.95	0.99	0.96	0.76	0.64	0.87	0.92	0.78	0.82	0.87

Table 34: A summary of the results obtained from the repeated measures ANOVA comparisons of the injured players, with the testing occasion as the independent factor and the gluteus medius to adductor longus muscle activation ratio (GM:AL) and the hip joint rotation in three planes as the dependent factors in given comparison; G-G - Greenhouse-Geisser correction used; df – degrees of freedom; L SHF – left standing hip flexion movement (injured leg stance); R SHF – right standing hip flexion movement (injured leg moving); R SLS – right single leg squat movement (injured leg stance); X – hip join rotation in coronal plane; Y – hip joint rotation in sagittal plane; Z – hip joint rotation in horizontal plane; ε – level of Greenhouse-Geisser correction. Highlighted cells indicate the significant findings (no significant findings found).

Kinematics

No significant effect was found in both two-way repeated measures ANOVA, with a testing occasion and phase of movement as independent factors and the hip joint kinematics in each plane as the dependent factor; as well as analysing each phase separately by one-way ANOVA with the testing occasion as an independent factor and the hip joint kinematics in each plane as the dependent factor (Table 34).

When comparing the injured participant with the uninjured controls, no significant interaction was found in a three-way mixed model ANOVA in SHF when the injured leg was stance in any plane (coronal: $F = 0.546$, $p = 0.703$; sagittal: $F = 0.697$, $p = 599$; horizontal: $F = 2.108$, $p = 0.103$), or moving (coronal: $F = 0.565$, $p = 0.69$; sagittal: $F = 0.677$, $p = 613$; horizontal: $F = 0.269$, $p = 0.879$); as well as during SLS (coronal: $F = 1.577$, $p = 0.111$; sagittal: $F = 0.732$, $p = 717$; horizontal: $F = 0.384$, $p = 0.966$). No significant differences were found when performing independent t-tests between the injured and uninjured participants.

Discussion

This study aimed to identify biomechanical imbalances in hip joint kinematics and muscle electromyography after acute groin injury, and following potential deficiencies along the course of rehabilitation. There was no significant effect of the testing occasion on the GM:AL sEMG ratio or hip joint kinematics in any plane during SHF movement when the injured leg was both stance and moving; as well as in SLS when the injured leg was stance (weight-bearing) although low subject numbers must be foregrounded as a caveat. Interestingly, no significant interaction was also found between the injured athletes and healthy controls in both sEMG and kinematic measures, at any testing occasion.

SRGP still remains a challenge for sports medicine. A number of research reported a previous groin injury as a major risk factor for SRGP (Arnason et al., 2004, Maffey and Emery, 2007),

which suggests that underlying imbalances still remain in athlete after his primary injury, despite positive outcomes of the clinical assessment (Holmich et al., 2004). It has been suggested that the SRGP recovery time with the exercise-focused rehabilitation programme is 8 - 12 weeks (Holmich et al., 1999), and this timeframe was also used in our study as sufficient time for the participants to recover from their injuries. Indeed, the study participants got significantly better between the first and last testing occasion. Interestingly, this improvement was in general not followed by the sEMG and kinematic changes in injured athletes.

A number of biomechanical imbalances in athletes with SRGP have been reported, including mainly strength and flexibility deficiencies (Malliaras et al., 2009, Mohammad et al., 2014, Nevin and Delahunt, 2013, Thorborg et al., 2010). Few studies have focused on the elctromyographic deficits (Morrissey et al., 2012a, Cowan et al., 2004b), reporting a clear association between existing sEMG deficits and SRGP; and none at all on movement pattern changes.

This suggests that despite a seemingly successful rehabilitation and minimal warning signs discovered during the clinical examination, there are underlying imbalances, which increase the athlete's risk of SRGP after acute episode. One study by Jansen et al (Jansen et al., 2009) mentions such phenomenon and reports no recovery of the transverse abdominal muscle thickness in athletes with SRGP despite successful clinical outcomes following a course of rehabilitation.

In this study, I found that despite a comprehensive exercise program and a significant improvement in clinical presentation of their injury, the athletes failed to improve their coronal plane hip muscle activation ratio.

The rehabilitation focussing on optimising the movement pattern has been reported successful in other multi-structural clinical entities, such as with lower back and shoulder pathologies

(Roussel et al., 2009, Mottram et al., 2009, Roussel et al., 2013, Worsley et al., 2013). There is also convincing evidence that the kinematic dysfunctions need to be addressed in order to optimise the rehabilitation and promote a successful recovery from pain –as has been shown in runners with patella-femoral pain (Noehren et al., Willy et al.).

The rehabilitation programme implemented in this study was based on previously published programmes, showing good clinical outcomes (Holmich et al., 1999, Weir et al., 2011b). The clinical examination followed by this study was also based on previously published research, using reliable clinical tests (Holmich et al., 2004). The programme focused on stretching and strengthening of certain structures, and no attention was given to movement patterns and their retraining. No previous research has identified the movement imbalances in SRGP athletes, or investigated the effects of the movement re-patterning on the effects of SRGP rehabilitation. However, given a still very high prevalence and morbidity of this debilitating condition, an increased focus on optimising the hip joint kinematics alongside the strengthening exercises seems to be a natural step forward.

It was surprising that no significant interaction existed when comparing the injured with the uninjured athletes. The reason for no significant interaction may be that the muscle activation and kinematic patterns are less affected by short compared with longer term groin pain. There is in fact no research investigating muscle activation patterns immediately after acute injury. A number of studies have, however, shown an association between the alteration of muscle activation and chronic pain or overload (Dingenen et al., 2015, Daly et al., 2015, Bourne et al., 2015, Morrissey et al., 2012a, Barton et al., 2012). It is therefore possible that an acutely injured muscle activation is not altered, particularly when measuring an electromyographic output from the whole muscle, with only two bipolar electrodes. Potentially, the healthy areas of the injured muscles put an increased effort to maintain a 'normal' level of muscle activation

in order to maximise function, which is also consistent with a lack of kinematic differences observed between the injured and uninjured players in this study.

Alternatively, lack of muscle activation or kinematic differences between testing occasions as well as between the injured cohort and healthy controls at the outset may suggest that the measurement method of this study was either not sensitive enough to explore the biomechanical patterns in the *acutely* injured athletes or that the differences do not exist at baseline and are acquired during recovery.

Limitations

A major limitation of this study is a low number of participants and therefore the results of this study should be treated with caution. A complexity and high amount of time that the participants were requested to sacrifice in order to part take in the study (three occasions of minimum two hour visit in the Human Performance Laboratory excluding the travel time), as well as stringent inclusion and exclusion criteria (for example only including participants who were able to arrive for the first testing occasion up to five days after the injury) limited the number of participants that could be recruited. It could be argued that the study should be treated as a case-series due to the low number of participants. However, in order to define a study a case-series I would have had to disregard the results from the healthy control participants. Although the comparison between the injured and uninjured players were not significant at any point I decided to include the healthy participants' data in the study as the results may become significant with larger participants numbers.

In this study the original acute injury was assessed only clinically, no imaging diagnostic tests were taken. This may bias the results, as there are a number of structures within the groin area that may potentially cause the injury and result in different biomechanical alterations.

Another limitation of the study was a small number of physiotherapy consultations that each participant was given (three on three testing occasions). This may have had a negative effect on the participants' motivation to closely follow the exercise program, as well as increased the chance for participants to make mistakes in their exercises, which could not have been corrected in time.

Conclusions

There is no relationship between the clinical outcomes of the athlete's recovery after an acute groin injury and the change in the coronal plane muscle activation and hip joint kinematics.

No change in the GM:AL activation ratio was found in the injured leg between any of the testing occasions, in SHF and SLS manoeuvres.

In the stance leg during SHF and SLS manoeuvres the athletes show an initial change of the hip joint kinematics into the abduction direction during the conservative treatment. However, they return to the degree of adduction presented during the initial testing after completing the rehabilitation course, while showing a significant improvement in their clinical measures.

Chapter 8: Discussion

Main findings

The aim of this thesis was to explore the biomechanical factors associated with sports related groin pain (SRGP) in order to guide rehabilitation and prevention strategies; firstly by summarising already reported biomechanical patterns in a systematic review with meta-analysis; secondly by investigating hip joint electromyographic and kinematic deficits specific to athletic sub-groups with SRGP; and thirdly, by observing those deficiencies among athletes recovering from groin injury.

Limitations

Five key limitations that apply to the overall thesis are worthy of further discussion. Firstly I did not use a patient rated outcome measure which, in retrospect, could have been useful to better characterise our patient groups and also as a potential covariate in statistical analysis. Although there were some measures available, these were either too vague to detect the functional deficits (Functional Measurement Screen) and some too focused on the hip joint (Harris Hip Score). A functional Copenhagen Hip and Groin Outcome Score (HAGOS), which is more targeted in the athletic deficits associated with groin pain, had just been published and would have been a suitable patient reported outcome measure in this study. This seemed very secondary to our main focus and methodological design but will be employed in future work.

The limitations associated with using the surface electromyography, presented in detail in Chapter 3: Methods (p. 85), were present when collecting data for the studies. In particular, our choice of temporal rather than amplitude normalisation could be viewed as an additional limitation, but a conscious decision was made to analyse the less conventional muscle ratios within standardised movement phases (van der Hulst et al., 2010b, van der Hulst et al., 2010c,

Mathiassen et al., 1995). None of the standard normalisation procedures were relevant for the injured muscles (Daly et al., 2015, Burden et al., 2003b); and the aim of the study was to assess the biomechanical imbalance of the coronal plane antagonist muscles rather than identify the exact level of muscle activation. Further reasoning of this decision and the advantages and disadvantages of thereof are detailed in Chapter 3: Methods (p. 85).

Focusing on coronal plane muscle activation data and tri-planar hip joint kinematics was a conscious choice, based on the existing evidence of mainly coronal plane deficits associated with SRGP, as shown in the systematic review. Ideally, we would also have established the muscle activation patterns in other planes, but this was not possible due to the difficulty of access and deemed less likely to be useful given the location of symptoms. Nonetheless, further investigations of other muscular imbalances affecting the pelvis and hip stability may give useful information, for example exploration of sagittal plane relationships in relation to pelvis tilt.

Although the number of participants was very high - especially when one considers that data collection and analysis typically took a day per subject per test not including the time spent recruiting – the number in the longitudinal study was low. This was despite concerted and persistent recruitment efforts. A recommendation for future work is aligning data collection with sports group with high numbers of injured athletes.

The wider context

Prior to summarising what has been found and relating this to the literature, it is important to revisit some key underpinning factors concerning diagnosis and assessment in order to fully understand the sampling criteria employed and therefore to whom the research findings are relevant.

There has been a lot of debate in the literature regarding terminology, diagnostic categories and definitions of athletic groin pain. Studies included in this thesis were designed in 2011, when there was still little agreement on the classification and diagnosis of SRGP. I was therefore faced with difficult decisions and deliberately designed an inclusive approach, requiring participants to respond positively or negatively to commonly used clinical tests in such a way as to localise the pathology to a defined range of muscular and soft-tissue related pathologies and exclude symptoms of bony or articular origin (Holmich, 2007). I was cognisant of needing to balance the risk of regression to the mean, in that sample diversity may have confounded clear movement pattern description with the possibility that we may maximise relevance and generalizability with an inclusive approach.

This approach is not unusual, with a range of authors commonly avoiding overly defined decisions regarding study inclusion and exclusion criteria (Malliaras et al., 2009, Nevin and Delahunt, 2013, Arnason et al., 2004, Mohammad et al., 2014, O'Connor, 2004). Interestingly, our approach has been indirectly validated by the recent Doha agreement on the definitions and terminology of athletic groin pain, which defines diagnostic sub-categories of very similar nature to the ones we selected (Weir et al., 2015). This provides a very strong argument for the validity of the thesis results. Effectively, our criteria map to the Doha-defined adductor-, iliopsoas-, inguinal- and pubic-related pathologies, which were combined in the thesis.

Although sub-grouping participants further depending on a more exact injury classification may potentially have altered the results, it is unlikely for a number of reasons. Firstly, the consensus is that groin pain is usually a multi-structural entity, and that the majority of injured athletes suffer from secondary and/or tertiary causes of pain (Holmich, 2007), therefore combining multiple categories. Secondly, our studies show strong, significant and consistent results, which are primarily sport- and level-specific. If there was a necessity to assess the exact and primary diagnosis of the injured players, our results may not have been so obvious

due to smaller sub-sample numbers. Additionally, one of the aims of the thesis was to guide rehabilitation and prevention strategies, and make the results applicable to clinical practice. Therefore the inclusion criteria for the study, and in consequence the investigated cohort, were based on combined, commonly applied, clinical tests. In order to diagnose groin pain for each separate category of pain (adductor-, iliopsoas-, inguinal- and pubic-related) a clinical test combination must reproduce the predominant symptom(s), which in the presence of secondary and/or tertiary causes may be challenging for a clinician. Therefore combining all of the tests together and not sub-dividing the participants' makes the findings more applicable and easier to implement in clinical settings. Most importantly, we have uncovered unique findings about sports and participation level specificity in terms of movement patterns that are not considered in current clinical guidelines. *Perhaps these factors are more important than diagnostic sub-groups?* This question may be provocative but is certainly worth posing, and our data provides a provisionally affirmative answer. Further confirmation would emerge from studies investigating the muscle activation and movement patterns focussed rehabilitation, and whether this treatment yields better outcomes than traditional conservative treatment.

Multiple muscle activation and kinematic patterns were found in the systematic review in professional and amateur athletes; as risk factors from prospective studies, as well as associations with existing SRGP. The high recurrence rate, and the fact that previous groin injury is reported to be a major risk factor in subsequent SRGP (Maffey and Emery, 2007, Arnason, 2004, Whittaker et al., 2015) suggest that current rehabilitation and management approaches do not address all potential deficits in SRGP. The results of the experimental studies in the thesis revealed some of these potential deficits. The coronal plane muscle activation and the hip joint kinematic patterns have not been extensively investigated in the association with SRGP. There were clear muscle activation and movement pattern imbalances, which should be considered in designing pre- and re-habilitation programmes for SRGP.

Additionally, muscle activation and kinematic patterns varied between the levels and types of sport. This was a surprising finding, as it was expected that the biomechanical effect on the athletes would be similar across all sports, and levels of athlete. Instead, different movement strategies were demonstrated depending on the sport and level of play. In particular, clear similarities within professional as opposed to amateur groups of players: the former group seeming to present with highly activated gluteal muscles while 'turning off' the adductors, whereas the latter group increased adductor activation alongside reduced gluteal muscle activation in the presence of pain.

The sport- and level- specific differences observed in the observational study may have occurred due to the differences in the treatment and playing load in different groups of athletes. As further discussed in the observational study chapter, professional players, in opposition to amateurs, tend to have well-structured, closely supervised and often gluteal-driven rehabilitation and prevention programmes. Therefore, they are likely to have well-developed, activated and strong gluteal muscles that are less likely to display deficits in function in SRGP. In amateurs, the gluteal muscles tend to be weaker (Niinimaki et al., 2015, Niemuth et al., 2005), so any added loading may need to be absorbed by the adductor muscles.

It is possible that the different training and participation levels, alongside potentially different genetic factors, may explain the associations identified and are irrelevant to SRGP. This seems unlikely for two main reasons. Firstly, professional athlete pre-habilitation does not differ significantly from rehabilitation in terms of a gluteal focus, with hip extensor and abductor dominant strength training being a strong feature of usual football preparation in the form of power squats, side-plank, gluteal activation and multi-directional activities (Styles et al., 2015, Sanchis-Moysi et al., 2011, Crow et al., 2012). Adductor strengthening may be a more salient feature of SRGP rehabilitation than usual sport, but would have resulted in adductor rather

than gluteal dominance in the results – the opposite of what was found. Secondly, the main training done by any elite sportsperson is participation in full, or deconstructed game situations (Jackman et al., 2013, McIntyre and Hall, 2005, Veale and Pearce, 2009). For these reasons, I am confident that the biomechanical patterns we see are likely to be injury related – either preceding pain onset or as a secondary adaptation.

The superiority of active exercise therapy for the SRGP treatment has long been established (Holmich et al., 1999, Jansen et al., 2008, Machotka et al., 2009), measured mainly by clinical outcomes of athletes completing rehabilitation (Jansen et al., 2008, Machotka et al., 2009). Although some biomechanical deficits, such as adductor muscle weakness, are recognised and included in published treatment strategies, usual practice does not include strongly advocate identifying and targeting potential biomechanical deficits. Specifically, I have found altered movement patterns and muscle imbalance to be strongly associated with SRGP. Therefore the focus and emphasis of the current guidelines need to be revisited.

Deficient neural drive may be associated with SRGP occurrence and is not explicitly addressed in SRGP rehabilitation programmes. The need of increased focus on the neuro-inhibitory mechanisms has been previously recognised in other sports-related injuries presenting a large challenge in sports, such as hamstring injures (Fyfe et al., 2013, Thelen et al., 2006, Daly et al., 2015). Implementing the heavily overloading, eccentric hamstring training (known as Nordic exercises), which maximises the hamstring muscle activation (Bourne et al., 2015) was reported to significantly reduce the first-time and recurrent hamstring injuries (Arnason et al., 2008, Petersen et al., 2011, van der Horst et al., 2015)

However limited, these papers give a novel approach and increasing evidence for the necessary elements of the hamstring injuries rehabilitation practice. A spectacular breakthrough in reducing the incidence of those injuries by Nordic exercises suggests that

similar mechanisms and, in consequence, treatment strategies should also be implemented in other persistent sports injuries.

Increasingly, published work describes neuro-muscular deficits associated with SRGP, (Morrissey et al., 2012a, Cowan et al., 2004b) with even more recent work providing and measuring a selection of exercises with an increasing muscle activation rate, focussing solely on the adductor muscles (Serner et al., 2013). This approach seems to be supported by the findings of this thesis, at least for the muscle activation deficits in professional athletes. Whether this approach would also change movement patterns has not yet been established. A similar approach may be warranted for the gluteal muscles in amateur athletes. We do not have an equivalent exercise to the Nordic hamstring for either the adductor or abductor muscle groups as yet – that is, an exercise that results in maximum activation to a break point. Perhaps this is unfeasible or perhaps it could represent a major step forward for SRGP management.

It suggests that there may be an underlying imbalance or deficit after the acute injury, which is not addressed in the current rehabilitation programmes. This imbalance is unrecognised in the clinical examination, leading to clinicians terminating the rehabilitation period potentially too early (Holmich et al., 1999) and allowing the athlete return to play prematurely. In the absence of clear clinical signs of any deficits, the athlete returns to his normal level of activity, and gets injured again. It may be that previously injured muscle has a propensity to become weak and inhibited without continued high load rehabilitation. Potentially, the risk for the injury recurrence may be decreased by a regular screening of the adduction strength, as it was reported to drop significantly two weeks before SRGP (Tyler et al., 2001).

In Chapter 7: Longitudinal study (p. 218) the findings indicate that neither the movement nor the muscle activation patterns were altered from baseline up to 8-12 weeks after injury. This

was surprising on the one hand given the trend for improved symptoms and function affecting 4 of the 5 players, but unsurprising in that no focus was given to muscle activation nor movement patterns in the traditional rehabilitation employed. It is worth noting that the sample size of the longitudinal study was very low, due to recruitment barriers discussed earlier, yet it can still be argued that the lack of change observed fits with other literature. For example, scapular retraining deficits are often found when measuring people with shoulder impingement syndrome but only change with very specifically targeted interventions (Worsley et al., 2013). Equally, only the targeted rehabilitation focussed on the kinematic patterns re-training was found to be effective in reducing one of the main risk factors in patella-femoral pain syndrome (Noehren et al., 2013), with the standard rehabilitation strategies failing to provide long-term success (Dolak et al., 2011).

However, as discussed in the longitudinal study section, the muscle activation imbalances may be secondary to the acute injury, and lack of proper focus on re-storing the coronal plane balance in the rehabilitation programmes may be the most important reason for the high recurrence of SRGP. The similarity of the (statistically insignificant) biomechanical pattern of the longitudinal study participants to the professional group in the observational study supports the hypothesis, that there is in fact no common adaptive response to the injury, pain or overload but rather that this relationship is level specific. Careful clinical examination and rehabilitation during functionally relevant manoeuvres may be key to not only improved rehabilitation success for SRGP, but also to reducing recurrence by re-storing and optimising biomechanical factors. The optimal methods for doing this need further study, and may include simplified versions of the complex measurements employed in this thesis. Potentially, modern sensors combined with a phone application of the dynamic goniometer measuring the changes in the hip joint kinematics in three planes, and/or basic muscle activation measures of

a very limited number of muscles could be useful and provide large amount of clinically applicable information.

A careful assessment of the actual muscle function in a functional setting is the more important as the relationship between muscle activation and force is yet to be established (Nishihara and Isho, 2012), in particular in a potentially injured muscle. Therefore potential strength deficits, or lack of thereof, may not be fully representative of actual functional deficits, and the treatment choices based purely on strength may be heavily biased. This provides another argument for a functional assessment of the movement and muscular 'behaviour' in functional tasks, which may be more sensitive to subtle abnormalities present in the athletes with SRGP (Boudreau et al., 2009, Crossley et al., 2011).

An additional and surprising finding of both observational and longitudinal study is the different pattern of the dominant versus non-dominant leg injured. All of the groups tended to injure their dominant leg, apart from the professional footballers, who showed a pattern of the non-dominant leg being more commonly injured, also reported in knee injuries (Krajnc et al., 2010). As the dominant leg was defined as the preferred kicking leg, it may raise a question whether the injury mechanisms may be associated with the training specificity. Among all of the investigated groups, the professional footballers perform the most kicking-specific training (Young and Rath, 2011, Kellis and Katis, 2007), which may then bias their self-reported dominance, as they are equally comfortable kicking with both legs. Alternatively, a high amount of kicking movement training puts more emphasis on open-chain movement patterns, meaning the standing leg is more challenged due to the higher loads associated with body deceleration and rotation (Mognoni et al., 1994, Orchard et al., 2002b).

Conclusions

My research shows clear biomechanical factors associated with SRGP that are participation level specific and partly sports specific. These include both muscle activation patterns and corresponding kinematic changes. My novel approach, and findings, represent a new dimension innovation in the clinical and research environments when designing and implementing prevention and rehabilitation programmes in athletes suffering from – or at high risk of - SRGP. Different levels of sport may require different approaches.

A strong argument can be made that coronal plane muscle activation and lower limb movement patterns need to be carefully assessed and addressed in the rehabilitation process, with the consideration that the imbalance may affect both the adductor and abductor muscles.

In order to plan and implement successful and efficient prevention and rehabilitation strategies for athletes, which include the hip coronal plane muscle activation and kinematic imbalances, simple and clinically applicable measuring devices may be needed.

This thesis also provides evidence that questions the conclusions of the recent Doha consensus, and make a case for extending them. We propose there are a number of imbalances and biomechanical deficits, which are level-and sport-specific, associated with SRGP. Research is needed to determine if addressing these gives better, more sustained, rehabilitation outcomes. The key to more successful prevention and rehabilitation programmes may be careful assessment of the pelvic girdle muscular and kinematic function and correction of the discovered imbalances.

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Appendix 1

Anatomy and pathology of the groin region

The definition of the anatomical landmarks of the groin area has always been troublesome. Anatomy books and dictionaries provide vague and non-specific definitions (LeBlanc and LeBlanc, 2003). However, a very useful, patho-anatomical 'groin triangle' model was proposed (Falvey et al., 2009), which summarises the anatomy of the area in the context of possible pathologies and provides a good anatomical guide for clinical diagnosis.

The groin triangle is based on the anterior aspect of the thigh and provides a clear reference to locate the structures and symptoms in the groin area. The apices of the triangle are the: anterior superior iliac spine (ASIS), pubic tubercle and '3G point', which is defined as the mid-point between ASIS and superior pole of the patella. The structures in the area are then described as lying within the triangle, medially, laterally or superiorly to it (Figure 36).

Additionally to simple anatomical description, this model also provides advice on how to conduct the optimal diagnostic process of the groin area and describes in more detail potentially serious entities, which should be investigated further.

Special attention is drawn to the pubic tubercle, the attachment site of several structures that can potentially cause groin symptoms. To facilitate the diagnosis of the pain arising from this region, Falvey introduced a model of "the pubic clock" (Figure 37). It gives clear and specific instructions on palpation of the pubic tubercle region and links the specific structures to the symptomatic areas. The pubic clock is in fact a simplified model of previously published Meyers' considerations about the structures attaching around the pubic symphysis joint (Meyers et al., 2005) with an additional layer of clinical application for diagnosis.

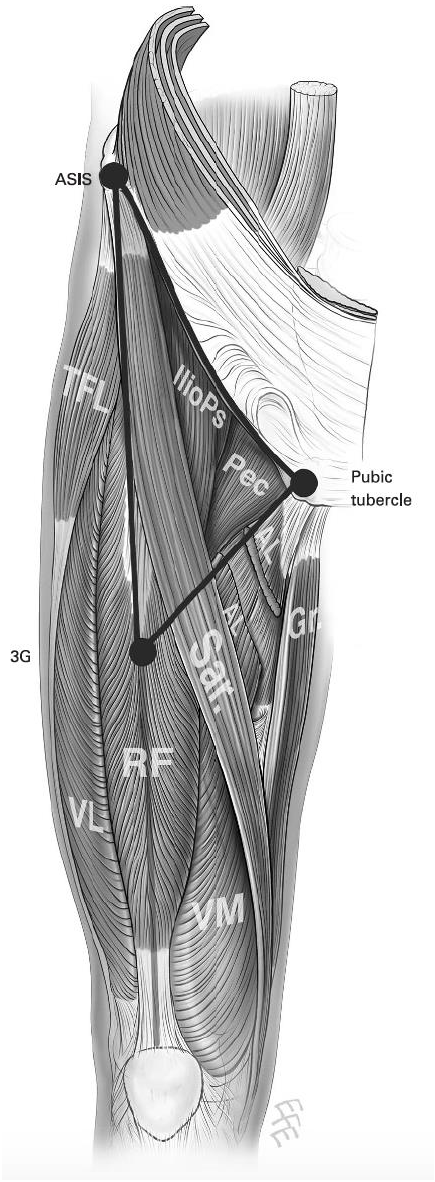


Figure 36: The groin triangle. AL - adductor longus; ASIS - anterior superior iliac spine; Gr - gracilis; IlioPS - iliopsoas; Pec - pectinius; RF – rectus femoris (Falvey et al., 2009).

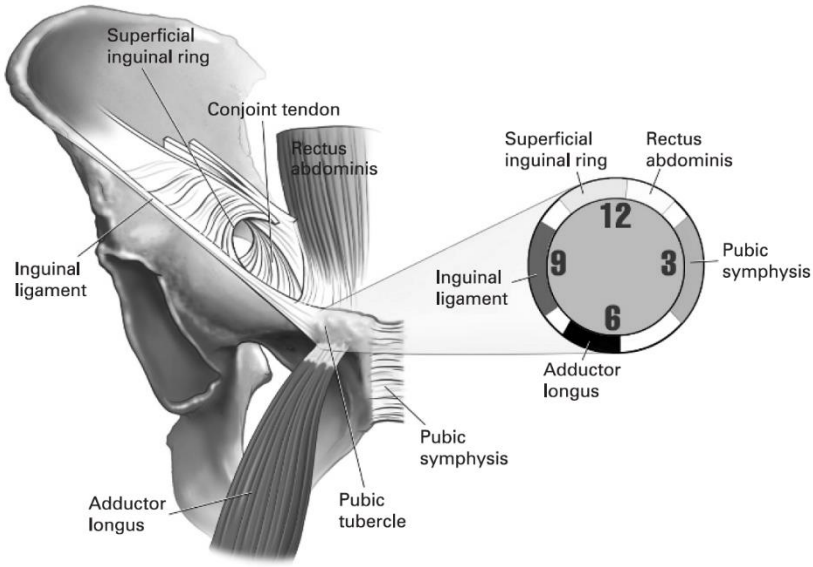


Figure 37: Pubic clock – a diagnostic clinical tool for SRGP diagnosis (Falvey et al., 2009)

Structures lying laterally to the triangle

The lateral border of the triangle extends from ASIS to a 3G point (Figure 36). Main structures, potentially triggering groin symptoms include: femoro-acetabular joint, trochanteric bursa, tensor fasciae latae muscle and iliotibial band. From a clinical and diagnostic perspective, pathologies in the femoro-acetabular (hip) joint, including the trochanteric bursa, need to be excluded early in the differential diagnostic process as they are likely to warrant different management (Anderson et al., 2012, Lahner et al., 2014). This was recognised in the Doha agreement, which separated hip-related pathologies from others in the terminology of patients groups. Similar approach was undertaken in this thesis, and testing positive on any of the common and validated hip joint tests was one of the main exclusion criteria for the study participants.

In fact, a differential diagnosis for groin pain should be particularly sensitive to any signs of the symptomatic femoro-acetabular impingement (FAI), morphological hip joint pathology of

unknown origin, presenting as an anatomical misalignment of the femoral head and acetabulum (Byrd, 2013, Monazzam et al., 2013, Hessel, 2014). It may present in two variants: CAM and PINCER. CAM is a form of deformity of the proximal part of the femur, with extra amount of bone in the area postero-inferiorly to the femoral head and in consequence – shallower femoral neck. PINCER presents as excessive acetabulum, which is either too deep or ill-oriented. Although CAM is classically associated with young, otherwise healthy athletic male population and PINCER in middle aged women (Amanatullah et al., 2015), they often co-exist and provide a mechanical misalignment between the femoral head and acetabulum, and a high potential for a hip joint pathology (Amanatullah et al., 2015). Importantly, FAI should not be used as a clinical diagnosis, as it may not be symptomatic; in fact there are studies reporting an asymptomatic presence of FAI in as many as 92% of healthy population (Schmitz et al., 2013, Kapron et al., 2011).

FAI, similarly to SRGP, is commonly observed in athletes participating in kicking and/or multi-directional sports disciplines (Lahner et al., 2014, Fraitzl et al., 2010, Hammoud et al., 2012, Hessel, 2014, Johnson et al., 2012, Keogh and Batt, 2008). Weir et al. (Weir et al., 2011a) showed a very high prevalence of FAI in athletes diagnosed with SRGP. In his study 64 out of 68 patients with groin pain (95%) have shown radiologically confirmed FAI. Nepple et al. (Nepple et al., 2012) retrospectively reviewed 123 cases of hip and groin pain and his findings are similar to Weir's. However, patients with both hip and groin symptoms were included in both studies, so the incidence of the hip pathologies may have been overestimated in this population.

However, another study reported a low prevalence of groin pain in patients with various hip malformations (Gosvig et al., 2010). This study also investigated radiographic signs of hip osteoarthrtitis, an entity that might as well be responsible for groin symptoms according to Falvey, and associated with pain beyond the lateral border of the groin triangle.

Other pathologies potentially causing groin pain and situated laterally to the triangle include: femoral neck stress fracture, proximal iliotibial band friction syndrome, femoral and lateral cutaneous nerve entrapment (Brukner et al., 2012). Although quite commonly reported, these entities have not been associated in relation to pain arising from the groin area.

A clear diagnostic and clinical entity potentially presenting as the pain in the groin is the acetabular tear. Narvani et al. (Narvani et al., 2003) found that 22% (4 out of 18) patients presenting with groin pain had a tear of the acetabular labrum on MRI, 3 located anterio-superiorly, in the lateral part of hip joint. Silvis et al (Silvis et al., 2011) found that out of 39 professional and non-professional hockey players with groin pain, 25 (64%) showed positive MRI findings of hip pathologies. 22% of these players were diagnosed with tear of the acetabular labrum based on MRI findings. Burnett et al. (Burnett et al., 2006) retrospectively reported groin pain symptoms in 61 out of 66 patients with as arthroscopically confirmed acetabular tear, whereas in a study published by Fitzgerald (Fitzgerald, 1995) 49 out of 55 patient with an acetabular tear identified with arthrography had groin symptoms. Bradshaw et al. (Bradshaw et al., 2008) has reported that hip joint pathology was the most common diagnosis among 218 patients with groin symptoms (45.9%).

Structures lying within the triangle

Pathologies, which may cause groin symptoms and arise from structures lying within the triangle include iliopsoas syndrome, rectus femoris tendinopathy and apophysitys, femoral hernia and genitofemoral and cutaneus nerve entrapments. In this thesis, consistently with the Doha agreement terminological guidelines, the participants for the observational study were included if presenting with the iliopsoas or, abdominal muscles pathologies. Participants wi th suspected hernias and nerve entrapment were excluded from the study.

Relationship between iliopsoas muscle pathologies and groin symptoms have been reported previously (Holmich, 2007, Lovell, 1995). Iliopsoas muscle-related pathologies were found to be the second most common entity responsible for groin symptoms in the Holmich (35.3%), but not the Lovell study (3%).

Abdominal pathologies have also been reported in association with groin pain (Jansen et al., 2010, Cowan et al., 2004b, Mens et al., 2006) and the role of the abdominal muscles (potentially due to their insertion to the superior part of the pubic bone and therefore the ability to affect the forces and loading travelling through) is commonly recognised.

Structures lying medially to the triangle

Structures located medially to the triangle are thought to be main causes of SRGP (Holmich, 2007). The area is mostly filled with hip adductor muscles, providing important stability mechanisms for the hip, groin and pelvis areas. According to Falvey et al. (Falvey et al., 2009) "the abnormal mechanics that arise as a result of adductor dysfunction play a critical role in the generation of a chronic pain/dysfunction cycle in the area".

A number of studies have reported differences in adductor muscle function associated with SRGP (Morrissey et al., 2012b, Crow et al., 2010, Mens et al., 2006, Malliaras et al., 2009).

The most common pathologies affecting the adductor muscles include the adductor and/or gracilis muscle ethensopathy and the pathology of the adductor muscle -tendon junction. According to the Doha agreement , adductor-related groin pain (including the pathology of all of the adductor muscles, with the adductor longus and gracilis being the most commonly injured) is one of the sub-groups identified within the SRPG.

The recommendations for diagnosing the adductor-related pathology include the pain provocation tests (active hip adduction) and the palpation of the potentially injured areas. Among many active hip adduction tests, the squeeze test is most commonly used, and is

reported to be a sensitive, but not very specific test for groin pathologies (Delahunt et al., 2011b, Delahunt et al., 2011a). Therefore, further pain reproduction by palpation of the painful areas is recommended to improve diagnostic confidence (Holmich, 2007, Falvey et al., 2009). Although those diagnostic criteria are widely used by clinicians, it is worth noting that SRGP is a multi-structural pathology and is likely to be related with more than one structure (Holmich, 2007).

Another common pathology in this area include an acute adductor muscle injury, which may lead to chronic groin pain and the 'groin pain cycle' (Renstrom and Peterson, 1980).

Pubic symphysis and pubic ramus are situated medially to the triangle and there is an established relation within authors between pathologies occurring in that region and groin symptoms (Verrall et al., 2007b, Verrall et al., 2005b, Slavotinek et al., 2005). Bradshaw et al. (Bradshaw et al., 2008) reported osteitis pubis in 20.6% of participants presenting groin symptoms, which was a second most common entity diagnosed in his study. "Osteitis pubis", "athletic pubialgia" are the common terms used to describe clinical entities in this region (Hiti et al., 2011, Kunduracioglu et al., 2007, Lovell et al., 2006, Mandelbaum and Mora, 2005, McCarthy and Vicenzino, 2003, Johnson, 2003, Rodriguez et al., 2001, Williams et al., 2000, Major and Helms, 1997, Fricker, 1997, Fricker et al., 1991). There is, though, still a lack of consensus regarding the terminology as some authors use these terms to describe a general pain in the pubis area, while others insist on leaving them to very specific pathologies to the pubic symphysis.

Structures lying superiorly to the triangle

Apart from already discussed abdominal muscle pathologies, other common clinical entities in the area superiorly to the triangle are hernias. The term "hernia" is not consistently used among authors and true abdominal, inguinal or femoral hernia is often confused with entities

such as sport's hernia or abdominal or inguinal wall deficiency. Although authors try to distinguish between these different entities, diagnosis is not straightforward. Holmich et al. (Holmich, 2007) found only one true hernia among 207 athletes with groin pain, his findings, though, are not supported by any other high quality study. Despite the lack of strong evidence, "sports" or "sportsman's" hernia has been investigated by several authors (Orchard et al., 1998, Steele et al., 2004, Caudill et al., 2008, Fon and Spence, 2000) and the term is still commonly used in relation to groin syndromes.

Appendix 2

Tables summarising the results of comparisons between the dominant and non-dominant legs of the healthy controls in each subgroup, in both movement manoeuvres.

A: Summary measurements of professional footballers during standing hip flexion; describing the stance, dominant leg with respect to the non-dominant leg of healthy controls.

Movement phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
Surface EMG									
GM:AL	0.72 (0.24)	-0.62 (0.5)	0.02*↓	0.27(0.24)	-1.03 (0.51)	0.03*↓	0.58 (0.24)	0.06 (0.46)	0.32
Kinematics									
Sagittal hip (Flex +)	1.18 (1.76)	3.92 (1.52)	0.25	-4.93 (1.37)	-6.26 (0.91)	0.42	-2.95 (1.16)	-1.22 (1.33)	0.33
Coronal hip (Add +)	0.83 (0.73)	0.26 (0.22)	0.69	-3.42 (0.75)	-3.43 (0.69)	1	-2.54 (0.98)	-1.85 (0.92)	0.61
Horizontal hip (IR +)	-14.97 (2.96)	-2.33 (1.87)	<0.01*↑	-3.38 (0.93)	-4.67 (0.92)	0.33	-16.28 (3.13)	-6.28 (1.82)	0.01*↑

B: Summary measurements of professional footballers during standing hip flexion; describing the moving, dominant leg with respect to the non-dominant leg of healthy controls.

Movement phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
Surface EMG									
GM:AL	0.19 (0.5)	0.16 (0.3)	0.95	-0.92 (0.35)	-0.73 (0.3)	0.69	-0.67 (0.37)	-0.67 (0.2)	0.99
Kinematics									
Sagittal hip (Flex +)	4.97 (2.05)	3.36 (2)	0.58	68.75 (1.88)	73.45 (1.82)	0.08**↑	74.04 (1.66)	76.81 (1.94)	0.28
Coronal hip (Add +)	0.13 (0.94)	-0.27 (0.72)	0.24	3.27 (1.47)	-0.46 (0.7)	6.22	1.88 (1.59)	-0.73 (0.92)	0.16
Horizontal hip (IR +)	-4.61 (1.9)	-11.17 (1.97)	0.02*↓	6.22 (1.87)	8.12 (0.78)	0.35	-0.28 (1.6)	-3.05 (1.69)	0.24

Table 35: Results from comparing surface electromyography and kinematic data between the dominant and non-dominant legs of the healthy professional footballers during standing hip flexion when the leg is weight bearing (stance) (A) and moving (B). Annotations: * = p < 0.05; ** = p < 0.1; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error. ↑ = increased in non–dominant leg; ↓ = decreased in non-dominant leg.

A: Summary measurements of professional footballers during standing hip flexion; describing the stance, dominant leg with respect to the non-dominant leg of healthy controls.

Movement phase	Early			Middle			Late		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
Surface EMG									
GM:AL	1.2 (0.18)	0.65 (0.15)	0.019*↓	0.86 (0.15)	0.4 (0.15)	0.04*↓	1.4 (0.19)	1 (0.19)	0.14
Kinematics									
Sagittal hip (Flex +)	5.93 (1.65)	6 (1.83)	0.98	-5.36 (0.75)	-5.9 (0.88)	0.65	0.63 (1.6)	0.11 (1.68)	0.62
Coronal hip (Add +)	-0.39 (0.82)	4.98 (0.8)	<0.01*↑	-5.44 (0.55)	-4.7 (0.62)	0.37	-5.92 (1.03)	0.27 (0.73)	<0.01*↑
Horizontal hip (IR +)	-2.37 (1.99)	-0.79 (1.62)	0.54	-4.75 (0.61)	-3.32 (0.6)	0.09	-6.72 (2.02)	-4.11 (1.84)	0.34

B: Summary measurements of amateur footballers during standing hip flexion; describing the moving, dominant leg with respect to the non-dominant leg of healthy controls.

Movement phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
Surface EMG									
GM:AL	-0.05 (0.15)	0.59 (0.15)	<0.01*↑	-0.96 (0.1)	-0.08 (0.13)	<0.01*↑	-0.92 (0.11)	-0.33 (0.14)	<0.01*↑
Kinematics									
Sagittal hip (Flex +)	7.93 (2.24)	10.62 (2.3)	0.4	61.63 (1.83)	51.54 (3.95)	0.02*↓	70.42 (1.5)	62.23 (2.9)	0.014*↓
Coronal hip (Add +)	-1.72 (0.11)	-6.92 (0.18)	<0.01*↓	-0.72 (1.33)	5.21(0.94)	<0.01*↑	-2.52 (0.83)	-1.76 (0.95)	0.65
Horizontal hip (IR +)	-4.04 (1.55)	-4.54 (2.75)	0.87	6.33 (1.66)	10.35 (1.93)	0.12	1.97 (2.59)	5.51 (2)	0.28

Table 36: Results from comparing surface electromyography and kinematic data between the dominant and non-dominant legs of the healthy amateur footballers during standing hip flexion when the leg is weight bearing (stance) (A) and moving (B). Annotations: * = p < 0.05; ** = p < 0.1; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error. ↑ = increased in non–dominant leg; ↓ = decreased in non-dominant leg.

A: Summary measurements of professional rugby players during standing hip flexion; describing the stance, dominant leg with respect to the non-dominant leg of the uninjured control group.

Movement phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
	Dominant	Non-dominant	Statistic (p)	Dominant	Non-dominant	Statistic (p)	Dominant	Non-dominant	Statistic (p)
Surface EMG									
GM:AL	1.29 (0.26)	0.77 (0.16)	0.11	0.86 (0.31)	1.02 (0.25)	0.68	1.67 (0.22)	1.97 (0.23)	0.34
Kinematics									
Sagittal hip (Flex +)	21.79 (0.58)	24.75 (1.14)	<0.05*↑	-7.32 (1.09)	-1.78 (0.96)	<0.01*↑	13.71 (1.73)	18.74 (1.46)	<0.05*↓
Coronal hip (Add +)	-2.29 (0.79)	-0.71 (1.28)	0.3	-7.55 (1)	-7.31 (0.97)	0.87	-8.24 (0.57)	-12.34 (2.58)	0.14
Horizontal hip (IR +)	-7.55 (2.1)	-3.33 (3.13)	0.26	-3.34 (0.75)	-0.85 (1.02)	0.06	-17.26 (2.1)	0.6 (2.76)	<0.01*↑

B: Summary measurements of professional rugby players during standing hip flexion; describing the moving, dominant leg with respect to the non-dominant leg of the uninjured control group.

Movement phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
Measured leg	Dominant	Non-dominant	Statistic (p)	Dominant	Non-dominant	Statistic (p)	Dominant	Non-dominant	Statistic (p)
Surface EMG									
GM:AL	-0.09 (0.29)	0.86 (0.15)	<0.05*↑	-1.34 (0.26)	-0.66 (0.15)	<0.05*↑	-1.24 (0.18)	-1.2 (0.15)	0.86
Kinematics									
Sagittal hip (Flex +)	22.05 (0.85)	18.06 (1.42)	<0.05*↓	60.7 (1.43)	67.2 (2.7)	<0.05*↑	83.66 (1.69)	80.83 (1.56)	<0.05*↓
Coronal hip (Add +)	-4.61 (1.1)	-5.9 (1)	0.39	8.88 (1.48)	-1.42 (3.47)	<0.05*↓	2.51 (2.02)	-4.73 (4.33)	0.15
Horizontal hip (IR +)	-11.83 (2.33)	-12.79 (2.69)	0.79	1.52 (1.63)	8.92 (2.43)	<0.05*↑	-7.61 (2.98)	-2.9 (7)	0.54

Table 37: Results from comparing surface electromyography and kinematic data between the dominant and non-dominant legs of the healthy professional rugby players during standing hip flexion when the leg is weight bearing (stance) (A) and moving (B). Annotations: * = p < 0.05; ** = p < 0.1; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error. ↑ = increased in non–dominant leg; ↓ = decreased in non-dominant leg.

A: Summary measurements of Ultimate Frisbee during standing hip flexion; describing the stance, dominant leg with respect to the non-dominant leg of healthy controls.

Movement Phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
	Dominant	Non-dominant	Statistic (p)	Dominant	Non-dominant	Statistic (p)	Dominant	Non-dominant	Statistic (p)
Surface EMG									
GM:AL	0.36 (0.37)	0.48 (0.27)	0.81	0.26 (0.38)	0.78 (0.33)	0.31	1.42 (0.46)	1.69 (0.26)	0.62
Comments									
Kinematics									
Sagittal hip (Flex +)	25.33 (1.02)	26.01 (1.24)	0.67	-3.37 (1.72)	-5.72 (0.81)	0.24	22.19 (1.53)	19.92 (2.27)	0.42
Coronal hip (Add +)	-2.54 (0.82)	4.17 (1.22)	<0.01*↑	-5.46 (2.32)	-11.59 (1.42)	<0.05*↓	-11.15 (2.68)	-10.71 (1.38)	0.89
Horizontal hip (IR +)	-11.49 (1.66)	7.87 (1.63)	<0.01*↑	-2.18 (1.47)	-4.89 (1.28)	0.19	-14.19 (3.33)	4.16 (2.23)	<0.01*↑

B: Summary measurements of Ultimate Frisbee during standing hip flexion; describing the moving, dominant leg with respect to the non-dominant leg of healthy controls.

Movement Phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
	Dominant	Non-dominant	Statistic (p)	Dominant	Non-dominant	Statistic (p)	Dominant	Non-dominant	Statistic (p)
Surface EMG									
GM:AL	0.87 (0.35)	0.35 (0.41)	0.34	-0.37 (0.31)	-0.59 (0.46)	0.69	0.02 (0.42)	0.08 (0.55)	0.93
Comments									
Kinematics									
Sagittal hip (Flex +)	24.99 (1.04)	22.17 (1.17)	0.08	67.38 (2.17)	71.68 (3.41)	0.3	93.52 (2.9)	92.81 (3.54)	0.88
Coronal hip (Add +)	0.62 (1.22)	-4.38 (0.91)	<0.01*↓	1.01 (2.42)	-0.03 (3.18)	0.8	5.85 (1.6)	-0.14 (2.62)	0.07
Horizontal hip (IR +)	6.2 (1.91)	-11.36 (2.14)	<0.01*↓	3.39 (3.88)	3.99 (2.67)	0.9	11.32 (2.36)	-10.71 (5.43)	<0.01*↓

Table 38: Results from comparing surface electromyography and kinematic data between the dominant and non-dominant legs of the healthy Ultimate Frisbee players during standing hip flexion when the leg is weight bearing (stance) (A) and moving (B). Annotations: * = p < 0.05; ** = p < 0.1; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error. ↑ = increased in non–dominant leg; ↓ = decreased in non-dominant leg.

A: Summary measurements of field hockey players during standing hip flexion; describing the stance, dominant leg with respect to the non-dominant leg of healthy controls.

Movement Phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)
Surface EMG									
GM:AL	0.36 (0.37)	0.48 (0.27)	0.81	0.26 (0.38)	0.78 (0.33)	0.31	1.42 (0.46)	1.69 (0.26)	0.62
Comments									
Kinematics									
Sagittal hip (Flex +)	25.33(1.02)	26.01 (1.24)	0.67	-3.37 (1.72)	-5.72 (0.81)	0.24	22.19 (1.53)	19.92 (2.27)	0.42
Coronal hip (Add +)	-2.54 (0.82)	4.17 (1.22)	<0.01*↓	-5.46 (2.32)	-11.59 (1.42)	<0.05*↓	-11.15 (2.68)	-10.71 (1.38)	0.89
Horizontal hip (IR +)	-11.49 (1.66)	7.87 (1.63)	<0.01*↑	-2.18 (1.47)	-4.89 (1.28)	0.19	-14.19 (3.33)	4.16 (2.23)	<0.01*↑

B: Summary measurements of field hockey players during standing hip flexion; describing the moving, dominant leg with respect to the non-dominant leg of healthy controls.

Movement Phase	Early (mean (SE))			Middle (mean (SE))			Late (mean (SE))		
	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)	Uninjured	Injured	Statistic (p)
Surface EMG									
GM:AL	0.87 (0.35)	0.35 (0.41)	0.34	-0.37 (0.31)	-0.59 (0.46)	0.69	0.02 (0.42)	0.08 (0.55)	0.93
Comments									
Kinematics									
Sagittal hip (Flex +)	24.99 (1.04)	22.17 (1.17)	0.08	67.38 (2.17)	71.68 (3.41)	0.3	93.52 (2.9)	92.81 (3.54)	0.88
Coronal hip (Add +)	0.62 (1.22)	-4.38 (0.91)	<0.01*↓	1.01 (2.42)	-0.03 (3.18)	0.8	5.85 (1.6)	-0.14 (2.62)	0.07
Horizontal hip (IR +)	6.2 (1.91)	-11.36 (2.14)	<0.01*↓	3.39 (3.88)	3.99 (2.67)	0.9	11.32 (2.36)	-10.71 (5.43)	<0.01*↓

Table 39: Results from comparing surface electromyography and kinematic data between the dominant and non-dominant legs of the healthy field hockey players during standing hip flexion when the leg is weight bearing (stance) (A) and moving (B). Annotations: * = $p < 0.05$; ** = $p < 0.1$; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in non–dominant leg; ↓ = decreased in non-dominant leg.

Phase: Moving I				
Measured leg	**Dom**	**NonDom**	**Statistic (p)**	
sEMG GM:AL	-0.7 (0.46)	0.63 (0.25)	<0.05*↑	
Sagittal hip	10.67 (2.7)	8.45 (1.93)	0.51	NB Flex +
Coronal hip	3.92 (1.12)	3.63 (1.05)	0.85	NB Add +
Horizontal hip	-1.38 (2)	-13.91 (1.76)	<0.01*	NB IR +

Phase: Moving II				Phase: Stance I		
Measured leg	**Dom**	**NonDom**	**Statistic (p)**	**Dom**	**NonDom**	**Statistic (p)**
sEMG GM:AL	-1.32 (0.37)	0.5 (0.24)	<0.01*↑	-1.21 (0.38)	0.13 (0.25)	<0.01*↑
Sagittal hip	11.32 (1.58)	20.17 (1.89)	<0.01*↑	20.89 (3.48)	28.88 (2.75)	0.08
Coronal hip	0.59 (0.33)	0.45 (0.71)	0.86	4.56 (1.28)	4.12 (1.19)	0.8
Horizontal hip	-1.38 (1.99)	-13.91 (1.76)	<0.01*↓	-3.26 (1.61)	-15.49 (1.96)	<0.01*↓

Phase: Moving III				Phase: Stance II		
Measured leg	**Dom**	**NonDom**	**Statistic (p)**	**Dom**	**NonDom**	**Statistic (p)**
sEMG GM:AL	-0.89 (0.35)	-0.11 (0.2)	0.38	-0.48 (0.44)	-0.06 (0.18)	0.38
Sagittal hip	45.53 (4.16)	40.45 (3.71)	0.37	67.33 (3.67)	67.74 (2.7)	0.93
Coronal hip	11.91 (1.16)	12.19 (1.01)	0.86	16.32 (2.03)	16.77 (1.11)	0.85
Horizontal hip	-3.2 (1.75)	1.08 (1.17)	<0.05*↑	-5.34 (2.64)	-14.28 (2.01)	<0.01*↑

Phase: Moving IV				Phase: Stance III		
Measured leg	**Dom**	**NonDom**	**Statistic (p)**	**Dom**	**NonDom**	**Statistic (p)**
sEMG GM:AL	-1.04 (0.43)	0.31 (0.21)	<0.01*↑	-1.33 (0.59)	0.68 (0.29)	<0.01*↑
Sagittal hip	-48.66 (3.64)	-52.05 (2.65)	0.46	17.44 (2.28)	16.56 (1.59)	0.75
Coronal hip	-14.77 (1.33)	-15.28 (1.24)	0.78	-0.21 (1.28)	0.8 (1.16)	0.56
Horizontal hip	1.21 (1.75)	0.84 (1.03)	0.86	-3.85 (1.63)	-14.13 (1.8)	<0.01*↓

Table 40: Results from comparing surface electromyography and kinematic data between the dominant and non-dominant legs of healthy professional footballers during single leg squat when the leg is moving. Annotations: * = p < 0.05; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in non–dominant leg; ↓ = decreased in non-dominant leg.

	Phase: Moving I					
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**			
sEMG GM:AL	0.62(0.24)	1.33(0.19)	<0.05*↑			
Sagittal hip	13.9(1.48)	12.95(1.57)	0.66	NB Flex +		
Coronal hip	7.49(0.94)	-1.66(1.12)	<0.01*↓	NB Add +		
Horizontal hip	-6.58(2.48)	-6.06(2.28)	0.86	NB IR +		
	Phase: Moving II			**Phase: Stance I**		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
sEMG GM:AL	0.48(0.19)	0.92(0.22)	0.13	0.55(0.17)	1.02(0.2)	0.08
Sagittal hip	26.46(2.52)	26.48(3.09)	1	39.94(3.07)	40.23(3)	0.95
Coronal hip	-0.57(0.83)	1.06(0.67)	0.13	7.04(1.07)	-0.47(1.29)	<0.01*↓
Horizontal hip	-3.83(0.76)	-0.81(0.89)	<0.05*↑	-6.58(2.48)	-4.36(2.77)	0.55
	Phase: Moving III			**Phase: Stance II**		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
sEMG GM:AL	0.5(0.08)	1.02(0.17)	<0.01*↑	0.4(0.1)	1.14(0.16)	<0.01*↑
Sagittal hip	24.78(2.67)	24.3(2.57)	0.9	64.89(2.17)	63.79(2.65)	0.75
Coronal hip	3.36(1.04)	7.38(0.96)	<0.01*↑	10.78(1)	6.58(1.53)	<0.05*↓
Horizontal hip	-0.48(1.07)	0.54(1.11)	0.51	-8.16(2.31)	-2.59(2.31)	0.09
	Phase: Moving IV			**Phase: Stance III**		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
sEMG GM:AL	0.72(0.1)	1.14(0.18)	<0.05*↑	0.52(0.18)	1.14(0.27)	0.07
Sagittal hip	-50.36(4)	-46.66(2.55)	0.44	14.98(3.27)	16.82(1.33)	0.6
Coronal hip	-6.79(1.32)	-10.53(1.01)	<0.05*↓	3.58(1.07)	-4.03(1.45)	<0.01*↓
Horizontal hip	1.13(1.89)	-1.02(1.27)	0.35	-6.06(2.28)	-3.68(2.57)	0.49

Table 41: Results from comparing surface electromyography and kinematic data between the dominant and non-dominant legs of healthy amateur footballers during single leg squat when the leg is moving. Annotations: * = p < 0.05; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in non–dominant leg; ↓ = decreased in non-dominant leg.

	Phase: Moving I					
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**			
sEMG GM:AL	1.4(0.22)	0.88(0.31)	0.17			
Sagittal hip	22.45(0.66)	21.37(0.5)	<0.01*↓	NB Flex +		
Coronal hip	0.01(0.87)	-2.44(0.72)	<0.05*↓	NB Add +		
Horizontal hip	-7.98(1.84)	-7.5(2.18)	0.87	NB IR +		
	Phase: Moving II			**Phase: Stance I**		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
sEMG GM:AL	1.39(0.17)	0.74(0.38)	0.13	1.67(0.16)	0.86(0.37)	0.06
Sagittal hip	1.19(0.75)	2.11(0.87)	0.43	26.64(1.14)	23.61(0.77)	<0.05*↓
Coronal hip	-2.03(0.68)	-1.19(0.69)	0.39	-2.03(1.3)	-3.75(0.96)	0.29
Horizontal hip	-1.74(0.5)	-0.73(0.63)	0.21	-9.73(1.93)	-7.89(2.09)	0.52
	Phase: Moving III			**Phase: Stance II**		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
sEMG GM:AL	0.59(0.15)	-0.09(0.26)	<0.05*↓	0.44(0.27)	-0.09(0.24)	0.15
Sagittal hip	43.43(3.19)	52.05(3.61)	0.097	68.99(3.97)	74.21(4.26)	0.39
Coronal hip	14.64(1.73)	14.4(3.3)	0.95	15.52(3.93)	9.23(3.59)	0.29
Horizontal hip	-1.83(3.49)	2.43(3.47)	0.4	-3.29(1.04)	1.88(4.71)	0.3
	Phase: Moving IV			**Phase: Stance III**		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
sEMG GM:AL	1.46(0.16)	0.75(0.23)	<0.05*↓	2.09(0.14)	0.81(0.38)	<0.01*↓
Sagittal hip	-40.74(4.1)	-54.04(4.3)	<0.05*↓	28.8(1.33)	22.48(0.96)	<0.01*↓
Coronal hip	-14.9(2.57)	-11.49(2.95)	0.4	-3.95(1.99)	-3.79(1.25)	0.95
Horizontal hip	2.23(3.26)	-3.2(3.73)	0.29	-7.99(1.91)	-8.53(2.22)	0.85

Table 42: Results from comparing surface electromyography and kinematic data between the dominant and non-dominant legs of healthy professional football players during single leg squat when the leg is moving. Annotations: * = p < 0.05; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in non–dominant leg; ↓ = decreased in non-dominant leg.

Phase: Moving I						
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**			
sEMG GM:AL	1.66(0.4)	0.87(0.38)	0.16			
Sagittal hip	30.8(1.35)	26.39(1.44)	<0.05*↓	NB Flex +		
Coronal hip	4.82(1.16)	-1.17(0.68)	<0.01*↓	NB Add +		
Horizontal hip	7.5(2.08)	-9.9(2.08)	<0.01*↓	NB IR +		
Phase: Moving II				**Phase: Stance I**		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
sEMG GM:AL	1.56(0.38)	1.35(0.55)	0.76	1.78(0.34)	1.51(0.54)	0.68
Sagittal hip	3.05(2.66)	5.86(3.34)	0.52	34.83(2.08)	31.81(2.82)	0.39
Coronal hip	-1.36(0.89)	-2.31(0.5)	0.36	4(1.34)	-3.27(0.65)	<0.05*↓
Horizontal hip	1.34(0.76)	0.97(1.48)	0.82	9.26(2.36)	-9.37(1.84)	<0.01*↓
Phase: Moving III				**Phase: Stance II**		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
sEMG GM:AL	0.48(0.27)	0.37(0.4)	0.83	0.57(0.36)	0.39(0.45)	0.76
Sagittal hip	38.96(2.69)	42.65(5.35)	0.54	71.85(2.77)	70.35(5.81)	0.82
Coronal hip	12.67(1.98)	3.91(2.39)	<0.05*↓	16.63(1)	1.27(2.97)	<0.01*↓
Horizontal hip	-1.85(1.38)	7.93(2.16)	<0.01*↑	6.25(2)	-2.5(3.31)	<0.05*↓
Phase: Moving IV				**Phase: Stance III**		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
sEMG GM:AL	1.21(0.22)	0.94(0.36)	0.53	1.43(0.4)	1.04(0.44)	0.51
Sagittal hip	-37.39(2.28)	-31.37(6.19)	0.37	35.98(1.73)	34.3(1.7)	0.49
Coronal hip	-14(2.02)	-4.82(3.7)	<0.05*↑	2.98(1.41)	-4.13(0.79)	<0.01*↓
Horizontal hip	1.89(1.38)	-6.53(0.97)	<0.01*↓	8.03(1.9)	-10.2(2.15)	<0.01*↓

Table 43: Results from comparing surface electromyography and kinematic data between the dominant and non-dominant legs of healthy Ultimate Frisbee players during single leg squat when the leg is moving. Annotations: * = p < 0.05; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in non–dominant leg; ↓ = decreased in non-dominant leg.

Phase: Moving I						
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**			
sEMG GM:AL	2.07(0.58)	2.68(0.52)	0.44			
Sagittal hip	20.45(1.52)	18.28(1.3)	0.29	NB Flex +		
Coronal hip	3.71(0.91)	0.52(0.91)	<0.05*↓	NB Add +		
Horizontal hip	0.04(0.75)	-5.84(3.17)	0.08	NB IR +		
Phase: Moving II				**Phase: Stance I**		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
sEMG GM:AL	1.38(0.38)	1.98(0.36)	0.27	1.91(0.39)	2.13(0.3)	0.66
Sagittal hip	1.13(1.34)	0.69(0.99)	0.79	22.58(2.3)	19.44(1.7)	0.28
Coronal hip	-5.35(1.71)	-4.23(1.06)	0.58	-0.67(1.56)	-3.84(0.83)	0.08
Horizontal hip	-1.36(1.96)	-3.02(1.29)	0.48	-1.36(1.74)	-9.46(2.3)	<0.01*↓
Phase: Moving III				**Phase: Stance II**		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
sEMG GM:AL	0.89(0.18)	1.1(0.28)	0.54	1.06(0.19)	1.16(0.31)	0.78
Sagittal hip	39.56(2.94)	42.93(1.38)	0.31	61.51(1.45)	62.73(1.51)	0.56
Coronal hip	14.64(1.51)	14.18(1.91)	0.85	15.14(1.97)	9.15(1.73)	<0.05*↓
Horizontal hip	-3.44(2.05)	6.59(1.29)	<0.01*↑	-2(1.24)	-1.48(3.06)	0.88
Phase: Moving IV				**Phase: Stance III**		
Measured leg	**Dominant**	**Non-dominant**	**Statistic (p)**	**Dominant**	**Non-dominant**	**Statistic (p)**
sEMG GM:AL	1.78(0.17)	1.62(0.23)	0.6	2.15(0.3)	2.12(0.29)	0.94
Sagittal hip	-36.78(2.97)	-39.75(0.98)	0.35	24.28(2.03)	21.84(1.51)	0.34
Coronal hip	-15.57(0.91)	-13.58(2)	0.37	-1.57(1.71)	-3.78(1.19)	0.3
Horizontal hip	-0.13(1,74)	-7.7(1.87)	<0.01*↓	-2.3(1.48)	-11.29(2.56)	<0.01*↓

Table 44: Results from comparing surface electromyography and kinematic data between the dominant and non-dominant legs of healthy filed hockey players during single leg squat when the leg is moving. Annotations: * = p < 0.05; sEMG = surface electromyography; arrows indicate the direction of difference; GM=gluteus medius; AL= adductor longus; Flex = flexion; Add = adduction; IR = internal rotation; SE = standard error; ↑ = increased in non–dominant leg; ↓ = decreased in non-dominant leg.

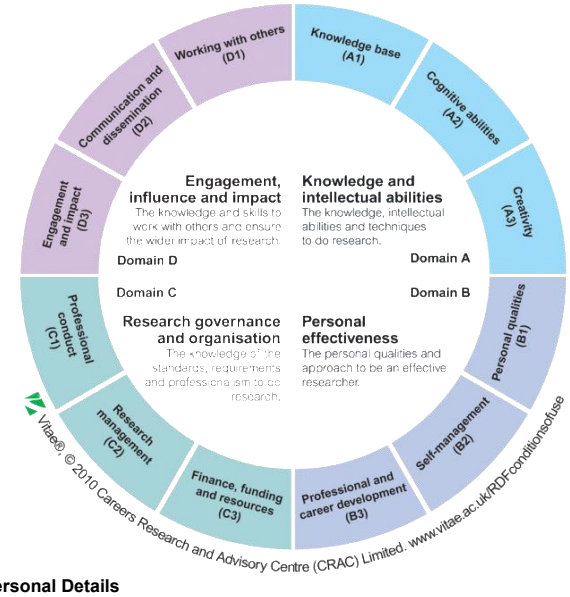
Appendix 3

Point based skills database

Miss PM Kloskowska (110624846)

Progress

Total target



Total

Personal Details

Full Name:	Paulina Maria Kloskowska	**Gender:**	Female
Username:	hhw819	**Email:**	p.kloskowska@qmul.ac.uk
Telephone:		**Mobile:**	07534133446
Enrolment Status:	R-E-E	**Programme:**	RRPF-QMWHRN1 PhD FT WHRI (Non-Clinical)
Course Name:	PhD FT William Harvey Research Institute (Non-Clinical)	**Award Code:**	RP
Start Date:	01-Nov-2011	**Expected End Date:**	01-Nov-2015
Route:	RSWHN		
Faculty:	Medicine and Dentistry	**School:**	William Harvey Research Institute
Department:	William Harvey Research Institute		

Supervisors

Title	Given Names	Last Name	Telephone	Email	Active
Dr	Dylan	Morrissey		d.morrissey@qmul.ac.uk	true
Prof	Bruce Lindsay	Kidd		b.l.kidd@qmul.ac.uk	true

Points Summary

Year	Type	Pts:	A	B	C	D	Total	Cap:	A	B	C	D	Total
0	*Teaching/demonstrating/marking/preparation*		0.0	2.5	0.0	2.5	5.0						
	Teaching sub-total		**0.0**	**2.5**	**0.0**	**2.5**	**5.0**						
	Year 0 Total (with caps applied)		0.0	2.5	0.0	2.5	5.0						
1st	*Conference Attendance (Half day)*		7.5	3.0	0.0	0.0	10.5						
	Conference Attendance (One day)		5.0	2.0	0.0	0.0	7.0						
	Conference attendance sub-total		**12.5**	**5.0**	**0.0**	**0.0**	**17.5**						
	CAPD Course		9.0	11.0	10.5	8.5	39.0						
	Course/event attendance sub-total		**9.0**	**11.0**	**10.5**	**8.5**	**39.0**						
	External funding application <£2,000		0.0	0.0	2.0	2.0	4.0						
	Funding application sub-total		**0.0**	**0.0**	**2.0**	**2.0**	**4.0**						
	Conference Presentation (oral/poster)		6.0	6.0	0.0	8.0	20.0						
	Ethical Approval for Study - Non-Clinical		0.0	0.0	2.0	0.0	2.0						
	Other sub-total		**6.0**	**6.0**	**2.0**	**8.0**	**22.0**						
	Mentoring/supervising of Project Student		8.0	4.0	0.0	8.0	20.0						
	Teaching/demonstrating/marking/preparation		0.0	6.5	0.0	6.5	13.0						
	Teaching sub-total		**8.0**	**10.5**	**0.0**	**14.5**	**33.0**						
	Year 1 Total (with caps applied)		35.5	32.5	14.5	33.0	115.5						

2nd	*Conference Attendance (Half day)*		2.5	1.0	0.0	0.0	3.5							
	Conference Attendance (Two days)		10.0	4.0	0.0	0.0	14.0							
	Conference attendance sub-total		**12.5**	**5.0**	**0.0**	**0.0**	**17.5**							
	CAPD Course		0.0	4.0	0.0	2.0	6.0							
	CILT Module 1		0.0	5.0	0.0	5.0	10.0							
	CILT Module 2		0.0	5.0	0.0	5.0	10.0							
	Course/event attendance sub-total		**0.0**	**14.0**	**0.0**	**12.0**	**26.0**							
	External funding application <£2,000		0.0	0.0	2.0	2.0	4.0							
	Funding application sub-total		**0.0**	**0.0**	**2.0**	**2.0**	**4.0**							
	Journal Club/Reading Group/lab meeting Presentation		3.0	0.0	0.0	1.0	4.0							
	Giving presentations sub-total		**3.0**	**0.0**	**0.0**	**1.0**	**4.0**							
	Conference Presentation (oral/poster)		12.0	12.0	0.0	16.0	40.0							
	Course/event Attendance		0.0	1.0	0.0	1.0	2.0							
	Ethical Approval for Study - Clinical		0.0	0.0	10.0	0.0	10.0							
	Ethical Approval for Study - Non-Clinical		0.0	0.0	4.0	0.0	4.0							
	Organising an event/seminar/conference		0.0	2.0	0.0	2.0	4.0							
	Presenting - internal to QM		1.0	1.0	0.0	2.0	4.0							
	Other sub-total		**13.0**	**16.0**	**14.0**	**21.0**	**64.0**							
	Mentoring/supervising of Project Student		6.0	3.0	0.0	6.0	15.0							
	Teaching/demonstrating/marking/preparation		0.0	11.5	0.0	11.5	23.0							
	Teaching sub-total		**6.0**	**14.5**	**0.0**	**17.5**	**38.0**							
	Year 2 Total (with caps applied)		**34.5**	**49.5**	**16.0**	**53.5**	**153.5**							
3rd	*Conference Attendance (Half day)*		2.5	1.0	0.0	0.0	3.5							
	Conference attendance sub-total		**2.5**	**1.0**	**0.0**	**0.0**	**3.5**							
	Ethical Approval for Study - Clinical		0.0	0.0	10.0	0.0	10.0							
	Presenting - internal to QM		1.0	1.0	0.0	2.0	4.0							
	Other sub-total		**1.0**	**1.0**	**10.0**	**2.0**	**14.0**							
	Teaching/demonstrating/marking/preparation		0.0	3.0	0.0	3.0	6.0							
	Teaching sub-total		**0.0**	**3.0**	**0.0**	**3.0**	**6.0**							
	Year 3 Total (with caps applied)		**3.5**	**5.0**	**10.0**	**5.0**	**23.5**							
Total	*Conference Attendance (Half day)*		12.5	5.0	0.0	0.0	17.5							
	Conference Attendance (One day)		5.0	2.0	0.0	0.0	7.0							
	Conference Attendance (Two days)		10.0	4.0	0.0	0.0	14.0							
	Conference attendance sub-total		**18.0**	**11.0**	**0.0**	**0.0**	**29.0**		**18.0**	**12.0**			**30.0**	
	CAPD Course		9.0	15.0	10.5	10.5	45.0							
	CILT Module 1		0.0	5.0	0.0	5.0	10.0							
	CILT Module 2		0.0	5.0	0.0	5.0	10.0							
	Course/event attendance sub-total		**9.0**	**25.0**	**10.5**	**20.5**	**65.0**							
	External funding application <£2,000		0.0	0.0	4.0	4.0	8.0				8.0	4.0	12.0	
	Funding application sub-total		**0.0**	**0.0**	**4.0**	**4.0**	**8.0**							
	Journal Club/Reading Group/lab meeting Presentation		3.0	0.0	0.0	1.0	4.0		6.0			6.0	12.0	
	Giving presentations sub-total		**3.0**	**0.0**	**0.0**	**1.0**	**4.0**							
	Conference Presentation (oral/poster)		18.0	18.0	0.0	24.0	60.0							
	Course/event Attendance		0.0	1.0	0.0	1.0	2.0							
	Ethical Approval for Study - Clinical		0.0	0.0	20.0	0.0	20.0							
	Ethical Approval for Study - Non-Clinical		0.0	0.0	6.0	0.0	6.0							
	Organising an event/seminar/conference		0.0	2.0	0.0	2.0	4.0							
	Presenting - internal to QM		2.0	2.0	0.0	4.0	8.0							
	Other sub-total		**20.0**	**23.0**	**26.0**	**31.0**	**100.0**							
	Mentoring/supervising of Project Student		8.0	4.0	0.0	8.0	20.0		8.0	4.0		8.0	20.0	
	Teaching/demonstrating/marking/preparation		0.0	15.0	0.0	15.0	30.0			15.0		15.0	30.0	
	Teaching sub-total		**8.0**	**19.0**	**0.0**	**23.0**	**50.0**							
	Total (with caps applied)		**58.0**	**78.0**	**40.5**	**79.5**	**256.0**							
	Target		60.0	20.0	15.0	30.0	210.0							

Pending Activities

Nothing found to display

Activity Record

Type	Code	Title	Provider	From	To	Hours	A	B	C	D	Total
Teaching/demonstrating/marking/preparation		Biomechanics - Lower Limb	CSEM	24-Jan-2011 14:30	24-Jan-2011 17:00	2.5	0.0	1.2	0.0	1.2	2.5
Teaching/demonstrating/marking/preparation		Biomechanics - students presentations	CSEM	31-Jan-2011 14:30	31-Jan-2011 17:00	2.5	0.0	1.2	0.0	1.2	2.5
Mentoring/supervising of Project Student		Mentoring/supervising a student's project (OR)	CSEM	01-Nov-2011 08:00	29-Jun-2012 09:00	0.0	2.0	1.0	0.0	2.0	5.0
Conference Attendance (Half day)		Attending lab meetings	Dr Dylan Morrissey	02-Nov-2011 08:30	06-Mar-2013 08:30	0.0	2.5	1.0	0.0	0.0	3.5
		Ethical approval - case control		08-Nov-	08-Jan-						

Ethical Approval for Study - Non-Clinical		study	QMUL	2011 09:00	2012 09:00	0.0	0.0	0.0	2.0	0.0	2.0
Conference Attendance (Half day)		Attending lab meetings	Dr Dylan Morrissey	09-Nov-2011 08:30	06-Mar-2013 08:30	0.0	2.5	1.0	0.0	0.0	3.5
Conference Attendance (One day)		CSEM-DJO Education Meeting	CSEM	09-Dec-2011 09:00	09-Dec-2011 17:00	0.0	5.0	2.0	0.0	0.0	7.0
CAPD Course	R244	Cafe Scientifique	The Learning Institute	14-Dec-2011 18:00	14-Dec-2011 20:30	0.0	0.0	1.0	0.0	0.0	1.0
Mentoring/supervising of Project Student		Mentoring/supervising a student's project (JC)	CSEM	02-Jan-2012 09:00	17-Aug-2012 09:00	0.0	2.0	1.0	0.0	2.0	5.0
CAPD Course	R160	Writing for Publication in Refereed Journals	The Learning Institute	23-Jan-2012 14:00	23-Jan-2012 17:00	0.0	0.0	0.0	0.0	3.0	3.0
Mentoring/supervising of Project Student		SMentoring/supervising a student's project (JA)	CSEM	08-Feb-2012 08:00	17-Aug-2012 09:00	0.0	2.0	1.0	0.0	2.0	5.0
Conference Presentation (oral/poster)		Football Association Medical Society conference - groin and hamstrings	Football Association Medical Society	13-Feb-2012 18:50	13-Feb-2012 21:30	0.0	3.0	3.0	0.0	4.0	10.0
Teaching/demonstrating/marking/preparation		Rehabilitation - lower limb	CSEM	21-Feb-2012 13:30	21-Feb-2012 15:30	2.0	0.0	1.0	0.0	1.0	2.0
Teaching/demonstrating/marking/preparation		Rehabilitation - upper limb	CSEM	28-Feb-2012 14:30	28-Feb-2012 18:00	3.5	0.0	1.8	0.0	1.8	3.5
Teaching/demonstrating/marking/preparation		Rehabilitation - spine	CSEM	06-Mar-2012 14:30	06-Mar-2012 18:00	3.5	0.0	1.8	0.0	1.8	3.5
Teaching/demonstrating/marking/preparation		Biomechanics - spine and pelvis	CSEM	13-Mar-2012 14:30	13-Mar-2012 16:30	2.0	0.0	1.0	0.0	1.0	2.0
Conference Attendance (Half day)		ootball Association Medical Society conference - Achilles problems in football	ootball Association Medical Society	16-Apr-2012 18:50	16-Apr-2012 21:30	0.0	2.5	1.0	0.0	0.0	3.5
CAPD Course	R175	PhD Induction Day 1	The Learning Institute	19-Apr-2012 09:30	19-Apr-2012 17:00	0.0	1.0	2.5	3.5	0.0	7.0
External funding application <£2,000		Boehringer Ingelheim Students Grant	Boehringer Ingelheim Foundation	01-May-2012 09:00	01-Jun-2012 09:00	0.0	0.0	0.0	1.0	1.0	2.0
CAPD Course	R121	Managing Your PhD	The Learning Institute	08-May-2012 10:00	08-May-2012 16:00	0.0	0.0	3.0	3.0	0.0	6.0
CAPD Course	R182	Presenting Your Research to an Audience (Day 1)	The Learning Institute	14-May-2012 10:00	14-May-2012 13:00	0.0	0.0	1.5	0.0	1.5	3.0
CAPD Course	DELUEL25p	How to use Endnote for Medicine and the Sciences	The Learning Institute	15-May-2012 14:00	15-May-2012 16:00	0.0	2.0	0.0	0.0	0.0	2.0
Conference Presentation (oral/poster)		WHRI Annual Research Review oral presentation	WHRI	10-Jul-2012 09:00	10-Jul-2012 16:00	0.0	3.0	3.0	0.0	4.0	10.0
Mentoring/supervising of Project Student		Mentoring/supervising a student's project (WM)	CSEM	10-Sep-2012 09:00	24-Jun-2013 09:00	0.0	2.0	1.0	0.0	2.0	5.0
External funding application <£2,000		ISB Brazil 2013 Student Travel Grant	International Society of Biomechanics	01-Oct-2012 09:00	01-Nov-2012 09:00	0.0	0.0	0.0	1.0	1.0	2.0
CAPD Course	ARP1	Introduction to Leadership & Managing Teams (Day 1)	The Learning Institute	08-Oct-2012 10:00	08-Oct-2012 16:00	0.0	0.0	2.0	1.0	3.0	6.0
Teaching/demonstrating/marking/preparation		Lower limb anatomy	CSEM	12-Oct-2012 11:00	12-Oct-2012 13:00	2.0	0.0	1.0	0.0	1.0	2.0
CAPD Course	RW202	WISE - Women in Leadership	The Learning Institute	24-Oct-2012 16:00	24-Oct-2012 18:00	0.0	0.0	1.0	0.0	1.0	2.0
CAPD Course	R209	Critical Thinking	The Learning Institute	29-Oct-2012 10:00	29-Oct-2012 16:00	0.0	6.0	0.0	0.0	0.0	6.0
CAPD Course	R137	Postgraduate Funding: Considering the Alternatives	The Learning Institute	31-Oct-2012 14:00	31-Oct-2012 17:00	0.0	0.0	0.0	3.0	0.0	3.0
Teaching/demonstrating/marking/preparation		Upper limb anatomy	CSEM	02-Nov-2012 11:30	02-Nov-2012 13:30	2.0	0.0	1.0	0.0	1.0	2.0
Ethical Approval for Study - Non-Clinical		Ethical approval - case control study	QMUL	08-Nov-2012 09:00	15-Feb-2013 09:00	0.0	0.0	0.0	2.0	0.0	2.0
CAPD Course	R243	Negotiating and Influencing Skills	The Learning Institute	12-Nov-2012 14:00	12-Nov-2012 17:00	0.0	0.0	3.0	0.0	0.0	3.0
Teaching/demonstrating/marking/preparation		Spine anatomy	CSEM	13-Nov-2012 13:00	13-Nov-2012 14:30	1.5	0.0	0.8	0.0	0.8	1.5
External funding application <£2,000		QMUL Postrgraduate Travel Grant	QMUL	03-Dec-2012	15-Jan-2013	0.0	0.0	0.0	1.0	1.0	2.0

				09:00	09:00						
Conference Attendance (Half day)		Cafe Scientifique	Jo Cordy	05-Dec-2012 18:00	05-Dec-2013 21:00	0.0	2.5	1.0	0.0	0.0	3.5
CILT Module 1		CILT Module 1 completion	LI	03-Jan-2013 15:00	03-Sep-2013 09:00	0.0	0.0	5.0	0.0	5.0	10.0
Teaching/demonstrating/marking/preparation		Biomechanics - lower limb	CSEM	22-Jan-2013 14:30	22-Jan-2013 17:00	2.5	0.0	1.2	0.0	1.2	2.5
Teaching/demonstrating/marking/preparation		Rehabilitation - lower limb	CSEM	25-Jan-2013 09:00	25-Jan-2013 11:30	2.5	0.0	1.2	0.0	1.2	2.5
Teaching/demonstrating/marking/preparation		Dance Medicine lecture	CSEMM	05-Feb-2013 09:00	05-Feb-2013 11:00	2.0	0.0	1.0	0.0	1.0	2.0
Teaching/demonstrating/marking/preparation		Rehabilitation - upper limb	CSEM	15-Feb-2013 09:00	15-Feb-2013 11:30	2.5	0.0	1.2	0.0	1.2	2.5
Ethical Approval for Study - Non-Clinical		Ethical approval - longitudinal study	QMUL	15-Feb-2013 09:00	15-Feb-2013 17:00	0.0	0.0	0.0	2.0	0.0	2.0
Teaching/demonstrating/marking/preparation		Anatomy practical: Lower limb	CSEM	25-Feb-2013 15:00	25-Feb-2013 17:00	2.0	0.0	1.0	0.0	1.0	2.0
Teaching/demonstrating/marking/preparation		Dance Medicine lecture	CSEM	05-Mar-2013 09:00	05-Mar-2013 11:00	2.0	0.0	1.0	0.0	1.0	2.0
External funding application <£2,000		QMPGRF Travel Grant successfully obtained	QMUL	19-Mar-2013 09:00	19-Mar-2013 11:00	0.0	0.0	0.0	1.0	1.0	2.0
Journal Club/Reading Group/lab meeting Presentation		Attending Lab Meetings	Dr Dylan Morrissey, CSEM	27-Mar-2013 08:30	19-Mar-2014 10:00	0.0	3.0	0.0	0.0	1.0	4.0
Course/event Attendance		Planning for difficult conversations	LI	10-May-2013 13:00	10-May-2013 15:00	0.0	0.0	1.0	0.0	1.0	2.0
Conference Attendance (Two days)		18th Annual Congress of European College of Sports Science	European College of Sports Science	26-Jun-2013 08:00	29-Jun-2013 20:00	0.0	10.0	4.0	0.0	0.0	14.0
Conference Presentation (oral/poster)		18th Annual Congress of European College of Sports Science- oral presentation	European College of Sports Science	28-Jun-2013 11:00	28-Jun-2013 12:00	0.0	3.0	3.0	0.0	4.0	10.0
Conference Presentation (oral/poster)		WHRI Annual Research Review	WHRI	04-Jul-2013 10:00	04-Jul-2013 18:00	0.0	3.0	3.0	0.0	4.0	10.0
Presenting - internal to QM		RIP presentation	QMUL	10-Jul-2013 13:00	10-Jul-2013 14:00	0.0	1.0	1.0	0.0	2.0	4.0
Mentoring/supervising of Project Student		Supervising iBSc student	CSEM, WHRI	02-Sep-2013 09:00	25-Jul-2014 09:00	0.0	2.0	1.0	0.0	2.0	5.0
CILT Module 2		CILT Module 2 completion	LI	02-Sep-2013 09:00	14-Mar-2014 14:00	0.0	0.0	5.0	0.0	5.0	10.0
Mentoring/supervising of Project Student		Supervising iBSc student	CSEM, WHRI	02-Sep-2013 09:00	24-Jul-2014 09:00	0.0	2.0	1.0	0.0	2.0	5.0
Conference Presentation (oral/poster)		QMUL in motion Conference	CSEM, WHRI	05-Sep-2013 09:00	05-Sep-2013 17:00	0.0	3.0	3.0	0.0	4.0	10.0
Conference Presentation (oral/poster)		CSEM Annual Scientific Conference presentation	CSEM, WHRI	06-Sep-2013 09:00	06-Sep-2013 18:00	0.0	3.0	3.0	0.0	4.0	10.0
Organising an event/seminar/conference		Organising CSEM Annual Scientific Conference	CSEM, WHRI	06-Sep-2013 09:00	06-Sep-2013 18:00	0.0	0.0	2.0	0.0	2.0	4.0
Mentoring/supervising of Project Student		Supervising MSc student	CSEM, WHRI	24-Sep-2013 09:00	27-Feb-2015 09:00	0.0	2.0	1.0	0.0	2.0	5.0
Teaching/demonstrating/marking/preparation		Anatomy Lecturing for iBSc students	CSEM, WHRI	27-Sep-2013 09:00	18-Dec-2013 12:00	6.0	0.0	3.0	0.0	3.0	6.0
Ethical Approval for Study - Clinical		Obtaining non-clinical ethics - successful	QMUL	01-Oct-2013 09:00	31-Oct-2013 09:00	0.0	0.0	0.0	10.0	0.0	10.0
CAPD Course	A206	Detecting and Deterring Plagiarism	Centre for Academic and Professional Development	24-Oct-2013 16:00	24-Oct-2013 17:00	0.0	0.0	1.0	0.0	2.0	3.0
Teaching/demonstrating/marking/preparation		Teching Biomechanics and Rehabilitation to iBSc students	CSEM, WHRI	19-Nov-2013 09:00	03-Mar-2014 12:00	6.0	0.0	3.0	0.0	3.0	6.0
Ethical Approval for Study - Clinical		Obtaining clinical (NHS) ethics through IRAS form -in progress	NHS	03-Jan-2014 09:00	24-Mar-2014 10:00	0.0	0.0	0.0	10.0	0.0	10.0
Conference Attendance (Half day)		WHRI New Year Celebration Conference	WHRI	31-Jan-2014 13:00	31-Jan-2014 18:00	0.0	2.5	1.0	0.0	0.0	3.5
Presenting - internal to QM		Internal meeting presentation	CSEM, WHRI	05-Mar-2014 08:00	05-Mar-2014 12:00	0.0	1.0	1.0	0.0	2.0	4.0

Appendix 4

Letter of ethical approval for the observational study.

Queen Mary, University of London
Room E16
Queen's Building
Queen Mary University of London
Mile End Road
London E1 4NS

Queen Mary Research Ethics Committee
Hazel Covill
Research Ethics Administrator
Tel: +44 (0) 20 7882 2207
Email: h.covill@qmul.ac.uk

Dr Dylan Morrissey
Department of Sports Medicine
Mile End Hospital
Bancroft Road
London E1 4NS

27th October 2015

To Whom It May Concern:

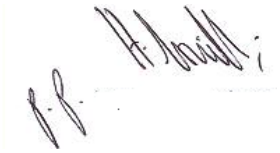
Re: QMREC2011/07 – Human performance measurement – a generic ethics application.

This is to confirm that the following study was agreed under the above ethical approval:

The biomechanical determinants of lumbo-pelvic muscle imbalance in footballers with adductor-related groin pain.

Date of approval.
This was noted and approved on the 1st March 2012.

Yours faithfully



Ms Elizabeth Hall – QMREC Chair.

Appendix 5

Approved ethical application for the observational study including the Participant Information Sheet and the Informed Consent.

For Office Use Only:
Rec Reference ……
Date received: ……

Queen Mary University of London

Application form – Queen Mary Research Ethics Committee

1 Name and email address of applicant
Miss Paulina Kloskowska MSc, Centre for Sports and Exercise Medicine, WHRI Email: p.kloskowska@qmul.ac.uk
2 Title of study

The biomechanical determinants of lumbo-pelvic muscle imbalance in Field Hockey players with adductor-related groin pain.
3 Investigators
Miss Paulina Kloskowska MSc, BSc Dr Dylan Morrissey PhD MSc MMACP MCSP Professor Roger Woledge, Professor Emeritus of Experimental Physiology
4 Proposed timetable
Preferred start date: October 2012 Projected date of completion: September 2014
5 Other organisations involved
Professional and amateur athletes The athletes will only be recruited once letters of approval have been granted.
6 Other REC approval
N/A
7 Nature of project e.g. undergraduate, postgraduate

The project is a postgraduate student research for the fulfilment of an intercalated BSc project. It will build the first part of the PhD project and develop collaboration between the Centre for Sports and Exercise Medicine and the sports clubs. Students who will work on this project include

- Miss Laura Middleton, BSc SEM student
- Miss Charlotte Hillary, BSc SEM student

8 Purpose of the research

The purpose of the study is to investigate muscle activation and kinematics during simple movement tests commonly used in the assessment of subjects with chronic groin pain and determine whether any systematic differences in electromyographically detected muscle onsets exist in muscle activation or movement patterns between:

a) symptomatic and non symptomatic sides

b) controls and subjects

c) dominant and non-dominant leg

The tests to be examined are:

- One Leg Standing – the ability to stand unsupported on one leg and lift the other leg to 90 degrees of hip flexion (Hungerford et al 03)

- Active Straight Leg Raise – the ability to lift one leg approximately 60 degrees from the supporting surface. Measurements of pain and effort are scored for all subjects (Mens et al 99)

- One Leg Squat – the ability to stand on one leg and perform a squat on the supporting leg (Crossley et al. 2011)

- Bent Knee Fall Out – the ability of the subject to abduct and externally rotate the hip joint from a position of hip and knee flexion (crook lying) (Sahrmann 98)

- other, similar, tests as indicated specific to the sport

The overall null hypothesis is that subjects with groin pain due to either articular or muscular presentations have no difference in movement patterns or muscle timing with respect to control subjects.

There are a range of movement or subject group alternative hypotheses including:

- that subjects with chronic groin pain will have an altered pattern of movement on the symptomatic side compared with the non-symptomatic side
- that subjects with chronic groin pain will have an altered ratio of hip adductor to hip abductor muscle activity compared to normal subjects
- that subjects with chronic groin pain will have altered ratios of hip adductor to hip extensor muscle activity
- that subjects with chronic groin pain will have an altered ratio of hip flexor to hip extensor muscle activity
- that the effect of dominance affects the muscle activation and muscle ratio in symptomatic subjects

Background:

Chronic groin pain is a common problem in football code athletes. Among professional soccer players the incidence of groin pain accounts up for 18% per year (Homlich 2007). There are many disorders potentially responsible for that symptom, including referred pain of thoracolumbar origin, hip arthrosis, hernia and sports hernia (Holmich 2007), pelvic nerve entrapments (Anderson et al 2001), urological diseases (Fon et al 2000), and many other, few of which are well investigated and described.

According to previous studies (Holmich 2007, Verral et al 2005) one of the most common clinical entities causing groin pain are adductor-related disorders (ARGP). This non-specific diagnosis contains wide range of alterations affecting adductor muscles and consequently, the adduction movement (Ibrahim et al 2007). The possible causes of ARGP include pathology of muscles, tendons, joints or bones. The variety of probabilities potentially responsible for this syndrome continues to present a significant diagnostic challenge (Holmich 2007, Fricker 1997).

Although there have been a number of studies trying to specify the initial cause of ARGP (Holmich 2007, Mens et al 2006, Verral 2001), few of them focus on the effect it has on the muscles around the groin and pelvic region. Several authors associated a decreased hip joint range of movement (ROM) with an increased risk of ARGP (Ibrahim et al 2007, Kettunen et al 2000, Gupta et al 2004), while others highlight the relationship between the features of the muscles responsible for core stability (e.g. transversus abdominis) and pathology of adductor muscles (Mens et al 2006). The outcomes, though, do not show significant relation in any of these studies – thus it appears to be rational to continue research in this field, which will help to uncover other relation between pelvic girdle muscles in ARGP.

Morrissey et al. (in review) carried out research showing the differences between the gluteus medius (GM) to adductor longus (AL) ratio in football players suffering from adductor-related groin pain compared to a matched control group (Figure 1). The data were collected during both moving and stance phase of standing hip flexion and show a significant change of the activation ratio between examined muscles in subjects with groin pain. The data suggests that there may be a common pattern concerning the electromyographic determinants in patients suffering and recovering from groin pain, which shows a significant decrease of GM:AL ratio in patients suffering from groin pain. Further analysis shows it is mainly due to a significant decrease of GM activation.

However, these outcomes have not been analysed in comparison to the kinematic determinants of the analysed movements. As well as EMG results, kinematic outcomes are also expected to show differences between participants with ARGP and healthy controls.

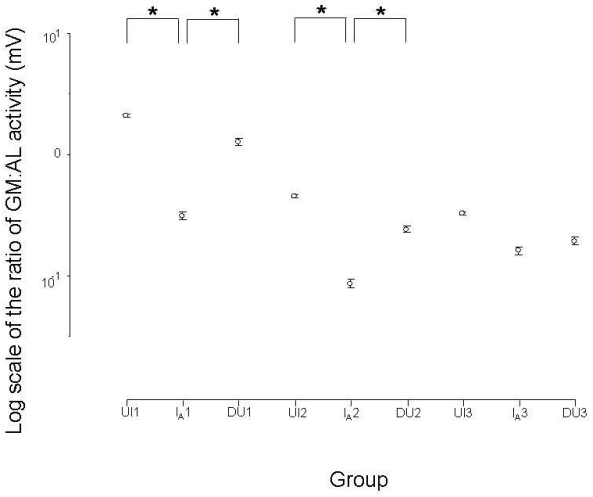


Figure 1: The ratio of GM:AL activity (mV) of injured and uninjured sides in ARGP patients and controls.

During these studies a number of data concerning ARGP will be collected. They will help to develop a quantifiable clinical test procedure and associated database to diagnose and assess the muscle imbalance occurring in ARGP. The outcomes would not only be useful to plan the accurate rehabilitation and proper treatment of this group of patients, but would also provide new insight into the mechanisms underlying ARGP.

References

Hölmich P. Long-standing groin pain in sportspeople falls into three primary patterns, a ‘‘clinical entity’’ approach: a prospective study of 207 patients. Br J Sports Med. 2007;41:247–52

Anderson K, Strickland SM, Warren R. Hip and groin injuries in athletes. Am J Sports Med. 2001;29(4):521-33

Fon LJ, Spence RA. Sportsman's hernia. Br J Surg 2000;87:545-52

Verrall GM, Slavotinek JP, Barnes PG, Fon GT. Description of pain provocation tests used for the diagnosis of sports-related chronic groin pain: relationship of tests to defined clinical (pain and tenderness) and MRI (pubic bone marrow oedema) criteria. Scand J Med Sci Sport. 2005;15:36-42.

Ibrahim A, Murrell GA, Knapman P. Adductor strain and hip range of movement in male professional soccer players. J Orthopaedic Surgery 2007;15(1):46-9

Fricker PA. Management of groin pain in athletes. Br J Sports Med.1997;31:97-101.

Mens J, Inklaar H, Koes BW, Stam HJ. A new view on adduction-related groin pain. Clin J Sport Med. 2006;16(1):15-9

Verral GM, Slavotinek JP, Fon GT. Incidence of pubic bone marrow oedema in Australian rules football players: relation to groin pain. Br J Sports Med. 2001;35(1):28-33

Kettunen JA, Kujala UM, Räty H, Videman T, Sarna S, Impivaara O, Koskinen S. Factors associated with hip joint rotation in former elite athletes. Br J Sports Med. 2000;34(1):44-8.

Gupta, A., Fernihough, B., Bailey, G., Bombeck, P., Clarke, A., and Hopper, D. An evaluation of hip external rotation strength and range of motion differences between female dancers and non-dancers. Br J Sports Med. 2004;38(6):778-83

Morrissey D, Graham J, Screen H, Sinha A, Small C, Twycross-Lewis R, Woledge R. Coronal plane hip muscle activation in football code athletes with chronic adductor groin strain injury during standing hip flexion. In press.

9 Study design, methodology and data analysis

Each potential participant will be provided with a consent form, information sheet and an explanation of the procedure before participating in the study.

Each subject will be asked to complete a written screening questionnaire to define their lower back, pelvic and groin injury status that has impacted on their ability to train or play in the last twelve months. The questionnaire will comprise of two parts; characterisation of participants and self-reported injury history.

Characterisation includes:

Biological data - age, age at puberty, height, weight

Sporting career – age at which commenced specialist sport, amount of playing / practice time, position played, level of competition.

Injury will be defined as any pain or dysfunction in the anterior hip and groin region, of at least 1 month's duration, that impacted the ability to do physical activity during the study period and/or the last twelve months. Each sportsman / woman will include a description and location of the injury and will be required to indicate their injury location on a body chart (attached). The duration of symptoms must have been at least 1 month.

It is at this point that consent will be taken and the questionnaire gone through with the subject. At this time, additional data will be collected on:

- Family history
- Past medical history
- Playing load – past / preseason / current
- Injury – onset / presence of prodromic symptoms
- Pain area and behaviour

A physical examination will then be undertaken to determine appropriate inclusion criteria as well as other associated features that may identify subgroups in analysis of the data. This will include:

- Spinal range of motion and manual segmental examination

- Hip joint range of motion and pain provocation tests

- SIJ Kinetic tests

- Isometric hip adduction force and symptom provocation

- Thomas test – muscle length and strength

- Squeeze test – resisted adduction (0 / 60 / 90 degrees of hip flexion)

- Unilateral Resisted Abduction test (30 degrees of hip flexion)

- Bilateral Resisted Abduction test (30 degrees of hip flexion)

- Palpation of the adductor tendons / pubic insertion

- SIJ passive motion analysis

- Response to ASLR

- Hip quadrant testing

- Labral “grind” test

Subjects will then undergo motion analysis measurements using non-invasive 3-dimensional infra-red cameras (Codamotion cx1, Charnwood Dynamics, Loughborough, UK) and force plates (Kistler, USA) - using standard marker placement protocols for the spine, pelvis and lower limb. In addition to motion analysis, electromyographic (EMG) readings will be taken using the wireless surface EMG device (Noraxon Telemyo 2400T, Scottsdale, Arizona, USA) of the following muscles :

- Hip adductors - 2 channels – Adductor longus and magnus
- Gluteus medius
- Gluteus maximus
- Abdominals – external oblique
- Rectus femoris
- Biceps femoris

Testing will take place in the Human Performance Laboratory at QMUL and should take no longer than 90 minutes. Only one test per participant will be required.

Simultaneous measurements of muscle strength will also be made using a hand held dynamometer.

Data analysis

Based on the results of the questionnaire and the physical examination, two sub-groups will be defined : one with a presentation of chronic groin pain of soft tissue origin and a control group. Controls will be age, height and activity matched.

Analysis of collected data for defined sub-groups will be done using a mathematical model written in MatLAb (Mathworks, USA).

Statistical analysis

The data will be assessed for normality and appropriate group comparison analysis undertaken accordingly. The power of the study will be 80% with statistical significance set at $p < 0.05$.

10 Participants to be studied

Number of participants – approximately 10 in each group

Lower age limit – 18

Upper age limit – 70

Sample Size

Based on the study by Cowan et al (2004) showing a difference in abdominal muscle activation of 45ms with a pooled standard deviation of 30ms, a sample size of 14 at a power of 80% and alpha error level of 5% is required in each group. We have allowed for an extra 6 subjects in case of data loss, unexpected sub-groups and to detect smaller significant differences.

11 Selection criteria

These inclusion / exclusion criteria reflect those used in previous studies examining potential mechanisms for chronic groin pain (Cowan et al 04, Holmich et al 99)

Chronic groin pain group

Inclusion criteria

- 18 yr of age or older
- Playing elite- or sub elite-level sport
- Activity-related, insidious onset groin pain that has been present for at least 4 weeks.

At least two of

1. Tenderness on palpation of either the adductor tendons, their insertion onto the pubic bone, or the pubic symphysis
2. Presence of groin pain during active hip adduction against resistance at the time of assessment – squeeze test
3. Presence of groin pain during active hip flexion against resistance at the time of assessment

4. Presence of groin pain during passive hip abduction (stretch)
5. Presence of groin pain during passive hip extension (stretch)
6. Labral grind test positive
7. Flexion adduction (FAbER) test positive
8. Proven muscular pathology on previously completed imaging tests

Exclusion criteria

- Groin pain that commenced as a result of an acute incident without prodromic symptoms
- Groin pain that commenced as a result of the articular pathology
- Surgery to the lower abdominal, hip or groin region
- Frank inguinal hernia
- Lumbar pain that predominates on physical examination
- Neurological symptoms
- Systemic disease
- Significant psychological condition

Control group

Inclusion criteria

- Over 18 years of age
- Playing elite- or sub elite-level sport

Exclusion criteria

- History of groin pain
- Surgery to their lower abdominal, hip, or groin region, or a frank inguinal hernia
- History of lumbar pain in the past year
- Neurological symptoms
- Systemic disease
- Significant psychological condition

.

12 Recruitment (including incentives and compensation)

Participants will be approached indirectly through an advert provided by email to the medical staff of various sports clubs. The medical staff at various clubs will have full details of the study and will have consented to provide this information to their academy teams.

The advert will include details of the research project; its purpose, objective and that participants are required. The advert will reflect the affiliation with QMUL and that the study has the full backing of the football clubs. This advert will be subject to consideration by Dr Morrissey prior to use.

Medical staff at the clubs will be asked to discuss the study with players and provide them with an information sheet detailing the study and the requirements of each participant, along with a consent form.

A contact telephone number will also be enclosed so that any questions or queries potential participants might have can be addressed through a follow up telephone interview with Dr Dylan Morrissey or Paulina Kloskowska.

If a player is happy to participate, details will be collected by the club's medical staff or Paulina Kloskowska. It is anticipated that a group of players will be tested at similar times and in the presence of the club physiotherapy staff. Consent will be documented at the time of testing.

Travelling expenses to a maximum of ten pounds per person will be given. The assessments will all be undertaken at the HPL, QMUL. As an incentive, each participant and their medical team will be offered an explanation of the findings. No financial or other reward will be given to participants.

13 Ethical considerations and risks to participants

The main ethical issue will be the need to ensure voluntary participation from acaedemy players within a club environment. As the clubs will not be incentivised in any way to participate, it is not anticipated that any form of coercion will occur.

The need to remove sufficient clothing to attach the motion markers to the torso and legs is also another consideration. In order for the EMG electrode pads to be well-adhered, small areas of the skin will need to be shaved and cleaned. Privacy in the data collection areas will be maximised and subjects will be encouraged to bring suitable clothing. The presence of the club's

physiotherapy staff should also ensure that players are confident in the testing being undertaken.

Full and informed consent will be obtained from each participant before entering the study. The participant will be given an information sheet detailing what the study entails and what is required of the participant. This will also be reiterated in person with the opportunity to ask any questions about the project.

Each participant will be protected from harm or injury with all measurements being undertaken in a controlled manner.

Each participant will have the right to withdraw from the research at any time, and for whatever reason.

14 Confidentiality, anonymity, and data storage

Each participant's confidentiality and privacy will be assured by the use of a code which will be characterized by each participant's initials and the date of the test. Each participant will be allocated their code on consenting to the study and each coded participant will also have the date that the assessment will be undertaken. This will ensure each participant's anonymity. Only the QMUL research team involved in the investigation will have access to the corresponding name/number data and any other personal information, which will be securely held on a separate server, requiring a password. The data will be securely stored, easily retrievable and well indexed.

Sensitive data will be stored on password protected server databases to which only the investigators will have access too, all such data will be handled in accordance with the provisions of the Data protection Act 1998.

15 Information for participants

Headed Paper

REC Protocol Number.**…….**

YOU WILL BE GIVEN A COPY OF THIS INFORMATION SHEET

We would like to invite you to participate in this research project. You should only participate if you want to; choosing not to take part will not disadvantage

you in any way. Your decision will not affect your access to treatment or services. Before you decide whether you want to take part, it is important for you to understand why the research is being done and what your participation will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information. If you do decide to take part, please let us know beforehand if you have been involved in any other study during the last year.

If you volunteer to take part you will be invited to meet the study team at the Human Performance Laboratory, Queen Mary's University of London. After answering any questions you may have, you will be asked to fill in a questionnaire. The first part will ask for you for personal details such as age, height, weight, other sporting activities past and present and amount of playing time. The second part will deal with self-reported injury, particularly pain or injury in the groin area.

A short physical examination will be undertaken to determine your suitability to participate.

We will then attach several electrodes that will be used to measure the electrical activity in your muscles. These electrodes do not carry any electricity into your body. These electrodes are self adhesive and designed to stick to skin and be removed easily and painlessly.
This will be followed by the application of 20 small infra-red motion sensors to your trunk and legs with medical grade double sided sticky tape. This will require you to wear clothing that reveals the skin of the lumbar spine, shoulder blades and legs. A pair of close fitting shorts would be ideal. We can provide these if necessary.
We will then make some measurements of your movement patterns during several movement tasks while standing or lying on a force plate that measures weight transfer. The total time required to attach markers and marker boxes and to measure the movement should be about one hour.

We do not anticipate any risk or discomfort by participating in this study. In order to participate in the study you will be asked to meet certain study inclusion/exclusion criteria.
If you participate in this study you will be given an identification number and so will remain completely anonymous throughout. All personal information linking you to this number will be kept separately and stored securely on a database server to which only I will have access to. All information will be handled in accordance with the provisions of the data protection act 1998 and your confidentiality assured.

My correspondence details are included in this application if you wish to contact me, to obtain further details or to ask any questions regarding the study:

Paulina Kloskowska
p.kloskowska@qmul.ac.uk 07428147932
Centre for Sport and Exercise Medicine
Mile End Hospital
Bancroft Road
LONDON E1 4DG

Alternatively, you can contact:
Dr Dylan Morrissey
Centre for Sport and Exercise Medicine
Mile End Hospital
Bancroft Road
LONDON E1 4DG
And on d.morrissey@qmul.ac.uk 02082238839

16 Consent

Please complete this form after you have read the Information Sheet and/or listened to an explanation about the research.

Title of Study: The biomechanical determinants of lumbo-pelvic muscle imbalance in footballers with adductor-related groin pain.

Queen Mary Research Ethics Committee Ref: _____

. • Thank you for considering taking part in this research. The person organizing the research must explain the project to you before you agree to take part.

. • If you have any questions arising from the Information Sheet or explanation already given to you, please ask the researcher before you decide whether to join in. You will be given a copy of this Consent

Form to keep and refer to at any time.

. • I understand that if I decide at any other time during the research that I no longer wish to participate in this project, I can notify the researchers involved and be withdrawn from it immediately.

. • I consent to the processing of my personal information for the purposes of this research study. I understand that such information will be treated as strictly confidential and handled in accordance with the provisions of the Data Protection Act 1998.

Participant's Statement:

I _____ agree that the research project named above has been explained to me to my satisfaction and I agree to take part in the study. I have read both the notes written above and the Information Sheet about the project, and understand what the research study involves.

Signed: Date:

Investigator's Statement:

I _____ confirm that I have carefully explained the nature, demands and any foreseeable risks (where applicable) of the proposed research to the volunteer.

Signed: Date:

17 Signature of applicant and authorising signatories.

Appendix 6

Letter of ethical approval for the longitudinal study.

Queen Mary, University of London
Room E16
Queen's Building
Queen Mary University of London
Mile End Road
London E1 4NS

Queen Mary Research Ethics Committee
Hazel Covill
Research Ethics Administrator
Tel: +44 (0) 20 7882 2207
Email: h.covill@qmul.ac.uk

Dr Dylan Morrissey
Department of Sports Medicine
Mile End Hospital
Bancroft Road
London E1 4NS

27th October 2015

To Whom It May Concern:

Re: QMREC2011/07 – Human performance measurement – a generic ethics application.

This is to confirm that the following study was agreed under the above ethical approval:

The biomechanical determinants of lumbo-pelvic muscle imbalance in athletes after adductor strain injury along the process of rehabilitation – a longitudinal study.

Date of approval.
This was noted and approved on the 18th February 2013.

Yours faithfully

Ms Elizabeth Hall – QMREC Chair.

Appendix 7

Approved ethical application for the longitudinal study including the Participant Information Sheet and the Informed Consent.

For Office Use Only:
Rec Reference ……
Date received: ……

Application form – Queen Mary Research Ethics Committee

1 Name and email address of applicant
Miss Paulina Kloskowska MSc, Centre for Sports and Exercise Medicine, WHRI E-mail: p.kloskowska@qmul.ac.uk
2 Title of study

The biomechanical determinants of lumbo-pelvic muscle imbalance in athletes after adductor strain injury along the process of rehabilitation – a longitudinal study.
3 Investigators
Miss Paulina Kloskowska MSc, BSc Dr Dylan Morrissey PhD MSc MMACP MCSP Professor Roger Woledge, Professor Emeritus of Experimental Physiology Centre for Sports and Exercise Medicine, Queen Mary University of London
4 Proposed timetable
Preferred start date: January 2013 Projected date of completion: Sept 2015
5 Other organisations involved
Saracens RUFC, Harlequins RUFC and other sports clubs Subjects from each of these groups will only be recruited once letters of approval have been granted.
6 Other REC approval
N/A
7 Nature of project e.g. undergraduate, postgraduate

The project is part of a PhD project. Students who will work on this project include

- Mr Waleed Moussa, BSc SEM student, Centre for Sports and Exercise Medicine, Queen Mary University of London

8 Purpose of the research

The purpose of the study is to investigate muscle activation and kinematics over a period of rehabilitation process during simple movement tests in subjects with acute groin pain and determine whether any systematic differences in electromyographically detected muscle onsets exist in muscle activation or movement patterns between:

a) symptomatic and non symptomatic sides

b) controls and subjects

c) dominant and non-dominant leg

d) different phases of rehabilitation process

The tests to be examined are:

- One Leg Standing – the ability to stand unsupported on one leg and lift the other leg to 90 degrees of hip flexion (Hungerford et al 03)

- Active Straight Leg Raise – the ability to lift one leg approximately 60 degrees from the supporting surface. Measurements of pain and effort are scored for all subjects (Mens et al 99)

- One Leg Squat – the ability to stand on one leg and perform a squat on the supporting leg (Crossley et al. 2011)

- Bent Knee Fall Out – the ability of the subject to abduct and externally rotate the hip joint from a position of hip and knee flexion (crook lying) (Sahrmann 98)

-concentric and eccentric hip adduction and abduction

- other, similar, tests as indicated specific to the sport

The overall null hypothesis is that subjects with groin pain due to either articular or muscular presentations have no difference in movement patterns or muscle timing with respect to control subjects in any phase of rehabilitation process.

There are a range of movement or subject group alternative hypotheses including:

- that subjects with acute groin pain will have an altered pattern of movement on the symptomatic side compared with the non-symptomatic side
- that subjects with acute groin pain will have an altered ratio of hip adductor to hip abductor muscle activity compared to normal subjects
- that subjects with acute groin pain will have altered ratios of hip adductor to hip extensor muscle activity
- that subjects with acute groin pain will have an altered ratio of hip flexor to hip extensor muscle activity
- that the effect of dominance affects the muscle activation and muscle ratio in symptomatic subjects
- that the muscle activation and kinematic patterns are different on a different stages of rehabilitation after adductor injury

Background:

Long-standing adduction-related groin pain (LSARGP) is a common problem in football code athletes. Among professional soccer players the incidence of groin pain accounts up for 18% per year (Homlich 2007). There are many disorders potentially

responsible for that symptom, including referred pain of thoracolumbar origin, hip arthrosis, hernia and sports hernia (Holmich 2007), pelvic nerve entrapments (Anderson et al 2001), urological diseases (Fon et al 2000), and many other, few of which are well investigated and described.

According to previous studies (Holmich 2007, Verral et al 2005) one of the most common clinical entities causing groin pain are adductor-related disorders (ARGP). This non-specific diagnosis contains wide range of alterations affecting adductor muscles and consequently, the adduction movement (Ibrahim et al 2007). The possible causes of LSARGP include pathology of muscles, tendons, joints or bones. The variety of probabilities potentially responsible for this syndrome continues to present a significant diagnostic challenge (Holmich 2007, Fricker 1997).

Although there have been a number of studies trying to specify the initial cause of LSARGP (Holmich 2007, Mens et al 2006, Verral 2001), few of them focus on the effect it has on the muscles around the groin and pelvic region. Several authors associated a decreased hip joint range of movement (ROM) with an increased risk of ARGP (Ibrahim et al 2007, Kettunen et al 2000, Gupta et al 2004), while others highlight the relationship between the features of the muscles responsible for core stability (e.g. transversus abdominis) and pathology of adductor muscles (Mens et al 2006). The outcomes, though, do not show significant relation in any of these studies – thus it appears to be rational to continue research in this field, which will help to uncover other relation between pelvic girdle muscles in LSARGP.

One of the main risk factors for LSARGP in athletes is a former acute injury to the adductor muscles (Engenretsen et al. 2010).

Morrissey et al. (2012) carried out research showing the differences between the gluteus medius (GM) to adductor longus (AL) ratio in football players suffering from adductor-related groin pain compared to a matched control group (Figure 1). The data were collected during both moving and stance phase of standing hip flexion and show a significant change of the activation ratio between examined muscles in subjects with groin pain. The data suggests that there may be a common pattern concerning the electromyographic determinants in patients suffering and recovering from groin pain, which shows a significant decrease of GM:AL ratio in patients suffering from groin pain. Further analysis shows it is mainly due to a significant decrease of GM activation.

However, these outcomes have not been analysed in comparison to the kinematic determinants of the analysed movements. As well as EMG results, kinematic outcomes are also expected to show differences between participants with ARGP and healthy controls.

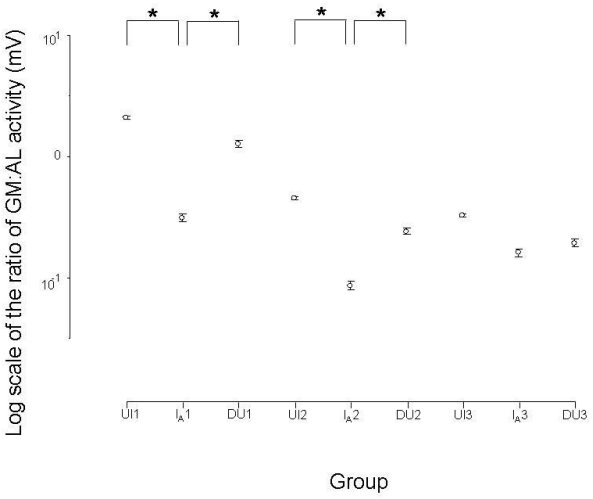


Figure 1: The ratio of GM:AL activity (mV) of injured and uninjured sides in ARGP patients and controls.

During this study a lot of data concerning acute adductor injury will be collected. They will help to complete our knowledge about the muscular changes in groin injuries. These data will allow us not only to complete the knowledge about the general muscle healing process, but will give us a clear picture of athlete's gradual return to health. We expect to find out why some particular groups of athletes fail to recover after such injuries. Based on the athletes' individual findings we will be provided with a clinical tool so that we can give targeted and individualised rehabilitation prescriptions.

References

Hölmich P. Long-standing groin pain in sportspeople falls into three primary patterns, a "clinical entity" approach: a prospective study of 207 patients. Br J Sports Med. 2007;41:247–52

Anderson K, Strickland SM, Warren R. Hip and groin injuries in athletes. Am J Sports Med. 2001;29(4):521-33

Fon LJ, Spence RA. Sportsman's hernia. Br J Surg 2000;87:545-52

Verrall GM, Slavotinek JP, Barnes PG, Fon GT. Description of pain provocation tests used for the diagnosis of sports-related chronic groin pain: relationship of tests to defined clinical (pain and tenderness) and MRI (pubic bone marrow oedema) criteria. Scand J Med Sci Sport. 2005;15:36-42.

Ibrahim A, Murrell GA, Knapman P. Adductor strain and hip range of movement in male professional soccer players. J Orthopaedic Surgery 2007;15(1):46-9

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Morrissey D, Graham J, Screen H, Sinha A, Small C, Twycross-Lewis R, Woledge R. Coronal plane hip muscle activation in football code athletes with chronic adductor groin strain injury during standing hip flexion. *Man Ther,* 17**,** 145-9.

ENGEBRETSEN, A. H., MYKLEBUST, G., HOLME, I., ENGEBRETSEN, L. & BAHR, R. 2010. Intrinsic risk factors for groin injuries among male soccer players: a prospective cohort study. Am J Sports Med, 38, 2051-7.

9 Study design, methodology and data analysis

Each potential participant will be provided with a consent form, information sheet and an explanation of the procedure before participating in the study.

Each subject will be asked to complete a written screening questionnaire to define their lower back, pelvic and groin injury status that has impacted on their ability to train or play. The questionnaire will comprise of two parts; characterisation of participants and self-reported injury history.

Characterisation includes:

Biological data - age, age at puberty, height, weight

Sporting career – age at which commenced specialist sport, amount of playing / practice time, position played, level of competition.

Injury will be defined as an acute adductor longus muscle strain (grade I,II or III) diagnosed by the team physiotherapist or a team physician and impacts the ability to do physical activity. Each sportsman/woman will include a description and location of the injury and will be required to indicate their injury location on a body chart (attached).

It is at this point that consent will be taken and the questionnaire gone through with the subject. At this time, additional data will be collected on:

- Family history
- Past medical history
- Playing load – past / preseason / current
- Injury – onset / presence of prodromic symptoms
- Pain area and behaviour

A physical examination will then be undertaken to determine appropriate inclusion criteria as well as other associated features that may identify subgroups in analysis of the data. This will include:

- Hip joint range of motion and pain provocation tests

- SIJ Kinetic tests

- Isometric hip adduction force and symptom provocation

- Thomas test – muscle length and strength

- Squeeze test – resisted adduction (0 / 60 / 90 degrees of hip flexion)

- Unilateral Resisted Abduction test (30 degrees of hip flexion)

- Bilateral Resisted Abduction test (30 degrees of hip flexion)

- Palpation of the adductor tendons, adductor muscles and pubic insertion

- SIJ passive motion analysis

- Response to ASLR

- Hip quadrant testing

- Labral “grind” test

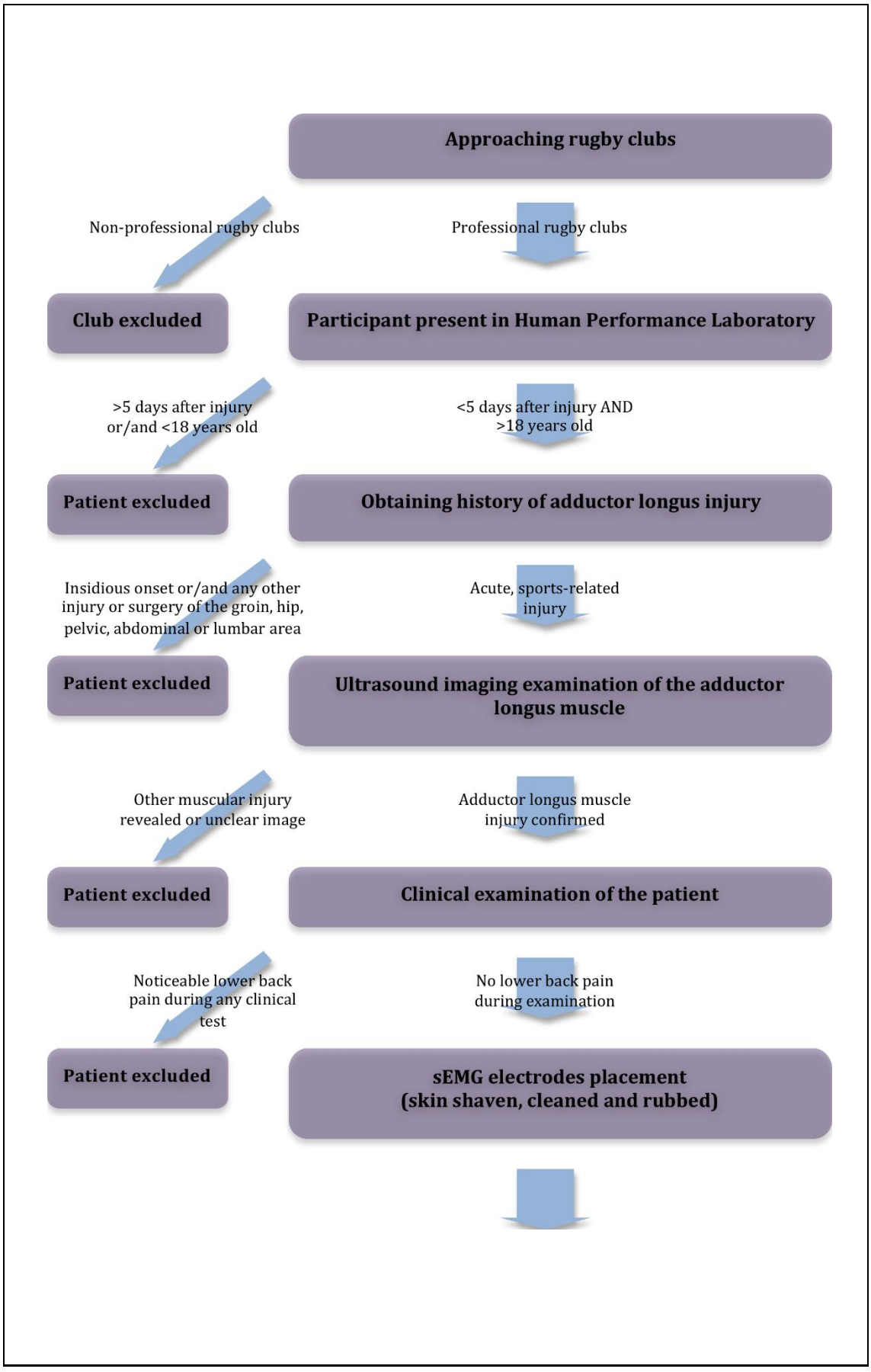
- ultrasound investigation

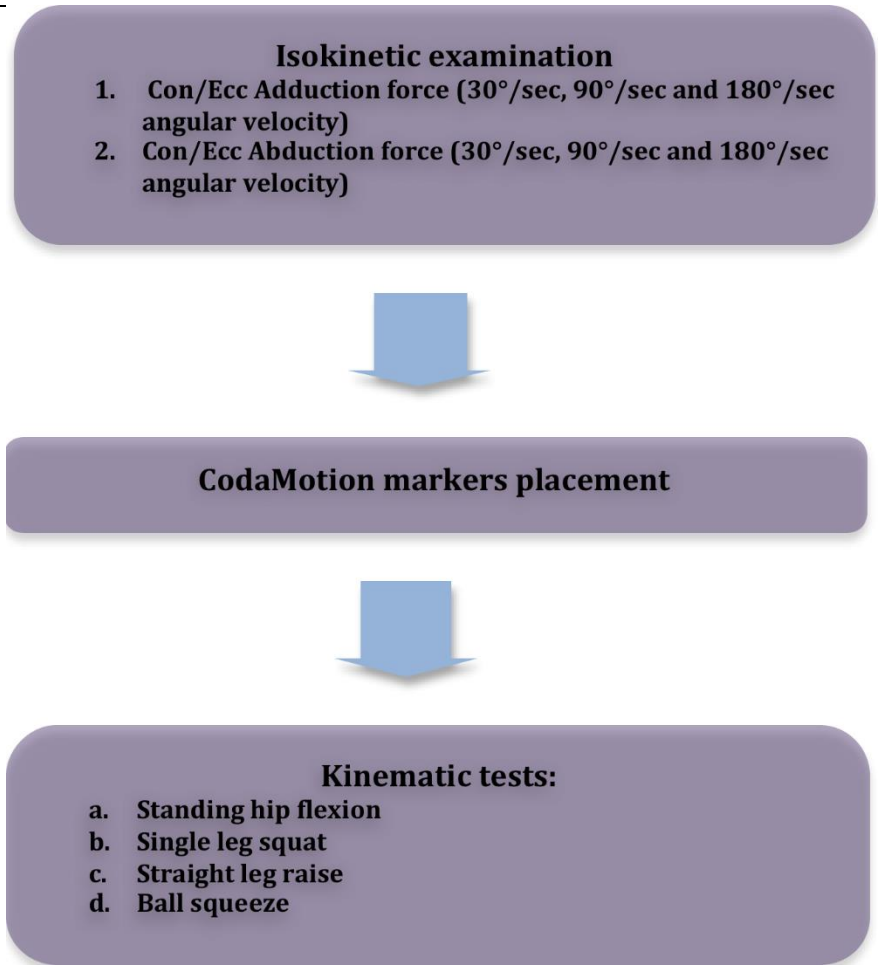
Subjects will then undergo concentric and eccentric peak torque measurements and motion analysis measurements using non-invasive 3-dimensional infra-red cameras (Codamotion cx1, Charnwood Dynamics, Loughborough, UK) and force plates (Kistler, USA) - using standard marker placement protocols for the spine, pelvis and lower limb. In addition to motion analysis, electromyographic (EMG) readings will be taken using the wireless surface EMG device (Noraxon Telemyo 2400T, Scottsdale, Arizona, USA) of the following muscles :

- Hip adductors - 2 channels – Adductor longus and magnus
- Gluteus medius
- Gluteus maximus
- Abdominals – external oblique
- Rectus femoris
- Biceps femoris

Testing will take place in the Human Performance Laboratory at QMUL and should take no longer than 120 minutes.

Simultaneous measurements of muscle strength will also be made using a hand held dynamometer.


Approaching rugby clubs
Non-professional rugby clubs
Professional rugby clubs
Club excluded
Participant present in Human Performance Laboratory
>5 days after injury or/and <18 years old
<5 days after injury AND >18 years old
Patient excluded
Obtaining history of adductor longus injury
Insidious onset or/and any other injury or surgery of the groin, hip, pelvic, abdominal or lumbar area
Acute, sports-related injury
Patient excluded
Ultrasound imaging examination of the adductor longus muscle
Other muscular injury revealed or unclear image
Adductor longus muscle injury confirmed
Patient excluded
Clinical examination of the patient
Noticeable lower back pain during any clinical test
No lower back pain during examination
Patient excluded
sEMG electrodes placement (skin shaven, cleaned and rubbed)



Data analysis

Based on the results of the questionnaire and the physical examination, two sub-groups will be defined: one with a presentation of acute groin pain of soft tissue origin and a control group. The sub-group of participants will be tested 4 times:

- Shortly after injury (maximum 5 days)
- 3 weeks after first test
- 6 weeks after first test
- 9 weeks after first test

Controls will be age, height and activity matched and will only be tested once.

Analysis of collected data for defined sub-groups will be done using a mathematical model written in MatLAb (Mathworks, USA).

Statistical analysis

The data will be assessed for normality and appropriate group comparison analysis undertaken accordingly. The power of the study will be 80% with statistical significance set at $p < 0.05$.

Attached to the application is a flowchart of the study (Attachment 1).

10 Participants to be studied

Number of participants – approximately 15 symptomatic players and 10 asymptomatic players to build a control group.

Lower age limit – 18

Upper age limit – 70

11 Selection criteria

Acute adductor longus injury

Inclusion criteria

- 18 yr of age or older
- Playing elite- or sub elite-level sport
- Activity-related, acute onset groin pain that has been diagnosed as adductor strain (grade I, II or III) confirmed by ultrasound imaging and/or MRI scan

Control group

Inclusion criteria

- Over 18 years of age
- Playing elite- or sub elite-level sport

Exclusion criteria

- History of groin pain or acute groin injury
- Surgery to their lower abdominal, hip, or groin region, or a frank inguinal hernia
- History of prolonged lumbar pain in the past year
- Lumbar pain during examination
- Neurological symptoms
- Systemic disease
- Significant psychological condition

.

12 Recruitment (including incentives and compensation)

Participants will be approached indirectly through an advert provided by email to the medical staff of various football clubs. The medical staff at various clubs will have full details of the study and will have consented to provide this information to their academy teams.

The advert will include details of the research project, its purpose, objective and that participants are required. The advert will reflect the affiliation with QMUL and that the study has the full backing of the football clubs. This advert will be subject to consideration by Dr Morrissey prior to use.

Medical staff at the clubs will be asked to discuss the study with players and provide them with an information sheet detailing the study and the requirements of each participant, along with a consent form.

A contact telephone number will also be enclosed so that any questions or queries potential participants might have can be addressed through a follow up telephone interview with Dr Dylan Morrissey or Paulina Kloskowska.

If a player is happy to participate details will be collected by the club's medical staff or Paulina Kloskowska. It is anticipated that a group of players will be tested at similar times and in the presence of the club physiotherapy staff. Consent will be documented at the time of testing.

Travelling expenses to a maximum of ten pounds per person will be given. The assessments will all be undertaken at the HPL, QMUL. As an incentive, each participant and their medical team will be offered an explanation of the findings. No financial or other reward will be given to participants.

13 Ethical considerations and risks to participants

The main ethical issue will be the need to ensure voluntary participation from players within a club environment. As the clubs will not be incentivised in any way to participate, it is not anticipated that any form of coercion will occur.

The need to remove sufficient clothing to attach the motion markers to the torso and legs is also another consideration. In order for the EMG electrode pads to be well adhered, small areas of the skin will need to be shaved and cleaned. Privacy in the data collection areas will be maximised and subjects will be encouraged to bring suitable clothing. The presence of the club's physiotherapy staff should also ensure that players are confident in the testing being undertaken.

Full and informed consent will be obtained from each participant before entering the study. The participant will be given an information sheet detailing what the study entails and what is required of the participant. This will also be reiterated in person with the opportunity to ask any questions about the project.

Each participant will be protected from harm or injury with all measurements being undertaken in a controlled manner. Participants will be encouraged to avoid potentially painful movements or range of motion.

Each participant will have the right to withdraw from the research at any time, and for whatever reason.

14 Confidentiality, anonymity, and data storage

Each participant's confidentiality and privacy will be assured by the use of a code which will be characterized by each participant's initials and the date of the test. Each participant will be allocated their code on consenting to the study and each coded participant will also have the date that the assessment will be undertaken. This will ensure each participant's anonymity. Only the QMUL research team involved in the investigation will have access to the corresponding name/number data and any other personal information, which will be securely held on a separate server, requiring a password. The data will be securely stored, easily retrievable and well indexed.

Sensitive data will be stored on password protected server databases to which only the investigators will have access too, all such data will be handled in accordance with the provisions of the Data protection Act 1998.

15 Information for participants

Headed Paper

REC Protocol Number.…….

YOU WILL BE GIVEN A COPY OF THIS INFORMATION SHEET

We would like to invite you to participate in this research project. You should only participate if you want to; choosing not to take part will not disadvantage you in any way. Your decision will not affect your access to treatment or services. Before you decide whether you want to take part, it is important for you to understand why the research is being done and what your participation will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information.

If you volunteer to take part you will be invited to meet the study team at the Human Performance Laboratory, Queen Mary's University of London. After answering any questions you may have, you will be asked to fill in a questionnaire. The first part will ask for you for personal details such as age,

height, weight, other sporting activities past and present and amount of playing time. The second part will deal with self-reported injury, particularly pain or injury in the groin area.

A short physical examination will be undertaken to determine your suitability to participate.

We will then attach several electrodes that will be used to measure the electrical activity in your muscles. These electrodes do not carry any electricity into your body. These electrodes are self adhesive and designed to stick to skin and be removed easily and painlessly.
After that you will be asked to perform a couple of movements and the force of these movement will be measured. This will be obtained by attaching your leg to the special machine, which is able to measure the strength of the movement.
This will be followed by the application of 20 small infra-red motion sensors to your trunk and legs with medical grade double sided sticky tape. This will require you to wear clothing that reveals the skin of the lumbar spine, shoulder blades and legs. A pair of close fitting shorts would be ideal. We can provide these if necessary.
We will then make some measurements of your movement patterns during several movement tasks while standing or lying on a force plate that measures weight transfer. The total time required to attach markers and marker boxes and to measure the movement should be about one hour.

We do not anticipate any risk or discomfort by participating in this study. You will be encouraged to avoid any movements that may reproduce your pain or make it worse. In order to participate in the study you will be asked to meet certain study inclusion/exclusion criteria.
If you participate in this study you will be given an identification number and so will remain completely anonymous throughout. All personal information linking you to this number will be kept separately and stored securely on a database server to which only I will have access to. All information will be handled in accordance with the provisions of the data protection act 1998 and your confidentiality assured.

My correspondence details are included in this application if you wish to contact me, to obtain further details or to ask any questions regarding the study:
Paulina Kloskowska
p.kloskowska@qmul.ac.uk 07428147932
Centre for Sport and Exercise Medicine
Mile End Hospital
Bancroft Road
LONDON E1 4DG

Alternatively, you can contact:

Dr Dylan Morrissey
Centre for Sport and Exercise Medicine
Mile End Hospital
Bancroft Road
LONDON E1 4DG
And on d.morrissey@qmul.ac.uk 02082238839

16 Consent

Please complete this form after you have read the Information Sheet and/or listened to an explanation about the research.

Title of Study: The biomechanical determinants of lumbo-pelvic muscle imbalance in footballers with adductor-related groin pain.

Queen Mary Research Ethics Committee Ref: _____

. • Thank you for considering taking part in this research. The person organizing the research must explain the project to you before you agree to take part.

. • If you have any questions arising from the Information Sheet or explanation already given to you, please ask the researcher before you decide whether to join in. You will be given a copy of this Consent Form to keep and refer to at any time.

. • I understand that if I decide at any other time during the research that I no longer wish to participate in this project, I can notify the researchers involved and be withdrawn from it immediately.

. • I consent to the processing of my personal information for the purposes of this research study. I understand that such information will be treated as strictly confidential and handled in accordance with the provisions of the Data Protection Act 1998.

Participant's Statement:

I _____ agree that the research project named above has been explained to me to my satisfaction and I agree to take part in the study. I have read both the notes written above and the Information Sheet about the project, and understand what the research study involves.

Signed: Date:

Investigator's Statement:

I _____ confirm that I have carefully explained the nature, demands and any foreseeable risks (where applicable) of the proposed research to the volunteer.

Signed: Date:

17 Signature of applicant and authorising signatories.

Appendix 8

Inclusion and exclusion criteria form for the observational study.

FORM A

Code:

Age:

Height:

Weight:

Dominant leg:

Symptomatic leg:

Level of competition now played:

Current/recent symptoms:

Please mark the area of your symptoms on the body chart attached on the last page.

How long have these symptoms been present / were they present for?

Describe how these symptoms started

Have you had any treatment for this condition? Please explain.

Have you had any investigations for this condition? Please explain.

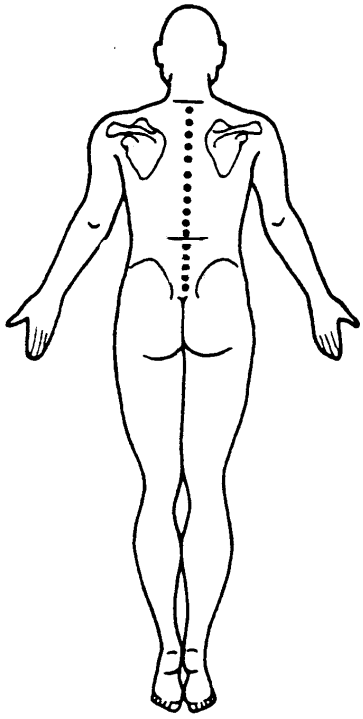
PART I: Inclusion criteria

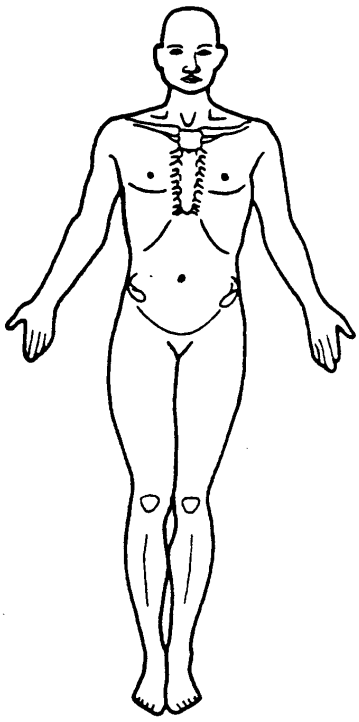
1. Tenderness on palpation:	
a. Adductor tendon	
b. Adductor insertion to pubic bone	
c. Pubic symphysis	
d. Iliopsoas muscle	
2. Presence of groin pain during active hip adduction against resistance at the time of assessment	
3. Presence of groin pain during active hip flexion against resistance at the time of assessment	
4. Presence of groin pain during passive hip abduction (stretch)	
5. Presence of groin pain during passive hip extension (stretch)	

6. Squeeze test positive (0°, 60° or 90° of flexion)	
7. Proven muscular pathology on imaging tests	

PART II: Exclusion criteria

1. Groin pain that commenced as a result of the articular pathology	
2. Surgery to the lower abdominal, hip or groin region	
3. Frank inguinal hernia	
4. Lumbar pain that predominates on physical examination	
5. Neurological symptoms	
6. Systemic disease	
8. Labral grind test positive	
9. Flexion adduction (FAbER) test positive	
7. Significant psychological condition	





Appendix 9

Inclusion and exclusion criteria form for the longitudinal study: first testing occasion.

FORM A

Code:

Age:

Height:

Weight:

Dominant leg:

Symptomatic leg:

Level of competition now played:

Current/recent symptoms:

Please mark the area of your symptoms on the body chart attached on the last page.

How long have these symptoms been present / were they present for?

Describe how these symptoms started

Have you had any treatment for this condition? Please explain.

Have you had any investigations for this condition? Please explain.

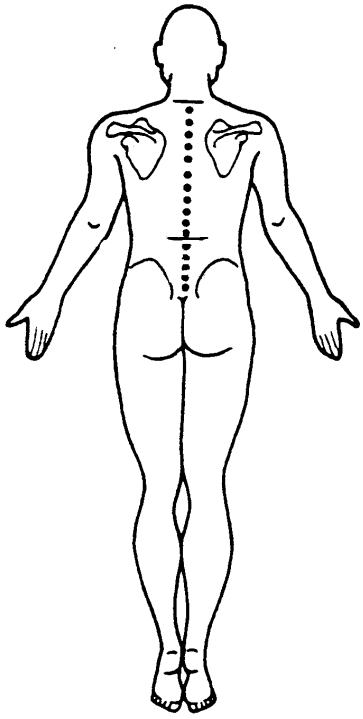
PART I: Inclusion criteria

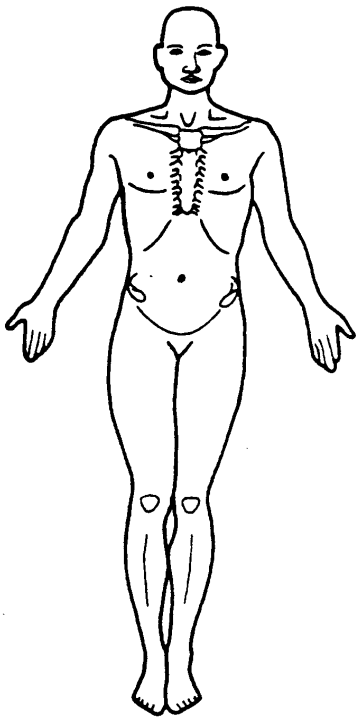
1. Tenderness on palpation:	
d. Adductor tendon	
e. Adductor insertion to pubic bone	
f. Pubic symphysis	
d. Iliopsoas muscle	
2. Presence of groin pain during active hip adduction against resistance at the time of assessment	
3. Presence of groin pain during active hip flexion against resistance at the time of assessment	
4. Presence of groin pain during passive hip abduction (stretch)	
5. Presence of groin pain during passive hip extension (stretch)	

6. Squeeze test positive (0°, 60° or 90° of flexion)	
7. Proven muscular pathology on imaging tests	

PART II: Exclusion criteria

1. Groin pain that commenced as a result of the articular pathology	
2. Surgery to the lower abdominal, hip or groin region	
3. Frank inguinal hernia	
4. Lumbar pain that predominates on physical examination	
5. Neurological symptoms	
6. Systemic disease	
8. Labral grind test positive	
9. Flexion adduction (FAbER) test positive	
7. Significant psychological condition	





Appendix 10

Inclusion and exclusion criteria form for the longitudinal study: subsequent testing occasions.

FORM A (subsequent testing)

Code:

1. Tenderness on palpation:	
g. Adductor tendon	
h. Adductor insertion to pubic bone	
i. Pubic symphysis	
d. Iliopsoas muscle	
2. Presence of groin pain during active hip adduction against resistance at the time of assessment	
3. Presence of groin pain during active hip flexion against resistance at the time of assessment	
4. Presence of groin pain during passive hip abduction (stretch)	

5. Presence of groin pain during passive hip extension (stretch)	
6. Squeeze test positive (0°, 60° or 90° of flexion)	

Appendix 11

Inclusion and exclusion criteria form for the control participants in observational and longitudinal studies.

FORM B

Code:

Age:

Height:

Weight:

Dominant leg:

Level of competition now played:

1. History of groin pain	
2. Surgery to their lower abdominal, hip, or groin region, or a frank inguinal hernia	
3. History of lumbar pain in the past year	
4. Neurological symptoms	

5. Systemic disease	

Appendix 12

Combine stats GUI – a custom-made MatLab program to statistically analyse the data for the observational and longitudinal studies.

```
function varargout = CombineStatsGUI(varargin)
```

Beginning of the GUI initialization code

```
gui_Singleton = 1;
gui_State = struct('gui_Name',       mfilename, ...
    'gui_Singleton',  gui_Singleton, ...
    'gui_OpeningFcn', @CombineStatsGUI_OpeningFcn, ...
    'gui_OutputFcn',  @CombineStatsGUI_OutputFcn, ...
    'gui_LayoutFcn',  [] , ...
    'gui_Callback',   []);
if nargin && ischar(varargin)
    gui_State.gui_Callback = str2func(varargin);
end

if nargout
    [varargout1:nargout] = gui_mainfcn(gui_State, varargin{:});
else
    gui_mainfcn(gui_State, varargin{:});
end
```

End of the GUI initialization code

Opening the interface

```
function CombineStatsGUI_OpeningFcn(hObject, eventdata, handles,
varargin)
handles.output = hObject;
guidata(hObject, handles);

function varargout = CombineStatsGUI_OutputFcn(hObject, eventdata,
handles)
varargout{1} = handles.output;
```

Choosing the desired comparisons from a drop-down menu available in the GUI

```
function Subject1_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Movement1_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Leg1_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Segment1_Callback(hObject, eventdata, handles)
```

```
MakeSet(hObject, handles)
function Time_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Site1_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Subject2_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Movement2_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Leg2_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Site2_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Site3_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Leg3_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Movement3_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Subject3_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Muscle1_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Muscle2_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function IsSubtract_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Source1_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Source2_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Source3_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function IsRatio_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function IsLog_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function Contra_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
function AvSub_Callback(hObject, eventdata, handles)
function AvMov_Callback(hObject, eventdata, handles)
function AvLeg_Callback(hObject, eventdata, handles)
function AvSite_Callback(hObject, eventdata, handles)
function AvTim_Callback(hObject, eventdata, handles)
function AvSource_Callback(hObject, eventdata, handles)
function Participant_Callback(hObject, eventdata, handles)
function ReportFile_Callback(hObject, eventdata, handles)
```

Reading the data

```
function SourceA_Callback(hObject, eventdata, handles)
sourcefileA=get(handles.SourceA, 'String');
[XLNum XLtxt] = xlsread(sourcefileA, 'AllData', 'A10:W3000');
Logic=XLNum(:,1:20);  rrlg=length(Logic);

XLNum = xlsread(sourcefileA, 'AllData', 'D10:FD3000');
XLNum=XLNum(:,21:end);
```

```
[rr,cc]=size(XLNum); Data=NaN*ones(rrlg,200); Data(1:rr,1:cc)=XLNum;
Data=reshape(Data,[rrlg 20 10]);
data=Data(:,14:15,:);
disp(length(find(data<5E-5)))
data(data<5E-5)=NaN; Data(:,14:15,:)=data;
disp(length(find(data<5E-5)))
XLNum = xlsread(sourcefileA, 'AllData', 'D10:NV3000');
XLNum=XLNum(:,284:end);
[rr,cc]=size(XLNum); DataK=NaN*ones(rrlg,100); DataK(1:rr,1:cc)=XLNum;
DataK=reshape(DataK,[rrlg 20 5]);
DataK(:,:,1:2:9)=DataK(:,:,:);
DataK(:,:,2:2:8)=DataK(:,:,3:2:9)-DataK(:,:,1:2:7);
handles.SegDat=DataK;
handles.EMGDat=Data(:,4:20,:);
handles.FileNames=XLtxt(:,2);
SportsGroup=XLtxt(:,1);
handles.isPat=strcmp('Pat',SportsGroup);
handles.isAm =strcmp('Am', SportsGroup);
handles.isPro=strcmp('Pro',SportsGroup);
handles.isUf =strcmp('Uf', SportsGroup);
handles.isRb =strcmp('Rb', SportsGroup);
handles.isFh =strcmp('Fh', SportsGroup);
handles.SportsGroup=SportsGroup;
handles.isinj   =Logic(:,1);
handles.isinjL  =Logic(:,2);
handles.isinjR  =Logic(:,3);
handles.pantnum=Logic(:,4);
handles.isleft  =Logic(:,5);
handles.isright =Logic(:,6);
handles.isone   =Logic(:,7);
handles.israis  =Logic(:,8);
handles.isbent  =Logic(:,9);
handles.isquick=Logic(:,10);
handles.isstand =Logic(:,11);
handles.isdom   =Logic(:,12);
handles.isinjipsi   =Logic(:,13);
handles.isinjcontra =Logic(:,14);

disp('Data loaded')
guidata(hObject, handles);
```

Setting the statistical analysis package

```
function MakeSet(hObject, handles)
set(handles.AnovaTableE, 'Visible', 'off'); set(handles.AnovaTableK,
'Visible', 'off')
set(handles.BonFerr, 'Visible', 'off'); set(handles.BonFerrK,
'Visible', 'off')
set(handles.text35, 'Visible', 'off'); set(handles.text113, 'Visible',
'off')
set(handles.text38, 'Visible', 'off'); set(handles.text114, 'Visible',
'off')
hhE=handles.axes1;   hhEp=handles.axes2;   hhK=handles.axes3;
hhKp=handles.axes4;
hhCor=handles.axes5; hhCor2=handles.axes6; hhCor3=handles.axes7;
hhCor4=handles.axes8;
```

```
cla(hhK, 'reset');   cla(hhE, 'reset');    cla(hhKp, 'reset');
cla(hhEp, 'reset');
cla(hhCor, 'reset'); cla(hhCor2, 'reset'); cla(hhCor3, 'reset');
cla(hhCor4, 'reset');
hhCorList=[hhCor hhCor2 hhCor3 hhCor4];
```

Reading the settings from the data chosen for further comparisons

```
Source(1)=get(handles.Source1, 'Value');
Source(2)=get(handles.Source2, 'Value');
Source(3)=get(handles.Source3, 'Value');
Subject(1)=get(handles.Subject1, 'Value');
Subject(2)=get(handles.Subject2, 'Value');
Subject(3)=get(handles.Subject3, 'Value');
MoveMent(1)=get(handles.Movement1,
'Value');MoveMent(2)=get(handles.Movement2, 'Value');
MoveMent(3)=get(handles.Movement3, 'Value');
Leg(1)=get(handles.Leg1, 'Value');
Leg(2)=get(handles.Leg2, 'Value');            Leg(3)=get(handles.Leg3,
'Value');
Site(1)=get(handles.Site1, 'Value');        Site(2)=get(handles.Site2,
'Value');          Site(3)=get(handles.Site3, 'Value');
Muscle(1)=get(handles.Muscle1, 'Value');
Muscle(2)=get(handles.Muscle2, 'Value');
Angle=get(handles.Segment1, 'Value');
Contra=get(handles.Contra, 'Value');
Time=get(handles.Time, 'Value');
if Time==1; TT=1; end
if Time==2; TT=2; end
if Time==3; TT=[1 3]; end
if Time==4; TT=[2 4]; end
if Time==5; TT=[1 3 5]; end
if Time==6; TT=[2 4 6]; end
if Time==7; TT=[1 3 5 7]; end
kk=length(TT);

cols={'b' 'r' 'g'};
if Source(3)==14; jj=2; else jj=3; end; handles.jj=jj;

Y4avE=[]; Y4avK=[]; Pant4av=[]; Sj4av=[];
M4av=[];L4av=[];St4av=[];T4av=[]; So4av=[];
```

Collecting the relevant sEMG data from the data set in the interface

```
p=0;
ReportE(1:4,1:15)={[]};ReportK(1:4,1:15)={[]};
TtestsE(1:4,1:15)={[]}; TtestsK(1:4,1:15)={[]};
for k=1:4; SubDatE{k}=[]; SubDatK{k}=[]; GList{k}=[]; end;
handles.XLTab=[];handles.XLTab{kk,jj}=NaN;
for j=1:jj
```

Choosing the source of the data (groups of

participants)

```
    if Source(j)==1;  SoUse= handles.isAm | handles.isPro |
handles.isUf | handles.isRb | handles.isFh;end % AllData
    if Source(j)==2;  SoUse= handles.isAm ; end % Amateurs only
    if Source(j)==3;  SoUse= handles.isPro ; end % Professionals only
    if Source(j)==4;  SoUse= handles.isRb ; end % Rugby
    if Source(j)==5;  SoUse= handles.isUf     ; end % Frisbee
    if Source(j)==6;  SoUse= handles.isFh     ; end % Field hockey
    if Source(j)==7;  SoUse=  handles.isPro|  handles.isRb |
handles.isFh;end % H P R
    if Source(j)==8;  SoUse= handles.isAm | handles.isUf ;end % A F
    if Source(j)==9;  SoUse= handles.isAm | handles.isPro ;end % A P
    if Source(j)==10;  SoUse= handles.isAm | handles.isPro |
handles.isRb ;end % A P R
    if Source(j)==11;  SoUse=  handles.isRb | handles.isFh;end % H R
    if Source(j)==12;  SoUse= handles.isAm | handles.isPro |
handles.isUf ;end % A P F
only

    if Subject(j)==1; SjUse= handles.isinj | ~handles.isinj ; end %
All
    if Subject(j)==2; SjUse= handles.isinj; end % Injured
    if Subject(j)==3; SjUse=~handles.isinj; end % Un-injured
    if Source(3)==14
        if Subject(1)==Subject(2) && Source(1)==Source(2)
            Paired=1;
        else
            Paired=0;
        end
    else
        if Subject(1)==Subject(2) && Source(1)==Source(2) && .....
                Subject(3)==Subject(1) && Source(3)==Source(1)  ;
            Paired=1;
        else
            Paired=0;
        end
    end
```

Choosing the source of the data (movement manoeuvre to be further analysed)

```
    if MoveMent(j)==1; MvUse=handles.isone | handles.isbent |
handles.israis| handles.isstand | handles.isquick;end % Any
    if MoveMent(j)==2; MvUse=handles.isone; end % One Leg Bend
    if MoveMent(j)==5; MvUse=handles.isstand; end % Hip Flexion
```

Choosing the source of the data (leg to be further analysed)

```
    if Leg(j)==1; LgUse=handles.isleft | handles.isright; end % Either
    if Leg(j)==2; LgUse=handles.isleft; end % Left
    if Leg(j)==3; LgUse=handles.isright; end % Right
    if Leg(j)==4; LgUse=handles.isdom; end % Dominant
```

```
    if Leg(j)==5; LgUse=~handles.isdom; end % Non-Dominant
    if Leg(j)==6; LgUse=handles.isinjipsi; end % Injured
    if Leg(j)==7; LgUse=~handles.isinjipsi; end % Uninjured
```

Choosing the source of the data (movement phase to be further analysed)

```
    for k=1:kk          handles.XLTab{k,j}=[];
        TmUse=TT(k);
        Use=SoUse & SjUse & MvUse & LgUse;
```

Choosing the source of the data (what is the leg status to be further analysed)

```
if Site(j)==2; ALorR=Use(Use); end             % Right;
        if Site(j)==3; ALorR=handles.isinjR(Use); end  % Injured;
        if Site(j)==4; ALorR=handles.isinjL(Use); end  % Uninjured;
        if Site(j)==5; ALorR=handles.isright(Use); end % Moving;
        if Site(j)==6; ALorR=handles.isleft(Use); end  % Not moving;
```

sEMG data processing for the formerly selected analysis combination(filtering, rectifying, smoothing and log transforming)

```
        EMGUse=squeeze(handles.EMGDat(Use,:,TmUse));
        SegDatUse=squeeze(handles.SegDat(Use,:,TmUse));
        PantUse{j,k}=handles.pantnum(Use);
        handles.SGUse{j,k}=handles.SportsGroup(Use);
        if Site(j)==1; ALorR=~Use(Use); end
        clear SetE RawSet
        Ratio=get(handles.IsRatio, 'Value'); if Ratio; qq=2; else
qq=1; end
        for q=1:qq
            if q==2 && Contra; ALorR=~ALorR; end
            MMuse=ones(size(EMGUse,1),1)*2*Muscle(q)-1;
            MMuse=MMuse+ALorR;
            if isempty(EMGUse); disp('No Data Available'); return; end
            RawSet(:,q)=EMGUse(sub2ind(size(EMGUse), 1:length(MMuse),
MMuse'));
        End

        if Ratio; SetE=RawSet(:,1)./RawSet(:,2); else SetE=
RawSet(:,1); end
        if get(handles.IsLog, 'Value') ; SetE=log(SetE); end
        MusNames=get(handles.Muscle1, 'String');
        lab2=MusNames(Muscle(1)); lab1=''; lab3='';
        if Ratio; lab3=MusNames(Muscle(2)); end
        if get(handles.IsLog, 'Value'); lab1='Log:'; end
```

```
        handles.MusName=[char(lab1) char(lab2) '/' char(lab3)];
```

Reading and setting the kinematic data: joints and rotations for the formerly chosen data combination to be analysed

```
Ause=  (mod(Angle-1,3)+1)     +   (ceil(Angle/3)-1)*6     + ALorR*3;
        SetK =  (SegDatUse(sub2ind(size(SegDatUse), 1:length(Ause),
Ause')))';

        if kk>1 && k==1; StartSet=SetK; end
        if kk>1 &&  get(handles.IsSubtract, 'Value'); SetK=SetK-
StartSet; end

        AngNames=get(handles.Segment1, 'String');
        lab2=AngNames(Angle); lab1=''; lab3=''; handles.AngName=lab2;
        if get(handles.IsLog, 'Value'); lab1='Log of '; end
        handles.AngName=[char(lab2) char(lab3)];
        Sets{j,k}=SetK;
        p=p+1; Tab(1:length(SetK),p)=SetK; count(p)=length(SetK);
```

Basic Set statistics – performing the statistical analysis

```
        SetEMean(k)={Mynanmean(SetE)};       SetESD=Mynanstd(SetE);
SetEN(k)={sum(~isnan(SetE))};
        SetEMiss(k)={length(SetE)-SetEN{k}};
SetESEM(k)={SetESD/sqrt(SetEN{k})};
        SetKMean(k)={Mynanmean(SetK)};       SetKSD=Mynanstd(SetK);
SetKN(k)={sum(~isnan(SetK))};
        SetKMiss(k)={length(SetK)-SetKN{k}};
SetKSEM(k)={SetKSD/sqrt(SetKN{k})};
=Ps=PantUse{j,k}; PList=unique(Ps); clear PSetK PSetE;
PN=length(PList);

        for p=1:PN;
            Obs4p=Ps==PList(p);
            PSetK(p)=nanmean(SetK(Obs4p)); PDatK{p}=SetK(Obs4p);
            PSetE(p)=nanmean(SetE(Obs4p)); PDatE{p}=SetE(Obs4p);
            GList{k}=[GList{k}; Ps(Ps==PList(p))];
        end
        SubDatE{k}=[SubDatE{k} PSetE]; SubDatK{k}=[SubDatK{k} PSetK];
        PairDatE{k,j}=PSetE;           PairDatK{k,j}=PSetK;
        SetENL(k)={nanN(PSetE)};       SetKNL(k)={nanN(PSetK)};
        SetEMeanL(k)={nanmean(PSetE)};
SetKMeanL(k)={nanmean(PSetK)};
        SetESEML(k)={nansem(PSetE)};     SetKSEML(k)={nansem(PSetK)};



        handles.GList=GList;
```

Accumulating data for ANOVA

```
        ZZ=zeros(size(PSetK));
        Y4avK=[Y4avK; PSetK']; Y4avE=[Y4avE; PSetE'];
        Pant4av=[Pant4av; PList];
        Sj4av= [Sj4av; ZZ'+Subject(j)];
        M4av=[M4av; ZZ'+MoveMent(j)];
        L4av=[L4av; ZZ'+Leg(j)];
        St4av=[St4av; ZZ'+Site(j)];
        T4av=[T4av; ZZ'+k];
        So4av=[So4av; ZZ'+Source(j)];
```

Plotting the data

```
        plot(hhE,SetE*0+k+j/5, SetE, [cols{j} '*']); xlim(hhE,[0.5
kk+1]); hold(hhE,'on')
        errorbar(hhE,k+j/5-0.1, SetEMean{k}, SetESEM{k},[cols{j} 'd'])
        plot(hhK,SetK*0+k+j/5, SetK, [cols{j} '*']); xlim(hhK,[0.5
kk+1]); hold(hhK,'on')
        errorbar(hhK,k+j/5-0.1, SetKMean{k}, SetKSEM{k},[cols{j} 'd'])

        for p=1:PN
            Spider(hhCorList(k),PDatK{p}, PDatE{p}, cols{j});
hold(hhCorList(k),'on');
            xx=PDatK{p}; yy=PDatE{p}; MeanX=nanmean(xx);
MeanY=nanmean(yy);
            pad=NaN*zeros(length(xx)-1,1);
            disp([k j p])
            handles.XLTab{k,j}=[handles.XLTab{k,j} ;[[PList(p); pad],
yy, [MeanY; pad], xx, [MeanX; pad]]];

        end
        if j==jj
            [Rval,Pval] = corr([SubDatE{k}' SubDatK{k}'], 'type',
'Pearson', 'rows', 'complete', 'tail', 'both');
            label=['R= ' num2str(Rval(1,2),3), ' P= '
num2str(Pval(1,2),3)];
            text(0.5,0.9, label, 'units', 'normalized', 'Parent',
hhCorList(k))

        end


        clear ReOrdered

        for q=1:size(SegDatUse,1)
            for a=[1:3 7:9];
                ReOrdered(q,a+ALorR(q)*3)=SegDatUse(q,a);
            end
            for a=[4:6 10:12];
                ReOrdered(q,a-ALorR(q)*3)=SegDatUse(q,a);
            end
        end

        handles.SegDatUse{j,k}=ReOrdered;
```

```
    end
```

Setting the output – report of the statistics

```
    Col=(j-1)*5+ (1:length(SetKN));
    ReportK(1, Col)=SetKN;                  ReportE(1, Col)=SetEN;
    ReportK(2, Col)=SetKMiss;               ReportE(2, ol)=SetEMiss;
    ReportK(3, Col)=SetKMean;               ReportE(3, Col)=SetEMean;
    ReportK(4, Col)=SetKSEM;                ReportE(4, Col)=SetESEM;
    ReportK(5, Col)=SetKNL;                 ReportE(5, Col)=SetENL;
    ReportK(6, Col)=SetKMeanL;              ReportE(6,Col)=SetEMeanL;
    ReportK(7, Col)=SetKSEML;               ReportE(7, Col)=SetESEML;


    handles.FileNamesUse{j}=handles.FileNames(Use);
end
```

Preparing the data for the t-test and performing the t-test

```
compare=[2 3 3; 1 1 2];
if jj==3;ncomp=3; else ncomp=1; end
for j=1:ncomp
    G1=compare(1,j)-1; G2=compare(2,j)-1;
    for k=1:kk
DFE=ReportE{1,5*G1+k}+ReportE{1,5*G2+k}-1;
        DFK=ReportK{1,5*G1+k}+ReportK{1,5*G2+k}-1;
        MeanDE=ReportE{3,5*G1+k}-ReportE{3,5*G2+k};
        MeanDK=ReportK{3,5*G1+k}-ReportK{3,5*G2+k};
        SEDE=sqrt(ReportE{4,5*G1+k}^2+ReportE{4,5*G2+k}^2);
        SEDK=sqrt(ReportK{4,5*G1+k}^2+ReportK{4,5*G2+k}^2);
        StudE=abs(MeanDE/SEDE);
        StudK=abs(MeanDK/SEDK);
        try
            PnullE=2*(1-tcdf(StudE,DFE));
            PnullK=2*(1-tcdf(StudK,DFK));
        catch
            PnullE=1;
            PnullK=1;
        end
                DFEL=ReportE{5,5*G1+k}+ReportE{5,5*G2+k}-1;
        DFKL=ReportK{5,5*G1+k}+ReportK{5,5*G2+k}-1;
        MeanDEL=ReportE{6,5*G1+k}-ReportE{6,5*G2+k};
        MeanDKL=ReportK{6,5*G1+k}-ReportK{6,5*G2+k};
        SEDEL=sqrt(ReportE{7,5*G1+k}^2+ReportE{7,5*G2+k}^2);
        SEDKL=sqrt(ReportK{7,5*G1+k}^2+ReportK{7,5*G2+k}^2);
        StudEL=abs(MeanDEL/SEDEL);
        StudKL=abs(MeanDKL/SEDKL);
         try
            PnullEL=2*(1-tcdf(StudEL,DFEL));
            PnullKL=2*(1-tcdf(StudKL,DFKL));
        catch
            PnullEL=1;
            PnullKL=1;
        end
```

```
        TtestsE(1:6,(j-1)*5+k)={MeanDE; SEDE; StudE;
num2str(PnullE,'%7.2g'); StudEL; num2str(PnullEL,'%7.2g')};
        TtestsK(1:6,(j-1)*5+k)={MeanDK; SEDK; StudK;
num2str(PnullK,'%7.2g'); StudKL; num2str(PnullKL,'%7.2g')};
    end
end

set(handles.Results, 'data', ReportE)
set(handles.ResultsK, 'data', ReportK)
set(handles.Ttests, 'data', TtestsE)
set(handles.TtestsK, 'data', TtestsK)
handles.Y4avE  = Y4avE;  handles.Y4avK  = Y4avK;
handles.Sj4av = Sj4av ; handles.M4av  = M4av ;
handles.L4av  = L4av; handles.St4av = St4av;  handles.T4av  = T4av ;
handles.Pant4av=Pant4av; handles.Sets=Sets; handles.PantUse=PantUse;
handles.Subjects=Subject; handles.MoveMent=MoveMent; handles.Tab=Tab;
handles.Leg=Leg; handles.Site=Site; handles.Source=Source;
handles.count=count;
handles.So4av=So4av;
```

Performing paired comparisons

```
if Paired
    set(handles.axes2, 'Visible', 'on')
    axes(handles.axes2);cla;hold on
    set(handles.PairedTests, 'Visible', 'on');
    lincol={'-k' ':m' '-c'};
    for j=1:ncomp
        G1=compare(1,j); G2=compare(2,j);
        for k=1:kk
            yy1=PairDatE{k,G1};
            plot((k+G1/5)+yy1*0, yy1, 'dr', 'MarkerFacecolor', 'r')
            yy2=PairDatE{k,G2};
            plot((k+G2/5)+yy2*0, yy2, 'db', 'MarkerFacecolor', 'b')
            for p=1:length(yy1); plot(k+[G1 G2]/5, [yy1(p) yy2(p)],
lincol{j}); end
            DF=2*length(find(~isnan(yy1)))-1; Ydiff=yy1-yy2;
MeanD=nanmean(Ydiff);
            SKD=nanstd(Ydiff)/sqrt(DF);
            Stud=abs(MeanD/SKD);
            try Pnull=2*(1-tcdf(Stud,DF));catch; Pnull=1;end
            PairedTests(1:5,(j-1)*3+k)={(DF+1)/2; MeanD; SKD; Stud;
Pnull};

        end
        set(handles.PairedTests, 'data', PairedTests);

    end
    axes(handles.axes4);cla;hold on
    for j=1:ncomp
        G1=compare(1,j); G2=compare(2,j);
        for k=1:kk
```

```
            yy1=PairDatK{k,G1};
            plot((k+G1/5)+yy1*0, yy1, 'dr', 'MarkerFacecolor', 'r')
            yy2=PairDatK{k,G2};
            plot((k+G2/5)+yy2*0, yy2, 'db', 'MarkerFacecolor', 'b')
            for p=1:length(yy1); plot(k+[G1 G2]/5, [yy1(p) yy2(p)],
lincol{j}); end
            DF=2*length(find(~isnan(yy1)))-1; Ydiff=yy1-yy2;
MeanD=nanmean(Ydiff);
            SKD=nanstd(Ydiff)/sqrt(DF);
            Stud=abs(MeanD/SKD);
            try Pnull=2*(1-tcdf(Stud,DF));catch; Pnull=1;end
            PairedTests(1:5,(j-1)*3+k)={(DF+1)/2; MeanD; SKD; Stud;
Pnull};
        end
        set(handles.PairedTestsK, 'data', PairedTests);
    end


else
    set(handles.PairedTests, 'Visible', 'off');
    axes(handles.axes2);cla;hold on
    set(handles.axes2, 'Visible', 'off')
end
%%
guidata(hObject, handles);
```

Plotting the kinematic data

```
function KinemPlot_Callback(hObject, eventdata, handles)

Labels={'Pelvis Moving' 'Pelvis Not Moving' 'Hip Moving' 'Hip Not
Moving'};
YLabels={'Coronal' 'Sagittal' 'Horizontal'};
figure(4);
cols='brg';
for j=1:handles.jj
    if j==1;  clf; end
    for k=1:size(handles.SegDatUse, 2)
        SDU=handles.SegDatUse{j,k};         SegDatMean=nanmean(SDU);
        SegDatSem=nanstd(SDU)/sqrt(size(SDU,1));
        sp=0;
        for pp=1:3
            for s=1:4
                sp=sp+1;
                yy=SegDatMean(sp); ee=SegDatSem(sp);
                subplot(3,4,sp)
                errorbar(k, yy, ee, cols(j)); hold on;
                plot(k, yy, [ 'o' cols(j)] )
                if pp==1; title(Labels(s)); end
                if s==1; ylabel(YLabels(pp)); end
                if j==2; axis tight; ylm=ylim; while ylm(2)-ylm(1)<20;
ylm=ylm+[-5 5]; end; ylim(ylm); end; xlim([0.7 3.3]);
            end
        end
    end
end
axes(handles.axes1)
```

```
function IndPlot_Callback(hObject, eventdata, handles)
```

Performing the ANOVA

```
function ANOVA_Callback(hObject, eventdata, handles)
MakeSet(hObject, handles)
set(handles.AnovaTableE, 'Visible', 'on'); set(handles.AnovaTableK,
'Visible', 'on')
set(handles.BonFerr, 'Visible', 'on'); set(handles.BonFerrK,
'Visible', 'on')
set(handles.text35, 'Visible', 'on'); set(handles.text113, 'Visible',
'on')
set(handles.text38, 'Visible', 'on'); set(handles.text114, 'Visible',
'on')
Y4avK=handles.Y4avK;  Y4avE=handles.Y4avE;
Sj4av =handles.Sj4av ;  M4av = handles.M4av  ;
L4av=handles.L4av;  St4av = handles.St4av;  T4av  = handles.T4av ;
Pant4av=(handles.Pant4av)'; So4av =handles.So4av ;
group={}; names={};
if get(handles.Participant, 'Value');  group=[group, Pant4av];
names=[names 'Ppt']; end
if get(handles.AvSource, 'Value');  group=[group, So4av]; names=[names
'Srce']; end
if get(handles.AvSub, 'Value');  group=[group, Sj4av]; names=[names
'Subj']; end
if get(handles.AvMov, 'Value');  group=[group, M4av];  names=[names
'Movt']; end
if get(handles.AvLeg, 'Value');  group=[group, L4av];  names=[names
'Leg'];  end
if get(handles.AvSite, 'Value'); group=[group, St4av]; names=[names
'Site']; end
if get(handles.AvTim, 'Value');  group=[group, T4av];  names=[names
'Time']; end
Dim=1:length(names);
try
    [~, tableE, statsE]=anovan(Y4avE, group, 'varnames', names,
'display', 'off', 'model','interaction',  'sstype',2 );
    [~, tableK, statsK]=anovan(Y4avK, group, 'varnames', names,
'display', 'off', 'model','interaction',  'sstype',2 );
catch
    disp('Stats Not Available .. Cuss Now & Try Again Later');
    return
end
[cE,~,~,~] =
multcompare(statsE,'display','off','ctype','bonferroni','dimension',Di
m );
[cK,~,~,Nms] =
multcompare(statsK,'display','off','ctype','bonferroni','dimension',Di
m);
set(handles.AnovaTableK, 'data', tableK)
set(handles.AnovaTableE, 'data', tableE)
Switches={....
    'Leg=1' 'MovAny';  'Leg=2' 'MovL';'Leg=3' 'MovR';'Leg=4'
'MovInj';'Leg=5' 'MovUn';....
    'Time=1' 'Point 1'; 'Time=2' 'Point 2';'Time=3' 'Point 3';.....
```

```
    'Site=1' 'Left' ;'Site=2' 'Right';'Site=3' 'Inj' ;'Site=4' 'Uninj'
;'Site=5' 'Moving' ;'Site=6' ' NotMov';......
    'Movt=1' 'Std&Lie' ;'Movt=2' 'Stand'; 'Movt=3'  'Lie';.....
    'Srce=1' 'A&B'; 'Srce=2' 'GrpA';'Srce=3' 'GrpB';....
    'Subj=1' 'All'; 'Subj=2' 'Inj'; 'Subj=3' 'UnInj'};
for q=1:length(Switches)
    Nms=regexprep(Nms, Switches{q,1}, Switches{q,2});
end
for EK=1:2;
```

Post hoc testing

```
    if EK==1; c=cE; else  c=cK; end
    R=size(c,1);
    BonTable={};
    q=0;
    for r=1:R
        if sign(c(r,3))==sign(c(r,5));
            q=q+1;
            BonTable(q,1)=Nms(c(q,1));
            BonTable(q,2)=Nms(c(q,2));
            BonTable(q,3)={num2str(c(q,4))};
            if sign(c(q,3))==sign(c(q,5)); Sig='Yes'; else Sig='No';
end
            BonTable(q,4)={Sig};
        end
        if q==0; BonTable(1,1:4)={'          No' 'significantly'
'different' 'pairs'}; end
    end
    if EK==1; set(handles.BonFerr, 'data', BonTable); else
set(handles.BonFerrK, 'data', BonTable); end
end
```

Performing the file output – writing to Excel file

```
function pushbutton2_Callback(hObject, eventdata, handles)
FileName=get(handles.ReportFile, 'String');
Subjects=handles.Subjects; MoveMent=handles.MoveMent;
Leg=handles.Leg; Site=handles.Site; Source=handles.Source;
TG=get(handles.Time, 'Value');
GroupNames={'Blue' 'Red' 'Green'};
SjLabs={'All' 'Injured' 'Uninjured'};
MvLabs={'Any' 'One Leg Bend' 'Bent Knee Fall Out' 'Straight Leg Raise'
'Hip Flexion' 'QuickHipFlexion'};
LgLabs={'Either' 'Left' 'Right' 'Dominant' 'Non-Dominant' 'Injured'
'Un-injured'};
StLabs={'Left' 'Right' 'Injured' 'Un-injured' 'Moving' 'Not Moving'};
TmLabs=cell(7,4);
TmLabs(1,1)={'Point 1'};
TmLabs(2,1:1)={'Range 1>2'};
TmLabs(3,1:2)={'Point 1' 'Point 2'};
TmLabs(4,1:2)={'Range 1>2' 'Range 2>3'};
TmLabs(5,1:3)={'Point 1' 'Point 2' 'Point 3'};
TmLabs(6,1:3)={'Range 1>2' 'Range 2>3' 'Range 3>4'};
TmLabs(7,1:4)={'Point 1' 'Point 2' 'Point 3' 'Point 4'};
```

```
SrceLabs={'All data' 'AmFootball' 'ProFootball' 'Rugby' 'Frisbee'
'Hockey' 'H+P+R' 'A+F' 'A+P' 'A+P+R' 'H+R' 'A+P+F' 'Patient'};
ReportE =get(handles.Results, 'data');
ReportK =get(handles.ResultsK, 'data');



col=0;
RowHeads={'Time'; 'Graph Colour';'Source'; 'Subject
group';'Movement';'Moving leg is';
          'Position of leg';'EMG
Source/Angle';'N';'Mean';'SEM';'';'Data Listing'};
OutTabGrouped(1:length(RowHeads),1)= RowHeads;
AA=handles.XLTab;
[kk jj]=size(AA);
for j=1:jj
    Group=GroupNames{j};
    if ~(Subjects(j)==4)
        for k=1:kk
            col=col+6;
            OutTabGrouped(9:11, col)=ReportE([1 3 4],k+(j-1)*5);
            OutTabGrouped(9:11, col+1)=ReportE(5:7,k+(j-1)*5);
            OutTabGrouped(9:11, col+2)=ReportK([1 3 4],k+(j-1)*5);
            OutTabGrouped(9:11, col+3)=ReportK(5:7,k+(j-1)*5);
            aa=AA{k,j}; [rr cc]=size(aa);
            OutTabGrouped(13+(1:rr), col-2+(1:cc))=num2cell(aa);
Labs={TmLabs{TG,k}; Group; SrceLabs{Source(j)};
SjLabs{Subjects(j)};.........
                  MvLabs{MoveMent(j)}; LgLabs{Leg(j)};
StLabs{Site(j)}; char(handles.MusName)};
            OutTabGrouped(1:8, col)=Labs;
            OutTabGrouped(8, col+2)={char(handles.AngName)};
        end
    end
end
a=1;
xlswrite(FileName, OutTabGrouped, 'Grouped data', 'C5')
```

Collecting all of the used functions in one place

```
function radiobutton3_Callback(hObject, eventdata, handles)

function Subject1_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function Movement1_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function Leg1_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
```

```
function Segment1_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function Time_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function Site1_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function SourceA_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function Subject2_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function Movement2_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function Leg2_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function Site2_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function Muscle2_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function Subject3_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function Movement3_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function Leg3_CreateFcn(hObject, eventdata, handles)

if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
```

```
    set(hObject,'BackgroundColor','white');
end
function Site3_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function ReportFile_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function LastRec_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end
function SourceB_CreateFcn(hObject, eventdata, handles)
function Source1_CreateFcn(hObject, eventdata, handles)
function Source2_CreateFcn(hObject, eventdata, handles)
function Source3_CreateFcn(hObject, eventdata, handles)
function Muscle1_CreateFcn(hObject, eventdata, handles)
function Names_CreateFcn(hObject, eventdata, handles)
if ispc && isequal(get(hObject,'BackgroundColor'),
get(0,'defaultUicontrolBackgroundColor'))
    set(hObject,'BackgroundColor','white');
end

function axes6_ButtonDownFcn(hObject, eventdata, handles)
cases=handles.GList(Vlasic et al.) ;
gname(cases)

function GroupStatsVis_Callback(hObject, eventdata, handles)
ON=get(hObject, 'Value');
if ON
    set(handles.Results, 'Visible', 'on')
    set(handles.ResultsK, 'Visible', 'on')
    set(handles.text32, 'Visible', 'on')
else
    set(handles.Results, 'Visible', 'off')
    set(handles.ResultsK, 'Visible', 'off')
    set(handles.text32, 'Visible', 'off')
end

function UnpTVis_Callback(hObject, eventdata, handles)
ON=get(hObject, 'Value');
if ON
    set(handles.Ttests, 'Visible', 'on')
    set(handles.TtestsK, 'Visible', 'on')
    set(handles.text33, 'Visible', 'on')
else
    set(handles.Ttests, 'Visible', 'off')
    set(handles.TtestsK, 'Visible', 'off')
    set(handles.text33, 'Visible', 'off')
end

function PaiTVis_Callback(hObject, eventdata, handles)
ON=get(hObject, 'Value');
```

```
if ON
    set(handles.PairedTests, 'Visible', 'on')
    set(handles.PairedTestsK, 'Visible', 'on')
    set(handles.text34, 'Visible', 'on')
else
    set(handles.PairedTests, 'Visible', 'off')
    set(handles.PairedTestsK, 'Visible', 'off')
    set(handles.text34, 'Visible', 'off')
end


function AnoVis_Callback(hObject, eventdata, handles)
ON=get(hObject, 'Value');
if ON
    set(handles.AnovaTableE, 'Visible', 'on')
    set(handles.AnovaTableK, 'Visible', 'on')
    set(handles.BonFerr, 'Visible', 'on')
    set(handles.BonFerrK, 'Visible', 'on')
    set(handles.text35, 'Visible', 'on')
     set(handles.text38, 'Visible', 'on')
      set(handles.text113, 'Visible', 'on')
     set(handles.text114, 'Visible', 'on')
else
    set(handles.AnovaTableE, 'Visible', 'off')
    set(handles.AnovaTableK, 'Visible', 'off')
    set(handles.BonFerr, 'Visible', 'off')
    set(handles.BonFerrK, 'Visible', 'off')
        set(handles.text35, 'Visible', 'off')
     set(handles.text38, 'Visible', 'off')
      set(handles.text113, 'Visible', 'off')
     set(handles.text114, 'Visible', 'off')
end
```

Appendix 13

```
% This function processed EMG:  filtered, rectified and smoothed.

% Filtering: needs two functions: NotchFilter and PassFilter
% Rectifying: Prof's idea of rectifying the negative values according
to
% the mean of de- and re-polarisation, rather than according to zero.

% Smoothing: according to the window size depending on the sample
% frequenct, but being always 0.1 sec (also following Prof - but maybe
needs to be checked)


function proEMG = EMGprocessing(rawEMG, SF)
% for TMSI following Prof
% windowSize is 0.1 sec (depending on the smapling frequency of the
input data)

windowSize=0.1*SF;


[FiltData1]=NotchFilter (rawEMG,50,SF);% 50 here - what frequency is
desired to be filtered out
[FiltData2]=PassFilter (FiltData1,[10 400],SF);
bls=mean(FiltData2(1000:length(FiltData2),:),1);
rectEMG=abs(FiltData2-repmat(bls,length(FiltData2),1));

proEMG=filter(ones(1,windowSize)/windowSize,1,rectEMG);
```

Functions called by this programme

1. NotchFilter

```
%% Notch filter
% input DirtyData = dirty data,
%       cutoo = target cutting frequency,
%       fs= sampling rate
%
function [FiltData]=NotchFilter(DirtyData,cutoff,Fs)

            Time=((1:length(DirtyData))-1)/Fs;
            Raw=timeseries(DirtyData,Time);
            Ints=[cutoff-2 cutoff+2]; % the frequency intervals, in
hertz, for filtering the data:
            Filt = idealfilter(Raw,Ints,'notch');
            FiltData=Filt.Data;
            figure
            plot(Time, DirtyData);
            hold on
            plot(Time, FiltData, 'r');
```

2. PassFilter

```
%% Band-Pass filter
% input DirtyData = dirty data,
%       pass = target cutting frequency,
%       fs= sampling rate
%

function [FiltData]=PassFilter(DirtyData,pass,Fs)

            Time=((1:length(DirtyData))-1)/Fs;
            Raw=timeseries(DirtyData,Time);
            Ints=pass; % the frequency intervals, in hertz, for
filtering the data:
            Filt = idealfilter(Raw,Ints,'pass');
            FiltData=Filt.Data;
            figure
            plot(Time, DirtyData);
            hold on
            plot(Time, FiltData, 'r');
```