



**Is Vertical Stiffness Associated with Common Traumatic
Musculoskeletal Injuries in the Football Codes? An
Observational Study with a Focus on Anterior Cruciate
Ligament Injury**

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STATEMENT OF ORIGINAL WORK

I declare that this submission is my own work and that, to the best of my knowledge, it contains no material previously published or written by another person except where acknowledged in the text. Nor does it contain material that has been accepted for the award of another degree or diploma from any university.

Signed, Benjamin Gordon Serpell

Date:

ACKNOWLEDGEMENTS

I started my education to be a researcher by completing an honours dissertation in my final year of my undergraduate degree for Occupational Therapy. I recall two very important passages in that time; firstly, that I selfishly chose to write a dissertation because I did not want to do the alternative which was to complete a research project as part of a group assignment. How things have changed!! I've come to realise that collaboration is the key to success whether that be in research or practice. Secondly, that my then supervisor, Professor Karen Stagnitti from Deakin University, stated that she wasn't convinced that I would be successful in completing my honours dissertation because she thought I'd lost interest in studying but she was willing to "give me a go". She was right, nevertheless my competitive personality took over and I was committed to proving her wrong. I'm not sure the intended meaning behind Karen's statement; whether it was her genuine belief or if it were used to motivate me. Regardless, I must thank her for starting me on the path to where I am now. Without her faith in me back then I would unlikely have been given the opportunity to complete this piece of work.

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ABSTRACT

Injury to the anterior cruciate ligament (ACL) is a career limiting event for many sports people. Irrespective of a person's activity level and whether they undergo surgery or conservative rehabilitation, ACL injury can lead to osteoarthritis later in life and other comorbidities. ACL injury rates remain high despite considerable research to reduce its incidence. Therefore, novel methods for preventing ACL injury are needed.

In a published literature review presented in the introduction of this thesis it is noted that ACL injury incidence is greater in athletic populations, particularly in field and court sports, with approximately 50-80% of those being non-contact in nature. It showed little disagreement exists about the importance of dynamic knee joint stability for reducing ACL injury risk. To date, little research into dynamic knee joint stability has been conducted. This could be because of difficulty in measuring knee joint stability dynamically *in-vivo*, or due to difficulty identifying novel practices/methods which incorporate dynamic knee joint stability. This work proposed musculotendinous stiffness, or 'stiffness' as it is referred to in this thesis, as novel, relevant and worthy of investigation.

A second literature review defined stiffness as a quantification of resistance against force. Specifically, vertical stiffness was described as the body's resistance to vertical displacement from ground reaction force, affected by the interaction of connective tissue, muscle and bone, as well as stiffness at each joint. Therefore, this thesis addresses the question "is vertical stiffness associated with common traumatic musculoskeletal injuries in the football codes?". A novel method of measuring dynamic knee joint stability and a surrogate measure of ACL loading *in-vivo* was applied

which involved image registration of computed tomography with fluoroscopy to build a 4-D model of knee joint motion.

This thesis is a compilation of published research papers. The study in chapter two showed hamstring and quadriceps pre-activation and co-activation is related to vertical stiffness for a task which simulates the manoeuvre typically observed when non-contact ACL injury occurs in field and court sports. Chapter three found no difference in vertical stiffness between a cohort of Australian Rules footballers who sustained a muscle strain injury and an uninjured cohort. This outcome was important because muscle inhibition can remain for 12 months following injury and, as identified in the previous study, thigh muscle function is important for vertical stiffness and, likely, knee joint stability. Chapter four used the novel technique described for measuring dynamic knee joint stability and ACL elongation *in-vivo* on a step-up task while also measuring hamstring and quadriceps activity. It argued hamstring and quadriceps co-activation is not associated with ACL elongation. Finally, chapter five used the same method to measure dynamic knee joint stability on a task similar to that used in chapter two and failed to find a link between vertical stiffness and ACL elongation.

This work found no evidence of an association between vertical stiffness and some common traumatic non-contact injuries. It concluded training programs which enhance vertical stiffness may be implemented without concern for injury. Suggestions for future research are also made.

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ABBREVIATIONS AND NOMECLATURE

(Symbols, then numerical order, then alphabetical order)

ΔL – Change in leg length

ω_0 – Natural frequency of a mass spring system representing a body

Δy – Displacement of centre of mass

ε - Constant

α – Leg angle relative to x-axis

α_0 – Leg angle relative to x-axis at touchdown

$\Delta\theta$ – Change in joint angle

W^- - Negative work

ω – Angular velocity

4-D – Four dimensional

ACL – Anterior cruciate ligament

AFL – Australian Football League

ATT – Anterior tibial translation

BF – Biceps femoris

BMI – Body mass index

COM – Centre of mass

CT – Computed tomography

d – Point of force translation distance

DF – Duty factor

EMG - Electromyography

F_{\max} – Maximum ground reaction force

g – Acceleration due to gravity

GRF – Ground reaction force

I – Body mass multiplied by length of the thigh squared

L_o – Leg length

L_{oA} – Actual leg length

L_{oR} – Relaxed leg length

K_{knee} – Knee stiffness

K_{leg} – Leg stiffness

K_{vert} – Vertical stiffness

m – Total body mass

M – Joint moment

MRI – Magnetic resonance imaging

ms – Milliseconds

MVC – Maximum voluntary contraction

NHMRC – National Health and Medical Research Council of Australia

NFL – National Football League

NWI – Notch width index

RMS – Root mean square

s – Horizontal velocity

SENIAM – Surface electromyography for the non-invasive assessment of muscles

SM - Semimembranosus

SPSS – Statistical package for social sciences

t – Time

t_c – Ground contact time

TD – Touch down

TEM – Typical error of measurement

t_f – Flight time

T_f – Time from take-off to touch down of same leg

TO – Take off

u – Vertical landing velocity or horizontal velocity of centre of mass

USA – United States of America

VL – Vastus lateralis

VM – Vastus medialis

CHAPTER 1

Introduction

1.1. Rationale

1.2. Mechanisms and Risk Factors for Noncontact ACL Injury in Age Mature Athletes Who Engage in Field or Court Sports: A Summary of the Literature Since 1980

1.3. Stiffness: A New Way of Thinking about Dynamic Stability?

1.4. A Review of Models of Vertical, Leg, and Knee Stiffness in Adults for Running, Jumping or Hopping Tasks

1.5. Aims of Thesis

1.1. RATIONALE

The anterior cruciate ligament (ACL) is one of the main ligaments of the knee (Butler et al., 1980), and without it the knee is rendered unstable (Tashman et al., 2007). It is commonly injured in sports and recreation activities and this injury can be career limiting for elite and professional sports people, or activity limiting for the general population (Tashman et al., 2007, Muaidi et al., 2007, Bjordal et al., 1997, Dallalana et al., 2007). As the ACL is so important to knee stability and function considerable research has been conducted into how it is injured, how injury to the ACL can be prevented, the potential amelioration of the injury, and the short, medium and long term consequences of ACL injury (Renstrom et al., 2008). The introduction to the thesis will describe the ligament and its importance in knee stability. It will describe the current research into the mechanisms of injury, with implications for prevention. It will then explore modelling knee stability in a relatively novel way, which could underpin explanations of knee stability or lack thereof. This introduction provides the basis on which to build the thesis.

The main function of the ACL is to constrain anterior displacement of the tibia relative to the femur in the knee (Butler et al., 1980). It is, however, supported in this function by surrounding structures such as the iliotibial band, capsular ligaments and the medial and lateral collateral ligaments as well as functioning skeletal muscle (Butler et al., 1980, Opar and Serpell, 2014). When the knee is fully extended the ACL is taut and it may therefore also assist to limit medial and lateral translations, varus/valgus movement, and rotations about the long axis (Bendjaballah et al., 1997, Brantigan and Voshell, 1946). It is easy to appreciate these mechanical functions of the ACL when one considers its location in the knee and its attachments. The proximal attachment of the ACL is on the medial wall of the lateral femoral condyle (Zantop et al., 2006) and the distal attachment attaches slightly anteriorly to the peak of the medial spine on the tibial plateau (Ferretti et al., 2012) (see figure 1).

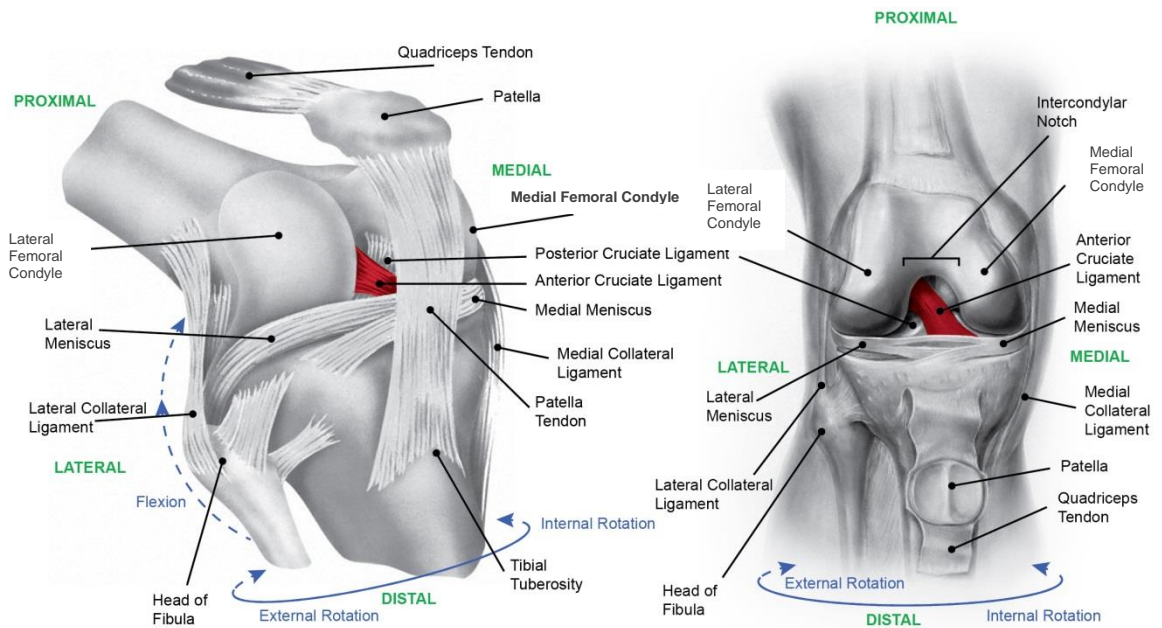


Figure 1. The anterior cruciate ligament in the knee and surrounding structures (adapted from Rosse and Gaddum-Rosse, 1997).

Note: this image appears again later in this thesis as a part of a published paper

Injury to the ACL can be severe and costly. Physical function is impaired acutely and many people who sustain an injury to the ACL develop osteoarthritis in the knee as a consequence (Ajuied et al., 2014, Tashman et al., 2007, Lohmander et al., 2004, Oiestad et al., 2010). More specifically, injury to the ACL will result in a period of inactivity (Gabbe et al., 2004, Deacon et al., 1997, Muaidi et al., 2007) and it may even lead to early retirement for many athletes irrespective of whether the injury is managed conservatively or with knee reconstructive surgery (Gabbe et al., 2004, Deacon et al., 1997, Muaidi et al., 2007). In fact recent research has shown that only 65% of athletes who sustain an ACL injury remain competing at the same level three years later (Walden et al., 2016). Research suggests permanent alterations to knee joint motion following rehabilitation from ACL injury are likely, leading to increased wear of the articular surfaces of the knee and subsequently hastening degenerative change (Scarvell et al., 2006, Tashman et al., 2007, Scarvell et al., 2005, Ajuied et al., 2014, Wexler et al., 1998, Andriacchi et al., 2004, Andriacchi and Dyrby, 2005). The rate at which degenerative change occurs in the knee is exacerbated by being

overweight or obese which may stem from the inactivity associated with the acute ACL injury, which could also lead to ongoing inactivity and some comorbidities (Myer et al., 2014, Osterberg et al., 2013). Finally, degeneration of the knee can ultimately require joint replacement surgery (Dieppe et al., 2011). Thus, the short and long term costs of ACL injury may be felt by both the individual and the health care system if one considers both the impact of knee reconstructive and joint replacement surgery as well as the impact of other health disorders which may arise.

The incidence of ACL injury, particularly in field and court sports, remains high and professional sport appears to not be ‘immune’ from the problem despite having scope for increased care and ability to implement prevention strategies. Mainstream media reported that by the start of November 2015, a total of 38 ACL injuries had been sustained by players in the National Football League (NFL) in the United States of America since the commencement of training camps that year (Steinberg, 2015). Training camps for some teams start toward the end of June. That equates to over two ACL injuries per week within the NFL over a 14-15 week period. The report stated that this was the highest number of this type of injury ever sustained in the competition (Steinberg, 2015). The Australian Football League (AFL) in their 2016 injury surveillance report of ten years leading up to and including the 2015 season reported an increasing trend in ACL injury incidence. In 2015 the incidence was 0.7 per club and was less (0.6 per club) in only 2007 and 2010 (Orchard and Seward, 2016). That equates to approximately 12 new ACL injuries per season across the competition. Given the AFL season runs for twenty-three weeks and starts in March each year, that amounts to nearly one new ACL injury every two weeks. While these statistics for ACL injury incidence have been reported in mainstream media and commissioned reports where ‘quality control’ for research may be questionable, it is supported by recent work published in internationally peer reviewed scientific journals which has shown that ACL injury incidence in professional football may not have changed since the turn of the century (Walden et al., 2016).

Given the consequences and subsequent costs associated with ACL injury, and that the incidence appears unchanged over time, investigating novel strategies of reducing ACL injury risk is necessary. However, to do so first requires a sound understanding of what is already known about the common mechanisms and risk factors for ACL injury. The next section of this chapter presents a literature review which describes and discusses the epidemiology of ACL injury, the common mechanisms and risk factors for ACL injury, and the direction for future research. This literature review was published in the *Journal of Strength and Conditioning Research* as 'Mechanisms and risk factors for noncontact ACL injury in age mature athletes who engage in field or court sports: A summary of the literature since 1980'.

**1.2. MECHANISMS AND RISK FACTORS FOR
NONCONTACT ACL INJURY IN AGE MATURE
ATHLETES WHO ENGAGE IN FIELD OR COURT SPORTS:
A SUMMARY OF THE LITERATURE SINCE 1980**

Statement confirming the authorship contribution of the PhD candidate

On behalf of all co-authors of the paper:

SERPELL, B.G., SCARVELL, J.M., BALL, N.B. & SMITH, P.N. 2012. Mechanisms and risk factors for noncontact ACL injury in age mature athletes who engage in field or court sports: A summary of the literature since 1980. *J Strength Cond Res*, 26, 3160-176.

I confirm that Benjamin Serpell has made the following contributions:

- Searching and collection of the literature
- Analysis of the literature and synthesis of concepts under supervision
- Wrote the first draft of the manuscript, and followed through to publication including proofing and final publication details of the manuscript

Professor Paul N. Smith Signed:..... Date:.....

MECHANISMS AND RISK FACTORS FOR NONCONTACT ACL INJURY IN AGE MATURE ATHLETES WHO ENGAGE IN FIELD OR COURT SPORTS: A SUMMARY OF THE LITERATURE SINCE 1980

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ABSTRACT

Serpell, BG, Scarvell, JM, Ball, NB, and Smith, PN. Mechanisms and risk factors for noncontact ACL injury in age mature athletes who engage in field or court sports: A summary of literature since 1980. *J Strength Cond Res* 26(11): 3160–3176, 2012—Epidemiological data show that in the last 10 years alone the incidence and rate of anterior cruciate ligament (ACL) injuries have not changed appreciably. Furthermore, many ACL injuries appear to be noncontact in nature and sustained while engaging in some field or court sport. Thus, the need to investigate novel methods and adopt training strategies to prevent ACL injuries is paramount. To do so, however, requires an understanding of the mechanisms and risk factors for the injury. The aim of this review was to investigate the mechanisms and risk factors for noncontact ACL injuries in age mature athletes who compete in field or court sports. A search of the entire MEDLINE database for biomedicine was performed, and an iterative reference check was also conducted. A total of 87 articles disclosed met the eligibility criteria. Articles were grouped into ‘themes’; ‘anatomical and biomechanical mechanisms and risk factors,’ ‘intrinsic mechanisms and risk factors,’ and ‘extrinsic mechanisms and risk factors.’ In this review, it is concluded that there are still a number of risk factors and mechanisms for noncontact ACL injury that are not well understood. However, the importance of dynamic knee joint stability is highlighted. It is also suggested that novel methods for preventing ACL injury be investigated and developed.

KEY WORDS knee, anterior cruciate ligament, trauma, cause

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INTRODUCTION

Anterior cruciate ligament (ACL) injury is a serious knee injury, which may impair functional ability (13,27,72,96,97,100) and is relatively common among active people worldwide. At the turn of the century, Griffin et al. reported an incidence of 80,000 ACL injuries per annum in the U.S.A. (39). More recently, an International Olympic Committee current concepts statement reported on similar data from European and Scandinavian nations, estimating an incidence of 34–80 noncontact ACL injuries per 100,000 persons in the general population over an 18-month period (92). Comparable data from New Zealand were also recently published (37). These data suggest that ACL injury incidence has not changed appreciably over time, and the notion is supported by data from the National Collegiate Athletics Association in the U.S.A. In the mid-1990s, ACL injury rates of 0.13 and 0.31, and 0.07 and 0.29 per 1,000 athlete exposures in men and women in basketball and soccer, respectively, were reported (4). Repeat studies conducted approximately 10 years later revealed similar results; an ACL injury rate of 0.11 and 0.27, and 0.11 and 0.27 per 1,000 athlete exposures in men and women in basketball and soccer, respectively, was observed in 1 study published in 2005 (2), and in another published in 2006, an incidence rate of 0.08 and 0.28, and 0.11 and 0.32 per 1,000 athlete exposures was observed (70).

Given the apparently unchanged incidence and injury rates, the need to investigate novel methods and adopt training strategies to prevent ACL injuries is paramount. To do so effectively requires first evaluating ACL injury risk factors (81). Anterior cruciate ligament injury in athletic populations is more common than in the general population (37,92,104), and they more commonly occur in field and court sports than in snow sports (34,76,104). Approximately, 50–80% of all ACL injuries in field and court sports are noncontact in nature (4,5,15,24,38,68,73–75,78,79,81,82,86,88,89,93,104).

The aim of this study was to review the literature describing mechanisms and risk factors for noncontact ACL injury for

athletes who engage in a field or court-based sport. We chose not to compare risk factors and mechanisms across sports. Articles were limited to those published since 1980 and articles with samples from age mature populations, because adolescents and children may respond to different risk factors or mechanisms of injury as a result of different hormonal and growth profiles (47,90). We chose to review the literature in this select population because of the implications findings may have for the physical preparation of amateur and professional athletes.

METHODS

Experimental Approach to the Problem

This study was a general literature review; however, a systematic approach to retrieving articles was adopted. Thus, rather than analyzing data from studies that were retrieved as would be the case in a systematic review, the results were summarized and strengths and limitations were discussed as were practical implications relevant to the physical preparation of athletes. Figure 1 summarizes the search strategy and how data were summarized in the Results section of this article.

Search Strategy

A search of the MEDLINE bibliographic database for biomedicine was performed using the following search terms and Boolean operators:

- (“*cruciate ligament” AND injur* AND caus*) NOT (posterior OR PCL)

The following restrictions were applied:

- Documents written in English only
- Published between the years 1980 and September 2011
- Limited to original articles and review articles only
- Limited to articles with human subjects only
- No duplicates

The search disclosed 1,479 articles, and the abstracts were reviewed for eligibility. From the review of abstracts, 114 articles that had the potential to meet eligibility requirements were read in full. Fifty-one met the eligibility criteria. An iterative reference check of eligible articles was performed. Articles that had been cited ≥ 5 times by eligible articles published before 2006, or ≥ 3 times by eligible articles published after 2006, were subsequently reviewed and included in this study provided they too met the eligibility criteria.

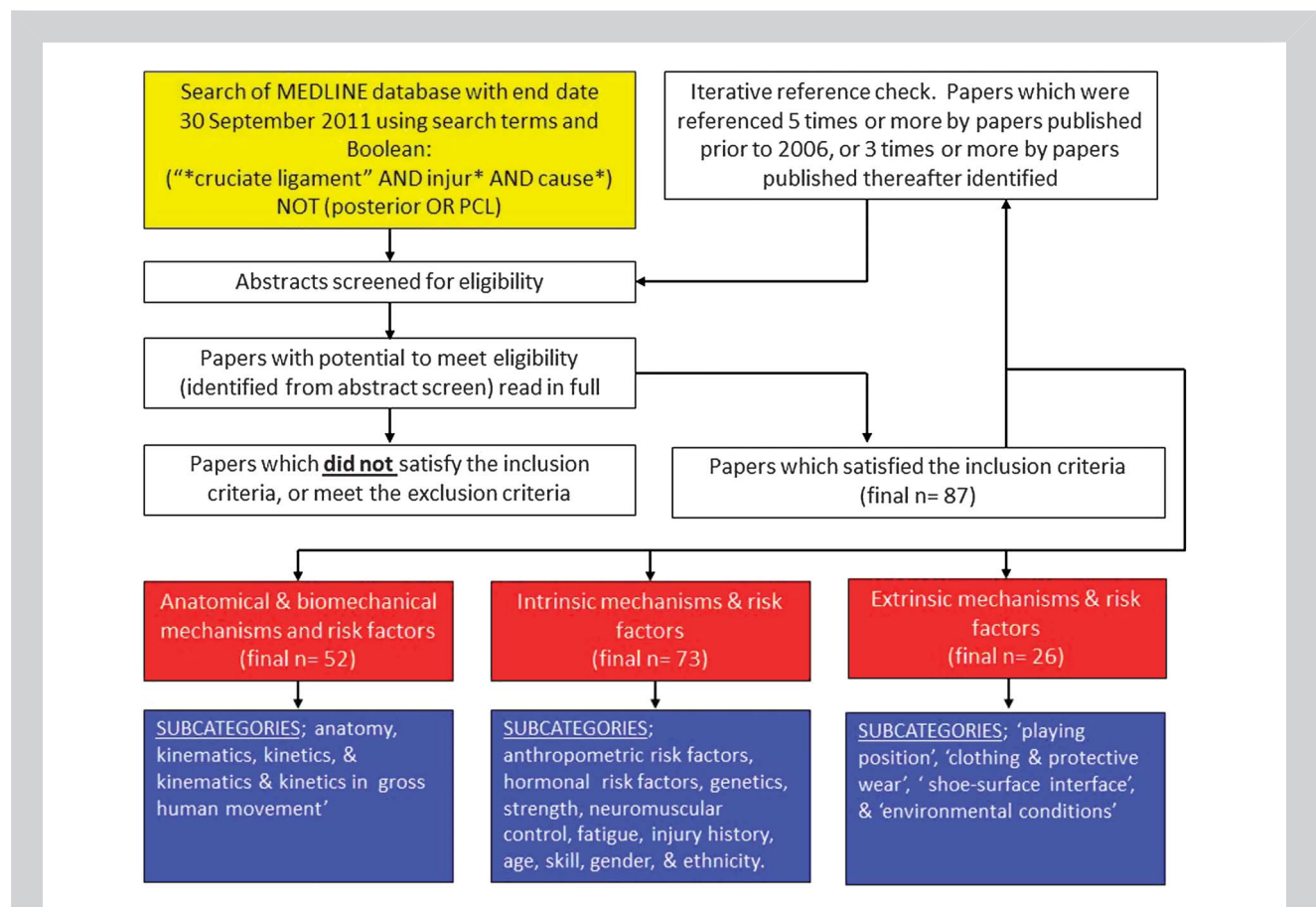


Figure 1. Summary of search strategy and subsequent grouping of articles for analysis and discussion. A number of articles were discussed in several ‘subcategories.’

Eligible articles identified in the initial iterative reference check were included in subsequent iterative reference checks. The iterative reference check process was repeated until exhaustion. No article published in 1979 was disclosed. In total, 87 articles published between the 1980 and September 2011 met the eligibility criteria.

Eligibility Criteria

Inclusion Criteria. Original articles, or systematic reviews that satisfied the definition of level 1 evidence by the National Health and Medical Research Council of Australia (NHMRC), were included. The NHMRC define level 1 evidence as a systematic review of randomized controlled trials or prospective cohort studies (26).

Articles were considered to be eligible for inclusion if their sample was from an adult population (i.e., mean sample age of ≥ 18 years).

Articles that discussed the effect of one risk factor on another were included in the present review. For example, knee joint laxity has been implicated as being a predictor for ACL injury (6,19,84,85); therefore, studies that discussed the effects of hormones on knee joint laxity were included.

Exclusion Criteria. To limit articles to field and court sports, articles describing ACL injuries from snow sports (e.g., skiing, snowboarding) and artistic sports (e.g., ballet, gymnastics) were excluded.

Studies limited to contact or indirect contact ACL injuries only were excluded. Noncontact ACL injuries are those that occur when no contact is made to the lower body of the athlete when the injury is sustained (78,81,93).

Data Extraction/Grouping of Articles

Eligible articles were grouped according to themes 'anatomical and biomechanical mechanisms and risk factors,' 'intrinsic mechanisms and risk factors,' and 'extrinsic mechanisms and risk factors.' A mechanism was considered anything that 'caused' injury to the ACL (78,93). Risk factors were considered any 'condition' that affects the likelihood of ACL injury (81). Articles were grouped by intrinsic and extrinsic risk factors; intrinsic risk factors are risk factors that are personal in nature, and extrinsic risk factors are environmental (81).

Articles that examined anatomical and biomechanical mechanisms and risk factors were divided into the subcategories of 'anatomy,' 'kinematics,' 'kinetics,' and 'kinematics and kinetics in human movements.' Anatomical articles could be included in this theme and intrinsic risk factor group. This group comprised 52 articles.

Articles that examined intrinsic mechanisms and risk factors were divided into the subcategories, 'anthropometric risk factors,' 'hormonal risk factors,' 'genetics,' 'strength,' 'neuromuscular control,' 'fatigue,' 'injury history,' 'age,' 'skill/,' 'gender,' and 'ethnicity.' This group comprised 73 articles.

Articles that discussed extrinsic mechanisms and risk factors were grouped into the subcategories 'playing position,'

'clothing and protective wear,' 'shoe-surface interface,' and 'environmental conditions'. This group comprised 26 articles.

RESULTS

Anatomical and Biomechanical Mechanisms and Risk Factors

Anatomy. Anterior cruciate ligament anatomy can be seen in Figure 2 (94). Eleven studies retrieved in the present review examined a connection between knee joint anatomy and ACL injury.

Intercondylar notch width and ACL injury were investigated in 2 studies. Notch width index (NWI) is a ratio of intercondylar notch width to femoral condyle width (35,58). One study that calculated NWI from measurements taken from x-ray films in a unilateral ACL deficient sample found NWI to be typically smaller in the injured knee compared with the noninjured knee (58). The other calculated NWI using magnetic resonance imaging (MRI) but found no relationship between NWI and elongation of the ACL when the knee was moved through kinematics believed to injure the ligament (35).

Five studies retrieved implicated knee joint laxity as a risk factor for ACL injury (6,7,19,84,85). Knee joint laxity can be passive or active. Passive laxity is the 'amount' of passive motion observed in any plane or rotation before plateauing of a displacement tension curve (14). Active laxity is the motion observed in a plane or rotation during active movement that is not associated with the primary movement (14). For example, some anterior-posterior displacement of the femur over the tibia can be observed during knee flexion; the primary movement is flexion, the amount of anterior-posterior displacement observed is the active laxity. One cadaveric study showed more passive knee joint laxity, determined by measuring anterior tibial translation, in knees with complete ACL rupture (19). Several other controlled laboratory studies showed that knees with greater passive laxity could more easily reach the kinematic extremes associated with ACL injury (84,85).

A number of factors contribute to knee joint laxity including hormones, neuromuscular control, and other anatomical structures. Of particular interest here is the role of other structures surrounding the knee. In one cadaveric study that retrieved the effects of the iliotibial band, capsular ligaments and the medial and lateral collateral ligaments on passive knee joint laxity was measured (19). This showed that the ACL provided most resistance against anterior tibial translation and those surrounding structures acted as secondary restraints (19). However, the relative contribution of each of those secondary restraints did not differ significantly between each other, and the contribution of each structure was minimal. Similar findings have been found elsewhere (6,7). Skeletal muscle may also act as a surrounding structure that can affect knee joint laxity. According to one retrospective survey that was reviewed, hamstring flexibility and knee recurvatum were typically greater in ACL-injured patients

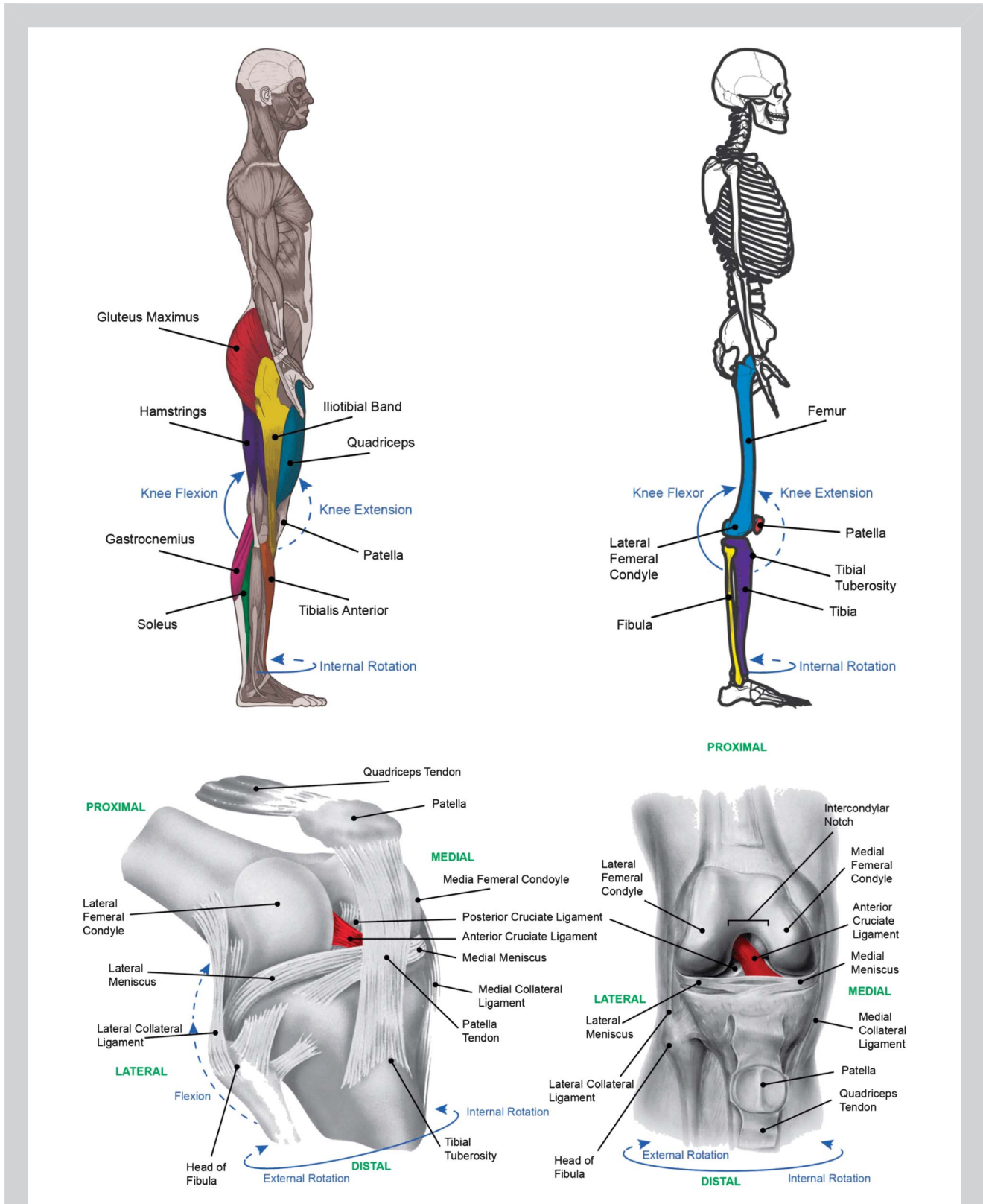


Figure 2. The anterior cruciate ligament and surrounding structures. The anterior cruciate ligament (ACL) attaches distally to the anterior aspect of the intercondylar eminence on the tibial plateau and passes posteriorly through the intercondylar notch to attach proximally to the posteromedial aspect of the lateral condyle of the femur (94).

(15). Another comparative cohort study showed greater tibialis anterior volume and cross-sectional area to exist in the injured leg of unilateral ACL deficient people was observed (11).

The only other anatomical risk factor for ACL injury discussed in the literature retrieved concerned micro-anatomy. In an immunohistological study, the presence of relaxin receptors in the ACL was observed but not in other ligamentous structures of the knee (36).

Kinematics. Kinematics is the description of human motion independent of the forces that cause the movement (103). In studies disclosed in this review, methods used to assess the kinematics of ACL injury included retrospective surveys (15,68,104), video analyses (15,24,78), and controlled laboratory studies (6,31,35,50,54,60,91). The participants in the retrospective surveys typically recalled a low knee flexion angle (15,68,78), knee joint rotation (68,78,104), and valgus collapse (78) when they injured their ACL.

Several studies that adopted a descriptive epidemiological video analysis method (15,24,78) had outcomes similar to those of the retrospective surveys; a low knee flexion angle (15,24,79), knee valgus (15,24,81), and in 1 study (15) minimal knee rotation (15,81) was typically seen when non-contact ACL injuries occurred. Notably, in one study, external rotation was not typically observed when the ACL ruptured, only internal rotation (24).

A number of laboratory studies in this review described knee joint kinematics likely to lead to ACL injury (6,31,35,50,54,57,60,87,91). Five cadaveric studies showed that ACL strain or anterior tibial translation increased with low flexion angles combined with knee rotation and valgus 'stress' (6,31,50,60,91). Two in vivo studies showed that anterior tibial translation was the greatest between 18° and 27° knee flexion (54) and stress placed on the ACL increased with varus-valgus movement (57). Finally, an MRI study revealed that impingement of the ACL against the lateral wall of the intercondylar notch can occur with external rotation, particularly when combined with valgus movement; elongating the ligament and putting it at a greater risk of rupture (35).

Kinetics. Kinetics is the description of forces that influence motion (103). Concerning noncontact ACL injuries, 2 types of forces should be considered: ground reaction forces (GRFs) and moments. The GRFs are the equal and opposite linear forces applied from the ground while weight bearing (103). Moments are the net forces of all ligaments and agonist and antagonist muscles that result in segment motion (103).

According to the literature reviewed, GRFs generated on impact with the ground likely contribute to noncontact ACL injury. One descriptive epidemiology study reported that noncontact ACL injuries typically occur when landing from a jump or when cutting, pivoting, or changing direction (15,24,34,74,75,78,79,81,93,104). Laboratory studies reviewed showed that greater GRFs were observed throughout stance

phases for populations at a high risk of sustaining ACL injury (e.g., women) (52,98,106) and for higher risk landing tasks (e.g., unanticipated sidestep cutting maneuvers) (45,65,80). One recently published laboratory study even argued that GRFs may play a more significant role in noncontact ACL injury than do knee extensor moments (87).

Eighteen laboratory studies examined the effects of moments on anterior tibial translation or the kinematic variables believed to result in ACL injury (9,10,18,21–23, 31,32,45,52,57,61–64,87,106). With the exception of one study, the literature retrieved discussed flexor and extensor moments generated by hamstring and quadriceps muscles (9,10,18,21–23,31,32,45,52,57,61–64,87,106). In the study that did not discuss knee flexor and extensor moments, it was argued that the soleus may elicit a posterior stabilizing force on the tibia when the foot is fixed, protecting the knee against ACL injury (87). In the other articles retrieved, it was seen that more anterior tibial translation typically occurred with smaller knee flexor moments (21,52,106), and a number of computer simulation studies showed that the ACL would not typically injure with sagittal plane moments alone (57,63,64,67).

Kinematics and Kinetics in Human Movement. Noncontact ACL injuries are reported to occur on impact when landing from a jump or when changing direction (15,24,34, 74,75,78,79,81,93,104).

Sidestep cutting has been shown to be the change of direction maneuver most associated with ACL injury. In an Australian Rules football descriptive epidemiology study, sidestepping accounted for more ACL ruptures than did a 'land and step' change of direction or a crossover cut (24) (Figure 3). No research comparing kinetic or kinematic variables between the 'land and step' maneuver and any other cutting maneuver was available in this review.

Finally, laboratory work has shown that the kinematic extremes and knee joint moments associated with ACL injury are more commonly reached when changes of direction are unplanned (8,9,17,18,65).

Intrinsic Mechanisms and Risk Factors

Anthropometric Risk Factors. The term 'anthropometry' is derived from the Greek terms 'anthropos,' meaning man, and 'metria,' meaning measure. Therefore, anthropometry is the measurement of man (77).

Relatively few studies retrieved investigated a link between anthropometry and ACL injury. Nevertheless, research has shown greater body mass to be a risk factor for ACL injury (33,81), and the risk appears to be greater for taller people (33,81); body mass index (BMI) has also been reported to be linked to ACL injury (33,81), and tibialis anterior cross-sectional area has also been shown to be associated with ACL injury (11).

Hormonal Risk Factors. Thirteen studies examined the effect of hormones on ACL injury risk. Although the literature



Figure 3. Description of change of direction maneuvers. A) Sidestep; the athlete pushes off his right foot to change the direction to the left. B) Crossover cut; the athlete pushes off his left leg to change the direction to the left. C) Jump and step cut; the athlete approaches the point where he wants to pivot, jumps, and lands and "sidesteps" off his right leg to change direction left.

was somewhat varied, the menstrual cycle was divided into 4 phases; menses, and the follicular, ovulatory, and luteal phases. A summary of the timing and duration of each phase is shown in Figure 4 (23,28,44,46,73,83–85,104).

Findings from the studies that measured a relationship between menstrual cycle and ACL injury risk are summarized in Table 1.

Three histochemical studies specifically investigated the effect of hormones on the human ACL; one examined relaxin (36), another estrogen (107), and the other estrogen and progesterone combined (108).

In the histochemical study that investigated relaxin, the existence of relaxin receptors in human ACL was reported (36). Relaxin contributes to the elasticity of connective tissue and may also reduce its collagen content (36,104). Both men and women secrete relaxin; however, concentrations of relaxin are greater for women, particularly during pregnancy. Furthermore, relaxin concentration fluctuates throughout the menstrual cycle peaking during the luteal phase (36). Therefore, the presence of relaxin receptors in the ACL makes it more susceptible to the effects of relaxin particularly during the luteal phase of the female menstrual cycle. It should also be noted that this study reported that estrogen might modulate relaxin receptors (36).

In the other histochemical studies that investigated estrogen and progesterone, it was reported that procollagen I synthesis can be inhibited by estradiol ('synthetic' estrogen) in a dose-dependent manner (107,108). Procollagen is the 'precursor' to collagen. Furthermore, the administration of progesterone attenuated the

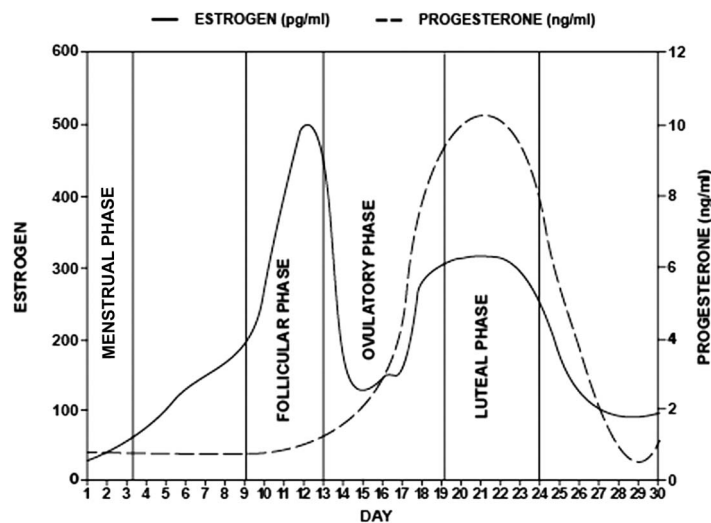


Figure 4. Estrogen and progesterone fluctuations throughout the menstrual, follicular, ovulatory, and luteal phases (44).

effect of estradiol regardless of progesterone dosage (108). However, progesterone administration in the absence of estradiol slightly increased procollagen I synthesis (108).

Genetics. This review disclosed 3 articles that examined a genetic link to ACL injury. Those studies showed that variants of the COL5A1 and COL1A1 genes were associated with ACL injury (53,88,89).

The COL5A1 gene codes for the $\alpha 1$ chain of type V collagen. Approximately 10% of the collagen content in ligaments is composed of type V collagen (89). One study reported a relationship between the COL5A1 BstUI genotype and ACL injury, noting that this genotype with the T allele was overrepresented in women who injured their ACL.

The COL1A1 gene regulates the production of type I collagen; another major constituent of cruciate ligaments. Several studies argued that those with the recessive homozygous genotype of the COL1A1 gene are believed to be at a decreased risk of sustaining ACL injury (53,88) because this variant facilitates increased collagen production that may result in greater ligamentous mechanical strength (88).

Strength. No study retrieved in the present review examined the effect of hamstring strength or knee joint strength ratios on ACL injury. One study described a training program that focused on hamstring, quadriceps, and gluteal strength (45), but it did not actually measure muscle strength; rather it assumed that increased strength in the gluteal and hamstring muscle groups was obtained after training. That study showed strength-alone training did not alter knee joint kinetics or kinematics for landing tasks, but strength training combined with skills training to landing 'safely' did (45).

Neuromuscular Control. Neuro-muscular control encompasses proprioception and timing, order and magnitude of muscle recruitment. Proprioceptive acuity refers to a person's ability to sense joint position and movement of segments relative to each other (71). Research in this area failed to find an association between proprioception and ACL injury risk. However, randomized controlled studies have shown that proprioceptive training can improve landing mechanics (86,99). Furthermore, except for one underpowered study (99), prospective cohort studies have revealed lower ACL injury rates in cohorts that have undergone proprioception training (20,73).

Recently, there has been a trend toward examining neuromuscular control specifically by measuring the order and magnitude of muscle recruitment (Table 2) (8,25,48,57,109).

Table 2 shows the importance of hamstring activation before foot strike for safer landings. This suggests that proprioceptive acuity is important for 'switching on' the hamstrings. This 'theory' is supported by one article retrieved in the present review, which showed that for unanticipated sidestepping tasks, 'safe' muscle recruitment strategies become compromised (8). Several training studies have shown that neuromuscular control adaptations can occur after intervention (22,38,45,80,86).

Fatigue. One descriptive epidemiology study showed that ACL injuries typically occur in the second half of a rugby match, when it is assumed players are typically more fatigued (27). A contrary study in team handball showed ACL injuries occurred in the first half (74). These epidemiological studies have produced conflicting results that have been explored further in the laboratory.

The effect of fatigue on the knee joint kinematics was studied in a number of laboratory studies and outcomes varied. Two in vivo laboratory studies showed no effect of fatigue on knee flexion angles (17,62). In one of those studies, a neuromuscular fatigue protocol was adopted (62), in the other, neuromuscular and mental fatigue was induced (17). In 2 other laboratory studies in which neuromuscular fatigue was induced, one showed a decline (21) and another showed an increase (52) in knee joint flexion angle throughout the contact phase of a jump landing or pivot. A greater valgus angle (17,62) was observed after a neuromuscular or mentally fatiguing protocol for a sidestep maneuver (17) and at peak

TABLE 1. Summary of studies investigating ACL injury risk throughout menstrual cycle.*

Reference	Level of evidence†	Summary of findings
Wojtys et al. (104)	Level 2 (retrospective case series)	A greater number of ACL injuries occurred during the luteal phase. The next greatest incidence of injuries was during the ovulatory phase. The least number of injuries occurred in the follicular phase.
Agel et al. (3)	Level 3-2 (cohort with retrospective and prospective arms)	No significant difference in the incidence of ACL injuries between the 'oral contraceptive' group and the 'nonoral contraceptive' group. The peak in injury incidence occurred 7-9 days postmenses onset.
Myklebust et al. (75)	Level 2 (prospective cohort)	Most injuries typically occurred within 7 days from the onset of menses or late in the luteal phase.
Myklebust et al. (73)	Level 2 (prospective cohort)	Most ACL injuries occurred during the menstrual phase, then follicular, then luteal phase. Trends in ACL injury incidence was the same for women using oral contraceptives and women not using oral contraceptives.
Heitz et al. (44)	Level 4 (prospective case series)	Mean passive knee laxity was greatest during the luteal phase, then follicular.
Park et al. (83)	Level 4 (prospective case series)	Passive knee laxity was typically greater during the ovulatory phase when compared with the luteal phase. Laxity during the ovulatory phase was also much greater than during the follicular phase.
Deie et al. (28)	Level 2 (prospective cohort)	Mean passive knee laxity was significantly greater in the ovulatory and luteal phases when compared with the follicular phase. No significant difference in mean anterior laxity between the ovulatory and luteal phases existed. Knee laxity for a male control group was performed at the same time measurements were taken for women. No difference in results was seen in men between trials.
Hertel et al. (46)	Level 4 (prospective case series)	No significant difference in knee flexion or extension peak torque, hamstring-quadriceps ratio, proprioception or passive knee joint laxity across the different phases of the menstrual cycle was observed.
Park et al. (84)	Level 4 (prospective case series)	Mean passive knee joint laxity during the ovulatory phase was greater than during the luteal phase. No significant differences in any knee joint kinetic or kinematic variables were observed.
Park et al. (85)	Level 4 (prospective case series)	No significant differences in knee joint kinetics or kinematics were observed across the different phases of the menstrual cycle.
Abt et al. (1)	Level 4 (prospective case series)	No significant differences for knee kinetics or kinematics, proprioception or hamstring-quadriceps ratios were observed between the different phases of the menstrual cycle.
Chaudhari et al. (23)	Level 2 (prospective cohort)	No significant differences for knee joint kinetics or kinematics were seen between the different phases of the menstrual cycle for women using oral contraceptive, women not using oral contraceptives, or men at the same time points. There was no significant differences between groups for the same variables.

*ACL = anterior cruciate ligament.

†Level of evidence according to the National Health and Medical Research Council of Australia (26).

TABLE 2. Summary of studies investigating the order and magnitude of muscle recruitment for high-risk landing tasks observed for noncontact ACL injuries.*

References	Measurement tool(s)	Summary of findings
Huston and Wojtys (48)	Custom-made jig that fixed the thigh and foot. Participants instructed to oppose a 30-N force applied to the tibia. Surface EMG measured quadriceps, hamstring and gastrocnemius activity.	The typical order of muscle recruitment when opposing the 30-N force was hamstrings, quadriceps then gastrocnemius. In higher ACL injury risk populations, initial quadriceps activity relative to initial hamstring activity was greater.
Lloyd and Buchanan (57)	Surface EMG applied to hamstrings, quadriceps and gastrocnemius. Intramuscular EMG measured adductor and abductor activity. Participants were seated and supported only by ischial tuberosities. Load cell fixed to ankle measured isometric forces in transverse plane. Data analyzed in biomechanical model.	For forces applied in the sagittal plane, flexor or extensor muscle activity was sufficient to overcome the opposing force. When forces were adjusted to require a varus or valgus component, skeletal muscle also contributed to oppose the applied force. However, skeletal muscle activity was typically insufficient to overcome varus/valgus forces. Other soft tissues were required to assist in dynamic knee joint stability.
Colby et al. (25)	Surface EMG measured quadriceps and hamstring activity for a sidestep, crossover cut maneuver, deceleration to a stop, and jump landing.	Quadriceps activity typically increased at foot strike, peaking at the mid eccentric phase (i.e., just before commencement of push off). Hamstring activity peaked just before foot strike and remained constant, or declined, throughout stance.
Besier et al. (8)	Surface EMG measured hamstrings, quadriceps, gastrocnemius, adductor, and abductor activity. A force plate and 6-camera motion analysis system used to measure joint loads participants completed a 30° and 60° sidestep 30 crossover cut and straight run.	Greater activation of all muscles for cutting maneuvers compared with the running task. The greater magnitude of activity coincided with increases in varus/valgus loads. For all tasks, muscle activation commenced before footstrike. The proportion of hamstring to quadriceps activity was greater for hamstrings before foot strike, and greater for quadriceps at weight acceptance. For the sidestepping tasks, the proportion of muscle activity in the medial muscle groups was greater than for the lateral muscle groups. No difference in medial-lateral muscle activity was observed between the running task and crossover cut task.
Zebis et al. (109)	Surface EMG measured hamstrings and quadriceps activity. A force plate was used to measure external joint loads. A flexible electrogoniometer was used to measure joint angles. The participants completed a sidestep cut. Prospective ACL injury data were recorded and muscle activation strategies for the ACL-injured group were analyzed.	The participants who sustained a noncontact ACL injury demonstrated lower hamstring activity before footstrike with concurrent greater quadriceps activity in preseason screening.

*EMG = electromyography.

stance for a double leg stop jump (62). Finally, an increase in internal rotation was typically reported postfatigue (17,62).

Mixed findings for knee joint moments have also been seen. Two studies showed no significant difference in knee joint extensor moments after neuromuscular fatigue (21,62), one showed a decline (52). Two studies showed a greater valgus moment (21,62) postfatigue, but in another, a decline was observed (52). For rotation moments, an increase in internal rotation moment has been reported after a neuromuscular fatiguing protocol (62).

In studies that measured the effect of fatigue on knee joint laxity, it was typically reported that that fatigue increased anterior tibial translation after neural and muscular fatigue (21,69,105). One study, however, showed a reduction in anterior tibial translation (52), though this study was underpowered.

Injury History. Four studies investigated ACL injury history as a risk factor for ACL injury. Three showed a greater risk of ACL injury in athletes with a history of ACL injury (27,33,81), and the other showed the opposite but was underpowered (38). One descriptive epidemiology study argued if ACL injury recurrence was within 12 months, the second was likely to occur in the same leg; if the injury occurred after 12 months, the distribution of ACL injury to the ipsilateral and contralateral leg was even (81). The reasons why recurrences occur was not clear, and several articles argued that it could be a function of poor surgical technique, improper rehabilitation, or a combination (33,81).

Age. The effect of age on ACL injury risk in populations with a mean sample age >18 years was discussed in 4 studies (13,27,81,93). Epidemiological studies in Norwegian and Swedish soccer leagues showed that ACL injury rates peaked in female athletes in their late teens and early twenties (13,102). For male athletes, with the exception of one (93), most studies showed the peak occurred in their mid to late 20s (13,27,81,102).

Skill. Skill was typically referred to interchangeably with competition level. Two studies suggested that ACL injury occurrence was greater at lower competition levels (76,93), 2 suggested the opposite (13,74). Given that athlete exposures were not typically reported, it is difficult to ascertain incidence rates and therefore come to any conclusions regarding competition level as a risk factor.

Skill in the context of 'landing ability' was loosely discussed in several studies showing that the kinetic and kinematic extremes which lead to noncontact ACL injuries are more easily reached in unanticipated conditions; with the assumption that athletes more skilled at safe landings, or skilled at being less exposed to unanticipated landing or change of direction tasks, would not as easily reach those extremes (8,9,17,18,65).

Gender. Overwhelmingly, gender has been the most researched risk factor for noncontact ACL injury. Thirty

articles discussed gender differences for ACL injury incidence, knee joint laxity, knee joint kinematics, or knee joint kinetics (2,4,5,12,13,15,18,21,23,28,30,40,41,48,52,59,62–66,70,74–76,79,95,98,102,106). Only one article showed a greater number of ACL injuries in a male population (13), in all others, incidence was equal or greater for women (2,4,5,13,15,30,40,41,70,74–76,79,102). Knee joint laxity was typically greater for women (28,48,95). Women demonstrated smaller flexion angles (21,52,59,65,98,106), more internal rotation (59,66), and more knee valgus (18,52,62,63,65,66) for jumping and cutting maneuvers, and GRFs (52,98,106), knee extension moments (21), internal rotation moments (62) and knee valgus moments (21,62,64) were greater for women.

Ethnicity. One study examined the effect of ethnicity on ACL injury. It showed a tendency for Hispanics and white-Europeans to be at a greater risk (101). However, the sample size was too small for clinical or statistical significance to be reached.

Extrinsic Mechanisms and Risk Factors

Playing Position. Six cohort studies reported on the association between playing position and noncontact ACL injury (13,33,74,75,81,93). A further 2 studies that investigated other mechanisms or risk factors of ACL injury also reported on playing position (73,79), and 4 laboratory studies discussed topics relevant to this area (8,9,18,65).

One laboratory study that showed an increase in GRFs, valgus collapse, and internal rotation and a decrease in knee flexion when a defensive opponent was added to a sidestepping task (65). The authors argued that this may be because when a defensive opponent was present the participants felt the need to change direction more rapidly (65).

Two soccer studies broke down playing position into striker, defender, midfielder, and goalkeeper (33,93). Data from both studies revealed that that strikers and defenders were more likely to get injured than midfielders and goalkeepers (33,93). One reported that defenders were more likely to injure their ACL when 'tackling' an opponent, whereas strikers were more likely to injure this when being tackled (93).

Clothing and Protective Wear. No article in the present review specifically examined the effect of clothing or protective wear on ACL injury rates. One controlled laboratory study examined the effect of a neoprene sleeve worn on the knee on proprioception (12). Results showed that for the active closed chain task there was a nonsignificant attenuation of knee joint kinesthesia. For the active open chain task, the attenuation became significant.

Shoe-Surface Interface. Where an increase in ground friction exists, the likelihood of ACL injury becomes greater (79,82). One laboratory study reported that shoes with a greater number and longer cleats distributed peripherally on the

sole of the shoe generate greater friction (55,56). The effects of these shoe designs were also exacerbated by increased downward pressure (56). Another study reported that grass thatches with longer and thicker blades that intertwine more so enable greater traction, which leads to increased friction (56,82).

Playing surface. Three descriptive epidemiological studies revealed a nonsignificant association between harder grounds and ACL injury (27,82,93). However, limitations within those 3 studies related to statistical power and homogenous samples makes it difficult to come to any real conclusions on this topic.

Environmental Conditions. Fewer ACL injuries were reported to have occurred in wetter conditions (15,27,81,82). However, other than 'dryness,' no study retrieved in the present review came to any conclusion with statistical significance related to environmental conditions and ACL injury. One descriptive epidemiological study in Australian Rules football showed a trend toward a greater rate of ACL injury in the northern states of Australia suggesting that climate might be an influencing factor (82). However, it was also shown that in northern states, the predominant grass on the field had a thicker thatch and the grounds typically had high evaporation rates for a greater proportion of the playing season (82).

DISCUSSION

In this article, we set out to review the literature describing mechanisms and risk factors for noncontact ACL injury for athletes who engage in field or court-based sports in recognition of the fact that the incidence of ACL injury has not changed appreciably over the last few decades despite considerable research on the topic. We proposed that novel strategies for preventing noncontact ACL injury be developed and, however, noted that doing so first requires effectively evaluating ACL injury mechanisms and risk factors (81). We chose to review the literature in this select population partly because of the implications that findings may have for the physical preparation of amateur and professional athletes. Although some mechanisms and risk factors appeared well defined, some notable limitations to current literature exist.

Anatomical and Biomechanical Mechanisms and Risk Factors

A number of anatomical risk factors were identified; NWI, knee joint laxity, hamstring flexibility and knee recurvatum, tibialis anterior cross-sectional area and volume, and the presence of relaxin receptors in human ACL. However, for most of those risk factors, no definitive conclusions should be reached. For instance, in 1 of the 2 studies that measured NWI and ACL injury, a smaller NWI was seen in ACL-injured knees (58), and the sample size in that study was larger than in the other (35); however, measurement technique was not

as accurate (x-ray as opposed to MRI); in the MRI study, no association between NWI and ACL injury was shown (35). In the studies that measured hamstring flexibility and knee recurvatum, and tibialis anterior cross-sectional area and volume in ACL-injured knees, it was not made clear whether those observed anatomical differences were present before the injury (11,15). Finally, where knee joint laxity was implicated as a risk factor for ACL injury, only passive laxity was measured (6,7,19,84,85). Nevertheless, given that passive knee joint laxity as a risk factor for noncontact ACL injury was not challenged within the literature reviewed, the presence of relaxin receptors in the ACL but not in other connective tissue structures surrounding the knee joint has been reported (36), and it has been shown that the ACL provides most resistance against anterior tibial translation, whereas surrounding structures act only as minor secondary restraints (6,7,19); evidence, although 'loose,' leans toward the importance of stability at the knee joint to prevent noncontact ACL injury. However, the question of whether passive knee joint laxity is related to dynamic knee joint stability still remains.

It should be noted that several other recently published articles not disclosed in the present review have discussed another anatomical risk factor; tibial plateau slope (16,42,43). One of those studies was not included because it was an opinion article (43), and another was omitted because it was a review article that did not meet the eligibility criteria (16). One article described a case-control study that, for reasons unknown, was not disclosed (42). It reported that increased posteriorly directed tibial plateau slope combined with shallow medial tibial plateau depth increased ACL injury risk (42). Several have argued that axial forces derived from GRFs cause posterior displacement of the femur over the tibia. Therefore, if GRF and all other factors remain constant, the greater the slope, the greater the risk of posterior displacement (16), and therefore, importance of dynamic knee joint stability is also greater.

Ground reaction forces were not the only forces that act across the knee joint which were noted in the present review, internal forces (moments) were also considered. Unfortunately, it was not made clear whether injury is more related to moments or to GRFs. However, it is worth pointing out that no article reviewed challenged the notion that injury risk appears to decline when knee flexor moments are greater (21,52,106). This may be a function of the fact that that less knee flexion (65,87,98,106) and more knee joint rotation and knee valgus has typically been observed when GRFs were greater (65).

Despite some differences in study methodology (i.e., patient recall, video analyses and controlled laboratory studies), from the literature reviewed, it appears that the kinematics which lead to ACL injury are a low knee flexion angle combined with internal or external rotation and valgus collapse. Some obvious measurement issues exist with patient recall studies related to memory and cognition, and the main

limitation to video analyses is that it is a descriptive methodology technique and in actual fact the timing of injury occurrence is impossible to identify. However, the arguments made by those studies are strengthened by 9 laboratory studies retrieved in this review, which described knee joint kinematics likely to lead to ACL injury (6,31,35,50,54,57,60,87,91). Although in each of the laboratory studies indirect measures such as ACL strain or elongation, or anterior tibial translation were used to predict the effects of kinematics.

How knee joint kinetics and kinematics present themselves in gross human movements is relatively unknown; as noted, no studies discussing the effect of different jump landings (e.g., 1 foot or 2 foot landings) on noncontact ACL injury were retrieved. Similarly, limited information was available about the change of direction maneuvers. Therefore, only a limited number of gross movements that stress the ACL were examined. Sidestep cutting was shown to be the change of direction maneuver most associated with ACL injury. This observation is consistent with laboratory studies that have revealed that valgus and internal rotation angle and moments at the knee are typically greater for sidestep cutting compared with crossover cut (8–10,59). However, in laboratory studies, no real difference in knee joint flexion angles or moments between the sidestep and crossover cut have typically been reported to exist (8–10,25,59). It is also worth highlighting again that the kinematic extremes and knee joint moments associated with ACL injury are more commonly reached when changes of direction are unplanned (8,9,17,18,65), highlighting skill as a risk factor.

Intrinsic Mechanisms and Risk Factors

A number of intrinsic risk factors were identified in the present review including anthropometry, hormones, genetics, strength and neuromuscular control, fatigue and injury history. The first intrinsic risk factor reported on was anthropometry; height, body mass and BMI were all shown to be associated with noncontact ACL injury. This is not surprising because taller people typically weigh more, and BMI is a function of height and body mass (33,81). Tibialis anterior cross-sectional area was also shown to be associated with ACL injury (11). No explanation was given in any study as to why any anthropometric variables are associated with noncontact ACL injury. Whether differences in tibialis anterior are present before injury is unknown, and it is certainly easy to assume that increased body mass increases GRF. Nevertheless, it is not surprising that skeletal muscle cross-sectional area has been linked to injury because muscle size has been shown to be related to strength, and strength may influence moments (51).

Several studies disclosed in the present review argued that knee extensor moments may contribute to noncontact ACL injury risk; and, according to the literature, when 'large' knee flexor moments are present then the risk declines (21,52,106). Thus, it is easy to assume that knee joint strength balance

would appear important. However, it should be pointed out that how knee joint strength ratios are measured is a point of contention with research showing that many traditional methods for doing so are questionable (29). Furthermore, in no study disclosed in the present review was knee joint strength ratio actually measured; strength gains were assumed after implementation of a strength training program and it was revealed that strength alone may not be sufficient, rather strength combined with appropriate neuromuscular control/landing skill to be important (45). This notion is consistent with other literature not disclosed in the present review (49).

Neuromuscular control was researched in a relatively high proportion of articles. In summary, they showed proprioceptive acuity to be important for 'switching on' the hamstrings before foot strike for safer landings. This 'theory' is supported by the fact that for unanticipated sidestepping tasks, 'safe' muscle recruitment strategies become compromised (8). That is, where skill is stressed, then proprioceptive acuity may become compromised and ability to switch on the hamstrings before foot contact declines. These results continue to point toward the importance of dynamic knee joint stability for preventing noncontact ACL injury.

Fatigue may also affect strength, skill, and neuromuscular control. However, from the studies disclosed, it appears that the association between ACL injury and fatigue is not very well understood. This may be related to no study specifically identifying the mechanism by which fatigue affects ACL injury risk, or placed considerable emphasis on the type of fatigue. Thus, manifestations of fatigue, rather than fatigue itself, may increase risk of ACL injury. For example, several studies showed fatigue impaired neuromuscular control (69,105).

A novel concept to this review was a genetic link to noncontact ACL injury. The literature showed that that variants of the COL5A1 and COL1A1 genes have been associated with increased ACL injury risk (53,88,89), most likely because those variants affect collagen production and therefore the tensile properties of the ACL. However, the notion of the genetic link to ACL is not entirely surprising because simply carrying the 'x' chromosome would suggest you are at a greater risk of noncontact ACL injury simply because you are female.

The most researched intrinsic risk factor was gender. There are many reasons why women appear to be at a greater risk of noncontact ACL injury, including manifestations of anthropometric and anatomical risk factors presenting themselves differently across genders. Some authors of the articles disclosed for the present review hypothesized that increased ACL injury rates may be related to increased sports participation among women without concurrent improvements in training practices that ultimately affects physical characteristics such as strength and neuromuscular control, skill, and some anthropometric variables (2,4,13,40,48,70,95). A similar observation could be made for ethnic minorities. Ultimately,

however, probably the most likely reason why women are at greater risk of noncontact ACL injury is because of different hormone profiles.

Hormonal risk factors have also been researched in a relatively high proportion of studies. However, most studies simply examined relationships between ACL injury incidence or ACL injury risk factors and phases of the menstrual cycle; when fluctuations in concentrations of the female sex hormones, estrogen and progesterone, are known. No article retrieved actually found a cause and effect relationship (1,3,23,28,44,46,73,75,83–85,104). Nevertheless, research argued that risk increases when the effects of estrogen were greater (107,108). It was revealed that estrogen may inhibit procollagen I synthesis in a dose-dependent manner (1,3,23,28,44,46,73,75,83–85,104,107,108). Procollagen is the ‘precursor’ to collagen. Furthermore, the administration of progesterone attenuated estradiol’s effect regardless of progesterone dosage (108). Therefore, it is not surprising that ACL injury risk appears to be greater during the luteal phase of the menstrual cycle, although estrogen concentration does not peak, progesterone concentration does (Figure 4) (44). However, it should also be noted that relaxin concentrations were also reported to be greater during the luteal phase (36). Furthermore, it was reported that estrogen might modulate relaxin receptors (36). Thus, the increased likelihood of noncontact ACL injury during the luteal phase is most likely to be because of the combined effects of estrogen on procollagen I synthesis and relaxin concentration; during this phase, the tensile properties of the ligament are likely to alter.

Finally, a history of ACL injury was shown to be a significant risk factor for noncontact ACL injury. This may be for a number of reasons; genetic link, poor rehabilitation, etc. However, no study retrieved investigated ACL injury risk after experiencing some other lower limb trauma. Given hamstring strength and function was argued to be useful in preventing ACL injuries, then it is possible that a history of hamstring injury may increase noncontact ACL injury risk. Damage to some of the secondary structures that provide stability to the knee joint may also increase risk, despite only contributing a small amount of stability to the knee.

Extrinsic Mechanisms and Risk Factors

Relative to ‘anatomical and biomechanical mechanisms and risk factors,’ and ‘intrinsic mechanisms and risk factors,’ the amount of research surrounding extrinsic mechanisms and risk factors for noncontact ACL injury was low. Only 5 risk factors were identified; playing position, clothing and protective wear, shoe-surface interface, playing surface, and environmental conditions.

It was seen that noncontact ACL injuries were more likely to occur during attacking phases of play relative to playing position (13,73–75,78,79). This explains why one study in soccer reported that defenders were more likely to injure their ACL when ‘tackling’ an opponent, whereas strikers were more likely to injure when being tackled (93).

Little was reported regarding clothing and protective wear. However, it does appear that some clothing can help with proprioception (12). More was reported on for studies that examined footwear. It was seen that with shoe designs that facilitated greater traction, and subsequently friction, ACL injury risk was greater (55,56). Increased friction requires greater opposing moments, particularly rotation moments. However, it was also reported that on grass surfaces with thatches with longer and thicker blades that intertwine more so enable greater traction, which leads to increased friction (56,82). Results from these studies suggest that friction, as a risk factor for noncontact ACL injury, may be a function of both shoe design and playing surface.

Studies that specifically examined playing surface and noncontact ACL injury were limited by statistical power and homogeneous samples, making it difficult to come to any real conclusions on this topic (27,82,93). However, given that climate can influence playing surface, it may be that ACL injury risk is more related to climate. One descriptive epidemiological study in Australian Rules football showed a trend toward a greater rate of ACL injury in the northern states of Australia suggesting that climate might be an influencing factor (82). However, it was also shown that in northern states the predominant grass on the field had a thicker thatch and the grounds typically had high evaporation rates for a greater proportion of the playing season (82). Therefore, the true effect of playing surface, climate, and shoe design remains unknown.

PRACTICAL APPLICATIONS

Early in the article, it was proposed that knee joint stability was important to protect against noncontact ACL injury. Specifically, it was revealed that anterior tibial translation was greater when the knee joint is ‘moved’ through the kinematic and kinetic extremes believed to stress the ACL, highlighting the importance of knee joint stability for dynamic tasks for preventing noncontact ACL injury (e.g., sidestep cutting tasks). However, it was also pointed out that studies that discussed knee joint stability as a risk factor typically only measured this ‘stability phenomenon’ in a passive state. To the knowledge of the authors of this study, no research has actually shown that passive stability correlates with dynamic stability. Many risk factors and mechanisms of noncontact ACL injury discussed thereafter were typically discussed in the context of their relationship to noncontact ACL injury incidence, and in the context of their influence on knee joint laxity or the kinetic and kinematic extremes which result in noncontact ACL injury. Thus, some research surrounding the mechanisms and risk factors of ACL injury becomes questionable simply because it is based on the assumption that passive knee joint stability is a sufficient measure of dynamic knee joint stability; some is questionable because it failed to find cause and affect relationships, rather it simply noted a higher incidence in experimental cohorts. This may help to explain why mixed findings for some commonly

thought of risk factors and mechanisms were often seen. Thus, this study highlights the value of continued research examining mechanisms and risk factors for noncontact ACL injury. In particular, research surrounding dynamic knee joint stability would be beneficial. Furthermore, given the apparent importance of knee joint stability, it would suggest the value of training for dynamic knee joint stability. Current literature suggests that this can be achieved through development of knee joint strength and neuromuscular training strategies that would ensure appropriate recruitment patterns. In particular, the preactivation of posterior chain muscles before footstrike appears important. However, because the incidence of noncontact ACL injury appears unchanged over the last 3 decades development of alternative training methods would also be beneficial. Finally, training for dynamic knee joint stability would be particularly important for athletes exposed to risk factors that are typically unavoidable such as genetic predisposition, high tibial plateau slope angle or high estrogen concentrations.

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Knee anatomy images in Figure 1 are Wellcome Images but has been altered to a derivative work by the authors by the addition of labels and for use in this journal. Figure 4 is from the Journal of Athletic Training and reproduced with permission. The authors would like to acknowledge the assistance of Margaret Morrison in preparation of the manuscript. No financial assistance was awarded for this article.

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1.3. STIFFNESS: A NEW WAY OF THINKING ABOUT DYNAMIC STABILITY?

Given the consequences of injury to the ACL, and because the rate at which it occurs remains an ongoing issue, novel methods for preventing ACL injury demand investigation. Based on some of the comments made in section 1.1., and the introduction of the literature review presented in section 1.2., research which aims to reduce ACL injury risk in field and court sports could have considerable impact on ACL injury rate reduction.

The literature review in section 1.2 gave an overview of the known mechanisms and risk factors for non-contact ACL injury in field and court sports and factors which may also contribute to the injury. A summary of the outcomes from this study has been adapted to table 1. In the ‘practical applications’ section of the literature review the importance of dynamic stability of the knee for protection of the ACL from injury was mentioned. Unfortunately, however, most research to date has measured knee joint stability in a passive state (See section 1.2. page 17, Arms et al., 1984, Bendjaballah et al., 1997, Butler et al., 1980, Park et al., 2009a, Park et al., 2009b). Therefore, it makes sense that the direction of research for ACL injury prevention should also focus on measurement of dynamic knee joint stability.

In defining dynamic knee joint stability, it may be useful to consider first the opposite to stability, which is laxity. In the literature review presented in section 1.2 passive laxity was defined as the amount of motion observed in a plane or axis before plateauing of a displacement tension curve and is often measured in a passive, or rested, state using devices such as knee arthrometers (Blankevoort et al., 1988). Active laxity, on the other hand, was defined as the motion observed in any plane or axis which is not associated with the primary movement (Blankevoort et al., 1988). For example, on a basic step up task some anterior-posterior or medial-lateral motion may be

observed. However, the primary movement is knee extension. Therefore, the translation or rotation observed on that basic step up task would be the active laxity. Greater active laxity would suggest lesser dynamic knee joint stability; or more specifically, reduced dynamic knee joint stability given that laxity is being considered on an active task. Therefore, for the remainder of this thesis dynamic knee joint stability will refer to the constraint of secondary motion(s) not associated with the primary movement.

Table 1. Summary of mechanisms and risk factors for non-contact ACL injuries in field and court sports

	Known Mechanisms & Risk Factors	Associated Mechanisms & Risk Factors
Intrinsic	<ul style="list-style-type: none"> - Increased body weight - Higher knee extensor moments relative to knee flexor moments - Rotation of the lower leg about the long axis of the femur - Gender (females at greater risk) - Luteal phase of menstrual cycle (i.e. when progesterone concentration is greater) - History of ACL injury 	<ul style="list-style-type: none"> - Intercondylar notch width of the femur - Hamstring Flexibility - Tibialis Anterior cross-sectional area and volume - Knee recurvatum - Tibial plateau slope - Presence of relaxin receptors in the ACL - Compromised integrity of structures in the Knee secondary to the ACL - Passive knee joint laxity - Strength and function imbalance of muscles surrounding the knee joint - Fatigue - Variants of COL5A1 and COL1A1 genes - Training and/or chronological age - Ethnicity - Skill/Playing ability
Extrinsic	<ul style="list-style-type: none"> - Footwear with a greater number and length of studs located peripherally on forefoot - Playing surface (longer grass length and thicker thatch increase risk) 	<ul style="list-style-type: none"> - Playing position (attackers typically at greater risk) - Hardness of playing surface

NOTE: Adapted from literature review presented in section 1.2. Known risk factors are those proven to increase ACL injury risk, associated risk factors are those thought to increase injury risk but not proven - they may be known to affect another risk factor (e.g. relaxin receptors may increase knee joint laxity). Intrinsic risk factors were those factors which are player/individual related and in this table includes anatomical and biomechanical risk factors; Extrinsic risk factors are those factors which are game/competition related (Orchard, 2001)

The reason why most research concerning ACL injury has typically only measured knee joint stability passively could be because of difficulty in measuring dynamic knee joint stability *in-vivo* while performing dynamic tasks. Dynamic knee joint stability has been implied from *in-vitro* cadaveric studies (MacWilliams et al., 1999), however these studies still fail to evaluate the effect of influence of active skeletal muscle. Some *in-vivo* work has used fluoroscopy and electromyography (EMG) to try explain anterior tibial translation and the importance of hamstring-quadriceps co-activation in an ACL deficient population (Isaac et al., 2005). However, the findings from that study are not conclusive because the EMG and fluoroscopy were not conducted concurrently and anterior tibial translation was ‘assumed’ by measuring patella tendon angle (Isaac et al., 2005). Novel image registration techniques offer the possibility of *in-vivo* measurement of dynamic knee joint stability while executing dynamic tasks with high precision via a method whereby computed tomography (CT) images are registered with fluoroscopy (video x-ray) to allow 4-D motion analysis of bone (Akter et al., 2012, Muhit et al., 2013, Scarvell et al., 2010). However, because this technology is relatively new it is yet to be used extensively in research.

A possible area of ACL injury risk mitigation which considers dynamic knee joint stability that has not been well researched is musculotendinous stiffness. The lack of evidence surrounding musculotendinous stiffness and its potential role in reducing ACL injury risk could be due to the difficulty of measuring dynamic knee joint stability. If CT-fluoroscopy image registration technology enables measurement of dynamic knee joint stability *in-vivo* while performing a dynamic task, and some measure of musculotendinous stiffness is concurrently examined then some insight into the role of musculotendinous stiffness for reducing ACL injury risk may be gained.

Musculotendinous stiffness, or ‘stiffness’ as it will be referred to in the remainder of this thesis, is a mechanical variable derived from Hooke’s law in physics which can be applied to human movement (Austin et al., 2002, Butler et al., 2003). Hooke’s law states that the force required to deform an object is related to a proportionality constant and the magnitude that object is

deformed (Butler et al., 2003). The proportionality constant is referred to as the spring constant (Butler et al., 2003). Thus, stiffness is a measure of resistance against force, and therefore the less 'spring' a structure or a system has the stiffer it is (Austin et al., 2002, Butler et al., 2003).

In the context of human movement, the outcome of the stiffness of one structure is likely to differ from the outcome of stiffness of an entire system. For example, when a force with magnitude great enough to elicit deformation is applied to the ACL in isolation, the ACL may fail if it has too little 'spring' and, therefore, ability to resist change in shape; similarly, to how a piece of wood would break. Conversely, too little stiffness and it may also fail because it is stretched excessively; similarly, to how a chewed piece of gum can stretch and break. However, if the knee is considered a system whereby stiffness is the sum of resistance to change in shape against force application from all structures combined (e.g. ligaments, functioning skeletal muscles, tendons), then a stiff knee may not be harmful to the ACL, rather it may contribute towards dynamic knee joint stability. This is in line with theory noted in section 1.1. that the ACL is supported by structures such as the iliotibial band, capsular ligaments and the medial and lateral collateral ligaments as well as functioning skeletal muscle (Butler et al., 1980, Opar and Serpell, 2014). Therefore, stiffness of the system may in fact protect the ACL from high magnitude forces and consequently over stretching. With this in mind, for the remainder of this thesis stiffness will also refer to stiffness of a system, not stiffness of a structure.

Evidence suggests that stiffness is a relatively easily trained quality. Training programs which focus on task knowledge of performance combined with movement across uneven or unstable surfaces and/or plyometric training can increase stiffness (Morin et al., 2009, Butler et al., 2003, Devita and Skelly, 1992, Moritz and Farley, 2004, Moritz and Farley, 2006, Spurrs et al., 2003). Importantly, it is also worth pointing out here that evidence is beginning to emerge which describes the benefits of plyometric training for protection against a number of injuries, not just non-contact ACL injury in field and court sports (Read et al., 2016).

If training stiffness has the potential to be an effective preventer of ACL injury, then more should be known about stiffness; how to measure it, how to train it, and how to evaluate its effectiveness as a mitigator of injury. However, methods for measuring stiffness can be varied. In section 1.4. of this thesis a literature review is presented which describes what stiffness is and details how it is best measured. This literature review was published in the Journal of Sports Sciences as 'A review of models of vertical, leg, and knee stiffness in adults for running, jumping or hopping tasks'.

1.4. A REVIEW OF MODELS OF VERTICAL, LEG, AND KNEE STIFFNESS IN ADULTS FOR RUNNING, JUMPING OR HOPPING TASKS

Statement confirming the authorship contribution of the PhD candidate

On behalf of all co-authors of the paper:

SERPELL, B.G., BALL, N.B., SCARVELL, J.M. & SMITH, P.N. 2012. A review of models of vertical, leg, and knee stiffness in adults for running, jumping or hopping tasks. *J Sports Sci*, 30, 3160-176.

I confirm that Benjamin Serpell has made the following contributions:

- Searching and collection of the literature
- Analysis of the literature and synthesis of concepts under supervision
- Wrote the first draft of the manuscript, and followed through to publication including proofing and final publication details of the manuscript

Professor Paul N. Smith

Signed:..... Date:.....

A review of models of vertical, leg, and knee stiffness in adults for running, jumping or hopping tasks

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Abstract

The ‘stiffness’ concept originates from Hooke’s law which states that the force required to deform an object is related to a spring constant and the distance that object is deformed. Research into stiffness in the human body is undergoing unprecedented popularity; possibly because stiffness has been associated with sporting performance and some lower limb injuries. However, some inconsistencies surrounding stiffness measurement exists bringing into question the integrity of some research related to stiffness. The aim of this study was to review literature which describes how vertical, leg and knee stiffness has been measured in adult populations while running, jumping or hopping. A search of the entire MEDLINE, PubMed and SPORTDiscus databases and an iterative reference check was performed. Sixty-seven articles were retrieved; 21 measured vertical stiffness, 51 measured leg stiffness, and 22 measured knee stiffness. Thus, some studies measured several ‘types’ of stiffness. Vertical stiffness was typically the quotient of ground reaction force and centre of mass displacement. For leg stiffness it was and change in leg length, and for the knee it was the quotient of knee joint moments and change in joint angle. Sample size issues and measurement techniques were identified as limitations to current research.

Keywords: *stiffness, neuromuscular, stretch shortening cycle*

Table I. Nomenclature.

K_{vert} = vertical stiffness	d = point of force translation distance
Δy = displacement of centre of mass	L _{oA} = actual leg length
F _{max} = maximum ground reaction force	L _{oR} = relaxed leg length
m = total body mass	TD = touch down
g = acceleration due to gravity	TO = take off
t _f = flight time	DF = duty factor
t _c = ground contact time	ε = constant
T _f = time from take-off to touch down of same leg	α = leg angle relative to x-axis
	α _o = leg angle relative to x-axis at touchdown
K_{leg} = leg stiffness	K_{knee} = knee stiffness
ΔL = change in leg length	M = joint moment
ω _o = natural frequency of a mass spring system representing a body	ΔΘ = change in joint angle
t = time	W ⁻ = negative work
u = vertical landing velocity	ω = angular velocity
L _o = Leg length	I = body mass multiplied by length of the thigh squared
s = horizontal velocity	
u = horizontal velocity of centre of mass	

Introduction

The concept of ‘stiffness’ is based on Hooke’s law which states that the force required to deform an object is related to a proportionality constant (spring) and the distance that object is deformed (Austin, Garrett, & Tiberio, 2002; Butler, Crowell, & Davis, 2003). Often the human body, or body segments, are modelled as a spring (Butler et al., 2003). Therefore stiffness in the human body, or body segments, describes its ability to resist displacement once ground reaction force or moments are applied. Thus, in this context, stiffness requires the interaction of anatomical structures such as tendons, ligaments, muscles, cartilage and bone (Butler et al., 2003; Rapoport, Mizrahi, Kimmel, Verbitsky, & Isakov, 2003) to resist change once ground reaction forces or moments are applied (Brughelli & Cronin, 2008). This notion is supported by studies which have discussed muscle pre-activation as being a contributing factor to stiffness (Horita, Komi, Nicol, &

Kyrolainen, 2002; Kuitunen, Komi, & Kyrolainen, 2002; Muller, Grimmer, & Blickhan, 2010) and work which has shown the importance of passive mechanical reactions to changes in stiffness (Moritz & Farley, 2004).

Vertical stiffness is commonly considered the 'first' or 'reference' stiffness measure from which models of leg and joint stiffness have been developed (Brughelli & Cronin, 2008; Butler et al., 2003; Morin, Dalleau, Kyrolainen, Jeannin, & Belli, 2005). Vertical stiffness is a measure of resistance of the body to vertical displacement after application of ground reaction force (Brughelli & Cronin, 2008; Butler et al., 2003). Leg stiffness, therefore, is considered resistance to change in leg length after application of internal or external forces; and joint stiffness, using the same theory, is resistance to change in angular displacement for flexion and rotation after application of joint moments (Brughelli & Cronin, 2008; Butler et al., 2003). Given stiffness in the human body is regulated by the interaction of a number of anatomical structures (Butler et al., 2003), leg stiffness must be modulated by stiffness at the joints (Arampatzis, Bruggemann, & Klapsing, 2001; Arampatzis, Bruggemann, & Metzler, 1999; Arampatzis, Schade, Walsh, & Bruggemann, 2001; Farley & Morgenroth, 1999; Hobara, Inoue, Muraoka, et al., 2010; Kuitunen, Komi, et al., 2002; Rapoport et al., 2003). There is some conjecture over which joint plays the most important role in modulating leg stiffness; several studies have shown that knee joint stiffness plays a more important role (Arampatzis, Bruggemann, et al., 2001; Arampatzis et al., 1999; Arampatzis, Schade, et al., 2001; Dutto & Braun, 2004; Hobara, Inoue, Muraoka, et al., 2010; Kuitunen, Komi, et al., 2002), some have shown that ankle stiffness is more important (Farley, Houdijk, Van Strien, & Louie, 1998; Farley & Morgenroth, 1999; Muller et al., 2010; Rapoport et al., 2003), and some were unclear or unsure (Gunther & Blickhan, 2002; Hobara et al., 2008). Notably, methodology varied considerably between those studies and the same model of joint stiffness was typically applied to all lower limb joints within those studies (Arampatzis, Bruggemann, et al., 2001; Arampatzis et al., 1999; Arampatzis, Schade, et al., 2001; Dutto & Braun, 2004; Farley et al., 1998; Farley & Morgenroth, 1999; Gunther & Blickhan, 2002; Hobara, Inoue, Muraoka, et al., 2010; Hobara et al., 2008; Kuitunen, Komi, et al., 2002; Muller et al., 2010; Rapoport et al., 2003). Nevertheless, evidence leans toward the relative contribution of ankle or knee joint stiffness to leg stiffness being task and velocity dependent (Arampatzis et al., 1999; Dutto & Braun, 2004; Hobara, Inoue, Muraoka, et al., 2010; Hobara et al., 2008; Kuitunen, Komi, et al., 2002).

Research in the area of stiffness in the human body is being published in sports science and medicine literature at an escalating rate. For example, in a literature review describing theoretical concepts surrounding the development of stiffness models published in 2008, 33 of the 62 cited references were published in the preceding eight years, and the remaining 29 were published in the 25 years preceding that (Brughelli & Cronin, 2008). It could be argued that this may be related to a relative increase in human movement research in general; however, given that in that literature review a notable increase in stiffness research output occurred after a defined point in time it is likely that it is more related to an increase interest in this area.

The reasons why this topic is of sudden interest could be many; one may be that research has associated stiffness with sporting performance (Bret, Rahmani, Dufour, Messonnier, & Lacour, 2002; Butler et al., 2003; Hobara et al., 2008; Hobara, Kimura, et al., 2010; Seyfarth, Friedrichs, Wank, & Blickhan, 1999; Spurrs, Murphy, & Watsford, 2003). For example, in a study which measured leg and joint stiffness from five consecutive double leg hops in endurance trained athletes and participants from the general population it was revealed endurance trained athletes had greater leg, knee and ankle stiffness than their counterparts from the general population (Hobara, Kimura, et al., 2010). In a similar study published two years earlier, the same authors revealed power athletes to have greater leg stiffness than endurance trained athletes (Hobara et al., 2008), suggesting that stiffness is important for force transmission especially where efficient force transmission is important for task outcome. This notion is supported by studies which have linked stiffness with 100 m sprint performance and long jump performance (Bret et al., 2002; Seyfarth et al., 1999). The interest in stiffness research may also be because it has been implicated in some common and severe lower limb musculoskeletal injuries (Butler et al., 2003).

Two recently published studies in Australian Rules football have aimed to find a link between muscle strains and stiffness (Pruyn et al., 2012; Watsford et al., 2010), and over time a number of studies have linked stiffness, or lack of, to anterior cruciate ligament injury (Arms et al., 1984; Bendjabballah, Shirazi-Adl, & Zukor, 1997; Butler, Noyes, & Grood, 1980; Park, Stefanyshyn, Ramage, Hart, & Ronsky, 2009a, 2009b). In the first of the Australian Rules football studies it was concluded that participants who sustained a hamstring injury during the season were likely to demonstrate greater stiffness than those who did not (Watsford et al., 2010), whereas the later produced conflicting results (Pruyn et al., 2012). The conflicting results may be

explained by some slight differences in methodology. In research which has implicated a lack of stiffness as a risk factor for anterior cruciate ligament injury studies have been limited to measuring stiffness passively using custom made jigs or knee arthrometers (Arms et al., 1984; Bendjaballah et al., 1997; Butler et al., 1980; Park et al., 2009a, 2009b; Serpell, Scarvell, Ball, & Smith, 2011). Given stiffness is dependent on the interaction of functioning skeletal muscle, connective tissue and bone (Butler et al., 2003) any passive or static measure of stiffness may be considered ecologically not valid as this would neglect the role of functioning skeletal muscle on stiffness. This limitation was recently discussed in detail in an anterior cruciate ligament injury mechanisms literature review which showed that no cause and effect relationship has actually been shown between passive knee stiffness and non-contact anterior cruciate ligament injury; arguing that further research in this area is important because a lot of assumptions are made in sports science and medicine research because of the perceived relationship between the injury and stiffness (Serpell et al., 2011).

Although a sudden interest in stiffness research has come about, differences in methods for measuring stiffness in the human body exist; this may ultimately bring into question the validity of some research. At the very least this notion is supported by the ambiguous results from studies which have associated stiffness to injury. This disparity is despite several review papers being published which have described models of stiffness; unfortunately they have been limited by a several factors (Brughelli & Cronin, 2008; Butler et al., 2003). Firstly, they have mainly focused on theoretical concepts and were limited to describing the development of the models rather than actually measuring stiffness. Secondly, they failed to limit to specific populations (e.g. adult populations) so that they could control for the effect of confounding factors (e.g. changing growth and hormone profiles) on the interaction of tendons, ligaments, muscles, cartilage, and bone for stiffness (Hewett, Myer, Ford, & Slauterbeck, 2006; Quatman, Ford, Myer, & Hewett, 2006), or they failed to account for other possible confounding factors such as shoe compressibility (Divert et al., 2008). Finally, and similarly to research which has implicated degree of stiffness to anterior cruciate ligament injury, they failed to limit to models which measure stiffness while engaging in tasks that stress the lower limbs through the stretch shortening cycle.

Given the sudden 'interest' in stiffness, and the potential relationship to sporting performance and injury, a review of literature which describes ways in which stiffness is modelled is timely; especially because if stiffness is related to injury sports

practitioners may become negligent by training stiffness for performance gains. Since ankle, knee, and hip joint stiffness is modelled the same and the relative contribution of stiffness at each joint is task dependent, in an arbitrary sense, reviewing how knee stiffness is modelled may be 'as good as' reviewing how ankle stiffness is modelled. Furthermore, as noted earlier, much of the research surrounding measurement of stiffness for knee joint trauma is limited in that it has typically been restricted to measuring stiffness in a passive state, and given serious knee joint injury remains one of the most severe of all injuries in field and court sports (Serpell et al., 2011), an argument for reviewing knee joint stiffness as opposed to hip or ankle stiffness can be made. Therefore, the primary purpose of this study was to review the literature and describe which models of vertical, leg and knee stiffness have been applied to measure stiffness in adult populations while engaging in running, jumping or hopping tasks and discuss some limitations to current methods. This led to a secondary aim which was to establish 'best practice' for measuring vertical, leg and knee stiffness.

Methods

A summary of the search strategy adopted and a description of how data was synthesized can be seen in Figure 1.

Search strategy

A search of the MEDLINE, PubMed and SPORTDiscus bibliographic databases for biomedicine was performed using the following search terms and Boolean operators:

(muscl* OR mechan*) AND (leg OR joint)
AND (stiff*) AND (run* OR jog* OR hop* OR
jump*)

The following restrictions were applied:

- Papers written in English only
- Studies with human samples
- No duplicates
- Journal articles only
- Articles published in January 2012 or earlier

The search disclosed 315 articles from the MEDLINE database, 163 from the PubMed database, and 184 from the SPORTDiscus database. Duplicates were removed and the abstracts were reviewed for eligibility. From the review of abstracts 63 were deemed to meet the eligibility criteria. An iterative reference check of eligible papers was performed. Papers which had been cited five or more times by

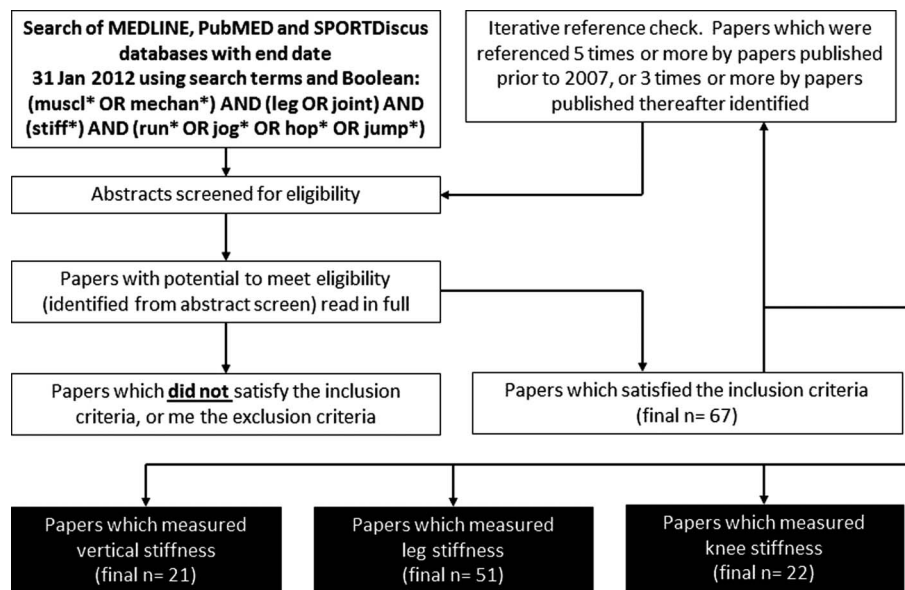


Figure 1. Summary of search strategy. The review commences with a search of the MEDLINE database. A number of papers were discussed in several stiffness 'sub-categories'.

eligible papers published before 2007, or three times or more by eligible papers published in 2007 or later, were subsequently reviewed and included in the present study provided they too met the eligibility criteria. Eligible papers identified in the initial iterative reference check were included in subsequent iterative reference checks. The iterative reference check process was repeated until exhaustion. In total 67 papers met the eligibility criteria.

Eligibility criteria

Inclusion criteria. Papers satisfied the inclusion criteria if:

- They were original articles or systematic reviews which satisfied the definition of level 1 evidence by the National Health and Medical Research Council of Australia (NHMRC) were included. The NHMRC define level 1 evidence as a systematic review of level 2 evidence (Coleman et al., 2009). Level 2 evidence is a randomized control study or prospective cohort study (Coleman, et al., 2009).
- Their sample was from an adult population (i.e. mean sample age of 18 years or older).
- They measured vertical, leg or knee joint stiffness while participants were executing tasks which actively worked lower limb muscles through the stretch shortening cycle were included.

Exclusion criteria. Papers were excluded if:

- The primary objective of the paper was to determine the effect of different types of footwear on stiffness.

- Stiffness was determined using static measures such as ultra sound, measures of joint laxity, or from squat jumps or counter movement jumps (i.e. where there was no rapid eccentric action prior to a concentric action, thus movement through the stretch shortening cycle was not achieved).
- The paper described conference proceedings.

Data extraction/grouping of articles

Eligible papers were grouped according to themes 'vertical stiffness', 'leg stiffness' and 'knee stiffness'. The number of papers retrieved which measured vertical, leg and knee stiffness was 21, 51 and 22 respectively. There were a number of studies that described more than one type of stiffness and were therefore discussed in several different sections of this paper.

Results

Vertical stiffness

Vertical stiffness was typically considered the quotient of maximum ground reaction force and centre of mass displacement (Arampatzis, et al., 1999; Austin et al., 2002; Divert et al., 2008; Dutto & Smith, 2002; Farley, Blickhan, Saito, & Taylor, 1991; Farley & Gonzalez, 1996; Ferris, Liang, & Farley, 1999; Girard, Racinais, Kelly, Millet, & Brocherie, 2011; He, Kram, & McMahon, 1991; Heise & Martin, 1998; Hobarra, Inoue, Gomi, et al., 2010; Hunter & Smith, 2007; Morin et al., 2005; Morin, Jeannin, Chevallier, & Belli, 2006; Moritz &

Farley, 2004; Slawinski, Heubert, Quievre, Billat, & Hanon, 2008; Williams & Riemann, 2009). That is:

$$K_{vert} = \frac{F_{max}}{\Delta y}$$

(Equation 1; see Table I for nomenclature)

In most studies maximum ground reaction force was measured using force platforms (Arampatzis et al., 1999; Austin et al., 2002; Divert et al., 2008; Dutto & Smith, 2002; Farley et al., 1991; Farley & Gonzalez, 1996; Ferris et al., 1999; He et al., 1991; Heise & Martin, 1998; Hunter & Smith, 2007; Morin et al., 2005; Moritz & Farley, 2004; Slawinski et al., 2008; Williams & Riemann, 2009), and centre of mass displacement was determined by double integration of vertical acceleration as described by McMahan and Cheng (Arampatzis et al., 1999; Ferris et al., 1999; Hunter & Smith, 2007; McMahan & Cheng, 1990), or by Cavagna (Austin et al., 2002; Cavagna, 1975; Divert et al., 2008; Dutto & Smith, 2002; Farley et al., 1991; Farley & Gonzalez, 1996; He et al., 1991; Morin et al., 2005; Slawinski et al., 2008). Two papers which measured ground reaction force using a force platform were unclear about the method by which calculation of centre of mass displacement was determined (Moritz & Farley, 2004; Williams & Riemann, 2009).

Several studies used force plate, pressure sensor or accelerometer technology and applied the same model to calculate vertical stiffness but modelled centre of mass displacement (Girard et al., 2011; Hobara, Inoue, Gomi, et al., 2010; Morin et al., 2005; Morin et al., 2006) from independent variables such as ground contact time, flight time etc. as follows:

$$t_f = \left(\frac{t_c + T_f}{2} \right) - t_c \quad (\text{Equation 2})$$

$$\Delta y = \frac{F_{max} t_c^2}{m[\pi^2]} + g \frac{t_c^2}{8} \quad (\text{Equation 3})$$

In the studies where accelerometer technology was used ground reaction force was also modelled (Girard et al., 2011; Hobara, Inoue, Gomi, et al., 2010):

$$F_{max} = mg \cdot \frac{\pi}{2} \cdot \left(\frac{t_f}{t_c} + 1 \right) \quad (\text{Equation 4})$$

Another model of vertical stiffness described (McMahan, Valiant, & Frederick, 1987) was:

$$K_{vert} = m\omega_0^2 \quad (\text{Equation 5})$$

With the natural frequency of a mass spring system representing a body solved by:

$$\frac{F}{mg} = \left(\frac{u\omega_0}{g} \right) \sin \omega_0 + 1 - \cos \omega_0 t \quad (\text{Equation 6})$$

A summary of the models of vertical stiffness used in previous research is presented in Table II. Papers which reported on trends only, and therefore did not present any descriptive statistics, are not shown. From data presented in Table II it appears that for running tasks stiffness is greater at higher running velocities.

Leg stiffness

Fifty-one studies retrieved discussed the measurement of leg stiffness. In 20 the model used to calculate leg stiffness was the quotient of ground reaction force and centre of mass displacement (Ambegaonkar et al., 2010; Arampatzis, Bruggemann, et al., 2001; Arampatzis, Schade, et al., 2001; Bret et al., 2002; Farley et al., 1998; Farley & Morgenroth, 1999; Granata, Padua, & Wilson, 2002; Hobara, Inoue, Muraoka, et al., 2010; Hobara et al., 2008; Hobara, Kimura, et al., 2010; Hobara et al., 2009; Hughes & Watkins, 2008; McLachlan, Murphy, Watsford, & Rees, 2006; Moritz & Farley, 2004, 2005, 2006; Padua, Carcia, Arnold, & Granata, 2005; Pruyne et al., 2012; Rabita, Couturier, & Lambertz, 2008; Rapoport et al., 2003). Some specifically noted centre of mass displacement was only measured during ground contact (Farley et al., 1998; Farley & Morgenroth, 1999; Granata et al., 2002; Hobara, Inoue, Muraoka, et al., 2010; Hobara et al., 2008; Hobara, Kimura, et al., 2010; Padua et al., 2005). The tasks required of participants in those studies varied from single or double leg hopping (Arampatzis, Bruggemann, et al., 2001; Farley et al., 1998; Farley & Morgenroth, 1999; Granata, et al., 2002; Hobara, Inoue, Muraoka, et al., 2010; Hobara, Kanosue, & Suzuki, 2007; Hobara et al., 2008; Hobara, Kimura, et al., 2010; McLachlan et al., 2006; Moritz & Farley, 2004, 2005, 2006; Padua et al., 2005), to drop jumps (Arampatzis, Schade, et al., 2001; Hobara, Inoue, Muraoka, et al., 2010) and over ground running (Bret et al., 2002); and calculation of centre of mass displacement varied between methods already described. A qualitative analysis of descriptive statistics revealed similar results to those noted in the 'vertical stiffness' sub section of the 'results' section of the present paper and will therefore not be discussed in further detail here.

Several studies used alternatives to measure leg stiffness. One study which used the quotient of

ground reaction force and centre of mass displacement as the model of stiffness for measuring leg stiffness also used another model (Granata et al., 2002):

$$K_{vert} = m \left(\frac{2}{t} \right)^2 \quad (\text{Equation 7})$$

The authors noted that both were used to permit comparison of models and facilitate validity of conclusions. It was noted that near identical results were observed from both models (Granata et al., 2002).

Several studies used a custom made sledge to measure leg stiffness (Comyns, Harrison, Hennessy, & Jensen, 2007; Flanagan & Harrison, 2007; Harrison & Gaffney, 2004; Harrison, Keane, & Cogan, 2004; Kuitunen, Kyrolainen, Avela, & Komi, 2007). The sledge was similar to a Pilates shuttle, with a force platform placed in the apparatus where foot contact would occur; thus subjects were 'fixed' superior to the hip. Leg stiffness was considered the quotient of ground reaction force and displacement of the sledge. Only one of those studies presented any descriptive statistics (Flanagan & Harrison, 2007), so measurement accuracy cannot be reported. However, it did show good day-to-day and leg-to-leg reliability.

Most other studies which measured leg stiffness considered leg stiffness as the quotient of change in leg length and maximum ground reaction force as their model of leg stiffness (Arampatzis et al., 1999; Avogadro, Chau, Bourdin, Dalleau, & Belli, 2004; Blum, Lipfert, & Seyfarth, 2009; Divert et al., 2008; Dutto & Smith, 2002; Farley & Gonzalez, 1996; Ferris et al., 1999; Girard et al., 2011; Grimmer, Ernst, Gunther, & Blickhan, 2008; Gunther & Blickhan, 2002; He et al., 1991; Heise & Martin, 1998; Hobara, Inoue, Gomi, et al., 2010; Hunter & Smith, 2007; Kerdok, Biewener, McMahan, Weyand, & Herr, 2002; Laffaye, Bardy, & Durey, 2005; Morin et al., 2005; Morin et al., 2006; Morin, Samozino, & Peyrot, 2009; Morin, Samozino, Zameziati, & Belli, 2007; Seyfarth et al., 1999; Slawinski et al., 2008; Stafilidis & Arampatzis, 2007). That is:

$$K_{leg} = \frac{F_{max}}{\Delta L} \quad (\text{Equation 8})$$

However, only three studies actually measured change in leg length (Grimmer et al., 2008; Rapoport et al., 2003; Stafilidis & Arampatzis, 2007); two measured change in leg length for over ground running using a three-dimensional motion capture system (Grimmer et al., 2008; Stafilidis &

Arampatzis, 2007), one used a 2D camera for double leg hopping (Rapoport et al., 2003). One of those studies considered change in leg length as displacement of the hip joint centre relative to a marker on the ball of the foot during ground contact (Grimmer et al., 2008), for another other it was displacement of the hip joint centre relative to the point of force application between foot contact and when ground reaction force reached maximum (Stafilidis & Arampatzis, 2007), and in the other it was considered vertical excursion of the hip joint centre relative to the ground (Rapoport et al., 2003). A reflective marker placed on the skin over the greater trochanter was used to identify hip joint centre (Grimmer et al., 2008; Rapoport et al., 2003; Stafilidis & Arampatzis, 2007). Thus, in two studies leg length was not measured perpendicular to the ground (Grimmer et al., 2008; Stafilidis & Arampatzis, 2007), in one it was (Rapoport et al., 2003). Variation in results, as suggested by standard deviation of the mean, was in the range of 4% to 28% (Grimmer et al., 2008; Rapoport et al., 2003; Stafilidis & Arampatzis, 2007).

For all other studies which modelled leg stiffness as the ratio between change in leg length and maximum ground reaction force change in leg length was predicted (Arampatzis et al., 1999; Avogadro et al., 2004; Blum et al., 2009; Divert et al., 2008; Dutto & Smith, 2002; Farley & Gonzalez, 1996; Ferris et al., 1999; Girard, et al., 2011; He et al., 1991; Heise & Martin, 1998; Hobara, Inoue, Gomi, et al., 2010; Kerdok et al., 2002; Laffaye et al., 2005; Morin et al., 2005; Morin et al., 2006; Morin et al., 2009; Morin et al., 2007; Seyfarth et al., 1999; Slawinski et al., 2008). Methods by which change in leg length was predicted and a summary of the descriptive statistics from those studies can be seen in Table III.

In one study, not presented in Table III, several variations of models of leg stiffness were compared (Blum et al., 2009). Findings from that paper are summarized in a separate table (Table IV). In that table it can be seen that for all of the models presented in that paper stiffness was considered the quotient of ground reaction force and predicted change in leg length; the method by which ground reaction force and predicted change in leg length was calculated differed. Some models were the same or similar to those identified from studies elsewhere (see Table III) some differed considerably. The authors conclude by stating that method (Equation 18) is the best and simplest approximation of stiffness and discuss the importance of a Froude correction factor when predicting change in leg length (Blum et al., 2009).

Finally, three other models used to measure leg stiffness were identified. However, the use of those

models was minimal. Nevertheless, those models are described in Table V.

Knee stiffness

Twenty-two papers retrieved measured knee stiffness. One study was not clear on how knee stiffness was measured (Kulig, Fietzer, & Popovich, 2011). The most commonly model adopted for measuring knee stiffness was:

$$K_{knee} = \frac{M}{\Delta\theta} \quad (\text{Equation 23})$$

That is, knee joint stiffness was considered the quotient of knee joint moments and change in knee joint angle (Ambegaonkar et al., 2010; Arampatzis, Bruggemann, et al., 2001; Arampatzis, Schade, et al., 2001; Clark 2009; Farley et al., 1998; Farley & Morgenroth, 1999; Gunther & Blickhan, 2002; Hobara, Inoue, Muraoka, et al., 2010; Hobara et al., 2008; Hobara, Kimura, et al., 2010; Hobara et al., 2009; Horita, Komi, Nicol, & Kyrolainen, 1996, 1999; Horita et al., 2002; Hughes & Watkins, 2008; Kuitunen, Avela, Kyrolainen, Nicol, & Komi, 2002; Kuitunen, Komi, et al., 2002; Milner, Hamill, & Davis, 2007; Muller et al., 2010; Rapoport et al., 2003). However, studies differed in the biomechanical model used to measure knee joint moments. A summary of those models is described in Table VI. Two studies were not clear on what biomechanical model was used (Ambegaonkar et al., 2010; Clark, 2009).

One study followed the same ‘theme’ as that described for the previous model (Equation 23) however considered knee stiffness for over ground running as the quotient of negative mechanical work in the knee joint (Arampatzis et al., 1999):

$$K_{knee} = \frac{2W^-}{\Delta\theta^2} \quad (\text{Equation 24})$$

A two-dimensional model was used to represent the human body and subsequently measure knee joint moments (Arampatzis & Bruggemann, 1998). No reason was given as to why this model of knee stiffness was used as opposed to another, and no descriptive statistics for knee joint stiffness were presented in the paper so measurement accuracy cannot be determined. However, the authors note that as increased running speed was achieved, so was leg stiffness and knee stiffness.

Finally, knee stiffness can be estimated from anthropometric measures and two-dimensional

kinematic parameters for treadmill running in endurance athletes (Dutto & Braun, 2004):

$$K_{knee} = I \frac{\Delta\omega^2}{\Delta\theta^2} \quad (\text{Equation 25})$$

Stiffness was only measured in the first half of the stance phase. The standard deviation of mean knee stiffness using this method was in the range of 8–10%. Compared to the more common method of measuring stiffness (Equation 23), variability of results using this method appears smaller (see Table VI). Thus, the external validity of this method may be greater. However, it should be pointed out that this method of estimating knee stiffness was not compared to methods which directly measured knee stiffness, therefore it cannot be assumed that this method (Equation 25) has greater external validity.

Discussion

The main aim of this study was to review literature which describes which models of vertical, leg and knee stiffness have been applied to measure stiffness in adult populations while engaging in running, jumping or hopping tasks. A secondary aim was to establish ‘best practice’ for measuring vertical, leg and knee stiffness. The outcomes of studies which measured stiffness will now be discussed segregated according to stiffness ‘type’. Recommendations on best practice will be made in the conclusion.

Vertical stiffness

The model of vertical stiffness most commonly used was the quotient of maximum ground reaction force and centre of mass displacement (Equation 1). An alternative was described (Equation 5), but unfortunately it was not adopted for use in later research and it did not present any descriptive statistics (McMahon et al., 1987) therefore a qualitative analysis of the accuracy of the model could not be determined.

For studies which used the model of the quotient of ground reaction force and centre of mass displacement (Equation 1) the main difference in measurement methodology was how centre of mass displacement was calculated; some used a method described by McMahon and Cheng (1990), others used a method described by Cavagna (1975). A qualitative analysis of data presented in Table I suggests that no argument can be made regarding which method is ‘better’. More specifically, the only statistical value that may be compared across all studies is standard deviation; the standard deviation of vertical stiffness as a proportion of the mean for studies which measured

centre of mass displacement using the McMahon and Cheng (1990) method was similar to what was recorded in studies which used the Cavagna (1975) method. Furthermore, if you were to compare absolute vertical stiffness and standard deviation from the Morin et al. (2005) studies, which used the Cavagna (1975) method to measure centre of mass displacement, with the Hunter and Smith (2007) study, which used the McMahon and Cheng (1990) method, the absolute vertical stiffness and standard deviations are reasonably similar; each of those studies required participants to execute similar tasks. It should be noted, however, that Cavagna's method was used in studies which required execution of a hopping task or running tasks whereas McMahon and Cheng's method was not used in any study which required execution of a hopping task.

Vertical stiffness measured while executing tasks which required greater force production (e.g. faster running velocities, single leg hopping as opposed to a double leg) was typically greater; however, measurement variation was also likely to be greater. This was evidenced by a larger standard deviation proportional to mean vertical stiffness seen in Table II. Thus, it is important to realize that for such tasks reliability issues may arise, or greater sample sizes might be required. Finally, studies which measured vertical stiffness using the model of the quotient of ground reaction force and centre of mass displacement (Equation 1) which also modelled ground reaction force or centre of mass displacement, or both, produced results similar to studies where ground reaction force and centre of mass were directly measured. This observation suggests that that modelling of those variables for measuring vertical stiffness may provide a suitable 'option' where direct measurement limitations exist. This can be confirmed by the Morin et al. (2005) study, seen in Table I, which revealed a small bias for results when ground reaction force and centre of mass displacement was modelled as opposed to when it was measured (Morin et al., 2005).

Leg stiffness

A number of studies stated they were measuring leg stiffness where in actual fact they were measuring vertical stiffness. That is, they stated they were measuring leg stiffness but measured the quotient of ground reaction force and centre of mass displacement (Equation 1). Or, they used other models which relied on centre of mass displacement to measure leg stiffness rather than measuring change in leg length (Arampatzis, Bruggemann, et al., 2001; Arampatzis, Schade, et al., 2001; Bret et al., 2002; Dalleau et al., 1998; Dutto & Braun,

2004; Farley et al., 1998; Farley & Morgenroth, 1999; Girard, Lattier, Micallef, & Millet, 2006; Granata et al., 2002; Hobara, Inoue, Muraoka, et al., 2010; Hobara et al., 2007; Hobara et al., 2008; Hobara, Kimura, et al., 2010; McLachlan, et al., 2006; Moritz & Farley, 2004, 2005, 2006; Padua et al., 2005; Rapoport et al., 2003).

Leg stiffness, strictly speaking, is not the same as vertical stiffness. Vertical stiffness is a measure of body stiffness through the whole gait cycle (i.e. stance and flight), whereas leg stiffness is a measure of stiffness of the lower limb; reliant on leg compression which can only be achieved during stance (Farley & Gonzalez, 1996; Heise & Martin, 1998; Slawinski et al., 2008). Some studies noted that they measured leg stiffness as the quotient of centre of mass displacement and ground reaction force during stance (Dalleau et al., 1998; Farley et al., 1998; Farley & Morgenroth, 1999; Granata et al., 2002; Hobara, Inoue, Muraoka, et al., 2010; Hobara et al., 2008; Hobara, Kimura, et al., 2010; Padua et al., 2005). However, this method is limited in that it assumes a rigid body superior to the hips and therefore does not consider flexion or extension at the hips or trunk.

Where leg stiffness was measured, the most commonly used model of leg stiffness was the quotient of ground reaction force and change in leg length (Equation 8), or variations of (Equations 16, 18). Unfortunately, only three studies actually measured change in leg length (Grimmer et al., 2008; Rapoport et al., 2003; Stafildis & Arampatzis, 2007); most predicted it. Where leg length was measured it was considered the distance between the hip joint and the distal point of the leg. Increased measurement accuracy from those studies may be assumed, however it should be pointed out that each adopted different methods for measuring leg length. Each considered the distal end of the leg at different points; one marked it as a point on the foot (Grimmer et al., 2008), another considered it the point of force application from ground reaction force (Stafildis & Arampatzis, 2007), and the other simply measured distance perpendicular to the ground (Rapoport et al., 2003). All marked the hip joint centre at the greater trochanter despite the fact that the greater trochanter is not the actual hip joint centre (Ehrig, Taylor, Duda, & Heller, 2006). Therefore, a more accurate approach would be to locate the hip joint centre using high-speed three-dimensional gait analysis technology (Ehrig et al., 2006) and measuring to the point of force application considering that the point of force application does not remain fixed throughout stance (Morin et al., 2007).

Most studies which considered leg stiffness as the quotient of change in leg length and maximum ground reaction force (Equation 8) predicted change

Table II. Summary of studies which have reported vertical stiffness values applying model $K_{vert} = \frac{F_{max}}{\Delta y}$.

Authors (year)	Sample	Task	Calculation of COM displacement	Mean $K_{vert} \pm \sigma$ (kN.M ⁻¹)
Austin et al. (2002)	10 healthy males	Single leg hopping at body mass, body mass + 10% and body mass + 20%	Double integration of vertical acceleration according to Cavagna (1975)	15.9 ± 5.7, 15.9 ± 5.5 and 15.3 ± 5.5 respectively
Farley et al. (1991)	4 healthy (2 male & 2 female)	Double leg hopping at frequency of 1.2 and 3.6 hops.second ⁻¹ on the spot, and with forward propulsion on treadmill	Double integration of vertical acceleration according to Cavagna (1975)	49.5 ± 1.5, 45.7 ± 1.5 and 45.0 ± 4.6 respectively*
Dutto & Smith (2002)	15 well trained distance runners (4 female & 11 male)	45 min treadmill running at 80% $\dot{V}O_{2MAX}$ pace. Measurements taken at beginning, and at 25, 50, 75 and 100% into test.	Double integration of vertical acceleration according to Cavagna (1975)	23.9 ± 3.2, 23.4 ± 3.0, 23.3 ± 3.0, 23.2 ± 2.3 and 23.1 ± 2.8 respectively
Farley & Gonzalez (1996)	4 healthy males	Treadmill running at preferred pace (9 km.hr ⁻¹) and at -5, -11, -18, -26, +17, +25, +30 and +36% of preferred running pace	Double integration of vertical acceleration according to Cavagna (1975)	15.1 ± 0.71 at low speed to 52.4 ± 2.3 at high speed
Morin et al. (2005)	8 healthy males for treadmill test, 10 trained distance runners for over ground test	Running on treadmill at velocities ranging from 12 to 24 km.hr ⁻¹ , and running over ground at velocities ranging from 14 to 25 km.hr ⁻¹ . Results were compared to stiffness calculated after modeling ground reaction force center of mass displacement from a double leg hop.	Double integration of vertical acceleration according to Cavagna (1975). For the model: $\Delta y = \frac{F_{max,c}^2}{m[\pi^2]} + g \frac{c}{8}$	37.7 ± 8.8, 37.7 ± 8.8 for treadmill running and model respectively (bias 0.1 ± 0.5). 51.4 ± 21.5, 50.2 ± 20.4 for over ground running and model respectively (bias 0.1 ± 0.5).
Slawinski et al. (2008)	9 distance runners (2 male, 2 female)	Over ground running for 2000 m (approx. 15 to 19 km.hr ⁻¹). Vertical stiffness measured before, during and after 2000 m effort	Double integration of vertical acceleration according to Cavagna (1975)	32.5 ± 6.4, 43.4 ± 9.6 and 32.6 ± 5.5 respectively
Ferris et al. (1999)	6 healthy females	Over ground running across a soft surface and hard surface (approx. 10 to 11 km.hr ⁻¹)	Double integration of vertical acceleration according to McMahon & Cheng (1990)	17.9 ± 0.8 and 18.4 ± 1.3 respectively
Heise & Martin (1998)	16 well trained distance runners	Over ground running following an aerobic running stimulus	Double integration of vertical acceleration according to McMahon & Cheng (1990)	34.5 ± 6.4
Hunter & Smith (2007)	16 trained distance runners (5 female, 11 male)	60 min treadmill running at 96-99% 10 km pace (approx. 10 to 17 km.hr ⁻¹). First 5 min at preferred frequency, last 10 min	Double integration of vertical acceleration according to McMahon & Cheng (1990)	36.5 ± 5.4 and 35.6 ± 5.1 respectively

(continued)

Table II. (Continued).

Authors (year)	Sample	Task	Calculation of COM displacement	Mean $K_{\text{vert}} \pm \sigma$ (kN.M ⁻¹)
Williams & Riemann (2009)	19 recreationally active participants (5 males, 14 females)	at manipulated stride frequency 30 sec double leg hopping at a self-selected pace, and 30 sec double leg hopping at 3.0 Hz	N/A	14.2 ± 4.87 to 15.2 ± 4.99, and 20.2 ± 4.5 to 20.3 ± 4.3 respectively
Morin et al. (2006)	8 healthy males	100 m over ground sprint. Mean vertical stiffness calculated for 20–40, 40–60, 60–80, 80–100 and 0–100 m	$\Delta y = \frac{F_{\text{max}} v_c^2}{m[\pi^2]} + g \frac{L_c^2}{8}$	93.9 ± 14.6, 98.3 ± 16.6, 93.8 ± 10.2, 89.6 ± 9.8 and 93.9 ± 12.4 respectively

NB * denotes maximum vertical stiffness

in leg length. Several methods for predicting change in leg length were reasonably similar (Equations 9, 10, 11, & 15, 17, 19), three differed considerably (Equations 12, 13, 14). A summary of the methods used to predict change in leg length and the studies which used those methods is presented in Table III. In those studies sample size was typically small, which may have led to an observed high variance in results indicated by a proportionally high standard deviation within studies. However, as noted already, it is also reasonable to assume reduced accuracy of measurement when change in leg length is being predicted as opposed to it being measured. The Morin et al. (2005) study presented in Table III supports this notion; it showed predicted change in leg length, although similar, is not quite the same as measured change in leg length

Despite evidence suggesting that measured change in leg length is not equal to predicted change in leg length, it should be pointed out that Table III shows that at higher constant velocities variation in leg stiffness decreased. It is also seen that stride frequency concurrently increased and contact time decreased. Change in leg length also increased at higher constant velocities. These results suggest that when power requirements are greater (e.g. when accelerating, jumping for distance or height, or when performing a single leg hop as opposed to a double leg hop, etc.) leg stiffness variation is also greater possibly due to increased contact time. Change in leg length will concurrently decrease, and therefore it can be assumed that metabolic energy expenditure is greater. These findings are consistent with others (Blum et al., 2009).

Finally, it should be noted that where change in leg length is predicted, and in the prediction equation if angle of attack or horizontal velocity is required, vertical stiffness may equal leg stiffness. For example, when jumping on the spot horizontal velocity or angle of attack is equal to zero; therefore to substitute zero in any of the formulas used to predict change in leg length will result in change in leg length is equal to displacement of centre of mass. Thus, vertical stiffness will equal leg stiffness despite the two measures being conceptually different.

In summary, results from studies which have measured leg stiffness highlight an inconsistency in terminology use, showing that the terms ‘vertical stiffness’, and ‘leg stiffness’ are often used interchangeably. Nevertheless, the best method for modelling leg stiffness appears to be the quotient of ground reaction force and change in leg length (Equation 8). In some instances vertical stiffness will equal leg stiffness (e.g. when change in leg length is predicted from vertical jumps or hops, or when using sledges); however, they are not the same – highlighting the

Table III. Summary of models of ΔL .

Model	Studies which used this model	Summary of sample(s)	σ for K_{leg}	Comments
$\Delta L = \Delta y + L_o - \sqrt{L_o^2 - \left(\frac{u_c}{2}\right)^2}$ where $L_o = 0.53 \times height$ (Equation 9)	Avogadro et al. (2007); Hunter & Smith (2007); Divert et al. (2008)	n = 12-16 males & females. Trained distance runners & recreational athletes	9-31%	Treadmill running only. σ as a percentage of mean K_{leg} typically higher for faster running velocities. Divert et al. considered L_o the distance from the ground to the greater trochanter. Over ground and treadmill running. σ as a percentage of mean K_{leg} typically lower over greater distances (ns). ΔL increased after ~40 m. Correlation coefficient between K_{leg} & stride length = -0.001 (ns). Bias for K_{leg} for modeled vs. measured ΔL greater for treadmill running compared to over ground running. Bias for over ground running ~20% of mean K_{leg} . Stiffness changes when subject more aware of task. Morin et al. (2005); Morin et al. (1999); Morin et al. (2006) considered L_o the distance from the ground to the greater trochanter.
$\Delta L = L_o - \sqrt{L_o^2 - \left(\frac{u_c}{2}\right)^2} + \Delta y$ where $L_o = 0.53 \times height$ (Equation 10)	Hobara, Inoue, Gromi et al. (2010); Morin et al. (2005); Morin et al. (2009); Morin et al. (2006); Girard et al. (2009)	n = 8-14 males. Well trained middle distance runners & recreational athletes	11-17%	
$\Delta L = L_o - \sqrt{L_o^2 - \left(\frac{u_c - d}{2}\right)^2} + \Delta y$ where L_o = distance from ground to greater trochanter (Equation 11)	Morin et al. (2007)	n = 10 male recreational athletes	12-33%	Treadmill running. Cadence manipulated at constant velocity. d considers point of force application is not fixed through stance phase. K_{leg} related to t_c ($p < 0.05$); increased stiffness with shorter t_c . K_{leg} unrelated to cadence frequency. Over ground & treadmill running. σ as a percentage of mean K_{leg} typically lower over greater distances (ns). Leg stiffness increased with higher stride frequencies. Stiffness greater on harder surfaces (i.e. surfaces where t_c is lower). Thus when stride frequency increased t_c decreased
$\Delta L = \Delta y + L_o(1 \cos - \theta)$ where $\theta = \sin^{-1}\left(\frac{u_c}{2L_o}\right)$ (Equation 12)	Arampatzis et al. (1999); Dutto & Smith (2002); Farley & Gonzalez (1996); Ferris et al. (1999); He et al. (1991); Heise & Martin (1998); Kerdok et al. (2002); L'affaye et al. (2005); Slawinski et al. (2008) Seyfarth et al. (1999)	n = 4-23 male & female recreational & well trained runners	12-13%	
$\Delta L = L_{oA} - L_{oR}$ where $L_{oA} = L_{oR} + e^{(\alpha - \alpha_o)}$ L_o = centre of gravity to ball of foot (Equation 13)	Seyfarth et al. (1999)	n = 30 long jumps from 18 males or females	25 & 23%	σ is for a numerical simulation of the long jump and an actual recorded long jump respectively. Mean K_{leg} was 14.6 and 16.2 kN.m ⁻¹ respectively.

NB ns = not significant

importance of actual measurement of change in leg length. The method by which leg length is measured requires additional consideration because current

methods are inconsistent and somewhat inaccurate. Furthermore, there appears to be an element of task dependency on variation in results which also requires

Table IV. Equations of leg stiffness and required additional calculations.

Equations	Additional Calculations	Comments
$K_{leg} = \frac{F_{max}}{\Delta L}$ (Equation 8)	$\Delta L = \Delta y + L_o - \sqrt{L_o^2 - \left(\frac{\Delta y}{2}\right)^2}$ (Equation 9)	Symmetric trajectories are assumed and resulting leg compression is calculated using COM displacement using method described by McMahon & Cheng 1990
$K_{leg} = \frac{F_{max}}{\Delta L}$ (Equation 8)	$\Delta L = L_{OAdj} - L_{Omin}$ $L_{OAdj} = L_{TD} + \frac{L_{OTD} - L_{OTO}}{t_c} \cdot t$ $L(t) = L_{Omin}$ (Equation 14)	Leg compression is defined as the difference of adjusted leg length (the rest is adjusted to match both initial and final leg length at TD and TO respectively) and COM-center of pressure
$K_{leg} = \frac{F_{max}}{\Delta L}$ (Equation 8)	$\Delta L = L_o + \frac{F_{max}}{m} \left(\frac{t_c}{\pi}\right)^2 - \frac{g}{8} t_c^2 - L_o \sin \alpha_{TD}$ (Equation 15)	Leg compression is estimated assuming a sinusoidal GRF
$K_{leg} = \frac{\Gamma \cdot F_{max}}{\Delta L}$ (Equation 16)	$\Delta L = L_o + \Gamma \frac{F_{max}}{m} \left(\frac{t_c}{\pi}\right)^2 - \frac{g}{8} t_c^2 - L_o \sin \alpha_{TD}$ $\Gamma = \frac{\pi}{2F_{max} t_c} \int_0^{t_c} F(t) dt$ (Equation 17)	Leg compression is estimated assuming a Γ -corrected sinusoidal GRF
$K_{leg} = \frac{F'_{max}}{\Delta L}$ (Equation 18)	$\Delta L = L_o + \frac{F'_{max}}{m} \left(\frac{t_c}{\pi}\right)^2 - \frac{g}{8} t_c^2 - L_o \sin \alpha_{TD}$ $F'_{max} = mg \frac{\pi}{4} \left(\frac{1}{DF}\right)$ $DF = \frac{t_c}{2(t_c + t_f)}$ (Equation 19)	Maximum vertical leg force is derived from the duty factor and represents the amplitude of the sinusoidal GRF. Leg compression is estimated as in (Equation 15).

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Table V. Alternative K_{leg} models.

Model	Studies which used this model	Summary of sample(s)	σ for K_{leg}	Comments
$K_{leg} = \frac{K_{knee}}{l^2 \sin \Delta \theta}$ (Equation 20) where $l = \text{length of thigh}$ $\Delta \theta = \text{change of knee angle}$	Dutto & Braun (2004)	9 well trained endurance athletes	9–15%	Treadmill running. Leg stiffness estimated from knee torsion stiffness. Authors chose to use this model because of constraints imposed from other laboratory equipment required in the study, but note this method has shown to produce similar results to the ‘conventional’ model (9).
$W_{CM^-} = 1/2 K_{vert} \cdot \Delta y^2$ (Equation 21) where $W_{CM^-} = \text{negative work}$ (i.e. work performed during eccentric phase)	Dalleau, Belli, Bourdin & Lacour (1998)	8 well trained middle distance runners	~30%	Treadmill running. Stiffness calculated from Δy during stance. Kinematic arm used and attached to dorsum of thorax. Displacement of kinematic arm assumed to be equal to Δy . Authors note that this method has shown to produce similar results to the ‘conventional’ model (9), but give no reason as to why they chose this approach.
$K = \frac{\pi \cdot (t_f + t_c)}{t_c^2 \cdot \left(\frac{t_f + t_c}{\pi} - \frac{t_c}{4}\right)}$ (Equation 22)	Bret et al. (2002); Hobara et al. (2007); Girard, Lattier, Micallef & Millet (2006)	19 male sprinters, 7 recreationally active males, & 12 well trained tennis players respectively	n/a	

some sensitivity too. Results from this section also allude the importance of consideration to sample size might to improve external validity

Knee stiffness

The most commonly used model of knee stiffness was the quotient of knee joint moments and change in joint angle (Equation 23). Only two others were identified; however, they followed the same theme. That is, one other effectively modelled knee stiffness as the quotient of knee joint moments and change in knee joint angle, however because it measured

negative work only (Equation 24) it only measured knee joint moments during the braking phase of stance. The other modelled knee stiffness the same way, however moments and joint angles were predicted (Equation 25).

The main difference between studies which modelled, and subsequently measured, knee stiffness was the biomechanical model used to measure knee joint moments (Table VI). Limitations in those biomechanical models bring into question the accuracy of results, and this is supported by a proportionally high standard deviation in all studies. For instance, few studies measured three-

Table VI. Summary of biomechanical models used to calculate knee joint moments for measuring knee joint stiffness.

Model / author of study describing model	Description of model	Studies which used this model, experimental tasks and methods of measurement	$K_{\text{knee}} \sigma$
Bresler & Frankel (1950)	The body consists of 4 rigid links – head arms and trunk, thigh, leg, and foot	Horita et al. (1996, 1999, 2002). Stiffness was measured from double leg drop jumps. Reflective markers were placed on anatomical landmarks of participants, drop jumps onto a force platform were recorded using a two-dimensional high speed video camera and kinematic data were digitised using specialist software	35–40%
Dempster (1955)	Rigid linked 4-segment model	Kuitunen, Avel et al. (2002), Kuitunen, Komi et al. (2002), Milner et al. (2007), Hobara et al. (2008), Hobara et al. (2009), Hobara, Inoue, Muraoka et al. (2010), and Hobara, Kimura et al. (2010). Stiffness was measured for a variety of tasks including over ground running, hopping drop jumps typically using a two-dimensional high speed camera. Reflective markers were placed on anatomical landmarks on participants' body and kinematic parameters were calculated using specialist digitizing software. Milner et al. (2007) was the only study which measured three dimensional kinematics using a six camera three-dimensional motion analysis system	47–50%
Gunther, Sholukha, Kessler, Wank & Blickan (2002)	Non rigid model which factors in skin motion and wobbling mass	Muller et al. (2010) and Gunther & Blickhan (2002). Stiffness was measured for over ground running using a two-dimensional high speed camera with digitizing software and a 12 camera three-dimensional motion analysis system respectively	3–37%
Zatsiorsky & Seluyanov (1983)	Rigid 15-segment 2 dimensional model	Farley et al. (1998), Farley, and Morgenroth (1999), Arampatzis, Bruggeman et al. (2001) and Arampatzis, Schade et al. (2001). Stiffness was measured for double leg hops and drop jumps. Reflective markers were attached to anatomical landmarks and kinematic data was recorded using two-dimensional high speed video cameras and specialist digitizing software	13–70%
Plug-in-gait (VICON, Oxford, England)	Rigid. Number of segments dependent on the number of models used (i.e. lower body vs. upper etc.)	Hughes & Watins (2008). Stiffness was measured from a jump landing using a 12 camera three-dimensional motion analysis system. Markers were placed over anatomical landmarks in accordance with the plug-in-gait marker set. Only knee joint moments in the sagittal plane were measured	26–50%
Rapoport et al (2003)	Rigid linked 4-segment two-dimensional model	Rapoport et al. (2003). Stiffness was measured for double leg hopping using a commercially available video camera and digitizing software.	7–32%

dimensional kinematics (Gunther & Blickhan, 2002; Milner et al., 2007; Muller et al., 2010), most simply measured kinematics using a two-dimensional camera from a lateral view (Arampatzis, Bruggemann, et al., 2001; Arampatzis, Schade, et al., 2001; Farley et al., 1998; Farley & Morgenroth, 1999; Hobara, Inoue, Gomi, et al., 2010; Hobara, Inoue, Muraoka, et al., 2010; Hobara et al., 2008; Hobara, Kimura, et al., 2010; Horita et al., 1996, 1999, 2002; Kuitunen, Avela, et al., 2002; Kuitunen, Komi, et al., 2002; Rapoport et al., 2003). Therefore, given the knee works in more than just the sagittal plane, then in most studies retrieved knee moments can be considered only an estimation because most of the models that were adopted neglected the effect of internal/external rotation and varus/valgus moments. Thus, it may have been assumed that the only moments which occur at the knee are flexion and extension, or varus/valgus and internal/external rotation moments may have only been predicted. The other major limitation to the biomechanical models used in the studies retrieved was that most were rigid-body models. That is they did not factor in wobbling masses (e.g. skeletal muscle, connective tissue etc.), and assumed that force production across the full length of a segment was equal.

In summary, the most commonly model for measuring knee stiffness is the quotient on knee moments and change in angle. However, methods for measuring knee joint moments should be improved. An appropriate method would be to use a three-dimensional biomechanical model which eliminates artefact from wobbling mass about the knee joint.

Conclusion

At the beginning of this review it was noted that research into stiffness in the human body has grown in interest exponentially. Several reasons for the exponential increase in interest were speculated however it was hypothesized that this is likely due to the relationship between stiffness and sporting performance, and a potential relationship between stiffness and injury; relatively considerable emphasis was placed on the stiffness and injury relationship. However, it was also pointed out that the validity of some of the currently available research on stiffness is questionable due to measurement limitations which may have arisen from confusion surrounding the definition of stiffness. Thus, it was suggested that this confusion be overcome, particularly because of the potential role stiffness may have in sporting injuries; if it shown to increase injury risk it would be negligent for sporting practitioners to 'encourage' stiffness for improved performance. Consequently, the aim of this paper was to review models and

methods for measuring vertical, leg and knee stiffness to establish a best practice for future research. As a result some recommendations can be made.

For vertical stiffness the most commonly used, and therefore recommended, model was the quotient of ground reaction force and centre of mass displacement (Equation 1). Force plates were typically used to measure ground reaction force. However, in a small number of studies ground reaction force was predicted and a small bias was observed in one which compared predicted ground reaction force and measured ground reaction force (Morin et al., 2005). Thus, where equipment limitations exist prediction of ground reaction force might suffice. Displacement of centre of mass was typically measured by double integration of vertical acceleration using methods described by McMahon and Cheng (1990) and Cavagna (1975). Neither method for measuring centre of mass displacement seemed to be 'better' than the other; however, according to the literature reviewed Cavagna's method has been used for a wider range of tasks without 'issues' and so this method can be recommended for future use. Where possible large samples should be recruited to help reduce variance, increase power and subsequently improve external validity.

For leg stiffness a similar 'theme' was observed in that leg stiffness was most commonly considered the quotient of ground reaction force and change in leg length (Equation 8). However, it was also shown that inconsistencies in terminology use exist with a number of studies using the terms 'vertical stiffness' and 'leg stiffness' interchangeably despite the measures being fundamentally different. It was also pointed out that relatively few studies actually measured change in leg length, most simply predicted it. It was suggested that one reason why the terms 'vertical stiffness' and 'leg stiffness' may be confused is that where change in leg length is predicted, and if the experimental task requires a vertical jump, then leg stiffness will often equal vertical stiffness. This highlights the necessity of actual measurement of change in leg length. For the studies which did measure change in leg length the method by which change in leg length was measured differed between each, and the accuracy of those methods were questioned; further confusing the 'concept' of leg stiffness. It was noted that leg length measurement requires accurate location of the hip joint centre and measuring the distance to the point of force application considering that the point of force application does not remain fixed throughout stance (Morin et al., 2007). Ground reaction force was typically measured using force plates and, similarly to observations made for vertical stiffness, relatively large sample sizes are recommended to

help reduce variance, and improve accuracy and external validity.

Finally, for knee joint stiffness the model typically used, and therefore recommended, was the quotient of knee joint moments and change in joint angle (Equation 23). However, similar observations to those made for leg stiffness in terms of measurement accuracy due to questionable measurement methods were noted; reported methods for measuring knee joint angles typically neglected movement out of sagittal plane. Improved measurement accuracy in the other two planes of motion would enable more accurate measurement of knee joint moments, and subsequently knee joint stiffness. Similarly to vertical and leg stiffness, relatively large sample sizes are recommended when measuring knee joint stiffness.

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1.5. AIMS OF THESIS

This chapter has shown that considerable research surrounding ACL injury has been undertaken yet incidence remains high (see section 1.2. page 6). Thus, novel research and development of better training methods to prevent ACL injuries from occurring is required. Based on what is being reported in scientific literature, work which aims to reduce incidence of non-contact ACL injury in field and court sports is likely to have most impact on reducing ACL injury rates, and dynamic knee joint stability is also important (see section 1.2. page 17).

In section 1.3. of this thesis, dynamic knee joint stability was defined as the constraint of secondary motion(s) not associated with a primary movement. Stiffness was also identified as a potential area of risk mitigation for non-contact ACL injury in field and court sports because it considers dynamic knee joint stability and appears easily trainable. Stiffness has been well researched in relation to sports performance, however it is a relatively novel concept when applied to musculoskeletal injury.

Section 1.3. and the literature review in section 1.4. described stiffness as a concept derived from physics, based on Hooke's law, and a measure of resistance to change against force. Specifically, knee stiffness was defined in section 1.4. as the ability to resist translations and change in joint angles following application of muscle moments, leg stiffness was the ability to resist change in leg length after application of ground reaction force, and vertical stiffness was resistance to vertical displacement of centre of mass after application of vertical ground reaction force. Based on the literature review presented in section 1.4. measurement of knee and leg stiffness is complex, requires use of high tech kinematic measurement systems, and is affected by the biomechanical model used to measure moments and joint angles. It was also noted that the terms 'leg stiffness' and 'vertical stiffness' are often used interchangeably, but the two measures are fundamentally different and therefore it is incorrect to do so. The literature review in section 1.4 showed that

vertical stiffness can be measured easily in the field with just a force platform, and typically/best calculated as the quotient of maximum ground reaction force and centre of mass displacement. Furthermore, the amount of research that has been published which has measured and reported on vertical stiffness using the same method is great, offering the possibility of comparing studies to each other. Finally, it may be hypothesized that a high level of effective vertical stiffness is linked, in part, to a high level of effective leg stiffness, which in turn is related to knee stiffness (see figure 2). Therefore, and in summary, stiffness, vertical stiffness in particular, may be of interest for ACL injury risk mitigation because it is easily measurable with just a force platform (see section 1.4.), may be trainable (see section 1.3.), and it may enhance dynamic knee joint stability. Consequently, for this body or work vertical stiffness was investigated.

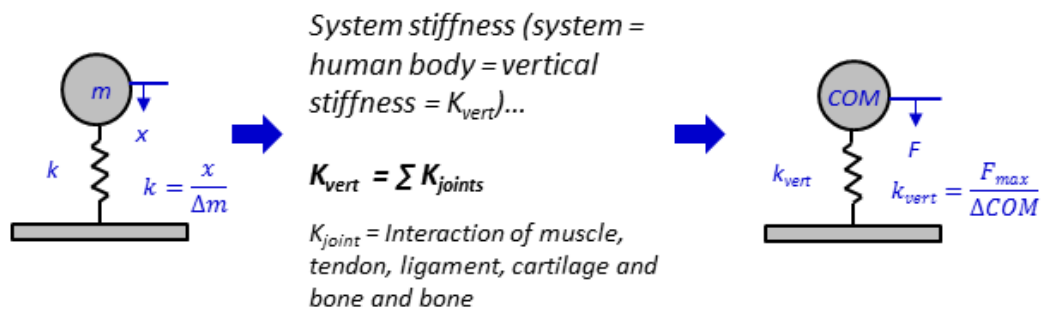


Figure 2. Vertical stiffness is a measure of whole body stiffness; it is dependent on stiffness at each of the joints which, in turn, is dependent on the interaction of muscle, tendon, ligament, cartilage and bone. (m = mass; k = stiffness; x = force; Δ = change; COM = centre of mass; vert = vertical; F = ground reaction force; Σ = sum)

Note: this image appears again later in this thesis as a part of a published paper

Although promising, some confusion about the role of vertical stiffness as an area of risk mitigation for traumatic non-contact injury, such as non-contact ACL injury, exists. Some have argued that the increased ground reaction force associated with increased vertical stiffness increases injury risk, citing osteoarthritis, stress fracture and tendinopathies as examples (Butler et al., 2003,

Bradshaw and Hume, 2012, Lorimer and Hume, 2016). However, these are overuse injuries, and they typically occur when strain below the single load-failure threshold occurs repetitively (Opar et al., 2012, Warden et al., 2006). A traumatic injury is considered an injury associated with a single incident (Junge and Dvorak, 2000, Fuller et al., 2006), such as a muscle strain or ligament injury. Given these definitions, it may be that overuse injuries are the result of improper loading as opposed to vertical stiffness. However, it is important to acknowledge that some believe increased ground reaction force does increase ACL injury risk (McLean et al., 2004, Onate et al., 2005, Podraza and White, 2010, Schmitz et al., 2007, Yu et al., 2001). If it were true that increased ground reaction force increases ACL injury risk then vertical stiffness could be a risk factor for traumatic non-contact injury. Furthermore, vertical stiffness is, in part, affected by knee joint stiffness (see figure 2) and there may be less knee flexion with a stiffer knee; previous research that has argued extended knee joint is more prone to ACL injury (see section 1.2), suggesting also that vertical stiffness may increase ACL injury risk. Adding strength to the argument that increased vertical stiffness is a risk factor for traumatic non-contact injury is some research which has specifically argued that risk of sustaining a hamstring injury increases with increased stiffness (Pruyn et al., 2012, Watsford et al., 2010). However, the argument that increased ground reaction force increases ACL injury risk is somewhat unsubstantiated, with the authors of those papers calling for further research to explain how or why increased vertical ground reaction force increases non-contact ACL injury. Furthermore, that argument neglects that stiffness also constrains joint motion and translations. In addition, increased vertical ground reaction force is likely to result in less decompression of the joint space and therefore enhanced joint congruency. This is also true of a stiffer knee. The constrained joint motion and translation and increased joint congruency will likely ensure the ACL is less taut, or 'less stressed'. Furthermore, it is also worth pointing out here that in the studies where increased vertical stiffness was argued to be a risk factor for hamstring strain, the mechanism behind the observation was not well established. Therefore, an argument about the 'protective nature' of vertical stiffness can be made, but whether this truly is the case is yet to be seen. Research investigating whether an association between vertical stiffness and

traumatic non-contact injury can be made, including for non-contact ACL injury, would therefore be beneficial.

The aim of this thesis was to explore whether vertical stiffness could be linked to some common traumatic musculoskeletal injuries in the football codes. This body of research did not intend to describe vertical stiffness as a mechanism for injury prevention. However, it was believed that if vertical stiffness was not linked to these types of injuries then the outcomes of this thesis could form the basis from which future research could be undertaken to examine the protective nature of vertical stiffness against injury. These outcomes could also be used to justify investigation into how best to train to enhance vertical stiffness.

The primary focus of thesis was on non-contact ACL injuries; partly because ACL injury rates are unchanged despite the considerable amount of research in the area demanding novel investigations (section 1.1.), and partly because ACL injuries are the most costly in sport (section 1.1., Orchard et al., 2001). This was done by examining the interaction between skeletal muscle function around the knee joint with vertical stiffness and knee joint kinematics and ACL elongation *in-vivo*, and by examining the interaction between vertical stiffness with knee joint kinematics and ACL elongation *in-vivo*. However, vertical stiffness has been shown to be related to functioning skeletal muscle (see section 1.2. page 17, and section 2.1.), and skeletal muscle injury can lead to neuromuscular inhibition of the injured muscle for up to twelve months (Opar et al., 2012). Muscle strain injury is the most prevalent non-contact injury in many field sports (Orchard et al., 2001). Therefore, whether vertical stiffness is associated to muscle strain injury also became of interest and was subsequently investigated.

This thesis is comprised of four distinct studies, each of which is presented in a separate chapter (chapters two to five). It concludes with a final chapter which draws together the outcomes of each of the studies presented in chapters two to five which explains how they combine to meet

the aims of this thesis. Figure 3 provides a concise schematic of the structure of the thesis, and a description of the studies presented in chapters two to five follows.

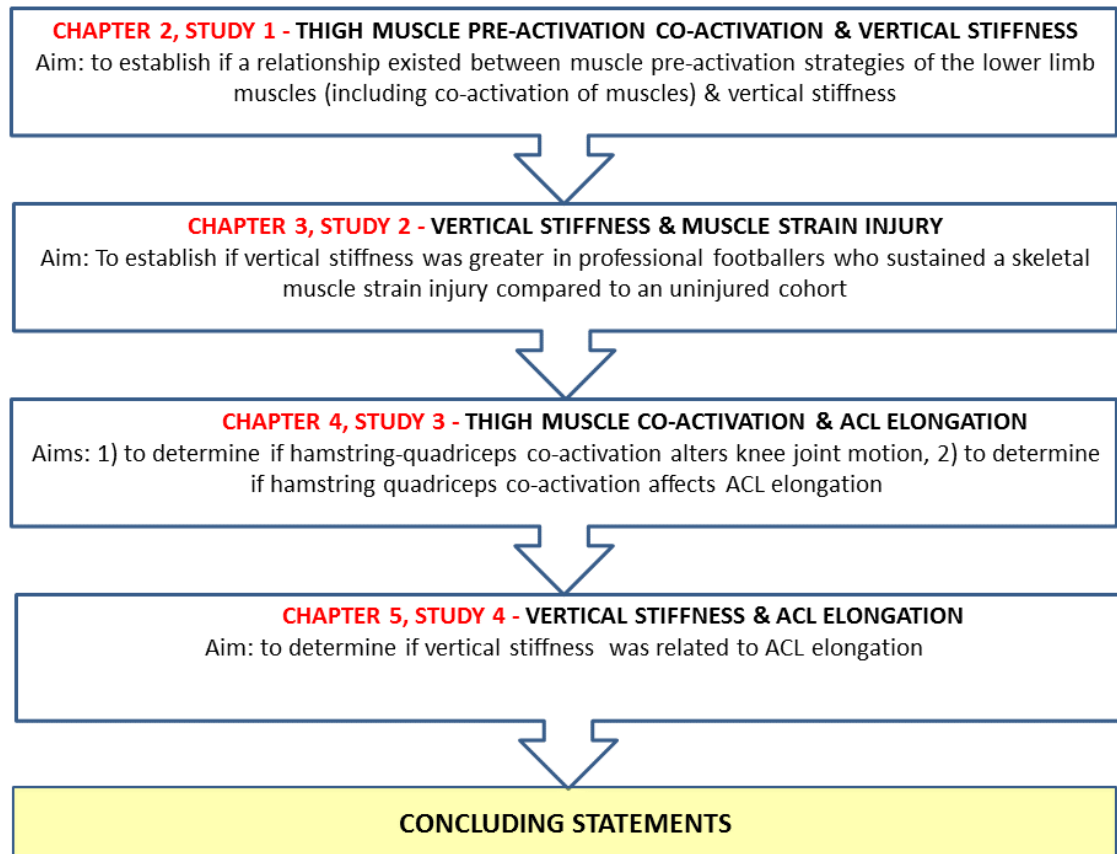


Figure 3. The structure of this thesis. Each study has been published in an internationally peer reviewed journal, and each study forms its own chapter

Chapter 2 - Thigh Muscle Pre-activation Co-activation and Vertical Stiffness

The study presented in this chapter was published in the Journal of Electromyography and Kinesiology as ‘Muscle pre-activation strategies play a role in modulating K_{vert} for change of direction manoeuvres: An observational study’.

In this study it was hypothesised that vertical stiffness for change of direction tasks would be greater when increased neuromuscular activation in the lower limb muscles was present prior to foot contact. The neuromuscular activation in the muscles prior to foot contact was termed ‘pre-

activation'. Therefore, the aim of this study was to establish if a relationship existed between pre-activation of the lower limb muscles and vertical stiffness. Twenty males aged 24.0 ± 4.4 years, height 185.3 ± 11.9 cm and weight 100.6 ± 18.5 kg were conveniently recruited to this study from a single professional rugby union club. Each participant performed three multidirectional hopping tasks onto a force platform on their preferred leg. The hops were designed to simulate the change of direction manoeuvre typically observed when non-contact ACL injury occurs. Each hop became progressively more difficult to simulate an increase in running intensity. Vertical stiffness was calculated from each hop and muscle activity was measured during the pre-activation period (i.e. the 100 ms prior to foot contact) from the gluteus maximus, vastus lateralis, vastus medialis, biceps femoris, semimembranosus, and medial gastrocnemius using EMG. Pearson's correlations were used to establish if a relationship existed between mean activation of each muscle and vertical stiffness. A pre-activation co-activation index (i.e. the ratio of agonist to antagonist muscle activation in the 100 ms prior to foot contact) was also calculated for several muscle groups and their relationship to vertical stiffness was also established using Pearson's correlations. Finally, the relationship for peak muscle activation for each muscle, and timing of peak activation relative to foot contact, with vertical stiffness was also described also using Pearson's correlations. In addition to this a series of independent samples t-tests were used to determine if there were significant differences between antagonistic muscles for peak muscle activation (proportional to maximum voluntary contraction), and timing of peak muscle activation relative to foot contact.

Chapter 3 – Vertical Stiffness and Muscle Strain Injury

The study presented in this chapter was published in the Journal of Sports Sciences as 'Vertical stiffness and muscle strain in professional Australian football'.

This study was important because if the work presented in this thesis found that non-contact ACL injury was unlikely associated with vertical stiffness then recommendations about to future research for examining in more detail the protective nature of vertical stiffness for ACL injury, or

recommendations to investigate training practices for enhancing vertical stiffness could be made, but only if vertical stiffness was not related to other non-contact traumatic injuries, such as muscle strain injury. Furthermore, because neuromuscular inhibition may remain for up to twelve months following muscle strain injury, it was thought that muscle strain injury could negatively affect vertical stiffness. This would be particularly concerning if vertical stiffness is shown to reduce ACL injury risk. Therefore, in this study it was hypothesised that vertical stiffness is not linked to muscle strain injury. Consequently, the aim of this study was to establish if vertical stiffness was greater in professional Australian Rules footballers who sustained a lower limb skeletal muscle strain compared to those who did not, and establish if a relationship for age and training history with vertical stiffness existed.

For this study thirty-one participants recruited from a single professional Australian Rules football club underwent weekly jump testing on a force platform over two seasons and results were compared between an uninjured and injured cohort. Mean age, height and weight at the commencement of this study for the uninjured cohort was 20.5 ± 2.1 years, 189.8 ± 8.3 cm and 85.4 ± 9.8 kg respectively. For the injured cohort it was 22.7 ± 3.1 years, 184.8 ± 8.2 cm and 83.1 ± 6.7 kg respectively. The jump test was always performed 48 hours following a game. For the jump test participants were required to stand on a force platform and complete two consecutive jumps without pausing between jumps. Vertical stiffness was calculated from the second jump. Data from participants who sustained a muscle strain injury was allocated to the injured cohort, whereas data from those who did not sustain an injury was added to an uninjured cohort. A series of independent t-tests was used to establish if any difference in means existed between groups for demographic variables. A 2-way ANOVA with interactions was performed to establish if there was any difference in means between or within groups for vertical stiffness one week prior to the injury occurring, three weeks prior to the injury occurring, and at the end of preseason. Effect size and upper and lower confidence limits for 95% confidence was also calculated for comparisons of

means. Finally, Pearson's correlations were used with pooled data to establish a relationship between age and vertical stiffness and training history and vertical stiffness.

Chapter 4 – Thigh Muscle Co-Activation and ACL Elongation

The study presented in this chapter was published in Biomed Central Musculoskeletal Disorders as 'Medial and lateral hamstrings and quadriceps co-activation affects knee joint kinematics and ACL elongation: A pilot study'.

It was intended that this study would build on the first study which aimed to find a relationship between lower limb muscle pre-activation strategies and vertical stiffness by exploring a relationship between co-activation of the muscles that surround the knee joint and measures which are known to load, or measures which represent loading, of the ACL. It was hypothesised that co-activation of the muscles around the knee joint would reduce excessive knee joint motion, and subsequently reduce ACL elongation. Therefore, this study had two aims; firstly, to determine if hamstring-quadriceps co-activation alters knee joint kinematics. The second aim was to establish if it affects ACL elongation. Five male participants aged 24.9 ± 4.1 years, height 184.8 ± 9.1 cm and weight 90.1 ± 16.3 kg were conveniently recruited to this study from a single professional rugby union club. A CT from each participant's dominant leg was acquired prior to performing two step-ups under fluoroscopy: one with 'natural' hamstring-quadriceps co-activation, one with deliberate co-activation. Muscle activity was measured during each step-up from the vastus lateralis, vastus medialis, biceps femoris, semimembranosus using EMG to confirm the deliberate co-activation. The CT images were registered to fluoroscopy using novel technology developed by engineers from the Australian Defence Force, and a 4-D model of knee joint motion was developed for each participant. Anterior cruciate ligament attachments were mapped to the 4-D models and its length was assumed from the distance between attachments. Knee joint kinematics were measured from the 4-D model and ACL elongation was derived from the change in distance between the attachment points as they moved relative to each other. Descriptive statistics were used to show

how knee joint motion changed when co-activation of the muscles occurred. Pearson's correlations were used on pooled data to describe relationships between knee joint kinematics and ACL elongation to describe construct validity of this measure, and also describe a relationship between co-activation index of the muscles and ACL elongation.

Chapter 5 – Vertical Stiffness and ACL Elongation

The study presented in this chapter was published in British Medical Journal Open Sport and Exercise Medicine as 'Vertical Stiffness is not related to anterior cruciate ligament elongation in professional rugby players'.

This was the final study of this thesis and it was designed to build on outcomes from all three studies presented to this point, studies one and three which are presented in chapters two and four respectively. For this study, it was predicted that vertical stiffness was not related to measures known to load, or which represent loading of, the ACL. Therefore, the main aim of the study was to describe a relationship for vertical stiffness with anterior tibial translation and ACL elongation. This was a cross-sectional observational study of 11 professional Australian Rugby union players (mean age, height and weight 26.1 ± 4.7 years, height 180.5 ± 9.1 cm and weight 85.4 ± 16.5 kg respectively). A CT scan was taken from each participant's dominant leg prior to them performing a multidirectional hopping task which simulated the change of direction manoeuvre typically observed when non-contact ACL injury occurs, under fluoroscopy. That is, a hopping task similar to that used in study one was performed under fluoroscopy. The same 4-D modelling technique used in the previous study was also used in this study and ACL attachments were also mapped to the 4-D models and its length was assumed from the distance between attachments. Vertical stiffness was calculated from force plate data and knee joint kinematics and ACL elongation was measured from image registration output. Pearson's correlations were used to describe the relationship between vertical stiffness and anterior tibial translation, and vertical stiffness and ACL elongation. A Pearson's correlation was also used to describe the relationship between anterior

tibial translation and ACL elongation to further investigate the construct validity of the measure used to describe ACL elongation in this study and the previous one.

CHAPTER 2

Thigh Muscle Pre-activation Co-activation & Vertical Stiffness

- 2.1. Muscle Pre-activation Strategies Play a Role in Modulating K_{vert} for Change of Direction Manoeuvres: An Observational Study
- 2.2. Context of study

2.1. MUSCLE PRE-ACTIVATION STRATEGIES PLAY A ROLE IN MODULATE K_{VERT} FOR CHANGE OF DIRECTION MANOEUVRES: AN OBSERVATIONAL STUDY

Statement confirming the authorship contribution of the PhD candidate

On behalf of all co-authors of the paper:

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I confirm that Benjamin Serpell has made the following contributions:

- Contributed to discussions on design of the study
- Collection, analysis and synthesis of literature
- Submission for ethics approval, recruitment of subjects, and collection of data under supervision
- Analysis and interpretation of data under supervisions
- Wrote the first draft of the manuscript, and followed through to publication including proofing and final publication details of the manuscript

Professor Paul N. Smith

Signed:..... Date:.....



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Muscle pre-activation strategies play a role in modulating K_{vert} for change of direction manoeuvres: An observational study



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ABSTRACT

The aim of the study presented in this paper was to establish if a relationship existed between lower limb muscle pre-activation strategies and vertical stiffness (K_{vert}). Participants from a professional rugby union club all performed a multidirectional hopping task on a force platform which measured K_{vert} . Muscle activity was concurrently measured for the gluteus maximus, vastus lateralis, vastus medialis, biceps femoris, semimembranosus, and medial gastrocnemius using electromyography and the activity of those muscles in the 100 ms prior to foot contact (pre-activation) was analysed. Moderate to strong positive relationships were typically seen for K_{vert} and muscle pre-activation for each muscle when normalized to maximum voluntary contraction. Pre-activation cocontraction of the muscles surrounding the knee joint also showed a typically moderate relationship with K_{vert} and peak muscle activation of antagonist muscles at the knee joint were typically similar. Results suggest that muscle pre-activation strategies play a role in modulating K_{vert} for change of direction manoeuvre.

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1. Introduction

Stiffness in the human body requires the interaction of anatomical structures such as tendons, ligaments, muscles, cartilage and bone to resist change once ground reaction forces or moments are applied (Brughelli and Cronin, 2008; Serpell et al., 2012). The ‘stiffness’ concept is derived from Hooke’s law which states that the force required to deform an object is related to a proportionality constant (spring) and the distance that object is deformed (Austin et al., 2002; Butler et al., 2003). Often the human body, or body segments, are modelled as a spring (Butler et al., 2003). For instance, vertical stiffness (K_{vert}) is considered the quotient of maximum ground reaction force (GRF) and centre of mass (COM) displacement (Serpell et al., 2012). Not surprisingly, therefore, it

has been argued that stiffness increases are partly due to increased muscle tension (Farley et al., 1998; Hobara et al., 2010; Horita et al., 2002; Spurrs et al., 2003). Some have argued that the increased muscle tension is a function of increased muscle pre-activation (particularly in the calves) (Hobara et al., 2010; Kuitunen et al., 2002, 2007; Muller et al., 2010; Spurrs et al., 2003), some have argued that it is a function of changed touch down angle at foot plant which may be modulated by knee joint muscle pre-activation (Farley et al., 1998; Horita et al., 2002; Muller et al., 2010). Regardless of the exact mechanism, results from these studies combined certainly suggest that K_{vert} is affected by muscle pre-activation at some level. Pre-activation in stiffness studies has typically been measured using electromyography (EMG) and considered muscle activity in the 100 ms prior to ground contact (Farley et al., 1998; Hobara et al., 2010; Horita et al., 2002; Kuitunen et al., 2002, 2007; Muller et al., 2010). However, it is important to note that while previous literature has postulated a link between muscle pre-activation and stiffness, some has only posed the connection theoretically, sometimes without even direct measurement of muscle activity (Farley et al., 1998; Spurrs et al., 2003). Other work has been largely inconsistent in measurement methods, and task selection has varied considerably which questions ecological validity of the studies (Hobara et al.,

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2010; Horita et al., 2002; Kuitunen et al., 2002, 2007; Muller et al., 2010).

Generally speaking, muscle activation prior to foot contact (that is, during the pre-activation period) is centrally programmed (Hobara et al., 2010; Mrachacz-Kersting et al., 2006). Activity in the early part of ground contact is a continuation of that centrally programmed function and also a function of the short latency stretch reflex response with the contribution of the pre-programmed function diminishing (Hobara et al., 2010; Mrachacz-Kersting et al., 2006). Thereafter it is likely to shift toward a supraspinal response of exponentially increasing contribution (Hobara et al., 2010). The magnitude of the short latency reflex response, therefore, may be affected by the amount of pre-activation. For instance, with high pre-activation, the 'amount' of activity 'allowable' from the short latency reflex response may decline given stretch may not be as great. Previous work which has measured muscle pre-activation has typically shown that gastrocnemius and soleus activation continually increases prior to ground contact (Horita et al., 2002; Kuitunen et al., 2002); that the level of pre-activation between the four quadriceps muscles and between the three hamstrings is not uniform (Butler et al., 2003; Hobara et al., 2010; Horita et al., 2002; Kuitunen et al., 2002); and that increased net quadriceps pre-activation relative to net hamstring pre-activation (antagonistic pre-activation co-contraction) may also be observed with increased speed and stiffness (Hobara et al., 2010; Kuitunen et al., 2002). However, studies which have discussed the role of muscle pre-activation K_{vert} or leg stiffness are limited by an inconsistency in analysis methodologies; with some reporting on filtered raw data (Horita et al., 2002; Kuitunen et al., 2002, 2007) whereas others have reported on muscle activation normalized to maximum voluntary contraction (MVC) (Farley et al., 1998; Hobara et al., 2010; Muller et al., 2010). Furthermore, only one study has actually discussed in detail the role of agonist to antagonist muscle activation for reducing COM displacement and subsequently increasing stiffness (Hobara et al., 2010); most typically only discuss pre-activation of individual muscles in isolation of each other. From a theoretical standpoint pre-activation of a single muscle is likely only to be loosely related to K_{vert} or leg stiffness as it does not provide any indication of muscle tension on either side of the joint. Where tension is not close to even on either side of the joint increased flexion angles will likely be observed and large displacements of COM or large reductions in leg length will ensue (Hobara et al., 2010); suggesting low stiffness considering Hooke's law.

A gap in the research also exists when it concerns task selection for measuring muscle pre-activation and stiffness. The relationship between stiffness and muscle activation has only been measured from straight line running tasks or hopping tasks, sometimes at controlled frequencies (Farley et al., 1998; Hobara et al., 2010; Horita et al., 2002; Kuitunen et al., 2002, 2007; Muller et al., 2010). Running tasks should be preferred due to their ecological validity, however it is understandable that hopping tasks are used in stiffness research as equipment and logistical constraints make it difficult to measure ground reaction force for over-ground running. If hopping tasks are used then hopping frequency should not be controlled as it is known that the natural frequency of the spring-mass system while hopping is equal to step frequency for slow gait tasks, but not for fast gaits (Cavagna et al., 1988). Therefore, controlling hopping frequency could slow the system from its natural running frequency, and consequently absolute stiffness of the spring mass system will not be measured rather just stiffness at submaximal pace. This is likely due to thigh muscle activation being able to modulate K_{vert} (Hobara et al., 2009). As such, athletes can consciously alter their vertical stiffness by increasing knee flexion (Butler et al., 2003). An argument for controlling hop frequency may be that by doing so athletes give less thought to

altering pre-activation strategies; however this is yet to be proven. Provided good reliability can be observed, it should be preferable to reduce conscious alteration of stiffness by simply requiring participants to hop with maximal effort with as little ground contact as possible.

Finally, as noted earlier, all work to date has examined stiffness and muscle pre-activation for straight line/sagittal plane tasks only. However, in sports where agility is a key performance indicator (e.g. the football codes and other field and court sports), stiffness while changing direction is important and therefore deserves more attention.

The aims of this project were firstly to establish if a relationship between muscle pre-activation strategies and K_{vert} for a single leg multidirectional hopping task existed which stressed the lower limbs similarly to change of direction running; and secondly, to determine if peak activation in the pre-activation period and the timing of that peak activation for each muscle was the same as that for their respective agonist muscles.

2. Methods

2.1. Experimental approach

The study presented in this paper was a cross-sectional correlational study with participants all from a single professional rugby club. They were asked to complete a 90 degree power-cut hop on and off a force platform at varying distances from the centre of the force platform, all bare foot. A power-cut hop was a single leg exercise requiring a jump at an angle of 45 degrees in the ipsilateral direction onto a designated point on the force platform, landing on the ipsilateral leg and hopping off as quick as possible at an angle of 90 degrees to land on the same leg at the set distance (see Fig. 1). Hops were performed at three distances to simulate change of direction at different speed. The test procedures were completed twice; the first occasion was for familiarisation. On the second testing occasion muscle activity was measured using EMG. The leg participants chose to hop on was self-selected.

2.2. Participants

Twenty males, all from a single professional rugby union club, agreed to participate in this study. Participant age, stature and body mass was 24.0 ± 4.4 years, 185.3 ± 11.9 cm and 100.6 ± 18.5 kg (mean \pm SD) respectively. All were healthy with no history of lower limb injury in the 12 months prior to data collection.

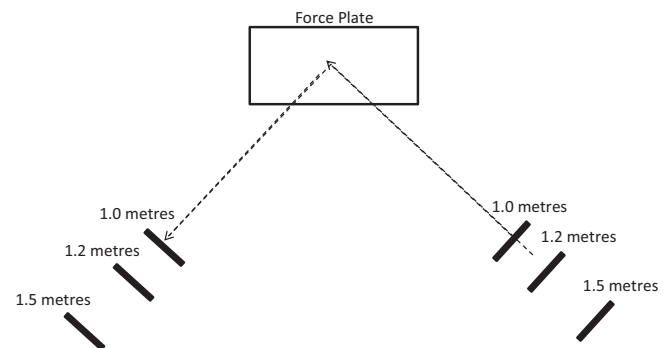


Fig. 1. Power-cut hop test. In the above diagram the participant would be completing a right foot 1.0 m power-cut hop. That is, off their right foot they would leave the 1.0 m mark on the right of the diagram and land on, and jump off, their right foot on the force plate as quick as possible before landing past the 1.0 m mark on the left of the diagram on their right foot.

2.3. Procedures

Ethical approval to conduct this research project was granted by the University and the local health department human research ethics committees in accordance with the Declaration of Helsinki. Each participant gave informed consent.

Muscle activity was measured using an eight-channel Megawin WBA telemetry EMG system (Mega Electronics, Kuopio, Finland) from six muscles (gluteus maximus, vastus lateralis, vastus medialis, biceps femoris, semimembranosus, and medial gastrocnemius) while participants performed the bare foot power-cut hopping task on and off a Kistler 9281C force platform (Kistler Group, Winterthur, Switzerland) at distances of 1.0 m, 1.2 m and 1.5 m from the centre of the force platform.

2.3.1. Electromyography data collection

Prior to application of electrodes skin was shaved and cleaned with alcohol swabs (Kendall Healthcare Products Company, USA). Two monopolar Ag-AgCl disc shaped surface electrodes with a 2 cm radius (Ambu, Denmark) were then placed at the approximate centre of each muscle belly with a minimum of 1 cm separation. Electrodes were pre-lubricated with ultrasound gel by the manufacturer. Electrode leads were secured to the leg with hypo-allergenic strapping tape (Elastoplast Sports, Australia) to minimize movement. The EMG signal was recorded by telemetry and pre-amplified using analogue differential amplification. It was converted from analogue to digital using an A/D converter (National Instruments NIUSM-6210, New South Wales, Australia) with a pre-amplifier gain of 305. A band pass filter 12–450 Hz and a sampling rate of 1000 Hz with a common mode rejection ratio of 60 dB was applied. The signal was saved to a personal computer using the Bioware software package (Kistler Group, Winterthur, Switzerland). In Bioware a 10 Hz high pass dual pass butterworth filter was applied to the EMG signal. The EMG signals were visually checked for artefacts, then a root mean squared (RMS) filter was applied at a non-overlapping moving window length of 20 ms.

The RMS EMG for MVC (mV) was established for each of the muscles prior to execution of the power-cut hopping tasks. Due to the known ineffectiveness of an isometric contraction to normalize dynamic movements (Ball and Scurr, 2010), MVC was established from dynamic maximal efforts. The RMS EMG MVC (mV) for the gluteus maximus, vastus lateralis and vastus medialis was recorded from a squat jump in accordance with methods previously reported (Ball and Scurr, 2012). The RMS EMG MVC (mV) for biceps femoris and semimembranosus was recorded from a nordic lower in accordance with methods previously reported (Ditroilo et al., 2013). The RMS EMG MVC (mV) for medial gastrocnemius was recorded from five single leg continuous hops over 30 cm hurdles.

For the power-cut hopping task EMG data was synchronised with force plate data to the instant of foot contact and pre-activation was considered mean RMS EMG (mV) in the 100 ms prior to foot contact. Mean normalized RMS EMG was the pre-activation RMS EMG (mV) relative to RMS EMG for the MVC (mV) and was presented as a proportion of MVC (%). Peak normalized RMS EMG (%) during the pre-activation and timing of peak RMS EMG relative to foot contact (ms) was also noted. Finally, a co-activation index, which is the ratio of mean normalized RMS EMG for antagonistic muscle activity to agonistic activity was calculated for the medial quadriceps and hamstring muscles (vastus medialis and semimembranosus), and the lateral quadriceps and hamstring muscle (vastus lateralis and biceps femoris).

2.3.2. Vertical stiffness data collection

Vertical stiffness was considered the quotient of maximum vertical GRF and whole body COM displacement. The force platform

was interfaced with a personal computer and Bioware software was used to record vertical GRF at 1000 Hz for each of the power-cut hops. A 10 Hz high pass dual pass butterworth filter was applied to the raw force plate data. Data was then exported to purpose built software (BioAlchemy, Adelaide, Australia) so that filtered data from Bioware could be processed for calculation of K_{vert} . In summary, vertical displacement of COM was established by double integration of vertical acceleration according to the protocol of Cavagna (1975); the cumulative sum of the vertical force (N/s) was integrated, then point by point integration of the previously integrated force was performed (Cavagna, 1975).

In order to test the reliability of K_{vert} measurement for the power-cut hopping task we tested 14 participants from a professional rugby union population with age, height and body mass of 26.0 ± 4.3 years, 168.8 ± 25.2 cm and 89.5 ± 14.3 kg (mean \pm SD) respectively separately to this study. A typical error of measurement (TEM) for vertical stiffness for the 1.0 m, 1.2 m and 1.5 m power-cut hops was 4.3%, 4.9% and 5.7% respectively was revealed. Typical error of measurement for contact time for the power-cut hops at each distance was 1.7%, 2.1% and 1.8% respectively.

2.4. Statistical analysis

All statistical analyses were performed using the Statistical Package for Social Sciences (SPSS) software (IBM, New York, USA) on an Acer laptop computer (Acer Australia, New South Wales, Australia). Descriptive statistics were calculated for K_{vert} , pre-activation RMS EMG (mV), pre-activation normalized RMS EMG (%), for co-activation index, for pre-activation peak RMS EMG (%), and for timing (ms) of peak RMS EMG during the pre-activation period. Pearson correlations were calculated between K_{vert} and pre-activation RMS EMG, K_{vert} and normalized pre-activation RMS EMG, and also for co-activation index and K_{vert} . Thresholds for correlations were considered according to those described by Hopkins et al. (2009) (0.1, 0.3 and 0.5 for small, moderate and large correlations respectively), and $\alpha < 0.05$ for all. Independent samples t-tests were applied to compare agonist and antagonist peak RMS EMG and its timing in the 100 ms prior to foot contact to establish if the magnitude of the muscle activation on each side of the joint was the same and if timing of peak activation occurred at the same time. Effect size was also calculated once t-tests were applied.

3. Results

Vertical stiffness (mean \pm SD) for the 1.0 m, 1.2 m and 1.5 m power-cut hops was 16.93 ± 8.55 , 12.72 ± 6.16 , and 14.39 ± 6.47 kN/m respectively. A typical RMS EMG trace for a 1.0 m power-cut hop may be seen in Fig. 2.

No obvious trend was observed for K_{vert} and pre-activation RMS EMG for each muscle, however typically a moderate positive relationship was seen for K_{vert} and quadriceps and hamstrings pre-activation normalized RMS EMG on the 1.0 and 1.2 m power-cut hops. (see Table 1). Pre-activation of the gluteus maximus was similar in observation to pre-activation of the hamstring muscles; the relationship between K_{vert} and RMS EMG was negligible, however the relationship between K_{vert} and gluteus maximus pre-activation normalized RMS EMG neared moderate. Finally, for the medial gastrocnemius no trend was observed for any relationship between pre-activation and K_{vert} .

When considering pre-activation in terms of co-activation of the medial and lateral quadriceps and hamstrings, a moderately positive relationship between the lateral knee flexors and extensors (vastus lateralis and biceps femoris respectively) co-activation index and K_{vert} was observed for the 1.0 m and 1.2 m power-cut

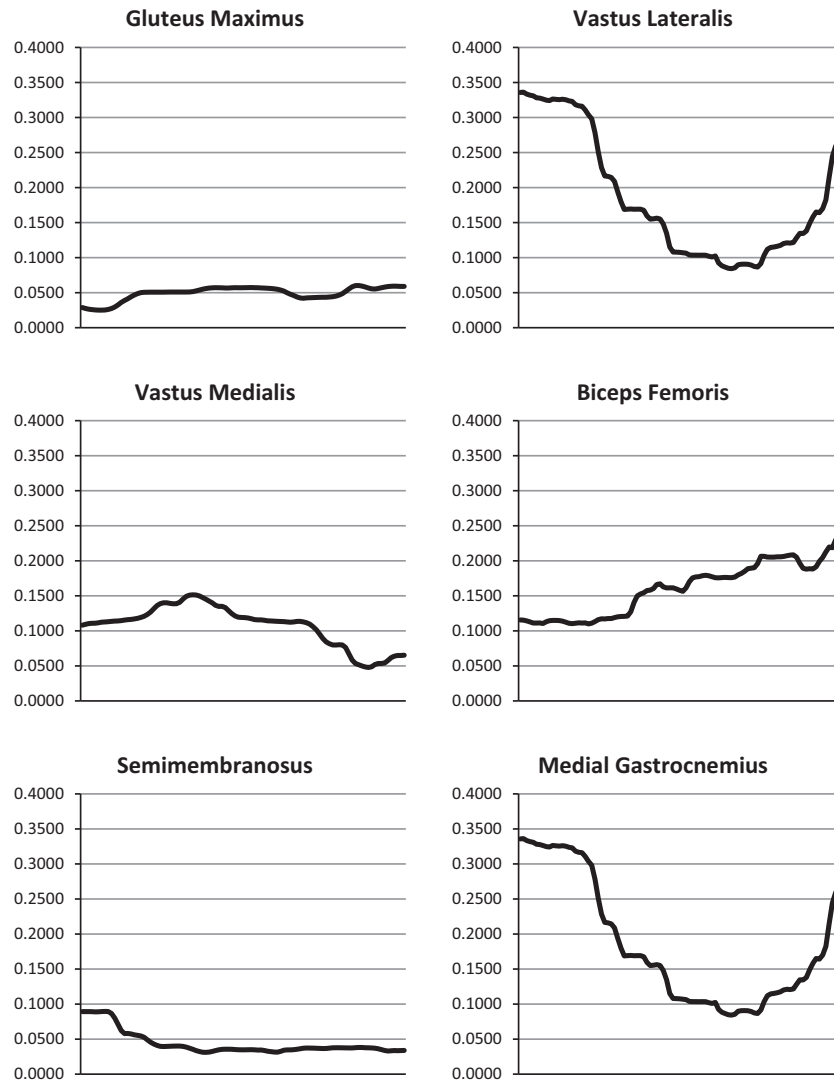


Fig. 2. Typical 10 Hz dual pass butterworth filtered 20 Hz RMS EMG signal for 1.0 m power-cut hop for the pre-activation period, that is the 100 ms prior to foot contact. Data is not normalized.

Table 1
Muscle pre-activation absolute (RMS EMG) and proportionate to MVC (mean \pm SD) and their relationship to K_{vert} .

	1.0 m Power-cut hop Pre-activation		1.2 m Power-cut hop Pre-activation		1.5 m Power-cut hop Pre-activation	
	RMS EMG (mV)	% MVC	RMS EMG (mV)	% MVC	RMS EMG (mV)	% MVC
Gluteus Maximus	0.0690 \pm 0.0776	25 \pm 20	0.0768 \pm 0.0804	28 \pm 23	0.0975 \pm 0.0809	33 \pm 22
(K_{vert} Correlation)	(-0.16)	(0.26)	(0.16)	(0.12)	(-0.05)	(0.09)
Vastus Lateralis	0.1437 \pm 0.0741	24 \pm 14	0.1770 \pm 0.1078	29 \pm 19	0.3303 \pm 0.2053	48 \pm 25
(K_{vert} Correlation)	(0.25)	(0.32)	(-0.19)	(0.32)	(0.41)	(0.27)
Vastus Medialis	0.1294 \pm 0.0735	25 \pm 16	0.1740 \pm 0.0835	36 \pm 20	0.2303 \pm 0.0968	47 \pm 22
(K_{vert} Correlation)	(0.45)	(0.36)	(0.23)	(0.25)	(0.18)	(0.15)
Biceps Femoris	0.1085 \pm 0.0531	32 \pm 19	0.1109 \pm 0.0414	33 \pm 18	0.1092 \pm 0.0530	33 \pm 21
(K_{vert} Correlation)	(-0.05)	(0.36)	(0.05)	(0.15)	(-0.27)	(0.00)
Semimembranosus	0.1496 \pm 0.0792	25 \pm 15	0.1383 \pm 0.0667	24 \pm 13	0.1478 \pm 0.1020	28 \pm 22
(K_{vert} Correlation)	(0.17)	(0.40)	(0.12)	(0.16)	(0.25)	(0.35)
Medial Gastrocnemius	0.1770 \pm 0.0724	32 \pm 15	0.1978 \pm 0.0657	37 \pm 17	0.1655 \pm 0.0670	30 \pm 15
(K_{vert} Correlation)	(-0.10)	(0.04)	(0.28)	(0.04)	(-0.05)	(-0.42)

NB: RMS = Root Mean Square; MVC = Maximum voluntary contraction; K_{vert} Correlation = Pearson's correlation $\alpha < 0.05$.

hops. There was a negligible relationship between the medial thigh muscles (vastus medialis and semimembranosus) co-activation index and K_{vert} (see Table 2).

Finally, no significant difference was typically seen for peak normalized RMS EMG during the pre-activation period between

the quadriceps or the hamstring muscles during the pre-activation period. The only statistically significant difference in timing observed was for peak RMS EMG of the biceps femoris occurring after vastus lateralis for the 1.0 m power-cut hop (Table 3). Descriptive statistics did, however, allude that timing of peak thigh

Table 2
Muscle pre-activation co-activation indexes (mean \pm SD) and their relationship to K_{vert} .

	1.0 m power-cut hop		1.2 m power-cut hop		1.5 m power-cut hop	
	Pre-activation co-activation Index	K_{vert} correlation	Pre-activation co-activation Index	K_{vert} correlation	Pre-activation co-activation Index	K_{vert} Correlation
Knee lateral flexors/extensors (BF/VL)	1.32 \pm 1.04	0.43	1.43 \pm 1.41	0.31	0.81 \pm 0.71	0.04
Knee medial flexors/extensors (SM/VM)	1.47 \pm 1.26	0.06	0.97 \pm 0.95	0.11	0.92 \pm 1.02	-0.07

NB: K_{vert} = Vertical stiffness; BF = Biceps Femoris; VL = Vastus Lateralis; VM = Vastus Medialis; SM = Semimembranosus; K_{vert} Correlation = Pearson's Correlation $\alpha < 0.05$.

Table 3
Peak muscle activation as a percentage of MVC (mean \pm SD) and timing of peak muscle activation (mean \pm SD) in pre-activation period for agonist/antagonist muscles.

	Knee lateral flexors/extensors (BF/VL)								
	1.0 m power-cut hop			1.2 m power-cut hop			1.5 m power-cut hop		
	Biceps femoris	Vastus lateralis	Effect size	Biceps femoris	Vastus lateralis	Effect size	Biceps femoris	Vastus lateralis	Effect size
Peak muscle activation (%MVC) (K_{vert} Correlation)	44 \pm 24 (0.34)	33 \pm 16 (0.50)	0.52	46 \pm 24 (0.19)	38 \pm 34 (-0.32)	0.13	41 \pm 22 (-0.01)	70 \pm 34 (0.19)	0.94
Timing of peak muscle activation (ms prior to foot contact) (K_{vert} correlation)	50 \pm 37 (0.01)	13 \pm 21* (-0.12)	1.03	51 \pm 33 (0.04)	34 \pm 40 (-0.01)	0.52	35 \pm 35 (-0.04)	27 \pm 27 (0.34)	0.25
	Knee medial flexors/extensors (VM/SM)								
	1.0 m power-cut hop			1.2 m power-cut hop			1.5 m power-cut hop		
	Semime-branosus	Vastus medialis	Effect size	Semime-branosus	Vastus medialis	Effect Size	Semime-branosus	Vastus medialis	Effect size
Peak muscle activation (%MVC) (K_{vert} Correlation)	40 \pm 25 (0.21)	44 \pm 25 (0.56)	0.14	37 \pm 26 (0.18)	56 \pm 23 (0.30)	0.74	40 \pm 32 (0.50)	67 \pm 28 (0.10)	0.81
Timing of peak muscle activation (ms prior to foot contact) (K_{vert} Correlation)	57 \pm 42 (0.15)	28 \pm 36* (-0.05)	0.71	54 \pm 36 (-0.19)	23 \pm 26* (0.15)	0.91	58 \pm 36 (0.32)	28 \pm 37 (-0.02)	0.22

NB: BF = Biceps Femoris; VL = Vastus Lateralis; SM = Semimembranosus; VM = Vastus Medialis; *significantly different to antagonist muscle $\alpha < 0.05$; K_{vert} Correlation = Pearson's correlation $\alpha < 0.05$.

muscle activation on either side of the knee joint did vary for flexors and extensors with peak activation of the hamstrings typically occurring after peak activation of the quadriceps (Fig. 2 and Table 3). The positive relationship between K_{vert} and magnitude of peak RMS EMG was stronger than that for K_{vert} and timing of peak RMS EMG (Table 3).

4. Discussion

There were two aims for the research project presented in this paper; (1) to establish if a relationship between muscle pre-activation strategies and K_{vert} for a single leg hopping task which stressed the lower limbs similarly to change of direction running existed, and (2) to determine if normalized peak RMS EMG and its timing for each muscle was the same as that for their respective agonist muscles. Results from this study showed that a typically moderate positive relationship between pre-activation RMS EMG and K_{vert} exist. The strength of that relationship increased when RMS EMG was normalized, and again when considering pre-activation co-activation index for the lateral hamstring and quadriceps (biceps femoris and vastus lateralis). That the strength of the relationship increased in such a manner was not unexpected given muscle force production is dependent on a range of factors including attachment sites, cross sectional area, fascicle length etc. Thus, while absolute force production by each muscle on either side of the joint may vary, relative to each other it is likely to near equal when little flexion and extension is observed and consequently increased K_{vert} will likely be seen as centre of mass displacement will not be as great. Furthermore, when executing change of direction

manoeuvres similar to those described in this project it would be expected that the lateral quadriceps and hamstring muscles 'work' more than the medial to facilitate lateral movement in the direction opposite to the stance leg, hence the moderate positive relationship between pre-activation co-activation index and K_{vert} for the lateral hamstrings–quadriceps but not the medial.

Another relatively novel aspect of the present study was related to timing of peak activation of the hamstrings and quadriceps during pre-activation for change of direction manoeuvres. Visual inspection of RMS EMG traces showed that timing of peak activation appeared to vary for the hamstrings and the quadriceps. It should be noted, however, that for only the lateral hamstrings and quadriceps on the 1.0 m power-cut hop this observation was made with statistical significance. In most instances the difference in timing was not statistically significant and did not appear to be related to K_{vert} despite descriptive statistics alluding to a difference for the lateral hamstring and quadriceps for the 1.2 m power-cut hop. Furthermore, magnitude of normalized peak RMS EMG for muscles on either side of the knee joint were not significantly different or related to K_{vert} . Consider that peak activation will influence mean activation, combined with the fact that a typically moderate relationship was seen between pre-activation co-activation index was seen for the lateral hamstring–quadriceps and K_{vert} , these results therefore suggest that on some level timing of peak activation influences K_{vert} .

Results from the present study were not surprising given previous work has shown that pre-activation strategies (Farley et al., 1998; Hobara et al., 2010; Horita et al., 2002; Kuitunen et al., 2002, 2007; Muller et al., 2010) and pre-activation co-activation of the quadriceps and hamstrings (Hobara et al., 2010; Mero and

Komi, 1987) plays a role in adjusting leg stiffness for sagittal plane tasks. Furthermore, it has been reported that loading of the muscles responsible for external rotation and valgus moments about the knee (i.e. lateral quadriceps and hamstrings) to be greater for power-cut change of direction type manoeuvres (Besier et al., 2003, 2001), and co-activation pre-activation can stabilize the knee joint for change of direction running (Besier et al., 2003). Finally, and also consistent with previous research for straight line running (Mero and Komi, 1987), it is worth highlighting that peak activation of the hamstrings was greater when stiffness was greater, and, although not typically statistically significant in the present study, it typically occurred after peak activation of the quadriceps.

Despite data from the present study implying a closed relationship of lateral hamstring and quadriceps muscle pre-activation co-activation (biceps femoris–vastus lateralis) with K_{vert} for change or direction tasks, it is more likely to be a general one. The role of each muscle (e.g. biceps femoris or vastus lateralis) in a given muscle group (hamstrings or quadriceps respectively) may be modulated by other synergistic muscles (e.g. semimembranosus or vastus medialis) (Neptune et al., 2001). Synergism is defined as the distribution of force among individual muscle to produce a given task (Zatsiorsky and Prilutsky, 2012). The central nervous system considers synergistic muscles as a functional unit as opposed to individual muscles when producing or maintaining force based on common drive (De Luca and Erim, 2002). Therefore, the greater pre-activation co-activation of the lateral and hamstring and quadriceps muscles (biceps femoris and vastus lateralis) relative to the medial (semimembranosus and vastus medialis) is not likely to have been to change knee joint angle, rather to stabilize the knee joint for the primary action of change of direction hopping or running. This theory is consistent with previous literature which has argued pre-activation and stabilization (Besier et al., 2003), and explains the stronger relationship of the biceps femoris–vastus lateralis co-activation index compared to the semimembranosus–vastus medialis co-activation index with K_{vert} . To go into further detail regarding synergisms is beyond the scope of this paper, however future research in this area may be beneficial.

Some may consider the strength of the relationships between pre-activation strategies and K_{vert} in the present study to be low or negligible. However, it should be considered that K_{vert} is a measure of whole body stiffness reliant upon flexion and extension of ankle, knee, hip and vertebral column joints as well as the interaction of anatomical structures including muscle, cartilage, ligaments, tendons and bone (Serpell et al., 2012). Therefore, it is not reasonable to expect more than a moderate positive relationship between the activation of muscles surrounding the knee joint and K_{vert} . It would be expected that similar relationships between pre-activation strategies from muscles surrounding other joints and K_{vert} exist. This is supported by work which has shown that a more erect posture increases K_{vert} (Devita and Skelly, 1992), work which has discussed the role of touch down angle at foot contact (which is also affected by hip joint angle and stiffness) for K_{vert} (Farley et al., 1998; Hobara et al., 2010; Muller et al., 2010), and work which has shown that vertical stiffness is also affected by ankle joint stiffness as well as knee joint stiffness (Farley et al., 1998; Hobara et al., 2010; Kuitunen et al., 2002; Muller et al., 2010). However, we do recognise that more rigorous analyses, especially non-linear analyses (Preatoni et al., 2013), would be beneficial for future studies to strengthen the theory of a relationship between K_{vert} and pre-activation strategies. Thus, while the strength of the relationships between K_{vert} and the muscles tested in the present study for pre-activation were not typically positively strong, results certainly do support the theory that muscle pre-activation plays a role in modulating K_{vert} especially if pre-activation co-activation is even on both sides of the joint whether that joint is the ankle, knee, hip or the vertebral column.

It is worth pointing out that the reliability of the measurement from the 1.5 m power-cut hop in the present study was borderline with a TEM of greater than 5%. Therefore, results from those hops should be interpreted with caution and as such have not drawn much attention for discussion in the present study. Consider also that the inclusion of three hops and requiring participants to hop as quickly as possible was a method used to simulate change of direction running at various paces. Hobara et al. (2010) has argued that step frequency for fast gaits is less than that for fast hopping frequencies. Therefore, it is possible that the 1.5 m power-cut hopping task does not accurately reflect rapid change of direction while running. Nevertheless, the reliability of K_{vert} measurements for the 1.0 m and 1.2 m power-cut hops combined with the reliability of contact time for those hops demonstrating K_{vert} was not typically different at the greater distance due to increased ground contact time and increased muscle moments, suggest the findings from the present study are ecologically valid. This is further supported by the effect sizes observed for results related to timing and magnitude of peak RMS EMG for the hamstrings and quadriceps (Table 3) suggesting that results can well be applied to the populations from which the sample was taken.

5. Conclusion

In summary, results from the present study suggest that pre-activation strategies play a role in modulating K_{vert} for change of direction running manoeuvres. Specifically, hamstring activation normalized to MVC will increase and near magnitude to that of normalized quadriceps activity as stiffness increases. Furthermore, for power-cut change of direction manoeuvres the lateral hamstring muscles (biceps femoris) become more active than their medial counterparts and this will consequently be reflected in the pre-activation co-activation index of the lateral quadriceps–hamstrings. Consequently, the pre-activation co-activation index of the biceps femoris–vastus lateralis is more related to K_{vert} for change of direction manoeuvres than the pre-activation co-activation index of semimembranosus–vastus medialis. However, this is likely due to synergism of the muscle groups to assist stabilizing the knee for the primary action for change of direction hopping or running. Therefore, the relationship of hamstring–quadriceps pre-activation co-activation and K_{vert} is likely to be a general one as opposed to a closed relationship which only exists with the lateral muscle groups (biceps femoris–vastus lateralis).

Conflict of interest

The fourth author is owner of BioAlchemy. No other conflicts exist and no financial assistance was awarded for this project.

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2.2. CONTEXT OF STUDY

This study described the relationship between pre-activation strategies of the lower limb muscles and vertical stiffness on a multidirectional hopping task which simulated the change of direction manoeuvre typically observed when non-contact ACL injury occurs. For this task pre-activation of lower limb muscles was typically positively related to vertical stiffness ($\alpha < 0.05$); the relationship was stronger when pre-activation was normalised to maximum voluntary contraction ($\alpha < 0.05$). The concept of co-activation was introduced and defined as a ratio of antagonistic to agonistic muscle activity, and a positive relationship was typically observed for vertical stiffness with co-activation of the muscles surrounding the knee joint ($\alpha < 0.05$). There also appeared to be a link for vertical stiffness with peak activation and timing of peak activation of the hamstring muscles. Consequently, it was argued that thigh muscle pre-activation strategies influenced vertical stiffness for multidirectional hopping tasks. However, it was also identified that the strength of the relationships for pre-activation with vertical stiffness differed between the medial and lateral hamstrings and quadriceps muscles.

That the strength of relationships with vertical stiffness was not uniform for each muscle or co-activation index was not concerning because activation does not equal force production. Force production is dependent on a range of factors including muscle attachment sites, cross sectional area, fascicle length etc. As such a smaller level of activation in a 'large' muscle with attachment sites nearing right angles to each other might produce a greater force than a high level of activation in a 'smaller' muscle with attachment sites near adjacent. Hence relationships for vertical stiffness with pre-activation of each muscle in isolation was typically stronger when normalised to maximum voluntary contraction. Furthermore, in this study it was noted that muscles rarely work in isolation. Rather they work synergistically.

Synergism refers to the distribution of force across different muscles (e.g. the three hamstring muscles work together to produce a flexion moment) (De Luca and Erim, 2002, Zatsiorsky and Prilutsky, 2012), and it is understood that the central nervous system considers synergistic muscles as a functional unit as opposed to individual muscles when producing or maintaining force based on common drive (De Luca and Erim, 2002). In this context, therefore, the role of each muscle in the hamstring and quadriceps muscle groups for the multidirectional hopping task could have been dependent on how other muscles in the muscle group operates (Zatsiorsky and Prilutsky, 2012, De Luca and Erim, 2002). This understanding of muscle synergism is assumed for the remainder of this thesis.

In summary, therefore, this study showed that pre-activation and co-activation of the muscles of the lower limbs for multidirectional tasks which simulate the change of direction manoeuvre typically observed when non-contact ACL injury occurs can be present. These pre-activation strategies are positively related to vertical stiffness. It could be thought that pre-activation co-activation of these muscles, and by extension vertical stiffness, constrains active laxity/enhance dynamic knee joint stability however no link between vertical stiffness and non-contact ACL injury, or the protective nature of vertical stiffness for non-contact ACL injury, could be made from this study.

CHAPTER 3

Vertical Stiffness & Muscle Strain Injury

3.1. Vertical Stiffness and Muscle Strain in Professional Australian Football

3.2. Context of Study

3.1. VERTICAL STIFFNESS AND MUSCLE STRAIN IN PROFESSIONAL AUSTRALIAN FOOTBALL

Statement confirming the authorship contribution of the PhD candidate

On behalf of all co-authors of the paper:

SERPELL, B.G., SCARVELL, J.M., BALL, N.B. & SMITH, P.N. 2014. Vertical stiffness and muscle strain in professional Australian football. *J Sports Sci*, 32, 1924-1930.

I confirm that Benjamin Serpell has made the following contributions:

- Contributed to discussions on design of the study
- Collection, analysis and synthesis of literature
- Submission for ethics approval, recruitment of subjects, and collection of data under supervision
- Analysis and interpretation of data under supervisions
- Wrote the first draft of the manuscript, and followed through to publication including proofing and final publication details of the manuscript

Professor Paul N. Smith

Signed:..... Date:.....

Vertical stiffness and muscle strain in professional Australian football

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Abstract

The purpose of this study was to establish if vertical stiffness was greater in professional Australian rules footballers who sustained a lower limb skeletal muscle strain compared to those who did not, and to establish if a relationship between age, or training history, and vertical stiffness existed. Thirty-one participants underwent weekly rebound jump testing on a force platform over two seasons. Vertical stiffness was calculated for injured players and the uninjured cohort 1 and 3 weeks prior to sustaining an injury and at the end of preseason. Eighteen athletes were in the “uninjured” cohort and 13 in the “injured” cohort. No significant difference in vertical stiffness was observed between groups ($P = 0.18$ for absolute stiffness; $P = 0.08$ for stiffness relative to body mass), within groups ($P = 0.83$ and $P = 0.88$, respectively) or for a time*cohort interaction ($P = 0.77$ and $P = 0.80$, respectively). No relationship between age and vertical stiffness existed ($r = -0.06$ for absolute and relative stiffness), or training history and vertical stiffness ($r = -0.01$ and 0.00 for absolute and relative stiffness, respectively) existed. These results and others lend to suggest that vertical stiffness is not related to lower limb muscle strain injury.

Keywords: injury, muscle mechanics, stiffness, neuromuscular, stretch shortening cycle

Introduction

The rate at which research into stiffness in the human body is being published in sports science and medicine literature is increasing exponentially (Serpell, Ball, Scarvell, & Smith, 2012), most likely because stiffness has been linked to superior sporting performance for running sports (Bret, Rahmani, Dufour, Messonnier, & Lacour, 2002; Hobara et al., 2008; Serpell et al., 2012). However, a trade-off between stiffness and injury might exist (Butler, Crowell, & Davis, 2003; Serpell et al., 2012). A recent prospective case control study reported mean bilateral leg stiffness at the end of preseason was greater in Australian rules footballers who sustained a hamstring injury during the subsequent season compared to those who did not (Watsford et al., 2010). However, other work has concluded with differing results (Pruyn et al., 2012). This conflict may be related to limitations with study methodology. Regardless, these results have called for clarity over whether stiffness should be considered a predictor for muscle strain injury.

The “stiffness” concept is derived from Hooke’s law which states that the force required to deform an

object is related to a proportionality constant (spring) and the distance that object is deformed (Austin, Garrett, & Tiberio, 2002; Butler et al., 2003). Often the human body or body segments are modelled as a spring (Butler et al., 2003). Thus, stiffness in the human body requires interaction of anatomical structures such as tendons, ligaments, muscles, cartilage and bone to resist change once ground reaction force or moments are applied (Brughelli & Cronin, 2008; Serpell et al., 2012). Vertical stiffness (i.e. the quotient of maximum ground reaction force and centre of mass displacement) is a measure of whole body stiffness. Given centre of mass displacement continues as a function of ground reaction force during flight then it is a measure of stiffness through the whole gait cycle (i.e. stance and flight) (Serpell et al., 2012). Leg stiffness (i.e. the quotient of maximum ground reaction force and change in leg length) is a measure of stiffness of the lower limb reliant on leg compression which can only be achieved during stance (Serpell et al., 2012). Vertical stiffness is affected by factors which do not affect leg stiffness (e.g. poor hip control, spinal cord flexion vertebral disks compression

etc.), and may be measured in the field with only a force platform. Accurate measurement of leg stiffness requires the use of specialised motion analysis equipment to measure leg compression during stance (Serpell et al., 2012). Nevertheless, the terms “vertical stiffness” and “leg stiffness” are often used interchangeably, exposing research which reports on measuring leg stiffness using methods for measuring vertical stiffness to threats to internal validity (Hébert-Losier & Eriksson, 2014; Serpell et al., 2012).

A limitation of aforementioned Australian rules football studies was that they claimed to measure leg stiffness when in fact vertical stiffness from single leg hopping was measured (Pruyn et al., 2012; Watsford et al., 2010). In the earlier of the two studies, it was argued that high mean bilateral leg stiffness was a risk factor for hamstring injury (Watsford et al., 2010). Bilateral leg stiffness was the mean of left and right leg stiffness. Leg stiffness was considered the quotient of ground reaction force and centre of mass displacement from a single leg hopping task requiring participants to hop in time to a metronome. Using similar methodology the latter study showed no difference in mean bilateral leg stiffness between their injured and uninjured groups throughout the season (Pruyn et al., 2012). In the earlier study no asymmetry in leg stiffness was observed for either group, while the latter study reported asymmetry from their injured group (Pruyn et al., 2012; Watsford et al., 2010). Therefore whether stiffness was a risk factor for hamstring injury or it were related to confounding factors remains unclear.

Further limitations to the Australian rules football studies were that neither controlled for injury history; a history of muscle strain injury is a risk factor for muscle strain injury (Opar, Williams, & Shield, 2012; Orchard, 2001; Pruyn et al., 2012; Watsford et al., 2010). In the study which showed a link between stiffness and injury the mean age of the injured cohort was greater than that of the uninjured cohort; age is a risk factor for hamstring injury (Opar et al., 2012; Orchard, 2001). In both studies participants were required to hop at a controlled constant frequency, and both studies defined injury differently.

By controlling hopping frequency precise measurement of the stiffness of a system will not be achieved, rather stiffness at a defined velocity. The natural frequency of a system while hopping is equal to the step frequency for slow gait tasks but not for fast gaits (Cavagna, Franzetti, Heglund, & Willems, 1988). Controlling hopping frequency could alter the system’s hopping frequency from its natural running frequency, and consequently absolute stiffness of the spring mass system will not be measured. This is likely because thigh muscle activation can modulate leg stiffness and vertical stiffness (Butler et al.,

2003; Hobarra et al., 2009). It may be argued that controlling hopping frequency ensures athletes give less thought to modulating knee flexion and therefore vertical stiffness; however, this is yet to be proven. It should be preferable to reduce conscious alteration of stiffness by requiring participants to hop with maximal effort with as little ground contact as possible

Finally, how an injury is defined may influence biases towards certain injuries and subsequently affect study outcomes (Bailey, Scase, Heynen, & Margarey, 2010). Arguably, the most commonly used definition of an injury is that described by Orchard and Seward (2002): any condition that prevents an athlete from competing in a regular event. Given that severity of injury is often described in terms of number of competitions missed (Orchard & Seward, 2002), then it is reasonable to suggest this definition appropriate. In earlier of the two Australian rules football studies, injury was defined as a palpable soreness which resulted in a missed game (Watsford et al., 2010), but because soreness on stretch and power was not assessed it is possible that the injury was not a muscle strain. In the latter study, injury was any recorded complaint made to medical staff (Pruyn et al., 2012). Therefore, whether vertical stiffness increases the risk of muscle strain injury remains unclear.

In the study presented in this paper, vertical stiffness was measured in age matched injured and uninjured groups in a professional Australian rules football population at the end of preseason, approximately 3 weeks prior to the injury occurring, and within a week of the injury occurring. The decision to analyse vertical stiffness 3 weeks prior to injury was arbitrary; no seasonal variation in stiffness exists for professional Australian rules footballers (Pruyn et al., 2013). Injury history was controlled for, hopping frequency was not, and injury was clearly defined. The primary aim of the study was to determine if vertical stiffness was a predictor for lower limb muscle strain in professional Australian rules footballers. We also sought to establish if a relationship between age, or training history in a professional training environment, and vertical stiffness existed. If vertical stiffness was not a predictor for muscle strain injury and was not related to age or training history then training for vertical stiffness to enhance performance may be recommended with reduced fear of injuring athletes.

Methods

Experimental approach

The study presented in this paper was a retrospective cohort study. Jump test data were collected at

a professional Australian rules football club prospectively as a standard procedure for monitoring training; however, it was analysed for vertical stiffness retrospectively. Two football seasons of data were analysed. Data were only collected through the competition phase of each season. The duration of each competition phase ran for 24 weeks commencing in February. The external validity of the method used to measure vertical stiffness has been discussed elsewhere (Cavagna, 1975; Serpell et al., 2012). A pilot analysis of two jump tests from 15 participants from the present study was performed separately to establish test–retest reliability. The second jump test for the test–retest reliability analysis was performed following a reasonable time to facilitate full recovery from the first jump test (5–10 min).

Participants

All data from all players were considered for analysis. However, data from players who sustained a lower limb muscle strain injury in the 12 months prior to the commencement of each competition phase were excluded, and if a participant sustained a muscle strain he was not re-entered into the study. If there were missing data at any of the three time points for any player, all data were excluded for that player.

Data from 31 participants from a single professional Australian rules football club were included in this study. The “injured” cohort comprised 13 participants, and the “uninjured” cohort comprised 18 participants. The number of participants was similar in the previous Australian rules football studies (Pruyn et al., 2012; Watsford et al., 2010).

Table I describes participant demographics. All were of Anglo-Saxon ethnicity.

Procedures

Ethical approval to conduct this project was granted by the university and the local health department human research ethics committees

Testing protocol. During both seasons all players underwent “jump testing” on a weekly basis 72 h after playing a game. For each jump test participants completed a standardised warm-up consisting of approximately 5 min of light jogging, followed by dynamic stretching. Before each test, players stood on a force platform for 4 s so that body mass could be measured. Then, holding a broomstick across their posterior shoulders, participants were asked to jump as high as they could twice without pausing between jumps. From the second jump maximum vertical ground reaction force (kN) and vertical acceleration ($\text{m} \cdot \text{s}^{-2}$) data were recorded. Centre of mass displacement was calculated by double integration of vertical acceleration according to the protocol of Cavagna (1975). Finally, vertical stiffness was calculated as the quotient of ground reaction force and centre of mass displacement.

Once an athlete sustained a soft tissue injury, vertical stiffness for that player and the uninjured cohort was noted from three data points; at the end of preseason, approximately 3 weeks prior to the injury occurring and within 1 week of the injury occurring. Several jumps from the uninjured cohort were included for analysis at each data point because not all injuries occurred at the same time. For example, two separate injuries may have occurred 4 weeks apart. Therefore, the time between the “end of preseason” measure and the occurrence of each injury would have been different. Therefore, vertical stiffness data for the uninjured cohort 3 weeks prior to the first injury was combined to vertical stiffness data for the uninjured cohort 3 weeks prior to the second injury occurring. Consequently, 13 jumps from 13

Table I. Participant demographics for the injured and uninjured cohort at the end of preseason, 3 weeks prior to the occurrence of a skeletal muscle strain and within one week prior to the occurrence of a skeletal muscle strain.

	End of preseason (mean \pm SD)			Near to 3 weeks prior to injury occurrence (mean \pm SD)			Within 1 week of injury occurrence (mean \pm SD)		
	Injured	Uninjured	Effect size	Injured	Uninjured	Effect size	Injured	Uninjured	Effect size
Number of days prior to injury occurring	82.2 \pm 56.2	90.5 \pm 56.0		19.4 \pm 8.7	18.1 \pm 7.7		6.8 \pm 5.0	6.3 \pm 4.6	
Body mass (kg)	85.4 \pm 9.8	83.1 \pm 6.7	0.27	87.7 \pm 9.3	83.8 \pm 6.4	0.49	87.7 \pm 9.3	83.6 \pm 6.6	0.51
Height (cm)	189.8 \pm 8.3	184.8 \pm 8.2	0.61	189.8 \pm 8.3	184.8 \pm 8.2	0.61	189.8 \pm 8.3	184.8 \pm 8.2	0.61
Age (years)	20.5 \pm 2.1	22.7 \pm 3.1	0.83	20.8 \pm 2.2	22.9 \pm 3.1	0.78	20.7 \pm 2.3	22.9 \pm 3.1	0.81
Professional training history	1.6 \pm 2.2	4.0 \pm 3.3	0.58	1.9 \pm 2.3	4.2 \pm 3.3	0.58	1.8 \pm 2.3	4.2 \pm 3.3	0.59

Notes: A series of independent samples *t*-tests were performed to determine if a statistically significant difference in means existed between the injured cohort and the uninjured cohort for all demographic variables. No significant difference in means between the injured group and the uninjured group for any demographic variable at any time point ($P > 0.07$).

players in the injured cohort were compared to 18 jumps from the uninjured cohort at the end of pre-season and 102 jumps from 18 players in the uninjured cohort 3 weeks and 1 week prior to the injury occurring.

Injury definition. Injuries were clinically diagnosed by two physiotherapists who each had 8 years' experience working with elite Australian rules footballers. A muscle strain was diagnosed as a palpable soreness which was also felt through passive and active range of motion, combined with compromised power on manual muscle testing. Where ambiguity arose then diagnoses were confirmed by magnetic resonance imaging 48 h after the injury was sustained. Only muscle strains which resulted in players missing a game were included. If an injury was sustained in the week prior to a bye or in the final game of the season and it was concluded that the injury would have led to a player missing a game, if there was one the following week then that injury was also included for analysis.

Equipment. A Fitness Technology 400 series force plate capable of sampling at 600 Hz and Ballistic Measurement System software (Fitness Technology, Adelaide, Australia) were used to measure body mass and ground reaction force. The force plate was calibrated according to manufacturer instructions. Purpose built software (BioAlchemy, Adelaide, Australia) was used to calculate vertical displacement of whole body centre of mass from data extracted from the Ballistic Measurement System software. A portable stadiometer was used to measure participant height (Mentone Educational, Melbourne, Australia).

Statistical analysis. Data from the pilot analysis revealed a typical error of measurement of $0.38 \text{ kN} \cdot \text{m}^{-1}$ (4.5%).

Data were analysed absolute ($\text{kN} \cdot \text{m}^{-1}$) and relative to body mass ($\text{N} \cdot \text{m} \cdot \text{kg}^{-1}$). Contact time for each jump was also noted so that we could establish the role of ground reaction force versus contact time on vertical stiffness. All data were shown to satisfy the assumptions required for parametric statistical analysis. A series of independent samples *t*-tests were performed to determine if a statistically significant difference in means existed between the injured cohort and the uninjured cohort for all demographic variables. A 2-way ANOVA with interactions was performed on vertical stiffness data to establish whether a difference in means existed between groups or within groups across the three time points and whether an interaction existed. The same analysis was performed for contact time data. Effect size and upper and lower confidence limits for a 95%

confidence interval were also calculated for all comparisons of means for vertical stiffness between groups at each time point, and also for contact times. Finally, Pearson's correlations were performed on pooled data to explore if a relationship existed between age and vertical stiffness, and training history and vertical stiffness. Significance for all statistical tests was set at $P = 0.05$.

All statistical analyses were performed using Statistical Package for the Social Sciences software version 19.0 (IBM, New York, NY, USA).

Results

Mean body mass, height, age and professional training history were not statistically different for the injured and uninjured cohorts at any stage throughout the study (Table I). Thirteen skeletal muscle strains across the two football seasons were sustained: eight hamstrings, three quadriceps, one calf (soleus) and one hip flexor (psoas). All injuries were non-contact. Eight injuries occurred in the first half of the season (soleus, two quadriceps, five hamstrings) and five occurred in the second half of the season (psoas, one quadriceps, three hamstrings).

No statistical difference in means was found for absolute or relative vertical stiffness between or within groups. There was also no significant time*cohort interaction (Table II).

A similar observation was made for contact time (Table III).

When data were pooled and analysed to establish if a relationship exist between age and absolute vertical stiffness, and training history and absolute vertical stiffness, no relationship was seen ($r = -0.06$, $P = 0.24$ and -0.01 , $P = 0.85$, respectively). Similarly, no relationship between relative vertical stiffness and age, and relative vertical stiffness and training history was seen ($r = -0.06$, $P = 0.58$ and 0.00 , $P = 0.97$, respectively).

Discussion

In this study, we set out to determine if vertical stiffness was greater for professional Australian rules footballers who sustained a lower limb skeletal muscle strain compared to those who did not. Vertical stiffness was measured weekly; however, only data from the end of pre-season, approximately 3 weeks prior to the injury occurring and within the week prior to injury occurring were analysed. Our results revealed no significant difference in means for vertical stiffness between the injured and uninjured cohorts at any point in time. Furthermore, mean vertical stiffness did not change for either the injured cohort or the uninjured cohort across the different time points, and there was no relationship between

Table II. Participant vertical stiffness.

	End of preseason (mean \pm SD)	3 weeks prior to injury occurrence (mean \pm SD)	Within 1 week of injury occurrence (mean \pm SD)
Injured ($\text{kN} \cdot \text{m}^{-1}$)	23.4 \pm 6.5	26.8 \pm 7.2	24.0 \pm 8.4
95% Confidence Interval			
• Lower limit	19.9	22.9	19.4
• Upper limit	27.0	30.7	28.6
Uninjured ($\text{kN} \cdot \text{m}^{-1}$)	27.7 \pm 11.8	27.5 \pm 14.4	28.0 \pm 14.1
95% Confidence Interval			
• Lower limit	25.4	24.7	25.2
• Upper limit	30.0	30.3	30.7
Effect size	0.45	0.06	0.34
Injured ($\text{N} \cdot \text{m} \cdot \text{kg}^{-1}$)	279.8 \pm 86.6	306.9 \pm 79.5	272.0 \pm 80.3
95% Confidence Interval			
• Lower limit	232.7	263.7	228.3
• Upper limit	326.9	350.1	315.6
Uninjured ($\text{N} \cdot \text{m} \cdot \text{kg}^{-1}$)	333.8 \pm 140.6	328.1 \pm 166.1	332.6 \pm 163.6
95% Confidence Interval			
• Lower limit	306.5	295.9	300.8
• Upper limit	361.0	360.3	364.3
Effect size	0.46	0.16	0.47

Notes: For absolute vertical stiffness ($\text{kN} \cdot \text{m}^{-1}$) a 2-way ANOVA with a time*cohort interaction revealed no significant difference in means between groups ($P = 0.18$) or within groups ($P = 0.83$), and no interaction ($P = 0.77$). For vertical stiffness relative to body mass ($\text{N} \cdot \text{m} \cdot \text{kg}^{-1}$), a 2-way ANOVA with a time*cohort interaction revealed no significant difference in means between groups ($P = 0.08$) or within groups ($P = 0.88$), and no interaction ($P = 0.80$).

Table III. Contact times for respective vertical stiffness measures.

	End of preseason (mean \pm SD)	3 weeks prior to injury occurrence (mean \pm SD)	Within 1 week of injury occurrence (mean \pm SD)
Injured (s)	0.31 \pm 0.12	0.22 \pm 0.04	0.26 \pm 0.07
95% Confidence Interval			
• Lower limit	0.24	0.20	0.22
• Upper limit	0.37	0.25	0.30
Uninjured (s)	0.28 \pm 0.17	0.23 \pm 0.09	0.24 \pm 0.08
95% Confidence Interval			
• Lower limit	0.24	0.21	0.21
• Upper limit	0.31	0.25	0.26
Effect size	0.24	0.12	0.29

Note: A 2-way ANOVA with a time*cohort interaction revealed no significant difference in means between groups ($P = 0.41$) or within groups ($P = 0.06$), and no interaction ($P = 0.68$).

age or professional training history and vertical stiffness. These results suggest that vertical stiffness was not a predictor for muscle strain.

Results from this study contradicts those from the Australian rules football study which investigated leg stiffness and soft tissue injury, and argued that stiffness was related to soft tissue injury (Watsford et al., 2010). The reasons for these contradictions may be related to differences in methodology. The present study attempted to account for many of confounding variables identified in previous works which may affect vertical stiffness or muscle strains; we

measured vertical stiffness from double leg rebound jumping at an “athlete selected” jumping frequency as opposed to the mean of both legs from a single leg hopping task at a controlled frequency. The significance of these differences in methodology will be discussed later in the discussion. Furthermore, the rigour for defining injury in the present study was greater than previous works. However, this still does not explain why results from this study are consistent with one of the previous Australian rules football studies (Pruyn et al., 2012), but not the other (Watsford et al., 2010) despite both previous studies

adopting similar methodology. This combined with the fact that no significant difference for vertical stiffness relative to body mass was observed in the present study for the injured and uninjured group over time or between groups but the ANOVA *P*-value was “borderline” at 0.08 lends some suggestions that some relationship between stiffness and muscle strain may remain. This is further supported by descriptive statistics which showed that the injured and uninjured mean absolute and mean relative vertical stiffness at the end of preseason was just outside 95% confidence limits. However, it is worth highlighting again that in the study where stiffness was associated with hamstring injury, the mean age of the injured group was greater than the uninjured group (Watsford et al., 2010) but in the other study it was not (Pruyn et al., 2012). Furthermore, there did not appear to be a relationship between age and vertical stiffness in the present study. Therefore, in previous work hamstring injury may have occurred due to other age-related risk factors. Thus, evidence still leans towards vertical stiffness not being a risk factor for muscle strain injury.

That vertical stiffness does not appear to be associated with muscle strain injury is not surprising given that stiffness is partly dependent on functioning skeletal muscle, specifically, pre-activation strategies (Farley, Houdijk, Van Strien, & Louie, 1998; Hobara et al., 2010; Horita, Komi, Nicol, & Kyrolainen, 2002; Kuitunen, Komi, & Kyrolainen, 2002; Kuitunen, Kyrolainen, Avela, & Komi, 2007; Müller, Grimmer, & Blickhan, 2010). It has been argued that muscle strains are associated with high force eccentric contractions (Opar et al., 2012). Therefore, muscle strain injuries would be less likely when a muscle resists stretch and/or when force production at long lengths is not excessive (Opar et al., 2012) or when a muscle is “pre-activated”. That is, when pre-activation occurs stiffness is greater and excess stretch of the muscle and/or force production at the outer ranges is not present, protecting the muscle against strain injury. This notion is supported by the fact that the contact time for both cohorts was not different between each other, and no seasonal variation was observed. Suggesting that pre-activation strategies throughout this study remained the same and vertical stiffness did not remain the same simply as a function of altering ground reaction force production or contact time.

This study was limited by several factors, namely task selection and task execution. Regarding task selection, measurement of unilateral leg stiffness might be a better measure as injuries typically occur during single leg stance. However, to overcome confounding factors such as hip and spinal cord flexion, vertebral disk compression and poor

hip control, accurate measurement of unilateral leg stiffness requires the use of specialised three-dimensional kinematic measurement systems which includes the use of a biomechanical model that can precisely locate hip joint centre to determine leg length (Serpell et al., 2012). That procedure would be largely impractical for a study such as this. With respect to task execution, the present study measured stiffness from a single double leg rebound jump whereas other studies required that participants execute several hops or jumps. However, in other studies which measured vertical stiffness only the jump that elicited the greatest ground reaction force was used to calculate stiffness (Farley & González, 1996). Furthermore, we demonstrated with sufficient reliability a single rebound jump to be adequate. In addition to this, we showed that contact time was not different between or within groups throughout this study, suggesting that the high reliability was not simply a function of a change in relative contribution of ground reaction force or contact time.

The results from this study support the use of training methods to improve vertical stiffness to enhance sporting performance without increasing risk of muscle strain injury. While the optimal method for improving vertical stiffness remains unclear there is evidence to suggest that plyometric training will be effective (Spurrs, Murphy, & Watsford, 2003). However, some have argued that stiffness may lead to some overuse injuries (e.g. shin splints, lower limb stress fractures) (Butler et al., 2003). Therefore, training for vertical stiffness must be implemented in a systematic progressive overload manner.

Conclusion

Research into stiffness in the human body is becoming popular likely due to performance gains attributed to stiffness. It has been hypothesised that an injury risk trade-off may exist with increased stiffness. However, results from the present study support the theory that vertical stiffness is not a risk factor for skeletal muscle strain in Australian rules footballers. Consideration must be given to the fact that in the present study statistical significance for the similarity in mean vertical stiffness relative to body mass for the injured and uninjured cohorts was “borderline”. Nevertheless, results from the present study are consistent with previous works which showed no difference in leg stiffness throughout the course of a season for a soft tissue injured cohort and an uninjured cohort in Australian rules football. It was argued in the present study that stiffer bodies adopt pre-activation strategies which prevent skeletal muscle being stressed at long lengths, when muscle

strains are likely to occur. Conflicting results from previous works were likely due to uncontrolled confounding variables in that work. Therefore, this work supports the use of training for vertical stiffness for enhancing sporting performance without risk of increasing muscle strain injury.

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3.2. CONTEXT OF STUDY

The link between this study and that presented in chapter two was not obvious. However, this work was important because if vertical stiffness is shown to be a mitigator of ACL injury (in this body of work or elsewhere), it would be negligent to recommend implementation of training programs which focus on enhancing vertical stiffness if vertical stiffness increases risk of other traumatic non-contact injuries such as muscle strain. In addition, considered in the context that neuromuscular inhibition could remain in damaged skeletal muscle for up to 12 months (Opar et al., 2012), then the link between the study presented in this chapter and the study presented in chapter two becomes clearer. It is possible that if neuromuscular inhibition remains ongoing following muscle strain injury pre-activation and co-activation could become compromised. This is problematic if co-activation of the muscles around the knee joint acts to reduce non-contact ACL injury by constraining active laxity/enhancing dynamic knee joint stability as was discussed in chapter two. Therefore, some clarity over whether muscle strain injury is associated with vertical stiffness was important.

The role of vertical stiffness for muscle strain injury is relatively novel, and until this study only two published research papers had discussed this concept (Pruyn et al., 2012, Watsford et al., 2010). However, some notable flaws in the methodology of those two studies existed. The present study overcame a number of those methodological issues and results showed that vertical stiffness was not greater in an injured cohort when compared to an uninjured cohort. An argument may be made that this study was underpowered and therefore no definitive conclusions should be made. However, the sample size in this study was similar to that in the previous two studies which examined hamstring strain injury and vertical stiffness. Therefore, the same argument may be made about previous work. Furthermore, if one considers that there was no statistically significant difference between the injured and uninjured groups for vertical stiffness in the present study; any

difference was trivial or small at best according to effect size, it reasonable to suggest that vertical stiffness is not associated with muscle strain injury. Effect size was not reported in the previous two studies.

To this point a story has begun to emerge that vertical stiffness may not be associated with traumatic non-contact musculoskeletal injury in the football codes. However, further evidence to substantiate this claim was still required. Certainly, evidence from the study presented in this chapter suggests that muscle strain injury is not linked to vertical stiffness, the next step would be to establish if joint injuries, particularly non-contact ACL injuries, are associated with vertical stiffness. However, because the incidence of ACL injury is far lower than muscle strain injury then conducting a study similar to that described in this chapter but for ACL injury would be impractical or not achievable in the time required to complete this body of work. Therefore, the direction of research from this point onwards was to establish if co-activation of the muscles of the lower limb enhances dynamic knee joint stability. If so, and because co-activation pre-activation is related to vertical stiffness, further research was necessary to answer the question ‘is there a relationship between vertical stiffness and measures which are known to load, or which represent loading of, the ACL?’

CHAPTER 4

Thigh Muscle Co-Activation & ACL Elongation

- 4.1. Medial and Lateral Hamstrings and Quadriceps Co-Activation Affects Knee Joint Kinematics and ACL Elongation: A Pilot Study
- 4.2. Context of Study

4.1. MEDIAL AND LATERAL HAMSTRINGS AND QUADRICEPS CO-ACTIVATION AFFECTS KNEE JOINT KINEMATICS AND ACL ELONGATION

Statement confirming the authorship contribution of the PhD candidate

On behalf of all co-authors of the paper:

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I confirm that Benjamin Serpell has made the following contributions:

- Contributed to discussions on design of the study
- Collection, analysis and synthesis of literature
- Submission for ethics approval and grant funding, recruitment of subjects, and collection of data under supervision
- Analysis and interpretation of data under supervisions
- Wrote the first draft of the manuscript, and followed through to publication including proofing and final publication details of the manuscript

Professor Paul N. Smith

Signed:..... Date:.....

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Medial and lateral hamstrings and quadriceps co-activation affects knee joint kinematics and ACL elongation: a pilot study

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Abstract

Background: Many injury prevention and rehabilitation programs aim to train hamstring and quadriceps co-activation to constrain excessive anterior tibial translation and protect the anterior cruciate ligament (ACL) from injury. However, despite strong clinical belief in its efficacy, primary evidence supporting training co-activation of the hamstrings and quadriceps muscles for ACL injury prevention and rehabilitation is quite limited. Therefore, the purpose of the study presented in this paper was to determine if hamstring-quadriceps co-activation alters knee joint kinematics, and also establish if it affects ACL elongation.

Methods: A computed tomography (CT) scan from each participant's dominant leg was acquired prior to performing two step-ups under fluoroscopy: one with 'natural' hamstring-quadriceps co-activation, one with deliberate co-activation. Electromyography was used to confirm increased motor unit recruitment. The CT scan was registered to fluoroscopy for 4-D modeling, and knee joint kinematics subsequently measured. Anterior cruciate ligament attachments were mapped to the 4-D models and its length was assumed from the distance between attachments. Anterior cruciate ligament elongation was derived from the change in distance between those points as they moved relative to each other.

Results: Reduced ACL elongation as well as knee joint rotation, abduction, translation, and distraction was observed for the step up with increased co-activation. A relationship was shown to exist for change in ACL length with knee abduction ($r = 0.91$; $p \leq 0.001$), with distraction ($r = -0.70$; $p = 0.02$ for relationship with compression), and with anterior tibial translation ($r = 0.52$; $p = 0.01$). However, ACL elongation was not associated with internal rotation or medial translation. Medial hamstring-quadriceps co-activation was associated with a shorter ACL ($r = -0.71$; $p = 0.01$), and lateral hamstring-quadriceps co-activation was related to ACL elongation ($r = 0.46$; $p = 0.05$).

Conclusion: Net co-activation of the hamstrings and quadriceps muscles will likely reduce ACL elongation provided that the proportion of medial hamstring-quadriceps co-activation exceeds lateral.

Keywords: ACL, Anterior cruciate ligament, Muscle activation

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Background

Excessive tibial translation has been implicated as the cause of serious knee injuries such as anterior cruciate ligament (ACL) injury [1]. Therefore, the focus of many injury prevention and rehabilitation programs is to train co-activation of the hamstrings and quadriceps to constrain this [2, 3]. However, primary evidence supporting the role of hamstring-quadriceps co-activation for constraining tibial translation and subsequent protection of the ACL from injury is limited. This absence of evidence in spite of strong clinical belief in the efficacy of co-activation is likely due to the difficulty of measuring *in-vivo* tibial translation or ACL elongation while performing a dynamic task.

Tibial translation is typically 'quantified' by measuring passive or active knee joint laxity. Passive laxity is the 'amount' of passive motion observed in any plane or rotation prior to plateauing of a displacement tension curve [4]. Active laxity is the secondary motion observed in a plane or rotation during active movement which is not associated with the primary movement [4]. For example, some tibial translation may be observed when performing a step-up; the primary movement is knee extension and tibial translation the secondary. Passive knee joint laxity is typically measured *in-vivo* with anterior draw tests using knee arthrometers or manual tests such as Lachman's test [1]. However, measures of passive laxity do not reflect functional instability as they are unable to evaluate the effect of muscular control. Active laxity has been implied from *in-vitro* cadaveric studies [5], however these studies still fail to evaluate the true effect of muscular influences [5]. More recently an *in-vivo* study which used fluoroscopy and electromyography (EMG) attempted to explain anterior tibial translation (ATT) and the role of hamstring-quadriceps co-activation in an ACL deficient population during both open and closed kinetic chain tasks (seated knee extension and step up respectively) [2]. However, the findings from that study are not conclusive since the EMG and fluoroscopy were not conducted concurrently and ATT was assumed from measuring patella tendon angle [2].

Recent advances in image registration techniques offer the possibility of real-time *in-vivo* measurement of ATT while executing dynamic tasks whereby computed tomography (CT) images are registered with fluoroscopy (video x-ray) to allow 4-D motion analysis of bone [6–8]. This methodology provides the opportunity for measuring kinematics with previously unachievable precision while concurrently measuring hamstring and quadriceps activity. Furthermore, by using a biomechanical model to locate the ACL attachments, measurement of the distance between those attachments can provide some insight into ACL length and tension. However, such a procedure is financially costly and requires some ethical

consideration due to the radiation dosage administered. Therefore, pilot research using this technique is required to establish its 'value'.

This pilot study had two primary aims; first, to establish if co-activation of hamstring and quadriceps muscles altered knee joint motion during a step-up task, and secondly to examine if ACL elongation (maximum change in distance between the ACL attachments) was related to co-activation of the hamstring-quadriceps muscles during a step-up. We hypothesized that co-activation of the hamstrings and quadriceps would constrain the knee in terms of rotation and translation and reduce the ACL elongation when performing a step-up.

Methods

Experimental approach

This was a descriptive cohort study of healthy males from a single professional rugby union club. A CT scan of each participant's dominant knee was acquired. Participants then performed two step-ups in view of the image intensifier of a fluoroscopy machine. The first step up was performed with a low level of co-activation; that is, participants stepped up onto a box as they typically would step-up onto a box or walk up a step. Prior to the second step-up, participants were taught how step-up with deliberate co-activation of their quadriceps and hamstring muscles. Muscle activity was recorded with EMG to confirm the increase in co-activation on the second step-up. The CT scan and fluoroscopy images were image-registered to enable kinematic analysis of knee rotations and translations as well as modelling of ACL length by mapping the distances between the bony attachment sites.

A step-up task was used in order to be consistent with previously published studies [2], and because tibial translation was more likely to be seen during a closed kinetic chain task as opposed to an open chain task. Only one repetition of each step-up was performed under fluoroscopy to keep radiation dose within ethical limits.

Participants

Five males all from a single professional rugby union club aged 24.9 ± 4.1 years, height 184.8 ± 9.1 cm and weight 90.1 ± 16.3 kg (mean \pm SD). All had ACL intact knees and were free of lower limb injury.

Procedures

Each participant gave written informed consent according to institutional ethics approval for this study prior to participating. Ethical approval to conduct the research was granted by the ACT Health human research ethics committee and also the Australian National University human research ethics committee.

CT data was collected from each participant's self-reported dominant leg at 0.5 mm slice intervals on an Aquilion 16 (Toshiba, Tokyo, Japan) 150 mm above and below the knee joint. Then, participants performed a 'typical' step-up onto a 30 cm box under fluoroscopy (Axiom Artis MP, Siemens, Munich, Germany) while muscle activity was measured using an eight-channel telemetry EMG system (Mega Electronics, Kuopio, Finland) from four muscles (vastus lateralis, vastus medialis, biceps femoris long head, and semimembranosus). Fluoroscopy was performed in the sagittal plane. The step-up procedure was then repeated following training to increase hamstring-quadriceps co-activation. In order to increase co-activation tactile stimulation was applied participants' quadriceps and hamstrings prior to them performing the 'deliberate co-activation step-up' (see Fig. 1; note both persons in this figure gave written and verbal consent to have their images published). They were then instructed to contract the muscles the experimenter was touching and hold that contraction as best they could for the duration of the step-up. Visual inspection, by the experimenter, of the raw EMG trace for the step-up with deliberate co-activation confirmed increased muscle activation relative to the 'typical' step-up. Participants were given as many practice trials they wanted on the deliberate co-activation step-up prior to performing the task under fluoroscopy however no participant took longer than five minutes to learn the task.

A 4-D model of the motion of the femur and tibia was created using an algorithm which produces a digitally reconstructed radiograph from CT data and then filters

it to construct an edge-enhanced image. It was then registered to an edge-enhanced version of each fluoroscopy frame using gradient-descent based image registration as described elsewhere [6–8]. Error associated with this CT-fluoroscopy image registration technique is a standard deviation of 0.38 mm for in-plane translations and 0.42 degrees for rotations [8].

Kinematic analysis

Anterior-posterior movement (e.g. flexion and ATT) was measured on the x-axis, superior-inferior movement on the y-axis (e.g. compression/distraction), and medial-lateral movement on the z-axis (e.g. medial translation, abduction). The long axis of the femur provided the reference for rotation co-ordinates for the tibia. ACL attachments were defined according the method used by Grood and Suntay [9]; the proximal attachment was assumed to be the most superior point of the intercondylar notch of the femur and the distal attachment was assumed at the most inferior point between tibial plateau spines. ACL length was therefore taken to be the distance between these points and the change in ACL length equated to the change in distance between those points as they moved relative to each other. Maximum knee joint translations, knee joint rotations and ACL elongation were recorded as the maximum change relative to the first measurement. An example of a typical 4-D model with descriptions of how the kinematic analysis was performed can be seen in Fig. 2.

EMG collection and analysis

Care was taken to avoid crosstalk; following skin preparation, monopolar Ag-AgCl disc surface electrodes with a 2 cm radius (Ambu, Denmark) were placed at the approximate center of each muscle belly with a minimum of 1 cm separation in accordance with guidelines outlined by the Surface Electromyography for the Non-Invasive Assessment of Muscles (SENIAM) project [10]. The EMG signal was recorded by telemetry then converted from analogue to digital using an A/D converter (National Instruments NIUSM-6210, NSW, Australia) with a pre-amplifier gain of 305. A band-pass filter 12–450 Hz and a sampling rate of 1000 Hz with a common mode rejection ratio of 60 dB was applied. The signal was amplified using double differential amplifiers and subsequently recorded using Megawin software (Mega Electronics, Kuopio, Finland). It was then visually checked for artefacts before being exported to Microsoft Excel where a root mean squared (RMS) filter was applied at a non-overlapping moving window length of 20 ms. Peak RMS EMG was recorded for each muscle for both step-ups. Electrode removal did not occur between step-up conditions.

A co-activation index, which is the ratio of peak RMS EMG for antagonistic to agonistic muscle activity, was

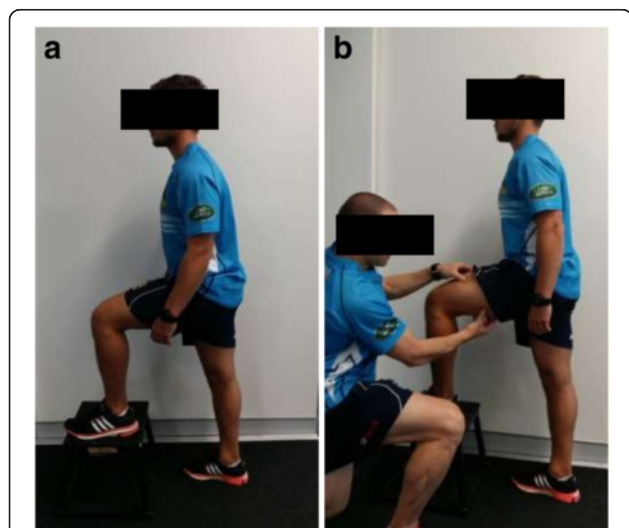
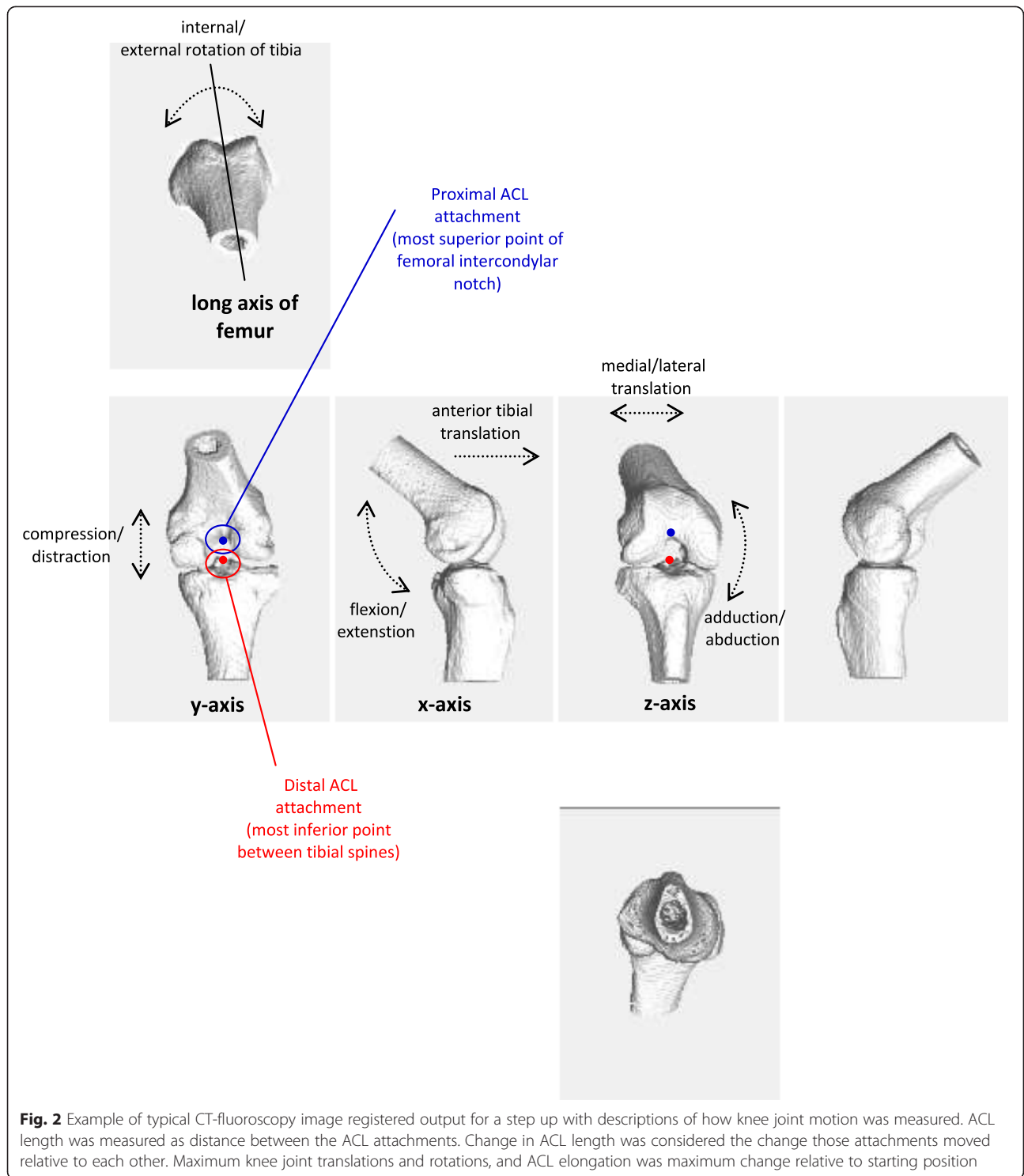


Fig. 1 **a** Starting position for a 'typical' step up. **b** Starting position for the step up with deliberate co-activation – the participant is receiving tactile feedback on how to co-activate his quadriceps and hamstrings prior to commencement of the step-up. NB: Both persons in this figure gave both verbal and written consent to have their images published



calculated for the medial hamstring and quadriceps muscles (semimembranosus-vastus medialis), the lateral hamstring and quadriceps muscles (biceps femoris-vastus lateralis), and the medial and lateral hamstring muscles (semimembranosus-biceps femoris) for both step-up conditions. Co-activation index for the medial and lateral

quadriceps was not calculated because data showed that for the step-up with deliberate co-activation muscle activity was predominantly from the hamstrings not quadriceps, therefore we were only interested in the role of the hamstring muscles in modulating ACL elongation. To remain consistent with other work, extensor muscle

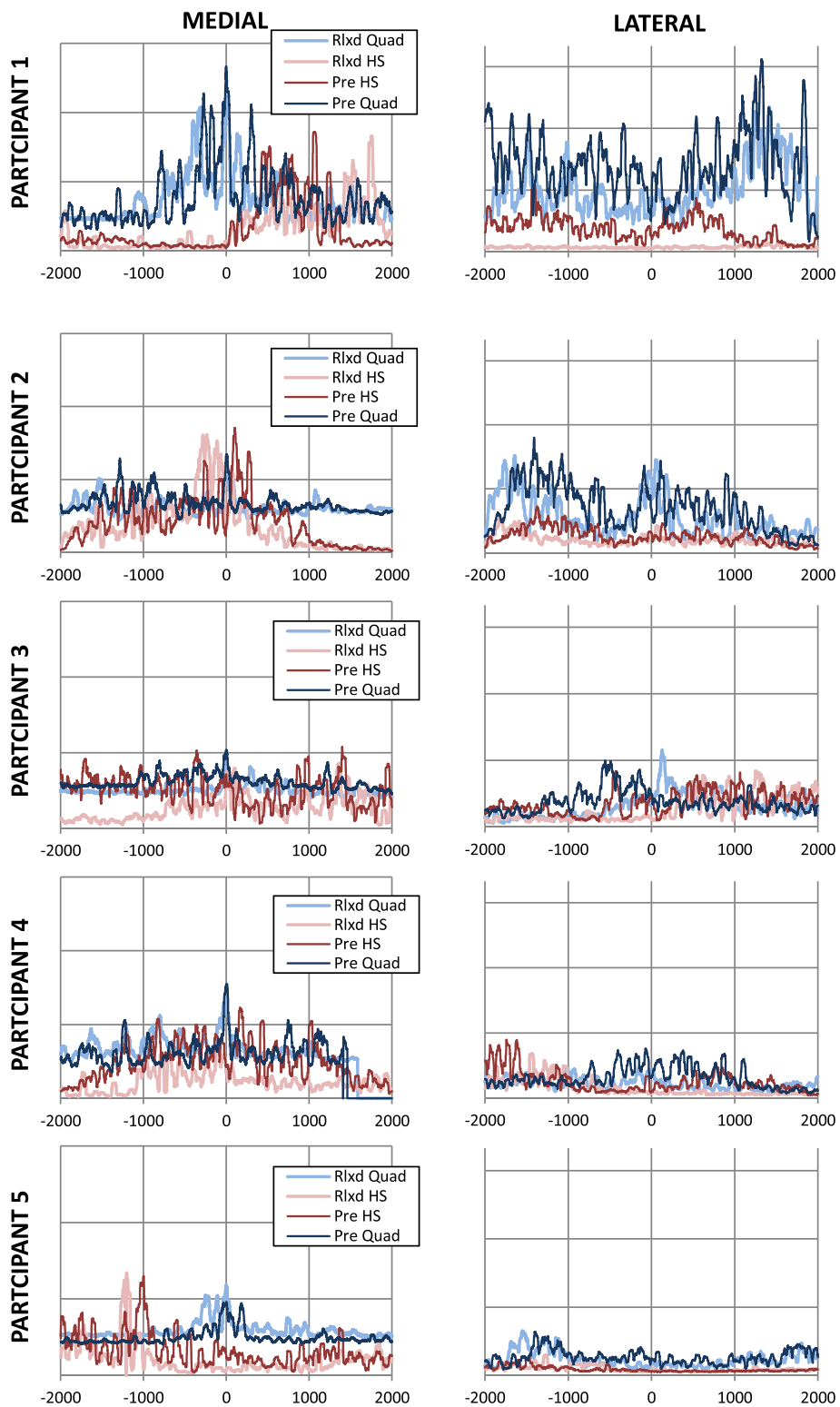


Fig. 3 EMG Traces for medial quadriceps and hamstrings (vastus medialis and semi-membranosus respectively), and lateral quadriceps and hamstrings (vastus lateralis and biceps femoris respectively). NB: Quad = quadriceps; HS = hamstring; Rlxd = relaxed and observed on first step-up; Pre = pre-activated and observed on step-up with deliberate co-activation. 0 = timing of peak vastus medialis activation for all graphs (msec)

activation was always the denominator for the hamstring-quadriceps co-activation indices [11]. For the purpose of consistency and ease of analysis, and because the denominator remained consistent for our flexor-extensor co-activation indices, the denominator was always the lateral hamstring for our medial-lateral hamstring co-activation index. Therefore, less valgus and knee rotation was expected for a smaller semimembranosus-biceps femoris co-activation index. Finally, timing of peak RMS EMG for each muscle relative to their co-activation index antagonist muscle was established for both step-up conditions to ensure the co-activation index was a true reflection of motor unit recruitment occurring at approximately the same time either side of the joint. Comparisons of co-activation between step-up conditions were based on no electrode removal.

Statistical analysis

Due to the small sample size only descriptive statistics were presented for comparison of means between step-up conditions for all EMG and kinematic data. However, data for both step-up conditions was pooled and a Pearson's correlation was used to test for relationships between ACL elongation and kinematic data, and ACL elongation with co-activation indices. Significance was set at $\alpha \leq 0.05$.

Results

The step-up with deliberate co-activation resulted in greater activation of the hamstrings, greater co-activation indices for semimembranosus-vastus medialis and biceps femoris-vastus lateralis, and a smaller co-activation index for semimembranosus-biceps (Fig. 3 and Table 1). Furthermore, the period of time between peak activation for each muscle in each co-activation index was smaller (Fig. 3 and Table 2).

Stepping-up with deliberate co-activation consistently resulted in reduced kinematic excursions and decreased elongation of the ACL during the step-up task (Table 3). Analysis of pooled data showed that as the ACL lengthened the knee abducted ($r = 0.91$; $p < 0.001$), distracted ($r = -0.70$; $p = 0.02$ for relationship between knee joint compression and ACL elongation) and anteriorly translated ($r = 0.52$; $p = 0.01$) (Table 4). However, no significant relationship was demonstrated between ACL elongation and internal rotation ($r = 0.07$; $p = 0.85$), or for ACL elongation and medial translation ($r = 0.44$; $p = 0.21$).

Stronger medial hamstring-quadriceps co-activation, demonstrated by a higher semimembranosus-vastus medialis co-activation index, resulted in a shorter ACL ($r = -0.71$; $p = 0.01$) (Fig. 4). With stronger lateral hamstring-quadriceps co-activation, demonstrated by biceps femoris-vastus lateralis co-activation index, the ACL lengthened ($r = 0.47$; $p = 0.05$). Finally, the ratio of medial to lateral hamstrings activity decreased as the

ACL lengthened ($r = -0.23$; $p = 0.03$) meaning that increased medial hamstrings activity was associated with a shorter ACL.

Discussion

The purpose of this pilot study was to investigate whether hamstring-quadriceps co-activation altered knee joint motion and limited ACL elongation during a step-up task. Although preliminary, the results of this study indicate that increasing co-activation of select hamstring and quadriceps muscles during a step-up task appears to reduce knee joint rotation, abduction, translation and distraction. Not surprisingly therefore, a lesser amount of ACL elongation was observed during the step-up with deliberate co-activation.

Change in ACL length correlated with co-activation of both lateral and medial muscle groups. However, because ACL elongation was positively correlated to the biceps femoris-vastus lateralis co-activation index and inversely correlated to the semimembranosus-vastus medialis co-activation index it is likely that medial hamstring-quadriceps co-activation, not lateral, is associated with smaller ACL elongation. This finding suggests that net co-activation of the hamstrings and quadriceps may reduce ACL elongation provided that the proportion of medial hamstring-quadriceps co-activation exceeds lateral. This hypothesis is supported by our finding that knee abduction, a movement influenced by vastus lateralis and biceps femoris [12–14], was positively correlated with ACL elongation (Table 4). These findings are meaningful when one considers that current knee reconstruction techniques involve harvesting medial hamstring tendon for ACL grafts.

The study presented in this paper is novel because this is the first time knee joint kinematics and active laxity, in the form of knee joint translations, have been measured *in-vivo* directly from bone. The methodology has a proven high degree of accuracy [8] and has the advantage of allowing concurrent EMG measurement of muscle activity. Previous studies have lacked accuracy because they have only been able to infer active laxity measurement from measures of patella tendon angle without concurrent measurements of muscle activity [2], or have had to extrapolate from *in-vitro* experiments [5].

The reductions in ACL length and ATT associated with co-activation are small but the implications are significant. Our research showed that, for a seemingly basic task such as a step-up, ATT and ACL elongation can be reduced by approximately 1.2 mm and 2.0 mm (respectively) with deliberate co-activation of select hamstring-quadriceps muscles. Previous studies have indicated that failure of the ACL is associated with relatively small changes in ACL length; an *in-vivo* study of passive laxity after ACL injury indicated that left-right

Table 1 Maximum absolute muscle activation (RMS EMG), and co-activation index for both step-up conditions (mean ± SD)

	Vastus medialis RMS EMG (mV)	Vastus lateralis RMS EMG (mV)	Semimembranosus RMS EMG (mV)	Biceps femoris RMS EMG (mV)	Co-activation index	
					Semimembranosus: Vastus Medialis activation index (mV)	Biceps Femoris: Vastus Lateralis Co-activation index (mV)
Low level co-activation step-up	280.67 ± 111.10	438.00 ± 347.63	248.84 ± 84.14	159.07 ± 82.14	0.94 ± 0.33	0.59 ± 0.47
95 % confident interval						2.28 ± 1.84
Upper limit	378.05	742.71	322.60	231.07	1.23	1.00
Lower limit	183.29	133.29	175.09	87.07	0.65	0.17
Step-up with deliberate co-activation	302.08 ± 137.74	430.48 ± 279.11	346.04 ± 143.47	311.70 ± 190.18	1.16 ± 0.14	0.88 ± 0.78
95 % confidence interval						1.50 ± 1.02
Upper limit	422.82	675.13	471.79	478.40	1.29	1.56
Lower limit	181.35	185.84	220.28	145.01	1.04	0.20

NB: RMS = Root Mean Square. Co-activation index values are hamstrings divided by quadriceps or medial hamstrings divided by lateral hamstrings

Table 2 Difference in timing of peak activation for each muscle in the co-activation indices (mean ± SD)

	Vastus Medialis – Semimembranosus (msec)	Vastus Lateralis – Biceps Femoris (msec)	Semimembranosus – Biceps Femoris (msec)
Low level co-activation step-up	0.55 ± 2.48	-2.53 ± 5.75	-1.57 ± 3.27
95% Confident Interval			
Upper Limit	3.63	4.62	2.49
Lower Limit	-2.53	-9.67	-5.63
Step-up with deliberate co-activation	-0.18 ± 8.52	-1.96 ± 11.04	-0.70 ± 5.34
95% Confidence Interval			
Upper Limit	10.39	11.74	5.93
Lower Limit	-10.76	-15.67	-7.32

NB: Values are hamstring prior to quadriceps or lateral hamstrings before medial hamstrings

3.0 mm differences in passive laxity on an anterior drawer test is indicative of ACL injury [15]. Cadaveric studies have shown a difference in ATT of approximately 7.0 mm pre and post ACL rupture [16] and primate model research showed the ACL began to fail when stretched by just 5.4 mm and this was exacerbated by the speed at which strain was applied [17]. Good comparisons between animal models and human ACL elongation patterns have been established [18]. Therefore, in view of the small length changes which appear to be required for failure of the ACL, the changes in ACL elongation detected in this study after very simple co-activation training should be considered clinically meaningful in terms of injury prevention and rehabilitation.

The potential for modulation of ACL elongation via neuromuscular training of the medial hamstring muscles is an important implication arising from of this study. There is a possibility that over activity of the lateral hamstrings and quadriceps could put the ACL at risk. This is of particular concern in the patient who has had an ACL repair using a medial hamstring graft given that muscle inhibition can persist for up to 12 months following a muscle strain injury [19]. Increased activity of the lateral hamstrings and quadriceps might ensue

following trauma to the medial tendon and could be a contributing factor to the fact that history of ACL injury is a significant risk for ACL injury [1]. This theory is also supported by some opinion which has presented a good argument for prior hamstring injury being a risk factor for ACL injury [14]. However, some caution must be exercised when considering and interpreting these findings because increased co-activation of the medial hamstrings and quadriceps muscle may be associated with osteoarthritis of the knee [20, 21], particularly when one considers that people with prior ACL injury are at increased risk of developing osteoarthritis of the knee later in life [22, 23]. Furthermore, the relationship between the muscles is not necessarily closed, it could be synergistic [24]. Synergism is defined as the distribution of force among individual muscles to produce a given task [24, 25]. The role of each muscle in a given muscle group may be modulated by a synergistic muscle [26], and it is known that the central nervous system considers synergistic muscles as a functional unit as opposed to single motor units [27].

This study has a number of limitations. Firstly the cohort studied was small but as a pilot study the results are promising and, in our view, because of the clinical

Table 3 Mean maximal change in knee joint kinematics from start position for both step-up conditions, including internal rotation, knee abduction, medial shift, joint distraction, anterior tibial translation and ACL length (mean ± SD)

	Internal rotation (degrees)	Knee abduction (degrees)	Medial translation (mm)	Joint distraction (mm)	Anterior tibial translation (mm)	Change in ACL length (mm)
Low level co-activation step-up	-11.54 ± 3.16	15.73 ± 2.25	9.78 ± 4.00	-20.55 ± 2.57	2.67 ± 1.48	15.73 ± 2.25
95 % confident interval						
Upper limit	-7.61	18.52	14.75	-17.36	4.50	18.52
Lower limit	-15.47	12.94	4.82	-23.74	0.83	12.94
Step-up with deliberate co-activation	-10.94 ± 4.26	13.92 ± 1.94	7.78 ± 3.60	-20.42 ± 2.51	1.22 ± 0.59	13.92 ± 1.94
95 % confidence interval						
Upper Limit	-5.68	16.33	12.25	-17.31	1.95	16.33
Lower Limit	-16.27	11.52	3.31	-23.54	0.50	11.52

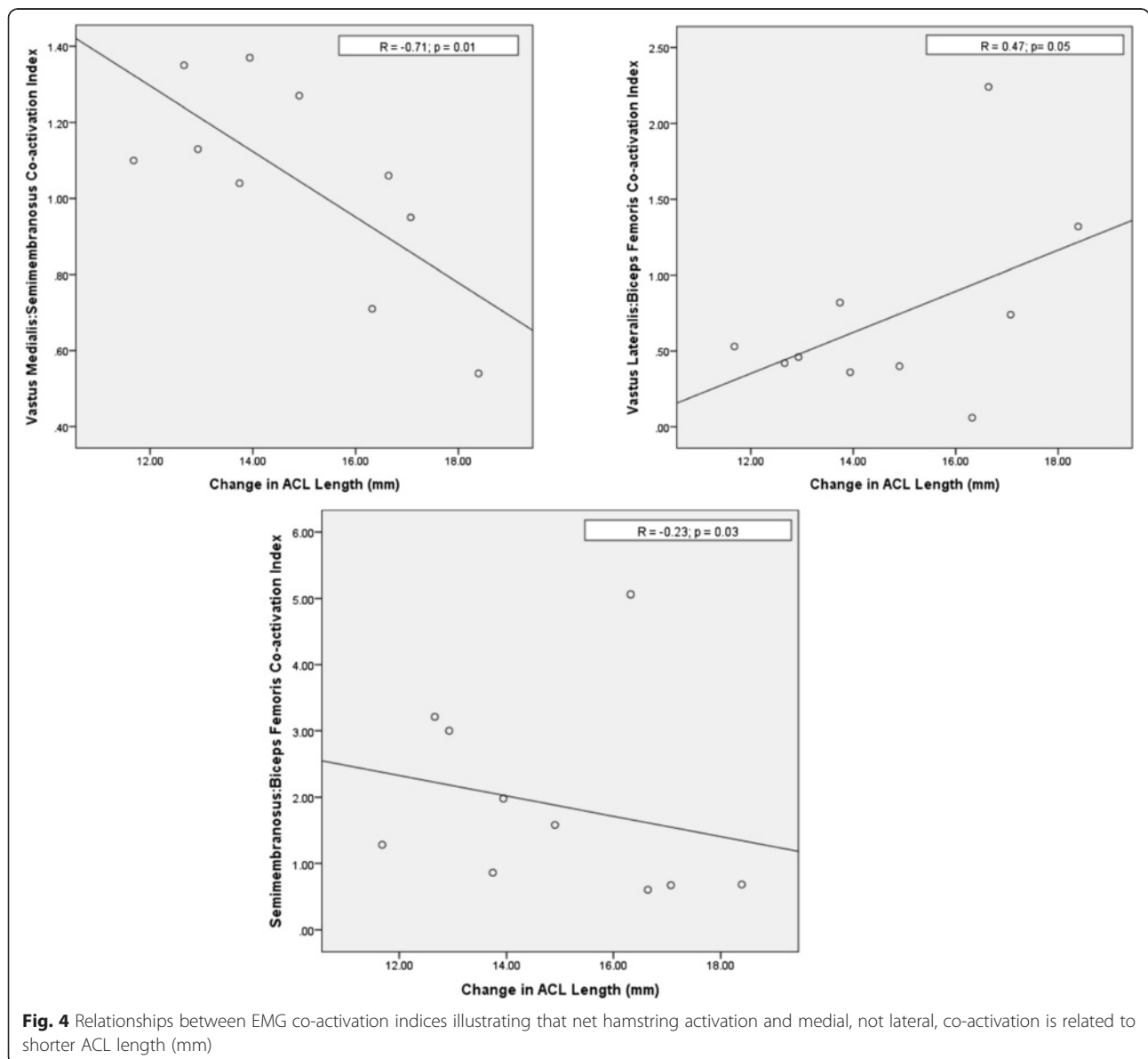
Table 4 Relationships between ACL Length and Internal Rotation, Knee Abduction, Medial Shift, Joint Compression and Anterior Tibial Translation

	Internal rotation (degrees)	Knee abduction (degrees)	Medial translation (mm)	Joint compression (mm)	Anterior tibial translation (mm)
Change in ACL Length (mm)	0.07	0.91	0.44	-0.70	0.52
p-value	0.85	≤0.001	0.21	0.02	0.01

NB: $\alpha = 0.05$

relevance of hamstring-quadriceps co-activation for ACL injury a larger study is justified despite financial and ethical considerations. Secondly, limitations surrounding EMG data collection were present. For instance, we did not quantify EMG cross-talk when measuring muscle activity. However, methods for measuring cross-talk, such as EMG signal cross-correlation, have been shown

to be ineffective in identifying cross-talk [28]. Therefore the likelihood of cross-talk measurement occurring was simply minimized by collecting EMG data according to SENIAM guidelines and applying a double differential signal amplifier which has been shown effective in minimizing cross talk [29]. In addition to this, only peak absolute RMS EMG data was presented; it could be



argued that peak RMS EMG normalized to maximum voluntary contraction should be presented as it describes better the magnitude of muscle activation. However, EMG was only used to confirm an increased level of co-activation of selected hamstring and quadriceps muscles for the step-up with deliberate co-activation. Given that the same electrodes were used on the same day on the same participants without removal between step-up conditions, and an increase in activity was seen for each muscle it can confidently be concluded that increased muscle activity was achieved for the step-up with deliberate co-activation. Furthermore, because the difference in timing of peak activation between muscles in each co-activation index reduced for the step-up with deliberate co-activation then we can state with confidence that a higher level of co-activation was achieved and not just increased activation of agonist and antagonist muscles occurring at significantly different time points. A third limitation is related to the statistical analysis for the comparison of means for EMG and kinematic data. We presented only descriptive statistics because the sample size was small. Parametric statistical analysis was not possible because the data did not satisfy the assumptions required for this type of analysis and a non-parametric analysis would likely return a type II error. A greater sample size would allow for statistical analysis for comparisons of means and is necessary to confirm our findings. Finally, timing of peak ACL elongation relative to hamstring-quadriceps co-activation, and muscle activity throughout the gait cycle was not reported. While we can confidently say that the step-up with deliberate co-activation resulted in a higher level of co-activation, we cannot be accurate about when this occurred. Unfortunately, however, it is not possible with currently available technology to synchronize EMG with the image registration technology described in this paper. Assumptions about muscle activation relative to commencement of movement have been well established elsewhere [24, 30–34] and therefore must be considered.

The results of this pilot study are promising. A future study powered for statistical examination and with some methodological improvements such as requiring participants to complete a more ecologically valid task relevant to ACL injury and enhancing EMG data collection is justified.

Conclusion

This pilot study sought to examine the clinical assumption that hamstring-quadriceps co-activation results in constraining knee from excessive ATT and other kinematic excursions therefore protecting the ACL from elongation. Our preliminary results suggest that medial hamstring-quadriceps co-activation may constrain ACL elongation, however if lateral activation exceeds medial then ACL

elongation might ensue. Although the results need confirmation with a larger study, the clinical implications are meaningful in terms of risk assessment and injury prevention.

Abbreviations

ACL: Anterior cruciate ligament; EMG: Electromyography; ATT: Anterior tibial translation; CT: Computed Tomography.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

All authors played a significant role in this project; all authors worked together to gain ethical approval and assisted with gaining funding to conduct this research. All contributed to the research design. Mark Pickering, Jennie Scarvell and Paul Smith were instrumental in developing the image registration technology used in this project. Jennie Scarvell, Nick Ball, Diana Perriman, John Warmerhoven and Ben Serpell all spent considerable time collecting data while Mark Pickering contributed significantly the processing of data. Finally, all authors read and approved the final manuscript.

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4.2. CONTEXT OF STUDY

To this point it has been argued that vertical stiffness is not linked to muscle strain injury, and this has been discussed at length in chapter three. This body of work has also shown that pre-activation and co-activation of muscles surrounding the knee joint is related to vertical stiffness. In order to establish if vertical stiffness is associated with non-contact ACL injury the next step was to determine whether co-activation of muscles surrounding the knee joint constrained active joint laxity/enhanced dynamic knee joint stability. The study presented in this chapter was a pilot study which used novel, high-tech medical imaging technology to measure knee joint motion on a basic step-up task while concurrently measuring muscle activity from muscles surrounding the knee joint. Knee joint kinematics were measured from a 4-D model which was created for each participant from image registration of CT with fluoroscopy frame by frame. ACL elongation and anterior tibial translation was measured while the step ups were being performed. It was hypothesised that with deliberate co-activation of the hamstrings and quadriceps muscles active laxity of the knee joint would be constrained (i.e. dynamic knee joint stability would be enhanced), and the ACL would not elongate.

This was just a pilot study, however results from this piece of work combined with other research (Isaac et al., 2005, MacWilliams et al., 1999) support the theory that co-activation of the hamstrings and quadriceps increases dynamic knee joint stability. Specifically, this study provided further evidence that hamstrings and quadriceps muscles operate synergistically, and net hamstrings-quadriceps co-activation constrains anterior tibial translation and subsequently ACL elongation is not seen. Given that net hamstring-quadriceps pre-activation and co-activation is also related to vertical stiffness, a story has begun to emerge from chapters two to four that vertical stiffness may not increase ACL injury risk. However, a final study to examine whether vertical stiffness and ACL injury can be associated with each other was required. Although, as identified in

chapter one, the incidence of ACL injury in sport remains high, and as stated in section 3.2 the incidence of ACL injuries which occur per year in a single professional football club is not great. Therefore, to conduct a study which examined the relationship between actual non-contact ACL injuries and vertical stiffness was not possible within the time constraints of needing to complete this thesis. Consequently, establishing a relationship between vertical stiffness and measures known to load, or which represent loading of, the ACL was necessary.

CHAPTER 5

Vertical Stiffness & ACL Elongation

5.1. Vertical Stiffness is not Related to Anterior Cruciate Ligament Elongation in Professional Rugby Players

5.2. Context of Study

5.1. VERTICAL STIFFNESS IS NOT RELATED TO ANTERIOR CRUCIATE LIGAMENT ELONGATION IN PROFESSIONAL RUGBY PLAYERS

Statement confirming the authorship contribution of the PhD candidate

On behalf of all co-authors of the paper:

SERPELL, B.G., SCARVELL, J.M., PICKERING, M.R., BALL, N.B., PERRIMAN, D., WARMENHOVEN, J. & SMITH, P.N. 2016. Vertical stiffness is not related to anterior cruciate ligament elongation in professional rugby union players. *BMJ Open Sport & Exercise Medicine*, 2.

I confirm that Benjamin Serpell has made the following contributions:

- Contributed to discussions on design of the study
- Collection, analysis and synthesis of literature
- Submission for ethics approval and grant funding, recruitment of subjects, and collection of data under supervision
- Analysis and interpretation of data under supervisions
- Wrote the first draft of the manuscript, and followed through to publication including proofing and final publication details of the manuscript

Professor Paul N. Smith

Signed:..... Date:.....

Vertical stiffness is not related to anterior cruciate ligament elongation in professional rugby union players

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ABSTRACT

Background: Novel research surrounding anterior cruciate ligament (ACL) injury is necessary because ACL injury rates have remained unchanged for several decades. An area of ACL risk mitigation which has not been well researched relates to vertical stiffness. The relationship between increased vertical stiffness and increased ground reaction force suggests that vertical stiffness may be related to ACL injury risk. However, given that increased dynamic knee joint stability has been shown to be associated with vertical stiffness, it is possible that modification of vertical stiffness could help to protect against injury. We aimed to determine whether vertical stiffness is related to measures known to load, or which represent loading of, the ACL.

Methods: This was a cross-sectional observational study of 11 professional Australian rugby players. Knee kinematics and ACL elongation were measured from a 4-dimensional model of a hopping task which simulated the change of direction manoeuvre typically observed when non-contact ACL injury occurs. The model was generated from a CT scan of the participant's knee registered frame by frame to fluoroscopy images of the hopping task. Vertical stiffness was calculated from force plate data.

Results: There was no association found between vertical stiffness and anterior tibial translation (ATT) or ACL elongation ($r=-0.05$; $p=0.89$, and $r=-0.07$; $p=0.83$, respectively). ATT was related to ACL elongation ($r=0.93$; $p=0.0001$).

Conclusions: Vertical stiffness was not associated with ACL loading in this cohort of elite rugby players but a novel method for measuring ACL elongation in vivo was found to have good construct validity.

INTRODUCTION

Anterior cruciate ligament (ACL) injury is a severe and common injury to the knee. In the USA, ~80 000 ACL injuries are reported per annum, which equates to 28 injuries per 100 000 people.¹ In Europe, the incidence of non-contact ACL injuries has been reported to be between 34 and 80 injuries per 100 000 people.² In addition, research from US collegiate sports and European professional

What are the new findings?

- Anterior tibial translation and anterior cruciate ligament elongation are strongly related.
- Vertical stiffness was not related to anterior tibial translation and anterior cruciate ligament elongation in muscular male elite rugby players.
- That vertical stiffness was not related to anterior tibial translation or anterior cruciate ligament elongation might be because vertical stiffness is associated with increased dynamic knee joint stability.

How might it impact on clinical practice in the near future?

- Vertical stiffness may be trained without fear of increased anterior cruciate ligament injury risk.
- Anterior cruciate ligament elongation may be modelled from anterior tibial translation using the equation $y=0.64x+0.24$; where y =anterior cruciate ligament elongation and x =anterior tibial translation.

football suggests that incidence of ACL injury has remained relatively unchanged over the past 30–40 years^{3 4} in spite of considerable research being undertaken in the area.⁴ These statistics are troubling given injury to the ACL leads to impairment of physical function acutely,³ and many people who sustain an ACL injury develop osteoarthritis in the knee later in life^{5–10} and other comorbidities^{11 12} making it a chronic issue also.

Unchanged ACL injury rates demand novel prevention strategies that concentrate on dynamic knee joint stability.⁴ A mechanism of ACL injury risk mitigation which has not been well studied is vertical stiffness. 'Stiffness' is a mechanical variable derived from Hooke's law in physics which can be applied to human movement. Hooke's law states that the force required to deform an object is related to a proportionality constant

(spring) and the distance that object is deformed.^{13 14} The ‘spring’ in this case reflects the viscoelastic properties of the various body tissues and the degree of stiffness is the result of the coordination and interaction of these tissues including tendons, ligaments, muscles, cartilage and bone, and their ability to resist change once force is applied.^{15–17} More specifically, vertical stiffness is a measure of whole body stiffness and is defined as the quotient of maximum ground reaction force and centre of mass displacement.^{16 18} Therefore, vertical stiffness is subject to the coordination and interaction of tendon, ligament, muscle, cartilage and bone, and the interaction and coordination of dynamic joint stability/stiffness at the spine, hip, knee and ankle joints^{16 19–25} (figure 1).

Vertical stiffness has been well researched in the area of sports performance because it has been linked to superior athletic ability,^{26–30} and because research has shown stiffness to be easily enhanced. Training programmes which focus on knowledge of performance, movement across uneven or unstable surfaces, strength training and/or plyometrics have all been shown to be effective at increasing stiffness.^{13 26 31–35} However, the study of vertical stiffness in the context of sudden or traumatic musculoskeletal injury is relatively novel. Nevertheless, it has been postulated that vertical stiffness is a risk factor for common sporting injuries due to increased vertical ground reaction force.^{13 36 37} Some research has argued a relationship between lower limb or vertical stiffness and bony injuries such as stress fracture.³⁸ However, stress fracture is an overuse injury which can be prevented by effective load monitoring.³⁹ Thus, stiffness may not be as problematic for overuse injuries, rather accelerated or exponential increases in training load and not adhering to progressive overload training principles might be. Vertical stiffness has also been implicated as a risk factor for hamstring strains in two separate research papers,^{40 41} but work by our research group which addressed notable flaws in those studies showed increased stiffness is unlikely a risk factor for muscle strain injury.⁴² To the authors’ knowledge, no evidence exists to suggest increased vertical stiffness is a risk factor for non-contact connective tissue injury such as ACL strains.

Given that vertical stiffness is partly regulated by joint stiffness, or dynamic joint stability, modifying vertical

stiffness may assist in preventing ACL injury particularly non-contact ACL injury. This concept is supported by other work previously undertaken by our research group which showed that greater vertical stiffness is related to increased hamstring and quadriceps preactivation and co-activation,¹⁵ and that increased co-activation of the hamstrings and quadriceps reduces ACL elongation and anterior tibial translation (ATT).⁴³ Therefore, when vertical stiffness is high knee joint stiffness/dynamic knee joint stability must also be high.^{16 25}

It is possible that vertical stiffness as a risk factor for ACL injury has not yet been investigated because measuring ACL stress in vivo has been very difficult and is either invasive or derived from indirect or inaccurate measures. In fact it is only that recent advances in image registration technology, whereby CT images are registered with fluoroscopy (video X-ray) to allow four-dimensional (4D) motion analysis of bone that non-invasive measures become more accurate. This technology, developed by our group, provides the opportunity for measuring kinematics with previously unachievable precision and, for the first time, enables in vivo measurement of ATT.^{44–46} Excessive ATT has been implicated in serious knee injuries such as ACL injury.⁴ Furthermore, by using a biomechanical model with the image registration technology to locate the ACL attachments, measurement of the distance between those attachments can provide some insight into change in ACL length, or ACL elongation. This is important because the ACL will fail when elongation, or consequent strain, is too great.^{43 47}

The aim of this study was to determine if vertical stiffness during a multidirectional hopping task was related to measures which represent loading of the ACL, specifically ACL elongation and ATT. ACL elongation and ATT were measured in vivo using image registration technology with known high precision.^{45 46} A secondary aim was to evaluate the relationship between ACL elongation and ATT.

MATERIALS AND METHODS

Experimental approach

This was a cross-sectional observational study of professional male rugby union players. Ethical approval was

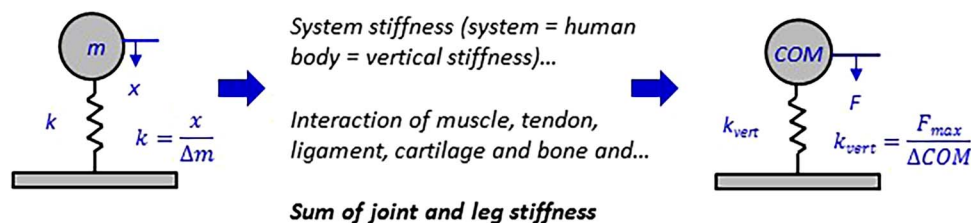


Figure 1 Stiffness (k) is equal to force (x) divided by change in length (Δm). Vertical stiffness (k_{vert}) is a measure of system/whole body stiffness and is equal to maximum vertical ground reaction force (F_{max}) divided by change in whole body centre of mass (ΔCOM). Vertical stiffness therefore is regulated by the function and interaction/coordination of individual anatomical structures and stiffness at joints.

given by the University Human Research Ethics Committee. Written informed consent was obtained from all participants prior to their involvement.

Participants

Participants were conveniently sampled and 11 men were subsequently recruited to this study aged 26.1 ± 4.7 years, height 180.5 ± 11.3 cm and mass 85.4 ± 16.5 kg (mean \pm SD). Each participant was screened by the rugby club's doctor and physiotherapist and deemed to be free of lower limb injury in the 24 months prior to data collection, and all had ACL intact knees.

Procedures

CT data were collected from participants' self-reported dominant leg at 0.5 mm slice intervals on an Aquilion 16 (Toshiba, Tokyo, Japan) 150 mm above and below the knee joint line prior to them performing a bare-foot power-cut hop under fluoroscopy (Axiom Artis MP, Siemens, Munich, Germany). The power-cut hop was a single-leg exercise requiring a 45° jump in the ipsilateral direction onto a designated point on a force platform (Kistler Group, Winterthur, Switzerland), landing on the ipsilateral leg and jumping off as quick as possible at an angle of 90° to land on the same leg at a set distance of 1.0 m (figure 2). A power-cut hop was required as opposed to a running change of direction manoeuvre due to spatial constraints and because this change of direction task best replicated the change of direction manoeuvre typically observed when non-contact ACL injury occurs.⁴ CT data were image registered to fluoroscopy and knee joint kinematics and ACL elongation were subsequently measured. Vertical stiffness was calculated

from force platform data for each hop and analysed with the image registration output.

Kinematic analysis

In summary, a 4D model of the motion of femur and tibia was constructed from CT and fluoroscopy data from the power-cut hop test using a technique whereby an algorithm which produces a digitally reconstructed radiograph from CT data and filters it to construct an edge-enhanced image is registered to edge-enhanced fluoroscopy using gradient descent-based image registration. This method has been well described elsewhere.^{45 46} Still image examples of image registered output can be seen in figure 3.⁴³ Knee joint kinematics were subsequently measured in 6-degrees-of-freedom; anterior–posterior movement (eg, flexion and ATT) was measured on the x-axis, superior–inferior movement on the y-axis (eg, compression/distraction) and medial–lateral movement on the z-axis (eg, medial translation, abduction). The long axis of the femur provided the reference for rotation coordinates for the tibia. The error associated with this CT fluoroscopy image registration technique is an SD of 0.38 mm for in-plane translations and 0.42° for rotation.⁴⁶

ACL attachments were mapped to the image-registered output and were defined according to the method used by Grood and Suntay;⁴⁸ the proximal attachment at the most superior point of the intercondylar notch of the femur and the distal attachment was assumed the most inferior point between tibial plateau spines. ACL length was considered the distance between those points. Thus, ACL elongation was the change in, or the difference between minimum and maximum, ACL length.

Vertical stiffness measurement

Vertical stiffness was calculated according to the protocol of Cavagna⁴⁹ and was therefore considered to be the quotient of maximum vertical ground reaction force and whole body centre of mass displacement. The force platform was interfaced with a personal computer and Bioware software (Kistler Group, Winterthur, Switzerland) was used to record vertical ground reaction force at 1000 Hz for each of the power-cut hops. A 10 Hz high-pass dual-pass Butterworth filter was applied to the raw force plate data. Data were exported from Bioware to purpose built software (BioAlchemy, Adelaide, Australia) for the calculation of vertical stiffness. To calculate the centre of mass displacement the cumulative sum of the vertical ground reaction force (N/s) was integrated, and then point-by-point integration of the previously integrated force was performed. Reliability of this method has been reported elsewhere with typical error of measurement (TEM) of 4.3%. TEM for contact time for the power-cut hopping task was also reported as 1.7%.¹⁵

Statistical analysis

ATT, change in ACL length and vertical stiffness data are presented as mean \pm SD. Prior to testing for correlations

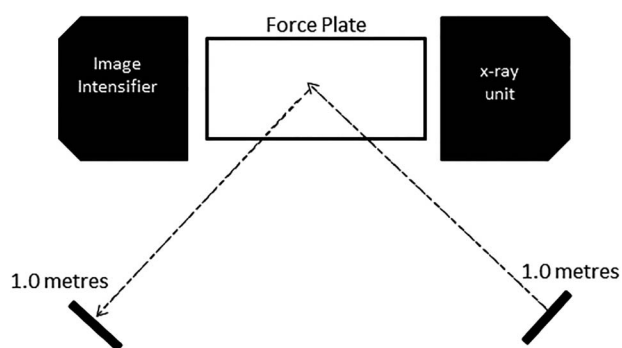


Figure 2 Power-cut hop test. For example, in the above diagram it shows that for a right leg power-cut hop participants would jump off their right leg from the 1.0 m mark on the right of the diagram to land on the centre of the force plate on their right leg, then jump off the force plate as quick as possible before landing on their right leg past the 1.0 m mark on the left of the diagram. The power-cut hop was performed under fluoroscopy to enable construction of a 4D model of the motion of the femur and tibia for knee joint kinematic analysis; hence the centre of the force platform was located in the C-arm of the image intensifier of a fluoroscopy machine. 4D, four-dimensional.

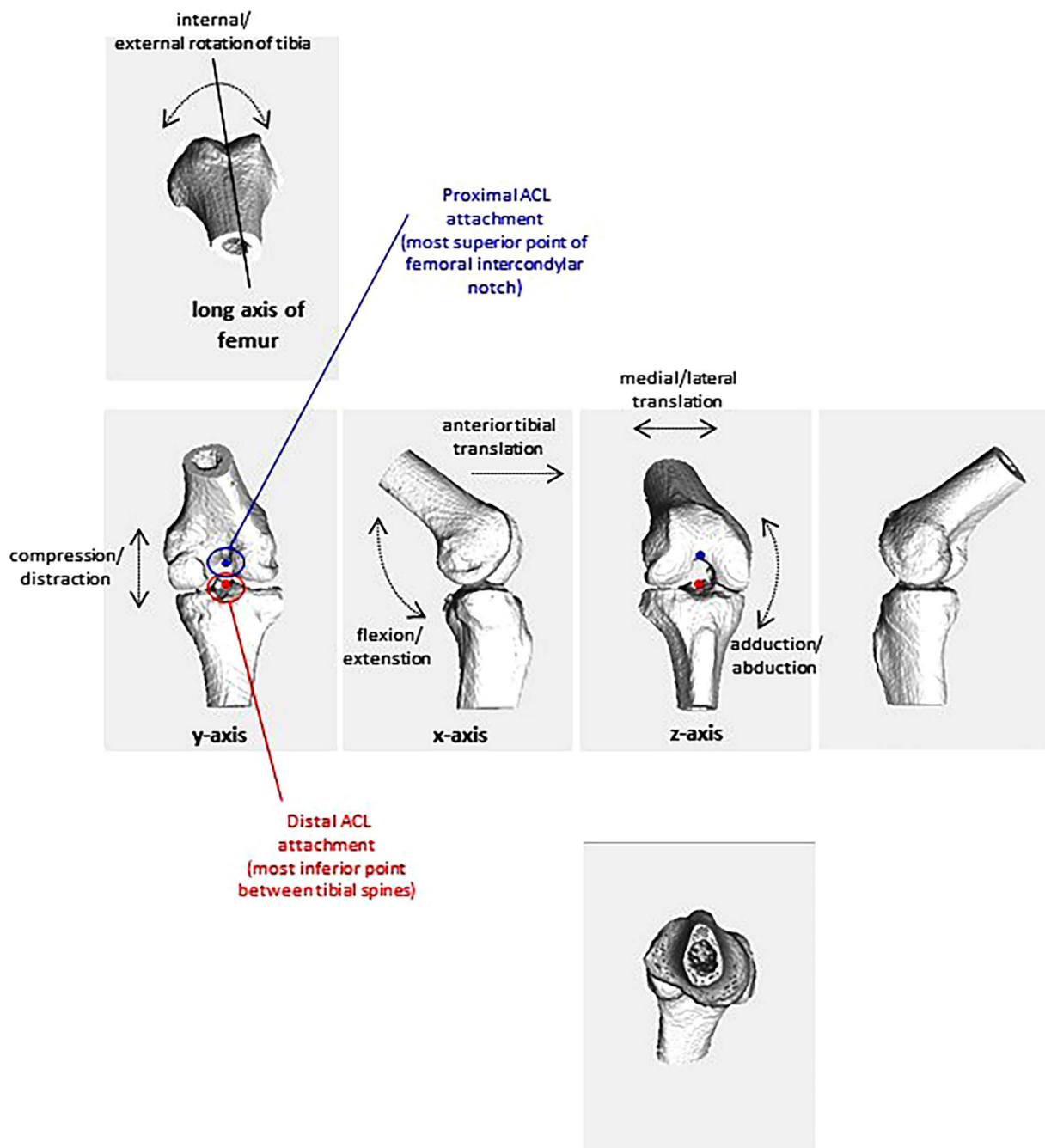


Figure 3 Example of typical CT fluoroscopy image registered output for a step up with descriptions of how the knee joint motion was measured. ACL length was measured as distance the ACL attachments moved relative to each other. ACL, anterior cruciate ligament.

data for ATT, change in ACL length and vertical stiffness were tested for normality with a Shapiro-Wilks test and a Levene's test for homogeneity of variance. Pearson's correlation coefficient was then used to test for the strength of relationship between vertical stiffness and both ATT and change in ACL length. Pearson's correlation coefficient was also used to test the relationship between ATT and change in ACL length. A scatterplot for change in ACL length versus ATT was generated and a linear regression analysis was performed to describe the relationship between ACL elongation and ATT. All statistical

analyses were performed using the Statistical Package for Social Sciences (SPSS) software V.19 (IBM).

RESULTS

Vertical stiffness (kN/m) for the power-cut hopping task was 68.31 ± 39.47 . Knee kinematics derived from the model showed that ATT was 0.78 ± 0.42 mm and the change in ACL length was 0.84 ± 0.61 mm.

Neither ATT nor ACL elongation appeared to be related to vertical stiffness as demonstrated by a non-

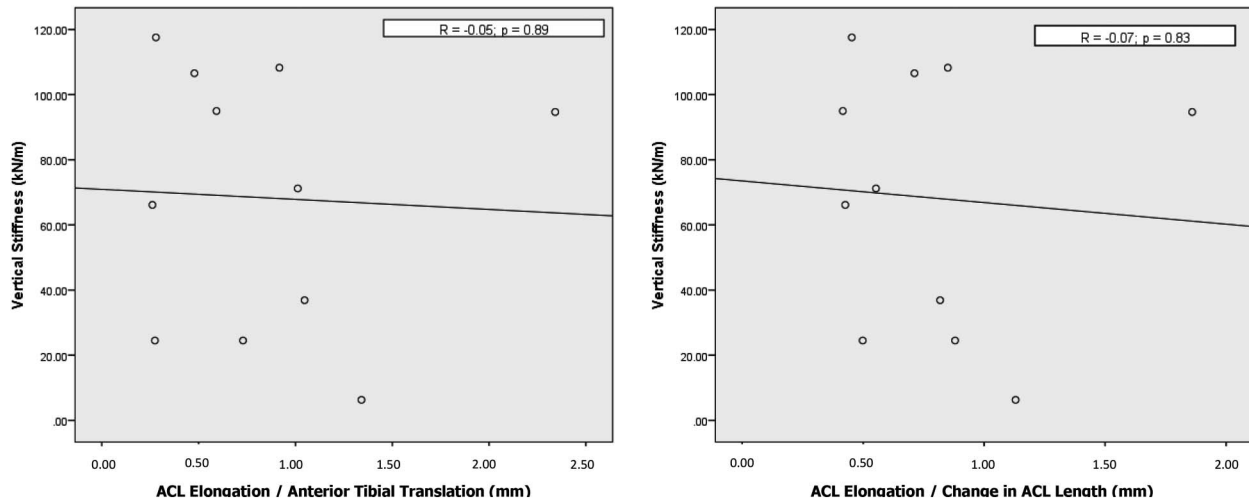


Figure 4 Relationships of vertical stiffness with anterior tibial translation and change in ACL length illustrating no relationship existed. ACL, anterior cruciate ligament.

significant and non-substantial inverse relationship between vertical stiffness and ATT ($r=-0.05$; $p=0.89$), and between vertical stiffness and change in ACL length ($r=-0.07$; $p=0.83$; [figure 4](#)).

ATT and ACL elongation were strongly related as demonstrated by a strong and significant relationship between ATT and change in ACL length ($r=0.93$; $p=0.0001$; [figure 5](#)). Furthermore, the linear regression analysis revealed that the relationship between ACL elongation and ATT is represented by the equation:

$$y = 0.64x + 0.24$$

where y is the ACL elongation/change in ACL length, and x is the ATT ([figure 5](#)) which explained 87% variation in the data.

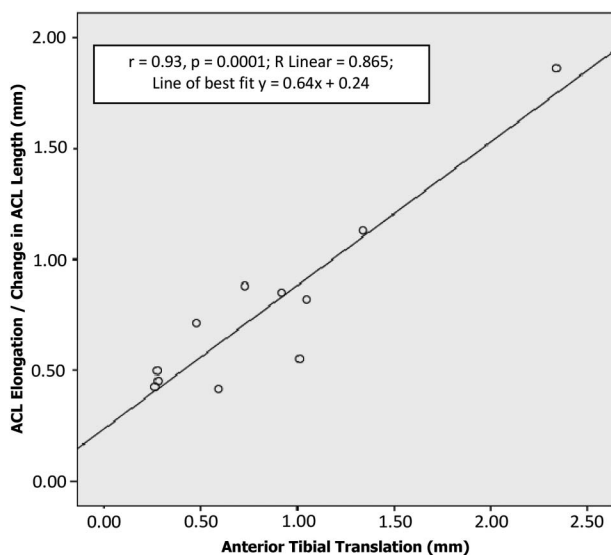


Figure 5 The relationship between ACL elongation (change in ACL length) and ATT. ACL elongation= $(0.64 \times \text{ATT}) + 0.24$. ACL, anterior cruciate ligament; ATT, anterior tibial translation.

DISCUSSION

The main finding of this study was that vertical stiffness was not related to measures which represent ACL loading; specifically ACL elongation and ATT. Furthermore, the novel in vivo method used in this study to measure ACL elongation was shown to have good construct validity as evidenced by a strong relationship between change in ACL length and ATT.

The aim of this study was to examine the theory that, because increased vertical stiffness is related to increased vertical ground reaction force, it is also related to ACL loading.^{13 36 37} Participants were tested using a multidirectional hopping task which simulated the change of direction manoeuvre typically seen when non-contact ACL injuries occur. Vertical stiffness was calculated from force plate measurements and ATT and ACL elongation were measured in vivo using a novel image registration method which has been previously validated for measurement of knee kinematics.^{45 46 48 49} No relationship between vertical stiffness and ATT or ACL elongation was observed. Therefore, our results do not support others' hypothesis that increased vertical stiffness may be related to increased ACL injury risk because of increased vertical ground reaction force. There are two possible explanations for this result; first and most obviously, vertical stiffness does not contribute to ACL injury risk. Second, our methods were insufficient to detect an association which was actually present.

This study is novel from the perspective that it is the first to measure ATT, ACL elongation and vertical stiffness in vivo while executing a task which simulates the change of direction manoeuvre observed when ACL injury typically occurs. To the best of the knowledge of the authors of the present study, a previous study which has discussed a link between vertical stiffness and ACL injury has only postulated this relationship theoretically.^{13 25 36 50 51} In a previous electromyography study, we suggested that vertical stiffness on similar hopping

tasks was likely to be related to increased preactivation of the hamstring and quadriceps muscles, particularly when they are co-activated.¹⁵ Furthermore, in another study by our group, and studies by others, have shown that increased co-activation of the hamstring and quadriceps muscles reduced ATT^{43 52 53} suggesting that dynamic factors were responsible for increased dynamic knee joint stability. Therefore, while increased vertical ground reaction force might occur with increased vertical stiffness, results from this study, and those of others, suggest that the ACL may not be subject to additional loading secondary to high levels of vertical stiffness because of the primary role played by dynamic knee joint stability. It should be acknowledged, however, that under conditions where extreme anterior–posterior, medial–lateral and/or rotational perturbations are present the magnitude of the vertical ground reaction force may not need to be as great for failure of the ACL to occur. This reasoning is consistent with a previous animal study which showed that ACL stretch and failure was exacerbated by extreme perturbations.⁴⁷

Another possible reason for not finding an association between vertical stiffness and ACL elongation is that our methodology was not sufficiently optimised. The ACL attachment sites used to model ACL elongation was based on those described by Grood and Suntay.⁴⁸ According to this method, the proximal ACL attachment is to the most superior point of the intercondylar notch of the femur and the distal attachment is to the most inferior point between tibial plateau spines.⁴⁸ However, recent anatomic studies have shown that the proximal attachment is on the medial wall of the lateral femoral condyle⁵⁴ and the distal attachment attaches slightly anteriorly to the peak of the medial spine on the tibial plateau.⁵⁵ These potential anatomical discrepancies may have affected measurement accuracy⁵⁶ and led to our failure to find a relationship between vertical stiffness and ACL elongation. Nevertheless, in this study, ATT was strongly related to ACL elongation indicating good construct validity for this novel method of measuring ACL length.

There were several limitations to this study. First, we did not measure muscle activity concurrently. It would be beneficial to establish further the relationship between thigh muscle activation and any synergistic relationship that may exist between the different quadriceps and hamstring muscles and how they affect ACL elongation on a task similar to that used in the present study. Combined with kinematic data, this may also enable modelling of moments which may provide further insight into the relative force production, and synergistic force production, between muscles surrounding the knee joint. However, with the image registration technology used in this, it is not possible to establish muscle activity relative to ACL elongation. Muscle activity on this task and similar other tasks has been established elsewhere¹⁵ and this must be considered currently. Second, although ATT and ACL elongation were strongly associated they are different measures and therefore can

only be surrogates for each other. This is hardly surprising, given that ATT occurs in one plane whereas the ACL length, although primarily modified by anteroposterior stress, is also influenced by mediolateral, rotational and decompressive stresses. Therefore, the relationship found in this study lends support to this novel method of measuring ACL elongation.

CONCLUSION

This study aimed to determine whether increased vertical stiffness is related to ACL loading. We used a novel in vivo method to measure ACL elongation in elite rugby players on a task which stressed the ACL similarly to that which would be observed when ACL injury occurs. This novel method was found to have good construct validity, and our results showed that ACL elongation was not related to vertical stiffness in this cohort of elite rugby players. This study argued that while peak vertical ground reaction force is likely to increase with increased vertical stiffness, it is unlikely to overload the ACL because it is relatively protected due to increased dynamic knee joint stability which is related to increased vertical stiffness. It is possible that the direction of force is more problematic to the ACL. Future studies should also aim to incorporate electromyography and to test more challenging activities where force direction is less predictable.

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Contributors MRP, JMS and PNS were instrumental in developing the image registration technology used in this project. JMS, NBB, DP, JW and BGS all spent considerable time collecting data while MRP contributed significantly to the processing of data. Finally, all authors read and approved the final manuscript.

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Competing interests None declared.

Ethics approval Approved by the ACT Health Human Research Ethics Committee (Protocol ETH.4.11.072), and the Australian National University Human Research Ethics Committee (Protocol 2011/396).

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5.1. CONTEXT OF STUDY

The final study in this body of research was an exploration of the existence of a relationship for vertical stiffness with measures that load, or which represent loading of, the ACL. Despite studies earlier in this thesis showing that vertical stiffness was related to pre-activation and co-activation strategies of the hamstring and quadriceps muscles, and that net co-activation of the hamstring and quadriceps reduces anterior tibial translation and ACL elongation, it did not necessarily follow that vertical stiffness is unrelated to ACL elongation. In this study participants executed a hopping task similar to that described in chapter two which was designed to simulate the change of direction manoeuvre typically observed when non-contact ACL injury occurs. Vertical stiffness was measured during this hopping task and knee joint motion (including anterior tibial translation) and ACL elongation was measured concurrently using kinematic measurement technology described in chapter four.

In the study presented in this chapter no relationship for vertical stiffness with anterior tibial translation or ACL elongation was observed. However, a very strong positive relationship was seen for anterior tibial translation with ACL elongation. This was not surprising because a considerable amount of ACL elongation is bourn from anterior tibial translation (Butler et al., 1980, Noyes et al., 1974a), and because a similar observation was made in the previous study. Therefore, good construct validity for how ACL elongation was measured in this study and the one previous may be argued; and because of this of this the argument that vertical stiffness is not related to measures known to load, or which represent loading of, the ACL is sound.

An unexpected, but exciting, outcome from this study was that a model of ACL elongation from measurement of ACL elongation was developed. This model offers scope to measure the effect of injury mitigation strategies or rehabilitation outcomes with respect to the ACL.

CHAPTER 6

Conclusion

7.1. Synthesis of Findings

- 7.1.1. Muscle pre-activation and co-activation increased vertical stiffness
- 7.1.2. Vertical stiffness was not greater in footballers who sustained a muscle strain injury
- 7.1.3. Muscle co-activation did not appear to increase loading of the ACL
- 7.1.4. Vertical stiffness did not increase loading of the ACL

7.2. Context of Findings

7.3. Practical Applications or Clinical Implications

7.4. Limitations

7.5. Directions for Future Research

7.6. Concluding Remarks

6.1. SYNTHESIS OF FINDINGS

In the introduction of this thesis it was argued that the incidence of ACL injury remains high and because of this, and the short and long term consequences associated with ACL injury, novel methods for preventing ACL injury demand investigation. A published literature review was presented (section 1.2.) which described the epidemiology of ACL injury, the common mechanisms and risk factors for ACL injury, and some direction for future research. It concluded that dynamic knee joint stability, in particular, was worthy of ongoing investigation. Section 1.3. noted that knee joint stability is typically only measured in a passive state but this was probably because of the difficulty of measuring dynamic knee joint stability *in-vivo*. However, a novel method for measuring knee joint motion that has a high level of known precision and reliability, which would enable measurement of dynamic knee joint stability *in-vivo*, was presented. This method involves image registration of CT and fluoroscopy. Section 1.3. also suggested stiffness as an area worthy of investigation because a high level of stiffness requires dynamic knee joint stability. It was subsequently argued that if the CT-fluoroscopy image registration technology was used to measure dynamic knee joint stability while stiffness was concurrently measured then some insight into the role of stiffness for reducing ACL injury rates could be gained. However, it was argued that more should be known about stiffness and hence a published literature review describing how it is best measured was presented in section 1.4. In section 1.5 it was suggested that stiffness, vertical stiffness in particular, may be of interest for ACL injury risk mitigation because it is easily measurable with just a force plate (see section 1.4.), is trainable (see section 1.3.), and it may enhance dynamic knee joint stability. However, it was also noted that some confusion exists about the role of vertical stiffness as an injury risk mitigator, with some believing that stiffness in fact increases injury risk. Therefore, the main aim of the thesis was to explore the role of vertical stiffness on some traumatic non-contact lower limb injuries. A focus was placed on non-contact ACL injury.

Prior to this research being conducted, the role of vertical stiffness as a risk factor for common traumatic non-contact injury was poorly understood. On one hand, vertical ground reaction force increases with increasing vertical stiffness and some believe that as a consequence injury risk increases (Bradshaw and Hume, 2012, Butler et al., 2003, Lorimer and Hume, 2016). Conversely, given that stiffness is dependent on the co-ordination and interaction of muscle, tendon, ligament, cartilage and bone, and subsequent joint stiffness, vertical stiffness serves to constrain joint motion and excessive stretch of soft tissue structures therefore vertical stiffness could be a mitigator of injury. Hence, this body of research set out to establish whether vertical stiffness could be associated with some common traumatic non-contact injuries. The research presented in this thesis focused primarily on trying to establish an association between vertical stiffness and non-contact ACL injury, however vertical stiffness and skeletal muscle strain was also briefly investigated mainly because vertical stiffness is modulated, in part, by functioning skeletal muscle. As will be discussed in the ensuing chapter, outcomes of the studies presented in this thesis suggest that vertical stiffness is not linked to common traumatic non-contact injury.

This thesis is comprised of four distinct studies, each of which was presented in separate chapters (chapters 2-5), and each study informed subsequent studies. Section 6.1.1. through to section 6.1.4. summarises the main outcomes of each of these studies and ties together the main concepts which, combined, tell a story of there being no evidence of an association between vertical stiffness and common traumatic non-contact injury. The reader should refer to each of the chapters for a more detailed discussion on each of the studies.

6.1.1. MUSCLE PRE-ACTIVATION AND CO-ACTIVATION INCREASED VERTICAL STIFFNESS

The study presented in chapter two described the relationship between pre-activation strategies of the lower limb muscles and vertical stiffness on a multidirectional hopping task. This study revealed that pre-activation of lower limb muscles was typically positively related to vertical stiffness on a multidirectional hopping task which simulated the change of direction manoeuvre typically observed when non-contact ACL injury occurs. Furthermore, the strength of the relationship typically increased when pre-activation was normalised to maximum voluntary contraction. In this study, a co-activation index was the mean normalised activation of the hamstrings divided by the mean normalised activation of their respective antagonist quadriceps muscle during the pre-activation period. Typically, a positive relationship was observed between co-activation index of the muscles around the knee joint and vertical stiffness. The strength of the relationship between lateral hamstring-quadriceps co-activation index (biceps femoris-vastus lateralis co-activation index) and vertical stiffness was greater than for the medial (semimembranosus-vastus medialis co-activation index). Finally, results point to there being a small but relevant relationship for vertical stiffness with peak activation and timing of peak activation of the medial hamstring muscles.

In the discussion section of this paper it was noted that the stronger relationship between lateral hamstring-quadriceps pre-activation co-activation index and vertical stiffness was not unexpected. It was not unexpected because previous work has shown that the lateral hamstring and quadriceps muscles typically ‘work harder’ for change of direction manoeuvres similar to the task required of participants in the present study (Besier et al., 2003). It should be acknowledged that those muscles have been implicated for knee joint external rotation and valgus/knee abduction; the knee joint kinematics that are observed often when ACL injury occurs (Besier et al., 2003).

However, as also noted in the discussion section of the paper, activation does not equal force production. Force production is dependent on a range of factors including muscle attachment sites, cross sectional area, fascicle length etc. This explains why the relationship between vertical stiffness and pre-activation of each muscle in isolation was more likely positive and typically stronger when normalised to maximum voluntary contraction. Furthermore, it was noted that muscles rarely work in isolation rather they work synergistically (Neptune et al., 2001), and the role of each muscle in each muscle group for any given task might be dependent on how other muscles in that muscle group operate (Zatsiorsky and Prilutsky, 2012, De Luca and Erim, 2002). In this study there was still a positive relationship between the medial hamstring and quadriceps muscles, and vertical stiffness for the multidirectional hopping task. A positive relationship between vertical stiffness and the co-activation index of the medial and lateral hamstring and quadriceps muscles was present, albeit weak for the medial co-activation index; and the magnitude of peak activation of the hamstrings, and the timing of peak activation of the medial hamstring, was positively related to vertical stiffness in this sample. These data suggest muscle synergism. This notion is consistent with other work which has described activation patterns of the knee flexors and extensors on various planned and unplanned running and change of direction tasks and argued that the central nervous system preferentially adopts generalised activation patterns to protect the ACL as opposed to selective patterns (Besier et al., 2003).

The study presented in chapter two clearly showed that pre-activation co-activation strategies of muscles of the lower limbs are adopted for multidirectional tasks which simulate the change of direction manoeuvre typically observed when non-contact ACL injury occurs. These pre-activation strategies are positively related to vertical stiffness. Based on the results from this study it could be hypothesised that when muscle activation on either side of the knee joint is balanced little knee joint motion will be observed and as a function vertical stiffness may be greater.

However, no link between vertical stiffness and non-contact ACL injury could be made from this study.

6.1.2. VERTICAL STIFFNESS WAS NOT GREATER IN FOOTBALLERS WHO SUSTAINED A MUSCLE STRAIN INJURY

The study presented in chapter three sought to determine a link between vertical stiffness and lower limb skeletal muscle strains. This study was important for several reasons; firstly, because if the body of research presented in this thesis showed that vertical stiffness was not linked to non-contact ACL injury then recommendations to train vertical stiffness may be made. However, if vertical stiffness was related to other non-contact traumatic injuries such as muscle strain then recommending implementation of training programs which focus on enhancing vertical stiffness would be negligent. Secondly, given neuromuscular inhibition could remain in damaged skeletal muscle for up to 12 months (Opar et al., 2012), and in the previous study it was shown that pre-activation was important for vertical stiffness, then it is possible that muscle strain injury can negatively affect vertical stiffness which could prove problematic if vertical stiffness acts to reduce joint injuries such as non-contact ACL injury.

The role of vertical stiffness in muscle strain injury is relatively novel, and until this study only two published research papers had discussed this concept (Pruyn et al., 2012, Watsford et al., 2010). However, as was pointed out in the introduction of the paper presented in chapter three, there were some quite notable flaws in the methodology adopted in the two previous studies. The most obvious flaw being in Watsford et al. (2010) which showed a link between vertical stiffness and hamstring muscle strain; the mean age of the injured cohort was significantly greater than the mean age of the uninjured cohort. Age is one of the greatest predictors of muscle strain injury, with

older people displaying increased incidence of muscle strain injury (Opar et al., 2012). The second major flaw present in both previous studies pertained to the definition of muscle strain injury that was used. The definition of ‘injury’ in those studies was broad, therefore it is possible that many of the injuries reported may not have actually been ‘injuries’ per se (Bailey et al., 2010), as the reported complaint may not have even been related to any local tissue damage.

For the study in chapter three, data was collected across two football seasons for vertical stiffness and muscle strain injury. Whether vertical stiffness was greater in the seven days prior to injury occurring, three weeks prior to injury occurring or at the end of pre-season in the injured cohort compared to the uninjured cohort was investigated. Furthermore, a tighter definition of muscle strain injury was adopted - the players must have been sore on palpation at the suspected site of injury, sore on stretch and a loss of power must have been observed. The clinical analysis was performed by several experienced sports therapists, and where ambiguity arose then MRI 48 hours post injury was used for confirmation. Finally, the player must have missed at least one game due to the complaint in accordance with previously published definitions of injury (Orchard and Seward, 2002). Results from this study showed that vertical stiffness was not greater at any time point for the injured cohort when compared to the uninjured cohort. Thus, it was concluded that vertical stiffness could not be associated with muscle strain injury.

6.1.3. MUSCLE CO-ACTIVATION DID NOT APPEAR TO INCREASE LOADING OF THE ACL

Results from the studies presented in chapters two and three showed that vertical stiffness was related to hamstring-quadriceps co-activation and pre-activation, and that vertical stiffness was not linked to muscle strain injury. The next step to this body of research was to establish if vertical stiffness could be linked to non-contact ACL injury. However, because the volume of ACL injuries

which occur per professional football club per season is far less than the number of muscle strain injuries (Opar and Serpell, 2014) a study similar to that described in chapter three using non-contact ACL injury as an outcome variable could not be conducted. Consequently, the studies conducted as part of this body of research after chapter three explored the relationship between vertical stiffness and measures known to load the ACL or measures which represent loading of the ACL; namely anterior tibial translation and ACL elongation respectively. This was done across two studies, the first of those, presented in chapter four, aimed to determine the effect of hamstring-quadriceps co-activation on knee joint motion and ACL elongation. As will be discussed in this section, hamstring-quadriceps co-activation constrained excessive joint motion and ACL elongation. Therefore, the second study, presented in chapter five, examined the relationship between vertical stiffness with anterior tibial translation and ACL elongation.

The study presented in chapter four was a pilot study which used novel, high-tech, medical imaging technology to measure knee joint motion on a basic step-up task while concurrently measuring muscle activity of the vastus medialis, vastus lateralis, semimembranosus and biceps femoris using EMG. Knee joint kinematics were measured from a 4-D model which was created for each participant from image registration of CT with fluoroscopy frame by frame. The technology used in this study differs from other kinematic measurement/motion analysis systems in that this technology measures knee joint motion directly from bone. Other motion analysis systems typically use skin marker sets with biomechanical models to predict translations and rotations. Skin markers are limited by the effect of wobbling mass (i.e. skeletal muscle and subcutaneous adipose tissue) (Reinschmidt et al., 1997, Begg et al., 1989, Windolf et al., 2008). The technology used for this project has known precision at the knee joint of only 0.38 mm error for in-plane translation and 0.42 degrees for rotation (Scarvell et al., 2010). In this study the attachments of the ACL were mapped to the 4-D model in accordance with methods described elsewhere (Grood and Suntay, 1983) for each frame. ACL elongation was considered the difference between maximum and

minimum ACL length throughout the step-up. To this point in time no other study had measured ACL elongation dynamically *in-vivo*, or anterior tibial translation with such precision *in-vivo*.

In this study it was hypothesised that with deliberate co-activation of the hamstrings and quadriceps muscles knee joint motion would be constrained, including anterior tibial translation, and ACL elongation would not be as great. It should be acknowledged, however, that this study revealed a strong inverse relationship between ACL elongation and medial hamstring-quadriceps co-activation, but positive relationship between ACL elongation and lateral hamstring-quadriceps co-activation. This may be concerning given the study presented in chapter two revealed greater activity of the lateral hamstring and quadriceps muscles on a hopping task which simulated the change of direction manoeuvre seen when non-contact ACL typically occurs. Furthermore, a strong positive relationship was observed between lateral hamstring-quadriceps co-activation index calculated and vertical stiffness in that study. Thus, evidence from this body of research to this point leans toward vertical stiffness being related to ACL elongation and therefore a risk factor for non-contact ACL injury. However, the studies presented in chapter two and in section 6.1.1. have shown that the relationship between hamstring function and vertical stiffness or hamstring function and ACL elongation is likely to be a general one; muscles do not operate in isolation, rather synergistically, and therefore this data should be interpreted with caution.

Careful thought has been given to differences in the methods used in chapter two, compared with chapter four, hence the call for caution when considering anterior tibial translation and ACL elongation with lateral hamstring-quadriceps co-activation. In the study presented in chapter four co-activation index was calculated from peak activation, not mean activation as was the case in chapter two. Worth noting also was that in the study presented in chapter two a strong positive relationship was seen for vertical stiffness with peak medial hamstring activation and timing of peak medial hamstring activation. Finally, in the present study a co-activation index of the medial and

lateral hamstring muscles was calculated from peak semimembranosus activation divided by peak biceps femoris activation; for flexion and extension semimembranosus and biceps femoris muscles are agonists but for knee rotation and for knee adduction/abduction they are antagonists. Therefore, it was expected that knee abduction and external rotation would not be as great when medial-lateral hamstring co-activation index was 'smaller', and this would manifest as an inverse relationship between ACL elongation and medial-lateral hamstring co-activation index. Indeed, as the ACL elongated the medial-lateral hamstring co-activation index reduced. When the findings of the four studies in this thesis are seen together, the data support the notion that hamstring and quadriceps function is synergistic in dynamic hopping and landing tasks. Therefore, the relationships for lateral hamstring and quadriceps activation, or for medial hamstring and quadriceps activation, with vertical stiffness and ACL elongation are not closed relationships. Rather, the relationships are co-dependent; it is the combined actions of the hamstrings and quadriceps which influence dynamic knee joint stability. This in turn will affect vertical stiffness (see section 1.5. of the introduction and figure 2).

Another important finding from the study in chapter four, was the strong positive relationship between anterior tibial translation and ACL elongation. Some limitations exist surrounding how ACL length and subsequent ACL elongation were measured in this study. These limitations were explored in the discussion section of the chapter. However, at the very least, it should be noted that anterior tibial translation is a measure known to load the ACL (Butler et al., 1980) and it is likely that a manifestation of this is ACL elongation. The anterior tibial translation and ACL elongation relationship provides validation of the findings of this chapter. Therefore, the strong positive relationship between the two measures adds strength to the argument that net co-activation of the hamstring and quadriceps muscles reduces ACL elongation.

Although the study presented in chapter four was just a pilot study, combined with other research (Isaac et al., 2005, MacWilliams et al., 1999), and read in context with studies presented in this body of research prior to this, it appears that net hamstrings-quadriceps co-activation constrains anterior tibial translation and subsequently reduces ACL elongation; a measure which represents loading of the ACL. Given that net hamstring-quadriceps co-activation is also related to vertical stiffness, a story begins to emerge from chapters two to four that vertical stiffness may not increase ACL injury risk.

6.1.4. VERTICAL STIFFNESS IS NOT RELATED TO MEASURES WHICH REPRESENT LOADING OF THE ACL

The next and final step for this body of research was to examine the relationship for vertical stiffness with anterior tibial translation and ACL elongation. Chapter two showed that vertical stiffness was related to pre-activation and co-activation strategies of the hamstring and quadriceps muscles, and chapter four demonstrated that net co-activation of the hamstring and quadriceps reduces anterior tibial translation and therefore ACL elongation. However, it does not necessarily follow that vertical stiffness and ACL elongation were unrelated.

To test whether vertical stiffness was associated with an increase or decrease in anterior tibial translation and ACL elongation, it was necessary to measure these in a specific experiment. In the study presented in chapter five of this thesis participants executed a hopping task similar to that described in chapter two which was designed to simulate the change of direction manoeuvre typically observed when non-contact ACL injury occurs. Vertical stiffness was measured during this hopping task and knee joint motion (including anterior tibial translation) was measured concurrently using kinematic measurement technology described in chapter four.

In the study presented in chapter five there was no relationship seen between vertical stiffness and anterior tibial translation or ACL elongation. However, a very strong positive relationship was seen for anterior tibial translation with ACL elongation. This was not surprising because a considerable amount of ACL elongation is bourn from anterior tibial translation (Butler et al., 1980, Noyes et al., 1974a), and because a moderate relationship was seen between the anterior tibial translation and ACL elongation in the study presented in chapter four. Therefore, good construct validity for how ACL elongation was measured in this study and the one previous may be argued. Adding strength to the argument that vertical stiffness is not related to measures known to load, or which represent loading of, the ACL.

An unexpected, but exciting, outcome from this study was the model that was developed enabling measurement of ACL length from anterior tibial translation. This model offers scope to measure the effect of other injury mitigation strategies or rehabilitation outcomes with respect to the ACL.

6.2. CONTEXT OF FINDINGS

This body of research set out to establish if vertical stiffness was linked to traumatic non-contact injury in elite football of various codes, particularly non-contact ACL injury. Some of the methods adopted in this thesis were novel (i.e. the CT-fluoroscopy image registration technology). These novel methods enabled us to do what no other study has previously done and that is to measure dynamic knee joint stability (i.e. anterior tibial translation) with high precision *in-vivo*, and gain some insight into *in-vivo* ACL elongation, while performing dynamic tasks. This work was also novel because of the measurement of muscle activity while concurrently measuring anterior tibial translation dynamically, and because of the hopping task required of participants in several studies

was designed to simulate the change of direction manoeuvre typically seen when non-contact ACL injury occurs.

This work was important because interest in research concerning stiffness is increasing considerably as noted in the literature review presented in section 1.4. Previous work has shown that greater stiffness can be linked to superior performance in athletic tasks requiring running and jumping (Bret et al., 2002, Morin et al., 2011, Morin et al., 2006, Seyfarth et al., 1999, Spurrs et al., 2003, Degache et al., 2016, Maloney et al., 2016, Pruyn et al., 2014), and work which has described how to enhance stiffness would suggest that some practice is already implementing training strategies to increase stiffness (Butler et al., 2003, Devita and Skelly, 1992, Millet et al., 2002, Morin et al., 2009, Moritz and Farley, 2004, Moritz and Farley, 2006, Spurrs et al., 2003). There is some academic opinion, however, that stiffness increases injury risk (Bradshaw and Hume, 2012, Butler et al., 2003, Lorimer and Hume, 2016, Pruyn et al., 2012, Watsford et al., 2010). If this were the case then it would be negligent to prescribe training to enhance stiffness. However, much of the academic discussions around stiffness and injury concerns only overuse injuries (Bradshaw and Hume, 2012, Butler et al., 2003, Lorimer and Hume, 2016). As noted in the introduction of this thesis overuse injuries are likely to arise because of exponential, or improper, loading; not because of increased vertical stiffness. Two research papers have argued hamstring muscle strain is associated with increased stiffness. However, as also noted in the introduction of this thesis, there was no explanation given by the authors of those studies as to why they believed stiffness increased hamstring strain injury risk (Pruyn et al., 2012, Watsford et al., 2010). Furthermore, as noted in section 3.1., there were some notable flaws in the studies by Watsford et al. (2010) and Pruyn et al. (2012) which could offer an alternative explanation as to why the injured cohort sustained a muscle strain injury but the uninjured cohort did not. Therefore, due to the ‘confusion’, or lack of certainty, around the role stiffness plays in non-contact injury this body of research sought to gain more clarity over whether or not non-contact injury could be linked to vertical stiffness. However, this

research only investigated traumatic non-contact injuries, and risk factors for traumatic non-contact injury.

The outcomes from the studies presented in this thesis, combined, suggest that traumatic non-contact injury is not linked to vertical stiffness. This is because data from this work revealed no significant difference in vertical stiffness for professional footballers who sustained a muscle strain injury when compared to a non-injured cohort, and because vertical stiffness was not related to measures known to load or which represent loading of the ACL. A definition of traumatic non-contact injury was applied for this body of research (section 1.5.) because, again, an argument may be made that overuse injuries are more the result of improper loading, not vertical stiffness. Furthermore, trying to establish a relationship between overuse injuries and vertical stiffness would require an extensive longitudinal study which would be impractical in a professional football environment and well beyond the scope of this study. Nevertheless, when one considers the outcomes of this study, and that stiffness is comprised of a number of components (i.e. functioning skeletal muscle, connective tissue etc.) so it therefore is a ‘global’ measure which may combine a number of risk factors (Lorimer and Hume, 2016), it is hard to conceive that stiffness is a risk factor for non-contact injuries regardless of whether they are overuse or traumatic in nature. Rather, it could be that dysfunction of one of the components that contribute to system stiffness which is the main cause of injury. It would, however, be beneficial to confirm this with research investigating the relationship between vertical stiffness and overuse injury. Such a study would need to be epidemiological and longitudinal in nature; sports injury data would need to be collected along with regular and ongoing measures of vertical stiffness.

Several other important observations can be made from the work presented in this thesis. The first being that vertical stiffness appeared to constrain excessive knee joint motion (i.e. anterior tibial translation) because of adoption of co-activation strategies of the hamstrings and quadriceps.

The second important observation was the development of a model for predicting ACL elongation from anterior tibial translation. These findings will now be explored in a little more detail.

Results from the studies presented in chapters two and four combined show that pre-activation and co-activation of the hamstrings and quadriceps is related to vertical stiffness, and that excessive knee joint motion is constrained by the net co-activation of these muscle groups. Furthermore, despite activity not being measured from all muscles in the thigh, data from this body of work suggests that medial hamstring activation relative to lateral hamstring and net quadriceps activation appears particularly important in this process of reducing anterior tibial translation and ACL elongation. However, this raises an issue that if medial hamstrings are important for constraining ACL elongation, then harvesting medial hamstrings for ACL reconstruction might be detrimental to future graft protection. Current knee reconstruction techniques involve harvesting medial hamstring tendons, usually semitendinosis but sometimes both semitendinosis and gracilis, leaving the medial hamstrings weaker. This could be a contributing factor as to why one of the greatest risk factors for ACL injury is a history of ACL injury, particularly when one considers that if an ACL injury is sustained within 12 months of a prior ACL injury is more likely to be in the same knee (Orchard, 2001) and it is known that skeletal muscle inhibition may remain for up to 12 months following trauma to the muscle (Opar et al., 2012). However, simply over emphasising strength and functional development of the medial hamstrings to prevent ACL injury from occurring, or in the rehabilitation process, might not be the answer. Excessive co-activation of the medial hamstrings and quadriceps may increase compression around the medial compartment of the knee, and consequently osteoarthritis of the knee could hasten (Lewek et al., 2004, Hodges et al., 2015). Osteoarthritis of the knee may require knee replacement surgery later in life (Scarvell et al., 2006, Tashman et al., 2007, Scarvell et al., 2005, Ajuied et al., 2014), or result in the development of co-morbidities secondary to the knee injury arising from inactivity due to severe knee joint pain (Myer et al., 2014, Osterberg et al., 2013). Hence, the role of the lateral hamstrings and quadriceps

in the co-activation process to reduce excessive knee joint motion (i.e. anterior tibial translation) is also important; not just to increase vertical stiffness, but also because it may contribute to knee and overall general health (e.g. early onset of osteoarthritis). This is despite the lateral hamstring and quadriceps having been shown to be important agonists for knee joint external rotation and valgus/knee abduction; the knee joint kinematics often observed when ACL injury occurs (Besier et al., 2003). Thus, there is enormous scope for research into the synergism of the muscles around the knee.

Finally, in the study presented in chapter five a model for predicting ACL elongation from anterior tibial translation was derived from a linear regression analysis the correlational analysis between anterior tibial translation and ACL elongation. This has not been done before, and the model offers some opportunity for new or more applied research concerning ACL injury. However, some caution must be applied because when the ACL is mostly lax or when it is at extreme elongation nearing failure, the relationship between ACL elongation and anterior tibial translation might not be linear. Thus, further research may also be conducted to develop the model with greater accuracy.

6.3. PRACTICAL APPLICATIONS AND CLINICAL IMPLICATIONS

Given the outcomes from studies presented in this thesis it would be remiss to not give some description of how stiffness can be trained. A sparsity of actual research describing how stiffness may be enhanced exists, however some papers published in scientific journals have suggested that

simply giving athletes feedback about how they perform a task (i.e. knowledge of performance) can assist in modifying stiffness because humans can consciously alter stiffness (Butler et al., 2003, Morin et al., 2009). Other papers have argued strength training can increase stiffness (Millet et al., 2002), as will plyometric training (i.e. jumping) (Butler et al., 2003, Spurrs et al., 2003, Saunders et al., 2006) and running on unstable and uneven surfaces (Butler et al., 2003, Moritz and Farley, 2004, Moritz and Farley, 2006). The volume and intensity of the different training modalities for optimising stiffness is unclear. Consequently, exploration of how to optimally enhance vertical stiffness also provides scope for further research.

That vertical stiffness may be enhanced by plyometric training lends further support to the theory presented in this thesis that vertical stiffness is not associated with traumatic non-contact injury because plyometric training may reduce injury risk. Previous research has argued that plyometric training reduces injury risk (Markovic and Mikulic, 2010), some specifically that plyometric training reduces ACL injury risk (Sugimoto et al., 2015). It has been suggested that the reason for this is because a carefully implemented and appropriately progressed plyometric training program facilitates development of neuromuscular control strategies (i.e. pre-activation and co-activation strategies) which limit valgus loading of the knee (Sugimoto et al., 2015, Struminger et al., 2013). The research presented in this thesis does not actually suggest vertical stiffness protects against injury, it simply argues that injury risk is not associated to increased vertical stiffness. However, given evidence suggests that plyometric training reduces injury risk, that plyometric training enhances pre-activation and co-activation strategies, and that plyometric training increases vertical stiffness, there is scope to research further the potential of vertical stiffness as a ‘protector’ against traumatic non-contact injury.

6.4. LIMITATIONS

As with all research, there are some limitations to the work presented in this thesis. To begin with, we did not investigate the relationship between vertical stiffness and overuse injury. However, this was a cross-sectional study. Overuse injury, defined in section 1.5., typically occurs when strain below the single load-failure threshold occurs repetitively (Opar et al., 2012, Warden et al., 2006), so to investigate overuse injury requires a longitudinal study design. As stated in section 6.2., a longitudinal study was beyond the scope of this body of work and would be difficult to conduct in a professional football environment. In addition, as vertical stiffness is the sum of resistance to change in shape against force application from all structures combined (e.g. ligaments, functioning skeletal muscles, tendons, bone etc.), it is a ‘global’ measure which may combine several injury risk factors. Therefore, it was clearer to investigate the link between vertical stiffness and traumatic non-contact injury only. Nevertheless, future research investigating the link between vertical stiffness and overuse injury may be beneficial.

Another limitation to this work relates to the fact that a lot of discussion has been devoted to the synergism between medial and lateral hamstring and quadriceps for net co-activation. It could be argued that the precise synergistic relationship should have been investigated and reported. However, the aim of this work was to examine the relationship between vertical stiffness and traumatic non-contact injury, not to describe the relationship between synergism of the hamstrings and quadriceps with knee joint motion. Nonetheless, the evidence in this body of research does support a relationship between synergistic hamstring-quadriceps co-activation and knee joint motion, and future research describing in detail the actual synergism would be particularly beneficial.

A considerable limitation to this body of research relates to the study presented in chapter four and the fact that statistical significance was not reached for differences in peak muscle activation for each muscle, differences in timing of peak activation for each muscle in the co-activation indices, and for differences in joint motion irrespective of whether the step-up was performed with co-activation or without. For each of those measures broad variation was observed as demonstrated by a large standard deviation and this was particularly the case for the variables concerning measurement of muscle activation. As noted in that paper this may be a result of not having normalised RMS EMG to maximum voluntary contraction. However, in design of the study, and as stated in the discussion of that paper, this was not believed necessary as the purpose behind measuring muscle activity was not to describe or quantify the amount of muscle activity which was occurring, rather just confirmation that a greater amount of activation was in fact happening for the co-activation step-up was needed. The standard deviation for muscle activation in the co-activation indices may also have been reduced by calculating co-activation index from mean RMS EMG from each muscle in the co-activation indices throughout the step-up tasks as opposed to calculation of co-activation index from peak RMS EMG. However, this was impossible because the technology used for measuring knee joint motion in that study does not have capacity to synchronise with EMG or force plates making it difficult to ascertain the start or end point of the step-up. Thus, mean RMS EMG could not have been accurately determined.

With regards to the variation observed for knee joint motion in the study presented in chapter four, it was typically not as great for the step-up with co-activation. This suggests that something as simple as increasing the sample size may reduce the variation for these measures and statistical significant may be reached. However, as stated in the introduction of that paper this was a pilot study only as there were ethical considerations to be made with regard to exposing young healthy participants to unnecessary doses of radiation hence the small sample size. Similar research with a greater sample size in the future might be beneficial. In the mean-time if the outcome of this

study is read in context with other studies similar (Isaac et al., 2005, MacWilliams et al., 1999) enough evidence to support the theory that co-activation of the hamstrings and quadriceps reduces ACL loading exists.

A final limitation was described by the reviewers from the internationally peer-reviewed journals for the study presented in chapter five. The reviewers asked why EMG was not concurrently used to measure muscle activation during the hopping task. While seen as a limitation to that study if read in isolation, when considered in the context of other studies in this thesis it was not necessary to measure EMG as well as ground reaction forces and knee motion in four dimensions. The studies presented in chapter two showed that net hamstring and quadriceps pre-activation co-activation increased vertical stiffness for multidirectional tasks, and study four showed that net co-activation of the hamstrings and quadriceps did not increase measures which represent loading of the ACL. Furthermore, measurement of muscle activity in the study presented in chapter five study was not actually part of the hypothesis being tested in this study. For these reasons the use of EMG to measure muscle activity was not necessary.

6.5. DIRECTIONS FOR FUTURE RESEARCH

The following areas have scope to be investigated further based on the results obtained from this body of research:

- What effect does neuromuscular inhibition following soft tissue strain have on hamstring and quadriceps synergism, vertical stiffness and subsequent anterior tibial translation and ACL elongation?
- How might vertical stiffness relate to overuse injury?
- This research has argued that vertical stiffness appears unrelated to traumatic non-contact injury, can we explore further to see if it actually protects against injury?
- How can vertical stiffness most effectively be trained without risk of sustaining overuse injury?
- How can the model developed for measuring ACL length from anterior tibial translation be used in other research?

6.6. CONCLUDING REMARKS

This thesis has taken the research question: “Is vertical stiffness a risk factor for common traumatic musculoskeletal injuries in the football codes?” and moved through a sequence of studies to answer the question with a focus on ACL injury. The first study was an observational study which showed that net hamstring and quadriceps pre-activation co-activation increased vertical stiffness for multidirectional tasks. The second study looked at retrospective injury data from Australian Rules football and drew the conclusion there was no difference in vertical stiffness between players

who sustained a muscle strain injury and those that did not. Study three showed that as net co-activation of the hamstrings and quadriceps increased, a surrogate measure for load on the ACL did not. Finally, while the studies presented in chapters two to four started to paint a picture that vertical stiffness was not a risk factor for muscle strain or ACL injury, it did not necessarily follow. Therefore, it was important to examine directly whether vertical stiffness increased ACL injury risk. Study four showed that vertical stiffness was not related to a same surrogate measure for load on the ACL.

Outcomes from this body of work suggest that co-activation strategies around the knee joint enhance dynamic knee joint stability and vertical stiffness. This dynamic knee joint stability appears to constrain the knee joint from secondary motion not associated with the primary movement, placing less 'stress' on the ACL and reducing its risk of failure/injury. Not surprisingly, therefore, ACL elongation was not related to vertical stiffness. This body of work also showed that vertical stiffness is not greater in professional footballers who sustain a muscle strain injury. Therefore, a story is emerging that vertical stiffness is not associated with traumatic non-contact injuries in field and court sports. It is important to note, however, it is not yet possible to say conclusively that increasing vertical stiffness through training will protect against non-contact musculoskeletal injury, work presented in this thesis has demonstrated that injury risk is not heightened because of increased vertical stiffness.

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