The impact of Achilles tendon pathology on lower limb joint stiffness regulation during hopping tasks

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Statement of sources

This thesis contains no material published elsewhere or extracted in whole or in part from a thesis by which I have qualified for or been awarded another degree or diploma.

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Abstract

Achilles tendinopathy (AT) is a debilitating running injury affecting 50% of distance runners over their lifetime. Ultrasound (US) imaging studies have shown that pathological changes are present in 11-52% of asymptomatic individuals. Impairments of the lower leg muscle-tendon function may develop with AT however alteration of lower limb loading strategies has not been examined in runners with asymptomatic Achilles tendon pathology. The primary aim of the thesis was to determine if Achilles tendon pathology changes lower limb loading patterns. It was hypothesised that there would be a different loading pattern between the ankle and knee in runners with Achilles tendon pathology. In order to investigate this primary aim, secondary aims were developed to determine the prevalence of Achilles tendon pathology in an asymptomatic running population and to determine any associated factors of tendon pathology. This study initially assessed the prevalence of tendon pathology and a number of associated risk factors for 37 experienced, high mileage male endurance runners with no history of Achilles tendon pain. The tendon was assessed using US by a musculoskeletal radiologist and classified as either normal or abnormal. Height, body mass, waist circumference, and weight bearing ankle dorsiflexion range of motion (ROM) with the knee in a flexed and extended position were measured. A survey quantified the running history of participants. Following the initial study, 14 runners with no history of Achilles tendon pain and a normal Achilles tendon on US imaging, and 12 runners with asymptomatic Achilles tendon pathology were assessed in a series of further studies. A third group of 12 runners with symptomatic AT were also studied. Each runner completed single leg hopping for both limbs on a level and inclined surface. Embedded in the surface was a force plate (1000Hz) that was synchronised with a three dimensional motion capture

system (250Hz). Nonparametric statistics were used to examine the effect of surface angle and group on hopping biomechanics. All results are reported as median and interquartile range (IQR). Almost half (46%) of the asymptomatic distance runners had at least one abnormal tendon. The runners with asymptomatic Achilles tendon pathology had significantly more years of running history (Median 20.0 years, IQR 6.0-25.5, p=0.024) than the runners with no pathology on US (Median 7.0 years, IQR 5.0-15.0). No significant differences between the groups was identified for age, height, mass, waist circumference, ankle ROM, number of weekly running sessions, weekly mileage and number of long distance (marathon and half marathon) running events completed in their lifetime. Symptomatic runners had significantly less active ankle joint stiffness (Level – Median = 8.2 Nm/kg/rad, IQR = 7.7-9.2; Incline - Median= 8.1 Nm/kg/rad, IQR = 7.2-9.7) when compared to the normal group (Level – Median = 9.8 Nm/kg/rad, IQR = 9.0-10.5; Incline - Median= 10.2 Nm/kg/rad, IQR = 8.7-10.4) for both the level (p=0.044) and inclined (p=0.042) surfaces. No differences were identified for leg stiffness between the three groups. Asymptomatic male distance runners had a high incidence of tendon pathology compared to other populations (e.g. soccer players) and cumulative load in running years is an associated factor of tendon change. This thesis identified that the presence of Achilles tendon pathology without a history or presence of pain did not alter lower limb loading strategies. However, runners with symptomatic AT had reduced ankle stiffness. This may explain the recalcitrant and recurrent nature of AT if runners are not able to increase their ankle stiffness following rehabilitation and recovery after injury.

Conference presentations

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Dedication

This thesis is dedicated to those closest to me, my family. The two people who I have missed spending more quality time with are my beautiful wife Lauren and my special little girl Olivia. I can't wait to spend more time with both of you upon the completion of this thesis. I would like to thank Lauren, who has been extremely supportive of my decision to pursue further study. Her patience and tolerance has definitely not gone unnoticed while I have tried my utmost to blend family, working full-time, studying and running. The first of many lessons I have learnt along the research road is that you learn very quickly who are dearest to you, and consequently it is essential to make the most of every moment in life.

Acknowledgements

- I sincerely thank Dr Elizabeth Bradshaw, who with her support and assistance has been instrumental in my research endeavours. Liz you have always exceeded what is expected of you and for that I am exceptionally grateful. Thank you for your patience, understanding and guidance to keep me on track or re-direct me back on track throughout the Masters program. Liz was always a great sounding board, especially when an apparent problem arose. Apart from your incredible knowledge of biomechanics I discovered your secret love for statistics that helped guide me along the correct path. Liz, you were always willing to find a solution in my many moments of stress.

- Professor Jill Cook, the tendon guru, you are one of the most humble individuals I have ever met. The wealth of knowledge you have and are always willing to share has enabled this research to reach its potential. Despite your busy schedule you always make yourself available. You have made me want to be not only a better researcher, but a better clinician. I have consequently challenged myself more than I ever thought I would and am now always looking at ways to improve what I do. Prior to this process I was becoming bored with my work as a physiotherapist, but thanks to you I have a passion and self belief in what I do.

- Dr Kade Paterson, who has given me a great deal of his time and support during the research process. He has been patient during times of frustrations and always provided encouragement allowing me to explore my particular

- My lab mates in the early days of my thesis that have now successfully completed their PhDs, Bianca Share and Eddy Crendel. They were both always offering their time and help to assist with my work

- ADIDAS for the supply of the footwear in every size available for my biomechanical analysis.

- Finally, I would like to thank the distance runners who were generous with their time to participate in this study. It is their willingness to be involved that has allowed for the completion of this thesis.

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coefficient, t-tests are paired samples t-tests, CV% is a coefficient of variation as a percentage of the inter-day mean score, and ES is a Cohen's effect size.

Glossary of Abbreviations

AT	Achilles tendinopathy
ASYM	Asymptomatic Achilles tendon pathology
BW	Body weight
cm	Centimetre
CC	Contractile component
CV	Coefficient of variation
COM	Centre of mass
DF	Dorsiflexion
ES	Effect size
Hz	Hertz
ICC	Interclass correlation
IQR	Interquartile range
k_{Joint}	Joint stiffness
k_{Vert}	Vertical leg stiffness
km	Kilometre
kN	Kilonewton
kN/m/kg	Kilonewton per meter per kilogram
m	Metre
ms	Millisecond
m/s	Metres per second
MDiff%	Mean difference as a percentage
ME	Margin of error
mm	Millimetre
n	Sample
Nm	Newton metres

Nm/kg/rad	Newton metre per kilogram per radian
PEC	Parallel elastic component
s	Second
SAT	Symptomatic Achilles tendinopathy
SD	Standard deviation
SEC	Series elastic component
SEM	Standard error of measurement
SPSS	Statistical package for the social sciences
US	Ultrasound
VISA-A	Victorian Institute of Sport Assessment - Achilles Questionnaire

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Chapter One

Introduction

1.1 Achilles Heel

The term "Achilles Heel" is frequently utilised to discuss a person's point of weakness. The term has been derived from Greek mythology where Achilles, a Greek hero of the Trojan War was killed after he was shot in his heel with an arrow. His heel was the only vulnerable part of his body that was not immersed in the river Styx when his mother attempted to immortalise him. For distance runners, the Achilles tendon is vulnerable to injury.

1.2 Statement of the problem

At the start of each year, distance runners have new reasons to run or to run more. The distance runner may have set a goal of completing their first marathon, whilst the habitual runner may have set a goal to break their own personal best running time or running further than they have previously (e.g. half marathon to full marathon or ultra marathon). Starting to run or simply increasing the intensity or volume of running places additional mechanical load on the body. For many runners, discomfort and pain develop and then subside if the body's tissues can adapt to the higher mechanical load. However, if the rate of tissue adaptation is less than the rate of continued tissue stress an overuse injury may develop.

Despite current injury prevention strategies including advancements in footwear technology, compression garments and accessibility to information on the internet, injury rates remain high and relatively unchanged from the 1970's. It is reported that up to 75% of runners are injured every year (Almonroeder, Willson, & Kernozek, 2013; Daoud et al., 2012; Hreljac, 2004; Taunton et al., 2003; Taunton et al., 2002). It is not known whether the high injury rate from running is because the

running population is very different now compared to the 1970's, a result of modern lifestyles (e.g. prolonged sitting), or because of poor training management strategies.

Achilles tendinopathy (AT: pain and dysfunction in a tendon) is one of the most common tendinopathies among endurance runners (Knobloch, Yoon, & Vogt, 2008). The condition also accounts for 4% of injuries presenting to sports medicine clinics and almost a quarter (18-24%) of all running injuries (Di Caprio, Buda, Mosca, Calabro, & Giannini, 2010; Magnussen, Dunn, & Thomson, 2009; Taunton et al., 2002; Van Ginckel et al., 2009). Whilst AT is a common injury amongst runners, the mechanisms for its development and its impact on lower limb function are poorly understood. Therefore AT is often difficult to manage clinically, taking prolonged periods of time to rehabilitate and is often recurrent.

Achilles tendinopathy is a debilitating injury that results in running athletes reducing volume, frequency and intensity of training. The consequence of AT on training, is a reduction in performance and unfortunately for many runners, cessation of activity. In addition to the compromised performance, the lifetime incidence of AT in endurance runners is approximately 50% (Kujala, Sama, & Kaprio, 2005). Achilles tendinopathy consequently has serious implications for individuals involved in running sports. In order to assist in the management and prevention of AT, research has focused on potential intrinsic and extrinsic risk factors. This research has investigated a number of associated factors for the development of Achilles tendon pathology.

Predisposed Runner
- **Intrinsic risk factors**
  - Gender
  - Older Age
  - Adiposity
  - Structural abnormalities
  - Ankle Dorsiflexion
  - Foot biomechanics (e.g. pronation)

Susceptible Runner
- **Extrinsic risk factors**
  - Mechanical overlaod
  - Fatigue
  - Inappropriate footwear
  - Environment (e.g. hilly terrain, soft surface)

Injured Runner
- **Inciting event**
  - Solitary intense run
  - Sudden increase in mileage
  - Sudden change of surface
  - Sudden change of footwear
  - Period of offloading followed by loading
  - Biomechanics (e.g. change of foot strike)

Figure 1.1 Combinations of intrinsic and extrinsic risk factors, in combination with a load stimulus may predispose some runners to injury (Modified from Bahr & Krosshaug (2005)).

Several risk and associated factors have been examined for AT and tendon pathology however the relationship between excessive Achilles tendon mechanical loading, tendon pathology and AT remains unclear. A prospective study design is required to identify risk factors, whereas an associated factor is identified during retrospective study designs (Cowan et al., 2004; Gabbe, Bennell, Finch, Wajswelner, & Orchard, 2006). The identified intrinsic and extrinsic factors are frequently multifactorial (summarised in Figure 1.1). Intrinsic factors include older age (Di Caprio et al., 2010; Gaida, Alfredson, Kiss, Bass, & Cook, 2010), gender (men > women)

(Astrom, 1998; Gaida et al., 2010), structural abnormalities (Comin et al., 2013), adiposity (waist measurments; Gaida et al., 2010), ankle dorsiflexion (DF) range of motion (Kaufman, Brodine, Shaffer, Johnson, & Cullison, 1999; Mahieu, Witvrouw, Stevens, Van Tiggelen, & Roget, 2006; McCrory et al., 1999) and excessive foot pronation (Clement, Taunton, & Smart, 1984; Donoghue, Harrison, Laxton, & Jones, 2008; McCrory et al., 1999). Extrinsic factors include mechanical overload (e.g. increase in running mileage or intensity) (Di Caprio et al., 2010; Knobloch et al., 2008; McCrory et al., 1999; Rompe, Furia, & Maffulli, 2008; Schepsis, Jones, & Haas, 2002), fatigue (Paavola et al., 2002), inappropriate footwear (Wyndow, Cowan, Wrigley, & Crossley, 2010), hilly terrain and running on soft surfaces (e.g. sand) (Knobloch et al., 2008; McCrory et al., 1999). Altered loading strategies have been identified to be associated with asymptomatic patellar tendon pathology (Edwards et al., 2010), which is a potential risk factor for pain. Loading strategies have yet to be examined in Achilles tendon pathology. Further research is required to assess the biomechanics of runners with asymptomatic tendon pathology to further our understanding of the effect of pathology and pain in the Achilles tendon on lower limb behaviour, a factor important for not only injury management but also injury prevention.

Investigation of the biomechanical variables that may be affected by pain and pathology of the tendon needs to be relevant to the function of the Achilles tendon. The Achilles tendon is an important structure in the lower limb during activities including running, jumping, hopping and bounding, contributing approximately 35% to the mechanical energy for each stride during running (Farley, Blickhan, Saito, & Taylor, 1991; Ker, Bennett, Bibby, Kester, & Alexander, 1987). During running the tendon stretches during the first portion of the stance phase of the gait cycle and

recoils to return that energy back to the individual at the time of push-off (Lichtwark & Wilson, 2006). This allows for storage of elastic strain energy during the elongation phase and a consequent conversion into kinetic energy upon recoil (Anderson, 1996; Child, Bryant, Clarke, & Crossley, 2010; Kyrolainen, Finni, Avela, & Komi, 2003; Rosager et al., 2002). Pathology of the tendon has the potential to alter the tendon's mechanical properties (Child et al., 2010; Wang, Lin, Su, Shin, & Huang, 2012), therefore investigating the impact of Achilles tendon pathology on the leg's spring-like characteristics may increase our understanding of biomechanical factors associated with tendon overload and pathology. Musculoskeletal stiffness is a mechanical parameter that may influence the storage and release of the elastic properties of the lower limb during activities such as running and hopping. Musculoskeletal stiffness, specifically increased stiffness is understood to be important for performance by optimising power output and reducing metabolic costs, however too much or too little may be associated with injury risk (Butler, Crowell, & Davis, 2003; Kuitunen, Ogiso, & Komi, 2011). Consequently further research is required to investigate the impact of tendon pathology on lower limb stiffness. Identifying alterations in loading strategies may ultimately aid in injury prevention and rehabilitation.

1.3 Aims and hypotheses

The primary aim of this research was to investigate the impact of Achilles tendon pathology on lower limb biomechanics, specifically stiffness regulation during hopping. It was hypothesised that a reduction in leg stiffness and ankle stiffness would be identified in athletes with Achilles tendon abnormalities.

In order to achieve the primary aim, a number of secondary aims were developed. A lack of ankle DF range is regularly reported as a risk factor of AT and due to the inconsistencies reported in previous studies (Kaufman et al., 1999; Mahieu et al., 2006) a novel measurement platform was tested as part of this thesis. Therefore the aim of assessing DF with the platform was to evaluate the influence of providing feedback for maintaining heel contact during DF measurements as well as assessing the reliability of measuring DF on the platform. It was hypothesised that the use of the novel platform would result in lower measurements of ankle DF in weight bearing as compared to the standard protocol, and be a reliable apparatus for ankle DF measurement.

The prevalence of asymptomatic Achilles tendon pathology is relatively common with reported frequencies in non distance running populations and a generally active population ranging from 11% to 52% (Gaida et al., 2010; Gibbon, Cooper, & Radcliffe, 1999; Nicol, McCurdie, & Etherington, 2006). Therefore the next aim was to investigate the prevalence of asymptomatic Achilles tendon pathology in a high risk running population and identify associated factors for the presence of tendon pathology. It was hypothesised that distance runners would display a high prevalence of tendon pathology. Also, it was hypothesised that years of running and adiposity would be associated factors of tendon pathology.

Finally as part of exploring the primary aim, the lower limb's spring-like behaviour during incline hopping was assessed. The aim was to compare lower limb kinematics and kinetics during hopping on a level surface and an inclined surface. It was hypothesised that hopping on an incline would increase the demands (increased joint moments and range of motion) on the ankle joint and reduce the demands (reduced joint moments and range of motion) on the knee joint.

This research ultimately aims to further develop our understanding of loading factors on the Achilles tendon, which may have implications for injury prevention and management strategies for clinicians involved in the management of running athletes.

1.4 Outline of thesis

Chapter 1	• Introduction
Chapter 2	• Literature review
Chapter 3	• A simple platform for measuring ankle joint dorsiflexion whilst weight bearing (Pilot study)
Chapter 4	• Prevalence and factors associated with Asymptomatic Achilles tendon pathology in male distance runners
Chapter 5	• Lower limb stiffness regulation during level and incline hopping
Chapter 6	• Stiffness regulation of the lower limb in distance runners with Achilles tendon pathology
Chapter 7	• Discussion, conclusions and further research

1.5 Limitations

The following limitations have been identified in this thesis:

1) Sample size was small due to difficulty in recruiting people prepared to participate in testing that took several hours and was being done predominantly on weekends during family time or during business hours.

2) Relatively homogeneous group with very similar loading volumes limits the ability to generalise the findings to women or novice runners. This also adds depth to the study because any significant positive findings have greater strength.

3) Years of cumulative loading was specific to distance running and did not evaluate previous running related sports loading (e.g. football, soccer).

4) The amount and type of running training participants were involved in at the time of testing was not controlled due to variations in running programs.

5) Mid-portion AT was only assessed due to its more frequent occurrence in comparison to insertional AT, therefore findings cannot be generalised to include insertional AT.

1.6 Delimiters

The following delimiters have been identified in this thesis:

1) Male distance runners aged 25-55 years,

2) Participants meeting all the inclusion criteria,

3) Runners with mid-portion AT,

4) Methodology utilised to calculate leg and joint stiffness.

Chapter Two

Literature review

2.1 Overview

This chapter reviews the literature on the structure and function of a healthy and pathologic Achilles tendon and its influence on lower limb stiffness. This includes a review of tendon structure, radiological imaging, and risk and associated factors for tendon pathology. Distance runners and athletes involved in running related sports place great demands on the tendons in their lower limbs, especially the Achilles tendon.

In this chapter lower limb biomechanics during running and bounding activities are also reviewed as they may be influenced by either pathology of a tendon and/or pain. It is not known whether the pathology and/or pain are as a consequence of the changes in biomechanics or a causative factor. Assessing the leg's spring-like behaviour with a high tendon loading activity such as hopping may allow for the identification of altered loading strategies in the presence of compromise tendon structure. In addition to level hopping, incline hopping may challenge the Achilles further allowing for examination of the lower limbs behaviour and response to increased load.

2.2 Tendon anatomy and structure

The Achilles tendon is one of the longest and strongest tendons in the human body (Jarvinen, Kannus, Maffulli, & Khan, 2005; Silbernagel, Gustavsson, Thomee, & Karlsson, 2006). It provides attachment for the triceps surae muscle complex (gastrocnemius and soleus muscles) to the calcaneal tuberosity and it transmits large forces to the foot (Figure 2.1a). The gastrocnemius muscle is a bi-articular muscle that crosses both the knee joint and the ankle joint, whereas the soleus muscle is

mono-articular, only crossing the ankle joint. A normal healthy tendon is predominantly made up of type 1 collagen fibres, which provides the tendon with its tensile strength (Khan & Cook, 2003). Tendon structure incorporates collagen bundles, cells (tenocytes) and proteoglycan rich extra cellular matrix (ground substance). Tendon is well organised in hierarchical order, with collagen fibres forming bundles, that then combine to create primary bundles (subfascicles), secondary bundles (fascicles) and finally tertiary bundles forming the tendon (Sharma & Maffulli, 2005)(Figure 2.1b).

(a)   (b)
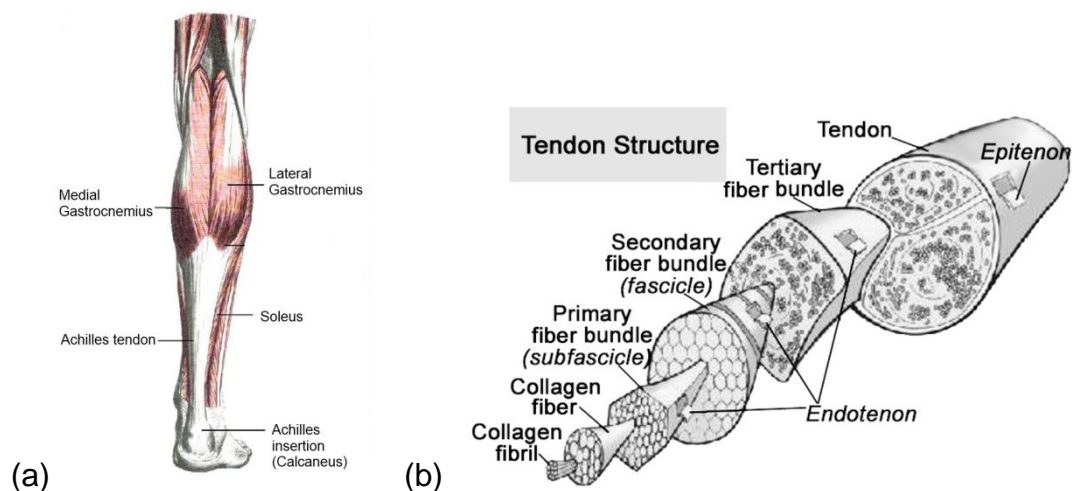
Figure 2.1 (a) Anatomy of the triceps surae (medial and lateral gastrocnemius and soleus) and Achilles tendon musculotendinous unit (modified from Lichtwark & Wilson, 2005). (b) Tendon structure (Sharma & Maffulli, 2005)

2.3 Biomechanical properties of tendons

A tendon transmits forces generated by a muscle to the bone to produce joint movement. Tendons also act as a buffer to limit muscle damage by absorbing

external forces during running (Maffulli, Sharma, & Luscombe, 2004). Tendons exhibit high mechanical strength, good compliance and an optimal level of elasticity to perform their unique role (Kirkendall & Garrett, 1997). The Achilles tendon behaves like a linear spring during running where the tendon lengthens and then shortens (Kyrolainen et al., 2003) (Figure 2.2). The Achilles complex allows for storage of elastic strain energy during elongation and a consequent conversion into kinetic energy upon recoil (Anderson, 1996; Child et al., 2010; Kyrolainen et al., 2003; Rosager et al., 2002). The elastic behaviour reduces the work required from the muscles and lowers the metabolic cost of locomotion (Ferris, Louie, & Farley, 1998).
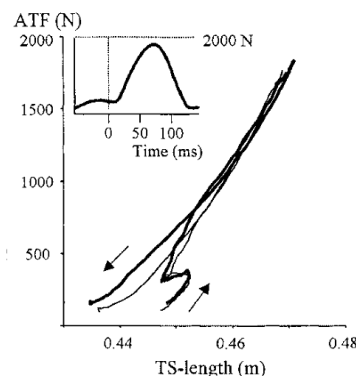
<u>Figure 2.2</u> Triceps surae muscle-tendon length in relation to Achilles tendon forces (Kyrolainen et al., 2003).

2.3.1 Tendon viscoelastic properties

The Achilles tendon exhibits viscoelastic properties during loading as it elongates. That is, the stress-strain characteristics of the Achilles tendon are not perfectly linear like a purely elastic structure (Hamill & Knutzen, 2009) (Figure 2.3a). The viscoelastic properties of the Achilles tendon include mechanical stiffness and

hysteresis. Tendon stiffness is determined by the change in length and change in force of a tendon, and the hysteresis is the region between the stored and released elastic energy that therefore represents energy loss or stored mechanical energy that is not fully returned when the applied force is removed (Hamill & Knutzen, 2009; Wang et al., 2012) (Figure 2.3b). Tendon stiffness is important for elastic activities such as running, hopping and jumping. It has been suggested that decreased tendon stiffness may negatively affect performance by increasing the time to stretch the series elastic component (Wang et al., 2012). Hysteresis is relevant to the efficiency of movement, power output in the stretch-shortening cycle and thermal stress within the tendon (Wang et al., 2012). Less hysteresis results in a lower level of dissipated energy and a greater return of stored energy.

(a)   (b)
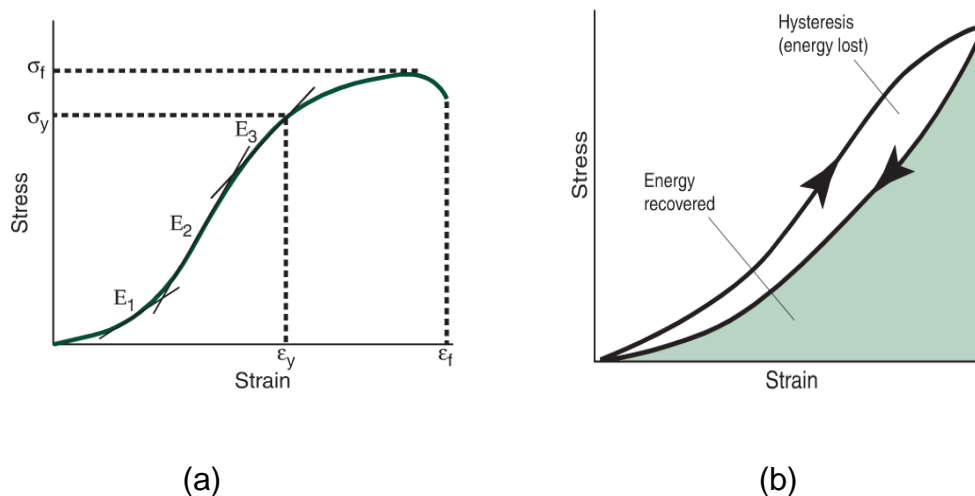
Figure 2.3 (a) A general stress-strain curve of a viscoelastic material and (b) a stress-strain curve where the energy recovered is less than the energy stored (Hamill & Knutzen, 2009).

2.3.2 Hill muscle model – the musculotendinous unit

The ability for a musculotendinous unit to tolerate different task difficulties, including speed and incline is dependent on the interaction between the muscle's

contractile and elastic components. The triceps surae and the Achilles tendon interaction can be described by an adapted version of the Hill muscle model (Figure 2.4) (Hamill & Knutzen, 2009). The Hill muscle model is comprised of a contractile component (i.e. the gastrocnemius and soleus muscles forming the triceps surae), a parallel elastic component (i.e. the surrounding connective tissue) and a series elastic component (i.e. the Achilles tendon). Running and hopping on an inclined surface results in the Achilles tendon being elongated to an even greater extent than during the same activity on a level surface in order to generate the propulsive forces for motion (Kannas, Kellis, & Amiridis, 2011; Lichtwark & Wilson, 2006) (Figure 2.5). Therefore incline loading has the potential to place greater demands on the Achilles tendon than level loading.

(a)   (b)
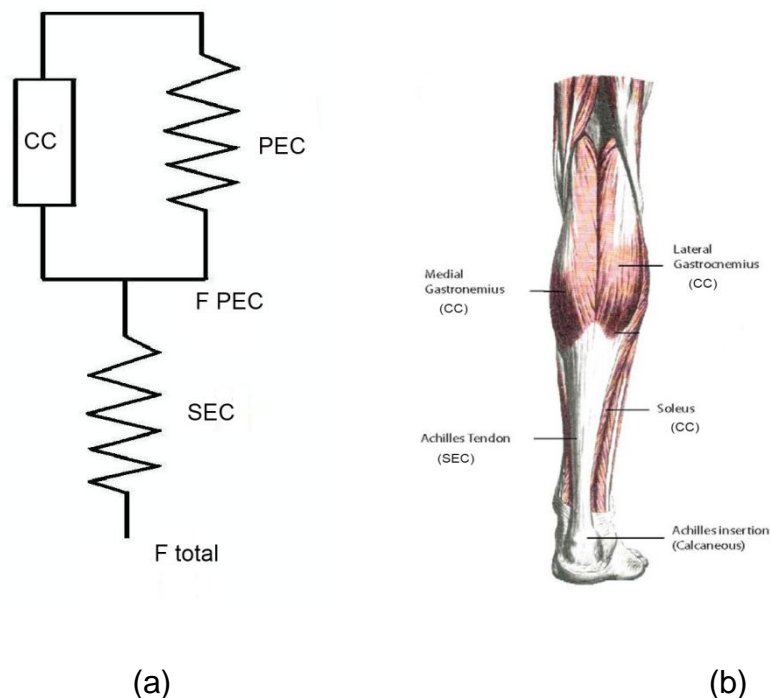
<u>Figure 2.4</u> (a)Adapted Hill muscle model (Hamill & Knutzen, 2009) where PEC is the parallel elastic component; CC is the contractile component; SEC is the series elastic component; F total is force total; and F PEC is the force of parallel elastic component. (b) The Hill type muscle model represented by the triceps surae (CC)

and Achilles tendon (SEC) musculotendinous unit (the PEC is made up of the surrounding connective tissue) (modified from Lichtwark & Wilson, 2005).
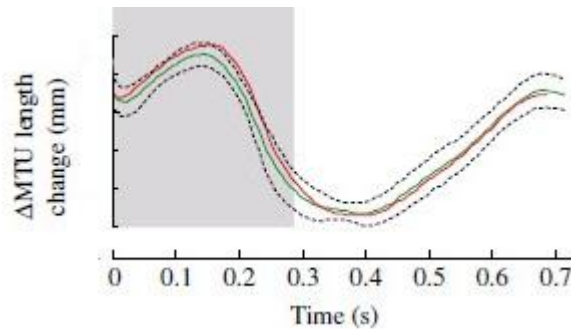
<u>Figure 2.5</u> Changes in muscle-tendon unit (MTU) length during running (green = level, red = uphill). Shaded area represents the stance phase (modified from Lichwark et al., 2006).

2.4 Achilles tendon function

The Achilles tendon behaves as a powerful spring during running and bounding activities, such as running and hopping (Farley et al., 1991). The Achilles tendon stores energy during the loading phase of gait before returning that energy as the tendon recoils during propulsion (Farley, Glasheen, & McMahon, 1993). The Achilles tendon is subjected to loads as high as 9 kN, which is approximately 12.5 times an individual's body weight during running (Komi, Fukashiro, & Jarvinen, 1992). During running and hopping the Achilles tendon is the prime contributor to the elastic energy in the musculotendinous unit as changes in the length of the triceps surae muscle are relatively minimal (concentric or isometric contraction) as compared to the change in length of the tendon (Hof, Van Zandwijk, & Bobbert, 2002; Lichtwark & Wilson, 2005; Novacheck, 1998; Roberts, Marsh, Weyand, & Taylor, 1997). When switching from walking to running there is increased recoil from the Achilles tendon,

reducing the demand on the muscle fascicles to generate increased force (Farris & Sawicki, 2012). Through its spring like properties, the tendon contributes approximately 35% to the mechanical energy for each stride during running (Farley et al., 1991; Ker et al., 1987).

2.5 Pathology of the Achilles tendon

In a sport such as long distance running, load accumulates over time (day, week, month, year). High repetitive loading is believed to be one of the main pathological stimuli causing AT (Silbernagel et al., 2006). With repetitive mechanical overloading of the Achilles tendon, there is potential to induce non-inflammatory pathology and degeneration (Khan & Cook, 2003). Histological change of a pathologic tendon described by Alfredson and Cook (2007) is characterised by four particular features, (1) increase in cell numbers, (2) increase in ground substance, (3) loss of characteristic hierarchical collagen structure and (4) neovascularisation.

2.5.1 Modelling tendon pathology

A continuum model has been developed by Cook and Purdam (2009) to describe the pathogenesis of tendon pathology (Figure 2.6). This pathology model hypothesises that tendons can transition forwards or backwards along the continuum, however they are unlikely to be reversible when they have become degenerative (Cook & Purdam, 2009). This has been observed in a number of studies for both the Achilles and patellar tendons. For example, Fredberg and Bolvig (2002) identified eleven tendons with abnormal imaging at the start of a soccer season. Four of the tendons (36%) reversed to normal on imaging at the end of the season. It was

theorised that pain is not caused by the structural changes in the tendon, but from a biochemical cause and/or sensitized cells (Cook & Purdam, 2009). It is unclear why Achilles tendons that are morphologically pathologic on US are not always painful (Cook, Khan, Kiss, Coleman, & Griffith, 2001; Rio et al., 2014). This suggests that the aetiology of Achilles tendon pain is different to the cause of pathology. However, given the presence of both tendon pathology and pain in an injured running population it is likely that there is a degree of commonality to the cause of both of these in AT.
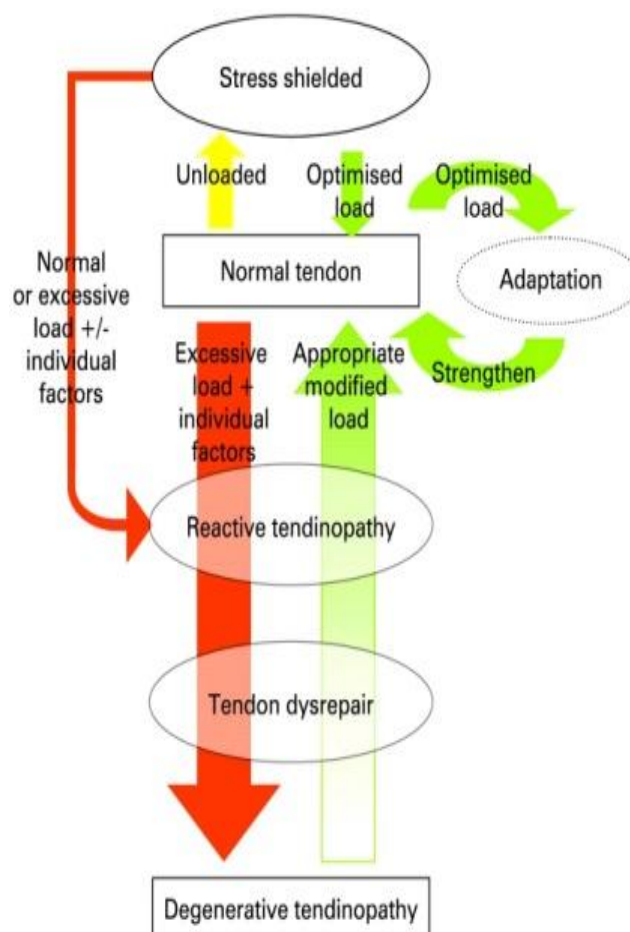
Figure 2.6 Tendon pathology continuum (from Cook & Purdam, 2009).

2.5.2 Imaging tendon pathology

The pathology and morphology of a tendon is determined by visualising the tendon with ultrasound (US) or magnetic resonance imaging (MRI). Ultrasonography can be used to confirm the diagnosis of AT, evaluate the pathology, assess thickness, hypoechogenicity and vascularisation, and is inexpensive and readily available (Cook, Khan, & Purdam, 2002; Khan et al., 2003; Maffulli, Regine, Angelillo, & Capasso, 1987; Ohberg, Lorentzon, & Alfredson, 2001) (Figure 2.7).

(a)   (b)

(c)   (d)
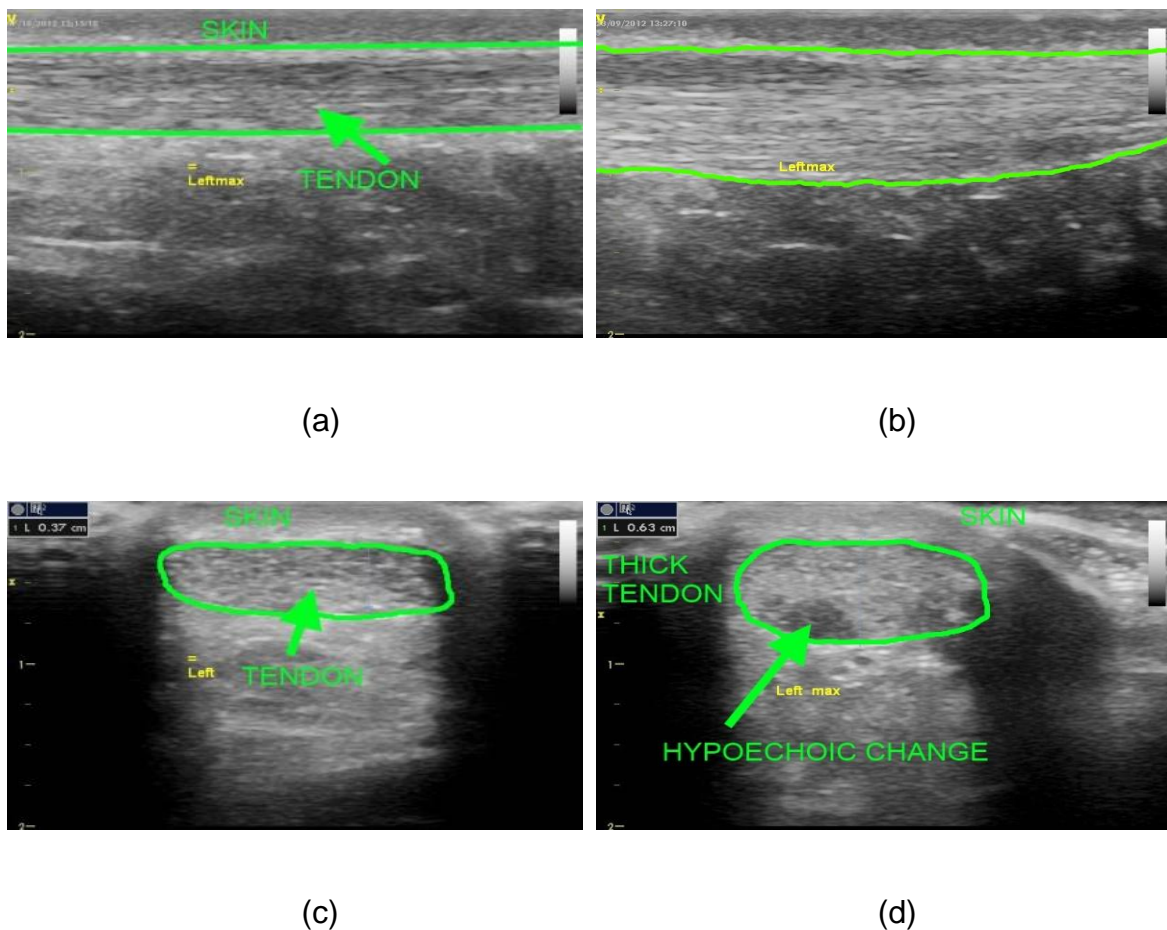
Figure 2.7 (a) Normal Achilles tendon (longitudinal view), (b) spindle shaped, thickened Achilles tendon (longitudinal view), (c) normal Achilles tendon (transverse view), and (d) Achilles tendon with a hypoechoic region marked by the arrow (transverse view).

Ultrasound imaging allows for assessment of tendon structure by transmitting sound waves from a probe into the underlying tissue and these sound waves are then reflected back as echoes. Tissue that reflects the soundwaves back strongly such as the collagen in tendons provides a brighter image, whilst degenerative changes in the matrix of the tendon do not reflect the echoes back as effectively and the image appears darker (Archambault et al., 1998). Ultrasound imaging of a normal healthy tendon reflects a homogenous echo texture with parallel margins (Archambault et al., 1998; Cook & Purdam, 2009) (Figure 2.7a). A reduction in reflection of the ultrasound waves due to changes in the matrix surrounding the collagen, results in hypoechoic regions that are identified in a pathologic tendon (Archambault et al., 1998; Cook & Purdam, 2009) (Figure 2.7d).

Reporting of US findings is user dependant, that is, the appearance of the US has to be interpreted. Therefore adequate training and experience is essential for accurate diagnosis of both normal and pathologic Achilles tendons. A common issue with US imaging is anisotropy (Smith & Finnoff, 2009). If an US probe is not positioned perpendicular to the structure of interest, the sound wave will not reflect back towards the probe. The reflection will be directed obliquely away from the probe and the desired structure will appear dark. The risk is that the dark region may be misinterpreted as hypoechoic and consequently the tendon incorrectly identified as pathologic (Filippucci et al., 2006).

2.6 Classification of Achilles tendinopathy

There are two classifications of AT, insertional (at the attachment to the calcaneus) and mid-portion (2-6cm from the calcaneal insertion). Mid-portion AT presents more frequently than insertional AT (Jarvinen et al., 2005; Kvist, 1994; Paavola et al., 2002), therefore mid-portion AT was selected for this research. Typically an athlete with AT presents with a common clinical pattern, including tenderness on palpation, morning stiffness and/or pain, and pain that progressively increases over weeks or months with exercise (Cook et al., 2002; Kountouris & Cook, 2007). Specifically, the progression of AT symptoms with exercise are (1) pain when commencing exercise that alleviates as the session progresses, which can progress to (2) ongoing pain during exercise, and then this may lead to (3) the cessation of activity (Kountouris & Cook, 2007). Running athletes with AT ultimately have to decrease volume, frequency and intensity of their training, often resulting in decreased performance (Cook & Purdam, 2014).

2.7 Risk and associated factors for Achilles tendon pathology

Some runners appear predisposed to tendinopathy whilst others can tolerate high load without ever developing tendinopathy. Treatment for AT can be costly and time consuming. Therefore identifying associated and risk factors may help develop injury prevention programs. Development of AT is often multifactorial, and both intrinsic and extrinsic risk and associated factors may not only affect the development of tendinopathy but also response to treatment. An associated factor is identified in a cross-sectional study where a difference is observed between two groups (Cowan et al., 2004). These factors are unable to be classified as predisposing or causative

factors due to the design (Gabbe et al., 2006). A prospective study design is required to identify risk factors (Gabbe et al., 2006).

Intrinsically, AT typically affects more men than women in both a physically active (Astrom, 1998) and a general non athletic population (Gaida et al., 2010). Oestrogen has been shown to protect the tendon from pathology in women (Cook, Bass, & Black, 2007). Oestrogen is not only linked to the difference in prevalence of AT between women and men, but also results in a lower incidence of AT in younger women compared to post-menopausal women (Cook et al., 2007).

Adiposity (high central adipose tissue accumulation measured with waist measurements) has been identified as a modifiable associated factor for the development of Achilles tendon pathology in men (Gaida et al., 2010). For weight bearing tendons like the Achilles tendon, it may be assumed that increased loading may be responsible for the association. However, an increase in upper limb tendinopathy is also an associated factor of adiposity suggesting a metabolic syndrome may be responsible (Gaida, Cook, & Bass, 2008). This is of particular interest given physical activity such as running has health benefits including weight loss. Greater concern exists if a runner with higher levels of central weight distribution develops symptoms and is unable to exercise, increasing weight gain and hence compounding a potential associated factor.

Older athletes display an increase in AT symptom development (Di Caprio et al., 2010; Paavola et al., 2002). Older age is also associated with greater life time load, which has been demonstrated to be a factor related to an increase in tendon cross-sectional area (Magnussen et al., 2003) and likelihood of tendon pathology in asymptomatic individuals (Gaida et al., 2010).

Structural abnormalities identified on ultrasound imaging have been identified as a risk factor for the development of pain (Comin et al., 2013). Focal hypoechoic regions rather than thickening of the tendon appears to be the structural morphological change that is predominantly predictive of an increased risk of tendon pain (Comin et al., 2013).

Biomechanical factors including foot biomechanics and decreased ankle dorsiflexion (DF) are regularly suggested as risk factors for the development of AT (Cook et al., 2002; Jarvinen et al., 2005; Kountouris & Cook, 2007). Foot biomechanics, particularly excess pronation has been suggested as a contributor to the repetitive overloading and consequential pathology and pain in the Achilles tendon (Clement et al., 1984). Pronation is a tri-planar movement that incorporates a combination of rearfoot eversion, DF and abduction of the foot (Wyndow et al., 2010). It has been suggested that excessive rear foot sub-talar joint motion causes a "whipping" action of the Achilles tendon resulting in micro-tearing of the tendon (Clement et al., 1984; Donoghue et al., 2008; Maffulli et al., 2004; McCrory et al., 1999). Donohue et al., (2008) identified that injured athletes with AT involved in running-related sports displayed greater rearfoot eversion range of movement (ROM) compared with healthy controls during running. McCrory et al. (1999) reported an increase in rearfoot inversion at initial contact, increased pronation and a greater rate of pronation during running. Despite these findings, the proposed "whipping" action that occurs has yet to be confirmed or refuted by research and the role of foot biomechanics remains unclear in the development of AT. In addition, it is difficult to measure foot biomechanics such as pronation during dynamic activities (Wyndow et al., 2010). This previous research on foot biomechanics has not explored the spring-like behaviour of the Achilles tendon.

Ankle joint DF may be another risk factor for AT. A reduction in DF may limit the capacity of the triceps surae to absorb load and may therefore result in greater loading rates (Wyndow et al., 2010). Alternatively, an increase in DF range may prolong loads on the Achilles tendon over a larger range. Although there is some support for these theories, there are conflicting findings in the literature. For example, McCrory et al. (1999) and Hein et al., (2014) found no difference with ankle DF in runners with AT. McCrory et al. (1999) assessed DF in supine but did not specify knee position, whereas Hein et al., (2014) assessed DF with the knee flexed. In contrast, Kaufmann et al., (1999) reported reduced DF when tested with the knee in an extended position in Navy SEAL candidates who developed AT, whereas Mahieu et al. (2006), who assessed ankle DF with both knee extension and flexion, reported the opposite with AT injured military recruits having greater ROM. Thus far, most research investigating AT has typically had non-weight bearing measurements of DF assessed (Hein et al., 2014; Kaufman et al., 1999; Mahieu et al., 2006; McCrory et al., 1999) and these non-weight bearing measurements are typically less reliable than the weight bearing methods (Bennell, Talbot, Wajswelner, Techovanich, & Kelly, 1998). Measurement of ankle DF in weight bearing is a common clinical and research test that has been shown to be reliable, however some inconsistencies with findings have been identified (Bennell et al., 1998; Munteanu, Strawhorn, Landorf, Bird, & Murley, 2009) (Figure 2.8). In addition there may be potential for over-estimation of ankle DF measurements if the heel lifts from the ground.

(a)   (b)
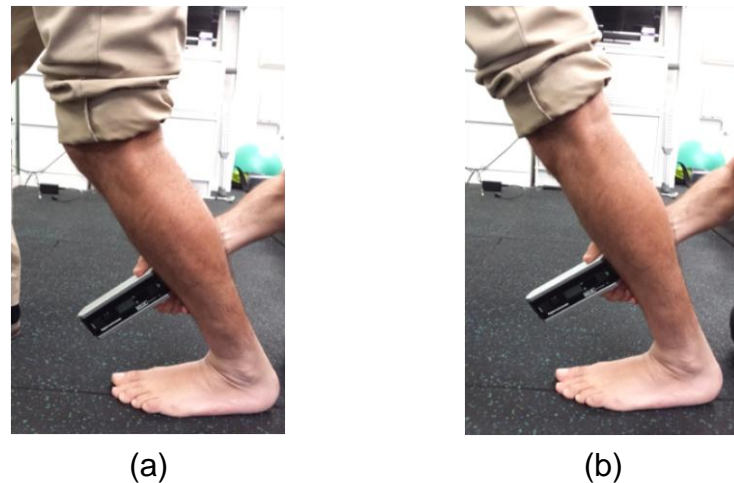
Figure 2.8 Measurement of ankle dorsiflexion in weight bearing where (a) is the standing lunge test (left leg) and (b) is the straight knee dorsiflexion (left leg).

A number of extrinsic risk factors have been associated with AT including training errors such as overload with a sudden increase in training mileage or intensity, a change of terrain (hill running or surface), an increase in interval training, a solitary intense run, years of running or a combination of these factors (Di Caprio et al., 2010; Knobloch et al., 2008; McCrory et al., 1999; Rompe et al., 2008; Rosager et al., 2002; Schepsis et al., 2002; Wyndow et al., 2010). Other extrinsic factors that compromise the muscle-tendon unit include running on soft surfaces such as sand (Knobloch et al., 2008; McCrory et al., 1999), worn footwear or footwear that are soft and has unstable heel cups (Wyndow et al., 2010) and fatigue (Komi, 2000; Paavola et al., 2002).

Overloading the Achilles tendon may expose the tendon to a stimulus that results in the development of pathology. Long term load exposure as a consequence of greater years of running is associated with AT (Knobloch et al., 2008) and increased running mileage is associated with a larger cross-sectional area of the

Achilles tendon (Rosager et al., 2002), both highlighting the influence of cumulative load of the Achilles tendon.

2.8 Asymptomatic tendon pathology

The association between symptoms and pathology seen on imaging is unclear. Athletes with asymptomatic tendon pathology have the typical pathological imaging findings described previously, however the individual reports no pain or stiffness despite prolonged exercise and activity. Degenerative tendon pathology, although asymptomatic, may result in regions of the tendon that are less tolerant to high load elastic tasks, with the remaining healthy tissue exposed to higher loads (Cook & Purdam, 2014).

Research exploring asymptomatic tendon pathology prior to symptom development is of clinical interest. Thus far, the presence of asymptomatic tendon pathology in the patellar tendon on imaging has been identified as a risk factor in a young basketball population (Cook et al., 2001; Cook, Khan, Kiss, Purdam, & Griffiths, 2000). Recent findings by Comin et al. (2013) identified that the presence of structural abnormalities, particularly hypoechoic regions within the tendon increased the risk of developing tendon pain. Fredberg and Bolvig (2002) showed that 45% of elite soccer players with asymptomatic Achilles tendon pathology at the start of the season developed clinical symptoms during the season. Several other studies have also shown the presence of asymptomatic Achilles tendon pathology and/or asymptomatic patellar tendon pathology in athletes participating in other sports, including volleyball (Kulig et al., 2013), soccer (Fredberg, Bolvig, & Andersen, 2008), basketball (Cook et al., 1998; Cook et al., 2001; Cook, Khan, Kiss, & Griffith, 2000),

badminton (Malliaras, Voss, Garau, Richards, & Maffulli, 2012), ballet (Comin et al., 2013), and gymnastics (Emerson, Morrisey, Perry, & Jalan, 2010). To date, no research has investigated the prevalence of asymptomatic tendon pathology in a distance running population. Identifying athletes at risk who are then regularly exposed to high tendon load activities, such as distance running may be an important injury prevention strategy as these athletes may become symptomatic over time.

2.9 Running biomechanics

Running is a complex form of a bounding gait. In a bounding gait the muscles, tendons and ligaments in the lower limb store and release elastic energy as an athlete travels along the ground in a spring like manner (Farley & Morgenroth, 1999). Biomechanical study of bounding gaits provides insight into locomotion mechanisms in healthy and injured populations.

During the running gait cycle there is a stance phase and a flight phase. Running gait is differentiated from walking by the presence of a flight phase (both feet are off the ground) between each foot strike. In contrast, walking has a double stance phase (both feet are on the ground). Typically as speed increases during running, initial ground contact shifts from the heel to the forefoot (Novacheck, 1998). The change in footstrike is observed when comparing running to sprinting. Only a small percentage of distance runners are natural forefoot strikers at initial contact, with most distance runners being rearfoot strikers, and some also strike the ground with a midfoot running technique (Hasegawa, Yamuchi, & Kraemer, 2007; Larson et al., 2011) (Figure 2.9). A rearfoot striker will make initial contact with their heel, a midfoot striker will make simultaneous contact with the heel and ball of their foot,

whereas a forefoot striker will contact the ground with the ball of their foot prior to their heel contacting the ground (Kubo, Miyazaki, Tanaka, Shimoju, & Tsunoda, 2015). As each foot impacts the ground, a vertical ground reaction force is generated. The leg compresses from initial contact until midstance to absorb this force and then the leg lengthens following midstance during the propulsion phase. The ratio of the body's centre of mass (COM) vertical displacement to the peak vertical ground reaction force (typically at mid-stance) is a measure of active, vertical musculoskeletal stiffness. Consequently, running has frequently been modelled and investigated by the spring-mass model to assess musculoskeletal stiffness (Farley et al., 1993; Farley & Gonzalez, 1996; Ferris et al., 1998; Hamill, Moses, & Seay, 2009).

Forefoot   Midfoot   Rearfoot

Figure 2.9 Footstrike patterns of distance runners (diagram modified from Larson et al., 2011)

Similar to running, hopping is a spring-like movement. As the foot contacts the ground, the leg begins to compress as the joints flex until midstance and then during rebound the leg lengthens as the joints extend. Some notable differences are apparent between the two tasks of running and hopping. At initial contact during running foot strike may be rearfoot, midfoot or forefoot, whereas during hopping the

forefoot typically strikes the ground at initial contact. Consequently footstrike will load different structures in the lower limb preferentially.

2.10 Leg stiffness and the spring-mass model

"Stiffness" is the main mechanical parameter studied when analysing the leg spring. Some researchers (Latash & Zatsiorsky, 1993; Shamaei, Sawicki, & Dollar, 2013a; Shamaei, Sawicki, & Dollar, 2013b) refer to the term "quasi-stiffness" to explore the leg spring, however this term is not commonly used in the literature. Running, hopping and trotting animals all ambulate in a spring-like manner (Farley & Morgenroth, 1999). Gait can be modelled using a simple spring-mass model (Figure 2.10), consisting of a single linear "leg spring" and body mass (Farley & Morgenroth, 1999). The spring-mass model is a biomechanical model often utilised to explore elastic energy storage and release. Hopping on the spot is commonly utilised for testing the spring-mass model because it has similar mechanics to running and is a much simpler test to use (Ferris & Farley, 1997).
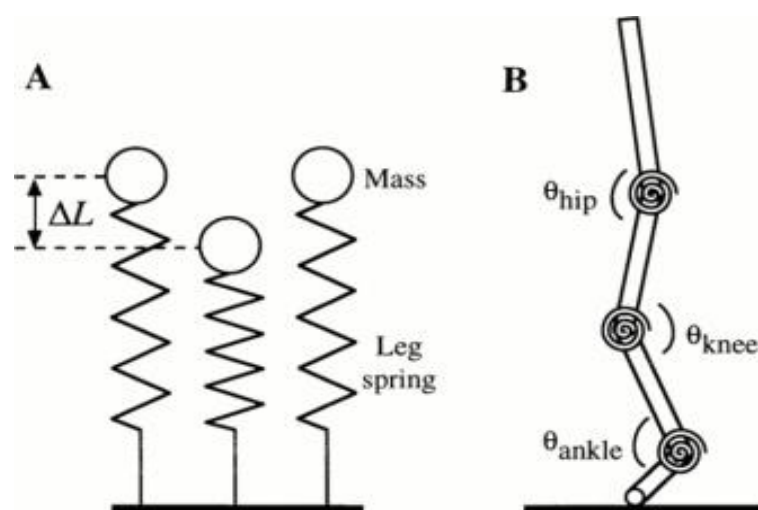
Figure 2.10 (A) Spring-mass model for hopping and (B) multi-joint torsional spring model of the joints (Farley, Houdijk, Van Strien, & Louie, 1998).

2.10.1 Leg and joint stiffness during hopping

Mechanical stiffness during hopping is calculated from the ratio of the peak vertical ground reaction force and the displacement of the centre of mass (COM) at the middle of the ground contact phase (Farley et al., 1998) (Figure 2.10A). Vertical leg stiffness, k_{Vert}, is calculated from force plate data using peak vertical ground reaction force and COM displacement during the compression phase of the hop. From initial ground contact to maximum leg compression (mid-stance) the calculation follows:

$$k_{Vert} = \frac{\text{Force (peak)}}{\text{COM (maximum displacemet of COM)}}$$

Maximum displacement of COM is obtained by double integration of the vertical acceleration with respect to time (Brughelli & Cronin, 2008; Divert, Baur, Mornieux, Mayer, & Belli, 2005; Hobara, Kimura, et al., 2010)

Leg stiffness depends on the torsional (angular) stiffness of the joints (Farley et al., 1998) (Figure 2.10B). If the joints of the lower limb (ankle, knee and hip) undergo smaller angular displacements during the ground contact phase, the joints are stiffer resulting in reduced leg compression and increased leg stiffness (Farley & Morgenroth, 1999). Joint stiffness, k_{Joint} is calculated using the torsional spring model. In this model it is assumed that four rigid segments (foot, shank, thigh and head-arms-trunk) are interconnected with torsional joint springs of the hip, knee and ankle (Farley et al., 1998). Therefore, joint stiffness are calculated as,

$$k_{Joint} = \frac{\text{Moment}_{Joint}}{\theta_{Joint}}$$

where moment$_{joint}$ and θ_{joint} are the changes in joint moment and the angular displacement at each joint from initial contact to mid-stance, respectively. The moment about a joint is determined by the magnitude of the applied force and the perpendicular distance from the line of action of the force to the joint axis of rotation (Hamill & Knutzen, 2009). The joint moments are influenced positively by both the magnitude of force applied about a joint axis and by an increased moment arm. A moment arm is the perpendicular distance of the joint axis from the force (Figure 2.11). Consequently, if the force is further from the joint axis, the moment arm is larger resulting in a greater joint moment. Alternatively if the force is larger with a constant moment arm, the joint moment will also be greater.
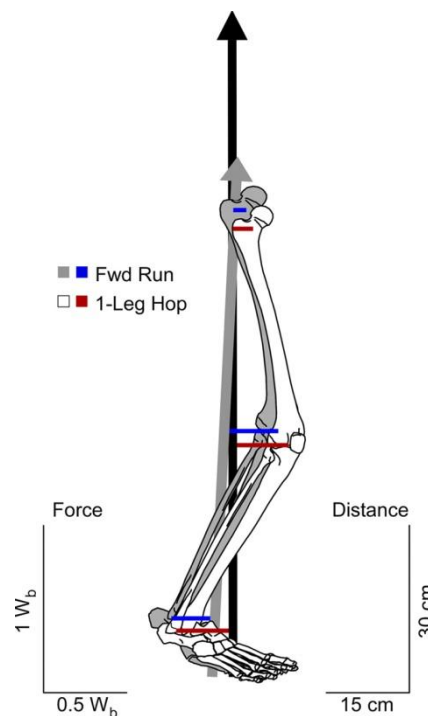
Figure 2.11 Moment arms for hopping and running (hopping: red bars and white limb, running: blue bars and grey limb). The moment arms are the distances from the joint axis to the ground reaction force (black arrow) during single leg hopping (Weyand, Sandell, Prime, & Bundle, 2010).

2.10.2 Leg stiffness regulation during running and hopping

The leg is comprised of a number of interconnecting joints that are influenced by a number of strategies to adjust leg stiffness (Farley & Morgenroth, 1999). Increased leg stiffness is thought to be beneficial for sporting performance (Butler et al., 2003) however in rebound jump performance, high levels of stiffness may reduce performance (Walshe & Wilson, 1997). These levels of stiffness have thus far been associated with increased or reduced performance, however they may not necessarily cause the performance change. It is important to consider how leg and joint stiffness is influenced by running and hopping.

During running, stiffness increases with increased stride frequency and reduced ground contact times (Farley & Gonzalez, 1996; Morin, Samozino, Zameziati, & Belli, 2007), running speed (Farley et al., 1993), body mass (Farley et al., 1993), whilst it is reduced by fatigue (Morin, Jeannin, Chevallier, & Belli, 2006) and wearing footwear (De Wit, De Clerq, & Aerts, 2000; Divert et al., 2005). Running technique can also influence stiffness. Forefoot striking during running increases knee joint stiffness whilst the ankle joint becomes more compliant (Hamill, Gruber, & Derrick, 2014). Conversely, rearfoot striking during running increases ankle joint stiffness and results in a more compliant knee joint (Hamill et al., 2014). Forefoot running also increases the load on the Achilles tendon in comparison to rearfoot runners (Almonroeder et al., 2013). In comparison to forefoot running, rearfoot running will typically increase the load on the patellofemoral joint (Kulmala, Avela, Pasanen, & Parkkari, 2013). Due to the increase in load on the Achilles tendon runners with or at risk of AT should be very cautious with adopting forefoot running due to the potential for exacerbating their condition.

During hopping, leg stiffness is influenced by a number of factors. During initial ground contact if the limb segments are configured in a more extended position (i.e. less flexed) leg stiffness will be increased (Farley et al., 1998; Hobara, Inoue, Omuro, Muraoka, & Kanosue, 2011; Moritz, Greene, & Farley, 2004). Hopping performance is modulated primarily by the ankle with increased muscle activation of the triceps surae (Farley & Morgenroth, 1999; Hobara, Kanosue, & Suzuki, 2007; Kuitunen et al., 2011). In addition pre-activation of the triceps surae including the short-latency stretch reflex that occurs at the point of contact enhances the musculoskeletal system to store and release energy (Hobara et al., 2007; Kuitunen et al., 2011). This pre-activation has been reported to contribute to leg and joint stiffness adjustments (Arampatzis, Schade, Walsh, & Bruggemann, 2001; Kuitunen et al., 2011). Consequently, the influence Achilles tendon pathology and AT has on the limb configuration and pre-activation of the triceps surae that may influence leg and joint stiffness is largely unknown and further research is warranted.

Leg stiffness also increases with higher hopping frequencies (Austin, Tiberio, & Garrett, 2002; Farley et al., 1991), wearing footwear (Bishop, Fiolkowski, Conrad, Brunt, & Horodyski, 2006) and also leg stiffness increases have been associated with increased strength (Hobara et al., 2008) and endurance training (Hobara, Kimura, et al., 2010). This indicates that conditioning of the lower limb may result in an increase in active limb stiffness. In addition, gender also influences leg stiffness during hopping, with men typically having higher levels of stiffness than women (Granata, Padua, & Wilson, 2002). Although leg stiffness has been suggested as an important factor in sporting performance, it is also understood that too much or too little may increase risk of injury (Butler et al., 2003).

2.10.3 Leg stiffness and injury

Higher levels of stiffness have been suggested to be associated with bony injuries, whereas lower levels of stiffness may be associated with soft tissue injuries (Butler et al., 2003; Williams, Davis, Shultz, Hamill, & Buchanan, 2004; Willliams, McClay, & Hamill, 2001). However the association between injury and stiffness has not been well developed. Although stiffness has not been well recognized as a risk factor, it is an area of increasing clinical interest due to the modifiable nature of leg stiffness, and its potential as a risk factor for injury. Improved understanding of alterations in leg stiffness may aid in injury prevention strategies for athletes (Hobara et al., 2008; Pruyn et al., 2012) and may also assist in the management of injuries. For example, it has been suggested that women are at a greater risk of anterior cruciate ligament (ACL) injury due to lower levels of leg stiffness (Granata et al., 2002).

Differences in leg stiffness do not demonstrate where different strategies in the kinetic leg chain occur. For examples, in athletes with patellar tendinopathy when compared to uninjured controls, a change in lower limb strategies during hopping has been identified with a shift to increased load at the hip and a reduction at the knee (Souza, Arya, Pollard, Salem, & Kulig, 2010). In addition during running, runners with low back pain and runners with tibial stress fractures have higher levels of knee stiffness (Hamill et al., 2009; Milner, Hamill, & Davis, 2007). During hopping tests female gymnasts with a previous injury history have been observed to have either higher or lower levels of ankle stiffness (Bradshaw & Hume, 2012). Therefore as highlighted by these findings when investigating leg stiffness as an associated factor of an injury, it is important to also consider joint stiffness because a difference in leg stiffness may not be identified as the stiffness of one joint may increase another joint

may decrease. These research findings are retrospective, therefore the stiffness may either be related to injury or alternatively it may be as a consequence of injury. Thus, a direct link is difficult to make between stiffness and injury.

2.10.4 Leg stiffness on level compared with inclined surfaces

Lower limb stiffness regulation has been primarily explored on a level surface with limited research exploring the impact of an incline surface on the leg spring. Incline hopping in comparison to level hopping results in loading of the ankle in greater ranges of ankle DF, and increased activation of the triceps surae with increased elongation of the Achilles tendon (Kannas et al., 2011). This may result in triceps surae functioning in a more optimal range as well as having greater levels of elastic storage and recoil of elastic energy (Kannas et al., 2011). However, their findings identified that incline hopping does not influence vertical leg stiffness (Kannas et al., 2011). The alteration observed in ankle function may influence joint stiffness regulation, a biomechanical variable that has yet to be explored during incline hopping. It is also unknown the influence of tendon pathology on stiffness regulation during incline hopping where increased elongation of the tendon occurs.

2.10.5 Tendon pathology and leg stiffness

The Achilles tendon is one of the main components of the leg spring during elastic tasks. Arya and Kulig (2010), Child et al. (2010) and Wang et al. (2012) all observed a reduction in tendon stiffness in mid-portion AT with an increase in strain occurring at the tendon-aponeurosis (Figure 2.12). Child et al. (2010) suggested that the pathologic changes to a tendon may alter the mechanical properties of the

tendon resulting in reduced stiffness or alternatively the increased compliance may be involved in the development of AT, however this remains unknown. Another factor that may reduce the capacity of the tendon is periods of unloading. Unloading may follow not only a break in training but some runners may be forced to unload due to pain. Unloading results in a reduction of mechanical stiffness and similar changes to the tendon matrix as overloading the tendon (Cook & Purdam, 2009). In addition to the change in stiffness, Wang et al., (2012) observed higher levels of hysteresis with AT, suggesting a reduced capacity to return stored energy. Increased hysteresis in combination with reduced tendon stiffness has the potential to reduce the elastic energy storage and increase the energy wasted in AT (Wang et al., 2012). The effect these alterations may have on lower limb mechanics during functional stretch-shortening cycle tasks is largely unknown. Therefore investigating the impact of Achilles tendon pathology on the leg's spring-like characteristics dynamically, may increase our understanding of biomechanical factors associated with tendon overload and pathology. Improved knowledge may ultimately aid in injury prevention and rehabilitation.
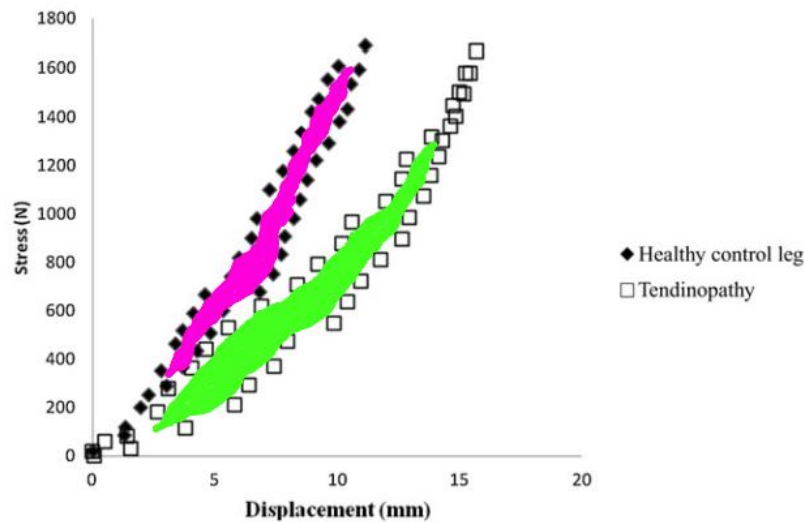
Figure 2.12 A stress-strain curve of the Achilles tendon for a patient with a normal tendon and a pathologic tendon (from Wang et al., (2012). The highlighted area within the curve (pink = healthy, green = AT) is hysteresis, which indicates the amount of energy loss.

Analysis of the impact that a pathological or painful Achilles tendon has on lower limb biomechanics requires an assessment protocol that challenges the elastic properties of the tendon. A simple test for assessing the elastic function of the Achilles tendon is hopping. Hopping allows for the assessment of musculoskeletal stiffness to analyse for potential changes that occur in the lower limb in the presence of Achilles tendon pathology and pain. Only limited research has examined the impact of Achilles tendon pathology on leg stiffness. Maquirriain (2012) identified that a reduction in leg stiffness was an associated factor of AT. His study compared the symptomatic leg and asymptomatic leg in individuals with unilateral AT and observed a difference in vertical leg stiffness between limbs. He suggested that the reduced leg stiffness was a consequence of an increase in ankle compliance however he had not assessed that as part of his research.

Pain is a defining symptom of AT (Rio et al., 2014), and the tendon pain causes muscle inhibition as well as a change in neuromuscular performance (Henrikson, Aaboe, Graven-Nielson, Bliddal, & Langberg, 2011). Pain compromises function and performance in AT (Cook & Purdam, 2014). Pain may also induce compensatory movement patterns that affect lower limb kinetic chain function and are likely to differ for each athlete. Some may compensate by transferring greater loads to other joints in the kinetic chain or alternatively modify loading patterns to the least or non-symptomatic leg. Henrikson et al., (2011) observed that when inducing Achilles tendon pain, motor inhibition occurs similar to motor responses that occur during fatigue. Unfortunately, Henrikson et al., (2011) did not use a stretch-shortening cycle task, however they were still able to highlight the impact of pain. Therefore assessing movement patterns in athletes with AT is complicated not only due to the effect of pain, muscle wasting and weakness, but potentially due to the structural tendon degeneration.

Altered lower limb strategies have been observed in athletes with asymptomatic patellar tendon pathology (Edwards et al., 2010). Edwards et al. (2010) identified different loading patterns in jumping athletes with asymptomatic patellar tendon pathology. They observed increased knee flexion, as well as hip extension rather than hip flexion during a horizontal landing task as compared to their control population. These loading strategies suggest a difference in load distribution between the hip, knee and ankle. It remains unclear as to the impact of Achilles tendon pathology, both symptomatic and asymptomatic on the alteration to lower limb strategies during challenging stretch-shortening cycle tasks, such as hopping on the flat or incline. Investigating biomechanical variables that are risk factors for an injury is very difficult, as it requires a prospective study design. Prospective study

designs are expensive due to the number of participants required to identify risk factors. A further complexity in the Achilles tendon is that there is often pathology without symptoms (Chapter 4). It is unknown whether Achilles tendon pathology may alter lower limb biomechanics or that symptoms (i.e. pain) are required to change the mechanics of the lower limb during high Achilles tendon loading activities. Previous studies exploring AT have examined biomechanics in symptomatic tendinopathy (Azevedo, Lambert, Vaughan, O'Conner, & Schwellnus, 2009; Child et al., 2010; Maquirriain, 2012). Further research is required to explore biomechanical factors associated with Achilles tendon pathology with no previous history of pain.

Therefore the purpose of this study was to explore the stiffness regulation (leg and joint stiffness) and joint interaction in the lower limb during level and incline hopping in distance runners with Achilles tendon pathology, both symptomatic and asymptomatic.

2.11 Summary

Achilles tendinopathy is a complex condition with many potential causative factors. Therefore treatment may be difficult, particularly given the condition is also renowned for its propensity for recurrence (Silbernagel, Thomee, Eriksson, & Karlsson, 2007b). The aim of clinical treatment is to reduce pain, regain full function of the Achilles tendon, return the athlete to their sport and minimise recurrence.

Research has identified that the presence of structural abnormalities in the Achilles tendon may predispose a runner to developing pain. Distance runners expose their Achilles tendons to regular bouts of high mechanical load that may

cause pathology. Distance runners who are at a high risk of developing tendon pathology and pain have yet to be investigated for the prevalence and associated factors of asymptomatic tendon pathology. Consequently, further research is required to examine distance runners..

Tendon pathology may not only be a risk factor for the development of symptoms, but it may change the mechanical properties of the tendon altering the mechanics of the lower limb during high tendon loading activities. In addition to pathology, previous or current Achilles tendon pain may affect motor control, such as the pre-activation of the triceps surae during hopping and running. The Achilles tendon is a very strong tendon that behaves like a spring during elastic activities by elongating to store energy, prior to rapid shortening as it recoils (e.g. running, hopping). Therefore tendon pathology and/or pain may compromise function and alter lower limb biomechanics. Identifying altered movement patterns that may occur in the presence of tendon pathology with and without pain aims to improve strategies for injury prevention and management.

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Chapter Three

A simple platform to improve accuracy of measuring ankle joint dorsiflexion whilst weight bearing

3.1 Aims and Hypotheses

The aims and hypotheses of this chapter were to:

- Develop a testing protocol using a novel platform that standardises ankle position to measure ankle dorsiflexion (DF)

- Determine if ankle DF range of motion (ROM) angles were different between the standard testing protocols and the platform. It was hypothesised that ankle DF angle would be smaller when using the platform.

- Determine the intra-rater reliability and the inter-rater measurement agreement for different raters when measuring ankle DF ROM using the standardised protocols and the platform, including the effect of experience on these measures. It was hypothesised that ankle DF measures would have high agreement between raters and, secondly, that reliability would be superior when using the platform compared to the standard protocol.

3.2 Introduction

Adequate range of ankle DF is required for basic activities of daily living such as walking, stair climbing and running. Further, a lack of DF has been reported as an independent risk factor for lower limb sporting injuries such as Achilles tendinopathy (Kaufman et al., 1999), patellar tendinopathy (Malliaras, Cook, & Kent, 2006) and plantar fasciitis (Riddle, Rulisic, Pidcoe, & Johnson, 2003). Restricted DF is also common following surgery, prolonged immobilisation and injury or surgery to the foot or ankle complex. Consequently assessment of DF has important clinical and research value.

Ankle DF is commonly assessed during athletic and clinical screening and also to assess the effectiveness of foot and ankle rehabilitation after an intervention. Clinically, such assessment is commonly performed using either the standing lunge test (Figure 3.1A) or a straight knee DF test (Figure 3.1B). The standing lunge test is done with the knee flexed and therefore it assesses the impact of joint restriction from bone, joint capsule and the soleus muscle (Munteanu et al., 2009). In contrast, the straight knee test assesses the addition of gastrocnemius muscle tightness on ankle DF (Munteanu et al., 2009). When combined these two tests provide clinicians and researchers with comprehensive insight into the soft tissue or joint restrictions that may limit ankle DF. The tests are inexpensive, time efficient and require minimal equipment (inclinometer). Additionally, the administration of the tests whilst the patient is weight bearing allows for increased torque compared to non-weight bearing methods. However, these weight-bearing DF tests are unable to separate talocrural DF from the combined ROM that includes subtalar and talar joint ROM (Bennell et al., 1998; Munteanu et al., 2009). Despite an inability to isolate talocrural DF, these

weight bearing tests better reflect the ROM required for activities of daily living (Bennell et al., 1998).

A B C
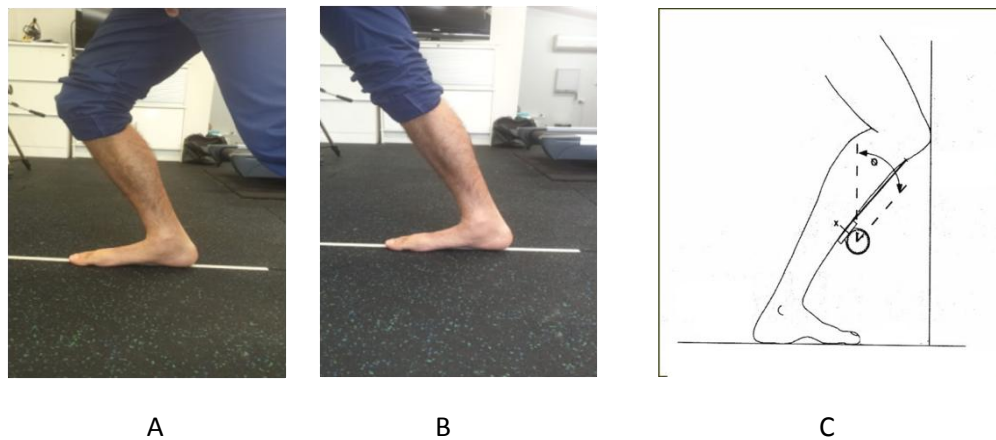
Figure 3.1 Ankle dorsiflexion, demonstrating (A) the standing lunge test (right leg), (B) straight knee dorsiflexion test (right leg) and (C) the placement of the standing lunge test inclinometer (modified from Bennell, et al. (1998))

The standing lunge test and straight knee DF tests have high inter-rater and intra-rater reliability for both experienced and inexperienced practitioners (Bennell et al., 1998; Munteanu et al., 2009). Munteanu et al. (2009) found that the reliability was higher in experienced compared to inexperienced practitioners. In contrast, Bennell et al. (1998) identified excellent inter-rater reliability regardless of the level of experience of the practitioner for the lunge test. However, despite the high level of reliability reported in the Bennell et al. (1998) study, they also found that one of the raters determined a significantly different (p<0.001) mean ankle DF angle when compared to the other three practitioners. Although both studies reported that the assessment of ankle DF was reliable, both measures appear to have some inconsistency in measurements between raters.

Measurement of DF requires a flat and level ground, with a straight line marked on the floor. The foot is placed over the line, ensuring the centre of the heel

and the second toe are on the line. The person is asked to lunge forward as far as possible with their knee and foot over the line and keep their heel on the ground, and measures of joint angle are then taken. Whilst the weight bearing DF test has been demonstrated to be reliable, one potential factor that may have contributed to the variability of values in the DF studies is that the participant being tested may not have maintained constant heel contact whilst the measurement was being recorded. As a result, we designed a custom built platform to allow for DF to be measured whilst ensuring heel contact. The platform provides a spring loaded mat under the heel that retracts if weight is lifted off the heel and ensures heel contact during testing, a possible cause for the inconsistencies noted in previous research.

The use of the platform may improve the consistency of measurements between assessors regardless of clinical or research experience. Consequently, the aim of this study was to evaluate the impact of the custom built platform on ankle DF measurement and to determine if ankle DF measurements were greater in the standard protocol due to the heel lifting from the ground. Inter-rater measurement agreement and intra-rater reliability and repeatability of the standing lunge and straight knee DF tests using the platform was also investigated.

3.3 Methods

3.3.1 Participants

Seventeen participants (9 men, 8 women), aged 19-28 years (mean = 21.4 ± 2.1 years) were recruited from the undergraduate student population within the School of Exercise Science, at the Australian Catholic University. Volunteers who

reported any acute or chronic lower limb musculoskeletal injury for which they had sought treatment in the previous six months were excluded from this study.

This study was approved by the Australian Catholic University Human Research Ethics Committee (Appendix A). Prior to testing, each volunteer was informed of the procedures, risks and benefits of the study before informed consent was attained (Appendix B).

3.3.2 Raters

Three raters were used for the study. At the time of this study Rater A was a physiotherapist with nine years of clinical experience, Rater B was a sports podiatrist with eleven years of clinical experience and Rater C was a final year Bachelor of Exercise Science student with no clinical experience. Prior to commencing the measurement protocol, each rater was instructed as to the appropriate technique and was provided with a 20 minute familiarisation session.

3.3.3 Protocol

3.3.3.1 Standing lunge test

The participants placed their foot on the ground with their foot aligned along a central line. The line passed through the centre of their heel and second toe. Then they were instructed to lunge forward, dorsiflexing their ankle as far as possible, whilst keeping their heel down in accordance with the knee to wall test described by

Bennell et al. (1998) (Figure 3.1A). The other leg was maintained in a self selected position to assist with balance.

3.3.3.2 Straight knee dorsiflexion

The participant placed their foot on the ground similarly to the standing lunge test and whilst keeping their knee straight. Then they leaned forwards until they could feel a maximal stretch in the back of their leg while ensuring they kept their heel in contact with the ground (Munteanu et al., 2009) (Figure 3.1B). The other leg was maintained in a self selected position to assist with balance.

3.3.3.3 Custom built platform

A platform was constructed using a 30 cm x 45 cm x 2 cm medium density fibre board (Figure 3.2). Four adjustable bolts were placed in each corner to allow for level calibration using a digital inclinometer (Baseline® Digital Inclinometer, Fabrication Enterprises, New York, USA). A plastic sheet was fixed to the platform with three light weight springs, and the top of the sheet had two holes for plugs in order to hold the sheet on tension prior to heel placement. A straight piece of tape was used to mark the centre of the platform and the platform was then placed on non-slip matting to reduce the potential for unwanted movement.
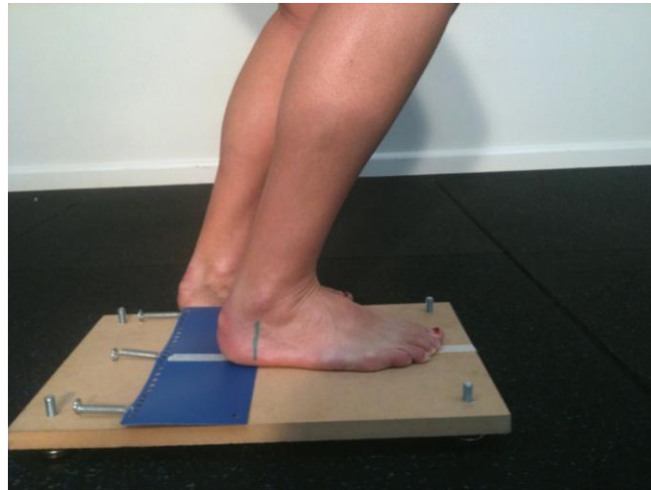
Figure 3.2 Custom designed platform for the standing lunge test that has a spring loaded sheet. The sheet displaced if the participant lifted their heel during the test.

3.3.4 Procedure

Prior to data collection, a vertical line was drawn from the right lateral malleolus to the lateral border of the participants' foot (Figure 3.2) and a point was marked on the anterior border of their right tibia 15cm below the tibial tuberosity using a non-permanent marker (Bennell et al., 1998). Only measures from the right leg were recorded. Four 30 second static stretches in both positions (standing lunge and straight knee DF tests) on the floor were completed prior to data collection to familiarise participants and precondition the tissue to minimise increases in DF ROM with repeated measures (Radford, Burns, Buchbinder, Landorf, & Cook, 2006; Taylor, Dalton, Seaber, & Garrett, 1990).

Participants then completed the standing lunge and straight knee DF tests, with and without the platform, with the order of conditions randomised between participants. For the trials without the platform, the floor was marked with a straight line and participants were asked to align their foot so the line passed through the centre of their heel and their second toe (Figure 3.1A & 3.1B). Once in position they

completed the two tests as described above. Each test was repeated three times with a ten second break between trials. The mean from the three trials was calculated and used for data analysis.

For the trials where the platform was used, participants were asked to place their heel on the plastic sheet on the platform with their foot aligned to the central tape line passing through the centre of the heel and the second toe (Figure 3.2). The heel placement was standardised so that the line from the participant's lateral malleolus to the lateral foot border was aligned with the front of the sheet (Figure 3.2). When completing the standing lunge and straight knee DF tests the measurement was repeated if the plastic sheet moved. Similar to the floor based testing, each test was repeated three times with a ten second break between trials. The mean from the three trials was calculated and used for data analysis.

In each test, the angle of DF was measured with a digital inclinometer with the centre of the inclinometer placed on the mark on the tibia (Figure 3.3) (Munteanu et al., 2009). All ankle DF tests were measured to the nearest 1°. Care was taken with the placement of the inclinometer to ensure it was not placed over the tibialis anterior muscle. Once measures were taken, all pen markings were removed using an alcohol wipe.

A   B
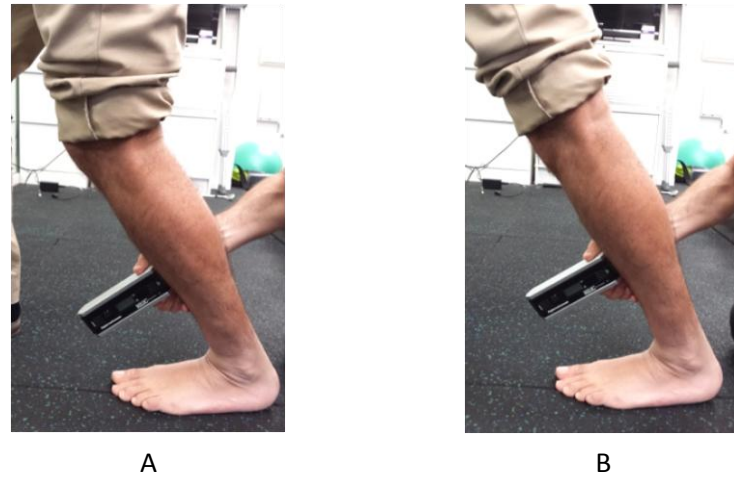
Figure 3.3 Positioning of digital inclinometer for measuring ankle dorsiflexion where (A) is the standing lunge test (left leg) and (B) is the straight knee dorsiflexion (left leg).

All participants returned one week later for repeat measurements for the reliability analysis. The protocol for the second testing session was identical to the first. The selected testing interval was considered large enough to avoid effects of the first testing session, such as changes in ankle joint ROM (Munteanu et al., 2009).

3.3.5 Statistical analysis

All angle measurements for the standing lunge test and straight knee DF test with and without the novel platform were collated for each day (Week 1 and 2) and rater (Rater A, B, and C). Statistical Package for Social Sciences software (SPSS, version 22.0 for Windows, IBM, Armonk, New York) was used, except where stated otherwise, with an alpha level of $p < 0.05$ set for all statistical analyses. The statistical procedures employed in this study are summarised in Figure 3.4.
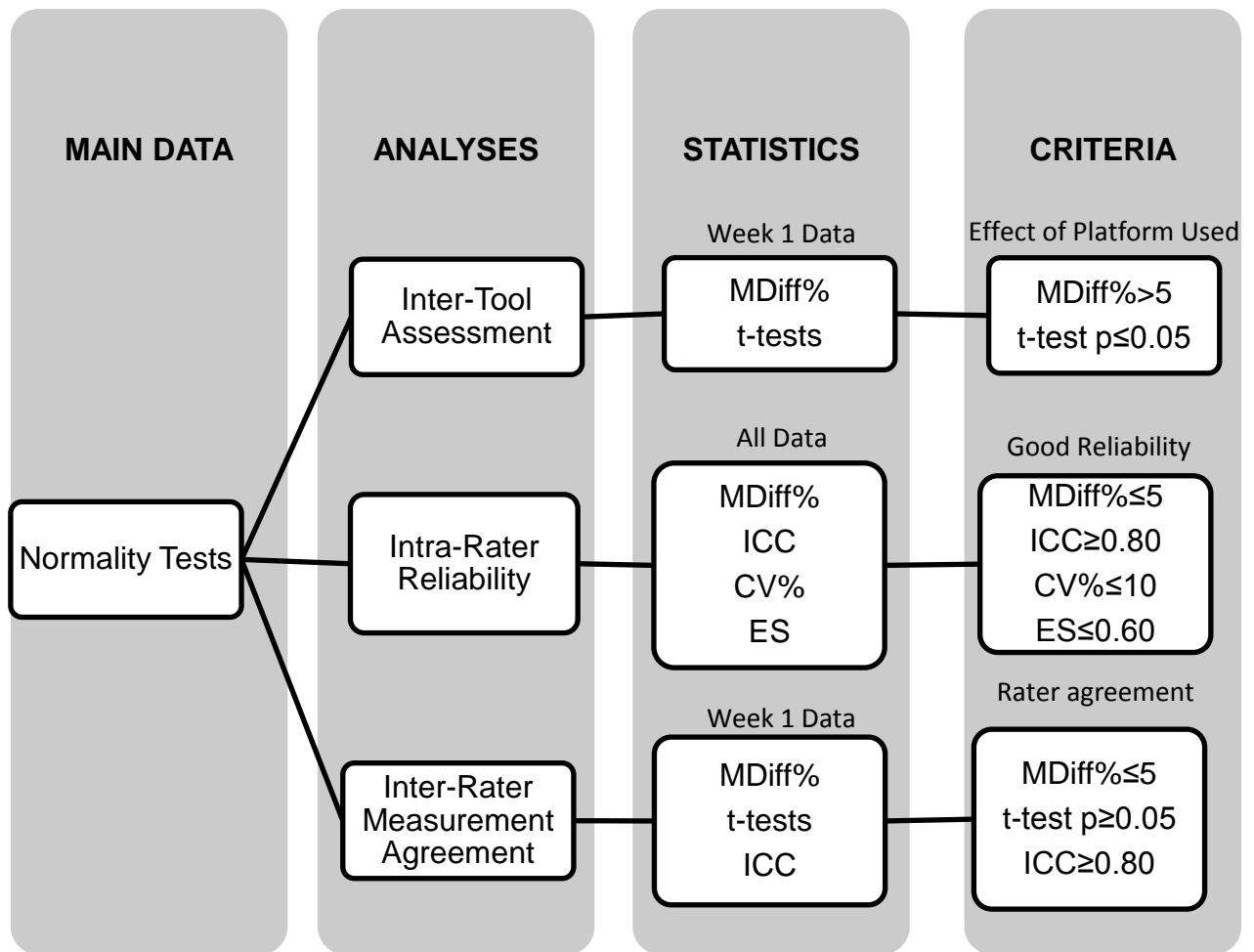
Figure 3.4 Schematic diagram of the statistical analyses employed in this study, where MDiff% is the mean difference score as a percentage of the mean score, ICC is the intra-class coefficient, t-tests are paired samples t-tests, CV% is a coefficient of variation as a percentage of the inter-day mean score, and ES is a Cohen's effect size.

All data were initially checked for normality using the difference between the mean and the median. Mean-median data within 10% was considered to be normally distributed (Bradshaw et al., 2009; Peat & Barton, 2005). Due to the small sample size, normality was further assessed using a Shapiro Wilks test (Peat & Barton, 2005; Riffenburgh, 2012). All data were required to be classified normal for both tests for parametric statistical testing to be applied. If a single test failed, non-parametric testing was applied. All data displayed a normal distribution and therefore parametric statistical methods were employed for the inter-rater reliability, intra-rater reliability,

and inter-platform assessment. Consistent with the recommendations of Atkinson and Neville (1998), more than one statistical method was used for each of these assessments. For that reason, criteria for the interpretation of the results were also set.

3.3.5.1 Inter-platform assessment

The effect of the standard protocol and novel platform on DF range was assessed for all of the data from week 1. Mean difference scores (MDiff%) and a paired samples t-tests (floor, novel platform) were calculated. Small differences in the mean (MDiff%) are indicative of similar measurements between the floor and the novel platform. An "effect of platform used" was concluded when the criteria threshold of MDiff% > 5% and $p \leq 0.05$ were both met.

3.3.5.2 Intra-rater reliability

The intra-rater reliability statistics were analysed in accordance with the methods of Hopkins (2000) for the full data set (both weeks, all raters). The range of statistical measures included MDiff%, ICC's, coefficient of variation as a percentage of the inter-day mean score (CV%) and Cohen's effect sizes (ES). Cohen's effect sizes (ES) and CV% were calculated consistent with the methods of Bradshaw *et al.* (2010). ES were interpreted as <0.1 as trivial, 0.1-0.6 as small, 0.6-1.2 as moderate and >1.2 as large (Bradshaw et al., 2010; Joseph, Bradshaw, Kemp, & Clark, 2013; Saunders et al., 2006). The methods of Bradshaw *et al.* (2010) and Joseph et al., (2013) were used for interpretation of the reliability statistics. In defining an overall

rating of "good reliability", the criteria threshold of MDiff% ≤ 5%, ICC ≥ 0.8, CV% ≤ 10% and ES ≤ 0.6 all had to be reached (Joseph et al., 2013). For "moderate reliability", three of the four criteria needed to be met, while "poor reliability" was defined as when at least two criteria were not met.

3.3.5.3 Inter-rater measurement agreement

Systematic differences between raters for measurements of DF (standing lunge and straight knee DF tests) with and without the novel platform were identified for week 1 data using MDiff% and paired samples t-tests. Consistency of measures between raters was determined using a two-way mixed model intra-class coefficient (ICC). An "effect of rater" was concluded when the criteria threshold of MDiff% > 5% and p≤0.05 (from t-test analysis). Good relative consistency was determined with a threshold of ICC≥0.80.

3.4 Results

3.4.1 Inter-platform assessment

Dorsiflexion angles were 2-10% smaller when the platform was used (Table 3.1). Inconsistent findings were revealed for the effect of the platform on the DF measures for each rater. The use of the platform had a significant effect on the DF measures for Rater A during the two tests. The use of the platform had an effect on the DF measures for Rater B only during the straight knee DF test. No effect was identified between measures with or without the platform for the straight knee DF test for Rater C. It was unclear whether the platform altered Rater C's measures for the standing

lunge test. As a group the effect of the platform was identified only for the straight knee DF test.

Table 3.1 Descriptive data (mean ± standard deviation) for ankle dorsiflexion (DF) range of movement from week 1 during the standing lunge test and the straight knee dorsiflexion test with and without the novel platform.

| Rater | Standing Lunge Test | | | | | Straight Knee Dorsiflexion Test | | | | |
	Ankle DF No Platform (°)	Ankle DF With Platform (°)	MDiff (%)	t-test	Effect of Tool Used	Ankle DF No Platform (°)	Ankle DF With Platform (°)	MDiff (%)	t-test	Effect of Tool Used
A	43.8 (5.8)	41.2 (5.4)	5.6	0.000	Yes	37.2 (5.7)	34.9 (4.3)	6.2	0.002	Yes
B	41.2 (5.6)	40.2 (5.2)	4.3	0.066	No	35.4 (5.0)	32 (4.7)	9.7	0.000	Yes
C	39.9 (6.0)	38.7 (6.3)	3.1	0.045	Unclear	33.7 (5.0)	32.9 (5.6)	2.4	0.340	No
Average	41.6 (5.9)	40 (5.6)	3.8	0.000	Unclear	35.4 (5.3)	33.2 (4.9)	6.1	0.000	Yes

Notes: MDiff is the normalised difference in the means between measures, and t-test is the alpha level revealed from the paired samples t-tests.

3.4.2 Intra-rater reliability

Intra-rater reliability was good for all raters for the standing lunge test when performed with or without the platform (Table 3.2). Good to moderate reliability was identified for the straight knee DF test (Table 3.2).

The inter-week reliability of Rater A's measures were all good with mean differences between weeks ranging from -2.7 to 0.1%. The mean differences between weeks for Rater B ranged from -6.7 to -2.5% indicating that Rater B's measures were consistently smaller in week 2. Rater B's straight knee DF measures had moderate reliability, regardless of whether the platform was or wasn't used. The mean differences between weeks for Rater C ranged from -7.2 to -2.4%. Rater C also took smaller measures in week 2. Rater C achieved good reliability for the straight knee DF test when not using the platform, but only moderate reliability when using the platform.

<u>Table 3.2</u> Intra-rater reliability of the measures of ankle dorsiflexion (DF) from weeks 1 and 2.

Test	Tool	ICC	SEM (^{0})	CV_{ME} (%)	ES	ES	MDiff (%)	Reliability
Rater A								
Standing Lunge Test	Ankle DF No Platform	0.87	2.2	6.5	-0.19	Trivial	-2.7	Good
	Ankle DF With Platform	0.93	1.4	4.9	0.06	Trivial	0.8	Good
Straight Knee DF Test	Ankle DF No Platform	0.94	1.5	5.7	-0.06	Trivial	-1.0	Good
	Ankle DF With Platform	0.93	1.3	5.1	0.00	Trivial	0.1	Good
Rater B								
Standing Lunge Test	Ankle DF No Platform	0.87	2.1	6.7	-0.17	Trivial	-2.5	Good
	Ankle DF With Platform	0.91	1.7	4.8	-0.33	Small	-4.8	Good
Straight Knee DF Test	Ankle DF No Platform	0.75	2.7	9.6	-0.26	Small	-4.2	Moderate
	Ankle DF With Platform	0.82	1.9	6.6	-0.45	Small	-6.7	Moderate
Rater C								
Standing Lunge Test	Ankle DF No Platform	0.88	2.2	7.3	-0.14	Trivial	-2.4	Good
	Ankle DF With Platform	0.89	2.1	7.2	-0.18	Trivial	-2.9	Good
Straight Knee DF Test	Ankle DF No Platform	0.81	2.4	9.5	-0.14	Trivial	-2.4	Good
	Ankle DF With Platform	0.86	2.1	7.4	-0.40	Small	-7.2	Moderate

Notes: ICC is intra-class coefficient, SEM is the standard error of measurement, CV_{ME} is the coefficient of variation, ES is Cohen's effect size, and MDiff is the normalised difference in the means between measures).

3.4.3 Inter-rater measurement agreement

Good relative agreement between the rater's measurements was identified for the week 1 data (Table 3.3). The intra-class correlations for the standing lunge tests

were 0.94 and 0.91 respectively when using or not using the platform. Similarly for the straight knee DF test the intra-class correlation was 0.86, regardless of whether the platform was used. The absolute measurement agreement between raters was inconsistent between raters with only Rater A and Rater C typically demonstrating poor agreement (Table 3.3).

Table 3.3: Inter-rater ankle dorsiflexion (DF) measurement agreement for the two tests with and without the platform

Test	Platform	Comparison	MDiff (%)	t-test	Absolute Agreement
Standing Lunge Test	No	Rater A vs B	5.9	0.002	Poor
		Rater B vs C	3.0	0.074	Good
		Rater A vs C	9.6	0.001	Poor
	Yes	Rater A vs B	2.6	0.057	Good
		Rater B vs C	3.7	0.031	Moderate
		Rater A vs C	6.7	0.004	Poor
Straight Knee Dorsiflexion Test	No	Rater A vs B	4.7	0.039	Moderate
		Rater B vs C	4.9	0.063	Moderate
		Rater A vs C	10.3	0.005	Poor
	Yes	Rater A vs B	8.2	0.001	Poor
		Rater B vs C	2.8	0.383	Good
		Rater A vs C	6.0	0.055	Moderate

Notes: MDiff is the normalised difference in the means between measures, and t-test is the alpha level revealed from the paired samples t-tests.

3.5 Discussion

The use of the platform had no consistent statistically significant effect on the DF ROM measures. Significantly smaller measures of ankle DF were recorded for both tests when using the platform in only one of the three raters (Rater A). The platform had no clear effect for the standing lunge test for the group of raters however it did for the straight knee DF test. Measurement of ankle DF in weight bearing is a reliable test, however using the platform is no more reliable than the standard protocol. It was identified that there was poor absolute measurement agreement however there was good relative measurement agreement between raters.

Differences between the measurements of DF using the platform were varied. Rater A recorded 2.6° (MDiff%=5.6%, $p<0.001$) less ROM in the standing lunge test when using the platform in comparison to the standard protocol, and 2.3°

(MDiff%=6.2%, p=0.002) less ROM in the straight knee test. The other experienced rater, Rater B, had lower measurements of ankle DF in only the straight leg DF test, measuring 3.4° (MDiff%=9.7%, p=<0.001) less ROM. No significant differences were identified between the measurements taken by the inexperienced rater, Rater C, with or without the platform. On average, lower measurements of ankle DF (2.2°) were recorded by the three raters for the straight knee DF test using the platform. The differences observed may also not be enough for the platform to be clinically useful in a young healthy population (19-28 years). These findings are in some agreement with the hypothesis that there would be smaller measurements of ankle DF when using the platform.

This research showed that the standing lunge test is a reliable measure regardless of the experience of the rater. Overall the inter-week reliability of the DF ROM measures were good for the standing lunge test when using the standard protocol or the platform, and moderate to good for the straight knee DF test. The findings of this study are in some agreement with two previous studies (Bennell et al., 1998; Munteanu et al., 2009) that have assessed the intra-rater reliability of assessing DF ROM in weight bearing. Further testing may have been appropriate for Raters B and C as they were not using the measurement protocol regularly and therefore may have needed more familiarisation with the tests. That may indicate that regular practice or use of these tests and equipment is important for reliable measures.

Consistent differences were identified for the DF ROM measures between raters in the four testing protocols rejecting the hypothesis that there would be high measurement agreement between raters. When assessing inter-rater measurement agreement there were consistent significant differences between results recorded by

Rater A when compared with Raters B and C. The differences identified could be explained by methodological differences between the raters. Rater A consistently recorded higher measures of DF. The differences appear to be a systematic difference since high ICC's (0.86-0.91) were displayed across the four tests despite the difference in raw measures. Similar to the previous studies, there is good relative agreement between raters indicated by these high ICC's (Bennell et al., 1998; Munteanu et al., 2009). The relative agreement suggests that although raters may record different measurements, they are consistently different. Therefore pooling results from a group of raters requires caution, however, if an individual rater of any level of experience is recording measures of ankle DF for a specific population (e.g. marathons runners), any significant findings in that population are likely to be in agreement with another rater.

The effect of experience was assessed and interpreted with caution in this study. There were two experienced raters and only a single inexperienced rater. Experience did not appear to have an effect in this study. Although there were consistent differences between the inexperienced rater (Rater C) and one of the experienced raters (Rater A), there was good measurement agreement between the Rater C and the second experienced rater (Rater B).

There were some limitations that may have affected the outcomes of this study. The varied results recorded using the platform may be as a consequence of the difficultly in using the apparatus. If the sheet slipped and a test was to be repeated, the process of resetting the device was awkward and became time consuming if it occurred repeatedly. Another potential issue may have been that the individual being tested may not push to their end of their DF ROM as they were too focussed on not allowing the sheet to slip. This suggests that a more appropriate

sensor for heel lift may be required if a difference would be clinically relevant, such as a contact sensor to assess the impact of heel lift more effectively. It was also observed that despite providing a familiarisation session, Raters B and C correctly positioned participants with the foot along the line as described in the methods however they did not align the inclinometer with the same central line that the foot was placed on. This may have been due to poor communication during the familiarisation session. This would have potentially had a greater impact on inter-rater measurement agreement, whilst it should have less of an impact on intra-rater reliability as the same protocol was implemented each time by each rater. Follow up research based upon this observation would provide insight into the impact of the information provided in the familiarisation session.

3.6 Conclusion

The platform tested in this study did not significantly affect measurements for all measurements of ankle DF. Intra-rater reliability for measuring ankle DF was generally good, especially when using the standing lunge test protocol. Implementing the current platform to ensure heel contact did not improve reliability. Measurement of ankle DF has good inter-tester consistency however measures were not in agreement. Greater confidence in detecting any differences or changes in DF measurements would occur if the tests are completed by the same clinician or researcher. It was therefore decided to use the floor based measure for the main part of this research thesis rather than the platform, with all measurements recorded by the same rater. Further research is required to determine if a platform with a different design that ensures heel contact during ankle DF ROM tests may reduce the

occurrence of overestimation of ankle DF or alternatively refute the occurrence of overestimation of ankle DF.

Chapter Four

Prevalence and factors associated with asymptomatic

Achilles tendon pathology in male distance runners

4.1 Aims and hypotheses

The aims of this chapter were:

- To determine the prevalence of Achilles tendon pathology in an asymptomatic male running population. It was hypothesised that prevalence of Achilles tendon pathology would be higher in this population compared to inactive individuals.

- To explore the interaction between anthropometric measures and running history variables with the development of Achilles tendon pathology. It was hypothesised that adiposity (waist measures), running years, weekly running mileage and age were associated with the presence of Achilles tendon pathology. It was also hypothesised that weight bearing ankle dorsiflexion (DF) would not be associated with tendon pathology.

4.2 Introduction

Endurance runners expose their Achilles tendons to both high volume (quantity) and high intensity (speed) running over many years. The nature of running exposes the tendon to loads of approximately 12.5 times body weight every time the foot strikes the ground (Komi et al., 1992). The potential therefore for cumulative stress and then injury to the tendon is high.

Achilles tendinopathy (AT) is a debilitating running injury affecting up to 50% of distance runners over their lifetime (Kujala et al., 2005). Diagnosis of AT is established from a clinical examination and diagnostic imaging using ultrasound (US) typically in runners who present with clinical signs. The pathology and morphology of a tendon is frequently determined by visualising the tendon with US. Ultrasonography is inexpensive and readily available and can be used to confirm the diagnosis of AT, evaluate the pathology, assess thickness, hypoechogenicity and vascularisation (Cook et al., 2002; Khan et al., 2003; Maffulli et al., 1987; Ohberg et al., 2001). Previous research investigating individuals with symptomatic and asymptomatic tendons has identified similarities with the imaging. Therefore, the pathophysiology of tendon injuries is complex and consequently the terminology selected to describe a tendon disorder is difficult (Khan et al., 2003).

The association between symptoms and imaging is unclear. Several studies have shown that the presence of Achilles tendon pathology identified on US without the presence or any history of pain is relatively common with a prevalence of 11-52% (Fredberg & Bolvig, 2002; Gaida et al., 2010; Gibbon et al., 1999; Nicol et al., 2006). Athletes with asymptomatic tendon pathology have the typical pathological imaging findings described previously (Chapter 2), however the individual reports no history of

pain or stiffness. Degenerative tendon pathology although asymptomatic, may result in regions of the tendon that are less tolerant to high load elastic tasks, with the remaining healthy tissue exposed to higher loads (Cook & Purdam, 2014). Recently, structural abnormalities in the Achilles tendon observed on US has been identified as a risk factor for tendon pain (Comin et al., 2013).

Treatment for AT is costly and time consuming. Identifying associated and risk factors may help develop injury prevention programs. Therefore, research exploring asymptomatic tendon pathology prior to symptom development is of clinical interest. Thus far, the presence of asymptomatic tendon pathology in the patellar tendon on imaging has been identified as a risk factor in a young basketball population (Cook et al., 2001; Cook, Khan, Kiss, Purdam, et al., 2000). Comin et al. (2013) identified that the presence of structural abnormalities, particularly hypoechoic regions within the Achilles tendon increased the risk of developing tendon pain. Fredberg and Bolvig (2002) showed that 45% of elite soccer players with asymptomatic Achilles tendon pathology at the start of the season developed clinical symptoms during the season. To date, no research has investigated the prevalence of asymptomatic tendon pathology or any associated factors of tendon pathology in a distance running population. Identifying athletes at risk, who are then regularly exposed to high tendon load activities such as distance running may be an important injury prevention strategy as these athletes may become symptomatic over time.

High repetitive loading is believed to be one of the main pathological stimuli causing AT (Silbernagel et al., 2006). Repetitive mechanical overloading of the Achilles tendon, particularly with a sport such as long distance running where load accumulates over time, has the potential to induce a non-inflammatory pathology and degeneration (Khan & Cook, 2003). Long term load exposure as a consequence of

greater years of running is associated with AT (Knobloch et al., 2008) and increased running mileage is associated with a larger cross-sectional area of the Achilles tendon (Rosager et al., 2002). Older individuals who have had a greater life time load have an increased tendon cross-sectional area (Magnussen et al., 2003). Older age is not only a factor for increased tendon cross-sectional area as discussed, but it is also a factor for the development of tendon pathology (Gaida et al., 2010).

Other factors that may predispose a runner to develop tendon pathology are adiposity (high central adipose tissue accumulation measured with waist measurements), ankle joint DF and gender. A reduction in DF may limit the capacity of the triceps surae to absorb load and may therefore result in greater loading rates. Alternatively, an increase in DF range may prolong loads on the Achilles tendon over a larger range. Although there is some support for these theories, there are conflicting findings in the literature (Hein et al., 2014; Kaufman et al., 1999; Mahieu et al., 2006; McCrory et al., 1999). Achilles tendinopathy also typically affects more men than women in both a physically active (Astrom, 1998) and the general population (Gaida et al., 2010), suggesting that gender may be another risk factor.

This study selected a high risk group for Achilles tendon pathology, male distance runners and excluded women due to the difficulty for controlling and measuring for the impact of oestrogen as a contributing factor (Cook et al., 2007). The purpose of this study was to determine the prevalence of asymptomatic tendon pathology in male distance runners and identify potential associated factors for the population, including age, adiposity, ankle DF and running history.

4.3 Methods

4.3.1 Participants

The endurance runners recruited for this study were classified as experienced (minimum running history of 3 years), high mileage (on average running a minimum of 30 km/week for the previous three months) athletes with no history of Achilles tendon pain. Other inclusion criteria for the study were that the participants must (1) be aged between 25-55 years, (2) have run a marathon or a half marathon in the last two years, (3) have had no lower limb injury for the previous six months that forced them to stop running for more than one week, and (4) no other significant medical condition (e.g. diabetes mellitus, systemic inflammatory disorders, previous Achilles trauma or rupture or insertional AT). Volunteers were recruited from advertising at running and triathlon clubs, online forums (www.coolrunning.com.au), attendance of running events and running training sessions providing brochures, as well as advertising through Athletics Australia.

This study was approved by the Australian Catholic University Human Research Ethics Committee (Appendix C). Prior to testing, each volunteer was informed of the procedures, risks and benefits of the study. Informed consent was then attained (Appendix D).

4.3.2 Procedure

Each participant completed a preliminary survey on their running history (Appendix E). The survey included details such as age, average running mileage per week, running history (approximate years), number of marathons/half-marathons

completed, and their injury history (defined as an injury that sidelined or required modified training for a period of 6 weeks or greater, and any injury that has forced them to stop running for a period of greater than one week in the previous six months). If the number of running sessions per week or mileage per week was reported as a range of two values on the running survey, the mean was recorded (e.g. 3-4 session/week: mean 3.5 sessions/week, or 50-60 km/ week: mean 55 km).

If the participant met the inclusion criteria for the study, their height was measured using a portable stadiometer (S+M Height Measure, 2m, AAXIS PACIFIC, Australia) and body mass was measured using portable scales (TANITA, Tokyo, Japan). Each participant then completed the Victorian Institute of Sport Assessment - Achilles Questionnaire (VISA-A survey: Appendix F). The VISA-A survey quantifies Achilles tendon pain, function, and activity through questions and simple exercise tests such as hopping. The VISA-A has been shown to be a reliable and valid measure for evaluating the severity of AT (Robinson et al., 2001; Silbernagel, Thomee, & Karlsson, 2005). Next, waist circumference was measured using a metal tape measure (Lufkin, W606PM, 2m) (Gaida et al., 2010). Waist circumference was measured in a horizontal plane at the midpoint between the iliac crest and lower costal margin. Each measurement was taken twice and the mean of the two measures recorded. If there was a discrepancy of greater than 1% between measures, a third measure was taken and the median recorded.

After completing the questionnaires and physical measures, participants' right and left ankle DF was measured using a standing lunge test (Figure 4.1A). Good inter and intra-rater reliability for evaluating weight bearing ankle joint DF ROM has previously been established for the standing lunge test by Bennell, Talbot, Wajwelner, Techovanich & Kelly (1998). Good intra-rater reliability was also

established in our previous study (chapter 3), with good relative inter-rater agreement despite differences in measurements recorded. Each participant aligned their heel and their second toe on a line. Participants were then instructed to dorsiflex their ankle as much as possible, ensuring that their heel remained flat on the ground.

(A)   (B)
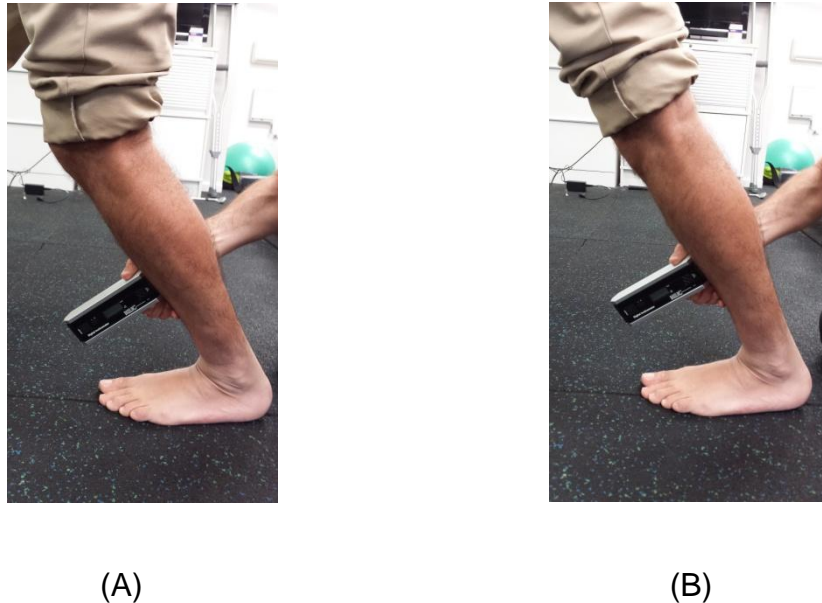
Figure 4.1 (A) Standing lunge test, and (B) straight knee dorsiflexion test.

The participants also completed a straight knee DF test based on the technique outlined by Munteanu et al. (2009). The straight knee DF test provided an indication of the influence of the gastrocnemius muscle on ankle DF ROM, and was shown by Munteanu et al., (2009) to have good inter-rater and intra-rater reliability. Our study (chapter 3) had similar findings for the straight knee DF test. The participant was instructed to keep their knee straight and lean forwards until maximal stretch was felt in the back of the leg while ensuring they kept their heel in contact with the ground (Munteanu et al., 2009). Their foot was aligned on the line similar to the standing lunge test.

Both measures of ankle DF ROM were measured with a digital inclinometer (Baseline® Digital Inclinometer, 12-1057, New York, USA) and the angle between the anterior border of the tibia and the vertical plane were recorded (Figure 4.1). The point of application for the middle of the inclinometer for measurement of ankle DF was 15 cm below the tibial tuberosity, consistent with Bennell *et al.* (1998). For both tests, the mean of three measures was recorded. Finally, the left and right Achilles tendon of each participant was examined using grey scale ultrasound (GE VIVID-I, GE, USA).

4.3.3 Ultrasound Imaging and Analysis

Left and right Achilles tendons were classified as abnormal or normal using a high-resolution grey scale (B-mode) ultrasound (GE VIVID-I, GE, USA) with a linear array probe at a frequency of 13 MHz. The images were taken by an experienced musculoskeletal radiologist (40 years radiology experience, 22 years in musculoskeletal imaging). The ultrasound images were collected with the participant in a prone position with their ankle at 90° (Figure 4.2). During scanning the probe was positioned perpendicular to the tendon to avoid anisotropy or artefacts. Consistent with Gaida et al., (2010) and Khan et al., (2003), a tendon was defined as abnormal if (1) one or more focal hypoechoic regions were visible in both the longitudinal and transverse scans, or (2) diffuse hypoechogenicity associated with bowing of the anterior tendon border was detected, (3) if diffuse hypoechogenicity associated with generalised thickening of the tendon in comparison to the contralateral tendon was found, or (4) if the tendon was thicker than 6mm (Section 2.5.2). The individuals identified to have abnormal tendons were classified to have asymptomatic Achilles tendon pathology (Gaida et al., 2010). A mark was made on the participants' leg with

a non-permanent marker one probe length from the position where the calcaneus was last visible in the longitudinal plane. A transverse image was taken at this point of the tendon. Another transverse image was taken at the thickest point of the tendon as determined by the radiologist. Thickness was measured inside the boundary of the tendon with the calliper measurement tool provided in the manufacturer's software (Echopac BT09, GE Vingmend Ultrasound Medical Systems, Milwaukee, Wisconsin, USA) preinstalled on the ultrasound equipment.

A   B
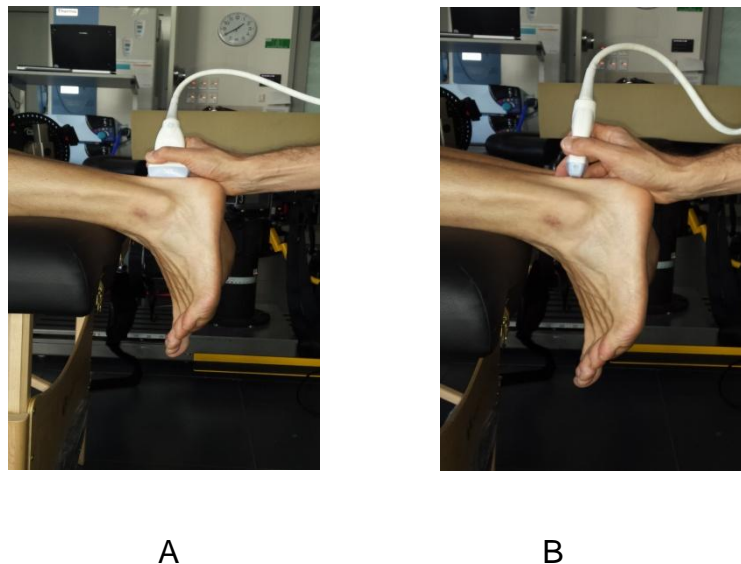
Figure 4.2 Positioning for ultrasonography; (A) Longitudinal scanning and (B) transverse scanning.

4.3.4 Data Analysis

From the ultrasound scans, participants were categorised into abnormal or normal groups based on the imaging findings. Achilles tendinopathy has many intrinsic risk factors, therefore if a participant had one or both tendons showing any tendon pathology they were classified as abnormal. For a classification of normal, both tendons were normal on US imaging. Measurements for waist girth, age, body

mass, height, mileage and training sessions per week, running history and DF ROM (standing lunge, straight knee) were analysed.

4.3.5 Statistical Analysis

Statistical analysis was completed using Statistical Packages for the Social Sciences (SPSS, version 22.0, IBM, Somers, New York). An alpha level of 0.05 was set for all analyses. For a statement of prevalence, a margin of error (ME) was calculated as follows:

$$Margin\ of\ error = \frac{2 * \sqrt{prev * (1 - prev)}}{\sqrt{N}}$$

Where *prev* is the prevalence of asymptomatic tendon pathology and *N* is the number of participants overall. All data were initially checked for normality using the difference between the mean and the median. Mean-median data within 10% was considered to be normally distributed (Bradshaw et al., 2009; Peat & Barton, 2005). Due to the small sample size, normality was further assessed using a Shapiro Wilks test (Peat & Barton, 2005; Riffenburgh, 2012). All data were required to be classified normal for both tests for parametric statistical testing to be applied. If a single test failed, non-parametric testing was applied. Not all of the data were normally distributed and due to the sample size it was not possible to assume that results from the running population recruited was a good reflection on the wider running population. Therefore non-parametric statistical procedures were employed for the data set that was obtained. Mann-Whitney U tests were utilised to compare the two groups (abnormal and normal tendons) across all measures. Data was described using medians and interquartile ranges (IQR). Effect sizes for the nonparametric data

were then calculated if any significant findings were identified (Effect size = $z/\sqrt{N}$) (Fritz, Morris, & Richler, 2012). Guidelines for effect sizes for the nonparametric data are that a small effect is 0.1, a medium effect is 0.3 and a large effect is 0.5 (Fritz et al., 2012). Due to the potential assumption that older runners have been running for longer periods of time, the association between age and years of running were examined with a Spearman's rho test. A post-hoc power calculation was done using G*power software (G*power, version 3.1.9.2, University of Kiel, Germany) to determine the power of the sample size and Cohen's d effect sizes for any significant findings. Cohen's effect sizes were interpreted as <0.1 as trivial, 0.1-0.6 as small, 0.6-1.2 as moderate and >1.2 as large (Bradshaw et al., 2010; Joseph et al., 2013; Saunders et al., 2006).

4.4 Results

Data were collected from 37 male distance runners with a median age of 36 years (IQR= 32-42 years), height of 180 cm (IQR=174.0-183.5cm) and a body mass of 77.4 kg (IQR= 73.8-83.4kg). Almost half (46%, ME = 17%) of these asymptomatic distance runners had at least one abnormal tendon (Table 4.1). More than a third (35%) of all tendons were abnormal on imaging with 47% of the athletes in the abnormal group having unilateral abnormalities. Tendon abnormalities observed on US imaging were predominantly regions of hypoechogenicity (84%) however five tendons were both hypoechoic and thickened (Figure 4.3 and Figure 4.4). One athlete had calcification identified within the tendon at the insertion of the Achilles tendon (Figure 4.5).

Table 4.1 Comparison of tendon measurements and classifications

	Abnormal tendons (n=17)	Normal tendons (n=20)
Total tendons n (%)	26 (35%)	48 (65%)
AP tendon thickness (mm)	5.4 (0.8)	4.7 (0.5)
Unilateral n (%)	8 (47%)	
Billateral n (%)	9 (53%)	
Abnormal tendon subgroups		
Hypoechoic n (%)	22 (84%)	
Fusiform n (%)	3 (12%)	
Thickened n (%)	1 (4%)	

A   B
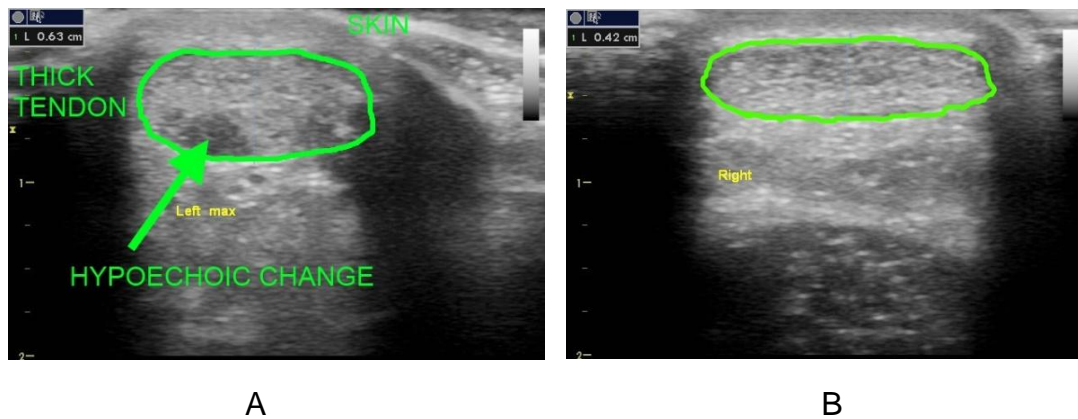
Figure 4.3 Example of transverse ultrasound images from (A) an asymptomatic tendon with thickening and hypoechoic change and (B) and normal tendon.

A   B
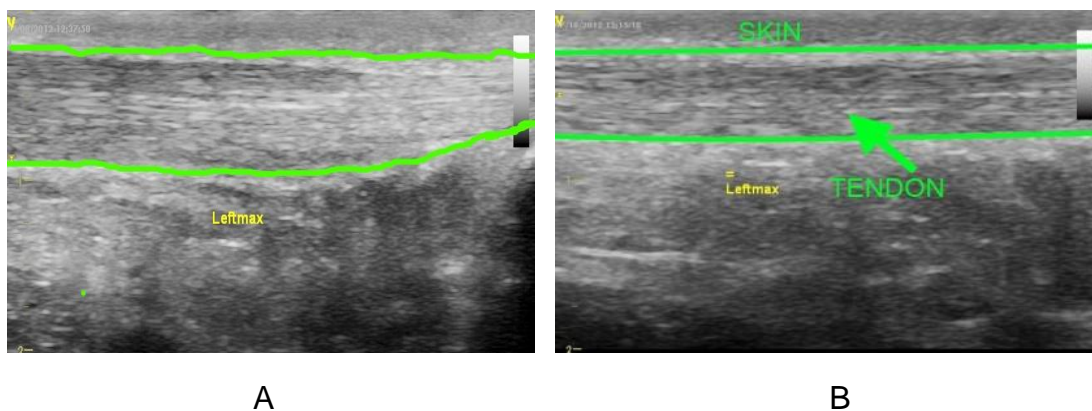
Figure 4.4 Example of longitudinal ultrasound images from (A) an asymptomatic tendon with hypoechoic change and fusiform in shape, and (B) a normal tendon.

A   B
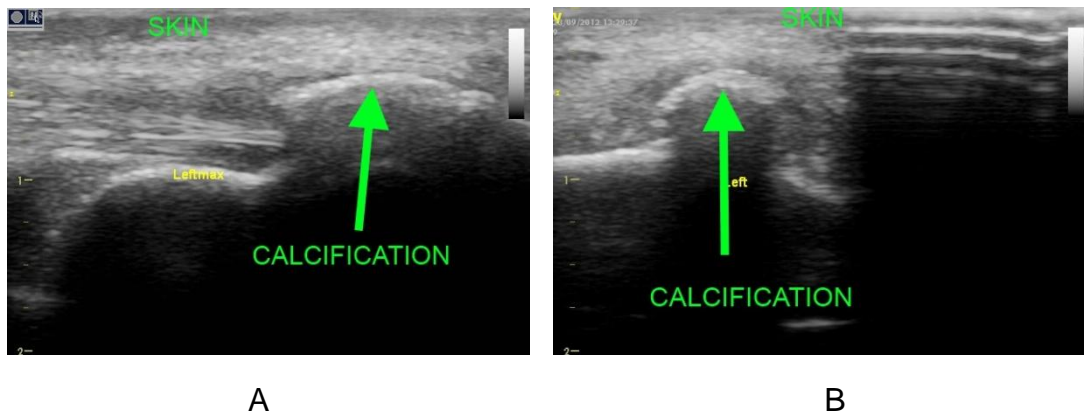
<u>Figure 4.5</u> Example of an athlete with calcification at the insertion of his Achilles tendon in the (A) longitudinal view and (B) the transverse view.

The runners with Achilles tendon pathology had significantly more years of running training (median= 20.0 years, IQR = 6.0-25.5, p=0.024) than the group of runners with no pathology on US imaging (median= 7.0 years, IQR= 5.0-15.0) (Table 4.2). The effect size was classified as medium (0.37). No significant differences between the groups was identified for age, adiposity, ankle ROM, number of weekly running sessions, weekly mileage and number of long distance (marathon and half marathon) running events completed (Table 3.2 and Table 4.3). Running years was moderately correlated with age (Spearman's rho=0.518, p=0.001). Post-hoc power calculation for years of running suggests that despite the sample size, the study was underpowered with a power of 0.70 and a moderate effect size of 0.86.

Table 4.2 Descriptive data of participants (Abnormal tendon group are participants with one or both tendons abnormal on imaging. Normal tendon group are participants with both tendons normal on imaging).

	Abnormal tendons (n=17)		Normal tendons (n=20)		p value	All tendons (n=37)	
	Median	IQR	Median	IQR		Median	IQR
Age (years)	39.0	33.5-46.0	35.5	32.0-38.8	0.110	36.0	32.0-42.0
Height (cm)	180.0	173.5-181.7	181.0	176.0-185	0.149	180.0	174.0-183.5
Mass (kg)	74.6	71.8-80.3	81.0	74.8-84.2	0.104	77.4	73.8-83.4
Waist (cm)	81.8	77.2-83.8	81.5	79.8-87.3	0.498	81.7	77.2-85.9
VISA-A	100	100-100	100	100-100	0.964	100	100-100
Running years	20.0	6.0-25.5	7.0	5.0-15.0	0.024*	10.0	5.0-21.5
Mileage (km/week)	60	45-66.3	47.5	36.3-63.8	0.270	55	40.0-65.0
Sessions/week	4.3	3.5-6.0	4.0	3.1-5.0	0.270	4.0	3.5-5.3
Half marathons	5	3-16	7	4-10	0.557	6	3-10
Marathons	3	1-8	2	0-6	0.598	2	0-7

Table 4.3 Ankle dorsiflexion measurements during standing lunge test and straight knee dorsiflexion (DF) test.

		Abnormal tendons (n=17)		Normal tendons (n=20)		p value	All tendons (n=37)	
		Median	IQR	Median	IQR		Median	IQR
Standing Lunge Test	Right ankle (°)	41.3	39.2-45.9	39.0	35.8-46.2	0.244	40.7	37.0-46.0
	Left ankle (°)	45.7	40.9-47.3	42.0	36.0-46.7	0.219	43.0	38.4-47.3
Straight Knee DF Test	Right ankle (°)	38.7	37.4-42.9	37.9	33.6-43.9	0.357	38.7	36.2-42.9
	Left ankle (°)	41.7	39.8-45.2	40.0	35.4-45.6	0.187	40.7	37.5-45.5

4.5 Discussion

The results of this study showed a high prevalence of asymptomatic tendon pathology in a male distance running population. Cumulative running years was an associated factor of tendon pathology rather than age. Waist measurements, body mass, ankle DF and weekly mileage as well as number of training sessions were not associated factors of Achilles tendon pathology. There was only a moderate correlation between age and years of running.

The prevalence of tendon pathology (46% of participants had at least one abnormal tendon and 35% of all tendons had an abnormality) was high. This finding may be due to a combination of the high loads that the runners expose their Achilles tendons in addition to the older age of participants recruited. Our findings were similar to Gibbon et al., (1999) who reported that 33% of the tendons in their asymptomatic volunteers (ages were not reported) had abnormalities on US imaging, and lower than Nicol et al. (2006), who reported 52% of tendons in an active asymptomatic population (mean age 33.1 years [standard deviation 6.8 years]) with no history of pain. In contrast Gaida et al., (2010) reported a much lower prevalence of 13% in a general non-athletic male population and identified an association between age and waist measurements in their population. Fredberg and Bolvig (2002) observed that 11% of tendons in a group of elite soccer players (18-35 years of age) had asymptomatic tendon pathology. Although it has been established that the presence of tendon pathology is relatively common, it is evident that it has only been identified as a risk factor for the development of tendon pain in limited populations (Comin et al., 2013; Cook, Khan, Kiss, Purdam, et al., 2000).

Ultrasonographic abnormalities do not always result in a symptomatic tendon, therefore the link between symptoms (pain and dysfunction) and pathology remains unclear. Tendon pathology was identified by Cook et al., (2000) as a risk factor for the development of tendinopathy in a young basketball population. More recently Comin et al., (2013) identified that specific changes in the tendon such as hypoechoic regions identified on US may increase the risk of developing tendinopathy. 84% of the runners with abnormal tendons identified on US in our study had hypoechoic regions, which may suggest these runners may be at risk of developing symptoms. Fredberg and Bolvig (2002) calculated the risk of developing symptoms of AT over the course of a single season as 45% when abnormal US changes were identified as compared to 1% for tendons with normal imaging. Although we did not collect prospective data, future studies should consider following participants over time to investigate whether tendon pathology is a risk factor for AT in distance runners.

Thickness of the Achilles tendon is affected by habitual long term loading. Male runners have been found to have an approximately 22% greater tendon cross-sectional area than non runners (Rosager et al., 2002). Furthermore, research has shown that athletes performing frequent high tendon load activities, such as running and jumping, have a tendon that is approximately 20% thicker than kayakers who experience a lower level of tendon loading (Kongsgaard, Aagaard, Kjaer, & Magnussen, 2005). Furthermore, Couppe et al., (2008) compared three distinct parts of the patellar tendon in athletes that predominantly loaded a single leg (e.g. Badminton and fencing), and identified an increase in cross-sectional area by 20-28% in the different regions of the tendon from the preferentially loaded leg. Long term tendon loading appears to induce tendon hypertrophy with the tendon adapting

over time through an increase in matrix protein production (Cook & Purdam, 2014). Unlike long term loading, short term loading (9 months of running) of the Achilles tendon does not appear to change tendon cross-sectional area in a sample of untrained novice runners (Hansen, Aagaard, Kjaer, Larsson, & Magnussen, 2003). Tendon adaptation that occurs may be identified as tendon abnormalities. Therefore in some individuals, these imaging abnormalities may simply be tissue adaptation to load rather than tendon pathology.

In this investigation the findings have shown that cumulative running years is an associated factor of Achilles tendon pathology. Interestingly, McCrory et al. (1999) also identified years of running as a risk factor for the development of AT. The cumulative running loads identified were only associated with running years, however it may also be in combination with weekly running mileage and running sessions per week. Although weekly running mileage was not identified as an associated factor it is a difficult measure to quantify via self-reporting. Weekly running mileage may be further influenced by the nature of running events throughout a calendar year. Endurance runners who participate in marathons and half marathons will have cyclical training plans that will be different from year to year depending on their goals, resulting in potential variations in running volume.

Despite running years being identified as an associated factor of tendon pathology, age was not identified as an associated factor in this population. Although it could also be assumed an older runner is likely to have run for more cumulative years, it was identified that running years and age was only moderately correlated (Spearman's rho = 0.518, p<0.001). These findings suggest that older runners have not always been running for a greater number of years. Distance runners frequently start running at an older age, which may explain the observation. Alternatively our

findings regarding the association between age and tendon pathology may have been influenced by the age of our cohort with a median of 36 years and an IQR of 32-42. Our finding may be more representative of this group rather than the general running population. Therefore in the clinical and research setting, running history needs to be explored in detail to avoid assuming running years is directly associated with the age of the runner.

Limited or increased ankle DF was not identified as an associated factor (Table 4.3). Our findings were in agreement with McCrory et al. (1999) and Hein et al., (2014), however these researchers utilised non-weight bearing positions for assessment of ankle DF. In contrast Kaufman et al., (1999) and Mahieu et al., (2006) who both prospectively utilised non-weight bearing DF tests, identified conflicting results, with reduced and increased ankle DF ROM respectively being identified as risk factors for developing AT. Further research into prospective studies using weight bearing methods to assess ankle DF is warranted.

A number of limitations were identified for this research. First the sample size was small in this study. Post-hoc testing revealed that the study was underpowered. Based on the difference between the years of running in the asymptomatic tendon pathology group and the normal tendon group, (Cohen's effect size = 0.86) with an alpha of 0.05, and a power of 0.80 requires a sample size of 48 with two groups 24 subjects. The small size may partly be due to strict inclusion criteria in addition to the limited availability of the population under investigation. However the criteria were set to be reflective of seasoned distance runners to add strength to the quality of the research. The consequence of the small sample means generalising findings to the broader distance running population should be done with caution. A further limitation of this study was interpreting years of running as a measure of cumulative load.

Participants were asked how many years they have been consistently doing long distance running with previous running-based sport not included. For example a distance runner with a swimming background will have exposed their tendon to far less cumulative load that of a retired Australian football player. In addition to loading history, other considerations that were not controlled or assessed were running surface and current running load. Whilst age was not identified as an associated factor, the limited age range of this cohort makes generalising this finding to all distance runners difficult. Soft surfaces are associated with AT as previously discussed and the terrain that distance runners select may be influenced by where they live or work, their running load tolerance, time available to commute for training and upcoming races making running surface difficult to control. The weekly running mileage may also be influenced by similar factors such as running load tolerance, time available to train due to external commitments (e.g. work, family) and recent running goals (e.g. half marathon versus ultra marathon).

4.6 Conclusion

There is a high prevalence of tendon pathology in an asymptomatic male running population with no history of Achilles tendon pain. Cumulative years of running appear to be an associated factor of Achilles tendon pathology. Further research is required to examine a running population with a greater spread of ages (e.g. 15-65 years) to explore if age is an associated factor of tendon pathology. Further research is required to determine if the presence of tendon pathology is a risk factor for developing symptomatic AT and secondly to determine if biomechanical loading strategies may be associated with or altered in the presence of tendon pathology. If there is an association between the development of symptoms in the

presence of tendon pathology in specific populations such as endurance runners, US

could be utilised as a screening tool to develop injury prevention programs.

Chapter Five

Lower limb stiffness regulation during level and incline hopping

5.1 Aims and Hypotheses

- To determine the effects of incline hopping on lower limb kinematic and kinetic parameters in runners with normal Achilles tendons. It was hypothesised that hopping on an incline would cause a reduction in knee joint angular displacement and an increase in ankle joint angular displacement. In addition it was hypothesised that altered joint kinematics at initial ground contact would be identified.

- To assess the effect of incline hopping on lower limb stiffness regulation. It was hypothesised that hopping on an incline would display an increase in knee joint stiffness and decrease in ankle joint stiffness.

5.2 Introduction

Running is a complex form of a bounding gait. In a bounding gait the muscles, tendons and ligaments in the lower limb store and release elastic energy as an athlete travels along the ground in a spring like manner (Farley & Morgenroth, 1999). Biomechanical study of bounding gaits provides insight on locomotion mechanisms in healthy and injured populations. Assessing running can be very difficult, and consequently hopping is a simple and commonly utilised test in both research and clinical settings to explore the spring-like function of the lower limb (Ferris & Farley, 1997). The spring-like behaviour of the leg in hopping has been explored extensively using the spring-mass model (Farley et al., 1991; Farley et al., 1993).

The spring-mass model is a biomechanical model that is utilised to analyse the integration of the musculoskeletal system as a single spring. The spring-mass model consists of a single linear "leg spring" and body mass (Farley & Morgenroth, 1999). During hopping the leg spring initially compresses and then lengthens during the ground contact phase. The limb joints flex and then extend to release the elastic energy stored. This pattern of movement is similar to that of running. When using the spring-mass model, the key parameter studied is the "stiffness" of the leg spring (Morin, Dalleau, Kyrolainen, Jeannin, & Belli, 2005).

Mechanical stiffness during hopping is calculated from the ratio of the peak ground reaction force produced when the centre of mass (COM) is at its lowest point, and the vertical displacement of the COM during the contact phase (Farley et al., 1998). The active, vertical leg stiffness of the leg spring represents the average stiffness of the overall musculoskeletal system during the ground contact phase.
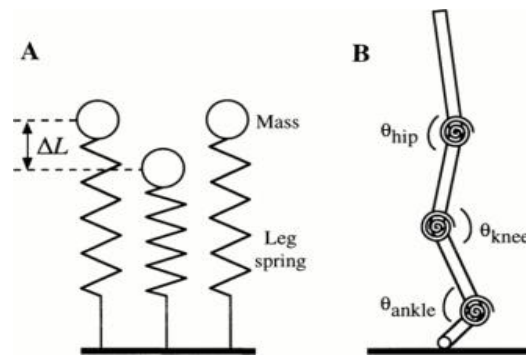
<u>Figure 5.2</u> (A) Spring-mass model for hopping and (B) multi-joint torsional spring model of the joints (Farley et al., 1998).

Leg stiffness is dependent on the stiffness of the joints, where the joints behave like springs (Farley et al., 1998: Figure 5.1B). If the ankle, knee, and hip are stiffer, they undergo smaller angular displacements during the loading phase, resulting in less leg compression and reduced displacement of the COM, and consequently higher leg stiffness (Farley & Morgenroth, 1999). In the multi-jointed musculoskeletal system, a variety of strategies can be used to adjust leg stiffness (Farley & Morgenroth, 1999).

During hopping at a person's preferred frequency or higher frequencies, the body behaves like a simple spring (Austin et al., 2002; Farley et al., 1991). When hopping at frequencies lower than the preferred rate there is a loss of the typical spring pattern (Austin et al., 2002; Farley et al., 1991). Stiffness has been shown to increase with higher hopping (unipedal and bipedal) frequencies (Austin et al., 2002; Farley et al., 1991), and both strength (Hobara et al., 2008) and endurance training (Hobara, Kimura, et al., 2010).

Leg stiffness has been related to injury. It has been suggested that higher levels of stiffness are associated with bony injuries, whereas lower levels of stiffness

are associated with soft tissue injuries (Butler et al., 2003; Williams et al., 2004; Willliams et al., 2001). Previous research exploring stiffness during running, runners with tibial stress fractures for example have been shown to have higher levels of knee stiffness (Milner et al., 2007), whilst runners with low back pain have also displayed increased stiffness, particularly at the knee during running (Hamill et al., 2009). There has been limited research investigating the association between injury and leg stiffness during hopping. It has been reported that too much or too little ankle stiffness during hopping is associated with injuries in young gymnasts (Bradshaw & Hume, 2012). Therefore too much or too little stiffness therefore has potential to contribute to injury (Butler et al., 2003).

Lower limb stiffness regulation has been primarily explored on a level surface with limited research examining the impact of an incline surface on the leg spring. . Hopping on an incline has the potential to increase the load on the ankle plantarflexors (Kannas et al., 2011) that may alter the biomechanical patterns observed during level hopping, therefore hopping on an incline surface to examine the spring-mass model may provide a model to explore incline loads on the lower limb. The behaviour of the spring-mass model during hopping on an incline surface has only been recently investigated (Kannas et al., 2011). Incline hopping has been observed to have no effect on leg stiffness (Kannas et al., 2011), however ankle and knee joint stiffness have yet to be investigated. Hopping on an incline surface potentially emphasises the demand of the triceps surae and ankle as it allows the ankle to go further into dorsiflexion (DF) as compared to level surface hopping (Kannas et al., 2011). In addition, it has been suggested that incline hopping results in greater elongation of the Achilles tendon that may have an impact on the elastic recoil and stretch-shortening cycle, as well as the potential stress placed on the

tendon (Kannas et al., 2011; Lichtwark & Wilson, 2006). The alteration of ankle function may impact the interaction between the knee and the ankle during incline hopping.

Testing protocols such as incline hopping are required to place greater loads on the triceps surae and thus through the Achilles tendon in both a research and clinical setting. Therefore the purpose of this study was to explore the loading strategies of the lower limb, with particular focus on the joint interplay between the ankle and the knee during incline hopping.

5.3 Methods

5.3.1 Participants

Experienced male endurance runners were recruited. The recruitment criteria were men aged between 25 and 55 years, a running history greater than 3 years, running at least 30 km/week, no history of an Achilles injury and injury free for the last 6 months. An injury was defined as any musculoskeletal problem that forced the runner to stop running for a period greater than one week. Participants with Achilles tendon pathology identified on imaging from Chapter 4 of this thesis were excluded.

This study was approved by the Australian Catholic University Human Research Ethics Committee (Appendix C). Prior to testing, each volunteer was informed of the procedures, risks and benefits of the study. Informed consent was then attained (Appendix D).

5.3.2 Procedure

The runners were tested in a single session in the School of Exercise Science Advanced Research Laboratory. Prior to biomechanical testing each participant had the following anthropometric measures taken and entered into the VICON Nexus software (VICON Nexus, Oxford Metrics Limited, United Kingdom): height, body mass, inter-anterior superior iliac spine (ASIS) distance, leg length (ASIS to medial malleolus), knee width and ankle width. ASIS distance and leg length were measured with a metal measuring tape (Lufkin, W606PM, 2m) and knee and ankle width were measured by using sliding bone callipers (TTM Martin's Human Body Measuring Kit, Mentone Educational Centre, Carnegie, Australia) to the nearest $\pm$1mm. Next, fourteen 14mm retroflective markers and two knee alignment devices (KADS) were placed on participants using double sided tape consistent with the Plug-in Gait model (Figure 5.2). The Plug-in Gait model is commercially available kinematic model of VICON. These sites were the ASIS, posterior superior iliac spine, lateral thigh, lateral leg/shank and lateral malleolus. The remaining markers were placed on each shoe overlying the posterior calcaneus and the 2nd metatarsal head. The KADS were used to define the alignment of the knee flexion axis prior to the hopping trials. All marker placements were completed by the same tester to avoid inter-tester variability (Schache, Blanch, Rath, Wrigley, & Bennell, 2002). A five second static capture was performed for estimation of joint centres in the VICON Nexus software prior to commencing the hopping test protocol. Following the static trial, the KADS were removed and replaced with two retroflective markers, with one being placed on each lateral femoral condyle.
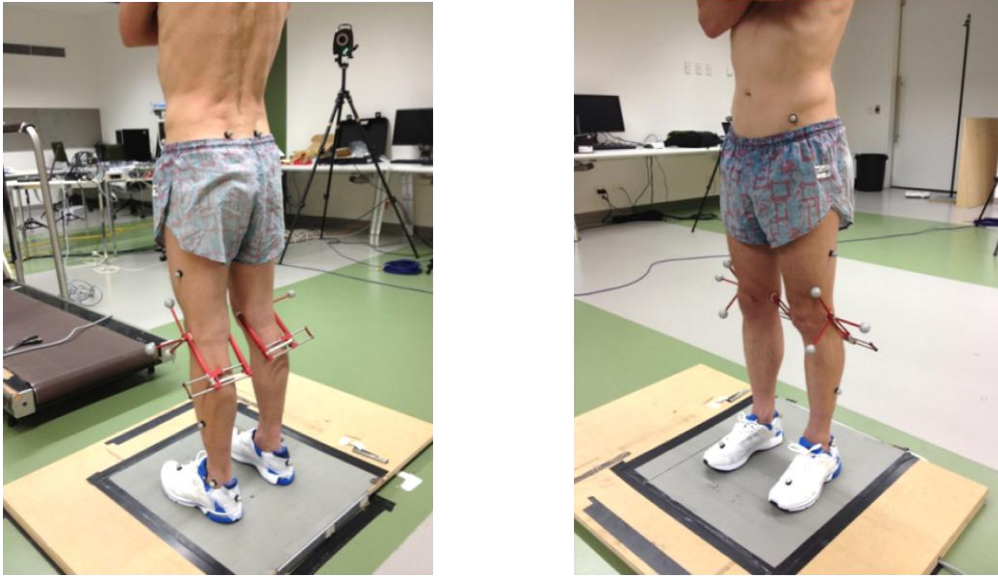
<u>Figure 5.2</u> Marker and knee alignment device placement for the static trial in accordance with the Plug in Gait model.

The participants then performed a running warm-up for ten minutes on a treadmill (H/P/Cosmos 3p 4.0, H/P/Cosmos Sports and Medical GmbH, Nussdorf-Traunstein, Germany) with a gradient of 1-2% at a self selected speed. Next, the participants hopped on a force plate (Kistler, 9286AA, Switzerland) with a single leg (both right and left) at 2.6Hz, using a digital metronome (Cherub, WMT-555C, Nanshan, China) for a period of 10 seconds. To emphasise the function of the triceps surae and to test the triceps surae more rigorously, individuals were instructed to keep their hands across their chest and to keep their knee "stiff" whilst hopping, with maximal effort and without the use of an aid (Hobara et al., 2007). Verbal reinforcement was given regularly during hopping to encourage the maintenance of a straight knee. Finally, participants completed a single trial of hopping on a custom built medium density fibre platform with an incline of 10°. Kannas et al. (2011) utilised an incline hopping protocol with a wooden box with an inclination of 15° relative to the ground. They justified the 15° angle as it would increase ankle DF during hopping

while avoiding an excessive range of motion (ROM) that could possibly cause an injury. The angle chosen in our study was twofold. In agreement with Kannas et al. (2011), safety is imperative but further to this the 10° angle was primarily selected because the demand on the triceps surae complex has been shown to be increased during the propulsive phase of running when the angle of inclination reaches 9° (Gottschall & Kram, 2005). A medium density fibre board with a cut out the size of the force plate (Figure 5.2 & Figure 5.4B) was placed on the floor as well as the incline platform around the force plate. The force plate within the cut out was sitting flush with the overlying board. The overlying board was utilised because during pilot testing volunteers reported a concern of hopping off the force plate. The reported sensation was negated with the use of the surrounding medium density fibre board.
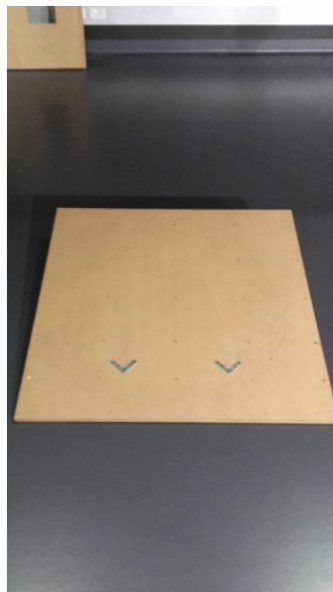
A   B

Figure 5.3 Custom built incline platform (A) from above and (B) from the side.

A   B
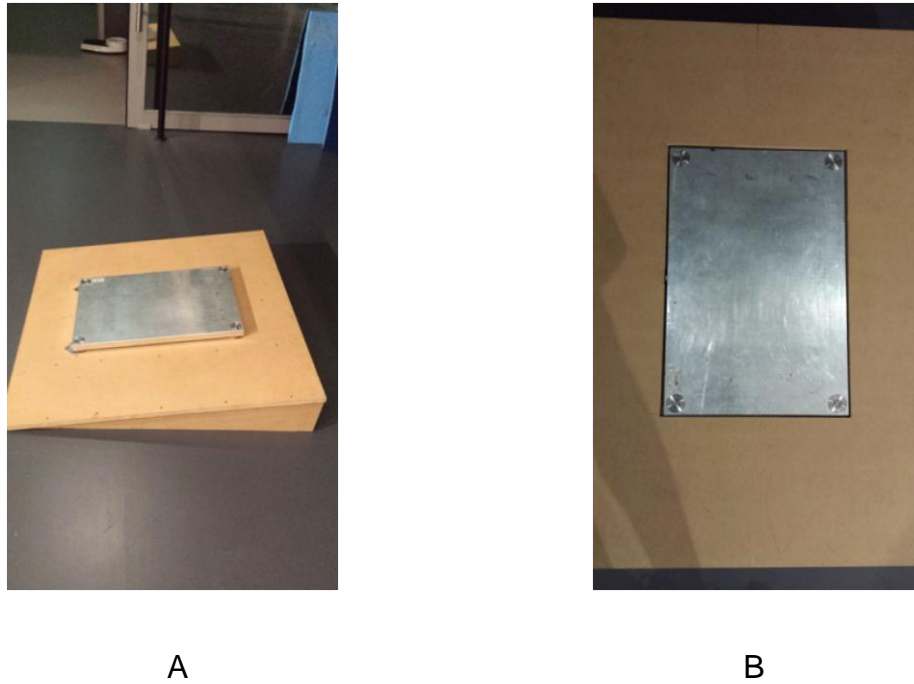
<u>Figure 5.4</u> Incline platform with (A) the force plate in place and (B) the force plate with the surrounding medium density fibre board.

Hopping stiffness regulation can be affected by footwear (Bishop et al., 2006), therefore all participants were supplied with the same brand and model of footwear (Adidas, Supernova Glide). The footwear was covered with tape (Hypafix or rigid strapping tape) to cover any reflective material that may be identified by the VICON cameras (Figure 5.2).

Similarly to Joseph et al. (2013) and Hobara et al. (2007) stiff legged hopping was selected to reduce the contribution of the knee and to primarily assess the function of the ankle plantarflexors. The natural frequency of hopping is approximately 2.2Hz, a frequency that is widely used in spring-mass model research. However during piloting, individuals found it extremely difficult to hop at a frequency below 2.6Hz whilst keeping their knees as straight as possible, and for this reason 2.6Hz was the selected frequency. Trials were accepted if the participants hopped within ±2% of the set frequency (Farley & Morgenroth, 1999; Joseph et al., 2013).

Participants were asked which their preferred kicking leg was and the non-preferred leg was selected for data analysis.

During all the hopping trials, kinematic data were recorded using a nine camera VICON three-dimensional motion analysis system at 250Hz. Kinetic force plate data was sampled at 1000Hz with a 20N threshold for ground contact. Kinematic data was filtered using a Woltring filter with a predicted *mean square error* of 10 mm (VICON).

5.3.3 Data Analysis

The kinematic and kinetic variables of interest for all hopping protocols on the level and incline surfaces included knee and ankle angles and moments at initial contact and mid-stance in the sagittal plane. Additionally, kinetic data was used to determine ground contact time, flight time, vertical acceleration and peak force. The hopping data were analysed from the 3rd through till the 10th second of testing. This provided 7 seconds of hopping data (allowing for 15 hops to be analysed), to account for the variability between hops.

The vertical leg stiffness, k_{Vert}, was calculated from the force plate data using peak vertical ground reaction force and COM displacement during the compression phase of the hop, from initial ground contact to maximum leg compression (mid-stance) as follows:

$$k_{Vert} = \frac{\text{Force (peak)}}{\text{Displacement of COM}}$$

Displacement of COM was obtained by integrating the vertical acceleration twice with respect to time as described in previous research (Dalleau, Belli, Viale, Lacour, & Bourdin, 2004; Hobara, Inoue, et al., 2010) (see Appendix G).

Joint stiffness, k_{Joint} was calculated using the torsional spring model. In this model it is assumed that four rigid segments (foot, shank, thigh and head-arms-trunk) are interconnected with torsional joint springs of the hip, knee and ankle (Farley et al., 1998). Similarly to Joseph et al., (Joseph et al., 2013) only the knee and ankle were investigated. Therefore, joint stiffness of the knee and ankle were calculated as,

$$k_{Joint} = \frac{Moment_{Joint}}{\theta_{Joint}}$$

where $Moment_{joint}$ and θ_{joint} are the changes in joint moment and the angular displacement at each joint from initial contact to mid-stance, respectively (see Appendix G).

VICON Nexus software was used to calculate the ankle and knee joint moments in the saggital plane using inverse dynamic equations (Davis, Ounpuu, Tyburski, & Gage, 1991). This involves integrating kinetic, kinematic and anthropometric data to calculate segment masses, segment centre of gravity locations, and segment moments of inertia. This process was completed similarly to Joseph et al. (2013).

<u>5.3.4 Statistical Analysis</u>

For statistical analysis all data was exported into the Statistical Package for the Social Sciences (SPSS, version 22.0, IBM, Somers, New York) and tested for normality, consistent with the methods used in Chapters 3 and 4 of this thesis. Some variables were not normally distributed and due to the small sample size non-parametric statistics were used. To examine whether there were differences between hopping on the incline and the level surface, a Wilcoxon signed rank test was used. Comparison between knee and ankle variables during both hopping protocols was also analysed using a Wilcoxon signed rank test. An alpha level of 0.05 was used to determine significance for all analyses.

5.4 Results

Data was collected from 14 male distance runners with a median age of 35.5 years (Interquartile range (IQR) = 32-39 years), a height of 180 cm (IQR = 174.2-186 cm) and a body mass of 81.6 kg (IQR = 74.6-84.4 kg). Hopping on an incline altered the interplay between the knee and ankle during the task. At initial contact the ankle was more dorsiflexed during incline hopping (level: median = -4.9°, IQR = -8.7- -3.1°, incline: median= -0.7°, IQR = -4.4-2.2°, p=0.011). Similarly, at midstance the ankle was more dorsiflexed during incline hopping (level: median = 17.0°, IQR = 15.3-18.6°, incline: median = 21.8°, IQR = 18.6-24.7°, p=0.001), however the joint angular displacement remained unchanged (level: median = 22.6°, IQR = 20.6-24.1°, incline: median = 23.4°, IQR = 20-24.8°, p=0.551). Consequently, the ankle joint functions in greater degrees of DF during incline hopping (Figure 5.5, individual recording). At initial contact the knee flexion was the same during level and incline hopping (level:

median = 19.5°, IQR = 18.1-21.1°, incline: median = 18.6°, IQR = 16.4-22.3°, p=0.701), however the knee remained significantly more extended on the incline platform during loading (level: median = 29.4°, IQR = 26.9-32.8°, incline: median = 26°, IQR = 24.2-27.9°, p=0.001). Hopping on an incline resulted in the knee joint having reduce joint excursion (level: median = 10.2°, IQR = 7.2-13°, incline: median= 7.2°, IQR = 5.3-9.2°, p=0.001) (Figure 5.6). A reduction in leg stiffness was observed during incline hopping (level: median = 32.2 kN/m, IQR = 29.3-35.2, incline: median = 29.0 kN/m, IQR = 25.9-34.4, p=0.011) (Table 5.1).

Table 5.1 Comparison of level and incline hopping at 2.6Hz (* indicate p<0.05).

| | Level surface | | Incline surface | | |
	median	IQR	median	IQR	p
Peak vertical force (N)	2185	1866-2472	2117	1850-2364	0.109
Leg stiffness kN/m	32.2	29.3-35.2	29.0	25.9-34.4	0.011*
Peak ankle moment Nm/kg	3.7	3.4-4.1	3.8	3.4-4.3	0.722
Peak knee moment Nm/kg	0.4	0.0-0.7	0.2	0.0-0.5	0.131
Ankle stiffness kNm/rad/kg	9.8	9.0-10.5	10.2	8.7-10.4	0.756
Knee stiffness kNm/rad/kg	2.8	0.0-4.4	2.5	0.8-4.7	0.594
Contact time (ms)	276	264-290	276	269-295	0.235
Flight time (ms)	114	99-123	111	95-126	0.730

During the hopping tasks greater moments were observed at the ankle in comparison to the knee on both the level surface (ankle: median = 3.7 Nm/kg, IQR = 3.4-4.1 Nm/kg, knee: median = 0.4 Nm/kg, IQR = 0.0-0.7 Nm/kg, p=0.003) and the incline surface (ankle: median = 3.8 Nm/kg, IQR = 3.4-4.3 Nm/kg, knee: median = 0.2 Nm/kg, IQR = 0.0-0.5, p=0.001) (Figure 5.5). In addition, ankle stiffness was greater than knee stiffness during both hopping on the level surface (ankle: median = 9.8 Nm/kg/rad, IQR = 9.0-10.5 Nm/kg/rad, knee: median = 2.8 Nm/kg/rad, IQR = 0.1-4.4

Nm/kg/rad, p=0.003) and the incline surface (ankle: median = 10.2 Nm/kg/rad, IQR = 8.7-10.4 Nm/kg/rad, knee: median = 2.5 Nm/kg/rad, IQR = 0.8-4.7 Nm/kg/rad, p=0.001) (Figure 5.8). No differences were identified between incline and level hopping for ankle and knee joint stiffness or ankle and knee joint moments (Table 5.1).
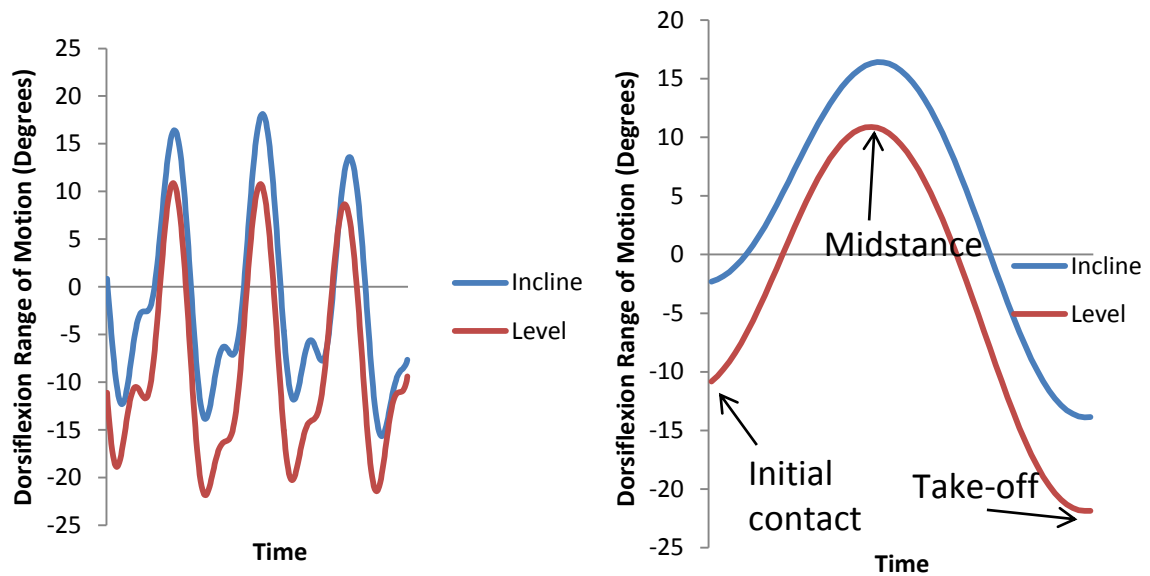
Figure 5.5 (A) Differences in ankle range of movement between hopping on an incline surface and a level surface during three individual hop cycles for a single participant and (B) for a single ground contact phase for the same participant
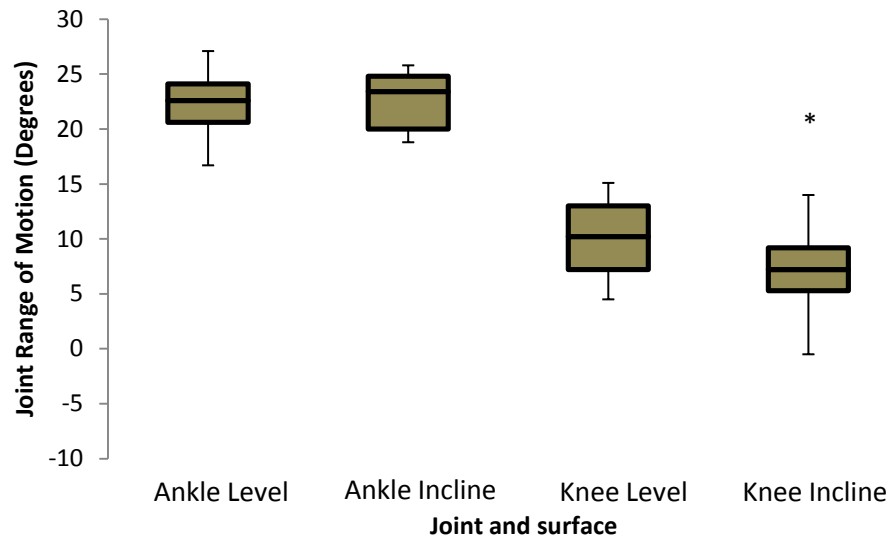
Figure 5.6 Ankle and knee joint excursion during hopping on level and incline surfaces (* indicate p<0.05).
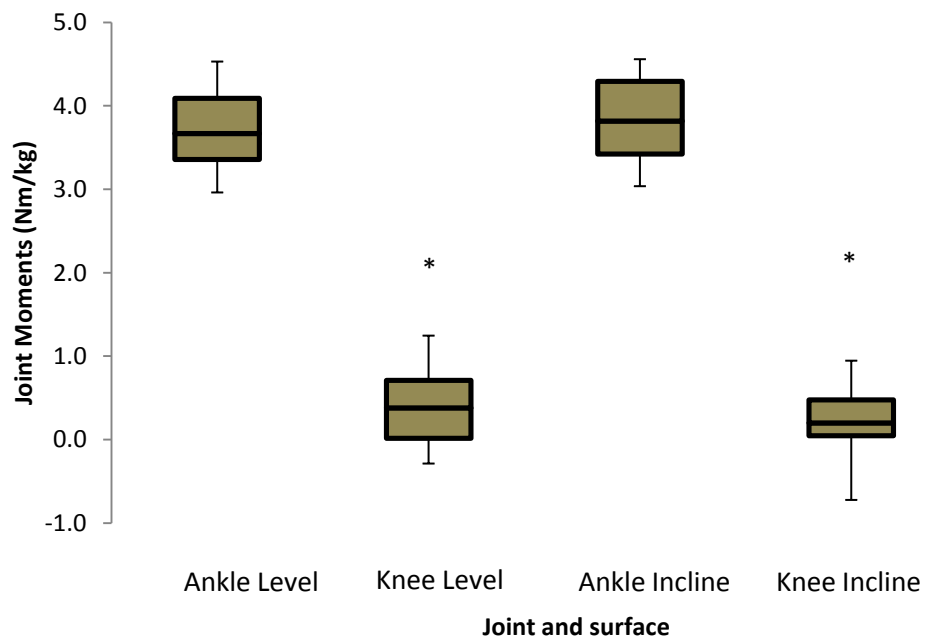
Figure 5.7 Ankle and knee moments during hopping on level and incline surfaces (* indicates p<0.05).
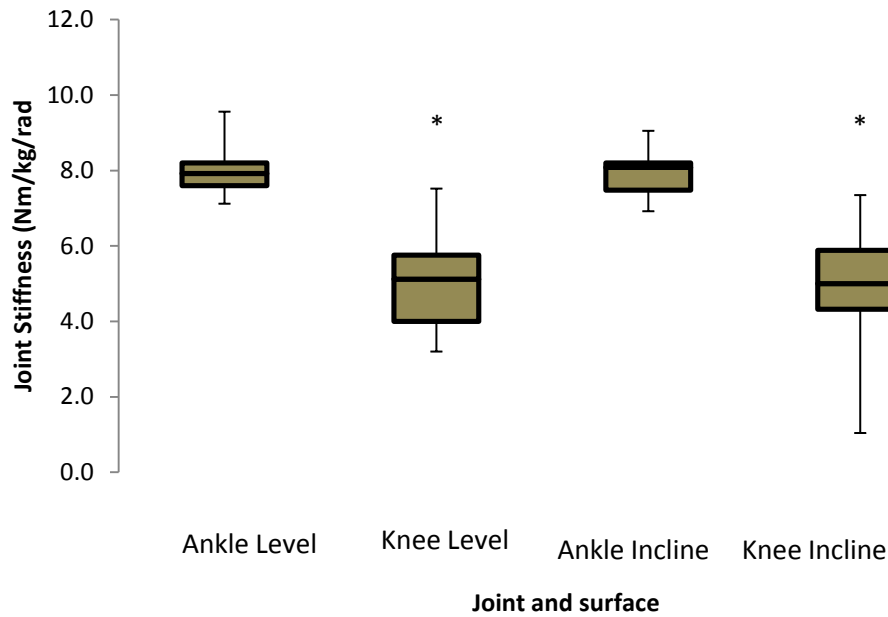
Figure 5.8 Ankle and knee joint stiffness during hopping on level and incline surfaces (* indicates p<0.05).

5.5 Discussion

The results of this study showed that the interplay between the knee and the ankle was different during incline and level hopping. The ankle joint functions in greater ranges of ankle DF throughout the loading phase of incline hopping without any change in angular displacement. The knee joint has less angular displacement during the loading phase of incline hopping. It was identified that although participants were instructed to keep their knee stiff, not only was their knee in flexion at initial contact, but during incline and level hopping the knee joint does not remain straight with some angular displacement of the joint. During incline hopping there was a reduction in vertical leg stiffness however no alterations were identified in joint stiffness at both the knee and ankle.

When hopping on an incline, while aiming to maintain a stiff knee, our results show the ankle typically loads in approximately 4-5° more DF during the stance phase from initial contact to midstance in comparison to a level surface (Figure 5.5). Although, the ankle typically landed and then flexed into greater DF, the range of motion was the same (Figure 5.9).The change in ankle DF is consistent with Kannas et al. (2011) who observed a similar shift with loading in greater angles of DF. Hopping on the incline, although having no increase in joint moments or joint excursions at the ankle, tends to shift the ankle in greater DF ROM in conjunction with reduced knee joint angular displacement. The change observed may place different and potentially greater stresses on the triceps surae musculotendinous unit. In addition to the change of functional ROM, Kannus et al. (2011) identified that the muscle fascicles in the gastrocnemius muscle were elongated at initial contact during incline hopping, however, by midstance there was no difference in the muscle fascicle lengths. The increase in DF ROM during incline hopping may be primarily due to the Achilles tendon increasing in length by midstance as suggested by Kannus et al. (2011). However, an increase in tendon length may also require an increase in muscle force indicated by increased ankle joint moments a finding that was not observed in our study. Although our study did not specifically assess changes in tendon length, given the consistent findings with the previous research (i.e. increase in ankle DF), our findings may have also been as a consequence of elongation of the Achilles tendon. Elongation of the Achilles tendon has potential to both impact loading of the tendon as well as affect the stretch-shortening cycle.
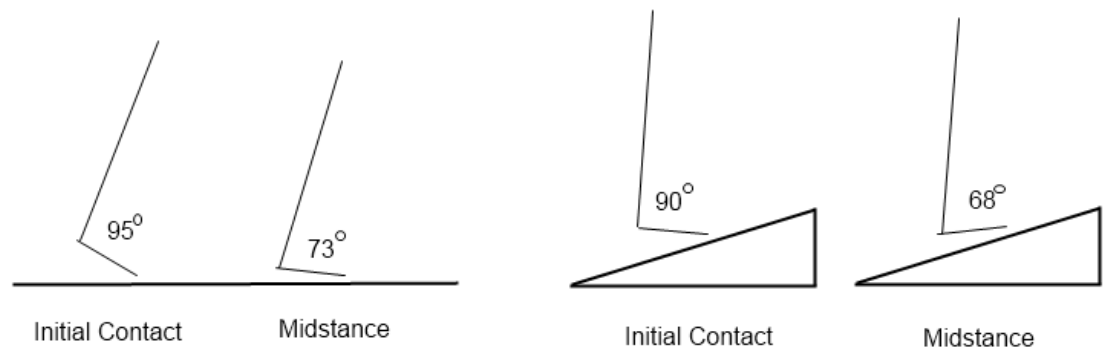
Figure 5.9 A schematic example of ankle angles during initial contact and midstance of hopping on a level surface (left) and incline surface (right).

Knee function during incline hopping is also altered. During ground contact, at midstance the knee remained more extended, resulting in reduced knee joint angular displacement. With a reduction of angular displacement of the knee (Figure 4.7), it would be expected that the knee would be stiffer. In this study, although not significant, the knee moments had a trend to be lower and as a consequence levels of knee stiffness did not increase despite the reduction in knee joint angular displacement. Incline hopping may also increase the joint moments at the hip, therefore further research is warranted to explore the impact of incline hopping at the hip and trunk..

Previous research by Hobara et al. (2007) has suggested that keeping the knee straight would result in minimal if any, contribution from muscles other than those about the ankle. The suggestion by Hobara et al., (2007) has been challenged by the findings of this investigation. The knee joint flexed through approximately 7° and 10° of angular displacement during the loading phase of incline and level

hopping respectively, and the knee typically loads in some flexion at initial contact. Previous research has identified much greater ranges of knee flexion ranging from 5-23° during hopping at frequencies from 2.0Hz to 3.0Hz (Farley & Morgenroth, 1999; Hobara, Inoue, et al., 2010; Hobara, Kimura, et al., 2010) with Joseph et al. (2013) observing as high as 44° of knee angular displacement at 2.2Hz. It is not surprising that the observed angular displacement in our study were lower than the other studies due to the instruction of keeping their knee straight. The key finding from our study was that the knee did not remain static, and therefore future researchers should be cautious in assuming the knee is not contributing to hopping tasks when participants are asked to maintain a stiff knee. This may be an important factor to consider when assessing different populations, especially those with lower limb pathologies. A further challenge to the assumptions of Hobara et al. (2007) was that they assessed participants at a hopping frequency of 2.2Hz as compared to 2.6Hz that was employed in our protocol. The lower frequency during their study would be more likely to result in increased knee flexion angular displacement. This suggests that during their study the contribution of the knee may have been even greater than that identified in this current study. In our study the significantly higher joint moments at the ankle suggests that the ankle was loaded to a greater magnitude in comparison to the knee (Figure 4.8).

Leg stiffness calculated in this study was much higher than that of Kannas et al. (2011) who also investigated incline hopping. The difference in measures may be due to two reasons. Firstly, in our study participants were instructed to keep their knee straight resulting in a stiffer leg spring, with reduced displacement of the COM, and secondly the frequency of hopping was markedly higher in our study, 2.6Hz compared with 1.6Hz. Hopping with a higher frequency results in a greater vertical

leg stiffness (Farley et al., 1991). In addition to the greater levels of leg stiffness measurements identified, our findings were not in agreement with their previous findings that incline hopping does not alter leg stiffness (Kannas et al., 2011). The difference in findings may be a result of different hopping protocols utilised.

Despite the changes in leg stiffness and the kinematics of the ankle and the knee there were no changes in ankle and knee joint kinetics. This may indicate that there was greater involvement from more proximal regions such as the trunk and the hip. In addition to similarities in leg stiffness, incline hopping was not accompanied by a change in joint stiffness. Stiffness at the ankle is proportionally much higher than knee stiffness (Table 5.1), despite less range of motion at the knee. The higher levels in ankle stiffness appears to be caused by much greater joint moments at the ankle in both level and incline hopping as compared to the knee (Table 5.1). These findings are in contrast to maximal hopping that results in larger angular displacement, higher joint moments and greater joint stiffness at the knee as compared to the ankle, resulting in greater demands on the knee extensor elastic component (Hobara et al., 2009). Our study utilised sub-maximal hopping that is predominantly dependant on the ankle, that therefore results in greater use of the plantar flexor elastic component (Farley & Morgenroth, 1999).

Our study revealed an interesting finding on the contact angle of the ankle during both hopping tasks. It was expected that the ankle would contact the ground both on a level surface and the incline in greater degrees of plantarflexion than actually observed and seen in previous studies (Farley & Morgenroth, 1999; Hobara, Inoue, et al., 2010; Joseph et al., 2013; Kannas et al., 2011). On review of the three dimensional video output frame by frame, at initial contact participants appear to have their thigh vertical relative to the ground. Their shin was angled behind (i.e. the

ankle was posterior to the knee) them allowing the forefoot to strike the ground under their COM. These identified observations may have been a postural and positional response to the verbal cue of instructing participants to keep their knee as straight as possible.

A limitation of our study was that measures of electromyography activity of the gastrocnemius and soleus muscles were not recorded. This could have provided further information about the activity of the muscles during the different tasks. Kannas et al. (2011) did examine electromyography activity and observed that greater electromyography activity of the soleus occurred during the propulsive phase of incline hopping. It is not known whether the different hopping protocol in our study (higher frequency, stiff knee) would have the same outcome. It had been observed in this study that knee function changed with incline hopping. Consequently, a change in the load of the triceps surae musculotendinous unit may have been demonstrated by alterations in electromyography activity levels between the mono-articular soleus and bi-articular gastrocnemius muscles. In addition to this, tibialis anterior electromyography activity was not assessed to determine co-activation, which has the potential to affect ankle joint stiffness during hopping. A further limitation for this study was that the hip joint was not examined in this study because the lower limb marker set used for the protocol did not allow for analysis of the hip. Although we were unable to assess the hip similarly to Kuitunen et al. (2011), they justified that that hip stiffness does not appear to play an important role in leg stiffness adjustments. A final limitation to the study was using a homogeneous group that have very similar loading patterns (i.e. distance running) makes it difficult to generalise these findings to all population groups.

5.6 Conclusion

Hopping on an incline altered lower limb loading strategies as compared to level hopping. Incline hopping results in reduced leg stiffness. The ankle joint loads in a more dorsiflexed position and the knee has reduced angular displacement during the loading phase. Incline hopping therefore may increase the load on the Achilles tendon. Incline hopping can therefore be used as a test to explore lower limb biomechanics in lower limb injuries such as Achilles tendinopathy. Further research is required to determine the impact of incline hopping at the hip and trunk.

Chapter Six

Regulation of lower limb stiffness in distance runners with Achilles tendon pathology

6.1 Aims and Hypotheses

- To determine the effects of mid-portion Achilles tendon pathology on lower limb stiffness regulation during level and incline hopping tasks. It was hypothesised that athletes with either Achilles tendinopathy (AT) or asymptomatic Achilles tendon pathology would display reduced leg and ankle stiffness when compared to athletes with normal Achilles tendons.

6.2 Introduction

Running and hopping require the Achilles tendon to transmit force from the triceps surae (calf muscle) to the calcaneus. The Achilles tendon behaves like a linear spring storing elastic strain energy during elongation and then converting this elastic energy into kinetic energy upon recoil (Anderson, 1996; Child et al., 2010; Kyrolainen et al., 2003; Rosager et al., 2002) (Figure 6.1). The Achilles tendon is exposed to very high loads during running and hopping (Chapter 2). These high loads are a potential cause for tendon degeneration and pain, resulting in the clinical presentation of Achilles tendinopathy (AT). There are two classifications of AT. Insertional AT occurs at the region where the tendon attaches to the calcaneus whereas mid-portion AT occurs 2-6cm above the insertion to the calcaneus. Achilles tendinopathy can result in impairments in lower leg muscle-tendon function (Silbernagel et al., 2006) that can compromise training and performance. Despite the prevalence of AT, the biomechanical changes that predispose an athlete to injury or the biomechanical changes that occur as a result of AT remain poorly understood. Investigating the spring-like characteristics of the Achilles tendon during hopping tasks may provide further understanding of biomechanical factors associated with tendon overload and pathology in high volume runners.
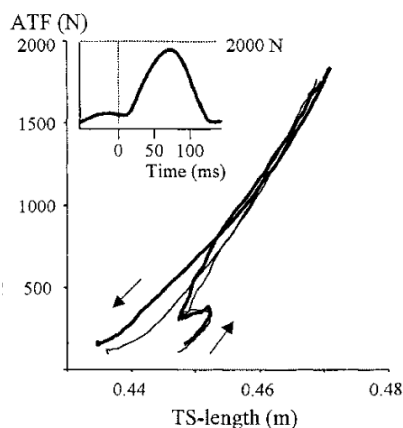
Figure 6.1 Triceps surae muscle-tendon length in relation to Achilles tendon forces (Kyrolainen et al., 2003).

Alterations to the viscoelastic properties of the Achilles tendon may influence ankle performance during elastic loading activities. Changes to the viscoelastic properties of the Achilles tendon in mid-portion AT that have been observed by previous research are firstly, a reduction in tendon stiffness with an increase in strain occurring at the tendon-aponeurosis (Figure 6.2) (Arya & Kulig, 2010; Child et al., 2010; Wang et al., 2012) and secondly, higher levels of hysteresis (energy loss), suggesting a reduced capacity to store energy (Wang et al., 2012). Tendon pathology may alter these mechanical properties of the tendon or alternatively the increased compliance may be involved in the development of AT. Increased hysteresis in combination with reduced tendon stiffness has the potential to reduce the elastic energy storage and increase the energy wasted in AT (Wang et al., 2012). The effect the alteration of tendon properties may have on lower limb mechanics during functional stretch-shortening cycle tasks is largely unknown.
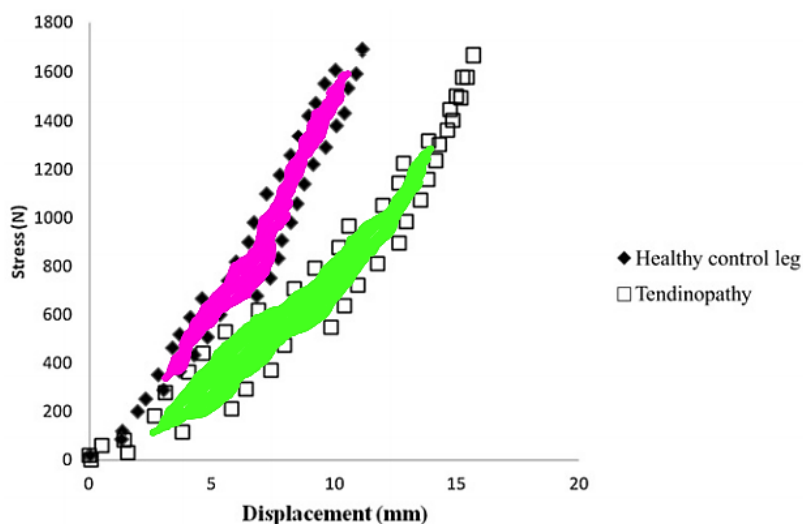
Figure 6.2 A stress-strain curve of the Achilles tendon for a patient with a normal tendon and a pathologic tendon. The highlighted area within the curve (pink = healthy, green = AT) is hysteresis, which indicates the amount of energy loss (from Wang et al., (2012)).

Analysis of the impact that a pathological or painful Achilles tendon has on lower limb biomechanics requires an assessment protocol that challenges the elastic properties of the tendon. A simple test that allows for the elastic properties in the lower limb is hopping. Maquirriain (2012) and Silbernagel et al. (2006) investigated the impact of AT during hopping and observed a reduction in leg stiffness and a reduction in hopping height, respectively. Maquirriain (2012) suggested that the reduction in stiffness was due to an increase in ankle compliance., unfortunately this was not assessed in the either study, therefore, the biomechanical consequence of tendon pathology and a history of tendon pain during elastic loading activities are largely unknown.

Pain is a definitive symptom of AT, and this pain causes muscle inhibition as well as a change in neuromuscular performance (Henrikson et al., 2011). Pain may induce compensatory movement patterns that affect lower limb kinetic chain function

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and are likely to be individual for each athlete. Some may transfer greater loads to other joints in the kinetic chain or alternatively modify loading patterns to the least or non-symptomatic leg. Therefore assessing movement patterns in athletes with AT that may be altered due to structural tendon degeneration is further complicated by the effect of pain, muscle wasting and weakness.

Altered lower limb strategies have been observed in athletes with asymptomatic patellar tendon pathology in jumping athletes during a challenging jumping protocol (Edwards et al., 2010). They observed increased knee flexion, as well as hip extension rather than hip flexion during a horizontal landing task as compared to their control population. These loading strategies suggest a difference in load distribution between the hip, knee and ankle. To date, no research has investigated the impact of symptomatic and asymptomatic Achilles tendon pathology upon lower limb strategies during challenging stretch-shortening cycle tasks, such as hopping on the flat or incline.

Investigating biomechanical variables that are risk factors for AT is difficult. A further complexity in the Achilles tendon is that there is often pathology without symptoms. It is unknown whether Achilles tendon pathology alters lower limb biomechanics, or whether symptoms (i.e. pain) are required to change the mechanics of the lower limb during high Achilles tendon loading activities as previous studies have only examined biomechanics in symptomatic tendinopathy (Azevedo et al., 2009; Child et al., 2010; Maquirriain, 2012). Our study is the first study that compares biomechanical factors associated with Achilles tendon pathology in runners with and without a previous history of pain.

The purpose of this study was to explore the stiffness regulation and joint interaction in the lower limb during level and incline hopping in distance runners with mid-portion Achilles tendon pathology, both symptomatic and asymptomatic. It was hypothesised that runners with symptomatic AT and asymptomatic Achilles tendon pathology would have both reduced leg stiffness and ankle stiffness..

6.3 Methods

6.3.1 Participants

Male runners were recruited for this study and classified into three test groups; symptomatic AT group, an asymptomatic Achilles tendon pathology group and a normal tendon (CONT) group. Participants found to have a mid-portion asymptomatic, yet pathologic, Achilles tendon as well as those with normal tendons on imaging (Chapter 4) were invited to participate in this follow up biomechanical study. To avoid bias, individuals were not told of their tendon status until the completion of the study. The symptomatic AT participants were recruited similarly to Chapter 4. Prior to ultrasound (US) imaging the symptomatic AT group completed the running survey and VISA-A questionnaire (Chapter 4). Then they had their symptomatic AT status confirmed by ultrasonography from the same musculoskeletal radiologist. Diagnostic and inclusion criteria were (1) pain over the Achilles tendon, (2) morning pain or stiffness (3) tenderness and thickening on palpation, (4) a VISA-A score less than 80 and (5) ultrasound findings (thickened and or hypoechoic regions).

The participants in the symptomatic AT group had to fulfil the running criteria (Chapters 4 and 5) with the exception of being symptomatic for a period of at least three months. The symptomatic AT participants were expected to have a VISA-A

score less than 80 points (maximum is 100), a rating previously used in patellar tendinopathy research (Visnes, Hoksrud, Cook, & Bahr, 2005). A VISA-A score of less than 80 aimed to eliminate runners with symptomatic AT who had minimal symptoms. Previous research by Child et al., (2010) recruited a similar population group that had a mean VISA-A score of 70.

The symptomatic AT participants completed a simple pre-test screening task via phone and/or email communication to ensure that the hopping component of this research would not aggravate their symptoms. The testing involved a ten minute warm-up jog, followed by two bouts of hopping on the affected limb(s), for a period of 30 seconds with a five minute rest between sets. During this physical task individuals were allowed to rate their pain during hopping up to a level of 5 on the visual analogue scale, where 0 in no pain and 10 is the worst imaginable pain, but must have subsided by the next morning (Silbernagel, Thomee, Eriksson, & Karlsson, 2007a). An increase in symptoms for the following day or pain that exceeded 5 excluded them from further participation in the study. There were no runners who were excluded following the pre-test screening.

This study was approved by the Australian Catholic University Human Research Ethics Committee (Appendix C). Prior to testing, each participant was informed of the procedures, risks and benefits of the study. Informed consent was then attained (Appendix D).

6.3.2 Procedure, Data Collection and Analyses

The procedure, data collection and analyses for this study were consistent with the methods outlined in Chapter 5 for both the participants with symptomatic AT and asymptomatic Achilles tendon pathology. The participants completed a ten minute running warm-up on a treadmill at a self selected speed. Then they hopped on a single leg for 10 seconds at 2.6 Hz on the level surface and the 10° platform. The right or left leg was randomly selected for the initial 10 second period of hopping, which was then followed up by the other limb for the second period of hopping. To emphasise the function of the triceps surae and to test the triceps surae more rigorously, the participants were instructed to keep their hands across their chest and to keep their knee "stiff" whilst hopping, with maximal effort and without the use of an aid (Hobara et al., 2007). Kinematic and kinetic data was collected using a VICON three-dimensional motion analysis system that was synchronised with a force plate. Footwear was standardised as in Chapter 5. The data collected and analysed in Chapter 4 were the control (CONT) group. The ten minute running warm-up was an important component of the protocol for the symptomatic AT group. The warm-up was aimed to reduce the impact of current pain on the hopping protocol. It is a common pattern in tendinopathy where symptoms will be initially painful, then subside with continuous activity (Kountouris & Cook, 2007).

All data was imported into the Statistical Package for Social Sciences (SPSS, version 22.0, IBM, Somers, New York) and tested for normality. Not all of the data were normally distributed, and due to the small sample size, non-parametric testing was applied. Kruskal-Wallis tests were employed to determine whether a difference existed between the three groups (CONT, symptomatic AT, asymptomatic tendon pathology) and post-hoc pairwise comparisons were performed using a Mann-

Whitney U test with a Bonferroni correction where between-group differences were identified. An alpha level of 0.05 was used to determine significance for all analyses. If any significant differences were identified between any of the groups, the participants with unilateral pathology in the asymptomatic tendon pathology group or the participants with unilateral symptoms in the symptomatic group had their pathological leg and non-pathological leg, or symptomatic leg and non-symptomatic leg compared. A difference between legs of less than 5% was considered small, a difference less than 10% but greater than 5% was interpreted as moderate and a difference greater than 10% was considered large. Finally post-hoc power calculation was done using G*power software (G*power, version 3.1.9.2, University of Kiel, Germany) to determine the power of the sample size and Cohen's d effect sizes for any significant findings. Cohen's effect sizes were interpreted as <0.1 as trivial, 0.1-0.6 as small, 0.6-1.2 as moderate and >1.2 as large (Bradshaw et al., 2010; Joseph et al., 2013; Saunders et al., 2006).

6.4 Results

Data were collected from 38 male distance runners with a median age of 37.0 years (Interquartile range (IQR) = 33.5-44.3 years), height of 177.8 cm (IQR = 173.1-182.5 cm), and a body mass of 75.8 kg (IQR = 71.0-82.4 kg). The CONT group comprised 14 participants and both the asymptomatic tendon pathology group and the AT group consisted of 12 participants (Table 6.1). Fifty percent of participants in the symptomatic AT and asymptomatic tendon pathology groups had unilateral symptoms. The AT group had a median VISA-A score of 75 (IQR= 63-79). Following the warm-up running protocol all participants in the symptomatic AT group reported

no pain during hopping except for one participant who reported 1 out of 10 on the VAS.

Table 6.1 Descriptive details for all participants

	Age (years)		Height (cm)		Body mass (kg)	
	Median	IQR	Median	IQR	Median	IQR
Control (N=14)	35.5	32.0-39.0	180.0	174.2-186.0	81.2	74.6-84.4
Aymptomatic tendon pathology (N=12)	38.5	31.8-42.0	177.0	173.1-182.2	76.0	72.6-80.5
Symptomatic Achilles tendinopathy (N=12)	41.5	34.3-46.8	177.2	167.7-180.7	71.1	69.2-78.1
p value	0.402		0.215		0.100	
Average (N=38)	37.0	33.5-44.3	177.8	173.1-182.5	75.8	71.0-82.4

The kinematic and kinetic variables between the three groups were similar during level hopping except for ankle stiffness (p=0.044, Table 6.2). Post-hoc testing identified lower levels of ankle stiffness in the symptomatic AT group in comparison to CONT group (p= 0.016) (Figure 6.3). During incline hopping there was a difference identified between groups for ankle stiffness and knee flexion angle at midstance (p=0.042, p=0.029 respectively, Table 6.3). Post-hoc testing identified increased knee flexion at midstance in the AT group when compared to the CONT (p=0.009) (Figure 6.4). The symptomatic AT group also had significantly lower levels of ankle stiffness in comparison to the CONT group (p= 0.013) (Figure 6.3).

Table 6.2 Kinematic and kinetic data for all participants during level hopping (*p<0.05)

	Control (N=14)		Asymptomatic tendon pathology (N=12)		Symptomatic Achilles tendinopathy (N=12)		
	Median	IQR	Median	IQR	Median	IQR	p
Peak vertical force (N)	2185	1866-2472	2110	1768-2336	1901	1817-2090	0.300
Peak vertical force/BW (N/BW	26.6	25.0-28.5	26.9	24.8-29.0	26.9	25.1-28.1	0.997
Ankle dorsiflexion contact (°)	-4.9	-8.7- -3.1	-6.8	-9.3- -1.9	-7.7	-11.0- -1.4	0.923
Ankle dorsiflexion midstance (	17.0	15.3-18.6	15.4	12.9-17.8	15.8	14.2-21.7	0.577
Knee flexion contact (°)	19.5	18.1-21.1	21.2	16.6-22.5	21.6	20.7-23.3	0.125
Knee flexion midstance (°)	29.4	26.9-32.8	30.4	28.6-35.8	32.2	28.9-34.9	0.255
Ankle excursion (°)	22.6	20.6-24.1	21.5	19.4-24.5	23.8	23-24.6	0.110
Knee excursion (°)	10.2	7.2-13	10.6	9.1-13.7	10.0	8.0-13.0	0.773
Peak ankle moment (Nm/kg)	3.67	3.36-4.09	3.73	2.90-3.94	3.55	3.17-3.88	0.644
Peak knee moment (Nm/kg)	0.38	0.02-0.71	0.72	0.28-0.91	0.50	0.27-0.85	0.285
Ankle stiffness (Nm/rad/kg)	9.8	9.0-10.5	9.4	7.8-10.7	8.2	7.7-9.2	0.044*
Knee stiffness (Nm/rad/kg)	2.82	<0.1-4.4	3.4	1.5-4.5	2.9	1.9-4.3	0.674
Leg stiffness (kN/m)	32.2	29.3-35.2	30.3	27.8-36.1	29	25.6-33.0	0.246
Leg stiffness/BW (kN/m/kg)	0.41	0.36-0.44	0.41	0.37-0.46	0.39	0.37-0.41	0.500
Contact time (ms)	276	253-282	274	264-279	267	253-281	0.483
Flight time (ms)	114	99-123	112	103-121	114	100-134	0.841

Note: IQR denotes interquartile range

No differences were identified between the asymptomatic tendon pathology group and either the CONT group or the symptomatic AT group. The ankle stiffness of the asymptomatic tendon pathology group during level hopping and incline hopping was typically between the CONT group and the symptomatic group (Figure 6.3). The same observation was identified for knee flexion at midstance during incline hopping (Figure 6.4).

Table 6.3 Kinematic and kinetic data for all participants during incline hopping (*p<0.05)

	Control (N=14)		Asymptomatic tendon pathology (N=12)		Symptomatic Achilles tendinopathy (N=12)		
	Median	IQR	Median	IQR	Median	IQR	p
Peak vertical force (N)	2117	1850-2364	2071	1775-2195	1777	1704-2060	0.220
Peak vertical force/BW (N/kg)	26.7	24.4-27.6	26.5	23.6-26.9	25.3	24.1-27.0	0.702
Ankle dorsiflexion contact (°)	-0.7	-4.4-2.2	-2.5	-6.2-8.5	0.3	-6.5-2.8	0.980
Ankle dorsiflexion midstance (	21.8	18.6-24.7	21.4	17.6-23.4	23.5	18.0-27.3	0.596
Knee flexion contact (°)	18.6	16.4-22.3	21.6	17.9-23.8	21.6	20.6-22.1	0.149
Knee flexion midstance (°)	26.0	24.2-27.9	29.9	25.6-33.8	29.6	27.6-33.6	0.029*
Ankle excursion (°)	23.4	20.0-24.8	22.1	20.3-25.4	23.5	22.1-24.9	0.518
Knee excursion (°)	7.2	5.3-9.2	8.9	6.2-12.5	7.5	6.3-11.5	0.444
Peak ankle moment (Nm/kg)	3.82	3.42-4.29	3.65	3.00-3.98	3.38	3.08-3.85	0.176
Peak knee moment (Nm/kg)	0.20	0.04-0.48	0.57	0.29-0.85	0.28	0.14-0.53	0.078
Ankle stiffness (Nm/rad/kg)	10.2	8.7-10.4	9.2	8.1-10.6	8.1	7.2-9.7	0.042*
Knee stiffness (Nm/rad/kg)	2.5	0.8-4.7	5.4	2.6-6.6	2.3	0.8-3.8	0.068
Leg stiffness (kN/m)	29.0	25.9-34.4	28.4	24.6-33.3	24.6	22.6-28.3	0.136
Leg stiffness/BW (kN/m/kg)	0.38	0.33-0.40	0.37	0.33-0.41	0.34	0.33-0.34	0.364
Contact time (ms)	276	269-295	272	264-285	281	264-289	0.588
Flight time (ms)	111	95-126	113	105-125	106	100-122	0.771
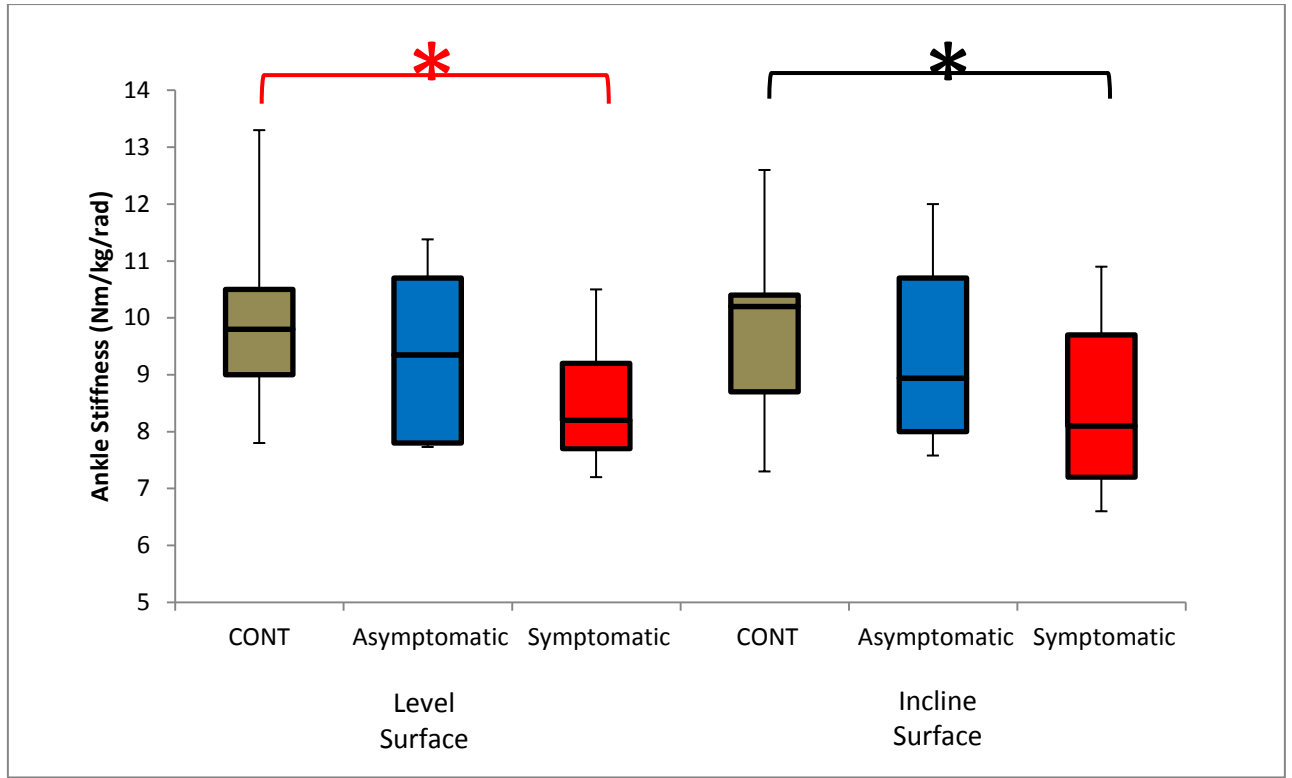
Figure 6.3 Ankle stiffness during level and incline hopping for participants with normal tendons (CONT), asymptomatic Achilles tendon pathology (Asymptomatic) and symptomatic Achilles tendinopathy (Symptomatic) (*p<0.05).
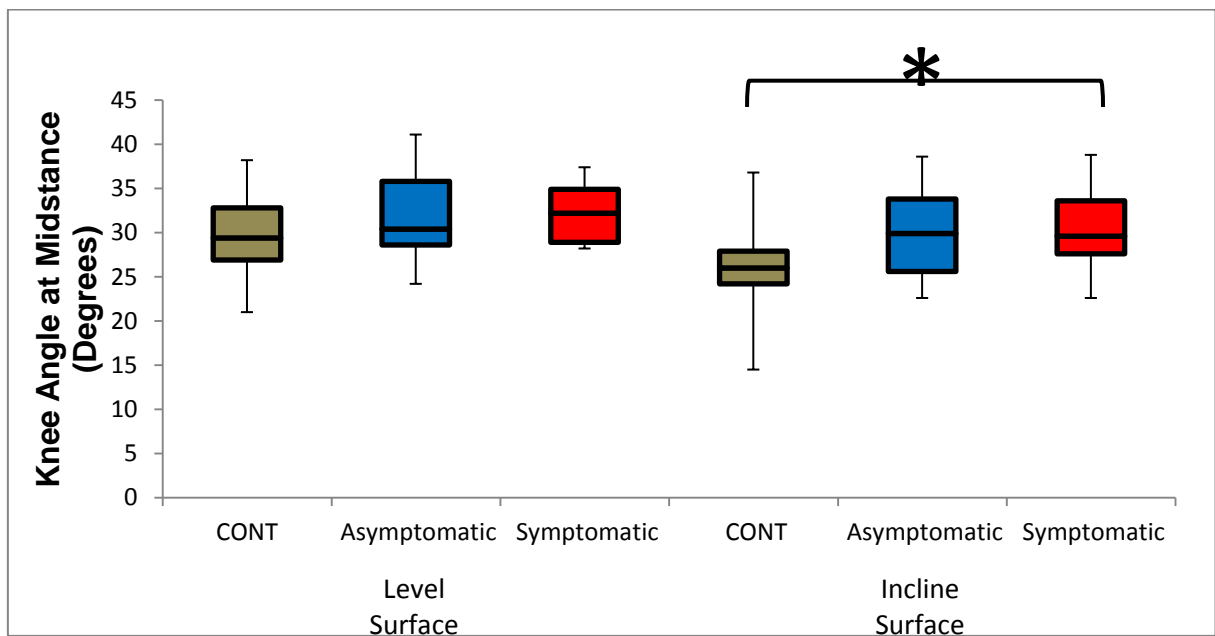
Figure 6.4 Knee flexion at midstance during level and incline hopping for participants with normal tendons (CONT), asymptomatic Achilles tendon pathology (Asymptomatic) and symptomatic Achilles tendinopathy (Symptomatic) (*p<0.05).

Significant differences were identified between the CONT group and the symptomatic AT group for both midstance knee flexion and ankle joint stiffness. Therefore comparison between the symptomatic and uninjured leg in runners with unilateral AT was completed. In the unilateral symptomatic AT participants, the affected leg was compared to the uninjured leg during incline and level hopping for both knee flexion at midstance (incline hopping only) and ankle stiffness (level and incline hopping). The difference between the symptomatic leg and the uninjured leg for knee flexion at midstance in the unilateral group during incline hopping (no difference was identified between groups in level hopping) was typically small to moderate (1-8% difference). Five participants landed with more knee flexion on the asymptomatic leg and a single participant loaded in more flexion on the symptomatic leg. During level hopping three participants had a large (> 10%) difference in ankle stiffness in the symptomatic leg (all measurements were lower in the runners with AT), two participants had a moderately (5% < 10%) less ankle stiffness in the symptomatic leg, whilst one participants had a small difference (< 5%) between the affected and uninjured legs (Figure 6.5). During incline hopping four participants had a large (> 10%) difference in ankle stiffness in the symptomatic leg (all measurements were lower in the runners with AT), whilst two participants had a small difference (< 5%) between the affected and uninjured legs (Figure 6.6).
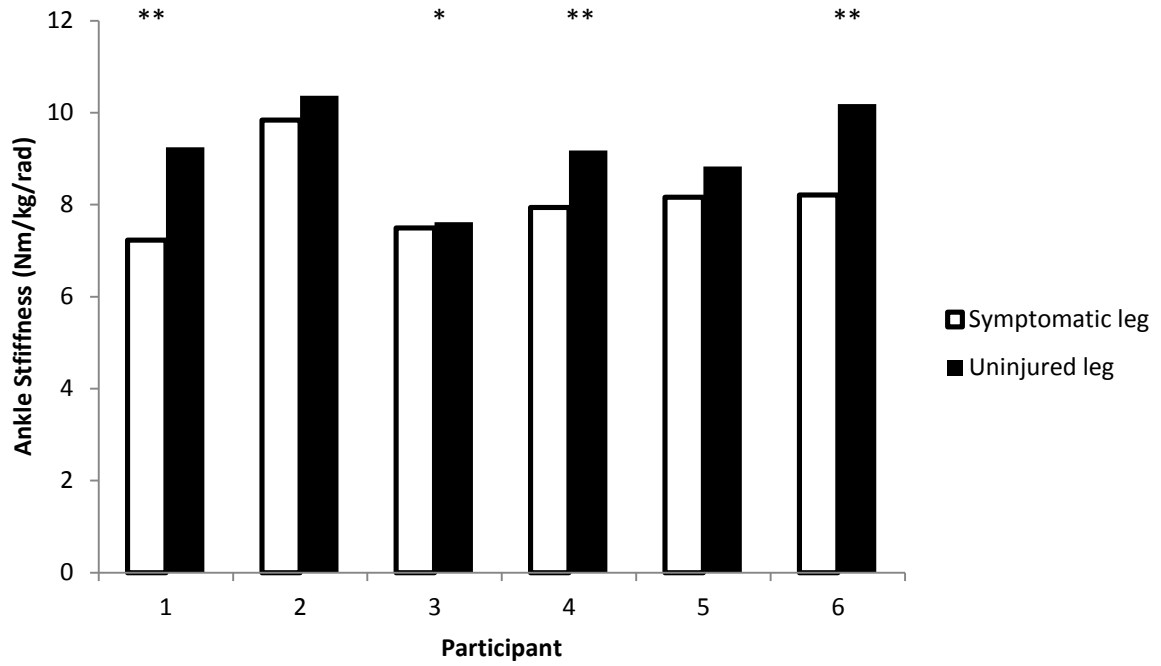
Figure 6.5 Individual ankle joint stiffness measures for participants with unilateral Achilles tendinopathy during level hopping (* difference <5%, **difference>10%).
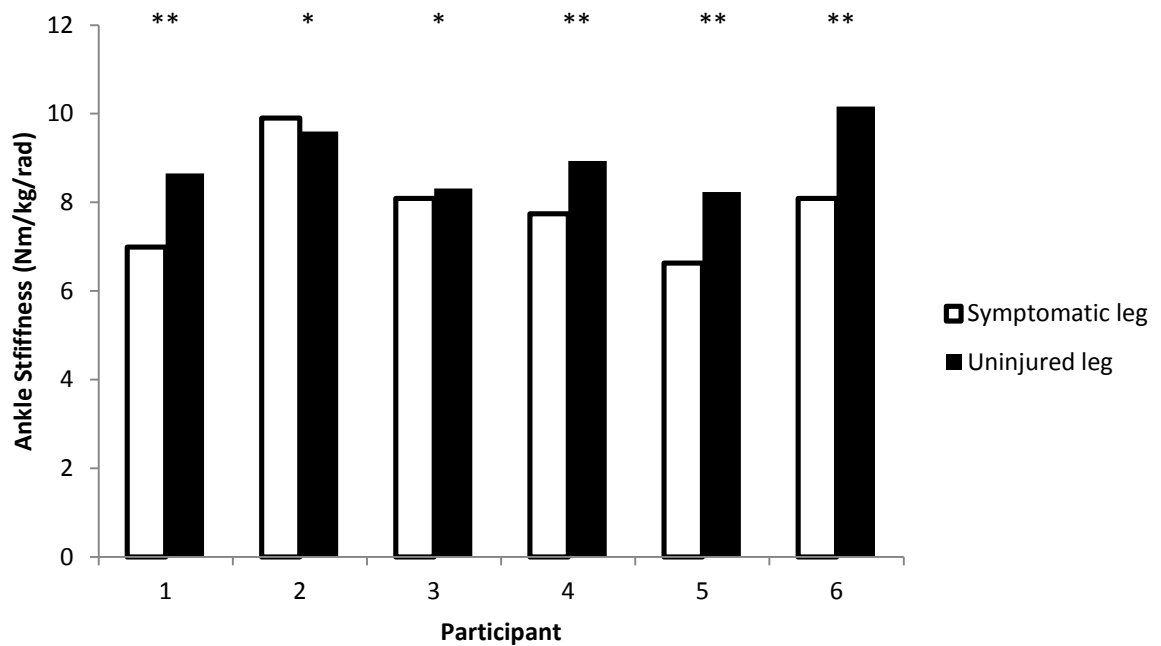
Figure 6.6 Individual ankle joint stiffness measures for participants with unilateral Achilles tendinopathy during incline hopping (* difference <5%, **difference>10%).

Post-hoc power calculations revealed that during level hopping the sample size for determining a difference in ankle stiffness was under powered with a power of 0.68 and a moderate effect size of 1.02. During incline hopping peak knee flexion at midstance was under powered with a power of 0.62 and a moderate effect size of 0.96, however ankle stiffness was under powered with a power of 0.69 and a moderate effect size of 1.04.

6.5 Discussion

Runners with symptomatic AT had different loading strategies for hopping when compared to uninjured runners with no tendon pathology. Specifically, those with symptomatic AT had a strategy of increased ankle joint compliance (lower ankle joint stiffness) during level and incline hopping. Furthermore, runners with symptomatic AT also had greater knee flexion during the limb loading phase (ground contact) during incline hopping. Whilst greater knee flexion often indicates a more compliant joint loading strategy, decreased knee joint stiffness was not identified. Interestingly, runners with symptomatic unilateral AT also typically had increased ankle compliance in the symptomatic limb. In contrast, those in the asymptomatic tendon pathology group did not show any biomechanical differences on either surface when compared to the control group, suggesting a history of pain may be involved in driving these altered movement strategies during hopping.

Ankle joint stiffness is one component of the multi-joint torsional spring model that has been recognised as an important factor that is influenced by AT. Joint stiffness is relative to the change in moment (torque) and the angular displacement from initial ground contact to midstance. A reduction in joint stiffness will occur either

with a reduction in joint moment or an increase in angular displacement, or a combination of both. The reduction in ankle stiffness in the runners with AT appeared to be predominantly driven by reduced ankle joint moments during incline hopping (Table 6.3) and a combination of reduced joint moments and increased angular excursion in level hopping (Table 6.2), albeit these changes were all not significant. The ankle joint angular displacements were similar between groups during incline hopping.

There is limited research that has investigated joint stiffness differences in the presence of an injury, especially AT. Research by Hamill et al. (2009) identified greatest joint stiffness levels at the knee in runners with lower back pain that was primarily a consequence of a reduction in ROM, rather than joint moments during running. They proposed that a knee that is stiffer during running attenuates force poorly, potentially increasing the shock experienced in the lower back (lumbosacral junction). In our running population with AT, the reduction in stiffness may be due to a number of factors acting about the ankle joint. Firstly, the reduction in ankle stiffness may have occurred as a result of changes in neuromuscular performance due to the consequences of current and/or past history of tendon pain. The consequences of pain are that pre-activation of the triceps surae prior to ground contact as well as the stretch reflex may be negatively influenced. This was not investigated in this research but has been shown to be an important factor in stiffness regulation (Hobara et al., 2007). Secondly, tendon pathology may cause dysfunction of the musculotendinous unit due to alterations in tendon compliance and an increase in hysteresis, however leg stiffness may not be directly related to tissue stiffness. Thirdly, a combination of these factors may influence the difference in ankle

stiffness observed. Another potential theory is that the injured runners had lower levels of ankle stiffness that predisposed them to injury.

A significant difference in loading strategies was identified during incline hopping (i.e. both ankle stiffness and greater peak knee flexion at midstance) between the participants with normal Achilles tendons and the runners with symptomatic AT (Figure 6.3 and 6.4), whereas level hopping only influenced ankle stiffness and did not influence knee flexion. This may be explained by the differences observed during incline hopping. Previous research has reported incline loading elongates the Achilles tendon and increases ankle dorsiflexion (DF) throughout the ground contact phase (Kannas et al., 2011). This may then influence the function of gastrocnemius as a bi-articular muscle. A strategy the runner with AT may utilise to reduce the tension on the Achilles tendon is to increase knee flexion. Not only was the knee more flexed at midstance, but there was a trend for the knee to be more flexed at initial contact. This will also potentially reduce the tension on the Achilles tendon. Although not statistically significant, there was also a trend for a similar loading strategy for the asymptomatic tendon pathology group in comparison to the runners with normal Achilles tendons (Table 6.3).

In addition to reduced ankle stiffness, a trend of lower vertical leg stiffness was observed in runners with AT however, the differences in leg stiffness in our study were not statistically significant. This was not consistent with the findings of Maquirriain (2012) who did observe a difference with reduced leg stiffness in active athletes. These differences between studies may be due to their larger sample size, different protocols (i.e. stiff knee hopping versus self selected hopping) resulting in higher levels of vertical leg stiffness in our study or because the reduction in ankle stiffness in our study was not large enough to influence overall leg stiffness.

Furthermore, the participants in Maquirriain's (2012) study had lower VISA-A scores, suggesting a lower level of function and increased pain, which may have also contributed to the different outcomes.

The study included a group of asymptomatic runners with Achilles tendon pathology that was identified using US. These participants were not exposed to the potential effect of previous or current Achilles tendon pain on motor control via compensatory movement patterns or altered motor recruitment. Biomechanical assessment of these asymptomatic runners allowed for identification of altered loading strategies that may be associated with Achilles tendon pathology, which is a potential risk factor for pain associated with AT. The presence of asymptomatic tendon pathology did not identify any significant difference between groups in this study. Whilst there were trends identified, no significant difference were suggesting further research may be warranted with a larger sample size.

To further explore the impact of AT on lower limb kinetics and kinematics, we reviewed the six participants with unilateral AT (only one symptomatic leg). In comparing the asymptomatic and symptomatic legs during level and incline hopping the findings were in agreement with earlier observations in this chapter with a trend towards a reduction in ankle stiffness observed in the symptomatic leg. Interestingly during level hopping one participant had similar ankle stiffness bilaterally (difference <5%), two had moderately less (5%-10%) ankle stiffness in the symptomatic leg and three participants had reductions of greater than 10% in the symptomatic leg. During incline hopping two participants had similar amounts of ankle stiffness bilaterally (difference <5%) and in four participants the symptomatic leg had less ankle stiffness (difference >10%). These findings further suggest that in the presence of

symptomatic AT there is a reduction in ankle stiffness during level and incline hopping in the symptomatic limb. If a reduction in ankle stiffness is a cause or a consequence of AT, the focus of rehabilitation should aim to increase the ankle joint stiffness. Hobara et al. (2008) identified that power trained athletes had an increase in ankle stiffness compared to endurance trained athletes. Other research into the effects of strength training has indicated that strengthening results in an increase in tendon stiffness (Albracht & Arampatzis, 2013; Kubo, Kanehisa, & Fukunaga, 2002). These previous findings suggest that increasing strength and power addresses both the decrease in ankle stiffness identified in this study and the decrease in tendon stiffness identified in previous research by Child et al. (2010) and Wang et al. (2012). Currently rehabilitation that addresses strength, endurance, power and sport specific function is the key to successful restoration of optimal muscle-tendon function (Kountouris & Cook, 2007). This rehabilitation process may have the capacity to increase ankle joint stiffness by increased tolerance to higher loads (e.g. joint moments). Failure to increase the tolerance to higher forces may explain the recalcitrant nature of AT. The Achilles tendon can often take months to recover and there are high recurrence rates of the condition.

In unilateral AT there was no difference identified in knee function. Earlier findings in this study identified that there was a significant difference in knee function between the runners with symptomatic AT and runners with normal tendons. A possible explanation for a change in knee function not being identified in the unilateral AT group may be that alterations at the knee are predominantly a consequence of Achilles tendon pathology. Although not observed in this study, previous research has identified that there is bilateral structural changes in unilateral AT (Docking, Rosengarten, Daffy, & Cook, 2014). Achilles tendon pathology may

alter the mechanical properties of the tendon by increasing compliance and causing higher levels of hysteresis (Child et al., 2010; Wang et al., 2012). Therefore, although the symptoms are unilateral the mechanical properties may be affected bilaterally, resulting in similar movement strategies of the knee (i.e. peak knee flexion).

A limitation of the study was the small sample size of the groups. It was difficult to recruit larger numbers of runners for this study. Post-hoc testing revealed that the study was underpowered. Based on the differences identified between the the symptomatic AT group and the control group, (Cohen's effect size = 0.96) with an alpha of 0.05, and a power of 0.80, a sample size with groups of 19 subjects was required. Large sample sizes are also somewhat prohibitive for a research design like this due to the time and cost involved in processing large quantities of data. Small sample sizes make it difficult to generalise findings to the greater running and sporting population. The homogeneous nature of this population with the symptomatic runners having low levels of symptoms and very similar running histories and current exercise load in comparison with the other participants may also be a limitation to the differences that may be identified between the groups. It is also difficult generalising the findings from our research into populations that are not involved in regular distance running. However this also adds strength to the study because any positive findings have greater power in the investigated population. Other limitations of this study included using hopping instead of running for biomechanical assessment. Although evaluating running biomechanics would be best achieved during task, due to methodological difficulties it would be very difficult to increase the elastic loading at the ankle during running to explore the impact of Achilles tendon pathology and pain. Increasing ankle loading was achieved in our study with straight knee hopping. Proximal regions such as the hip were not

assessed to determine if there may have been an increase in hip joint stiffness to account for the reduction in ankle stiffness. Finally, current training was not controlled due to the varied running programs and upcoming event participation. Training is very difficult to control unless the testing is all done on the same day to a group with the same training volume and intensity with the same race plan, a factor almost impossible to organise.

6.6 Conclusion

Incline hopping results in different loading strategies in distance runners with symptomatic AT. Runners with AT have reduced ankle stiffness and absorb load with a knee that is more flexed during incline hopping. During level hopping runners with symptomatic AT have lower levels of ankle stiffness, but no difference in knee function. It is difficult to speculate whether these changes are a cause or a consequence of AT. The differences identified in level and incline hopping may encourage clinicians to utilise incline hopping as a clinical test if level hopping does not provide them with adequate clinical findings. Further research is required to assess if ankle stiffness in runners with AT will increase following a successful rehabilitation program.

Chapter Seven

Discussion, conclusions and further research

The primary aim of this thesis was to investigate the impact of Achilles tendon pathology on lower limb joint stiffness regulation, but specifically the ankle and the knee. It was identified that the presence of Achilles tendon pathology without a history or presence of pain did not alter lower limb loading strategies. However, differences in these strategies were identified in runners with symptomatic Achilles tendinopathy (AT). In a cross-sectional design such as this study, it is difficult to determine if these differences identified were due to the condition or whether they may contribute to the development of AT.

Alteration of lower leg stiffness regulation was identified in running athletes with AT, but not in running athletes with asymptomatic tendon pathology. Lower levels of ankle joint stiffness during both incline and level hopping was identified. The reduction in ankle stiffness identified may explain the recalcitrant and recurrent nature of AT especially if athletes are unable to increase their ankle stiffness following rehabilitation. Maquirriain (2012) identified a reduction of leg stiffness during hopping with AT, and suggested that this was caused by an increase in ankle compliance. This research is partly in agreement with their conclusion that an increase in compliance (reduced stiffness) at the ankle was responsible for the reduction in leg stiffness. However, our work revealed no statistical difference in leg stiffness, a finding that was not consistent with Maquirriain (2012). The difference may be due to different populations with differing levels of symptoms. The athletes in this study had higher Victorian Institute of Sport Assessment - Achilles Questionnaire scores, indicating higher levels of function with lower levels of pain. Despite the different findings in our research, the trend of the results in our study suggest that athletes with AT have lower levels of vertical leg stiffness when hopping on either a

level or an inclined surface. Further research with larger sample sizes may be required to confirm or challenge the current findings regarding vertical leg stiffness.

Runners with AT flexed their knees more at midstance during incline hopping in comparison to runners (control group) with normal tendons. Similar to the runners with AT, the runners with asymptomatic tendon pathology typically landed with a knee that was more flexed than runners with normal tendons however that was not statistically significant. It has been identified by Kannas et al. (2011) that incline hopping results in elongation of the Achilles tendon, therefore this change in loading strategy by increasing knee flexion may reduce the tension in the Achilles tendon by reducing the length of the bi-articular gastrocnemius.

The secondary aim of this study was to determine the prevalence of Achilles tendon pathology in an asymptomatic running population with no history of tendon pain and to identify differences in loading strategies between level and incline hopping. We identified that there is a high prevalence of tendon pathology in an asymptomatic running population with no history of Achilles tendon pain and that hopping on an incline surface reduced leg stiffness. Almost half (46%) of the runners had at least one tendon that had pathologic changes and 35% of all tendons had abnormalities that were detected on ultrasound (US) imaging. The potential consequences of tendon pathology are an increased risk of developing tendon pain (Comin et al., 2013) and an alteration of lower limb biomechanics (Edwards et al., 2010). Greater years of running was identified as an associated factor of tendon pathology (p=0.024). This is in agreement with Knobloch et al. (2008) who identified that AT was associated with greater duration of load exposure. Although older age is suggestive of a greater lifetime exposure to load, in this group of runners the correlation was only moderate (r=0.518, p=0.001). This suggests that increased age

does not always equate to increased years of running, with many distance runners starting to run later in life. In both the research and clinical setting it is important to consider the age of an athlete, however years of previous loading is also of great importance.

Lower limb stiffness adjustments and biomechanics were assessed during level and incline hopping in the control group. A reduction in leg stiffness was observed during incline hopping however no differences in ankle joint stiffness and knee joint stiffness were seen. This may be explained by more proximal changes at the hip and trunk, components that were not assessed as part of this research. The lower levels of leg stiffness observed was not in agreement with Kannas et al. (2011) who utilised a different hopping protocol. Our research found that during incline hopping the ankle is loaded at initial contact, midstance and take-off in more dorsiflexion (DF) than level hopping. This finding was consistent with previous research (Kannas et al., 2011). There was also reduced angular displacement at the knee during incline hopping. Although a reduction in range of motion (ROM) would be expected to yield an increase in stiffness, this did not occur due to a reduction in joint moments. This reduction in ROM at the knee may however increase the demands placed on the ankle. This would be in agreement with previous research findings that identified increased elongation of the Achilles tendon and increased activity in soleus during incline hopping (Kannas et al., 2011).

The main limitations to this study were firstly the small sample size. Recruiting larger numbers of participants proved to be more difficult than anticipated and therefore further research is warranted. Secondly, it was difficult to make conclusions about the role of years of running as an associated factor of tendon pathology. Years of running does not evaluate previous running load from other running related sports

such as football, soccer or hockey. Thirdly, acquiring three homogeneous groups with similar loading histories and volumes is both a limitation and a strength of the study. It is therefore difficult to generalise the findings, however any findings that are identified are more clinically relevant in this particular population.

In conclusion, there was a high prevalence of tendon pathology in male distance runners with no history of Achilles tendon pain. Cumulative running years was an associated factor of asymptomatic tendon pathology. Male distance runners with AT have reduced levels of ankle stiffness during both level and incline hopping. Consequently when assessing vertical leg stiffness in athletes with AT, vertical leg stiffness alone is not adequately sensitive to identify differences, therefore assessing the knee, but especially the ankle is essential. Athletes with AT alter their lower limb biomechanics to load with increased knee flexion during midstance only when hopping on an incline surface and not when hopping on a level surface. Therefore, both biomechanics researchers and clinicians may use an inclined surface to challenge hopping for lower limb biomechanical assessments because it may be a more sensitive test for the analysis of lower limb injuries.

Further research

These findings warrant further investigation of ankle stiffness in athletes before and after a successful rehabilitation program that is predominantly focussed on improving strength and capacity of the musculotendinous unit. Assessment of athletes who are more symptomatic than this population recruited and are more functionally limited (e.g. unable to run more than 10 km/week) may provide further insight into alterations of lower limb stiffness. In addition long term follow up of

runners that have been identified to have asymptomatic tendon pathology to determine if the presence of structural abnormalities is a risk factor and if any of the assessed factors in this thesis are risk factors for the development of AT.

APPENDICES

Appendix A – Australian Catholic University Human Research Ethics Committee
Approval – Reliability of measuring ankle dorsiflexion

⊕ACU

AUSTRALIAN CATHOLIC UNIVERSITY

Human Research Ethics Committee
Committee Approval Form

Principal Investigator/Supervisor: Elizabeth Bradshaw   Melbourne Campus

Co-Investigators: Melbourne Campus

Student Researcher: Kevin Lieberthal   Melbourne Campus

Ethics approval has been granted for the following project:
Reliability of measuring ankle joint dorsiflexion using a simple tool in weight bearing with the knee flexed and extended

for the period: 05/05/2011-17/07/2011

Human Research Ethics Committee (HREC) Register Number: V2011 35

Special Condition/s of Approval

Prior to commencement of your research, the following permissions are required to be submitted to the ACU HREC:
N/A

The following standard conditions as stipulated in the National Statement on Ethical Conduct in Research Involving Humans (2007) apply:

(i)   that Principal Investigators / Supervisors provide, on the form supplied by the Human Research Ethics Committee, annual reports on matters such as:
   • security of records
   • compliance with approved consent procedures and documentation
   • compliance with special conditions, and

(ii) that researchers report to the HREC immediately any matter that might affect the ethical acceptability of the protocol, such as:
   • proposed changes to the protocol
   • unforeseen circumstances or events
   • adverse effects on participants

The HREC will conduct an audit each year of all projects deemed to be of more than low risk. There will also be random audits of a sample of projects considered to be of negligible risk and low risk on all campuses each year.

Within one month of the conclusion of the project, researchers are required to complete a Final Report Form and submit it to the local Research Services Officer.

If the project continues for more than one year, researchers are required to complete an Annual Progress Report Form and submit it to the local Research Services Officer within one month of the anniversary date of the ethics approval.

Signed:..Date: ....05/05/2011............
          (Research Services Officer, Melbourne Campus)

Appendix B – Informed consent forms (Reliability of measuring ankle dorsiflexion)

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PARTICIPANT CONSENT FORM

Participant's Copy

TITLE OF PROJECT: Reliability of measuring ankle joint dorsiflexion using a simple tool in weight bearing with the knee flexed and extended.

PRINCIPAL INVESTIGATOR (supervisor): Dr Elizabeth Bradshaw

CO-SUPERVISORS: Dr Kade Paterson, Professor Jillian Cook

STUDENT RESEARCHER: Kevin Lieberthal

I .... have read and understood the information provided in the Letter to Participants. Any questions I have asked have been answered to my satisfaction. I agree to participate in this 30 minute assessment of ankle range of movement which I understand will not be audio or videotaped, and data that will be collected will not identify me in any form. I realise and understand that I can withdraw my consent at any time without any adverse consequences. I agree that research data collected for the study may be published or may be provided to other researchers in a form that does not identify me in any way.

NAME OF PARTICIPANT: ....

SIGNATURE .... DATE ....

SIGNATURE OF PRINCIPAL INVESTIGATOR (or SUPERVISOR): ....

DATE:......................................

SIGNATURE OF STUDENT RESEARCHER:...

DATE:..................................

Appendix C – Australian Catholic University Human Research Ethics Committee Approval – The impact of Achilles tendinopathy on lower limb joint stiffness regulation during hopping tasks

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Human Research Ethics Committee
Committee Approval Form

Principal Investigator/Supervisor: Dr Liz Bradshaw

Co-Investigators:

Student Researcher: : Mr Kevin Lieberthal

Ethics approval has been granted for the following project:
The impact of Achilles tendinopathy on lower limb joint stiffness regulation during hopping tasks

for the period: 06/06/2012-31/12/2013

Human Research Ethics Committee (HREC) Register Number: 2012 78V

Special Condition/s of Approval
Prior to commencement of your research, the following permissions are required to be submitted to the ACU HREC:
N/A

The following standard conditions as stipulated in the *National Statement on Ethical Conduct in Research Involving Humans* (2007) apply:

(i) that Principal Investigators / Supervisors provide, on the form supplied by the Human Research Ethics Committee, annual reports on matters such as:
  • security of records
  • compliance with approved consent procedures and documentation
  • compliance with special conditions, and

(ii) that researchers report to the HREC immediately any matter that might affect the ethical acceptability of the protocol, such as:
  • proposed changes to the protocol
  • unforeseen circumstances or events
  • adverse effects on participants

The HREC will conduct an audit each year of all projects deemed to be of more than low risk. There will also be random audits of a sample of projects considered to be of negligible risk and low risk on all campuses each year.

Within one month of the conclusion of the project, researchers are required to complete a *Final Report Form* and submit it to the local Research Services Officer.

If the project continues for more than one year, researchers are required to complete an *Annual Progress Report Form* and submit it to the local Research Services Officer within one month of the anniversary date of the ethics approval.

Signed: ...... *.K. Pashley.* ...... Date: .... 18/09/2014.....
(Research Services Officer, McAuley Campus)

U:\Ethics\APPLICATIONS\1Old Applications\Ethics Application 2012\2012 78V Bradshaw Paterson Lieberthal\2012 78V Approval Form.doc

Appendix D – Informed consent forms (The impact of Achilles tendinopathy on lower limb joint stiffness regulation during hopping tasks –Study A)

ACU
AUSTRALIAN CATHOLIC UNIVERSITY

PARTICIPANT CONSENT FORM

Participant's Copy

TITLE OF PROJECT: The impact of Achilles tendinopathy on lower limb joint stiffness regulation during hopping tasks

PRINCIPAL INVESTIGATOR (supervisor): Dr Elizabeth Bradshaw

CO-SUPERVISORS: Dr Kade Paterson, Professor Jillian Cook

STUDENT RESEARCHER: Kevin Lieberthal

I .... have read and understood the information provided in the Letter to Participants. Any questions I have asked have been answered to my satisfaction. I agree to participate in this study that has been detailed in the Letter to participants. I agree to participate in this 30 minute assessment of my Achilles tendon that will be assessed by Ultrasound. I understand that my ankle range of movement and waist will be measured. I will then be required to complete a running history survey and Achilles questionnaire. I agree to being randomly invited to participate in a second study that has been detailed in the letter to the participant. I understand that the second study is approximately a 2 hour biomechanical assessment that involves running and a number of hopping tests. I understand that I will not be audio or videotaped, and data that will be collected will not identify me in any form. I realise and understand that I can withdraw my consent at any time without any adverse consequences. I agree that research data collected for the study may be published or may be provided to other researchers in a form that does not identify me in any way. I agree to be contacted by email or phone in a year for a follow up.

NAME OF PARTICIPANT: ....

EMAIL...

PHONE..

SIGNATURE ....   DATE ....

SIGNATURE OF PRINCIPAL INVESTIGATOR (or SUPERVISOR):...

DATE:..

SIGNATURE OF STUDENT RESEARCHER:...

DATE:..

Appendix E – Running history survey

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Appendix I

Running Survey

PLEASE COMPLETE THIS QUESTIONNAIRE TO THE BEST OF YOUR KNOWLEDGE.

Email:..

Phone Contact:...

1. What is your age today?

2. How many years have you been running for?

3. How many sessions do you run each week?

4. How many kilometres do you run each week?

5. How many half marathons have you run?

6. How many marathons have you run?

7. When was your most recent half or full marathon run?

8. Have you had any lower limb injury that has forced you to stop running for a period of greater than one week in the last six months?

9. Have you ever had a lower limb injury that has sidelined you for a period of at least six weeks? If yes please explain further (diagnosis, duration, recurrence, management). Include details of all injuries if more than one.

Appendix F – VISA-A Questionnaire

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The VISA-A questionnaire

IN THIS QUESTIONNAIRE, THE TERM PAIN REFERS SPECIFICALLY TO PAIN IN THE ACHILLES TENDON REGION

1. For how many minutes do you have stiffness in the Achilles region on first getting up?

100 mins — 0 mins

0 1 2 3 4 5 6 7 8 9 10

2. Once you are warmed up for the day, do you have pain when stretching the Achilles tendon fully over the edge of a step? (keeping the knee straight)

Strong severe pain — No pain

0 1 2 3 4 5 6 7 8 9 10

3. After walking on flat ground for 30 minutes, do you have pain within the next 2 hours? (If unable to walk on flat ground for 30 minutes because of pain, score 0 for this question).

Strong severe pain — No pain

0 1 2 3 4 5 6 7 8 9 10

4. Do you have pain walking downstairs with a normal gait cycle?

Strong severe pain — No pain

0 1 2 3 4 5 6 7 8 9 10

5. Do you have pain during or immediately after doing 10 (single leg) heel raises from a flat surface?

Strong severe pain — No pain

0 1 2 3 4 5 6 7 8 9 10

6. Do you have pain during or immediately after doing 10 single leg hops?

Strong severe pain/unable — No pain

0 1 2 3 4 5 6 7 8 9 10

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7. Are you currently undertaking sport or other physical activity?
0 Not at all
4 Modified training ± modified competition
7 Full training ± competition but not at same level as when symptoms began
10 Competing at the same or higher level as when symptoms began
POINTS ☐

8. Please complete EITHER A, B or C in this question.
If you have no pain while undertaking Achilles tendon loading sports please complete Q8a only.
If you have pain while undertaking Achilles tendon loading sports but it does not stop you from completing the activity, please complete Q8b only.
If you have pain which stops you from completing Achilles tendon loading sports, please complete Q8c only.

A. If you have no pain while undertaking Achilles tendon loading sports, for how long can you train/practise?

Nil	1-10 mins	11-20 mins	21-30 Mins	>30 mins
0	7	14	21	30

OR

B. If you have some pain while undertaking Achilles tendon loading sport, but it does not stop you from completing your training/practice for how long can you train/practise?

Nil	1-10 mins	11-20 mins	21-30 Mins	>30 mins
0	4	10	14	20

OR

C. If you have pain that stops you from completing your training/practice in Achilles tendon loading sport, for how long can you train/practise?

Nil	1-10 mins	11-20 Mins	21-30 Mins	>30 mins
0	2	5	7	10

TOTAL SCORE (/100)

Appendix G – Calculations (leg stiffness, joint stiffness)

Leg stiffness

To calculate leg stiffness COM displacement needs to be calculated.

To determine COM displacement, the touchdown velocity of the COM needs to be calculated at the point of initial contact. This was determined from the previous flight phase for each individual hop. The COM is assumed to reach its highest point at the middle of the flight phase where the velocity is 0 m/s prior to free fall. Acceleration is determined by gravity (9.81 m/s²) and time is half of the flight duration, therefore:

$$\text{Final velocity} = \text{Initial velocity} + (\text{Acceleration} \times \text{Time})$$

$$\text{Touchdown velocity} = 0 + 9.81 \times \frac{\text{Flight time (seconds)}}{2}$$

The vertical acceleration of the COM was then calculated by subtracting 1 bodyweight from the from the ground reaction forces.

$$\text{Resultant force} = \text{Ground reaction force} - \text{Bodyweight}$$

The vertical acceleration was then calculated by modifying the equation Force=Mass x Acceleration. Therefore:

$$\text{Vertical acceleration} = \frac{\text{Resultant force}}{\text{Mass}}$$

Integrating the acceleration with respect to time to get the vertical velocity was then completed. Then average velocity at each time interval was calculated using the touch-down velocity and final velocity, thereafter the average of the initial velocity (point before) and final velocity (next point). This generated the velocity-time curve.

$$\text{Velocity} = \text{Initial velocity} + \text{Acceleration x time}$$

Then finally the integration of the velocity curve was completed to determine the final displacement of the COM.

$$\text{Displacement} = \frac{\text{Velocity initial} + \text{Velocity final}}{2} \text{ x time}$$

If the peak ground reaction force and peak leg compression did not occur simultaneously, leg stiffness was calculated as a ratio of peak ground reaction force and COM displacement at the point of peak ground reaction force.

<u>Joint angular displacement</u>

Joint angular displacement = Midstance angle - Initial contact angle

Where initial contact is the point where the threshold of 20N occurs as the foot touches the force plate and midstance is the point that peak force occurs where the COM is at its lowest point

<u>Joint stiffness</u>

For joint stiffness calculations angles were converted to radians

$$\text{Radians} = \text{Degrees} \times \frac{\pi}{180°}$$

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