School of Physiotherapy and Exercise Science

'Spring Theory'- The Role of the Ankle during Sub-Maximal Hopping and its Modulation by Pathology, Fatigue and Training

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DECLARATION

To the best of my knowledge and belief this thesis contains no material previously published by any other person except where due acknowledgment has been made.

This thesis contains no material which has been accepted for the award of any other degree or diploma in any university.

Human Ethics (For projects involving human participants/tissue, etc.) The research presented and reported in this thesis was conducted in accordance with the National Health and Medical Research Council National Statement on Ethical Conduct in Human Research (2007) – updated March 2014. The proposed research study received human research ethics approval from the Curtin University Human Research Ethics Committee (EC00262), Approval Numbers # HR 151/2009 (Study 1), HR28/2010 (Study 2) and HR17/2010 (Study 3 & 4)

James R Debenham 21 October 2016

THESIS SYNOPSIS

The understanding of human locomotion has been simplified by the spring mass model (SMM). The SMM is a biomechanical representation of the lower limb which describes its spring-function to produce bouncing terrestrial gait for running and hopping. Intimately linked to the SMM is the stretch shortening cycle (SSC), a neuromechanical phenomenon, where during the ground contact phase of gait, passive and active elements of the lower limb spring interact in a co-ordinated manner to produce locomotion that is efficient in nature and simplified in its execution. Collectively, lower limb behaviour of this nature in functional and clinical settings can be termed 'Spring Function'.

Spring function can be regulated in real-time in response to changes in task (e.g. running faster) and environment (e.g. change in ground surface quality) in order to optimise locomotor efficiency in real time. In addition, spring function can be modulated in response to a variety of positive (e.g. training) and negative (e.g. pathology) factors that result in a systematic shift in the parameters of spring function, although our understanding of the modulation of spring function is currently incomplete. During sub-maximal SSC-tasks such as running and hopping, the ankle is the region where lower limb spring function is regulated, but there is an absence of knowledge regarding specific changes in ankle mechanics in response to SSC-modulation. As such, the focus of this thesis was to specifically explore the modulation of spring function in response to a series of clinical and experimental circumstances that impact human function as it relates to SSC activities such as running. The first study of this thesis (Study 1) is essentially a methods paper, where a protocol involving a single-leg sub-maximal hopping task performed upon a custom-built sledge jump system (SJS) is described and its reliability established. Subsequently, a series of related clinical and experimental conditions that affect spring function are presented; Achilles tendinopathy (Study 2), plantarflexor fatigue (Study 3), and eccentric loading of the plantarflexors (Study 4). The Discussion of this thesis compares and contrasts the collective thread of these studies, before presenting a novel theoretical model for understanding clinical aspects of spring function, termed 'Spring Theory'.

The first step of the thesis was to establish robust methods upon which to base future studies. For this, sub-maximal hopping was chosen as the measurement task of choice. In order to facilitate close inspection of sagittal plane ankle mechanics during a low load SSC-task under precise conditions, all but one degree of freedom was removed and

fatigue was eliminated by the utilisation of a custom-built SJS. Given the novelty of this task and the potential confounding impact of familiarisation and fatigue, measurement reliability was established within 3 temporal frames: 1) within one trial lasting 30 seconds; 2) between 10 consecutive trials; 3) between 2 testing occasions separated by 1 week. Vertical leg spring stiffness (k_{vert}) and joint kinematics were measured using a three-dimensional (3D) motion analysis system and analysed for temporal reliability. Comparisons using a three-level intraclass correlation coefficient model were made within hopping trials, between multiple trials and between the 2 hopping occasions. With the exception of knee stretch amplitude, no significant differences were present within trials. Across trials, k_{vert}, ankle angle 80 ms pre-contact, ankle angle at contact and ankle stretch amplitude all demonstrated strong reliability (ICC's = 0.77, 0.86 and 0.71 respectively); ankle angle at take-off demonstrated moderate reliability (ICC = 0.68); knee stretch amplitude and hip stretch amplitude demonstrated poor reliability (ICC = 0.11 and 0.28 respectively). At the conclusion of this preliminary study it was deemed that the methods developed were robust and reliable for investigating SSC behaviour.

Study 2 was conducted based on the premise that collectively the current evidence incompletely describes how spring-function changes in accordance with Achilles tendinopathy (AT). This was achieved by comparing spring function in individuals with AT and healthy volunteers. Using a between-subjects case-controlled design, fifteen participants with AT (mean age 41.2 ± 12.7 years) and eleven healthy volunteers (CON) (mean age 23.2 ± 6.7 years) performed sub-maximal single-limb hopping on the SJS. Utilising methods developed in study 1, temporal kinematic (k_{vert}, ankle angle at 80 ms pre-contact, ankle angle at contact, peak ankle angle, ankle stretch amplitude) and surface electromyographic (sEMG) measures (onset, offset and peak times relative to contact) were captured. Data between AT and CON were compared statistically using a linear mixed model. Patients with AT exhibited significantly increased k_{vert} when compared to healthy volunteers ($p<0.001$) and their hopping range was shifted towards a more dorsiflexed position ($p<0.001$). Furthermore, ankle stretch amplitude was greater in AT compared with healthy volunteers ($p<.001$). A delay in muscle activity was also observed; soleus onset ($p<.001$), tibialis anterior peak ($p=0.026$) and tibialis anterior offset ($p<.001$) were all delayed in AT compared with CON. These findings indicated that individuals with AT exhibited altered spring function during sub-maximal hopping when compared with healthy volunteers. It is suggested that these adaptations represented a neuromuscular attempt by the spring system to reduce exposure to the threatening

stimulus (ground contact), whilst lacking the neuromuscular apparatus (e.g. musculotendinous stiffness) with which to do so. These findings have implications for understanding the pathogenesis, clinical features and rehabilitation of this challenging condition.

Study 3 considered aspects of fatigue and spring function, with potential applications to the understanding of the pathogenesis of AT. The role of fatigue in injury development is an important consideration for clinicians. In particular, the role of eccentric fatigue in SSC-activities may be linked to overuse lower limb conditions. The purpose of this study was to explore the influence of ankle plantarflexor eccentric fatigue on spring function during a sub-maximal hopping task in healthy volunteers. Eleven healthy volunteers (23.2 ± 6.7 years) performed the above-described hopping task with 3D motion capture and sEMG utilised to measure k_{vert}, temporal kinematic measures and muscle timing measures at baseline and immediately following an eccentric fatigue protocol with a linear mixed model used to test whether measures differed between conditions. Compared to baseline, eccentric fatigue induced increased k_{vert} during the hopping task (+ 15.3%; $P<0.001$). Furthermore, ankle stretch amplitude decreased (- 9.1%; $P<0.001$), whilst all other ankle kinematic measures remained unchanged. These changes were accompanied by a temporal shift in onset of activity in soleus and tibialis anterior muscles (- 4.6 to - 8.5%; $p<0.001$), suggesting that eccentric fatigue alters spring function in healthy volunteers. It is suggested that these adaptations represent a neuromuscular attempt to protect spring function in a reactive manner to the local fatigue. These findings have implications in terms of understanding the nature of spring function modulation and also the pathogenesis of overuse injuries such as AT.

The objective of Study 4 was to investigate how eccentric loading may influence spring function during the same sub-maximal hopping task, with a view to exploring whether the mechanisms underpinning this popular and efficacious intervention exert their positive influence due to changes in spring function. To do so, k_{vert}, sagittal plane ankle kinematics, and temporal muscle activity of the agonist (soleus) and antagonist (tibialis anterior) muscles were measured during sub-maximal hopping on the SJS in eleven healthy adults. Differences were compared using a linear mixed model before and 7 days after a single eccentric loading intervention. Following the intervention, peak ankle angle shifted to a less dorsiflexed position by 2.9° (44%) ($p<0.001$) and ankle angle 80ms pre-contact decreased by 4.4° (20%) ($p<0.001$). Vertical stiffness significantly increased

+15% ($p<0.001$). sEMG measures of soleus occurred 44%, 17% and 14% earlier for onset, peak and offset value respectively. Tibialis anterior onset was delayed by 13% ($p=0.03$), tibialis anterior peak was not significantly different ($p=1.00$) and tibialis anterior offset was earlier ($p<0.001$), suggesting that eccentric loading does alter spring function in healthy volunteers. It is suggested that these adaptations represent a positive neuromuscular adaptation that provide the spring system with enhanced spring 'capacity', conferring protection upon the Achilles tendon via the 'buffering' mechanism. These findings have implications in terms of understanding spring function modulation in response to training and the mechanisms underpinning eccentric loading.

The collective findings of all these studies provide insight into the manner by which human movement responds to a variety of clinically relevant scenarios. The primary finding of this thesis was that under all conditions (pathology, fatigue, and training) k_{vert} increased. It is speculated that despite the different contexts, stiffness increases to provide the system with protection and an efficient means of locomotion. Under the threatening conditions of pathology and fatigue, it is suggested that individuals stiffen the area in attempt to protect the area ('reactive-protective'), whereas following a loading intervention, stiffness increases as a result of positive changes to the neuromechanical environment ('proactive-protective'). In both instances, it is suggested that the increase in stiffness provides protection to the region whilst optimising locomotor efficiency. Kinematic and sEMG variables varied depending on the condition and did so in a manner that is consistent with certain theories of the pathogenesis and managements of overuse lower limb injuries. In participants with AT the increased peak dorsiflexion and stretch amplitude (accompanied by hastened sEMG activity) may represent a failed attempt of plantarflexor performance to limit excursion into dorsiflexion. In the fatigued state, decreased stretch amplitude and hastened sEMG activity may represent the successful attempt of the plantarflexors to continue executing the task safely under acute stress. Finally, following eccentric loading, the shift of ankle kinematics to a more plantarflexed position without a clinically meaningful change in sEMG activity most likely reflects a positive mechanical adaptation in the plantarflexor neuromuscular unit to protect the ankle from yielding into a more dorsiflexed position.

The findings of this thesis have several independent yet relatable implications regarding lower limb function. Firstly, the findings of Study 2 (AT- increased stiffness and increased peak dorsiflexion) has implications in terms of informing the understanding of the

pathogenesis of AT and its subsequent management. Associations between these measures do not imply causation, but there is biological plausibility to the suggestion that training and rehabilitation might be justified in aiming to positively address these potentially maladaptive features. Secondly, the findings from Study 3 (Fatigue- increased stiffness and decreased stretch amplitude) inform our understanding of motor control and its response to fatigue as a potential legitimate threat. Finally, the findings from Study 4 (Loading- increased k_{vert} and decreased ankle excursion) represent an adaptive response to eccentric loading, supporting the theory that mechanisms underpinning eccentric loading for tendinopathy are in part via improved 'buffering' of the tendon. These findings suggest that eccentric loading alters spring function in a manner reflective of improved motor performance. In addition, with a deeper understanding of the interdependence of these modulatory relationships, an opportunity is provided to present a theoretic model that connects these findings and simplify these complex phenomena. It is suggested in this thesis by 'Spring Theory', that if the lower limb under functional and pathological conditions is considered as an adaptable spring, understanding the pathogenesis, clinical features and management is simplified and optimised for clinicians, educators and researchers.

In summary, the understanding of the modulation of spring function was incomplete; in particular, until now, close inspection of the ankle had received little attention. This thesis provides insight into how in response to the modulatory influences of pathology, fatigue and a loading intervention, spring function alters in a manner that reflects adaptive control strategies in response to threat and adaptive mechanical strategies in response to therapeutic loading. These findings have implications in the understanding both of the physiology of human movement, but also in informing pathogenetic models of pathology and mechanisms of efficacy for therapeutic exercises. These findings are novel and significant in this field.

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"…think like a man of action, act like a man of thought."

Henri Bergson, French Philosopher (1937)

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PUBLICATIONS

JOURNAL ARTICLES

Debenham JR, Travers MJ, Campbell A, Gibson, WI, Grisbrook T, Bulsara M. (2015) The reliability of a sledge jump system for measuring stretch-shortening cycle behaviour during sub-maximal single-limb hopping. Under preparation.

Debenham JR, Travers MJ, Gibson W, Campbell A, Allison GT. (2014) Achilles tendinopathy alters stretch shortening cycle behaviour during a sub-maximal hopping task. J Sci Med Sport 19(1):69-73

Debenham JR, Travers M, Gibson W, Campbell A, Allison GT. (2016) Eccentric fatigue modulates stretch-shortening cycle effectiveness - a possible role in lower limb overuse injuries. Int J Sports Med 37(1):50-55

Debenham JR, Gibson WI, Travers MJ, Campbell A, Allison GT. (2016) Eccentric loading of the triceps surae modulates stretch shortening cycle behaviour- A possible therapeutic mechanism. J Sport Rehab (2017. 26(2):151-158

CONFERENCE PROCEEDINGS

Debenham J, Travers M, Gibson W, Campbell A, Allison GT (2014). Achilles tendinopathy alters stretch shortening cycle behaviour during a sub-maximal hopping task. International Scientific Tendinopathy Symposium, Oxford UK

Debenham J, Travers M, Gibson W, Campbell A, Allison GT (2014). Eccentric fatigue alters stretch-shortening cycle behaviour during a sub-maximal hopping task. International Scientific Tendinopathy Symposium, Oxford UK

ADDITIONAL PUBLICATIONS RELEVANT TO THE THESIS BUT NOT FORMING PART OF IT

Travers M, Debenham J, Gibson W, Campbell A, & Allison G (2013) Stability of lower limb minimal perceptible difference in floor height during hopping stretch-shortening cycles. Physiological Measurement, 34(10), 1375 - 1386.

Grisbrook TL, Cui L, Travers MJ, Debenham JR, Allison GT (2015) Estimates of leg stiffness during low-load plyometrics. J Biomech (Submitted)

Wellisch M, Hamer P, Hopper L, Debenham JR (2015). Eccentric loading positively shifts peak torque angle of the ankle plantarflexors in healthy volunteers. International Journal of Sports and Exercise Medicine.;1:2

STATEMENT OF CONTRIBUTION

The author contributed to the development of the research idea, methods, pilot testing, recruitment, data collection, data management, data analysis and preparation of all chapters within this manuscript and the associated publications.

James Debenham, January 2017

LIST OF ABBREVIATIONS

AT	Achilles tendinopathy
CNS	Central nervous system
DOMS	Delayed onset muscle soreness
EIMD	Exercise-induced muscle damage
k_{vert}	Vertical leg spring stiffness
m	Body mass
MIVC	Maximal voluntary isometric contraction
RFE	Residual force enhancement
sEMG	Surface electromyography
SJS	Sledge jump system
SMM	Spring mass model
SSC	Stretch shortening cycle
SSR	Spinal stretch reflex
t_c	Contact time
t_f	Flight time
VISA-A	Victorian Institute of Sport Assessment- Achilles

CONTENTS

1. INTRODUCTION AND THESIS OBJECTIVES

1.1. SPRING THEORY- A UNIFYING MODEL FOR ACHILLES TENDINOPATHY

In the past decade, attention has shifted towards the global impact of the musculoskeletal burden of disease as being the highest contributor to morbidity in developed nations, and second only to childhood infectious diseases worldwide (Murray et al., 2012). This burden is shifting in part due to our ageing population, in which the incidence of musculoskeletal conditions rises (Vos et al., 2012). As such, there is a growing social requirement to improve our understanding of all musculoskeletal conditions in order to better prevent and manage them once established. It is clear that whilst exercise is responsible for the prevention and treatment of many serious health conditions (Trost et al., 2014), it is ironically responsible for the development of other conditions, principally those sustained due to the participation in sports and exercise. One condition, prevalent in the active ageing population is Achilles tendinopathy (AT) (Longo et al., 2009); no more or less important than other conditions, it represents a common overuse lower limb condition that is yet to be fully understood in terms of its development and subsequent management. As such, further study of this condition is warranted.

Overuse injuries, including tendinopathies, represent 7% of all primary care visits (Skjong et al., 2012) and 30-50% of lower limb injuries (Sobhani et al., 2013) with a prevalence of AT in active individuals of 9-40% depending on the type and level of their sporting activity (Kujala et al., 2005). AT is a chronic musculoskeletal condition of multifactorial origin (Magnan et al., 2014); although the contribution to it by physiological factors that impair tissue patency and healing capacity ought not to be underestimated, given the relationship of the condition to movement, overuse is considered the primary pathogenic trigger. 'Overuse' is a clinically nebulous term, but involves repetitive submaximal loading of a particular musculoskeletal tissue, resulting in fatigue-mediated tissue breakdown (Shepherd and Screen, 2013). Surprisingly then, the role that fatigue plays in the pathogenesis of AT has remained largely unexplored. Furthermore, compared with other conditions, AT is not one that is managed consistently or well. Conservative management is considered the first and optimal strategy (Sussmilch-Leitch et al., 2012), although given the numerous reported conservative interventions, and the likely need for integrated multimodal care (Rowe et al., 2012), conservative management is also in this instance, a

nebulous clinical term. Whilst most conservative interventions such as injections (high-volume saline, prolotherapy, and platelet-rich plasma), shock-wave therapy and topical nitroglycerin are generally not well-supported (Kearney et al., 2015, Mani-Babu et al., 2015, Magnussen et al., 2009), eccentric loading has withstood scientific scrutiny better than others, and is recommended as the primary intervention for this condition (Couppe et al., 2015). Given the poor understanding of the condition and modest recorded treatment outcomes, it is not out of the question that either something is being missed, or more likely, we need to reconceptualise our understanding of the condition. AT is intimately linked to movement, so there is justification to examine the lower limb neuromusculoskeletal system in greater depth. Specifically, it is a condition that is connected to running and jumping, which are characterised by bouncing gait. For some time, bouncing gait has been well described mechanically using the Spring Mass Model (SMM) (Blickhan, 1989) and physiologically by the stretch shortening cycle (SSC) (Komi, 1984). Therefore, there is strong logic in pursuing a model that connects AT with the spring function of the musculoskeletal system, both in terms of its pathogenesis and its management.

The SMM simply and accurately describes the complex nature of bouncing gait by equating locomotion to a point mass (representing body weight), bouncing on a massless linear spring (representing the lower limb) (Nikooyan and Zadpoor, 2011). Furthermore, the SMM is associated with the SCC, a physiological phenomenon where the anti-gravity muscles associated with gait (e.g. ankle plantarflexors) are pre-activated, actively-stretched during the first phase of ground contact, before finally shortening in the push-off phase (Nicol et al., 2006). This phenomenon exists to simplify motor control strategies (Taube et al., 2012) and optimise locomotor metabolic efficiency (Bobbert and Casius, 2011), thus generating the highly effective bouncing gait described by the SMM. Collectively, 'spring function' is a novel term, referring to the behaviour of the lower limb as a spring, as driven by its neuromuscular apparatus during the SSC. Spring function is known to be adaptable based upon real-time requirements (Farley and Morgenroth, 1999), and is known also to have the capacity to systematically change, based on loading history (Nicol et al., 2006) or pathology (Maquirriain, 2012). One such pathology is AT, characterised by pain, disability, and biomechanical features indicative of impaired spring function (Roche and Calder, 2013, Maquirriain, 2012). In this condition though, our understanding of these interactions are far from complete. In summary, a model related to spring function to explain the pathogenic triggers of AT does not exist. Furthermore,

an equivalent robust model for the management of AT also does not exist. There is biomechanical and physiological plausibility that 'Spring Theory', the model proposed in this PhD explains the connection between AT and the spring function of human locomotion, helping to explain the pathogenesis of AT, and subsequently direct management strategies that may ultimately benefit the tendon and the active individual.

This thesis begins with a narrative review presenting the argument that spring function is intimately related to AT. Following from that, the first study explores a novel method of investigating spring function utilising an unloaded sledge-jump system (SJS). Having made these introductions, three main studies are presented, where the aforementioned methods are utilised to; 1) examine changes in spring function that exist in individuals with AT; 2) investigate how experimentally-induced fatigue alters spring function, and 3) investigate how an eccentric loading intervention alters spring function. In the discussion, these findings and implications will be considered collectively with a view to supporting the proposed 'Spring Theory'. It is hoped this thesis will provide a novel and significant contribution to the understanding of Achilles tendinopathy, the role fatigue plays in its development, and the mechanisms underpinning how therapeutic exercise confers its benefits.

A few caveats;

in this thesis, AT is used as a clinical model of an overuse injury. Given the role of the Achilles tendon in locomotion it is a most suitable condition to study. However, it is prospectively acknowledged that this model may apply to any/most overuse lower limb injuries associated with spring functions such as running and jumping (e.g. patellar tendinopathy, medial tibial stress syndrome, etc.). In addition, the pathogenesis of AT is multifactorial; whilst this thesis considers fatigue to be a primary pathogenic trigger (i.e. it's basically synonymous with overuse), it is certainly recognised that other non-mechanical risk factors play an important role in determining who is more susceptible to the effects of fatigue damage. Finally, eccentric loading has been selected to illustrate in this model the influence that improving motor performance has on spring function. It is recognised that in terms of clinical management, it is not the only suitable rehabilitation intervention, and nor is rehabilitation the only proposed intervention for AT.

1.2. THESIS OBJECTIVES

See Figure 1.1

I. To describe, and establish the reliability, of a sub-maximal hopping task on a custom-built sledge-jump system as a method to evaluate spring function
II. To explore, how AT affects spring function
III. To explore how fatigue affects spring function
IV. To explore how eccentric loading effects spring function
V. To describe 'Spring Theory' as an integrated model for the pathogenesis and management of lower limb overuse injury

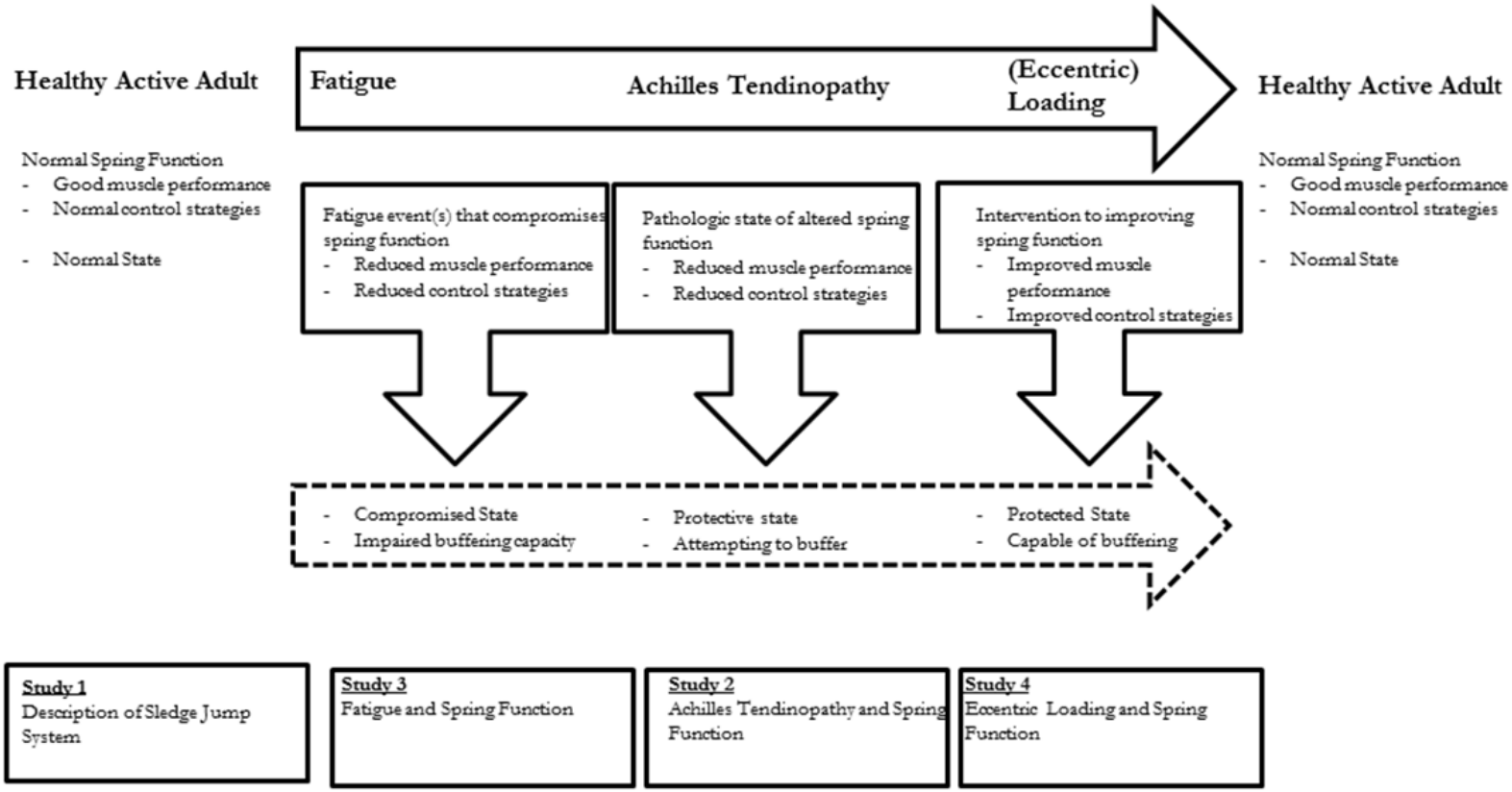

Figure 1.1 This thesis supports the theory that the pathogenesis and management of AT can be explained by modulation of lower limb spring function in accordance to fatigue, pathology and exercise. It is derived from four studies reported in four papers, including the development of a novel testing method (Study 1), the effect of AT on spring function (study 2), the effect of fatigue on spring function (Study 3), and the effect of eccentric loading on spring function (study 4).

1.3. THESIS STRUCTURE AND OUTLINE

This thesis has been structured as follows; following a narrative review of the pertinent literature, each subsequent chapter represents each individual study. It has been structured to be read chronologically and given that shared methods exists between chapters, they are described on their initial use, and to avoid repetition referred back to in subsequent chapters. The following is an outline of the four chapters that constitute the body of this thesis:

CHAPTER 3- THE SLEDGE JUMP SYSTEM

The use of a sledge jump system (SJS) is not novel to biomechanics research (Aura and Komi, 1986, Horita et al., 1996, Kuitunen et al., 2007, Furlong and Harrison, 2013); however, the studies of this thesis employed a unique, custom-built device. Furthermore, whilst the employed measurement tools are also common, many of the specific measures chosen to explore spring function are novel. Finally, the measurement task of sub-maximal single-limb hopping on the sleigh was employed for specific reasons. With these issues in mind, Chapter 3 is effectively a methods chapter, describing these tools and tasks and was achieved by answering the following questions that sought to establish the reliability of these methods:

1. Are measures of spring function consistent within a 30 second period of sub-maximal hopping on the SJS?
2. Are measures of spring function consistent across 10 consecutive hopping trials on the SJS?
3. Are measures of spring function consistent between 2 testing occasions, separated by 1 week?

This chapter has been reported on in the following journal articles and thesis:

- Debenham JR, Campbell AC, Travers MJ, Gibson WI, Grisbrook TL, Bulsara M, Allison GT (under review). "Reliability of a Sledge-Jump System for Measuring Stretch-Shortening Cycle Behaviour"
- Grisbrook TL, Cui L, Travers MJ, Debenham JR, Allison GT (under review). "Estimates of leg stiffness during low-load plyometrics"

- Travers MJ (2014). "An investigation into perception of change in the foot-floor interface during repeated stretch-shortening cycles" PhD Thesis, Curtin University Western Australia.

CHAPTER 4- ACHILLES TENDINOPATHY AND SPRING FUNCTION

AT is a common overuse condition affecting active (ageing) adults. Its pathogenesis is not clearly understood, and its management is subsequently sub-optimal. AT has clear links to spring dysfunction, although much remains to be learnt and the causal nature of these relationships determined. Chapter 4 presents a cross-sectional study where measures of spring function during sub-maximal hopping on the SJS are compared with a healthy population, with a view to answering the following question(s):

- During a sub-maximal hopping task on a SJS, are measures of spring function altered in individuals with AT?
    - Is vertical stiffness altered?
    - Are ankle kinematics altered?
    - Is agonist-antagonist muscle behaviour altered?

This chapter has been reported on in the following journal article and conference paper:

- Debenham JR, Travers MJ, Gibson WI, Campbell AC, Allison GT (2014). "Achilles tendinopathy alters stretch shortening cycle behaviour during a sub-maximal hopping task". *J Sci Med Sport* (In Press).
- Debenham JR, Travers MJ, Gibson WI, Campbell AC, Allison GT (2014). "Achilles tendinopathy alters stretch shortening cycle behaviour during a sub-maximal hopping task". International Scientific Tendinopathy Symposium (ISTS), Oxford, UK

CHAPTER 5- FATIGUE AND SPRING FUNCTION

'Overuse' is regularly cited clinically as a key pathogenic trigger for AT; however it has received surprisingly little empirical attention. Overuse is a consequence of fatigue, and fatigue has been demonstrated to affect various aspects of neuromuscular performance, including measures and correlates of spring function. Therefore exploration of the role that fatigue plays in the pathogenesis of AT can be explored using an experimental model of fatigue and its effect on spring function. To achieve this, the following questions were answered:

- During a sub-maximal hopping task on a SJS, does eccentric fatigue of the ankle plantarflexors alter measures of spring function?
    - Is vertical stiffness altered?
    - Are ankle kinematics altered?
    - Is agonist-antagonist muscle behaviour altered?

This chapter has been reported on in the following journal article and conference paper:

- Debenham JR, Travers MJ, Gibson WI, Campbell AC, Allison GT (2015). "Eccentric fatigue modulates stretch-shortening cycle effectiveness - A Possible Role in Lower Limb Overuse Injuries". *Int J Sports Med* (In Press).
- Debenham JR, Travers MJ, Gibson WI, Campbell AC, Allison GT (2014). "Eccentric fatigue alters stretch-shortening cycle behaviour during a sub-maximal hopping task". International Scientific Tendinopathy Symposium (ISTS), Oxford, UK

CHAPTER 6- ECCENTRIC LOADING AND SPRING FUNCTION

Eccentric loading has demonstrated efficacy in the management of AT. Despite this, the mechanisms underpinning its efficacy are unclear. One theory gaining traction is that eccentric loading improves the ability of the ankle plantarflexors, via various neural and structural means, to 'buffer' the tendon, thus removing relative load and potentially providing it with the opportunity to return to good health. This theory is consistent with 'Spring Theory' presented here but warrants further exploration. Chapter 5 does so using an experimental intervention study and answering the following question(s):

- Following an eccentric loading intervention, are measures of spring function altered in healthy participants during a sub-maximal hopping task on a SJS?
    - Is vertical stiffness altered?
    - Are ankle kinematics altered?
    - Is agonist-antagonist muscle behaviour altered?

This chapter has been reported on, and supported by the following journal articles:

- Debenham JR, Gibson WI, Travers MJ, Campbell AC, Allison GT (2015). "Eccentric loading of triceps surae modulates stretch-shortening cycle behaviour- a possible therapeutic mechanism". *J Sports Rehab* (In Press).

- Wellisch M, Hamer P, Hopper L, Debenham JR (2015). "Eccentric loading positively shifts peak torque angle of the ankle plantarflexors in healthy volunteers". International Journal of Sports and Exercise Medicine; 1:2 (see Appendix iii)

CHAPTER 7- DISCUSSION

The discussion chapter has two objectives. Its first objective is to collectively evaluate the meaning of the findings of the 4 studies, and explore common threads that provide insight into the manner by which spring function is modulated in response to different conditions. The second objective is to present 'Spring Theory' as a novel theoretical model that simplifies complex multi-dimensional conditions providing a potential framework for clinicians, educator and researchers to optimise management, treatment strategies and research directions.

2. LITERATURE REVIEW- PERSPECTIVES ON SPRING FUNCTION

2.1. INTRODUCTION

Locomotion is a fundamental human function, and from an evolutionary perspective arguably one of the most ubiquitous and important manifestations of life, equally important to feeding as to escaping predators (Hoppeler and Herzog, 2014). Although locomotion has been studied extensively to understand deficiencies in the musculoskeletal system, the understanding of the physiology of locomotion and its deficiencies is incomplete. Efficient locomotion involves a complex interplay between the musculoskeletal and neural control systems meaning that modelling gait/locomotion is incredibly difficult. However, given that the lower limb behaves like a compression spring during bouncing gait it has been biomechanically modelled using a spring mass model (SMM) (Blickhan, 1989), which is thought to be optimised in terms of efficiency by the physiological phenomenon of the stretch-shortening cycle (SSC) (Komi, 1984). With these two phenomena in mind, this thesis employs the term 'Spring Function' as a simple descriptor of lower limb function during running and hopping which recognises the collective considerations of the SMM and SSC.

Spring function can be regulated in accordance with real-time task and environmental requirements, and it can be modulated, with systematic changes in spring function occurring in response to loading history (fatigue and training) and pathologic states. Given the influence that loading and pathology have on spring function modulation, a better understanding of these relationships may help explain the pathogenesis of conditions affecting spring function such as overuse conditions of the lower limb. Likewise, addressing deficits in spring function in training and clinical practice may optimise treatment outcomes and preventions strategies.

This literature review consists of four main sections. Initially, the SMM and SSC will be introduced, with focus being given to the physiology underpinning the regulation and modulation of spring function. Secondly, Achilles tendinopathy (AT) will be discussed as a suitable clinical model with which to explore how an overuse musculoskeletal pathologic state can result from and be related to, impairments in spring function. Thirdly, the role of fatigue as a primary pathogenic mediator of AT is considered with specific interest being paid to the modulatory impact of fatigue on spring function. Finally, eccentric loading is a commonly prescribed exercise intervention that currently enjoys respect as the primary intervention for AT. However, the mechanisms underpinning its efficacy are unclear, and its capacity to alter spring function is presented to support the emerging

theory that eccentric loading exerts its positive influence via changes in muscle structure and neural function, subservient to spring function.

2.2. THE SPRING MASS MODEL AND THE STRETCH SHORTENING CYCLE

2.2.1. THE SPRING MASS MODEL (SMM)

In search of general principals underpinning bouncing gaits, biomechanists utilise the SMM, in view of the fact that during bouncing gaits such as running and hopping, legs behave like compression springs (Bobbert and Casius, 2011). During the first half of ground contact phase, leg length (the distance between the hip and toe) decreases whilst the ground reaction force (GRF) increases, and during the second half of the ground contact phase, leg length increases whilst GRF decreases. Originally described by Blickhan (1989), the SMM is defined by representing the entire mass of the animal as a single point mass and the entire musculoskeletal system as a single linear spring (Farley and González, 1996). The stiffness of the 'leg spring', as it is typically referred, is determined from the relationship between the magnitude of the GRF and the distance between the centre of mass and the centre of pressure on the ground (Bobbert and Casius, 2011).

Single-body and multi-body SMM's have been described and remain under development (models under development outside the scope of this thesis have been reviewed by Nikooyan and Zadpoor (2011)). The single body SMM is most commonly used to study lower extremity spring function during simple human-ground interactions such as running and hopping, where real-time regulation and systematic changes in response to interventions can easily be observed. A one-dimensional (1D) SMM is used for hopping and the 2 dimensional (2D) SMM is used for running yet conform to the same SMM principals (see Figure 1.1). Despite its simplicity, the SMM is remarkably accurate in predicting the characteristics of running and hopping (Nikooyan and Zadpoor, 2011, Farley and González, 1996). The single-body SMM was quantitatively evaluated by Bullimore and Burn (2007), who concluded that it predicts the parameters of human running (vertical peak GRF, stance time, contact time, etc.) 'reasonably well', with only a slight overestimation of certain parameters.

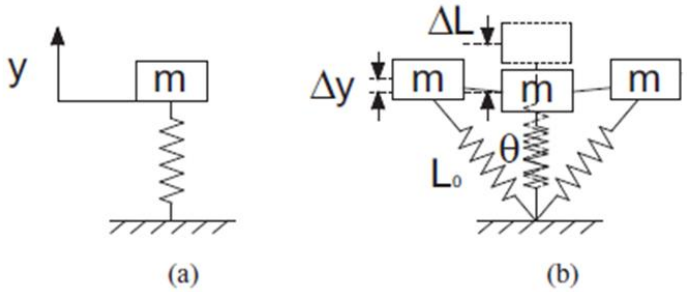

Figure 1.1 Schematic representation of the (a) 1D motion and (b) 2D motion passive one-body mass-spring models (Nikooyan and Zadpoor, 2011)

The primary advantage of single-body SMM's over multi-body SMM's is in their simplicity, based on the need for few input parameters; however, they are not without fault. The simplicity of the single-body SMM contrasts with the complexity of the actual musculoskeletal system, which consists of a complex jointed skeleton with muscles, tendons and ligaments acting across nearly every joint (Farley and González, 1996), so it cannot fully explain various aspects of the complex system that is human locomotion (Nikooyan and Zadpoor, 2011). For instance, the single-body SMM assumes ground reaction force (GRF) to act as a sine wave where in fact it actually commonly exhibits it has two peaks (Nikooyan and Zadpoor, 2011). Likewise, the human leg doesn't act as a linear temporal spring as the SMM assumes; it is driven by a musculoskeletal system that has multiple components involving complex active and passive processes interplaying to produce bouncing gait in what is in reality, non-linear. For example, fascicle-tendon interaction during ground contact is muscle-specific, task-specific and load history-specific (Lichtwark et al., 2013). Further still, the SMM assumes that the mechanical properties of the human body are constant regardless of task and environment, and of volitional and subconscious control. This is certainly not the case (see next section) as neuromuscular activity during the SSC varies considerably. Whilst an optimally accurate SMM should take muscle activity into account, active-body SMM's are excessively complex and yet to be fully validated (Nikooyan and Zadpoor, 2011), rendering them practically redundant for clinical applications. As such, it is widely accepted that despite its simplicity, the single-body passive SMM is appropriate to study many aspects of human locomotion (Lamontagne and Kennedy, 2013).

2.2.2. THE STRETCH SHORTENING CYCLE (SSC)

By making the musculoskeletal system behave globally like a spring, physiological advantages are enjoyed in terms of simplification of motor control and reductions in energy expenditure (Bobbert

and Casius, 2011). It is the SSC that is the physiological phenomenon associated with the SMM that facilitates this mechanical behaviour (Nicol et al., 2006).

The SSC is defined as "a natural muscle function in which the pre-activated muscle-tendon complex is lengthened in the eccentric phase followed immediately by muscle-tendon shortening in the concentric phase" (Taube et al., 2012). The SSC has three specific components; (1) pre-activation, (2) eccentric phase, and (3) concentric phase (Nicol et al., 2006). The pre-activation phase stiffens the extensor muscle groups to prepare the limb for ground contact while in the eccentric (braking) phase, lasting 50-120 ms (Nicol et al., 2006), the same muscles act like shock-absorbing structures as they absorb mechanical work while eccentrically lengthening (LaStayo et al., 2003). However, when muscles contract eccentrically as part of a SSC, they no longer act purely as shock-absorbers; rather they perform more like springs (Lindstedt et al., 2002). The concentric (functional or push-off) phase is the final phase, and the performance of this phase, which is the ultimate determinant of locomotor performance is in turn mediated predominantly by events that occur during the eccentric phase (Cormie et al., 2010). The SSC exists for the single well-recognised purpose of enhancing locomotor performance, which it does so by enhancing the concentric phase (Nicol et al., 2006).

The combination of pre-activation, followed by eccentric and concentric muscle activity generates a stronger concentric muscle contraction compared with one that occurred without the preceding contractions. The enhancement of concentric muscle activity during the SSC is known as residual force enhancement (RFE) (Herzog, 2014) and is a fundamental feature of spring function. It is created by multiple interacting physiological features including the storage/return of elastic energy, utilisation of the spinal stretch reflex (SSR), muscle activation and neural control.

Whilst no longer considered to be the only contributor to RFE during the SSC, the storage and return of elastic energy remains of primary importance (Kubo et al., 1999, Lichtwark et al., 2013). However, it has become apparent that the muscle tissue itself is also a significant contributor to this; during the SSC, muscle itself stores and recovers elastic strain energy, which has even been demonstrated to occur in the absence of tendon (Cavagna et al., 1985), responsible for over half of the elastic contribution to the SSC (Maganaris and Paul, 2002). With this knowledge comes an important concept, that of 'muscle buffering'. Muscle buffering is a clinical term, used to describe how muscle tissue acts as a 'shock-absorber' during movement, absorbing kinetic energy during movement, storing it as elastic energy before returning it or dissipating it as heat (Lindstedt et al., 2002). In performing this function, load is re-distributed away from other passive tissues such as joint, bone and tendon, which is less capable of withstanding, and responding to such loads. In

performing this buffering function, injury-risk is minimised and the importance of neuromuscular function is highlighted, and this concept is gaining traction in the understanding of tendinopathy (O'Neill et al., 2015).

Utilisation of the spinal stretch reflex (SSR) is critical to SSC performance (Komi and Gollhofer, 1997). During the pre-activation phase, muscle spindle sensitivity is increased (Cronin et al., 2011), leading to augmented reflex potentiation and increasing stiffness throughout the SSC (Kyröläinen and Komi, 1995, Cronin et al., 2011). SSR contributions appear to be most important during sub-maximal SSC activities. During sub-maximal SSC-activities (e.g. sub-maximal hopping rather than rebound jumping), there are reduced levels of pre-activation. An augmented SSR in this instance provides the muscle with an automated mechanism of stiffness generation that ensures adequate muscle activation occurs during the eccentric phase. If this did not occur and a judgemental error occurred where pre-activation levels were reduced, the tissue may yield as the muscle, with reduced activation, is unable to buffer the tissue during the eccentric phase. This may lead to elevated risk of tissue injury (Horita et al., 1996).

The role of muscle in contributing to elastic storage and recoil has already been discussed and its importance in this respect cannot be understated. However, muscles are primarily contractile tissues; hence they provide an important contribution to RFE through their contractile capabilities. Because it requires time to develop contractile force, the pre-activation and eccentric phases provide the muscle with the requisite time to develop the force required for task completion in the concentric phase. The level of contraction is related to SSC efficiency (Arampatzis et al., 1999). More recently, attention has turned to intramuscular physiology to help explain RFE (Seiberl et al., 2015). In brief, it has been suggested that Ca^{2+} -dependent increase in titin-stiffness, the development of half sarcomere non-uniformities, a stretch-induced increase in the number of attached cross bridges, or an increase in average cross bridge force may explain how muscle activity contributes to RFE during the SSC (Herzog, 2014) although further exploration in this realm is clearly required.

The SSC clearly operates under complex neural control based on feedforward and feedback mechanisms (Taube et al., 2012). Given the constantly changing environmental and task requirements of locomotion, these two mechanisms are highly adaptable. Furthermore, these processes are highly accurate, as evidenced by the highly efficient and effective nature of gait. Pre-programmed activation of supraspinal structures (and probably sub-cortical structures such as the cerebellum) contribute to the initiation and execution of the SSC (Taube et al., 2012) and whilst

not studied extensively, it would be unlikely if subcortical brain motor regions (cerebellum, basal ganglia, brainstem) were not involved in the production of co-ordinated SSC tasks.

2.2.3. REGULATION AND MODULATION OF SPRING FUNCTION

Locomotion takes place in an ever-changing environment, with constant changes in task requirements. Likewise, systematic changes over time can occur in spring function in response to a variety of single or multiple loading events. The next part of this review considers two key issues of spring function; (1) the real-time **regulation** of spring function changes in response to task and environmental requirements, and; (2) the systematic **modulation** of spring function in accordance with loading events such as fatigue, training and pathology.

2.2.3.1. REGULATING SPRING FUNCTION

SSC's involve high impact loads, with very short phases. Furthermore, as described above, the initiation of any SSC is determined by centrally-held feedforward motor patterns. However, spring function requires continual context-dependent regulation. For example, a runner will alter spring function with each step depending on changes in task (e.g. running faster) and environment (e.g. changing from bitumen to grass). In order to accommodate these constant changes, spring function is regulated by reflex and central neural pathways which operate simultaneously (Nicol et al., 2006).

Spring function is regulated for three reasons: (1) to optimise/stabilise locomotor efficiency by minimising metabolic and mechanical energy expenditure (Dalleau et al., 1998); (2) to simplify control strategies of locomotion (Bobbert and Casius, 2011)); and (3) to protect the musculoskeletal apparatus of the 'spring' and minimise the possibility of (overload) injury (Taube et al., 2012). Regulation is achieved through a complex interplay between control strategies, viscoelastic properties of the constituent musculoskeletal apparatus, and changes in geometry that occur in response to changes in environment and task (Bobbert and Casius, 2011).

Regulation of spring function in order to optimise locomotor efficiency is well-understood, but regulation for the purposes of minimising injury-risk is less so (Hobara et al., 2008). During locomotor tasks involving high loads or fatiguing loads, the primary extensor tendons (Achilles and patellar) work in ranges and under loads relatively close to their point of failure (Bohm et al., 2014, Fletcher and MacIntosh, 2015, Lai et al., 2014, Wren et al., 2001). For example, whilst running it has been reported that muscle-tendon forces going through the Achilles tendon are up

to 9000N, 12.5 times body weight and close to the ultimate strength of the tendon (Scott and Winter, 1990), and with strain levels of up to 13% (Rosager et al., 2002). Given these potentially damaging mechanical events that are 'normal', it is muscle activity, and their regulation by the CNS that confers protection on the tendon courtesy of this buffering phenomenon. For instance, research has indicated that tendon stiffness is regulated by muscle activity; increasing muscle activation increases tendon stiffness (Sugisaki et al., 2011). Without muscle activity the tendon is less stiff, subjecting it to loads, strains and heat shock that may be detrimental to tendon health (Slane and Thelen, 2014, Nell et al., 2012).

Vertical leg spring stiffness (discussed in depth later) is the primary measure of spring function and is considered to be the most important determinant of injury protection (Alentorn-Geli et al., 2009), with lower levels of stiffness associated with increased risk of soft-tissue injury (Hobara et al., 2008). Therefore in order to protect musculoskeletal tissues, spring function must be capable of being adjusted in real time via the regulation of muscular activation strategies with both time and phase-specificity.

Spring function is regulated according to task requirements and much work has been done in this area. For example, running velocity, leg stiffness and knee stiffness are correlated (Arampatzis et al., 1999). Likewise, during sub-maximal hopping or running, cadence is correlated with leg and ankle stiffness (Farley et al., 1991, Farley and González, 1996); a 65% increase in hopping/stride cadence is generated by a twofold increase in stiffness. Spring function is also regulated according to environmental requirements. For example, adaptations made in order to run successfully at a constant velocity over a surface of irregular height is achieved by regulating leg stiffness, which in turn is regulated at the ankle, and it is in muscle pre-activation where these alterations in stiffness as a response to the environment are produced (Muller et al., 2010). Likewise, during sub-maximal hopping tasks, it has been shown that humans adjust their leg stiffness inversely to changes in surface stiffness (i.e. leg stiffness increases as surface stiffness reduces) to maintain hopping performance (Ferris and Farley, 1997). Finally, spring function can be regulated by conscious choice; for example, based on specific instructions for an external source to either increase or decrease their ground contact time, participants in the study by Arampatzis et al. (2001) were able to achieve this and in doing so, alter their leg stiffness.

As established, SSC regulation is achieved by altering spring stiffness in accordance with environmental and task requirements to maintain an optimally efficient linear spring mass system (Bobbert and Casius, 2011). Stiffness is both generated and regulated by integration of feedforward motor commands (originating from the CNS), with feedback received from the periphery. In fact,

97% of stiffness regulation during the SSC can be explained by the magnitude of feedforward and SSR responses of the lower limb extensor muscles (Oliver and Smith, 2010).

Spring function regulation is predominantly generated with the integration of feedforward and feedback mechanisms (see Figure 1.2). It is thought the CNS holds an internal blueprint model of the dynamics of the musculoskeletal system in order to compute the necessary motor output for any given movement (which often includes SSC activity as an inherent component of the movement/task) across the multiplicity of environments encountered (Schwoebel et al., 2001). These motor commands are considered to exist independent of feedback systems and are required basically as a consequence of time delays of sensorimotor loops that limit the rapidity with which the motor system can respond to sensory events (Taube et al., 2012). The commands are regulated according to the expected environmental setting (e.g. surface stiffness) and task requirements (e.g. to jump as high as possible) determined by pre-existing schema. The primary mechanism of feedforward output during movement execution is muscle pre-activation, which is used to generate active stiffness (Kuitunen et al., 2002a). Musculotendinous units, composed of both contractile and non-contractile materials, are collectively 'tuned' by the nervous system to the properties of the spring system in order to optimise the storage and recovery of elastic strain energy during locomotion (see previous sections). Unfortunately though, the feedforward commands can never be 100% accurate. This is due to subtle variations in biomechanics, environment and task such that errors will inevitably occur. In light of this reality, during ground contact, peripheral feedback is generated and integrated into the movement schema to provide online reinforcement and/or adjustment (e.g. if the original schema miscalculated surface height/stiffness etc.) (Travers et al., 2013) and this reinforcement/readjustment is believed to be achieved via spinal and supraspinal pathways (Taube et al., 2012, Moseley, 2011). In other words, the predicted and actual consequences of the movement are compared, and adjustments are made to the internal model if disagreements present. The error between predicted and actual consequences of the individual SSC is used for task recalibration, which can be done for sequential SSC's whilst hopping or running (Marquez et al., 2014, Fiolkowski et al., 2005, Marquez et al., 2010, Lee et al., 2014, Blum et al., 2009).

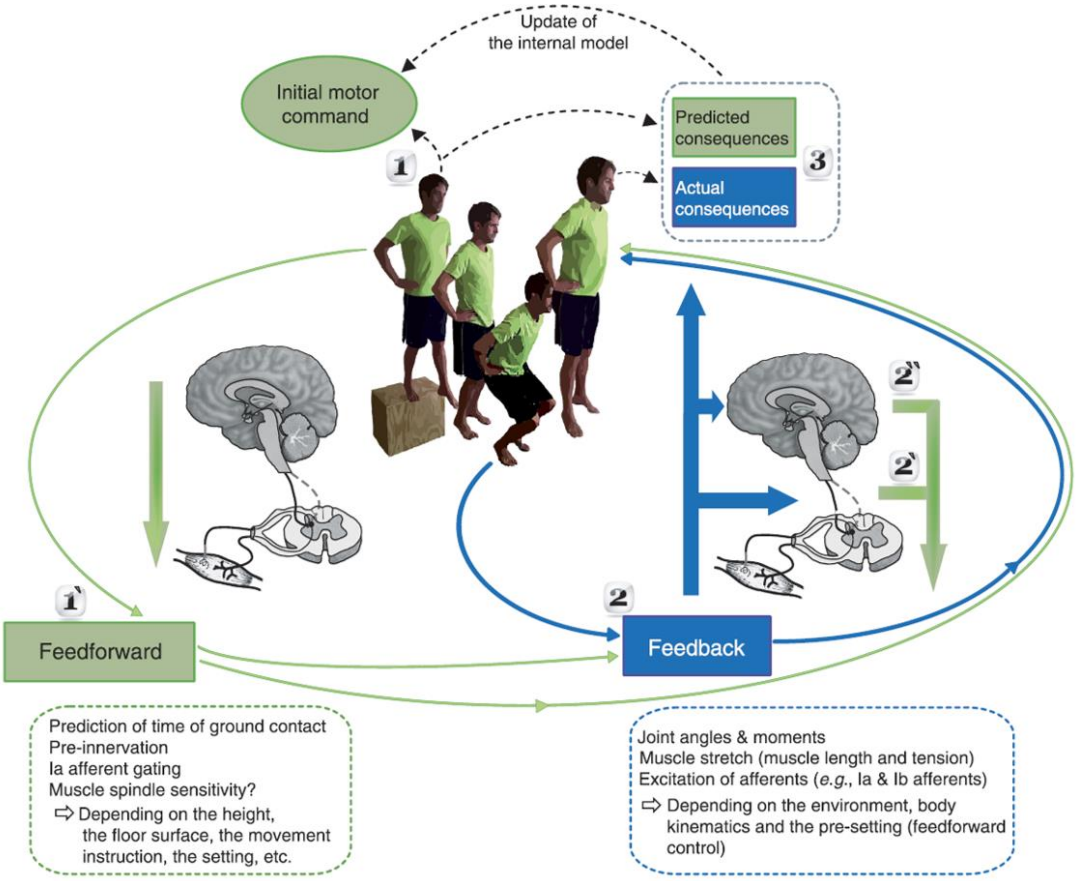

Figure 1.2 Schematic representation of the integration of feedforward and feedback processes to produce and regulate safe, efficient and effective spring function (from (Taube et al., 2012).

It is well know that the human CNS responds very quickly to stretches of a muscle-tendon unit (Taube et al., 2012) and SSR-activity is regulated in a context-specific manner (Voigt et al., 1998). Muscle spindles detect changes in muscle length and alter firing frequencies in Ia afferent fibres proportionate to the velocity and amplitude of the change in length of the muscle. The increased activity of Ia afferents after muscle stretch depolarises α-motorneurones at the spinal level, which elicit an agonistic monosynaptic stretch reflex–induced contraction. Of pertinence to the SSC, muscle pre-activation enhances SSR's (Cronin et al., 2011) and whilst outside the scope of this review, several experimental observations support the hypothesis of an integrated SSR within the SSC (see (Taube et al., 2012)). Furthermore it is the Ia afferent pathway (primary muscle spindle) that is the dominant pathway in generating the SSR during SSC tasks, more so than other structures like the Golgi tendon organ or cutaneous receptors (Taube et al., 2012). This reinforces the suggestion that appropriate muscle activation (intensity and temporally) during the pre-activation and eccentric phases is vital in terms of tuning the muscle (via SSR regulation) in order to provide accurate sensory information regarding task execution via Ia pathways.

As alluded to earlier, leg stiffness is regulated at different joints in a task dependent manner. During maximal activities (e.g. sprinting, drop jumps), stiffness of the lower limb is regulated at the knee (Kuitunen et al., 2002b), whilst during submaximal activities (e.g. running, sub-maximal hopping), stiffness is regulated at the ankle (Farley et al., 1998, Arampatzis et al., 2001). Leg stiffness is very sensitive to changes in in ankle stiffness compared to the hip and knee, because ankle stiffness is comparatively lower and in a system with multiple springs, the spring with least stiffness will undergo the largest displacement in response to a force and has the most influence on the overall stiffness (Maquirriain, 2012).

2.2.3.2. MODULATION OF SPRING FUNCTION

When the spring system is subjected to repeated task and environmental conditions that deviate from its natural homeostatic state, systematic alterations in spring function can occur. The three main modulatory factors of spring function are pathology, fatigue, and training (age and gender also modulate spring function but are not considered in this thesis due to their non-modifiable nature). It is unsurprising that these factors are interdependent, and it is this relationship that forms the keystone of this thesis, with each of these factors discussed in detail in subsequent sections of this review. In brief, overuse pathology of the lower limb musculoskeletal system (e.g. Achilles tendinopathy (AT) as a definitive example of overuse pathology) is characterised by changes in spring function (Maquirriain, 2012). Whilst such pathologies are multifactorial in origin, SSC-mediated fatigue is considered to play a significant role as a pathogenic trigger in AT, mediated in turn by its negative modulatory influence on spring function (Horita et al., 1996, Hayes and Caplan, 2014, Padua et al., 2006, Nicol et al., 2006) that ultimately overloads the musculoskeletal tissue beyond its patent capacity. The primary therapeutic intervention for such conditions is therapeutic exercise (e.g. eccentric loading), and emergent theory exists that the mechanisms underpinning the clinical efficacy of such interventions relate to the positive modulatory influence on spring function that this and other exercise-based interventions may have (O'Neill et al., 2015). However, much of this remains conjecture and requires significant investigation; this thesis seeks to contribute to this endeavour.

2.2.4. MEASURING SPRING FUNCTION

With an interest in modulation of spring function in mind, it is advantageous to measure spring function in its most natural manner. If the function of human muscle (the spring driver) is measured under pure isometric, concentric or eccentric actions, the principle of natural conditions will not apply (Nicol et al., 2006). Likewise, given that spring function can be influenced by multiple

factors, in order to fully reflect this, measurement should aim to combine neurophysiological measures and biomechanical measures so that interrelations of neuronal control and musculotendinous properties can be identified (Taube et al., 2012).

In the literature, numerous methods of investigation have been employed to quantify spring function. This thesis focuses on stiffness as the primary determinant of spring function, which in turn is influenced by measures of motion (kinematics) and muscle activity (surface electromyography (sEMG)). Whilst outside the scope of this review, where relevant, reference will be made to other measures that shed relevant light on the modulation of spring function.

STIFFNESS AS A MEASURE OF SPRING FUNCTION

The SMM models the lower limb as a linear spring of uniform stiffness. As such, biomechanical research interested in aspects of spring function, use stiffness as a primary measure (Nikooyan and Zadpoor, 2011, Brughelli and Cronin, 2008, Butler et al., 2003, Serpell et al., 2012). In its simplest terms, stiffness is defined as the resistance of an object or body to a change in length (McMahon and Cheng, 1990). Stiffness is related to Hooke's law, which states that the force required to deform an object is related to a proportional constant (spring) and the distance that the object is deformed (Brughelli and Cronin, 2008). A growing volume of evidence (discussed below) demonstrates that in accordance with pathology (e.g. AT), fatigue and training, spring function is modulated and can be observed via changes in measures of stiffness. Historically, several methods have been described to measure stiffness and whilst this thesis employs only one measure, for completeness the most common are now briefly discussed.

VERTICAL STIFFNESS

Vertical stiffness is commonly referred to as the 'reference' stiffness value; it serves as a measure of the resistance of the body to vertical displacement after the application of ground reaction force and is the primary measure of spring function in one-dimensional one-body SMM's. This review will focus on vertical stiffness as it is most commonly used (Brughelli and Cronin, 2008, Lamontagne and Kennedy, 2013, Serpell et al., 2012) due to its simplicity accuracy and utility. Vertical stiffness is defined as "the ratio of the vertical leg spring compression to peak vertical ground reaction force at the middle of ground contact" (Blickhan, 1989, Brughelli and Cronin, 2008). This measure is commonly calculated in one of four ways (McMahon and Cheng, 1990, McMahon et al., 1987, Dalleau et al., 2004, Cavagna, 1985), however three methods are considered to have the greatest validity and utility.

(McMahon and Cheng, 1990) recommends the calculation of stiffness based on the following:

$K_{vert} = \frac{Fmax}{\Delta Ymax}$ (McMahon and Cheng, 1990)

Where k_{vert} is measured in Nm^{-1}, F_{Max} represents the peak impact force and ΔY_{Max} represents vertical displacement of the centre of mass. Of note, this method has been criticised for over-estimation of the leg length change (distance change of CoM and the force vectors point of origin) (Arampatzis et al., 1999). Alternatively, and as a development on the method described by McMahon and Cheng (1990), Cavagna (1985) derived the following method:

$K_{vert} = m \left(\frac{2\pi}{T}\right)^2$ (Cavagna, 1985)

This method uses the subjects mass *m* and the period of the vertical vibration *T* to calculate k_{vert}, based on the assumption that the vertical GRF is a sine wave.

Finally, (Dalleau et al., 2004) validated a simple field test for k_{vert} where force data is not required:

$K_{vert} = \frac{m \times \pi(T_f + T_c)}{T_c^2 \left(\frac{T_f + T_c}{\pi} \frac{T_c}{4}\right)}$

In this equation, T_f and T_c represent flight time and contact time respectively. It has demonstrated a strong correlation ($r = 0.94$) with the reference method of (Cavagna, 1985), although it does slightly underestimate k_{vert} at hopping frequencies above 1.8 Hz, limited by the approximation *per se* of the force signal by a sine wave (Dalleau et al., 2004). This method is the one employed in this thesis.

Leg stiffness refers to the measurement of k_{vert} during two-dimensional activities such as running, where the same SMM rules apply although (see Figure 1.1), the leg spring is considered as an inverted pendulum, accounting for the forward displacement and angle of attack and forward velocity that occurs during running. Finally, joint stiffness, a measure of the torque required to induce angular displacement provides insight into how k_{vert} can be regulated by alterations in behaviour between constituent joint stiffness (Arya et al., 2006, Needle et al., 2014).

KINEMATIC AND MUSCLE ACTIVITY MEASURES OF SPRING FUNCTION

Whilst k_{vert} provides the best measure of spring function in its entirety, other measures of spring function exist that can highlight the musculoskeletal behaviour that drives spring function. The most commonly used of these are kinematic measures using motion capture systems (Morio et al., 2015, Kuitunen et al., 2002b) and measures of muscle activity using sEMG (Masood et al., 2014b).

OTHER MEASURES OF SPRING FUNCTION

Whilst outside the scope of this review, other musculoskeletal correlates are measured and reported in the literature and to briefly look at these other aspects of musculoskeletal behaviour can provide additional relevant insight regarding spring function. For instance, musculotendinous stiffness provides information on how the viscoelastic behaviour of muscle-tendon tissues influences locomotor capacity (Lichtwark et al., 2013, Suydam et al., 2015). Kinetics are measured to explore variations in force behaviour during SSC tasks (Wang, 2008), and from a more clinical perspective, isolated measures of muscle strength (Suydam et al., 2015, Miyaguchi and Demura, 2008, Lastayo et al., 1999, Alfredson et al., 1996), and measures of functional performance (Silbernagel et al., 2006) provide insight into force output and functional performance of the spring system respectively.

2.2.5. SUMMARY

In summary, human locomotion is based on a spring system that has been successfully explored utilising the SMM to measure aspects of its primary driver, the SSC. Given the ever-changing landscape in which humans perform various tasks, spring function requires an agile neuromuscular system that can regulate efficiently and effectively. Furthermore, spring function will systematically change if it is repeatedly loaded outside of inherent homeostatic boundaries (with due consideration acknowledged in terms of tissue condition). This change/adaptation can be positive following training, or negative if excessive loading results in uncompensated tissue fatigue and possibly pathology as a final result. The relationships that exist between spring function and these modulatory factors are complex and far from fully understood. This review now considers each of these factors in turn.

2.3. OVERUSE PATHOLOGIES: ACHILLES TENDINOPATHY – A DEFINITIVE PATHOLOGIC MODEL TO EXPLORE MODULATION OF SPRING FUNCTION

The Achilles tendon is the primary series elastic component of the ankle antigravity (i.e. spring) muscle group and it is through this muscle group (as the agonist) that the ankle joint regulates k_{vert} (Farley and Morgenroth, 1999). It is apparent that excessive load through the plantarflexor-Achilles tendon unit may result in injury, and Achilles tendinopathy (AT) is a common condition experienced by active individuals engaged in SSC activities (Kujala et al., 2005). As such, AT is the

ideal clinical experimental model for exploring the relationship between spring dysfunction and overload injury. Whilst principals discussed in this review relate specifically to AT, many extrapolations might be made to other lower limb overuse conditions (Butler et al., 2003).

Physical activity is a recommended and common pursuit in modern society, although excessive levels of activity often results in the development of overuse injuries (Mahieu et al., 2006, Hespanhol Junior et al., 2015). Overuse injuries occur as a result of a series of fatiguing events (Neviaser et al., 2012), where the tissue breakdown (caused naturally by the activity) and tissue production (in response to the activity) relationship is in net favour of tissue breakdown (Magnusson et al., 2010). Due to the fatigue-generated impairment placed upon muscle, load is redistributed through passive tissues (tendon and bone) and this ultimately results in tissue breakdown, clinical presentation and potentially, complete tissue failure (e.g. stress fracture (Clansey et al., 2012, Jacobs et al., 2014, Pamukoff and Blackburn, 2015)). It remains unclear what additional risk factors are required to cause one individual to sustain a bony injury (e.g. stress reaction injury) versus a soft tissue injury (e.g. AT), although differences in spring function modulation have already been suggested (Butler et al., 2003).

2.3.1. CLINICAL ASPECTS OF ACHILLES TENDINOPATHY

Tendon is composed of fibroblastic tendon cells (tenocytes), which produce, and are surrounded by, an extensive extracellular matrix (ECM). The ECM is predominantly made up of tightly packed collagen fibres (Type I mainly) that are orientated along the primary loading direction. Proteoglycans, mainly decorin and other non-collagenous proteins are also present. Tendons have mechanotransductive (load sensing) properties, mediated by the tenocytes, that subsequent to loading, lead to adaptive (strengthening) or maladaptive (tissue breakdown and failed healing (i.e. tendinopathy)) responses.

Tendinopathy is an umbrella term that indicates a non-rupture injury in the tendon that is exacerbated by mechanical loading (Scott et al., 2015). Tendinopathy is defined as 'a chronic clinical syndrome characterised by a combination of tendon pain and swelling and impaired performance arising from overuse and corresponding with histological evidence of tendinosis (van Dijk et al., 2011). Importantly, it has also been described as an overuse injury, "one caused by repeated micro-trauma without a single, identifiable cause" (Rae and Orchard, 2007)

Although AT can affect sedentary individuals of all ages, 70% of people with AT are recreationally active middle-aged men (Kujala et al., 2005). Overuse injuries, including tendinopathies, represent 7% of all primary care visits (Skjong et al., 2012) and 30-50% of lower limb injuries are related to

overuse (Sobhani et al., 2013). In general practice the incidence of AT is 2.35 per 1000 (de Jonge et al., 2011) and the prevalence of AT in active individuals is 9-40% depending on the type and level of sporting activity (Kujala et al., 2005), with a lifetime prevalence of 50% in elite endurance runners (Lopes et al., 2012).

AT is characterised by transient localised tendon pain associated temporally with high-level spring loading (e.g. running and jumping). Typically, the tendon 'warms-up' becoming less painful over the course of an activity, probably reflecting mechanisms associated with pain perception rather than actual mechanical changes associated with the activity (Rio et al., 2014). Symptoms are commonly preceded by a change (increase) in load frequency or intensity (overuse) during SSC activities (Bagge et al., 2011) and on examination, there is clear tenderness to local palpation (Hutchison et al., 2013).

The risk factors for AT have been extensively, if not systematically explored. They will be briefly presented here, contextualised in a manner relevant to their relationships with spring function. Although highly plausible, impairments in spring function as a risk factor for AT have received relatively little attention. Loading history (relative volume, frequency and intensity) is considered the most important risk factor for AT (Visnes and Bahr, 2011, Lorimer and Hume, 2014, Magnan et al., 2014, Gajhede-Knudsen et al., 2013), which is a behavioural factor, measured relative to an individual's musculoskeletal capacity (i.e. optimal spring function capacity) and the amount of fatigue they subject their system to (Gajhede-Knudsen et al., 2013). Looking at simple proxy measures of spring function, plantarflexor strength below 50 NM is predictive of AT development (Mahieu et al., 2006); furthermore, surface stiffness and a supinated foot have been found to be protective against AT (Lorimer and Hume, 2014). Both of these protective features make biomechanical sense in terms of spring function as they result in a more predictable environment in which to tune spring function at the foot-ground interface. A series of largely non-modifiable intrinsic risk factors for AT have been widely explored, all of which can be considered to negatively influence the structural patency of the muscle-tendon unit and/or its ability to recover from repeated SSC loads. These factors include age (de Jonge et al., 2011), genetics (September et al., 2009), male gender (Lorimer and Hume, 2014), co-existing systemic/metabolic disease markers such as diabetes mellitus (Mahieu et al., 2006), the use of certain drugs (Magnan et al., 2014) and adiposity (Gaida et al., 2009). Unfortunately, these measures are highly variable and as a result of the multifactorial nature of AT individual risk factor analysis is limited.

The pathogenesis of AT has long been under debate due to its multifactorial/heterogeneous nature (Magnan et al., 2014). To consider pathogenesis outside of mechanical issues is beyond the scope

of this review, which will concentrate on the issue of 'overuse' as the key trigger (Magnan et al., 2014, Gross, 1992), which is realised through reduced tissue resilience and a susceptibility to fatigue (Shepherd and Screen, 2013).

Tendon responds positively or negatively to the type/nature of load (Docking et al., 2015) that is placed upon it and the response is individualised to the tendons tissue capacity and its recent acute and sub-acute loading history (Lichtwark et al., 2013). The tendon responds positively (adapts) if the load is appropriate (Kubo et al., 2006, Docking et al., 2015) but excessive load results in negative tendon adaptations, leading to alterations in the structure and mechanical properties of the tendon (Wang et al., 2012, Arya and Kulig, 2010, Cortes et al., 2015); a response mediated by tenocytes (Docking et al., 2015). Repetitive suprathreshold tissue loading, results in initial tissue damage that sets up a healing process, driven by tenocyte activation and proliferation. This predominantly occurs in the tendon mid-portion due to the increased levels of transverse and rotational strain that occur in that location (Obst et al., 2014). However, for reasons that are not fully understood (Fu et al., 2010, Kingma et al., 2007), there is a failure in the normal healing response resulting in typical pathohistological features. These features include collagen disorganisation reflective of degeneration (fibre disorientation, separation and necrosis), increased proteoglycan and water content, alterations in cell density (i.e. areas of hyper- and hypocellularity) and phenotype (increased osteoblasts, chondroblasts and adipocytes) and areas of neovascularisation (Fu et al., 2010). The pain of tendinopathy is a controversial topic, with evidence to suggest that adaptive and maladaptive peripheral and central process contribute (Rio et al., 2014). Peripheral sources of nociception may include changes in the tendon matrix; vascular, cellular, and biochemical structure and/or function; more recently though, alterations in body schema and internal load calculation may represent explanations underpinning central changes associated with tendinopathy (Rio et al., 2016).

The management of AT remains challenging for clinicians and numerous studies have explored the myriad interventions that have been proposed (Magnussen et al., 2009, Sussmilch-Leitch et al., 2012). In AT, medical treatment is generally unsatisfactory and the outcome of surgical procedures is unpredictable (Frizziero et al., 2014, Khan et al., 2015). Conservative management involves many therapeutic options, ranging from non-steroidal anti-inflammatory drugs and eccentric loading, to corticosteroids, sclerosing, platelet-rich plasma, prolotherapy, and high-volume injections, extracorporeal shockwave therapy, splinting-bracing, active rest, low level laser therapy, concentric exercise, orthoses, therapeutic ultrasound, deep transverse frictions massage and topical glycerine (Rowe et al., 2012, Frizziero et al., 2014, Couppe et al., 2015, Sinnott et al., 2015). This plethora of

suggested treatment options may be reflective of the lack of effect of any one option over the other. Likewise, the lack of clear efficacy of any single intervention possibly reflects the fact that studies of single interventions for a condition of multifactorial origin are unlikely to demonstrate more than modest effect sizes. Despite this, there is current clinical consensus that the intervention with the highest level of evidence is exercise therapy, and it is recommended that all patients be initially treated with exercise for at least 3 months prior to consideration of other treatment options (Silbernagel and Crossley, 2015). Furthermore, and of note, recent studies combining injections with rehabilitation have shown elevated efficacy for this multimodal approach (Wetke et al., 2014).

Most patients with AT are treated conservatively, including activity modification and strengthening of the lower limb muscles (Sussmilch-Leitch et al., 2012) and it is becoming increasingly apparent that whilst loading-based rehabilitation is the cornerstone of conservative management, 'one size does not fit all' in this regard (Silbernagel and Crossley, 2015). Exercise that loads the muscle-tendon unit are promoted as being beneficial, with isolated eccentric exercise having received most attention (Malliaras et al., 2013, Kingma et al., 2007, Habets and van Cingel, 2014).

Eccentric exercise for tendinopathy was first described over 30 years ago (Stanish et al., 1986) after gaining traction based on anecdotal evidence reported by a handful of surgeons and sports physicians. The seminal paper by Alfredson et al. (1998), thrust eccentric loading into the clinical limelight, where it has since become the most studied intervention for AT (Malliaras et al., 2013, Kingma et al., 2007, Habets and van Cingel, 2014). Despite its popularity, its underpinning mechanisms remain poorly understood. Although the majority of researchers and clinicians explain these results in terms of the effect eccentric loading has on tendon healing via its unique loading influence on the tendon (e.g. (Rees et al., 2008, Grigg et al., 2014)), recent studies highlighting equivalency between eccentric loading and traditional heavy slow resistance training (Beyer et al., 2015) suggest this may not provide an adequate explanation (O'Neill et al., 2015) and the theory that loading, regardless of its specific action, but more dependent on its dose parameters (load, sets, reps, etc.) (Stevens and Tan, 2014) mediate their benefit through improvements in neuromuscular performance of the plantarflexors, and in doing so restore the plantarflexors capacity to buffer the Achilles tendon from excessive load, providing it with an opportunity to return to relative homeostasis (see later section).

2.3.2. THE RELATIONSHIP BETWEEN ACHILLES TENDINOPATHY AND SSC BEHAVIOUR

Over the past few years, interest has increased on the biomechanical dysfunctional that occurs with AT and in doing so, a reasonable amount of literature now exists providing insight regarding the relationship between AT and spring dysfunction.

Only a handful of studies have examined the relationship between AT and spring stiffness and they consistently demonstrate significant alterations (Maquirriain and Kokalj, 2014, Maquirriain, 2012, Arya et al., 2006, Child et al., 2010). Individuals with AT have reduced k_{vert} (Maquirriain, 2012) and this reduction is typically 10% when compared with the 'control healthy' side and that levels of stiffness reduction are correlated with clinical status and levels of pain (Maquirriain and Kokalj, 2014). This stiffness reduction is proposed to be mediated through reduced ankle stiffness and reduced plantarflexor muscle-tendon stiffness (Child et al., 2010, Arya et al., 2006), leading to conclusions that AT affects overall leg stiffness probably by increasing ankle compliance. Of interest, it appears that in the presence of AT, there is redistribution of stiffness away from the painful, less stiff region towards the knee (Arya et al., 2006), which may represent an adaptive response, in an attempt to reduce loads going through the tendon whilst maintaining adequate spring function.

AT has consistently been associated with altered spring kinematics that are consistent with the concept of impaired spring function (Kulig et al., 2011, Donoghue et al., 2008b, Donoghue et al., 2008a, Ryan et al., 2009). For example, AT is associated with greater frontal plane foot-ankle displacement movement (pronation) during running (Ryan et al., 2009, McCrory et al., 1999) and is associated with greater frontal and transverse plane displacements during the ground contact phase of a jumping during dance (Kulig et al., 2011). These findings all indicate joint yielding in the direction of spring displacement. Collectively, these findings suggest the existence of impaired capacity of the muscle-tendon systems of joints responsible for k_{vert} to resist the normal forces associated with SSC tasks.

Consistent with kinematic findings are alterations in muscle activity (Masood et al., 2014a, Franettovich Smith et al., 2014, Silbernagel et al., 2007, Wyndow et al., 2013, Grigg et al., 2013, Masood et al., 2014b). Again these findings are most pronounced locally to the plantarflexors, but impact throughout the lower limb, affecting all segments responsible for normal spring function.

Soleus offset is hastened during running (Wyndow et al., 2013) and despite reduced force-producing capabilities, there is increased sEMG in the soleus AT during a maximal voluntary isometric contraction (MIVC) (Masood et al., 2014a, Masood et al., 2014b). Likewise, when participants with AT performed a MVIC, soleus was their dominant plantarflexor, compared with medial gastrocnemius in healthy controls (Masood et al., 2014a). It has been suggested that increased muscle activity may be a neural attempt to increase Ia activity and provide improved sensory feedback in the presence of potential peripheral errors and errors in central processing/integration (Chang and Kulig, 2015b). Finally, from a pathogenic perspective, imbalanced activation of triceps surae may result in non-homogenous Achilles tendon stress, resulting in focal regions of excessive tendon stress, which may in part be responsible for pathology development (Obst et al., 2014).

Early studies of biomechanical measures of spring function focussed on measures in isolation, and these continue to this day, although when viewed collectively, provide important knowledge regarding spring dysfunction in the presence of the condition. In the presence of AT, tendons are less stiff and exhibit higher strain rates (Arya and Kulig, 2010, Child et al., 2009, Child et al., 2010, Wang et al., 2012, Chang and Kulig, 2015b, Cortes et al., 2015). Likewise, in the presence of AT there is reduced eccentric and concentric torque (Alfredson et al., 1996, Ohberg et al., 2001) although eccentric torque reduction is greater than concentric (Haglund-Akerlind and Eriksson, 1993). With initial insight into the interaction between feedforward processes and spring function, it has been consistently demonstrated that in the presence of AT, plantarflexor sEMG is greater (Reid et al., 2012, Masood et al., 2014a, Yu, 2014). Tying much of this work together is the eloquent recent study by Chang and Kulig (2015b), who in measuring neuromechanical properties of the plantarflexors (affected side vs. unaffected side) in individuals with AT, observed increased electromechanical delay (29 ± 2 vs. 39 ± 4 ms), as a result of reduced tendon stiffness (165 ± 44 vs. 295 ± 60 Nmm^{-1}), which was in turn compensated for increased pre-plantarflexor preactivation, presumably in an attempt to offset the impaired afferent feedback delivered by muscle and tendon sensory afferents (see Figure 1.3).

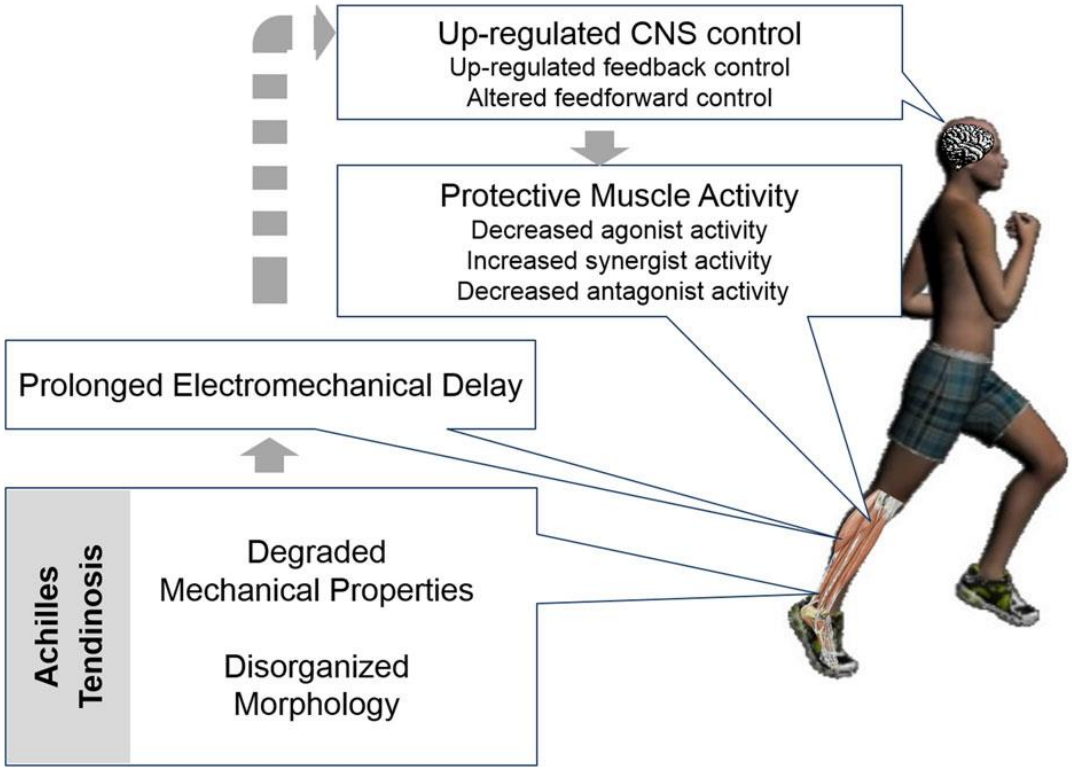

Figure 1.3 Interaction of feedforward and feedback processes derived from various hierarchies of the 'spring system', demonstrating the relationship between impaired sensory afferent input from the periphery and accommodating up-regulation of CNS control (Chang and Kulig, 2015a).

2.3.3. SUMMARY

In summary, whilst the problem of AT and its clinical presentation is quite clear, its pathogenesis and pathophysiology, whilst being understood to be multifactorial, is far from understood. As a result there is little consensus regarding optimal treatment strategies. Despite this it is known that in the presence of AT, alterations in spring function are consistently present, such as altered stiffness patterns, kinematics and muscle activity. The modulation of spring function in the presence of AT warrants further investigation so as to elucidate whether these changes are cause or consequence of AT, and if they are adaptive and/or maladaptive. Of particular interest is the role that fatigue may play in inducing this modulation in spring function.

2.4. FATIGUE

Fatigue is in many respects a somewhat nebulous concept, given the multitude of body systems involved in the development, recognition, and action of fatigue. Fatigue as a pathogenic trigger for AT via its modulatory influence is compelling, yet has received to date little attention. This review highlights the nature of neuromuscular fatigue and its modulatory influence on spring function. In

doing so, it is argued that fatigue can lead to adaptations that ultimately lead to the development of this pathologic state.

2.4.1. PHYSIOLOGY OF FATIGUE

Neuromuscular fatigue has been defined as "any exercise-induced reduction in the ability of skeletal muscle to produce force or power irrespective of task completion" (Gandevia, 2001). It is well-known that fatigue causes considerable changes in several neuromechanical parameters. These changes are commonly described in terms of **structural changes** and **neural changes** that whilst described separately, occur in parallel.

Structural fatigue refers specifically to changes that occur distal to the neuromuscular junction. It has also been referred to as peripheral fatigue, defined as a 'transient decrease in a muscles capacity for exercise' (Asmussen, 1979) and is related specifically to the muscles contractile components and mechanisms (Nicol et al., 2006).

Fatigue results in alterations in the structure and function of the nervous system. These neural changes, sometimes called central changes (Nicol et al., 2006) relate to the 'central governor theory', a theory where the CNS limits the recruitment of motor units to prevent extensive homeostasis disturbance, muscle damage, and biological harm (Noakes et al., 2004). The CNS holds a 'critical threshold' of peripheral fatigue that is tolerated by the CNS, as determined via activation of group III/IV afferents (Noakes et al., 2004). If this threshold is exceeded, the CNS limits the recruitment of motor units to prevent further peripheral fatigue and potential damage (Noakes et al., 2004). Fatigue modulates neural behaviour and activation pathways at different levels (Bigland-Ritchie, 1981). These include excitatory input to supraspinal motor centres, excitatory drive to α-motorneurones, modulation of interneuronal circuits, motorneurone excitability, peripheral reflex activity from small diameter afferents and Ia muscle spindle activity.

These neural changes do occur in a task-dependent and flexible manner and occur for what are believed to be two reason: to compensates for contractile failure and/or to induce contractile failure in a fatigued muscle (Nicol et al., 2006). Ultimately, these neural changes are likely an attempt to protect the musculotendinous system from (further) damage (Regueme et al., 2005b). Neural fatigue is mediated by multiple biochemical factors related to metabolic fatigue and mechanical damage (Nicol et al., 2006). It results in increased levels of extracellular potassium phosphate and lactic acid which stimulate muscle metaboceptors. Likewise, muscle damage results in primary release of biochemical substances such as bradykinin and prostaglandin and secondary

release (due to nociceptor stimulation) of substance P and histamine, all of which exert numerous inhibitory and excitatory influences on neural control of movement (Enoka, 2012).

The eccentric phase of the SSC is considered to be the most critical phase in terms of RFE (Cormie et al., 2010). With this in mind, it has been demonstrated that eccentric fatigue is unique and has specific additional neuromuscular consequences especially in terms of neural adjustments (Gonzalez-Izal et al., 2014). For a matched force, sEMG activity increases following eccentric fatigue (Semmler et al., 2007); metabolic fatigue is unlikely to be responsible for this as the changes remain after 2 hours, when short-lasting metabolic fatigue will have resolved (Semmler, 2014). This increase in relative sEMG following eccentric fatigue may reflect a decrease in motor unit conduction velocity, an increase in motor unit activity, or reflect increased activity that is required to produce the same force in the presence of increased antagonist activity (Semmler, 2014, Semmler et al., 2007, Bigland-Ritchie, 1981). Finally, the increased sEMG may reflect recruitment of additional motor units to compensate for the fatigue-induced decline in the ability of the active muscle fibres to generate force (Semmler, 2014). This area certainly would benefit from further exploration but it is clear that eccentric fatigue results in unique neuromuscular alterations that could influence spring function negatively. The studies from this thesis will attempt to highlight further the changes that occur in response to eccentric fatigue.

2.4.2. THE RELATIONSHIP BETWEEN (ECCENTRIC) FATIGUE AND SPRING FUNCTION

Comparatively is known about how eccentric fatigue might modulate spring function; however, several studies of fatigue have looked at pertinent measures of SSC behaviour from which it is possible to draw inferences.

In the pathogenesis of overuse conditions such as AT, fatigue of spring function may be a primary pathogenic trigger (Obst et al., 2013). Theory indicates that if a repeated SSC activity (e.g. running) occurs, fatigue will develop and several possibilities arise. (1) Do the subsequent adaptations in spring function provide a plausible explanation for increased tendon load to damaging levels?; (2) do the observed responses in spring function represent an attempt by the system to create a 'protective state', in attempt to protect the tendon from further damage? And if so; (3) is the system effective in doing so?

Four studies to date have associated fatigue with either altered leg or k_{vert} (Oliver et al., 2014, Kuitunen et al., 2007, Dutto and Smith, 2002, Fischer et al., 2015). Whilst the response is somewhat

dependent on the fatiguing task, fatigue generally results in decreased stiffness in the short term followed by a subsequent increase in the longer term. Sub-maximal SSC tasks such as jumping (Kuitunen et al., 2007) and running (Dutto and Smith, 2002), are commonly used to induce SSC fatigue after which spring function is observed. In these studies, stiffness decreases and is associated with decreased peak force, and an increase in ground contact time. This alteration in stiffness is driven by fatigue-induced changes in muscle activation during all phases of the SSC (Avela and Komi, 1998, Kuitunen et al., 2007), caused by insufficient energy production and associated accumulation of muscle metabolites. In addition to reductions in k_{vert} following fatigue, there is also a redistribution of stiffness contributions from different joints and this is believed to be a control strategy to 'offload' work from an increasingly fatigued region to a relatively less fatigued region (Bonnard et al., 1994). Once metabolic stress has resolved, it appears that fatigue results in increased k_{vert}, driven by reductions in joint excursions and increased extensor muscle activity (Morio et al., 2015). It is interesting that this seems to reflect strategies observed in the pathologic state, most likely representing an attempt by the system to maintain performance and protect tissues by offloading the threatened area. Alternatively, the increased muscle activity may represent a neuromuscular strategy of the same extensor muscles to facilitate Ia muscle spindles to gather accurate environmental data under increasingly challenging/compromising / threatening physiological conditions where feedforward and feedback strategies are likely to be compromised by fatigue (Chang and Kulig, 2015b). These theories warrant further investigation.

Despite these typical findings, additional evidence is emerging that stiffness responses to fatigue are variable at the individual level, with some individuals actually increasing stiffness as they fatigue (Oliver et al., 2014) suggesting the possibility of a potentiated state within the neuromuscular system (Hobara et al., 2007). These changes are associated with performance, confirming that fatigue-resilience mediates stiffness alterations and given the capacity to do so, individuals attempt to increase stiffness for protective purposes and do so until fatigue exceeds the individuals critical threshold.

Ample work has demonstrated that fatigue is associated with altered spring kinematics (Morin et al., 2010, Joseph et al., 2014, Padua et al., 2006, Kuitunen et al., 2002a, Willson et al., 2015) and muscle activity (Morio et al., 2015, Oliver et al., 2014, Horita et al., 1996, Regueme et al., 2005a) that may accompany these stiffness changes. Fatigue results in increased soleus activation during the pre-activation and eccentric phases of the SSC (Regueme et al., 2005a) as well as an in increase in activation of all lower limb extensors during sub-maximal running (Morio et al., 2015). This shift of muscle activation is considered to reflect a neural strategy to protect recovering

musculotendinous tissues from the (threatening) stretching phase, while securing the rebound performance by increased work during the push-off phase (Nicol et al., 2006).

2.4.3. SUMMARY

Fatigue is a predictable consequence of work in excess of an individual's capacity. Fatigue, mediated principally by neural adaptations occurs to maintain a performance profile whilst protecting tissues from potential/further damage. Fatigue clearly has the capacity to modulate spring function although much remains to be determined about under what circumstance, in what way and for what purposes these changes occur. The modulatory nature of fatigue emphasises the flexibility of the neural adjustments within and across homonymous muscles to meet the functional requirements of the peripheral system (Nicol et al., 2006). It appears that the modulatory influence of fatigue on spring function as a primary pathogenic trigger for overuse conditions such as AT has yet to be formally presented but has plausibility, warranting further consideration. Finally, it is noted that many changes associated with the fatigued state reflect those of the pathologic state, potentially suggesting commonality in cause and effect, but also that the individual simply perceives both states as threatening and employs a protective response within the confines of the (dys)functional apparatus it has.

2.5. ECCENTRIC LOADING

Management of disease, regardless of its nature, requires interventions directed at eliminating the primary driver(s) that have developed, or are maintaining those conditions. This review so far has argued that similarities exist between a potential pathologic trigger (fatigue) and pathology (AT). Following this logic, it's reasonable to explore the possibility that eccentric loading, the primary management intervention for AT, may exert its beneficial effect by positively influencing the neuromuscular deficits in spring function that are observed in AT.

PHYSIOLOGY OF ECCENTRIC LOADING

ECCENTRIC CONTRACTIONS

The true definition of an eccentric contraction indicates that the muscle must be active during stretch (Nicol et al., 2006). These eccentric, or lengthening contractions are a part of normal function performed regularly in our everyday lives and occur whenever we run downhill, walk downstairs or perform any slow braking movements (Semmler, 2014). Our understanding of the physiology of eccentric contractions remains somewhat limited; for instance it is established that

sliding filament theory and cross-bridge theories fail to explain the phenomenon of eccentric contractions but without an obvious alternative explanation (Hoppeler and Herzog, 2014). Eccentric contractions require different activation strategies and programming processes by the CNS with cortical activation higher in amplitude and area dimension (Vogt and Hoppeler, 2014, Guilhem et al., 2010) and they are capable of producing high forces at low neural and metabolic costs (Bigland-Ritchie, 1981). Fewer motor units are required to produce the same tension as in concentric contractions, leading to increased mechanical stress per motor unit (Vogt and Hoppeler, 2014). Eccentric contractions in isolation have two purposes; to dissipate energy for deceleration and to convert kinetic and potential energy into elastic energy within the muscle-tendon unit. Within the SSC they have the additional role of developing force in preparation for a concentric contraction.

EXERCISE-INDUCED MUSCLE DAMAGE

During eccentric exercise, skeletal muscle is subjected to both stretch and overload. If repeated and unaccustomed, the high levels of mechanical tension produces muscle micro-lesions, resulting in substantial damage to contractile, structural and subcellular skeletal muscle components (Hedayatpour and Falla, 2015). In addition to this, the excitation-coupling process is impaired (Allen, 2001), and collectively these result in a phenomenon known as exercise-induced muscle damage (EIMD). Following EIMD, four predictable phases of recovery occur; 'initial', 'autogenetic', 'phagocytic' and 'regenerative'. It is known that this damage results in short-term and long-term consequences to both structural and neural control mechanisms (Nicol et al., 2006) as well as the temporary perception of soreness, known as delayed-onset muscle soreness (DOMS). The whole process is considered as a protective muscle response/adaptation, providing a stimulus for beneficial neuromusculotendinous changes to occur (LaStayo et al., 2003). A number of investigators have reported that there is a shift in the optimal peak torque joint angle such that peak force occurs at longer muscle lengths following eccentric contractions (Wellisch et al., 2015, Brockett et al., 2001, McHugh and Tetro, 2003). This phenomenon is most often attributed to an increase in series elastic compliance in the damaged muscle that is thought to occur as a result of sarcomere disruption, but may also result from fatigue (Proske and Morgan, 2001).

ECCENTRIC LOADING

Muscle is a highly mutable tissue in that its structure and function adapt to the demands placed upon it. Eccentric loading (aka eccentric exercise) is the therapeutic application of selectively dosed repeated eccentric muscle contraction, performed as a specific intervention for a pathologic condition, or for performance training purposes. Eccentric loading results in adaptive

physiological, structural and neural changes that help explain clinical efficacy (Hedayatpour and Falla, 2015).

PHYSIOLOGICAL ADAPTATIONS

Mechanical tension, and possibly ischaemia, produced by the combination of force generation and stretch, is an essential factor to stimulate signalling pathways involved in increased muscle performance and the combination of force generation and stretch, unique to eccentric loading are thought to be additive (Vandenburgh, 1987). Histochemical adaptations to eccentric loading include increased free-radical population and increased activity of growth-oriented transcription factors as well as increased muscle fibre membrane permeability to calcium ions and myocyte activity. In response to eccentric loading the hormonal environment changes with enhanced levels of growth factors such as insulin-like growth factor and enhanced activity in tension-sensitive anabolic pathways mediated by growth hormones such as testosterone being observed (Hedayatpour and Falla, 2015, Vandenburgh, 1987).

STRUCTURAL ADAPTATIONS

Following eccentric loading, muscle fibres change their optimal length, becoming more compliant via the addition of sarcomeres in series, known as sarcomerogenesis (Welsh et al., 2015, Wellisch et al., 2015). Eccentric loading of the hamstrings has been shown to increase fascicle length by 34% (Potier et al., 2009). Sarcomerogenesis allows muscle fibres to operate at longer lengths while avoiding the descending limb of the length-tension curve (Brockett et al., 2001), enabling them to more comprehensively fulfil their role in buffering adjacent tendon tissue to repeated loads, especially in outer range, where tendon tissue is typically at greater risk, due to muscle diminished capacity. For example, consider a runner: during ground there will be a SSC of the plantarflexors. During the eccentric phase, which is known to provide over 50% of the muscle-tendon units capability for withstanding tensile load, muscle progressively contracts on the descending limb of the length-tension curve (Proske and Allen, 2005). The further down that curve (Lichtwark et al., 2013), the greater degree of load will be placed on the tendon. In contrast, following eccentric loading, the location of this descending limb shifts to the right and for the same joint angle the muscle is better capable of producing force, thus removing load that would otherwise be placed on the tendon and providing it with the opportunity for improved function. Finally, eccentric loading results in changes in fibre type composition; more fragile, stress-susceptible muscle fibres are reduced in number while stronger fibres survive, a process known as preferential apoptosis. Preferential apoptosis results in a shift towards a faster muscle phenotype (Vogt and Hoppeler, 2014), and this is believed to serve a protective effect (LaStayo et al., 2003). This is further

enhanced by titin isoforms altering to stronger varieties (Monroy et al., 2012) shifting to forms with greater stiffness.

NEURAL ADAPTATIONS

Neural adaptations to loading is defined as "…changes within the nervous system that allow an individual to more fully activate prime movers in specific movements and to better co-ordinate the activation of all relevant muscles, thereby affecting a greater net force" (Sale, 1988). These adaptations may occur at the level of the motor cortex, spinal cord, neuromuscular junction and/or sarcolemma (Hedayatpour and Falla, 2015) and explain the discordant relationship between strength gains and structural muscle changes in the early period of resistance training. Observed cortical changes include earlier onset of cortical activities, attributed to the planning and execution of the more complex contraction (Liu et al., 2000) and increased cortical activities, associated with processing feedback, which are larger during eccentric than concentric actions, likely due to the higher degree of movement complexity and/or stretch-related transcortical reflexes (Liu et al., 2000, Yue et al., 2000). Furthermore, eccentric loading results in reduced intracortical inhibition (37%) and increased corticospinal excitability (51%) when compared with concentric exercise (Kidgell et al., 2015). At the motor unit level, eccentric loading results in changes in motor unit recruitment patterns (Clarkson et al., 1992), shifting towards faster motor units, (Hodson-Tole and Wakeling, 2009) in addition to increased motor unit discharge rate and increased muscle fibre conduction velocity.

Eccentric loading also produces alterations in SSR regulation (Engardt et al., 1995). There is a modulatory increase in the amplitude of the maximal muscle H reflex (muscle reaction after electrical stimulation muscle spindle sensory afferents) (Vangsgaard et al., 2014), which these occurs within 5 weeks, and is associated with increased MVC. This explains, at least in part, the neural explanation of increased force production in the early phase of eccentric loading when compared with other loading forms (Vangsgaard et al., 2014). This change in regulation strategy is difficult to explain; it may reflect increased motorneurone excitability, or recruitment of additional 'fringe' motorneurones due to an altered balance between Ia (muscle spindle) excitatory and Ib (Golgi tendon organ) inhibitory input (Vangsgaard et al., 2014).

These functional changes result in altered neuromuscular function with the ultimate outcome of these changes being potential improvements in spring function. It has been clearly demonstrated that following eccentric loading, the muscle increases its stiffness, and this occurs independently of any increases in muscle size and strength (Lindstedt et al., 2002).

THERAPEUTIC APPLICATIONS OF ECCENTRIC LOADING

Eccentric loading is applied therapeutically in a number of clinical scenarios besides AT, including hamstring strains (Askling et al., 2013), ACL rehabilitation (Lepley et al., 2015), hip/knee replacement rehabilitation (LaStayo et al., 2003), sub-acromial impingement syndrome (Valier et al., 2014) and lateral epicondylalgia (Cullinane et al., 2014). Although eccentric loading has not been established as a superior management strategy for all tendinopathies (Couppe et al., 2015, Woodley et al., 2007), it has demonstrated efficacy for conditions associated with spring dysfunction such as AT. The most recent systematic review identified 29 studies that evaluated the efficacy of eccentric loading (Frizziero et al., 2014). Whilst the conclusions of this review were limited by the methodological quality of some of the studies, it confirmed the wide use of eccentric loading for AT, with generally successful outcomes (Frizziero et al., 2014). Most authors report successfully returning 60% of participants back to sport (Sussmilch-Leitch et al., 2012, Kingma et al., 2007). The most recent well-constructed trial of eccentric loading for Achilles tendinopathy was conducted by Beyer et al. (2015). Following a traditional 12-week eccentric loading intervention alone, clinical status (measured by the VISA-A questionnaire where 100 represents a perfect score and no disability) reported a mean improvement from 58 ± 3.9 to 72 ± 3.7 at 12 weeks and 84 ±3.5 at 52 weeks.

Despite the well-studied general adaptations associated with eccentric loading the mechanisms underpinning the effectiveness of eccentric loading in AT (and other clinical populations) are unclear. Recent reviews of the topic by O'Neill et al. (2015), Allison and Purdam (2009) and Couppe et al. (2015) provide a broad consensus that many of the suggested mechanisms popular in the clinical and research community are unlikely explanations for the efficacy for eccentric loading. These mechanisms include structural tendon adaptation (Drew et al., 2012), tendon length changes, alterations to neurovascular ingrowth (Zanetti et al., 2003), and alterations in fluid movement (Grigg et al., 2009). If these mechanisms are placed to one side, there is greater plausibility that eccentric loading confers it benefits through neuromuscular adaptations (i.e. changes in structure and function of the neuro-muscle-tendon unit) subservient to spring function. In addition to the mechanisms described above, based on studies on healthy volunteers, a plethora of work has provided plausible mechanisms underpinning eccentric loading for AT. Grigg et al. (2014) demonstrated that eccentric loading can optimise motor control of the eccentric phase, and smooth tendon loading; Masood et al. (2014b) observed that eccentric loading resulted in the normalisation of altered sEMG activity in triceps surae, redistributing activity away from the overactive soleus, to the underactive lateral gastrocnemius, resulting in comparably homogenous

muscle activity of the triceps surae. Furthermore, eccentric loading can increase fascicle length by 9% Crill et al. (2014), shifting the peak torque angle by a clinically meaningful 3.2° towards dorsiflexion (Wellisch et al., 2015). The collective mechanistic and clinical work on eccentric muscle contractions, provide a compelling argument that it is indeed neuromuscular adaptations that are responsible for the clinical benefit of eccentric loading programmes.

By improving the structural and functional properties of muscle capable of contributing to tendon buffering, and by improving muscular co-ordination, locomotion can occur in a safe and efficient manner, with load being appropriately placed through the muscular elements that appear to be optimally designed to fulfil this role.

THE RELATIONSHIP BETWEEN ECCENTRIC LOADING AND SPRING FUNCTION

Given that there seems to be merit in exploring the clinical benefit of eccentric loading by improving spring function, the next section of this review will consider some of the research conducted in this area in order to shed light on this possibility.

The effect of eccentric loading on k_{vert} has received limited attention with only two studies to date. Elmer et al. (2012) demonstrated that eccentric loading on a cycle ergometer increases k_{vert} by 10% during a hopping task. Lindstedt et al. (2002) observed that eccentric loading resulted in a 12% increase in natural hopping frequency rate, which would theoretically be associated with increased stiffness Dalleau et al. (2004). To date, no research has directly explored the kinematic or sEMG changes that occur in response to eccentric loading during a SSC-task. The only work has been mechanistic, related to the work discussed in previous section of this review. As such, a great opportunity exists to explore how eccentric loading alters spring function, particularly given the potential for eccentric loading to positively influence neuromuscular properties of the musculotendinous apparatus and neural control networks associated with spring function. This thesis aims to contribute in the field.

SUMMARY

In summary, the specific effects of eccentric loading on spring function have received limited attention. Despite this, there is a long held theory that eccentric loading, via the mechanisms reviewed above improves the economy and safety of spring function and does so quite rapidly, within 7 days of a single bout of eccentric loading (Timmins et al., 2015) suggesting that a combination of neural and structural factors contribute to these changes. Furthermore, Lindstedt

et al. (2001) demonstrated that eccentric training confers increased hopping economy to the equivalent tune of reducing body mass by 50%.

A theory is emerging that eccentric loading confers its clinical effect via positive adaptations in neuromuscular function that would likely result in positive modulation of spring function. This clearly warrants further exploration.

2.6. IMPLICATIONS OF THESE RELATIONSHIPS

The objective of this thesis literature review to this point was to present the evidence that a modulatory relationships exists between pathology, fatigue, and eccentric loading on spring function.

IS ECCENTRIC FATIGUE A PATHOGENIC TRIGGER FOR ACHILLES TENDINOPATHY?

Eccentric fatigue may well be a pathogenic feature of an overuse injury such as AT. It is known that fatigue is associated with increased injury risk (Oliver et al., 2014), and Clement et al. (1984) were the first to suggest that high eccentric loads during SSC activities may provide an 'overuse' stimulus for AT. It seems most likely that the loss of structural and neural functionality is responsible for this fatigue-induced pathogenic trigger leading to prolonged and repeated redistribution of load away from the muscle and towards the tendon (see Figure 2.3) resulting in progressive catabolic breakdown (Magnusson et al., 2010).

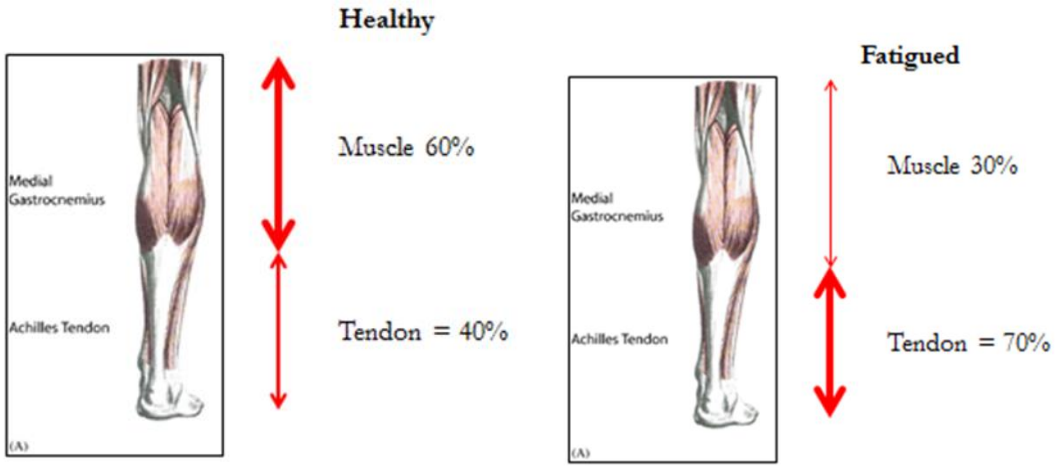

Figure 2.3. Schematic representation of load redistribution during SSC tasks following fatigue, indicating the increased relative loads passing through the muscle and tendon respectively.

From a structural perspective, it has been demonstrated that reduced plantarflexor strength (a surrogate measure of fatigue-resistance) is a risk factor for AT (Mahieu et al., 2006). As such neuromuscular changes in response to fatigue results in redistribution of load within the muscle tendon unit towards the tendon. Fatigue presumably alters spring function by reducing stiffness behaviour at the tissue, joint and lower limb levels resulting in net 'yielding' of the leg spring. Knowing that the entire muscle-tendon system participates in the storage-recovery and dissipation of this energy during the SSC, in a fatigued state, the muscles capacity to contribute to this process is compromised, and therefore without the muscle to act as a buffer, that same force is absorbed increasingly through the passive structures, principally the tendon (Dutto and Smith, 2002). From a neural perspective, it is known that fatigue may alter sensory feedback from muscle spindles resulting in imprecisions of motor control integration of feedforward and feedback strategies, with the realities of the task and environment. Likewise, fatigue may present a threat, and the neural changes may reflect an adaptive response as the system attempts to protect itself from the perceived threat (Chang and Kulig, 2015b).

DOES ECCENTRIC LOADING CONFER ITS CLINICAL BENEFIT VIA IMPROVED SPRING FUNCTION?

It is understood that altering neuromuscular function in response to a given stimulus (e.g. training) is an important component of reducing injury risk. Therapeutic exercise may provide such a stimulus to the Achilles tendon muscle-tendon unit, providing that component of the spring with enhanced buffering capacity. The eccentric phase is the most important phase of the SSC (Cormie et al., 2010) so it is logical that therapeutic exercise in the form of eccentric loading is likely to be one of the most effective means of improving spring function by these means. AT is associated with deficiencies in spring function (Wyndow et al., 2013, Maquirriain, 2012, Baur et al., 2011) and it has been posited here that eccentric loading may address these various deficits in neuromuscular function.

2.7. SUMMARY

In summary of this literature review, the role of the SMM and its physiological counterpart, the SSC as the biological phenomena of locomotion have been explored. Furthermore the physiology underpinning the regulation and modulation of spring function has been discussed with explicit reference to modulation in accordance with pathology, fatigue, and eccentric loading. It is clear that our understanding of the capacity of these modulatory factors to influence spring function is limited, but there is compelling evidence to justify its future exploration. Likewise, a model was

suggested where, fatigue may serve as a pathogenic trigger to Achilles tendinopathy, and eccentric loading as a therapeutic intervention, may confer its clinical benefit by address the deficits in spring function observed in the pathologic condition.

3. THE RELIABILITY OF A SLEDGE JUMP SYSTEM

INTRODUCTION

Understanding the mechanical behaviour of the lower limb during functional tasks is important to optimise athletic performance, minimise injury risk and improve rehabilitation strategies (Hobara et al., 2012). The spring mass model (SMM) (Blickhan, 1989), has enabled researchers to investigate lower limb behaviour in a simple and robust manner.

The SMM describes human locomotion based on a point mass supported by a single, 'massless' linear 'leg spring' (Butler et al., 2003) (see Figure 2). In this model, lower limb stiffness refers to the ratio between force and linear displacement of the lower limb and is highly representative of running mechanics (Blickhan, 1989). The utility of the SMM is that it accurately, but simply, describes the behaviour of the lower limb as an entire functional unit during ground contact. As such it is intimately linked to the stretch shortening cycle (SSC).

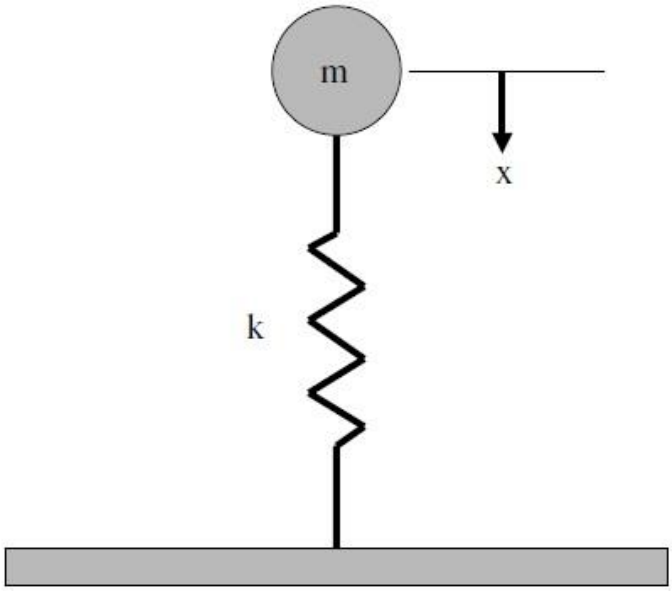

Figure 2: Spring mass model (from Butler et al. (2003) with permission), where m is mass, k is stiffness and x is displacement.

The SSC is the physiological phenomenon that provides the lower limb with its spring-like attributes. It is characterised by the pre-activation, lengthening and then shortening of a muscle-tendon unit during the ground contact phase (Nicol et al., 2006) and is a universal characteristic of mammalian locomotion (Shen and Seipel, 2015). The role of the SSC is to simplify the motor

control of locomotion while concurrently optimising locomotor efficiency (Farley et al., 1991, Bobbert and Casius, 2011). This is achieved by the utilisation of muscle pre-activation, storage/return of elastic energy, and the spinal stretch reflex (Cormie et al., 2010). The SSC is regulated in real-time according to changes in task (Schache et al., 2014, Arampatzis et al., 1999) and environment (Marquez et al., 2014), and can be modulated in response to training (Miyaguchi et al., 2014) and pathologic conditions (Debenham et al., 2016c, Ryan et al., 2006). In order to gain meaningful insight into the factors associated with these variations in SSC behaviour, reliable measurement methods are required. SSC behaviour can be measured in a number of ways including observations of muscle activity and joint kinematics either in isolation, or during SSC tasks (Wyndow et al., 2013, Williams et al., 2008). Another valid measure of SSC behaviour is lower limb stiffness, which refers to the force required to deform the lower limb during the stance phase of gait. The advantage of this measure is its functional relevance, incorporating all neuromusculoskeletal components as inferred by the SMM. Lower limb stiffness can be measured in a number of ways during a multitude of SSC tasks, with the most common being running (Farley et al., 1993) and hopping (Dalleau et al., 2004).

Sub-maximal hopping is an advantageous model for measuring the SSC; in comparison to running, it allows for more reliable measures due to the restriction of the centre of mass movement (McLachlan et al., 2006) and the elimination of horizontal translation. However, the sub-maximal upright hopping model may still demonstrate undesired variability in performance due to difficulty in controlling centre of mass trajectory, and the rapid onset of fatigue (Horita et al., 1996). A sledge jump system (SJS) offers an experimental model whereby individuals hop while the body is partially supported, restricting the degrees of freedom of the task and limiting the impact of fatigue for an equivalent upright hopping task. An additional benefit of a SJS is that in de-loading the participant, enables examination of more subtle variations in behaviour that might be 'drowned' by the higher forces associated with upright hopping. Therefore, it affords improved reliability whilst maintaining the dynamic environment required for SSC-tasks.

Given the utility of this method, many different SJS's have been employed in the past (Aura and Komi, 1986, Ertelt and Blickhan, 2009, Furlong and Harrison, 2013). However, there is limited information on the reliability of the derived measurements (Furlong and Harrison, 2013). As such, it is important to quantify the reliability of the SJS system and its associated standard error of measurement (SEM). The purpose of this study therefore was to measure the temporal reliability and SEM of measures of SSC behaviour during sub-maximal single-limb hopping on a SJS. We hypothesised that after a period of familiarisation, the measures of SSC behaviour would become

highly reliable. To test this hypothesis, we measured lower limb stiffness and joint kinematics during this task, consisting of 10 trials of 30 hops, over two identical testing occasions, 1 week apart. With established reliability of this tool, it can be applied to confidently identify true changes in SSC behaviour as a result of regulatory or modulating factors associated with activity, training and lower limb injury.

MATERIALS AND METHODS

This study employed a within-subjects, repeated measures design. The independent variable was time (30 hops, 10 trials, and 2 testing occasions), whilst dependent variables were lower limb stiffness and sagittal plane joint angles of the ankle, knee and hip (80 ms pre-contact, ground contact, take-off and stretch amplitude respectively).

PARTICIPANTS

Fifteen (8 females and 7 males; mean age 27.4 ± 5.6 years, height 171.2 ± 10.7 cm, mass 71.1 ± 15.9 kg) healthy volunteers participated in this study. Potential participants were excluded if they had a lower quadrant neuromusculoskeletal condition in the preceding 12 months, a significant visual or motor impairment, or were pregnant. All participants provided written informed consent, with procedures being approved by the local University human research ethics committee.

PROCEDURES

Participants attended two separate data collections, one week apart at the same time of day (Figure 1 & b). They were instructed to continue their normal everyday activities but to refrain from undertaking any unfamiliar physical activity in the week prior to, and between testing occasions. In addition, they were not to undertake vigorous physical activity in the 24 hours prior to testing.

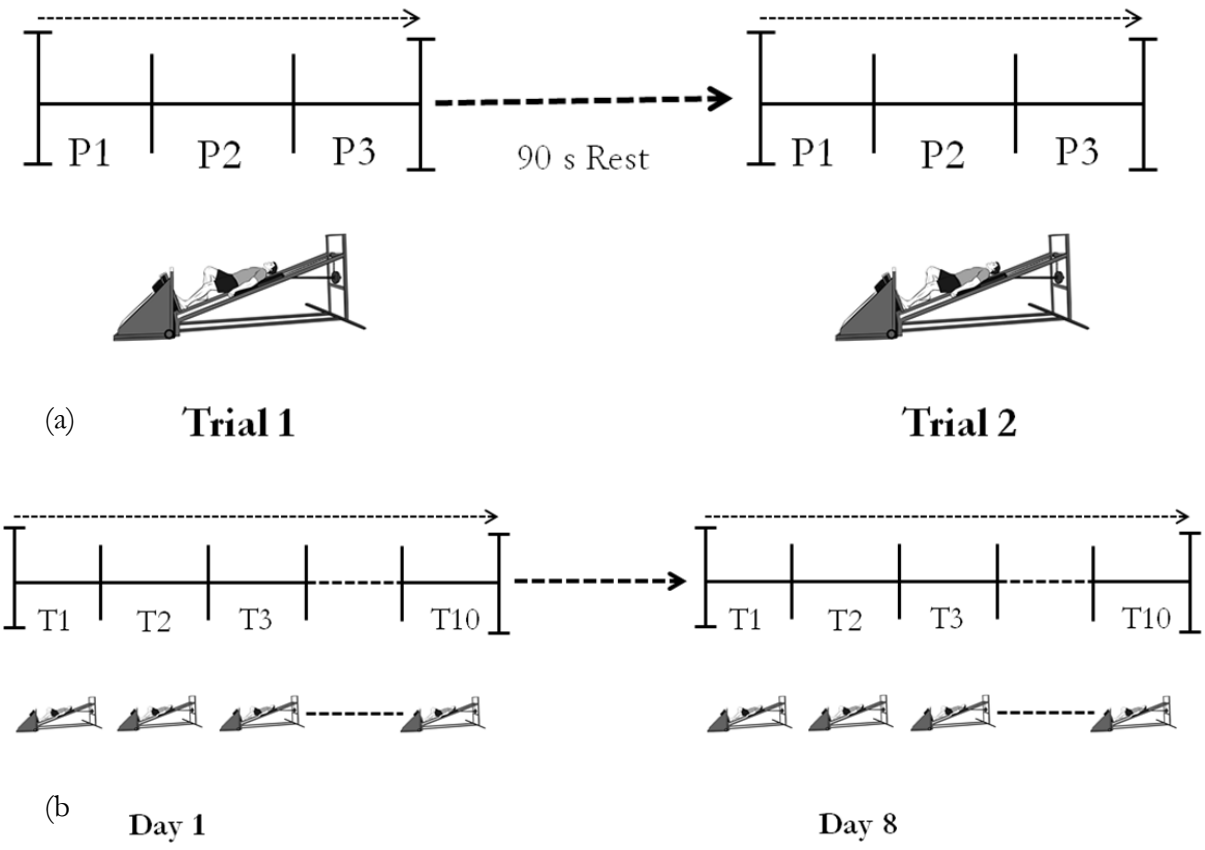

Figure 1: Experimental protocol: During this experiment, participants were tested on 2 seperat days, separated by 1 week (Day 1 and Day 8). On each testing occasion 10 trials were collected (T1-T10). Within a within a single trial, 30 hops were completed, which were divided into 3 equal period (P1-P3).

Retro-reflective markers were fixed to the skin of the participants according to a customised marker set and model for the lower limbs and pelvis, which was in accordance with an established cluster based method (Besier et al., 2003, Wu et al., 2002) (see Chapter 4, Figure 1b for illustration). Single markers were placed on the head of the first and fifth metatarsals, calcaneus, anterior superior iliac spines and posterior superior iliac spines. Marker clusters (three markers attached to a semi-rigid plastic base plate) were attached to the lateral aspects of both thighs and legs. The three-dimensional position of these markers was tracked using a 14-camera Vicon MX motion analysis system (Vicon, Oxford Metrics, Oxford, UK) operating at 250 Hz. Prior to dynamic trial collection, a static calibration trial was performed using a foot-calibration rig to measure foot abduction/adduction and inversion/eversion angles. The additional use of medial and lateral malleoli and medial and lateral femoral condyle marker locations determined anatomically relevant ankle, knee and hip joint axes of rotation and joint centres (Besier et al., 2003).

Ten hopping trials of 30s were completed interspersed by a 90 s rest period to provide full metabolic recovery (Nicol et al., 2006). The task involved continuous sub-maximal single-limb hopping on a custom-built SJS that included sled with low-friction wheels on corresponding tracks, reclined at 20° to horizontal. Perpendicular to the sled is a base plate, with an embedded 0.50 m x 0.50 m timing sensor operating at 1000 Hz capable of measuring temporal events of spring mass behaviour relative to ground contact (AccuGait; AMTI, Watertown, MA) (Figure 3).

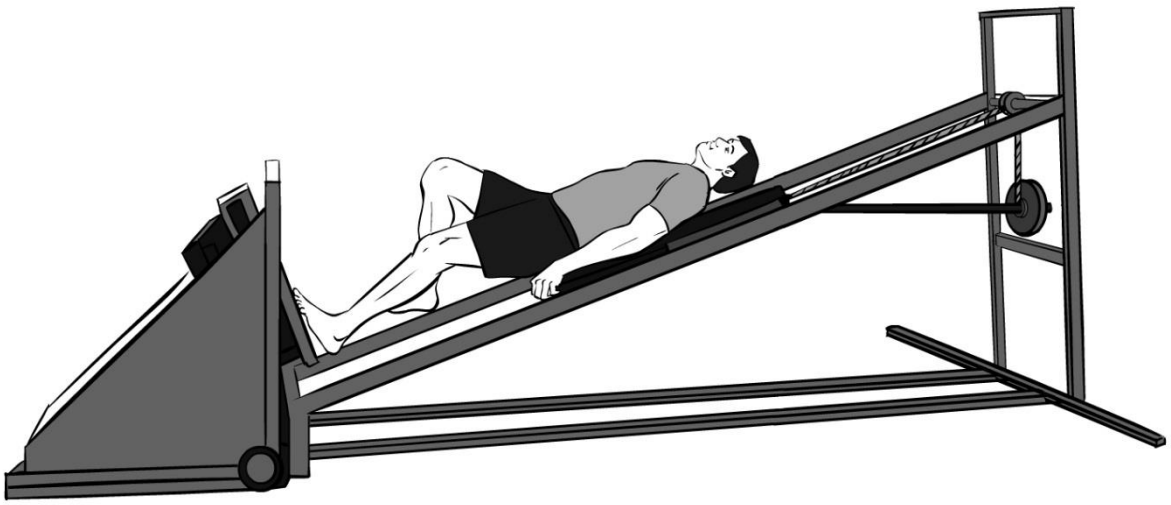

Figure 3: Custom-Built Low-Friction Sledge Jump System (from Gibson et al. (2013) with permission).

Participants were instructed to keep their non-hopping limb in a flexed position. Their foot rested on the SJS and they held onto the sliding component of the SJS in order to stabilise the thorax and upper limbs. Participants hopped on their preferred leg, as recommended by Flanagan and Harrison (2007). Whilst hopping, participants were instructed to keep the hip and knee as straight as possible, isolating the task to the ankle. They were instructed to hop at their natural frequency (recorded mean 1.16 ±0.09 Hz) and at a sub-maximal effort level, described as an effort they could maintain 'indefinitely'. Given the task was novel, a demonstration and familiarisation period was employed until they could hop as instructed comfortably; this process typically took less than 10

minutes. To ensure consistent hopping performance, instructions/feedback were kept consistent between participant, as variations in verbal cues can modulate lower limb stiffness in similar tasks (Arampatzis et al., 2001). The protocol was repeated exactly 7 days later.

DATA PROCESSING, ANALYSIS AND STATISTICS

Kinematic data were processed using Vicon Nexus motion analysis software (Vicon, Oxford Metrics, Oxford, UK). Data were filtered using a fourth-order low-pass Butterworth filter operating at a cut-off frequency of 20 Hz for the marker trajectories. All lower limb anatomical and joint coordinate systems were calculated in accordance with previously described standards outlined by the International Society of Biomechanics (Wu et al., 2002, Besier et al., 2003). Data were exported from Nexus for further analysis using a customised LabVIEW program (National Instruments, Austin, TX, 2011). For each hopping trial, ankle dorsi-plantar flexion angle at three time points, determined using touch pad data (80 ms prior to foot strike, at contact, and at take-off), as well as ankle, knee and hip stretch amplitude (joint range of motion between initial contact and maximum flexion) were calculated. In addition, lower limb stiffness was calculated using the method described by Dalleau et al. (2004) using the following equation:

$$K_n = \frac{\mathrm{M} \times \pi(T_f + T_c)}{T_c^2 \left(\frac{T_f + T_c}{\pi} \frac{T_c}{4}\right)}$$

K_n: Lower limb stiffness (Nm^{-1})
M: Mass (kg)
T_f: Flight time (s)
T_c: Contact time (s)

This calculation, designed for the study of variations in lower limb stiffness during sub-maximal hopping is calculated by modelling the ground reaction force as a sine wave as is expected from oscillation of a pure spring-mass model. Stiffness can therefore be calculated by measuring ground contact time and flight time for any given hop and validating support has been leant to this method (Riese et al., 2013). A 30 s epoch was chosen for each block of hopping ensuring at least 30 consecutive hops for all participants. Individual trials were then sub-divided into 3 periods of 10 hops (1-10, 11-20 and 21-30). Individual period values were the lowest level of analysis and a median value of each period was selected for comparison.

Reliability data were performed using Stata (StataCorp. 2011. Stata Statistical Software: Release 12. College Station, TX: StataCorp LP). All other statistical analyses were conducted using SPSS 19 (SPSS, Chicago, IL, USA). Data was checked for normality and values outside of two standard

deviations from the mean were considered outliers and were removed for the purposes of statistical analysis as described in previous literature employing SJS's (Ertelt and Blickhan, 2009).

Data were compared between the 3 periods, averaged across the 10 trials and 2 testing occasions, using a generalised linear mixed model with an alpha level set at 0.05. Within-trial and between-week reliability was assessed by generating ICC values for each trial. Week was taken into consideration using the variance of intraclass correlation coefficients in three-level model as described by Hedges et al. (2012). In this model, reliability values are attributed to contributing sources (subject and week) for each trial. Previous research (McLachlan et al., 2006, Joseph et al., 2013), supported by our own pilot testing indicated trial 1 may demonstrate poorer stability than trials 2-10; therefore an additional pooled analysis was conducted for trials 1-10 and trials 2-10. ICC values above 0.70 were considered to represent strong reliability; values between 0.50-0.69 were considered to represent moderate reliability and values below 0.50 were considered to represent poor reliability (Portney and Watkins, 2008). Finally a comparison by week was undertaken. According to the model by Hedges et al. (2012), low ICC values represent minimal influence on reliability between weeks and indicate higher reliability. Between-week consistency was assessed by calculating the SEM and minimal detectable difference (MDD) between weeks.

RESULTS

RELIABILITY ACROSS INDIVIDUAL TRIALS

The results from the within trials analysis revealed that there was no significant difference in lower limb stiffness, ankle and hip kinematics between periods (Table 1). A main effect was detected between periods for knee stretch amplitude ($p < 0.05$), and further analysis revealed periods 1, 2 and 3 were all significantly different.

RELIABILITY ACROSS MULTIPLE TRIALS

ICC values for all 7 measures are represented in Table 2. Lower limb stiffness demonstrated strong reliability; lowest ICC value was for trial 1 (0.69), besides which individual trials ranged from 0.74-0.84, with pooled trials 2-10 ICC of 0.77. Ankle angle 80 ms pre-contact demonstrated strong reliability; lowest ICC value was trial 8 (0.81), besides which individual trials ranged from 0.83-0.87, with pooled trials 2-10 of 0.86. Ankle angle at contact demonstrated strong reliability; lowest ICC value was for trial 4 (0.77), besides which values for individual trials ranged from 0.78-0.88 with pooled trials 2-10 ICC of 0.83. Ankle angle at take-off demonstrated moderate reliability;

lowest ICC value was for trial 2 (0.56), besides which ICC values for individual trials ranged from 0.64-0.79, with trials 2-10 ICC of 0.68. Ankle stretch amplitude demonstrated strong reliability; lowest ICC value was for trial 1 (0.38), besides which ICC values for individual trials ranged from 0.56-0.78, with trials 2-10 ICC of 0.71. Knee stretch amplitude demonstrated poor reliability; lowest ICC value was for trials 1-3 (0.00), besides which, ICC values for individual trials ranged from 0.05-0.30, with pooled trials 2-10 ICC of 0.11. Hip stretch amplitude also demonstrated poor reliability; lowest ICC value was for trial 2 (0.06), besides which individual trials ranged from 0.18-0.51, with pooled trials 2-10 ICC of 0.28.

RELIABILITY BETWEEN WEEKS

The results regarding the influence of week demonstrated a weak effect on the variation within the results for lower limb stiffness (ICC trials 2-10 pooled = 0.19), ankle angle 80 ms pre-contact (ICC trials 2-10 pooled = 0.11), ankle angle at contact (ICC trials 2-10 pooled = 0.12), ankle angle at take-off (ICC trials 2-10 pooled = 0.27) and ankle stretch amplitude (ICC trials 2-10 pooled = 0.24). Week demonstrated a moderate influence on variation within the results for knee stretch amplitude (ICC trials 2-10 pooled = 0.50) and hip stretch amplitude (ICC trials 2-10 pooled = 0.52). Table 3 shows the SEM and MDD values for variables between weeks 1 and 2. These indicate that with the exception of knee stretch amplitude (SEM 5.39° and SDD 14.94°), the values were indicative of good consistency (lower limb stiffness SEM 0.42 kNm^{-1}, MDD 1.16 kNm^{-1}; ankle and hip angles SEM 0.97-1.45°, MDD 2.69-4.02°).

DISCUSSION AND IMPLICATIONS

Understanding lower limb mechanics during the SSC is crucial for gaining insight into injury and rehabilitation of the lower limb. The SMM offers a robust model by which to explore this. Whilst SJS's have advantages over running and hopping in terms of isolating specific components of the SSC, such methods require robust measurement properties. Our goal was to determine the temporal reliability of lower limb stiffness and kinematics within trial, between ten trials, and across two testing occasions. Whilst McLachlan et al. (2006) have measured SSC reliability and Furlong and Harrison (2013) have measured within trial SJS reliability, this is the first study exploring the reliability of a SSC task on a SJS including between trials and between occasion measures. We found that the SJS is reliable within trial, between trials and across testing occasions. Our data shows that excluding knee stretch amplitude, no significant differences were present between hopping periods (see Table 1). Across trials, lower limb stiffness and 3 out of 4 ankle kinematic measures demonstrated strong reliability (ICC's ranging from 0.71 to 0.86 (see Table 2)). Ankle

angle at take-off demonstrated moderate reliability (ICC = 0.68), whilst knee and hip stretch amplitude demonstrated poor reliability (ICC = 0.11 and 0.28 respectively) (see Table 2). Testing occasion had minimal influence on measures that had moderate/strong inter-trial reliability and a moderate influence on measures that had poor inter-trial reliability (see Table 2).

RELIABLE MEASURES

Consistent with our hypothesis, for our main parameters of interest (lower limb stiffness and ankle kinematics), reliability was high. Furthermore, our results demonstrate that for lower limb stiffness, ankle stretch amplitude, and knee stretch amplitude, exclusion of the first trial optimises reliability. The exclusion of the first trial improved reliability presumably by mitigating familiarisation/learning that may have occurred thereby providing the first objective evidence supporting the empirically-derived practice of excluding first and final hops from hopping trials using this methodology (Joseph et al., 2013, Furlong and Harrison, 2013). The findings of high reliability and low SEM (relative to the overall total possible range of kinematic scores) demonstrated that this model is stable and judgements about true change in outcome measures across time can be confidently made.

Whilst the use of SJS's is not new (Aura and Komi, 1986, Ertelt and Blickhan, 2009, Kramer et al., 2010), ours is the first to report measures of temporal performance reliability. Harrison et al. (2004) investigated differences in SSC-function on a SJS between power and endurance athletes, reporting reliability data from 4 drop jumps (ICC 0.996). Likewise, Flanagan and Harrison (2007) performed a reliability analysis of SSC behaviour demonstrating strong reliability of vertical stiffness, flight time and reactive strength index (ICC > 0.85). Our reliability levels were slightly lower than theirs and this may reflect differences in testing protocols; those by Flanagan and Harrison (2007) were based on a minimal number of maximal SSC's, whereas ours were based on a high volume of sub-maximal SSC's. Bearing most resemblance to our own study, Furlong and Harrison (2013) explored within-trial reliability of SJS sub-maximal hopping, observing good reliability of kinematic measures of ankle function (ICC >0.95). This study differed from our own by employing an inverted SJS where participants lay stationary and projected a mobile weighted SJS, which may explain the differences in ICC values. Another possible explanation for our lower ICC values is the increased movement that is likely to occur in our study, likely representing increased variability as task complexity increases.

UNRELIABLE MEASURES

Contrary to our hypothesis, some of our measures demonstrated sub-optimal reliability (ankle angle at take-off, knee and hip stretch amplitudes). Ankle angle at take-off demonstrated moderate reliability; this may be expected as this measure is taken at the final stage of the task, where variation could be expected to be greatest as the foot leaves the ground and the task is completed (Austin et al., 2002). Likewise, poor reliability for knee and hip kinematics were observed. The most likely explanation for this finding lies in the range of motion in which the hip and knee operated during this task. In order to isolate the SSC task to the ankle, the joint where SSC regulation principally occurs (Farley and Morgenroth, 1999), we instructed participants to constrain hip and knee motion. This was successful, with stretch amplitude values of 9° and 6° per trial for the knee and hip respectively. Given the small ranges involved any variations between individuals and between trials would magnify poor ICC reliability. For example, low knee stretch amplitude ICC's were generated by a corresponding SEM of 5.4°.

IMPLICATIONS

Given the high reliability of our measures of lower limb stiffness and ankle kinematics, this experimental model can be confidently applied for studies where repeated measures of SSC behaviour are required. Given optimal reliability (excluding the first trial), we recommend such exclusion in future studies utilising a SJS. A further benefit of using the SJS over upright hopping for a sub-maximal task is that it limits the impact of fatigue that may directly influence results. The use of an upright hopping model could conceivably result in fatigue towards the end of any given 30-hop trial, as indicated by an increase in performance variability. In using the SJS, our data (excluding knee stretch amplitude) remains stable in the final period of hopping. In the presence of discernible fatigue, one might expect to see a change in data, such as an increase in variability or a drift in performance. The absence of such patterns indicates that participants performing this task do not experience fatigue sufficient to contaminate performance. Therefore, this current study provides the basis for using this methodology to predict true change in performance across time/repeated trials. Finally, this is a recommended model for measuring lower limb stiffness where two specific areas of interest exist. Firstly, if the ankle is the primary region of interest, the hip and knee can be well-constrained. This would be advantageous if researchers were interested in modulatory triggers believed to occur predominantly at the ankle, such as pathological conditions (e.g. Achilles tendinopathy), or training of the ankle muscles (e.g. strengthening). Secondly, given the unloaded nature of the task, it may be possible to 'magnify' changes occurring

as a result of these modulatory triggers. For instance, it is known that Achilles tendinopathy modulates leg stiffness (Maquirriain, 2012) during an upright sub-maximal hopping task but the role of stiffness modulation at the ankle is unknown. This model could provide further insight into how the pathology affects SSC-behaviour locally to the ankle.

where the impact of changes at the ankle are likely to be the predominant modulator of lower limb stiffness such as pathological conditions (e.g. Achilles tendinopathy), or training of the ankle (e.g. strengthening, proprioceptive exercise).

LIMITATIONS

Historically SJS's have been criticised for task unfamiliarity contributing to lack of ecological validity; we attempted to account for this by sampling a large number of hops within trials and a large number of trials (10 trials of 30 hops). This contrasts previous similar studies where typical sampling values vary from 3 trials of 20 hops (Lloyd et al., 2009) to 10 trials of 10 hops (Joseph et al., 2013). In addition, our instructions to participants indicated they should hop at a sub-maximal level, described as an effort they could maintain 'indefinitely'. Removing the subjectivity of this instruction may further improve reliability. Furlong and Harrison (2013) quantified effort, where optimal reliability was achieved based on the instructions to hop at a level of 2 out of 5 on a numerical rating scale (5 representing maximal force they could apply whilst still successfully completing the task). Horita et al. (1996) have attempted to quantify/define sub-maximal hopping. Using the SJS described by Aura and Komi (1986), they defined sub-maximal hopping as hopping at 70% of the maximal countermovement jump. Using this method, participants fatigued after 3 minutes of hopping. Given these results we can be confident that our protocol would not have resulted in significant fatigue, reflected in the reliability of our results. Our study required participants to hop at a self-selected pace. Whilst hopping at a self-selected frequency results in poorer reliability than a forced frequency (Joseph et al., 2013), Dalleau et al. (2004) suggests a difference of 0.6 Hz in hopping frequency is required to significantly alter stiffness values. Participants in our study did not vary to this extent, implying that the SJS has the advantage of yielding reliable measures while allowing participants to hop in a 'natural/preferred' manner. Regarding statistics, the use of a three-level intraclass correlation coefficient model presents a novel interpretation of our data, which is in contrast to conventional interpretations of ICC data. It is important to recognise that using this model, the low ICC values observed between weeks represent high levels of stability when comparing measures between weeks. This is because the value reflects the influence that week has on the stability, which our data indicate is small. Finally, it must be remembered that whilst this SJS yields reliable data indicative of motor performance

during a SSC-task, findings using a tool of this nature ought not to be directly extrapolated to performance in other SSC-tasks (e.g. upright hopping, running). However, this paper supports the use of this reliable tool employing within- and between-participant study designs and its utility would be highest where SSC-behaviour is evaluated in response to temporal or interventional factors.

CONCLUSION

In summary, we have shown that the use of a SJS to measure SSC behaviour yields reliable results within multiple hops and trials, and between testing occasions. Likewise, our description of SEM of this task provides a firm basis upon which future methodologies utilising this protocol may infer true change across experimental conditions. As such we recommend that future studies utilising a SJS employ similar methods of measurement as described here. Notably, a familiarisation period is recommended where data from the initial trial is not analysed. Otherwise, multiple-trial and multiple-occasion testing is an appropriate strategy for measuring SSC behaviour.

CONFLICT OF INTEREST

The authors of this study have no professional relationships with companies or manufacturers who will benefit from the results of the present study.

ACKNOWLEDGEMENTS

The authors would like to thank Paul Davey (Curtin University) for his assistance with data processing, and the research volunteers for their participation.

TABLES

Table 1: Means (and standard deviations) for different periods during sleigh hopping (weeks 1&2 pooled) and standard error of measurement and minimal detectable difference between Weeks 1 and 2

Means (± SD) for hopping periods (weeks 1&2 pooled). * Significant difference between periods 1-3 ($p < 0.05$)				SEM and MDD between Weeks 1 and 2	
Variable	**Period 1**	**Period 2**	**Period 3**	**SEM**	**MDD**
Lower limb stiffness (kNm^{-1})	9.4 ±3.9	9.4 ±3.9	9.4 ±3.9	0.42	1.16
Ankle angle 80 ms pre-contact (dorsiflexion)	-19.6 ±10.5°	-19.7 ±10.4°	-20.3 ±10.7°	0.97	2.69
Ankle angle @ contact (dorsiflexion)	-21.2 ±10.5°	-21.2 ±10.2°	-20.7 ±10.2°	1.25	3.46
Ankle angle @ take-off (dorsiflexion)	-30.4 ±8.3°	-30.5 ±8.5°	-30.2 ±8.5°	1.28	3.55
Ankle stretch amplitude	28.5 ±9.6°	28.6 ±10.2°	29.8 ±9.5	1.45	4.02
Knee stretch amplitude	9.0 ±4.4°*	9.8 ±4.8°*	10.6 ±5.0°*	5.39	14.94
Hip stretch amplitude	6.3 ±2.3°	6.3 ±2.3°	6.3 ±2.3°)	1.26	3.49

SD Standard deviation
SEM Standard error of measurement
MDD Minimal detectable difference

Table 2: Reliability of Stiffness Measures (ICC) and SEM for Trial, Session and Week

(SEM); [95% Confidence Intervals]
* SEM for lower limb stiffness in kNm^{-1}
# SEM for angular measurements in degrees
Text highlighted in blue represents trial findings with ICC value over 0.70

Trial(s)	Stiffness *	Ankle Angle 80 ms Pre-Contact #	Ankle Angle at Contact #	Ankle Angle at Take-Off #	Ankle Stretch Amplitude #	Knee Stretch Amplitude #	Hip Stretch Amplitude #
1	0.69 [0.41-0.96] (0.45)	0.84 [0.70-0.99] (1.28)	0.83 [0.68-0.99] (1.44)	0.69 [0.42-0.95] (1.18)	0.38 [0.00-0.82] (1.43)	0.00 [0.00-0.00] (2.97)	0.27 [0.00-0.72] (0.63)
2	0.75 [0.53-0.98] (0.37)	0.86 [0.73-0.99] (1.33)	0.83 [0.67-0.98] (1.74)	0.56 [0.20-0.91] (0.89)	0.61 [0.29-0.93] (1.33)	0.00 [0.00-0.00] (2.07)	0.06 [0.00-0.54] (0.74)
3	0.80 [0.61-0.99] (0.30)	0.87 [0.74-0.99] (1.12)	0.85 [0.71-0.99] (1.38)	0.65 [0.36-0.95] (0.96)	0.68 [0.40-0.96] (1.05)	0.00 [0.00-0.00] (1.84)	0.18 [0.00-0.65] (0.71)
4	0.76 [0.54-0.98] (0.42)	0.84 [0.68-1.00] (1.16)	0.77 [0.53-1.00] (1.24)	0.64 [0.33-0.95] (1.10)	0.70 [0.43-0.96] (1.27)	0.05 [0.00-0.51] (2.17)	0.19 [0.00-0.65] (0.80)
5	0.75 [0.52-0.97] (0.41)	0.87 [0.75-0.99] (1.40)	0.83 [0.67-0.99] (1.33)	0.79 [0.60-0.98] (1.27)	0.72 [0.47-0.97] (1.25)	0.12 [0.00-0.55] (1.97)	0.25 [0.00-0.70] (0.67)
6	0.79 [0.59-0.98] (0.30)	0.83 [0.66-1.00] (0.89)	0.88 [0.76-1.00] (1.13)	0.71 [0.42-1.00](0.93)	0.75 [0.52-0.98] (1.22)	0.23 [0.00-0.69] (1.51)	0.42 [0.00-0.90] (0.62)
7	0.84 [0.69-0.99] (0.35)	0.86 [0.72-1.00] (1.27)	0.83 [0.67-1.00] (1.45)	0.68 [0.41-0.95] (1.46)	0.56 [0.17-0.95] (1.27)	0.13 [0.00-0.59] (1.74)	0.34 [0.00-0.76] (0.87)
8	0.74 [0.51-0.97] (0.38)	0.81 [0.62-1.00] (1.29)	0.78 [0.56-1.00] (1.40)	0.69 [0.43-0.95] (1.58)	0.78 [0.58-0.99] (1.46)	0.13 [0.00-0.63] (1.58)	0.25 [0.00-0.75] (0.72)
9	0.78 [0.57-0.98] (0.34)	0.86 [0.73-1.00] (1.16)	0.84 [0.68-0.99] (1.12)	0.68 [0.40-0.96] (1.42)	0.76 [0.52-0.99] (1.15)	0.30 [0.00-0.76] (1.13)	0.51 [0.14-0.88] (0.56)
10	0.76 [0.53-0.98] (0.31)	0.85 [0.72-0.99] (1.03)	0.80 [0.62-0.98] (1.23)	0.62 [0.30-0.94] (1.11)	0.63 [0.29-0.97] (0.99)	0.30 [0.00-0.77] (1.38)	0.34 [0.00-0.81] (0.57)
1-10	0.76 [0.56-0.96] (0.87)	0.86 [0.74-0.98] (1.97)	0.83 [0.69-0.97] (2.28)	0.68 [0.42-0.93] (1.99)	0.68 [0.43-0.92] (2.33)	0.07 [0.00-0.37] (3.17)	0.28 [0.00-0.64] (1.10)
2-10	0.77 [0.57-0.96] (0.82)	0.86 [0.74-0.98] (1.86)	0.83 [0.69-0.97] (2.17)	0.68 [0.42-0.93] (1.88)	0.71 [0.47-0.94] (2.05)	0.11 [0.00-0.43] (2.91)	0.28 [0.00-0.67] (1.03)

Table 3: Standard Error of Measurement and Minimal Detectable Difference between Weeks 1 and 2

	Standard Error of Measure (SEM)	Minimal Detectable Difference (MDD)
Lower limb stiffness (kNm^{-1})	0.42	1.16
Ankle angle (degrees) 80 ms pre-contact (dorsiflexion)	0.97	2.69
Ankle angle (degrees) @ contact (dorsiflexion)	1.25	3.46
Ankle angle (degrees) @ take-off (dorsiflexion)	1.28	3.55
Ankle stretch amplitude (degrees)	1.45	4.02
Knee stretch amplitude (degrees)	5.39	14.94
Hip stretch amplitude (degrees)	1.26	3.49

4. THE MODULATORY INFLUENCE OF ACHILLES TENDINOPATHY ON SPRING FUNCTION

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Abstract

Objectives

To describe stretch shortening cycle behaviour of the ankle and lower limb in patients with Achilles tendinopathy (AT) and establish differences with healthy volunteers.

Design

Between-subjects case-controlled

Methods

Fifteen patients with AT (mean age 41.2 ± 12.7 years) and eleven healthy volunteers (CON) (mean age 23.2 ± 6.7 years) performed sub-maximal single-limb hopping on a custom built sledge-jump system. Using 3D motion analysis and surface EMG, temporal kinematic (lower limb stiffness, ankle angle at 80 ms pre-contact, ankle angle at contact, peak ankle angle, ankle stretch amplitude) and EMG measures (onset, offset and peak times relative to contact) were captured. Data between AT and CON were compared statistically using a linear mixed model.

Results

Patients with AT exhibited significantly increased lower limb stiffness when compared to healthy volunteers ($p<0.001$) and their hopping range was shifted towards a more dorsiflexed position ($p<0.001$). Furthermore, ankle stretch amplitude was greater in AT compared with healthy volunteers ($p<.001$). A delay in muscle activity was also observed; soleus onset ($p<.001$), tibialis anterior peak ($p=0.026$) and tibialis anterior offset ($p<.001$) were all delayed in AT compared with CON.

Conclusions

These findings indicate that patients with AT exhibit altered stretch-shortening cycle behaviour during sub-maximal hopping when compared with healthy volunteers. Patients with AT hop with greater lower limb stiffness, in a greater degree of ankle dorsiflexion and have a greater stretch amplitude. Likewise, delayed muscle activity is evident. These findings have implications in terms of informing the understanding of the pathoaetiology and management of AT.

Keywords

Achilles tendon

Tendinopathy

Overuse injury

Plyometric exercise

Stretch shortening cycle

Hopping

Main Text

Introduction

Achilles tendinopathy (AT) is a common clinical syndrome experienced by active individuals, characterised by a combination of pain, diffuse or localised swelling, and impaired performance arising from overuse. Whilst AT is a challenging condition to manage and evidence supports a conservative approach [1], frequent reports of sub-optimal clinical outcomes (e.g. van der Plas, de Jonge, de Vos, van der Heide, Verhaar, Weir, Tol [2]) suggest our understanding of the condition is incomplete.

The pathoaetiology of AT is complex [3] and whilst multiple factors clearly interact in the development of AT, mechanical factors dominate where the cumulative load placed upon the tendon exceeds its mechanical capacity, resulting in a 'failed loading response' [4]. Given the central role that the Achilles tendon plays in the stretch-shortening cycle (SSC), it seems reasonable that aberrations in SSC behaviour and AT may be related.

The stretch shortening cycle (SSC) is a phenomenon that describes the natural pre-activation of a musculotendinous unit, followed by an eccentric phase and a subsequent concentric phase [5]. Its role is to simplify the motor control of locomotion and optimise locomotor efficiency [6]. It has been suggested that aberrations in SSC performance may result in injury [7] and evidence exits describing how AT is associated with alterations in measures indicative of altered SSC behaviour [8-10]. Unfortunately, these studies collectively provide an incomplete picture of the biomechanical changes that occur in the presence of Achilles tendinopathy. In particular kinematic evaluation of the ankle in the sagittal plane, and the associated activity of key agonist/antagonist muscles during a SSC task have yet to be explored in this population. Given that the principal plane of motion for the SSC during running is sagittal, a greater depth of understanding at this level would provide further insight into the relationship between AT and SSC behaviour.

In this study we compared SSC behaviour during sub-maximal single limb hopping in individuals with AT and in a group of healthy volunteers (CON). We hypothesised that in the AT group

alterations in SSC behaviour would be observed. Specifically, we hypothesised that when compared with CON, the AT group would exhibit decreased lower limb stiffness, hop in a greater degree of dorsiflexion and have a greater stretch amplitude. Likewise, we hypothesised that in AT, delayed muscle activity in both the agonist and antagonist muscles would be observed. To test these hypotheses, we measured SSC behaviour during a sub-maximal hopping task on a sledge-jump system (SJS). The utility of such a system is its capacity to limit variability of movement and mitigate fatigue; such systems have been used in the past to explore a variety of conditions influencing SSC behaviour [11].

Achilles tendinopathy is a common injury and challenging to treat in part due to our incomplete understanding of the pathoaetiological drivers of the condition. The findings of this study may have connotations in both deepening our understanding of the mechanical pathoaetiology of AT, and potentially informing the development and refinement of therapeutic interventions for AT.

Methods

This study employed a between-subjects case-controlled design and included 15 patients with AT (mean age 41.2 ± 12.7 years; 9 male: 6 female; affected side 4 left: 11 right) and 11 CON volunteers (mean age 23.2 ± 6.7 years; 5 male: 6 female). CON volunteers were recruited from the local university community and AT patients were recruited from local medical practices in Perth, Western Australia. AT inclusion criteria included a >3 month history of unilateral mid-portion Achilles tendon pain, a VISA-A score <80/100, with mid-portion pain and thickening identified on palpation. Exclusion criteria for both groups included an absence of co-existing lower quadrant musculoskeletal pathology or other visual/motor impairment(s). Informed consent approved by the Human Research Ethics Committee of Curtin University was obtained from all participants prior to testing (HR28/2010).

Retro-reflective markers were fixed to the skin of participants according to a customised marker set and model for the lower quadrant (see Figure 1b), set according to an established cluster-based method [12]. This established set-up enabled determination of anatomically-relevant ankle, knee and hip

joint axes of rotation and joint centres [12], and subsequent motion capture was performed using a 14-camera Vicon MX motion analysis system (Vicon, Oxford Metrics, Oxford, UK) operating at 250 Hz.

Temporal measures of soleus and tibialis anterior muscle activity were recorded using an AMTI-8 (Bortec Biomedical Ltd) surface EMG (sEMG) system. Bipolar differential surface electrodes (Ag / AgCL) were placed on the belly of each muscle with the reference electrode on the medial malleolus. Skin impedance (< 15kOhms) was achieved with skin preparation and signals were pre-amplified, analogue filtered (10 – 500Hz band pass) and then digitised using an 18 bit A-D card with a sampling rate of 1000Hz. All data was temporally synchronised and recorded on dedicated hardware running a customised Labview program (National Instruments, Austin, Texas, 2011).

Participants attended on a single testing occasion. They were instructed to continue with their normal everyday activities but to refrain from undertaking unfamiliar activities in the week prior to testing. In addition, they were instructed to avoid vigorous physical activity in the 24 hour period prior to data collection. AT participants were further instructed to not receive novel therapeutic interventions in the 2 weeks prior to testing.

Participants were instructed on the performance of sub-maximal hopping on a custom-built SJS (see Figure 1a). The task involved continuous sub-maximal single-limb hopping on the SJS keeping the knee fully extended; participants hopped on their affected (AT) or dominant (CON) limb for 15 seconds trials, before a 30 s rest period. Five trials were repeated.

Data was processed using Vicon Nexus motion analysis software (Vicon, Oxford Metrics, Oxford, UK). Kinematic data were filtered using a fourth order Butterworth filter operating at a frequency cut-off of 20 Hz for the marker trajectories and 50 Hz for the ground contact data as determined by residual analysis [13]. All lower limb anatomical and joint coordinates were calculated in accordance with the standards outlined by the International Standards of Biomechanics and have been previously described [12]. Data was exported from Nexus for further analysis using a customised LabVIEW program (National Instruments, Austin, Texas, 2011). For each hopping trial, the following ankle kinematic measures were calculated; ankle angle 80 ms prior to ground contact, ankle angle at ground

contact, peak ankle angle and ankle stretch amplitude. In addition, lower limb stiffness was calculated using the method described by Dalleau, Belli, Viale, Lacour, Bourdin [14] (figure 1c). In addition, temporal measures of muscle activity for soleus and tibialis anterior were calculated relative to ground contact; onset, peak and offset.

The EMG signal was full wave rectified and onsets detected using an integrated protocol [15]. Trial linear envelopes (LE) were created using a fourth-order, zero-lag Butterworth low-pass filter (10 Hz) and temporally synchronised to (T=0) foot contact.

Statistical analysis was conducted using SPSS version 20 (SPSS, Chicago, Il, USA). Descriptive statistics were used to establish mean values for all variables in each group (AT vs. CON). A linear mixed model was used for all statistical comparisons between groups. Age, gender height and body mass were input as covariates and adjusted for within the model. A fixed main effects model was fitted, with a type III sum of squares used to assess statistical significance. Parameter estimates were utilised, and main effects were compared as pairwise comparisons using a Bonferoni correction. The residuals were tested for normality as required by the linear mixed model.

Results

Mean (and standard deviation) values for our biomechanical measures are presented in Table 1. Patients with AT exhibited increased lower limb stiffness when compared to CON ($p<0.001$) and their hopping range was shifted towards a more dorsiflexed position ($p<0.001$). Ankle stretch amplitude was greater in AT compared with CON ($p<.001$). A delay in muscle activity was observed in soleus onset ($p<.001$), tibialis anterior peak ($p=0.026$) and tibialis anterior offset ($p<.001$) in AT compared with CON

Discussion

This is the first study to describe SSC-behaviour during a sub-maximal hopping task in patients with AT with a detailed focus on sagittal plane behaviour. AT is a common injury whose pathoaetiology is unclear and as a result management remains sub-optimal. Whilst we have some understanding of the changes in SSC behaviour that correspond with the pathology, our understanding of sagittal plane

SSC behaviour has been to this point somewhat limited. In this comparative study, we found that when compared to healthy volunteers, individuals with AT exhibit altered SSC behaviour during a sub-maximal hopping task. This has been demonstrated in the following ways. Firstly, individuals with AT hopped with increased lower limb stiffness. In addition they hopped in greater dorsiflexion, and with greater overall stretch amplitude. We also found that soleus onset, tibialis anterior peak, and tibialis anterior offset timing is delayed in AT (see Table 1). Whilst changes in SSC behaviour has been investigated in AT [8-10], this is the first study that has isolated SSC performance to the ankle in such a manner that has enabled detailed examination of sagittal plane ankle behaviour in this manner.

Contrary to our hypothesis and the existing literature, lower limb stiffness was increased in AT. Lower limb stiffness has only been previously measured in individuals with AT on limited occasions, and in all studies stiffness was found to be reduced. For example, Maquirriain [10] measured lower limb stiffness during an upright hopping task in athletes with AT, observing reductions in stiffness of the affected, compared with the unaffected leg. Arya, Solnik, Kulig [16] conducted the only study to date where stiffness has been compared with a healthy control group, which they did using an upright hopping model. They found that overall lower limb stiffness reduced, achieved by shifting to a knee strategy. As such, the most likely explanation for our findings is that in the presence of AT, the change in behaviour is done so with the aim of limiting exposure of the tendon to the painful stimulus. Assuming the strategy used to do so is task-dependent, our participants were likely attempting to reduce overall ankle load by limiting both ground contact time and reaction forces. Our findings, combined with those of Arya, Solnik, Kulig [16] suggest that one possible solution is that ankle stiffness increases to increase lower limb stiffness whilst knee stiffness reduces to reduce peak loading. If this theory is correct, our findings of increased stiffness could therefore be explained by the fact that our experimental model largely removes the ability of participants to redistribute a stiffness strategy to knee. Other less likely possible explanations for the increased stiffness values observed include the novel nature of the task, the absence of pain due to unloading, and the lack of perceived threat due to the secure nature of the task.

Consistent with our hypothesis, the AT group hopped in greater dorsiflexion at all recorded time points including; 4.3° at contact and 7.4° at peak. Our findings are consistent with those of Ryan, Grau, Krauss, Maiwald, Taunton, Horstmann [17], who investigated ankle range of motion in patients with AT compared with healthy volunteers, finding that runners with AT had comparatively increased dorsiflexion range of motion. Whilst we observed an increase in dorsiflexion stretch amplitude, Ryan, Grau, Krauss, Maiwald, Taunton, Horstmann [17] found similar findings on observation of eversion stretch amplitude. When combined with the findings of increased stiffness during the hopping task, it might be suggested that whilst increasing stiffness as a strategy to limit exposure to ground contact, individuals with AT lack the structural apparatus to achieve this in the most effective manner. During SSC tasks, elongation of the TA occurs in the presence of a 'quasi-isometric' plantarflexion contraction [18]. However, it has been reported that AT is associated with reduced tendon stiffness [19, 20], so the increased stretch amplitude may be viewed as an indicator of reduced tendon stiffness.

Our findings on temporal muscle activity partially supported our hypothesis with 3 of our 6 measures demonstrating delays in AT (see Table 1). The most likely explanation for this observation is that the pain experienced during the contact phase of the SSC can trigger inhibition of neuromuscular activity [21] resulting in delays or reductions in EMG activity [9]. Alternatively, it is possible that the earlier offsets observed are a manifestation of a learnt behaviour, adopted in response to chronic changes in muscular performance or alterations in sensory input secondary to changes in tendon compliance [22], which in turn affect feedforward muscle activity. Likewise, this sensory input may also be negatively influenced by the increased compliance observed in patients with AT [20]. Our findings are consistent with those of Azevedo, Lambert, Vaughan, O'Connor, Schwellnus [23], who observed reduced muscle activity in the tibialis anterior of runners with AT performing a running task. Likewise, Baur, Muller, Hirschmuller, Cassel, Weber, Mayer [9], investigating neuromuscular control of tibialis anterior, fibularis and gastrocnemius muscles in runners with AT reported that whilst no differences were observed in pre-activation of any muscles studied when compared with controls, gastrocnemius activity was reduced during the eccentric phase of the SSC. Finally, Wyndow, Cowan, Wrigley, Crossley [24] observed a delay in soleus offset in AT compared to controls during a running task in the

order of 18 (± 22) ms. The delay observed in tibialis anterior offset might suggest a global strategy of muscle delay in AT. The global nature of this strategy supports the findings of Smith, Honeywill, Wyndow, Crossley, Creaby [25] who observed delayed activation of gluteus medius and gluteus maximus in runners with AT.

Some methodological issues require consideration. The descriptive nature of this study limits causative interpretations; it is not possible to elucidate the temporal relationship between altered SSC behaviour, AT symptoms and pathology. Whilst age and gender matching was not ideal, the utilisation of the linear mixed model accounted for by its inclusion as a covariate in the model. We did not match participants for activity levels, which would have improved the homogeneity of groups. However, benefits exist in terms of external validity when using a heterogeneous group, which nevertheless remained reflective of the AT population. Finally, in this study, participants hopped rather than ran. In doing so, this enabled us to make a detailed exploration of SSC behaviour and whilst this is the first study to have conducted such an analysis in AT, its use in other experimental models does exist [11].

We speculate that many of our findings support the theory that biomechanical changes result in altered tendon loading and may be pathogenetic for AT. During SSC tasks, the plantarflexors control ankle dorsiflexion eccentrically and the shift in operating range towards greater dorsiflexion may increase the task demands of the plantarflexors. This may be further magnified by the possibility of a shift in the angle to peak torque that has been observed in other lower limb conditions [26]. This is an area of enquiry that justifies further exploration.

It has been speculated that muscle (pre-)activation is the strategy employed to increase stiffness to absorb impact forces [27], and the delays in muscle activity observed in our study could indicate an increase in tendon loading during the eccentric phase of the SSC. Although our findings require further confirmation, it is possible that differential stress generated by altered muscular activation generate altered intratendinous loads and may be associated with the pathogenesis of AT, as suggested by Wyndow, Cowan, Wrigley, Crossley [24].

Since no prospective data exists, we can only speculate if the observed changes in SSC behaviour in AT is a cause or consequence of the condition. Regardless of whether or not the observed changes in SSC behaviour are causative or not, they do inform clinical applications. It seems clear that focus should be placed on developing interventions to optimise SSC behaviour in line with modifying the impairments identified in this and other studies as a key factor to improving patient-centred functional outcomes.. In particular, our recommendations would include strategies to encourage regulation of stiffness strategies, reductions in dorsiflexion during ground contact and appropriate enhancement of agonist/antagonist timing around the ankle.

When considering these descriptive findings, it is recommended that prospective studies are undertaken to further explore whether the altered SSC behaviour observed in patients with AT is a consequence or a predisposing factor. In line with the clinical applications suggested by our findings, further study is also recommended exploring therapeutic interventions that modify SSC behaviour, such as strength training and plyometric training, developed with the relevant precautions required for this population in mind.

Conclusions

Our observation of SSC behaviour changes in Achilles tendinopathy showed relevant changes in lower limb stiffness, ankle joint kinematics and muscle activity. These findings support the theory of a mechanical pathoaetiological mechanism contributing to the development of Achilles tendinopathy and support the use of therapeutic interventions designed to optimise SSC behaviour in this patient population. Although these findings support these theories, further prospective studies are recommended to clarify causality.

Practical implications

- The shift in ankle mechanics during sub-maximal hopping towards a more dorsiflexed position with larger stretch amplitude and the associated global delay in muscle activity is likely to result in excessive load being placed on the Achilles tendon.

- The observed alterations in stretch shortening cycle behaviour in patients with Achilles tendinopathy lend support to the theory that failed loading is a pathoaetiological component of the condition.
- Clinicians should consider applying therapeutic interventions that optimise SSC behaviour in patients with AT.

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326 **Tables**

Table 1. Mean (standard deviation) values for participant characteristics and biomechanical derived variables

Participant Characteristics	**Achilles Tendinopathy**	**Healthy Volunteers**
n = (male: female)	15 (9:6)	11(3:8)
Age (years)	41.2 (± 12.7)[a]	23.2 (± 6.7)
Height (cm)	174.1 (± 9.6) [a]	170.1 (± 8.2)
Mass (kg)	82.0 (± 12.2) [a]	70.7 (± 13.3)
VISA-A (0-100)	64.7 (± 12.7) [a]	100
Tendinopathy duration (months)	12.06 (±8.24)	na
Stiffness		
Lower Limb Stiffness (kNm^{-1})	8.8 (1.3) [a]	4.5 (2.6)
Ankle Kinematics		
Ankle Angle 80 ms pre-contact (° dorsiflexion)	-15.3 (9.97) [a]	-19.5 (8.97)
Ankle Angle at contact (° dorsiflexion)	-12.9 (10.0) [a]	-17.0 (9.3)
Peak Ankle Angle (° dorsiflexion)	18.4 (7.65) [a]	10.9 (9.97)
Ankle Stretch Amplitude (°)	29.9 (8.87) [a]	26.1 (6.82)
Muscle Activity		
Soleus Onset (ms)	82 (62) [a]	72 (66)
Soleus Peak (ms)	245 (69)	241 (72)
Soleus Offset (ms)	346 (67)	342 (66)
Tibialis Anterior Onset (ms)	46 (113)	38 (130)
Tibialis Anterior Peak (ms)	212 (114) [a]	201 (154)
Tibialis Anterior Offset (ms)	371 (74) [a]	347 (77)

[a] Significant difference between AT and CON means ($p<0.05$) adjusted for age, gender, height, mass and tendinopathy duration

VISA-A- Victorian Institute of Sport Assessment- Achilles

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Figure Legends

Fig 1. (a) Custom-built low-friction sledge jump system (adapted with permission from Gibson, Campbell, Allison [28]; (b) 3D motion analysis marker set configuration and; (b) lower limb stiffness derivation [14]

5. THE MODULATORY INFLUENCE OF ECCENTRIC FATIGUE ON SPRING FUNCTION

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Eccentric Fatigue Modulates Stretch-shortening Cycle Effectiveness – A Possible Role in Lower Limb Overuse Injuries

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Key word
- fatigue
- stretch shortening cycle
- eccentric contraction
- stiffness

Abstract

The role of fatigue in injury development is an important consideration for clinicians. In particular, the role of eccentric fatigue in stretch-shortening cycle (SSC) activities may be linked to lower limb overuse conditions. The purpose of this study was to explore the influence of ankle plantarflexor eccentric fatigue on SSC effectiveness during a hopping task in healthy volunteers. 11 healthy volunteers (23.2±6.7 years) performed a sub-maximal hopping task on a custom-built sledge system. 3D motion capture and surface EMG were utilised to measure lower limb stiffness, temporal kinematic measures and muscle timing measures at baseline and immediately following an eccentric fatigue protocol. A linear mixed model was used to test whether measures differed between conditions. Compared to baseline, eccentric fatigue induced increased stiffness during the hopping task (+15.3%; $P<0.001$). Furthermore, ankle stretch amplitude decreased (−9.1%; $P<0.001$), whilst all other ankle kinematic measures remained unchanged. These changes were accompanied by a temporal shift in onset of activity in soleus and tibialis anterior muscles (−4.6 to −8.5%; $p<0.001$). These findings indicate that eccentric fatigue alters SSC effectiveness in healthy volunteers. These findings may be applied to inform pathogenetic models of overuse injury development.

Introduction

Modulation of stretch-shortening cycle (SSC) effectiveness in response to loading history implies it has a role in athletic performance, injury prevention and rehabilitation [10]. For example, SSC effectiveness improves with training [26], and deteriorates in the presence of overuse injuries. Whilst SSC dysfunction has been demonstrated in several overuse conditions [20,35,38], due to its intimate relationship with the SSC, the condition that has received the most attention is Achilles tendinopathy (AT) [14,33,41]. An important physiological phenomenon that links SSC effectiveness and injury is fatigue. Further understanding the relationship between fatigue and SSC effectiveness may assist in the rehabilitation and prevention of overuse injuries such as AT.

The SSC is a phenomenon associated with human locomotion, describing the muscle function in which a pre-activated musculotendinous unit lengthens (eccentric phase) then immediately shortens (concentric phase) [42]. It simplifies and optimises the neural efficiency of terrestrial locomotion and is dependent upon co-ordinated storage/return of elastic energy and muscle activation under the control of neural strategies. The most meaningful measure of SSC effectiveness is stiffness of the lower limb [8]. Stiffness increases or decreases, regulated in accordance with task and environmental changes by the modulation of muscle activity via feedforward and feedback neural activity [16,32]. Furthermore, stiffness is modified in response to acute and chronic loading histories [39,40], as well as in accordance with pathologic conditions such as AT [14], where as a rule, increased stiffness is positive, and decreased stiffness is negative to the individual's performance.

The pathogenesis of lower limb overuse injuries such as AT are complex and multifactorial [30]. However, one important feature appears to be SSC dysfunction [14,33,41] where impairments in the spring mass system [7] result in abnormal loading of musculoskeletal tissues [10]. Whilst SSC dysfunction is likely mediated by multiple factors, fatigue appears to be a significant com-

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ponent of this process [11,19]. For instance, exhaustive running decreases lower limb stiffness [15], mediated by alterations in the neural control of the SCC. It has been suggested that this fatigue leads to increased passive tissue loading and an increased risk of injury [37].

Whilst optimal SSC efficiency requires all components to act in unison, the eccentric phase is the primary determinant of effective SSC effectiveness [12], with approximately two-thirds of extensor muscle activity occurring during the braking phase [37]. As such, a close exploration of fatigue, with a focus on the eccentric phase is warranted, which to our knowledge has yet to be done. In line with the importance of the eccentric phase of the SSC, preliminary data exists suggesting that impairments in eccentric muscle performance are an important feature of overuse injuries [1,11,34]. However, research to date has only explored the effect of fatigue on SSC modulation using SSC fatigue models. However, SSC fatigue models such as exhaustive running induce significant systemic fatigue [19], which makes divorcing the role of systemic fatigue from the neural response to fatigue not possible. Given the potential importance of eccentric fatigue in the modulation of SSC effectiveness and its potential to contribute to a number of lower limb overuse pathologies, this paper explores the modulatory effect of eccentric fatigue on temporal measures of SSC effectiveness.

SSC effectiveness can be measured in several ways: at a tissue level, at a kinematic/muscle activation level and at a systems level [9]. In our exploration, we measured the temporal changes in lower limb stiffness, and corresponding changes in associated ankle kinematic and agonist/antagonist muscle activity during a sub-maximal single-limb hopping task on a sledge-jump system. Comparisons between baseline (BAS) values and those taken immediately after an ankle plantarflexor eccentric-only fatiguing protocol (FAT) were performed.

Materials and Methods

Participants

This was an observational study, employing a within-subjects, repeated measures design. We recruited healthy university students in Perth, Western Australia (see **Table 1** for participant characteristics). Participants were excluded if they had a history of AT, lower limb surgery in the preceding 12 months, co-existing lower quadrant musculoskeletal disorder, or a significant visual or motor impairment. Participants received a full explanation of the procedures before providing written consent. Our study meets the ethical standards of this journal as described by Harriss and Atkinson [21].

Measurements

Sub-maximal hopping task

SSC effectiveness was investigated using a submaximal single-limb hopping model on a custom-built sledge-jump system (**Fig. 1a, c**). Such systems are employed to optimise reliability by reducing the degrees of freedom in the task and eliminate task fatigue as a confounding factor [18]. Furthermore, given our interest in focusing the task to the ankle, this model unloads participants, enabling this process. Following familiarisation, participants were instructed to hop on the sledge using their dominant limb, at a submaximal effort level, consciously maintaining a neutral hip and knee. Pilot testing demonstrated that this was achieved; mean joint excursion angles for the ankle, knee and hip were 28.6° (±10.2), 9.8° (±4.8) and 6.3° (±2.3) respectively, with high reliability (e.g., ICC of 0.87 of ankle stretch amplitude 0.77 for within, between and across trial measures). Participants hopped continuously for 15 s, repeated 5 times, with 45 s rest between trials. This task was performed before and immediately after (within 5 min) the fatigue intervention.

Table 1 Participant characteristics.

n = (male:female)	11 (3:8)
age	23.2 (±6.7) years
height	170.1 (±8.2) cm
mass	70.7 (±13.3) kg

Kinematic measures

Sagittal plane ankle kinematics were recorded using a 14-camera Vicon MX motion analysis system (Vicon, Oxford Metrics, Oxford, UK) operating at 250 Hz. Retro-reflective markers were fixed to participants' skin according to a customised marker set and model for the lower quadrant, according to an established cluster-based method [6]. This established set-up enabled determination of anatomically-relevant ankle, knee and hip joint axes of rotation and joint centres [6].

Electromyographic measures

Soleus and tibialis anterior muscle activity were recorded, using an AMT-8 (Bortec Biomedical Ltd) surface electromyography (sEMG) system. Bipolar differential surface electrodes (Ag/AgCL) were placed on the belly of each muscle with the reference electrode on the medial malleolus. Skin impedance (<15 kOhms) was achieved by skin preparation and signals were pre-amplified, analogue-filtered (10–500 Hz band pass) and digitised using an 18-bit A-D card utilising a sampling rate of 1 000 Hz. All data was temporally synchronised and recorded on dedicated hardware running a customised Labview program (National Instruments, Austin, Texas, 2011).

Interventions

Fatiguing protocol

Positioned in a commercial seated calf-raise machine (**Fig. 1c**) and using only their dominant limb, participants completed a warm-up of 3 sets of 10 repetitions using a 10-kg weight before a '6 Repetition Maximum Test' (6 RM; the maximum weight a participant can lift 6 times) was conducted. Participants performed 6 isotonic repetitions, beginning at 12.5 kg; on successful completion, weight was increased by 2.5 kg. All sets were separated by 60 s rest and continued until the participant could not successfully complete the task. Successful completion of the task was judged by whether or not the participant could complete the 6 repetitions through full range without employing compensatory strategies such as hip flexion or trunk movements that might facilitate hip flexor contribution. The final successful weight, which for each participant was achieved within 3 trials, was considered their 6RM (group mean 27.4 kg±6.6). Participants performed 5 sets of 10 eccentric contractions at their 6RM [5]. An assistant raised the weight so that the participant only had to perform the eccentric component. The aim of the protocol was to induce standardised eccentric fatigue in the plantarflexors. If a participant fatigued early or late, sets were added or removed at the discretion of the chief investigator. All partici-

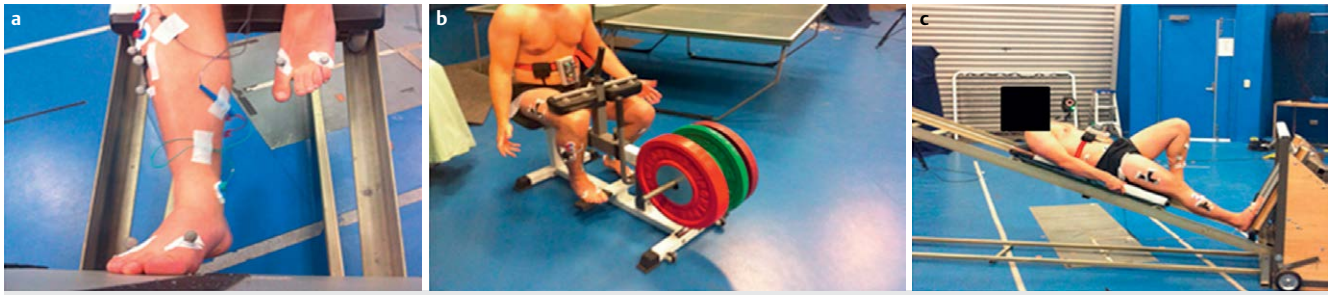

Fig. 1 Experimental set-up **a** Participant hopping on sledge-jump system, viewed from above; sEMG and 3D motion-analysis marker set in situ. **b** Participant performing a loaded single-leg calf raise. **c** Participant hopping sledge jump system – hip and knee remain in a neutral position.

pants completed 5 or 6 sets. Neither during this protocol, nor on re-testing, did participants experience pain or discomfort beyond the transient discomfort associated with muscle fatigue.

Data analysis

Kinematic and sEMG data were processed using Vicon Nexus motion analysis software (Vicon, Oxford Metrics, Oxford, UK). Kinematic data were inspected for broken trajectories that can occur as a result of marker occlusion. All breaks < 20 frames in length were filled using standard procedures (i.e., cubic spline interpolation). Data was filtered using a fourth-order Butterworth filter operating at a frequency cut-off of 20 Hz for the marker trajectories and 50 Hz for the ground contact data as determined by residual analysis [44]. Lower limb anatomical and joint coordinates were calculated in accordance with the standards outlined by the previously described International Standards of Biomechanics [6,45]. Data was exported from Nexus for further analysis using a customised LabVIEW program (National Instruments, Austin, Texas, 2011). For each trial, the following ankle kinematic measures were calculated; ankle angle 80 ms prior to ground contact, ankle angle at ground contact, peak ankle angle and ankle stretch amplitude. Lower limb stiffness was calculated using the following validated method [13]:

$$K_n = \frac{M \times \pi (T_f + T_c)}{T_c^2 \left(\frac{T_f + T_c}{\pi} \frac{T_c}{4} \right)}$$

K_n: Lower limb stiffness (Nm^{-1})
M: Mass (kg)
T_f: Flight time (s)
T_c: Contact time (s)

Temporal measures of muscle activity for the soleus and tibialis anterior were calculated relative to ground contact: onset, peak and offset. The EMG signal was full-wave rectified and onsets detected using an integrated protocol [2]. Trial linear envelopes (LE) were created using a fourth-order, zero-lag Butterworth low-pass filter (10 Hz) and temporally synchronised to (T = 0) foot contact.

Statistics

Statistical analysis was conducted using SPSS version 20 (SPSS, Chicago, Il, USA). Descriptive statistics were used to establish mean values for all variables in each group (BASE vs. FAT). A linear mixed model was used for all statistical comparisons between groups. Age, gender, height and body mass were input as covariates and adjusted for within the model. A fixed main effects model was fitted, with a type III sum of squares used to assess statistical significance. For each dependent variable parameter, estimates were utilised and main effects were compared as pairwise comparisons using a Bonferoni correction for repeated measures. The residuals were tested for normality as required by the linear mixed model with a significance level of $p < 0.05$.

Results

Following eccentric fatigue, lower limb stiffness increased +15.3 % from 5.9 (±1.3) to 6.8 (±1.7) Nm^{-1} ($p < 0.001$; 95 % CI 0.7 to 1.1) (**Fig. 2b**). Our primary kinematic variable, stretch amplitude (**Fig. 2a**) decreased −9.1 % from 25.2 (±8.9) to 23.4 (±8.5)° ($p < 0.001$; 95 % CI −1.1 to −2.4). There was no statistically significant difference in any other kinematic measure between BAS and FAT.

For all temporal sEMG measures (**Fig. 3**), relative timings occurred between 4.6–8.5 % earlier following the fatigue intervention (soleus onset: 76 (±62) to 61 (±51) ms ($p < 0.001$; 95 % CI 9 to 21); soleus peak: 242 (±69) to 222 (±62) ms ($p < 0.001$; 95 % CI 14 to 27); soleus offset: 343 (±67) to 322 (±58) ms ($p < 0.001$; 95 % CI 14 to 27); tibialis anterior onset 44 (±113) to 32 (±102) ms ($p = 0.03$; 95 % CI 1 to 23); tibialis anterior peak 207 (±114) to 193 (±111) ms ($p = 0.01$; 95 % CI 3 to 25); tibialis anterior offset 347 (±74) to 331 (±68) ms ($p < 0.001$; 95 % CI 8 to 23).

Discussion

The impact of eccentric fatigue on SSC effectiveness is potentially an important pathogenetic component for lower limb overuse conditions such as AT and to our knowledge, this is the first study that has explored how eccentric fatigue modulates SSC effectiveness. We found that eccentric fatigue results in increased lower limb stiffness and a corresponding decrease in stretch amplitude, accompanied by a hastening of muscle activity. Lower limb stiffness increased by 15.3 % (0.9 Nm^{-1}), stretch amplitude decreased by 9.1 % (1.8°) (**Fig. 2**) and all temporal measures of muscle activity for the tibialis anterior and soleus hastened by 4.6–8.5 % (12–21 ms) (**Fig. 2**).

Whilst the findings on stiffness are inconsistent with the existing literature [4,22,28], findings on muscle activity are consistent [28]. A number of possible explanations for these findings exist. Whilst the majority of other studies investigating the effect of fatigue on lower limb stiffness have observed decreases in stiffness [25,28,37], systemic fatigue was induced by the methodology employed. Our experimental model attempted to mitigate the influence of systemic fatigue and isolate changes to

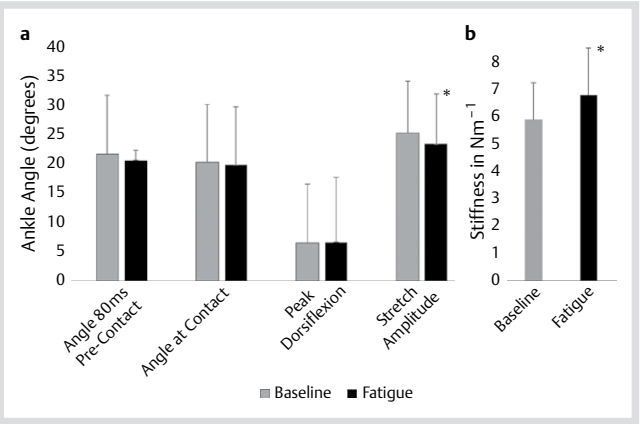

Fig. 2 Mean (±SD) for **a** kinematic and **b** lower limb stiffness values at baseline and fatigue during the hopping task; * denotes significant difference ($P<0.05$).

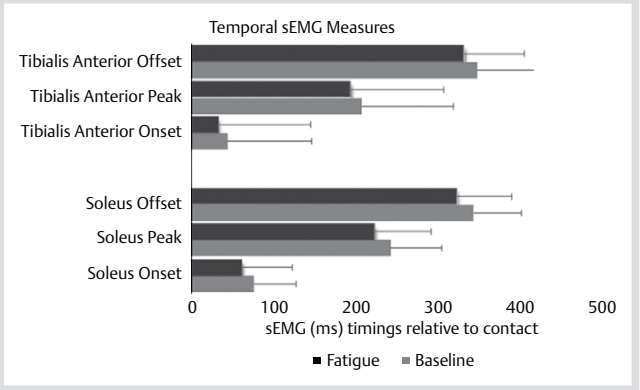

Fig. 3 Mean (±SD) sEMG values at baseline and fatigued, for tibialis anterior and soleus during the hopping task. All measures are significantly different ($P<0.05$).

the neural control of the SSC following fatigue. Fatigue appears to generate feed-forward changes in agonist and antagonist muscle activity, which drives the increase in stiffness. According to Oliver, De Ste Croix, Lloyd, et al. [37], increased stiffness may represent a potentiated state within the neuromuscular system, where in response to fatigue, neural control is modulated in a positive (i.e., protective) manner. The ability to maintain short ground contact time appears to be a key determinant to maintaining SSC performance under fatigue [22] and participants may have increased stiffness in order to achieve this.

Fatigue is a pathogenetic feature of lower limb overuse conditions (e.g., AT [36]) and may be perceived at a systems level as a threatening stimulus. Using our experimental model, it has been suggested that in the presence of an overuse injury, a threat is perceived and individuals attempt to limit exposure to that threat by increasing stiffness [14]. The findings of the current study support this theory, suggesting that fatigue may be perceived at a 'control-level' as a threatening stimulus in the same way that pain is [24].

We observed a decrease in ankle stretch amplitude, but no other changes in ankle kinematic measures. The only other study to explore ankle kinematics during an SSC following fatigue was performed by Kuitunen, Avela, Kyrolainen, et al. [27]. They measured lower limb kinematics during sub-maximal hopping on a sledge system following a hopping fatigue protocol. Once fatigued, they observed increased ankle stretch amplitude in the region of 15% relative to baseline, compared to increases at the knee and hip of 50 and 300% respectively. These findings indicate that in the presence of fatigue, individuals absorb load at the hip and knee as opposed to the ankle, which our findings also support. In our model, participants were unable to yield at the hip or knee; nevertheless, relative attempts were made to limit yielding at the ankle, as appeared to occur in the study by Kuitunen, Avela, Kyrolainen, et al. [27] showing in fact a slight reduction in stretch amplitude at the ankle. Whilst the fatiguing nature of the 2 studies differed, in combination these findings support the theory that in the presence of a threatening stimulus, a local stiffening strategy is employed to limit such exposure [24].

Limitations of our study require acknowledgement. Our fatiguing protocol isolated the plantarflexors in an eccentric manner, meaning that fatigue of other muscles (antagonist, synergists) in a manner that would induce SSC and systemic fatigue limit the generalisability of our findings. However, this model was specifically chosen in order to isolate the eccentric role of the plantarflexors in the absence of systemic fatigue. This is important given that eccentric plantarflexor activity is the primary modulator of SSC effectiveness [12,23]. Similarly, this protocol would inevitably cause exercise-induced muscle damage, which will have contributed to mechanisms responsible for the changes observed. This study is unable to distinguish between this and other-related phenomena (e.g., motor drive [43]), and future studies should attempt to divorce such mechanisms from one another. We employed a submaximal single-limb hopping task on a sledge jump system, which has been used previously to investigate a number of neural and mechanical properties of the apparatus of the SSC [18,29]. Whilst this limits external validity, it does facilitate the intimate exploration of the mechanics and control of the SSC. Finally, we employed the method described by Dalleau, Belli, Viale, et al. [13] to measure lower limb stiffness. However, participants were instructed to hop with a static hip and knee, which was mostly successful (pilot testing demonstrated mean hip and knee excursion of 6.3° (±2.3) and 9.0° (±4.4). Given mean ankle excursion was around 25°, we believe this method can be considered to provide a surrogate measure of ankle stiffness.

Our findings have a number of potential clinical implications, surrounding firstly the pathogenesis, and secondly the management of lower limb overuse conditions (e.g., AT). These are based on the theory that deficits in muscle performance underpin the fatigue damage observed in such conditions [36], and fatigue resilience is a critical requirement in the rehabilitation of such conditions [31]. In the context of endurance sport (e.g., running) if reduced muscle performance is considered a risk factor for the development of 'AT', when combined with excessive activity (i.e., running), the musculotendinous unit eventually loses its resilience to repeated impact. Given that muscles act as 'buffers to passive tissues, (joint, tendon and bone) (add buffer ref), fatigue of the muscle will result in increased load being placed on the passive tissues, leading to tissue breakdown once the individual load tolerance of that tissue has been exceeded. In the same light, given the association between such pathologic conditions and associated motor dysfunction [14], a clinical paradigm exists where 'normalising' such motor performance deficits is an integral component of rehabilitation [31]. Our study provides affirming data, albeit in experimental terms, that a muscle's capacity to tolerate fatigue is a critical component of the pathogenesis and rehabilitation of lower limb overuse condi-

tions. We observed increased lower limb stiffness and reduced stretch amplitude, which is in contrast to many previous studies [15,17,22]. This most likely reflects a novel phenomenon that in the absence of metabolic fatigue, it is observed that motor control strategies exist where the lower limb responds to fatigue by increasing stiffness. Given that fatigue loading reduces tendon stiffness [3], motor performance may be the most important mediator of ankle mechanics in the presence of fatigue (i.e., fatigue resilience). If tendon fatigue was the dominant feature we would have expected an increase in stretch amplitude and a decrease in lower limb stiffness indicative of tendon yielding. During conditioning and rehabilitation, consideration should be given to ensuring an adequate motor performance profile of the plantarflexors to protect against fatigue damage and the predisposition to developing an overuse injury.

To our knowledge, fatigue as a perceived threatening stimulus in the same light as pain has yet to be considered. Our findings allow us to make the suggestion that this may be the case. In the presence of experimental and clinical pain, changes at multiple levels within the neuromusculoskeletal system produce a redistribution of activity within and between muscles, utilising a most commonly observed stiffening strategy, which is believed to be an attempt to protect the painful region [24]. Pain is a perceptual experience elicited in response to actual or threat of tissue damage. If fatigue is contextualised as a threatening stimulus, which is appropriate given that it elevates the risk of injury [46], and it is acknowledged that to experience pain in the presence of fatigue would be inappropriate from an evolutionary perspective, it would make sense for the neuromusculoskeletal system to elicit a protective motor response to such a threat, as has been demonstrated in this study.

In summary, we have shown that eccentric fatigue of the ankle plantarflexors alters SSC effectiveness. Specifically, lower limb stiffness increases mediated by an increase in muscle activity, whilst ankle stretch amplitude decreases. These changes occur in a manner that appears to be independent of systemic responses and that recognises that fatigue may pose a potential injury threat to the musculoskeletal system.

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6. THE MODULATORY INFLUENCE OF ECCENTRIC LOADING ON SPRING FUNCTION

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Article Title: Eccentric Loading of Triceps Surae Modulates Stretch Shortening Cycle Behaviour- A Possible Therapeutic Mechanism

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Title Page

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Eccentric loading of triceps surae modulates stretch shortening cycle behaviour- A possible therapeutic mechanism

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Abstract

Context: Eccentric exercises are increasingly being used to treat lower limb musculoskeletal conditions such as Achilles tendinopathy. Despite widespread clinical application and documented efficacy, mechanisms underpinning clinical benefit remain unclear. Positive adaptations in motor performance are one potential mechanism. **Objective:** To investigate how an eccentric loading intervention influences measures of stretch-shortening cycle (SSC) behaviour during a hopping task. **Design:** Within subjects repeated measures observational study. **Setting:** University motion analysis laboratory. **Participants:** Healthy adults. **Interventions:** A single intervention of 5 sets of 10 eccentric plantarflexion contractions at 6 RM using a commercial seated calf raise machine. **Main outcome measures:** Lower limb stiffness, sagittal plane ankle kinematics, and temporal muscle activity of the agonist (soleus) and antagonist (tibialis anterior) muscles, measured during sub-maximal hopping on a custom-built sledge-jump system. **Results:** Eccentric loading altered ankle kinematics during sub-maximal hopping; peak ankle angle shifted to a less dorsiflexed position by 2.9° and ankle angle pre-contact shifted by 4.4° ($p<0.001$). Lower limb stiffness increased from 5.9 to 6.8 Nm^{-1} ($p<0.001$), whilst surface EMG measures of soleus occurred 14 to 44% earlier ($p<0.001$) following the loading intervention. **Conclusions:** These findings suggest that eccentric loading alters SSC behaviour in a manner reflective of improved motor performance. Decreased ankle excursion, increased lower limb stiffness and alterations in motor control may represent a positive adaptive response to eccentric loading. These findings support the theory that mechanisms underpinning eccentric loading for tendinopathy may in part be due to improved 'buffering' of the tendon by the neuromuscular system.

Keywords: Achilles tendon, Eccentric exercise, Lower limb stiffness, Motor control, Rehabilitation

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Main Text

Introduction

Eccentric contractions are defined as muscle activity that occurs when the force applied to the muscle exceeds the momentary force produced by the muscle itself [1] and are used to decelerate, brake or absorb energy [2]. The intentional elicitation of eccentric contractions under load is referred to as eccentric loading and has for many years been used in both training and rehabilitative settings [3]. One common application of eccentric loading is in the management of Achilles tendinopathy (AT). First reported on in the early 1990's [4] and made popular by Alfredson, et al. [5], eccentric loading involves the patient performing progressive, loaded, eccentric contractions of the ankle plantarflexors. Whilst efficacy for eccentric loading for AT has been demonstrated [6], the mechanisms underpinning its effect are not fully understood [7]. Proposed mechanisms include structural tendon adaptation, tendon length changes, neurovascular ingrowth, neurochemical alterations, fluid movement, and neuromuscular changes (see [8] for a recent review).

The stretch-shortening cycle (SSC) describes the pre-activation, lengthening and shortening of a musculotendinous unit during ground contact [9] and is a common feature of terrestrial locomotion [10]. It exists to simplify the motor control of locomotion and enhance its efficacy through the utilisation of muscle pre-activation, storage of elastic energy, and utilisation of the spinal stretch reflex [11]. SSC behaviour can be regulated according to changes in task [12] and environment [13], and can be modulated in response to training [14] or pathologic conditions such as AT [15]. Recently, the role of the SSC in the development and management of AT has received increased attention [15, 16], based on the premise that whilst AT has a multifactorial pathogenesis [17], deficits in motor performance are considered a major pathogenic component [18]. In terms of regulating SSC behaviour, the most important phase is the eccentric phase [11]; whilst it is well-known that eccentric loading improves motor performance [19], we have limited understanding on how SSC behaviour can be modulated by eccentric loading of the plantarflexors [20].

The purpose of this study was to investigate how an eccentric loading intervention of the plantarflexors affects SSC behaviour. To do this, we measured several correlates of SSC-behaviour (lower limb stiffness, ankle kinematics and agonist/antagonist muscle activity) during a controlled SSC task (sub-maximal hopping on a sledge jump system) before and 7 days after an eccentric loading protocol. We hypothesised that following the intervention lower limb stiffness would increase driven by alterations of muscle activity. Furthermore, we hypothesised that ankle kinematics would demonstrate a shift towards a more plantarflexed position. A clearer understanding of how eccentric loading effects SSC behaviour may assist in explaining the mechanisms underpinning its efficacy and inform their clinical applications in conditions such as AT. For instance, it can be used to inform clinical decision making about when and why eccentric loading can be incorporated into patient management, providing the potential to improve clinical outcomes in this challenging population.

Methods

This study drew upon a previously described protocol investigating the effect of eccentric fatigue on SSC behaviour [21].

Design

This was a within subjects repeated measures observational study. The independent variable was eccentric loading status (Baseline vs. Eccentric) and the dependent variables were temporal measures of lower limb stiffness (k), ankle kinematics (angle 80 ms prior to foot contact, ankle angle at contact, peak ankle angle, and stretch amplitude) and surface electromyographic (sEMG) activity (onset, peak, and offset timings for soleus and tibialis anterior).

Participants

We recruited 11 healthy volunteers (5 males and 6 females; mean age 23.2 ± 6.7years) and excluded those with a history of AT, lower limb surgery in the preceding 12 months, a co-existing

lower quadrant musculoskeletal disorder, or a significant visual or motor impairment. All participants provided written informed consent, with procedures being approved by the local university human research ethics committee.

Procedures

Sub-Maximal Hopping Task

Participants performed continuous sub-maximal hopping on a custom-built sledge jump system. Such systems have demonstrated validity and reliability [22] and the details of the protocol employed in this study have been described in detail elsewhere [15]. The advantage of hopping on such a system as this is that the SSC can be isolated to the ankle and the confounding effects of fatigue can be eliminated due to the low load nature of the task. Following a warm-up, participants hopped continuously at a sub-maximal level (one that could be sustained 'indefinitely') for 15 seconds. Following a 45 second rest period, this was repeated, for a total of 5 trials (see fig 1). Five trials were chosen as pilot testing had established that exclusion of the first trial, as well as the initial hop for any given trial results in stable measures within-trial, between multiple trials, and across multiple testing occasions. With this protocol we were able to analyse the full series of trials and given that participants hopped at 1.3 Hz, provided approximately 20 hops per trial.

Kinematics

Sagittal plane ankle kinematics were recorded using a 14-camera Vicon MX motion analysis system (Vicon, Oxford Metrics, Oxford, UK) operating at 250 Hz. Retro-reflective markers were fixed to participants' skin according to a customised marker set and model for the lower quadrant which established anatomically-relevant ankle, knee and hip joint axes of rotation and joint centres [23]. Ankle angles at specific time-points relative to initial ground contact (80 ms pre-contact and contact) were captured along with peak dorsiflexion angle. Ankle dorsiflexion stretch amplitude was calculated as the difference between the ankle angle at contact, and peak dorsiflexion angle.

Electromyographic Measures

Soleus and tibialis anterior muscle activities were recorded, using an AMT-8 (Bortec Biomedical Ltd) surface electromyography (sEMG) system. Bipolar differential surface electrodes (Ag / AgCl) were placed on the belly of each muscle with the reference electrode on the medial malleolus. Skin impedance (< 15kOhms) was achieved by skin preparation and signals were pre-amplified, analogue filtered (10 – 500Hz band pass) and digitised at sampling rate of 1000Hz. All data was synchronised on dedicated hardware running a customised program (Labview, National Instruments, Austin, Texas, 2011).

Intervention- Eccentric Loading Protocol

The employed eccentric loading protocol has previously been comprehensibly described elsewhere [21], which was applied on a single occasion using the dominant limb. Using a commercial seated calf raise machine participants warmed-up (3 x 10 repetitions at 4/10 RPE) and completed a 6 RM test upon which the eccentric loading dose was based. The 6 RM was always achieved within 3 attempts (group mean 6 RM 27.4 kg ± 6.6). Following a 10 minute break, participants performed a single eccentric loading protocol consisting of 5 sets of 10 eccentric plantarflexion repetitions at their 6 RM, interspersed by 60 second rest periods.

Protocol

Participants attended for baseline testing and the eccentric loading intervention on day 1. Participants returned for post-intervention testing 7 days later with testing performed at a consistent time of day on both occasions. This model was chosen to best isolate neuromuscular changes rather than those that may occur at days 1-3 with delayed onset muscle soreness (DOMS) following a novel exercise or tendon changes that may occur with multiple training occasions [24]. All participants were instructed to continue with their normal daily activities over the intervening week, and specifically instructed not to undertake any change in physical activities. Likewise,

participants were asked if any experienced DOMS was still evident at day 7 testing and none reported so.

Statistical Analyses

Kinematic and sEMG data were processed in accordance with international standards [23]. For each trial, the following ankle kinematic measures were calculated; ankle angle 80 ms prior to ground contact, ankle angle at ground contact, peak ankle angle and ankle stretch amplitude and with the exclusion of the first trial, mean values of the remaining 4 trials were analysed. Lower limb stiffness was calculated using the following method [25]:

$$K_n = \frac{\mathrm{M} \times \pi(T_f + T_c)}{T_c^2 \left(\frac{T_f + T_c}{\pi} \frac{T_c}{4}\right)}$$

K_n = lower limb stiffness (Nm^{-1}), M = Mass (kg), T_f = Flight time (s) and T_c = Contact time (s)

Temporal measures of muscle activity for soleus and tibialis anterior were calculated relative to ground contact; onset, peak and offset, using the protocol of Allison [26] and described by Debenham, et al. [15].

Statistical analysis was conducted using SPSS version 20 (SPSS, Chicago, Il, USA). Descriptive statistics were used to establish mean values for all variables in each group (Baseline vs. Eccentric). A linear mixed model was used for all statistical comparisons between groups. Age, gender height and body mass were input as covariates and adjusted for within the model. A fixed main effects model was fitted, with a type III sum of squares used to assess statistical significance. For each dependent variable parameter estimates were utilised, and main effects were compared as pairwise comparisons using a Bonferoni correction for repeated measures. The residuals were tested for normality as required by the linear mixed model with a set significance level of $p<0.05$. Based on a previous study employing these methods [15] a minimum sample size of 10 participants was determined sufficient to test the null hypothesis with a type I error of five percent, with power set at 80%.

Results

Descriptive statistics are presented in Table 1. Following the eccentric loading protocol, lower limb stiffness significantly increased by 0.9 Nm^{-1} ($p<0.001$; 95% CI 0.7 to 1.1) (Fig 2b). Ankle angle 80ms pre-contact significantly decreased by 4.4° ($p<0.001$; 95% CI 3.1 to 5.8°) although ankle angle at contact did not change significantly. There was a shift in peak ankle angle towards a less dorsiflexed position (Fig 2a); this angle significantly decreased by 2.9° ($p<0.001$; 95% CI 1.5 to 4.3°) and ankle stretch amplitude significantly increased by 1.0° ($p=0.001$; 95% CI 0.3 to 1.7°).

sEMG measures of soleus occurred 44.7%, 17.4% and 14.6% earlier for onset, peak and offset value respectively (soleus onset: $p<0.001$; 95% CI 28 to 40; soleus peak: $p<0.001$; 95% CI 36 to 49; soleus offset: $p<0.001$; 95% CI 14 to 27 (Fig 3)). Tibialis anterior onset was delayed by 13.4% ($p=0.03$; 95% CI 1 to 23); tibialis anterior peak was not significantly different ($p=1.00$; 95% CI -13 to 8) and tibialis anterior offset was earlier ($p<0.001$; 95% CI 28 to 43)) (Fig 3).

Discussion

Whilst the efficacy of eccentric loading for AT has been demonstrated [6] and theories regarding its efficacy have been explored [27], this is the first study to determine in which way SSC behaviour is modulated by eccentric loading. Our findings demonstrate that following eccentric loading, peak ankle angle shifts to less dorsiflexion (6.5 to 3.6º dorsiflexion) and lower limb stiffness increases (5.9 to 6.8 Nm^{-1}), whilst agonist/antagonist muscle activity moves to a more agonist-dominant pattern (e.g. soleus onset 34 ms earlier vs. tibialis onset 6 ms later) (see Figs 2 and 3).

Consistent with our hypothesis, we found that peak ankle angle shifted towards a position of relative plantarflexion. Whilst modest, these findings likely represent a clinically meaningful change when compared with the 7.7° and 3.2° changes in peak torque angle that have been previously reported at the hamstrings [28]and plantarflexors [29] respectively following eccentric

loading. Previous research has found that eccentric loading leads to increases in muscle strength and power [27] and an improved ability to produce force in the descending limb of the muscle fascicle length-tension curve [30]; our findings are consistent with these phenomena. Furthermore, our findings may reflect an increase in force producing capacity of the muscle similar to those seen by Masood, et al. [31] as increases in strength imply the plantarflexors have an increased capacity to resist excursion into dorsiflexion. Improved muscle performance also protects the tendon by conferring increased stiffness upon the tendon, thereby improving its capacity to resist strain [32]. As such, these increases ensure tendon strain is not excessive, as is believed to be pathogenetic with AT [33].

Also consistent with our hypothesis, we observed a 15% increase in lower limb stiffness following the eccentric loading intervention from 5.9 to 6.8 Nm^{-1} which was achieved by decreasing contact time and increasing flight time. Our findings are consistent with Elmer, et al. [20] who in their study of healthy volunteers observed a 10% increase in lower limb stiffness following an eccentric loading task. Increases in stiffness are generally associated with improvements in both performance in healthy individuals, and clinical improvements in patients with a pathologic condition [34]. Our findings therefore likely reflect a true positive change in spring behaviour associated with the aforementioned alterations in muscular function following the intervention. Furthermore, given the 1 week timeframe involved, it is most likely that changes are neural rather structural in origin. Whilst we did not measure sEMG amplitude, the alterations in timing imply an integration of motor activity, which in turn results in increased stiffness [35]. Whilst it may be speculated that the increase in stiffness may be in part due to the impact that eccentric loading has on the connective tissues, the observed changes in SSC behaviour following a single loading event are unlikely to have occurred due to changes in in tendon material or mechanical properties [36].

Consistent with our hypothesis, following the eccentric loading intervention, temporal muscle events occurred earlier in 4 out of our 6 measures and this is the first time temporal changes

in sEMG have been demonstrated following an eccentric loading intervention. These findings most likely reflect training-induced changes in neural activation as described by Markovic, et al. [35]. Such changes include increased neural drive to the agonist muscle, as we observed with the hastening of soleus activity at all-time points. Likewise, our observed changes toward a more agonist dominant pattern of SSC performance may reflect changes in activation strategies (i.e. improved intermuscular co-ordination) [35]. The only other study exploring plantarflexor sEMG following eccentric loading is that of Masood, et al. [31]. Their findings are consistent with our own; they observed an increase in plantarflexor sEMG amplitude following an eccentric loading intervention. Given that Cadore, et al. [37] observed no increase in sEMG amplitude following eccentric loading, it may be that eccentric loading induces changes in muscle timing rather than amplitude. This theory is supported by Masood, et al. [31] who suggest that eccentric loading-induced changes in amplitude ultimately normalises muscle activity (i.e. return dysfunctional motor performance to 'normal').

Limitations to our study must be acknowledged. Our study was conducted on healthy volunteers; whilst this limits the generalisability of our findings to the target clinical population (AT), our findings form the basis upon which comparisons can be made with equivalent studies on the clinical population [31]. Although conventional sEMG is widely used to measure the electrical activity of skeletal muscle during activity, the information it provides may not reflect the activity of the whole muscle [38]. Given that intramuscular variations in muscle activity exist, findings such as ours may not reflect the true change in muscle behaviour [39]. Whilst errors in kinematic measures may also occur due to soft tissue artefact of skin-attached markers, high within-subject reliability implies our methods accommodate this. Likewise, whilst the one week might be expected to result in natural variation in measures, pilot testing demonstrated high reliability (e.g. ICC of 0.77 for ankle stretch amplitude). Finally, whilst our study is designed to inform mechanisms underpinning a clinical intervention, the employed loading protocol is not reflective of standard clinical practice[5].

This design however was purposeful, reflecting our interest in isolating our observations to changes in motor behaviour in response to loading rather than inducing chronic changes in tendon structure. Finally, our study did not measure the activity profile of gastrocnemius. Whilst this data would add value it was chosen to measure soleus in isolation due to its primary role in low level SSC-activities such as sub-maximal hopping and running [40].

The eccentric phase of the SSC appears to be its critical phase [11]. Likewise, it is known that eccentric loading improves eccentric, but not concentric muscle performance [37]. Our study has demonstrated that eccentric loading induces what appear to be positive changes in SSC behaviour. Varied opinions exist regarding the mechanisms that underpin eccentric loading for AT [7] with theories directed towards resolution of pathologic tendon structure and function [41] or improvements in motor performance [42]. Our findings provide support to the theory that changes in motor performance underpin such benefits. We also suggest that improvements in tendon structure that may occur in response to eccentric loading are secondary to improvements in motor performance. Our findings of reduced excursion into dorsiflexion and increased stiffness may represent the 'buffering' capacity of the muscle to protect the tendon as proposed by Lindstedt, et al. [1]. Furthermore, AT is associated with excessive loading into dorsiflexion [15]. Regardless of whether this kinematic phenomenon is a cause or consequence of tendinopathy it represents a maladaptive biomechanical state; excessive dorsiflexion during the SSC is sub-optimal for the muscles length-tension properties and under such circumstances excessive load is placed upon the passive structures. Our findings of reduced dorsiflexion indicate that this phenomenon reduces following eccentric loading and may play an important role in protecting the tendon from excessive tensile loading. Admittedly, given these methods it is difficult to ascertain whether these findings are unique to eccentric loading, or may be observed with combined eccentric-concentric loading. The theory that clinical benefits are conferred by improvements in motor performance (i.e. strength) is gaining traction, with Beyer, et al. [43] observing superior outcomes with resistance

loading (combined heavy eccentric and concentric contractions) rather than by eccentric loading. Replication of this study with a combined group is warranted; the remaining mechanism of efficacy being related to the changes in peak torque angle that occur following eccentric, but not concentric loading [29].

Finally, whilst our study has no direct link to the pathogenesis of AT, it is relevant to speculate that our findings support those of McCrory, et al. [44] who stated that reduced plantarflexor performance acts as a factor to increase the prevalence of AT. Combining our findings with those of Masood, et al. [45] observing improvements in symptoms and motor performance following eccentric loading in patients with AT, we suggest that impaired plantarflexor performance may be a significant pathogenic component of AT.

Conclusions

In summary, we have shown that in healthy adults, eccentric loading results in positive changes in ankle kinematics, lower limb stiffness, and agonist/antagonist muscle activity. These findings may reflect an increase in force producing capacity which leads to decreased loading in end range dorsiflexion, and possibly protecting the tendon from excessive load and providing an opportunity to heal.

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Conflict of Interest

The authors of this study have no professional relationships with companies or manufacturers who will benefit from the results of the present study.

The results of the present study do not constitute endorsement by ACSM

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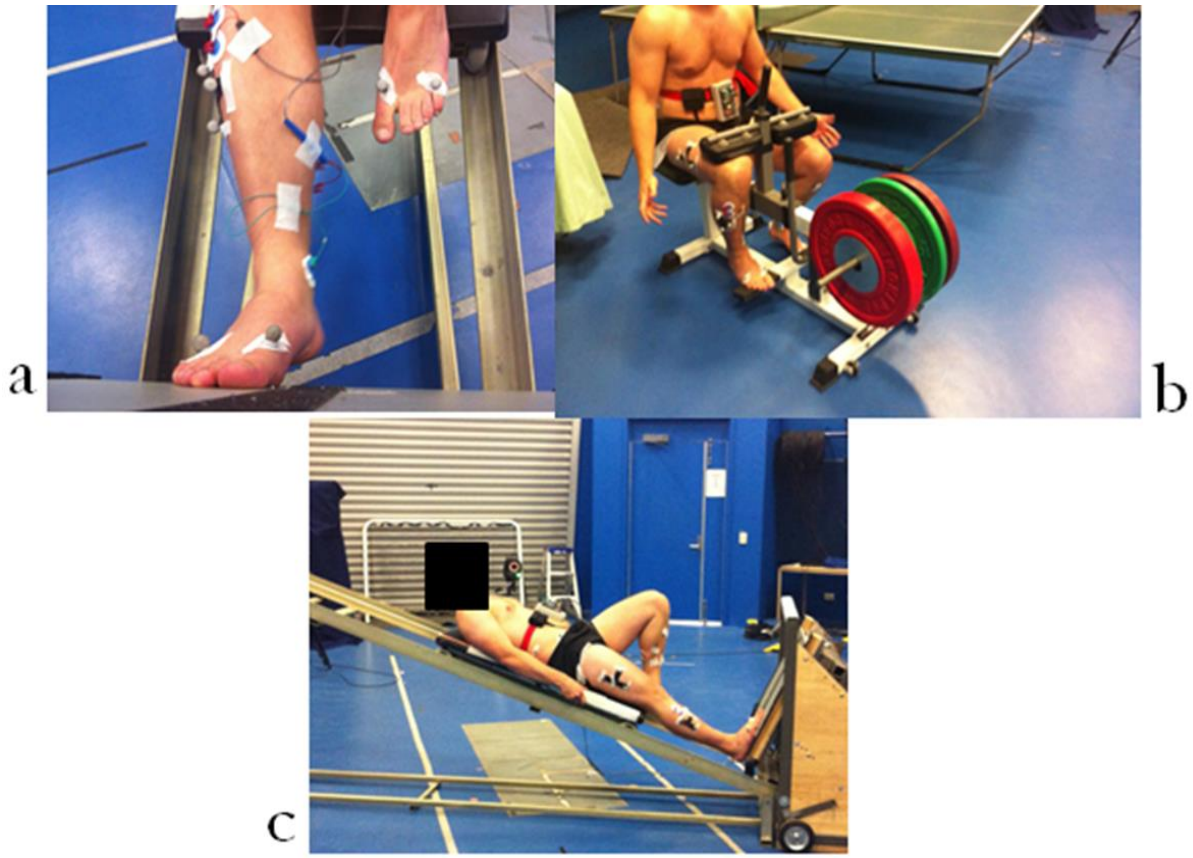

Figure 1) Experimental set-up **a)** participant hopping on sledge-jump system, viewed from above; sEMG and 3D motion-analysis marker set in situ; **b)** participant performing a loaded single-leg calf raise; **c)** participant hopping sledge jump system- hip and knee remain in a neutral position (used by permission[20]).

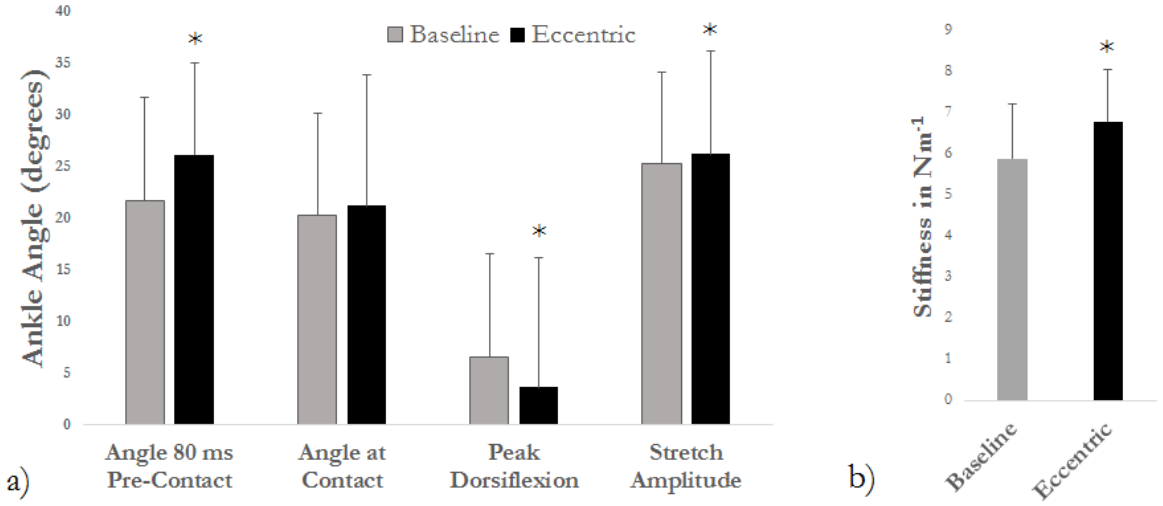

Figure 2- a) Ankle kinematics and **b)** lower limb stiffness at baseline and following the eccentric loading protocol.

* denotes significant difference ($p<0.05$)

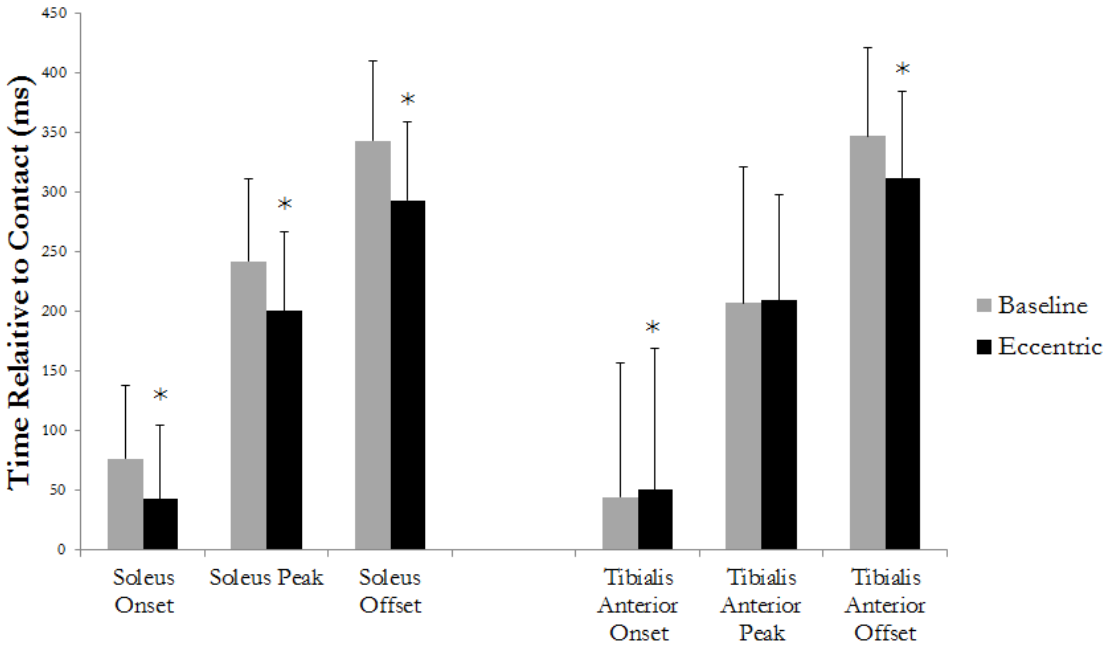

Fig 3- Soleus and tibialis anterior muscle onset timings at baseline and following the eccentric loading protocol.

* denotes significant difference ($p<0.05$)

Table 1 – Descriptive Statistics (Mean ±SD)

	Baseline	**Post-Loading (7 days)**
Hopping frequency (Hz)	1.3 (.16)	1.3 (.24)
Flight time (ms)	341 (73)	371 (101)*
Contact time (ms)	455 (60)	416 (61)*
Stiffness (Nm^{-1})	5.9 (1.34)	6.8 (1.26)*
Ankle angle 80ms pre-contact (ºDF)	-27.1 (10.1)	-26.1 (8.9)*
Ankle angle at contact (ºDF)	-20.2 (10.0)	-21.2 (12.7)
Peak ankle angle (ºDF)	6.5 (10.0)	3.6 (12.5)*
Ankle stretch amplitude (º)	25.2 (8.9)	26.2 (10.0)*
Soleus onset (ms)	76 (62)	42 (62)*
Soleus peak (ms)	242 (69)	200 (67)*
Soleus offset (ms)	343 (67)	293 (66)*
Tibialis anterior onset (ms)	44 (113)	50 (119)
Tibialis anterior peak (ms)	207 (114)	209 (89)
Tibialis anterior offset (ms)	347 (74)	312 (73)*

DF dorsiflexion
* significant difference ($p<.05$)

INTRODUCTION

Eccentric contractions are defined as muscle activity that occurs when the force applied to the muscle exceeds the momentary force produced by the muscle itself (Lindstedt et al., 2002) and are used to decelerate, brake or absorb energy (Vogt and Hoppeler, 2014). The intentional elicitation of eccentric contractions under load is referred to as eccentric loading and has for many years been used in both training and rehabilitative settings (LaStayo et al., 2014). One common application of eccentric loading is in the management of Achilles tendinopathy (AT). First reported on in the early 1990's (Niesen-Vertommen et al., 1992) and made popular by Alfredson et al. (1998), eccentric loading involves the patient performing progressive, loaded, eccentric contractions of the ankle plantarflexors. Whilst efficacy for eccentric loading for AT has been demonstrated (Magnussen et al., 2009), the mechanisms underpinning its effect are not fully understood (Allison and Purdam, 2009). Proposed mechanisms include structural tendon adaptation, tendon length changes, neurovascular ingrowth, neurochemical alterations, fluid movement, and neuromuscular changes (see (O'Neill et al., 2015) for a recent review).

The stretch-shortening cycle (SSC) describes the pre-activation, lengthening and shortening of a musculotendinous unit during ground contact (Nicol et al., 2006) and is a common feature of terrestrial locomotion (Shen and Seipel, 2015). It exists to simplify the motor control of locomotion and enhance its efficacy through the utilisation of muscle pre-activation, storage of elastic energy, and utilisation of the spinal stretch reflex (Cormie et al., 2010). SSC behaviour can be regulated according to changes in task (Farley and González, 1996) and environment (Marquez et al., 2014), and can be modulated in response to training (Vaczi et al., 2014) or pathologic conditions such as AT (Debenham et al., 2016c). Recently, the role of the SSC in the development and management of AT has received increased attention (Debenham et al., 2016c, Maquirriain and Kokalj, 2014), based on the premise that whilst AT has a multifactorial pathogenesis (Magnan et al., 2014), deficits in motor performance are considered a major pathogenic component (Munteanu and Barton, 2011). In terms of regulating SSC behaviour, the most important phase is the eccentric phase (Cormie et al., 2010); whilst it is well-known that eccentric loading improves motor performance (Lastayo et al., 1999), we have limited understanding on how SSC behaviour can be modulated by eccentric loading of the plantarflexors (Elmer et al., 2012).

The purpose of this study was to investigate how an eccentric loading intervention of the plantarflexors affects SSC behaviour. To do this, we measured several correlates of SSC-behaviour (lower limb stiffness, ankle kinematics and agonist/antagonist muscle activity) during a controlled SSC task (sub-maximal hopping on a sledge jump system) before and 7 days after an eccentric loading protocol. We hypothesised that following the intervention lower limb stiffness would increase driven by alterations of muscle activity. Furthermore, we hypothesised that ankle kinematics would demonstrate a shift towards a more plantarflexed position. A clearer understanding of how eccentric loading effects SSC behaviour may assist in explaining the mechanisms underpinning its efficacy and inform their clinical applications in conditions such as AT. For instance, it can be used to inform clinical decision making about when and why eccentric loading can be incorporated into patient management, providing the potential to improve clinical outcomes in this challenging population.

METHODS

This study drew upon a previously described protocol investigating the effect of eccentric fatigue on SSC behaviour (Wellisch et al., 2015).

DESIGN

This was a within subjects repeated measures observational study. The independent variable was eccentric loading status (Baseline vs. Eccentric) and the dependent variables were temporal measures of lower limb stiffness (k), ankle kinematics (angle 80 ms prior to foot contact, ankle angle at contact, peak ankle angle, and stretch amplitude) and surface electromyographic (sEMG) activity (onset, peak, and offset timings for soleus and tibialis anterior).

PARTICIPANTS

We recruited 11 healthy volunteers (5 males and 6 females; mean age 23.2 ± 6.7years) and excluded those with a history of AT, lower limb surgery in the preceding 12 months, a co-existing lower quadrant musculoskeletal disorder, or a significant visual or motor impairment. All participants provided written informed consent, with procedures being approved by the local university human research ethics committee.

PROCEDURES

SUB-MAXIMAL HOPPING TASK

Participants performed continuous sub-maximal hopping on a custom-built sledge jump system. Such systems have demonstrated validity and reliability (Furlong and Harrison, 2013) and the details of the protocol employed in this study have been described in detail elsewhere (Debenham et al., 2016c). The advantage of hopping on such a system as this is that the SSC can be isolated to the ankle and the confounding effects of fatigue can be eliminated due to the low load nature of the task. Following a warm-up, participants hopped continuously at a sub-maximal level (one that could be sustained 'indefinitely') for 15 seconds. Following a 45 second rest period, this was repeated, for a total of 5 trials (see Figure 1). Five trials were chosen as pilot testing had established that exclusion of the first trial, as well as the initial hop for any given trial results in stable measures within-trial, between multiple trials, and across multiple testing occasions. With this protocol we were able to analyse the full series of trials and given that participants hopped at 1.3 Hz, provided approximately 20 hops per trial.

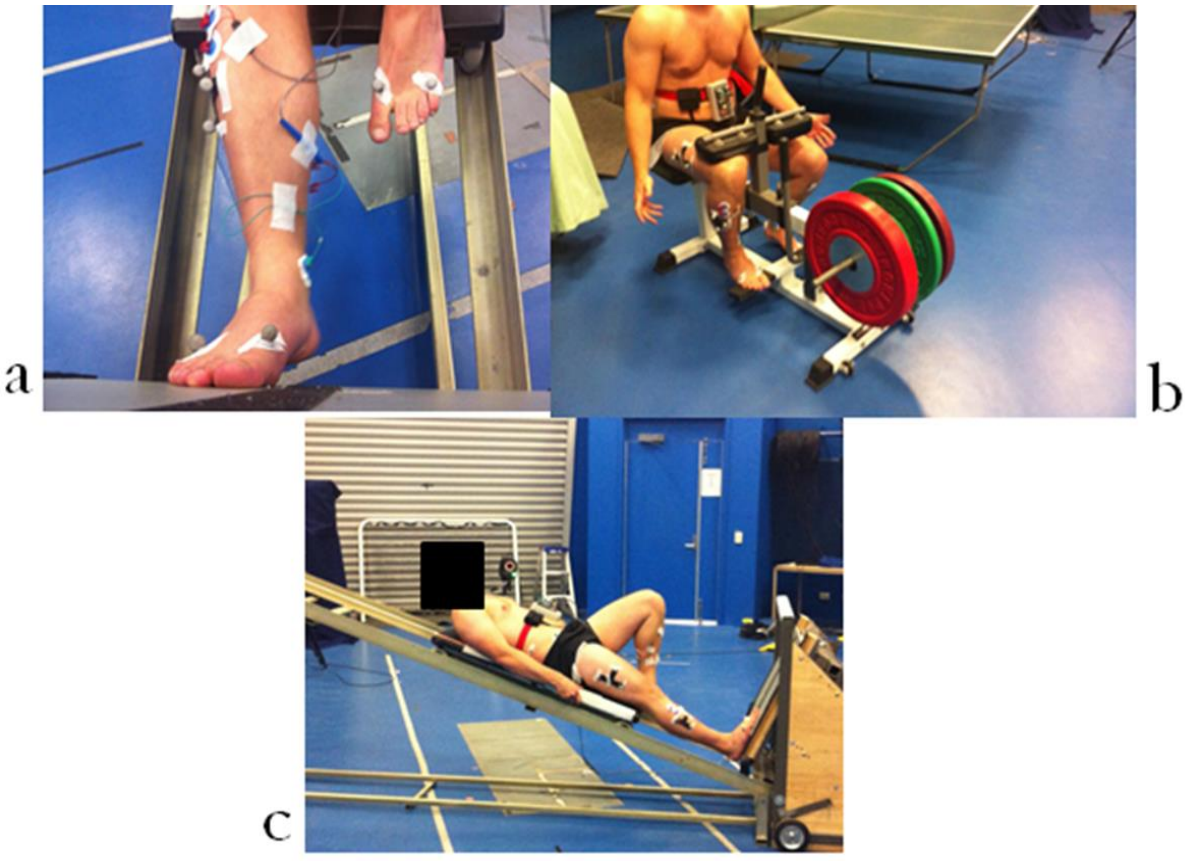

Figure 1: Experimental set-up **a)** participant hopping on sledge-jump system, viewed from above; sEMG and 3D motion-analysis marker set in situ; **b)** participant performing a loaded single-leg calf raise; **c)** participant hopping sledge jump system- hip and knee remain in a neutral position (used by permission (Wellisch et al., 2015)).

KINEMATICS

Sagittal plane ankle kinematics were recorded using a 14-camera Vicon MX motion analysis system (Vicon, Oxford Metrics, Oxford, UK) operating at 250 Hz. Retro-reflective markers were fixed to participants' skin according to a customised marker set and model for the lower quadrant which established anatomically-relevant ankle, knee and hip joint axes of rotation and joint centres (Besier et al., 2003). Ankle angles at specific time-points relative to initial ground contact (80 ms pre-contact and contact) were captured along with peak dorsiflexion angle. Ankle dorsiflexion stretch amplitude was calculated as the difference between the ankle angle at contact, and peak dorsiflexion angle.

ELECTROMYOGRAPHIC MEASURES

Soleus and tibialis anterior muscle activities were recorded, using an AMT-8 (Bortec Biomedical Ltd) surface electromyography (sEMG) system. Bipolar differential surface electrodes (Ag / AgCl) were placed on the belly of each muscle with the reference electrode on the medial malleolus. Skin impedance (< 15kOhms) was achieved by skin preparation and signals were pre-amplified, analogue filtered (10 – 500Hz band pass) and digitised at sampling rate of 1000Hz. All data was synchronised on dedicated hardware running a customised program (Labview, National Instruments, Austin, Texas, 2011).

INTERVENTION- ECCENTRIC LOADING PROTOCOL

The employed eccentric loading protocol has previously been comprehensibly described elsewhere (Wellisch et al., 2015), which was applied on a single occasion using the dominant limb. Using a commercial seated calf raise machine participants warmed-up (3 x 10 repetitions at 4/10 RPE) and completed a 6 RM test upon which the eccentric loading dose was based. The 6 RM was always achieved within 3 attempts (group mean 6 RM 27.4 kg ± 6.6). Following a 10 minute break, participants performed a single eccentric loading protocol consisting of 5 sets of 10 eccentric plantarflexion repetitions at their 6 RM, interspersed by 60 second rest periods.

PROTOCOL

Participants attended for baseline testing and the eccentric loading intervention on day 1. Participants returned for post-intervention testing 7 days later with testing performed at a consistent time of day on both occasions. This model was chosen to best isolate

neuromuscular changes rather than those that may occur at days 1-3 with delayed onset muscle soreness (DOMS) following a novel exercise or tendon changes that may occur with multiple training occasions (Mahieu et al., 2008). All participants were instructed to continue with their normal daily activities over the intervening week, and specifically instructed not to undertake any change in physical activities. Likewise, participants were asked if any experienced DOMS was still evident at day 7 testing and none reported so.

STATISTICAL ANALYSES

Kinematic and sEMG data were processed in accordance with international standards (Besier et al., 2003). For each trial, the following ankle kinematic measures were calculated; ankle angle 80 ms prior to ground contact, ankle angle at ground contact, peak ankle angle and ankle stretch amplitude and with the exclusion of the first trial, mean values of the remaining 4 trials were analysed. Lower limb stiffness was calculated using the following method (Dalleau et al., 2004):

$$K_n = \frac{\mathrm{M} \times \pi(T_f + T_c)}{T_c^2 \left(\frac{T_f + T_c}{\pi} \frac{T_c}{4}\right)}$$

K_n = lower limb stiffness (Nm^{-1}), M = Mass (kg), T_f = Flight time (s) and T_c = Contact time (s)

Temporal measures of muscle activity for soleus and tibialis anterior were calculated relative to ground contact; onset, peak and offset, using the protocol of Allison (2003) and described by Debenham et al. (2016c).

Statistical analysis was conducted using SPSS version 20 (SPSS, Chicago, Il, USA). Descriptive statistics were used to establish mean values for all variables in each group (Baseline vs. Eccentric). A linear mixed model was used for all statistical comparisons between groups. Age, gender height and body mass were input as covariates and adjusted for within the model. A fixed main effects model was fitted, with a type III sum of squares used to assess statistical significance. For each dependent variable parameter estimates were utilised, and main effects were compared as pairwise comparisons using a Bonferoni correction for repeated measures. The residuals were tested for normality as required by the linear mixed model with a set significance level of $p<0.05$. Based on a previous study employing these methods (Debenham et al., 2016c) a minimum sample size of 10

participants was determined sufficient to test the null hypothesis with a type I error of five percent, with power set at 80%.

RESULTS

Descriptive statistics are presented in Table 1. Following the eccentric loading protocol, lower limb stiffness significantly increased by 0.9 Nm^{-1} ($p<0.001$; 95% CI 0.7 to 1.1) (Figure 2b). Ankle angle 80ms pre-contact significantly decreased by 4.4° ($p<0.001$; 95% CI 3.1 to 5.8°) although ankle angle at contact did not change significantly. There was a shift in peak ankle angle towards a less dorsiflexed position (Figure 2a); this angle significantly decreased by 2.9° ($p<0.001$; 95% CI 1.5 to 4.3°) and ankle stretch amplitude significantly increased by 1.0° ($p=0.001$; 95% CI 0.3 to 1.7°).

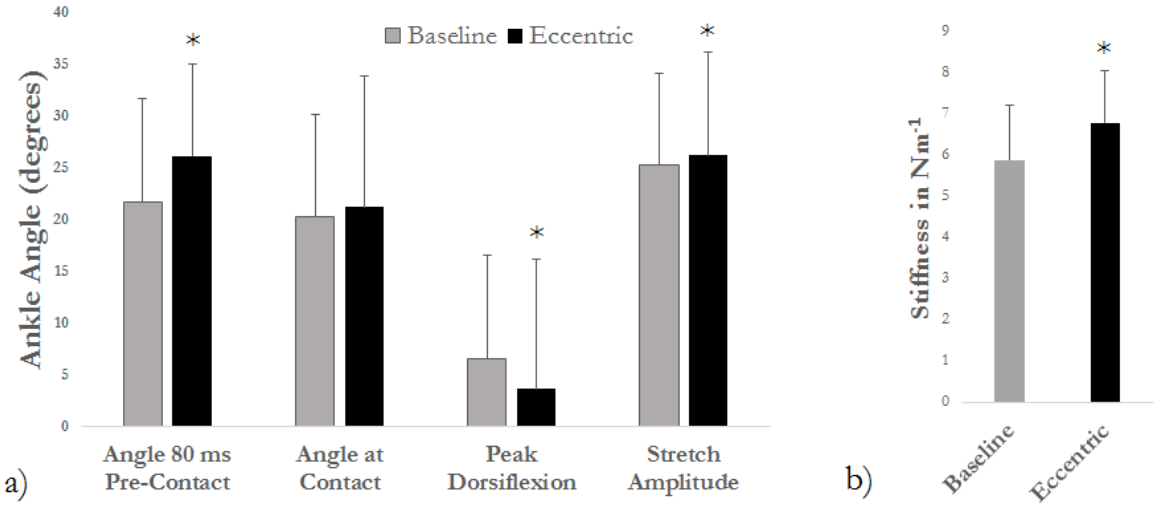

Figure 2: a) Ankle kinematics and **b)** lower limb stiffness at baseline and following the eccentric loading protocol.

* denotes significant difference ($p<0.05$)

sEMG measures of soleus occurred 44.7%, 17.4% and 14.6% earlier for onset, peak and offset value respectively (soleus onset: $p<0.001$; 95% CI 28 to 40; soleus peak: $p<0.001$; 95% CI 36 to 49; soleus offset: $p<0.001$; 95% CI 14 to 27 (Figure 3)). Tibialis anterior onset was delayed by 13.4% ($p=0.03$; 95% CI 1 to 23); tibialis anterior peak was not significantly different ($p=1.00$; 95% CI -13 to 8) and tibialis anterior offset was earlier ($p<0.001$; 95% CI 28 to 43)) (Figure 3).

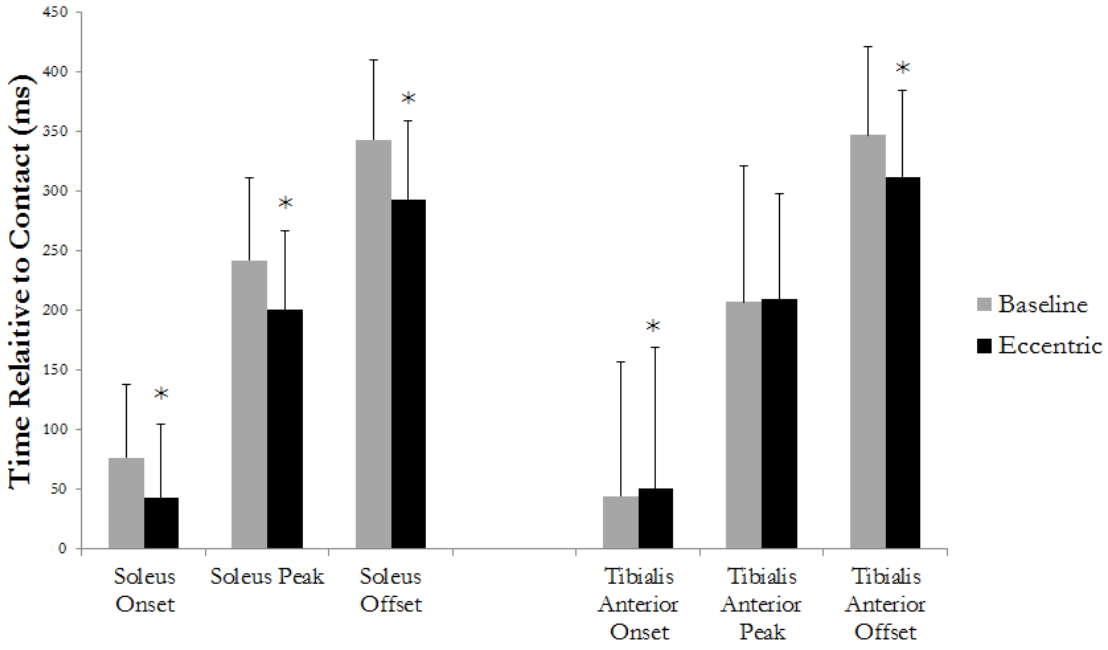

Figure 3: Soleus and tibialis anterior muscle onset timings at baseline and following the eccentric loading protocol.

* denotes significant difference ($p<0.05$)

DISCUSSION

Whilst the efficacy of eccentric loading for AT has been demonstrated (Magnussen et al., 2009) and theories regarding its efficacy have been explored (Crill et al., 2014), this is the first study to determine in which way SSC behaviour is modulated by eccentric loading. Our findings demonstrate that following eccentric loading, peak ankle angle shifts to less dorsiflexion (6.5 to 3.6° dorsiflexion) and lower limb stiffness increases (5.9 to 6.8 Nm^{-1}), whilst agonist/antagonist muscle activity moves to a more agonist-dominant pattern (e.g. soleus onset 34 ms earlier vs. tibialis onset 6 ms later) (see Figures 2 and 3).

Consistent with our hypothesis, we found that peak ankle angle shifted towards a position of relative plantarflexion. Whilst modest, these findings likely represent a clinically meaningful change when compared with the 7.7° and 3.2° changes in peak torque angle that have been previously reported at the hamstrings (Brockett et al., 2001)and plantarflexors (Wellisch et al., 2015) respectively following eccentric loading. Previous research has found that eccentric loading leads to increases in muscle strength and power (Crill et al., 2014) and an improved ability to produce force in the descending limb of the muscle fascicle length-tension curve (Reeves et al., 2004); our findings are consistent with these phenomena. Furthermore, our findings may reflect an increase in force producing

capacity of the muscle similar to those seen by Masood et al. (2014b) as increases in strength imply the plantarflexors have an increased capacity to resist excursion into dorsiflexion. Improved muscle performance also protects the tendon by conferring increased stiffness upon the tendon, thereby improving its capacity to resist strain (Mahieu et al., 2007). As such, these increases ensure tendon strain is not excessive, as is believed to be pathogenetic with AT (Pamukoff and Blackburn, 2015).

Also consistent with our hypothesis, we observed a 15% increase in lower limb stiffness following the eccentric loading intervention from 5.9 to 6.8 Nm^{-1} which was achieved by decreasing contact time and increasing flight time. Our findings are consistent with Elmer et al. (2012) who in their study of healthy volunteers observed a 10% increase in lower limb stiffness following an eccentric loading task. Increases in stiffness are generally associated with improvements in both performance in healthy individuals, and clinical improvements in patients with a pathologic condition (Butler et al., 2003). Our findings therefore likely reflect a true positive change in spring behaviour associated with the aforementioned alterations in muscular function following the intervention. Furthermore, given the 1 week timeframe involved, it is most likely that changes are neural rather structural in origin. Whilst we did not measure sEMG amplitude, the alterations in timing imply an integration of motor activity, which in turn results in increased stiffness (Markovic and Mikulic, 2010). Whilst it may be speculated that the increase in stiffness may be in part due to the impact that eccentric loading has on the connective tissues, the observed changes in SSC behaviour following a single loading event are unlikely to have occurred due to changes in in tendon material or mechanical properties (Arampatzis et al., 2007).

Consistent with our hypothesis, following the eccentric loading intervention, temporal muscle events occurred earlier in 4 out of our 6 measures and this is the first time temporal changes in sEMG have been demonstrated following an eccentric loading intervention. These findings most likely reflect training-induced changes in neural activation as described by Markovic and Mikulic (2010). Such changes include increased neural drive to the agonist muscle, as we observed with the hastening of soleus activity at all-time points. Likewise, our observed changes toward a more agonist dominant pattern of SSC performance may reflect changes in activation strategies (i.e. improved intermuscular co-ordination) (Markovic and Mikulic, 2010). The only other study exploring plantarflexor sEMG following eccentric loading is that of Masood et al.

(2014b). Their findings are consistent with our own; they observed an increase in plantarflexor sEMG amplitude following an eccentric loading intervention. Given that Cadore et al. (2014) observed no increase in sEMG amplitude following eccentric loading, it may be that eccentric loading induces changes in muscle timing rather than amplitude. This theory is supported by Masood et al. (2014b) who suggest that eccentric loading-induced changes in amplitude ultimately normalises muscle activity (i.e. return dysfunctional motor performance to 'normal').

Limitations to our study must be acknowledged. Our study was conducted on healthy volunteers; whilst this limits the generalisability of our findings to the target clinical population (AT), our findings form the basis upon which comparisons can be made with equivalent studies on the clinical population (Masood et al., 2014b). Although conventional sEMG is widely used to measure the electrical activity of skeletal muscle during activity, the information it provides may not reflect the activity of the whole muscle (Hodson-Tole et al., 2013). Given that intramuscular variations in muscle activity exist, findings such as ours may not reflect the true change in muscle behaviour (Tucker and Hodges, 2010). Whilst errors in kinematic measures may also occur due to soft tissue artefact of skin-attached markers, high within-subject reliability implies our methods accommodate this. Likewise, whilst the one week might be expected to result in natural variation in measures, pilot testing demonstrated high reliability (e.g. ICC of 0.77 for ankle stretch amplitude). Finally, whilst our study is designed to inform mechanisms underpinning a clinical intervention, the employed loading protocol is not reflective of standard clinical practice (Alfredson et al., 1998). This design however was purposeful, reflecting our interest in isolating our observations to changes in motor behaviour in response to loading rather than inducing chronic changes in tendon structure. Finally, our study did not measure the activity profile of gastrocnemius. Whilst this data would add value it was chosen to measure soleus in isolation due to its primary role in low level SSC-activities such as sub-maximal hopping and running (Ishikawa et al., 2006).

The eccentric phase of the SSC appears to be its critical phase (Cormie et al., 2010). Likewise, it is known that eccentric loading improves eccentric, but not concentric muscle performance (Cadore et al., 2014). Our study has demonstrated that eccentric loading induces what appear to be positive changes in SSC behaviour. Varied opinions exist regarding the mechanisms that underpin eccentric loading for AT (Allison and Purdam, 2009) with theories directed towards resolution of pathologic tendon structure and

function (Rees et al., 2008) or improvements in motor performance (Yu et al., 2013). Our findings provide support to the theory that changes in motor performance underpin such benefits. We also suggest that improvements in tendon structure that may occur in response to eccentric loading are secondary to improvements in motor performance. Our findings of reduced excursion into dorsiflexion and increased stiffness may represent the 'buffering' capacity of the muscle to protect the tendon as proposed by Lindstedt et al. (2002). Furthermore, AT is associated with excessive loading into dorsiflexion (Debenham et al., 2016c). Regardless of whether this kinematic phenomenon is a cause or consequence of tendinopathy it represents a maladaptive biomechanical state; excessive dorsiflexion during the SSC is sub-optimal for the muscles length-tension properties and under such circumstances excessive load is placed upon the passive structures. Our findings of reduced dorsiflexion indicate that this phenomenon reduces following eccentric loading and may play an important role in protecting the tendon from excessive tensile loading. Admittedly, given these methods it is difficult to ascertain whether these findings are unique to eccentric loading, or may be observed with combined eccentric-concentric loading. The theory that clinical benefits are conferred by improvements in motor performance (i.e. strength) is gaining traction, with Beyer et al. (2015) observing superior outcomes with resistance loading (combined heavy eccentric and concentric contractions) rather than by eccentric loading. Replication of this study with a combined group is warranted; the remaining mechanism of efficacy being related to the changes in peak torque angle that occur following eccentric, but not concentric loading (Wellisch et al., 2015).

Finally, whilst our study has no direct link to the pathogenesis of AT, it is relevant to speculate that our findings support those of McCrory et al. (1999) who stated that reduced plantarflexor performance acts as a factor to increase the prevalence of AT. Combining our findings with those of Masood et al. (2014a) observing improvements in symptoms and motor performance following eccentric loading in patients with AT, we suggest that impaired plantarflexor performance may be a significant pathogenic component of AT.

CONCLUSIONS

In summary, we have shown that in healthy adults, eccentric loading results in positive changes in ankle kinematics, lower limb stiffness, and agonist/antagonist muscle activity. These findings may reflect an increase in force producing capacity which leads to

decreased loading in end range dorsiflexion, and possibly protecting the tendon from excessive load and providing an opportunity to heal.

ACKNOWLEDGEMENTS

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7. DISCUSSION

7.1. INTRODUCTION

This thesis has presented a series of studies that relate to spring function; the concept that the bouncing nature of gait can be described using the spring mass model (SMM) and the stretch-shortening cycle (SSC) and can be modulated in response to a variety of factors. The crux of the thesis was to present a series of studies exploring this idea of spring function modulation and that this broad model can simplify the understanding of how fatigue can result in pathology, and how pathology might resolve in response to therapeutic exercise directed at improving spring function.

Achilles tendinopathy (AT) is a common clinical condition that affects active adults (de Jonge et al., 2011, Rio et al., 2014). Its pathology is complex, multifactorial and yet to be fully understood (Magnan et al., 2014) and as such, its optimal management is unclear. In recent times, evidence is building demonstrating that in the presence of pathology, the capacity of the lower limb to operate as a spring becomes dysfunctional (Kim and Yu, 2015, Maquirriain and Kokalj, 2014). Clearly, further work in this area is required to establish the extent and nature of these relationships (O'Neill et al., 2015). Furthermore, this complex multifactorial condition could benefit from a theoretical model with simplicity and utility (McEwan and Bigelow, 1997), one that could be employed by the clinical, research, and educational communities with which to better understand and ultimately assist in managing this challenging condition. This thesis has discussed the conceptual idea of 'spring function', which recognises that bouncing gait can be simply modelled mechanically using the SMM (Blickhan, 1989) and its dynamic behaviour explained physiologically by the SSC (Komi, 1984). Furthermore, the concept that whilst spring function is regulated according to real-time requirements (Taube et al., 2012), it can also be modulated, systematically changing in response to the nature and extent of the spring's functional (i.e. loading) history (Nicol et al., 2006).

This chapter will re-iterate the critical novel findings of this series of studies that explored the modulation of spring function. Collectively, the studies presented here demonstrate that in response to varying modulatory factors (pathology, fatigue, training), consistent neuromuscular modulation occurs in spring function that appears to be directed towards ensuring the maintenance of; 1) efficient task-oriented locomotion and; 2) protection of locomotor apparatus that are either compromised (pathology), under threat of

compromise (fatigue), or in a manner that confers protection (training). This chapter will also utilise the opportunity to present a theoretical model, 'Spring Theory', to explain the complex relationship that exists between pathogenic triggers, the development of an overuse condition, and its subsequent logical management. This chapter will re-iterate key findings of individual studies and their place within the existing literature (7.1 to 7.5), as well as speculate on the collective meaning of these studies and their role in supporting 'Spring Theory' (7.6 and 7.7).

7.2. CHAPTER 3- THE RELIABILITY OF A SLEDGE JUMP SYSTEM

This chapter described and established the reliability of a research tool that could explore spring function in a novel way, focusing attention at the ankle in an unloaded manner, enabling magnification of subtle neuromuscular alterations that may occur in response to modulatory triggers. In addition to describing the details of the sledge-jump-system (SJS) and the sub-maximal hopping task employed as the measurement task in this thesis, reliability was also established. No significant variation in key measures (stiffness and ankle kinematics) was observed between hopping periods, and strong reliability was established across trials for 4 out of 5 measures; likewise, testing occasion had minimal influence on the reliability of measures that had moderate/strong inter-trial reliability. Whilst reliability of SSC measurement tasks (using SJS and otherwise) have been conducted (Pappas et al., 2014, Moresi et al., 2014, McLachlan et al., 2006, Furlong and Harrison, 2013, Joseph et al., 2013, Waxman et al., 2015, Maloney et al., 2015, Kockum and Heijne, 2014, Gupta et al., 2014), this study provided detailed reliability of temporal measures of spring function pertinent to investigating neuromuscular changes in response to modulatory triggers.

7.3. CHAPTER 4- THE MODULATORY INFLUENCE OF ACHILLES TENDINOPATHY ON SPRING FUNCTION

Chapter 4 presented data on how AT modulates spring function. Importantly, AT was used as a representative model of an overuse condition relevant to spring function; likewise, it was important to present this pathologic model first, in order to highlight the intended clinical relevance of this thesis. This study found that in the presence of AT,

spring function is associated with an increase in stiffness, a shift in range towards greater dorsiflexion and an increase in joint excursion during ground contact, with a delay in associated muscle activity. This study (Debenham et al., 2016c) is now added to the several studies that have explored a variety of measures of spring function in individuals with AT, finding reductions in stiffness (Maquirriain and Kokalj, 2014, Maquirriain, 2012, Arya et al., 2006), and alterations in muscle activity (Masood et al., 2014a, Franettovich Smith et al., 2014, Wyndow et al., 2013, Wang et al., 2012, Baur et al., 2011, Azevedo et al., 2009) and joint kinematics (Kulig et al., 2011, Ryan et al., 2009, Azevedo et al., 2009, Williams et al., 2008, Donoghue et al., 2008b, Donoghue et al., 2008c, Donoghue et al., 2008a, Ryan et al., 2006). The novel findings from this study included the first demonstration that when unloaded, stiffness was increased, with perhaps a view to limiting exposure to the potentially threatening stimulus. Furthermore, the muscular and kinematic changes indicate that despite this attempt, yielding occurred at the ankle, suggesting that the plantarflexors lack the neuromuscular apparatus to resist spring loading, even when in an unloaded state.

These findings complement the recent findings of Chang and Kulig (2015b), who demonstrated similar changes in muscle activity, which in turn were associated with reduced tendon stiffness and a resultant electromechanical delay. These authors suggest that AT results in impaired afferent feedback (due to the compliant tendon), with delays and potential inaccuracies in information, further resulting in reduced efficiency and effectiveness of the execution of feedforward motor control, manifested as earlier pre-activation (Chang and Kulig, 2015b). The significance of this being that the neuromuscular system attempts to buffer the mechanically compromised Achilles tendon yet lacks the capacity to do so. Furthermore, with the addition of a de-loaded task (sub-maximal SJS hopping), these findings indicate that the neuromuscular adaptation is an attempt (increased stiffness) to maintain locomotor function whilst protect the spring system from the current threat (pathology). The consequences of such a pathologic state are that without appropriate neuromuscular capacity, the tendon has very little chance of regaining its function without intervention. With this in mind, there is logic that interventions should be designed that have the capacity to improve this neuromuscular function.

7.4. CHAPTER 5- THE MODULATORY INFLUENCE OF ECCENTRIC FATIGUE ON SPRING FUNCTION

Chapter 5 explored the modulatory role of fatigue on spring function in healthy volunteers, utilised to explore the possible role that fatigue may play in the pathogenesis of overuse conditions such as AT. This model recognised that that whilst multiple factors play a role, one of the most significant is the fatigue that an individual subjects their tissues to, which is determined by their local capacity (neuromuscular function) and the nature/magnitude of applied load (Lorimer and Hume, 2014). In this study, participants subjected to an eccentric loading fatigue protocol demonstrated increased stiffness, decreased ankle stretch amplitude during ground contact, and a hastening of muscle activity. In utilising the unloaded sub-maximal hopping task, much of the mechanical work was eliminated with the effective reduction of bodyweight. In doing so, it was possible to explore with greater acuity, the neural adaptations that occur in response to acute fatigue. Past studies have demonstrated inconsistent findings in terms of stiffness: decreased (Oliver et al., 2014, Fourchet et al., 2014, Hayes and Caplan, 2014, Girard et al., 2013, Comyns et al., 2011, Kuitunen et al., 2007, Kuitunen et al., 2002a, Dutto and Smith, 2002, Horita et al., 1996, Hobara et al., 2010), increased (Peng et al., 2015, Rumpf et al., 2013), and no change (Fischer et al., 2015, McBride et al., 2014, Joseph et al., 2014, Hunter and Smith, 2007, Padua et al., 2006, Morin et al., 2006) have all been reported in terms of stiffness changes associated with fatigue. These variations no doubt reflect differences in methodology. Of note, it appears that the studies finding reductions in stiffness employed functional methods of fatigue-induction (e.g. sports participation (Oliver et al., 2014), run to exhaustion (Hayes and Caplan, 2014)), highlighting the role of metabolic fatigue and central governance. In the study reported in this thesis, extreme local fatigue was induced using eccentric contractions in the (relative) absence of systemic metabolic fatigue. With the addition of an unloading task (sub-maximal SJS hopping), the findings indicate that the neuromuscular adaptation may be viewed as an attempt (increased stiffness, reduced contact time) to maintain locomotor function whilst protect the spring system from the current threat (fatigue).

7.5. CHAPTER 6- THE MODULATORY INFLUENCE OF ECCENTRIC LOADING ON SPRING FUNCTION

Chapter 6 explored, using these novel methods, how an eccentric loading protocol might confer a positive neuromuscular adaptation to spring function, in the context that this may explain the mechanism underpinning the clinical efficacy of eccentric loading for AT. The key findings of this study were an increase in stiffness and a shift in peak joint

angle towards a more plantarflexed position, with greater stretch amplitude and a hastening of associated agonist muscle activity. The findings from this study were consistent with the only other studies measuring stiffness following eccentric loading (Elmer et al., 2012, Lindstedt et al., 2002). However, this study is the first to report kinematic and muscle activation alterations during a SSC task. These findings can be incorporated within the existing literature that has demonstrated a number of changes reflective of improved spring function following eccentric loading, including positive alterations in neural drive (Cadore et al., 2014, Baroni et al., 2013), tendon mechanical properties (Obst et al., 2015, Morrissey et al., 2011, Porter et al., 2002) and muscular structure and function (Wellisch et al., 2015, Potier et al., 2009) as well as consistent and significant improvements in strength profile (Hortobagyi et al., 1996, Cadore et al., 2014, Roig et al., 2009). This study therefore provides supporting evidence that eccentric loading confers its clinical benefit by improving spring function, conferring a redistribution of relative load away from the tendon, towards the muscle, which acts as a mechanical 'buffer' to the tendon, providing capacity to the spring system that optimises its need to maintain locomotor function and protect it from the potential threat (overload).

7.6. SPRING FUNCTION MODULATION- AN ADAPTABLE SYSTEM

This thesis presented a series of studies that considered modulation of spring function under different, but inter-related conditions, and to see whether, in light of these conditions, consistencies could be observed in the manner by which spring function is modulated, speculating that the changes that occur could be related to the nature of the input, but occurring with a view to maintaining locomotor efficiency and protecting tissues from actual or potential harm. As has been demonstrated, this was achieved using a reliable experimental model (study 1), and each condition provided the opportunity to explore a different modulatory factor, representative of a common trajectory of overuse lower limb injury. In this trajectory excessive fatigue (study 3), within the right context, leads to an overuse injury (AT (study 2)), which can be treated with a related intervention (eccentric loading (study 4)) that confers protection upon the spring. These studies found that the lower limb spring does consistently modulate in response to triggers with varying biomechanical and contextual meaning. In all studies, stiffness of the spring increased, and it is suggested here that in the same way the lower limb regulates function by

adjusting stiffness, it does so also in response to modulatory triggers in order to maintain locomotor efficiency and protection. In the fatigue study and the AT study, it is most likely that this was done in order to minimise contact time and thus reduce exposure to ground contact, which would be perceived as the 'threatening' event (a **'reactive-protective' response**). Conversely, increased stiffness was also observed in the eccentric loading study, but for different biomechanical and contextual reasons. In this case, it is likely that the eccentric loading protocol provides a training stimulus that provides improved spring function, enabling optimised locomotor function and protection as occurs in the presence of increased stiffness (a **'proactive-protective'** response). It has been suggested that an optimal level of stiffness is required for safe and efficient locomotion, with insufficient and excessive levels of stiffness being detrimental to both performance, and injury risk (Butler et al., 2003). It is suggested here though that stiffness can be represented as an asymmetric Laffer curve, skewed to the right (see Figure 7.1). In this scenario a number of comments need to be made.

1. There is an optimal stiffness level for safe and efficient locomotion
2. In response to modulatory stimuli, stiffness increases for protective purposes
3. If stiffness is sub-optimal, locomotor efficiency is reduced and injury risk elevated
4. The systems' response to external factors is an attempt to modulate stiffness towards optimal
5. Most external factors result in
    a. …a loss in mechanical capacity to generate increased stiffness
    b. …a compensatory neural attempt to generate increased stiffness
6. Training (if performed appropriately) increases local neuromuscular capacity and stiffness potential

Figure 7.1. Representation of stiffness and its modulatory factors as an asymmetric Laffer curve. An optimal level of stiffness is desired for safe and efficient locomotion. Certain factors will systematically reduce stiffness (negative modulation), and a neural response to this will occur that attempts to restore stiffness. Conversely, training has the potential to provide the neuromuscular apparatus that provides the system with optimal stiffness (positive modulation).

In addition, the consistent changes in stiffness are accompanied by other more subtle kinematic and sEMG changes that potentially highlight that whilst the systems primary goal is one of safe and efficient locomotion, there are differences that might reflect the multiple modulatory influences at play. Under fatigue, we see a reduction in stretch amplitude; this potentially reflects the fact that despite the presence of local fatigue, the system has the structural architecture with which to reduce threat-exposure (i.e. musculotendinous passive capacity is largely retained), and the unloaded nature of the task enables this to be highlighted. Conversely, in the pathological state, we see an increase in stiffness but an increase in stretch amplitude, potentially reflecting the systems attempt to limit threat exposure, yet **without** the structural apparatus with which to do so (i.e. musculotendinous stiffness). Finally, following eccentric loading, where we would expect to observe systematic improvements in neuromuscular capacity, we see associated with increased stiffness a shift in ground contact range of motion towards greater plantarflexion and with increased stretch amplitude, a position where the SSC can be performed with optimal utilisation of its musculotendinous apparatus. This co-ordinated neuromuscular response, which appears to be context-dependent describes a system where the structural apparatus of the spring system (musculotendinous stiffness), under the careful control of the nervous system provide protection to the spring system in

either a reactive manner (fatigue and pathology) or a proactive manner (post-loading), whilst continuing to produce efficient locomotion.

7.7. SPRING THEORY- AN INTEGRATED MODEL OF OVERUSE LOWER LIMB INJURY

The series of results presented in this thesis in the context of the literature review provide the opportunity to present novel theoretical model, termed 'Spring Theory'. Theoretical models attempt to make sense of complex sets of (clinical) phenomena, and in this model an attempt is made to explain AT, but can be extrapolated to many other overuse lower limb injuries. Theoretical models such as this are beneficial for clinicians, seeking to simplify and optimise the management of often complex patient presentations, and for students and educators, seeking to transfer meaningful knowledge on a complex subject. Finally, a theory is merely a theory until it is subjected to scientific scrutiny and this model directs researchers towards opportunities to conduct hypothesis testing of ideas that relate to this theory (see section 7.8).

AT is a complex clinical condition, with a multifactorial pathogenesis. This pathogenesis is primarily mechanical (Scott et al., 2015), but has significant contributions from systemic sources (Magnan et al., 2014) and emerging evidence of CNS involvement (Plinsinga et al., 2015). Furthermore our understanding of its clinical presentation is unclear, typified by the discordant relationship between clinical severity and pathohistological findings (Ryan et al., 2015, De Jonge et al., 2014). Subsequently there is a myriad of clinical interventions used to manage this condition. Yet convincing models on which to base clinical interventions remain incomplete or elusive. The model described by 'Spring Theory' is deliberately simple, in order to achieve its primary aim of providing a platform upon which to superimpose clinical and research 'models' that have the agility to adjust to the multifactorial nature of the condition.

'Spring Theory' is a clinical/research/educational model to help explain lower limb overuse injury and AT is used as a clinical illustration. This theoretical model is based on the conceptualisation that the lower limb operates mechanically as a spring, based on the SMM (Blickhan, 1989). However, it also recognises that this spring is driven by a neuroplastic SSC (Komi, 1984), which can be modulated (systematic changes in neuromuscular properties) in response to factors such as pathology, fatigue and training (Debenham et al., 2016a, Debenham et al., 2016c) in order to produce locomotion that

is safe and efficient. In essence, Spring Theory is a three part model. (1) Initially, it recognises fatigue, a reflection of the intrinsic local capacity of the neuromuscular apparatus and the load it is subjected to, modulates spring function to the point where despite reactive protective neural strategies, structural function becomes sufficiently impaired such that muscle is no longer able to protect tissues to the extent that is required. Unlike other pathogenic models, this one purposefully removes focus from the terminal tissue (i.e. tendon), considering this to be an 'outcome' of a broader functional problem of the lower limb musculoskeletal system (i.e. the leg spring). (2) In the second stage, resultant tissue breakdown occurs with insufficient opportunity to recover and ultimately a pathologic state ensues. In the case of AT, this is further contributed to by the unique nature of tendinopathy that is characterised by a 'failed healing response' (Fu et al., 2010). This state is characterised by impaired spring function, where the system attempts to maintain safe, locomotor efficiency but in the absence of sufficient neuromuscular function, pathology persists. This model recognises that in *status quo* there is no logical reason why the condition should improve independently as spring function will remain dysfunctional until purposefully attended to. (3) Finally, it is suggested that interventions for conditions that are manifestations of spring dysfunction need to be based on appropriately addressing the primary drivers of the condition. This model suggests that the dominant driver is the neuromuscular apparatus of the lower limb given that its role is to confer this protection; therefore rehabilitation should be specifically directed at improving spring function. Again using the example of AT, a large (but not sole) feature using this model would be to improve the neuromuscular function of the plantarflexors in isolation, then consider the remote regions of the spring (knee and hip extensors), before combining these with exercise-based interventions to improve global spring function. This thesis explored one commonly prescribed intervention under this paradigm, but much work needs to be done to clarify the exact place where eccentric loading sits in a rehabilitation model based on 'Spring Theory'. For instance, whilst not questioning efficacy, dosages prescribed originally by Alfredson et al. (1998) (180 reps x bodyweight ± load daily) are inconsistent with current best practice for increasing strength (e.g. 3-5 sets of 4-8 repetitions (Rhea et al., 2003), or peak torque angle (Wellisch et al., 2015), which are potentially more likely operational mechanisms underpinning improvements in spring function.

7.8. THESIS LIMITATIONS

This thesis has attempted to provide a narrative that integrates the findings of independent studies to offer a novel and significant knowledge contribution. This thesis is not without limitations. Many of these have already been discussed in individual studies, but below, general thesis limitations are hereby recognised.

Chapter 4 - 6 measure only the EMG of the soleus muscle and not of gastrocnemius, the other major plantarflexor contributor to spring function. This is an unfortunate omission that fell outside the scope of this PhD as its inclusion would provide significant additional insight into the modulatory processes that occurred as a result of the different 'states' studied here.

Chapter 4 makes a comparison between a young healthy group and a relatively older (middle-aged) group of individuals with Achilles tendinopathy. This limitation has been recognised within the manuscript and whilst it does not significantly impact the implications of the findings, it bares further recognition at this stage of the thesis. This reflects the fact that it is known that there is a change in tendon structure and mechanical properties with increasing age and some difference might be due to age and some due to injury. Furthermore, this study did not explore the influence that changes in tendon structure, or tendon pain (both important components of Achilles tendinopathy) may, or may not have had on spring function modulation. This would be a highly valuable area for future scientific investigation.

Chapter 5, in its exploration of fatigue on spring function, explored the effect of soleus fatigue using a bent-knee heel raise task. However, the testing task was upright hopping, where gastrocnemius plays a relevant role. This discordance between fatiguing protocol and testing procedure was chosen to bias the scrutiny to the effect of soleus; however, inclusion of EMG evaluation of gastrocnemius would have advanced this understanding.

Chapters 5 and 6 exploring the effect of fatigue, and eccentric loading did utilise common participants. It is possible that the sampled healthy participants are not completely representative of the larger population. Whilst independent samples would have been preferable, study constraints restricted this and represents only a minor overall limitation; reproduction of these findings would strengthen the conclusions of both studies. Likewise, speculation has been made on the implications that these findings may have for the prevention and management of Achilles tendinopathy. Repeating these studies on

individuals with Achilles tendinopathy would provide significant further insight in terms of corroborating the relevance of these findings in a healthy population.

7.9. SUMMARY OF THE THESIS

This thesis has presented a series of studies exploring modulation of spring function. The series of studies demonstrate that all stimuli (pathology, local plantarflexor fatigue and eccentric loading) modulated spring function, principally by increasing lower limb stiffness. This can be interpreted as an adaptive change in the spring system to optimise locomotor efficiency under the present circumstances whilst minimising the risk of current, potential or future tissue damage. Furthermore, individual variations in kinematic and muscle activity possibly reflect subtle changes in strategies that are context dependent. That is, dependent upon the integrity of the neuromuscular system under that condition, with specific adaptations made toward a 'best attempt' to optimise safe and efficient locomotion. In two of the investigated conditions (pathology and fatigue), a '**reactive-protective**' state was achieved, whilst in the third (eccentric loading), a '**proactive-protective**' state was achieved. These findings are based on novel but robust and reliable methods, and are significant in terms of the contribution they make to the concept of spring function modulation. In addition, the results of these studies allowed the proposition of a novel theory for clinical, educational and research within the topic of overuse lower limb injury. This 'Spring Theory' refers to spring function as a combination of the spring mass model and the stretch shortening cycle phenomenon, recognising the capacity for spring function to modulate in accordance with its intrinsic capacity, and the functional loading it is subjected to. This model provides a platform upon which findings relating to the pathogenesis, clinical features and rehabilitation of overuse lower limb injuries such as AT can be built. This model has the potential to simplify and optimise clinical practice of these injuries, create educational strategies for teaching lower limb overuse injuries to physiotherapists, and potentially direct hypothesis testing research streams.

APPENDICES

APPENDIX I- CONTRIBUTION OF OTHERS

PROFESSOR GARRY T ALLISON

	SJS Reliability	Achilles Tendinopathy	Fatigue	Eccentric Loading
Conception and design of research	✓	✓	✓	✓
Performed experiments				
Analysed data				
Interpreted results of experiments				
Prepared figures				
Drafted manuscript				
Edited and revised manuscript	✓	✓	✓	✓
Approved final version of manuscript	✓	✓	✓	✓

PROFESSOR MAX BULSARA

	SJS Reliability	Achilles Tendinopathy	Fatigue	Eccentric Loading
Conception and design of research				
Performed experiments				
Analysed data	✓	✓	✓	✓
Interpreted results of experiments	✓			
Prepared figures				
Drafted manuscript				
Edited and revised manuscript	✓			
Approved final version of manuscript	✓			

DR. AMITY CAMPBELL

	SJS Reliability	Achilles Tendinopathy	Fatigue	Eccentric Loading
Conception and design of research	✓	✓	✓	✓
Performed experiments				
Analysed data				
Interpreted results of experiments	✓	✓	✓	✓
Prepared figures				
Drafted manuscript				
Edited amd revised manuscript	✓	✓	✓	✓
Approved final version of manuscript	✓	✓	✓	✓

DR. WILLIAM GIBSON

	SJS Reliability	Achilles Tendinopathy	Fatigue	Eccentric Loading
Conception and design of research	✓	✓	✓	✓
Performed experiments	✓	✓	✓	✓
Analysed data				
Interpreted results of experiments	✓	✓	✓	✓
Prepared figures				
Drafted manuscript				
Edited and revised manuscript	✓	✓	✓	✓
Approved final version of manuscript	✓	✓	✓	✓

DR. TIFFANY GRISSBROOK

	SJS Reliability	Achilles Tendinopathy	Fatigue	Eccentric Loading
Conception and design of research				
Performed experiments				
Analysed data	✓	✓	✓	✓
Interpreted results of experiments				
Prepared figures				
Drafted manuscript				
Edited and revised manuscript	✓			
Approved final version of manuscript	✓			

DR MERVYN TRAVERS

	SJS Reliability	Achilles Tendinopathy	Fatigue	Eccentric Loading
Conception and design of research	✓	✓	✓	✓
Performed experiments	✓	✓	✓	✓
Analysed data				
Interpreted results of experiments				
Prepared figures				
Drafted manuscript				
Edited and revised manuscript	✓	✓	✓	✓
Approved final version of manuscript	✓	✓	✓	✓

APPENDIX II- PARTICIPANT INFORMATION SHEETS AND CONSENT FORMS

SJS RELIABILITY

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PARTICIPANT INFORMATION SHEET

Study Title: "An investigation into the reliability of measuring muscle and joint behaviour during single-limb hopping"

Principal investigator: James Debenham
Co-investigators: Associate Professor Garry Allison; Dr William Gibson; Dr Amity Campbell

Purpose of Research

This study is part of a series of studies looking at the way in which muscles and joints work and behave when humans hop. The aim of the research is to help increase understanding of how some people sustain chronic lower limb injuries like Achilles tendon problems. It may also assist in the development of new strategies to prevent and/or treat this condition. The studies employ a device, known as an inclined-sleigh, or 'sleigh' for short. The sleigh allows subjects to freely hop on an incline with their bodyweight supported. This enables measurement of how the muscles and joints behave whilst subjects hop in a much more accurate way than during upright hopping. Because the sleigh has never been used before, the accuracy and reliability of the measurements being taken needs to be determined. By knowing this future studies can be performed in the knowledge of how precise the sleigh is for measuring muscle activity and joint movement.

Your Role

Testing takes place in the Motion Analysis Laboratory at Curtin University of Technology's Bentley campus. You will be required to attend testing on 3 different occasions (Day 1, 3 and 7). On each occasion you will be required to wear shorts and a t-shirt. You will have various markers placed on your legs to allow us to take measurements. If necessary, this may require shaving small patches of hair (approximately 5 cm) so the markers stick well. You will be given an explanation and a demonstration regarding the hopping task you will be performing. You may ask questions at any point. Following that you will be positioned on the sleigh and required to hop for 30 second periods when instructed. This will be repeated over a period of 20 minutes. This process will be repeated identically on the subsequent occasions that we test. The whole process will take less than an hour on each occasion.

Risks and Discomfort

There is no inherent risk in the activity you will be requested to perform. You may feel some very mild fatigue towards the end of testing. The various markers placed on your skin may cause some mild discomfort when they are removed.

Benefits

There is no direct benefit to you from participating in this study. However information gained from this study may ultimately provide insights into the mechanisms underlying certain musculoskeletal lower

limb conditions. It may also aid in the development of rehabilitation protocols for such conditions in the general community.

Consent to Participate

Your involvement in this research is entirely voluntary. If you choose not to participate or at any point in the study you wish to withdraw, you have the right to do so without explanation, justification or consequence of any kind. After reading this form and asking any questions you may have, you will be asked to sign a consent form indicating you voluntarily agree to participate in this study and you are aware of all your rights in relation to participation.

Confidentiality

Your privacy is greatly respected. We will not ask any personal information of you beyond information such as height, weight, age and past musculoskeletal injury history. The data collected from you will be kept separate from your personal details, and only the investigators will have access to this. The data collected from you will have all identifying information removed. This is in adherence to University policy. All data will be securely stored for a period of 7 years, following which it will be destroyed. Any publications or presentations resulting from this study will be presented as averages, thus not personally identifiable to any one individual.

Further Information

This study has been approved by the Curtin University Human Research Ethics Committee (Approval Number xxxxxx). The Committee is comprised of members of the public, academics, lawyers, doctors and pastoral carers. Its main role is to protect participants. If needed, verification of approval can be obtained either by writing to the Curtin University Human Research Ethics Committee, c/- Office of Research and Development, Curtin University of Technology, GPO Box U1987, Perth, 6845 or by telephoning 9266 2784 or by emailing hrec@curtin.edu.au . If you would like further information about the study, please feel free to contact me on 08 9266 3667 or by email: j.debenham@curtin.edu.au.

Thank you very much for your involvement in this research. Your participation is greatly appreciated.

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Consent Form

- I understand the purpose and procedures of the study
- I have been provided with the participation information sheet
- I understand that the study itself may not benefit me
- I understand that my involvement is voluntary and I can withdraw at any time without problem
- I understand that no personal identifying information like my name and address will be used and that all information will be securely stored for 7 years before being destroyed
- I agree that research gathered from this study may be published provided that any information that may identify me is not used
- I have been given the opportunity to ask questions
- I agree to participate in the study outlined to me

This study has been approved by the Curtin University Human Research Ethics Committee (Approval Number PT0151/2009 (Part 1) and PT0145/2009 (Part 2)). The Committee is comprised of members of the public, academics, lawyers, doctors and pastoral carers. Its main role is to protect participants. If needed, verification of approval can be obtained either by writing to the Curtin University Human Research Ethics Committee, c/- Office of Research and Development, Curtin University of Technology, GPO Box U1987, Perth, 6845 or by telephoning 9266 2784 or by emailing hrec@curtin.edu.au . If you would like further information about the study, please feel free to contact me on 08 9266 3667 or by email: j.debenham@curtin.edu.au, m.travers@curtin.edu.au

All participants are informed that, if they have any complaint regarding the manner, in which a research project is conducted, it may be given to the research supervisor (Ass Proc GT Allison, 9266 3626) or, alternatively to the Secretary, Human Research Ethics Committee, School of Physiotherapy, Curtin University (Tel: 9266 2784). All study participants will be provided with a copy of the information sheet and the consent form for their personal records.

Name: _____ Signature: _____ Date: _____

Witness: _____ Signature: _____ Date: _____

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PARTICIPANT INFORMATION SHEET

Study Title: "The influence of Achilles Tendinopathy on muscle and joint behaviour during single-limb hopping"

Principal investigator: James Debenham
Co-investigators: Associate Professor Garry Allison; Dr William Gibson; Dr Amity Campbell

Purpose of Research

Currently, we have little understanding of how humans perceive pain and by which mechanisms humans control movement whilst experiencing pain. This study is part of a series of studies looking at the way in which muscles and joints behave when humans move whilst experiencing pain. The aim of the research is to help increase understanding of how some people sustain chronic lower limb injuries. It may also assist in the development of new strategies to prevent and/or treat such conditions.

This study employs a device, known as an inclined-sleigh, or 'sleigh' for short. The sleigh allows subjects to hop as they would in standing, yet while laying at an incline. This means that the sleigh supports a large proportion of their bodyweight and they can hop for longer without feeling tired. During this controlled hopping we can assess how the muscles and joints work in a much more accurate way than during upright hopping.

We do not yet know if, or to what extent pain affects muscle and joint behaviour during this movement. To aid in our investigation of this, we would like to observe individuals with Achilles Tendinopathy and see how muscle and joint behaviour changes over the course of the condition.

Your Role

Testing will take place in the Motion Analysis Laboratory at Curtin University of Technology's Bentley campus. You will be required to attend for testing on every 3 months following the first testing session until the condition has resolved. On each occasion you will be required to wear shorts and a t-shirt. You will have various markers placed on your legs to allow us to take measurements. If necessary, this may require shaving small patches of hair (approximately 5 cm) so the markers stick well. You will be given an explanation and a demonstration regarding the hopping task you will be performing. You may ask questions at any point.

You will perform a series of 8 hopping trials on each leg, each lasting 15 seconds. Between each trial you will rest for 30 seconds. Following this your participation in the study will be complete.

Risks and Discomfort

We are interested in factors related to the pain you are experiencing in you Achilles tendon. As such, testing will cause some discomfort. However, the levels of discomfort that you feel will be similar to those that you would be feeling walking up a set of stairs. Available research suggests that this testing should present no risk to the patency of your Achilles tendon, nor should it exacerbate or prolong the condition. However, given that this experiment has not been done before under these conditions, you must be warned that a very small risk exists that either of these adverse events may take place.

Curtin
University of Technology

Benefits

There is no direct benefit to you from participating in this study. However information gained from this study may ultimately provide insights into the mechanisms underlying Achilles tendinopathy. It may also aid in the development of rehabilitation protocols for the condition.

Consent to Participate

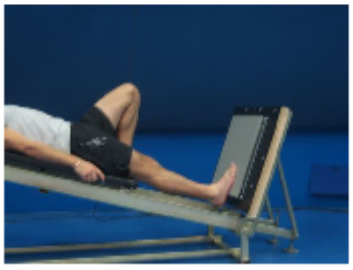
Your involvement in this research is entirely voluntary. If you choose not to participate or at any point in the study you wish to withdraw, you have the right to do so without explanation, justification or consequence of any kind. After reading this form and asking any questions you may have, you will be asked to sign a consent form indicating you voluntarily agree to participate in this study and you are aware of all your rights in relation to participation.

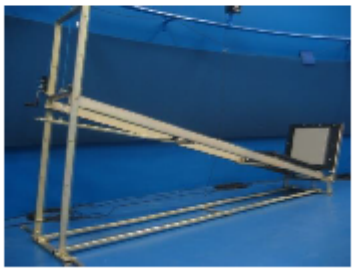
Confidentiality

Your privacy is greatly respected. We will not ask any personal information of you beyond information such as height, weight, age and past musculoskeletal injury history. The data collected from you will be kept separate from your personal details, and only the investigators will have access to this. The data collected from you will have all identifying information removed. This is in adherence to University policy. All data will be securely stored for a period of 7 years, following which it will be destroyed. Any publications or presentations resulting from this study will be presented as averages, thus not personally identifiable to any one individual.

Further Information

This study has been approved by the Curtin University Human Research Ethics Committee (Approval Number HR28/2010). The Committee is comprised of members of the public, academics, lawyers, doctors and pastoral carers. Its main role is to protect participants. If needed, verification of approval can be obtained either by writing to the Curtin University Human Research Ethics Committee, c/- Office of Research and Development, Curtin University of Technology, GPO Box U1987, Perth, 6845 or by telephoning 9266 2784 or by emailing hrec@curtin.edu.au . If you would like further information about the study, please feel free to contact me on 08 9266 3667 or by email: jdebenham@nd.edu.au.





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- I understand that my involvement is voluntary and I can withdraw at any time without problem
- I understand that no personal identifying information like my name and address will be used and that all information will be securely stored for 7 years before being destroyed
- I agree that research gathered from this study may be published provided that any information that may identify me is not used
- I have been given the opportunity to ask questions
- I agree to participate in the study outlined to me

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All participants are informed that, if they have any complaint regarding the manner, in which a research project is conducted, it may be given to the research supervisor (Ass Proc GT Allison, 9266 3626) or, alternatively to the Secretary, Human Research Ethics Committee, School of Physiotherapy, Curtin University (Tel: 9266 2784). All study participants will be provided with a copy of the information sheet and the consent form for their personal records.

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PARTICIPANT INFORMATION SHEET

Study Title: "The influence of muscle soreness on muscle and joint behaviour during single-limb hopping"

Principal investigator: James Debenham
Co-investigators: Associate Professor Garry Allison; Dr William Gibson; Dr Amity Campbell

Purpose of Research

Musculoskeletal pain is a significant burden to the community in terms of loss of function and the effect this has on work performance. In addition, it poses a significant burden to the health care system. Currently, we have little understanding of human pain perception and the mechanisms behind muscle control of movement whilst experiencing pain.

This study is part of a series of studies looking at the way in which muscles and joints behave when humans hop whilst experiencing pain. The aim of the research is to help increase understanding of how some people sustain chronic lower limb injuries. It may also assist in the development of new strategies to prevent and/or treat such conditions.

This study employs a device, known as an inclined-sleigh, or 'sleigh' for short. The sleigh allows subjects to hop as they would in standing, yet while laying at an incline. This means that the sleigh supports a large proportion of their bodyweight and they can hop for longer without feeling tired. During this controlled hopping we can assess how the muscles and joints work in a much more accurate way than during upright hopping.

We do not yet know if, or to what extent pain affects this movement or the muscle behaviour. For us to be able to investigate this, we will use an experimental pain model that imitates clinical pain conditions for a period of time.

To achieve this, we will induce what is known as 'delayed onset muscle soreness', or DOMS for short. This is similar to the pain you might have experienced in your muscles in the past after performing an unaccustomed activity. It is perfectly safe and once the soreness subsides, you will feel no different to before the test. We intend to induce DOMS in your calf or shin muscle by getting you to perform a simple exercise procedure where the muscle is exercised to fatigue. 24 hours after performing this exercise, you will begin to experience DOMS in the muscle and this pain will last approximately 1 week.

Your Role

Testing will take place in the Motion Analysis Laboratory at Curtin University of Technology's Bentley campus. You will be required to attend testing on 6 different occasions. On each occasion you will be required to wear shorts and a t-shirt. You will have various markers placed on your legs to allow us to take measurements. If necessary, this may require shaving small patches of hair (approximately 5 cm) so the

markers stick well. You will be given an explanation and a demonstration regarding the hopping task you will be performing. You may ask questions at any point.

You will first be required to perform a simple 'calf raise' exercise to fatigue your muscle. You will be guided through this exercise, which takes only a few minutes. Following that you will be positioned on the sleigh and required to hop for 30 second periods when instructed. This will be repeated 10 times immediately, and then repeated 1 hour later. During testing, you will be asked to provide information regarding the presence and severity of any soreness you are experiencing. The whole process on this first occasion will take 90 minutes. You will be asked to return for testing 2 days and 7 days later. On these occasions the exercise will not be performed, only the testing. These subsequent sessions will last approximately 30 minutes. You will be requested to repeat the process for 2 further muscles, but this must be done no sooner than 1 week after the previous muscle.

Risks and Discomfort

There is no inherent risk to you or your muscle in the activity you will be requested to perform. You will feel fatigue towards the end of the exercise which will settle within about 10-20 minutes. The hopping trial itself will not be tiring. Whilst experiencing DOMS the hopping activity may enhance soreness. However, this will not be severe and will be no worse than what you would experience walking up some stairs when you have DOMS. Markers will be fixed to your skin with low-allergenic tape. This may cause some mild discomfort when they are removed.

Benefits

There is no direct benefit to you from participating in this study. However information gained from this study may ultimately provide insights into the mechanisms underlying certain musculoskeletal lower limb conditions. It may also aid in the development of rehabilitation protocols for such conditions in the general community.

Consent to Participate

Your involvement in this research is entirely voluntary. If you choose not to participate or at any point in the study you wish to withdraw, you have the right to do so without explanation, justification or consequence of any kind. After reading this form and asking any questions you may have, you will be asked to sign a consent form indicating you voluntarily agree to participate in this study and you are aware of all your rights in relation to participation.

Confidentiality

Your privacy is greatly respected. We will not ask any personal information of you beyond information such as height, weight, age and past musculoskeletal injury history. The data collected from you will be kept separate from your personal details, and only the investigators will have access to this. The data collected from you will have all identifying information removed. This is in adherence to University policy. All data will be securely stored for a period of 7 years, following which it will be destroyed. Any publications or presentations resulting from this study will be presented as averages, thus not personally identifiable to any one individual.

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Consent Form

Project Title: The Influence of Endogenous Pain on Stiffness Modulation during Sub-Maximal Single Limb Hopping

Principal Investigator: James Debenham, PhD Candidate
Supervisors: Associate Professor Garry Allison, Dr William Gibson, Dr Amity Campbell

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APPENDIX III- ECCENTRIC LOADING INCREASES PEAK TORQUE ANGLE OF THE ANKLE PLANTAR FLEXORS IN HEALTHY VOLUNTEERS

International Journal of
Sports and Exercise Medicine

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Eccentric Loading Increases Peak Torque Angle of the Ankle Plantar Flexors in Healthy Volunteers

Matthew Wellisch[1], Peter Hamer[1], Luke Hopper[2], Max Bulsara[1] and James Debenham[1]*

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Abstract

Eccentric loading of the ankle plantar flexor's (PF) has demonstrated clinical efficacy in the conservative treatment of Achilles tendinopathy, however, its mechanism of therapeutic benefit remains unclear. The purpose of this study was to examine the effects of PF eccentric loading on PF angle to peak torque (AtPT), peak torque (PT) and lower limb vertical stiffness. Thirty healthy volunteers were randomised to an eccentric (n=15) or concentric (n=13) exercise group. A 10-week loading programme of the ankle plantar flexors was completed. AtPT, PT and vertical stiffness were compared within and between groups before and after the interventions. AtPT increased in the eccentric group by 3.2° dorsiflexion (p=0.001) and decreased by 0.7° dorsiflexion (p=0.528) for the concentric group with significant post-intervention group differences (p<0.001). PT levels were unchanged following the interventions for both groups (p>0.2); however, post-intervention the eccentric group showed a greater PT than the concentric group (p>0.05). Between group comparison showed no significant difference in vertical stiffness (p>0.5). However, the concentric group demonstrated a vertical stiffness increase of 765kNm^{-1} (p ≥ 0.05). This study demonstrates that a clinically-derived eccentric loading programme can produce an adaptive shift in AtPT of the ankle plantar flexors in a healthy population. These results support the theory that in part, eccentric loading derives its therapeutic benefit from mechanisms that influence plantar flexor motor performance.

Keywords

Achilles tendinopathy, Eccentric exercise, Plantar flexors, Muscle performance

Introduction

Achilles tendinopathy (AT) is a common overuse condition characterised by impaired physical function due to pain [1]. The incidence of AT in registered patients presenting to general practitioners is 1.85 per 1000, with average presentation age of forty-three [2]. Although the natural history of AT is unclear, early conservative treatment is thought to be optimal, with eccentric exercise demonstrated to be one of the more efficacious conservative treatments currently available [3-5]. Furthermore, eccentric loading protocols offer superior outcomes to concentric protocols in terms of pain and function [6-8] and the eccentric loading protocol outlined by Alfredson et al. [9] is widely used in the literature and clinically. Whilst the clinical efficacy of eccentric loading as an intervention for AT has been established the underpinning mechanisms behind its benefit are unclear. A number of theories have been proposed to explain the mechanism of efficacy for eccentric loading; one theory suggests mechanisms influencing pathology [10,11], whilst another suggests mechanisms influencing tendon structure [12-14]. However, it appears that these explanations alone are insufficient to account for the positive outcomes observed following eccentric loading, leaving a third biomechanical theory as an alternative explanation. This theory suggests mechanisms that positively influence motor performance [15].

Eccentric loading has been shown to improve many measures of motor performance, including peak torque (PT) [8,9], angle to peak torque (AtPT) [16,17] and vertical stiffness [18,19]. Interestingly, motor performance deficits in PT and vertical stiffness have been observed in individuals with AT [9,20,21]. Even though the mechanism of efficacy is not yet clearly understood, it is known that eccentric contractions serve as a mechanical stimulus that results in muscle fibre damage and a subsequent adaptive shift in the muscles length-tension relation to longer muscle lengths [22].

Whilst, eccentric loading has been demonstrated to increase PT, AtPT and vertical stiffness in other regions, this has not been demonstrated at the ankle; furthermore, it has not been demonstrated whether these changes are a unique feature of eccentric loading over concentric loading. Therefore the aim of this study was to investigate alterations in motor performance of the plantar flexors to a therapeutic eccentric loading protocol as compared to an equivalent concentric loading protocol. The logical foundation of this biomechanical investigation is to collect data from a healthy population to provide a baseline for the measures of interest. We hypothesised that eccentric loading would result in an increase in PT, AtPT and vertical stiffness of the ankle plantar flexors compared to a comparable concentric exercise. Given the relevance of eccentric loading in the management of AT, a better understanding of the mechanisms underpinning its efficacy should be sought. With a greater understanding of this, prescriptions of this therapeutic intervention can be progressed.

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APPENDIX IV- EXPANDED METHODS FOR CHAPTER 3

METHODS

PARTICIPANTS

Fifteen healthy participants (8 females and 7 males; mean age 27.4 ± 5.6 years, height 171.2 ± 10.7 cm, mass 71.1 ± 15.9 kg) took part in this study. Participants were excluded if they were pregnant, had significant medical or psychological illness, significant visual impairment, or if medications that may affect motor performance were being taken. Likewise, participants were excluded if they had had significant lower quadrant neuromusculoskeletal pathology in the preceding 12 months, or if they had ever undergone lower limb surgery. The study was approved by Curtin University Human Research Ethics Committee (#PT0151) and informed consent was obtained from all individual participants included in the study. No participants were excluded or withdrew from this study.

PROCEDURES

Participants attended two separate data collections, one week apart. They were instructed to continue with their normal everyday activities, but to refrain from undertaking any unfamiliar physical activity in the week prior to, and between testing occasions. In addition, they were not to undertake vigorous physical activity in the 24 hours prior to testing. Data collection was performed in the motion analysis laboratory at the School of Physiotherapy, Curtin University, Western Australia.

Initially, relevant demographic and anthropometric data was recorded (age, height, body mass, lower limb dominance). Retro-reflective markers were then affixed to the skin of the participants according to a customised marker set and model for the lower limbs and pelvis (see Figure 1), which was in accordance with an established cluster based method

(Besier et al., 2003, Wu et al., 2002). Single markers were placed on the head of the first and fifth metatarsals, calcaneus, anterior superior iliac spines (ASIS) and posterior superior iliac spines (PSIS). Marker clusters (three markers attached to a semi-rigid plastic base plate) were attached to the lateral aspects of both thighs and legs. The three-dimensional position of these markers was tracked using a 14-camera Vicon MX motion analysis system (Vicon, Oxford Metrics, Oxford, UK) operating at 250 Hz. Prior to dynamic trial collection, a static calibration trial was performed using a foot-calibration rig to measure foot abduction/adduction and inversion/eversion angles. The additional use of medial and lateral malleoli and medial and lateral femoral condyle marker locations determined anatomically relevant ankle, knee and hip joint axes of rotation and joint centres (Besier et al., 2003).

Ten 30 second (s) dynamic hopping trials were then completed with a 90 s rest period between trials. The task involved continuous sub-maximal single-limb hopping on a custom-built SJS that included a low-friction sled, reclined to 20° relative to the base which was composed of a 50.2 cm x 50.2 cm force plate (AccuGait; AMTI, Watertown, MA) operated at 1000 Hz (Figure 2). The apparatus has been developed locally to quantify the mechanical properties of the lower limb musculoskeletal structures in vivo during sub-maximal dynamic exercise.

Participants were instructed to keep their non-hopping limb in a flexed position. Their foot rested on the SJS and they held onto the sliding component of the SJS in order to stabilise the thorax and upper limbs. Participants hopped on their non-dominant leg; this was defined as the side opposite to the participants preferred jumping leg as recommended by Flanagan and Harrison (2007). Whilst hopping, participants were instructed to keep the hip and knee as straight as possible, effectively isolating the task to the ankle. They were instructed to hop at a sub-maximal level, described as an effort

they could maintain 'indefinitely'. Given the uniqueness of the task, participants were provided with a demonstration and familiarisation period until they could hop as instructed comfortably; this process typically took 10 minutes. To ensure that hopping performance was consistent for each participant, instructions/feedback were kept consistent, as variations in verbal cues can modulate lower limb stiffness in similar tasks (Arampatzis et al., 2001). This testing protocol was repeated exactly 7 days later.

DATA PROCESSING

The Vicon data was processed using Vicon Nexus motion analysis software (Vicon, Oxford Metrics, Oxford, UK). Data were filtered using a fourth-order low-pass Butterworth filter operating at a cut-off frequency of 20 Hz for the marker trajectories and 50 Hz for the force plate data as determined by residual analysis (Winter, 1990). All lower limb anatomical and joint coordinate systems were calculated in accordance with the standards outlined by the International Standards of Biomechanics (Wu et al., 2002) and have been previously described (Besier et al., 2003).

Data was exported from Nexus for further analysis using a customised LabVIEW program (National Instruments, Austin, TX, 2011). For each hopping trial, ankle dorsi-plantar flexion angle at 3 time points, determined using force plate data (80 ms prior to foot strike, at foot strike, and at take-off), as well as ankle, knee and hip stretch amplitude (range of excursion between contact and maximum dorsiflexion) were calculated. In addition, lower limb stiffness was calculated using the method thoroughly described by Dalleau et al. (2004) (Figure 3). This simple field-measurement has been validated for the study of intra- and inter-individual variations in lower limb stiffness during sub-maximal hopping and is calculated by modelling the ground reaction force as a sine wave as is expected from oscillation of a pure spring-mass model. Support has recently been leant to this assumption by Riese et al. (2013) and enables lower limb stiffness to be calculated

by measuring ground contact time (t_c) and flight time (t_f) for any given hop. This method has been recently validated against reference standards (Grisbrook et al., 2014).

A 30 s epoch was chosen for each block of hopping as pilot testing established that this would ensure at least 30 consecutive hops for all participants. Individual trials were then sub-divided into 3 periods of 10 hops (1-10, 11-20 and 21-30). Individual period values were the lowest level of analysis and a median value of each period was selected for comparison.

STATISTICAL ANALYSIS

Reliability data analyses were performed using Stata (StataCorp. 2011. Stata Statistical Software: Release 12. College Station, TX: StataCorp LP). All other statistical analyses were conducted using SPSS 19 (SPSS, Chicago, IL, USA). Data was checked for normality and values outside of two standard deviations from the mean were considered outliers and were removed for the purposes of statistical analysis as described in previous literature employing SJS's (Ertelt and Blickhan, 2009). On average, 4% of data was removed from each trial, consistent with typical data processing errors experienced using this model (Ertelt and Blickhan, 2009).

Data were compared between the 3 periods, averaged across the 10 trials and 2 testing occasions, using a generalised linear mixed model. An alpha less than 0.05 was utilised to represent statistical significance. Within-trial and within-week reliability was assessed by generating ICC values for each trial. Week was taken into consideration using the variance of intraclass correlation coefficients in three-level model as described by Hedges et al. (2012). In this model, reliability values are attributed to contributing sources (subject and week) for each trial. Previous research (McLachlan et al., 2006, Joseph et al., 2013), supported by our own pilot testing indicated trial 1 may demonstrate poorer stability than trials 2-10; therefore an additional pooled analysis was conducted for trials 1-10 and trials

2-10. ICC values above 0.70 were considered to represent strong reliability; values between 0.50-0.70 were considered to represent moderate reliability and values below 0.50 were considered to represent poor reliability (Portney and Watkins, 2008). Finally a comparison by week was undertaken. According to the model by Hedges et al. (2012), low ICC values represent minimal influence on reliability between weeks and indicate higher reliability. Between-week consistency was also assessed by calculating the standard error of measurement (SEM) and minimal detectable difference (MDD) between weeks.

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