

Musculoskeletal stiffness and Achilles tendon mechanical property changes following exercise-induced muscle damage

Corey William Julian Joseph

Bachelor of Applied Science - Sport and Exercise Science (Honors)

A thesis submitted in total fulfillment of the requirement of the degree
of Doctor of Philosophy

School of Exercise Science
Faculty of Health Science

ACU National
Research Services
Locked Bag 4115,
Fitzroy, Victoria 3065
Australia

February 2012

Statement of sources

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

No parts of this thesis have been submitted towards the award of any other degree or diploma in any other tertiary institution.

No other person's work has been used without due acknowledgement in the main text of the thesis.

All research procedure reported in the thesis received the approval of the relevant Ethics/Safety Committees (where required).

Full Name: Corey William Julian Joseph

Signed:

Date:

Abstract

This thesis investigated the affect of exercise induced muscle damage (EIMD) on musculoskeletal stiffness (MSS), Achilles tendon (AT) stiffness and AT strain. Furthermore, this thesis determined the reliability of a protocol used to measure MSS with the aim to apply this protocol in the investigation of the EIMD associated changes in MSS, AT stiffness and AT strain. Three studies were conducted as part of this thesis.

Study 1: A number of methods are used to measure lower extremity musculoskeletal stiffness (MSS), but there is a paucity of research examining the reliability of these techniques. The aim of study 1 was to assess the reliability of vertical MSS (K_{vert}), leg MSS (K_{leg}), knee MSS (K_{knee}) and ankle MSS (K_{ank}) during over-ground running, hopping and jumping. Twenty active males were required to run on a 10 m over-ground runway at 3.83 m/s, hop in-place at 2.2 Hz and at a self-selected frequency, and jump at 2.2 Hz and at a self-selected frequency. Reliability was determined using the intra-class correlation coefficient (ICC), coefficient of variation ($CV_{\text{ME}}\%$), mean differences (MDiff%), and Cohen's effect sizes (ES). There was good reliability for K_{vert} , moderate reliability for K_{leg} and poor reliability for K_{knee} and K_{ank} during the running task. Similar results were observed during the 2.2 Hz hopping and jumping tasks, with good reliability displayed for K_{vert} and poor reliability for K_{knee} and K_{ank} . In conclusion, our results suggest that K_{vert} is a reliable MSS measure when running at 3.83 m/s and hopping and jumping at 2.2 Hz.

Study 2: Spring energy is used to enhance musculoskeletal performance and minimise the amount of mechanical energy required for movement. However, the eccentric contractions that provide storage and recoil of spring energy can also cause muscle damage. The aim of study 2 was to explore the changes in leg and vertical stiffness associated with an eccentric exercise bout that induces muscle damage. Twenty active males performed a backwards-walking eccentric exercise protocol to induce muscle damage that consisted of 60 minutes of walking at 0.67 m/s on a treadmill at a gradient of -8.5° . Tests were performed immediately before, immediately post, 24, 48 and 168 hours post eccentric exercise. The testing battery included running at 3.83 m/s, and hopping at 2.2 Hz using a single-and a double-legged action. Leg and vertical stiffness were measured from kinetic and kinematic data, and electromyography (EMG) of five muscles of the dominant limb were recorded during hopping. No significant ($P<0.05$) differences in leg or vertical stiffness were observed between baseline and any post exercise time points. No significant ($P<0.05$) differences were found in EMG activity. However, significant ($P<0.05$) differences in knee kinematics during single-legged hopping were observed. These results indicate that knee mechanics may be altered to maintain consistent levels of leg and vertical stiffness when eccentric exercise-induced muscle damage is present in the lower legs.

Study 3: Daily activities and common sporting movements consist of eccentric contractions, which can result in exercise induced muscle damage (EIMD). This may alter the mechanical properties of the musculotendon unit.

The aim of study 3 was to investigate the biomechanical response of the Achilles tendon (AT) aponeurosis following an exercise intervention designed to induce EIMD. Twenty active males performed an eccentric exercise protocol that consisted of 60 minutes of inclined (-8.5°), backwards walking on a treadmill at 0.67 m/s. Tests were performed immediately before, immediately post, 24, 48 and 168 hours post eccentric exercise. The AT elongation was measured using ultrasonography during a ramped maximal voluntary plantar flexor contraction of the dominant limb by tracking the myotendinous junction of the medial gastrocnemius and the Achilles tendon (ATJ). The elongation of the ATJ was used to determine AT strain and AT stiffness was calculated by a ratio of tendon force (from inverse dynamics) and elongation. There were increases in AT stiffness at 24 hours post EIMD ($p = 0.004$) and decreases in AT strain at 24 – 48 hours post EIMD ($p > 0.05$), with no significant change in muscle force production at any time point. These results suggest that the EIMD protocol affected the mechanical properties of musculotendinous unit resulting in either an increase in tendon stiffness and strain or an increase in muscle compliance. This may improve force transmission capabilities and provide a defence mechanism protecting the AT against high levels of strain.

LIST OF PUBLICATION SUBMISSIONS

Joseph, C., Bradshaw, E., Kemp, J. & Clark, R. (Accepted). The inter-day reliability of ankle, knee, leg, and vertical musculoskeletal stiffness during hopping, jumping and over-ground running. *Journal of Applied Biomechanics*.

CONFERENCE PRESENTATIONS

Joseph, C., Bradshaw, E., and Clark, R. (2011). The inter-day reliability of leg, knee and ankle stiffness during hopping. *International Society of Biomechanics in Sports Conference*. University of Porto, Portugal, June 27th – July 1st, p. 989-992.

Joseph, C., Bradshaw, E., and Clark, R. (2010). The inter-day reliability of leg, knee and ankle stiffness during running. *International Society of Biomechanics in Sports Conference*. Northern Michigan University, Marquette, July 19-23, p. 286-89.

Dedication

I would like to dedicate this thesis to my family.

To my grandparents, I am so glad you are here to share this with.

To my mother and father for their unconditional support throughout my endeavours in life, sport and career.

Lastly, I dedicate this to my best friend, travel buddy and pillar of strength, Zara. I wouldn't be who I am today without you. Words can't express.

Acknowledgements

This and the dedication sections have been undoubtedly the hardest parts of this thesis to write. Writing, and certainly emotive writing, is not my strong point and whilst these words acknowledge and offer thanks to a number of people for their guidance and support, my appreciation and gratitude goes beyond the limits of my ability to pen them. To anyone who I may have left out, forgive me but believe me, you are not forgotten. My deepest thanks go to the following –

I would like to sincerely thank my supervisors for their support. Liz, from my introduction to ACU though to the completion of this thesis, I have been fortunate enough to have your supervision throughout. You planted the musculoskeletal stiffness seed and my interest grew from there. You have been an enormous support and I thank you for giving me the freedom and trust to work autonomously and in finding time for me when I really needed it amongst the massive workload you already had. I wish you all the happiness and health with the birth of your first child. Ross, thank you for agreeing to meet me on that summer day in early 2009 and somehow seeing my vision and agreeing to become involved. You have been the link in this project that made it all possible. Your expertise in tendon ultrasonography, technical knowledge in experimental design, and skill and advice in turning my research into something worthy of peer-review has been invaluable. Furthermore, programming is a skill I have always wanted to develop and all your tips and help with LabVIEW have been amazing. Thank you for also considering the

bigger picture for me in my life after this thesis, this I sincerely appreciate. Justin, for all your unconditional time and support and your knowledge in muscle physiology and writing prowess, thank you.

Jeri, I don't think there are many PhD students who could get the unconditional assistance and support of their Professor. Your encouragement, positivity and humility have taught me an enormous amount about research and life. You have been a huge mentor and I can't thank you enough. To my main man, Trent. Where do I begin? Your camaraderie has been tremendous. You have an amazing outlook on life and I look forward to continuing our success together. A marvellous change agent! You and Jeri are both true friends for life.

Many students don't have the luxury of sharing an office with fantastic people who are on the same journey as your self. Dan and Bianca, I wish you both the best of luck with your PhD's and thank you in particular to Bianca for assisting in my data collection when you had more important things to do. I appreciate it.

Thank you to the nursing staff at ACU Melbourne for your help with the venepuncture consumables, and for the tips and encouragement.

Taryn and the staff within the School of Exercise Science at ACU, thank you. In particular Kade, who assisted me in the lab and provided me

with many gems experienced through his thesis write-up. Also to Mark Moresi for our limited but extremely helpful chats.

This would not have been possible without participants and of equal importance, to my helpers, Rob, Tom, Courtney, Lauren, Scott, Chelsea, Paul, and Aaron. Thank you for your time and if I can be of any assistance in the future, please, reach out.

My parents have provided unwavering support and have always put my brothers and I first, even when they couldn't afford to. My father has taught me to have an incredible hardened work ethic and my mother has given me an amazing gift of tolerance. These are qualities that have made it possible for me to complete and endure the ups and downs of this PhD. To Zara, your support, strength and belief in me has been astounding. Thank you for putting up with the 3am nights while I was in the final preparations of this work, I know we will look back and laugh about these times. You are amazing and I love you very much. And finally, to my brothers and friends, who understand only that I have been completing my doctorate and won't make any sense out of what lines these pages, thanks for your support.

Table of contents

STATEMENT OF SOURCES	ii
ABSTRACT	iii
LIST OF PUBLICATIONS	vi
DEDICATION	vii
ACKNOWLEDGEMENTS	viii
TABLE OF CONTENTS	xi
LIST OF TABLES	xix
LIST OF FIGURES	xxi
GLOSSARY OF ABBREVIATIONS	xxiv
CHAPTER 1. GENERAL INTRODUCTION	1
1.1 Statement of the problem	1
1.2 General aims of the study	4
1.3 Specific aims of the study	5
1.4 Hypotheses	5
1.5 Limitations	6
1.6 Delimiters	7
CHAPTER 2. LITERATURE REVIEW	8
2.1 Musculoskeletal injury	8
2.1.2 Causes of injury	9
2.1.2.1 Load and loading characteristics	12
2.1.2.1.a Direction	13
2.1.2.1.b Magnitude	15
2.1.2.1.c Type	17
2.1.2.1.d Loading rate	19

2.1.2.1.e	Frequency	20
2.1.3	Biological properties of muscle and tendon	22
2.1.3.1	Muscle	23
2.1.3.2	Tendon	30
2.1.4	Summary	33
2.2	Musculoskeletal stiffness and tendon mechanics	34
2.2.1	Measurement and calculation of global MSS	37
2.2.1.1	Vertical MSS	38
2.2.1.2	Leg MSS	43
2.2.1.3	Joint MSS	46
2.2.2	MSS and performance	48
2.2.2.1	Hopping	51
2.2.2.2	Jumping	53
2.2.2.3	Running	54
2.2.3	Injury and MSS	56
2.2.4	Measurement and calculation of tendon stiffness	59
2.2.4.1	Methods used to measure tendon loads	60
2.2.4.2	Ultrasonography of the muscle-tendon structures	61
2.2.5	Tendon mechanics and performance	65
2.2.6	Summary	67
2.3	Injury and exercise induced muscle damage	67
2.3.1	Proposed mechanisms underlying EIMD	71
2.3.1.1	Contraction type	72

2.3.1.2	Mechanical factors	73
2.3.2	Indicators of damage	75
2.3.2.1	Creatine kinase activity	75
2.3.2.2	Muscular strength	77
2.3.2.3	Delayed onset muscular soreness	78
2.3.3	Exercise models used to illicit EIMD	79
2.3.4	EIMD and sports performance	83
2.3.4.1	Joint kinematics	83
2.3.4.2	Neuromuscular control	84
2.3.4.3	Strength and power	85
2.3.4.4	Functional measures of performance	85
2.3.5	Summary	86
2.4	Chapter summary	87
CHAPTER 3.	METHODS	90
3.1	Ethics approval	90
3.2	Participant recruitment	90
3.2.1	Inclusion criteria	90
3.2.1.1	Chapter 4 inclusion criteria	90
3.2.1.2	Chapter 5 & 6 inclusion criteria	91
3.3	Methods and procedure	92
3.3.1	Participant screening	92
3.3.1.1	Medication	93
3.3.1.2	Self reported medical and surgical history	93

3.3.1.3	Anthropometric data	93
3.3.1.4	Supplement usage	93
3.3.1.5	Eccentric exercise history	94
3.3.2	Laterality assessment	94
3.3.3	Hopping and jumping assessment	95
3.3.4	Running assessment	96
3.3.5	Eccentric exercise protocol	97
3.3.6	Muscle damage assessment	97
3.3.7	Experimental set-up and isometric plantar flexion procedure	98
3.3.8	Determination of muscle-tendon junction position	101
3.4	Data analysis	104
3.4.1	Musculoskeletal stiffness analysis	105
3.4.2	EMG collection and analysis	106
3.4.3	Achilles tendon moment arm length and Achilles tendon thickness	112
3.4.4	Achilles tendon force analysis	114
3.4.5	Achilles tendon strain and stiffness	114
3.5	Statistical analysis	118
3.5.1	Statistical power	118

CHAPTER 4. STUDY 1

The inter-day reliability of ankle, knee, leg, and vertical musculoskeletal stiffness during hopping, jumping and over-ground running.	120
---	------------

4.1	Introduction	120
4.1.1	Aims and hypothesis	121
4.2	Methods and procedure	121
4.2.1	Participants	122
4.2.2	Procedure	122
4.2.3	Data collection and analysis	124
4.2.4	Statistics	125
4.3	Results	126
4.4	Discussion	134
4.4.1	Conclusion	138

CHAPTER 5. STUDY 2

Eccentric exercise-induced alterations of lower limb stiffness during dynamic tasks.

5.1	Introduction	139
5.1.1	Aims and hypothesis	141
5.2	Methods and procedure	141
5.2.1	Participants	142
5.2.2	Procedure	142
5.2.3	Data Collection and Analysis	145
5.2.4	Statistics	146
5.3	Results	146
5.3.1	Muscle damage markers	146
5.3.1.1	MSS	148

5.3.1.2	EMG	151
5.3.1.3	Joint kinetics and kinematics	154
5.4	Discussion	157
5.4.1	Conclusion	160

CHAPTER 6. STUDY 3

Behaviour of the Achilles tendon *in-vivo* following exercise induced

muscle damage		161
6.1	Introduction	161
6.1.1	Aims and hypothesis	164
6.2	Methods	165
6.2.1	Participants	165
6.2.2	Procedure	166
6.2.2.1	Eccentric exercise protocol and muscle damage indicators	167
6.2.2.2	Ultrasound experimental set-up and isometric plantar flexion procedure	168
6.2.3	Data Collection and Analysis	168
6.2.3.1	Achilles tendon moment arm length and Achilles tendon thickness	169
6.2.3.2	Determination of muscle-tendon junction position	170
6.2.3.3	Achilles tendon force	171
6.2.3.4	Achilles tendon strain and stiffness	171

6.2.4	Statistics	172
6.3	Results	173
6.4	Discussion	176
6.4.1	Conclusion	182
 CHAPTER 7. FINAL DISCUSSION		 184
7.1	Introduction and general overview	184
7.1.1	Thesis sequence	184
7.2	Summary of major findings	185
7.3	Discussion	188
7.3.1	The inter-day reliability of K_{ank} , K_{knee} , K_{leg} and K_{vert} , during hopping, jumping and over-ground running	188
7.3.2	New insights into the effect of DOMS on the behaviour of the musculoskeletal system.	190
7.4	Future directions	193
7.5	Concluding remarks	195

APPENDICES	196
Appendix A Informed consent form	196
Appendix B Information letter to participants (Study 1)	199
Appendix C Information letter to participants (Study 2 & 3)	202
Appendix D Health screening form	205
Appendix E Calculation of vertical displacement	213
Appendix F Dalleau and colleagues (2004) method of calculating K_{vert}	216
Appendix G Exemplars of raw data used to calculate vertical and joint MSS	219
REFERENCES	222

LIST OF TABLES

Table 2.1.	Summary of formulas for calculating MSS.	39
Table 2.2.	Summary of MSS and running performance.	49
Table 2.3.	Summary of MSS and hopping and jumping performance.	30
Table 4.1.	Musculoskeletal stiffness reliability statistics and overall determination of reliability during the running task.	127
Table 4.2.	Musculoskeletal stiffness reliability statistics and overall determination of reliability for continuous jumping and hopping	128
Table 4.3.	Kinetic, kinematic and VGRF results during the jumping (CJ) and hopping (CH) tasks using both legs.	131
Table 4.4.	Kinetic, kinematic and VGRF results during the jumping (CJ) and hopping (CH) tasks using single legs.	132
Table 4.5.	Kinetic, kinematic and VGRF results during the running (RUN) task.	133
Table 5.1.	Double-legged hopping EMG activity in the dominant limb.	152
Table 5.2.	Single-legged hopping EMG activity in the dominant limb.	153
Table 5.3.	Kinematic and kinetic results in the dominant (DOM) and non-dominant (ND) legs during the hopping tasks (double and single legged) at 2.2 Hz across the five testing sessions (Pre, Post, 24 hr, 48 hr and 168 hr).	155
Table 5.4.	Kinematic and kinetic results in the dominant (DOM) and non-dominant (ND) legs during the running task across the 5 testing sessions (Pre, Post, 24 hr, 48 hr and 168 hr).	156

Table 6.1. Plantar flexor force and muscle activity (EMG) measurements during MVC across the 5 testing sessions (Pre, Post, 24 hr, 48 hr and 168 hr). 175

LIST OF FIGURES

Figure 2.1. A model of the interaction between intrinsic and extrinsic risk factors and injury.	10
Figure 2.2. Risk factors for injury.	11
Figure 2.3. Examples of the directions that loads can be applied to a system.	14
Figure 2.4. Stress-strain curves of musculoskeletal tissue under tensile loading.	16
Figure 2.5. Compression (a), tension (b) and shear loading (c).	18
Figure 2.6. Fatigue curve showing the relationship between loading, frequency and injury risk.	21
Figure 2.7. Stress-strain curve of tendon showing the regions of the curve that represent a different mechanical characteristic.	23
Figure 2.8. Schematic diagram of skeletal muscle composed of a number muscle fascicles.	24
Figure 2.9. An isolated myofibril marked where a single sarcomere lies (top).	25
Figure 2.10. Examples of fibre pennation angle (black line).	26
Figure 2.11. Organisation of the sarcomere during a relaxed and contracted state.	27
Figure 2.12. An example of the force-length relationship of a sarcomere (middle).	28
Figure 2.13. The force-velocity relationship of skeletal muscle.	29
Figure 2.14. A specimen of normal tendon.	30

Figure 2.15. The mechanical properties of tendon.	31
Figure 2.16. The spring-mass system used to model vertical MSS.	36
Figure 2.17. Spring-mass model showing a single linear leg spring representing the mechanical behaviour of the musculoskeletal system during ground contact.	40
Figure 2.18. The leg model used to calculate leg MSS during ground contact in non-linear movements such as running.	45
Figure 2.19. Multi-jointed model indicating the hip, knee and ankle joints and their torsional springs.	46
Figure 2.20. Three-element Hill-type muscle model of the medial gastrocnemius and Achilles tendon (A).	62
Figure 2.21. Shows the anatomical landmarks of the gastrocnemius and Achilles tendon.	63
Figure 2.22. Longitudinal ultrasonography image of the triceps surae muscle during resting conditions, 1000N and 2000N of force.	64
Figure 2.23. Muscle strain injury.	67
Figure 2.24. A model of the relationship between exercise and injury.	70
Figure 3.1. The customised squat rack in detail.	100
Figure 3.2. Anatomy of the gastrocnemius and Achilles tendon.	102
Figure 3.3. The custom built brace moulded to the shape of the ultrasound probe and to the shape of an average gastrocnemius muscle.	103
Figure 3.4. EMG electrode placement.	108
Figure 3.5. An example of the time points at which EMG was analysed during hopping.	109

Figure 3.6.	Justification for EMG filtering technique.	111
Figure 3.7.	Sagittal plane ultrasound image of the Achilles tendon, skin and calcaneus.	113
Figure 3.8.	Sagittal plane ultrasound images over the distal myotendinous junction of the medial gastrocnemius muscle for one subject.	115
Figure 3.9.	Calculation for the displacement of the Achilles tendon junction that occurs during ankle joint rotation.	117
Figure 5.1.	Changes (mean \pm SD) in A: Muscle soreness, B: Maximal voluntary isometric contraction in the plantar flexor muscles of the dominant limb, and C: Plasma CK activity at each time point between baseline (Pre) and 7 days (168 hours) post eccentric exercise protocol.	148
Figure 5.2.	Changes in A: K_{Vert} in the dominant and non-dominant legs during the running task, B: K_{Leg} in the dominant and non-dominant legs during the running task, and C: K_{Leg} in the double-legged (Both) and single-legged (Dominant and Non-dominant) hopping task between baseline (Pre) and 7 days (168 hours) post eccentric exercise protocol.	150
Figure 6.1.	Changes (mean \pm SD) in A: Achilles tendon stiffness, B: Achilles tendon strain, and C: Achilles tendon force at each time point between baseline (Pre) and 7 days (168 hours) post eccentric exercise protocol.	174

GLOSSARY OF ABBREVIATIONS

AT	Achilles tendon
ATJ	Achilles tendon junction
BGA	Background electromyography activity
BW	Body weight
CH	Continuous hopping
CH2.2	Continuous hopping at 2.2 hertz
CHSS	Continuous hopping at self-selected frequency
CJ	Continuous jumping
CJ2.2	Continuous jumping at 2.2 hertz
CI	Confidence interval
CK	Creatine kinase
COM	Centre of mass
CSA	Cross-sectional area
CV	Coefficient of variation
CV_{ME}%	Coefficient of variation as a percentage of measurement error
EMG	Electromyography
EIMD	Exercise-induced muscle damage
ES	Effect size
F	Force
hr	Hours
Hz	Hertz
ICC	Interclass correlation
K_{knee}	Knee musculoskeletal stiffness

K_{leg}	Leg musculoskeletal stiffness
kN	Kilonewtons
kN/kg/m	Kilonewton per kilogram per meter
K_{vert}	Vertical musculoskeletal stiffness
LG	Lateral gastrocnemius muscle
MG	Medial gastrocnemius muscle
M1	Short-latency reflex electromyography activity
M2	Long-latency reflex electromyography activity
m	Meter
Mdiff%	Mean difference as a percentage
MHz	Megahertz
mm	millimeter
m/s	meters per second
MSS	Musculoskeletal stiffness
mV	Millivolts
MVC	Maximal voluntary contraction
Nm	Newtons per meter
Nm/kg	Newtown meters per kilogram
N/mm	Newtons per millimeter
PL	Peroneus longus muscle
PRE	Pre-contact electromyography activity
RUN	Running task
SD	Standard deviation
s	Seconds
SEM	Standard error of measurement

SOL	Soleus muscle
SPSS	Statistical package for the social sciences
SS	Self-selected
RMS	Root mean squared
TA	Tibialis anterior muscle
VAS	Visual analogue scale
VGRF	Vertical ground reaction force
VL	Vastus lateralis muscle

1. General introduction

1.1 Statement of the problem

Injury can result when a load applied to musculoskeletal tissue exceeds its failure tolerance (Bartlett, 1999, p. 3). In a sporting context, it can be defined by any injury resulting from participation in a sport or exercise that causes either a reduction in activity or a need to seek medical advice (Brooks & Fuller, 2006). In 2006, approximately 25% (5.2 million) of Australians suffered a sports related injury (Medibank, 2006). These injury figures translated to a \$2 billion cost to the Australian community per year (Medibank, 2006).

National sports injury figures indicate that 57% of all sporting injuries in Australia occur to the lower limbs (ABS, 2006), with muscle-tendon injuries being most common in sports that involve high intensity efforts (Flood & Harrison, 2009; Orchard, Best, & Verrall, 2005). However, despite an increasing awareness of the high occurrence of lower limb muscular strain and tendinopathy, the precise mechanisms that cause these injuries are unknown (Finch & Orchard, 2002).

Muscle damage or injury brought upon by unaccustomed eccentric exercise may be the most common injury resulting from athletic activity (Cheung, Hume, & Maxwell, 2003). The mechanism that causes delayed onset muscle soreness (DOMS) following exercise induced muscle damage (EIMD) is unclear, with a combination of factors being suggested (Armstrong, Warren, & Warren, 1991; Cleak & Eston, 1992; Smith, 1991). This spectrum of mechanisms include lactic acid theory, connective tissue damage, inflammation, enzyme efflux theory and muscle spasm (Cheung, et al., 2003).

The symptoms associated with this type of muscle damage have helped to shed light on the potential causes of the damage. Common symptoms following EIMD include reductions in force production capability, increases in inflammation, increases in plasma creatine kinase levels, increases in muscle soreness, and reductions in joint range of motion (Clarkson & Hubal, 2002). Early investigations into muscle contractile activity have demonstrated that eccentric contractions result in a greater magnitude of EIMD than concentric or isometric contractions (Newham, McPhail, Mills, & Edwards, 1983).

With respect to muscle force production, concentric contractions occur when a muscle shortens, while an eccentric contraction occurs when the muscle elongates under tension. Physical activity involves both eccentric and concentric contractions and the combination of these muscle actions is known as the stretch-shortening cycle (SSC). During the SSC, elastic-strain energy is stored throughout the active, eccentric contraction phase and then recovered during the subsequent concentric contraction phase (Komi, 2000). The high level of shock-absorbing, negative mechanical work that the muscle-tendon unit performs during the eccentric component of the SSC can cause damage within the muscle-tendon unit (Newham, Jones, Ghosh, & Aurora, 1988). Furthermore, performance can be enhanced by SSC exercise and this is partly attributed to the ability of the muscle-tendon unit to store and reuse elastic strain energy (Komi & Bosco, 1978). The musculoskeletal system possesses the ability to modulate the level of spring energy required for athletic performance, and musculoskeletal stiffness (MSS) regulates the storage and reuse of elastic energy.

MSS is defined as the collective ability of muscles, tendons, ligament, cartilage and bone to resist deformation from an applied force (Kuitunen, Avela, Kyrolainen, Nicol, & Komi, 2002). A growing body of research has been able to establish clear links between musculoskeletal stiffness and sports performance (see reviews by Brughelli & Cronin, 2008a, 2008b; Butler, Crowell, & McClay-Davis, 2003). Observations have emerged from retrospective studies that identify MSS as a potential mechanism for lower limb sporting injuries (Bradshaw, Le Rossignol, Williams, & Lorenzen, 2006). However, only one study has attempted to *prospectively* link injury with musculoskeletal stiffness (Watsford, et al., 2010) whilst the mechanisms related to tendon injury are well known (Maganaris, Narici, Almekinders, & Maffulli, 2004).

The function of human tendon is two-fold: (1) to transmit contractile force to create movement about a joint, and; (2) to store and reuse elastic strain energy (Ker, Alexander, & Bennett, 1988). The Achilles tendon is the strongest tendon in the body and specialises in the storage and reuse of elastic energy (Komi, Fukashiro, & Jarvinen, 1992). The Achilles tendon has the ability to adapt to loading and unloading over time (Kubo, et al., 2004a; Kubo, Tabata, Yata, Tsunoda, & Kanehisa, 2010). The location of the Achilles tendon makes it responsible for withstanding high forces and, therefore, high magnitudes of strain. However, high levels of strain in the Achilles tendon can result in degradation and injury (Rolf & Movin, 1997). Past studies have explored the effect of repeated strain on the mechanical behaviour of the Achilles tendon by altering strain rate, contraction mode and contraction duration (Arampatzis, Karamanidis, & Albracht, 2007; Duclay,

Martin, Duclay, Cometti, & Pousson, 2009; Mademli, Arampatzis, & Walsh, 2006). The mechanical and morphological behaviour of tendon following mechanical loading can alter the force generating capacity of contractile muscle by affecting the force-length-velocity relationship (Ettema, 1996). Furthermore, mechanical loading may affect tendon health and cause degradation (Arya & Kulig, 2010; Butler, Grood, & Noyes, 1978). Therefore, understanding the response of tendon to mechanical loading may improve the process of tendon anabolism via adaptation or healing. Nonetheless, only one study has attempted to *prospectively* link injury with Achilles tendon stiffness (Arya & Kulig, 2010).

The effects of repetitive, unaccustomed, eccentric contractions on muscle function and neuromuscular control are well established. However, there is a lack of empirical evidence investigating the effect of eccentric EIMD on the function of the muscle-tendon unit to absorb mechanical energy and store and reuse elastic strain energy. Therefore, there is merit in exploring the relationship between EIMD and MSS, tendon stiffness and tendon strain in order to better understand their roles in performance and injury.

1.2 General aims of the study

The general aim of this study was to investigate whether there is an association between EIMD and MSS and Achilles tendon stiffness and Achilles tendon strain. Understanding this association allows us to advance the knowledge in athletic performance, injury risk and injury prevention. To achieve this, three studies with the following objectives were proposed.

1.3 Specific aims of the study

The specific aims of this study were to (in active males):

1. Investigate and establish the reliability of musculoskeletal stiffness measures during running and hopping tasks, particularly when performed at a self-selected velocity or frequency respectively. This was to be explored during a laboratory-based biomechanical measurement that assessed vertical, leg, knee and ankle joint MSS during running and hopping tasks in active males.
2. Investigate over 5 testing sessions (immediately before, immediately post, 24, 48 and 168 hours post induced damage) the eccentric exercise associated changes leg and vertical MSS (during hopping) following a downhill backwards-walking protocol.
3. Investigate over 5 testing sessions, (immediately before, immediately post, 24, 48 and 168 hours post induced damage) whether Achilles tendon stiffness and Achilles tendon strain are altered as a result of the EIMD caused by a downhill backwards walking protocol.

1.4 Hypotheses

The following hypotheses were forwarded:

- (i) MSS measures (vertical, leg) of running and hopping at a set pace (velocity or frequency) are reliable, whereas the same tasks performed at a self-selected pace exhibit reduced reliability.

- (ii) Joint MSS measures (ankle and knee) of running and hopping at either a set or self-selected pace are less reliable than global measures due to increased task performance variation in joint movement patterns.
- (iii) There is a reduction in leg MSS, compared to baseline, following eccentric muscle damage and that these changes are associated with changes in ankle kinematics and muscle activity during the hopping task.
- (iv) There is no change in leg MSS, knee and ankle joint kinematics, and muscle activity during the running task.
- (v) Achilles tendon stiffness is reduced following exercise induced muscle damage.
- (vi) Achilles tendon strain is increased following exercise induced muscle damage.

1.5 Limitations

The following may be recognised as limiting factors in the research conducted in this thesis:

- (i) The volume, intensity and type of training routinely undertaken by each participant varied according to the type of sport or physical activity they were commonly involved in.
- (ii) Nutrition status of each participant
- (iii) Motivational and psychological status of each participant
- (iv) Influence of genetic factors or ethnicity
- (v) The design of this study limits the ability to generalise the findings to only young adult, active males only.

- (vi) The type of activity that participants performed prior to the initial testing period. However, participants were required to abstain from any form of activity after the eccentric exercise protocol.
- (vii) Tendon behaviour of this study is limited to measurements taken *in vivo*.
- (viii) The mechanical property changes in the AT of this study could be a result of the either modulation or changes in the stiffness of the tendon, muscle fascicle or the aponeurosis. We did not account for changes in the aponeurosis or muscle.

1.6 Delimiters

Due to the presence of limiters, this thesis was delimited to the study of:

- (i) Male participants aged 18 to 26 years;
- (ii) Participants meeting all inclusion criteria and passing all screening criteria prior to recruitment;
- (iii) Common methodology used to measure muscle damage;
- (iv) Common methodology used to measure MSS;
- (v) Common methodology used to measure tendon displacement and account for joint rotation;
- (vi) Participants not consuming any nutritional supplements, ibuprofen, anti-inflammatory or pain relief medication, and avoiding massage, contrast therapy or stretching, to prevent any influence of other recovery interventions on the results of the studies.
- (vii) The use of backwards walking as the stimulus for EIMD.

2. Literature Review

This chapter is comprised of three sections that will critically review the literature of each topic. The first section will review the loads common to the musculoskeletal system during athletic performance and the causes of injury as a result of these loads (Chapter 2.1). The second section will review musculoskeletal stiffness (Chapter 2.2), and the third part will cover exercise-induced muscle damage (Chapter 2.3).

Searches of electronic databases were conducted for all papers published from inception until February 2012. The electronic databases that were searched were PubMed, SPORTDiscus, MEDLINE, CINAHL, ISI Web of Science and Google Scholar. Search terms used included 'load', 'adaptation', 'stiffness', 'musculoskeletal', 'musculotendinous', 'leg stiffness', 'vertical stiffness', 'quasi-stiffness', 'eccentric', 'eccentric exercise', 'eccentric muscle damage', 'injury', 'DOMS', 'delayed onset muscle soreness', 'muscle', 'muscle damage', 'muscle soreness', 'tendon', 'tendon behaviour', 'tendon stiffness', 'tendon strain', 'human', 'gastrocnemius', 'Achilles tendon', 'triceps surae', 'muscle-tendon complex', 'muscle-tendon unit', 'muscle-tendon' 'mechanics', 'ultrasound'. There was no restriction on publication date.

2.1 Musculoskeletal injury

Musculoskeletal injury can result when a load applied to body tissue exceeds its failure tolerance (Bartlett, 1999, p. 3). However, in a sporting

context, it can be defined as any injury that results from participation in a sport or exercise that causes either a reduction in activity or a need to seek medical advice (Brooks & Fuller, 2006). Injury affects all types of tissues that make up the musculoskeletal system and includes; muscle, bone, tendon, ligament, and cartilage. As will be explained later in this chapter, each tissue within the body displays its own biomechanical properties and responds differently to loading. Where sports participation is concerned, the loading applied to and by the body often result in musculoskeletal injury and therefore, it is important to understand the mechanisms that cause injury so that risk of injury can be minimised.

2.1.2 Causes of injury

Physical activity has many health benefits and leads to a number of chemical and biological adaptations within the musculoskeletal system (Maffulli & King, 1992; Penedo & Dahn, 2005). However, the stresses or loads the body undergoes during movement can reach a threshold at which adaptations no longer take place and injury occurs (McGinnis, 2005, p. 348) (Figure 2.1).

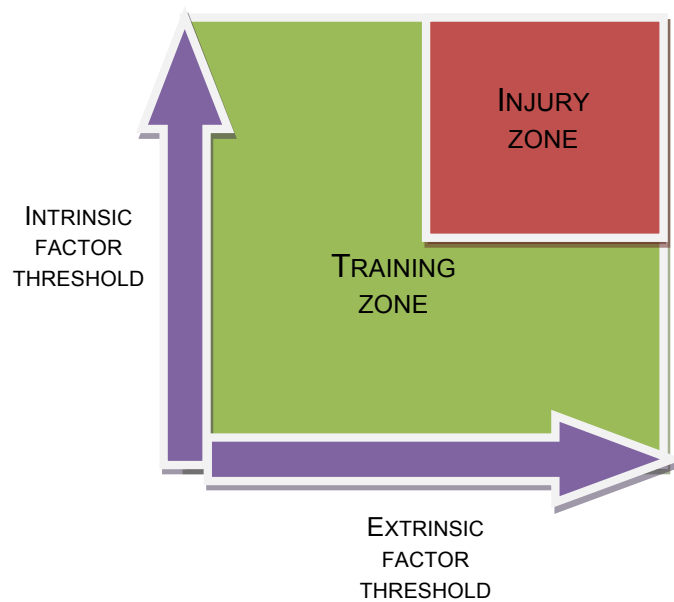


Figure 2.1. A model of the interaction between intrinsic and extrinsic risk factors and injury. Diagram adapted from Messier et al. (1991), as cited in McGinnis (2005, p. 348).

Intrinsic and extrinsic factors are two groups of risk factors that directly influence the occurrence and frequency of injury. These factors not only act in isolation during human movement, but they act in concert, increasing injury risk (McGinnis, 2005, p. 348). Intrinsic factors can be categorised as biological or psychological factors that individually make up and distinguish one person from the other. Extrinsic factors are those related to external, environmental conditions and the manner in which the load is applied to the human body (Bahr & Krosshaug, 2005) (Figure 2.2).

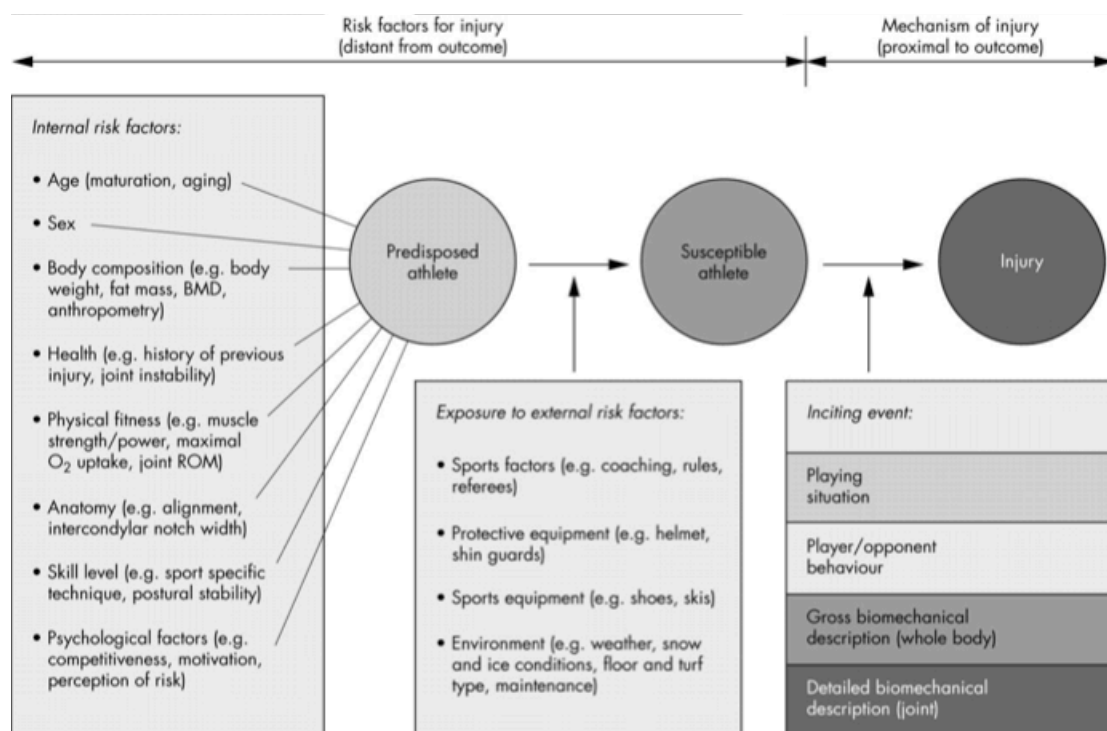


Figure 2.2. Risk factors for injury. Diagram from Bahr & Krosshaug (2005, p. 327).

One example of the interaction between internal and external risk factors is ankle sprain injuries in female volleyball players. Ankle sprain injuries are one of the most common sporting injuries in female volleyball players and are mainly caused by landing on the foot of a team mate or opponent (Bahr & Bahr, 1997). Gender has been shown to be a risk factor due differences in mechanics and neuromuscular control (Krosshaug, et al., 2007) therefore, in this example, the player's gender is the intrinsic risk factor and the playing environment (crowded court area) is the extrinsic risk factor. This is an example of an acute type of injury that occurs from a single, isolated incident however, chronic exposure to intrinsic and extrinsic risk

factors may also lead to injury. For example, previous investigations into ankle injury risk factors have shown that a relationship exists between taping or bracing and previous injury history (Olmsted, Vela, Denegar, & Hertel, 2004), and that the interaction between these two factors influences injury risk whereby, taping and bracing benefits those with a history of ankle sprain. In that example the previous injury history is the internal risk factor and the use of taping or bracing is the external risk factor.

As alluded to, during sports participation, loading the musculoskeletal system causes stresses on the biological tissues that make up the human body. However, there is a fine balance in the relationship between loading, adaptation, and recovery, whereby loading without adequate time for recovery and adaptation can lead to injury (Bahr & Krosshaug, 2005). The characteristics of internal and external loads also have an important effect on injury as a result of their effect on the biological responses within human tissue. Therefore, understanding the effect of different, unique types of loading on the musculoskeletal system enables insight into the possible mechanisms of injury.

2.1.2.1 Load and loading characteristics

The effects of loading on the human body can positively and negatively influence the musculoskeletal system. Positive effects include normal growth and maturation and increased functional capacity of the body's tissues

(Strong, et al., 2005), as well as increased bone mineral density and reduced risk of osteoporosis later in life (Sinaki, et al., 2010). However, negative effects can also occur such as abnormal growth and bone fracture (Hills, King, & Armstrong, 2007; McNair, Prapavessis, & Callender, 2000). The nature and severity of injury is influenced by a range of factors that characterise the applied load and these characteristics include; load direction, load magnitude, type of load, and the rate and frequency of loading (Whiting & Zernicke, 1998, p. 58).

2.1.2.1.a Direction

Human movement causes many different internal and external load patterns on the musculoskeletal system. These loads do not always act in the same directions and are therefore, categorised into; linear loads (a), parallel loads (b), concurrent loads (c), general loads (not a, b or c) (d), and force couple loads (parallel and opposite) (e), as illustrated in Figure 2.3.

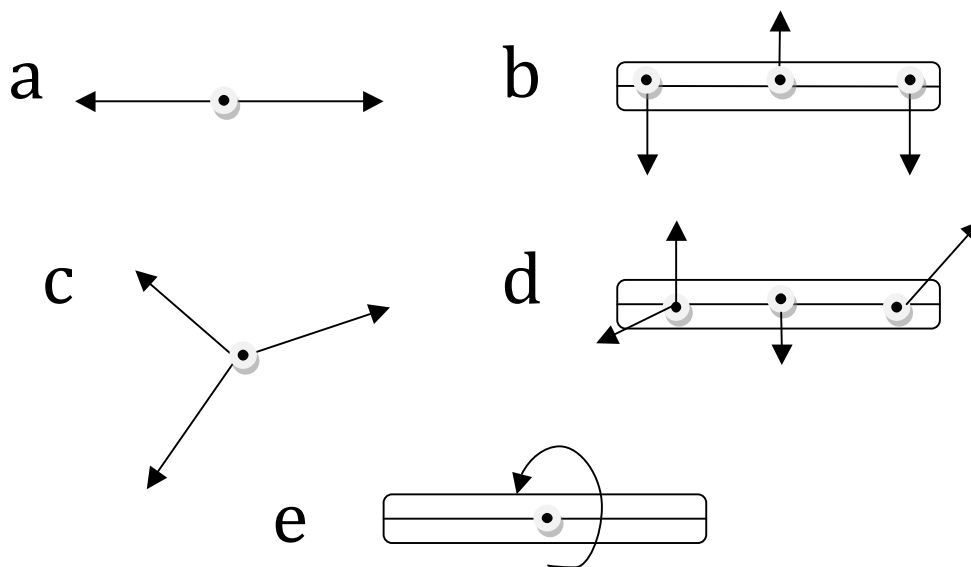


Figure 2.3. Examples of the directions that loads can be applied to a system. Diagram from Whiting & Zernicke (1998, p. 59).

The directions by which loads are applied to systems are important because they directly influence the type of loading undergone by the system (tension, compression or shear). The directions of loads applied determine how each biological tissue responds to the loading and therefore, their risk of failure (Whiting & Zernicke, 1998, p.60). Furthermore, loads that act in the same axis are called uniaxial loads (Figure 2.3a), loads that act on multiple axis are called multi-axial loads (Figure 2.3b, 2.3c, 2.3d) and loads that act in directions which cause twisting are called bending or torsional loads (Figure 2.3e) (Whiting & Zernicke, 1998, p. 87).

2.1.2.1.b Magnitude

A direct relationship exists between the magnitude of load and the failure of a structure (Fung, 1993, p. 25). Biological tissues such as bone, muscle, cartilage, ligament, tendon and muscle can withstand different magnitudes of loads and this depends on the characteristics of each biological tissue (Ashby, Gibson, Wegst, & Olive, 1995). These characteristics will be discussed later in this chapter however; the greater the magnitude of load on a material, the greater the amount of deformation occurs and, if this magnitude of load exceeds the strength of a material, it will fail (Fung, 1993, p. 34). Failure of a biological tissue of the musculoskeletal system results in injury and figure 2.4 shows an example of the different mechanical responses and failure levels of tissue under loading.

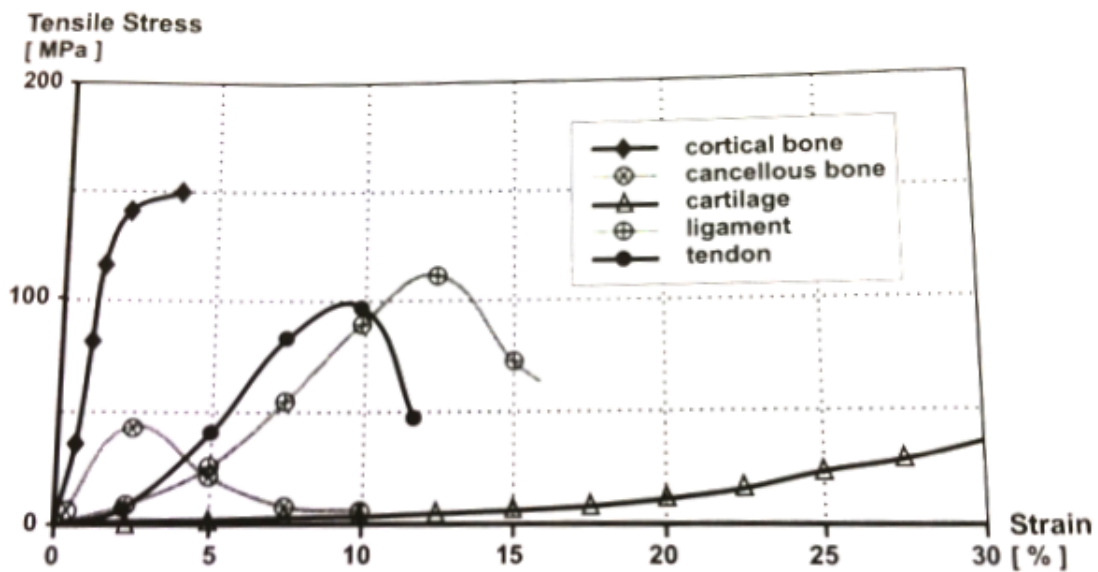


Figure 2.4. Stress-strain curves of musculoskeletal tissue under tensile loading. Diagram from Nigg (2000, p. 287).

An example of this is commonly seen in running where overuse injuries are caused from anatomical or biomechanical factors that lead to high magnitudes of impact forces experienced during repetitive ground impact (Hreljac, Marshall, & Hume, 2000). This high level of impact forces have been shown to cause overuse injuries (Cavanagh & LaFortune, 1980), and stress fractures in runners (Grimston, Nigg, Hanley, & Engsberg, 1993).

Although the magnitude of loading alone can cause injury, failure of a biological material may also occur when a material is stretched, compressed or bent beyond its failure limit. Therefore, the type of load applied to the musculoskeletal system is also an important mechanism of injury.

2.1.2.1.c Loading Type

All biological tissues deform when subjected to loading (Burr, 2011; Humphrey, 2003). The deformation caused by loading a biological tissue is a result of three types of mechanical loading; tension, compression and shear (Figure 2.5), and these are all commonly experienced during human movement.

Compression occurs when a load is acting to compress the molecules of the material together and can occur during weight bearing gravity or muscle force production (Hamill & Knutzen, 2009, p. 40). Load applied to the ends of a material causes it to shorten in the direction the load is applied (Figure 2.5a). Common compressive forces experienced in sports participation have been found to result in spinal fracture (Leucht, Fischer, Muhr, & Mueller, 2009) and muscle contusion (Feeley, et al., 2008), and cartilage damage (Yeow, Cheong, Ng, Lee, & Goh, 2008).

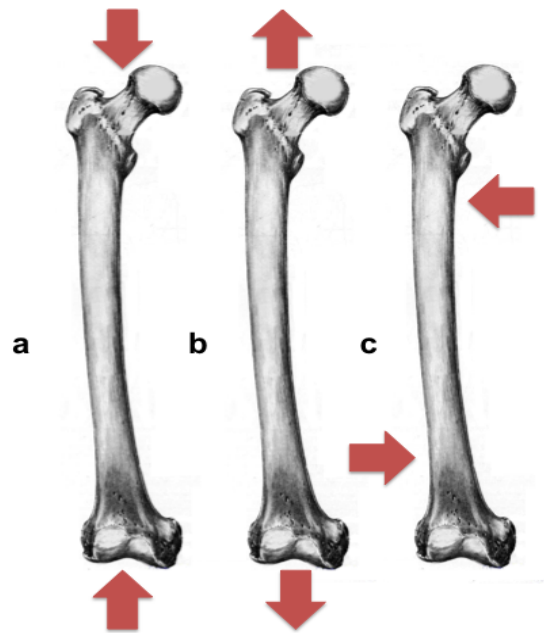


Figure 2.5. Compression (a), tension (b) and shear loading (c).

Alternately, tensile stresses are caused in a material by pulling apart the molecules that bond a material together (Hamill & Knutzen, 2009, p. 42) (Figure 2.5b). Tension occurs when the load is applied along the longitudinal axis where each end of the material is pulled, and as a result, deforms by stretching or elongating in the direction of the applied load. Common sporting injuries caused by tensile loads include tendon rupture (Maffulli, Wong, & Almekinders, 2003), muscle strain (Orchard, 2001), ligament tears (Kirkendall & Garrett, 2000) and bone fracture (Nattiv & Armsey Jr, 1997).

The aforementioned loads are axial and act longitudinally to a material however, shear loads act parallel. Shear loading causes molecules in the material to slide apart past each other and rather than stretching or squashing it causes skewing of the orientation of the material (Hamill & Knutzen, 2009,

p. 43) (Figure 2.5c). Tendon and bone injuries are a common result of shearing loads particularly because they are fragile loads applied parallel to their fibre direction (Braun, et al., 2010; Gluck, Bendo, & Spivak; Turner, Wang, & Burr, 2001). These types of injury commonly occur during landing (Wang, Gu, Chen, & Chang, 2010) or direct impact caused by equipment or other players (Emery, Hagel, Decloe, & Carly, 2010). Whilst the type of load is important, the rate at which the load is applied is another important loading characteristic that effects musculoskeletal adaptation and injury.

2.1.2.1.d Loading rate

Along with the magnitude of loads that are applied to the musculoskeletal system, the rate by which the loading occurs also influences injury risk (Hreljac, et al., 2000). Loading rate refers to the relationship between force and the duration over which the force is applied, and will determine how a biological tissue mechanically responds to that applied force (Ewers, Jayaraman, Banglmaier, & Haut, 2000). For example, it requires more energy to break a bone in a short time than in a relatively long time and landing is an example of a high energy event due to the large magnitude of impact force that is applied over a short period of time (Bartlett, 1999, p. 41). Evidence of the effect of loading rate on injury exists in sports such as running (Hreljac, et al., 2000), weight lifting (Zernicke, Garhammer, & Jobe), volleyball (Bisseling, Hof, Bredeweg, Zwerver, & Mulder, 2007) and Australian rules

football (Schache, Wrigley, Baker, & Pandy, 2009). A jump landing causes ground reaction impact forces to exceed 10 to 12 times body weight in less than 10 ms (Hreljac et al., 2000) whilst repetitive foot striking in running results in 2 to 4 times BW (Bennell, et al., 2004). The difference between the two is the latter results in stress fracture injury. Furthermore, whilst the timeframe of an applied load may cause injury, so too will frequency. This will be covered in the next section.

2.1.2.1.e Frequency

Single, high magnitude loads commonly exceed the failure limit of biological tissues and cause injury (Renstrom, 1993, p. 84). Small loads applied frequently to the musculoskeletal system can be tolerated however, these exact loads can also result in injury (Rolf, 1995). Furthermore, low frequency, high magnitude loads can breach the same injury threshold as small magnitude, high frequency loads (Chandrashekar, Slauterbeck, & Hashemi, 2011) (Figure 2.6).

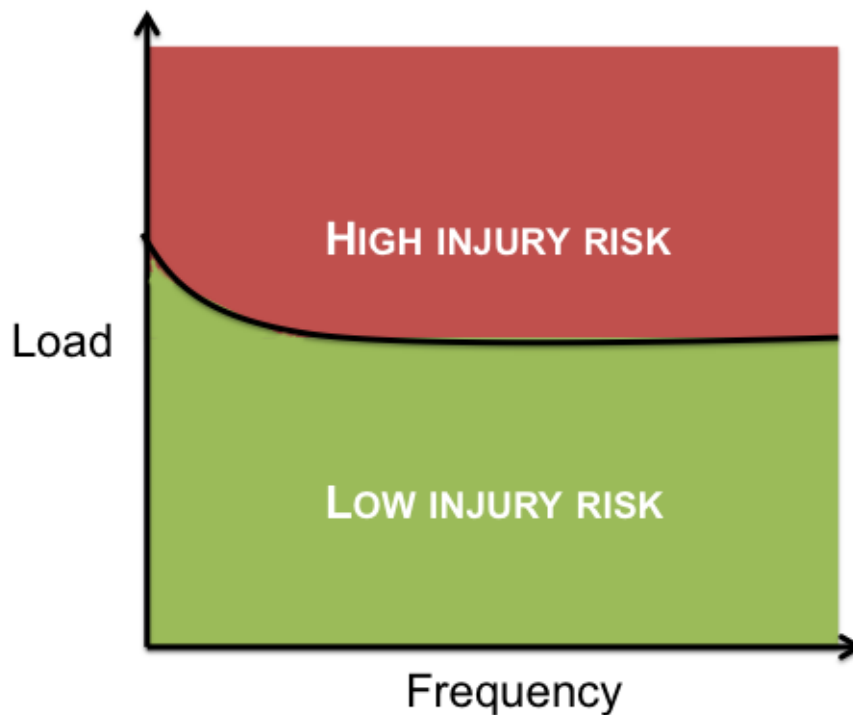


Figure 2.6. Fatigue curve showing the relationship between loading magnitude, frequency of loading and injury risk. Diagram adapted from Hreljac, et al. (2004, p. 846).

Repeated loading causes a breach in the ability of the musculoskeletal system to remodel tissue, overruling the repair process, and resulting in the emergence of clinical symptoms and tissue injury (Hreljac, et al., 2000). To avoid reaching the injury threshold, the combination of loading and frequency must be kept below the limit that the material fatigues. This is difficult to control, because individuals respond differently to loading or have different injury threshold levels thus, making injury risk complex to track and monitor.

To gain a deeper knowledge of the mechanisms that cause musculoskeletal injury, it is important to understand the mechanical properties of the tissues that make up the system. Exploring the mechanical properties of a material whilst under loading allows a causal relationship between loading and failure to be determined therefore, providing insights into musculoskeletal injury mechanisms.

2.1.3 Biological properties of muscle and tendon

The musculoskeletal system has an innate ability to withstand and tolerate loading as well as develop biological tissue in response to the loading (Layne & Nelson, 1999). Loading of the body then becomes very important for injury prevention as the biological structures of the human body must not only learn to endure loads but to also adapt to the applied load (Whiting & Zernicke, 1998). Therefore, in order to gain a deeper understanding of the response of the human body to loading, it is important to consider the characteristics of the biological materials under loading (Figure 2.7). Two structures that make up the musculoskeletal system and undergo repeated loading from the production and transfer of force are muscle and tendon. However, muscle and tendon exhibit different mechanical characteristics and therefore, respond differently to loading.

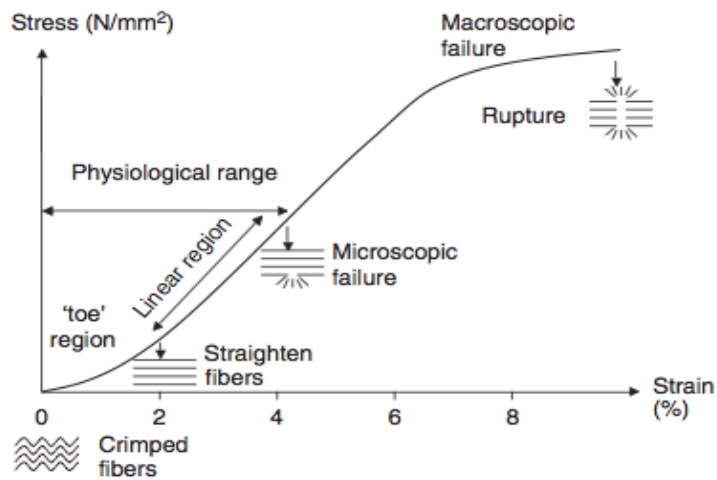


Figure 2.7. Stress-strain curve of tendon showing the regions of the curve that represent a different mechanical characteristic. Diagram from Wang (2006, p. 1567).

2.1.3.1 Muscle

Human skeletal muscle is made up of a number of muscle fibres consisting of proteins that are essential for contraction. The size of a muscle is proportional to the amount of muscle fibres and therefore the amount of myofibrils within each muscle fibre. Myofibrils consist of a systemic arrangement of filaments that form the sarcomere (Figure 2.8).

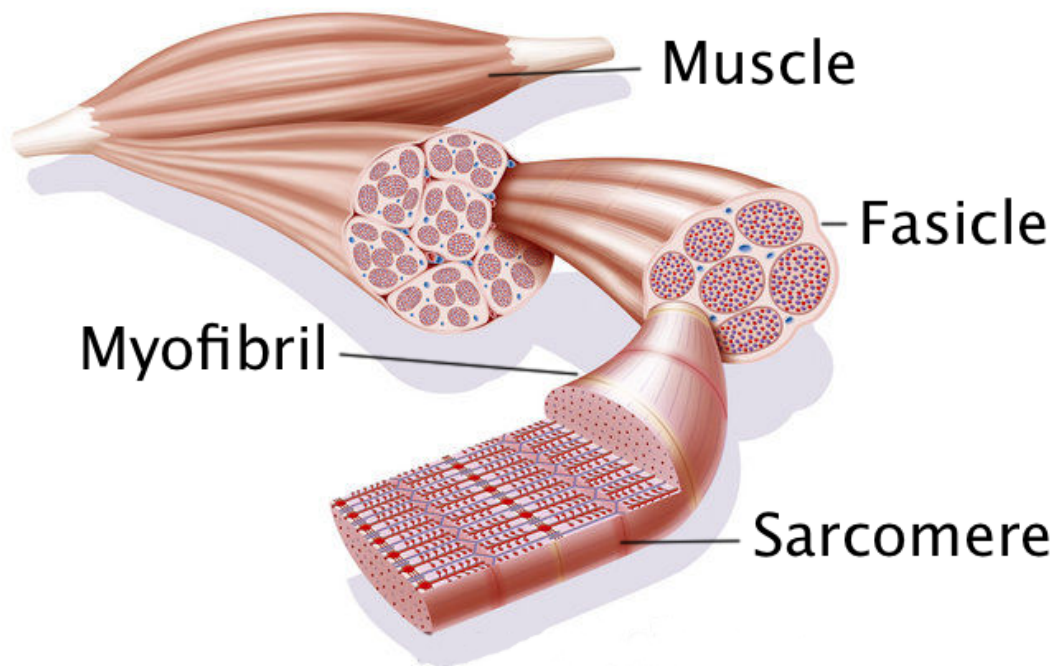


Figure 2.8. Schematic diagram of skeletal muscle composed of a number of muscle fascicles. Muscle fascicles are made up of myofibrils that consist of sarcomeres in series. Adapted from *Skeletal muscle* [image]. (2011). Retrieved from <http://www.sciencephoto.com/media/127052/enlarge.jpg>

Within muscle fibres, there are a number of sarcomeres in series and the length of the fibre depends of the number of sarcomeres (MacIntosh, Gardiner, & Mc Comas, 2006, p. 5). A sarcomere contains elements that form the mechanics of contraction and consists of an I band, A band, Z line, M band and thick and thin filaments (Figure 2.9). The thick filaments that are located in the centre of the sarcomere and are made up of myosin proteins, and the thin filaments are made up of actin, myosin and troponin proteins (Herzog, 2000, p. 270).

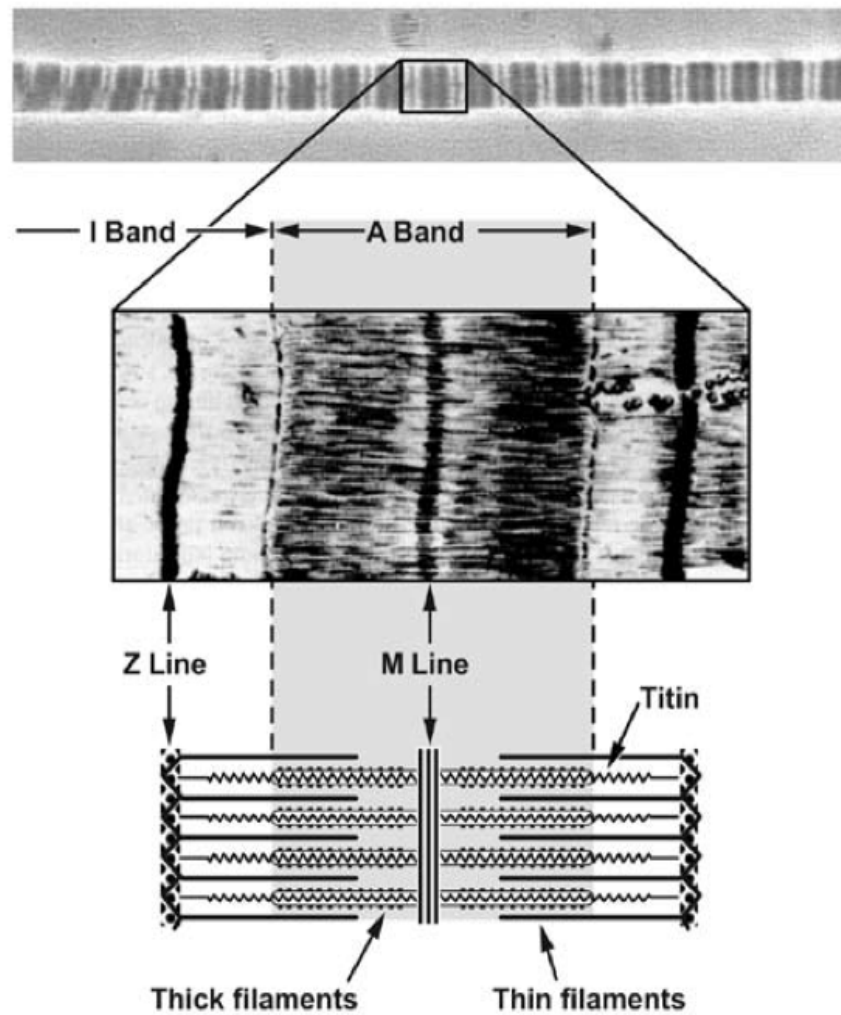


Figure 2.9. An isolated myofibril marked where a single sarcomere lies (top). Myofibril (middle) and schematic diagram (bottom) showing the location of the Z line, M line, thick and thin filaments. Diagram from Herzog, Leonard, Joumaa, & Mehta (2008, p. 8).

Skeletal muscle fibres can also be arranged in unique patterns and can be categorised into fusiform, unipennate and bipennate formations (Figure 2.10). Fusiform arrangement is when the fibres run parallel to the direction of the muscle; unipennate is when the fibre arrangement is parallel but not in the

direction of the muscle; and bipennate arranged fibres are similar to the unipennate arrangement but possess attachments at two opposing sites on the tendon (Whiting & Rugg, 2006, p.75). Uni and bipennate muscle allows more fibres in a given volume and therefore allows more force to be produced furthermore, they are also stronger as they have more fibres arranged in parallel (Herzog, 2000, p. 272).

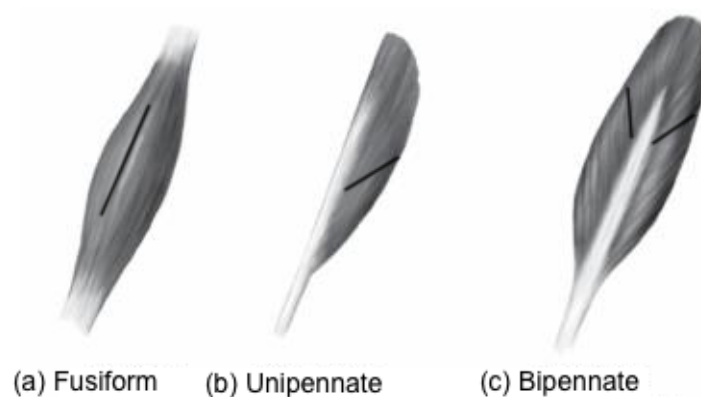


Figure 2.10. Examples of fibre pennation angle (black line) in fusiform (a), unipennate (b) and bipennate (c) muscles. Diagram adapted from Whiting and Rugg (2006, p. 75).

Muscle contraction can be explained by two theories reported in the literature. These theories are known as the cross-bridge cycling theory and the sliding filament theory, respectively. The cross-bridge cycling theory describes contraction by the idea that the thick and thin filaments are sliding over each other as a result of myosin filaments attaching to actin filaments, forming cross-bridges (Huxley, 1969). The force generated by contraction in

this theory is dependent on the number of simultaneous interactions between actin heads and myosin filaments and this is varied by the amount the muscle fibre is stretched and therefore, the amount of overlap between actin and myosin filaments (Huxley, 1969).

The sliding filament theory describes contraction as occurs due to actin and myosin filaments sliding past each other (Huxley, 1969). Shortening of the muscle occurs due to the thick filaments remaining in a fixed position as the thin filaments slide in and out and therefore, force is proportional to the amount of filament overlap as described by sarcomere length (Figure 2.11). There is a low level of force at short muscle lengths however, this force then rises to a maximum point and then upon lessening of overlap due to the sarcomere being stretched, force levels decrease (Figure 2.12).

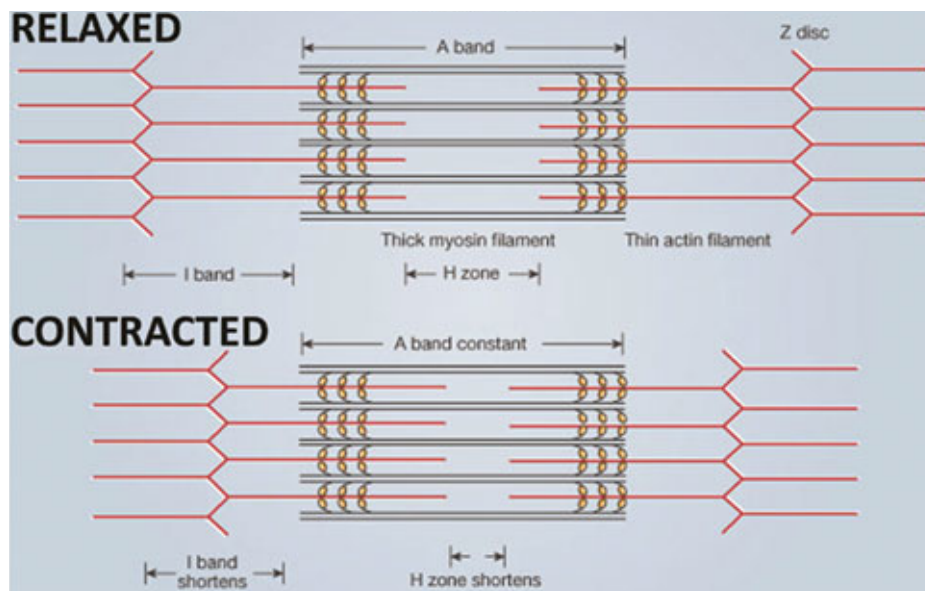


Figure 2.11. Organisation of the sarcomere during a relaxed and contracted state. The change in structure of the sarcomere during contraction shows the

actin (red) and myosin filaments (black) sliding over each other. There is shortening of the I band and H zone that leads to a shortening of the distance between the Z discs. Diagram adapted from Krans, (Krans, 2010 , p. 1).

There are two properties that explain the nature of contraction. These are the muscle force and shortening velocity relationship and the relationship between the tension and change in length of a muscle. The length-tension relationship is important to contraction because it explains the length at which sarcomeres can produce optimal force. Upon increasing the length of a fibre, the amount of active force able to be produced increases to a maximum and then decreases once the optimum length has passed (Figure 2.12) (Rassier, MacIntosh, & Herzog, 1999).

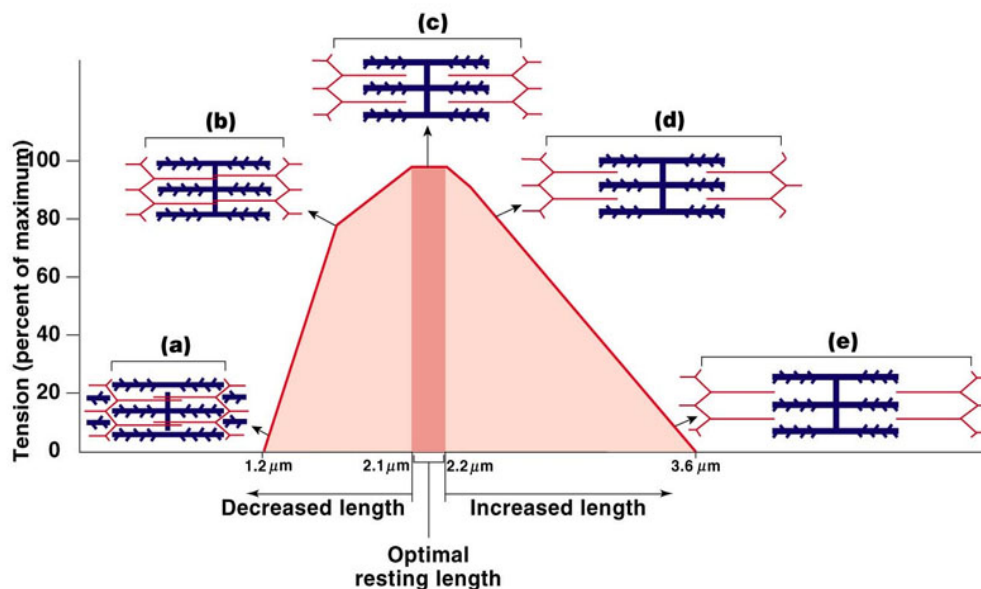


Figure 2.12. An example of the force-length relationship of a sarcomere (middle). At short muscle lengths the filaments are overlapping (a) and with the increasing of length (b), optimum force production occurs (c), and from

there on the sarcomeres are stretched too far and force production decreases (d & e). Diagram adapted from (Martini & Nath, 2009, p. 312).

The velocity of force production is also an important factor in contraction mechanics and particularly so during dynamic movement (Hill, 1938). When a muscle shortens or lengthens, the force that can be produced by the muscle will depend on the rate at which the muscle changes length. However, this relationship differs depending on contraction type. Eccentric contractions can create larger amounts of force than isometric and these eccentric forces increase faster in relation to the speed of lengthening. Conversely, concentric contractions produce force at lower velocities (Figure 2.14).

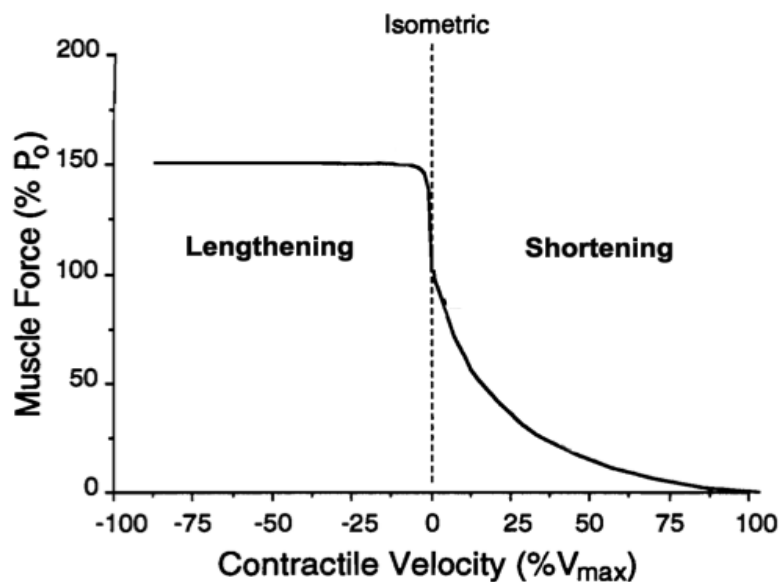


Figure 2.13. The force-velocity relationship of skeletal muscle illustrating the lengthening contractions can be performed at a higher velocity (%V_{max}) and

higher force (P_0) than shortening. Diagram from (Lieber & Bodine-Fowler, 1993, p. 847).

2.1.3.2 Tendon

Tendons insert muscle to bone and transfer forces generated by muscular contraction (Wang, 2006). Tendon is categorised into two types; energy-absorbing and joint positioning. Tendons are made up of collagen fibres that typically run parallel with the length of the tendon and consist of water, collagen, ground substance and elastin (Birch, 2007) (Figure 2.14).

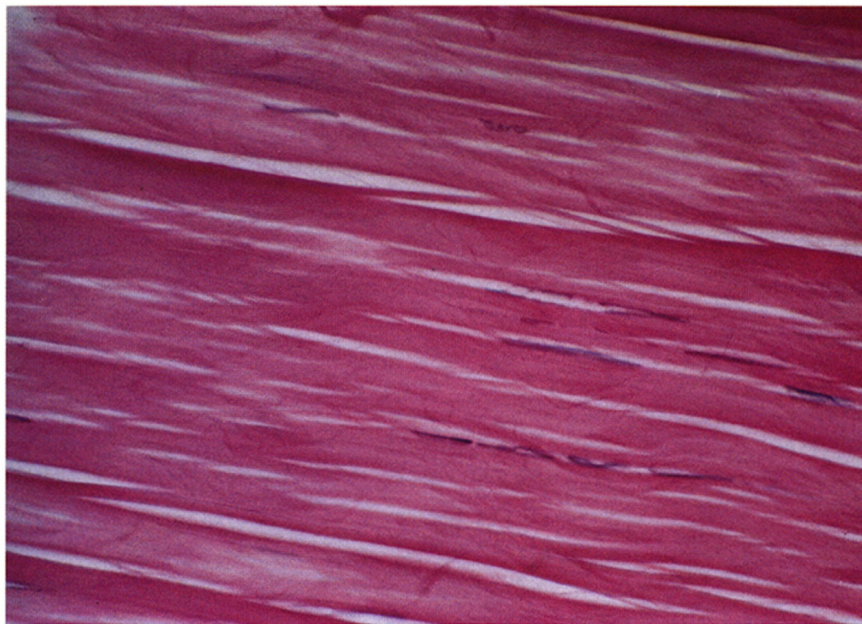


Figure 2.14. A specimen of normal tendon showing parallel bundles of uniform-appearing collagen oriented along the long axis of the tendon. (Kraushaar & Nirschl, 1999, p. 263).

As a result of the direction at which collagen fibres are arranged, tendons have the ability to withstand high tensile loads (strain) from muscle contraction however, tendon is weak under shear or compressive loading (Chan & Hsu, 1993; Williams, Krahenbuhl, & Morgan, 1991).

At resting levels, tendon is crimped and as load is applied the tendon stretches and the crimped slack is taken up so that the fibres appear more parallel to each other (Wang, 2006). This can be seen in the stress-strain curve of tendon and is called the 'toe region' (Figure 2.15a).

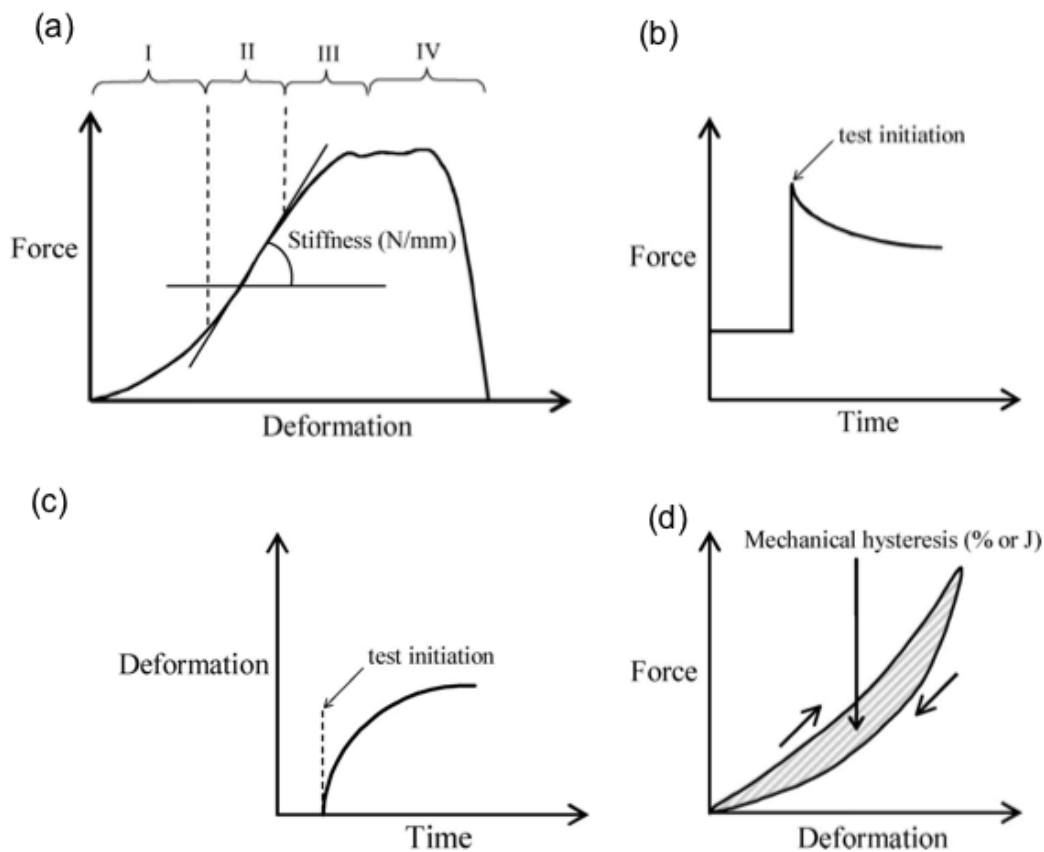


Figure 2.15. The mechanical properties of tendon. (a) force-displacement curve in tendon showing the 'toe' region (I), 'linear' region (II), and failure region (III and IV); (b) force-relaxation curve; (c) creep, and (d) mechanical

hysteresis loop. Diagram adapted from Maganaris, Narici, & Maffulli (2008, p. 1543).

Once it has surpassed the toe region, tendon will then deform at a rate proportional to the load applied. This phase is known as the 'linear' region and represents the stiffness of the tendon (Maganaris, Narici, & Maffulli, 2008). If the loading continues, tendon fibres that are now elongated may become overstretched and begin to fail towards the end of this linear region. Further increasing the deformation of the tendon then brings the tendon into an area where complete failure occurs (Maganaris, et al., 2008).

The intrinsic mechanical properties of tendon that are commonly reported are Young's modulus, ultimate stress and ultimate strain. Young's modulus is the slope of the 'linear region' of stress-strain curve and is a measure of the stiffness of the tendon in proportion to its cross sectional area (Figure 2.16a). Young's modulus of tendon ranges between 1 – 2 GPa (Butler, et al., 1978). Tendon stress represents the ratio of force to cross sectional area of a tendon. Ultimate stress is the point at which failure occurs, typically at around 100 MPa (Bennett, Ker, Imery, & Alexander, 1986; Butler, et al., 1978). Tendon strain is the amount of deformation or elongation that occurs under loading and ultimate tendon strain is the point at which tendon fails and ranges between 4 and 10% of resting length (Bennett, Ker, Imery, & Alexander, 1986; Butler, et al., 1978).

The function of energy absorbing tendons is to store and recoil elastic energy to provide efficient return of stored energy (Maganaris, et al., 2004). Upon stretching, tendon does not display perfectly elastic behaviour and this has been demonstrated by experiments into the force-relaxation, creep and hysteresis characteristics of tendon (Figure 2.16b, c, d). The curvilinear force-relaxation curve shows that a force required to cause a given elongation decreases over time (Figure 2.16b), referred to as creep (Maganaris, et al., 2008). Also, under stretch, tendon loses a certain amount of energy through heat (Maganaris, et al., 2008). This is known as hysteresis and is evidenced by the area of the force-elongation loop when tendon is loaded and unloaded (Figure 2.16.c). The value for hysteresis has been shown to range from 7 to 39% and represents the proportion of energy loss compared to total work done on the tendon (Lichtwark & Wilson, 2005; Maganaris & Paul, 2002; Pollock & Shadwick, 1994).

2.1.4 Summary

Participation in sporting activities often results in musculoskeletal loads that are beyond the norms of sedentary life. Loads applied to and by the human body during sporting activities have particular characteristics and cause tissues that make up the musculoskeletal system to deform, bend, stretch or compress. To examine the effect of these types of loads on the musculoskeletal system, the mechanical properties of the tissue of interest

must be understood. Furthermore, the influence of loading on the ability of the system to control and maintain movement is also important. One measure that estimates neuromuscular control during athletic performance is musculoskeletal stiffness (Cavagna, 1977).

2.2 Musculoskeletal stiffness

During human movement, the limbs are moving relative to the body and the body relative to the surroundings (Cavagna, 1977). Movement is ultimately due to muscular contraction, yet there are certain mechanisms within these contractions that play a role in movement. Storage and utilization of elastic potential energy is one mechanism that is present in certain movements. One example is during locomotion where there is a cyclic transfer between kinetic and potential (gravitational or elastic) energy. When the speed of locomotion is low, a transfer exists from kinetic to gravitational potential energy due to vertical oscillation (rise and fall) of the centre of gravity. As locomotion speed increases, the energy transfer then cycles from kinetic to elastic potential energy, where more energy is being stored as elastic potential than as gravitational (McMahon, 1984, p. 204). Movement is a result of the muscle-tendon unit forcibly contracting and stretching (eccentric contraction) whereby immediately before shortening, it stores elastic energy. A portion of this stored energy is dissipated as heat with the remaining utilised

during the shortening (concentric) contractions. This series of events is known as the stretch shortening cycle (SSC).

The SSC enhances the concentric phase of movement. This stretch-induced muscle enhancement provides an increase in work and power during the final phase of ground contact (Fukunaga, et al., 2001; Lichtwark, Bougoulas, & Wilson, 2007). However, the SSC cannot be present in isolated eccentric or concentric actions, but only in the combination of the two, with concentric being preceded by eccentric contraction. For example, a basic pure concentric contraction, such as a seated knee extension from 90 degrees flexion, does not involve any transfer of gravitational energy to elastic potential. Conversely, during the initial phase of ground contact in running or jumping tasks, external forces are lengthening the muscle-tendon unit while it is active resulting in an eccentric action. This external force is attenuated by the eccentric contraction, followed by a concentric action (contraction) that propels the body forward. The neuromuscular system is able to regulate the ability of the muscle-tendon unit to stretch and recoil and this phenomenon is known as musculoskeletal stiffness.

Musculoskeletal stiffness (MSS) is understood to be an important factor in performance and injury (Butler, et al., 2003). During locomotion, there is an energy exchange between the muscles, tendons, ligaments and bone in order to provide efficient movement (Cavagna, 1977; Ferris & Farley, 1997). MSS is essential in this exchange of energy and described in part of Hooke's law representing the relationship between the deformation of a body and a given

force. It is modelled on the basis that the extent of stretching of a sample of any biological or non-biological substance is directly proportional to the applied force (Gibilisco, 2002, p. 249). It is defined as:

$$F = kx \quad (\text{Eq. 2.1})$$

It states that, the force (F) required to deform a material is related to a proportionality 'spring stiffness' constant (k) and the distance (x) the material is deformed (McMahon & Cheng, 1990)(Figure 2.16).

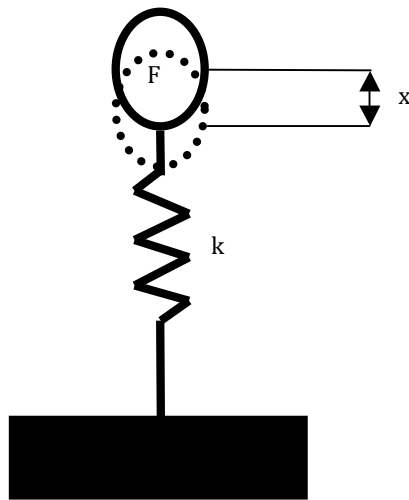


Figure 2.16. The spring-mass system used to model vertical MSS. F represents vertical force; x , the amount of deflection as a result of the force; and k , is the stiffness constant of the spring.

In the context of human movement, MSS is defined as the combination of muscles, tendons, ligaments, cartilage and bones and their collective ability to temporarily deform under a given force (Kuitunen, Komi, & Kyrolainen, 2002; Latash & Zatsiorsky, 1993). In this context, the leg is often modelled as the spring and the supporting mass modelled as the body. Additional factors are also influential in the behaviour of the spring-mass model such as central nervous system control, viscosity, muscle reflex time delays, series and parallel components of muscles, and bi-articular muscles (Latash & Zatsiorsky, 1993). Furthermore, because the model is so complex and involves the consideration of a number of components that influence movement, it is very difficult to experimentally account for all of these factors. Latash and Zatsiorsky have identified this issue and subsequently suggested a shift in terminology that takes into account the differences in the physical nature of the system, and the methods of measurement of MSS. In their work, “quasi-stiffness” is the term used to describe MSS, as the methods of measurement are not performed in equilibrium (Latash & Zatsiorsky, 1993). However, from here on it will be termed “musculoskeletal stiffness” (MSS).

2.2.1 Measurement and calculation of MSS

There are several different calculations of MSS used in the biomechanics literature. The array of calculation methods is mainly due to the availability of equipment of each research group, and to date, no study has

attempted to compare and determine the accuracy of each method. However, of these calculations, three types will be detailed in the following section; vertical, leg and joint MSS. Table 2.1 provides an overview of the various methods of calculation.

2.2.1.1 Vertical MSS

Vertical MSS (k_{vert}) is used to describe jumping or hopping tasks that only involve linear movement in the vertical direction. The calculations for lower extremity vertical MSS are numerous and inconsistent in the literature.

Table 2.1. Summary of formulas for calculating MSS

Vertical MSS (k_{vert})		Reference
$k_{vert} = \frac{F_{peak}}{\Delta y} = \frac{F_{peak}}{\Delta L}$	(Eq. 2.2)	McMahon & Cheng (1990)
$k_{vert} = m \frac{2\pi}{P}^2$	(Eq. 2.3)	Cavagna et al. (1988)
$k_{vert} = m\omega_0^2$	(Eq. 2.4)	McMahon et al. (1987)
$k_{vert} = \frac{m \times \pi (T_f + T_c)}{T_c^2 \frac{T_f + T_c}{\pi} - \frac{T_f}{4}}$	(Eq. 2.5)	Dalleau et al. (2004)
Leg MSS (K_{Leg})		
$k_{leg} = \frac{F_{peak}}{\Delta L}$	(Eq. 2.6)	McMahon & Cheng (1990)
Torsional/Joint MSS (k_{joint})		
$k_{joint} = \frac{2W}{\Delta\theta}$	(Eq. 2.7)	Arampatzis et al. (1999)
$k_{joint} = I \frac{\Delta\omega^2}{\Delta\theta^2}$	(Eq. 2.8)	Williams et al. (2004)
$k_{joint} = \frac{\Delta M}{\Delta\theta}$	(Eq. 2.9)	Farley et al. (1998)

F_{peak} = peak ground reaction force; ΔL = change in leg length or spring compression during ground contact; Δy = change in COM displacement during ground contact; m = body mass; P = the period of oscillation; ω_0 = the natural frequency of oscillation of the system; T_f = time in flight; T_c = time in ground contact; L_o = leg length; u = horizontal velocity; t_c = time in ground contact; θ = joint angular displacement; W = negative mechanical work; ΔM = net muscle moment; I = the product of the length of segment and body mass.

Some research uses a calculation of the period of vibration of the mass-spring system (body) (Eq. 2.3) or the natural frequency of oscillation of the system (Eq. 2.4) (Cavagna, Franzetti, Heglund, & Willems, 1988; McMahon, Valiant, & Frederick, 1987). However, the simplest form of calculation for hopping and jumping tasks is performed by computing the ratio of ground reaction force (F_{peak}) and leg spring displacement (ΔL) (Figure 2.17; Eq. 2.2) (Ferris & Farley, 1997). This method was used in the studies of this thesis.

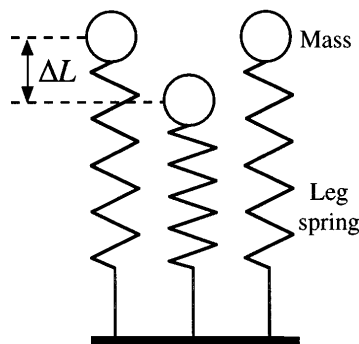


Figure 2.17. Spring-mass model showing a single linear leg spring representing the mechanical behaviour of the musculoskeletal system during ground contact. Mass is equivalent to subject body mass. The three springs represent initial, mid stance and final compression during ground contact or leg spring displacement (ΔL) (Farley, Houdijk, Van Strien, & Louie, 1998).

When a participant hops using both legs on a hard surface, the maximum vertical displacement of the centre of mass (COM) of the body

during the ground contact phase (Δy) is equal to the maximum displacement of the leg spring compression (ΔL) (Ferris & Farley, 1997).

$$k_{vert} = \frac{F_{peak}}{\Delta y} = \frac{F_{peak}}{\Delta L} \quad (\text{Eq. 2.2})$$

The calculated MSS is equal to the combined MSS of both legs, with the vertical displacement of the COM of the body during the ground-contact phase equal to the maximum displacement of the leg spring (ΔL) (Ferris & Farley, 1997). For a detailed explanation of this calculation, see Appendix E.

The period of oscillation method can be used without kinematics (Cavagna, et al., 1988). It is also commonly used by setting the frequency (f) of the task as a constant (e.g. hopping in time with a metronome at 2.0 Hz) (Ferris & Farley, 1997). When combined with the body mass (m) of the participant and vertical force data (F_z), vertical MSS (k_{vert}) can then be calculated. This is done by assuming that the vertical force-time curve is a sine wave, with peak force occurring at the midpoint of the stance phase. The period of oscillation (P) is then used to determine the time taken to reach peak force (F). Table 1 shows the equation (Eq. 2.3) for this method.

$$k_{vert} = m(2\pi/P)^2 \quad (\text{Eq. 2.3})$$

A third method of calculating k_{vert} was developed by (McMahon, et al., 1987) during running with a crouched posture (Groucho running). This

method requires known measures of ground contact time (t_{cont}) and flight time (t_{flight}) (time not in contact with the ground). From these quantities, the natural frequency of oscillation (ω_0) for the system (body) can be calculated and combined with the body mass of the subject to calculate k_{vert} (Table 1, Eq. 2.4).

$$k_{vert} = m\omega_0^2 \quad (\text{Eq. 2.4})$$

However, this methodology is only applicable when the frequency of the activity is constant.

A fourth method was developed for the application of hopping MSS assessment in the field. MSS is first calculated by modelling ground reaction force as a sine wave (Dalleau, Belli, Lacour, & Bourdin, 2004). Therefore, k_{vert} is calculated by:

$$k_{vert} = \frac{m \times \pi (T_f + T_c)}{T_c^2 \frac{T_f + T_c}{\pi} \frac{T_f}{4}} \quad (\text{Eq. 2.5})$$

where m is body mass, T_c is ground contact time and T_f is flight time. A more detailed description on this method can be found in Appendix F.

As mentioned, other methods used to assess k_{vert} involve calculating the period of oscillation of a system. The methods proposed by Cavagna et al., (1988), Dalleau, et al., (2004), and McMahon, et al., (1987) assume that

the COM of the body is behaving like a sine wave with the peak of COM occurring during the middle of the stance phase and thus, making them only relevant when hopping frequency is set, or when force plates are not available. Furthermore, they are not commonly employed and hence, they were not used in the studies of this thesis.

As illustrated, there are a number of ways to determine vertical MSS however, vertical MSS equations can only be used during jumping or hopping when there is virtually no horizontal (forward or lateral) motion present. To account for the forward motion during movement, leg MSS calculations are necessary.

2.2.1.2 Leg MSS

The spring-mass model can be extended to include forward motion. Again, it is defined as the ratio of maximal vertical force to maximal leg spring compression. Movement that results in motion in both the horizontal and vertical directions (e.g. running) must consider these two separate components.

A method of calculating MSS including both the vertical and horizontal components was developed by McMahon and Cheng (1990) and is termed 'leg' MSS (k_{leg}). This method requires the known values of peak vertical ground reaction force (F_{peak}) and the displacement of the compression of the leg spring (ΔL).

$$k_{leg} = \frac{F_{peak}}{\Delta L} \quad (\text{Eq. 2.6})$$

In order to determine ΔL , the displacement of the COM (Δy) must be taken into account. To calculate this, the vertical displacement of COM is determined by double integration of vertical acceleration obtained from force plate data, as shown earlier in Equation 2.2. Displacement of the leg spring is then calculated from the COM displacement (Δy), initial leg length (L_0 , the distance between the hip and the foot) and half the angle swept by the leg spring while in contact with the ground (θ) (Farley, Glasheen, & McMahon, 1993) (Figure 2.18).

$$\Delta L = \Delta y + L_0 (1 - \cos \theta) \quad (\text{Eq. 2.10})$$

with

$$\theta = \sin^{-1} \frac{ut_c}{2L_0} \quad (\text{Eq. 2.11})$$

where u is horizontal velocity, t_c is ground contact time and L_0 is the standing leg length (i.e. greater trochanter to the floor).

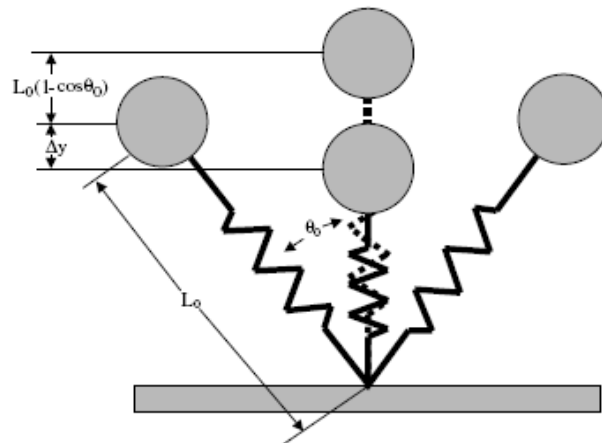


Figure 2.18. The leg model used to calculate leg MSS during ground contact in non-linear movements such as running. Diagram from (Butler, et al., 2003, p. 513).

Similarly, leg spring compression (ΔL) can also be found by:

$$\Delta L = \Delta y + L_0 - \sqrt{L_0^2 - \left(\frac{1}{2} ut_c\right)^2} \quad (\text{Eq. 2.12})$$

where, horizontal velocity (u), time of ground-contact (t_c) and leg length measured from the greater trochanter to the floor (L_0) in addition to the vertical displacement of the COM (Δy) is derived from the double integration of vertical acceleration data (Divert, Baur, Mornieux, Mayer, & Belli, 2005).

Vertical and leg MSS are both “global” measures of MSS. When wanting to consider the individual contributions of each joint during a movement, single joint MSS is investigated. Joint MSS enables a “local”

assessment of MSS and, where the lower body is concerned, the hip, knee and ankle joints are commonly explored.

2.2.1.3 Joint MSS

Movement is controlled by a number of muscles that, in turn, result in rotations about single or multiple joints. Consequently, individual joints contribute to the overall MSS of the system (body). Each joint plays a role in MSS and their contribution is known as either joint or torsional MSS (k_{joint}) (Figure 2.19). The previous methods mentioned refer to the force-displacement ratio in the vertical direction. However at the joint level, moment and the change in angle are the angular equivalents (Farley, et al., 1998).

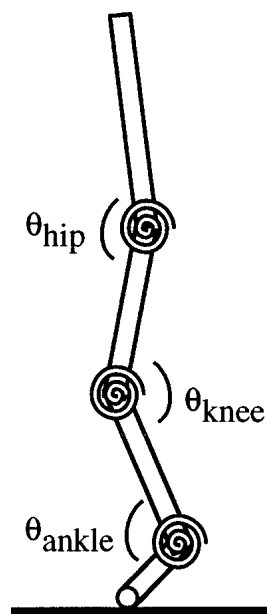


Figure 2.19. Multi-jointed model indicating the hip, knee and ankle joints and their torsional springs. θ representing joint angle at touchdown. Diagram adapted from (Farley, et al., 1998, p. 1045).

Joint MSS can be calculated using three different methods. These are:

(a) A work-energy approach (Arampatzis, Bruggemann, & Metzler, 1999),

$$k_{joint} = \frac{2W}{\Delta\theta} \quad (\text{Eq. 2.7})$$

where W is negative mechanical work at the joint and $\Delta\theta$ is the change in joint angle.

(b) By the equation:

$$k_{joint} = I \frac{\Delta\omega^2}{\Delta\theta^2} \quad (\text{Eq. 2.8})$$

where I is the length of the segment multiplied by body mass, $\Delta\omega$ is the change in angular velocity of the segment, and $\Delta\theta$ is the change in angular position (Williams, McClay Davis, Scholz, Hamill, & Buchanan, 2004).

And, (c) by using the ratio of change in the net muscle moment (ΔM) to joint angular displacement ($\Delta\theta$) in the sagittal plane (Farley, et al., 1998), using the equation:

$$k_{joint} = \frac{\Delta M}{\Delta\theta} \quad (\text{Eq. 2.9})$$

where ΔM is the change in joint momentum and $\Delta\theta$ is the change in joint angle. This is the most commonly used methodology.

However, Arampatzis et al., (1999) are the only authors to employ method (a) during running and it has been argued, but not explained, that it is not reasonable to divide a work integral with a change in joint angle to calculate MSS (Gunther & Blickhan, 2002). Furthermore, Williams et al., (2004) are also the only authors to adopt method (b).

2.2.2 MSS and performance

During hopping, running and jumping tasks, MSS is essential to force development, storage and utilization of elastic energy (Butler, et al., 2003). The relationship between MSS and performance has indicated that the greater the demands of the activity (e.g. increases in running velocity, stride frequency, hopping frequency, drop jump height), the greater the MSS expressed (Tables 2.2 & 2.3).

Table 2.2. Summary of MSS and running performance

Type MSS	Result	Reference
Vertical	Increases with running velocity	McMahon et al., 1984; Cavagna et al., 1988 McMahon & Cheng, 1990; He at al., 1991; Farley et al., 1991; Farley et al., 1998; Gunther et al., 2002; Kuitunen et al. 2002, Walker & Blair., 2002; Cavagna et al., 2005; Morin et al., 2005 & 2006
	Increases as metabolic cost decreases	Dutto & Smith, 2002; Kerdok et al., 2002
	Unchanged across different surfaces	Ferris, Louie & Farley, 1998; Ferris, Liang & Farley, 1999
	Decreases with exhaustion	Dutto & Smith, 2002
Leg	Unchanged/decreases with increased running velocity	McMahon et al., 1987; McMahon & Cheng, 1990; He et al., 1991; Farley et al., 1993; Kuitunen et al., 2002; Avogadro et al., 2004; Cavagna et al, 2005; Morin et al., 2005 & 2006 Ferris, Louie & Farley, 1998; Ferris, Liang & Farley, 1999
	Increased with decreased in surface stiffness	Dutto & Smith, 2002
	Decreases with exhaustion	Dutto & Smith, 2002
Knee	Increases with running velocity	Arampatzis et al., 1999
Ankle	Unchanged with running velocity	Kuitunen et al., 2002

Table 2.3. Summary of MSS and hopping and jumping performance

Type MSS	Result	Reference
HOPPING		
Vertical	Increases with hopping frequency	Farley et al., 1991 & Granata et al., 2002; Dalleau et al., 2004
	Increases with decreases in surface stiffness	Ferris & Farley, 1997; Farley, Houdijk, Van Strien & Louie, 1998
Leg	Increases with hopping forward velocity	Farley et al., 1991
Knee	Unchanged with decreases in surface stiffness	Farley, Houdijk, Van Strien & Louie, 1998
Ankle	Increases with hopping height	Farley & Morgenroth, 1999
	Increases with decreases in surface stiffness	Farley, Houdijk, Van Strien & Louie, 1998
Hip	Unchanged with decreases in surface stiffness	Farley, Houdijk, Van Strien & Louie, 1998
JUMPING		
Vertical	Unchanged with changes in surface stiffness	Arampatziz et al., 2004
Leg	Increases as contact time decreases	Arampatzis et al. 2001a, b; Yoon, Tauchi & Takamatsu, 2007
	Increases as target height increases	Laffaye et al., 2005
	Decreases as rebound height increases	Arampatzis et al. 2001a, b; Yoon, Tauchi & Takamatsu, 2007
Knee	Increases as contact time decreases	Arampatzis et al. 2001a, b
	Unchanged with changes in surface stiffness	Arampatziz et al., 2004
Ankle	Increases as contact time decreases	Arampatzis et al. 2001a, b
	Unchanged with changes in surface stiffness	Arampatziz et al., 2004

2.2.2.1 Hopping

Hopping is a classic movement illustrating the storage and return of elastic energy in the musculoskeletal system. As a result, many studies have been conducted investigating the relationship between MSS and hopping in place. For example, evidence suggests that vertical MSS is affected by gender (Granata, Padua, & Wilson, 2002). When hopping at preferred, 2.5 and 3.0 Hz frequencies, females have displayed less vertical MSS than males. This difference in vertical MSS is due to a larger body mass in males compared to females, as the stiffness of the spring-mass model is proportionate to the mass of the system as shown by the elimination of differences in vertical stiffness when normalised for body mass (Padua, Carcia, Arnold, & Granata, 2005). Gender is one factor that is a manipulator of MSS however, there are other factors which also influence MSS levels.

Vertical MSS has been shown to increase with increases in hopping frequency (Dalleau, et al., 2004; Farley, Blickhan, Saito, & Taylor, 1991; Granata, et al., 2002). It has been reported that the preferred hopping frequency for adult humans is 2.2 Hz because this is the frequency whereby the body behaves like a spring-mass system and efficiently uses elastic energy and reduce the metabolic cost (Farley, et al., 1991). Research has also shown that as hopping frequency increases from 2.2 Hz to 3.6 Hz, vertical MSS levels more than double (Farley, et al., 1991). Similarly, when hopping forward, leg MSS is reported to increase with treadmill speeds above 2.0 m/s, where it doubles with every 2m/s speed increase. This increase in

treadmill speed and hopping frequency has been shown to decrease ground contact time by 30% and lead to an increase in the rate of force development, however the magnitude of increase was not reported (Farley, et al., 1991). To examine why these changes in leg MSS were occurring, studies have focussed their investigation at a more local level to determine the individual joint contributions of the lower extremity.

During hopping, changes in leg MSS have been shown to be determined by changes in the MSS of the ankle (Farley & Morgenroth, 1999). These changes in ankle MSS are due to increases in net muscle moment and displacement about the ankle. Furthermore, individual joint contributions have also been determined in another study which compared power and endurance trained athletes (Hobara, et al., 2008). It was reported that at 1.5 Hz, knee MSS was greater in the power-trained athletes however, hip and ankle MSS between the two groups were reported to be the same. Similarly, during hopping at 3.0 Hz, knee MSS was greater in the power-trained than the endurance trained with no differences found in ankle or hip MSS between the groups.

The literature has demonstrated that there are numerous factors that influence MSS during hopping performance. One similar activity to hopping which has received less scrutiny is jumping.

2.2.2.2 Jumping

A small number of studies (Arampatzis, Schade, Walsh, & Bruggemann, 2001; Walshe & Wilson, 1997; Yoon, Tauchi, & Takamatsu, 2007) have investigated drop or rebound jumping and the associated differences in MSS. Subsequently, it has been shown that reducing ground contact time whilst still attempting to jump as high as possible can result in increased leg and ankle MSS (Arampatzis, Schade, et al., 2001; Yoon, et al., 2007).

Further, it has been suggested that if a cohort is split into a 'stiff' (MSS > 16.0 kN/m) and a 'compliant' (MSS < 15.0 kN/m) group then rebound height for both groups displayed an inverse parabolic shape (Walshe & Wilson, 1997). As a result, rebound height increased up to a height of 60 cm that resulted in the optimum rebound jump height. Moreover, when comparing the stiff and compliant groups at the higher loads (80 to 100 cm), the stiff group significantly underperformed when compared to the compliant group illustrating that the stiff group have an impaired ability to attenuate high eccentric loads. However, the methodology adopted to measure MSS was performed using an alternate method that models the MTU as a damped spring and, therefore, the findings may not be comparable to other studies.

Single-leg jumping involving a run-up approach has also been performed (Laffaye, Bardy, & Durey, 2005). Athletes from four different sports (high jump, volleyball, handball, basketball) and a group of novices were recruited and asked to run 5 m, jump with one foot, and touch a target with the

top of their head. As the target height was increased from 55% to 95% of maximum jump height, there was a 15% decrease in leg MSS. This is in contrast to hopping research which shows there to be an increase in leg MSS with hopping height (Farley, et al., 1991). However, the contradictory results may be specifically related to the task in that: (a) one was a hop and the other was a jump, (b) one performed the task with both legs and the other was with a single leg, and (c) one had a run-up and the other required hopping on the spot. Given the constraints imposed by the run-up, there is an increase in contact time which causes a shift in energy storage from kinetic to gravitational potential, leading to a non-efficient SSC and a limited amount of kinetic energy able to be stored and reused. Therefore, during a single-leg jump, a reduction in leg MSS may actually enhance performance.

2.2.2.3 Running

Similar to hopping, running velocity has been shown to influence MSS. Vertical MSS has been reported to increase with running velocity (Cavagna, et al., 1988; Cavagna, Heglund, & Willems, 2005; Farley, et al., 1991; Farley, et al., 1998; Gunther & Blickhan, 2002; He, Kram, & McMahon, 1991; Kuitunen, Komi, et al., 2002; McMahon, 1984; McMahon & Cheng, 1990; Morin, Dalleau, Kyrolainen, Jeannin, & Belli, 2005; Morin, Jeannin, Chevallier, & Belli, 2006; Walker & Blair, 2002). In contrast, leg MSS has been shown to remain unchanged (Cavagna, 2006; Farley, et al., 1993; He, et al., 1991;

Kuitunen, Komi, et al., 2002; McMahon & Cheng, 1990; McMahon, et al., 1987; Morin, et al., 2005; Morin, et al., 2006) or decrease with running velocity (Avagadro, Chaux, Bourdin, Dalleau, & Belli, 2004). The associated levels of leg MSS during running are lesser than vertical MSS. This is due to the fact the leg MSS takes into account the change in leg length which is always greater than the change in COM that is used to measure vertical MSS. Moreover, leg MSS also takes into account the angle swept by the contact limb, horizontal velocity and resting leg length.

Running velocity has also been reported to influence joint MSS. Knee joint MSS increases with running velocity (Arampatzis, et al., 1999) while ankle MSS remains unchanged (Kuitunen, Komi, et al., 2002), or increases at low velocities (Arampatzis et al.). The alteration of knee stiffness suggests that the changes in leg MSS are a result of changes in knee MSS as confirmed by a number of other studies (Gunther & Blickhan, 2002; Stefanyshyn & Nigg, 1998).

With the exception of one study (Heise & Martin, 1998), running economy has also been shown to be related to MSS (Dutto & Smith, 2002; Kerdok, Biewener, McMahon, Weyand, & Herr, 2002). Typically, a runner will adopt a stride rate that minimises their metabolic cost (Clark, Crossley, Buckley, Bartold, & Bryant, 2007; Hamill, Derrick, & Holt, 1995). Decreases in stride rate are accompanied by decreases in leg and vertical MSS over an exhausting run (Dutto & Smith, 2002; Hunter & Smith, 2007). Furthermore, it has been reported that the metabolic cost of running decreased with an

increase in vertical MSS (Kerdok, et al., 2002). The decrease in metabolic cost indicates that increasing stride rate leads to increased levels of MSS that in turn improve running economy and metabolic cost. Hence, the association between metabolic cost and vertical MSS may be explained by the storage and reutilization of elastic energy. As MSS increases, the musculoskeletal system becomes more efficient at storing and reusing elastic energy and, therefore, running economy and metabolic cost improves. By reducing the ability to store and reuse elastic energy efficiently, the task then requires mechanical energy, via muscular contraction, to maintain the same work rate. This then results in an earlier fatigued state through the reduction of economy and increased metabolic cost.

2.2.3 Injury and MSS

The direct relationship between MSS and performance is well established (Barber-Westin, Noyles, & Galloway, 2006; Brughelli & Cronin, 2008a, 2008b; Butler, et al., 2003), however the relationship between MSS and injury is not. It is understood that a specific level of MSS is required for optimal performance and, to date, only one prospective study exists that investigating MSS levels and injury (Watsford, et al., 2010). Other research is mainly retrospective (Williams McClay & Hamill, 2001; Williams, McClay Davis, Scholz, Hamill & Buchanan; 2003; Bradshaw, Le Rossignol, Williams & Lorenzen, 2006) or provides indirect evidence (Grimston, Engsborg, Kloiber,

& Hanley, 1991; Hewett, Stroupe, Nance, & Noyes, 1996) to suggest there is a link between MSS and injury. Furthermore, indirect evidence has been used to assist in explaining the link between MSS and injury.

Biomechanical measures such as ground reaction forces and other kinematic variables indirectly associated with MSS have been investigated in relation to injury. Specifically, ground reaction forces have been associated with stress fracture injury rates. One study showed that ground reaction forces in female runners who had stress fractures were significantly higher than those females who had not suffered from a stress fracture (Grimston, et al., 1991). In support of these findings it has also been shown that the magnitude and rate of loading was significantly greater in the injured group compared to the non-injured group of runners (Hreljac, et al., 2000). However, the cohort in this study was predominantly male. In other research, arch structure of the foot was examined to investigate MSS and injury rates and found that male and female runners who exhibited high arches were more commonly injured (Williams, et al., 2004). Furthermore, runners who exhibited high arches displayed significantly higher levels of leg MSS, ground reaction forces and loading rates (Williams, et al., 2004). Similarly, female gymnasts who exhibited highest and lowest levels of ankle MSS displayed previous injury history (Bradshaw, et al., 2006), thus indicating that there may be a possible optimal or normal range of MSS. However, these studies only present retrospective findings and there is a paucity of published research directly linking MSS with injury (Butler, et al., 2003).

Two studies have investigated MSS and injury (Pruyn, et al., 2012; Watsford, et al., 2010). Australian Football League players were prospectively assessed over a playing season for hamstring stiffness and leg stiffness (Watsford, et al., 2010). Interestingly, the players who developed a hamstring injury had increased bilateral levels of leg and hamstring stiffness (5% and 11% increase, respectively) than those who remained uninjured. The players who became injured also displayed a 5% lower level of hamstring stiffness in the injured limb compared to the non-injured. However, while Watsford and colleagues (2010) discovered a relationship between MSS and injury, they could only speculate on the causal mechanisms, suggesting that reduced muscular strength in the injured limb, altered neuromechanical properties between limbs, age, training history and previous hamstring injury history as mechanistic possibilities.

Additionally, injured Australian Rules Football players had a greater mean bilateral difference in leg stiffness than non-injured (Pruyn, et al., 2012). However, there was no difference in mean leg stiffness between injured and non-injured players. Interestingly, the injured players were more at risk of a sprint/running or a bending/twisting non-contact soft-tissue injury and, therefore, shedding some light on the potential mechanisms behind injury. Yet, small sample sizes for each injured group (running/sprinting $n = 7$, twisting/bending $n = 2$) may limit the power of these findings.

2.2.4 Measurement and calculation of tendon stiffness

The primary role of tendons is two-fold: (1) to transmit forces created from muscular contraction to the skeleton to generate joint movement, and (2) to store and release elastic strain energy (Pollock & Shadwick, 1994). The interaction between muscle and tendon is critical as it facilitates force production and regulates economical movement (Lichtwark, et al., 2007). Tendons do not behave like rigid bodies but more like elastic bands or springs, storing and returning energy upon stretch and recoil (Ker, et al., 1988). Evidence in humans has revealed that when the gastrocnemius muscle contracts, it behaves isometrically whilst the Achilles tendon lengthens and shortens (Fukunaga, Kawakami, Kubo, & Kanehisa, 2002). This reduces the amount of work the muscle has to perform because the tendon is storing and returning elastic energy which, in turn, contributes to the production of force (Fukunaga, et al., 2002).

The mechanical properties of human tendons have been studied *in vitro* (Zajac, 1989). The intrinsic material properties of tendon are measured by stress or stiffness, strain and Young's modulus at maximum isometrical force. Human tendon exhibits 13-30 MPa of stress, 2% of strain and 1.2 GPa of Young's modulus (Zajac, 1989). However, the nature of *in vitro* testing requires tendon samples to be frozen which may alter their mechanical properties (Smith, Young, & Kearney, 1996), thus not being a realistic representation of *in vivo* behaviour (Zajac, 1989). Therefore, numerous

methods have been applied to investigate the mechanical characteristics of human tendons still attached and *in vivo*.

2.2.4.1 Methods used to measure tendon loads

Tendon and muscular interactions involved in human and animal movement have been of scientific interest for some time. It is beyond the scope of this thesis to review all of the methods used to investigate tendon loading, however a brief summary will be provided.

The earliest experiments investigating tendon forces *in vivo* involved animals and the use of the buckle transducer technique (Salmons, 1969). This method requires a force transducer to be surgically implanted on the tendon of interest, and following appropriate healing, *in vivo* recordings are able to be collected during movement. Until 1984, force transducers were only used on animals; however, the first experiment on humans involved transducers being surgically implanted in tendons under local anaesthesia (Komi, 1984; Komi, Salonen, & Jarvinen, 1984). Due to the invasive nature of the procedure and the size of the force transducers, there were major limitations to the method. Optic fibres have also been used to measure the forces in tendons during movement (Komi, et al., 1996). This method requires optic fibres to be inserted into the tendon, and changes in the geometric properties of the fibre cause a change in light sensitivity and, therefore, the amount of force in the tendon can be measured and calculated. Again, this

technique is invasive and requires local anaesthesia and specialised medical equipment.

Advances in medical imaging have allowed images of the musculoskeletal system to be captured. Through X-ray and magnetic resonance imaging, static images can be produced. However, the use of ultrasonography has enabled real-time, quasi-static imaging to be used to investigate human tissue behaviour *in vivo*.

2.2.4.2 Ultrasonography of the muscle-tendon structures

Ultrasonography is a non-invasive method used to examine muscle-tendon behaviour. To investigate the mechanics of the muscle-tendon unit, a three-element Hill muscle model can be adopted (Figure 2.20) (Lichtwark & Wilson, 2007).

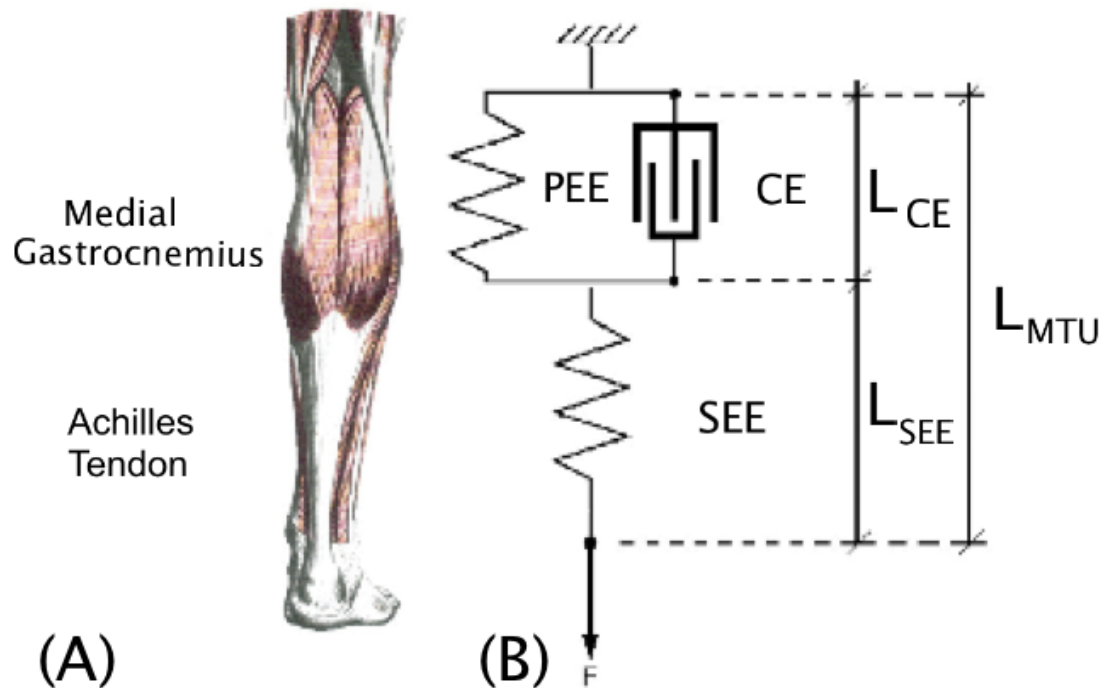


Figure 2.20. Three-element Hill-type muscle model of the medial gastrocnemius and Achilles tendon (A). The muscle-tendon unit length (L_{MTU}) consists of a contractile element (CE; medial gastrocnemius) of length L_{CE} in parallel with an elastic element (PEE; Achilles tendon); and a series elastic element (SEE) of length L_{SEE} (B)(Achilles tendon). Adapted from Lichtwark and Wilson (2007, p. 1769).

This model allows power production, efficiency, work and elastic properties to be assessed in muscle fascicle and tendon. As a result, there have been a number of studies which have used the ultrasound technique to investigate the behaviour of muscle-tendon structures and the changes in muscle fascicle and tendon length during muscular contraction (Fukashiro, Rob, Ichinose, Kawakami, & Fukunaga, 1995; Ito, Kawakami, Ichinose, Fukashiro, & Fukunaga, 1998).

Measuring muscle-tendon mechanics using ultrasonography requires an ultrasound probe to be positioned on the skin, allowing a specific anatomical landmark of interest, such as the junction on the gastrocnemius muscle (GA) and the Achilles tendon (AT) to be located, imaged and tracked (Figure 2.21).

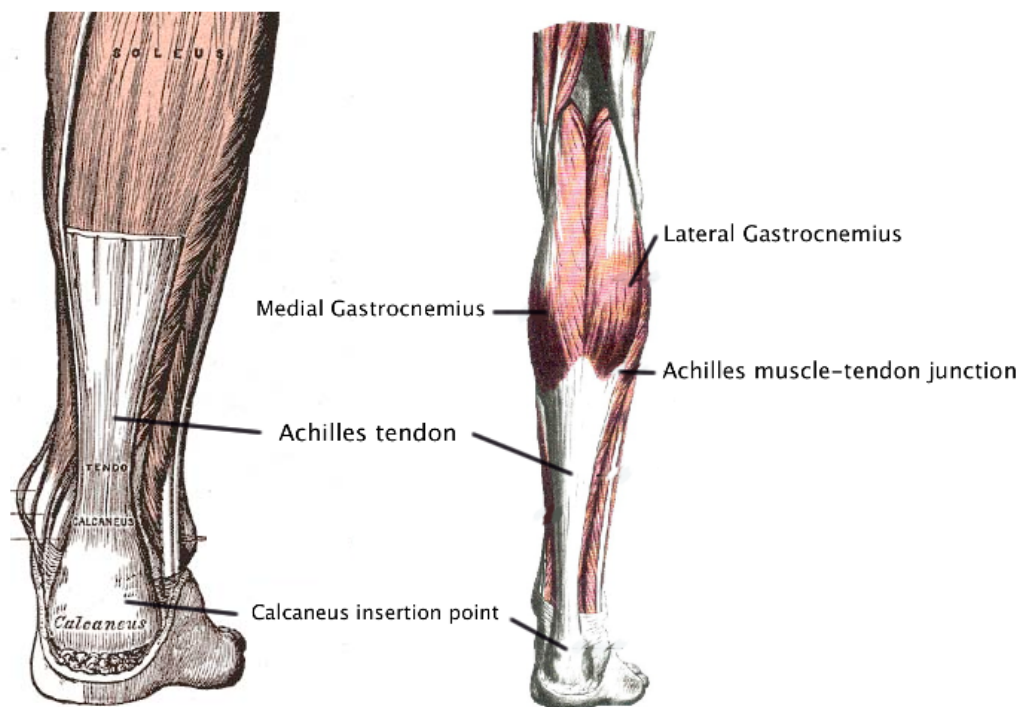


Figure 2.21. Shows the anatomical landmarks of the gastrocnemius and Achilles tendon (Adapted from *Grey's Anatomy*).

The behaviour of this landmark can then be seen in real-time during static and quasi-dynamic activities as long as the probe remains still and in

the same position as it was in the original, static view. For example, Figure 2.22 shows the displacement of the AT and GA junction during rest, with 1000N and 2000N of force applied via plantar-flexion. Here, the ultrasound probe is positioned so that a longitudinal image of the GA junction can be seen, enabling the displacement of the landmark of interest to be quantified.

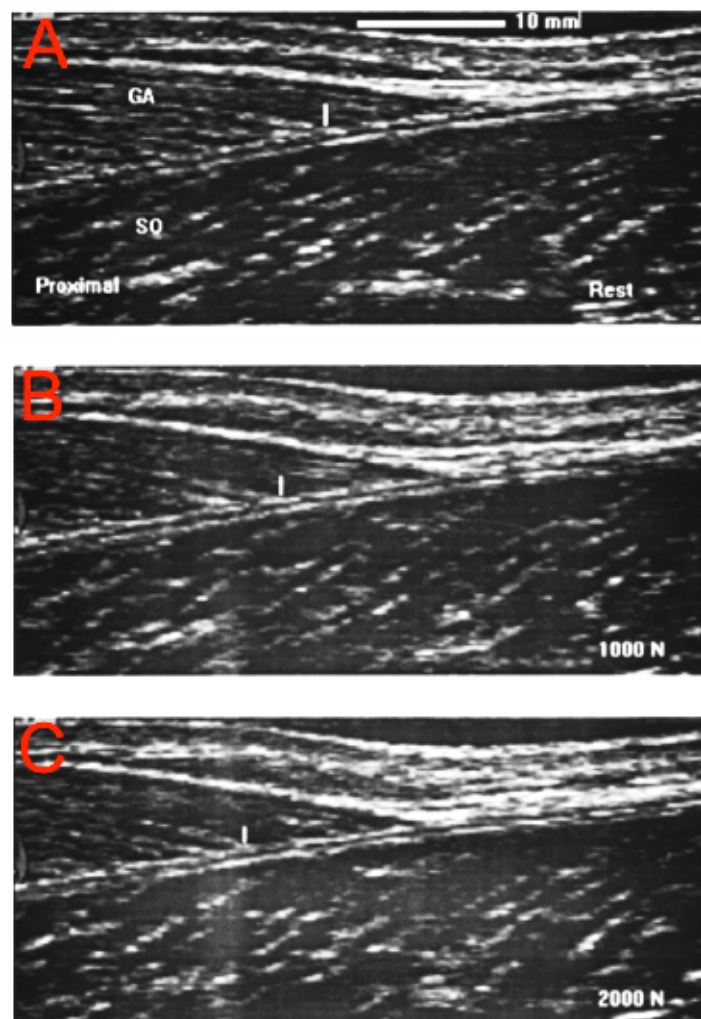


Figure 2.22. Longitudinal ultrasonography image of the triceps surae muscle during **A.** Resting conditions, **B.** 1000N, and **C.** 2000N of force. GA, gastrocnemius muscle; SO, soleus muscle. The vertical marker is used to

measure displacement during contraction. Image from (Magnusson, Aagaard, Rosager, Dyhre-Poulsen, & Kjaer, 2001, p. 279).

The mechanical characteristics of tendon are often measured via a maximal voluntary contraction (Magnusson, et al., 2001) or electrical stimulation of the muscles acting on the tendon of interest (Maganaris, 2001) to explore tendon behaviour during controlled, static experimentation. Furthermore, actual, real-life dynamic tasks have also been researched to understand more about the mechanics of the muscle-tendon interaction during common physical activities (Ishikawa, Finni, & Komi, 2003; Ishikawa & Komi, 2007; Ishikawa & Komi, 2004; Ishikawa, Niemela, & Komi, 2005; Kurokawa, Fukunaga, & Fukashiro, 2001; Kurokawa, Fukunaga, Nagano, & Fukashiro, 2003; Lichtwark & Wilson, 2005, 2006). Therefore, the ability to measure tendon behaviour allows the strain, stress, Young's modulus, hysteresis and stiffness of a tendon to be investigated. The next section will focus on tendon mechanics during real-life activities.

2.2.5 Tendon mechanics and performance

Until recently, information about the behaviour of tendon *in vivo* has been limited. The elastic nature of tendon influences the overall function of the muscle-tendon complex thus, affecting the ability of tendon to store energy and transmit force. This is important because the ability of tendon to transfer force and provide elastic energy affects the time-dependent

characteristics and overall function of the muscle-tendon complex (Magnusson, Narici, Maganaris, & Kjaer, 2008). Therefore, with the progression of ultrasound technology, explorations have been made into tendon function during dynamic movement.

During hopping, AT stiffness has been shown to range between 122 and 234 N/mm (Lichtwark & Wilson, 2005). Furthermore, AT strain and displacement during single-legged hopping at 2 Hz has been shown to be approximately 12% and 20 mm respectively. During squat and countermovement jumping, the AT undergoes a displacement of approximately 30 mm, leading to strain levels of approximately 6% and 4.4% and storage of 4.9 to 5.6 J of elastic energy (Kurokawa, et al., 2001; Kurokawa, et al., 2003). Tendon behaviour has also been investigated during drop jump behaviour, with tendon displacements of 25 to 51 mm in the vastus lateralis (VL) (Ishikawa, et al., 2003; Ishikawa & Komi, 2004; Ishikawa, et al., 2005) and 40 mm in the AT (Ishikawa, et al., 2005) observed. Moreover, walking and running have also been used to investigate tendon mechanics and shown displacements of 32 mm and 46 mm in the medial gastrocnemius (MG) tendon (Ishikawa & Komi, 2007) and in the AT average strain levels of 4.6% and an average tendon displacement of approximately 11 mm (Lichtwark & Wilson, 2006).

2.2.6 Summary

Tendon mechanics and MSS have been investigated throughout a number of functional tasks, and it has been suggested that they have a major influence on performance. However, the levels of MSS that cause muscle damage and injury, or the optimal levels required for performance enhancement are relatively unknown. Furthermore, there is limited prospective research attempting to investigate MSS and injury (Pruyn, et al., 2012; Watsford, et al., 2010). Additionally, there is a paucity of research exploring the relationship between muscle damage and the effects on tendon mechanics such as stiffness and strain. This type of research is important to facilitate the development of screening protocols identifying the optimal levels of MSS and tendon stiffness deemed to be safe. Consequently, this will in the development of specific training programs to reduce injury and help maintain appropriate MSS levels.

2.3 Injury and exercise induced muscle damage (EIMD)

Muscle strain injuries have been reported as the most commonly occurring sports-related injury (Orchard, et al., 2005). This is especially the case in sports that involve high intensity efforts. Muscle strains are graded from 1 to 3 depending on the severity (Figure 2.23). A grade 1 injury is the least severe and usually involves micro tears in muscle fibres. A grade 2

injury involves a partial tear, while a grade 3 injury involves a complete rupture (Brukner & Kahn, 2007; Close, Ashton, McArdle, & MacLaren, 2005).

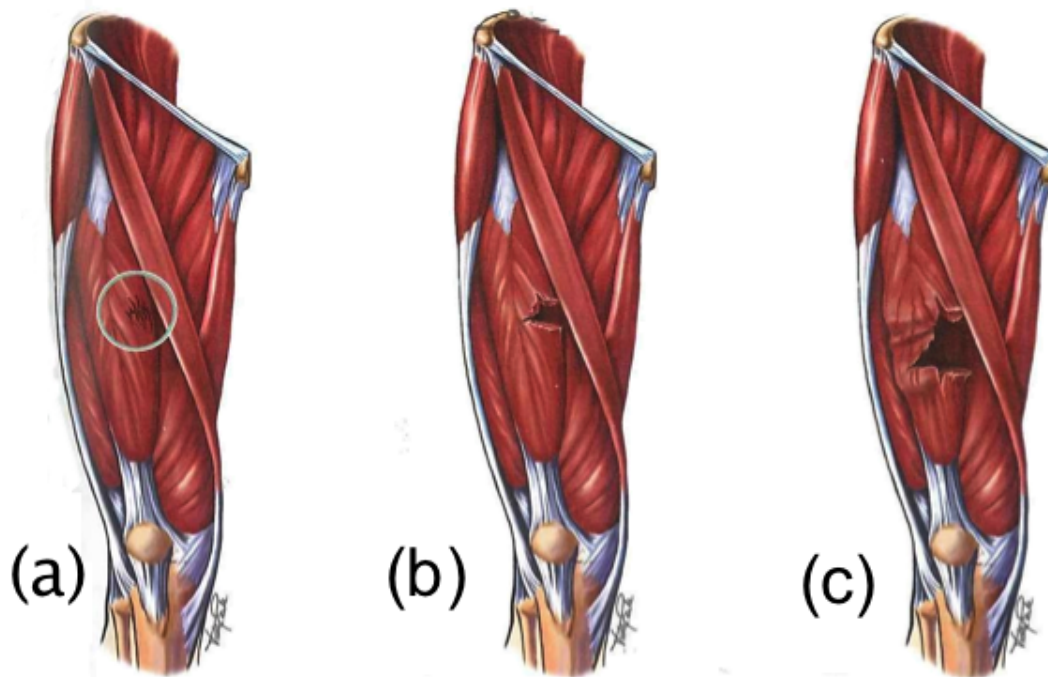


Figure 2.23. Muscle strain injury. Grade 1 (a), grade 2 (b), and grade 3 (c) muscle strain injury. Diagram from (Brukner & Kahn, 2007, p. 13).

Despite the increasing awareness of the high prevalence of muscle strains, little is known about the exact mechanisms causing these injuries (Finch & Orchard, 2002). Human and animal studies use muscular contraction to examine the mechanisms behind skeletal muscle injury (Warren, Hayes, Lowe, & Armstrong, 1993), and many models have been proposed to induce muscular injury in humans (Clarkson, Nosaka, & Braun, 1992; Cleak & Eston, 1992). These models have used single and multiple

contractions to induce muscle injury and have discovered that muscle can adapt to the stimulus following the initial injury (Nottle & Nosaka, 2005b). However, the balance between returning to sports whilst this damage is present and remodelling or adaptation is still occurring can be problematic (Figure 2.24). As a result, a substantial amount of research exists investigating eccentric exercise as a model to induce muscle injury (see Chapter 2.3.3).



Figure 2.24. A model of the relationship between exercise and injury. This demonstrates the grey area between being injured (yellow & red) and uninjured (green). Adapted from Williams, Krahenbuhl, & Morgan (1991) by Blackah, (2011, p 9).

Unaccustomed, eccentric contractions have been characterised as a type 1 muscle strain (Cheung, et al., 2003). The subsequent injury following unaccustomed, eccentric contractions is thought to occur as a result of

excessive levels of strain applied to muscle fibres concurrent with the length that the muscle fibre is at during this application of strain (Newham, et al., 1988). The magnitude of stress causes mechanical failure within the muscle fibre structures and a host of biochemical changes have been shown to follow (Koh, 2008). These changes to the muscle structure have led to individuals experiencing a dull, aching pain, combined with tenderness of the muscle and a sensation of stiffness (Nosaka, 2008). This sensation of skeletal muscle tenderness and pain is often referred to as delayed onset of muscle soreness (DOMS).

2.3.1 Proposed mechanisms underlying EIMD

There has been considerable interest in sports science and medical research regarding the mechanisms behind muscle damage brought on by unaccustomed, high-force, eccentric contractions. Injury to muscle fibres can occur as a result of direct trauma (Koh, Cassidy, & Watkinson, 2003), inflammatory processes (MacIntyre, Reid, & McKenzie, 1995), intense exercise (Noakes, Kotzenberg, McArthur, & Dykman, 1983) or by myotoxic agents such as local anaesthetics (Lieber & Friden, 2002; Zink & Graf). A number of experimental models have been used on humans and animals to investigate the causes, mechanisms and prevention of muscle damage, (Warren & Palubinskas, 2008, pp. 14, 15, 17 & 19). As a result of these models, exercise-induced injuries in humans have been confirmed using

various protocols with various contraction types (Golden & Dudley, 1992; Newham, Mills, Quigley, & Edwards, 1983b). However, the predominantly used model is one where repetitive eccentric contractions unaccustomed to the participant are performed (LaStayo, et al., 2003).

2.3.1.1 Contraction type

Muscle actions whereby an activated skeletal muscle is elongated whilst producing tension are called eccentric contractions (Lieber & Friden, 2002). Eccentric muscle actions result in greater evidence of muscle damage than isometric or concentric contraction (Golden & Dudley, 1992; Jones, Newham, & Torgan, 1989; Newham, McPhail, et al., 1983). When muscle fibres are lengthened under tension, such as in an eccentric contraction, there is a reduction in motor unit recruitment which results in a smaller cross-section of fibres activated to handle the load (Enoka, 1996). As a result of the excessive load and reduction in recruitment, structural damage to the contractile and cytoskeletal components of the muscle fibre occurs. To induce structural damage to muscle fibres, a range of testing protocols have been adopted to investigate muscle damage and contraction type. One study consisting of four participants (3 male, 1 female) employed a stepping protocol to elicit muscle damage via eccentric and concentric contractions in the quadriceps muscles (Newham, McPhail, et al., 1983). It was found that the eccentric muscle actions performed during stepping caused changes in

the muscle architecture such as sarcomere disruption and z-line streaming and increases in the sensations of pain and tenderness. Another study reported decreases in elbow flexor strength and increases in tenderness following eccentric when compared to isometric and concentric contractions (Jones, et al., 1989). However, the actions performed by Jones and colleagues (1989) were uni-articular and specific only to the elbow flexors when compared to the lower-extremity, multi-joint actions, of the stepping protocol in Newham, et al., (1983).

Studies as early as 1953 (Asmussen) have used eccentric contractions as models to induce muscle injury and research suggests that the early events which cause muscle injury are mechanical in nature (Lieber & Friden, 2002). As a result, scientific focus has been focused upon the mechanical factors that are leading to muscle damage.

2.3.1.2 Mechanical factors

There are numerous mechanical factors that lead to and determine the magnitude of muscle contraction-induced injury. As mentioned earlier, eccentric contractions have been shown to cause significantly more muscle fibre damage than other contraction types. Consequently, research has focused on the mechanical factors behind eccentric contraction-induced injury.

One study used isolated soleus muscle from rats to investigate the mechanical factors associated with eccentric contractions (Warren, et al.,

1993). A dose-response relationship was explored whereby levels of muscle force, muscle length and lengthening velocity were altered. It was established that the greater the force produced during the eccentric contraction, the greater the level of muscle damage as measured by a deficit in force. This indicated that the muscle damage was dependent upon the tension produced in the muscle fibre.

An animal model was used to investigate the effect of work and average force on contraction-induced injury (Brooks, Zerba & Faulkner, 1995). It was shown that the work done to stretch the muscle was the best predictor of muscle damage and stretching the muscle to a strain of more than 50% fibre length passively, and 30% actively, produced a significant deficit in muscle force production. However, other investigators (Lieber & Friden, 1993) have suggested that the change in length of the activated muscle fibre is associated with contraction-induced injury and that muscle injury is related to the magnitude of strain or the change in length of the muscle fibre, not work done. Furthermore, it has been reported that the greater the amount of stretch during contraction, the greater the injury (Talbot & Morgan, 1998). This is supported by evidence of more damage being produced after eccentric contractions at longer muscle lengths than shorter (Child, Saxton, & Donnelly, 1998; Newham, et al., 1988).

2.3.2 Indicators of damage

Indicators of muscle damage can be separated into histological, bio-chemical, histo-chemical, and functional measures. Histological evidence requires muscle biopsies or magnetic resonance imaging (MRI), bio-chemical and histo-chemical analysis assesses bodily fluids and muscle tissue, and functional tests focus on functional evidence of muscle damage. Some indicators are complex to measure and require expensive or specialised equipment, while others are simpler to administer whilst still being valid and reliable. Predominantly, because muscle damage is multi-factorial, more than one indicator is used to measure muscle damage. It is beyond the scope of this study to review and assess all the relevant indicators of muscle damage in the literature; therefore, the most common and reliable indicators have been selected (Nosaka & Clarkson, 1996; Nosaka & Newton, 2002; Nosaka, Newton, & Sacco, 2002). These are creatine kinase (CK) activity, muscular strength and muscular soreness (Warren, Lowe, & Armstrong, 1999).

2.3.2.1 Creatine kinase activity

Muscle damage is commonly assessed through muscle specific proteins such as CK (Clarkson & Hubal, 2002). CK is an intramuscular enzyme responsible for maintaining adequate adenosine triphosphate levels during muscle contraction (Lieber & Friden, 2002). Disruption of skeletal muscle architecture, which is common after eccentric exercise, enables the

diffusion of soluble muscle enzymes such as CK into the extracellular space (Cheung, et al., 2003) through increased permeability or breakdown of the muscle cell membrane (Lieber & Friden, 2002). As a result, CK is reported to be elevated in blood serum following eccentric exercise due to the micro-damage experienced by muscle tissue. Levels of CK in blood plasma following eccentric activity are dependent upon the intensity, type and duration of the exercise, and the training status of the individual (Brancaccio, Maffulli, & Limongelli, 2007). Marathon injury models, where average performance duration was 251 mins, have demonstrated peak levels of CK at 3880 IU/L (Akimoto, et al., 2002), while downhill running for 45 mins which resulted in peak blood CK levels of 940 IU/L (Yu, Malm, & Thornell, 2002) when compared to resting levels of around 300 IU/L (Brancaccio, et al., 2007). Investigations into eccentric contraction intensity have revealed that plasma CK levels were significantly higher during eccentric MVC than at 50% of eccentric MVC (Nosaka & Newton, 2002). Meanwhile, trained individuals have been reported to have significantly lower plasma CK levels than untrained after 3 days post-eccentric exercise (Newton, Morgan, Sacco, Chapman, & Nosaka, 2008).

Time course changes in CK indicate that levels commonly peak 1 to 4 days post-eccentric exercise and remain elevated for several days (Clarkson, 2006; Clarkson, Kearns, Rouzier, Richard, & Thompson, 2006; Nosaka, et al., 2002; Paschalis, et al., 2005). However, caution must be exercised when using CK as a marker of muscle damage as there is larger individual

variability in the response to exercise, regardless of sex or muscle mass (Clarkson & Hubal, 2002). Furthermore, the relationship of CK to muscle soreness, muscular strength loss and magnitude of the injury is not strong, indicating that damage to skeletal muscle architecture is only one explanation for the onset of DOMS (Friden & Leiber, 2002).

2.3.2.2 Muscular strength

Reductions in muscular strength are thought to be a reliable indicator of muscle injury following eccentric-based muscle injury (Warren, et al., 1993). However, the mechanism by which the strength/force loss occurs remains unclear (Cheung, et al., 2003). Regardless of the mechanisms behind the loss of strength, muscle strength loss has been shown to be a reliable and valid indirect marker of muscle damage (Kellis & Baltzopoulos, 1995).

Immediately post-eccentric exercise, a loss of muscular strength is observed and can continue for weeks, depending on the mode, intensity and duration of the activity (Sayers & Hubal, 2008). Reductions in strength can range from 11% (Etheridge, Philp, & Watt, 2008) to 40% (Child, et al., 1998) and can remain significantly lower than baseline levels for up to 12 days (Child, et al., 1998). However, the magnitude of strength reduction is primarily dependent upon the intensity of the exercise protocol. To assess decrements in muscular strength, the majority of literature uses open-kinetic chain dynamometry protocols to isolate a specific joint and group of muscles while

performing a maximal voluntary contraction (MVC) (Warren, et al., 1999). Open-kinetic chain movements involve the end of a segment to be free and the load to be moved away, from the body (e.g. bench press) (Prokopy, et al., 2008). However, torque output is velocity dependent because of the force-velocity relationship of muscles. MVC measurements are therefore joint-angle and angular velocity dependent during isokinetic testing.

2.3.2.3 Delayed onset muscular soreness

Muscle soreness has been cited as the most common measure of muscle damage (Warren, et al., 1999) and is common among recreational and elite athletes. It is possible that soreness experienced from eccentric-induced muscle damage is a result of swelling and pressure in the muscle (Friden, Sfikianos, Hargens, & Akeson, 1988) or the release of chemicals which activate nerves that carry messages of pain to the central nervous system (O'Connor & Cook, 1999). DOMS is pain or discomfort associated with EIMD and is associated with high-force, eccentric muscular work usually peaking within 24 to 72 hours post-exercise and lasting for 5 to 7 days (Byrne & Eston, 2002b).

Muscle soreness can be assessed subjectively via a rating system such as the visual analogue scale or objectively by measuring the amount of force applied to cause discomfort (MacIntyre, et al., 1995). Subjective scores are commonly rated on a scale of 1 (no soreness) to 10 (very sore), and peak

soreness values vary depending on the type and duration of the muscle damaging exercise (Newham, et al., 1988; Newham, Mills, Quigley, & Edwards, 1983a). Consistent with blood CK levels and muscular strength, changes from baseline are seen following eccentric contraction-induced muscle damage (MacIntyre, et al., 1995). However, there is no similarity in time-course changes or magnitude changes from baseline between muscular soreness, muscular strength or CK levels (Ebbeling & Clarkson, 1989).

2.3.3 Models used to illicit EIMD

To investigate the mechanisms behind muscle injury and henceforth, cause and prevention, an experimental model of muscle injury needs to be applied. Consequently, there have been a number of models proposed via animal (Armstrong, Ogilvie, & Schwane, 1983) and human examples (Komi & Viitasalo, 1977). Ethical considerations surrounding animal and human models differ and as a result animal models have been used to explore EIMD in a more invasive manner (e.g. *in vivo*) (Armstrong, et al., 1983; McBride, Gorin, & Carlsen, 1995). However, reviewing the literature regarding animal models of muscle damage is beyond the scope of this theses and therefore, only literature involving humans will be covered in this section.

Human models have used repeated eccentric contractions involving elbow flexion and knee extension actions to induce injury (Byrne & Eston, 2002a; Nosaka & Clarkson, 1996). Repeated eccentric contractions of the

elbow flexors at varying intensities, angular velocities and angular excursions have been used to induce muscle damage via dynamometry or custom built apparatus (Lambert, Marcus, Burges, & Noakes, 2002; Newham, et al., 1988; Nosaka, Sakamoto, Newton, & Sacco, 2001). Protocols have varied from 2 to 125 contraction repetitions across a range of intensities based on MVC strength, body weight or 1 repetition maximum (1RM) strength (Lambert, et al., 2002; Nosaka, et al., 2001). Moreover, angular velocity and angular excursion of elbow flexion has ranged from 20 to 120 deg/sec and spanned from 40 to 180 deg (Bloomer, Goldfarb, McKenzie, You, & Nguyen, 2004; Evans, Knight, Draper, & Parcell, 2002; Nosaka & Clarkson, 1996). However, the majority of the literature investigating elbow flexion and muscle damage is difficult to compare due to a lack of consistency in contraction repetition, load, angular excursion and angular velocity. Furthermore, whilst the volume of literature is not as large as studies on elbow flexion, muscle damage has also been explored via knee extension models to replicate actions performed during sporting movements.

Isokinetic dynamometry has been used to investigate repeated eccentric contractions of the knee extensors and muscle damage. Similar to elbow flexor experiments, contraction repetition, load, angular excursion and angular velocity have been manipulated to induce muscle damage. Studies have used a volume ranging from 40 to 300 eccentric contractions at speeds of 5 to 90 deg/s, and at varying intensities (e.g. percentages of MVC, 1RM and body weight) (Babul, Rhodes, Taunton, & Lepawsky, 2003; Byrne, Eston,

& Edwards, 2001; Komi & Viitasalo, 1977). However, similarly to elbow flexion models, the majority of the literature investigating muscle damage in knee flexors is difficult to compare due to a lack of consistency between studies.

Whilst these single joint experiments are relatively easy to control, administer and evaluate, they are limited to actions performed by muscles that act about a uniaxial joint and focus only on damage performed by acute eccentric contractions. Therefore, other models have been adopted that consist of whole body exercise which are not limited to just eccentric contractions in isolation.

Using whole body exercise as a model for injury may be more ecologically valid. By simulating actual athletic performance, an accurate picture of muscle damage that occurs during sports participation can be seen. As a result, downhill running, downhill backwards walking, downward stepping and prolonged endurance exercise models have been used to induce muscle injury. Downhill running models have been performed at a percentage of maximal oxygen consumption or maximal heart rate on a treadmill with an incline grade of -5 to -25% for up to 60 minutes (Akimoto, et al., 2002; Braun & Dutto, 2003; Byrnes, et al., 1985; Koller, et al., 1998). The actions of downhill running cause damage to the muscles involved in hip and knee extension. Similarly, stepping models have focussed on damage to the hip and knee extensor musculature (Duarte, et al., 1999; Newham, Jones, & Edwards, 1983; Newham, Jones, Tolfree, & Edwards, 1986). These

experiments have involved stepping downward from a box set at a height of 110% of leg length at a rate of 15 per minute for around 300-900 repetitions or until exhaustion (Newham, Jones, et al., 1983; Newham, et al., 1986). By lowering the body and stepping down with one leg, eccentric contractions are being performed and therefore, inducing muscle injury. Furthermore, prolonged exercise has also been used to facilitate muscle damage to hip and knee joint musculature during marathon and ultra-marathon events (Akimoto, et al., 2002; Noakes, et al., 1983). These models have consisted of 192 to 615 minutes or running over distances of 42.2 to 160 km (Akimoto, et al., 2002; Cummins, et al., 1987; Noakes, et al., 1983) however, the large variations in time and distance of these protocols, make them very difficult to compare. Lastly, downhill backward walking has been used to induce damage within the musculature of the ankle joint (Nottle & Nosaka, 2005a; Racinais, Bringard, Puchaux, Noakes, & Perrey, 2008; Whitehead, Allen, Morgan, & Proske, 1998; Whitehead, Weerakkody, Gregory, Morgan, & Proske, 2001). These studies have involved walking backwards, on a downhill slope of 7 to 14 degrees, at a speed of 1.3 to 3.6 km/hr and for between 30 and 60 minutes.

Given the nature of the whole-body exercise model that encompasses the use of a number of joints and muscle groups, it may therefore be more difficult to evaluate injury. Furthermore, the levels of strain experienced within the musculature may also not be of a high enough magnitude and hence, only a low level of EIMD and DOMS will occur (Nottle & Nosaka, 2005a). With that

in mind, it is not only necessary to explore EIMD and DOMS caused by whole-body exercise, it is equally important to investigate the actual affect it has on performance of a true sporting task.

2.3.4 EIMD and sports performance

EIMD and its side effects are a familiar experience for both novice and experienced athletes. Although the exact mechanisms responsible for causing EIMD are unknown, the impact of EIMD on performance has been investigated to some degree. Treatment of type 1 muscle strains often does not involve complete withdrawal from exercise (Brukner & Kahn, 2007, p. 195). Because of the mild severity of a type 1 muscle strain, many athletes play or train with some level of soreness or sensation of pain, loss of force and increased levels of CK. These factors may significantly alter an athlete's kinematics and kinetics and lead to decreases in performance at training or in competition. Areas identified as being altered following EIMD are joint kinematics, neuromuscular recruitment patterns and the production of strength and power (Cheung, et al., 2003; Smith, 1992), which all contribute to a reduction in scores on functional measures of performance.

2.3.4.1 Joint kinematics

Joint kinematics have been shown to be altered as a result of EIMD. Research investigating the effect of EIMD on running gait has demonstrated

that muscle damage causes reduction in maximal hip flexion at touch-down, a reduction in maximum knee joint flexion in the swing and stance phases, an increase in maximum dorsiflexion angle, and a reduction in plantar flexion angle during the support phase (Hamill, Freedson, Clarkson, & Braun, 1991; Paschalis, et al., 2007). It is suggested that the alterations may be a result of the body choosing “safer” movement patterns to protect itself from further damage (Paschalis, et al., 2007). Furthermore, reductions in maximum knee and hip joint flexion have been attributed to the increased swelling of muscle tissue (Hamill, et al., 1991) and these reductions in kinematics following EIMD have been suggested to decrease the body’s ability to attenuate shock in the legs and head (Goff, Hamill, & Clarkson, 1998; Hamill, et al., 1991).

2.3.4.2 Neuromuscular control

Neuromuscular control is affected by EIMD (Byrne, Twist, & Eston, 2004). Proprioception has been demonstrated to be impaired as a result of eccentric exercise whereby investigations into the perception of muscular force and joint position have suggested that force development is underestimated, and joint position is either under or over estimated (Forestier, Teasdale, & Nougier, 2002; Walsh, Hesse, Morgan, & Proske, 2004). Moreover, reductions in neuromuscular efficiency resulting in a greater central activation required to produce force have also been observed (Deschenes, et al., 2000).

2.3.4.3 Strength and power

Production of strength and power in sports performance is crucial. EIMD has been shown to significantly reduce measures of muscular strength and power (Byrne, et al., 2004). Knee and ankle joints have been shown to exhibit a reduction in strength following EIMD, with a 30 to 40% reduction shown in knee extensor strength (Byrne & Eston, 2002a) and a 30% reduction in ankle extensor strength (Avela, Kyrolainen, Komi, & Rama, 1999a). Additionally, isokinetic measures of muscular strength at set angular velocities (e.g. 0.52, 1.05, 3.14, 4.19 rad/sec) have also been shown to be reduced (Byrne & Eston, 2002a; Deschenes, et al., 2000). However, isokinetic dynamometry is limited in its ability to replicate sports-specific movements and velocities. Isolated, open-kinetic chain, single joint movements and restricted angular joint velocities do not replicate closed-kinetic chain, sport-specific multi-joint movements; therefore, other performance measures (e.g. jump performance) have been investigated.

2.3.4.4 Functional measures of performance

To translate results into a sporting context, functional measures of performance and EIMD have been investigated (Avela, et al., 1999a; Byrne & Eston, 2002a, 2002b; Horita, Komi, Nicol, & Kyrolainen, 1999; Nicol, Komi, & Marconnet, 1991a, 1991b). In terms of vertical jump performance, reductions

have occurred in peak force production, muscle activity, ground reaction force, stretch-reflex sensitivity, MSS and jump height (Avela, et al., 1999a; Byrne & Eston, 2002a; Horita, et al., 1999; Nicol, et al., 1991a, 1991b). The influence of EIMD on sprinting performance has also been investigated (Semark, Noakes, Gibson, & Lambert, 1999), with no effect of EIMD on sprint time observed. Performance in endurance tasks after EIMD has also indicated elevated physiological responses (e.g. one minute ventilation, breathing frequency, respiratory exchange ratio, heart rate, blood lactate, plasma cortisol, core temperature)(Gleeson, Blannin, Walsh, Field, & Pritchard, 1998; Gleeson, Blannin, Zhu, Brooks, & Cave, 1995). However, there were no differences shown in aerobic fitness (VO₂max), endurance time or exercise efficiency.

2.3.5 Summary

The susceptibility of muscle injury through eccentric contraction has been well established. However, the exact mechanism responsible is currently contentious and it is likely that no single mechanism is responsible for exercise-induced muscle damage. It is understood that alterations in joint kinematics, co-ordination, and strength and power measures are all risk factors associated with returning to sports participation following EIMD (Byrne, et al., 2004; Cheung, et al., 2003). However, there is limited research attempting to relate functional performance decrements and their mechanisms

with athletic performance. Additionally, due to the nature of EIMD being a mild, sub-clinical type of injury, it is tolerated and treated very differently from person to person and practitioner to practitioner. As a result, there is potential for individuals to become injured to a greater extent due to the weaknesses in tissue structure.

2.4 Chapter summary

Injury from sports participation incurs a high cost to the individual and the community. It often results in a cessation or reduction of physical activity and an increased risk of re-injury. Muscle damage caused from exercise is a common side effect of physical activity and is experienced by every level of athlete and individual. However, whilst a large body of research is invested in this area, the causes and risk factors of injury are relatively unknown. Therefore, exploring and attempting to gain an insight into the mechanisms behind injury is of significant value to both the individual and the community.

Research suggests that excessively high and low levels of MSS may increase the risk of injury. Bony injuries are thought to be related to high levels of MSS and soft tissue injuries, such as muscle damage, are potentially related to low levels of MSS. The role of MSS during athletic performance is well researched and the storage and return of elastic energy within the musculoskeletal system has been shown during running, jumping and hopping and has illustrated the benefit of MSS to mechanical energy

conservation. Whilst the importance of MSS to human performance is known, the level at which MSS becomes problematic in causing injury is probably highly individualistic, but is unknown and rarely researched prospectively. Hence this illustrates an importance and merit in exploring any association between muscle damage (injury) and MSS.

Tendon mechanics have been used to describe the muscle-tendon interaction during movement. Tendon and muscle interact to control the position and velocity of joint rotation and tendon specifically provides the transfer of contractile force and storage and reuse of elastic energy to allow this movement. Under force, tendons have the capacity to change length, affecting their ability to control joint position and velocity. Therefore, given the influence of the muscle-tendon interaction on movement, the effect of muscle injury on the dynamics of tendon provides an interesting area to consider, particularly because high levels of strain lead to tendon injury. However, there is a paucity of research exploring the effect of changes in muscle and the transferred influence on the mechanical properties of tendon.

Unaccustomed, eccentric exercise is known to cause muscle damage, with common symptoms following a bout of EIMD including swelling and soreness. However, the exact mechanisms behind the cause of EIMD are speculative. In terms of athletic performance, EIMD has been shown to result in reductions in range of joint motion, neuromuscular control and proprioception, as well as reductions in muscular strength and power production. Furthermore, EIMD has also been shown to result in reductions in

vertical jump, therefore illustrating the practical implications of EIMD on sporting performance.

The compensatory mechanisms that occur in the presence of EIMD may provide important information in regards to injury prevention and rehabilitation. MSS is indirectly linked with injury mechanisms and therefore, may potentially play a part in the compensatory mechanisms that occur when EIMS is present. By exploring the relationship between MSS and tendon mechanics and EIMD, the implications of muscle damage on the ability of the musculoskeletal system to utilise elastic energy can be provided. This is of value because compensatory mechanisms during sporting performance may increase injury risk, particularly if a return to sport is premature. Therefore, a great benefit will be gained by understanding the change in MSS and in the mechanical properties of tendon that occur within a timeframe when EIMD is present. Currently, a paucity of literature exists exploring the effect of EIMD on MSS and tendon mechanics and hence, the aim of this investigation was to advance knowledge about this relationship.

3. Methods

3.1 Ethical approval

This study was approved by the Human Research Ethics Committee of the Australian Catholic University. All participants completed and signed informed consent forms (Appendix A).

3.2 Participant recruitment

Based on power calculations (see Chapter 3.5.1), twenty active male participants were recruited for each study reported in this thesis. The recruitment methods employed included posting notices on university noticeboards and attending lectures where information letters outlining the study were handed out (Appendix B & C). Interested participants contacted the lead investigator (the author of this thesis) where a time was arranged that they could begin testing. Females were excluded from recruitment due to the effect of oestrogen on musculoskeletal stiffness (MSS) and the mechanical properties of tendon (Bryant, et al., 2008; Granata, et al., 2002).

3.2.1 Inclusion criteria

3.2.1.1 Chapter 4 inclusion criteria

Males were included into the study of Chapter 4 if they:

- (i) were aged between 18 – 30 years;

- (ii) were injury-free at the time of testing and had not missed a training session or game in their respective sport for six weeks preceding the time of testing (Noyes, Lindenfeld, & Marshall, 1988);
- (iii) had no current history of medication usage or medical conditions;
- (iv) had no recent hospitalisation or surgery in the last 12 months;

3.2.1.2 Chapter 5 & 6 inclusion criteria

Males were recruited for the studies in Chapter 5 and 6 if they:

- (i) were aged between 18 – 30 years;
- (ii) were injury-free at the time of testing and had not missed a training session or game in their respective sport for six weeks preceding the time of testing;
- (iii) had no current history of medication usage or medical conditions;
- (iv) had no recent hospitalisation or surgery in the last 12 months;
- (v) had not participated in downhill backwards walking training or resistance training of the plantar-flexor muscle in the 6 months prior to the study;
- (vi) agreed to not consume any nutritional supplements, ibuprofen, anti-inflammatory or pain relief medication;
- (vii) agreed to avoid any massage, contrast therapy or stretching, to prevent any influence of other recovery interventions on the results of the study;

- (viii) agreed to abstain from any physical activity that they would normally participate in.

3.3 Methods and procedure

Upon entry into the study, participants attended the research laboratory of the School of Exercise Science at the Australian Catholic University. They were asked to wear loose fitting clothing and footwear that they would normally wear for sports participation. The involvement as a participant required completion of a consent form and a health screening questionnaire. The questionnaire contained information in regards to medication, medical and surgery history, injury history, supplementation use, eccentric exercise history and laterality (Appendix D). Each of these are outlined in the following section.

3.3.1 Participant screening

All participants were required to fill out a health screening questionnaire (Appendix D). This questionnaire took approximately 20 minutes to complete and contained questions relating to the sections outlined below.

3.3.1.1 Medication

Participants were asked to record all medication use including non-prescribed medications. Medications such as pain relief or anti-inflammatory medications affect the ability to measure muscle damage (Mishra, Friden, Schmitz, & Lieber, 1995) and, therefore, participants who were taking these were excluded from the study.

3.3.1.2 Self reported medical and surgical history

Participants were asked to record all information about their medical and surgical history over the previous 12 months. Any participant who displayed a medical or surgery history within the last 12 months was excluded.

3.3.1.3 Anthropometric measurements

Anthropometric data were collected for standardisation purposes and for the requirement of the VICON® three-dimensional motion analysis software. Anthropometric data measured were: height, weight, leg length, inter-anterior superior iliac spine distance, anterior superior iliac spine to greater trochanter distance, knee width and ankle width of each leg.

3.3.1.4 Supplement usage

Supplementation use such as caffeine has been shown to affect adenosine receptors and, therefore, reduce the intensity of and sensation of

pain (Gliottoni, Meyers, Arngrimsson, Broglio, & Motl, 2009). Therefore, participants who displayed moderate to high daily dosages of caffeine (≥ 400 mg/day) were excluded from this study due to the importance of monitoring pain in determining the existence of muscle damage (Gliottoni, et al., 2009). This was only asked and recorded for the use in the studies of Chapter 5 and 6.

3.3.1.5 Eccentric exercise history

Participants were asked to record their participation in any form of eccentric exercise training in the previous 12 months for the study reported in Chapters 5 and 6. Eccentric exercise has been shown to result in an adaptation response whereby there is a lessened level of muscle damage that occurs upon repeating the same bout of exercise (Nosaka, 2008; Nosaka, et al., 2001; Nottle & Nosaka, 2005b). Therefore, any participant who indicated that they had performed any eccentric exercise training were required to explain what it was, and were subsequently excluded if it involved training of the lower limbs.

3.3.2 Laterality assessment

For the purpose of delineating results into dominant and non-dominant limbs, a laterality questionnaire was used (Coren, 1993) which included questions such as “Which foot would you use to kick a ball at a target?”. Responses for right-footedness were assigned a value of 1 and responses for

left-footedness were assigned a -1. Scores were totalled and negative scores signified a left-sided preference, positive scores a right-sided preference and, a score of zero represented ambilaterality.

3.3.3 Hopping and jumping assessment

The continuous hopping (CH) and continuous jumping (CJ) tasks were performed at the participant's self-selected frequency (CHSS and CJSS, respectively) and also at a set frequency of 2.2 Hz (CH2.2 and CJ2.2, respectively). For these two tasks:

(1) participants were required to perform straight-legged CH, locking their knees to reduce the input of the knees and, thus, predominantly requiring the ankle joint to perform the task. This test was used to assess ankle flexor/extensor input (Hobara, Kanosue, & Suzuki, 2007);

(2) perform CJ bent-legged without attempting to restrict the movement of any joint in the lower extremity. Although the CJ task is technically a hopping action when performed on one leg, for ease of reading and displaying results it will be referred to in this study as a continuous jump.

Participants were asked to perform the CH and CJ tasks on a force platform (Kistler, model 9268BA, Switzerland) for 20 continuous hops or jumps (Hobara, et al., 2007). Set frequency hopping and jumping was controlled by a digital metronome and trials were accepted if they were within $\pm 2\%$ of the set frequency (Farley, et al., 1998; McLachlan, Murphy, Watsford, & Rees, 2006).

The CH and CJ tasks were performed on the left, right and both legs together. The first and last two hops/jumps of the 20 trials were excluded from analysis and participants were instructed during all hops/jumps to maximise jump height and minimise contact time (Dalleau, et al., 2004). For this protocol, participants were instructed to keep their hands on their hips to remove the influence of the arms.

3.3.4 Running assessment

Kinetics, kinematics and vertical ground reaction forces (VGRF) were analysed during ten trials of running (RUN). Participants were required to run at 3.35 m/s (12.06 km/hr) (Williams, et al., 2004) along a 10 m runway. This speed was set due to the influence of running velocity on vertical, leg and joint stiffness (Brughelli & Cronin, 2008a). Running speed was verified by timing gates (Smart Speed, 1.8 MHz, Fusion Sport, Australia), placed 7 m apart. The force platform was positioned midway between the two timing gates and trials were only accepted if they were within $\pm 5\%$ of the target speed (Williams, et al., 2004). Participants contacted the force plate with their left foot for five trials and their right foot for the remaining five trials, and were instructed to run and look ahead to avoid targeting the force plate (Abendroth-Smith, 1996). Only trials that exhibited a clean (whole) foot contact on the force plate were included in the analysis. Participants were instructed to alter their starting feet by the principal researcher (CJ) and the order in which feet the participant started was randomised to negate an order effect. This was

performed to control which foot hit the force plate. Any trials that exhibited a forefoot ground contact pattern were excluded from analysis due to its affect on running kinetics and kinematics and, therefore, MSS (Gunther & Blickhan, 2002).

3.3.5 Eccentric exercise protocol

The eccentric exercise protocol consisted of 60 minutes of downhill backwards walking on a treadmill (h/p/cosmos, Quasar It, Germany). The treadmill speed was 0.67 m/s (2.4 km/hr) and declined to a gradient of -8.5° . This protocol has been used previously and shown to cause an acceptable level of eccentric muscle damage, whereby approximately 30-35 strides per minute occur and, therefore, around 1800 to 2100 eccentric contractions are performed during the 60-minute exercise protocol (Nottle & Nosaka, 2005b). Participants were asked to take long, searching, backward strides, making sure to contact the treadmill initially with their forefoot in order to emphasise the length and duration of eccentric contractions.

3.3.6 Muscle damage assessment

Perceived muscle soreness was assessed from self reports via a visual analogue scale (VAS) which ranged from one (no soreness) to 10 (very, very sore) (Cleather & Guthrie, 2007). This score was reported prior to blood sampling at the beginning of each testing session.

Functional performance of the plantar flexor muscles was assessed via a maximal voluntary contraction (MVC) in a customised squat rack (Figure 3.1 A). Three isometric plantar flexor contractions were held for 3 sec, with a 2 min rest period between trials, and were completed using the participant's dominant leg. Peak force was recorded at the steadiest point on the force profile and averaged across the three trials, as described in Pua et al. (2010). The reliability of this MVC protocol was determined prior to the study from seven untrained male participants (ICC=0.85).

Blood samples were drawn from an antecubital vein using a 5 ml evacuated, heparinised vacutainer tube by a qualified phlebotomist. The blood sample was then centrifuged at 1500×g for 10 min (Spintron, Australia, GT-175BR) to obtain plasma, and the plasma layer was removed and frozen at -80°C until analysed. Plasma CK activity was determined spectrophotometrically using a Reflotron analyser (Boehringer-Mannheim, Indiana, USA, Reflotron).

3.3.7 Experimental set-up and isometric plantar flexion procedure

Achilles tendon mechanics was determined for the dominant leg of each participant. Participants performed an isometric MVC of the plantar-flexors in a custom built rack (Figure 3.1 A). The customised squat rack consisted of a barbell (1.82 m [6 ft], 20 kg) fixed by a chain around each end so that the height of the set-up could be adjusted to each participant and

vertical movement could be controlled. Both chains were fitted with a turnbuckle (Figure 3.1 B, C & D) to adjust length and restrict movement to allow a pure isometric plantar flexion contraction to be performed without any changes in ankle angle. Each participant was custom-fitted to the rack prior to testing so that minimal movement occurred vertically, anteroposteriorly or mediolaterally. Feet were positioned wholly on the force platform (no overhang) and with the ankle joint at 90 degrees. The testing protocol consisted of the participant gradually ramping force from rest to MVC within approximately 5 seconds (Kubo, Kanehisa, & Fukunaga, 2005). This was repeated until three technically correct trials were performed. Each trial was separated by a 2 min rest period and all measured values were averaged across the three trials. Ground reaction force data were collected at 1200 Hz (Kistler, model 9268BA, Switzerland).

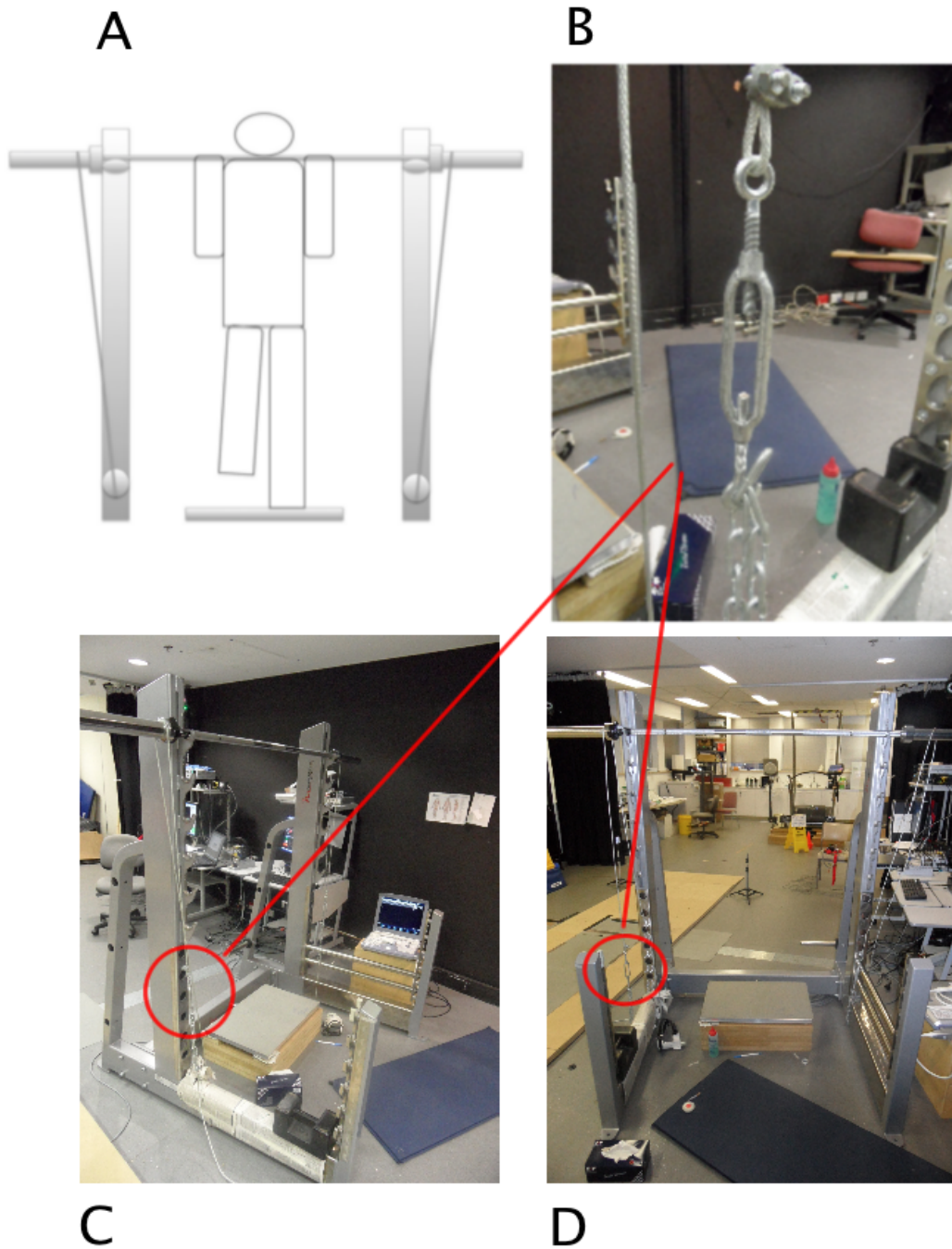


Figure 3.1. The customised squat rack in detail (A). The barbell is locked in place by a cable and chain on either side and tightened by a turnbuckle (B, C & D), so that the contraction is isometric and there is no movement in the bar. Each participant was fitted into the rack prior to testing and kept the same

throughout testing. Participants were fitted so that their knees were straight and there was minimal heel lift during plantar flexion.

3.3.8 Determination of muscle-tendon junction position

The triceps surae consists of two muscles; soleus and gastrocnemius. The gastrocnemius is made up of two heads; medial and lateral (Gray's anatomy). The medial head originates from the medial condyle of the femur and the lateral head originates at the lateral condyle of the femur (Gray's anatomy). Both heads form a common tendon, the Achilles tendon, which originate at the posterior surface of the calcaneus (Figure 3.3). The position of the Achilles tendon-muscle junction (ATJ) of the medial gastrocnemius muscle was determined using real-time B-mode ultrasonography (Vivid-i, GE Healthcare, United Kingdom) with an electric linear array probe (5-13 MHz wave frequency, linear array transducer, 42 × 7 mm footprint; 12L-RS, GE Healthcare, United Kingdom). Sagittal plane images of the myotendinous ATJ were recorded at 40 frames per second and stored on the ultrasonography computer system. The position of the ATJ was located via surface palpation and marked with a non-permanent pen. The ultrasound probe was then positioned so that the ATJ appeared clearly in the inferior third of the ultrasound image (Figure 3.4). The probe was then longitudinally attached to the dermal surface over the location of the ATJ insertion point with a custom-built brace and adhesive foam. The custom-built plastic brace was moulded to the shape of the ultrasound probe and to the size of an average

gastrocnemius muscle (Figure 3.4). The brace was precast in such a way that the angle of the head of the probe was fixed to reduce the parallax error that occurs when probe angle is altered, and so that direct contact with the skin was maintained during recording. Achilles tendon length was measured at rest whilst standing between the marked insertion point at the calcaneus and the marked location of the ATJ using a measuring tape. The validity (Maganaris, 2005) and reliability of this protocol has been previously established (ICC = 0.89) (Clark, et al., 2007).

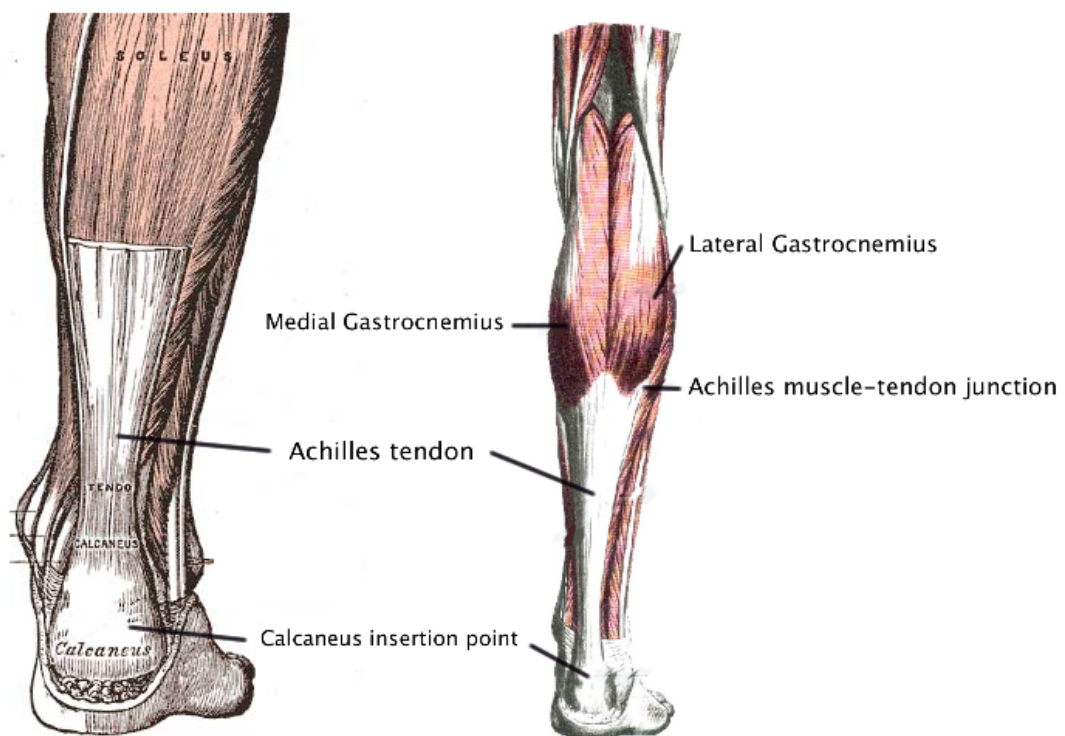


Figure 3.2. Anatomy of the gastrocnemius and Achilles tendon (Adapted from *Grey's Anatomy*).

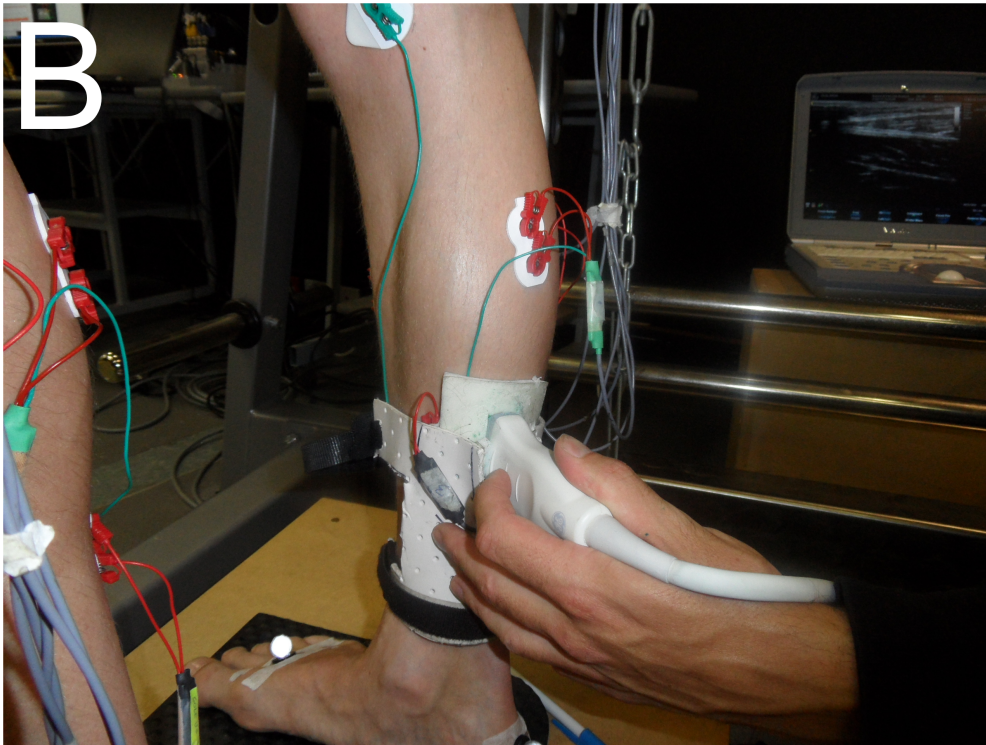
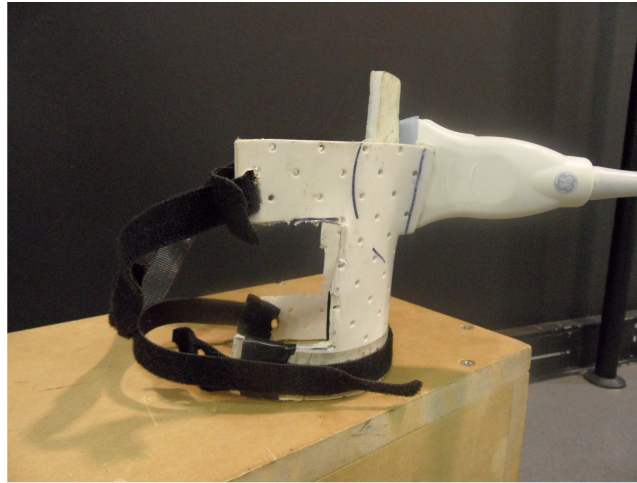
A

Figure 3.3. The custom-built brace moulded to the shape of the ultrasound probe and to the shape of an average gastrocnemius muscle (A). The velcro straps were used to fasten the brace firmly with the leg and the ultrasound probe is held to maintain the clearest and most consistent image and to reduce parallax error (B).

3.4 Data analysis

A six-camera VICON[®] motion analysis system was used to measure the hopping, jumping and running tests (Oxford Metrics Limited, U.K.). Retroreflective markers were placed unilaterally on each segment of the lower body according to the requirements of the plug-in-gait model of VICON[®]. The results reported in this study include error that may be present in external factors (skin marker movement), within the measurement system or, inherent in physiological performance. It is difficult to separate these out and, therefore, they are included in the reliability assessments of measurements of this thesis. However, it was possible for marker placement to be considered and to reduce potential sources of extrinsic error, the principal researcher (CJ) performed all anatomical measurements and undertook all marker placements on participants during all trials. Inter-session reliability of the principal researcher in charge for marker placement and anthropometry was established during pilot testing and all error in joint angles and rotations were less than 5° (McGinley, Goldie, Greenwood, & Olney, 2003). During hopping, participants were instructed to hop on a force platform (Kistler, model 9268BA, Switzerland) and kinetic and kinematic data were processed using custom-written software (LabVIEW, National Instruments, Version 8.2, U.S.A.). All kinematic data were filtered using a zero-lag low-pass, fourth-order Butterworth filter with a cut-off frequency of 8 Hz, and force data were filtered at 50 Hz (Williams, et al., 2004). In Chapter 4, kinematic data were sampled at 100 Hz and ground reaction forces were sampled at 1000 Hz,

while kinematic data in Chapters 5 & 6 were sampled at 120 Hz and ground reaction forces were sampled at 1200 Hz. All force platforms were set with a 15 N ground contact threshold.

3.4.1 Musculoskeletal stiffness analysis

The spring-mass model was used to represent the overall stiffness of the leg (McMahon & Cheng, 1990). Vertical leg stiffness (K_{vert}) is the ratio of peak vertical ground reaction force (F_{peak}) and the displacement of the centre of mass (Δy) calculated by integrating the vertical acceleration twice with respect to time (McMahon & Cheng, 1990):

$$K_{vert} \left(\frac{N}{m} \right) = \frac{F_{peak}}{\Delta y} \quad (\text{Equation 3.1})$$

During running, the behaviour of the leg spring system is not completely vertically linear (McMahon & Cheng, 1990), and so forward motion must be taken into account when calculating the compression of the leg spring (ΔL). Appendix E details the method of calculation for ΔL .

Leg stiffness was calculated by:

$$K_{leg} \left(\frac{N}{m} \right) = \frac{F_{peak}}{\Delta L} \quad (\text{Equation 3.2})$$

In this study, we assumed that the ankle and knee joints act like torsional springs (Farley & Morgenroth, 1999). Therefore, joint stiffness of the ankle and knee (K_{ankle} and K_{knee}) were calculated as the ratio of joint moment (ΔM) to angular displacement ($\Delta\theta$) (Gunther & Blickhan, 2002):

$$K_{\text{joint}} \left(\frac{\text{Nm}}{\text{kg}} \right) = \frac{\Delta M}{\Delta\theta} \quad (\text{Equation 3.3})$$

Leg stiffness was calculated during the braking phase (touch-down to maximum knee flexion) of ground contact (Appendix G). Joint stiffness of the knee and ankle was also calculated during the braking phase of ground contact from touch-down to the peak joint flexion of each respective joint (Farley & Morgenroth, 1999) (Appendix G). The kinetic, kinematic and VGRF data were then post-processed using custom-written software (LabVIEW, National Instruments, Version 8.2, U.S.).

3.4.2 EMG collection and analysis

Electromyographic activity (EMG) was measured from the medial gastrocnemius (MG), lateral gastrocnemius (LG), tibialis anterior (TA), soleus (SOL), peroneus longus (PL) and vastus lateralis (VL) of each leg of each participant (Figure 3.4). During all MVC trials, a visual inspection of the VL muscle activity was performed to ensure that no knee flexion was occurring. The reference electrode was placed on the medial epicondyle of the femur of

each leg. The EMG data were amplified at the source (1000 Hz) and collected at 1200 Hz using a Bortec AMT-16 surface EMG measurement system (Bortec Medical, Alberta, Canada) and synchronised with the VICON[®] capture system. Placement of each disposable Ag/AgCl circular bipolar electrode (Myotronics, Kent, Washington) was in line with the standards outlined by SENIAM (SENIAM, 2005). The electrodes, 10 mm in diameter with an inter-electrode distance of 19 mm (center to center), and an adhesive conducting gel, were positioned on the skin covering the muscles of interest. Prior to attaching the electrodes, the skin surface at the electrode location was shaved, abraded, and cleaned with alcohol. Skin impedance was checked to ensure correct preparation and electrodes were replaced if greater than 5 k Ω . All electrodes were reinforced with tape (Micropore, 3M, St. Paul, Minneapolis) to ensure optimal skin contact.

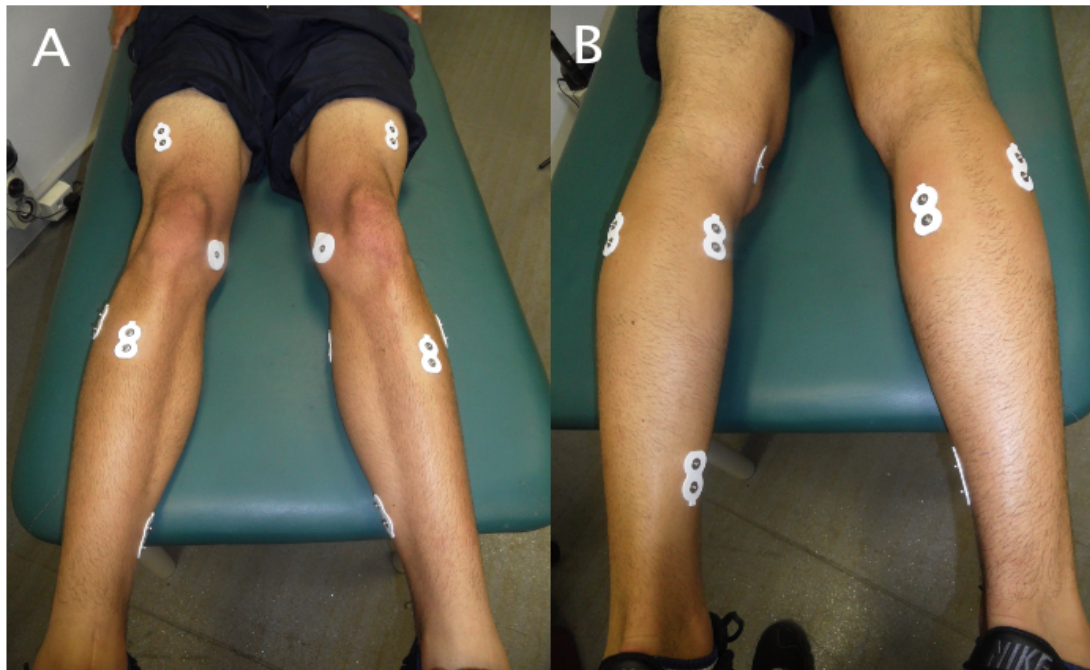


Figure 3.4. EMG electrode placement. Placement of the reference electrode, tibialis anterior and, peroneus longus can be seen in A. Placement of the soleus, medial and lateral gastrocnemius can be seen in B.

Post processing of the EMG data involved filtering using a 16-500 Hz bandpass filter to remove signal artefact, followed by the application of a 10-point moving average filter, before root mean square (RMS) full-wave rectification (Ritchie, Paterson, Bryant, Bartold, & Clark, 2011). These data were then normalised to the values obtained during an isometric ankle plantar flexion MVC performed in the modified squat rack. Mean EMG results were then calculated based on the work of Hobara (2010), whereby this measure was calculated from 100 ms prior to ground contact until ground contact (pre-activity; PRE), from ground contact until 30 ms after ground contact

(background activity; BGA), between 30 and 60 ms after ground contact (short-latency reflex activity; M1), and from 60 to 90 ms after ground contact (long-latency reflex activity; M2) (Figure 3.5).

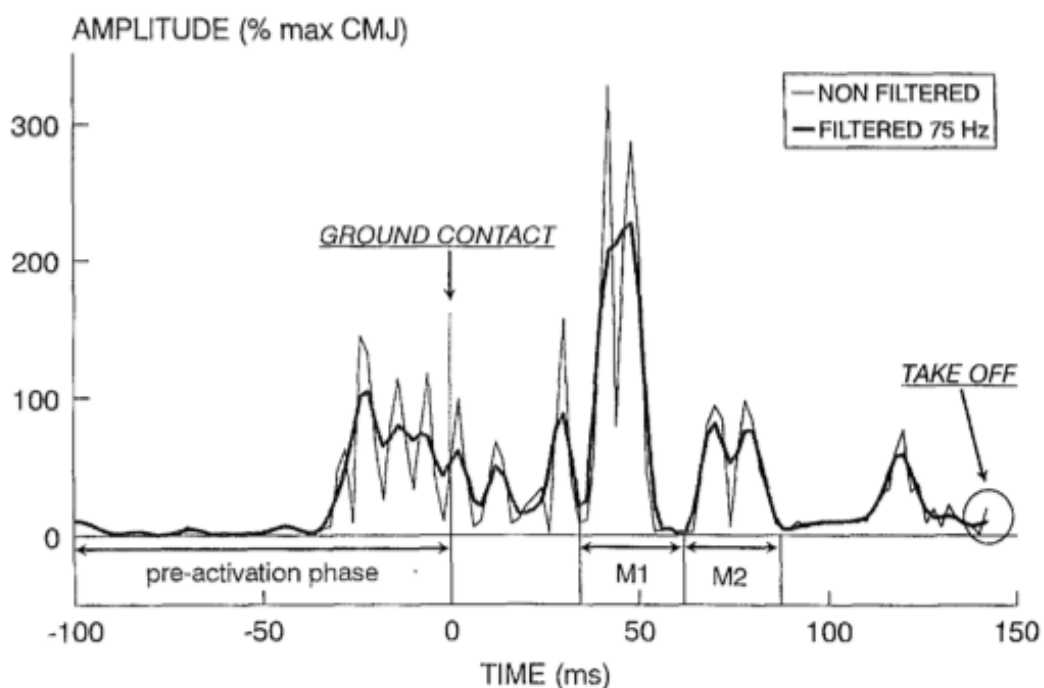


Figure 3.5. An example of the time points at which EMG was analysed during hopping. Pre-activation phase (PRE) was during the 100 ms before ground contact; background activity (BGA) was during ground contact and 30 ms; M1 was during the 30 to 60 ms after ground contact, and M2 was analysed during the 60 to 90 ms after ground contact (Horita, Komi, Nicol, & Kyrolainen, 1996).

Whilst the protocol of Hobara (2010) was replicated, EMG data in this study were not filtered using a 10 Hz envelope. Hopping is a dynamic task

and, therefore, RMS rectification and smoothing (10-point moving average) is sensitive enough to provide an accurate method to measure any differences that may occur at each time point. This procedure is described and illustrated in Figure 3.6. The procedure for the treatment of raw EMG values has been described in the previous paragraph.

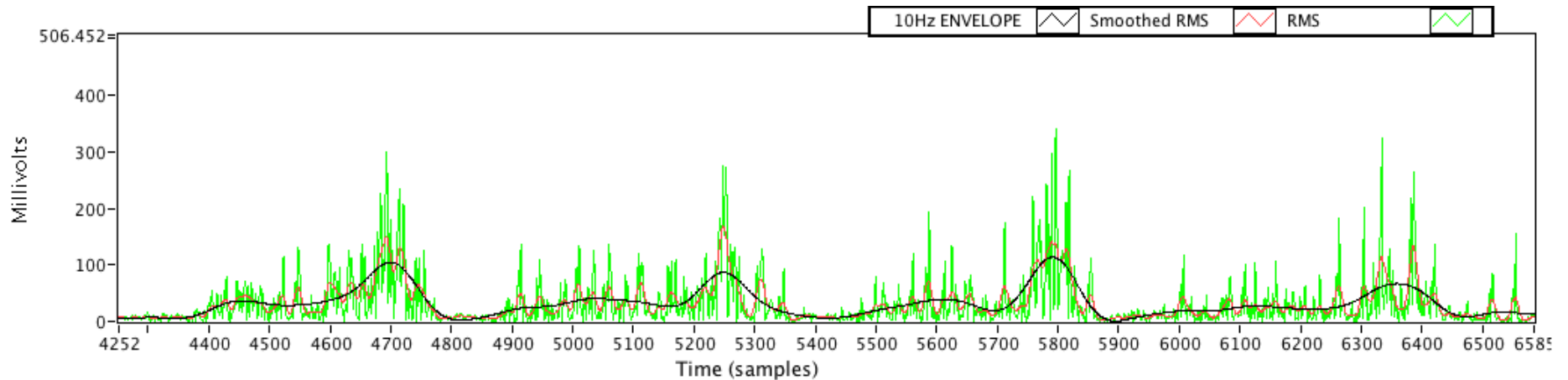


Figure 3.6. Justification for EMG filtering technique. This figure is a sample of soleus EMG data taken from four hops taken after 4 seconds of straight-legged hopping at 2.2 Hz on the dominant limb. Here the difference between RMS rectified raw EMG data (green; RMS), EMG data smoothed using a 10-point moving average filter (red) and, EMG data filtered using a 10Hz linear envelope (black) is shown. As illustrated in the above figure, the smoothed RMS method is able to detect rapid fluctuations in EMG data unlike, the linear 10 Hz envelope method. Therefore, the smoothed RMS technique was chosen as the method used to treat the EMG data in this study because during dynamic movements, such as hopping, it is sensitive enough to detect higher frequency EMG fluctuations.

3.4.3 Achilles tendon moment arm length and Achilles tendon thickness

Achilles tendon moment arm length and Achilles tendon thickness can be measured by magnetic resonance imaging (MRI) (Maganaris, Baltzopoulos, & Sargeant, 1998). However, MRI was not available during this study and, therefore, we estimated these using a combination of ultrasonography and anthropometry (Bryant, et al., 2008). Achilles tendon thickness was measured from stored ultrasound images. Thickness was determined from the distance between the anterior margin and the posterior margin of the tendon (Figure 6.7) and directly horizontal to the predetermined medial malleoli of the ankle (Bryant, et al., 2008).

The ankle joint centre was located by marking the surface of the skin, along an imaginary line that passes through the transmalleolar axis, at the location of the lateral and medial malleoli. To determine Achilles tendon moment arm length, the thickness of the skin together with the midpoint of tendon thickness, as determined from stored ultrasonography images, was subtracted from the distance to the medial malleoli of the ankle (Figure 3.7). This was again performed using ImageJ software (version 1.44O). This method cannot be considered as accurate as MRI but, it has been determined to be reliable (ICC = 0.89) (Clark, et al., 2007), and provides at least the ability to consider the effect of mechanical leverage on Achilles tendon strain.

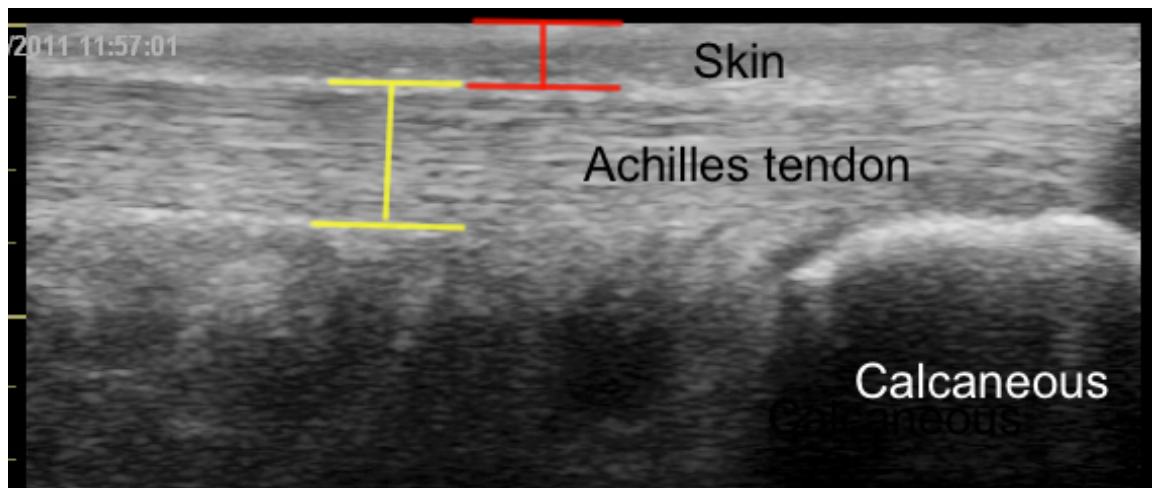


Figure 3.7. Sagittal plane ultrasound image of the Achilles tendon, skin and calcaneus. To estimate Achilles tendon moment arm length, Achilles tendon thickness was measured between the anterior margin and the posterior margin of the tendon (yellow lines). The midpoint of the Achilles tendon was calculated along with the thickness of the skin (red lines) and subtracted from the distance between the medial malleoli of the ankle and the skin over the posterior surface of the Achilles tendon.

During isometric plantar flexion, a small heel lift can occur and, as a result, a change in ankle angle follows (Magnusson, et al., 2001; Muramatsu, et al., 2001). This change in ankle angle translates to an increase in Achilles tendon moment arm and, therefore, affects the vertical displacement of the tendon (Maganaris, et al., 1998; Magnusson, et al., 2001). Therefore, ankle joint rotation was measured using the VICON[®] system and incorporated to calculate the change in Achilles tendon moment arm length shift and then correct for the vertical displacement of the ATJ. This was then deducted from total ATJ displacement. Horizontal displacement of tendon was also calculated however, it was not corrected for as all horizontal displacement was < 6 mm. This range is deemed negligible to ATJ displacement as it is similar to the actual horizontal movement of the Achilles tendon during

isometric MVC of the plantar flexors from rest (Maganaris, et al., 1998). There was no change in ankle angle during this study.

3.4.4 Achilles tendon force analysis

Achilles tendon force was calculated by dividing the ankle joint moment by the Achilles tendon moment arm (the point between the Achilles tendon insertion point and the ankle joint center of rotation, as described in the previous section) (Maganaris & Paul, 1999). This was calculated at MVC and assumed that all of the plantar flexor moment is contributed via the Achilles tendon structure (Lichtwark & Wilson, 2005). Achilles tendon stiffness, strain and plantar flexor force reliability were measured on five individuals over 2 days, 5-7 days apart. Results revealed high reliability (from ICC analysis) for AT stiffness (ICC = 0.86), AT strain (ICC = 0.88) and plantar flexor force (ICC = 0.85).

3.4.5 Achilles tendon strain and stiffness

The ultrasound video of the ATJ taken from rest to MVC of the plantar flexor was converted into individual frames using VLC software (Version 2, Boston, USA) (Figure 3.8). The individual frames were then analysed using a free image analysis software tool developed by the US National Institute of Health (ImageJ, Version 1.44O, <http://rsb.info.nih.gov/nih-image/>).

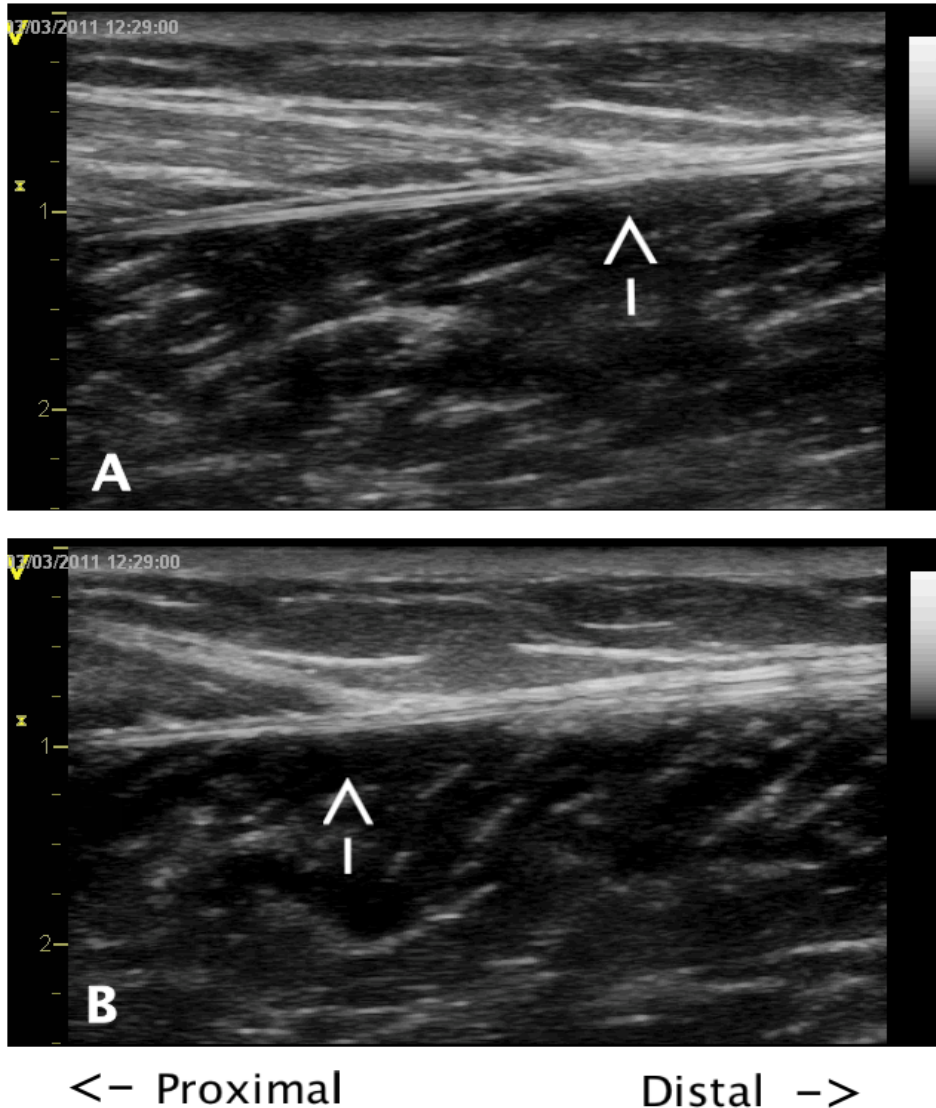


Figure 3.8. Sagittal plane ultrasound images over the distal myotendinous junction of the medial gastrocnemius muscle for one participant. The myotendinous junction, indicated by white arrows, moves proximally in the transition from rest (A) to maximal voluntary isometric plantar flexion (B).

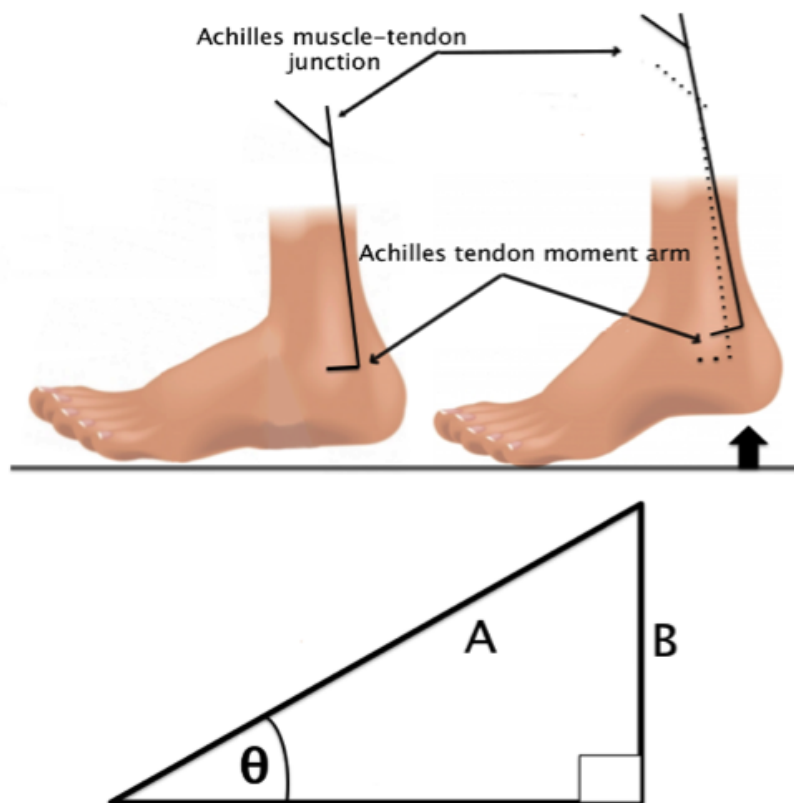
A custom-written program in LabVIEW[®] was used to determine the point at which MVC force occurred and, therefore, the corresponding ultrasound video frame of the ATJ. Force data recorded during the plantar flexor MVC were filtered using a 10-point moving average filter and any trials that exhibited a transient force peak of > 15% of the observed force peak plateau or which did not reach a stable plateau in peak force levels (assessed via visual inspection) were repeated. The

peak force plateau was determined as the segment in the force trace that had the lowest coefficient of variation over a one second timeframe. Three frames corresponding to this peak force plateau were identified and averaged to determine ATJ position; (1) the first data sample, (2) the median data sample, and (3) the final sample of the force plateau (Bryant, et al., 2008). The respective matching force values for these time points were also recorded.

To account for a proximal-distal and anterior-posterior shift in ATJ during MVC, trigonometry was used to solve and correct any effect vertical or horizontal displacement may have had on ATJ displacement and, therefore, strain results (Figure 3.9) (Maganaris, et al., 1998; Magnusson, et al., 2001). The pixel position of the ATJ at rest was recorded and subtracted from the three ATJ pixel positions during MVC to determine the corrected location. Achilles tendon strain was then calculated by:

$$\text{Achilles tendon strain (\%)} = [(L_{MVC} / L_i) - 1] \times 100 \quad (\text{Eq. 3.1})$$

where L_{MVC} is the tendon length at MVC and L_i is the resting tendon length. Achilles tendon stiffness was calculated as the ratio of AT force, as determined from the steadiest part of the peak in the force trace, and the corresponding ATJ displacement. This was taken during the final 20% of MVC.



A = distance from the metatarsalphalangeal joint to the ankle joint center

$$B = \sin\theta \times A$$

θ = change in ankle angle

Figure 3.9. Calculation for the displacement of the Achilles tendon junction that occurs during ankle joint rotation. To correct for the heel lift that occurs during MVC of the plantar flexors, B is deducted from total Achilles tendon displacement.

It has been suggested that the initial resting length of the Achilles tendon should be taken at approximately 5% of average maximum force due to the tow region of tendon behaviour (Lichtwark & Wilson, 2005). However, it is likely to only affect strain results by 1% (Lichtwark & Wilson, 2005) and, therefore, Achilles tendon length was measured whilst standing at rest.

Although there were slight movements vertical and horizontally in the leg during MVC, given that the probe is attached to the leg and fixed to the skin, any movement in the leg is occurring at the probe simultaneously and, therefore, correction procedures are not necessary (Maganaris, 2005; Maganaris, Baltzopoulos, & Sargeant, 2006). Additionally, during MVC there is some shift in the skin covering the ATJ and fixing the probe to the skin does not account for this movement. Without fixing a reference point such as a bony landmark, this movement is difficult to measure however, it is thought to be negligible (Maganaris, et al., 2006).

3.5 Statistical analysis

Prior to statistical analysis, all data were checked for normality based on a critical appraisal approach (Peat & Barton, 2005, p. 46). Normality was assessed by checking the differences between mean and median values, skewness and kurtosis values, Shapiro-Wilk test results, multiplied two standard deviations compared to means, and exploring plots. If two or more criteria were breached then non-parametric statistical tests were used. All data were analysed using SPSS (Version 17.0, Chicago, IL) and statistical significance was set at $p < 0.05$. Specific analyses for each study are described in Chapters 4 to 6.

3.5.1 Statistical power

For the first thesis study in Chapter 4, twenty participants were recruited. This sample size was selected to detect differences at a significance level of 0.05

and a power of 90% and was based on results of earlier work investigating the reliability of MSS (McLachlan, et al., 2006).

The sample size estimation for the studies in Chapter 5 & 6 were based on previously reported data of Dutto and Braun (2004) who investigated the DOMS associated changes in MSS. Based on the between group mean difference of 4.2 kN/m and SD of 2.2 kN/m in leg MSS, a size of 8 subjects were required for a significance of $p < 0.01$ and 90% power. However, to be conservative twenty participants were recruited. All sample size and power calculations were performed using G*Power software (Faul, Erdfelder, Buchner, & Lang, 2009).

Chapter 4. The inter-day reliability of ankle, knee, leg, and vertical musculoskeletal stiffness during hopping, jumping and over-ground running.

4.1 Introduction

The biomechanical behaviour of the lower extremity during running and hopping activities has been suggested to simulate that of a spring-mass system (Blickhan, 1989). Part of this spring model is the concept known as musculoskeletal stiffness (MSS) which describes the ability of the leg spring to resist a change in length when loaded (McMahon & Cheng, 1990). Musculoskeletal stiffness has been shown to be important to athletic performance however, optimal levels for performance are not clearly defined (Brughelli & Cronin, 2008a, 2008b). During running at speeds of 2.5 to 10.26 m/s, increases in vertical and knee MSS are observed with increases in running velocity and stride rate; however, leg and ankle MSS can remain unchanged (Farley & Gonzalez, 1996; He, et al., 1991; Kuitunen, Komi, et al., 2002). A number of studies have also assessed MSS during hopping or jumping tasks (Arampatzis, Schade, et al., 2001; Farley, et al., 1991; Hobara, et al., 2007; McLachlan, et al., 2006). Levels of MSS are greatly influenced by hopping frequency and hop displacement. Increases in hopping frequency and/or displacement leads to overall increases in leg MSS (Farley, et al., 1991; Farley & Gonzalez, 1996), which is predominantly due to increases in ankle joint MSS (Farley & Morgenroth, 1999).

While reliability has been determined for some specific biomechanical methods of MSS assessment (Allison, et al., 1998; Hunter & Spriggs, 2000; McLachlan, et al., 2006; Murphy, Watsford, Coutts, & Pine, 2003), there is a paucity of research investigating the reliability of MSS during running and hopping tasks,

particularly when performed at a self-selected velocity or frequency. Furthermore, when using interventions to explore changes in MSS, it is imperative to understand the reliability of the chosen testing procedures and the session-to-session variation in MSS inherent in a given test protocol and method of analysis. Having this knowledge would better enable the researcher to determine the changes in MSS that may be a result of the intervention itself, the inherent method error or the natural, day-to-day variation in task performance. Moreover, providing absolute and relative results of reliability enables the experimenter to compare equipment or testing procedures, perform *a priori* sample size estimations, and provides an opportunity to make methodological adjustments to prevent resources and participant's time being wasted with unreliable testing procedures.

4.1.1 Aims and hypothesis

The aim of this study was to establish the reliability of laboratory-based biomechanical measurements of vertical, leg, knee and ankle joint MSS during running and hopping tasks. It was hypothesised that global MSS measures (vertical, leg) of running and hopping at a set pace (velocity or frequency) would be reliable, whilst the same tasks performed at a self-selected pace would exhibit poor reliability. Further it was hypothesised that joint MSS measures of running and hopping at either a set or self-selected pace would be less reliable than global measures due to increased task performance variation in joint movement patterns.

4.2 Methods and procedure

To ensure the aims of this study were met, the suitability of participants for this study was assessed as outlined in Chapter 3.2. In summary, each participant

filled out a health screening questionnaire that included questions regarding any current or pre-existing medical condition or injury. If a potential candidate had any condition or injury that was likely to affect their performance in this study then, they were excluded.

4.2.1 Participants

Twenty active men (age: 22.3 ± 3.0 y; mass: 74.7 ± 5.6 kg; height: 178.7 ± 7.1 cm) from various sporting backgrounds were recruited and were all right-side dominant. As described in Chapter 3.5.1, this sample size was selected to detect differences at a significance level of 0.05 and a power of 90% and was based on results of earlier work investigating the reliability of musculoskeletal stiffness (McLachlan, et al., 2006). The University Ethics Committee approved the study and all participants provided written informed consent (approval reference V200708 109). All participants were injury-free at the time of testing and had not missed a training session or game in their respective sport for six weeks preceding the time of testing. During the initial testing session, anthropometric measurements were taken and participants were assessed for height, weight and laterality (Coren, 1993).

4.2.2 Procedure

Two testing sessions were completed by participants, separated by 3 to 7 days (Mean = 4.35 ± 1.42 days) to avoid any effects from the previous session. Participants returned for their second testing session at the same time of day as the first session. For all sessions, a warm-up was performed consisting of 5 min of

cycling at a self-selected velocity on an ergometer (Monark AB, Sweden) and 5 minutes of self-selected dynamic and static stretching. This protocol was repeated during the subsequent testing session. Participants were given as many practice trials as required to become familiar with all testing protocols. The order of all tasks (running, hopping and jumping) were randomly allocated to each participant. For a detailed description of the hopping, jumping and running protocols, see Chapter 3.3.3 and 3.3.4, respectively. However, the protocols are briefly described below.

During ten trials of running (RUN), kinetics, kinematics and vertical ground reaction forces (VGRF) were recorded. Each participant was required to run at 3.35 m/s (Williams, et al., 2004) along a 10 m runway. Participants contacted the force plate with their left foot for five trials and their right foot for the remaining five trials. Only trials that exhibited a clean foot contact with the force plate were included in the analysis. The order in which leg the participant began on was randomised by the researcher so that a random order of force plate foot strikes occurred. This was performed to negate a possible order effect

Continuous straight-legged hopping (CH) and continuous bent-legged jumping (CJ) tasks were performed by all participants. The CH and CJ tasks were performed at the participant's self-selected frequency (CHSS and CJSS) and also at a frequency of 2.2 Hz (CH2.2 and CJ2.2). For these two tasks: (1) participants were required to perform straight-legged hopping, locking their knees to reduce the input of the knees and, thus, predominantly requiring the ankle joint to perform the task - this test was used to assess ankle flexor/extensor input (Hobara, et al., 2007); (2) perform bent-legged hopping without attempting to restrict the movement of any joint. Participants were asked to perform the CH and CJ tasks on the force platform for 10 continuous hops or jumps (Hobara, et al., 2007). The CH and CJ

tasks were performed on the left, right and both legs together and a digital metronome controlled hopping frequency.

4.2.3 Data collection and analysis

Greater detail of the methods used in this chapter are outlined in Chapter 3.4 and 3.4.1, respectively. For the RUN task, means for individual legs were not different ($p>0.05$) and results were subsequently pooled. Data from the CJSS task were removed from analysis due to the inability of the participants to correctly perform the task resulting in large variations in all variables.

Retroreflective markers were placed unilaterally on each segment of the lower body according to the requirements of the plug-in-gait model of VICON®. Kinematic data was collected at 100 Hz and kinetic data at 1000 Hz. To reduce potential sources of extrinsic error, the principal researcher (CJ) performed all anatomical measurements and undertook all marker placements on participants during all trials.

Vertical, leg, knee and ankle stiffness were examined in this study. Vertical stiffness was calculated by the ratio of peak vertical ground reaction force (F_{peak}) to centre of mass displacement (Δy):

$$K_{vert} \left(\frac{N}{m} \right) = \frac{F_{peak}}{\Delta y} \quad (\text{Eq. 3.2})$$

During running, leg stiffness was calculated by:

$$K_{leg} \left(\frac{N}{m} \right) = \frac{F_{peak}}{\Delta L} \quad (\text{Eq. 3.3})$$

where, ΔL is the compression of the leg spring. Joint stiffness of the ankle and knee (K_{ankle} and K_{knee}) were also calculated as the ratio of joint moment (ΔM) to angular displacement ($\Delta\theta$) (Gunther & Blickhan, 2002):

$$K_{\text{joint}} \left(\frac{\text{Nm}}{\text{kg}} \right) = \frac{\Delta M}{\Delta\theta} \quad (\text{Eq. 3.4})$$

Joint moments were calculated via the plug-in-gait model of the VICON[®] software. For calculation of the variables reported in this study, these data were aligned by down sampling the kinetic data to 100Hz to match the kinematic data. Although this reduces the resolution of the kinetic data traces, the relevant kinetic and kinematic data at the time point of interest (i.e. peak joint flexion) is not fluctuating at a high magnitude or frequency and, therefore, this down sampling has negligible effect on the values derived.

Data from the bent-legged hopping task (unrestricted knee flexion) at self-selected frequency were removed from analysis due to the inability of the participants to correctly perform the task resulting in large variations in all variables.

4.2.4 Statistics

All data were normally distributed based on based on a critical appraisal approach (Peat & Barton, 2005). Descriptive statistics including means and standard deviations were calculated for all variables and the differences between means for all variables were assessed using paired samples t-tests. All data were

analysed using SPSS (Version 17.0, Chicago, IL) and statistical significance was set at $p < 0.05$.

To determine reliability, criteria of 'relative' and 'absolute' reliability measures were chosen (Atkinson & Nevill, 1998; Bradshaw, Hume, Calton, & Aisbett, 2010). These measures were; difference in the mean (Mdiff%), intraclass correlation coefficients ($ICC_{3,1}$) (Hopkins, 2000), measurement error (typical error) as expressed as a coefficient of variation percentage ($CV_{ME}\%$) (Hopkins, 2000), and Cohen's effect size (ES).

Cohen's ES were interpreted as < 0.1 being trivial, 0.1 to 0.6 being small, 0.6 to 1.2 being moderate, and > 1.2 being large (Saunders, Pyne, Telford, & Hawley, 2004). Furthermore, to help in the comparison of the present study with other methodologies, 95% limits of agreement (95% LOA) were also reported (Atkinson & Nevill, 1998; Bland & Altman, 1999). In defining an overall rating of 'good' reliability, the criteria threshold of $Mdiff\% < 5\%$, $ICC \geq 0.80$, $CV_{ME}\% \leq 10\%$ and $ES < 0.6$ all had to be satisfied. For 'moderate' reliability, all but one of the four criteria had to be met, while 'poor' reliability was defined as two or more set criteria not being met.

4.3 Results

No differences were identified for any of the stiffness measures between day 1 and day 2 ($p > 0.05$). Summaries of the descriptive and reliability statistics for the MSS variables (K_{leg} , K_{vert} , K_{ank} , and K_{knee}) during all tasks (RUN, CH2.2, CJ2.2 and CHSS) are shown in Tables 4.1 and 4.2.

Table 4.1. Musculoskeletal stiffness reliability statistics and overall determination of reliability for the running task.

Test	Day 1	Day 2	ICC	Mdiff%	CV_{ME}%	ES	95% LOA	Reliability
RUN								
K _{vert}	30.14 (6.53)	31.13 (7.81)	0.87	3.27	9.4	0.14	26.20	Good
K _{leg}	5.91 (6.96)	5.97 (6.91)	0.85	4.80	49.8	0.04	137.90	Moderate
K _{ank}	0.22 (0.06)	0.22 (0.06)	0.51	0.00	23.3	0.01	64.53	Poor
K _{knee}	0.29 (0.09)	0.30 (0.10)	0.56	1.48	25.4	0.05	70.34	Poor

RUN refers to the running task; K_{leg} and K_{vert} values are measured in kN/m; K_{ank} and K_{knee} values in Nm/kg/deg for all tasks. Day 1 and 2 values are mean and standard deviations (SD). Intraclass correlation (ICC); differences between means (Mdiff%), coefficient of variation (CV_{ME}%), limits of agreement (95% LOA) and effect size (ES).

Table 4.2. Musculoskeletal stiffness reliability statistics and overall determination of reliability for continuous jumping and hopping tasks.

Test	Day 1	Day 2	ICC	Mdiff%	CV_{ME}%	ES	95% LOA	Reliability
CJ2.2								
Both legs								
K _{vert}	56.46 (3.35)	54.71 (7.67)	0.82	3.10	6.9	-0.25	19.18	Good
K _{ank}	0.17 (0.11)	0.21 (0.17)	0.92	25.52	60.3	-0.03	167.28	Poor
K _{knee}	0.26 (0.12)	0.28 (0.15)	0.79	7.59	51.0	0.22	141.34	Poor
Left leg								
K _{vert}	47.45 (6.44)	48.90 (5.58)	0.84	3.04	5.5	0.34	15.49	Good
K _{ank}	0.16 (0.11)	0.15 (0.08)	0.81	8.32	31.9	0.23	88.50	Poor
K _{knee}	0.35 (0.15)	0.35 (0.14)	0.69	0.68	23.2	-0.05	64.45	Poor
Right leg								
K _{vert}	46.94 (5.74)	46.05 (10.14)	0.89	1.90	5.4	-0.02	14.97	Good
K _{ank}	0.21 (0.10)	0.20 (0.11)	0.81	4.24	24.9	0.85	67.52	Poor
K _{knee}	0.19 (0.07)	0.18 (0.09)	0.68	6.86	29.1	-0.17	80.55	Poor
CH2.2								
Both legs								
K _{vert}	57.72 (6.46)	57.87 (5.15)	0.82	0.25	5.5	0.03	15.19	Good
K _{ank}	0.31 (0.12)	0.27 (0.14)	0.61	13.40	27.9	-0.62	77.34	Poor
K _{knee}	0.44 (0.20)	0.44 (0.21)	0.88	1.27	22.1	0.82	60.20	Poor
Left leg								
K _{vert}	49.62 (9.73)	51.03 (8.17)	0.84	2.83	9.2	0.09	20.90	Good
K _{ank}	0.14 (0.09)	0.13 (0.07)	0.71	3.26	38.0	0.41	105.34	Poor
K _{knee}	0.27 (0.13)	0.24 (0.14)	0.79	11.63	51.0	0.22	141.34	Poor
Right leg								
K _{vert}	46.82 (7.17)	48.90 (6.23)	0.89	4.45	7.6	0.04	20.90	Good
K _{ank}	0.22 (0.09)	0.18 (0.07)	0.68	18.54	28.1	-0.29	77.80	Poor
K _{knee}	0.17 (0.09)	0.20 (0.09)	0.68	17.16	29.1	-0.17	80.55	Poor
CHSS								
Both legs								
K _{vert}	50.05 (10.65)	47.84 (10.18)	0.86	4.42	10.2	-0.21	28.24	Moderate
K _{ank}	0.25 (0.09)	0.30 (0.08)	0.86	17.91	353	0.53	97.72	Poor
K _{knee}	0.32 (0.14)	0.32 (0.20)	0.85	0.71	24.6	0.01	68.25	Poor
Left leg								
K _{vert}	37.62 (9.42)	39.27 (6.73)	0.61	4.38	16.1	0.20	54.62	Poor
K _{ank}	0.16 (0.10)	0.18 (0.10)	0.91	12.30	22.4	0.12	63.70	Poor
K _{knee}	0.32 (0.20)	0.34 (0.13)	0.83	5.27	26.3	0.75	72.71	Poor
Right leg								
K _{vert}	39.51 (7.67)	41.71 (9.14)	0.20	5.58	19.7	0.26	54.62	Poor
K _{ank}	0.16 (0.08)	0.16 (0.06)	0.86	4.12	23.0	0.09	63.70	Moderate
K _{knee}	0.26 (0.13)	0.25 (0.13)	0.82	2.01	26.0	0.10	72.06	Moderate

CJ2.2 refers to the continuous jumping task at 2.2 Hz; CH2.2 refers to the continuous hopping task at 2.2 Hz; CHSS refers to the continuous jumping task at self-selected frequency. K_{leg} and K_{vert} values are measured in kN/m; K_{ank} and K_{knee} values in Nm/kg/deg for all tasks. Day 1 and 2 values are mean and standard deviations (SD). Intraclass correlation (ICC); differences between means (Mdiff%), coefficient of variation (CV_{ME}%), limits of agreement (95% LOA) and effect size (ES).

Global MSS results exposed 'good' to 'moderate' reliability during the set paced tasks (RUN, CJ2.2 and CH2.2). Vertical MSS (K_{vert}) exhibited 'good' reliability between testing sessions for all the RUN, CJ2.2, and CH2.2 tasks, with small $M_{\text{diff}}\%$ (0.25 to 4.80%), trivial to small Cohen's ES (0.02 to 0.34), and acceptable ICC ($r \geq 0.80$) and CV% ($\leq 10\%$). During the RUN task, K_{leg} had 'good' reliability [(ICC = 0.85; $M_{\text{diff}}\%$ = 4.80%; ES = 0.04)], despite a CV% of 49.8.

In contrast, K_{vert} during the self-paced CHSS task, performed using a single leg, showed 'poor' reliability, and only moderate reliability when performed two-legged. Reliability results were 'poor' to 'moderate' for K_{ank} and K_{knee} during all tasks. Only two of the set criteria for K_{ank} and K_{knee} were met concurrently. ICCs were within the acceptable range ($r \geq 0.80$) only during the CJ2.2 and CHSS conditions. Scores for $CV_{\text{ME}}\%$ ranged from 22.1 to 68.2%, ES from 0.02 to 0.85, and $M_{\text{diff}}\%$ from 0.68 to 18.90%.

Assessment of joint kinetics and kinematics were performed to provide insight into the reliability results of joint MSS. Differences from day 1 to day 2 were identified in knee angular displacement during the CH2.2 two-legged task ($p = 0.048$) and ankle angular displacement during the CJ2.2 right-legged task ($p = 0.025$). Peak VGRF and centre of mass (COM) displacement demonstrated 'good' ICCs during the CH, CJ and RUN tasks; however, peak knee moment had acceptable ICCs ($r \geq 0.80$) only during the RUN task and the CH2.2 task in one-legged hopping (Tables 4.3, 4.4 and 4.5). Peak ankle moment displayed 'good' ICCs during the left-legged CJ2.2 task, while ankle angular displacement reported 'good' reliability during the right-legged CH2.2 task. All other kinematic variables displayed 'moderate' to 'poor' ICCs ($r = 0.14$ to 0.79). Mean difference results were largely mixed. Peak VGRF showed only small changes in means between days

(Mdiff% \leq 5%); however, results for kinetics and kinematics showed greater differences (Mdiff% = 0.57 to 20.89). Interestingly, large changes in means from day 1 to day 2 still led to relative reliability ICC scores being above $r = 0.80$ (e.g. RUN peak ankle moment: Mdiff% = 10.31 and ICC = 0.83). Hopping frequency had 'good' reliability and showed little day-to-day variation during the CJSS task (Tables 4.3 and 4.4).

Table 4.3. Kinetic, kinematic and VGRF results during the jumping (CJ) and hopping (CH) tasks using both legs.

	Both Day 1	Day 2	Mdiff%	ICC
2.2 Hz				
CJ				
Peak VGRF (kN)	2.84 (0.37)	2.92 (0.42)	2.18	0.83
Peak VGRF (Bodyweights)	34.38 (12.68)	39.25 (5.73)	1.79	0.86
COM displacement (m)	0.05 (0.01)	0.05 (0.01)	0.57	0.99
Peak ankle moment (Nm/kg)	1.76 (0.74)	1.93 (0.89)	5.19	0.14
Peak knee moment (Nm/kg)	6.22 (2.55)	5.68 (1.62)	10.31	0.52
Ankle angular displacement (deg)	19.81(4.80)	19.09 (5.64)	1.90	0.66
Knee angular displacement (deg)	42.26 (4.86)	41.49 (5.98)	6.02	0.78
2.2 Hz				
CH				
Peak VGRF (kN)	2.80 (0.45)	2.89 (0.32)	0.43	0.83
Peak VGRF (Bodyweights)	39.12 (5.28)	38.88 (4.05)	0.61	0.85
COM displacement (m)	0.05 (0.01)	0.05 (0.01)	8.49	0.97
Peak ankle moment (Nm/kg)	6.34 (2.48)	6.00 (3.15)	4.19	0.45
Peak knee moment (Nm/kg)	5.88 (2.84)	5.94 (2.23)	1.68	0.57
Ankle angular displacement (deg)	15.01 (3.81)	13.96 (3.97)	1.81	0.73
Knee angular displacement (deg)	32.64 (6.17)	34.83 (3.23)*	0.79	0.78
Self-selected bent				
CJ				
Frequency (Hz)	1.90 (0.28)	1.89 (0.23)	0.79	0.87
Peak VGRF (kN)	3.76 (0.50)	3.89 (0.42)	2.91	0.85
Peak VGRF (Bodyweights)	50.76 (7.44)	52.18 (5.84)	2.14	0.87
COM displacement (m)	0.08 (0.03)	0.08 (0.02)	2.91	0.91
Peak ankle moment (Nm/kg)	8.66 (2.84)	10.27 (3.41)	13.31	0.70
Peak knee moment (Nm/kg)	9.82 (4.40)	8.91 (3.33)	0.19	0.77
Ankle angular displacement (deg)	19.87 (7.51)	17.82 (5.64)	8.12	0.79
Knee angular displacement (deg)	42.53 (11.60)	43.56 (5.68)	6.78	0.78

* Signifies a significant difference between day 1 and day 2 ($p < 0.05$). CJ2.2 refers to the continuous jumping task at 2.2 Hz; CH2.2 refers to the continuous hopping task at 2.2 Hz and CHSS refers to the continuous jumping task at self-selected frequency. Day 1 and 2 values are mean and standard deviations (SD). Reliability results are intraclass correlations (ICC) and differences between means (Mdiff%).

Table 4.4. Kinetic, kinematic and VGRF results during the jumping (CJ) and hopping (CH) tasks using single legs.

	Left				Right			
	Day 1	Day 2	Mdiff%	ICC	Day 1	Day 2	Mdiff%	ICC
2.2 Hz								
CJ								
Peak VGRF (kN)	2.20 (0.29)	2.23 (0.25)	1.31	0.80	2.21 (0.28)	2.26 (0.27)	2.01	0.84
Peak VGRF (Bodyweights)	29.51 (3.46)	29.94 (3.28)	1.40	0.85	29.66 (3.50)	30.28 (3.64)	2.09	0.83
COM displacement (m)	0.05 (0.01)	0.05 (0.01)	2.69	0.97	0.05 (0.01)	0.06 (0.04)	12.01	0.81
Peak ankle moment (Nm/kg)	3.78 (2.70)	4.72 (2.78)	15.17	0.83	5.51 (2.22)	6.79 (2.59)	11.88	0.64
Peak knee moment (Nm/kg)	7.38 (2.67)	7.28 (2.54)	2.50	0.82	5.24 (2.59)	4.50 (2.73)	20.89	0.26
Ankle angular displacement (deg)	23.19 (3.98)	21.61 (2.97)	8.38	0.51	22.91 (3.78)	21.36 (2.87)*	7.70	0.71
Knee angular displacement (deg)	43.11 (4.12)	41.44 (3.76)	3.57	0.66	42.29 (3.45)	42.78 (4.00)	4.31	0.72
2.2 Hz								
CH								
Peak VGRF (kN)	2.32 (0.26)	2.35 (0.34)	1.33	0.88	2.24 (0.30)	2.34 (0.33)	2.87	0.87
Peak VGRF (Bodyweights)	31.15 (3.15)	31.31 (4.19)	0.52	0.89	30.03 (3.60)	31.26 (4.44)	2.18	0.87
COM displacement (m)	0.05 (0.01)	0.05 (0.01)	6.56	0.91	0.05 (0.01)	0.06 (0.04)	6.32	0.88
Peak ankle moment (Nm/kg)	4.37 (2.95)	5.36 (2.76)	0.31	0.61	6.08 (2.59)	7.57 (3.09)	12.3	0.61
Peak knee moment (Nm/kg)	8.19 (3.74)	7.23 (2.53)	7.32	0.46	4.82 (2.63)	5.23 (2.16)	0.28	0.81
Ankle angular displacement (deg)	18.13 (8.40)	18.36 (4.36)	2.80	0.64	20.20 (3.93)	18.09 (4.16)	6.79	0.82
Knee angular displacement (deg)	37.54 (5.16)	37.83 (4.95)	3.92	0.43	39.20 (4.10)	38.56 (3.72)	1.11	0.56
Self-selected bent								
CJ								
Frequency (Hz)	2.05 (0.33)	2.14 (0.20)	4.35	0.83	2.07 (0.24)	2.14 (0.21)	3.08	0.92
Peak VGRF (kN)	2.46 (0.43)	2.40 (0.41)	2.42	0.87	2.47 (0.30)	2.43 (0.47)	1.38	0.88
Peak VGRF (Bodyweights)	33.09 (5.73)	32.16 (5.01)	1.94	0.85	33.25 (4.42)	32.67 (5.95)	0.83	0.89
COM displacement (m)	0.07 (0.02)	0.08 (0.02)	6.41	0.92	0.07 (0.01)	0.07 (0.02)	3.82	0.20
Peak ankle moment (Nm/kg)	5.85 (3.02)	6.62 (3.06)	13.01	0.78	7.22 (3.19)	7.75 (3.01)	7.29	0.68
Peak knee moment (Nm/kg)	9.06 (3.01)	8.25 (21.99)	8.50	0.52	7.00 (3.27)	6.67 (2.61)	4.75	0.74
Ankle angular displacement (deg)	20.76 (12.09)	21.65 (5.07)	5.14	0.74	22.61 (6.53)	20.28 (3.96)	9.93	0.69
Knee angular displacement (deg)	42.81(9.84)	42.95 (4.87)	1.46	0.66	44.78 (7.47)	42.39 (4.91)	5.33	0.69

* Signifies a significant difference between day 1 and day 2 ($p < 0.05$). CJ2.2 refers to the continuous jumping task at 2.2 Hz; CH2.2 refers to the continuous hopping task at 2.2 Hz and CHSS refers to the continuous jumping task at self-selected frequency. Day 1 and 2 values are mean and standard deviations (SD). Reliability results are intraclass correlations (ICC) and differences between means (Mdiff%).

Table 4.5. Kinetic, kinematic and VGRF results during the running (RUN) task.

RUN	Day 1	Day 2	Mdiff%	ICC
Peak VGRF (kN)	2.02 (0.22)	2.00 (0.18)	0.93	0.92
Peak VGRF (Bodyweights)	27.15 (2.64)	26.89 (2.08)	0.94	0.90
COM displacement (m)	0.08 (0.02)	0.08 (0.02)	1.53	0.87
Peak ankle moment (Nm/kg)	1.94 (0.99)	1.91 (0.67)	1.71	0.54
Peak knee moment (Nm/kg)	4.43 (0.15)	3.98 (0.14)	10.31	0.83
Ankle angular displacement (deg)	17.52 (4.01)	17.24 (3.02)	1.58	0.52
Knee angular displacement (deg)	30.46 (3.14)	30.70 (2.25)	0.81	0.58
Ankle angle at touch-down (deg)	28.36 (1.98)	28.22 (2.02)	0.50	0.50
Knee angle at touch-down (deg)	49.57 (2.67)	50.17 (2.93)	1.21	0.23
Leg angle at touch-down (deg)	14.53 (3.76)	15.62 (3.77)	6.98	0.89

Day 1 and 2 values are mean and standard deviations (SD). Reliability results are intraclass correlations (ICC) and differences between means (Mdiff%).

4.4 Discussion

There are a range of methodological approaches currently used to measure MSS (Brughelli & Cronin, 2008b), including a number of open and closed kinetic-chain movement protocols (Butler, et al., 2003). However, the reliability of these tests has not been well established. Therefore, the aim of this study was to examine the inter-day reliability of a testing protocol used to assess MSS during over-ground running, jumping and hopping tasks across two testing sessions. The most reliable measure of MSS was K_{vert} during over-ground running and during single- and double-legged hopping and jumping at either a fixed (2.2 Hz) or self-selected frequency. All other variables exhibited moderate to poor reliability.

To our knowledge, this is the first study to investigate magnitudes of K_{leg} , K_{vert} , K_{knee} and K_{ank} during over-ground running. Furthermore, this is the first study to report an array of reliability statistics that account for the absolute and relative reliability of musculoskeletal stiffness. The results for K_{leg} and K_{vert} in this study were similar to previous research (Arampatzis, et al., 1999; Divert, et al., 2005; Morin, et al., 2005), as were those for K_{ank} and K_{knee} data (Arampatzis, et al., 1999; Gunther & Blickhan, 2002). This consistency suggests a level of conformity between the methods adopted in the present study and those of previous methodological approaches.

As hypothesised, 'good' reliability was shown for K_{vert} with high ICC, low $M_{\text{diff}}\%$, low $CV_{\text{ME}}\%$ and small ES results during RUN. However, K_{leg} , K_{ank} and K_{knee} all produced less than acceptable reliability based on the criteria set in this study. Perhaps, the highly variable contributions of K_{ank} and K_{knee} are a result of the joints individually adjusting to maintain an overall consistent K_{leg} .

Given that we did not measure stiffness at the hip joint, it can only be assumed that individual joint stiffness is modulated to maintain an overall level of K_{leg} . The ICC values for the RUN task showed that peak vertical ground reaction force had good repeatability from day-to-day repeatability, consistent with previous research (Ferber, McClay Davis, Williams, & Laughton, 2002; Queen, Gross, & Liu, 2006). Further investigations into the RUN task indicate that sources of variability exist in the kinetic and kinematic results. Centre of mass displacement and peak knee moment displayed 'good' reliability (ICC = 0.83 to 0.97), yet all other kinematic and kinetic variables exhibited 'moderate' to 'poor' ICC scores (0.23 to 0.58). These results are not in agreement with previous research where ICCs ranged from 0.83 to 0.94 (Ferber, et al., 2002). However, it is difficult to compare past work with the methodology in the present study due to differences in the participant cohort, marker set used, runway length and running velocity (Ferber, et al., 2002). Whilst there were no differences between day 1 and 2 for any MSS, VGRF, kinematic or kinetic variable during RUN, there was an increase (3 to 20%) in all MSS means from day 1 to day 2. This suggests that a separate familiarization session may be of benefit to improve between-session reliability therefore, researchers are encouraged to adopt this approach. In addition, there is potential that repeatability in VGRF, kinematic and kinetic data may be improved by participants running at a self-selected pace (Queen, et al., 2006), although this would likely compromise the data due to the effect of running velocity on MSS (Brughelli & Cronin, 2008a).

The single- and double-legged hopping (CH) and jumping (CJ) tasks at 2.2 Hz demonstrated reliable K_{vert} results that compare well with past research

(Farley & Morgenroth, 1999; Hobara, et al., 2010; Hobara, et al., 2008; Padua, et al., 2006). All means, ICCs and ESs for K_{vert} (Hobara, et al., 2010; Lloyd, Oliver, Hughes, & Williams, 2009; McLachlan, et al., 2006) and means for K_{ank} and K_{knee} also compared well with past studies (Hobara, et al., 2010). However, a greater than desired $CV_{\text{ME}}\%$, 'moderate' to 'good' ICC (0.61 to 0.92) and moderate to trivial ES (0.03 to 0.85) were evident and, therefore, deem K_{ank} and K_{knee} during the CH2.2 and CJ2.2 tasks to have a 'poor' overall reliability based on the criteria set in this paper. This finding confirms our second hypothesis. As previously suggested, a potential explanation for the 'poor' reliability in K_{ank} and K_{knee} is a result of the modulation of individual joint MSS contributions to maintain a consistent K_{vert} .

Musculoskeletal stiffness data during the CHSS task resulted in moderate to 'poor' reliability. This finding confirms our second hypothesis. During the single- and double-legged CHSS tasks, K_{vert} met the set reliability criteria, however, K_{ank} and K_{knee} displayed mixed reliability results. Intraclass correlation coefficients for K_{ank} and K_{knee} were good (0.82 to 0.91), while $CV_{\text{ME}}\%$ ($> 10\%$) and $M_{\text{diff}}\%$ ($> 5\%$) were 'poor', except during the right-legged condition. Given that all participants had a right-legged preference (Coren, 1993), it is suggested that during the CHSS activity, the right leg is task dependent and, therefore, predominantly performs either a propulsive or stabilization role during landing. This type of task dependence is seen in gait research whereby each limb performs a particular task, either absorbing power for the purpose of controlling the movement or providing power for propulsion (Sadeghi, Allard, & Duhaime, 1997). Furthermore, a potential explanation as to why the CHSS task displayed 'moderate' reliability may be

that this task was not indicative of movements similar to the spring-mass model, consequently requiring a greater amount of joint control and input from muscle contraction rather than from elastic energy.

For kinematic and kinetic data during all hopping and jumping tasks, the high ICC and low Mdiff% results suggest that VGRF data were reliable. This is consistent with previous studies which have shown ground reaction force data to have greater repeatability and less variability than kinetic and kinematic data (Ferber, et al., 2002; Queen, et al., 2006). Moreover, this lack of consistency in kinetics and kinematics between sessions is evident in the joint angle and moment values of this study and may explain the lack of reliability in K_{ank} and K_{knee} in active men. Intra-class correlation values for joint moment and angle data range from 0.14 to 0.83 and mean differences range from 0.19 to 20.89%. However, there were no clear patterns to emerge in the data to provide insight as to why there was a lack of consistency between days. Furthermore, it is difficult to compare these reliability data with previous research due to the differences in methodological approaches and the lack of published reliability data in hopping and jumping tasks (McLachlan, et al., 2006).

A limitation of this study is that the results reported include error that may be present within the measurement system (e.g. soft tissue artefact) or inherent in physiological performance (e.g. natural day-to-day fluctuations). While attempts to minimise these errors were made, based on our data it is difficult to differentiate the contributions each source of error made to the lack of consistency between testing sessions for many of the outcome measures. Furthermore, there may also be error inherent within the testing protocol itself

and it may be reasonable to suggest that the lower limb marker set adopted in this study also contributed as a source of error.

4.4.1 Conclusion

In summary, it is concluded that, in active men, K_{vert} is a reliable measure of MSS during over-ground running at 3.83 m/s and during hopping and jumping at 2.2 Hz. The reliability of all other MSS variables may be improved with the inclusion of a prior familiarization session or additional intersession trials, but further research is required to verify this. Future work might also explore other hopping and jumping frequencies to ascertain a more reliable frequency. In addition, as the $CV_{\text{ME}}\%$ suggests, there is evidence of measurement or movement variability within this protocol when measuring K_{ank} and K_{knee} and this may be an important finding in itself. Researchers may find these results relevant in study sample size estimation and when comparing the reliability of other testing procedures and equipment. Finally, a more detailed lower extremity marker set or alternate methodological protocol, such as intra-day reliability testing or increasing sampling frequency, may also be worthwhile investigating in the future.

Chapter 5. Eccentric exercise-induced alterations of lower limb stiffness during dynamic tasks.

5.1 Introduction

During shock absorption or braking, muscles contract eccentrically. This allows for controlled lengthening of the muscle which, depending on the movement requirements, will result in dissipation of heat as well as storage of elastic energy (LaStayo, et al., 2003). This type of contraction is common during sporting activities, with the release of elastic energy during a subsequent concentric contraction improving performance via force and power production (Komi, 2000). However, eccentric contractions have also been shown to cause muscle damage due to the high levels of strain placed on the muscle architecture, and the production of negative work by the associated muscles (LaStayo, et al., 2003). This induced muscle trauma is thought to be caused by a combination of mechanical and metabolic mechanisms, with no single mechanism alone responsible for the muscle damage (Allen, 2001; Cheung, et al., 2003; Gulick & Kimura, 1996). The presence of muscle damage is signalled by losses in muscle function, increased presence of muscle proteins and muscle-related enzymes in the blood stream, structural changes in muscle architecture, losses in range of motion, swelling, and/or delayed onset of muscle soreness (DOMS) (Cheung, et al., 2003). However, the time course of these symptoms are not alike and, alone, do not represent the magnitude of muscle damage; instead they are indicators that damage is present (Byrne, et al., 2004). Importantly, sports performance following unaccustomed eccentric exercise can be affected through the alteration of muscle function and joint mechanics (Byrne, et al., 2004; Cheung, et al., 2003). It has been suggested that measures of muscle

function are the best indicators of eccentric exercise-induced muscle damage (EIMD) (Warren, et al., 1999), with reductions in force and power production (Byrne & Eston, 2002b; Ebbeling & Clarkson, 1989), alterations in stretch reflex sensitivity (Avela, Kyrolainen, Komi, & Rama, 1999b), reductions in joint range of motion (Cleak & Eston, 1992), and altered muscle recruitment patterns (Deschenes, et al., 2000) being reported previously.

High force contractions cause neuromuscular fatigue and present symptoms similar to EIMD (Nicol, Avela, & Komi, 2006). The ability of the musculoskeletal system to adapt to EIMD, and how sports performance is enhanced through high-force eccentric contractions, is well researched (Komi, 2000; Nosaka, 2008). For example, previous findings have indicated that, following fatigue, there is a significant reduction in leg stiffness (Kuitunen, Kyrolainen, Avela, & Komi, 2007). These changes in leg stiffness during hopping are a result of changes in ankle kinetics and kinematics (Arampatzis, Bruggemann, & Klapsing, 2001; Farley, et al., 1998), and can be evidenced by changes in muscle activity (Hobara, et al., 2007). Moreover, a study assessing EIMD-associated changes in kinematics during running have suggested that there is no change in leg or joint stiffness 48 hours post EIMD (Dutto & Braun, 2004).

There is a paucity of literature considering the effect of eccentric exercise on musculoskeletal stiffness (MSS) over a short- or long-term time frame. One previous study observed changes after 48 hours in knee kinematics following a downhill running model that elicited muscle damage primarily to the knee flexor and extensor muscles (Dutto & Braun, 2004). Ankle joint function is essential in propulsion and support during movement (Meinders, Gitter, & Czerniecki, 1998; Neptune, Kautz, & Zajac, 2001; Rodgers, 1988); however, no study has explored the effect of EIMD on

the musculature of the ankle joint. Walking, running, hopping and jumping are fundamental sporting activities and are commonly investigated, thereby, understanding the effect of EIMD on the function of the ankle is important, particularly given the role of the ankle joint in these tasks.

5.1.1 Aims and hypothesis

The aim of this study was to investigate, over seven days (immediately before, immediately post, 24, 48 and 168 hours post induced damage), the effects of an eccentric exercise protocol targeting the plantar flexor muscles of the ankle (following a downhill backwards walking protocol) on: (1) leg stiffness during hopping; (2) leg stiffness during running; and (3) vertical stiffness during running in active males. It was hypothesised that following eccentric exercise aimed at producing eccentric muscle damage (1) there will be a reduction in leg stiffness during hopping, compared to baseline, and that these changes are associated with changes in ankle kinematics and muscle activity, and (2) there will be no change in leg and vertical stiffness, knee and ankle joint kinematics, and muscle activity during the running task.

5.2 Methods and procedure

To ensure the aim of this study was met, the suitability of participants for this study was assessed as outlined in Chapter 3.2. In summary, each participant filled out a health screening questionnaire that included questions regarding any current or pre-existing medical condition or injury, supplement usage and eccentric exercise background. If a potential candidate had a current or recent medical condition or

injury, a high level of caffeine ingestion or previous exposure to eccentric exercise, then, they were excluded from this study.

5.2.1 Participants

Twenty active males from various sporting backgrounds were recruited to participate in this study (age: 22.3 ± 3.0 years; mass: 74.7 ± 5.6 kg; stature: 1.79 ± 0.07 m). Of the 20 participants, 17 were right-legged and 3 were left-legged. As described in Chapter 3.5.1, this sample size was selected to detect differences at a significance level of 0.05 and a power of 90% and was based on results of earlier work of Dutto and Braun (2004). The University Ethics Committee approved the study and all participants provided written informed consent (approval reference V2009 78). All participants were injury-free at the time of testing and had not missed a training session or game in their respective sport for six weeks preceding the time of testing. During the initial testing session, anthropometric measurements were taken and participants were assessed for height, weight and laterality (Coren, 1993).

5.2.2 Procedure

To examine the hypotheses, a test-retest model over five sessions was employed which included: (1) baseline data taken immediately before eccentric exercise, (2) immediately post, and (3) 24 hours (1 day), (4) 48 hours (2 days), and (5) 168 hours (7 days) post eccentric exercise. Each participant returned for the four repeat-testing sessions at the same time of day as their exercise session, and during these sessions MSS and muscle damage criteria were assessed.

For all sessions, a warm-up was performed consisting of 5 minutes of cycling at a self-selected velocity on an ergometer and 5 minutes of self-selected dynamic

and static stretching. The same warm-up protocol was repeated at each subsequent testing session. Following the completion of the warm up, participants were required to perform the baseline testing protocol consisting of MSS, muscle damage and EMG measurements. Upon completion of baseline testing participants, performed the eccentric exercise protocol. For a detailed description of the exercise protocols and the assessment of MSS, muscle damage and EMG, see Chapter 3.3 and 3.4, respectively. However, the protocols are briefly described below.

The measures of MSS were recorded during a running task and a series of hopping tasks. The hopping tasks were single (dominant and non-dominant) leg, and double-legged hopping using a bent-legged action. These tasks were selected due to their 'good' reliability determined in Chapter 4. The participants were required to hop at 2.2 Hz with their hands on their hips and were requested to 'hop as high as possible' whilst maintaining hopping frequency. Hopping frequency was controlled by a metronome and trials were accepted if hopping frequency was within $\pm 2\%$ (Farley, et al., 1998). One trial of 20 single leg (dominant and non-dominant) and double-legged hops were performed and the order of each task was randomised between participants to reduce an order effect.

Kinetics and kinematics were analysed during ten trials of overground running (RUN). Participants were required to run at 3.83 m/s (Williams, et al., 2004) along a 10 m runway. Running speed was verified by timing gates placed 7 m apart and a force platform was positioned midway between the two timing gates. Participants contacted the force plate with their foot of each leg for five trials, and were instructed to run and look ahead and avoid targeting the force plate (Abendroth-Smith, 1996). Only trials that exhibited clean foot contact with the force plate were included for

analysis. The order in which leg the participant started on was randomised and any trials that exhibited a forefoot ground contact pattern were excluded from analysis.

Leg and vertical stiffness were examined in this study. Leg stiffness was calculated by

$$K_{leg} (N/m) = \frac{F_{peak}}{\Delta L} \quad (\text{Eq. 3.1})$$

where F_{Peak} is the peak VGRF during landing, and ΔL is the vertical change in leg length during the braking phase of ground contact. Vertical stiffness was calculated by

$$K_{vert} (N/m) = \frac{F_{peak}}{\Delta y} \quad (\text{Eq. 3.2})$$

where Δy is vertical displacement of the centre of mass (COM).

The eccentric exercise protocol consisted of 60 minutes of downhill backwards walking on a treadmill. The treadmill speed was 0.67 m/s (30-35 strides per minute) and inclined to a gradient of -8.5° . This protocol has been shown to cause muscle damage as a result of the 1800 to 2100 eccentric contractions over the 60-minute time frame (Nottle & Nosaka, 2005b).

A battery of indicators were used to measured muscle damage. Perceived muscle soreness was assessed via a visual analogue scale (VAS). Functional performance of the plantar flexor muscles was assessed via a maximal voluntary contraction (MVC) in a customised squat rack (Figure 3.1). Three isometric plantar flexor contractions were held for 3 sec, with a 2 min rest period between trials, and were completed using the participant's dominant leg. Peak force was recorded at the steadiest point on the force profile and averaged across the three trials, as

described in Pau et al. (2010). Venous blood samples were drawn using a 5 ml evacuated, heparinised vacutainer tube by a qualified phlebotomist. Blood samples were centrifuged to obtain plasma, and the plasma layer was removed and frozen at -80°C until analysed for creatine kinase (CK) activity level.

5.2.3 Data Collection and Analysis

A six-camera motion analysis system (VICON[®]) with retroreflective markers placed unilaterally according to the requirements of the lower limb plug-in-gait model of VICON[®]. The participants were instructed to hop on a force platform and kinetic and kinematic data were processed using custom-written LabVIEW. Kinematic data were sampled at 120 Hz and ground reaction forces were sampled at 1200 Hz with a 15 N threshold. To reduce potential sources of extrinsic error, the principal researcher (CJ) performed all anatomical measurements and undertook all marker placements on participants during all trials.

Electromyographic activity (EMG) was collected at 1200 Hz and measured from the medial gastrocnemius (MG), lateral gastrocnemius (LG), tibialis anterior (TA), soleus (SOL) and peroneus longus (PL) of the dominant leg of each participant. The reference electrode was placed on the medial epicondyle of the femur of each leg and all EMG data were synchronised with the VICON[®] capture system. Electrode placement was in line with the standards outlined by SENIAM (SENIAM, 2005).

Post processing of EMG data involved filtering using a 16 - 500Hz bandpass filter to remove signal artefact followed by root mean squared (RMS) full-wave rectification (Ritchie, et al., 2011). These data were then normalised to the values obtained during an isometric ankle plantar flexion MVC performed in the modified

squat rack. Mean EMG results were then calculated based on the work of Hobara (2010), whereby this measure was calculated from 100 ms prior to ground contact until ground contact (pre-activity; PRE), from ground contact until 30 ms after ground contact (background activity; BGA), between 30 and 60 ms after ground contact (short-latency reflex activity; M1), and from 60 to 90 ms after ground contact (long-latency reflex activity; M2).

5.2.4 Statistics

Upon assessment of normality, the changes in MSS and muscle damage markers (MVC, plasma creatine kinase [CK] activity and soreness) over time (immediately before, immediately post, and 24, 48 and 168 hr post) were assessed. The majority of data breached the normality criteria and, therefore, non-parametric statistics were administered. The changes in each dependent measure were analysed using a Friedman's test (measure \times time). Due to breaches of normality, all EMG data were assessed via non-parametric statistics and reported as median (range). All other data is reported as mean (SD).

5.3 Results

5.3.1 Muscle damage markers

Changes from baseline in the muscle damage indicators are shown in Figures 5.2A and 5.2B, respectively. Soreness scores assessed via a visual analogue scale (VAS) were increased immediately post ($p < 0.001$) exercise, and remained elevated at 24 hours ($p < 0.001$) and 48 hours ($p < 0.001$) post eccentric exercise. Isometric maximal voluntary contraction (MVC) plantar flexor force decreased ($p = 0.019$) immediately post eccentric exercise, and returned to baseline at all other time

intervals. There were no changes in plasma creatine kinase (CK) activity from baseline (159 IU/L); however, CK activity was elevated at 24 hours (221 ± 232.75 IU/L, $\Delta+39\%$), 48 hours (219 ± 236.85 IU/L, $\Delta+38\%$) and 168 hours (291 ± 232.75 IU/L, $\Delta+83\%$) post eccentric exercise (Figure 5.2C).

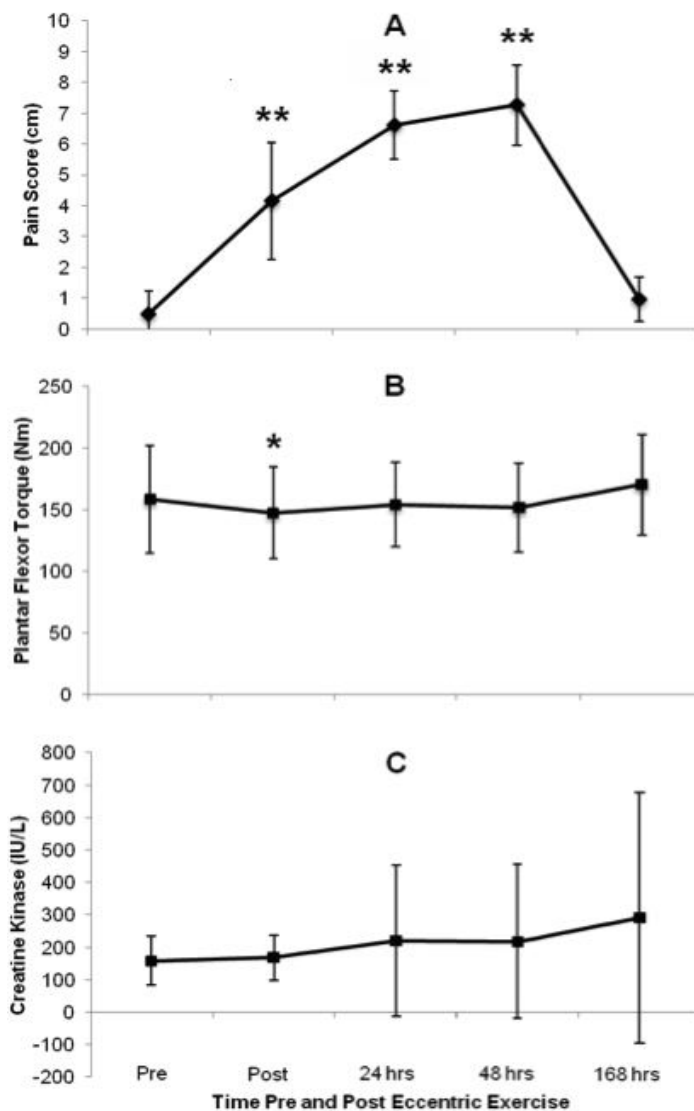


Figure 5.1. Changes (mean \pm SD) in A: Muscle soreness, B: Maximal voluntary isometric contraction in the plantar flexor muscles of the dominant limb, and C: Plasma CK activity at each time point between baseline (Pre) and 7 days (168 hours) post eccentric exercise protocol. * $p<0.05$; ** $p<0.01$.

5.3.1.1 MSS

Across the 168 hours post eccentric exercise, there were no statistically significant differences in K_{leg} or K_{vert} during the running or the hopping tasks. Figures 5.3A and 5.3B depict the changes in K_{vert} and K_{leg} in the dominant and non-dominant

legs across each of the time intervals for the running task. Figure 5.3C shows the response in K_{leg} over the time intervals during the double-legged and single-legged hopping tasks.

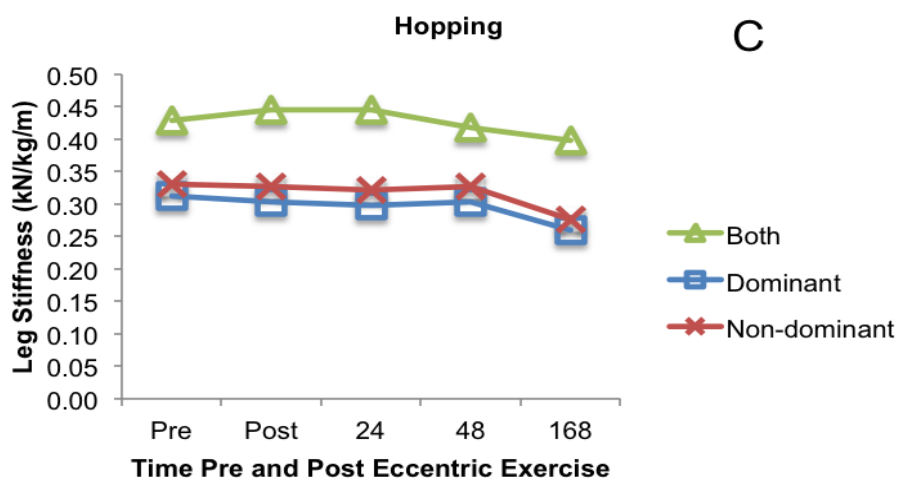
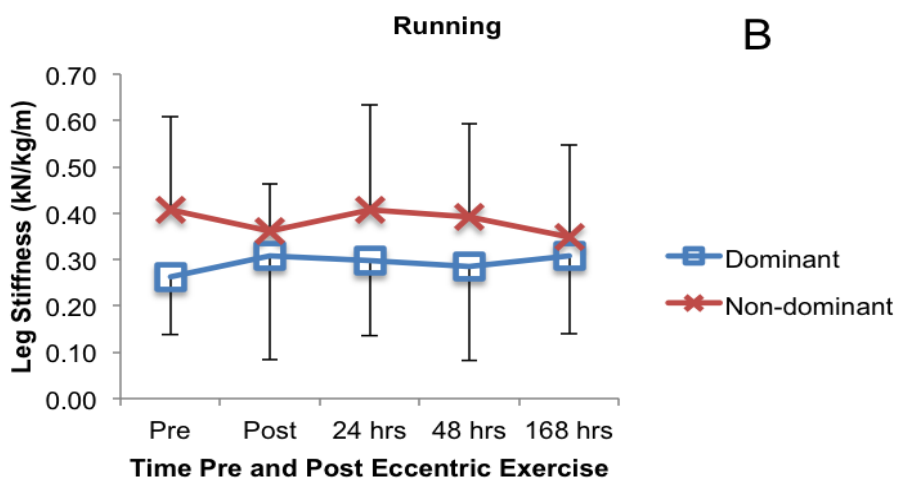
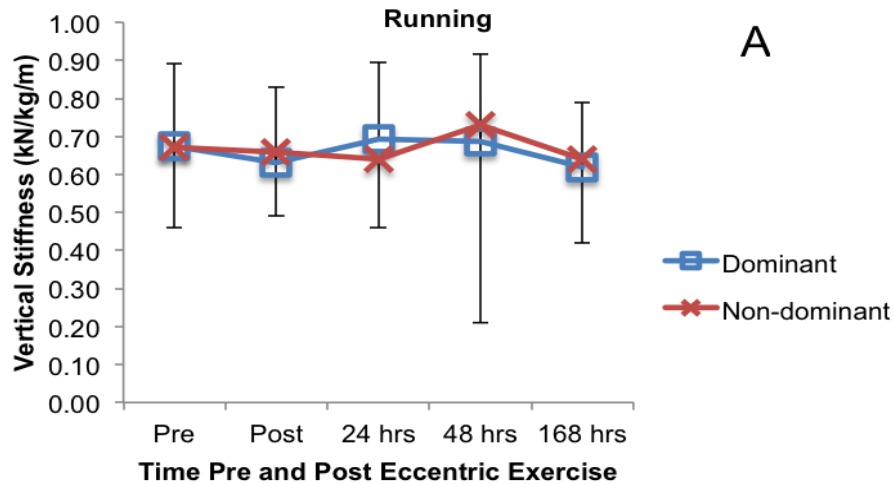


Figure 5.2. Mean changes in A: K_{vert} in the dominant and non-dominant legs during the running task, B: K_{leg} in the dominant and non-dominant legs during the running task, and C: K_{leg} in the double-legged (Both) and single-legged (Dominant and Non-dominant) hopping task between baseline (Pre) and 7 days (168 hours) post eccentric exercise protocol.

5.3.1.2 EMG

As described in Chapter 3, EMG results were determined during four time segments: (1) 100 ms prior to ground contact until ground contact (pre-activity; PRE), (2) from ground contact until 30 ms after ground contact (background activity; BGA), (3) between 30 and 60 ms after ground contact (short-latency reflex activity; M1), and (4) from 60 to 90 ms after ground contact (long-latency reflex activity; M2).

There were no differences in the EMG activity of any muscles of the dominant limb during any component of EMG analysis (PRE, BGA, M1 and M2). Muscle activity in all muscles recorded, across all testing sessions, and during all phases, is shown in Tables 5.1 and 5.2.

Table 5.1. Double-legged hopping EMG activity in the dominant limb. EMG activity (%MVC) in the medial gastrocnemius, lateral gastrocnemius, soleus, tibialis anterior and peroneus longus muscles prior to ground contact (PRE), background activity (BGA), short-latency stretch reflex (M1) and long-latency stretch reflex (M2) across the five testing sessions (Pre, Post, 24 hr, 48 hr and 168 hr).

Double-legged hop	Pre	Post	24 hr	48 hr	168 hr
PRE					
Medial gastrocnemius	103.52 (258.42)	151.81 (276.89)	138.14 (182.91)	87.64 (182.91)	122.24 (231.47)
Lateral gastrocnemius	60.63 (415.73)	97.62 (243.78)	89.62 (93.15)	161.71 (487.21)	70.64 (407.55)
Soleus	54.49 (150.61)	94.50 (412.05)	67.89 (737.53)	129.37 (183.36)	76.91 (210.01)
Tibialis anterior	97.53 (562.82)	77.52 (252.71)	88.62 (88.09)	95.24 (349.05)	78.41 (272.18)
Peroneus longus	86.51 (300.26)	105.26 (325.51)	98.73 (153.87)	111.58 (555.67)	101.15 (350.81)
BGA					
Medial gastrocnemius	117.13 (256.39)	145.09 (225.02)	172.11 (127.27)	141.09 (262.48)	155.71 (512.52)
Lateral gastrocnemius	103.47 (438.91)	114.28 (443.63)	111.32 (535.64)	179.63 (655.67)	87.56 (457.81)
Soleus	97.66 (193.16)	113.30 (479.14)	88.95 (208.30)	142.48 (189.69)	82.01 (302.62)
Tibialis anterior	100.33 (478.26)	85.10 (755.16)	96.35 (91.33)	99.23 (202.00)	97.14 (450.72)
Peroneus longus	133.90 (507.22)	122.04 (614.95)	117.13 (124.46)	131.01 (230.40)	82.13 (393.55)
M1					
Medial gastrocnemius	56.25 (170.38)	77.71 (160.57)	82.58 (73.99)	71.09 (99.00)	77.15 (123.75)
Lateral gastrocnemius	50.87 (419.62)	68.83 (772.33)	78.85 (200.60)	138.62 (548.11)	75.52 (184.08)
Soleus	86.18 (115.27)	75.63 (265.12)	61.19 (223.14)	101.45 (172.36)	116.30 (203.52)
Tibialis anterior	116.30 (203.52)	129.65 ((356.71)	79.58 (108.01)	105.01 (113.37)	86.44 (587.02)
Peroneus longus	86.56 (379.24)	92.73 (258.91)	106.91 (63.87)	106.91 (241.75)	58.66 (174.02)
M2					
Medial gastrocnemius	5.08 (57.05)	23.32 (65.62)	6.56 (138.30)	13.26 (137.39)	9.00 (114.71)
Lateral gastrocnemius	7.69 (37.83)	11.07 (61.35)	9.16 (125.64)	20.95 (147.54)	9.89 (99.56)
Soleus	16.36 (19.25)	19.28 (145.49)	11.10 (142.09)	19.68 (164.90)	9.03 (183.39)
Tibialis anterior	19.95 (107.75)	31.12 (108.79)	51.48 (82.40)	73.91 (89.47)	37.25 (73.02)
Peroneus longus	14.38 (32.75)	12,35 (62.35)	15.54 (99.09)	18.08 (209.38)	31.54 (100.27)

Values are median (range).

Table 5.2. Single-legged hopping EMG activity in the dominant limb. EMG activity (%MVC) in the medial gastrocnemius, lateral gastrocnemius, soleus, tibialis anterior and peroneus longus muscles prior to ground contact (PRE), background activity (BGA), short-latency stretch reflex (M1) and long-latency stretch reflex (M2) across the five testing sessions (Pre, Post, 24 hr, 48 hr and 168 hr).

Single-legged hop	Pre	Post	24 hr	48 hr	168 hr
PRE					
Medial gastrocnemius	104.68 (80.96)	104.33 (71.50)	139.73 (73.37)	126.43 (154.58)	155.36 (144.09)
Lateral gastrocnemius	59.85 (144.60)	85.80 (87.85)	99.61 (48.73)	163.37 (48.73)	93.89 (168.20)
Soleus	150.82 (112.64)	91.92 (499.67)	82.33 (138.50)	146.88 (263.98)	100.39 (170.03)
Tibialis anterior	115.00 (159.74)	129.57 (167.00)	68.24 (65.39)	79.29 (95.97)	77.86 (53.53)
Peroneus longus	99.85 (174.55)	93.77 (90.08)	115.70 (144.01)	110.99 (134.08)	92.69 (163.46)
BGA					
Medial gastrocnemius	122.37 (43.39)	117.33 (61.09)	148.75 (92.38)	135.36 (157.33)	149.04 (90.57)
Lateral gastrocnemius	93.22 (125.88)	91.17 (77.00)	110.06 (16.21)	180.00 (93.00)	130.84 (202.47)
Soleus	179.03 (104.67)	115.53 (309.88)	98.47 (216.45)	204.04 (292.51)	197.82 (179.28)
Tibialis anterior	113.23 (137.66)	107.97 (118.66)	70.38 (79.79)	82.78 (120.88)	89.90 (56.35)
Peroneus longus	118.22 (216.67)	131.28 (36.80)	137.75 (77.09)	150.88 (179.20)	109.89 (174.26)
M1					
Medial gastrocnemius	77.04 (38.07)	88.84 (46.99)	106.17 (59.60)	81.62 (140.01)	112.66 (102.39)
Lateral gastrocnemius	56.48 (143.87)	72.99 (30.74)	108.25 (88.61)	141.56 (73.62)	92.81 (188.25)
Soleus	124.39 (75.84)	91.97 (208.15)	94.10 (250.62)	143.37 (220.89)	173.83 (236.03)
Tibialis anterior	122.04 (184.41)	133.40 (187.02)	122.00 (81.41)	110.32 (130.97)	96.60 (148.21)
Peroneus longus	89.16 (166.20)	79.36 (66.47)	115.57 (54.14)	98.84 (114.69)	78.44 (128.38)
M2					
Medial gastrocnemius	17.66 (57.05)	14.91 (94.42)	14.80 (143.97)	13.42 (194.87)	8.08 (259.13)
Lateral gastrocnemius	13.02 (81.24)	14.59 (132.24)	17.85 (71.27)	15.92 (59.25)	15.31 (14.31)
Soleus	27.74 (125.67)	23.36 (168.62)	20.29 (191.67)	25.55 (178.74)	24.24 (210.77)
Tibialis anterior	41.60 (72.38)	34.46 (49.68)	62.92 (78.99)	25.56 (31.20)	43.33 (29.06)
Peroneus longus	23.73 (251.11)	29.58 (132.39)	30.32 (195.44)	47.16 (261.47)	29.07 (168.87)

Values are median (range).

5.3.1.3 Joint kinetics and kinematics

Results for joint kinetics and kinematics for the hopping and running tasks are shown in Tables 5.3 and 5.4. There were no differences between baseline and any follow up sessions, except for an increase in touchdown angle of the knee during single-legged hopping in the dominant limb ($p < 0.05$) at the 168hr testing timepoint.

Table 5.3. Kinematic and kinetic results in the dominant (DOM) and non-dominant (ND) legs during the hopping tasks (double and single legged) at 2.2 Hz across the five testing sessions (Pre, Post, 24 hr, 48 hr and 168 hr).

Hopping	Type	Pre	Post	24 hr	48 hr	168 hr
Double-legged						
F _{Peak} (N)	BOTH	3049.21 (412.33)	3104.68 (401.89)	3045 (411.70)	3032.04 (386.61)	2919.01 (412.65)
F _{Peak} (BW)	BOTH	38.27 (5.91)	39.73 (5.20)	38.37 (10.47)	38.92 (10.46)	34.12 (15.74)
COM (m)	BOTH	0.09 (0.02)	0.09 (0.02)	0.08 (0.02)	0.09 (0.01)	0.08 (0.01)
Peak Ankle Moment (Nm/kg)	BOTH	6.10 (2.77)	5.84 (2.48)	6.12 (2.90)	5.95 (2.63)	5.73 (2.21)
Peak Knee Moment (Nm/kg)	BOTH	6.49 (1.68)	6.33 (1.39)	6.17 (2.36)	6.48 (1.64)	6.35 (2.11)
Ankle excursion θ (deg)	BOTH	12.87 (7.07)	13.73 (6.33)	11.13 (7.29)	11.59 (5.31)	11.41 (5.81)
Knee excursion θ (deg)	BOTH	35.30 (9.49)	35.14 (8.47)	34.44 (8.53)	34.10 (6.07)	33.41 (6.30)
Ankle touch-down θ (deg)	BOTH	15.72 (10.13)	11.11 (8.70)	12.11 (9.23)	11.86 (9.44)	13.88 (10.10)
Knee touch-down θ (deg)	BOTH	21.89 (13.56)	20.37 (10.13)	19.82 (10.18)	17.86 (9.10)	16.45 (9.32)
Single-legged						
Peak Ankle Moment (Nm/kg)	DOM	5.11 (0.96)	4.60 (1.09)	5.11 (0.93)	5.13 (0.92)	5.23 (1.17)
	ND	5.15 (1.22)	4.72 (1.05)	5.08 (1.39)	5.06 (1.15)	5.45 (1.40)
Peak Knee Moment (Nm/kg)	DOM	3.77 (1.47)	3.58 (1.30)	3.80 (1.10)	3.94 (1.31)	3.90 (0.78)
	ND	3.56 (1.59)	3.68 (1.38)	3.56 (1.30)	4.01 (1.21)	3.56 (1.46)
Ankle excursion θ (deg)	DOM	13.32 (5.91)	15.43 (5.17)	14.45 (5.90)	13.57 (6.04)	14.19 (4.33)
	ND	14.79 (6.36)	17.47 (4.21)	16.50 (5.17)	15.90 (4.09)	16.88 (2.98)
Knee excursion θ (deg)	DOM	34.56 (7.22)	34.60 (7.87)	36.93 (8.16)	35.52 (7.51)	36.32 (4.45)
	ND	33.76 (8.79)	35.48 (9.04)	33.72 (10.35)	35.08 (6.18)	35.15 (6.56)
Ankle touch-down θ (deg)	DOM	12.35 (5.14)	13.76 (5.32)	12.61 (4.52)	12.57 (3.68)	12.97 (4.13)
	ND	11.67 (7.00)	13.64 (6.49)	10.11 (8.16)	12.78 (4.66)	12.36 (5.52)
Knee touch-down θ (deg)	DOM	28.58 (9.25)	30.84 (7.98)	32.34 (9.30)	31.23 (8.58)	30.43 (6.82)*
	ND	29.93 (9.24)	32.04 (9.81)	28.88 (9.65)	32.15 (5.65)	30.27 (7.32)

Values displayed are mean (SD). * Represents significant difference from baseline ($P < 0.05$).

Table 5.4. Kinematic and kinetic results in the dominant (DOM) and non-dominant (ND) legs during the running task across the 5 testing sessions (Pre, Post, 24 hr, 48 hr and 168 hr).

Running	Type	Pre	Post	24 hr	48 hr	168 hr
F _{Peak} (N)	DOM	2684.74 (958.50)	2825.29 (1239.77)	2637.69 (841.71)	2659.18 (874.98)	2572.83 (839.68)
	ND	2738.57 (953.30)	2642.24 (769.30)	2614.19 (897.13)	2791.47 (1237.37)	2671.95 (1041.40)
F _{Peak} (BW)	DOM	34.01 (7.64)	33.44 (7.67)	33.54 (6.21)	33.72 (6.70)	32.70 (6.07)
	ND	34.69 (7.30)	33.68 (5.51)	33.16 (7.10)	35.11 (11.09)	33.80 (8.54)
COM (m)	DOM	0.05 (0.01)	0.05 (0.01)	0.05 (0.01)	0.05 (0.01)	0.05 (0.01)
	ND	0.05 (0.01)	0.05 (0.01)	0.05 (0.01)	0.05 (0.01)	0.05 (0.01)
Touch-down θ (deg)	DOM	34.61 (6.20)	34.28 (8.13)	35.23 (6.91)	34.58 (7.78)	37.03 (6.40)
	ND	34.41 (6.60)	35.10 (6.62)	35.01 (6.86)	33.38 (8.08)	35.27 (7.66)
Peak Ankle Moment (Nm/kg)	DOM	3.76 (0.70)	3.43 (0.84)	3.90 (0.85)	3.77 (0.60)	3.84 (0.75)
	ND	3.71 (0.98)	3.70 (1.03)	3.84 (0.64)	3.70 (1.05)	3.92 (0.11)
Peak Knee Moment (Nm/kg)	DOM	4.25 (1.64)	4.07 (1.50)	3.70 (1.17)	4.41 (1.31)	4.33 (0.96)
	ND	4.15 (1.38)	4.18 (1.47)	4.33 (0.98)	4.15 (1.29)	4.47 (0.91)
Ankle excursion θ (deg)	DOM	15.87 (6.20)	15.93 (6.19)	17.01 (4.97)	16.82 (5.31)	16.31 (5.15)
	ND	16.33 (6.24)	18.06 (8.03)	18.50 (7.66)	17.74 (6.87)	17.76 (6.25)
Knee excursion θ (deg)	DOM	29.47 (5.37)	28.07 (8.17)	28.41 (4.67)	29.54 (5.31)	30.52 (5.13)
	ND	30.40 (5.96)	29.70 (7.65)	29.58 (5.45)	30.52 (5.46)	30.81 (4.55)
Peak ankle θ (deg)	DOM	24.22 (2.84)	23.35 (5.68)	25.21 (2.96)	24.30 (3.48)	24.48 (2.60)
	ND	25.12 (3.97)	25.70 (5.05)	24.85 (5.56)	24.50 (3.29)	25.91 (3.39)
Peak knee θ (deg)	DOM	45.34 (7.07)	42.56 (11.22)	45.53 (7.52)	45.22 (9.08)	46.32 (8.45)
	ND	43.79 (11.20)	44.74 (11.82)	43.18 (9.58)	44.34 (7.61)	45.95 (7.47)

Values displayed are means (SD).

5.4 Discussion

The purpose of this study was to investigate the changes in K_{vert} and K_{leg} during running and hopping following eccentric exercise-induced muscle damage. EIMD was induced to the plantar-flexor muscles after downhill backward walking as demonstrated by changes in the muscle damage indicators such as VAS scores, MVC force and plasma CK activity (non-significant). Our results for MSS were similar to previous research investigating MSS during running (Arampatzis, et al., 1999; Divert, et al., 2005; Morin, et al., 2005) and hopping tasks (Farley & Morgenroth, 1999; Hobara, et al., 2010; McLachlan, et al., 2006). Despite significant differences in self-reported muscle soreness in the days after the eccentric training protocol, there were no significant differences in K_{leg} or K_{vert} between baseline and any of the post-exercise testing sessions for the hopping tasks. This finding did not support our first hypothesis. The absence of any significant changes in K_{leg} or K_{vert} could be attributed to an alteration of the stretch reflex that is acting as a protective mechanism for the musculoskeletal system (Byrne, et al., 2004). Additionally, supporting the second hypothesis, there were no differences in K_{leg} during the running task. This is in agreement with past research (Dutto & Braun, 2004) however, EIMD during this study was elicited in knee flexor and extensor muscles. Visual inspection of our MSS results suggests that K_{leg} and K_{vert} did not change following eccentric muscle damage (Figure 5.3).

It may be reasonable to assume that the absence of any change in K_{leg} and K_{vert} could be due to the musculoskeletal system's attempt to maintain functional performance in the presence of muscle damage (Padua, et al., 2006). Previously, fatigue has been used to investigate changes in MSS and neuromuscular control. Levels of K_{leg} and K_{vert} have been shown to decrease following a run to fatigue via

changes in kinematics and temporal results (Dutto & Smith, 2002). Our results suggest that shifts in kinematics did not occur as no changes in center of mass displacement, vertical ground reaction forces or leg length displacement were evident. This also strengthens the theory that the musculoskeletal system is attempting to maintain a set level of MSS to optimize functional efficiency (Padua et al., 2006).

Downhill, backward walking causes muscle damage in the muscles about the ankle joint (Nottle & Nosaka, 2005a). To determine a potential mechanism in the modulation of MSS following eccentric muscle damage, we recorded EMG activity of five muscles of the ankle joint (lateral gastrocnemius, medial gastrocnemius, soleus, tibialis anterior and peroneus longus), specifically because the ankle joint is known to be susceptible to muscle damage post eccentric exercise (Nottle & Nosaka, 2005a; Whitehead, et al., 1998). The absence of any change in K_{vert} and K_{leg} was confirmed by the lack of differences in EMG activity in the muscles of the lower leg. Previous research has shown that K_{leg} is partially dependent on the level of pre-activation (Dyhre-Poulsen, Simonsen, & Voigt, 1991); however, in the present study eccentric exercise-induced damage caused by downhill walking did not alter pre-activation, preparation for stretch reflex activity or the need to store and re-use elastic energy via short and long latency stretch reflex. Specifically, no differences were found in muscle activity during the PRE phase, reflecting that there is no change in the commands from central motor system to enhance muscle spindle sensitivity and prepare for ground contact (Gottlieb, Agarwal, & Jaeger, 1981), following eccentric exercise-induced muscle damage. Additionally, there was no change in the preparation of the muscles for stretch reflex activation, or in the level of short and long latency stretch reflex as characterised by the BGA, M1 and M2 phases of EMG

activity during stretch-shortening cycle actions (Gollhofer, Strojnik, Rapp, & Schweizer, 1992; Voigt, Chelli, & Frigo, 1998).

The ankle joint has been described as the principal factor in the changes that occur in K_{leg} during submaximal hopping (Arampatzis, Bruggemann, et al., 2001; Farley, et al., 1998; Farley & Morgenroth, 1999). The observation of no differences in MSS in the present study can partly be confirmed by the absence of significant changes in the EMG activity of the ankle joint. However, observed differences in the touchdown angle of the knee during single-legged hopping were seen in the dominant leg between baseline and 168 hours. An increased range of motion of the knee has also been reported to allow an increase in shock absorption during running (Derrick, Hamill, & Caldwell, 1998) and a hopping type task (Lafortune, Hennig, & Lake, 1996). Furthermore, increased joint angle of the knee at touchdown leads to a greater ground reaction force moment arm and, thus, a decrease in K_{leg} (Farley, et al., 1998). Therefore, participants in the present study were altering the touchdown knee angle to either maintain MSS levels, or else this was an adaptive response to the eccentric exercise. For example, altered knee angle may have been adopted as a protective strategy to reduce pain or further damage. This finding is in contrast to our first hypothesis that any changes in MSS would be a result of changes at the ankle joint during hopping.

As suggested by previous authors (Nottle & Nosaka, 2005a), the submaximal nature of the eccentric exercise protocol of this study may not be enough to elicit the levels of muscle damage required to see large changes in the response of the musculoskeletal system, even though the protocol is one that reflects the eccentric actions of daily activities. Furthermore, due to soft tissue artefact, it may not have been possible to detect any subtle changes in kinematics or neuromuscular

activation that may have changed across testing sessions. Additionally, normalization of the EMG to the isometric ankle flexion MVC performed at each testing session assumes that the co-contraction strategy of the involved muscles is similar at each time point, which may not be the case in the presence of eccentric induced muscle damage. There is also potential that the population used in this study may be more accustomed to eccentric contractions and, therefore, only small changes were elicited by the eccentric protocol.

5.4.1 Conclusion

In conclusion, our results suggest that the muscle damage caused by downhill, backward walking does not alter K_{leg} whilst running over-ground at 3.83 m/s in either the dominant or the non-dominant leg of active males. Furthermore, during hopping at 2.2 Hz using single and double-legged tests, K_{leg} and K_{vert} are not affected by the same muscle damage. Deeper examination is required to explain the consequences of eccentric exercise-induced muscle damage and the mechanisms behind MSS regulation.

Chapter 6. Behaviour of the Achilles tendon *in-vivo* following exercise induced muscle damage.

6.1 Introduction

Muscles commonly act eccentrically, performing negative work through muscle stretching, and absorbing energy or dissipating it as heat (Lindstedt, LaStayo, & Reich, 2001). Eccentric contractions occur when the load torque acting on the muscle or muscles is greater than that of the activated motor units (Enoka, 1996). Reduced motor unit recruitment for the same amount of force is observed in eccentric contractions, when compared to the other contraction types, and exercise induced muscle damage often transpires (Enoka, 1996). Eccentric exercise-induced muscle damage (EIMD) is well documented and common symptoms include decreased force generating capacity (Warren, et al., 1999), decreased range of motion (Hamill, et al., 1991), increased inflammation (Friden, et al., 1988), increased sensation of pain (Newham, et al., 1988), and increased plasma creatine kinase activity (Clarkson, et al., 1992). The exact cause of muscle damage is unknown; however, there is evidence that supports both metabolic mechanisms (Gulick & Kimura, 1996) and structural and mechanical disruption or damage within muscle architecture (Armstrong, et al., 1991; Friden & Lieber, 1992; Jones, et al., 1989; Lieber & Friden, 2002). Mechanical loading from exercise consistent with that inducing muscle damage can cause several changes to muscle-tendon structure and mechanics (Arampatzis, et al., 2005; Chandrashekar, et al., 2011; Paschalis, et al., 2007). Within this complex interaction, tendons play a vital role, as “not only are tendons

essential for movement - movement is also essential for tendons” (Heinemeier & Kjaer, 2011, p. 115).

Tendon provides two roles; transmission of contractile force to bone resulting in movement about a joint, and the storage and reuse of strain energy (Ker, et al., 1988). As Hill’s muscle-model (Hill, 1974) suggests, in-series muscular contraction causes forces to be transferred to tendons to generate joint motion. Tendons exhibit viscoelastic properties and store and reuse elastic strain energy to provide economical movement (Alexander & Bennet-Clark, 1977; Bobbert, 2001; Maganaris, et al., 2008), and adapt to stimuli and respond to use and disuse (Kjær, et al., 2009; Kubo, et al., 2004a; Kubo, et al., 2010). Isometric resistance training at high-strain magnitudes has been investigated in healthy adults and was shown to increase tendon stiffness when compared to a low-strain condition (Arampatzis, et al., 2007). Additionally, eccentric resistance training has been shown to increase tendon stiffness (Duclay, et al., 2009). Furthermore, isotonic resistance training via leg press and leg extension exercises have also been shown to decrease tendon elongation and strain and, subsequently, increase tendon stiffness (Reeves, Maganaris, & Narici, 2003).

Accompanying the research investigating the changes in the morphological and mechanical properties of tendon following repeated mechanical loading is research exploring the relationship between fatigue and tendon behaviour. Under *in vitro* conditions, human tendon rupture induced by fatigue has been well researched (Schechtman & Bader, 1997, 2002; Wren, Lindsey, Beaupre, & Carter, 2003). However, the behaviour of tendon in living

tissue during *in vivo* conditions may provide more relevant information, particularly as there is limited research investigating tendon fatigue *in vivo*. Studies exploring the effect of static and cyclical loading on tendon have been used to investigate tendon fatigue and have uncovered mixed findings. Endurance exercise consisting of fatiguing eccentric and isometric contractions of varying duration and intensity have been shown to reduce vastus lateralis tendon stiffness (Kubo, Kanehisa, Kawakami, & Fukunaga, 2001). Furthermore, fatigue caused by repeated isometric contraction also significantly reduced vastus lateralis tendon stiffness (Kubo, et al., 2005). However, no differences were found in Achilles tendon stiffness following submaximal repeated isokinetic contractions to fatigue or after maintaining a submaximal isometric contraction until fatigue (Mademli, et al., 2006; Mademli, Arampatzis, & Walsh, 2008).

Sport-like activities have also been used to investigate tendon behaviour (Peltonen, Cronin, Avela, & Finni, 2010). Similar to the effect of resistance training on Achilles tendon stiffness, the cyclical loading caused during hopping led to no differences between pre- and post-exercise levels of Achilles tendon stiffness. However, to the author's knowledge, Peltonen and colleagues (2010) are the only authors to test the relationship between tendon behaviour and the effect of fatigue during an ecologically valid, sports-specific, dynamic exercise task, rather than eccentric and concentric contractions in isolation.

In summary, the mechanical properties of tendon can be altered via mechanical loading. However, human tendon responds differently based on location, contraction mode, contraction duration and the rate of strain applied to

the tendon, and all of these factors may contribute to the severity of tendon fatigue. Muscle contraction mode or amount of force production does not affect tendon stiffness, but increased contraction duration makes tendon more compliant, thereby reducing stiffness (Kubo, Kanehisa, Ito, & Fukunaga, 2001). Greater tendon elongation under a given force translates to an inverse level of tendon stiffness and, therefore, a greater level of elastic strain energy is able to be stored in the tendon (Maganaris, et al., 2008). However, high levels of tendon elongation can cause tendon rupture (Butler, et al., 1978). Advances in technology have made it possible to investigate the mechanical properties of fascicle and tendon *in vivo*. Through the use of real-time ultrasonography, it is possible to image the structure of the musculoskeletal system and, specifically, the tendon-aponeurosis complex. While the effect of training and fatigue on the behaviour of the tendon-aponeurosis complex has been widely researched, so far, little is known about the relationship between EIMD and the behaviour of the Achilles tendon, and especially when EIMD is caused during common sporting activities.

6.1.1 Aims and Hypotheses

Exercise induced muscle damage affects muscular force production and has a direct causal effect on in-series elastic structures such as tendon. Therefore, the purpose of this study was to investigate whether two mechanical properties of the Achilles tendon, (1) strain, and (2) stiffness, are altered as a result of the EIMD caused by a downhill backwards walking protocol. Based on

previous research investigating tendon response to tendinopathy and fatigue, it is hypothesised that Achilles tendon stiffness decreases following EIMD as a result of increases in Achilles tendon strain levels.

6.2 Methods and procedure

To ensure the aims of this study were met, the suitability of participants for this study was assessed as outlined in Chapter 3.2. In summary, each participant filled out a health screening questionnaire that included questions regarding any current or pre-existing medical condition or injury, supplement usage and eccentric exercise background. If a potential candidate had a current or recent medical condition or injury, a high level of caffeine ingestion or previous exposure to eccentric exercise, then, they were excluded from this study.

6.2.1 Participants

Twenty active males from various sporting backgrounds were recruited to participate in this study (age: 22.3 ± 3.0 years; mass: 74.7 ± 5.6 kg; height: 1.79 ± 0.70 m). Of the 20 participants, 17 were right-legged and 3 were left-legged. As described in Chapter 3.5.1, this sample size was selected to detect differences at a significance level of 0.05 and a power of 90% and was based on results of earlier work of Dutto and Braun (2004). The University Ethics Committee approved the study and all participants provided written informed consent (approval reference V2009 78). All participants were injury-free at the time of testing and had not missed a training session or game in their respective

sport for six weeks preceding the time of testing. During the initial testing session, anthropometric measurements were taken and participants were assessed for height, weight and laterality (Coren, 1993).

6.2.2 Procedure

To examine the hypotheses, a test-retest model over five sessions was employed which included data taken: (1) immediately before eccentric exercise, (baseline), (2) immediately post, and (3) 24 hours (1 day), (4) 48 hours (2 days), and (5) 168 hours (7 days) post eccentric exercise. Participants returned for the four repeat-testing sessions at the same time of day as their exercise session, and during these sessions maximal plantar flexor voluntary contraction (MVC) force, MVC electromyography (EMG) and Achilles tendon displacement were assessed. Muscle damage caused by the eccentric exercise protocol was determined via a battery of indicators.

For all sessions, a warm-up was performed consisting of 5 minutes of cycling at a self-selected pace on a stationary ergometer. The same warm-up protocol was repeated at each subsequent testing session. Following the completion of the warm up, participants were required to perform the baseline testing protocol consisting of Achilles tendon (AT) stiffness, AT strain, muscle damage and EMG measures. Upon completion of baseline testing, participants performed the eccentric exercise protocol. For a detailed description of the exercise and testing protocol of this study, see Chapters 3.3 and 3.4, respectively. Furthermore, detailed descriptions of the assessment of AT strain,

AT stiffness, muscle damage and EMG, and of the eccentric exercise protocols, can also be found in Chapter 3.3 and 3.4. However, they will be briefly described below.

6.2.2.1 Eccentric exercise protocol and muscle damage indicators

The eccentric exercise protocol consisted of 60 minutes of downhill backwards walking on a treadmill. The treadmill speed was 0.67 m/s (30-35 strides per minute) and inclined to a gradient of -8.5° . This protocol has been shown to cause muscle damage as a result of the 1800 to 2100 eccentric contractions over the 60-minute time frame (Nottle & Nosaka, 2005b).

A battery of indicators were used to measured muscle damage. Perceived muscle soreness was assessed via a visual analogue scale (VAS). Functional performance of the plantar flexor muscles was assessed via a maximal voluntary contraction (MVC) in a customised squat rack (Figure 3.1). Three isometric plantar flexor contractions were held for 3 sec, with a 2 min rest period between trials, and were completed using the participant's dominant leg. Peak force was recorded at the steadiest point on the force profile and averaged across the three trials, as described in Pau et al. (2010). Venous blood samples were drawn using a 5 ml evacuated, heparinised vacutainer tube by a qualified phlebotomist. The blood sample was then centrifuged to obtain plasma, and the plasma layer was removed and frozen at -80°C until analysed for creatine kinase (CK) activity level.

6.2.2.2 Ultrasound experimental set-up and isometric plantar flexion procedure

Achilles tendon behaviour was measured on the dominant leg of each participant. Participants performed an isometric MVC of the plantar-flexors in a custom built rack (Figure 3.1 A). The customised squat rack consisted of a barbell fixed by a chain around each end so that the height of the set-up could be adjusted to each participant and vertical movement could be controlled. Each participant was custom-fitted to the rack prior to testing so that a pure isometric plantar flexion contraction to be performed without any changes in ankle joint angle. The testing protocol consisted of the participant gradually ramping force from rest to MVC within approximately 5 seconds (Kubo, et al., 2005). This was repeated until three technically correct trials were performed. Each trial was separated by a 2 min rest period and all measured values were averaged across the three trials.

6.2.3 Data Collection and Analysis

A six-camera motion analysis system (VICON[®]) was used to measure any change in ankle angle that occurred during MVC. Retroreflective markers were placed unilaterally according to the requirements of the lower limb plug-in-gait model of VICON[®]. Kinematic data were sampled at 120 Hz and ground reaction forces were sampled at 1200 Hz with a 15 N threshold and kinetic and kinematic data were processed using custom-written LabVIEW.

Electromyographic activity (EMG) was collected at 1200 Hz and measured from the medial gastrocnemius (MG), lateral gastrocnemius (LG), tibialis anterior (TA), soleus (SOL) and peroneus longus (PL) of the dominant leg of each participant. Muscle activity of the vastus lateralis (VL) was also collected to allow for visual inspection to ensure no knee extension was performed during the MVC trials. All EMG data were synchronised with the VICON[®] capture system and electrode placement was in line with the standards outlined by SENIAM (SENIAM, 2005).

6.2.3.1 Achilles tendon moment arm length and Achilles tendon thickness

Achilles tendon moment arm length was estimated using a combination of ultrasonography and anthropometry. Achilles tendon thickness was measured from stored ultrasound images. Thickness was determined from the distance between the anterior margin and the posterior margin of the tendon (Figure 6.7) and directly horizontal to the predetermined medial malleoli of the ankle (Bryant, et al., 2008).

The ankle joint centre was located by marking the surface of the skin, along an imaginary line that passes through the transmalleolar axis, at the location of the lateral and medial malleoli. To determine AT moment arm length, the thickness of the skin together with the midpoint of tendon thickness, as determined from stored ultrasonography images, was subtracted from the distance to the medial malleoli of the ankle (Figure 3.10). This was again

performed using ImageJ software and provides at least the ability to consider the effect of mechanical leverage on AT strain.

During isometric MVC of the plantar flexors, a small heel lift can occur causing a change in ankle angle. The change in ankle angle then translates to an increase in AT moment arm and, therefore, affects the vertical displacement of the tendon (Maganaris, et al., 1998; Magnusson, et al., 2001). To account for this, ankle joint rotation was measured using the VICON[®] system and incorporated to calculate the change in Achilles tendon moment arm length shift. This was then deducted from total ATJ displacement to correct AT moment arm length for any vertical displacement that may have occurred.

6.2.3.2 Determination of muscle-tendon junction position

The position of the Achilles tendon-muscle junction (ATJ) of the medial gastrocnemius muscle was determined using real-time B-mode ultrasonography. Sagittal plane images of the myotendinous ATJ were recorded and stored on the ultrasonography computer system. The position of the ATJ was located and the ultrasound probe was then positioned so that the ATJ appeared clearly in the inferior third of the ultrasound image (Figure 3.4). The probe was then longitudinally attached to the dermal surface over the location of the ATJ insertion point with a custom-built brace and adhesive foam. Achilles tendon length was measured at rest whilst standing between the marked insertion point at the calcaneus and the marked location of the ATJ using a measuring tape.

6.2.3.3 Achilles tendon force

Achilles tendon force was calculated by dividing the ankle joint moment by the Achilles tendon moment arm length. This was calculated at MVC and assumed that all of the plantar flexor moment is contributed via the Achilles tendon structure (Lichtwark & Wilson, 2005).

6.2.3.4 Achilles tendon strain and stiffness

The ultrasound video of the ATJ taken from rest to MVC of the plantar flexor was converted into individual frames and the individual frames were then analysed using ImageJ freeware. A custom-written program in LabVIEW[®] was used to determine the point at which MVC force occurred and, therefore, the corresponding ultrasound video frame of the ATJ. The peak force plateau was determined as the segment in the force trace that had the lowest coefficient of variation over a one second timeframe. Three frames corresponding to this peak force plateau were identified and averaged to determine ATJ position; (1) the first data sample, (2) the median data sample, and (3) the final sample of the force plateau (Bryant, et al., 2008). The respective matching force values for these time points were also recorded.

To account for a proximal-distal and anterior-posterior shift in ATJ during MVC, trigonometry was used to solve and correct any effect vertical or horizontal displacement may have had on ATJ displacement and, therefore, strain results (Figure 3.9) (Maganaris, et al., 1998; Magnusson, et al., 2001). The pixel position of the ATJ at rest was recorded and subtracted from the three ATJ pixel

positions during MVC to determine the corrected location. Achilles tendon strain was then calculated by:

$$\text{Achilles tendon strain (\%)} = [(L_{MVC} / L_i) - 1] \times 100 \quad (\text{Eq. 3.1})$$

where L_{MVC} is the tendon length at MVC and L_i is the resting tendon length. Achilles tendon stiffness was calculated as the ratio of peak AT force and the corresponding AT displacement during the final 20% of MVC.

6.2.4 Statistics

Changes in AT strain, AT stiffness, EMG and muscle damage markers (MVC, CK activity and soreness) over time (immediately before, immediately post, and 24, 48 and 168 hr post) were assessed. All dependant measures were tested for normality via a critical appraisal approach (Peat & Barton, 2005). In cases where data breached the normality criteria, non-parametric statistics were administered and, where appropriate, descriptive statistics are displayed as median (range). The changes in each dependent measure (measure × time) were analysed parametrically using a repeated measures ANOVA and non-parametrically using a Friedman's test. Post-hoc analysis with a Bonferroni adjustment was used to determine where differences existed between each testing session and baseline.

6.3 Results

Achilles tendon force ($p = 0.10$) did not change from baseline levels at any post-exercise time periods (Figure 6.7 A), while Achilles tendon strain decreased at 24 hours ($p = 0.008$) and 48 hours ($p = 0.002$) post eccentric EIMD (Figure 6.7 B). Achilles tendon stiffness increased at 24 hours ($p = 0.004$) post EIMD (Figure 6.7 C).

There were no differences ($p > 0.05$) in peak MVC plantar flexor force between pre-exercise levels and any post-exercise levels (Table 6.1). There were no differences ($p > 0.05$) in EMG activity of the medial gastrocnemius (MG), lateral gastrocnemius (LG), tibialis anterior (TA), soleus (SOL) or peroneus longus (PL) of the dominant leg of participants across any of the testing sessions.

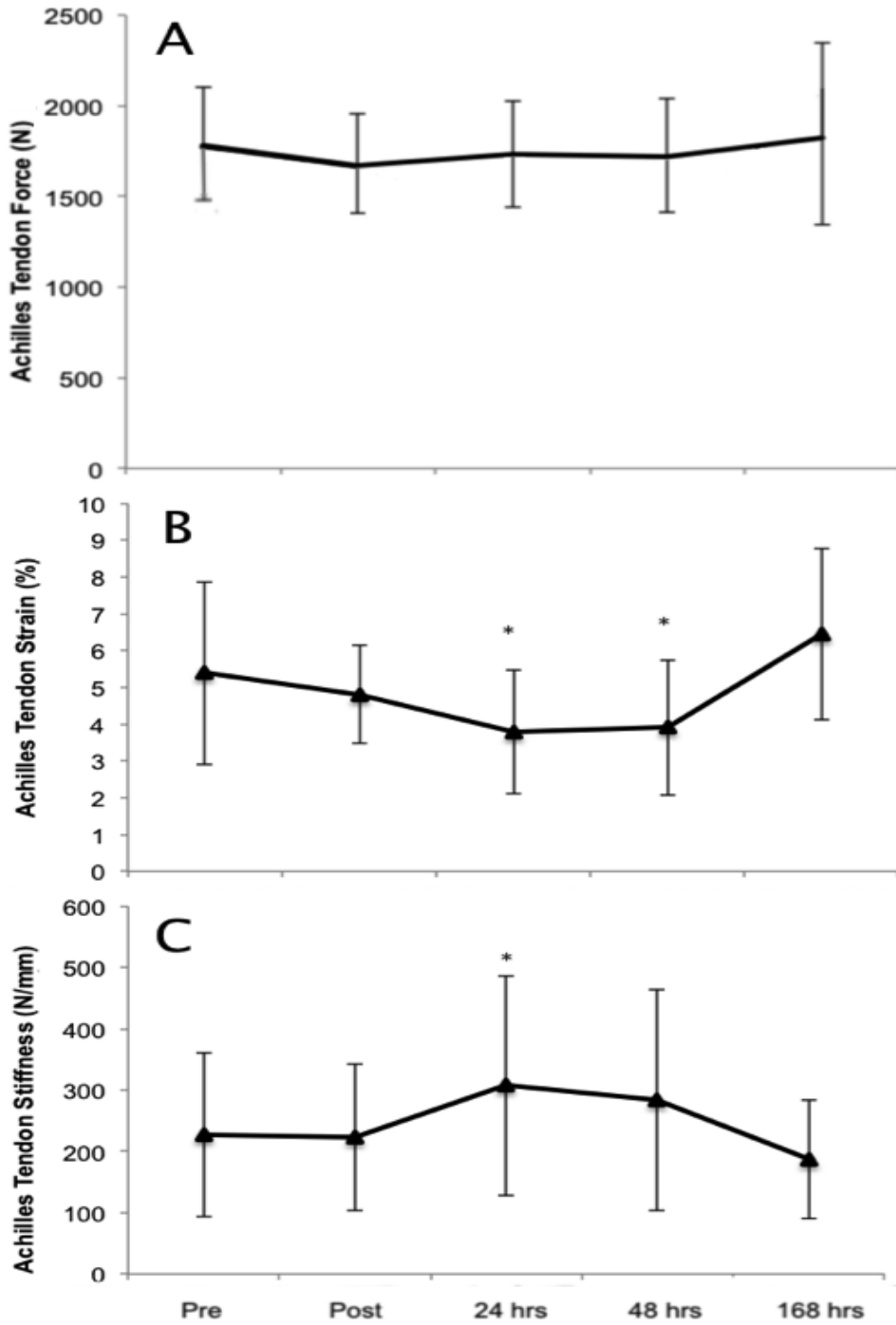


Figure 6.1. Changes (mean±SD) in A: Achilles tendon force, B: Achilles tendon strain, and C: Achilles tendon stiffness at each time point between baseline (Pre) and 7 days (168 hours) post eccentric exercise protocol. * $p < 0.05$.

Table 6.1. Plantar flexor force and muscle activity (EMG) measurements during MVC across the 5 testing sessions (Pre, Post, 24 hr, 48 hr and 168 hr) for the dominant limb.

	Pre	Post	24 hr	48 hr	168 hr	p value
Force (kN)	1.71 (0.41)	1.75 (0.29)	1.79 (0.31)	1.78 (0.42)	1.94 (0.44)	0.246
EMG (mV)						
MG	0.65 (0.40)	0.76 (0.72)	0.63 (0.48)	0.73 (0.69)	0.91 (0.76)	0.898
LG	0.73 (0.72)	0.99 (0.48)	0.58 (0.69)	0.47 (0.76)	0.70 (0.63)	0.282
SOL	0.86 (0.48)	0.88 (0.69)	0.58 (0.76)	0.49 (0.63)	0.61 (0.86)	0.637
TA	0.42 (0.69)	0.52 (0.76)	0.36 (0.63)	0.26 (0.86)	0.28 (0.60)	0.592
PL	0.60 (0.76)	0.87 (0.63)	0.54 (0.86)	0.49 (0.60)	0.62 (0.44)	0.576

Values are mean (\pm SD). MG, medial gastrocnemius; LG, lateral gastrocnemius; SOL, soleus; TA, tibialis anterior and PL, peroneus longus.

6.4 Discussion

The purpose of this study was to investigate the effect of eccentric EIMD on AT stiffness and strain. This is the first study to investigate the mechanical behaviour of tendon following an exercise activity specifically used to induce muscle damage. The main findings of this study were that AT stiffness increased 24 hours post-EIMD and AT strain levels decreased at 24 and 48 hours post-EIMD. These results were in contrast to both hypotheses.

High levels of strain in tendon are known to cause microtrauma to collagen fibres within tendon and increase the risk of tendon rupture injury accordingly (Arya & Kulig, 2010; Butler, et al., 1978). The reduction in the level of tendon strain following EIMD in this study potentially suggests there is a protective mechanism that alters elongation and, consequently, strain. This protective mechanism may be explained by progressive collagen fibre recruitment that has been shown to prevent ligament damage (Thornton, Oliynyk, Frank, & Shrive, 1997; Thornton, Shrive, & Frank, 2002). The recruitment of more collagen fibres is thought to reduce tendon stress (force/area) by spreading the stress over a greater area. As a result, an increase in collagen fibre recruitment may have lead to a reduction in tendon elongation and, hence, strain.

Moreover, whilst force production of the AT across the five testing sessions was comparable with previous research (Lichtwark & Wilson, 2005; Magnusson, et al., 2001; Muraoka, Muramatsu, Fukunaga, & Kanehisa, 2005; Rosager, et al., 2002), there was no difference in force production between baseline and any of the sessions. The absence of change in force was

confirmed by no change in the EMG activity of the medial gastrocnemius, lateral gastrocnemius, soleus, tibialis anterior or peroneus longus following EIMD, implying no change in neural input or muscle activation patterns. Taken together, this strengthens the theory that there is a mechanism occurring within the muscle-tendon unit to protect the AT against high levels of strain.

The AT stiffness levels measured during this study ranged from 187 to 307 N/mm and were similar to those of other studies (49 – 390 N/mm) (Hansen, Aagaard, Kjaer, Larsson, & Magnusson, 2003; Kubo, et al., 2004b; Maganaris & Paul, 1999; Muraoka, et al., 2005; Rosager, et al., 2002). The mechanical properties of tendon are important to the time course of torque development (Reeves, et al., 2003). For example, a more compliant tendon will require more time to stretch and, hence, more time to stretch other series elastic components within the muscle-tendon unit, decreasing the rate of torque development. Tendon stiffness is lower in older individuals and is thought to result in a less rapid execution of motor tasks and, therefore, increase the risk of falling (Reeves, et al., 2003). Therefore, the increase in AT stiffness shown in this study is potentially occurring to provide more efficient and economical torque development by reducing tendon compliance (Reeves, et al., 2003).

Additionally, mechanical stretching of tendon has been shown to alter the cellular production of collagen type I and induce tendon remodelling (Kim, Akaike, Sasagawa, Atomi, & Kurosawa, 2002). Perhaps the reduction in tendon elongation may be a result of remodeling of the tendon architecture to compensate for the mechanical weakness caused by the EIMD (Miller, et al.,

2005). Other possible explanations for the increase in AT stiffness include increased tendon collagen content (Miller, et al., 2005), increased crimp angle of collagen fibrils (Wood, Cooke, & Goodship, 1988), and increased tendon water content (Haut & Haut, 1997); however, these characteristics are difficult to measure *in vivo* and were not measured and, thus, are only speculation.

A higher level of AT stiffness corresponded to a lower level of AT strain in this study. Achilles tendon strain was reduced at 24 and 48 hours post EIMD and the levels ranged between 3.8 to 6.5%, which is consistent with previous research reporting levels ranging from 0.8 to 10.3% (Arampatzis, et al., 2005; Kubo, et al., 2004a; Kubo, Kanehisa, & Fukunaga, 2003; Lichtwark & Wilson, 2005; Magnusson, et al., 2001; Muramatsu, et al., 2001; Muraoka, et al., 2005; Rosager, et al., 2002). The AT strain results of this study are similar to previous research that investigated an eccentric training program performed over 18 weeks (Duclay, et al., 2009). These findings may imply that following EIMD, there was an increase in cross-sectional area (CSA) of the AT which is associated with increased levels of tendon stiffness (Couppe, et al., 2008). However, this is unlikely and, while we did measure the intrinsic mechanical properties of AT; we did not measure the geometric changes (e.g. CSA). Therefore, cannot confirm the implications of these findings and they are merely speculative in nature and, therefore, reader caution is advised.

In the present study, AT stiffness remained unchanged immediately post EIMD exercise. This is similar to past research investigating the effect of fatigue and low strain rates caused by exercise on AT stiffness (Mademli, et al., 2006,

2008; Peltonen, et al., 2010). Previous studies have explored the long-term effects of fatigue on tendon and shown that AT stiffness is unchanged after the chronic mechanical loading of a training program. Repeated, cyclical loading (Arampatzis, et al., 2007) and eccentric training (Mahieu, et al., 2008) over 6 -14 weeks of training did not change AT stiffness. In addition, acute, short-term intervention studies have demonstrated that exercise consisting of a single isometric plantar flexion contraction until fatigue does not effect AT stiffness levels (Mademli, et al., 2006, 2008). Furthermore, no acute changes in AT stiffness have been found in double-legged hopping until fatigue (Peltonen, et al., 2010), with the approximate 15 minutes of hopping in Peltonen, et al. (2010) consisting of 1150 – 2600 contractions, comparing well with the 1800 – 2100 contractions performed by walking backward in this study. Moreover, perhaps the AT is resistant to submaximal contractions without altering its mechanical properties by exhibiting a strain threshold at which tension homeostasis of the tendon's extracellular matrix exists that leads to anabolic responses of tendon (Wang, 2006). Consequently, our results may suggest that the AT displays a capability to resist fatigue. However, caution must be taken in interpreting the results this way, as we did not account for changes that may have occurred in the stiffness of the gastrocnemius muscle or tendon aponeurosis.

Tendon degeneration resulting in tendinopathy has been shown to weaken the mechanical characteristics and alter the material properties of the human AT (Arya & Kulig, 2010; Child, Bryant, Clark, & Crossley, 2010). Injured or degenerated AT exhibits a lower level of tendon stiffness and a higher level of

tendon strain than healthy tendons. This suggests that the changes that have occurred in this study may be a result of changes in the stiffness of the gastrocnemius muscle. One investigation into the changes in AT strain levels between participants with Achilles tendinopathy and those without suggested that the differences between groups could not be solely accounted for by changes in the mechanical behaviour of tendon (Child, et al., 2010). Moreover, tendon and aponeurosis and muscle offer separate functional roles in force transmission, highlighting the possibility of changes occurring in the muscle (Magnusson, et al., 2003). Therefore, any changes that are occurring in the stiffness and strain of the AT of this study may not be occurring in the tendon alone.

Furthermore, eccentric exercise has been used to investigate changes in the mechanical behaviour of muscle. Following repeated eccentric contractions similar to those performed in this study, changes in the length-tension or torque-angle relationship of muscle have been found to occur (Morgan & Proske, 2004; Whitehead, et al., 2001). Eccentric contractions cause damage to sarcomeres within the myofibrils of muscle by stretching them beyond their optimal length (Morgan & Proske, 2004). During experiments on muscle fibre, short or weak sarcomeres are disrupted during active lengthening and stretching beyond their capabilities. Furthermore, upon muscle relaxation following contraction, there is potential that the rapid recoil of tendon also causes sarcomere disruption due to muscles still being partially active (Gregory, Morgan, Allen, & Proske, 2007). As a result of these structural changes, muscle increases both its compliance and the length at which it is able to optimally produce tension (Gregory, et al., 2007),

suggesting that changes can occur within muscle following EIMD. Again, caution must be taken in interpreting these results as the findings of Gregory et al., were from conducted *in vitro* on cats.

The notion that, in this study, EIMD-associated changes are occurring in the muscle is supported by time-course changes observed in muscle following EIMD. Repeated high-force eccentric contraction causes the force-length characteristics of muscle not only to shift to longer lengths but to last for up to four days (Philippou, Bogdanis, Nevill, & Maridaki, 2004). These shifts in optimal muscle length have also been shown to return to resting levels after eight days (Prasartwuth, Allen, Butler, Gandevia, & Taylor, 2006). The results in this study showed that changes in the muscle-tendon unit occurred at 24 and 48 hours post EIMD. Upon considering the time-course changes that have occurred and, the results of previous research, there is reason to suggest and support the theory that these changes are occurring in muscle alone. However, the measurements in this study were taken during a constant muscle length (isometric), therefore, it is purely speculative that the force-length characteristics of the gastrocnemius were altered to longer lengths.

To put in perspective the mechanics of the muscle-tendon unit after EIMD, consider the muscle and tendon being a combination of two springs joined in series. For a given level of force or tension, an increase in compliance of muscle would lead to an increase in the stiffness of tendon, just as a decrease in tendon compliance would lead to an increase in muscle compliance. Whilst it can be theorised that the changes in this study have occurred in either the muscle or the

tendon, we cannot confirm that the changes have occurred exclusively in one alone and, therefore, changes in both may be possible. Repeated eccentric contraction alters the mechanical properties of muscle to produce force at longer lengths (Whitehead, et al., 2001) and become more compliant (Gregory, et al., 2007). Moreover, the repeated application of strain on tendon during eccentric contraction can cause collagen synthesis or degradation (Kim, et al., 2002), and alter the biological and mechanical properties of tendon, thereby affecting tendon compliance (Kubo, Kanehisa, Ito, et al., 2001). However, because tendon is less deformable and malleable than muscle, there is also potential that changes are occurring within the muscle and the tendon concurrently. Consequently, as suggested previously, the EIMD-associated changes in the muscle-tendon unit may be a result of a protective mechanism occurring to reduce high tendon strain levels. Therefore, not only are the repeated contractions resulting in EIMD, causing changes in tendon, but also causing changes in muscle mechanics.

6.4.1 Conclusion

Studies investigating tendon stiffness and the influence of training have found it difficult to draw conclusions on the exact mechanisms behind the mechanical changes in tendon (Foure, Nordez, & Cornu, 2010; Kubo, et al., 2007). However, this study found that changes in AT stiffness and strain levels of active males are associated with EIMD. There were no changes in AT stiffness or strain immediately post exercise; however, at 24 and 48 hours,

changes were evident. Plantar flexor force and muscle activity were not affected by the repeated eccentric contractions of walking backwards downhill for 60 minutes. These findings suggest that the changes in AT stiffness and strain are occurring as a protective mechanism against increasing levels of strain on the tendon or to provide effective and efficient force transmission capabilities. However, we cannot discount that the changes may be occurring within muscle independently, or that changes are a result of a combination of shifts in the mechanical properties of both muscle and tendon.

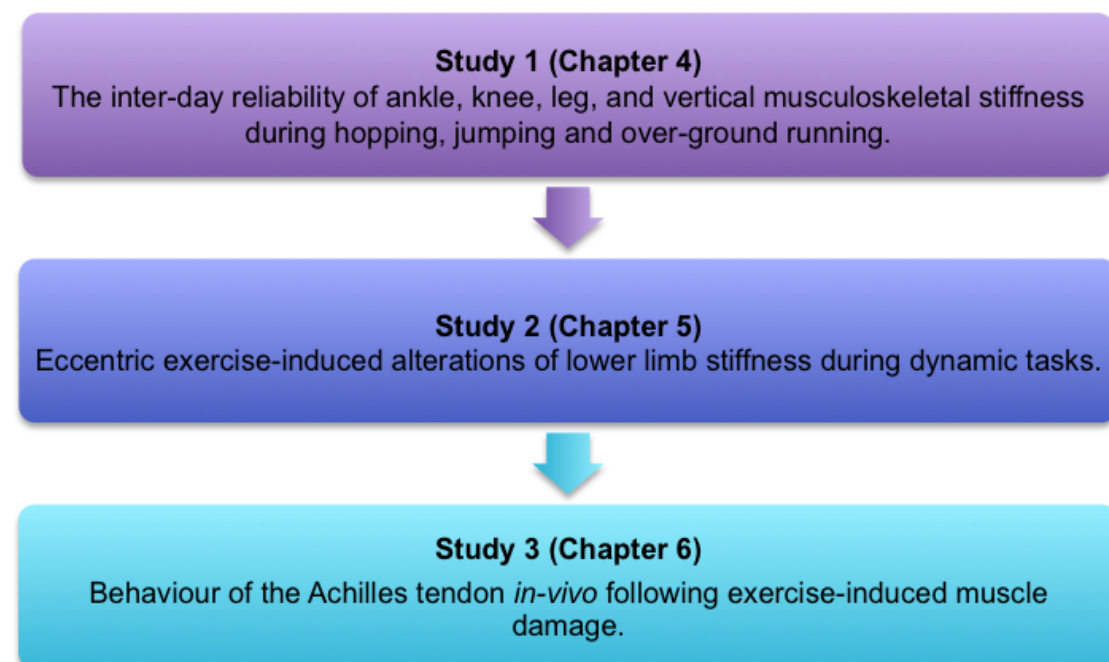
7 Summary and conclusion

7.1 Introduction and general overview

This study explored the acute effects of muscle damage on levels of MSS (global measures) and tendon mechanical properties (local measure). The major aim was to investigate whether changes in musculoskeletal stiffness (MSS), Achilles tendon (AT) strain and AT stiffness, in a sample of active males, are associated with exercise-induced muscle damage (EIMD) caused by repeated eccentric contractions. The following section provides a review of the sequence of the studies comprising this thesis, a synthesis of the hypotheses and major outcomes and a general discussion of the findings of each of the studies.

7.1.1 Thesis sequence

THESIS SEQUENCE



7.2 Summary of hypotheses and major findings

Study 1: The inter-day reliability of ankle, knee, leg, and vertical musculoskeletal stiffness during hopping, jumping and over-ground running (Chapter 4).

Hypothesis (i) MSS measures (vertical, leg) of running and hopping at a set pace (velocity or frequency) are reliable, whereas the same tasks performed at a self-selected pace lack sufficient reliability.

This hypothesis was partially supported by the results of this study. There was ‘good’ reliability identified for measures of K_{vert} and ‘moderate’ reliability for K_{leg} during the set-paced running task. Similar results during hopping and jumping at 2.2 Hz were observed with ‘good’ reliability identified for K_{vert} . Furthermore, as hypothesised, hopping at a self-selected frequency displayed ‘poor’ to ‘moderate’ reliability for K_{vert} . Therefore, taken together, these results suggest that K_{vert} is a reliable MSS measure when running at 3.83 m/s and hopping and jumping at 2.2 Hz.

Hypothesis (ii) Joint MSS measures (ankle and knee) of running and hopping at either a set or self-selected pace are less reliable than global measures due to increased task performance variation in joint movement patterns.

‘Poor’ reliability was revealed for K_{knee} and K_{ank} during the running task. Similarly, ‘poor’ reliability was shown for K_{knee} and K_{ank} during hopping and jumping at 2.2 Hz. These findings support the proposed hypothesis that joint

stiffness measures are not reliable during set-paced tasks. Additionally, hopping at a self-selected frequency displayed 'poor' to 'moderate' reliability for K_{knee} and K_{ank} , also supporting our hypothesis. The lack of reliability in joint MSS results were substantiated by the large variation in joint kinematics and joint kinetics between day 1 and 2. Joint moment and angular displacement results indicated that there was a large variation in task performance displayed by the large $M_{diff\%}$ results ($>5\%$) and/or unacceptable ICCs (<0.80) during running, hopping and jumping. These findings also support the hypothesis that the lack of reliability in joint MSS may be explained by an inconsistent movement pattern and task performance variation.

Study 2: Eccentric exercise-induced alterations of lower limb stiffness during dynamic tasks (Chapter 5).

Hypothesis (iii) *There is a reduction in leg MSS, compared to baseline, with EIMD and these changes are associated with changes in ankle kinematics and muscle activity during the hopping task.*

This experiment identified no differences in K_{leg} , ankle kinetics and ankle kinematics between baseline and the immediately post-exercise, 24 hr, 48 hr and 168 hr post-exercise time points. Furthermore, no differences were found in EMG activity of the plantar flexor muscles of the dominant limb or in ankle kinematics with EIMD. The results of this study, therefore, do not support the hypothesis. However, differences in knee kinematics during single-legged hopping were observed. This suggests, therefore, that knee

mechanics may be altered to maintain consistent levels of K_{leg} and K_{vert} when EIMD is present in the lower legs.

Hypothesis (iv) *There is no change in leg MSS, vertical MSS, knee and ankle joint kinematics, or muscle activity during the running task with EIMD.*

The results of this experiment showed no differences in K_{leg} or K_{vert} between baseline and the immediately post-exercise, 24 hr, 48 hr and 168 post-exercise time points during running. No differences were found in ankle or knee joint displacement and, therefore, the hypotheses of this study that there would be no change in leg MSS, vertical MSS, knee and ankle kinematics was supported by these results. It is suggested, therefore, that K_{leg} , K_{vert} , knee and ankle mechanics are not altered when EIMD is present in the lower legs.

Study 3: Behaviour of the Achilles tendon *in-vivo* following exercise induced muscle damage (Chapter 6).

Hypothesis (v) *Achilles tendon strain is increased with EIMD.*

The hypothesis that AT strain would increase with EIMD was not supported by the findings of this study. Significant decreases in the level of AT strain were identified at 24 and 48 hours post EIMD.

Hypothesis (vi) *Achilles tendon stiffness is reduced with EIMD.*

Results from this study indicated that there was an increase in AT stiffness at 24 and 48 hours post EIMD. Therefore, the hypothesis that AT stiffness would be reduced in the presence of EIMD was not supported.

The alterations in AT stiffness and strain suggest that the EIMD protocol affected the mechanical properties of the muscle-tendon unit resulting in alterations to maintain the force transmission capabilities of the muscle-tendon unit.

7.3 Discussion

The purpose of this section is to provide an integration of the findings of this thesis with the outcomes of previous research relevant to MSS and EIMD. The relevance of these findings will also be discussed and suggestions for future research will be presented.

7.3.1 The inter-day reliability of K_{ank} , K_{knee} , K_{leg} and K_{vert} , during hopping, jumping and over-ground running.

As outlined in Chapter 2.2.1, there are a number of methodologies adopted in the literature to assess MSS; however, only a small number of studies have attempted to rigorously evaluate the reliability of these measures (Allison, et al., 1998; Hunter & Spriggs, 2000; McLachlan, et al., 2006; Watsford, et al., 2003). Furthermore, the methods adopted in this thesis are commonly used throughout the literature (see reviews by Brughelli & Cronin, 2008a, 2008b; Butler, et al., 2003) and contain one component that has been previously determined as reliable (McLachlan, et al., 2006). Therefore, to fully

understand the outcomes of study 2 and 3, the reliability of the methods to be used was examined.

This study identified the most reliable MSS measures during common tasks used to investigate MSS. These findings informed the development of the methodology that was subsequently used for studies 2 and 3 of this thesis. Tasks that were performed under controlled speeds or frequencies led to a larger number of reliable measures. Furthermore, the MSS variables that were measures of more global, whole leg-spring behaviour were more reliable than local, joint-spring behaviour. Although reliability of MSS variables has been assessed previously, this was the first study to examine the inter-day reliability of a testing protocol used to assess MSS during over-ground running. This was also the first study to explore the reliability of MSS at set and self-selected frequencies during jumping and hopping tasks using a single and double-legged action.

The findings of this research (study 1) can be applied in practice by researchers when considering the suitability of MSS measures that may be of interest during investigations into stretch-shortening cycle tasks. Furthermore, reporting the reliability data of study 1 is important because MSS is often used to investigate changes in athletic performance or to potentially link MSS with injury. Moreover, it allows *a priori* sample size analysis and reveals the suitability of certain measures of MSS to be used in research where an intervention effect is of interest.

7.3.2 New insights into the effect of DOMS on the behaviour of the musculoskeletal system.

The purpose of study 2 and 3 of this thesis was to investigate the effect of eccentric EIMD on kinematics, kinetics, MSS, AT strain and AT stiffness. To the author's knowledge, this is the first study to explore the changes in MSS and in the mechanical behaviour of the AT over a short- or long-term time frame following an exercise activity specifically implemented to induce muscle damage. Furthermore, this study is the first to investigate the effect of muscle damage caused by repeated eccentric contractions on the kinematics and kinetics of jumping at 2.2 Hz and hopping at 2.2 Hz.

Whilst there is evidence concerning the effect of EIMD on gait dynamics (Dutto & Braun, 2004; Paschalis, et al., 2007; Tsatalas, et al., 2010), the effects are unknown on other fundamental sporting actions such as hopping and jumping. Additionally, fatigue similar to that caused by EIMD has been shown to affect MSS (Kuitunen, et al., 2007); however, evidence is lacking on the effect over an extended time frame. Repeated mechanical loading that is common to EIMD has been shown to alter tendon properties (Duclay, et al., 2009), yet the direct causal effect that EIMD has on in-series elastic structures, such as tendon, is unknown. Consideration of these previous findings and the results of this thesis may help elucidate the impact that EIMD has on the mechanical and positional changes of the joints and the mechanical and neuromuscular behavior of the lower leg during a range of sporting activities.

EIMD had no effect on the kinematic, kinetic or MSS variables during the hopping and jumping movements tested in this study, except for

decreases in knee touchdown angle during dominant-legged hopping. The finding that MSS was not altered by EIMD is confirmed by the kinematic and kinetic results of study 2. This is not surprising given that MSS is determined by a combination of kinematic and kinetic variables. Additionally, running kinematics have been shown to be altered in the presence of EIMD (Hamill, et al., 1991). However, study 2 indicated that the EIMD protocol adopted may not have been intense enough to cause large changes in joint behaviour during running, hopping or jumping.

The results from study 3 showed that there were changes in the mechanical properties of the AT following EIMD. But, whilst the behaviour of the AT was primarily measured in this study, the changes seen may not be a result of changes in tendon alone but potentially in the aponeurosis or in the muscle belly, or a result of changes in all three. Furthermore, neuromuscular control, as measured by muscular EMG, was not altered in the presence of EIMD.

These findings are relevant to performance and injury paradigms. Specifically, the kinematic and kinetic findings of study 2 have practical relevance to mechanisms associated with injury prevention. The ability of the musculoskeletal system to perform safe movements can be determined by the proprioceptive capacity of the system. If the capacity to perform a movement is altered, then perturbations during common, fundamental daily activities may lead to an increased risk of injury. Also, changes in kinematics and kinetics caused by EIMD have been suggested to increase the risk of injury, as well as alter factors related to injury such as force production and muscle activation (Deschenes, et al., 2000; Nottle & Nosaka, 2005a). With respect to knee

mechanics, these may be altered as a protective mechanism against pain or further damage when EIMD is present in the lower legs. Reduced knee joint angle during the stance phase of running has been suggested to reduce the shock absorption ability and transfer loads to articular joints, the head and neck, increasing the potential for musculoskeletal injury (Lafortune, Hennig, et al., 1996; Lafortune, Lake, & Hennig, 1996). Therefore, the findings of this thesis provide some insight into injury risk following EIMD during hopping, jumping and running and make highlight an area for future research.

Spring energy harnessed from eccentric contractions is used to enhance musculoskeletal performance and minimise the amount of mechanical energy required for movement. The utilization of this type of spring energy is facilitated by tendon and controlled by the neuromuscular system through MSS regulation. Therefore, when considering the paradigm of sporting performance, the changes that occurred in the muscle-tendon unit observed in this thesis, as a result of EIMD, have implications for the ability of the musculoskeletal system to provide elastic energy for effective and efficient movement, and on the ability of tendon to transfer contractile force.

Whilst spring energy is essential to athletic performance, the repeated eccentric contractions that facilitate the storage and recoil of spring energy can cause muscle damage. The changes observed occur in the muscle-tendon unit following EIMD may be taking place to protect the tendon against high magnitudes of strain that lead to injury. This, therefore, has implications for the theories relating to, and the application of, rehabilitation and recovery principles following exercise that induces muscle damage, as well as sporting performance. Specifically, these findings may provide some value to

clinicians when grading muscle strain injuries and in making safe return-to-play decisions particularly, when low levels of muscle damage develop into more serious injuries.

Consequently, the outcomes of this thesis provide relevance to performance and injury prevention through the importance of understanding the neuromuscular and mechanical responses within the musculoskeletal system following repeated mechanical loading, and particularly loading that causes muscle damage.

7.4 Future directions

Whilst this thesis has established the inter-day reliability of MSS measures during hopping, jumping and running tasks, the lack of reliability in the majority of joint variables provides avenues for further research. Therefore, the use of several re-tests would help determine the effect of familiarisation on reliability. Moreover, an appraisal with regards to the reliability that individual participants exhibit would help to determine whether the participant or the methodology is the primary source of questionable reliability. In light of pursuing a methodology that is reliable in assessing joint stiffness, manipulating protocol variables such as sampling rate or hopping frequency may be fruitful in revealing improvements in reliability.

To date, there is a paucity of research that investigates the effect of EIMD on actual competitive performance (Kyrolainen, Takala, & Komi, 1998; Semark, et al., 1999). A deeper understanding of the effect of EIMD on performance will provide clinicians, athletes, and sports scientists with

practical knowledge of these effects and, consequently, the implications with respect to injury. The majority of studies that have investigated EIMD in untrained individuals and the protocols used to induce EIMD are either dissimilar to any action performed during sports participation or not practical for investigating sports performance. Therefore, studies employing actions that are common within the cohort of interest and studying a population that is highly trained will enable the extent of muscle damage from relevant movement patterns on athletic performance to be interpreted. Moreover, exploring EIMD caused by actual competition or training may provide more relevant insights, as current protocols typically use specific activities to target individual muscle groups.

The mechanical properties of tendon investigated in this thesis were limited to stiffness and strain. Future research might consider the effect of EIMD on other mechanical properties of tendon such as hysteresis, cross-sectional area (CSA), Young's modulus and stress. This will help to give a more complete picture of the changes in the mechanical properties of tendon and, thus, the implications of these changes on tendon behaviour. Furthermore, to gain a clearer understanding of the properties that are altered following EIMD, the mechanical behaviour of tendon and aponeurosis and muscle should all be considered. This will provide a more complete overview of the alterations in mechanical properties and interactions between these components of the muscle-tendon unit following repeated eccentric exercise that causes damage. Due to the location, role and characteristics (e.g. tendon thickness) of the AT in locomotion, it is least affected by low strain

magnitudes (Arampatzis, et al., 2007). Therefore, exploration into the effect of EIMD on other tendons may provide interesting results concerning the influence of EIMD on tendons that perform different functions within the musculoskeletal system and the consequences of these changes to movement.

7.5 Concluding remarks

In conclusion, the findings of this thesis advance knowledge in the musculoskeletal behaviour of active males following EIMD. EIMD causes changes in the mechanical properties of tendon in a cohort of young, active males. Other musculoskeletal properties such as leg MSS, vertical MSS, force production and muscle activity do not appear to change. Furthermore, there was no difference between the characteristics of MSS or any kinematic and kinetic variables in the dominant and non-dominant limbs following EIMD. These findings suggest that repeated eccentric contractions of the plantar flexors cause changes in the mechanical properties of the AT, aponeurosis or muscle but do not alter neuromuscular control. Knowledge gained from the data in this thesis is provided through an insight into behavior of the musculoskeletal system with muscle damage present and, therefore, provides information relevant to injury prevention and rehabilitation programs. Deeper examination is required to explain the mechanisms behind altered MSS regulation and into the specific mechanisms changed by EIMD within the AT and the muscle-tendon unit.

Appendix A: Informed consent form

CONSENT FORM (PARTICIPANT'S COPY)

TITLE OF PROJECT: Is delayed onset muscle soreness associated with musculoskeletal stiffness?

NAME OF PRINCIPAL SUPERVISOR: Dr Elizabeth Bradshaw

NAME OF STUDENT RESEARCHER: Mr. Corey Joseph

I have read (*or, where appropriate, have had read to me*) and understood the information provided in the Letter to Participants. Any questions I have asked have been answered to my satisfaction. I give consent to participate in this activity, acknowledging that whilst participating I will be recorded via 3D motion analysis equipment. I understand that the study will require approximately a total of 6 hours attendance spread over 5 separate days (e.g. approximately 1 hr per day) and that I can withdraw at any time from the study without any consequences of withdrawal. I also give consent to having my blood sampled on each of the 5 days and give consent to providing the researches with personal information in questionnaires. I understand that eccentric exercise often results in mild muscular discomfort, consistent with the discomfort experienced with returning to sport after a lengthy break. I understand that all information collected will remain private and that all data will be re-identified by codes only known by the researchers. I agree that research data collected for the study may be published or may be provided to other researchers in a form that does not identify me in any way.

NAME OF PARTICIPANT:

(Block letters)

SIGNATURE: DATE:

SIGNATURE OF RESEARCHER:

..... DATE:

CONSENT FORM (RESEARCHER'S COPY)

TITLE OF PROJECT: Is delayed onset muscle soreness associated with musculoskeletal stiffness?

NAME OF PRINCIPAL SUPERVISOR: Dr Elizabeth Bradshaw

NAME OF STUDENT RESEARCHER: Mr. Corey Joseph

I have read (*or, where appropriate, have had read to me*) and understood the information provided in the Letter to Participants. Any questions I have asked have been answered to my satisfaction. I give consent to participate in this activity, acknowledging that whilst participating I will be recorded via 3D motion analysis equipment. I understand that the study will require approximately a total of 6 hours attendance spread over 5 separate days (e.g. approximately 1 hr per day) and that I can withdraw at any time from the study without any consequences of withdrawal. I also give consent to having my blood sampled on each of the 5 days and give consent to providing the researches with personal information in questionnaires. I understand that eccentric exercise often results in mild muscular discomfort, consistent with the discomfort experienced with returning to sport after a lengthy break. I understand that all information collected will remain private and that all data will be re-identified by codes only known by the researchers. I agree that research data collected for the study may be published or may be provided to other researchers in a form that does not identify me in any way.

NAME OF PARTICIPANT:
(Block letters)

SIGNATURE: DATE:

SIGNATURE OF RESEARCHER:

..... DATE:

Appendix B: Information letter to participants (Study 1)

INFORMATION LETTER TO PARTICIPANTS

TITLE OF PROJECT: The inter-day reliability of a functional protocol used to investigate lower extremity musculoskeletal stiffness in Males.

NAME OF SUPERVISOR: Dr. Elizabeth Bradshaw

NAME OF STUDENT RESEARCHER: Mr Corey Joseph

PROGRAMME IN WHICH ENROLLED: Doctorate of Philosophy

Dear Participant,

You are invited to take part in a project which will examine the reliability of a testing method which assesses lower extremity musculoskeletal stiffness. This study follows past research related to stiffness however; it is exploring an area of ideas that has not yet been covered a great deal. This area of ideas involves investigating the most reliable, accurate and consistent method of determining stiffness. This method will then be used in future testing protocols of the student researchers Doctorate to determine the link between stiffness and injury.

This study is deemed to be of low risk and requires drop jumps from three standardised heights, a 15m running test, and single and double legged hopping to be performed. Your suitability for participating in this test will be determined on the completion of a Health Screening questionnaire. No injured athletes will be required. It is important that this form is completed as accurately as possible.

Testing will be undertaken at the Australian Catholic University Exercise Science Laboratory, (LG) Lower ground (115 Victoria Parade, Fitzroy, MDC, VIC, 3065). Testing will require participants to come 2 days (possibly a third) and will take an hour on each day. Simple anthropometric measurements of height, sitting height, weight, and bone lengths will be taken, followed by the placement of reflective markers on the lower legs and a detailed, 3D analysis of the running and jumping tasks.

Participation in this study will help provide valuable information on the accuracy of the testing procedure and whether it is acceptable to be used in future testing of the student researchers Doctorate. There is no direct benefit to participants however the experience of participating in a research activity may provide individuals with insight into the rigours of scientific research. Feedback on your performance during the tasks undertaken can be provided upon request.

As this study is volunteer based, you are free to refuse consent altogether without having to justify your reason. Withdrawal of consent will also be regarded the same way. Withdrawal

will not prejudice your future with coaches or sport. Any feedback on the results of the test will be provided to you upon request.

It is anticipated that results from these tests will be published in scientific and/or medical journals. However, all information you provide will remain confidential. A coding system will be used to ensure individuals can't be identified. All data will be stored in a locked filing cabinet only accessed by the principal investigator.

Any questions regarding this study, or any issues raised in this information letter may be directed to any of the following:

Mr Corey Joseph (Student supervisor) corey.joseph@acu.edu.au
Dr. Elizabeth Bradshaw (Principal investigator) elizabeth.bradshaw@acu.edu.au

School of Exercise Science, ACU National
115 Victoria Parade, Fitzroy, Vic, 3065.

This study has been approved by the Human Research Ethics Committee at Australian Catholic University. In the event you may have a complaint or concern about the way you have been treated during the study, or if you have any query that the Investigators have not been able to satisfy, you may write to the Chair of the Human Research Ethics Committee care of the address below:

Chair HREC
C/o Research Services
Australian Catholic University
Melbourne Campus
Locked Bag 4115
Fitzroy, Vic, 3065
Tel: 03 9953 3158
Fax: 03 9953 3315

Any complaint or concern will be treated in confidence and fully investigated. The participant will be informed of the outcome.

If you agree to participate in this project, you should sign both copies of the Consent Form, retain one copy for your records and return the other copy to the Investigator or Student Researcher. Thank you for your Co- Operation with this research

Yours sincerely,

Dr. Elizabeth Bradshaw
Principal Supervisor

Mr. Corey Joseph
Student Researcher

Appendix C: Information letter to participants (Study 2 & 3)

INFORMATION LETTER TO PARTICIPANTS

TITLE OF PROJECT: Is delayed onset muscle soreness associated with musculoskeletal stiffness?

NAME OF SUPERVISORS: Dr. Elizabeth Bradshaw & Dr. Justin Kemp

NAME OF STUDENT RESEARCHER: Mr Corey Joseph

PROGRAMME IN WHICH ENROLLED: Doctorate of Philosophy

Dear Participant,

You are invited to take part in a project which will examine the relationship between delayed onset muscle soreness (DOMS) and musculoskeletal stiffness. Currently, musculoskeletal stiffness has predominantly focussed on sports performance. However, the relationship between DOMS and musculoskeletal stiffness is unknown, even though DOMS is a common sensation following sports participation. The aim of this study, therefore, is to determine the effect that DOMS has on musculoskeletal stiffness.

This study is deemed to be of low risk; however, some discomfort is expected which is not deemed to be unfamiliar or high. Your suitability for participating in this test will be determined on the completion of a Health Screening questionnaire and it is important that this form is completed as accurately as possible. Injured athletes are not required. The study will require participants to perform a number of tests over 5 separate days and it is expected that your participation in this study will involve approximately 6 hours in total.

Testing will be undertaken at the Australian Catholic University Exercise Science Laboratory, Lower ground level (115 Victoria Parade, Fitzroy, MDC, VIC, 3065). Testing will require participants to come to this address for 5 sessions. The first session will involve a maximal oxygen uptake test (VO_2 max), where your aerobic power will be assessed. Session 2 will involve some baseline measures of musculoskeletal stiffness during a running and hopping task, followed by downhill running and backward walking. This will then be immediately followed with the same pre-test measures. Over the next 3 sessions (24, 48 hrs and 7 days post) the same pre-post test measures will be performed. All sessions will require intravenous blood samples to be collected.

Your participation in this study will help provide valuable information concerning the relationship between DOMS and musculoskeletal stiffness, which is currently unknown. From these results, prospective screening of potential injury risk may be developed. Further,

participants will be exposed to gold-standard maximal oxygen uptake and biomechanical laboratory-based testing and analysis at no cost. Feedback on performance during the tasks can be provided upon request.

As this study is volunteer based, you are free to refuse consent altogether without having to justify your reason. Withdrawal of consent will also be regarded the same way. Withdrawal will not prejudice your future with coaches or sport.

It is anticipated that results from these tests will be published in scientific and/or medical journals. However, all information you provide will remain confidential. A coding system will be used to ensure that individuals cannot be identified. All data will be stored in a locked filing cabinet or password protected computer files only accessed by the principal investigator.

Any questions regarding this study, or any issues raised in this information letter, may be directed to any of the following:

Mr Corey Joseph (Student researcher) corey.joseph@acu.edu.au
Dr. Elizabeth Bradshaw (Principal investigator) elizabeth.bradshaw@acu.edu.au

School of Exercise Science, ACU National
115 Victoria Parade, Fitzroy, Vic, 3065.

This study has been approved by the Human Research Ethics Committee at Australian Catholic University. In the event you may have a complaint or concern about the way you have been treated during the study, or if you have any query that the Investigators have not been able to satisfy, you may write to the Chair of the Human Research Ethics Committee care of the address below:

Chair HREC
C/o Research Services
Australian Catholic University
Melbourne Campus
Locked Bag 4115
Fitzroy, Vic, 3065
Tel: 03 9953 3158
Fax: 03 9953 3315

Any complaint or concern will be treated in confidence and fully investigated. The participant will be informed of the outcome.

If you agree to participate in this project, you should sign both copies of the Consent Form, retain one copy for your records and return the other copy to the Investigator or Student Researcher. Thank you for your cooperation with this research.

Yours sincerely,

Dr. Elizabeth Bradshaw
Principal Supervisor

Mr. Corey Joseph
Student Researcher

Appendix D: Health screening questionnaire

Health Screening Questionnaire – Part A

1. Personal Details

Name: _____

Address: _____

Telephone: _____ (AH) _____ (W)

Date of Birth: _____

2. Medication

Are you currently taking any medication or drugs (circle) ?

Yes **No**

If yes, please list name, duration and reason for use. Please include any anti-inflammatory drugs, ibuprofen or pain killers.

3. Medical and Surgical History

- Have you ever been hospitalised? If yes, when and why?

- Do you have any allergies (circle)?

Yes **No**

If yes, please specify:

- Have you ever experienced abnormal responses during exercise out of proportion to the activity undertaken (e.g. chest pain, abnormal shortness of breath, fainting, abnormal bleeding or bruising) (circle)?

Yes **No**

If yes, please specify:

- Do you have, or have you ever had, any of the following conditions (circle)?

If yes, specify (e.g. where, when, for how long, etc)

Musculo-skeletal dysfunction (e.g. cerebral palsy)	Yes	No	
Neuromuscular dysfunction (e.g. multiple sclerosis)	Yes	No	
Overuse injuries (e.g. tendonitis)	Yes	No	
High blood pressure	Yes	No	
Blood or heart disorder	Yes	No	
Asthma or other respiratory condition	Yes	No	
Traumatic injuries/surgeries	Yes	No	
Diabetes	Yes	No	
Arthritis	Yes	No	
Visual impairment	Yes	No	
Persistent vertigo	Yes	No	
Visual impairment	Yes	No	
Light-headedness	Yes	No	
Stroke	Yes	No	
Other	Yes	No	

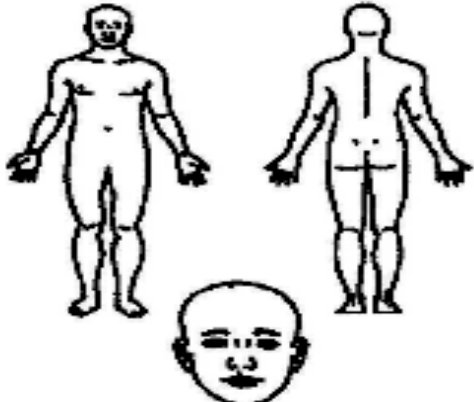
- Have you ever experienced any musculoskeletal injuries?

Yes

No

If NO, skip to page 5. If YES, fill out the following section.... PTO

Sport/Activity in which injury occurred: _____
 Event / Position played (if appropriate): _____
 Approx hrs of training / competition per week: _____

<p>Date of Injury ___/___/___</p> <p>Type of activity at time of injury</p> <ul style="list-style-type: none"> <input type="checkbox"/> Warm-up <input type="checkbox"/> Game <input type="checkbox"/> Supervised training <input type="checkbox"/> Unsupervised training or play <p>When did the injury occur?</p> <ul style="list-style-type: none"> <input type="checkbox"/> start of sport /training <input type="checkbox"/> about the middle of your sport/training <input type="checkbox"/> towards the end of sport/ training <p>Injury History</p> <ul style="list-style-type: none"> <input type="checkbox"/> new injury <input type="checkbox"/> exacerbated / aggravated injury <input type="checkbox"/> recurrent injury <input type="checkbox"/> chronic condition <p>If you have had this injury before, how long ago was it?</p> <ul style="list-style-type: none"> <input type="checkbox"/> < 1 month ago <input type="checkbox"/> 1 - 3 months ago <input type="checkbox"/> 3 - 6 months ago <input type="checkbox"/> 6 - 12 months ago <input type="checkbox"/> >12 months ago <p>Body Region Injured Tick or circle body part/s injured and name</p> <div style="text-align: center;">  </div> <p>Body Part/s (List as much detail as possible)</p> <p>_____</p> <p>_____</p> <p>_____</p>	<p>Mechanism of Injury</p> <ul style="list-style-type: none"> <input type="checkbox"/> collision with fixed object <input type="checkbox"/> collision with person <input type="checkbox"/> sudden stopping <input type="checkbox"/> struck by ball / or other sports equipment <input type="checkbox"/> jumping <input type="checkbox"/> during a fall <input type="checkbox"/> swerving / pivoting <input type="checkbox"/> overuse <input type="checkbox"/> whilst running <input type="checkbox"/> other <p>_____</p> <p>_____</p> <p>Explain exactly how the injury occurred</p> <p>_____</p> <p>_____</p> <p>_____</p> <p>_____</p> <p>_____</p> <p>Nature of Injury (tick all applicable boxes)</p> <ul style="list-style-type: none"> <input type="checkbox"/> Fracture (including suspected) <input type="checkbox"/> Stress fracture / reaction <input type="checkbox"/> Ligament sprain / tear <input type="checkbox"/> Muscle strain / tear <input type="checkbox"/> Tendonitis / inflammation <input type="checkbox"/> Spinal or suspected spinal injury <input type="checkbox"/> Dislocation / subluxation <input type="checkbox"/> Cut / Laceration <input type="checkbox"/> Muscle 'cork' <input type="checkbox"/> Thermal related (heat stress) <input type="checkbox"/> <p>Other _____</p> <p>_____</p> <ul style="list-style-type: none"> <input type="checkbox"/> Not known <p>Result of the injury</p> <ul style="list-style-type: none"> <input type="checkbox"/> had to stop playing / training immediately <input type="checkbox"/> continued playing / training but gradually worsened until had to stop playing / training
---	---

<hr/> <hr/>	<input type="checkbox"/> after immediate treatment went back to playing <input type="checkbox"/> continued playing / training until end of session with some discomfort <input type="checkbox"/> continued playing / training until end of session with minimal discomfort
-------------	--

Injury Treatment Required (tick all applicable boxes)

- Self-treated
- Massage therapy
- Physiotherapy / rehabilitation exercises
- Anti-inflammatory medication
- Surgery / Operation
- Injection (eg. quarterzone or other)
- other (please specify)

Resuming Activity Assessment

- minor (return to activity after treatment)
- mild (1-7 days modified activity)
- moderate (8-21 days modified activity)
- severe (> 21 days modified or lost)

Protective Equipment

Was protective equipment or taping used on the injured body part?

Yes No

If Yes, what type e.g. ankle brace, taping , helmet

Other medical conditions

Do you suffer from any long term medical conditions eg epilepsy, asthma, diabetes, arthritis, haemophilia (please list)

Have you been diagnosed with any growth-related musculoskeletal condition eg Osgood Schlatter disease (knee), Sever's Disease (heel) Little Leaguer's elbow, Perthes Disease (hip) (Please List)

Do you take any regular medications or supplements?
If YES please list
Name of medication or supplement

4. Further Information

- Have you participated in any sporting activity over the last 12 months that caused you to have any subsequent muscle soreness over the period of a week (circle)?

Yes No

If so, what was the activity?

Is there any other reason that you know of that would prevent you from participating in this project (circle)?

Yes No

If yes, please specify:

- In an *average* day, do you drink the following (circle)?

Coffee Yes No

How many? _____

Tea Yes No

How many? _____

Energy drinks Yes No

How many? _____

Chocolate drinks Yes No

How many? _____

- Are you taking any supplements (circle)?

Yes

No

If yes, please specify and explain:

I believe the information I have provided to be true and correct:

Signed:

Date:

N.B. The following sections will be completed by the investigator.

Health Screening Form – Part B

Testing commenced at:

	am/pm
--	-------

Anthropometric Data

Weight (kg)		
Height (mm)		
Inter-ASIS (mm)		
	LEFT (mm)	RIGHT (mm)
Leg Length		
ASIS-greater troch		
Knee Width		
Ankle Width		
Sole Thickness		

Limb preference:

- | | | |
|--------------------------------------|---|---|
| • What hand do you write with? | L | R |
| • What hand do you throw with? | L | R |
| • What foot do you kick a ball with? | L | R |

- What foot would you use to crush a can? L R
- What foot would you use to hop on? L R
- What foot do you use to lead when walking? L R

Appendix E: Calculation of vertical displacement

To calculate K_{vert} the vertical displacement of the COM of the body must also be calculated. In this case, COM displacement or ΔL can be calculated in two methods – indirectly via the double integration of the vertical force (F_z) (Cavagna, 1985) or directly via kinematic analysis. One limitation of the integration method is that the displacement of the COM is assumed to be the same at ground contact as it is at toe/foot off. This method must therefore be approached with caution. Below is the double integration method:

$$F_z = ma$$

Therefore:

$$a = \frac{F_z}{m}$$

where F_z is the vertical component of ground reaction force, m is body mass and a is acceleration. The integration of a , with respect to time (t), gives velocity (v).

$$v = a \cdot dt$$

The vertical velocity is then integrated again with respect to time to obtain vertical displacement.

$$L = v \cdot dt$$

Therefore, the double integration of vertical acceleration of the COM with respect to time gives maximal vertical displacement of the leg spring (ΔL).

$$\Delta L = \frac{F_z - \text{body mass}}{m} . dt$$

This method has been shown to be valid (Cavagna, 1985).

Appendix F: Dalleau and colleagues (2004) method of calculating K_{vert}

To calculate K_{vert} without the use of force platforms, a methodology was developed to measure and assess MSS whilst in the field. This method requires measuring contact and flight times during hopping and MSS is calculated by modelling ground reaction force as a sine wave (Dalleau, et al., 2004),

$$F(t) = F_{\text{peak}} \times \sin \frac{\pi}{t_c} \times t$$

where F_{peak} is peak ground reaction force, t_c is contact time and t is the half period of the sine wave. Assuming that the area under the force-time curve is equal to the impulse of the ground reaction force, F_{peak} is calculated by:

$$F_{\text{peak}} = mg \times \frac{\pi}{2} \times \frac{T_f}{T_c} + 1$$

where m is body mass, g is gravity, T_f is flight time and T_c is ground contact time. From here, vertical displacement (ΔL) can be calculated from knowing that the vertical velocity (v) of the body is zero at the middle of contact (or stance phase) shown by:

$$v t = - \frac{F_{\text{peak}} T_c}{m \pi} \times \cos \frac{\pi}{T_c} \times t - gt + g \frac{T_c}{2}$$

By integrating the above expression of vertical velocity, knowing that vertical displacement is zero at touchdown or foot contact with the ground, the equation for vertical displacement (z) is as follows:

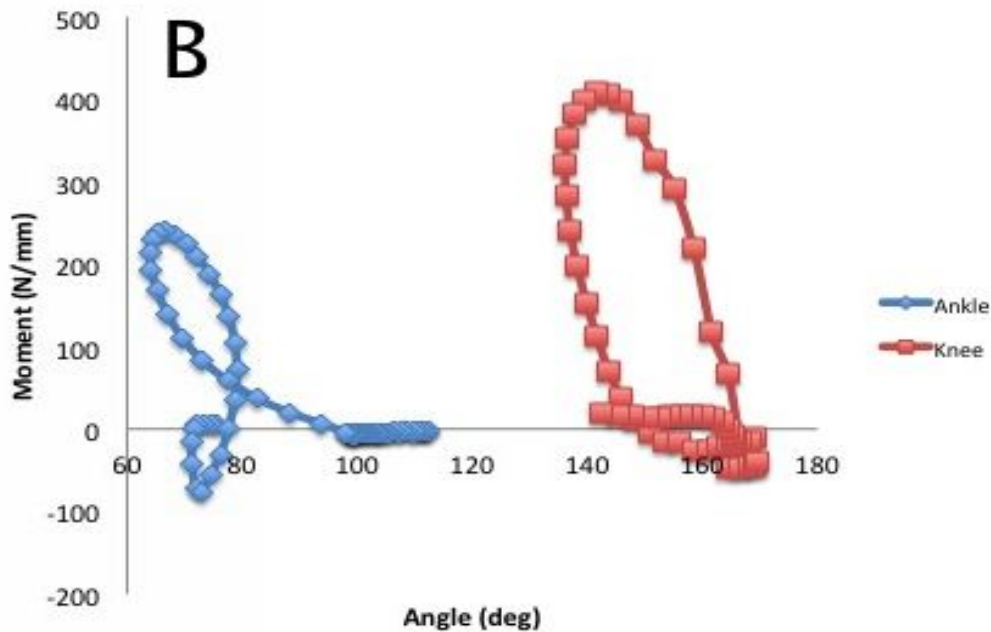
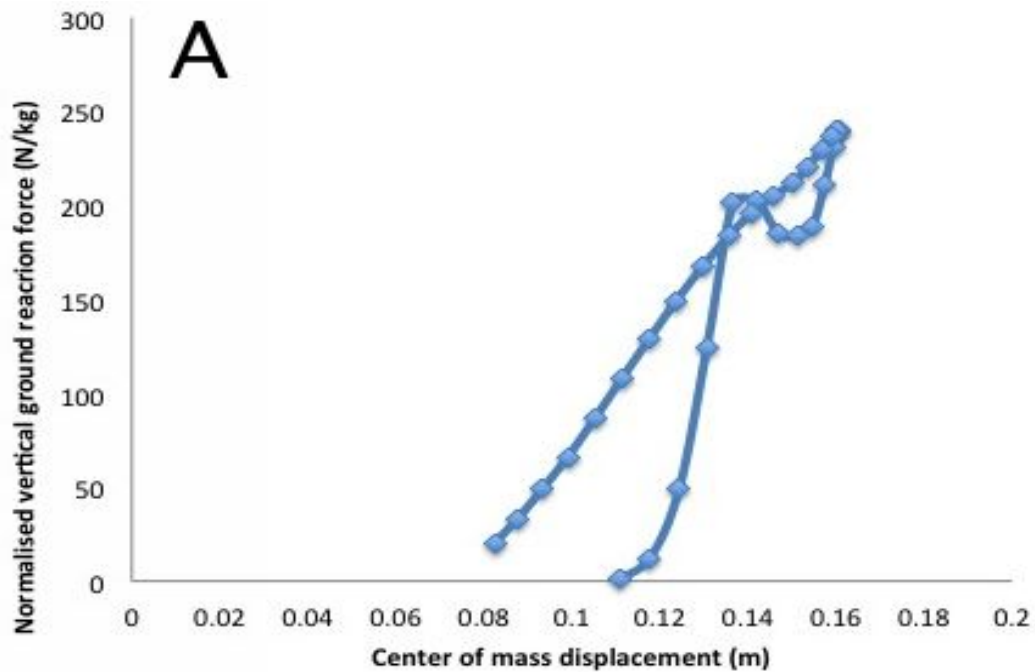
$$z(t) = -\frac{F_{peak}}{m} \frac{T_c^2}{\pi^2} \times \sin\left(\frac{\pi}{T_c} \times t\right) - \frac{1}{2} g \times t^2 + g \frac{T_c}{2} t$$

Therefore, knowing that k_{vert} is a ratio of force and displacement, the final equation is:

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

where m is body mass, T_c is ground contact time and T_f is flight time.

**Appendix G: Exemplars of raw data used to calculate vertical and joint
MSS**



Exemplar x-y graph used to calculate vertical stiffness (A), and knee and ankle joint stiffness (B). The ratio of ground reaction force and centre of mass displacement is used to calculate vertical stiffness. The ratio of moment and angle was used to calculate joint stiffness. MSS (leg, vertical & joint) is

calculated only during the braking phase of the movement (touchdown to peak knee flexion or touchdown to minimum centre of mass displacement).

REFERENCES

- Abendroth-Smith, J. (1996). Stride adjustments during a running approach toward a force plate. *Research Quarterly for Exercise and Sport*, 61(1), 97-101.
- ABS. (2006). *Physical Activity in Australia: A Snapshot, 2004 -05*. Unpublished manuscript, Canberra, Australian Capital Territory.
- Akimoto, T., Furudate, M., Saitoh, M., Sugiura, K., Waku, T., Akama, T., et al. (2002). Increased plasma concentrations of intercellular adhesion molecule-1 after strenuous exercise associated with muscle damage. *European Journal of Applied Physiology*, 86, 185-190.
- Alexander, R., & Bennet-Clark, H. (1977). Storage of elastic strain energy in muscle and other tissues. *Nature*, 265, 114-117.
- Allen, D. (2001). Eccentric muscle damage: mechanisms of early reduction of force. *Acta Physiologica Scandinavica*, 171, 311-319.
- Allison, G., Weston, R., Shaw, R., Longhurst, J., James, L., Kyle, K., et al. (1998). The reliability of Quadriceps muscle stiffness in individuals with osgood-schlatter disease. *Journal of Sports Rehabilitation*, 7, 258-266.
- Arampatzis, A., Bruggemann, G., & Klapsing, G. (2001). Leg stiffness and mechanical energetic processes during jumping on a sprung surface. *Medicine and Science in Sports and Exercise*, 33(6), 923-931.
- Arampatzis, A., Bruggemann, G., & Metzler, V. (1999). The effect of speed on leg stiffness and joint kinetics in human running. *Journal of Biomechanics*, 32, 1349-1353.
- Arampatzis, A., Karamanidis, K., & Albracht, K. (2007). Adaptational responses of the human Achilles tendon by modulation of the applied cyclic strain magnitude. *Journal of Experimental Biology*, 210(15), 2743-2753.
- Arampatzis, A., Schade, F., Walsh, M., & Bruggemann, G. (2001). Influence of leg stiffness and its effect on myodynamic jumping performance. *Journal of Electromyography and Kinesiology*, 11, 355-364.
- Arampatzis, A., Stafilidis, S., DeMonte, G., Karamanidis, K., Morey-Klapsing, G., & Bruggemann, G. (2005). Strain and elongation of the human gastrocnemius tendon and aponeurosis during maximal plantarflexion effort. *Journal of Biomechanics*, 38(4), 833-841.
- Armstrong, R. B., Ogilvie, R. W., & Schwane, J. A. (1983). Eccentric exercise-induced injury to rat skeletal muscle. *Journal of Applied Physiology*, 54(1), 80-93.
- Armstrong, R. B., Warren, G. L., & Warren, J. A. (1991). Mechanisms of exercise-induced muscle fibre injury. *Sports Medicine*, 12(3), 184-207.
- Arya, S., & Kulig, K. (2010). Tendinopathy alters mechanical and material properties of the Achilles tendon. *Journal of Applied Physiology*, 108(3), 670-675.
- Ashby, M. F., Gibson, L. J., Wegst, U., & Olive, R. (1995). The Mechanical Properties of Natural Materials. I. Material Property Charts. *Proceedings of the Royal Society of London. Series A: Mathematical and Physical Sciences*, 450(1938), 123-140.
- Asmussen, E. (1953). Positive and Negative Muscular Work. *Acta Physiologica Scandinavica*, 28(4), 364-382.

- Atkinson, G., & Nevill, A. (1998). Statistical methods for assessing measurement error (reliability) in variables relevant to sports medicine. *Sports Medicine*, 26(4), 217-238.
- Avagadro, P., Chaux, C., Bourdin, M., Dalleau, G., & Belli, A. (2004). The use of treadmill ergometers for extensive calculation of external work and leg stiffness during running. *European Journal of Applied Physiology*, 92, 182-185.
- Avela, J., Kyrolainen, H., Komi, P., & Rama, D. (1999a). Reduced reflex sensitivity persists several days after long-lasting stretch-shortening cycle exercise. *Journal of Applied Physiology*, 96, 1292-1300.
- Avela, J., Kyrolainen, H., Komi, P. V., & Rama, D. (1999b). Reduced reflex sensitivity persists several days after long-lasting stretch-shortening cycle exercise. *Journal of Applied Physiology*, 86(4), 1292-1300.
- Babul, S., Rhodes, E. C., Taunton, J. E., & Lepawsky, M. (2003). Effects of Intermittent Exposure to Hyperbaric Oxygen for the Treatment of an Acute Soft Tissue Injury. *Clinical Journal of Sport Medicine*, 13(3), 138-147.
- Bahr, R., & Bahr, I. A. (1997). Incidence of acute volleyball injuries: a prospective cohort study of injury mechanisms and risk factors. *Scandinavian Journal of Medicine & Science in Sports*, 7(3), 166-171.
- Bahr, R., & Krosshaug, T. (2005). Understanding injury mechanisms: a key component of preventing injuries in sport. *British Journal of Sports Medicine*, 39(6), 324-329.
- Barber-Westin, S. D., Noyles, F. R., & Galloway, M. (2006). Jump-land characteristics and muscle strength development in young athletes: a gender comparison of 1140 athletes 9 to 17 years of age. *The American Journal of Sports Medicine*, 34(3), 375-385.
- Bartlett, R. (Ed.). (1999). *Sports Biomechanics. Reducing Injury and Improving Performance*. New York: E & FN Spon.
- Bennell, K., Crossley, K., Jayarajan, J., Walton, E., Warden, S., Kiss, Z. D., et al. (2004). Ground reaction forces and bone parameters in females with tibial stress fracture. *Medicine & Science in Sports & Exercise*, 36(3), 397-404.
- Bennett, M. B., Ker, R. F., Imery, N. J., & Alexander, R. M. (1986). Mechanical properties of various mammalian tendons. *Journal of Zoology*, 209(4), 537-548.
- Birch, H. L. (2007). Tendon matrix composition and turnover in relation to functional requirements. *International Journal of Experimental Pathology*, 88(4), 241-248.
- Bisseling, R., Hof, A., Bredeweg, S., Zwerver, J., & Mulder, T. (2007). Relationship between landing strategy and patellar tendinopathy in volleyball. *British Journal of Sports Medicine*, 41(7), e8.
- Bland, J. M., & Altman, D. G. (1999). Measuring agreement in method comparison studies. *Statistical Methods in Medical Research*, 8(2), 135-160.
- Blickhan, R. (1989). The spring-mass model for running and hopping. *Journal of Biomechanics*, 22(11-12), 1217-1227.
- Bloomer, R. J., Goldfarb, A. H., McKenzie, M. J., You, T., & Nguyen, L. (2004). Effects of antioxidant therapy in women exposed to eccentric

- exercise. *International Journal of Sport Nutrition and Exercise Metabolism*, 14(4), 377-388.
- Bobbert, M. (2001). Dependence of human squat jump performance on the series elastic compliance of the triceps surae: a simulation study. *Journal of Experimental Biology*, 204(3), 533-542.
- Bradshaw, E., Hume, P., Calton, M., & Aisbett, B. (2010). Reliability and variability of day-to-day vault training measures in artistic gymnastics. *Sports Biomechanics*, 9(2), 79 - 97.
- Bradshaw, E. J., Le Rossignol, P., Williams, M., & Lorenzen, C. (2006). Novel insights on lower limb musculoskeletal health and performance in pre-adolescent and adolescent gymnasts. *International Society of Biomechanics Symposium, XXIV*, 413-417.
- Brancaccio, P., Maffulli, N., & Limongelli, F. (2007). Creatine kinase monitoring in sport medicine. *British Medical Bulletin*, 1-22.
- Braun, S., Millett, P. J., Yongpravat, C., Pault, J. D., Anstett, T., Torry, M. R., et al. (2010). Biomechanical Evaluation of Shear Force Vectors Leading to Injury of the Biceps Reflection Pulley. *The American Journal of Sports Medicine*, 38(5), 1015-1024.
- Braun, W., & Dutto, D. J. (2003). The effects of a single bout of downhill running and ensuing delayed onset of muscle soreness on running economy performed 48 h later. *European Journal of Applied Physiology*, 90(1), 29-34.
- Brooks, J., & Fuller, C. (2006). The Influence of Methodological Issues on the Results and Conclusions from Epidemiological Studies of Sports Injuries: Illustrative Examples. *Sports Medicine*, 36(6), 459-472.
- Brooks, S., Zerba, E., & Faulkner, J. (1995). Injury to muscle fibres after single stretches of passive and maximally stimulated muscles in mice. *Journal of Physiology*, 488(2), 459-469.
- Brughelli, M., & Cronin, J. (2008a). Influence of running velocity on vertical, leg and joint stiffness: Modelling and recommendations for future research. *Sports Medicine*, 38(8), 647-657.
- Brughelli, M., & Cronin, J. (2008b). A review of research on the mechanical stiffness in running and jumping: methodology and implications. *Scandinavian Journal of Medicine and Science in Sports*, 18, 417-426.
- Brukner, P., & Kahn, K. (Eds.). (2007). *Clinical Sports Medicine* (three ed.). New South Wales: McGraw-Hill.
- Bryant, A., Clark, R., Bartold, S., Murphy, A., Bennell, K., Hohmann, E., et al. (2008). Effects of estrogen on the mechanical behaviour of the human Achilles tendon *in vivo*. *Journal of Applied Physiology*, 105(4), 1035-1043.
- Burr, D. (2011). Why bones bend but don't break. *Journal of Musculoskeletal and Neuronal Interactions*, 11(4), 270-285.
- Butler, R. J., Crowell, H. P., & McClay-Davis, I. (2003). Lower extremity stiffness: implications for performance and injury. *Clinical Biomechanics*, 18, 511-517.
- Butler, R. J., Grood, E., & Noyes, F. R. (1978). Biomechanics of ligaments and tendons. *Exercise and sport sciences reviews*, 6, 125-181.

- Byrne, C., & Eston, R. (2002a). The effect of exercise-induced muscle damage on isometric and dynamic knee extensor strength and vertical jump performance. *Journal of Sports Sciences*, 20(5), 417-425.
- Byrne, C., & Eston, R. (2002b). Maximal-intensity isometric and dynamic exercise performance after eccentric muscle actions. *Journal of Sports Sciences*, 20, 951-959.
- Byrne, C., Eston, R. G., & Edwards, R. H. T. (2001). Characteristics of isometric and dynamic strength loss following eccentric exercise-induced muscle damage. *Scandinavian Journal of Medicine & Science in Sports*, 11(3), 134-140.
- Byrne, C., Twist, C., & Eston, R. (2004). Neuromuscular function after exercise induced muscle damage. Theoretical and applied implications. *Sports Medicine*, 34(1), 49-69.
- Byrnes, W. C., Clarkson, P. M., White, J. S., Hsieh, S. S., Frykman, P. N., & Maughan, R. J. (1985). Delayed onset muscle soreness following repeated bouts of downhill running. *Journal of Applied Physiology*, 59(3), 710-715.
- Cavagna, G. A. (1977). Storage and Utilization of Elastic Energy in Skeletal Muscle. *Exercise and Sport Sciences Reviews*, 5(1), 89-130.
- Cavagna, G. A. (1985). Force platforms as ergometers. *Journal of Applied Physiology*, 39(1), 174-179.
- Cavagna, G. A. (2006). The landing-take-off asymmetry in human running. *Journal of Experimental Biology*, 209(20), 4051-4060.
- Cavagna, G. A., Franzetti, N. C., Heglund, N. C., & Willems, P. (1988). The determinants of step frequency in running, trotting and hopping in man and other vertebrates. *Journal of Physiology*, 399, 81-92.
- Cavagna, G. A., Heglund, N., & Willems, P. (2005). Effect of an increase in gravity on the power output and the rebound of the body in human running *Journal of Experimental Biology*, 208, 2333-2346.
- Cavanagh, P., & LaFortune, M. (1980). Ground reaction forces in distance running. *Journal of Biomechanics*, 13(5), 397-406.
- Chan, K., & Hsu, C. (1993). *Cartilage and ligament injuries in sports injuries*. London, Oxford: University Press.
- Chandrashekar, N., Slauterbeck, J., & Hashemi, J. (2011). Effects of cyclic loading on the tensile properties of human patellar tendon. *The Knee*.
- Cheung, K., Hume, P. A., & Maxwell, L. (2003). Delayed onset muscle soreness. Treatment strategies and performance factors. *Sports Medicine*, 33(2), 145-164.
- Child, R., Saxton, J., & Donnelly, A. (1998). Comparison of eccentric knee extensor muscle actions at two muscle lengths on indices of damage and angle-specific force production in humans. *Journal of Sports Sciences*, 16, 301-308.
- Child, S., Bryant, A. L., Clark, R. A., & Crossley, K. M. (2010). Mechanical Properties of the Achilles Tendon Aponeurosis Are Altered in Athletes With Achilles Tendinopathy. *The American Journal of Sports Medicine*, 38(9), 1885-1893.
- Clark, R., Crossley, K., Buckley, L., Bartold, S., & Bryant, A. (2007). Reliability of an In Vivo measure of Achilles Tendon strain. *Journal of Science and Medicine in Sport*, 10, 78.

- Clarkson, P. (2006). Case Report of Exertional Rhabdomyolysis in a 12-Year-Old Boy. *Medicine and Science in Sports and Exercise* 38(2), 197-200.
- Clarkson, P., & Hubal, M. (2002). Exercise-induced muscle damage in humans. *American Journal of Physical Medicine and Rehabilitation*, 81(Suppl), s52-s69.
- Clarkson, P., Kearns, A., Rouzier, P., Richard, R., & Thompson, P. (2006). Serum creatine kinase levels and renal function measures in exertional muscle damage. *Medicine and Science in Sports and Exercise*, 38(4), 623-627.
- Clarkson, P. M., Nosaka, K., & Braun, B. (1992). Muscle function after exercise-induced muscle damage and rapid adaptation. *Medicine and Science in Sports and Exercise*, 24(5), 512-520.
- Cleak, M., & Eston, R. (1992). Muscle soreness, swelling, stiffness and strength loss after intense eccentric exercise. *British Journal of Sports Medicine*, 26(4), 267-272.
- Cleather, D., & Guthrie, S. (2007). Quantifying delayed-onset muscle soreness: A comparison of unidimensional and multidimensional instrumentation. *Journal of Sports Sciences*, 25(8), 845-850.
- Close, G., Ashton, T., McArdle, A., & MacLaren, D. (2005). The emerging role of free radicals in delayed onset muscle soreness and contraction-induced muscle injury. *Comparative Biochemistry and Physiology, Part A* 142, 257-266.
- Coren, S. (1993). The lateral preference inventory for measurement of handedness, footedness, eyedness, and earedness: norms for young adults. . *Bulletin of the Psychonomic Society*, 31(1), 1-3.
- Coupe, C., Kongsgaard, M., Aagaard, P., Hansen, P., Bojsen-Moller, J., Kjaer, M., et al. (2008). Habitual loading results in tendon hypertrophy and increased stiffness of the human patellar tendon. *Journal of Applied Physiology*, 105(3), 805-810.
- Cummins, P., Young, A., Auckland, M. L., Michie, C. A., Stone, P. C. W., & Shepstone, B. J. (1987). Comparison of serum cardiac specific troponin-I with creatine kinase, creatine kinase-MB isoenzyme, tropomyosin, myoglobin and C-reactive protein release in marathon runners: cardiac or skeletal muscle trauma? *European Journal of Clinical Investigation*, 17(4), 317-324.
- Dalleau, G., Belli, A. V., F., Lacour, J. R., & Bourdin, M. (2004). A simple method for field measurements of leg stiffness in hopping. *International Journal of Sports Medicine*, 25, 170-176.
- Derrick, T. R., Hamill, J., & Caldwell, G. E. (1998). Energy absorption of impacts during running at various stride lengths. *Medicine & Science in Sports & Exercise*, 30(1), 128-135.
- Deschenes, M., Brewer, R., Bush, J., McCoy, R., Volek, J., & Kraemer, W. (2000). Neuromuscular disturbance outlasts other symptoms of exercise-induced muscle damage. *Journal of the Neurological Sciences* 174(2), 92-99.
- Divert, C., Baur, H., Mornieux, G., Mayer, F., & Belli, A. (2005). Stiffness adaptations in shod running. *Journal of Applied Biomechanics*, 21, 311-321.

- Duarte, J., Magalhaes, J., Monteiro, L., Almeida-Dias, A., Soares, J., & Appell, H. (1999). Exercise-induced signs of muscle overuse in children. *International Journal of Sports Medicine*, *20*, 103-108.
- Duclay, J., Martin, A., Duclay, A., Cometti, G., & Pousson, M. (2009). Behavior of fascicles and the myotendinous junction of human medial gastrocnemius following eccentric strength training. *Muscle & Nerve*, *39*(6), 819-827.
- Dutto, D. J., & Braun, W. (2004). DOMS-associated changes in ankle and knee joint dynamics during running. *Medicine and Science in Sports and Exercise*, *36*(4), 560-566.
- Dutto, D. J., & Smith, G. A. (2002). Changes in spring-mass characteristics during treadmill running to exhaustion. *Medicine and Science in Sports and Exercise*, *34*(8), 1324-1331
- Dyhre-Poulsen, P., Simonsen, E., & Voigt, M. (1991). Dynamic control of muscle stiffness and H reflex modulation during hopping and jumping in man. *Journal of Physiology*, *437*, 287-304.
- Ebbeling, C., & Clarkson, P. (1989). Exercise-induced muscle damage and adaptation. *Sports Medicine*, *7*(4), 207-234.
- Emery, C. A., Hagel, B., Decloe, M., & Carly, M. (2010). Risk factors for injury and severe injury in youth ice hockey: a systematic review of the literature. *Injury Prevention*, *16*(2), 113-118.
- Enoka, R. (1996). Eccentric contractions require unique activation strategies by the nervous system. *Journal of Applied Physiology*, *81*(6), 2339-2346.
- Etheridge, T., Philp, A., & Watt, P. (2008). A single protein meal increases recovery of muscle function following an acute eccentric exercise bout. *Applied Physiology, Nutrition, and Metabolism*, *33*, 483-488.
- Ettema, G. J. (1996). Mechanical efficiency and efficiency of storage and release of series elastic energy in skeletal muscle during stretch-shorten cycles. *Journal of Experimental Biology*, *199*(9), 1983-1997.
- Evans, R., Knight, K., Draper, D., & Parcell, A. (2002). Effects of warm-up before eccentric exercise on indirect markers of muscle damage. *Medicine & Science in Sports & Exercise*, *34*(12), 1892-1899.
- Ewers, B., Jayaraman, V., Banglmaier, R., & Haut, R. (2000). The effect of loading rate on the degree of acute injury and chronic conditions in the knee after blunt impact. *Stapp Car Crash Journal*, *44*, 299-313.
- Farley, C., Blickhan, R., Saito, J., & Taylor, C. (1991). Hopping frequency in humans: a test of how springs set stride frequency in bouncing gaits. *Journal of Applied Physiology*, *71*(6), 2172-2132.
- Farley, C., Glasheen, J., & McMahon, T. A. (1993). Running springs: speed and animal size. *Journal of Experimental Biology*, *185*, 71-86.
- Farley, C., & Gonzalez, O. (1996). Leg stiffness and stride frequency in human running. *Journal of Biomechanics*, *29*(2), 181-186.
- Farley, C., Houdijk, H., Van Strien, C., & Louie, M. (1998). Mechanism of leg stiffness adjustment for hopping on surfaces of different stiffnesses. *Journal of Applied Physiology*, *85*(3), 1044-1055.
- Farley, C., & Morgenroth, D. (1999). Leg stiffness primarily depends on ankle stiffness during human hopping. *Journal of Biomechanics*, *32*, 267-273.

- Faul, F., Erdfelder, E., Buchner, A., & Lang, A.-G. (2009). Statistical power analyses using G*Power 3.1: Tests for correlation and regression analyses. *Behavior Research Methods* 41, 1149-1160.
- Feeley, B. T., Powell, J. W., Muller, M. S., Barnes, R. P., Warren, R. F., & Kelly, B. T. (2008). Hip Injuries and Labral Tears in the National Football League. *The American Journal of Sports Medicine*, 36(11), 2187-2195.
- Ferber, R., McClay Davis, I., Williams, D., & Laughton, C. (2002). A comparison of within- and between-day reliability of discrete 3D lower extremity variables in runners. *Journal of Orthopaedic Research*, 20(6), 1139-1145.
- Ferris, D. P., & Farley, C. T. (1997). Interaction of leg stiffness and surface stiffness during human hopping. *Journal of Applied Physiology*, 82(1), 15-22.
- Finch, C., & Orchard, J. (2002). Australia needs to follow New Zealand's lead on sports injuries. *Medical Journal of Australia*, 177, 38-39.
- Flood, L., & Harrison, J. (2009). Epidemiology of basketball and netball injuries that resulted in hospital admission in Australia, 2000-2004. *Medical Journal of Australia*, 190(19), 87-90.
- Forestier, N., Teasdale, N., & Nougier, V. (2002). Alteration of the position sense at the ankle induced by muscular fatigue in humans. *Medicine and Science in Sports and Exercise*, 34(1), 117-122.
- Foure, A., Nordez, A., & Cornu, C. (2010). Plyometric training effects on Achilles tendon stiffness and dissipative properties. *Journal of Applied Physiology*, 109(3), 849-854.
- Friden, J., & Lieber, R. (1992). Structural and mechanical basis of exercise-induced muscle injury. *Medicine & Science in Sports & Exercise*, 24(5), 521-530.
- Friden, J., Sfakianos, P., Hargens, A., & Akeson, W. (1988). Residual muscular swelling after repetitive eccentric contractions. *Journal of Orthopaedic Research*, 6, 493-498.
- Fukashiro, S., Rob, M., Ichinose, Y., Kawakami, Y., & Fukunaga, T. (1995). Ultrasonography gives directly but noninvasively elastic characteristic of human tendon in vivo. *European Journal of Applied Physiology and Occupational Physiology*, 71(6), 555-557.
- Fukunaga, T., Kawakami, Y., Kubo, K., & Kanehisa, H. (2002). Muscle and tendon interaction during human movements. *Exercise and Sports Science Reviews*, 30(3), 106-110.
- Fukunaga, T., Kubo, K., Kawakami, Y., Fukashiro, S., Kanehisa, H., & Maganaris, C. (2001). In vivo behaviour of human muscle tendon during walking. *Proceedings of the Royal Society of London. Series B: Biological Sciences*, 268(1464), 229-233.
- Fung, Y.-c. (1993). *Biomechanics: mechanical properties of living tissues*. USA: Springer.
- Gibilisco, S. (2002). *Physics demystified*. New York: McGraw Hill.
- Gleeson, M., Blannin, A., Walsh, N., Field, C., & Pritchard, J. (1998). Effect of exercise-induced muscle damage on the blood lactate response to incremental exercise in humans. *European Journal of Applied Physiology and Occupational Physiology*, 77(3), 292-295.

- Gleeson, M., Blannin, A., Zhu, B., Brooks, S., & Cave, R. (1995). Cardiorespiratory, hormonal and haematological responses to submaximal cycling performed 2 days after eccentric or concentric exercise bouts. *Journal of Sports Sciences*, 13(6), 471 - 479
- Gliottoni, R., Meyers, J., Arngrimsson, S., Broglio, S., & Motl, R. (2009). Effect of caffeine on quadriceps muscle pain during acute cycling exercise in low versus high caffeine consumers. *International Journal of Sports Nutrition and Exercise Metabolism*, 19(2), 150-161.
- Gluck, G. S., Bendo, J. A., & Spivak, J. M. (2008). The lumbar spine and low back pain in golf: a literature review of swing biomechanics and injury prevention. *The Spine Journal*, 8(5), 778-788.
- Goff, D., Hamill, J., & Clarkson, P. (1998). Biomechanical and biochemical changes after downhill running. *Medicine & Science in Sports & Exercise*, 30(5), Suppl. 101.
- Golden, C., & Dudley, G. (1992). Strength after bouts of eccentric or concentric actions. *Medicine & Science in Sports & Exercise*, 24(8), 926-933.
- Gollhofer, A., Strojnik, V., Rapp, W., & Schweizer, L. (1992). Behaviour of triceps surae muscle-tendon complex in different jump conditions. *European Journal of Applied Physiology and Occupational Physiology*, 64(4), 283-291.
- Gottlieb, G. L., Agarwal, G. C., & Jaeger, R. J. (1981). Response to sudden torques about ankle in man. IV. A functional role of alpha-gamma linkage. *Journal of Neurophysiology*, 46(1), 179-190.
- Granata, K., Padua, D., & Wilson, S. (2002). Gender differences in active musculoskeletal stiffness. Part II: quantification of leg stiffness during functional hopping tasks. *Journal of Electromyography and Kinesiology*, 12(2), 127-135
- Gregory, J., Morgan, D., Allen, T., & Proske, U. (2007). The shift in muscle's length-tension relation after exercise attributed to increased series compliance. *European Journal of Applied Physiology*, 99(4), 431-441.
- Grimston, S. K., Engsborg, J. R., Kloiber, R., & Hanley, D. A. (1991). Bone mass, external loads, and stress fracture in female runners. *International Journal of Sport Biomechanics*, 7(3), 293-302.
- Grimston, S. K., Nigg, B., Hanley, D. A., & Engsborg, J. R. (1993). Differences in ankle joint complex range of motion as a function of age. *Foot & Ankle*, 14(4), 215-222.
- Gulick, D., & Kimura, I. (1996). Delayed onset muscle soreness: What is it and how do we treat it? *Journal of Sport Rehabilitation*, 5, 234-243.
- Gunther, M., & Blickhan, R. (2002). Joint stiffness of the ankle and the knee in running. *Journal of Biomechanics*, 35, 1459-1474.
- Hamill, J., Derrick, T. R., & Holt, K. (1995). Shock attenuation and stride frequency during running. *Human Movement Science*, 14(1), 45-60.
- Hamill, J., Freedson, P., Clarkson, P., & Braun, B. (1991). Muscle Soreness During Running: Biomechanical and Physiological Considerations. *Journal of Applied Biomechanics*, 7(2), 125-137.
- Hamill, J., & Knutzen, K. (2009). *Biomechanical Basis of Human Movement* (Third ed.). Baltimore: Lippincott, Williams and Wilkins.

- Hansen, P., Aagaard, P., Kjaer, M., Larsson, B., & Magnusson, S. P. (2003). Effect of habitual running on human Achilles tendon load-deformation properties and cross-sectional area. *Journal of Applied Physiology*, *95*(6), 2375-2380.
- Haut, T., & Haut, R. (1997). The state of tissue hydration determines the strain-rate-sensitive stiffness of human patellar tendon. *Journal of Biomechanics*, *30*(1), 79-81.
- He, J., Kram, R., & McMahon, T. A. (1991). Mechanics of running under simulated low gravity. *Journal of Applied Physiology*, *71*(863-870).
- Heinemeier, K., & Kjaer, M. (2011). In vivo investigation of tendon responses to mechanical loading. *Journal of Musculoskeletal and Neuronal Interactions*, *11*(2), 115-123.
- Heise, G., & Martin, P. (1998). "Leg spring" characteristics and the aerobic demand of running. *Medicine and Science in Sports and Exercise*, *30*(5), 750-754.
- Herzog, W. (2000). *Force production in skeletal muscle*. Champaign, IL: Human Kinetics.
- Herzog, W., Leonard, T. R., Joumaa, V., & Mehta, A. (2008). Mysteries of Muscle Contraction. *Journal of Applied Biomechanics*, *24*(1), 1-13.
- Hewett, T., Stroupe, A., Nance, T., & Noyes, F. R. (1996). Plyometric Training in Female Athletes. *The American Journal of Sports Medicine*, *24*(6), 765-773.
- Hill, A. (1938). The heat of shortening and the dynamic constants of muscle. *Proceedings of the Royal Society B: Biological Sciences*, *128*(136-195).
- Hill, T. L. (1974). Theoretical formalism for the sliding filament model of contraction of striated muscle. Part I. *Progress in Biophysics and Molecular Biology*, *28*, 267.
- Hills, A. P., King, N. A., & Armstrong, T. P. (2007). The Contribution of Physical Activity and Sedentary Behaviours to the Growth and Development of Children and Adolescents: Implications for Overweight and Obesity. *Sports Medicine*, *37*(6), 533-545.
- Hobara, H., Inoue, K., Muraoka, T., Omuro, K., Sakamoto, M., & Kanosue, K. (2010). Leg stiffness adjustment for a range of hopping frequencies in humans. *Journal of Biomechanics*, *43*(3), 506-511.
- Hobara, H., Kanosue, K., & Suzuki, S. (2007). Changes in muscle activity with increase in leg stiffness during hopping. *Neuroscience Letters*, *418*(1), 55-59.
- Hobara, H., Kimura, K., Omuro, K., Gomi, K., Muraoka, T., Iso, S., et al. (2008). Determinants of difference in leg stiffness between endurance and power-trained athletes. *Journal of Biomechanics*, *41*, 506-514.
- Hopkins, W. (2000). Measures of reliability in sports medicine and science. *Sports Medicine*, *30*(1), 1-15.
- Horita, T., Komi, P., Nicol, C., & Kyrolainen, H. (1996). Stretch shortening cycle fatigue: interactions among joint stiffness, reflex, and muscle mechanical performance in the drop jump. *European Journal of Applied Physiology*, *73*, 393-403.
- Horita, T., Komi, P., Nicol, C., & Kyrolainen, H. (1999). Effect of exhausting stretch-shortening cycle exercise on the time course of mechanical

- behaviour in the drop jump: possible role of muscle damage. *European Journal of Applied Physiology*, 79(2), 160-167.
- Hreljac, A., Marshall, R. N., & Hume, P. A. (2000). Evaluation of lower extremity overuse injury potential in runners. *Medicine and Science in Sports and Exercise*, 32(9), 1635-1641.
- Humphrey, J. D. (2003). Review Paper: Continuum biomechanics of soft biological tissues. *Proceedings of the Royal Society of London. Series A: Mathematical, Physical and Engineering Sciences*, 459(2029), 3-46.
- Hunter, D., & Spriggs, J. (2000). Investigation into the relationship between the passive flexibility and active stiffness of ankle and plantar-flexor muscles. *Clinical Biomechanics*, 15, 600-606.
- Hunter, I., & Smith, G. (2007). Preferred and optimal stride frequency, stiffness and economy: changes with fatigue during a 1-h high-intensity run.
- Huxley, H. E. (1969). The Mechanism of Muscular Contraction. *Science*, 164(3886), 1356-1366.
- Ishikawa, M., Finni, T., & Komi, P. (2003). Behaviour of vastus lateralis muscle-tendon during high intensity SSC exercise in vivo. *Acta Physiological Scandinavia*, 178, 205-213.
- Ishikawa, M., & Komi, P. (2007). The role of the stretch reflex in the gastrocnemius muscle during human locomotion at various speeds. *Journal of Applied Physiology*, 12, 1030-1036.
- Ishikawa, M., & Komi, P. V. (2004). Effects of different dropping intensities on fascicle and tendinous tissue behavior during stretch-shortening cycle exercise. *Journal of Applied Physiology*, 96(3), 848-852.
- Ishikawa, M., Niemela, E., & Komi, P. V. (2005). Interaction between fascicle and tendinous tissues in short-contact stretch-shortening cycle exercise with varying eccentric intensities. *Journal of Applied Physiology*, 99(1), 217-223.
- Ito, M., Kawakami, Y., Ichinose, Y., Fukashiro, S., & Fukunaga, T. (1998). Nonisometric behavior of fascicles during isometric contractions of a human muscle. *Journal of Applied Physiology*, 85(4), 1230-1235.
- Jones, D., Newham, D., & Torgan, C. (1989). Mechanical influenced on long-lasting human muscle fatigue and delayed-onset pain. *Journal of Physiology*, 412, 415-427.
- Kellis, E., & Baltzopoulos, V. (1995). Isokinetic eccentric exercise. *Sports Medicine*, 19(3), 202-222.
- Ker, R., Alexander, R., & Bennett, M. (1988). Why are mammalian tendons so thick? *Journal of Zoology*, 216(2), 309-324.
- Kerdok, A. E., Biewener, A. A., McMahon, T. A., Weyand, P. G., & Herr, H. M. (2002). Energetics and mechanics of human running on surfaces of different stiffnesses. *Journal of Applied Physiology*, 92(2), 469-478.
- Kim, S., Akaike, T., Sasagaw, T., Atomi, Y., & Kurosawa, H. (2002). Gene expression of type I and type III collagen by mechanical stretch in anterior cruciate ligament cells. *Cell Structure and Function*, 27, 139 - 144.
- Kirkendall, D. T., & Garrett, W. E. (2000). The Anterior Cruciate Ligament Enigma: Injury Mechanisms and Prevention. *Clinical Orthopaedics and Related Research*, 372, 64-68.

- Kjær, M., Langberg, H., Heinemeier, K., Bayer, M., Hansen, M., Holm, L., et al. (2009). From mechanical loading to collagen synthesis, structural changes and function in human tendon. *Scandinavian Journal of Medicine and Science in Sports*, *19*, 500-510.
- Koh, J. O., Cassidy, J. D., & Watkinson, E. J. (2003). Incidence of concussion in contact sports: a systematic review of the evidence. *Brain Injury*, *17*(10), 901-917.
- Koh, T. (2008). Physiology and mechanisms of skeletal muscle damage. In P. Tiidus (Ed.), *Skeletal Muscle Damage and Repair*. Illinois: Human Kinetics.
- Koller, A., Mair, J., Schobersberger, W., Wohlfarter, T., Haid, C., Mayr, M., et al. (1998). Effects of prolonged strenuous endurance exercise on plasma myosin heavy chain fragments and other muscular proteins. Cycling vs running. *The Journal of Sports Medicine and Physical Fitness*, *38*(1), 10-17.
- Komi, P. (1984). Physiological and biomechanical correlates of muscle function: the effects of muscle structure and stretch-shortening cycle force and speed. *Exercise & Sports Science Review*, *12*, 81-121.
- Komi, P. (2000). Stretch-shortening cycle: a powerful model to study normal and fatigued muscle. *Journal of Biomechanics*, *33*(10), 1197-1206.
- Komi, P., Belli, A., Huttunen, V., Bonnefoy, R., Geysant, A., & Lacour, J. R. (1996). Optic fibre as a transducer of tendomuscular forces. *European Journal of Applied Physiology and Occupational Physiology*, *72*(3), 278-280.
- Komi, P., & Bosco, C. (1978). Utilization of stored elastic energy in leg extensor muscles by men and women. *Medicine and Science in Sports*, *10*(4), 261-265.
- Komi, P., Fukashiro, S., & Jarvinen, M. (1992). Biomechanical loading of Achilles tendon during normal locomotion. *Clinical Journal of Sport Medicine*, *11*, 521-531.
- Komi, P., Salonen, M., & Jarvinen, M. (1984). In Vivo-Measurements of Achilles Tendon Forces in Man. *Medicine & Science in Sports & Exercise*, *16*(2), 165.
- Komi, P. V., & Viitasalo, J. T. (1977). Changes in Motor Unit Activity and Metabolism in Human Skeletal Muscle during and after Repeated Eccentric and Concentric Contractions. *Acta Physiologica Scandinavica*, *100*(2), 246-254.
- Krans, J. (2010). The Sliding Filament Theory of Muscle Contraction. *Nature education*, *3*(9), 66.
- Kraushaar, B., & Nirschl, R. (1999). Tendinosis of the elbow (tennis elbow). Clinical features and findings of histological, immunohistochemical, and electron microscopy studies. *Journal of Bone and Joint Surgery. American Volume*, *82*(2), 259-278
- Krosshaug, T., Nakamae, A., Boden, B. P., Engebretsen, L., Smith, G., Slauterbeck, J. R., et al. (2007). Mechanisms of Anterior Cruciate Ligament Injury in Basketball. *The American Journal of Sports Medicine*, *35*(3), 359-367.
- Kubo, K., Akima, H., Ushiyama, J., Tabata, I., Fukuoka, H., Kanehisa, H., et al. (2004a). Effects of 20 days of bed rest on the viscoelastic properties

- of tendon structures in lower limb muscles. *British Journal of Sports Medicine*, 38, 324-330.
- Kubo, K., Akima, H., Ushiyama, J., Tabata, I., Fukuoka, H., Kanehisa, H., et al. (2004b). Effects of resistance training during bed rest on the viscoelastic properties of tendon structures in the lower limb. *Scandinavian Journal of Medicine and Science in Sports*, 14, 296-302.
- Kubo, K., Kanehisa, H., & Fukunaga, T. (2003). Gender differences in the viscoelastic properties of tendon structures. *European Journal of Applied Physiology*, 88, 520-526.
- Kubo, K., Kanehisa, H., & Fukunaga, T. (2005). Effects of viscoelastic properties of tendon structures on stretch-shortening cycle exercise *in vivo*. *Journal of Sports Sciences*, 23(8), 851-860.
- Kubo, K., Kanehisa, H., Ito, M., & Fukunaga, T. (2001). Effects of isometric training on the elasticity of human tendon structures *in vivo*. *Journal of Applied Physiology*, 91, 26-32.
- Kubo, K., Kanehisa, H., Kawakami, Y., & Fukunaga, T. (2001). Influences of repetitive muscle contractions with different modes on tendon elasticity *in vivo*. *Journal of Applied Physiology*, 91(1), 277-282.
- Kubo, K., Morimoto, M., Komuro, T., Yata, H., Tsunoda, N., Kanehisa, H., et al. (2007). Effects of plyometric and weight training on muscle-tendon complex and jump performance. *Medicine and Science in Sports and Exercise*, 39(10), 1801-1810.
- Kubo, K., Tabata, T., Yata, H., Tsunoda, N., & Kanehisa, H. (2010). Time course of changes in muscle and tendon properties during strength training and detraining. *The Journal of Strength & Conditioning Research*, 24(2), 322-331.
- Kuitunen, S., Komi, P. V., & Kyrolainen, H. (2002). Knee and ankle joint stiffness in sprint running. *Medicine and Science of Sport and Exercise*, 34(1), 166-173.
- Kuitunen, S., Kyrolainen, H., Avela, J., & Komi, P. V. (2007). Leg stiffness modulation during exhaustive stretch-shortening cycle exercise. *Scandinavian Journal of Medicine and Science in Sports*, 17, 67-75.
- Kuitunen, S. K., Avela, J. A., Kyrolainen, H., Nicol, C. N., & Komi, P. K. (2002). Acute and prolonged reduction in joint stiffness in humans after exhausting stretch-shortening cycle exercise. *European Journal of Applied Physiology*, 88(1), 107-116.
- Kurokawa, S., Fukunaga, T., & Fukashiro, S. (2001). Behaviour of fascicles and tendinous structures of human gastrocnemius during vertical jumping. *Journal of Applied Physiology*, 90, 1349-1358.
- Kurokawa, S., Fukunaga, T., Nagano, A., & Fukashiro, S. (2003). Interaction between fascicles and tendinous structures during counter movement jumping investigated *in vivo*. *Journal of Applied Physiology*, 95, 2306-2314.
- Kyrolainen, H., Takala, T., & Komi, P. (1998). Muscle damage induced by stretch-shortening cycle exercise. *Medicine and Science in Sports and Exercise* 30(3), 415-420.
- Laffaye, G., Bardy, B. G., & Durey, A. (2005). Leg stiffness and expertise in men jumping. *Medicine and Science in Sports and Exercise*, 37(4), 536-543.

- Lafortune, M. A., Hennig, E. M., & Lake, M. J. (1996). Dominant role of interface over knee angle for cushioning impact loading and regulating initial leg stiffness. *Journal of Biomechanics*, 29(12), 1523-1529.
- Lafortune, M. A., Lake, M. J., & Hennig, E. M. (1996). Differential shock transmission response of the human body to impact severity and lower limb posture. *Journal of Biomechanics*, 29(12), 1531-1537.
- Lambert, M., Marcus, P., Burges, T., & Noakes, T. (2002). Electro-membrane microcurrent therapy reduces signs and symptoms of muscle damage. *Medicine & Science in Sports & Exercise*, 34(4), 602-607.
- LaStayo, P., Woolf, J., Lewek, M., Snyder-Mackler, L., Reich, R., & Lindstedt, S. (2003). Eccentric muscle contractions: Their contribution to injury, prevention, rehabilitation and sport. *Journal of Orthopaedic and Sports Physical Therapy*, 33, 557-571.
- Latash, M. L., & Zatsiorsky, V. M. (1993). Joint stiffness: Myth or reality? *Human Movement Science*, 12, 653-692.
- Layne, J., & Nelson, M. (1999). The effects of progressive resistance training on bone density: a review. *Medicine & Science in Sports & Exercise*, 31(1), 25-30.
- Leucht, P., Fischer, K., Muhr, G., & Mueller, E. (2009). Epidemiology of traumatic spine fractures. *Injury*, 40(2), 166-172.
- Lichtwark, G., Bougoulas, K., & Wilson, A. (2007). Muscle fascicle and series elastic element length changes along the length of the human gastrocnemius during walking and running. *Journal of Biomechanics*, 40, 157-164.
- Lichtwark, G., & Wilson, A. (2005). In vivo mechanical properties of the human Achilles tendon during one-legged hopping. *The Journal of Experimental Biology*, 208, 4715-4725.
- Lichtwark, G., & Wilson, A. (2006). Interactions between the human gastrocnemius muscle and the Achilles tendon during incline, level, decline locomotion. *The Journal of Experimental Biology*, 209, 4379-4388.
- Lichtwark, G., & Wilson, A. (2007). Is Achilles tendon compliance optimised for maximum muscle efficiency during locomotion? *Journal of Biomechanics*, 40, 1768-1775.
- Lieber, R., & Bodine-Fowler, S. (1993). Skeletal muscle mechanics: implications for rehabilitation. *Physical Therapy*, 73(12), 844-856.
- Lieber, R., & Friden, J. (1993). Muscle damage is not a function of muscle force but active muscle strain. *Journal of Applied Physiology*, 74(2), 520-526.
- Lieber, R., & Friden, J. (2002). Morphologic and mechanical basis of delayed-onset muscle soreness. *Journal of the American Academy of Orthopaedic Surgeons*, 10, 67-73.
- Lindstedt, S., LaStayo, P., & Reich, T. (2001). When active muscles lengthen: Properties and consequences of eccentric contractions. *News in Physiological Sciences*, 16(6), 256-261.
- Lloyd, R., Oliver, J., Hughes, M., & Williams, C. (2009). Reliability and validity of field-based measures of leg stiffness and reactive strength index in youths. *Journal of Sports Sciences*, 27(14), 1565-1573.

- MacIntosh, B., Gardiner, P., & Mc Comas, A. (2006). *Skeletal muscle: Form and function*. Champaign, IL: Human Kinetics.
- MacIntyre, D. L., Reid, W. D., & McKenzie, D. C. (1995). Delayed muscle soreness. The inflammatory response to muscle injury and its clinical implications. *Sports Medicine*, 20(1), 24-40.
- Mademli, L., Arampatzis, A., & Walsh, M. (2006). Effect of muscle fatigue on the compliance of the gastrocnemius medialis tendon and aponeurosis. *Journal of Biomechanics*, 39(3), 426-434.
- Mademli, L., Arampatzis, A., & Walsh, M. (2008). Age-Related Effect of Static and Cyclic Loadings on the Strain-Force Curve of the Vastus Lateralis Tendon and Aponeurosis. *Journal of Biomechanical Engineering*, 130(1), 011007.
- Maffulli, N., & King, J. (1992). Effects of physical activity on some components of the skeletal system. *Sports Medicine* 13(6), 393-407.
- Maffulli, N., Wong, J., & Almekinders, L. C. (2003). Types and epidemiology of tendinopathy. *Clinics in Sports Medicine*, 22(4), 675-692.
- Maganaris, C. (2001). Force-length characteristics of in vivo human skeletal muscle. *Acta Physiologica Scandinavica*, 172(4), 279-285.
- Maganaris, C. (2005). Validity of procedures involved in ultrasound-based measurement of human plantarflexor tendon elongation on contraction. *Journal of Biomechanics*, 38, 9-13.
- Maganaris, C., Baltzopoulos, V., & Sargeant, A. (1998). Changes in Achilles tendon moment arm from rest to maximum isometric plantarflexion: in vivo observations in man. *Journal of Physiology*, 510(3), 977-985.
- Maganaris, C., Baltzopoulos, V., & Sargeant, A. (2006). Human calf muscle response during repeated isometric plantarflexions. *Journal of Biomechanics*, 39, 1249-1225.
- Maganaris, C., Narici, M., Almekinders, L., & Maffulli, N. (2004). Biomechanics and Pathophysiology of Overuse Tendon Injuries: Ideas on Insertional Tendinopathy. *Sports Medicine*, 34(14), 1005-1017.
- Maganaris, C., Narici, M., & Maffulli, N. (2008). Biomechanics of the Achilles tendon. *Disability and Rehabilitation*, 30(20-22), 1542-1547.
- Maganaris, C., & Paul, J. (1999). In vivo human tendon mechanical properties. *Journal of Physiology*, 521(1), 307-313.
- Maganaris, C., & Paul, J. (2002). Tensile properties of the in vivo human gastrocnemius tendon. *Journal of Biomechanics*, 35(12), 1639-1646.
- Magnusson, S., Aagaard, P., Rosager, S., Dyhre-Poulsen, P., & Kjaer, M. (2001). Load-displacement properties of human triceps surae aponeurosis in vivo. *Journal of Physiology*, 531(1), 277-288.
- Magnusson, S., Hansen, P., Aagaard, P., Brønd, J., Dyhre-Poulsen, P., Bojsen-Moller, J., et al. (2003). Differential strain patterns of the human gastrocnemius aponeurosis and free tendon, in vivo. *Acta Physiologica Scandinavica*, 177(2), 185-195.
- Magnusson, S., Narici, M., Maganaris, C., & Kjaer, M. (2008). Human tendon behaviour and adaptation, in vivo. *The Journal of Physiology* 586, 71-81.
- Mahieu, N., McNair, P. J., Cools, A., D'Haen, C., Vendermeulen, K., & Witvrouw, E. (2008). Effect of Eccentric Training on the Plantar Flexor

- Muscle-Tendon Tissue Properties. *Medicine & Science in Sports & Exercise*, 40(1), 117-123.
- Martini, F., & Nath, J. (2009). *Fundamentals of Anatomy and Physiology* (Eighth ed.). New York: Benjamin Cummings.
- McBride, T. A., Gorin, F. A., & Carlsen, R. C. (1995). Prolonged recovery and reduced adaptation in aged rat muscle following eccentric exercise. *Mechanisms of Ageing and Development*, 83(3), 185-200.
- McGinley, J. L., Goldie, P. A., Greenwood, K. M., & Olney, S. J. (2003). Accuracy and Reliability of Observational Gait Analysis Data: Judgments of Push-off in Gait After Stroke. *Physical Therapy*, 83(2), 146-160.
- McGinnis, P. (2005). *Biomechanics of sport and exercise* (Second ed.). USA: Human Kinetics.
- McLachlan, K., Murphy, A., Watsford, M., & Rees, S. (2006). The Interday Reliability of Leg and Ankle Musculotendinous Stiffness Measures. *Journal of Applied Biomechanics* 22, 296-304.
- McMahon, T. A. (1984). *Muscles, Reflexes and Locomotion*. Princeton, New Jersey: Princeton University Press.
- McMahon, T. A., & Cheng, G. C. (1990). The mechanics of running: how does stiffness couple with speed? *Journal of Biomechanics*, 23(Supplementary 1), 65-78.
- McMahon, T. A., Valiant, G., & Frederick, E. C. (1987). Groucho running. *Journal of Applied Physiology*, 62(6), 2326-2337.
- McNair, P. J., Prapavessis, H., & Callender, K. (2000). Decreasing landing forces: effect of instruction. *British Journal of Sports Medicine*, 34(293-296).
- Medibank (Producer). (2006, October 18th, 2007) Medibank Private Safe Sports Report. retrieved from http://www.medibank.com.au/Client/Documents/Pdfs/Medi_Safe_Sports_Report_2006.pdf
- Meinders, M., Gitter, A., & Czerniecki, J. M. (1998). The role of the ankle plantar flexor muscle work during walking. *Scandinavian Journal of Rehabilitation Medicine*, 30(1), 39-46.
- Messier, S. P., Davis, S. E., Curl, W. W., Lowery, R. B., & Pack, R. J. (1991). Etiologic factors associated with patellofemoral pain in runners. *Medicine and Science in Sports and Exercise*, 23(9), 1008-1015.
- Miller, B., Olesen, J., Hansen, M., Dossing, S., Cramer, R., Welling, R., et al. (2005). Coordinated collagen and muscle protein synthesis in human patella tendon and quadriceps muscle after exercise. *The Journal of Physiology*, 567(3), 1021-1033.
- Mishra, D., Friden, J., Schmitz, M., & Lieber, R. (1995). *Anti-inflammatory medication after muscle injury : a treatment resulting in short-term improvement but subsequent loss muscle function* (Vol. 77). Boston, MA, ETATS-UNIS: Journal of Bone and Joint Surgery Incorporated.
- Morgan, D., & Proske, U. (2004). Popping sarcomere hypothesis explains stretch-induced muscle damage. *Clinical and Experimental Pharmacology and Physiology*, 31, 541-545.

- Morin, J. B., Dalleau, G., Kyrolainen, H., Jeannin, T., & Belli, A. (2005). A simple method for measuring stiffness during running. *Journal of Applied Biomechanics*, *21*, 167-180.
- Morin, J. B., Jeannin, T., Chevallier, B., & Belli, A. (2006). Spring-mass model characteristics during spring running: correlation with performance and fatigue-induced changes. *International Journal of Sports Medicine*, *27*, 158-165.
- Muramatsu, T., Muraoka, T., Takeshita, D., Kawakami, Y., Hirano, Y., & Fukunaga, T. (2001). Mechanical properties of tendon and aponeurosis of human gastrocnemius muscle in vivo *Journal of Applied Physiology*, *90*(5), 1671-1678.
- Muraoka, T., Muramatsu, T., Fukunaga, T., & Kanehisa, H. (2005). Elastic properties of human Achilles tendon are correlated to muscle strength. *Journal of Applied Physiology*, *99*, 665-669.
- Murphy, A., Watsford, M., Coutts, A., & Pine, M. (2003). Reliability of a test of musculotendinous stiffness for the triceps-surae. *Physical Therapy in Sport*, *4*, 175-181.
- Nattiv, A., & Armsey Jr, T. (1997). Stress injury to bone in the female athlete. *Clinics in Sports Medicine*, *16*(2), 197-224.
- Neptune, R. R., Kautz, S. A., & Zajac, F. E. (2001). Contributions of the individual ankle plantar flexors to support, forward progression and swing initiation during walking. *Journal of Biomechanics*, *34*(11), 1387-1398.
- Newham, D., Jones, D., & Edwards, R. (1983). Large delayed plasma creatine kinase changes after stepping exercise. *Muscle & Nerve*, *6*(5), 380-385.
- Newham, D., Jones, D., Ghosh, G., & Aurora, P. (1988). Muscle fatigue and pain after eccentric contractions at long and short length. *Clinical Science*, *74*, 553-557.
- Newham, D., Jones, D., Tolfree, S., & Edwards, R. (1986). Skeletal muscle damage: a study of isotope uptake, enzyme efflux and pain after stepping. *European Journal of Applied Physiology and Occupational Physiology*, *55*(1), 106-112.
- Newham, D., McPhail, G., Mills, K., & Edwards, R. (1983). Ultrastructural changes after concentric and eccentric contractions of human muscle. *Journal of the Neurological Sciences*, *61*, 109-122.
- Newham, D., Mills, K., Quigley, B., & Edwards, R. (1983a). Muscle pain and fatigue after concentric and eccentric muscle contractions. *Clinical Science*, *64*, 55-62.
- Newham, D., Mills, K., Quigley, B., & Edwards, R. (1983b). Pain and fatigue after concentric and eccentric muscle contractions. *Clinical Science*, *64*, 55-62.
- Newton, M., Morgan, G., Sacco, P., Chapman, D., & Nosaka, K. (2008). Comparison of responses to strenuous eccentric exercise of the elbow flexors between resistance-trained and untrained men. *Journal of Strength and Conditioning*, *22*(2), 597-607.
- Nicol, C., Avela, J., & Komi, P. (2006). The stretch-shortening cycle. A model to study naturally occurring fatigue. *Sports Medicine*, *11*, 977-999.

- Nicol, C., Komi, P., & Marconnet, P. (1991a). Fatigue effects of marathon running on neuromuscular performance. *Scandinavian Journal of Medicine and Science in Sports*, 1, 10-17.
- Nicol, C., Komi, P., & Marconnet, P. (1991b). Fatigue effects of marathon running on neuromuscular performance. II. Changes in force, integrated electromyographic activity and endurance capacity. *Scandinavian Journal of Medicine and Science in Sports*, 1, 195-204.
- Nigg, B. (2000). *Mechanical effects of forces*. Champaign: Human Kinetics.
- Noakes, T., Kotzenberg, G., McArthur, P., & Dykman, J. (1983). Elevated serum creatine kinase MB and creatine kinase BB-isoenzyme fractions after ultra-marathon running. *European Journal of Applied Physiology and Occupational Physiology*, 52(1), 75-79.
- Nosaka, K. (2008). Muscle soreness and damage and the repeat-bout effect. In P. Tiidus (Ed.), *Skeletal Muscle Damage and Repair*. Illinois: Human Kinetics.
- Nosaka, K., & Clarkson, P. (1996). Changes in indicators of inflammation after eccentric exercise of the elbow flexors. *Medicine and Science in Sports & Exercise*, 28(8), 953-961.
- Nosaka, K., & Newton, M. (2002). Difference in the magnitude of muscle damage between maximal and submaximal eccentric loading. *Journal of Strength and Conditioning*, 16(2), 202-208.
- Nosaka, K., Newton, M., & Sacco, P. (2002). Delayed-onset muscle soreness does not reflect the magnitude of eccentric exercise-induced muscle damage. *Scandinavian Journal of Medicine and Science in Sports*, 12, 337-345.
- Nosaka, K. N., Sakamoto, K. S., Newton, M. N., & Sacco, P. S. (2001). The repeated bout effect of reduced-load eccentric exercise on elbow flexor muscle damage. *European Journal of Applied Physiology*, 85(1), 34-40.
- Nottle, C., & Nosaka, K. (2005a). The magnitude of muscle damage induced by downhill backward walking. *Journal of Science and Medicine in Sport*, 8(3), 264-273.
- Nottle, C., & Nosaka, K. (2005b). Repeated bout effect conferred by downhill backward walking. *Journal of Exercise Physiology*, 8(1), 1-10.
- Noyes, F. R., Lindenfeld, T. N., & Marshall, M. T. (1988). What determines an athletic injury (definition)? Who determines an injury (occurrence)? *American Journal of Sports Medicine*, 8(1 (15 supplement)), 65-68.
- O'Connor, P., & Cook, D. (1999). Exercise and Pain: The neurobiology, measurement, and laboratory study of pain in relation to exercise in humans. *Exercise and Sport Sciences Reviews*, 27(1), 119-166.
- Olmsted, L., Vela, L., Denegar, C., & Hertel, J. (2004). Prophylactic Ankle Taping and Bracing: A Numbers-Needed-to-Treat and Cost-Benefit Analysis. *Journal of Athletic Training*, 39(1), 95-100.
- Orchard, J. (2001). Intrinsic and Extrinsic Risk Factors for Muscle Strains in Australian Football. *The American Journal of Sports Medicine*, 29(3), 300-303.
- Orchard, J., Best, T., & Verrall, G. (2005). Return to play following muscle strain. *Clinical Journal of Sports Medicine*, 15, 436-411.

- Padua, D., Arnold, B. L., Perrin, D., Gansneder, B., Carcia, C. R., & Granata, K. (2006). Fatigue, Vertical Leg Stiffness, and Stiffness Control Strategies in Males and Females. *Journal of Athletic Training, 41*(3), 294-304.
- Padua, D., Carcia, C. R., Arnold, B. L., & Granata, K. P. (2005). Gender differences in leg stiffness and stiffness recruitment strategy during two-legged hopping. *Journal of Motor Behaviour, 37*(2), 111-125.
- Paschalis, V., Giakas, G., Baltzopoulos, V., Jamurtas, A., Theoharis, V., Kotzamanidis, C., et al. (2007). The effects of muscle damage following eccentric exercise on gait biomechanics. *Gait and Posture, 25*, 236-242.
- Paschalis, V., Koutedakis, Y., Baltzopoulos, V., Mougios, V., Jamurtas, A., & Giakas, G. (2005). Short vs. long length rectus femoris during eccentric exercise in relation to muscle damage in healthy males. *Clinical Biomechanics, 20*, 617-622.
- Peat, J., & Barton, B. (2005). *Medical statistics: A guide to data analysis and critical appraisal*: BMJ Publishing group.
- Peltonen, J., Cronin, N., Avela, J., & Finni, T. (2010). In vivo mechanical response of human Achilles tendon to a single bout of hopping exercise. *Journal of Experimental Biology, 213*(8), 1259-1265.
- Penedo, F. J., & Dahn, J. R. (2005). Exercise and well-being: a review of mental and physical health benefits associated with physical activity. *Current Opinion in Psychiatry, 18*(32), 189-189.
- Philippou, A., Bogdanis, G. C., Nevill, A. M., & Maridaki, M. (2004). Changes in the angle-force curve of human elbow flexors following eccentric and isometric exercise. *European Journal of Applied Physiology, 93*(1), 237-244.
- Pollock, C. M., & Shadwick, R. E. (1994). Relationship between body mass and biomechanical properties of limb tendons in adult mammals. *American Journal of Physiology - Regulatory, Integrative and Comparative Physiology, 266*(3), R1016-R1021.
- Prasartwuth, O., Allen, T. J., Butler, J. E., Gandevia, S. C., & Taylor, J. L. (2006). Length-dependent changes in voluntary activation, maximum voluntary torque and twitch responses after eccentric damage in humans. *The Journal of Physiology, 571*(1), 243-252.
- Prokopy, M. P., Ingersoll, C. D., Nordenschild, E., Katch, F. I., Gaesser, G. A., & Weltman, A. (2008). Closed-Kinetic Chain Upper-Body Training Improves Throwing Performance of NCAA Division I Softball Players. *The Journal of Strength & Conditioning Research, 22*(6), 1790-1798
1710.1519/JSC.1790b1013e318185f318637.
- Pruyn, E., Watsford, M., Murphy, A., Pine, M., Spurrs, R., Cameron, M., et al. (2012). Relationship between leg stiffness and lower body injuries in professional Australian football. *Journal of Sports Sciences, 30*(1), 71-78.
- Pua, Y.-H., Clark, R. A., & Bryant, A. L. (2010). Physical Function in Hip Osteoarthritis: Relationship to Isometric Knee Extensor Steadiness. *Archives of Physical Medicine and Rehabilitation, 91*(7), 1110-1116.

- Queen, R., Gross, M., & Liu, H. (2006). Repeatability of lower extremity kinetics and kinematics for standardized and self-selected running speeds. *Gait and Posture*, 23, 282-287.
- Racinais, S., Bringard, A., Puchaux, K., Noakes, T., & Perrey, S. (2008). Modulation in voluntary neural drive in relation to muscle soreness. *European Journal of Applied Physiology*, 102(4), 439-446.
- Reeves, N., Maganaris, C., & Narici, M. (2003). Effect of strength training on human patella tendon mechanical properties of older individuals. *The Journal of Physiology*, 548(3), 971-981.
- Renstrom, P. (1993). *Clinical practice of sports injury prevention and care*. Oxford: Blackwell Scientific.
- Ritchie, C., Paterson, K., Bryant, A. L., Bartold, S., & Clark, R. A. (2011). The effects of enhanced plantar sensory feedback and foot orthoses on midfoot kinematics and lower leg neuromuscular activation. *Gait & Posture*, 33(4), 576-581.
- Rodgers, M. M. (1988). Dynamic Biomechanics of the Normal Foot and Ankle During Walking and Running. *Physical Therapy*, 68(12), 1822-1830.
- Rolf, C. (1995). Overuse injuries of the lower extremity in runners. *Scandinavian Journal of Medicine & Science in Sports*, 5(4), 181-190.
- Rolf, C., & Movin, T. (1997). Etiology, histopathology, and outcome of surgery in achillodynia. *Foot and Ankle International*(18), 565-569.
- Rosager, S., Aagaard, P., Dyhre-Poulsen, P., Neergaard, M., Kjaer, M., & Magnusson, S. (2002). Load-displacement properties of the human triceps surae aponeurosis and tendon in runners and non-runners. *Scandinavian Journal of Medicine and Science in Sports*, 12, 90-98.
- Sadeghi, H., Allard, P., & Duhaime, M. (1997). Functional gait asymmetry in able-bodied subjects. *Human Movement Science*, 16, 243.
- Salmons, S. (1969). *The 8th International Conference on Medical and Biological Engineering - meeting report*, 4, 467-474.
- Saunders, P., Pyne, D., Telford, R., & Hawley, J. (2004). Reliability and Variability of Running Economy in Elite Distance Runners. *Medicine and Science in Sports and Exercise*, 36(11), 1972-1976.
- Sayers, S., & Hubal, M. (2008). Histological, chemical, and functional manifestations of muscle damage. In P. Tiidus (Ed.), *Skeletal Muscle Damage and Repair*. Illinois: Human Kinetics.
- Schache, A., Wrigley, T., Baker, R., & Pandy, M. G. (2009). Biomechanical response to hamstring muscle strain injury. *Gait & Posture*, 29(2), 332-338.
- Schechtman, H., & Bader, D. L. (1997). In vitro fatigue of human tendons. *Journal of Biomechanics*, 30(8), 829-835.
- Schechtman, H., & Bader, D. L. (2002). Fatigue damage of human tendons. *Journal of Biomechanics*, 35(3), 347-353.
- Semark, A., Noakes, T., Gibson, A., & Lambert, M. (1999). The effect of a prophylactic dose of flurbiprofen on muscle soreness and sprinting performance in trained subjects *Journal of Sports Sciences*, 17(3), 197-203.
- SENIAM. (2005). Project-Sensor placement. Retrieved 20/1/2011, from <http://www.seniam.org>

- Sinaki, M., Pfeifer, M., Preisinger, E., Itoi, E., Rizzoli, R., Boonen, S., et al. (2010). The Role of Exercise in the Treatment of Osteoporosis. *Current Osteoporosis Reports*, 8(3), 138-144.
- Smith, C., Young, I., & Kearney, J. (1996). Mechanical Properties of Tendons: Changes With Sterilization and Preservation. *Journal of Biomechanical Engineering*, 118(1), 56-61.
- Smith, L. (1991). Acute inflammation: the underlying mechanism in delayed onset muscle soreness? *Medicine & Science in Sports & Exercise*, 23(5), 542-551.
- Smith, L. (1992). Causes of Delayed Onset Muscle Soreness and the Impact on Athletic Performance: A Review. *Journal of Strength & Conditioning Research*, 6(3), 135-141.
- Stefanyshyn, D. J., & Nigg, B. M. (1998). Dynamic angular stiffness of the ankle joint during running and sprinting. *Journal of Applied Biomechanics*, 14(3), 292-299.
- Strong, W. B., Malina, R. M., Blimkie, C. J. R., Daniels, S. R., Dishman, R. K., Gutin, B., et al. (2005). Evidence Based Physical Activity for School-age Youth. *The Journal of Pediatrics*, 146(6), 732-737.
- Talbot, J., & Morgan, D. (1998). The effects of stretch parameters on eccentric exercise-induced damage to toad skeletal muscle. *Journal of Muscle Research and Cell Motility* 19(3), 237-245.
- Thornton, G., Oliynyk, A., Frank, C., & Shrive, N. (1997). Ligament creep cannot be predicted from stress relaxation at low stress: A biomechanical study of the rabbit medial collateral ligament. *Journal of Orthopaedic Research*, 15(5), 652-656.
- Thornton, G., Shrive, N., & Frank, C. (2002). Ligament creep recruits fibres at low stresses and can lead to modulus-reducing fibre damage at higher creep stresses: a study in rabbit medial collateral ligament model. *Journal of Orthopaedic Research*, 20(5), 967-974.
- Tsatalas, T., Giakas, G., Spyropoulos, G., Paschalis, V., Nikolaidis, M., Tsaopoulos, D., et al. (2010). The effects of muscle damage on walking biomechanics are speed-dependent. *European Journal of Applied Physiology*, 110(5), 977-988.
- Turner, C., Wang, T., & Burr, D. (2001). Shear strength and fatigue properties of human cortical bone determined from pure shear tests. *Calcified Tissue International*, 69(9), 373-378.
- Voigt, M., Chelli, F., & Frigo, C. (1998). Changes in the excitability of soleus muscle short latency stretch reflexes during human hopping after 4 weeks of hopping training. *European Journal of Applied Physiology and Occupational Physiology*, 78(6), 522-532.
- Walker, C. A., & Blair, R. (2002). Leg stiffness and damping factors as a function of running speed. *Sports Engineering*, 5, 139-139.
- Walsh, L., Hesse, C., Morgan, D., & Proske, U. (2004). Human forearm position sense after fatigue of elbow flexor muscles. *Journal of Physiology*, 558(2), 705-715.
- Walshe, A. D., & Wilson, G. J. (1997). The influence of musculotendinous stiffness on drop jump performance. *Canadian Journal of Applied Physiology*, 22(2), 117-132.

- Wang, J. (2006). Mechanobiology of tendon. *Journal of Biomechanics*, 39, 1563-1582.
- Warren, G., Hayes, D., Lowe, D., & Armstrong, R. (1993). Mechanical factors in the initiation of eccentric contraction-induced injury in rat soleus muscle. *Journal of Physiology*, 464, 457-475.
- Warren, G., Lowe, D., & Armstrong, R. (1999). Measurement tools used in the study of eccentric contraction-induced injury. *Sports Medicine*, 27(1), 43-59.
- Warren, G., & Palubinskas, L. (2008). Human and animal experimental models. In P. Tiidus (Ed.), *Skeletal Muscle Damage and Repair*. Illinois: Human Kinetics.
- Watsford, M. L., Murphy, A. J., McLachlan, K. A., Bryant, A. L., Cameron, M. L., Crossley, K. M., et al. (2010). A Prospective Study of the Relationship Between Lower Body Stiffness and Hamstring Injury in Professional Australian Rules Footballers. *The American Journal of Sports Medicine*, 38(10), 2058-2064.
- Whitehead, N. P., Allen, T. J., Morgan, D. L., & Proske, U. (1998). Damage to human muscle from eccentric exercise after training with concentric exercise. *The Journal of Physiology*, 512(2), 615-620.
- Whitehead, N. P., Weerakkody, N. S., Gregory, J. E., Morgan, D. L., & Proske, U. (2001). Changes in passive tension of muscle in humans and animals after eccentric exercise. *The Journal of Physiology*, 533(2), 593-604.
- Whiting, W., & Rugg, S. (2006). *Dynatomy: dynamic human anatomy*. Champaign, IL: Human Kinetics.
- Whiting, W., & Zernicke, R. (1998). *Biomechanics of musculoskeletal injury* (Second ed.). USA: Sheridan Books.
- Williams, D., McClay Davis, I., Scholz, J., Hamill, J., & Buchanan, T. (2004). High-arched runners exhibit increased leg stiffness compared to low-arched runners. *Gait and Posture*, 19, 263-269.
- Williams, T., Krahenbuhl, G., & Morgan, D. (1991). Daily variation in running economy of moderately trained male runners. *Medicine & Science in Sports & Exercise*, 23(8), 944-948.
- Wood, T., Cooke, P., & Goodship, A. (1988). The effect of exercise and anabolic steroids on the mechanical properties and crimp morphology of the rat tendon. *The American Journal of Sports Medicine*, 16(2), 153-158.
- Wren, T., Lindsey, D., Beaupre, G., & Carter, D. (2003). Effects of Creep and Cyclic Loading on the Mechanical Properties and Failure of Human Achilles Tendons. *Annals of Biomedical Engineering*, 31(6), 710-717.
- Yeow, C. H., Cheong, C. H., Ng, K. S., Lee, P. V. S., & Goh, J. C. H. (2008). Anterior Cruciate Ligament Failure and Cartilage Damage During Knee Joint Compression. *The American Journal of Sports Medicine*, 36(5), 934-942.
- Yoon, S., Tauchi, K., & Takamatsu, K. (2007). Effect of ankle joint stiffness during eccentric phase in rebound jumps on ankle joint torque at midpoint. *International Journal of Sports Medicine*, 28, 66-71.

- Yu, J.-G., Malm, C., & Thornell, L.-E. (2002). Eccentric contractions leading to DOMS do not cause loss of desmin nor fibre necrosis in human muscle. *Histochemistry and Cell Biology*, 118(1), 29-34.
- Zajac, F. E. (1989). Muscle and tendon: properties, models, scaling, and application to biomechanics and motor control. *Critical Reviews in Biomedical Engineering*, 17(4), 359-411.
- Zernicke, R., Garhammer, J., & Jobe, F. Human patellar tendon rupture: A kinetic analysis. *Medicine & Science in Sports & Exercise*, 13, 243-245.
- Zink, W., & Graf, B. M. Local anesthetic myotoxicity. *Regional Anesthesia and Pain Medicine*, 29(4), 333-340.