

2013

## Neuromuscular Fatigue and Biomechanical Alterations during High-Intensity, Constant-Load Cycling

Amanda Julie Overton  
*Edith Cowan University*

Follow this and additional works at: <https://ro.ecu.edu.au/theses>



Part of the [Sports Sciences Commons](#)

---

### Recommended Citation

Overton, A. J. (2013). *Neuromuscular Fatigue and Biomechanical Alterations during High-Intensity, Constant-Load Cycling*. Edith Cowan University. Retrieved from <https://ro.ecu.edu.au/theses/612>

This Thesis is posted at Research Online.  
<https://ro.ecu.edu.au/theses/612>

# Edith Cowan University

## Copyright Warning

You may print or download ONE copy of this document for the purpose of your own research or study.

The University does not authorize you to copy, communicate or otherwise make available electronically to any other person any copyright material contained on this site.

You are reminded of the following:

- Copyright owners are entitled to take legal action against persons who infringe their copyright.
- A reproduction of material that is protected by copyright may be a copyright infringement. Where the reproduction of such material is done without attribution of authorship, with false attribution of authorship or the authorship is treated in a derogatory manner, this may be a breach of the author's moral rights contained in Part IX of the Copyright Act 1968 (Cth).
- Courts have the power to impose a wide range of civil and criminal sanctions for infringement of copyright, infringement of moral rights and other offences under the Copyright Act 1968 (Cth). Higher penalties may apply, and higher damages may be awarded, for offences and infringements involving the conversion of material into digital or electronic form.

**EDITH COWAN UNIVERSITY**

**School of Exercise and Health Sciences**

# **Neuromuscular Fatigue and Biomechanical Alterations during High-Intensity, Constant-Load Cycling**

**By**

**Amanda J. Overton BPhEd. (Hons)**

This thesis is presented for the award of Doctor of Philosophy (Sport Sciences) from the  
School of Exercise and Health Sciences; Faculty of Computing, Health and Science;  
Edith Cowan University, Western Australia

**Principal Supervisor:** Assoc. Prof. Anthony J. Blazevich (Edith Cowan University)

**Co-Supervisor:** Dr Chris R. Abbiss (Edith Cowan University)

**Date of Submission:** 14<sup>th</sup> June 2013



## ABSTRACT

Neuromuscular fatigue is an inevitable process at play during prolonged exercise, and may be caused by multiple alterations within the central nervous system and peripheral musculature. As fatigue develops, the neuromuscular system must adapt to these changes by making compensatory movement pattern adjustments so as to use motor pathways that are less fatigued in an effort to maintain task performance; motor variability is thus increased. The primary purpose of the four studies contained within this doctoral thesis was to detail the progression of exercise-induced neuromuscular fatigue, and to improve our understanding of the muscle activation and joint kinematic alterations that occur as fatigue accumulates. Within this context, cycling was used as the exercise model, and the relationship between physiological and biomechanical aspects of high-intensity, moderate duration (<10 min) cycling were specifically examined.

The first two studies of this thesis were aimed at understanding the progression of neuromuscular fatigue as well as the associated motor control and biomechanical (i.e. muscle activation and kinematic) changes that occur during exhaustive cycling. Specifically, the time course and relative contributions of central and peripheral fatigue mechanisms, and the associated changes in muscle activation and both lower (i.e. hip, knee and ankle joint) and upper (i.e. trunk) limb kinematics were examined during a high-intensity cycling time to exhaustion (TTE) test. This was performed at 90% maximal aerobic power ( $P_{\max}$ ) with nine well-trained cyclists. Temporal relationships between joint kinematics and changes in markers of central and peripheral fatigue were also examined. Peripheral fatigue (i.e. impaired contractile function: reduced peak twitch torque, -39.9%; twitch contraction time, -10.7%; and the average rates of twitch torque development -34.7% and relaxation -36.7% at task failure i.e.,  $T_{100}$ ) developed early in the exercise bout from 60% of the time to task failure ( $p < 0.05$ ). However, a central facilitation, measured as an increase in peak vastus medialis (38.9%) and gluteus maximus electromyogram (87.2%) amplitudes at  $T_{100}$ , rather than central fatigue, occurred towards the end of the exercise task ( $p < 0.05$ ). Thus, neuromuscular fatigue development was associated with an increase in the magnitude of lower limb muscle activity, which may have represented an attempt to increase muscle force to maintain the required power output of the cycling task. Increases in trunk flexion were observed from 60% of the time to task failure ( $p < 0.05$ ), and were therefore notable at or after the

point of significant peripheral fatigue. Conversely, increases in trunk medio-lateral sway (lateral flexion), hip abduction/adduction and knee valgus/varus were observed only from 80% of the time to task failure ( $p < 0.05$ ), which paralleled the increase in central motor drive. The results of this study therefore indicate that significant trunk kinematic changes in the sagittal plane occurred at or after the point of significant peripheral fatigue development, whereas, significant changes at the trunk, hip and knee joints in the coronal plane occurred later in the exercise task and paralleled the facilitation of central motor drive during the cycling task.

In the third study, the effects of real-time, kinematic feedback provision for trunk flexion ( $TTE_{Tflex}$ ), trunk medio-lateral sway ( $TTE_{Tsway}$ ) and hip abduction/adduction ( $TTE_{Habd/add}$ ) during a high-intensity TTE cycling test (90%  $P_{max}$ ) in nine well-trained cyclists were examined. The times taken to reach task failure were compared to a TTE test completed with no feedback. The times taken to reach task failure were not significantly different when provided with trunk flexion ( $TTE_{Tflex}$ ) and hip abduction/adduction ( $TTE_{Habd/add}$ ) feedback compared to the non-feedback condition ( $p > 0.05$ ). There was, however, a significant decrease in the time to task failure during the  $TTE_{Tsway}$  test ( $p < 0.05$ ). Not all participants could maintain trunk and/or hip movement within a set movement pattern criteria; and three participants were therefore excluded from the kinematic analyses for both the  $TTE_{Tflex}$  and  $TTE_{Tsway}$  tests ( $n = 6$ ) as were two participants from the  $TTE_{Habd/add}$  test ( $n = 7$ ). For participants who correctly used the kinematic feedback, no differences in the times taken to reach failure were observed in between the feedback ( $TTE_{Tflex}$ ,  $TTE_{Tsway}$  and  $TTE_{Habd/add}$ ) and non-feedback test conditions ( $p > 0.05$ ). Despite being given feedback, changes in joint kinematics were similar across all test conditions; significant alterations were observed at the trunk and knee joints in the sagittal plane and at the hip and knee joints in the coronal plane ( $p < 0.05$ ). Given trunk flexion feedback ( $TTE_{Tflex}$ ), significant increases in left hip flexion and trunk medio-lateral sway ROM were observed ( $p < 0.05$ ), whereas given trunk medio-lateral sway feedback ( $TTE_{Tsway}$ ), increases in right hip flexion ROM also occurred ( $p < 0.05$ ). These results indicate that, regardless of whether or not well-trained cyclists are able to control the level of kinematic variability when fatigued, acute exposure to real-time kinematic feedback to limit trunk or hip movement during high-intensity cycling may influence cycling kinematics (i.e. technique) and, in some cases (e.g. trunk medio-lateral sway), may reduce performance.

The final study examined the relationship between joint kinematics, measured in non-fatigued and fatigued high-intensity cycling, and the cyclists' physiological profiles

(i.e., physiological attributes indicative of successful cycling ability, including both maximal oxygen consumption and peak power output relative to body mass, maximal heart rate, both power output and heart rate at the first and second ventilatory thresholds and cycling economy at 100 W) and the time taken to reach task failure. Submaximal physiological attributes were correlated with hip (abduction/adduction angle and ROM), knee (flexion angle) and ankle (flexion ROM) kinematics measured in a non-fatigued state at the start of the trial ( $r > 0.40$ ;  $p < 0.05$ ). However, both physiological attributes associated with maximal exercise capacity and cycling economy were correlated with trunk (flexion angle) and ankle (flexion angle and ROM) kinematics measured in a fatigued state at the end of the test ( $r > 0.40$ ;  $p < 0.05$ ). Trunk flexion and medio-lateral sway ROM in a non-fatigued state, and trunk flexion angle in a fatigued state, were associated with the time to task failure ( $r > 0.50$ ;  $p < 0.05$ ). Thus, the degree of trunk flexion and medio-lateral sway may be important kinematic variables that are indicative of cycling performance. These findings reveal an interdependence between cycling kinematics and both the physiological attributes indicative of successful cycling performance and the time taken to reach task failure during high-intensity, constant-load cycling.

In conclusion, the findings presented in this thesis indicate that the temporal patterns of central and peripheral neuromuscular fatigue differ (Study 1; Chapter 3). Task failure during high intensity cycling appears to be associated with the development of peripheral fatigue despite the presence of an increase in central motor drive. Subsequent to the development of neuromuscular fatigue, muscle activation and joint kinematic alterations can be observed, which may represent compensatory mechanisms employed by the neuromuscular system to continue task performance (Studies 1 and 2; Chapters 3 and 4). Joint kinematic alterations in the sagittal plane were associated with the development of peripheral fatigue whereas coronal plane adjustments occurred in parallel with central facilitation, and/or when a more substantial level of peripheral fatigue accumulated. Such compensatory kinematic strategies are also associated with an athlete's physiological attributes and their cycling performance (i.e., time to task failure) (Study 4; Chapter 6). Importantly, imposing specific joint kinematic restrictions (trunk flexion, trunk medio-lateral sway and hip abduction/adduction) during exhaustive cycling, influenced cycling kinematics (i.e. technique) and, in some cases (e.g. trunk medio-lateral sway), reduced the time taken to reach task failure for well-trained cyclists (Study 3; Chapter 5). Such findings enhance our understanding of how the neuromuscular system copes with fatigue development, and should assist coaches

and/or occupational health practitioners to better understand the fatigue process and neuromuscular strategies utilised during exercise tasks with similar characteristics to that used in the current studies.



## DECLARATION

I certify that this thesis does not, to the best of my knowledge and belief:

- i. incorporate without acknowledgement any material previously submitted for a degree or diploma in any institution of higher education;
- ii. contain any material previously published or written by another person except where due reference is made in text; or
- iii. contain any defamatory material

I also grant permission for the Library at Edith Cowan University to make duplicate copies of my thesis as required.

Signature: \_\_\_\_\_

Date: \_\_\_\_\_

## USE OF THESIS

The Use of Thesis statement is not included in this version of the thesis.

*This thesis is dedicated to Robert, my Mum, my brother Darren  
and to the Taylor family for always believing in me  
– thank you for your love and support.*

*In loving memory of Alan John Overton*

*23.06.1958 – 10.04.2003*

## ACKNOWLEDGMENTS

I am so proud to even be writing the acknowledgements section of this thesis, as this means that I am nearly finished my long, challenging and at times rewarding PhD journey.

I want to start by thanking the two most important people in my academic life – my supervisors, Anthony Blazeovich and Chris Abbiss. Without these two individuals there would be no Doctor before my name! I am so very grateful to both of you, and appreciated the different ideas and approaches you brought to my research; I feel that this has only made me a stronger researcher and know this will benefit me in the future. Tony, your enthusiasm and love of research astounds me. You have been the best supervisor I could ever wish to have, you are honest, encouraging and a great teacher, and if not for your guidance and support I would not have come this far. And to Chris, the same goes for you, I want to thank you for your ability to listen, I always felt I could come to you with any problem and this really helped me in the ‘troughs’ of my PhD. You are an excellent researcher who I am so glad to have been able to work with. I want to thank you both for all your hard work in helping me to achieve this PhD, it is extremely appreciated.

Next I would like to thank those individuals who without their help this research would not have been possible. The lab technicians past and present, Nadija Vrdoljak, Jack Burns, Elizabeth Depetro and Helen Alexander; your tireless effort, willing attitude and genuine attempt to make my data collection run as smoothly as possible was very much appreciated. To my MATLAB guru Gavin Kennedy; the coding you helped me with was essential for the completion of my data analysis and therefore this thesis, so I am immensely grateful. I would also like to thank Jodie Wilkie in particular; you have been such a role model for me over the years and I have always appreciated your advice and support. Finally, I want to thank all my participants – without your participation none of this work would be possible, so thank you for volunteering numerous hours of your time and putting in 110% effort during testing!

Next, I would like to thank my fellow research companions, the post-graduate team, past and present. I arrived into the group of “Tony’s girls” that included Marika Noorkoiv, Jo Trezise and I. How lucky I was to share my research experience with both of you, the research rollercoaster was made the better with you there alongside me, so thank you. There have been other post-graduates who have offered me personal and academic guidance. These individuals have helped me towards gaining this PhD, but

more importantly, have become my friends. Thanks for understanding the research issues we all face and making me see that we are all in the same boat. In no particular order I would like to extend my gratitude to Rod Siegel, Luis Penailillo, Harry Banyard, Tania Spiteri, Gabriel Trajano, Laurent Seitz, Anders Stavnsbo, Julia Skleryk, Sam Goh, Chris Joyce, Joe Mate, Nic Hart, Mike Barker, Jake Earp, and Roy Chan - I wish all of you the best in your chosen careers.

Last but definitely not least, to the most important people in my life – Robert, my family and friends. These words are the hardest to write of the entire thesis. Robert, you are such an amazing support to me, through both the ups and downs over the past four (and a bit) years you have always been there for me, so thank you. I know you have waited a long time for this to be finished, and I hope that I have made you proud. I am so looking forward to beginning the next chapter of our lives together! Mum and Darren, thank-you for always supporting and believing in me, I don't know where I would be without either of you in my life. To the Taylor family, and in particular Ken and Vickie, I know that your initial encouragement to do this PhD is why I took this path – so thank you for making me realise the value of further education. I also want to thank my beautiful friends Kate and Jessie; you have been such a big support to me throughout this journey, particularly when Robert has been away, I am so grateful and honoured to have you both as friends. Gabrielle, I'd also like to thank-you for lending me your University of Otago library details, I have found numerous, helpful journal articles because of that! Finally, to my Dad, who sadly is no longer here with us – I hope that I have made you proud. To all of you, thank you for keeping me sane and reminding me that this doctorate is only one aspect of my life and definitely not the most important.

## TABLE OF CONTENTS

ABSTRACT .....	i
DECLARATION .....	v
COPYRIGHT AND ACCESS STATEMENT .....	vi
ACKNOWLEDGMENTS .....	viii
TABLE OF CONTENTS .....	x
LIST OF TABLES .....	xiv
LIST OF FIGURES .....	xv
LIST OF PUBLICATIONS .....	xvi
CHAPTER ONE .....	1
Introduction .....	1
1.1 Overview .....	1
1.2 Background .....	1
1.3 Significance of the Research .....	4
1.4 Purpose of the Research .....	5
1.5 Research Questions .....	5
1.5.1 Study 1 (Chapter 3) .....	6
1.5.2 Study 2 (Chapter 4) .....	6
1.5.3 Study 3 (Chapter 5) .....	6
1.5.4 Study 4 (Chapter 6) .....	7
1.6 Research Hypotheses .....	7
1.6.1 Study 1 (Chapter 3) .....	7
1.6.2 Study 2 (Chapter 4) .....	7
1.6.3 Study 3 (Chapter 5) .....	8
1.6.4 Study 4 (Chapter 6) .....	8
1.7 Limitations .....	8
1.8 Delimitations .....	10
1.9 Definitions of Selected Terms .....	11
CHAPTER TWO .....	13
Review of Literature .....	13
2.1 Introduction .....	13
2.2 Mechanisms Underpinning Exercise-Induced Neuromuscular Fatigue .....	14
2.2.1 Peripheral fatigue .....	15
2.2.2 Central fatigue .....	16

2.2.4	Interaction between peripheral and central fatigue .....	17
2.2.4	Measurement of neuromuscular fatigue during exercise .....	21
2.3	Alterations in Neuromuscular Fatigue .....	24
2.3.1	Motor variability and reorganisation during fatigue .....	25
2.3.2	Changes in muscle activation with fatigue.....	27
2.3.3	Changes in joint kinematics with fatigue .....	30
2.4	Relationships between Physiological Attributes of an Individual and Biomechanical Alterations in Response to Neuromuscular Fatigue.....	33
2.5	Summary and Conclusion .....	35
CHAPTER THREE.....		37
Evidence of peripheral fatigue and up-regulation of central motor drive during high-intensity, constant-load cycling.....		37
3.1	Abstract .....	37
3.2	Introduction .....	39
3.3	Methods .....	40
3.3.1	Participants.....	40
3.3.2	Experimental protocol.....	41
3.3.3	Statistical analyses .....	47
3.4	Results .....	47
3.5	Discussion .....	54
CHAPTER FOUR.....		60
The temporal relationship between joint kinematics and neuromuscular fatigue in high-intensity, constant-load cycling.....		60
4.1	Abstract .....	60
4.2	Introduction .....	61
4.3	Methods .....	63
4.3.1	Participants.....	63
4.3.2	Experimental protocol.....	63
4.3.3	Data analyses.....	65
4.3.4	Statistical analyses .....	66
4.4	Results .....	66
4.5	Discussion .....	72
CHAPTER FIVE.....		76
Effects of real-time, visual kinematic feedback on the time taken to reach task failure during high-intensity, constant-load cycling.....		76

5.1	Abstract .....	76
5.2	Introduction .....	78
5.3	Methods .....	79
5.3.1	Participants.....	79
5.3.2	Experimental protocol.....	80
5.3.3	Data analyses.....	83
5.3.4	Statistical analyses .....	85
5.4	Results .....	85
5.5	Discussion .....	88
CHAPTER SIX .....		94
Interdependence of physiological and biomechanical factors in determining high-intensity cycling performance in well-trained cyclists .....		94
6.1	Abstract .....	94
6.2	Introduction .....	96
6.3	Methods .....	98
6.3.1	Participants.....	98
6.3.2	Experimental protocol.....	98
6.3.3	Statistical analyses .....	99
6.4	Results .....	100
6.5	Discussion .....	104
CHAPTER SEVEN.....		109
General Discussion .....		109
7.1	Directions for future research.....	116
7.2	Conclusion.....	121
CHAPTER EIGHT .....		122
References .....		122
CHAPTER NINE.....		151
Appendices .....		151
APPENDIX A .....		152
APPENDIX B .....		154
APPENDIX C .....		156
APPENDIX D .....		158
APPENDIX E .....		159
APPENDIX F.....		163
APPENDIX G.....		168



APPENDIX H .....	170
APPENDIX I.....	173
APPENDIX J .....	176
APPENDIX K .....	178
APPENDIX L .....	179
APPENDIX M .....	180
APPENDIX N .....	181
APPENDIX O .....	184

## LIST OF TABLES

<b>Table 3.1</b>	Percent changes in peak and mean electromyogram (EMG) amplitudes and the EMG impulse of seven lower limb muscles during a high-intensity, constant-load cycling bout for ten pedal cycles from $T_0$ to $T_{20}$ , $T_{40}$ , $T_{60}$ , $T_{80}$ and $T_{100}$ .....	50
<b>Table 3.2</b>	Changes in the mean electromyogram frequency of seven lower limb muscles during a high-intensity, constant-load cycling bout for ten pedal cycles at $T_0$ , $T_{20}$ , $T_{40}$ , $T_{60}$ , $T_{80}$ and $T_{100}$ . ....	51
<b>Table 3.3</b>	Percent changes in isometric maximal voluntary contraction (MVC) mechanical and electromyogram (EMG) responses during a high-intensity, constant-load cycling bout from $T_0$ to $T_{20}$ , $T_{40}$ , $T_{60}$ , $T_{80}$ and $T_{100}$ .....	53
<b>Table 4.1</b>	Kinematic changes at the trunk, hip, knee and ankle joints in the sagittal and coronal planes during a high-intensity, constant-load cycling bout for ten pedal cycles at $T_0$ , $T_{20}$ , $T_{40}$ , $T_{60}$ , $T_{80}$ and $T_{100}$ . ....	69
<b>Table 5.1</b>	Power output, cadence and the times taken to reach task failure for the time to exhaustion (TTE) test with no feedback and the TTE tests with real-time, kinematic feedback for trunk flexion ( $TTE_{Tflex}$ ), trunk medio-lateral sway ( $TTE_{Tsway}$ ) and hip abduction/adduction ( $TTE_{Habd/add}$ ) for all participants and for those who met the movement pattern inclusion criteria for each feedback condition. ....	87
<b>Table 5.2</b>	Trunk, hip, knee and ankle joint kinematics in the sagittal and coronal planes at the start ( $T_0$ ) and at task failure ( $T_{100}$ ) of high-intensity, constant-load cycling bouts performed with either no feedback (TTE test) or real-time, kinematic feedback for trunk flexion ( $TTE_{Tflex}$ test) – Appendix A. ....	152
<b>Table 5.3</b>	Trunk, hip, knee and ankle joint kinematics in the sagittal and coronal planes at the start ( $T_0$ ) and at task failure ( $T_{100}$ ) of high-intensity, constant-load cycling bouts performed with either no feedback (TTE test) or real-time, kinematic feedback for trunk medio-lateral sway ( $TTE_{Tsway}$ test) – Appendix B. ....	154
<b>Table 5.4</b>	Trunk, hip, knee and ankle joint kinematics in the sagittal and coronal planes at the start ( $T_0$ ) and at task failure ( $T_{100}$ ) of high-intensity, constant-load cycling bouts performed with either no feedback (TTE test) or real-time, kinematic feedback for hip abduction/adduction ( $TTE_{Habd/add}$ test) – Appendix C. ....	156
<b>Table 6.1</b>	Physiological attributes of 18 well-trained cyclists measured during the incremental cycling test to exhaustion. ....	100
<b>Table 6.2</b>	Trunk, hip, knee and ankle joint kinematics for ten pedal cycles during non-fatigued ( $T_0$ ) and fatigued ( $T_{100}$ ) high-intensity, constant-load cycling. ....	101

## LIST OF FIGURES

<b>Figure 3.1</b>	Schematic representations of the study design and neuromuscular assessments. ....	45
<b>Figure 3.2</b>	Percent changes in gluteus maximus peak and mean electromyogram (EMG) amplitude and EMG impulse during a high-intensity, constant-load cycling bout from $T_0$ to $T_{20}$ , $T_{40}$ , $T_{60}$ , $T_{80}$ and $T_{100}$ . ....	49
<b>Figure 3.3</b>	Peak twitch torque ( $T_{p,twitch}$ ) (A), twitch contraction time ( $CT_{twitch}$ ) (B), and the average rates of twitch torque development ( $RTD_{twitch}$ ) (C) and relaxation ( $RTR_{twitch}$ ) (D) during a high-intensity, constant-load cycling bout from $T_0$ to $T_{20}$ , $T_{40}$ , $T_{60}$ , $T_{80}$ and $T_{100}$ . ....	54
<b>Figure 4.1</b>	Schematic representations of joint angle definitions. ....	65
<b>Figure 4.2</b>	Trunk flexion and medio-lateral sway, hip abduction/adduction and knee valgus/varus joint angles during a high-intensity, constant-load cycling bout. ....	68
<b>Figure 5.1</b>	Set-up for the provision of real-time, kinematic feedback during a high-intensity, constant-load cycling bout. ....	82
<b>Figure 6.1</b>	The relationship between mean trunk flexion angle at $T_{100}$ (i.e., task failure) and maximal heart rate during high-intensity cycling. ....	102
<b>Figure 6.2</b>	The relationships between left and right ankle joints range of motion at $T_{100}$ (i.e., at task failure) and cycling economy measured at 100 W during high-intensity cycling. ....	103
<b>Figure 6.3</b>	The relationship between mean trunk flexion angle at $T_{100}$ (i.e., at task failure) and the time taken to reach task failure during high-intensity cycling. ....	104

## **LIST OF PUBLICATIONS**

Certain chapters of this thesis have been presented at conferences; these publications are outlined below.

### **Conference presentations**

17th annual Congress of the European College of Sport Science, 4-7 July 2012, Bruges – Belgium. Poster presentation: Overton AJ, Abbiss CR, and Blazevich AJ. Neuromuscular fatigue development and biomechanical changes during constant-load, high-intensity cycling exercise (Appendix O).

1  
2  
3  
4  
5  
6  
7  
8  
9  
10  
11  
12  
13  
14  
15  
16  
17  
18  
19  
20  
21  
22  
23  
24  
25  
26  
27  
28  
29  
30

**CHAPTER ONE**

**Introduction**

**1.1 Overview**

This doctoral thesis contains four research studies with the underlying focus of better understanding the development of exercise-induced neuromuscular fatigue and its association with movement pattern variation. All studies included in this thesis were controlled experimental studies. The first two studies aimed to understand the progression of neuromuscular fatigue and associated motor control and biomechanical (i.e. muscle activation and kinematic) changes that occur during high-intensity, constant-load cycling. Specifically, Study 1 was developed to quantify the development of central and peripheral fatigue and describe the associated temporal alterations in muscle activation patterns during high intensity exercise. The relationships between alterations in cycling kinematics and the progression of neuromuscular fatigue were then examined in Study 2. Following this, Study 3 examined the effect of real-time, visual kinematic feedback of kinematic patterns on cycling performance. In the final study of this thesis (Study 4), relationships between physiological attributes that are commonly considered indicative of superior cycling ability (i.e. peak power output ( $W_{peak}$ ), maximal oxygen consumption ( $VO_{2max}$ ), metabolic thresholds (i.e. lactate or ventilation thresholds) and economy) and joint kinematics of well-trained male cyclists during high-intensity cycling were examined. In addition, the relationship between joint kinematics and cycling performance (i.e. the time taken to reach task failure) was examined.

**1.2 Background**

For more than a century, scientists have attempted to understand the mechanisms responsible for the development of exercise-induced fatigue (144, 217, 251). The majority of earlier research indicated that muscular, i.e. peripheral, fatigue may result from either metabolic limitations or the formation of ‘fatigue biproducts’. Indeed, Mosso stated that ‘fatigue is a chemical process’ (217), and Hill described this phenomenon as ‘oxygen want’ (144). However, scientists also acknowledged the

1 importance of central factors in the development of fatigue, and in 1928 Reid and  
2 colleagues showed that both central and peripheral fatigue contributed to the ‘fatigue  
3 point’ (251). Over the past century, our understanding of fatigue has significantly  
4 improved, however the precise mechanisms responsible for task failure/exhaustion (i.e.  
5 the point at which an individual voluntarily terminates exercise) remain unclear.

6       Recent research examining exercise-induced fatigue has focused on alterations  
7 occurring within the neuromuscular system (for example, (124, 178, 205, 240, 242,  
8 296)). Neuromuscular fatigue can be defined as an inability to maintain a given force or  
9 power output and has been found to have both central and peripheral origins (6, 117,  
10 294). Central fatigue is defined as a progressive exercise-induced reduction in voluntary  
11 activation of muscle and occurs within the central nervous system (CNS) at cortical and  
12 spinal levels (117, 294). Peripheral fatigue, by contrast, occurs at or distal to the  
13 neuromuscular junction and is described as any decline in muscle performance (6, 117).  
14 To date, our ability to understand the development and relative contributions of central  
15 and peripheral fatigue mechanisms has been somewhat limited by the methodological  
16 designs used in research. Exercise-induced fatigue has primarily been investigated at  
17 task failure; such methodology does not adequately capture the time course of fatigue  
18 development and inherently assumes that fatigue development is relatively constant  
19 across the entire exercise duration. However, a relatively linear increase in  
20 neuromuscular fatigue is unlikely to occur during high-intensity exercise and it is  
21 therefore important to examine the temporal changes in central and peripheral fatigue  
22 mechanisms during a repetitive, high-intensity exercise bout. Indeed, Decorte et al.  
23 (2012) has recently assessed the time course of central and peripheral fatigue  
24 development during intermittent bouts of constant-load cycling (82). This is the only  
25 study that has shown that reduced efficacy of excitation-contraction (E-C) coupling was  
26 “compensated for” by an increase in central motor drive, as indicated by an increase in  
27 the electromyogram (EMG) amplitude (82). Although this study provided some insight  
28 into the temporal pattern of neuromuscular fatigue development and the magnitude of  
29 change in fatigue mechanisms, it is still unclear whether these changes are comparable  
30 during continuous exercise bouts.

31       As neuromuscular fatigue develops, associated changes in joint rigidity, the  
32 relative activity of muscles and the timing of muscle activity are observed during  
33 dynamic multi-joint exercise (117). Such changes in muscle activation likely enforce  
34 changes in joint kinematics and increase the variability of movement patterns, which is  
35 commonly referred to as motor variability (282). Movement pattern changes have

1 historically been considered as noise in the neuromuscular system and thus were viewed  
2 as a negative outcome of fatigue and therefore detrimental for exercise performance (25,  
3 282). However, recent data have indicated that variations in movement patterns may in  
4 fact be a beneficial adaptation within the neuromuscular system that may assist in  
5 delaying further fatigue development, avoiding injury and maintaining task performance  
6 (25, 282). These adjustments occur presumably in an effort to increase the activation of  
7 less fatigued muscles or muscle groups and maintain task performance. To date few  
8 studies have documented kinematic alterations throughout a fatiguing cycling bout (37,  
9 38, 87, 192, 266) and the associated changes in lower limb muscle recruitment (EMG)  
10 strategies (87). In addition, there are limited data describing the association between the  
11 site (i.e., mechanism) of neuromuscular fatigue and changes in muscle activation (82)  
12 and/or joint kinematics, and thus alterations in movement variability during cycling.  
13 Therefore, the effects of modulating the level of movement variability within the system  
14 on skill execution, and thus cycling performance, are not known. Further work is  
15 needed to identify muscle activation and kinematic strategies utilised during fatiguing  
16 exercise, and to determine whether allowing changes in joint kinematics, and thus  
17 encouraging kinematic variability, is beneficial to performance.

18         It has been well established that an individual's physiological profile largely  
19 dictates their exercise performance/capacity. Indeed,  $W_{peak}$ ,  $VO_{2max}$ , metabolic  
20 thresholds (i.e. lactate or ventilation thresholds), maximal lactate steady-state (i.e. the  
21 highest exercise intensity at which blood lactate concentration is stable) and efficiency  
22 or economy have all been found to be significantly correlated with performance in  
23 exercise tasks such as cycling (21, 23, 73, 103, 104, 135, 183). In addition, such  
24 characteristics are also likely to be associated with both the timing and magnitude of  
25 muscle activation and kinematic changes during fatigue. Indeed, it is believed that the  
26 application of an effective pedal force profile through the use of an 'optimum cycling  
27 technique' is a key factor influencing cycling efficiency, and should reduce fatigue and  
28 thus enhance performance (173). However, few studies have examined the relationship  
29 between the physiological characteristics of cyclists and changes in biomechanics (i.e.  
30 kinetics and/or kinematics) during cycling (73, 173). Of the studies that have examined  
31 the association between physiological characteristics and cycling kinetics, no significant  
32 relationships have been observed (73, 173). In addition, no studies have examined the  
33 relationship between the physiological attributes of a cyclist and cycling technique (i.e.  
34 kinematics). However, the relationships between kinematic variables and physiological  
35 characteristics, such as  $VO_{2max}$ , heart rate and blood pressure responses, blood lactate

and economy, have been examined during other locomotive tasks (i.e. running (167, 222, 228, 286, 322), swimming (24, 68, 239, 328) and alpine skiing (268)). Knowledge of the relationship between a cyclist's physiological attributes and cycling kinematics during exhaustive cycling would allow for a better understanding of the interdependence of the physiological response of a cyclist and cycling technique. It is also plausible that specific physiological characteristics may be related to an athlete's ability to resist fatigue and thus to maintain cycling technique towards the end of a high-intensity exercise bout.

### 1.3 Significance of the Research

Neuromuscular fatigue occurs during many manual labour (e.g. lifting, or hammer, shovel or axe use) and sporting or exercise tasks that require moderate periods (<10 min) of relatively high intensity work (e.g. >80% of  $\text{VO}_{2\text{max}}$  or power output). During fatigue the neuromuscular system can employ numerous neurally-mediated (or compensatory) strategies in order to maintain performance and complete a motor task (30, 235). Research examining the precise mechanisms underpinning the development of neuromuscular fatigue during high-intensity, constant-load exercise, the muscle activation and kinematic strategies employed, and their potential benefit is limited. Consequently, it is not known whether it is advantageous to maintain a uniform technique or promote kinematic variability during fatiguing exercise, to delay fatigue progression and optimise exercise performance.

Enhancing our understanding of exercise-induced fatigue and compensatory neuromuscular strategies has implications for a wide range of motor tasks and subject populations. Indeed, the ability to sustain exercise is particularly important for athletes, but perhaps more importantly poor exercise capacity is associated with an increased risk of cardiovascular disease, disability and mortality in the general population (190). As such, this research is of importance to those with poor movement capacity (e.g. those who suffer from diseases or chronic conditions, or the elderly), individuals whose jobs involve repetitive actions or inadequate postures often leading to work-related muscular skeletal disorders (71, 131, 218), and athletes/coaches aiming to optimise performance of cyclic, repetitive tasks that involve moderate periods of high intensity work. As a result, the ability to identify and effectively employ compensatory strategies during fatiguing work bouts may be of significant importance to personal health, safety and performance. Finally, given the known relationships between biomechanical and



physiological variables in other locomotor tasks (24, 68, 167, 222, 228, 239, 268, 286, 321, 328), it is interesting that there is presently no research examining the relationship between movement kinematics of well-trained cyclists during cycling and either the physiological attributes previously shown to be indicative of successful cycling performance or the time taken to reach task failure. It is of importance to determine whether any relationships exist between the physiological response of a cyclist and cycling technique, and to examine whether there are certain physiological attributes that relate to specific kinematic changes in cycling technique and/or one's ability to continue exercise and thus resist fatigue.

#### **1.4 Purpose of the Research**

The overall purpose of the research presented in this thesis is to enhance our understanding of exercise-induced neuromuscular fatigue and the compensatory neuromuscular strategies that may be adopted in order to prevent fatigue development and thus improve exercise performance. Specifically, the purpose of Study 1 was to determine the time-course of the development of both central and peripheral fatigue, as well as changes in lower limb muscle activation during high-intensity, constant-load cycling in well-trained male cyclists. Following this, the temporal association between the development of central and peripheral fatigue and alterations in movement kinematics during high-intensity, constant-load cycling was examined in Study 2. The purpose of Study 3 was to determine whether limiting the variation of trunk flexion, trunk medio-lateral sway (lateral flexion) and hip abduction/adduction using real-time, visual kinematic feedback influenced the time taken to reach task failure during high-intensity, constant-load cycling. Subsequently, the purpose of Study 4 was to examine the association between cycling kinematics and the physiological attributes of well-trained male cyclists, and the time taken to reach task failure during high-intensity, constant-load cycling.

#### **1.5 Research Questions**

The research questions asked in this PhD thesis have been divided into four separate studies, as listed below:

1 **1.5.1 Study 1 (Chapter 3)**

2  
3 **Evidence of peripheral fatigue and up-regulation of central motor drive during**  
4 **high-intensity, constant-load cycling.**

- 5  
6 i. What are the time courses of the development, and relative  
7 contribution, of both central and peripheral fatigue during high-  
8 intensity, constant-load cycling?  
9 ii. Are there associated changes in the level of muscle activity and/or the  
10 timing of muscle activation in the lower limb during this type of  
11 exercise?  
12

13 **1.5.2 Study 2 (Chapter 4)**

14  
15 **The temporal relationship between joint kinematics and neuromuscular fatigue in**  
16 **high-intensity, constant-load cycling.**

- 17  
18 i. What are the temporal relationships between kinematic alterations and  
19 changes in key indicators of neuromuscular fatigue (identified from  
20 Study 1)?  
21 ii. What is the magnitude of change in lower (i.e. hip, knee and ankle  
22 joint) and upper (i.e. trunk) limb kinematics in both the sagittal and  
23 coronal planes during high-intensity, constant-load cycling?  
24

25 **1.5.3 Study 3 (Chapter 5)**

26  
27 **Effects of real-time, visual kinematic feedback on the time taken to reach task**  
28 **failure during high-intensity, constant-load cycling.**

- 29  
30 i. Does limiting the variation of specific kinematic variables (trunk  
31 flexion, trunk medio-lateral sway and hip abduction/adduction;  
32 identified from Study 2) using real-time, visual kinematic feedback  
33 influence the time taken to reach task failure during high-intensity,  
34 constant-load cycling?

#### 1.5.4 Study 4 (Chapter 6)

##### **Interdependence of physiological and biomechanical factors in determining high-intensity cycling performance in well-trained cyclists.**

- i. Are there relationships between physiological attributes that are considered indicative of successful cycling performance (i.e. power output and heart rate at the first ( $VT_1$ ) and second ( $VT_2$ ) ventilatory thresholds,  $W_{peak}$ , maximal heart rate ( $HR_{max}$ ),  $VO_{2max}$  and cycling economy), and lower (i.e. hip, knee and ankle joint) and upper (i.e. trunk) limb kinematics measured during high-intensity cycling in both a non-fatigued (i.e. at the start of exercise;  $T_0$ ) and fatigued (i.e. at task failure;  $T_{100}$ ) state?
- ii. Are there relationships between joint kinematics and the time taken to reach task failure during high-intensity, constant-load cycling?

### **1.6 Research Hypotheses**

The research hypotheses tested in the four studies that make up this PhD thesis, are as follows:

#### **1.6.1 Study 1 (Chapter 3)**

- i. Peripheral fatigue will occur earlier in the high-intensity, constant-load cycling bout, whereas factors related to central fatigue will develop later, towards exhaustion.
- ii. There will be both an increase in the degree of lower limb muscle activation, and an alteration in the timing of this muscle activity, during the high-intensity, constant-load cycling bout.

#### **1.6.2 Study 2 (Chapter 4)**

- i. The development of neuromuscular fatigue, including changes in lower limb muscle activity, will precede changes in joint kinematics.

- ii. Changes in joint kinematics will be observed at the trunk segment, and at the hip, knee and ankle joints in the sagittal plane, in addition to changes at the knee joint in the coronal plane.

### 1.6.3 Study 3 (Chapter 5)

- i. Limiting kinematic variation in trunk flexion, trunk medio-lateral sway and hip abduction/adduction through the use of real-time, visual kinematic feedback will either have no effect on, or be detrimental to, the time taken to reach task failure during high-intensity, constant-load cycling.

### 1.6.4 Study 4 (Chapter 6)

- i. There will be significant negative relationships between joint kinematics measured during high-intensity cycling in both a non-fatigued ( $T_0$ ) and fatigued ( $T_{100}$ ) state and at least some (e.g.  $W_{peak}$  and cycling economy) physiological attributes that are considered indicative of successful cycling performance.
- ii. There will be significant negative relationships between joint kinematics and the time taken to reach task failure during high-intensity, constant-load cycling.

## 1.7 Limitations

A number of limitations exist in the methods employed in the studies of this thesis. In Study 1, neuromuscular function was measured during both involuntary (i.e., electrically stimulated) and voluntary contractions of the quadriceps performed following exercise. A short time delay was present between exercise cessation and the initial neuromuscular assessment. This is a common limitation observed in research examining mechanisms of neuromuscular fatigue (10, 11, 13-15, 166, 240, 254, 275). While some recovery will occur during this time period, the methodology in the present

study allowed for the temporal pattern of neuromuscular fatigue to be examined in detail throughout the exercise bout.

In Studies 1 and 2, muscle activation and joint kinematic changes were assessed from 10 consecutive pedal cycles at distinct time intervals throughout the high-intensity, constant-load cycling bout. This was done because of the high volume of data recorded by three-dimensional (3D) motion analysis systems (Vicon Motion Systems, USA). As such, kinematic changes in this thesis were only assessed at 20% time intervals (0, 20, 40, 60, 80 and 100% of the time taken to reach task failure;  $T_0$ - $T_{100}$ ) and may not capture minor fluctuations in movement that could have occurred during the exercise task. Despite this, the methods employed in these studies still allowed for temporal descriptions of changes in joint kinematics. Similarly, previous reports have also examined neuromuscular fatigue at 20% time intervals during a knee extension/flexion task (112), and changes in muscle activation (36, 88, 91, 185) and joint kinematics (37, 38, 266) from 10-30 consecutive pedal cycles at specific time points throughout cycling bouts.

To examine the relationship between physiological attributes indicative of successful cycling performance and joint kinematics in Study 4, two different exercise tests were employed. In order to assess the physiological attributes of cyclists (i.e.  $W_{peak}$ ,  $VO_{2max}$ , metabolic thresholds (i.e. lactate or ventilation thresholds) and cycling economy) an incremental test protocol was performed. However, in order to be consistent with the previous studies of this thesis, and given the difficulty in recording 3D joint kinematic data and ventilatory responses simultaneously, changes in joint kinematics were measured during a constant-load cycling test. Therefore, the directionality of associations observed in this study could not be determined. However, the results revealed novel insights regarding the interdependence between lower limb joint kinematics and both the physiological attributes of cyclists and the time taken to reach task failure during high-intensity cycling in well-trained male cyclists.

Finally, participants performed constant-load cycling tests in all studies of this thesis. A constant intensity exercise test was performed so that the temporal pattern of neuromuscular fatigue mechanisms could be closely examined. In addition, a constant intensity exercise test allowed for fatigue-induced changes in muscle activation and joint kinematics to be assessed compared to a self-paced trial, where such changes may have resulted from fatigue and/or variations in exercise intensity. Further research examining the progression and mechanisms associated with fatigue during self-paced exercise is warranted.

## 1.8 Delimitations

Only well-trained male cyclists were included in the studies of this thesis. Reasons for this were that: (i) well-trained individuals would exhibit consistent muscle recruitment and kinematic patterns (56, 57, 59), (ii) neuromuscular fatigue measurements during involuntary (i.e., electrically stimulated) and voluntary contractions of the quadriceps would not be affected by the reproductive hormone oestrogen; the effect of changing oestrogen levels during the menstrual cycle may influence the force generating capacity of skeletal muscle in females (262), and (iii) central and peripheral fatigue develops differently in men and women during cycling (126). Therefore, the results reported within this thesis are only directly applicable to this subject population. In addition, the number of kinematic feedback conditions in Study 3 (Chapter 5) was delimited to three. This was chosen as it would allow for the investigation of altered joint kinematics on task performance, but still allow participants to cope with the degree of motor learning involved.

1		<b>1.9 Definitions of Selected Terms</b>
2		
3	3D:	Three-dimensional
4	ANOVA:	Analysis of variance
5	BF:	Biceps femoris
6	$\text{Ca}^{2+}$ :	Calcium
7	CNS:	Central nervous system
8	$\text{CT}_{\text{twitch}}$ :	Twitch contraction time
9	E-C:	Excitation-contraction
10	EMG:	Electromyography
11	$\text{EMG:M}_{\text{max,MVC}}$ :	EMG to M-wave ratio
12	$f_{\text{mean}}$	Mean frequency
13	$f_{\text{median}}$	Median frequency
14	GL:	Gastrocnemius lateral head
15	GM:	Gastrocnemius medial head
16	GMax:	Gluteus maximus
17	$\text{H}^{+}$ :	Hydrogen ion
18	$\text{HR}_{\text{max}}$ :	Maximal heart rate
19	MAP:	Maximal aerobic power
20	$\text{M}_{\text{dur}}$ :	M-wave peak-to-peak duration
21	MFI:	Multidimensional fatigue inventory
22	$\text{M}_{\text{max}}$ :	M-wave peak-to-peak amplitude from resting twitch
23	$\text{M}_{\text{max,MVC}}$ :	Superimposed M-wave peak-to-peak amplitude during MVC
24	MVC:	Maximal voluntary contraction
25	M-wave:	Maximal compound muscle action potential
26	$\text{P}_i$ :	Inorganic phosphate
27	$\text{P}_{\text{max}}$ :	Power output at $\text{VO}_{2\text{max}}$
28	REI:	Rate of EMG increase
29	RF:	Rectus femoris
30	ROM:	Range of motion
31	RPE:	Rating of perceived exertion
32	$\frac{1}{2}\text{RT}$ :	One-half relaxation time of twitch torque
33	rpm	$\text{Revolutions} \cdot \text{min}^{-1}$
34	$\text{RTD}_{\text{MVC}}$ :	Rate of torque development during MVC

1	RTD <sub>twitch</sub> :	Rate of twitch torque development
2	RTR <sub>twitch</sub> :	Rate of twitch torque relaxation
3	SD:	Standard deviation
4	SOL:	Soleus
5	T <sub>0</sub> :	Start of exercise
6	T <sub>20</sub> :	20% of the time taken to reach task failure (i.e. T <sub>100</sub> )
7	T <sub>40</sub> :	40% of the time taken to reach task failure (i.e. T <sub>100</sub> )
8	T <sub>60</sub> :	60% of the time taken to reach task failure (i.e. T <sub>100</sub> )
9	T <sub>80</sub> :	80% of the time taken to reach task failure (i.e. T <sub>100</sub> )
10	T <sub>100</sub> :	Time taken to reach task failure i.e., voluntary termination of
11		exercise
12	TA:	Tibialis anterior
13	TDC:	Top-dead-centre
14	T <sub>p,MVC</sub> :	Peak MVC torque
15	T <sub>p,twitch</sub> :	Peak twitch torque
16	TTE:	Time to exhaustion
17	TTE <sub>Tflex</sub> :	Time to exhaustion test with trunk flexion feedback
18	TTE <sub>Tsway</sub> :	Time to exhaustion test with trunk medio-lateral sway feedback
19	TTE <sub>Habd/add</sub> :	Time to exhaustion test with hip abduction/adduction feedback
20	VCO <sub>2</sub> :	Carbon dioxide production
21	VE:	Ventilation
22	VL:	Vastus lateralis
23	VM:	Vastus medialis
24	VO <sub>2</sub> :	Oxygen consumption
25	VO <sub>2max</sub> :	Maximal oxygen consumption
26	VT <sub>1</sub> :	First ventilatory threshold (aerobic threshold)
27	VT <sub>2</sub> :	Second ventilatory threshold (anaerobic threshold)
28	%VA:	Percent voluntary activation
29	W <sub>peak</sub> :	Peak power output
30		
31		



## CHAPTER TWO

### Review of Literature

The present review of literature provides information relevant to the studies of this thesis. The main topics discussed include the time course and mechanisms of exercise-induced neuromuscular fatigue as well as the strategies employed by the neuromuscular system to combat fatigue during exercise tasks, with a focus on cycling. The importance of movement variability during exercise tasks is also subsequently discussed.

#### 2.1 Introduction

Neuromuscular fatigue can be defined as an inability to maintain a given force or power level and has been hypothesised to have both central and peripheral origins (6, 117, 294). Central fatigue occurs within the CNS at cortical and spinal levels, and is defined as a progressive exercise-induced reduction in the voluntary activation of skeletal muscle (117, 294). Peripheral fatigue is defined as any decline in muscle performance that occurs with alterations occurring at or distal to the neuromuscular junction (6, 117). Neuromuscular fatigue is therefore caused by multiple processes within both the CNS and peripheral musculature and is an inevitable process that occurs during exercise (6, 117, 294). As fatigue develops, it is speculated that the human neuromuscular system must adapt to these changes by altering muscle activity and movement technique (i.e., joint kinematics) in order to increase movement variability and maintain task performance. The purpose of this review is to i) provide a brief overview of the known mechanisms underpinning neuromuscular fatigue, (ii) discuss the role and importance of movement variability during fatiguing exercise tasks, (iii) examine the literature pertaining to possible muscle activation and kinematic compensatory strategies utilised by the neuromuscular system during fatiguing exercise tasks, and (iv) examine the association between movement biomechanics (i.e., kinetics and kinematics) and the physiological attributes indicative of an individual's ability to resist fatigue.

## 2.2 Mechanisms Underpinning Exercise-Induced Neuromuscular Fatigue

The mechanisms responsible for exercise-induced neuromuscular fatigue have been investigated by scientists for more than a century (144, 217, 251). In the early 1900s, fatigue was thought to occur as a result of metabolic limitations or the formation of ‘fatigue biproducts’ in the peripheral musculature (144, 217, 251). Even in these early stages of neuromuscular fatigue research, central factors related to ‘brain fatigue’ (217) were also recognised as important contributors to the ‘fatigue point’ (251). This point of fatigue, when an individual can no longer complete an exercise task, has more recently been referred to as the point of exhaustion or task failure. While our knowledge of the factors influencing fatigue development has drastically improved, the precise mechanisms responsible for exercise-induced fatigue and task failure remain unclear. This uncertainty results from the complex processes involved in muscle contraction and exercise-induced fatigue. Voluntary skeletal muscle contraction occurs via the activation of the motoneurone pool, through synaptic inputs provided by descending cortical pathways, spinal interneurons and afferent feedback from the periphery. To initiate the contraction process, action potentials are generated in corticospinal neurones, and sent to the specific motor units recruited within the skeletal muscle. Briefly, once the action potential reaches the neuromuscular junction it propagates into the transverse tubular system. This causes a rapid depolarisation and activation of dihydropyridine receptors, which results in calcium ( $\text{Ca}^{2+}$ ) release from the  $\text{Ca}^{2+}$  release channels (ryanodine receptors) located in the terminal cisternae of the sarcoplasmic reticulum. Action potential-induced  $\text{Ca}^{2+}$  release allows for the formation of cross-bridges, and results in muscular contraction. Following this,  $\text{Ca}^{2+}$  is then resequestered into the sarcoplasmic reticulum and the muscle then relaxes; this process is known as excitation-contraction (E-C) coupling.

The process of fatigue is complicated because it can occur at any point within the chain of neuromuscular events from the motor cortex to the contractile proteins within the muscle fibres. Furthermore, multiple factors can influence muscle output and fatigueability during exercise and thus the following factors need to be considered: muscle perfusion; core body temperature; environmental conditions; the competing needs of physiological systems; and the psychological and emotional characteristics of the individual, including arousal, motivation and both tolerance to pain and sensitivity to stress (117). Although these are important concerns in fatigue research, these are beyond the scope of the current review.

Irrespective of the precise mechanisms responsible for fatigue, it is acknowledged that fatigue either occurs within the CNS at cortical and spinal levels (i.e. central fatigue), or distal to the neuromuscular junction (i.e. peripheral fatigue) (6, 117, 294). Indeed, several theories exist with regards to the location along the chain of events where failure or deficient occurs, including the ‘neuromuscular propagation’ and ‘peripheral failure’ theories, as well as the ‘central activation failure’ theory (1). The neuromuscular propagation and peripheral failure theories hold that peripheral mechanisms (i.e., alterations at the sarcolemmal or action-myosin levels) are involved in the reduced ability of a muscle to respond to neural input and thus to produce force (1). The central activation failure theory suggests that the diminished ability of muscle to produce force is related to a reduction in neural drive, which may be controlled by a governor located within the CNS (1). While the relative contribution of these mechanisms in the development of exercise-induced fatigue is widely debated, it is likely that both peripheral and central factors, and the interaction of these factors, are involved in neuromuscular fatigue development.

### **2.2.1 Peripheral fatigue**

Peripheral fatigue is defined as any decline in muscle performance occurring at or distal to the neuromuscular junction (6, 117). Numerous mechanisms at the muscle level can underpin peripheral fatigue and these will be discussed hereafter. At the onset of muscular contraction, phosphocreatine and adenosine tri-phosphate are hydrolysed resulting in an immediate increase in inorganic phosphate ( $P_i$ ) (47, 48, 51, 72). As exercise continues, anaerobic glycolysis becomes the primary energy source resulting in an increase in lactate and hydrogen ions ( $H^+$ ) (i.e. low muscle pH) (258). Contrary to early belief (for review see, (108)), lactate has been reported to have a minimal effect on force production and thus is not considered to be directly related to muscular fatigue (6). Earlier experiments were performed on muscle fibres at non-physiological temperatures and results indicated a close association between the accumulation of both lactate and  $H^+$  and the decline in muscle performance (6, 108). However, recent findings from experiments performed on muscle fibres at physiological temperatures (i.e.  $\sim 30^\circ\text{C}$ ) have shown that increases in metabolites ( $P_i$  and  $H^+$ ), but not lactate, affect fatigueability during exercise (6, 108). Exercise-induced increases in  $H^+$  and  $P_i$  affect multiple sites in muscle cells, which can ultimately lead to inefficient E-C coupling during contraction. These metabolites (i) impair sarcoplasmic reticulum  $Ca^{2+}$  release ( $P_i$ ) (4-6, 92, 113,

114); (ii) decrease myofibrillar  $\text{Ca}^{2+}$  sensitivity (4, 5, 109); (iii) decrease fibre shortening velocity ( $\text{H}^+$ ) (102, 109, 202); (iv) increase fibre relaxation rates (5, 6); and (v) activate group III and IV muscle afferents ( $\text{H}^+$ ) (76, 77, 116, 193, 275, 293, 317). Consequently, fatigue-induced increases in  $\text{P}_i$  and  $\text{H}^+$  are likely to negatively affect the rate of force development, the force output of active cross-bridges and force relaxation rate (7, 47, 48, 67, 72, 80, 81, 102, 109, 163, 202, 234, 236, 318). Furthermore, since these metabolites act on different myofilament sites their effects are likely to be additive (109, 227).

During exercise, fatigue at the sarcolemmal level takes longer to develop (6, 52). The combined, additive effects of exercise-induced ionic changes in extracellular potassium, intracellular sodium and chloride concentrations can potentially influence action potential transmission, and therefore muscle cell activation and contraction (6, 52). During muscular contraction, the repeated depolarization of the muscle cell leads to an increase in extracellular potassium in the T-tubules and thus an inactivation of sodium channels, which would impact action-potential induced sarcoplasmic reticulum  $\text{Ca}^{2+}$  release (66, 233, 319). However, numerous compensatory mechanisms (e.g. sodium-potassium pump activation (64-66), chloride leakage channels (237, 238),  $\text{Ca}^{2+}$  release feedback (6) and motor unit discharge rates (28, 31, 141)) tightly regulate extracellular potassium levels during exercise. As a result it has been suggested that cellular ionic changes are not likely to be responsible for the development of peripheral fatigue during high intensity exercise in humans (155). Indeed, such changes are typically only observed after prolonged bouts of strenuous exercise (i.e., 2-4 h) (10, 11, 13, 14, 175, 176, 254). Coupled with the other metabolic changes that transpire within the muscle fibre itself ( $\text{P}_i$  and  $\text{H}^+$ ), such ionic changes will accelerate peripheral fatigue development. However, considering the research together, it appears that alterations in muscle excitability are not as important as changes in cellular  $\text{P}_i$  accumulation and muscle acidosis (elevated  $\text{H}^+$  levels).

### 2.2.2 Central fatigue

Central fatigue is defined as a progressive exercise-induced reduction in the voluntary activation of skeletal muscle (117, 294). The inability to drive muscles voluntarily results from fatigue-induced processes that reduce descending cortical output from the motor cortex, or result in a reduction in the efficacy of output from the motor cortex (117, 118, 295). These processes may include (i) changes to motoneurone

1 intrinsic properties and thus changes in the responsiveness of these motoneurons (116,  
2 117, 193), (ii) modifications to reflex excitatory and inhibitory inputs to motoneurons  
3 (particularly of group III and IV muscle afferents, which respond to fatigue-induced  
4 metabolic biproducts (76, 77, 116, 117, 193, 293)), (iii) changes in cerebral oxygenation  
5 (249), and/or (iv) biochemical changes to neurotransmitters within the CNS such as  
6 serotonin (55, 78, 200, 253), choline (78, 281) dopamine (253) and noradrenaline (253).  
7 During fatiguing muscular contractions, the neuromodulators noradrenaline and  
8 serotonin are released from descending neural tracts and act primarily on the dendritic  
9 regions of motoneurons to generate persistent inward currents (136-139, 226). It has  
10 been suggested that persistent inward currents in spinal motoneurons also act to  
11 increase motoneurone excitability (27, 139, 188), amplify excitatory synaptic input  
12 (137, 139, 170, 171, 188, 287) and alter motoneurone firing patterns (137, 139).  
13 However, these effects are proportionate to the level of descending monoaminergic  
14 drive (139). Therefore during exercise, particularly when performed at high-intensities  
15 where monoaminergic levels increase above resting levels (9, 12, 128, 288), it is  
16 possible that the development of persistent inward currents may help to delay  
17 performance declines in the presence of substantial peripheral fatigue. Overall, the net  
18 change in supraspinal drive will depend on the balance of excitatory and inhibitory  
19 inputs, and it is likely that excitatory inputs (i.e., increases in cortical activity and  
20 motoneurone excitability) are compensated for by inhibitory ones (i.e., increases in  
21 intracortical inhibition and down-regulation of afferent feedback from the peripheral  
22 musculature) (97, 139). Thus, the precise mechanisms underpinning the changes in the  
23 CNS during fatiguing exercise are complicated and yet to be fully determined.

24

#### 25 **2.2.4 Interaction between peripheral and central fatigue**

26 It is probable that neuromuscular fatigue development during strenuous exercise  
27 is regulated by a complex interplay between both central and peripheral factors. For  
28 example, it is possible that a negative feedback loop exists between the peripheral  
29 musculature and the CNS such that group III and IV muscle afferents are activated as  
30 peripheral fatigue develops which: (i) facilitate and/or reduce spinal motoneurone  
31 excitability (193, 275); (ii) inhibit motor cortical output cells ‘upstream’ (116, 117, 193,  
32 275); and (iii) project through multiple ascending pathways to subcortical and cortical  
33 centres and thus may affect motivation (272), motor processing (60) and/or learning and  
34 memory (8). For example, during dynamic exercise fatigued-induced increases in

1 sensory feedback from group III and IV muscle afferents have been shown to facilitate,  
2 and thus maintain, motoneurone excitability during an elbow flexion/extension task  
3 (193) and after repeated-sprint cycling (275). As exercise continues and fatigue  
4 develops, however inactivation of fast voltage-gated sodium channels (203), slowing of  
5 the sodium-potassium pump (27, 161) and/or inactivation of persistent inward currents  
6 (27, 136, 188) has also been observed. Such changes can cause an increase in the  
7 motoneurone recruitment threshold, which may contribute to the motoneurone's  
8 inability to maintain its discharge without additional synaptic input (27, 188).  
9 Simultaneously, afferent feedback from muscle receptors and associated pre-synaptic  
10 modulations may also cause inhibitory responses on the spinal motoneurons (139).  
11 Taken together these interactive effects between central and peripheral fatigue act to  
12 increase one's perception of effort and influence behavioural decisions associated with  
13 continuing exercise (8, 116, 275). Thus, the CNS senses peripheral fatigue and  
14 consequently modulates central motor drive to control the extent of further peripheral  
15 fatigue development. Such adaptations in neural drive are presumably made in order to  
16 avoid cellular disturbances beyond sensory tolerance limits, and to ensure that  
17 homeostasis is maintained (10, 11, 13, 14, 50, 118, 132, 193, 275, 283, 295).

18 This limit of peripheral fatigue has been termed by some researchers as an  
19 'individual's critical threshold' or 'sensory tolerance limit' and is dependent upon both  
20 the exercise modality and the individual's perception and/or sensitivity to fatigue, but is  
21 also independent of the exercise task characteristics and the rate of change in  
22 intramuscular metabolic perturbations (9). Numerous reports during the last few years  
23 have revealed a similar degree of peripheral fatigue at the point of exhaustion, and this  
24 is rarely exceeded under normal conditions (9). Amann and colleagues have shown that  
25 the same level of peripheral fatigue exists at the end of a 5-km cycling time-trial  
26 regardless of the level of pre-existing fatigue (induced by constant-load cycling) (10).  
27 However, with the absence of afferent feedback from the exercising lower limbs,  
28 induced by fentanyl injection to selectively block group III/IV sensory information to  
29 the CNS, a greater degree of peripheral fatigue accumulates (14). Accordingly, it is  
30 proposed that exercise is voluntarily terminated once an individual's critical threshold is  
31 reached (during open-loop tasks, when the exercise duration is unknown) or once a  
32 critical rate of fatigue development is reached and the exercise intensity, and thus  
33 central motor drive, is reduced or regulated (during closed-loop tasks, when the exercise  
34 duration is known) under normal exercising conditions (9). In other words, based on  
35 sensory feedback from the periphery, the Central Governor would sub-consciously

1 'choose' to increase skeletal muscle recruitment, or change the pattern of muscle  
2 recruitment to achieve the same level of muscular force (open-loop tasks), or to  
3 decrease the rate of increase in muscle recruitment (closed-loop tasks) (9). However, it  
4 should be noted that the relative importance and limiting effects of afferent feedback  
5 from the locomotor muscles is less significant when exercising in extreme environments  
6 (i.e., at altitude (12) or in the heat (128, 298)) or when faced with debilitating  
7 physiological conditions (i.e., hypoglycaemia (230) or mental stress/fatigue (191))  
8 because of the more substantial threat posed to homeostasis within the CNS (9).

9 A number of models have been developed to describe these possible interactions  
10 between central and peripheral mechanisms of fatigue, including the Central Governor  
11 Model (223, 304) and the Complex Systems Model of Fatigue (168, 224, 283). These  
12 models suggest that exercise is regulated to ensure that homeostasis is maintained at rest  
13 and during exercise, and predicts that humans will exercise with an 'emergency reserve  
14 capacity' to prevent metabolic or cardiorespiratory failure (168, 223, 224, 283, 289,  
15 304). The Central Governor Model, first suggested by Ulmer in 1996 (304) and then  
16 updated by Noakes in 2001 (223), hypothesises that exercise intensity is controlled via a  
17 'Central Governor' located in the CNS. Afferent signals from the periphery are  
18 continually forwarded to the central controller via somatosensory pathways, and the  
19 central controller will alter central motor drive to optimise performance and to avoid  
20 injury or damage to the vital organs based on these inputs and past experiences (132,  
21 223, 304). The Complex Systems Model of Fatigue, proposed by Lambert (168) and St  
22 Clair Gibson and Noakes in 2004/2005 (224, 283), extends upon the Central Governor  
23 Model and implies that muscle fatigue is regulated via the integration of numerous  
24 physiological systems, and that constant feed-forward and feedback loops exist to  
25 monitor and control central motor drive to maintain homeostasis (168, 224, 283). The  
26 key components or extensions of this theory are that (i) knowledge of the exercise end  
27 point is a key controlling variable regulating neural drive, and (ii) fatigue is considered  
28 a sub-conscious sensation (168, 224, 283, 288, 289). As described above central motor  
29 drive is reduced or regulated to ensure the exercise bout is completed within a safe level  
30 of physical functioning when the exercise duration is known. Conversely, in exercise  
31 tasks where the duration is unknown, there is evidence to suggest that the CNS  
32 increases descending neural drive to the muscles to maximise skeletal muscle  
33 recruitment (9), despite the progressive and possible acceleration of peripheral fatigue.  
34 According to the Central Governor Model, an initial pacing strategy cannot be set if the  
35 end point of exercise is not known, which leaves only afferent feedback to regulate



1 power output and thus central motor drive (196). Indeed, increases in neural drive have  
2 been shown during open-loop, constant-load cycling to exhaustion, where significant  
3 increases in knee extensor and flexor EMG amplitudes were observed across the  
4 exercise bout (84, 185). Under normal exercising conditions, the effects of fatigue on  
5 the CNS are task dependant, and prior knowledge of the task duration or the exercise  
6 end point are likely to be key determinants in the regulation of central motor drive.

7 An alternative way of considering the proposed feedback loop between the  
8 peripheral musculature and the CNS is that the level of peripheral fatigue, afferent  
9 feedback and central motor drive will influence the pacing strategy chosen during  
10 closed-loop exercise. These pacing strategies can be thought of as a reflection, or the  
11 physical expression, of the sensation of fatigue (224, 283). Consequently, it is difficult  
12 to examine changes in central motor drive, and thus exercise intensity, without  
13 commenting on the nature of pacing during exhaustive exercise. Pacing strategies are  
14 defined as the variation in speed that occurs during an exercise bout or a period of  
15 physical work via the regulation of energy expenditure in order to reduce fatigue  
16 development and thus enhance performance (143). Therefore, pacing strategies ‘chosen’  
17 by the CNS reflect an integration of both the feed-forward commands in conjunction  
18 with afferent feedback (168, 224, 283, 288, 289). Importantly, the level of sensory  
19 feedback acts to continually reset the pacing strategy ‘chosen’ during exercise to  
20 complete the task within a safe level of physical functioning (168, 224, 283, 288, 289).  
21 Recent evidence has shown that task familiarity and knowledge of the exercise end  
22 point are important regulators of pacing strategies. For example, cycling time-trial  
23 performance has been shown to improve (196, 285), or remain similar (3), for trained  
24 cyclists despite being provided with either incorrect (3, 285) or no (196) distance (3,  
25 196) or power output (285) feedback. In addition, during repeated-sprint running, prior  
26 knowledge of the sprint number to be completed affects the pacing strategy chosen (34).  
27 However, a recent study has shown that incorrect distance feedback during 4-km  
28 cycling time trials is detrimental for performance (197).

29 Other reports have also shown that ratings of perceived exertion (RPE) (288)  
30 and/or pacing (196) strategies ‘chosen’ during cycling time-trial performance become  
31 more aggressive with increased task familiarity and, presumably, confidence in one’s  
32 ability to complete the task. It is commonly observed that an increase in pace can be  
33 achieved in the final 5-10% of exercise during tasks where the duration is known (204,  
34 224). This increase in pace, accompanied by an increase in neural drive, has been  
35 suggested to represent a ‘neural or metabolic reserve’ which is only accessible during



1 the final stages of an exercise bout. An important prediction of the Central Governor  
2 Model is that exercise is always performed submaximally, hence why this  
3 ‘neural/metabolic reserve’ exists. Therefore, it appears that afferent feedback can be  
4 ignored by the CNS in the final stages of an exercise bout (9). Evidence for this neural  
5 reserve has been shown during a 60-min cycling time-trial effort, where 1-min sprints  
6 were dispersed over the exercise bout every 10 min (158). Cyclists were able to increase  
7 neural drive to complete the final sprint at the same power output as the first sprint  
8 (158). Additionally, neural drive to the exercising muscles also increases in the final  
9 stages of 5 km (10, 11, 13, 14) and 40 km (36) cycling time-trials, and 5 km running  
10 trials (229), and is accompanied by a concomitant increase in cycling power output (10,  
11 11, 13, 14, 36) or running velocity (229). It is suggested that individuals can predict the  
12 course profile and access this neural reserve in an attempt to enhance performance at the  
13 end of the exercise bout. This type of pacing strategy has also been observed during 2-  
14 km rowing races, where rowers increased their boat speed in the final 500 m of the race  
15 (119). Overall, these studies highlight that individuals are better able to identify the  
16 metabolic requirements for the remaining duration of the exercise bout and successfully  
17 complete the task within their own physiological limits with increased task familiarity  
18 and certainty of the end point of exercise, and therefore ‘choose’ an effective  
19 pacing/neural drive strategy.

20

#### 21 **2.2.4 Measurement of neuromuscular fatigue during exercise**

22 Another important factor that needs to be considered is that it is difficult to  
23 precisely measure, and thus describe, the exact mechanisms responsible for task failure  
24 in exercising humans. In addition, the divergence in ideas for the role and interaction of  
25 central and peripheral fatigue has made it difficult for researchers to establish a  
26 comprehensive test protocol (i.e. specific methodology) to examine aspects of  
27 neuromuscular fatigue development during exercise. Given this, indirect and non-  
28 invasive techniques have been developed (201, 243). In order to better understand  
29 localised muscle fatigue and advance our knowledge of the mechanisms responsible for  
30 force decrements, studies have examined in vitro intact single skeletal muscle fibres or  
31 the function of specific muscle(s) at a single joint (243). These studies are important  
32 given the difficulty in examining the fatigue effects of whole-body exercise (243).  
33 Indeed, the methods typically used for the assessment of neuromuscular function are  
34 particularly difficult to obtain during exercise given that an individual is often required

1 to change from an ergometer on which the exercise bout is performed to a different  
2 ergometer or test device for neuromuscular assessment (e.g., twitch contractile  
3 properties, muscle membrane properties and isometric maximal voluntary force  
4 (MVC)). An inherent issue with this methodology is the time delay between exercise  
5 cessation and the neuromuscular assessment, which is typically 2-10 min (for examples  
6 see (10, 166, 175, 206, 240)). During this time delay recovery will occur and thus and  
7 measurements of post-exercise fatigue may underestimate the true extent of  
8 neuromuscular fatigue present (112, 243). Therefore, it is implied that the results of  
9 studies examining fatigue effects on single muscle fibres, or muscle(s) at a single joint  
10 allows indications as to the fatigue-induced changes in the neuromuscular system that  
11 occur during whole-body exercise (243).

12         Given the above, neuromuscular fatigue has primarily been determined by  
13 comparing the torque produced during involuntary (i.e., electrically stimulated) and  
14 voluntary contractions of various muscle groups (primarily the knee extensors, but also  
15 the plantar flexors or dorsiflexors after running exercise) at the onset and termination of  
16 whole-body exercise. The majority of research has focused on continuous running and  
17 cycling of 30 min to 8 h (82, 166, 174-178, 206, 207, 209, 242, 248, 255, 256, 259, 275,  
18 296), with the exception of one study that investigated fatigue effects following a 24-h  
19 treadmill run (194). In addition, some studies have also investigated fatigue during  
20 repeated-sprint running (240), skiing (208) and racquet sports (122, 123, 125). All  
21 studies have found decrements in MVC force of -8 to 40% (82, 122, 123, 125, 166,  
22 174-178, 194, 206, 207, 209, 240, 242, 248, 255, 256, 259, 275, 296) and the majority  
23 of studies have also found a reduction in peak twitch torque ( $T_{p,twitch}$ ) elicited by  
24 electrical stimulation of a muscle or nerve, of -9 to 45% (82, 123, 125, 166, 174-178,  
25 194, 207, 240, 248, 256, 275, 296). Depending on the exercise modality and the time  
26 delay between exercise cessation and the neuromuscular assessment, potentiation of the  
27  $T_{p,twitch}$  response has also been observed after prolonged running (206, 242) and skiing  
28 exercise (208). In addition, post-exercise reductions of -9 to 34% in the muscle  
29 compound action potential (M-wave) peak-to-peak amplitude ( $M_{max}$ ) have been  
30 reported (125, 178, 194, 207, 240, 242, 248, 296), indicating a decrease in muscle  
31 membrane excitability and thus action potential transmission, and decrements in the  
32 level of voluntary muscle activation of -3 to 62% have been reported (82, 122, 123,  
33 125, 166, 176-178, 194, 206, 207, 240, 242, 248, 255, 256, 259, 275, 296), suggesting a  
34 reduction in central motor drive. Some studies also report no change in  $T_{p,twitch}$  (122,  
35 209, 255, 259), voluntary muscle activation (208) and/or M-wave characteristics (122,

1 123, 175-177, 194, 206, 255) after exercise. Therefore, the magnitude of exercise-  
2 induced fatigue is likely to be highly dependent upon the duration and type of exercise  
3 task, the intensity at which the task is performed and the type of muscular contraction  
4 involved (205). Although these studies have provided valuable information with regards  
5 to the central and peripheral mechanisms underpinning fatigue development, this  
6 methodology does not allow for temporal descriptions of the fatigue process and  
7 implicitly assumes that the progression to exhaustion occurs relatively constantly across  
8 the exercise duration.

9         Few studies have attempted to assess the time course of exercise-induced central  
10 and peripheral fatigue development during whole-body exercise (82, 176, 242, 255).  
11 During prolonged running exercise, alterations in neuromuscular function (i.e., reduced  
12 knee extensor MVC, voluntary activation and M-wave characteristics) were found only  
13 after the fourth hour of a 5-h running bout (242). Similarly during a 20-km self-pace  
14 run, a significant reduction in knee extensor MVC force was only found in the final 5  
15 km, as well as a reduced level of voluntary activation at exhaustion (255). In that study,  
16 no changes in the function of the contractile elements (twitch properties) or the muscle  
17 membrane (M-wave characteristics) were observed following the running bout (255).  
18 Therefore, this study indicates that reductions in neural drive (i.e., central fatigue) to the  
19 exercising muscle occur later in prolonged running with no notable impairment in  
20 contractile function, and therefore central changes are likely to be responsible for  
21 decrements in voluntary force capacity and ultimately task failure.

22         During prolonged cycling, however, the mechanisms responsible for task failure  
23 and fatigue development differ. Such differences likely relate to the different type of  
24 muscular action involved between these two tasks (i.e., eccentric-concentric muscle  
25 action during running vs. the involvement of the stretch-shortening cycle in cycling).  
26 During 5-h cycling impairments of the contractile elements were observed after the first  
27 hour of exercise whereas reductions in muscle membrane excitability and central motor  
28 drive (voluntary activation and the EMG:M-wave ratio) appeared to occur in the final  
29 hour of exercise (176). Therefore, fatigue occurs at both central and peripheral sites  
30 during prolonged cycling, compared to the preponderance of central fatigue in  
31 prolonged running. However, during short-duration, high-intensity cycling (4-8  
32 intermittent 6-min bouts performed at 80% maximum aerobic power (MAP)) the pattern  
33 of neuromuscular fatigue development differs (82). Measures of contractile function  
34 (i.e.  $T_{p,twitch}$ , time to peak force, and maximal rates of force development and relaxation)  
35 were found to be significantly reduced near the beginning of exercise (within the first

40% of exercise time), but measures of central drive (i.e. voluntary activation) were augmented near exercise termination (82). The authors suggested that the reduced efficacy of E-C coupling observed during exercise was “compensated for” by an increase in central motor drive, as indicated by an increase in the EMG amplitude. Therefore, exercise duration may have differential effects on the CNS and thus the development of central fatigue and/or facilitation at least in exercise tasks such as cycling. The assessment of neuromuscular fatigue during prolonged (3-h) tennis playing has also been investigated with similar findings to cycling in that both central (impaired voluntary activation and modulations in spinal loop properties) and peripheral (impaired twitch contractile properties and M-wave characteristics) factors were linked to decrements in neuromuscular function (122, 125). All of the above studies required the exercise bout to be interrupted in order to assess neuromuscular function and thus are, effectively, repeated-bout protocols. Therefore, it remains unknown whether the temporal pattern of fatigue development and the magnitude of change in central and peripheral fatigue is similar during continuous exercise tasks. Thus, the precise mechanisms underpinning fatigue and factors resulting in task failure during continuous work bouts remains elusive.

### **2.3 Alterations in Neuromuscular Fatigue**

In the presence of fatigue, compensatory alterations occur in the neuromuscular system that increases motor variability. Motor variability refers to the level of intrinsic variability present in all actions controlled by the sensorimotor system (186, 195). Historically, cognitive motor control theorists have considered movement variability as noise or errors in the neuromuscular system when performing a task, and thus variability in movement patterns was viewed as a negative consequence of fatigue and unfavourable for performance (25, 282). In addition, this view of motor variability sees that, with learning, motor variability should be reduced and movement execution will progress towards an ‘optimal’ movement pattern (25). However, more recent research by ecological motor control theorists depicts motor variability as a potentially beneficial adaptation in movement execution and control in regards to delaying fatigue development, alleviating muscular pain, prolonging task performance and reducing potential injury risk (25, 282). Importantly, motor variability allows the neuromuscular system to adjust or adapt to changes in the external environment or physiological systems, i.e. fatigue (25, 86, 252), and permits the individual to discover new,

alternative movement solutions (282). The following sections of this literature review will discuss the effects of neuromuscular fatigue on motor variability with a focus on the changes in muscle activation and joint kinematic strategies employed during fatiguing manual labour and exercise tasks.

### **2.3.1 Motor variability and reorganisation during fatigue**

Motor variability occurs at multiple levels of the neuromuscular system during the execution of a movement task. Even in the early part of the nineteenth century motor control experts, including Bernstein, who proposed the degrees of freedom model, acknowledged these motor adjustments and suggested that the joints involved in the movement of a specific task did not act independently but that they corrected for each other's errors (30). Motor variability is evident in (i) muscle activation magnitude and timing, (ii) joint kinematics, (iii) movement kinetics, (iv) multi-joint coordination, and (v) performance outcomes (282). Such changes can be considered as gross motor variability adaptations that occur at a 'global level' (i.e. those associated with high degrees of freedom during multi-joint tasks; joint kinematics, movement kinetics and multi-joint coordination), whereas specific changes in motor variability also occur at a 'local level' (i.e. motor unit activation). These changes in movement strategies, and thus potential increases in motor variability, are possible given the redundant number of degrees of freedom available in the human neuromuscular system that ensure many joint combinations can be used to complete a motor task (30, 235, 282).

Motor variability is proposed to play a key functional role in preserving task performance by preventing, delaying or alleviating fatigue. Increases in the variation of movement execution will inherently alter the motor strategies employed by the neuromuscular system and it is suggested that these occur with the purpose of preserving task performance and/or the task goal (120, 282). In manual labour tasks such as repetitive hammering (69, 71) and sawing (70) changes in movement patterns occur to maintain performance; in particular end-point and movement trajectories are preserved despite fatigue. Similarly, in exercise tasks such as repetitive hopping (42) and throwing (146) aspects of the motor skill important to performance or the task goal (i.e., hopping amplitude and frequency or throwing accuracy) are maintained with the adoption of alternative motor, or compensatory, strategies during fatigue. Collectively, these studies indicate that increasing motor variability can potentially prolong performance. Despite this, the potential to deliberately increase kinematic variability to

1 delay the progression of fatigue and thus to enhance task performance has not been  
2 addressed in the literature. This may partly be because changes in movement patterns  
3 (i.e., increased motor variability) in basic accuracy tasks (154, 210) have been  
4 associated with a decrease in performance with fatigue.

5 In all of these studies an increase or decrease in the variability of one joint may  
6 be offset by another, and thus the neuromuscular system utilises the available degrees of  
7 freedom in order to perform the task successfully. During repetitive hammering, sawing  
8 and throwing, where the arm and upper body are predominantly involved, changes at  
9 the elbow joint that occur with fatigue are typically compensated for by increases in the  
10 contribution from the trunk, shoulder and wrist joints (69-71, 146). During repetitive  
11 load lifting, decreased contributions at the hip and knee joints during fatigue are  
12 compensated for by an increase in trunk movement (279, 280, 300). Therefore, it is  
13 clear that movements are reorganised by the neuromuscular system, and thus  
14 compensatory motor strategies employed, during fatigue in these tasks.

15 Recent research indicates that the magnitude of motor variability may also  
16 depend on the site of muscular fatigue. Some studies have investigated the  
17 compensatory adjustments that occur when local muscular fatigue is induced (i.e.,  
18 fatigue of the muscles that act at a specific joint) at different joints involved in  
19 movement execution. For example, muscular fatigue was induced in either the proximal  
20 or distal agonist muscle of the throwing arm during a repetitive throwing task (146).  
21 Wrist movement was maintained by alterations at the elbow joint in the distal fatigue  
22 condition, however in the proximal fatigue condition an increase in wrist joint  
23 contribution was observed (146). Other studies have assessed the effects of different  
24 sites of muscular fatigue during short duration running (e.g. 10-s) (160), or the time  
25 taken to complete five running strides (61)). Local fatigue of the foot invertors had no  
26 effect on joint contributions to the running stride; however there was an effect on the  
27 magnitude of the push-off and impact forces generated (61). With local fatigue of the  
28 dorsiflexors an increase in ankle dorsiflexion at heel strike was observed (61). In  
29 addition, an increase in knee flexion during the swing phase of the running stride was  
30 reported when fatigue was localised to the ankle joint (160). Conversely, fatigue at the  
31 knee joint increased knee flexion during the stance phase of the running stride, as well  
32 as both hip and knee flexion and angular velocity of the leg during the swing phase  
33 (160). Therefore, the compensatory strategies employed by the neuromuscular system  
34 may depend on where in the kinetic chain the fatigue occurs.



1           Given the above, it appears that fatigue triggers a restructuring of the inter-  
2 segmental organisation of the neuromuscular system. Thus, through the use of global  
3 and local motor variability strategies, the neuromuscular system may utilise previously  
4 redundant components of the neuromuscular system when fatigued, in order to maintain  
5 the basic characteristics of the motor skill being performed (146). It is likely that the  
6 task characteristics and the physiological capacity of the individual performing the task  
7 to cope with fatigue (45) will determine the magnitude of motor variability, the motor  
8 strategies employed and, subsequently the effect on task performance. Motor variability  
9 may thus be considered a potentially important compensatory strategy utilised by the  
10 neuromuscular system during fatigue.

11

### 12   **2.3.2   Changes in muscle activation with fatigue**

13           As previously indicated, motor variability can occur at multiple levels within the  
14 sensorimotor system. At the muscular level, global and local muscle activation  
15 compensatory strategies can also be observed during performance of a fatiguing task  
16 (199). Global strategies may include (i) load-sharing within and between synergist  
17 muscles (75, 164, 199); (ii) trade-offs between muscles within a synergy acting at a  
18 single joint or between muscles acting across multiple joints (42, 69, 70, 110, 192); (iii)  
19 recruitment of additional muscles that were previously inactive (or less active) prior to  
20 fatigue development, which consequently employs additional degrees of freedom in  
21 order to maintain task performance (42, 69-71, 110, 120, 146) and is considered a  
22 characteristic of a healthy CNS (96); and/or (iv) co-activation mechanisms of flexor and  
23 extensor muscles, which increases joint rigidity (primarily in the distal joint) and has  
24 been suggested to simplify movement control because of a decrease in the number of  
25 degrees of freedom that the neuromuscular system needs to control to perform a motor  
26 task (30, 69, 87, 110, 130, 146, 192, 245, 308). Indeed, fatigue takes longer to develop  
27 during isometric exercise in participants with more variable muscle activation strategies  
28 (i.e., cyclic changes in activation between synergist muscles) in erector spinae (306),  
29 triceps-surae (199, 292) and quadriceps (277, 311) muscle groups. In cycling, variability  
30 of muscle activation patterns are evident throughout the pedal stroke even in highly  
31 trained cyclists (148, 150, 152, 257); however no attempts have been made to  
32 understand the importance or purpose of such variability in such complex motor tasks.

33           In addition, changes within the active muscle itself can occur with fatigue  
34 accumulation. These local muscular strategies may include changes in the motor unit

firing patterns, such as motor unit rotation and/or substitution (6, 32, 117, 193, 199). During prolonged, fatiguing muscular contractions a rotation or temporary substitution between motor units occurs as an attempt to maintain the desired force level (27, 188, 273, 320). A positive consequence of this is that it permits less fatigued motor units to be recruited in order to maintain the desired force level whilst allowing the capacity of previously activated motor units to recover. Such a motor unit recruitment strategy is particularly important given the increase in motoneurone recruitment that is often observed with fatigue (27, 188, 203, 244). Motor unit rotation and/or substitution has been observed during sustained isometric contractions at 5 to 50% MVC within a variety of muscle groups, including the erector spinae (306), knee extensors (277, 313) and triceps-surae (115, 199, 292). These same strategies are also observed in the plantar flexors during dynamic whole-body exercise (309). Specifically in non-fatiguing cycling (30-s blocks of cycling at varying loads and velocities), motor units within the muscle belly of both the medial and lateral gastrocnemii (GM and GL, respectively) and soleus (SOL) are differentially recruited and are dependent upon the load and cadence of the cycling task (309). Therefore, motor unit rotation or temporary substitution is not only a consequence of fatigue but may also be preventative against it.

As a result of the global and local strategies described above, alterations in the level, timing and/or frequency characteristics of the EMG signal are often observed in fatiguing exercise (98, 117). Typically, exercise-induced fatigue results in an increase in the level of EMG activity, a prolonged time period of muscle activity and a decrease in the frequency component of the EMG signal during prolonged submaximal contractions (98, 117). An increase in the EMG amplitude is considered to reflect the recruitment of additional non-fatigued motor units and/or an increase in motor unit discharge rate (94, 117). This increase in the EMG amplitude may also reflect changes within the muscle itself such as prolongation of the intramuscular action potential, which would increase synchronisation of motor unit action potentials (85, 107). However, during multi-joint movements it is likely that an increase in the EMG signal is observed in some, but not all, muscles that contribute to power production. Indeed, during exercise tasks such as cycling, activity in the knee extensors (84, 89, 145, 185, 241, 261, 263, 312), biceps femoris (BF) (89, 291, 312), gluteus maximus (GMax) (88, 89, 312) and adductor magnus and longus (312) typically increases, but activity in GM, tibialis anterior (TA) (89) and rectus femoris (RF) (88) decreases with fatigue accumulation. In some studies, the activities of GL, SOL, and RF have also been shown to remain consistent (89).



1           Another strategy that occurs in an attempt to compensate for fatigue  
2 development is an alteration in the timing of muscle activity. Few studies have  
3 investigated the changes in muscle activation timing during cycling. One study reported  
4 a significant forward shift in the timing of muscle activation with progressive fatigue  
5 development during constant-load cycling (89) and, similarly, during incremental (312),  
6 repeated-sprint (33) and sprint (compared to submaximal cycling) (90) cycling where  
7 duration and timing parameters of muscle activation appear to change with fatigue.  
8 Conversely, other studies have reported no changes in the timing of muscle activation  
9 (162, 261). The discrepancies in findings for both the level and timing of muscle  
10 activity may be explained by the different cycling protocols employed, variations in the  
11 methods for quantifying the level and timing of muscle activation, and/or the specific  
12 characteristics of the participants.

13           In addition, a slowing of the muscle fibre conduction velocity has been observed  
14 with fatigue accumulation (79), and is typically indirectly estimated as a change in the  
15 mean ( $f_{\text{mean}}$ ) or median ( $f_{\text{median}}$ ) frequency of the EMG signal. While EMG frequency  
16 characteristics are not well examined in manual labour tasks, some studies have shown  
17 significant decreases in EMG frequency characteristics for erector spinae (174) and  
18 certain lower limb muscles (i.e. vastus lateralis (VL) (53); GL and BF (87)) during  
19 fatiguing incremental (53) and constant-load (87, 174) cycling to exhaustion. Recent  
20 studies have used multi-channel EMG systems as a more advanced and direct technique  
21 to measure muscle fibre conduction velocity during cycling (106, 185, 267, 270, 284).  
22 With this methodology, researchers have reported a decrease in knee extensor muscle  
23 fibre conduction velocity that is sensitive to changes in load and cadence (106, 267,  
24 284), however other studies have reported no changes (185, 270). These differential  
25 findings may relate to the characteristics of the cycling task and thus the extent of  
26 muscle acidosis and muscular fatigue-induced by the exercise bout, which is evidenced  
27 by the direct relationship between muscle acidosis and muscle fibre conduction velocity  
28 during cycling (270).

29           Numerous compensatory strategies occur at the muscle level, and are observed  
30 between different muscle synergists (global) or within the muscle itself (local). Global  
31 and local muscular compensatory strategies are therefore employed in an attempt to  
32 delay fatigue and prolong task performance. During dynamic whole-body exercise, e.g.  
33 cycling, it is difficult to describe a ‘typical’ pattern of muscle recruitment, given the  
34 differences in cycling protocols used in the research to date and, in particular, the effects  
35 of workload, cadence, body position, seat height, shoe-pedal interface, training status

(for review see (149)) and chain ring shape (220). In particular, and of importance to this review, the development of fatigue interferes with the ability to describe common muscle activation patterns of the lower limb as well. Given this, constant-load cycling protocols have been employed by researchers in an attempt to identify changes in muscle activation during fatigue, in order to eliminate the effects of changes in exercise intensity, i.e., changes in power output and/or cadence. The majority of studies cited above have used somewhat fatiguing exercise protocols, but the true effects of fatigue development itself can only be investigated during constant-load tests. At present, only a few studies have investigated alterations in muscle recruitment in fatiguing constant-load cycling protocols (80 or 100% MAP to exhaustion) (82, 84, 87). Thus, our knowledge is limited in regards to the effects of physiological fatigue on changes in muscle activation during cycling.

### **2.3.3 Changes in joint kinematics with fatigue**

Fatigue-induced compensatory adjustments are also evident in the changes that occur in joint kinematics (i.e., the movement patterns) of an exercise task. Similar to muscle activation strategies, changes in joint kinematics presumably occur to maintain task performance and may result from alterations in muscle activation and thus the development of muscular fatigue (146). In other words, it is likely that an individual will change their movement pattern, i.e. kinematics, to limit the use of specific muscle(s) groups in an attempt to continue task performance when they become fatigued and/or experience localised muscular fatigue. The majority of research to date, however, has not documented the time course of such changes and thus there is little evidence as to which compensatory strategies (i.e., joint kinematics vs. muscle activation) occur first and, thus, which one influences the other. However, the few studies that have attempted to examine this have reported that muscle fatigue (i.e., decreases in the EMG  $f_{\text{mean}}$  (326) and  $f_{\text{median}}$  (87)) precedes changes in gait rhythm (326) and lower limb joint angles or joint range of motion (ROM) during fatiguing cycling (87), respectively. Thus, it is likely that kinematic adjustments occur secondary to, and possibly as a consequence of, muscular fatigue. Regardless, significant compensatory kinematic adjustments occur with fatigue development and these have been observed during numerous exercise tasks. Nonetheless, it is difficult to gain an understanding of the kinematic adjustments that occur during different types of exercise tasks given that (i) the changes in kinematics are likely to be dependent upon, and specific to, the

1 exercise modality, (ii) the extent of fatigue resulting from the exercise bout and/or  
2 induced by previous exercise prior to task performance varies, and (iii) there is a variety  
3 of kinematic variables examined.

4 Gait-based exercise tasks such as skiing, walking and running have  
5 predominately been investigated. In sprint cross-country skiing, length parameters of  
6 the skiing movement (stride, pole and gliding length) remain stable, whereas velocity  
7 parameters including stride, pole and gliding velocity and hip flexion velocity, as well  
8 hip flexion and trunk extension decreased with fatigue development (330). Thus,  
9 changes in trunk and hip movements were associated with fatigue development and may  
10 have contributed to the decrease in skiing performance (330). In exhaustive skiing,  
11 however, an increase in inter-cycle variability of arm and leg angular displacement was  
12 associated with fatigue (63). These findings are similar to prolonged walking (3-h),  
13 where an increase in gait rhythm variability is observed with fatigue development (326).  
14 The majority of research in running has focused on injury prevention, given that  
15 muscular fatigue may reduce the shock absorption capacity of the lower limb and in turn  
16 alter running kinematics. In exhaustive running bouts, decreases in ankle dorsiflexion  
17 (61, 83, 93, 95, 121, 160) and increases in knee flexion (160, 212) at heel strike, as well  
18 as increases in rear foot motion (ankle pronation) (61, 83, 93, 95, 121) and angular  
19 velocity of the shank (83, 212) have been shown to occur with fatigue. Likewise,  
20 increases in hip and knee extension at toe-off are observed (133, 160), which can also  
21 affect running stride characteristics (133, 167, 211). In addition, variations in hip and  
22 knee joint angles are also observed with progressive fatigue (157). These kinematic  
23 changes (particularly an increase in ankle dorsiflexion and knee flexion) likely occur as  
24 a safety mechanism to reduce the ground reaction forces and thus improve energy  
25 absorption in the lower limb when fatigued (2, 157, 160, 212). Conversely, other studies  
26 have failed to find any relationship between fatigue magnitude and leg kinematics (2,  
27 222), which is suggested to be due to the variation in running styles within and between  
28 participants (222) or the differences in running task characteristics and/or research  
29 methodologies (2). It should also be noted that changes in joint kinematics with fatigue  
30 development in running may be affected by the running protocol employed (i.e. graded  
31 vs. constant-load tests, and/or differences in running speed or surface incline), and thus  
32 these factors should be considered when interpreting these findings. Regardless, the  
33 relationship between fatigue magnitude and changes in running kinematics remains  
34 unclear.

Kinematic adjustments have also been reported in sport-specific movements. In baseball pitching, changes in shoulder, elbow and trunk positioning are commonly reported during the arm cocking and acceleration phase or as foot contact occurs during the pitch (100, 219). Changes in the degree of knee extension at ball release (219) in response to prolonged play (219) or muscular fatigue (100) have also been reported. The coordination of the lower limb during a soccer kick is affected by fatigue, where reduced angular velocity of the shank (18, 159) and/or foot/toe (18, 172) accounts for a significantly slower ball velocity (18, 159, 172). All of these studies indicated that muscular fatigue impaired the ability of the kicking leg to produce force, which altered the biomechanics and inter-segmental coordination of the kick (18, 159, 172). Fatigue-induced adjustments in the kinematics of a free-throw (305) or long shot (99) in basketball have also been briefly investigated. No effects of fatigue were found in college basketball players on trunk, elbow, knee and ankle joint angles before, at, or after ball release, and thus it was suggested that these players could continue to perform coordinated movements despite being fatigued (305). Nonetheless, a case study on an elite basketball player revealed decreases in shoulder and wrist height of the release arm as well as elbow and upper arm joint angles with the progression of fatigue (99). Regardless of the exercise modality, the majority of studies have reported increases in the variability of and/or changes in limb kinematic parameters with fatigue development. Thus, these can be considered as common neuromuscular adjustments made during fatigue that may, in some instances, help to maintain performance.

Despite its popularity, cycling has received less attention in the literature in this regard with few studies examining fatigue-induced changes in cycling kinematics (37, 38, 87, 266). This is somewhat surprising since it is generally believed that an ‘optimum cycling technique’ is associated with improved cycling efficiency and thus enhanced performance (173). During incremental cycling, an increase in ankle dorsiflexion and ROM as well as an increase in hip flexion angle and reduced hip ROM have been observed with increases in workload leading to fatigue (37). Similar findings at the ankle and hip joint have also been reported, as well as an increase in knee flexion angle towards the end of a high-intensity cycling bout (i.e., cycling at 100% MAP) (38). Both of these studies examined the fatigue-induced changes in cycling kinematics at specific time points throughout the exercise bout (37, 38), however another study examined such changes over the entire exercise period and reported increases in trunk forward flexion, knee valgus/varus and ankle dorsiflexion (87). Furthermore, all of the studies investigated the changes in joint kinematics occurring throughout the entire pedal

1 revolution. An alternative approach is to only examine the changes in joint kinematics  
2 during the drive or power production phase of the pedal stroke. Using this approach  
3 Sayers and colleagues (2012) reported an early decrease in ankle joint ROM along with  
4 a later increase in mean tibial rotation during a prolonged (60 min) cycling time-trial  
5 (266). Despite these findings, there is still limited information pertaining to both the  
6 sagittal and coronal plane kinematic changes that occur during fatiguing, high-intensity,  
7 constant-load cycling. Regardless of the kinematic changes that occur, it is commonly  
8 suggested that cycling technique modifications are made in an effort to cope with the  
9 level of fatigue experienced, alter muscle recruitment strategies and/or improve the  
10 application of force to the pedal in an effort to continue cycling performance.

11

## 12     **2.4     Relationships between Physiological Attributes of an Individual and** 13     **Biomechanical Alterations in Response to Neuromuscular Fatigue**

14         Successful performance of an exercise task is typically determined by a variety  
15 of factors, including race tactics, the exercising environment, movement biomechanics  
16 and the physiological attributes and psychological characteristics of the athlete (21, 103,  
17 104). Given this, it is assumed that an individual's ability to resist fatigue whilst  
18 maintaining desirable movement biomechanics is largely dependent upon the  
19 characteristics of both the individual and the exercise task (11, 205). The relationship  
20 between an individual's physiological attributes and movement biomechanics (i.e.,  
21 kinetics and kinematics) has been investigated in a number of sports, including running  
22 (167, 222, 228, 286, 322), alpine skiing (268) and swimming (24, 68, 239, 328). In  
23 short-duration running (8 min), more efficient runners tended to run with a greater trunk  
24 lean, although a high variability in movement technique (i.e., joint kinematics) and  
25 physiological responses of the runners was reported (322). In addition, the study  
26 reported that 54% of the inter-individual variability in running economy could be  
27 explained by between-subject variability in kinematic factors (322). Conversely, other  
28 studies have found no relationship between running kinematics (hip, knee and ankle  
29 angular displacements, velocities and/or joint angles) and running economy (54, 134,  
30 167, 222). Also, no relationships were reported between running stride parameters  
31 (stride frequency and length) and running economy (134, 167, 286), even though  
32 significant increases in stride frequency and decreases in stride length were observed  
33 across the running bout (167). However, other studies have reported significant

relationships between the physiological attributes of runners and running kinetics (286, 322). For example, running economy has been correlated with ground contact time (165, 228) and the magnitude of ground reaction forces generated during the running stride (140, 286), as well as submaximal oxygen consumption ( $\text{VO}_2$ ) which has been correlated with peak medial forces in the ground support phase (322). These findings indicate that the kinetic patterns of the running stride may be better related to the physiological attributes of runners than kinematic variables. In skiing, blood lactate concentration was found to be positively related to knee joint ROM in older, recreational alpine skiers, and the skiers' average heart rates were also negatively correlated with mean knee joint angles, although no relationships were found between physiological variables and skiing kinetics (268). These results indicate that muscle recruitment, heart rate responses and substrate metabolism may be influenced by knee joint ROM during alpine skiing (268). In swimming, the energy cost of exercise at a given velocity typically increases with an increase in stroke rate (24, 328) but decreases with an increase in stroke length (24, 68, 239, 328). These results indicate that the kinematics of the stroke influences swimming efficiency/economy, and most probably performance. The above examples provide clear evidence that the physiological attributes of runners, skiers and swimmers are related to movement biomechanics, and therefore it is of importance to understand these relationships for other work- and exercise-related tasks.

In cycling, movement technique (i.e., kinematics) and the efficiency of force application at the pedals (i.e., kinetics) can greatly influence physiological responses, and therefore cycling performance (173). Despite this, few studies have examined the relationships between a cyclist's physiological attributes and their movement biomechanics (73, 173), and both of these studies found no significant associations. During a 1-h cycling trial at lactate threshold, no relationship between cycling ability (i.e., 'elite cyclists' were designated as those with a greater power production, anaerobic threshold and  $\text{VO}_{2\text{max}}$  compared to 'good cyclists') and pedalling effectiveness (i.e., the proportion of force applied to the pedal that results in propulsive torque) was observed (73). In addition, no influence of technique (body orientation/seat position) on the efficiency or effectiveness of pedal force application was reported during repeated-bout cycling at 80% MAP (173). These studies indicate that cyclists who are considered to have better physiological profiles do not necessarily cycle with a more effective or superior technique, and that factors other than body/seat position and/or pedal force application patterns may be more important in regards to cycling



performance. No studies have examined these relationships under conditions of fatigue, which is important given that previous reports have highlighted fatigue-induced changes in cycling kinematics (37, 38, 87, 266). Thus, the research to date does not allow for a clear understanding of whether or not there is an interdependence of the physiological attributes of a cyclist and cycling biomechanics, and in particular during fatiguing exercise conditions.

## 2.5 Summary and Conclusion

Neuromuscular fatigue accumulates in both the CNS and the periphery during exercise, and this can have a detrimental effect on exercise performance. Although it has been well established that the development of neuromuscular fatigue is likely to be dependent upon the characteristics of both the individual and the exercise task, the temporal pattern of fatigue development and the magnitude of change in both central and peripheral fatigue mechanisms during high-intensity, constant-load exercise tasks are not well described in the literature. Thus, the precise mechanisms underpinning neuromuscular fatigue development and the factors resulting in task failure during this type of exercise remain elusive.

Despite the detrimental effects of fatigue on exercise performance, the neuromuscular system appears to adapt to fatigue by utilising a variety of neural (i.e., compensatory) strategies in an attempt to prolong task performance and/or alleviate the effects of fatigue accumulation. Compensatory strategies can modulate the effects of fatigue on task performance and have been observed in regards to alterations in muscle activation, joint kinematics, and the degree of motor variability in numerous manual labour and exercise tasks. Further work is needed to identify the muscle activation and joint kinematic compensatory strategies utilised during high-intensity, constant-load tasks, and the relationship between these strategies and neuromuscular fatigue mechanisms. In addition, given that increases in motor variability have been shown to improve and/or prolong performance, further research is needed to ascertain whether allowing changes in motor strategies, such as joint kinematics, and thus encouraging kinematic variability is beneficial in regards to delaying fatigue accumulation and prolonging exercise performance.

Finally, the physiological capacity of the exercising human and therefore the ability to resist fatigue, as well as the neuromuscular adaptations to fatigue employed (i.e., movement technique), likely interrelate to affect exercise performance. Further

1 research is necessary to determine the relationships between an individual's  
2 physiological attributes and both kinematic and kinetic variables during fatiguing  
3 exercise.  
4  
5



## CHAPTER THREE

### Evidence of peripheral fatigue and up-regulation of central motor drive during high-intensity, constant-load cycling

#### 3.1 Abstract

**Purpose:** The time course and relative contribution of central and peripheral fatigue, and the associated muscle activation changes, were examined during high-intensity, constant-load cycling. **Methods:** Nine well-trained male cyclists performed seven testing sessions, including an incremental cycling test to exhaustion, a time to exhaustion (TTE) test at 90%  $P_{\max}$  (power output at  $VO_{2\max}$ ) at a constant cadence, and tests to 20, 40, 60, 80 and 100% ( $T_{20}$ – $T_{100}$ ) of the time taken to reach task failure in the TTE test ( $5:49 \pm 0:51$  min:s). Neuromuscular function pre- ( $T_0$ ) to post-exercise (i.e.  $T_{20}$ – $T_{100}$ ), and surface electromyography (EMG) of seven lower limb muscles were assessed during cycling. **Results:** During cycling, EMG mean frequencies ( $f_{\text{mean}}$ ), EMG onset and offset times and duration did not change ( $p > 0.05$ ), but peak vastus medialis (38.9% at  $T_{100}$ ) and gluteus maximus (87.2% at  $T_{100}$ ) EMG amplitudes increased ( $p < 0.05$ ). Measured during a maximal isometric knee extension, peak twitch torque ( $T_{p,\text{twitch}}$ ) and the average rate of twitch torque relaxation ( $RTR_{\text{twitch}}$ ) increased from  $T_0$  to  $T_{20}$  (15.0% and 17.0% at  $T_{20}$ , respectively), and the average rate of twitch torque development ( $RTD_{\text{twitch}}$ ) increased to  $T_{40}$  (11.0%), then declined to  $T_{100}$  ( $T_{p,\text{twitch}}$ : –39.9%;  $RTR_{\text{twitch}}$ : –36.7%; and  $RTD_{\text{twitch}}$ : –34.7%) ( $p < 0.05$ ). Twitch contraction time showed a similar progression, with decrements below pre-exercise values from  $T_{20}$  (9.7%) onwards before an increase from  $T_{80}$ – $T_{100}$  (–10.7% at  $T_{100}$ ) ( $p < 0.05$ ). Neither maximal compound muscle action potential peak-to-peak amplitude ( $M_{\max}$ ) and duration, nor twitch one-half relaxation time changed ( $p > 0.05$ ). No changes were observed in voluntary peak torque or rate of torque development ( $p > 0.05$ ). Trends towards increases in vastus lateralis EMG amplitude (5.7% at  $T_{100}$ ) were not significant ( $p > 0.05$ ) and no changes were observed in voluntary muscle activation, peak EMG amplitudes, rate of EMG increases or EMG  $f_{\text{mean}}$  ( $p > 0.05$ ). **Conclusions:** Peripheral fatigue developed early in the exercise bout from 60% of the time to task failure, and central facilitation, rather than central fatigue, occurred towards the end of the high-intensity, constant-load cycling bout near exhaustion. Neuromuscular fatigue was

- 1 compensated for by an increase in the magnitude of lower limb muscle activity as an
- 2 attempt to maintain the required power output of the cycling task.
- 3
- 4

## 3.2 Introduction

Exercise-induced fatigue is a complex biological process involving alterations in neuromuscular function both proximal (i.e. central fatigue) and distal to the neuromuscular junction (i.e. peripheral fatigue) (6, 117, 294). Many manual labour (e.g. hammer or axe use) and sporting or exercise tasks require moderate periods (<10 min) of relatively high intensity work (e.g. >80% of  $\text{VO}_{2\text{max}}$  or power output), so it is of particular interest to understand the factors influencing fatigue development, and ultimately task performance, during such work bouts. Given that the methods required for the assessment of neuromuscular function (e.g. twitch contractile properties, isometric MVC) are difficult to obtain during dynamic exercise, neuromuscular fatigue has primarily been examined at the onset and termination of exercise (243). For instance, studies have shown that exercise can result in reduced MVC (82, 123, 166, 175, 177, 194, 240, 256, 275, 296),  $T_{p,\text{twitch}}$  (82, 125, 166, 177, 178, 194, 240, 256, 275),  $M_{\text{max}}$  (125, 178, 194, 207, 240, 296) and voluntary muscle activation (82, 123, 125, 166, 178, 194, 240, 256, 275, 296). Although these studies have provided valuable information with regards to the central and peripheral mechanisms underpinning fatigue development, this paradigm does not allow for adequate temporal descriptions of the fatigue process, and implicitly assumes that the progression to exhaustion occurs relatively constantly across the exercise duration.

One study has attempted to assess the time course of exercise-induced central and peripheral fatigue development during intermittent bouts of high-intensity, constant-load cycling (4-8, 6-min bouts performed at 80% MAP) (82). Measures of contractile function (i.e.  $T_{p,\text{twitch}}$ , time to peak force and maximal rates of force development and relaxation) were found to be significantly reduced within the first 40% of exercise time, while measures associated with central drive (i.e. voluntary activation) changed only near exercise termination (82). In this study the authors suggested that the reduced efficacy of E-C coupling observed during exercise was compensated for by an increase in central motor drive, as indicated by an increase in the EMG amplitude (82). Nonetheless, it is still unclear whether the temporal pattern of fatigue development and the magnitude of change in central and peripheral fatigue are similar during continuous exercise tasks. Thus, the precise mechanisms underpinning fatigue and factors resulting in task failure during continuous work bouts remains elusive.

In addition to alterations in central and peripheral fatigue development, the timing of muscle activity, the relative contribution of muscles and joint rigidity may

also be altered during dynamic multi-joint exercise (117, 282), presumably in an effort to increase the activation of less fatigued muscles or muscle groups and maintain task performance. For instance, during cycling the activation of lower limb muscles typically increases with fatigue development (84, 89, 145, 185, 241, 263, 291, 312), and the timing of muscle activation occurs significantly later in the crank cycle (89, 90, 312).

In the current study it was hypothesised that peripheral fatigue would occur early in the exercise bout, whereas factors related to central fatigue would develop later, towards exhaustion. In addition, it was hypothesised that the degree of lower limb muscle activity would increase, and that alterations in the timing of muscle activity will occur during cycling. As such, the aims of the present study were to: i) examine the time course of the development, and relative contribution, of central and peripheral fatigue during high-intensity, constant-load cycling, and ii) determine the associated changes in muscle activation (i.e. the degree and timing of muscle activity) in the lower limb during this type of exercise. A novel exercise model that involved measuring neuromuscular function during multiple, constant-load cycling tests of different durations on separate days was used to build a model of fatigue progression to exhaustion.

### 3.3 Methods

#### 3.3.1 Participants

Nine well-trained male cyclists (i.e., club level competitive cyclists (17)) (mean  $\pm$  SD; age  $36 \pm 9$  y, height  $180 \pm 9$  cm, body mass  $77.0 \pm 16.6$  kg,  $\text{VO}_{2\text{max}}$   $55.9 \pm 7.3$   $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) currently cycling  $366 \pm 125$   $\text{km}\cdot\text{wk}^{-1}$  and with  $5 \pm 2$  y of cycling experience volunteered to participate in the study. Experienced cyclists were recruited to ensure that they had superior movement control, reflected in consistent muscle recruitment patterns (56, 57, 59). The participants were asked to avoid the consumption of alcohol, caffeine or other stimulants for 12 h, and to record and follow a similar diet in the 24 h period prior to all testing sessions (Appendix K). Strenuous exercise was also avoided in the 48 h prior to all testing sessions. Prior to testing this study was approved by the institution's Human Research Ethics Committee and procedures conformed to the Declaration of Helsinki (Appendix D). Participants provided informed written consent prior to participation (Appendices E and G).

### 3.3.2 Experimental protocol

The participants performed a total of seven testing sessions (Figure 3.1) which were separated by at least 48 h. The average number of days to complete all testing sessions was  $26 \pm 11$  days. In the first session participants performed familiarisation of neuromuscular assessments and an incremental cycling test to exhaustion. In the following session participants performed a high-intensity constant-load cycling time to exhaustion test (TTE). The five subsequent sessions consisted of constant-load cycling tests to 20, 40, 60 and 80% ( $T_{20}$ – $T_{80}$ ) of the time taken to reach task failure in the TTE test ( $T_{100}$ ) as well as a second TTE test.  $T_{20}$  to  $T_{80}$  were performed in a single-blind and randomised order with the second TTE test always being performed in the last (seventh) session. This was done to create the expectation in the participants that they may have to complete the second TTE test on any given session, which would ensure that their pre-exercise anticipatory response would be the same for each session (302, 303). In addition, it was determined whether improvements in performance and/or physiological adaptations occurred over the testing period; no change in TTE between sessions 2 and 7 was observed (session 2 =  $5:49 \pm 0:51$  min; session 7 =  $6:22 \pm 1:42$  min;  $p = 0.120$ ). Neuromuscular function was assessed before and after (average time between exercise cessation and neuromuscular assessment =  $82 \pm 9$  s) all constant load cycling tests (described below).

To quantify subjective feelings of fatigue, participants completed the Multidimensional Fatigue Inventory (MFI) psychometric questionnaire (278) prior to each testing session (Appendix J). All participants scored  $\geq 12$  for all test sessions ( $17 \pm 1$ ; average general fatigue score), indicating no/minimal ‘general fatigue’, which refers to general expressions/subjective feelings of fatigue (278).

All cycling tests were performed on an electromagnetically-braked cycle ergometer (Velotron, RacerMate, USA), which was configured to resemble the participant’s own bike set-up. The ergometer configuration was kept constant for all testing sessions. The participants also used their own clip-in cycling shoes and pedals.

29

#### 3.3.2.1 Incremental cycling test

Participants warmed up for a minimum of 5 min at  $1.25 \text{ W} \cdot \text{kg}^{-1}$  ( $96 \pm 18 \text{ W}$ ) followed by three 5-10 s maximal sprints with a self-paced active recovery before performing the incremental cycling test. The test started at 100 W, and then increased by 15 W every 30 s until voluntary exhaustion or the cadence dropped below 70 revolutions·min<sup>-1</sup> (rpm). Ventilation, expired gas concentrations and heart rate were

recorded as 15-s averages throughout the test (ParvoMedics TrueOne 2400 diagnostic system, USA; RS800 Polar Heart Rate Monitor, Finland). RPE were measured using Borg's 15 point (6-20) scale (44) every minute. The test was completed at a freely chosen cadence.  $\text{VO}_{2\text{max}}$  (the highest  $\text{VO}_2$ ) reading averaged across two consecutive readings (169)),  $P_{\text{max}}$  (the power output that elicits a  $\text{VO}_2$  reading within  $2.1 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$  of the subsequent reading despite an increase in workload (169)), and the target cadence for subsequent constant-load cycling tests (average self-selected cadence between 10 and 30 s prior to exhaustion) were determined.

### **3.3.2.2 Constant-load cycling tests**

Prior to all constant-load cycling tests (sessions 2-7), participants warmed up as described above. The constant-load cycling tests required participants to cycle at 90% of  $P_{\text{max}}$  and within  $\pm 5\%$  of their target cadence (calculated from the incremental cycling test). The participants were instructed to remain seated for the duration of the test. To ensure they reached their target cadence at test commencement an initial 10 s unloaded period and subsequent 10 s ramp period (10% of 90%  $P_{\text{max}}$  every 1 s) was provided until 90% of  $P_{\text{max}}$  was reached. The participants were instructed to continue cycling until cadence dropped below 5% of their target cadence for longer than 3 s, or they were stopped spontaneously by the experimenter (i.e.  $T_{20}$ – $T_{80}$  tests). The participants were provided with verbal encouragement and visual feedback on cadence throughout the test. RPE was recorded using Borg's 15 point (6-20) scale (44) at the beginning ( $T_0$ ) and end of exercise ( $T_{100}$ ) of the first TTE test, and at completion of the remaining constant-load cycling tests ( $T_{20}$ – $T_{80}$ ).

### **3.3.2.3 Electromyography during cycling**

For all constant-load cycling tests, EMG signals were recorded from VL and vastus medialis (VM), TA, GL, SOL, BF and GMax of the right leg during cycling using pairs of silver chloride surface electrodes (10 mm diameter; Kendall Healthcare, Medi-Trace™ 200 Series, USA) through a wireless EMG system (Zero Wire System, Aurion, Italy). Electrodes were placed according SENIAM guidelines (142). Electrode positions were marked with ink to ensure reliable electrode replacement in subsequent test sessions. EMG signals were sampled online at 2000 Hz using Vicon Nexus software (Vicon Motion Systems, USA). In order to reduce the necessity to collect EMG signals during the full duration of the first TTE constant-load cycling test, data were recorded in the first 20 s (i.e. at  $T_0$ ) and then when RPE increased above 16 and/or

1 cadence approached 95% of the target cadence ( $T_{100}$ ). During the remaining constant-  
2 load cycling tests, EMG signals were recorded during the final 20 s of each test ( $T_{20}$  –  
3  $T_{80}$ ).

4 EMG signals from 10 consecutive pedal cycles at each time point (the first 10  
5 pedal cycles at  $T_0$  and the final 10 pedal cycles at  $T_{20}$ - $T_{100}$ ) were filtered with a 4<sup>th</sup> order  
6 zero-lag band pass (20 - 480 Hz) Butterworth filter (26). A customised four-way  
7 polarity switch attached to the crank with individual sensors on each quadrant was used  
8 to identify 0°/360° (top-dead-centre (TDC)), 90°, 180° (bottom-dead centre) and 270°  
9 crank angles. EMG data for each quadrant of the pedal cycle were time normalised  
10 using cubic splines to a time base of 100 samples. EMG onset and offset crank angles  
11 for individual pedal cycles were used to calculate primary (i.e. a significant EMG burst  
12 common to all participants) and secondary (i.e. smaller EMG bursts observed in only  
13 some participants) bursts of EMG activity (57, 59). Crank angles at which EMG onset  
14 and offset occurred were identified and verified visually from the filtered data. EMG  
15 onset and offset were defined as the points at which EMG amplitude increased  $\geq 3$  SD  
16 or remained  $\leq 3$  SD below the average baseline EMG activity for  $\geq 10\%$  of the crank  
17 cycle, respectively (57, 59, 149). The time elapsed between the crank angles at which  
18 EMG onset and offset occurred (duration of EMG activity), the mean EMG amplitudes  
19 of both primary and secondary EMG bursts, the EMG  $f_{\text{mean}}$  of the primary EMG burst,  
20 and the EMG impulse (EMG amplitude multiplied by the primary EMG burst duration  
21 (1, 47)) were calculated from individual pedal cycles and then averaged for each time  
22 point ( $T_0$ - $T_{100}$ ). In addition, the peak EMG amplitude of the primary EMG burst from  
23 all 10 pedal cycles at each time point ( $T_0$ - $T_{100}$ ) was calculated. Calculations for all data  
24 analyses including those described below were performed in MATLAB (Mathworks  
25 Inc., USA) using custom-built programmes.

26

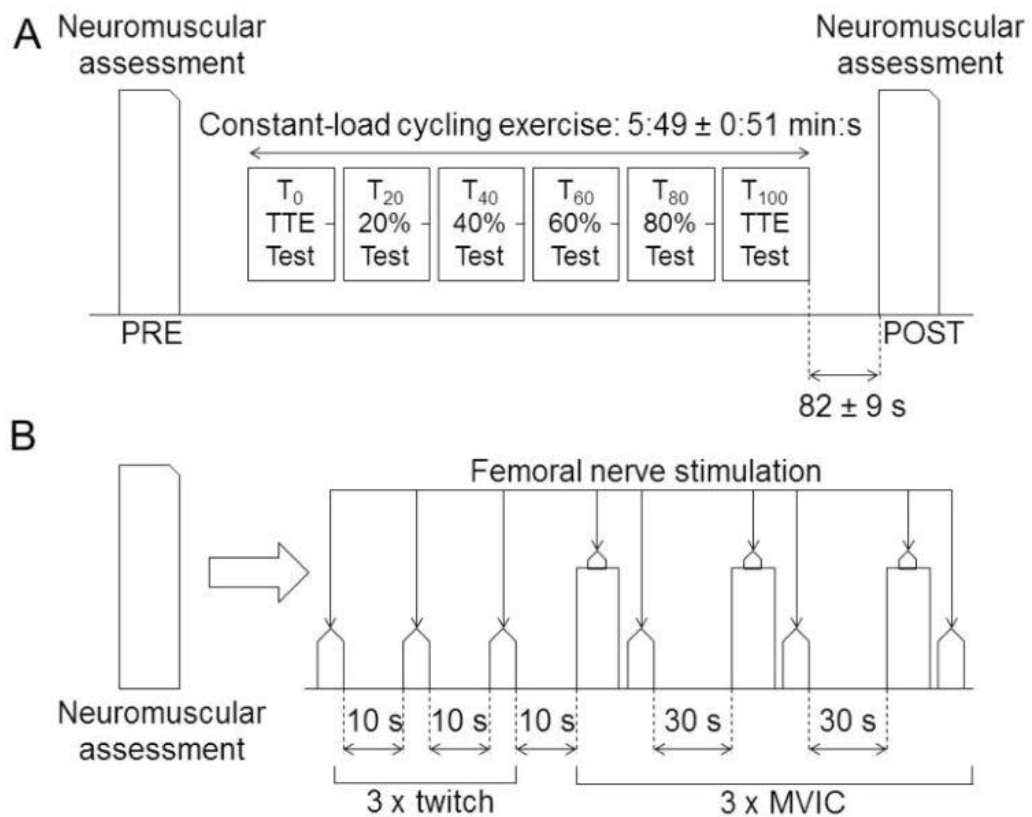
#### 27 **3.3.2.4 Neuromuscular assessment**

28 The neuromuscular assessment protocol is shown in Figure 3.1. Mechanical and  
29 EMG responses of the quadriceps were recorded from muscular contractions elicited by  
30 electrical stimulation of the femoral nerve and then by isometric MVCs. Knee extensor  
31 force was measured via a strain gauge attached to the lever arm of a custom-built chair.  
32 The participants were seated upright with the right hip at 90° flexion and the knee at 60°  
33 flexion (0° = full knee extension). The axis of the knee joint was aligned with the axis  
34 of rotation of the lever arm and the participants were secured with crossover shoulder  
35 and waist straps. The chair set-up was measured and kept constant for all testing

1 sessions. Knee joint torques were subsequently calculated as force multiplied by the  
2 length of the participant's shank from the lateral condyle of the fibula to the point of the  
3 force transducer attachment (approximately 10 cm superior to the lateral malleolus).

4 At the beginning of each test session, the intensity required to elicit a maximal  
5 motor response ( $M_{\max}$ ) by electrical femoral nerve stimulation was established by  
6 performing single stimulations in the relaxed condition using 2-ms (400 V maximum)  
7 rectangular stimuli generated by a high-voltage, constant-current stimulator (Digimeter,  
8 model DS7AH, Hertfordshire, UK). The self-adhesive cathode and anode electrodes  
9 (rectangular 1 x 3 cm; Dura-Stick® II, Chattanooga Group, USA) were placed along the  
10 femoral fold with the middle of the electrode positioned over the femoral artery and on  
11 the lateral side of the hip over the tensor fascia latae inferior to the iliac spine.  
12 Throughout the measurements the cathode was manually pressed into the femoral  
13 triangle, in which lateral pressure was applied to the medial aspect of the femur. This  
14 was shown during pilot testing to elicit more reliable motor responses with the  
15 participant seated upright. Stimulation intensity started at 50 mA and increased in 10  
16 mA steps until a plateau in the  $M_{\max}$  was observed, despite a further increase in  
17 stimulation intensity. After the determination of  $M_{\max}$  and as a warm-up to stabilise the  
18 mechanical properties of the quadriceps (40, 187), the participants completed three 10-s  
19 isometric ramp contractions of the knee extensors and three 3 s MVCs separated by 60  
20 s. Subsequent to this warm-up, three single supra-maximal stimuli (120% of  $M_{\max}$ ),  
21 separated by 10 s rest, were delivered while the participant was relaxed. Following this,  
22 participants performed three 3-s knee extensor MVCs separated by 30 s rest. A single  
23 supra-maximal stimulus (120% of  $M_{\max}$ ) was delivered during the torque plateau of the  
24 MVC (superimposed twitch) and repeated 5 s post-MVC (potentiated twitch) to  
25 evaluate voluntary activation. For all MVCs participants were provided with visual  
26 feedback of torque magnitude and were given verbal encouragement. The participants  
27 were instructed to contract as hard and fast as possible, and to hold their maximal force  
28 level as steady as possible during the MVC. The same experimental procedure but  
29 without the warm-up contractions was repeated after exercise cessation.





**Figure 3.1** Schematic representations of the study design and neuromuscular assessments.

(A): The experimental protocol for the constant-load, time to exhaustion (TTE) cycling tests performed at 90% of  $P_{\max}$  (power output at  $VO_{2\max}$ ) and within  $\pm 5\%$  of the target cadence (sessions 2-6). Neuromuscular function was assessed before (pre) and after (post) all tests. (B): The neuromuscular assessment involved three single supra-maximal stimuli (120% of the maximal compound muscle action (M-wave) peak-to-peak amplitude ( $M_{\max}$ )), separated by 10 s rest, which were delivered while the participants were relaxed (twitch) followed by three 3-s knee extensor isometric maximal voluntary contractions (MVCs) separated by 30 s rest. A single supra-maximal stimulus (120% of  $M_{\max}$ ) was delivered during the torque plateau of the MVC (superimposed twitch) and repeated 5 s post-MVC (potentiated twitch) to evaluate voluntary activation.

For all neuromuscular assessments, EMG signals and force data were synchronously recorded at a 1000 Hz analogue-digital conversion rate using PowerLab hardware (PowerLab System, ADInstruments, Australia) to a computer running LabChart Pro v.7.1 software (PowerLab System, ADInstruments, Australia). EMG signals resulting from maximal femoral nerve stimulation and MVCs were recorded from the right VL using a pair of silver chloride surface electrodes (10 mm diameter; Kendall Healthcare, Medi-Trace™ 200 Series, USA). The cathode electrode was placed directly proximal and the anode directly distal to the electrodes that were previously positioned to record electrical signals during the cycling exercise (described above). The

reference electrode was placed on the right lateral condyle of the fibula. EMG and nerve stimulation electrodes and cables remained attached during all constant-load cycling tests.

From the three stimuli delivered in the relaxed condition,  $T_{p,twitch}$ , twitch contraction time ( $CT_{twitch}$ ; the time elapsed between twitch onset, defined as a torque increase  $\geq 0.5$  Nm, and  $T_{p,twitch}$ ), one-half relaxation time of twitch torque ( $\frac{1}{2}RT$ ; half the time elapsed between  $T_{p,twitch}$  and twitch offset, defined as the torque decrease to  $\leq 0.5$  Nm), and both the average rate of twitch torque development ( $T_{p,twitch}/CT_{twitch}$ ;  $RTD_{twitch}$ ) and relaxation ( $T_{p,twitch}/\frac{1}{2}RT$ ;  $RTR_{twitch}$ ), measured as the greatest and least values of the first derivative of the torque signal, respectively, were calculated as the average response of the three stimuli delivered at rest prior to MVCs. In addition, the  $M_{max}$  and peak-to-peak duration ( $M_{dur}$ ) of the VL M-wave were calculated as the average response of the three stimuli delivered at rest.

From an averaged response of the three MVCs, the peak torque recorded in the 500 ms prior to the superimposed twitch ( $T_{p,MVC}$ ), the rate of torque development ( $RTD_{MVC}$ ) measured from baseline (defined as a torque increase  $> 5.0$  Nm) to early (0-50 ms), middle (100-200 ms) and late phases (0-250 ms), and the muscle's voluntary activation percentage (VA%), calculated using Merton's formula (Equation 1) (201), were determined. VA% was considered an indicator of the level of voluntary efferent (i.e. central) drive to the muscle during the MVC (117).

$$VA\% = [1 - (\text{superimposed twitch} / \text{post-MVC resting twitch})] \times 100$$

$M_{max}$  from the superimposed twitch ( $M_{max,MVC}$ ), the mean VL EMG amplitude during the torque plateau (500 ms period prior to stimulation) and the ratio of these ( $EMG:M_{max,MVC}$ ) (19) were calculated from the three MVCs.  $EMG:M_{max,MVC}$  ratio was considered an indicator of central drive (efferent drive to the neuromuscular junction) that is independent from fatigue distal to the neuromuscular junction (117). Thus to distinguish central from peripheral fatigue, this measure was employed in conjunction with changes in the VA% to gain a clearer picture of central and peripheral fatigue origins (19, 329). After smoothing with a 70-ms RMS filter, the rate of increase in EMG amplitude (rate of EMG increase; REI) to early (0-15 ms), middle (0-30 ms) and late phases (0-50 ms) were calculated as indicators of the rate of muscle activity increase (i.e. rate of muscle activation).  $EMG f_{mean}$  were also calculated in the same 500-ms period during torque plateau using Fast-Fourier transformation as an indicator of muscle fatigue resulting from changes in muscle fibre conduction velocity (85, 107).

### 3.3.3 Statistical analyses

Data are expressed as mean values  $\pm$  standard deviation (SD). Percent change for all fatigue variables recorded during the neuromuscular assessments from pre- to post-exercise for each constant-load cycling test were statistically analysed using a one-way analysis of variance (ANOVA) with repeated measures at each time point ( $T_{20}$ ,  $T_{40}$ ,  $T_{60}$ ,  $T_{80}$  and  $T_{100}$ ). EMG variables recorded during cycling were also statistically analysed by one-way ANOVAs with repeated measures at each time point ( $T_0$ ,  $T_{20}$ ,  $T_{40}$ ,  $T_{60}$ ,  $T_{80}$  and  $T_{100}$ ). When significant main effects for time were found, Bonferroni pairwise comparisons were computed post-hoc to test differences between individual time points. All statistical analyses were performed using standard statistical software program (SPSS v 17 for Windows, SPSS, Inc., Chicago, IL, USA). Significance was set at  $p < 0.05$ .

## 3.4 Results

### 3.4.1 Incremental cycling test

$P_{\max}$ , maximal heart rate and RPE at the completion of the incremental cycling test were  $371 \pm 42$  W,  $184 \pm 10$  beats $\cdot$ min $^{-1}$  and  $18.7 \pm 1.1$ , respectively.

### 3.4.2 Constant-load cycling tests

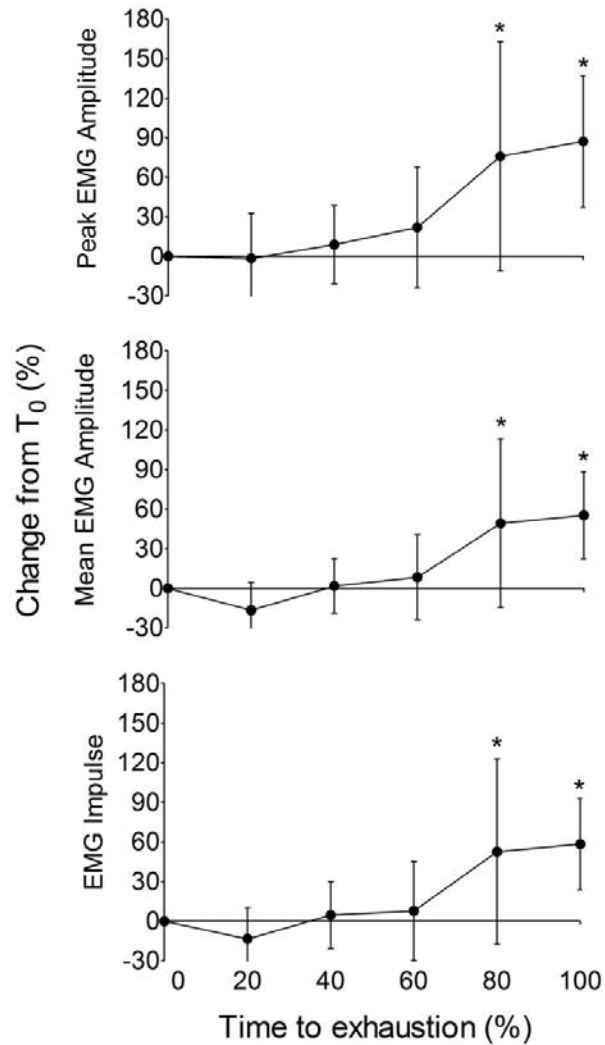
The participants cycled at an average power output of  $337 \pm 38$  W (90%  $P_{\max}$ ) for  $5:49 \pm 0:51$  min:s ( $T_{100}$ ). Target cadence was  $89 \pm 10$  rpm, which was maintained across all constant-load cycling tests. RPE increased throughout the exercise bout, and at  $T_{100}$  (termination of the constant-load cycling TTE test) was  $19.6 \pm 0.5$ .

### 3.4.3 Electromyography (EMG) during cycling

Muscle activation timing variables (onset and offset crank angles) did not change significantly across the exercise bout ( $p > 0.05$ ), and thus the duration of activation for all muscles remained constant ( $p > 0.05$ ). VL and VM were activated primarily in the propulsive phase of the down-stroke from just prior to TDC at  $349 \pm 4^\circ$  to  $123 \pm 2^\circ$  and  $351 \pm 2^\circ$  to  $120 \pm 3^\circ$ , respectively. SOL was also activated during the propulsive phase of the down-stroke between  $38 \pm 2^\circ$  and  $180 \pm 9^\circ$ . GL was activated concurrently with SOL, and continued activation was observed into the early stages of the up-stroke from  $56 \pm 4^\circ$  to  $208 \pm 3^\circ$ . TA was activated in the transition of the up- to down-stroke between  $305 \pm 3^\circ$  and  $27 \pm 4^\circ$ , and again for six of the nine participants in

1 the later stages of the transition of the down- to up-stroke between  $162 \pm 7^\circ$  and  $252 \pm$   
2  $9^\circ$  (i.e. secondary burst of EMG activity). In this case, analyses were focused only on  
3 the primary burst of EMG activity. BF was activated during the latter stages of the  
4 down-stroke and continued activation was shown into the early stages of the up-stroke  
5 between  $53 \pm 13^\circ$  and  $254 \pm 14^\circ$ . Finally, GMax was activated in the mid portion of the  
6 down-stroke between  $13 \pm 2^\circ$  and  $128 \pm 2^\circ$ .

7 The peak EMG amplitude of each muscle increased across the exercise bout.  
8 VM ( $p = 0.039$ ) and GMax ( $p = 0.002$ ) activation increased significantly throughout the  
9 exercise bout, but the trend for an increase in VL activation was not statistically  
10 significant ( $p = 0.087$ ) (Table 3.1 and Figure 3.2). Pairwise comparisons showed a  
11 significant effect for time between  $T_0$ - $T_{100}$  ( $p = 0.012$ ),  $T_{20}$ - $T_{100}$  ( $p = 0.027$ ),  $T_{40}$ - $T_{100}$  ( $p$   
12  $= 0.001$ ) and  $T_{60}$ - $T_{100}$  ( $p = 0.021$ ) for GMax peak EMG amplitude. Only the mean EMG  
13 amplitude and impulse of the primary EMG burst for GMax significantly increased  
14 across the exercise bout (EMG amplitude:  $p = 0.003$ ; EMG impulse:  $p = 0.004$ ) (Table  
15 3.1 and Figure 3.2). Pairwise comparisons revealed a significant effect for time between  
16  $T_0$ - $T_{100}$  ( $p = 0.015$ ),  $T_{20}$ - $T_{100}$  ( $p = 0.012$ ),  $T_{40}$ - $T_{100}$  ( $p = 0.009$ ) and  $T_{60}$ - $T_{100}$  ( $p = 0.013$ ),  
17 and between  $T_0$ - $T_{100}$  ( $p = 0.015$ ),  $T_{20}$ - $T_{100}$  ( $p = 0.014$ ),  $T_{40}$ - $T_{100}$  ( $p = 0.011$ ) and  $T_{60}$ - $T_{100}$   
18 ( $p = 0.008$ ) for GMax EMG amplitude and impulse, respectively. No significant  
19 differences were observed for the EMG  $f_{\text{mean}}$  across the exercise bout ( $p > 0.05$ ) (Table  
20 3.2).



**Figure 3.2** Percent changes in gluteus maximus peak and mean electromyogram (EMG) amplitude and EMG impulse during a high-intensity, constant-load cycling bout from T<sub>0</sub> to T<sub>20</sub>, T<sub>40</sub>, T<sub>60</sub>, T<sub>80</sub> and T<sub>100</sub>.

Data are the average from 10 pedal cycles at each time point (the first 10 pedal cycles at T<sub>0</sub> and the final 10 pedal cycles at T<sub>20</sub>-T<sub>100</sub>). There was a significant increase in GMax peak and mean EMG amplitude and impulse from T<sub>80</sub> onwards.

\* Denotes a significant difference from T<sub>0</sub> ( $p < 0.05$ ).

**Table 3.1** Percent changes in peak and mean electromyogram (EMG) amplitudes and the EMG impulse of seven lower limb muscles during a high-intensity, constant-load cycling bout for ten pedal cycles from  $T_0$  to  $T_{20}$ ,  $T_{40}$ ,  $T_{60}$ ,  $T_{80}$  and  $T_{100}$ .

	$T_{20}$	$T_{40}$	$T_{60}$	$T_{80}$	$T_{100}$
Peak EMG amplitude					
VL	$27.3 \pm 45.0$	$27.4 \pm 39.1$	$27.9 \pm 26.9$	$27.9 \pm 27.3$	$34.4 \pm 44.9$
VM*	$18.3 \pm 32.9$	$26.5 \pm 43.8$	$33.3 \pm 37.4$	$48.5 \pm 55.3$	$38.9 \pm 33.1$
TA	$18.6 \pm 52.6$	$30.4 \pm 48.5$	$29.3 \pm 49.0$	$33.3 \pm 48.3$	$28.2 \pm 37.2$
GL	$16.3 \pm 29.9$	$16.3 \pm 34.3$	$36.5 \pm 43.9$	$37.4 \pm 33.1$	$21.3 \pm 33.7$
SOL	$14.9 \pm 42.6$	$8.8 \pm 33.9$	$33.7 \pm 70.0$	$41.0 \pm 62.4$	$47.9 \pm 85.4$
BF	$9.2 \pm 42.0$	$19.0 \pm 48.0$	$29.3 \pm 45.5$	$26.4 \pm 42.0$	$32.7 \pm 52.3$
GMax*	$-1.6 \pm 34.2$	$8.9 \pm 29.8$	$22.0 \pm 45.9$	$75.9 \pm 87.2$	$87.2 \pm 50.1$
Mean EMG amplitude					
VL	$23.7 \pm 45.1$	$21.7 \pm 34.0$	$20.6 \pm 33.0$	$20.1 \pm 30.9$	$29.0 \pm 26.1$
VM	$30.6 \pm 50.2$	$36.0 \pm 41.0$	$36.9 \pm 32.1$	$49.8 \pm 60.2$	$40.7 \pm 53.5$
TA	$17.0 \pm 34.1$	$21.5 \pm 36.0$	$32.7 \pm 52.4$	$29.4 \pm 50.2$	$37.1 \pm 54.3$
GL	$11.9 \pm 28.6$	$14.1 \pm 24.9$	$25.3 \pm 31.8$	$28.9 \pm 31.4$	$6.9 \pm 21.6$
SOL	$23.8 \pm 34.6$	$19.5 \pm 29.8$	$28.1 \pm 49.8$	$30.8 \pm 44.9$	$35.4 \pm 57.7$
BF	$11.1 \pm 42.4$	$15.3 \pm 35.4$	$-7.7 \pm 17.5$	$4.5 \pm 17.5$	$3.8 \pm 44.1$
GMax*	$-16.6 \pm 21.0$	$1.9 \pm 20.8$	$8.4 \pm 32.2$	$49.4 \pm 64.0$	$55.4 \pm 32.9$
EMG impulse					
VL	$12.3 \pm 34.9$	$13.0 \pm 23.8$	$13.9 \pm 34.8$	$8.6 \pm 25.4$	$24.1 \pm 27.9$
VM	$19.7 \pm 43.2$	$31.1 \pm 37.1$	$29.3 \pm 35.5$	$40.0 \pm 69.7$	$38 \pm 63.7$
TA	$18.1 \pm 61.7$	$27.9 \pm 43.2$	$45.9 \pm 49.8$	$29.7 \pm 67.3$	$41.6 \pm 41.3$
GL	$11.2 \pm 25.9$	$8.7 \pm 24.7$	$26.4 \pm 32.9$	$28.4 \pm 32.0$	$8.7 \pm 22.5$

**Table 3.1** Cont.

	T <sub>20</sub>	T <sub>40</sub>	T <sub>60</sub>	T <sub>80</sub>	T <sub>100</sub>
EMG impulse					
SOL	16.3 ± 43.0	18.5 ± 28.8	20.7 ± 53.0	45.4 ± 73.1	53.5 ± 81.4
BF	25.4 ± 57.3	16.1 ± 39.0	30.8 ± 66.5	34.0 ± 45.9	39.1 ± 57.0
GMax*	-13.4 ± 23.4	4.7 ± 25.3	7.8 ± 37.5	52.5 ± 70.1	58.4 ± 34.6

Data are mean ± SD for nine participants.

Values are expressed as a percentage change from T<sub>0</sub> to T<sub>20</sub>, T<sub>40</sub>, T<sub>60</sub>, T<sub>80</sub> and T<sub>100</sub> for each constant-load cycling test.

VL, vastus lateralis; VM, vastus medialis; TA, tibialis anterior; GL, gastrocnemius lateral head; SOL, soleus; BF, biceps femoris; GMax, gluteus maximus.

\* Main effect of time ( $p < 0.05$ ).

**Table 3.2** Changes in the mean electromyogram frequency of seven lower limb muscles during a high-intensity, constant-load cycling bout for ten pedal cycles at T<sub>0</sub>, T<sub>20</sub>, T<sub>40</sub>, T<sub>60</sub>, T<sub>80</sub> and T<sub>100</sub>.

	T <sub>0</sub>	T <sub>20</sub>	T <sub>40</sub>	T <sub>60</sub>	T <sub>80</sub>	T <sub>100</sub>
VL	48.1 ± 5.0	49.3 ± 4.6	46.0 ± 4.5	50.7 ± 4.8	45.8 ± 7.1	44.6 ± 4.7
VM	48.7 ± 10.5	50.6 ± 6.4	48.5 ± 7.2	48.2 ± 7.9	46.4 ± 7.7	45.5 ± 6.5
TA	61.5 ± 6.0	68.8 ± 7.7	64.9 ± 8.0	66.9 ± 6.3	64.7 ± 10.8	62.4 ± 8.2
GL	52.6 ± 9.5	51.9 ± 5.6	52.2 ± 7.0	49.7 ± 5.2	52.6 ± 7.4	50.5 ± 6.8
SOL	53.3 ± 6.3	53.9 ± 8.5	53.6 ± 7.5	52.0 ± 6.1	52.1 ± 7.1	53.8 ± 8.2
BF	52.6 ± 9.7	50.2 ± 8.2	50.7 ± 6.5	49.7 ± 9.3	46.9 ± 8.7	48.5 ± 5.2
GMax	46.7 ± 7.6	52.6 ± 9.8	51.2 ± 6.7	52.8 ± 7.7	49.1 ± 9.5	48.7 ± 10.3

Data are mean ± SD for nine participants.

VL, vastus lateralis; VM, vastus medialis; TA, tibialis anterior; GL, gastrocnemius lateral head; SOL, soleus; BF, biceps femoris; GMax, gluteus maximus.

### 3.4.4 Neuromuscular assessment

#### 3.4.4.1 Voluntary force measurements

No significant differences were observed throughout the exercise bout for  $T_{p,MVC}$  and  $RTD_{MVC}$  to early (0-50 ms), middle (100-200 ms) or late phases (0-250 ms) measured during isometric knee extension (Table 3.3) ( $p > 0.05$ ).

#### 3.4.4.2 Twitch contractile properties

All twitch contractile properties except  $\frac{1}{2}RT$  changed significantly throughout the exercise bout ( $T_{p,twitch}$ :  $p < 0.001$ ;  $CT_{twitch}$ :  $p = 0.007$ ;  $RTD_{twitch}$ :  $p = 0.002$ ;  $RTR_{twitch}$ :  $p = 0.010$ ; Figure 3.3).  $T_{p,twitch}$  and  $RTR_{twitch}$  increased until  $T_{20}$ , and  $RTD_{twitch}$  increased until  $T_{40}$ , followed by a decline to below pre-exercise values at  $T_{100}$ . Pairwise comparisons showed a significant effect for time between  $T_{20}$ - $T_{80}$  ( $p = 0.026$ ),  $T_{20}$ - $T_{100}$  ( $p < 0.001$ ) and  $T_{40}$ - $T_{100}$  ( $p < 0.001$ ) for  $T_{p,twitch}$ , and  $T_{20}$ - $T_{100}$  ( $p = 0.008$ ;  $p = 0.010$ ) and  $T_{40}$ - $T_{100}$  ( $p = 0.004$ ;  $p = 0.010$ ) for  $RTD_{twitch}$  and  $RTR_{twitch}$ , respectively.  $CT_{twitch}$  showed a similar progression, except decrements below pre-exercise values were observed from  $T_{20}$  onwards before an increase from  $T_{80}$ - $T_{100}$ . Pairwise comparisons showed a significant effect for time between  $T_{20}$ - $T_{60}$  ( $p = 0.006$ ).

#### 3.4.4.3 Electromyogram and M-wave changes

No significant changes were observed in  $M_{max}$  or  $M_{dur}$  through the exercise bout measured ( $p > 0.05$ ) ( $M_{max}$ :  $-11.3 \pm 28.0\%$ ;  $-8.7 \pm 14.0\%$ ;  $-12.7 \pm 13.2\%$ ;  $-29.8 \pm 29.8\%$ ; and  $-30.6 \pm 38.3\%$  for  $T_{20-100}$ , respectively;  $M_{dur}$ :  $-0.8 \pm 11.9\%$ ;  $3.6 \pm 15.5\%$ ;  $7.0 \pm 16.3\%$ ;  $5.6 \pm 28.1\%$ ; and  $8.1 \pm 13.9\%$  for  $T_{20-100}$ , respectively). No significant changes were observed in the VL EMG amplitude, EMG: $M_{max,MVC}$  ratio,  $M_{max,MVC}$ , VA%, peak EMG amplitudes, REI to early (0-15 ms), middle (0-30 ms) or late phases (0-50 ms) (Table 3.3), or EMG  $f_{mean}$  measured during the isometric knee extension task ( $p > 0.05$ ). However, there was a trend towards an increase in VL EMG amplitude ( $p = 0.065$ ). The lack of significant change might partly be a consequence of the observed inter-individual variability (e.g.  $5.7 \pm 19.3\%$  at  $T_{100}$ ; see Table 3.3).



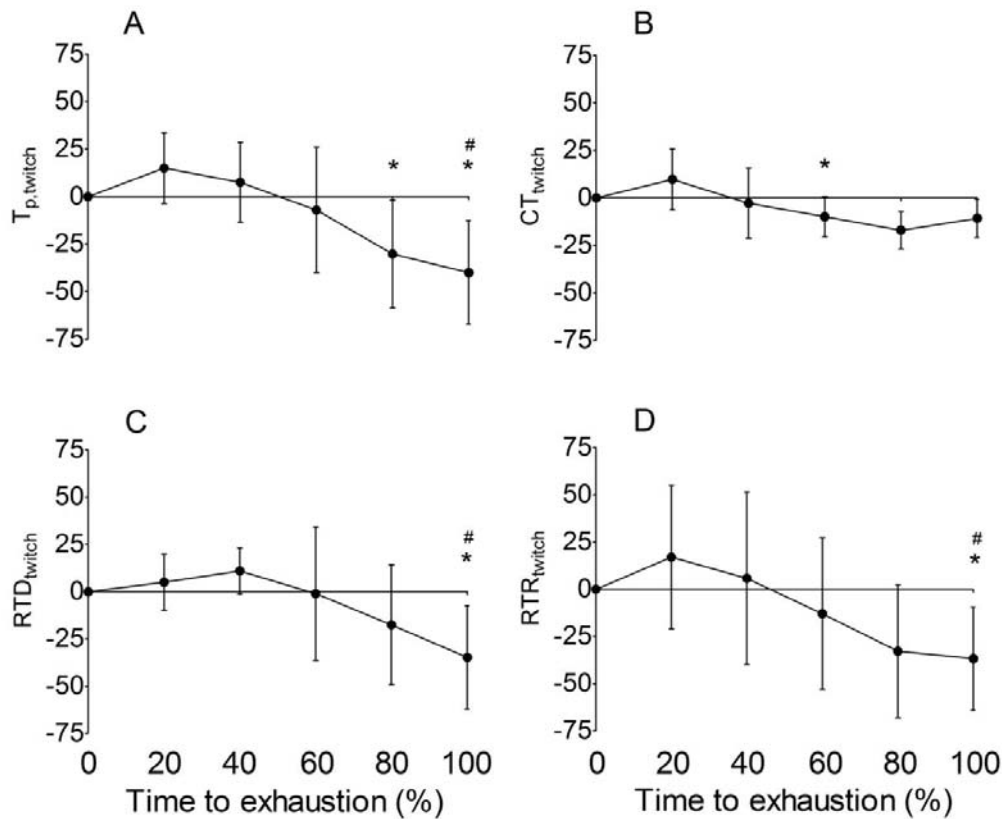
**Table 3.3** Percent changes in isometric maximal voluntary contraction (MVC) mechanical and electromyogram (EMG) responses during a high-intensity, constant-load cycling bout from T<sub>0</sub> to T<sub>20</sub>, T<sub>40</sub>, T<sub>60</sub>, T<sub>80</sub> and T<sub>100</sub>.

Percent Change	T <sub>20</sub>	T <sub>40</sub>	T <sub>60</sub>	T <sub>80</sub>	T <sub>100</sub>
T <sub>p,MVC</sub>	-4.7 ± 8.3	-7.0 ± 8.1	-9.4 ± 6.9	-9.2 ± 11.4	-12.5 ± 12.0
VA	-1.7 ± 2.4	-3.1 ± 5.6	-2.4 ± 5.9	-2.1 ± 4.7	-2.7 ± 9.0
RTD					
Early phase	-11.0 ± 7.9	-12.4 ± 13.3	-6.2 ± 16.8	-16.8 ± 24.3	-21.33 ± 15.7
Middle phase	1.8 ± 13.8	-4.4 ± 12.1	-2.3 ± 15.0	-8.1 ± 15.8	3.6 ± 24.2
Late phase	-5.7 ± 10.3	-6.8 ± 10.0	-5.9 ± 14.1	-10.9 ± 12.2	-11.4 ± 12.1
VL EMG amplitude					
M <sub>max,MVC</sub>	-14.8 ± 7.6	-3.4 ± 11.9	-0.4 ± 20.9	1.7 ± 11.0	5.7 ± 19.3
EMG:M <sub>max,MVC</sub>	-15.7 ± 15.7	-14.9 ± 14.6	-16.4 ± 31.1	-17.7 ± 22.7	-27.2 ± 20.9
REI	4.6 ± 22.2	16.4 ± 22.8	44.7 ± 89.7	34.3 ± 45.1	61.3 ± 71.4
Early phase	-12.8 ± 36.1	-18.5 ± 43.6	-7.0 ± 39.3	-8.7 ± 34.6	-7.5 ± 38.3
Middle phase	-18.9 ± 29.5	-18.2 ± 34.7	-14.3 ± 26.4	-14.0 ± 30.5	-2.9 ± 31.4
Late phase	-17.9 ± 28.7	-25.7 ± 34.2	-17.6 ± 17.1	-4.6 ± 30.0	-0.1 ± 28.6

Data are mean ± SD for nine participants.

MVC mechanical and EMG responses were assessed via supramaximal electrical stimulation of the femoral nerve before and after exercise. Values are expressed as a percentage change from T<sub>0</sub> to T<sub>20</sub>, T<sub>40</sub>, T<sub>60</sub>, T<sub>80</sub> and T<sub>100</sub> for each constant-load cycling test.

T<sub>p,MVC</sub>, peak torque; VA, percent voluntary activation estimated from the interpolated twitch technique; RTD, rate of torque development, early phase (0-50ms), middle phase (100-200ms) and late phase (0-250ms); Vastus lateralis (VL) EMG amplitude, mean EMG activity of VL during torque plateau of the MVC; M<sub>max,MVC</sub>, M-wave peak-to-peak amplitude from the superimposed twitch during the MVC; EMG:M<sub>max,MVC</sub>, ratio of the mean VL EMG amplitude to the M<sub>max,MVC</sub>; REI, rate of EMG increase, early phase (0-15ms), middle phase (0-30ms) and late phase (0-50ms).



**Figure 3.3** Peak twitch torque ( $T_{p,twitch}$ ) (A), twitch contraction time ( $CT_{twitch}$ ) (B), and the average rates of twitch torque development ( $RTD_{twitch}$ ) (C) and relaxation ( $RTR_{twitch}$ ) (D) during a high-intensity, constant-load cycling bout from  $T_0$  to  $T_{20}$ ,  $T_{40}$ ,  $T_{60}$ ,  $T_{80}$  and  $T_{100}$ .

There was a potentiation effect until  $\sim T_{40}$  followed by a marked decline in all twitch parameters from  $\sim T_{60}$  (see 3.4.4.2 for more detail). \* Denotes a significant difference from  $T_{20}$  and # denotes a significant difference from  $T_{40}$  ( $p < 0.05$ ).

### 3.5 Discussion

This study examined the temporal progression of physiological fatigue and changes in muscle activation during high-intensity, constant-load cycling to exhaustion in humans. The main findings of this investigation were that: i) there were no changes in the patterns of muscle activity and coactivity measured during cycling, suggesting a rigid temporal activation pattern was retained throughout the exercise bout; ii) there were increases in VM and GMax muscle activity measured during cycling, as well as an increase in the VL EMG amplitude measured during isometric knee extension, indicating a possible up-regulation of central motor drive across the exercise bout; iii) twitch contractile properties ( $T_{p,twitch}$ ,  $CT_{twitch}$ ,  $RTD_{twitch}$  and  $RTR_{twitch}$ ) were

1 significantly impaired from  $T_{60}$  onwards, indicating an increase in peripheral fatigue;  
2 and iv) there were no detectable changes across the exercise bout in voluntary torque  
3 ( $T_{p,MVC}$  and  $RTD_{MVC}$ ) or VA% measured during isometric knee extension, indicating  
4 that there was little or no effect on the ability to produce voluntary muscle force.  
5 Collectively, these results suggest that peripheral fatigue becomes significant from  $T_{60}$   
6 onwards and central facilitation, rather than central fatigue, occurs towards the end of  
7 the exercise bout near task failure during high-intensity, constant-load cycling.

8         The neuromuscular pattern employed during cycling can change with  
9 progressive fatigue, and the strategies used to overcome this may relate to the level,  
10 timing and/or frequency characteristics of activation of the muscles involved. In the  
11 current study, highly consistent patterns of muscle recruitment were evident, with no  
12 significant changes in muscle timing observed despite the development of significant  
13 peripheral fatigue. These patterns of muscular recruitment were typical of those  
14 previously observed in lower limb muscles during cycling (for review see (149)). Given  
15 that the load and cadence of the cycling task was fixed, it could be suggested that  
16 participants maintained the same neuromuscular recruitment pattern to allow peak  
17 torque to be applied to the pedal at the same crank angle, as a change in muscle  
18 activation timing could result in inefficient pedal torque application (221, 261).  
19 Therefore, a central strategy may be to ensure mechanical efficiency at the required  
20 power output during cycling by using a rigid neuromuscular pattern, regardless of the  
21 level of neuromuscular fatigue developed. Whether such a strategy is used by lesser-  
22 trained individuals should be examined in future research.

23         Nonetheless, the level of muscle activity increased substantially with exercise  
24 progression, as reflected by the significant increase in the mean EMG amplitude and  
25 impulse of GMax and the peak VM and GMax EMG amplitudes (Table 3.1 and Figure  
26 3.2). Similarly, the mean VL EMG amplitude increased significantly during isometric  
27 knee extension (see below). In general, activity in the knee extensors (84, 89, 145, 185,  
28 241, 261, 263, 312), BF (89, 291, 312), GMax (88, 89, 312) and adductor magnus and  
29 longus (312) have previously been shown to increase during cycling, whilst activity in  
30 GM, TA (89) and RF (88) have been shown to decrease. In some studies, the activities  
31 of GL, SOL, and RF (89, 263) have been shown to remain consistent during cycling.  
32 However, only a few of these studies used a constant-load cycling protocol (89, 185),  
33 and the EMG amplitudes measured in the present study increased more than reported in  
34 other studies. These discrepancies may result from the different exercise protocols  
35 employed, the time period in which the EMG amplitude was calculated and/or the

specific characteristics of the participants. Regardless, these data support the notion that the observed increase in lower limb EMG, and thus central motor drive, acts as a compensatory mechanism to overcome the progression of peripheral fatigue during open-loop intensive cycling where the exercise duration is unknown. It also signifies a change in the muscle activation strategy that may be necessary to maintain the required power output of the task. These phenomena have also been shown during constant-load cycling to exhaustion, where significant increases in knee extensor and flexor EMG amplitude were associated with progressive muscle contractile failure (10, 84, 185). These results clearly demonstrate that peripheral muscle fatigue can be partly overcome by an increase in central motor drive during high-intensity, open-loop, constant-load exercise.

The present results also provide evidence that the development of neuromuscular fatigue during this type of exercise induces differential changes in the amplitude (increase) and frequency (no change) components of the EMG signal (53, 156, 214), because no changes in EMG  $f_{\text{mean}}$  were found during cycling (Table 3.2). Since there were also no changes in M-wave characteristics measured during the isometric knee extension task ( $M_{\text{max}}$ ,  $M_{\text{dur}}$  and  $M_{\text{max,MVC}}$ ) (see below), membrane excitability was probably maintained (53, 111, 156). These results are consistent with previous reports during fatiguing constant-load cycling (185, 261), although significant decreases in EMG frequency characteristics for some lower limb muscles (i.e. VL (53); GL and BF (87)) have been observed during fatiguing incremental (53) and constant-load (87) cycling to exhaustion.

An important goal is to more clearly elucidate the mechanisms underpinning the change in muscle activity and reduced exercise capacity. A novel study design was used that allowed a temporal picture of fatigue to be developed, whereby the subjects were unexpectedly stopped at different time intervals and a neuromuscular exam was completed. One difficulty that has not yet been overcome is how to test muscle contractile properties instantaneously upon whole-body exercise cessation. Thus, while the study has provided details on the temporal profile of changes in contractile function, the actual magnitude of alterations in neuromuscular function are likely to be somewhat greater than those reported here (9, 112). An important next step is to establish a method by which such data can be obtained immediately upon exercise cessation, without removing the participant from the cycle ergometer.

Nonetheless using this design, a distinctively sigmoidal pattern of peripheral fatigue development was observed, with potentiation effects from  $T_0$ - $T_{40}$  followed by a

1 progressive decline to  $T_{100}$ . Twitch contractile properties measured during an isometric  
2 knee extension test, including  $T_{p,twitch}$  and the parameters related to force production  
3 ( $RTD_{twitch}$ ) and relaxation ( $RTR_{twitch}$ ) rates displayed similar temporal patterns of fatigue  
4 progression, with increases in force production observed until  $T_{40}$  before a decrease to  
5 below pre-exercise values at exhaustion. Nonetheless,  $CT_{twitch}$  was significantly  
6 impaired from  $T_{60}$  onwards while no changes in M-wave characteristics were detected;  
7 together these results are suggestive that E-C coupling was less effective towards  
8 exercise termination (Figure 3.3). Previous research has indicated that such potentiating  
9 effects (i.e. to  $T_{40}$ ) may result from an increased myosin light chain phosphorylation  
10 (213, 250, 301, 307), which typically results from an increased  $Ca^{2+}$  sensitivity of actin  
11 and myosin filaments leading to an improved rate of cross-bridge attachment (250). The  
12 significant decrement in  $T_{p,twitch}$  (-40%) and  $RTD_{twitch}$  (-35%) from  $T_{60}$  onwards  
13 indicates an impairment in the E-C coupling process, which has been shown to be  
14 caused by multiple mechanisms including reduced  $Ca^{2+}$  released from the sarcoplasmic  
15 reticulum (5, 6, 92, 113, 117, 232, 247, 317), reduced  $Ca^{2+}$  affinity of the contractile  
16 elements (5, 6, 109, 117, 234) and/or a reduced capacity of actin and myosin filaments  
17 to form strong cross-bridge bonds (6, 108). The significant decline in relaxation rates  
18 ( $RTR_{twitch}$ ) (-37%) cannot be explained using data collected in the present study, but  
19 previous reports have suggested that these changes may result from a reduction in  
20 myosin ATPase activity, decreased functioning of the sarcoplasmic reticulum pump (i.e.  
21 a reduction in the rate of  $Ca^{2+}$  re-uptake) and/or a slowed cross-bridge detachment rate  
22 (109, 202, 315, 316). Nonetheless, no changes were observed in  $\frac{1}{2}RT_{twitch}$ , probably  
23 because both the peak twitch force and rate of relaxation decreased in tandem. In this  
24 context, a lack of change in  $\frac{1}{2}RT_{twitch}$  should not be considered to reflect a lack of  
25 change in muscle relaxation characteristics; a lack of change in the  $\frac{1}{2}RT$  has previously  
26 been reported after longer bouts of cycling (43, 175, 177).

27 In the present study, the robustness of the M-wave ( $M_{max}$  and  $M_{dur}$ ) measured  
28 during isometric knee extension indicates that the transmission and propagation of  
29 action potentials along the muscle fibre were probably unaffected by the cycling bout.  
30 However, a trend towards a decrease in  $M_{max}$  (i.e., lower amplitude) and increase in  
31  $M_{dur}$  (i.e., extended duration) were observed and may indicate that the development of  
32 fatigue at the sarcolemmal level progresses relatively constantly throughout the exercise  
33 bout and may be significant during longer bouts of exercise, or that it was able to  
34 recover rapidly before the data could be obtained; future research is required to more  
35 successfully test M-wave changes. Significant decrements in M-wave characteristics

1 have typically been observed in whole-body endurance exercise after many hours (175,  
2 207, 242) or repeated bouts of high-intensity exercise (240). Nonetheless, it is unlikely  
3 that the small changes in action potential propagation seen in the current study would  
4 have affected the voltage-triggered release of  $\text{Ca}^{2+}$  from the sarcoplasmic reticulum (5).  
5 Importantly, during high-intensity, shorter-term exercise, it is probable that an increased  
6 level of catecholamines provides some level of protection against fatigue at the  
7 sarcolemmal level (52, 64, 180, 198). Taken together, these results strongly suggest that  
8 neuromuscular fatigue mechanisms at the muscle level, distal to the neuromuscular  
9 junction (i.e. decreased E-C coupling efficiency) contributed to fatigue development.

10 Despite evidence for an increase in peripheral fatigue development with  
11 increasing task duration, no change in voluntary knee extensor torque ( $T_{p,MVC}$  and  
12  $RTD_{MVC}$ ) was observed in the present study. Indeed, although non-significant, there was  
13 a trend towards a decline in  $T_{p,MVC}$  of the knee extensors across the exercise bout (-13%  
14 at  $T_{100}$ ) (Table 3.3). This reduction is comparable to previous studies involving cycling  
15 exercise of similar duration and workload (-10%; 7.3 min at 347 W (10); -14%; 7.6 min  
16 at 351 W (11)), but is less than has been shown after prolonged lower intensity cycling  
17 (-21% (82); -23% (275)). Similarly, no changes in  $RTD_{MVC}$  to early (0-50 ms), middle  
18 (100-200 ms) or late phases (0-250 ms) were observed. Although this finding might  
19 partly be explained by the ~82 s delay between cycling exercise cessation and the  
20 neuromuscular assessments, it is speculated that the increase in central motor drive at  
21 exhaustion was adequate to allow the participants to produce voluntarily knee extensor  
22 torque similar to pre-exercise values despite significant peripheral fatigue. As evidence  
23 of this, a trend towards an increase in the VL EMG amplitude when measured during  
24 the isometric knee extension task was observed. Similarly, as discussed above, during  
25 cycling the EMG amplitudes of all muscles tested were maintained or augmented  
26 throughout the exercise bout, and in particular VM and GMax. It is important to note  
27 that the present results provide evidence that neuromuscular fatigue development  
28 measured during both the cycling and the isometric knee extension task were similar  
29 and thus fatigue measured post-exercise was reflective of that that occurred *during* the  
30 exercise task.

31 Finally, it should be pointed out that a stronger central motor drive may have  
32 contributed to a greater subjective level of exertion and ultimately limited exercise  
33 performance (16, 189). RPE increased throughout the exercise bout, resulting in a  
34 maximal end-exercise perceived exertion of  $19.6 \pm 0.5$ . It is likely that high RPE values  
35 are related to an individual's critical sensory threshold, and that peripheral fatigue is

1 consciously or sub-consciously regulated via ‘adjustments’ in central motor drive  
2 during self-paced closed-loop exercise (10, 11, 132, 168, 224, 283, 288, 289). However,  
3 participants in the present study were unable to regulate exercise intensity given that the  
4 exercise duration was unknown (9). Therefore, instead it seems as though central motor  
5 drive was increased in order to maintain the constant power output of the test. This  
6 effect would maximise skeletal muscle recruitment to enable continuation of exercise  
7 performance, albeit at the expense of increased muscle fatigue (264, 283). It is therefore  
8 likely that the conscious decision to terminate the exercise bout was a direct  
9 consequence of the increased sense of exertion (290), motivational factors (272, 290)  
10 and/or increased muscle afferent (group III/IV) input and associated perception of  
11 muscle pain (8, 116).

12 In conclusion, these findings show that during high-intensity, constant-load  
13 cycling, a compensatory increase in the magnitude of lower limb muscle activity  
14 occurred as an attempt to maintain the required power output, while no changes in the  
15 timing of muscle activity were observed. It should be acknowledged that the present  
16 findings are somewhat limited to cycling exercise of similar intensity and duration.  
17 Regardless, impairments in muscle function following cycling appeared to be primarily  
18 associated with peripheral fatigue development from about 60% of the time to task  
19 failure, while a central facilitation, rather than fatigue, occurred near exhaustion. Using  
20 a novel repeat-test exercise model, there is evidence to indicate that reduced E-C  
21 coupling efficiency and muscle relaxation rates (i.e., fatigue distal to the neuromuscular  
22 junction) may have impaired exercise capacity; a reduction in the time taken between  
23 exercise cessation and neuromuscular testing should be a prime goal of future research.  
24 It is speculated that changes such as those reported here might also occur during other  
25 manual tasks performed for moderate durations at relatively high intensities.

26

27

28

29

30

31

32



## CHAPTER FOUR

### The temporal relationship between joint kinematics and neuromuscular fatigue in high-intensity, constant-load cycling

#### 4.1 Abstract

**Purpose:** The temporal relationships between lower and upper limb kinematics and changes in markers of central and peripheral fatigue (described in Chapter 3), as well the temporal progression of changes in joint kinematics, were examined during high-intensity, constant-load cycling. **Methods:** Nine well-trained male cyclists performed seven testing sessions, including an incremental cycling test to exhaustion, a time to exhaustion (TTE) test at 90%  $P_{\max}$  (power output at  $VO_{2\max}$ ) at a constant cadence, and tests to 20, 40, 60, 80 and 100% ( $T_{20}$ – $T_{100}$ ) of the time taken to reach task failure in the TTE test ( $5:49 \pm 0:51$  min:s). Lower (i.e. hip, knee and ankle joint) and upper (i.e. trunk) limb kinematics in the sagittal and coronal planes were assessed during cycling ( $T_0$ – $T_{100}$ ), to determine joint kinematic changes throughout the test. Representative markers of central (EMG amplitudes of vastus lateralis, vastus medialis and gluteus maximus) and peripheral (peak twitch torque) fatigue from Chapter 3 were assessed as previously described. **Results:** During cycling, significant increases in trunk flexion mean, maximum and minimum joint angles were observed from  $T_{60}$  ( $p < 0.05$ ). Increases in trunk medio-lateral sway (lateral flexion) maximum and minimum joint angles and range of motion (ROM), left and right hip abduction/adduction ROM and right hip abduction/adduction minimum joint angle were observed from  $T_{80}$  ( $p < 0.05$ ). Increases in right knee valgus/varus ROM were observed at  $T_{100}$  (i.e., at the termination of exercise) ( $p < 0.05$ ). Finally, at  $T_{100}$ , a significant decrease in the right knee valgus/varus ROM was also observed ( $p < 0.05$ ). **Conclusions:** Significant trunk kinematic changes were observed in the sagittal plane from 60% of the time to task failure, and were thus notable at or after the point of significant peripheral fatigue development, whereas significant changes at the trunk, hip and knee joints in the coronal plane were observed from 80% of the time to task failure, which therefore paralleled the facilitation of central motor drive during the cycling task. Kinematic changes in the sagittal and coronal plane are temporally dissociated, and may be in part be related to the site of fatigue (i.e. peripheral or central) induced by the exercise bout.



## 4.2 Introduction

An effective movement pattern that optimises movement efficiency is considered to be extremely important for the successful performance of complex, repetitive tasks and activities of daily living. Cycling has been commonly used as a model in research aimed at understanding the effect of movement patterns on exercise performance, with numerous studies examining the influence of rider technique on neuromuscular control strategies, ventilatory/metabolic responses and cycling power output (20, 88, 127, 231, 274, 299, 314). Many of these studies have focused on trunk position, and found that an increase in trunk flexion results in: (i) changes in lower limb muscle recruitment (58, 88, 265), (ii) a reduction in power production (299), (iii) a change in the application of pedal force (88), (iv) minimal effects on lower limb joint kinematics (58), and either (v) an increase in the metabolic cost of cycling (greater heart rate and/or  $\text{VO}_2$  responses) (101, 127), or (vi) a minimal change in heart rate, ventilatory and metabolic ( $\text{VO}_2$ ) responses (20, 88, 101, 129, 231, 274, 314).

Nonetheless, few studies have examined the potential changes in kinematics that occur *during* a fatiguing cycling bout (37, 38, 87, 192, 266). Within these studies the kinematic changes observed with fatiguing cycling are typically measured in the sagittal plane and include an increase in ankle dorsiflexion (37, 38, 192, 266), trunk lean (i.e. flexion) (87), hip and knee extension (37, 38, 87, 266) and in the coronal plane knee splay (i.e. abduction/adduction) (87). These alterations in the movement pattern are typically associated with changes in joint rigidity and the timing and relative activity of muscles (133, 219, 271), suggesting that the neuromuscular system utilises specific strategies to combat fatigue accumulation in order to maintain task performance (120). To date, only two studies have detailed the temporal progression of kinematic changes during cycling (38, 266). Bini et al. (2010) found significant kinematic changes from 90% of the time to fatigue during high-intensity, constant-load cycling ( $405 \pm 90$  s), which included a decrease in mean ankle angle, an increase in ankle ROM, an increase in mean hip and knee angle, and a decrease in hip ROM (38). Conversely, an early decrease in ankle joint ROM along with a later increase in mean tibial rotation has been observed during prolonged (60 min) time-trial cycling (266). While these studies have provided some information regarding the changes in cycling kinematics, it is still not known how sagittal *and* coronal plane kinematics change and what temporal relationship exists between these changes the progression of fatigue throughout a high-intensity, constant-load cycling bout.

Whilst kinematic alterations are often associated with the development of neuromuscular fatigue, previous reports have primarily examined neuromuscular mechanisms of fatigue and/or changes in cycling biomechanics separately (37, 38, 82, 87, 166, 175, 176, 178, 192, 209, 266, 296). The association between neurophysiological and biomechanical changes are of interest because this knowledge will improve our understanding of how individuals respond, and adapt, to fatiguing exercise. At present, only one study has described the temporal relationship between neuromuscular function and kinematic adjustments during high-intensity cycling (87). Dingwell et al. (87) found that alterations in lower limb muscle recruitment (EMG) strategies during cycling at 100% MAP were preceded by significant alterations in joint kinematics. However, muscle fatigue was quantified using changes in the EMG  $f_{\text{median}}$  (87), which provides limited information regarding the contribution of central and peripheral fatigue mechanisms and its use is controversial during dynamic, fatiguing muscular contractions (105, 107). In Chapter 3, the association between changes in muscle activation and both the magnitude and timing of central and peripheral fatigue was investigated during short duration, high-intensity exercise (i.e. cycling at 90% of MAP). It was found that peripheral fatigue (i.e. impaired muscle function) was significant from 60% of the time to task failure and appeared to be the dominant limiting factor in regards to exercise capacity. A central facilitation, rather than inhibition (i.e. significant increases in VM and GMax muscle activity during cycling exercise), was found to occur later in the exercise bout, with GMax activation significant from 80% of the time to task failure. Given that changes in joint kinematics may influence force transfer to the pedals, and thus power production, it is of interest to investigate whether the temporal development of neuromuscular fatigue is associated with changes in kinematics.

In Chapter 3, changes in neuromuscular function were measured during multiple, constant-load cycling tests of different durations on separate days to build a temporal ‘fatigue profile’ that reflected fatigue progression to exhaustion. In the present study the results of a detailed biomechanical analysis of the cyclists during each exercise bout will be reported, as well as the temporal relationships between kinematic changes and changes in markers of central and peripheral fatigue; thus, some of the data has been reported in Chapter 3. Therefore, the overall aim of the present study was to examine the temporal relationship between neuromuscular fatigue and kinematic changes observed during high-intensity, constant-load cycling. Accordingly, the specific purposes of the present study were to: i) assess the temporal relationship between joint

1 kinematics, and changes in key indicators of peripheral fatigue and central facilitation  
2 (lower limb muscle activity), and ii) determine the magnitude of change in lower (i.e.  
3 hip, knee and ankle joint) and upper (i.e. trunk) limb kinematics in both the sagittal and  
4 coronal planes during high-intensity, constant-load cycling.

## 6 **4.3 Methods**

### 7 **4.3.1 Participants**

8 As described in Study 1 (Chapter 3).

### 9 **4.3.2 Experimental protocol**

10 As described in Study 1 (Chapter 3).

#### 12 **4.3.2.1 Incremental cycling test**

13 As described in Study 1 (Chapter 3).

#### 15 **4.3.2.2 Constant-load cycling tests**

16 As described in Study 1 (Chapter 3).

#### 18 **4.3.2.3 Neuromuscular function**

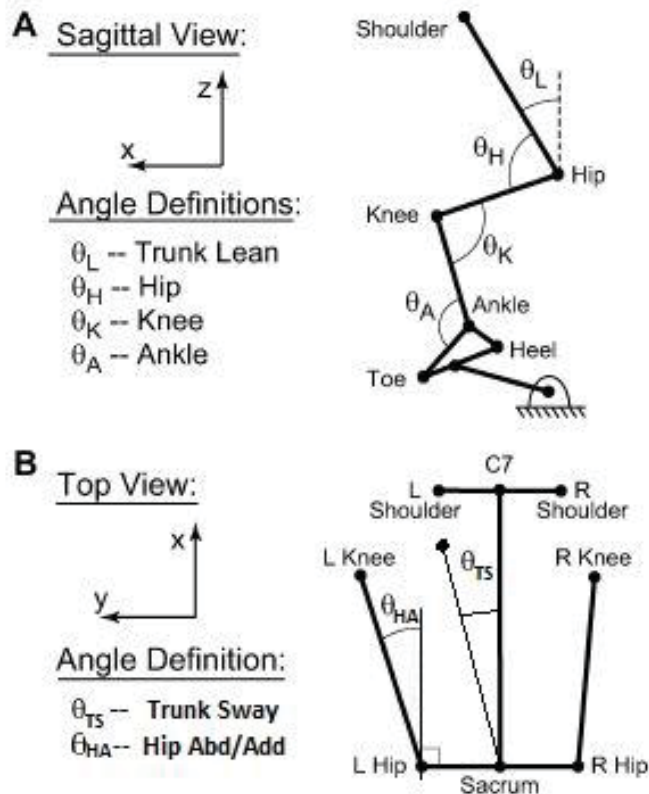
19 As described in Study 1 (Chapter 3); however see Section 4.2.3 for the inclusion  
20 of representative measures of neuromuscular function that will be used and discussed in  
21 the present study of this thesis.

#### 23 **4.3.2.4 Kinematics during cycling**

24 Sagittal and coronal plane motions of the trunk and the left and right lower limbs  
25 were measured during all constant-load cycling tests using a full-body, 15-segment  
26 marker set and a 10-camera VICON MX motion analysis system (Oxford Metrics Ltd.,  
27 UK). Reflective markers were placed on anatomical landmarks according to the Plug-in-  
28 Gait® model and secured using medical tape to minimise movement relative to the skin.  
29 The upper and lower body anatomical coordinate systems were defined using the Plug-  
30 in-Gait® model and were modelled as 15 segments, including the head, thorax, upper  
31 and lower arms, hands, pelvis, upper and lower legs and feet. Marker coordinates were  
32 sampled at 250 Hz and time synchronised with EMG signals using Vicon Nexus  
33 software (Vicon Motion Systems, USA). Hip, knee and ankle angles were calculated for

1 both the left and right sides of the body and trunk angles were calculated relative to the  
2 pelvis; please note, trunk medio-lateral sway joint angle was calculated as the medio-  
3 lateral displacement of the thorax segment relative to the pelvis segment, hip  
4 abduction/adduction joint angles were calculated as the medio-lateral displacement of  
5 the upper leg segment (thigh) relative to the pelvis segment, and knee valgus/varus joint  
6 angles were calculated as the medio-lateral displacement of the upper leg segment  
7 (thigh) relative to the lower leg segment (shank) (see Figure 4.1). All joint angles were  
8 expressed relative to the anatomical position.

9         Marker coordinates were sampled at 250 Hz using Vicon Nexus software (Vicon  
10 Motion Systems, USA). Marker coordinates were recorded in the first 25 s of each 30 s  
11 period and then near exhaustion when cadence dropped close to 5% below the target  
12 cadence to ensure the final period of the test was recorded. Pedal cycle quadrants  
13 ( $0^{\circ}/360^{\circ}$  (TDC),  $90^{\circ}$ ,  $180^{\circ}$  (bottom-dead centre), and  $270^{\circ}$ ) were identified using a  
14 customised four-way polarity switch attached to the crank with individual sensors on  
15 each quadrant. Following data collection the time taken to reach task failure for each  
16 test ( $T_{100}$ ) was divided into 20% time periods ( $T_{20}$ – $T_{80}$ ). Joint angle data from 10  
17 consecutive pedal cycles at each time point (the first 10 pedal cycles at  $T_0$  and the final  
18 10 pedal cycles at  $T_{20}$ – $T_{100}$ ) were filtered with a 4<sup>th</sup> order zero-lag Butterworth filter  
19 with low-pass cut-off frequency of 10 Hz. Joint angle data for each quadrant of the  
20 pedal cycle were time normalised using cubic splines to a time base of 100 samples.  
21 The mean, maximum and minimum joint angles, and joint ROM (minimum joint angle  
22 subtracted from the maximum joint angle) throughout the pedal cycle were calculated  
23 from individual pedal cycles and then averaged for each time point ( $T_0$ – $T_{100}$ ) for all  
24 constant-load cycling tests.



**Figure 4.1** Schematic representations of joint angle definitions (adapted from Dingwell et al (2008) (87). (A): Sagittal plane view showing trunk flexion/lean, hip, knee and ankle joint angles. (B): Coronal plane view showing trunk medio-lateral sway and hip abduction/adduction joint angles (please note knee valgus/varus joint angles are not shown but are described in text).

### 4.3.3 Data analyses

As per reported in Chapter 3, peripheral fatigue (i.e. decrements in twitch contractile properties) developed early in the exercise bout from 60% of the time to task failure, and a central facilitation (i.e. increases in VM and GMax peak EMG amplitudes during cycling exercise) occurred towards the end of the exercise bout. In the present study, the temporal association between selected measures of neuromuscular function and kinematic changes are reported.  $T_{p,twitch}$  was chosen as a representative indicator of peripheral fatigue, given that all twitch contractile measures were found to follow a similar trend of fatigue development.  $T_{p,twitch}$  increased until 20% of the time to task failure ( $15.0 \pm 18.6\%$ ) and was then followed by a linear decline to below pre-exercise values at task failure ( $-39.9 \pm 27.3\%$ ). Peak EMG amplitudes of knee extensors (VM and VL) and GMax were chosen as representative indicators of central motor drive

1 facilitation. There were significant increases in VM ( $38.9 \pm 33.1\%$ ) and GMax ( $87.2 \pm$   
2  $50.1\%$ ) peak EMG amplitudes throughout the cycling exercise bout with fatigue  
3 accumulation, and GMax activation was significant from 80% of the time to task  
4 failure.

#### 6 **4.3.4 Statistical analyses**

7 Data are expressed as mean values  $\pm$  SD. Kinematic variables recorded during  
8 cycling at each time point ( $T_0$ - $T_{100}$ ) were analysed using a one-way repeated measures  
9 ANOVA. When significant main effects were found, Bonferroni pairwise comparisons  
10 were performed. All statistical analyses were performed using standard statistical  
11 software program (SPSS v 17 for Windows, SPSS, Inc., Chicago, IL, USA).  
12 Significance was set at  $p < 0.05$ .

### 14 **4.4 Results**

#### 15 **4.4.1 Incremental cycling test**

16 As reported in Study 1 (Chapter 3) of this thesis.

#### 18 **4.4.2 Constant-load cycling tests**

19 As reported in Study 1 (Chapter 3) of this thesis.

#### 21 **4.4.3 Neuromuscular function**

22 As reported in Study 1 (Chapter 3) of this thesis.

#### 24 **4.4.4 Kinematics during cycling**

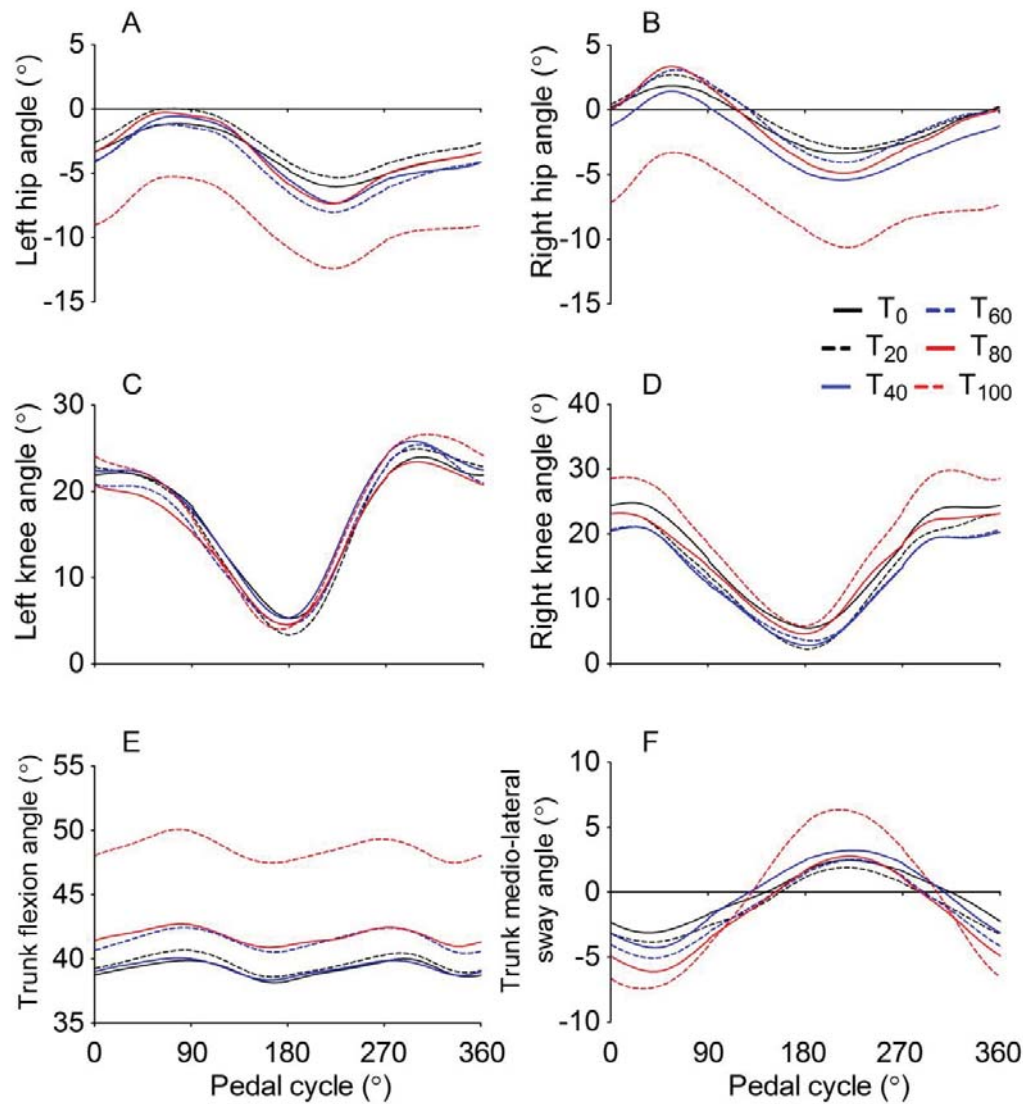
25 Changes in joint kinematics observed throughout the exercise bout for the trunk,  
26 hip, knee and ankle are shown in Table 4.1 and Figure 4.2. Trunk mean, maximum and  
27 minimum angles in the sagittal plane increased significantly (i.e. towards a greater  
28 degree of trunk flexion) throughout the exercise bout (MA,  $p = 0.001$ ; maximum angle,  
29  $p = 0.001$ ; minimum angle,  $p < 0.001$ ). Pairwise comparisons revealed a significant  
30 effect for time between  $T_0$ - $T_{100}$  ( $p = 0.002$ ),  $T_{20}$ - $T_{100}$  ( $p < 0.001$ ),  $T_{40}$ - $T_{100}$  ( $p = 0.006$ )  
31 and  $T_{60}$ - $T_{100}$  ( $p = 0.001$ ) for mean trunk flexion angle, between  $T_0$ - $T_{100}$  ( $p = 0.003$ ),  $T_{20}$ -  
32  $T_{100}$  ( $p < 0.001$ ),  $T_{40}$ - $T_{100}$  ( $p = 0.009$ ) and  $T_{60}$ - $T_{100}$  ( $p = 0.003$ ) for maximum trunk

1 flexion angle, and between T<sub>0</sub>-T<sub>100</sub> ( $p = 0.001$ ), T<sub>20</sub>-T<sub>100</sub> ( $p < 0.001$ ), T<sub>40</sub>-T<sub>100</sub> ( $p =$   
2 0.003) and T<sub>60</sub>-T<sub>100</sub> ( $p = 0.002$ ) for minimum trunk flexion angle.

3 Trunk medio-lateral sway maximum angle and joint ROM in the coronal plane  
4 increased (maximum angle,  $p = 0.013$ ; ROM,  $p < 0.001$ ), and minimum angle decreased  
5 significantly throughout the exercise bout ( $p = 0.002$ ). Pairwise comparisons showed a  
6 significant effect for time between T<sub>0</sub>-T<sub>100</sub> ( $p = 0.003$ ) for maximum trunk medio-lateral  
7 sway angle, between T<sub>0</sub>-T<sub>100</sub> ( $p = 0.005$ ) and T<sub>20</sub>-T<sub>80</sub> ( $p = 0.009$ ) for trunk medio-lateral  
8 sway ROM, and between T<sub>0</sub>-T<sub>80</sub> ( $p = 0.022$ ) and T<sub>40</sub>-T<sub>80</sub> ( $p = 0.043$ ) for trunk medio-  
9 lateral sway minimum angle. In addition, coronal plane left and right hip joint ROM and  
10 right hip minimum angle (i.e., hip abduction/adduction) and right knee joint ROM (i.e.,  
11 knee valgus/varus) increased significantly throughout the exercise bout (left hip ROM,  
12  $p = 0.004$ ; right hip ROM,  $p = 0.001$ ; right hip minimum angle,  $p = 0.036$ ; right knee  
13 ROM,  $p = 0.020$ ). Pairwise comparisons showed a significant effect for time between  
14 T<sub>0</sub>-T<sub>80</sub> ( $p = 0.006$ ) and T<sub>20</sub>-T<sub>80</sub> ( $p = 0.021$ ) for right hip ROM.

15  
16  
17  
18  
19  
20  
21  
22





**Figure 4.2** Trunk flexion and medio-lateral sway, hip abduction/adduction and knee valgus/varus joint angles during a high-intensity, constant-load cycling bout.

Coronal plane left (A) and right (B) hip (abduction/adduction), coronal plane left (C) and right (D) knee (valgus/varus), and sagittal plane trunk flexion (E) and coronal plane trunk medio-lateral sway (F) joint angles expressed as a function of pedal cycle duration for  $T_0$  (solid black line),  $T_{20}$  (broken black line),  $T_{40}$  (solid blue line),  $T_{60}$  (broken blue line),  $T_{80}$  (solid red grey line) and  $T_{100}$  (broken red grey line) for all participants during the time to exhaustion constant-load cycling tests. Data are the average from 10 pedal cycles at each time point (the first 10 pedal cycles at  $T_0$  and the final 10 pedal cycles at  $T_{20}$ - $T_{100}$ ). Significant kinematic changes were observed from  $\sim T_{60}$  in the sagittal plane and  $\sim T_{80}$  in the coronal plane (see 4.4.4 for more detail).



**Table 4.1** Kinematic changes at the trunk, hip, knee and ankle joints in the sagittal and coronal planes during a high-intensity, constant-load cycling bout for ten pedal cycles at T<sub>0</sub>, T<sub>20</sub>, T<sub>40</sub>, T<sub>60</sub>, T<sub>80</sub> and T<sub>100</sub>.

Joint	Side	Kinematic variable	Cycle duration					
			T <sub>0</sub>	T <sub>20</sub>	T <sub>40</sub>	T <sub>60</sub>	T <sub>80</sub>	T <sub>100</sub>
<b>Sagittal Plane</b>								
Hip	Left	Mean angle (°)	66.6 ± 6.8	68.1 ± 7.3	68.3 ± 5.1	67.8 ± 6.3	67.6 ± 6.2	65.2 ± 6.2
		Maximum angle (°)	90.1 ± 8.1	91.1 ± 7.3	91.7 ± 5.4	91.3 ± 6.7	91.3 ± 7.4	89.4 ± 7.5
		Minimum angle (°)	42.4 ± 6.9	44.0 ± 8.4	43.6 ± 5.8	43.5 ± 6.6	43.2 ± 6.9	40.2 ± 6.6
		ROM (°)	47.9 ± 4.5	47.1 ± 2.9	48.1 ± 2.9	47.8 ± 3.1	48.1 ± 3.5	49.1 ± 4.7
	Right	Mean angle (°)	63.3 ± 4.6	63.3 ± 5.4	65.7 ± 4.8	63.9 ± 5.5	64.2 ± 4.9	64.9 ± 5.5
		Maximum angle (°)	88.8 ± 5.3	89.2 ± 6.2	91.3 ± 5.7	90.1 ± 6.8	89.9 ± 5.4	90.7 ± 5.6
		Minimum angle (°)	41.1 ± 5.0	41.4 ± 5.6	42.6 ± 5.1	41.3 ± 5.4	41.8 ± 5.5	41.3 ± 5.1
		ROM (°)	47.8 ± 3.0	47.8 ± 2.9	48.7 ± 2.9	48.7 ± 4.0	48.1 ± 3.5	49.6 ± 4.2
Knee	Left	Mean angle (°)	72.7 ± 7.5	73.7 ± 7.4	72.7 ± 8.6	72.8 ± 8.3	72.2 ± 9.0	70.2 ± 6.3
		Maximum angle (°)	110.3 ± 8.4	111.7 ± 8.7	110.5 ± 8.5	112.9 ± 9.2	110.5 ± 9.4	109.5 ± 8.3
		Minimum angle (°)	28.9 ± 7.7	29.2 ± 8.0	28.2 ± 8.1	27.2 ± 9.2	27.4 ± 8.6	24.8 ± 6.6
		ROM (°)	81.3 ± 6.4	82.5 ± 6.3	82.2 ± 3.2	85.7 ± 10.0	83.1 ± 6.8	84.7 ± 7.5
	Right	Mean angle (°)	68.7 ± 6.5	67.8 ± 7.5	67.4 ± 7.6	66.8 ± 8.4	66.7 ± 7.7	67.6 ± 6.5
		Maximum angle (°)	111.7 ± 7.8	110.4 ± 8.3	110.2 ± 8.6	109.3 ± 10.0	110.2 ± 1.0	110.6 ± 7.5
		Minimum angle (°)	27.7 ± 6.5	26.2 ± 7.3	25.3 ± 7.5	24.6 ± 8.0	23.7 ± 7.5	24.3 ± 5.7
		ROM (°)	83.9 ± 6.4	84.2 ± 4.4	84.9 ± 5.3	84.7 ± 6.4	86.5 ± 7.1	86.3 ± 7.4

**Table 5.1** Cont.

Joint	Side	Kinematic variable	Cycle duration					
			T <sub>0</sub>	T <sub>20</sub>	T <sub>40</sub>	T <sub>60</sub>	T <sub>80</sub>	T <sub>100</sub>
<b>Sagittal Plane</b>								
Ankle	Left	Mean angle (°)	0.7 ± 9.1	1.3 ± 8.3	2.1 ± 6.7	2.2 ± 8.0	2.7 ± 8.4	4.5 ± 7.3
		Maximum angle (°)	12.8 ± 9.3	15.6 ± 6.7	15.0 ± 4.5	15.9 ± 3.9	16.1 ± 5.3	18.3 ± 5.0
		Minimum angle (°)	-10.5 ± 10.6	-11.1 ± 13.2	-9.9 ± 11.7	-10.0 ± 14.7	-8.6 ± 13.9	-13.0 ± 26.2
		ROM (°)	23.3 ± 8.5	26.7 ± 10.2	24.9 ± 10.6	25.9 ± 11.9	24.7 ± 10.4	26.2 ± 12.0
	Right	Mean angle (°)	0.5 ± 5.0	3.1 ± 4.3	2.6 ± 4.7	2.9 ± 5.3	4.6 ± 5.8	3.1 ± 4.5
		Maximum angle (°)	12.2 ± 5.4	14.7 ± 5.0	13.9 ± 3.9	14.7 ± 4.4	16.5 ± 4.2	15.8 ± 4.5
		Minimum angle (°)	-9.4 ± 5.7	-6.9 ± 8.6	-7.8 ± 8.0	-7.3 ± 8.6	-5.8 ± 8.4	-7.2 ± 6.4
		ROM (°)	21.7 ± 5.3	21.6 ± 6.1	21.7 ± 6.7	22.0 ± 7.1	22.3 ± 6.0	23.0 ± 7.4
Trunk		Mean angle (°)*	39.2 ± 6.1	39.7 ± 7.2	39.3 ± 6.5	41.5 ± 7.2	41.8 ± 6.5	48.6 ± 5.6
		Maximum angle (°)*	40.3 ± 6.0	41.0 ± 7.4	40.5 ± 6.7	43.0 ± 7.5	43.2 ± 6.7	50.5 ± 5.8
		Minimum angle (°)*	37.8 ± 6.2	38.3 ± 6.9	37.9 ± 6.3	40.1 ± 7.1	40.5 ± 6.4	47.0 ± 5.4
		ROM (°)	2.6 ± 1.0	2.6 ± 1.2	2.5 ± 0.7	3.0 ± 1.4	2.7 ± 1.3	3.5 ± 1.4
<b>Coronal Plane</b>								
Hip	Left	Mean joint angle (°)	-3.6 ± 3.0	-2.7 ± 2.8	-3.9 ± 3.1	-4.5 ± 3.3	-3.6 ± 2.4	-8.8 ± 9.4
		Maximum angle (°)	-0.9 ± 3.1	-0.4 ± 2.3	-0.2 ± 3.0	-0.6 ± 3.5	-0.6 ± 2.7	-4.7 ± 9.6
		Minimum angle (°)	-6.4 ± 3.0	-5.9 ± 3.5	-7.7 ± 3.4	-8.4 ± 3.6	-7.9 ± 2.7	-13.0 ± 9.5
		ROM (°)*	5.5 ± 1.0	6.3 ± 2.0	7.5 ± 2.1	7.7 ± 2.2	8.5 ± 2.4	8.3 ± 2.6

**Table 4.1** Cont.

Joint	Side	Kinematic variable	Cycle duration					
			T <sub>0</sub>	T <sub>20</sub>	T <sub>40</sub>	T <sub>60</sub>	T <sub>80</sub>	T <sub>100</sub>
Coronal Plane								
Hip	Right	Mean angle (°)	-1.0 ± 3.9	-0.5 ± 3.6	-2.4 ± 3.8	-0.5 ± 2.6	-1.2 ± 4.2	-7.2 ± 9.8
		Maximum angle (°)	2.1 ± 4.3	3.0 ± 4.0	1.7 ± 3.1	3.2 ± 2.6	3.5 ± 4.1	-2.8 ± 9.9
		Minimum angle (°)*	-3.6 ± 3.6	-3.2 ± 3.1	-5.7 ± 4.4	-3.8 ± 2.9	-4.0 ± 8.8	-9.2 ± 8.4
		ROM (°)*	5.8 ± 1.8	6.2 ± 2.0	7.4 ± 1.9	7.0 ± 2.4	8.8 ± 1.4	8.4 ± 2.0
Knee	Left	Mean angle (°)	16.8 ± 7.5	16.5 ± 7.9	17.6 ± 8.2	16.2 ± 13.3	15.7 ± 10.0	17.4 ± 8.9
		Maximum angle (°)	24.9 ± 9.0	25.6 ± 10.1	27.1 ± 10.3	26.1 ± 16.2	25.1 ± 12.0	28.0 ± 11.0
		Minimum angle (°)	5.1 ± 6.0	3.2 ± 6.2	4.5 ± 5.5	4.1 ± 9.3	3.6 ± 6.9	3.7 ± 6.9
		ROM (°)	19.8 ± 6.9	22.4 ± 9.1	22.7 ± 7.3	22.1 ± 10.5	21.5 ± 7.3	24.3 ± 9.7
	Right	Mean angle (°)	16.3 ± 19.4	14.0 ± 16.8	12.9 ± 17.8	13.2 ± 16.6	15.5 ± 18.3	19.6 ± 18.9
		Maximum angle (°)	28.0 ± 18.7	27.1 ± 15.1	24.5 ± 16.0	24.9 ± 14.8	27.7 ± 17.8	33.6 ± 18.2
		Minimum angle (°)	3.1 ± 17.7	0.9 ± 15.6	0.5 ± 17.0	1.4 ± 16.3	3.1 ± 17.1	4.2 ± 17.0
		ROM (°)*	24.9 ± 8.6	26.3 ± 9.7	23.9 ± 5.7	23.6 ± 6.3	24.6 ± 7.8	29.4 ± 9.1
Trunk		Mean angle (°)	-0.2 ± 3.6	-1.0 ± 3.5	-0.2 ± 2.7	-1.2 ± 2.6	-1.5 ± 2.6	-0.5 ± 3.2
		Maximum angle (°)*	3.6 ± 4.8	3.3 ± 3.3	5.1 ± 3.2	4.1 ± 3.2	4.6 ± 2.9	6.6 ± 4.5
		Minimum angle (°)*	-4.4 ± 2.6	-5.3 ± 4.0	-6.2 ± 3.0	-6.8 ± 2.7	-8.0 ± 3.1	-7.8 ± 4.3
		ROM (°)*	8.0 ± 3.3	8.6 ± 2.1	11.3 ± 3.3	10.9 ± 2.7	12.6 ± 3.1	14.4 ± 5.3

Data are mean ± SD for nine participants.

ROM, range of motion.

\* Main effect of time ( $p < 0.05$ ).

#### 4.4.5 Temporal relationships

$T_{p,twitch}$  magnitude, which was used as an indicator of peripheral fatigue, followed a sigmoidal pattern; it increased from  $T_0$ - $T_{20}$  then progressively decreased to  $T_{100}$ . Impairments in muscle function therefore occurred prior to significant kinematic changes at the trunk in the sagittal plane, which occurred from 60% of the time to task failure. Indicators of central facilitation (i.e. peak EMG amplitudes of VM and GMax) increased throughout the exercise bout and GMax activation was significant from  $T_{80}$ , which therefore occurred in parallel with kinematic alterations at the trunk, hip and knee joints in the coronal plane, which became significant from 80% of the time to task failure.

### 4.5 Discussion

The purpose of the present study was to describe the temporal relationship between kinematic changes and neuromuscular fatigue during high-intensity, constant-load cycling. The main findings were that: i) significant trunk kinematic changes were observed in the sagittal plane from 60% of the time to task failure, and were thus notable at or after the point of significant peripheral fatigue development, and ii) significant changes at the trunk, hip and knee joints in the coronal plane were observed from 80% of the time to task failure, which therefore paralleled the facilitation of central motor drive during the cycling task. These observations are suggestive that the effects of peripheral fatigue may lead to sagittal plane alterations, whilst coronal plane adjustments occur in parallel with central facilitation, and/or are only observed when a more substantial level of peripheral fatigue accumulates. Therefore, kinematic changes in the sagittal and coronal plane are temporally dissociated, and may be partly related to the site of fatigue (i.e., peripheral or central) induced by the exercise bout.

Within the present study, sagittal plane joint angle changes were observed only at the trunk (Table 4.1 and Figure 4.2). These results are somewhat in opposition to previous studies that have reported changes in hip (37, 87, 266) and knee (38) joint kinematics during 60-min time trial (266), and incremental (37) and constant-load (38, 87) cycling to exhaustion. A further contradiction (37, 38, 153, 192, 266) was that no changes were observed in the magnitude of ankle dorsiflexion ROM throughout the trial. It is possible that differences in cadence and/or power-duration profiles of the cycling task may contribute to the differential findings, and warrant further investigation. Despite this, a progressive increase in trunk flexion occurred from 60% of

1 the time to task failure, which possibly reflects a fatigue-mediated response to maintain  
2 the necessary propulsive force during the down stroke. Indeed, such changes in sagittal  
3 plane kinematics were evident at or after significant peripheral fatigue accumulation,  
4 and thus may consequently act as an efficient way to recruit previously inactive  
5 muscle(s) without compromising pedal force application efficiency. Indeed, an increase  
6 in trunk flexion increases the length of the muscles spanning the hip and knee joints (20,  
7 265, 299), which will affect force production during the propulsive phase of the pedal  
8 cycle. Increases in peak force during the down-stroke have previously been observed  
9 during cycling in positions of considerable trunk flexion (i.e. dropped or aerodynamic  
10 postures) and are associated with alterations in lower limb muscle activity (i.e. increases  
11 in VM and GMax activity) (88). Consequently, the increase in VM and GMax EMG  
12 observed in the present study was likely to be associated with the progressive increase  
13 in trunk flexion angle. Furthermore, given that the increase in lower limb EMG  
14 (Chapter 3) was hypothesised to be a compensatory mechanism for the increase in  
15 peripheral fatigue, it appears that a change in muscle activation patterns and proximal  
16 limb kinematics is necessary to maintain performance and prevent task failure during  
17 high-intensity, constant-load cycling.

18 In the coronal plane, left and right hip and right knee joint ROM (i.e., hip  
19 abduction/adduction and knee valgus/varus) and trunk medio-lateral sway maximum  
20 and minimum angles and ROM increased significantly towards the end of exercise  
21 (Table 4.1 and Figure 4.2). These results indicate that the participants progressively  
22 changed their cycling technique to a more abducted hip-varus knee position and  
23 increased the degree of side-to-side flexion of the trunk as fatigue accumulated. These  
24 changes were evident once central facilitation was significant, or at least when a more  
25 substantial level of peripheral fatigue accumulated. At present, only two studies have  
26 examined the effects of fatiguing cycling on technique changes in the coronal plane (87,  
27 266). The increase in hip abduction/adduction observed in the present study is in  
28 agreement with the study of Dingwell et al. (2008), who observed an increase in knee  
29 splay during fatiguing high-intensity cycling (87). However, an increased axial rotation  
30 of the shank, without a change in coronal plane kinematics, has also been observed  
31 towards the end of a 60-min constant-load cycling test (266). It should be noted,  
32 however, that changes in kinematics were measured only during the drive phase of the  
33 pedal stroke (i.e. from TDC to 180°) and not throughout the entire pedal revolution, as  
34 in the current study and that of Dingwell et al. (87).

1           It is generally considered that a break-down or change in movement technique  
2 during exercise will have a negative influence on the efficiency of motion and thus task  
3 performance (173). Indeed, in cycling the kinematic changes observed may influence  
4 the distribution of force throughout the pedal cycle, particularly for those changes that  
5 occurred in the coronal plane. It was interesting to note that a greater degree of trunk  
6 medio-lateral sway toward the left was observed from the start of the test to 80% of the  
7 time to task failure. It is believed that this increase in trunk sway was the result of  
8 participants shifting their centre of gravity over the pedal during each corresponding  
9 down-stroke, in an attempt to increase the down-stroke force produced. Furthermore,  
10 the greater degree of medio-lateral sway to the left is likely to be due to participants  
11 attempting to assist or maintain power output with their weakest leg. Indeed, eight of  
12 the nine participants reported that their right leg was their dominant leg, which is of  
13 interest as leg dominance has not typically been considered as a concern in cycling  
14 research. It could be speculated that the ability to maintain an effective tangential pedal  
15 force towards the end of the exercise bout would be compromised with increasing  
16 lateral hip, knee and trunk motion. Thus, the ability to maintain performance at the set  
17 workload and cadence would have become more difficult, ultimately resulting in the  
18 termination of exercise. However, while an increased lateral motion across multiple  
19 joints may be undesirable with regards to pedal force application, and thus cycling  
20 efficiency, this kinematic strategy may have been important for the participants to  
21 maintain the necessary workload.

22           The significant kinematic changes observed in the present study may assist in  
23 delaying task failure by increasing the recruitment of muscles (or motor units within  
24 muscles) that were not previously highly active. This is particularly true for those  
25 muscles or motor units used to control lateral motion of the thigh and trunk. Previous  
26 reports have shown an increase in the recruitment of adductors longus and magnus  
27 during incremental cycling to exhaustion (312), and an increase in adductor longus  
28 activation during constant-load cycling in an exaggerated position of knee joint  
29 adduction (35). It is therefore likely that the alterations in coronal plane hip and knee  
30 joint kinematics observed in this study were associated with the development of fatigue  
31 and alterations in the recruitment of hip adductors/abductors. As well as increasing  
32 recruitment of additional muscle groups, it is also probable that motor units within  
33 various regions of individual muscles were differentially activated during the fatiguing  
34 cycling task (309). In support of this, changes in the relative activity between different  
35 regions of GM and GL have been shown during cycling, and relate to changes in the

1 torque-velocity demands of the task (309). As such, the progressive change in cycling  
2 kinematics observed in the present study could have induced beneficial compensatory  
3 muscle activation strategies that were reflected in either (i) region-specific muscle  
4 activation changes, and/or (ii) recruitment of additional muscles, i.e. adductor longus  
5 and magnus, beyond those lower limb muscles that are typically considered to  
6 contribute to the synergist muscle groups used in cycling (151, 246, 310). It should also  
7 be noted that kinematic and muscle activation changes occurred without alterations in  
8 the temporal pattern of muscular activity (see Chapter 3). It is therefore plausible that  
9 either the increase in VM and GMax EMG, and/or differences in the rates of fatigue  
10 between muscles were sufficient to cause the observed changes in cycling kinematics.

11 In conclusion, the results of this study revealed that sagittal plane kinematic  
12 alterations at the trunk were observed from 60% of the time to task failure and were thus  
13 notable at, or secondary to, significant peripheral fatigue development. In addition,  
14 kinematic changes in the coronal plane at the trunk, hip and knee joints occurred from  
15 80% of the time to task failure and thus progressed together with increases in central  
16 motor drive until task failure, or at least when a more substantial level of peripheral  
17 fatigue accumulated. These findings indicate that kinematic changes in the sagittal and  
18 coronal planes are temporally dissociated, and could be related to the site of exercise-  
19 induced fatigue. Furthermore, it appears that potentially beneficial compensatory  
20 increases in lower limb muscle activity occurred as a result of a change in body position  
21 (i.e. greater trunk lean and medio-lateral sway, hip abduction and knee varus angle)  
22 when the participants approached exhaustion. It is hypothesised that, consciously or  
23 sub-consciously, participants chose to alter their cycling technique to compensate for  
24 the level of fatigue experienced during the cycling bout. Further research is necessary to  
25 understand whether these sagittal and coronal plane kinematic changes occur alongside  
26 peripheral fatigue and central facilitation in other exercises, and particularly during  
27 other high-intensity and repetitive tasks. Overall, this study provides novel insights in  
28 relation to how the CNS responds to fatigue accumulation by employing temporally  
29 dissociated kinematic strategies and therefore modulating movement patterns during a  
30 fatiguing exercise task.

31  
32

## CHAPTER FIVE

### Effects of real-time, visual kinematic feedback on the time taken to reach task failure during high-intensity, constant-load cycling

#### 5.1 Abstract

**Purpose:** To examine the effects of real-time, kinematic feedback for trunk flexion, trunk medio-lateral sway (lateral flexion) and hip abduction/adduction on the time taken to reach task failure during high-intensity, constant-load cycling. **Methods:** Nine well-trained male cyclists performed five testing sessions, including (i) an incremental cycling test to exhaustion, (ii) a time to exhaustion (TTE) test with no feedback and (iii) three TTE tests with kinematic feedback provided for trunk flexion ( $TTE_{Tflex}$ ), trunk medio-lateral sway ( $TTE_{Tsway}$ ) and hip abduction/adduction ( $TTE_{Habd/add}$ ). All TTE tests were performed at 90%  $P_{max}$  (power output at  $VO_{2max}$ ) at a constant cadence. Real-time, kinematic feedback was provided via a laser beam projected onto a screen, and the participants were instructed to limit the movement of a specific joint segment(s) (i.e. the trunk ( $TTE_{Tflex}$  and  $TTE_{Tsway}$  tests) or left and right thigh ( $TTE_{Habd/add}$  test)) within a set target zone; thus they were asked to not to vary joint kinematics as the trials progressed. Lower (i.e. hip, knee and ankle joint) and upper (i.e. trunk) limb kinematics in the sagittal and coronal planes were assessed during cycling at the start ( $T_0$ ) and at 20, 40, 60, 80 and 100% ( $T_{20}-T_{100}$ ) of the time taken to reach task failure, to quantify joint kinematic changes throughout the trials. Participants who failed to meet predetermined criteria to ensure joint segments moved within the target zone were excluded from the kinematic analyses; three participants were excluded from both the  $TTE_{Tflex}$  and  $TTE_{Tsway}$  tests ( $n = 6$ ) and two participants from the  $TTE_{Habd/add}$  test ( $n = 7$ ). **Results:** The times taken to reach task failure were not significantly different between the feedback and non-feedback TTE test conditions for participants who met the inclusion criteria or for all participants in the  $TTE_{Tflex}$  and  $TTE_{Habd/add}$  tests ( $p > 0.05$ ). However, task failure occurred earlier during the trunk medio-lateral sway feedback condition when all participants were included ( $p < 0.05$ ). Despite being given feedback, changes in joint kinematics were similar across all test conditions; significant alterations were observed at the trunk and knee joints in the sagittal plane and at the hip and knee joints in the coronal plane ( $p < 0.05$ ). With the provision of trunk flexion and hip



1 abduction/adduction feedback no changes were observed in knee flexion joint angles or  
2 range of motion (ROM) ( $p > 0.05$ ). Trunk flexion feedback resulted in significant  
3 increases in left hip flexion and trunk medio-lateral sway ROM ( $p < 0.05$ ). In addition,  
4 increases in right hip flexion ROM were observed in the trunk medio-lateral sway  
5 feedback test ( $p < 0.05$ ). **Conclusions:** These results indicate that regardless of whether  
6 or not well-trained cyclists are able to control the level of kinematic variability when  
7 fatigued, acute exposure to real-time kinematic feedback to limit trunk or hip movement  
8 during high-intensity cycling may influence cycling kinematics (i.e. technique) and, in  
9 some cases (e.g. trunk medio-lateral sway), may reduce performance.  
10  
11  
12

## 5.2 Introduction

The human neuromuscular system can utilise a variety of neural strategies to execute a motor skill (30, 235, 282). In the presence of fatigue, the neural strategies typically employed include alterations in joint rigidity and muscle activation, which, in turn, changes movement technique (i.e., joint kinematics) and increases motor variability (282). It is likely that these changes compensate for fatigue by changing and/or increasing the variation in kinematic patterns to limit the use of specific muscle(s) groups in an attempt to continue exercise. Such strategies are therefore crucial for optimising and preserving performance in fatiguing exercise tasks, from manual labouring skills (e.g., lifting (41, 49, 279, 280, 300), hammering (69, 71) and sawing (70, 131)) to complex sporting movements (18, 87, 133, 219, 271). During cycling, these compensatory kinematic adjustments include increases in trunk flexion (87), hip and knee extension (37, 38, 87, 266), knee valgus/varus (87) and ankle dorsiflexion (37, 38, 192, 266). Indeed, in Chapter 4 increases in trunk flexion, trunk medio-lateral sway, hip abduction/adduction and knee valgus/varus were reported during a fatiguing high-intensity, constant-load exercise task (i.e. cycling at 90% of MAP). The ability to utilise these compensatory strategies will inherently alter the motor strategies employed by the neuromuscular system, and are considered to assist in preventing, delaying or alleviating neuromuscular fatigue and maintaining exercise performance (25, 282).

Nonetheless, sports coaches and health and safety practitioners often encourage the maintenance of a consistent technique in order to maintain movement efficiency and possibly avoid injury (25). However it could be speculated that consistency of technique may prevent the neuromuscular system from using compensatory strategies to cope with fatigue, and therefore compromise task performance (282). While, a number of studies have examined the influence of kinematic manipulation (i.e. trunk position (aerodynamic posture)) on power production (127, 299, 314), physiological responses (20, 101, 127, 231, 274, 314) and changes in lower limb muscle activation and joint kinematics (58, 88, 265) during exercises such as cycling, the kinematic adjustments made are often associated with the use of an aerodynamic posture and are not necessarily the kinematic changes that occur in response to fatigue (37, 38, 87, 192, 266). Therefore, there is a paucity of research examining the influence of technique alteration and/or increasing kinematic variability on the progression of fatigue during cycling.

Systematically increasing or decreasing kinematic variation through the use of augmented knowledge of performance feedback would allow for an examination of these changes on fatigue progression and the ability to continue task performance. Augmented knowledge of performance feedback is a type of external feedback that is focused on the movement pattern and how the task is performed (for review see (269)). It is typically provided via visual or verbal media and is more effective in eliciting immediate improvements in performance (i.e., compared to skill retention, or learning) when the desired movement involves multiple joint segments and is presented concurrently to the learner. Concurrent feedback is continuously provided to the learner as a real-time representation of the coordinative pattern of the movement being performed and can thus indicate transitory errors in performance. The use of knowledge of performance feedback training programmes to optimise pedal force application patterns (46, 215, 260, 297) and cadence selection (62) in cycling have been shown to improve performance. Consequently, knowledge of performance feedback might be an ideal way to entrain optimal kinematic variability to potentially delay fatigue progression and optimise exercise performance.

Given the above, the present study investigates the effect of visual knowledge of performance feedback to control the variation in trunk flexion, trunk medio-lateral sway and hip abduction/adduction during high-intensity, constant-load cycling to exhaustion. These kinematic variables were chosen as they were identified in Chapter 4 to be the most significant kinematic adjustments observed during this type of cycling, and were thus deemed important compensatory joint kinematic strategies employed to deal with neuromuscular fatigue development. Given this, the purpose of the present study was to determine whether limiting the variation of trunk flexion, trunk medio-lateral sway and hip abduction/adduction using real-time, visual kinematic feedback influences the time taken to reach task failure during high-intensity, constant-load cycling.

### 5.3 Methods

### 5.3.1 Participants

Nine well-trained male cyclists (mean  $\pm$  SD; age  $36 \pm 7$  y, height  $179 \pm 5$  cm, body mass  $79.6 \pm 8.3$  kg,  $\text{VO}_{2\text{ max}}$   $55.0 \pm 3.1$  ml $\cdot$ kg $^{-1}\cdot$ min $^{-1}$ ) with  $6 \pm 6$  y of cycling experience and currently cycling  $356 \pm 98$  km $\cdot$ wk $^{-1}$  volunteered to participate in the study. Experienced cyclists were chosen because they were more likely to have superior

movement control, reflected in consistent kinematic and muscle recruitment patterns (56, 57, 59). The participants were asked to avoid strenuous exercise for 48 h, and the consumption of alcohol, caffeine or other stimulants for 12 h prior to all testing sessions. The participants were also requested to record and follow a similar diet in the 24 h period prior to all testing sessions (see Appendix K). The participants were informed of the study requirements and written consent was gained prior to participation (see Appendices F and H). All procedures were approved by the institution's Human Research Ethics Committee and conformed to the Declaration of Helsinki (see Appendix D).

### 5.3.2 Experimental protocol

The participants performed a total of five testing sessions that were separated by at least 48 h (average time to complete all testing sessions:  $20 \pm 8$  days). In the first session, participants were briefed about the study design and potential outcomes, and then performed an incremental cycling test to exhaustion. In the subsequent visit, the participants performed a high-intensity, constant-load cycling TTE test and were familiarised with the kinematic feedback for all three conditions. The three subsequent sessions were randomised (counterbalanced) and consisted of the same constant-load cycling TTE test, during which participants were provided with feedback on either trunk flexion ( $TTE_{Tflex}$ ), trunk medio-lateral sway ( $TTE_{Tsway}$ ) or hip abduction/adduction ( $TTE_{Habd/add}$ ) (described below). The time taken to reach task failure (i.e., volitional exhaustion;  $T_{100}$ ) was divided into 20% time periods ( $T_{20}$ – $T_{80}$ ) to allow for joint kinematics to be compared across time and between participants. All cycling tests were performed on an electromagnetically-braked cycle ergometer (Velotron, Racermate, USA), which was configured to resemble the participant's own bike set-up. The participants used their own pedals and clip-in cycling shoes. Prior to all tests, the participants also performed a warm-up that consisted of cycling for at least 5 min at  $1.25 \text{ W} \cdot \text{kg}^{-1}$  ( $95 \pm 5 \text{ W}$ ) followed by three 5-10 s maximal sprints with a self-paced active recovery.

Prior to each testing session the participants also completed the MFI psychometric questionnaire (278) to determine subjective feelings of fatigue (see Appendix J). All participants scored  $\geq 12$  for all test sessions ( $17.7 \pm 0.4$ ; average general fatigue score), indicating no/minimal 'general fatigue' (i.e. general expressions/subjective feelings of fatigue) (278). In addition, before performing any of

the experimental TTE trials, the participants were asked to complete a questionnaire to determine their performance expectations (see Appendix M). In brief, participants were provided with a written explanation of the potential benefits of either holding a ‘correct’ technique or changing technique during an exhausting cycling exercise bout, and were asked “Do you think that maintaining or holding ‘good cycling technique’, or changing cycling technique during cycling exercise is more beneficial to performance?”

#### **5.3.2.1 Incremental cycling test**

As described in Study 1 (Chapter 3).

#### **5.3.2.2 Constant-load cycling tests**

As described in Study 1 (Chapter 3); however the participants were instructed to continue cycling until their cadence dropped below 5% of their target cadence for longer than 3 s, or they were stopped by the experimenter because they consistently moved beyond the target zone for longer than 10 s during the  $TTE_{Tflex}$ ,  $TTE_{Tsway}$  and  $TTE_{Habd/add}$  tests (described below). In addition, the participants were provided with verbal encouragement to maintain movement within the target zone.

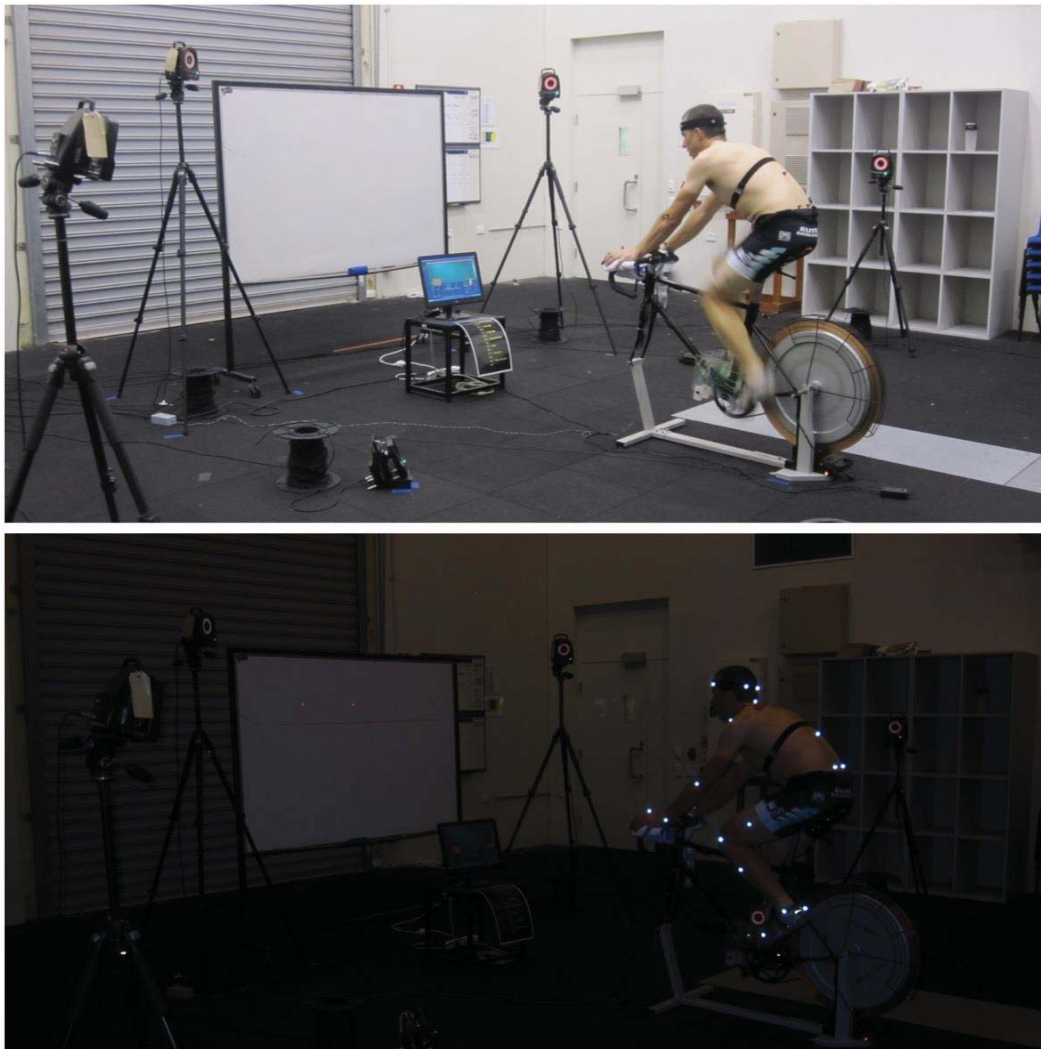
#### **5.3.2.3 Kinematics during cycling**

As described in Study 2 (Chapter 4); however only joint mean angles and ROM were calculated for all constant-load cycling tests.

#### **5.3.2.4 Kinematic feedback**

During the experimental constant-load cycling tests, real-time kinematic feedback for either trunk flexion, ( $TTE_{Tflex}$ ) trunk medio-lateral sway ( $TTE_{Tsway}$ ) or hip abduction/adduction ( $TTE_{Habd/add}$ ) (see Figure 5.1) was continuously displayed in front of the participant. This type of feedback allows the participant to maintain their joint segment motions within a target zone in real time, and consequently adjust their cycling technique during the exercise bout. The kinematic feedback was provided via a laser beam, whereby laser pointers were attached to the participant and the displacement of the lasers were projected onto a whiteboard positioned in front of the cycle ergometer. Participants were instructed to control the movement of each joint segment, i.e. the trunk ( $TTE_{Tflex}$  and  $TTE_{Tsway}$  tests) or thigh ( $TTE_{Habd/add}$  test) in the sagittal ( $TTE_{Tflex}$  test) or coronal ( $TTE_{Tsway}$  and  $TTE_{Habd/add}$  tests) plane of motion. The use of laser beams

has previously been used to provide kinematic feedback when learning movement patterns of the hip and knee joints during a double-leg squat (325).



**Figure 5.1** Set-up for the provision of real-time, kinematic feedback during a high-intensity, constant-load cycling bout.

The participants were asked to keep the displacement of the laser beam within a ‘target zone’ throughout the test. The target zone was determined prior to each TTE feedback test, and encompassed the area in which the laser beam displayed during a 30-45 s period of cycling in the non-fatigued state ( $T_0$ ). For the  $TTE_{Tflex}$  and  $TTE_{Tsway}$  tests, two laser pointers (left and right) were positioned on the participant over the trapezius muscle, with the laser pointers lying parallel to the spinal column. For the  $TTE_{Habd/add}$  test the participant wore modified tubular support bandages (13 cm in length) over the left and right thighs, positioned superior to the upper border of the patella. The laser

1 pointers were placed in line with the femur inside a pocket-like compartment on the  
2 bandage on the dorsal side of the thigh and were elevated using 2 cm blocks to ensure  
3 that the laser beam projected onto the whiteboard during pedalling. The target zone for  
4 trunk flexion ( $TTE_{Tflex}$  test) kinematic feedback was the area above a horizontal line that  
5 was drawn across the width of the whiteboard, and represented the position of the trunk  
6 in the sagittal plane (see Appendix N; photo A). The target zone for trunk medio-lateral  
7 sway ( $TTE_{Tsway}$  test) and hip abduction/adduction ( $TTE_{Habd/add}$  test) kinematic feedback  
8 was the area between two vertical lines (one target zone for trunk medio-lateral sway  
9 feedback, and two target zones for hip abduction/adduction feedback), which were  
10 drawn down the length of the whiteboard and represented the position of the trunk (see  
11 Appendix N; photo B) or the left and right leg/thigh in the coronal plane (see Appendix  
12 N; photo C).

13 In session two, after completion of the TTE test with no feedback and prior to  
14 each TTE feedback test, participants practiced using the feedback to control the  
15 movement of the trunk and/or thigh (all kinematic variables were practiced in session  
16 two and only the variable to be tested on that day in sessions 3-5). Prior to the tests,  
17 detailed instructions were given to the participants to ensure they were aware of the task  
18 goal and what was expected. The participants practiced using the feedback at 50-W  
19 increments from 100-250 W, and then at their individual test power output (90% of  
20  $P_{max}$ ); the participants were told that they could practice at each workload for as long as  
21 required to feel comfortable and accustomed to the feedback. It was intended that this  
22 practice period would provide adequate time for the participants to become familiar  
23 with the correct position of the trunk or thigh segment during the cycling exercise, but  
24 separate sessions were not allowed so that ‘training’ with the feedback could not occur.  
25 Thus, the aim was to determine whether acute exposure to kinematic feedback  
26 influenced cycling technique and performance. Prior to the experimental sessions, the  
27 experimenter judged that the participant was able to control the movement of the trunk  
28 or thigh within the target zone and confirmed with the participant that they felt  
29 comfortable using the feedback.

30

### 31 **5.3.3 Data analyses**

32 Kinematic data were analysed post hoc to determine whether the participants  
33 were able to use the feedback correctly during each condition, i.e. they controlled the  
34 movement of the trunk or thigh segment to a similar degree of that during the non-



fatigued state at the start of the trial (when the target zone was set); it was assumed that this represented movement within the target zone. In addition to the standard analyses (described below), separate analyses were done excluding participants who were unable to meet the set movement pattern inclusion criteria. For the  $TTE_{Tflex}$  test the criterion was set that the change in mean trunk flexion angle must not be greater than that which occurred during the TTE test with no feedback. For the  $TTE_{Tsway}$  and  $TTE_{Habd/add}$  tests the criterion was set that the change in trunk medio-lateral sway or hip abduction/adduction ROM must not be  $\geq 15\%$  of that which occurred during the TTE test with no feedback (for the  $TTE_{Habd/add}$  test the criterion needed to be met for both the leg and right legs). This analysis was necessary given that: (i) the study objectives were to control the motion of the trunk and thigh segment to that which would occur in a non-fatigued state, and (ii) the real-time subjective assessment by the experimenter may not have recognised small changes in joint angles during the test.

Subsequently it was determined that six of the nine participants were able to correctly minimise movement of the trunk ( $TTE_{Tflex}$  and  $TTE_{Tsway}$  tests,  $n = 6$ ), and seven of the nine participants were able to minimise movement of the hip joints ( $TTE_{Habd/add}$  test,  $n = 7$ ) to a similar degree of that during the non-fatigued state at the start of the trial, i.e. within the target zones, throughout the duration of the test. Of the participants who met the inclusion criteria in the trunk flexion condition ( $n = 6$ ), the change in mean trunk flexion angle from the start of the trial to at fatigue prior to volitional exhaustion was  $4.9 \pm 4.5^\circ$  ( $T_0$ :  $34.3 \pm 4.6^\circ$ ;  $T_{100}$ :  $39.2 \pm 7.3^\circ$ ) in the TTE test with no feedback, compared to  $2.1 \pm 2.8^\circ$  ( $T_0$ :  $33.2 \pm 5.3^\circ$ ;  $T_{100}$ :  $35.5 \pm 5.5^\circ$ ) in the  $TTE_{Tflex}$  test ( $p = 0.034$ ). Of the participants who met the inclusion criteria in the trunk medio-lateral sway condition ( $n = 6$ ), the change in trunk medio-lateral sway ROM from the start of the trial to at fatigue prior to volitional exhaustion was  $2.2 \pm 2.3^\circ$  ( $T_0$ :  $3.3 \pm 1.3^\circ$ ;  $T_{100}$ :  $5.6 \pm 1.8^\circ$ ) in the TTE test with no feedback, compared to  $1.7 \pm 2.1^\circ$  ( $T_0$ :  $2.8 \pm 0.7^\circ$ ;  $T_{100}$ :  $4.5 \pm 2.1^\circ$ ) in the  $TTE_{Tsway}$  test ( $p > 0.05$ ). Of the participants who met the inclusion criteria in the hip abduction/adduction condition ( $n = 7$ ), the change in hip abduction/adduction ROM from the start of the trial to at fatigue prior to volitional exhaustion was  $2.9 \pm 2.0^\circ$  ( $T_0$ :  $7.1 \pm 2.2^\circ$ ;  $T_{100}$ :  $9.9 \pm 2.3^\circ$ ) for the left leg and  $2.9 \pm 1.5^\circ$  ( $T_0$ :  $5.9 \pm 1.7^\circ$ ;  $T_{100}$ :  $8.8 \pm 2.0^\circ$ ) for the right leg in the TTE test with no feedback, compared to  $2.0 \pm 1.0^\circ$  ( $T_0$ :  $6.5 \pm 2.6^\circ$ ;  $T_{100}$ :  $8.5 \pm 3.0^\circ$ ) for the left leg and  $1.4 \pm 1.8^\circ$  ( $T_0$ :  $5.4 \pm 1.9^\circ$ ;  $T_{100}$ :  $6.8 \pm 2.3^\circ$ ) for the right leg in the  $TTE_{Habd/add}$  test ( $p > 0.05$ ). Thus, even though some participants were unable to achieve the movement pattern criteria set,



there were few differences in the kinematic patterns of the group as a whole compared to the group that included only those who met the criteria.

#### 5.3.4 Statistical analyses

Data are expressed as mean values  $\pm$  SD. Times taken to reach task failure and RPE at task failure ( $T_{100}$ ) between the non-feedback and feedback ( $TTE_{Tflex}$ ,  $TTE_{Tsway}$  and  $TTE_{Habd/add}$ ) TTE tests were analysed using paired-samples t-tests. Effect sizes were calculated (SD/mean) on near significant effects ( $p < 0.10$ ). Kinematic variables for each constant-load cycling test were statistically analysed by one-way ANOVAs with repeated measures at each time point ( $T_0$ - $T_{100}$ ). All statistical analyses were performed using standard statistical software program (SPSS v 17 for Windows, SPSS, Inc., Chicago, IL, USA). Significance was set at  $p < 0.05$ .

### 5.4 Results

#### 5.4.1 Pre-testing task expectations questionnaire

The majority of participants (eight of nine) believed that either changing cycling technique (five participants), or both holding ‘good cycling technique’ and changing technique in certain situations can be beneficial (three participants) to cycling performance. Only one participant believed that holding a ‘good technique’ would be “more efficient and would require less energy”.

#### 5.4.2 Incremental cycling test

At completion of the incremental cycling test  $P_{max}$ , maximal heart rate and RPE were  $374 \pm 37$  W,  $180 \pm 5$  beats·min<sup>-1</sup> and  $19.0 \pm 1.0$ , respectively.

#### 5.4.3 Constant-load cycling tests

When including all participants in the analysis, the times taken to reach task failure ( $T_{100}$ ) in the  $TTE_{Tflex}$  and  $TTE_{Habd/add}$  tests were not different to that in the non-feedback condition ( $p > 0.05$ ) (Table 5.1). However, the  $TTE_{Tsway}$  test was significantly shorter than that of the TTE test with no feedback ( $p = 0.019$ ) (Table 5.1). When only those who met the movement pattern inclusion criteria were included, the times taken to reach task failure for all constant-load cycling tests with feedback ( $TTE_{Tflex}$ ,  $TTE_{Tsway}$  and  $TTE_{Habd/add}$  tests) were not different to the TTE test with no feedback ( $p > 0.05$ ).

(Table 5.1). Therefore, the mean reduction in the time taken to reach task failure in the  $TTE_{Tsway}$  test was no longer statistically significant ( $p = 0.092$ ), which was possibly more associated with the reduction in sample size, and thus statistical power (effect sizes for the  $TTE_{Tsway}$  test for all participants was 0.41 and for those who met the inclusion criteria was 0.44). The aerobic capacity, workloads and cadences attained by the participants are shown in Table 5.1. RPE for all participants increased throughout the exercise bout for all constant-load cycling tests, and at  $T_{100}$  the RPE for the TTE with no feedback,  $TTE_{Tflex}$ ,  $TTE_{Tsway}$  and  $TTE_{Habd/add}$  test conditions were  $19.8 \pm 0.4$ ,  $19.9 \pm 0.3$ ,  $19.7 \pm 0.5$ ,  $19.9 \pm 0.3$ , respectively ( $p > 0.05$ ).

**Table 5.1** Aerobic capacity, power output, cadence and the times taken to reach task failure for the time to exhaustion (TTE) test with no feedback and the TTE tests with real-time, kinematic feedback for trunk flexion (TTE<sub>Tflex</sub>), trunk medio-lateral sway (TTE<sub>Tsway</sub>) and hip abduction/adduction (TTE<sub>Habd/add</sub>) for all participants and for those who met the movement pattern inclusion criteria for each feedback condition.

	Time to exhaustion test condition							
	VO <sub>2max</sub> (ml·kg <sup>-1</sup> ·min <sup>-1</sup> )	Power output (W)	Power output (W·kg <sup>-1</sup> )	Cadence (rpm)	TTE (min)	TTE <sub>Tflex</sub> (min)	TTE <sub>Tsway</sub> (min)	TTE <sub>Habd/add</sub> (min)
All participants	55.0 ± 3.1	337 ± 33	3.39 ± 0.21	89 ± 6	7.40 ± 2.30	7.00 ± 3.10	6.40 ± 2.60*	7.00 ± 3.00
Met criterion								
Trunk flexion	53.9 ± 2.1	347 ± 33	3.34 ± 0.22	91 ± 6	8.00 ± 2.60	7.40 ± 3.70		
Trunk medio-lateral sway	53.8 ± 2.2	329 ± 27	3.33 ± 0.22	88 ± 6			6.90 ± 3.00	
Hip abduction/adduction	55.1 ± 3.4	334 ± 36	3.36 ± 0.20	89 ± 7	7.90 ± 2.60 6.90 ± 1.50			6.60 ± 1.90

Data are mean ± SD. All participants (n = 9); met criterion = participants who met the movement pattern inclusion criteria for each feedback condition: trunk flexion (n = 6), trunk medio-lateral sway (n = 6), hip abduction/adduction (n = 7).

Tflex, trunk flexion; Tsway, trunk medio-lateral sway; Hip abd/add, Hip abduction/adduction

\* TTE<sub>Tsway</sub> significantly different to the TTE test ( $p = 0.019$ ).

#### 5.4.4 Kinematics during cycling

Changes in joint kinematics were relatively similar for each of TTE tests with no feedback (i.e. for each corresponding feedback test; see Section 5.3.3). Increases in mean trunk flexion angles and ROM, and left knee flexion ROM were observed throughout all exercise tests ( $p < 0.05$ ), except in the hip abduction/adduction feedback condition where no significant increase in mean trunk flexion angle was observed ( $p > 0.05$ ). In the coronal plane, left and right hip abduction/adduction ROM, and left and right mean knee valgus/varus angles increased throughout the exercise bout in all tests ( $p < 0.05$ ). In addition, for the trunk flexion and medio-lateral sway feedback conditions, increases in right knee flexion ROM and mean angle (trunk flexion feedback condition only) and left knee valgus/varus ROM were observed ( $p < 0.05$ ). There was also a trend towards an increase in trunk medio-lateral sway ROM ( $p = 0.057$ ) for the trunk medio-lateral sway feedback condition. Thus, the cyclists allowed kinematic changes other than those that occurred during the TTE tests with no feedback. Joint kinematics at  $T_0$  and  $T_{100}$  are presented in Tables 5.2 – 5.4 (Appendices A – C).

With the provision of trunk flexion feedback (TTE<sub>Tflex</sub> test), no significant changes were observed in mean trunk and knee flexion angles or ROM, or left knee valgus/varus mean angles ( $p > 0.05$ ). However, significant increases in trunk medio-lateral sway and right knee valgus/varus ROM were observed ( $p < 0.05$ ). With the provision of trunk medio-lateral sway feedback (TTE<sub>Tsway</sub> test), increases in right hip flexion and abduction/adduction ROM ( $p < 0.05$ ), but no changes in right knee flexion and valgus/varus mean angles ( $p > 0.05$ ) were observed. With the provision of hip abduction/adduction feedback (TTE<sub>Habd/add</sub> test) there were no changes in trunk flexion, right hip abduction/adduction or left knee flexion ROM ( $p > 0.05$ ), however significant increases were observed in mean trunk flexion angles and left knee valgus/varus ROM ( $p < 0.05$ ). All other changes in joint kinematics during the kinematic feedback TTE tests were similar to the TTE test with no feedback. Joint kinematics at  $T_0$  and  $T_{100}$  are presented in Tables 5.2 – 5.4 (Appendices A – C).

### 5.5 Discussion

This study examined the effect of real-time, kinematic feedback on the time taken to reach task failure during high-intensity, constant-load cycling. The main findings of the present study were that: i) for all participants the provision of trunk and hip joint kinematic feedback either had no discernible (TTE<sub>Tflex</sub> and TTE<sub>Habd/add</sub> tests),

or a significant negative ( $TTE_{Tsway}$ ), impact on the time taken to reach task failure when compared to a condition where no feedback was provided, indicating that purposefully restricting kinematic variation may be detrimental to performance; ii) for participants who correctly used the kinematic feedback (i.e., maintained joint movement within the target zone), the times to task failure were not significantly different to the non-feedback condition; iii) these well-trained cyclists, with very consistent and well-learned kinematic patterns, found it difficult to reduce kinematic variability even with the provision of visual feedback; and iv) kinematic variability at the hip joint in the sagittal plane was either the same or greater than in the non-feedback conditions with the provision of trunk flexion and medio-lateral sway feedback ( $TTE_{Tflex}$  and  $TTE_{Tsway}$  tests). In addition, with the provision of trunk flexion feedback ( $TTE_{Tflex}$  test) an increase in the variation of trunk medio-lateral sway was observed compared to when no feedback was provided. Such changes may represent compensatory adjustments at the hip joint or trunk when movement of the trunk is limited in either the sagittal or coronal plane. These results indicate that regardless of whether or not well-trained cyclists are able to control the level of kinematic variability when fatigued, acute exposure to real-time, kinematic feedback to limit movement of the trunk/hip joints during high-intensity cycling may influence cycling technique and, in some cases (e.g. trunk medio-lateral sway), may reduce performance.

This is the first study to determine whether the use of real-time, kinematic feedback during high-intensity, constant-load cycling affects the time taken to reach task failure. Given that the neuromuscular system was not able to ‘freely’ employ joint kinematic compensatory strategies that have typically been observed with fatiguing exercise (see Chapter 4, (37, 38, 87, 192, 266)), it was hypothesised that the use of kinematic feedback would result in either no change or a reduction in the time taken to reach task failure; this is at odds with the strategy to ‘maintain technique’ in the face of fatigue accumulation. The results confirm this hypothesis because there was little influence on the times taken to reach task failure when the participants were provided with trunk flexion or hip abduction/adduction kinematic feedback and there was a decrease in performance ( $TTE_{Tsway}$  test) when trunk medio-lateral sway was minimised (Table 5.1). When a retrospective analysis was done to exclude those subjects who did not meet the movement pattern feedback criteria perfectly, the reduction in performance in the  $TTE_{Tsway}$  condition was no longer statistically significant, however the effect sizes of the differences in performance ( $TTE_{Tsway}$  vs. no feedback) were very similar (0.41 vs. 0.44), indicating that the lack of statistical significance might be explained by the

reduced sample size, and thus statistical power. Further research with a greater sample size would confirm this hypothesis. These findings reveal that purposefully restricting kinematic variation through instruction and feedback provision can be detrimental to performance in cyclists, and that some (well-trained) cyclists might be unable to control their kinematic patterns within a set target zone in the face of significant fatigue. Thus, it could be hypothesised that kinematic variation occurs despite the conscious attempt to limit such variation during fatigue. While sports coaches and health and safety officers may encourage the maintenance of a ‘good technique’ in order to preserve movement efficiency and avoid injury, the present results indicate that the provision of acute kinematic feedback may be problematic, or at least not beneficial for performance. Whilst an important focus of the present research was to determine the effect of acute feedback, it would be interesting to determine whether the use of prolonged periods (e.g. several weeks) of feedback training would lead to different outcomes.

The results of the present study indicate that limiting the degree of trunk flexion, trunk medio-lateral sway, or hip abduction/adduction has little influence on the exercise capacity during high-intensity cycling. Indeed, the times taken to reach task failure were not significantly different between the feedback and non-feedback conditions ( $p > 0.05$ ), except for when all participants were considered in the trunk medio-lateral sway condition ( $TTE_{Tsway}$  test). As such, restrictions to the degree of trunk flexion, trunk medio-lateral sway, or hip abduction/adduction do not appear to impose any problems for the neuromuscular system to either: (i) continue to employ compensatory strategies similar to that during a TTE test performed with no feedback, or (ii) make compensatory kinematic adjustments to continue exercise performance. By way of example, compensatory adjustments were observed at the hip joint in the sagittal plane with the provision of trunk flexion and medio-lateral sway feedback ( $TTE_{Tflex}$  and  $TTE_{Tsway}$  tests), where kinematic variability was either the same or greater than in the non-feedback conditions. Similarly, increases in the variation of trunk medio-lateral sway were observed when movement of the trunk in the sagittal plane was limited ( $TTE_{Tflex}$  test), compared to when no feedback was provided.

Despite the provision of kinematic feedback, significant changes in mean joint angles and/or ROMs were predominately observed at the trunk and knee joints in the sagittal plane and at the hip and knee joints in the coronal plane during all constant-load cycling tests. These results largely confirm the findings of Study 2 (Chapter 4), and previously published data of constant-load cycling at 100% MAP (38, 87). While the kinematic changes that occurred during tests where feedback was provided occurred at

multiple joints, and not only the joint for which feedback was given, changes were similar across all conditions. However, with the provision of trunk flexion ( $TTE_{Tflex}$  test) and hip abduction/adduction ( $TTE_{Habd/add}$  test) feedback no changes were observed in the degree of knee flexion. The only additional kinematic changes noted with both trunk flexion and medio-lateral sway feedback ( $TTE_{Tflex}$  and  $TTE_{Tsway}$  tests) were increases in left and right hip flexion ROM, respectively. These kinematic changes at the hip may represent compensatory adjustments that occur when trunk flexion is limited. Likewise, an increase in trunk medio-lateral sway ROM observed in the trunk flexion feedback ( $TTE_{Tflex}$ ) test could be an attempt to accommodate for a limited sagittal plane trunk movement.

In the current investigation at least two participants in each feedback condition were unable to correctly use the kinematic feedback provided in order to maintain the desired technique (i.e., minimise movement of the trunk or thigh segment(s) to within the target zone). As such, it appears that some well-trained cyclists who exhibited consistent alterations in movement patterns during a fatiguing exercise task found it difficult to adopt a new kinematic strategy that restricted the degree of trunk flexion, trunk medio-lateral sway or hip abduction/adduction. However, it should be noted that the degree of change in mean trunk flexion angle was significantly less than that which occurred in the non-feedback test ( $p = 0.019$ ), as was the degree of trunk medio-lateral sway and hip abduction/adduction ROM, although this was not statically significant ( $p > 0.05$ ). Thus, although participants were satisfied with their opportunity to cycle with the feedback prior to testing, it appears that such a small exposure period was not substantial enough. In future studies, it would be interesting to determine whether the use of a smaller target zone might force the cyclists to maintain the required technique. In the present study, post-hoc analyses were used to eliminate participants who failed to fulfil the imposed movement pattern inclusion criteria. Although this procedure reduced the sample size it provided a clearer indication as to the true effects of restricting joint movements during the cycling task. It is worth noting that subjective interpretations/feedback from participants indicated that hip abduction/adduction in the  $TTE_{Habd/add}$  test was more difficult to control than the kinematic feedback provided for the trunk. Indeed, increases in left hip abduction/adduction and knee valgus/varus ROM, and left and right knee mean valgus/varus angles during the  $TTE_{Habd/add}$  test, remained significant even after employing the rigorous inclusion criteria. It is possible that this greater difficulty was the result of the increased mental load associated with simultaneously controlling the movement of two separate limbs, a greater number of

degrees of freedom involved in hip abduction/adduction compared with trunk flexion, and/or a reduced ability of participants to use muscle groups not directly involved in the task (i.e. upper body) in order to resist against the handle bars and hold the trunk in the desired position during the fatiguing task. Interestingly, although the participants perceived that this feedback condition was harder to perform, RPE was similar at task failure at the end of all tests ( $p > 0.05$ ). Further research is warranted in order to better understand what factors influence the difficulty of performing exercise tasks with kinematic feedback.

It was not possible to blind participants to the feedback condition within the present study and as such the participant's expectations of the kinematic alterations are likely to have affected their performance. In the present study, four participants stated that maintaining an 'efficient' cycling technique throughout an exercise bout would, in most situations, improve performance, but the majority of these participants also believed that in certain situations (i.e. hill climbing and/or time-trials) varying technique may also be beneficial. The remainder of the participants believed that altering cycling technique may assist in maintaining exercise capacity and they therefore could be expected to perform worse in the trials with kinematic feedback, compared to the non-feedback condition. Despite this, significant kinematic changes were observed for the majority of participants across all trials. Furthermore, the time to task failure and end-exercise perceived exertion was not different between trials for participants who met the movement pattern inclusion criteria ( $p > 0.05$ ). Interestingly, two of the six participants who were excluded from the analyses believed that holding a specific technique or movement strategy would be beneficial for performance. These results indicate that such fatigue-induced compensatory adjustments may be regulated sub-consciously and are therefore very difficult to control. Clearly further research examining the influence of prior expectations and perceived fatigue on performance and the ability to attenuate or restrict kinematic variability during fatiguing exercise is warranted.

In conclusion, when all participants were examined, the acute exposure to kinematic feedback for trunk flexion and hip abduction/adduction had no effect on the times taken to reach task failure in the high-intensity, constant-load cycling task and there was a significant decrease in performance when trunk medio-lateral sway feedback was provided. Thus, kinematic variation occurred in these well-trained cyclists, who otherwise had very consistent and well-learned kinematic patterns, despite visual feedback provision and the conscious attempt to limit variation during fatigue. However, some well-trained cyclists were unable to correctly use the kinematic



feedback provided perfectly, and their removal from analysis resulted in no changes in the times taken to reach task failure between the feedback ( $TTE_{Tflex}$ ,  $TTE_{Tsway}$  and  $TTE_{Habd/add}$ ) and non-feedback test conditions. Nonetheless, the lack of significance in the  $TTE_{Tsway}$  was probably related to the reduced sample size, and thus statistical power. In addition, regardless of the joint movement restrictions imposed by the feedback, the majority of kinematic changes observed throughout the exercise bout were similar between the feedback and non-feedback trials. Increases in kinematic variability in hip flexion and trunk medio-lateral sway was observed during the feedback conditions ( $TTE_{Tflex}$  and  $TTE_{Tsway}$  tests), compared to when no feedback was provided. Such changes may represent compensatory adjustments at the hip joint or trunk when movement of the trunk is limited in either the sagittal or coronal plane. These results indicate that, regardless of whether or not well-trained cyclists are able to control the level of kinematic variability when fatigued, acute exposure to real-time, kinematic feedback to limit trunk flexion, trunk medio-lateral sway, and hip abduction/adduction during high-intensity cycling may influence cycling technique and, in some cases (e.g. trunk medio-lateral sway), may reduce performance.

## CHAPTER SIX

### Interdependence of physiological and biomechanical factors in determining high-intensity cycling performance in well-trained cyclists

#### 6.1 Abstract

**Purpose:** The relationship between physiological attributes previously shown to be indicative of successful cycling performance and lower and upper limb kinematics measured in non-fatigued and fatigued high-intensity, constant-load cycling were examined. In addition, the relationship between joint kinematics and the ability to continue exercise (i.e. the time taken to reach task failure) was determined. **Methods:** 18 well-trained male cyclists performed an incremental cycling test to exhaustion from which numerous physiological attributes were determined, including cycling economy, power output and heart rate at the first ( $VT_1$ ) and second ( $VT_2$ ) ventilatory thresholds, peak power output ( $W_{peak}$ ), maximal heart rate ( $HR_{max}$ ) and maximal oxygen consumption ( $VO_{2max}$ ). In the subsequent session, participants performed a time to exhaustion (TTE) test at 90%  $P_{max}$  (power output at  $VO_{2max}$ ) at a constant cadence. Lower (i.e. hip, knee and ankle joint) and upper (i.e. trunk) limb kinematics were recorded in a non-fatigued state at the start of the trial and a fatigued state at end of the test. **Results:** Hip abduction/adduction, and knee and ankle flexion kinematics measured during a non-fatigued state were correlated with submaximal physiological attributes; left hip abduction/adduction ROM ( $r = -0.529$ ) and right hip abduction/adduction mean angle ( $r = -0.556$ ) were correlated with power output at  $VT_1$ ; left ( $r = 0.473$ ) and right ( $r = 0.655$ ) mean knee flexion angles were correlated with heart rate at  $VT_1$ ; and cycling economy at 100 W was correlated with left ankle ROM ( $r = 0.609$ ) ( $p < 0.05$ ). However, trunk and ankle flexion kinematics measured at fatigue were correlated with both physiological attributes associated with maximal exercise capacity and cycling economy; mean trunk flexion angle was correlated with heart rate at  $VT_2$  ( $r = 0.470$ ) and  $HR_{max}$  ( $r = 0.651$ ); mean ankle flexion angles were correlated with heart rate at  $VT_2$  (left:  $r = -0.552$ ; right:  $r = -0.476$ ),  $HR_{max}$  (left:  $r = -0.548$ ; right:  $r = -0.531$ ), and  $W_{peak}$  ( $W \cdot kg^{-1}$ ) (left:  $r = -0.486$ ); and ankle ROM was correlated with  $W_{peak}$  ( $W \cdot kg^{-1}$ ) (left:  $r = 0.593$ ; right:  $r = 0.549$ ),  $VO_{2max}$  ( $ml \cdot kg^{-1} \cdot min^{-1}$ ) (left:  $r = 0.689$ ; right:  $r = 0.59$ ) and cycling economy at 100 W (left:  $r = 0.564$ ; right:  $r = 0.479$ ) ( $p <$

0.05). Trunk flexion ( $r = -0.505$ ) and medio-lateral sway ( $r = -0.509$ ) ROM in a non-fatigued state, and mean trunk flexion angle ( $r = -0.503$ ) in a fatigued state, were associated with the time to task failure. **Conclusions:** These findings reveal an interdependence between cycling kinematics and both the physiological attributes indicative of successful cycling performance and the time taken to reach task failure during high-intensity, constant-load cycling in well-trained male cyclists.

## 6.2 Introduction

Successful cycling performance is dictated by a variety of factors, including race tactics, technique and the physiological attributes of the athlete (21, 103, 104). Indeed, the physiological characteristics of cyclists measured in laboratory-based tests have been used to monitor the success of specific training interventions (324) and predict cycling performance (21, 23, 73, 103, 104, 135, 183). From this research a number of physiological attributes have become synonymous with better cycling performance, including  $W_{\text{peak}}$ ,  $VO_{2\text{max}}$ , metabolic thresholds (i.e. lactate or ventilation thresholds), maximal lactate steady-state (i.e. the highest exercise intensity at which blood lactate concentration is stable) and efficiency/economy (21, 23, 73, 103, 104, 135, 183). Notable examples are the moderate to strong relationships ( $r^2 > 0.80$ ) observed between prolonged cycling time trial performance and  $VO_2$  or power output at either submaximal (i.e. metabolic thresholds) or maximal ( $VO_{2\text{max}}$  or  $W_{\text{peak}}$ ) workloads measured during graded cycling (23, 73, 135).

While this research has provided valuable information as to the physiological attributes that may be important to cycling performance, movement technique (kinematics) and the efficiency of force application at the pedals (kinetics) can also greatly influence physiological responses, and in turn, exercise performance (173). A number of studies have examined the association between physiological and biomechanical variables during various exercise tasks, e.g. alpine skiing (268), running (167, 222, 228, 286, 322) and swimming (24, 68, 239, 328), however the relationship between physiological attributes and cycling kinetics (pedal force application) is limited (73, 173). Perhaps more notably, the relationship between an individual's physiological attributes and cycling kinematics is not known; therefore it is not clear whether there is an interdependence of the physiological attributes of a cyclist and cycling technique.

This is somewhat surprising since it is generally believed that effective pedal force application provided by use of an 'optimum cycling technique' is associated with improved cycling efficiency and reduced fatigue, and thus enhanced performance (173). Nonetheless, a relationship between the physiological attributes of a cyclist and cycling biomechanics is not always seen. Indeed, Coyle et al. (73) did not observe a relationship between the superior cycling performance ability of 'elite cyclists' (i.e., those with greater power production, anaerobic threshold and  $VO_{2\text{max}}$  within a cycling group) and pedalling effectiveness (i.e., the proportion of force applied to the pedal that results in propulsive torque) during a 1 h cycling trial at lactate threshold, although peak torque

production during the downstroke remained higher in ‘good cyclists’. The authors suggested that other aspects of the cycling movement, such as joint kinematics and muscular coordination, should also be considered as important factors related to pedal force application. More recently, Leirdal et al. (173) found no influence of technique (body orientation/seat position) on the efficiency or effectiveness of pedal force application during repeated-bout cycling at 80% MAP. These studies suggest that cyclists who typically have better physiological profiles do not necessarily utilise a different technique, and that body/seat position and/or pedal force application patterns may not be important factors related to performance.

However, these data were obtained on cyclists performing non-fatiguing exercise. Competitive cycling is a demanding sport often performed at high-intensities (181), resulting in significant fatigue-induced changes in movement kinematics (Chapters 4 and 5, (37, 38, 87, 192, 266)). The ability to maintain a desirable technique as fatigue accumulates may have a greater influence on overall cycling performance. It is therefore important to examine the relationship between physiological attributes related to cycling performance and joint kinematics in both fatigued and non-fatigued cycling. A detailed understanding of this relationship, measured in these states, would allow for a better understanding of the interdependence of the physiological response of a cyclist and cycling technique. It is possible that specific physiological characteristics may be associated with an athlete’s ability to resist fatigue and thus to maintain cycling technique towards the end of a high-intensity exercise bout. As such, the aims of the present study were to: i) determine whether there are any relationships between physiological attributes that are commonly considered indicative of successful cycling performance, and lower (i.e. hip, knee and ankle joint) and upper (i.e. trunk) limb kinematics measured during high-intensity cycling in both a non-fatigued (i.e. start of exercise) and fatigued (i.e. task failure) state, and ii) determine whether there are relationships between joint kinematics and cycling performance (i.e. the time taken to reach task failure in the constant-load cycling test). It should be noted that the present analyses were done on pooled data obtained during Studies 2 and 3 (Chapters 4 and 5).

## 6.3 Methods

### 6.3.1 Participants

18 well-trained male cyclists (mean  $\pm$  SD; age  $36 \pm 8$  y, height  $179 \pm 7$  cm, body mass  $78.4 \pm 12.1$  kg,  $\text{VO}_{2\text{max}}$   $55.4 \pm 5.5$  ml $\cdot$ kg $^{-1}\cdot$ min $^{-1}$ ) with  $6 \pm 4$  y of cycling experience and currently cycling  $361 \pm 109$  km $\cdot$ wk $^{-1}$  volunteered to participate in the study. Experienced cyclists were chosen because they were more likely to have efficient and reliable motor patterns reflected in consistent kinematic adjustments (56). The participants were asked to report their training history (i.e. distance cycled per week and years of cycling experience) and to record and follow a similar diet in the 24 h period prior to all test sessions (see Appendix K). The participants were also asked to avoid the consumption of alcohol, caffeine and other stimulants for 12 h and strenuous exercise for 48 h prior to all test sessions. The participants were informed of the study requirements and written consent was gained prior to participation (see Appendices E-H). All procedures were approved by the institution's human research ethics committee and conformed to the Declaration of Helsinki (see Appendix D).

### 6.3.2 Experimental protocol

The participants performed two test sessions which were separated by at least 48 h. An incremental cycling test to exhaustion was performed in the first session, and a high-intensity constant-load cycling TTE test was completed in the second session. Cycling exercise was performed on an electromagnetically-braked cycle ergometer (Velotron, RacerMate, USA), which was configured to resemble the participant's own bike set-up; this was kept constant for both sessions. The participants used their own pedals and clip-in cycling shoes.

#### 6.3.2.1 Incremental cycling test

The participants performed the same incremental cycling test as described in Study 1 (Chapter 3). Several physiological attributes that have been previously reported to correlate with submaximal and high-intensity or maximal cycling performance (21, 23, 73, 103, 104, 135, 183) were calculated from the incremental exercise test.  $\text{VO}_{2\text{max}}$  was calculated as the highest  $\text{VO}_2$  reading averaged across two consecutive readings (169).  $\text{VT}_1$  or aerobic threshold was taken as the point at which a non-linear increase in the ventilatory equivalent of  $\text{VO}_2$  was first observed ( $V_E/\text{VO}_2$ ), and  $\text{VT}_2$  or anaerobic

threshold was taken as the point at which there was a concomitant increase in the ventilatory equivalent for  $\text{VO}_2$  and carbon dioxide ( $\text{VCO}_2$ ) production ( $\text{V}_E/\text{VCO}_2$ ) (182). Ventilatory thresholds were determined by two independent researchers and the opinion of a third researcher was sought where discrepancies were observed. Power output and heart rate at the first and second ventilatory thresholds and the end of the test (i.e.  $\text{W}_{\text{peak}}$  and  $\text{HR}_{\text{max}}$ ) were determined. Cycling economy was calculated based on the average  $\text{VO}_2$  over the final 30 s of the 100 W stage using the formula below and ensuring that respiratory exchange ratio values were below 1.0 (216):

$$\text{Cycling economy (W} \cdot \text{L} \cdot \text{min}^{-1}) = \text{power (W)} / \text{average } \text{VO}_2 (\text{L} \cdot \text{min}^{-1})$$

### **6.3.2.2 Constant-load cycling test**

As described in Study 1 (Chapter 3).

### **6.3.2.3 Kinematics during cycling**

Kinematic data were recorded, processed and analysed as described in Study 2 (Chapter 4). However, in order to reduce the need to collect marker coordinate data during the full duration of the test, data were recorded in the first 20 s of the trial (i.e. at  $T_0$ ) and again when the RPE increased above 16 and/or the cadence slowed to 5% below the target cadence in the final period of the test ( $T_{100}$ ). Joint mean angles and ROM were calculated as per Study 2 for each time point ( $T_0$  and  $T_{100}$ ).

### **6.3.3 Statistical analyses**

Data are expressed as mean values  $\pm$  SD. Pearson's product moment correlation's were computed to characterise the relationship between physiological attributes recorded during the incremental cycling test and kinematic variables recorded during the constant-load cycling test in both non-fatigued (i.e. at  $T_0$ ) and fatigued (i.e. at  $T_{100}$ ) states. The relationships between kinematic variables at  $T_0$  and  $T_{100}$  and the time taken to reach task failure in the constant-load cycling test, as well as training history (i.e. the distance cycled per week and years of cycling experience), were also examined using Pearson's product moment correlations. The strengths of correlations were ranked according to the guidelines of Dancey and Reidy (74): perfect correlation,  $r = 1.0$ ; strong correlation,  $r = 0.7 - 0.9$ ; moderate correlation,  $r = 0.4 - 0.6$ ; weak correlation,  $r = 0.1 - 0.3$ ; no correlation,  $r = 0.0$ . Joint mean angles and ROM between  $T_0$  and  $T_{100}$  were analysed using paired-samples t-tests. All statistical analyses were performed using

standard statistical software program (SPSS v 17 for Windows, SPSS, Inc., Chicago, IL, USA). Significance was set at  $p < 0.05$ .

## 6.4 Results

### 6.4.1 Incremental cycling test

Physiological variables measured during the incremental cycling test are presented in Table 6.1.

**Table 5.1** Physiological attributes of 18 well-trained cyclists measured during the incremental cycling test to exhaustion.

Physiological attributes	Mean $\pm$ SD
VO <sub>2max</sub> (L·min <sup>-1</sup> )	4.3 $\pm$ 0.5
VO <sub>2max</sub> (ml·kg <sup>-1</sup> ·min <sup>-1</sup> )	55.4 $\pm$ 5.5
W <sub>peak</sub> (W)	408 $\pm$ 33
W <sub>peak</sub> (W·kg <sup>-1</sup> )	5.3 $\pm$ 0.5
PO at VT <sub>1</sub> (W)	229 $\pm$ 36
PO at VT <sub>2</sub> (W)	335 $\pm$ 32
HR <sub>max</sub> (beats·min <sup>-1</sup> )	183 $\pm$ 9
Heart rate at VT <sub>1</sub> (beats·min <sup>-1</sup> )	144 $\pm$ 12
Heart rate at VT <sub>2</sub> (beats·min <sup>-1</sup> )	168 $\pm$ 9
Cycling economy (W·L·min <sup>-1</sup> )	55.8 $\pm$ 7.6

VO<sub>2max</sub>, maximal oxygen consumption; W<sub>peak</sub>, peak power output; PO, power output; VT<sub>1</sub>, first ventilatory threshold; VT<sub>2</sub>, second ventilatory threshold; HR<sub>max</sub>, maximal heart rate; HR, heart rate.

### 6.4.2 Constant-load cycling test

The participants cycled at an average power output of 337  $\pm$  35 W (90% P<sub>max</sub>) and at a cadence of 89  $\pm$  8 rpm. Time to task failure (T<sub>100</sub>) was 396  $\pm$  113 s. Trunk, hip, knee and ankle joint kinematics are presented in Table 6.2.



**Table 6.2** Trunk, hip, knee and ankle joint kinematics for ten pedal cycles during non-fatigued ( $T_0$ ) and fatigued ( $T_{100}$ ) high-intensity, constant-load cycling.

	Non-fatigued	Fatigued	p-value
Mean joint angle (°)			
Left hip flexion	70.4 ± 6.5	69.8 ± 7.3	0.596
Right hip flexion	68.0 ± 6.1	69.3 ± 6.5	0.250
Left hip abduction/adduction	- 4.3 ± 3.5	- 7.1 ± 7.1	0.112
Right hip abduction/adduction	- 2.0 ± 3.7	- 5.1 ± 7.5	0.096
Left knee flexion	76.3 ± 6.6	74.2 ± 6.3	< 0.001*
Right knee flexion	72.8 ± 6.6	72.1 ± 6.7	0.187
Left knee valgus/varus	16.6 ± 8.5	17.9 ± 9.0	0.266
Right knee valgus/varus	13.4 ± 15.8	16.1 ± 15.3	0.001*
Left ankle flexion	2.9 ± 7.8	4.5 ± 6.6	0.199
Right ankle flexion	0.9 ± 5.7	2.8 ± 6.5	0.091
Trunk flexion	37.0 ± 5.9	44.0 ± 7.8	< 0.001*
Trunk medio-lateral sway	0.4 ± 3.2	- 0.1 ± 6.6	0.278
Joint range of motion (°)			
Left hip flexion	46.9 ± 3.9	48.4 ± 4.3	0.066
Right hip flexion	46.5 ± 3.6	48.1 ± 4.6	0.062
Left hip abduction/adduction	5.9 ± 2.0	8.7 ± 2.6	< 0.001*
Right hip abduction/adduction	5.6 ± 1.7	8.1 ± 2.3	< 0.001*
Left knee flexion	81.2 ± 5.4	84.0 ± 6.4	0.001*
Right knee flexion	81.8 ± 5.9	84.1 ± 7.1	0.008*
Left knee valgus/varus	19.4 ± 5.4	23.2 ± 7.1	0.005*
Right knee valgus/varus	21.2 ± 7.7	25.4 ± 8.5	0.001*
Left ankle flexion	23.2 ± 7.3	25.7 ± 8.7	0.097
Right ankle flexion	22.8 ± 5.5	23.4 ± 7.5	0.597
Trunk flexion	2.3 ± 0.9	36. ± 1.2	< 0.001*
Trunk medio-lateral sway	5.7 ± 3.5	9.8 ± 6.1	< 0.001*

Data are mean ± SD.

\* Significantly different at  $T_{100}$  from  $T_0$  ( $p < 0.05$ ).

### 6.4.3 Relationships between physiological attributes and kinematic variables

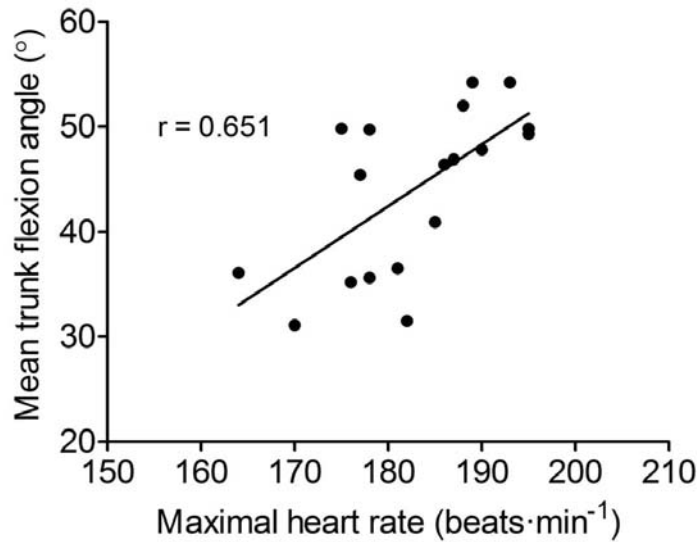
#### 6.4.3.1 Submaximal physiological attributes

Significant correlations were observed between physiological attributes associated with submaximal cycling performance and joint kinematics at the hip, knee and ankle joints measured in the non-fatigued state (i.e.  $T_0$ ). Moderate correlations were observed between power output at  $VT_1$  and both left hip abduction/adduction ROM ( $r = -0.529$ ,  $p = 0.024$ ) and right hip abduction/adduction mean angle ( $r = -0.556$ ,  $p = 0.017$ ). A trend toward a significant correlation was also observed for power output at  $VT_1$  and right hip abduction/adduction ROM ( $r = -0.450$ ,  $p = 0.061$ ). In addition, heart rate at  $VT_1$  was correlated with left ( $r = 0.473$ ,  $p = 0.048$ ) and right ( $r = 0.655$ ,  $p =$

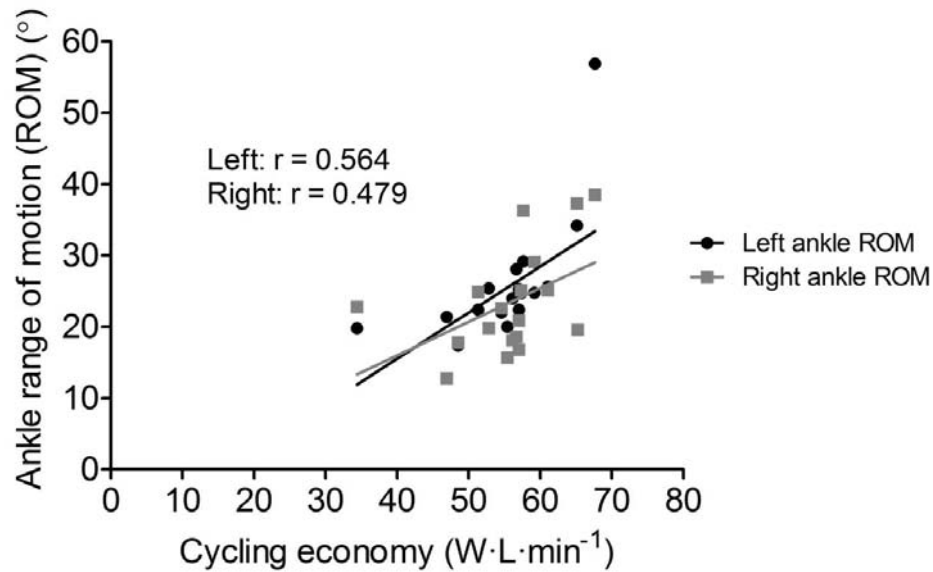
0.003) mean knee flexion angle. Cycling economy was moderately correlated with left ankle ROM ( $r = 0.609$ ,  $p = 0.007$ ), but not the right ankle ROM ( $p > 0.05$ ).

#### 6.4.3.2 Maximal exercise capacity physiological attributes

Moderate to strong correlations were observed between mean trunk flexion angle observed at  $T_{100}$  and heart rate at  $VT_2$  ( $r = 0.470$ ,  $p = 0.049$ ) and  $HR_{max}$  ( $r = 0.651$ ,  $p = 0.003$ ; Figure 6.1). Heart rate at  $VT_2$  (left:  $r = -0.552$ ,  $p = 0.018$ ; right:  $r = -0.476$ ,  $p = 0.046$ ) and  $HR_{max}$  (left:  $r = -0.548$ ,  $p = 0.018$ ; right:  $r = -0.531$ ,  $p = 0.023$ ) were also correlated with left and right mean ankle flexion angles at  $T_{100}$ .  $W_{peak}$  ( $W \cdot kg^{-1}$ ) was moderately correlated with both left ( $r = 0.593$ ,  $p = 0.009$ ) and right ( $r = 0.549$ ,  $p = 0.018$ ) ankle flexion ROM, and left mean ankle flexion angle ( $r = -0.486$ ,  $p = 0.041$ ). Likewise, moderate to strong relationships were observed between  $VO_{2max}$  ( $ml \cdot kg^{-1} \cdot min^{-1}$ ) and left ( $r = 0.689$ ,  $p = 0.002$ ) and right ( $r = 0.593$ ,  $p = 0.010$ ) ankle flexion ROM. Cycling economy was moderately correlated with both left ( $r = 0.564$ ,  $p = 0.015$ ) and right ( $r = 0.479$ ,  $p = 0.044$ ) ankle flexion ROM (Figure 6.2).



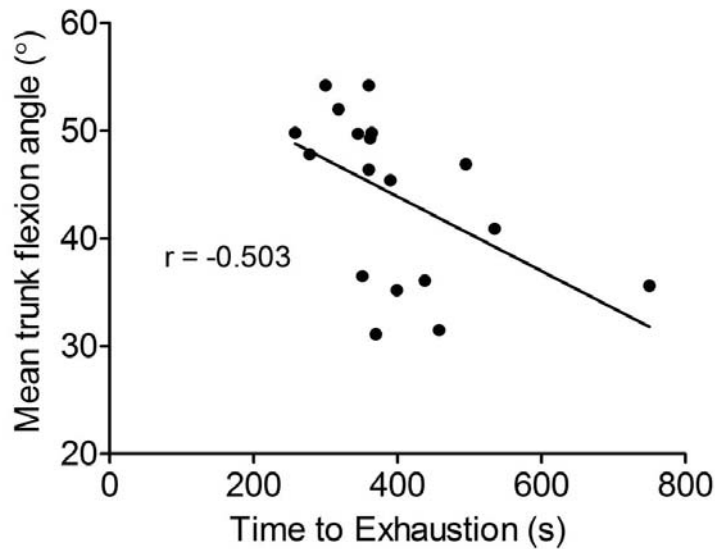
**Figure 6.1** The relationship between mean trunk flexion angle at  $T_{100}$  (i.e., task failure) and maximal heart rate during high-intensity cycling.



**Figure 6.2** The relationships between left and right ankle joints range of motion at  $T_{100}$  (i.e., at task failure) and cycling economy measured at 100 W during high-intensity cycling.

#### 6.4.4 Relationships between kinematic variables and performance

Moderate correlations were observed between trunk flexion ( $r = -0.505$ ,  $p = 0.032$ ) and medio-lateral sway ( $r = -0.509$ ,  $p = 0.031$ ) ROM at  $T_0$  and the time to task failure in the constant-load cycling test. Mean trunk flexion angle was the only kinematic variable measured at  $T_{100}$  that was correlated with the time to task failure ( $r = -0.503$ ,  $p = 0.033$ ; Figure 6.3). It should be noted that these correlations may have been influenced by a single cyclist who had the longest time to exhaustion; however the correlations showed a trend toward significance at the beginning of exercise ( $T_0$ ) (trunk flexion ROM,  $r = -0.444$ ,  $p = 0.074$ ; trunk medio-lateral sway ROM,  $r = -0.453$ ,  $p = 0.068$ ), and remained significant at fatigue ( $T_{100}$ ) (mean trunk flexion angle,  $r = -0.485$ ,  $p = 0.048$ ) even after removal of this subject.



**Figure 6.3** The relationship between mean trunk flexion angle at  $T_{100}$  (i.e., at task failure) and the time taken to reach task failure during high-intensity cycling.

#### 6.4.4 Relationships between kinematic variables and cycling training history

Moderate correlations were observed between trunk flexion ( $r = -0.508$ ,  $p = 0.031$ ) and left knee valgus/varus mean angles ( $r = -0.476$ ,  $p = 0.046$ ) observed at  $T_0$ , and the distance (km) cycled by participants per week. Left hip abduction/adduction ( $r = 0.588$ ,  $p = 0.010$ ) and right ankle flexion ( $r = 0.469$ ,  $p = 0.049$ ) ROM at  $T_0$  were greater in cyclists with a longer cycling training history (years of training). No correlations were found between cycling training history and any kinematic variable measured at  $T_{100}$ .

### 6.5 Discussion

The aims of the present study were to: i) examine the relationships between physiological attributes measured during an incremental cycling test previously shown to be indicative of successful cycling performance and both lower (i.e. hip, knee and ankle joint) and upper (i.e. trunk) limb kinematics measured during high-intensity cycling in non-fatigued and fatigued states, and ii) describe the relationship between joint kinematics and the cyclists' abilities to continue exercise (i.e. the time taken to reach task failure). An important finding of this investigation was that hip abduction/adduction ROM, knee mean flexion angles and ankle flexion ROM measured during non-fatigued cycling were correlated with physiological attributes measured at

submaximal cycling intensities (i.e., power output and heart rate at  $VT_1$ , and cycling economy at 100 W). These results indicate that cyclists with (i) higher heart rate responses at aerobic threshold cycled with an increased degree of knee flexion, (ii) higher aerobic threshold (power output at  $VT_1$ ) displayed less lateral movement at the hip joint and (iii) relatively lower  $VO_2$  (increased economy) exhibited an increase in ankle joint ROM. A second important finding was that mean trunk flexion angles and ankle mean flexion angles and ROM measured at fatigue (immediately prior to volitional exercise termination) were correlated with both physiological attributes associated with maximal exercise capacity (i.e.,  $W_{peak}$ ,  $VO_{2max}$ ,  $HR_{max}$ , heart rate at  $VT_2$ ) and cycling economy at 100 W. Such findings suggest that cyclists with (i) higher work outputs, aerobic capacity and relatively lower  $VO_2$  displayed a greater degree of ankle flexion and ROM and (ii) higher heart rate responses at anaerobic threshold cycled with a greater degree of trunk and ankle flexion. Furthermore, trunk kinematics measured in both non-fatigued (trunk flexion and medio-lateral sway ROM) and fatigued (mean trunk flexion angle) states were associated with the time taken to reach task failure. These findings reveal (i) an interdependence between the physiological attributes indicative of successful cycling performance and cycling kinematics, indicating that either better cyclists' adopt different movement patterns than lesser cyclists or that cycling kinematics influences physiological measurements, and (ii) exercise capacity during high-intensity cycling is related to greater trunk flexion angles and medio-lateral sway ROM in these well-trained male cyclists.

In the present study, a number of significant relationships were observed between lower limb kinematics measured in the non-fatigued state (i.e., at the beginning of exercise) and physiological attributes typically associated with submaximal cycling performance including power output and heart rate at  $VT_1$ , and cycling economy. For instance, participants with a higher aerobic threshold (power output at  $VT_1$ ) displayed less lateral movement at the hip joint. A reduction in lateral hip movement may allow for a greater component of the total force to be directed perpendicular to the crank arm, improving effective pedal force application (173). It is therefore possible that cyclists who perform a significant volume of their training at low exercise intensities, which will improve their aerobic threshold, have also developed a more efficient pedalling technique when cycling in a non-fatigued state. However, it is also possible that an effective delivery of force to the pedal reduced oxygen consumption leading to a better test result. Interestingly, neither the average distance cycled per week nor years of cycling experience were correlated with hip joint kinematics in this study suggesting

that the latter may be a stronger possibility, but this speculation needs to be explicitly studied in future. The measurement of  $\text{VO}_2$  was not done in the present study given the likely influence on movement kinematics from wearing the equipment to record ventilatory responses (mouthpiece, head support, nose clip and respiratory tubing), and the difficulty in obtaining accurate measurements of 3D joint kinematic data and ventilatory responses simultaneously during exercise with the metabolic cart in the capture zone for 3D analysis.

As the exercise bout progressed and significant neuromuscular fatigue developed (see Chapter 3), the relationships between physiological attributes and joint kinematics changed. At the termination of exercise, significant correlations were observed between trunk and ankle flexion kinematics and both physiological attributes typically associated with maximal exercise capacity (i.e.  $W_{\text{peak}}$ ,  $\text{VO}_{2\text{max}}$ ,  $\text{HR}_{\text{max}}$ , heart rate at  $\text{VT}_2$ ) and cycling economy at 100 W. Importantly, previous studies have reported significant changes in both trunk (Chapter 4, (87)) and ankle (37, 38, 192, 266) joint angles and ROM during fatiguing cycling. In the present study, greater ankle flexion ROM was associated with higher peak work output ( $W_{\text{peak}}$ ,  $\text{W}\cdot\text{kg}^{-1}$ , left ankle:  $r = 0.593$ ; right ankle:  $r = 0.549$ ) and aerobic capacity ( $\text{VO}_{2\text{max}}$ ,  $\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ , left ankle:  $r = 0.689$ ; right ankle:  $r = 0.593$ ), and lower oxygen cost in both fatigued ( $\text{W}\cdot\text{L}\cdot\text{min}^{-1}$ ; left ankle:  $r = 0.564$ ; right ankle:  $r = 0.479$ ) and non-fatigued ( $\text{W}\cdot\text{L}\cdot\text{min}^{-1}$ ; left ankle:  $r = 0.609$ ) states. Since the plantar flexors are responsible for transferring the power produced at the hip and knee joints to the pedals (247, 327) it might be that the distribution of joint-specific power production in the lower limb differs depending on the fitness (i.e. physiological characteristics) of cyclists. Furthermore, given the relationship between a greater ROM at the ankle joint and superior physiological characteristics, it is possible these cyclists may have adopted a different pedalling technique. Indeed, cyclists with more cycling experience (i.e., years of training) ( $r = 0.469$ ) had a greater right ankle flexion ROM, which provides further support for this hypothesis.

Another finding of the present study was that participants who cycled with a greater degree of trunk flexion at the end of exercise ( $T_{100}$ ) had higher heart rate responses in the incremental cycling test (i.e.  $\text{HR}_{\text{max}}$ :  $r = 0.651$ ; heart rate at anaerobic threshold ( $\text{VT}_2$ ):  $r = 0.470$ ). One possible hypothesis to explain these results is that increases in trunk flexion increase the requirement for oxygen delivery. Indeed, it was shown in both Chapter 3 and in previous studies (58, 88, 265) that an increase in trunk flexion is associated with an increase in lower limb EMG activity. Given that other researchers have also reported a relationship between the degree of EMG activity and

muscle length in lower limb muscles (22, 184), it can be hypothesised that the increased muscle length associated with a greater degree of trunk flexion may also result in an increase in muscle activity. Alternatively, it has been reported that an excessive level of intermittent hip hyper-flexion occurs in conjunction with a high blood flow demand to the lower limbs during exhaustive cycling in an aerodynamic posture; this can cause extreme loading and blood flow limitations of the iliac blood vessels (29, 179). An increase in trunk flexion and resultant pressure on these vessels may increase venous pooling in the lower limbs during cycling and thus compromise venous return. Thus, it is possible that the cyclists in the present study who exhibited a greater degree of trunk flexion were required to have a higher heart rate to maintain cardiac output and oxygen delivery when cycling at high exercise intensities. Indeed, higher heart rates have been observed when cycling in an aerodynamic posture compared to an upright position in highly trained cyclists (127), but not in non-cyclists (20, 231, 314), and thus differences between participants likely resulted from the variation in training status between them. It should be noted, however, that this hypothesis assumes the cyclists adopted a similar degree of trunk flexion during the incremental and TTE tests, and require further investigation.

An additional consequence of cycling in positions of significant trunk flexion may have been the reduced ability to maintain the required power output of the test (299). Interestingly, the cyclists in the present study who performed best during the TTE test (i.e. longest time to task failure) also had the lowest degree of trunk flexion at both the beginning (joint ROM at  $T_0$ ,  $r = -0.505$ ) and end (mean angle at  $T_{100}$ ,  $r = -0.503$ ) of exercise. A greater degree of trunk flexion, and the possibility of compromised lower limb blood flow, may have made it more difficult for participants to maintain the required power output. Interestingly, given that an increase in trunk medio-lateral sway ROM measured during cycling in a non-fatigued state was also associated with the time taken to reach task failure ( $T_0$ ,  $r = -0.509$ ), it could be proposed that the degree of trunk flexion and medio-lateral sway at the start of exercise is an indicator of (poorer) performance ability.

In conclusion, physiological attributes associated with submaximal exercise performance, including power output and heart rate at  $VT_1$  and cycling economy, were negatively correlated with hip abduction/adduction ROM and positively correlated with knee mean flexion angles and ankle flexion ROM measured in a non-fatigued state. However, trunk mean flexion angles and ankle mean flexion angles and ROM in the fatigued state were correlated with both physiological attributes indicative of maximal

exercise capacity (i.e.,  $W_{\text{peak}}$ ,  $VO_{2\text{max}}$ ,  $HR_{\text{max}}$ , heart rate at  $VT_2$ ) and cycling economy. Additionally, the degree of trunk flexion and medio-lateral sway was related to, and possibly affects, cycling performance during high-intensity, constant-load cycling. These findings indicate a clear interdependence between the physiological attributes of cyclists and the kinematics of the cycling movement. While the directionality of these associations cannot be determined from the present study, these data indicate that joint kinematic strategies may significantly influence measurements of an athlete's physiological attributes and/or the physiological profile of a cyclist determined during incremental cycling tests may influence the joint kinematics adopted during exercise. Further research is required in order to better understand the directionality of these associations and thus improve our understanding of the kinematic and physiological factors that influence cycling performance. The current results provide novel evidence that an interdependence exists between specific lower limb joint kinematics and both the physiological attributes of cyclists and that the time taken to reach task failure during high-intensity cycling in well-trained male cyclists.



## **CHAPTER SEVEN**

### **General Discussion**

During sport-related or manual labour exercise tasks, a certain degree of fatigue occurs within the neuromuscular system. It is important to understand the factors influencing the development of this fatigue and how an individual might delay fatigue progression through motor control and biomechanical (muscle activation and kinematic) strategies. This knowledge would be useful and applicable for a wide range of motor tasks and subject populations. Despite this, research examining neuromuscular fatigue mechanisms, and the muscle activation and kinematic strategies utilised by the CNS during high-intensity, constant-load exercise, is limited. As such, the possible role of enhancing kinematic variability to delay the progression of fatigue and enhance exercise performance has not been addressed in the literature. Indeed, the association between one's ability to resist fatigue and neural (i.e., compensatory) strategies during manual or exercise tasks has not previously been explored. Therefore, the main purpose of the research contained within this thesis was to examine exercise-induced neuromuscular fatigue and to gain a greater understanding of neuromuscular strategies employed during high-intensity, constant-load cycling. The role of such strategies in the possible attenuation of fatigue progression and increase in exercise capacity was examined. Finally, the association between an individual's physiological attributes and cycling kinematics was explored. As outlined in Chapter 1, the first two studies included in this thesis aimed to examine the temporal pattern, and relative contribution, of central and peripheral fatigue mechanisms as well as changes in muscle activation and joint kinematics, and the relationship of these biomechanical changes to neuromuscular fatigue mechanisms (Chapters 3 and 4). The third study of this thesis aimed to determine whether or not these kinematic changes influenced exercise performance (i.e. the time taken to reach task failure) (Chapter 5). Subsequently, the final study assessed the relationships between joint kinematics measured in non-fatigued and fatigued states and both the physiological attributes of cyclists indicative of superior cycling ability and the time taken to reach task failure (Chapter 6).

The major findings of this thesis were that: i) peripheral fatigue was significant from 60% of the time to task failure and a possible up-regulation of central motor drive (increases in VM and GMax muscle activity), rather than central fatigue, occurred

towards the end of a high-intensity, constant-load cycling bout; ii) significant trunk kinematic changes were observed in the sagittal plane from 60% of the time to task failure, and were thus notable at or after the point of significant peripheral fatigue development; iii) significant changes at the trunk, hip and knee joints in the coronal plane were observed from 80% of the time to task failure, which therefore paralleled the facilitation of central motor drive during the cycling task; iv) when considered as a whole group, the provision of trunk and hip joint kinematic feedback either had no discernible ( $TTE_{Tflex}$  and  $TTE_{Habd/add}$  tests), or a negative ( $TTE_{Tsway}$ ), impact on the times taken to reach task failure when compared to a condition where no feedback was provided, indicating that purposefully restricting kinematic variation can be detrimental to performance; v) for participants who correctly used the kinematic feedback (i.e., maintained joint movement within the target zone), the times to task failure were not significantly different in the feedback conditions ( $TTE_{Tflex}$ ,  $TTE_{Tsway}$  and  $TTE_{Habd/add}$  tests), when compared to the non-feedback condition; vi) compensatory increases in kinematic variation at the trunk or hip joint occur when movement of the trunk is limited in either the sagittal or coronal plane ( $TTE_{Tflex}$  and  $TTE_{Tsway}$  tests), indicating that some well-trained cyclists, with very consistent and well-learned kinematic patterns, found it difficult to reduce kinematic variability even with the provision of visual feedback; vii) hip (abduction/adduction angle and ROM), knee (flexion angle) and ankle (flexion ROM) kinematics measured during a non-fatigued state (i.e., at the start of exercise) were correlated with submaximal physiological attributes including power output and heart rate at  $VT_1$ , and cycling economy at 100 W; viii) trunk (flexion angle) and ankle (flexion angle and ROM) kinematics measured at fatigue (i.e., at task failure) were correlated with both physiological attributes associated with maximal exercise capacity ( $W_{peak}$ ,  $VO_{2max}$ ,  $HR_{max}$ , heart rate at  $VT_2$ ) and cycling economy at 100 W; and ix) the degree of trunk flexion and medio-lateral sway may be indicative kinematic variables associated with cycling performance ability. This chapter summarises the answers to the research questions detailed in Chapter 1 and associated purposes described above. In addition, this chapter will aim to highlight the significant findings and limitations of this research, and to propose future research directions.

The results of Study 1 (Chapter 3) were that variables indicative of peripheral fatigue were significant from 60% of the time to task failure and, as the high-intensity, constant-load cycling bout progressed, a possible up-regulation of central motor drive, rather than central fatigue, occurred. This is one of the first study to detail the time course of the development, and relative contributions, of central and peripheral fatigue

mechanisms during high-intensity, constant-load cycling. It is important to examine how the human body adapts to fatigue development from both sporting, exercise and occupational perspectives. Using the novel repeat-test exercise design, a non-linear progression of fatigue development was observed. The majority of twitch contractile properties ( $T_{p,twitch}$ ,  $CT_{twitch}$ ,  $RTD_{twitch}$  and  $RTR_{twitch}$ ) were significantly impaired from 60% of the time to task failure, indicating that peripheral mechanisms at the muscle level and distal to the neuromuscular junction were found to contribute to fatigue. In addition, M-wave propagation and transmission ( $M_{max}$  and  $M_{dur}$ ) were preserved throughout the exercise bout, indicating that fatigue at the sarcolemmal level did not occur during this type of high-intensity exercise. Despite significant peripheral fatigue, voluntary measures of neuromuscular function were maintained ( $T_{p,MVC}$ ,  $RTD_{MVC}$  and %VA); this may relate to the increase or maintenance of central motor drive, although might also indicate that a modicum of recovery occurred in the time period between exercise cessation and the initial neuromuscular assessment. Regardless, these assessments enhance our understanding of the specific processes occurring within the neuromuscular system during fatiguing exercise.

Nonetheless, these neuromuscular assessments were performed prior to and following exercise and thus it was also important to match these with measures of neuromuscular function obtained during the cycling task itself. As such, EMG of lower limb muscles was assessed throughout the exercise bout. This study was one of the first to simultaneously examine neuromuscular function recorded from EMG measurements during the cycling exercise alongside measurements obtained during involuntary (i.e., electrically stimulated) and voluntary isometric contractions of a specific muscle(s) at a single joint during a neuromuscular assessment post-exercise (82). Significant increases in VM and GMax muscle activity were observed during cycling exercise; however the temporal pattern of muscle activation remained stable. These changes may have reflected a central strategy to ensure adequate mechanical efficiency in regards to pedal force application (221, 261), and in turn maintain exercise performance. A significant increase in VL EMG amplitude measured during the isometric knee extension task also occurred across the exercise bout. Collectively, these results highlight that peripheral fatigue may be partly overcome by region-specific muscle activation changes during cycling including the recruitment of additional motor units and/or rotation amongst active motor units (309). This possible up-regulation of central motor drive would allow the desired test power output to be maintained in the event of advancing peripheral fatigue. An important finding of this research is that participants were unable to regulate

exercise intensity and therefore had to increase central motor drive to continue exercise performance (9), despite leading to significant muscular fatigue. Ultimately, this may lead to the conscious decision to terminate the exercise bout.

The time delay between cycling exercise cessation and neuromuscular assessment from the isometric knee extension may have been a limitation in this study design. However, it is important to note that neuromuscular fatigue measured during both the cycling exercise and the isometric knee extension task were similar in progression and thus fatigue measured post-exercise was reflective of that which occurred during the exercise task. Indeed, it is plausible that the degree of change in fatigue variables measured during the neuromuscular assessment post-exercise may have been greater than that observed (112, 243), and it is crucial for researchers in the future to develop techniques whereby neuromuscular function can be measured immediately post-exercise, ideally whilst the participant remains on the cycle ergometer. Regardless, this study has led to important advances in our understanding of neuromuscular fatigue, specifically the contribution, and timing, of central and peripheral fatigue mechanisms during high-intensity, constant-load cycling.

A subsequent aim of this thesis was to examine the temporal relationship between the development of neuromuscular fatigue observed in Study 1 with changes in task kinematics, and therefore to also describe the magnitude of change in kinematics during high-intensity, constant-load cycling. In Study 2 (Chapter 4) a significant increase in trunk flexion occurred from 60% of the time to task failure, whilst changes in trunk medio-lateral sway, hip abduction and knee varus kinematics were notable from 80% of the time to task failure. These observations indicate that peripheral fatigue accumulation may lead to alterations in the sagittal plane, whilst coronal plane adjustments occur once either central facilitation is significant and/or a more substantial level of peripheral fatigue accumulates. Importantly, kinematic changes in the sagittal and coronal plane appear to be temporally dissociated, and may be in part be related to the site of exercise-induced fatigue (i.e. peripheral or central). It can therefore be hypothesised that the joint kinematic changes observed throughout the exercise bout reflect fatigue-induced motor control and biomechanical strategies employed to maintain exercise performance. For instance, the increase in trunk flexion observed with increasing fatigue would alter the force production capacity of the muscles acting at the hip/knee joints (20, 265, 299), and thereby require a concomitant increase in lower limb (VM and GMax) muscle activity to maintain the necessary propulsive force generated during the down-stroke (88). Given that kinematic changes in the sagittal plane occurred

first, such neural strategies are believed to assist in the application of force at the pedal through the activation of previously inactive muscle(s). Alternatively, coronal plane changes were evident later in the exercise bout, and thus such changes could be considered as final strategies employed by the neuromuscular system to continue exercise as long as possible. Conversely, it is also plausible that such coronal plane kinematic changes may have decreased the effectiveness of pedal force application, and consequently increased fatigued development and been responsible for task failure. However, no research to date has investigated the combined effects of fatigue and medio-lateral motion on pedal force application and task performance. Regardless, this study revealed neural strategies that adopted during fatiguing exercise, and is the first to indicate that the differential temporal effects of fatigue accumulation on sagittal and coronal plane joint kinematics.

The results of Studies 1 and 2 highlight that, during high-intensity, constant-load cycling, significant peripheral fatigue and increases in central motor drive occur with subsequent changes in joint kinematics at the trunk, and hip and knee joints. For the first time, these studies provide detailed data suggesting the existence of a relationship between specific neuromuscular fatigue mechanisms and technique (i.e., joint kinematic) changes in cycling. Taken together, these findings extend our understanding of biomechanical and motor control strategies employed by the neuromuscular system under fatiguing conditions. It is suggested that the neuromuscular system may compensate for such fatigue and allow maintenance of the task goal through alterations in muscle activation and joint kinematic strategies. The implications of these findings are that during high-intensity, short-duration exercise performance coaches and/or occupational health practitioners should be aware that the changes in movement technique observed during exercise are most likely compensatory strategies sub-consciously utilised by the neuromuscular system to overcome fatigue development. After identifying the kinematic strategies utilised during this type of exhaustive cycling, it was then important to consider whether allowing these compensatory changes, or holding technique was more advantageous in terms of improving exercise capacity.

Given these findings, Study 3 of this thesis examined the effect of real-time, kinematic feedback for trunk flexion, trunk medio-lateral sway and hip abduction/adduction on the time taken to reach task failure during high-intensity, constant-load cycling. The results of this study revealed that there was little influence on the times taken to reach task failure when the participants were provided with trunk flexion or hip abduction/adduction kinematic feedback, but when provided with trunk

medio-lateral sway feedback ( $TTE_{Tsway}$  test) there was a significant reduction in performance. However, some well-trained cyclists were unable to correctly use the kinematic feedback provided perfectly (i.e., maintain trunk/hip movement within the target zone), particularly for the hip abduction/adduction condition ( $TTE_{Habd/add}$  test). As such, it was suggested that some (well-trained) cyclists who exhibited consistent alterations in joint kinematics during a fatiguing exercise task found it difficult to adopt a new kinematic strategy that restricted the degree of trunk flexion, trunk medio-lateral sway or hip abduction/adduction. Furthermore, in regards to the hip abduction/adduction feedback condition, it was hypothesised that this condition was more difficult given the increased mental load associated with simultaneously controlling the movement of two separate limbs, or that perhaps this is a more difficult kinematic adjustment for the neuromuscular system to control because of the greater number of degrees of freedom involved. Thus, when a retrospective analysis was done to exclude those subjects who did not meet the movement pattern criteria perfectly, the reduction in performance in the  $TTE_{Tsway}$  condition was no longer statistically significant, however this was probably related to the reduced sample size, and thus statistical power. These findings reveal that purposefully restricting kinematic variation through instruction and feedback provision can, in some cases, be detrimental to performance. Thus, it is suggested that kinematic variation occurs despite the conscious attempt to limit such variation during fatigue.

Significant changes in joint kinematics were observed in Study 3 and were largely similar for all feedback conditions. Of interest, during the trunk flexion and medio-lateral sway feedback conditions ( $TTE_{Tflex}$  and  $TTE_{Tsway}$  tests), left and right hip flexion variation increased, respectively, and were hypothesised to be compensatory adjustments at the hip joint when trunk flexion was limited. Furthermore, compensatory increases in the variation of trunk medio-lateral sway were observed when movement of the trunk in the sagittal plane was limited ( $TTE_{Tflex}$  test). Provided that the participants held their own beliefs in regards to how the kinematic feedback would affect their cycling performance it was interesting that, regardless of participant's task expectations, performance was not affected for those cyclists who met the inclusion criteria. Given these findings, it might be suggested that the ability, or lack of, to control movement at specific joint segments may be a subconscious fatigue-induced adjustment. These data therefore support the notion that motor control and biomechanical patterns (i.e., fatigue-related adjustments) are entrenched in this group of well-trained cyclists. Regardless, these results provide novel insights of how kinematic feedback can be used to

manipulate an individual's movement technique during exercise, and that such changes have minimal effects on task performance. One final consideration is that it is also possible that kinematic variability may assist in reducing injury risk (25, 282), and thus might be considered as another potential favourable aspect to motor variability during exhaustive cycling. In combination the knowledge gained in the present research may be of use for coaches and/or occupational health practitioners whereby encouraging the use of a consistent technique may not be of benefit. Nonetheless, it is possible that changing technique/increasing kinematic variability excessively could be undesirable in regards to exercise capacity.

The first three studies of this thesis investigated the neuromuscular adjustments that occur during fatiguing cycling exercise. The development of neuromuscular fatigue and subsequent changes in movement kinematics observed (Studies 1 and 2) are likely to be influenced by an individual's fitness/physiology. Likewise, however, the results of Study 2 also indicated that cycling kinematics may influence muscle function (i.e., changes in force/length characteristics), muscle recruitment (i.e., level of muscle activity) and thus fatigue development. Indeed, according to the biomechanical model of fatigue, fatigue during cycling is governed by the efficiency of motion and consequently cycling technique will influence physiological responses during exercise (1). Therefore, the final study of this thesis (Chapter 6) aimed to further our understanding of the association between the physiological characteristics of cyclists and biomechanical (joint kinematics) aspects of cycling performance. This is the first study to present findings on these relationships, and this was considered as an important next step to understand the possible influence of the identified kinematic changes from Studies 2 and 3 on physiological indices of cycling performance ability. The results of this study revealed that joint kinematics in a non-fatigued state (i.e., recorded at the start of the exercise bout) including hip abduction/adduction and both knee and ankle flexion, were significantly correlated with submaximal physiological attributes (power output and heart rate at  $VT_1$ , and cycling economy at 100 W). Such findings suggest that cyclists with (i) higher heart rate responses at aerobic threshold cycled with an increased degree of knee flexion, (ii) higher aerobic threshold (power output at  $VT_1$ ) displayed less lateral movement at the hip joint and (iii) relatively lower  $VO_2$  (increased economy) exhibited an increase in ankle joint ROM. Conversely, trunk and ankle flexion kinematics measured during fatigue (i.e., at task failure) were correlated with both maximal physiological attributes (i.e.  $W_{peak}$ ,  $VO_{2max}$ ,  $HR_{max}$ , heart rate at  $VT_2$ ) and cycling economy at 100 W. These results indicate that cyclists with (i) higher work



outputs, aerobic capacity and relatively lower  $\text{VO}_2$  displayed a greater degree of ankle flexion and ROM and (ii) higher heart rate responses at anaerobic threshold cycled with a greater degree of trunk and ankle flexion. Another interesting outcome of this study was that trunk kinematics measured in both non-fatigued and fatigued states were associated with the time taken to reach task failure, indicating that cyclists who rode with a greater degree of trunk flexion and medio-lateral sway cycled for the least amount of time. One possibility is that poorer cyclists deliberately utilise a greater degree of trunk flexion in order to prolong work output. However, it is also possible that such kinematic changes exacerbated fatigue and may have been a causative factor in the shorter performance time. This may have been due to, for example, an increase in the requirement for oxygen delivery that may relate to compromised iliac artery and vein blood flow limitations (29, 179) and/or increases in muscle length (22, 184) in positions of increased trunk flexion. Overall, these findings indicate a clear interdependence between the physiological attributes of cyclists and the kinematics of the cycling movement. In addition, these data indicate that joint kinematic strategies may significantly influence measurements of an athlete's physiological attributes, and/or that the physiological profile of a cyclist determined during incremental cycling tests may influence the joint kinematics adopted during exercise; however, the current study does not allow an indication to the directionality of these associations. Despite this, the outcomes of this novel study provide valuable information regarding the relationships, and thus interdependence, of physiological and biomechanical aspects of cycling performance, and provide a platform for future research to extend upon these initial and innovative findings. It is important to acknowledge and further investigate the importance of joint kinematics on physiological responses and performance during cycling.

## **7.1 Directions for future research**

The findings from the present research contribute to the existing literature; however, they also highlight some possible areas of future research. There are several avenues for future projects and these will be discussed below.

The results of Study 1 provided valuable information regarding the time course and relative contribution of central and peripheral fatigue mechanisms. Although this study extended our knowledge of neuromuscular fatigue, the time delay between cycling cessation and the initial neuromuscular assessment may have allowed some



recovery of force production. It is important to note that this methodology is commonly employed in neuromuscular research and studies typically cite a 2-10 min time delay (10, 11, 13-15, 166, 240, 254, 275). Recently, Sidhu et al. (276) examined corticospinal contributions to central motor drive during sustained whole-body exercise whilst participants cycled upright on a modified cycle ergometer. Although that study (276) has advanced our knowledge of the contribution the motor cortex has to central motor drive during dynamic whole-body exercise, peripheral contributions to changes in corticospinal excitability were not assessed and the exercise bout was not performed under fatiguing conditions. Thus, it is vital for researchers to develop techniques, similar to those of Sidhu and colleagues (276), to measure neuromuscular function immediately post-exercise, ideally whilst the participant remains on the cycle ergometer. This knowledge would provide a clearer indication of the effects of whole-body exercise on the magnitude of change in neuromuscular function, the impact of the time delay between exercise cessation and neuromuscular assessment, and whether or not previous research studies have underestimated the magnitude neuromuscular fatigue (112, 243).

The results of the present research also indicate that increases in lower limb muscle activation (particularly GMax) occur during exhaustive cycling. If such increases in muscle activity are indeed employed to cope with muscular fatigue, it is hypothesised that earlier recruitment of these muscles during the exercise bout may enhance exercise capacity. It would be of interest in future studies to examine the use of EMG biofeedback during cycling, to determine if earlier recruitment of hip/knee extensors would alleviate muscular work and thus fatigue in the power-producing vastii muscles, and therefore enhance exercise capacity. That is, it is of interest to determine whether or not it is possible to prolong exercise performance if an individual volitionally changes their technique early in an exercise bout.

In Study 2 it was revealed that muscle activation and joint kinematic compensatory strategies are employed in response to neuromuscular fatigue development. For the first time, these changes were matched with specific neuromuscular fatigue mechanisms and their relationships examined. To extend upon this knowledge it would also be beneficial to understand how these compensatory changes impact on pedalling kinetics. Only one study has investigated the changes in muscle activity/coordination and pedalling efficiency, with changes in joint kinematics during short-duration (3 min) cycling performed at various exercise intensities ranging from 25-90%  $\text{VO}_{2\text{max}}$  (39). The results of that study indicated that an increased

pedalling efficiency is achieved through greater ankle dorsiflexion during the upstroke and greater ankle plantar flexion at the top and bottom of the pedal stroke, as well as a change in the coordination of muscles acting at the same joint and sequential muscle activation from the knee to hip to ankle, but is independent of the pattern of pedal force application (39). These findings (39) highlight the direct association between changes in muscle activation, cycling kinematics and pedalling technique. Therefore, a subsequent step in this line of research would be to examine muscle activation, pedalling efficiency and joint kinematics, and the interrelationships between these during a fatiguing cycling task. Such research could result in a greater understanding of the influence of neuromuscular fatigue on biomechanical compensatory strategies (i.e., muscle recruitment, joint kinematics and pedal force application) employed during fatiguing cycling. In addition, future research is necessary to understand whether the association between sagittal plane kinematic changes and peripheral fatigue, and coronal plane kinematic changes and central facilitation, are evident during other movement patterns, particularly during high-intensity and repetitive tasks. This knowledge would allow indications to whether such changes are commonly employed during human movement by the neuromuscular system to cope with fatigue accumulation.

Within this thesis it was revealed for the first time in Study 3 (Chapter 5) that regardless of whether or not well-trained cyclists are able to control the level of kinematic variability when fatigued, acute exposure to real-time, kinematic feedback to limit movement of the trunk/hip joints during high-intensity cycling may influence cycling kinematics (i.e., technique) and performance and, in some cases (e.g. trunk medio-lateral sway), may be detrimental to exercise capacity. It is important to acknowledge that a primary purpose of this research was to examine the acute effects of real-time, visual kinematic feedback provision. In future, it would be ideal to determine alternative ways to provide specific joint angle feedback in real-time, as this may assist in improving the ability of athletes to correctly use the feedback provided. In addition, to determine whether or not the use of prolonged kinematic feedback exposure i.e., training programmes, would alter movement kinematics, the development of fatigue and overall exercise performance. Given that cycling is a complex motor skill and that modifying cycling kinematics would require substantial motor learning, training programmes would need to be long enough and appropriately provide feedback (e.g. a faded-frequency feedback schedule that considers the exposure time to feedback and when the feedback is given within the training programme) to promote intrinsic processes that develop error-detecting mechanisms and consistent kinematic patterns

(269, 323). This would allow for enhanced long-term retention, and thus the likelihood of learning the novel cycling technique (269, 323). Consideration of kinematic feedback conditions where the feedback involves the use of both limbs, i.e. hip, knee or ankle kinematics, may also be important. This is suggested given that the current results indicated that the feedback condition where participants had to control the extent of both the left and right hip movement ( $TTE_{Habd/add}$  test) was more difficult to perform, when compared to the trunk feedback conditions ( $TTE_{Tflex}$  and  $TTE_{Tsway}$  tests). This information would further our understanding of the potential benefits to promoting kinematic variability during exercise, and answers the question of whether it is possible for well-trained individuals to learn, and use, kinematic patterns that may enhance task performance.

It is also possible that more advanced methods of providing kinematic feedback are required to ensure that participants perform the cycling task within the specified joint range. Thus, future research should develop alternative ways of providing specific real-time, kinematic feedback. Recent studies have used the SwayStar™ balance system, which uses angular velocity transducers (147) and Vicon Nexus software streaming directly into MATLAB to calculate, and provide in real-time, kinematic feedback on trunk angle during walking (153), as well as hip adduction position in running (225). Indeed, for the present research attempts were made to use Vicon Nexus software to provide real time displacement of certain joint markers. However, difficulties arose when attempting to register and reconstruct joint markers correctly in real-time given the high velocity nature of the cycling movement. Therefore, it was deemed inaccurate to provide real-time joint marker displacement feedback. Future research should aim to use similar, or develop new, technology to provide real-time feedback during exercise tasks on specific kinematic variables. This may enhance correct kinematic responses during exercise, improve skill retention, and provide further understanding of manipulating technique via kinematic feedback on the time taken to complete a motor task.

The final study of this thesis was one of the first to examine the relationships between physiological and biomechanical aspects of cycling exercise (73, 173) and the results provide numerous concepts for future research studies to expand upon. For example, significant relationships were found between trunk segment kinematics and cycling performance (i.e. the time taken to reach task failure). These results were potentially related to an increased requirement for oxygen delivery to the exercising lower limbs, which may have made it difficult for cyclists to maintain power output in

positions of high trunk flexion. Future research could investigate the possible effects of an increase in trunk flexion and/or medio-lateral sway on oxygen delivery and/or iliac artery/vein blood flow during fatiguing cycling, and/or power production, during self-paced exercise tests, and its consequent impacts on cycling performance.

The overall outcomes of this thesis also lead to other important future research directions. Firstly, the studies within this thesis used cycling as a model of exercise; however, it is important to also investigate whether the temporal progression, and relative contribution, of neuromuscular fatigue mechanisms develop similarly in other exercise modalities and manual tasks. A few studies have examined neuromuscular fatigue development during prolonged tennis (122, 125) and running (242, 255). However, although these data provide valuable information in regards to the development of fatigue during prolonged work bouts, there appears to be no research examining the temporal progression of fatigue during high-intensity running, or other whole-body exercises or manual labour tasks. Such studies would allow us to better determine whether the development of neuromuscular fatigue occurs in the same way in short-term, high-intensity exercise tasks regardless of exercise modality. Secondly, specialist cyclists were recruited as participants in all studies included in this thesis, and therefore the current results are only applicable to well-trained individuals. This group of participants were recruited because it was assumed they would possess well-learned and consistent muscle activation and kinematic patterns (56, 57, 59). It is likely that novice cyclists would respond differently to this type of fatiguing exercise and exhibit different muscle activation (56, 57) and joint kinematic (56) patterns, which warrants further investigation. Finally, an important methodological concern was the type of cycling exercise task used. Constant-load cycling tests were chosen within this thesis to allow the effects of neuromuscular fatigue on muscle activation and joint kinematic changes to be examined during exercise, whilst eliminating the effects of changes in exercise intensity, i.e., changes in power output and/or cadence. For the purposes of this thesis, the decision to use constant-load exercise tests was satisfactory and allowed for reliable results, however it would be interesting in future research to examine the effects of neuromuscular fatigue on muscle activation and joint kinematic changes during self-paced exercise. This information would more accurately reflect, and replicate, cycling performance in real-world conditions, i.e., training and competition situations. Clearly, the present research has provided numerous possible directions for future research and has built upon the existing body of literature.

## 7.2 Conclusion

Collectively, the studies of this thesis have shown that the development of central and peripheral neuromuscular fatigue occurs in different temporal patterns, and that a facilitation of central motor drive, rather than central fatigue, together with peripheral fatigue may lead to task failure. With the development of significant neuromuscular fatigue, muscle activation and joint kinematic compensatory strategies appear to be employed and thus increases in movement variability occur. It is likely that these kinematic alterations occur sub-consciously since well-trained athletes are not easily able to override these changes even with clear visual feedback. Importantly, kinematic strategies employed to combat fatigue are not necessarily detrimental for exercise performance. The relationship between these kinematic strategies and physiological attributes associated with better cycling performance is different during cycling in non-fatigued and fatigued states.

Such findings improve our understanding of how the neuromuscular system acutely adapts in response to increasing levels of fatigue. This knowledge may allow coaches and/or occupational health practitioners to better understand the fatigue process during sporting and manual exercise tasks with similar characteristics to that of the current study, and help them make more informed decisions in regards to techniques that help to maintain task performance in fatiguing conditions. Despite this, it is still not known whether this pattern of fatigue development is similar during other sporting and/or manual exercise tasks, whether the effects of neuromuscular fatigue on compensatory biomechanical strategies are similar during prolonged and/or self-paced exercise, or whether the effects of long-term training with kinematic feedback on exercise performance is similar to the effects of acute kinematic feedback exposure. Clearly, further research examining these topics will provide a better indication to how individuals adapt to fatigue on a broader scale, and whether kinematic variability has the potential to enhance exercise performance.

## CHAPTER EIGHT

### References

1. Abbiss CR, Laursen PB. Models to explain fatigue during prolonged cycling. *Sports Medicine*. 2005;35(10):865-98.
2. Abt JP, Sell TC, Chu Y, Lovalekar M, Burdett RG, Lephart SM. Running kinematics and shock absorption do not change after brief exhaustive running. *Journal of Strength and Conditioning Research*. 2011;25(6):1479-85.
3. Albertus Y, Tucker R, St Clair Gibson A, Lambert EV, Hampson DB, Noakes TD. Effect of distance feedback on pacing strategy and perceived exertion during cycling. *Medicine & Science in Sports & Exercise*. 2005;37(3):461-8.
4. Allen DG, Fitts RH. Role of phosphate and calcium stores in muscle fatigue. *The Journal of Physiology*. 2001;536(3):657-65.
5. Allen DG, Lamb GD, Westerblad H. Impaired calcium release during fatigue. *Journal of Applied Physiology*. 2008;104:296-305.
6. Allen DG, Lamb GD, Westerblad H. Skeletal muscle fatigue: cellular mechanisms. *Physiological Reviews*. 2008;88:287-332.
7. Allen DG, Lannergren J, Westerblad H. Muscle cell function during prolonged activity: cellular mechanisms of fatigue. *Experimental Physiology*. 1995;80:497-527.
8. Almeida TF, Roizenblatt S, Tufik S. Afferent pain pathways: a neuroanatomical review. *Brain Research*. 2004;1000:40-56.
9. Amann M. Central and peripheral fatigue: interaction during cycling exercise in humans. *Medicine & Science in Sports & Exercise*. 2011;43(11):2039-45.
10. Amann M, Dempsey JA. Locomotor muscle fatigue modifies central motor drive in healthy humans and imposes a limitation to exercise performance. *The Journal of Physiology*. 2008;586(1):161-73.

11. Amann M, Eldridge MW, Lovering AT, Stickland MK, Pegelow DF, Dempsey JA. Arterial oxygenation influences central motor output and exercise performance via effects on peripheral locomotor muscle fatigue in humans. *The Journal of Physiology*. 2006;575(3):937-52.
12. Amann M, Kayser B. Nervous system function during exercise in hypoxia. *High Altitude Medicine and Biology*. 2009;10(2):149-64.
13. Amann M, Proctor LT, Sebranek JJ, Eldridge MW, Pegelow DF, Dempsey JA. Somatosensory feedback from the limbs exerts inhibitory influences on central neural drive during whole body endurance exercise. *Journal of Applied Physiology*. 2008;105:1714-24.
14. Amann M, Proctor LT, Sebranek JJ, Pegelow DF, Dempsey JA. Opioid-mediated muscle afferents inhibit central motor drive and limit peripheral muscle fatigue development in humans. *The Journal of Physiology*. 2009;587(1):271-83.
15. Amann M, Romer LM, Pegelow DF, Jacques AJ, Hess CJ, Dempsey JA. Effects of arterial oxygen content on peripheral locomotor muscle fatigue. *Journal of Applied Physiology*. 2006;101:119-27.
16. Ament W, Verkerke GJ. Exercise and fatigue. *Sports Medicine*. 2009;39:389-422.
17. Ansley L, Cangle P. Determinants of "optimal" cadence during cycling. *European Journal of Sport Sciences*. 2009;9(2):61-85.
18. Apriantono T, Nunome H, Ikegami Y, Sano S. The effect of muscle fatigue on instep kicking kinetics and kinematics in association football. *Journal of Sports Sciences*. 2006;24(9):951-60.
19. Arabadzhiev TI, Dimitrov VG, Dimitrova NA, Dimitrov GV. Interpretation of EMG integral or RMS and estimates of "neuromuscular efficiency" can be misleading in fatiguing contraction. *Journal of Electromyography and Kinesiology*. 2010;20(2):223-32.
20. Ashe MC, Scroop GC, Frisken PI, Amery CA, Wilkins MA, Khan KM. Body position affects performance in untrained cyclists. *British Journal of Sports Medicine*. 2003;37:441-4.
21. Atkinson G, Davison R, Jeukendrup A, Passfield L. Science and cycling: current knowledge and future directions for research. *Journal of Sports Sciences*. 2003;21:767-87.



22. Babault N, Pousson M, Michaut A, Van Hoecke J. Effect of quadriceps femoris muscle length on neural activation during isometric and concentric contractions. *Journal of Applied Physiology*. 2003;94:983-90.
23. Balmer J, Davidson RC, Bird SR. Peak power predicts performance power during an outdoor 16.1-km cycling time trial. *Medicine & Science in Sports & Exercise*. 2000;32(8):1485-90.
24. Barbosa TM, Keskinen KL, Fernandes R, Colaco P, Carmo C, Vilas-Boas JP. Relationships between energetic, stroke determinants and velocity in butterfly. *International Journal of Sports Medicine*. 2005;26:841-6.
25. Bartlett R, Wheat J, Robins M. Is movement variability important for sports biomechanists? *Sports Biomechanics*. 2007;6(2):224-43.
26. Basmajian JV, De Luca CJ. *Muscles alive: their functions revealed by electromyography*. Baltimore, MD: Williams & Wilkins; 1985, 57-68 p.
27. Bawa P, Murnaghan C. Motor unit rotation in a variety of human muscles. *Journal of Neurophysiology*. 2009;102:2265-72.
28. Bellemare F, Woods JJ, Johansson R, Bigland-Ritchie B. Motor-unit discharge rates in maximal voluntary contractions of three human muscles. *Journal of Neurophysiology*. 1983;50:1380-92.
29. Bender MHM, Schep G, de Vries WR, Hoogeveen AR, Wijn PFF. Sports-related flow limitations in the iliac arteries in endurance athletes: aetiology, diagnosis, treatment and future developments. *Sports Medicine*. 2004;34(7):427-42.
30. Bernstein NA. *The coordination and regulation of movements*. Oxford, UK: Pergamon Press; 1967.
31. Bigland-Ritchie B, Johansson R, Lippold OC, Smith S, Woods JJ. Changes in motoneurone firing rates during sustained maximal voluntary contractions. *The Journal of Physiology*. 1983;340(335-346).
32. Bigland-Ritchie B, Woods JJ. Changes in muscle contractile properties and neural control during human muscular fatigue. *Muscle & Nerve*. 1984;7:691-9.
33. Billaut F, Basset FA, Falgairette G. Muscle coordination changes during intermittent cycling sprints. *Neuroscience Letters*. 2005;380(3):265-9.



34. Billaut F, Bishop DJ, Schaerz S, Noakes TD. Influence of knowledge of sprint number on pacing during repeated-sprint exercise. *Medicine & Science in Sports & Exercise*. 2011;43(4):665-72.
35. Bini RR, Carpes FP, Diefenthaler F. Effects of knee frontal plane position on pedal forces during cycling: a preliminary study. *Journal of Kinanthropometry and Human Performance*. 2009;11(2):142-50.
36. Bini RR, Carpes FP, Diefenthaler F, Mota CB, Guimarães ACS. Physiological and electromyographic responses during 40-km cycling time trial: relationship to muscle coordination and performance. *Journal of Science and Medicine in Sport*. 2008;11(4):363-70.
37. Bini RR, Diefenthaler F. Kinetics and kinematics analysis of incremental cycling to exhaustion. *Sports Biomechanics*. 2010;9(4):223-35.
38. Bini RR, Diefenthaler F, Mota CB. Fatigue effects on the coordinative pattern during cycling: kinetics and kinematics evaluation. *Journal of Electromyography and Kinesiology*. 2010;20(1):102-7.
39. Blake OM, Champoux Y, Wakeling JM. Muscle coordination patterns for efficient cycling. *Medicine & Science in Sports & Exercise*. 2012;44(5):926-38.
40. Bojsen-Moller J, Hansen P, Aagaard M, Kjaer M, Magnusson SP. Measuring mechanical properties of the vatus lateralis tendon-aponeurosis complex in vivo by ultrasound imaging. *Scandinavian Journal of Medicine and Science in Sports*. 2003;13:259-65.
41. Bonato P, Ebenbichler GR, Roy SH et al. Muscle fatigue and fatigue-related biomechanical changes during a cyclic lifting task. *Spine*. 2003;28(16):1810-20.
42. Bonnard A, Sirin AV, Oddsson L, Thorstensson A. Different strategies to compensate for the effects of fatigue revealed by neuromuscular adaptation processes in humans. *Neuroscience Letters*. 1994;166:101-5.
43. Booth J, McKenna MJ, Ruell PA et al. Impaired calcium pump function does not slow relaxation in human skeletal muscle after prolonged exercise. *Journal of Applied Physiology*. 1997;83:511-21.
44. Borg GAV. *Borg's perceived exertion and pain scales*. Champaign, IL: Human Kinetics; 1998.

45. Bosch T, Mathiassen SE, Visser B, de Looze MP, van Dieen JH. The effect of work pace on workload, motor variability and fatigue during simulated light assembly work. *Ergonomics*. 2011;54:154-68.
46. Broker JP, Gregor JP, Schmidt RA. Extrinsic feedback and the learning of kinetic patterns in cycling. *Journal of Applied Biomechanics*. 1993;9:111-23.
47. Bruton JD, Westerblad H, Katz A, Lannergren J. Augmented force output in skeletal muscle fibres of *Xenopus* following a preceding bout of activity. *The Journal of Physiology*. 1996;493:211-7.
48. Bruton JD, Wretman C, Katz A, Westerblad H. Increased tetanic force and reduced myoplasmic [Pi] following a brief series of tetani in mouse soleus muscle. *American Journal of Physiology: Cell Physiology*. 1997;272:C870-C4.
49. Burgess-Limerick R, Abernethy B, Neal RJ, Kippers V. Self-selected manual lifting technique - functional consequences of the interjoint coordination. *Human Factors*. 1995;37(2):395-411.
50. Butler JE, Taylor JL, Gandevia SC. Responses of Human Motoneurons to Corticospinal Stimulation during Maximal Voluntary Contractions and Ischemia. *The Journal of Neuroscience*. 2003;23(32):10224-30.
51. Cady EBJ, D A, Lynn J, Newham DJ. Changes in force and intracellular metabolites during fatigue of human skeletal muscle. *The Journal of Physiology*. 1989;418:311-25.
52. Cairns SP, Lindinger MI. Do multiple ionic interactions contribute to skeletal muscle fatigue? *The Journal of Physiology*. 2008;586(17):4039-54.
53. Camic CL, Housh TJ, Johnson GO et al. An EMG frequency-based test for estimating the neuromuscular fatigue threshold during cycle ergometry. *European Journal of Applied Physiology*. 2010;108(2):337-45.
54. Cavanagh PR, Williams KR. The effect of stride length variation on oxygen uptake during distance running. *Medicine & Science in Sports & Exercise*. 1982;14:30-5.
55. Chaouloff F. Effects of acute physical exercise on central serotonergic systems. *Medicine & Science in Sports & Exercise*. 1997;29(1):58-62.

56. Chapman AR, Vicenzino B, Blanch P, Hodges P. Do differences in muscle recruitment between novice and elite cyclists reflect different movement patterns or less skilled muscle recruitment? *Journal of Science and Medicine in Sport*. 2008;12(1):31-4.
57. Chapman AR, Vicenzino B, Blanch P, Hodges PW. Patterns of leg muscle recruitment vary between novice and highly trained cyclists. *Journal of Electromyography and Kinesiology*. 2008;18:359-71.
58. Chapman AR, Vicenzino B, Blanch P, Knox JJ, Dowlan S, Hodges PW. The influence of body position on leg kinematics and muscle recruitment during cycling. *Journal of Science and Medicine in Sport*. 2008;11(6):519-26.
59. Chapman AR, Vicenzino B, Blanch P, Knox JJ, Hodges PW. Leg muscle recruitment in highly trained cyclists. *Journal of Sports Sciences*. 2006;24(2):115-24.
60. Chaudhuri A, Behan PO. Fatigue and basal ganglia. *Journal of the Neurological Sciences*. 2000;179:34-42.
61. Christina KA, White SC, Gilchrist LA. Effect of localized muscle fatigue on vertical ground reaction forces and ankle joint motion during running. *Human Movement Science*. 2001;20:257-76.
62. Chu DPK. The effects of augmented feedback training in cadence acquisition. *Research in Sports Medicine*. 2006;14:135-47.
63. Cignetti F, Schena F, Rouard A. Effects of fatigue on inter-cycle variability in cross-country skiing. *Journal of Biomechanics*. 2009;42(10):1452-9.
64. Clausen T.  $\text{Na}^+\text{-K}^+$  pump regulation and skeletal muscle contractility. *Physiological Reviews*. 2003;83:1269-324.
65. Clausen T, Andersen SL, Flatman JA.  $\text{Na}^+\text{-K}^+$  pump stimulation elicits recovery of contractility in  $\text{K}^+$ -paralysed rat muscle. *The Journal of Physiology*. 1993;472:521-36.
66. Clausen T, Nielsen OB. Potassium,  $\text{Na}^+$ ,  $\text{K}^+$ -pumps and fatigue in rat muscle. *The Journal of Physiology*. 2007;584:295-304.
67. Cooke R, Pate E. The effects of ADP and phosphate on the contraction of muscle fibers. *Biophysical Journal*. 1985;48:789-98.

68. Costill DL, Kovaleski J, Porter D, Kirwin J, Fielding R, King D. Energy expenditure during front crawl swimming: predicting success in middle-distance events. *International Journal of Sports Medicine*. 1985;6:266-70.
69. Cote JN, Feldman AG, Mathieu PA, Levin MF. Effects of fatigue on intermuscular coordination during repetitive hammering. *Motor Control*. 2008;12(2):79-92.
70. Cote JN, Mathieu PA, Levin MF, Feldman AG. Movement reorganization to compensate for fatigue during sawing. *Experimental Brain Research*. 2002;146:394-8.
71. Cote JN, Raymond D, Mathieu PA, Feldman AG, Levin MF. Differences in multi-joint kinematic patterns of repetitive hammering in healthy, fatigued and shoulder-injured individuals. *Clinical Biomechanics*. 2005;20:581-90.
72. Coupland ME, Puchert E, Ranatunga KW. Temperature dependance of active tension in mammalian (rabbit psoas) muscle fibres: effect of inorganic phosphate. *The Journal of Physiology*. 2001;536:879-91.
73. Coyle EF, Feltner ME, Kautz SA et al. Physiological and biomechanical factors associated with elite endurance cycling performance. *Medicine & Science in Sports & Exercise*. 1991;23(1):93-107.
74. Dancey C, Reidy J. *Statistics without Maths for Psychology: using SPSS for Windows*. London: Prentice Hall; 2004.
75. Danion F, Latash ML, Li ZM, Zatsiorsky VM. The effect of fatigue on multifinger coordination in force production tasks in humans. *The Journal of Physiology*. 2000;523(2):523-32.
76. Darques JL, Decherchi P, Jammes Y. Mechanisms of fatigue-induced activation of group IV muscle afferents: the roles played by lactic acid and inflammatory mediators. *Neuroscience Letters*. 1998;257:109-12.
77. Darques JL, Jammes Y. Fatigue-induced changes in group IV muscle afferent activity: differences between high- and low-frequency electrically induced fatigues. *Brain Research*. 1997;750:147-54.
78. Davis MJ, Bailey SP. Possible mechanisms of central nervous system fatigue during exercise. 1997.
79. De Luca CJ. Myoelectrical manifestations of localized muscular fatigue in humans. *Critical Reviews in Biomedical Engineering*. 1984;11:251-79.

80. Debold EP, Dave H, Fitts RH. Fiber type and temperature dependence of inorganic phosphate: implications for fatigue. *American Journal of Physiology: Cell Physiology*. 2004;287:C673-C81.
81. Debold EP, Romatowski J, Fitts RH. The depressive effect of Pi on the force-pCa relationship in skinned single muscle fibers is temperature dependent. *American Journal of Physiology: Cell Physiology*. 2006;290:C1041-C50.
82. Decorte N, Lafaix PA, Millet GY, Wuyam B, Verges S. Central and peripheral fatigue kinetics during exhaustive constant-load cycling. *Scandinavian Journal of Medicine and Science in Sports*. 2012;22(3):381-91.
83. Derrick T, Dereu D, McLean S. Impacts and kinematic adjustments during an exhaustive run. *Medicine & Science in Sports & Exercise*. 2002;34(6):998-1002.
84. Diefenthaeler F, Coyle EF, Bini RR, Carpes FP, Vaz MA. Muscle activity and pedal force profile of triathletes during cycling to exhaustion. *Sports Biomechanics*. 2012;11(1):10-9.
85. Dimitrova NA, Dimitrov GV. Interpretation of EMG changes with fatigue: facts, pitfalls, and fallacies. *Journal of Electromyography and Kinesiology*. 2003;13(1):13-36.
86. Dingwell JB, Cusumano JP, Cavanagh PR, Sternad D. Local dynamic stability versus kinematic variability of continuous overground and treadmill walking. *Journal of Biomechanical Engineering*. 2001;123:27-32.
87. Dingwell JB, Joubert JE, Diefenthaeler F, Trinity JD. Changes in muscle activity and kinematics of highly trained cyclists during fatigue. *IEEE Transactions on Biomedical Engineering*. 2008;55(11):2666-74.
88. Dorel S, Couturier A, Hug F. Influence of different racing positions on mechanical and electromyographic patterns during pedalling. *Scandinavian journal of medicine & science in sports*. 2009;19(1):44-54.
89. Dorel S, Drouet JM, Couturier A, Champoux Y, Hug F. Changes in pedaling technique and muscle coordination during an exhaustive exercise. *Medicine & Science in Sports & Exercise*. 2009;41(6):1277-86.
90. Dorel S, Guilhem G, Couturier A, Hug F. Adjustment of muscle coordination during an all-out sprint cycling task. *Medicine & Science in Sports & Exercise*. 2012;44(11):2154-64.

91. Duc S, Betik A-C, Grappe F. EMG activity does not change during a time trial in competitive cyclists. *International Journal of Sports Medicine*. 2005;26(2):145-50.
92. Dutka TL, Cole L, Lamb GD. Calcium phosphate precipitation in the sarcoplasmic reticulum reduces action potential-mediated  $\text{Ca}^{2+}$  release in mammalian skeletal muscle. *American Journal of Physiology: Cell Physiology*. 2005;289:C1502-C12.
93. Dutto DJ, Levy M, Lee KK, Sidthalaw S, Smith GA. Effect of fatigue and gender on running mechanics. *Medicine & Science in Sports & Exercise*. 1997;29:S82.
94. Edwards RG, Lippold OC. The relation between force and integrated electrical activity in fatigued muscle. *The Journal of Physiology*. 1956;132:677-81.
95. Elliott B, Ackland T. Biomechanical effects of fatigue on 10,000 meter running technique. *Research Quarterly for Exercise and Sport*. 1981;52:160-6.
96. Enoka RM. Mechanisms of muscle fatigue: central factors and task dependency. *Journal of Electromyography and Kinesiology*. 1995;5(3):141-9.
97. Enoka RM, Baudry S, Rudroff T, Farina D, Klass M, Duchateau J. Unraveling the neurophysiology of muscle fatigue. *Journal of Electromyography and Kinesiology*. 2011;21(2):208-19.
98. Enoka RM, Stuart DG. Neurobiology of muscle fatigue. *Journal of Applied Physiology*. 1992;75(5):1631-48.
99. Erculi F, Supej M. Impact of fatigue on the position of the release arm and shoulder girdle over a longer shooting distance for an elite basketball player. *Journal of Strength and Conditioning Research*. 2009;23(3):1029-36.
100. Escamilla RF, Barrentine SW, Fleisig GS et al. Pitching biomechanics as a pitcher approaches muscular fatigue during a simulated baseball game. *American Journal of Sports Medicine*. 2007;35(1):23-33.
101. Evangelisti MI, Verde TJ, Andres FF, Flynn MG. Effects of handlebar position on physiological responses to prolonged cycling. *Journal of Strength and Conditioning Research*. 1995;9(4):243-6.
102. Fabiato A, Fabiato F. Effects of pH on the myofilaments and sarcoplasmic reticulum of skinned cells from cardiac and skeletal muscles. *The Journal of Physiology*. 1978;276:233-55.

103. Faria EW, Parker DL, Faria IE. The science of cycling: factors affecting performance – part 2. *Sports Medicine*. 2005;35(4):313-37.
104. Faria EW, Parker DL, Faria IE. The science of cycling: physiology and training - part 1. *Sports Medicine*. 2005;35(4):285-312.
105. Farina D. Interpretation of the surface electromyogram in dynamic contractions. *Exercise and Sport Science Reviews*. 2006;34:121-7.
106. Farina D, Macaluso A, Ferguson RA, De Vito G. Effect of power, pedal rate, and force on average muscle fiber conduction velocity during cycling. *Journal of Applied Physiology*. 2004;97:2035-41.
107. Farina D, Merletti R, Enoka RM. The extraction of neural strategies from the surface EMG. *Journal of Applied Physiology*. 2004;96(4):1486-95.
108. Fitts RH. Cellular mechanisms of muscle fatigue. *Physiological Reviews*. 1994;74(1):49-93.
109. Fitts RH. The cross-bridge cycle and skeletal muscle fatigue. *Journal of Applied Physiology*. 2008;104:551-8.
110. Forestier N, Nougier V. The effects of muscular fatigue on the coordination of a multijoint movement in human. *Neuroscience Letters*. 1998;252:187-90.
111. Fortune E, Lowery MM. Effect of extracellular potassium accumulation on muscle fiber conduction velocity: a simulation study. *Annals of Biomedical Engineering*. 2009;37(10):2105-17.
112. Froyd C, Millet GY, Noakes TD. The development of peripheral fatigue and short-term recovery during self-paced high-intensity exercise. *The Journal of Physiology*. 2013;591(Pt 5):1339-46.
113. Fryer MW, Owen VJ, Lamb GD, Stephenson GD. Effects of creatine phosphate and  $P_i$  on  $Ca^{2+}$  movements and tension development in rat skinned skeletal muscle fibres. *The Journal of Physiology*. 1995;482:123-40.
114. Fryer MW, West JM, Stephenson DG. Phosphate transport into the sarcoplasmic reticulum of skinned fibres from rat skeletal muscle. *Journal of Muscle Research and Cell Motility*. 1997;18:161-7.



115. Gallina A, Merletti R, Vieira TM. Are the myoelectric manifestations of fatigue distributed regionally in the human medial gastrocnemius muscle? *Journal of Electromyography and Kinesiology*. 2011;21(6):929-38.
116. Gandevia SC. Neural control in human muscle fatigue: changes in muscle afferents, moto neurones and moto cortical drive. *Acta Physiologica Scandinavica*. 1998;162:275-83.
117. Gandevia SC. Spinal and Supraspinal Factors in Human Muscle Fatigue *Physiological Reviews*. 2001;81(4):1725-89.
118. Gandevia SC, Allen GB, Butler JE, Taylor JL. Supraspinal factors in human muscle fatigue: evidence for suboptimal output from the motor cortex. *The Journal of Physiology*. 1996;490(2):529-36.
119. Garland SW. An analysis of the pacing strategy adopted by elite competitors in 2000 m rowing. *British Journal of Sports Medicine*. 2005;39(1):39-42.
120. Gates DH, Dingwell JB. The effects of neuromuscular fatigue on task performance during repetitive goal-directed movements. *Experimental Brain Research*. 2008;187:573-85.
121. Gheluwe BV, Madsen C. Frontal rearfoot kinematics in running prior to volitional exhaustion. *Journal of Applied Biomechanics*. 1997;13:66-75.
122. Girard O, Lattier G, Maffiuletti NA, Micallef JP, Millet GP. Neuromuscular fatigue during a prolonged intermittent exercise: application to tennis. *Journal of Electromyography and Kinesiology*. 2008;18:1038-46.
123. Girard O, Micallef JP, Noual J, Millet GP. Alteration of neuromuscular function in squash. *Journal of Science and Medicine in Sport*. 2010;13(1):172-7.
124. Girard O, Millet G. Neuromuscular fatigue in racquet sports. *Neurologics Clinics*. 2008;26:181-94.
125. Girard O, Racinais S, Micallef JP, Millet GP. Spinal modulations accompany peripheral fatigue during prolonged tennis playing. *Scandinavian Journal of Medicine and Science in Sports*. 2011;21(3):455-64.
126. Glace BW, Kremenik IJ, McHugh MP. Sex differences in central and peripheral mechanisms of fatigue in cyclists. *European Journal of Applied Physiology*. 2013;113(4):1091-8.



127. Gnehm P, Reichenbach S, Altpeter E, Widmer H, Hoppeler H. Influence of different racing positions on metabolic cost in elite cyclists. *Medicine & Science in Sports & Exercise*. 1997;29:818-23.
128. Gonzalez-Alonso J. Hyperthermia impairs brain, heart and muscle function in exercising humans. *Sports Medicine*. 2007;37(4-5):371-3.
129. Grappe F, Candau R, Busso T, Rouillon JD. Effect of cycling position on ventilatory and metabolic variables. *International Journal of Sports Medicine*. 1998;19(5):336-41.
130. Hagbarth KE, Bongiovanni LG, Nordin M. Reduced servo-control of fatigued human finger extensors and flexor muscles. *The Journal of Physiology*. 1995;485:865-72.
131. Hammarskjöld E, Harms-Ringdahl K. Effect of arm-shoulder fatigue on carpenters at work. *European Journal of Applied Physiology and Occupational Therapy*. 1992;64(5):402-9.
132. Hampson DB, St Clair Gibson A, Lambert MI, Noakes TD. The influence of sensory cues on the perception of exertion during exercise and central regulation of exercise performance. *Sports Medicine*. 2001;31:935-52.
133. Hanley B, Mohan A. Gait parameter changes during 1000 metre treadmill running. In: *Proceedings of the 24 International Symposium on Biomechanics in Sports* 2006: Salzburg, Austria. p. 518-21.
134. Hausswirth C, Bigard AX, Guezennec CY. Relationships between running mechanics and energy cost of running at the end of a triathlon and a marathon. *International Journal of Sports Medicine*. 1997;18(5):330-9.
135. Hawley JA, Noakes TD. Peak power output predicts maximal oxygen uptake and performance time in trained cyclists. *European Journal of Applied Physiology*. 1992;65:79-83.
136. Heckman CJ, Gorassini MA, Bennett DJ. Persistent inward currents in motoneuron dendrites: Implications for motor output. *Muscle & Nerve*. 2005;31(2):135-56.
137. Heckman CJ, Johnson M, Mottram C, Schuster J. Persistent inward currents in spinal motoneurons and their influence on human motoneuron firing patterns. *The Neuroscientist*. 2008;14(3).

138. Heckman CJ, Lee RH, Brownstone RM. Hyperexcitable dendrites in motoneurons and their neuromodulatory control during motor behavior. *Trends in Neurosciences*. 2003;26(12).
139. Heckman CJ, Mottram C, Quinlan K, Theiss R, Schuster J. Motoneuron excitability: the importance of neuromodulatory inputs. *Clinical Neurophysiology*. 2009;120(12):2040-54.
140. Heise GD, Martin PE. Are variations in running economy in humans associated with ground reaction force characteristics? *European Journal of Applied Physiology*. 2001;84(5):438-42.
141. Hennig R, Lomo T. Firing patterns of motor units in normal rats. *Nature*. 1985;314:164-6.
142. Hermens HJ, Freriks B, Disselhorst-Klug C, Rau G. Development of recommendations for SEMG sensors and sensor placement procedures. *Journal of Electromyography and Kinesiology*. 2000;10:361-74.
143. Hettinga FJ, De Koning JJ, Broersen FT, Van Geffen P, Foster C. Pacing strategy and the occurrence of fatigue in 4000-m cycling time trials. *Medicine & Science in Sports & Exercise*. 2006;38(8):1484-91.
144. Hill AV. *Muscular activity*. Baltimore, MD: Williams and Wilkins; 1926.
145. Housh TJ, Perry SR, Bull AJ et al. Mechanomyographic and electromyographic responses during submaximal cycle ergometry. *European Journal of Applied Physiology*. 2000;83(4-5):381-7.
146. HufFenus F, Amarantini D, Forestier N. Effects of distal and proximal arm muscles fatigue on multi-joint movement organization. *Experimental Brain Research*. 2006;170:438-47.
147. Huffman L, Norton LE, Adkin AL, Allum JH. Directional effects of biofeedback on trunk sway during stance tasks in healthy young adults. *Gait & posture*. 2010;32(1):62-6.
148. Hug F, Bendahan D, Le Fur Y, Cozzone PJ, Grelot L. Heterogeneity of muscle recruitment pattern during pedaling in professional road cyclists: a magnetic resonance imaging and electromyography study. *European Journal of Applied Physiology*. 2004;92(3):334-42.
149. Hug F, Dorel S. Electromyographic analysis of pedaling: a review. *Journal of Electromyography and Kinesiology*. 2009;19(2):182-98.

150. Hug F, Drouet JM, Champoux Y, Couturier A, Dorel S. Interindividual variability of electromyographic patterns and pedal force profiles in elite cyclists. *European Journal of Applied Physiology*. 2008;104:667-78.
151. Hug F, Turpin NA, Couturier A, Dorel S. Consistency of muscle synergies during pedaling across different mechanical constraints. *Journal of Neurophysiology*. 2011;106:91-103.
152. Hug F, Turpin NA, Guevel A, Dorel S. Is interindividual variability of EMG patterns in trained cyclists related to different muscle synergies? *Journal of Applied Physiology*. 2010;108:1727-36.
153. Hunt MA, Simic M, Hinman RS, Bennell KL, Wrigley TV. Feasibility of a gait retraining strategy for reducing knee joint loading: increased trunkle angle guided by real-time biofeedback. *Journal of Biomechanics*. 2011;44:943-7.
154. Huysmans MA, Hoozemans MJ, van der Beek AJ, de Looze MP, van Dieen JH. Fatigue effects on tracking performance and muscle activity. *Journal of Electromyography and Kinesiology*. 2008;18:410-9.
155. Jones DA. High- and low- frequency fatigue revisited. *Acta Physiologica Scandinavica*. 1996;156:265-70.
156. Juel C. Muscle action potential propagation velocity changes during activity. *Muscle & Nerve*. 1988;11:714-9.
157. Katsavelis D, Kyvelidou A, Stergiou N. The effect of fatigue on movement variability during running. Omaha, NE: University of Nebraska Omaha; 2004.
158. Kay D, Marino FE, Cannon J, St Clair Gibson A, Lambert MI, Noakes TD. Evidence for neuromuscular fatigue during high-intensity cycling in warm, humid conditions. *European Journal of Applied Physiology*. 2001;84(1-2):115-21.
159. Kellis E, Katis A, Vrabas IS. Effects of an intermittent exercise fatigue protocol on biomechanics of soccer kick performance. *Scandinavian Journal of Medicine and Science in Sports*. 2006;16:334-44.
160. Kellis E, Liassou C. The effect of selective muscle fatigue on sagittal lower limb kinematics and muscle activity during level running. *Journal of Orthopaedic and Sports Physical Therapy*. 2009;39(3):210-20.

161. Kiernan MC, Lin CS, Burke D. Differences in activity-dependant hyperpolarization in human sensory and motor axons. *The Journal of Physiology*. 2004;558(Pt 1):341-9.
162. Knaflitz M, Molinari F. Assessment of muscle fatigue during biking. *IEEE Transactions on Biomedical Engineering*. 2003;11(1):17-23.
163. Knuth ST, Dave H, Peters JR, Fitts RH. Low cell pH depresses peak power in rat skeletal muscle fibres at both 30°C and 15°C: implications for muscle fatigue. *The Journal of Physiology*. 2006;575:887-99.
164. Kouzaki M, Shinohara M, Masani K, Kanehisa H, Fukunaga T. Alternate muscle activity observed between knee extensor synergists during low-level sustained contractions. *Journal of Applied Physiology*. 2002;93:675-84.
165. Kram R, Taylor CR. Energetics of running: a new perspective. *Nature*. 1990;346(6281):265-7.
166. Kremenik IJ, Glace BW, Ben-Avi SS, Nicholas SJ, McHugh MP. Central fatigue after cycling evaluated using peripheral magnetic stimulation. *Medicine & Science in Sports & Exercise*. 2009;41(7):1461-6.
167. Kyrolainen H, Pullinen T, Candau R, Avela J, Huttunen P, Komi PV. Effects of marathon running on running economy and kinematics. *European Journal of Applied Physiology*. 2000;82:297-304.
168. Lambert EV, St Clair Gibson A, Noakes TD. Complex systems model of fatigue: integrative homeostatic control of peripheral physiological systems during exercise in humans. *British Journal of Sports Medicine*. 2005;39:52-62.
169. Laursen PB, Shing CM, Jenkins DG. Reproducibility of the cycling time to exhaustion at  $\text{VO}_{2\text{peak}}$  in highly trained cyclists. *Canadian Journal of Applied Physiology*. 2003;28(4):605-15.
170. Lee RH, Heckman CJ. Influence of voltage-sensitive dendritic conductances on bistable firing and effective synaptic current in the cat spinal motoneurons in vivo. *Journal of Neurophysiology*. 1996;76:2107-10.
171. Lee RH, Heckman CJ. Adjustable amplification of synaptic input to dendritic currents on input-output processing in spinal motoneurons in vivo. *Journal of Neurophysiology*. 2000;89:27-39.
172. Lees AD, T. The effects of fatigue on soccer kick performance. *Journal of Sports Sciences*. 1988;8:156-7.

173. Leirdal S, Ettema G. The relationship between cadence, pedalling technique and gross efficiency in cycling. *European Journal of Applied Physiology*. 2011;111:2885–93.
174. Lepasalu L, Ereline J, Gapeyeva H, Kums T, Paasuke M. Changes in electromyographic parameters during cycling exercise at constant intensity. *Acta Kinesiologiae Universitatis Tartuensis*. 2011;17:99-108.
175. Lepers R, Hausswirth C, Maffiuletti NA, Brisswalter J, Van Hoecke J. Evidence of neuromuscular fatigue after prolonged cycling exercise. *Medicine & Science in Sports & Exercise*. 2000;32(11):1882-6.
176. Lepers R, Maffiuletti NA, Rochette L, Brugniaux J, Millet G. Neuromuscular fatigue during a long-duration cycling exercise. *Journal of Applied Physiology*. 2002;92:1487-93.
177. Lepers R, Millet G, Maffiuletti NA. Effect of cycling cadence on contractile and neural properties of knee extensors. *Medicine & Science in Sports & Exercise*. 2001;33:1882-8.
178. Lepers R, Theural J, Hausswirth C, Bernard T. Neuromuscular fatigue following constant versus variable-intensity endurance cycling in triathletes. *Journal of Science and Medicine in Sport*. 2008;11(4):381-9.
179. Lim CS, Gohel MS, Shepherd AC, Davies AH. Iliac artery compression in cyclists: mechanisms, diagnosis and treatment. *European Journal of Vascular and Endovascular Surgery* 2009;38:180-6.
180. Lindinger MI. Determinants of surface membrane and transverse-tubular excitability in skeletal muscle: implications for high-intensity exercise. *Equine and Comparative Exercise Physiology*. 2005;2:209-17.
181. Lucia A, Hoyos J, Chicharro JL. Physiology of professional road cycling. *Sports Medicine*. 2001;31(5):325-37.
182. Lucia A, Joyos H, Chicharro JL. Physiological response to professional road cycling: climbers vs. time trialists. *International Journal of Sports Medicine*. 2000;21:505-12.
183. Lucia A, Pardo J, Durantez A, Hoyos J, Chicharro JL. Physiological differences between professional and elite road cyclists. *International Journal of Sports Medicine*. 1998;19(5):342–8.

184. Lunnen JD, Yack J, LeVeau BF. Relationship between muscle length, muscle activity, and torque of the hamstring muscles. *Physical Therapy*. 1981;61(2):190-5.
185. MacDonald JH, Faria D, Marcora SM. Response of electromyographic variables during incremental and fatiguing cycling. *Medicine & Science in Sports & Exercise*. 2008;40(2):335-44.
186. Madeleine P. On functional motor adaptations: from the quantification of motor strategies to the prevention of musculoskeletal disorders in the neck-shoulder region. *Acta Physiologica*. 2010;199(Suppl. 679):1-46.
187. Maganaris CN, Paul JP. In vivo human tendon mechanical properties. *The Journal of Physiology*. 1999;521(1):307-13.
188. Manning CD, Miller TA, Burnham ML, Murnaghan CD, Calancie B, Bawa P. Recovery of human motoneurons during rotation. *Experimental Brain Research*. 2010;204:139-44.
189. Marcora SM. Perception of effort during exercise is independent of afferent feedback from skeletal muscles, heart, and lungs. *Journal of Applied Physiology*. 2009;106:2060-2.
190. Marcora SM, Staiano W. The limit to exercise tolerance in humans: mind over muscle? *European Journal of Applied Physiology*. 2010;109(4):763-70.
191. Marcora SM, Staiano W, Manning V. Mental fatigue impairs physical performance in humans. *Journal of Applied Physiology*. 2009;106:857-64.
192. Martin JC, Brown NAT. Joint-specific power production and fatigue during maximal cycling. *Journal of Biomechanics*. 2009;42(4):474-9.
193. Martin PG, Weerakkody SC, Gandevia SC, Taylor JL. Group III and IV muscle afferents differentially affect the motor cortex and motoneurons in humans. *The Journal of Physiology*. 2008;586(5):1277-89.
194. Martin V, Kerhervé H, Messonnier LA et al. Central and peripheral contributions to neuromuscular fatigue induced by a 24-h treadmill run. *Journal of Applied Physiology*. 2010;108:1224-33.
195. Mathiassen SE, Møller T, Forsman M. Variability in mechanical exposure within and between individuals performing a highly constrained industrial work task. *Ergonomics*. 2003;46:800-24.

196. Mauger AR, Jones AM, Williams CA. Influence of feedback and prior experience on pacing during a 4-km Cycle Time Trial. *Medicine & Science in Sports & Exercise*. 2009;41(2):451-8.
197. Mauger AR, Jones AM, Williams CA. The effect of non-contingent and accurate performance feedback on pacing and time trial performance in 4-km track cycling. *British Journal of Sports Medicine*. 2011;45:225-9.
198. McKenna MJ, Bangsbo J, Renaud J. Muscle  $K^+$ ,  $Na^+$ , and  $Cl^-$  disturbances and  $Na^+-K^+$  pump inactivation: implications for fatigue. *Journal of Applied Physiology*. 2008;104:288-95.
199. McLean L, Goudy N. Neuromuscular response to sustained low-level muscle activation: within- and between-synergist substitution in the triceps surae muscles. *European Journal of Applied Physiology*. 2004;91:204-16.
200. Meeusen R, Watson P, Hasegawa H, Roelands B, Piacentini MF. Central fatigue: The central fatigue hypothesis and beyond. *Sports Medicine*. 2006;36(10):881-909.
201. Merton PA. Voluntary strength and fatigue. *The Journal of Physiology*. 1954;123:553-64.
202. Metzger JM, Moss RL. Greater hydrogen ion-induced depression of tension and velocity in skinned single fibres of rat fast than slow muscles. *The Journal of Physiology*. 1987;393:727-42.
203. Miles GB, Dai Y, Brownstone RM. Mechanisms underlying the early phase of a spike frequency adaptation in mouse spinal motoneurons. *The Journal of Physiology*. 2005;566:519-32.
204. Millet G. Can neuromuscular fatigue explain running strategies and performance in ultra-marathons? The flush model. *Sports Medicine*. 2011;41(6):489-506.
205. Millet GY, Lepers R. Alterations of neuromuscular function after prolonged running, cycling and skiing exercises. *Sports Medicine*. 2004;34(2):105-16.
206. Millet GY, Lepers R, Maffiuletti NA, Babault N, Martin V, Lattier G. Alterations of neuromuscular function after an ultramarathon. *Journal of Applied Physiology*. 2002;92:486-92.
207. Millet GY, Martin V, Lattier G, Ballay Y. Mechanisms contributing to knee extensor strength loss after prolonged running exercise. *Journal of Applied Physiology*. 2003;94:193-8.



208. Millet GY, Martin V, Maffiuletti NA, Martin A. Neuromuscular fatigue after a ski skating marathon. *Canadian Journal of Applied Physiology*. 2003;28(3):434-45.
209. Millet GY, Millet GP, Lattier G, Maffiuletti NA, Candau R. Alteration of neuromuscular function after a prolonged road cycling race. *International Journal of Sports Medicine*. 2003;24:190-4.
210. Missenard O, Mottet D, Perrey S. The role of cocontraction in the impairment of movement accuracy with fatigue. *Experimental Brain Research*. 2008;185:151-6.
211. Mizrahi J, Verbitsky O, Isakov E. Fatigue-induced changes in decline running. *Clinical Biomechanics*. 2001;16:207-12.
212. Mizrahi J, Verbitsky O, Isakov E, Daily D. Effect of fatigue on leg kinematics and impact acceleration in long distance running. *Human Movement Science*. 2000;19:139-51.
213. Moore RL, Stull JT. Myosin light chain phosphorylation in fast and slow skeletal muscles in situ. *American Journal of Physiology: Cell Physiology*. 1984;247:C462-C71.
214. Moritani T, Nagata A, Muro M. Electromyographic manifestations of muscular fatigue. *Medicine & Science in Sports & Exercise*. 1982;14:198-202.
215. Mornieux G, Stapelfeldt B, Gollhofer A, Belli A. Effects of pedal type and pull-up action during cycling. *International Journal of Sports Medicine*. 2008;29(10):817-22.
216. Moseley L, Jeukendrup AE. The reliability of cycling efficiency. *Medicine & Science in Sports & Exercise*. 2001;33(4):621-7.
217. Mosso A. *Fatigue*. Third Ed ed. London: George Allen & Unwin Ltd.; 1915.
218. Muir M, Hrynkow P, Chase R, Boyce D, McLean D. The nature, cause, and extent of occupational musculoskeletal injuries among sonographers recommendations for treatment and prevention. *Journal of Diagnostic Medical Sonography*. 2004;20:317-25.
219. Murray TA, Cook TD, Werner SL, Schlegel TF, Hawkins RJ. The effects of extended play on professional baseball pitchers. *The American Journal of Sports Medicine*. 2001;29(2):137-42.



220. Neptune RR, Herzog W. Adaptation of muscle coordination to altered task mechanics during steady-state cycling. *Journal of Biomechanics*. 2000;33:165-72.
221. Neptune RR, Kautz SA, Hull ML. The effect of pedaling rate of coordination in cycle. *Journal of Biomechanics*. 1997;30:1051-8.
222. Nicol C, Komi PV, Marconnet P. Effects of marathon fatigue on running kinematics and economy. *Scandinavian Journal of Medicine and Science in Sports*. 1991;1:195-204.
223. Noakes TD, Peltonen JE, Rusko HK. Evidence that a central governor regulates exercise performance during acute hypoxia and hyperoxia. *Journal of Experimental Biology*. 2001;204(Pt 18):3225-34.
224. Noakes TD, St Clair Gibson A, Lambert EV. From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans: summary and conclusions. *British Journal of Sports Medicine*. 2005;39:120-4.
225. Noehren BS, J Davis, I. The effect of real-time gait retraining on hip kinematics, pain and function in subjects with patellofemoral pain syndrome. *British Journal of Sports Medicine*. 2011;45(9).
226. Nordstrom MA, Gorman RB, Laouris Y, Spielmann JM, Stuart DG. Does motoneuron adaptation contribute to muscle fatigue? *Muscle & Nerve*. 2007;35:135-58.
227. Nosek TM, Fender KY, Godt RE. It is diprotonated inorganic phosphate that depresses force in skeletal muscle fibres. *Science*. 1987;236:191-3.
228. Nummela A, Keranen T, Mikkelsen LO. Factors related to top running speed and economy. *International Journal of Sports Medicine*. 2007;28(8):655-61.
229. Nummela AT, Paavolainen LM, Sharwood KA, Lambert MI, Noakes TD, Rusko HK. Neuromuscular factors determining 5 km running performance and running economy in well-trained athletes. *European Journal of Applied Physiology*. 2006;97(1):1-8.
230. Nybo L, Secher NH. Cerebral perturbations provoked by prolonged exercise. *Progress in Neurobiology*. 2004;72(4):223-61.

231. Origenes MM, Blank SE, Schoene RB. Exercise ventilatory response to upright and aero-posture cycling. *Medicine & Science in Sports & Exercise*. 1993;25(5):608-12.
232. Ortenblad N, Nielsen J, Saltin B, Holmberg C. Role of glycogen availability in sarcoplasmic reticulum  $\text{Ca}^{2+}$  kinetics in human skeletal muscle. *The Journal of Physiology*. 2011;589:711-25.
233. Overgaard K, Nielsen OB, Flatman JA, Clausen T. Relations between excitability and contractility in rat soleus muscle: role of the  $\text{Na}^+\text{-K}^+$  pump and  $\text{Na}^+/\text{K}^+$  gradients. *The Journal of Physiology*. 1999;518(215-225).
234. Palmer S, Kentish JC. The role of troponin C in modulating the  $\text{Ca}^{2+}$  sensitivity of mammalian skinned cardiac and skeletal muscle fibres. *The Journal of Physiology*. 1994;480:45-60.
235. Patda AE. Some neuromuscular strategies characterising adaptation process during prolonged activity in humans. *Canadian Journal of Sport Sciences*. 1987;12:33S-44S.
236. Pate E, Bhimani M, Franks-skiba K, Cooke R. Reduced effect of pH on skinned rabbit psoas muscle mechanics at high temperatures: implications for fatigue. *The Journal of Physiology*. 1995;486:689-94.
237. Pedersen TH, de Paoli F, Nielsen OB. Increased excitability of acidified skeletal muscle: role of chloride conductance. *The Journal of General Physiology*. 2005;125:237-46.
238. Pedersen TH, Nielsen OB, Lamb GD, Stephenson DG. Intracellular acidosis enhances the excitability of working muscle. *Science*. 2004;305:1144-7.
239. Pendergast D, Zamparo P, di Prampero PE et al. Energy balance of human locomotion in water. *European Journal of Applied Physiology*. 2003;90:377-86.
240. Perrey S, Racinais S, Saimouaa K, Girard O. Neural and muscular adjustments following repeated running sprints. *European Journal of Applied Physiology*. 2010;109(6):1027-36.
241. Perry SR, Housh TJ, Weir JP, Johnson GO, Bull AJ, Ebersole KT. Mean power frequency and amplitude of the mechanomyographic and electromyographic signals during incremental cycle ergometry. *Journal of Electromyography and Kinesiology*. 2001;11:299-305.

242. Place N, Lepers R, Deley G, Millet G. Time course of neuromuscular alterations during a prolonged running exercise. *Medicine & Science in Sports & Exercise*. 2004;36(8):1347-56.
243. Place N, Yamada T, Bruton JD, Westerblad H. Muscle fatigue: from observations in humans to underlying mechanisms studied in intact single muscle fibres. *European Journal of Applied Physiology*. 2010;110(1):1-15.
244. Powers RK, Binder MD. Input-output functions of mammalian motoneurons. *Reviews of Physiology, Biochemistry and Pharmacology*. 2001;143:137-263.
245. Psek JA, Cafarelli E. Behaviour of co-actives muscles during fatigue. *Journal of Applied Physiology*. 1993;74:170-5.
246. Raasch CC, Zajac FE. Locomotor strategy for pedaling: muscle groups and biomechanical functions. *Journal of Neurophysiology*. 1999;82:515-25.
247. Raasch CC, Zajac FE, Ma B, Levine WS. Muscle coordination of maximum-speed pedaling. *Journal of Biomechanics*. 1997;30:595–602.
248. Racinais S, Girard O, Micallef JP, Perrey S. Failed excitability of spinal motoneurons induced by prolonged running exercise. *Journal of Neurophysiology*. 2007;97:596-603.
249. Rasmussen P, Nielsen J, Overgaard M et al. Reduced muscle activation during exercise related to brain oxygenation and metabolism in humans. *The Journal of Physiology*. 2010;588:1985-95.
250. Rassier DE, MacIntosh BR. Coexistence of potentiation and fatigue in skeletal muscle. *Brazilian Journal of Medical and Biological Research*. 2000;33:499-508.
251. Reid C. The mechanisms of voluntary muscle fatigue. *The Journal of Physiology*. 1928;29:17-42.
252. Riley MA, Turvey MT. Variability and determinism in motor behavior. *Journal of Motor Behaviour*. 2002;34:99-125.
253. Roelands B, Meeusen R. The possible role of serotonin, dopamine and noradrenaline in central fatigue. In: *Proceedings of the European College of Sport Science*. 2009: Oslo.

254. Romer LM, Haverkamp HC, Lovering AT, Pegelow DF, Dempsey JA. Effect of exercise-induced arterial hypoxemia on quadriceps muscle fatigue in healthy humans. *American Journal of Physiology: Cell Physiology*. 2006;290:R365-R75.
255. Ross EZ, Goodall S, Stevens A, Harris I. Time course of neuromuscular changes during running in well-trained subjects. *Medicine & Science in Sports & Exercise*. 2010;42(6):1184-90.
256. Ross EZ, Middleton N, Shave R, George K, Nowicky A. Corticomotor excitability contributes to neuromuscular fatigue following marathon running in man. *Experimental Physiology*. 2007;92(2):417-26.
257. Ryan MM, Gregor RJ. EMG profiles of lower extremity muscles during cycling at constant workload and cadence. *Journal of Electromyography and Kinesiology*. 1992;2(2):69-80.
258. Sahlin K, Harris RC, Nylinde B, Hultman E. Lactate content and pH in muscle obtained after dynamic exercise. *Pflugers Arch*. 1976;367:143-9.
259. Saldanha A, Nordlund Ekblom MM, Thorstensson A. Central fatigue affects plantar flexor strength after prolonged running. *Scandinavian Journal of Medicine and Science in Sports*. 2008;18(3):383-8.
260. Sanderson DJ, Cavanagh PR. Use of augmented feedback for the modification of the pedaling mechanics of cyclists. *Canadian Journal of Sport Sciences*. 1990;15(1):38-42.
261. Sarre G, Lepers R, Van Hoecke J. Stability of pedalling mechanics during a prolonged cycling exercise performed at different cadences. *Journal of Sports Sciences*. 2005;23(7):693-701.
262. Sarwar RN, B B, Rutherford OM. Changes in muscle strength, relaxation rate and fatiguability during the human menstrual cycle. *The Journal of Physiology*. 1996;493(Pt 1):267-72.
263. Saunders MJ, Evans EM, Arngrimsson SA, Allison JD, Warren GL, Cureton KJ. Muscle activation and the slow component rise in oxygen uptake during cycling. *Medicine & Science in Sports & Exercise*. 2000;32(12):2040-5.
264. Saunders PU, Pyne DB, Telford RD, Hawley JA. Factors affecting running economy in trained distance runners. *Sports Medicine*. 2004;34(7):465-85.

265. Savelberg HHCM, Van de Port IGL, Willems PJB. Body configuration in cycling affects muscle recruitment and movement pattern. *Journal of Applied Biomechanics*. 2003;19:310-24.
266. Sayers MGL, Tweddle AL, Every J, Wiegand A. Changes in drive phase lower limb kinematics during a 60 min cycling time trial. *Journal of Science and Medicine in Sport*. 2012;15:169-74.
267. Sbriccoli P, Sacchetti M, Felici F et al. Non-invasive assessment of muscle fiber conduction velocity during an incremental maximal cycling test. *Journal of Electromyography and Kinesiology*. 2009;19(6):380-6.
268. Scheiber P, Seifert J, Muller E. Relationships between biomechanics and physiology in older, recreational alpine skiers. *Scandinavian Journal of Medicine and Science in Sports*. 2012;22:49-57.
269. Schmidt RA, Lee TD. *Motor control and learning: a behavioral emphasis* 4th ed. Champaign, IL: Human Kinetics; 2005.
270. Schmitz JP, van Dijk JP, Hilbers PA, Nicolay K, Jeneson JA, Stegeman DF. Unchanged muscle fiber conduction velocity relates to mild acidosis during exhaustive bicycling. *European Journal of Applied Physiology*. 2012;112(5):1593-602.
271. Schnitzler C, Seifert L, Chollet D. Variability of coordination parameters at 400-m front crawl swimming pace. *Journal of Sports Science and Medicine*. 2008;8:203-10.
272. Schultz W, Tremblay L, Hollerman JR. Reward prediction in primate basal ganglia and frontal cortex. *Neuropharmacology*. 1998;37:421-9.
273. Senn W, Wyler KC, H P, Kleinle J, Luscher HR, Muller L. Size principal and information theory. *Biological Cybernetics*. 1997;76(1):11-22.
274. Sheel AW, Lama I, Potvin P, Coutts KD, McKenzie DC. Comparison of aero-bars versus traditional cycling postures on physiological parameters during submaximal cycling. *Canadian Journal of Applied Physiology*. 1996;21(1):16-22.
275. Sidhu SK, Bentley DJ, Carroll TJ. Locomotor exercise induces long-lasting impairments in the capacity of the human motor cortex to voluntarily activate knee extensor muscles. *Journal of Applied Physiology*. 2009;106:556-65.

276. Sidhu SK, Hoffman BW, Cresswell AG, Carroll TJ. Corticospinal contributions to lower limb muscle activity during cycling in humans. *Journal of Neurophysiology*. 2012;107:306-14.
277. Sjogaard G, Kiens B, Jorgensen K, Saltin B. Intramuscular pressure, EMG and blood flow during low-level prolonged static contraction in man. *Acta Physiologica Scandinavica*. 1986;128:475-84.
278. Smets EMA, Garssen B, Bonke B, de Haes JCJM. The multidimensional fatigue inventory (MFI) psychometric qualities of an instrument to assess fatigue. *Journal of Psychosomatic Research*. 1995;39:315-25.
279. Sparto PJ, Parnianpour M, Reinsel TE, Simon S. The effect of fatigue on multijoint kinematics, coordination and postural stability during a repetitive lifting test. *Journal of Orthopaedic and Sports Physical Therapy*. 1997;25(1):3-12.
280. Sparto PJ, Parnianpour M, Reinsel TE, Simon S. The effects of fatigue on multijoint kinematics and load sharing during a repetitive lifting task. *Spine*. 1997;22:2647-54.
281. Spector SA, Jackman MR, Sabounjian LA, Sakkas C, Landers DM, Willis WT. Effect of choline supplementation on fatigue in trained cyclists. *Medicine & Science in Sports & Exercise*. 1995;27(5):668-73.
282. Srinivasan D, Mathiassen SE. Motor variability in occupational health and performance. *Clinical Biomechanics*. 2012;27(10):979-93.
283. St Clair Gibson A, Noakes TD. Evidence for complex system integration and dynamic neural regulation of skeletal muscle recruitment during exercise in humans. *British Journal of Sports Medicine*. 2004;38:797-806.
284. Stewart D, Farina D, Shen C, Macaluso A. Muscle fibre conduction velocity during a 30-s Wingate anaerobic test. *Journal of Electromyography and Kinesiology*. 2011;21(3):418-22.
285. Stone MRK, Wilkinson TM, Jones AM, St Clair Gibson A, Thompson KG. Effects of deception on exercise performance: implications for determinants of fatigue in humans. *Medicine & Science in Sports & Exercise*. 2012;44(3):534-41.
286. Storen O, Helgerud J, Hoff J. Running stride peak forces inversely determine running economy in elite runners *Journal of Strength and Conditioning Research*. 2011;25(1):117-23.

- 287. Svirskis G, Hounsgaard J. Depolarization-induced facilitation of a plateau-generating current in ventral horn neurons in the turtle spinal cord. *Journal of Neurophysiology*. 1997;78:1740-2.
- 288. Swart J, Lamberts RP, Lambert MI et al. Exercising with reserve: exercise regulation by perceived exertion in relation to duration of exercise and knowledge of endpoint. *British Journal of Sports Medicine*. 2009;43(775-781).
- 289. Swart J, Lamberts RP, Lambert MI et al. Exercising with reserve: evidence that the central nervous system regulates prolonged exercise performance. *British Journal of Sports Medicine*. 2009;43(10):782-8.
- 290. Swart J, Lindsay TR, Lambert MI, Brown JC, Noakes TD. Perceptual cues in the regulation of exercise performance – physical sensations of exercise and awareness of effort interact as separate cues. *British Journal of Sports Medicine*. 2012;46:42-8.
- 291. Takaishi T, Yamamoto T, Ono T, Ito T, Moritani T. Neuromuscular, metabolic, and kinetic adaptations for skilled pedaling performance in cyclists. *Medicine & Science in Sports & Exercise*. 1998;30(3):442-9.
- 292. Tamaki HK, K, Akamine T, Murata F, Sakou T, Kurata H. Alternate activity in the synergist muscles during prolonged low-level contractions. *Journal of Applied Physiology*. 1998;84:1943-51.
- 293. Taylor JL, Butler JE, Gandevia SC. Changes in muscle afferents, motoneurons and motor drive during human muscle fatigue. *European Journal of Physiology*. 2000;83:106-15.
- 294. Taylor JL, Gandevia SC. A comparison of central aspects of fatigue in submaximal and maximal voluntary contractions. *Journal of Applied Physiology*. 2008;104:542-50.
- 295. Taylor JL, Todd G, Gandevia SC. Evidence for a supraspinal contribution to human muscle fatigue. *Clinical and Experimental Pharmacology and Physiology*. 2006;33:400-5.
- 296. Theural J, Lepers R. Neuromuscular fatigue is greater following highly variable versus constant intensity endurance cycling. *European Journal of Applied Physiology*. 2008;103:461-8.



297. Theurel J, Crepin M, Foissac M, Temprado JJ. Effects of different pedalling techniques on muscle fatigue and mechanical efficiency during prolonged cycling. *Scandinavian Journal of Medicine and Science in Sports*. 2012;22(6):714-21.
298. Todd G, Butler JE, Taylor JL, Gandevia SC. Hyperthermia: a failure of the motor cortex and the muscle. *The Journal of Physiology*. 2005;563(2):621-31.
299. Too D. The effect of trunk angle on power production in cycling. *Research Quarterly for Exercise and Sport*. 1994;65(4):308-15.
300. Trafimow JH, Schipplein OD, Novak GJ, Andersson GBJ. The effects of quadriceps fatigue on the technique of lifting. *Spine*. 1993;18(3):364-7.
301. Tubman L, MacIntosh BR, Maki WA. Myosin light chain phosphorylation and posttetanic potentiation in fatigued skeletal muscle. *Pflugers Archiv - European Journal of Physiology*. 1996;431:882-7.
302. Tucker R. The anticipatory regulation of performance: the development of a perception-based model for exercise performance. *British Journal of Sports Medicine*. 2009;43:392-400.
303. Tucker R. The physiological regulation of pacing strategy during exercise: a critical review. *British Journal of Sports Medicine*. 2009;43:e1.
304. Ulmer HV. Concept of an extracellular regulation of muscular metabolic rate during heavy exercise in humans by psychophysiological feedback. *Experientia*. 1996;52:416-20.
305. Uygur M, Goktepe A, Ak E, Karabörk H, Korkusuz F. The effect of fatigue on the kinematics of free throw shooting in basketball. *Journal of Human Kinetics*. 2010;24:51-6.
306. van Dieën JH, Oude Vrielink HH, Housheer AF, Lotters FB, Toussaint HM. Trunk extensor endurance and its relationship to electromyogram parameters. *European Journal of Applied Physiology*. 1993;66:388-96.
307. Vandenboom R, Houston ME. Phosphorylation of myosin and twitch potentiation in fatigued skeletal muscle. *Canadian Journal of Physiology and Pharmacology*. 1996;74(12):1315-21.
308. Vereijken B, Whiting HT, Beek WJ. A dynamical systems approach to skill acquisition. *The Quarterly Journal of Experimental Psychology A*. 1992;45(2):323-44.



309. Wakeling JM. The recruitment of different compartments within a muscle depends on the mechanics of the movement. *Biology Letters*. 2009;5(1):30-4.
310. Wakeling JM, Horn T. Neuromechanics of muscle synergies during cycling. *Journal of Neurophysiology*. 2009;101(2):843-54.
311. Watanabe K, Akima H. Neuromuscular activation of vastus intermedius muscle during fatiguing exercise. *Journal of Electromyography and Kinesiology*. 2010;20(4):661-6.
312. Watanabe K, Katayama K, Ishida K, Akima H. Electromyographic analysis of hip adductor muscles during incremental fatiguing pedaling exercise. *European Journal of Applied Physiology*. 2009;106(6):815-25.
313. Watanabe K, Kouzaki M, Moritani T. Region-specific myoelectric manifestations of fatigue in human rectus femoris muscle. *Muscle & Nerve*. 2012; Accepted Article doi: 10.1002/mus.23739.
314. Welbergen E, Clijsen LPVM. The influence of body position on maximal performance in cycling. *European Journal of Applied Physiology*. 1990;61:138-42.
315. Westerblad H, Allen DG. The contribution of  $[Ca^{2+}]_i$  to the slowing of relaxation in fatigued single fibres from mouse skeletal muscle. *The Journal of Physiology*. 1993;468:729-40.
316. Westerblad H, Allen DG. Relaxation,  $[Ca^{2+}]_i$  and  $[Mg^{2+}]_i$  during prolonged tetanic stimulation of intact, single fibres from mouse skeletal muscle. *The Journal of Physiology*. 1994;480:31-43.
317. Westerblad H, Allen DG, Lannergren J. Muscle fatigue: lactic acid or inorganic phosphate the major cause? *News in Physiological Sciences*. 2002;17(1):17.
318. Westerblad H, Bruton JD, Lannergren J. The effect of intracellular pH on contractile function of intact, single fibres of mouse muscle declines with increasing temperature. *The Journal of Physiology*. 1997;500:193-204.
319. Westerblad H, Lee JA, Lamb GD, Bolsover SR, Allen DG. Spatial gradients of intracellular calcium in skeletal muscle during fatigue. *Pflugers Arch*. 1990;415:734-40.
320. Westgaard RH, De Luca CJ. Motor unit substitution in long-duration contractions of the human trapezius muscle. *Journal of Neurophysiology*. 1999;82:501-4.

321. Williams K, Smow R, Agruss C. Changes in distance running kinematics with fatigue. *International Journal of Sports Biomechanics*. 1991;7:138-62.
322. Williams KR, Cavanagh PR. Relationship between distance running mechanics, running economy, and performance. *Journal of Applied Physiology*. 1987;63(3):1236-124.
323. Winstein CJ, Schmidt RA. Reduced frequency of knowledge of results enhances motor skill learning. *Journal of Experimental Psychology: Learning, Memory, and Cognition*. 1990;16(4):677-91.
324. Wolfe AE, Brown LE, Coburn JW, Kersey RD, Bottaro M. Time course of the effects of static stretching on cycling economy. *Journal of Strength and Conditioning Research*. 2011;25(11):2980–4.
325. Yoon J, Kang M, Oh J. Effects of visual biofeedback using a laser beam on the EMG ratio of the medial and lateral vasti muscles and kinematics of hip and knee joints during a squat exercise. *Journal of Physical Therapy Science*. 2011;23:559-63.
326. Yoshino K, Motoshige T, Araki T, Matsuoka K. Effect of prolonged free-walking fatigue on gait and physiological rhythm. *Journal of Biomechanics*. 2004;37(8):1271-80.
327. Zajac FE, Neptune RRK, S A. Biomechanics and muscle coordination in human walking. Part 1: Introduction to concepts, power transfer, dynamics, and simulations. *Gait and Posture*. 2002;16:215–32.
328. Zamparo P, Pendergast D, Mollendorf J, Termin A, Minetti A. An energy balance of front crawl. *European Journal of Applied Physiology*. 2005;94:134-44.
329. Zory R, Boerio D, Jubeau M, Maffiuletti NA. Central and peripheral fatigue of the knee extensor muscles induced by electromyostimulation. *International Journal of Sports Medicine*. 2005;26:847-53.
330. Zory R, Vuillerme N, Pellegrini B, Schena F, Rouard A. Effect of fatigue on double pole kinematics in sprint cross-country skiing. *Human Movement Science*. 2009;28(1):85-98.

## CHAPTER NINE

### Appendices

<b>Appendix</b>	<b>Page</b>
A     Table 5.2     Trunk, hip, knee and ankle joint kinematics in the sagittal and coronal planes at the start ( $T_0$ ) and at task failure ( $T_{100}$ ) of high-intensity, constant-load cycling bouts performed with either no feedback (TTE test) or real-time, kinematic feedback for trunk flexion ( $TTE_{Tflex}$ test). ....	152
B     Table 5.3     Trunk, hip, knee and ankle joint kinematics in the sagittal and coronal planes at the start ( $T_0$ ) and at task failure ( $T_{100}$ ) of high-intensity, constant-load cycling bouts performed with either no feedback (TTE test) or real-time, kinematic feedback for trunk medio-lateral sway ( $TTE_{Tsway}$ test). ....	154
C     Table 5.4     Trunk, hip, knee and ankle joint kinematics in the sagittal and coronal planes at the start ( $T_0$ ) and at task failure ( $T_{100}$ ) of high-intensity, constant-load cycling bouts performed with either no feedback (TTE test) or real-time, kinematic feedback for hip abduction/adduction ( $TTE_{Habd/add}$ test). ....	156
D     Ethics approval for PhD - ECU Human Research Ethics Committee.....	158
E     Information letter – Chapters 1 and 2.....	159
F     Information letter – Chapter 3.....	163
G     Document of informed consent – Chapters 1 and 2.....	168
H     Document of informed consent – Chapter 3.....	170
I     Pre-exercise medical screening questionnaire (Chapters 1-3) .....	173
J     Multidimensional fatigue inventory (Chapters 1-3) .....	176
K     24-hour diet history record sheet (Chapters 1-3) .....	178
L     Final check-list (Chapters 1-3) .....	179
M     Prior task expectations questionnaire (Chapter 3) .....	180
N     Photos of kinematic feedback set-up and subject performing cycling protocol with the kinematic feedback.....	181
O     Copy of conference presentation (Chapters 1 and 2) .....	184

## APPENDIX A

**Table 5.2** Trunk, hip, knee and ankle joint kinematics in the sagittal and coronal planes at the start ( $T_0$ ) and at task failure ( $T_{100}$ ) of high-intensity, constant-load cycling bouts performed with either no feedback (TTE test) or real-time, kinematic feedback for trunk flexion (TTE<sub>Tflex</sub> test).

Joint	Side	Kinematic variable	TTE test		TTE <sub>Tflex</sub> test	
			T <sub>0</sub>	T <sub>100</sub>	T <sub>0</sub>	T <sub>100</sub>
Sagittal Plane						
Hip	Left	Mean angle	73.6 ± 3.6	74.4 ± 5.2	75.9 ± 5.6	75.9 ± 5.6
		ROM	46.3 ± 3.5	48.8 ± 4.0	46.3 ± 2.8	48.9 ± 2.3
	Right	Mean angle	72.6 ± 3.1	73.7 ± 4.8	73.3 ± 5.1	74.0 ± 4.5
		ROM	45.6 ± 4.8	47.8 ± 4.6	46.4 ± 3.3	48.9 ± 4.0
Knee	Left	Mean angle	80.4 ± 3.3	78.3 ± 3.5	82.2 ± 2.5	80.1 ± 2.7
		ROM	82.1 ± 4.5	85.2 ± 4.6	79.4 ± 2.6	82.6 ± 2.5
	Right	Mean angle	78.3 ± 2.8	76.8 ± 3.3*	76.6 ± 1.6	75.6 ± 1.4
		ROM	79.4 ± 5.6	82.4 ± 6.3	79.1 ± 3.0	82.1 ± 5.4
Ankle	Left	Mean angle	6.2 ± 5.9	7.0 ± 4.0	7.3 ± 5.4	7.8 ± 4.4
		ROM	22.4 ± 6.8	25.1 ± 4.8	22.8 ± 7.1	25.3 ± 4.7
	Right	Mean angle	3.8 ± 6.1	4.9 ± 6.6	4.3 ± 4.0	5.7 ± 6.4
		ROM	22.4 ± 7.1	23.5 ± 7.7	23.5 ± 7.6	22.4 ± 5.9
Trunk		Mean angle	34.3 ± 4.6	39.2 ± 7.3*	33.2 ± 5.3	35.3 ± 5.5
		ROM	1.8 ± 0.7	3.4 ± 0.7*	1.6 ± 0.4	4.0 ± 0.9*

**Table 5.2** Cont.

Joint	Side	Kinematic variable	TTE test		TTE <sub>Tflex</sub> test	
			T <sub>0</sub>	T <sub>100</sub>	T <sub>0</sub>	T <sub>100</sub>
Coronal Plane						
Hip	Left	Mean angle	-5.3 ± 4.5	-5.6 ± 3.9	-4.0 ± 3.7	-4.2 ± 2.6
		ROM	6.3 ± 3.1	8.7 ± 3.1 <sup>*</sup>	5.8 ± 3.5	8.4 ± 2.8 <sup>*</sup>
	Right	Mean angle	-2.8 ± 3.4	-1.9 ± 2.9	-3.5 ± 2.2	-3.2 ± 1.7
		ROM	5.5 ± 2.2	7.4 ± 3.1 <sup>*</sup>	5.3 ± 1.9	6.9 ± 2.4 <sup>*</sup>
Knee	Left	Mean angle	17.4 ± 12.1	19.1 ± 12.0 <sup>*</sup>	12.1 ± 10.4	14.1 ± 10.0
		ROM	19.2 ± 3.3	23.4 ± 3.0 <sup>*</sup>	17.6 ± 3.3	20.6 ± 3.7 <sup>*</sup>
	Right	Mean angle	11.1 ± 11.9	13.2 ± 10.9 <sup>*</sup>	11.9 ± 6.1	14.2 ± 5.4 <sup>*</sup>
		ROM	17.6 ± 5.1	22.5 ± 6.2	18.7 ± 3.0	23.2 ± 5.2 <sup>*</sup>
Trunk		Mean angle	0.8 ± 3.4	0.7 ± 3.2	-0.2 ± 3.8	-0.7 ± 3.3
		ROM	3.1 ± 1.4	4.6 ± 2.1	2.7 ± 0.7	4.5 ± 1.9

Data are mean ± SD for six participants.

ROM, range of motion.

\* Main effect of time ( $p < 0.05$ ).

## APPENDIX B

**Table 5.3** Trunk, hip, knee and ankle joint kinematics in the sagittal and coronal planes at the start ( $T_0$ ) and at task failure ( $T_{100}$ ) of high-intensity, constant-load cycling bouts performed with either no feedback (TTE test) or real-time, kinematic feedback for trunk medio-lateral sway (TTE<sub>Tsway</sub> test).

Joint	Side	Kinematic variable	TTE test		TTE <sub>Tsway</sub> test	
			T <sub>0</sub>	T <sub>100</sub>	T <sub>0</sub>	T <sub>100</sub>
Sagittal Plane						
Hip	Left	Mean angle	74.7 ± 3.2	74.5 ± 5.2	74.4 ± 3.9	73.0 ± 4.2
		ROM	46.4 ± 3.5	48.1 ± 3.1	49.9 ± 2.9	49.3 ± 3.4
	Right	Mean angle	72.6 ± 3.1	73.0 ± 4.3	73.4 ± 3.1	72.4 ± 2.4
		ROM	46.5 ± 4.2	49.2 ± 4.0	46.3 ± 3.5	51.0 ± 4.5*
Knee	Left	Mean angle	80.1 ± 3.5	77.7 ± 3.7	79.9 ± 4.0	75.2 ± 3.5
		ROM	81.7 ± 4.1	84.5 ± 4.0*	80.1 ± 3.3	84.5 ± 3.5*
	Right	Mean angle	77.0 ± 3.7	75.8 ± 3.2	76.8 ± 3.8	73.1 ± 2.4
		ROM	81.2 ± 4.0	85.1 ± 3.5*	80.1 ± 5.2	86.6 ± 5.1*
Ankle	Left	Mean angle	4.1 ± 7.2	4.8 ± 7.7	5.9 ± 7.4	5.2 ± 9.2
		ROM	22.2 ± 6.9	25.1 ± 4.8	25.6 ± 6.2	26.5 ± 5.0
	Right	Mean angle	2.4 ± 6.8	5.1 ± 6.4	2.4 ± 6.5	4.9 ± 6.7
		ROM	23.0 ± 7.1	22.1 ± 8.1	25.7 ± 5.8	24.6 ± 7.5
Trunk		Mean angle	36.1 ± 4.1	40.8 ± 6.2*	36.4 ± 5.2	42.1 ± 4.5*
		ROM	1.9 ± 0.7	3.8 ± 1.4*	2.2 ± 0.6	4.0 ± 0.9*

**Table 5.3** Cont.

Joint	Side	Kinematic variable	TTE test		TTE <sub>T<sub>sway</sub></sub> test	
			T <sub>0</sub>	T <sub>100</sub>	T <sub>0</sub>	T <sub>100</sub>
Coronal Plane						
Hip	Left	Mean angle	-4.5 ± 3.1	-4.9 ± 2.7	-5.4 ± 1.3	-5.0 ± 1.1
		ROM	5.9 ± 3.1	9.3 ± 3.2 <sup>*</sup>	6.2 ± 3.5	8.4 ± 3.3 <sup>*</sup>
	Right	Mean angle	-3.7 ± 3.5	-3.7 ± 3.5	-1.6 ± 2.6	-2.5 ± 2.7
		ROM	5.4 ± 2.1	7.7 ± 3.4 <sup>*</sup>	4.8 ± 2.3	7.8 ± 2.5 <sup>*</sup>
Knee	Left	Mean angle	15.8 ± 10.8	17.4 ± 10.6 <sup>*</sup>	14.2 ± 3.1	16.0 ± 2.5 <sup>*</sup>
		ROM	18.0 ± 4.1	22.5 ± 3.0 <sup>*</sup>	19.1 ± 3.1	24.3 ± 4.8 <sup>*</sup>
	Right	Mean angle	12.7 ± 12.0	14.9 ± 11.2 <sup>*</sup>	7.6 ± 10.4	9.0 ± 9.6
		ROM	17.2 ± 5.5	21.0 ± 7.0	15.1 ± 3.0	19.4 ± 2.3 <sup>*</sup>
Trunk		Mean angle	0.1 ± 2.7	-0.5 ± 2.3	0.8 ± 0.7	0.1 ± 0.2
		ROM	3.3 ± 1.3	5.5 ± 1.8	2.8 ± 0.7	4.5 ± 2.0

Data are mean ± SD for six participants.

ROM, range of motion.

\* Main effect of time ( $p < 0.05$ ).

## APPENDIX C

**Table 5.4** Trunk, hip, knee and ankle joint kinematics in the sagittal and coronal planes at the start ( $T_0$ ) and at task failure ( $T_{100}$ ) of high-intensity, constant-load cycling bouts performed with either no feedback (TTE test) or real-time, kinematic feedback for hip abduction/adduction (TTE<sub>Habd/add</sub> test).

Joint	Side	Kinematic variable	TTE test		TTE <sub>Habd/add</sub> test	
			T <sub>0</sub>	T <sub>100</sub>	T <sub>0</sub>	T <sub>100</sub>
Sagittal Plane						
Hip	Left	Mean angle	74.0 ± 4.0	75.2 ± 4.9	74.4 ± 2.9	75.8 ± 5.7
		ROM	46.4 ± 2.9	48.2 ± 4.6	46.5 ± 3.9	48.0 ± 4.0
	Right	Mean angle	73.5 ± 2.2	74.9 ± 2.4	73.7 ± 2.1	75.3 ± 4.6
		ROM	45.4 ± 4.4	46.5 ± 5.4	45.8 ± 3.2	46.8 ± 3.5
Knee	Left	Mean angle	79.0 ± 2.4	77.6 ± 3.0	78.9 ± 3.0	78.9 ± 2.5
		ROM	82.2 ± 4.5	84.1 ± 5.8*	80.6 ± 4.5	83.2 ± 6.4
	Right	Mean angle	76.5 ± 3.6	76.6 ± 3.1	76.5 ± 2.6	77.1 ± 3.0
		ROM	78.7 ± 4.4	79.9 ± 5.8	79.8 ± 4.7	78.8 ± 6.2
Ankle	Left	Mean angle	5.2 ± 5.4	3.0 ± 6.1	5.5 ± 3.9	4.1 ± 4.0
		ROM	23.0 ± 5.9	25.6 ± 4.8	22.1 ± 5.5	23.9 ± 5.1
	Right	Mean angle	0.5 ± 6.5	0.1 ± 7.9	0.8 ± 4.0	1.1 ± 8.6
		ROM	24.1 ± 5.7	24.5 ± 8.9	22.5 ± 5.1	20.8 ± 5.4
Trunk		Mean angle	34.8 ± 5.9	38.6 ± 6.8*	38.3 ± 5.6	34.4 ± 7.6*
		ROM	1.9 ± 0.5	3.9 ± 1.1*	2.0 ± 0.5	4.4 ± 3.1



**Table 5.4** Cont.

Joint	Side	Kinematic variable	TTE test		TTE <sub>Habd/add</sub> test	
			T <sub>0</sub>	T <sub>100</sub>	T <sub>0</sub>	T <sub>100</sub>
Coronal Plane						
Hip	Left	Mean angle	-6.2 ± 3.7	-6.5 ± 3.1	-4.7 ± 2.1	-4.4 ± 1.4
		ROM	7.0 ± 2.2	9.9 ± 2.3*	6.5 ± 2.6	8.5 ± 3.0*
	Right	Mean angle	-1.9 ± 2.9	-2.4 ± 3.9	-3.1 ± 1.5	-3.6 ± 2.0
		ROM	5.9 ± 1.7	8.8 ± 2.0*	5.4 ± 1.9	6.8 ± 2.3
Knee	Left	Mean angle	18.6 ± 3.6	20.6 ± 5.8*	14.7 ± 9.9	16.8 ± 9.4*
		ROM	19.4 ± 3.9	21.7 ± 3.2	17.2 ± 4.7	19.0 ± 5.4*
	Right	Mean angle	7.4 ± 9.3	9.8 ± 8.9*	11.3 ± 11.0	13.8 ± 11.0*
		ROM	16.1 ± 4.0	19.5 ± 4.9	14.9 ± 5.0	16.5 ± 6.2
Trunk		Mean angle	1.6 ± 2.2	0.9 ± 2.4	0.5 ± 1.1	-0.3 ± 1.0
		ROM	3.5 ± 1.3	5.1 ± 2.6	3.8 ± 1.5	3.7 ± 2.2*

Data are mean ± SD for seven participants.

ROM, range of motion.

\* Main effect of time ( $p < 0.05$ ).

## APPENDIX D

Dear Amanda

Project Number: 4399 OVERTON

Project Name: Neuromuscular Fatigue and Kinematics during Constant Workload High-Intensity Endurance Exercise

Ethics approval for your research project was granted from 09 December 2009 to 02 August 2012.

The *National Statement on Ethical Conduct in Human Research* requires that all approved projects are subject to monitoring conditions. This includes completion of an annual report (for projects longer than one year) and completion of a final report at the completion of the project.

A FINAL REPORT was due on 02 August 2012.

A copy of the ethics report form can be found on the [Ethics Website](#)

Please complete the ethics report form and return the signed form to the Research Ethics Office.

Note that ethics approval is required for both the collection and use (analysis) of data. If the project is still continuing, please complete the form and apply for an extension of ethics approval.

Hi Amanda

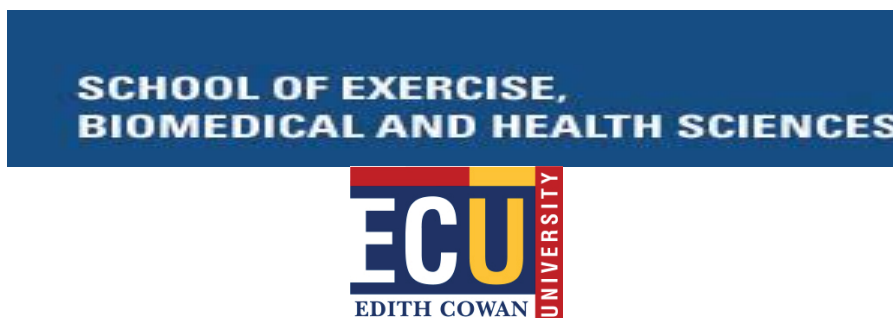
Thank you for the request for an extension to your project titled 'Neuromuscular Fatigue and Kinematics during Constant Workload High-Intensity Endurance Exercise'. The extension has been approved until 31<sup>st</sup> December 2012.

Regards

Kerry

Kerry Blowfield  
Senior Centre Officer  
Office of Research and Innovation  
Edith Cowan University  
Building 1B, 225  
Joondalup WA 6027  
Phone 08 6304 2721  
Fax 08 6304 5044  
CRICOS IPC 00279B

## APPENDIX E



### INFORMATION LETTER FOR PARTICIPANTS

#### **Studies 1 and 2: Neuromuscular fatigue and kinematics during high-intensity, constant-load cycling exercise**

Thank you for showing an interest in this research project. Please read this information letter carefully before deciding whether or not to participate. If you decide not to take part there will be no disadvantage to you of any kind and we thank you for your consideration.

#### **Aims of the Research Project**

This project is being undertaken as part of the requirements of a Doctor of Philosophy in Sports Science at Edith Cowan University (ECU). At present it is not known how central (brain and spinal cord) and peripheral (muscle) fatigue develop throughout high-intensity endurance exercise (HIEE) (maximal exercise lasting approximately 5 minutes). Furthermore, during fatiguing exercise we can change our technique to compensate for fatigue and to continue exercise; however it is not known whether or not these changes in our technique are beneficial or detrimental for exercise performance. Therefore, this project aims to determine the time-course and relative contribution of the development of both central (brain and spinal cord) and peripheral (muscle) fatigue, as well as changes in technique that occur during constant workload HIEE.

#### **Participants**

This project will include well-trained, healthy male cyclists aged between 18 and 45 years. You must have at least one year of cycling experience (track/road cycling or triathlons, inclusive of all sub-disciplines) and currently be cycling in excess of 250 km/week, and have been over at least the last year. You must also be free from major illness or injury at the time of participation.

#### **Research Project Outline**

Should you agree to take part in this project, you will be asked to participate in seven testing sessions, each separated by at least 48 hours. All testing sessions will take place in Building 19 at ECU (Joondalup campus). You will be asked to refrain from strenuous exercise/hard training 24 hours before and caffeine, alcohol and other stimulants 12 hours before each testing session. You will also be asked to ingest the same diet 12 hours before each testing session.

#### Day 1:

- Body mass, height, date of birth, training status and 24-h diet history will be recorded.
- Familiarisation with fatigue measurements will be given (electrical nerve stimulation and maximal voluntary contractions (MVC); described below).
- 5-minute warm-up on a stationary bike will be completed.
- Progressive exercise test to exhaustion. This test will involve cycling on a stationary bike as the workload/intensity of exercise increases over time. This intensity will continue to increase until you can no longer perform the workload (i.e. you voluntarily terminate the test). During the test, heart rate will be recorded using a heart rate monitor, oxygen consumption and carbon dioxide production will be measured using a gas analysis system where you breathe continuously into a mouth piece and expiratory gases are collected for analysis, and a rating of perceived exertion will be recorded.
- Time to exhaustion (TTE) test. This test will involve cycling on a stationary bike at your constant, individualised power output (the power output or workload you achieve at your maximal oxygen consumption level) until you can no longer exercise (i.e. you voluntarily terminate the test). Immediately after the TTE test, post-fatigue measurements will be taken as quickly as possible (described below).
- Total testing on this day will take approximately 3 hours of your of your time.

#### Day 2-7:

- Body mass and 24-h diet history will be recorded.
- Multidimensional Fatigue Inventory psychometric questionnaire, which provides a measure of how 'fatigued' you are feeling before the test, will be completed.
- 5-minute warm-up on a stationary bike, followed by three 3-second maximal sprints with 57 seconds recovery between sprints, will be completed.
- Multiple TTE tests
  - On the first day you will perform a TTE test (described above).
  - On the remaining five days you will be required to perform five TTE tests to *20, 40, 60, 80 or 100% of your first day TTE test duration*. Therefore instead of exercising until you cannot exercise any longer, you will be stopped by the experimenter at a specific time. Please be aware that you will not be told which test duration you will be performing on each day.
- Electrical stimulation of your femoral nerve (the nerve that runs at the front of your hip near the top of your leg) before and immediately after each test. Electrical stimulation involves placing a small electrode on the skin over the nerve, then a brief electrical current passes from the electrical stimulator, creating an electrical pulse which stimulates the nerve. The pulses are delivered in very short bursts lasting only 2 milliseconds each. The nerve will be stimulated three times, with 10 seconds rest in between each stimulus. During this test you will be seated in a chair and secured in with waist and shoulder crossover straps.
  - *This procedure is not painful*, but will cause your quadriceps to contract and you may notice involuntary movements of the knee and/or hip joint.
- 3 x MVC of the quadriceps held for 5 seconds before and immediately after each test, seated in the chair described above. During these contractions you will be stimulated again: one stimulus during the MVC and one stimulus 3 seconds after the MVC. You will be given a 30 second recovery period in between each MVC.

- Muscle activity of eight lower leg muscles, one buttocks muscle and one trunk muscle will be recorded using surface electrodes. These are gel-like pads which are stuck onto the muscle and are used to record the level of muscle activation.
- Cycling technique will be recorded using 3D motion analysis. Reflective surface markers will be stuck on various landmarks of the body over the skin and will be recorded by ten infra-red cameras. This will be used to record changes in joint angles and joint range of motion.
- HR, oxygen consumption and carbon dioxide production and a rating of perceived exertion will be recorded the same as Day 1.
- Total testing on this day will take approximately 1.5-2 hours of your of your time.

There are very few discomforts associated with these tests. As you are trained cyclists you will be accustomed to performing exercise at high intensities and this should not pose any more physical or psychological discomfort than that typically experienced during hard training sessions. You may find that breathing into the mouthpiece is a little inconvenient; however this will not be of any major discomfort. Electrical nerve stimulation may be inconvenient; however this procedure is not painful and will pose only minimal discomfort.

You are volunteering to participate in this project and therefore can withdraw from this project without explanation at any time without prejudice and with no disadvantage to yourself. Any data collected before withdrawal from the project will be destroyed. If you feel any pain or discomfort during the testing sessions please notify the experimenter immediately.

### **Risk Considerations**

There is a low risk and possible discomfort involved in performing maximal exertion exercise. This type of exercise could in rare cases lead to disastrous events, including cardiac events leading to death, and could also lead to severe exhaustion and/or fainting, however this is unlikely. There is also a low risk and possible discomfort in performing maximal voluntary contractions of the quadriceps. This type of contraction could lead to a minor injury, such as a muscle strain, however this is unlikely. There is again a low risk and possible discomfort associated with electrical nerve stimulation that would lead to minimal discomfort, but no injury, however again this is unlikely. Numerous precautionary procedures are put in place to avoid and minimise the likelihood of these risks occurring, including medical screening questionnaires, regular equipment checks, safe operating practices and physiological measures of your response to exercise/level of fatigue.

### **Benefits and Reimbursement**

By participating in this project you will receive numerous measures of your current physiological status, including your maximal knee extensor strength, maximal oxygen consumption ( $\text{VO}_2 \text{ max}$ ), maximal heart rate ( $\text{HR}_{\text{max}}$ ), peak power output and ventilatory thresholds. You will also be provided with specific information about your cycling technique, and how it changes with fatigue. You will also get the chance to observe interesting research techniques such as electrical nerve stimulation, electromyography (muscle activity) and 3D motion analysis. The results of the project will also be provided to all participants in a summary format and/or a copy of journal articles. Furthermore, after all testing sessions you will be provided with a 600 ml sports drink (Gatorade) to encourage recovery (carbohydrate, fluid and electrolyte replacement) post- exercise.

## **Ethical Considerations**

This project will comply with the guidelines set out by the National Health and Research Council. Ethical approval of this project has been granted by the Human Research Ethics Committee of ECU. All data collected during the project will be formatted so that it is unidentifiable (coded) and kept on password protected computers (soft copies) and/or in a lockable cabinet at ECU Joondalup campus (hard copies). At completion of the project all data will be retained for 5 years stored in a lockable cabinet at ECU Joondalup campus. Data collected may be used in future approved research projects, if you give permission to do so. Data will only ever be accessed by the investigators of this research project including myself and my supervisors Assoc Prof. Anthony Blazeovich and Dr. Chris Abbiss. The results of this project will be published in journal articles and my thesis, and presented at conferences, but any data included will in no way be linked to any specific participant.

Should you have any questions, concerns, or would like further information regarding this project, or would like to participate please contact me. If you would like to speak to the research supervisors please contact Assoc Prof. Anthony Blazeovich (08) 6304 5472, or Dr. Chris Abbiss (08) 6304 5740. If you have any concerns or complaints about the project, and you wish to talk to an independent person please contact the ECU Research Ethics Officer on (08) 6304 2170.

Kind Regards,

**Amanda Overton BPhEd (Hons) (PhD Candidate)**

Faculty of Computing, Health and Science  
School of Exercise, Biomedical and Health Sciences  
Edith Cowan University  
100 Joondalup Drive, Joondalup WA 6027  
Email: [a.overton@ecu.edu.au](mailto:a.overton@ecu.edu.au)  
Phone (W): (08) 6304 5097  
Mobile: 04 5096 3527

## APPENDIX F



### INFORMATION LETTER FOR PARTICIPANTS

#### **Study 3: Effects of real-time, visual kinematic feedback on time to exhaustion during high-intensity, constant-load cycling exercise**

Thank you for showing an interest in this research project. Please read this information letter carefully before deciding whether or not to participate. If you decide not to take part there will be no disadvantage to you of any kind and we thank you for your consideration.

#### **Aims of the Research Project**

This project is being undertaken as part of the requirements of a Doctor of Philosophy in Sports Science at Edith Cowan University (ECU). During fatiguing exercise we can change our technique to compensate for fatigue and to continue exercise; however it is not known whether these changes in our technique are beneficial or detrimental for exercise performance. Therefore, this project aims to determine how different techniques influence exercise performance in terms of the time taken to reach exhaustion.

#### **Participants**

This project will include well-trained, healthy male cyclists aged between 18 and 45 years. You must have at least one year of cycling experience (track/road cycling or triathlons, inclusive of all sub-disciplines) and currently be cycling in excess of 250 km/week, and have been over at least the last year. You must also be free from major illness or injury at the time of participation.

#### **Research Project Outline**

Should you agree to take part in this project, you will be asked to participate in multiple testing sessions, each separated by at least 48 hours. All testing sessions will take place in Building 19 at ECU (Joondalup campus). You will be asked to refrain from strenuous exercise/hard training 24 hours before and caffeine, alcohol and other stimulants 12 hours before each testing session. You will also be asked to follow a similar diet 12 hours before each testing session.

Day 1:

- Body mass, height, date of birth, training status and 24-h diet history will be recorded.
- 5-minute warm-up on a stationary bike will be completed.



- Progressive exercise test to exhaustion. This test will involve cycling on a stationary bike as the workload/intensity of exercise increases over time. This intensity will continue to increase until you can no longer perform the workload (i.e. you voluntarily terminate the test). During the test, heart rate will be recorded using a heart rate monitor, oxygen consumption and carbon dioxide production will be measured using a gas analysis system where you breathe continuously into a mouth piece and expiratory gases are collected for analysis, and ratings of perceived exertion will be recorded.
- Total testing on this day will take approximately 1 hour of your of your time.

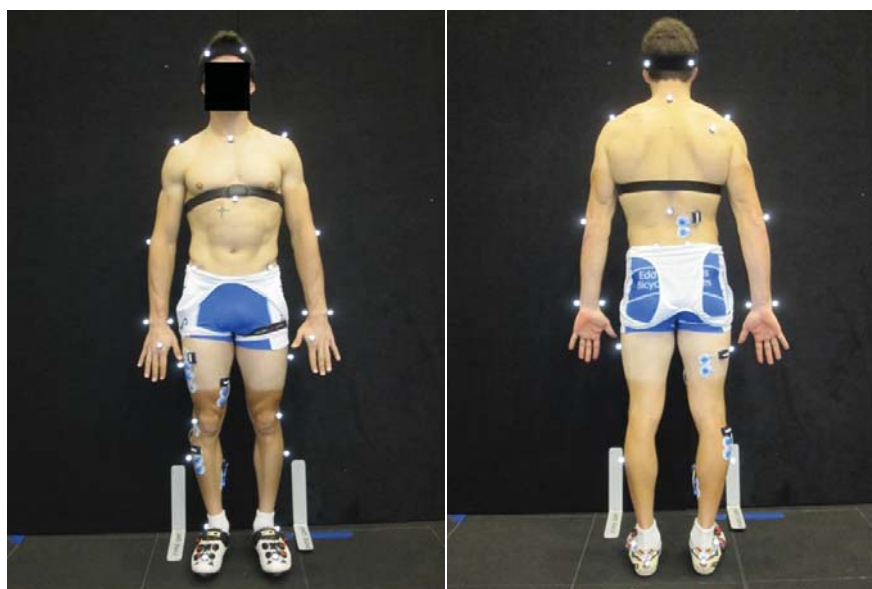
#### Day 2:

- Body mass and 24-h diet history will be recorded.
- Multidimensional Fatigue Inventory psychometric questionnaire, which provides a measure of how ‘fatigued’ you are feeling before the test, will be completed.
- A minimum of a 5-minute warm-up on a stationary bike, followed by three 3-second maximal sprints with a self-paced recovery between sprints, will be completed.
- Time to exhaustion (TTE) test. This test will involve cycling on a stationary bike at your constant, individualised power output (90% of the power output or workload you achieve at your maximal oxygen consumption level) until (1) you can no longer exercise (i.e. you voluntarily terminate the test), or (2) your cadence dropped below 5% of the target cadence (average cadence from the progressive exercise test) for longer than 3 s. HR and ratings of perceived exertion will be measured the same as the progressive exercise test.
- Muscle activity of six lower leg muscles and one buttocks muscle will be recorded using surface electrodes. These are gel-like pads which are stuck onto the muscle and are used to record the level of muscle activation (see Figure 1).
- Cycling technique will be recorded using 3D motion analysis. Reflective surface markers will be stuck on various landmarks of the body over the skin and will be recorded by ten infra-red cameras. This will be used to record changes in joint angles and joint range of motion (see Figure 2).
- Familiarisation with feedback on all techniques (described below).
- Total testing on this day will take approximately 2-2.5 hours of your of your time.



**Figure 1. Measurement of muscle activity during cycling exercise**





**Figure 2. Measurement of technique during cycling exercise**

Day 3-5:

- Body mass and 24-h diet history will be recorded.
- Multidimensional Fatigue Inventory psychometric questionnaire will be completed.
- A minimum of a 5-minute warm-up on a stationary bike, followed by three 3-second maximal sprints with a self-paced recovery between sprints, will be completed.
- Same as TTE test on Day 2 except that during each test you will be provided with real-time (immediate), visual feedback on three different techniques, which will be different for each TTE test. Therefore, feedback will be given for each technique during separate TTE tests, on separate days and will be randomised for each participant.
  - Three techniques will be investigated including trunk lean; trunk sway and knee sway (see Figure 3).
  - For all TTE tests when provided with technique feedback you will be asked to stay within an individualised ‘target range’ for each technique that is based on how you cycle when in a ‘non-fatigued’ state. The kinematic feedback will be provided via a laser beam, which will track the movement of the joint segment(s) (trunk or hip) throughout the test. The laser beam(s) will be attached to your trunk or knee and will track onto a whiteboard placed in front of you. You will need to try as hard as possible to limit your movement of the specific technique to within the ‘target range’ throughout the test. The test will continue until (1) you can no longer exercise (i.e. you voluntarily terminate the test) and have remained within the ‘target range’, (2) your cadence dropped below 5% of the target cadence for longer than 3 s, or (3) you are stopped by the experimenter because you moved beyond the ‘target zone’ consistently for longer than 10 s (if this occurs you may be asked to return for another test session to repeat the test).
- Muscle activity, cycling technique, HR and ratings of perceived exertion will be recorded the same as Day 2.
- Total testing on this day will take approximately 1-1.5 hours of your of your time.



**Figure 3. Three techniques that feedback will be provided on during cycling exercise (Note. Photos taken during Study 1 of this PhD project)**

**1: Trunk lean**

**2: Trunk sway**

**3: Knee sway**

There are very few discomforts associated with these tests. As you are trained cyclists you will be accustomed to performing exercise at high intensities and this should not pose any more physical or psychological discomfort than that typically experienced during hard training sessions. You may find that breathing into the mouthpiece is a little inconvenient; however this will not be of any major discomfort.

You are volunteering to participate in this project and therefore can withdraw from this project without explanation at any time without prejudice and with no disadvantage to yourself. Any data collected before withdrawal from the project will be destroyed. If you feel any pain or discomfort during the testing sessions please notify the experimenter immediately.

### **Risk Considerations**

There is a low risk and possible discomfort involved in performing maximal exertion exercise. This type of exercise could in rare cases lead to disastrous events, including cardiac events leading to death, and could also lead to severe exhaustion and/or fainting, however this is unlikely. Numerous precautionary procedures are put in place to avoid and minimise the likelihood of these risks occurring, including medical screening

questionnaires, regular equipment checks, safe operating practices and physiological measures of your response to exercise/level of fatigue.

### **Benefits and Reimbursement**

By participating in this project you will receive numerous measures of your current physiological status, including your maximal oxygen consumption ( $\text{VO}_{2\text{ max}}$ ), maximal heart rate ( $\text{HR}_{\text{max}}$ ), peak power output, ventilatory thresholds and suggested training zones in relation to your heart rate and power output. You will also be provided with details about which technique could be beneficial for you to use under fatiguing conditions, if the results indicate this. You will also get the chance to observe interesting research techniques such as electromyography (muscle activity), and 3D motion analysis, and will be the first to use the feedback system designed for this research project. The results of the project will also be provided to all participants in a summary format and/or a copy of journal articles, once data analysis is complete.

### **Ethical Considerations**

This project will comply with the guidelines set out by the National Health and Research Council. Ethical approval of this project has been granted by the Human Research Ethics Committee of ECU. All data collected during the project will be formatted so that it is unidentifiable (coded) and kept on password protected computers (soft copies) and/or in a lockable cabinet at ECU Joondalup campus (hard copies). At completion of the project all data will be retained for 5 years stored in a lockable cabinet at ECU Joondalup campus. Data collected may be used in future approved research projects, if you give permission to do so. Data will only ever be accessed by the investigators of this research project including myself and my supervisors Assoc Prof. Anthony Blazeovich and Dr. Chris Abbiss. The results of this project will be published in journal articles and my thesis, and presented at conferences, but any data included will in no way be linked to any specific participant. Furthermore, the results of this project will be made available to all participants in a summary format and again will not be linked to any specific participant.

Should you have any questions, concerns, or would like further information regarding this project, or would like to participate please contact me. If you would like to speak to the research supervisors please contact Assoc Prof. Anthony Blazeovich (08) 6304 5472, or Dr. Chris Abbiss (08) 6304 5740. If you have any concerns or complaints about the project, and you wish to talk to an independent person please contact the ECU Research Ethics Officer on (08) 6304 2170.

Kind Regards,

**Amanda Overton BPhEd (Hons) (PhD Candidate)**

Faculty of Computing, Health and Science  
School of Exercise, Biomedical and Health Sciences  
Edith Cowan University  
100 Joondalup Drive, Joondalup WA 6027  
Email: [a.overton@ecu.edu.au](mailto:a.overton@ecu.edu.au)  
Phone (W): (08) 6304 5819  
Mobile: 0450 963 527

## APPENDIX G



### INFORMED CONSENT FORM

#### **Studies 1 and 2: Neuromuscular fatigue and kinematics during high-intensity, constant-load cycling exercise**

Thank you for deciding to participate in this research project. Please read this consent form carefully before signing.

As the participant, you are aware and agree to the following statements:

- I have been provided with a copy of the Information Letter explaining the research project.
- I have read and understood the information provided.
- I have been given the opportunity to ask questions and have had any questions answered to my satisfaction.
- I am aware that if I have any additional questions I can contact the research team.
- I understand that my participation in the research project will involve:
  - Recording of body mass, height, date of birth, training status and 24-h diet history
  - Multidimensional Fatigue Inventory psychometric questionnaire
  - A progressive exercise test to exhaustion
  - A time to exhaustion test (TTE) and 5 tests to 20, 40, 60, 80 or 100% of your first TTE test duration
  - Magnetic stimulation of the femoral nerve
  - Maximal voluntary contractions of the quadriceps
  - Recording of HR using a heart rate monitor
  - Recording of oxygen consumption and carbon dioxide production using a gas analysis system
  - Recording of a rating of perceived exertion
  - Recording of muscle activity using surface electrodes
  - Recording of technique using 3D motion analysis
- I understand that the information provided will be kept confidential, and that the identity of participants, including myself will not be disclosed without consent.
- I understand that the information provided will only be used for the purposes of this research project, and I understand how the information is to be used.
- I understand that I am free to withdraw from further participation at any time, without explanation or penalty.
- I freely agree to participate in the project.

\_\_\_\_\_  
Name of the Participant                      Signature of the Participant                      Date

\_\_\_\_\_  
Signature of the Investigator                      Date

### CONSENT FOR FUTURE RESEARCH PROJECTS

It is possible that the data collected from this research project may be used in the future in other research projects. Data collected will remain formatted so that it is unidentifiable (coded) and only myself and my supervisors will have access to this data. If necessary ethical approval will be sought for any other researchers involved in future research projects who warrant access to the data.

Please consider this request and choose which statement is appropriate for you:

- ☐ The data collected for the purposes of this research project may be used in further approved research projects provided my name and any other identifying information is removed.
- ☐ The data collected for the purposes of this research project may not be used in further approved research projects without my consent.
- ☐ The data collected may be used only for the purposes of this research project.

\_\_\_\_\_  
Name of the Participant                      Signature of the Participant                      Date

\_\_\_\_\_  
Signature of the Investigator                      Date

#### **Amanda Overton BPhEd (Hons) (PhD Candidate)**

Faculty of Computing, Health and Science  
School of Exercise, Biomedical and Health Sciences  
Edith Cowan University  
100 Joondalup Drive, Joondalup WA 6027  
Email: [a.overton@ecu.edu.au](mailto:a.overton@ecu.edu.au)  
Phone: (08) 6304 5097  
Mobile: 04 5096 3527

#### **Research Team**

Assoc Prof Anthony Blazeovich  
Faculty of Computing, Health and Science  
Email: [a.blazeovich@ecu.edu.au](mailto:a.blazeovich@ecu.edu.au)  
Phone: (08) 6304 5472  
Mobile: 04 338 09339

Dr. Chris Abbiss  
Faculty of Computing, Health and Science  
Email: [c.abbiss@ecu.edu.au](mailto:c.abbiss@ecu.edu.au)  
Phone: (08) 6304 5740  
Mobile: 04 0429 9331

## APPENDIX H



### INFORMED CONSENT FORM

#### **Study 3: Effects of real-time, visual kinematic feedback on time to exhaustion during high-intensity, constant-load cycling exercise**

Thank you for deciding to participate in this research project. Please read this consent form carefully before signing.

As the participant, you are aware of and agree to the following statements:

- I have been provided with a copy of the Information Letter explaining the research project.
- I have read and understood the information provided.
- I have been given the opportunity to ask questions and have had any questions answered to my satisfaction.
- I am aware that if I have any additional questions I can contact the research team.
- I understand that my participation in the research project will involve:
  - Recording of body mass, height, date of birth, training status and 24-h diet history
  - Multidimensional Fatigue Inventory psychometric questionnaire
  - A progressive exercise test to exhaustion (Day 1)
  - A time to exhaustion test (TTE) (Day 2)
  - Multiple time to exhaustion tests performed with technique feedback (Days 3-5) (with the possibility that you may have to return on a separate day to complete another test if not performed to test protocol correctly).
  - Recording of HR using a heart rate monitor
  - Recording of oxygen consumption and carbon dioxide production using a gas analysis system
  - Recording of ratings of perceived exertion
  - Recording of muscle activity using surface electrodes
  - Recording of technique using 3D motion analysis
- I understand that the information provided will be kept confidential, and that the identity of participants, including myself will not be disclosed without consent.
- I understand that the information provided will only be used for the purposes of this research project, and I understand how the information is to be used.
- I understand that I am free to withdraw from further participation at any time, without explanation or penalty.
- I freely agree to participate in the project.



\_\_\_\_\_  
Name of the Participant                      Signature of the Participant                      Date

\_\_\_\_\_  
Signature of the Investigator                      Date

### **CONSENT FOR CONTACT TO PARTICIPATE IN FUTURE RESEARCH PROJECTS**

Please indicate below whether you would like to be contacted about future research projects. All research projects you will be contacted for will be approved by the Human Research Ethics Committee of ECU.

Please consider this request and choose which statement is appropriate for you:

- ☐ I would like to be contacted about future research projects at Edith Cowan University within the School of Exercise, Biomedical and Health Sciences.
- ☐ I only wish to be contacted about future research studies involved in this project (Neuromuscular Fatigue and Kinematics during Constant Workload High-Intensity Endurance Exercise).
- ☐ I do not wish to be contacted about future research projects.

\_\_\_\_\_  
Name of the Participant                      Signature of the Participant                      Date

\_\_\_\_\_  
Signature of the Investigator                      Date

### **CONSENT FOR FUTURE RESEARCH PROJECTS**

It is possible that the data collected from this research project may be used in the future in other research projects. Data collected will remain formatted so that it is unidentifiable (coded) and only myself and my supervisors will have access to this data. If necessary ethical approval will be sought for any other researchers involved in future research projects who warrant access to the data.

Please consider this request and choose which statement is appropriate for you:

- ☐ The data collected for the purposes of this research project may be used in further approved research projects provided my name and any other identifying information is removed.
- ☐ The data collected for the purposes of this research project may not be used in further approved research projects without my consent.
- ☐ The data collected may be used only for the purposes of this research project.

\_\_\_\_\_  
Name of the Participant                      Signature of the Participant                      Date

\_\_\_\_\_  
Signature of the Investigator                      Date

**Amanda Overton BPhEd (Hons) (PhD Candidate)**

Faculty of Computing, Health and Science  
School of Exercise, Biomedical and Health Sciences  
Edith Cowan University  
100 Joondalup Drive, Joondalup WA 6027  
Email: [a.overton@ecu.edu.au](mailto:a.overton@ecu.edu.au)  
Phone: (08) 6304 5097  
Mobile: 04 5096 3527

**Research Team**

Assoc Prof Anthony Blazeovich  
Faculty of Computing, Health and Science  
Email: [a.blazeovich@ecu.edu.au](mailto:a.blazeovich@ecu.edu.au)  
Phone: (08) 6304 5472  
Mobile: 04 338 09339

Dr. Chris Abbiss  
Faculty of Computing, Health and Science  
Email: [c.abbiss@ecu.edu.au](mailto:c.abbiss@ecu.edu.au)  
Phone: (08) 6304 5740  
Mobile: 04 0429 9331



## APPENDIX I



### PRE-EXERCISE MEDICAL SCREENING QUESTIONNAIRE

The following questionnaire is designed to establish a background of your medical history, and identify any injury and/ or illness that may influence your testing and performance. Please answer all questions as accurately as possible, and if you are unsure about anything please ask for clarification. All information provided is strictly confidential.

#### Personal Details

Participant ID: \_\_\_\_\_

Date of Birth (DD/MM/YYYY): \_\_\_\_\_

#### PART A

- |   |     |    | If YES, please<br>provide details |
|---|-----|----|-----------------------------------|
| 1. Are you a regular smoker or have you quit in the last 6 months?  | YES | NO | _____                             |
| 2. Did a close family member have heart disease or surgery, or stroke before the age of 60 yrs?   | YES | NO | _____                             |
| 3. Do you have, or have you ever been told you Have blood pressure above 140/90 mmHg, or do you currently take blood pressure medication? | YES | NO | _____                             |
| 4. Do you have, or have you ever been told you have a total cholesterol level above 5.20 mm/L (200 mg/dL)?                                | YES | NO | _____                             |
| 5. Is your BMI (weight/height <sup>2</sup> ) greater than 30 kg/m <sup>2</sup> ?  | YES | NO | _____                             |

#### PART B

- |   |     |    |       |
|---|-----|----|-------|
| 1. Have you ever had a serious asthma attack during exercise? | YES | NO | _____ |
| 2. Do you have asthma that requires medication?               | YES | NO | _____ |

- |  |     |    |       |
|--|-----|----|-------|
| 3. Have you had an epileptic seizure in the last 5 yrs?  | YES | NO | _____ |
| 4. Do you have any moderate or severe allergies?   | YES | NO | _____ |
| 5. Do you, or could you reasonably, have any infectious disease?                                       | YES | NO | _____ |
| 6. Do you, or could you reasonably, have an infection or disease that might be aggravated by exercise? | YES | NO | _____ |

### PART C

1. Are you currently taking any prescribed or non-prescribed medications?  
 YES                      NO

\_\_\_\_\_

2. Have you had, or do you currently have, any of the following?  
 If YES, please provide details

- |   |     |    |       |
|---|-----|----|-------|
| • Rheumatic fever   | YES | NO | _____ |
| • Heart abnormalities   | YES | NO | _____ |
| • Diabetes  | YES | NO | _____ |
| • Epilepsy  | YES | NO | _____ |
| • Recurring back pain that would make exercise problematic, or where exercise may aggravate pain?                         | YES | NO | _____ |
| • Recurring neck pain that would make exercise problematic, or where exercise may aggravate pain?                         | YES | NO | _____ |
| • Any neurological disorders that would make exercise problematic, or where exercise may aggravate the condition?         | YES | NO | _____ |
| • Any neuromuscular disorders that would make exercise problematic, or where exercise may aggravate the condition?        | YES | NO | _____ |
| • Recurring muscle or joint injuries that Would make exercise problematic, or where exercise may aggravate the condition? | YES | NO | _____ |

- A burning or cramping sensation in your legs when walking for short distances? YES NO \_\_\_\_\_
- Chest discomfort, unreasonable breathlessness, dizziness or fainting, or blackouts during exercise? YES NO \_\_\_\_\_

#### **PART D**

1. Have you had the flu in the last week? YES NO \_\_\_\_\_
2. Do you currently have an injury that might affect, or be affected by exercise? YES NO \_\_\_\_\_

#### **PART E**

1. Have you ever had significant periods of dizziness or disorientation after performing maximal exercise? YES NO \_\_\_\_\_
2. Do you have any injuries or medical conditions that you believe might be problematic for maximal exercise performance? YES NO \_\_\_\_\_
3. Are you a diabetic? YES NO \_\_\_\_\_
4. Have you ever been told by a medical practitioner or health care professional that you have a nerve disorder? YES NO \_\_\_\_\_
5. Do you have a heart pace maker? YES NO \_\_\_\_\_
6. Do you have any metallic implants? YES NO \_\_\_\_\_  
(e.g. bone pins)

\* Is there any other condition not previously mentioned that may affect your ability to participate in this study?

YES NO \_\_\_\_\_  
\_\_\_\_\_

\_\_\_\_\_  
Name of the Participant

\_\_\_\_\_  
Signature of the Participant

\_\_\_\_\_  
Date

\_\_\_\_\_  
Signature of the Investigator

\_\_\_\_\_  
Date

## APPENDIX J

### MULTIDIMENSIONAL FATIGUE INVENTORY

\*\*\*MFI-20\*\*\*

Participant ID: \_\_\_\_\_ Session: \_\_\_\_\_ Date: \_\_\_\_\_

#### Instructions:

By means of the following statements we would like to get an idea of how you have been feeling lately. There is for example the statement:

‘I FEEL RELAXED’

If you think this is entirely true, that you indeed have been feeling relaxed lately, please, place an X in the extreme left box like this:

Yes, that is true 

X				
---	--	--	--	--

 No, that is not true

The more you disagree with the statement, the more you can place an X in the direction of ‘No, that is not true’. Please, do not miss out a statement and place one X next to each statement.

- |   |  |  |  |  |  |  |
|---|--|--|--|--|--|--|
| 1. I feel fit   | Yes, that is <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td></td><td></td><td></td><td></td><td></td></tr></table> No, that is not |  |  |  |  |  |
|   |  |  |  |  |  |  |
| 2. Physically I feel only able to do a little                 | Yes, that is <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td></td><td></td><td></td><td></td><td></td></tr></table> No, that is not |  |  |  |  |  |
|   |  |  |  |  |  |  |
| 3. I feel very active   | Yes, that is <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td></td><td></td><td></td><td></td><td></td></tr></table> No, that is not |  |  |  |  |  |
|   |  |  |  |  |  |  |
| 4. I feel like doing all sorts of nice things                 | Yes, that is <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td></td><td></td><td></td><td></td><td></td></tr></table> No, that is not |  |  |  |  |  |
|   |  |  |  |  |  |  |
| 5. I feel tired   | Yes, that is <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td></td><td></td><td></td><td></td><td></td></tr></table> No, that is not |  |  |  |  |  |
|   |  |  |  |  |  |  |
| 6. I think I do a lot in a day                                | Yes, that is <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td></td><td></td><td></td><td></td><td></td></tr></table> No, that is not |  |  |  |  |  |
|   |  |  |  |  |  |  |
| 7. When I am doing something, I can keep<br>my thoughts on it | Yes, that is <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td></td><td></td><td></td><td></td><td></td></tr></table> No, that is not |  |  |  |  |  |
|   |  |  |  |  |  |  |
| 8. Physically I can take on a lot                             | Yes, that is <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td></td><td></td><td></td><td></td><td></td></tr></table> No, that is not |  |  |  |  |  |
|   |  |  |  |  |  |  |
| 9. I dread having to do things                                | Yes, that is <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td></td><td></td><td></td><td></td><td></td></tr></table> No, that is not |  |  |  |  |  |
|   |  |  |  |  |  |  |
| 10. I think I do very little in a day                         | Yes, that is <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td></td><td></td><td></td><td></td><td></td></tr></table> No, that is not |  |  |  |  |  |
|   |  |  |  |  |  |  |
| 11. I can concentrate well                                    | Yes, that is <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td></td><td></td><td></td><td></td><td></td></tr></table> No, that is not |  |  |  |  |  |
|   |  |  |  |  |  |  |
| 12. I am rested   | Yes, that is <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td></td><td></td><td></td><td></td><td></td></tr></table> No, that is not |  |  |  |  |  |
|   |  |  |  |  |  |  |

13. It takes a lot of effort to concentrate on things      Yes, that is ☐☐☐☐☐☐ No, that is not
14. Physically I feel I am in a bad condition      Yes, that is ☐☐☐☐☐☐ No, that is not
15. I have a lot of pains      Yes, that is ☐☐☐☐☐☐ No, that is not
16. I tire easily      Yes, that is ☐☐☐☐☐☐ No, that is not
17. I get little done      Yes, that is ☐☐☐☐☐☐ No, that is not
18. I don't feel like doing anything      Yes, that is ☐☐☐☐☐☐ No, that is not
19. My thoughts easily wonder      Yes, that is ☐☐☐☐☐☐ No, that is not
20. Physically I feel I am in an excellent  
condition      Yes, that is ☐☐☐☐☐☐ No, that is not

## APPENDIX K

## 24-H DIET HISTORY RECORD SHEET

Participant ID: \_\_\_\_\_ Session: \_\_\_\_\_ Date: \_\_\_\_\_ / \_\_\_\_\_ / \_\_\_\_\_

*Please record all food and beverages consumed over the past 24-h period.*

[illegible]

## APPENDIX L



### FINAL CHECKLIST FOR PARTICIPANT

- |   |     |    |
|---|-----|----|
| 1. Are you aware that if you feel uncomfortable with any testing procedure you should tell the researcher immediately, and that <b>YOU CAN STOP</b> your participation at any time? | YES | NO |
| 2. Are you aware although very rare, maximal exercise can result in fainting, severe exhaustion or cardiac events leading to death?   | YES | NO |
| 3. Are you aware that the fatigue caused by the exercise can impair your ability to perform tasks such as driving for a short while after the cessation of exercise?                | YES | NO |
| 4. Have you fasted for longer than 6 hours?   | YES | NO |
| 5. Are you aware the electrical stimulation will be applied to your nerve?  | YES | NO |
| 6. Are you aware that, depending on the intensity if the stimulation, the procedure may cause variable levels of discomfort or pain?  | YES | NO |

\_\_\_\_\_  
Name of the Participant

\_\_\_\_\_  
Signature of the Participant

\_\_\_\_\_  
Date

\_\_\_\_\_  
Signature of the Investigator

\_\_\_\_\_  
Date

## APPENDIX M

### Task Expectations

**Do you think that maintaining or holding ‘good cycling technique’, or changing cycling technique during cycling exercise is more beneficial to performance? In other words, in which scenario do you think you would be able to cycle longer (improved performance)?**

### Before you answer this you should consider the following points:

Traditionally, it is often encouraged by health and safety officers, practitioners and sports coaches to maintain ‘good technique’ in order to maintain movement efficiency and avoid injury, and therefore to improve performance.

However, on the other hand, scientific research has shown that the human neuromuscular system (your central nervous system control of all muscle groups) is programmed in a way to deal with fatigue effects by allowing changes in movement patterns, which are beneficial for delaying further fatigue development, avoiding injury and maintaining task performance. For example, the neuromuscular system uses “compensatory strategies” against the development of fatigue and therefore allow for continued exercise performance. These include:

- Changes to the way in which your muscle “fires” to prolong muscular contraction
- The use of additional muscles which were previously inactive prior to fatigue development and thus allowing the other muscles that have become fatigued to recover
- Co-activation between flexor and extensor muscles, which increases joint rigidity and has been suggested to simplify movement control.

Subject ID: \_\_\_\_\_

### **Do you think...?**

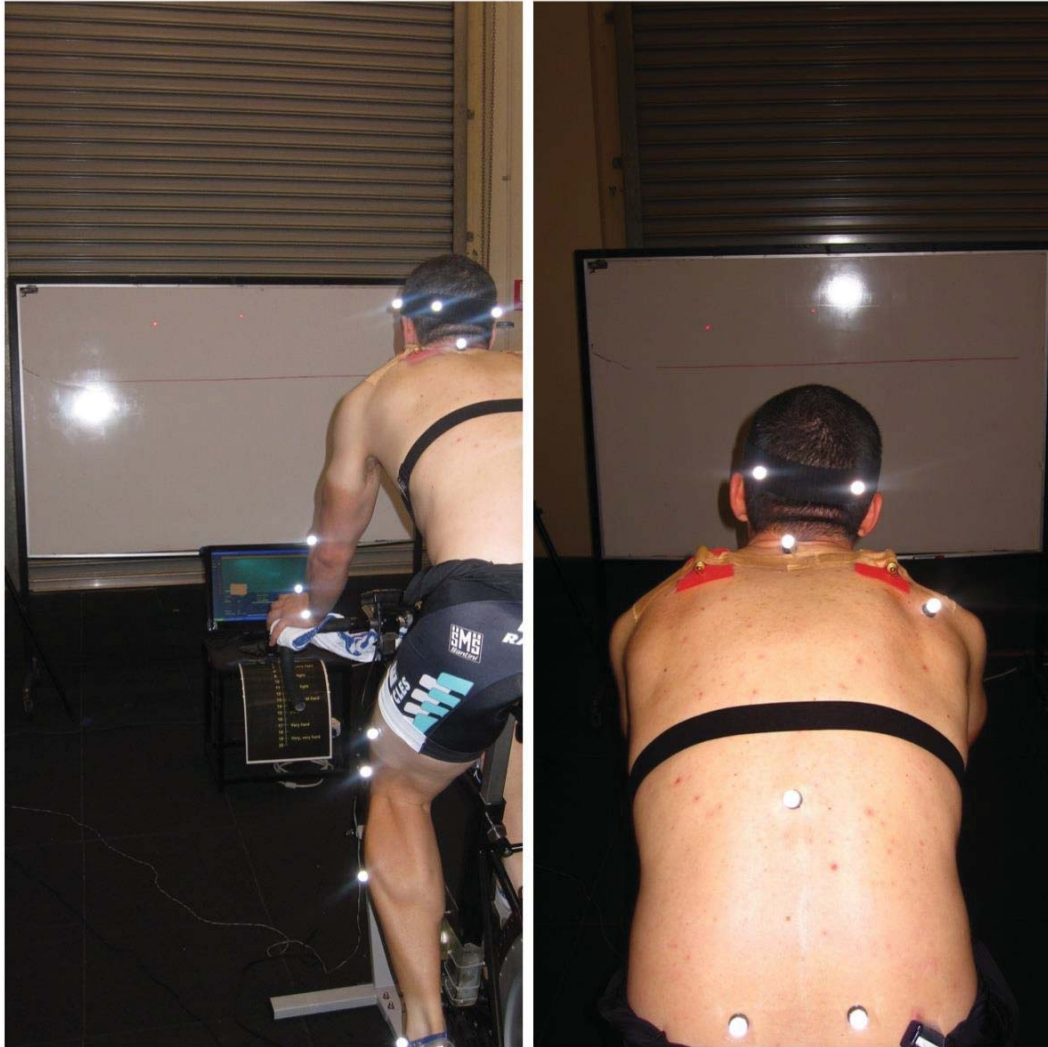
Holding ‘good cycling technique’ is more beneficial ☐ OR

Changing cycling technique is more beneficial ☐

WHY: \_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_



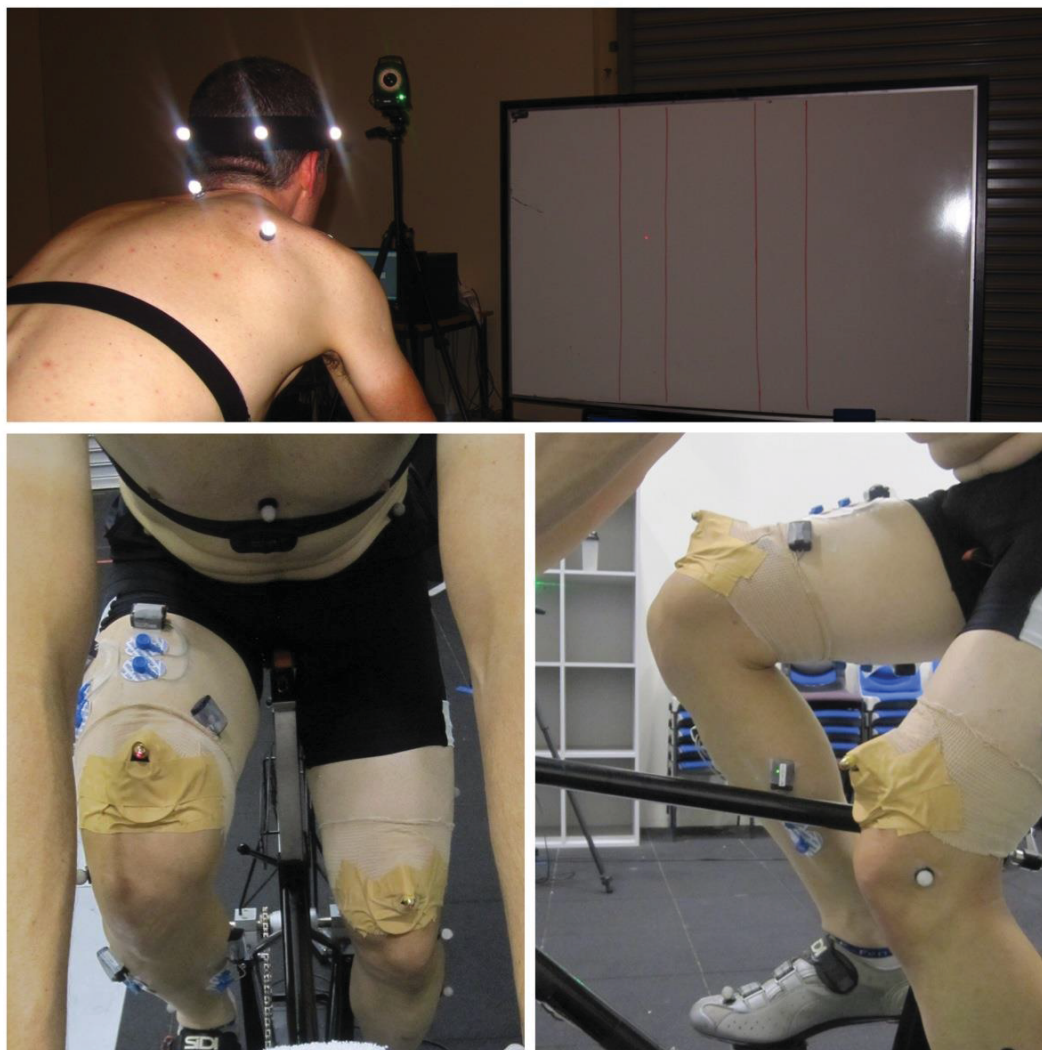
## APPENDIX N



(A) Cyclist using the real-time, visual kinematic feedback during the constant-load, cycling test for trunk flexion (target zone was above the horizontal line).



(B) Cyclist using the real-time, visual kinematic feedback during the constant-load, cycling test for trunk medio-lateral sway (target zone was in between the two vertical lines).



(C) Cyclist using the real-time, visual kinematic feedback during the constant-load, cycling test for hip abduction/adduction (target zones were in between the two vertical lines for both the right and left legs).



## INTRODUCTION

Neuromuscular fatigue can develop at both central and peripheral levels, and has mostly been assessed at the onset and termination of exercise. However, this does not capture the time course, or relative contribution, of fatigue mechanisms throughout an exercise bout.

Changes in the timing and magnitude of muscle activation, and subsequent changes in kinematics, are used as compensatory strategies to maintain power output during fatigue [1].

Changes in neuromuscular fatigue markers has not been investigated simultaneously with muscle activation and kinematics during high-intensity, constant-load cycling exercise.

## METHODS

Nine well-trained male cyclists ( $\text{VO}_{2\text{max}}$ ,  $55.9 \pm 7.3 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) performed seven test sessions: (1) incremental cycling test to exhaustion, (2) constant-load cycling time to exhaustion (TTE;  $5:49 \pm 0:51 \text{ min}$ ), (3-6) constant-load cycling tests to 20, 40, 60 and 80% ( $T_{20-T_{80}}$ ) of the time taken to complete the first TTE test ( $T_{100}$ ) in a random order, and (7) a TTE test. Constant-load cycling tests were performed at 90% of the power output that elicited  $\text{VO}_{2\text{max}}$  (cadence,  $89 \pm 10 \text{ rpm}$ ). EMG activity of seven lower limb muscles and 3D joint kinematics were recorded throughout the constant-load cycling tests ( $T_0-T_{100}$ ). Mechanical and EMG responses of the quadriceps to voluntary and electrically stimulated (femoral nerve) contractions were made before and after exercise. Voluntary muscle activation (%VA) was using the ITT.

## RESULTS

Twitch contractile properties changed significantly throughout the exercise bout (Fig. 1). No change in M-wave amplitude ( $M_{\text{max}}$ ) and duration ( $M_{\text{dur}}$ ), nor twitch one-half relaxation time was observed. Peak torque and the rate of torque development measured during voluntary knee extension did not change. Trends towards increases in vastus lateralis EMG amplitude and the EMG: $M_{\text{max}}$  from  $T_{80}-T_{100}$  were not significant. No significant changes were observed in %VA, peak EMG amplitudes, rate of EMG rise or EMG mean frequency ( $f_{\text{mean}}$ ). During cycling, EMG onset and offset times and duration, and EMG  $f_{\text{mean}}$  did not change, but peak vastus medialis and gluteus maximus (GMax) EMG amplitude, GMax mean EMG amplitude and GMax EMG impulse significantly increased (Fig. 2). Trunk flexion significantly increased from  $T_{60}$ , and trunk lateral flexion, hip abduction/adduction (Fig. 3) and knee valgus/varus increased from  $T_{80}$ .

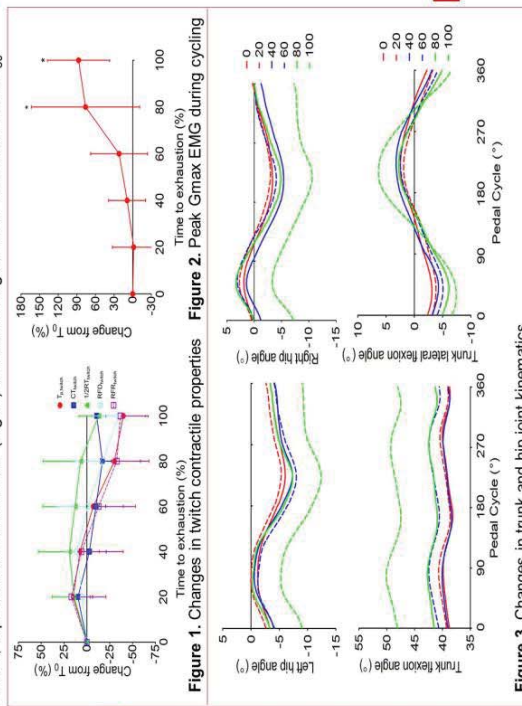


Figure 1. Changes in twitch contractile properties

Figure 2. Peak Gmax EMG during cycling

Figure 3. Changes in trunk and hip joint kinematics

## DISCUSSION

Twitch contractile properties were significantly impaired from  $T_{60}$  onwards despite a lack of change in  $M_{\text{max}}$  and  $M_{\text{dur}}$ . Thus peripheral fatigue significantly influenced exercise capacity but was temporally dissociated from the kinematic changes.

VL EMG amplitude and the EMG: $M_{\text{max}}$  ratio were increased in the final stages of the exercise bout ( $T_{80}-T_{100}$ ), indicating an increase in central motor drive. We speculate that this may act as a compensatory mechanism to overcome peripheral fatigue during intensive cycling exercise.

There were only moderate changes in the patterns of muscle activity measured during cycling. An increase in lower limb muscle (VM and Gmax) activity was observed, possibly as an attempt to employ motor units that were not previously maximally active to delay exhaustion.

There were significant sagittal plane trunk kinematic changes from  $T_{60}$  and trunk, hip and knee joint coronal plane changes from  $T_{80}$ , indicating that changes in cycling kinematics were secondary to peripheral fatigue development and were possibly associated with the facilitation of central drive.

## REFERENCES

- [1] Gandevia, S.C., *Spinal and Supraspinal Factors in Human Muscle Fatigue* Physiological reviews, 2001. **81**(4): p. 1725-1789.