

18

The Regulation of Exercise Performance by a Complex Anticipatory System

by
Ross Tucker

This thesis is presented for the degree of
DOCTOR OF PHILOSOPHY
in the Department of Human Biology,
University of Cape Town
Cape Town, SOUTH AFRICA
AUGUST 2006

MRC/UCT Research Unit for Exercise Science and Sports Medicine
Sports Science Institute of South Africa
Boundary Road
Newlands 7700
Cape Town, SOUTH AFRICA

The copyright of this thesis vests in the author. No quotation from it or information derived from it is to be published without full acknowledgement of the source. The thesis is to be used for private study or non-commercial research purposes only.

Published by the University of Cape Town (UCT) in terms of the non-exclusive license granted to UCT by the author.

Table of Contents

Acknowledgements	i
Declaration	iii
List of Publications	iv
Conference proceedings.....	vi
<i>International</i>	vi
<i>Local</i>	vi
Abstract.....	viii
Summary of studies in this thesis.....	ix
List of abbreviations used in this thesis	xiii
Chapter 1 Review of the Literature Pacing strategy during self-	
paced exercise performance.....	1
Introduction	2
Studies of pacing strategy	4
<i>Observational studies of pacing strategy</i>	6
<i>Experimental manipulations of pacing strategy</i>	8
<i>Computer simulations of pacing strategy</i>	13
<i>Method differences between studies</i>	13
<i>Conclusion</i>	16
Physiological basis for the observed optimal pacing strategy during long	
duration exercise bouts	18
<i>Exercise in the heat</i>	19
<i>The critical internal temperature hypothesis</i>	19
<i>Pacing strategies during self-paced exercise performance in the heat</i>	24
<i>Exercise with different inspired oxygen content</i>	27
<i>Energy substrate availability</i>	30
<i>Provision of incorrect distance or duration feedback</i>	33
<i>Conclusion</i>	38
Physiological basis for positive pacing strategy during short duration exercise	
.....	39

<i>Evidence for declining muscle force production during short duration self-paced exercise</i>	<i>40</i>
<i>Evidence for pacing strategies during short duration, high intensity exercise.....</i>	<i>41</i>
<i>Conclusion.....</i>	<i>47</i>
The proposed role for the RPE in the regulation of exercise	47
Summary	51
Research questions	53
Chapter 2 Impaired exercise performance in the heat is associated with an anticipatory reduction in skeletal muscle activation	57
Introduction	58
Methods	62
Results	70
Discussion	79
Chapter 3 Hyperoxia improves 20 km cycling time trial performance by increasing muscle activation levels while perceived exertion stays the same	90
Introduction	91
Methods	95
Results	102
Discussion	111
Chapter 4 The rate of heat storage mediates an anticipatory reduction in exercise intensity during cycling at a fixed rating of perceived exertion	121
Introduction	122
Methods	125
Results	133
Discussion	143
Chapter 5 Skeletal muscle electrical activity is not maximal at maximal volitional effort/exhaustion	153

Introduction	154
Methods	157
Results	163
Discussion	167
Chapter 6 An analysis of pacing strategies during men's world record performances in track athletics	173
Introduction	174
Methods	176
Results	177
Discussion	183
Chapter 7 Summary Conclusion including a proposed anticipatory- feedback model for the regulation of exercise incorporating the rating of perceived exertion	196
Exercise at a fixed workrate to fatigue.....	200
Self-paced exercise time-trials.....	202
Testing the model using the RPE clamp protocol.....	208
Further applications of the anticipatory-feedback model.....	211
<i>The endspurt phenomenon</i>	<i>211</i>
<i>The effects of central nervous system drugs on performance.....</i>	<i>212</i>
<i>Application to studies where misinformation is provided.....</i>	<i>214</i>
<i>Actual duration is shorter than the expected/informed duration.....</i>	<i>217</i>
<i>Actual duration exceeds the expected/informed duration.....</i>	<i>218</i>
<i>No duration information is provided.....</i>	<i>220</i>
<i>Limitations of the studies in the present thesis</i>	<i>222</i>
<i>Suggestions for future research</i>	<i>224</i>
Conclusion.....	227
References	228

Acknowledgements

The journey to the completion of this thesis began when I was still a high school student who made up my mind to pursue a career in Exercise Science. Since that day, so many people have supported, steered and guided me that it would be impossible to name all of them. However, a few deserve special mention, for their direct involvement and support in the last 12 years. In no particular order:

Tim Noakes, for all the obvious reasons - supervisor, director of ESSM, creative inspiration. But most of all, Tim wrote *Lore of Running*, which was the very first book I ever read on the subject of sports science. I was inspired to come to UCT by that book. Tim was with me at the start of this journey and has seen it to completion, as my very first and biggest scientific inspiration.

Mike Lambert. I met Mike as a 16-year old a day visit, when I announced my intention to come to ESSM in 6 years time! Mike was the same then as he is now, reassuring and inspiring. Anyone else may have put me off before I began, but Mike gave me confidence and assurance that lasted for the next 6 years, and he was always going to be my supervisor. Thank you for your patience and guidance.

The entire staff at ESSM. Not just for teaching me science, but for developing me as a person. Everyone has played a role, but special thanks go to Vicki and Zig. Vicki is impossibly positive, always enthusiastic and I hope to discover the secret to that one day! You are an inspiration, and were always a good morale booster when needed it. Zig is a visionary, who, as I progressed in this thesis, earned more and more respect because there were many moments where I felt that I had come across a novel theory or idea, only to find that Zig had said the same two years earlier! I was always one step behind, I hope to catch up one day!

My office mates, Jonathan, Tami and George. The best thing that happened to me in my University career was being thrown into an office with these three in my first year as a Postgraduate student. I have learned so much, and it's an honour to have shared ideas and time with you. Tami, you are a huge inspiration for your personality and

spirit, and you have become the scientist I respect most. George, you have a wisdom and approach to life that has changed mine, and Jonathan, I've not met anyone else so capable and so willing to help and get involved with anything. Three great examples, three great team mates in the dream team!

My Honours year classmates – Yumna, Lize, Tina, Liesl, Jamie, Jacques, Liane and Hitesh. We had a great year, the definitive year of my life up to now and you were an integral part of it.

All the other honours students who I have had the privilege of working with in the last four years. Most of what I have learned has been through your experiences, and I owe you a debt of gratitude for that.

Sebastian, Rob Lamberts, Lotte and Gunnaar. I hope to be there when you graduate. Thank you for everything.

Karen Sharwood, Sacha West, Yolande Harley, Dale Rae and Mark Kirkman were the senior students when I began. It's funny to think that I am now someone's 'senior student', but I hope that I can make the impact you made on me. A special thanks to Sacha, the best listener in the world, I hope that we graduate together.

Ant Saunders. Unfortunately, Ant left Exercise Science for the 'greener' pastures of accounting. Made it look routine, just like exercise science, but you were the catalyst for many of my landmark moments and 'epiphanies'.

Erin Rae. You probably don't even realise it, but you are one of the biggest inspirations on my life, I learned that much from you. If I had a choice of people to help me in a

time of crisis, you would be the first name on the list. During the good times, you would be on that list too. Unique.

People who have picked me up in the difficult times. In 8 years of university, there are invariably moments of doubt, anxiety or frustration, usually not work related. At those times, I have been amazed at how people step in and a word of encouragement here, some advice there, and the journey continued. Many people have carried me, often without knowing, but thanks to Wayne Viljoen, Julia Goedecke, Liane Berretta, Yumna, Carrie Blackburn and Malcolm Collins, for advice that made a difference.

My classmates in Sports Management Postgrad course 2006. It's been a difficult year, and I often wonder whether I would have been better off waiting a year to do the course. But that would have meant missing out on this group, and you are one of the two reasons I'm glad I didn't.

Rob van der Valk and Nick Randall – the other reason I am glad I didn't wait. You have provided me with a learning curve so steep, I have lost sight of where I left from! I have never been so stimulated and it's been a rare privilege to learn from you guys.

To my closest support base, Lauren, Bianca and Maria. Quite what I did to earn your loyalty I don't know, but if I could control time, I would run the last two years over and over.

To Tabea, who has supported me with loyalty that I don't deserve. Two years ago,

Sorry to all those who I have forgotten – it is not intentional. I hope that I have made it known to you that your part in this thesis and in my life cannot be justified or explained in the acknowledgements section of an ultimately limited thesis. Thank you, I hope I repay you in kind.

I could not even imagine myself as I am today, and that's because of you.

My sister, Heather, who has been with me throughout school and university. It's impossible to know how different things might have been without a (twin) sister, but to have had a tolerant and supportive companion the whole way is a real blessing.

My Mom and Dad. It's not even possible to measure the contribution they have made to my life, let alone this work. My thinking, values, attitudes and perceptions have been shaped over 25 years and I can't even begin to describe how grateful I am. Had it not been for my father's participation in the Two Oceans Marathon, my interest in running would not have been so great, and both my parents taught me the value of logic and thought.

Ian Harries, the greatest athletics coach around. Ian picked me up while I was still at school and made his library and his mind available to me, allowing me to read and research topics such as training, altitude and exercise performance. We will one day write that book together! Ian also taught me the value of thinking practically and most of all, challenged my thinking in such a way that it threw up Chapter 6 of this thesis, and may one day be responsible for starting off an entire career. A visionary.

To my grandmother, Joyce. She picked me up from primary school on my very first day as a five-year old, and continued to influence my life for the next twenty years. This thesis is for you.

Declaration

The Regulation of Exercise Performance by a Complex Anticipatory System

I _____ (full name) hereby:

- (a) grant the University of Cape Town free licence to reproduce the above thesis in whole or in part, for the purpose of research;

- (b) declare that:

The above thesis is my own unaided work, both in concept and execution, and that apart from the normal guidance from my supervisor.

Neither the substance nor any part of the above thesis has been submitted in the past, or is being, or is to be submitted for a degree at this University or at any other university.

I am now presenting the thesis for examination for the degree of PhD.

SIGNED:

Signed by candidate

DATE: 29/11/2006

List of Publications

Albertus Y, Tucker R, St Clair Gibson A, Lambert EV, Hampson DB & Noakes TD (2005). Effect of distance feedback on pacing strategy and perceived exertion during cycling. *Med Sci Sports Exerc* 37, 461-468.

Baden DA, McLean TL, Tucker R, Noakes TD & St Clair Gibson, A (2005). Effect of anticipation during unknown or unexpected exercise duration on rating of perceived exertion, affect, and physiological function. *Br J Sports Med* 39, 742-746.

The findings of this original research study are described in detail in Chapter 1 and Chapter 7 of the present thesis

Noakes TD & Tucker R (2004). Inverse relationship between VO_2max and economy in world-class cyclists. *Med Sci Sports Exerc* 36, 1083-1084.

Saunders AG, Dugas JP, Tucker R, Lambert MI & Noakes TD (2005). The effects of different air velocities on heat storage and body temperature in humans cycling in a hot, humid environment. *Acta Physiol Scand* 183, 241-255.

St Clair Gibson A, Lambert EV, Rauch LHG, Tucker R, Baden DA, Foster C & Noakes TD (2006). The role of information processing between the brain and peripheral physiological systems in pacing and perception of effort. *Sports Med* 36, 705-722.

Tucker R, Rauch L, Harley YX & Noakes TD (2004). Impaired exercise performance in the heat is associated with an anticipatory reduction in skeletal muscle recruitment. *Pflugers Arch* 448, 422-430

The findings of this original research study form the basis for Chapter 2 of the present thesis

Tucker R, Marle T, Lambert EV & Noakes TD (2006). The rate of heat storage mediates an anticipatory reduction in exercise intensity during cycling at a fixed rating of perceived exertion. *J Physiol* 574, 905-915

The findings of this original research study form the basis for Chapter 4 of the present thesis

Tucker R, Lambert MI & Noakes TD (2006). An analysis of pacing strategies during men's world record performances in track athletics. *Int J Sports Physiol Performance* 1, 297-309.

The findings of this original research study form the basis for Chapter 6 of the present thesis

Tucker R, Bester A, Lambert EV, Noakes TD, Vaughan CL & St Clair Gibson A (2006). Non-random fluctuations in power output during self-paced exercise. *Br J Sports Med* (In Press).

Conference proceedings

International

52nd Meeting of the American College of Sports Medicine, Nashville, Tennessee, USA, 1 – 5 June 2005.

Ross Tucker, Bester A, Lambert EV, Noakes TD, Vaughan CL, St Clair Gibson A.
Non-random fluctuations in power output during self-paced exercise

9th Annual Meeting of the European College of Sports Sciences (ECSS), Clermont-Ferrand, France, 4 – 7 July, 2004.

Ross Tucker, Laurie Rauch, Yolande X Harley, Timothy D Noakes. Impaired exercise performance in the heat is associated with an anticipatory reduction in skeletal muscle recruitment.

Ross Tucker, Anthony G. Saunders, Jonathan P. Dugas, Mike I. Lambert, Timothy D. Noakes. The effects of different air velocities on heat storage and body temperature in humans cycling in a hot, humid environment

Local

SSISA/UCT Expedition report back evening: The world's northernmost long distance swim, 3 October 2005

Ross Tucker. The physiology of cold-water immersion.

UCT Faculty of Health Sciences School of Biomedical Sciences Research Day 2004, 1 September, 2004

Ross Tucker, Laurie Rauch, Yolande X Harley, Timothy D Noakes. Impaired exercise performance in the heat is associated with an anticipatory reduction in skeletal muscle recruitment.

Ross Tucker, Erin Rae, Laruie Rauch, Andrew Bosch, Alan Thomas, Madeleine Murphy, Timothy Noakes. Hyperoxia alters pacing strategy and skeletal muscle recruitment during a 20 km cycling time trial

Sport Sciences Conference, in Association with the 3rd Annual Discovery Vitality Fitness Convention, Wits Education Campus, Johannesburg, South Africa, 7 – 9 October, 2004

Ross Tucker, Denise Baden, Tracy-Lee Mclean, Alan St Clair Gibson, Timothy D. Noakes. Anticipated exercise duration influences psychological variables and running economy.

Abstract

The Regulation of Exercise Performance by a Complex Anticipatory System

Ross Tucker

9 August 2006

The present thesis examined the hypothesis that self-paced exercise performance and pacing strategies are regulated by a complex intelligent system in advance of a failure to maintain homeostasis in one or more physiological systems.

In the first study, ten trained cyclists performed 20 km cycling time-trials in hot (35°C) and cool (15°C) conditions. The power output was reduced in the heat despite core temperatures that were sub-maximal and not different from those measured in the cool condition. Significantly, the reduction in power output was associated with a lower IEMG activity in the active muscle, suggesting that the brain recruited less muscle even at sub-maximal body temperatures. Thus, self-paced exercise in the heat was regulated in advance of thermoregulatory failure.

This model was then applied to conditions where the oxygen content of the air was elevated (hyperoxia). Eleven subjects performed 20 km time-trials, and it was found that a higher power output was maintained throughout hyperoxic (F_{iO_2} 0.4) trials than in normoxia (F_{iO_2} 0.21), and that the IEMG activity was elevated in hyperoxia. The subjective rating of perceived exertion (RPE), measured using the Borg scale, was similar in both this and the first study, despite differences in power output. It was suggested that the RPE may play a mediatory role.

This hypothesis was evaluated by developing a novel RPE clamp model, in which cyclists rode at a fixed RPE of 16 on the Borg scale. The rate of heat storage was similar when subjects cycled this way in temperatures of 15°C, 25°C and 35°C. It was concluded that the RPE was sensitive to the rate of heat storage and was a key mediator in a proposed anticipatory-feedback model.

In the fourth study, I evaluated the hypothesis that the activation of muscle was not maximal, even at maximal volitional effort. Cyclists performed incremental cycling trials to exhaustion, followed by intermittent sprint bouts at power outputs higher than those achieved in the incremental bouts. The iEMG activity increased linearly at these supramaximal power outputs, suggesting that volitional fatigue had occurred at substantially sub-maximal levels of muscle activity.

Finally, I analysed the pacing strategies adopted during world record performances in the 800 m, 5 000 m and 10 000 m track events. It was found that in the shorter 800 m event, no increase in running speed occurred in the second lap, whereas in the longer distance events, an endspurt was observed. This has implications for understanding how pacing strategies may be regulated depending on the duration or length of exercise.

Summary of studies in this thesis

Study 1

The aim of the first study of this thesis was to examine whether centrally regulated skeletal muscle recruitment was altered during dynamic exercise in hot (35°C) compared to cool (15°C) environments. Ten male subjects performed two self-paced 20-km cycling time-trials, one at 35°C (HOT condition) and one at 15°C (COOL condition). Power output and integrated electromyographic activity (iEMG) of the quadriceps muscle began to decrease early in the HOT trial, when core temperatures, heart rates and ratings of perceived exertion (RPE) were not different from those measured in COOL. iEMG was significantly lower in HOT than in COOL at 10 and 20 km, while power output was significantly reduced from 80% to 100% of the trial duration in the HOT compared to COOL condition. Thus, reduced power output and iEMG activity during self-paced exercise in the heat occurs before there is any abnormal increase in rectal temperature, heart rate or perception of effort. This adaptation appears to form part of an anticipatory response which adjusts muscle recruitment and power output to reduce heat production, thereby ensuring that thermal homeostasis is maintained during exercise in the heat.

Study 2

Increasing inspiratory oxygen tension improves exercise performance. Study 2 of this thesis tested the hypothesis that this is due to changes in muscle activation levels. Eleven male subjects performed two 20-km cycling time-trials, one in hyperoxia (HI, F_{iO_2} 40%) and one in normoxia (NORM, F_{iO_2} 21%). Power output, heart rate, blood lactate, integrated vastus lateralis EMG activity (iEMG) and ratings of perceived exertion (RPE) were measured every 2 km. Performance was improved by 5% in HI compared to NORM ($P < 0.01$). Power output was maintained throughout the trial in HI, but decreased progressively in NORM from 4 km onwards ($P < 0.01$). In both conditions, power output increased significantly

in the last kilometre ($P < 0.0001$). IEMG activity was proportional to power output and was significantly greater in HI than in NORM. IEMG activity increased significantly in the final kilometer of both trials ($P < 0.001$). Heart rate, plasma lactate concentration and rating of perceived exertion (RPE) were not different between conditions. In conclusion, improved exercise performance in hyperoxia is due to higher power output from increased muscle activation, resulting in altered pacing strategies between conditions. Such adaptation of pacing suggests the existence of an integrative property of the central nervous system which controls and limits muscle activation level commensurate with prevailing exercise conditions, anticipated remaining exercise time and development of fatigue.

Study 3

Study 3 aimed to examine the regulation of exercise intensity in hot environments when exercise is performed at a predetermined, fixed subjective rating of perceived exertion (RPE). Eight cyclists performed cycling trials at 15°C (COOL), 25°C (NORM) and 35°C (HOT) (Humidity 65%), during which they were instructed to cycle at a Borg Rating of Perceived exertion (RPE) of 16, increasing or decreasing their power output in order to maintain this RPE. Power output declined linearly in all three trials, and the rate of decline was significantly higher in HOT than in NORM and COOL (2.35 ± 0.73 W/min vs. 1.63 ± 0.70 vs. 1.61 ± 0.80 W/min respectively, $P < 0.05$). The rate of heat storage was significantly higher in HOT for the first four minutes of the trials only, as a result of increasing skin temperatures. Thereafter, no differences in heat storage were found between conditions. It is concluded that the regulation of exercise intensity is controlled based on initial afferent feedback regarding the rate of heat storage, which is used to regulate exercise intensity and hence the rate of heat storage for the remainder of the anticipated exercise bout. This regulation maintains thermal homeostasis by reducing the exercise workrate, and utilized the subjective RPE specifically to ensure that excessive heat accumulation does not occur and cellular catastrophe is avoided.

Study 4

Study 4 aimed to examine whether skeletal muscle activation, measured indirectly using IEMG measurement, was maximal at volitional fatigue. Ten male cyclists performed an incremental cycling trial to fatigue, classically used to measure the peak power output (PPO). Ten minutes after the completion of PPO, subjects performed a series of intermittent 20-second sprint bouts at power outputs ranging from 80% to the maximal power output that could be maintained for 20 seconds (SUP). During both PPO and SUP, IEMG activity in the active quadriceps muscles was measured, together with heart rate and RPE. Mean peak power output during PPO was 426 ± 26 W. During the supramaximal cycling bouts (SUP), the maximal power output was 905 ± 60 W. IEMG activity increased linearly across the entire range of power outputs of which the subjects were capable in both PPO and SUP. At fatigue during SUP, the IEMG activity in vastus lateralis and vastus medialis were $177 \pm 27\%$ and $191 \pm 28\%$ of the value measured at volitional fatigue during PPO respectively. Volitional fatigue during PPO therefore occurred at submaximal levels of skeletal muscle activation, suggesting that volitional fatigue is the result of a limitation in the degree of skeletal muscle activation (central regulation), rather than simply being the consequence of a failure of maximally activated muscle to sustain a required work rate (peripheral failure). Exercise is thus regulated centrally by a complex system, the function of which is to ensure that exercise terminates before homeostasis fails and a catastrophic organ damage develops.

Study 5

Study 5 of this thesis analysed pacing strategies employed during men's world record performances for 800 m, 5 000 m and 10 000 m. In the 800 m event, lap times were analysed for 26 world record performances from 1912 to 1997. In the 5 000 m and 10 000 m events, times for each kilometer were analysed for 32 (1922 to 2004) and 34 (1921 to 2004) world records respectively. The second lap in the 800 m event was significantly slower than the first lap (52.0 ± 1.7 s vs $54.4 \pm$

4.9 s, $P < 0.00005$). In only two world records was the second lap faster than the first lap. In the 5 000 m and 10 000 m events, the first and final kilometers were significantly faster than the middle kilometer intervals, resulting in an overall even pace with an endspurt at the end. The optimal pacing strategy during world record performances differs for the 800 m event compared to the 5 000 and 10 000 m events. In the 800 m event, greater running speeds are achieved in the first lap, and the ability to increase running speed on the second lap appears limited. In the 5 000 and 10 000 m events, an endspurt occurs due to the maintenance of a reserve during the middle part of the race. In all events, pacing strategy is regulated in a complex system which balances the demand for optimal performance with the requirement to defend homeostasis during exercise.

List of abbreviations used in this thesis

cm	Centimeters	RPE	Rating of Perceived
°C	Degrees Celsius		Exertion
EEG	Electroencephalogram	s	Seconds
EMG	Electromyogram	T	Temperature (Dry bulb)
F _I O ₂	Fractional content of	T _{body}	Body temperature
	oxygen	T _{core}	Core temperature
g	Grams	T _{rec}	Rectal temperature
h	Hours	T _{skin}	Skin temperature
IEMG	Integrated electromyogram	TT	Time Trial
HR	Heart rate	VO ₂	Oxygen consumption
kJ	Kilojoules	VO ₂ max	Maximal oxygen
km	Kilometers		consumption
L	Liters	W	Watts
m	Meters		
min	Minutes		
mL	Milliliters		
MVC	Maximal Voluntary Contraction		
PPO	Peak power output		
Q _c	Heat content		
Q _s	Heat storage		
RH	Relative Humidity		

Chapter 1

Review of the Literature

Pacing strategy during self-paced exercise performance

University of Cape Town

Introduction

Pacing strategy has been described as the efficient use of energetic resources during athletic competition, so that all available energy stores are “used before finishing a race, but not so far from the end of a race that a meaningful slowdown can occur” (Foster *et al.* 2003). This description can be extended to include the regulation of other physiological variables, such as the rate of heat storage (Marino *et al.* 2000; Marino *et al.* 2004), into the role of the appropriate pacing strategy during self-paced exercise.

It is apparent from this definition that pacing strategies must be regulated to prevent changes in physiological systems that may be limiting or detrimental to performance. This regulation and control is not well understood, however, and provides a challenge to classical models of fatigue and the factors that are considered to limit exercise performance (Noakes, 2000). For example, during endurance exercise in hot and humid environments, it has been proposed that fatigue and a resultant decrease in force production, running speed or power output occurs because the body temperature rises to reach critically high levels, above which volitional exercise is not possible (Nielsen *et al.* 1990; Nielsen, 1996; Galloway & Maughan 1997; Nybo & Nielsen 2001a). The proposed mechanism for the failure to maintain force output is that the activation of skeletal muscle motor units by the brain is reduced above a critical core temperature of approximately 40°C.

In contrast, studies of self-paced exercise have found that motor unit activation and performance are reduced before the body temperature reaches critically high levels. For example, Morrison *et al.* (2004) found that isometric force production and voluntary activation percentage decrease progressively during a passive heating protocol, even at body temperatures below 39°C (Morrison *et al.* 2004). Todd and colleagues (2005) have shown a reduction in force output due to a failure of voluntary drive during passive heating despite the availability of additional motor cortical output, which would, in theory, allow an increased force output. Finally, Marino *et al.* (2004) found that African runners maintained higher running speeds during self-paced 8 km running trials in the heat than did Caucasian runners, despite core temperatures that were not significantly elevated or even different from the Caucasians'.

Collectively, these studies provide evidence for a pacing strategy that is regulated in advance of, in this case, the attainment of a critically high body temperature. According to this definition, the pacing strategy is not simply the result of a change in a physiological system, but is in fact mediated specifically to prevent that change from occurring. It has been suggested that the pacing strategy is a key component of a proposed teleoanticipatory system (Ulmer, 1996; St Clair Gibson *et al.* 2001b), in which a central governor (Noakes *et al.* 2001) regulates exercise intensity and alters the adopted pacing strategy specifically to ensure that potentially catastrophic derangements to homeostasis do not occur (St Clair Gibson & Noakes 2004; Lambert *et al.* 2005; Noakes *et al.* 2005). Studies of the

pacing strategies of elite and well-trained athletes during competitive or laboratory controlled maximal exercise time-trials may allow insight into the physiological and regulatory processes that underlie exercise performance. The present thesis aims to identify and propose potential mechanisms for the regulation and control of pacing strategies during self-paced exercise under various conditions.

Accordingly, the aim of the present review is to examine the regulation of pacing strategy during both short duration and long duration self-paced exercise bouts. It examines studies which have experimentally altered the pacing strategy, as well as studies which have examined how various interventions such as high temperatures, hyperoxia, hypoxia and altered substrate availability influence the adopted pacing strategy. Specific emphasis is placed on exercise performed in hot and humid environments.

Studies of pacing strategy

Pacing strategy can be examined in one of two ways. In the first method, which is the focus of the present thesis, the self-selected pacing strategy can be observed during either laboratory time-trials or competitive events. Because pacing strategy is dependent on external factors such as environment, race situation and the influence of other competitors, these studies may not necessarily reflect optimal pacing strategy for a given set of circumstances. However, they do allow factors

such as ambient temperature (Tatterson *et al.* 2000; Marino *et al.* 2004; Tucker *et al.* 2004), oxygen content of the inspired air (Peltonen *et al.* 1997; Brosnan *et al.* 2000) and expected exercise duration (Ansley *et al.* 2004) to be manipulated, and the potential physiological mechanisms that could explain any alternations in pacing strategy and performance induced by these changes, can be postulated.

In the second method, the pacing strategy can be experimentally altered by forcing the athlete to consciously begin a trial at a pace that is either faster or slower than the self-selected pace. This allows the effects of experimental manipulations on overall performance to be studied, and inferences can be drawn regarding the optimal pacing strategy. Owing to the difficulty of accurately controlling the exercise intensity for long periods, such experimental interventions are typically used for shorter duration exercise (less than 4 minutes) only. A variation on experimental manipulations of the actual pacing strategy is the use of computer models which predict pacing strategies during competitions. These computer models are used to simulate competitive events, and they generate predicted split times which can then be compared to actual performances.

As described, the focus of the present thesis is on those studies that have examined alterations in pacing strategy and described proposed mechanisms as a result of interventions including changes in the environmental temperature, oxygen content, muscle glycogen concentrated, or expected exercise duration. In the current review, these methods will be considered separately.

Observational studies of pacing strategy

Pacing strategy can be observed by studying changes in exercise intensity (running speed and power output, for example) during self-paced exercise performance. Studies in which the exercise workrate is free to vary show a characteristic pacing strategy depending on the distance or the duration of exercise. In shorter duration events (< 4 minutes), the selected strategy typically involves a fast start, with power output declining progressively until completion of the trial (Ferro *et al.* 2001; Foster *et al.* 2003; Ansley *et al.* 2004). For example, during a 1 500 m cycling time-trial (approximately 2 minutes), power output and velocity peaked in the first 300 m, and then decreased progressively so that the lowest velocity was recorded at the completion of the trial (Foster *et al.* 2003). Exercise trials such as the Wingate Anaerobic test, classically used to assess anaerobic (oxygen independent) exercise performance, show a similar pattern, with power output decreasing by almost 50% from the start to finish of a 30 second trial (Ansley *et al.* 2004).

Analysis of the sprinting events (100 m, 200 m and 400 m) at the 7th IAAF World Championships of Athletics showed that every single athlete adopted a positive pacing strategy, in which the highest speeds are achieved early on during the event, with a progressive slowing down until the finish line (Ferro *et al.* 2001). These results are in agreement with computer simulations and studies in which the pacing strategy has been manipulated, since they support the notion that for

shorter duration events, optimal pacing requires a fast start, even if this results in a reduction in velocity at the end of exercise.

As exercise duration increases (> 4 minutes), the self-selected pacing strategy becomes more even, and during longer duration exercise, self-selected pacing is characterized by the ability to significantly elevate power output or running speed at the end of the event (Marino *et al.* 2000; Tatterson *et al.* 2000; St Clair Gibson *et al.* 2001b; Marino *et al.* 2004). More specifically, these events typically begin with a relatively high power output, followed by a reduction in power output during the middle part of the trial or race, before power output increases significantly towards the end of the exercise bout, a phenomenon termed an endspurt (Baden, 2002; Albertus *et al.* 2005). The physiological basis for this characteristic pacing strategy is discussed subsequently.

To our knowledge, only one study has examined pacing strategy during a single bout of ultra-endurance exercise (> 6 hours) (Lambert *et al.* 2004). This study found that running speeds were relatively constant for the first 50 km of a 100 km running race, but then declined progressively over the second half of the race, resulting in an overall positive pacing strategy. The best athletes showed the smallest reduction in running speed during the second half of the race. It must also be noted that it is possible that an endspurt was present at the end of the 100 km race, but this was not detected because split times were available for 10 km intervals only.

Experimental manipulations of pacing strategy

Considering that the appropriate pacing strategy is essential for optimal exercise performance (Foster *et al.* 1994; Bishop *et al.* 2002), few studies have systematically evaluated the effect of experimentally altering the pacing strategy on performance. Those studies that have often differ with regards to how the desired pacing strategy is achieved, and consequently, there is equivocal evidence regarding which pacing strategy (that is, slower or faster start compared to even pace throughout the event) achieves the best overall performance (Foster *et al.* 1993; Bishop *et al.* 2002; Foster *et al.* 2003; Thompson *et al.* 2004).

Some studies in which the pacing strategy is manipulated by forcing athletes to start either faster or slower than their average speeds for their best performances have found that an even pace or slower start is optimal (Foster *et al.* 1993; Thompson *et al.* 2004). Others have found that a faster start improves overall performance (Bishop *et al.* 2002). It appears that the length of the exercise bout is an important determinant of which strategy is optimal (Ingen Schenau *et al.* 1992; Ingen Schenau *et al.* 1994). However, very few studies have examined pacing strategy in longer duration exercise (Lambert *et al.* 2004), with most studies focusing on exercise events lasting between one and four minutes.

The first recorded study of the effects of experimental manipulations of pacing strategy on physiology was performed in 1958 by Robinson *et al.* (1958). Three

well-trained men ran the same distance (approximately 1200 m, four minutes) using a faster start (107% of even pace for the first minute), an even paced start or a slow start (97% of even pace for the first minute). It was found that VO_2 , post exercise blood lactate concentrations and the subjective effort rating were all higher with the faster start. It was concluded that a faster start would result in sub-optimal performances, due to these relatively larger changes and so even pacing strategies were recommended for best performances.

More recently, Foster and colleagues examined the effect of imposing different pacing strategies on performance during a 2000 m cycling time-trial (approximately 2.5 minutes) (Foster *et al.* 1993). Well-trained subjects first performed a maximal self-paced trial, and the split times from this trial were used to prescribe pacing strategies for subsequent experimental trials. In these trials, the pace over the first 1000 m was controlled so that athletes completed the first 1000 m in times ranging from 55% (Very Slow) to 48% (Very Fast) of their overall self-paced performance time for the total distance. Subjects were instructed to complete the final 1000 m as fast as possible. Optimal performances were achieved with the even pacing strategy, in which the first kilometer was completed in 50.9% of the overall time. In the Very Fast trial, in which the first kilometer was completed in 48% of the total time, overall performance was impaired by 2.9%. When the speed over the first kilometer was regulated to be significantly slower, the overall performance was impaired by 7.2 seconds, which represents a 4.3% impairment in performance compared to the optimal even pacing strategy. This represents the

difference between first and eleventh in the 1992 Olympic pursuit cycling competition. This study reinforces the importance of appropriate pacing strategy to achieve superior performances at the elite level (Foster *et al.* 1994).

In contrast to that study (Foster *et al.* 1993), 2 minute kayaking performance was improved when a faster start was imposed (Bishop *et al.* 2002). Experienced male kayak paddlers performed two maximal 2-minute trials, during which power output was recorded for use in subsequent manipulated trials. Subjects then performed two experimental trials using either an all-out pacing strategy or an even paced strategy. For the all-out pacing strategy, subjects were required to work at maximum effort for the first 10 seconds, followed by a 5 second transition to an even pace, which was maintained for the remainder of the first minute. During the even paced trial, subjects accelerated as quickly as possible to the stipulated pace, and then maintained this until the end of the first minute. In both trials, subjects were instructed to complete the second minute as fast as possible.

It was found that the average power output was 3.5% higher in the trial using the all-out pacing strategy (348 W for all-out vs. 336 W for even pace, $P < 0.05$), as a result of a significantly higher power output in the first 60 seconds (12% greater in all-out pacing than even pacing). Interestingly, the power output during the second half of the all-out trial declined by 14%, whereas in the even paced trial, power output was maintained compared to the first half. This resulted in a significantly higher power output during the second half of the even paced trial

than the all out trial (337 W in Even vs. 323 W in All-out, 4.3% greater, $P < 0.05$). However, this 14% reduction in power output in the second half of the all-out trial and the improved ability to finish faster in the even paced trial did not compensate for the time that was 'lost' during the first minute in the more conservative start compared to the all-out trial, resulting in the overall improvement in performance of 3.5% in the All-out trial.

Similarly, 200 m breaststroke performance (approximately 2.5 minutes) was improved when a faster start was forced (Thompson *et al.* 2004). In that study, well-trained swimmers were required to perform a paced 200 m breaststroke trial at speeds corresponding to 98% (slow trial), 100% (even paced) and 102% (fast trial) of a previously performed self-paced trial. It was found that the fast trial was significantly faster than the even paced trial (160.0 s vs. 161.3 s, a 0.8% improvement). Interestingly, this occurred even though subjects slowed significantly in the second half of the fast trial and were unable to match the target times set for that section of the trial. That is, the actual time in the fast paced trial after 150 m was 1.0 seconds slower than the target time, and after 200 m, actual time was 4.0 seconds slower than the target time.

However, despite the inability to maintain the faster speeds at the end of the trials, overall performance was still significantly faster than when the trial was performed at even pace. This was similar to the findings in the kayak study of Bishop *et al.* (2002). Thus, in both examples, overall performance was optimized by a fast start despite a progressive reduction in speed during the trial.

Finally, Foster *et al.* (1994) examined speed-skaters during experimentally paced 1 500 m competitions (approximately 120 s), and found that in contrast to 2 km cycling time trials (150 s) (Foster *et al.* 1993), performance was optimized by a faster start. The authors attributed this different outcome (skating vs. cycling) to the reportedly very good ice conditions and to the altitude at which trials were performed. It can be assumed that the air resistance was lower at altitude, which meant that skating velocity did not decrease as much as would be expected for any decline in power output at sea-level. This study indicated that in addition to the duration of the event, other factors contribute to the optimal pacing strategy.

Therefore, under certain conditions, a faster start with a progressive decline in power output may be the favoured pacing strategy due to an ability to maintain speed despite relatively large reductions in power output as a result of fatigue (Foster *et al.* 1993). Indeed, this is supported by an analysis of the pacing strategy during two different Olympic Games speed skating competitions. In the high altitude and good ice conditions of the Calgary Olympics of 1988, 1 500 m speed skaters achieved higher velocities early on during the race and slowed down less than in the Albertville Games, held at a lower altitude four years later (Foster *et al.* 1993).

Computer simulations of pacing strategy

Computer-based models have also been used as a tool to study pacing strategy during various competitive events (Ingen Schenau *et al.* 1992; de Koning *et al.* 1999; de Koning *et al.* 2005). These models depend on assumptions about how the non-oxidative energy capacity is used during exercise, and are based on the premise that velocity at the end of the event is wasted kinetic energy. Despite these assumptions, it has been found that the models accurately predict performances and even split times during competitive speed skating and cycling events (Ingen Schenau *et al.* 1992; de Koning *et al.* 1999). Using such models, the effects of different pacing strategies on performance can also be examined. It has been found that in 1 000 m cycling time trials, best results are achieved with an all out pacing strategy and a deceleration towards the end of the event. In 4 000 m cycling time trials, lasting between 4 and 5 minutes, performance is optimized by an all out start lasting 12 seconds, followed by a constant power output and overall even pacing strategy (Ingen Schenau *et al.* 1992; de Koning *et al.* 1999). This is similar to the simulated optimal power output in the kayaking study of Bishop *et al.* (2002).

Method differences between studies

To my knowledge, these are the only studies which have systematically examined the effects of controlled manipulations of power output or velocity on exercise

performance, either during actual performance or in computer-generated models. It must be noted, however, that differences between the methods in the studies make definitive conclusions regarding the optimal pacing strategy difficult. For example, the faster paced trial in the study of Foster et al (1993) was performed with a first kilometer (50% of the total trial distance) which was faster than the overall pace of a previously performed even-paced trial, and did not improve performance compared to an even-pacing strategy.

In contrast, in the study of Bishop et al. (2002) and in the simulations based on modelled energy flow (Ingen Schenau *et al.* 1992; de Koning *et al.* 1999), the faster paced trials that optimize performance were performed with an all out start lasting between 12 and 15 seconds. Bishop et al. (2002) have suggested that this brief, all-out start results in greater work output compared to the sustained, though relatively sub-maximal start employed by Foster et al. (1993). This then explains why Bishop found that the faster start improved performance whereas Foster found that an even-pace was optimal. Interestingly, both studies found that power output declined substantially in the second half of the trial, resulting in an overall positive pacing strategy, but in the 2 minute kayaking trial, overall performance was still improved, because of the supramaximal power output in the first 15 seconds of the trial.

In another example, a faster start improved 200 m breaststroke swimming performance by 0.8% compared to an even paced trial (Thompson *et al.* 2004), but

in neither trial were subjects instructed to complete the trial as fast as possible. In contrast, in the studies of both Bishop et al. (2002) and Foster et al. (1993), subjects were instructed to perform the final half of the trial with maximal effort. Therefore, the authors suggested that in the even paced trial, swimmers might have been able to swim faster in the final 50 m if instructed to do so (Thompson et al. 2004). However, inspection of the results suggests that this would not be the case, since they were unable even to meet the target time in the even paced trial in the final 50 m, falling behind by 1.3 seconds at 200 m (Thompson et al. 2004). There was thus a progressive slowing down in the even paced trial, despite efforts to maintain a required target pace, which suggests that it is unlikely that the swimmers would have had the ability to increase pace, even if instructed to do so. Regardless of this, however, this study did not allow an all out start for 10 to 15 seconds followed by even pace, which has been found to optimize performance. Under such conditions, it is possible that performance would be further improved by the faster pacing strategy.

Conclusion

Based on studies that have experimentally manipulated the pacing strategy, the length of the exercise bout is a critical factor determining the optimal pacing strategy. It appears that shorter events lasting less than two minutes require a fast start, even if power output or speed decline rapidly towards the end of the race (Ingen Schenau *et al.* 1992; Ingen Schenau *et al.* 1994; de Koning *et al.* 1999). In contrast, events lasting longer than two minutes appear to be optimized by even pacing strategies (Foster *et al.* 1993; Ingen Schenau *et al.* 1994). The physiological basis for these findings, and the apparent differences in the optimal pacing strategy between short and long duration exercise bouts, will be discussed in detail subsequently.

In addition to exercise duration, factors such as altitude and surface conditions have been proposed to determine ideal pacing strategy (Foster *et al.* 1993; Foster *et al.* 1994), since they influence the degree of slowdown that would occur if power output declined at the end of the event. These factors would depend on the activity being performed, and so it has been suggested that running, swimming and rowing performance would benefit from an even pacing strategy because of the large drag forces which would cause large reductions in speed if power output decreased. In contrast, in cycling and speed skating, the relatively small frictional losses might favour a more positive pacing strategy (Foster *et al.* 1993).

While this may be true in theory, it does not appear to be supported by the limited number of studies in which pacing is experimentally manipulated, since 2.5 minute cycling performance was improved by an even pacing strategy (Foster *et al.* 1993), whereas 2 minute kayaking performance (Bishop *et al.* 2002) and 2.5 minute swimming performance (Thompson *et al.* 2004), with higher drag forces, were improved with a faster start. However, as described, different methods and different levels of athletes have been used in these studies, and further studies are required to fully understand how different activities may be influenced by different pacing strategies.

Finally, it is notable that very few studies have examined the effect of manipulations of pacing strategy on performance in events longer than four minutes, probably due to the difficulty in controlling pacing strategies and split times in longer events. Further research is required to determine at which distances or durations the optimal pacing strategy changes from an all out start with a progressive reduction in power output, to a more even pacing strategy. However, an understanding of the regulation of pacing strategy during these longer duration events can be gained from physiological studies of self-paced exercise trials.

Physiological basis for the observed optimal pacing strategy during long duration exercise bouts

As described previously, it is typically observed that athletes elevated power output significantly at the end of longer duration exercise bouts. This observation has physiological significance, since it indicates that the exercise intensity during the middle part of the exercise bout is sub-maximal. This finding supports the concept that exercise intensity is regulated in a complex, feedforward manner, rather than being the result of a catastrophic failure in one or more homeostatic systems (Noakes & St Clair Gibson 2004; Noakes *et al.* 2005). In such a complex system, the observed pacing strategy may fulfil a teleological role, being the consequence of the underlying physiological regulatory processes occurring during exercise (St Clair Gibson & Noakes 2004; Lambert *et al.* 2005), while at the same being the means by which homeostasis is regulated. Thus, changes in the external environment which influence homeostatically regulated variables such as body temperature or oxygen availability would be expected to induce a change in the pacing strategy.

Numerous studies have examined the effects of changing ambient temperature (Marino *et al.* 2000; Tatterson *et al.* 2000; Marino *et al.* 2004), oxygen content of the inspired air (Peltonen *et al.* 1995; Peltonen *et al.* 1997; Brosnan *et al.* 2000), muscle glycogen content (Rauch *et al.* 2005) and feedback regarding distance or

duration (Ansley *et al.* 2004; Albertus *et al.* 2005; Baden *et al.* 2005) on exercise performance. The following section describes these studies, evaluating the physiological basis for the characteristically observed pacing strategies during endurance exercise.

Exercise in the heat

The critical internal temperature hypothesis

Environmental temperature has long been recognised as a critical factor affecting endurance exercise performance. It is known that hot (30°C to 40°C) conditions markedly impair exercise performance compared with cool (3°C to 20°C) conditions (Fink & Costill 1975; Savard *et al.* 1988; Nielsen *et al.* 1990; Galloway & Maughan 1997; Nybo & Nielsen 2001a; Drust *et al.* 2005). Originally it was believed that the impairment of exercise performance in the heat was the result of a reduction in skeletal muscle blood flow (Rowell *et al.* 1966; Fink & Costill 1975) as a result of reduced stroke volume and cardiac output (Rowell *et al.* 1966), due to the challenges imposed on the circulatory system by the hot environment, in particular the need to perfuse both working muscle to maintain the power output and the skin in order to thermoregulate.

However, it is now known that the termination of exercise in the heat is not caused by reductions in cardiac output or exercising muscle blood flow, by

impaired substrate availability or utilization, or by the accumulation of lactate or K^+ (Savard *et al.* 1988; Nielsen *et al.* 1990; Nielsen *et al.* 1993; Gonzalez-Alonso *et al.* 1999; Drust *et al.* 2005). But, such fatigue in well-trained individuals has been observed to occur at a core temperature of approximately 40°C (Nielsen *et al.* 1990; Nielsen *et al.* 1993; Galloway & Maughan 1997; Nybo & Nielsen 2001a), irrespective of the rate of heat storage, the pre-exercise core temperature (Gonzalez-Alonso *et al.* 1999), or the extent of prior heat acclimatization (Nielsen *et al.* 1993; Nielsen *et al.* 1997). In moderately fit individuals, this 'limit' has been established at a rectal temperature of approximately 38.7°C, regardless of hydration or acclimation status (Cheung & McLellan 1998).

Symptoms commonly associated with volitional exhaustion during exercise include confusion, loss of coordination, and syncope (Cheung & Sleivert 2004b), suggesting a possible involvement of the central nervous system in fatigue. Accordingly, it has been proposed that fatigue during exercise in the heat is associated with a "critical internal temperature limiting exercise performance" (Bruck & Olschewski 1987; Fuller *et al.* 1998; Gonzalez-Alonso *et al.* 1999), in which a high body temperature directly affects central nervous functions (Nielsen *et al.* 1990; Cheung & Sleivert 2004b), including a failure to maintain central drive to continue exercise (Nybo & Nielsen 2001a).

In 2001, Nybo & Nielsen (2001a) showed that force production and voluntary activation percentage in the exercised muscle groups (knee extensors) were

lower during a sustained isometric maximal voluntary contraction (MVC) following cycle exercise in hot (40°C, sufficient to raise body temperature to 40°C) than in temperate (18°C, final core temperature 38°C) conditions. Significantly, the overall force produced when electrical stimulation was superimposed upon voluntary contraction was unchanged from values measured during the temperate trial. This indicates that the force-generating capacity of the exercised muscle was unaffected by the elevated core and muscle temperatures after exercise in the heat. It was concluded that exercised-induced hyperthermia caused a form of “central fatigue”, in which elevated body temperature (> 40°C) caused reduced central activation of the exercised muscles by the motor cortex leading to a lower force production.

Recent novel research (Nielsen *et al.* 2001; Nybo & Nielsen 2001b; Rasmussen *et al.* 2004) has demonstrated a possible effect of hyperthermia on arousal levels (a proxy for motivation or ‘drive’) by examining changes in the EEG signal during exercise in hot (40°C) and cool (~19°C) conditions. Subjects cycled to volitional fatigue at a fixed workrate, and the α -to- β wave ratio was measured as an index of arousal levels, with an increase in the ratio suggesting that arousal levels are reduced (Nielsen *et al.* 2001; Rasmussen *et al.* 2004). It was found that the α -to- β ratio increased during exercise in the heat, and this increase was strongly correlated with the increase in core temperature (Nielsen *et al.* 2001; Rasmussen *et al.* 2004), and to the increase in the rating of perceived exertion (Nybo & Nielsen 2001b; Rasmussen *et al.* 2004).

The strong linear correlation ($r^2 = 0.98$, $P < 0.001$) between the reduction in arousal (as measured by the increase in α -to- β ratio) and the increased body temperature (Nielsen *et al.* 2001) is of interest, because it suggests that arousal levels decrease progressively as body temperature increases, rather than simply falling after the core temperature reaches 40°C. Therefore, at a slightly elevated body temperature of 38.5°C, arousal levels are already reduced compared to a body temperature of 37.5°C.

This may have implications for pacing strategies and performance when exercise workrate can be self-selected, since any reduction in arousal would presumably cause the exercising athlete to voluntarily reduce their exercise intensity, rather than waiting for the body temperature to reach 40°C before arousal levels decreased sufficiently to cause volitional fatigue. The authors did not acknowledge this possibility, suggesting instead that the data supported the notion that volitional exhaustion occurred at a body temperature of approximately 40°C due to a reduction in arousal levels, leading to reduced voluntary activation levels. This explanation fits the catastrophic model of how exercise performance is regulated (Noakes & St Clair Gibson 2004; Noakes *et al.* 2005).

The observation that the RPE was strongly correlated to both the body temperature ($r = 0.98$, $P < 0.001$) and to the α -to- β ratio ($r = 0.98$, $P < 0.001$) (Nybo & Nielsen 2001b) is also noteworthy, for it suggests that the RPE may be an important variable that is progressively influenced by the increase in body

temperature. The authors suggested that changes in core temperature and α -to- β index are the best predictors of an increase in RPE, which is “associated with increased difficulty to maintain the required exercise intensity” (Rasmussen *et al.* 2004). If the increase in RPE suggested the workrate was becoming more difficult to maintain, then it seems probable that the subjects would reduce the workrate, if they had the opportunity to do so, as during self-paced exercise. This possibility is discussed subsequently.

Finally, there was no change in EMG activity during the trials (Nielsen *et al.* 2001; Nybo & Nielsen 2001b) and the authors concluded that hyperthermia does not affect the electrical activation pattern of the active skeletal muscles. However, because these trials used a fixed work rate protocol, in which the subject cycled at a predetermined, fixed power output, it may be argued that this finding is expected, and shows simply that the degree of muscle activation required to maintain the power output was not different between hot and cool conditions. Instead, the authors might have concluded that not allowing the subjects to alter their work rate during exercise in the heat resulted in a faster rise in body temperature and a reduced exercise time to fatigue.

Pacing strategies during self-paced exercise performance in the heat

A different picture emerges when exercise is self-paced and the athlete is able to increase or decrease the exercise workrate volitionally. It has been found that power output during cycling (Tattersson *et al.* 2000; Kay *et al.* 2001) and running speed (Marino *et al.* 2000; Marino *et al.* 2004) are reduced soon after the onset of exercise in hot compared to cool conditions. More importantly, these changes occur before body temperatures reach values that are commonly associated with a performance limitation or bodily harm (Nielsen *et al.* 1997; Nybo & Nielsen 2001a).

For example, the pacing strategy in hot conditions is different between African and Caucasian runners (Marino *et al.* 2004), and between larger and smaller runners (Marino *et al.* 2000). The smaller African runners paced themselves similarly in hot and cool conditions, whereas larger Caucasian runners showed a significant decrease in running speed in hot conditions, but not cold conditions. Since body size is known to be an important determinant of the rate of heat storage (Dennis & Noakes 1999), it was proposed that the difference in pacing strategy occurred because the brain was sensitive to the rate of heat storage. As a result, it reduced the running speed of the larger Caucasian runners during time-trials in hot conditions to ensure that excessive heat storage did not occur.

Similarly, Tatterson et al. (2000) found that the power output during self-paced cycling decreased in hot (32°C) compared to temperate (23°C) conditions even though body temperatures were not different between the conditions. It was concluded that the brain was sensitive to the rate of heat storage and mediated a reduction in power output to prevent excessive heat storage during the hot trials. The involvement of the brain in the reduction of power output or running speed in these studies could not be proven, however, since no measures of muscle activation were obtained.

Morrison *et al.* (2004) did measure voluntary muscle activation during self-paced exercise in the heat. They examined the effect of core and skin temperature on maximal voluntary contractions (MVC) during an isometric knee-extension, performed while subjects were passively heated from a core temperature of 37.5°C to 39.5°C and then gradually cooled again to starting temperatures. A 10-second MVC was performed at every 0.5°C increase in core temperature, with two muscle stimulations performed during each MVC to determine the magnitude of voluntary activation of the muscle. Their results showed a gradual and progressive decrease in force production during the MVC and electrical stimulation as body temperature increased, with a subsequent return to baseline with cooling. Therefore, the brain selectively activated a smaller muscle mass throughout the range of increasing body temperatures, rather than only after a 'critical temperature' had been reached.

This result (Morrison *et al.* 2004) does not support the hypothesis that a reduction in skeletal muscle recruitment occurs only after a critically high core temperature is reached. Rather, the data contributes to the growing body of evidence for anticipatory regulation of exercise in the heat (Marino, 2004), which suggests that during exercise where force output is selected by the individual and is free to vary, motor command and voluntary activation are reduced incrementally as core temperature rises.

Todd *et al.* (2005) attributed hyperthermia-induced fatigue to a combination of factors in both the muscle and the motor cortex. They measured a reduction in force output which occurred due to a failure of voluntary drive during passive heating despite the availability of additional motor cortical output, which would, in theory, allow increased force output (Todd *et al.* 2005). Therefore, fatigue occurs in the presence of a 'motor cortex' reserve, and a motor unit reserve (Nybo & Nielsen 2001a; Morrison *et al.* 2004), suggesting the role of the brain in this process.

Even more significantly, exercise workrate may be altered even though there are no measurable differences in body temperatures between hot or cool conditions (Marino *et al.* 2000; Tatterson *et al.* 2000; Marino *et al.* 2004). This supports the anticipatory theory of pacing strategy, which holds that a reduction in exercise workrate occurs before the body temperature rises to reach critical or limiting values (Marino, 2004; Marino *et al.* 2004). The classic 'limitations' or catastrophe

model (Noakes & St Clair Gibson 2004; St Clair Gibson & Noakes 2004; Noakes *et al.* 2005) makes no provision for such an occurrence, since it posits that skeletal muscle recruitment and workrate are reduced only after the body temperature has risen beyond a critical limit. This model predicts that pacing strategy will be altered only after body temperatures become different between conditions, yet this is clearly not the case. Accordingly, some other mechanism must exist to explain impaired self-paced exercise performance in the heat.

In conclusion, self-paced exercise performance in the heat is impaired in advance of the attainment of a critically high body temperature. It has been suggested that a centrally-mediated mechanism exists to decrease muscle recruitment and hence exercise workrate when the rate of heat storage is high early on during exercise, or when there is a risk that the core temperature will rise to limiting levels before the anticipated end of the exercise bout (Marino *et al.* 2000; Tattersson *et al.* 2000; Marino, 2004; Marino *et al.* 2004). The mechanism for such a reduction is however unclear.

Exercise with different inspired oxygen content

Changes in the oxygen content of the inspired air also alters the pacing strategy (Peltonen *et al.* 1995; Brosnan *et al.* 2000) and skeletal muscle activation patterns (Peltonen *et al.* 1997) during self-paced exercise. Peltonen *et al.* (1997) examined IEMG activity in seven active muscles during a 2500 m rowing ergometry trial, and

found that hypoxia (F_{iO_2} 15.8%) impaired overall performance as a result of differences in pacing strategy during the trials. That is, force output during maximal rowing strokes decreased progressively during the trials, but the reduction was greater in hypoxia than in normoxia (Peltonen *et al.* 1997). Furthermore, the decline in force production in hypoxia was accompanied by a reduced IEMG activity, suggesting that the level of muscle activation was influenced by the oxygen content of the inspired air.

Kayser *et al.* (1994) have provided evidence for this effect, showing that when cyclists were given hyperoxic air at the point of volitional exhaustion in hypoxic conditions, while at the same moment the exercise load was increased, the subjects were able to continue cycling at the higher power output. The continuation of exercise was associated with an immediate increase in the IEMG activity of one of the active muscles (vastus lateralis). The authors proposed that the extent of skeletal muscle activation during exercise is influenced by the F_{iO_2} .

Interestingly, Peltonen *et al.* (1997) found that altered the pacing strategy and improved performance in hyperoxia were not associated with any differences in IEMG activity compared to normoxia. This finding was attributed to “other factors related to the availability of oxygen”, or due to a neural limitation of muscle recruitment, because “full motor unit recruitment is achieved during normoxia” (Peltonen *et al.* 1997). However, inspection of the results indicates that the IEMG activity was never greater than 75% of the IEMG activity measured

during a maximal effort stroke before the trial, and so skeletal muscle recruitment was clearly sub-maximal during the trial. It may however be that the method of measurement of IEMG in that study (Peltonen *et al.* 1997), which summed seven active muscles during rowing, was not sensitive enough to detect differences in activation in the hyperoxic condition, since only small differences in power output were observed and these differences may have been the result of increased activation of only one of the seven muscle groups that were studied.

The theory that muscle activation levels and exercise intensity are regulated differently in hypoxia and hyperoxia does not discount the observation that peripheral factors such as changes in metabolite levels may result in 'myographical signs of muscle fatigue', in which the measured IEMG activity increased despite no change in force production (Kayser *et al.* 1994; Taylor *et al.* 1997; Kayser, 2003). For example, Taylor *et al.* (1997) found that IEMG activity was greater during sub-maximal cycling at a fixed power output in hypoxic ($F_{I}O_2$ 11.6%) compared to normoxic conditions, suggesting that the force-generating capacity of the muscle was impaired in hypoxia. Further, the ratio of Force/EMG activity decreased progressively during exercise in hypoxia. This suggests that an increase in motor unit recruitment was required to maintain the power output due to a progressive reduction in muscle force generating capacity.

However, the critical point is that power output and IEMG activity can be increased volitionally at the end of exercise time-trials in hyperoxia or hypoxia

(Peltonen *et al.* 1997; Marino *et al.* 2000), indicating that the reduction in power output during the middle part of the trial is not solely due to a failure of muscle contractility but must be part of a regulated process since a greater power output could have been achieved with a greater level of muscle recruitment. Thus, these studies provide evidence for regulation of the degree of motor unit recruitment which is sensitive to the oxygen content of the inspired air (Kayser *et al.* 1994; Peltonen *et al.* 1997).

Energy substrate availability

While the present thesis has not directly examined the role of energy substrate availability on performance and pacing strategy during exercise, substrate availability is often implicated as a limiting factor during exercise performance, and is thus important in a discussion of the control of pacing strategy. Volitional fatigue during exercise at a constant workload is often thought to coincide with muscle or liver glycogen depletion (Bergstrom *et al.* 1967; Havemann *et al.* 2006; Rauch *et al.* 2005). Therefore, if the pacing strategy is regulated to prevent absolute or 'catastrophic' fatigue, as proposed in previous examples, then the overall pacing strategy during self-paced exercise should be altered by dietary interventions that result in either i) different amounts of stored energy (particularly muscle and liver glycogen) before exercise or ii) altered substrate utilization during exercise.

Indeed, a high-fat diet for six days, followed by a single day of high carbohydrate feeding to normalize muscle glycogen stores, impairs performance during repeated 1 km sprints during a self-paced 100 km time trial when compared to performance after a seven day high carbohydrate diet (Havemann *et al.* 2006). However, the slower overall performance (3 minutes and 44 seconds) in the 100 km time-trials following the high fat diet was not significantly different from the high carbohydrate diet, although five out of eight subjects improved while eating a high carbohydrate diet.

It was suggested that the higher intensity sprints were impaired because of an increase in sympathetic activation and consequent increase in effort perception (Stephens *et al.* 2002; Havemann *et al.* 2006) following the high fat intake. Inspection of the results from the study of Havemann (2006) shows that the measured RPE during trials was not however different. Thus, if the high fat diet exerted effects on sympathetic activity and the RPE, then it might have done so by altering the self-selected workrate that could be sustained in order to generate the given RPE. This hypothesis is examined later in the present thesis.

Previous studies using a similar dietary regime have however failed to find an effect of dietary regimes on time-trial performance (Carey *et al.* 2001; Burke *et al.* 2002). These studies are often affected by large individual responses to the different diets, thereby reducing the statistical power. For example, in the study of Havemann *et al.* (2006), five out of eight subjects improved on a high

carbohydrate diet. In contrast, in studies by Carey et al. (2001) and Burke et al. (2002), most subjects improved performance on a high fat diet, with this improvement being attributed to a muscle glycogen sparing effect as a result of increased rates of fat oxidation on the high fat diet. However, these studies differ in that they have used a constant effort time-trial following prolonged submaximal exercise (Carey *et al.* 2001; Burke *et al.* 2002), whereas the study of Havemann (2006) included high intensity sprints during endurance exercise in an attempt to simulate pacing strategies during competition. In all these studies, it appears that altering the pre-exercise glycogen levels and utilization of substrates through dietary manipulations may cause changes in pacing strategy and self-selected power output, but individually different responses make definitive conclusions difficult.

A putative role for glycogen concentration as a signaller and regulator of pacing strategy has been proposed by Rauch et al. (2005), who found that one hour cycling time-trial performance was improved by a high carbohydrate diet which elevated muscle glycogen content. The difference in performance was evident from the onset of the time-trial, as power output in the normal diet group decreased and became lower than in the carbohydrate loaded group after the first minute. Therefore, subjects did not become glycogen 'depleted' before the differences in performance were observed, but slowed down, apparently in advance of such an effect developing. Interestingly, each subject ended the trials with similar muscle glycogen concentrations, irrespective of whether they had

been carbohydrate loaded or depleted. Therefore, subjects made use of the extra carbohydrate in the loaded trials, leading to the hypothesis that subjects paced themselves to reach a critical level of muscle glycogen at the termination of the exercise trial. Pacing was suggested to be the result of afferent feedback, signalling alterations in total substrate availability, thereby allowing a higher power output to be maintained in the carbohydrate loaded state (Rauch *et al.* 2005).

In conclusion, large individual differences in response to energy substrate availability have made definitive conclusions regarding its effects on pacing strategy difficult. Evidence does exist that self-paced exercise performance and pacing strategies are sensitive to alterations in muscle substrate utilization. A role of glycogen as a signaller has been proposed in this regard. Pacing strategy may be regulated during endurance exercise to ensure that a limiting level of glycogen depletion does not occur.

Provision of incorrect distance or duration feedback

In previous examples of the regulation of pacing strategy, a model has been described in which the pacing strategy is altered by a central controller to prevent limiting physiological changes from occurring before the known endpoint of exercise is reached. Implicit in this model is that the endpoint of exercise must be known prior to the commencement of the exercise bout, since any anticipatory calculation cannot be made unless the duration of exercise is known with some

accuracy. That is, if the adjustments in pacing strategy serve to prevent harmful or limiting disturbances to homeostasis before the end of exercise, as is described in this review, then the expected duration of exercise would serve as the 'anchor point' against which this regulation would occur.

To confirm this, the following two hypotheses must be valid: Firstly, if the athlete is **correctly** informed of the upcoming exercise duration before the commencement of exercise, then the provision of incorrect information regarding time and distance intervals during exercise would not be expected to result in changes in performance, provided the mismatch created by the misinformation is sufficiently small so as to not be consciously detected by the subjects. Secondly, if the athlete is **incorrectly** informed about the duration of exercise about to be undertaken, then performance will be negatively affected if the mismatch is eventually revealed or detected (either consciously or sub-consciously), because the allocation of physiological resources will have been based on an incorrect expectation of exercise time before exercise began. Alternatively, if the athlete is **incorrectly** informed of duration, but the discrepancy between expected and actual duration is small and not detected, then performance would be expected to be the same as when the athlete is reliably informed of the exercise duration.

To test the first hypothesis, Albertus et al. (2005) had well-trained male cyclists perform five 20-km cycling time-trials, during which they received either correct or incorrect distance feedback every kilometer. In the incorrect feedback trials,

subjects were told they had completed a kilometer when in fact the actual distance was either shorter or longer, by up to 250 m per kilometer. It was found that overall performance, pacing strategies and the subjective ratings of perceived exertion were not different at any stage in the different trials. This indicates that the control of pacing strategy is rigorous and unaffected by the provision of incorrect feedback, and might be set prior to exercise based on the anticipated exercise duration, at least in exercise of this duration. Presumably, if the mismatches between actual and informed distances were larger, or if the subjects had been aware of their split times at each incorrectly informed kilometer, then the pacing strategy would have been altered in response to these conscious cues. However, because only distance feedback was provided, pacing strategy was based on conscious expectation of the overall exercise duration of 20 km and the sub-conscious regulation based on afferent feedback, as discussed previously.

In contrast, the second requirement of the present model is that exercise performance should be altered when the overall exercise duration differs from what was anticipated before the start of the exercise bout, and large mismatches are detected by the exercising athlete. In support of this, Ansley et al (2004) found evidence of a pre-programmed, centrally regulated pacing strategy during supramaximal exercise lasting only 36 seconds. This study found that when subjects performed a supramaximal cycling trial lasting 36 seconds after being informed that they would be cycling for only 30 seconds, their power output in the final 6 seconds was significantly lower than when they were correctly informed

of the correct duration of the activity – that is, 36 seconds. Thus, when the actual duration of the exercise bout exceeded the anticipated duration by 20%, a significant impairment in performance occurred, suggesting that the physiological resources had been ‘incorrectly’ allocated. It was concluded that a pacing strategy was set based on the anticipated duration of exercise as a result of previous experience.

A third example that workrate is set in anticipation of exercise duration was provided by Nikolopoulos *et al.* (2001), who informed well-trained cyclists that they would be completing 40 km time-trials when the actual distances were 34 km, 40 km and 46 km (a difference of 15% compared to the expected duration). It was found that the pacing strategies and performances during the 34 km and 46 km rides were not different from those measured during the 40 km time-trial, suggesting that the subjects were able to maintain a power output based on the expected distance (40 km) from the onset of exercise. Performances during subsequent 34 km and 46 km time-trials in which subjects were correctly informed of duration were not different from those trials with incorrect distance information (Nikolopoulos *et al.* 2001). This may suggest that the difference in distance between the actual 34 km or 46 km and the expected 40 km was not sufficiently large to allow the subjects to detect it and force them to alter their pacing strategy.

Also, subjects were provided with distance feedback in the form of the percentage of the distance remaining – this would allow the pacing strategy to be modified constantly with respects to the end-point, such that the discrepancy of 6 km in the beginning is progressively reduced as the trial progresses. This study (Nikolopoulos *et al.* 2001) does however provide evidence that well-trained cyclists self-select power outputs and pacing strategies based on their perception or expectation of distance, and not solely on the physiological feedback during exercise.

A final interesting illustration of the importance of expectation of exercise duration, even during exercise at a fixed workrate, was provided by Baden *et al.* (2005). This study suggested that the anticipated exercise duration influences running economy (Baden *et al.* 2005), since the oxygen consumption (VO_2) at a submaximal running speed (corresponding to 75% of previously measured peak treadmill running speed) was lower when subjects were told they would be running for an indeterminate length of time compared to trials in which they were told they would run for only 10 minutes. It was suggested that when an unknown, perhaps longer exercise bout was undertaken, subjects attempted to conserve their resources by improving their energy efficiency (Baden *et al.* 2005). It has also been shown that EMG activity in the biceps muscle is lower during a task of long duration than a task of short duration, despite similar work being performed (Vidacek & Wishner 1971), supporting the idea of greater muscular efficiency in longer tasks.

Conclusion

In conclusion, there is evidence that during long duration exercise, the overall pacing strategy is mediated to prevent premature fatigue caused by a failure of one or more physiological systems. The resulting pacing strategy is thus proposed to be a marker of underlying physiological regulation, and alterations in pacing strategy occur due to changes in muscle activation in an anticipatory manner, based on afferent feedback from the various physiological systems and prior experience. These changes occur in advance of the attainment of limiting increases in body temperature (Tatterson *et al.* 2000; Marino *et al.* 2004), decreases in substrate availability (Rauch *et al.* 2005) or the development of a limiting metabolic acidosis. Knowledge of the exercise duration is required to adjust exercise intensity appropriately. Near the end of an endurance exercise bout, muscle activation can be increased, resulting in the characteristically observed endspurt in which power output or running speed increase significantly.

Critically, this complex central regulation of exercise does not completely prevent homeostatic disturbances from occurring. For example, exercise intensity is not reduced so much that heat storage becomes zero (Marino *et al.* 2004) – a certain amount of heat gain is 'allowed'. Similarly, energy substrate levels (Havemann *et al.* 2006; Rauch *et al.* 2005) and oxygen saturation levels (Peltonen *et al.* 1999) do decline during self-paced exercise, and there is evidence that muscle force generating capacity is reduced during dynamic cycling exercise (Kayser *et al.* 1994;

Taylor *et al.* 1997). However, these changes do not reach those critical levels at which exercise would terminate or bodily harm would occur. Rather a reserve exists in which skeletal muscle activation can be increased to cause increases in whole limb power output even in the presence of such peripheral changes.

Physiological basis for positive pacing strategy during short duration exercise

In contrast to the endspurt observed during longer duration exercise, both observational studies and studies in which initial pacing strategy is manipulated have shown that during short duration exercise bouts lasting less than two minutes, there is an apparent inability to increase workrate at the end of exercise (Foster *et al.* 1993; Ferro *et al.* 2001; Bishop *et al.* 2002; Thompson *et al.* 2004). Instead, there is characteristically a progressive reduction in power output or velocity in these events. This may suggest that exercise intensity is not regulated during short duration exercise, but decreases as a result of a progressive failure of the muscle to produce force (Nummela *et al.* 1992; Taylor *et al.* 1997). This argument is evaluated subsequently.

Evidence for declining muscle force production during short duration self-paced exercise

Kayser *et al.* (1994) found that EMG activity increases progressively during cycling exercise at a constant power output. They interpreted this as evidence for a 'myographical sign of muscle fatigue' (Kayser, 2003). It was suggested that muscle can develop signs of metabolic fatigue but only if the volume of muscle at work is small, or if the workload achieved with larger muscle groups is very high (Kayser, 2003), as would occur during short duration, high intensity exercise. Taylor *et al.* (1997) have found that during high intensity cycling exercise in hypoxia, the ratio of Force/EMG activity decreased progressively, suggesting that an increase in motor unit recruitment was required to maintain the power output caused by a progressive reduction in muscle contractility.

Similarly, Nummela *et al.* (1992) found that drop jump performance was impaired by 39% following a maximal 400 m sprint, and the reduction was correlated negatively with increases in blood lactate concentrations. The EMG activity in the active sprinting muscles increased significantly over the course of the run. It was concluded that additional motor units were being activated to compensate for the progressive reduction in muscle force production as a result of metabolic acidosis in the muscle (Nummela *et al.* 1992). This supports the notion that the progressive decline in power output or running speed during shorter duration exercise bouts is the result of a failure of muscle contractility.

Evidence for pacing strategies during short duration, high intensity exercise

While these studies (Nummela *et al.* 1992; Kayser *et al.* 1994; Taylor *et al.* 1997) provide evidence that exercised muscle is less capable of producing force, studies have also shown an anticipatory component to short duration, high intensity exercise. Most significant is that when short duration exercise is undertaken, the initial power output is lower than is possible if the athlete is instructed to perform an all-out effort with no regard for overall performance (Foster *et al.* 1994).

Therefore, some form of pacing must be present to regulate the initial exercise intensity, even though a progressive reduction in intensity still occurs as exercise continues.

Foster *et al.* (2003) evaluated the pattern of energy system contributions to power output during high intensity cycling lasting less than two minutes, and found that energetic resources were distributed over the duration of the event, apparently to preserve the contribution of nonoxidative energy production to power output throughout the exercise bout. Therefore, rather than depleting the nonoxidative energy stores early in the trials, there was some form of pacing which preserved these sources. It was suggested that the intracellular changes occurring during exercise, such as metabolite accumulation (Diamant *et al.* 1968; Karlsson & Saltin 1970; Jacobs & Kaiser 1982; Jacobs *et al.* 1983) or phosphagen depletion (McLester, Jr., 1997), were being monitored continually and that power output

was reduced in advance of these changes becoming critical or harmful (Foster *et al.* 2003), in agreement with the model proposed for longer duration exercise.

Evidence that the pacing strategy during shorter duration exercise bouts has an anticipatory component comes from the study of Ansley *et al.* (2004), described previously, which showed that performance during a supramaximal exercise bout was impaired only after the expected exercise duration had elapsed. This shows that the appropriate allocation of physiological resources is essential in even very short (36 seconds) exercise bouts, and suggests that the reduction in power output in the first 30 seconds occurs as part of this subconscious allocation, since a greater reduction in power output occurred after the pacing strategy had 'failed'.

Collectively, these studies indicate that an anticipatory pacing strategy exists during exercise lasting less than two minutes, even though power output decreases progressively during exercise (Bishop *et al.* 2002; Foster *et al.* 2003; Thompson *et al.* 2004). As described, the progressive reduction in work output has classically been attributed to impaired ability of muscle to produce force due to changes in metabolite levels, so called 'peripheral fatigue' (Nummela *et al.* 1992; Taylor *et al.* 1997; Noakes, 2000; Lambert *et al.* 2005). This is based on the theory that impaired oxygen delivery or uptake results in the accumulation of metabolites, leading to a reduction in muscle pH, which impairs glycolysis (Hermansen, 1981) and muscle contractile processes (Fabiato & Fabiato 1978).

As such, much of the early focus on the optimal pacing strategy was on the effect of faster or slower starts on oxygen kinetics, including oxygen uptake (VO_2), incurred oxygen debt, post exercise lactate levels and concentrations of other metabolites, including phosphocreatine (PCr) and adenosine triphosphate (ATP). Thus, Robinson et al. (1958) suggested that even pacing was optimal for middle distance running events, based on the finding that a faster start resulted in elevated blood lactate concentrations and oxygen uptake.

Similarly, Thompson et al. (2004) found higher lactate levels, respiratory exchange ratio and Rating of Perceived Exertion (RPE) after 200 m breaststroke trials which began at speeds corresponding to 102% of a previously performed self-paced effort compared to trials performed at 100% of previous swimming times. The authors suggested that this impaired muscle function results from proton accumulation and reduced muscle pH (Thompson *et al.* 2004), and caused the large reduction in speed in the second half of the trial. Regardless of the potential factors responsible for this reduction in swimming speed, this conclusion (Thompson *et al.* 2004) fails to acknowledge that the best overall performance was achieved with the faster starting pace, and so performance was optimized despite these apparent 'limitations' to muscle function as a result of higher lactate concentrations.

Bishop et al. (2002) showed that performance was improved and initial and total oxygen consumption were greater when a kayaking trial was performed with an all

out start compared to an even paced start. No differences were found in accumulated oxygen deficit, blood lactate concentrations or pH. They proposed that the higher initial VO_2 was the result of greater rates of PCr breakdown. Interestingly, as with the study of Thompson et al. (2004), the power output declined significantly in the second half of the trial, but overall performance was still improved with the faster start. Therefore, rather than being detrimental to performance, the proposed elevation in PCr breakdown (and consequent reduction in PCr levels), as well as changes in the levels of other metabolites such as ATP and lactate as a result of the faster start, were associated with improved overall performance.

It can therefore be argued that if an exercise trial is to be performed in the shortest possible time, these metabolic changes are simply the consequences of the high work output required early on during exercise. While it must be acknowledged that the later reduction in power output could occur in part due to the effect of metabolic changes in the muscle, it is suggested that pacing strategies are still present in these events, and that the observed reduction in power output is 'tolerated' or controlled as a consequence of the overall pacing strategy which ensures a balance between protecting against harmful disturbances to homeostasis and the optimization of performance.

It is interesting to speculate whether the requirements to defend homeostasis and optimize performance are in conflict. That is, in order to prevent large

disturbances in metabolic and physiological systems, a relatively lower workrate would have to be selected from the onset of exercise and would negate any possibility of achieving the best possible performance. During shorter duration bouts, an even pacing strategy may fall into this category, with the initial exercise intensity being relatively submaximal and the resultant performance sub-optimal (Foster *et al.* 1993). In contrast, excessively high workrates early on would result in a greater threat to the maintenance of homeostasis, causing a rapid decline in exercise workrate, leading to impaired performance, as demonstrated by Foster *et al.* (1993).

Consequently, if the pacing strategy is a marker of the complex regulation of physiological systems and performance during exercise, then optimal pacing strategy does not necessarily imply fastest performance which might occur, for example, if the systems were pushed beyond their limits. Nor does it suggest minimal physiological derangements. In longer duration exercise, in which there may for example be complete failure to continue exercise when a critical core temperature is reached (Nybo & Nielsen 2001a), or when energy substrates are depleted (Havemann *et al.* 2006), performance would clearly be sub-optimal if these 'limitations' occurred before the exercise bout was completed. Therefore, the maintenance of homeostasis is an essential requirement for optimal performance.

However, in shorter duration exercise bouts, it is possible that metabolic changes such as metabolite accumulation or acidosis, sufficient to cause a progressive and gradual decline in exercise intensity even at the same level of muscle activation, are controlled in order to optimize performance, possibly because these changes in metabolite concentrations are short-lived and reversible. The greater kinetic energy in these events (Foster *et al.* 1993; Foster *et al.* 1994) may also mean that reductions in power output do not affect overall performance time to the same extent as in longer duration exercise.

A further hypothesis is that exercise training may alter pacing strategy, particularly during long duration exercise bouts, by reducing the skeletal muscle reserve that is maintained during the middle part of the trial. Therefore, in addition to altering the rate of change in physiological variables such as body temperature and metabolite concentrations, training would allow the exercising athlete to more closely approach the limits to performance imposed by his or her physiology without reaching them prematurely. Thus, prior experience is vital in determining the optimal pacing strategy, since it ensures that the exercise intensity is neither too high to result in failure to maintain homeostasis, nor too low to result in sub-optimal performance while attempting to prevent changes in physiological variables. Indeed, the anticipatory component of the pacing strategy relies on previous experience of exercise. Further research is required to investigate the effects of training on pacing strategies during self-paced exercise.

Conclusion

In conclusion, it is suggested that there exists a centrally-mediated pacing strategy for self-paced exercise of all durations, the role of which is to balance the requirement for completing an exercise bout in the shortest possible time with the requirement to complete the task without causing irreparable harm to the muscle and other organs. This results in a certain degree of physiological disturbance being tolerated, which may cause a progressive reduction in power output as exercise continues, particularly during short duration (< 4 minutes) exercise. However, since the pacing strategy is regulated based on prior experience and afferent information from the periphery, the task can be completed without the development of bodily harm.

The proposed role for the RPE in the regulation of exercise

Often measured as an index of subjective perception of effort during exercise, the RPE has been causally linked to physiological variables such as muscular force (Cafarelli, 1982), heart rate, ventilation, respiratory rate, oxygen uptake and blood lactate concentrations (Robertson, 1982). Borg has stated that the RPE is the "single best indicator of physical strain", and "integrates various information, including the many signals elicited from the peripheral working muscles and joints, from the central cardiovascular and respiratory functions, and from the central nervous system" (Borg, 1982). The RPE also incorporates other mediators, most

notably psychological and affective components (Rejeski & Ribisl 1980; Hardy & Rejeski 1989). Thus, the overall sensation of exertion measured during exercise is the conscious/verbal manifestation of the integration of these psychological and physiological cues (See Hampson *et al.* (2001) for complete review).

This biological link between the subjective sensation of effort and the physiological changes occurring during exercise is important, since it provides a mechanism by which the RPE could, in theory, contribute to the anticipatory regulation of exercise intensity and pacing strategy. As described earlier in this review, the increase in RPE is linearly correlated to increases in core body temperature during exercise in hot and cool conditions, and to the increase in the α -to- β ratio suggesting reduced arousal levels (Nielsen *et al.* 2001; Nybo & Nielsen 2001b; Rasmussen *et al.* 2004). If exercise at a fixed workrate in the heat is limited due to a failure of central drive (Nybo & Nielsen 2001a; Nybo & Nielsen 2001b), as proposed, then the finding that arousal levels decrease progressively while RPE increases in direct proportion to body temperature, invites the suggestion that if exercise was self-paced, any reduction in arousal (or increase in RPE) caused by physiological changes, could exert effects by enforcing a reduction in the self-selected workrate.

Noakes (2004) recently argued from the data of Baldwin *et al.* (2003) that when exercise was performed at a constant workrate (thus negating any effect of pacing strategy) to volitional fatigue in either a glycogen-loaded or glycogen-depleted

state, the initial and final RPE (at the point of exhaustion) were similar between conditions, but the time to fatigue was significantly shorter in the glycogen depleted state (Baldwin *et al.* 2003). Time to fatigue was however predicted by the rate at which the RPE increased, with RPE increasing more rapidly in the glycogen depleted state, and therefore reaching the final, maximal RPE sooner when glycogen levels were lower. However, when expressed relative to the total trial duration, the RPE increased linearly and at the same rate in both conditions (Noakes, 2004).

This finding has two important implications. First, it reinforces the assertion that the RPE is influenced by afferent physiological feedback, since it increased more rapidly in the glycogen depleted state. Noakes suggested that the absolute rate of increase in RPE reflected “the rate at which fuel reserves were being depleted” (Noakes, 2004), though it may be more appropriate to speculate that the magnitude of the RPE during exercise was dependent on the muscle glycogen content at that time, and was hence higher when glycogen levels were reduced. Studies of exercise performance in the heat have also found that the RPE is linearly correlated with body temperature (Nielsen *et al.* 2001; Nybo & Nielsen 2001b), and maximal values of RPE occur at the same time as the core temperature reaches approximately 40°C (Nielsen *et al.* 2001; Nybo & Nielsen 2001b), irrespective of the rate at which it increases. Also, interventions such as high fat diets (Burke *et al.* 2002; Havemann *et al.* 2006) and hypoxia (Linossier *et*

al. 2000) result in increased effort perception and reduced time to volitional fatigue when the exercise workrate is fixed.

Secondly, the observation that the RPE increased linearly but at different rates in both the glycogen-depleted and glycogen-loaded states (Noakes, 2004) suggests that from the onset of exercise, the conscious perception of effort increases at a rate that will ensure that a maximal tolerable RPE is attained before the complete and limiting depletion of muscle glycogen stores. If exercise was ultimately limited by the depletion of energy substrates, with no anticipatory component, then it would be expected that the RPE would increase rapidly only at the end of exercise when the muscle glycogen stores were approaching critically low levels. However, this was not the case, and instead, the RPE rose at a constant rate throughout exercise in each condition.

Therefore, the rate of RPE increase, and hence the duration of exercise, may have been set from the very beginning of the trial in order to ensure that the maximal RPE occurs before any potentially harmful level of muscle glycogen depletion can occur. Afferent feedback would inform the brain of the availability of muscle glycogen, and the rate of increase in RPE would be set in an 'anticipatory fashion' to ensure that the maximal RPE occurs before glycogen levels are depleted. For this reason, the RPE increases more rapidly from the onset of exercise in the glycogen depleted state. The RPE is therefore not merely the direct result of afferent feedback from peripheral systems, as proposed previously (St Clair

Gibson *et al.* 2001a; St Clair Gibson *et al.* 2003), but plays a role in the anticipatory regulation of exercise, even when the workrate is fixed.

Critically, these studies (Nielsen *et al.* 2001; Nybo & Nielsen 2001b; Baldwin *et al.* 2003; Noakes, 2004; Rasmussen *et al.* 2004) have all examined exercise at a fixed workrate to fatigue. It is not known whether the same phenomenon would occur when exercise is self-paced. It seems reasonable to suggest that rather than simply maintaining a high workrate until the RPE does rise to reach near-maximal levels, the exercising athlete would instead reduce the workrate under conditions of elevated body temperature (Nielsen *et al.* 2001; Nybo & Nielsen 2001b; Rasmussen *et al.* 2004) or depleted muscle glycogen concentrations (Baldwin *et al.* 2003), giving rise to the possibility that the regulation of self-paced exercise may utilize the RPE as an important mediator of pacing strategy.

Summary

The evidence presented here suggests that pacing strategy during self-paced exercise is regulated in a dynamic, complex system which utilizes both feedforward (anticipatory) and feedback information. The optimal pacing strategy depends on numerous factors, most importantly the duration of the exercise bout being performed. During shorter exercise bouts (less than 4 minutes), optimal pacing appears to require a fast start, even though power output may decrease significantly towards the end of the task.

During longer duration exercise bouts, there is evidence that the overall pacing strategy is mediated to prevent premature fatigue due to the failure of one or more physiological systems. The resulting pacing strategy is thus proposed to be a marker of underlying physiological regulation, and alterations in pacing strategy occur due to changes in muscle activation in an anticipatory manner, based on afferent feedback from the various physiological systems and prior experience.

The mechanism by which this anticipatory pacing strategy is regulated is however unknown. It does not appear to be based solely on physiological afferents such as high body temperatures or metabolite concentrations, since differences in performance have been detected in the absence of differences in any measured variables compared to control trials. Nor is the regulation of exercise workrate and muscle function (the effector component of a regulatory system) completely understood.

During exercise to volitional fatigue at a fixed workrate, exercise terminates when the maximal tolerable RPE is reached, and the rate of increase in the RPE determines the length of exercise. The finding that the rate of increase in RPE is related to physiological changes such as body temperature, changes in arousal levels, the availability of energy substrates and the expectation of exercise duration invites the hypothesis that when exercise is self-paced, the RPE may be an important regulator of pacing strategy and performance.

Therefore, the hypothesis of the present thesis is that self-paced exercise is regulated in an anticipatory manner, with the brain regulating skeletal muscle motor unit activation and workrate in order to ensure that exercise can be completed without a 'catastrophic' failure in one or more physiological systems.

The hypothesis is that measures of skeletal muscle activation will reveal that environmental interventions such as high ambient temperatures or elevated inspired oxygen content will alter pacing strategy and performance by means of alterations in muscle activation. Further, I aimed to investigate the potential role of the RPE as a mediator in this anticipatory regulatory process.

Research questions

The following specific research questions will be examined in the present thesis:

Question 1

What is the mechanism for the characteristically observed impairment in exercise performance in hot compared to cool conditions?

As described, it has been postulated that central fatigue, in which the hot brain reduces skeletal muscle recruitment (measured using IEMG), explains the observed impairment in performance (Nybo & Nielsen 2001a; Nybo & Nielsen 2001b; Rasmussen *et al.* 2004). However, this model is catastrophic (Noakes & St Clair Gibson 2004), and does not allow for the self-selection of a lower power output in advance of thermoregulatory failure. Evidence exists that pacing strategy

is altered before core temperatures reach 'limiting' levels (Tattersson *et al.* 2000; Marino, 2004; Marino *et al.* 2004), and that muscle activation levels are reduced progressively across a range of body temperatures below the critical level (Cheung & Sleivert 2004; Morrison *et al.* 2004). However, this has not been demonstrated during dynamic exercise.

Question 2

Are the pacing strategy and skeletal muscle activation levels altered by the oxygen content of the inspired air?

To date, only Peltonen *et al.* (1997) have shown an effect of a change in $F_{I}O_2$ on skeletal muscle activation and performance. They showed that hypoxia reduced muscle activation levels and hence performance was impaired during rowing time-trials. Numerous studies have measured an increase in muscle activation levels at a fixed power output in hypoxia, suggesting that muscle force-producing capacity is impaired (Nummela *et al.* 1992; Taylor *et al.* 1997). This model is also catastrophic, and is challenged by the findings of Kayser (1994), who showed that muscle activation, measured using EMG, could increase at the point of fatigue, if subjects were given hyperoxic air to breathe. Therefore, I aimed to establish whether self-paced exercise performance would be improved by hyperoxic breathing, and whether this was associated with an increase in skeletal muscle activation levels.

Question 3

Does the subjective Rating of Perceived Exertion (RPE) play a role in the regulation of exercise in advance of physiological failure?

Studies have shown that the RPE is linearly correlated to increases in core body temperature during exercise in hot and cool conditions, and to the increase in the α -to- β ratio of the EEG signal, suggesting reduced arousal levels (Nielsen *et al.* 2001; Nybo & Nielsen 2001b; Rasmussen *et al.* 2004). During self-paced exercise in hot conditions, any increase in RPE (or reduction in arousal) caused by a progressive increase in body temperature could enforce a reduction in the self-selected workrate before the attainment of a critically high temperature causes complete failure to continue exercise. Therefore, the RPE may play an important mediatory role, and I aimed to examine this role using a novel method of exercise, in which the RPE is 'clamped' throughout exercise in hot conditions.

Question 4

Is the activation of skeletal muscle maximal at maximal volitional effort?

A premise of the theory that exercise is regulated in an anticipatory fashion is that the activation of skeletal muscle is regulated throughout the exercise bout to ensure that physiological derangements do not become harmful or limiting. Therefore, muscle activation is not maximal during exercise, even at maximal volitional effort. I aimed to examine this hypothesis by measuring IEMG activity during progressive cycling tests to exhaustion and during sprint cycling at supramaximal power outputs.

Question 5

How are world record track event performances paced? Are there differences in the pacing strategies during world record performances in the shorter events (800 m) compared with long distance events (mile, 5 000 m and 10 000 m events)?

In laboratory studies, pacing strategies are characterized by a fast start, a period of reduced power output or running speed during the middle part of a trial, and a significant increase in power output at the end of the trial (Albertus *et al.* 2005).

However, when shorter duration exercise (< 120 seconds) is performed, the increase in power output at the end is absent, and power output declines progressively (Foster *et al.* 1993; Ferro *et al.* 2001). I examined world record performances in track athletics to establish how elite athletes paced themselves, and to identify factors that may account for any differences in pacing strategy between shorter and longer events.

Finally, based on the studies of this thesis, and the research reviewed in the present chapter, a model for the regulation of exercise by a complex, anticipatory system is proposed.

Chapter 2

Impaired exercise performance in the heat is associated with an anticipatory reduction in skeletal muscle activation

The research findings from this chapter have been published as an Original Investigation in the *European Journal of Physiology (Pflügers Arch)* **448**: 422-430, 2004.

Introduction

Exercise performance is impaired during both self-paced (Marino *et al.* 2000; Tatterson *et al.* 2000; Marino *et al.* 2004) and externally regulated (Febbraio *et al.* 1994; Galloway & Maughan 1997; Parkin *et al.* 1999; Nybo & Nielsen 2001b) exercise performance in the heat. The biological mechanisms explaining this impairment are however poorly understood. Originally it was believed that an increase in the oxygen-independent contribution to energy production (Brown *et al.* 1982), resulting from a reduction in skeletal muscle blood flow (Rowell *et al.* 1966; Fink & Costill 1975) secondary to reduced stroke volume and cardiac output (Rowell *et al.* 1966), explained this phenomenon.

However, it is now known that fatigue during exercise in the heat is not caused by reductions in cardiac output or exercising muscle blood flow, or by impaired substrate availability or utilization, or by the accumulation of lactate or K^+ (Savard *et al.* 1988; Nielsen *et al.* 1990; Nielsen *et al.* 1993; Gonzalez-Alonso *et al.* 1999).

Furthermore, it is known that fatigue occurs at a core temperature of approximately 40°C (Galloway & Maughan 1997; Nybo & Nielsen 2001a), irrespective of the rate of heat storage, the pre-exercise core temperature (Gonzalez-Alonso *et al.* 1999), or the extent of prior heat acclimatization (Nielsen *et al.* 1993; Nielsen *et al.* 1997). It has thus been proposed that fatigue during exercise in the heat is associated with a “critical core temperature limiting exercise performance” (Gonzalez-Alonso *et al.* 1999), in which a high body

temperature directly affects central nervous functions (Nielsen *et al.* 1990; Nybo & Nielsen 2001a).

Recently, Nybo and Nielsen (2001a) showed that force production and voluntary activation percentage in the exercised muscle groups (knee extensors) were lower during a sustained isometric maximal voluntary contraction (MVC) following cycle exercise in hot (40°C, sufficient to raise body temperature to 40°C) than in temperate (18°C, final core temperature 38°C) conditions. Significantly, the overall force produced when electrical stimulation was superimposed upon voluntary contraction was unchanged from values measured during the temperate trial. This indicates that the force-generating capacity of the exercised muscle is unaffected by the elevated core and muscle temperatures after exercise in the heat.

It was concluded that exercised-induced hyperthermia caused a form of “central fatigue”, in which elevated body temperature (> 40°C) caused reduced central activation in the exercised muscles leading to a lower force production. In support of the proposed role for the brain in fatigue, Todd *et al.* (2005) recently found that reduced force output during passive heating occurred due to a failure of voluntary drive despite the availability of additional motor cortical output, which would, in theory, allow increased force output. It was concluded that fatigue during hyperthermia was the result of a failure of the motor cortex and the muscle (Todd *et al.* 2005).

A key factor in interpreting the results of these studies (Nielsen *et al.* 1990; Gonzalez-Alonso *et al.* 1999; Nybo & Nielsen 2001a) is that the workrate is externally regulated and not free to vary during exercise. Under these conditions, fatigue is the consequence of excessive heat accumulation which ultimately forces the termination of exercise, or a failure to maintain the required force output (Nielsen, 1996; Nybo & Nielsen 2001a). In contrast, recent studies have shown that isometric force production and voluntary activation percentage decrease progressively during a passive heating protocol (Morrison *et al.* 2004), even at body temperatures below 39°C. It has also been found that running speed (Marino *et al.* 2004) and cycling power output (Tatterson *et al.* 2000) are reduced in hot conditions before any differences in core temperatures are measured compared to cool conditions. Based on these studies, it has been suggested that when exercise is self-paced, the brain acts pre-emptively to reduce work output specifically to prevent body temperatures from rising to reach critical or limiting levels (Marino *et al.* 2004). A pacing strategy is thus regulated by the brain in anticipation of a potentially limiting hyperthermia. This theory has yet to be tested during dynamic exercise.

Accordingly, the aim of this study was to investigate whether centrally-regulated recruitment of skeletal muscle motor units is altered during dynamic exercise in hot (35°C, HOT) compared to cool (15°C, COOL) environments. To evaluate this effect during, as opposed to after the completion of exercise, cyclists were studied during a self-paced 20-km laboratory cycling time-trial in which they

received no verbal or visual feedback other than the distance covered (every km). It has previously been shown that this form of testing produces pacing strategies during exercise that are highly reproducible when the testing conditions are identical (Schabert *et al.* 1998).

It was hypothesized that in order to prevent core temperatures from reaching harmful levels during exercise, subjects would subconsciously select a lower power output soon after the start of the time-trial in the HOT compared with the COOL environment, when core temperatures were still significantly lower than levels associated with bodily harm or diminished central drive in previous studies (Nielsen *et al.* 1990; Gonzalez-Alonso *et al.* 1999). Furthermore, it was hypothesized that electromyographic (IEMG) activity in the exercising muscle would be lower in the HOT than in the COOL condition. It has been shown that within an individual, IEMG activity is roughly proportional to the number and diameter of active muscle fibres (Bigland-Ritchie, 1981; Häkkinen, 1993), and IEMG measurements during exercise therefore allow insight into the degree of muscle recruitment and muscle recruitment patterns. A reduction in IEMG activity and power output early on in the hot condition, before rectal temperatures increase to potentially harmful levels would indicate that skeletal muscle recruitment and power output are down-regulated in advance of thermoregulatory failure. This contrasts with the prevailing hypothesis of fatigue in the heat, which predicts that “central fatigue” develops only after the homeostatic regulation of body temperature has failed and a critical level of hyperthermia is reached.

Methods

Subjects

Ten male cyclists were recruited from local cycling clubs and gymnasias to participate in the study. All subjects were physically active and were fully informed of the risks associated with the study. Subjects signed an informed consent before participating in the study, and upon completion of the trials, were remunerated for their participation. The study was approved by the Research and Ethics Committee of the Faculty of Health Sciences of the University of Cape Town. The mean age, height, mass and peak power output of the subjects were 24.7 ± 4.6 years (mean \pm S.D.), 176.2 ± 6.5 cm, 72.4 ± 8.6 kg, and 376 ± 47 W respectively.

Preliminary testing

Subjects reported to the laboratory for preliminary testing consisting of anthropometric measurements and to perform a peak power output trial. Stature (cm) and body mass (kg) were recorded using a precision stadiometer and balance (Model 770, Seca, Bonn, Germany). Percent body fat was calculated using the equations of Durnin & Womersley (1974) from skinfold measurements taken at seven sites (Ross & Marfell-Jones 1992). Lean thigh volume for each subject was

calculated according to the method of Katch & Katch (1974), based on the assumption that the thigh is a truncated cone.

Peak power output (PPO) was determined on a Kingcycle ergometer (described later) using a modified protocol as described by Hawley & Noakes (1992).

Subjects performed a self-paced warm-up for 10 minutes before beginning the test at a starting power output of 3 W/kg body weight. The workload was increased by 20 W per minute until exhaustion. Subjects were required to match a continuously increasing power output displayed in analogue form on the computer monitor. The test was terminated when the subject was unable to match the required power output. PPO was recorded as the highest mean power output achieved over a period of one minute during the test. Subjects were requested to remain in a seated position throughout the test.

Kingcycle ergometry system

All trials were conducted on a Kingcycle ergometer (Kingcycle Ltd., High Wycombe, U.K.), which allows the subjects to ride their own bicycles in the laboratory. After removing the front wheel, the cycle was firmly secured to the ergometry system by the front fork and supported by an adjustable pillar under the bottom bracket. The bottom bracket support was used to position the rolling resistance of the rear wheel correctly on an air-braked flywheel. A photo-optic sensor monitored the velocity of the flywheel in revolutions per second (RPS),

from which an IBM-compatible computer calculated the power output (W) that would be generated by a cyclist riding at that speed on a level terrain, using the following equation:

$$W = 0.000136 \text{ RPS}^2 + 1.09 \text{ RPS}$$

The Kingcycle was calibrated before both the peak power output test and the 20 km time-trials. For the calibration, subjects were required to accelerate to a power output of 220 W while seated in their normal cycling position. Once they reached this power output, subjects were instructed to stop pedaling immediately and remain in their riding position. The bottom bracket support was then adjusted until the computer display indicated that the slowing of the flywheel matched a pre-determined reference power decay curve.

Familiarization trial

Within one week of the PPO determination, subjects reported to the laboratory for a familiarization trial, during which they became accustomed to the equipment and laboratory conditions for the remaining two trials. Subjects completed a familiarization 20 km time-trial at an ambient temperature of 20°C, relative humidity of 60% and wind velocity of 10 km/h. Subjects were able to drink water ad libitum during the trial. All conditions and procedures were identical to those used in subsequent experimental trials.

Experimental protocol

Within one week, subjects reported to the laboratory for the experimental trials, which were conducted in an environmental chamber (Scientific Technology Corporation, Cape Town, South Africa). Each subject performed two experimental 20 km time-trials, a trial at 35°C (HOT) and a trial at 15°C (COOL). Relative humidity was 60% and wind speed 10 km/h for both conditions. Five subjects performed the HOT trial first while five performed the COOL trial first. For each subject, trials were conducted at the same time of day so that the effect of circadian variation could be minimized. Trials were separated by between 4 and 7 days in all subjects to allow sufficient recovery. It was also assumed that subjects were not naturally heat acclimatized as the experiments were conducted between the months of July and October, at which time the outside air temperature ranged from 12 to 25°C. Subjects were requested not to modify their training for the duration of their involvement in the trial, and to refrain from heavy physical exercise the day before the trial. During trials, subjects were allowed to drink water ad libitum. The only feedback given to subjects during the trials was the elapsed distance at the completion of each kilometer.

Maximal voluntary contraction testing

Prior to each 20 km time-trial, subjects performed a maximal voluntary contraction (MVC) for normalization of the EMG signal obtained during the

subsequent trial. Subject's right knee extensor strength was measured on an isokinetic dynamometer (Kin-Com, Chattanooga Group Inc., USA), while the electromyographic (EMG) activity of the vastus lateralis muscle was recorded. Subjects sat on the dynamometer with their arms folded across their chest, and their hips, thighs and upper bodies were firmly strapped to the seat. In this position, the hip angle was 100° flexion. The right leg was then attached to the arm of the dynamometer at a level slightly above the lateral malleolus and the axis of rotation of the arm was aligned with the lateral femoral condyle. The arm was then set so that the knee was at a 60° angle from full leg extension. Each subject then performed four sub-maximal familiarization contractions prior to performing four five-second maximal contractions, separated by 5-second recovery periods. Subjects were verbally encouraged to exert the maximal possible force during each contraction. The contraction producing the highest force was recorded and used for normalization of the EMG signal obtained during the 20 km time-trial.

EMG testing

During each MVC and subsequent 20 km time-trial, the EMG activity of the vastus lateralis muscle was recorded. Before placement of the electrode, the skin was shaved and cleaned with 95% ethanol, according to methods previously described (Kay *et al.* 2001; St Clair Gibson *et al.* 2001b). A triode electrode (Thought Technology, West Chazy, N.Y., USA) was placed over the muscle belly of the vastus lateralis and connected to a pre-amplifier. The electrode was firmly taped

to the skin using micropore tape, and a bandage (Flexwrap) was wrapped around the electrode to ensure that sweat interference was minimized. Outputs from the pre-amplifier were relayed to a Flexcomp/DSP EMG apparatus (Thought Technology USA) via a fiber optic cable and stored by an online computer. EMG signals were captured at 1984 Hz during the MVC and the time-trials. EMG activity was captured for five-second periods during the MVC. During the 20 km time-trials, EMG activity was measured at 1, 5, 10, 15 and 20 km. For analysis of the signal, five seconds of data were analyzed because subjects selected their own cadence while cycling.

The raw EMG signals were full wave rectified, movement artifact removed using a high-pass second order Butterworth filter with a cut off frequency of 15 Hz, then smoothed with a low-pass second order Butterworth filter with a cut-off frequency of 5 Hz. This was performed using MATLAB™ gait analysis software. This IEMG was used for subsequent analysis. All EMG data were normalized by dividing the EMG value obtained at each measurement point during the time-trials by the EMG value obtained during the MVC performed before the start of each time-trial. IEMG data were therefore expressed as a percentage of this MVC data. This method of EMG normalization have previously been shown to be reliable and valid for use in cycling trials, (Hunter *et al.* 2002b), and it has been found that the neuromuscular responses (IEMG) during self-paced cycling in the heat are reproducible between trials using this methodology (Kay *et al.* 2001). Because of technical problems with the EMG computer, a complete set of EMG

measurements could not be obtained during two of the time-trials and so EMG data from two subjects were rejected and the EMG data reported here represents the results from eight subjects.

Temperature measurements

Following MVC testing, subjects reported to the environmental chamber and inserted a rectal thermometer (YSI 409AC, Yellow Springs, OH, USA) 10 cm beyond their anal sphincter. Saltin and Hermansen have shown that the measurement of rectal temperature is as good an index core temperature during cycling at high work rates as oesophageal temperature (Saltin & Hermansen 1966). Four surface thermocouples (YSI 427, Yellow Springs, OH, USA) were securely taped to the sternum region, left mid-thigh, left calf and forehead for measurement of skin temperature. Three of these sites (sternum, mid-thigh and calf) are typically measured during temperature-related studies (Ramanathan, 1964), as they measure skin temperature over the heart (sternum thermocouple) and the working skeletal muscle (thigh and calf thermocouples). The forehead site was selected since it was speculated that the temperature of the head may be important in monitoring temperature and regulating exercise intensity, according to the current hypothesis.

Upon entering the chamber, the Kingcycle was calibrated as described, and subjects performed a self-paced two-minute warm-up. The duration of the warm-

up was restricted to two minutes to ensure that the initial values for heart rate, RPE and core temperature were not different between HOT and COOL conditions. The initial values of skin temperature, rectal temperature and heart rate were obtained at the completion of the warm-up. Temperatures were recorded at 1, 5, 10, 15 and 20 km during the trial using a digital telethermometer, accurate to 0.1°C (YSI 400 series).

Measurements of power output, heart rate and RPE

Power output during the time-trials was recorded by the Kingcycle equipment. To allow comparisons to be made between power outputs during trials of different duration, the recorded power output was normalized by dividing the trial into intervals of 5% of the total trial duration. Power output is thus reported as the average power output over each of these intervals of 5% total trial duration.

Heart rate was recorded at the start of the trial, and at 1, 5, 10, 15 and 20 km using a Polar Accurex NV heart rate monitor (Polar Electro OY, Kempele, Finland).

A rating of perceived exertion (RPE) was recorded at the start of the trial and at 1, 5, 10, 15 and 20 km, using the Borg category ratio scale (Borg, 1982).

Subjects recorded their nude body mass before each trial, and again after completion of the trial after wiping off sweat with a towel. The volume of water

ingested during each trial was also recorded. Rate of weight loss (in kg/hour) was estimated by the change in body mass adjusted for fluid consumption. This weight loss was considered a proxy for sweat rate, but was not corrected for other body weight losses caused by irreversible fuel oxidation, since it was assumed that such losses would be essentially similar in both trials.

Statistical analysis

Power outputs, EMG data, temperatures and heart rate data were analyzed using a two-way ANOVA for repeated measures, to examine the interaction of temperature and time. Where a significant effect was detected, post-hoc comparisons were made with a Tukey's HSD for pairwise comparisons.

Performance times, average power output, pre and post body weights and fluid ingestion were analyzed using a dependent t-test. For all analyses significance was accepted at $P < 0.05$. Data are presented as means \pm S.D.

Results

Time-trial performance and power outputs

The time taken to complete the 20-km trial was significantly greater in the HOT than in the COOL condition ($P < 0.001$) (Table 1). The average power output in

HOT was correspondingly lower; 255 ± 47 W compared to 272 ± 45 W in COOL ($P < 0.01$) (Table 1).

Table 1. Time and average power output during trials in HOT (35°C) and COOL (15°C) conditions.

	HOT	COOL
Time (min)	29.6 ± 1.9	28.8 ± 1.8 *
Average power (W)	255 ± 47	272 ± 45 †

Values are means \pm S.D. of ten subjects. * Significantly different from HOT ($P < 0.001$); † Significantly different from HOT ($P < 0.01$).

Fig. 1 shows the normalized power output recorded during the time-trials, at intervals of 5% of the total duration of the time-trial. Power outputs were identical for the first 30% of the trials in both conditions. In HOT, power output declined progressively from 30% to 80%, whereas in COOL, power output remained constant from 10% to 90% of the time-trial. The power output in HOT became significantly lower than in COOL at 80%, 90%, 95% and 100% of the total duration ($P < 0.05$). In both HOT and COOL conditions, the power output in the final 5% interval was significantly greater than in the previous intervals ($P < 0.05$). The highest power output over any 5% interval was recorded in the final 5% in both HOT and COOL conditions.

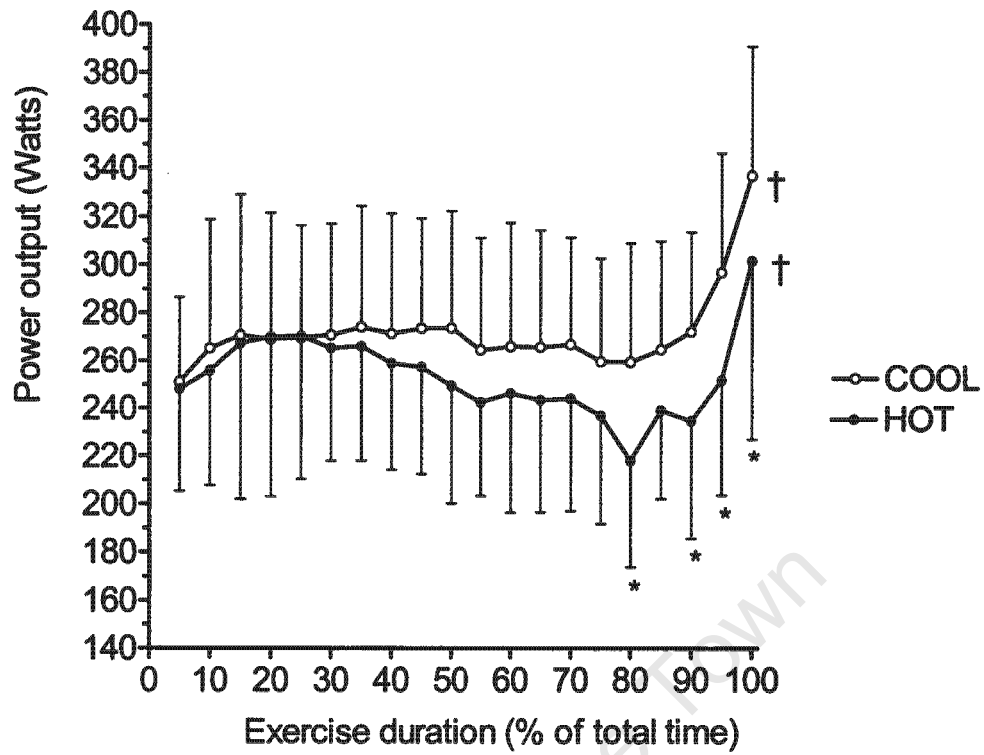


Figure 1. Power output (W) at intervals of 5% of total trial duration in HOT (35°C) and COOL (15°C).

Values are means \pm S.D. for ten subjects. * Significantly different from COOL ($P < 0.05$); † significantly different from preceding time intervals within the same environmental condition ($P < 0.05$)

Thermoregulatory responses

Rectal temperature increased significantly over time ($P < 0.001$) in both HOT and COOL conditions (Fig. 2). In HOT, the final rectal temperature was $39.2 \pm 0.6^\circ\text{C}$, compared to $38.8 \pm 0.4^\circ\text{C}$ in COOL ($P < 0.005$). At all other intervals, rectal temperatures were not significantly different between conditions.

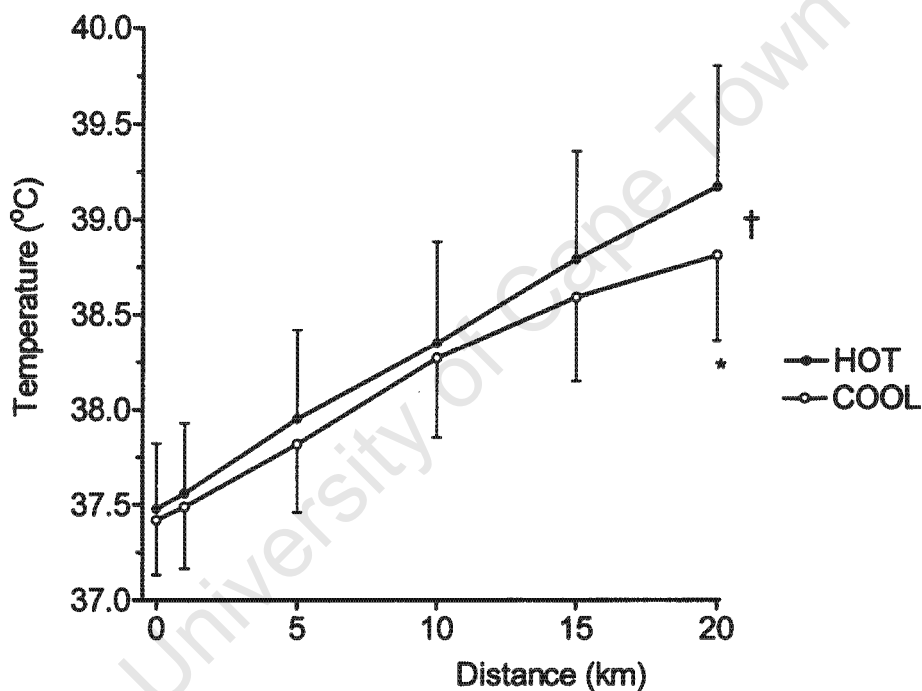


Figure 2. Rectal temperatures at 0, 1, 5, 10, 15 and 20 km during trials in HOT (35°C) and COOL (15°C) conditions.

Values are means \pm S.D for ten subjects. † Significant time main effect ($P < 0.001$); * significantly different from HOT ($P < 0.005$).

Fig. 3 shows the rate of increase in rectal temperature calculated at 2-km intervals. There were no significant differences in rate of increase in rectal temperature over any of the intervals. The average rate of rise in rectal temperature in HOT was

$0.085 \pm 0.030^{\circ}\text{C}/\text{km}$, compared to an average rate of increase in COOL of $0.070 \pm 0.017^{\circ}\text{C}/\text{km}$. These were not significantly different.

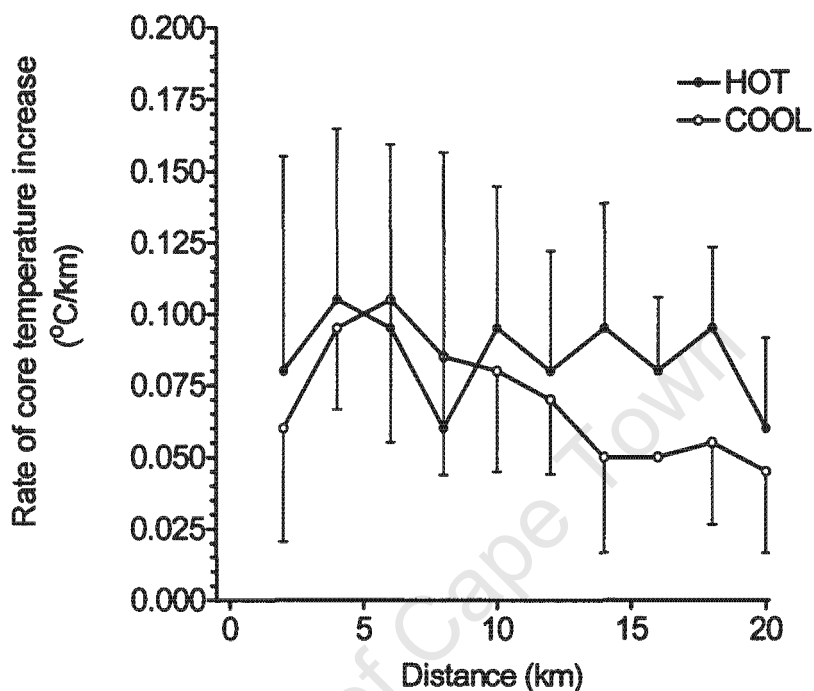


Figure 3. Rate of increase in rectal temperature ($^{\circ}\text{C km}^{-1}$) calculated at 2 km intervals during trials in HOT (35°C) and COOL (15°C) conditions

Values are means \pm S.D. for ten subjects.

All four skin temperatures (chest, thigh, calf, forehead) were significantly greater in HOT than in COOL conditions throughout the time-trials ($P < 0.001$) (Fig. 4. a - d).

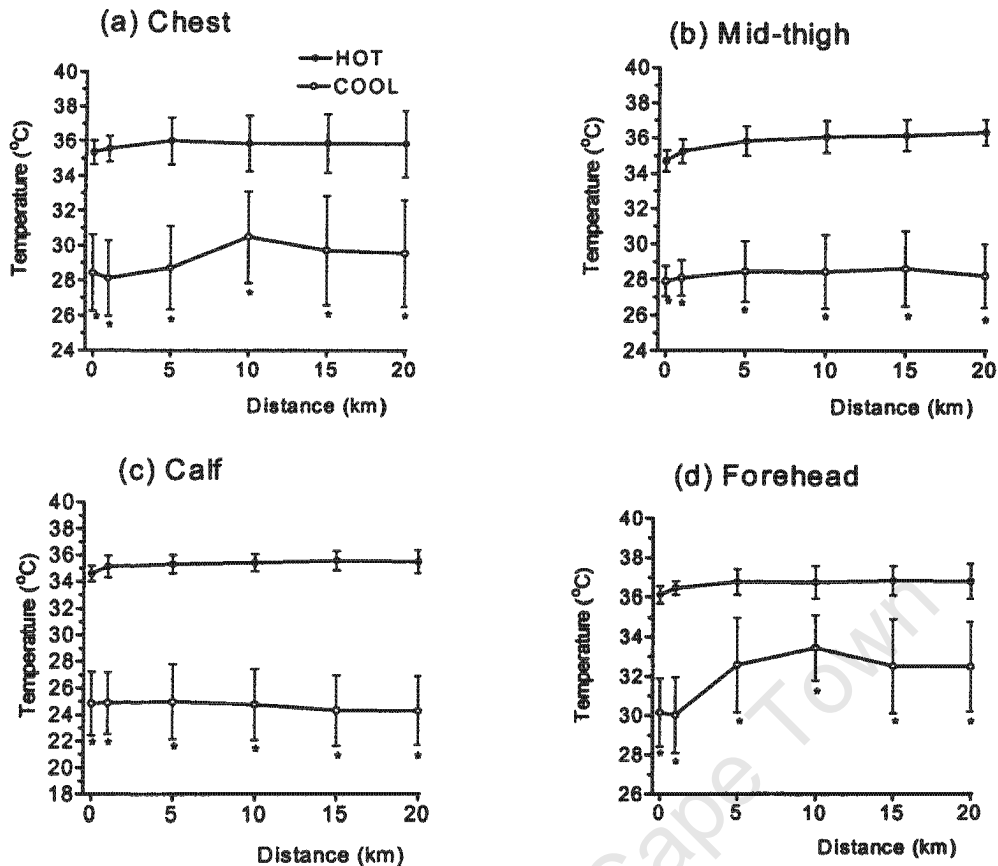


Figure 4 a - d. Skin temperatures over the chest (a), mid-thigh (b), calf (c) and forehead (d) at 0, 1, 5, 10, 15 and 20 km during trials in HOT (35°C) and COOL (15°C) conditions.

Values are means \pm S.D. for ten subjects. * Significantly different from HOT ($P < 0.001$)

IEMG amplitude

IEMG amplitude, expressed as a percentage of the IEMG amplitude during the MVC prior to each trial, is shown in Fig. 5. IEMG activity was lower in HOT than in COOL at 10 km and 20 km ($P < 0.05$). At 15 km, there was a tendency for IEMG to be lower in HOT than in COOL, but this was not significant ($P = 0.1$). IEMG

did not change during the HOT trial, whereas IEMG in COOL at 20 km was significantly greater than at 1, 5, 10 or 15 km ($P < 0.005$).

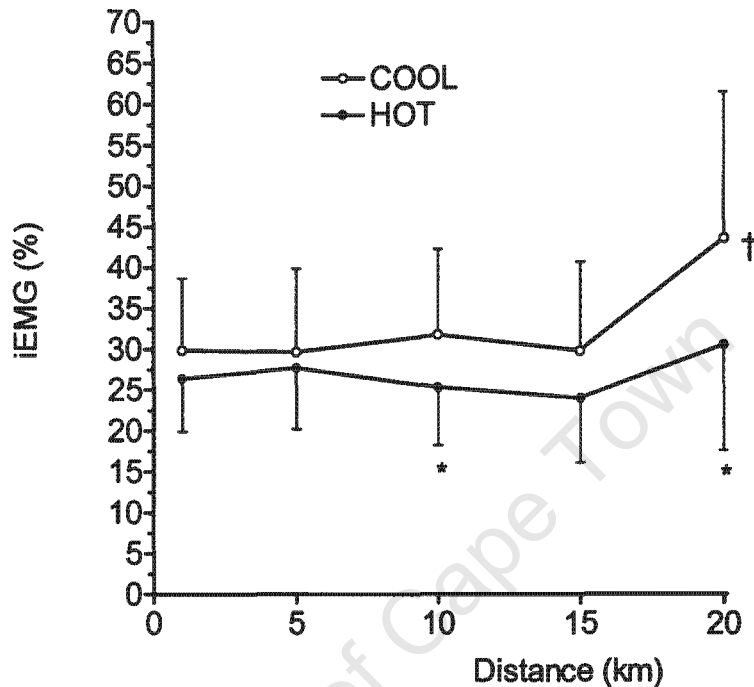


Figure 5. Integrated electromyogram (IEMG) at 1, 5, 10, 15 and 20 km during trials in HOT (35°C) and COOL (15°C) conditions.

Values are means \pm S.D. for eight subjects. * Significantly different from COOL ($P < 0.05$); † significantly different from other points during COOL.

Heart rates

Heart rate in both conditions increased similarly over time, and final heart rates were significantly greater than at rest ($P < 0.0001$) (Fig. 6). Final heart rates in HOT and COOL were 184 ± 8 and 181 ± 10 beats per minute respectively. These were not significantly different.

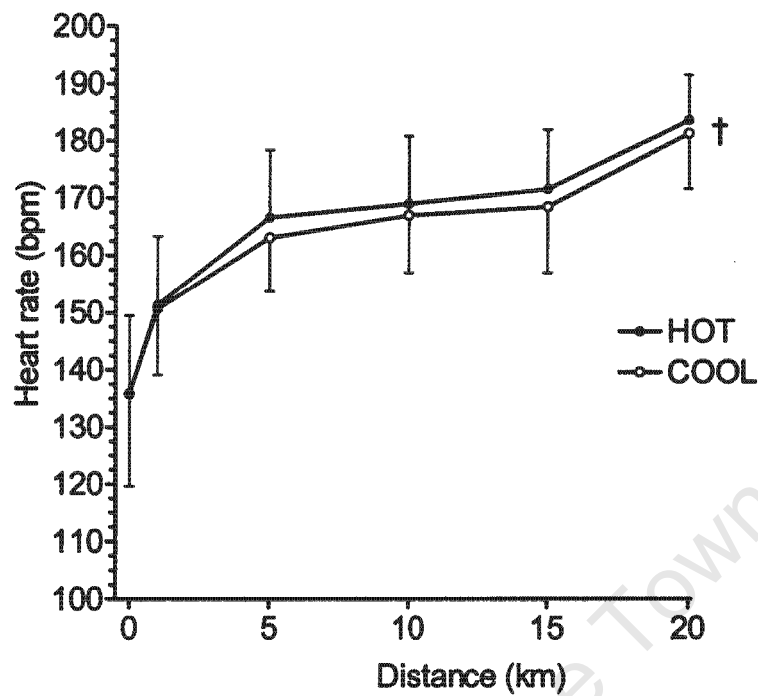


Figure 6. Heart rate at 0, 1, 5, 10, 15 and 20 km during trials in HOT (35°C) and COOL (15°C) conditions.

Values are means \pm S.D. for ten subjects. † Significant time main effect ($P < 0.0001$)

Ratings of perceived exertion

RPE increased significantly over time in both trials (Fig. 7), but were not significantly different between HOT and COOL. The final RPE in HOT and COOL were 9.0 ± 1.5 and 9.6 ± 1.2 respectively.

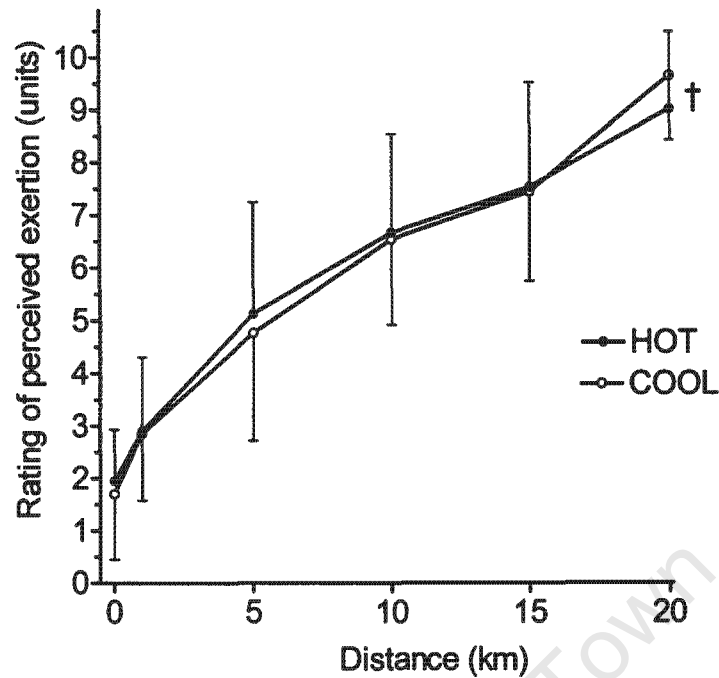


Figure 7. Ratings of perceived exertion (RPE) at 0, 1, 5, 10, 15 and 20 km during trials in HOT (35°C) and COOL (15°C) conditions

Values are means \pm S.D. for ten subjects. † Significant time main effect ($P < 0.0001$).

Fluid intake and weight loss

Body weight changes during the trials, total fluid intakes and rates of weight loss are presented in Table 2. There were no significant differences in pre- and post-trial body weights between conditions, or in changes in body weight during the trials. Fluid intake in HOT was significantly greater than in COOL ($P < 0.001$).

Total weight losses and rates of weight loss were not significantly different between conditions.

Table 2. Body weight, fluid intakes and weight loss during trials in HOT (35°C) and COOL (15°C) conditions

	HOT	COOL
Pre-trial body mass (kg)	72.3 ± 8.8	72.1 ± 8.7
Post-trial body mass (kg)	71.8 ± 8.8	71.5 ± 8.7
Change in body mass (kg)	0.4 ± 0.4	0.6 ± 0.4
Fluid intake (ml)	581 ± 171	244 ± 177 *
Rate of mass loss (kg/hour)	2.06 ± 0.58	1.73 ± 0.77

Values are means ± S.D. of ten subjects. * Significantly different from HOT ($P < 0.001$).

Discussion

Exercise-related fatigue attribute fatigue or exhaustion has been attributed to homeostatic failure in one or more organ systems that are crucial for sustained exercise performance (Rowell et al. 1966; Noakes, 2000). Accordingly, it was originally proposed that fatigue during exercise in the heat developed after a limitation in blood supply, oxygen delivery or fuel utilization had developed. It was subsequently shown that such homeostatic failure does not occur, since skeletal muscle blood flow and metabolism do not reach limiting values in the heat (Savard *et al.* 1988). More recently, it has been proposed that central (Bruck & Olschewski 1987; Nielsen *et al.* 1993) neural recruitment of skeletal muscle motor units is reduced when core body temperature rises to reach 'critical' levels (Nielsen *et al.* 1990; Nybo & Nielsen 2001a). Thus, according to this model, a

failure of body temperature regulation causes critical brain and/or core temperatures to be reached. Central fatigue then occurs, as the “hot brain” is no longer able to recruit a sufficient number of motor units to sustain the previous or expected power output.

To our knowledge, this is the first study to show that this central fatigue model, while still relevant when exercise is performed to exhaustion at a fixed workrate (Nybo & Nielsen 2001a), does not accurately explain self-paced exercise in the heat. This study has found that when self-paced exercise is performed in the heat, work output and skeletal muscle recruitment are down-regulated early during the trial, before body temperature is significantly elevated, with the result that thermal homeostasis is maintained similarly during exercise in both temperate and hot conditions. The critical findings that support this novel interpretation are the following:

First, despite marked differences in the environmental conditions in which the time-trials were performed, rectal temperatures were not different between HOT and COOL for the first 15 km of the 20 km time-trial (Fig. 2). Rectal temperatures were different at the end of the trials, but did not reach temperatures measured in heatstroke ($> 41^{\circ}\text{C}$) in either condition. Hence, relative thermal homeostasis was maintained during exercise, and the exercise bout was completed without dangerous levels of hyperthermia being reached.

Second, power output began to decline in the hot condition after only 30% of the total trial duration, and was significantly less than in the cool condition from 80% of the trial duration until the finish (Fig. 1). However, rectal temperatures (Fig. 2) were not different until the final kilometer during the trials, suggesting that the observed reduction in power output in the heat could not have been caused by a higher core temperature acting directly on the active skeletal muscles or the brain to cause fatigue, as has been hypothesized (Nybo & Nielsen 2001a). Indeed, the highest power output in both conditions was achieved during the final 5% of the time-trial, when core temperatures were at their highest. The core temperature during the final kilometer of the trial in the cool condition was in fact significantly greater than the core temperatures in the hot condition at 5 and 10 km, when the power output had begun to decline in that trial. Yet, power output was maintained throughout the cool trial, and increased by 20% during the final 10% of the trial (Fig. 1). Hence, an elevated core temperature cannot be the direct cause of the lower power outputs achieved in the hot than in the cool condition. Rather, it appears that power output is decreased in the heat in the absence of any thermal distress.

Third, IEMG activity was significantly reduced at 10 and 20 km in the hot condition. This indicates that the recruitment of motor units was decreased even when core temperatures were below 40°C. In fact, at 10 km, the core temperatures were remarkably similar between conditions ($38.4 \pm 0.5^\circ\text{C}$ vs. $38.3 \pm 0.4^\circ\text{C}$ in HOT and COOL respectively) (Fig. 2). Therefore, the reduced skeletal

muscle recruitment in the hot trial can not be explained by the direct effect of a “critical” core temperature producing “central fatigue” in the motor regions of the brain, as has previously been found at volitional exhaustion when the exercise workrate is imposed (Nielsen *et al.* 1990).

Rather, it is proposed that the early decline in power output in the absence of any thermoregulatory disturbance in the heat forms part of an anticipatory response in the brain, which mediates a reduction in skeletal muscle recruitment to ensure that the rate of heat production is reduced. This would allow relative thermal homeostasis to be maintained so that exercise can be safely completed without the development of premature fatigue or heat stroke, even in severe environmental conditions.

Indeed, the rate of increase in rectal temperature, a measure of the rate of heat storage, was not significantly different between the hot and cool conditions (Fig. 3). This finding is speculatively interpreted to indicate that the reductions in skeletal muscle recruitment and power output in the hot condition may have been mediated to ensure that similar rates of heat accumulation occurred in hot and cool conditions. If this is indeed the case, then the rate of heat storage in the body may provide crucial afferent sensory inputs to a central controller, which adjusts work rate and skeletal muscle motor unit recruitment during exercise in both hot and cool conditions.

Others have reported similar findings to our own. Tatterson et al. (2000) showed a reduction in power output after only fifteen minutes of a 30-minute self-paced cycling time-trial in the heat, even though core temperatures rose at similar rates in a hot and temperate environment. They postulated that the brain was sensitive to the rate of increase in arterial blood temperature, and selected a power output relative to the rate of rise in core temperature. However, they did not measure EMG activity to confirm this hypothesis.

A similar finding was reported by Marino *et al.* (2000), who showed that runners with a lower body mass outperformed heavier runners during a self-paced 8-km time-trial that followed 30 minutes of running at 70% peak treadmill speed in hot (35°C) conditions. A significant correlation was also found between the rate of heat storage and body mass during the time-trial, suggesting that the runners with a low body mass stored less heat at the same running speed. It was concluded that this reduced rate of heat storage allowed the lighter runners to run faster before reaching a limiting rectal temperature (Marino *et al.* 2000). This suggests that the rate of heat storage may contribute to the afferent input responsible for a reduction in work rate in the heat.

Subsequently, Marino et al. (2004) found that African runners, who have a lower rate of heat storage at a given running speed than Caucasian runners, were able to outperform the Caucasian runners in hot (35°C), but not in cool (15°C) conditions during an 8 km time-trial. The difference in running speed between the

groups in the heat was present from the onset of the time-trial, despite rectal temperatures which were only moderately elevated ($\sim 38^{\circ}\text{C}$) and not different between groups. It was suggested that the early reduction in running speed in the heat occurs due to an anticipatory exercise response which would “control the exercise work rate by regulating the number of motor units that are recruited or derecruited during prolonged exercise in the heat” (Marino *et al.* 2004).

These findings are compatible with the existence of a centrally-regulated pacing strategy that reduces motor command and exercise intensity specifically to prevent excessive heat storage. Presumably, a central controller calculates the optimum rate of heat production and hence heat storage that will allow the self-paced exercise bout to be completed without the development of a harmful level of hyperthermia (Fig. 2), and then regulates skeletal muscle motor unit recruitment to adjust the metabolic rate accordingly. Indeed, Kayser (2003) has proposed that the CNS integrates afferent signals from various sources including the heart, muscles, respiratory system and thermoreceptors, and adjusts motor command to protect the integrity of the organism during exercise. This general model for the role of the CNS during exercise is applicable to exercise in the heat.

If this theory is correct, it is not clear how the brain is able to detect the initial increased rate of heat storage in the hot condition. The skin temperatures at all four measured sites were significantly higher in the heat (Fig. 4). The afferent

sensory input from the thermoreceptors in the skin must form part of the integrated response which mediates the decreased central recruitment and power output in the heat. That is, a high skin temperature may inform the brain that the capacity for heat dissipation is reduced (Nielsen, 1996), and so heat production is reduced in order to prevent body temperature from rising too rapidly to harmful levels.

It has been found that the exercise-induced increase in muscle sympathetic nerve activity (MSNA) is augmented during muscle heating (Ray & Gracey 1997). The increase in MSNA was attributed to the stimulation of mechanically sensitive muscle afferents which were sensitized by heating (Ray & Gracey 1997). Muscle temperature was not measured directly in this study, though it can be expected that muscle temperature would be elevated to similar or greater levels than rectal temperature, based on previous studies of exercise in the heat (Saltin & Hermansen 1966). Thus, the sensitization of skeletal muscle afferents may play a role in increased signaling to the CNS during exercise in the heat (Ray & Gracey 1997). A 'central programmer', proposed by Ulmer (1996), would integrate this and other afferent signals arising from the muscle and peripheral organs and alter movement or force output to optimize performance. This integral control, termed teleoanticipation (Ulmer 1996), would regulate efferent commands based on the afferent feedback from the periphery and knowledge of the 'finishing points' (St Clair Gibson *et al.* 2006).

It must also be assumed that all the subjects had prior experience of exercise in the heat, so that any anticipatory reductions in power output may occur as part of a learned response.

The reductions in power output and IEMG activity were not associated with reductions in heart rate (Fig. 6) or ratings of perceived exertion (Fig. 7), which were similar at all points during the two trials. The final RPE values were near maximum in both conditions, indicating that subjects performed to their own maximal volitional capacity. It is also significant that power output and IEMG activity were reduced in the heat despite RPE values which were similar and well below maximal in hot and cool conditions. Therefore, the rating of perceived exertion or, alternatively, the conscious sensation of fatigue, does not merely track changes in power output, but is related to the central neural processes involved in the maintenance of, in this case, thermal homeostasis. It clearly makes no sense for the conscious perception of effort to be reduced at the same time that power output is decreasing (Fig. 1). The athlete's natural response to a falling RPE would be to consciously override this effect, thereby increasing the power output, the rate of heat production, and the probability that homeostatic failure would develop. Thus, for thermal homeostasis to be maintained, the central processes responsible for adjusting power output and muscle recruitment must simultaneously increase the conscious perception of exertion, in order to discourage any conscious overriding of this subconscious control.

It was also found that the power output in both the hot and cool conditions increased during the final 5% of the trial as previously reported by Tatterson et al. (2000) and Kay *et al.* (2001). This is a characteristic finding during self-paced exercise, with the increase at the end of exercise being termed the 'endspurt' (Baden *et al.* 2005). In the present study, the endspurt was associated with a significant increase in IEMG activity at the end of the cool, but not the hot trial. This difference was possibly due to methodological limitations. The IEMG activity was measured during the final 20 seconds of the time-trial, whereas power output was averaged over the final 5% of the trial, and thus reflects an average of the final few minutes of the trial. Another limitation of the present study is that the IEMG activity was measured in one muscle, and it is possible that altered skeletal muscle recruitment patterns in the heat, or the relatively small number of subjects may also explain the lack of a significant increase in IEMG activity in the heat.

Nevertheless, the increased IEMG activity in the cool condition indicates that the athletes were able to increase motor unit recruitment at the end of exercise, despite core temperatures, heart rates and RPE values which were significantly higher than at the start of the trial. This suggests that the afferent inputs which mediate the initial decreases in recruitment and power output can be consciously overridden in the maximal effort at the end of the trial, and supports the existence of a system which adjusts exercise performance by altering efferent motor command during exercise (Ulmer, 1996). Such a system would ensure that under exercise conditions, a skeletal muscle reserve is maintained. Indeed, it was found

that the measured EMG activity increased by approximately 40% and 50% in the final kilometer of the trials in HOT and COOL conditions respectively (Fig. 5), suggesting that muscle activation levels were submaximal in the earlier, preceding kilometers. Hence, a cardinal feature of prolonged exercise is the presence of motor unit recruitment reserve; a feature which is not always recognized (Noakes *et al.* 2001).

In conclusion, the pacing strategy adopted during a self-paced 20 km time trial was different in hot compared to cool conditions. Power output began to fall within the first 30% of a maximal self-paced time-trial in the heat, reaching significance after 80% of the trial had been completed. Skeletal muscle IEMG activity was also reduced from 10 km during the trial in the hot condition. These decreases were not associated with altered rectal temperature, heart rate or perception of effort compared with exercise in the cool, and occurred well before rectal temperature reached 40°C. It is proposed that this response occurs as part of a centrally controlled neural mechanism, which anticipates an abnormal elevation in body temperature, and alters skeletal muscle recruitment to allow completion of the exercise bout whilst thermal homeostasis is maintained.

Impaired exercise performance in the heat is thus not the result of a limiting core temperature, but occurs as part of the central regulation of pacing strategy and skeletal muscle recruitment, which controls the rate of heat storage, thereby

preventing the development of thermoregulatory derangements during self-paced exercise in the heat.

University of Cape Town

Chapter 3

***Hyperoxia improves 20 km cycling
time trial performance by increasing
muscle activation levels while
perceived exertion stays the same***

Introduction

Contrary to explosive type efforts, endurance exercise performance is influenced by the pressure of oxygen in the inspired air. A decrease in oxygen tension (hypoxia) leads to impaired performance (Steinacker *et al.* 1986; Taylor *et al.* 1997; Wilber *et al.* 2003) and an increase (hyperoxia) to an improvement in performance compared to normoxia (Peltonen *et al.* 1995; Peltonen *et al.* 1997).

A generally accepted explanation for this dependence of endurance performance on inspired oxygen tension is based on a model that holds that endurance type activity relies almost essentially on aerobic metabolism. Since aerobic metabolism depends on pressure gradient-dependent oxygen transport from the inspired air to the site of oxidation in the mitochondria of the contracting muscles, a decrease in oxygen flux would impose a decrease in oxygen use and therefore a decrease in sustainable aerobic metabolism, greater dependence on anaerobic metabolism and earlier development of muscle fatigue. For example, Taylor *et al.* (1997) reported that the integrated EMG activity (IEMG), an indirect measure of skeletal muscle recruitment (Häkkinen, 1993), of one of the active muscles was greater during sub-maximal cycling at a fixed power output in hypoxic (F_{iO_2} 11.6%) than in normoxic conditions, suggesting that the force-generating capacity of the muscle was impaired in hypoxia, so that an increase in muscle activation was required to maintain power output (Taylor *et al.* 1997).

Conversely, endurance exercise performance is improved in hyperoxia (Steinacker *et al.* 1986). This increase in performance has been attributed to a hyperoxia-dependent increase in oxygen consumption (VO_2) and associated aerobic ATP energy production (Wilber *et al.* 2003), and to a protective effect of hyperoxia on the contractile properties of the muscle as a result of reduced metabolic acidosis and lactate production (Linossier *et al.* 2000).

Recently, it has been suggested that oxygen-induced changes in metabolism and muscle function may not provide the sole explanation for improved performances in hyperoxia and impaired performances in hypoxia. For example, Kayser *et al.* (1994) found that when subjects were given hyperoxic air at the point of volitional exhaustion during cycling exercise in hypoxic conditions, while at the same moment the exercise load was increased, the subjects were able to continue cycling at the higher power output. The continuation of exercise was associated with an immediate increase in the IEMG activity in the one active muscle that was studied (vastus lateralis). This suggested that volitional fatigue in hypoxia occurred at a submaximal level of skeletal muscle activation, and that the activation of skeletal muscle was influenced by the $F_{\text{I}}\text{O}_2$. It was concluded that under these conditions, the limiting factor to endurance exercise performance was not the development of muscle fatigue (Taylor *et al.* 1997), but rather the alterations in the motor drive to the active muscle (Kayser *et al.* 1994).

During self-paced exercise, alterations in performance in hypoxia and hyperoxia are associated with changes in the pacing strategy (Peltonen *et al.* 1997). Peltonen *et al.* (1997) found that the force output of maximal rowing strokes was maintained throughout hyperoxic trials, but decreased progressively during trials in normoxia and hypoxia. This finding could be attributed to a progressive reduction in the force generating ability of the muscle in hypoxia and normoxia, as has been proposed (Taylor *et al.* 1997; Linossier *et al.* 2000). However, Peltonen *et al.* (1997) found that the IEMG activity of the active muscles was lower during trials in hypoxia than in normoxia, indicating that performance in hypoxia was regulated, at least in part, by a centrally-mediated reduction in skeletal muscle activation. Thus, it has been suggested that during self-paced exercise, changes in oxygen content exert effects on performance by altering centrally regulated muscle activation levels (Gandevia, 2001).

In Chapter 2 of the present thesis, it was shown that during a self-paced 20 km cycling time-trial, power output and IEMG activity are reduced well before body temperatures are different between hot and cool conditions. It was proposed that the central nervous system regulates the pacing strategy by determining sustainable muscle activation levels (Noakes & St Clair Gibson 2004) in anticipation of changes in body temperature (Chapter 2). It has also been suggested that changes in pacing strategy are mediated in advance of potentially harmful or limiting changes in energetic resources and metabolite accumulation (Foster *et al.* 2003). Thus, when exercise is performed in hyperoxia, it may be

that altered pacing strategies (Peltonen *et al.* 1997) are the result of a protective effect of hyperoxia on muscle contractility (Taylor *et al.* 1997; Linossier *et al.* 2000), as well as a neural control strategy which monitors intracellular changes such as metabolite accumulation (Diamant *et al.* 1968; Karlsson & Saltin 1970; Jacobs & Kaiser 1982; Jacobs *et al.* 1983) or phosphagen depletion (McLester, Jr., 1997), and then mediates anticipatory adjustments in the degree of skeletal muscle activation and power output in order to prevent these changes from becoming critical or harmful (Ingen Schenau *et al.* 1991; Foster *et al.* 2003; Noakes & St Clair Gibson 2004; Lambert *et al.* 2005). This hypothesis has yet to be tested.

Accordingly, the present study first examined the influence of hyperoxia on pacing strategy and skeletal muscle activation during self-paced cycling time trials in normoxic and hyperoxic air. It was hypothesized that exercise performance during a self-paced 20-km cycling time-trial would be improved in hyperoxia (F_{iO_2} 0.40) compared to normoxia (F_{iO_2} 0.21), as a result of an altered pacing strategy and an increase in IEMG amplitude in the muscles responsible for force generation (i.e. quadriceps). It was further hypothesized that the adjustments in power output and IEMG activity would occur soon after the onset of exercise, as part of an anticipatory regulatory process, as was proposed in Chapter 2 of the present thesis.

The effects of cycling at a constant workrate in normoxia and hyperoxia on IEMG activity and physiological measurements were also examined. This was performed

to allow comparisons to be made between the measured physiological variables in hyperoxia and normoxia when exercise workrate was free to vary versus when workrate was imposed. The aim of this design was to attempt to understand how any differences in pacing strategy between hyperoxia and normoxia may be mediated, as hypothesized in the first part of the study.

Methods

Subject selection

Eleven well trained male subjects (Age 24 ± 3 years, height 177 ± 7 cm, mass 72 ± 7 kg and peak power output 395 ± 33 W) were recruited on the basis of performance in local cycling races and in previous laboratory studies. Athletes who were used to cycle racing were purposefully recruited in order to study pacing strategies during time trial-like challenges. Subjects were fully informed of the risks associated with the study and gave written informed consent before participation. The study was in accordance with the declaration of Helsinki and was approved by the Research and Ethics Committee of the Faculty of Health Sciences of the University of Cape Town.

Preliminary testing

After recording stature and body mass using a precision stadiometer and balance (Model 770, Seca, Bonn, Germany), subjects performed an incremental peak power output (PPO) test. All exercise trials were conducted on a Kingcycle ergometer (Kingcycle Ltd., High Wycombe, U.K.), which allows the subjects to ride their own bicycles in the laboratory. After completing a self-paced warmup, the test began at a power output of 2.5 W/kg body weight. The workload was increased by 20 W per minute until exhaustion. Subjects were required to match a continuously increasing power output displayed in analogue form on the computer monitor by increasing the speed of cycling through the changing of gears and pedal rate. The test was terminated when the subject was unable to match the required power output. PPO was recorded as the average power output achieved over the final of minute of the test.

Three to five days after the PPO determination, subjects performed a self-paced 20 km time-trial to become accustomed to the equipment and procedures during the actual experimental testing.

Experimental Exercise trials

All subjects first performed two 20-km time-trials: one time-trial in normoxia (NORM-TT, $F_iO_2 = 0.21$) and one time-trial in hyperoxia (HI-TT, $F_iO_2 = 0.40$).

The F_iO_2 of 40% was selected since ethical and safety requirements limited the duration and exposure of subjects to high oxygen contents. Upon completing the time-trials, eight of the eleven subjects performed cycling trials to fatigue at a constant workload in both normoxia (NORM-CW) and hyperoxia (HI-CW). These trials were performed at a constant power output which was set equal to each subject's average power output during their best 20 km time-trial performance. The power output during constant workload trials was thus similar in HI and NORM. Subjects were instructed to ride for as long as possible at the required power output.

Control of inspired oxygen tension

All trials were performed inside a Multi-place Class "A" 18 000 L hyperbaric chamber (National Hyperbarics (Pty.) Ltd., Hull, U.K.), of length 3.5 m and diameter 2.5 m built to Lloyd's and ASME 1 PVHO specifications. The oxygen concentration of the air was continuously monitored using an oxygen sensor (Oxa 001, Scottish Anglo Environmental Protection Ltd.) placed at the level of the subject's head while in the riding position. When the F_iO_2 decreased 1.0% below the required percentage, 100% oxygen was fed into the chamber until the required F_iO_2 was achieved. Temperature and humidity were maintained between 19 and 21°C and 60 to 70% respectively during all trials, and pressure was maintained at sea level values for all trials.

The order of testing was randomized so that six subjects performed NORM-TT first and five subjects performed HI-TT first. The order of the constant workload trials, NORM-CW and HI-CW, was also randomized. Subjects were blinded to the oxygen content of the air and were not informed of the hypothesis of the study. Trials were separated by a minimum of three days to allow recovery between trials, and subjects refrained from heavy physical exercise the day before trials and maintained their training for the duration of their involvement in the study.

Maximal voluntary contraction and EMG activity

Prior to each experimental trial, subjects performed two maximum voluntary contractions (MVCs) to allow normalization of the EMG signal obtained during the subsequent cycling bouts as previously described (Tucker *et al.* 2004). Briefly, the electromyogram (EMG) activity of the vastus lateralis muscle was recorded during maximal isometric contractions using an isokinetic dynamometer (Bio-Dex dynamometer, Bio-Dex, United Kingdom). The skin over the muscle was shaved, abraded and cleaned with ethanol. Two electrodes (Medicotest A/S, Denmark) were placed over the belly of the muscle with an inter-electrode distance of 20 mm and were connected to a transmitter which relayed a telemetric signal to an antenna connected to an online computer (MyoResearch 2.02, Noraxon, USA) on which raw data were captured at 2000 Hz and stored for subsequent processing. Subjects were firmly strapped into the dynamometer and the right leg attached to

the arm of the dynamometer at a level slightly above the lateral malleolus. The arm was set so that the knee was at a 60° angle from full leg extension. Subjects then performed two isometric knee extensions and the EMG activity from the contraction producing the highest force was used for normalization of the EMG signal obtained during the subsequent cycling trials.

Normalising each subject's EMG activity relative to their own EMG signal before the trial reduces the effect of variables such as electrode positioning, skin impedance and differences in distribution of body fat, allowing comparisons to be made between conditions and subjects. This method was reported to be reliable for normalization of the EMG signal obtained both during maximal (Hunter *et al.* 2002b; Hunter *et al.* 2003) and sub-maximal cycling exercise (St Clair Gibson *et al.* 2001b; Tucker *et al.* 2004).

During the cycling trials, EMG activity was measured for a five second period every 2 km. EMG was measured over a set time interval so that changes in pedal rate could be reflected in the IEMG, since at higher rates; a greater number of contractions would be included in this five-second measurement period. For subsequent analysis of the raw EMG signal, the root mean square (RMS) was calculated over 50 ms periods and the signal was filtered using a 10-200 Hz filter. The IEMG amplitude was calculated using the MyoResearch software (MyoResearch 2.02, Noraxon, USA) and the value obtained from five seconds of cycling at each measurement point was divided by the value obtained during the

MVC. IEMG thus measured is independent of pedal rate within a range of 80-100 rpm (references) and therefore the freely chosen pedal rates used by the subjects were monitored to ensure that any differences in EMG activity between conditions could not be attributed to changes in cadence.

Due to technical problems with the EMG measurement computer, EMG data for one subject were not captured during the constant workload trials. The EMG data are thus presented for ten subjects during the time-trials and for seven subjects during the constant workload trials.

Plasma lactate concentration

A 20-G cannula (Nissho Corporation, Zaventem, Belgium) was inserted into a forearm vein prior to each trial for blood sampling every 2 km during the trials. Blood was later analysed for plasma lactate concentrations. Samples for analysis were stored on ice until centrifuged at 3000 g for 10 min at 4°C and the plasma stored at -20°C. Plasma lactate concentrations were determined by the spectrophotometric (Beckman Spectrophotometer – M35) enzymatic assay method using a lactate kit (Lactate PAP, bioMérieux Kit, Marcey l'Etoile, France). Blood samples could not be obtained from 2 subjects during time-trials, and so lactate concentrations are thus reported as means \pm S.D for nine subjects.

Power output and Performance

During the trials, the power output was recorded every 200 m and used to calculate average power output for two kilometre intervals. Performance was defined as the time taken to complete the time trial, and the distance covered during the constant load trial.

Heart rate

Average heart rate for each kilometre was measured using a Polar S410 heart rate monitor (Polar Electro OY, Kempele, Finland), and averaged for each two kilometre interval.

Perceived exertion

A rating of perceived exertion (RPE) was recorded every two kilometers using the Borg category-ratio scale (Borg, 1982). This scale (0-10) was explained to the subjects on their first visit to the laboratory, during the familiarization session and prior to each trial. Upon a prompt from an experimenter the subjects would look at the scale and indicate the perceived level of exertion which was then recorded.

Statistical analyses

Power output, IEMG amplitude, heart rate, plasma lactate concentrations and ratings of perceived exertion were analysed using a two-way analysis of variance (ANOVA) to examine the interaction of oxygen concentration and time. Where a significant effect was detected, post-hoc comparisons were made using a Tukey's HSD for pairwise comparisons. Performance times and average power output were analysed using a student's paired T-test. For the constant workrate trials, subjects did not all complete equal distances before volitional fatigue, and so data were analysed up to 14 km, which is the shortest distance completed by all nine subjects, as well as the final measurement taken at volitional fatigue. For all analyses significance was accepted at $P < 0.05$. Data are presented as means \pm S.D. for eleven subjects during time-trials, and for eight subjects during constant workrate trials, except where noted.

Results

Time-trial performance and power output

20 km Time-trial performance was significantly improved in the hyperoxic condition (27 minutes 34 seconds in HI vs. 28 minutes 8 seconds in NORM, $P < 0.005$). The average power output in HI was correspondingly higher; 292 ± 36 W

in HI compared to 277 ± 35 W in NORM ($P < 0.001$, Fig. 8). Of the eleven subjects, ten subjects achieved their best performance in the hyperoxic condition, while one subject performed similarly in both conditions. The individual and mean average power output recorded for each subject in HI and NORM are shown in Fig 8.

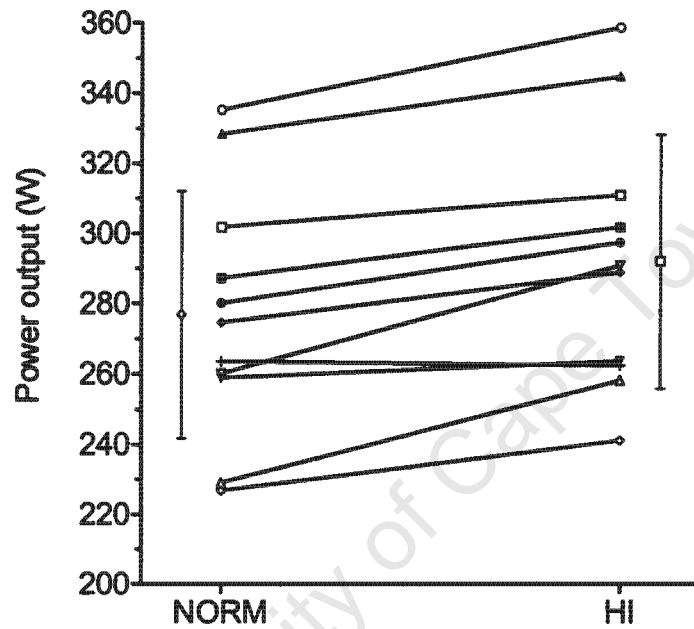


Figure 8. Average power output for each subject during 20 km time-trials in HI ($F_{I}O_2$ 0.4) and NORM ($F_{I}O_2$ 0.21) conditions. Average power outputs for the group are shown for NORM and HI

Values are means \pm S.D. for eleven subjects

The average of the individual power outputs recorded during each two kilometer interval during the time-trials is shown in Fig 9. The pacing strategy differed between HI and NORM. In NORM, power output began to decrease from 2 km onwards, and was significantly lower than at the start of the trial from 10 km until 18 km ($P < 0.05$), before increasing significantly for the final 2 km interval. In HI,

power output was maintained throughout the trial, until the final two kilometers when it increased significantly ($P < 0.05$). This resulted in a significantly greater power output in HI than in NORM from 12 km until the end of the trial ($P < 0.05$). In HI, the highest power output during the trial occurred in the final 2 km, compared to NORM, where the highest power output was recorded in the first 2 km and the final 2 km intervals (Fig. 9).

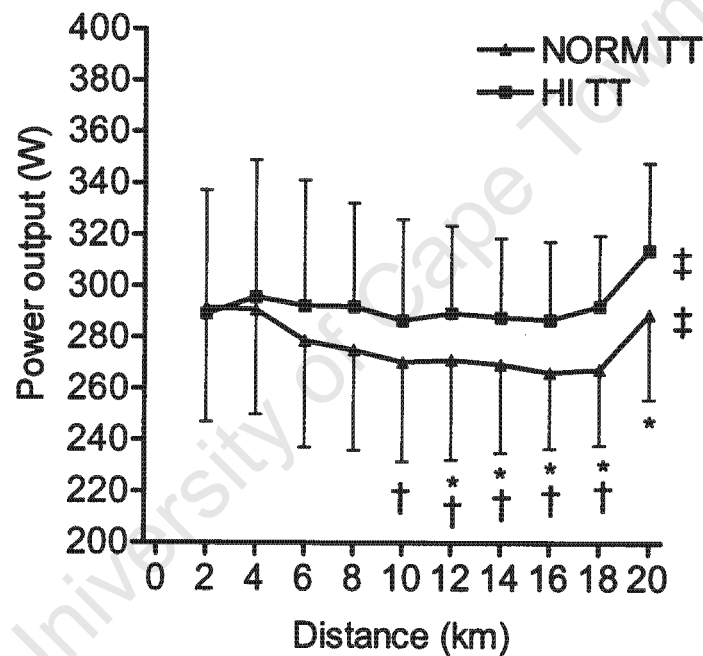


Figure 9. Average power output for 2 km intervals during time-trials in HI and NORM.

Values are means \pm S.D. for eleven subjects. † Significantly different from starting power output in NORM ($P < 0.05$); * Significantly different from HI ($P < 0.05$); ‡ Significantly different from preceding values within the same condition ($P < 0.05$)

During constant workload trials, the power output, based on the average power output from the 20 km time-trials, was 292 ± 40 and 293 ± 41 W in HI and

NORM, respectively. Hyperoxia resulted in improved performance during the constant workload trials to fatigue (Fig. 10), with five out the eight subjects cycling for longer in hyperoxia, and three subjects cycling similar distances in the two conditions. Five out the eight subjects were also able to cycle further than 20 km when cycling at a constant workrate in hyperoxia (Range 14 to 28 km).

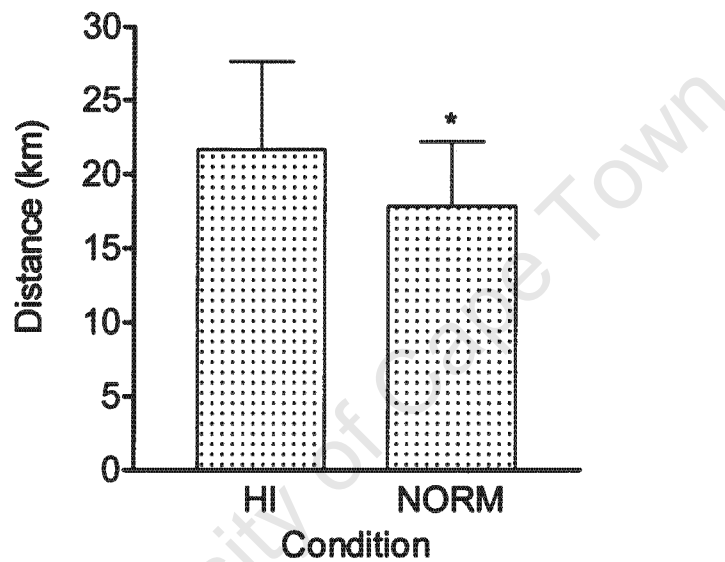


Figure 10: Distance cycled to fatigue during constant workrate trials in HI and NORM.

Values are means \pm S.D. for eight subjects. * Significantly different from HI ($P < 0.05$)

IEMG activity

Cadence ranged between 85 and 100 revolutions per minute and was not different between conditions for each subject. A significant time x trial interaction was found for IEMG during the time-trials (Fig 11A). That is, IEMG was maintained

during the trial in NORM, and increased slightly, though non-significantly, over time in HI. The IEMG was greater in HI than in NORM at 12, 16 and 18 km (Fig. 11A). There was also a tendency for IEMG to be greater in HI than in NORM at 14 km ($P = 0.08$). IEMG increased significantly during the final kilometer of trials in both conditions ($P < 0.005$), with peak values being recorded at 20 km (Fig. 11A).

During the constant workload trials, IEMG activity was not different between HI and NORM (Fig 11B). IEMG activity increased significantly in both HI and NORM, with values at fatigue significantly greater than during the first 8 km in both conditions ($P < 0.05$, Fig 11B).

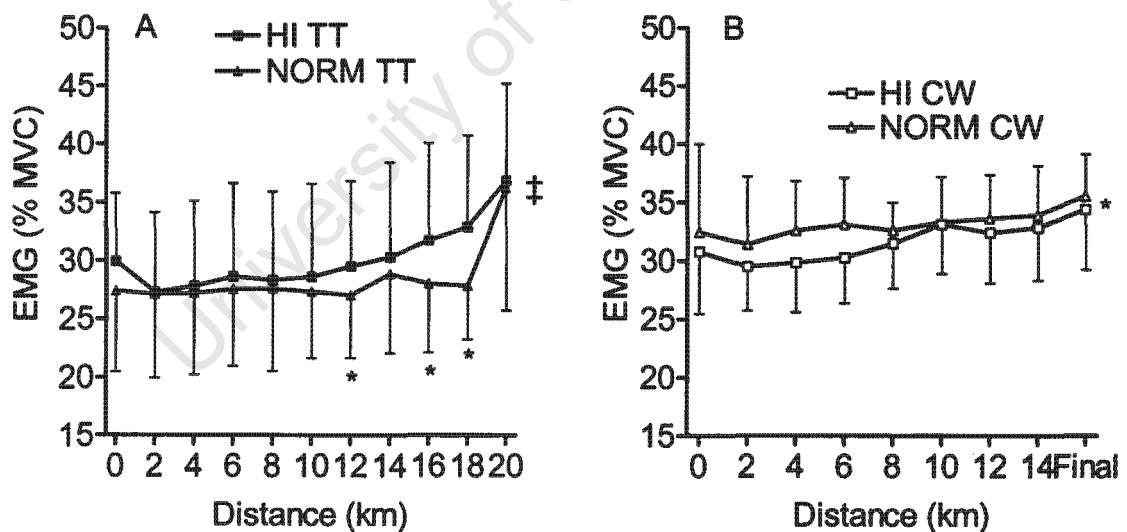


Figure 11 A: Integrated electromyogram (IEMG) activity in the vastus lateralis (VLO) at 2 km intervals during 20 km time-trials in HI and NORM.

Values are means \pm S.D. for ten subjects. * Significantly different from HI ($P < 0.05$); ‡ Significantly different from preceding values within HI and NORM ($P < 0.005$).

Figure 11B: Integrated electromyogram (IEMG) activity in the vastus lateralis (VLO) at 2 km intervals during constant workrate trials to fatigue in HI and NORM.

Values are means \pm S.D. for seven subjects. * Significant increase over time in both conditions ($P < 0.05$). No significant difference between conditions.

Fig 12A and B show the calculated ratio of power output to IEMG during the 20 km time-trials (Fig 12A) and constant workrate trials (Fig 12B). During the time-trials, there were no differences in this ratio between HI and NORM at any time, though the ratio decreased over time in both conditions (Fig 12A). During constant work rate trials, the ratio of power output to IEMG activity also decreased progressively over time, and was similar between HI and NORM (Fig 12B).

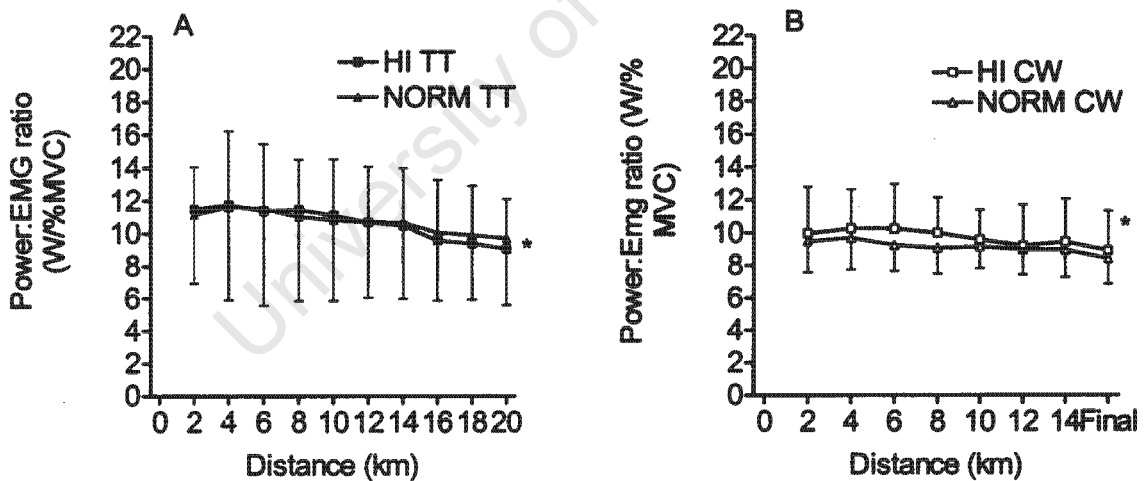


Figure 12A. Ratio of power output to IEMG activity during 20 km time-trials in HI and NORM.

Figure 12B. Ratio of power output to IEMG activity during constant workrate trials to fatigue in HI and NORM.

Values are means \pm S.D. for seven subjects. * Significant decrease over time in both conditions ($P < 0.05$). No significant difference between conditions.

Plasma lactate concentrations

Figure 13 depicts the plasma lactate concentrations during 20 km time-trials (13A) and constant workrate trials (13B). During the 20 km time-trials, plasma lactate concentrations were not significantly different between conditions (Fig. 13A), and increased significantly during the trials ($P < 0.0005$). During the constant workrate trials, plasma lactate levels were significantly greater ($P < 0.01$) in NORM than in HI from 6 km until volitional fatigue (Fig 13B).

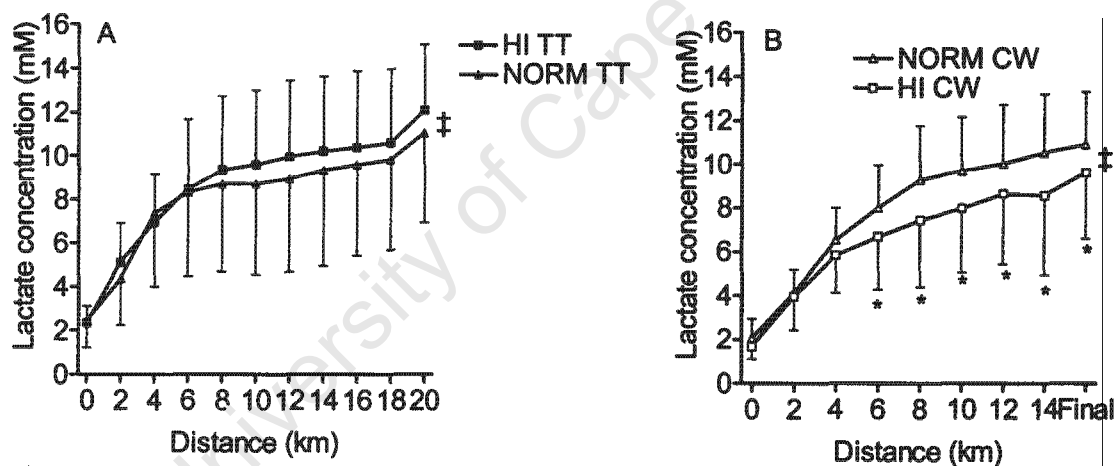


Figure 13A. Plasma lactate concentrations during 20 km time-trials in HI and NORM.

Values are means \pm S.D. for nine subjects. ‡ Significant increase over time in both conditions ($P < 0.0005$). No differences between conditions.

Figure 13B. Plasma lactate concentrations during constant workrate trials to fatigue in HI and NORM.

Values are means \pm S.D. for eight subjects. * Significantly different from HI ($P < 0.01$); ‡ Significant increase over time in both conditions ($P < 0.0005$).

Heart rate

Heart rate increased over time during both self-paced and constant workrate trials, and there were no differences between HI and NORM in any of the trials ($P < 0.0005$, Fig. 14).

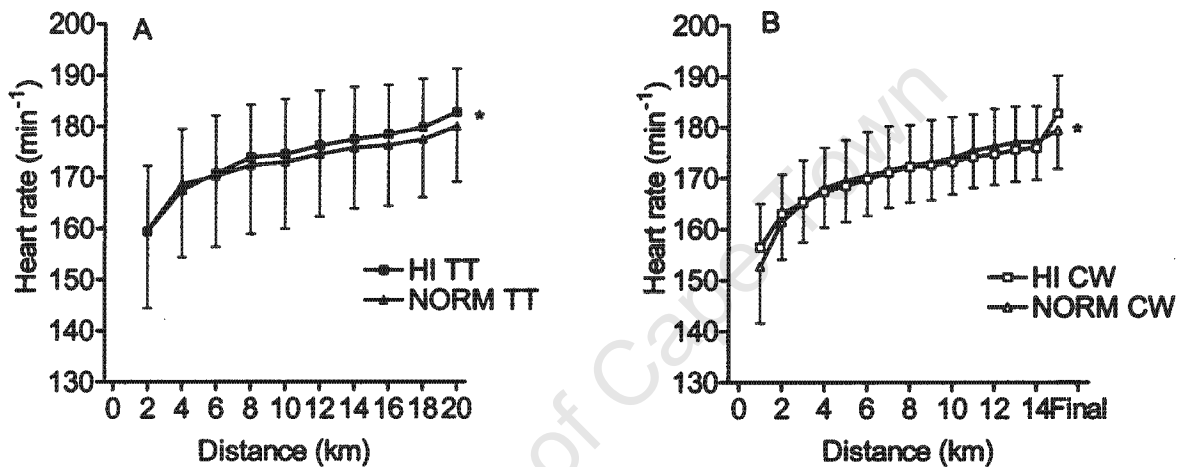


Figure 14A. Heart rate responses during 20 km time-trials in HI and NORM.

Values are means \pm S.D. for eleven subjects. * Significant increase over time in both conditions ($P < 0.0001$). No differences between conditions.

Figure 14B. Heart rate responses during constant workrate trials to fatigue in HI and NORM.

Values are means \pm S.D. for eight subjects. * Significant increase over time in both conditions ($P < 0.0001$). No differences between conditions.

Ratings of perceived exertion (RPE)

RPE increased significantly over time during time-trials ($P < 0.00005$, Fig. 15A) and during constant workrate trials ($P < 0.00005$, Fig 15B). There were no differences between HI and NORM in any trials. The final RPE values during the 20 km time-trials in HI and NORM were 10 ± 1 and 10 ± 1 respectively, while volitional fatigue during the constant workrate trials occurred at RPE values of 10 ± 1 in HI and 10 ± 1 in NORM.

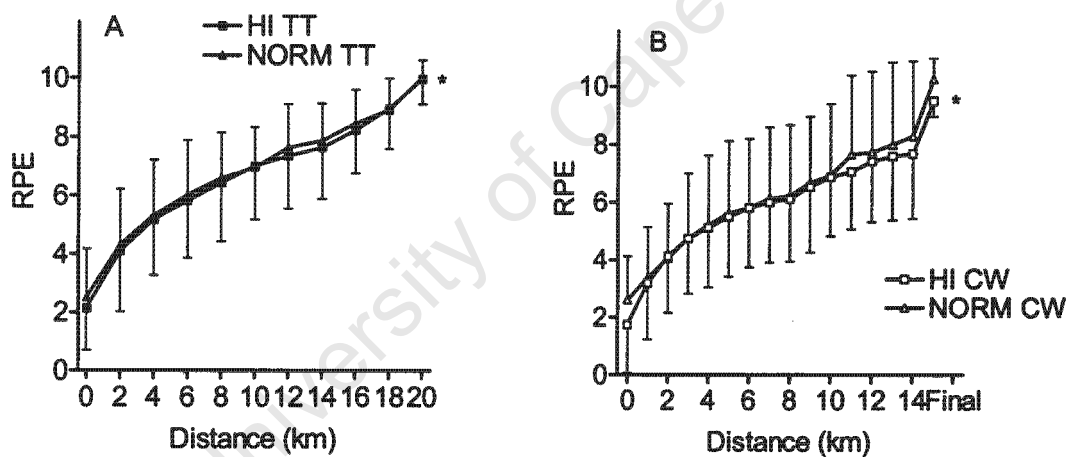


Figure 15A. Ratings of perceived exertion measured during time-trials in HI and NORM.

Values are means \pm S.D. for eleven subjects. * Significant increase over time in both conditions ($P < 0.00005$).

Figure 15B. Ratings of perceived exertion measured during constant workrate trials to fatigue in HI and NORM.

Values are means \pm S.D. for eight subjects. * Significant increase over time in both conditions ($P < 0.00005$).

Discussion

The first important finding of the present study was that both self-paced 20 km cycling time-trial performances (Fig. 8 and 9) and constant workrate performances (Fig. 10) were improved in hyperoxia; a $F_{I}O_2$ of 40% was thus sufficient to exert significant effects on cycling performance. In the self-paced 20 km time-trials, the improvement in performance was associated with an alteration in pacing strategy, since power output decreased progressively from the start of the trial in NORM, but was maintained throughout the trial in HI (Fig. 9). This was not associated with any differences in heart rate, plasma lactate concentrations or ratings of perceived exertion between conditions (Figs. 13 – 15), but was associated with an increase in the IEMG activity, reflecting increased skeletal muscle activation in hyperoxia (Fig. 11A).

Thus, in NORM-TT, IEMG activity remained constant throughout the trial, but increased progressively in HI-TT (Fig. 11A), resulting in significant differences between conditions at 12, 16 and 18 km, the same time as the difference in power output between HI and NORM reached statistical significance (Fig. 9). In contrast, during CW trials, the IEMG activity increased similarly during both HI and NORM, suggesting that similar levels of muscle activation were necessary to produce the same power output, despite differences in the oxygen content of the inspired air.

Impaired endurance exercise performance has been attributed to a progressive reduction in muscle force production ability caused by increased metabolite levels (Taylor *et al.* 1997; Haseler *et al.* 1998; Hogan *et al.* 1999; Linossier *et al.* 2000). For example, it has been found that improved performances in hyperoxia are associated with reduced P_i accumulation (Haseler *et al.* 1998). It is possible that dynamic exercise is affected in a similar way. However, it is significant that the measured reductions in power output and skeletal muscle activation levels in NORM-TT (Fig. 9 and Fig. 11A) were reversible, since activation levels increased during the final 2 km in both HI and NORM, allowing power output to return to the level measured during the first 2 km of the time trial. Hence, peripheral muscle fatigue alone cannot explain our results. If the greater reduction in power output in NORM-TT (Fig. 9) were solely the result of a metabolite-induced peripheral inhibition or impairment of muscle contractility compared to HI, then it would not be possible to increase power output at the end of exercise, unless the impaired force production could suddenly be overridden in the affected muscle fibres.

Instead, it is suggested that the increase in power output in the final 2 km is the result of an increase in muscle activation levels, as occurs during cycling time-trials in the heat (Chapter 2). By extension, the ability to maintain a greater power output during hyperoxia than normoxia (Fig. 9) may be the result of an increased capacity for the activation of motor units in hyperoxia, as suggested by the increased IEMG activity in that condition (Fig. 11A). A similar phenomenon was

proposed in Chapter 2 of the present thesis for exercise in hot conditions, and here this finding is extended to hyperoxia.

On the other hand, when peripheral muscle fatigue develops, power output can only be maintained by activating more muscle. The fact that the power-IEMG relationship decreased in all trials (Fig. 12A and B) supports the notion that the force producing ability of the muscle is indeed progressively reduced during all trials. Interestingly, however, it was found that the IEMG activity was similar between NORM-CW and HI-CW when power output was the same between conditions (Fig. 11B). If there was some oxygen-sensitive factor that acted peripherally to impair muscle force production, it would be expected that the IEMG activity would be greater in normoxia than in hyperoxia in order to produce the required power output, yet this was not the case. As a result, the Power/EMG ratio was not different between HI and NORM for time-trials (Fig 12A) or constant workload trials (Fig. 12B).

The ratio of force output to EMG activity has been used previously to examine the relative changes in muscle function during exercise (Taylor *et al.* 1997). It was found that the Force/EMG ratio was lower in hypoxia than in normoxia, which was interpreted as evidence that hypoxia impaired muscle function (Taylor *et al.* 1997). Similarly, Nummela *et al.* (1992) found that EMG activity in the active muscles during a 400 m sprint increased significantly over the course of the run, even though speed decreased, and it was concluded that additional motor units

were being activated to compensate for the progressive reduction in muscle force production as a result of metabolic acidosis in the muscle (Nummela *et al.* 1992).

The present results support the notion that peripheral changes in the muscle may necessitate an increase in skeletal activation to maintain a given force output.

However, using similar reasoning (Nummela *et al.* 1992; Taylor *et al.* 1997), our finding of a similar Power output/EMG ratio in HI and NORM during both self-paced and constant work rate cycling (Fig. 12) suggests that hyperoxia does not decrease the rate of development of peripheral muscle fatigue compared to normoxia. The fall in this ratio is thus not related to the oxygen content of the inspired air and therefore unlikely to be caused by oxygen-dependent metabolic differences

It is acknowledged that the measurement and interpretation of EMG during dynamic exercise is difficult and may be influenced by experimental and physiological bias. Nevertheless, in the absence of alternative ways to quantify muscle activation levels during long duration dynamic exercise, changes in IEMG amplitude can be attributed to changes in either centrally-mediated motor unit recruitment or to changes in the motor unit firing rate and, in experimental conditions like ours, are a reflection of changes in muscle activation (Farina *et al.* 2004).

A decrease in the central drive to the motor neuron pool could be the result of two possibilities: there may be a reduction in descending message from the higher brain centres, or there may be inhibition of the descending signal in the spinal cord, where afferent feedback may decrease the excitability of the motor neurons (Cheung & Sleivert 2004b). However, if the latter were true, then it is unclear how the amplitude of the signal could be increased again at the end of exercise (Fig. 11A), unless the inhibition could be overridden. I am unaware of any mechanism that would allow the inhibition to be suddenly reversed. A more reasonable explanation would be that the reduced IEMG in normoxia is at least in part due to lower central drive from the brain compared to the hyperoxic condition.

The possibility that changes in IEMG activity are the result of altered motor neuron firing rates must also be acknowledged. These changes can be mediated either centrally (Bilodeau *et al.* 2003) or peripherally, in response to fatigue (Marsden *et al.* 1983). That is, firing rates decline to compensate for a reduction in muscle relaxation time and contraction speed (Marsden *et al.* 1983; Gandevia, 2001), secondary to peripheral changes in the muscle (MacLaren *et al.* 1989; Hepple, 2002; Westerblad *et al.* 2002). Thus, the difference in IEMG activity between HI-TT and NORM-TT may be attributed to this phenomenon. It is not clear, however, how the IEMG could increase significantly in the final 2 km of both TT if this phenomenon were occurring, since peripheral changes in the muscle, which are presumably responsible for the reduction, are still present and would

likely increase progressively to a maximum at the end of exercise. Conversely, the higher IEMG activity during HI-TT may be due to increased motor unit firing rates, possibly as a result of reduced peripheral metabolite levels, or to altered central activation patterns as a result of direct effects of hyperoxia on the CNS.

During fatiguing isometric exercise, the median frequency (MF) of the EMG signal is often measured since it provides a measure of changes in motor unit firing rate with fatigue (Bigland-Ritchie, 1981; Bilodeau *et al.* 2003). However, during dynamic exercise, this method is probably not reliable since muscle fibre conduction velocity and hence MF may be influenced by temperature changes in the muscle (Bigland-Ritchie, 1981) and the continuous changes in muscle length and position during such dynamic exercise. Other promising techniques such as functional MRI, multimodal imaging and Transcranial Magnetic Stimulation are, at least for now, not feasible because of the large movement component during dynamic exercise, leaving only IEMG as an indirect means to measure activation levels. Thus, while the EMG data must be interpreted with caution, the finding that pacing strategy is altered in association with changes in IEMG activity suggests that altered skeletal muscle activation, in combination with peripheral changes in the muscle, is responsible for the altered exercise performance.

If such a hypothesis is correct, it is not clear from the present data how this differential regulation of muscle activation and exercise performance is achieved in the hyperoxic condition. Much debate exists regarding the effects of the

accumulation of metabolites on muscle function and exercise performance

(Nummela *et al.* 1992; Taylor *et al.* 1997; Linossier *et al.* 2000; Foster *et al.* 2003).

Previous studies have shown that when exercise is performed in hyperoxia, fatigue

occurs at similar arterial pH levels and lactate concentrations as in normoxia

(Linossier *et al.* 2000). Nielsen *et al.* (1999) have suggested that elevated

inspiratory O₂ fraction improves exercise performance by maintaining cerebral

oxygenation and it is possible that our subjects showed this phenomenon.

Similarly, lactate accumulation (Linnarsson *et al.* 1974; Hogan & Welch 1984),

ventilation (Peltonen *et al.* 1999), the β -adrenergic response to exercise (Howley

et al. 1983), systemic (Peltonen *et al.* 1999) arterial desaturation (Nielsen *et al.*

1999), and reductions in skeletal muscle ATP, ADP and total NADH

concentrations (Linossier *et al.* 2000) are all attenuated in hyperoxia. Thus, it has

been proposed that hyperoxia improves performance to fatigue by reducing the

biochemical and physiological disturbances to homeostasis (Wilber *et al.* 2003),

which would allow exercise to continue for longer before 'limiting' concentrations are reached.

Indeed, it was found that when exercise was performed at the same constant

power output in HI and NORM, plasma lactate concentrations were lower in HI

compared to NORM (Fig. 13B). However, when exercise was self-paced, blood

lactate concentrations were similar (Fig. 13A), presumably because the exercise

workrate was higher in hyperoxia. Foster *et al.* (2003) have suggested that

athletes are sensitive to proton accumulation and that the pacing strategy during

high intensity exercise is regulated to ensure that a harmful metabolic acidosis does not occur until the end of the event. While there are no measurements of lactate concentrations in the active muscles, it seems reasonable to suggest that hyperoxia attenuated the degree of metabolic acidosis at a given power output (Fig. 13B). Therefore, during the self-paced time-trials, a greater power output could be maintained before a harmful acidosis could occur.

Other variables, including ventilation (Peltonen *et al.* 1999), β -adrenergic activation (Howley *et al.* 1983), systemic (Peltonen *et al.* 1999) and cerebral arterial desaturation (Nielsen *et al.* 1999), and the reduction in skeletal muscle ATP, ADP and total NADH concentrations (Linossier *et al.* 2000), all of which are proposed to limit exercise performance, may be similarly regulated by this neural control process when exercise is self-paced and the workrate is free to vary. The present hypothesis therefore holds that changes in power output and skeletal muscle recruitment are adjusted to ensure that these variables, which are often implicated as “limiting” factors for exercise performance, are instead the very variables that are regulated similarly in the two conditions.

The novel aspect of this model is that the changes in workrate and muscle activation occur in advance of the attainment of critical changes in the physiological systems and processes they are ultimately responsible for regulating (Chapter 2). Changes in pacing strategy and performance are thus the markers of the integration of afferent sensory feedback with a feed forward, anticipatory

adjustment in power output. This has previously been shown during exercise in the heat (Chapter 2), where exercise intensity and IEMG activity are reduced in advance of differences and excessive increases in core temperature compared to cool conditions (Marino *et al.* 2004; Tucker *et al.* 2004), and the major contribution of the present study is to suggest that a similar phenomenon occurs during exercise in hyperoxia, possibly due to the regulation of metabolite accumulation.

A final interesting observation in the present study was that five out of eight subjects were able to cycle for more than 20 km when power output was held constant at the same average power output that they achieved during self-paced exercise. This resulted in greater work being done during the constant workload trials compared to the time-trials in hyperoxia, and suggests that those subjects did not perform optimally during the time-trials. This finding may be due to the neural control system which is suggested to regulated the characteristic pacing strategy that is observed during self-paced time-trials, both in the present study (Chapter 2) and in previous research (Foster *et al.* 2003; Marino *et al.* 2004).

That is, exercise intensity is maintained at sub-maximal levels for most of the trial, before a significant increase in power output occurs in the final kilometer (Fig. 9). The maintenance of this 'reserve' during the self-paced trials does not therefore result in optimal performance, though factors such as training may alter the pacing

strategy that is adopted. Further research is required to examine the regulation of pacing strategy and its implications for performance.

In conclusion, self-paced exercise performance is improved in hyperoxia as a result of differences in pacing strategy compared to normoxia. Power output was maintained in HI but decreased progressively in NORM, until the final 2 km. This characteristic pacing strategy, together with the associated changes in IEMG activity, suggests that hyperoxia improves exercise performance through a combination of peripheral effects and an enhanced ability to activate skeletal muscle and hence to exercise with a greater power output. Exercise performance is therefore regulated in a complex system, sensitive to peripheral changes and to the oxygen content of the inspired air.

Chapter 4

***The rate of heat storage mediates an
anticipatory reduction in exercise
intensity during cycling at a fixed
rating of perceived exertion***

The research findings from this chapter have been published as an Original

Investigation in the *Journal of Physiology* 574: 905-915, 2006

Introduction

Exercise performance is impaired in hot compared to cool conditions (Nielsen *et al.* 1990; Marino *et al.* 2000; Tatterson *et al.* 2000; Marino *et al.* 2004)(Chapter 2), and this has been attributed to 'central fatigue' (Nybo & Nielsen 2001a), in which there is a failure of motor unit recruitment after body temperature reaches a critical limiting value of approximately 40°C (Nielsen *et al.* 1990; Nybo & Nielsen 2001a). In support of this, if exercise is performed at a constant workrate until volitional fatigue, skeletal muscle motor unit recruitment, measured indirectly using surface electromyography (EMG) during sustained isometric contractions, is lower in hyperthermic (40°C) compared to normothermic (38°C) subjects (Nybo & Nielsen 2001a), suggesting that the 'hot brain' is unable to recruit motor units to allow exercise to continue at the required workrate.

Chapter 2 of the present thesis examined the regulation of exercise performance and pacing strategy during self-paced exercise in the heat. It was found that power output and IEMG activity during self-paced cycling exercise in the heat decreased well before the core temperature reaches 40°C. Further, the power output and IEMG activity were greatest in the final kilometer of the 20 km cycling time-trial, when the rectal temperatures were also the highest, suggesting that high body temperatures do not directly impair centrally regulated muscle activation (Chapter 2). More recently, isometric force production and voluntary activation

percentage have been found to decrease progressively during a passive heating protocol (Morrison *et al.* 2004), even at body temperatures below 39°C.

Todd *et al.* (2005) found that reduced force output occurred due to a failure of voluntary drive during passive heating despite the availability of additional motor cortical output, which would, in theory, allow increased force output.

Collectively, these studies (Tattersson *et al.* 2000; Marino, 2004; Morrison *et al.* 2004; Cheung & Sleivert 2004a) suggest that when force output or exercise workrate are self-selected rather than being fixed, an anticipatory mechanism (Chapter 2) adjusts the workrate by regulating the degree of motor unit recruitment to prevent body temperature from rising to levels which may cause harm or premature fatigue (Marino *et al.* 2004). The brain thus acts pre-emptively to ensure that a catastrophic failure of thermoregulation does not occur.

Such regulation represents homeostatic control, the goal of which is to prevent an abnormal rise in body temperature by regulating the rate of heat production (Marino *et al.* 2004; Tucker *et al.* 2004). It has also been found that the conscious sensation of fatigue, measured as the rating of perceived exertion (RPE), is correlated with changes in brain electrical rhythms (Nybo & Nielsen 2001b) and increases in body temperature during both dynamic exercise ((Galloway & Maughan 1997; Nybo & Nielsen 2001a) and passive heating (Gonzalez-Alonso *et al.* 1999; Armada-da-Silva *et al.* 2004).

In Chapter 2 of this thesis, the RPE was found to increase similarly during time-trials in hot and cool conditions, even though power output was lower in the hot condition. Similarly, in Chapter 3, it was found that the RPE rose similarly during time-trials in hyperoxia and normoxia, despite differences in pacing strategy and skeletal muscle activation that were present throughout the trials. It was proposed that rather than acting solely as a measure of exercise intensity, the conscious perception of effort plays a regulatory function to ensure that the work rate remains at an intensity that can be safely sustained for the expected duration of the exercise. If this is correct, then this regulatory control should be evident during exercise at a constant RPE. This has been termed this the RPE clamp protocol.

Accordingly, Chapter 4 of the present thesis aimed to examine the regulation of exercise performance in hot (35°C) compared to normal (25°C) and cool (15°C) conditions during cycling at a predetermined, fixed rating of perceived exertion (RPE). In this protocol the subject is free to vary the work rate, but only to ensure that the RPE remains constant throughout exercise. It has previously been shown that the RPE clamp produces repeatable results with respect to trial duration, rate of power output decline, average power output, heart rate, oxygen consumption and skeletal muscle recruitment (unpublished observations).

It was hypothesized that during exercise at a fixed RPE, self-selected power output would decrease most rapidly in the hot condition, which would ensure that the

rate of heat storage would be similar in all environmental conditions. The more rapid decrease in power output in the heat would occur soon after the onset of exercise, and even when the rectal temperature was not greatly elevated but was the same as values measured during exercise in the cooler conditions.

Methods

Subjects

Eight well-trained male cyclists were recruited from local cycling clubs, training facilities and from participation in previous studies. The subjects' mean age, height, body mass and peak power output were 23.4 ± 4.2 , 177.1 ± 7.1 cm, 70.57 ± 7.14 kg and 369.1 ± 32.8 W respectively. Prior to participation in the study, the subjects were informed of the risks associated with the study, and informed consent was obtained in writing prior to the initiation of the study. Subjects were also required to refrain from any strenuous exercise on the day prior to or on the day of a trial. The Research and Ethics Committee of the Faculty of Health Sciences of the University of Cape Town Medical School approved the study.

Testing Procedure

Subjects were required to report to the laboratory on five separate occasions.

During the first two occasions, subjects underwent preliminary testing consisting of peak power output tests, familiarization trials and anthropometric measurements. The subject's height (cm) and body mass (kg) were measured using a precision stadiometer and balance (Model 770, Seca, Bonn, Germany, accurate to 10g). The third, fourth and fifth trials were experimental trials, during which subjects performed three cycling trials in random order, in an environmental chamber at ambient temperatures of 15°C (COOL), 25°C (NORM) and 35°C (HOT). All cycling trials were performed on a Kingcycle ergometer system, which allows subjects to cycle their own bicycles in the laboratory.

Preliminary testing

Each subject's Peak Power Output (PPO) was determined using a modified protocol as described by Hawley & Noakes (1992). Subjects performed a self-paced warm-up for 10 minutes prior to beginning the test at a starting power output of 2.5 W/kg body weight. The workload was increased by 20 W per minute until exhaustion. The test was terminated when the subject was unable to match the required power output. PPO was recorded as the highest mean power output achieved over a one-minute period. The subjects were also requested to refrain from standing throughout the test.

Within a week of performing the PPO trial, subjects reported to the laboratory for a familiarization session, during which they underwent procedures identical to the experimental trials. Subjects completed a cycling trial at a fixed RPE in ambient temperatures of 24°C, relative humidity of 60% and a wind velocity of 10km/h under the same conditions and procedures as the experimental trials (described below). During both the PPO and the familiarization trials, the subjects were familiarized with both the Borg RPE scale and instructions for the subsequent trials. A standard set of instructions was given to subjects during these two trials.

Experimental Protocol

Environmental conditions

Subjects performed three randomized experimental trials, separated by three to seven days, in an environmental chamber (Scientific Technology Corporation, Cape Town, South Africa), at ambient temperatures of 15°C, 25°C and 35°C and relative humidity and wind speed of 60% and 10km/h respectively. Temperature, humidity and wind velocity were measured every minute. The wind velocity was measured at the level of the subject's head while in their riding position. We have previously shown that this wind speed is sub-optimal and causes increased heat storage during exercise in the heat (Saunders *et al.* 2005). It was assumed that subjects were not heat acclimatized as the testing was conducted during the winter months between April and September, when the average maximum air

temperature ranges from 16 to 19°C. Subjects were asked to refrain from strenuous physical exercise, caffeine and alcohol on the day of and prior to the trials, and from all products containing ephedrine for the duration of their involvement in the trials. Euhydration was confirmed by a body weight within 200 g of the preceding trials, a resting rectal temperature of within 0.2°C of the preceding trials, and a resting heart rate within six beats of the previous trials (Montain & Coyle 1992). During the trials, subjects were allowed to ingest water ad libitum.

Cycle trials at a fixed rating of perceived exertion (RPE)

During experimental trials, subjects cycled on the Kingcycle ergometer at a fixed rating of perceived exertion (RPE). Subjects were instructed to cycle from the outset at a power output which was perceived by them to represent an RPE of 16 on the Borg 6 to 20 Rating of Perceived Exertion scale (Borg, 1982). This rating corresponded to the verbal cue of between “hard” and “very hard” on the Borg scale. The average of the power output measured during the first three minutes of the trial was calculated and defined as the starting power output. Subjects continued cycling until their power output declined to a value corresponding to 70% of the starting power output. The trial was terminated when the power output, measured every minute during the trial, decreased below 70% of the initial value for three consecutive minutes. Immediately after the trial was terminated, subjects were requested to perform a maximal sprint, lasting 30 seconds, during

which time they were verbally encouraged to produce the highest power output possible. No feedback in terms of distance covered, time elapsed or power outputs and heart rates was provided to the subject at any time during any trial.

IEMG testing

Prior to each experimental session, subjects performed two maximum voluntary contractions (MVC) for normalization of the IEMG signal obtained during the subsequent cycling bouts. This method for normalization of the IEMG signal has been used previously for both maximal (Hunter *et al.* 2002b; Hunter *et al.* 2003) and sub-maximal (St Clair Gibson *et al.* 2001b; Tucker *et al.* 2004) cycling exercise. Briefly, the electromyographic (EMG) activity of the vastus lateralis muscle was recorded during the two five-second maximal isometric contractions using an isokinetic dynamometer (Bio-Dex dynamometer, Bio-Dex, United Kingdom). Subjects were firmly strapped into the dynamometer and the right leg attached to the arm of the dynamometer at a level slightly above the lateral malleolus. The arm was set so that the knee was at a 60° angle from full leg extension (0°). The IEMG activity recorded from the contraction producing the highest force was used for normalization of the IEMG signal obtained during the subsequent cycling trials.

During each MVC and the subsequent cycling trials, the IEMG activity of the vastus lateralis muscle was recorded. Before placement of the electrodes, the skin was shaved and cleaned with 95% ethanol, according to methods previously described

(Kay *et al.* 2001; St Clair Gibson *et al.* 2001b). A triode electrode (Thought Technology, West Chazy, N.Y., USA) was placed over the muscle belly of the vastus lateralis and connected to a pre-amplifier. Outputs from the pre-amplifier were relayed to a Flexcomp/DSP IEMG apparatus (Thought Technology USA) via a fiber optic cable and stored by an online computer. IEMG signals were captured at 1984 Hz and analysed for five-second periods during the MVC and for five second periods at each measurement period during the trials. For analysis, the raw IEMG signals were filtered using a second order 15Hz Butterworth high-pass filter to remove movement artefact, rectified, then smoothed with a low-pass second order Butterworth filter with a cut-off frequency of 5 Hz. This was performed using MATLAB™ software.

All processed IEMG data were normalized by dividing the IEMG value obtained at each workload during the trials by the IEMG value obtained during the MVC performed before the start of the trial. IEMG data are therefore expressed as a percentage of the IEMG measured during the MVC. This method of IEMG normalization has been shown to be reliable and valid for use in cycling trials, (Hunter *et al.* 2002b), and the neuromuscular responses (IEMG) during self-paced cycling in the heat are reproducible between trials using this methodology (Kay *et al.* 2001).

Temperature measurements

Following Kingcycle calibration and the measurement of body mass, the subjects inserted a rectal thermometer (YSI 409AC, Yellow Springs, OH, USA) 10 cm beyond their anal sphincter. Four surface skin thermocouples (YSI 427, Yellow Springs, OH, USA) were taped to the medial calf, anterior mid-thigh, anterior mid-bicep and on the chest at a point midway between the acromium process and the nipple. The subject was then instructed to perform a self-paced 10-minutes warm-up on the bicycle outside the chamber at room temperature. The duration was standardized to ensure that initial core and skin temperature values taken prior to entering the chamber were not different between the HOT, NORM and COOL conditions. At the completion of the warm-up, initial heart rate and temperature readings were obtained. Skin and rectal temperatures were recorded at one minute intervals throughout the trial using a telethermometer (YSI 400 series, Yellow Springs, OH, USA, accurate to 0.1°C).

The weighted skin temperature from four sites was calculated by the following equation (Mitchell & Wyndham 1969; MaClean, 1993):

$$T_{sk} = 0.3(T_{chest} + T_{arm}) + 0.2(T_{thigh} + T_{leg})$$

The total body temperature used in the study was calculated from skin and core temperatures using the following equation (Colin *et al.* 1971):

$$T_{body} = 0.79(T_{rec}) + 0.21(T_{sk}) \quad (\text{Ramanathan, 1964})$$

Heat content was calculated every minute during exercise using the following equation in which Q_c was the heat content, T_b was the body temperature in °C, m was body mass in kg, and 3.47 was constant in $\text{kJ } ^\circ\text{C}^{-1} \text{ kg}^{-1}$.

$$Q_c = T_b \times m \times 3.47$$

Heat Storage was calculated with the following equation, in which Q_s is heat storage in kJ, Q_{cT1} is heat content at time 1 and Q_{cT2} is the heat content at time 2.

$$Q_s = Q_{cT1} - Q_{cT2}$$

Heart rates were measured at the start of the trial and every minute throughout the duration of the trial, using a Polar S720i heart rate monitor (Polar Electro OY, Kempele, Finland). Total exercise time was also recorded from the heart rate monitor.

During the RPE clamp part of each trial, power output readings were recorded every minute by the Kingcycle ergometer. During the 30 second maximal sprint bout, power output was measured every 5 seconds and then averaged for the 30 second sprint.

As required by the RPE clamp, subjects were required to ride constantly at an RPE of 16 using the Borg 6 to 20 rating of perceived exertion scale (Borg, 1982). This RPE was confirmed every two-minute interval. In addition a rating of thermal comfort was also recorded at two-minute intervals, using a modified Borg

category scale. This scale ranged from values of 1, which corresponded to 'much too cool', to 7, which corresponded to 'much too hot.'

Statistical analysis

All statistical analyses were performed using Statistica 6.0 (Statsoft Inc. 1284 – 2001). Data are presented as means \pm S.D. Power output, skin and body temperatures, heat storage, heart rates, and IEMG were analyzed using repeated measures ANOVA (Trial \times time). Where significant interaction effects were found, a post-hoc analysis was performed using a Tukey's HSD test for pairwise comparisons. Because trials varied in duration, trials were time-normalized by expressing the time of each measurement as a percentage of the total trial duration. Significance was accepted at $P < 0.05$.

Results

The temperatures for COOL, NORM and HOT were 15.1 ± 0.3 , 24.9 ± 0.4 and $35.2 \pm 0.6^\circ\text{C}$ respectively. The average humidity was $68 \pm 4\%$, $66 \pm 4\%$ and $65 \pm 3\%$ for COOL, NORM and HOT respectively.

Exercise Performance

Total trial duration was significantly shorter in the HOT compared to the NORM and COOL conditions (Fig. 16A) (34.0 ± 10.4 , 48.6 ± 14.1 and 50.2 ± 16.3 minutes, $P < 0.001$). Power output decreased at a significantly greater rate in HOT compared to the other two conditions; 2.35 ± 0.73 W/min compared to 1.63 ± 0.70 and 1.61 ± 0.80 W/min for the COOL and NORM trials respectively ($P < 0.05$).

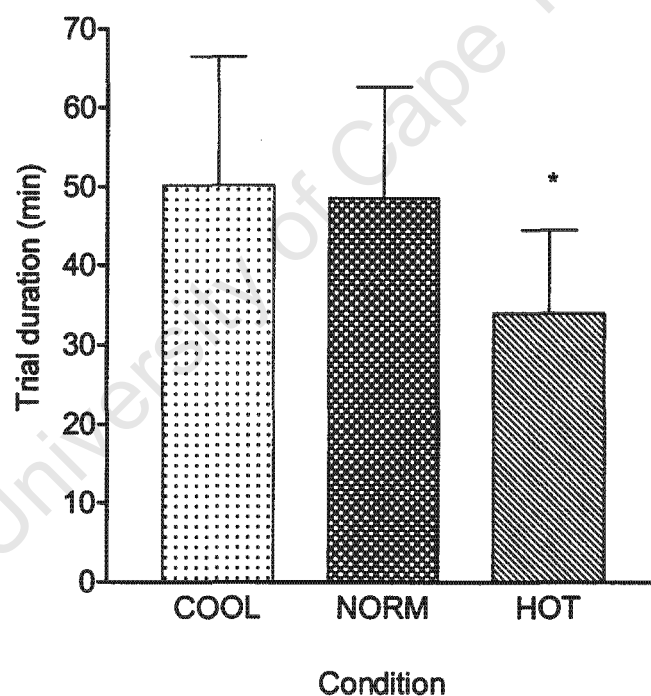


Figure 16. Total trial duration during trials in HOT (35°C), NORM (25°C) and COOL (15°C) conditions.

Values are means \pm S.D. for eight subjects. * Significantly different from NORM and COOL ($P < 0.001$)

Fig. 17 shows the decrease in power output expressed as a percentage of total trial duration. The starting power outputs were not different between trials (245 ± 35 , 250 ± 43 and 261 ± 33 W for COOL, NORM and HOT respectively). Power output decreased linearly in all trials, as seen by correlation co-efficients of $r = 0.99$, 0.97 , and 0.95 for the HOT, NORM and COOL trials, respectively. The rate of decrease in power output tended to be highest in HOT, though this was not significant (Fig. 17).

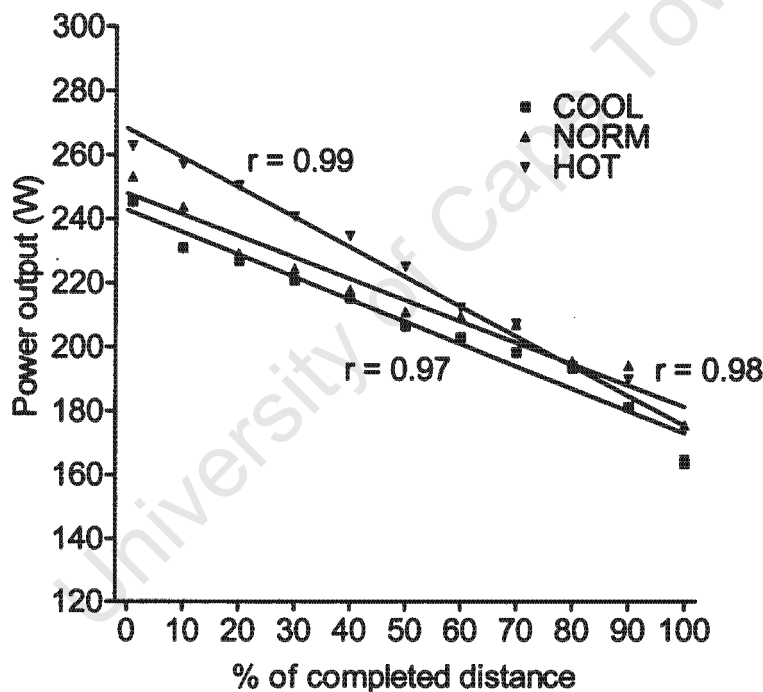


Figure 17. Mean power output expressed relative to total trial duration in HOT, NORM and COOL conditions.

During the 30 second sprint at the end of the trial, peak power outputs of 437 ± 61 W, 492 ± 78 W and 454 ± 88 W were achieved in COOL, NORM and HOT,

respectively. The power outputs achieved during these 30 second sprints were not significantly different between conditions.

Heat Storage

During the first four minutes of the trials, the rate of heat storage was significantly greater in the HOT condition than in NORM and COOL (Fig. 18A, $P < 0.05$).

From five minutes until trial completion, there were no differences in rates of heat storage between conditions. When expressed relative to total trial duration, heat storage was significantly higher in HOT at 1% and again at 90 and 100% relative trial duration (Fig. 18B, $P < 0.05$). No significant differences were found between conditions between 1% and 90%.

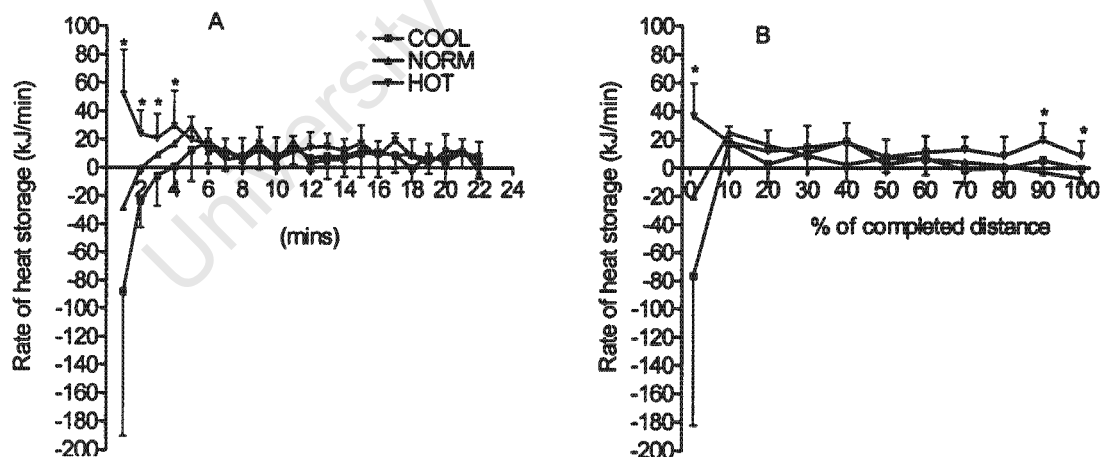


Figure 18. Rate of heat storage (kJ/min) during the first 22 minutes of the trials (A) and expressed relative to total trial duration (B) for trials in HOT, NORM and COOL conditions.

Values are means \pm S.D. for eight subjects. * Significantly different from NORM and COOL ($P < 0.001$); * Significantly different from NORM and COOL ($P < 0.05$)

The rate of heat storage was significantly higher during the first ten minutes in the HOT compared to the NORM and COOL conditions (Fig. 19A, $P < 0.05$). From 10 to 20 minutes, rate of heat storage decreased significantly in the HOT condition and increased significantly in the COOL, resulting in a similar rate of heat storage between conditions during this time period. There were no differences in heat storage between conditions from 20 to 30 minutes (Fig. 19A).

Fig. 19B depicts power output, expressed as a percentage of initial power output during at 10 minutes intervals during trials. Overall, there was a significant decrease in power output in all conditions, with the greatest reduction occurring in the HOT condition ($P < 0.01$). Over the first ten minutes, the decrease in power output from starting values was not different between conditions. From 10 to 20 minutes, power output decreased significantly in the HOT condition but not in the NORM or COOL conditions. From 20 to 30 minutes power output continued to decrease at a significantly faster rate in the HOT condition than in the NORM and COOL conditions, so that the decrease from initial power output was significantly greater in HOT compared to NORM and COOL ($P < 0.01$).

The change in power output over 10 minute periods was plotted against the rate of heat storage during the preceding 10 minute interval (Fig. 19C). There was a linear relationship between the change in power output and the change in heat content, with a correlation co-efficient of $r = 0.92$ (Fig. 19C).

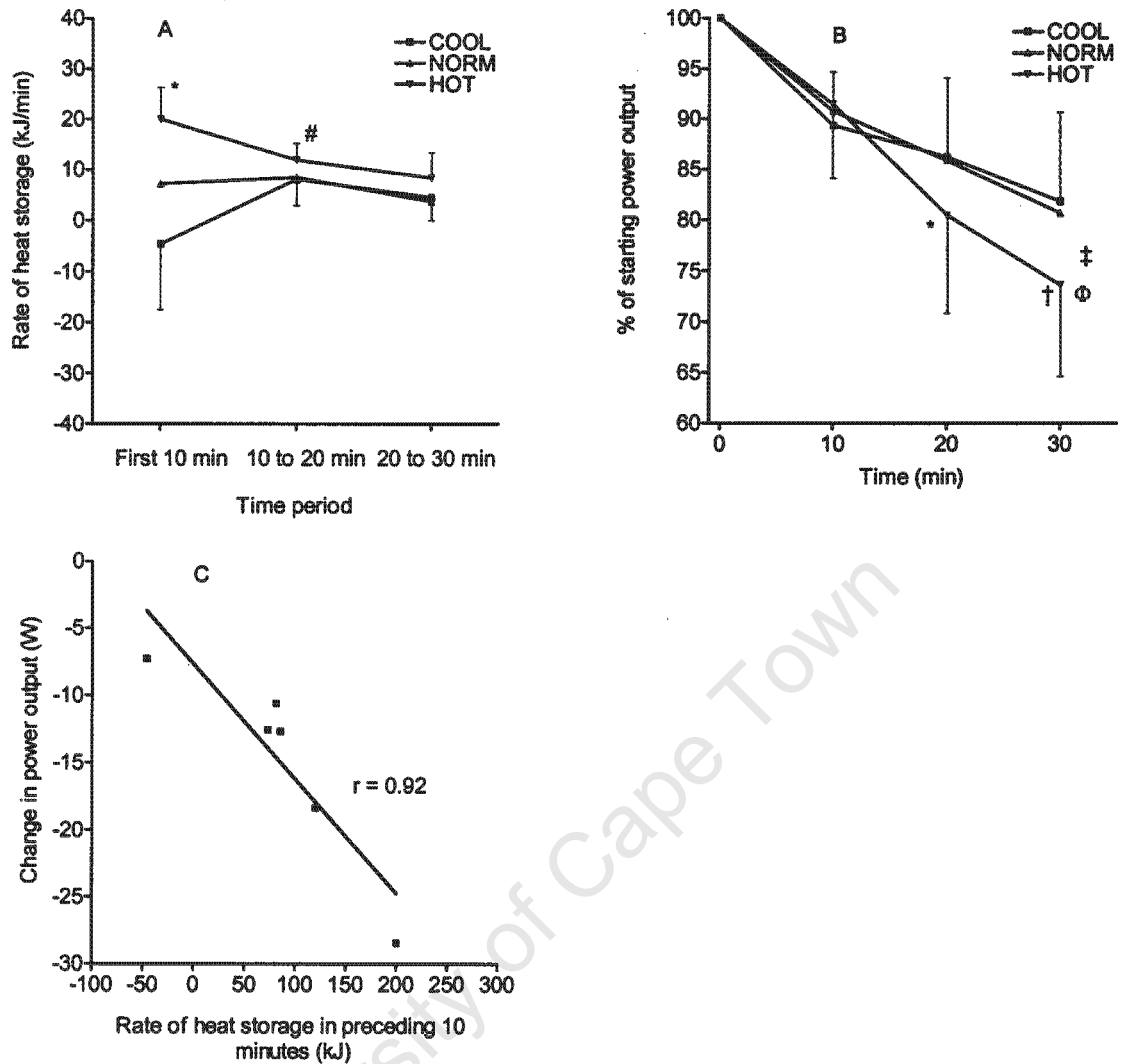


Figure 19. Heat storage (A), power output expressed over 10 minute intervals (B) and change in power output plotted against rate of heat storage in the preceding 10 minute interval during trials in HOT, NORM and COOL.

Values are means \pm S.D. for eight subjects. * Significantly different from COOL and NORM ($P < 0.0005$). # Significantly different from first 10 minutes in HOT and COOL ($P < 0.05$). † Significantly different from 20 minutes in HOT ($P < 0.05$). ‡ Significantly lower than in COOL at 30 minutes. † Significant time main effect, decrease over time in all conditions ($P < 0.005$).

Thermoregulatory variables

Starting rectal temperatures were 37.5 ± 0.2 , 37.5 ± 0.2 and $37.6 \pm 0.3^\circ\text{C}$ for COOL, NORM and HOT respectively. Rectal temperatures increased significantly during the first 22 minutes of the trial, but there was no difference between HOT, COOL and NORM conditions (Fig. 20). When rectal temperature was expressed as a percent of total trial duration, there was no difference between HOT, NORM and COOL during the first 80% of the trials. At 90% and 100%, the rectal temperatures were significantly higher in the HOT condition ($P < 0.05$). The highest rectal temperature recorded in HOT was $39.1 \pm 0.6^\circ\text{C}$. Mean skin temperatures increased upon exposure to the hot environment and decreased in COOL, resulting in significantly higher skin temperatures in the HOT compared to the NORM and COOL conditions ($P < 0.05$, Fig. 21). Initial heat content was not different between conditions, but as a result of the significantly higher skin temperatures from the first minute onwards in the HOT condition, the heat content was significantly greater in the HOT condition throughout the trial ($P < 0.01$, Trial effect).

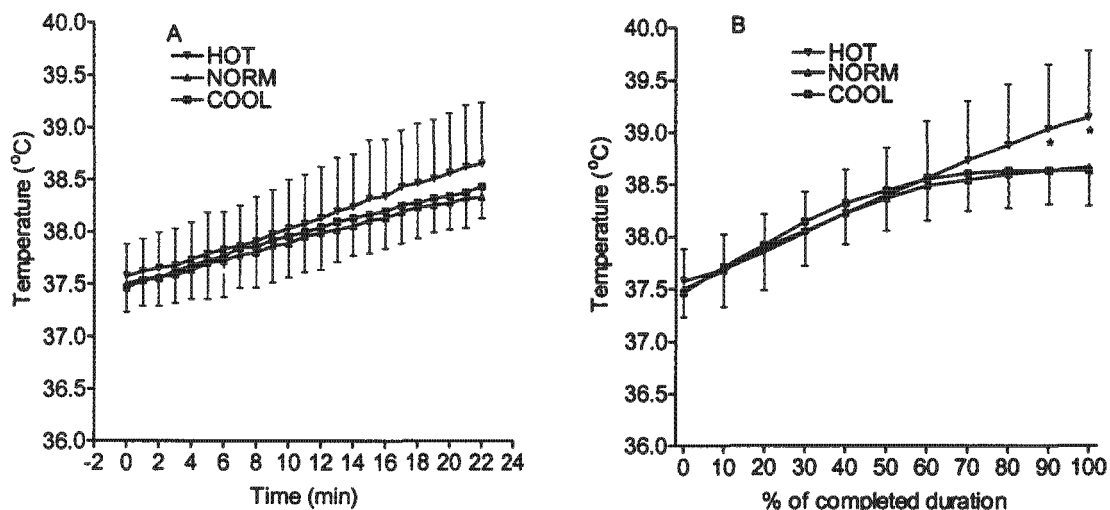


Figure 20: Rectal temperature during the first 22 minutes (A) and relative to total trial duration (B) in HOT, NORM and COOL conditions.

Values are means \pm S.D. for eight subjects. * Significantly different from NORM and COOL ($P < 0.05$)

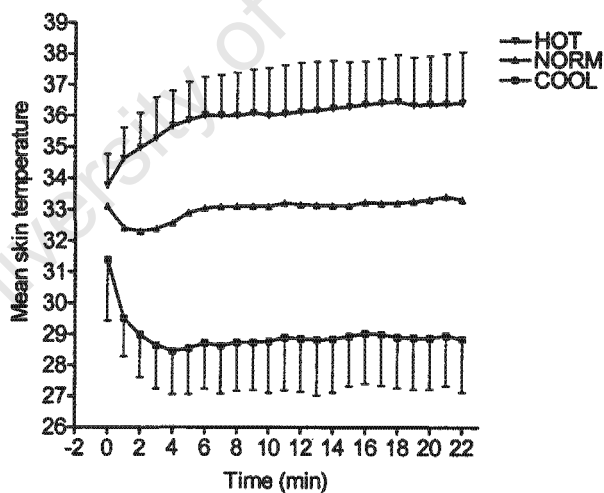


Figure 21: Skin temperatures during the first 22 minutes during trials in HOT, NORM and COOL conditions

Values are means \pm S.D. for eight subjects ($P < 0.00001$, group main effect)

IEMG activity

IEMG amplitude was not different at the start of the trials, and no significant differences were found during the first 22 minutes of trials between conditions (Fig. 22A). Expressed relative to total trial duration, IEMG activity decreased significantly in HOT, but not in NORM and COOL and the IEMG amplitude was lower in HOT than in COOL at 60, 90, and 100% of total trial duration (P < 0.05, Fig. 22B). During the final 30 second sprint, IEMG activity increased significantly in all conditions (P < 0.0001). There were no differences between conditions during the 30 second sprint.

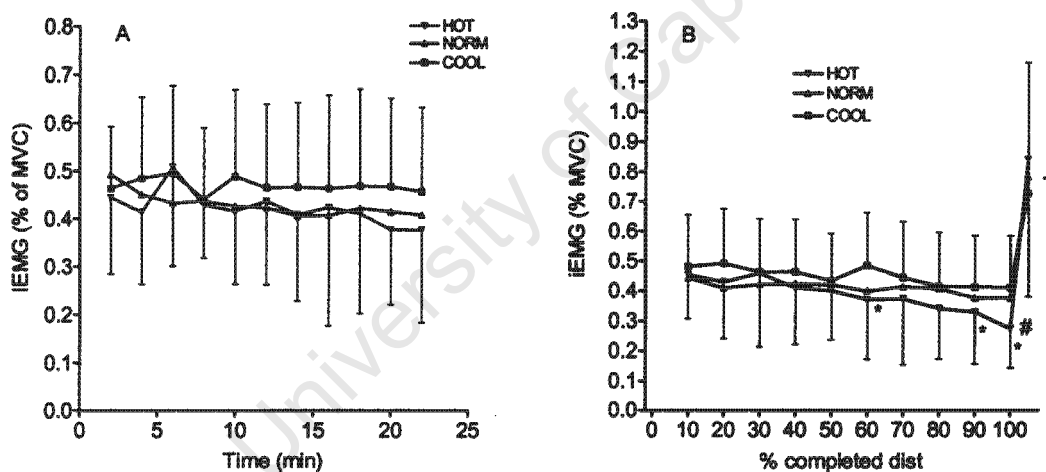


Figure 22: IEMG activity during the first 22 minutes (A) and relative to total trial duration (B) in HOT, NORM and COOL conditions.

Values are means \pm S.D. for eight subjects. * Significantly different from COOL (P < 0.05). # Significant decrease over time in HOT (P < 0.05). † Significant increase in all conditions (P < 0.0001)

Heart rates and Thermal Comfort

Fig. 23A shows heart rate during the three trials. Mean heart rate was significantly higher in the HOT compared to NORM and COOL ($P < 0.05$).

Fig. 23B shows that thermal comfort scores were significantly higher in the HOT compared to the COOL and NORM conditions ($P < 0.01$). Similarly, thermal comfort scores were significantly lower in the COOL compared to the NORM conditions ($P < 0.01$). Mean thermal comfort scores were 3.26 ± 0.85 , 4.76 ± 0.49 and 5.95 ± 1.05 for COOL, NORM and HOT respectively.

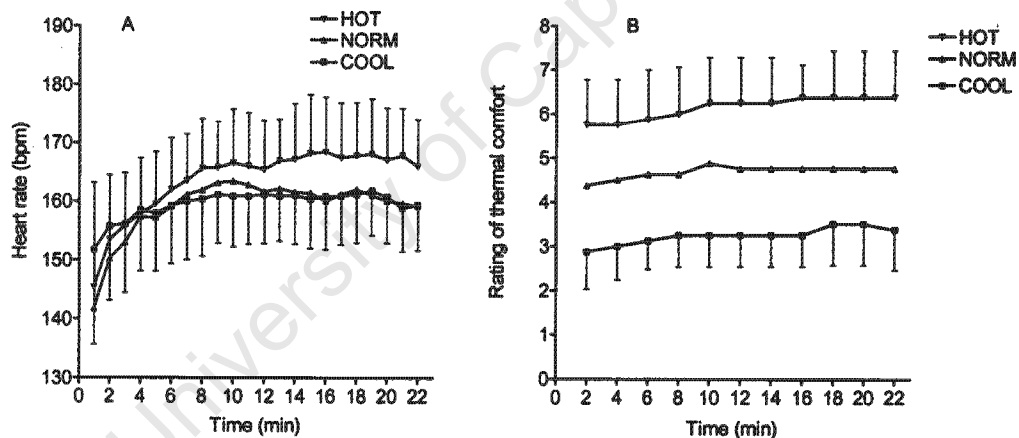


Figure 23: Heart rates (A) and thermal comfort scores (B) during trials in HOT, NORM and COOL conditions.

Values are means \pm S.D. for eight subjects ($P < 0.0005$, Group main effect)

Discussion

Currently, two different exercise models are used to evaluate the impairment of exercise performance in the heat. In the first model, the exercise workrate is fixed, and fatigue occurs when a critically high core temperature is reached, resulting in a failure to maintain the required level of skeletal muscle recruitment necessary to maintain the work rate (Bruck & Olschewski 1987; Nybo & Nielsen 2001a; Nybo & Nielsen 2001b). Fatigue is thus the consequence of excessive heat accumulation which ultimately forces a centrally-mediated reduction in exercise intensity (Nielsen, 1996; Nybo & Nielsen 2001a). In the second model, developed during Chapter 2 of the present thesis, exercise is self-paced, and exercise workrate (Tatterson *et al.* 2000; Marino *et al.* 2004) or force output (Morrison *et al.* 2004) are reduced in advance of excessive heat accumulation to ensure that a catastrophic rise in body temperature does not occur. In this model, skeletal muscle power output does not decline as a direct result of high body temperatures, but is instead regulated specifically to prevent excessive heat storage. The present chapter used a third exercise model to evaluate the regulation of self-paced exercise performance in the heat. Subjects were allowed to adjust their workrate only to maintain their subject perception of effort (RPE) at a fixed, predetermined level, in order to evaluate the interdependencies of power output, the rate of heat storage and the development of fatigue.

The first important finding of this study was that during exercise at a fixed RPE, the work rate decreased as a linear function of exercise duration (Fig. 17) and the absolute rate of decline was fastest in the HOT condition, resulting in a significantly shorter exercise time in the heat, a characteristic finding (Nielsen *et al.* 1984). This suggests that the RPE is not simply a marker of exercise intensity, since power output was falling at different rates in the different environmental conditions even though the RPE was fixed and identical. This linear decrease in power output in the present study began shortly after the onset of exercise (Fig. 17), suggesting that exercise performance is altered well in advance of a critical increase in body temperature (Fig. 20). The present finding therefore supports the existence of anticipatory regulation of exercise intensity, which occurs before the body temperature is abnormally elevated (Chapter 2) (Marino *et al.* 2000; Tatterson *et al.* 2000; Marino, 2004; Marino *et al.* 2004), rather than a model which proposes that reductions in performance occur only body temperature has risen to critical levels as a result of excessive heat storage (Nielsen, 1996; Nybo & Nielsen 2001a).

The second important finding was that the rate of heat storage was different only during the first four minutes of exercise in the different environmental conditions, after which time the rate of heat storage was similar despite a difference in ambient temperature of 20°C between the HOT and COOL conditions (Fig. 18A). Equal rates of heat storage during exercise in very different environmental conditions are achieved by matching rates of heat production to the different rates

of heat loss achievable in the different environmental conditions, and it is proposed that this is the result of alterations in intensity in the present study.

The rectal temperature in the present study reached a plateau value of 38.5°C in COOL conditions after approximately 60% of the exercise duration (Fig. 20B).

However, in the HOT condition, the rectal temperature continued to rise throughout exercise, reaching a value of 39.1°C (Fig. 20B) when the trial was terminated. In order for the rectal temperature to continue rising so that it eventually reached the heatstroke range of 41°C, exercise would have had to continue at this same rate of heat storage for a total of 76 minutes (extrapolated from the linear increase in rectal temperature in the HOT condition in Fig. 20A), more than twice the actual exercise duration. Assuming the power output decreased at the same rate during this time, the power output after 76 minutes would be only 70 W (or 27% of starting power output). It seems more probable that prior to reaching that temperature, the power output and hence rate of heat storage, would have declined sufficiently that the rectal temperature would have stabilized.

However, an essential requirement in order for rates of heat storage and body temperatures to be regulated in anticipation of a limiting hyperthermia is that the precise duration of exercise must be known. In the present study, this was not the case, and it is therefore speculated that the exercise intensity was adjusted to ensure that the rate of heat storage was close to zero in all environmental

conditions (Fig. 19A). The high rate of heat storage during the first 10 minutes of the HOT trial led to an anticipatory reduction in power output which reduced the rate of heat storage to a level that would not produce a lethal hyperthermia.

Indeed, in all environmental conditions, the rate of heat storage in the previous ten minutes predicted the reduction in power output in the following ten minute exercise period (Fig. 19C). This indicates that the reduction in exercise intensity during a subsequent 10 minute period results from calculations based on the rate of heat storage during the previous 10 minutes, as part of feedback-feedforward control.

The final important finding was the IEMG activity decreased during exercise in all conditions (Fig. 22), although the fall was significant only in the HOT condition.

The reduction in IEMG activity in the HOT condition occurred before the core temperature reached the critical value (Nielsen *et al.* 2001; Nybo & Nielsen 2001a; Drust *et al.* 2005) and is similar to the finding reported in Chapter 2. It suggests that impaired performance in the heat is at least in part the result of reduced skeletal muscle activation based on sensory feedback to the central controller.

That IEMG activity began to fall early in the exercise bout in the HOT condition when the rectal temperatures were the same in all conditions (Fig. 20A) indicates that this was not due to a direct effect of temperature on the brain, but occurred in anticipation of a critical rise in temperature.

In COOL and NORM, the decrease in IEMG activity was not significant. It may be that the reduction in skeletal muscle activation levels occurred to a lesser extent in these cooler conditions, a possibility that is supported by the finding that power output declined less in these conditions. That is, the relationship between IEMG activity and power output is influenced by numerous factors, including changes in peripheral metabolite levels. It is thus possible that a small reduction in power output could occur in the absence of changes in IEMG activity, resulting in 'myographical signs of muscle fatigue' (Kayser *et al.* 1994). I do not dispute that such changes are responsible, at least in part, for the observed reduction in power output.

However, the important point is that during a 30 second sprint at the end of the trial, both IEMG activity (Fig. 22B) and power output increase significantly in all three conditions. Indeed, power output increased three fold, whereas the IEMG activity doubled compared to values measured at trial completion. Thus, the power output decreased during the RPE clamp protocol in all conditions despite the presence of a sizeable reserve capacity for force output and skeletal muscle recruitment. This suggests that the reductions in power output and in IEMG activity were not the direct result of any failure of either muscle function or motor unit recruitment, but must instead be part of a regulatory process which maintains a motor unit reserve and is sensitive to hot ambient conditions or high rates of heat storage shortly after the initiation of exercise.

The totality of the present results support the hypothesis that the rate of heat storage, determined by skin and rectal temperatures, is the sensed variable that regulates the exercise response in all environmental conditions when the endpoint is unknown before the exercise bout begins. This is most apparent when exercise in HOT and COOL conditions is compared. Thus, upon first exposure to the hot environment, the significant increase in skin temperature produces a significantly higher rate of heat storage in the first few minutes in the HOT condition. Power output did not however change differently between conditions, until 10 minutes onwards, when the power output decreased more rapidly in the HOT trial, leading to an overall impairment in performance (Fig. 16A). The change in power output was thus predicted by the rate of heat storage during the preceding ten minute interval in all three conditions (Fig. 19C), suggesting that the continual regulation of power output is based on feedback that measures the rate of heat storage.

Since the work rate in this study was selected on the basis of the subject's perception of effort, the finding that the initial power output and the early decline in power output were not different between conditions indicates that the regulation of the work output is initially feedforward and determined by factors other than heat-related variables. However, the workrate was then altered during exercise, on the basis of continual afferent feedback from temperature sensors and the rate of heat storage. The novel aspect of the present study was that any changes in workrate were achieved specifically to maintain the RPE at the

predetermined level, suggesting that afferent feedback, RPE and exercise workrate are interdependent.

It is therefore suggested that during exercise in the heat, afferent feedback in the form of increasing skin temperatures and the significantly higher rate of heat storage in HOT result in a relative 'up-regulation' of the RPE. However, since the RPE must be kept constant in this RPE clamp protocol, the power output declined more rapidly in HOT than in NORM and COOL. As a result, the rate of heat storage was reduced, and from five minutes onwards, there were no differences in the rates of heat storage, and hence in body temperature changes, until completion of the trials. This was demonstrated by a similar increase in the rectal temperature in all trials (Fig. 20). The regulation of exercise intensity is thus the result of both afferent feedback signalling and a feedforward anticipatory strategy which serves to defend thermal homeostasis whilst using the RPE as the mediator of this response.

Previous research supports the concept that the RPE is sensitive to changes in core and skin temperature, and hence to changes in the rate of heat storage. Nybo & Nielsen (2001b) have shown a close correlation between changes in brain electrical rhythms, RPE and increases in body temperature indicating that sensations of fatigue can be explained by changes occurring in the brain. It is known that exercise in the heat at a predetermined and fixed workrate is accompanied by elevated ratings of perceived exertion compared to exercise in

cool conditions (Galloway & Maughan 1997; Nybo & Nielsen 2001a), and that increases in the rating of perceived exertion are closely correlated with core temperature during passive heating (Gonzalez-Alonso *et al.* 1999; Armada-da-Silva *et al.* 2004). Head cooling during exercise reduces thermal strain (Nunneley *et al.* 1982) and RPE (Armada-da-Silva *et al.* 2004) without altering rectal or brain temperatures or hypothalamic function (Nybo *et al.* 2002). It has been suggested that this effect is mediated by sensory information from skin thermoreceptors (Armada-da-Silva *et al.* 2004).

Marino *et al.* (2004) have found that African and Caucasian runners ran an 8 km time-trial in hot conditions at similar ratings of perceived exertion, but the African runners maintained a faster running speed at a given RPE (Marino *et al.* 2004). This was attributed to the smaller body size of the Africans, which resulted in a reduced rate of heat storage at a given running speed (Dennis & Noakes 1999). This identifies the relationship between the RPE and heat storage as an important component in the regulation of exercise intensity during self-paced exercise, since excessively high RPE values associated with inappropriately high rates of heat storage would have developed in the larger Caucasian runners had they attempted to run faster. Furthermore, differences in running speed in the HOT trial were measured within the first minutes of the time-trials, confirming the presence of an anticipatory (feedforward) component of this regulation.

Chapter 2 of the present thesis described the same phenomenon during cycling exercise in hot and cool conditions, while in Chapter 3, it was shown that the RPE increased at a similar rate in hyperoxia and normoxia despite differences in pacing strategy, suggesting that the proposed role for the RPE as a mediator of self-paced exercise is applicable to exercise in different conditions.

Further support for the hypothesis that the RPE is an important mediator of exercise in the heat comes from the study of Watson *et al.* (2005), who found that the administration of a dopamine/noradrenaline reuptake inhibitor improved cycling performance in the heat. It was suggested that the administration of the drug acted on dopamine and noradrenaline neurotransmission to maintain arousal, motivation and reward (Watson *et al.* 2005), which would be reflected in a reduced RPE for a given exercise intensity. However, that study found that the RPE during exercise was similar whether or not the re-uptake inhibitor had been ingested. Since the exercise was self-paced, subjects clearly chose to increase their power output when using the drug, thereby exercising at a higher intensity but at the same RPE. Thus, the drug-induced 'downregulation' of RPE allowed the power output to be increased at the level of discomfort (RPE) that the subjects were voluntarily prepared to sustain during exercise. However, the drug did not increase the level of discomfort subjects were prepared to accept during exercise. The undesirable consequence was that temperature rose more rapidly in the drug trial, indicating i) that homeostatic regulation had been over-ridden, and ii) the rating of perceived exertion plays a role in the homeostatic regulation.

In summary, the present study provides evidence that the regulation of exercise intensity in the heat is achieved through a combination of afferent feedback from the skin and perhaps blood thermoreceptors and the rate of heat storage and a feedforward calculation of the rate of heat storage, which, together, maintain thermal homeostasis by reducing the exercise workrate specifically to ensure that excessive heat accumulation does not occur. This shows that exercise is regulated in anticipation by a complex, intelligent system (St Clair Gibson & Noakes 2004).

University of Cape Town

Chapter 5

***Skeletal muscle electrical activity is
not maximal at maximal volitional
effort/exhaustion***

University of Cape Town

Introduction

Chapters 2, 3 and 4 of the present thesis have provided evidence that self-paced exercise performance is regulated in advance of the development of limiting homeostatic changes. This regulation occurs through a complex, intelligent system, which maintains a sub-maximal level of skeletal muscle recruitment during exercise. The characteristic pacing strategy observed during self-paced exercise is thus the result of an anticipatory system that reduces muscle recruitment as a result of afferent feedback during exercise. I have suggested a role for the rating of perceived exertion (RPE) as a mediator in this process (Chapter 4).

While this theory has been tested during self-paced endurance exercise (Chapter 2 and 3) and using a novel RPE-clamp protocol (Chapter 4), it may not adequately explain how maximal exercise to exhaustion is regulated. This type of exercise, in which the exercising athlete is forced to exercise at progressively increasing workrates until volitional fatigue, is often used to measure the maximal oxygen consumption. It has been proposed that fatigue occurs due to a limitation in oxygen delivery to the exercising muscles (Bassett & Howley 1997; Bassett & Howley 2000). This model holds that when oxygen delivery to the exercising muscles reaches a limit, a developing skeletal muscle anaerobiosis causes an increase in muscle lactic acid production (Taylor *et al.* 1997). This lactic acidosis then directly impairs skeletal muscle contractile function causing the termination of exercise. This model has been termed the A.V Hill

Cardiovascular/Anaerobic/Catastrophic model after its originator (Noakes, 1998; Noakes, 2000; Noakes & St Clair Gibson 2004).

An unrecognized prediction of this model is that this effect can only occur after full motor unit recruitment has been achieved, since there is no known biological mechanism, or any postulated, by which skeletal muscle anaerobiosis and lactic acidosis can inhibit the function of quiescent motor units that have yet to be recruited by the motor cortex. Rather, logic demands that if skeletal muscle function is indeed regulated by such metabolic events occurring in the periphery, then any potential influence of central (brain) factors must already have been surpassed. In particular, there must be complete skeletal muscle activation. For in the absence of maximal activation, central (brain) command should be able to further increase the power output by activating those remaining motor units that are quiescent, even if the function of those additionally recruited motor units was somewhat impaired by anaerobiosis and lactate acidosis.

More recently, a contrasting model has been proposed. This central governor model (Noakes *et al.* 2001) predicts that under all circumstances of exercise, the brain will regulate motor unit activation in the exercising skeletal muscles to ensure that all exercise, regardless of intensity or duration, or the environmental conditions in which it is undertaken, will be completed with the maintenance of homeostasis in all bodily systems (Noakes, 2000; Noakes & St Clair Gibson 2004; Noakes *et al.* 2005). This model therefore predicts that since subjects are able to

increase their power outputs beyond those at which the peak power output is measured, the peak power output test will terminate at submaximal values of muscle activation.

To our knowledge, only one study has quantified the extent of motor unit activation during progressive exercise up to and above the peak power output. In 1974, Henriksson and Bonde-Petersen measured EMG activity as an index of motor unit activation in the vastus lateralis and rectus femoris muscles during cycling at six workloads between 25 and 120% of the peak sustainable power output at which VO_2 max was measured (Henriksson & Bonde-Petersen 1974). It was found that EMG activity increased linearly with increasing power output ($r = 0.96$). At a workload corresponding to 120%, the EMG activity in the vastus lateralis and rectus femoris muscles was 126% and 118% of that measured at the maximal power output (Henriksson & Bonde-Petersen 1974). The authors concluded that the quadriceps muscle is recruited proportionally to changes in oxygen uptake. They drew no conclusion from their findings that EMG activity continued to increase at workrates above the peak power output measured during the progressive trials to exhaustions.

Accordingly, the aim of this study was to quantify the relationship between EMG activity and power output over a wider range of power outputs than tested in the study of Henriksson and Bonde-Pedersen (1974). Specifically, I aimed to determine whether, considering the multiple limitations of this method (Farina *et*

al. 2004), the use of surface EMG measurements would be able to distinguish between the two theories for fatigue. Surface EMG activity was measured during intermittent, short-duration cycle exercise at power outputs up to 200% of the peak sustainable power output achieved during progressive incremental cycling exercise to exhaustion.

It was hypothesized that the EMG activity would continue to increase linearly across the range of power outputs of which our subjects were volitionally capable.

This finding would indicate that motor unit recruitment is sub-maximal at volitional fatigue, as predicted by the central governor model (Noakes & St Clair Gibson 2004; Noakes *et al.* 2005), and as suggested by the pioneering findings of Henriksson and Bonde-Pedersen (1974).

Methods

Ten healthy male cyclists were recruited from local cycling clubs and gymnasia to participate in the study. All subjects were well trained and were fully informed of the risks associated with the study. Subjects signed an informed consent before commencing the study, The study was approved by the Research and Ethics Committee of the Faculty of Health Sciences of the University of Cape Town.

Experimental procedure

Maximal voluntary contraction testing

Upon reporting to the laboratory, subjects performed a maximum voluntary contraction (MVC) for normalization of the EMG signal obtained during the subsequent cycling bouts. This method for normalization of the EMG signal has been shown to be valid during maximal (Hunter *et al.* 2002b; Hunter *et al.* 2003) and sub-maximal (St Clair Gibson *et al.* 2001b; Tucker *et al.* 2004) cycling exercise.

The right knee extensor muscle strength of the subject was measured on an isokinetic dynamometer (Bio-Dex dynamometer, Bio-Dex, United Kingdom), while the electromyographic (EMG) activity of the vastus lateralis and vastus medialis muscles was recorded. Subjects were firmly strapped into the dynamometer and the right leg attached to the arm of the dynamometer at a level slightly above the lateral malleolus. The arm of the dynamometer was set so that the knee was at a 60° angle from full leg extension. Each subject performed a standardized warmup consisting of four sub-maximal contractions prior to performing two five-second maximal contractions, separated by a one minute recovery period. Subjects were verbally encouraged to exert the maximal possible force during each contraction. The EMG activity recorded from the contraction producing the highest force was used for normalization of the EMG signal obtained during the subsequent cycling trials.

EMG testing

During each MVC and the subsequent cycling trials, the EMG activity of the vastus lateralis and vastus medialis muscles was recorded. Before placement of the electrodes, the skin was shaved and cleaned with 95% ethanol, according to methods previously described (Kay *et al.* 2001; St Clair Gibson *et al.* 2001b). A triode electrode (Thought Technology, West Chazy, N.Y., USA) was placed over the belly of the vastus lateralis and medialis muscle and connected to a pre-amplifier. Outputs from the pre-amplifier were relayed to a Flexcomp/DSP EMG apparatus (Thought Technology USA) via a fiber optic cable and stored by an online computer. EMG signals were captured at 1984 Hz during the MVC and the cycling trials. EMG activity was analysed for five-second periods during the MVC and for five second periods at each workload during the trials. The raw EMG signals were full wave rectified, movement artefact removed using a high-pass second order Butterworth filter with a cut off frequency of 15 Hz, then smoothed with a low-pass second order Butterworth filter with a cut-off frequency of 5 Hz. This was performed using MATLAB™ software.

All processed EMG data were normalized by dividing the EMG value obtained at each workload during the trials by the EMG value obtained during the MVC performed before the start of the trial. EMG data are therefore expressed as a percentage of the EMG measured during the MVC. While it is acknowledged that the MVC is not representative of the cycling movement, and hence no direct

inferences regarding the normalized values can be made. This method of EMG normalization has previously been shown to be valid for use in cycling trials, (Hunter *et al.* 2002b), and the EMG measurements during self-paced cycling in the heat are reproducible between trials using this methodology (Kay *et al.* 2001).

Incremental cycling exercise to fatigue

All cycling trials were performed on an electrically braked cycle ergometer (Lode, Groningen, Holland). For the first cycling bout, subjects performed a peak power output trial (PPO). During this trial, subjects began cycling at a workload of 220W and were required to maintain a cadence between 85 and 90 revolutions per minute. This workload was increased by 20W every minute, until the subject was no longer able to maintain a cadence of at least 85 rpm. This cadence was selected since the subjects would be required to maintain the same cadence during the subsequent intermittent supramaximal trials (described later). In those trials, a high cadence is necessary to produce the required workrate. The PPO was calculated as the highest completed workload, plus the fraction of time spent in the final incomplete workload.

During all trials, subjects were encouraged to perform maximally. This method of PPO testing is typically used for the determination of the maximal oxygen uptake (VO_2max) as well as maximal sustainable power output. During the present study however, oxygen consumption (VO_2) was not measured, since I wanted to ensure

that subjects completed the PPO test maximally, without any restrictions to breathing that may affect their maximal performance. Further, comparisons between the PPO test and the intermittent trials at workrates greatly in excess of the PPO would be made for EMG and not for VO_2 data, since measurements of VO_2 during a twenty second sprint would not reveal any novel information.

EMG activity in the vastus lateralis and vastus medialis was sampled for five second periods during the middle of each workload.

Heart rate was measured continuously during trials (Polar Electro OY, Kempele, Finland), and is reported at the mid-point of each workload.

The rating of perceived exertion was obtained at the mid-point of each workload using the Borg scale for rating of perceived exertion (Borg 1982).

Intermittent supramaximal cycling exercise

Following the PPO trial, subjects rested for ten minutes before performing the intermittent supramaximal bout (SUP). During this trial, subjects were required to cycle for 20 seconds at workloads starting at 320 W (approximately 80% of the anticipated PPO), and increasing by 40W in each interval, up to 1000 W or until the subject could no longer complete the 20 second bout. Between cycling bouts, subjects rested for two minutes to minimize the effects of fatigue on the EMG

signal. It is acknowledged that this two minute rest period, and the ten minute period after the PPO test, would not allow complete recovery, and so the maximal power output achieved during SUP will represent a relative underperformance. However, since the EMG activity would be compared across a range of power outputs both lower and higher than the PPO, the actual peak power output achieved during SUP was not of great consequence.

The workloads were selected to ensure that the workloads measured during the PPO and SUP bouts would overlap to allow comparison of EMG activity at those workloads common to both trials. Thus, if fatigue due to metabolite accumulation during PPO was responsible for increasing the measured EMG signal during SUP, it would be observed when cycling at the same power output during SUP as during PPO. During each 20 second sprint, subjects were required to maintain their cadence at the same level as during the previously described PPO test. It has been found that EMG activity is not significantly altered by changes in cadence (Marsh & Martin 1995; Sarre *et al.* 2003), but cadence was controlled to ensure that any differences in the EMG activity measured during the two cycling bouts could not attributed to changes in cadence.

EMG activity was sampled for the middle five seconds of each 20-second exercise bout. Heart rate was recorded as the highest measured heart rate either during or immediately following completion of each sprint. RPE was measured immediately upon completion of each workload.

Statistical analysis

All statistical analysis was performed using Statistica 6.0 (Statsoft Inc. 1284 – 2001).

Data are presented as means \pm S.D. The slope of the EMG-power output relationship was determined for each measured muscle group during PPO and SUP using linear regression analysis. The slopes were compared between PPO and SUP using a repeated measures ANOVA (Trial X Power output). A Tukey's Post Hoc test was performed where a significant interaction effect was found. Significance was accepted at $P < 0.05$.

Results

The mean peak power output measured during the incremental bouts to fatigue (PPO) was 426 ± 26 W. During the supramaximal cycling bouts (SUP), the mean power output was 905 ± 60 W. All ten subjects were able to reach a minimum power output of 400 W during PPO and 840 W during SUP. Therefore, data are presented for power outputs up to 400 W and 840 W for PPO and SUP respectively.

Figure 24 shows the EMG activity in the vastus lateralis (Fig. 24A) and vastus medialis (Fig. 24B) expressed over the entire range of power outputs from 220 W up to 840W during both PPO and SUP. EMG activity in both muscle groups increased linearly with power output. During PPO, the correlation co-efficients

were calculated as $r^2 = 0.92$ and 0.81 for v. lateralis and v. medialis, respectively.

During SUP, r^2 was calculated as 0.95 for v. lateralis and 0.96 for v. medialis.

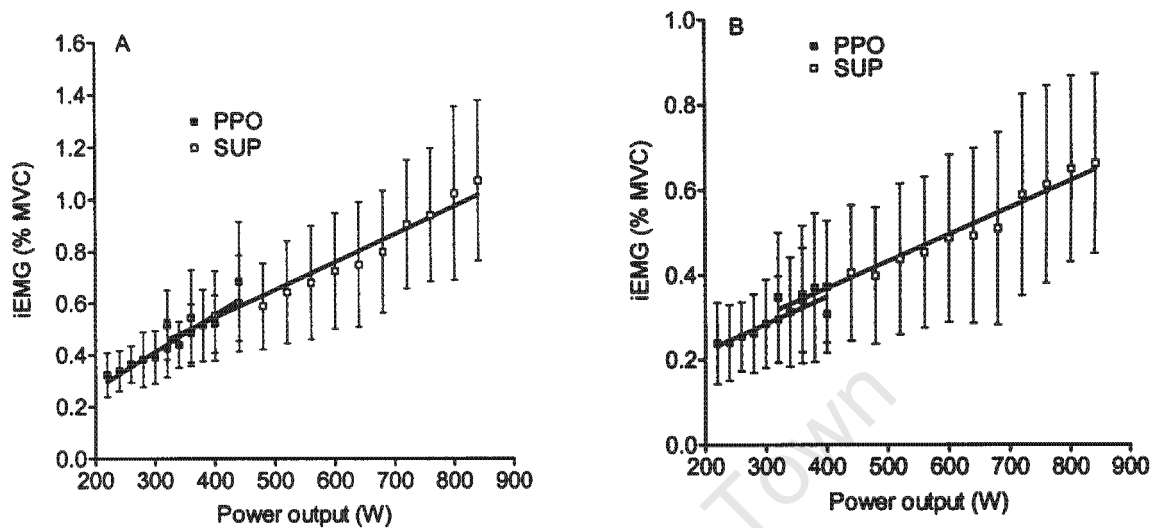


Figure 24. IEMG activity expressed over the range of power outputs measured during incremental exercise to fatigue (PPO) and during intermittent supramaximal exercise (SUP) in vastus lateralis (A) and vastus medialis (B).

Values are means \pm S.D. for ten subjects

For vastus lateralis, the slope of the EMG-Power output relationship during PPO was determined as 0.0014 ± 0.0001 %/W, compared to 0.0011 ± 0.0001 %/W for the slope during the SUP exercise bout. These slopes were not significantly different.

For vastus medialis, the slopes of the EMG-Power output relationship were 0.0053 ± 0.0001 and 0.0063 ± 0.0001 for PPO and SUP respectively. These slopes were not significantly different.

Figure 25 shows the EMG activity measured during SUP expressed as a percentage of the EMG activity measured at the highest power output achieved during PPO. EMG activity continued to increase beyond values measured at volitional fatigue at a power output of 400 W during PPO. At fatigue during SUP, the EMG activity in vastus lateralis was $177 \pm 27\%$ of that measured at fatigue during PPO, while vastus medialis EMG activity increased by a further $91 \pm 28\%$ compared to the value measured at volitional fatigue during PPO.

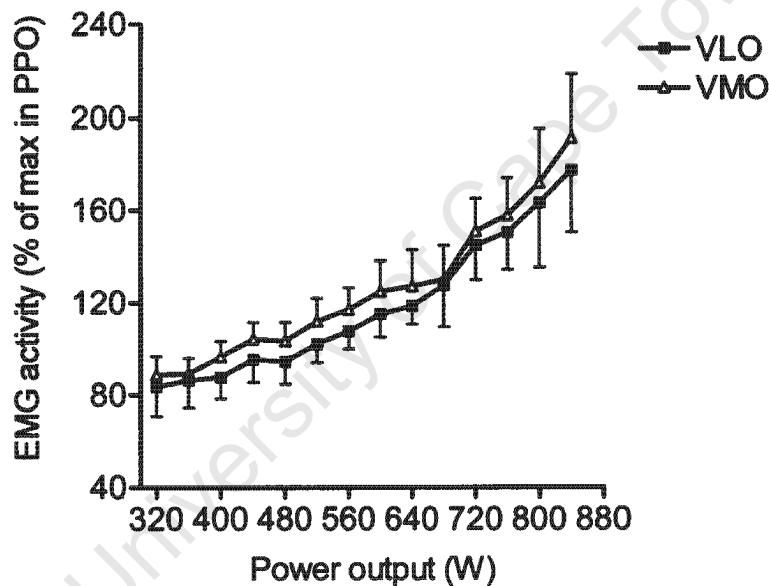


Figure 25. EMG activity in vastus lateralis (VLO) and vastus medialis (VMO) during intermittent supramaximal exercise (SUP) expressed relative to EMG activity measured at the maximal workrate achieved during incremental exercise to fatigue (PPO).

Values are means \pm S.D. for ten subjects

Figure 26A shows the RPE (left panel) and heart rate (right panel) measured during the PPO trial. Figure 26B depicts the RPE (left panel) and heart rate (right

panel) measured during the SUP cycling bouts. Both heart rate and RPE increased significantly with power output, and reached maximal values at the highest workrates sustained during the respective trials.

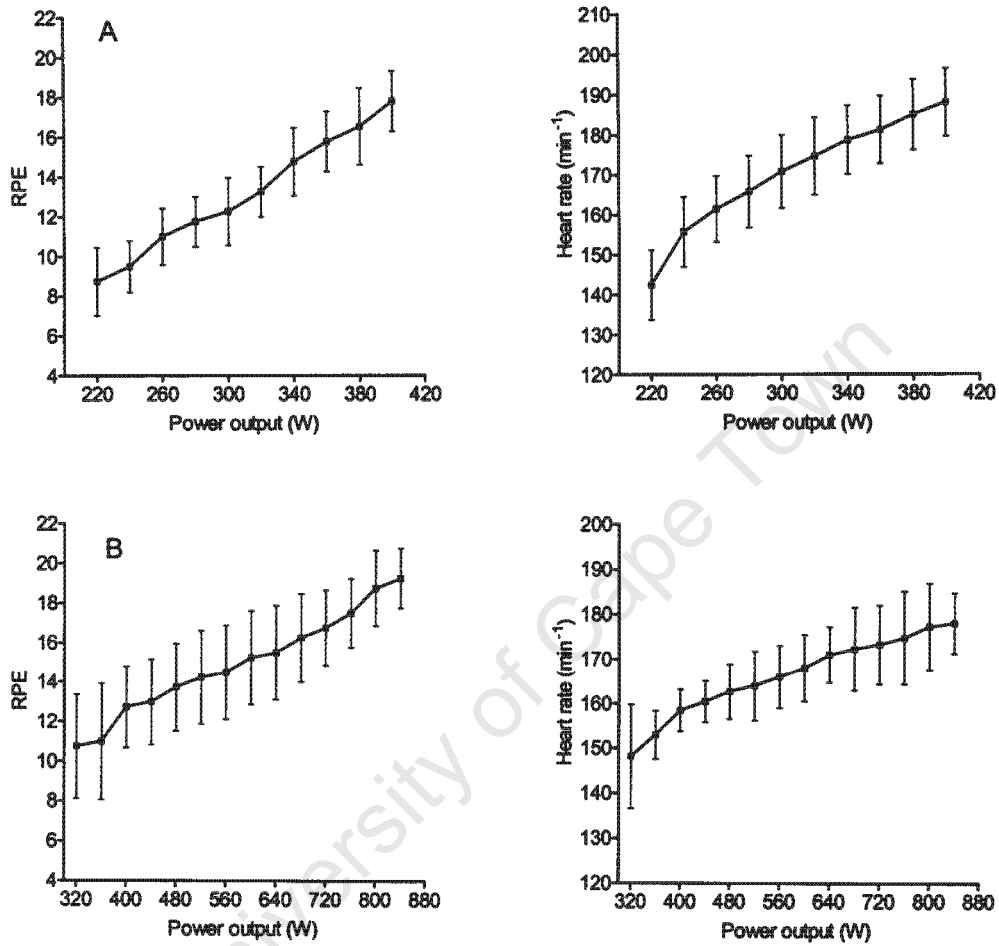


Figure 26. Ratings of perceived exertion (left panel) and heart rates (right panel) measured during incremental exercise to fatigue (PPO, 3A) and during intermittent supramaximal exercise (SUP, 3B).

Values are means \pm S.D. for ten subjects

Discussion

If A.V. Hill's Cardiovascular/Anaerobic/Catastrophic model is correct, then skeletal muscle motor unit recruitment must be maximal at exhaustion during progressive exercise, because a peripheral metabolite like lactic acid cannot regulate the function of motor units that have yet to be recruited by the central nervous system (Noakes & St Clair Gibson 2004; Noakes *et al.* 2005). If motor unit recruitment is maximal during progressive maximal exercise to exhaustion, then surface EMG activity, a measure of skeletal muscle activation levels (Bigland-Ritchie, 1981), should also reach a peak at the peak achieved workrate during progressive exercise with no further capacity to increase muscle activation at any workrate above that 'maximal' workrate.

Accordingly, the first important finding of this study is that EMG activity measured in two different lower limb muscle groups increases as a linear function of the power output at all power outputs of which our subjects were capable (Fig. 24). This confirms and extends the findings of Henriksson and Bonde-Petersen (1974). These authors found that if the workrate was increased beyond the power output which elicited a VO_2max , the EMG activity in vastus lateralis muscle continued to increase. That study (Henriksson & Bonde-Petersen 1974) measured EMG activity at a power output of 120% of the peak sustainable power output. Those findings are now extended to show that EMG activity continues to increase linearly to the very highest workrates of which the present subjects were capable and which

exceeded by 200% the maximal workrates achieved during a continuous peak output test.

The second important finding was therefore that EMG activity was substantially submaximal at volitional fatigue during the PPO (Fig. 25). Indeed, this value was only approximately 50% of the value achieved at the highest workrates of approximately 900W that were sustainable by these athletes for a minimum of 20 seconds. Indeed, it seems highly probable that EMG activity would have increased even further if our subjects had been capable of achieving higher workrates, since there was no evidence that EMG activity had reached a 'plateau', even at these high workrates (Fig. 24 and Fig. 25). Thus, the extent of skeletal muscle activation even during exhaustive supramaximal exercise is sub-maximal.

Previous studies have found that EMG activity increases over time when exercise is performed at a constant workrate (Nummela *et al.* 1992; Kayser *et al.* 1994; Taylor *et al.* 1997). This has been interpreted as a "myographical sign of muscle fatigue" (Kayser, 2003). For example, Nummela *et al.* (1992) found that drop jump performance was impaired by 39% following a maximal 400 m sprint, and the reduction in jump performance was negatively correlated with blood lactate concentration. The EMG activity in the active sprinting muscles also increased significantly over the course of the run and it was concluded that additional motor units were being activated to compensate for the progressive reduction in muscle force production as a result of metabolic acidosis in the muscle (Nummela *et al.*

1992). Taylor *et al.* (1997) have also suggested that an increase in EMG activity during exercise at a constant power output represents an increase in muscle recruitment to maintain a given force output, usually due to a progressive failure in muscle contractility caused by metabolic changes at the muscle level.

While it is acknowledged that factors at the muscle level (Enoka, 1995; Farina *et al.* 2004) can influence the degree of muscle recruitment for a given EMG signal, our finding that EMG activity increases almost two-fold as power output increases beyond the levels measured at volitional fatigue (Fig. 25) can only be interpreted to show that muscle activation was not maximal at volitional fatigue. Regardless of whether the increased EMG activity was produced by increased motor unit firing rates or increased numbers of motor units recruited, the progressive increase in the EMG signal shows that motor activation, however achieved, was not maximal during the so-called maximal aerobic exercise.

We do not therefore dispute the assertion that metabolite accumulation results in a progressive decline in muscle force output (Nummela *et al.* 1992; Taylor *et al.* 1997; Linossier *et al.* 2000). However, the present results suggest that even in the presence of this 'muscle fatigue', the activation of skeletal muscle, and the power output, can still be increased compared to what is achieved at maximal effort during sustained exercise.

Much of the present conclusions are based on the assumption that surface EMG activity provides a reasonable estimation of motor unit activation during dynamic exercise. This has been regarded as a controversial issue, since numerous factors can influence the EMG-Force output relationship (Marsden *et al.* 1983; Bilodeau *et al.* 2003). For example, it has been argued that peripheral changes in the muscle cause the motor neuron firing rate to decrease (MacLaren *et al.* 1989; Hepple, 2002; Westerblad *et al.* 2002), causing slower contraction and relaxation times (Marsden *et al.* 1983; Gandevia, 2001), producing a reduction in EMG that is not associated with a reduced activation level of skeletal muscle, and which is not measured using surface EMG techniques. This process, termed muscle wisdom, actually compensates for the decline in muscle relaxation and contraction speed, enabling force output to be sustained to a greater degree than if the firing rate remained the same. However, this has only been examined during static isometric muscle contractions, and during dynamic exercise such as cycling, where muscle contracts at a rate that is determined by the cycling cadence (which was held within a narrow predefined range in the present study), it is not clear how contraction speed could slow down in this manner.

However, it must still be acknowledged that a decrease in motor neuron firing rates as a result of fatigue can produce a lower measured EMG activity. Similarly, an increase the motor neuron firing rate could increase the amplitude of the measured EMG signal. Thus, in the present study, it is possible that the increase in EMG activity at supramaximal power outputs is due to an increase in firing rate of

the motor neuron, not detected using the present methods. However, considering that the muscle wisdom theory posits that firing rate decreases due to metabolite accumulation, it is difficult to explain how firing rate would be greatest at the end of exercise to fatigue, when metabolite levels are known to be at their highest. Therefore, it is still suggested that the only explanation for the observed increase in EMG amplitude as power output increases beyond the peak sustainable level is that motor unit activation increases, as the brain continues to increase the recruitment of previously inactive motor units.

Furthermore, the process of amplitude cancellation may result in a relative underestimation of the real increase in muscle recruitment (Farina *et al.* 2004). Thus, Farina *et al.* (2004) have suggested that “the amplitude change of the surface EMG actually underestimates the associated change in motor unit activity underlying the modulation of muscle force” (Farina *et al.* 2004). Nevertheless, they acknowledge that “*Both the force exerted by a muscle and the amplitude of the surface EMG depend on the number of recruited motor units (MU's) and the discharge rate of active MU. Accordingly, it is reasonable to expect that muscle force can be estimated from the surface EMG*” (Farina *et al.* 2004).

However, by showing that EMG activity increases as a linear function of work rate across the entire spectrum of power output it is confirmed that: i) skeletal muscle activation increases across the entire range; and ii) that motor unit activation cannot be maximal during maximal ‘aerobic’ exercise, as is required by the model

which holds that such exercise is always limited by the development of skeletal muscle anaerobiosis.

In summary, EMG activity, an accepted indirect measure of the extent of skeletal muscle recruitment (Farina *et al.* 2004) rises as a linear function of work rate, across the complete range of work rates of which our subjects were capable. This indicates that the principal method by which humans increase their skeletal muscle power outputs is through an increase in the extent of motor unit recruitment, in keeping with the traditional teaching in motor control. Since the extent of motor unit recruitment is submaximal during 'maximal' exercise tests which are traditionally used to measure the VO_2 max, the factor 'limiting' such exercise is the extent of motor unit recruitment, which may be regulated in response to physiological variables such as oxygen delivery or lactate acidosis, rather than being the direct result of these changes. This finding supports our hypothesis that human exercise is regulated centrally by a complex system, the function of which is to ensure that exercise terminates before homeostasis fails and a catastrophic organ damage develops.

Chapter 6

An analysis of pacing strategies during men's world record performances in track athletics

The research findings from this chapter have been published as an Original
Investigation in the International Journal of Sports Physiology and Performance 1:
297-309, 2006

Introduction

An optimal pacing strategy refers to the efficient use of physiological resources during athletic competition, and is essential for optimal exercise performance (Foster *et al.* 1993; Foster *et al.* 2003). This optimal pacing strategy depends on factors such as the length of the exercise bout (Ingen Schenau *et al.* 1994; de Koning *et al.* 1999; Foster *et al.* 2004), the type of exercise being performed (Foster *et al.* 1993; Foster *et al.* 2003), ambient temperature (Chapter 2) and altitude (Foster *et al.* 1993). Studies in which the pacing strategy is manipulated by forcing athletes to start either faster or slower than their average speeds for their best performances have found that in shorter events (less than 80 seconds (Ingen Schenau *et al.* 1994)) and in sports such as cycling and speed skating, where resistive drag forces are lower, best performances may occur with a faster start so that kinetic energy is maximized early in the trial (Ingen Schenau *et al.* 1992; Ingen Schenau *et al.* 1994; de Koning *et al.* 1999; Bishop *et al.* 2002; Foster *et al.* 2003). Once high velocities are achieved, a reduction in power output may cause only a small reduction in speed compared to running or rowing (Bishop *et al.* 2002), where higher drag forces result in a greater reduction in speed if power output decreases (Foster *et al.* 2003).

In contrast, performance during longer duration events may be optimized by an even or negative pacing strategy (Foster *et al.* 1993; Bishop *et al.* 2002; Thompson *et al.* 2004), which is often characterized by an increase in the power output or

speed at the end of the event. For example, best performances during a 2000 m cycling trial are achieved when athletes are made to complete the first 1000 m either slightly slower or at the same pace as the second 1000 m (Foster *et al.* 1993). During self-paced laboratory trials lasting approximately 30 minutes, the pacing strategy is characterized by a significant increase in power output (Marino *et al.* 2000; Tatterson *et al.* 2000) or running speed (Marino *et al.* 2004) at the end of the trial.

It has been proposed that the self-selected pacing strategy plays a key role in a complex, regulatory system (Ulmer, 1996), in which a central governor regulates exercise intensity specifically to ensure that potentially catastrophic derangements to cellular function do not occur (St Clair Gibson & Noakes 2004; Lambert *et al.* 2005; Noakes *et al.* 2005). Chapter 2 and Chapter 4 of the present thesis have found that power output and skeletal muscle motor unit activation decrease soon after the onset of self-paced cycling trials in hot compared to cool environments, even though body temperature, heart rate or perceived exertion were not yet different between conditions. Similarly, pacing strategy is altered when the oxygen content of the inspired air is either increased (Chapter 3) or decreased (Brosnan *et al.* 2000), supporting the notion that the observed changes in power output or exercise intensity during self-paced exercise serve a regulatory function.

Therefore, understanding the pacing strategies of elite athletes during competitive or maximal exercise may allow insight into the underlying physiological and

regulatory processes. We have previously analysed 32 world record performances from the mile event (Noakes and Lambert, in review), and found that the first and final laps were significantly faster than the second and third laps. In contrast, in the sprint events (100, 200 and 400 m) at the 7th IAAF World Athletics Championships, the fastest split times for every single athlete ($n = 16$ for each event) were recorded early on and running speed decreased progressively until the finish (Ferro *et al.* 2001).

Accordingly, the aim of the present study was to describe the pacing strategy adopted during world record performances for the 800 m, 5000 m and 10 000 m events for males. It was hypothesized that in the longer distance events (5000 m and 10 000 m), the running speed would decline during the middle portion of the race before increasing again at the end of the race, with the fastest speeds occurring at the end of the race. In contrast, in the shorter event, the 800 m (< 110 s), the first lap would be significantly faster than the second lap, resulting in an overall positive pacing strategy.

Methods

Overall performances and split times for male world record performances for the 800 m, 5000 m and 10 000 m events were obtained. In the 800 m event, lap times were analysed for 26 world record performances from 1912 to 1997. Twelve of these world record performances had data for 200 m intervals. In the 5 000 m

and 10 000 m events, times for each kilometer were analysed for 32 (1922 to 2004) and 34 (1921 to 2004) world records respectively. While it is acknowledged that some world records may be set in competitive races where tactics may influence the pacing strategy, it is considered that generally, these races utilize pacemakers in structured, planned world record attempts, and so represent maximal efforts.

Statistical analysis

An analysis of variance with repeated measures and a Tukey's HSD post hoc test were used to determine differences in lap times for the 800 m event and kilometer times for the 5 000 and 10 000 m events. Statistical significance was accepted as $P < 0.05$. Values are shown as means \pm S.D.

Results

800 m event

Figure 27 depicts the time for each lap from 26 world record performances. The second lap was significantly slower than the first lap (52.0 ± 1.7 s vs 54.4 ± 4.9 s, $P < 0.00005$). In only two of the 26 world records set since 1912, has the second lap been faster than the first lap, once in 1966 (Jim Ryun) and again in 1972 (Dave Wottle).

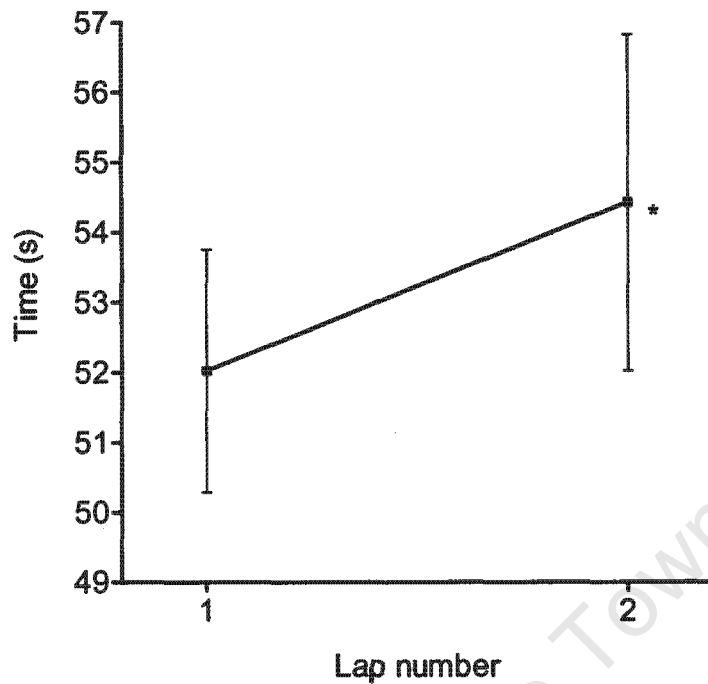


Figure 27: Lap times from 26 world record performances in the 800 m event

Values are means \pm S.D. for 26 performances. * Significantly different from first lap ($P < 0.0005$)

The times from the 200 m intervals of 12 world record performances are shown in Figure 28. The second and fourth 200 m intervals were significantly slower than the first and third 200 m interval ($P < 0.05$).

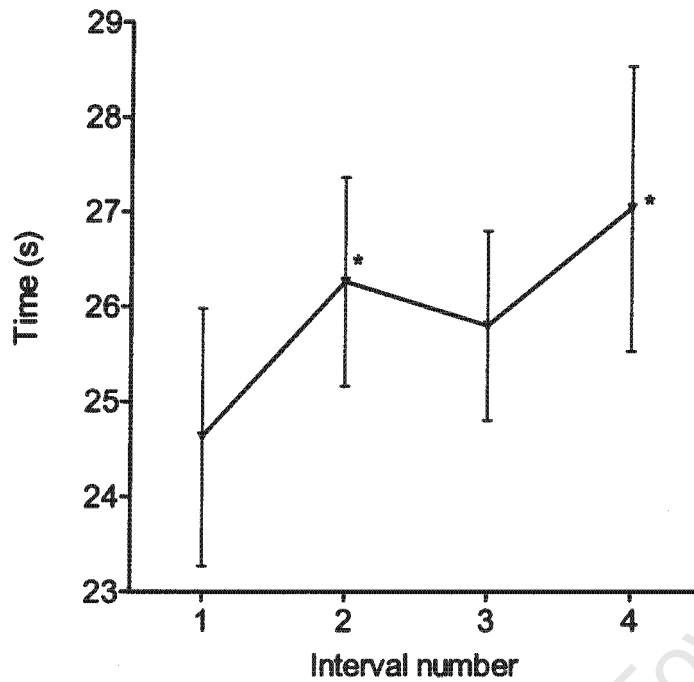


Figure 28: 200 m interval times from 12 world records in the 800 m.

Values are means \pm S.D. for 12 performances. * Significantly different from intervals 1 and 3 ($P < 0.05$)

5 000 m event

The kilometer splits from 32 world record performances are shown in Figure 29.

The first and final kilometers were significantly faster than kilometers 2, 3 and 4 ($P < 0.005$).

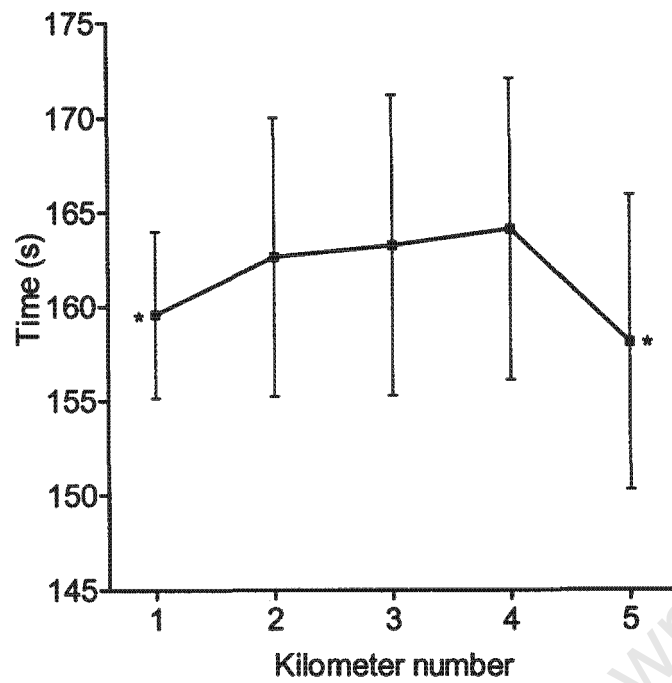


Figure 29: Kilometer times from 32 world record performances in the 5000 m event.

Values are means \pm S.D. for 32 performances. * Significantly different from kilometers 2, 3 and 4 ($P < 0.005$)

10 000 m event

Figure 30 depicts the kilometer times from 34 world record performances in the 10 000 m event. The kilometer times increased progressively after the start, and became significantly greater than at the start from 3 km onwards, before decreasing significantly in the final kilometer, resulting in the final kilometer being the fastest of the race ($P < 0.005$).

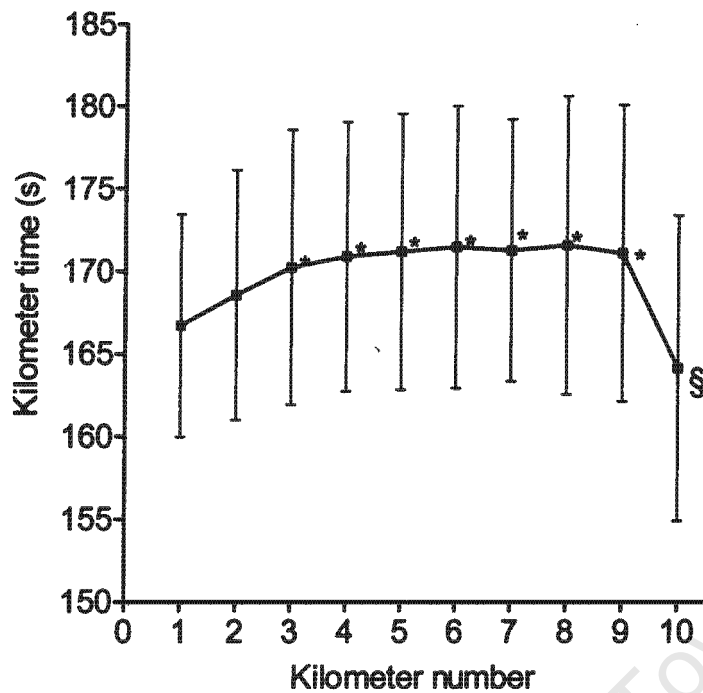


Figure 30: Kilometer times from 34 world record performances in the 10 000 m event.

Values are means \pm S.D. for 34 performances. * Significantly slower than kilometers 1, 2 and 10 ($P < 0.05$); § Significantly faster than the preceding 9 km ($P < 0.005$)

The average running speeds for each event are shown in Figure 31. For the 800 m event, the interval refers to each lap, while for the 5 000 and 10 000 m events, running speeds are plotted for each kilometer. In the 800 m event, the overall pacing strategy is positive, with a reduction in running speed in the final interval, compared to the 5 000 and 10 000 m events, where running speed increases significantly at the end of the race. I have also included the average data from the mile event (Noakes and Lambert, in review), as the mile event represents a 'transitional duration' event.

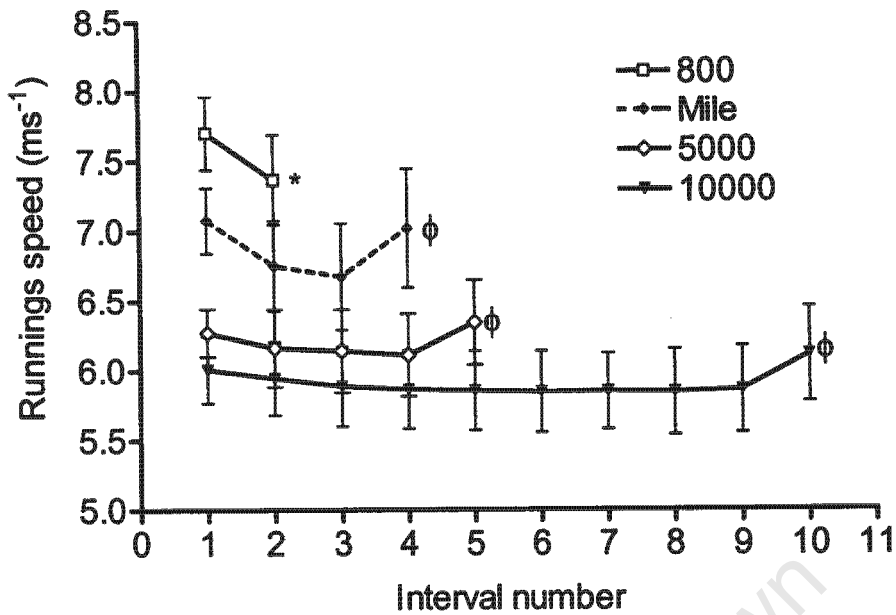


Figure 31: Average running speed for each interval during world record performances in 800 m, 1-mile, 5 000 m and 10 000 m events. The running speeds for the mile event are shown with a dashed line (from Noakes & Lambert, in review).

Values are means \pm S.D. for 26 (800 m), 32 (5000 m), 34 (10 000 m) and 32 (1 mile) performances. * Significantly slower than the first lap ($P < 0.005$); Φ Significantly faster than preceding intervals

Figure 32 shows the first and second lap times during world record performances in the 800 m event. The fastest second lap time ever recorded during a world record was 51.6 seconds in 1972 (Figure 32 right panel), following a first lap of 52.9 seconds.

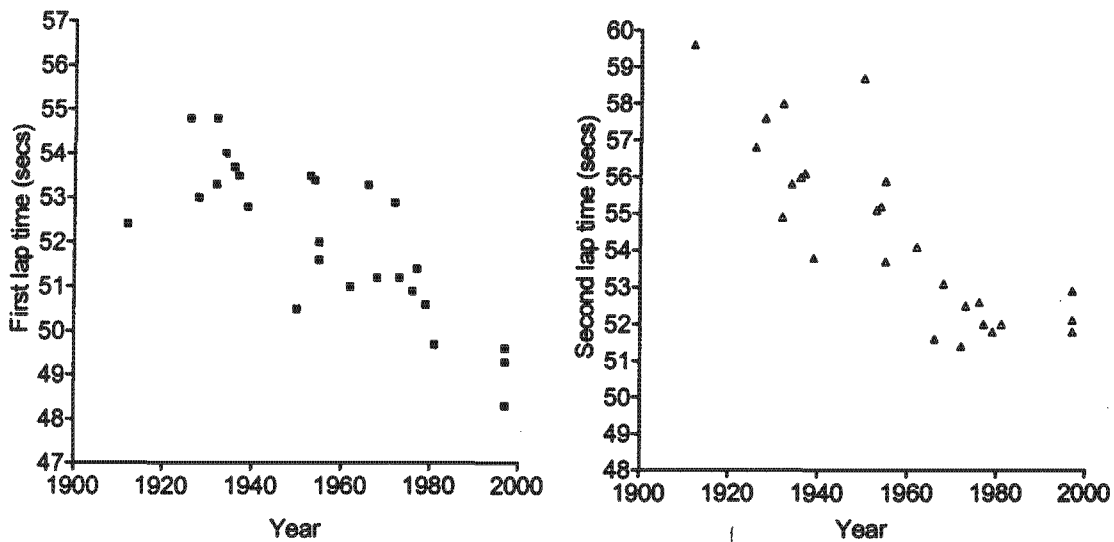


Figure 32: Times recorded during the first lap (32A) and second lap (32B) of 32 world record performances in the 800 m event since 1912.

Discussion

A key factor determining the optimal pacing strategy is the duration of the exercise bout (Foster *et al.* 1993; Ingen Schenau *et al.* 1994; de Koning *et al.* 1999; Foster *et al.* 2004). Thus, a maximal start followed by a progressive, but not drastic, slowing down has been proposed to be optimal for shorter events (Ingen Schenau *et al.* 1992; Ingen Schenau *et al.* 1994; de Koning *et al.* 1999), while an even pace is suggested to be more beneficial during exercise of longer duration (Foster *et al.* 1993; Ingen Schenau *et al.* 1994). Accordingly, the first significant finding of this study was that during the 800 m event, world records are mostly achieved with a second lap which is significantly slower than the first lap (Fig. 27 and 28). Indeed, in only two world records has the second lap been run at a faster pace than the first lap. This supports the notion that performance in

shorter events (< 110s) is improved by a faster start, even if the running pace declines towards the end of the race.

It is of interest that the two fastest second lap times ever achieved in 800 m world record performances were run in 1972 and 1966 respectively (Fig. 32 right panel).

The second lap time of a world record performance has therefore not improved in 35 years, since Dave Wottle broke the world record with a time of 1:44.3 (min:s) and a second lap of 51.40 seconds in 1972. The current world record holder, Wilson Kipketer, has broken the world record on three occasions, with second lap times of 52.12, 52.90 and 51.80 seconds. Therefore, a 3.2 second reduction in the world record in the 800 m event between 1966 and 1997, from 1:44.3 to 1:41.11, has been achieved by running the first lap significantly faster, rather than an improved ability to increase running speed on the second lap (Fig. 32 left panel). Collectively, these findings suggest that the ability to run faster during the second lap of an 800 m is limited, and so the optimal pacing strategy may consist of a faster start followed by a relatively slower second lap.

This finding agrees with previous research of pacing strategies adopted during competitive sprinting competitions (Ferro *et al.* 2001). This research has shown that during 100 m, 200 m and 400 m races, athletes reached peak running speeds before reaching halfway, and then slowed down progressively until the finish (Ferro *et al.* 2001). This was the case for every single athlete in both male and female sprint events at the IAAF World Championships, suggesting that an

underlying physiological process is responsible for the reduction in running speed, and that optimal performance in these relatively short duration events requires the faster start, even at the cost of a progressive reduction in running speed towards the end of the race.

Furthermore, in that study (Ferro *et al.* 2001), the athletes who won were not necessarily the ones who began races conservatively and then slowed down the least during the final part of the race. Rather, those athletes who won the races adopted similar pacing strategies as the athletes who finished last, but were faster throughout the race. Similarly, in the 800 m event in the present study, the strategy has not changed since the first documented record in 1912, and improvements in the time in the past 30 years have been achieved by increasing the speed on the first lap, and maintaining the same overall pacing strategy. If running an even paced race with a more conservative first lap was the optimal strategy for the 800 m event, then the improvements in the world record would be expected to occur with a faster second lap, but this is clearly not the case, as seen by Fig. 32.

Therefore, while it is acknowledged that world record performances are not necessarily achieved with 'optimal' pacing strategies, this observation does suggest that optimal performance in shorter duration exercise, such as the 800 m event in the present study, requires the attainment of peak speeds early during the bout, with a progressive reduction in the second half of exercise. Similar findings exist

in laboratory studies of performance and pacing strategy. For example, Foster et al.(2003) showed that during self-paced 1500 m cycling time-trials (130 – 135s), power output and velocity peaked within the first 100 and 300 m respectively, and then decreased progressively, with the lowest power outputs being recorded in the final 400 m, a pattern similar to that of the 800 m (Fig. 27). In a subsequent study, Foster et al (2004) showed that even when power output decreased during cycling time-trials ranging in length from 500 to 3000 m, athletes maintained an ability to increase anaerobic energy production for a terminal acceleration. It was suggested that athletes monitor and then regulate their energetic output over time in order to optimize performance.

Finally, Fukuba and Whipp (1999) have demonstrated an inability to make up lost time during the second half of middle and long distance running events. They showed that if the initial running velocity was slower than the running speed at a predefined fatigue threshold (Fukuba & Whipp 1999), the athlete would be unable to make up for the lost time with a final spurt. The fatigue threshold running speed was conjectured to represent the running speed for each event at which a steady state could be maintained for pulmonary gas exchange, blood acid-base status and blood lactate concentration (Fukuba & Whipp 1999). In the 800 m event, however, it appears that the running speed required on the first lap is even greater than a 'steady state threshold speed', because athletes actually slow down on the second lap. Therefore, the present results support the notion that athletes cannot recover lost time from a slower start (Fukuba & Whipp 1999), but also

appear to indicate that in the 800 m, the first lap must be run at a speed that forces the athlete to slow down on the second lap, without any ability to accelerate.

In contrast, world records in the 5 000 m and 10 000 m events are characterized by fast starts, a period of slower running during the middle of the race, followed by a significant increase in speed towards the end (Fig. 30 and 31). This is similar to the pacing strategies adopted by elite rowers during 2000 m rowing races and time-trials (Garland, 2005). That study found that the first 500 m of a 2000 m time-trial was completed at an average speed of 103.3% of the average speed for the whole race, with a progressive decrease in speed for the second and third 500 m sectors, before the speed increased slightly in the final 500 m. These 2000 m rowing events typically last between six and eight minutes, and therefore lie midway between the 800 m and the 5000 m and 10 000 m events in the present study. This may account for the relatively slower final sector in the rowing trials, since in our study, the fastest kilometer sector occurred at the end of exercise rather than at the beginning.

Laboratory studies have also found that performance in events longer than 120 s is optimized by a more even pacing strategy. For example, optimal performances during a 2000 m cycling time-trial (150 s) were achieved with an imposed even pacing strategy, which was in fact not different from the self-selected pacing strategy. Overall performance was however impaired by 7.2 seconds (4.3%) when

the imposed speed for the first kilometer was significantly slower. When the first kilometer was completed significantly faster, overall performance was impaired by 4.9 seconds (2.9%) (Foster *et al.* 1993).

The pacing strategy adopted in the 5000 m and 10 000 m world records has been remarkably consistent. For example, in the 5000 m event, the final kilometer of the race has been the fastest of the race in 21 out of 32 world records, while it has been the second fastest kilometer of the race (behind the opening kilometer) in the other 11 world record performances. Therefore, the middle part of the race from 2 to 4 km have never been faster than either the first or final kilometer in any world record performance at 5 000 m. Similarly, in the 10 000 m, the first or final kilometers have been the fastest of the race in 33 out of 34 world records. Only during the world record performance of Paul Tergat in 1997 (26:27.85) was any kilometer other than the first or final kilometer the fastest, when the ninth kilometer was run one second faster than the final kilometer. In 25 out of the 34 world record performances at 10 000 m, the final kilometer has been the fastest of the race.

The characteristic pacing strategy during these longer duration events has physiological significance. In particular, the presence of an endspurt at the end of the 5000 and 10000 m races is important, since it suggests that a reserve capacity is maintained during the exercise bout. The present thesis has provided evidence to suggest that the exercise workrate and levels of skeletal muscle recruitment are

regulated in advance of potentially limiting or harmful physiological changes, and the generation of a characteristic pacing strategy is a consequence of this regulation (Chapters 2 to 5). The characteristic endspurt would be the result of the athlete overriding this regulation at the very end of exercise when utilizing this reserve. Clearly, the athlete cannot run at a faster running speed early on, for this would result in premature fatigue, but the mechanism by which a sub-maximal running speed is 'selected' during the race and how the optimal pace is regulated during such longer duration exercise is not yet understood. It cannot be explained by the direct effects of any physiological variable such as metabolite accumulation (Nummela *et al.* 1992) or high body temperatures (Nybo & Nielsen 2001a) on the ability of the muscle to produce force, for if these variables determined the pacing strategy during the earlier part of the race, they would also prevent any increases in running speed at the end of the trial.

Previous experience and training are important factors that must influence the athlete's decision to either slow down or speed up during an event. Tactics are also important, and in the present study, changes in running speed based on tactical situations cannot be accounted for. Unfortunately, data regarding race tactics do not exist for all world record performances. However, the majority of world records are set in planned record attempts, with race tactics playing a relatively minor role. Also, in only one of 66 world records in the 5000 m and 10 000 m events has the fastest kilometer split occurred outside of the first or final kilometer. If race-tactics affected world record performances, it may be expected

that there would be occasions where the middle kilometers would be fastest, yet this is not the case. It may also be argued that tactically-motivated adjustments in workrate would tend to manifest towards the end of the race, as athletes alter their pace in response to the tactics of other athletes, and so the reductions in running speed which occur early on are less likely to be influenced by tactics than increases in speed later on during races.

The reasons for the difference in pacing strategy between the 800 m event and the longer 5 000 and 10 000 m events are not clear. The mile event, which lasts approximately four minutes, is characterized by a fast first lap, two significantly slower laps, and a fast final lap, for an overall even pacing strategy (Fig. 31). It is thus similar to the 5000 m and 10 000 m events, though in the longer events, changes in running speed do not appear to be as great as in the mile event (Fig. 31). This may suggest that the transition from an optimal negative pacing strategy and an even pacing strategy occurs in events that are shorter than the mile (that is, less than four minutes). This is supported by laboratory based studies, which have shown that 2000 m cycling events, lasting approximately three minutes, are optimized by an even pacing strategy (Foster *et al.* 1993), whereas the performance in the 1500 m event, lasting approximately 3 minutes, is optimized with a faster start (Foster *et al.* 1994).

Shorter, higher intensity exercise is traditionally considered to be anaerobic, resulting in metabolite accumulation (Jacobs & Kaiser 1982; Jacobs *et al.* 1983) and

depletion (Karlsson & Saltin 1970; McLester, Jr., 1997), which are presumed to impair muscle contractility, resulting in a progressive decline in force output (McLester, Jr., 1997). For example, Nummela et al. (1992) found that drop jump performance was impaired by 39% following a maximal 400 m sprint, and the reduction was correlated with increases in blood lactate concentration. The EMG activity in the active sprinting muscles increased significantly over the course of the run, and it was concluded that additional motor units were being activated to compensate for the progressive reduction in muscle force production as a result of metabolic acidosis in the muscle (Nummela *et al.* 1992). It was concluded that fatigue in the 400 m sprint was mainly due to processes within skeletal muscle rather than in the central nervous system (Nummela *et al.* 1992).

However, studies of the pattern of energy system contributions to power output during high intensity cycling lasting less than two minutes suggest a more complex form of regulation. It has been shown that energetic resources are distributed over the duration of the event in order to preserve the contribution of nonoxidative energy production to power output until the end of the exercise bout (Foster *et al.* 2003; Foster *et al.* 2004). It was suggested (Foster *et al.* 2003) that the intracellular changes occurring during exercise, such as metabolite accumulation (Diamant *et al.* 1968; Karlsson & Saltin 1970; Jacobs & Kaiser 1982; Jacobs *et al.* 1983) or phosphagen depletion (McLester, Jr., 1997), were being monitored continually and that power output was reduced in advance of these changes becoming critical or harmful, a notion which supports the existence of

pacing, even in the presence of a falling power output. This regulation of energetic resources would allow athletes to expend energy anaerobically for a terminal acceleration (Foster *et al.* 2004), and suggests that pacing strategy is not 'all-out', but regulated, even in the presence of a falling power output or running speed, as may occur in the 800 m event in the present study.

Also, in the study of Nummela *et al.* (1992), running speed during a maximal 400 m time-trial decreased progressively even though motor unit activation (measured as EMG activity) was able to increase to compensate for the apparent failure of muscle contractility. Early reductions in running speed therefore occurred despite a capacity to increase motor unit activation, which indicates that a neural control strategy exists even during maximal sprint exercise.

Such a complex neural strategy, which ensures the maintenance of a motor unit reserve, would also explain the finding that when short duration exercise is undertaken, the initial power output is lower than would be possible if the athlete is instructed to perform an all-out effort with no regard for overall performance (Foster *et al.* 1994). This is observable in the pacing strategies of 400 m sprinters (Ferro *et al.* 2001), who run the first 50 m of the race at speeds slower than they can achieve for the first 50 m during a 200 m race. Running speed still however decreases progressively in the 400 m event (Ferro *et al.* 2001). Therefore, pacing strategies are evident even in the presence of a reduction in running speed.

These findings implicate an anticipatory pacing strategy during even supramaximal exercise, a finding which is supported by Ansley *et al.* (2004), who found evidence for a pre-programmed, centrally regulated pacing strategy during supramaximal exercise lasting only 36 seconds. These observations challenge the notion that fatigue during short duration exercise is “mainly due to processes within skeletal muscle rather than the central nervous system” (Nummela *et al.* 1992). Instead, the progressive reduction in running speed or power output in these studies (Nummela *et al.* 1992) and in the 800 m event in the present study must be due to the integration of changes occurring in the muscle into a complex regulation by the central nervous system.

Thus, even though a possible contribution of metabolic derangements to fatigue cannot be discounted, reductions in power output, and presumably, running speed during the 800 m event would occur as part of a centrally-regulated control mechanism based on afferent feedback (Noakes *et al.* 2001; St Clair Gibson *et al.* 2001a; Lambert *et al.* 2005; Noakes *et al.* 2005) to protect against harmful disturbances to homeostasis while still optimizing performance. The balance between preventing catastrophic disturbances to homeostasis and optimizing performance may allow greater running speeds early on during shorter duration exercise, even though this may cause a certain degree of metabolite accumulation and depletion to occur, and may increase the perception of effort according to the proposed model. However, since the 800 m event is short, both the metabolic changes and the elevated RPE may be tolerable over the anticipated time of

exercise, with the result that higher running speeds occur early, before a progressive reduction towards the end.

In longer duration exercise, the pacing strategy is regulated to ensure a reserve is present at the end, with the result that running speed can be increased consequent to an increase in skeletal muscle recruitment. Support for this theory was provided by Chapter 2 and Chapter 3 of the present thesis, as well as in the study of Albertus et al. (2005), which measured an increase in the EMG activity during final kilometer of 20 km time-trials.

The precise reasons explaining why the second lap in 800 m world records has not improved in 30 years are not clear. According to the current discussion, the apparent inability to increase the running speed in the second lap may be due to a combination of metabolic changes in the muscle and a centrally-regulated pacing strategy, which monitors the use of energetic resources and the degree of metabolic derangement during high intensity exercise. It may thus be that advances in training and technology of shoes and track surfaces have improved the ability of athletes to run faster with similar levels of metabolic derangements. As a result, the brain 'allows' faster running speeds before a perceived level of metabolite accumulation or depletion is reached, causing a reduction in speed over the second lap.

Further, the increased contribution of runners of African descent to world record performances since the mid-1960's cannot be discounted, because it has been shown that these runners have improved running economy compared to Caucasian runners. Therefore, it is possible that physiological differences, including improved running economy, which manifest as reduced rates of metabolite accumulation at a given running intensity, are responsible for different pacing strategies in recent years.

In conclusion, the optimal pacing strategy during world record performances differs for the 800 m event compared to the 5 000 and 10 000 m events. In the 800 m, greater running speeds are achieved in the first lap, and it appears that time that may be lost on the first lap cannot be recovered by a faster second lap, since there appears to be little capacity to run the second lap faster than the first. In longer duration events, pacing strategy is regulated to ensure that a reserve is maintained, which allows significant increases in running speed at the end of the event. It is proposed that these findings support the concept that the pacing strategy is regulated in an anticipatory manner by a central governor which ensures that physiological reserves are maintained. During both longer and shorter duration maximal effort events, certain levels of non-catastrophic derangements may be tolerated in order to optimize kinetic energy and performance. This suggests that exercise may be regulated by a complex, intelligent system (St Clair Gibson & Noakes 2004).

Chapter 7

Summary

***Conclusion including a proposed
anticipatory-feedback model for the
regulation of exercise incorporating the
rating of perceived exertion***

The present thesis has provided evidence for the existence of a centrally-mediated system that regulates exercise performance and pacing strategies by means of adjustments in skeletal muscle motor unit recruitment, protecting against the development of harmful or limiting changes in physiological systems. The findings of each study are summarised briefly:

In Chapter 2, it was shown that 20 km cycling time-trial performance is impaired in hot compared to cool conditions despite rectal temperatures, heart rates and ratings of perceived exertion that were not different from the cool condition. The ability to maintain a higher power output in the cool was associated with an increase in the IEMG activity of the active muscle. Further, the IEMG activity and power output increased in the final kilometer of the trials, producing a characteristic endspurt which suggests the regulation of motor unit recruitment during the preceding kilometers. I proposed the existence of an anticipatory system that regulates the degree of motor unit recruitment specifically to ensure that a limiting temperature would not be attained before the anticipated end of the exercise bout.

In Chapter 3, this model was applied to exercise in hyperoxia. It was found that the power output maintained during a 20 km time-trial in hyperoxia (F_{iO_2} 40%) was greater than in normoxia (F_{iO_2} 21%), and this elevation in power output was again associated with a greater level of skeletal muscle activation. It was therefore

proposed that the brain allowed a greater level of muscle activation in hyperoxia, perhaps as a result of reductions in metabolite accumulation.

In both Chapter 2 and Chapter 3, it was found that the Rating of Perceived Exertion (RPE) was not different at any stage during time-trials, despite differences in power output between conditions. A possible role for the RPE as a mediator of the proposed anticipatory regulatory system was suggested. In Chapter 4, this proposed role was studied using a novel RPE clamp protocol, in which cyclists exercised in a range of environmental temperatures at a pre-determined and fixed RPE. This study provided evidence that the RPE was sensitive to the rate of heat storage, and it was proposed that the RPE played a mediatory role in an anticipatory-feedback model for exercise, being influenced by the rate of heat storage during the early part of the trial, and then mediating reductions in workrate to maintain the required RPE.

In Chapter 5, the possibility that the regulation of skeletal muscle motor unit recruitment occurs even during maximal exercise to volitional exhaustion was examined. It was found that during an incremental exercise trial to exhaustion, the IEMG activity at fatigue was only half that achieved during maximal 20-second sprints when the power output was doubled compared to the peak power output at fatigue. It was concluded that even at maximal effort, muscle activation was submaximal, supporting the theory that all forms of exercise are regulated by a complex system, rather than being the result of a catastrophic failure in one or

more physiological system, and which occurs only after all motor units are recruited. This study validated the proposed hypothesis that skeletal muscle recruitment was most likely submaximal during all forms of self-paced exercise, an important component of the argument presented in Chapters 2 to 4. The maximal level of muscle activation also coincided with the attainment of a maximal RPE during both the sustained exercise and the intermittent sprint bouts, suggesting that the RPE does not merely track the exercise workrate, but takes into account the expected duration of exercise.

In Chapter 6, I examined the pacing strategies of elite men's world records at distances ranging from 800 m to 10 000 m. This study showed that during longer duration exercise (5000 m and 10 000 m), pacing strategies were characterized by the presence of an endsprint, a significant increase in running speed at the end of the race. This agrees with Chapters 2 to 4, and supports the interpretation that exercise workrate is maintained at submaximal levels during the middle part of the exercise. In contrast, during the 800 m race, there appears to be no ability to increase speed in the second half of the race. Therefore, the optimal pacing strategy depends on the length of exercise, and Chapter 6 proposed potential reasons for this observation.

Based on the findings of the present thesis, together with recent research that was discussed in more detail in Chapter 1 of the present thesis, the following models

are proposed for the regulation of exercise performance by a complex intelligent system.

Exercise at a fixed workrate to fatigue

Figure 33 depicts a model for the regulation of exercise performance at an imposed workrate to fatigue, including incremental exercise to exhaustion as used in Chapter 5. At the onset of exercise, afferent information from various physiological systems and external/environmental cues (A) is used by the brain to forecast the duration of exercise that can be safely completed without causing harmful homeostatic derangements (B). The afferent feedback from physiological systems depends on the exercise intensity and environmental conditions, including factors such as temperature and the partial pressure of oxygen of the inspired air. Simultaneously, the initial rate of increase in RPE is set as a consequence of a subconscious anticipatory calculation of the safe exercise duration (C). The initial 'setting' of exercise duration and the rate of increase in RPE represent the anticipatory component of the model.

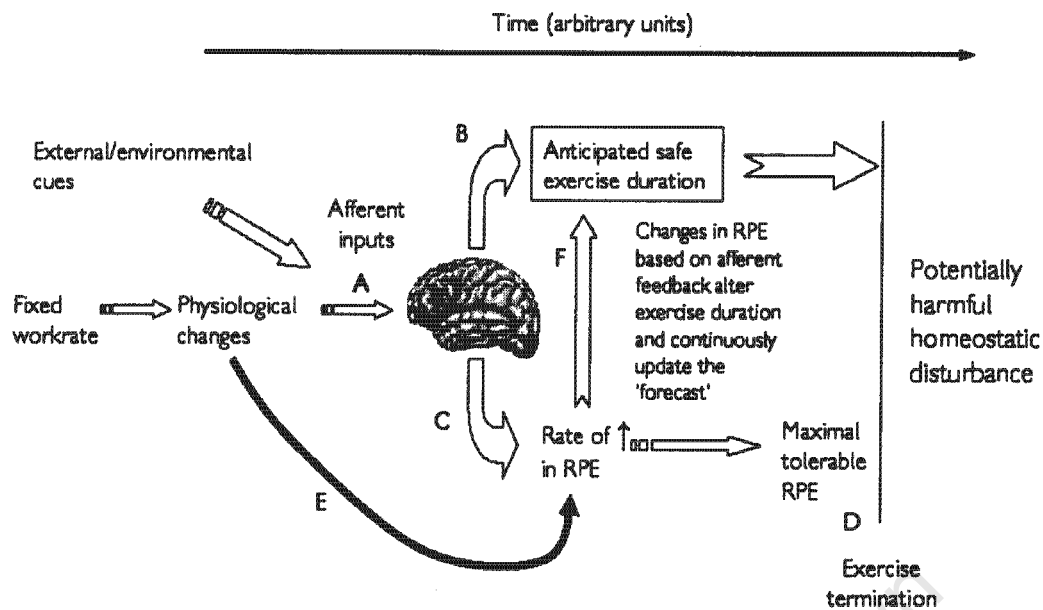


Figure 33. Schematic diagram showing the proposed model for the anticipatory regulation of exercise performance during exercise to fatigue at a fixed workrate.

Since exercise terminates when the maximal tolerable RPE is attained (D), the time to exhaustion is determined by the rate of increase in RPE, which is continuously modified based on the regular integration of afferent feedback signals from numerous physiological systems, include those described in Chapter 1 (E). The 'safe' exercise duration is thus determined by a combination of anticipatory forecasting and afferent feedback. The maximal tolerable RPE (D) occurs before harmful changes to homeostasis can occur. Such changes include the attainment of a critically high core temperature, as described in Chapters 2 and 4. The high core temperature thus acts as an 'off-switch', mediated, importantly, by the RPE.

The novel aspect of this model is that physiological changes do not directly limit the ability of the athlete to perform exercise (a catastrophic model (Noakes & St Clair Gibson 2004; Noakes *et al.* 2005)), nor are they the singular cause for the

generation of the RPE. Rather, afferent feedback, expected exercise duration and physiological changes during exercise are used by a central controller to forecast future physiological changes. Based on this feedback, an RPE is generated and then increased at a rate that ultimately determines the safe exercise duration. This model can be applied to the studies described in Chapter 1 of the present thesis, which showed the influence of high temperatures (Nielsen *et al.* 2001; Nybo & Nielsen 2001a; Nybo & Nielsen 2001b; Rasmussen *et al.* 2004) and low muscle glycogen concentrations (Baldwin *et al.* 2003; Noakes, 2004) on exercise performance to fatigue at a fixed workrate.

Self-paced exercise time-trials

The previous model (Figure 33) is only applicable to exercise at a fixed workrate, in which the central control processes cannot alter the pacing strategy in response to experimental perturbations, but can only influence the duration of exercise. If the exercise workrate is free to vary, then experimental interventions such as changes in ambient temperature, oxygen content of the inspired air, energy substrate availability or the provision of incorrect distance feedback all alter the power output (pacing strategy), whereas the rate of increase in RPE has been found to be similar between conditions (Chapter 2 and 3) (Peltonen *et al.* 1997; Tatterson *et al.* 2000; Marino *et al.* 2004; Tucker *et al.* 2004; Albertus *et al.* 2005; Havemann *et al.* 2006).

In Figure 33, the RPE is set by the brain at the onset of exercise, and the time to fatigue is determined by the rate at which the RPE rises to reach a maximal tolerable value. In contrast, self-paced exercise with a known duration would allow the workrate to be altered in response to a high or subjectively unsustainable RPE. It seems reasonable to suggest that rather than simply maintaining a high self-selected workrate until the RPE rises to reach near-maximal levels, the exercising athlete would instead reduce the workrate in certain conditions to prevent hyperthermia or substrate depletion from occurring before the end of exercise. This would have the effect of regulating the RPE, so that it would not differ from control trials in which the exercise workrate was higher, as occurred in Chapter 2 and 3 of the present thesis.

Accordingly, the fixed workrate model in Figure 33 is extended to propose that during self-paced exercise, the brain integrates afferent information from various physiological systems (described previously) to generate a conscious RPE, and then regulates the workrate to ensure that this conscious RPE does not increase excessively at any stage during exercise, since this would lead to premature exercise termination (Noakes, 2004). Importantly, in order to prevent the conscious RPE from exceeding acceptable levels, there must be some expected or acceptable RPE during exercise, against which the conscious RPE can be continuously compared. This is termed the 'template RPE' and, it is proposed that it is generated as a result of previous experience and knowledge of the upcoming exercise duration (St Clair Gibson *et al.* 2006). It is important to note that this

'template RPE' is a construct which cannot be measured, but, as proposed below, is necessary in order for the conscious RPE to be interpreted. In contrast, the conscious RPE is a verbalized rating, usually on the Borg scale (Borg, 1982), which is measured during exercise.

According to this model, shown in Figure 34, the regulation of exercise workrate during self-paced exercise is achieved by means of a combination of feedback integration (which generates the conscious RPE) and anticipatory forecasting (which produces a 'template' for the RPE against which the conscious RPE is compared).

The anticipatory component requires previous experience and training, as well as afferent input from the environment, which may include physiological inputs such as reduced muscle glycogen levels or increased skin temperature, as well as psychological inputs such as motivation levels, arousal and the presence of competitors (A in Figure 34). In addition, at the onset of self-paced exercise, the exercising athlete is usually aware of the distance or duration of the upcoming exercise bout. Based on these factors, the exercising athlete self-selects an initial exercise intensity, which is anticipated to be optimal for the expected duration of exercise (B).

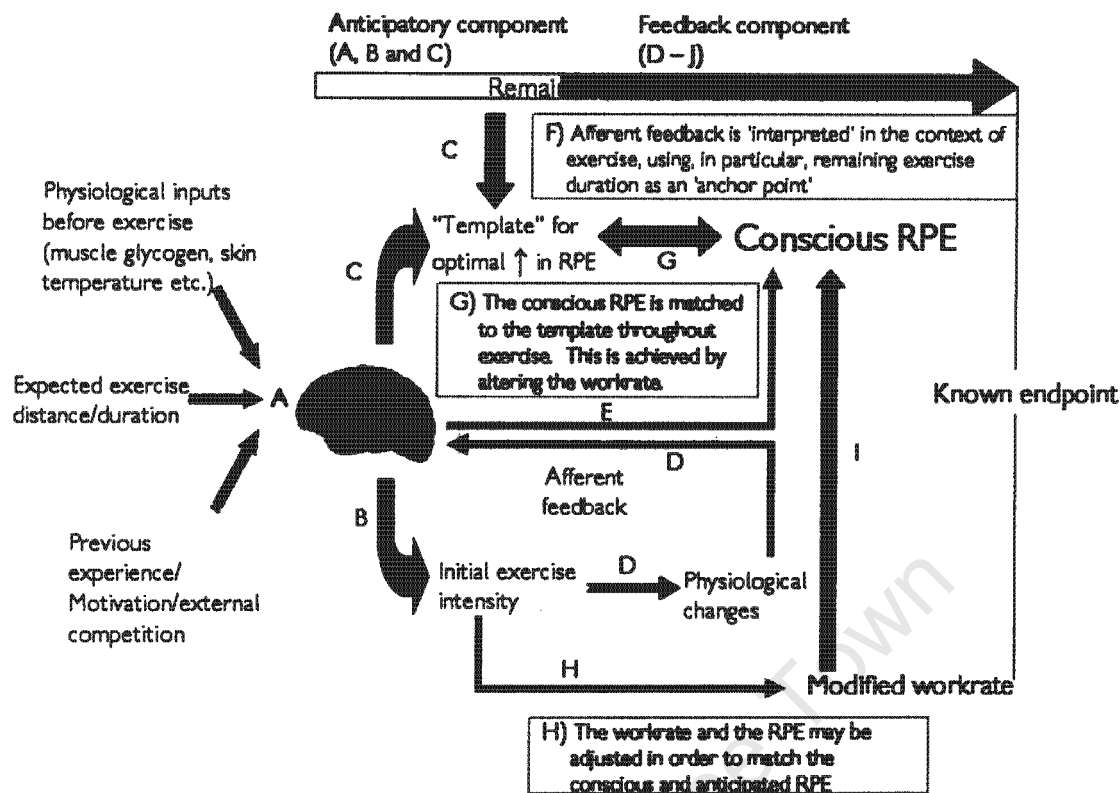


Figure 34. Schematic diagram showing the proposed model for the anticipatory regulation of exercise performance during self-paced exercise. Black shading denotes input to the brain, grey denotes output or efferent processes.

At the same time, the brain creates a 'template' for the ideal or optimal rate of increase in the RPE during the exercise bout (C). That is, optimal performance requires an increase in the RPE which ensures that a maximal tolerable RPE is reached at the moment exercise is completed, but not before. If the maximal RPE occurred before the endpoint, performance would be impaired by a reduction in the workrate or premature fatigue (Noakes, 2004). The 'template RPE' provides a means by which the conscious RPE can be interpreted – it is a construct based on memory or previous experience, which is updated continuously throughout exercise based on the remaining exercise duration (C).

As soon as exercise commences, changes in physiological variables, including body temperature, skin temperature, rates of heat storage, metabolite concentrations, arterial saturation levels, increased ventilatory rates and increased heart rates, and mechanical afferents from muscle generate afferent signals to the brain (D). These afferent signals are 'interpreted' by the brain in the context of the exercise bout, and result in the generation of a conscious RPE (E). The duration of exercise remaining is one of the key 'anchors' against which the afferent feedback is interpreted (F). The conscious RPE is thus a consequence of both the duration of exercise that remains, as well as the exercise intensity, since the degree of afferent feedback is a function of the exercise workrate (D and F).

The key aspect of the present model is that the conscious RPE is then 'matched' with the template RPE (or previous experience) throughout exercise (G, Figure 34). This would allow alterations in workrate to occur specifically to ensure that the conscious RPE does not differ from what the central controller considers 'acceptable' at any stage during the exercise bout, effectively incorporating previous experience, motivation and physiological afferent information into the regulatory process (G).

Adjustments to the workrate (H) would be achieved by alterations in the degree of skeletal muscle recruitment, as is supported by studies which measure EMG activity, a measure of skeletal muscle activation (Chapter 2 and 3)(Kayser *et al.* 1994; Peltonen *et al.* 1997; Kayser, 2003). Ultimately, this regulatory system will

prevent harmful or limiting physiological changes from occurring before the end of exercise, while still optimizing performance. The conscious RPE is an ideal candidate to perform a regulatory role, since it is a consequence of the total physiological changes occurring during exercise (St Clair Gibson *et al.* 2003), is specific to the conditions under which exercise is being performed (Hampson *et al.* 2001), and is potentially the only link between the physiological changes occurring during exercise and the athlete's conscious awareness of these changes.

If, for example, the initial exercise workrate is excessively high, it will produce physiological changes which result in the conscious RPE being greater than that which the central controller considers optimal for a particular stage during an exercise trial. The anticipatory-feedback control system would result in a reduction in motor unit recruitment and workrate (H) until the conscious RPE returns to an 'acceptable' level, as shown by I on Figure 34. As a result of continuous dynamic interaction between the subconscious anticipatory component and the afferent feedback component (G), power output is continuously modified in order to regulate the increase in the RPE. Because the RPE is influenced by physiological changes (Hampson *et al.* 2001), the matching of RPE to an anticipatory calculation successfully prevents catastrophic derangements to homeostasis. Thus, only power output is ever observed to change during self-paced exercise, while RPE is similar in various experimental interventions (Chapter 2 and Chapter 3). The pacing strategy is therefore a measure of how this

regulatory control process allocates physiological resources and prevents the sensation of fatigue from rising excessively during exercise.

Testing the model using the RPE clamp protocol

This model (Figure 34) can be evaluated using the novel RPE clamp method, described in Chapter 4 of the present thesis. Clamping the RPE at a constant predetermined level effectively replaces the generation of a template for an 'optimal' rise in RPE with an externally imposed 'template' (C on Figure 34). Thus, the athlete must regulate their workrate and conscious RPE so that it remains at the predetermined level (G on Figure 34). This allows two potential predictions of our model to be evaluated. Firstly, it allows the anticipatory or forecasting component of our model to be examined. If the initial workrate and RPE are set in a feedforward or anticipatory manner as is suggested (B and C on Figure 34), then the starting power output should be similar whenever exercise is performed at a given RPE, irrespective of experimental interventions, such as increased ambient temperatures, hypoxia, and dietary interventions. In contrast, if the initial workrate and RPE are the result of afferent feedback, then differences in workrate would be expected at the onset of exercise, as soon as differences between conditions are detected.

Secondly, the RPE clamp method allows the effects of afferent feedback on the regulation of exercise workrate to be studied. Our model proposes that afferent

feedback is 'interpreted' by the brain (D, Figure 34) and also directly influences the conscious RPE, resulting in continuous adjustments to the power output in order to return the conscious RPE to an anticipated optimal level. If the RPE is 'clamped', then power output would be reduced to prevent the conscious RPE from increasing above this predetermined level. Therefore, the hypothesis is that any intervention which results in differences in one or more physiological systems will alter the nature of the afferent feedback to the central controller and will thus result in greater changes in power output than during a control trial.

The findings of Chapter 4 validate these hypotheses. Firstly, it was found that the power output at the immediate onset of exercise was similar between the three conditions, suggesting that the initial selection of workrate is set in a feedforward manner (Figure 34), based perhaps on previous experience and expectations of exercise, rather than an instantaneous afferent input from an elevated skin temperature in the heat.

Further, the decrease in power output over the first ten minutes of the trial was also similar between conditions, again supporting the notion that the initial workrate is set in an anticipatory manner. However, between ten and thirty minutes, power output decreased more rapidly in the hot (35°C) condition, resulting in a reduced trial duration compared to the other two conditions. Over the course of the trial, power output declined linearly in all conditions, though at different rates, which suggests that the RPE is not simply a marker of exercise

intensity, since power output was falling at different rates in the different environmental conditions even though the RPE was fixed and identical between conditions, in agreement with our proposed model.

Secondly, the rate of heat storage was greatest in the hot condition only during the first ten minutes of the trial. Thereafter, heat storage was similar between all three trials, despite a 20°C difference in ambient temperature between conditions. A similar rate of heat storage despite a decreased potential heat loss in the hot condition would be the result of a reduction in the rate of heat production, which can be achieved by reducing the workrate. As described, this was the case in this study (Chapter 4), since the power output from ten to thirty minutes fell more rapidly in the hot than in the temperate and cool conditions (Figure 19B in Chapter 4). This finding therefore supports the feedback component of the proposed model (Figure 34), which posits that afferent feedback causes an increase in the conscious RPE, which, in this exercise study, forces a reduction in workrate in order to maintain the RPE at the predetermined level.

Finally, it is notable that the rapid reduction in power output in the hot condition occurred even though body temperatures were similar between the three conditions (Chapter 4). This suggests that anticipatory control is present even when afferent feedback has begun to adjust the workrate, as described. There is thus no distinct transition between anticipatory and feedback regulation, but rather a continuous integration of these two components. This has been

described in previous studies of self-paced exercise in the heat (Marino *et al.* 2004), including the results of Chapter 2 of the present thesis.

Further applications of the anticipatory-feedback model

The endspurt phenomenon

The proposed model is also able to account for the characteristically observed 'endspurt' phenomenon, where power output or running speed increase significantly during the final stages of self-paced trials (Marino *et al.* 2000; Tatterson *et al.* 2000; St Clair Gibson *et al.* 2001b; Marino *et al.* 2004; Albertus *et al.* 2005). Our model posits that changes in workrate during exercise are the result of the continuous interpretation of afferent feedback during exercise combined with an anticipatory calculation which identifies the optimal rate of increase in RPE and the initial exercise intensity. The afferent feedback is interpreted in the context of the exercise bout, and influences the RPE accordingly. In particular, the distance or duration of exercise that remains is a crucial 'anchor' against which the RPE is interpreted (F on Figure 34).

Throughout exercise, however, there is a degree of 'uncertainty', because it can never be precisely known when the exercise bout will be completed, or what physiological demands may be incurred after an exercise bout has been

completed. The interpretation of the afferent feedback and resultant regulation of workrate are therefore subject to a level of uncertainty which is greatest when exercise duration is great, and becomes progressively smaller as the known endpoint approaches. Since the role of the regulatory processes is to ensure that catastrophic derangements to homeostasis do not occur, this uncertainty results in the maintenance of a motor unit and metabolic reserve throughout exercise. As the exercising athlete approaches the known end-point, the uncertainty is reduced, and so the motor unit and metabolic reserve need not be maintained any longer, resulting in significant increases in motor unit recruitment and power output.

The effects of central nervous system drugs on performance

Our model also explains the performance-enhancing effect of dopamine/noradrenaline re-uptake inhibitors on exercise performance in hot conditions, as shown by Watson *et al.* (2005). These drugs are known to increase arousal and motivation and reduce the sensation of fatigue (Watson *et al.* 2005). This would act at two potential sites in the proposed model, as shown in Figure 35. Firstly, the initial anticipatory setting of workrate would be altered, since arousal and motivational levels would be elevated (A), resulting in an increase in the initial power output (B). Secondly, the conscious RPE at a given workrate would be reduced (C), resulting in a mismatch between the actual conscious RPE and the 'template RPE', which is based on previous experience (D). The consequence of

this mismatch would be an increase in the workrate (E), which will elevate the conscious RPE (F) until it returns to the anticipated levels for that exercise bout. At the same time, the elevated workrate will increase heat production, causing thermoregulatory failure or hyperthermia (G).

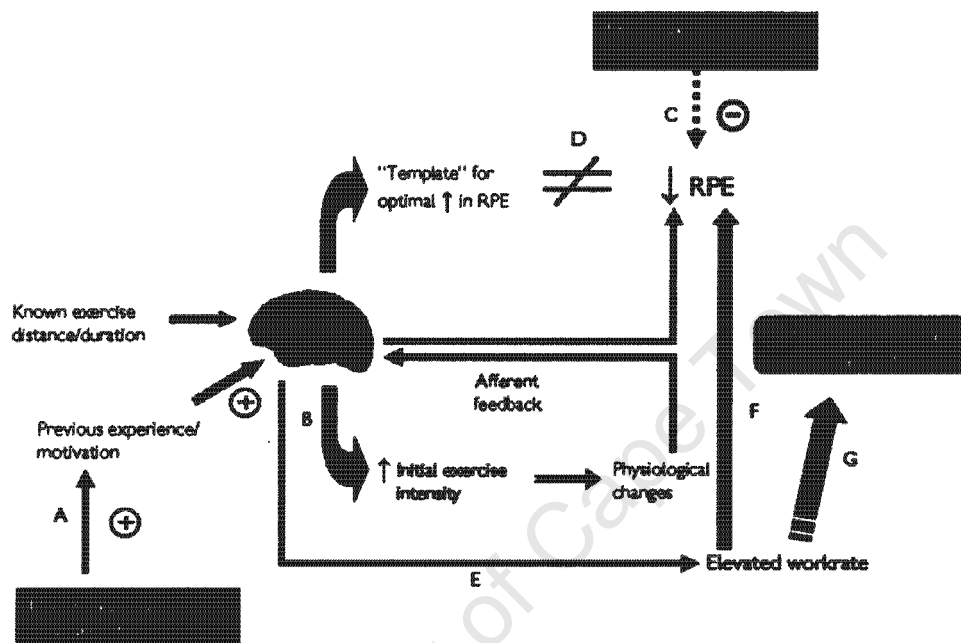


Figure 35. Schematic diagram depicting how the performance enhancing effects of dopamine/noradrenaline reuptake inhibitors can be explained according to the proposed model. Black shading denotes input to the brain, grey denotes output from the brain. (+) denotes stimulation, (-) denotes inhibition.

Since the purpose of this control system is to regulate exercise performance to prevent potentially harmful changes in homeostasis, a further prediction of the model would be that any improvements in performance which result from the use of these drugs will also cause a failure to regulate physiological variables compared to when exercise is performed without such drugs. Indeed, Watson et al. (2005) found a significant increase in power output during time-trials in hot conditions

(30°C) when the reuptake inhibitor was administered, and this was associated with a significantly elevated rectal temperature. Seven out of the nine subjects were in fact able to exceed what is usually considered the critical core temperature of 40°C without volitional fatigue, which represents an overriding of the usual control process occurring in the heat (Patterson *et al.* 1994). This observation can be accounted for by the model (G on Figure 35). A similar phenomenon may explain the effects of amphetamines on exercise performance (Noakes, 2004), and may account for the observed effect of hypnosis on performance.

Application to studies where misinformation is provided

Finally, the present model is also able to account for the findings of studies in which athletes are provided with incorrect or misleading feedback regarding exercise duration (Nikolopoulos *et al.* 2001; Ansley *et al.* 2004; Albertus *et al.* 2005; Baden *et al.* 2005). These studies, which were described earlier, are particularly interesting because they allow the anticipatory components of the model to be evaluated (A, B and C in Figure 34), since they alter the expected exercise distance or duration (A, Figure 34). As proposed, the anticipatory setting of the initial workrate and the generation of a template for the increase in RPE are based upon the expectation of exercise duration (B and C in Figure 34).

In addition, the continuous updating of this RPE 'template' and the interpretation of afferent feedback depends on knowledge of the exercise duration that remains

(St Clair Gibson *et al.* 2006) (C and F, Figure 34), and so incorrect expectation of distance remaining prevents the appropriate interpretation of physiological afferents. Therefore, an intervention which deceives the subject with regards to exercise duration creates numerous errors in the anticipatory calculations and the interpretation of afferent feedback, which would alter the model as proposed in Figures 36 and 37, with the errors in the anticipatory calculations denoted by crosses.

University of Cape Town

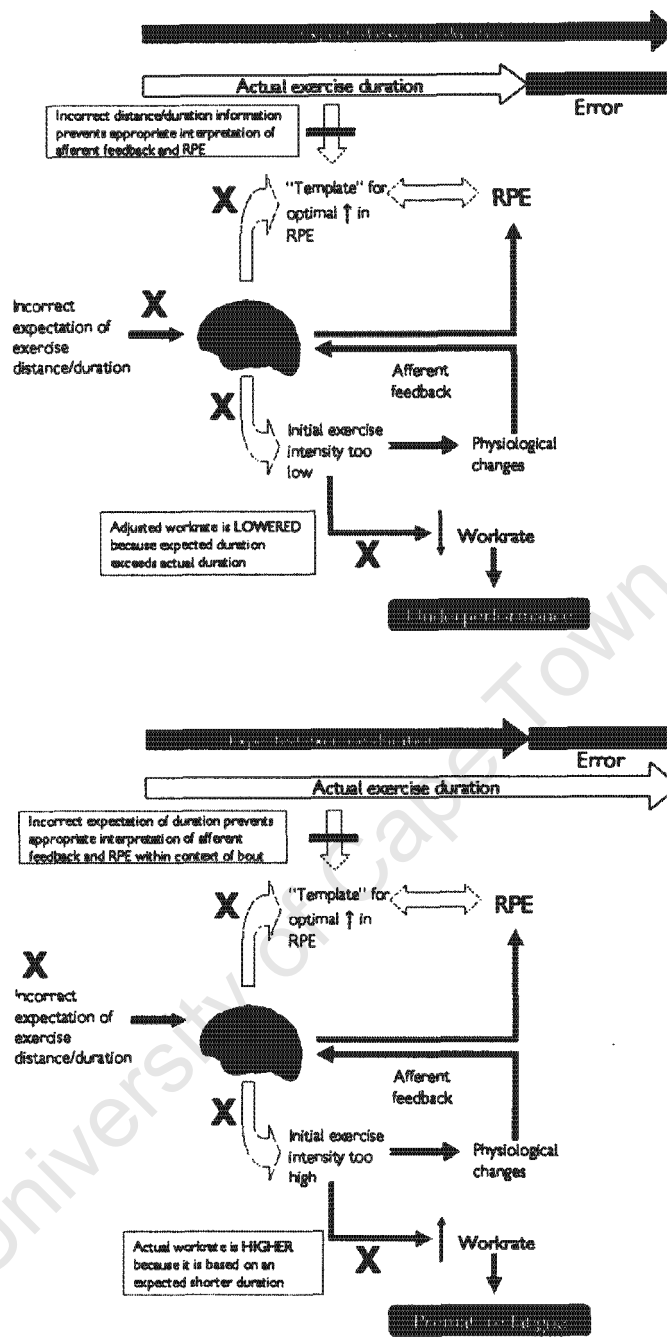


Figure 36. Schematic diagram showing how the anticipatory component of the proposed model would be influenced when athletes are incorrectly informed of exercise duration before the exercise bout commences. The top panel depicts the proposed model when the actual duration is shorter than the athlete is informed. The bottom panel depicts the proposed model when the actual duration exceeds the expected duration. Both

situations result in relative underperformances (see text for details). Black shading denotes input to the brain, grey denotes output from the brain. Dashed white arrows depict processes that have been removed/ablated by the provision of incorrect information.

Actual duration is shorter than the expected/informed duration

If the exercising athlete is incorrectly informed about the distance of exercise BEFORE they begin, the overall effect will be to alter the initial workrate and the 'template RPE', since both are based on the expectation of exercise duration, as shown in Figure 36. That is, the athlete's conscious RPE must be compared to some expected RPE, but if the duration has been incorrectly 'set', this comparison will be incorrect. If no further distance feedback is provided during exercise, then there are two possible outcomes: First, if the actual length of exercise is shorter than what the athlete was informed, an overall under-performance will occur (Figure 36 top panel). This occurs because the anticipatory setting of workrate, the optimal rate of increase in RPE and the interpretation of afferent feedback will have been based on the expectation of a longer exercise bout than is really the case. This will result in the relative under utilization of physiological resources, and an excessively large reserve will be maintained during exercise (Figure 36 top panel).

Actual duration exceeds the expected/informed duration

The second possible outcome arises if the actual duration exceeds that which the athlete had been told (Figure 36 bottom panel). In this case, the anticipatory setting of the initial workrate, the generation of the template for the RPE and the interpretation of feedback will have been made with the expectation of a shorter exercise bout. This will result in the maintenance of very high workrates for the first part of the exercise bout in order to match the conscious RPE with the 'template RPE', until the actual duration exceeds the expected duration.

Thereafter, excessive afferent feedback as a result of the high initial workrates will cause premature fatigue, a rapid reduction in power output and a relative underperformance compared to what could be achieved when duration is correctly known (Figure 36 bottom panel).

In support of this, Ansley et al. (2004) found that when cyclists performed 36 seconds of maximal cycling after being informed that they would be cycling for only 30 seconds, their power output in the final 6 seconds was significantly lower than when they were correctly informed of the exercise duration. According to the present model, this occurs because the subjects interpreted afferent information feedback incorrectly, based on the expected duration of 30 seconds, and so physiological resources were incorrectly allocated from the onset of exercise. Similarly, when subjects ran on a treadmill at a fixed velocity and were deceived about the length of exercise and instructed to run for longer than

anticipated, their RPE and affect scores increased suddenly (Baden *et al.* 2005). As described, this occurs when the exercise workrate is fixed. When it is free to vary, then the power output or running speed would be reduced.

Another study design which allows distance or duration manipulation is to correctly inform the athlete of the exercise duration before the trial, but to provide inaccurate distance feedback during the trial. Albertus *et al.* (2005) performed a study in which trained cyclists performed 20 km time-trials with either correct or incorrect distance feedback every kilometer. It was found that overall performance, pacing strategies and the subjective ratings of perceived exertion were not different at any stage between trials, despite the provision of inaccurate distance splits (Albertus *et al.* 2005). This suggests that the anticipatory setting of the initial workrate and the subconscious calculation of the optimal rate of increase in RPE, both of which are based on previous experience and knowledge of exercise duration (A, B and C, Figure 34), are particularly important (St Clair Gibson *et al.* 2006). It also suggests that the interpretation of afferent feedback in the context of distance remaining may have a less important role in regulating the pacing strategy under the conditions evaluated in that study, since subjects did not alter their power output in response to incorrect distance splits (Albertus *et al.* 2005).

It was speculated that the exercising athlete did not take the incorrect distance information into account, but based their pacing strategy on previous experience,

the initial knowledge of exercise duration and on physiological afferent inputs throughout the exercise bout (D in Figure 34). This strongly supports the model that the workrate during self-paced exercise is the result, at least in part, of anticipatory calculations. Since there was no experimental intervention other than the provision of incorrect distance information, the interpretation of the afferent feedback may have played less of a role. If trials are conducted in the heat, or with reduced oxygen content, for example, then continuous knowledge of the distance remaining may become more important as an anchor point (St Clair Gibson *et al.* 2006) in the regulation of workrate.

No duration information is provided

Finally, the model predicts that if the subject is not informed of the exercise duration before exercise begins, the initial workrate will be reduced compared to the normal situation, also resulting in an underperformance (Figure 37), similar to that which would be observed when the actual duration is shorter than what is expected (Figure 36 top panel, discussed previously). This occurs because of the high level of uncertainty when the endpoint is not accurately known. Ignorance of exercise duration negates the role of previous experience and training, and once the exercise bout has commenced, it prevents the correct 'interpretation' of afferent feedback. That is, the 'template RPE' cannot be set and nor can afferent feedback be interpreted in the absence of an anchor point provided by knowledge of the end-point of exercise. In the absence of this 'template', the conscious RPE

cannot be interpreted, and so the overall effect will be to reduce the workrate compared to a control trial in which the athlete is aware of the duration.

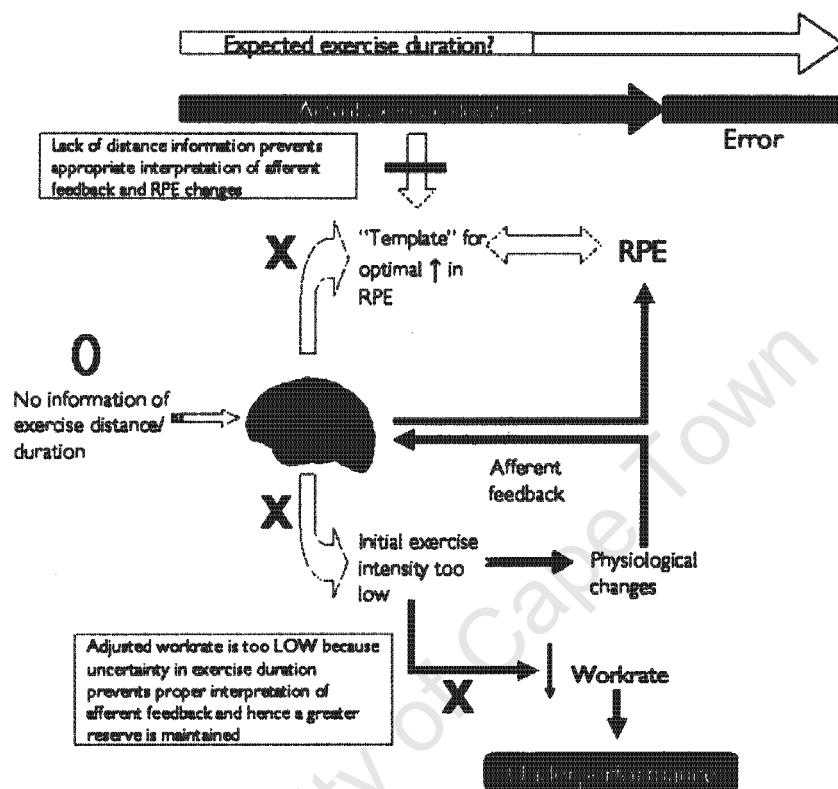


Figure 37. Schematic diagram showing how exercise performance would be impaired if no information regarding the exercise duration was provided prior to the commencement of exercise. Black shading denotes input to the brain, grey denotes output from the brain, dashed white lines depict processes which have been removed and are absent due to the lack of information.

Further, if the athlete is informed that the end of exercise is approaching, it is expected that the workrate will increase significantly, much more than would usually be the case in an 'endspurt'. This will occur because the uncertainty will have been instantly reduced from high levels to almost zero and the reserve that was maintained can then be instantly accessed to produce high workrates.

Limitations of the studies in the present thesis

A limitation in the present thesis is the use of surface EMG techniques to measure the extent of skeletal muscle activation. As described in the relevant chapters (Chapters 2 to 5), surface EMG provides an indirect measure of skeletal muscle activation, not muscle recruitment, and numerous factors can influence the EMG-Force output relationship (Marsden *et al.* 1983; Bilodeau *et al.* 2003). For example, it has been argued that peripheral changes in the muscle cause the motor neuron firing rate to decrease (MacLaren *et al.* 1989; Hepple, 2002; Westerblad *et al.* 2002), causing slower contraction and relaxation times (Marsden *et al.* 1983; Gandevia, 2001), producing a reduction in EMG that is not associated with a reduced activation level of skeletal muscle, and which is not measured using surface EMG techniques. Also only one active muscle was measured in three of the four studies using EMG techniques. This may mean that other muscles are being activated or recruited differently in the different conditions, but are not being detected. These limitations are described extensively in Chapters 2 and 5 of the present thesis.

At present, a number of promising techniques such as functional MRI, multimodal imaging and Transcranial Magnetic Stimulation are being investigated as methods to examine the central nervous system directly during exercise. However, at least for now, these are not feasible because of the large movement component during dynamic exercise, leaving only IEMG as an indirect means to measure activation

levels. As a result, it is not yet possible to definitively conclude that the motor unit recruitment is altered in anticipation of physiological changes that may be harmful.

Another limitation of the present thesis concerns the measurement of the proposed physiological afferents, particularly body temperature. Rectal temperatures have been measured during this thesis, and there is evidence that rectal temperature may show a delayed response to changes in internal and external temperature due to vascular perfusion of the rectum being lower than that of the deeper core (Rabinowitz et al. 1996; Blatteis 1998). This may affect the ability to infer which thermoregulatory variables are being monitored in the presently proposed anticipatory regulation model, since it is the timing of the changes in workrate that is essential, and rectal temperature may not provide the necessary resolution of measurement.

Similarly, in Chapter 3 of the present thesis, where it was shown that increasing the oxygen content of the inspired air improved time-trial performance, the physiological afferents such as β -adrenergic activation, systemic and cerebral arterial desaturation, and the reduction in skeletal muscle ATP, ADP and total NADH concentrations that may mediate this regulation (Figure 9 in Chapter 7) are difficult to measure during dynamic exercise. As a result, the potential mediators of the proposed regulatory system remain unelucidated, and future

research is required to identify with certainty whether these physiological afferents are responsible for mediating the proposed anticipatory regulation.

Suggestions for future research

The present thesis has proposed numerous models which explain how exercise performance of all types is regulated in anticipation of the potential loss of homeostasis in one or more physiological systems. Future studies must evaluate these models, either refuting them altogether or contributing to the understanding of how this regulation is achieved.

The main area of future research revolves around the limitations of the presently described studies. For example, as methods become available to examine the function of the motor cortex or muscle recruitment directly during dynamic exercise, it will become possible to evaluate the proposed models of the present thesis. Of specific interest in the present thesis is the regulation of short duration exercise lasting less than 120 seconds. This is of interest since there is now evidence that running speed or cycling power output decline even in the presence of a motor unit reserve and with evidence for peripheral fatigue (see Chapter 5 for details). Thus, the regulation of pacing strategy is a combination of both central and peripheral factors, but the mechanism is not known, and is unlikely to be fully understood until methods are available to quantify brain function and motor unit recruitment very accurately. However, there are some other possible

interventions that will contribute to a deeper understanding of the balance between the feedforward and feedback components of the prescribed models.

Firstly, a key assumption in the model for self-paced exercise is that the knowledge of the endpoint is a critical 'anchor' which allows the template RPE to be generated, and the consequent RPE to be interpreted in the context of the overall exercise bout. Thus, studies can manipulate the subject's anticipation of exercise duration. In particular, the interaction between the RPE and the knowledge of the endpoint is an interesting one, and it seems logical to assess this interaction using an RPE clamp protocol where the duration of exercise is either known or unknown (as was the case in Chapter 4). The hypothesis would be that performance will be improved when the exercise duration is known, even though the RPE would be identical in the two conditions. This would indicate that the RPE is in fact dependent on the known end-point of exercise, as proposed in Chapter 7 (Figure 34).

Another possible intervention involves manipulating the environmental conditions mid-way through the exercise bout. With particular reference to the present thesis, the oxygen content of the inspired air or the ambient temperature could be increased or decreased at the halfway point of a self-paced time-trial. This would alter the timing and the nature of the physiological feedback (D on Figure 34 in Chapter 7), and the resultant adjustments to pacing strategy (if any) could be

examined. This would provide a deeper understanding of the interaction between the feedforward and feedback components of the model.

Thirdly, future studies should also measure mechanical efficiency, particularly during shorter duration exercise, for this is a potential reason why the pacing strategy for these short duration events differs from that of longer bouts (See Chapter 5). There may very well be a relationship between the RPE and the athlete's efficiency, a function of technique. In particular, with fatiguing exercise, or at the end of self-paced trials when the RPE is highest, the mechanical efficiency may also be impaired.

Finally, a key component of the present model is previous experience and training, since the initial 'forecasting' of exercise duration and the interpretation of the resultant physiological feedback would both be influenced by the athlete's experience and learned responses to exercise. Studies should therefore examine how pacing strategy is different between trained and untrained populations, and should also investigate how familiarity with an exercise task alters both pacing strategy and perceived exertion. This may have applications for training of athletes in the future.

Conclusion

Based on the findings of the present thesis, and on an analysis of similar studies in the literature, various models have been proposed to explain how the anticipatory regulation of pacing strategies is achieved. This model incorporates anticipatory/feedforward as well as feedback components, using an expectation of exercise duration to set an initial workrate and to generate what has been termed a subconscious 'template' for the rate of increase in the RPE. During exercise, afferent feedback from numerous physiological systems is responsible for the generation of the conscious RPE, which is continuously matched with the subconscious template by means of adjustments in power output. This subjective rating is biologically linked, allowing pacing strategy to be adjusted to prevent catastrophic changes in the monitored physiological variables (homeostats).

In conclusion, the present thesis has provided evidence for the existence of a complex and intelligent system that regulates exercise performance and pacing strategies by means of adjustments in skeletal muscle motor unit recruitment, in order to protect against the development of harmful or limiting changes in physiological systems.

References

- Albertus Y, Tucker R, St Clair Gibson A, Lambert EV, Hampson DB & Noakes TD (2005). Effect of distance feedback on pacing strategy and perceived exertion during cycling. *Med Sci Sports Exerc* 37, 461-468.
- Ansley L, Robson PJ, St Clair GA & Noakes TD (2004). Anticipatory pacing strategies during supramaximal exercise lasting longer than 30 s. *Med Sci Sports Exerc* 36, 309-314.
- Armada-da-Silva PAS, Woods J & Jones DA (2004). The effect of passive heating and face cooling on perceived exertion during exercise in the heat. *Eur J Appl Physiol* 91, 563-571.
- Baden, DA (2002). Goals and expectations: Psychological and physiological effects of anticipating the end. PhD Thesis, University of Southampton.
- Baden DA, Mclean T, Tucker R, Noakes TD & St Clair Gibson A (2005). Effect of anticipation during unknown or unexpected exercise duration on rating of perceived exertion, affect, and physiological function. *Br J Sports Med* 39, 742-746.
- Baldwin J, Snow RJ, Gibala MJ, Garnham A, Howarth K & Febbraio M (2003). Glycogen availability does not affect the TCA cycle or TAN pools during prolonged, fatiguing exercise. *J Appl Physiol* 94, 2181-2187.
- Bassett DR & Howley ET (1997). Maximal oxygen uptake: 'classical' versus 'contemporary' viewpoints. *Med Sci Sports Exerc* 29, 591-603.
- Bassett DR & Howley ET (2000). Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Med Sci Sports Exerc* 32, 70-84.
- Bergstrom J, Hermansen L, Hultman E & Saltin B (1967). Diet, muscle glycogen and physical performance. *Acta Physiol Scand* 71, 140-150.

References

- Bigland-Ritchie B (1981). EMG/force relations and fatigue of human voluntary contractions. *Exerc Sports Sci Rev* 11, 75-117.
- Bilodeau M, Schindler-Ivens S, Williams DM, Chandran R & Sharma SS (2003). EMG frequency content changes with increasing force and during fatigue in the quadriceps femoris muscle of men and women. *J Elec Kinesiol* 13, 83-92.
- Bishop D, Bonetti D & Dawson B (2002). The influence of pacing strategy on VO₂ and supramaximal kayak performance. *Med Sci Sports Exerc* 34, 1041-1047.
- Blatteis CM (1998). *Physiology and pathophysiology of temperature regulation*. Singapore, World Scientific Publishing, 14-24
- Borg GA (1982). Psychophysical bases of perceived exertion. *Med Sci Sports Exerc* 14, 377-381.
- Brosnan MJ, Martin DT, Hahn AG, Gore CJ & Hawley JA (2000). Impaired interval exercise responses in elite female cyclists at moderate simulated altitude. *J Appl Physiol* 89, 1819-1824.
- Brown NJ, Stephenson LA, Lister G & Nadel ER (1982). Relative anaerobiosis during heavy exercise in the heat. *Fed Proc* 41, 1677-1682.
- Bruck K & Olschewski H (1987). Body temperature related factors diminishing the drive to exercise. *Can J Physiol Pharmacol* 65, 1274-1280.
- Burke LM, Hawley JA, Angus DJ, Cox GR, Clark SA, Cummings NK, Desbrow B & Hargreaves M (2002). Adaptations to short-term high-fat diet persist during exercise despite high carbohydrate availability. *Med Sci Sports Exerc* 34, 83-91.
- Cafarelli E (1982). Peripheral contributions to the perception of effort. *Med Sci Sports Exerc* 14, 382-389.

- Carey AL, Staudacher HM, Cummings NK, Stepto NK, Nikolopoulos V, Burke LM & Hawley JA (2001). Effects of fat adaptation and carbohydrate restoration on prolonged endurance exercise. *J Appl Physiol* 91, 115-122.
- Cheung SS & McLellan TM (1998). Influence of short-term aerobic training and hydration status on tolerance during uncompensable heat stress. *Eur J Appl Physiol* 78, 50-58.
- Cheung SS & Sleivert GG (2004a). Lowering of skin temperature decreases isokinetic maximal force production independent of core temperature. *Eur J Appl Physiol* 91, 723-728.
- Cheung SS & Sleivert GG (2004b). Multiple triggers for hyperthermic fatigue and exhaustion. *Exerc Sport Sci Rev* 32, 100-106.
- Colin J, Timbal J, Houdas Y, Boutelier C & Guieu JD (1971). Computation of mean body temperature from rectal and skin temperatures. *J Appl Physiol* 31, 484-489.
- de Koning JJ, Bobbert MF & Foster C (1999). Determination of optimal pacing strategy in track cycling with an energy flow model. *J Sci Med Sport* 2, 266-277.
- de Koning JJ, Foster C, Lampen J, Hettinga F & Bobbert MF (2005). Experimental evaluation of the power balance model of speed skating. *J Appl Physiol* 98, 227-233.
- Dennis SC & Noakes TD (1999). Advantages of a smaller body mass in humans when distance-running in warm, humid conditions. *Eur J Appl Physiol* 79, 280-284.
- Diamant B, Karlsson J & Saltin B (1968). Muscle tissue lactate after maximal exercise in man. *Acta Physiol Scand* 72, 383-384.
- Drust B, Rasmussen P, Mohr M, Nielsen B & Nybo L (2005). Elevations in core and muscle temperature impairs repeated sprint performance. *Acta Physiol Scand* 183, 181-190.

References

- Durnin JV & Womersley J (1974). Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *Br J Nutr* 32, 77-97.
- Enoka RM (1995). Morphological Features and Activation Patterns of Motor Units. *J Clin Neurophysiol* 12, 538-559.
- Fabiato A & Fabiato F (1978). Effects of pH on the myofilaments and the sarcoplasmic reticulum of skinned cells from the cardiac and skeletal muscles. *J Physiol* 276, 233-255.
- Farina D, Merletti R & Enoka RM (2004). The extraction of neural strategies from the surface EMG. *J Appl Physiol* 96, 1486-1495.
- Febbraio MA, Snow RJ, Stathis CG, Hargreaves M & Carey MF (1994). Effect of heat stress on muscle energy metabolism during exercise. *J Appl Physiol* 77, 2827-2831.
- Ferro A, Rivera A, Pagola I, Ferreruela M, Martin A & Rocandio V (2001). Biomechanical analysis of the 7th World Championships in Athletics Seville 1999. *IAAF New Studies in Athletics* 16, 25-60.
- Fink WJ & Costill DL (1975). Leg muscle metabolism during exercise in the heat and cold. *Eur J Appl Physiol* 34, 247-248.
- Foster C, de Koning JJ, Hettinga F, Lampen J, La Clair KL, Dodge C, Bobbert M & Porcari JP (2003). Pattern of energy expenditure during simulated competition. *Med Sci Sports Exerc* 35, 826-831.
- Foster C, deKoning JJ, Hettinga F, Lampen J, Dodge C, Bobbert M & Porcari JP (2004). Effect of competitive distance on energy expenditure during simulated competition. *Int J Sports Med* 25, 198-204.

Foster C, Schrager M, Snyder AC & Thompson NN (1994). Pacing strategy and athletic performance. *Sports Med* 17, 77-85.

Foster C, Snyder AC, Thompson NN, Green MA, Foley M & Schrager M (1993). Effect of pacing strategy on cycle time trial performance. *Med Sci Sports Exerc* 25, 383-388.

Fukuba Y & Whipp BJ (1999). A metabolic limit on the ability to make up for lost time in endurance events. *J Appl Physiol* 87, 853-861.

Fuller A, Carter RN & Mitchell D (1998). Brain and abdominal temperature at fatigue in rats exercising in the heat. *J Appl Physiol* 84, 877-883.

Galloway SD & Maughan RJ (1997). Effects of ambient temperature on the capacity to perform prolonged cycle exercise in man. *Med Sci Sports Exerc* 29, 1240-1249.

Gandevia SC (2001). Spinal and supraspinal factors in human muscle fatigue. *Physiol Rev* 81, 1725-1789.

Garland SW (2005). An analysis of the pacing strategy adopted by elite competitors in 2000 m rowing. *Br J Sports Med* 39, 39-42.

Gonzalez-Alonso J, Teller C, Andersen SL, Jensen FB, Hyldig T & Nielsen B (1999). Influence of body temperature on the development of fatigue during prolonged exercise in the heat. *J Appl Physiol* 86, 1032-1039.

Häkkinen K (1993). Neuromuscular fatigue and recovery in male and female athletes during heavy resistance exercise. *Int J Sports Med* 14, 53-59.

Hampson DB, St Clair Gibson A, Lambert MI & Noakes TD (2001). The influence of Sensory Cues on the Perception of Exertion During Exercise and Central Regulation of Exercise performance. *Sports Med* 31, 935-952.

References

- Hardy CJ & Rejeski WJ (1989). Not what, but how one feels - the measurement of affect during exercise. *J Sports Exerc Psych* 11, 304-317.
- Haseler LJ, Richardson RS, Videen JS & Hogan MC (1998). Phosphocreatine hydrolysis during submaximal exercise: the effect of $F_{I}O_2$. *J Appl Physiol* 85, 1457-1463.
- Havemann L, West SJ, Goedecke J, Lambert EV & Noakes TD (2006). Fat adaptation followed by carbohydrate-loading compromises high-intensity sprint performance. *J Appl Physiol* 100, 194 – 202.
- Hawley JA & Noakes TD (1992). Peak power output predicts maximal oxygen uptake and performance time in trained cyclists. *Eur J Appl Physiol* 65, 79-83.
- Henriksson J & Bonde-Petersen F (1974). Integrated electromyography of quadriceps femoris muscle at different exercise intensities. *J Appl Physiol* 36, 218-220.
- Hepple RT (2002). The role of O_2 supply in muscle fatigue. *Can J Appl Physiol* 27, 56-69.
- Hermansen, L. (1981). Muscle fatigue during maximal exercise of short duration. In *Physiological Chemistry of Exercise and Training*, eds. Di Prampero, P. E. & Poortmans, J., pp. 45-52. Karger, Basel, Switzerland.
- Hogan MC, Richardson RS & Haseler LJ (1999). Human muscle performance and PCr hydrolysis with varied inspired oxygen fractions: a ^{31}P -MRS study. *J Appl Physiol* 86, 1367-1373.
- Hogan MC & Welch HG (1984). Effect of varied lactate levels on bicycle ergometer performance. *J Appl Physiol* 57, 507-513.
- Howley ET, Cox RH, Welch HG & Adams RP (1983). Effect of hyperoxia on metabolic and catecholamine responses to prolonged exercise. *J Appl Physiol* 54, 59-63.

- Hunter AM, St Clair Gibson A, Lambert DL, Nobbs L & Noakes TD (2003). Effects of supramaximal exercise on the electromyographic signal. *Br J Sports Med* 37, 296-299.
- Hunter AM, St Clair Gibson A, Mbambo Z, Lambert MI & Noakes TD (2002a). The effects of heat stress on neuromuscular activity during endurance exercise. *Pflugers Arch* 444, 738-743.
- Hunter AM, St Clair GA, Lambert M & Noakes TD (2002b). Electromyographic (EMG) normalization method for cycle fatigue protocols. *Med Sci Sports Exerc* 34, 857-861.
- Ingen Schenau GJ, de Koning JJ & de Groot G (1992). The distribution of anaerobic energy in 1000 and 4000 metre cycling bouts. *Int J Sports Med* 13, 447-451.
- Ingen Schenau GJ, de Koning JJ & de Groot G (1994). Optimisation of sprinting performance in running, cycling and speed skating. *Sports Med* 17, 259-275.
- Ingen Schenau GJ, Jacobs R & de Koning JJ (1991). Can cycle power predict sprint running performance? *Eur J Appl Physiol Occup Physiol* 63, 255-260.
- Jacobs I & Kaiser P (1982). Lactate in blood, mixed skeletal muscle, and FT or ST fibres during cycle exercise in man. *Acta Physiol Scand* 114, 461-466.
- Jacobs I, Tesch PA, Bar-Or O, Karlsson J & Dotan R (1983). Lactate in human skeletal muscle after 10 and 30 s of supramaximal exercise. *J Appl Physiol* 55, 365-367.
- Karlsson J & Saltin B (1970). Lactate, ATP, and CP in working muscles during exhaustive exercise in man. *J Appl Physiol* 29, 596-602.
- Katch VL & Katch FI (1974). A simple anthropometric method for calculating segmental leg limb volume. *Res Q* 45, 211-214.

References

- Kay D, Marino FE, Cannon J, St Clair GA, Lambert MI & Noakes TD (2001). Evidence for neuromuscular fatigue during high-intensity cycling in warm, humid conditions. *Eur J Appl Physiol* 84, 115-121.
- Kayser B (2003). Exercise starts and ends in the brain. *Eur J Appl Physiol* 90, 411-419.
- Kayser B, Narici M, Binzoni T, Grassi B & Cerretelli P (1994). Fatigue and exhaustion in chronic hypobaric hypoxia: influence of exercising muscle mass. *J Appl Physiol* 76, 634-640.
- Lambert EV, St Clair Gibson A & Noakes TD (2005). Complex systems model of fatigue: integrative homeostatic control of peripheral physiological systems during exercise in humans. *Br J Sports Med* 39, 52-62.
- Lambert MI, Dugas JP, Kirkman MC, Mokone GG & Waldeck MR (2004). Changes in running speeds in a 100 km ultra-marathon race. *J Sports Sci Med* 3, 167-173.
- Linnarsson D, Karlsson J, Fagraeus L & Saltin B (1974). Muscle metabolites and oxygen deficit with exercise in hypoxia and hyperoxia. *J Appl Physiol* 36, 399-402.
- Linossier MT, Dormois D, Arsac L, Denis C, Gay JP, Geysant A & Lacour JR (2000). Effect of hyperoxia on aerobic and anaerobic performances and muscle metabolism during maximal cycling exercise. *Acta Physiol Scand* 168, 403-411.
- MacLaren DP, Gibson H, Parry-Billings M & Edwards RH (1989). A review of metabolic and physiological factors in fatigue. *Exerc Sport Sci Rev* 17, 29-66.
- MaClean BD (1993). The relationship between frontal surface area and anthropometric parameters in racing cyclists. *Proceedings, 14th Congress International Society of Biomechanics Paris*, 853-857.

- Marino FE (2004). Anticipatory regulation and avoidance of catastrophe during exercise-induced hyperthermia. *Comp Biochem Physiol B Biochem Mol Biol* 139, 561-569.
- Marino FE, Lambert MI & Noakes TD (2004). Superior performance of African runners in warm humid but not in cool environmental conditions. *J Appl Physiol* 96, 124-130.
- Marino FE, Mbambo Z, Kortekaas E, Wilson G, Lambert MI, Noakes TD & Dennis SC (2000). Advantages of smaller body mass during distance running in warm, humid environments. *Pflugers Arch* 441, 359-367.
- Marsden CD, Meadows JC & Merton PA (1983). 'Muscular wisdom' that minimizes fatigue during prolonged effort in man: peak rates of motoneuron discharge and slowing of discharge during fatigue. *Adv Neurol* 39, 169-211.
- Marsh AP & Martin PE (1995). The relationship between cadence and lower extremity EMG in cyclists and noncyclists. *Med Sci Sports Exerc* 27, 217-225.
- McLester JR, Jr. (1997). Muscle contraction and fatigue. The role of adenosine 5'-diphosphate and inorganic phosphate. *Sports Med* 23, 287-305.
- Mitchell D & Wyndham CH (1969). Comparison of weighting formulas for calculating mean skin temperature. *J Appl Physiol* 26, 616-622.
- Montain Sj & Coyle EF (1992). Influence of graded dehydration on cardiovascular drift and hyperthermia during exercise. *J Appl Physiol* 73, 1340-1350.
- Morrison S, Sleivert GG & Cheung SS (2004). Passive hyperthermia reduces voluntary activation and isometric force production. *Eur J Appl Physiol* 91, 729-736.
- Nielsen B (1996). Olympics in Atlanta: a fight against physics. *Med Sci Sports Exerc* 28, 665-668.

References

- Nielsen B, Hales JR, Strange S, Christensen NJ, Warberg J & Saltin B (1993). Human circulatory and thermoregulatory adaptations with heat acclimation and exercise in a hot, dry environment. *J Physiol* 460, 467-485.
- Nielsen B, Hyldig T, Bidstrup F, Gonzalez-Alonso J & Christoffersen GR (2001). Brain activity and fatigue during prolonged exercise in the heat. *Pflugers Arch* 442, 41-48.
- Nielsen B, Rowell LB & Bonde-Petersen F (1984). Cardiovascular responses to heat stress and blood volume displacements during exercise in man. *Eur J Appl Physiol Occup Physiol* 52, 370-374.
- Nielsen B, Savard G, Richter EA, Hargreaves M & Saltin B (1990). Muscle blood flow and muscle metabolism during exercise and heat stress. *J Appl Physiol* 69, 1040-1046.
- Nielsen B, Strange S, Christensen NJ, Warberg J & Saltin B (1997). Acute and adaptive responses in humans to exercise in a warm, humid environment. *Pflugers Arch* 434, 49-56.
- Nielsen HB, Boushel R, Madsen P & Secher NH (1999). Cerebral desaturation during exercise reversed by O₂ supplementation. *Am J Physiol* 277, H1045-H1052.
- Nikolopoulos V, Arkinstall MJ & Hawley JA (2001). Pacing strategy in simulated cycle time-trials is based on perceived rather than actual distance. *J Sci Med Sport* 4, 212-219.
- Noakes TD (1998). Maximal oxygen uptake: 'classical' versus 'contemporary' viewpoints: a rebuttal. *Med Sci Sports Exerc* 30, 1381-1398.
- Noakes TD (2000). Physiological models to understand exercise fatigue and the adaptations that predict or enhance athletic performance. *Scand J Med Sci Sports* 10, 123-145.

Noakes TD (2004). Linear relationship between the perception of effort and the duration of constant load exercise that remains. *J Appl Physiol* 96, 1571-1572.

Noakes TD, Peltonen JE & Rusko HK (2001). Evidence that a central governor regulates exercise performance during acute hypoxia and hyperoxia. *J Exp Biol* 204, 3225-3234.

Noakes TD & St Clair Gibson A (2004). Logical Limitations to the "Catastrophe" Models of Fatigue during Exercise in Humans. *Br J Sports Med* 38, 648-649.

Noakes TD, St Clair Gibson A & Lambert EV (2005). From catastrophe to complexity: a novel model of integrative central neural regulation of effort and fatigue during exercise in humans: summary and conclusions. *Br J Sports Med* 39, 120-124.

Nummela A, Vuorimaa T & Rusko HK (1992). Changes in force production, blood lactate and EMG activity in the 400-m sprint. *J Sports Sci* 10, 217-228.

Nunneley SA, Reader DC & Maldonado RJ (1982). Head-temperature effects on physiology, comfort, and performance during hyperthermia. *Aviat Space Environ Med* 53, 623-628.

Nybo L & Nielsen B (2001a). Hyperthermia and central fatigue during prolonged exercise in humans. *J Appl Physiol* 91, 1055-1060.

Nybo L & Nielsen B (2001b). Perceived exertion is associated with an altered brain activity during exercise with progressive hyperthermia. *J Appl Physiol* 91, 2017-2023.

Nybo L, Secher NH & Nielsen B (2002). Inadequate heat release from the human brain during prolonged exercise with hyperthermia. *J Physiol* 545, 697-704.

Parkin JM, Carey MF, Zhao S & Febbraio MA (1999). Effect of ambient temperature on human skeletal muscle metabolism during fatiguing submaximal exercise. *J Appl Physiol* 86, 902-908.

References

- Patterson MJ, Warlters D & Taylor NA (1994). Attenuation of the cutaneous blood flow response during combined exercise and heat stress. *Eur J Appl Physiol Occup Physiol* 69, 367-369.
- Peltonen JE, Leppavuori AP, Kyro K-P, Makela P & Rusko HK (1999). Arterial haemoglobin oxygen saturation is affected by F_{iO_2} at submaximal running velocities in elite athletes. *Scand J Med Sci Sports* 9, 265-271.
- Peltonen JE, Rantamäki J, Niittymäki SPT, Sweins K, Viitasalo JT & Rusko HK (1995). Effects of oxygen fraction in inspired air on rowing performance. *Med Sci Sports Exerc* 27, 573-579.
- Peltonen JE, Rantamäki J, Niittymäki SPT, Sweins K, Viitasalo JT & Rusko HK (1997). Effects of oxygen fraction in inspired air on force production and electromyogram activity during ergometer rowing. *Eur J Appl Physiol* 76, 495-503.
- Rabinowitz RP, Cookson SL, Wasserman SS & Mackowiak PA (1996). Effects of anatomic site, oral stimulation and body position on estimates of body temperature. *Arch Int Med* 156, 777-780.
- Ramanathan NL (1964). A new weighting system for mean surface temperature of the human body. *J Appl Physiol* 19, 531-533.
- Rasmussen P, Stie H, Nybo L & Nielsen B (2004). Heat induced fatigue and changes of the EEG is not related to reduced perfusion of the brain during prolonged exercise in humans. *J Therm Biol* 29, 731-737.
- Rauch HG, St Clair Gibson A, Lambert EV & Noakes TD (2005). A signalling role for muscle glycogen in the regulation of pace during prolonged exercise. *Br J Sports Med* 39, 34-38.

- Ray CA & Gracey KH (1997). Augmentation of exercise-induced muscle sympathetic nerve activity during muscle heating. *J Appl Physiol* 82, 1719-1725.
- Rejeski WJ & Ribisl PM (1980). Expected task duration and perceived effort: An attributional analysis. *J Sport Psychol* 39, 249-254.
- Robertson RJ (1982). Central signals of perceived exertion during dynamic exercise. *Med Sci Sports Exerc* 14, 390-396.
- Robinson S, Robinson DL, Mountjoy RJ & Bullard RW (1958). Influence of fatigue on the efficiency of men during exhausting runs. *J Appl Physiol* 12, 197-201.
- Ross, W. D. & Marfell-Jones, M. J. (1992). Kinanthropometry. In *The Physiological Assessment of High Performance Athletes*, eds. MacDougall, J. D., Wenger, H. A., & Green, H. J., Human Kinetics, Champaign, IL.
- Rowell LB, Marx HJ, Bruce RA, Conn RD & Kusumi F (1966). Reductions in cardiac output, central blood volume, and stroke volume with thermal stress in normal men during exercise. *J Clin Invest* 45, 1801-1816.
- Saltin B & Hermansen L (1966). Esophageal, rectal and muscle temperature during exercise. *J Appl Physiol* 21, 1757-1762.
- Sarre G, Lepers R, Maffiuletti N, Millet G & Martin A (2003). Influence of cycling cadence on neuromuscular activity of the knee extensors in humans. *Eur J Appl Physiol* 88, 476-479.
- Saunders AG, Dugas JP, Tucker R, Lambert MI & Noakes TD (2005). The effects of different air velocities on heat storage and body temperature in humans cycling in a hot, humid environment. *Acta Physiol Scand* 183, 241-255.

References

- Savard GK, Nielsen B, Laszczynska J, Larsen BE & Saltin B (1988). Muscle blood flow is not reduced in humans during moderate exercise and heat stress. *J Appl Physiol* 64, 649-657.
- Schabort EJ, Hawley JA, Hopkins WG, Mujika I & Noakes TD (1998). A new reliable laboratory test of endurance performance for road cyclists. *Med Sci Sports Exerc* 30, 1744-1750.
- St Clair Gibson A, Baden DA, Lambert MI, Lambert EV, Harley YX, Hampson D, Russell VA & Noakes TD (2003). The conscious perception of the sensation of fatigue. *Sports Med* 33, 167-176.
- St Clair Gibson A, Lambert EV, Rauch LHG, Tucker R, Baden DA, Foster C & Noakes TD (2006). The role of information processing between the brain and peripheral physiological systems in pacing and perception of effort. *Sports Med* 36, 705-722.
- St Clair Gibson A, Lambert MI & Noakes TD (2001a). Neural Control of Force Output During Maximal and Submaximal Exercise. *Sports Med* 31, 637-650.
- St Clair Gibson A & Noakes TD (2004). Evidence for complex system integration and dynamic neural regulation of skeletal muscle recruitment during exercise in humans. *Br J Sports Med* 38, 797-806.
- St Clair Gibson A, Schabort EJ & Noakes TD (2001b). Reduced neuromuscular activity and force generation during prolonged cycling. *Am J Physiol Reg Int Comp Physiol* 281, R187-R196.
- Steinacker JM, Marx TR, Marx U & Lormes W (1986). Oxygen consumption and metabolic strain in rowing ergometer exercise. *Eur J Appl Physiol* 55, 240-247.