Regulation of Pacing Strategies during Intermittent-Sprint Exercise: The Effects of Altered Physiological and Perceptual States on Team Sport Performance.

Melissa Skein
B.Ex Sci (Honours)

This thesis is submitted as requirement for the Doctor of Philosophy – Exercise Science Degree at Charles Sturt University

June 2012

Faculty of Education
School of Human Movement Studies
Table of Contents
Table of Contents........................................................................................................................................... i
Certificate of Authorship ............................................................................................................................ iii
Acknowledgements ......................................................................................................................................... iv
Publications .................................................................................................................................................. vi
Abbreviations .............................................................................................................................................. vii
List of Figures ................................................................................................................................................ x
List of Tables ................................................................................................................................................ xii
Abstract .................................................................................................................................................... xiv

Chapter 1: Introduction ............................................................................................................................. 1
  1.1 Introduction .............................................................................................................................................. 2
  1.2 Pacing Strategies ................................................................................................................................. 3
  1.3 Mechanisms Regulating Pacing Strategies .......................................................................................... 3
  1.4 Pacing Strategies in ISE and Alterations in Pre-Exercise Physiological and Perceptual States ... 5
  1.5 Sleep Deprivation ............................................................................................................................... 7
  1.6 Carbohydrate Ingestion ....................................................................................................................... 9
  1.7 Thermoregulation ............................................................................................................................... 10
  1.8 Study 1 – Sleep Deprivation .............................................................................................................. 12
  1.9 Study 2 – Carbohydrate Ingestion ..................................................................................................... 13
  1.10 Study 3 – Thermoregulation ............................................................................................................ 13
  1.11 Thesis Aims ......................................................................................................................................... 14
  1.12 Justification of the thesis .................................................................................................................. 14
  1.13 Limitations ........................................................................................................................................ 15
  1.14 Delimitations .................................................................................................................................... 16

Chapter 2: Review of Literature............................................................................................................. 17
  2.1 Introduction .......................................................................................................................................... 18
  2.2 Team Sport Exercise ............................................................................................................................ 20
  2.3 Pacing Strategies and Regulation of Fatigue during Self-Paced Exercise ............................................ 27
  2.4 Sleep deprivation ............................................................................................................................... 57
  2.5 Carbohydrate Ingestion ..................................................................................................................... 74
  2.6 Thermoregulation ............................................................................................................................. 92
  2.7 Conclusion ......................................................................................................................................... 123

Chapter 3: Reliability and Validity of a Laboratory-Based Self-Paced Intermittent-Sprint Exercise Protocol......................................................................................................................... 125
  Abstract ................................................................................................................................................... 126
  Introduction ............................................................................................................................................... 127
  Methods ................................................................................................................................................... 128
  Results .................................................................................................................................................... 131
  Discussion .............................................................................................................................................. 133

Preface
Preface

Chapter 4: Intermittent-Sprint Performance and Muscle Glycogen after 30 h of Sleep

Deprivation .................................................................................................................. 135
  Abstract .................................................................................................................. 136
  Introduction ........................................................................................................... 137
  Methods ................................................................................................................. 139
  Results .................................................................................................................... 146
  Discussion ............................................................................................................. 156

Chapter 5: The Effects of Carbohydrate Intake and Muscle Glycogen Content on Self-Paced Intermittent-Sprint Exercise Despite No Knowledge of Carbohydrate Manipulation .......... 163

  Abstract ............................................................................................................... 164
  Introduction .......................................................................................................... 165
  Methods ............................................................................................................... 167
  Results .................................................................................................................. 175
  Discussion ........................................................................................................... 185

Chapter 6: Self-paced Intermittent-Sprint Performance and Pacing Strategies Following Respective Pre-Cooling and Heating ................................................................................. 191

  Abstract ............................................................................................................... 192
  Introduction .......................................................................................................... 193
  Methods ............................................................................................................... 195
  Results .................................................................................................................. 202
  Discussion ........................................................................................................... 212

Chapter 7: General Discussion ..................................................................................... 220

  7.1 Overview of the Thesis ...................................................................................... 221
  7.2 Major Findings of the Respective Studies .......................................................... 222
  7.3 Descriptions of the Regulation of Pacing Strategies ........................................... 225
  7.4 Mechanisms and Models to Explain Pacing Strategies during ISE ..................... 231
  7.5 Summary of Pacing Strategies during Intermittent-Sprint Exercise ................. 245

Chapter 8: Summary and Conclusions ....................................................................... 246

  8.1 Overview ........................................................................................................... 247
  8.2 Research Aims – Chapter 3 .............................................................................. 247
  8.3 Research Aims – Chapter 4 .............................................................................. 247
  8.4 Research Aims – Chapter 5 .............................................................................. 248
  8.5 Research Aims – Chapter 6 .............................................................................. 249
  8.6 Thesis Aims ..................................................................................................... 250
  8.7 Summary and Conclusions .............................................................................. 252
  8.8 Practical Applications ...................................................................................... 253
  8.9 Recommendations for Future Research ........................................................... 254

Chapter 9: References .................................................................................................. 255

  Appendix A ............................................................................................................. 280
  Appendix B ............................................................................................................. 295
  Appendix C ............................................................................................................. 299
  Appendix D ............................................................................................................. 305
Certificate of Authorship

I, Melissa Skein,

“I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person nor material which to a substantial extent has been accepted for the award of any other degree or diploma at Charles Sturt University or any other educational institution, except where due acknowledgment is made in the thesis Regulation of Pacing Strategies during Intermittent-Sprint Exercise: The Effects of Altered Physiological and Perceptual States on Team Sport Performance. Any contribution made to the research by colleagues with whom I have worked at Charles Sturt University or elsewhere during my candidature is fully acknowledged. I agree that this thesis be accessible for the purpose of study and research in accordance with the normal conditions established by the Executive Director, Library Services or nominee, for the care, loan and reproduction of theses.” *

Signature ……………………………………………… Date ………………………

* Subject to confidentiality provisions as approved by the University
Acknowledgements

There have been so many people who have helped me through the completion of my PhD, and I thank you all sincerely for your contributions, collaborations and guidance.

Firstly, to Dr Rob Duffield, I don’t think I could thank you enough for all your time, support, guidance and patience you have given me during my PhD, or say how much I truly appreciate having you as my supervisor. I am indebted to you for all the time you have spent towards meetings, emails and phone calls, not to mention reading proposals, manuscripts and chapters. As my supervisor you lead by example with your work ethic and passion for research; while teaching many of life’s little lessons along the way. I am also very thankful for all the opportunities you have been able to give me that have helped me grow not only as a researcher but as a person, something I hope I can do when I have students of my own one day.

To Professor Frank Marino, thank you very much for all your help and guidance throughout my PhD. Despite your busy schedule, your door is always open to be of assistance and for one of your thought-provoking discussions; many of which times I walked out with more questions than what I walked in with! I am also very grateful for both the teaching and research all the opportunities you have provided me and helping me open my eyes to the world of academia.

To all the post-graduates in the SHMS both past and present, thanks for your help and support. It certainly makes it easier knowing you are not the only one going through the highs and lows of a PhD. To Katrina, Monique, Geoff, Cheyne, Amy, Danni, Alistair and Pete, your help with data collection, solving the problems of the world over a coffee, and social catch-ups at the Church Bar have been critical in me keeping me sane, thank you!

Thank you to the SHMS academic and administrative staff, I have appreciated the support each of you has provided me during my PhD both in teaching and research. A special thank you to Dr Jack Cannon for your assistance setting up and writing the scripts for my neuro data, this aspect of my work would not have been possible without you.
Thank you to the crew in the School of Sport and Exercise at Massey University for the use of your labs and students for data collection; but more importantly your friendship and support during each of my visits. Palmy North has become my home away from home the past few years thanks to each of you opening your homes and lives to a young, naïve PhD student from Oz. A special thank you to Dr Toby Mündel for your advice, guidance and particularly your friendship over the past few years. Also, thank you to the late Dr Hans Edge for your advice towards my studies and your words of wisdom on how to survive a PhD, your help was invaluable and like you, will never be forgotten.

To all research participants, thank you for volunteering to participate in my studies, none of this could have been completed without your involvement. Thank you to all my research assistants and the lab staff for your help. A special thanks also to Brad Kelly at UWA for your time and expertise in helping me to complete my muscle glycogen analysis.

To Mum, Dad, Lou, Muzz, Amanda and Jake, I can’t thank you enough for your support throughout my PhD. In your own personal way, each of you has given me timely encouragement, support or a necessary distraction to get through this PhD. I truly appreciate it and it is probably something I didn’t say often enough. Also, thank you Jean and Kerry for your unquestionable support for Mark and I during my PhD which we are most grateful for.

Finally, to Mark, thank you for all the love and support you have given me throughout this PhD. You gave me the belief that I could do this, particularly when times got tough and overwhelming. The support and help you have given me during the PhD has surpassed anything I ever expected. You have been patient and understanding about the endless hours at work and have been so supportive of me trying to maximise any opportunities at work, including disappearing to NZ a few times. This PhD would not have been possible without you and I hope one day I can return the love, support and encouragement you have given me as my best friend and husband.
Publications


### Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADP</td>
<td>Adenosine Di-phosphate</td>
</tr>
<tr>
<td>ATP</td>
<td>Adenosine Tri-phosphate</td>
</tr>
<tr>
<td>AFL</td>
<td>Australian Football League</td>
</tr>
<tr>
<td>AH</td>
<td>Active Heating</td>
</tr>
<tr>
<td>ANOVA</td>
<td>Analysis of Variance</td>
</tr>
<tr>
<td>au</td>
<td>Arbitrary Units</td>
</tr>
<tr>
<td>BP</td>
<td>Blood Pressure</td>
</tr>
<tr>
<td>bpm</td>
<td>Beats Per Minute</td>
</tr>
<tr>
<td>bw</td>
<td>Body Weight</td>
</tr>
<tr>
<td>CAR</td>
<td>Central Activation Ratio</td>
</tr>
<tr>
<td>CD</td>
<td>Contraction Duration</td>
</tr>
<tr>
<td>CHO</td>
<td>Carbohydrate</td>
</tr>
<tr>
<td>CNS</td>
<td>Central Nervous System</td>
</tr>
<tr>
<td>CONT</td>
<td>Control</td>
</tr>
<tr>
<td>CV</td>
<td>Coefficient of Variation</td>
</tr>
<tr>
<td>CWI</td>
<td>Cold Water Immersion</td>
</tr>
<tr>
<td>dw</td>
<td>Dry Weight</td>
</tr>
<tr>
<td>EMG</td>
<td>Electromyography</td>
</tr>
<tr>
<td>ex</td>
<td>Exercise</td>
</tr>
<tr>
<td>FFA</td>
<td>Free Fatty Acids</td>
</tr>
<tr>
<td>Glu</td>
<td>Glucose</td>
</tr>
<tr>
<td>GXR</td>
<td>Graded Exercise Run</td>
</tr>
<tr>
<td>HCHO</td>
<td>High Carbohydrate</td>
</tr>
<tr>
<td>HCl</td>
<td>Hydrochloric Acid</td>
</tr>
<tr>
<td>HEAT</td>
<td>Passive Heating</td>
</tr>
<tr>
<td>HIA</td>
<td>High-Intensity Activities</td>
</tr>
<tr>
<td>HK</td>
<td>Hexokinase</td>
</tr>
<tr>
<td>HR</td>
<td>Heart Rate</td>
</tr>
<tr>
<td>HRT</td>
<td>Half Relaxation Time</td>
</tr>
<tr>
<td>HT</td>
<td>Half-Time</td>
</tr>
<tr>
<td>ICE</td>
<td>Ice Bath</td>
</tr>
<tr>
<td>iEMG</td>
<td>Integrated Electromyography</td>
</tr>
<tr>
<td>IMP</td>
<td>Inosine monophosphate</td>
</tr>
<tr>
<td>ISE</td>
<td>Intermittent-Sprint Exercise</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Description</td>
</tr>
<tr>
<td>--------------</td>
<td>-------------</td>
</tr>
<tr>
<td>ISOK</td>
<td>Isokinetic contraction</td>
</tr>
<tr>
<td>ISOM</td>
<td>Isometric contraction</td>
</tr>
<tr>
<td>K$_2$CO$_3$</td>
<td>Potassium Carbonate</td>
</tr>
<tr>
<td>La$^-$</td>
<td>Lactate</td>
</tr>
<tr>
<td>LCHO</td>
<td>Low Carbohydrate</td>
</tr>
<tr>
<td>LIA</td>
<td>Low Intensity Activities</td>
</tr>
<tr>
<td>LIST</td>
<td>Loughborough Intermittent Shuttle Test</td>
</tr>
<tr>
<td>LoA</td>
<td>Limits of Agreement</td>
</tr>
<tr>
<td>MAP</td>
<td>Mean Arterial Pressure</td>
</tr>
<tr>
<td>MCHO</td>
<td>Moderate Carbohydrate</td>
</tr>
<tr>
<td>MIA</td>
<td>Moderate Intensity Activities</td>
</tr>
<tr>
<td>MVC</td>
<td>Maximal Voluntary Contraction</td>
</tr>
<tr>
<td>MVF</td>
<td>Maximal Voluntary Force</td>
</tr>
<tr>
<td>MVT</td>
<td>Maximal Voluntary Torque</td>
</tr>
<tr>
<td>PCr</td>
<td>Creatine Phosphate</td>
</tr>
<tr>
<td>PFK</td>
<td>Phosphofructokinase</td>
</tr>
<tr>
<td>PH</td>
<td>Passive Heating</td>
</tr>
<tr>
<td>PLA</td>
<td>Placebo</td>
</tr>
<tr>
<td>$P_{\text{max}}$</td>
<td>Power at maximal aerobic oxygen consumption</td>
</tr>
<tr>
<td>$P_{\text{mean}}$</td>
<td>Mean power output</td>
</tr>
<tr>
<td>PO</td>
<td>Power Output</td>
</tr>
<tr>
<td>POMS</td>
<td>Profile of Mood States</td>
</tr>
<tr>
<td>Post-Int</td>
<td>Post-Intervention</td>
</tr>
<tr>
<td>Post-Ex</td>
<td>Post-Exercise</td>
</tr>
<tr>
<td>PPO</td>
<td>Peak Power Output</td>
</tr>
<tr>
<td>Pre-Int</td>
<td>Pre-Intervention</td>
</tr>
<tr>
<td>PSI</td>
<td>Physiological Strain Index</td>
</tr>
<tr>
<td>Pt</td>
<td>Peak Torque</td>
</tr>
<tr>
<td>$r$</td>
<td>Intra-Class Correlation</td>
</tr>
<tr>
<td>RER</td>
<td>Respiratory Exchange Ratio</td>
</tr>
<tr>
<td>RPE</td>
<td>Rating of Perceived Exertion</td>
</tr>
<tr>
<td>RR</td>
<td>Rate of Relaxation</td>
</tr>
<tr>
<td>RTD</td>
<td>Rate of Torque Development</td>
</tr>
<tr>
<td>SD</td>
<td>Standard Deviation</td>
</tr>
<tr>
<td>SDEP</td>
<td>Sleep Deprivation</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Description</td>
</tr>
<tr>
<td>--------------</td>
<td>------------------------------------------</td>
</tr>
<tr>
<td>SEM</td>
<td>Typical Error</td>
</tr>
<tr>
<td>SwR</td>
<td>Sweat Rate</td>
</tr>
<tr>
<td>T_{arm}</td>
<td>Forearm Skin Temperature</td>
</tr>
<tr>
<td>T_{calf}</td>
<td>Calf Skin Temperature</td>
</tr>
<tr>
<td>T_{core}</td>
<td>Core Temperature</td>
</tr>
<tr>
<td>TCP</td>
<td>Twitch Contractile Properties</td>
</tr>
<tr>
<td>T_{es}</td>
<td>Oesophageal Temperature</td>
</tr>
<tr>
<td>TDC</td>
<td>Total Distance Covered</td>
</tr>
<tr>
<td>TMA</td>
<td>Time Motion Analysis</td>
</tr>
<tr>
<td>TMS</td>
<td>Transcranial Magnetic Stimulation</td>
</tr>
<tr>
<td>TPt</td>
<td>Time to Peak Torque</td>
</tr>
<tr>
<td>T_{skin}</td>
<td>Skin Temperature</td>
</tr>
<tr>
<td>T_{stern}</td>
<td>Sternum Skin Temperature</td>
</tr>
<tr>
<td>T_{thigh}</td>
<td>Thigh Skin Temperature</td>
</tr>
<tr>
<td>TT</td>
<td>Time Trial</td>
</tr>
<tr>
<td>TTE</td>
<td>Time to Exhaustion</td>
</tr>
<tr>
<td>USG</td>
<td>Urine Specific Gravity</td>
</tr>
<tr>
<td>VA</td>
<td>Voluntary Activation</td>
</tr>
<tr>
<td>VCO_2</td>
<td>Carbon Dioxide Production</td>
</tr>
<tr>
<td>V_E</td>
<td>Minute Ventilation</td>
</tr>
<tr>
<td>V_{Emax}</td>
<td>Maximal Ventilation</td>
</tr>
<tr>
<td>VO_2</td>
<td>Oxygen Consumption</td>
</tr>
<tr>
<td>VO_{2max}</td>
<td>Maximal aerobic oxygen consumption</td>
</tr>
<tr>
<td>VO_{peak}</td>
<td>Peak aerobic oxygen consumption</td>
</tr>
<tr>
<td>vVO_{2max}</td>
<td>Velocity at maximal aerobic oxygen</td>
</tr>
<tr>
<td>WBGTT</td>
<td>Wet Bulb Globe Temperature</td>
</tr>
<tr>
<td>WU</td>
<td>Warm-Up</td>
</tr>
</tbody>
</table>
List of Figures

**Figure 2.1:** General profiles of a) Even Pacing Strategy, b) Positive Pacing Strategy, c) Negative Pacing Strategy, d) ‘All-out’ Pacing Strategy exhibited during self-paced exercise 32

**Figure 2.2:** Parabolic-shaped pacing strategies, most commonly exhibited during endurance events (Abbiss and Laursen, 2005) 35

**Figure 2.3:** Cardiovascular/Anaerobic model of fatigue (Abbiss and Laursen, 2005) 40

**Figure 2.4:** Overview of the Teleoanticipation Model proposed by Ulmer (1996) (Lambert et al., 2005) 45

**Figure 2.5:** Overview of the Central Governor Model during which centrally-mediated factors from the periphery and external environment (feedback) are interpreted by the brain for an appropriate feed-forward regulation of neural drive and pacing strategies (Noakes, 2011) 47

**Figure 2.6:** The Psychological-Motivation Model as proposed by Marcra (2008, 2009) 50

**Figure 3.1:** Overview of the Self-Paced Intermittent-Sprint Exercise (ISE) protocol that includes a 15 m maximal sprint at the start of each minute, followed by sub-maximal, self-paced exercise (hard running, jogging, walking, bounds) of various intensities for the remainder of the minute 130

**Figure 4.1:** Mean ± SD a) total distance covered during self-paced bouts during 10 min phases of intermittent-sprint exercise (ISE), b) mean distance covered during hard running efforts, c) mean sprint times during ISE with (CONT1 and CONT2) or without sleep (SDEP1 and SDEP2) 149

**Figure 4.2:** Mean ± SD a) maximal voluntary force (MVF) and b) voluntary activation (VA), measured pre-GXR (PRE) and post-ISE (POST) either with (CONT1 and CONT2) or without sleep (SDEP1 and SDEP2) 152

**Figure 4.3:** Mean ± SD a) heart rate, b) core temperature, c) RPE pre-exercise and during the 50-min ISE protocol either with (CONT1 and CONT2) or without sleep (SDEP1 and SDEP2) 153

**Figure 5.1:** Mean ± SD muscle glycogen concentration pre and post intermittent-sprint protocol on Day 2 for high (HCHO) and low (LCHO) conditions, respectively (n=7) 176

**Figure 5.2:** Mean ± SD a) total distance covered during each 10 min phase of the intermittent-sprint protocol and b) distance covered during the hard running efforts during the intermittent-sprint protocol for high (HCHO) and low (LCHO) carbohydrate conditions respectively 179

**Figure 5.3:** Mean ± SD a) heart rate and b) rating of perceived exertion (RPE) on Day 1 (cycling), c) heart rate and d) RPE on Day 2 (intermittent-sprint protocol) for high (HCHO) and low (LCHO) conditions, respectively 181
Figure 6.1: Mean ± SD a) sprint efforts during the ISE; b) hard running efforts during the ISE; and c) total distance covered during respective 10 min phases of the ISE for control (CONT), ice-bath (ICE) and passive-heating (HEAT) conditions .................................................. 203

Figure 6.2: Mean ± SD a) maximal voluntary torque (MVT) and b) voluntary activation (VA) during 15 superimposed maximal isometric contractions pre-intervention (Pre Int), post-intervention (Post Int), and post-exercise (Post Ex) for control (CONT), ice-bath (ICE) and passive-heating (HEAT) conditions .................................................................................................................. 207

Figure 6.3: Mean ± SD a) heart rate (HR); b) core temperature (Tcore); and c) skin temperature (Tskin) throughout the intervention and exercise during the control (CONT), ice-bath (ICE), and passive-heating (HEAT) conditions respectively .......................................................................................................................... 210

Figure 7.1: Mean ± SEM % peak speed during each 3 min block of sub-maximal efforts (hard running, jogging, walking) of the intermittent-sprint protocol for A) sleep deprivation (SDEP) and control (CONT); B) high CHO diet (HCHO) and low CHO diet (LCHO) conditions; C) pre-cooling (ICE), passive heating (HEAT) and control (CONT) conditions, respectively …… 228

Figure 7.2: Mean ± SD % peak speed for each hard running effort during A) sleep deprivation (SDEP) and control (CONT); B) high CHO diet (HCHO) and low CHO diet (LCHO) conditions; C) pre-cooling (ICE), passive heating (HEAT) and control (CONT) conditions, respectively .......................................................................................................................... 229

Figure 7.3: Mean ± SD % peak speed for each sprint during A) sleep deprivation (SDEP) and control (CONT); B) high CHO diet (HCHO) and low CHO diet (LCHO) conditions; C) pre-cooling (ICE), passive heating (HEAT) and control (CONT) conditions, respectively …… 230

Figure 7.4: A diagrammatic summary of the mechanisms that contributed to the regulation of pacing strategies during the present self-paced intermittent-sprint exercise (ISE) protocol and the use of pre-exercise circumstances to highlight the contributions each of the respective (central, peripheral and conscious) factors had on ISE pacing and the strategies during specific modes of the ISE protocol........................................................................................................................................ 234
List of Tables

**Table 2.1:** Summary of studies examining the effect of acute sleep deprivation on self-paced exercise performance and the effects of sleep loss on physiological and perceptual responses during steady state exercise……………………………………………………………………………………………… 65

**Table 2.2:** Summary of studies examining the effect of acute sleep deprivation on muscle strength and power and the concomitant effects of physiological and perceptual responses. 67

**Table 2.3:** Summary of the studies examining the effect of pre-exercise carbohydrate ingestion on physiological and perceptual responses and intermittent-sprint exercise performance……………… 78

**Table 2.4:** Summary of the studies examining the effect of pre-exercise carbohydrate ingestion on physiological and perceptual responses and self-paced exercise performance …………… 83

**Table 2.5:** Summary of the studies examining the effect of pre-exercise carbohydrate ingestion on physiological and perceptual responses and muscle strength and power performance……………… 84

**Table 2.6:** Summary of Studies Examining the Effect of Environmental Heat on Repeated-Sprint and Intermittent-Sprint Exercise Performance………………………………………………………... 102

**Table 2.7:** Summary of Studies Examining the Effect of Pre-Cooling and Passive Heating on Intermittent-Sprint Exercise Performance………………………………………………………... 103

**Table 2.8:** Summary of Studies Examining the Effect of Environmental Heat on Self-Paced Exercise Performance…………………………………………………………………………………… 108

**Table 3.1:** Mean ± SD session 1 and 2 performance values, intra-class correlation (ICC), coefficient of variation (CV), typical error (r) and limits of agreement (LOA) for performance, physiological and perceptual measures……………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………
Table 5.2: Mean ± SD venous and capillary blood glucose (Glu) and lactate (La) concentrations for the High CHO (HCHO) and Low CHO (LCHO) conditions on Day 1 (cycling) and Day 2 (ISE) of the experimental trials  ........................................................................................................ 182

Table 5.3: Mean ± SD peak maximal voluntary torque (MVT), voluntary activation (VA), and twitch contractile properties (TCP) including peak torque (Pt), time to peak torque (TPt), rate of torque development (RTD), rate of relaxation (RR), half relaxation time (HRT) and contraction duration (CD) for High CHO (HCHO) and Low CHO (LCHO) conditions on Day 1 (cycling) and Day 2 (ISE) of the experimental sessions ........................................................................................................ 184

Table 6.1: Mean ± SD total distance covered, hard running, jogging, and walking efforts and bounds during the control (CONT), ice-bath (ICE), and passive-heating (HEAT) conditions, respectively ........................................................................................................ 204

Table 6.2: Mean ± SD neuromuscular assessment of potentiated twitch contractile properties of peak torque (Pt), time to peak torque (TPt), rate of torque development (RTD), rate of relaxation (RR), half relaxation time (HRT) and contraction duration (CD) pre-intervention (Pre-Int), post-intervention (Post-Int) and post-exercise (Post-Ex) during the control (CONT), ice-bath (ICE) and passive-heating (HEAT) conditions ........................................................................................................ 208

Table 6.3: Mean ± SD physiological strain index (PSI) and perceptual ratings of perceived exertion (RPE), thermal stress (Thermal) and capillary blood measure of pH, lactate (La), and glucose (Glu) during the control (CONT), ice-bath (ICE), and passive-heating (HEAT) conditions, respectively ........................................................................................................ 211

Table 7.1: Overview of pacing strategies adopted during the sub-maximal, hard running, and sprint efforts of the self-paced intermittent-sprint exercise protocol completed during the respective studies of the present thesis ........................................................................................................ 227
Abstract

Pacing strategies are the regulation of exercise intensities throughout a given bout of (continuous or intermittent) self-paced exercise in response to the exercise and environmental-induced demands placed on the athlete. Models that have been proposed to explain the development of fatigue and the regulation of exercise intensities include i) peripheral perturbations directly affecting the ability to sustain a given intensity; ii) a centrally-mediated manifestation regulating peripheral perturbations via changes in neural drive from the central nervous system (CNS) and iii) the conscious decision to adjust exercise intensity based on the perception of the physical demands. These mechanisms associated with fatigue-regulation have been investigated in prolonged, continuous exercise, but less so in team sports. Such investigations are relevant to team sport athletes, as competition matches are self-paced, with diverse physical demands and thus pacing strategies may be important to ensure optimal competition performance. Accordingly, as pacing strategies are suggested to be either due to changes at the periphery, or a central regulation of these peripheral changes; an effective method to assess the pacing mechanisms in team sports is via the manipulation of athlete’s pre-exercise state to examine the effects on subsequent performance and pacing. The pre-exercise circumstances used to alter physiological and perceptual states in the present thesis are commonly experienced by team sport athletes participating in the various football codes and alter central, peripheral or conscious states. Accordingly, this thesis examined the effects of sleep deprivation, carbohydrate (CHO) ingestion, and thermoregulation on pacing strategies and self-paced intermittent-sprint performance in team sport athletes.

Initially, the reliability of the intermittent-sprint exercise (ISE) protocol used throughout the thesis was examined. On two separate occasions, ten male moderately trained, team sport athletes completed a 40 min ISE protocol within an enclosed laboratory and controlled environmental conditions. The ISE protocol included 15 m maximal sprints every minute and was interspersed with self-paced exercise of various intensities (hard running, jogging, walking and double-leg bounding) with heart
rate and rating of perceived exertion (RPE) measured throughout. Results indicate the intra-class correlation ($r$) and coefficient of variation (CV) for maximal sprint times and total distance covered were 0.90 and 0.96, and 1.9% and 1.5% respectively. Further heart rate and RPE were of high reliability with $r= 0.94$ and 0.92, and CV= 1.2 and 6.6%, respectively. Overall, the test-retest reliability of performance, physiological and perceptual variables during the ISE protocol are within an acceptable range.

The first study of the thesis examined the effects of 30 h sleep deprivation on pacing strategies and self-paced intermittent-sprint performance in team sport athletes. Following a familiarisation session, ten male subjects completed two, consecutive-day experimental trials that consisted of exercise on both days and was separated by either a normal night sleep (CONT) or overnight (30 h) sleep deprivation (SDEP). Exercise protocols on both days included 30 min graded exercise run (GXR) and 50 min self-paced, intermittent-sprint exercise (ISE). The ISE protocol included a 15 m maximal sprint every minute that was proceeded by sub-maximal, self-paced exercise of varying exercise intensities (hard running, jogging, walking, and bounds) for the remainder of the minute. Muscle biopsies were obtained pre and post exercise on Day 2 of the trials, while neuromuscular assessments of maximal voluntary force (MVF), voluntary activation (VA) were obtained on both days. Results showed significantly slower sprints times (CONT2: $2.74 \pm 0.14$ s vs SDEP2: $2.78 \pm 0.17$ s; $P= 0.01$) and less distance was covered during the sub-maximal bouts of the first (CONT2: $776 \pm 63$ m vs SDEP2: $763 \pm 56$ m) and final (CONT2: $663 \pm 77$ m vs SDEP2: $635 \pm 56$ m) 10 min of the ISE protocol following SDEP. Further, significant reductions in pre-exercise mood states, muscle glycogen concentration, MVF and VA were evident following SDEP. These findings suggest that negative mood states and reduced muscle glycogen following acute exposure to sleep deprivation reduces maximal sprint performance and down-regulates pacing strategies during the self-paced, sub-maximal efforts of the ISE protocol. Due to the moderate differences in glycogen, it is likely that the
altered mood states primarily affected pacing strategies, thus suggesting changes in exercise intensity following sleep loss is predominately a conscious decision.

The second study of the thesis examined the effects of high and low CHO diets and muscle glycogen content on intermittent-sprint performance and pacing strategies. Uniquely, the manipulation of CHO diets and glycogen content were blinded to the subjects. Ten males completed two experimental trials that each included consecutive days of exercise. Day 1 included 95 min of a glycogen-depleting upper and lower body cycling bout, while Day 2 included 60 min of the previously mentioned ISE protocol. Following Day 1, subjects consumed either a high (HCHO; 7g/kg bw) or low (LCHO; 2g/kg bw) CHO diet. Pre and post-ISE muscle biopsies were obtained on Day 2, while pre and post exercise maximal voluntary torque (MVT), VA, twitch contractile properties (TCP), blood glucose (Glu) and lactate (La), heart rate (HR) and rating of perceived exertion (RPE) were recorded throughout the exercise protocols on both days. Pre-ISE muscle glycogen was greater in HCHO compared with LCHO (597 ± 115 v 318 ± 72mmol kg⁻¹ dw; P=0.001), which resulted in significantly more hard running (HCHO: 2279 ± 203 m vs LCHO: 2094 ± 251 m) and total distance (HCHO: 4750 ± 485 m vs LCHO: 4518 ± 544 m) compared to LCHO (P=0.02–0.04). Peak MVT, VA, HR, La and RPE were not different between conditions (P>0.05). This study adds to previous literature, showing that improvements in pacing strategies (particularly hard running) are evident despite no knowledge of dietary manipulation or metabolic status. Due to the blinded study design, exercise intensities seem manipulated due to peripheral perturbations regulated based on anticipation of demands associated with CHO content rather than a conscious manipulation of exercise intensities.

The final study examined the effects of pre exercise cooling and passive heating on pacing strategies and intermittent-sprint performance. Ten male, team-sport athletes completed three conditions followed by 50 min ISE protocol in the heat (31 ± 1°C; 33 ± 5% relative humidity). The respective conditions included (i) seated in a thermo-neutral environment (CONT); (ii) whole body submersion
in an ice-bath (ICE) and (iii) passive-heating in a hot environment (HEAT). Pre and post-intervention, and post exercise maximal isometric contractions were performed to determine MVT, VA, and TCP. Physiological measures including HR, core (T_{core}) and skin (T_{skin}) temperatures, capillary blood La and Glu, and perceptual ratings were recorded during the intervention and exercise protocols. Mean sprint times were significantly slower during ICE (2.83 ± 0.16 s) compared to HEAT (2.78 ± 0.14 s; \( P <0.05 \)) with no difference compared to CONT (2.80 ± 0.15 s). Total distance covered was not different between conditions but less distance was covered during HEAT during the final 20 min of the protocol (\( P <0.05 \)). Reductions in post-exercise MVT were present in CONT (172 ± 83 Nm) and HEAT (169 ± 21 Nm), with no effect of time on ICE (185 ± 40 Nm), whilst VA was reduced post-intervention and post-exercise in HEAT (\( P <0.05 \)). TCP including peak torque and relaxation rates were negatively affected by ICE compared to HEAT. HR, T_{core} and T_{skin} during exercise were lower in ICE compared to CONT and HEAT (\( P <0.05 \)). The findings from the present study further highlight the paradox between cooling and heating procedures prior to team sport exercise; with pre-cooling reducing sprint times and muscle contractile function but maintaining endurance performance compared to HEAT. Conversely, passive heating augmented maximal sprint efforts but accelerated the rate of rise in thermal strain, leading to less distance covered during the latter stages of the ISE protocol.

Collectively, these findings highlight alterations in pre-exercise physiological and perceptual states lead to significant alterations in pacing strategies and intermittent-sprint performance in team sport athletes participating in various football codes. The closer examination of pacing strategies indicates that overall pacing strategies of the sub-maximal efforts and pacing of individual modes (sprinting and hard running) were altered in response to the change in pre-exercise state. Further, the regulation of such exercise intensities was dependent upon both the pre-exercise physiological and perceptual states, while the knowledge of these states also influenced pacing when minimal physiological differences were present. In relation to the mechanisms responsible for pacing during ISE, the
delivery method of each of the pre-exercise circumstances demonstrates that rather than one mutually exclusive mechanism being responsible for changes in performance, pacing is the result of a complex integration of central, peripheral and conscious factors. Accordingly, pre-exercise circumstances that alter the muscle itself such as temperature and substrate availability can have direct effect on high-intensity performance; however, the overall pacing of self-paced intermittent-sprint exercise appears to be centrally regulated based on both peripheral feedback and knowledge of the circumstances. Therefore, team sport athletes in sports need to ensure they are in an optimal physiological and perceptual state prior to exercise to minimise peripheral and centrally-mediated reductions in exercise intensity that present as the implementation of slower pacing strategies and reduced performance.
Chapter 1

Introduction
Chapter 1 - Introduction

1.1 Introduction

The nature of team-sports, as represented by intermittent-sprint exercise (ISE), involves a complex interplay of self-paced efforts ranging from very low to very high intensities; all completed within the tactical demands of the sport (Bangsbo et al. 1991; Spencer et al. 2005). These highly variable exercise intensities invoke substantial loads on both physiological and perceptual systems (Bangsbo et al. 2006; Cunniffe et al. 2009). Given the high loads and prolonged durations, some evidence suggests that athletes regulate exercise intensities throughout a match based on factors such as the context of match loads, opposition, tactics, period of play, and/or environmental stress (Coutts et al. 2010; Duffield et al. 2009; Rampinini et al. 2007). For example, Mohr et al. (2003) reported the period of lowest exercise intensity within a soccer match occurs directly following a period of the highest intensity. Furthermore, the notion of pacing during self-paced intermittent-sprint exercise has been supported by laboratory-based protocols showing that a regulation of intensities (of predominately higher-intensities) is a result of altered interventions such as environmental heat, fluid ingestion and cooling (Duffield and Marino, 2007; Minett et al. 2011; Skein and Duffield, 2010).

The mechanisms responsible for the regulation of intermittent-sprint exercise suggest the physical demands may either i) directly affect the peripheral active musculature i.e. substrate depletion (Saltin, 1973) or metabolite accumulation (Krustrup et al. 2006); ii) result in a centrally-mediated reductions in muscle recruitment in a feedback/feed-forward manner (Tucker and Noakes, 2009); iii) increase the perceived effort or reduce the motivation to perform (Smith et al. 2011); or a combination of all three factors. All of these factors may exclusively or collectively contribute to the reduction in exercise intensity and resultant observation of ‘fatigue’. Accordingly, the fluctuating exercise modes and high physiological and perceptual loads during team sports create an intriguing model to study the regulation and interplay of exercise intensities and the mechanisms responsible for pacing strategies during such exercise.
1.2 Pacing Strategies

Pacing strategies have been defined as the efficient use of resources and the sufficient utilisation of energy expenditure during the completion of exercise without significant compromise of performance outcomes (Foster et al. 2003). Tucker and Noakes (2009) further elaborate that pacing can also include the regulation of other physiological variables such as the rate of heat storage. The most frequent assessment of pacing strategies involves (during continuous protocols) sampling power output at set distance intervals (Tucker et al. 2006), at a percentage of exercise duration completed during a time trial (Tucker et al. 2004), or during repeated sprints (Kay et al. 2001). It is understandable that the examination of such pacing strategies and the mechanisms responsible have primarily used continuous protocols due to the ability to perform the simultaneous collection of performance, physiological, perceptual, and neuromuscular data. However, such findings may not be appropriately transferred to intermittent-sprint exercise due to the varying and sporadic physical and physiological requirements of team sport exercise compared to continuous exercise protocols. Accordingly, further understanding of the pacing of intermittent-sprint exercise and mechanisms regulating such strategies during team-sport activities is required.

1.3 Mechanisms Regulating Pacing Strategies

The mechanisms responsible for pacing strategies during self-paced exercise are somewhat unclear, but have primarily been classified into two distinct areas regarding central and peripheral control, respectively (Davis and Bailey, 1997; Green, 1997; Lambert et al. 2005; Noakes et al. 2005). The traditional models proposed to explain fatigue are generally peripherally derived, in which fatigue and exercise termination are due to limitations within the muscles and organs (Davis and Bailey, 1997). Examples of these physiological changes include the inability of the heart to supply sufficient oxygenated blood to the active musculature (Calbet et al. 2004) and the inability to remove accumulated intramuscular metabolites (Green, 1997). These exercise-induced physiological perturbations that negate performance may also be accelerated due to additional factors such as
depleted muscle glycogen concentrations (Hargreaves 2004; Jeukendrup, 2008) or exercise in the heat resulting in a faster rate of rise in core temperature (Gonzalez-Alonso et al. 1999). In relation to team sports, decreases in intermittent-sprint performance have been related to lower glycogen content (Balsom et al. 1999b; Bangsbo et al. 1992) and increased thermoregulatory stress from exercise in the heat (Drust et al. 2005; Maxwell et al. 1996). Accordingly, the negative effects of exercise-induced increases in cardiovascular and metabolic loads are relevant to team sport athletes due to the prolonged physical and environmental loads encountered. However, it is unclear if these perturbations affect either overall physical performance or specific exercise modes within a match; or whether these physiological responses affect performance exclusively or in conjunction with other proposed mechanisms.

Compared to the traditional peripheral theories related to fatigue, more recent research suggests the central nervous system (CNS) has an integral role in regulating skeletal muscle recruitment and exercise performance (Noakes et al. 2005; St Clair Gibson, 2001). As such, centrally mediated models suggest that pacing is regulated by feed-forward changes in neural drive and muscle recruitment in response to feedback regarding the physiological load of exercise (St Clair Gibson and Noakes, 2004). Furthermore, Noakes et al. (2005) suggests that the feed-forward regulation of muscle recruitment to regulate physiological perturbations to protect the body and minimise disturbances from homeostasis. This protective mechanism has also been shown through the down-regulation of pacing prior to the increase in physiological stressors; suggesting the CNS anticipates the physiological loads required until completion of exercise and adjusts neural drive accordingly to avoid cellular damage (Marino, 2004; Ulmer, 1996). Similar to continuous exercise protocols, this pre-emptive down-regulation of exercise intensities may also be present during intermittent-sprint exercise in the heat (Castle et al. 2006; Morris et al. 2000). Accordingly, while the literature has shown the regulation of pacing strategies is present during both continuous and intermittent-sprint self-paced exercise, the mechanisms responsible for such responses are still somewhat elusive.
While previous theoretical models suggest the central regulation of fatigue is a sub-conscious process, research also supports the conscious regulation of exercise intensities (Marcora and Staiano, 2010). It is suggested that athletes consciously alter their exercise intensity based on motivation, perceived exertion, and the knowledge available to them regarding the impeding exercise bout, exercise/match context and physiological state (Billaut et al. 2011; Hampson et al. 2004). The use of placebos and deception interventions reveal that knowledge regarding one’s physiological state and/or the potential effects of that state can influence ensuing exercise performance (Clark et al. 2000; Nassif et al. 2008). Similar to previously mentioned physiological perturbations associated with intermittent-sprint exercise, the regulation of self-paced exercise could potentially incorporate the athletes’ RPE, motivation to perform, and knowledge of current physical demands.

Collectively, the avocation of each of these respective models and the applicability to various tasks means it is unclear whether performance and pacing changes following different pre-exercise interventions on intermittent-sprint performance are a result of (1) physiological changes directly affecting the contractile unit of the active musculature, (2) afferent feedback regarding these physiological changes are influencing the feed-forward neural drive to the active musculature or (3) is pacing all perceptual and in the mind of the athlete? Despite evidence that team sport athletes may regulate exercise intensities during a match (Carling et al. 2008; Coutts et al. 2010; Krustrup et al. 2005) minimal research has examined the mechanisms responsible for pacing regulation during self-paced intermittent-sprint exercise, and will therefore be explored through the studies completed in this thesis.

1.4 Pacing Strategies in ISE and Alterations in Pre-Exercise Physiological and Perceptual States

The examination of pacing strategies has been primarily used in continuous endurance protocols, often incorporating external interventions such as heat, nutrition, and prior fatigue to gain further
understanding of the mechanisms responsible for the regulation of exercise intensities (Marcora et al. 2009; Tucker, 2004, 2006). The use of externally manipulated conditions allow the alteration of factors related to central, peripheral, and/or conscious control of exercise intensity selection. However, it is likely the mechanism(s) regulating exercise intensity may be dependent upon the type of intervention administered, the effect on physiological and metabolic variables (Tucker and Noakes, 2009), and the subsequent task (Enoka 1995; St Clair Gibson et al. 2001). Accordingly, interventions that alter either central or peripheral factors, and manipulate the regulation of exercise intensities may provide further insight into the mechanisms responsible for pacing strategies. However, the use of interventions to study pacing strategy regulation in team-sports should be tailored to relate to ecologically valid circumstances that would confront athletes in the ‘real world’. To date, few studies examine the role of pacing during self-paced intermittent-sprint exercise with the use of pre-exercise circumstances to alter physiological and perceptual states (Castle et al. 2006; Duffield and Marino, 2007).

The development of ‘fatigue’ and the regulation of pacing strategies within the context of prolonged intermittent-sprint exercise are proposed to relate to a range of physiological perturbations such as a decrease in fuel substrates and accumulation of metabolites (Bangsbo et al. 2006; 2007; Mohr et al. 2003). In conjunction with the physiological changes associated with intermittent-sprint exercise, athletes participating in team sports such as soccer and rugby are also exposed to environmental and physiological circumstances that may be either advantageous or deleterious to performance. Such exposures may include altered nutritional interventions, increased thermoregulatory loads, and alterations to perceptual mood states (Balsom et al. 1999a; Bangsbo et al. 1992; Drust et al. 2005; Filaire et al. 2001; Maxwell et al. 1999). Specifically, altered perception and mood states are commonly observed in athletes following sleep deprivation (SDEP) (Angus et al. 1985; Myles, 1985; Scott et al. 1996). Sleep deprivation appears to have variable effects on physiological responses; however, research consistently reports changes in mood following sleep loss may be an important
regulator affecting conscious control of exercise intensity (Oliver et al. 2009). In relation to nutritional interventions, reductions in high-intensity activity during intermittent-sprint exercise have been linked to the depletion of muscle glycogen stores (Balsom et al. 1999b), whilst the reduction in pacing strategies during cycling trials have suggested a role for central regulation based on glycogen utilisation (Rauch et al. 2005). Finally, in the context of thermoregulation, declines in exercise performance in the heat have been associated with increased cardiovascular system load (Gonzalez-Alonso and Calbet, 2003; Rowell et al. 1966). However, more recent research suggests increased core temperature has a direct effect on the CNS, resulting in reduced neural drive to the active musculature (Nybo and Nielson, 2001; Thomas et al. 2006).

The aforementioned conditions (sleep deprivation, altered carbohydrate nutritional status and thermal stress) are ecologically valid to team sport athletes participating in football codes. Furthermore, given their hypothesised effects on conscious perception, peripheral and central factors regulating exercise, the use of these conditions creates a novel approach to understand how external factors affect the regulation of pacing in intermittent-sprint exercise. Specifically, the studies in this thesis will examine the effect of sleep deprivation, carbohydrate (CHO) diet manipulation, and thermoregulatory stress on pacing strategies during self-paced exercise and use these respective circumstances to provide information regarding the mechanisms responsible for exercise intensity regulation.

1.5 Sleep Deprivation

Acute sleep deprivation can be experienced by athletes the night before or following training and competition (Richmond et al. 2004), and can range from several hours to an entire night of sleep loss. The reasons responsible for such sleep loss in team sport athletes may include stress and anxiety, time schedules, or an athlete being forced into environments that are not conducive to sleep, such as travelling across multiple time zones (Reilly et al. 2005; Richmond et al. 2004). These bouts of acute sleep loss may be of significant interest to team sport athletes, as sleep loss has been associated with
declines in cognition, mood, and disruption to energy expenditure and hormone regulation; all of which may potentially negate subsequent exercise performance (Bonnet, 1980; Myles 1985; Opstad et al. 1991; Scott et al. 1996; Waterhouse et al. 2007). Despite the prevalence of sleep deprivation experienced by team sport athletes, no study to date has examined the effects of sleep deprivation on self-paced intermittent-sprint exercise performance.

The majority of research literature examining sleep deprivation have utilised fixed paced, endurance protocols or single-efforts of maximal strength. The examination of sleep loss on endurance exercise has highlighted that physiological perturbations associated with sleep loss have minimal effect on performance, as observed with the lack of differences in physiological responses during steady-state exercise following sleep loss (Martin, 1986; Plyley et al. 1987). On the contrary, exercise intensity selection (pacing strategy) appears to be reduced during self-paced exercise. These differences in performance findings between exercise modes may be due to the mechanisms responsible. As performance variables can be altered during self-paced and voluntary force efforts, the conscious awareness of sleep deprivation and the associated deleterious effects on mood states and RPE may influence exercise performance (Martin and Haney, 1982; Oliver et al. 2009 Reilly and Piercy, 1994; Symons et al. 1988; Takeuchi et al. 1985). Another proposed mechanism for performance declines following sleep deprivation has been related to metabolic and hormonal factors such as increased energy expenditure during hours of wakefulness affecting glucose metabolism (Driver and Taylor, 2000). Notwithstanding, it appears that the influence of these perceptual and metabolic factors on performance may be task dependent and have greater influence on tasks in which athletes can self-select their own pace or force output, thus explaining the discrepancies between studies.

The findings from previous literature suggest that sleep loss may have a greater effect on perceptual exertion and mood states than physiological responses. Consequently, sleep deprivation may be an appropriate model to assess the perceptual and conscious regulation of pacing strategies and
intermittent-sprint performance; however, the effect of sleep deprivation on these variables during self-paced intermittent-sprint performance has not been previously examined. Furthermore, given the concern regarding sleep quality for team sport athletes (Richmond et al. 2004), sleep deprivation also provides an ecologically valid model to understand the influence of negative mood states on the regulation of exercise intensities during self-paced intermittent-sprint exercise.

1.6 Carbohydrate Ingestion

Another pre-exercise state that is commonly experienced by team sport athletes such as soccer players is the nutritional manipulation of CHO ingestion and muscle glycogen content prior to exercise (Bangsbo et al. 2007). The importance of increased muscle glycogen content was first highlighted by Bergström and colleagues; with a series of studies highlighting a post-exercise reduction in glycogen concentrations in active musculature was a determinant of prolonged exercise performance (Bergström and Hultman, 1967). Following this initial work using laboratory-based studies, the importance of CHO intake and muscle glycogen content for football (soccer) players was highlighted using field-based research. Satlin (1973) showed that muscle glycogen concentrations are significantly reduced during a soccer match and a greater rate of depletion is present in Type I fibres. Later research has also reported high CHO (HCHO) diets prior to exercise are effective at increasing muscle glycogen stores and maintaining intermittent-sprint performance (Balsom et al. 1999b; Bangsbo et al. 2006). However, the influence of pre-exercise CHO ingestion between consecutive days of intermittent-sprint exercise and the effects on pacing strategies have not been previously examined. Given the ecological importance of optimal nutritional intake for athletes in various football codes, the role of CHO intake prior to self-paced, intermittent-sprint exercise is of interest as a method of improving exercise performance and understanding the mechanisms related to the development of fatigue during a match.
Time trial studies to elucidate the mechanisms responsible for performance improvements during self-paced exercise following CHO ingestion have reported conflicting results in regards to the conscious vs subconscious regulation of exercise intensities (Johnson et al. 2006; Rauch et al. 2005). Rauch et al. (2005) reported a pre-exercise CHO diet increased muscle glycogen content and improved 1 h cycling time-trial performance due to an increased maintenance of power output following the initial minute compared to the low CHO condition. Further, post-exercise glycogen was similar between conditions, with the authors suggesting there was an anticipatory regulation of exercise to avoid critically low glycogen levels based on the theoretical presence of a ‘gluco-stat’. On the contrary, Johnson et al. (2006) reported minimal differences in power output during the initial 80% of a 2 h cycling time-trial when subjects were blinded to their CHO condition, suggesting that initial changes in pacing are due to the knowledge of CHO ingestion rather than changes at the periphery. Such findings highlight the discrepancies in the mechanisms proposed to regulate self-paced exercise performance in response to changes in muscle glycogen content and the knowledge of such changes. Furthermore, the relative contribution of physiological perturbations compared to the conscious manipulation due to prior knowledge of CHO ingestion on pacing strategies in a team sport context is still unclear. Accordingly, the manipulation of nutritional CHO interventions provides an avenue to assess peripheral contributions to pacing strategies during team sport exercise, while blinding subjects to such manipulation may exclude the conscious regulation of exercise intensities.

1.7 Thermoregulation

A further issue often facing team-sport athletes relates to environmental stress of the climatic environment and the negative effects on intermittent-sprint performance (Drust et al. 2005; Maxwell et al. 1999). The increased exposure to environmental heat may be due to extended seasons, as well as pre-season training and competitions scheduled in the summer months. Accordingly, pre-cooling is a common strategy team sport athletes engage in to minimise the deleterious effects of intermittent-sprint exercise in the heat. Previous literature reports pre-cooling as advantageous to team sport
performance, primarily due to increased exercise intensities throughout the protocol (Castle, et al. 2006; Minett et al. 2011). The main premise for pre-cooling is to reduce an athlete’s core and peripheral temperatures, thus increasing the gradient between initial temperatures and attainment of excessive thermal stress that may be detrimental to performance (Gonzalez-Alonso et al. 1999). Despite the potential for cooling-induced slowing of nerve conduction rates or increased muscle stiffness (Bishop et al. 2003), pre-cooling is reported to be effective in reducing thermal strain associated with exercise in the heat and allowing selection of higher intensity pacing strategies during self-paced exercise (Duffield et al. 2010).

Team sport athletes also engage in more traditional ‘warm-up’ practices, for the known benefits of increased muscle temperature to improve high-intensity exercise performance (Gray et al. 2002). Further, passive heating (via exposure to warm environment) is proposed to provide similar temperature-dependent benefits to an active warm-up but without concerns of substrate depletion or metabolite accumulation (Bishop 2003). However, passive heating is also prone to increases in core temperature, which can be problematic given significant increases in core temperature are associated with declines in prolonged intermittent-sprint performance (Drust et al. 2005; Maxwell et al. 1996). Further, elevated core temperature has been related to reductions in neural drive to the active musculature and reduction in maximal force recruitment (Morrison et al. 2004). The contradictory pre-exercise interventions (cooling and heating) used by team sport athletes poses the question as to which intervention augments intermittent-sprint performance and pacing during the self-paced efforts. As warm environmental conditions are becoming increasingly evident during competition of all football codes, the use of pre-exercise thermoregulatory strategies that may have both positive and negative effects on performance are important to assist athletes monitor temperature-dependent changes to exercise performance. Accordingly, due to both central and peripheral contributions to fatigue during exercise in the heat, the manipulation of pre-exercise thermal stress may be an effective tool to assess the regulation pacing strategies in team sport athletes and the contributing mechanisms.
In summary, team sport athletes in sports such as soccer, rugby league and rugby union are subject to high physical loads resulting in stress on physiological and perceptual variables. Such changes to cardiovascular, thermoregulatory, metabolic and perceptual loads associated with repetitive, high-intensity exercise have been associated with performance declines (Krstrup et al. 2006; Mohr et al. 2003). Team sport athletes have the ability to regulate exercise intensities during the maximal sprint and sub-maximal efforts, with evidence suggesting such pacing occurs during a match (Mohr et al. 2003) and within laboratory settings (Skein and Duffield, 2010). Numerous models have been proposed in order to understand the regulation of exercise-induced fatigue and may potentially be extrapolated to intermittent-sprint exercise. Altering one’s physiological or perceptual state prior to exercise may provide insight into the mechanisms related to the regulation of pacing strategies during intermittent-sprint exercise. Interventions that can provide the greatest insight into the mechanisms responsible are those that are ecologically valid to team sports and can alter both central and peripheral mechanisms, such as nutritional interventions, thermoregulation and altering mood states.

Therefore, this thesis aims to examine pacing strategies during self-paced intermittent-sprint exercise and the associated mechanisms. Furthermore, altering pre-exercise physiological and perceptual states will provide an avenue to examine the mechanisms proposed to be responsible for the regulation of exercise intensities, whilst also providing information appropriate to improve team sport performance. Accordingly, the research aims and hypotheses for the respective studies and the thesis are:

1.8 Study 1 – Sleep Deprivation

Research Aims

1) The aim of this study was to examine the effect of ~30h sleep deprivation on self-paced intermittent-sprint performance and pacing strategies.
2) A secondary aim of the study was to examine the effect sleep deprivation on mood states and neuromuscular function, and the implications these factors may have on subsequent team sport exercise.

*Hypotheses*

1) It was hypothesised that ~30h sleep deprivation would result in a significant decline in intermittent-sprint performance and pacing strategies would be down regulated earlier during the protocol.

2) Compared to the control condition, sleep deprivation would negatively affect mood states, with minimal effects on physiological responses during exercise.

### 1.9 Study 2 – Carbohydrate Ingestion

*Research Aim*

1) The aim of this study was to examine the effect of a blinded pre-exercise CHO diet resulting in altered muscle glycogen concentration on intermittent-sprint performance and pacing strategies in team sport athletes on consecutive days of exercise.

*Hypothesis*

1) It was hypothesised that reduced CHO ingestion and resultant reduction in muscle glycogen content, without subject knowledge, would reduce exercise performance and pacing strategies during intermittent-sprint exercise.

### 1.10 Study 3 – Thermoregulation

*Research Aims*

1) This study aimed to examine the effect of pre-cooling and passive pre-heating on pacing and intermittent-sprint performance in warm environmental conditions.

2) A further aim was to determine the respective neuromuscular responses to the altered pre-exercise thermal state as a potential mechanism for changes in performance.
Chapter 1 - Introduction

Hypotheses

1) It was hypothesised that pre-cooling would result in the maintenance of higher exercise intensities throughout the intermittent-sprint protocol compared to the control or heating condition.
2) Additionally, it was hypothesised that the maintenance of exercise intensities would be due to a reduction in thermal strain and an associated maintenance of muscle recruitment.

1.11 Thesis Aims

Research Aims

1) Examine the effect of pre-exercise interventions, commonly experienced by team sport athletes, on pacing strategies and intermittent-sprint performance.
2) Examine the mechanisms responsible for pacing and how these respective interventions influence the relative contributions to physiological and perceptual stress and prior knowledge on the regulation of pacing.

1.12 Justification of the thesis

Although pacing strategies have been observed during team sport exercise (Carling et al. 2008; Mohr et al. 2003), laboratory-based, self-paced intermittent-sprint protocols (Duffield and Marino 2007; Skein and Duffield 2010), and prolonged continuous exercise protocols (Tucker, 2006); there remains robust debate regarding the mechanisms responsible for such exercise intensity regulation. Specifically, within a team-sport context, the complex interplay of high-intensity exercise interspersed with sub-maximal efforts may complicate the understanding of the contributing mechanisms responsible for the adoption of pacing strategies. The physical, physiological and perceptual demands placed on team sport athletes means the use of interventions to alter these respective demands may further elucidate how and why pacing strategies are regulated during team sports. The use of nutritional interventions, sleep deprivation, and thermoregulatory strain are also of high ecological validity as they are commonly experienced by many team sport athletes. Further, these respective pre-
exercise circumstances may be useful in helping explain the central and peripheral contributions to team sport exercise and the interplay of pacing between the different self-paced modes within a match. Accordingly, gaining further understanding of the implementation of pacing strategies in team sport athletes may assist coaches and scientists provide athletes with environments that are conducive for optimal performance and pacing.

1.13 Limitations

- Moderate-to-well trained male, team-sport athletes were the only subjects recruited for the respective studies, thus limiting the ability to extrapolate findings to female, older and youth athletes;

- Due to methodological constraints, neuromuscular tests were unable to be assessed during the self-paced exercise protocols, and therefore tests were completed before and immediately after exercise. The timing of these measures means that these are only an indication of the effects that the pre-exercise circumstances and exercise protocol had on neuromuscular properties both before and after exercise;

- In order to complete assessment of voluntary activation (VA) neuromuscular tests included maximal voluntary isometric contractions. Therefore, caution needs to be applied to extrapolating findings during isometric neuromuscular tests to changes in dynamic exercise;

- Single twitch pulses were used for the resting and superimposed twitches delivered during neuromuscular tests. Although single pulses are not as stable as double pulses, for subject comfort, single pulses were used. Further, single stimuli can be advantageous as they can reveal small declines in VA when high-resolution measurements of force are employed. Additionally, spinal reflexes have more time to influence superimposed twitch force when multiple stimuli are delivered during a maximal voluntary contraction (MVC);

- Due to methodological constraints of implementing the respective interventions, methodological design was different between each study, including consecutive days of
exercise during sleep deprivation and CHO studies. Furthermore, a 30 min graded exercise run (GXR) was completed during the sleep deprivation study, while the ISE protocol duration varied between chapters. These differences may create difficulties in comparing pacing strategies between studies.

1.14 Delimitations

- The protocol used during all three studies of the thesis has been designed to simulate the physical and physiological demands of team sport exercise. Time motion analysis (TMA) data and physiological variables during previous literature supports the workloads completed during protocol and the self-paced nature allowing pacing strategies to be implemented within the protocol and between conditions. However it is acknowledged whilst the protocol is self-paced, it is not a competitive match or with any tactical or technical element involved;

- Interventions used throughout the thesis were included as they are ecologically valid pre-exercise circumstances that are experienced and/or employed by team sport athlete;

- The use of pre-exercise circumstances that have a significant effect on physiological variables and/or perceptual state were used in all studies to provided an effective tool to assess the regulation of pacing strategies during self-paced intermittent-sprint performance;

- Sampling of subjects was consistent between all studies, with male amateur, club-representative level team sport athletes recruited;

- Within the Discussion Chapter, to allow the comparison of pacing strategies between studies, pacing strategies during sub-maximal, hard running, and sprints are represented as relative speed (% peak speed) for each individual subject rather than absolute speeds due to the different population groups;

- Pre-exercise circumstances were controlled with food and fluid abstained 3 h prior to exercise, and alcohol, caffeine and physical activity minimised 24 h prior to each experimental session.
Chapter 2

Review of Literature
2.1 Introduction

The initial setting and continuous manipulation of exercise intensities during an exercise bout are controlled by the athlete and are a characteristic of self-paced exercise. It is suggested that athletes alter pacing strategies during self-paced exercise to regulate physiological and perceptual responses, with the primary aim of minimising disturbances to homeostasis (Noakes et al. 2004; Tucker and Noakes, 2009). In research settings, pacing is measured in regards to the change in power output or speed during continuous, prolonged and often sub-maximal exercise (Atkinson and Brunskill, 2000; Tucker et al. 2004). Similar to endurance athletes, team sport athletes are also able to manipulate exercise intensities due to the self-paced nature of their respective sports (rugby league, union, AFL, soccer). Where different to continuous exercise, the sporadic intermittent nature of team sports creates difficulties in the assessment of pacing during team sports and the mechanisms responsible for alterations in exercise intensities.

Generally, team sports consist of prolonged exercise consisting of repeated short-bursts of maximal sprints interspersed with sub-maximal exercise of varying, self-selected intensities (Duthie et al. 2005; Spencer et al. 2004). The regulation of pacing strategies, the development of fatigue, and the mechanisms responsible are likely to be dependent on the specific tasks (task dependency) during a match or training. The high physical and physiological demands on the athlete can often result in performance reductions in the latter stages of the match; whilst transient fatigue may be reflected in the acute alteration in pacing strategies of exercise modes within short time frames of a match (Carling et al. 2009; Coutts et al. 2010; Duffield et al. 2010). Given the infinite permutations of match-specific activity profiles, the assessment of pacing strategies along with collection of physiological and perceptual data can be difficult in a field setting. Therefore, laboratory-based intermittent-sprint protocols may be used to simulate the physical and physiological demands of match-play, whilst also providing a controlled environment for the assessment of the research focus. Regardless, the first section of this literature review will briefly assess the physical and physiological
loads of team sports and the effect of such loads on the development of cumulative and transient fatigue (Carling et al. 2008; Mohr et al. 2005).

The regulation of exercise performance and pacing strategies during self-paced exercise is reported to be a central regulation of peripheral perturbations (St Clair Gibson, 2003). While it has been suggested that similar factors may be present during intermittent-sprint exercise (Maxwell et al. 1996; Krustrup et al. 2006), there is limited research examining the mechanisms responsible for the development of fatigue in intermittent-sprint compared to its continuous exercise counterparts. While extrapolation of findings from continuous exercise protocols may provide some insight, caution must be applied as exercise protocols, intensities, metabolic requirements, and thus causes of fatigue may differ i.e. task dependency (Enoka, 1995; Weir et al. 2006). Therefore, the second section of this literature review examines the models proposed to explain fatigue and exercise intensity regulation from both peripheral and central origins.

A popular method to assess pacing strategies during intermittent-sprint exercise is to intentionally alter an athlete’s pre-exercise physiological and/or perceptual state (Clarke et al. 2010; Maxwell et al. 1996; Skein and Duffield, 2010). The application of pre-exercise interventions may provide new perspectives on the mechanisms responsible for pacing in response to physiological and perceptual changes. Accordingly, the final sections of the literature review explore the effects of sleep deprivation, pre-exercise CHO intake, and altering pre-exercise thermal stress on exercise performance and the respective effects on central, peripheral and conscious regulation of exercise intensity. Within each intervention the potential mechanisms for performance and pacing alterations will be explored to provide further insight into the regulation of pacing during intermittent-sprint exercise. Overall this literature review will highlight the physical, physiological and perceptual loads associated with team sports, how these loads contribute to fatigue, and discuss the models proposed to explain the development of fatigue in relation to intermittent-sprint exercise. Finally, as pacing
appears to be a regulation of an athlete’s internal state in response to the external environment, this review will also outline the effects of central, peripheral and conscious factors of sleep deprivation, CHO intake and thermal stress as related to pacing during intermittent-sprint exercise.

2.2 Team Sport Exercise

Introduction

While extensive research interest is paid to team sport performance, a detailed understanding of the mechanisms responsible for development and regulation of fatigue are somewhat elusive. The ability to identify the timing and magnitude of reductions in performance during a match, in conjunction with the assessment of performance, may provide insight into the development of fatigue. To date, analysis of movement activities and physical demands suggest the presence of reduced distances covered during the second half of a match (cumulative fatigue) and temporary reductions in performance throughout the match (transient fatigue) (Coutts et al. 2010; Mohr et al. 2005). Due to the self-paced stochastic nature of team sports, the mechanisms responsible for these observations remain somewhat equivocal. Notwithstanding, there is evidence to suggest that exercise intensities may be regulated during self-paced intermittent-sprint performance, particularly via changes in sprint times and hard running distance covered (Duffield and Marino, 2007; Minett et al. 2011; Skein and Duffield, 2010).

Therefore, this section of the literature review will discuss the physical and physiological loads associated with team sports and how these loads affect the regulation of fatigue via changes in pacing strategies.

Physical Loads and Activity Patterns during Team Sports

Team sports differ considerably in terms of rules, surfaces, structure, and organisation; however, most team-based sports involve the repeated bouts of maximal to near-maximal efforts interspersed with recovery periods of varying intensities (Duthie et al. 2005; Glaister, 2005). Numerous motion analysis studies have been conducted on a wide range of team sports to quantify movement patterns, performance analysis, and determine physical and physiological demands for training loads to
simulate competitive bouts (Duthie et al. 2005; Spencer et al. 2004, 2005). One key characteristic of
team sports such as soccer and rugby, which differs to most laboratory intermittent-sprint exercise
protocols, is the self-paced nature of the exercise. During such exercise athletes are able to manipulate
exercise intensities throughout a given match, with intensities ranging from standing to maximal
sprinting. Often the physical demands of team sports are examined in relation to the overall distance
covered during a match, and distances and speeds within specific intensity zones. The total distance
covered during the various football codes is primarily dependent on the sport and level of
competition, with elite soccer players reported to covered approximately 10.86 ± 0.18 km for a 90 min
match, with slightly less distance covered by moderate-level players (10.33 ± 0.26 km) (Mohr et al.
2003). Compared to soccer, AFL players cover more distance during a match (11.7 – 12.3km), which
may in-part be due to the longer game duration (~120 min). Finally, rugby union players cover
substantially less distance (6.7 – 7.2 km) (Cunniffe et al. 2009) compared to other team sports,
possibly explained by the match duration and position requirements such as completion of physically
taxing rucks, mauls and scrums.

A closer analysis of team sport movement patterns highlights that athletes complete repeated bouts of
maximal efforts (sprints) throughout the match. During soccer matches, mean sprint durations are 2 -
4 s (Stolen, 2005) and the time between these sprints is approximately 40 – 90 s of active recovery
(Bangsbo et al. 2006; Mohr et al. 2003). Total distance of these sprint efforts during a soccer match
appear to be greater than rugby union with 670 - 695 m vs 94 - 524 m, respectively (Cunniffe et al.
2009; Deustch, 1998; Stolen, 2005). Other high-intensity activities during team sports (excluding
sprinting) are generally 3-5 s in duration, with variations between sports and player positions within
respective sports (Duthie et al. 2005; Mohr et al. 2005). Finally, lower intensity activities account for
the majority of the match time with reports of jogging, walking and standing accounting for ~16%,
42% and 19.5% of total match time, respectively (Gabbett and Mulvey, 2008; Mohr et al. 2005).
Accordingly, analysis of movement patterns in team sports shows that athletes perform a considerable
volume of work during a match within a range exercise intensities and durations. Due to the self-paced nature of team sports, athletes are able to dictate the work completed during a match, albeit within the technical and tactical context of the match; thus self-paced regulation of exercise intensities should be accounted for in any laboratory-based intermittent-sprint exercise protocol.

**Physiological and Perceptual Demands of Team Sport Exercise**

Physiological responses during team sport (football) exercise are reflective of the physical demands of repeated-bouts of high-intensity exercise over a prolonged period of time. Heart rate responses to field-based team sports have shown mean heart rates of approximately 166 – 180 bpm, with variation reflective of player position and movement patterns (Coutts et al. 2010; Deutsch et al. 2007; Duthie et al. 2003; Krustup, 2005). Heart rate responses during rugby union are approximately 173 bpm during the first half and 169 bpm during the second half of the match (Cunniffe et al. 2009). Rugby league mean heart rate values are similar to union, with Coutts et al. (2003) reporting mean heart rate of 166 ± 10 bpm for semi-professional players. During under 19’s rugby union matches, Deutsch et al. (1998) reported heart rates based on positional differences; with higher percentages of time at heart rates ≥ 85% of maximum for props, locks, and back row forwards, while outside backs spent the most time of a match (~20%) at heart rates <75% heart rate maximum. Finally, field hockey and basketball have reported mean heart rate responses of 159 ± 8 bpm and 171 ± 4 bpm, respectively (Abdelkrim et al. 2007; Spencer et al. 2004), all highlighting a prolonged and elevated cardiovascular load.

Although measurement of oxygen consumption (\(\dot{VO}_2\)) is not possible during a match, estimated \(\dot{VO}_2\) and energy expenditure has been conducted based on heart rate responses. Coutts et al. (2003) estimated oxygen consumption and energy expenditure from extrapolated heart rate responses and heart-rate-\(\dot{VO}_2\) regression equations obtained during an incremental treadmill test. Results indicate estimated mean \(\dot{VO}_2\) during a rugby league match was 47.1 ± 3.4ml kg\(^{-1}\) min\(^{-1}\), which was 81.0 ± 5.8% \(\dot{VO}_{2\text{max}}\) and an estimated energy expenditure of 7.9 ± 0.4MJ. Additionally, Krustup (2005) reported
during a female soccer match players exercised at an estimated oxygen consumption of 49.4ml kg$^{-1}$ min$^{-1}$ based on each individual’s HR-$\dot{V}O_2$ relationship from a preceding incremental $\dot{V}O_{2max}$ test. Accordingly, the estimated $\dot{V}O_2$ during team sports represent a significant and prolonged metabolic load in association with high cardiovascular demands throughout a given match.

Given the above highlighted metabolic demands and the high-intensity nature of various football codes, CHO sources are the primary fuel source during a match (Saltin, 1973). Depletion of muscle glycogen content has been reported during a soccer match, which have negative implications on high-intensity exercise performance during both the first and second halves of the match (Saltin, 1973; Krustrup et al. 2006). As muscle metabolites have been suggested to limit exercise performance, Krustrup et al. (2006) also examined water content, adenosine triphosphate (ATP), inosine monophosphate (IMP), creatine phosphate (PCr), lactate (La$'$), hydrogen, and pH from the muscle biopsy samples. Post-match ATP and PCr concentrations decreased while water content, venous blood and muscle lactate were all significantly higher compared to pre-match values. However, no definite conclusions were able to be drawn on the effects of changes muscle metabolites on fatigue during a soccer match. Regardless, it appears that muscle glycogen may have some role in the transient and progressive declines in high-intensity efforts during a soccer match (Krustrup et al. 2006; Saltin, 1973). Accordingly, the overall physiological load imposed on team sport athletes is representative of the high physical demands of repeated high-intensity exercise which may contribute to the development and regulation of fatigue in a match.

Similar to the aforementioned physiological variables, the sporadic, high intensity nature of team sports also invokes high perceptual loads on athletes. Mean sessional rating of perceived exertion (RPE) after an AFL match has been recorded as high as 8.6 ± 1.4 using the CR-10 rating scale (Duffield et al. 2009). Furthermore, Hill-Haas et al. (2009) have shown RPE responses to small-sided games to range from 10.5 – 12.2au (6 – 20 RPE scale). Coutts et al. (2009) have also highlighted that
elevated RPE during small-sided games is significantly correlated with higher lactate concentrations and percentage of peak heart rate. These high perceptual responses in the field are similar to RPE responses by athletes during intermittent-sprint performance in both thermo-neutral (Cheung and Robinson, 2004) and hot environmental conditions (Bishop et al. 2009; Castle et al. 2006; Pointon et al. 2011). More specifically, Castle et al. (2006) and Duffield and Marino (2007) reported peak RPE values during intermittent-sprint exercise in the heat of 18.0 ± 0.5 and 18.4 ± 1.6, respectively. Collectively, it appears that the high physiological demands associated with prolonged intermittent-sprint exercise are also associated with high perceptual strain.

Effects of the Physical and Physiological Loads on Development of Fatigue

As outlined above, team sport athletes are exposed to intense physiological responses as a result of prolonged bouts of high-intensity, intermittent activities. These alterations in physiological and perceptual states may have an effect on the development of fatigue and the regulation of pacing strategies during a given match (Carling et al. 2008; Mohr et al. 2005). However, due to the complex and field-based nature of soccer and rugby, it is difficult to quantify the mechanisms responsible for the cumulative and transient fatigue evident throughout the match. Efforts to explain these mechanisms in the field have included examination of pacing strategies during the respective movement patterns, while the use of team-sport simulated intermittent-sprint protocols have also been used to elucidate the mechanisms of fatigue within a more controlled environment.

Cumulative Fatigue

The most distinctive observation of cumulative fatigue during team sport matches is the reduction in distance covered during the second half (Carling et al. 2008). Time motion analysis has shown a 5-10% reduction in distance covered during the second half compared to the first half of a soccer match (Stolen et al., 2005), which is supported by Carling et al. (2008) who reported a 3.1% decline between halves during an elite soccer match. Similar to soccer, other team sports have also reported declines in
latter stages of the match including AFL (Wisbey et al. 2010), rugby union (McLean et al. 1992) and rugby league (King et al. 2009). More specifically, the presence of cumulative fatigue via the reduction in distance covered has primarily been associated with higher-intensity activities, with maintenance of moderate and lower intensity activities. For example, Rampinini et al. (2007) reported a reduction in high-intensity running during the second half of an elite level soccer match was responsible for a 25% reduction in total distance during the second half. Similarly Coutts et al. (2010) reported reductions in high intensity running during an AFL match were evident with a ~49% reduction in total distance covered during the second half of a match. Mohr et al. (2003) also reported that the declines in performance during an elite soccer match coincided with a 35-45% reduction in high-intensity running during the final 15 min of a match. Application of field-based findings to the laboratory have shown that self-paced intermittent-sprint protocols have reported similar results, with a progressive decline in distance covered of sub-maximal efforts, most notably during hard running efforts (Duffield and Marino, 2007; Skein and Duffield, 2010). These studies indicate that cumulative fatigue is present during intermittent-sprint exercise, and the pacing of specific exercise modes may contribute to the development of such decrements in physical performance.

**Transient Fatigue**

Within a given team sport match there is also evidence of transient fatigue, with undulations in exercise intensities (pacing strategies) implemented by the athlete in response to demands associated with the match. Most notably is the reduction in intensities following a bout of high-intensity exercise during a match (Carling et al. 2008; Mohr et al. 2005). Mohr et al. (2005) reported that in elite and moderate level soccer players, a reduction in intensity is present following a 5 min period of high-intensity exercise compared to the match mean. Furthermore, Coutts et al. (2010) reported during an elite AFL match, players down-regulated total distance, high intensity distance and work rate during a match quarter that was preceded by a quarter that accumulated the highest amount of work for the entire match. However, Aughey (2010) has suggested when game time is presented per minute of a
game, there are no differences in total work and work during low-intensity efforts are not of sufficient magnitude to affect a match; although it was noted that performance of maximal sprints and high-intensity efforts are reduced during the latter stages of an AFL match. Notwithstanding, similar findings to Coutts et al. (2010) have been reported during self-paced intermittent-sprint exercise in which subsequent exercise performance is reduced following high-intensity efforts. Skein and Duffield (2010) showed that sprint performance was dependent upon the preceding sub-maximal effort, with fastest sprint times following the walking efforts and slowest sprint times following hard running efforts. Collectively, these findings suggest that team sport athletes are continuously exposed to factors that are fatigue-invoking and result in transient reductions in exercise intensities.

The development of fatigue during team sports has been identified with a progressive reduction in performance during the second half of a match (cumulative fatigue) and has been associated with (but not exclusive to) muscle glycogen depletion, dehydration and increases in thermal strain (Edwards and Clark, 2006; Krstrup et al. 2005) Further, pacing strategies are also evident with fluctuations in performance of specific modes throughout a given match (transient fatigue). For example, the attainment of peak speeds at the end of a match/exercise protocol similar to efforts during the initial stages of exercise (Duffield et al. 2009) and reductions in distance covered during specific exercise modes (high-intensity efforts) for respective quarters of an AFL match when high volumes of work are completed in the preceding quarter (Coutts et al. 2010). Therefore, the examination of laboratory-based intermittent-sprint exercise with alterations in pre-exercise physiological and perceptual states may be an effective method to assess the development of fatigue throughout a given exercise bout and examine the interplay of exercise modes on overall performance.

**Conclusion**

In summary, team sport athletes are exposed to high physiological and perceptual loads, as evidenced by prolonged elevation of heart rate, estimated $\dot{VO}_2$, and substantial muscle glycogen depletion. These
elevated demands are in response to athletes completing prolonged bouts of exercise comprised of brief bouts of maximal sprints and active recovery of various exercise intensities. Accordingly, time motion analysis data reveals that the frequency, duration, and distances vary between sports, but all result in the development of both cumulative and transient fatigue. Further, it may be possible that the transient fatigue observed during team sports indicates modal pacing strategies in order to maintain overall performance. While cumulative and transient fatigue are present in team sports, it is unclear the mechanisms that are responsible for the development of fatigue and how and why athletes regulate pacing strategies. Therefore, the investigation into self-paced, intermittent-sprint exercise that allows the determination of performance and pacing strategies during respective modes may provide insight into the regulation of pacing by team sport athletes.

2.3 Pacing Strategies and Regulation of Fatigue during Self-Paced Exercise

Introduction
As outlined previously, team sport athletes are exposed to high physical demands with concomitant stressors on physiological and perceptual variables. The examination of how physical states affect team sport performance can be completed via the application of models that have been previously proposed to describe pacing strategies in prolonged continuous endurance exercise. The following section will outline the definitions of fatigue and pacing strategies, the types of pacing strategies, and how pacing is measured during continuous and intermittent exercise. Finally, the peripheral and centrally derived models proposed to explain fatigue and pacing strategies during self-paced exercise will be discussed with specific reference to the implications on self-paced intermittent-sprint exercise.

Definitions

Fatigue
The ability to delay the point at which fatigue or performance declines become present is important to athletes, coaches and exercise scientists. An understanding of fatigue and the ability to counter or
defend against excessive fatigue development may provide for further understanding of human physiological responses to exercise. An extensive volume of research has been completed on the timing and regulation of fatigue, and the type and location of causative mechanisms (Davis and Bailey, 1997; McKenna and Hargreaves, 2008; Noakes et al. 2005; Taylor and Gandevia, 2008; Westerblad et al. 2002). However, many research questions and hypotheses surrounding the research of fatigue during exercise are clouded by the interpretation and definition of the term ‘fatigue’. Definitions of fatigue seem to vary depending upon the context, the discipline (i.e. neuromechanics, psychology and physiology), and are task dependent (Enoka, 1995; St Clair Gibson et al. 2001). Generally, within a given exercise bout fatigue is defined as a “decrease in force production or the inability to regenerate the original force” (St Clair Gibson and Noakes, 2004, p797). In relation to this notion of fatigue, traditional mechanisms of fatigue are highlighted as peripheral perturbations that are responsible for the reduced force production and the termination of exercise (volitional exhaustion or task failure) (Hunter et al. 2004; Kent-Braun et al. 1999). Conversely, more recent theories have referred to fatigue as a process and thus regulated throughout the entirety of the exercise bout rather than perceived as an end-point (Noakes et al. 2005). Therefore, the regulation of fatigue may be represented as the inability to maintain a given power output or force production in response to physiological perturbations or in anticipation of imposed stress (Marino, 2004; Noakes, 2005; Lambert et al. 2005).

While the physical manifestations of fatigue, such as reduced force production have been extensively reported, the mechanisms related to the development of fatigue are less clear. Peripheral fatigue is defined as a decrease in the capacity of the skeletal muscle to generate force due to processes distal to the neuromuscular junction (Taylor and Gandevia, 2008). These changes may include action potential failure, excitation-contraction coupling failure, or impairments of cross-bridge cycling (St Clair Gibson and Noakes, 2004). On the contrary, central fatigue is considered as the reduction in force production due to processes proximal to the neuromuscular junction, including reduction in neural
drive from the CNS to the active musculature (Taylor and Gandevia, 2008). The vast majority of literature examining fatigue have developed an ‘either/or’ approach in which development of fatigue is referred to as exclusively either central or peripheral in origin (Marino et al. 2011). This approach to understanding fatigue may in itself create issues with the ability to identify and acknowledge all contributing factors to performance declines during a given exercise bout.

**Pacing Strategies**

As the development of fatigue is a process throughout an exercise bout, it may be possible that pacing strategies are the manifestation of the fatigue regulation (Marino et al. 2011). Pacing strategies have been defined as a within-race distribution of work rate (power output) (Atkinson and Brunskill, 2000). Foster et al. (2003) further elaborates that pacing strategies are the efficient use of fuel substrates to ensure optimised distribution of energetic resources to allow successful completion of the race. This definition has been extended to highlight that the regulation of energetic resources can also include other physiological variables; including heat storage, substrate availability, hypoxia, and dehydration (Billaut et al. 2010; Marino, 2004; Nybo and Nielsen, 2001; Steams et al. 2009; Tucker and Noakes, 2009). It has been suggested that the primary aim of regulating such pacing strategies is to prevent a change in physiological systems that may limit or be detrimental to performance and physiological functioning (Tucker and Noakes, 2009). Of particular note, pacing strategies are only evident during self-paced exercise, owing to the ability of an athlete to freely implement and adjust exercise intensities. Such a response is not possible during externally-paced exercise because, as the name suggests, exercise intensity is dictated by an external controller. The regulation of such pacing strategies have been examined extensively during individual sports such as cycling and running (Ansley et al. 2004; Hettinga et al. 2006; Nummela et al. 2008), although less attention has been paid to pacing during various modes of intermittent-sprint exercise (Skein and Duffield, 2010).
Types of Pacing Strategies

The examination of pacing strategies during self-paced exercise has gained increased interest over recent years (Abbiss and Laursen 2008; Ansley et al. 2004; Foster et al. 1993; Tucker and Noakes, 2009); however, is relatively unexplored compared to research examining fatigue using task failure and time to exhaustion protocols (Gonzalez-Alonso, 1999; Hunter et al. 2004). It has been suggested that the search for the optimal pacing strategy for an event/match may have significant benefits to overall performance (Abbiss and Laursen, 2008). While numerous pacing strategies can be adopted by an athlete, pacing strategies are often defined based on categories including, a) negative, b) ‘all-out’, c) positive, d) even, e) parabolic-shaped and f) variable (Abbiss and Laursen, 2008; St Clair Gibson et al. 2006). The ensuing section of this literature review will describe and discuss these respective pacing strategies.

Even Pacing

As shown in Figure 2.1A, an even paced strategy is present when athletes maintain constant sub-maximal exercise intensity throughout a bout of exercise (St Clair Gibson et al. 2006). Abbiss and Laursen (2008) outline that an even pacing strategy may be most beneficial to events that are > 2 min in duration, often observed in sports such as swimming, running, rowing and cycling. Thompson et al. (2003) examined even, positive, and negative pacing strategies during a 200 m breaststroke swimming event and reported even strategies resulted in lower lactate concentrations and RPE, without differences in VO₂ compared to the other two strategies. However, these strategies were externally dictated to maintain a constant %VO₂max, therefore, the effect of pacing on performance could not be determined. Even pacing strategies have been reported to be beneficial to 1 h track cycling, with Padilla et al. (2000) reporting that maintaining a steady velocity with minimal deviations in lap times improves performance. It was suggested that significant increases in velocity (above the even strategy) may be sufficient to exceed an athlete’s physiological limit (critical power) and reduce performance. Due to variable exercise intensities during team sport exercise, it is unlikely that an even
pacing strategy would be exhibited by a team sport athlete during a match; however, specific modes may exhibit even pacing strategies such as a relatively constant jogging pace throughout the match.

**Positive Pacing**

Positive pacing strategies are characterised by an athlete commencing exercise at maximal intensity, followed by a progressive decline in performance for the remainder of the exercise bout (Figure 2.1B; Abbiss and Laursen, 2008). Positive pacing strategies are often present in moderate distance events such as 100-200m swimming, 2000m rowing and 800m running, and are approximately 60 – 150 s in duration. As expected, positive pacing strategies result in increased $\dot{V}O_2$, accumulation of metabolites, and higher RPE’s at earlier stages of the exercise bout, followed by the reduction in performance for the remainder of the bout. Bishop et al. (2002) showed a positive pacing strategy adopted during a 2 min kayaking trial improved performance compared to an even pacing strategy. Further, De Koning et al. (1999) reported an all-out strategy was optimal for 1000 m track cycling event, while an all-out followed by an even power output is optimal for a 4000 m event. Positive pacing strategies have also been shown in endurance protocols, and progressive declines in exercise intensity have been suggested to be the result of peripheral changes such as glycogen depletion (Johnson et al. 2006), altered substrate utilisation (Havermann et al. 2005), neuromuscular fatigue (Lepers et al. 2002) and/or psychological stress (Marcora et al. 2009).
Figure 2.1 General profiles of A) Even Pacing Strategy, B) Positive Pacing Strategy, C) Negative Pacing Strategy, D) ‘All-out’ Pacing Strategy exhibited during self-paced exercise.
Negative Pacing

A negative pacing strategy occurs when an athlete progressively increases exercise intensity over the duration of the protocol (Abbiss and Laursen, 2008). As shown in Figure 2.1C, this strategy is described as a ‘slow start’ by St Clair Gibson (2006) and shows that the athlete commences exercise at sub-maximal values and increases pace with duration. A negative pacing strategy is most apparent during middle-distance events where the final increase in intensity is a result of increased motor unit recruitment (Kay et al. 1999; Tucker et al. 2004) and use of anaerobic energy reserves (Foster et al. 2004; Green 1997). For example, Mattern et al. (2001) examined the effect of a negative pacing strategy in which power output commenced at 15% below mean power output, but resulted in a significantly improved overall performance during a 20 km cycling time trial. Accordingly, the nature of a negative pacing strategy may suggest that it is a strategy that is centrally mediated in which athletes are able to anticipate forthcoming workloads and thus complete the exercise bout at maximal or near-maximal efforts (Marino, 2004).

‘All-out’ Pacing

An ‘all-out’ pacing strategy is most prevalent during short duration exercise where acceleration is the predominate portion of the event, such as a 100 m sprint (Abbiss and Laursen, 2008). Characteristics of an ‘all-out’ pacing strategy include a rapid increase in power output/velocity within a short period of time, showing a maximal effort at the commencement of exercise. This is followed by a plateau or slight decline in power/velocity when the athlete is attempting to maintain that intensity for the remainder of the event (Figure 2.1D). As an example, Foster et al. (2003) examined energy expenditure during a 1500 m cycling trial and showed an ‘all-out’ strategy for velocity during the trial peaked within the first third of the trial with a relatively minor decline in velocity thereafter. It has been suggested that this type of pacing strategy is representative of energy expenditure requirements for acceleration compared to energy required to maintain a given intensity, with energy best distributed at the start of short duration events (Abbiss and Laursen, 2008).
Parabolic-Shaped Pacing

A ‘parabolic-shaped’ or ‘variable’ (as described by St Clair Gibson et al. 2006) pacing strategy is when an athlete produces a maximal effort during the initial efforts, power output is then down-regulated during the mid-section of the protocol, and then increases again toward the end of the given exercise bout (Abbiss and Laursen, 2008; St Clair Gibson et al. 2006; Tucker and Noakes, 2009). Recent development of more accurate and reliable split times, improved frequency of power output sampling, and mathematical interpretation of results has allowed for closer examination of this parabolic-shaped pacing strategy. Generally, parabolic-shaped pacing can be further segmented to 3 curves, the ‘U-shaped’, ‘J-shaped’, and ‘reverse J-shaped’ (Figure 2.2). Recent cycling time trial studies have exhibited these distinct pacing strategies. Kay et al. (2001) examined 1 h cycling time trial, and while adjustment of power output was not reported, power output during the 6 sprints that were interspersed at 10 min intervals throughout the protocol exhibited a ‘U-shaped’ curve. Specifically, power output during maximal sprints exhibited a progressive decline in sprint performance following the initial sprint, although power output during the final sprint was at values similar to the first sprint. Additionally, Tucker et al. (2004) showed a ‘J-shaped’ pacing strategy, in which the final power output was higher than at the start of exercise during a cycling time trial in both warm and cool conditions. The mechanisms for these parabolic pacing strategies have primarily been explained by centrally mediated mechanisms where an athlete regulates intensity and muscle recruitment in response to their physiological load and for exercise to be completed with a superseded increase in work at the end (end-spurt) (Noakes, 2011).
Chapter 2 – Review of Literature

Accordingly, many of the previous studies examining pacing strategies have externally-imposed the respective strategies and examined the physiological and perceptual responses, which are not indicative of ‘real world’ settings. On the contrary, there are numerous studies that allow the self-selection of pacing strategies in response to physiological and perceptual loads, primarily with the use of laboratory-based cycling time trials (Kay et al. 1999; Tucker et al. 2004, 2006). However, few studies have attempted to relate pacing strategies to performance outcomes in prolonged intermittent-sprint exercise or applied the current collection of central and peripherally derived models used to explain fatigue on pacing within such exercise (Castle et al. 2006; Skein and Duffield, 2010). These mechanisms and models that attempt to explain fatigue and pacing will be outlined later in this section of the literature review.

**Measurement of Pacing Strategies**

While the previous section described the common pacing strategies observed in the literature, the assessment of pacing is often varied between studies. As cycling time trials are the most common self-paced exercise mode, assessment of pacing strategies have generally involved a preview of power output at percentages of distance covered or time lapsed (Atkinson and Brunskill, 2000). For example,
Duffield et al. (2010) reported mean power output every minute during a 40 min cycling time trial, while physiological variables were measured at 5 min intervals. Additionally, Tucker et al. (2006) have assessed alterations in power output during self-paced exercise during a 20 km cycling time trial, sampling power output every 200 m. Furthermore, Clark et al. (2007) assessed pacing strategies via changes in power output alongside physiological variables every 30 s split of a 5 km cycling time trial. Similarly, power output was measured every 100 m and 200 m of a 1500 m cycling trial by Hettinga et al. (2007) and Foster et al. (2003), respectively. However, as Tucker et al. (2006) highlight, the use of vastly sporadic data collection points during self-paced exercise makes it difficult to measure pacing strategies and examine the mechanisms associated with exercise regulation, such as feed-forward and feedback control that may underpin the observed pacing strategy.

The measurement of exercise intensity, and more specifically, pacing strategies during team sports can be more difficult than prolonged continuous events in set distance events or as an individual. Methods to assess performance and pacing strategies during team sports have included total distance covered, with more specific segmentation into distance covered during high-intensity activities (HIA), moderate-intensity activities (MIA), and low-intensity activities (LIA) (Hill-Haas et al. 2009; Mohr et al. 2003). TMA has also included the segmentation of these activities into frequency, mean and total duration, and % total time of each exercise mode within a match (Mohr et al. 2003). The examination of overall distance covered during a match and during each half can provide information regarding the cumulative fatigue present during the match. Further, the inspection of specific, individual exercise modes within a match has been shown to provide insight into which exercise intensities primarily contribute to the changes in overall match performance (Krustrup et al. 2005; Rampinini et al. 2007). For example, several field-based studies have shown a reduction in total distance covered, however LIA has been reduced while HIA performance is maintained during an AFL match in the heat (Duffield et al. 2009). Further evidence of exercise regulation during team sports has been supported
Chapter 2 – Review of Literature

by reductions in distance covered during the second half of team sport matches (Coutts et al. 2010; Rampinini et al. 2007).

Given the difficulties measuring pacing strategies in match-based scenarios, examination of pacing during repeated-sprint efforts within a laboratory setting can be segmented into maximal sprint efforts with passive recovery or sprint efforts that are interspersed with (active) sub-maximal efforts. However, the examination of these repeat-sprint protocols in this area is rather limited. Billaut et al. (2011) examined prior knowledge on sprint number on exercise performance and recorded peak power output, mechanical work, and sum of intergrated electromyography (iEMG) for all 10 sprints completed. Similarly, Mendez-Villaanueva et al. (2007) reported peak power, total work, and EMG data during each of 10 x 6 s sprints. However, these repeated-sprint protocols only assess peak power during a relatively short period of time, thus do not examine pacing strategies during intermittent-sprint exercise over a prolonged period or assess the inter-mode effect of regulating high and low intensity efforts. Therefore, most previously used intermittent-sprint protocols do not allow the examination of pacing strategies or the interplay of various exercise intensities on overall performance, as noted during competitive matches.

The Loughborough Intermittent Shuttle Test (LIST) was one the first laboratory-based running intermittent-sprint tests designed to replicate the demands of soccer (Nicholas et al. 2000). Despite successful and popular research use of the protocol, the LIST is externally regulated (other than maximal sprints) and therefore reduces the ability to measure self-paced efforts. Duffield and Marino (2007) used an alternative intermittent-sprint protocol that included over-the-ground running in which all exercise modes were self-paced, allowing the implementation and regulation of pacing strategies including sprint times and distance covered during hard running, jogging and walking efforts (Duffield and Marino, 2007). Further, Skein and Duffield (2010) have segmented distance covered during the sub-maximal efforts during each 10 min phase for further examination of the pacing
strategies developed during the 50 min intermittent-sprint protocol. Accordingly, it could be viewed that a laboratory-based intermittent-sprint exercise protocol for team sport athletes should replicate the physical, physiological, and perceptual demands of a match outlined in the previous section, in conjunction with the inclusion of self-paced exercise to allow the assessment of pacing strategies. The assessment of pacing strategies and the corresponding physiological and perceptual loads can allow the examination of the mechanisms related to self-paced intermittent-sprint exercise regulation.

**Models to Explain Pacing and Regulation of Fatigue**

The considerable variation in exercise modes and intensities during team sport exercise provides an interesting environment for the assessment of the mechanisms responsible for fatigue. Previous literature has shown that peripheral perturbations are evident during team sports such as muscle glycogen depletion, accumulation of various metabolites including lactate and hydrogen ions, and reductions in blood and muscle pH (Krustrup et al. 2006). Factors such as dehydration and the associated changes to the cardiovascular and thermoregulatory responses are also commonly experienced during intermittent-sprint exercise have also been linked to performance declines (McGregor et al. 1999). While the volume of literature examining the regulation of fatigue and pacing during intermittent-sprint exercise is relatively small, there is growing support of the notion that exercise is regulated throughout all self-paced exercise modes in an anticipation of the impending loads (Hampson et al. 2001; Marino, 2004; Tucker and Noakes, 2009). Accordingly, this section will investigate the proposed models attempting to explain fatigue and exercise regulation to gain perspective of the mechanisms responsible for regulating performance.

**Cardiovascular/Anaerobic Model**

As team sport athletes are exposed to increased cardiovascular, thermoregulatory, and metabolic stress through prolonged bouts of high-intensity exercise, the perturbations associated with the Cardiovascular/Anaerobic model may have implications on intermittent-sprint performance (Shirreffs
et al. 2005). Some of the earliest research recognising the cardiovascular system as a limiting factor includes work by Hill and colleagues (1924, 1927). The key concept of Hill’s theory was that there is an upper limit of cardiac output that cannot be exceeded; resulting in a limited amount of blood that can be pumped to the muscles. When exercise is maximal, the oxygen demand exceeds supply from cardiac output and the muscles are forced to work anaerobically. As shown in Figure 2.3, when cardiac output is limited as a result of a reduction in heart rate and/or stroke volume, there is an impaired delivery and utilisation of oxygen and the ability to remove lactate and hydrogen ions are compromised (Abbiss and Laursen, 2005). In support of the Cardiovascular/Anaerobic model, Gonzalez-Alonso and Calbet (2003) examined cardiovascular responses to cycling to exhaustion with high or normal pre-exercise skin and core temperatures in healthy males. Results indicated that exercise is terminated when declines in cardiac output, mean arterial pressure (MAP), muscle blood flow, oxygen delivery and oxygen uptake are present. In relation to team sport athletes, increased physiological loads including workloads in excess of ~80% $\dot{V}O_{2\text{max}}$ and presence of hypohydration due to excessive sweat loss (Shirreffs et al. 2005) may contribute to compromised cardiovascular load and performance declines (McGregor et al. 1999).
The Cardiovascular/Aerobic model is one of the most accepted models in Exercise Physiology to explain fatigue and performance declines during exercise (Coyle, 1999). Moreover, this model has been further supported by training studies that have shown improvements in exercise performance when oxygen transport and utilisation are increased (Blomqvist and Saltin, 1983). Significant increases in cardiac output have been reported following endurance training due to increased stroke and blood volumes (Convertino, 1991; Sawka et al. 2000). Further, hemodynamic variables in the blood have also been reported to change as a result of aerobic training, with increased circulatory haemoglobin and improved performance through enhanced oxygen carrying capacity (Coyle, 1999).

Endurance training has also shown to improve intracellular oxygen utilisation (Rud et al. 2011) with greater size and volume of mitochondria, increased aerobic enzyme activity and capillarisation (Baar, 2006). The higher oxygen consumption capacities exhibited by elite players’ compared to sub-elite players may in part allow for the increased distance covered specifically during the high intensity efforts of a team sport match (Helgerud et al. 2001). Accordingly, improvements in oxygen utilisation
have been related to performance improvements via the ability to exercise at higher intensities before attaining the anaerobic threshold when fatigue may become apparent; possibly also explaining performance outcomes present during team sport exercise (Helgurud et al. 2001).

As outlined within this model, when cardiac output is compromised and an insufficient volume of blood (and thus oxygen) is present within the active muscle, an anaerobic environment results leading to accelerated metabolite accumulation (Green, 1997). Accordingly, the metabolic processes in the muscle aim to maintain the availability of ATP for myosin cross-bridge cycling and the by-products of muscular work stimulate a cascade of metabolic processes to re-establish ATP levels from adenosine diphosphate (ADP) using inorganic phosphates (Green, 1997). During high-intensity exercise there is an accumulation of intracellular metabolites that are suggested to compromise the contractile unit and excitation-contraction coupling, leading to an interruption of continued muscular contraction (Green, 1997). The primary metabolites highlighted as being fatigue causing agents relate to the accumulation of hydrogen ions and inorganic phosphates. The excess accumulation of hydrogen ions is associated with a decrease in intramuscular pH, which has been linked to decreased cell excitability (Orchardson, 1937), increased calcium requirement for similar tension requirements (in skinned fibre studies) (Fabiato and Fabiato, 1978; Robertson and Kerrick, 1976), and decreases in sarcoplasmic reticulum release and reuptake of calcium (MacLaren et al. 1989). Furthermore, rate limiting enzymes associated with glycolysis, particularly phosphofructokinase (PFK), are inhibited by a decrease in pH and reduce the rate of ATP synthesis (Green, 1997). Each of these respective factors associated with increased hydrogen ion accumulation and reductions in pH have been linked to a reduced capacity to produce contractile force and consequently declines in exercise performance (Green, 1997). Accordingly, previous research has shown significant reductions in muscle ATP, PCr, glycogen concentrations, pH, and sarcoplasmic reticulum calcium reuptake, while muscle lactate and IMP is significantly increased following 3 x 30 s maximal sprints (Hargreaves et al. 1998). These findings are supported by field-based research, with Krstrup et al. (2006) highlighting post-exercise
reductions in ATP, PCr and increased muscle lactate in relation to pre-exercise values. Whilst not significantly different from pre-exercise values, trends were also evident for reductions in post-exercise pH and increased intramuscular hydrogen ions. Collectively, accumulation of such metabolites are present during repeated bouts of high-intensity exercise and may be detrimental to team sport performance, particularly if insufficient recovery period durations are present between maximal sprint efforts. Accordingly, an understanding of the peripheral perturbations associated with the Cardiovascular/Aerobic model may assist further elucidate the mechanisms responsible for exercise regulation during intermittent-sprint exercise.

Energy Supply/Energy Depletion Model

The Energy Supply model poses that the development of fatigue and decline in performance are a direct result of inadequate energy supply (ATP) via respective metabolic pathways (Abbiss and Laursen, 2005; Noakes, 2000). More specifically, the inability to supply ATP at sufficient rates can occur during the phosphagen, anaerobic glycolysis, aerobic glycolysis, and aerobic lipolysis pathways, although is dependent on exercise intensity (Noakes, 2000). The effect of energy supply on exercise performance for team sport athletes is important as repeat-sprint exercise has been shown to be taxing on these energy pathways (Glaister, 2005). Energy provision during sprint performance is obtained through the breakdown of ATP (Glaister, 2005) while PCr is required for the fast resynthesis of ATP. Accordingly, reductions in ATP and PCr may be detrimental to high-intensity exercise performance particularly if inadequate recovery time is not present between successive high-intensity efforts (Bishop and Spencer, 2004). Furthermore, Glasiter (2005) has reported that PCr degradation accounts for approximately 50% of the total ATP provision during single, short maximal sprints; although during repeated bouts of maximal sprints, PCr degradation is predominately determined by rate of resynthesis and recovery periods following exercise. Accordingly, energy provision is important for all exercise modes and durations; however, the inability to supply ATP during intermittent bouts of maximal sprints may compromise performance.
The Energy Depletion model is related to the Energy Supply model, although differing in regards to exercise performance being limited by the depletion of fuel substrates, namely, liver and muscle glycogen (Abbiss and Laursen, 2005). Some of the first research to directly examine muscle glycogen depletion on exercise performance using the muscle biopsy technique was Bergström and colleagues in 1967. During a series of studies Bergström and Hultman (1967) highlighted that (i) exercise capacity decreases when glycogen stores are depleted, (ii) glycogen concentration in resting muscle is unchanged when exercised muscles have been depleted with exercise, (iii) if glucose is infused during exercise, glycogen consumption is significantly lower compared to no glucose infusion and (iv) liver glucose production increases towards latter stages of continuous exercise but is relatively small compared to total CHO metabolism (Bergström and Hultman, 1967). Following this initial work, there has been a plethora of research examining the effects of muscle glycogen content on exercise performance (Burke et al. 2000; Hawley et al. 1997a; Mosley et al. 2003; Williams et al. 1992; Winnick et al. 2005). For example, increased pre-exercise CHO ingestion has been shown to have direct effects on muscle glycogen availability, increase muscle glycogen use (utilisation) and rate of glucose uptake (Steensberg et al. 2002). Furthermore, the ingestion of post-exercise CHO ingestion supports a rapid resynthesis of glycogen stores (Burke et al. 1995, 2004; Ivy et al. 1988) and improves ensuing exercise performance (Nielsen et al. 2011). These findings are of particular interest to team sport athletes who experience performance declines due to muscle glycogen depletion during matches (Balsom et al. 1999b; Krstrup et al. 2005). Notwithstanding, energy supply (muscle glycogen) has been shown to be important for exercise of prolonged durations and/or high intensities that rely on glycogen utilisation and CHO oxidation. Accordingly, the manipulation of pre-exercise CHO ingestion and muscle glycogen content (high vs low) creates a method of assessing the direct effects of peripheral perturbations on self-paced intermittent-sprint performance. Furthermore, the high demands associated with intermittent-sprint exercise may also result in compromised energy supply with the potential to reduce consecutive bouts of high-intensity exercise. Therefore, team sport athletes may have a specific interest in understanding the Energy Supply/Depletion model as
peripheral factors discussed in these models may have detrimental effects on specific exercise modes of a match and/or overall exercise performance.

**Teleoanticipatory Model**

The physiological changes associated with team sport exercise may have direct consequences on match performance (Bangsbo et al. 1992). The aforementioned peripherally-based models show that altered physical states as a result of repeated high-intensity exercise can have a direct effect on performance (Glaister et al. 2005; Green, 1997). However, centrally mediated models suggest that exercise intensities are manipulated in order to regulate these physiological states before they become catastrophic (Noakes et al. 2000). The notion of an anticipatory regulation of work rate was originally suggested by Ulmer (1996) who stated that that exercise is controlled by a governor that is able to estimate the optimal work rate to complete an exercise bout. Accordingly, this system anticipates the demands of the preceding task in response to the finishing point or goal, hence the name ‘teleoanticipatory’, (teleo= final or last). It is suggested that the primary variable that is regulated during exercise is metabolic rate, with feedback regarding the actual metabolic rate compared with the preferred rate. However, in order for this teleoanticipation of metabolic demands to be completed, a template is required in order to determine the preferred metabolic rate (Ament and Verereke, 2009). Ulmer (1996) further suggests that feedback may be provided from other somatosensory sources such as the muscles and organs which are also taken into account during the projection of imposed loads associated with a given pacing strategy.

The theory of teleoanticipation has been supported by Marino et al. (2004), who examined self-paced exercise performance in African and Caucasian runners with starting core temperatures of 38.4°C. At the start of the self-paced run, the Caucasian runners immediately down-regulated their running pace compared to their African counterparts. As both groups finished with similar rectal temperatures, it was concluded that the Caucasian runners reduced pacing strategies (intensity) in anticipation of the
forthcoming imposed loads to maintain a similar rate of heat storage as the African runners (Marino et al. 2004). As shown by Marino et al. (2004), there is an anticipatory regulation of thermal stress and these findings may be extrapolated to team sports. Duffield and Marino (2007) reported a down regulation of exercise intensities (hard running) during self-paced intermittent-sprint exercise, with the completion of exercise at similar core and skin temperatures. In relation to team sports, it may be suggested that exercise intensities during various modes within the match (i.e. sprinting, jogging) are anticipatorily regulated based on feedback from the periphery and a feed-forward template derived from prior matches and training sessions in order to complete the match but avoid substantial physiologic deviations from homeostasis.

Figure 2.4 Overview of the Teleoanticipation Model proposed by Ulmer (1996). (Lambert et al. (2005)

It has been suggested that this anticipatory template is formed from previous experiences (i.e. training and matches for team sports athletes) or is an ability/characteristic of the individual to predict forthcoming physical loads imposed on the body during exercise (Hampson et al. 2001; Ulmer, 1996). Hampson et al. (2001) further elucidates that the sub-conscious setting of exercise intensities is based
on knowledge regarding the biomechanical and metabolic stress associated with the given exercise bout from previous experiences. Systematically, the feed-forward motor drive is initiated and efferent commands (motor output) are sent to the periphery (such as the skeletal muscle), during which alterations in pacing strategies via changes in skeletal muscle recruitment occur. As shown in Figure 2.4, afferent feedback from the periphery regarding physiological state associated with the present pacing strategy is interpreted by the CNS and compared to the preferred metabolic template. In response to the difference between the present and preferred physiological and metabolic states, appropriate changes are again made to neural drive and thus pacing strategies (Figure 2.4). This feed-forward/feedback loop will be discussed in greater detail in the Central Governor Model section of this literature review. In summary, the Teleoanticipatory model suggests that pacing is the regulation of physiological loads, therefore the application of pre-exercise physiological and perceptual states may be an effective method to assess the potential regulation of exercise intensities. In respect to team sport athletes, the application of ecologically valid circumstances such as changes in thermal stress and muscle glycogen content can provide insight into the anticipatory regulation of various exercise modes in order to protect the body from cellular catastrophe.

Central Governor Model

In more recent decades, the Central Governor Model has been developed by Professor Tim Noakes and colleagues and is derived from the original work by A.V. Hill and colleagues (1924). Hill et al. (1924) proposed that the development of anaerobiosis in the active musculature is responsible for the reduction in performance during maximal exercise performance. Although Hill et al. (1924) also suggested that some mechanism located either in the periphery or CNS, restricted cardiac output when arterial oxygen saturation levels were reduced. Noakes and colleagues have continued such investigations and discussions about the concept of a ‘central governor’ with suggestions that the CNS regulates all physiological systems (rather than just the cardiovascular system). Accordingly, the regulation of such physiological systems is through the manipulation of exercise intensities (pacing
strategies) via changes in neural drive to the active musculature (neuromuscular recruitment) (Tucker and Noakes, 2009; Weir et al. 2006). As outlined in Figure 2.5 (Noakes, 2011) there are numerous centrally-mediated factors that are encountered by athletes before and during exercise that can influence performance and pacing strategies. Many of these specific factors may be experienced by team sport athletes, such as prior match experiences, sleep deprivation, and glucose ingestion or may alter the afferent sensory feedback, including heat, dehydration and glycogen stores (Krystrup et al. 2006; Shirreffs et al. 2010). Therefore these respective performance modifiers are pertinent to understanding the central regulation of pacing strategies in team sport athletes.

Figure 2.5 Overview of the Central Governor Model during which centrally-mediated factors from the periphery and external environment (feedback) are interpreted by the brain for an appropriate feed-forward regulation of neural drive and pacing strategies (Noakes, 2011).
Noakes et al. (2011) suggests several observations that are evident during exercise cannot be explained by the previously suggested peripheral models that do not incorporate the CNS in explaining exercise regulation. These observations include (i) different pacing strategies for different exercise durations, (ii) the presence of an end-spurt during self-paced exercise, (iii) development of fatigue despite homeostasis being present, (iv) sub-maximal muscle recruitment at $\dot{V}O_{2\text{max}}$, (v) interventions acting on the CNS that alter pacing strategies and exercise performance, and (vi) RPE is in response to exercise duration rather than intensity (Noakes, 2011). For example, in regards to the cardiovascular model, the first organ to be affected by reductions in cardiac output and thus induce anaerobic metabolism would be the cardiac muscle, however, no study to date has reported hypoxia and ischemia in the heart during maximal exercise (Noakes, 2000). According to the Central Governor Model, the findings that cannot be explained by peripherally-mediated mechanisms can be done so by centrally-mediated mechanisms that monitor and regulate motor drive in order to minimise disruptions from homeostasis and events such as ischemia and hyperthermia occurring (Noakes 2000, 2005, 2011).

The Central Governor Model extends from Ulmer’s (1996) previously explained Teleoanticipatory Model in that sub-conscious feed-forward control mechanisms regulate exercise. The model proposes that afferent feedback from the periphery (such as the heart, muscle, blood markers) is continuously provided to the brain, which is interpreted and an appropriate feed-forward central drive (efferent motor drive) determines the extend of skeletal muscle recruitment (Noakes, 2011; St Clair Gibson and Noakes 2004). The model also allows afferent feedback to be recognised prior to exercise in order to determine the initial pace set. These factors include prior experience, pre-exercise physiological state, sleep state, expected exercise duration/distance and motivation (Figure 2.5). Afferent feedback regarding factors outlined in Figure 2.5 such as fuel substrate availability, rate of heat accumulation, hydration status, cerebral oxygenation levels, continue to be interpreted by the CNS with the appropriate neural drive sent to the active musculature (Noakes, 2011).
According to the Central Governor Model, the purpose of the feed-forward/feedback loop is to monitor and control physiological responses during exercise and ultimately, protect the body from cellular damage (Noakes, 2005; Marino, 2004). Furthermore, protective mechanisms are in response to the concurrent physiological perturbations, but also in an anticipatory regulation of exercise-induced stresses that are expected to be placed on the body (Lambert et al. 2005; Marino, 2004; Noakes et al. 2005). For example, self-paced exercise protocols have shown a reduction in power output during exercise in hot environment conditions compared to cool conditions (Abbiss et al. 2010) and differences in pacing strategies have been evident prior to significant differences in physiological variables such as core temperature (Tucker et al. 2006). Additionally, studies examining hyperthermia on exercise capacity have shown exercise exhaustion coincides with attainment of the critically-limiting temperature (Galloway and Maughan, 1997; Gonzalez-Alonso et al. 1999). Furthermore, when subjects are required to complete self-paced exercise, interestingly, subjects do not obtain or supersede this critical limiting temperature, but rather a reduction in pacing strategies are observed well before attaining a hyperthermic state (Marino, 2004; Nybo, 2008; Tucker et al. 2004). As a specific example, these findings suggest that during exercise in the heat, exercise is severally compromised (terminated) at critically-limiting temperatures, and during self-paced exercise, athletes protectively down-regulate exercise intensities to avoid reaching this temperature.

The Central Governor Model has provided insight into the complex regulation of exercise intensities via the integration of various physiological systems which are controlled by the brain. The notion of exercise regulation rather than a peripherally-derived exercise terminator is of importance to team sport athletes in which exercise is self-paced and match cessation is governed by the ‘final whistle’ rather than physiological load. Furthermore, the various exercise modes and intensities during a match cause high physiological strain on the body and therefore induce disruptions from homeostasis. Therefore, knowledge of whether cumulative and transient fatigue developed during a match is...
peripherally derived or centrally-mediated will be important in the development of countermeasures to improve pacing strategies and minimise fatigue.

**Psychological-Motivational Model**

Following the development of Noakes and colleagues’ Central Governor Model where exercise is believed to be sub-consciously regulated in response to afferent feedback from the periphery, Marcora (2008) has proposed an alternative model in which the conscious brain controls exercise regulation. This Psychological-Motivational Model suggests that during exercise power output and completion of that bout of exercise is a voluntary decision. More specifically, during incremental exercise to exhaustion, an athlete terminates exercise when the perception of effort is greater than the level that they are willing to exert themselves (Marcora, 2008). In relation to self-paced exercise, it would be thought that athletes consciously regulate pacing strategies throughout the protocol rather than pacing being a sub-conscious regulation of physiological perturbations (Noakes, 2000; St Clair Gibson and Noakes, 2005). More specifically, in the context of this model, it would be thought that the exercise frequency, intensity, and duration of the exercise modes present during team sports are consciously controlled by the athlete in relation to their willingness and motivation to exert themselves based on the context of match technical and tactical demands.

![Figure 2.6 The Psychological-Motivation Model as proposed by Marcora (2008, 2009).](image-url)
The theory of conscious exercise regulation was examined by Marcora and colleagues (2009) with the use of mental fatigue rather than conventional methods of manipulating physiological variables. Subjects cycled to exhaustion following 90 min of a cognitive demanding task (mental fatigue), or watching emotionally neutral documentaries (control). Mental fatigue significantly reduced time to exhaustion compared to the control condition with a significantly higher RPE. Therefore, the authors concluded that mental fatigue reduces exercise capacity due to higher perception of effort. Obviously for methodological constraints the subjects were not able to be blinded to the conditions; therefore, it may be likely that there may have been some placebo effect of the mental fatigue condition with the expectation of a greater decrease in performance (Beedie et al. 2009). Notwithstanding, this study does suggest that when physiological differences are not present, mental fatigue can reduce exercise performance. In regards to team sport exercise, which requires high cognitive demands and decision-making processes, cognitive perception of fatigue may be present within a match and thus affect performance in conjunction with or independent to physiological perturbations (Smith et al. 2011). Furthermore, the alteration of perception and mood states without substantial alterations in physiological perturbations may provide an interesting method of assessing the effects of conscious vs sub-conscious regulation of exercise intensities.

Summary of Models Explaining Fatigue

In summary, the models that attempt to elucidate the mechanisms responsible for fatigue and exercise regulation have been primarily categorised as two distinct origins pertaining to peripheral and central, respectively. The peripheral models, which include the Cardiovascular/Anaerobic Model and Energy Supply/Depletion Model theorise that declines in exercise performance are associated with catastrophic events at the periphery. More specifically, these deleterious changes are primarily evident in the cardiovascular system or intramuscular changes such as metabolite accumulation or substrate depletion. On the contrary, centrally-mediated models suggest that the CNS is able to monitor and regulate exercise intensities in order to prevent these catastrophic events from occurring. Accordingly,
feedback from the periphery is interpreted by the CNS and sends a corresponding efferent motor drive to the active musculature to monitor muscle recruitment and pacing strategies. A third model proposed to explain fatigue that has gained notoriety includes the conscious regulation of exercise intensity based on motivation and perception of effort. While all respective models have credence, to date no one model exclusively explains the development of fatigue, possibly in-part due to task dependency in which different task demands may result in different mechanisms of fatigue (Weir et al. 2006). In relation to team-sports, few studies have attempted to apply such models to explain self-paced intermittent-sprint activity. Accordingly, the present thesis will use a series of pre-exercise interventions to alter physiological and perceptual states to assess the effects on pacing strategies during self-paced, intermittent-sprint exercise and elucidate the mechanisms responsible for pacing and the development of fatigue.

**Influential Factors Regulating Pacing Strategies**

*Effect of Task Dependency on Pacing Strategies*

In recent published research there has been a noticeable increase in the use of the term ‘task dependency’ in exercise science, and more specifically, during investigations of mechanistic agents of fatigue (Abbiss and Laursen, 2008; Enoka, 1995; Hunter et al. 2004). Task dependency describes the complex relationship between speed, duration, and intensity of different exercise bouts (St Clair Gibson et al. 2001). Enoka and Stuart (1992) highlight that fatigue is not the result of a single system, but rather different exercise protocols may result in different mechanisms being responsible for the fatigue within that given exercise bout. Therefore, as different tasks provoke different physiological and perceptual responses, the concomitant pacing strategies adopted to regulate such changes may also be affected. More specifically, physiological responses that may be dictated by the specific exercise task include substrate utilisation (Gollnick et al. 1974), metabolite accumulation, energy system contribution and cardiovascular, thermoregulatory and perceptual responses (Gastin et al. 2001; Gollnick et al. 1974; Kondo et al. 1998; Montain et al. 1995; Van Loon et al. 2001). Therefore,
the effect of the aforementioned physiological changes (induced by different pre-exercise circumstances) on the manipulation of pacing strategies may be dependent upon the physical, physiological and psychological demands of the particular task.

The Effects of Physiological and Perceptual States on Pacing Strategies

Changes to an athlete’s physiological state can have a significant effect on pacing strategies during self-paced exercise (Castle et al. 2010; Duffield and Marino, 2007; Marino et al. 2010). These physiological variables may include substrate usage, recovery durations for ATP resynthesis, and cardiovascular and thermoregulatory responses. For example, muscle glycogen utilisation and following alterations in pre-exercise muscle glycogen concentration will differ during short-duration, high-intensity exercise, which is primarily CHO reliant, compared to prolonged, sub-maximal exercise (Coyle et al. 1986). Therefore, examination of pacing strategies during intermittent-sprint exercise in team sport athletes should incorporate the physical, physiological and perceptual demands of team sports for valid assessments of the likely mechanisms related to fatigue.

Unfortunately, the background literature examining intermittent-sprint exercise regulation in response to physiological perturbations is rather limited. Duffield and Marino (2007) examined pre-cooling interventions on self-paced intermittent-sprint exercise and reported an increase in hard running distance covered following submersion in an ice bath and warm-up with an ice vest compared to no cooling (control). Similarly, Clarke et al. (2010) examined pre-exercise cooling with CHO ingestion during a soccer-specific intermittent-sprint protocol compared to CHO ingestion alone, a placebo CHO ingestion and pre-cooling with a CHO placebo and reported a higher intensity pacing strategy was present when Tcore, Tmus and Tskin were significantly lower. Minett et al. (2011) also highlighted that whole-body pre-cooling was effective in reducing pre-exercise thermal strain, resulting in an increased maintenance of sprint times and distance covered during the hard running bouts of the intermittent-sprint protocol. The inclusion of additional physical stressors such as contacts and
collisions can also invoke greater physiological stress including muscle damage (creatine kinase), which can have negative consequences on self-paced intermittent-sprint performance (Pointon et al. 2011). In conjunction with these physiological responses, intermittent-sprint performance has been shown to be altered after mental fatigue without changes at the periphery. Smith et al. (2011) examined performance during a 45 min intermittent-sprint protocol on a non-motorised treadmill following either a mentally demanding task or an emotionally neutral documentary. Results indicate that mental fatigue reduces low-intensity activity velocity, with minimal effect on high-intensity activities or sprint velocity. Collectively, research indicates that changes to external environment or internal stress that deviate from homeostasis or mental fatigue can induce a change in pacing strategies during continuous and intermittent-sprint exercise (Tucker and Noakes, 2009).

The Effects of Knowledge and Deception on Pacing Strategies

Knowledge can have a powerful influence on exercise performance. Previous research has identified that the knowledge of an impending exercise task, such as time to completion, can influence the pacing strategy set during continuous (Clark et al. 2000) and repeat-sprint (Billaut et al. 2011) protocols. Examining the influence of knowledge on exercise performance has primarily been achieved using deception methodology, in which subjects are blinded and/or deceived about when the given exercise task will be completed. For example, Paterson and Marino (2004) examined the effect of prior distance deception on 30 km cycling time trial performance. All subjects completed a 30 km time trial, and were then segmented into three groups and were told they were completing 30 km despite actually completing either (i) 36 km, (ii) 30 km, or (iii) 24 km. Interestingly, when subject completed a third trial of a known 30 km ride, time to complete was increased when subjects previously rode 24 km, while the 30 km trial time was shorter after the 36 km ride and control times remained unchanged. Moreover, there were no differences in RPE between the first and final 30 km rides. These findings strongly support Ulmer’s (1996) Teleoanticipatory Model, as the subject’s exercise ‘template’ for a 30 km time trial based on previous experience was compromised, subsequent
performance was altered accordingly. Swart et al. (2009) acknowledges the importance of RPE on pacing strategies during cycling time trial performance; however, suggests it is dependent upon the information provided about the duration until exercise completion. Results from this study indicated that when feedback regarding time to completion is withheld from an athlete, more conservative increases in RPE and pacing strategies were implemented.

Similar to continuous protocols, the use of deception during repeated sprint protocols have also shown effects on performance and pacing. Billaut et al. (2011) reported that deceiving subjects about the number of sprints they are required to complete altered pacing strategies during repeated-sprint efforts. Subjects completed (i) 10 x 6 s sprints (with 24 s recovery) followed by 10 x 6 s sprints (control), (ii) were told to complete 5 sprints, but were then told to complete the remaining 5 x 6 s sprints (deception), (iii) were not told how many sprints they would be performing but were stopped after 10 sprints (unknown). Results highlight that more work was completed during the first 5 sprints of the deception trial and less work was completed during the unknown trial, suggesting an anticipatory regulation of work that can be dependent upon the knowledge of exercise duration. Further, Billaut et al. (2011) reported EMG values followed similar responses to the power and work completed during the maximal sprints between conditions, while RPE responses were similar between conditions. Therefore, similar to prolonged continuous exercise protocols, it appears that repeated sprints are paced in anticipation of the number of sprints to that are expected to be completed.

Blinding of information or deception regarding ergogenic aids and the concomitant physiological response have also shown that an athlete’s knowledge of the internal or external demands can influence pacing strategies adopted during exercise. Accordingly, Clark et al. (2000) recruited 43 subjects to complete a 40 km cycling time trial on two separate occasions. All subjects completed trial 1 with the same conditions (baseline). During trial 2, subjects were divided into two groups, either consuming i) 16ml kg\(^{-1}\) of 7.6% CHO solution during the trial, or ii) the same volume of a placebo.
Within these respective groups, subjects were further sub-divided into (i) told they were ingesting CHO, (ii) told they were ingesting a placebo, and (iii) told nothing. This intricate study design further highlighted that the knowledge provided to an athlete regarding CHO ingestion is important, as no knowledge increased pacing variability (compared to baseline); and even when not in a physiologically advantageous state, a placebo effect had some performance improvement. In support of these findings, Skein and Duffield (2010) reported (non-significant) trends of increased distance covered when subjects were informed that they would be able to ingest water at half time of an intermittent-sprint protocol rather than receive no water. However, exercise only continued for 15 min between the information being provided and the hydration interventions being implemented which may not have been sufficient time for significant effects on pacing strategies to become evident. Finally, Castle et al. (2011) examined the effect of core temperature feedback on 30 min cycling performance in hot environmental conditions. Distance covered was reduced when subjects were cycling in the hot condition (31°C) and aware of the ambient temperature, compared to the cool condition (22°C) and cycling in the heat (31°C) yet informed it was 26°C. Furthermore, power output was higher and RPE at 3 min was lower during the deceived trial compared to knowingly cycling in the hot condition. The findings by Castle et al. (2011) in conjunction with the placebo and other deception trials create an interesting paradigm indicating that pacing strategies during self-paced exercise are a combination of both sub-conscious and conscious regulation, rather than the previously suggested sub-conscious feedback from the periphery.

**Summary**

The regulation of fatigue may be considered a continuous process throughout a given exercise bout rather than considered as the ‘end-point’ of exercise. When fatigue is examined in this way, it is likely that during self-paced exercise, pacing strategies are the physical manifestation of fatigue regulation. As such, there are several types of pacing strategies that can be implemented during a given exercise bout which lead to different physiological responses and performance outcomes. When self-paced
continuous exercise protocols are utilised, parabolic pacing strategies are primarily evident. The mechanisms that explain these pacing strategies and the regulation of fatigue are still somewhat elusive. Rather than one mechanism being exclusively responsible for performance, it may be likely that exercise regulation is a complex interaction of central, peripheral and conscious factors. This may be particularly relevant to team sport athletes, where the undulating and variable nature of intermittent-sprint exercise may suggest a dynamic interplay of mechanisms is present. Therefore, the application of various pre-exercise circumstances to alter physiological and perceptual states can provide an effective means of examining peripheral perturbations on performance and the central regulation of such perturbations. While a dynamic interplay of mechanisms responsible for intermittent-sprint performance and pacing may be present, minimal research has been completed in this type of exercise and the alteration of pre-exercise states. Accordingly, this thesis will utilise several specific pre-exercise interventions (sleep deprivation, carbohydrate intake and thermal stress) to alter physiological and perceptual load to investigate ensuing pacing strategies in intermittent-sprint exercise.

2.4 Sleep deprivation

The importance of adequate sleep before exercise has been highlighted in previous research, with acute sleep deprivation and/or disruption of a few hours potentially having negating consequences for exercise performance (Myles, 1985; Oliver et al. 2009). However, there are some discrepancies regarding the mechanisms responsible for changes in performance following sleep loss; including impaired cognition, increased energy expenditure, altered hormone regulation and negative mood states. These proposed mechanisms explaining the effects of sleep loss on exercise performance stem from previous theories attempting to determine the explicit role of sleep *per se*. One theory suggests that sleep is essential for the restorative process of the immune and endocrine systems (Horne, 1988). A second theory also encompasses the notion of a restorative process, although suggests that sleep assists in the recovery from the demands placed on the metabolic and nervous systems during the
hours of wakefulness (Vanltallie, 2006). Thirdly, it is also thought that sleep is associated with the ability to maintain cognition, specifically tasks such as short and long-term memory, motor learning, reaction times, and accuracy (Thomas et al. 2000). Notwithstanding, sleep is imperative for psychological and physiological restoration. Due to increased physical and mental demands of an athlete’s schedule, it may be likely that inadequate sleep prior to training and/or competition may dampen the athlete’s ability for complete physical and psychological restoration (from the previous day) and thus have negative implications on subsequent exercise performance.

The deprivation of sleep is common among a variety of populations and in society it is primarily associated with shift workers and military personnel (Alfred et al. 2010; Akerstedt et al. 2008, 2009). Athletes also experience acute bouts of sleep deprivation due to variety of reasons, including travel commitments across multiple time zones, experiencing environments not conducive to sleep, and anxiety or apprehension about training or competition commitments. This notion has been highlighted by Richmond et al. (2004) who examined sleep patterns during an AFL season and reported elite AFL players have significantly reduced sleep duration the night before and night of a competition match. Further, it has been reported air travel which is associated with sleep disruption, can also be deleterious to subsequent exercise performance (Bishop, 2004; Reilly and Waterhouse, 2005; Youngstedt and O’Connor, 1999). While sleep deprivation is associated with performance declines in athletes, the effect of an acute bout of sleep loss on pacing strategies during intermittent exercise is unknown and the mechanisms responsible for these performance outcomes are unclear. Further, the use of sleep deprivation represents a useful model to examine pacing strategies as a manifestation of physical regulation due the substantial effects sleep loss can have on restorative processes including physiological and perceptual states prior to exercise.
**Effects of Sleep Deprivation on Exercise Performance**

As athletes are often required to repeatedly compete and/or train at high intensities for prolonged periods of time, they may be subjected to increased stress during exercise and thus exhibit delays in recovery. If adequate sleep is not achieved following a bout of exercise, subsequent recovery may be further dampened, as sleep deprivation increases energy expenditure and metabolic demands (Berger and Phillips, 1995). While there is a growing collection of research that has shown that sleep deprivation attenuates exercise performance, the specific cause of these declines is less clear (Chen, 1991; Martin and Gaddis, 1981; Martin and Haney, 1982). It has been suggested that changes in various cardiovascular, thermoregulatory and metabolic processes following sleep deprivation may explain performance reductions (Chen, 1991; Sawka et al. 1984). Additionally, altered cognitive processes following sleep have been proposed as a possible cause, whilst changes in mood states and perception of effort have also been linked to performance declines (Angus et al. 1985; Myles, 1985). Notwithstanding, no research has examined the effect of sleep deprivation on team sports, however existing literature (as outlined in Tables 2.1 and 2.2) suggests that performance may suffer as a result of sleep deprivation.

**Self-Paced Exercise Performance**

Understanding the effect of sleep deprivation on self-paced exercise and the mechanisms responsible are important for team sport athletes, as it may provide insight into the regulation of physiological and perceptual responses during exercise. Oliver et al. (2009) examined one night sleep deprivation on 30 min fixed paced exercise at 60% \(\dot{V}O_{2\text{max}}\), followed by 30 min self-paced running. Results indicate that one night sleep loss was sufficient to reduce distance covered during the self-paced exercise protocol (SDEP: 6037 ± 759 vs CONT: 6224 ± 818m), however core and skin temperatures, cardiovascular responses and RPE were not different between conditions at rest or during exercise. Due to the similar RPE responses, despite different exercise intensities during the self-paced protocol, the authors suggested that altered pacing for similar RPE may have reduced endurance performance. In relation to
the previously proposed models, it may be that that the conscious awareness of exercise in a sleep deprived state, as evident by the presence of negative mood states, results in a down-regulation of exercise intensity during self-paced exercise. These findings are supported by Martin and Haney (1982) who examined blinded changes of treadmill grade during exercise following 30 h sleep deprivation or a normal night sleep. The study design was utilised due to the inability to blind subjects from their respective sleep condition; therefore, Martin and Haney (1982) removed knowledge of the self-selected treadmill grade (thus the pacing strategy) whilst requiring subjects to maintain a RPE of 14. The authors reported sleep deprivation had no effect on workload for a set RPE during treadmill exercise. Therefore, in conjunction with findings by Oliver et al. (2009), it is suggested that pacing is likely to be a conscious regulation of exercise intensities due to the knowledge of sleep disruption rather than just physiological variables alone.

Physiological and Perceptual Responses to Steady-State and Incremental Exercise

To gain understanding of the contribution that sleep deprivation and the associated perturbations have on exercise performance and pacing, an examination of responses to fixed-paced exercise protocols may be of some benefit. Time to exhaustion has been shown to be reduced following sleep loss; with Martin (1981) reporting time to volitional exhaustion was decreased by 11% during heavy treadmill walking following 36 h sleep deprivation. Mougin et al. (2001) also reported partial sleep loss due to late bedtimes and early rises resulting in reduced exercise time to exhaustion. Further, Chen (1991) also examined cardiorespiratory function during incremental cycle ergometry until exhaustion and endurance exercise tests at 75% peak power following 30 h sleep loss. Results suggest time to exhaustion was reduced following sleep deprivation while resting heart rate, plasma catecholamine (epinephrine and norepinephrine) concentrations and blood pH were decreased, and minute ventilation ($V_E$) and carbon dioxide production ($V_{CO_2}$) were increased. Collectively, these findings suggest that reductions in incremental exercise to exhaustion are a result of compromised cardiorespiratory function. However, Mougin et al. (1991) reported contrasting results, suggesting no
effect on time to exhaustion following a night of disrupted sleep in which subjects were woken for 3 hours during the night. The present data highlight the mixed research findings on the effects of sleep deprivation on exercise performance, although the conflicting results may be due to the level of sleep loss, with subjects in Mougin et al. (1991) study only experiencing disrupted sleep (3 hours sleep loss) as opposed to an acute bout of prolonged sleep deprivation.

In contrast to the observed changes in cardiorespiratory responses during incremental exercise to exhaustion, substantial (30–72 h) sleep deprivation has minimal effect on cardio-respiratory responses to steady-state exercise (Horne and Pettitt, 1984; Martin and Gaddis, 1981). Despite substantial sleep deprivation durations (72 h), Horne and Pettitt (1984) reported no effect of sleep loss on maximal and sub-maximal \( \dot{V}\text{O}_2 \), gross mechanical efficiency or respiratory quotient during steady state cycling. Interestingly, despite additional exercise during the sleep deprivation period increasing energy expenditure, the inclusion of exercise while sleep deprived had no additive effect on cardio-respiratory responses during exercise. Further, Plyley et al. (1987) reported 64 h sleep deprivation plus intermittent bouts of exercise during the sleep deprivation period reduced \( \dot{V}\text{O}_2 \text{max} \) following the sleep deprivation period, but without differences in maximal heart rate, Respiratory Exchange Ratio (RER) and blood lactate concentration. Martin and Gaddis (1981) also included intermittent bouts of cycling exercise for 8 min at various intensities during the 72 h of sleep deprivation and reported \( \dot{V}\text{O}_2 \), \( \dot{V}\text{CO}_2 \), \( \dot{V}\text{E} \), HR and blood pressure (BP) were not different between conditions. Furthermore, no differences in HR, \( \dot{V}\text{O}_2 \), \( \dot{V}\text{E} \) and core temperature responses were evident during lower intensity treadmill walking. Unlike incremental exercise to exhaustion, sleep loss has minimal effect on cardiovascular responses to steady-state exercise possibly due to the lesser strain placed on the body during steady-state exercise (Table 2.1).

Similar to cardiovascular responses, thermoregulatory responses to exercise following sleep deprivation have demonstrated equivocal results. Sawka et al. (1984) reported during 40 min of
cycling in warm conditions, sleep deprivation reduces evaporative and dry heat loss. As sleep deprivation is associated with increased physical activity (Driver and Taylor, 2000) it may be that the increased metabolic heat production may result in increased body temperature, although a collection of results have suggested otherwise (Martin and Chen, 1984; Meney et al. 1998). For example, Martin and Chen (1984) and Kolka et al. (1988) both reported that core and skin temperatures were not affected by sleep loss during steady-state exercise. Similarly, Meney et al. (1998) reported no effect of sleep deprivation on body temperature and further highlighted that when activity-related rises in core temperature were portioned out sleep loss still had no effect on thermoregulatory responses. Therefore, such findings indicate that thermoregulation during fixed-paced exercise is not altered following sleep deprivation.

The comparison of previous studies examining the effects of sleep deprivation on subsequent incremental and steady-state exercise performance is difficult due to the considerable variation in study design; including, exercise type and duration, duration of sleep deprivation, alterations in diets and the use of sleep medications (Horne and Pettitt, 1984; Martin and Gaddis, 1981; Martin and Hanley 1982). The general overview of the literature suggests sleep disruption and/or deprivation can negatively affect exercise performance; although these conclusions and mechanisms responsible remain equivocal. Furthermore, differences in findings relating to exercise performance have been linked with various physiological and metabolic perturbations although to date the precise relationships remain ambiguous and inconsistent.

**Strength, Power and Anaerobic Exercise Performance**

In comparison with endurance exercise protocols the literature examining the effect of sleep deprivation on muscle strength and power is rather limited, with the existing research varying considerably in terms of types of protocols performed, amount of sleep deprivation, and time of day the tests were completed (Table 2.2). Reilly and Piercy (1994) reported that disrupted sleep for 3
consecutive nights reduces strength during both upper and lower body exercises. Contrary to these findings, 60 h sleep loss has been shown to have no effect on 25 repetitions of maximal isokinetic and isometric force of the upper and lower body (Symons et al. 1988). Further, isometric handgrip strength is not affected by one night sleep deprivation (Meney et al. 1998) or 64 h sleep deprivation with additional exercise intermittently spaced throughout the sleep deprivation period (Takeuchi et al. 1985). The attenuation of maximal strength and power may have performance implications to specific tactical demands during a match, such as jumping, agility maneuvers and sprinting. Accordingly, Takeuchi et al. (1985) reported 64 h sleep deprivation with or without additional exercise reduced vertical jump height. Waterhouse et al. (2007) has also reported that 20 m sprint times can be improved following a 30 min post-lunch nap when someone is suffering from sleep loss. Similar to aerobic performance, the direct effect of sleep deprivation itself or the associated perturbations on anaerobic exercise performance is unclear. However, this collection of studies indicates sleep deprivation has minimal effect on isometric contractions, but impairs muscular power of the lower extremities. These findings are of importance for team sports where the required demands may include explosive movements throughout a match.

Similar to studies examining the effect of sleep deprivation on strength, the investigation of peak power are limited and predominately use cycle ergometry (Mougin et al. 1996; Souissi et al. 2003). From these data it seems that time of day may influence Wingate test performance following sleep loss with afternoon testing compared to morning testing resulting in more deleterious effects following sleep deprivation, possibly due to the longer duration of deprivation. Mougin et al. (1996) reported partial sleep deprivation (retiring at 03:00) did not affect power output during a Wingate test performed at 09:00 or 12:00 the following day compared to the reference night. Further, Souissi et al. (2003) examined one night sleep deprivation on subsequent Wingate test performance during the morning (06:00) and evening (18:00) for measures of peak and mean power. Results indicated morning performances were not affected by sleep loss, although 36 h sleep loss (18:00) negatively
affected peak and mean power performance. Collectively, these studies suggest that exercise time-of-day and/or sleep duration effect peak power, with later testing and thus longer sleep deprivation duration having larger effects. However, Symons et al. (1988) reported that greater sleep deprivation (60 h) had no effect on anaerobic performance (performed at 13:00), which included no differences in upper and lower body strength, lactate and cardiorespiratory responses to steady state exercise.

Summary

The examination of sleep deprivation on team sport (intermittent-sprint) exercise has not been previously completed, although previous literature examining components of team sports may provide some insight into the potential effects. It appears that self-paced exercise performance following sleep deprivation is related to the conscious regulation of exercise intensity, as differences between conditions dissipate when the change in performance (i.e. change in treadmill grade) is blinded to the subject. These findings may indicate sleep loss may affect the self-paced component of intermittent-sprint exercise as a conscious down-regulation of exercise intensities. Furthermore, the changes in cardio-respiratory variables during incremental exercise support the suggestion that self-paced exercise may be down-regulated to minimise changes in these respective variables; however, this has not been examined during intermittent-sprint exercise. Finally, literature in Table 2.2 highlights lower body power is reduced following sleep deprivation, particularly with more pronounced sleep deprivation durations. Therefore, team sport exercise performance may potentially be negated by sleep deprivation; however the effect of sleep deprivation on self-paced intermittent-sprint performance is yet to be examined.
Table 2.1: Summary of studies examining the effect of acute sleep deprivation on self-paced exercise performance and the effects of sleep loss on physiological and perceptual responses during steady state exercise.

<table>
<thead>
<tr>
<th>Author</th>
<th>n=</th>
<th>SDEP Duration</th>
<th>Exercise Protocol</th>
<th>Effects of Sleep Deprivation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Self-Paced Exercise</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Martin and Haney</td>
<td>24</td>
<td>30 h</td>
<td>Constant treadmill speed, adjust grade to maintain ‘very hard’ RPE</td>
<td>- No effect on treadmill grade (17.1 vs 17.5%)</td>
</tr>
<tr>
<td>Oliver et al. (2009)</td>
<td>11</td>
<td>30 h</td>
<td>30 min at 60% $\dot{V}O_{2\text{max}}$ and 30 min ‘self-paced’ treadmill running</td>
<td>- Less distance covered&lt;br&gt;- No effect on RPE, core and skin temperatures, HR&lt;br&gt;- Increased $\dot{V}O_{2}$ at end of fixed-paced exercise</td>
</tr>
<tr>
<td><strong>Steady State Exercise</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chen (1991)</td>
<td>15</td>
<td>30 h</td>
<td>a) 1 min incremental exercise until exhaustion&lt;br&gt;b) Endurance time at 75% $\dot{V}O_{2\text{max}}$</td>
<td>- Resting HR, catecholamine levels, blood pH decreased&lt;br&gt;- Resting minute ventilation and $\dot{V}CO_{2}$ increased&lt;br&gt;- Reduced time to exhaustion&lt;br&gt;- No effect on endurance performance</td>
</tr>
<tr>
<td>Horne and Pettitt (1984)</td>
<td>12</td>
<td>72 h</td>
<td>Cycling at 40, 60, 80% $\dot{V}O_{2\text{max}}$ for total of 40 min duration</td>
<td>- Greater variability of mechanical efficiency&lt;br&gt;- Minimal effect on $\dot{V}O_{2}$</td>
</tr>
<tr>
<td>Kolka et al. (1988)</td>
<td>6</td>
<td>33 h</td>
<td>30 min at 60% $\dot{V}O_{2\text{peak}}$ on a cycle ergometer</td>
<td>- Tes not effected&lt;br&gt;- Depression in forearm blood flow&lt;br&gt;- No effect on arm skin temperature&lt;br&gt;- Reduction in reflex cutaneous vasodilation during exercise</td>
</tr>
<tr>
<td>Martin et al. (1986)</td>
<td>8</td>
<td>a) 30 min heavy treadmill walking&lt;br&gt;b) 3 h treadmill walking</td>
<td>- No effect on HR, core temperature, $\dot{V}O_{2}$ or $\dot{V}E$, cortisol or $\beta$-endorphins&lt;br&gt;- Disturbed mood states</td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Hours</td>
<td>Condition/Procedure</td>
<td>Findings</td>
<td></td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-------</td>
<td>-------------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Martin and Chen (1984)</td>
<td>8</td>
<td>Steady state walking following by walking until exhaustion</td>
<td>Reduced time to exhaustion, no effect on $\dot{V}O_2$, $\dot{V}CO_2$, HR, ( \dot{V}_E ), blood La', epinephrine, norepinephrine or dopamine</td>
<td></td>
</tr>
<tr>
<td>Martin and Gaddis (1981)</td>
<td>6</td>
<td>8 min each at 25, 50, 75% $\dot{V}O_{2\text{max}}$ on a cycle ergometer</td>
<td>No effect on $\dot{V}O_2$, $\dot{V}CO_2$, HR, $\dot{V}_E$, BP</td>
<td></td>
</tr>
<tr>
<td>Meney et al. (1988)</td>
<td>11</td>
<td>One night (~30h) 5 min on a cycle ergometer at a work rate they felt they could maintain for 30 min</td>
<td>No effect on selected work rate or heart rate, reduced RPE</td>
<td></td>
</tr>
<tr>
<td>Mougin et al. (1991)</td>
<td>7</td>
<td>Partial sleep loss (3 h during the night) 20 min at 75% $\dot{V}O_{2\text{max}}$, followed by incremental exercise to exhaustion</td>
<td>Increased HR, $\dot{V}_E$ at sub-maximal and maximal exercise, increased blood lactate during maximal and sub-maximal exercise</td>
<td></td>
</tr>
<tr>
<td>Plyley et al. (1987)</td>
<td>12</td>
<td>Pre and post sleep deprivation $\dot{V}O_{2\text{max}}$ test. Additional 1h treadmill walking every 3 h during the 64 h SDEP.</td>
<td>Reduced $\dot{V}O_{2\text{max}}$ and $\dot{V}_{E\text{max}}$, no effect on HR, RER and blood lactate, additional exercise had no effect</td>
<td></td>
</tr>
<tr>
<td>Sawka et al. (1984)</td>
<td>5</td>
<td>40 min at 50% $\dot{V}O_{2\text{peak}}$ on a cycle ergometer</td>
<td>Greater rise in esophageal temperature ($T_{es}$) during exercise, lower sweat rate, local sweat rate and chest thermal conductance lower in final 20 min of exercise</td>
<td></td>
</tr>
</tbody>
</table>

BP = blood pressure; HR= Heart Rate; La' = Lactate; RER = Respiratory Exchange Ratio; RPE = Rating of Perceived Exertion; SDEP = sleep deprivation; $T_{es}$ = oesophageal temperature; $\dot{V}CO_2$ = carbon dioxide production; $\dot{V}_E$ = minute ventilation; $\dot{V}_{E\text{max}}$ = maximal ventilation; $\dot{V}O_2$ = oxygen consumption; $\dot{V}O_{2\text{max}}$ = maximal oxygen consumption; $\dot{V}O_{\text{peak}}$ = peak oxygen consumption
Table 2.2: Summary of studies examining the effect of acute sleep deprivation on muscle strength and power and the concomitant effects of physiological and perceptual responses.

<table>
<thead>
<tr>
<th>Author</th>
<th>n=</th>
<th>SDEP Duration</th>
<th>Exercise Protocol</th>
<th>Effects of Sleep Deprivation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Muscular Strength and Power</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bulbulian et al. (1996)</td>
<td>24</td>
<td>30 h (plus computer tasks and walking)</td>
<td>45 consecutive contractions at 3.14rad s(^{-1})</td>
<td>- Peak Torque reduced and with added walking &lt;br&gt; - No effect on fatigue index</td>
</tr>
<tr>
<td>Reilly and Piercy (1994)</td>
<td>8</td>
<td>Partial sleep loss (3 h during the night)</td>
<td>Maximal and submaximal strength: bicep curl, bench press, leg press, dead lift</td>
<td>- Greater strength declines during sub-maximal lifts</td>
</tr>
<tr>
<td>Souissi et al. (2003)</td>
<td>13</td>
<td>24 – 36 h</td>
<td>Wingate Anaerobic Power Test</td>
<td>- Morning Wingate performance was not affected (24 h sleep loss) &lt;br&gt; - Afternoon (36 h sleep loss) peak, mean and max power were significantly reduced</td>
</tr>
<tr>
<td>Symons et al. (1988)</td>
<td>11</td>
<td>60 h</td>
<td>Maximal and sub-maximal isometric strength; Wingate Anaerobic capacity test; reaction time; 20 min treadmill exercise at 70% (\text{VO}_{2\max})</td>
<td>- Increased HR and RPE during steady state exercise &lt;br&gt; - No effect of muscular strength or power output during Wingate, reaction times</td>
</tr>
<tr>
<td>Takeuchi et al. (1985)</td>
<td>12</td>
<td>64 h (plus intermittent exercise)</td>
<td>40 m sprint, isometric handgrip strength, balance, vertical jump and isokinetic extension force</td>
<td>- No effect on 40m sprint, hand grip strength or balance &lt;br&gt; - Significantly reduced vertical jump height and extension force at 60° s(^{-1}) and 180° s(^{-1})</td>
</tr>
</tbody>
</table>

HR= Heart Rate; RPE = Rating of Perceived Exertion; rad s\(^{-1}\) = radians per second; SDEP = sleep deprivation; \(\text{VO}_{2\max}\) = maximal oxygen consumption
Potential Mechanisms Explaining the Effect of Sleep Deprivation on Performance

Energy Expenditure and Metabolic Demands of Sleep Deprivation

One of the most accepted theories regarding the purpose of sleep relates to the processes of tissue restoration and energy conservation (Bonnet, 1980; Driver and Taylor, 2000). Consequently, sleep deprivation disrupts the restorative process and the ability to conserve energy due to increased energy expenditure and metabolic demands. The conservation of energy during sleep is achieved through the reduction in energy expenditure 10-15% below values observed during rest; thus allowing the restoration of energy that has been expended during the hours of wakefulness (Driver and Taylor, 2000). Specifically, energy conservation during sleep includes the resynthesis of fuel substrates via glycogenesis (Van Cauter et al. 1997). In conjunction with glycogen resynthesis, the importance of sleep for resting metabolic processes has been further highlighted with reduced glucose tolerance following both acute and chronic bouts of sleep deprivation (Spiegel et al. 2005; Van Cauter et al. 1997). It is believed changes in the circadian rhythms of responses to exogenous glucose may be responsible for the reduced glucose tolerance as glycogenesis is suppressed during sleep. Furthermore, the circadian rhythm of glucose utilisation may also be disrupted due to sleep deprivation, as utilisation is greatest during waking and lowest during sleep (Knuston et al. 2007). The changes in glucose tolerance and utilisation due to disrupted circadian rhythms may lead to an inability to increase intracellular glucose and resynthesis depleted muscle glycogen stores; thus having potential implications for successive exercise performance (Hargreaves, 2004; Hawley et al. 1997b). However, the majority of the research on sleep deprivation and glucose metabolism and tolerance has examined the effects on metabolic diseases such as diabetes mellitus (Spiegel et al. 2005; Van Cauter et al. 1997). Additionally, no research has examined the effect of muscle glycogen content following sleep deprivation on exercise performance.

The literature above highlights the altered regulation of blood glucose following sleep disruption; however, Bonnet (1980) was the first to acknowledge the role of increased energy expenditure during
sleep deprivation on subsequent exercise performance. Accordingly, Bonnet (1980) designed a study that matched the energy expended during activities including marching while not in a sleep deprived state to energy expended during 40 hours of sleep deprivation in a sedentary state. Results suggest that the (similar) increased energy expenditure during both the marching and the inactive sleep deprivation reduced performance on mathematical addition tasks, vigilance, choice reaction time, short-term memory tasks and mood states. Such findings suggest the energy expenditure associated with sleep deprivation may play a primary role in impaired cognitive performance. These findings are of importance for team sport athletes, as technical processes within a match which require higher cognitive processes and decision making skills may lead to altered performance outcomes. In conjunction with the previously mentioned effects on glucose tolerance while sleep deprived, although not directly measured, the increased energy expenditure associated with sleep deprivation may compromise muscle glycogen resynthesis via glycogenesis (Knuston et al 2007). Despite sleep deprivation removing one’s ability to be in a state of complete rest, increased energy expenditure and potentially compromised resynthesis of fuel substrates, no studies to date have specifically examined the effect of sleep deprivation on muscle glycogen concentration as a potential cause of decline in subsequent exercise performance.

**Mood and Perception**

In conjunction with physiological changes, sleep deprivation can also affect mood states and perception, both of which have been suggested to have negative effects on subsequent performance (Myles, 1985). Accordingly, sleep deprivation can be a source of irritability, mental fatigue and loss of motivation (Meney et al. 1998); and as expected, sleep disruption also results in increased feelings of sleepiness (Blagrove et al. 1995; Waterhouse et al. 2007). Sleep loss has also been associated with negative mood states including increased perceptions of fatigue, vigor and confusion (Bonnet 1980; Meney et al. 1998; Reilly and Piercy, 1994). Several studies have also examined the additive effects of sleep deprivation and intermittent-exercise throughout the sleep loss period on mood states,
Chapter 2 – Review of Literature

reporting that 30 h sleep deprivation negatively affects subjective vigor, fatigue and depression assessed using the Profile of Mood States (POMS) questionnaire (Scott et al. 1996). Participants who also completed intermittent, sub-maximal exercise throughout the 30 h sleep deprivation were more vulnerable to negative mood states (Scott et al. 1996). Angus et al. (1985) also examined sleep deprivation and exercise every 3 h on subsequent mood states over a 60 h period, reporting sleep deprivation resulted in negative effects on ratings of fatigue, sleepiness, and overall mood, although additional exercise had no further effect on mood states. If athletes are subject to acute or partial sleep deprivation, negative mood states and level of alertness induced by the sleep loss can be alleviated with caffeine ingestion (Penetar et al. 1993) and 30 min naps (Waterhouse et al. 2007). Therefore, if environments are not conducive to sleep and sleep deprivation and associated negative mood states are unavoidable by athletes, the potential deleterious effects on performance may be reduced via the use of short duration naps and caffeine respectively.

Similar to research examining the effects of sleep deprivation on mood states, RPE also appears to be affected by sleep loss. Myles (1985) suggests that RPE responses to shorter duration protocols are not affected by sleep deprivation, while significantly higher sleep deprived RPE values are present during prolonged exercise durations and incremental exercise to exhaustion. Accordingly, Myles (1985) examined the combined effects of 60 h sleep deprivation and physical fatigue (50min at 28% \( \dot{V}O_{2\text{max}} \) every 3 h of sleep loss) on RPE responses during sub-maximal exercise bouts. Results showed RPE increased progressively with sleep loss. During the second phase of the experiment by Myles (1985), female subjects performed 10 x 30 s leg cycling bouts before and after 54 h sleep loss, reporting no effect of sleep on RPE during exercise. Finally, Myles (1985) examined 2 sets of 10 x 30s bouts of leg cycling before and after a treadmill protocol at 70% \( \dot{V}O_{2\text{max}} \) until exhaustion. Increased RPE during the treadmill running was reported following sleep deprivation with minimal changes during the pre and post cycling efforts. This collection of studies completed by Myles (1985) shows RPE is primarily affected during prolonged exercise durations and may be of importance to team sport
athletes who are required to complete > 60 min of exercise. Consequently, a down-regulation in pacing strategies may be present following sleep deprivation to protect RPE responses compared to exercise following a normal night sleep.

In relation to the previously proposed models, the mechanisms responsible for increased perception of effort for a given exercise bout following sleep deprivation may be centrally mediated, and more specifically indicate the presence of a conscious regulation of exercise intensities. Previous literature broader than sleep deprivation has shown increased RPE is associated with increased physiological and metabolic demands (Borg, 1987). Furthermore, RPE is considered the conscious perception of fatigue in response to the sub-conscious regulation of pacing strategies during self-paced exercise (St Clair Gibson et al. 2003). Studies examining the effect of sleep deprivation on RPE responses during self-paced exercise have indicated a conscious protection of RPE following sleep deprivation (Oliver et al. 2009). These findings support previous literature of a (feed-forward) anticipatory regulation to monitor and protect RPE throughout the given exercise bout (Tucker, 2009). Martin and Haney (1982) blinded participants to their perceived exertion through altering treadmill grade to match a set RPE. These findings are interesting in that sleep loss had no effect on RPE, represented by changes in treadmill grade. This study design was original in that it blinded the conscious effects of sleep deprivation on performance; however this is not likely in a ‘real-world’ setting. More recently Oliver et al. (2009) reported significantly less distance during 30 min of self-paced running and no differences in RPE, indicating that alterations in perception of effort may have accounted for the performance decline. The lack of difference between conditions during the Martin and Haney (1982) study, in conjunction with findings by Oliver et al. (2009), suggests that reduced exercise performances associated with sleep deprivation have some conscious regulatory origin.

In summary, the effect of sleep deprivation induced reductions on mood states and perceived exertion is evident; however, the effect of mood states on exercise performance *per se* remains more equivocal.
During self-paced exercise protocols, the manipulation of exercise intensity (pacing strategies) in response to a conscious RPE (Tucker, 2009) appears consistent with findings from sleep deprivation research. This anticipatory ‘feedback model’ suggests pacing strategies are set due to feedback regarding physiological changes and pacing is altered in order for the conscious RPE to match the ‘template RPE’. However, quite often minimal physiological differences are evident following sleep deprivation; with the main variable influenced is mood. Therefore, it may be suggested that exercise intensities are regulated following sleep deprivation, and reductions in performance may be due to maintenance of a conscious RPE (evident with similar RPE ratings despite different conditions).

Cognition

Another variable influenced by sleep deprivation and associated with team sport performance is mental cognition, particularly given the high volume of technical and tactical match demands. Such detrimental effects can include a wide range of psychological and cognitive functions; and the severity appears to be dependent on the complexity of the subsequent task (Ikegami et al. 2009). It has been shown that the negative effects of sleep deprivation are exacerbated when the tasks to be carried out are relatively simple, monotonous and involve minimal environmental stimulation (Folkard, 1990; Rosekind, 2008; Van Dongen and Dinges 2005; Waterhouse et al. 2001). Simple skills affected by sleep deprivation include vigilance, simple and choice reaction times (Scott et al. 2006; Waterhouse et al. 2007) and short-term memory (Kim et al. 2001; Waterhouse et al. 2007). Additionally, studies have highlighted sleep deprivation to negatively affect auditory vigilance, logical reasoning, mental addition and visual search tasks (Angus et al. 1985; Bonnet, 1980). Collectively, sleep disruptions seem to have lesser effect on tasks of greater complexity and that are rule-based, but become more sensitive to sleep deprivation when tasks increase in familiarity (Harrison and Horne 2000).

While the previous research has indicated that simple tasks are primarily affected by sleep deprivations, there is increasing evidence that one night sleep deprivation leads to the deterioration of
more complex skills (Kim et al. 2001; Thomas et al. 2000). The deterioration of complex skills following sleep deprivation has been suggested to be related to decreased brain activity, predominately within the prefrontal region and cerebral cortex (Harrison and Horne, 2000). More specifically, the decrease in neural activity in the sub-cortical area may be linked to reductions in alertness and attention, and the reduced prefrontal cortex activity associated with higher order cognitive processes (Thomas et al. 2000). Thomas et al. (2000) further highlights that alertness and cognitive performance associated with a Serial Addition/Subtraction task were reduced in alignment with reduced global brain activity, particularly cortical regions, during 85 h sleep deprivation. Kim et al. (2001) also reported cognitive functions such as motor, rhythm, receptive and expressive speech, memory and complex verbal arithmetic function are decreased following 24 h sleep deprivation. Kim et al. (2001) further discusses that some of these functions are primarily associated with right anterior hemisphere and sub-cortical areas. Interestingly, the right hemisphere is also associated with emotion, mood and perceived mood states, which are also affected by sleep. Finally, Leproult et al. (2003) reported performance during selective attention task (subjective alertness) and sustained attention task (objective alertness) were reduced following 27 h sleep deprivation. In summary, it appears that both simple and complex cognitive tasks are affected by sleep deprivation which is associated with declines in neural activity within certain regions of the brain. This may be of interest to team sport athletes as high cognitive demands including decision marking, reaction times, and alertness can be compromised when in a sleep deprived state, leading to performance declines and an alteration in the conscious regulation of pacing strategies.

**Summary**

Sleep deprivation may be experienced by team sport athletes; however, minimal research has examined the effect of acute sleep deprivation on performance of these athletes. Previous literature examining the effects on sleep deprivation on various exercise modes that are components of team sports suggests that sleep loss may potentially affect intermittent-sprint exercise performance. The
mechanisms that attempt to explain performance decrements following sleep loss are unclear, possibly due to the vast range of methodological practices used, including different exercise types, intensities, durations and exercise time-of-day, sleep deprivation durations and the use of intermittent exercise during the sleep deprivation period. Despite the inconsistencies in sleep deprivation research methodology, the general consensus is that sleep deprivation affects prolonged exercise performance, possibly due to altered glucose metabolism responses, without substantial influences on cardiovascular or thermoregulatory variables. Furthermore, conscious awareness of altered mood states and perception following sleep loss may be a significant contributor to changes in exercise performance. In relation to the previously outlined models, the use of sleep deprivation may be an effective intervention that can affect both physiological and perceptual states, and thus alter pacing strategies during intermittent-sprint exercise. To date, the effect of sleep deprivation on pacing strategies during self-paced exercise and the mechanisms responsible have not been examined, and more specifically, no research has been completed in relation to team sports.

2.5 Carbohydrate Ingestion

The practice of increasing muscle glycogen concentration prior to exercise via CHO ingestion is used extensively by team sport athletes. The primary aim of increasing such fuel substrates is to delay the development of fatigue that is often associated with reduced muscle glycogen content (Karlsson and Saltin 1971; Widdick et al. 1993; Williams et al. 1992). The examination of CHO ingestion on self-paced intermittent-sprint performance and pacing strategies is also an effective tool to examine the previously proposed models explaining the regulation of fatigue during self-paced exercise (section 2.3.5). For example, specific discussions regarding the direct effect of substrate availability has on the active musculature (Energy Supply/Energy Depletion Model), the proposed central regulation of glycogen content (Central Governor and Teleoanticipatory Models), and the conscious regulation of exercise due to the knowledge of CHO ingestion (Psychological/Motivation Model). As team sport athletes experience prolonged bouts of high-intensity activity that are predominately fuelled by CHO,
the use of nutritional interventions to minimise performance declines associated with muscle glycogen depletion may further elucidate the mechanisms responsible for overall and modal pacing during intermittent-sprint exercise.

In order for CHO ingestion to provide maximal benefits for team sport athletes, athletes should ensure adequate CHO is ingested to match or exceed the muscle and liver glycogen required for the subsequent exercise demands. Similar to continuous exercise protocols, the importance of CHO ingestion prior to intermittent-sprint exercise has been associated with increased substrate availability and the elevated exogenous glucose oxidation during subsequent exercise (Krstrup et al. 2006). However, the effectiveness of ingestion of this macronutrient prior to intermittent-sprint exercise appears to be dependent upon several dietary factors including CHO type, timing and volume, and the duration and intensity of the subsequent exercise bout (Burke et al. 2004). The primary aim of manipulating these dietary factors have been linked with maximising endogenous CHO stores within the muscle and liver and thus minimising exercise-induced depletion of muscle glycogen stores that are prevalent during team sport exercise (Saltin, 1973).

The effect of CHO ingestion on exercise performance is of particular interest to team sport athletes. Previous literature has shown that muscle glycogen concentrations are significantly reduced during a soccer match (Krstrup et al. 2005; Saltin, 1973). The prevalence of glycogen depletion during team sports is likely to be associated with the higher demand on glycogen as the primary fuel source (due to aerobic glycolysis), thus increasing muscle glycogen utilisation and accelerating the depletion of glycogen stores (Kopke et al. 1985). Such outcomes create an environment in which team sport athletes must monitor CHO intake to maximise substrate availability and avoid impairing performance that is associated with muscle glycogen depletion (Balsom et al. 1999b). Accordingly, the use of CHO as an intervention in this thesis provides an appropriate environment to assess the mechanisms responsible for the effects of pre-exercise physiological and perceptual states in relation
to the previously mentioned models of fatigue and examine the influence of pacing on specific modes (namely high intensity efforts) during self-paced intermittent-sprint exercise.

**Effects of Pre-Exercise CHO Ingestion on Exercise Performance**

*Intermittent-Sprint Exercise Performance*

In relation to team sports, increased muscle glycogen due to increased pre-exercise CHO ingestion has been associated with improvements in intermittent exercise performance (Balsom et al. 1999a; Bangsbo et al. 1992; Table 2.3). Field-based studies examining pre-exercise CHO intake on competition match performances are limited, but have reported improvements in match performance compared to more moderate CHO intakes (Balsom et al. 1999b; Bangsbo et al. 1992). Balsom et al. (1999b) reported CHO ingestion (65% total dietary intake) 48 h prior to a 90 min match increased pre-exercise muscle glycogen compared a low CHO diet (30% total dietary intake); resulting in 33% greater high-intensity exercise during the match. Bangsbo et al. (1992) utilised field and laboratory based protocols and examined distance covered during a soccer match followed by a treadmill run to exhaustion. Results showed distance covered during the match was higher following a 2 day high CHO (65% CHO) diet compared to a control (39% CHO) diet. These studies suggest a high CHO diet and increased muscle glycogen content significantly improves repeated, high-intensity efforts. While the maintenance of high intensity efforts is apparent in team sport exercise following a high CHO diet, such effects do not seem to extend to technical match skills. Abt et al. (1998) reported a high CHO diet for 48 h prior to exercise had no effect on soccer skills such as shooting and dribbling following a 60 min intermittent treadmill protocol. These findings suggest that glycogen content has minimal effect on technical skill performance, although may alter physical work performed. It is also likely that the 60 min treadmill protocol intensity and duration was not sufficient to deplete muscle glycogen stores to a point at which soccer skill performance would be detrimentally affected. Collectively, although minimal effects are evident on skills performance, these field-based studies suggest that higher CHO intake is important for the maintenance of high-intensity efforts during team sports.
As outlined in Table 2.3, laboratory-based intermittent-sprint exercise protocols designed to simulate team sports support field-based studies in that increased CHO ingestion increases physical performance during high-intensity activities. While some inferences from these repeat-sprint protocols regarding high-intensity exercise performance can be made to team sports; most studies have examined power output during maximal sprint efforts without inclusion of an active recovery, and thus do not investigate pacing strategies of specific modes. Balsom et al. (1999a) reported a high CHO diet 48 h prior to intermittent-sprint exercise increased glycogen concentrations compared to a low CHO diet. The increased CHO intake and glycogen content improved work during repeated 6 s sprints every 30 s over short (10min) and longer (30min) durations. Additionally, Jenkins et al. (1993) reported a 5.6% and 2.3% increase in total work completed during 5 x 60 s maximal cycling efforts following 3 day high (83% of energy was CHO) and moderate (58% CHO) CHO diets compared to a 5.4% decline during the low (12% CHO) CHO condition. Finally, Casey et al. (1996) similarly reported a high CHO diet for 3 days that was preceded by a depleting bout of exercise increased total work during 3 x 30 s maximal sprints; although no effect was evident on a 4th sprint. Unfortunately, muscle biopsies were not collected during this study to determine the effects of muscle metabolism on performance differences. Similar to field-based research, laboratory-based intermittent-sprint protocols indicate that high-intensity exercise is primarily affected by reduced CHO intake and muscle glycogen content. Despite these findings, minimal research has examined the dynamic interplay of all modes of prolonged intermittent-sprint exercise following CHO ingestion and the mechanisms responsible for any altered pacing strategies observed (Balsom et al. 1999b).
Table 2.3: Summary of the studies examining the effect of pre-exercise carbohydrate ingestion on physiological and perceptual responses and intermittent-sprint exercise performance.

<table>
<thead>
<tr>
<th>Author</th>
<th>n=</th>
<th>CHO Diet</th>
<th>Exercise Protocol</th>
<th>Effect of HCHO on Physiological Responses</th>
<th>Effect of HCHO on Performance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intermittent-Sprint Exercise Performance</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abt et al. 1998</td>
<td>6</td>
<td>80% CHO (HCHO); 40% CHO (LCHO) 48 h before ex (preceded by glycogen depleting ex)</td>
<td>- Modified Zelenka Functional Performance Test - 60 min intermittent treadmill protocol</td>
<td>↔ Glu or La^-</td>
<td>- Post-treadmill skills test completion faster during LCHO compared to pre-treadmill - No effect of condition on soccer skills</td>
</tr>
<tr>
<td>Balsom et al. 1999b</td>
<td>6</td>
<td>65% CHO (HCHO); 30% CHO (LCHO) 48 h before ex</td>
<td>90 min soccer match</td>
<td>↑ pre-ex muscle glycogen ↓ FFA, glycerol and glucose concentrations during ex ↔ La^- or HR</td>
<td>- 33% greater high-intensity ex - No effect on technical variables</td>
</tr>
<tr>
<td>Balsom et al. 1999a</td>
<td>7</td>
<td>67% CHO (HCHO); 4% CHO (LCHO) 48 h before ex</td>
<td>6s sprints every 30s over 10 min and 30 min (until fatigue)</td>
<td>↑ pre-ex muscle glycogen ↔ post-ex FFA, glycerol and La^- ↔ O2 but higher RER during 30 min protocol</td>
<td>- Increased work during sprints during 10 min and greater number of sprints during 30 min protocol</td>
</tr>
<tr>
<td>Bangsbo et al. 1992</td>
<td>7</td>
<td>65% CHO (HCHO); 39% CHO (LCHO)</td>
<td>Soccer match followed by treadmill run TTE</td>
<td>↑ pre-ex muscle glycogen ↔ blood La^- or Glu ↑ RER during treadmill run</td>
<td>- Longer running distance</td>
</tr>
<tr>
<td>Casey et al. 1996</td>
<td>11</td>
<td>81.5% CHO (HCHO); 7.8% CHO (LCHO); 3 day diet before ex (preceded by depleting bout of ex)</td>
<td>4 x 30 s maximal cycling efforts</td>
<td>↑ blood La^- and ammonia accumulation during LCHO (unchanged in HCHO)</td>
<td>- Increased work during sprints during sprints 1-3 - No effect on sprint 4</td>
</tr>
<tr>
<td>Jenkins et al. 1993</td>
<td>14</td>
<td>83% CHO (HCHO); 58% CHO (MCHO); 12% CHO (LCHO) 3 day diet before ex</td>
<td>5 x 60 s maximal cycling efforts</td>
<td>↔VO2, pH, Glu or La^-</td>
<td>- 5.6% and 2.3% increase total work for HCHO and MCHO compared to LCHO</td>
</tr>
</tbody>
</table>

CHO = Carbohydrate; ex = Exercise; Glu = Glucose; HCHO = High Carbohydrate; La^- = Lactate; LCHO = Low Carbohydrate; MCHO = Moderate Carbohydrate; HR = Heart Rate; FFA = Free Fatty Acids; RER = Respiratory Exchange Ratio; TTE = Time to Exhaustion; VO2 = oxygen consumption
While team sports include self-paced exercise, as mentioned previously, many laboratory-based intermittent-sprint protocols designed to simulate team sport exercise do not incorporate any self-paced element. Regardless, numerous studies have been completed on continuous, self-paced exercise with evidence of exercise regulation following altered CHO and muscle glycogen states (Table 2.4). For example, previous studies have shown pre-exercise CHO ingestion to improve 30 km self-paced running performance (Karlsson and Saltin 1971; Williams et al. 1992). These improvements were associated with faster speeds in the last 5 km of the trial (end-spurt), while no end-spurt was observed in the control trial (Williams et al. 1992). Cycling trials further support the notion that pre-exercise glycogen content can influence exercise performance during the latter stages of a bout. Widdick et al. (1993) reported that during a 70 km self-paced cycling time trial, power output during the final 14% of the trial was reduced when pre-exercise CHO ingestion and muscle glycogen was significantly reduced. Accordingly, it seems CHO intake can alter pacing strategies during prolonged self-paced exercise, and these changes are evident during the final stages of the protocol when it would be expected that differences in muscle glycogen content are greatest.

While previous studies highlight that differences in exercise performance are primarily evident in the final stages of a prolonged exercise bout, the adoption of pacing strategies during self-paced exercise may also be evident in earlier stages of any exercise bout (Rauch et al. 2005). Rauch et al. (2005) reported that power output was greater after the first minute of a 1h cycling time trial following a 3-day diet consuming high volumes of CHO. Rauch et al. (2005) further explained that the performance decrements in the early stages of the time trial (following a 2 h externally-paced protocol) suggest feedback from the periphery regarding muscle glycogen content manipulated the central drive to the active musculature, reducing muscle recruitment and power output in anticipation of reaching significantly low glycogen concentrations. These findings support the theory of a sub-conscious anticipatory regulation of exercise intensities from early stages of the exercise protocol in order to
regulate physiological perturbations, in this case muscle glycogen. In relation to team sport exercise, the transient and cumulative fatigue present during matches may in part be a similar regulation of metabolic processes throughout the match. However, these findings are in contrast to Johnson et al. (2006) who reported performance differences during a ~2 h cycling protocol were not evident until 80% of trial was completed (~96 min). During the study, subjects were blinded to the knowledge that CHO was being manipulated and therefore, Johnson and colleagues (2006) suggest that changes in performance during early stages of a self-paced protocol are due to the knowledge of CHO manipulation rather than physiological perturbations or anticipatory regulation of muscle glycogen. Although the mechanisms responsible are not as clear, collectively these studies indicate that low muscle glycogen content is detrimental to self-paced exercise performance and the ingestion of CHO pre and during exercise may alleviate the negative effects by increasing available fuel stores.

On the contrary, numerous studies have reported no effects of CHO ingestion on self-paced exercise performance of various distances and durations. Hawley et al. (1997a) examined 3 days CHO loading compared to a normal diet on 1 h cycling time trial performance. Results indicate that muscle glycogen was significantly increased during the CHO loading trial, without differences in performance, CHO utilisation or oxidation between conditions. Hawley et al. (1997a) suggested that the high post-exercise glycogen values indicate that whole-muscle depletion did not determine fatigue. Furthermore, differences in pre-exercise glycogen concentration between conditions were only moderate (106 mmol kg\(^{-1}\) \(dw\)). Additionally, the pre-exercise glycogen content of 459 mmol kg\(^{-1}\) \(dw\) following the normal diet would have been sufficient to complete a 1 h cycling time trial without substrate depletion causing detrimental effects to performance (Hawley et al. 1997a). Similarly, Burke et al. (2000) also utilised a 3 day CHO-loading diet (9g kg\(^{-1}\) day\(^{-1}\)) compared to a placebo (6g kg\(^{-1}\) day\(^{-1}\)) on 100 km time trial performance and reported significantly greater pre-exercise glycogen content during the loading trial. Regardless of the loading protocol, time to complete the 100 km cycling trial was not affected, which may also be due to the relatively small difference in pre-exercise glycogen content.
content between conditions (87 mmol kg\(^{-1}\) dw) and the implementation of CHO feedings during exercise. In summary, performance differences during self-paced exercise performance appear to only be apparent when significant depletion is present during one of the conditions, and there is a substantial difference in pre-exercise glycogen content between conditions. Therefore, the down-regulation of pacing strategies during team sports may only be present following a low CHO diet compared to high HCHO diet, and moderate CHO intakes may still be sufficient to maintain performance during the sub-maximal, self-paced efforts.

Muscle Power and Strength Performance

In comparison to prolonged exercise protocols, the effect of pre-exercise high and low CHO diets for ~24 h prior to short duration, high-intensity exercise and effect on muscular power/strength have not been investigated extensively. Of the research completed, CHO appears to have a minimal effect on force production or work completed during short duration, high-intensity efforts (Mitchell et al. 1997; Walberg et al. 1988). However, Nybo et al. (2003) examined the neuromuscular properties associated with hypoglycemia, reporting significant reduction in force production during a 2 min maximal voluntary contraction (MVC) of the knee extensors. While Nybo et al. (2003) did not measure muscle glycogen, it was assumed the 3 h cycling protocol prior to the MVC sufficiently reduced muscle glycogen content. Interestingly, differences in MVC force were accompanied with reductions in CNS activation; suggesting that reduced force production is a result of centrally mediated declines in muscle recruitment. These findings are of importance to athletes, as evidence of down-regulation of muscle recruitment in response to peripheral feedback, including muscle glycogen and blood glucose concentrations may influence pacing strategies during competition.

The effects of pre-exercise CHO ingestion on power output during maximal, short-duration efforts are somewhat unclear, although may be dependent upon the volume of pre-exercise CHO feedings and the subsequent exercise duration. Hargreaves et al. (1997) reported following a depleting bout of
exercise, a 24 h high CHO diet (80% CHO) increased pre-exercise glycogen content compared to a low (25% CHO) diet, but these differences in glycogen content had no effect on peak power during a 75 s maximal cycling effort. Hargreaves and colleagues (1997) suggest that these findings may be due to glycogenolysis not being affected by pre-exercise glycogen availability during brief, intense exercise (Bangsbo et al. 1992; Symons and Jacobs 1989). However, previous studies have also reported a 35% reduction in glycogen content is apparent following a 30 s maximal sprint (Bogdanis et al. 1995) and during 3 x 30s cycling sprints (Spriet et al. 1989). These findings suggest there is a high dependence on muscle glycogen utilisation during maximal sprints that leads to glycogen depletion and may be amplified when sprints are repeated successively over a prolonged period of time (Saltin, 1973). Conversely, Langfort et al. (1997) reported an 8% decline in power output during a 30 s Wingate test following a 3 day diet of substantially lower CHO (5% CHO) content compared to a moderate CHO (50% CHO) diet. Irrespective of the mechanisms responsible for performance declines, it appears that glycogen depletion may be of concern to team sport athletes who engage in repeated maximal sprints over a prolonged period of time (see Tables 2.3 – 2.5).
Table 2.4: Summary of the studies examining the effect of pre-exercise carbohydrate ingestion on physiological and perceptual responses and self-paced exercise performance.

<table>
<thead>
<tr>
<th>Author</th>
<th>n=</th>
<th>CHO Diet</th>
<th>Exercise Protocol</th>
<th>Effect of HCHO on Physiological Responses</th>
<th>Effect of HCHO on Performance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Self-Paced Exercise Performance</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Burke et al. 2000</td>
<td>7</td>
<td>9g kg day⁻¹ (HCHO); 6g kg day⁻¹ (PLA); 3 day diet before ex (plus CHO consumed during ex)</td>
<td>100 km cycling TT (plus 4 x 4km sprints and 5 x 1km sprints)</td>
<td>↑ pre-ex muscle glycogen - Small differences in pre-ex glycogen (87mmol kg⁻¹ dw)</td>
<td>↔ Glycogen utilisation and Glu</td>
</tr>
<tr>
<td>Hawley et al. 1997a</td>
<td>6</td>
<td>9.3g kg⁻¹ (HCHO) ; 5.9g kg⁻¹ CHO (NORM); 3 day diet before ex</td>
<td>1 h cycling TT</td>
<td>↑ pre-ex muscle glycogen - Moderate differences in pre-ex glycogen (106mmol kg⁻¹ dw)</td>
<td>No effect on TT performance</td>
</tr>
<tr>
<td>Johnson et al. 2006</td>
<td>8</td>
<td>9g kg⁻¹ CHO (HCHO); 0.6g kg⁻¹ CHO (LCHO) 2 days before exercise (preceded by glycogen depleting ex)</td>
<td>Cycling TT until fixed amount of external work (measured in kJ)</td>
<td>↓ sense of tiredness</td>
<td>No differences in PO until 80% of a ~2 h protocol (at approx 96 min) - Increased PO during final 20% of protocol</td>
</tr>
<tr>
<td>Rauch et al. 2005</td>
<td>8</td>
<td>3 day diet before ex</td>
<td>1 h cycling TT (preceded by 2 h depleting ex)</td>
<td>↑ Muscle glycogen concentration ↔ Post-ex muscle glycogen ↔ Blood Lactate or Glu</td>
<td>Increased PO after 1st minute of exercise - End-spurt present in both conditions</td>
</tr>
<tr>
<td>Williams et al. 1992</td>
<td>18</td>
<td>566-424g CHO (HCHO) or 349-364 CHO (LCHO) diet for 7 days before ex</td>
<td>30 km running TT</td>
<td>- Maintained blood Glu compared to LCHO</td>
<td>Improved TT performance - Increased speed in final 5km</td>
</tr>
</tbody>
</table>

CHO = Carbohydrate; ex = Exercise; Glu = Glucose; HCHO = High Carbohydrate; LCHO = Low Carbohydrate; LA⁻ = Lactate; RER = Respiratory Exchange Ratio; PO = Power Output; TT = Time Trial
Table 2.5: Summary of the studies examining the effect of pre-exercise carbohydrate ingestion on physiological and perceptual responses and muscle strength and power performance.

<table>
<thead>
<tr>
<th>Author</th>
<th>n=</th>
<th>CHO Diet</th>
<th>Exercise Protocol</th>
<th>Effect of HCHO on Physiological Responses</th>
<th>Effect of HCHO on Performance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle Power and Strength Performance</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hargreaves et al. 1997</td>
<td>9</td>
<td>80% CHO (HCHO); 25% CHO (LCHO)</td>
<td>75 s maximal cycling effort</td>
<td>↑ pre-exercise muscle glycogen ↔ Venous and muscle La' concentrations</td>
<td>No effect on peak or mean PO</td>
</tr>
<tr>
<td>Langfort et al. 1997</td>
<td>8</td>
<td>50% CHO (HCHO); 5% CHO (LCHO) 3 day diet before ex</td>
<td>30 s Wingate test</td>
<td>↔ blood Glu and La’</td>
<td>8% increase in mean PO</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>No effect on peak power during first 5 s</td>
<td></td>
</tr>
<tr>
<td>Mitchell et al. 1997</td>
<td>11</td>
<td>80% CHO (HCHO); 4% CHO (LCHO) 48 h before ex</td>
<td>5 sets of 3 exercises to failure @ 15RM</td>
<td>↑ Blood Glu ↔ Blood La’</td>
<td>No effect on strength performance</td>
</tr>
<tr>
<td>Symons and Jacobs, 1989</td>
<td>8</td>
<td>‘Normal Diet’ (HCHO); 37-49% CHO (LCHO) for 48 h (preceded by depleting bout of ex)</td>
<td>Performance Evaluation Tasks (PET) including: max ISOK strength and endurance, ISOM force, twitch force</td>
<td>↑ pre-exercise muscle glycogen ↔ blood Glu and La’</td>
<td>No effect of PET measures</td>
</tr>
</tbody>
</table>

CHO = Carbohydrate; ex = Exercise; Glu = Glucose; HCHO = High Carbohydrate; ISOK = Isokinetic; ISOM = isometric; La’ = Lactate; LCHO = Low Carbohydrate; HR = Heart Rate; PET = Performance Evaluation Tasks; PO = Power Output; RM = Repetition Maximum
**Mechanisms for CHO Ingestion Improving Exercise Performance**

As previously outlined, increased pre-exercise CHO ingestion has been shown to improve subsequent endurance exercise performance due to increased pre-exercise muscle glycogen concentrations and glycogen availability during exercise. The effect of pre-exercise CHO ingestion on exercise performance may be dependent on a wide range of variables including amount, type and timing of CHO ingestion, and type and intensity of exercise completed (Jentjens and Jeukendrup, 2003; Sparks et al. 1998). Collectively, these variables influence blood glucose, insulin and catecholamine concentrations, and rates of muscle glycogen utilisation during exercise (Tsintzas and Williams, 1998). On the contrary, investigations into self-paced exercise have provided insight that peripheral perturbations associated with different pre-exercise metabolic states may be centrally regulated (Nybo, 2003). Accordingly, the CNS interprets afferent feedback from the periphery regarding muscle glycogen content and utilisation and provides appropriate feed-forward regulation of the active musculature and thus alters pacing strategies (Rauch et al. 2005). The following section of the literature review examines the proposed mechanisms related to exercise performance and pacing strategies in response to the previously outlined models of fatigue and outlines the implications these mechanisms may have on team sport athletes.

**Substrate Availability and Utilisation**

The relationship between muscle glycogen concentrations and endurance performance has been well established since the work by Bergström and colleagues (1967). Since then, a plethora of studies have shown that the increase in substrate availability due to CHO ingestion plays an integral role in the maintaining or improving exercise performance (Balsom et al. 1999a; Balsom et al. 1999b; Bangsbo et al. 1992; Jenkins et al. 1997; Langfort et al. 1997; Williams et al. 1992). The general consensus is that exercise, depending on intensity, needs to exceed ~90 min in order for prior CHO ingestion to be beneficial to performance; as muscle glycogen availability is reduced to a level that is critical in the development of fatigue (Hargreaves, 2004). In contrast, performance improvements have also been
shown during shorter duration (~60 min) intermittent-sprint exercise due to the increased reliance of CHO oxidation (Van Loon et al. 2001) and increased power output following the first minute of exercise during self-paced cycling trials (Rauch et al. 2005). Despite an increased intramuscular utilisation of glycogen following higher CHO ingestion during the days prior to exercise, absolute concentrations are greater compared to lower CHO intake during the days prior to exercise (Hargreaves, 2004). The high availability of fuel substrates is important for team sport athletes as increased rates of utilisation are observed due to the high physical demands (Saltin, 1973). Therefore, athletes may be able to indulge in higher intensity pacing strategies for longer durations before critically low muscle glycogen concentrations become apparent, resulting in excessive fatigue observed during later stages of matches (Krustrup et al. 2005).

Another mechanism relating to performance improvements following CHO intake is the increased glycogen availability increasing utilisation during exercise, particularly when substrates and hormone values remain constant between conditions (Blomstrand and Saltin, 1999; Steensberg et al. 2002). Blomstrand and Saltin (1999) reported that during 1 h of two-legged cycling, where one leg had a normal glycogen content and the other a low glycogen content, muscle glycogen utilisation was ~60% lower during the low glycogen leg. Additionally, the rate of glucose uptake during the low glycogen leg was ~30% higher. These findings are supported by Steensberg et al. (2002) who also utilised a protocol in which contralateral limbs differed in glycogen content. These protocol designs minimised the influence of other circulatory and metabolic factors that may have been present when testing on two separate occasions. Steensberg et al. (2002) reported that the exercised leg (with less muscle glycogen) had increased glucose uptake compared to the control leg. Steensberg and colleagues (2002) also examined high vs low CHO diets (on two separate occasions) on exercise metabolism, reporting similar rates of glucose uptake between high and low CHO diets and a decrease in blood glucose and insulin concentrations during the low CHO diet condition. In contrast, Hargreaves et al. (1995) reported that muscle glycogen utilisation is reduced during exercise, and despite changes in
glycogenolysis, reported no effect on glucose uptake between conditions. This study used methodology similar to the second study by Steensberg et al. (2002), with the use of diet manipulation, but subjects returned on separate occasions; therefore not allowing the tighter control of other factors that may influence glucose uptake compared to the contralateral cycling protocol. Collectively, these studies suggest that pre-exercise glycogen content affects glycogen utilisation during exercise, but only when substrate, circulatory, and hormonal influences are constant.

While it has been shown that increased glycogen availability increases utilisation during exercise, the rate of utilisation may also be task dependent. Accordingly, exercise intensities ≥ 60% \( \dot{V}O_{2\text{max}} \) have an increased dependence on CHO as a fuel source and the aforementioned discrepancies in performance between studies (Tables 2.3 – 2.5) may be partially explained by the variation of exercise intensity and modes (Romijin et al. 1993; Van Loon et al. 2001). Accordingly, Havemann et al. (2006) examined a 6 day high fat or high CHO diet, both followed by 1 day of high CHO on 100 km cycling time trial performance. RER data indicated an increased fat utilisation following the high fat diet, without effects on time trial performance compared to high CHO. However, maximal sprints were completed intermittently throughout the protocol, and the high-intensity sprints were significantly improved during the CHO condition, suggesting CHO ingestion is crucial for high-intensity exercise performance that is interspersed with sub-maximal efforts. This task dependency of fuel utilisation is of particular relevance to team sport athletes as they are engaged in high-intensity exercise (~80% \( \dot{V}O_{2\text{max}} \)) for ≥ 60 min (Coutts et al. 2003; Krstrup et al. 2005). Accordingly, the reliance on muscle glycogen utilisation during team sports has been shown with field-based reports of significantly reduced muscle glycogen concentrations and utilisation (Krustup et al. 2005; Saltin, 1973).

Muscle biopsy staining procedures have highlighted the extent of utilisation and depletion of muscle glycogen varies between muscle fibre types. Vøllestad and Blom (1985) examined glycogen depletion
in the vastus lateralis muscle during cycling exercise at 43%, 61%, and 91% \( \text{VO}_2\text{max} \). Results show that after 60 min at the lowest intensity, most of the glycogen depletion was evident in slow twitch (Type I) fibres and approximately 20% depleted in Type IIA fibres. As intensity increased (61% \( \text{VO}_2\text{max} \)) full depletion of Type I fibres were present and approximately 65% of Type IIA fibres were depleted. Finally, during high intensity exercise, glycogen depletion was present in both Type I and IIA fibres and 50% depletion during Type IIB fibres. Therefore, muscle glycogen depletion is dependent upon the recruitment patterns associated with a given exercise intensity. In relation to team sport exercise, it is likely that Type I muscle fibres are predominately recruited during the sub-maximal efforts, while recruitment of Type IIA and IIB muscle fibres during the maximal sprints would result in a decrease glycogen in the respective fibres. Consequently, following the demands of team sport match-play, it is not surprising that significant reductions in muscle glycogen content are apparent, which in turn may relate to reduced intermittent-sprint performance (Krustrup et al. 2006).

**Central Factors**

The previously outlined mechanisms of substrate availability and utilisation related to exercise performance are based on the notion that declines in exercise performance become apparent when exercise durations are long enough to cause significant declines in muscle glycogen content (Hawley et al. 1997b). However, reductions during the early periods of endurance exercise, as well as during shorter exercise durations (≤ 90 min) raise questions regarding the direct peripheral contribution muscle glycogen content has on exercise performance. It has been proposed that centrally-mediated mechanisms may also be responsible for performance differences and altered pacing strategies during exercise, rather than exclusively peripherally-mediated factors. Rauch et al. (2005) reported that during a 1 h time trial, a high pre-exercise CHO diet and muscle glycogen content significantly improved time trial performance; with differences in power output observed after the first minute of exercise. As suggested by Rauch et al. (2005), the different pacing strategies, despite similar final glycogen concentrations, heart rates and blood glucose and lactate values, indicate an anticipatory
regulation of power output. Furthermore, this was the first study to suggest the presence of a ‘gluco-stat’, whereby subjects regulated intensities in order to complete exercise at similar glycogen levels that were not substantially low enough to be catastrophic to performance for the athlete.

The notion of a ‘gluco-stat’ is not unlike theories in other exercise physiology sub-disciplines such as thermoregulation where it is suggested that exercise is regulated in order to avoid attaining a ‘critically limiting temperature’ (Gonzalez-Alonso et al. 1999; Marino, 2004). As fuel substrate availability is imperative to prolonged exercise performance, it may not be surprising that the CNS monitors and controls muscle glycogen content and utilisation, as suggested by Noakes et al. (2005). Performance differences during exercise protocols of shorter durations (< 60 min) which cannot be sufficiently explained by glycogen depletion alone (Langford et al. 1997) further supports the notion of exercise regulation in anticipation of excessive reductions in glycogen content. Accordingly, some role for centrally-mediated control of exercise intensities based on pre-exercise muscle glycogen concentration and utilisation may be present during self-paced exercise. Furthermore, the regulation of the respective self-paced modes during a team sport match may also be influenced, with reductions in high-intensity efforts the most likely affected mode, as noted in field-based research (Balsom et al. 1999b).

In contrast to findings by Rauch et al. (2005), Johnson et al. (2006) reported on subjects who were required to cycle at a self-selected pace until they had completed at fixed amount of external work (determined in kJ). All subjects were blinded to any feedback during the trial and were blinded to which CHO condition they were completing. The authors reported no differences in power output during the initial 80% of the time trial (~96min) and as the subjects were blinded to their respective conditions, such findings suggest initial changes in pacing strategies during exercise are due to the knowledge of CHO ingestion. However, subjects were provided a glucose polymer solution during the time trial to avoid hypoglycaemia. The increased availability of exogenous glucose supply during the
exercise, irrespective of the CHO condition, may have allowed sparing of muscle glycogen utilisation, thus making the differences in pre-exercise muscle glycogen, in part, redundant. Furthermore, the pacing profile of the power output, highlights an end-spurt was present during the final 10% of the protocol. As it can be assumed that this end-spurt occurs when glycogen would have been at its lowest, it is likely that power output was regulated throughout the protocol to allow the end-spurt to occur, irrespective of the CHO condition. Unfortunately, muscle biopsies were not obtained during this study and thus it is difficult to substantiate the effects of CHO intake during exercise and the role of glycogen utilisation on exercise regulation. However, the discrepancies between Rauch et al. (2005) and Johnson et al. (2006) data highlights an intriguing paradigm regarding the conscious and sub-conscious regulation of exercise intensities in relation to knowledge of pre-exercise physiological and perceptual states.

Conscious Decision Making

While changes at the periphery associated with CHO ingestion and the central regulation of the peripheral perturbations are integral for observed performance improvements, the conscious manipulation of exercise intensities cannot be discounted. As mentioned previously, Johnson et al. (2006) provides some insight into the effects of blinding subjects on CHO intake on ensuing performance, while other research (excluding the use of nutritional interventions) has further explored the idea of a conscious regulation of exercise intensity with the use of deception studies (Billaut et al. 2011; Paterson and Marino, 2004). These deception studies have successfully highlighted that knowledge of expected performance improvements with CHO manipulation can alter pacing strategies and performance during self-paced exercise. Clark et al. (2000) examined the placebo effect of CHO ingestion during a 40 km time trial performance. The methodology included 43 subjects completing a baseline session followed by grouping into either a CHO drink or a placebo. Subjects were further subdivided into being told they were consuming CHO, a placebo or no information.
Power output during the second trial compared to baseline was improved once informed of CHO and placebo intake (4.3 and 0.5%, respectively) while no information reduced power output by 1.1%.

However, using different methodology Nassif et al. (2008) examined the effectiveness of a placebo of CHO ingestion during exercise on cycling performance. The blinding was achieved with ingestion of double blind placebo and CHO capsules and a known CHO capsule. Results show that there was no performance difference between the blinded conditions indicating the physiological effects associated with CHO ingestion during exercise were not sufficient to affect performance outcomes and the knowledge of CHO ingestion during exercise has some placebo effect on time to exhaustion. Additionally, the comparison of the blinded and known CHO condition indicated no differences in performance, thus suggesting that when an athlete is in a physiologically enhanced state by ingesting CHO, knowledge of that state does not affect performance. The final comparison between conditions showed that time to exhaustion was significantly longer during the known CHO condition compared to the blinded placebo. Therefore, CHO ingestion during exercise influences time to exhaustion when differences in CHO feedings are present and the subject is aware of the CHO ingestion. These findings suggest that performance alterations associated with CHO ingestion are both conscious and sub-conscious, although the effects on intermittent-sprint exercise remain equivocal.

**Summary**

In summary, it is well established that reductions in pre-exercise muscle glycogen concentration significantly impair subsequent endurance exercise performance. Although dependent on intensity, these performance declines appear to be most apparent in exercise durations that exceed 90 min; as this exercise duration is substantial enough to significantly deplete muscle glycogen concentrations to a level to impair exercise performance. Obviously, starting exercise in a reduced glycogen state accelerates the time until these performance decrements are apparent during self-paced exercise. These observations in continuous exercise protocols have also been shown during team sport exercise,
with significant declines in glycogen content during soccer matches (Krustrup, 2005; Saltin, 1973). Although team sports generally exhibit shorter durations compared to endurance exercise, glycogen depletion during intermittent-sprint exercise may be due to the high-intensity activities performed during such exercise bouts, increasing the reliance on CHO as the primary fuel source (Gollnick et al. 1974; Van Loon et al. 2001). Accordingly, the mechanisms responsible for performance improvement when increased pre-exercise CHO feedings are provided to athletes are associated with increasing glycogen availability (stores) and increased exogenous glucose supply, thus sparing muscle glycogen stores. However, significant reductions in power output during early stages of cycling time-trial efforts and no differences in glycogen concentration at the completion of exercise suggests an anticipatory regulation of pacing strategies may also be present in response to glycogen availability and utilisation (Rauch et al. 2005). Furthermore, studies showing minimal differences in time trial performance, despite different pre-exercise muscle glycogen content suggest that the knowledge of CHO ingestion and enhanced metabolic state may contribute to an increased maintenance of performance during self-paced exercise (Burke et al. 2000; Hawley et al. 1997b). However, the effects of increased pre-exercise CHO ingestion and muscle glycogen on pacing strategies in team sport athletes have yet to be investigated. Furthermore, the influence of knowledge about such changes has also not been investigated to date.

2.6 Thermoregulation

*Exercise in the Heat and the Effect of Pre-Exercise Thermal State*

Exercise in warmer environmental temperatures is a common occurrence for team sport athletes due to teams located in warmer climates, extended competition seasons, off-season training and pre-season games. These higher environmental temperatures can be of concern to team sport athletes given exercise in the heat is associated with exacerbated declines in intermittent-sprint performance compared to thermo-neutral conditions (Drust et al. 2005; Maxwell et al. 1996; Morris et al. 2005). These performance declines throughout either a match or laboratory-based intermittent-sprint exercise
in the heat are proposed to be due to the accelerated rate of rise in core temperature from increased metabolic heat production coupled with the inability to dissipate accumulated heat to the surrounding environment (Nybo, 2008). The compromised heat dissipation during exercise may be of greater detriment to team sport athletes as it has been reported that core temperature increases at quicker rates compared to continuous exercise for a comparable exercise intensity (Drust et al. 2000b; Ekblom et al. 1971; Kraning and Gonzalez, 1991). In conjunction with thermoregulatory stress, performance declines in the heat have been associated with increased circulatory strain (Gonzalez-Alonso and Calbet, 2003) and centrally-mediated behaviour modification (pacing strategies) in response to and in anticipation of increased thermal stress (Marino, 2004; Schlader et al. 2010).

The ability for a team sport athlete to lose heat that is accumulated during exercise is dependent on the temperature gradient between the skin and the external environment (Gagge et al. 1977). Mechanisms of heat dissipation and environmental conditions may potentially determine the extent to which the body is able to remove heat from the core and dissipate through the skin (Maxwell et al. 1996). During uncompensable warm environments, the gradient between heat loss and gain is compromised and heat gain is faster than the ability to dissipate heat (Brotherhood et al. 2008). The accumulation of internal heat stress is predominately evident through the measurement of the rise in core temperature. During fixed-paced exercise protocols, an accelerated rate of rise in core, skin and muscle temperatures are evident in the heat compared to thermo-neutral conditions (Galloway and Maughan, 1997; Gonzalez-Alonso et al. 1999; Kraning and Gonzalez, 1991). However, during self-paced exercise, similar physiological responses are often observed compared to cooler environments but with a reduction in exercise intensity in the heat (Marino, 2004). These observations have also been noted during intermittent-sprint exercise, with increased thermal stress proposed to negatively affect exercise performance (Drust et al. 2005; Maxwell et al. 1996; Mohr et al. 2003).
The aforementioned detrimental effects of high endogenous body temperatures on exercise performance can be further exacerbated by exposure to warm ambient conditions prior to exercise in the heat (pre-heating). Paradoxically, increased thermal load prior to exercise has also been shown to improve high-intensity exercise performance and therefore is employed by team sport athletes via passive and active means (Bishop, 2003). Some common temperature-related mechanisms for the (intentional) exposure to environmental heat include increased muscle, skin and core temperatures, decreased resistance to muscles and joints, speeding of metabolic reactions, and improved nerve conduction rates (Bishop, 2003). Therefore, team sport athletes engage in warm-up practices to increase muscle temperature and augment subsequent intermittent-sprint performance. For example, Lovell et al. (2011) reported that elevated muscle temperature compared to baseline values at half time of a soccer match via whole-body vibration and active warm-up significantly improved sprinting, jumping and dynamic strength performance during the second half of the match. Accordingly, there appears to be temperature-dependent effects on high-intensity exercise and intermittent-sprint performance; therefore applying pre-exercise passive heating prior to self-paced intermittent-sprint exercise may provide further insight into the mechanisms responsible for pacing regulation, specifically in relation to regulating thermal stress.

Due to the well-established literature highlighting that exercise in warm, humid conditions results in an increase in endogenous thermal load (Nybo, 2008), a common strategy that team sport athletes employ to counter the negative effects of exercise in the heat is the use of pre-cooling. Pre-cooling modalities include whole-body or body segments submerged into ice and/or cold water, application of ice vests and towels, ingestion of ice slushies, or exposure to cold air (Cheung and Robinson, 2004; Duffield and Marino, 2007; Minett et al. 2011; Siegel et al. 2011; Stanley et al. 2010). As outlined by Brück and Olschewski, (1987), the premise behind engaging in these maneuvers is for athletes to reduce core and skin temperatures prior to exercise and thus create a ‘heat sink’ in which they able to exercise at a higher intensity for a given period of time (exercise performance). While reducing an
athlete’s core temperature before exercise is beneficial to longer exercise durations, there may also be negative effects of decreasing muscle temperature, which may be a by-product of many of the aforementioned cooling practices (Kay et al. 1999). Accordingly, reductions in muscle temperature have been linked to reduced power output during maximal cycling efforts and reductions in sprint times (Sleivert et al. 2001). The contrasting effects of pre-cooling on endurance and sprint performance is of interest to team sports who are required to complete both modes within a given match.

The examination of pre-exercise thermal stress and thermoregulatory responses to exercise has been shown to be an effective method to investigate the models proposed to explain fatigue and exercise regulation (Cheung and Sleivert, 2004; Kay et al. 2001; Marino, 2004; Nybo, 2008; Todd et al. 2005). More traditional viewpoints suggest that peripheral perturbations including physiological changes to the cardiovascular system during exercise in the heat are detrimental to performance (Gonzalez and Calbet, 2003). Further, hyperthermia-induced changes to muscle metabolism and metabolite accumulation during the heat may directly affect the force generating capacity of the active musculature (Nybo, 2008). More recent models propose that the attainment of a higher core temperature has an effect on the CNS, resulting in a down-reduction in neural drive and exercise intensity in order to avoid this critically high temperature (Saboisky et al. 2003). Accordingly, exercise in the heat affects intermittent-sprint performance and creates a model to examine the temperature-dependent effects on the periphery in conjunction with the central regulation of exercise intensities to monitor such changes during self-paced intermittent-sprint exercise.

**Exercise Performance in the Heat**

*Interrittent-Sprint Performance*

The increased thermoregulatory and cardiovascular stresses associated with exercise in the heat have negative implications on intermittent-sprint performance due to excessive increases in core
temperature (Drust et al. 2005; Maxwell et al. 1996; Table 2.6). Field-based research has indicated core temperatures during rugby and soccer players increase to 38.5 – 39.3°C in neutral conditions (Edwards and Clark 2006; Meir et al. 2003) and peak at 39.6°C during soccer matches in warm conditions (Mohr et al. 2004). A study by Duffield et al. (2009) reported that during an elite AFL game in the heat (29.5 ± 1.3°C and 64.9 ± 16.7% relative humidity) mean peak core temperature was 39.3°C, which occurred towards the end of the first quarter and with several players reaching almost 40.0°C. Duffield and colleagues (2009) further highlight strong correlations between running velocity, moderate-intensity velocity and very-high intensity running with core temperature during respective quarters of the match. These findings suggest that thermal strain is evident during team sport exercise, with significant elevations in core temperature. Further, reductions in the exercise intensities during a match were associated with the increased thermal load, thus attempting to minimise further gains in heat stress. While field-based research has shown elevations in core temperature down regulate running velocities during a match, research in this area is still relatively sparse (Coutts et al. 2010; Duffield et al. 2009; Reilly et al. 2008). However, laboratory-based intermittent-sprint protocols in the heat further supports the existing field-based research in that team sport athletes suffer from thermal stress that negates exercise performance.

The laboratory-based intermittent-sprint protocols within the literature vary considerably, ranging from repeated sprints on a cycle ergometer to over-ground running of various exercise intensities. Laboratory-based protocols that have examined the effect of environmental heat on sprint performance without the inclusion of self-paced exercise between sprints have reported conflicting results, with differences possibly due to the variation in study designs and training status of the subjects. Falk et al. (1998) examined a series of 5 x 15 s cycling sprint bouts separated by 30 s of active recovery between the bouts, which was then repeated 60 min later in hot and neutral conditions. Results indicate peak power during the initial series was higher in the hot compared to neutral conditions, and despite higher core temperatures, minimal differences were evident between
conditions during sprints completed 60 min later. Further, Drust et al. (2005) completed a 40 min intermittent-sprint protocol on a cycle ergometer in normal and hyperthermic conditions that consisted of work (sprint) to rest ratio of 1:1 with sprint durations of 15 s. Repeat sprint performance was reduced during the hyperthermic condition and also resulted in significant elevations in core and muscle temperatures, HR and RPE. Collectively, initial sprint performance may be maintained or improved during hyperthermic conditions; however repeated sprints interspersed with passive recovery are negatively affected by environmental heat (Table 2.7).

Extending on the above mentioned repeat-sprint protocols, intermittent-sprint protocols that include exercise of various intensities between maximal sprints have also shown performance to be negated during hyperthermic conditions. Maxwell et al. (1996) examined warm-up and exercise in cool and warm conditions on intermittent running performance until exhaustion. The findings indicate that time to exhaustion was reduced when exercising in warm conditions, with significant elevations in core and mean skin temperatures. However, this protocol assessed intensities above \( \dot{V}O_2\text{max} \), therefore the mean incremental test time to exhaustion was 140 – 151 s duration. Furthermore, Morris et al. (2000) utilised a modified 75 min LIST followed by a series of 60 s runs at 100% \( \dot{V}O_2\text{max} \) with 60 s rest, performed until exhaustion in hot or moderate environmental conditions. During the LIST, less distance was covered and sprints were slower during the hot condition, and there was a significant correlation between rate of rise of core temperature and distance covered (\( r = -0.93 \text{ to } -0.94; P < 0.01 \)).

Morris et al. (2005) again used a modified LIST; however, in this study the exercise: rest pattern was continued until volitional exhaustion either in hot or moderate environmental conditions. Similar to their previous findings Morris and colleagues (2005) reported slower sprint times and less distance covered in the heat, with increased heart rate, core and muscle temperatures, muscle glycogen utilization, and blood lactate, glucose and catecholamine concentrations. Collectively, these studies indicate that intermittent-sprint performance is compromised in the heat compared to thermo-neutral conditions (Table 2.6). However, despite the findings that exercise in the heat increased thermal strain
and reduced sprint performance, these activity patterns (other than maximal sprints) were externally controlled which is not indicative of team sport exercise.

The effects of hyperthermia on intermittent-sprint performance are further highlighted by increases in pre-exercise core temperature due to external methods of heating. Bishop and Maxwell (2009) examined the effects of no warm-up, 10 min warm-up or a 20 min warm up on intermittent-sprint test performance including 2 repeated-sprint bouts (5 x 2s sprints separated by 20s). No significant differences in mean work during the intermittent-sprint test were present, however higher core temperatures and less work was completed during the repeat-sprint test following a 20 min warm-up. The authors concluded that athletes should avoid elevations in pre-exercise core temperature due to excessive warm-up durations to minimise the negative effects of hyperthermia on repeat-sprint performance. Similar to repeated sprints, temperature dependent effects of passive heating also reduce intermittent running to exhaustion. Gregson and colleagues (2005) suggest a decline in performance was associated with increased heat storage and rectal temperatures following pre-exercise warming conditions compared to the control condition. While there are some issues regarding the ecological validity to team sports of some of the intermittent-sprint protocols used to examine the effects of pre-heating, data supports the notion that intermittent-sprint exercise performance in the heat is potentially negated due to elevations in pre-exercise temperature and the accelerated rise in body temperature once exercise commences.

While passive heating resulting in high endogenous thermal loads negates intermittent-sprint performance, the resultant increase in muscle temperature is reported to have performance benefits to maximal exercise of short durations (Bishop, 2003; Gray et al. 2002; Racinais et al. 2007). Brown et al. (2008) examined active (10 min at 70% \( \dot{V}O_{2\text{max}} \)) and passive (water submersion) warm-ups on 10 x 6 s maximal sprints every 40 s on a non-motorised treadmill. Results indicated that both warm-ups improved peak and mean sprint times compared to no warm-up; however, no differences were noted
between the active and passive warm-up routines. Based on these findings, Brown et al. (2008) suggested temperature and non-temperature dependent mechanisms are responsible for performance improvements, primarily the maintenance of neuromuscular facilitation and improved efficiency of the contractile processes. Further, Gray et al. (2002) examined the effect of elevated muscle temperature via passive heating (water immersion and electric blankets) on 6 s sprint performance on a cycle ergometer. Maximal power output and pedal rate were greater following passive heating, possibly via an elevated rate of anaerobic ATP turnover and muscle fibre conduction velocity (Gray et al. 2002). These findings are supported by Racinais et al. (2009) who reported 7 s cycling sprint performance was reduced following 30 min submerged in an ice bath, whilst 30 min passive heating improved power output. It is believed the performance improvements were attributed to increases in muscle temperature rather than core temperature. Furthermore, Lacerda et al. (2006) reported similar findings in that passive heating improves 30 s cycling sprint performance without changes to core temperature or heart rate. The authors suggested that changes in muscle temperature were the likely cause of performance improvements following heating rather than non-temperature factors. Collectively, these studies highlight that increases in muscle temperature via passive heating improve subsequent one-off maximal sprint performance due to increased ATP turnover and muscle contraction velocities compared to control and cooling conditions. Such findings are important for team sport athletes who complete bouts of maximal intensity exercise; although, when sprints are repeated over a prolonged duration, performance benefits of passive heating may result in ergolytic responses due to concomitant elevations in core temperature.

In contrast to passive heating, previous research examining the effect of pre-cooling on team sport exercise has reported that with sufficient cooling temperature and duration, pre-cooling provides ergogenic effects on prolonged, intermittent-sprint exercise performance (Castle et al. 2006; Duffield et al. 2009; Duffield and Marino 2007; Table 2.7). Drust et al. (2000a) examined pre-cooling via a cold shower (26°C) for 60 min in thermo-neutral conditions, no pre-cooling in neutral conditions, and
no pre-cooling in a heated environment. The authors reported a significantly lower rectal temperature during exercise following pre-cooling compared to the heated condition. However, minimal physiological differences were observed during the 90 min soccer-specific exercise protocol, including heart rate, \( \dot{\text{VO}}_2 \), \( \dot{\text{V}}_\text{E} \), RPE, and blood lactate, glucose and FFA utilisation between all conditions. Unfortunately, while the exercise protocol simulated the demands of a soccer match and examined sprint performance, the fixed-paced exercise during the sub-maximal efforts did not allow for intermodal pacing strategies to be examined.

Similar to Drust et al. (2000a), most intermittent-sprint protocols allow the examination of the effect of pre-cooling on maximal sprint performance. Duffield et al. (2003) reported pre-cooling with an ice-vest for 5 min prior to exercise and during the recovery periods did not improve work completed or power output during an 80 min intermittent-sprint cycling protocol. Duffield and colleagues (2003) reported that the cooling methodology was not sufficient enough to create differences in core and skin temperatures, heart rate, blood lactate, sweat rate, RPE or thermal stress; therefore minimising the effect on performance. However, using longer cooling durations, Castle et al. (2006) examined 20 min of no cooling, pre-cooling via an ice vest, cold water immersion, and ice packs on the upper legs on a 40 min intermittent-sprint cycling performance. A reduction in peak power output (PPO) was observed during the no cooling condition, while a ~4\% (P< 0.05) increase in PPO was evident following application of the ice packs. These performance findings were associated with a faster rate of heat strain during no cooling compared to lower body cooling methods (excluding ice vests) while the use of ice packs and water immersion blunted increases in muscle temperature. Although this study did not specifically utilise a self-paced running protocol indicative of team sports, the findings from this study may suggest that lower body and whole body cooling is advantageous to intermittent-sprint exercise due to reduced thermal strain.
Unlike previous intermittent-sprint protocols, Duffield and Marino (2007) examined self-paced sub-maximal exercise performance in conjunction with sprint performance following either 15 min whole body submersion in an ice bath, application of an ice-vest, or control. The authors reported minimal differences between conditions for 15 m sprint performance during a 60 min free-paced intermittent-sprint running protocol; however, following submersion in the ice-bath, participants covered more distance during the self-paced hard running efforts. These performance improvements were in conjunction with lower core and skin temperatures, heat storage and RPE for a vast portion of the protocol. Further, Minett et al. (2011) examined mixed-method, whole-body cooling, and no cooling prior to a comparable intermittent-sprint exercise protocol. Whole-body pre-cooling maintained 15 m sprint performance and distance covered during hard running and jogging bouts throughout a 85 min self-paced protocol compared to control, cooling of head, and cooling of head and hands combined. Accordingly, pre-cooling maintains intermittent-sprint performance in the heat, particularly during higher-intensity efforts. Furthermore, it seems that the magnitude of effect that pre-cooling has on performance is dependent upon the total surface area of the body that is being cooled. Further elaboration on the studies involving pre-cooling and resultant effects on intermittent-sprint performance are presented in Table 2.7.
Table 2.6: Summary of Studies Examining the Effect of Environmental Heat on Repeated-Sprint and Intermittent-Sprint Exercise Performance

<table>
<thead>
<tr>
<th>Author</th>
<th>n=</th>
<th>Ambient Conditions</th>
<th>Exercise Protocol</th>
<th>Performance Outcomes in the Heat</th>
<th>Physiological Responses to Exercise in the Heat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ball et al. 1999</td>
<td>7</td>
<td>Hot (30.1°C) vs Neutral (18.7°C)</td>
<td>2 x 30 s sprints on cycle ergometer (4 min passive recovery)</td>
<td>↑ Mean and Peak PO during both sprints</td>
<td>↔ La⁻</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Faster rate of fatigue</td>
<td></td>
</tr>
<tr>
<td>Drust et al. 2005</td>
<td>7</td>
<td>Hot (40°C) vs Neutral (20°C)</td>
<td>40 min intermittent-sprint protocol on cycle ergometer followed by 5 x 15 s sprint (15 s recovery)</td>
<td>↔ PO during sprint 1</td>
<td>↑ T&lt;sub&gt;core&lt;/sub&gt;, T&lt;sub&gt;mus&lt;/sub&gt;, HR, norepinephrine concentration</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>↓ PO during sprint 2-5</td>
<td>↑ RPE</td>
</tr>
<tr>
<td>Duffield et al. 2009</td>
<td>10</td>
<td>Hot (29°C dry; 27°C WBGT)</td>
<td>Elite AFL match</td>
<td>T&lt;sub&gt;core&lt;/sub&gt; correlated with 1&lt;sup&gt;st&lt;/sup&gt; qtr HIRV and MIV, 2&lt;sup&gt;nd&lt;/sup&gt; qtr MIV, 4&lt;sup&gt;th&lt;/sup&gt; qtr VHIR and MIV</td>
<td>Peak T&lt;sub&gt;core&lt;/sub&gt; of 39.3 ± 0.7°C, Total rise in T&lt;sub&gt;core&lt;/sub&gt; of 2.1 ± 0.7°C</td>
</tr>
<tr>
<td>Falk et al. 1998</td>
<td>11</td>
<td>Hot (35°C) vs Neutral (22°C)</td>
<td>Series 1: 5 x 15 s cycling sprints separated by 30s active recovery and repeated 60 min later in hot or neutral conditions (series 2)</td>
<td>↑ Peak Power during series 1</td>
<td>↑ T&lt;sub&gt;core&lt;/sub&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>↔ Peak Power during series 2</td>
<td>↔ La⁻, HR, VO&lt;sub&gt;2&lt;/sub&gt;, electrolyte concentration, osmolality</td>
</tr>
<tr>
<td>Maxwell et al. 1996</td>
<td>12</td>
<td>WU and ex in heat (HH); WU and ex in cool (CC); WU in cool &amp; ex in heat (CH)</td>
<td>Intermittent running to exhaustion (20 s runs at increasing intensity with 100 s passive recovery)</td>
<td>↓ TTE (HH: 140 ± 5s; CC: 151 ± 4s; CH: 144 ± 5 s)</td>
<td>↑ SwR during HH</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>↑ T&lt;sub&gt;core&lt;/sub&gt;, T&lt;sub&gt;skin&lt;/sub&gt; during HH</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>↔ La⁻, ammonia and plasma volume during HH</td>
</tr>
<tr>
<td>Morris et al. 2000</td>
<td>16</td>
<td>Hot (30°C) vs Neutral (16°C)</td>
<td>Part A: 75 min LIST Part B: 60s runs at 100% VO&lt;sub&gt;2&lt;/sub&gt;max (60s recovery) until fatigue</td>
<td>Part A: 25% less work completed Part B: Slower sprint times</td>
<td>↑ T&lt;sub&gt;core&lt;/sub&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>↔ La⁻ and ammonia</td>
</tr>
<tr>
<td>Morris et al. 2005</td>
<td>9</td>
<td>Hot (33°C) vs Neutral (17°C)</td>
<td>Part A: Modified LIST for 14.8 ± 0.1 min followed by a 3min rest, then continued exercise until exhaustion</td>
<td>↓ less distance covered Slow(er) sprint times</td>
<td>↑ HR, T&lt;sub&gt;core&lt;/sub&gt;, T&lt;sub&gt;mus&lt;/sub&gt;, La⁻, Glu, catecholamines</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>↑ muscle glycogen utilization</td>
</tr>
</tbody>
</table>

AFL = Australian Football League; Ex = Exercise; HIRV = High Intensity Running Velocity; LIST= Loughborough Intermittent Shuttle Test; MIV = Moderate Intensity Velocity; PO = Power output; T<sub>core</sub> = core temperature; T<sub>mus</sub> = muscle temperature T<sub>skin</sub> = skin temperature; TTE = Time to Exhaustion; VHIR = Very High Intensity Running; WBGT= Wet Bulb Globe Temperature; WU = Warm up
Table 2.7: Summary of Studies Examining the Effect of Pre-Cooling and Passive Heating on Intermittent-Sprint Exercise Performance.

<table>
<thead>
<tr>
<th>Author</th>
<th>n=</th>
<th>Ambient Temp</th>
<th>Cooling/Heating Protocol</th>
<th>Exercise Protocol</th>
<th>Performance Outcomes</th>
<th>Physiological and Perceptual Responses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Castle et al. 2006</td>
<td>12</td>
<td>34°C</td>
<td>20 min of No cooling (CONT); Ice-vest (Vest); CWI; ice packs on upper legs (Packs)</td>
<td>40 min ISE including repeated bouts of 10s passive rest, 5s sprint, 105s active recovery</td>
<td>↓ PPO during CONT ↑ PPO during Packs (~4%)</td>
<td>↓ rate of heat strain for CWI and Packs ↓ T_{mus}, T_{core} and T_{skin} for CWI and Packs ↓ Thermal Strain and PSI following cooling ↓ T_{core}, T_{mus}, Thermal sensation following cooling</td>
</tr>
<tr>
<td>Clarke et al. 2010</td>
<td>12</td>
<td>30.5°C</td>
<td>Pre-cool + CHO during exercise (CHOc); Pre-cool + placebo during exercise (PLAc); No-cooling + CHO (CHO); no cooling + placebo (PLA)</td>
<td>90 min soccer-specific intermittent protocol</td>
<td>↑ self-chosen pace during CHOc compared to CHO and PLAc ↑ HIA in CHOc and CHO compared to PLAc ↑ mental concentration during CHOc compared to PLA</td>
<td></td>
</tr>
<tr>
<td>Drust et al. 2000a</td>
<td>6</td>
<td>Hot (26°C) vs Neutral (20°C)</td>
<td>60 min of no cooling (CONT); cold shower (26°C)</td>
<td>90 min soccer-specific ISE protocol on a non-motorised treadmill</td>
<td>Sprint performance was not reported</td>
<td>↑ T_{core} following CONT ↔ VO_{2}, HR, V_{\dot{E}}, RPE, plasma La, Glu, FFA concentrations</td>
</tr>
<tr>
<td>Duffield et al. 2003</td>
<td>7</td>
<td>32°C</td>
<td>5 min of no-cooling (CONT); Ice-Vest (Vest) before and during recovery</td>
<td>80 min intermittent sprint cycling</td>
<td>↔ work done or PO during sprint performance</td>
<td>↑ chest T_{skin}, thermal discomfort, rating of thirst ↔ HR, La, T_{core}, mean T_{skin}, RPE, SwR, ratings of fatigue</td>
</tr>
<tr>
<td>Duffield and Marino 2007</td>
<td>9</td>
<td>32°C</td>
<td>15 min of no cooling (CONT); Ice-bath (CWI) plus vest during WU and HT; Ice-vest (Vest)</td>
<td>60 min ISE including 15 m sprint every min and free-paced, sub-max efforts (hard run, jog, walk)</td>
<td>↔ sprint times and %decline ↓ distance covered during sub-max efforts in CONT ↓ distance covered during hard run efforts</td>
<td>↑ T_{core}, T_{skin}, HR, SwR ↑ Thermal Comfort ↔ blood La, pH, K or Na+</td>
</tr>
<tr>
<td>Minett et al. 2011</td>
<td>10</td>
<td>33°C</td>
<td>20 min whole-body (WB); Head + Hand (HH); Head (H); No cooling (CONT)</td>
<td>85 min free-paced intermittent sprint protocol</td>
<td>↓ % decline and faster sprint times after WB ↓ distance covered in CONT</td>
<td>↓ T_{core} and T_{skin} during WB and HH ↓ HR during WB compared to CONT ↓ RPE and Thermal stress during WB and H</td>
</tr>
<tr>
<td>Passive Heating</td>
<td>Brown et al. 2008</td>
<td>10</td>
<td>20°C</td>
<td>~10 min No warm-up (CONT); Active pre-heating (AH); Passive pre-heating (PH)</td>
<td>10 x 6 s sprints with 34 s recovery</td>
<td>↑ peak mean 1s maximum speed following PH and AH ↔ % decline in fatigue</td>
</tr>
<tr>
<td>----------------</td>
<td>------------------</td>
<td>----</td>
<td>------</td>
<td>----------------------------------------------------------------</td>
<td>----------------------------------</td>
<td>--------------------------------------------------------------------------------</td>
</tr>
<tr>
<td></td>
<td>Gregson et al. 2005</td>
<td>6</td>
<td>22°C</td>
<td>Active pre-heating (AH); passive pre-heating (PH); no heating (CONT)</td>
<td>30 s bouts at 90%VOC$_{\text{max}}$ with 30 s passive recovery to exhaustion</td>
<td>TTE different between all conditions (CONT &gt; AH &gt; PH)</td>
</tr>
<tr>
<td></td>
<td>Sleivert et al. 2001</td>
<td>9</td>
<td>33°C</td>
<td>No cooling (CONT); Pre-cool torso and thigh (LC); pre-cool torso with thighs warm (LW)</td>
<td>45 s high-intensity exercise</td>
<td>↓ peak and mean PO during LC compared to CONT ↔ peak and mean PO during LW</td>
</tr>
</tbody>
</table>

AH = active pre-heating; CHO = carbohydrate; CHOc = pre-cooling + CHO intake; CONT = Control; CWI = cold water immersion; FFA = Free Fatty Acids; Glu = Glucose; H = Head; HH = Head + Hand; HR = heart rate; HT = half-time; ISE = Intermittent-Sprint Exercise; K$^+$ = potassium; La$^-$ = lactate; LC = Lower-body cooling; LW = Lower-body warming; Na$^+$ = sodium; Packs = Ice-Packs; PH = passive pre-heating; PLA = placebo; PLAc = pre-cooling + placebo CHO intake; PPO = Peak Power Output; PSI = Physiological Strain Index; SwR = sweat rate; T$_{core}$ = core temperature; T$_{\text{max}}$ = muscle temperature; T$_{skim}$ = skin temperature; $\dot{V}_E$ = minute ventilation; Vest = Ice-Vest; VOC = oxygen consumption; VOC$_{\text{max}}$ = maximal aerobic capacity; WB = whole body; WU = warm-up;
Self-Paced Exercise Performance

A component of team sport exercise that is not commonly incorporated into laboratory-based intermittent-sprint protocols is self-paced exercise. The examination of pacing strategies during continuous exercise protocols may provide insight into the regulation of pacing in response to altered thermal stress. As outlined in Table 2.8, previous literature has indicated that cycling time trial performance and pacing are negatively affected in the heat (Kay et al. 2001; Marino et al. 2001; Tatterson et al. 2000; Tucker et al. 2004; Tucker et al. 2006). It has been suggested that the earlier reductions in power output are in response to increased core temperature (Tatterson et al. 2000) and in anticipation of attaining a critically high body temperature (Tucker et al. 2004). More specifically, Tatterson et al. (2000) reported a significant (6.5%) decline in power output during a 30 min cycling time trial in the heat compared to moderate conditions; despite similar rectal temperatures throughout the protocol. Similarly, Tucker et al. (2004) compared two 20 km cycling time trials in either hot (35°C) or cool (15°C) conditions, and reported power output was significantly reduced in the final 20% (following 23min of cycling) of the trial duration in the hot condition. However, iEMG became reduced substantially earlier (10 km) despite no difference between conditions for rectal temperature, heart rate or RPE.

These aforementioned results suggest an anticipatory reduction in pacing strategy and muscle recruitment to maintain thermoregulatory strain and RPE during self-paced time trials. These findings are further supported by later work by Tucker and colleagues (2006) who examined hot, normal, and cool conditions on self-paced cycling exercise; however, power output was manipulated by the participant in order to maintain an RPE of 16. While power output declined over time to maintain the set RPE in all three conditions, a greater decline was evident during the hot condition despite heat storage only being significantly higher in the first 4 min of the trial. Collectively, these studies suggest that due to afferent feedback regarding heat storage, there is an anticipatory regulation of thermoregulatory strain, predominately via absolute changes or the rate of rise in core temperature.
To counter the negative effects of hyperthermia on self-paced exercise performance, pre-cooling allows the selection of higher pacing strategies for a given period of time for the same physiological and perceptual responses compared to a control condition. As previously mentioned, the manipulation of exercise intensity and pacing strategies may be crucial for a team-sport athletes’ performance during a match; however, the majority of self-paced exercise includes continuous cycling protocols. Similar to self-paced intermittent-sprint exercise, these continuous protocols have reported the adoption of higher exercise intensities for a given time trial following pre-cooling (Duffield et al. 2010; Kay et al. 1999; Quod et al. 2008; Schlader et al. 2011a). Quod et al. (2008) examined the effects of no-cooling, cooling via ice jacket for 40 min, or ice-bath for 30 min following by a cooling jacket for 40 min on a 40min cycling time trial performance in warm ambient conditions. Pre time-trial rectal temperature was lower in the combined cooling condition and time trial performance was faster compared to the no cooling. Booth et al. (1997) similarly reported a greater distance covered during a 30 min treadmill run in the heat following whole-body water submersion for 60 min at ~23°C. Performance improvements were associated with lower rectal, skin and mean body temperatures and heart rate; suggesting an enhanced rate of heat storage and decreased thermal strain were responsible for the performance differences. In support of these findings, Duffield et al. (2010) reported that lower-body pre-cooling improved 40 min cycling time trial performance in the heat, with lower core, skin and body temperatures up to the final 20 min of the exercise protocol. Notably, performance differences became most apparent in the latter stages of the protocol, when physiological differences were no longer apparent. These data suggest that similar to ISE, pre-cooling improves pacing strategies during continuous self-paced exercise with the adoption of higher intensity pacing strategies (Table 2.8).

Similar to core temperature, changes in skin temperature, independent of changes in core temperature play a crucial role in the regulation of pacing strategies during self-paced exercise. Kay et al. (1999) examined 30 min self-paced cycling trial performance following pre-cooling of the skin via water
immersion, without differences in pre-exercise core temperature. Participants cycled significantly further following pre-cooling and recorded lower heat storage and sweat rates, thus suggesting skin cooling is sufficient for performance improvements with the absence of changes to core temperature. This study is further supported by Schlader et al. (2011b) who reported significantly greater work was completed following skin cooling (without changes to core temperature) due to a higher initial power output. Collectively, these studies suggest that skin temperature associated with pre-cooling may also provide valuable input for behaviour modification and the selection of higher intensity pacing strategies, void of core temperature manipulation.

In summary, these studies indicate that prolonged exercise in the heat is negated compared to exercise in thermo-neutral conditions. The performance decrements prevalent during both continuous and intermittent-sprint protocols have mainly been attributed to the accelerated increase in core temperature due to compromised ability to dissipate endogenous heat production to the surrounding environment. Accordingly, pre-cooling has been shown to alleviate some of these physiological perturbations associated with exercise in the heat, but at a cost to maximal sprint performance due to reductions in muscle temperature. On the contrary, passive heating may augment sprint performance, however once sprints are repeated over a prolonged period of time, the concomitant elevations in core temperature reduce endurance capacity. Accordingly, the successive section of the literature review will discuss the proposed mechanisms related to these performance changes in the heat and how changing pre-exercise thermal strain (via pre-cooling and heating) may influence pacing strategies of specific modes during self-paced intermittent-sprint performance in team sport athletes.
Table 2.8: Summary of Studies Examining the Effect of Environmental Heat on Self-Paced Exercise Performance.

<table>
<thead>
<tr>
<th>Author</th>
<th>n=</th>
<th>Ambient Conditions</th>
<th>Exercise Protocol</th>
<th>Performance Outcomes in the Heat</th>
<th>Physiological and Perceptual Responses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marino et al. 2004</td>
<td>12</td>
<td>Hot (35°C) vs Cool (15°C)</td>
<td>30 min treadmill running at 70% $\dot{V}O_{2\text{max}}$ followed by 8 km running time trial</td>
<td>↓ time trial performance</td>
<td>↑ $T_{\text{core}}$, $T_{\text{skin}}$, SwR</td>
</tr>
<tr>
<td>Tatterson et al. 2000</td>
<td>11</td>
<td>Hot (32°C) vs Neutral (23°C)</td>
<td>30 min cycling time trial</td>
<td>↓ PO (6.5%) ↓ PO during last 10 min ↔ PO during initial 10 min</td>
<td>↑ mean $T_{\text{skin}}$ and SwR ↔ $T_{\text{core}}$ ↑ blood La$\text{-}$ and ↓ pH during first and final 10min of exercise</td>
</tr>
<tr>
<td>Tucker et al. 2004</td>
<td>10</td>
<td>Hot (35°C) vs Cool (15°C)</td>
<td>20 km cycling time trial</td>
<td>↓ PO in final stages of protocol ↓ $i\text{EMG}$ early in the protocol</td>
<td>↔ $T_{\text{core}}$, HR, RPE</td>
</tr>
<tr>
<td>Tucker et al. 2006</td>
<td>8</td>
<td>Hot (35°C) vs Neutral (25°C) vs Cool (15°C)</td>
<td>Cycling trial adjusting PO to maintain an RPE of 16</td>
<td>↓ PO Hot compared to Neutral and Cool conditions</td>
<td>↑ rate of heat storage in first 4 min due to ↑ $T_{\text{skin}}$ ↔ $T_{\text{core}}$ ↑ Thermal comfort</td>
</tr>
</tbody>
</table>

HR = heart rate; $i\text{EMG}$ = integrated electromyography; La$\text{-}$ = lactate; PO = power output; RPE = rating of perceived exertion; SwR = sweat rate; $T_{\text{core}}$ = core temperature; $T_{\text{skin}}$ = skin temperature; $\dot{V}O_{2\text{max}}$ = maximal aerobic capacity
Mechanisms Related to Exercise Performance in Hyperthermic Conditions

Thermoregulatory and Cardiovascular Stress

The ability to effectively thermoregulate at rest and regulate core temperature within homeostatic limits involves various processes (convection, conduction, evaporation and radiation) to maintain the balance between heat accumulation and dissipation (Marino, 2008). The integration of these systems becomes more important once an athlete commences exercise, as the accumulation of endogenous and exogenous heat production occurs. Mechanisms associated with heat loss during exercise are dependent on the temperature gradient between the skin and the external environment (Kraning and Gonzalez, 1991). Therefore, exercise in the heat, compared to thermo-neutral conditions, compromises the temperature gradient between the body and ambient surroundings and reduces one’s ability to dissipate internal heat to the external environment (Wendt et al. 2007). The inability to dissipate heat to the environment leads to a quicker rate of rise in thermal stress for a given exercise intensity. This increase in thermal stress is observed with elevations in core, skin and muscle temperatures (Sawka et al. 1996), calculations of mean body temperature (Ramanathan, 1964), physiological strain index (PSI; Moran et al. 1988) and percentage heat storage accumulation (Tucker et al. 2006). Increases in such markers of thermal stress have been linked to reductions in exercise time to exhaustion (Galloway and Maughan, 1997), time trial performance (Altareki et al. 2009), and intermittent-sprint performance (Maxwell et al. 1996). Accordingly, the completion of team sports in hot conditions may compromise an athlete’s ability to dissipate heat to the external environment, leading to an accelerated rate of rise in core, skin and muscle temperatures compared to moderate environmental conditions.

Increases in thermal stress associated with exercise in the heat have concomitant and deleterious effects on the cardiovascular system and may be further exacerbated by pre-exercise heating. Independent of the presence of environmental heat; the commencement of exercise results in an increase in cardiac output via increases in stroke volume and heart rate (Gonzalez-Alonso et al. 2008).
The primary aim of this increase in cardiac output is to increase blood flow to the active musculature (Astrand et al. 1964; Kovacs et al. 2009). Concomitantly, as a result of exogenous and endogenous heat production and increases in core, muscle and skin temperature, there is a greater redistribution of blood flow to the skin to assist in heat dissipation (Gonzalez-Alonso et al. 2008). However, the redistribution of blood flow to the skin for heat loss and the increase in evaporative cooling reduces blood volume, venous return and MAP (Nielsen et al. 1993). Accordingly, in order for cardiac output to be maintained in the presence of a reduced stroke volume, a compensatory increase in heart rate becomes evident, otherwise known as cardiovascular drift (Trinity et al. 2010). As exercise in the heat continues there becomes an increased competition for blood flow between the skin (for heat dissipation), the active musculature (for aerobic metabolism), the heart (to maintain central blood volume) and cerebral blood flow (Gonzalez-Alonso et al. 2008). Accordingly, vasoconstriction at the periphery to maintain central blood volume eventually reduces evaporative heat loss and further accelerates heat accumulation. Further, reduced blood flow to the active musculature has been shown to affect fuel utilisation and metabolite accumulation (Gonzalez-Alonso and Calbet, 2003; Mortensen et al. 2005). These negative effects of exercise in the heat on cardiovascular function are evident during intermittent-sprint exercise. Accordingly, it has been reported team sport athletes lose approximately 1.2 ± 0.5 kg (0.5 – 2.6 kg) during small-sided matches (Sherriffs et al. 2005) and 1.0 – 1.4 kg during intermittent-sprint exercise in the heat (Skein and Duffield, 2010). These reductions in nude mass may be an indication of an elevated sweat rates (Edwards et al. 2007) and elevated HR responses during repeat-sprint exercise in the heat compared to thermo-neutral conditions (Drust et al. 2005). Collectively, these studies indicate increased cardiovascular stress during exercise the heat may potentially lead to performance decrements due to compromised blood flow, altered muscle metabolism and reduced cardiac output.

During self-paced exercise, minimal differences in physiological variables such as heart rate, and core and skin temperatures are often observed between environmental conditions (Tucker et al. 2004,
2006). The lack of difference is due to the nature of the exercise in which athletes are able to regulate pacing strategies in order to govern the rate of rise in thermal and cardiovascular stress (Tucker et al. 2004). Within these exercise settings, cardiovascular stress is noted with a decline in workload in order to maintain a given physiological stress (Tucker and Noakes, 2009). This notion has been supported with several studies highlighting that self-paced exercise in the heat is significantly reduced without differences in cardiovascular responses during exercise (Crewe et al. 2008; Tucker et al. 2006). Schlader et al. (2011b) examined the effects of exercise modality on body temperature regulation in the heat and reported no difference in circulatory responses between modalities. Accordingly, the authors examined fixed-paced exercise at 70% \( \dot{V}O_{2\text{max}} \) to exhaustion compared to self-paced exercise of the same amount of work, but power output was freely adjustable. Findings suggested exercise time to exhaustion was longer and power output and RPE were reduced during the self-paced trial, while core temperatures finished at similar values (39.4 vs 39.1°C). Further, cardiac output, stroke volume, and HR did not differ between conditions. These findings indicate that pacing strategies during self-paced exercise are a compensatory protection of thermoregulatory stress, rather than circulatory stress (Marino, 2004; Schlader et al. 2011b).

In contrast to previous findings, Périard et al. (2010) examined 40 km self-paced cycling time trial performance in hot (35°C) versus moderate (20°C) conditions. Data indicated that time trial duration was significantly reduced in the heat, which included a reduction in power output from 20 min onwards and a significantly higher final rectal temperature (39.8 ± 0.3 vs 38.9 ± 0.2°C). Further, mean skin temperature, heart rate and skin blood flow were higher in the heat; while stroke volume, cardiac output, and MAP were reduced. The findings suggest that rises in cardiovascular stress reduce power output, peak \( \dot{V}O_2 \) and pacing strategies during self-paced exercise in the heat, rather than the previously suggested anticipatory reduction in musculature recruitment and power output. Marino (2011) however provides an alternative interpretation of Périard and colleagues’ (2010) data and draws attention to the increase in power output in the final data collection point compared to the
penultimate point in both conditions. Interestingly, a greater increase was evident in the hot condition, suggesting an anticipatory regulation of exercise intensity was present, and subjects did not give a maximal effort during the first data collection point, but only did so at the end. Finally, Marino (2011) contradicts the notion proposed by Périard et al. (2010) that the similar RPE and HR responses indicate a similar level of motor outflow/central neural drive in both tests, considering power output values were different between conditions for the vast majority of the protocol. In summary, it appears that changes to cardiovascular stress associated with exercise in the heat can compromise performance; however, when exercise is self-paced, the CNS regulates such cardiovascular responses via changes in pacing strategies and muscle recruitment to minimise substantial disruptions in homeostasis that could be detrimental to the athlete’s health (Lambert et al. 2005; Marino, 2004).

Effects of Hyperthermia on Muscle Metabolism

Hargreaves & Febbraio (1998) have previously suggested that metabolic and cellular processes within the contracting muscle are compromised by hyperthermia and thus affect skeletal muscle function. The increased muscle temperature may induce structural and functional alterations in various proteins that are involved in electrolyte distribution across the sarcolemma, calcium release and reuptake by the sarcoplasmic reticulum, actin-myosin interactions and mitochondrial respiration (Hargreaves & Febbraio, 1998). Further, an elevated environmental temperature may create an increased reliance on CHO utilisation, which has been demonstrated by increased CHO oxidation (Febbraio et al. 1994; Jentjens et al. 2002), accelerated glycogenolysis and lactate accumulation, and elevated hepatic glucose output without changes to glucose uptake, leading to hyperglycemia (Febbraio et al. 1998; Hargreaves & Febbraio, 1998). These suggestions are supported by Drust et al. (2005) reporting muscle glycogen utilisation was greater during an intermittent-sprint protocol in hot conditions compared to a (thermo-neutral) control condition. It was proposed the enhanced utilisation could have resulted in inadequate energy provision due to the effect on glycolytic rates (Drust et al. 2005). Despite these changes in glycolysis during repeat sprint work in the heat, Drust et al. (2005)
concludes simultaneous elevations in core and muscle temperatures impaired the ability to complete high intensity, intermittent exercise due to the effect on the function of the CNS. Furthermore, it is evident excessive elevations in core temperature can impair endurance performance before muscle glycogen stores are depleted; therefore implicating that an increase in core temperature is more likely to limit intermittent sprint performance when performed in the heat (Reilly et al., 2006).

**Attainment of Critical-Limiting Temperature**

Recent studies have suggested that exercise performance declines are due to the attainment of a critical core temperature of approximately 39.5 – 40.0°C (Gonzalez-Alonso et al. 1999). Further, attainment of the critically limiting temperature reduces central drive to the active musculature and thus fatigue becomes evident (Nielsen et al. 2001; Nybo, 2008). This notion of a critically limiting temperature has been supported by several studies reporting that exercise terminates at a consistent temperature despite alterations in pre-exercise core temperature (Gonzalez-Alonso et al. 1999) and environmental conditions (Febbraio et al. 1994; Parkin et al. 1997). Gonzalez-Alonso et al. (1999) reported that high internal temperatures, due to exercise in uncompensable hot environments, reduced time to exhaustion and was related to the initial core temperature and ensuing rate of heat storage. During this study, participants’ pre-exercise core temperature was altered (35.9 ± 0.2, 37.4 ± 0.1, or 38.2 ± 0.1°C); however, all participants reached exhaustion at the same core temperature (40.1 - 40.2°C). Moreover, a 10 day heat acclimation study by Nielsen et al. (1993) showed core temperature at exhaustion was not different between pre (39.8 ± 0.13°C) and post acclimation (39.7 ± 0.15°C), although following acclimation participants were able to increase time to exhaustion from 48 min to 80 min. The difference in time to exhaustion with the same core temperature supports the notion that performance declines are attributed to high body temperature and performance improvements can be achieved by slowing the rate of rise of thermal stress until attaining a critically high temperature.
The reasoning for fatigue when an athlete reaches the critical limiting temperature has been related to the reduced central drive (Nybo, 2008). Nielsen et al. (1990) reported reduced time to exhaustion in the heat was due to impaired mental functioning and core temperatures at ~40°C reducing motivation and neural drive to skeletal muscle. Further, Nybo and Nielsen (2001) examined the effects of exercise at 60% of $\dot{V}O_2_{\text{max}}$ until exhaustion in warm and neutral conditions. Results indicated that time to exhaustion (50 ± 3 min) during the hot condition coincided with core temperature of 40.0 ± 0.1°C, whilst core temperature did not rise above ~38.0 ± 0.1°C during the neutral condition and exercise exceeded 60 min without the onset of fatigue. Further, more pronounced reductions in post-exercise MVC and voluntary activation of the exercised (leg extensors) muscles were observed and indicated that the onset of fatigue was attributed to a reduction in muscle recruitment. These more recent studies are in agreement with previous thermoregulatory work by Brück and Olschewski (1987) who reported time to exhaustion coincided with critically high body temperatures (~40°C) and suggested a potential impairment in neurological functioning due to experiences of dizziness and a lack of coordination at exhaustion. These latter observations, which were similar to Nielsen et al. (1990), further suggest hyperthermia has a direct effect on the CNS. Collectively, the findings from these studies suggest athletes may not be able to exceed this critical limiting temperature during exercise without significant performance impairments due to reductions in neural drive (Lambert et al. 2005). Further, the manipulation of ambient temperature such as hot environmental conditions reduces time to fatigue, with termination occurring at similar core temperatures (Gonzalez-Alonso et al. 1999; Nielsen et al. 2001). However, this is primarily evident during externally-paced protocols in which behaviour cannot be manipulated to avoid attaining a critically limiting temperature.

On the contrary, Ely et al. (2009) suggest that attainment of the critical limiting temperature is not associated with a reduction in exercise performance. Ely and colleagues (2009) reported that 17 highly trained athletes completed two 8 km track runs in either cool or warm environmental conditions. Time elapsed was recorded every 200 m, and core and skin temperatures and heart rate
were recorded throughout. The authors compared the running velocity during the final 600 m between subjects who had attained a core temperature greater than 40°C compared to those subjects whose core temperature was less than 40°C. Findings indicate that performance (via running velocity) was not different between core temperatures and therefore questions the notion of a critically limiting temperature affecting performance. However, Ely and colleagues (2009) seemed to have not accounted for the responses during the first 7.4 km of the running trial. Firstly, data used for the >40°C group was primarily from the warm environmental group. Interestingly, although not significantly different, core temperatures were different at the start of exercise by approximately 0.5°C between warm (~38.3°C) and cool (~37.8°C) conditions, with a similar rise during exercise as final core temperatures were ~39.6°C vs 40.2°C, respectively. These similar rates of rise in core temperature coincided with lower running velocities, thus suggesting that exercise velocity was regulated by the rate of thermal stress (Marino, 2004). Secondly, previous research has not explicitly outlined that a critical limiting temperature is 40°C for all athletes but rather report a range from 39.5 to 40°C (Cheung and Sleivert, 2004; Gonzalez-Alonso et al. 1999; Marino, 2004), and as evident with heart rate responses to exercise, it may be possible that there is some inter-subject variability in ‘critical’ core temperature which is not taken into account by Ely and colleagues (2009), possibly explaining the lack of differences in performances observed. Regardless, the attainment of high endogenous thermal loads may be present during prolonged intermittent-sprint in the heat and can potentially reduce performance and pacing strategies due to increased thermal strain.

Anticipatory Regulation to Avoid Hyperthermia

Steady-state or incremental time to exhaustion protocols have provided valuable information regarding the peripheral perturbations associated with hyperthermia-induced reductions in exercise performance (Galloway and Maughan, 1997; Nielsen et al. 1993). In relation to the models previously discussed, exercise is generally terminated at a critical limiting temperature during external-paced protocols; however, during self-paced exercise a centrally mediated regulation of pacing strategies
appears to be present to govern the rate of rise in thermal strain (Tatterson et al. 2000). As already mentioned, Tucker et al. (2004) examined 20 km cycling time trial performance in hot and neutral conditions and reported that while rectal temperature increased over time in both conditions, power output and iEMG of the quadriceps was significantly reduced in the hot condition before differences in rectal temperature, heart rate or RPE were apparent. The reduction in power output and muscle recruitment suggests there is an anticipatory neuromuscular response to exercise in the heat to regulate the same rate of rise of thermal stress to align with (less physiologically taxing) responses in cooler conditions. In support of the notion of anticipatory regulation is the study by Marino et al. (2004) who reported that lighter African runners outperform heavier Caucasian runners during an 8 km time-trial in hot environmental conditions due to an earlier reduction in running speed in the Caucasian runners. The findings suggest that the accelerated rate of rise of heat storage is responsible for anticipatory declines in exercise intensities in the heat. Similarly, self-paced cycling protocols have also provided insight into the regulation of pacing strategies in anticipation of thermal stress and have indicated predictive declines in power output are reflective of changes in neural drive (via EMG) (Tucker et al. 2004). These observations suggest the CNS is pivotal in the role of interpreting afferent feedback and providing appropriate efferent motor command to the active musculature during exercise in the heat (Altareki et al. 2009; Werner et al. 2010). While the specific mechanism for an anticipatory regulation of pacing strategies during self-paced exercise is still somewhat equivocal, previous literature has shown significant reductions in work rate prior to significant elevations in thermal stress during exercise in the heat.

Similar to these continuous exercise protocols, team sport athletes are also able to adopt pacing strategies throughout a match in response to and/or anticipation of attaining critically high thermal loads. Team sport athletes have been reported to experience significant elevations in core temperature, with previous literature rarely reporting values exceeding 39.5°C (Duffield et al. 2009; Edwards and Clark, 2006), but with suggestions of a down-regulation of intensity during a match to induce a
plateau in core temperature (Duffield et al. 2009). Furthermore, the notion of an anticipatory regulation of exercise intensity due to environmental heat and elevated core temperatures have also been observed during self-paced intermittent-sprint exercise. Skein and Duffield (2010) reported a reduction in pacing strategies via less distance covered during the hard running of a 50 min intermittent-sprint exercise protocol in the heat during no fluid ingestion condition, compared to fluid ingestion. These differences were observed prior to any differences in physiological variables, including core temperature and heart rate.

**Neuromuscular Responses to Hyperthermia**

The premise of centrally-mediated reductions in pacing strategies and exercise performance is the feed-forward decline in efferent motor output from the CNS to the active musculature (Swart et al. 2011; Tucker et al. 2004). More specifically, hyperthermia has been linked to the inability to sustain a given force (during an isometric contraction), reduction in the selection of a given exercise intensity/power output (during self-paced exercise) or match required demands which are externally governed (during steady state or incremental exercise protocols) (Cheung, 2008). Furthermore, the effect of hyperthermia on the neuromuscular system and exercise performance are also dependent on the given exercise task (Tucker, 2008), method of heating (active v passive) (Bishop, 2003), and degree of temperature change at the core, muscle and skin (Gonzalez-Alonso et al. 1999). The most common methods of assessing the effect of hyperthermia on neuromuscular function is the use of EMG on active and non-active musculature during exercise or evoked muscle stimulation of the peripheral nerve, primarily during resting and maximal isometric contractions. The latter technique allows the investigation of peripheral fatigue due to changes in twitch contractile properties (TCP) of the muscle. Contractile properties specifically examine peak torque development, time to attain peak torque, contraction duration, and rate of relaxation, thus providing information about the contractile unit *per se* (Cheung, 2008). Further, the comparison of the superimposed twitch during the MVC and
resting/potentiated twitch allows the use of the interpolation technique and by default, allows insight into the level of central fatigue via the measurement of voluntary activation (Merton, 1954).

Despite these limitations, results appear to be consistent with assessments completed during exercise in that hyperthermia reduced maximal voluntary torque and voluntary activation, indicating hyperthermia may have a direct effect on the CNS (i.e. central fatigue) (Nybo and Nielsen, 2001; Morrison et al. 2004; Thomas et al. 2006; Todd et al. 2005). Nybo and Nielsen (2001) examined the effect of (exercise induced) hyperthermia on MVC and central activation during a 2 min sustained isometric contraction of the knee extensors and ‘non-exercised’ muscle group (handgrip). Reductions in post-exercise MVC of the knee extensors were more pronounced in the hyperthermic condition, due to a greater decline in central activation ($54 \pm 7\%$) compared to the control condition ($82 \pm 6\%$). Further, MVC in the ‘non-exercise’ forearm flexors was reduced in the hyperthermic condition and followed a similar pattern of fatigue development to the knee extensors (Nybo and Nielsen, 2001). Additionally, Martin et al. (2004) and Saboisky et al. (2003) both examined ‘exercised’ knee-extensors and ‘non-exercised’ forearm flexors following exercise to exhaustion in hot environmental conditions. Findings by Martin et al. (2004) showed force and voluntary activation were significantly reduced post-exercise in shortening and lengthening phases of muscular contraction of the leg extensors but not the forearm flexors. Similarly, Saboisky et al. (2003) indicated reductions in MVC and central activation ratio (CAR) in knee extensors during maximal isometric contractions but no effect on the forearm flexors. Consequently, Saboisky et al. (2003) and Martin et al. (2004) report similar findings to Nybo and Nielsen (2001), in that maximal force production of active musculature following exercise is reduced, primarily due to reductions in neural drive. However, Saboisky et al. (2003) and Martin et al. (2004) results also suggest that the CNS is able to selectively reduce neural drive to specific muscle during exercise-induced hyperthermia.
Compromised CNS function as a result of hyperthermia has been further highlighted using passive heating methodologies (Morrison et al. 2004; Periard et al. 2011). The examination of maximal isometric force development of an isolated muscle group (knee extensors) following passive heating appears to be a result of reductions in neural drive evident via reduced voluntary activation (VA) associated with an increased core temperature (Morrison et al. 2004; Todd et al. 2005). Morrison et al. (2004) reported passive heating of core temperature from 37.4 to 39.4°C reduced maximal voluntary contraction force and voluntary activation by 13 ± 18 and 11 ± 11%, respectively. Interestingly, skin cooling with reductions in cardiovascular and psychophysical strain did not restore MVC and VA values, although when core temperature returned near to baseline values, MVC and VA were restored to baseline levels. These data suggest elevated core temperature associated with passive heating reduces maximal voluntary force due to a reduction in neural drive (i.e. VA), thus indicating central mechanisms may be responsible rather than changes to the contractile unit influencing performance during isometric contractions.

Further investigations into the central and peripheral contributions to hyperthermia on maximal force production have been conducted by Periard et al. (2011). Periard et al. (2011) examined MVC and VA following either active or passive heating until rectal temperature reached 39.5°C and reported force production during a 45 s MVC to be reduced after both forms of heating compared to control, but more pronounced after an active warm-up. Similar to previous findings (Morrison et al. 2004), VA was also suppressed following warm-up conditions, suggesting hyperthermia has a detrimental effect on neuromuscular function, and thus a suppression of maximal isometric force. Accordingly, significant reductions in MVC are a result of reductions in neural drive to the active musculature. While the relationship between isolated, single muscle function and full-body dynamic movements are tenuous, these aforementioned ergolytic neuromuscular responses appear to be due to an increased core temperature; suggesting the reduction in intermittent-sprint performance and pacing of sub-maximal efforts may be a result of reduction in neural drive to the active musculature.
Mechanisms Related to Pre-cooling to Reduce Hyperthermia during Exercise

Previous literature suggests there is a direct relationship between the volume of cooling used with the magnitude of change in thermal load and ensuing exercise performance improvement; evident in whole-body ice-bath submersion seemingly the most ergogenic (Castle et al. 2006; Duffield and Marino, 2007; Minett et al. 2011). It appears that in order for pre-cooling to be effective on subsequent performance, cooling durations need to be 15-30 min; although dependent on the cooling temperature, mode and surface area cooled. The ergogenic effects of pre-cooling for exercise performance have been linked to the reductions in the aforementioned strain on cardiovascular and thermoregulatory systems (Booth et al. 2001; Cotter et al. 2001). Therefore, athletes are able to exercise at higher work rates for the same or similar thermal and physiological stress during self-paced exercise. It has been well-established that exercise in warm, humid environmental conditions increases the accumulation of endogenous and exogenous heat, with methods of heat dissipation to the external environment compromised (Kenny et al. 2011). Previous literature indicates that the act of pre-cooling the core and peripheral tissues allows an increased gradient between starting thermoregulatory strain and the level of internal heat stress where performance is compromised (Marino, 2002). On the contrary, cooling of core temperature due to ice-bath submersion also involves the cooling of the periphery, more specifically, skeletal muscle (Faulkner et al. 1990). With this in mind, the effect of reduced muscle temperature may be deleterious to team sport athletes as previous research has highlighted reduced muscle temperature negatively effects high-intensity exercise performance (Sleivert et al. 2001).

Previous literature has shown the increased use of conductive and convective methods following cooling allows a reduced reliance on evaporative methods (sweating) (Casa, 1999; Reilly and Cable, 2008) and thus preserving blood volume and MAP (Nybo, 2008). Such physiological outcomes have been related to improved maintenance of central blood volume, stroke volume and cardiac output, increased peripheral vasoconstriction and redistributing blood flow to exercising muscles (Roberts et
al. 1977). Furthermore, numerous studies have shown pre-cooling reduces heart rates at a given workload (Lee and Haymes, 1995; Schmidt and Brück, 1981) or exercising at a higher power output for a similar heart rate compared to no cooling (Duffield et al. 2010). Collectively, previous literature indicates that pre-cooling is effective in allowing an increased reliance on alternative methods of heat dissipation than evaporation to minimise thermoregulatory and associated cardiovascular stress; and thus maintain exercise performance in the heat (Nybo, 2008).

More recent research indicates that pre-cooling also improves performance through curbing the negative effect of heat stress on CNS function (Nybo et al. 2002). Tucker et al. (2004) has shown reducing core temperature helps maintain recruitment of active musculature during exercise in the heat. This notion of improved central drive after pre-cooling has been shown with the reduction of voluntary drive during maximal isometric contractions when in a hyperthermic state (Moran et al. 1998; Nybo and Nielsen 2001; Saboisky et al. 2003), but returns to baseline values once cooling is applied and core temperature is reduced (Morrison et al. 2004; Thomas et al. 2006). These changes in EMG and muscle recruitment during MVC’s may be extrapolated to a team-sport context, with several reports of maintained neural drive pre-exercise and during half-time of an ISE protocol (Minett et al. 2011). Accordingly, the positive effects associated with pre-cooling are related to an increased exercise performance through the adoption of higher intensities for the same rate of rise in thermal stress compared to cooler conditions. In relation to team sports, pre-cooling may provide an advantage via the ability to exercise at higher intensities throughout the match with similar degree of thermal stress.

Pre-cooling often includes cooling of the periphery, and more specifically the reduction of skeletal muscle temperature which has been linked to performance decrements during short duration, high-intensity exercise (Racinais et al. 2009). Such a physiological consequence may be problematic for team-sport athletes who are required to complete maximal sprints on a repeated basis over a
prolonged period of time. There are some suggestions that reduced muscle temperature reduces muscle blood flow (Peiffer et al. 2009), which in turns affects muscle metabolism and glycolytic enzyme activity (Febbario et al. 1994). Further, reduced muscle temperatures are primarily associated with negative effects on nerve conduction velocity, resulting in lower evoked twitch force, delayed time to peak twitch force and rate of relaxation (Cheung, 2008). Therefore, while there are potential benefits, it appears pre-cooling prior to ISE should be conducted with caution and delivery of such pre-cooling maneuvers should be performed with maximal potential changes to core temperature while preventing excessive reduction in muscle temperature (Duffield and Lovell, 2009).

**Conclusion**

In summary, exercise in the heat has detrimental effects on exercise performance, including intermittent-sprint exercise. Proposed mechanisms to explain performance decrements include the cardiovascular system being compromised and the increased reliance of evaporative heat loss via sweating. Hyperthermia has also been linked to having a direct effect on CNS and thus reducing reductions in neural drive from the brain. The negative effects of hyperthermia have been further supported by studies showing that increasing pre-exercise core temperature via passive methods can be detrimental to endurance and intermittent-sprint performance due to exacerbated increases in core temperature. These heating methods are also beneficial to shorter duration exercise due to the concomitant increase in muscle temperature and nerve conduction rates (Bishop, 2003). On the contrary, pre-cooling has also shown that reducing one’s internal temperature can alleviate some of the negative physiological implications associated with exercise-induced hyperthermia. However, these pre-exercise interventions need to be applied with caution as they have ergolytic and ergogenic effects to performance. Overall, the use of altered thermal stress prior to intermittent-sprint exercise in the heat can provide an effective means of examining the effect of thermal stress on intermittent-sprint performance and the peripheral and central contributions to altered pacing during team sport exercise.
2.7 Conclusion

In conclusion, team sport athletes encounter physical and physiological stress during training and competition due to prolonged bouts of intermittent-sprint exercise of various exercise intensities, ranging from maximal sprints to walking. As team sports are self-paced, it is likely that exercise intensities throughout the entire match and between respective modes (i.e. pacing strategies) are regulated in order to ensure match completion without sacrificing critical (match-specific) performance outcomes. Previous literature has attempted to explain the regulation of pacing strategies in both continuous and intermittent-sprint protocols via the use of different fatigue models. As the name suggests, the main premise of peripheral models are physiological perturbations that directly affect the skeletal muscle and impair performance. On the contrary, centrally-mediated models suggest that the CNS regulates these physiological responses to avoid reaching catastrophic levels that are described in the peripheral models. Accordingly, the sub-conscious regulation of the physiological responses may be via changes in neural drive to active musculature, which in turn alters the pacing strategies. Finally, the regulation of pacing has also been suggested to be a conscious regulation in which the athlete dictates their work rate based on their conscious perception of effort.

A common method to assess these pacing strategies is via altering the central, peripheral, and/or perceptual states to observe how subsequent pacing strategies are altered. The methods used in the present thesis and outlined in this literature review are sleep deprivation, which primarily influences perception and mood states; CHO intake, which is primarily associated with changes at the periphery; and thermal stress which appears to affect central and peripheral components. Accordingly, this thesis will investigate the effect of changes in pre-exercise physiological and perceptual states via various circumstances on intermittent-sprint performance. Furthermore, due the limited literature examining the mechanisms responsible for pacing during such exercise, this thesis will also examine the models proposed to explain fatigue and regulation of exercise intensities. Collectively, the findings from the literature review suggests that pacing strategies during self-paced intermittent-sprint exercise are
combination of central, peripheral and conscious factors and the magnitude of effect will be dependent upon the task, the pre-exercise physiological state of the athlete, and the level of knowledge about their pre-exercise state and their forthcoming exercise task.
Chapter 3

Reliability and Validity of a Laboratory-Based Self-Paced Intermittent-Sprint Exercise Protocol
Abstract

The use of self-paced, intermittent-sprint exercise protocols within a controlled laboratory setting for simulation of team-sports is rare. Accordingly, the aim of this study was to assess the reliability of a self-paced, intermittent-sprint protocol designed to simulate the physical and physiological demands of team sport exercise. Following a full familiarisation with the protocol, 10 moderately trained males (20.5 ± 0.9 years, 181.0 ± 4.8 cm, 81.3 ± 7.9 kg) each completed two identical sessions within an enclosed laboratory. The protocol was 40 min of self-paced intermittent-sprint exercise that included a 15-m sprint each minute, followed by sub-maximal and self-paced exercise including hard running, jogging, walking or deep-squat bounds for the remainder of the minute; with only one exercise mode completed each minute. Performance measures recorded included sprint time (s), mean and total distance covered (m) during each sub-maximal effort, and distance covered during double-leg bounds (m). Furthermore, heart rate (HR) and rating of perceived exertion (RPE) were recorded every 5 min. Results indicated no significant differences between the repeated sessions for all performance and physiological measures ($P>0.05$). All performance measures showed a high level of reliability, with intra-class correlations ($r$) ranging between 0.73 - 0.96, and coefficient of variations (CV) between 1.5 - 3.2%. Furthermore, mean difference between sessions were within technical error and limits of agreement (LoA) values for all variables. LoA values for sprint times were 0.17s, while ranging from 1.23 – 9.93 m for sub-maximal efforts. Overall, results indicated that this self-paced intermittent-sprint exercise protocol designed to simulate physical and physiological demands of team sports has a high level of reliability for sprint times, total and mean distance covered and HR and RPE responses.
Introduction

Team sports (such as soccer, rugby league and union) involve prolonged bouts of exercise (60 – 90 min) with sporadic changes in exercise intensity (Bishop and Spencer, 2004). Time motion analysis suggests that teams sports generally are comprised of repeated maximal sprints of ~2 – 5 s in duration, performed every 1 – 2 min and interspersed by active recovery of a self-selected pace and duration (Glaister, 2005; Spencer et al. 2005). Such self-regulation of pacing strategies permits athletes to monitor and adjust exercise intensity, duration and frequency according to match demands and within the context of their own physical ability. Regardless, match-specific data suggests athletes in team sports cover a total of 6.7 – 7.2 km during rugby union (Cunniffe et al. 2009) and 10 – 12 km during soccer matches (Bangsbo et al. 1991; Krstrup 2005; Stolen et al. 2005). Further, physiological responses indicate athletes experience high physiological strain with heart rate responses of 166 – 180 bpm (Coutts et al. 2010; Deutsch et al. 2007; Duthie et al. 2003), relative intensities of 81.0 ± 5.8% \( \dot{V}O_{2\text{max}} \) (Coutts et al. 2003) and rises in core temperatures to ~39.5°C (Duffield et al. 2009). Recent data from match environments suggest some regulation of exercise intensities to protect maximal efforts late in a match (Coutts et al. 2010; Duffield et al. 2009); despite some disagreement regarding the occurrence and extent of within-match pacing strategies (Aughey, 2010).

While many intermittent-sprint laboratory protocols exist that include over-ground running (Bishop et al. 2001; McGregor et al. 1999; Nicholas et al. 2000) or non-motorised treadmills (Hughes et al. 2006; Sirotic and Coutts, 2008; Thatcher and Batterham, 2004); few allow the athlete to freely manipulate the exercise intensity, and thus often exclude the regulation of intensity other than maximal sprints at pre-set intervals. On the contrary, an increasing volume of endurance exercise protocols are utilising self-paced exercise protocols, such as cycling time trials (Atkinson and Brunskill, 2000; El-Sayed et al. 1997; Jeukendrup et al. 1997); however, few protocols incorporating intermittent-sprint exercise are indicative of free-paced team sports. The use of a self-paced laboratory-based exercise protocol that simulates the physical and physiological demands of team sports allows external factors to be
monitored and controlled, thus creating an environment supportive of examining performance and pacing. However, many of these protocols exclude various physical and physiological demands of a game such as self-paced exercise, eccentric loading, and change of direction (Hughes et al. 2006; Sirotic and Coutts 2008). Accordingly, the aim of this study was to assess the reliability of a self-paced intermittent-sprint exercise protocol which has been used in previous published work (Duffield and Marino, 2007; Skein and Duffield, 2010) that is designed to allow self-paced activity.

**Methods**

Following full protocol and equipment familiarisation, ten moderately trained males (age 20.5 ± 0.9 years, height 181.0 ± 4.8 cm, and mass 81.3 ± 7.9 kg) completed a 40 min self-paced intermittent-sprint protocol on two separate occasions. Each testing session was separated by 5 – 7 days of recovery, performed within an enclosed laboratory under controlled environmental conditions (21.7 ± 1.2 °C) at the same time of day to minimise diurnal variation. Participants abstained from caffeine, alcohol and strenuous exercise 24 h prior to each session and were required to record dietary intake 24 h prior to the initial session, which was replicated for all ensuing sessions.

**Intermittent-Sprint Protocol**

The intermittent-sprint protocol included 40 min of self-paced exercise at various intensities ranging from maximal sprints to walking. Participants were required to commence each minute of the ISE protocol behind a marked line at the start of a synthetic running track. On cue, participants completed a 15 m maximal sprint at the commencement of each minute followed by a 5 m deceleration zone before impacting with a high-jump mat placed upright against a wall. As depicted in Figure 3.1, each minute following the maximal sprint, participants completed self-paced sub-maximal exercise at varying paces (hard running, jogging, and walking) in a shuttle-run format (over the 15 m running track) for the remainder of the minute. Participants were required to place one foot over the line at either end of the 15 m track before turning to complete the next shuttle. When instructed, participants
returned to the starting position with 10 s remaining in each minute to complete the ensuing sprint. Only one sub-maximal mode was completed each minute and was rotated as per the order above. Self-paced, sub-maximal efforts included ‘hard running’ in which participants were instructed to ‘cover as much distance as possible’ for the given minute, and jogging and walking efforts that were covered at the participant’s self-selected intensity. At the completion of 2 rotations of each sub-maximal mode (every 7 min), participants completed a 15 m maximal sprint and 8 deep-squat, double-leg bounds aiming to cover as much distance as possible to invoke an additional eccentric load. Participants were provided verbal encouragement during maximal sprints and hard running bouts.

**Measures**

Performance measures recorded each minute included sprint time(s) using an infra-red timing system (Speed-Light, Swift, Australia), while distance covered during each respective sub-maximal bout and double leg bounds (m) were manually counted with 1 m markings on the running track. Further, heart rate (HR) was recorded with a chest heart rate monitor and wrist watch receiver (F1, Polar Electro-Oy, Finland), and Rating of Perceived Exertion (RPE) were recorded throughout the protocol.

**Statistical Analysis**

Data are reported as mean ± standard deviation (SD). A repeated measures ANOVA was used to determine significant differences between two testing sessions (session 1 vs session 2), with significance set at $P=0.05$. Reliability of performance and physiological measures were determined using intra-class correlations ($r$) and coefficient of variation (CV) of the log-transformed variable with confidence intervals (CI) set at 90% (Hopkins, 2000). The typical error (SEM) to assess within-subject differences and limits of agreement (LoA) to assess a reference range for changes were calculated for performance and physiological variables, with a 95% level of agreement (Atkinson and Nevill, 2000; Hopkins, 2000). Acceptable reliability was deemed when $r \geq 0.8$, $CV \leq 5\%$ and/or within the 95% LoA, respectively.
Figure 3.1: Overview of the Self-Paced Intermittent-Sprint Exercise (ISE) protocol that includes a 15 m maximal sprint at the start of each minute, followed by sub-maximal, self-paced exercise (hard running, jogging, walking, bounds) of various intensities for the remainder of the minute. HR = Heart Rate; RPE = Rating of Perceived Exertion
Results

Table 3.1 shows the mean sprint time, mean distance covered during sub-maximal exercise bouts and total distance covered during each session. There were no significant differences between sessions for any performance measure ($P>0.05$). Further, there were no differences evident between sessions for HR or RPE ($P>0.05$; Table 1). Results of reliability analyses indicated that performance variables, HR and RPE measures recorded during the self-paced intermittent-sprint protocol were highly reliable, with intra-class correlations ranging from $r=0.73 – 0.96$ and CV of 1.5 – 6.6% (Table 3.1). As shown, typical error for mean sprint times was 0.05 s while LOA was 0.17 s. Further, the typical error for mean distance covered during the respective exercise modes ranged from 0.38 – 3.10 m, while the LoA ranged from 1.23 – 9.93 m. Finally, HR and RPE typical error values were 2.0 bpm and 0.4, and LoA were 6.4 bpm and 1.3, respectively.
Table 3.1: Mean ± SD session 1 and 2 values and intra-class correlation (r), coefficient of variation (CV), typical error (SEM), typical error (SEM) and limits of agreement (LoA) for performance and physiological measures.

<table>
<thead>
<tr>
<th></th>
<th>Session 1</th>
<th>Session 2</th>
<th>r</th>
<th>CV (%)</th>
<th>SEM</th>
<th>LoA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sprint time (s)</td>
<td>2.72 ± 0.18</td>
<td>2.68 ± 0.15</td>
<td>0.90</td>
<td>1.9</td>
<td>0.05</td>
<td>0.17</td>
</tr>
<tr>
<td>Mean distance covered during each bout (m)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hard Running</td>
<td>135.8 ± 6.1</td>
<td>136.5 ± 7.8</td>
<td>0.82</td>
<td>2.2</td>
<td>2.9</td>
<td>9.4</td>
</tr>
<tr>
<td>Jogging</td>
<td>94.1 ± 12.8</td>
<td>96.0 ± 15.4</td>
<td>0.95</td>
<td>3.2</td>
<td>3.1</td>
<td>9.9</td>
</tr>
<tr>
<td>Walking</td>
<td>50.7 ± 4.7</td>
<td>50.3 ± 3.7</td>
<td>0.89</td>
<td>2.5</td>
<td>1.3</td>
<td>4.3</td>
</tr>
<tr>
<td>Bounds</td>
<td>17.4 ± 0.9</td>
<td>17.5 ± 0.6</td>
<td>0.73</td>
<td>2.3</td>
<td>0.3</td>
<td>1.2</td>
</tr>
<tr>
<td>Total distance covered during ISE protocol (m)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total distance</td>
<td>3362 ± 235</td>
<td>3393 ± 282</td>
<td>0.96</td>
<td>1.5</td>
<td>49.9</td>
<td>159.8</td>
</tr>
<tr>
<td>Hard Running</td>
<td>1630 ± 74</td>
<td>1638 ± 94</td>
<td>0.82</td>
<td>2.2</td>
<td>35.3</td>
<td>113.0</td>
</tr>
<tr>
<td>Jogging</td>
<td>1129 ± 154</td>
<td>1152 ± 185</td>
<td>0.95</td>
<td>3.2</td>
<td>37.5</td>
<td>119.1</td>
</tr>
<tr>
<td>Walking</td>
<td>603 ± 59</td>
<td>604 ± 44</td>
<td>0.85</td>
<td>3.2</td>
<td>19.9</td>
<td>63.7</td>
</tr>
<tr>
<td>Bounds</td>
<td>87 ± 4</td>
<td>88 ± 3</td>
<td>0.73</td>
<td>2.3</td>
<td>1.9</td>
<td>6.1</td>
</tr>
<tr>
<td>Distance covered during 10min phases of ISE protocol</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0-10min</td>
<td>900 ± 87</td>
<td>898 ± 90</td>
<td>0.95</td>
<td>2.2</td>
<td>19.3</td>
<td>7.3</td>
</tr>
<tr>
<td>11-20min</td>
<td>835 ± 65</td>
<td>835 ± 81</td>
<td>0.87</td>
<td>3.0</td>
<td>24.2</td>
<td>10.0</td>
</tr>
<tr>
<td>21-30min</td>
<td>761 ± 53</td>
<td>785 ± 66</td>
<td>0.80</td>
<td>3.6</td>
<td>26.4</td>
<td>12.0</td>
</tr>
<tr>
<td>30-40min</td>
<td>866 ± 57</td>
<td>875 ± 60</td>
<td>0.71</td>
<td>3.7</td>
<td>31.1</td>
<td>12.2</td>
</tr>
<tr>
<td>Heart Rate (bpm)</td>
<td>177 ± 9</td>
<td>176 ± 7</td>
<td>0.94</td>
<td>1.2</td>
<td>2.0</td>
<td>6.4</td>
</tr>
<tr>
<td>RPE</td>
<td>6.5 ± 1.2</td>
<td>6.8 ± 1.4</td>
<td>0.92</td>
<td>6.6</td>
<td>0.4</td>
<td>1.3</td>
</tr>
</tbody>
</table>

No significant differences between sessions
Discussion

The aim of this study was to examine the test-retest reliability of the performance, physiological and perceptual parameters during a 40 min self-paced, intermittent-sprint exercise protocol utilised in the subsequent experimental chapters (Chapters 4-6). Test-retest reliability was examined using intra-class correlations (r), coefficient of variation (CV) and limits of agreement (LoA). The self-paced intermittent-sprint exercise protocol was shown to have high repeatability between sessions. The test-retest reliability was particularly high in the measurement of total distance covered (r= 0.96, CV=1.5%) and when reported as individual hard running, jogging and walking efforts (r= 0.82 - 0.95, CV= 2.2 - 3.2%). Distance covered during the bounds was moderately reliable between sessions (r= 0.73, CV= 2.3%). Additionally, HR and RPE also demonstrated high reliability between sessions (r= 0.92 - 0.94, CV= 1.2 - 6.6%), suggesting the physiological load and perceived exertion between sessions were similar. The mean differences between sessions were within typical error and LoA values for all performance and physiological variables. Such findings suggest the reliability of the physiological, perceptual and performance responses to a self-paced intermittent-sprint protocol are acceptable.

The present results also indicate the exercise protocol had comparable physiological responses to data reported for team sport matches of similar competition standard. Ali and Farrally (1991) have reported mean heart rate responses of 172 ± 12 bpm for semi-professional soccer players, which correspond with the 177 ± 9 and 176 ± 7 bpm reported in the present study. The current protocol demonstrates similarities to the physical demands of previously reported team sport exercise (Glaister, 2005; Spencer et al. 2005). Mean sprint times for the respective sessions were 2.72 ± 0.18 and 2.68 ± 0.15 s, which compares well with the 2 – 5s reported in previous time-motion analysis data (Spencer et al. 2005). Moreover, the total distance covered during the (40 min) protocol (3362 and 3393m) is representative of the first half of many team sport matches (Cunniffe et al. 2009). Specifically, TMA data report total distance during an 80 min rugby league match ranges from 6.7- 7.2 km (Cunniffe et
al. 2009) and ~10 km during an 80 min match for top-class soccer players (Mohr et al. 2003). These values have been shown to be subject to variability depending on the playing position and competition status (Bangsbo et al. 1991; King et al. 2009). The validity of laboratory protocol is difficult given the controlled nature of such environments compared to the high volumes of permutations of movement patterns in the field. However, the current data suggests a similar load and physiological responses to previously reported match-based data (Deutsch et al. 2007; Duthie et al. 2003).

The variations in speed and distance covered (pacing strategies) throughout team sport exercise and during the intermittent-sprint exercise protocol are due to the self-paced nature of the game. Further, the manipulation of these pacing strategies during team sports may be dependent upon the internal (physiological) and external (environmental) and/or perception of such conditions (Nicholas et al. 2000). The manipulation of pacing strategies was permitted in the present intermittent-sprint protocol, allowing the novelty of participants setting and altering their exercise intensity in order to complete exercise bout based on both internal and external feedback. Accordingly, this highly reliable self-paced intermittent-sprint protocol may allow for the controlled laboratory investigation into the effects of an intervention (ergogenic aids, environments, etc) on the performance of intermittent-sprint exercise in team sport athletes.
Chapter 4

Intermittent-Sprint Performance and Muscle Glycogen after 30 h of Sleep Deprivation

As published in Medicine and Science in Sport and Exercise

Chapter 4 – Sleep Deprivation Study

Abstract

The aim of this study was to determine the effects of 30 h sleep deprivation on consecutive day intermittent-sprint performance and muscle glycogen content. Ten male, team-sport athletes performed a single day ‘baseline’ session, and two consecutive-day experimental trials separated either by a normal night’s sleep (CONT1 and CONT2) or no sleep (SDEP1 and SDEP2). Each session included a 30min graded exercise run (GXR) and 50 min intermittent-sprint exercise (ISE) protocol, including a 15m maximal sprint every minute and self-paced exercise bouts of varying intensities. Muscle biopsies were extracted pre and post exercise during the baseline session and pre-exercise on Day 2 during experimental trials. Maximal voluntary force (MVF) and activation (VA) of the right quadriceps, nude mass, heart rate (HR), core temperature (Tcore), capillary blood lactate (La’) and glucose (Glu), rating of perceived exertion (RPE) and a modified Profile of Mood States (POMS) were recorded pre, post and during the exercise protocols. Mean sprint times were slower on SDEP2 (2.78 ± 0.17 s) compared to SDEP1 (2.70 ± 0.16s) and CONT2 (2.74 ± 0.15s; P <0.05). Distance covered during self-paced exercise was reduced on SDEP2 during the initial 10 min compared to SDEP1 and during the final 10min compared to CONT2 (P <0.05). These performance differences were associated with significant reductions in muscle glycogen concentration on SDEP2 (209 + 60mmol kg dw) compared to CONT2 (274 + 54mmol kg dw; P= 0.05) and pre-exercise negative mood states. MVF and VA were reduced on Day 2 of both conditions; however, were lower in SDEP2 compared to CONT2 (P <0.05). In conclusion, sleep loss and associated reductions in muscle glycogen and increased perceptual stress reduced sprint performance and slowed pacing strategies during intermittent-sprint exercise for male team-sport athletes.

Keywords: sleep loss; pacing; team sports; glycogen content
Introduction

Athletes involved in team sports may be subject to varying degrees of sleep deprivation either before or after training and competition (Bishop, 2004; Richmond et al. 2004). The extent of this sleep disruption or loss can range from minor (2 – 4 h) to quite extensive (overnight), depending on the prevailing circumstances. As sleep is known to be critical in the restoration of metabolic processes (Driver and Taylor, 2000) and regulation of hormone secretion (growth hormone, prolactin and cortisol) (Mougin et al. 2001), the effect of sleep loss prior to exercise may be detrimental to team sport exercise. Moreover, sleep disruption has been associated with the increased perception of negative mood states (Martín and Haney, 1982), suppression of resting heart rate and core temperature (Vaara et al. 2009), reductions in aerobic oxidation capacity and decreased metabolic enzyme activity (Vondra et al. 1981). Despite negative metabolic and physiological consequences at rest and during exercise following sleep loss, and the belief among athletes and coaches of the importance of adequate sleep for ensuing performance, the effect of sleep loss on team sport exercise remains unclear.

The reality of many competition and training schedules results in athletes performing prolonged, high-intensity exercise bouts on consecutive days. Given the high physical, physiological and metabolic demands associated with team sports (Spencer et al. 2005), recovery from such exercise may be prolonged. Recovery may be further dampened if adequate sleep is not achieved as sleep deprivation has been associated with a 10-15% increase in energy expenditure and metabolic demands (Berger and Phillips, 1995). Furthermore, given muscle glycogen depletion is evident following prolonged, repeated-sprint exercise (Krustrup et al. 2006), the increased energy expended during prolonged hours awake may affect rate of resynthesis of the depleted muscle glycogen. Additionally, sleep deprivation may also alter physiological functions including a decreased resting heart rate and body temperature (Vaara et al. 2009) while decreased evaporative heat loss (Sawka et al. 1984), maximal heart rate (Chen, 1991) and peak oxygen consumption (Chen, 1991; Plyley et al. 1987) have been observed.
during exercise. Despite sleep deprivation affecting these physiological parameters at rest and during exercise, the effect on team sport athletes and subsequent self-paced, intermittent-sprint performance is not well understood.

The implications of altered physiological states due to sleep deprivation may negatively affect subsequent exercise performance (Horne and Pettitt, 1984; Martin, 1981; Reilly and Piercy, 1994; Souissi et al. 2003). While no studies to date have specifically examined self-paced intermittent-sprint exercise, several studies have examined components of team sport exercise. For example, prolonged, self-paced exercise protocols have shown sleep deprivation reduces distance covered during 30 min free-paced treadmill running in 11 healthy male participants (Oliver et al. 2009); however, 30 h of sleep deprivation had minimal effect on exercise workload manipulation at a set RPE in 24 healthy students (Martin and Haney, 1982). Fixed-paced protocols also demonstrate contradictory findings, with 72 h sleep loss having minimal effect on steady state exercise at 40, 60, and 80% \( \dot{V}_O_{2\text{max}} \) in untrained subjects (Horne and Pettitt, 1984); while Martin (1981) reported sleep deprivation reduced time to exhaustion following 36 h sleep loss in healthy (19 – 27 yrs) subjects. Additionally, peak and mean power during a 30 s Wingate anaerobic test is reduced following 36 h of sleep loss (Souissi et al. 2003). Finally, studies examining muscular strength have reported 60 h sleep loss to have minimal effect on 25 maximal isokinetic contractions of the upper or lower body in 11 healthy subjects (Symons et al. 1988). Conversely, Reilly and Piercy (1994) reported sleep restrictions of 3 h for 3 consecutive days reduced maximal strength during bench press, leg press and dead lift and reduced sub-maximal lift capacity as well. While previous studies provide insight into the possible effects of sleep deprivation on intermittent-sprint exercise, the specific effect on actual or simulated (intermittent-sprint) team-sport exercise performance remains unclear. Furthermore, the underlying mechanisms responsible for alterations in exercise intensity and thus pacing strategies is not well understood, as majority of studies have used constant-intensity protocols which may not be indicative of team sport exercise.
Despite the possibility of team sport athletes experiencing sleep loss during the course of a normal training or competition schedule, the relationship between sleep deprivation, recovery and subsequent intermittent-sprint performance remains equivocal. Therefore, the aim of this study was to examine the effect of 30 h sleep deprivation on self-paced intermittent-sprint performance and pacing strategies. A secondary aim of the study was to examine sleep deprivation on muscle glycogen restoration, mood states and neuromuscular function, and implications on subsequent team sport exercise. We hypothesized that ~30h sleep deprivation would negatively affect intermittent-sprint performance and recovery of muscle glycogen compared to a normal night’s sleep.

**Methods**

**Participants**

Ten male team sport athletes, playing at club and representative levels with competitive matches and formal training sessions ≥ 3 x per week took part in this study. Mean ± standard deviation (sd) characteristics were: age 21 ± 3 yrs, mass 81.5 ± 9.5 kg, height 178.6 ± 9.2cm and maximal aerobic capacity ($\dot{V}O_{2max}$) 56.8 ± 5.3 ml kg$^{-1}$min$^{-1}$ (45.3 – 63.1 ml kg$^{-1}$min$^{-1}$). All participants were questioned about their sleeping patterns and baseline sleep data was collected 2 days prior to initial testing, with participants excluded if substantial variation in sleeping patterns were evident. Participants were informed of the requirements and demands of the study and written informed consent was obtained prior to the commencement of testing. This study was approved by the Institutional Human Ethics Committee.

**Overview**

Initially participants completed a familiarisation session that also included a graded exercise test to determine $\dot{V}O_{2max}$ and velocity of $\dot{V}O_{2max}$ ($v\dot{V}O_{2max}$). Following familiarisation, participants completed a one day (baseline) trial (n= 7), and two consecutive-day experimental trials (n= 10) in a counter-balanced, cross-over design. Experimental trials consisted of a normal night’s sleep (CONT1 and
CONT2) or no sleep (SDEP1 and SDEP2) between testing days. All testing procedures and exercise protocols were identical between the baseline and experimental sessions except for the inclusion of the muscle biopsy procedures. The exercise bouts performed on each day included a 30 min graded exercise run (GXR) followed by a 50 min intermittent-sprint exercise (ISE) protocol. Muscle biopsies were obtained pre-GXR and 30 min post-ISE during the baseline session, while a single biopsy was obtained pre-GXR on Day 2 of both experimental trials. As all food, fluid, activity and testing procedures were standardised, the baseline session served to provide information regarding representative muscle glycogen concentration of the vastus lateralis following exercise on Day 1 for both conditions of the experimental trials. Accordingly, no biopsies were obtained on Day 1 during actual experimental trials to avoid any delayed muscle soreness on Day 2 due to the biopsy procedure. The experimental trials involved two consecutive-day testing sessions of identical procedures separated by either one night of ‘normal’ sleeping hours relative to the participant’s sleep patterns (CONT), or a night without sleep (SDEP). While extreme, the study was designed to simulate a night of minimal sleep due to extended travel commitments. Following exercise on CONT1, participants remained supervised within the laboratory until after the meal provision, before returning home for a ‘normal’ night sleep and returning to the laboratory by 08.30am the following morning. Conversely, during SDEP participants were required to remain awake in the laboratory for the duration of the experimental trial and were supervised by the research team. Each trial was completed at the same time of day (15:00), and was separated by 7 days to ensure adequate recovery and to allow participants to regain normal sleep patterns.

All food and fluid during the consecutive day trials were matched and standardised between the two conditions. The diet consumed by the participants was controlled by the research team ~24 h prior to each session and between testing sessions during the experimental trials (total dietary control: ~60h) with a carbohydrate (CHO) intake of ~3g/kg body weight (bw). Consumption and timing of food and fluid was recorded by participants in a diary, and inspected by the research team. Food and fluid were
provided to participants and were matched for caloric intake between conditions; however, during SDEP a smaller portion of the food allocated for dinner was consumed at the same time as CONT, with the remaining food spaced intermittently throughout the ensuing evening. Participants abstained from food, fluid and caffeine 3 h prior to each testing session and no strenuous activity was completed 24 h before each session, excluding the exercise protocol. Participants consumed 500 ml of water 1 h prior to testing to ensure the participants presented in a euhydrated state and consumed an additional 500 ml between the GXR and ISE protocols. Sleep patterns were monitored ~2 nights prior to the initial testing session to determine ‘normal’ sleeping patterns for each participant, through the use of sleep diaries and sleep watches (Actiwatch, Philips Respironics, PA, USA). The sleep watches were also worn and diaries completed throughout the experimental trials to ensure compliance with the experimental interventions. All testing sessions were completed within a controlled environment with an ambient temperature during the GXR and ISE of 19 ± 1°C and 17 ± 1°C, respectively.

**Exercise Protocol**

**Treadmill Protocol**

For each respective day, participants commenced with a 30 min graded exercise run (GXR) on a motorised treadmill (True 825 S.D.F.T System, ETL Testing Laboratories INC., NY, USA) at 60, 70, 80% v\(\text{VO}_{2\text{max}}\) for 10 min at each respective intensity. Exercise intensities were calculated based on the relative percentage intensity determined from v\(\text{VO}_{2\text{max}}\). The inclusion of the GXR was designed to simulate a warm up and ensured the entire exercise completed and energy expended during a respective testing session was similar to a competition or training scenario.

**Intermittent-Sprint Exercise (ISE) Protocol**

Following a 10 min recovery, participants then completed a 50 min free-paced intermittent-sprint exercise protocol with 1 min breaks every 10 min. The self-paced exercise protocol was completed on a synthetic surface 20 m in length and involved a 15 m maximal sprint each minute, with a 5 m
deceleration zone before impacting with a crash mat placed upright against a wall. Immediately following impact with the mat, participants completed a self-paced exercise at varying intensities for the remainder of the minute (~50 s) (Skein and Duffield, 2010). Exercise bouts were sub-maximal, free-paced activities of hard running, jogging or walking. During the hard running bout, participants were instructed to ‘cover as much distance as possible’, while selecting their own pace during the jogging and walking bouts, respectively. These bouts were completed in a shuttle-run format with only one mode completed each minute, rotating through each minute in the above order. The intra-class correlation \((r)\) and co-efficient of variation (CV) for the sprint times and total distance covered for this ISE protocol are \(r = 0.90\) and \(0.96\) and \(CV = 1.9\) and \(1.5\)%, respectively (unpublished observations, Chapter 3). Participants returned at 50 s of each minute to complete the ensuing sprint.

In order to invoke a greater eccentric load, every 7 min participants completed 8 deep-squat, double-leg bounds, aiming to cover as much distance as possible. Participants were given verbal support and encouragement during sprints and hard running bouts, and were not aware that distance was being counted to ensure no conscious manipulation of exercise intensity was implemented.

**Measures**

*Intermittent-Sprint Performance and Pacing Strategies*

Performance measures recorded during the intermittent-sprint protocol included 15 m sprint time and distance covered during each sub-maximal exercise bout and double leg bounds. Maximal 15 m sprint performances were recorded with an infra-red timing gate system (Speed-Light, Swift, Australia), with mean and total sprint time calculated. Mean and total distance covered throughout each respective sub-maximal exercise bout (hard running, jogging, and walking) were measured by manually counting metres covered using 1 m floor markings. Finally, double leg bounds were recorded by manually measuring the distance covered with a tape measure to determine mean and total distance covered. To assess pacing strategies implemented by the participants, mean sprint times
and distance covered during self-paced exercise were reported as mean ± SD of each 10 min of the ISE protocol.

**Maximal Isometric Contractions and Muscle Activation**

Maximal isometric voluntary contraction (MVC) and evoked twitch properties of the right knee extensors were assessed pre-GXR and ~5-min post-ISE. During pre-GXR assessment, participants completed a 3 min moderate-intensity (60W) warm-up on a cycle ergometer (828E Monark, Stockholm, Sweden). The neuromuscular test was completed on a modified dynamometer, in which participants were seated on a straight back chair with the hip and knee angle at 90° (0° represents full extension). A Velcro strap was placed at the ankle, 1cm above the lateral malleolus, which attached onto a suspended load cell (6000, ICI, Sensortronics, Covina, California, USA). The load cell was attached to the undercarriage frame of the chair and detected the force, which was amplified and recorded by a signal acquisition system (PowerLab, 8/30 and Chart v6.1.1, ADInstruments, Australia). The load cell was zeroed and calibrated before each testing session. Participants were secured to the chair with a waist strap and during testing participants were instructed to place their arms across their chest to minimise additional forces contributing to the MVC.

Muscle stimulation was achieved using two 50 x 90 mm self-adhesive surface electrodes (Verity Medical, Ltd., Stockbridge, Hampshire, UK), placed on the anterior aspect of the right thigh, ~2cm below the inguinal fold and ~3cm above the superior border of patella. Electrode placements were marked on the participant’s skin with a permanent ink maker and instructed to maintain the marks to ensure consistency between sessions. The current was delivered via a stimulator (Model DS 7A, Digitimer Ltd, Weleyn, Garden City, England) using a doublet square wave pulse with a width of 200µs. Initially, the current was applied in incremental steps until a plateau in twitch force was reached. The current was then increased by a further 25% to ensure supra-maximal stimulation was used for all tests. MVC testing consisted of a series of 15 maximal isometric contractions of right
knee extensors. Participants were instructed to produce a maximal isometric effort for duration of ~3 s for each contraction, with the start of each contraction 10 s apart. The first and final 5 contractions included a superimposed electrical stimulus manually delivered ~1 s after initiation of each contraction and within 2 s of relaxation of MVC a second stimulus was delivered, with the muscle at complete rest. All data was processed using a customised, formulated spreadsheet (Excel 2007, Microsoft Corp, Washington, USA). Initially, the effect of gravity of the lower leg was corrected by calculating average load applied to the force transducer during the 50 ms period immediately prior to the force onset. Maximal voluntary force (MVF) was determined as the maximal force exerted prior to the delivery to electrical stimulation and voluntary activation (VA) was calculated using the twitch interpolation technique (Merton, 1954). Mean ± SD MVF and VA for all contractions were assessed within and between conditions.

**Physiological measures**

On arrival, subjects provided a urine sample to measure hydration status (Refractometer 503, Now. Nippon Optical, Works Co, Tokyo, Japan). Nude mass was recorded pre-GXR and 20 min post ISE protocol on a set of calibrated scales accurate to 10g (Fitness Technology Inc, O’Fallon, Missouri, USA). Participants were instructed to ‘towel down’ as much sweat as possible before stepping onto the scales, and the difference in mass was used to calculate sweat rate. Heart rate (HR) was recorded pre-GXR, pre-ISE and every 10 min during the ISE protocol with a heart rate monitor and wrist watch receiver (F1, Polar Electro-Oy, Finland). Core temperature (T_core) was measured with a telemetric capsule that was ingested 4 – 5 h prior to each testing session to ensure it had passed into the gastrointestinal tract and was unaffected by the ingested food and/or fluid. Core temperature was recorded from a hand-held monitor receiving a telemetric measure from the ingested capsule pre-GXR and ISE and every 10 min during the ISE protocol (VitalSense, Mini Mitter, Oregon, USA). Following sterilisation of the fingertip with an alcohol swab, the skin was perforated using a lancet and a 35μL sample of capillary blood from the fingertip was obtained pre-GXR, pre-ISE, 30 min, and
post-ISE. Capillary blood samples were measured for lactate (La’, Lactate Pro, Arkray Inc. Kyoto, Japan) and glucose (Glu, Accu-Chek Advantage, Roche, Mannheim, Germany) as per the manufacturer’s instructions. Coefficient of variation (CV) for the Lactate Pro and Accu-Chek Advantage device were 3.1% and 2.6%, respectively.

Muscle biopsies were obtained using the Bergström needle-biopsy technique pre-GXR and 30 min post-ISE during the single (baseline) session and pre-GXR on Day 2 of both experimental sessions. During biopsy extraction, participants laid supine with the biopsy site selected in the belly of the vastus lateralis muscle. Once the site was identified the muscle was anaesthetized by injection of 1.5ml Xylocaine under the skin. A small incision was made whereby a muscle biopsy needle was inserted and with manual suction a small piece (~80-100 mg) of muscle was extracted from the vastus lateralis muscle of the left thigh (to avoid interference with the electrical stimulation site). Pre and post exercise samples were extracted from the same incision, while post-exercise with the biopsy needle angled proximal to the previous pre-GXR sample site. Subsequent biopsy incision was made distal to the previous incisions. Biopsy samples were immediately frozen with liquid nitrogen and stored at -80°C until further analysis of muscle glycogen concentration. A small piece (4 – 6 mg) of freeze-dried muscle was removed and dissected of visible blood, fat and connective tissue. One aliquot of freeze-dried muscle was homogenised for one minute in perchloric acid. To determine muscle glycogen, homogenate was diluted with 350µL of 2M HCl, and incubated in a heating block at 100°C for 2 hours. Following incubation the homogenate was reweighed vortexed and neutralized to a pH 7-8 with 2M K2CO3. The glycogen aliquots were centrifuged and supernatant used for analysis. Muscle glycogen concentration was assessed using a spectrophotometer to assess absorbance with the addition of glucose-6-phosphate dehydrogenise followed by hexokinase. Glycogen concentration was expressed as millimoles of glycogen per kilogram of dry weight (dw).
Perceptual measures of Rating of Perceived Exertion (RPE) and a modified Profile of Mood States (POMS) were used throughout the protocol. RPE was recorded every 10 min during ISE protocol, while mood state was assessed using the modified POMS test assessing moods (lively, alert, energetic and fatigued) that were deemed applicable to this particular study pre-GXR and 20 min post ISE protocol.

Statistical Analysis
Data are reported as mean ± SD. Initially, a Shapiro-Wilk test was used to confirm that data not differ substantially from a Gaussian distribution. A repeated measures analysis of variance (ANOVA) (time x day x condition) was used to determine main effects within and/or between each experimental condition. Significance was set at \( P = 0.05 \). A paired t-test was used to determine differences in muscle glycogen concentration between conditions. A Tukey’s post hoc was then applied where appropriate to determine statistical significance. Additionally, Pearson product moment correlation was completed to assess the relationship between pre-exercise muscle glycogen concentration and mean sprint time, total distance covered and hard running distance covered.

Results
Sleep Duration
No differences were present in mean sleep duration during the two nights preceding the experimental sessions, with \( 8.1 \pm 1.6 \) h sleep during CONT and \( 8.4 \pm 1.5 \) h sleep during SDEP \( (P > 0.05) \), indicating regular and consistent sleeping patterns were evident for all participants. Sleep duration was significantly different between CONT \( (8.5 \pm 1.7 \) h) and SDEP \( (0 \pm 0 \) h) conditions during the evening of the experimental trials \( (P = 0.01) \).
**Intermittent-Sprint Performance and Pacing Strategies**

Sleep deprivation resulted in significant differences between conditions for mean sprint times, with slower times recorded for 8 of the 10 participants during SDEP2 compared to CONT2 ($P= 0.05$; Table 4.1). Further, within the SDEP condition, mean and total sprint times were significantly slower during SDEP2 compared to SDEP1 ($P= 0.04$). Pacing of maximal sprint efforts appeared to be affected by sleep loss, with mean sprint times slower during the initial and final 10 min during SDEP2 compared to CONT2 ($P= 0.01 – 0.03$, respectively). Within the respective SDEP and CONT conditions, mean sprint times were slower during the initial 10 min on Day 2 compared to Day 1 (CONT1: 2.63 ± 0.11; CONT2: 2.68 ± 0.12; SDEP1: 2.64 ± 0.18; SDEP2: 2.74 ± 0.15 s; $P< 0.02$). Moreover, during the 11-20 min, 31-40 min, and 41-50 min phases, mean sprint times were slower during SDEP2 than SDEP1 ($P= 0.03 - 0.05$).

Total distance covered during the self-paced, sub-maximal exercise bouts was not significantly different between conditions, with 6 of 10 participants performing worse during SDEP2 compared to CONT2 ($P > 0.05$). However, distance covered during SDEP2 was significant lower during initial and final 10 min compared to SDEP1 ($P= 0.01$; Figure 4.1A) and lower during final 10 min compared to CONT2 ($P= 0.02$; Figure 4.1A). No significant differences were evident within or between conditions for mean and total hard running, jogging and walking distance covered with 6, 4, and 5/10 participants respectively performing worse after sleep deprivation ($P > 0.05$; Table 4.1). Finally, distance covered during double leg bounds are presented in Table 4.1 and significant differences were noted within and between conditions, with less mean and total distance covered by 8 of 10 participants during SDEP2 compared to CONT2 ($P= 0.01$) and SDEP1 ($P= 0.01$) respectively.
Table 4.1: Mean ± SD mean sprint times and total distance covered (TDC) during respective sub-maximal exercise modes for the control condition on Day 1 (CONT1) and Day 2 (CONT2) and the sleep deprivation condition on Day 1 (SDEP1) and Day 2 (SDEP2) and percentage change (% change) between CONT2 and SDEP2.

<table>
<thead>
<tr>
<th></th>
<th>CONT1</th>
<th>CONT2</th>
<th>SDEP1</th>
<th>SDEP2</th>
<th>% change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean sprint time (s)</td>
<td>2.70 ± 0.14</td>
<td>2.74 ± 0.15</td>
<td>2.70 ± 0.16 ^</td>
<td>2.78 ± 0.17*</td>
<td>1.7 ± 2.2%</td>
</tr>
<tr>
<td>TDC (m)</td>
<td>3616 ± 306</td>
<td>3575 ± 337</td>
<td>3611 ± 279</td>
<td>3540 ± 303</td>
<td>-0.8 ± 3.5%</td>
</tr>
<tr>
<td>TDC hard run (m)</td>
<td>1896 ± 163</td>
<td>1883 ± 216</td>
<td>1917 ± 166</td>
<td>1833 ± 199</td>
<td>-2.3 ± 6.4%</td>
</tr>
<tr>
<td>TDC jogging (m)</td>
<td>1065 ± 156</td>
<td>1044 ± 146</td>
<td>1034 ± 147</td>
<td>1047 ± 115</td>
<td>1.0 ± 7.7%</td>
</tr>
<tr>
<td>TDC walking (m)</td>
<td>656 ± 59</td>
<td>649 ± 76</td>
<td>659 ± 53</td>
<td>659 ± 63</td>
<td>2.0 ± 5.4%</td>
</tr>
<tr>
<td>TDC bounds (m)</td>
<td>132 ± 13</td>
<td>135 ± 16</td>
<td>130 ± 13 ^</td>
<td>128 ± 13 *</td>
<td>-5.2 ± 5.4%</td>
</tr>
</tbody>
</table>

* Significant difference between CONT2 and SDEP2 (P= 0.01)
^ Significant difference between SDEP1 and SDEP2 (P= 0.01)
Figure 4.1: Mean ± SD A) total distance covered during self-paced bouts during 10 min phases of intermittent-sprint exercise (ISE), B) mean distance covered during hard running efforts, C) mean sprint times during ISE with (CONT1 and CONT2) or without sleep (SDEP1 and SDEP2).

* Significant difference between CONT2 and SDEP2 ($P=0.02$)

^ Significant difference between SDEP1 and SDEP2 ($P=0.01$)
Maximal Voluntary Force and Voluntary Activation

MVF was reduced post-ISE during SDEP1 compared to CONT1 ($P = 0.02$; Figure 4.2A), and post-ISE in SDEP2 compared to CONT2 in 8 of 10 participants ($P = 0.04$). MVF was reduced pre-GXR on SDEP2 compared to SDEP1 ($P = 0.01$). Percentage change between CONT2 and SDEP2 pre-exercise MVF was $11.7 \pm 9.0\%$ while post-exercise was reduced to $4.7 \pm 9.7\%$. VA was significantly reduced in SDEP2 compared to CONT2 for both pre-GXR and post-ISE in 8 of 10 participants ($P = 0.03$ and 0.05, respectively; Figure 4.2B). Moreover, VA was reduced pre-GXR in SDEP2 compared to SDEP1 ($P = 0.01$). Pre-exercise VA percentage change between CONT2 and SDEP2 was $9.9 \pm 10.2\%$ while post-exercise was $6.3 \pm 9.7\%$.

Physiological

Muscle glycogen concentration was significantly reduced in all participants post-exercise ($122 \pm 64$ mmol/kg $dw$) compared to pre-exercise ($310 \pm 67$ mmol/kg $dw$) during the baseline session ($P = 0.001$). Pre-exercise glycogen content was significantly lower in SDEP2 ($209 \pm 60$ mmol/kg $dw$) compared to baseline ($P = 0.03$) in all participants, while no differences were present between baseline and CONT2 ($274 \pm 54$ mmol/kg $dw$) ($P > 0.05$). Further, muscle glycogen was significantly lower pre-exercise in SDEP2 compared to CONT2 ($P = 0.05$) and percentage change between conditions was $24.5 \pm 10.7\%$. No significant correlations ($P > 0.05$) were evident between pre-GXR muscle glycogen concentration and mean sprint times ($r = 0.13$), total distance covered ($r = 0.22 - 0.28$) and hard running distance covered ($r = 0.2 - 0.3$).

No significant differences were evident within or between conditions for pre-GXR HR (CONT1: $68 \pm 5$; CONT2: $68 \pm 10$; SDEP1: $66 \pm 8$; SDEP2: $62 \pm 9$bpm; $P > 0.05$) or pre-ISE HR (CONT1: $98 \pm 5$; CONT2: $97 \pm 11$; SDEP1: $104 \pm 8$; SDEP2: $100 \pm 9$bpm; $P > 0.05$). Mean HR throughout the ISE protocol was significantly lower on Day 2 compared to Day 1 within both SDEP and CONT conditions, respectively (CONT1: $176 \pm 8$; CONT2: $173 \pm 8$; SDEP1: $178 \pm 9$; SDEP 2: $171 \pm$
Moreover, mean heart rate was lower at 10, 20, 30 and, 50 min of ISE during SDEP2 compared to SDEP1 ($P = 0.01 - 0.03$) and lower at 20 min during CONT2 ($P = 0.04$; Figure 4.3B). Peak heart rate was not significantly different within or between conditions (CONT1: $184 \pm 8$; CONT2: $183 \pm 8$; SDEP1: $185 \pm 8$; SDEP2: $179 \pm 9$bmp; $P > 0.05$). $T_{core}$ (Figure 4.3b) was not different ($P >0.05$) within or between conditions at any stage of the exercise protocol on respective days. There were also no differences in USG within or between conditions (CONT1: $1.005 \pm 0.002$; CONT2: $1.004 \pm 0.002$; SDEP1: $1.011 \pm 0.013$; SDEP2: $1.004 \pm 0.002$; $P >0.05$).

Differences in sweat rate, indicated by change in nude mass were not different within the SDEP condition or between conditions, however, were evident within CONT with greater sweat rate on Day 2 ($1.4 \pm 0.6$ L) compared to Day 1 ($1.1 \pm 0.4$ L) ($P = 0.008$). Mean ISE blood lactate concentrations were lower on Day 2 within CONT condition compared to Day 1 ($P = 0.01$; Table 4.2). Finally, blood glucose concentrations were higher on Day 2 compared to Day 1 within the CONT and SDEP conditions ($P = 0.01$ and 0.04, respectively), but was lower in SDEP2 compared to SDEP1 at 30 min ISE ($P = 0.05$; Table 4.2).

**Perceptual**

No significant differences and trivial effects were evident within and between conditions for RPE ($P >0.05$; Figure 4.3C). Differences were noted within and between conditions for the moods ‘lively’, ‘alert’, ‘energetic’ and ‘fatigued’ assessed in the modified POMS questionnaire. Between conditions, participants rated to be less ‘lively’ pre-GXR on SDEP2 compared to CONT2 ($P = 0.01$; Table 4.3). Furthermore, pre-GXR participants were less ‘alert’, ‘energetic’, ‘lively’ and more ‘fatigued’ on SDEP2 compared to SDEP1 ($P = 0.01 - 0.02$). Finally, participants felt more ‘fatigued’ pre-GXR on CONT2 compared to CONT1 ($P=0.04$; Table 4.3).
Figure 4.2: Mean ± SD A) maximal voluntary force (MVF) and B) voluntary activation (VA), measured pre-GXR (PRE) and post-ISE (POST) either with (CONT1 and CONT2) or without sleep (SDEP1 and SDEP2).

* Significant difference between CONT2 and SDEP2 ($P = 0.03 – 0.05$)

^ Significant difference between SDEP1 and SDEP2 ($P = 0.01$)

# Significant difference between CONT1 and SDEP1 ($P = 0.04$)
Figure 4.3: Mean ± SD A) heart rate, B) core temperature, C) RPE ratings pre-exercise and during the 50-min ISE protocol either with (CONT1 and CONT2) or without sleep (SDEP1 and SDEP2).

* Significant difference between CONT2 and SDEP2 ($P = 0.04$)

^ Significant difference between SDEP1 and SDEP2 ($P = 0.01-0.03$)
Table 4.2: Mean ± SD capillary blood glucose (Glu) and lactate (La') measured throughout the 50-min intermittent-sprint protocol for the control condition on Day 1 (CONT1) and Day 2 (CONT2) and the sleep deprivation condition on Day 1 (SDEP1) and Day 2 (SDEP2).

<table>
<thead>
<tr>
<th></th>
<th>Pre GXR</th>
<th>Pre-ISE</th>
<th>30min ISE</th>
<th>50min ISE</th>
<th>Mean ISE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Glu (mmol/L)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT1</td>
<td>5.3 ± 0.4</td>
<td>5.6 ± 1.0</td>
<td>6.8 ± 1.4</td>
<td>6.5 ± 1.0</td>
<td>5.8 ± 0.8 #</td>
</tr>
<tr>
<td>CONT2</td>
<td>5.0 ± 0.5</td>
<td>5.5 ± 1.0</td>
<td>6.6 ± 0.9</td>
<td>6.0 ± 0.7</td>
<td>6.3 ± 0.8</td>
</tr>
<tr>
<td>SDEP1</td>
<td>5.7 ± 0.9</td>
<td>5.9 ± 0.9</td>
<td>7.3 ± 1.7 ^</td>
<td>6.8 ± 1.7</td>
<td>6.0 ± 0.8 ^</td>
</tr>
<tr>
<td>SDEP2</td>
<td>5.2 ± 0.4</td>
<td>5.8 ± 1.0</td>
<td>6.8 ± 1.4</td>
<td>6.1 ± 0.9</td>
<td>6.4 ± 1.0</td>
</tr>
<tr>
<td><strong>La' (mmol/L)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT1</td>
<td>1.7 ± 0.9</td>
<td>4.1 ± 1.3</td>
<td>9.6 ± 4.3</td>
<td>11.7 ± 5.0</td>
<td>10.9 ± 3.8 #</td>
</tr>
<tr>
<td>CONT2</td>
<td>1.6 ± 0.7</td>
<td>3.9 ± 1.8</td>
<td>9.9 ± 4.2</td>
<td>9.9 ± 4.2</td>
<td>9.1 ± 3.7</td>
</tr>
<tr>
<td>SDEP1</td>
<td>1.5 ± 0.8</td>
<td>3.5 ± 2.1</td>
<td>12.7 ± 5.4</td>
<td>10.2 ± 3.3</td>
<td>11.4 ± 3.9</td>
</tr>
<tr>
<td>SDEP2</td>
<td>1.9 ± 0.3</td>
<td>4.0 ± 2.8</td>
<td>9.2 ± 3.2</td>
<td>9.2 ± 4.2</td>
<td>9.2 ± 3.3</td>
</tr>
</tbody>
</table>

^ Significant difference between SDEP1 and SDEP2 (P = 0.05)
# Significant difference between CONT1 and CONT2 (P = 0.01)
Table 4.3: Mean ± SD of moods assessed in a modified POMS questionnaire pre-GXR and post-ISE for the control condition on Day 1 (CONT1) and Day 2 (CONT2) and the sleep deprivation condition on Day 1 (SDEP1) and Day 2 (SDEP2).

<table>
<thead>
<tr>
<th></th>
<th>CONT1</th>
<th>CONT2</th>
<th>SDEP1</th>
<th>SDEP2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lively</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre GXR</td>
<td>3.2 ± 0.9</td>
<td>3.1 ± 1.0</td>
<td>3.2 ± 0.6 ^</td>
<td>2.2 ± 0.8 *</td>
</tr>
<tr>
<td>Post ISE</td>
<td>2.4 ± 0.9</td>
<td>2.3 ± 0.8</td>
<td>2.4 ± 1.1</td>
<td>2.3 ± 0.2</td>
</tr>
<tr>
<td>Alert</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre GXR</td>
<td>3.6 ± 1.1</td>
<td>2.7 ± 1.0</td>
<td>3.1 ± 0.7 ^</td>
<td>2.3 ± 0.7</td>
</tr>
<tr>
<td>Post ISE</td>
<td>3.0 ± 0.8</td>
<td>2.7 ± 0.5</td>
<td>3.0 ± 0.7</td>
<td>2.7 ± 1.0</td>
</tr>
<tr>
<td>Energetic</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre GXR</td>
<td>3.3 ± 1.1</td>
<td>2.5 ± 0.9</td>
<td>3.1 ± 1.1 ^</td>
<td>2.0 ± 0.9</td>
</tr>
<tr>
<td>Post ISE</td>
<td>2.3 ± 1.2</td>
<td>1.6 ± 1.0</td>
<td>2.3 ± 1.1</td>
<td>1.6 ± 1.0</td>
</tr>
<tr>
<td>Fatigued</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre GXR</td>
<td>1.7 ± 0.9 #</td>
<td>2.9 ± 1.4</td>
<td>2.0 ± 0.2 ^</td>
<td>3.5 ± 1.0</td>
</tr>
<tr>
<td>Post ISE</td>
<td>3.2 ± 1.4</td>
<td>3.9 ± 0.9</td>
<td>3.1 ± 0.9</td>
<td>4.1 ± 0.7</td>
</tr>
</tbody>
</table>

* Significant difference between CONT2 and SDEP2 (P = 0.01)
^ Significant difference between SDEP1 and SDEP2 (P = 0.01 – 0.02)
# Significant difference between CONT1 and CONT2 (P = 0.04)
Discussion

The aim of the present study was to determine the effect of sleep deprivation on pacing strategies, intermittent-sprint performance and physiological and perceptual recovery on consecutive days of exercise. Results indicated that ~30 h sleep deprivation between simulated team sport exercise bouts resulted in slowed pacing strategies, reduced intermittent-sprint performance, muscle glycogen content, peak MVF and VA, and increased negative mood states. The reduction in performance was most evident with slower sprint times, significantly less distance covered during double leg bounds and a reduction in distance covered within SDEP during initial 10 min and compared to CONT during the final 10min of self-paced bouts of the ISE. Despite the findings, it is difficult to determine if sleep loss exclusively, or associated factors were directly responsible for the observed performance decrements.

Some variation regarding experimental design and subsequent results are evident within the previous literature examining the effects of sleep disruption on exercise performance (Martin, 1981; Martin and Haney, 1982; Mougin et al, 1991; Souissi et al. 2003). The current study utilised a self-paced exercise protocol, designed to allow participant manipulation of exercise intensities throughout the protocol, similar to team sport exercise. In agreement with the decline in self-paced distance covered in the present study, Oliver et al., (2009) reported reductions in distance covered during a 30 min self-paced treadmill run following one-night sleep deprivation with no significant effects on $T_{core}$, $T_{skin}$ and HR. Martin and Haney (1982) also used a self-paced protocol, however, self-selection was achieved through the manipulation of the treadmill grade to exercise at a set RPE following either 30 h sleep loss or a normal sleep. The differing protocols may explain the dissimilar findings, as Martin and Haney (1982) reported sleep loss had no effect on absolute treadmill grades. On the contrary, examinations of incremental tests have reported similar findings to the present study in that sleep loss negatively affects exercise performance (Mougin et al. 1991). The present study highlighted sleep loss reduced the volume of work completed, while Martin (1981) reported an 11% decrease in time to
exhaustion during heavy treadmill walking and Mougin et al., (2001) reported partial sleep loss through late bedtimes and early rises reduced maximal work rate compared to baseline during an incremental test to exhaustion. That said, a previous study by Mougin and colleagues (1991) reported no effect on time to exhaustion test when exposed to a disrupted night sleep. The inconsistencies within the existing literature may be due to the variations in protocol design, and therefore the degree of influence on physiological and perceptual parameters and thus performance responses remain unclear. Regardless, this study is the first to utilise simulated team-sport activity, with sleep deprivation seemingly having a negative effect on intermittent-sprint activity.

While differences in muscle glycogen content were small (65mmol kg\(^{-1}\) dw) between conditions in the present study, sleep loss significantly reduced muscle glycogen concentrations pre-exercise on Day 2 compared to a normal night sleep. As the exercise protocol and food consumption for the baseline session and experimental trials were identical it could be assumed muscle glycogen depletion would be similar following Day 1 during the experimental trials. Interestingly, despite the consumption of an identical diet between conditions, participants presented with reduced muscle glycogen concentrations in SDEP2. Due to the standardisation of the diet and supervision by the research team, either a difference in the timing of food consumed and/or the additional energy expended while in a sleep deprived state may be responsible for the reduced muscle glycogen concentration. It has been reported sleep aids in the reduction of energy expenditure below rest alone by 10 – 15% (Opstad et al. 1980), reduces metabolic requirements, and is involved in energy conservation (Driver and Taylor, 2000). Moreover, it has also been reported that when the same volume of food and CHO are ingested, the frequency of food ingestion may not be a major factor on muscle glycogen concentration (Burke et al. 1996). Accordingly, it seems evident that the additional energy expenditure while awake may be responsible for the reduced glycogen content evident on Day2 during SDEP. Accordingly, although functionally the differences in glycogen content may not have been large, it is worth noting that
following sleep deprivation, athletes may commence exercise bouts with lower muscle glycogen content, which may be harmful to prolonged endurance bouts (Babault et al. 2006).

Previous research indicates a state of reduced muscle glycogen may negatively affect exercise performance (Balsom et al. 1999a); thus the reduction in muscle glycogen during SDEP2 may have had a contributing effect on the subsequent performance decline. Balsom et al. (1999b) reported a low-CHO diet significantly reduced pre-exercise muscle glycogen compared to a high-CHO diet, resulting in a 33% reduction in high-intensity exercise during small-sided football games. Additionally, Winnick et al. (2005) reported the ingestion of a CHO solution improved shuttle running and vertical jumps in team sport athletes and maintained central nervous system (CNS) function in the latter stages of exercise compared to a placebo. Although the present study involved a standardised diet, similar findings are evident in that reduced muscle glycogen contributes towards a reduction in high-intensity efforts during self-paced exercise. Similar to the present study, Rauch et al., (2005) reported a down-regulation of pacing strategies during a 2 h cycling time trial during a non-CHO loaded compared to a CHO loaded diet. Rauch et al., (2005) suggests the down-regulation in pacing may be related to integrated feedback from the periphery regarding muscle glycogen content. However, due to the smaller difference in muscle glycogen concentration between conditions in the present study, and the supporting analysis which highlighted no correlation between glycogen content and performance parameters, it is likely muscle glycogen content was not the sole contributing factor to performance declines. It may be suggested that performance and pacing declines were attributed to a combination of sleep deprivation itself and reduced glycogen content and perceptual/mood states following sleep loss.

The reductions in pre-exercise peak MVF on SDEP2 may suggest that sleep loss and/or muscle glycogen content had a direct effect on recruitment of the exercising muscles (Nybo, 2003; Rauch et al. 2005). It is, however unclear as to the level of contribution sleep itself and/or reduced muscle
glycogen had on exercise performance, either of which may relate to the reductions observed in MVF. In relation to the effect of sleep loss on peak force, Bulbulian et al., (1996) previously reported a combined 30 h sleep loss and exercise to reduce flexion and extension peak torque but no effect on a calculated fatigue index; while Symons et al. (1988) reported no effect of 60 h sleep loss on maximal isometric or isokinetic strength of the upper and lower body. Alternatively, St Clair Gibson et al., (2001) reported reduced muscle glycogen concentration to have no effect on maximal isometric contraction force, while Nybo (2003) reported a reduction in mean force and following placebo compared to glucose supplementation. Nybo (2003) also reported exercise-induced hypoglycaemia reduced CNS activation during muscular contractions which may be influenced by feedback signals from the active muscles. These findings by Nybo (2003) concur with the present study in which reductions in VA and pacing strategies were evident during the self-paced exercise bouts; however, the extent to which sleep loss \textit{per se} affected, as opposed to the consequence of reduced glycogen content affect muscle recruitment remains equivocal.

Another possible contributor to the reduction in performance is the elevation of negative mood states and suppression of positive feelings. Sleep loss has been reported to be associated with detrimental effects on mood and perception, primarily assessed through the use of the Profile of Mood States (POMS) questionnaire (Martin and Haney, 1982). The present study used a modified, shortened POMS questionnaire with the moods included based on applicability for the study design. Despite this shortened version, the results suggested similar findings to Martin and Haney (1982) in which participants’ moods were affected by sleep loss, despite minimal differences in reported RPE. Given only a small difference in muscle glycogen content was present, it may be suggested that the increased perceptual strain following sleep disruption may have negatively affected pacing strategies and thus reduced intermittent-sprint performance. The effect of negative mood states on exercise performance is supported by Marcora (2009) who reported a state of mental fatigue reduced exercise performance and time to exhaustion during a cycling trial at 80% peak power output. In the present study, despite
increased levels of fatigue and tiredness during SDEP2, it appeared sleep loss had minimal affect on RPE during the ISE. The minimal effect of sleep on RPE may be due to the participants not exercising at perceived maximal effort at any stage of the exercise protocol, with a progressive increase in RPE during the ISE protocol. The lack of difference in perceived exertion within and between conditions may also be associated with the reduction in work which was completed on Day 2 (Oliver et al. 2009). Due to the self-paced nature of the protocol, participants reduced their workload during SDEP2 to defend a similar rating of perceived exertion to that in CONT2, at the expense of performance.

As self-paced exercise allows the continual adjustment in exercise intensity athletes may also have greater control over physiological responses to the given exercise bout through manipulation of pacing strategies. During the present study, heart rate was significantly lower on Day 2 within both experimental trials which may be due to the slower sprint speeds and reduced distance covered during the self-paced exercise efforts. Vaara et al., (2009) also reported sleep loss reduced heart rate; however, no exercise protocol was present in this study and was in a rested state which is difficult to compare to the present study. Oliver et al., (2009) reported dissimilar findings to the present study in that core temperature was reduced during 30 min of self-paced treadmill running following sleep deprivation. However, core temperature in the present study was not different between conditions even at rest, suggesting sleep deprivation had minimal effect on thermoregulatory function during the subsequent day (Oliver et al. 2009). Despite the increased hours of wakefulness, USG measures showed all subjects presented in a euhydrated state prior to each testing session, excluding differences in hydration as a possible cause of reductions in pacing strategies. Once exercise commenced, sweat rate was greater in CONT2 compared to CONT1 but not different at any other time within or between conditions. These findings are similar to Sawka et al., (1984) who reported a reduction in evaporative heat loss in moderate environmental conditions was due to sleep deprivation despite no differences in core temperature. It appears in the present study that due to the self-paced nature of the protocol, decreased physiological responses following sleep loss may be explained by the reduced sprinting
speed, distance covered during sub-maximal bouts and muscular activation, and in moderate conditions, sleep loss has minimal effect on thermoregulation.

The effect of sleep loss on pacing strategies during intermittent-sprint exercise has not been previously examined, however previous research unrelated to sleep loss suggests factors such as reduced muscle glycogen content (Opstad et al. 1980) and negative perceptual stress (Marcora et al. 2009) to be linked to declines in pacing strategies and exercise performance. In relation to the present study, pacing appeared to be manipulated during the exercise protocol with a progressive decline in distance covered throughout the protocol in both conditions. Interestingly though, participants were able to increase exercise intensity during the final self-paced efforts, with less distance covered during this ‘end-spurt’ during SDEP2. These findings were mirrored during the hard running efforts with a progressive decline in distance covered and a final ‘end-spurt’ by an average of 12.5 ± 2.7m, with the lowest increase during SDEP2. These findings suggest sleep deprivation and/or associated factors reduced the exercise intensity during self-paced exercise and the capacity to perform a high-intensity ‘end-spurt’, which is similar to previous work examining self-paced cycling (Kay et al. 2001). The determinants which affect the chosen intensity and allow this end-spurt are unclear, but may be related to feedback regarding the actual sleep loss, reduction in muscle glycogen concentrations (Rauch et al. 2005) or increased perceptual stress (Marcora et al. 2009). The notion of feedback from the periphery is also supported by participants covering the same distance on Day 1 between conditions, however once changes in muscle glycogen and perceptual stress were present, there appeared to be a reduction in exercise intensity. This reduction in exercise intensity coincided with reductions in MVF and VA of the right knee extensors following sleep loss. However, potential limitations of the study is that these findings may not be able to be generalised to other sub-populations due to the sample size and it is difficult to differentiate whether these changes in exercise intensity were due to the sleep deprivation and/or contributing factors associated with a lack of substantial sleep.
In conclusion, sleep deprivation negatively affects pacing strategies and intermittent-sprint performance, which appears to be associated with integrated feedback from the periphery and/or presence of negative mood states. Afferent feedback including reduced muscle glycogen concentration and increased perceptual strain may have reduced recruitment of active musculature which was evident pre-exercise during SDEP2. The level of contribution of these factors including sleep deprivation itself is however unclear. From the findings it is suggested team-sport athletes provide themselves with adequate time and conditions conducive to sleep, and if not possible, ensure additional CHO are consumed to counteract the reduction in muscle glycogen content.
Chapter 5

The Effects of Carbohydrate Intake and Muscle Glycogen Content on Self-Paced Intermittent-Sprint Exercise Despite No Knowledge of Carbohydrate Manipulation

As Published in the *European Journal of Applied Physiology*

Abstract

The aim of this study was to determine the effects of carbohydrate (CHO) ingestion and muscle glycogen content, without the influence of knowledge of CHO consumption on intermittent-sprint performance. Following familiarisation, ten males completed two conditions of differing CHO diets on two consecutive days. Day 1 involved 2 x 40 min of leg cycling separated by 15 min of arm cycling, followed by an overnight diet consuming either a high (HCHO; 7g kg^-1 bw) or low (LCHO; 2g kg^-1 bw) CHO diet. Participants were blinded to the knowledge CHO was being examined or manipulated. Day 2 included a 60 min intermittent-sprint exercise (ISE) protocol that included 15 m maximal sprints every minute and self-paced efforts of varying intensities. Pre and post-ISE muscle biopsies were obtained on Day 2 while pre and post exercise maximal voluntary torque (MVT), voluntary activation (VA) and twitch contractile properties (TCP) were assessed during 15 maximal isometric contractions. Further, blood glucose and lactate, heart rate (HR) and rating of perceived exertion (RPE) were also recorded during exercise. Pre-ISE muscle glycogen was greater in HCHO compared with LCHO (597 ± 115 vs 318 ± 72mmol kg^-1 dw; P=0.001). Performance variables show that total distance and hard running distance covered were 4.9 and 8.1% greater in HCHO, respectively (P= 0.02–0.04) while peak MVT, VA, HR and RPE were not different between conditions (P> 0.05). Blood glucose was higher pre-ISE for LCHO but lower post-ISE compared with HCHO (P< 0.05). These results indicate HCHO improved self-paced exercise intensities despite no knowledge of dietary manipulation. Due to the blinded study design, exercise intensities seem manipulated due to peripheral perturbations associated with CHO content rather than a conscious manipulation of exercise intensities.

Keywords: deception; team sports; glycogen; pacing strategies
**Introduction**

Carbohydrate (CHO) ingestion during the days before exercise is known to result in the maintenance of higher muscle glycogen content and improve the performance of both endurance (Walker et al. 2000; Rauch et al. 2005) and intermittent-sprint exercise (Bangsbo et al. 1992; Balsom et al. 1999a). Increasing dietary CHO may benefit ensuing team-sports performance given the prolonged, intermittent nature of match (and training) demands resulting in reduced muscle glycogen stores at both half (Saltin, 1973) and full-time (Krstrup et al. 2006) of a soccer match. Performance declines during prolonged, repeated-sprint exercise have been attributed to muscle glycogen depletion, including impaired sprint times that follow periods of high-intensity (> 18km h\(^{-1}\)) exercise (Krstrup et al. 2006). Further, the higher CHO availability achieved by additional CHO prior to exercise is reported to minimise the decline in glycogen stores and improve the volume of high-intensity work completed by up to 33% (Balsom et al. 1999b), increase running distance covered during a field-based intermittent-sprint test (Bangsbo et al. 1992) and increase work rate during simulated laboratory-based protocols of both short (< 10 min) and prolonged (> 30 min) durations (Balsom et al. 1999a). Although the evidence linking enhanced intake of CHO prior to exercise to improve intermittent-sprint performance is robust; few studies have provided insight into whether the mechanisms responsible for the regulation of self-paced intermittent-sprint activity are centrally or peripherally derived. Furthermore, recent research suggests that a subject’s knowledge of whether their CHO intake has been altered before and during exercise may affect performance (Clark et al. 2000; Johnson et al. 2006). To date, few studies have investigated the effect of CHO ingestion on self-paced intermittent-sprint performance while disguising the knowledge of dietary manipulation of CHO content.

Increased pre-exercise muscle glycogen content has been shown to improve pacing strategies with an improved maintenance of power output during self-paced exercise (Rauch et al. 2005). In contrast, some studies have reported no effect of increased pre-exercise glycogen stores on exercise
performance (Hawley et al. 1997a; Burke et al. 2000). These discrepancies in the literature raise questions as to the mechanisms responsible for altered performance in the presence of increased CHO intake and muscle glycogen content (Hawley et al. 1997a; Rauch et al. 2005; Winnick et al. 2005). Rauch et al. (2005) suggest that exercise intensity may be regulated throughout a given bout based on feedback from the skeletal muscle regarding glycogen content. Specifically, they suggested that based on an internal ‘gluco-stat’, exercise intensity is regulated to avoid reaching critically low glycogen stores (Rauch et al. 2005). However, the influence of perceptual factors cannot be excluded from influencing conscious exercise regulation. Johnson et al. (2006) reported minimal differences in power output during the initial 80% (2 h) of a cycling time trial when subjects were blinded to their CHO condition, leading to suggestions that initial changes in pacing during self-paced exercise may be due to their knowledge of pre-exercise CHO ingestion. Further, studies involving blinding of increased CHO availability have shown that subjects may consciously manipulate exercise intensity based on the perception of their CHO status (Clark et al. 2000; Nassif et al. 2008). Clark et al. (2000) reported that participants who were deceived into thinking they had consumed CHO during exercise had greater performance improvements, than subjects who consumed the actual CHO beverage under the pretence of it being a placebo. Collectively, these studies demonstrate that CHO ingestion, both prior to and during exercise, maintains self-paced exercise performance; however, the contribution of the physiological effects as opposed to the conscious regulation of pacing is unclear and is yet to be investigated with respect to intermittent-sprint exercise (ISE).

To date, most studies have attributed the improvements in exercise performance preceded by CHO ingestion to the increased availability of muscle glycogen stores, which permit selection of higher exercise intensities (pacing strategies) within a given time frame (El-Sayed et al. 1997; Williams et al. 1992). However, the regulation of pacing strategies during intermittent-sprint exercise based on CHO availability is yet to be closely investigated. The mechanisms responsible for altered pacing strategies including the effect of knowledge of CHO manipulation on ensuing self-paced, intermittent-sprint
performance remain unclear. Therefore, the aim of this study was to examine the effect of high vs low pre-exercise CHO ingestion following a glycogen-depleting bout of exercise and resulting muscle glycogen concentration on intermittent-sprint performance in team sport athletes without knowledge of altered CHO availability. We hypothesised that reduced CHO ingestion and resultant reduction in muscle glycogen content would reduce exercise performance and pacing strategies during intermittent-sprint exercise, despite no knowledge of altered CHO dietary intake.

Methods

Participants
Ten, physically active and moderately-trained male team-sport athletes were recruited to participate in this study. The mean ± standard deviation (SD) age, mass, height and peak aerobic capacity (VO\textsubscript{2peak}) were 20.7 ± 2.4 yrs, 73.7 ± 8.8 kg, 180.5 ± 4.5 cm and 47.2 ± 5.8 ml kg\textsuperscript{-1} min\textsuperscript{-1}, respectively. All participants were actively involved in various team-sports, participating in club representative competition and trainings sessions 3 – 4 times per week. The study was approved by Human Ethics Committee of Charles Sturt University. Verbal and written consent was obtained from all participants.

Overview
Participants initially completed a familiarisation session which also included a maximal incremental test on a cycle ergometer (LODE Excalibur Sport, LODE BV, Groningen, The Netherlands) to determine VO\textsubscript{2peak} and maximal aerobic power output (P\textsubscript{max}) to then calculate the workload to be used during the subsequent cycling protocol. Following a 3-min warm up, the VO\textsubscript{2peak} test commenced at 50W and increased by 40W every minute until volitional exhaustion (VO\textsubscript{2peak} test duration 8.5 ± 0.8min). Participants then attended the laboratory for two experimental conditions, with each condition consisting of two consecutive days of exercise. On day 1, following initial physiological, perceptual and neuromuscular measures, participants were required to complete a 95 min intermittent cycling trial. Overnight, participants then completed one of the two respective, blinded CHO
interventions; consuming either a high CHO (HCHO; 7g/kg bw) or low CHO (LCHO; 2g/kg bw) diet for the remainder of the day and breakfast the following morning. On arrival (10:00) on Day 2, participants completed resting physiological measures, including a muscle biopsy and assessments of neuromuscular function, followed by a 60 min intermittent-sprint exercise (ISE) protocol. Post-exercise assessment of neuromuscular function was completed ~ 5 min following the ISE, and a further muscle biopsy was obtained 20 min following exercise. Due to subject availability, only 7 of the 10 participants were able to complete the muscle biopsy procedures on Day 2. Muscle biopsies were not obtained on Day 1 to avoid residual muscle soreness associated with the biopsy procedures negatively affecting intermittent-sprint performance and pacing strategies on Day 2. Participants completed exercise and food diaries 24 h prior to the initial testing session, which were inspected by the research team and replicated by the subjects for the following session. Participants also abstained from strenuous exercise, alcohol, and caffeine for 24 h prior to each testing session and abstained from food and fluid (other than water) 3 h prior to each session. Environmental conditions during the cycling and intermittent-sprint protocols were standardised at 20 ± 2°C, 40 ± 5 % Relative Humidity.

Nutritional Interventions

Participants completed the two conditions in a counter-balanced, randomised order. The designation of the respective CHO interventions was double-blind and delivery of the diets was completed by an independent person who had no connection to the study. Diets were manipulated relative to the participant’s body mass, with participants consuming either a HCHO diet comprising 7g/kg bw of CHO or a LCHO diet limited to 2g/kg bw of CHO. The CHO content of each diet was quantified using food analysis software (Food Works ®, Xyris Software, Melbourne, Australia). Diets consumed during each condition were predominately liquid based, matched for volume and to disguise differences in CHO content, were similar in taste and texture and delivered in sealed opaque bottles. Both diets consisted of a small volume of food relative to body mass and 5 x 500ml liquid beverages which were manipulated to contain high or low volumes of CHO. Participants were advised of
preferred times to consume drinks (commencing ~15 min post cycling) to ensure consistency between subjects, and timing of drinks/food were documented and replicated in the following experimental session. Diets were also controlled 24 h prior to the cycling protocol, with participants instructed to maintain their normal diet and record all food and fluid ingested and physical activity completed, which was replicated during the 24 h prior to the following experimental trial. Participants also consumed 500 ml of water 1 hour prior to exercise to ensure commencement of exercise was in a euhydrated state and participants were able to consume water ad libitum during the cycling protocol, while no fluid was consumed during the intermittent-sprint protocol on Day 2. Such regulation of intake ensured the physiological or psychological effects of fluid ingestion did not interfere with pacing strategies or intermittent-sprint performance.

A novel component of this study was that participants were not aware of the actual aim of the study. Despite the study investigating the manipulation of CHO ingestion and muscle glycogen concentration, participants were informed the study would be examining two different ‘meal replacement diets for sporting teams travelling overseas’ to justify the predominately liquid diet. The study was designed and advertised with this deception to eliminate participant’s awareness of CHO content and accordingly, making a conscious decision to manipulate pacing strategies throughout the exercise based on pre-conceived ideas as to known differences of the effects of CHO intake. Participants were not informed of the study design and aim until all participants had completed testing, with later questioning confirming no participant was aware of the true aim of the study. No participant detected any difference in liquid meals relating to CHO content and no participant had any particular preference or aversion for either meal. Accordingly, the novel design and blinding of subjects to both the condition and the dietary manipulation allows investigation of the physiological and performance responses to altered CHO intake, without influence of conscious regulation.
**Exercise Protocols**

**Day 1: Cycling Protocol**

The cycling trial on Day 1 consisted of 2x40 min of lower-body cycling (Monark 828E, Monark Exercise AB, Varburg, Sweden) alternating between 12 min at 40% $P_{\text{max}}$ and 3 min at 60% $P_{\text{max}}$, determined from the prior $\bar{VO}_2\text{peak}$ test. Immediately following the initial 40 min cycling trial, participants completed 15 min of arm ergometry cycling at 25 W before repeating the 40 min leg cycling trial. The aim of this session was to deplete active musculature of glycogen content, as previous research highlights cycling protocols which are at least 90 min in duration (Burke et al. 1996; Ivy et al. 2002) and include intermittent variations in workload (Ivy et al. 1988; Symons and Jacobs, 1989) of the upper and lower-body (Keller et al. 2001) significantly reduce muscle glycogen concentration.

**Day 2: Intermittent-Sprint Exercise (ISE) Protocol**

On Day 2, participants completed a 60 min self-paced, intermittent-sprint protocol with a 1 min break every 10 min within an enclosed gymnasium. The protocol consisted of a 15 m maximal sprint every minute followed by a 5 m deceleration zone before impacting with a large upright high jump mat. Immediately after impact with the mat, participants completed one self-paced exercise bout for the remainder of the minute (~50 s) in 15 m shuttle runs along the running track. The self-paced bouts consisted of sub-maximal activity including hard running, jogging and walking. Only one self-paced exercise intensity was completed each minute and was rotated in the above order. During the hard running efforts participants were asked to cover as much distance as possible, while selecting their own pace during the jogging and walking bouts. To simulate the eccentric loading that is evident during team-sport exercise, every 7 min immediately following the 15 m maximal sprint, participants completed 8 double-leg bounds aiming to cover as much distance as possible. Participants were provided verbal encouragement during the sprints and hard running efforts that remained consistent between subjects and conditions. Participants were not advised pacing strategies were being assessed.
during the protocol to ensure no conscious manipulation of pacing strategies was implemented. The intra-class correlation ($r$) and coefficient of variation (CV) for the sprint times and total distance covered during the protocol are $r = 0.90$ and 0.96 and CV = 1.9 and 1.5\% respectively (Chapter 3).

**Measures**

*Intermittent-Sprint Performance and Pacing Strategies*

Maximal 15 m sprint time, and distance covered during respective self-paced exercise bouts were recorded each minute of the protocol. Sprint times were measured using an infra-red timing system (Speed Light, Swift, Australia), with mean and total sprint time calculated. Mean and total distance covered (TDC) throughout the respective sub-maximal exercise bout (hard running, jogging, and walking) were measured by manually counting meters covered. Double leg bounds were recorded by manually measuring the distance covered with a tape measure to determine mean and total distance covered over the 8 consecutive bounds. Pacing strategies implemented by the participants were assessed by segmentation of the mean and total distance covered during self-paced exercise into 10 min phases. This method allowed a closer inspection of performance decrements and changes in exercise intensity implemented throughout the ISE protocol.

*Muscle Biopsies*

Muscle biopsies were obtained using the Bergström needle-biopsy technique pre and ~20 min post-ISE on Day 2 of the experimental trials (following collection of other post-exercise measures). During biopsy extraction, participants laid supine with the biopsy site selected in the belly of the vastus lateralis muscle of the left thigh. Once the site was identified the muscle was anaesthetized by injection of 1.5ml (1\%) Xylocaine under the skin. A small incision was made whereby a muscle biopsy needle was inserted and with manual suction a small piece (~60-100 mg wet weight) of muscle was extracted. A post-exercise muscle biopsy was taken from the same incision with the needle
angled distal to the previous biopsy site. Biopsy samples were immediately frozen with liquid nitrogen and stored at -80°C until further analysis of muscle glycogen concentration.

Prior to determination of muscle glycogen concentration, muscle samples were freeze-dried and muscle fibres separated from any traces of non-muscular connective tissues, blood or fat. The (4–6mg) freeze-dried muscle samples were homogenized for one minute in 100 volumes of 6% perchloric acid using a homogenizer (1KA, Labortechnik, Staufen) and kept on ice. To determine free glucose in the muscle, a 200µL aliquot was removed from this homogenate and neutralized to a pH of 7-8 with 2M K₂CO₃ and stored in a -20°C freezer. For determination of glycogen plus free glucose, a separate 100µL of remaining homogenate was diluted with 350µL of 2M HCl, and incubated in a heating block (Ratek DBH20D Dry Block Heater, Victoria, Australia) at 90°C for 2 h. Following incubation, a 200µL aliquot from the homogenate was removed to be neutralized to a pH 7-8 with 2M K₂CO₃. This glycogen plus free glucose aliquot and the glucose aliquot were centrifuged and the supernatant was used in the ensuing analysis. Analysis of the respective glycogen and glucose assays and duplicates were performed using a Shimadzu U.V. 120-02 Spectrophotometre. The concentration of each metabolite was calculated from the changes in absorbance at a wave length of 340nm. Absorbance was recorded for initial samples and duplicates and 10 min after the addition of glucose 6-phosphate (G6P), and subsequent addition of hexokinase (HK). Glycogen plus free glucose absorbance was calculated and dilutions accounted for, with total glucose concentration subtracted from the glycogen plus free glucose concentration for determination of muscle glycogen concentration. Muscle glycogen is expressed as mmol.kg⁻¹ dry weight (dw).

**Physiological and Perceptual**

A mid-stream urine sample was collected pre-exercise on Day 1 and Day 2 of the experimental trials and measured for urine specific gravity (USG) to determine hydration status (Atago Digital Handheld Pocket Refractometer, PAL-10S, USA). Further, participants removed as much sweat as possible and
nude mass was recorded on a set of calibrated scales (HW 150 K, A&D, Tokyo, Japan) pre and post exercise on both days of the trials to determine sweat rate via change in nude mass. Heart rate was recorded via a chest monitor and wrist watch receiver (Polar FS1, Polar Electro, Oy, Finland) pre-exercise and every 5 min during the cycling and the ISE protocols. Rating of perceived exertion (RPE) was obtained using the 6 – 20 Borg RPE Scale (Borg, 1982) every 10 min during the cycling and ISE protocols.

Venous blood (1.5ml) samples were collected pre and ~15 min post-exercise on Day 1 and 2 from an antecubital forearm vein and samples were syringed from the vacutainer system for the measurement of glucose (Glu) and lactate (La) (ABL825 Radiometer, Copenhagen, Denmark). Additionally, a 100µL sample of capillary blood was collected from a hyperaemic ear lobe pre and post exercise on both days, as well as at 40 min during the cycling protocol and 30 min during the ISE protocol for the measurement of blood glucose (Glu) and lactate (La) (ABL825 Radiometer, Copenhagen, Denmark).

Neuromuscular tests of peak maximal voluntary torque (MVT), voluntary activation (VA) and evoked muscle twitch properties (TCP) during the 15 maximal voluntary isometric contractions (MVC) on the right knee extensors were assessed pre and ~5min post exercise on both days. A 3 min warm up on a cycle ergometer was performed prior to the pre-exercise neuromuscular assessment. Participants were seated upright on an isokinetic dynamometer (KinCom, Model 125, Chattanooga Group Inc, Hixon, TN, USA) with the knee and hip positioned at 90° (0° represents full extension). Participants were secured with a shoulder and waist strap and a strap placed at the ankle 1 cm above the lateral malleolus. The axis of rotation of the dynamometer was aligned with the lateral epicondyle of the femur. During all MVC tests, participants completed 15 MVC’s of the right knee extensors for ~3 s, with a ~6 s recovery between each contraction. During all MVC’s, participants placed arms across their chest to ensure additional forces did not contribute to the knee extensors performance. The first and final 5 MVCs were superimposed with the electrical stimulus when peak torque was achieved (~1
s after initiation of the contraction). A potentiated evoked twitch was delivered (~1 s) after the contraction was completed when the muscle was at complete rest.

Muscle activation was achieved using a double felt-tip electrode (Bipolar felt-tip electrode, Nicolet, Cardinal Health, Madison, WI, USA) placed over the femoral nerve on the anterior thigh ~1.5 cm below the inguinal fold with pressure manually applied to the electrode at 1.5 kg (Force One FDIX, RS232, Wagner Instruments, Greenwich, CT, USA). The current was delivered via a stimulator (Digitimer Ltd, Welwyn Garden City, Hertfordshire, England) linked to a BNC2100 terminal block and connected to a signal acquisition system (PXI1024; National Instruments, Austin, Texas) which sampled all data at 2000Hz. Electrical stimulus was delivered using a single square-wave pulse with a width of 200µs, which was driven using customized software (v8.0, LabView; National Instruments). The current was applied in incremental steps until a plateau in peak twitch torque, and then the current was further increased by 10% to ensure supramaximal stimulation was achieved.

MVT was determined as the mean torque value produced during the MVC in the 50 ms prior to the delivery of the stimulus. VA levels were calculated using the twitch interpolation technique (Gandevia and McKenzie, 1988; Merton, 1954). Peak superimposed torque was determined as peak torque produced during the MVC in the 50 – 150 ms period after delivery of the stimulus. Interpolated twitch torque was determined as the peak superimposed torque minus peak voluntary torque. Mean torque-time curves from the potentiated evoke twitch contractions determined 1) peak potentiated twitch torque (Pt; highest torque obtained); 2) rate of torque development (RTD; the mean tangential slope of the twitch torque-time curve between the onset of torque development and Pt); 3) time to peak torque (TPt; time from evoke torque onset to Pt); 4) rate of relaxation (RR; the mean tangential slope of the twitch torque-time between Pt and half-relaxation time); 5) contraction duration (CD; TPt plus half relaxation time). These procedures were performed using MatLab™ Software (R2009b 7.9.0.529, The Mathworks Inc, Natick, USA).
**Statistical Analysis**

Data are presented as mean ±SD. Initially, a Shapiro-Wilk test was used to confirm data did not differ substantially from a normal distribution. A repeated-measures analysis of variance (ANOVA) (time x condition) was used to determine main effects within and/or between experimental condition. One-way ANOVA was completed to determine significant differences for means of performance markers between conditions. Significance was set at $P= 0.05$. A Tukey’s post hoc was then applied where appropriate to determine the source of significance.

**Results**

**Muscle Glycogen**

Muscle glycogen concentration was significantly greater pre-exercise on Day 2 for HCHO (597 ± 115 mmol kg$^{-1}$dw) compared to LCHO (318 ± 72 mmol kg$^{-1}$dw; $P= 0.002$). While both conditions reduced muscle glycogen content, a significantly greater post-exercise content was still evident in HCHO (HCHO: 413 ± 139; LCHO: 246 ± 96 mmol kg$^{-1}$dw; $P= 0.01 - 0.03$; Figure 5.1). Additionally, muscle glycogen utilisation was significantly different between HCHO (3.1 ± 1.6 mmol kg$^{-1}$min$^{-1}$) and LCHO conditions (1.2 ± 0.6 mmol kg$^{-1}$min$^{-1}$; $P= 0.04$).
Figure 5.1: Mean ± SD muscle glycogen concentration pre and post intermittent-sprint protocol on Day 2 for high (HCHO) and low (LCHO) conditions, respectively (n=7).

* Significant difference between HCHO and LCHO conditions (P= 0.002)
# Significantly different to pre-exercise value (P= 0.01 – 0.03)
Intermittent-Sprint Performance and Pacing Strategies

Mean and total maximal 15 m sprint times were not significantly different between conditions \((P=0.57; \text{Table 5.1})\). Self-paced efforts were negatively affected by low CHO ingestion with total distance covered during the self-paced efforts significantly greater during the HCHO condition compared to LCHO \((P=0.02; \text{Table 5.1})\). Further, greater distance was covered during self-paced efforts during most 10 min phases of the protocol \((P=0.02 - 0.04; \text{Figure 5.2A})\). These differences were mainly attributed to greater distance covered during the hard running bouts, with significantly greater mean and total distance covered during HCHO \((P=0.03; \text{Figure 5.2B})\). Specifically, reduced distances covered during most individual efforts were present following the initial effort for most subjects. While hard running was affected by pre-exercise CHO ingestion, there were no significant differences between conditions for distance covered during the jogging and walking bouts \(P = 0.31-0.46; \text{Table 5.1}\) and a trend for increased distance covered during the double-leg bounds was not significant during HCHO compared to LCHO \((P=0.06; \text{Table 5.1})\).

Physiological and Perceptual

No significant differences between conditions were evident for pre-exercise USG on Day 1 or Day 2 of the experimental trials (HCHO Day 1: 1.016 ± 0.010, Day 2: 1.008 ± 0.007; LCHO Day 1: 1.014 ± 0.007, Day 2: 1.010 ± 0.008; \(P=0.41-0.77\)). Sweat rates, measured via changes in nude mass were significantly greater on Day 2 (HCHO: 1.5 ± 0.7 kg; LCHO: 1.5 ± 0.6 kg) compared to Day 1 (HCHO: 0.6 ± 0.6 kg; LCHO: 0.4 ± 0.5 kg; \(P=0.001\)) within both experimental trials. No differences in sweat rate were evident between conditions \((P=0.72)\). Heart rate was not significantly different between conditions during the cycling \((P=0.06 – 0.80; \text{Figure 5.3A})\) or ISE protocols \((P=0.19 – 0.83; \text{Figure 5.3C})\). Further, RPE was not significantly different between conditions during the cycling \((P=0.21 – 0.81; \text{Figure 5.3B})\) and ISE protocols \((P=0.30 – 0.88; \text{Fig 5.3D})\).
Table 5.1: Mean ± SD sprint times, total distance covered (TDC), and distance covered during the hard running, jogging, walking and bounding efforts during the High CHO (HCHO) and Low CHO (LCHO) conditions.

<table>
<thead>
<tr>
<th></th>
<th>HCHO</th>
<th>LCHO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean sprint times (s)</td>
<td>2.79 ± 0.09</td>
<td>2.81 ± 0.11</td>
</tr>
<tr>
<td>Mean hard running (m)</td>
<td>127 ± 11*</td>
<td>116 ± 14</td>
</tr>
<tr>
<td>Mean jogging (m)</td>
<td>91 ± 11</td>
<td>89 ± 11</td>
</tr>
<tr>
<td>Mean walking (m)</td>
<td>54 ± 8</td>
<td>53 ± 7</td>
</tr>
<tr>
<td>Mean bounds (m)</td>
<td>17.8 ± 1.5</td>
<td>17.1 ± 1.4</td>
</tr>
<tr>
<td>TDC (m)</td>
<td>4750 ± 485 *</td>
<td>4518 ± 544</td>
</tr>
<tr>
<td>TDC hard running (m)</td>
<td>2279 ± 203 *</td>
<td>2094 ± 251</td>
</tr>
<tr>
<td>TDC jogging (m)</td>
<td>1548 ± 186</td>
<td>1519 ± 191</td>
</tr>
<tr>
<td>TDC walking (m)</td>
<td>923 ± 141</td>
<td>905 ± 122</td>
</tr>
<tr>
<td>TDC bounds (m)</td>
<td>142 ± 12</td>
<td>137 ± 11</td>
</tr>
</tbody>
</table>

* Significant difference between HCHO and LCHO ($P = 0.02 - 0.03$)
Figure 5.2: Mean ± SD a) total distance covered during each 10 min phase of the intermittent-sprint protocol and b) distance covered during the hard running effort during the intermittent-sprint protocol for high (HCHO) and low (LCHO) carbohydrate conditions respectively.  

* Significant difference between HCHO and LCHO conditions ($P = 0.001 – 0.04$)
Venous and capillary blood glucose were significantly lower pre-ISE during HCHO compared to LCHO ($P = 0.01$; Table 5.2). Capillary blood glucose was higher post-ISE during HCHO compared to LCHO ($P = 0.01$). Within both conditions, capillary blood glucose was lower post-cycling compared to pre-cycling ($P = 0.001 – 0.05$). Within HCHO, venous and capillary blood glucose were reduced pre-ISE compared to post-ISE and 30 min capillary blood glucose values ($P = 0.003$ - $0.01$). No effect of time on venous or capillary blood glucose was evident within the LCHO condition ($P = 0.51 – 0.95$; Table 5.2).

Venous and capillary blood lactate were not significantly different pre or post exercise between conditions on Day 1 or Day 2 ($P = 0.06 – 0.83$; Table 5.2). On Day 1, venous and capillary blood lactate concentrations were lower pre-exercise compared to post-exercise ($P = 0.01$) and 40 min capillary blood values ($P = 0.001$). Post-exercise capillary lactate was lower compared to 40 min within both conditions ($P = 0.001$). Within both conditions on Day 2 pre-ISE capillary blood lactate was significantly lower compared to 30 min ($P = 0.001$) and post-ISE ($P=0.001$), however only within HCHO was 30 min lactate higher than post-ISE ($P = 0.03$).
Figure 5.3: Mean ± SD a) heart rate and b) rating of perceived exertion (RPE) on Day 1 (cycling), c) heart rate and d) RPE on Day 2 (intermittent-sprint protocol) for high (HCHO) and low (LCHO) conditions, respectively.

No significant differences were evident between HCHO and LCHO conditions ($P>0.05$)
Table 5.2: Mean ± SD venous and capillary blood glucose (Glu) and lactate (La') concentrations for the High CHO (HCHO) and Low CHO (LCHO) conditions on Day 1 (cycling) and Day 2 (ISE) of the experimental trials.

<table>
<thead>
<tr>
<th></th>
<th>Day 1</th>
<th>Day 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>40 min</td>
</tr>
<tr>
<td>Venous Glu (mmol/L)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HCHO</td>
<td>4.7 ± 0.6</td>
<td>-</td>
</tr>
<tr>
<td>LCHO</td>
<td>5.2 ± 1.1</td>
<td>-</td>
</tr>
<tr>
<td>Venous La' (mmol/L)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HCHO</td>
<td>2.4 ± 3.4</td>
<td>-</td>
</tr>
<tr>
<td>LCHO</td>
<td>2.5 ± 1.3</td>
<td>-</td>
</tr>
<tr>
<td>Capillary Glu (mmol/L)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HCHO</td>
<td>5.5 ± 0.6</td>
<td>5.0 ± 0.7</td>
</tr>
<tr>
<td>LCHO</td>
<td>5.8 ± 0.9</td>
<td>5.0 ± 0.8</td>
</tr>
<tr>
<td>Capillary La' (mmol/L)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HCHO</td>
<td>1.5 ± 0.9</td>
<td>6.2 ± 1.8#^</td>
</tr>
<tr>
<td>LCHO</td>
<td>1.4 ± 0.3</td>
<td>6.2 ± 2.0#^</td>
</tr>
</tbody>
</table>

* Significant difference between HCHO and LCHO ($P=0.01$)
# Significantly different from pre exercise values within the respective conditions ($P=0.001–0.05$)
^ Significantly different to post-exercise values within the respective conditions ($P=0.001–0.03$)
Maximal Voluntary Torque, Activation and Twitch Contractile Properties

Peak MVT during the 15 isometric contractions was not significantly different between conditions pre or post-exercise on Day 1 ($P= 0.06 – 0.40$) or Day 2 ($P= 0.08 – 0.38$; Table 5.3). Within HCHO condition, post-ISE MVT was reduced compared to pre-ISE ($P= 0.05$); however, no differences were evident within LCHO condition ($P= 0.09$). Additionally, pre and post-exercise VA were not significantly different between conditions on either day of the experimental protocol ($P= 0.24 – 0.99$; Table 5.3) and no significant differences were evident within each condition on either day of exercise ($P= 0.61$).

Twitch contractile properties were not significantly different between conditions on either day of the experimental trial ($P= 0.07 – 0.84$). Within both conditions on Day 1, Pt was reduced, RTD was faster, and CD was shorter post-cycling compared to pre-cycling ($P= 0.01 - 0.02$). Within LCHO, TPt was quicker post-cycling compared to pre ($P= 0.01$), while within HCHO, RR was slower pre-cycling compared to post ($P= 0.02$). On Day 2, Pt was reduced, and TPt and RR were faster post-ISE compared to pre-ISE within both conditions ($P= 0.01 - 0.04$). CD was also shorter post-ISE compared to pre-ISE within LCHO ($P= 0.02$) while no differences were evident within HCHO.
Table 5.3: Mean ± SD peak maximal voluntary torque (MVT), voluntary activation (VA), and twitch contractile properties (TCP) including peak torque (Pt), time to peak torque (TPt), rate of torque development (RTD), rate of relaxation (RR), half relaxation time (HRT) and contraction duration (CD) for High CHO (HCHO) and Low CHO (LCHO) conditions on Day 1 (Cycling) and Day 2 (ISE) of the experimental sessions.

<table>
<thead>
<tr>
<th></th>
<th>Day 1 (Cycling)</th>
<th>Day 2 (ISE)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td></td>
<td>HCHO</td>
<td>LCHO</td>
</tr>
<tr>
<td>MVT (Nm)</td>
<td>190 ± 41</td>
<td>197 ± 57</td>
</tr>
<tr>
<td>VA (%)</td>
<td>93 ± 5</td>
<td>94 ± 9</td>
</tr>
<tr>
<td><strong>TCP</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pt (Nm)</td>
<td>70 ± 15</td>
<td>64 ± 8</td>
</tr>
<tr>
<td>TPt (ms)</td>
<td>98 ± 13</td>
<td>101 ± 20</td>
</tr>
<tr>
<td>RTD (% Pt s⁻¹)</td>
<td>1041 ± 128</td>
<td>1048 ± 213</td>
</tr>
<tr>
<td>RR (% Pt s⁻¹)</td>
<td>-673 ± 143</td>
<td>-629 ± 157</td>
</tr>
<tr>
<td>CD (ms)</td>
<td>177 ± 12</td>
<td>189 ± 21</td>
</tr>
</tbody>
</table>

# Significant difference to pre-exercise value within the respective conditions (P= 0.001 - 0.05)
Discussion

The novel aspect of the present study was that the participants were completely blind to the knowledge that CHO ingestion and muscle glycogen concentrations were being manipulated or examined. Therefore, reductions in total and hard running distances were likely to be the result of peripheral and metabolic changes rather than a conscious manipulation of exercise intensities based on knowledge of CHO availability. The reduction in exercise intensities observed immediately following the initial hard running effort, combined with the discontinuous regulation of pacing strategies and the presence of an ‘end-spurt’ in both conditions suggests that exercise intensities were regulated based on afferent feedback from the periphery regarding glycogen content (Rauch et al. 2005). It is likely such adjustments were relative to muscle glycogen content, and regulated by availability and utilisation in the absence of knowledge of CHO manipulation. No significant differences in pre-exercise neuromuscular properties suggest that peak muscle recruitment and contractile properties were not affected by muscle glycogen content or prior CHO ingestion. Moreover, the lack of differences in skeletal muscle recruitment might also explain the similar mean sprint times during the exercise protocol.

Performance variables indicate that increased pre-exercise CHO ingestion and muscle glycogen content improves self-paced intermittent-sprint exercise performance. A significant increase in total distance covered was noted in the HCHO condition and was mainly attributable to an increased distance during hard running bouts. Similarly, Bangsbo et al. (1992) reported that following a 2 day high-CHO diet (8g kg\(^{-1}\) day\(^{-1}\)), professional soccer players’ improved running distance to fatigue by ~1km compared to a control diet (4.5g kg\(^{-1}\) day\(^{-1}\)) following a 90 min intermittent running protocol. Consistent with the increased hard running efforts observed during the present study, Balsom et al. (1999b) reported a 33% improvement in high-intensity exercise during small-sided football games preceded by a high CHO diet. While the present study highlighted LCHO to reduce hard running distances, no effects on jogging and walking efforts were apparent. The lack of difference in the lower
exercise intensities compared to differences in the hard running may be due to a greater dependence on glycogen as a fuel source during the higher exercise intensities (van Loon et al. 2001); and supported by the aforementioned field-based studies observing reductions in the higher-intensities during a match (Balsom et al. 1999b; Bangsbo et al. 1992). Similarly, training studies have reported 3 weeks of training with high muscle glycogen content to improve maintenance of power output during high-intensity exercise compared to training with low glycogen content (Hulston et al. 2010; Yeo et al. 2008). Collectively, given the significant difference in self-paced performance, the present study supports previous research that low CHO intake and glycogen content may be deleterious to high-intensity, self-paced efforts during intermittent-sprint exercise (Krustrup et al. 2006; Saltin, 1973; Winnick et al. 2005).

Distance covered during the high-intensity self-paced efforts was greater following a high CHO diet; however, sprint times were not significantly different between conditions. These findings are in contrast to Balsom et al. (1999a) who reported lower CHO intake and glycogen reduced work completed during maximal 6s sprints during long (30min) and short (10min) duration protocols. Pre-exercise glycogen content during low CHO condition was substantially lower ($180 \pm 14\text{mmol}\text{l}^{-1}\text{dw}$) than the present study which may explain different performance outcomes. The inclusion of self-paced efforts between maximal sprints may also explain the difference among conditions during the hard running despite no difference in maximal sprint times. As the participants were aware of the recording of sprint times, it is possible that they made a conscious effort to maintain sprint speeds, whilst sacrificing the hard running efforts in order to do so. Further, while muscle glycogen concentrations were significantly different, participants were not completely depleted pre-exercise on Day 2 during LCHO condition, suggesting a physiological capacity to maintain sprint times, but only by compromising hard running performance. Additionally, the maintenance of sprint times during the present study may be due to the minimal effect CHO ingestion had on muscle contractile properties or muscle recruitment, evident during pre-exercise assessments of voluntary torque and activation.
Overall, the findings indicate that intermittent-sprint efforts were not significantly affected by higher or lower CHO ingestion when pre-exercise glycogen content is not depleted to very low concentrations; although the regulated and immediate decline in hard running performance was a likely contributor to the maintenance of sprint performance.

Compared with the low CHO diet, the high CHO intake of 7g/kg bw between successive days of exercise resulted in a greater pre-exercise muscle glycogen concentration. Despite no biopsy sample obtained on Day 1 (to ensure no residual soreness on Day 2), the cycling protocol on Day 1 to reduce skeletal muscle glycogen content was similar to previous studies that have shown significantly reduced muscle glycogen content (Burke et al. 1996; Ivy et al. 1988; Keller et al. 2001). Physiological and blood data on Day 1 were not significantly different, suggesting a similar physiological and metabolic state between conditions. Accordingly, it is likely the diets contributed to the differences in pre-exercise muscle glycogen content on Day 2 as HCHO met previous recommendations that post-exercise CHO ingestion of 7-12g/kg bw is needed to optimise the rate of glycogen resynthesis and restore glycogen stores to pre-exercise values (Burke, 1997; Burke et al. 2004; Fairchild et al. 2003; Hargreaves et al. 2004; Jeukendrup, 2008). Previous studies examining team sport performance (Balsom et al. 1999b; Nicholas et al. 1995; Winnick et al. 2005), and self-paced exercise performance (Johnson et al. 2006; Rauch et al. 2005) have also reported that reduced CHO ingestion between consecutive day exercise bouts attenuates glycogen concentrations prior to subsequent exercise. Although not measured, it should be noted that the glycogen depleting protocol and low CHO diet may have also reduced pre-exercise liver glycogen content, which may also be associated with reductions in subsequent exercise capacity (Casey et al. 2000) and influence pacing strategies during the present study. Notwithstanding, the current results corroborate previous findings and highlight a successful dietary intervention to increase muscle glycogen in one condition compared to the other.
Despite differences in glycogen content prior to exercise, a down regulation of pacing strategies (within the hard running bouts) became evident only after the first minute of the exercise protocol. These similar initial efforts were accompanied by no effect of CHO on neuromuscular function and suggests muscle recruitment and the initial high-intensity effort of the protocol were unaffected by muscle glycogen content. The initial maintenance of high-intensity performance is similar to previous reports that pre-exercise glycogen concentrations do not affect maximal 75 s high-intensity efforts (Hargreaves et al. 1997), or maximal strength and twitch properties of the leg extensors (Symons and Jacobs, 1989). During the present study, the reduced glycogen concentration did not impair high-intensity exercise of the initial ~60s; however, within the ensuing minutes an almost immediate regulatory adjustment was made, potentially in response to altered substrate availability (Lambert et al. 2005; Noakes et al. 2004). This is evidenced in the present study by the blinded nature of the design, providing some evidence for an adjustment in pacing possibly due to afferent feedback from the periphery regarding physiological and metabolic perturbations (Rauch et al. 2005, Tucker et al. 2009).

Due to the experimental design, glycogen stores were the predominant pre-exercise physiological difference evident between conditions on Day 2. The subsequent reduced glycogen utilisation during the LCHO condition suggests a down-regulation of pacing strategies, possibly to protect already depleted fuel substrates (Rauch et al. 2005, Tucker et al. 2009). Further, the discontinuous pacing strategies throughout the self-paced exercise (Figure 5.2a) shows that rather than a progressive, linear decline in exercise intensity during the exercise protocol, exercise intensity was regulated and adjusted continuously throughout the protocol. As suggested in previous studies (Rauch et al. 2005), the preservation of muscle glycogen content creates a ‘reserve’ to ensure completion of the exercise protocol, ideally with minimal disruption to tolerable homeostatic limits. Physiological data and RPE responses from the present study also show the regulation of exercise intensities to minimise differences in heart rate or blood lactate concentrations and RPE despite significantly different
exercise intensities. Further support for this reserve capacity is the progressive increase in RPE during the protocol for which the end-point was known, but without attaining ‘maximal’ perceived exertion. The ability to maintain a reserve may also explain the presence of an ‘end-spurt’ during the final hard running bout of the intermittent-sprint protocol. This findings was similarly reported by Rauch et al. (2005), suggesting that an end-spurt following a reserve in exercise intensity during an exercise bout may be to prevent excessive declines in muscle glycogen content. Collectively, these data suggest pacing strategies were regulated throughout the protocol to maintain similar physiological and perceptual responses and ensure completion. To this end, the difference in muscle glycogen content due to the HCHO augmented selected pacing strategies, despite no knowledge of a difference in consumed diets or glycogen content.

Such adjustments in pacing strategies during the present study were evident despite athletes having no knowledge of possible alterations in CHO manipulation or glycogen content. Interestingly, the observed reduction in exercise intensity in LCHO occurs at a very early stage of the exercise protocol, with 8 of the 10 participants exhibiting reduced distance covered during the LCHO condition following the initial hard running effort. These findings are in contrast to those of Johnson et al. (2006) who examined a double-blinded, pre-exercise CHO diet on a cycling time trial performance with no difference in power output between the high and low CHO conditions until 80% of the time trial was completed. The authors concluded that pre-exercise glycogen depletion does not influence initial work-rates during self-paced exercise and any alteration in initial selected work rate is due to the knowledge of CHO ingestion. Additionally, Marcora (2008, 2009) suggests that changes in exercise intensity are controlled by the conscious brain rather than afferent feedback from the periphery, including active musculature and the cardio-respiratory system. In the present study, a key contributor affecting the conscious regulation of exercise was missing (i.e. knowledge of CHO ingestion); therefore, alterations in exercise intensity were likely to have not been explicitly conscious control. Notwithstanding all other elements of the protocol remaining standard, removing the
provision of knowledge suggests that exercise intensities were regulated from an early stage of exercise based on afferent glycogen feedback or possibly a ‘gluco-stat’ (Rauch et al. 2005). While some conscious regulation of exercise intensity is a likely factor in any exercise bout, the present findings suggest that with a successfully blinded intervention resulting in an altered peripheral physiological state, feedback on metabolic status seems to be integrated into the regulation of self-paced exercise.

In conclusion, the present study shows that reduced pre-exercise CHO ingestion and muscle glycogen content results in altered pacing strategies, although with minimal effect on maximal sprint performance during prolonged intermittent-sprint exercise. The novel study design highlights that higher intensity pacing strategies are present despite the removal of knowledge concerning CHO manipulation. Accordingly, the alterations in pacing strategies following the initial hard running bout and overall decline in distance covered were not likely to be due to a conscious regulation of exercise intensity based on knowledge of CHO, but rather a result of perturbations at the periphery associated with prior CHO ingestion. Such changes suggest the regulation of exercise intensity may be in anticipation of further depletion from a state of substantially reduced pre-exercise glycogen content. Therefore, we conclude that the study both supports the importance of pre-exercise CHO intake for team-sport athletes, and suggests that self-paced, intermittent-sprint intensities may be regulated based on skeletal muscle glycogen content despite the removal of a conscious regulation of pacing due to the knowledge of CHO ingestion.
Chapter 6

Self-paced Intermittent-Sprint Performance and Pacing Strategies Following Respective Pre-Cooling and Heating

As published in *European Journal of Applied Physiology*

Abstract

This study examined the effects of pre-exercise cooling and heating on neuromuscular function, pacing and intermittent-sprint performance in the heat. Ten male, team-sport athletes completed three randomised, counter-balanced conditions including, a thermo-neutral environment (CONT); whole body submersion in an ice-bath (ICE) and passive-heating in a hot environment (HEAT) before 50min intermittent-sprint exercise (ISE) in the heat (31 ± 1°C). Exercise involved repeated 15 m maximal sprints and self-paced exercise of varying intensities. Performance was measured by sprint times and distance covered. Maximal isometric contractions were performed to determine maximal voluntary torque (MVT), activation (VA), and contractile properties. Physiological measures included heart rate (HR), core (T$_{core}$) and skin (T$_{skin}$) temperatures, capillary blood, and perceptual ratings. Mean sprint times were slower during ICE compared to HEAT ($P <0.05$). Total distance covered was not different between conditions but less distance was covered during HEAT in 31-40min compared to CONT and 41-50min compared to ICE ($P <0.05$). MVT was reduced post-exercise compared to post-intervention in CONT and HEAT, while VA was reduced post-intervention in HEAT compared to CONT and ICE and post-exercise compared to ICE ($P <0.05$). HR, T$_{core}$ and T$_{skin}$ during exercise were lower in ICE compared to CONT and HEAT ($P <0.05$). Sprint times and distance covered were not affected by ICE and HEAT conditions compared to CONT. However, initial sprint performance was slowed by pre-cooling, with improvements following passive heating possibly due to altered contractile properties. Conversely, pre-cooling improved exercise intensities, while HEAT resulted in greater declines in muscle recruitment and ensuing distance covered.

Key words: free-paced exercise, ice-bath, passive heating, repeat-sprint, neuromuscular function
Chapter 6 – Thermoregulation Study

Introduction

Exercise in the heat increases thermal strain and results in premature reductions in intensity during continuous (Tatterson et al. 2000; Tucker et al. 2006) and intermittent-sprint exercise (Drust et al. 2005; Maxwell et al. 1996). Despite these findings, either deliberately or unintentionally, team sport athletes often increase their pre-competition thermal load prior to exercise in the heat; primarily through passive exposure to the conditions and/or engagement in warm up protocols for the reported ergogenic benefits for high intensity activities (Bishop 2003). Conversely, pre-cooling methods are often employed by team sport athletes to counter the negative performance outcomes, allowing for improved performance and reduced physiological and perceptual consequences of exercise in the heat (Duffield and Marino 2007; Quod et al. 2006). As these conflicting heating and cooling methods are often used individually or simultaneously prior to team sport exercise, it presents a paradox for which strategy is optimal prior to intermittent-sprint exercise in the heat (Duffield and Lovell 2009). Further, the opposing nature of pre-exercise cooling and heating and the resultant effects on pacing and performance may assist in highlighting the underlying mechanisms responsible for the regulation and pacing of intermittent-sprint exercise in the heat.

Athletes often train and compete in warm environmental conditions and pre-exercise exposure to such conditions is normal practice. While athletes use active warm up procedures to obtain pre-exercise physiological benefits, passive heating can provide the benefits of increased muscle temperature, nerve conduction velocity, and decreased musculo-tendonous stiffness with minimised depletion of energy substrates (Bishop 2003). Moreover, evidence has shown that increased muscle temperature improves maximal muscle strength and power via improvements in contractile function (Bergh and Ekblom 1979). However, when the beneficial effects of increased muscle temperature contribute to an excessive elevation of core temperature ($T_{\text{core}}$), declines in exercise performance may become apparent (Gonzalez-Alonso and Calbet 2003), as observed through the earlier reduction in exercise intensity (Tucker et al. 2004). The concomitant increase in $T_{\text{core}}$ during exercise in the heat has been
related to reductions in time to exhaustion (Gregson et al. 2002), intermittent-sprint performance (Drust et al. 2005) and muscle recruitment (Morrison et al. 2004; Todd et al. 2005). These reductions are exacerbated when high exogenous or endogenous thermal loads are present prior to exercise (Gregson et al. 2002). In contrast, Ely et al. (2009) have reported a maintenance and/or improvement in running velocity in highly trained athletes despite $T_{\text{core}}$ exceeding 40°C and stable skin temperatures at 30 – 34°C. Regardless, exposure to passive heating or increased thermal load prior to exercise has potential benefits (Bishop 2003) for shorter duration, high-intensity exercise typical of team sports (Spencer et al. 2005). However, increased pre-exercise thermal load may also result in reductions in skeletal muscle recruitment and performance declines in prolonged exercise (Nybo and Nielsen 2001; Saboisky et al. 2003).

Pre-cooling interventions such as ice baths (Duffield and Marino 2007), ice vests (Cheung and Robinson 2004; Duffield and Marino 2007) or mixed methods (Duffield et al. 2010) are used to minimize exercise-induced increases in thermal load and to prevent the declines in exercise performance. Laboratory-based intermittent-sprint exercise protocols have highlighted the possible benefits of pre-cooling, with increases in prolonged intermittent-sprint peak power (Castle et al. 2006) and distance covered during sub-maximal exercise bouts (Duffield and Marino 2007). The benefits of pre-cooling interventions on performance are likely due to greater heat storage capacity to allow increased muscle recruitment and the maintenance of higher intensity pacing strategies (Cheung and Robinson 2004). Furthermore, afferent feedback from peripheral cues including thermoregulatory and cardiovascular strain may influence the exercise intensity selected during a subsequent exercise bout (Tucker et al. 2006). However, pre-cooling primarily involves methods that cool the periphery, resulting in concomitant reductions in skin and muscle temperature (Duffield et al. 2010) which are thought to reduce short duration, high-intensity exercise performance (Bishop 2003; Sleivert et al. 2001). Therefore, while pre-cooling may counter the increased thermal load exacerbated by
subsequent exercise in the heat, the reduced muscle temperature may also have negative performance consequences for team sport athletes requiring high peak power efforts (Mohr et al. 2004).

The contrasting responses of pre-cooling and pre-heating on core and muscle temperatures and subsequent intermittent-sprint exercise present a conundrum for practitioners. However, this paradox of pre-exercise thermal control also presents an interesting model to assist in understanding self-paced regulation of (team-sport) exercise intensity in the heat. Accordingly, this study aimed to examine the effect of pre-cooling and passive pre-heating on pacing and intermittent-sprint performance in warm environmental conditions. A further aim was to determine the respective neuromuscular responses to the altered pre-exercise thermal state as a potential mechanism for changes in performance. It was hypothesized that pre-cooling would reduce the decline in exercise intensity throughout the intermittent-sprint protocol compared to that observed in the control or heating condition. Further, it is also hypothesized that passive heating prior to intermittent-sprint exercise will reduce distance covered during sub-maximal efforts but maintain maximal sprint performance. Finally, it is suggested that the greater maintenance of exercise intensity observed following pre-cooling and reduction following passive heating will be relative to the extent of thermal strain and muscle recruitment.

**Methods**

**Participants**

Ten healthy, physically active males who participated in regular sub-elite team-sport exercise were recruited for this study. All participants completed on average \( \geq 3 \) training sessions and 1-2 competition games each week prior to the study. The mean \( \pm \) standard deviation (SD) age, height and mass of the participants were 20 \( \pm \) 1 years, 182 \( \pm \) 6 cm and 84 \( \pm \) 11 kg, respectively. Participants were informed of the requirements and demands of the study, and informed verbal and written consent were obtained prior to commencement of testing and following approval from the Institutional Human Ethics Committee.
Overview

Participants completed a familiarisation session to ensure acquaintance with the protocol, all testing equipment and procedures prior to the experimental trials. Participants then completed three experimental trials, with each trial conducted at the same time of day and separated by 5 – 7 days recovery. Participants were required to abstain from food and caffeine 3 h prior to testing and from physical activity and alcohol 24 h prior to testing. Food and activity diaries were completed 24 h prior to the initial testing session, replicated during the subsequent trials, and checked for conformity by the research team. A 500 ml bolus of water was ingested 60 min prior to each experimental trial to ensure a standardized hydration status on arrival and was allocated a total of 400 ml of water to consume intermittently throughout the exercise protocol. Each testing session included pre-intervention physiological measures of nude mass, urine specific gravity (USG), heart rate (HR), core and skin temperatures (T\textsubscript{core} and T\textsubscript{skin}). Neuromuscular properties were assessed during 15 maximal isometric contractions both pre and post-intervention. Participants then completed one of the 15 min thermal interventions including seating in a thermo-neutral environment (CONT), submersion into an ice bath (ICE), or seated in hot, humid environmental conditions (HEAT). Following this, participants completed a 50 min self-paced, intermittent-sprint exercise protocol in warm conditions (31 ± 1°C; 33 ± 5% relative humidity). Finally, post-exercise physiological measures were collected and neuromuscular assessments were again completed.

Cooling and Heating Interventions

Each intervention was performed for 15 min prior to the intermittent-sprint exercise (ISE) protocol. During the ice bath (ICE) condition, participants were immersed in a cold water bath (10 ± 1°C; Custom design, CSU, Australia) (Booth et al. 1997) to the suprasternal notch. During the post-intervention neuromuscular test, ice towels (5 ± 2 °C) were placed over the shoulders and torso to maintain the effects of the ice bath. During the passive-heating (HEAT) condition, participants remained seated, fully clothed with a blanket around the body and directly in front of an electric
heater within a hot, humid climate chamber (38 ± 1°C and 58 ± 4% RH). An electric floor heater was placed within 1.5 m of the participants and blanket remained around the body during the post-intervention neuromuscular test. During the control (CONT) condition, participants remained seated in a thermo-neutral environment for 15 min and during the neuromuscular test (19 ± 2 °C and 24 ± 6% RH).

**Intermittent-Sprint Exercise (ISE) Protocol**

Participants completed a 3 min jogging warm-up, increasing the intensity each minute. While warm-up is normal practice for most athletes, to determine the respective effects of the pre-exercise interventions, the warm-up was restricted to minimize the time between the intervention and commencement of the protocol, maximizing the physiological and perceptual effects of the interventions. The self-paced ISE protocol was 50 min in duration with a 1 min recovery every 10 min and was performed along a synthetic running track within an enclosed laboratory, similar to Skein and Duffield (2010). The ISE protocol included a 15 m maximal sprint each minute of the 50 min along the running track with a 5 m deceleration zone before impacting with a large crash mat. Following each maximal sprint, participants completed one self-paced, sub-maximal exercise mode consisting of either hard running, jogging, or walking in a shuttle run format along the running surface. Only one self-paced, sub-maximal exercise mode was completed each minute and rotated through each minute in the above order. Participants returned at 50 s of each minute to complete the subsequent 15 m sprint and sub-maximal effort. Every 7 min participants completed the 15 m maximal sprint followed by 8 deep-squat double-leg bounds, aiming to cover as much distance as possible. During the hard running bout, participants were instructed to ‘cover as much distance as possible’, while selecting their own pace during the jogging and walking bouts. The exercise protocol was designed to induce similar physical, physiological and perceptual demands associated with team sport exercise (Spencer et al. 2005). The intra-class correlation (r) and coefficient of variation (CV) for the maximal sprints, total distance covered and hard running efforts of this ISE protocol were r = 0.90, 0.96 and 0.82 and CV =
1.9, 1.5 and 2.2%, respectively (Chapter 3). Participants were given verbal encouragement during sprints and hard running bouts, and were aware performance measures were being recorded however were not aware that pacing strategies were being assessed to minimise conscious manipulation of exercise intensity during the self-paced efforts.

**Measures**

*Intermittent-Sprint Performance and Pacing Strategies*

Maximal 15 m sprint times, and distance covered during respective self-paced exercise bouts were recorded each minute. Sprint times were measured using an infra-red timing system (Speed Light, Swift, Lismore, Australia) with mean sprint time calculated. Mean and total distance covered throughout each respective sub-maximal exercise bout (hard running, jogging, and walking) were measured by counting meters covered using 1 m markings on the running surface. Double leg bounds were recorded by measuring the distance covered with a tape measure to determine mean and total distance over the 8 consecutive bounds. Pacing strategies implemented were assessed by segmentation of the mean sprint times and total distance covered during self-paced exercise into 10 min phases, allowing a closer inspection of performance decrements and changes in exercise intensity throughout the protocol.

*Maximal Isometric Contractions and Muscle Activation*

Neuromuscular tests of maximal voluntary torque (MVT), voluntary activation (VA) and evoked muscle twitch contractile properties (TCP) during maximal voluntary isometric contractions (MVC) on the right knee extensors were assessed pre-intervention, post-intervention and ~5 min post exercise. During all tests, participants were seated upright on an isokinetic dynamometer (KinCom, Model 125, Chattanooga Group Inc, Hixon, USA) with knee and hip positioned at 90° (0° represents full extension). Participants were secured with a shoulder and waist strap and the leg secured with a strap placed at the ankle 1 cm above the lateral malleolus. The axis of rotation of the dynamometer
was aligned with the lateral epicondyle of the femur. During all MVCs, participants placed arms across their chest to minimise additional forces. Prior to the pre-intervention MVC, a 3 min cycling warm up at 60 W was performed (Monark 828E, Monark Exercise AB, Varburg, Sweden) and once seated on the dynamometer, 2 isometric contractions each at 60, 70, 80 and 100% of maximal effort were performed. During the MVC test, participants completed 15 maximal isometric contractions of the right knee extensors and were instructed to produce a maximal effort for ~3 s, with a ~6 s recovery between each contraction. The first and final 5 MVCs were superimposed with the electrical stimulus. The stimulus was delivered when peak torque was achieved (~1 s after initiation of the contraction) and a potentiated twitch was delivered immediately after the contraction was completed when the muscle was at complete rest. Due to participants preparation the pre-intervention MVC test was ~15 min in duration, while post intervention and post-exercise MVC tests were completed within ~6 min. A ~5 min recovery was allowed between post-intervention MVC and the commencement of the ISE warm up.

Muscle activation was achieved using a double felt-tip electrode (Bipolar felt-tip electrode, Nicolet, Cardinal Health, Madison, WI, USA) placed over the femoral nerve on the anterior thigh ~2 cm below the inguinal fold with pressure manually applied to the electrode at 1.5 kg (Force One FDIX, RS232, Wagner Instruments, Greenwich, CT, USA). The current was delivered via a stimulator (Digitimer Ltd, Welwyn Garden City, Hertfordshire, England) linked to a BNC2100 terminal block and connected to a signal acquisition system (PXI1024; National Instruments, Austin, Texas, USA) which sampled data at 2000Hz. The electrical stimulus was delivered using a single square-wave pulse with a width of 200µs, which was driven using customized software (v8.0, LabView; National Instruments). The current was applied in incremental steps until a plateau in peak twitch torque, and then the current was further increased by 10% to ensure supramaximal stimulation was achieved.
MVT was determined as the mean torque value produced during the MVC in the 50 ms prior to the delivery of the stimulus. VA levels were calculated using the twitch interpolation technique (Merton, 1954). Mean torque-time curves from the potentiated evoke twitch contractions determined 1) peak potentiated twitch torque (Pt; highest torque obtained); 2) rate of torque development (RTD; the mean tangential slope of the twitch torque-time curve between the onset of torque development and Pt); 3) time to peak torque (TPt; time from evoke torque onset to Pt); 4) rate of relaxation (RR; the mean tangential slope of the twitch torque-time between Pt and half-relaxation time and relative to Pt); 5) half relaxation time (HRT; time required for Pt to decline by half and relative to Pt); 6) contraction duration (CD; TPt plus HRT). These procedures were performed using MatLab™ Software (R2009b 7.9.0.529, The Mathworks Inc, Natick, USA).

**USG, Nude Mass, and Heart Rate**

A mid-stream urine sample was obtained for measurement of urine specific gravity (USG) pre-intervention to ensure all participants commenced exercise in a euhydrated state (Refractometer 503, Now. Nippon Optical, Works Co, Tokyo, Japan). To estimate changes in body mass due to sweat loss nude mass was measured before and after exercise on a set of calibrated scales accurate to 10g (HW 150 K, A&D, Tokyo, Japan), with participants ‘toweling down’ as much as possible before stepping on the scales. Heart rate (HR) was recorded pre, every 3 min during the intervention and every 5 min during the exercise protocol with a chest heart rate monitor and wrist watch receiver (F1, Polar Electro-Oy, Finland).

**Core and Skin Temperatures and Physiological Strain Index (PSI)**

$T_{core}$ was recorded via a telemetric capsule ingested 4 – 5 h prior to exercise to ensure it had passed into the gastrointestinal tract and minimally affected by ingested food and fluid. Skin temperature ($T_{skin}$) was recorded from telemetric patches adhered 1cm below the suprasternal notch of the sternum ($T_{stern}$); mid-point of the anterior surface at the maximal girth of the forearm ($T_{arm}$); mid-point of the
anterior aspect of the quadriceps ($T_{\text{thigh}}$); and medial aspect of the maximal girth of the calf ($T_{\text{calf}}$). $T_{\text{core}}$ and $T_{\text{skin}}$ were measured pre, every 3 min during the intervention and every 10 min during the ISE protocol by a hand-held monitor which telemetrically received core and skin temperatures from the respective capsules and patches (Ingestible core temperature capsule; wireless dermal temperature patch; VitalSense®, Mini Mitter, Bend, OR, USA). $T_{\text{skin}}$ was calculated using the temperatures obtained from the four skin sites using the formula by Ramanathan (1964). Physiological Strain Index (PSI) was used to estimate increased cardiovascular and thermoregulatory strain was calculated and categorized from 0 (no strain) to 10 (very high strain) (Moran et al. 1998).

**Capillary Blood**

A 100µL sample of capillary blood was collected from a hyperaemic ear lobe pre and post-intervention, 30-min during exercise and post-exercise for the measurement of blood lactate ($L_a$), glucose (Glu) and pH (ABL825 Radiometer, Copenhagen, Denmark).

**Perceptual Ratings**

Subjective ratings were recorded pre and every 5 min during the ISE protocol including Rating of Perceived Exertion (RPE; 6 – 20 point scale) (Borg, 1982) and a 10 point Likert scale to assess Rating of Thermal Stress ranging from 1 (unbearably cold) to 10 (unbearably hot).

**Statistical Analysis**

Data are presented as mean ± standard deviation (SD). Data were assessed using a Shapiro-Wilk test to confirm normal distribution. A repeated-measures (condition x time) ANOVA was used to compared the intervention effect within and between conditions. A Tukey’s post hoc test was applied to determine the source of significance. Statistical significance was set a priori at $P=0.05$. Effect sizes (Cohen’s $d$) were calculated to determine the magnitude of effect of the interventions with $<0.20$ considered ‘trivial’, $0.20 – 0.39$ as ‘small’, $0.40 – 0.79$ as ‘moderate’, and $>0.80$ as ‘large’.
Results

Intermittent-Sprint Performance and Pacing Strategies

Maximal sprint performance was negatively affected during the ISE protocol such that overall mean sprint times were significantly slower in the ICE condition compared to HEAT because of slower mean sprint times during the initial 10 min in ICE ($P=0.01$-$0.02$; $d=0.80$; Fig 6.1A). Moreover, a significant interaction effect was present for condition x time during the maximal sprints ($P=0.001$). Total distance covered during the sub-maximal, self-paced exercise bouts were not significantly different between conditions ($P >0.05$; Table 6.1). Mean and total distance covered during the respective exercise modes (hard running, jogging, walking) were not different between conditions and main effects of time x condition interaction were not significantly different ($P >0.05$). However, reduced performance was evident in HEAT, with significantly less distance covered compared to CONT during 31-40 min and compared to ICE during 41-50 min of the protocol ($P=0.02$; $d=1.30$; Figure 6.1C). The main effect of time x condition interaction for 10-min pacing for each respective phase was significantly different ($P=0.001$). Large effects were also noted with more distance covered during ICE compared to CONT during the final 10 min of exercise ($d=0.80$). No significant differences and small to trivial effects were noted during the double leg bounds amongst conditions ($P > 0.05$; $d \leq 0.20$; Table 6.1), while the interaction between time and condition was significant ($P=0.001$).
Figure 6.1: Mean ± SD a) sprint efforts during the ISE; b) hard running efforts during the ISE; and c) total distance covered during respective 10 min phases of the ISE for control (CONT), ice-bath (ICE) and passive-heating (HEAT) conditions.

* Significant difference between ICE and HEAT conditions ($P=0.01 – 0.05$)
† Significant difference between CONT and ICE conditions ($P=0.02$)
# Significant difference between CONT and HEAT conditions ($P=0.02 – 0.04$)
Δ Large effects between CONT and ICE conditions ($d=0.8$)
Table 6.1: Mean ± (sd) total distance covered, hard running, jogging, and walking efforts and bounds during the control (CONT), ice-bath (ICE), and passive-heating (HEAT) conditions, respectively.

<table>
<thead>
<tr>
<th></th>
<th>CONT</th>
<th>ICE</th>
<th>HEAT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total distance covered (m)</td>
<td>3825 ± 329</td>
<td>3915 ± 343</td>
<td>3778 ± 309</td>
</tr>
<tr>
<td>Total sprint time (s)</td>
<td>139.1 ± 7.5</td>
<td>141.3 ± 8.1 *</td>
<td>136.3 ± 8.6</td>
</tr>
<tr>
<td>Mean sprint time (s)</td>
<td>2.80 ± 0.15</td>
<td>2.83 ± 0.16 *</td>
<td>2.78 ± 0.14</td>
</tr>
<tr>
<td>Mean hard running (m)</td>
<td>127 ± 13</td>
<td>130 ± 15</td>
<td>124 ± 16</td>
</tr>
<tr>
<td>Mean jogging (m)</td>
<td>87 ± 12</td>
<td>89 ± 12</td>
<td>87 ± 10</td>
</tr>
<tr>
<td>Mean walking (m)</td>
<td>51 ± 4</td>
<td>52 ± 5</td>
<td>51 ± 4</td>
</tr>
<tr>
<td>Mean bounds (m)</td>
<td>17.07 ± 1.33</td>
<td>17.08 ± 1.47</td>
<td>17.27 ± 1.55</td>
</tr>
<tr>
<td>Total hard running (m)</td>
<td>1898 ± 198</td>
<td>1946 ± 228</td>
<td>1855 ± 245</td>
</tr>
<tr>
<td>Total jogging (m)</td>
<td>1212 ± 174</td>
<td>1245 ± 169</td>
<td>1210 ± 144</td>
</tr>
<tr>
<td>Total walking (m)</td>
<td>715 ± 62</td>
<td>725 ± 71</td>
<td>713 ± 52</td>
</tr>
<tr>
<td>Total bounds (m)</td>
<td>119.52 ± 9.31</td>
<td>119.54 ± 10.27</td>
<td>120.87 ± 10.88</td>
</tr>
</tbody>
</table>

* Significant differences between ICE and HEAT conditions (P <0.05)
Maximal Voluntary Torque and Voluntary Activation

MVT was not significantly different among conditions pre or post-intervention or post-exercise ($P > 0.05$; Figure 6.2A). Within CONT and HEAT, MVT was lower post-exercise compared to pre- and post-intervention ($P = 0.02$; Figure 6.2A) but no difference was evident within ICE. Relative percentage change from pre-intervention to post-exercise was not significantly different between conditions but large effects were present between ICE ($3 \pm 14\%$) compared to the HEAT ($15 \pm 15\%$; $d = 1.20$) and CONT ($15 \pm 14\%$; $d = 1.20$) conditions. Mean VA was reduced post-intervention during HEAT compared to CONT and ICE ($P = 0.01 – 0.03$; $d = 0.90 – 1.80$; Figure 6.2B), and reduced post-exercise compared to ICE ($P = 0.01$; $d = 1.10$). Within CONT, VA was reduced post-exercise compared to post-intervention ($P = 0.03$; Figure 6.2B). Finally, percentage change from pre-intervention to post-exercise was significantly different between ICE ($5 \pm 19\%$) and HEAT ($-7 \pm 12\%$; $P = 0.01$; $d = 1.10$) but not different compared to CONT ($0 \pm 11\%$; $d = 0.50$).

Twitch Contractile Properties

Pt was higher post-intervention during HEAT compared to ICE ($P = 0.01$; $d = 1.60$; Table 6.2) and CONT ($d = 0.90$). Within the ICE condition, Pt was reduced post-intervention compared to pre-intervention ($P = 0.01$), and within CONT and HEAT, post-exercise Pt was reduced compared to pre and post-intervention ($P = 0.01$; Table 6.2). TPt was longer post-intervention during the HEAT condition compared to ICE ($P = 0.04$) and longer post-intervention compared to post-exercise within HEAT ($P = 0.04$). RTD was not significantly different among conditions, however, RTD was significantly greater post-exercise compared to pre and post-intervention within HEAT ($P = 0.03$; Table 6.2). RR was increased post-intervention during HEAT compared to ICE ($P = 0.05$). Within the respective CONT and ICE conditions, RR was greater post-exercise compared to pre-intervention ($P = 0.01 – 0.03$) and greater post-exercise compared to post-intervention during ICE and HEAT conditions ($P = 0.01 – 0.03$). Within all conditions, post-exercise HRT was significantly slower compared to pre- and post-intervention ($P = 0.01 – 0.04$; Table 6.2). No significant differences were
evident within or between conditions for HRT, but large effects were noted post-intervention with a shorter HRT during ICE compared to CONT and HEAT \((d= 0.90-1.20)\). Finally, CD was not significantly different among conditions pre or post intervention or post-exercise \((P >0.05; d <0.20)\); however was reduced post-exercise compared to pre- and post-intervention within all conditions \((P= 0.03; \text{Table 6.2})\).

**USG, Nude Mass/Sweat Rate, Heart Rate**

Pre-exercise USG did not differ among conditions (CONT: 1.008 ± 0.006; ICE: 1007 ± 0.003; HEAT: 1.007 ± 0.003, \(P >0.05\)). Differences in sweat rate, determined by changes in nude mass, were reduced during ICE \((0.6 ± 0.2 \text{ kg})\) compared to HEAT \((1.1 ± 0.5 \text{ kg})\) and CONT \((1.0 ± 0.5 \text{ kg})\) \((P= 0.01 – 0.02; d= 1.50 – 2.10)\). Mean heart rate during the ISE and at time points 10 – 40 min were lower during ICE compared to HEAT \((P= 0.01 – 0.05)\) and from 10 – 30 min compared to CONT \((P= 0.01 – 0.04; \text{Fig 6.3A})\). HR was lower during CONT compared to HEAT at 12 - 15 min of the intervention \((P= 0.03 – 0.05)\); however, was lower pre-exercise during HEAT \((P= 0.03; d= 0.50; \text{Figure 6.3A})\). Large effects were also noted for a lower mean ISE HR during ICE compared to CONT and HEAT \((d= 0.80)\).
Figure 6.2: Mean ± SD a) peak voluntary torque (MVT) and b) voluntary activation (VA) during 15 superimposed maximal isometric contractions pre-intervention (Pre Int), post-intervention (Post Int), and post-exercise (Post Ex) for control (CONT), ice-bath (ICE) and passive-heating (HEAT) conditions.

* Significant difference between ICE and HEAT conditions ($P=0.01$)
# Significant difference between CONT and HEAT conditions ($P=0.03$)
¥ Significant difference between post-ex compared to pre and post-int ($P=0.02$)
§ Significant difference between post-int and post-ex ($P=0.03$)
Table 6.2: Mean ± SD neuromuscular assessment of potentiated twitch contractile properties of peak torque (Pt), time to peak torque (TpT), rate of torque development (RTD), rate of relaxation (RR), half relaxation time (HRT) and contraction duration (CD) pre-intervention (Pre-Int), post-intervention (Post-Int) and post-exercise (Post-Ex) during the control (CONT), ice-bath (ICE) and passive-heating (HEAT) conditions.

<table>
<thead>
<tr>
<th></th>
<th>Pt (Nm)</th>
<th>TpT (ms)</th>
<th>RTD (% Pt s⁻¹)</th>
<th>RR (% Pt s⁻¹)</th>
<th>HRT (ms)</th>
<th>CD (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Int</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>46.9 ± 9.0</td>
<td>94.7 ± 14.6</td>
<td>1086 ± 165</td>
<td>-864 ± 154</td>
<td>61 ± 13</td>
<td>156 ± 27</td>
</tr>
<tr>
<td>ICE</td>
<td>47.5 ± 7.5</td>
<td>88.5 ± 6.5</td>
<td>1165 ± 104</td>
<td>-762 ± 177</td>
<td>70 ± 16</td>
<td>157 ± 13</td>
</tr>
<tr>
<td>HEAT</td>
<td>52.4 ± 10.3</td>
<td>93.6 ± 8.0</td>
<td>1054 ± 115</td>
<td>-851 ± 153</td>
<td>60 ± 12</td>
<td>153 ± 19</td>
</tr>
<tr>
<td>Post-Int</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>45.7 ± 9.1</td>
<td>89.1 ± 6.3</td>
<td>1159 ± 103</td>
<td>-946 ± 217</td>
<td>57 ± 17</td>
<td>147 ± 19</td>
</tr>
<tr>
<td>ICE</td>
<td>41.5 ± 8.1</td>
<td>88.6 ± 6.6</td>
<td>1133 ± 85</td>
<td>-794 ± 271$</td>
<td>70 ± 20 Δ</td>
<td>158 ± 22</td>
</tr>
<tr>
<td>HEAT</td>
<td>52.0 ± 10.5*◊</td>
<td>94.9 ± 12.1*</td>
<td>1073 ± 114</td>
<td>-958 ± 229*$§</td>
<td>56 ± 11^</td>
<td>150 ± 21</td>
</tr>
<tr>
<td>Post-Ex</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>36.8 ± 4.1¥</td>
<td>89.9 ± 15.6</td>
<td>1172 ± 156</td>
<td>-1160 ± 152△</td>
<td>45 ± 6¥</td>
<td>134 ± 19¥</td>
</tr>
<tr>
<td>ICE</td>
<td>36.4 ± 7.2</td>
<td>88.5 ± 8.4</td>
<td>1144 ± 104</td>
<td>-1075 ± 125△</td>
<td>48 ± 6¥</td>
<td>137 ± 10¥</td>
</tr>
<tr>
<td>HEAT</td>
<td>36.3 ± 3.6¥</td>
<td>86.5 ± 8.6§</td>
<td>1165 ± 111¥</td>
<td>-1143 ± 153</td>
<td>46 ± 5¥</td>
<td>132 ± 10¥</td>
</tr>
</tbody>
</table>

* Significant difference between ICE and HEAT conditions (P= 0.01-0.05)
^ Large effects between ICE and HEAT conditions (d=1.20)
◊ Large effects between CONT and HEAT conditions (d= 0.90)
Δ Large effects between ICE and CONT conditions (d=0.90)
☆ Significant difference between pre-int and post-int within respective conditions (P= 0.01)
§ Significant difference between pre-int and post-ex within respective conditions (P= 0.04)
△ Significant difference between pre-int and post-ex within respective conditions (P=0.01-0.03)
¥ Significant difference post-ex compared to pre and post-int within respective conditions (P=0.01-0.03)
**Core and Skin Temperatures, PSI and Capillary Bloods**

Pre-intervention $T_{core}$ was not different among conditions ($P > 0.05$; Figure 6.3B); however $T_{core}$ was significantly lower post-intervention during ICE compared to CONT and HEAT ($P = 0.01$). During the ISE, $T_{core}$ remained lower in ICE at 10 and 20 min compared to CONT and HEAT ($P = 0.01 - 0.05$) and at 30 min compared to HEAT ($P = 0.01$). Mean ISE $T_{core}$ was also lower during ICE ($37.93 \pm 0.36 ^\circ C$) compared to CONT ($38.50 \pm 0.80 ^\circ C$) and HEAT ($38.62 \pm 0.49 ^\circ C$) ($P = 0.01 - 0.5$; $d = 1.30 - 2.30$). $T_{skin}$ was significantly lower during ICE compared to HEAT and CONT at all time points of the intervention ($P = 0.01$; Figure 6.3C). During the ISE, $T_{skin}$ was significantly lower until 20 min ($P = 0.01 - 0.03$) and mean $T_{skin}$ was reduced during ICE compared to HEAT ($d = 0.60$). Mean PSI during the ISE was significantly reduced during ICE compared to CONT ($P = 0.03$; $d = 1.60$; Table 6.3) and HEAT ($P = 0.01$; $d = 2.40$). Furthermore, PSI was reduced at 10 – 30min of ISE during ICE compared to CONT and HEAT ($P < 0.04$; $d = 1.60 - 3.80$), and CONT was lower than HEAT at 10min ($P = 0.01$; $d = 1.80$). Finally, no significant differences were evident between conditions for capillary blood samples of pH, lactate or glucose at any stage of the experimental trials ($P > 0.05$; Table 6.3).

**Perceptual**

RPE was significantly lower during ICE at 10 min compared with HEAT ($P = 0.02$; Table 6.3) and at 15 min compared with CONT ($P = 0.02$). No significant differences were evident at any stage throughout the exercise protocol between conditions for ratings of Thermal Stress ($P > 0.05$; Table 6.3).
Figure 6.3: Mean ± SD a) heart rate (HR); b) core temperature (T_core); and c) skin temperature (T_skin) throughout the intervention and exercise during the control (CONT), ice-bath (ICE), and passive-heating (HEAT) conditions respectively.

* Significant difference between ICE and HEAT conditions ($P = 0.01 - 0.05$)
# Significant difference between CONT and HEAT conditions ($P = 0.03 - 0.05$)
† Significant difference between CONT and ICE conditions ($P = 0.01 - 0.05$)
Table 6.3: Mean ± SD physiological strain index (PSI) and perceptual ratings of perceived exertion (RPE), thermal stress (Thermal) and capillary blood measure of pH, lactate (La\(^{-}\)), and glucose (Glu) during the control (CONT), ice-bath (ICE), and passive-heating (HEAT) conditions, respectively.

<table>
<thead>
<tr>
<th></th>
<th>Pre-ex</th>
<th>10min</th>
<th>20min</th>
<th>30min</th>
<th>40min</th>
<th>50min</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PSI</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>-</td>
<td>5.8 ± 0.6 #</td>
<td>7.5 ± 1.1</td>
<td>8.3 ± 1.6</td>
<td>8.9 ± 2.1</td>
<td>7.8 ± 1.4</td>
</tr>
<tr>
<td>ICE</td>
<td>-</td>
<td>3.9 ± 1.2 *†</td>
<td>5.5 ± 1.0 *†</td>
<td>6.7 ± 1.1 *†</td>
<td>8.3 ± 1.4</td>
<td>6.4 ± 1.0</td>
</tr>
<tr>
<td>HEAT</td>
<td>-</td>
<td>6.7 ± 0.8</td>
<td>8.0 ± 1.3</td>
<td>8.7 ± 1.3</td>
<td>8.9 ± 1.4</td>
<td>8.2 ± 1.1</td>
</tr>
<tr>
<td><strong>RPE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>-</td>
<td>14.1 ± 1.4</td>
<td>16.2 ± 1.4</td>
<td>17.2 ± 1.4</td>
<td>18.1 ± 1.5</td>
<td>18.6 ± 1.4</td>
</tr>
<tr>
<td>ICE</td>
<td>-</td>
<td>13.9 ± 1.4*</td>
<td>15.8 ± 1.6†</td>
<td>17.0 ± 1.6</td>
<td>18.1 ± 1.4</td>
<td>18.8 ± 1.3</td>
</tr>
<tr>
<td>HEAT</td>
<td>-</td>
<td>14.6 ± 1.6</td>
<td>15.9 ± 1.5</td>
<td>16.9 ± 1.0</td>
<td>17.8 ± 1.0</td>
<td>18.4 ± 1.3</td>
</tr>
<tr>
<td><strong>Thermal</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>4.7 ± 0.8</td>
<td>6.0 ± 0.3</td>
<td>6.6 ± 0.4</td>
<td>7.0 ± 0.8</td>
<td>7.2 ± 0.6</td>
<td>7.3 ± 0.7</td>
</tr>
<tr>
<td>ICE</td>
<td>4.5 ± 0.8</td>
<td>5.7 ± 0.6</td>
<td>6.3 ± 0.7</td>
<td>6.8 ± 0.5</td>
<td>7.2 ± 0.6</td>
<td>7.5 ± 0.5</td>
</tr>
<tr>
<td>HEAT</td>
<td>4.3 ± 0.6</td>
<td>5.8 ± 0.7</td>
<td>6.3 ± 0.7</td>
<td>6.7 ± 0.5</td>
<td>7.0 ± 0.7</td>
<td>7.2 ± 0.6</td>
</tr>
<tr>
<td><strong>pH</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>7.34 ± 0.02</td>
<td>-</td>
<td>-</td>
<td>7.28 ± 0.04</td>
<td>-</td>
<td>7.31 ± 0.05</td>
</tr>
<tr>
<td>ICE</td>
<td>7.34 ± 0.02</td>
<td>-</td>
<td>-</td>
<td>7.29 ± 0.04</td>
<td>-</td>
<td>7.31 ± 0.03</td>
</tr>
<tr>
<td>HEAT</td>
<td>7.34 ± 0.01</td>
<td>-</td>
<td>-</td>
<td>7.30 ± 0.05</td>
<td>-</td>
<td>7.32 ± 0.05</td>
</tr>
<tr>
<td><strong>La(^{-})</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>1.2 ± 0.2</td>
<td>-</td>
<td>-</td>
<td>7.7 ± 2.9</td>
<td>-</td>
<td>7.3 ± 3.7</td>
</tr>
<tr>
<td>ICE</td>
<td>1.1 ± 0.3</td>
<td>-</td>
<td>-</td>
<td>6.9 ± 2.5</td>
<td>-</td>
<td>6.9 ± 2.6</td>
</tr>
<tr>
<td>HEAT</td>
<td>1.3 ± 0.4</td>
<td>-</td>
<td>-</td>
<td>6.9 ± 3.1</td>
<td>-</td>
<td>6.6 ± 3.0</td>
</tr>
<tr>
<td><strong>Glu</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CONT</td>
<td>5.4 ± 0.5</td>
<td>-</td>
<td>-</td>
<td>6.8 ± 1.9</td>
<td>-</td>
<td>6.5 ± 1.5</td>
</tr>
<tr>
<td>ICE</td>
<td>5.8 ± 0.7</td>
<td>-</td>
<td>-</td>
<td>6.5 ± 1.0</td>
<td>-</td>
<td>6.3 ± 0.9</td>
</tr>
<tr>
<td>HEAT</td>
<td>5.7 ± 0.7</td>
<td>-</td>
<td>-</td>
<td>6.9 ± 1.3</td>
<td>-</td>
<td>6.7 ± 1.5</td>
</tr>
</tbody>
</table>

* Significant difference between ICE and HEAT conditions (P= 0.01 – 0.04)
# Significant difference between CONT and HEAT conditions (P= 0.01)
† Significant difference between CONT and ICE (P= 0.01-0.04)
Discussion

The present study highlights the opposing effects of pre-exercise thermal interventions, in which there are both positive and negative responses to pre-cooling and passive heating prior to prolonged, self-paced intermittent-sprint exercise in the heat; despite minimal difference to the control condition. In an ergogenic sense, pre-cooling maintains higher self-selected exercise intensities during prolonged self-paced exercise compared to passive heating (Duffield et al. 2010; Kay et al. 1999) but retards sprint speed during the early phase of an exercise bout; particularly if not preceded by a warm-up of sufficient duration or intensity (Sleivert et al. 2001). Further, pre-cooling manoeuvres had minimal effect on repeated sprint times compared to a control condition. Conversely, exposure to hot environmental conditions and the resultant passive heating effect provides some augmentation of early sprint speeds, but may also result in the earlier reduction of self-paced exercise intensity, as evident between the HEAT, ICE and CONT conditions in the final 20 min of the protocol. Underlying these divergent exercise responses are alterations to the recruitment of skeletal muscle, potentially related to the endogenous thermal load and physiological state induced by the hot environmental temperatures. As such, passive heating may invoke the largest physiological and thermal load, possibly predisposing athletes to greater performance reductions during self-paced exercise. However, pre-cooling interventions to counter these exacerbated loads still require some engagement in sufficient warm-up procedures to prevent possible negative consequences on early peak speed or high-intensity efforts.

The present findings indicate that 15 min pre-cooling had a divergent effect on the respective performance-based maximal sprints and self-paced efforts during intermittent-sprint exercise. That is, the ice bath was effective in maintaining distance covered during the self-paced exercise bouts during the latter stages of the exercise protocol compared to CONT and HEAT (Figure 6.1C). Similarly, previous studies have reported pre-cooling to be beneficial for maintaining performance during prolonged intermittent-sprint exercise (Castle et al. 2006; Duffield and Marino, 2007), time to
exhaustion (Gonzalez-Alonso et al. 1999) and self-paced cycling trial performance in the heat (Duffield et al. 2010). The increased exercise intensity evident during the final stages (20 min) of the exercise protocol compared to the CONT and HEAT conditions were primarily attributed to differences in the hard running efforts and minimal effect during the jogging and walking bouts. Duffield and Marino (2007) similarly reported a smaller decline in self-paced, hard running exercise in the heat when ice-bath and ice-vests were used before exercise, and during the warm-up and half-time periods. The findings suggest pre-exercise exposure to different thermal conditions did not affect overall distance covered; however, a down regulation of exercise intensity was adapted to the respective conditions. The present study highlights overall distance and distance covered during the lower intensity jogging and walking bouts were not affected by pre-exercise thermal interventions. However, pre-cooling prior to intermittent-sprint exercise in the heat increased distance covered during the self-paced high-intensity efforts in the latter stages of a prolonged exercise; particularly compared to pre-exercise exposure to warm environmental conditions.

Although pre-cooling was advantageous during the self-paced efforts, the likely reduction in muscle temperature (Castle et al. 2006) following the ice bath negatively affected sprint performance compared to passive heating. These declines may be due to changes in post-intervention muscle contractile properties, including reductions in peak torque and time to peak torque, and longer relaxation times between ICE and HEAT conditions. Additionally, Wittekind et al. (2011) highlighted that all out pacing strategies are implemented during sprints up to 15 s; although noted a reduction in power during sprints ≥ 30sec which appears to be due to an initial pacing strategy. Based on these findings, it may be suggested that differences in maximal sprint times between conditions were not reduced due to centrally mediated factors, but rather were affected at the periphery, resulting from temperature-dependent effects on twitch contractile properties. Alternatively, cold-induced physiological disruptions such as reduced muscle blood flow, glycolytic enzyme activity or nerve conduction velocity (Bishop 2003) have also previously been reported to reduce peak force
Chapter 6 – Thermoregulation Study

development. Similarly, Sleivert et al. (2001) reported that pre-cooling the thighs and torso combined
with 6 min warm up reduced peak and mean power during a subsequent 45 s high-intensity exercise
bout; although more relevant, performance was further reduced when no warm-up was completed.
The authors suggested that the lower muscle temperature and reduction in contractile function and/or
anaerobic metabolism following the thigh/torso pre-cooling and no active warm-up was responsible
for the greatest performance decrements (Sleivert et al. 2001).

It is likely the reduced sprint times following ICE in the present study may be due to the minimal (3
min) warm-up provided. Such duration was used to reduce the time between the intervention and
exercise and thus maximize the physiological effects of the respective interventions. While not a
likely scenario in a competitive environment, the reduced warm up ensured it did not blur the thermal
effects of the intervention. However, repeat-sprint performance findings were consistent with previous
studies reporting whole body (Drust et al. 2000a; Duffield and Marino, 2007) and upper body
(Cheung and Robinson 2004) cooling to have minimal effect on maximal sprint times compared to a
control. Furthermore, the differences in twitch properties between the respective conditions further
supports the previous notion that temperature dependent changes in twitch contractile properties may
affect subsequent sprint performance. Accordingly, pre-cooling without a warm up may slow sprint
times due to lower muscle temperatures (Castle et al. 2006; Sleivert et al. 2001) and corresponding
reductions in peak torque compared to passive heating, despite also suppressing $T_{core}$ and maintaining
higher self-selected intensities during exercise in the heat.

In contrast to pre-cooling, passive heating resulted in improved initial performance during the
repeated sprints, but retarded self-paced efforts later in the exercise bout compared to ICE and CONT.
Similarly, previous reports have highlighted that passive heating via whole-body water submersion
improves mean power output during a maximal 30 s sprint (Linnane et al. 2004) and 10 x 6 s sprints
with a 34 s recovery compared to no warm-up (Brown et al. 2008). It is proposed the heating-induced
benefits to high-intensity efforts result from an increased muscle temperature contributing to vasodilatation, increased blood flow and improved muscle contractile properties (Bishop, 2003). The present study observed temperature-dependent changes to post-intervention potentiated twitch contractile properties including peak torque, time to peak torque, rate of relaxation and half relaxation time compared to the ICE condition. However, no differences were evident between conditions post-exercise once the direct thermal effects of the respective interventions had dissipated. These findings may suggest alterations in muscle stiffness, musculo-tendinous elasticity and contractile characteristics following HEAT compared to ICE. Accordingly, pre-exercise exposure to warmer thermal environments may be initially beneficial for high-intensity demands. However, the exacerbated rise in $T_{\text{core}}$ can contribute to a down regulation of exercise intensity (Tucker et al. 2004), and a reduced distance covered during the final stages of the protocol compared to CONT and ICE was observed. Previous research confirms that pre-exposure to hot environments results in an earlier performance declines during intermittent-sprint exercise (Morris et al. 1998), prolonged, continuous exercise (Craig and Froehlich 1968; Gonzalez-Alonso et al. 1999) and sub-maximal endurance performance compared to moderate conditions (Gregson et al. 2002). As evident in the present study, the accelerated rate of rise of thermal load during self-paced exercise may induce reductions in exercise intensity. Consequently, passive heating in hot conditions may improve initial peak speed or early repeated efforts due to increased muscle temperatures (Bishop 2003; Brown et al. 2008); however due to increased thermal load, may result in reduced exercise intensities during prolonged exercise in the heat.

Reductions in exercise intensity observed following passive heating are possibly due to the level of muscle recruitment from feedback regarding the imposing environmental conditions and/or endogenous load (Morrison et al. 2004; Todd et al. 2005) and may have direct effects on the central nervous system (CNS) (Nybo and Nielsen 2001). Although neuromuscular tests were not able to be performed during exercise, assessments completed pre and post intervention and post exercise
indicate a condition-specific response of voluntary torque and activation. Previous studies have indicated that hyperthermia reduces voluntary drive during maximal isometric contractions (Moran et al. 1998; Nybo and Nielsen 2001; Saboisky et al. 2003), and returns to baseline values once cooling is imposed and T\textsubscript{core} is reduced (Morrison et al. 2004; Thomas et al. 2006). In the present study, when pre-exercise T\textsubscript{core} was elevated, reductions in the volume of dynamic exercise during the final 20 min were evident with concomitant declines in post-exercise voluntary torque and activation. These results suggest centrally mediated reductions in muscle recruitment are evident when passive heating is applied prior to intermittent-sprint exercise. Despite passive heating reducing voluntary recruitment, increases in potentiated twitch peak torque, time to peak torque and relative relaxation rates suggest that increased local muscle temperatures improved nerve conduction rates and improved contractile properties of the active musculature. These changes at the muscle may explain the quicker sprint times during the starting sprints of the intermittent-sprint protocol but dissipated as exercise progressed. Therefore, contractile properties data may suggest that increases in muscle temperature associated with passive heating posed benefits to the initial maximal sprint performance and the excessive elevation in pre-exercise T\textsubscript{core} resulted in attenuated distances covered in sub-maximal modes of self-paced exercise.

The findings from the present study suggest that a down regulation of intensity during intermittent-sprint exercise may be associated with an increase in thermal load due to exercise in the heat, which are exacerbated by pre-heating. Therefore, pre-cooling may be utilized to reduce core and muscle temperatures and increase the gradient between starting temperatures and the attainment of hyperpyrexia (Wilson et al. 2002). Findings from the present study indicate the accelerated increase in T\textsubscript{core}, T\textsubscript{skin} and PSI due to pre-heating resulted in faster rates of heat accumulation and increased sweat rates compared to ICE, with minimal changes when exposed to a thermo-neutral environment. The pre-exercise exposure to warm environmental conditions may have led to a more rapid decline in distance covered during the self-paced efforts. Interestingly, similar to Duffield et al. (2010),
differences in $T_{\text{core}}$ and $T_{\text{skin}}$ had dissipated between passive heating and pre-cooling by the final stages of the protocol when differences in pacing were present. These findings suggest increased pre-exercise thermal strain may lead to anticipatory reductions in neural drive due to afferent feedback from the periphery regarding cardiovascular load, $T_{\text{core}}$ (Tucker et al. 2006) and skin temperature (Schlader et al. 2011b). Further evidence of this protective mechanism has been supported by studies showing exercise terminates at a critical temperature (Gonzalez-Alonso et al. 1999) and an anticipatory regulation of muscle recruitment during self-paced exercise to govern the rate of rise of endogenous thermal load (Marino et al. 2004; Tucker et al. 2006). However, more recent work has shown that in highly trained athletes, exercise intensity is able to be maintained and improved in the final 600m of an 8 km running trial despite core temperatures exceeding 40°C in some cases (Ely et al. 2009). In relation to the present study, the overall pacing strategy was not different between conditions; however, an attenuation of exercise intensities during the HEAT condition was evident during the latter stages of the exercise protocol when physiological perturbations had dissipated between conditions and $T_{\text{core}}$ was well below 40°C. Therefore it appears exercise intensities were regulated in response to the imposing thermoregulatory loads; however the conscious and/or subconscious contribution towards this exercise regulation is not clear.

Increased thermoregulatory strain while exercising in the heat and/or in a hyperthermic state also elevates the strain on the cardiovascular system (Gonzalez-Alonso and Calbet, 2003). These limitations to the cardiovascular system include reduced blood volume, venous return and redistribution of blood flow to the skin for heat dissipation and have been linked to performance reductions (Linnane et al. 2004; Nielsen et al. 1984). Despite less distance covered during the self-paced efforts of the intermittent-sprint protocol following HEAT, cardiovascular strain was higher compared to the ICE and CONT conditions. These elevations were observed through higher heart rates and sweat rates in conjunction with increases in core and skin temperatures and calculated PSI. Capillary blood metabolites of glucose and lactate, and pH indicate that despite working at higher
intensities during ICE, participants regulated their physiological stress to similar levels to CONT and HEAT. These results suggest that manipulation of exercise intensities following cooling and heating are possibly due to differences in cardiovascular and thermoregulatory load during the intermittent-sprint protocol. In support of this, the similar RPE and thermal stress throughout the protocol indicate that despite working at higher intensities following ICE, participants perceived the demands of the exercise and environment to be similar to HEAT. This ability to work at a higher intensity for a given RPE during the self-paced efforts may be attributed to the reduced cardiovascular and thermal strain following the ice bath rather than the observed reductions during the passive heating or control conditions.

The present study highlights the paradox between pre-exercise exposures to warm environmental conditions and pre-cooling respectively, both of which regularly occur in team sport environments, despite minimal effect compared to a thermo-neutral environment. Pre-cooling is beneficial to self-paced efforts during intermittent-sprint exercise and maintain muscular recruitment (MVT and VA), but negatively affects early sprint performance and contractile properties of the active musculature if a sufficient warm-up is not present. Conversely, pre-heating improves early sprint performance but results in earlier reductions in self-paced exercise intensities compared to control and cooling conditions. The regulation of these exercise intensities seem to be partially governed by endogenous load, and altered performance following passive heating may be linked with a down-regulation of muscle recruitment due to a faster rise in thermoregulatory strain. The present findings suggest team-sport athletes engage in pre-cooling manoeuvres before selected competition and training in the heat. However, pre-cooling should either be applied to non-active musculature to minimise temperature-dependent declines in twitch contractile properties and high-intensity exercise performance. Additionally, to capitalise on both pre-exercise cooling and heating manoeuvres for subsequent performance, athletes should incorporate warm-up procedures that increase muscle fibre temperature, while maintaining reduced core temperature. This may be achieved through the application of
portable cooling procedures including ice slushies or ice-vests, while completing active or passive warm-up procedures. In conclusion, pre-exercise exposure to warm environmental condition augmented initial sprint times, however reduced sub-maximal exercise intensities; while pre-cooling reduced cardiovascular and thermoregulatory strain and allowed for the self-selection of higher exercise intensities.
Chapter 7

General Discussion
7.1 Overview of the Thesis

Team sport athletes are subject to high physical demands that are associated with concomitant increases in physiological and perceptual loads (Coutts et al. 2010; Krstrup et al. 2006; Mohr et al. 2003). Due to the self-paced nature of team sports, exercise intensity may be continuously altered in order to regulate physiological and perceptual responses, whilst meeting the tactical demands of the match (Coutts et al. 2010). Despite the notion of athletes implementing pacing strategies during matches (Carling et al. 2008; Coutts et al. 2010), minimal research has examined the presence of pacing during intermittent-sprint exercise (Duffield and Marino, 2007; Skein and Duffield, 2010). Furthermore, lesser attention has been paid to elucidate the mechanisms responsible for pacing during intermittent-sprint exercise or apply models purporting to explain ‘fatigue’ in such exercise modes. As intermittent-sprint exercise is physically, physiologically and perceptually taxing on an athlete, an effective method to assess the mechanisms responsible for the regulation of exercise intensities during such exercise may be to selectively exacerbate these states with various pre-exercise interventions.

Accordingly, the purpose of this thesis was to examine pacing strategies during self-paced intermittent-sprint exercise. The specific aims of this thesis were to (1) examine if alterations to pre-exercise circumstances common to team sport athletes influence pacing strategies during self-paced intermittent-sprint exercise; and (2) examine the mechanisms responsible for the manipulation of pacing strategies by team sport athletes. In relation to thesis aim (1), the 3 ecologically valid pre-exercise circumstances to exacerbate pre-exercise loads included; sleep deprivation (Chapter 4), high or low CHO intake (Chapter 5) and alterations in heat stress via pre-cooling and heating (Chapter 6). In accordance with thesis aim (2) the delivery of each intervention was different between studies in order to examine if the knowledge of a given intervention or an altered physiological or perceptual state were responsible for the manipulation of pacing strategies during intermittent-sprint exercise.
7.2 Major Findings of the Respective Studies

Chapter 4 - Sleep Deprivation

As outlined in Chapter 4, the first study of this thesis examined the effect of 30 h sleep deprivation between consecutive days of exercise on pacing strategies and intermittent-sprint performance. We found that following sleep deprivation, sprint times were significantly slower and bounding distance was less compared to a normal night sleep. Total distance covered and distance covered during the respective hard running, jogging and walking efforts were not significantly different between conditions, although closer inspection of pacing strategies indicates that significantly less distance was covered during the initial and final 10 min of the protocol in a sleep deprived state. In accordance with thesis aim (1), the study successfully highlighted that sleep deprivation is detrimental to intermittent-sprint performance and pacing strategies employed compared to a normal night sleep.

In relation to thesis aim (2), the mechanisms responsible for the performance declines and changes in pacing are mainly attributed to negative mood states and moderately reduced muscle glycogen following sleep deprivation. Previous studies have reported similar findings with a noted deterioration of mood states following sleep deprivation (Meney et al. 1998; Reilly and Piercy, 1994) and an increased perception of effort for a given workload (Myles, 1985). Furthermore, neuromuscular tests on SDEP2 highlighted a reduction in force production during repeated MVC’s due to centrally mediated reductions in neural drive to the active musculature. It is likely that a combination of negative mood states and moderate declines in muscle glycogen content may be responsible for the reduced pacing strategies and sprint performance. Further, the conscious awareness of negative mood states and the relatively minor differences in physiological variables (including muscle glycogen) suggests that the alteration in pacing strategies may have been predominately a conscious decision based on knowledge and experience of being sleep deprived rather than sub-conscious feedback from the periphery.
Chapter 7 – General Discussion

Chapter 5 - Carbohydrate Ingestion

In the second study (Chapter 5), the effects of high or low CHO diet and muscle glycogen between consecutive days of exercise on self-paced intermittent-sprint performance were examined. Uniquely, participants were blind to all knowledge of CHO diet and muscle glycogen manipulation to remove the expectation that one condition was superior to the other (high vs low CHO). Such a model allowed some insight into the conscious vs sub-conscious regulation of pacing. In relation to thesis aim (1), pre-exercise muscle biopsies on Day 2 showed that muscle glycogen concentration was significantly reduced following the LCHO diet compared to HCHO. Total and hard running distance covered were significantly greater following HCHO and pacing strategies indicate a greater distance was covered during alternating 10 min phases of the protocol. These findings are consistent with previous field-based studies reporting a significant increase in high-intensity running following a high CHO diet (Balsom et al. 1999b; Bangsbo et al. 2006). However, this was the first study to examine the effects of blinded high or low CHO diets before a laboratory-based, intermittent-sprint protocol specifically examining pacing strategies.

In relation to thesis aim (2), it is suggested that pacing strategies were continuously regulated throughout the protocol in response to afferent feedback regarding muscle glycogen content rather than an explicit conscious decision to manipulate exercise intensities. While it has been suggested that performance declines during the early aspects of an exercise protocol are due to the knowledge of CHO ingestion (Johnson et al. 2006), the present study indicates pacing strategies are down-regulated after the initial minute of exercise despite no knowledge of CHO manipulation. Similar to the present findings, Rauch et al. (2005) reported a decline in power output during a cycling trial after the first minute of exercise; suggesting that exercise was regulated in anticipation of declining muscle glycogen content. This notion of a ‘gluco-stat’ suggests that muscle glycogen utilisation and thus glycogen depletion is regulated throughout self-paced exercise via changes in exercise intensity to minimise substantial declines in post-exercise glycogen concentrations. A ‘gluco-stat’ regulation of
muscle glycogen may also be present in the current carbohydrate study, as noted by an early decline in intensity, a discontinuous pacing strategy throughout the protocol, and a lower utilisation rate during LCHO; suggesting the altered pacing was based on lowered muscle glycogen content. Accordingly, this study highlights the role of a feed-forward/feedback regulation of exercise intensity that does not seem to be predominately conscious control.

Chapter 6 - Thermoregulation

The third study (Chapter 6) examined the effect of exposure to different pre-exercise thermal conditions on pacing strategies and intermittent-sprint performance. This study was designed due to the paradox between the ergogenic effects of pre-cooling to prolonged intermittent-sprint exercise (Castle et al. 2006; Duffield and Marino, 2007) but the advantage of increased muscle temperature (via passive heating) on high-intensity efforts (Bishop, 2003; Lovell et al. 2011). Changes to thermal stress were achieved by the exposure of subjects to 15 min of pre-cooling, passive heating or thermo-neutral conditions before the ISE protocol. Results from this study highlight the potential benefits of these thermal interventions and the effect on pacing strategies adopted during each of the respective modes in the ISE protocol. Greater distances were covered during the sub-maximal efforts throughout the final 20 min of the protocol during ICE, with differences mainly attributed to an increase in hard running distance compared to HEAT. On the contrary, HEAT reduced sub-maximal exercise performance but improved sprint times during the initial 10 min of the protocol compared to ICE. Post-intervention TCP results suggest the improved sprint times following HEAT were due to increases in muscle temperature facilitating muscle contractile properties (Cheung, 2008). Collectively, these findings suggest that temperature-dependent effects on ISE performance are mode specific with ICE improving sub-maximal efforts while HEAT augments sprint performance.

As differences in distance covered were not evident until the final 20 min of exercise when physiological variables (T_{core}, T_{skin} and PSI) had dissipated, it is possible that pacing strategies were
regulated in anticipation of the imposed loads placed on the body (thesis aim 2). Furthermore, an increased maintenance of post-exercise MVC during ICE compared to pre-exercise values was associated with significantly higher VA values compared to HEAT. Similarly, previous work has highlighted that increased core temperature reduces MVC due to declines in neural drive (Morrison et al. 2004; Thomas et al. 2006). Collectively, these findings suggest that the mechanisms responsible for intermittent-sprint performance and pacing strategies are likely both central and peripheral in origin; as pre-cooling maintains an increased neural drive compared to passive heating, but cooling of the periphery also leads to temperature-dependent effects on the contractile unit and impairs sprint performance.

7.3 Descriptions of the Regulation of Pacing Strategies

Prior to discussion of the mechanisms regulating intermittent-sprint exercise performance, it is worth noting the observed similarities and differences between studies and conditions with respect to the pacing profiles adopted. Within this chapter, to allow comparison between studies (and populations), pacing is assessed through the presentation of individual sprint and sub-maximal efforts as a percentage of the fastest speed recorded throughout an individual’s testing (% peak speed). Mean speed was calculated based on the distance covered within each time frame and then divided by the peak speed from sprint data, and as presented in Figures 7.1 – 7.3, mean % peak speed for 3 consecutive minutes (50 s of each min) were calculated to correspond to the total distance covered in each cycle of hard running, jogging and walking.

Previous methods that have assessed pacing during cycling time trials, such as a sample of power output at a selected time point, may not be sensitive enough for the sporadic interplay of various exercise intensities evident in team sports (Abbiss et al. 2008; St Clair Gibson et al. 2001). Therefore, the above method of modal segmentation provided means of examining the profile of the pacing strategies employed throughout the entire ISE protocol and the contribution of each mode to overall performance. Whilst minute-by-minute reporting of exercise intensity was considered, due to the wide
range of exercise intensities performed i.e. walking to sprinting, deciphering the changes in pacing based on such variation was difficult. Hence, the mean of one cycle of sub-maximal and associated sprint efforts were calculated to compare pacing strategies. As outlined in Table 7.1, and discussed in further detail in the ensuing sections, there were distinct similarities and differences both within and between the respective studies. Of note, the manipulation of exercise intensity following the intentional alteration of central, peripheral and/or conscious factors may have contributed to specific pacing strategy adjustments determined by the physical, physiological and perceptual states. More specifically, within the sleep deprivation study, alterations in pacing were evident between conditions due to slower sprint times and less distance covered during the sub-maximal and hard running efforts at the start and end of the protocol when sleep deprived. During the carbohydrate study, differences in pacing were present due to less distance covered during the sub-maximal and hard running efforts throughout the protocol following a LCHO diet compared to HCHO. Finally, pre-cooling within the thermoregulatory study significantly reduced initial sprint performance due to the even pacing strategy present during ICE; however, greater hard running and sub-maximal distances were covered during the final 20 min of the protocol compared to HEAT.

Table 7.1 summarises that the pacing profile during intermittent-sprint exercise is regulated specifically by the pre-exercise perceptual and physiological states. When either peripheral perturbations are ‘extreme’, or perceived as ‘extreme’, exercise intensity is regulated down accordingly. However, as exercise continues the differences in intensity between conditions are often reduced when the actual physiological differences are minimal, suggesting exercise regulation may potentially be a central control of the peripheral state (Duffield et al. 2010; Tucker et al. 2004). The representation of sprint data as % peak speed indicates only slight variations in sprint performance between conditions, with each study indicating a general trend for sprints to be quickest during the first 3 min with progressive declines thereafter. However, sprint performance during ICE was an exception with changes in performance appearing to be a direct result of altered contractile properties associated with reduced muscle temperature (Cheung, 2008).
Table 7.1: Overview of pacing strategies adopted during the sub-maximal, hard running, and sprint efforts of the self-paced intermittent-sprint exercise protocol completed during the respective studies of the present thesis.

<table>
<thead>
<tr>
<th>Sub-Maximal Efforts (Figure 7.1)</th>
<th>Chapter 4 – Sleep Deprivation Study</th>
<th>Chapter 5 - Carbohydrate Study</th>
<th>Chapter 6 - Thermoregulation Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Even pacing strategy was present for both conditions.</td>
<td>- Relatively even pacing strategy was present during both conditions.</td>
<td>- Reverse ‘J-shaped’ strategy was present during ICE and CONT due to an increase in distance covered during the final stages of the protocol.</td>
<td></td>
</tr>
<tr>
<td>- Trends of a reverse ‘J shape’ parabolic curve for both conditions.</td>
<td>- Trends of a slight positive curve during the latter stages of the ISE protocol during both conditions.</td>
<td>- Positive pacing strategy was present during HEAT due to a progressive decline until the end of exercise.</td>
<td></td>
</tr>
<tr>
<td>- SDEP resulted in a slower pacing strategy during initial (4-13min) and final (39-48min) stages of the protocol compared to CONT.</td>
<td>- HCHO resulted in higher pacing strategies throughout the entire ISE protocol compared to LCHO.</td>
<td>- ICE resulted in higher pacing strategies compared to HEAT during the final ~20 min of exercise.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Hard Running Efforts (Figure 7.2)</th>
<th>Chapter 4 – Sleep Deprivation Study</th>
<th>Chapter 5 - Carbohydrate Study</th>
<th>Chapter 6 - Thermoregulation Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Parabolic ‘U-shaped’ pacing strategy was evident during both conditions due to similar initial hard running efforts, a down-regulation during the mid-section and an end-spurt.</td>
<td>- Parabolic ‘U-shaped’ pacing strategy was evident during both conditions due to similar initial hard running efforts, a down-regulation during the mid-section and an end-spurt.</td>
<td>- Parabolic ‘J-shaped’ pacing curve was evident during all conditions due to similar initial hard running efforts, a down-regulation during the mid-section and an end-spurt.</td>
<td></td>
</tr>
<tr>
<td>- SDEP resulted in lower pacing strategies during the initial and final efforts compared to CONT.</td>
<td>- HCHO resulted in higher pacing strategies throughout the entire ISE protocol compared to LCHO.</td>
<td>- ICE resulted in a greater increase in end-spurt distance compared to HEAT.</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sprint Efforts (Figure 7.3)</th>
<th>Chapter 4 – Sleep Deprivation Study</th>
<th>Chapter 5 - Carbohydrate Study</th>
<th>Chapter 6 - Thermoregulation Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Even pacing strategy was present for both conditions.</td>
<td>- Relatively even strategy during both conditions.</td>
<td>- Relatively even strategy was present during ICE (due to slower sprint times in the first ~13 min of the protocol)</td>
<td></td>
</tr>
<tr>
<td>- Trends of a more exaggerated ‘U-shaped curve’ during CONT due to quicker sprint times during the initial and final 10 min phases of the protocol compared to SDEP.</td>
<td>- Trends of a slight positive curve were also present in both conditions.</td>
<td>- Positive pacing strategy was present during CONT and HEAT (a greater maintenance of % peak speed was present during HEAT).</td>
<td></td>
</tr>
</tbody>
</table>
Figure 7.1: Mean ± SEM % peak speed during each 3 min block of sub-maximal efforts (hard running, jogging, walking) of the intermittent-sprint protocol for A) sleep deprivation (SDEP) and control (CONT); B) high CHO diet (HCHO) and low CHO diet (LCHO) conditions; C) pre-cooling (ICE), passive heating (HEAT) and control (CONT) conditions, respectively. * Significant difference between conditions ($P < 0.05$)
Figure 7.2: Mean ± SD % peak speed for each hard running effort during A) sleep deprivation (SDEP) and control (CONT); B) high CHO diet (HCHO) and low CHO diet (LCHO) conditions; C) precooling (ICE), passive heating (HEAT) and control (CONT) conditions, respectively.

* Significant difference between conditions ($P < 0.05$)
Figure 7.3: Mean ± SD % peak speed for each sprint during A) sleep deprivation (SDEP) and control (CONT); B) high CHO diet (HCHO) and low CHO diet (LCHO) conditions; C) pre-cooling (ICE), passive heating (HEAT) and control (CONT) conditions, respectively.
* Significant difference between conditions ($P < 0.05$)
The minimal differences in sprint performance within the respective studies, despite differences in hard running performance, highlight the interplay between the different modes within an ISE protocol. Accordingly, the data show a potential conscious regulation of pacing, with the awareness of sprint times being measured (with the use of timing gates) giving a greater perceived importance and conscious decision to maintain sprint times. Such a decision making process may be replicated in match conditions with particular match-based tactical demands dependent upon the ability to perform maximal sprint efforts. However, in order for sprint times to be maintained over the duration of a match, and as suggested in field-based evidence, other exercise modes may need to be sacrificed (Coutts et al. 2010; Duffield et al. 2009). Within the present thesis, hard running distance covered seemed to be reduced to assist in the maintenance of sprint times. In summary, these findings show that pacing strategies can be evident between the various exercise modes during self-paced ISE, and athletes may place a conscious effort to maintain performance during certain aspects of a match (i.e. sprints). Furthermore, the within and between study alterations in pacing strategies highlight that both the perception of an intervention as well as the physiological effects of the respective interventions alter sub-maximal efforts during intermittent-sprint exercise. The comparison of pacing during the sub-maximal and hard running efforts within each condition suggests that overall pacing is primarily dependent upon the regulation of hard running efforts in combination with sprint efforts. The ensuing section will discuss the mechanisms potentially associated with the aforementioned descriptions of pacing strategies adopted within and between studies.

7.4 Mechanisms and Models to Explain Pacing Strategies during ISE

The mechanisms proposed to explain the development of fatigue and pacing strategies during continuous and intermittent-sprint exercise are somewhat elusive, with considerable debate regarding the appropriateness of respective models to explain performance outcomes (Abbiss and Laursen, 2005; McKenna and Hargreaves 2008; Noakes et al. 2000; Tucker, 2009). Specifically, the examination of pacing strategies during self-paced ISE is limited, with the understanding of the
underlying mechanisms complicated by both the vast range of exercise modes utilised and the associated physiological demands. The findings from the present thesis highlight that during self-paced ISE there is a dynamic interplay of pacing strategies within and between the respective exercise modes of the protocol (sprints and sub-maximal efforts). Such interplay of exercise modes may also reflect a concomitant interaction of mechanisms responsible for the regulation of such pacing strategies and ISE performance. The ability to elucidate the contribution of each of the proposed mechanisms was achieved by altering the athlete’s physiological state (central vs peripheral, conscious vs sub-conscious) and examining the subsequent pacing strategies adopted during the sub-maximal efforts.

Comparisons between studies suggest that different physiological and perceptual states associated with the pre-exercise circumstances resulted in specific adjustment to both the overall pacing strategy and individual strategies adopted within the respective modes (as highlighted in the previous section). In relation to changes of such pre-exercise physiological and perceptual states, it is acknowledged that prior experience can also influence subsequent performance and the pacing template adopted during exercise. Accordingly, the influence of prior experience was accounted for during all studies by recruiting a new cohort of subjects for each study (with all familiarisation sessions standardised within and between studies), thus removing the possibility of prior experience of the specific ISE protocol. Further, the within-subject and randomised design of the studies removed the influence of prior experience on subsequent, self-paced ISE performance. Therefore, with the standardisation of prior experience, it is assumed the adoption of pacing during ISE protocol was a result of the changes in pre-exercise physiological and perceptual states. Accordingly, as shown in Figure 7.4, the respective pre-exercise circumstances resulted in changes in the (conscious) perception of mood states following sleep deprivation (Chapter 4); blinded changes in muscle glycogen content (peripheral) during the carbohydrate study (Chapter 5); and changes in neural drive (central) and contractile
properties (peripheral) during thermoregulatory study (Chapter 6); which assumingly contributed to the alteration of the overall pacing strategy and the pacing of specific modes within the ISE protocol. Models that have been proposed over recent decades to explain the onset and development of fatigue are generally classed as either central or peripheral (Ament and Verkerke, 2009; Davis and Bailey, 1997; Vollestad et al. 1997). Unfortunately, many previous studies examining fatigue have deduced a ‘either/or’ approach which may lead to potentially neglecting the complex interaction of mechanisms related to the development of fatigue. In the present thesis, the investigation of previously proposed models of fatigue via the application of various pre-exercise circumstances has uniquely shown that rather than one exclusive mechanism being responsible, the regulation of pacing is likely to be an integration of all previously proposed models (McKenna and Hargreaves, 2008). However, the level of contribution that each of these mechanisms may have on performance and pacing are task dependent (overall exercise protocol and specific modes), and dependent upon both any physical change as well as the knowledge of the change in pre-exercise state.

As highlighted in Figure 7.4, centrally mediated factors contributed to overall and modal pacing strategies during all studies of the present thesis. Within these central factors, it appears that dependent upon the knowledge provided to the athlete regarding their pre-exercise circumstance, both sub-conscious and conscious regulation of pacing are present. While observations listed below (Figure 7.4) highlight that the regulation of pacing strategies were of central control, changes in pre-exercise physiological states can also have a direct effect on the periphery (substrate availability and muscle temperature) and ensuing ISE performance. These peripheral changes appeared to have the greatest effect on the pacing of specific modes, including sprints and hard running. Accordingly, the remainder of this section will discuss in further detail the observations outlined in Figure 7.4 based on previous models and mechanisms explaining fatigue and exercise regulation.
Figure 7.4: A diagrammatic summary of the mechanisms that contributed to the regulation of pacing strategies during the present self-paced intermittent-sprint exercise (ISE) protocol and the use of pre-exercise circumstances to highlight the contributions each of the respective (central, peripheral, and conscious) factors had on ISE pacing and the strategies during specific modes of the ISE protocol.
Peripheral

Throughout the present thesis evidence is presented to support the notion that changes to the periphery (skeletal musculature) can be directly responsible for changes in pacing strategies and ISE performance. Accordingly, the pre-exercise sleep deprivation, CHO diets and thermoregulatory interventions altered skeletal muscle properties, with changes to substrate availability and the contractile unit, respectively. As presented in Chapter 6, pre-cooling directly compromised the contractile unit, including temperature-dependent changes to twitch contractile properties such as lower peak torque, slower time to peak torque and relaxation rates. These changes in peak torque and relaxation rates may account for the reduction in sprint performance during the first 10 min of the ISE protocol. Cheung (2008) has previously discussed that changes in twitch contractile properties are representative of changes to the excitation-contraction coupling and contractile properties of the muscle. For example, the peak twitch torque and rate of relaxation may be related to calcium release and uptake within the sarcoplasmic reticulum (Cheung, 2008). Although not directly measured, differences in sprint times dissipated once muscle temperature was likely to have increased with sufficient exercise duration (Young, 1990). These findings suggest that reduced muscle temperature resulted in an even pacing strategy following ICE compared to a positive pacing strategy during CONT and HEAT, where muscle temperature was likely to be greater. Accordingly, findings from Chapter 6 are an example where altered peripheral functioning is likely to have overtly affected ensuing pacing and (sprint) performance.

Similar to changes in internal and external temperatures during the thermoregulatory study, alterations in substrate availability within the muscle also appear to have had a direct effect on ISE performance. Within the carbohydrate study there was a significant reduction in muscle glycogen and glycogen utilisation during the LCHO condition. It is likely that the significantly increased total distance and distance covered during the hard running efforts were due to increased muscle glycogen availability providing a means for increased glycogen utilisation via glycogenolysis (Hargreaves, 2004).
Specifically, the increase in hard running performance may have been related to the increased reliance on CHO oxidation during high intensity efforts compared to lower intensities (Romijin et al. 1993; Van Loon et al. 2001). In support of the findings that high-intensity effects are predominately affected by changes in muscle glycogen content, Havemann et al. (2006) reported that pre-exercise diets with either high volumes of CHO or fat had no effect on 100 km cycling time trial performance. However, the maximal sprints interspersed during the trial were significantly higher during the HCHO condition, suggesting that only high-intensity efforts are compromised by low muscle glycogen content during self-paced exercise. As such, in the present thesis the selective pacing of specific modes (hard running) may be reflective of the fuel substrate availability for the given exercise intensity, suggesting the role of peripheral functioning to regulate performance of specific exercise modes; although minimal differences in sprint times despite differences in hard running also highlights some central regulation of exercise intensities.

In summary, evidence presented throughout the respective studies supports theories that exercise performance can be influenced by peripheral factors including, skeletal muscle temperature and substrate availability. The changes in performance and pacing appeared to be present specifically during the sprints and hard running efforts in which reduced muscle temperature and muscle glycogen generally have the greatest influence. These findings highlight that the mechanisms responsible for fatigue may be task dependent, with modal pacing strategies (high-intensity efforts) affected by peripheral mechanisms. While the effect of peripheral factors on performance in team sport athletes have been recognised in the present thesis, there are also observations that challenge the notion that self-paced ISE is governed exclusively by peripheral perturbations. For example, peripheral explanations regarding increased glycogen availability and utilisation increasing hard running distance covered does not account for the observation that sprint times were not affected by muscle glycogen manipulation. While acknowledging the effect of these peripheral perturbations following each of the pre-exercise circumstances, it appears that a centrally mediated regulation of these
physiological responses via changes in overall and modal pacing strategies were also present throughout ISE.

Central

In relation to the central regulation of pacing strategies, it has been proposed that the CNS matches the feedback from the periphery with the aim of the exercise outcome, creating a feed-forward/feedback loop in which exercise is regulated via changes in muscle recruitment (Lambert et al. 2005). Such evidence of centrally mediated regulation of exercise intensities appears to be applicable to the current ISE protocol, particularly based on several key observations throughout the respective studies. The key findings that support this view are (1) within all conditions an end-spurt was present during the final hard running efforts, and was preceded by a down-regulation of sub-maximal and hard running distances during the mid-section of the protocol; (2) changes in pacing strategies were adopted during the early stages of the protocol rather than concluding stages when internal strain would presumably be at its highest; (3) end-exercise physiological and RPE responses were similar between conditions despite different exercise intensities throughout the protocol; (4) no changes in post-exercise twitch contractile properties were evident between conditions, although differences in MVC and VA were observed. Accordingly, the central regulation of exercise intensities and muscle recruitment during the ISE protocol appears to be a combination of an anticipatory regulation of the forthcoming loads in conjunction with a protective regulation of peripheral perturbations; which will be discussed subsequently.

As outlined in the preceding section of the discussion (section 7.3), distinctive pacing strategies dependent on the pre-exercise state were present both within and between studies. As different physiological, perceptual and conscious states were present following the respective pre-exercise circumstances, a reasonable assumption is that the strategies adopted during the ISE protocol were in response to these states. The first observation to support centrally-mediated pacing relates to the
changes in pacing strategies despite minimal physiological differences during the sleep deprivation study. The differences in pre-exercise muscle glycogen were modest between conditions, and not likely to be sufficient to solely affect exercise performance (Burke et al. 2000; Hawley et al. 1997a). It is more likely the knowledge or experience of being sleep deprived and the associated negative mood states were responsible for the change in subsequent pacing strategies (Oliver et al. 2009). Accordingly, the increased perceptual stress was associated with sleep deprivation induced modification of pacing strategies; which cannot be solely explained by peripheral models of fatigue. Furthermore, the reduction in MVF and VA pre and post exercise during SDEP2 compared to CONT2 indicate the reduction in force production, slower sprint times and less distance covered during the sub-maximal efforts were primarily centrally derived rather than changes within the contractile unit.

The second observation which supports a centrally mediated regulation of exercise intensities includes the dissipation (or significant reduction) of physiological differences between conditions as exercise protocols progressed. For instance, due to reduced substrate availability, muscle glycogen utilisation was lower in the LCHO condition, yet post-exercise glycogen content was not completely depleted, possibly suggesting an anticipatory maintenance of glycogen availability (Rauch et al. 2005). Additionally, during the thermoregulatory study, differences in post-intervention $T_{\text{core}}$, $T_{\text{skin}}$ and PSI between ICE and HEAT had disappeared by 30 min of exercise, remaining similar for the final 20 min of exercise. These diminishing differences in thermoregulatory variables between conditions coincided with a reduction in pacing during HEAT compared to an increase in pacing during the ICE condition. These findings suggest that pacing strategies were continuously regulated (lower and higher, respectively) in order to maintain similar levels of thermal strain ($T_{\text{core}}$, $T_{\text{skin}}$ and PSI) during exercise in the heat. Similarly, Duffield et al. (2010) reported performance differences in the latter part of a cycling time trial between pre-cooling and control conditions, despite thermoregulatory variables disappearing prior to performance differences. Furthermore, within all studies of the present thesis there were no differences in cardiovascular and biochemistry blood markers ($\text{pH}$, glucose and
lactate) at the completion of exercise, despite different distances covered between conditions. The similar physiological responses during exercise despite the difference in pre-exercise states may support the notion that pacing strategies during ISE are down regulated when an athlete is not in an ‘optimal’ pre-exercise state and this down-regulation is to ensure exercise is completed but within homeostatic boundaries (Noakes, 2011).

Another observation during the respective studies that confirms pacing strategies were predominately centrally rather than peripherally regulated relates to the lack of difference in post-exercise neuromuscular assessments. It is evident that there were peripheral perturbations that contributed to performance outcomes during specific elements of the ISE protocol (sprints and hard running). However, the lack of difference between conditions in post-exercise twitch contractile properties suggests that the observed reductions in performance and pacing throughout the ISE protocol and post-exercise MVC values were not due to compromised contractile unit within the active musculature (knee extensors) (Andersen and Aagaard, 2006). While it is accepted that the use of isometric contractions of an isolated muscle group may not be directly representative of the dynamic movement patterns present during the ISE protocol; the use of neuromuscular assessment immediately after exercise may provide some indication of the state of the contractile unit during the final stages of exercise. Similar to the findings in Chapter 6, Saboisky et al. (2003) reported that exercise-induced hyperthermia (38.8 ± 0.2°C) during incremental exercise in the heat resulted in a reduction in MVC and central activation ratio; however, post-exercise TCP including Pt, TPt and HRT were not affected by hyperthermia. The authors concluded that the CNS reduces neural drive to skeletal muscles as a result of hyperthermia, which also appears to be evident in the results of the present thesis. Accordingly, it has been previously discussed that changes in Pt, RTD, CD and RR are associated with cross-bridge cycling rate and the sarcoplasmic reticulum’s release and uptake of calcium (Andersen and Aagaard, 2006; Cheung, 2008); although no differences in any of the aforementioned contractile properties were evident between conditions in any study. Therefore, if the changes in pre-
exercise contractile unit properties were exclusively responsible for the reductions in performance during the ISE protocol observed during the more deleterious conditions, this would have been evident during the post-exercise neuromuscular assessments.

In light of the minimal differences in post-exercise twitch contractile properties, an alternative explanation for the down-regulation of exercise intensities during exercise is that central factors contributed to ISE pacing. Although not measured during the ISE protocol due to methodological limitations, cycling time trial protocols that have measured muscle recruitment with EMG have shown that reductions in power output during self-paced exercise are due to declines in neural drive to the active musculature (Kay et al. 2001; Tucker et al. 2004). Furthermore, comparable to the present ISE protocol, assessment of pacing strategy profiles during time trials have shown that an end-spurt is the result of increased neural drive (Ansley et al. 2004; Noakes, 2011; Tucker et al. 2004). The post-exercise neuromuscular assessment findings of the present thesis support the interpretations from cycling trials in that pacing strategies are a result of changes in neural drive. Within the respective studies VA was reduced pre and post exercise during the SDEP (Chapter 4) and post-intervention and post-exercise during the HEAT (Chapter 6) condition. In conjunction with previous literature, post-exercise neuromuscular recruitment suggests that the pacing strategies during the ISE protocol (including down-regulation during the mid-section and the end-spurt for all studies) may be due to alterations in neural drive to the active musculature without differences in the contractile unit after exercise (Andersen and Aagaard, 2006). Accordingly, during the present thesis, the lack of differences in post-exercise twitch contractile properties despite a down-regulation of pacing during the mid-section of the protocol and the end-spurt suggests that changes in pacing strategies throughout the ISE protocol were likely to be centrally derived.

Similar to the centrally mediated down-regulation of pacing and muscle recruitment to minimise differences in the physiological responses, perceptual ratings throughout the three studies also show
evidence of central regulation. The similar RPE responses between conditions (within the respective studies) suggest that pacing strategies may have been altered as a protective mechanism of RPE and/or physiological perturbations (Hampson et al. 2001; St Clair Gibson, 2003). More specifically, it is proposed that RPE is a conscious sensation of fatigue and is correlated with sub-conscious control of physiological perturbations including heart rate, metabolite accumulation and respiratory rate (Hampson et al. 2001; St Clair Gibson et al. 2003). The protection of RPE is further supported by the progressive increase in perceived exertion with exercise duration, despite RPE values remaining sub-maximal (< 20) during all time points of the protocol and across all studies. In the present thesis, RPE and physiological responses were not different between conditions and remained ‘sub-maximal’ throughout the ISE protocol; suggestive of an anticipatory mechanism operating throughout the exercise bout to maintain similar and sub-maximal perceptual stress (Hampson et al. 2001).

The down-regulation of exercise intensities during the sub-maximal and hard running efforts of the ISE protocol and final end-spurt highlights that centrally mediated mechanisms were predominately responsible for the regulation of pacing strategies. As shown in Figure 7.2, the increase in distance during the final hard running effort compared to the penultimate minute was evident, irrespective of the condition or study. Further, a greater end-spurt distance was covered during the conditions that were considered to be favourable to the sub-maximal efforts (CONT, HCHO, and ICE) compared to the less favourable conditions of the respective studies (SDEP, LCHO and HEAT). These findings are supported by studies using self-paced protocols that have reported greater end-spurt in neutral conditions compared to the heat (Tucker et al. 2004) and with increased fluid availability (Dugas et al. 2009). Self-paced intermittent-sprint studies have also reported increases in final hard running efforts following pre-cooling (Duffield and Marino, 2007) and fluid ingestion during exercise (Skein and Duffield, 2010) compared to a control. The findings of the present thesis suggest that more advantageous pre-exercise physiological (Chapter 5 and 6) and perceptual (Chapter 4) states allow for
Chapter 7 – General Discussion

the maintenance of higher intensity pacing strategies throughout the protocol and during the final hard running effort.

The increase in hard running distance during the end-spurt in conjunction with the down-regulation of distance covered during the preceding mid-section of the protocol confirms that such regulation was pre-emptive in nature. The regulation of pacing strategies via a down-regulation of sub-maximal exercise intensity during the protocol mid-section was accentuated by changes in pre-exercise states including peripheral (i.e. muscle glycogen depletion or increased thermal stress) and perceptual states (mood states). While these peripheral changes were present, it appears the CNS was able to consider the different conscious and sub-conscious responses associated with the respective pre-exercise states and regulate intensities to allow for the successful completion of the protocol (Lambert et al. 2005; Noakes, 2011). In relation to the present thesis, a down-regulation of pacing and an end-spurt were evident in all conditions, irrespective of the pre-exercise circumstance and the knowledge of such circumstances. Accordingly, if peripheral mechanisms were exclusively responsible for changes in performance and pacing, exercise intensities should have continued to decline with the reduction in substrate availability, increased perception of effort, or increase in thermal stress. However, when physiological and perceptual stresses were at their highest, distance covered was able to be increased to similar distances covered at the start of exercise. Therefore, the differences in pre-exercise physiological and perceptual states were attenuated during subsequent exercise due to different pacing strategies set during the sprinting and sub-maximal efforts; thus suggesting an anticipatory protection (of conscious or sub-conscious control) of physiological and perceptual states during exercise.

Conscious vs Sub-conscious Regulation

Within centrally derived explanations of fatigue, there are questions regarding the contribution of the conscious awareness compared to the sub-conscious regulation of pacing strategies (Marcora, 2010; St Clair Gibson, 2004). Marcora and colleagues (2008, 2009) have most recently examined this
concept of conscious exercise regulation, suggesting that exercise is primarily influenced by motivation and perception of effort; for example, during exhaustive exercise subjects ‘give up’ when they exceed their greatest perceived effort (Marcora and Staiano, 2010). In the context of self-paced exercise, it would be assumed that a down-regulation of exercise intensity is of conscious control as opposed to the sub-conscious feed-forward decline in neural drive and intensity in response to afferent feedback. This discrepancy between contributions of sub-conscious and conscious regulation of pacing strategies was assessed within the present thesis through the manipulation of knowledge regarding altered pre-exercise circumstances. Collectively, the findings indicate that pacing strategies are likely to be both conscious and sub-consciously regulated during self-paced exercise. However, the level of contribution is dependent on the knowledge (and expectation) of the pre-exercise circumstance and the magnitude of change in physiological and perceptual variables.

The delivery of interventions during Chapters 5 and 6 indicate that the regulation of pacing strategies during the ISE protocol in response to the pre-exercise circumstances were predominately sub-conscious. By removing the knowledge about the CHO diets, it is assumed that changes in pacing were a direct result of sub-conscious manipulation of exercise intensities. Further, during the thermoregulatory study, subjects were aware of the conditions; however, were unlikely to be fully aware of the potential advantageous or deleterious effects the respective cooling and heating interventions may have of the pacing of respective modes (sprints and hard running). While it is noted some conscious regulation may be present, as subjects were either completely or partially blinded to their conditions, it is likely that the regulation of pacing during the carbohydrate and thermoregulatory studies was primarily sub-conscious. Further, the sub-conscious regulation might be in response to feedback from the periphery regarding the current physical state and in anticipation of the imposed loads for the remaining exercise duration (Noakes, 2011). For example, the blinding of all knowledge that CHO and muscle glycogen were manipulated (Chapter 5) indicates that changes in performance and pacing were sub-consciously regulated. Further, the decreased utilisation during the LCHO
Chapter 7 – General Discussion

Condition and the discontinuous pacing strategies between conditions suggests that pacing was continuously altered throughout the entire protocol to regulate and preserve muscle glycogen content. Collectively, the sub-conscious protection of internal strain during exercise is supported by evidence of a reserve, unknown protection of muscle glycogen content, and similar physiological and RPE responses in all conditions.

Alternatively, findings from the present thesis also indicate that a conscious regulation of exercise was evident during the ISE protocol. As outlined in Chapter 4, the moderate difference between conditions in muscle glycogen and substantial differences in mood states following sleep deprivation suggest pacing strategies were primarily of conscious regulation. This finding supports previous literature on the effect of sleep deprivation on self-paced exercise performance (Oliver et al. 2009), which is further supported by studies that have indicated a conscious regulation of exercise intensities following the use of placebos and deception trials. For example, Paterson and Marino (2004) showed that pacing strategies were altered when subjects were deceived about the time to completion of a time trial, whilst Billaut et al. (2011) have reported changes in pacing of repeated sprints when deceived about the number of sprints to be completed. Further, Clark et al. (2000) examined the placebo effect of CHO ingestion on 40 km time trial performance, during which subjects were either told they were ingesting CHO during exercise, given a placebo, or provided no information. Power output was improved during informed CHO and placebo groups while no information resulted in reduced power output. Additionally, Skein and Duffield (2010) reported (not statistically different) trends indicative of increased maintenance in pacing strategies when subjects were informed of the availability to consume water during the second half of a 50 min intermittent-sprint performance exercise protocol in the heat, despite no differences in physiological variables. These aforementioned studies concur with the current data highlighting that an athlete’s knowledge regarding their physiological state and/or the potential effects an intervention may have on performance can significantly affect exercise performance and the conscious manipulation of pacing strategies.
Alternatively, if the subject is unaware of the intervention and the physiological perturbations are significant, some role of sub-conscious regulation of exercise intensity seems evident.

7.5 Summary of Pacing Strategies during Intermittent-Sprint Exercise

The examination of pacing strategies during self-paced ISE indicates that intensities are regulated throughout a given exercise protocol. The regulation of such pacing strategies appears to be altered between and within the respective sleep deprivation, CHO consumption and thermoregulatory studies. These differences are due to the influence of the respective pre-exercise circumstances on central, peripheral and perceptual variables during the subsequent exercise bout. When comparing to previously proposed models and mechanisms to explain fatigue; data from each of the studies indicate the presence of both peripheral and central influences on pacing strategies during self-paced ISE. It is evident peripheral limitations to performance influence specific exercise modes within the protocol; however, it appears overall pacing strategies and the integration of all modal strategies were primarily central. These findings are supported by i) altered pacing strategies during early stages of the exercise protocol (after the first minute); ii) a down-regulation of pacing during the mid-section of the protocol followed by an end-spurt during hard running; iii) no differences between conditions in post-exercise TCP’s; and iv) similar physiological and RPE responses despite different pacing strategies. Accordingly, team sport athletes regulate their pacing strategies during self-paced ISE, with strategies dependent upon the magnitude of the physiological and perceptual stress, the influence on specific exercise modes (particularly sprinting and hard running), and the knowledge available to the athlete regarding their pre-exercise physical state. Overall pacing strategies may be centrally mediated but specific peripheral factors and/or knowledge of the environment will influence performance and pacing of specific modes. Therefore, it remains advisable that athletes ensure they are in optimal pre-exercise physiological and perceptual states to maintain higher intensity pacing strategies in all components of a match.
Chapter 8

Summary and Conclusions
8.1 Overview

The purpose of the thesis was to examine the effects of various pre-exercise physiological and perceptual states on pacing strategies during self-paced, intermittent-sprint exercise in team sport athletes. Secondly, this thesis examined the mechanisms responsible for the alterations in pacing strategies in response to altered physiological and perceptual stress in team sport athletes. Accordingly, the outcomes of the research aims are addressed as follows:

8.2 Research Aims – Chapter 3

1) The aim of this study was to assess the reliability of a self-paced intermittent-sprint exercise protocol that is designed to allow self-paced intermittent-sprint activity.

The self-paced intermittent-sprint exercise protocol utilised during all experimental chapters of the thesis was shown to have high repeatability between sessions. More specifically, the test-retest reliability of total distance covered and distance covered during individual hard running, jogging, and walking about was particularly high ($r = 0.82-0.96$, CV=1.5-3.2%). The test-retest reliability of heart rate and RPE also demonstrated high reliability between sessions ($r = 0.92 - 0.94$, CV = 1.2 - 6.6 %), suggesting the physiological load and perceived exertion between sessions were similar. Such findings suggest the reliability of the physiological, perceptual and performance responses to a self-paced intermittent-sprint protocol were acceptable.

8.3 Research Aims – Chapter 4

1) The aim of this study was to examine the effect of ~30h sleep deprivation on self-paced intermittent-sprint performance and pacing strategies.

Acute sleep deprivation of ~30 h significantly reduced maximal sprint performance and altered pacing strategies during the 50 min self-paced, intermittent-sprint exercise protocol. There was no significant difference between conditions for total distance covered or distance covered during the respective
sub-maximal efforts. However, differences in pacing strategies were observed during the first and final 10 min phases of the exercise protocol, with reduced intensities in the SDEP condition.

2) A secondary aim of the study was to examine sleep deprivation on mood states and neuromuscular function, and the implications these factors may have on subsequent team sport exercise.

The study highlighted that ~30 h sleep deprivation reduced pre-exercise muscle glycogen concentration on Day 2 compared to a normal (~8h) night sleep. Further, mood states were negatively affected and maximal voluntary contraction and activation were suppressed pre and post exercise on SDEP2. These differences in glycogen content and mood states appear to be responsible for the reductions in sprint performance and alterations in pacing strategies. Furthermore, the known disruptions to mood states suggests that changes in pacing were due to a conscious regulation rather than a sub-conscious regulation in response to feedback from the periphery. These findings suggest that negative mood states and to a lesser extent, muscle glycogen content associated with sleep deprivation are deleterious to maximal force production, intermittent-sprint performance and pacing strategies.

8.4 Research Aims – Chapter 5

1) The aim of this study was to examine the effect of a blinded pre-exercise CHO diet resulting in reduced muscle glycogen concentration on intermittent-sprint performance and pacing strategies in team sport athletes on consecutive days of exercise.

Results from Chapter 6 indicate that pre-exercise diets of either high (HCHO; 7g/kg bw) or low (LCHO; 2g/kg bw) CHO intake after a glycogen depleting exercise altered pre-exercise glycogen concentrations on Day 2. The reductions in CHO and glycogen content reduced total distance covered and distance covered during the hard running efforts compared to the HCHO, without differences in sprint times. Interestingly, pacing strategies during the sub-maximal efforts were only different during alternate 10 min phases of the protocol. Due to the blinded nature of the study, in which subjects were
not aware of CHO and muscle glycogen manipulation, it is suggested that the pacing strategies adopted were due to sub-conscious feedback from the periphery regarding muscle glycogen content and utilisation. Additionally, the reduction in hard running pacing in the early stages of the protocol and the reduced glycogen utilisation during LCHO suggests pacing was down-regulated to avoid critically low muscle glycogen concentrations.

8.5 Research Aims – Chapter 6

1) This study aimed to examine the effect of pre-cooling and passive heating on pacing on intermittent-sprint performance in warm environmental conditions.

Chapter 7 highlighted the paradox between pre-exercise cooling and passive heating, which are both interventions commonly encountered by team sport athletes. As observed via reductions in $T_{core}$ and $T_{skin}$ and PSI, pre-cooling reduced thermal strain, allowing higher exercise intensities during sub-maximal efforts in the final 20 min of the protocol. However, pre-cooling negated sprint times during the initial 10 min of the protocol, likely due to temperature-dependent changes in excitation-contraction coupling of the active musculature. Alternatively, passive heating increased thermal stress, leading to reduced pacing strategies in the latter stages of the protocol but with augmented sprint times compared to cooling. Interestingly, compared to the control condition, cooling and heating conditions had minimal effect on intermittent-sprint performance or pacing strategies.

2) A further aim was to determine the respective neuromuscular responses to the altered pre-exercise thermal state as a potential mechanism for changes in performance.

Pre-cooling negatively affected post-intervention evoked twitch contractile properties including peak torque, time to peak torque, and rate of relaxation. These declines in twitch properties indicate that temperature-dependent changes to contractile unit within the musculature were compromised (Cheung 2008). Although not measured during exercise due to methodological constraints, it is likely the reduced sprint performance following ICE was due to reduced muscle temperature and a
compromised contractile unit. Conversely, a greater maintenance of post-exercise voluntary force via an increased maintenance in neural drive during ICE condition was evident compared to HEAT and CONT. Accordingly, the increased muscle recruitment may explain the significant differences in distance covered during the final 20 min of exercise protocol; however, further investigation examining muscle recruitment during intermittent-sprint performance may need to be completed.

8.6 Thesis Aims

1) Examine the effect of pre-exercise interventions, commonly experienced by team sport athletes, on pacing strategies and intermittent-sprint performance.

The respective pre-exercise circumstances resulted in changes in the conscious perception of mood states and peripheral physiology (muscle glycogen) following sleep deprivation (Chapter 4); blinded changes in muscle glycogen content during the carbohydrate study (Chapter 5); and changes in neural drive (central) and contractile properties (peripheral) during thermoregulatory study (Chapter 6). These pre-exercise circumstances and the associated effects on central, peripheral and perceptual variables significantly altered intermittent-sprint performance and pacing strategies during respective studies.

- Within the sleep deprivation study, sprint times were significantly slower and less distance was covered during the initial and final 10 min phases of the exercise protocol, primarily due to reduced distances covered during the hard running efforts in the SDEP condition.
- During the carbohydrate study, significant differences in total and mean distances covered during hard running were evident between conditions, and reductions in distance during alternate 10 min phases of the protocol following the LCHO compared to HCHO diet.
- Pre-cooling within the thermoregulatory study resulted in a significant reduction in sprint performance, alongside an augmentation of endurance performance, with greater distance covered during final 20 min of the protocol compared to HEAT. However when compared to
the CONT condition, the cooling and heating interventions had no effect on intermittent-sprint performance and pacing strategies.

Collectively these results from Chapters 4 – 6, within the high protocol reliability outlined in Chapter 3, highlight pre-exercise circumstances that alter central, peripheral and perceptual factors affect pacing strategies and intermittent-sprint performance in team sport athletes. These changes in pacing strategies of the entire ISE protocol appear to be the result of intermodal pacing of the respective efforts; most notably the sprinting and hard running efforts, with a consistent strategy between conditions present in the lower intensity efforts.

2) Examine the mechanisms responsible for pacing and how these respective interventions influence the relative contributions of physiological and perceptual stress and prior knowledge have on the regulation of pacing strategies.

Findings from the present thesis illustrate that team sport athletes regulate pacing strategies during self-paced intermittent-sprint exercise. Closer inspection of pacing strategies into 10 min and 3 min phases and of the individual modes (sprinting, hard running, jogging, walking) revealed that the respective modes were influenced by both central and peripheral factors. Whilst changes associated with pre-exercise circumstances in the present thesis were predominately at the periphery and had a direct effect of performance of specific modes; there appears to be central manipulation of overall performance and pacing to regulate peripheral perturbations. This theory is supported by observations in each of the studies that participants covered the same distances during the initial hard running effort, despite differences in thermal strain and substrate availability. Following the similar initial effort, during all studies and conditions, a down-regulation of sub-maximal exercise intensities was observed during the mid-section of the ISE protocol, followed by an end-spurt during final hard running effort comparable to the initial efforts. These observations indicate that exercise intensities during the ISE protocol were anticipatorily regulated to ensure completion of the exercise bout at
intensities that superseded the penultimate effort. Additionally, the similar physiological responses for different strategies suggest that pacing was also regulated to result in similar physical and perceptual strain between conditions during the exercise bout.

8.7 Summary and Conclusions

It is clear that pacing strategies exist during prolonged intermittent-sprint exercise, and are regulated in response to various external stimuli such as environmental heat (Tucker et al. 2006), substrate availability (Rauch et al. 2005), and mental fatigue (Marcora and Staiano, 2010). However, little attention has been paid to the presence of pacing strategies during intermittent-sprint exercise that are reflective of team sport exercise. Further, the mechanism(s) responsible for changes in pacing strategies during self-paced intermittent-sprint exercise have remained relatively undefined and may be task dependent. Based on the results presented in Chapters 4 - 6, it may be concluded that pacing strategies are evident during prolonged, self-paced intermittent-sprint exercise and these strategies are set and constantly readjusted throughout the given exercise bout, not unlike continuous self-paced exercise protocols. The mechanisms responsible for the regulation of exercise intensity during intermittent-sprint exercise collectively include peripheral, central and perceptual responses. From these pre-exercise circumstances, it can be suggested that team sport athletes alter pacing strategies dependent upon the physiological and perceptual stress placed on the body and this conscious regulation is in part dependent upon the level of prior knowledge of the pre-exercise state. Further, the pacing strategies implemented appear to be centrally regulated in that exercise intensities are manipulated in an attempt to minimise the imposed loads of the given exercise task (such as thermal stress or substrate depletion), and in anticipation of the remaining exercise in order to ensure optimal completion within homeostatic boundaries.
8.8 Practical Applications

- In order for team sport athletes to avoid a significant down-regulation in pacing strategies during competition, coaches, sports scientists and athletes should provide a pre-exercise environment that promotes an optimal physical, physiological and perceptual state. This environment should include adequate sleep, CHO ingestion and reduced thermal stress.

- An athlete’s knowledge regarding their pre-exercise environment and physical state may influence subsequent pacing strategies; therefore, when possible athletes should be blinded to potentially detrimental circumstances while made aware of perceived benefits of advantageous circumstances/interventions.

- In relation to the findings from Chapter 4, team sport athletes should engage in environments that are conducive to sleep before and after competition matches to avoid potentially suffering from acute sleep deprivation. If sleep deprivation cannot be avoided, athletes should supplement their diet with additional CHO to counter the minor but potentially negative effects sleep loss has on muscle glycogen content. Further, interventions that improve perceived mood states may also be of benefit to athletes when in a sleep deprived state.

- In relation to findings from Chapter 5, high CHO diets (i.e. 7g kg\(^{-1}\) bw) should be provided to team sport athletes prior to training and/or competition irrespective of whether the athlete is aware of the increase in CHO ingestion and glycogen content.

- In relation to findings from Chapter 6, pre-cooling is recommended to team sport athletes when competing in the heat; however, warm-up duration and intensity should be sufficient to increase muscle temperature, whilst pre-cooling prevents an excessive increase in core temperature. This technique could be achieved with the application of upper-body cooling, such as an ice vest during the warm-up and half-time periods of a match.
8.9 Recommendations for Future Research

- Findings from Chapter 6 indicate changes in performance and pacing are evident despite no knowledge of an altered metabolic state; while Chapter 5 highlights that with minimal physiological differences but known altered mood states, pacing can also be affected. Therefore, further research examining the effects of prior knowledge using placebos and deception protocols may elucidate the conscious vs sub-conscious regulation of pacing strategies during self-paced exercise.

- Neuromuscular assessments were completed pre and post exercise with the use of maximal isometric contractions superimposed with electrical stimulus to determine MVC, VA and TCP. However, this assessment of neuromuscular function only allows inferences to the muscle recruitment during the intermittent-sprint exercise. Therefore, future research that examines EMG recruitment during ISE may provide insight regarding the relationship between muscle recruitment and pacing strategies during team sports. Furthermore, more recent techniques to assess neuromuscular function may also further elucidate the origin of changes in neural drive i.e. transcranial magnetic stimulation (TMS).

- While it is evident that pacing strategies responses are different depending on whether an athlete has experienced sleep deprivation, CHO ingestion or thermal stress, other interventions may also provide further insight into the mechanisms responsible for alterations in pacing strategies, such as exercise in hypoxic conditions with assessments of cerebral and tissue oxygenation in conjunction with neuromuscular assessments.
Chapter 9

References
References


261


Appendix A

Consent Forms

Information Sheets
CONSENT FORM

Experiment Title: The effects of sleep deprivation on the recovery of muscle and performance

I have read the Participant Information Sheet for the above experiment and had the procedures and potential risks explained to me by the researchers. I am satisfied that my concerns and questions have been addressed fully.

Yes ☐ No ☐

I understand that I have the right to withdraw my consent for being a participant at any time without giving reasons and without penalty.

Yes ☐ No ☐

I have read the information sheet describing this project and I have no known medical or other condition which would exclude me from being a participant in this experiment.

Yes ☐ No ☐

I would like my blood samples returned to me after analysis

Yes ☐ No ☐

I have been given one week to consider my involvement in the project.

Yes ☐ No ☐

I agree to participate as an experimental subject

Yes ☐ No ☐

I understand that thereafter I can withdraw at any time without reason and without penalty.

Yes ☐ No ☐

Signed: .............................
Name: .............................
Date: .............................
Appendix A

Participant Information Sheet

Project Title: The effects of sleep deprivation on the recovery of muscle and performance

Researchers:
Dr. Johann Edge
Ph (06) 350 4336 ext 7582
J.A.Edge@massey.ac.nz

Dr. Toby Mundel
Ph (06) 350 4336 ext 7763
T.mundel@massey.ac.nz

You have been invited to participate in a study investigating the effects of sleep deprivation on the recovery of muscle and running performance. Participation in this study is on a voluntary basis and all participants have the right to pull out, or ask questions at any time.

Why are we doing this study?
Many team-sports involve high-intensity exercise that often results in post-game fatigue, which may take days to recover from. Due to a number of circumstances such as post-competition travel and other outside stressors (i.e. family, alcohol intake, anticipation of competition), sleep disturbance is an occurrence encountered by many athletes. Despite the added stress that sleep disturbance has on the body, little is known regarding the acute effects that sleep deprivation has on recovery of performance or muscle.

What is the aim of this study?
The purpose of this study is to compare the effects of sleep deprivation versus habitual sleep on the recovery of muscle and running performance.

If I agree to take part, what will I have to do?
You will be asked to come into the laboratory of 4 occasions:

Visit 1. You will come in for an initial familiarisation session to ensure you are comfortable/familiar with all the facilities, equipment and protocol. Included in this familiarisation session will be a treadmill test of maximal aerobic capacity (VO₂peak), which will be used to determine the exercise intensities during an ensuing exercise trial.

Visit 2. You will come in for a single session that will include an 18-min graded exercise test (GXT) consisting of increasing intensities every 6 min (at 50, 65, 80% VO₂peak). During the GXT measures of
heart rate (via a chest strap), core temperature (via a swallowed non-toxic temperature pill) and respiratory gas will be measured. Following this GXT, you will complete a 50 minute intermittent-sprint exercise (ISE) protocol. The ISE consists of a 15-m maximal sprint each minute followed by sub-maximal exercise at varying intensities for the remainder of the minute (to simulate team-sport games such as hockey and the various football codes). Throughout the exercise protocol, performance measures (i.e. sprint time) will be recorded every minute. This session will also include a pre-exercise and post-exercise muscle biopsy from the vastus lateralis (thigh) muscle (2 muscle biopsies in total).

**Visits 3 & 4.** The remaining two sessions will involve two conditions with either (1) normal or (2) deprived sleep states. Each respective condition will be conducted over 2 consecutive days (one night sleep). For both sessions you will be asked to report to the laboratory at ~3pm. You will have swallowed the above-mentioned core temperature pill (approximately the size of a jelly bean) 5 hours prior to testing. Your weight, heart rate, core temperature and maximal voluntary and electrically-stimulated muscle contractions of your leg will be measured. The electrical stimulation will be achieved by placing two large pads covered with electrode gel onto the skin on the belly of the quadriceps (thigh) muscles. Pulses will be programmed into a series of timed pulses through a commercially available electrical stimulator to the electrodes. A suitable current will be obtained by increasing the current slowly until you find the sensation to be just tolerable.

You will then complete the 18-min GXT (as mentioned above for Visit 2) on a treadmill with measures of gas, heart rate and core temperature measured. A blood sample will be taken from a vein in your arm before and after the GXT.

Following the GXT, you will complete the 50-min ISE protocol (as mentioned above for Visit 2). Before and after the ISE, heart rate, core temperature, a blood sample and maximal voluntary and electrically-stimulated muscle contractions of your leg will be measured.

After the completion of this (day 1) protocol, you will be provided with all meals and required to remain in the laboratory for the remainder of the evening and morning under supervision of the researchers. During the normal sleep (control) condition, you will be encouraged to adhere to your normal sleeping pattern – this will be aided by an eye-mask and ear-plugs and of course a bed provided in laboratory. During the sleep deprivation trial you will be required to stay awake and will be provided with entertainment including DVD’s and video game to assist with this. Sleep will be monitored with motion detecting wrist watches (Actiwatches™) to examine the quantity and quality of sleep.

The following day (day 2), you will again complete the same testing protocol as on day 1 commencing at ~3pm. The only difference is that one hour before the day 2 exercise protocol a muscle biopsy will be sampled from the vastus lateralis (thigh) muscle.

*On completion of the study, the researchers will arrange for you to be taken home via taxi and in the case of the sleep deprivation trial you should under no circumstance undertake any activity that might cause you harm e.g. driving, handling machinery etc.*
What are the risks?

**Muscle Biopsy.** This procedure will be performed by an experienced medical Doctor (Dr. Michael Short). This is a common procedure used to determine muscle adaptations to exercise.

- Upon arrival to the laboratory the sites will be prepared for muscle samples. There will be a total of 4 muscle biopsies during the whole study.
- The muscle biopsy will involve the administering of a local anaesthetic to the outside part of the upper thigh. This will be followed by a small incision made through the skin. A 5 millimetre biopsy needle will then be inserted through the incision and into the muscle, in order to remove a small piece of muscle tissue (~80 – 100 mg, size of a match head).
- Finally the incision will be closed with steri-strips (like a band aid). After the last muscle biopsy appropriate recovery processes will be performed including, rest, ice, compression and elevation of the leg.
- The muscle biopsy process may result in slight discomfort (a mild “cork”), which may last for the following 1-3 days. This may be accompanied by local temporary bruising (although normally rare), along with the very small risk of superficial nerve damage in the skin (caused by the incision), which if present, may cause a temporary (approximately 1 - 10 days) loss of sensation to the area. However, this is also extremely rare. You should not perform vigorous exercise for the next 24-48 hours. However, you may resume light exercise approximately 24 hours after the procedure. If you get a haematoma due to the biopsy procedure the Doctor will apply pressure to the sample area to reduce the bleeding and follow the RICE procedures of rest, ice, compression and elevation. There is the extremely remote risk of a small decrease in muscle size at the site of the muscle biopsy. In the event that the intramuscular bleeding is not reduced using RICE procedures, we will book an immediate appointment to see Dr. Michael Short, who will recommend appropriate procedures to deal with this issue.”
- In the circumstance you feel unwell at any stage during the recovery period you should immediately call Johann Edge (Chief Investigator, see phone numbers at the end of this information sheet)

**Venous Blood Sample.** This procedure will be performed by an experienced phlebotomist (Dr. Toby Mundel). This is a common procedure used to determine physiological changes carried by the blood and will be very similar to any blood sample you have had taken at a doctor’s surgery or in hospital.

- There will be a total of 12 blood samples taken from a forearm vein during the whole study.
- There is a small needle prick and therefore, discomfort associated with placement of a needle. The risk of infection is extremely low, however there is chance of minor bruising. You can request to have any/all portions of your samples returned to you.

**Sleep Deprivation.** You may or may not experience effects of missing a night of sleep. Most often this will result in severe tiredness for a matter of hours to a day, however this could affect your energy and sleeping patterns for up to a week. Your balance, decision-making and concentration may also be affected therefore we strongly advise that you do not undertake any activity that might
cause you harm (such as driving, handling machinery etc.) until you have slept for a considerable time.

**What are the benefits?**

For the time you invest in this study, you will receive a $200 voucher. You will also have your peak aerobic fitness (VO$_{2}$peak) score and sprint performance given to you at the end of the study. Additionally, a meal will be provided both before exercise for study control purposes and after exercise to aid in your recovery. From the muscle and blood we will measure proteins (Mammalian target of rapamysin; mTOR) and hormones (growth hormone, insulin-like growth factor) important to the growth of your muscle, your personal results will be provided to you.

**What are my rights?**

- You can ask questions on any aspect of the project at any time, and we will do our best to answer them to your satisfaction.
- As a participant in the study you will provide information on the understanding that your name will not be used unless you give permission to the researcher.
- You have the right to view your own data at any stage and have it explained to you.
- You have the right to have any blood samples returned to you after they have been analyzed.
- You will also be given access to a summary of the project findings when it is concluded.
- You can withdraw from the project at any time, without giving any reason and without penalty.

**What about compensation for injury?**

If physical injury results from your participation in this study, you should visit a treatment provider to make a claim to ACC as soon as possible. ACC cover and entitlements are not automatic and your claim will be assessed by ACC in accordance with the Injury Prevention, Rehabilitation and Compensation Act 2001. If your claim is accepted, ACC must inform you of your entitlements, and must help you access those entitlements. Entitlements may include, but not be limited to, treatment costs, travel costs for rehabilitation, loss of earnings, and/or lump sum for permanent impairment. Compensation for mental trauma may also be included, but only if this is incurred as a result of physical injury. If your ACC claim is not accepted you should immediately contact the Johann Edge. Johann Edge will initiate processes to ensure you receive compensation equivalent to that to which you would have been entitled had ACC accepted your claim.

**Am I eligible?**

To participate in this study we require you are a competitive male or female in-season team-sport player (e.g. hockey, rugby, soccer) with a moderate to high fitness level and void of injury. It is also important for the female participants that you have a regular menstrual cycle. You must also be of good health. You will not be eligible to participate if any of the following apply:

- You have any known heart or cardiovascular condition or if a member of your family died below the age of fifty (50) as a result of a heart condition.
- In the last six months you have suffered from any painful injury or condition that lasted more than one week.
- You have ever had an injury or any medical condition that you think may affect your ability to sense pain or discomfort.
Appendix A

- You have ever had persistent or regular lower back pain.
- You are taking prescribed medication.
- You have cultural or religious sensitivities about human body measurements.
- You have any other reason to consider that you are not in good health and of average, or better than average, fitness.
- You are diabetic
- You are pregnant
- You or a family member has a bleeding disorder

Anything else I need to know?

You will be required to wear suitable sporting clothing and footwear that you feel comfortable exercising in. Water will be provided throughout the testing procedure and showers are also available should you need them. We also require you to fill in a food diary and training diary. For female participants we also ask that you map your menstrual cycle prior to testing.

All data obtained from this study will be kept strictly confidential. Data will be identified as a code only. Results will be made available to you at the completion of the study.

If you are interested in taking part:

Contact:
  Dr. Johann Edge
  Sport and Exercise Science lecturer
  Massey University, Palmerston North Campus
  Palmerston North, New Zealand
  Phone: +64 6 350 5799 ext 7582
  Mobile: 021 0236 7300
  e-mail: J.A.Edge@massey.ac.nz

  Dr. Toby Mundel
  Sport and Exercise Science lecturer
  Massey University, Palmerston North Campus
  Palmerston North, New Zealand
  Phone: (06) 350 5799
  e-mail: T.mundel@massey.ac.nz

This project has been reviewed and approved by the Massey University Human Ethics Committee: Southern A, Application 07/46. If you have any concerns about the conduct of this research, please contact Professor John O’Neill, Chair, Massey University Human Ethics Committee: Southern A, telephone 06 350 5799 x 8771, email humanethicsoutha@massey.ac.nz.
CONSENT FORM
Meal Replacement Diets on Intermittent-Sprint Performance in Team Sport Athletes

1) As a subject you are free to withdraw your consent to participate at any time.

2) The researchers will answer any questions you may have in regard to the study at any time.

Questions concerning the study can be directed to:

Principal Investigator (PhD Student):
Ms Melissa Skein
School of Human Movement Studies
Charles Sturt University
Ph: (02) 63386101
Ah: 0408274028

Supervisor:
Dr Rob Duffield
School of Human Movement Studies
Charles Sturt University
Ph: (02) 63384939

I, (print your name) _________________________________ have read the information contained within this consent form and any questions I have asked have been answered to my satisfaction.

I agree to participate in this project, realising I am free to withdraw my participation at any time without being subject to any penalty or discriminatory treatment.

I have been given the opportunity to ask questions about the research and received satisfactory answers.

I agree that the purpose of this research and potential risks or discomforts involved with the testing procedures have been sufficiently explained to me, with the opportunity to ask questions.

I understand that any information or personal details gathered in the course of this research about me is confidential and that neither my name nor any other identifying information will be used or published without my written permission.

Charles Sturt University’s Human Research Ethics Committee has approved this study.

I understand that if I have any complaints or concerns about this research I can contact:

Executive Officer
Human Research Ethics Committee, Office of Academic Governance
Charles Sturt University
Panorama Avenue, Bathurst NSW 2795
Ph: (02) 63384628 Fax: (02) 63384194

____________________ _______________________
Signature of participant (and parent/guardian if under 18 years of age)  Date

____________________ _______________________
Signature of investigator             Date
INFORMATION SHEET

Meal Replacement Diets on Intermittent-Sprint Performance in Team Sport Athletes

Principal Investigator (PhD Student): Melissa Skein
School of Human Movement Studies
Ph: (02) 63386101
Mob: 0408274028
mskein@csu.edu.au

Supervisor: Dr Rob Duffield
School of Human Movement Studies
Ph: (02) 63384939
rduffield@csu.edu.au

Purpose of the Study:
The purpose this investigation is to examine the effect of pre-exercise meal replacement diets and muscle glycogen concentration on intermittent-sprint (team sport) performance.

Procedures:

Overview
- Participants will be required to undertake a familiarisation session followed by 2 testing sessions which involves consecutive days of exercise.
- Conditions that will be completed include the consumption of two different meal replacement diets matched for volume between consecutive days of testing.
- All testing procedures will be completed in a controlled environment of 22°C

Pre-Exercise
- Participants will be required to be in a rested state prior to all testing sessions and avoid any consumption of food, alcohol or caffeine at least 3 hours prior to testing.
- All food, drink and activity in the 24-h prior to each testing session will be recorded and replicated for following sessions

Exercise Protocol
On arrival participants will complete the following measures and exercise protocol in the order below
- Resting physiological measures of urine specific gravity (USG), nude mass, heart rate, and collection of capillary and venous blood samples, and muscle biopsy (Day 2 only)
- Resting physiological measures and endurance muscle strength test of quadriceps, involving 15 maximal voluntary contractions (MVC) with electrical stimulation to knee extensors on first and final 5 contractions.
- Day 1 – Muscle glycogen depletion exercise protocol includes 90 min of continuous cycling on a stationary bike exercising at rotating intensities of 60% and 75% VO2max (maximal aerobic capacity) every 15min.
- Day 2 - Intermittent-sprint protocol consisting of 50-min continuous intermittent sprints, with a 1-min breaks every 10min. The protocol included a 15-m maximal sprint every minute, separated by recoveries of varying intensities of hard running, jogging and walking until the end of each minute. During the ‘hard running’, participants will be required to cover as much distance as possible up and down the 15-m running
track, while only required to jog or walk respectively in the allocated minute. Participants will perform a 15-m sprint into the crash mat against the laboratory wall to help and protect the participant when coming to a halt.

- Immediately following the repeat sprint protocol, all physiological measures will be recorded (and a muscle biopsy on day 2), immediately followed by 15 endurance contractions of knee extensors.

**Measures**

- **Performance measures** include 15-m sprint time and distance covered during self-paced exercise bouts which will be recorded each minute. 15-m sprint time will recorded with timing gates placed at 0 and 15-m of running track while distance covered throughout each sub-maximal bout will be manually measured with 1-m markings on the track. Double leg bounds will be measured by manually measuring the distance covered over 8 consecutive bounds.

- **Voluntary activation** measures will be completed pre and post intervention and immediately post exercise with use of endurance maximal voluntary contractions (MVC) with electrical stimulation. Participants will be seated with knee position at 90° flexion (0° represents full extension) with stimulus electrodes placed on the knee extensors. Participants will complete 15 maximal contractions of knee extensors (quadriceps) for 3 sec with 5 sec rest between each. The initial and final 5 MVC will be superimposed with an electrical stimulus to knee extensors. The electrical stimulation is a transient sensation to the quadriceps muscle and is completed with minimal harm.

- **Muscle Biopsy** samples will be taken by local medical doctor, Dr Neil Meulman pre and post exercise from the vastus lateralis (thigh) muscle pre and post exercise on Day 2 of the sessions. The site will be located in the belly of the muscle and numbed with a local anaesthetic, with only a feeling of pressure experienced during the biopsy procedure. During the procedure the Dr will make a small (~1cm) incision in the skin and advance the biopsy needle into the incision, through the fascia into the muscle where a small piece of muscle will be extracted.

- **Venous blood** samples will be taken pre and post exercise with a sterilised needle into a superficial vein in the forearm, measuring heat shock protein (HSP70) and S100B.

- **Capillary blood** sample will be taken with a small incision with a lancet to the earlobe pre and post exercise and 30-min break of protocol and will be measured for pH, lactate, glucose, bicarbonate (HCO₃⁻), oxygen concentration and haemoglobin.

- **Nude mass** will be recorded pre and post exercise within an enclosed private area.

- **Heart rate** will be measured pre, post and every 5min during protocol with a chest heart rate monitor.

- **Rating of Perceived Exertion (RPE)** This perceptual measure indicates how hard to perceived to be working at a given time point. This will be asked every 10-min during the intermittent-sprint protocol.

**Experimental Conditions (completed in a double-blind, semi-randomised order)**

**Session 1 and 2:** Participants will be required to consume two different liquid based diets that will be matched for volume between exercise sessions completed on Day 1 and Day 2. All food and fluid will be provided by the research team and this is to be the only food that is to be consumed during this time. A food and exercise diary will also be completed during each testing session.

Prior consent will be gained for any visual recording of any testing session (photographs/videos etc.) from the subjects involved and these recordings will remain under confidential storage and only published with the express permission of the subject involved. If the data is published in any form, participant names and distinguishing features will remain confidential.

**Subject Benefit:**

Participants will receive information pertaining to their repeat sprint ability performance, peak sprinting speed, peak and mean force output and indications of match-fitness and areas for improvement in fitness and conditioning as related to team sport scenarios. Participants will also receive a School of Human Movement Studies water bottle at the conclusion of the testing.
Appendix A

Major Issues:

- Muscle biopsy extraction will be taken pre and post exercise on Day 2 of each experimental trial. A local anaesthetic will be administered to the biopsy site which will cause some discomfort. Once the site is numb the participant will only feel pressure and minimal discomfort during the biopsy procedure. This procedure will be completed by a medical doctor (Dr Meulman) within sterilised equipment and environment.
  - Approximately 8 hours after the first biopsy, the muscle is likely to be moderately sore for about 24 hours, similar to muscle soreness following unusually vigorous exercise or a muscle injury. Complications accompanying this procedure are rare. The primary concern would be prolonged bleeding which could produce a bruise and could extend the period of muscle soreness, but is adequately treated with rest, ice, compression, and elevation.
  - Although the muscle selected for biopsying (vastus lateralis) has no major blood vessels or nerves in the areas where the biopsy needle will be inserted, there is the rare occurrence of compressing or cutting small nerve branches which can sometimes cause temporary tingling and numbness in the skin. These responses, when they have occurred, have dissipated in a few days or weeks. Every effort will be made to minimize the length and degree of muscle soreness.
- Venous blood samples (5mL) will be taken with a needle from a superficial vein of the forearm. While procedure may cause some discomfort to the participant, procedure is brief and with be completed by a research investigator experienced in the procedure.
- Capillary blood sample (100µL) will be taken with a small lancet from the earlobe. This procedure may cause a brief feeling of discomfort however is of minimal harm.
- Electrical stimulation applied to the knee extensors will cause a brief (~0.3sec) painful sensation to the muscle (quadriceps). This is a transient sensation and is of minimal harm to the participant. Participants will be fully informed and familiarised with the procedure prior to testing.
- Nature of the testing protocol requires prolonged, high intensity exercise in warm environmental conditions. All participants in the study are of adequate fitness to complete the protocol and will be familiarised with protocol and conditions prior to testing.

Time Commitments:
Each testing session will take approximately 2 hours and will be organised for a time that is convenient for both subject and investigator. Sessions will be separated by a week, with no less than 5 days between sessions. However, if for some reason, the participant can not finish all testing requirements, they are free to withdraw from the project at any time without penalty or discriminatory treatment.

Contact Details:
If participants have any queries through out the testing procedures, they can contact the researcher on:

Mrs Melissa Skein  
School of Human Movement  
Charles Sturt University  
Ph: 6338 6101 (wk)  
Email: mskein@csu.edu.au

Dr Rob Duffield  
School of Human Movement  
Charles Sturt University  
Ph: 6338 4939  
Email: rduffield@csu.edu.au

Note: Charles Sturt University's Ethics in Human Research Committee has approved this project. If you have any complaints or reservations about the ethical conduct of this project, you may contact the Committee treatment through the executive Officer:

The Executive Officer  
Ethics in Human Research Committee  
Charles Sturt University  
Private Mail Bag 29  
Bathurst, NSW, 2795. Tel: (02) 6338 4628  Fax: (02) 6338 4194

Any issues you raise will be treated in confidence and investigated fully and you will be informed of the outcome.
CONSENT FORM

The Effect of Pre-cooling and Passive Heating on Intermittent-Sprint Exercise

1) As a subject you are free to withdraw your consent to participate at any time.
2) The researchers will answer any questions you may have in regard to the study at any time.

Questions concerning the study can be directed to:

**Principal Investigator (PhD Student):**
Ms Melissa Skein  
School of Human Movement Studies  
Charles Sturt University  
Ph: (02) 63386101  
Ah: 0408274028

**Supervisor:**
Dr Rob Duffield  
School of Human Movement Studies  
Charles Sturt University  
Ph: (02) 63384939

I, (print your name) _________________________________ have read the information contained within this consent form and any questions I have asked have been answered to my satisfaction.

I agree to participate in this project, realising I am free to withdraw my participation at any time without being subject to any penalty or discriminatory treatment.

I have been given the opportunity to ask questions about the research and received satisfactory answers.

I agree that the purpose of this research and potential risks or discomforts involved with the testing procedures have been sufficiently explained to me, with the opportunity to ask questions.

I understand that any information or personal details gathered in the course of this research about me is confidential and that neither my name nor any other identifying information will be used or published without my written permission.

Charles Sturt University’s Human Research Ethics Committee has approved this study.

I understand that if I have any complaints or concerns about this research I can contact:

**Executive Officer**  
Human Research Ethics Committee, Office of Academic Governance  
Charles Sturt University  
Panorama Avenue, Bathurst NSW 2795  
Ph: (02) 63384628 Fax: (02) 63384194

______________________________  ________________  ________________
Signature of participant (and parent/guardian if under 18 years of age)  Date

______________________________  ________________
Signature of investigator  Date
INFORMATION SHEET

The Effect of Pre-cooling and Passive Heating on Intermittent-Sprint Exercise

Principal Investigator (PhD Student): Melissa Skein
School of Human Movement Studies
Ph: (02) 63386101
Mob: 0408274028
mskein@csu.edu.au

Supervisor: Dr Rob Duffield
School of Human Movement Studies
Ph: (02) 63384939
rduffield@csu.edu.au

Purpose of the Study:
The purpose this investigation is to examine the effect of pre-exercise cooling and passive heating on intermittent-sprint (team sport) performance in the heat.

Procedures:

Overview
- Participants will be required to undertake a familiarisation session followed by 3 testing sessions.
- Conditions that will be completed include hot (CONT) condition; hot conditions with pre-exercise passive heating (PHEAT) and hot condition with pre-exercise cooling (COOL). All conditions will be completed in a controlled environment of 33°C and 30% relative humidity.

Pre-Exercise
- Participants will ingest a small, non-toxic core temperature capsule (previously approved by Ethics Committee) 4-hours prior and consume 500ml water 1 h prior to testing
- Participants will be required to be in a rested state prior to all testing sessions and avoid any consumption of food, alcohol or caffeine at least 3 hours prior to testing.
- All food, drink and activity in the 24-h prior to each testing session will be recorded and replicated for following sessions

Exercise Protocol
On arrival participants will complete the following measures and exercise protocol in the order below
- Resting physiological measures and endurance muscle strength test of quadriceps, involving 15 maximal voluntary contractions (MVC) with electrical stimulation to knee extensors on first and final 5 contractions.
- Completion of the respective intervention (control, passive heating or pre-cooling) followed by endurance muscle strength test again.
- Intermittent-sprint protocol consisting of 60-min continuous intermittent sprints, with a 1-min breaks every 10min. The protocol included a 15-m maximal sprint every minute, separated by recoveries of varying intensities of hard running, jogging and walking until the end of each minute. During the ‘hard running’, participants will be required to cover as much distance as possible up and down the 15-m running track, while only required to jog or walk respectively in the allocated minute. Participants will perform a 15-m sprint into the crash mat against the laboratory wall to help and protect the participant when coming to a halt.
Immediately following the repeat sprint protocol, all physiological measures will be recorded, immediately followed by 15 endurance contractions of knee extensors.

**Measures**

**Performance measures** include 15-m sprint time and distance covered during self-paced exercise bouts which will be recorded each minute. 15-m sprint time will recorded with timing gates placed at 0 and 15-m of running track while distance covered throughout each sub-maximal bout will be manually measured with 1-m markings on the track. Double leg bounds will be measured by manually measuring the distance covered over 8 consecutive bounds.

**Voluntary activation** measures will be completed pre and post intervention and immediately post exercise with use of endurance maximal voluntary contractions (MVC) with electrical stimulation. Participants will be seated with knee position at 90° flexion (0° represents full extension) with stimulus electrodes placed on the knee extensors. Participants will complete 15 maximal contractions of knee extensors (quadriceps) for 3 sec with 5 sec rest between each. The initial and final 5 MVC will be superimposed with an electrical stimulus to knee extensors. The electrical stimulation is a transient sensation to the quadriceps muscle and is completed with minimal harm.

**Nude mass** will be recorded pre and post exercise within an enclosed private area.

**Heart rate** will be measured pre, post and every 5min during protocol with a chest heart rate monitor.

**Core temperature** will be measured through the ingestion (non-harmful) telemetric capsule the size of a jelly bean 4 hour prior to exercise and temperature detected with a Vital-Sense monitor.

**Skin temperature** will record on the same monitor with patches placed on the chest, forearm and calf.

**Venous blood** samples will be taken pre and post exercise with a sterilised needle into a superficial vein in the forearm, measuring insulin-like growth factor (IGF-1), heat shock protein (HSP70) and interleukin-6 (IL-6).

**Capillary blood** sample will be taken with a small incision with a lancet to the earlobe pre and post exercise and 30-min break of protocol and will be measured for pH, lactate, glucose, bicarbonate (HCO₃⁻), oxygen concentration and haemoglobin.

**Perceptual measures** using subjective rating scales will be assessed, including Rating of Perceived Exertion (RPE) and Thermal Stress using a 1 – 10 Likert scale. RPE and Thermal Stress will be recorded pre and post exercise and every 10-min during protocol.

**Experimental Conditions**

**CONT Condition:** Participants will be seated in a thermo-neutral (22°C) room for the same duration as passive heating condition (~10min) prior to exercise protocol in warm environmental conditions.

**PHEAT Condition:** Participants will be seated in hot environmental conditions (40°C) to raise core temperature by ~1°C from resting measures prior to exercise protocol in warm environmental conditions.

**COOL Condition:** Participants will be submerged in an ice bath (10°C) up to the suprasternal notch (shoulder height) for 15-min prior to testing protocol in warm environmental conditions.

Prior consent will be gained for any visual recording of any testing session (photographs/videos etc.) from the subjects involved and these recordings will remain under confidential storage and only published with the express permission of the subject involved. If the data is published in any form, participant names and distinguishing features will remain confidential.

**Subject Benefit:**

Participants will receive information pertaining to their repeat sprint ability performance, peak sprinting speed, peak and mean force output and indications of match-fitness and areas for improvement in fitness and conditioning as related to team sport scenarios. Participants will also receive a School of Human Movement Studies water bottle at the conclusion of the testing.
Major Issues:

- Venous blood samples (5mL) will be taken with a needle from a superficial vein of the forearm. While procedure may cause some discomfort to the participant, procedure is brief and will be completed by a research investigator experienced in the procedure.
- Capillary blood sample (100µL) will be taken with a small lancet from the earlobe. This procedure may cause a brief feeling of discomfort however is of minimal harm.
- Electrical stimulation applied to the knee extensors will cause a brief (~0.3sec) painful sensation to the muscle (quadriceps). This is a transient sensation and is of minimal harm to the participant. Participants will be fully informed and familiarised with the procedure prior to testing.
- Nature of the testing protocol requires prolonged, high intensity exercise in warm environmental conditions. All participants in the study are of adequate fitness to complete the protocol and will be familiarised with protocol and conditions prior to testing.

Time Commitments:
Each testing session will take approximately 2 hours and will be organised for a time that is convenient for both subject and investigator. Sessions will be separated by a week, with no less than 5 days between sessions. However, if for some reason, the participant can not finish all testing requirements, they are free to withdraw from the project at any time without penalty or discriminatory treatment.

Contact Details:
If participants have any queries throughout the testing procedures, they can contact the researcher on:

Ms Melissa Skein
School of Human Movement
Charles Sturt University
Ph: 6338 6101 (wk)
0408274028 (ah)
Email: mskein@csu.edu.au

Dr Rob Duffield
School of Human Movement
Charles Sturt University
Ph: 6338 4939
Email: rduffield@csu.edu.au

Note: Charles Sturt University’s Ethics in Human Research Committee has approved this project. If you have any complaints or reservations about the ethical conduct of this project, you may contact the Committee treatment through the executive Officer:

The Executive Officer
Ethics in Human Research Committee
Academic Secretariat
Charles Sturt University
Private Mail Bag 29
Bathurst, NSW, 2795.

Tel: (02) 6338 4628
Fax: (02) 6338 4194

Any issues you raise will be treated in confidence and investigated fully and you will be informed of the outcome.
Appendix B

Rating Scales

Questionnaires
Borg 6-20 Rating of Perceived Exertion Scale

On the scale below, rate how easy or hard you think your body is working right now

6
7  Very, very light
8
9  Very light
10
11  Fairly Light
12
13  Somewhat Hard
14
15  Hard
16
17  Very Hard
18
19  Very, Very Hard
20
## Thermal Sensations

<table>
<thead>
<tr>
<th>Value</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.0</td>
<td>Unbearably Cold</td>
</tr>
<tr>
<td>0.5</td>
<td></td>
</tr>
<tr>
<td>1.0</td>
<td>Very Cold</td>
</tr>
<tr>
<td>1.5</td>
<td></td>
</tr>
<tr>
<td>2.0</td>
<td>Cold</td>
</tr>
<tr>
<td>2.5</td>
<td></td>
</tr>
<tr>
<td>3.0</td>
<td>Cool</td>
</tr>
<tr>
<td>3.5</td>
<td></td>
</tr>
<tr>
<td>4.0</td>
<td>Comfortable</td>
</tr>
<tr>
<td>4.5</td>
<td></td>
</tr>
<tr>
<td>5.0</td>
<td>Warm</td>
</tr>
<tr>
<td>5.5</td>
<td></td>
</tr>
<tr>
<td>6.0</td>
<td>Hot</td>
</tr>
<tr>
<td>6.5</td>
<td></td>
</tr>
<tr>
<td>7.0</td>
<td>Very Hot</td>
</tr>
<tr>
<td>7.5</td>
<td></td>
</tr>
<tr>
<td>8.0</td>
<td>Unbearably Hot</td>
</tr>
</tbody>
</table>
## POMS Rating Scale

How do you feel **right now?**

<table>
<thead>
<tr>
<th></th>
<th>Not at all</th>
<th>A Little</th>
<th>Moderately</th>
<th>Quite a bit</th>
<th>Extremely</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tense</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Worn Out</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Unhappy</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Clear-headed</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Lively</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Listless</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Sad</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Active</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Grouchy</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Energetic</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Hopeless</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Relaxed</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Uneasy</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Restless</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Unable to concentrate</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Fatigued</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Annoyed</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Discouraged</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Nervous</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Miserable</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Muddled</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Cheerful</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Bitter</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Exhausted</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Anxious</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Gloomy</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Sluggish</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Helpless</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Weary</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Bewildered</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Alert</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Vigorous</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
</tbody>
</table>
Appendix C

Conference Abstracts
Conference Proceedings

2012  Skein M, Duffield R, Kelly B, Marino F.E  
*Exercise and Sports Science Australia (ESSA/SDA) Conference – Gold Coast, Australia*  
Lowered carbohydrate intake and muscle glycogen content reduces intermittent-sprint performance despite no conscious awareness of diet manipulation

2011  Skein M, Duffield R, Marino F.E, Cannon J  
*European Conference of Sports Sciences (ECSS) – Liverpool, UK*  
The effect of pre-cooling and passive heating on pacing strategies and intermittent-sprint performance in team sport athletes  
Mündel T, Skein M, Duffield R, Edge J  
*European Conference of Sports Sciences (ECSS) – Liverpool, UK*  
Physiological responses to consecutive day treadmill running following one night of sleep deprivation

2010  Skein M, Duffield R, Edge J, Short M.J, Mündel T  
*Exercise and Sports Science Australia (ESSA/SDA) Conference – Gold Coast, Australia*  
The effects of sleep deprivation on pacing strategies and intermittent-sprint performance (Poster Presentation – Awarded Young Investigators Award)

2009  Skein M, Duffield R, Edge J, Short M.J, Mündel T  
*European Conference of Sports Sciences (ECSS) – Oslo, Norway*  
The effects of sleep deprivation on intermittent-sprint performance and pacing strategies
The Effect of Sleep Deprivation on Pacing Strategies and Intermittent-Sprint Performance.

Skein Melissa¹, Duffield, Rob¹, Mundel, Toby², Edge, Johann²,³ & Short, Michael²
¹ School of Human Movement Studies, Charles Sturt University, Bathurst, AUSTRALIA.
² Institute of Food, Nutrition, and Human Health, Massey University, Palmerston North, NEW ZEALAND.
³ Department of Sport and Exercise, The University of Auckland, Auckland, NEW ZEALAND.

Correspondence: mskein@csu.edu.au

* Awarded Young Investigators Award – Poster Presentation

Introduction:
Team sport athletes may often experience sleep loss prior to training or competition. Accordingly, this study examined the effects of sleep deprivation on pacing strategies and performance during intermittent-sprint exercise.

Methods:
Following Ethics clearance and informed consent, 10 male, team-sport athletes completed 3 trials of a 30-min treadmill run and 50-min intermittent-sprint protocol (ISP). Participants completed a baseline session and 2 identical trials of consecutive day exercise, separated by no sleep (SDEP) or normal sleep (CONT). The ISP included a 15-m sprint followed by self-paced exercise each minute. Muscle biopsies from the vastus lateralis were assessed for muscle glycogen, while voluntary force (VF) and activation (VA) of right knee extensors were assessed pre- and post-exercise. Various physiological and perceptual measures were recorded throughout. A repeated-measure ANOVA was used to determine differences between conditions.

Results:
Sprint times were reduced on Day2 in both conditions, with a greater decline within SDEP. Distances covered during free-paced efforts were lower in SDEP Day2 v Day1. Muscle glycogen was lower pre-exercise Day2 SDEP compared to CONT. RPE and POMS were more adversely affected in SDEP on Day2. SDEP pre-exercise VF and VA on Day2 were reduced compared to CONT.

Conclusions:
ISP performance and VF were negatively affected on Day2 by SDEP. This may be due to the hindered repletion of muscle glycogen before exercise on day 2 following sleep loss. Further, increased psychological strain prior to exercise may also result in the reduced VF and VA and the slower pacing strategies adopted following SDEP.
The effect of pre-cooling and passive heating on pacing strategies and intermittent-sprint performance in team sport athletes

Melissa Skein, Rob Duffield, Frank E Marino, Jack Cannon

School of Human Movement Studies, Charles Sturt University, Bathurst, AUSTRALIA

Introduction
Team sport athletes often engage in pre-cooling prior to exercise in the heat to reduce thermoregulatory strain and improve performance. Conversely, warm-up practices are used amongst athletes to increase endogenous temperature and improve subsequent high-intensity exercise performance. This paradox in pre-exercise interventions raises questions concerning which thermal state is optimal for subsequent intermittent-sprint performance. Therefore, this study examined the effects of pre-exercise cooling and heating on pacing and intermittent-sprint performance in the heat.

Methods
Ten male, team-sport athletes completed three randomized conditions including, a thermo-neutral environment (CONT); whole body submersion in an ice-bath (ICE) and passive-heating in a hot environment (HEAT) prior to 50min intermittent-sprint exercise (ISE) in the heat (31 ± 1°C). Exercise involved repeated 15 m maximal sprints and self-paced exercise of varying intensities (hard running, jogging, walking, and bounding). Performance was measured by sprint times and total distances covered (TDC), and pacing strategies were assessed via distance covered during 10min phases of the ISE. 15 maximal contractions were performed to determine maximal voluntary torque (MVT), activation (VA), and twitch contractile properties (TCP). Physiological measures included heart rate (HR), core (Tcore) and skin (Tskin) temperatures, physiological strain index (PSI), capillary blood, and perceptual ratings.

Results
Mean sprint times, especially during the initial 10min, were slower during ICE compared to HEAT (P<0.05). TDC was not different between conditions but less distance was covered during HEAT in the final 10min compared to ICE and during 31-40min compared to CONT (P<0.05). Post-ex MVT was reduced within CONT and HEAT, and VA was higher post-intervention and post-ex during ICE. Peak torque and relaxation rates were improved post-intervention during HEAT compared to ICE. Finally, HR, Tcore, Tskin and PSI during exercise were lower in ICE compared to CONT and HEAT (P<0.05).

Conclusion
Initial sprint performance was faster following HEAT and slowed by ICE, possibly due to altered contractile properties at the start of exercise. Conversely, pre-cooling maintained endurance performance later in the protocol, while HEAT resulted in greater performance declines. ICE maintained MVT and VA post-exercise compared to HEAT, suggesting improved skeletal recruitment. Accordingly, passive heating augmented initial sprint times yet reduced pacing strategies later in the protocol, while pre-cooling reduced cardiovascular and thermoregulatory strain and allowed for selection of higher exercise intensities throughout the protocol.
Lowered carbohydrate intake and muscle glycogen content reduces self-paced intermittent-sprint performance and pacing strategies despite no conscious awareness of diet manipulation.

Skein Melissa¹, Duffield Rob¹, Kelly Bradley², Marino Frank E¹

¹ School of Human Movement Studies, Charles Sturt University, Bathurst, AUSTRALIA.
² School of Sport Science, Exercise and Health, The University of Western Australia, Crawley, WA, AUSTRALIA.

Correspondence: mskein@csu.edu.au

Introduction:
The aim of this study was to examine the effect of high and low carbohydrate diets following a depleting bout of exercise on muscle glycogen and self-paced intermittent-sprint performance in team-sport athletes. Secondly, the study aimed to examine these effects of carbohydrate ingestion and muscle glycogen content without the subject’s knowledge in order to exclude potential conscious regulation of exercise intensities from knowledge of such events.

Methods:
10 healthy, male, team-sport athletes volunteered to participate in the study. Subjects completed two testing sessions, each involving two consecutive days of exercise. Day 1 included a glycogen depleting 95min intermittent, upper and lower body cycling protocol, followed by an assigned diet for the remainder of the day and breakfast on day 2. Respective diets contained either high (HCHO; 7g kg bw) or low (LCHO; 2g kg bw) carbohydrate and were allocated in a double-blind, semi-randomised order. Subjects were deceived that carbohydrate intake and muscle glycogen content were altered or assessed during the study. On day 2, subjects completed a 60 min self-paced intermittent-sprint protocol, including 15m maximal sprints every minute and self-paced efforts at sub-maximal intensities (hard running, jogging, walking and bounds). Muscle biopsies were obtained from the pre and post exercise on day 2 and heart rate and rating of perceived exertion (RPE) were recorded during exercise on day 1 and 2. Assessment of neuromuscular function of the right knee extensors including maximal voluntary torque (MVC) and voluntary activation (VA) were obtained pre and post exercise. Testing procedures were approved by the Research Ethics Committee and written and verbal informed consent was obtained from the subject prior to commencement of any trial.

Results:
The HCHO diet resulted in a significantly increased muscle glycogen content compared to LCHO prior to the intermittent-sprint protocol (597 ± 115 vs 318 ± 72 mmol kg⁻¹ dw; P<0.05). On day 2, total distance covered during the self-paced efforts, particularly during the hard running bouts were significantly greater following HCHO (P<0.05). Interestingly, distances covered during initial efforts were similar between conditions; however, greater declines in pacing became evident in LCHO within minutes, and remained throughout the protocol. Carbohydrate intake had no effect on maximal sprint times, jogging, walking and bounding distance. Further, no differences in pre or post MVC or VA, or heart rate and RPE during exercise were evident between conditions (P>0.05).

Conclusion/Discussion:
A high carbohydrate diet improves subsequent pacing strategies and self-paced intermittent-sprint exercise as evidenced by increased total distance covered, particularly during the high-intensity, hard running efforts. As subjects were deceived to the manipulation of carbohydrate intake, it is suggested that the regulation of exercise intensities were due to feedback from the periphery regarding physiological and metabolic perturbations associated with reduced muscle glycogen content rather than a conscious decision to manipulate pacing strategies due to knowledge of CHO intake.
Lowered carbohydrate intake and muscle glycogen content reduces self-paced intermittent-sprint performance and pacing strategies despite no conscious awareness of diet manipulation.

Skein Melissa¹, Duffield Rob¹, Kelly Bradley², Marino Frank E¹

¹ School of Human Movement Studies, Charles Sturt University, Bathurst, AUSTRALIA.
² School of Sport Science, Exercise and Health, The University of Western Australia, Crawley, WA, AUSTRALIA.

Correspondence: mskein@csu.edu.au

Introduction:
The aim of this study was to examine the effect of high and low carbohydrate diets following a depleting bout of exercise on muscle glycogen and self-paced intermittent-sprint performance in team-sport athletes. Secondly, the study aimed to examine these effects of carbohydrate ingestion and muscle glycogen content without the subject’s knowledge in order to exclude potential conscious regulation of exercise intensities from knowledge of such events.

Methods:
10 healthy, male, team-sport athletes volunteered to participate in the study. Subjects completed two testing sessions, each involving two consecutive days of exercise. Day 1 included a glycogen depleting 95min intermittent, upper and lower body cycling protocol, followed by an assigned diet for the reminder of the day and breakfast on day 2. Respective diets contained either high (HCHO; 7g kg⁻¹ bw) or low (LCHO; 2g kg⁻¹ bw) carbohydrate and were allocated in a double-blind, semi-randomised order. Subjects were deceived that carbohydrate intake and muscle glycogen content were altered or assessed during the study. On day 2, subjects completed a 60 min self-paced intermittent-sprint protocol, including 15m maximal sprints every minute and self-paced efforts at sub-maximal intensities (hard running, jogging, walking and bounds). Muscle biopsies were obtained from the pre and post exercise on day 2 and heart rate and rating of perceived exertion (RPE) were recorded during exercise on day 1 and 2. Assessment of neuromuscular function of the right knee extensors including maximal voluntary torque (MVC) and voluntary activation (VA) were obtained pre and post exercise. Testing procedures were approved by the Research Ethics Committee and written and verbal informed consent was obtained from the subject prior to commencement of any trial.

Results:
The HCHO diet resulted in a significantly increased muscle glycogen content compared to LCHO prior to the intermittent-sprint protocol (597 ± 115 vs 318 ± 72 mmol kg⁻¹ dw; P<0.05). On day 2, total distance covered during the self-paced efforts, particularly during the hard running bouts were significantly greater following HCHO (P<0.05). Interestingly, distances covered during initial efforts were similar between conditions; however, greater declines in pacing became evident in LCHO within minutes, and remained throughout the protocol. Carbohydrate intake had no effect on maximal sprint times, jogging, walking and bounding distance. Further, no differences in pre or post MVC or VA, or heart rate and RPE during exercise were evident between conditions (P>0.05).

Conclusion/Discussion:
A high carbohydrate diet improves subsequent pacing strategies and self-paced intermittent-sprint exercise as evidenced by increased total distance covered, particularly during the high-intensity, hard running efforts. As subjects were deceived to the manipulation of carbohydrate intake, it is suggested that the regulation of exercise intensities were due to feedback from the periphery regarding physiological and metabolic perturbations associated with reduced muscle glycogen content rather than a conscious decision to manipulate pacing strategies due to knowledge of CHO intake.
Appendix D

Photographs
A) Subject completing maximal sprint during the self-paced intermittent-sprint exercise (ISE) protocol; B) subject completing maximal voluntary contraction (MVC) superimposed with electrical stimulus to the femoral nerve that was completed during all studies; C) muscle biopsy procedure completed during the sleep deprivation and carbohydrate studies.
D) Subjects seated and within the laboratory during the sleep deprivation condition; E) pre-exercise cooling via submersion in an ice bath and passive heating via seated in a hot, humid climate chamber during the thermoregulation study.