A Multi-Disciplinary Approach to Alternative Training Methods for Endurance Athletes and Their Relationship with Performance

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Abstract

Title: A Multi-Disciplinary Approach to Alternative Training Methods for Endurance Athletes and Their Relationship with Performance

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The physiological determinants most closely associated with successful endurance performance are factors primarily related to oxygen uptake including $\dot{V}O_{2\text{max}}$, economy of movement and lactate threshold. As a consequence, endurance athletes typically spend extended periods of training utilising modalities designed to improve aerobic capacity. Despite this, successful endurance performance in sports such as rowing requires not only a high aerobic capacity but also muscular strength and anaerobic power necessary for attacking, pace changing and final sprints. Through a multi-disciplinary approach, the aim of this research was to investigate the impact of alternative methods of training on well-trained endurance athletes in order to maximise training adaptations of the key biomechanical and physiological determinants associated with endurance performance.

The effect of maximal stretch shortening cycle (SSC) fatigue on the biomechanical performance of both well-trained endurance and strength trained athletes was investigated. Maximal SSC fatigue had an immediate, debilitating effect on the performance of subsequent SSC activities for both strength and endurance athletes. This effect was realised through reductions in performance outcome and the biomechanical performance of the jump indicating that the effectiveness of the SSC was significantly reduced resulting in a decrease in performance. During recovery an enhancement above baseline values was observed for both groups of athletes. This enhancement may be attributable to a post activation potentiation (PAP) effect, leading to an acute improvement in performance as a result of prior muscle activation, whereby subsequent SSC activities can be performed with a more effective SSC.

To successfully prescribe training for well-trained endurance athletes an in-depth knowledge of the specific demands of the sport is pertinent and therefore the physiological determinants of 2000 m rowing ergometer performance were also investigated. The main finding was that $W\dot{V}O_{2\text{max}}$ was the strongest correlate of performance and was the variable with the most influence on performance. It has been suggested that in well-trained athletes, additional increases in aerobic training may not result in any further improvement in endurance performance or associated physiological variables and therefore the effect of high intensity interval training (HIIT) on well-trained rowers was investigated. The results revealed that eight weeks of HIIT performed at 100% peak power output was a more effective means than traditional, long, slow distance training to elicit improvements in $\dot{V}O_{2\text{max}}$ and power output associated with lactate threshold. HIIT also resulted in a significant improvement in 2000 m time trial improvement, although this improvement was not significantly greater than that observed with traditional training.

In summary, the findings of this thesis indicate that well-trained endurance athletes have the potential to elicit a similar PAP effect to that observed in strength trained athletes following maximal SSC fatigue. HIIT was also identified as an effective modality to optimise the development of aerobic characteristics and enhance rowing performance. In conclusion, this research adds to the existing body of research by showing that, compared to traditional training modalities adopted, alternative training methods such as maximal SSC activities and HIIT may further enhance biomechanical and physiological function and performance in well-training endurance athletes.
Authors Declaration

I hereby declare that the work contained in this thesis is my own, and was completed with counsel of my supervisors, Dr. AJ Harrison of the Department of Physical Education and Sport Sciences, University of Limerick and Dr. GD Warrington of the Department of Health and Human Performance, Dublin City University. The work has not been submitted to any other University or higher education institution, or for any other academic award with this University.

____________________           _____________  __________________
Niamh Ní Chéilleachair    Dr A.J. Harrison    Dr G.D. Warrington
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# Table of Contents

Abstract ....................................................................................................................................................... i

Authors Declaration .................................................................................................................................. ii

Acknowledgements ................................................................................................................................... iii

Table of Contents ....................................................................................................................................... iv

List of Figures ............................................................................................................................................ ix

List of Tables .............................................................................................................................................. xi

List of Equations ....................................................................................................................................... xiii

List of Abbreviations ................................................................................................................................ . xiv

Glossary of Terms ..................................................................................................................................... xvi

Preface ......................................................................................................................................................... xvii

Chapter 1. Introduction .............................................................................................................................. 1

  1. 1. Background and Rationale for Research ................................................................................... 2
  1. 2. Aim of Research ............................................................................................................................ 3
  1. 3. Objectives of Research ................................................................................................................ 4
  1. 4. Research Questions ...................................................................................................................... 4
  1. 5. Thesis Outline ............................................................................................................................... 5

Chapter 2. Literature Review ..................................................................................................................... 7

  2. 1. Introduction ................................................................................................................................... 8
  2. 2. Background .................................................................................................................................... 8
  2. 3. Concurrent Training ...................................................................................................................... 9
      2. 3. 1. Concurrent Training and Strength Athletes ................................................................ 10
      2. 3. 2. Concurrent Training and Endurance Athletes............................................................ 10
  2. 4. Stretch-Shortening Cycle .......................................................................................................... 12
      2. 4. 1. SSC Mechanisms ............................................................................................................. 13
      2. 4. 1. 1. Time Available for Force Development ........................................................... 13
      2. 4. 1. 2. Stretch Reflexes .................................................................................................... 14
      2. 4. 1. 3. Storage and Reutilisation of Elastic Energy ..................................................... 15
      2. 4. 2. Stretch-Shortening Cycle Fatigue ............................................................................... 17
      2. 4. 2. 1. Models for Investigating SSC Fatigue ................................................................... 19
<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. 4. 2. 2. SSC Fatigue and Force Production</td>
<td>19</td>
</tr>
<tr>
<td>2. 4. 2. 3. SSC Fatigue and Ground Contact Time</td>
<td>20</td>
</tr>
<tr>
<td>2. 4. 2. 4. SSC Fatigue and Leg Spring Stiffness</td>
<td>21</td>
</tr>
<tr>
<td>2. 4. 2. 5. Recovery from SSC Fatigue</td>
<td>22</td>
</tr>
<tr>
<td>2. 5. Post Activation Potentiation</td>
<td>23</td>
</tr>
<tr>
<td>2. 5. 1. Mechanisms for Post Activation Potentiation</td>
<td>25</td>
</tr>
<tr>
<td>2. 5. 1. 1. Phosphorylation</td>
<td>25</td>
</tr>
<tr>
<td>2. 5. 1. 2. Neural Factors</td>
<td>26</td>
</tr>
<tr>
<td>2. 5. 2. Factors Influencing Post Activation Potentiation</td>
<td>27</td>
</tr>
<tr>
<td>2. 5. 2. 1. Exercise Intensity</td>
<td>27</td>
</tr>
<tr>
<td>2. 5. 2. 2. Recovery Periods</td>
<td>28</td>
</tr>
<tr>
<td>2. 5. 2. 3. Muscle Fibre Composition</td>
<td>29</td>
</tr>
<tr>
<td>2. 5. 2. 4. Training Status</td>
<td>29</td>
</tr>
<tr>
<td>2. 5. 3. Post Activation Potentiation and Strength Trained Athletes</td>
<td>30</td>
</tr>
<tr>
<td>2. 5. 4. Post Activation Potentiation and Endurance Trained Athletes</td>
<td>31</td>
</tr>
<tr>
<td>2. 5. 5. Post Activation Potentiation and Performance</td>
<td>32</td>
</tr>
<tr>
<td>2. 6. Predictors of Rowing Performance</td>
<td>33</td>
</tr>
<tr>
<td>2. 6. 1. Introduction to the Physiology of Rowers</td>
<td>33</td>
</tr>
<tr>
<td>2. 6. 2. Maximal Aerobic Capacity ($\dot{V}O_{2\text{max}}$)</td>
<td>35</td>
</tr>
<tr>
<td>2. 6. 3. Power at $\dot{V}O_{2\text{max}}$ ($\dot{W}\dot{V}O_{2\text{max}}$)</td>
<td>37</td>
</tr>
<tr>
<td>2. 6. 4. Blood Lactate Indices</td>
<td>38</td>
</tr>
<tr>
<td>2. 6. 5. Max Strength and Max Force</td>
<td>41</td>
</tr>
<tr>
<td>2. 6. 6. Anaerobic Capacity</td>
<td>42</td>
</tr>
<tr>
<td>2. 6. 7. Body Mass</td>
<td>44</td>
</tr>
<tr>
<td>2. 6. 8. Economy</td>
<td>45</td>
</tr>
<tr>
<td>2. 7. Rowing Training</td>
<td>46</td>
</tr>
<tr>
<td>2. 7. 1. Mechanisms Underlying High Intensity Interval Training (HIIT)</td>
<td>48</td>
</tr>
<tr>
<td>2. 7. 2. Prescription of HIIT</td>
<td>51</td>
</tr>
<tr>
<td>2. 7. 2. 1. HIIT Intensity</td>
<td>51</td>
</tr>
<tr>
<td>2. 7. 2. 2. HIIT Duration</td>
<td>53</td>
</tr>
<tr>
<td>2. 7. 2. 3. HIIT Recovery</td>
<td>54</td>
</tr>
<tr>
<td>2. 7. 3. HIIT and Well-Trained Athletes</td>
<td>55</td>
</tr>
<tr>
<td>2. 7. 4. HIIT and $\dot{V}O_{2\text{max}}$</td>
<td>57</td>
</tr>
<tr>
<td>2. 7. 5. HIIT and Lactate Indices</td>
<td>58</td>
</tr>
</tbody>
</table>
Chapter 4. The Effect of Maximal Stretch Shortening Cycle Fatigue on The Biomechanical Properties of Endurance Trained Athletes

4. 1. Abstract
4. 2. Introduction
4. 3. Method
4. 3. 1. Subjects
4. 3. 2. Research Design
4. 3. 3. Instrumentation
4. 3. 4. Test Procedure
4. 3. 5. Data Collection
4. 3. 6. Calculation of the Dependent Variables
4. 3. 7. Statistical Analyses
4. 4. Results
4. 4. 1. Drop Jump Results
4. 4. 2. Rebound Jump Results
4. 5. Discussion
4. 6. Conclusions
4. 7. Summary

Chapter 5. The Physiological Determinants of 2000 m Rowing Ergometer Performance

5. 1. Abstract
5. 2. Introduction
5. 3. Method
5. 3. 1. Subjects
5. 3. 2. Research Design
5. 3. 2. 1. Body Mass and Stature
5. 3. 2. 2. Body Composition
5. 3. 2. 3. 2000 m Time Trial
5. 3. 2. 4. Incremental Step Test
5. 3. 2. 5. Gas Analysis
5. 3. 2. 6. Blood Lactate Analysis
5. 3. 2. 7. Seven Stroke Power Test
5. 3. 3. Statistical Analyses
5. 4. Results
5. 5. Discussion
Chapter 6. The Effect of High Intensity Interval Training on Well-Trained Rowers

6. 1. Abstract ..................................................................................................................................... 134
6. 2. Introduction .............................................................................................................................. 134
6. 3. Method ...................................................................................................................................... 136
6. 3. 1. Subjects ................................................................................................................................ 136
6. 3. 2. Overview of Research Design .............................................................................................. 137
6. 3. 2. 1. Body Mass and Stature ................................................................................................. 138
6. 3. 2. 2. Body Composition .......................................................................................................... 138
6. 3. 2. 3. 2000 m Time Trial .......................................................................................................... 139
6. 3. 2. 4. Incremental Step Test ..................................................................................................... 139
6. 3. 2. 5. Gas Analysis .................................................................................................................... 140
6. 3. 2. 6. Blood Lactate Analysis .................................................................................................... 140
6. 3. 2. 7. Blood Gas Analysis ........................................................................................................ 141
6. 3. 2. 8. Rate of Perceived Exertion ............................................................................................. 141
6. 3. 2. 9. Time to Exhaustion ......................................................................................................... 142
6. 3. 2. 10. Seven Stroke Power Test ............................................................................................. 142
6. 3. 2. 11. Eight Week Training Intervention .................................................................................. 142
6. 3. 2. 11. 1. Long, Slow Distance Group Training ...................................................................... 143
6. 3. 2. 11. 2. High Intensity Interval Training Group .................................................................... 143
6. 3. 3. Statistical Analyses ............................................................................................................... 144
6. 4. Results ....................................................................................................................................... 144
6. 5. Discussion .................................................................................................................................. 149
6. 6. Summary and Conclusion ......................................................................................................... 155

Chapter 7. Thesis Summary, Conclusions and Future Recommendations

7. 1. Introduction ............................................................................................................................... 158
7. 2. Thesis Summary and Main Findings ......................................................................................... 159
7. 3. Thesis Conclusions .................................................................................................................... 163
7. 4. Thesis Limitations ...................................................................................................................... 164
7. 5. Recommendations for Future Research .................................................................................. 165

Bibliography ............................................................................................................................................. 167
Appendices .............................................................................................................................................. 197
List of Figures

Figure 2.1: SSC action of lower limb, adapted from Komi (1992). A is the pre-activation phase, B is the eccentric phase and C is the concentric phase ........................................... 12
Figure 2.2: Events leading to performance deterioration due to SSC fatigue adapted from Komi (2000) and the proposed coupling between SSC exercise induced muscle damage and performance reduction, adapted from Komi and Nicol (2000) ........................... 18
Figure 2.3: A model of the hypothetical relationship between PAP and fatigue (Tillin and Bishop 2009) ............................................................................................................................ 25
Figure 3.1: Specially designed sledge apparatus ........................................................................ 68
Figure 3.2: Camera set up and reflective marker on sledge....................................................... 69
Figure 3.3: Four point affine scaling frame (1.5 m x 1.1 m) used for calibration ................. 69
Figure 3.4: Schematic diagram of test protocol ......................................................................... 70
Figure 3.5: Mean (± 95% CI) GRF difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. **p < 0.01; *p < 0.05 ...................... 77
Figure 3.6: Mean (± 95% CI) height jumped difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. ***p < 0.001 ............................... 77
Figure 3.7: Mean (± 95% CI) CT difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. ***p < 0.001 ........................................... 78
Figure 3.8: Mean (± 95% CI) RSI difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. ***p < 0.001 ........................................... 78
Figure 3.9: Mean (± 95% CI) k_vrt difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. **p < 0.01 ......................................... 79
Figure 3.10: Mean (± 95% CI) GRF difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. **p < 0.01 ......................................... 81
Figure 3.11: Mean (± 95% CI) height jumped difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. **p < 0.01 ......................................... 81
Figure 3.12: Mean (± 95% CI) CT difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. **p < 0.01 ......................................... 82
Figure 3.13: Mean (± 95% CI) RSI difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. **p < 0.01 ......................................... 82
Figure 3.14: Mean (± 95% CI) k_vrt difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. **p < 0.01 ......................................... 83
Figure 4.1: Mean (± 95% CI) GRF difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue .............................................................. 98
Figure 4.2: Mean (± 95% CI) height jumped difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. ***p < 0.001 ........................................... 98
Figure 4.3: Mean (± 95% CI) CT difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue .............................................................. 99
Figure 4.4: Mean (± 95% CI) RSI difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. **p < 0.01 ......................................... 99
Figure 4.5: Mean (± 95% CI) $k_{\text{vent}}$ difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. *p < 0.05 ......................................... 100
Figure 4.6: Mean (± 95% CI) GRF difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. ***p < 0.001; **p < 0.01 .............. 101
Figure 4.7: Mean (± 95% CI) height jumped difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. **p < 0.01 ................................ 102
Figure 4.8: Mean (± 95% CI) CT difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue.............................................................. 102
Figure 4.9: Mean (± 95% CI) RSI difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. **p < 0.01 ......................................... 103
Figure 4.10: Mean (± 95% CI) $k_{\text{vent}}$ difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. *p < 0.05 ......................................... 103
Figure 5.1: Schematic representation of the experimental design ......................................... 117
Figure 6.1: Schematic representation of the experimental design ......................................... 138
Figure 6.2: Mean (± 95% CI) percentage change in measured variables from pre to post testing. ***p < 0.001; **p < 0.01; *p < 0.05 Significant change from pre to post training within groups. † p < 0.05 Significant change from pre to post training between groups. ............................................................................................................................ 146
Figure 6.3: Baseline pH and HCO$_3$ levels pre and post training. ......................................... 148
Figure 6.4: Peak blood lactate, post exercise pH and HCO$_3$ following a 2000 m TT pre and post training. .......................................................................................................................... 148
List of Tables

Table 3.1: Physical characteristics of the participants ................................................................. 67
Table 3.2: Table showing the classification of $\eta_p^2$ as an effect size ........................................ 75
Table 3.3: Performance scores of fatigue workout ....................................................................... 76
Table 3.4: Mean baseline DJ and RBJ values for each dependent variable ................................. 76
Table 3.5: Percentage change for the DJ dependent variables from baseline values to minimum and maximum values achieved during recovery .............................................. 79
Table 3.6: $\eta_p^2$ values for the DJ dependent variables and classification of the magnitude of the effect size according to Hopkins (2002) ................................................................. 80
Table 3.7: Percentage change for the RBJ dependent variables from baseline values to minimum and maximum values achieved during recovery. **$p < 0.01$; *$p < 0.05$ .......... 83
Table 3.8: $\eta_p^2$ values for the RBJ dependent variables and classification of the magnitude of the effect size according to Hopkins (2002) ................................................................. 84
Table 4.1: Physical characteristics of the subjects (n=9) ............................................................... 95
Table 4.2: Performance scores of fatigue workout ....................................................................... 96
Table 4.3: Mean baseline DJ and RBJ values for each dependent variable ................................. 97
Table 4.4: Percentage change for the DJ dependent variables from baseline values to minimum and maximum values achieved during recovery .............................................. 100
Table 4.5: $\eta_p^2$ values for the DJ dependent variables and classification of the magnitude of the effect size according to Hopkins (2002) ................................................................. 101
Table 4.6: Percentage change for the RBJ dependent variables from baseline values to minimum and maximum values achieved during recovery .............................................. 104
Table 4.7: $\eta_p^2$ values for the RBJ dependent variables and classification of the magnitude of the effect size according to Hopkins (2002) ................................................................. 104
Table 5.1: Physical characteristics of the subjects ....................................................................... 116
Table 5.2: Mean values for all variables measured for all subjects and by gender ......................... 122
Table 5.3: Correlation coefficients for determinants of 2000 m rowing ergometer speed ................... 123
Table 5.4: Multiple regression to predict 2000 m rowing velocity and the influence of parameters on 2000 m rowing speed for all subjects ................................................................. 124
Table 5.5: Multiple regression to predict 2000 m rowing speed and the influence of parameters on 2000 m rowing velocity for male subjects ................................................................. 125
Table 5.6: Multiple regression to predict 2000 m rowing velocity and the influence of parameters on 2000 m rowing velocity for female subjects .......................................................... 126
Table 6.1: Physical characteristics of the subjects (n=19) .......................................................... 136
Table 6.2: Mean baseline values for all variables measured ....................................................... 145
Table 6.3: Changes in all measured variable from pre to post testing .................................... 147
List of Equations

Equation 2.1: The Fick equation................................................................................................... 35
Equation 3.1: Calculation of flight time....................................................................................... 73
Equation 3.2: Calculation of height jumped (Harrison and Gaffney 2004, Flanagan and Harrison 2007) ................................................................................................................................ . 73
Equation 3.3: Calculation of leg spring stiffness (McMahon and Cheng 1990) ............... 74
Equation 3.4: Calculation of partial eta-squared................................................................. 75
Equation 3.5: The conversion of $f$-values to $\eta_p^2$.............................................................. 75
Equation 5.1: Equation predicting 2000m rowing ergometer velocity ............................ 124
Equation 5.2: Equation predicting 2000 m rowing ergometer velocity for male rowers ... 125
Equation 5.3: Equation predicting 2000m rowing ergometer velocity for female rowers 126
### List of Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>2000 m TT</td>
<td>2000 m time trial</td>
</tr>
<tr>
<td>95% CI</td>
<td>95% confidence interval</td>
</tr>
<tr>
<td>BF%</td>
<td>Percent body fat</td>
</tr>
<tr>
<td>CT</td>
<td>Contact time</td>
</tr>
<tr>
<td>DJ</td>
<td>Drop jump</td>
</tr>
<tr>
<td>DJ/RBJ set</td>
<td>Set consisting of 1 DJ immediately followed by a RBJ</td>
</tr>
<tr>
<td>ECON</td>
<td>Oxygen cost of movement</td>
</tr>
<tr>
<td>FF%</td>
<td>Percent fat free mass</td>
</tr>
<tr>
<td>$F_{\text{max}}$</td>
<td>Maximal force</td>
</tr>
<tr>
<td>GLM ANOVA</td>
<td>General linear model analysis of variance</td>
</tr>
<tr>
<td>GRF</td>
<td>Ground reaction force</td>
</tr>
<tr>
<td>HCO$_3$</td>
<td>Bicarbonate</td>
</tr>
<tr>
<td>HIIT</td>
<td>High intensity interval training</td>
</tr>
<tr>
<td>k$_{\text{vert}}$</td>
<td>Leg spring stiffness</td>
</tr>
<tr>
<td>Lean%</td>
<td>Percent lean body mass</td>
</tr>
<tr>
<td>LSD</td>
<td>Long slow distance training</td>
</tr>
</tbody>
</table>

xiv
**LT**  Lactate threshold

**Peak La**  Peak lactate

**PPO**  4 minute all-out power output

**RBJ**  Rebound jump

**RSI**  Reactive strength index

**SSC**  Stretch shortening cycle

**$T_{\text{max}}$**  Time to exhaustion at $\dot{V}O_{2\text{max}}$

**$\dot{V}O_{2\text{AT}}$**  Oxygen consumption at anaerobic threshold

**$\dot{V}O_{2LT}$**  Oxygen consumption at lactate threshold

**$\dot{V}O_{2\text{max}}$**  Maximal oxygen uptake

**$W\dot{V}O_{2\text{max}}$**  Power at maximal oxygen uptake

**$W_{2\text{mmol/l}}$**  Power output at blood lactate concentration of 2 mmol.l$^{-1}$

**$W_{4\text{mmol/l}}$**  Power output at blood lactate concentration of 4 mmol.l$^{-1}$

**$W_{\text{AT}}$**  Power output at anaerobic threshold

**$W_{\text{LT}}$**  Power output at lactate threshold

**$W_{\text{max}}$**  Maximal power

**$\eta_p^2$**  Partial eta squared
Glossary of Terms

**Buffering Capacity**
The ability of the tissue to resist pH changes during an acid load.

**Concurrent Training**
The performance of both strength and endurance training during the same phase of a training programme in order to achieve multiple training goals.

**High Intensity Interval Training (HIIT)**
Short to long bouts of high intensity exercise interspersed with recovery periods.

**Leg Spring Stiffness ($k_{vert}$)**
Stiffness is the relationship between the deformation of an object and a given force. Leg spring stiffness represents an integration of the stiffness of all the lower limb musculoskeletal structures and describes those structures’ ability to interact in unison in a spring-like fashion.

**Maximal Oxygen Uptake ($\bar{VO}_{2max}$)**
The maximal ability of an individual to take up, transport and utilise oxygen by the working muscles.

**Post Activation Potentiation (PAP)**
An acute transient improvement in performance as a result of prior muscle activation.

**Reactive Strength Index (RSI)**
The ability to change quickly from an eccentric to concentric contraction.

**Rowing Economy (ECON)**
The volume of oxygen consumed by the working muscles at a given steady-state exercise intensity.

**Stretch Shortening Cycle (SSC)**
A natural type of muscle function in which an eccentric contraction immediately precedes a concentric contraction with minimum delay between the eccentric and concentric phase.
Preface

The work of this thesis was published in the following peer reviewed international conference proceedings:


Chapter 1. Introduction
1.1. Background and Rationale for Research

The principal objective of training is to stress various bodily systems in order to elicit positive adaptations to enhance performance and in accordance with the principles of training specificity, strength and endurance training induce distinctly different muscular adaptations (Tanaka and Swensen 1998). This has led to the classification of athletes into two broad categories, namely strength and endurance, based on distinctly different and opposing phenotypes. Endurance is the ability to perform repeated, continuous skeletal muscle contractions for prolonged periods, at the highest mean power output or velocity (Hawley 2002). Accordingly, traditional training methods for endurance based sports typically involve low resistance, high repetition exercise which is performed continuously for extended periods. The primary goal of endurance training is to improve aerobic capacity through increases in $\dot{V}O_{2\text{max}}$, efficiency, economy, lactate threshold and long-term endurance capacity. These enhancements in key determinants in endurance performance are achieved through a number of adaptations including increases in capillary density, mitochondrial density, intramuscular substrate stores and oxidative enzyme activity, and a reduction in the activity of the glycolytic enzymes with negligible affects on muscular strength and anaerobic power (Tanaka and Swensen 1998, Hickson 1980, Dudley and Djamil 1985). In contrast, strength is the ability to exert force (Brooks et al. 2000, p424). Traditional strength training involves high resistance, low repetition exercise for very brief periods. This type of training paradigm aims to increase muscular strength, to initiate hypertrophy and to improve motor performance (Komi 2003a). These improvements are realised through increased muscle mass, increased cross-sectional area of muscle through increased myofibrils, increased motor unit firing rate, alteration of type II fibre characteristics and a negligible change in aerobic capacity (Tanaka and Swensen 1998).

Due to the distinctly disparate nature of strength and endurance training, it is proposed that training programmes should be designed to maximise the adaptive response most closely related to the performance of the sport and athletes should train with an optimal degree of specificity, ensuring the essential energy system is well trained and that biomechanical factors are considered (Glowacki et al. 2004). Low intensity, long duration training would therefore appear to be effective in stimulating essential physiological adaptations for endurance athletes and well-trained endurance athletes, including runners, cyclists, cross-country skiers and rowers, are reported to perform $\sim$75% of their training at
intensities below the lactate threshold, despite competing at much higher intensities (Esteve-Lanao et al. 2007). To be successful in endurance sports, however, requires more than an enhanced long term work capacity in a physiologically homogenous group of athletes. Successful performance in intense endurance events such as 2000 m rowing or 5000 m running also requires muscular strength and anaerobic power, necessary for attacking, pace changing and final sprints and in a homogenous group of athletes there appears to be an increasing importance on the anaerobic contribution to performance as a result of less variation in aerobic endurance due to increased aerobic training hours observed over the past thirty years (McNeely 2011a). The ability of endurance athletes to effectively utilise the stretch shortening cycle (SSC) is also important for endurance performance (Harrison et al. 2004). The changes in neuromuscular characteristics induced during SSC fatigue may alter the manner in which the SSC is utilised and it has been suggested that endurance performance may be affected by the ability of the neuromuscular system to produce power or contractility factors affected by the interaction of neuromuscular and anaerobic characteristics (Noakes 1988, Paavolainen et al. 1999).

One of the main challenges facing endurance athletes is to enhance aerobic endurance while simultaneously maintaining strength and anaerobic gains. It is therefore apparent that alternative methods of training for endurance athletes already completing high volumes of low-intensity training should be investigated in order to optimise performance. This is especially important for rowers who, in contrast to other traditional endurance sports such as running and cycling have received little comparative attention in research. It is envisaged that the body of work in this thesis will increase the understanding of endurance performance optimisation which will enable scientists, coaches and athletes alike to make more informed decisions about the potential benefits of acute and chronic effects of alternative training methods for well-trained endurance athletes.

1.2. Aim of Research

The primary aim of this thesis is to investigate the impact of alternative methods of training for well-trained endurance athletes to elucidate optimal training prescription in order to maximise performance.
1.3. Objectives of Research

- To investigate the acute effects of maximal SSC fatigue on the biomechanical performance of strength trained athletes.

- To examine the recovery process of strength trained athletes following maximal SSC fatigue.

- To investigate the acute effects of maximal SSC fatigue on the biomechanical performance of endurance trained athletes.

- To examine the recovery process of endurance trained athletes following maximal SSC fatigue.

- To establish the physiological determinants of 2000 m rowing performance in well-trained rowers.

- To determine the relationship between rowing performance and selected physiological measures.

- To compare the effects of traditional endurance training with high intensity interval training in well-trained rowers.

1.4. Research Questions

For the studies completed, the research questions were as follows:

**Study 1.** The effect of maximal stretch shortening cycle fatigue on the biomechanical properties of strength trained athletes.

1. Does maximal SSC fatigue affect the biomechanical performance of strength trained athletes?

2. What is the pattern of recovery for strength trained athletes following maximal SSC fatigue?
Study 2. The effect of maximal stretch shortening cycle fatigue on the biomechanical properties of endurance trained athletes.

1. Does maximal stretch shortening cycle fatigue affect the biomechanical performance of endurance trained athletes?
2. What is the pattern of recovery for endurance trained athletes following maximal SSC fatigue?

Study 3. The physiological determinants of 2000 m rowing ergometer performance.

1. What physiological parameters best predict 2000 m rowing ergometer performance?

Study 4. The effect of high intensity interval training on well-trained rowers.

1. Does high intensity interval training have the same effect as long, slow distance training on the 2000 m rowing ergometer performance and the key physiological parameters of performance of well-trained rowers.

1. 5. Thesis Outline

This thesis is comprised of seven chapters. Following a brief introduction to the research topic and an outline of the primary aim and objectives of the thesis in the current chapter (Chapter 1) a comprehensive review of pertinent literature is presented (Chapter 2). Chapter 3-6 comprises of four original research studies which are presented in a manuscript format and finally a synopsis of thesis findings is presented in addition to recommendations for the direction of future research (Chapter 7).

Chapter 2: presents a review of relevant literature on the stretch shortening cycle (SSC), SSC fatigue, post activation potentiation (PAP), predictors of rowing performance and high intensity interval training. This review identifies the existing research in the area with a particular focus on well-trained endurance athletes.
Chapter 3: investigates the acute effects of maximum SSC fatigue on the biomechanical performance of well-trained strength athletes. The study also investigates if a maximum SSC workout can elicit a post activation potentiation response in a group of well-trained strength trained athletes.

Chapter 4: employs a methodology analogous to Chapter 3 while investigating the acute effects of maximal SSC fatigue on the biomechanical processes involved with the execution of the SSC in well-trained endurance athletes. Similar to Chapter 3, this study examines the recovery processes following maximal SSC fatigue with the aim of establishing if well-trained endurance athletes can elicit a post activation potentiation response. This methodology has not previously been utilised with endurance athletes and affords the opportunity to evaluate the immediate effects of maximal SSC fatigue.

Chapter 5: determines the physiological determinants of 2000 m rowing ergometer performance in well-trained rowers and investigates the key predictors of performance. There is a dearth of research involving well-trained rowers and this information could prove useful when designing effective training interventions.

Chapter 6: monitors and evaluates changes in rowing performance and associated physiological variables in response to traditional rowing training and high intensity interval training while addressing a significant gap in the existing literature regarding the effect of high intensity interval training on well-trained rowers.

Chapter 7: critically appraises the results observed and the research completed throughout this thesis. The practical implications of these findings are explored and recommendations for future research are presented.
Chapter 2. Literature Review
2. 1. Introduction

The aim of this thesis is to investigate the impact of alternative training modalities for well-trained endurance athletes and the purpose of this chapter is to provide a critical review of the pertinent literature relating to the most important concepts of this thesis. A brief introduction to concurrent training, the performance of both strength and endurance training, is provided to establish the effect of strength training on endurance athletes and endurance performance. The stretch shortening cycle (SSC) is the basis of plyometrics, a popular method of resistance training, and as such a detailed review of the SSC and SSC fatigue is presented. In coexistence with fatigue is the potential of potentiation, whereby the time when an athlete may be best prepared to perform is when fatigue has dissipated and potentiation prevails. Therefore the concept of post activation potentiation (PAP) is examined. The physiology of rowers and the key determinants of rowing performance are discussed and finally high intensity interval training, an alternative method of training for well-trained endurance athletes will conclude the review.

2. 2. Background

Athletes are typically divided into two broad categories, namely endurance and strength, based on their distinctly different and opposing phenotypes. However, many athletes require a high degree of both strength and endurance for optimal performance. It is well recognised that the key physiological determinants of prolonged endurance performance are those related to oxygen uptake, including \( \dot{V}O_{2\text{max}} \), oxygen kinetics, exercise economy and lactate threshold (Jones 2006) and therefore training for endurance events has, traditionally, focused largely on improvements in aerobic capacity (Esteve-Lanao et al. 2007). However, it has been suggested that \( \dot{V}O_{2\text{max}} \) and endurance performance may not only be limited by factors related to oxygen uptake but also by muscle power, the ability of the neuromuscular system to produce power during maximal exercise, or contractility factors affected by the interaction of neuromuscular and anaerobic characteristics (Noakes 1988, Green and Patla 1992, Paavolainen et al. 1999).

Noakes (2000) proposed that endurance performance is not only limited by factors related to oxygen uptake but also by factors related to force production capacity and central activation. It has been observed that power and force production may be a major factor
separating endurance athletes (Noakes 1988, Tanaka et al. 1993, Bulbulian et al. 1986). Paavolainen et al. (1999) introduced a hypothetical model relating to the determinants of endurance performance and among the factors influencing endurance performance were neuromuscular factors including neural control, muscle force and elasticity and running mechanics. During endurance events several neuromuscular factors of force production play a vital role including muscle contractility, ability to utilise the potential energy stored in the muscle structures during the pre-stretch, force-time characteristics of the muscles and reactive ability (Paavolainen et al. 1999, Noakes 1988, Noakes 2003, Green and Patla 1992).

Endurance athletes need a proficient level of strength and anaerobic ability upon which they can call when needed throughout a race. A high level of velocity and sustainable power in addition to the ability to change pace are now prerequisites of high level endurance performance placing an emphasis on muscular ability. It has therefore, in recent times become evident that in endurance sports there is an optimal level of strength and power required for optimal performance and endurance athletes may need to utilise strength and anaerobic training in order to maximise their performance. It has in fact been suggested that strength training (Hoff et al. 1999, Hickson et al. 1988, Hickson et al. 1980, Millet et al. 2002) or high intensity interval training (Esfarjani and Laursen 2007, Smith et al. 1999, Billat et al. 2001, Smith et al. 2003) may lead to an improvement in endurance performance.

2.3. Concurrent Training

Concurrent training involves the performance of both strength and endurance training during the same phase of a training programme in order to achieve multiple training goals and ultimately improve performance. To date, investigations into the compatibility of strength and endurance training are contradictory and inconclusive. Several concurrent training studies have reported an interference effect (Bell et al. 2000, Craig et al. 1991, Dudley and Djamil 1985, Sale et al. 1990). In contrast some have reported no interference effect (McCarthy et al. 1995, Mikkola et al. 2007, Hickson et al. 1980, Gravelle and Blessing 2000) whilst other studies have indicated that concurrent training may result in an improvement in performance (Hickson et al. 1988, Marcinik et al. 1991, Chtara et al. 2005).
2. 3. 1. Concurrent Training and Strength Athletes

Benefits associated with concurrent training for strength athletes have been identified and primarily involve the effect of aerobic training on recovery capabilities through increased aerobic response, improved lactate removal and enhanced phosphocreatine regeneration (Tomlin and Wenger 2001). While it therefore may seem logical the strength athletes could benefit from incorporating endurance training into their training program, some researchers have reported that endurance and strength training are not compatible (Bell et al. 2000, Hakkinen et al. 2003, Hickson 1980). Other studies, meanwhile, have reported no interference effect in strength training gains with the addition of endurance training (McCarthy et al. 1995, McCarthy et al. 2002, Hunter et al. 1987, Gravelle and Blessing 2000). However, recent findings have in fact suggested that there may be a large inter-individual variation in strength training adaptations following concurrent training (-12 to 87%) indicating that while some individuals experience strength decrements following concurrent training, others experience substantial gains (Karavirta et al. 2011). Wilson et al. (2012b) propose that overall power is the major variable which is affected by concurrent training and if maximal strength and hypertrophy are the primary goals, then concurrent training may not lead to significant decrements, provided the correct modality of endurance training is utilised.

2. 3. 2. Concurrent Training and Endurance Athletes

Research into the effect of strength training on endurance has also been equivocal. Several studies have reported that endurance adaptation may be compromised by concurrent strength and endurance training (Gravelle and Blessing 2000, Kyrolainen and Komi 1995, Nelson et al. 1990), in contrast, other investigators have demonstrated that strength training does not interfere with the development of maximal oxygen uptake (Hickson et al. 1988, Hickson et al. 1980, Marcinik et al. 1991, McCarthy et al. 1995, Hunter et al. 1987, Sale et al. 1990, Mikkola et al. 2007, Dudley and Djamil 1985, Chtara et al. 2005). However, it has been suggested by Mikkola et al. (2007) that if the training mode, volume and intensity are appropriate and the training is programmed correctly, endurance athletes can avoid any interference effect.
Indeed strength training could potentially lead to improvements in endurance performance and it has been shown that concurrent strength and endurance training can result in an improvement in running and cycling performance without a concomitant change in maximal aerobic capacity (Hickson et al. 1988, Hickson et al. 1980, Marcinik et al. 1991, Spurrs et al. 2003, Paavolainen et al. 1999, Mikkola et al. 2007). Hickson et al. (1980), in one of the first studies examining the effects of strength training on endurance, reported that following strength training there was a 47% increase in endurance time to exhaustion while cycling and 12% while running, with no significant change in \( \dot{V}O_2 \text{max} \) when expressed relative to body mass. Marcinik et al. (1991) also found that strength training improved endurance performance independently of \( \dot{V}O_2 \text{max} \) changes and that the improvement was associated with increases in both leg strength and lactate threshold. However, the participants in both of these studies were untrained. More recently, Mikkola et al. (2007) found that endurance athletes displayed improvements in explosive force associated with increased rapid neural activation in addition to a more economical sports performance following strength training while Osteras et al. (2002) suggested that improved endurance performances may not only be associated with maximal strength increases, but may be associated with increases in power and rate of force development.

Previous research has suggested that concurrent strength and endurance training may result in improvements in neuromuscular characteristics (Hoff et al. 1999, Spurrs et al. 2003, Osteras et al. 2002, Paavolainen et al. 1999, Mikkola et al. 2007, Paavolainen et al. 1991). Paavolainen et al. (1991) proposed that the neuromuscular changes result in improvements in rapid isometric force production. Similarly, Mikkola et al. (2007) reported similar enhanced neuromuscular performance evident by improvements in rapid force production but also reported an increase in rapid neural activation. Paavolainen et al. (1999) suggested that it was the improvements in neuromuscular characteristics that may be converted into improved economy, whereby improved muscle stiffness as a result of strength training enhances work economy as improved muscle stiffness may develop the body’s ability to store and utilise elastic energy, thereby reducing the energy cost of the movement. Millet et al. (2002) also reported an improvement in the economy of movement following concurrent strength and endurance training and determined that the improvement was the result of an improvement in leg spring stiffness regulation.
2. 4. Stretch-Shortening Cycle

In real life situations, due to the influence of gravity, compression and impact forces, exercise seldom involves a pure form of isometric, concentric or eccentric actions (Komi 2000). In natural locomotion and activities such as running, throwing or jumping, active muscles undergo consecutive stretching and shortening actions, called the stretch-shortening cycle (SSC). The SSC is a natural type of muscle function in which an eccentric contraction immediately precedes a concentric contraction with a minimum delay between the eccentric and concentric phase. Komi (2000) indicated that SSC movements involve (a) pre-activation of the lower limb extensor muscles before contact with the ground is made, (b) stretching of the muscles during the active braking or eccentric phase, followed by (c) shortening of the extensor muscles during the concentric phase. These movements are illustrated in figure 2.1. The time period between the eccentric and concentric phases has been referred to as the amortization phase (Potach and Chu 2008) and Roozen and Suprak (2012) suggest it may be the most critical phase in the SSC.

Figure 2.1: SSC action of lower limb, adapted from Komi (1992). A is the pre-activation phase, B is the eccentric phase and C is the concentric phase

The ability of the SSC to enhance human movement has been recognised since the pioneering research of Marey and Demeny (1885) who observed that in two successive jumps, the second was higher than the first because it involved a more intense eccentric muscular action than the first. In the SSC, the eccentric contraction influences the performance of the subsequent concentric phase, so that the final concentric contraction can be more powerful than that resulting from the concentric action alone. The addition of an eccentric contraction prior to a concentric contraction has been found to increase the force, speed and power output of the concentric contraction in addition to increasing take-
off velocity, and consequently, the height of a vertical jump (Komi and Bosco 1978, Van Ingen Schenau et al. 1997). Cavagna et al. (1975) reported that an eccentric stretch prior to concentric muscle action may increase the effectiveness of the SSC by up to 23% while Anderson (1996) reported that the pre-stretch during the eccentric phase is likely to enhance performance in jumping activities by 5-15%. In earlier research Asmussen and Bonde-Peterson (1974) observed an 11.5% increase in height jumped due to the use of the SSC. Meanwhile, Wilson et al. (1991) reported a mean increase of 14.5% in the maximum weight lifted in a bench press when using a SSC movement, as compared to a lift performed without an eccentric phase.

2. 4. 1. SSC Mechanisms

The increased performance benefit associated with muscle contractions that take place during the SSC has been the focus of much research in order to determine the true nature of this enhancement. Several mechanisms have been proposed to explain the greater positive work that can be done through the use of the SSC. Three common mechanisms that have been proposed include: elastic energy (Cavagna et al. 1965, Cavagna et al. 1977, Cavagna 1978, Komi 1984), time available for force development (Asmussen and Sorensen 1971, Bobbert et al. 1996, Van Ingen Schenau et al. 1997) and stretch reflexes (Komi 2000, Komi and Gollhofer 1997, Komi 2003b, Taube et al. 2012b).

2. 4. 1. 1. Time Available for Force Development

The concept that the SSC may increase the time available for maximal or near maximal force development was first introduced by Asmussen and Sorensen (1971). Bobbert et al. (1996) also determined that the performance enhancement in the SSC is probably caused by the increased time the muscle has to become fully activated when there is an initial eccentric contraction. The eccentric phase prior to the concentric contraction permits the development of a high level of active state, with more cross-bridge attachment. As a result, the subsequent force developed and joint moments are greater at the beginning of the concentric phase and more work can be produced in the first part of the concentric phase. Bobbert et al. (1996) concluded that the storage and reutilisation of elastic energy could be eliminated as an explanation for the enhancement in performance observed with the SSC and proposed that the crucial contribution was in fact the high level of active state and
force the muscles built up to prior to the start of shortening therefore producing more work over the first part of the shortening distance. Van Ingen Schenau et al. (1997) also proposed that the time available to develop force may contribute to the enhancement of work during the SSC. The muscle requires time to reach its maximum force, due to time constraints in the excitation and contraction dynamics of muscle, which are dependent on muscle fibre type and series elastic compliance, and the limitations in the rate at which the central nervous system generates signals. During the SSC the extra time required to build up a maximum active state in the muscle is attained before the concentric contraction resulting in a more powerful action than that resulting from a concentric action alone.

2. 4. 1. 2. Stretch Reflexes

The role of stretch reflexes in force potentiation associated with the SSC has been proposed to explain the benefit of the SSC. The stretch reflex is a neurophysiological response to the stimulation of muscle spindles (Cronin et al. 2011) and the efficiency of the SSC is dependent on recoil properties of the muscle-tendon complex, which can be influenced by the central nervous system (Taube et al. 2012a). In addition, stretch reflexes during the eccentric phase of the SSC have been proposed to enhance muscular stiffness thereby increasing the performance during the concentric phase when compared with isolated concentric action (Voigt et al. 1998). The stretch reflexes are suggested to be evoked by the forced lengthening of the muscle at the beginning of the SSC as the central nervous system responds instantaneously to stretches of a relaxed muscle.

It is proposed that the muscle spindle, acting as a mechanoreceptor facilitator, reacts to rapid changes in the muscle’s length to protect the muscle-tendon complex. As eccentric stretching approaches a rate that could potentially damage the muscle-tendon complex, the muscle spindle activates and reflexively stimulates an opposite contraction of the agonist proportionate to the velocity of the change in length and to a smaller degree in relation to the amplitude (Taube et al. 2012a).

The role of stretch reflexes is an emerging theory but it remains controversial. The time constraints involved in the nature of the events that must occur for this mechanism to influence the force exerted by the muscle during the concentric contraction of the SSC has lead to controversy regarding the mechanism. Van Ingen Schenau et al. (1997) have
proposed that these time constraints limit the possibility of stretch reflexes to have any functional meaning and that a SSC faster that 130 ms does not experience a contribution from the stretch reflex.

In contrast, Komi (2000) argues that there is enough evidence to suggest that stretch reflexes play an important role and that they could have plenty of time to be instrumental during the SSC. Furthermore, Komi (2003b) emphasizes that the reflexes contribute to the efficiency of the motor output by making the force output more powerful. More recently Zuur et al. (2010) confirmed stretch-induced reflex activity in the SSC during hopping. The authors changed the time of ground contact by changing the height of the floor surface while subjects were airborne and unaware of the change in floor height and concluded that there was a contribution from stretch reflexes. Several authors have proposed that in SSC movements, stretch reflex contributions are an important factor in the ability to transfer energy from the pre-activated and eccentrically stretched muscle-tendon complex to the concentric push-off phase (Taube et al. 2012b, Gollhofer et al. 1992, Voigt et al. 1998).

2.4.1.3. Storage and Reutilisation of Elastic Energy

Arguably the most popular mechanism to explain the benefit of the SSC is storage and reutilisation of elastic energy. It has been reported that, when in the eccentric phase of the SSC and an active muscle is forcibly stretched, part of the imposed energy may be stored in the series elastic elements as potential energy and can then reappear during a subsequent shortening of the muscle (Cavagna 1978, Komi 1984). The ability of the muscle to use this stored elastic energy is influenced by the rate and magnitude of the stretch, the level of activation and resulting stiffness of the muscle tendon unit prior to the concentric phase, the change in muscle length during the stretch, and the time lag between the completion of the stretch and the initiation of the concentric contraction (Anderson 1996). Zatsiorsky (1995) has also suggested that the level of stored energy is proportional to the applied force and velocity of the stretch while the magnitude of the stretch is a function of muscle and tendon stiffness.

The time interval between the stretch and shortening, the amortization phase, has been identified as an important component of the SSC, because if this time interval or phase is too long, the stored elastic energy can be wasted and the mechanical efficiency of the
concentric phase will decrease. Cavagna et al. (1965) suggested that a short transition from the braking to the push-off phase is a necessity for the efficient use of elastic recoil in human movement. In addition Wilson et al. (1991) reported that stored elastic energy dissipates as an exponential function of delay time with a 0.85 s half-life decay and that to maximise the use of elastic energy in SSC movements, delay periods should be eliminated or at least minimised and the movement should rapidly continue from the eccentric to the concentric phase.

The ability to change quickly from an eccentric to a concentric contraction has been identified as the reactive strength index (RSI) and is derived from height jumped and ground contact time (Young 1995). It is a means of quantifying SSC performance (Flanagan and Harrison 2007) and has been developed as a mechanism to monitor the stress on the musculotendinous complex during plyometric activities (McClomant 2003). It has been demonstrated that there should be no time delay between the eccentric and concentric contractions due to the fact that some of the stored energy will be dissipated Cavagna (1977). The lost energy associated with a longer amortization phase has been attributed to the detachment and reattachment of cross-bridges during the delay such that, following reattachment, the myofibrils are under less stretch (Enoka 1994). Enoka (1994) further suggests that if the magnitude of the lengthening contraction is too great, fewer cross-bridges remain attached following the stretch and therefore less energy is stored. However, provided the cross-bridges remain attached, the greater the velocity of stretch, the greater the storage of elastic energy.

Despite the popularity of the storage and utilisation of elastic energy as a possible explanation for the greater work associated with the SSC, it has been suggested that elastic energy does not explain the enhancement of work during the concentric phase of the SSC (Bobbert et al. 1996, Van Ingen Schenau et al. 1997, Anderson and Pandy 1993, Avis et al. 1986) whereby the stored elastic energy increases the efficiency of doing positive work but not the total amount of positive work that can be produced. Van Ingen Schenau et al. (1997) proposed that dynamics of force development determine the amount of work that can be produced and not the storage and reutilisation of elastic energy. Furthermore Chapman and Sanderson (1990) explain how it has been demonstrated that work enhancement is not dependent on elastic energy through simulations with models that do not possess series elastic components. The authors propose that without the activation of
the contractile component the series elastic component would store no energy and suggest that the storage and release of energy is a consequence of the SSC and not a reason for its use.

While Van Ingen Schenau et al. (1997) propose that elastic energy does not explain the enhancement of work observed with the SSC in discrete movements the authors attenuate the fact that elastic energy may play a role in the conservation of mechanical energy. It is possible that mechanical energy can be conserved in elastic structures during repetitive SSC’s and as a consequence of effective utilisation of the elastic energy it has been suggested that the metabolic demands of muscles may decrease resulting in an increase in mechanical efficiency (Aura and Komi 1986, Kyrolainen et al. 1991). It has been observed that effective utilisation of stored elastic energy reduces energy expenditure with Cavagna et al. (1968) estimating that without the contribution from the SSC, $\dot{V}O_{2max}$ during running could be 30-40% higher. As such the storage and reutilisation of elastic energy in repetitive SSC’s may therefore improve the economy of movement (Van Ingen Schenau et al. 1997) and Noakes (2003) has postulated that runners with poor economy are less effective at utilizing the impact energy produced.

2.4.2. Stretch-Shortening Cycle Fatigue

Fatigue is one factor which contributes to decrements in performance. It has been described as the inability to maintain a required or expected force (Asmussen 1979). Fatigue can occur either proximally (central fatigue) or distally (peripheral fatigue) and is largely dependent on the type of exercise being performed. Traditionally fatigue research has focused on conditions involving pure isometric, concentric and eccentric muscle actions. However, human movement rarely involves pure forms of these muscle actions with the basic human muscle function involving the stretch-shortening cycle (SSC). The SSC, therefore, provides a model to study neuromuscular fatigue and Komi (2000) has suggested that the SSC gives unique possibilities to study normal and fatigued muscle function. Gollhofer et al. (1987a) was one of the first researchers to use the SSC model to study the effect of fatigue on muscle action and found that repeated SSC actions do induce fatigue effects.
Fatiguing SSC exercise has a strong loading effect on skeletal muscle and the fatigue responses of repeated SSC actions are complex. Repeated high-force SSC muscle actions, used in many sporting activities, can lead to acute and long-term impairments in neuromuscular performance. SSC fatigue has been compared with fatigue following eccentric exercise but is more complex due to the number of possibilities for fatigue adjustments. In SSC activities, impact loads may be repeatedly applied resulting in all of the major elements: metabolic, mechanical and neural, being stressed (Komi 2000). Komi (2000) also suggests that SSC fatigue has considerable impact on muscle mechanics, joint and muscle stiffness and reflex intervention while Horita et al. (1996) reported that exhaustive SSC exercise induces local muscle impairment, which results in modulation of stretch reflexes and stiffness interaction as well as compensation by the central motor command. Komi (2000) proposed a coupling between SSC exercise induced muscle damage and performance reduction whereby interactions between muscle damage, reduced stretch-reflex sensitivity and reduced stiffness regulation lead to a decrease in efficiency of the SSC behaviour and consequently muscle performance following SSC fatigue. Figure 2.2 illustrates the proposal by Komi (2000) of the events leading to SSC performance deterioration.

Figure 2.2: Events leading to performance deterioration due to SSC fatigue adapted from Komi (2000) and the proposed coupling between SSC exercise induced muscle damage and performance reduction, adapted from Komi and Nicol (2000).
2. 4. 2. 1. Models for Investigating SSC Fatigue

Both short and long duration exercise can induce SSC fatigue which can in turn induce neuromuscular fatigue and potentially lead to deterioration in performance. There are two main models used to study exhaustive SSC exercise. A special sledge ergometer, originally designed by Gollhofer et al. (1987b) on which repeated SSC activities are performed, has been extensively used to investigate short-duration SSC fatigue (Gollhofer et al. 1987a, Gollhofer et al. 1987b, Horita et al. 1996, Horita et al. 1999, Horita et al. 2003, Kuitunen et al. 2004, Comyns et al. 2011, Harrison et al. 2004). Meanwhile, events such as marathon running and cross-country skiing have been used as models to study long-duration SSC fatigue (Avela and Komi 1998, Nicol et al. 1991a, Nicol et al. 1991b). Both SSC fatigue models, the sledge apparatus investigating short-duration fatigue and marathon running investigating long-duration fatigue, have given similar results indicating that SSC fatigue is characterised by changes in mechanical performance and changes in performance outcomes. Immediate changes in mechanical performance have clearly revealed a loss of tolerance to imposed stretch loads during the SSC in addition to changes in ground reaction force, contact time and leg spring stiffness (Nicol et al. 1991a, Avela et al. 1999, Comyns et al. 2011, Gollhofer et al. 1987a, Avela and Komi 1998, Horita et al. 2003, Harrison and Gaffney 2004).

2. 4. 2. 2. SSC Fatigue and Force Production

Performance in several sports is determined by the ability to generate force and the speed of force production. Similarly prolonged endurance activities, which undoubtedly require specialized metabolic capacity, often involve a series of brief, explosive spikes in power output. The SSC contributes to effective development of concentric muscle force in many sporting activities. However, the force potentiation associated with the SSC has been shown to significantly diminish following fatiguing SSC exercise. SSC fatigue can cause considerable decreases in force production and ground reaction forces and according to Nicol et al. (1991a), SSC fatigue is likely to affect force production in both isometric and dynamic type performances. Numerous fatigue studies have shown that SSC fatigue can lead to reductions in maximal activation of the muscle and also the force generated in maximal voluntary contractions (Nicol et al. 1991a, Avela et al. 1999, Comyns et al. 2011). In fact, most SSC fatigue studies have observed an immediate drop of $22 \pm 11\%$ in the
force generated in maximal voluntary contractions following SSC fatigue (Nicol et al. 2006).

Declines in maximum voluntary force have been considered one of the most important signs of fatigue (Bigland-Ritchie and Woods 1984) and in addition to decreases in force production following SSC fatigue, detailed graphic analyses have shown that the force-time curve is also influenced by fatigue. In several studies post fatigue graphic analysis revealed a progressive increase in impact spike force followed by a coincidental decrease in post impact force-spike which became more pronounced (Gollhofer et al. 1987a, Nicol et al. 1991a, Nicol et al. 1991b, Komi et al. 1986). Komi (2000) suggested that the drop in force after impact is probably an indicator of a reduction in tolerance to repeated stretch loads as fatigue progresses, and that in order to maintain SSC performance, greater work must be performed which may lead to an even faster progression of fatigue.

2. 4. 2. 3. SSC Fatigue and Ground Contact Time

In the SSC, the time interval between the stretch and shortening of the muscle has been identified as the amortization phase. This phase corresponds to ground contact time (CT) and has been identified as an important component of the SSC. Komi and Gollhofer (1997) identified an immediate transition between the eccentric and concentric phases as one of the three fundamental conditions of effective SSC function. Schmidtbleicher (1992) also reported that a short amortization phase is required for the subsequent concentric contraction to elicit the advantages of both the stored elastic energy and stretch reflex. If this time interval or phase is too long, the stored elastic energy can be wasted and the mechanical efficiency of the concentric phase will decrease.

During and after SSC exercise, fatigue has been characterised by progressive increases in CT (Gollhofer et al. 1987a, Nicol et al. 1991a, Avela and Komi 1998, Horita et al. 2003, Comyns et al. 2011). Such results may indicate a loss in the effectiveness of the SSC as the effectiveness of the SSC is somewhat influenced by the time lag between the completion of the stretch and the initiation of the concentric contraction. A decrease in the magnitude of the SSC potentiation has been observed as the transition time between the eccentric and concentric contraction increases (Wilson et al. 1991). Comyns et al. (2011) reported a significant increase in CT immediately following a maximum SSC fatigue protocol for both
the drop jump (p < 0.001, 29.5%) and the rebound jump (p = 0.007, 12.9%) and concluded that for an effective SSC function minimising ground CT is imperative.

2.4.2.4. SSC Fatigue and Leg Spring Stiffness

Leg spring stiffness represents an integration of the stiffness of all the lower limb musculoskeletal structures during locomotion and describes those structures’ ability to interact in unison in a spring-like fashion (Dalleau et al. 1998, Ferris and Farley 1997). The leg spring stiffness model is highly representative of the mechanics of running gait (Farley et al. 1993, Ferris and Farley 1997) and Farley & Morgenroth (1999) describe how leg stiffness influences the mechanics and kinematics of the body’s interaction with the ground whereby a greater leg stiffness leads to a shorter ground contact time and a smaller vertical displacement of centre of mass during the ground contact phase. Increased leg spring stiffness has been correlated with faster stride frequencies (Farley and Gonzalez 1996) and running velocity (Arampatzis et al. 1999, Bret et al. 2002) and an inverse relationship between stiffness and the metabolic cost of running has been established (Dalleau et al. 1998, Heise and Martin 1998).

The control of stiffness plays an important role in exploiting the full benefits of the SSC and the relative stiffness of the musculotendinous system may determine the body’s ability to store and utilise elastic energy (Wilson et al. 1991). A significant relationship between leg spring stiffness and the active work done during SSC activity has been identified (Liu et al. 2006) and increased utilisation of the SSC with increases in leg spring stiffness has been observed (Harrison and Gaffney 2004). An association between reduced stretch reflex sensitivity and decreased stiffness following highly fatiguing SSC exercise has also been reported (Avela and Komi 1998) and several studies have supported the view that muscle damage, as apparent after SSC fatigue, alters stiffness regulation through a change in the afferent input from the muscle (Millet et al. 2003, Girard et al. 2008, Avela et al. 1999).

A proper adjustment in stiffness allows for the absorption of high impact forces and the storage of elastic energy during the active braking phase (Kuitunen et al. 2004). Leg spring stiffness is an important component of mechanical efficiency and decreases in performance may result partly from alterations in stiffness regulation. Therefore, leg spring stiffness must be well regulated in order to meet external loading conditions during SSC exercise.
and repeated SSC’s, both sub-maximal and maximal have been reported to alter leg spring stiffness (Nicol et al. 1991a, Harrison and Gaffney 2004, Comyns et al. 2011).

With the progression of fatigue during SSC fatiguing exercise, a change in the stiffness characteristics have been reported and, hence, a reduction in the SSC performance (Komi et al. 1986, Comyns et al. 2011). Avela and Komi (1998) have proposed that decreases in performance following SSC fatigue may result somewhat from alterations in stiffness regulation and impaired ability to utilise stiffness-related elastic energy. Repeated SSC’s may eventually become so fatiguing that the neuromuscular system may change its stiffness regulation. During the initial stages of fatigue, leg spring stiffness has been reported to increase in an attempt to utilise elastic energy (Harrison and Gaffney 2004). However, several SSC fatigue studies have reported a weakened SSC behaviour after fatigue due to impaired utilisation of elastic energy and reduced stiffness (Avela and Komi 1998, Horita et al. 1996, Horita et al. 1999, Nicol et al. 1991a).

2. 4. 2. 5. Recovery from SSC Fatigue

SSC fatigue can cause considerable changes in neural, mechanical and metabolic parameters (Komi and Nicol 2000). As highlighted in previous sections SSC fatigue can be characterised by a decrement in performance, a decrease in ground reaction force, an increase in CT and alterations in leg spring stiffness. Submaximal SSC fatigue studies to date have indicated that recovery from SSC fatigue occurs in a bimodal fashion, involving a dramatic decline immediately post-fatigue followed by a short-lasting recovery and a subsequent secondary decline, which may peak around the second or third day post fatigue (Horita et al. 1999, Komi 2000). The immediate reduction in performance is suggested to be primarily related to metabolic disturbances while the secondary decline has been associated with the inflammatory process of muscle damage (Faulkner et al. 1993).

In a review of 26 SSC fatigue studies, Nicol et al. (Nicol et al. 2006) reported that, with the exception of two studies, a general immediate drop (22 ± 11%) in maximal voluntary contraction (MVC) was observed. When MVC was re-examined 2 hours post-fatigue, a partial or even complete recovery was frequently observed. In addition, when MVC was re-
examined during subsequent days in 12 studies, a secondary decline 1-2 days post-fatigue was observed in eight of the studies.

More recently Comyns et al. (2011) investigated the recovery process of a maximum SSC fatigue protocol. The authors found that while there was an initial decline in performance immediately post-fatigue, there was also a significant enhancement in some biomechanical variables, namely ground reaction force (p = 0.002, 9.6% in the drop jump; p = 0.031, 9.6% in the rebound jump) leg spring stiffness (p = 0.004, 19.4% in the drop jump; p = 0.078, 12.7% in the rebound jump) and CT (p = 0.011, 9.6% in the drop jump), approximately 300 seconds post-fatigue. Although no performance outcome was reported in the drop jump the jumping process was altered with a shorter, stiffer and more elastic leg spring action being used. The authors concluded that the maximal fatigue workout altered the way the muscle tendon unit behaved resulting in an effective SSC behaviour 300 seconds post fatigue. This is the first study to demonstrate that while maximum SSC fatigue inhibits performance immediately post fatigue, there is also a potentiation effect associated with maximal SSC fatigue.

2.5. Post Activation Potentiation

Post activation potentiation (PAP) is an acute transient improvement in performance as a result of prior muscle activation (Hodgson et al. 2005, Robbins 2005). It is an activity dependent enhancement and it is proposed that the execution of high intensity contractions prior to the performance of an athletic activity can enhance the performance of that activity (Baker 2003, Gullich and Schmidtbleicher 1996, Young et al. 1998). The subsequent activity is typically plyometric in nature with an emphasis on the SSC. Performance enhancements can come in the form of applied performance (e.g. jumping, throwing, squatting), contractile property output (peak force, peak twitch force, rate of force development, contraction time) and/or neural function (neural conduction velocity, motor unit recruitment patterns). Research has shown that athletes can use exercise sequencing to elicit acute, transient neural adaptations, to generate greater power output or generate faster rates of force development (Duthie et al. 2002, Hamada et al. 2000b, Gullich and Schmidtbleicher 1996). If utilised effectively, PAP could be implemented into power-training to enhance the training stimulus or it may prove to be better than conventional warm-up routines at enhancing performance of explosive sports activities.
(Tillin and Bishop 2009). However, it should be noted that research findings concerning the effect of PAP on performance are equivocal. Several studies have reported a successful PAP effect with improvements in performance being observed (Young et al. 1998, Chatzopoulos et al. 2007, Gourgoulis et al. 2003, McBride et al. 2005) while in contrast others have reported no improvement in performance (Gossen and Sale 2000, Jensen and Ebben 2003, Jones and Lees 2003, Scott and Docherty 2004) and in addition a large inter-individual response has also been observed (Till and Cooke 2009).

The magnitude of potentiation is influenced by both the methods used to evoke it and the characteristics of the muscle. The contractile history of skeletal muscle plays an important role in subsequent performance and also influences the mechanical performance of subsequent muscle contractions. Fatiguing muscle contractions impair muscle performance while non-fatiguing muscle contractions, usually at high loads for brief durations, may enhance performance (Stone et al. 2008). Fatigue is the most apparent effect of contractile history, evidenced by a depression in performance or loss of ability to generate force. However, in coexistence with fatigue is potentiation and the time when an athlete is best prepared to perform is when fatigue has dissipated and potentiation appears, indicating that an optimal recovery time is required to diminish fatigue and realize PAP.

Rassier et al. (2000) demonstrated that fatigue and potentiation could exist simultaneously. However, Tillin and Bishop (2009) have explained that the PAP-fatigue relationship and its effects on subsequent performance are complex. Contractile activity produces both fatigue and PAP and the balance between these two factors determines whether the subsequent contractile response is enhanced, diminished or unchanged. Figure 2.3 illustrates the PAP-fatigue relationship. When the prior muscle activation volume is low, PAP is more dominant than fatigue and a potentiation in subsequent performance can be realised immediately (window 1). As the prior muscle activation volume increases fatigue becomes dominant, having a negative effect on subsequent performance. However, while the fatigue after-effect manifests as an immediate decrease in performance, the fatigue dissipates at a faster rate than PAP and therefore as fatigue subsides a potentiation of performance can be realised (window 2) (Chiu et al. 2003).
Figure 2.3: A model of the hypothetical relationship between PAP and fatigue (Tillin and Bishop 2009).

2. 5. 1. Mechanisms for Post Activation Potentiation

2. 5. 1. 1. Phosphorylation

One of the two principal mechanisms proposed to be responsible for PAP is phosphorylation of myosin regulatory light chains (Hodgson et al. 2005, Sale 2004, Sale 2002, Hamada et al. 2000b, Chiu et al. 2003). This biochemical mechanism involves myosin phosphorylation working to increase the myosin cross-bridge binding with the actin filaments. It is catalysed by the enzyme myosin light chain kinase, which is activated when calcium molecules, released from the sarcoplasmic reticulum during muscle contraction, bind to the calcium regulatory protein calmodulin (Hodgson et al. 2005, Tillin and Bishop 2009). Hodgson et al. (2005) suggest that phosphorylation of the myosin regulatory light chain facilitates PAP through alteration of the myosin head structure and subsequently moving it away from its thick filament backbone. Greater activation of the muscle results in an increased and prolonged calcium release and therefore increased phosphorylation. Grange et al. (1995) reported that a five-fold increase in phosphorylation of myosin regulatory light chain, resulted in a potentiation of the rate of force development, maximal force production and peak velocity. The phosphorylation of the myosin light chains may also make the actin-myosin interaction more sensitive to calcium during further consecutive contractions, resulting in an increase in force of each successive contraction. Morana and Perrey (2009) therefore propose that potentiation may counteract the effects of reduced calcium release that can be seen with fatigue. It must be noted however, that while an acute increase in phosphorylation of myosin regulatory light chains and a parallel
potentiation has been reported in animal studies, the significance of phosphorylation of myosin regulatory light chains in human skeletal muscle remains unclear (Tillin and Bishop 2009), and therefore it may be reasonable to suggest that alternative mechanisms may be responsible for PAP (Stuart et al. 1988).

2.5.1.2. Neural Factors

While the performance enhancement associated with PAP has been attributed to mechanisms within the muscle, it has been noted that neural factors cannot be ruled out especially when involving voluntary muscle actions. Docherty et al. (2004) proposed that PAP is possibly the result of interactions between both neural and muscular mechanisms and Gullich and Schmidtbleicher (1996) have suggested that potentiation may result from an increase in neural excitability when they observed an increase in vertical jump height following maximum voluntary contractions.

The central nervous system, in response to initial muscle activation, has been reported to produce increased contractile function with a subsequent increase in muscle twitch force (Alway et al. 1987). If a prior activation could induce an increase in higher order motor neuron recruitment this might theoretically increase fast twitch fibre contribution to muscular contraction, and therefore enhance subsequent performance (Gullich and Schmidtbleicher 1996). This neural mechanism involves the H-reflex, which is an excitation of a spinal reflex elicited by the Group Ia muscle nerves that conduct impulses to the muscles. The H-reflex amplitude is purported to be a function of the number and size of the motor units recruited which may help place a theoretical link between the H-reflex and the corresponding enhancement in force production (Hodgson et al. 2005). It has been proposed that the initial muscle activation that results in potentiation enhances the H-reflex, and subsequently increases the efficiency and rate of the nerve impulses to the muscle (Hodgson et al. 2005). Comyns et al. (2007) suggested that the results of their study investigating the optimal resistive load for complex training in male rugby players also provided evidence to support the theory of increased neural excitability for potentiation. The authors reported a significant reduction in contact time and an improvement in leg stiffness as a result of heavy weightlifting as the prior contractile activity and proposed that these changes could be due to an increase in neuromuscular activation owing to the prior contractile activity. Comyns et al. (2007) further suggested that the increased activation
resulted in the athletes being able to modulate leg spring stiffness and subsequently reducing ground contact time thereby altering the way the muscle tendon unit behaved during jumping.

2.5.2. Factors Influencing Post Activation Potentiation

The elicitation of PAP is dependent on the balance between fatigue and potentiation which can be affected by several factors including exercise intensity (Masiulis et al. 2007, Vandervoort and McComas 1983, Chiu et al. 2003, Parry et al. 2008), rest intervals (Gossen and Sale 2000, Gullich and Schmidtbleicher 1996) and subject characteristics such as muscle fibre composition (Hamada et al. 2000b, Tillin and Bishop 2009) and training status (Gullich and Schmidtbleicher 1996, Hamada et al. 2000b, Young et al. 1998).

2.5.2.1. Exercise Intensity

The intensity of the potentiation activity is an important factor in eliciting potentiation. Masiulis et al. (2007) stated that voluntary contractions at less than 75% maximum voluntary contraction produce little or no potentiation. Comyns et al. (2011) recently credited the PAP effect they observed in their study to the greater stimulus of a maximum fatigue protocol. Vandervoot and McComas (1983) also concluded, when comparing maximal and sub-maximal contractions, that MVC’s, lasting approximately 10 seconds, result in the greatest potentiation effect. Similar findings were reported by Chiu et al. (2003) who reported that repeated sub-maximal contractions had no effect on power output and Parry et al. (2008) who concluded that the absence of potentiation after sub-maximal contractions could be due to the intensity of the stimulus being too low. The basis of this may be Henneman’s size principle whereby fewer motor units are activated during sub-maximal contractions, in particular type II motor units, and type II fibres may have the greatest potential magnitude to elicit potentiation (Gullich and Schmidtbleicher 1996).

Despite these findings, it is worth noting that some authors have reported potentiation effects following sub-maximal protocols (Masiulis et al. 2007, Morana and Perrey 2009). Indeed a meta-analysis conducted by Wilson et al. (2012a) indicated that moderate intensity (ES = 1.06) exercise is ideal for eliciting PAP when compared to very high intensity (ES = 0.31). In addition, Lowery et al. (2012) reported a similar PAP effect in jump performance.
and power following both moderate and high intensity potentiation squat activities. However, the authors did note that, compared to the moderate intensity workload, the high intensity workload prolonged the duration of PAP.

2.5.2.2. Recovery Periods

The amount of time between the potentiation activity and the performance is critical to PAP. If the potentiation activity is too close to the performance, the effectiveness of the activity will be diminished as a result of the prevalence of fatigue. Gossen and Sale (2000) reported that if there is insufficient time between the potentiation activity and the performance, the effectiveness of the potentiation activity might be attenuated as a result of cumulative fatigue. A study by Gullich & Schmidtbleicher (1996) concluded however, that the time course of potentiation varied greatly between individuals and therefore it may be necessary to examine subjects as individuals rather than groups. It should also be considered that the longer the recovery period between the end of the potentiation activity and the beginning of the performance, the greater the recovery from fatigue but also the greater the decay of potentiation and it is likely that the ability to potentiate performance completely dissipates 30 minutes following the potentiation activity (Rixon et al. 2007).

Several researchers have indicated that a 20 minute rest period was the most successful method of minimising fatigue with the least possible decay of potentiation (Hamada et al. 2000b, Young et al. 1998). Chiu et al. (2003) reported similar findings when they observed that the mean force and power as well as peak power were significantly greater at 18.5 minutes post-activation compared to 5 minutes post-activation. More recently Lowrey et al. (2012) observed an increase in vertical jump height and power 4-8 minutes post activation, which is similar to Kilduff et al. (2007) and Jo et al. (2010) who found that PAP was optimised 8-12 minutes and 5-10 minutes respectively in trained athletes. Comyns et al. (2011) also demonstrated a potentiation effect in ground reaction force, contact time, leg spring stiffness and reactive strength index 5 minutes post activation. In a meta-analysis Wilson et al. (2012a) identified moderate rest periods of 7-10 minutes as the optimal rest period to augment power output, however, the authors did concede that the findings changed based on training status with trained athletes peaking in power output at 7-10 minutes and experienced athletes peaking at 3-7 minutes.
2. 5. 2. 3. Muscle Fibre Composition

The magnitude of PAP may be affected by muscle fibre composition. Studies have reported that individuals with a greater percentage and cross-sectional area of type II muscle fibres and a faster twitch time to maximal force can benefit to a greater degree by PAP (Hamada et al. 2000b, Vandervoort and McComas 1983). Hamada et al. (2000b) investigated the correlation between muscle fibre type distribution and potentiation in human knee extensors and found that potentiation was most effective in subjects with a greater percentage of type II muscle fibres. The authors noted that these subjects also elicited a greater fatigue response following the potentiation activity. Tillin and Bishop (2009) explain that although subjects with a higher percentage of type II muscle fibres have the potential to elicit PAP to a greater degree, due to a higher initial anaerobic ATP turnover rate, they are also likely to show greater fatigue due to the greater utilisation of anaerobic energy sources and the production of metabolites associated with fatigue. In addition the neural excitation associated with heavy lifting is reported to be greatest in type II muscle fibres (Hamada et al. 2000b, Sale 2004) and therefore potentiation may provide the most benefit for athletes participating in events which require sudden, brief efforts of activity.

2. 5. 2. 4. Training Status

Training status has also been suggested to affect the manifestation of potentiation (Gullich and Schmidtbleicher 1996, Hamada et al. 2000b, Young et al. 1998). Hamada et al. (2000b) reported how trained athletes, long distance runners and triathletes, showed greater potentiation than recreationally trained and sedentary individuals while Duthie et al. (2002) reported that stronger individuals improved vertical jump performance to a greater degree than weaker individuals, through means of peak force and peak power. Similarly, Chiu et al. (2003) found that when subjects were separated into athletic and recreationally trained groups, performance improved in the athletic group. The authors reported that following the potentiation activity, performance for the athletic group was greater than 100%, while for the recreational group performance was near or below 100% (-1 to -4% decrease). Chiu et al. (2003) suggested that those subjects in the athletic group were training at higher levels of resistance thereby developing a resistance to fatigue and enhancing the ability to realize PAP.
Young et al. (1998) also found that stronger individuals had a greater vertical jump increase following a potentiation activity than weaker subjects. Similarly, Stone et al. (2008) observed potentiation in very well trained strength/power athletes and Tillin and Bishop (2009) have proposed that there may be a power-strength ratio threshold above which subjects do not benefit from PAP. Schneiker et al. (2006) reported a significant negative correlation between the power-strength ratio and potentiation of peak power ($r^2 = 0.065; p < 0.05$), suggesting that subjects less able to effectively convert their strength into power are more likely to benefit from PAP.

2.5.3. Post Activation Potentiation and Strength Trained Athletes

It has been proposed by several researchers that power-trained and strength-trained athletes are better able to evoke PAP than endurance trained athletes (Smith and Fry 2007, Chiu et al. 2003, Paasuke et al. 2007). The histochemical differences between strength/power and endurance athletes have long been documented with a general consensus that endurance athletes typically have a higher percentage of type I muscle fibres while strength/power athletes typically have a higher percentage of type II muscle fibres. Indeed, as discussed earlier, one the most important muscular characteristic found to affect potentiation is muscle fibre type (Hamada et al. 2000b, Vandervoort and McComas 1983).

Type II muscle fibres elicit a greater potentiation effect than type I fibres due to the fact that type II fibres undergo greater myosin light chain phosphorylation which was earlier identified as one of the primary mechanisms behind PAP. Past research has shown a greater potentiation response in athletes engaging in activities which involve more type II muscle fibres, with muscles with the highest amount of type II fibres and shortest contraction times showing the greatest PAP (O'Leary et al. 1997, Hamada et al. 2000b, Comyns et al. 2006, Comyns et al. 2011). Hamada et al. (2003) did note however, that although muscles with a higher percentage of type II fibres exhibited greater potentiation early in recovery from a potentiation activity, they also experienced greater fatigue later in the recovery.
2. 5. 4. Post Activation Potentiation and Endurance Trained Athletes

The results of several studies have contradicted the concept that strength trained athletes experience the greatest PAP and have observed endurance trained athletes eliciting a higher PAP effect (Hamada et al. 2000a, Morana and Perrey 2009, Boullosa and Tuimil 2009). Hamada et al. (2000a) reported that endurance trained athletes have shown an ability to increase maximum shortening velocity of their type I muscle fibres after potentiation interventions. Morana and Perrey (2009) found that their exercise program did in fact elicit an identical PAP effect, early in the exercise, in both power and endurance athletes. In this study the authors used sub-maximal conditions and observed a PAP effect through a 52% increase in peak twitch torque accompanied by a decrease in time to peak torque.

Endurance athletes may develop a resistance to fatigue as an adaptation to repeated prolonged activity. Even at relatively high force production, endurance athletes may be able to offset fatigue and recover quicker due to the nature of their training (Chiu et al. 2003). Morana and Perrey (2009) reported, from the findings in their study, that the enhanced fatigue resistance of endurance athletes allows the PAP effect to prevail longer over the fatigue effect. In addition, endurance athletes possess a high percentage of type I muscle fibres which have greater resistance to fatigue and may assist in causing less decreases in force production. Hamada et al. (2000a) suggested that elite endurance trained athletes showed evidence of PAP through enhanced isometric force due to a greater level of fatigue resistance but the authors suggested that the potentiation enhancement is limited to trained muscle groups and is somewhat proportional to the training status of the individual.

As mentioned earlier, Hamada (2000a) suggested that endurance athletes may be able to increase the maximum shortening velocity of Type I fibres. This increase in maximum shortening velocity has been associated with an increased content of fast myosin light chains in slow-twitch fibres. This adaptation could increase the capacity of myosin light chain phosphorylation, a proposed mechanism of PAP. In search of alternative explanations, Morana and Perrey (2009) proposed that it is possible that changes in firing rates, a control strategy used by endurance athletes during low-frequency fatigue, may have reduced the fatigue effect and allowed for PAP to occur. Sale (2002) also recognised this mechanism and suggested that a decrease in motor unit firing rate could, by reducing the
number of nerve impulses and muscle action potentials per unit time, delay the onset of fatigue and allow for the prevalence of PAP.

2.5.5. Post Activation Potentiation and Performance

Research findings to date are unclear as to whether potentiation enhances human performance. Sale (2002) has suggested that, theoretically, potentiation would increase the rate of force development leading to an increase in acceleration and velocity. In theory, Sale (2002) suggests that there would be a shift in the force velocity curve upward and to the right, potentially leading to improvements in strength and performance. Meanwhile, Comyns et al. (2011) proposed that the potentiation activity may alter the biomechanics of performance changing the way the muscle tendon unit behaves, resulting in a more effective SSC behaviour with a shorter, stiffer and more elastic leg-spring action.

Numerous studies describe and report the potentiation effects of voluntary contractions (Hamada et al. 2000a) and evoked tetanic contractions (Grange et al. 1993). Despite this, studies involving the ability of athletes to enhance performance through PAP are equivocal. Several studies which have failed to observe PAP (Gossen and Sale 2000, Gullich and Schmidtbleicher 1996). In some of these studies it has been proposed that perhaps the duration or volume of the potentiation contractions were not sufficient to stimulate improvements in voluntary performance in all individuals. Gossen and Sale (2000) found no potentiation effects when investigating the impact of potentiation on dynamic knee extension but concluded that the rest period which was utilised was too short to allow fatigue to subside and therefore any possible potentiation benefits would have been minimal. Also after sub-maximal contractions, the absence of potentiation may be due to the fact that the intensity was too low. Generally, fewer motor units are activated, particularly type II muscle fibres, during sub-maximal contractions, and type II fibres have been reported to elicit the most potentiation (Gullich and Schmidtbleicher 1996). In addition to insufficient stimulation intensity and insufficient rest or latency periods, other factors have been suggested to be the responsible for the failure of PAP to be observed. These include insufficient familiarity with the potentiation protocol, lack of genetic ability of the subjects, insufficient training status of the subjects or lack of specificity of the potentiation activity.
PAP may indeed be a physiological phenomenon, but unless it can be optimally used to enhance performance on a consistent basis its potential may never be realised. The concept of potentiation can be incorporated into training programs and the main purpose of using it is to increase both rate and magnitude of force development in order to maximise explosive power for performance. It has been hypothesised that PAP can be used in order to achieve short-term enhancement of power performance or to achieve chronic adaptation through training and thereby improve performance. Numerous researchers have indicated the acute effects of PAP, but it has also been suggested that potentiation may be manipulated to produce chronic adaptation (Duthie et al. 2002, Gullich and Schmidtbleicher 1996, Young et al. 1998). Any training intervention which has the capacity of acutely enhancing power performance has the potential to directly enhance sports performance. Athletes are continually searching for way to improve performance, often to result in very small margins of improvement, and therefore the concept of potentiation to elicit enhancements in performance, either acute or chronic, in the form of complex training, should not be dismissed. However, given the large variation in individual responses, PAP should be considered on an individual basis.

2.6. Predictors of Rowing Performance

2.6.1. Introduction to the Physiology of Rowers

The physiological qualities of rowers have been reported to be amongst some of the highest recorded (Hagerman et al. 1979). Despite this the uncontrollable nature of the environment in which the sport takes place has restricted research being implemented. Rowing ergometer performance is considered a valuable indicator of a rower’s sport specific fitness and as a result of this rowing ergometer testing is regularly used for the assessment of rowing performance, including the selection of rowers for competition (Smith and Spinks 1995), with 2000 m ergometer time trials the most widely used measure of rowing performance (Maestu et al. 2005).

A 2000 m rowing event could be classed as an intense exercise whereby it involves a near maximal energy delivery for a sustained period of time. Laursen (2010) described an intense exercise event as one lasting between 1 and 8 minutes where there is a mix of ATP derived energy from both aerobic and anaerobic energy systems. Rowing performance over a 2000
m race lasts, depending on boat type and weather conditions, ~5.5 – 7 minutes and is dependent on both the aerobic and anaerobic energy pathways (Steinacker 1993). During competition rowers stress their aerobic and anaerobic capacities to the maximum.

In terms of the physiological demands required to row 2000 m, several researchers have reported that 70-75% of the energy yield necessary to row 2000 m is derived from the aerobic energy system, with the remaining 25-30% being achieved through anaerobic metabolism (Hagerman 1984, Secher 1990, Hagerman et al. 1978). Hagerman (1984) suggested that anaerobic metabolism is largely responsible for the provision of energy during the initial spurt of a race when stroke rates of 40-50 strokes per minute are executed. Rowers then rely on a highly developed aerobic capacity to meet the necessary energy requirements for the next four to six minutes. During the final 30-60 seconds of the race rowers begin a sprint to the finish with the contribution of aerobic metabolism decreasing and the anaerobic pathways supplying a high proportion of energy requirements. It is interesting to note however, that although the same race strategy described by Hagerman (1984) is used by rowers today, more recent studies have suggested a higher aerobic energy contribution. Pripstein et al. (1999) estimated that 88% of energy contribution came from aerobic metabolism while Russel et al. (1998) reported an aerobic contribution of 84%. More recently, de Campos Mello et al. (2009) reported an 84% contribution from aerobic metabolism. McNeely (2011a) has proposed that the observed increase in aerobic contribution is the result of rowers possessing a higher level of aerobic fitness and achieving higher power outputs at $\dot{V}O_{2\text{max}}$ both of which allow a rower to race without having to rely as much on the anaerobic system.

Rowing is one of the most physically demanding of all sports, with almost all muscle groups contributing to the development of propulsive force in each stroke. It is a strength-endurance sport which demands a high degree of both muscular strength, for the acceleration of the boat, and endurance to maintain boat velocity. Research into the physiological predictors of rowing performance of elite and non-elite rowers has shown that greater absolute $\dot{V}O_{2\text{max}}$ ($l\text{min}^{-1}$); power at $\dot{V}O_{2\text{max}}$; peak power output; $\dot{V}O_{2}$ at lactate threshold; percentage of slow twitch fibres; body mass and power output at a blood lactate concentration of 4 mmoll$^{-1}$ are all related to performance (Ingham et al. 2002, Nevill et al. 2011, Secher et al. 1983, Kramer et al. 1994, Cosgrove et al. 1999, Womack et al. 1996). It has therefore been proposed that in order to successfully identify the key factors
influencing rowing performance over 2000 m, that performance determinant models
should consider a contribution from a low-intensity metabolic threshold, a maximal and/or
functional aerobic capacity, and an indicator of anaerobic/maximal power capability (Nevill
et al. 2011).

2. 6. 2. Maximal Aerobic Capacity ($\dot{V}O_{2\text{max}}$)

$\dot{V}O_{2\text{max}}$ is the maximal ability of an individual to take up, transport and utilise oxygen by the
working muscle (Åstrand 2003). It is the product of cardiac output ($Q$), which is the
product of heart rate and stroke volume, and arteriovenous oxygen difference (a-vO$_2$), and
can be calculated by the Fick equation:

$$\dot{V}O_{2\text{max}} = Q_{\text{max}} \times (\text{a-vO}_2)_{\text{max}}$$

Equation 2.1: The Fick equation

Traditionally, $\dot{V}O_{2\text{max}}$ was regarded by many researchers and coaches as the best single
measure of cardiorespiratory endurance and endurance performance. It has been used
extensively as an objective measure of physical work capacity and has long been believed to
be the “gold standard” measurement of performance (Costill 1976, Wilmore and Costill
1994, Saltin and Astrand 1967). Like many other endurance sports, $\dot{V}O_{2\text{max}}$ has commonly
been used for the assessment and prediction of rowing performance (Nevill et al. 1992,
Secher et al. 1982, Secher et al. 1983). Several studies have defined $\dot{V}O_{2\text{max}}$ and the
maximum aerobic capacity as the best predictors of 2000 m rowing performance,
accounting for 49-81% of performance variance (Cosgrove et al. 1999, Ingham et al. 2002,
Kramer et al. 1994) and according to Secher et al. (1982) the correlation between rowing
performance and $\dot{V}O_{2\text{max}}$ is $r = 0.87$. Jurmiiae et al. (2002) and Ingham et al. (2002) reported
similar correlations of $r = 0.85$ and $r = 0.88$ respectively.

According to the scientific literature, elite rowers have produced some of the highest
absolute $\dot{V}O_{2\text{max}}$ values ever recorded with several researchers reporting values of over 6
l/min$^{-1}$ in male elite rowers (Lacour et al. 2009, Hagerman 1984, Di Prampero et al. 1971,
over 7 l min⁻¹ in two elite male rowers and over 5 l min⁻¹ in three elite female rowers (Hagerman, 1984). These absolute $\dot{V}O_{2\text{max}}$ values for rowers are remarkable, however, when $\dot{V}O_{2\text{max}}$ is scaled relative to the body mass (ml kg⁻¹ min⁻¹), the values are less impressive and hence an issue as to which unit of $\dot{V}O_{2\text{max}}$ best predicts rowing performance has been brought into question. The large absolute $\dot{V}O_{2\text{max}}$ values of rowers is mainly due to large body dimensions and muscle mass and have been shown to correlate well with rowing performance, but when this value is expressed in relative terms the correlation has been shown to decrease and smaller, less successful rowers show similar or slightly larger values (Secher et al. 1982, Jurimae et al. 2002). Secher et al. (1982) found a strong relationship between $\dot{V}O_{2\text{max}}$ (l min⁻¹) and placing in an international regatta (r = 0.87) but when $\dot{V}O_{2\text{max}}$ was expressed relative to body mass no significant relationship was found (r = 0.38). In contrast Klusiewicz et al. (1991) reported that $\dot{V}O_{2\text{max}}$ was only significantly related to rowing performance (r = 0.70) when expressed relative to body mass, however this may reflect the heterogeneous nature of the group in terms of training experience and also their smaller body mass.

A number of studies support the argument that absolute $\dot{V}O_{2\text{max}}$ results may be more important than relative values in the assessment of a rower’s maximal aerobic capacity since bodyweight is supported in the boat (Secher et al. 1982, Hagerman 1984, Secher et al. 1983, Nevill et al. 1992). Absolute $\dot{V}O_{2\text{max}}$ has been found to be the best single predictor of rowing performance (Kramer et al. 1994, Warrington et al. 2003, Jurimae et al. 2000). However, it has been noted that $\dot{V}O_{2\text{max}}$ does not explain all of the variance in rowing performance and that by incorporating other variables, such as muscle mass (Jurimae et al. 2002), peak blood lactate (Warrington et al. 2003), body composition (Purge et al. 2004) or velocity at a blood lactate of 4 mmolL⁻¹ (Cosgrove et al. 1999), with $\dot{V}O_{2\text{max}}$ and placing them in regression equations increases the amount of variance that can be accounted for. Nevill et al. (2011) have explained that the selection of both aerobic and anaerobic variables in models to explain ergometer performance are necessary to describe the interplay between the dual aerobic and anaerobic energy pathways during 2000 m performance.

In addition to impressive absolute $\dot{V}O_{2\text{max}}$ values, Hagerman et al. (1978) suggested that the most impressive physiological attribute of rowers appears to be their ability to sustain an
extremely high percentage of their absolute $\dot{V}O_2\max$ despite exceeding their “anaerobic threshold” levels. Elite rowers have a highly developed aerobic capacity which allows them to work at very high percentage of their maximal aerobic capacity. Hagerman et al. (1979) observed elite rowers working at 96-98% of their maximal aerobic capacity during the majority of a race. This high fractional utilisation, which is the ability of rowers to work at a high percentage of their maximal aerobic capacity, is a significant determinant of performance and on-water rowing performance has been associated with the ability to maintain a high percentage of maximal aerobic power and high metabolic efficiency (Jurimae et al. 2000).

2.6.3. Power at $\dot{V}O_2\max$ ($\dot{W}O_2\max$)

The power or velocity at $\dot{V}O_2\max$ has been identified as an index of performance in several endurance disciplines (Hill and Rowell 1997, Noakes et al. 1990, Morgan et al. 1989) with Noakes et al. (1990) reporting that velocity at $\dot{V}O_2\max$ ($v\dot{V}O_2\max$) was the best laboratory measured predictor of distance running performance. Daniels et al. (1985) proposed that $v\dot{V}O_2\max$, which combines $\dot{V}O_2\max$ and economy into one variable, may be important in explaining performance in athletes of mixed ability. However, Noakes (1988) did note that $v\dot{V}O_2\max$ is not only influenced by aerobic power and running economy but also by the muscle power factor related to neuromuscular and anaerobic characteristics.

Maximal aerobic power and the power associated with $\dot{V}O_2\max$, calculated in several studies by solving the regression equation describing $\dot{V}O_2$ and power for incremental intensities of exercise during an incremental step test (Ingham et al. 2002, Nevill et al. 2011), have been identified as important parameters in predicting 2000 m rowing ergometer performance (Nevill et al. 2011, Ingham et al. 2002, Bourdin et al. 2004, Cosgrove et al. 1999, Jurimae et al. 2000). Nevill et al. (2011) reported that the best single predictor of 2000 m ergometer performance is the power at $\dot{V}O_2\max$, explaining 95.3% of the variance in rowing speed. Jurimae et al. (2000) also found that power at $\dot{V}O_2\max$ was the strongest correlate of 2000 m rowing performance in experienced rowers ($r = -0.97, p < 0.05$) while Ingham (2002) reported similar findings with elite rowers ($r = 0.95, p < 0.001$). Jurimae et al. (2000) further reported that the power at $\dot{V}O_2\max$ was significantly related to on-water 2000 m rowing performance ($r = -0.70, p < 0.05$).
Power at $\dot{V}O_{2\text{max}}$ may be a more valuable tool for monitoring performance than $V\dot{O}_{2\text{max}}$ alone in athletes who appear to have reached a plateau in $V\dot{O}_{2\text{max}}$ as it has been demonstrated that the power at $\dot{V}O_{2\text{max}}$ is sensitive to training changes (Ingham et al. 2002). Mikulic (2011) recently support this with the results of a six year follow-up study of a world-class rowing crew. The results illustrated that while $V\dot{O}_{2\text{max}}$ did not change in the final two years of assessment, the power at $\dot{V}O_{2\text{max}}$ over the same period increased by 5% from 459 to 481 W.

### 2.6.4. Blood Lactate Indices

Lactate is the product of anaerobic glycolysis at all rates of muscle metabolism from rest to maximal exercise. Unlike other endurance sports with similar energy demands, rowers quickly achieve very high energy expenditures and therefore induce high lactate levels very early in the race (Hagerman et al. 1978). Blood lactate concentration during standardized exercise tests is an important parameter for the determination of endurance capacity (Hildebrand et al. 2000). The accumulation of blood lactate during incremental exercise tests is a measure commonly used to evaluate the effects of training, to establish individualised training intensities and to predict performance and the way in which blood lactate levels change with increased exercise intensity can provide valuable information about how an athlete is adapting to training (Bourdon 2000).

Weltman (1995) has argued, that due to fact that the blood lactate response to exercise appears to be limited by peripheral adaptations at specific skeletal muscles, the blood lactate response to exercise may be a more sensitive estimate of endurance performance capability than $\dot{V}O_{2\text{max}}$. Other authors have also suggested that the blood lactate response to incremental exercise, and in particular the highest sustainable exercise intensity without a gradual increase in blood lactate level, may predict endurance performance more accurately than $V\dot{O}_{2\text{max}}$ (Coyle et al. 1991, Farrell et al. 1979, LaFontaine et al. 1981). Secher et al. (2007) recently added that blood lactate is a more precise performance predictor because it reflects not only $\dot{V}O_{2\text{max}}$ but also the ability to work without affecting the oxyhemoglobin dissociation curve.

The power or velocity eliciting a blood lactate concentration of 4 mmol\textsuperscript{l}\textsuperscript{-1} has been identified as being closely related to 2000 m rowing performance (Ingham et al. 2002, Womack et al. 1996, Faff et al. 1993, Klusiewicz 1993, Cosgrove et al. 1999) and corresponds to approximately 75-85% of the power at $\dot{V}O_{2\text{max}}$ in successful rowers (Steinacker 1993). Steinacker (1993) reported that the submaximal aerobic capacity measured as the power that elicits a blood lactate level of 4 mmol\textsuperscript{l}\textsuperscript{-1} is the most predictive parameter of competition performance in trained rowers. In a study of junior rowers, Faff et al. (1993) found a strong correlation between mean power output achieved during a 2000 m ergometer time trial and the velocity attained at a blood lactate level of 4 mmol\textsuperscript{l}\textsuperscript{-1} ($r = -0.89$). Womack et al. (1992) and Ingham et al. (2002) reported similar findings. Ingham et al. (2002) found a very strong correlation between the velocity attained at a blood lactate level of 4 mmol\textsuperscript{l}\textsuperscript{-1} and 2000 m ergometer performance ($r = 0.92, p < 0.001$) while Womack et al. (1992) observed the velocity attained at a blood lactate level of 4 mmol\textsuperscript{l}\textsuperscript{-1} to be the best predictor of 2000 m rowing performance ($r = 0.91$).

The $\dot{V}O_{2}$ at a blood lactate concentration of 4 mmol\textsuperscript{l}\textsuperscript{-1} has been shown to have a positive relationship with 2000 m ergometer performance (Cosgrove et al. 1999, Steinacker 1993, Bourdin et al. 2004). Secher et al. (2007) described how rowers with high $\dot{V}O_{2}$ values at the power that elicits a blood lactate concentration of 4 mmol\textsuperscript{l}\textsuperscript{-1} perform better in 6-7 minute maximal tests than rowers with lower values of $\dot{V}O_{2}$. Other parameters of the lactate response to exercise have also demonstrated a strong relationship to rowing performance including power at a blood lactate concentration of 2 mmol\textsuperscript{l}\textsuperscript{-1} (Ingham et al. 2002,
Warrington et al. 2003) and power at a blood lactate concentration of 2.5 mmol\textsuperscript{l-1} (Womack et al. 1996).

The power output attained at lactate threshold has demonstrated a strong relationship with 2000 m rowing performance (Ingham et al. 2002, Nevill et al. 2011). Fixed blood lactate concentrations such as 2.0 mmol\textsuperscript{l-1}, 2.2 mmol\textsuperscript{l-1}, 2.5 mmol\textsuperscript{l-1}, 3.0 mmol\textsuperscript{l-1} and 4.0 mmol\textsuperscript{l-1} have been used to establish lactate threshold (Weltman 1995) with the workload associated with the fixed blood lactate concentration being determined by interpolation from visual plots of workload versus blood lactate. However, the use of fixed blood lactate concentrations in the determination of lactate threshold has been criticised (Coyle 1995, Stegmann et al. 1981). Fixed blood lactate values do not take into account individual kinetics of the blood lactate concentration curve. Marked individual variations in these threshold values have been reported. Stegmann and Kindermann (1982) found in a study of 19 rowers, that 15 failed to endure the work rate associated with a lactate concentration of 4 mmol\textsuperscript{l-1} for a prolonged period. The athletes ceased exercising after 14.4 minutes where mean lactate levels of 9.6 mmol\textsuperscript{l-1} were observed. As a result individual lactate thresholds were established for each athlete with a mean blood lactate value of 2.3 mmol\textsuperscript{l-1} being identified as the new lactate threshold.

Womack et al. (1996) suggested that lactate related variables may be more sensitive indicators of adaptations to training and performance than $\dot{V}O\textsubscript{2}\text{max}$. Prior to training, velocity at the blood lactate concentration of 4 mmol\textsuperscript{l-1} was found to be closely correlated to 2000 m rowing performance ($r = -0.90$), while a weaker relationship was reported between 2000 m rowing performance and $\dot{V}O\textsubscript{2}\text{max}$ ($r = -0.84$). After 12 weeks of training, a higher correlation was observed between the blood lactate concentration of 4 mmol\textsuperscript{l-1} and 2000 m rowing performance ($r = -0.93$) while a weaker relationship was reported between $\dot{V}O\textsubscript{2}\text{max}$ and 2000 m rowing performance ($r = -0.82$). This finding agreed with other studies which reported that, as a consequence of annual planning, the velocity associated with a blood lactate level of 4.0 mmol\textsuperscript{l-1} may increase to higher values in response to seasonal training (Klusiewicz 1993, Secher et al. 1982, Mickelson and Hagerman 1982, Lacour et al. 2009, Messonnier et al. 2005). This work rate increases with training and appears to be dependent on the muscle fibre composition of the rower as Secher et al. (2007) explains that those with many slow twitch fibres are capable of exercising at a high intensity with a blood lactate value of no more than 4 mmol\textsuperscript{l-1}. 

40
2. 6. 5. Max Strength and Max Force

Rowing differs from other endurance sports with similar energy component relationships due to the fact that rowers are required to generate much higher forces for the duration of competition. Despite the source of energy in a 2000 m rowing race being predominantly aerobic, anaerobic parameters such as maximal strength and muscle power are also suggested to have an impact on elite rowing performance (Ingham et al. 2002, de Campos Mello et al. 2009, Secher 1993) as performance depends on the functional capacity of both aerobic and anaerobic pathways.

Strength can be defined as the “ability of a muscle to develop sufficient force to overcome a resistance” while power can be defined as the “ability of a muscle to generate force quickly” (McArthur 1997). Success in rowing involves an element of dynamic strength and the ability to produce a large force during each stroke with rowers required to perform more than 240 strokes throughout a race with a peak force of 1000 to 1500 N per stroke for the first 20 – 30 strokes and 500 – 700 N during the middle of the race (Steinacker 1993). Rowers have been shown to possess exceptional isokinetic leg strength and power when compared to other elite athletes (Hagerman 1984, Steinacker 1993) and Bourgois et al. (2000) have reported that the most successful rowers can be distinguished by skeletal robustness and muscular development.

Trained rowers develop more force and power than most other endurance athletes at relatively low contraction velocities (Steinacker 1993). Unlike other endurance sports, such as cycling and running, rowing involves the use of both legs simultaneously and an adaptive response has been observed in rowers whereby the strength realised when both legs work simultaneously has been demonstrated to be greater than the sum of the strength measured in each of the two legs (Secher et al. 1983). As a result of the leg drive during each stroke being a major source of power, the strength and velocity of contraction of the quadriceps during the forceful extension of the legs is extremely important (Hagerman 1984). Secher (1983) proposed that the presence of very few fast twitch muscle fibres in rowers and the ability of rowers to repeatedly perform muscle contractions is due to the specificity of rowing. Rowers perform a 2000 m rowing race at an average stroke rate of approximately 33 strokes per minute with each stroke having a duration of approximately 0.9 seconds. Secher et al. (1983) concluded that this duration of time allows enough time for force to be
developed in the slow twitch fibres, while the fast twitch fibres only contribute to the first part of each stroke. The reduced muscular performance of rowers at fast contraction velocities may be accounted for by the predominance of slow-twitch fibres.

Short duration rowing power tests have been found to be strongly correlated with 2000 m rowing ergometer performance (Ingham et al. 2002, Riechman et al. 2002). Ingham et al. (2002) reported that maximal force and maximal power produced during a seven stroke power test were the strongest correlates \((r = 0.95, p < 0.001)\) of performance measured. Similarly, Nevill et al. (2011) suggest that maximal power output averaged over the five main strokes of the seven stroke power test would provide an estimate of anaerobic capability. The authors found that when the aerobic component of power at \(\dot{V}O_{2\text{max}}\) was the only variable in the model of determinants of 2000 m performance, the model was inadequate and incapable of explaining differences due to gender or weight class. However, when further aspects, in particular maximal power, were incorporated into the model a more comprehensive model capable of explaining performance of all athletes, irrespective of gender and weight class emerged.

2.6.6. Anaerobic Capacity

Anaerobic capacity has been defined as the maximal amount of ATP that can be supplied to the anaerobic energy system (Medbo 1996) and is an important parameter in competitive rowing (Maestu et al. 2005). Anaerobic contribution to energy production in rowing is typically 20 to 30% in a 2000 m race and is of particular importance during the critical stages of a race, the start and the final sprint, but has been reported to explain only 10-20% of performance (Steinacker 1993). The high anaerobic capacity in elite rowers illustrates how they are capable of exercising at intensities exceptionally close to their \(\dot{V}O_{2\text{max}}\) without experiencing the debilitating effects of metabolic acidosis and Mickelson and Hagerman (1982) concluded that world class rowers generate 72% of their total power output by utilizing 83% of their anaerobic capacity.

Traditionally, the relative contribution of anaerobic metabolism to rowing performance was evaluated by peak blood lactate (Secher 1990) with anaerobic metabolism being indicated by a high peak blood lactate concentration. Mickelson and Hagerman (1982) proposed that the high anaerobic threshold of trained athletes may indicate their increased capacity to
utilise lactate as a fuel during exercise. Hagerman et al. (1979) reported peak blood lactate concentrations on 14-18 mmol\textsuperscript{l}\textsuperscript{-1} in rowers following 6 minutes of maximal ergometer rowing while Secher et al. (2007) explained how values of 11 mmol\textsuperscript{l}\textsuperscript{-1} after treadmill running, 15 mmol\textsuperscript{l}\textsuperscript{-1} after a national regatta and 17 mmol\textsuperscript{l}\textsuperscript{-1} after World championship have been reported for a group of rowers. The elevated blood lactate concentrations (15 – 17 mmol\textsuperscript{l}\textsuperscript{-1}) recorded at the end of international competitions (Vaage 1977) indicate that glycolytic processes are an important factor in the energy supply (Messonnier et al. 1997) and have been observed to be accompanied by changes in the buffering system of the blood with decreases in bicarbonate from 26 to 13 mmol\textsuperscript{l}\textsuperscript{-1} and decreases in pH from a normal value of 7.4 to 7.1 mmol\textsuperscript{l}\textsuperscript{-1} being observed (Secher et al. 2007).

While blood lactate values may indicate the importance of a rower’s anaerobic capacity, they do not indicate the amount of anaerobic metabolism or the overall anaerobic energy contribution to rowing. For this reason the calculation of the oxygen deficit, the difference between the estimated total oxygen requirement and the actual oxygen uptake established during exercise, has been proposed for determining anaerobic capacity (Secher et al. 2007). Of the limited data available, the oxygen deficit of rowers has been reported to be 88-97 ml/kg (Hagerman et al. 1979) which is higher than those recorded for runners (Medbo et al. 1988, Scott et al. 1991) and cyclists (Graham and McLellan 1989) and may reflect the whole body nature of rowing (Koutedakis and Sharp 1985).

McNeely (2011a) identified two main aspects of anaerobic fitness: peak power and mean power. In recent years peak power has begun to emerge as an important predictor of rowing performance. Data collected from Canadian heavyweight male rowers illustrated that while power at $\dot{V}O_{2\text{max}}$ was the best predictor of ergometer performance, peak power on the rowing ergometer was more highly correlated with on-water rowing performance ($r = 0.82$) than $\dot{V}O_{2\text{max}}$ ($r = 0.72$) or ventilatory threshold ($r = 0.70$) (McNeely 2011a). Mean anaerobic power has also been reported to be highly correlated with rowing performance. Riechman et al. (2002) found that mean power from a 30-second ergometer sprint explained 75.5% of the variation in 2000 m rowing performance while $\dot{V}O_{2\text{max}}$ explained only 12.1%.

Several studies have reported that a high anaerobic capacity is of limited value, is not among the most essential factors determining rowing work capacity and, therefore, may not
need to be developed above a certain level (Secher et al. 1983, Klusiewicz 1993, Klusiewicz et al. 1991). This, however, is not the consensus of all researchers in the area. Nevill (2011) proposed that in order to successfully identify the key determinants of 2000 m rowing performance, the best performance determinant models of rowing should include an indicator of anaerobic power capability. In addition McNeely (2011a) suggests due to the fact that in the last 30 years there is a greater emphasis on aerobic capacity, there is less variation in the aerobic capacity between rowers and more variation in the anaerobic capacity resulting in a possible increased importance on anaerobic capacity during a 2000 m race, which as Babraj and Voliantis (2007) explained can help improve an athlete’s chance of winning a specific race.

Babraj and Volianitis (2007) propose than an improvement in anaerobic capacity can occur through two main pathways, an increase in the rate of glycolysis increasing the rate of lactic acid production and/or an improvement in lactate tolerance. In the last 250-300 m of a 2000 m race a change in boat velocity is required for the final sprint. The effectiveness of this sprint will be determined by how fast energy can be released from glycogen also meaning how rapidly lactate can be produced. An improvement in lactate tolerance involves an improvement in buffering capacity which would ultimately enable the athlete to work at a higher intensity before severe acidosis occurs.

2.6.7. Body Mass

Elite rowers are generally tall and muscular and tend to be lean, with very low percentages of body fat (Secher 1990). Ingham et al. (2002) investigated the determinants of 2000 m rowing ergometer performance with 41 World Championship finalists and reported that heavy weight male rowers averaged 190.6 cm (± 6.0) in height, 92.6 kg (± 8.6) in body mass and had 13.5% (± 2.5) body fat while their female counterparts averaged 178.8 cm (± 5.6) in height, 74.5 kg (± 5.5) in body mass and had 22.3% (± 3.1%) body fat. Mikulic (2011) more recently reported values of 188 cm (± 2), 95 kg (± 5) and 9.4% (± 2.8), for height, body mass and percentage body fat respectively, for four heavy weight world champions.

Body size and mass have been identified to be related to rowing performance (Hagerman 1984, Steinacker 1993). As a large proportion of body mass is involved in rowing, it has
been proposed that body size and body mass, at least with heavyweight rowers, are key performance related characteristics. Russell et al. (1998) reported that anthropometric variables (height, body mass and the sum of skinfold measurements) alone predicted 2000 m rowing ergometer performance time best. Similarly, Jurimae et al. (2002) reported that there was a strong correlation between body mass and rowing performance ($r = -0.85$).

Nevill et al. (2010) found that body mass provides a positive contribution to 2000 m rowing ergometer performance ($r = 0.68$, $p < 0.001$) but only a small non-significant contribution to 2000 m on-water single scull performance ($r = 0.039$, $p = 0.79$). Jurimae et al. (2002) reported similar findings when they compared ergometer rowing with on-water rowing. They found that while almost every anthropometric and body composition variable was correlated to 2000 m ergometer performance, only lean muscle mass was correlated to 2000 m on-water single scull performance.

Although a large body mass appears to be important for rowing performance, it is also important to ensure that a high proportion of the body mass consists of lean body mass. Lean body mass has been found to be a predictor of 2000 m rowing performance (Cosgrove et al. 1999, Mikulic 2011) and male heavyweight rowers have been reported to have a low body fat, of between 9 and 10% (Hagerman 1984). Cosgrove et al. (1999) reported that lean body mass had the highest correlation ($r = 0.85$) with mean 2000 m ergometer velocity. Jurimae et al. (2002) had similar findings and concluded that lean body mass presented the highest shared variance with rowing performance ($r^2 = 0.77$). Meanwhile Mikulic (2011) explained that there is a well-established positive relationship between fat free mass and both 2000 m ergometer and on-water performance and that a relatively low percentage of body fat seems to be important for medal winning performance at major competitions. In Mikulic’s (2011) six year case study on world class rowers the author reported a 10% (9kg) increase in body mass and an increase of 15% (11kg) in fat free mass with a concomitant decrease in percentage body fat of 4%.

### 2. 6. 8. Economy

Economy is defined as the volume of oxygen consumed by the working muscles at a given steady-state exercise intensity (Cosgrove et al. 1999). It has been suggested that success in endurance sports is dependent on the economical utilisation of a high aerobic capacity and
the ability to employ a large fraction of that capacity during competition (Costill et al. 1973). Economy has been associated with success in distance running (Di Prampero et al. 1993, Conley and Krahenbuhl 1980, Conley et al. 1984). However, several studies have failed to show any significant relationship between running economy and endurance performance (Williams and Cavanagh 1987, Farrell et al. 1979, Sjodin and Svedenhag 1985) and it has been highlighted that running economy may only be a good predictor of performance in runners of comparable ability (Saunders et al. 2004).

The economy, or oxygen cost of rowing, can be assessed by calculating the mean oxygen uptake per watt (ml/W) of the submaximal stages of the incremental step test (Nevill et al. 2011). However, there is a dearth of information regarding the importance of economy to rowing performance. Cosgrove et al. (1999) speculated that if rowers could reduce the oxygen cost of rowing at a given power, they could probably enhance performance. One longitudinal study conducted by Warrington et al. (1992) found a significant improvement in rowing economy in elite female rowers following 3 months of training. With no concurrent improvement in $\dot{V}O_2\text{max}$, the authors suggested that rowing economy may be a more sensitive measure of change in endurance in elite rowers. More recently however, Nevill et al. (2011) found that economy was not significantly correlated with 2000 m rowing speed ($r = -0.33$).

2. 7. Rowing Training

Similar to other endurance athletes, well-trained rowers perform ~75% of their training at intensities below the lactate threshold, despite competing at much higher intensities (Esteve-Lanao et al. 2007). Due to the nature of the sport of rowing, aerobic training is traditionally the focus of elite rowers. However, Mikulic (2011) suggests that the training of successful rowers should have a focus on aerobic training with a proper relationship of strength and anaerobic training. Due to the fact that elite rowers spend the majority of training time enhancing aerobic endurance one of the main challenges is to simultaneously maintain strength and anaerobic gains.

Endurance training which elicits a blood lactate concentration of 2-4 mmol l$^{-1}$ is the foundation of success in rowing. Steinacker et al. (1998) observed that extensive endurance training (60-120 minute sessions at <2 mmol l$^{-1}$ blood lactate) dominated the training
volume of German, Danish, Dutch and Norwegian elite rowers. In a 31 year study on elite Norwegian rowers from 1970 to 2001 several changes occurred in the training characteristics of elite rowers (Fiskerstrand and Seiler 2004). Extensive endurance training at a low blood lactate (<2 mmolL⁻¹) increased from 30 to 50 hours per month while race pace and supra-maximal intensity training (~8-14 mmolL⁻¹) decreased from 23 to ~7 hours per month in addition to overall training volume increasing by ~20% from 924 to 1128 hours per year.

From the limited number of published observations of elite rowers a polarized training approach is evident, whereby total training volume is comprised of a large volume of extensive endurance training and a small volume of high intensity training (Fiskerstrand and Seiler 2004, Steinacker 1993, Steinacker et al. 1998) and intensity distribution becomes more polarized in the competition period (Guellich et al. 2009). However, despite the fact that maximum aerobic capacity, $\dot{V}_O_{2max}$, is identified as one of the main determinants of performance in rowing (Cosgrove et al. 1999, Ingham et al. 2002, Kramer et al. 1994), $\dot{V}_O_{2max}$ appears to plateau in elite endurance athletes and increases in training volume do not yield any further improvements. For rowers it has been demonstrated that kilometres of training are positively related to successful performance in championships (Jensen et al. 1990, Steinacker 1993) and $\dot{V}_O_{2max}$ is reported to increase with training distance per year but levels off at training volumes of approximately 5000-6000 km per year (Steinacker 1993).

It has therefore become apparent that due to the plateau observed in $\dot{V}_O_{2max}$ in elite endurance athletes, improvements in performance through increased $\dot{V}_O_{2max}$ or the velocity associated with $\dot{V}_O_{2max}$ may only be achieved through high intensity interval training (Laursen and Jenkins 2002). In well-trained athletes, high intensity interval training (HIIT) in addition to an already high training volume appears to be extremely effective (Acevedo and Goldfarb 1989, Weston et al. 1997, Driller et al. 2009, Westgarth-Taylor et al. 1997, Stepto et al. 1999, Lindsay et al. 1996, Smith et al. 1999). Training at a high intensity has the potential to improve rowing power by transferring strength acquired in weight training into explosive high rate stroke power and improvements in maximal intensity have the potential to increase speed particularly at the start of a race (Babraj and Volianitis 2007). McNeely (2011a) suggests that, in a homogenous group of rowers, there may be an increasing importance on the anaerobic contribution to rowing performance as a result of less
variation in aerobic fitness levels and more variable anaerobic fitness levels in elite rowers
due to the decrease in anaerobic training hours observed over the past 30 years.

Despite the fact that, according to the limited research, elite rowers appear to polarize
training and that high intensity interval training may help realise improvements in
performance which increases in training volume cannot, there is a paucity of research
investigating the effect of high intensity interval training on well-trained rowers.

2. 7. 1. Mechanisms Underlying High Intensity Interval Training (HIIT)

The mechanisms responsible for performance changes in well-trained athletes from HIIT
are not clear. Adaptations which have been purported to be responsible for the
improvement in endurance performance associated with HIIT include improvements in
anaerobic capacity (Laursen et al. 2005, Tabata et al. 1996) improvements in ventilatory
thresholds (Acevedo and Goldfarb 1989, Laursen et al. 2002a) and lactate thresholds
(Esfarjani and Laursen 2007, Driller et al. 2009), an increased ability to engage a greater
volume of muscle mass (Creer et al. 2004), greater fat oxidation relative to carbohydrate
oxidation (Westgarth-Taylor et al. 1997) as well as increased buffering capacity (Weston et
al. 1997, Gibala et al. 2006). Other reported physiological adaptations include the ability to
utilise oxygen efficiently, neuromuscular system adaptations and improvements in economy
(Esfarjani and Laursen 2007). It is evident from the research investigating the effect of
HIIT on well-trained athletes that the mechanisms responsible for the improved
performance requires further investigation and it may be that more than one mechanism is
responsible for the improvement.

Recent studies investigating the effects of HIIT have suggested that there are superior
central adaptations to short-term HIIT compared to traditional endurance training at low
intensity (Daussin et al. 2007, Helgerud et al. 2007). Daussin et al. (2007) reported an
improvement in cardiac output through an enhancement of both maximal heart rate and
stroke volume while Helgerud (2007) also reported improvements in stroke volume. Stroke
volume is known to have the potential to increase in two ways, through a higher left
ventricle contractile force and/or through an increase in cardiac filling pressure raising end
diastolic volume and resultant stroke volume through the Frank-Starling mechanism. Such
adaptations would facilitate improved delivery of oxygen to working muscles. However, while Helgerud (2007) noted the change in physically active subjects, Daussin et al. (2007) observed the change in sedentary subjects. Potential changes in stroke volume in well-trained athletes have not been investigated extensively, with Laursen and Jenkins (2002) speculating that even if stroke volume did increase following HIIT in well-trained it may be difficult to detect.

Following HIIT an increase in muscle oxidative potential indicated through changes in maximal activities of enzymes such as citrate synthase, a commonly used marker of oxidative potential, has been reported (Jacobs et al. 1987, MacDougall et al. 1998, Rodas et al. 2000, Burgomaster et al. 2005, Gibala et al. 2006). Rodas et al. (2000) found that a very short, daily high intensity training programme increased aerobic enzyme activity (citrate synthase) in just two weeks. Similarly Burgomaster et al. (2005) and Gibala et al. (2006) reported that over a two week period, all out exercise activity increased skeletal muscle oxidative capacity as observed by the maximal activity and/or protein content of mitochondrial enzymes. It is also possible that HIIT increases the oxidative capacity of type II fibres (Henriksson and Reitman 1976). The results of these studies indicate that improvements in aerobic energy metabolism can be stimulated rapidly by brief bouts of HIIT.

This improvement in aerobic energy metabolism has also been observed in well-trained athletes. Shepley et al. (1992) examined some of the physiological and performance effects of three different tapers in highly trained middle distance runners and showed that a high intensity taper significantly improved citrate synthase activity by 18% suggesting that HIIT may enhance oxidative enzyme activity even in well-trained athletes. However, this change in well-trained athletes is equivocal. Weston et al. (1997) revealed no change in oxidative enzyme activity despite significant improvements in endurance performance in six well trained cyclists following six HIIT sessions over a three week period.

Improvements in performance following HIIT have been attributed to enhanced fat oxidation at the same absolute exercise intensity (Westgarth-Taylor et al. 1997, Essen et al. 1977). HIIT has been demonstrated to be more effective than continuous training for increasing rates of fatty acid oxidation leading to the assumption that the activity of enzymes involved in fatty acid oxidation increase more than those involved in pyruvate
oxidation (Billat 2001a). Essen et al. (1977) suggested that during later intervals in a HIIT session, lactate and citrate may inhibit glycogenolysis leading to an greater reliance on fatty acid oxidation. The authors demonstrated less glycogen was utilised and lipids contributed more to oxidative metabolism with a smaller depletion of glycogen and a larger depletion of intramuscular triglycerides when HIIT was compared to continuous training. Henriksson and Reitman (1976) hypothesized that a repeated stimulation of fatty acid oxidation with HIIT may lead to an up-regulation of this pathway, resulting in a greater stimulation of mitochondrial respiration in the presence of fatty acids.

Anaerobic capacity, the maximum amount of energy available from anaerobic sources determined by maximal accumulated oxygen deficit is purported to be an important contributor to performance in well-trained endurance athletes. Tabata et al. (1996) investigated the effects of 6 weeks of HIIT on anaerobic capacity and reported a 28% improvement. This improvement was smaller than that found by Laursen et al. (2005) who reported improvements in anaerobic capacity of 104% and found that the changes were significantly associated with 40 km time-trial performance. In addition to these findings, Rhodas et al. (2000) found that 2 weeks of HIIT increased the enzymatic activity of anaerobic alactic (creatine kinase) and anaerobic lactic pathways (phosphofructokinase and lactate dehydrogenase).

Hoogeveen (2000) explained that the ventilatory threshold has commonly been accepted as a key indicator in endurance performance and interval training has been found to be more effective in improving the ventilatory threshold than continuous training. The ventilatory threshold has been expected to correspond to the anaerobic threshold (Poole and Gaesser 1985) or to be an indicator of the lactate threshold (Laursen et al. 2005). Smith et al. (2003) proposed that an improvement in the ventilatory threshold would enable athletes to perform at a higher percentage of their $\dot{V}O_{2\text{max}}$. The authors found improvements in 3000 m and 5000 m running performance of 17 and 25 seconds respectively following four weeks of HIIT training and speculated that a 6.8% improvement in the ventilatory threshold was the mechanism explaining the effectiveness of the training. Significant improvements in the ventilatory threshold were also observed by Laursen et al. (2005) in well-trained cyclists following four weeks of HIIT. From these results Laursen and colleagues, using the controversial assumption that the ventilatory threshold is an indicator of the lactate threshold, suggested that the improvements in performance and associated
improvements in the ventilatory threshold were achieved through peripheral skeletal muscle adaptations that enhanced fat oxidation relative to carbohydrate oxidation at the same absolute exercise intensity.

It has been found that well-trained athletes have a higher capacity to transport lactate than less trained subjects, indicating that training can affect lactate/H+ transport (Pilegaard et al. 1994). Pilegaard et al. (1999) concluded from the results of their study that the muscle lactate/H+ transporters can be altered by high intensity training with the rate of sarcolemmal lactate/H+ increasing, playing an important role in the regulation of lactate and pH. This was similar to the assumption made by Jeul et al. (1998), that the capacity for pH regulation via the lactate/proton co-transporter appears to adapt with training. In addition Billat et al. (2001) concluded that in addition to aerobic transport benefits, HIIT stimulates the rate of lactate removal which is dependent directly on its concentration and therefore HIIT which increases blood lactate levels will also stimulate an improvement in lactate removal (Brooks et al. 2000).

2.7.2. Prescription of HIIT

Various high-intensity interval training programmes have been shown to induce improvements in endurance performance and associated physiological variables in well-trained athletes (Driller et al. 2009, Laursen et al. 2005, Laursen and Jenkins 2002, Billat et al. 2002a, Smith et al. 1999, Westgarth-Taylor et al. 1997, Stepto et al. 1999). The extent of these improvements in performance and physiology appears to be dependent upon the duration, the intensity and the frequency of the interval bouts in addition to the type and duration of the recovery period between the interval bouts. However, due to the infinite HIIT options available it is still somewhat unclear how to optimise these factors.

2.7.2.1. HIIT Intensity

The prescription of intensity for intervals is often based on power output, speed or velocity at \( \dot{V}O_{2\text{max}} \). Velocity at \( \dot{V}O_{2\text{max}} \) (v\( \dot{V}O_{2\text{max}} \)) is an index of sustainable aerobic power and was originally defined by Daniels et al (1984) as the minimum velocity needed to reach \( \dot{V}O_{2\text{max}} \) (cited in Billat and Koralsztein 1996). v\( \dot{V}O_{2\text{max}} \) is known to predict performance in middle and long distance runners (Noakes et al. 1990, Morgan et al. 1989) and it has been
suggested that it may be an optimal training stimulus for distance runners (Billat 2001a) when the goal is to increase maximal aerobic power by training for as long as possible at $v\dot{V}O_{2\text{max}}$ or by repeating shorter bouts at $v\dot{V}O_{2\text{max}}$ with minimal fatigue (Hill and Rowell 1996).

The rationale for using $v\dot{V}O_{2\text{max}}$ as the interval intensity in HIIT is based on the assumption that it is the slowest velocity at which $\dot{V}O_{2\text{max}}$ is elicited and that further improvements in $\dot{V}O_{2\text{max}}$ will only result from training at or above $\dot{V}O_{2\text{max}}$ (Hill and Rowell 1997). Billat et al. (2000b) suggested that training at velocities around $v\dot{V}O_{2\text{max}}$ not only has the potential to maximise improvements in $\dot{V}O_{2\text{max}}$, but may also induce significant improvements in mitochondrial density. Meanwhile Smith et al. (2003) explained that the mechanisms responsible for the performance improvement associated with training at $v\dot{V}O_{2\text{max}}$ are believed to include a reduction in oxygen deficit with less anaerobic contribution at the onset of exercise, an improvement in critical power and increases in ventilatory or lactate thresholds.

An exercise intensity of 100% $v\dot{V}O_{2\text{max}}$ has been used in studies which have reported improvements in performance following HIIT (Esfarjani and Laursen 2007, Smith et al. 1999, Billat et al. 2001, Smith et al. 2003). Denadai et al. (2006) concluded that improvements in $v\dot{V}O_{2\text{max}}$, running economy and running performance seem to be dependent on HIIT being completed at 100% $v\dot{V}O_{2\text{max}}$ as opposed to 95% $v\dot{V}O_{2\text{max}}$. Power output at $\dot{V}O_{2\text{max}}$ has also been used in studies involving athletes other than runners (Driller et al. 2009, Laursen et al. 2002b). In a study investigating well-trained rowers, Driller et al. (2009) used intervals of 90% peak power output and concluded that HIIT sessions at or close to the velocity corresponding to $\dot{V}O_{2\text{max}}$ may be the most effective means of eliciting additional improvements in $\dot{V}O_{2\text{max}}$ in already well-trained athletes. Indeed Laursen et al. (2002b), investigating the optimal HIIT program for highly trained cyclists, also found that training at the minimal power output eliciting $\dot{V}O_{2\text{max}}$ produced the most consistent improvements in both endurance performance and $\dot{V}O_{2\text{max}}$.  

52
2.7.2.2. HIIT Duration

The duration of the exercise stimulus plays an important role in the adaptations that occur due to HIIT. The time to exhaustion at $\dot{V}O_{2max}$, $T_{max}$, is a concept closely related to $\dot{V}O_{2max}$ and has been reported to be significant when selecting the duration of HIIT (Smith et al. 2003, Hill and Rowell 1996, Laursen and Jenkins 2002, Billat 2001a). $T_{max}$ is the length of time exercise at $\dot{V}O_{2max}$ can be sustained and studies have shown that increasing $T_{max}$ through HIIT may result in substantial improvements in performance (Billat et al. 2000b, Billat et al. 2001). $T_{max}$ is also said to be related to the lactate threshold and the lactate steady state velocity. Billat (2001a) proposed that the use of $T_{max}$ in interval prescription may allow elite runners to run longer distances at $\dot{V}O_{2max}$ during HIIT and due to the fact the $T_{max}$ is different among runners with the same $\dot{V}O_{2max}$ it may be a rational basis for determining the duration of intervals. Therefore, the use of $\dot{V}O_{2max}$ and $T_{max}$ allows for individualised intensity and duration for HITT sessions.

Hill and Rowell (1996) found that there is no physiological rationale for the prescription of exercise at $\dot{V}O_{2max}$ for durations that are less than 60% of $T_{max}$ and several studies have suggested that the most beneficial exercise duration for HIIT sessions is between 60 and 75% of an athlete’s $T_{max}$ (Smith et al. 1999, Laursen et al. 2002b, Esfarjani and Laursen 2007, Smith et al. 2003). Smith et al. (1999) observed that by utilizing between 60 and 75% of $T_{max}$ as the interval duration for four weeks of HIIT at $\dot{V}O_{2max}$, there was a significant improvement in 3000 m running performance (17.2 s; $P < 0.05$). Smith et al. (2003) more specifically investigated the effects of training at $\dot{V}O_{2max}$ for a duration of 60% $T_{max}$ compared to 70% $T_{max}$. The authors reported a significant improvement of 17 seconds ($P < 0.05$) in 3000 m running performance in subjects in the group training with interval durations of 60% $T_{max}$ compared to a non-significant improvement of 7 seconds ($P < 0.05$) in the subjects using interval durations of 70% $T_{max}$. Additionally, the 60% $T_{max}$ group experienced a 24 second improvement in 5000 m running time compared to just 4 seconds in the 70% $T_{max}$ group, although both of these results were non-significant ($P = 0.12$ and $P = 0.50$ respectively). Smith et al. (2003) speculated that the mechanism responsible for the differences in the groups was the significant improvement in the ventilatory threshold of the 60% $T_{max}$ group (6.8%; $P < 0.05$) compared to a non-significant 1.7% improvement in the 70% $T_{max}$ group.
Esfarjani and Laursen (2007) also compared the effects of two HIIT programmes and found that HIIT prescribed using $\dot{V}O_{2\text{max}}$ and 60% of $T_{\text{max}}$ as the interval intensity and duration improved 3000 m running performance time more that supramaximal sprint interval training, with concomitant increases in $\dot{V}O_{2\text{max}}$, $\dot{V}O_{2\text{max}}$, $T_{\text{max}}$ and the running velocity at lactate threshold. The improvement in 3000 m running performance of 50 seconds (-7.3%; $P < 0.05$) for the subjects in this study was greater than the improvement in the previously mentioned study by Smith et al. (1999) who reported a 17 second (-2.7%; $P < 0.05$) improvement using a similar HIT prescription.

Similar results have been observed by other research groups. Laursen et al. (2002b) conducted a study to investigate interval training program optimization in highly trained endurance cyclists. From the results of this study the authors concluded that the use of $P_{\text{max}}$ ($\dot{V}O_{2\text{max}}$ power output) as the intensity for the intervals and a duration of 60% of $T_{\text{max}}$ elicited the most consistent improvements in already highly trained cyclists.

2. 7. 2. 3. HIIT Recovery

There are two common approaches to recovery duration between HIIT work bouts. These include fixed work recovery ratios (i.e. 2:1, 1:1, 1:2) (Smith et al. 1999, Billat et al. 1999, Stepto et al. 2001) or recovery durations based on heart rate returning to a fixed percentage of its maximum (Driller et al. 2009, Acevedo and Goldfarb 1989, Laursen et al. 2002b). The recovery duration may alter the training stimulus of HIIT. A longer rest period between high intensity intervals allows for a greater removal of $H^+$ prior to subsequent intervals reducing the $H^+$ accumulation during training (Edge et al. 2006b). This is an important factor to consider as a large accumulation of $H^+$ during HIIT bouts has been suggested to be an important stimulus to improve muscle buffer capacity (Weston et al. 1996).

The mode of recovery (i.e. passive or active) and the intensity of work during recovery also require consideration. Billat (2001a) suggests active recovery between intervals may be more beneficial due to its ability to maintain $\dot{V}O_{2\text{max}}$ and stimulate lactate removal. Menzies et al. (2010) reported similar findings when they demonstrated that active recovery after strenuous HIIT leads to faster blood lactate clearance than passive recovery. The authors
also found that the rate of blood lactate clearance was dependent on the intensity of the active recovery, with peak lactate clearance occurring at intensities close to the lactate threshold. This intensity will not only influence the rate of lactate elimination but also the rate of recovery of phosphocreatine and the oxygen kinetics at the start of the subsequent interval (Seiler and Sjursen 2004). Billat et al. (2000a) reported that the intensity during recovery typically ranges from rest to 50% $\dot{V}O_{2\text{max}}$ and studies have shown that the rate of lactate removal increases with increased intensity of active recovery up to approximately 40% of $\dot{V}O_{2\text{max}}$ (Koutedakis and Sharp 1985).

2. 7. 3. HIIT and Well-Trained Athletes

For well-trained athletes, further performance improvements are often difficult to attain. An additional increase in training volume does not appear to further improve endurance performance or associated physiological variables such as $\dot{V}O_{2\text{max}}$, anaerobic threshold, economy or oxidative muscle enzymes (Billat et al. 2001). Consequently, additional HIIT training on top of an already high training volume appears to be extremely effective (Acevedo and Goldfarb 1989, Weston et al. 1997, Driller et al. 2009, Westgarth-Taylor et al. 1997, Stepto et al. 1999, Lindsay et al. 1996, Smith et al. 1999). However, Seiler (2010) warned that an established endurance base built from high volumes of training is an important precondition for tolerating and responding well to an increase in training intensity.

Londeree (1997) reported that once an individual reaches a $\dot{V}O_{2\text{max}}$ of greater the 60 mlkg$^{-1}$min$^{-1}$, endurance performance is not improved by a further increase in submaximal training volume. Billat et al. (2001) also suggested that well-trained athletes can reach a point where their performance no longer improves with traditional long slow distance training. Such athletes would be considered to have a high endurance index described by Perronnet and Thibault (1989) as the ability to use a high fraction of $\dot{V}O_{2\text{max}}$ for a prolonged period of time. It appears that in these athletes, improvements in performance through increased $\dot{V}O_{2\text{max}}$ or the velocity associated with $\dot{V}O_{2\text{max}}$ may only be achieved through HIIT.
Laursen (2010) explained how recent work is revealing that the combination of both traditional endurance training and HIIT may optimize the development of aerobic muscle characteristics and enhance intense exercise performance. In addition, Bulbulian et al. (1986) reported that anaerobic capacity is also an important variable in the performance of well-trained endurance athletes. Tabata et al. (1996) found that aerobic training does not change anaerobic capacity and concluded that in order to improve anaerobic capacity HIIT is necessary. The authors reported a 28% increase in anaerobic capacity following six weeks of HIIT.

In a review article of the scientific basis for HIIT Laursen and Jenkins (2002) summarised that HIIT, but not continuous submaximal training, elicits significant improvements in endurance performance in well-trained athletes and that these improvements are often paralleled with improvements in peak aerobic power output and ventilatory threshold. HIIT has been shown to improve running performance in middle- and long-distance runners. Acevedo and Goldfarb (1989) conducted one of the first studies investigating the effects of HIIT on well-trained distance runners. They found that eight weeks of HIIT improved both 10,000 m running performance and run time to exhaustion although there was no improvement in $\dot{V}O_{2\text{max}}$ leading the authors to conclude that improvements in athletic performance can be independent of increases in $\dot{V}O_{2\text{max}}$. Smith et al. (2003) reported similar findings with a statistically significant ($p < 0.05$) increase in 3000 m running performance following four weeks of HIIT.

The majority of research regarding well-trained athletes and HIIT has focused on distance runners. However, there is research to suggest that well-trained cyclists can also benefit from HIIT (Laursen et al. 2002b, Westgarth-Taylor et al. 1997, Lindsay et al. 1996). Lindsay et al. (1996) investigated the effect of HIIT on well-trained cyclists by replacing approximately 15% of their 300 km/week endurance training with HIIT consisting of six to eight five minute repetitions at 80% of peak power output. The authors found that 4 weeks of HIIT significantly improved 40 km time trial performance ($P < 0.0001$), peak power output ($P < 0.01$) and time to exhaustion at 150% peak power output ($P < 0.01$) and also reported that the improvement in time trial performance was due to significant increases in both the cyclists absolute and relative peak power output. These improvements were similar to those observed by Westgarth-Taylor et al. (1997) who reported significant
improvements in 40 km time trial performance (P < 0.05) and peak power output (P < 0.01) following 12 HIIT sessions completed over a six week period.

While the body of research around HIIT and cycling has expanded, there is a dearth of information regarding HIIT and well-trained rowers. Driller et al. (2009) found that four weeks of HIIT improved 2000 m time trial performance and relative $\dot{V}O_{2\text{max}}$ in well-trained rowers, more than the traditional rowing training approach. The authors highlighted that the ~6 second greater improvement in 2000 m time that the HIIT group achieved could be realised with a 3.5 boat length improvement in a 2000 m single sculling race. The effect of short duration interval training has also been investigated with elite male rowers (Gullstrand 1996). The HIIT sessions included five sets of eight cycles of 15 seconds work at competition intensity and 15 seconds rests. From the investigation Gullstrand (1996) concluded that the HIIT model used demanded relatively high aerobic loading and low glycolytic activity and could be considered an alternative model for training allowing rowers to work for prolonged periods of time at or slightly above competition intensity.

2.7.4. HIIT and $\dot{V}O_{2\text{max}}$

There is a dearth of information regarding the most effective training intensity to elicit an enhancement of $\dot{V}O_{2\text{max}}$ in well-trained athletes. HIIT has been shown to be a very effective means of increasing $\dot{V}O_{2\text{max}}$ in untrained subjects (Laursen and Jenkins 2002, Hickson et al. 1977, MacDougall et al. 1998, Tabata et al. 1996). However, the effect of HIIT on $\dot{V}O_{2\text{max}}$ in well-trained athletes is equivocal. Acevedo and Goldfarb (1989) and Daniels et al. (1978) both reported improvements in performance following HIIT with no increase in $\dot{V}O_{2\text{max}}$ in well-trained participants while other studies have reported an improvement in $\dot{V}O_{2\text{max}}$ following HIIT (Driller et al. 2009, Brooks et al. 2000, Billat et al. 2002a, Laursen et al. 2005, Smith et al. 1999). Two such studies include Billat et al. (2002a) who reported an improvement in $\dot{V}O_{2\text{max}}$ of 5.4% (p < 0.01) in elite marathon runners following eight weeks of HIIT and Driller et al. (2009) who reported a 7% (p < 0.05) increase in $\dot{V}O_{2\text{max}}$ in well-trained rowers following four weeks of HITTT.

In a review investigating if there is an optimal training intensity for enhancing $\dot{V}O_{2\text{max}}$ in well-trained distance runners Midgley et al. (2006) suggested that while the enhancement of
\( \dot{V}O_{2\text{max}} \) is highly dependent on an individual’s initial \( \dot{V}O_{2\text{max}} \) well trained athletes probably need to train at relatively high percentages of their \( \dot{V}O_{2\text{max}} \) to elicit further improvements. Following a review of 59 training studies, Wenger and Bell (1986) made similar conclusions, that the degree of improvement in \( \dot{V}O_{2\text{max}} \) was positively related to training intensities in the range of 50-100% \( \dot{V}O_{2\text{max}} \). Several authors have since corroborated this view and have suggested that well-trained athletes approaching their trainable limit for \( \dot{V}O_{2\text{max}} \) may not only need to train at high percentages of their \( \dot{V}O_{2\text{max}} \) but may even need to attain and maintain \( \dot{V}O_{2\text{max}} \) during training to elicit further increments (Billat 2001b, Laursen and Jenkins 2002, Hill and Rowell 1997).

Billat (2001a) has hypothesised that the benefit of HIIT on \( \dot{V}O_{2\text{max}} \) is dependent on both the time spent at \( \dot{V}O_{2\text{max}} \) during the HIIT sessions and the distance covered at a high velocity. Tabata et al. (1997) proposed that it is conceivable that it is not the exercise intensity per se but the high oxygen uptake that is usually found during HIIT that results in the improved maximal oxygen uptake. The authors further claim that it may be reasonable to assume that the high oxygen uptake obtained during some types of HIIT leads to the significant stress on the aerobic system and results in the large increase in \( \dot{V}O_{2\text{max}} \).

2.7.5. HIIT and Lactate Indices

It has been suggested that the blood lactate response to exercise may be a sensitive estimate of endurance performance capability (Weltman 1995). Poole and Gaesser (1985) speculated that it is possible that training at a very high intensity would delay the accumulation of lactate in the blood to a greater extent than low intensity continuous training by increasing the oxidative capacity of a greater number of muscle fibres. Meanwhile, Brooks et al. (2000) proposed that HIIT affords the ability to tolerate the presence of lactate enhancing lactate removal and allowing better tolerance of high-intensity activity.

A meta-analysis examining the effect of training on lactate and ventilatory thresholds indicated that greater improvements in the lactate threshold are associated with HIIT, but this analysis accounted for mainly sedentary populations (Londeree 1997). Evertsen et al. (2001) investigated the effect of HIIT compared to continuous training on 20 elite cross country skiers and found greater improvements in aerobic fitness through improved lactate
thresholds in the HIIT group while Acevedo and Goldfarb (1989) demonstrated that the performance of well-trained endurance athletes was improved in relation to a decrease in blood lactate at 85 and 90% v\(\dot{V}\)\(\text{O}_{2\text{max}}\) and hypothesised that lactate production and clearance had improved.

2.7.6. HIIT and Buffering Capacity

Buffering capacity has been defined by Jeul (1998) as the ability of the tissue to resist pH changes during an acid load. The removal and regulation of H\(^+\) occurs via a number of different transport systems and via muscle buffering capacity. These mechanisms include ion co-transport and exchange mechanisms and both intra- and extra-cellular buffers (Juel 1998, Bishop et al. 2008). Heisler (2004) proposed that buffering of surplus H\(^+\) ions is a valuable mechanism for minimising transient acid-base disturbances with the intra- and extra-cellular buffers acting to reduce the build-up of H\(^+\) during high intensity exercise therefore aiding in the regulation of intracellular pH.

Buffering capacity has been observed to be higher in anaerobically trained individuals than endurance trained or sedentary subjects indicating that anaerobically trained individuals have an enhanced ability to work under high intensity exercise (Bell and Wenger 1988, Edge et al. 2006c). The ability of muscle to buffer increased concentrations of H\(^+\) is an important factor during intense exercise and therefore those with a greater ability to regulate H\(^+\) during intense exercise may be better able to maintain intense muscle contractions (Edge et al. 2006c). From the results of their study Edge et al. (2006c) suggest that regular high-intensity exercise training performed by team-sport athletes may result in an elevated buffering capacity.

High intensity exercise results in large ionic changes and an increased non-mitochondrial ATP turnover, contributing to the accumulation of H\(^+\) (Bishop et al. 2009). The ability to sustain high intensity exercise is dependent upon the ability to minimise increases in cellular and blood H\(^+\) concentrations. Training at intensities above the lactate threshold causes the production of lactate and H\(^+\) to exceed their removal resulting in an accumulation of H\(^+\) and a decrease in pH, placing stress on the mechanisms involved in pH regulation. This large accumulation of H\(^+\) during exercise has been suggested to be an important stimulus to improve buffering capacity (Weston et al. 1996). These changes in pH are not dependent
on acid production alone but are also influenced by buffering capacity, which determines the degree of acidification with a given acid load.

Bishop et al. (2009) argue that while there are studies which indicate that the role of H⁺ accumulation during fatigue processes may be limited, H⁺ accumulation has been shown to impair exercise performance by affecting oxidative phosphorylation, the perception of effort, enzyme activity and ion regulation during some exercise tasks. The reduction in intracellular pH may also contribute to a decline in power output as reported by Bishop et al. (2003) who found a significant correlation between the change in H⁺ and power decrement (\(r=0.75, P<0.05\)). H⁺ efflux out of the muscle cell has been reported to be inhibited by extracellular acidosis and enhanced by a greater extracellular buffer concentration (Bishop et al. 2003).

The capacity of working muscle to buffer H⁺ ions is related to performance in well-trained individuals (Weston et al. 1997). Laursen and Jenkins (2002) proposed that due to the fact that a high concentration of H⁺ ions has an inhibitory effect on enzyme activity including phosphofructokinase (PFK) activity, that improved buffering capacity may indirectly contribute to an improved glycolytic ATP yield and higher exercise intensity by improving the activity of PFK. Although the authors have admitted that further research investigating this theory is required.

While some studies have reported no change in muscle buffering capacity following a period of HIIT (Harmer et al. 2000, Nevill et al. 1989), according to several studies which have used high intensity intervals, HIIT is responsible for increasing resting muscle buffering capacity (Bishop et al. 2009, Edge et al. 2006a, Edge et al. 2006b, Weston et al. 1997, Gibala et al. 2006). Edge et al. (2006b) hypothesized that a training program that significantly improves muscle buffer capacity has the potential to improve physical performance in a range of sports as buffer capacity has been associated with short and long sprint performance (Bell and Wenger 1988, Sharp et al. 1986, Nevill et al. 1989) and cycling endurance performance (Weston et al. 1997).

A large accumulation of H⁺ during HIIT bouts has been suggested to be an important stimulus to improve muscle buffer capacity (Weston et al. 1996) as has training intensity (Edge et al. 2006a). Edge et al. (2006c) propose that a greater buffering capacity may
improve high intensity exercise performance by allowing anaerobic glycolysis to continue during maximal exercise, resulting in a larger lactate production without an associated increase in H⁺ accumulation, as an improvement in muscle buffer capacity should delay the accumulation of H⁺.

Weston et al. (1997) found a significant increase in buffering capacity in addition to a strong relationship between buffering capacity and 40 km time-trial performance (r=-.82; P<.05) following only 4 weeks of HIIT in well-trained cyclists. These results suggest that the improvements in performance could be related to an increased ability to buffer H⁺. It is believed that the increased buffering potential of the blood enhances the efflux of H⁺ from the contracting muscle into the blood thereby reducing the intracellular accumulation of H⁺, which has been implicated as a possible cause of muscular fatigue (Bishop and Claudius 2005). An increased buffering capacity may therefore improve performance by preventing a large drop in pH.

Edge et al. (2006b) investigated the effects of 5 weeks of HIIT training on females and reported a ~25% increase in buffering capacity while Gibala et al. (2006) also reported an improvement in buffering capacity after four weeks of interval training and speculated that the change may represent a relatively rapid muscle adaptation that contributed to the improvement in exercise capacity which the authors observed. Sharp et al. (1986) also found significant improvements in buffer capacity, with no change in muscle pH despite increased lactate levels, following eight weeks of sprint training suggesting that the buffering capacity can be improved with training.

2. 7. 6. 1. Blood Gas Analysis and Buffering Capacity

Blood buffering capacity can be examined through changes in blood pH, HCO₃⁻ and blood lactate. An increase in post-exercise blood pH in addition to a greater decrease in blood HCO₃⁻, measured by blood gas analysis, and a greater increase in blood lactate concentration indicates that the buffer capacity of the blood has been increased. A significant correlation between blood and muscle pH was reported by Allsop et al. (1990) who found that there was a similar pattern of response between blood and muscle pH at rest and during exercise. McNaughton et al. (2008) also reported that the similar slope and time to critical pH exhibited between muscle and blood pH justifies the continued use of
blood pH kinetics which is more practical in an applied sport setting. Similarly Egde et al. (2002) observed significant correlations (P < 0.05) between post exercise blood and muscle pH (r = 0.64) and blood and muscle in vitro and in vivo buffering capacity (r = 0.57 and r = 0.50 respectively) and concluded that after maximal sprints blood pH and buffering capacity reflected corresponding measures within the muscle.

2. 8. Summary

In order to maximise training adaptations knowledge of the key biomechanical and physiological determinants of performance is required. The importance of the SSC, a key biomechanical variable, to sprinting and jumping is well established (Kubo et al. 2000, Kyrolainen and Komi 1995) and the SSC provides a unique opportunity to investigate the effects of neuromuscular fatigue on performance (Komi 2000). Typically SSC fatigue has been investigated at submaximal intensities from which a bimodal pattern of recovery has emerged. However, SSC fatigue at maximal intensities may in fact elicit a PAP effect (Comyns et al. 2011) although further research is required to substantiate this. It is also apparent that endurance athletes may benefit from the inclusion of SSC-related activities in their training (Harrison et al. 2004). Increased knowledge of the parameters which influence the facilitating effect of acute SSC fatigue on biomechanical performance will enhance the ability to prescribe effective training interventions to induce performance improvements. Therefore this thesis will investigate the acute effects of SSC fatigue, and due to the fact that it is proposed that power-trained and strength-trained athletes are better able to evoke PAP than endurance athletes, the acute effects of SSC fatigue on both strength and endurance trained athletes will be investigated.

It is also clear that gaps in the knowledge of the physiology of well-trained rowers and the most effective training methods for well-trained rowers remain. Although there is evidence that HIIT is a beneficial method of training for well-trained endurance athletes it is clear from the findings presented that further research on the effects of HIIT in well-trained rowers is required. Furthermore the mechanisms responsible for performance improvements associated with HIIT in well-trained endurance athletes remain ambiguous. Therefore this research will also investigate the key physiological determinants of 2000 m rowing performance and the impact of HIIT on well-trained rowers.
Chapter 3. The Effect of Maximal Stretch Shortening Cycle Fatigue on The Biomechanical Properties of Strength Trained Athletes.
3.1. Abstract

The purpose of this study was to investigate the effects of maximal fatigue on the mechanical performance of strength trained athletes. Ten strength trained athletes performed four initial sets of drop (DJ) and rebound jumps (RBJ) to establish baseline values. The athletes then performed a maximal SSC fatigue protocol on a sledge and force plate apparatus. DJ and RBJ sets were then performed 15, 45, 120, 300 and 600 seconds following the maximal workout to establish the effect of the maximal workout. Measurements of peak ground reaction force (GRF), contact time (CT), height jumped, reactive strength index (RSI) and leg-spring stiffness ($k_{vert}$) were calculated. The maximal fatigue workout resulted in a significant reduction in GRF peak ($p < 0.01$), height jumped ($p < 0.001$), RSI ($p < 0.001$) and $k_{vert}$ ($p < 0.01$) and a significant increase in CT ($p < 0.001$) in the DJ. The fatigue workout had similar effects on the RBJ with significant reductions in GRF peak ($p < 0.01$), height jumped ($p < 0.01$), RSI ($p < 0.01$) and $k_{vert}$ ($p < 0.01$) and a significant increase in CT ($p < 0.01$). The results also indicated a post activation potentiation effect with a significant increase in GRF peak ($p < 0.05$) in the DJ.

3.2. Introduction

Human movement, and indeed complex sports movements, seldom involve pure forms of isometric, concentric and eccentric muscle actions, with basic human muscle function typically involving the stretch-shortening cycle (SSC). The SSC is a phenomenon which involves both eccentric and concentric contractions whereby an eccentric contraction precedes a concentric contraction with minimum delay between the eccentric and concentric phase. The SSC occurs where body segments are subjected to impact or stretch forces. Everyday examples of the body’s utilisation of the SSC include running and hopping while more advanced skills which utilise it may include cross country skiing or flat water kayaking.

The importance of the SSC to sprinting and jumping performance is well established (Harrison et al. 2004, Kyrolainen and Komi 1995, Kubo et al. 2000). Research has demonstrated that the eccentric or pre-stretching phase can enhance force production and power output during subsequent concentric contractions (Cavagna et al. 1965, Komi and Bosco 1978). Strength athletes continually rely on their ability to effectively utilise the SSC.
in order to maximise performance as the SSC is essential to the production of explosive, powerful and fast movements.

The SSC is a form of muscle function in which all the key components of performance sources, mechanical, neural and metabolic, are stressed and therefore the SSC provides a unique model to investigate the effects of neuromuscular fatigue on performance (Komi 2000). SSC effectiveness is influenced by the rate and magnitude of the stretch, the level of activation, resulting stiffness of the muscle tendon unit prior to the concentric phase, the change in muscle length during the stretch, and the time lag between the completion of the stretch and the initiation of the concentric contraction (Anderson 1996). Avela and Komi (1998) hypothesised that decreased performances due to SSC fatigue may be due, in part, to a loss in recoil characteristics associated with the SSC and also alterations in muscle stiffness. Komi et al. (1986) suggested that with the progress of fatigue, there is a change in the stiffness characteristics and hence, a reduction in SSC-type performances.

Fatiguing SSC exercise has a strong loading effect on skeletal muscle and the fatigue responses of repeated SSC actions are complex. Repeated high-force SSC muscle actions, used in many sporting activities, can lead to acute and long-term impairments in neuromuscular performance. In addition to alterations in stiffness regulation, SSC fatigue has been characterised by increases in contact time, changes in peak GRF, decreases in RSI, decreases in performance parameters in addition to metabolic changes (Komi and Nicol 2000). Gollhofer et al. (1987a) performed one of the first SSC fatigue studies and found that the fatigue was characterised by increases in contact time and that the force-time curves were influenced by the fatigue. Nicol et al. (1991b) also reported that the ground reaction force-time curves revealed a clear drop in the vertical force after the impact peak with a simultaneous increase in contact time.

Both the acute and delayed recovery following submaximal SSC fatigue have been investigated (Gollhofer et al. 1987a, Gollhofer et al. 1987b, Avela et al. 1999) (Skurvydas et al. 2002, Nicol et al. 2003) and it has been observed that recovery from SSC fatigue occurs in a bimodal fashion, whereby there is a dramatic decline post fatigue followed by a short-lasting recovery and then a subsequent decline, which may peak around the 2nd or 3rd day post fatigue (Horita et al. 1999, Komi 2000). Faulkner et al. (1993) have suggested that the initial immediate reduction in performance is primarily related to metabolic disturbances
while the secondary decline is associated with the inflammatory process of muscle damage. More recently Comyns et al. (2011) investigated the recovery process from a maximum SSC fatigue protocol. The authors found that while there was an initial decline in performance immediately post-fatigue, there was also a significant enhancement in some biomechanical variables, namely GRF, leg spring stiffness and CT, approximately 300 seconds post-fatigue. Although no performance outcome was reported the jumping process was altered with a shorter, stiffer and more elastic leg spring action being used.

Despite the importance of the SSC to strength athletes, few studies have investigated the effect of maximal SSC fatigue on performance and the subsequent recovery from fatigue. The majority of past research has focused on submaximal SSC fatigue. However, the mechanisms in maximal SSC fatigue may differ from the mechanisms in sub-maximal fatigue as maximal fatigue workouts involve greater GRF, greater muscle activation and shorter time periods. Strojnik & Komi (1998) used a maximal SSC fatigue workout to investigate the possible mechanisms of neuromuscular fatigue, while Comyns et al. (2011) investigated the effect of a maximal SSC fatigue workout on elite male rugby players. Consequently the aim of this study was to examine the effect of a maximal SSC fatigue workout on the biomechanical performance of well-trained strength trained athletes. In addition the study aimed to investigate the recovery of well-trained strength trained athletes following maximal SSC fatigue in an attempt to identify if a post activation potentiation effect could be achieved from a maximal SSC fatigue workout.

3. 3. Method

3. 3. 1. Subjects

Ten well-trained male rugby players contracted to the Munster sub-academy participated in this study. All participants were members of the academy for a minimum of one year and were involved in a structured rugby training program for a minimum of three years. The physical characteristics of the athletes are presented in Table 3.1. All participants were fully informed verbally and in writing of the requirements, and the potential risks and benefits of participating in the study (Appendix A). An informed consent form (Appendix A) and pre-test questionnaire (Appendix C) were completed by each participant. All participants were required to undertake no more than 30 minutes light training and to refrain from any
strength training or plyometrics during the 24 hours prior to the test. All experimental procedures were approved by the University of Limerick Ethics Committee.

Table 3.1: Physical characteristics of the participants

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>Mass (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>20 ± 1</td>
<td>180.9 ± 4.9</td>
<td>89.8 ± 12.8</td>
</tr>
</tbody>
</table>

Data expressed as mean ± SD

3.3.2. Research Design

The participants attended the laboratory for two sessions, the first of which was a familiarization session. All testing took place on the second visit to the laboratory and was performed on a sledge and force plate apparatus and recorded on 50 Hz SVHS videocassettes via a Panasonic AGDP800 camera (Panasonic, Osaka, Japan). Each participant performed four sets of one-legged drop jumps (DJ) and one-legged rebound jumps (RBJ), with each set consisting of one DJ from a height of 30 cm followed immediately by one RBJ (DJ/RBJ set). These initial four sets were to establish each participant’s mean baseline scores. A maximum fatigue workout was then performed which was followed immediately with a DJ/RBJ set at 15, 45, 120, 300 and 600 seconds after. The dependable variables for each jump included peak GRF, CT, height jumped, RSI and $k_{vert}$.

3.3.3. Instrumentation and Data Collection

3.3.3.1. Sledge Apparatus

All testing was performed on a specially designed sledge apparatus, as seen in figure 3.1, consisting of 3 main parts; a sledge frame, sliding chair and a force platform. The sledge apparatus has been specially designed to enable the quantification of the mechanical properties of the musculoskeletal systems during dynamic exercise. The sledge frame consists of a box metal frame with sledge rails inclined at 30º. A sliding chair is mounted upon the sledge rails on steel rollers in order to minimise friction. The chair includes a harness and Velcro straps which are used to secure participants in the chair and to assist in eliminating any movement of the upper body. A winch with a quick pull-release mechanism
is located at the top of the sledge rails. This can be attached to the sledge chair and used to hoist the participants to desired dropping heights for DJ.

![Figure 3.1: Specially designed sledge apparatus](image)

**3. 3. 3. 2. Force Platform**

A force platform (AMTI OR6-5, AMTI, MA, USA) is secured at 60° to the sledge frame, or right angles to the slope of the frame as can be seen in figure 3.1. The force platform was sampled at 1000 Hz to give values for vertical ground reaction forces. For each set of jumps, data recorded from the force plate was exported to Microsoft Excel (Release 9.0.6926). In Excel, the force traces recorded were graphed with respect to time and instances of initial foot contact, full crouch depth, take-off and landing were identified. Identification of these instances allowed for the calculation of the dependent variables namely, height jumped, contact time and reactive strength index.

**3. 3. 3. 3. Video Analysis**

Each set of jumps was videotaped to assist in the calculation of leg spring stiffness. In order to calculate stiffness the maximum vertical displacement of the centre of the mass during limb contact must be identified. This involved 50 Hz sagital plane SVHS recordings
which were made through a Panasonic AGDP800 camera mounted on a tripod perpendicular to the sledge, as seen in figure 3.2. In addition a reflective marker, also seen in figure 3.2, was attached to the sledge which could be tracked throughout the testing. The video recordings were digitised using Peak Motus® (Peak Performance Technologies, Colorado, USA) in order to determine the displacement of the sledge at the bottom of the crouch for both the DJ and RBJ. For calibration purposes a four point “Affine scaling” frame, as seen in figure 3.3, which measured 1.51 m by 1.1 m was placed in front of the sledge apparatus and filmed for 4 frames.

Figure 3.2: Camera set up and reflective marker on sledge

Figure 3.3: Four point affine scaling frame (1.5 m x 1.1 m) used for calibration
3.3.4. Test Procedure

The following is a schematic summary (figure 3.4) of the testing procedure.

![Schematic Diagram of Test Protocol]

**Figure 3.4: Schematic diagram of test protocol**

3.3.4.1. General and Specific Warm-Up

Upon arrival at the laboratory each participant’s personal details, height and weight were recorded. A general and specific warm-up was then completed. The general warm-up involved a light 5 minute jog at the self-selected pace. All participants, proficient in the DJ and RBJ technique following the familiarisation session, then performed a specific warm-up of four practice sets of drop and rebound jumps on the sledge apparatus.
3.3.4.2. Pre-test

Participants selected the dominant/preferred leg and while seated in the sledge apparatus, with the selected leg fully extended, a mark was placed on the sledge 30 cm up from the seat. The participant was dropped from this 30 cm mark for each DJ/RBJ set. A height of 30 cm was selected based on the results of previous research that used this experimental design (Comyns et al. 2006, Harrison and Gaffney 2004). The participants were instructed to keep arms across the chest at all times and once dropped to make initial contact on the force plate with a straight leg and to focus on minimising ground contact time and maximising jump height for each jump. A straight leg landing was also required following the RBJ. Each participant performed 4 DJ/RBJ sets with 90 seconds rest between each to ensure a full recovery. The 4 pre-test sets of jumps established the baseline values for each dependent variable.

3.3.4.3. Fatigue Workout

Each pre-test DJ/RBJ set was analysed immediately. The height jumped for each RBJ was calculated and the jump with the maximum height was selected. 90% of this jump height was calculated and marked on the sledge from a position where the subject was seated in the sledge apparatus with the leg fully extended. 90% was selected based on previous research that used this experimental design (Comyns et al. 2011). The mean 90% level was 31.4 ± 0.5 cm. The fatigue workout began with the participant being dropped from a height of 30 cm. Participants were instructed to rebound as long as possible to the 90% mark, until they failed to reach 90% for three consecutive jumps. The 90% height mark was marked with reflective tape. This reflective tape, along with an OMRON Opto-Switch (EE-SY410) enabled the identification of jumps which did not reach the 90% mark, as each time the Opto-Switch passed the reflective tape it flashed on. Therefore, when the desired height was not achieved the light did not show. The fatigue workout was recorded on a 50 Hz SVHS videocassette via a Panasonic AGDP800 camera for later analyse in order to identify the duration of each participant’s fatigue workout and also the number of jumps completed.
3. 3. 4. 4. Post Test

Immediately following the fatigue workout the participant was prepared to perform 5 post test sets of DJ/RBJ. These sets were performed 15, 45, 120, 300 and 600 seconds post the fatigue workout.

3. 3. 4. 5. Cool Down

A cool-down of light jogging and stretching was completed at the end of the testing session.

3. 3. 5. Calculation of the Dependent Variables

3. 3. 5. 1. Peak Ground Reaction Force (GRF)

The peak GRF was recorded by the AMTI force plate and was obtained from the force-time data. It is the maximum force recorded by a person when performing a jump on the force plate.

3. 3. 5. 2. Contact Time (CT)

Ground contact time is the length of time the foot is in contact with the force plate during the jumps. It is the time difference between the initial landing and subsequent take-off. It can be calculated from the reaction force-time traces obtained from the force plate.

3. 3. 5. 3. Height Jumped

Height jumped is the greatest height participants reach after take-off from the force plate. It is a product of flight time (FT) which is calculated from inspection of the vertical ground reaction force-time traces for each jump. FT is the difference between the initial take-off and subsequent landing for each jump. From this calculation of FT, height jumped can be calculated using the second mathematical equation of linear motion (Comyns et al. 2011).
\[ s = ut + \frac{1}{2} at^2 \]

**Equation 3.1: Calculation of flight time**

Where:
\( s \) = displacement
\( u \) = initial velocity = 0 m.s\(^{-1}\)
\( t \) = time to top of jump = FT/2
\( a \) = acceleration = gravity (gravity = 9.81/2 due to the sledge being angled at 30° to the horizontal (Harrison and Gaffney 2004, Flanagan and Harrison 2007))

Therefore:

\[ s = 0 + \frac{1}{2} \left( \frac{9.81}{2} \right) \left( \frac{FT}{2} \right)^2 \]

\[ = \frac{1 \times 9.81 \times FT^2}{16} \]

\[ = \frac{9.81 \times FT^2}{16} \]

**Equation 3.2: Calculation of height jumped (Harrison and Gaffney 2004, Flanagan and Harrison 2007)**

3. 3. 5. 4. Reactive Strength Index (RSI)

Reactive strength can be defined as the ability to change quickly from an eccentric to a concentric contraction (Young 1995). It is a calculated figure derived by dividing the height jumped by the ground contact time (height jumped/CT) (Young 1995).

3. 3. 5. 5. Leg-Spring Stiffness

Stiffness is the relationship between the deformation of an object and a given force (Butler et al. 2003). The stiffness of a human system it suggested to have two distinct components, namely vertical stiffness \( k_{vert} \) and overall leg-spring stiffness \( k_{leg} \). For vertical hopping \( k_{vert} \) and \( k_{leg} \) are identical. This study involved the use of a sledge and force plate apparatus for all jumps with the sledge apparatus only allowing for movement along one axis and in one
plane and with no forwards, backwards or side movements. Therefore the jumps
performed in this study can be viewed as vertical hopping movements and the spring-mass
model for vertical hopping can be used to analyse leg-spring stiffness on the sledge
apparatus. Because of the spring-like nature of the leg during DJs, the peak GRF and the
peak leg-spring displacement both occur simultaneously at the middle of the ground
contact phase (Ferris and Farley 1997, McMahon and Cheng 1990). Vertical stiffness can
be defined as the ratio of peak force in the spring ($F_{y_{peak}}$), to the displacement of the leg-
spring ($\Delta L$), at the instant the leg-spring is maximally compressed (McMahon and Cheng
1990).

$$k_{vert} = \frac{F_{y_{peak}}}{\Delta L}$$

Equation 3.3: Calculation of leg spring stiffness (McMahon and Cheng 1990)

3. 3.6. Statistical Analyses

All statistical calculations were performed with the statistical package SPSS (Release 16.0).
Individuals respond to training stimulus and fatigue in different ways and it has previously
been suggested that the optimal rest interval may vary from person to person. For this
reason each subject’s post test jumps, which comprised of a DJ/RBJ set at 15, 45, 120, 200
and 300s post fatigue, were analysed. From these recovery intervals, each subject’s
minimum and maximum score for each dependent variable, irrespective of time, was
identified. This allowed for the identification of fatigue and any possible potentiation
without the interference of individual variation across recovery time.

Normality of all variables was verified using the Shapiro-Wilk test. A one-way analysis of
variance (ANOVA) with repeated measures was used for each dependent variable to
evaluate the differences between the average of the baseline scores and the minimum and
maximum scores achieved in the post tests. The GLM ANOVA had 1 within-subjects
factor, namely Condition, with 3 levels (baseline, minimum and maximum). This analysis
was carried out for both the DJ and RBJ.

Effect sizes were obtained for each dependent variable from the (GLM) ANOVA using
partial eta$^2$ ($\eta_p^2$). Partial eta$^2$ can be defined as the ratio of variance accounted for by an
effect and that effect plus its associated error variance within an ANOVA study. Partial eta-squared is computed as follows:

\[ \eta_p^2 = \frac{SS_{effect}}{SS_{effect} + SS_{error}} \]

\textbf{Equation 3.4: Calculation of partial eta-squared}

Where:
- \( SS_{effect} \) = effect variation
- \( SS_{error} \) = error variation.

Interpretation of the effect size was based on the scale for effect size classification as presented by Hopkins (2002). This scale is based on \( f \)-values for effect size and these were converted to \( \eta_p^2 \) using the formula:

\[ \eta_p^2 = \sqrt{\frac{f^2}{1 + f^2}} \]

\textbf{Equation 3.5: The conversion of \( f \)-values to \( \eta_p^2 \) }

Using this formula the scale for classification of \( \eta_p^2 \) was as follows:

\textbf{Table 3.2: Table showing the classification of \( \eta_p^2 \) as an effect size}

<table>
<thead>
<tr>
<th>( \eta_p^2 )</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 0.04</td>
<td>Trivial</td>
</tr>
<tr>
<td>0.041 – 0.249</td>
<td>Small</td>
</tr>
<tr>
<td>0.25 – 0.549</td>
<td>Medium</td>
</tr>
<tr>
<td>0.55 – 0.799</td>
<td>Large</td>
</tr>
<tr>
<td>&gt; 0.8</td>
<td>Very Large</td>
</tr>
</tbody>
</table>

\textbf{3. 4. Results}

Table 3.3 presents the performance results of the fatigue workout.
Table 3.3: Performance scores of fatigue workout

<table>
<thead>
<tr>
<th>No. of jumps</th>
<th>Duration of workout(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>55 ± 24.9</td>
<td>69.3 ± 21.2</td>
</tr>
</tbody>
</table>

Data expressed as mean ± SD

The pre-fatigue and post-fatigue results of this investigation are presented in two sections. The results of the DJ are reported in section 3.4.1 and the results of the RBJ are reported in section 3.4.2. The baseline scores (mean ± SD) for each of the dependent variables for both the DJ and RBJ jump are presented in Table 3.4.

Table 3.4: Mean baseline DJ and RBJ values for each dependent variable

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Drop Jump</th>
<th>Rebound Jump</th>
</tr>
</thead>
<tbody>
<tr>
<td>GRF (N)</td>
<td>1624.34 ± 250.5</td>
<td>1770.58 ± 258.13</td>
</tr>
<tr>
<td>Height jumped (cm)</td>
<td>37.7 ± 4.44</td>
<td>36.51 ± 4.35</td>
</tr>
<tr>
<td>CT (s)</td>
<td>0.544 ± 0.121</td>
<td>0.531 ± 0.126</td>
</tr>
<tr>
<td>RSI</td>
<td>0.721 ± 0.167</td>
<td>0.719 ± 0.167</td>
</tr>
<tr>
<td>$k_{vert}$ (kN.m$^{-1}$)</td>
<td>7.587 ± 2.889</td>
<td>6.927 ± 2.469</td>
</tr>
</tbody>
</table>

Data expressed as mean ± SD

The following graphs in sections 3.4.1 and 3.4.2 illustrate the effect of maximal fatigue on the dependent variables, GRF, height jumped, CT, RSI and $k_{vert}$. Each graph represents an individual dependent variable with each x-axis representing the baseline scores. The graphs display the change from the baseline scores to the minimum and maximum scores achieved in the post-fatigue recovery intervals. The minimum and maximum scores across all 5 recovery intervals (15, 45, 120, 300 and 600 seconds) were selected in an attempt to identify the effects of fatigue and potentiation without the interference of individual variation across recovery time. In each graph the mean baseline score was subtracted from the minimum and maximum score achieved in order to show a representation of the actual change that occurred. The actual percentage changes and effect sizes of maximal fatigue on performance are also included.
3. 4. 1. Drop Jump Results

The GRF results for the DJ are presented in figure 3.5. The GLM ANOVA results showed a significant reduction ($p = 0.005$) in the peak GRF produced immediately following a maximal fatigue workout. There was also a significant improvement ($p = 0.041$) in peak GRF during the latter recovery phases.

![Figure 3.5: Mean (± 95% CI) GRF difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. **$p < 0.01$; *$p < 0.05$](image)

Figure 3.6 illustrates the results for height jumped during the DJ. There was a significant reduction ($p < 0.001$) following maximal fatigue.

![Figure 3.6: Mean (± 95% CI) height jumped difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. ***$p < 0.001$](image)
The results for CT during the DJ are presented in figure 3.7. There was a significant increase (p < 0.001) in CT immediately following the maximal fatigue workout. There was also a decrease in CT during the recovery intervals, however, this decrease was not significant (p > 0.05).

**Figure 3.7**: Mean (± 95% CI) CT difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. ***p < 0.001

Figure 3.8 illustrates the results for RSI during the DJ. Immediately following maximal fatigue, there was a significant reduction (p < 0.001) in RSI. Throughout the recovery intervals, it can be seen that RSI did not recover to baseline levels and the maximum post-fatigue value remained below pre-fatigue baseline levels.

**Figure 3.8**: Mean (± 95% CI) RSI difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. ***p < 0.001
The $k_{ver}$ results of the DJ are illustrated in the figure 3.9. There was a significant reduction ($p = 0.003$) in $k_{ver}$ following the maximal fatigue workout. During the recovery intervals $k_{ver}$ did increase above baseline levels as shown by the maximum jump. This increase, however, was not significant ($p > 0.05$).

![Figure 3.9: Mean (± 95% CI) $k_{ver}$ difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. **$p < 0.01$](image)

The actual percentage changes in performance for each of the dependent variables during the DJ can be seen in Table 3.5. This percentage change represents the change from the mean baseline value to the minimum and maximum values achieved during the recovery intervals.

Table 3.5: Percentage change for the DJ dependent variables from baseline values to minimum and maximum values achieved during recovery.

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>% Change Baseline → Minimum</th>
<th>% Change Baseline → Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>GRF</td>
<td>11.9%**</td>
<td>7.9%*</td>
</tr>
<tr>
<td>Height Jumped</td>
<td>21.0%***</td>
<td>-2.1%</td>
</tr>
<tr>
<td>CT</td>
<td>21.9%***</td>
<td>7.9%</td>
</tr>
<tr>
<td>RSI</td>
<td>31.8%***</td>
<td>-0.4%</td>
</tr>
<tr>
<td>$k_{ver}$</td>
<td>31.4%**</td>
<td>23.6%</td>
</tr>
</tbody>
</table>

***$p < 0.001$; **$p < 0.01$; *$p < 0.05$
The $\eta^2$ values for the DJ can be seen in Table 3.6. Measures of effect sizes using $\eta^2$ are measures of the degree of association between an effect and the dependent variable (i.e. the effect of maximal fatigue workout on each of the dependent variables). If the value of the measure of association is squared it can be interpreted as the proportion of variance in the dependent variable that is attributable to each effect.

Table 3.6: $\eta^2$ values for the DJ dependent variables and classification of the magnitude of the effect size according to Hopkins (2002).

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Baseline → Minimum</th>
<th>Baseline → Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>GRF</td>
<td>0.595: medium**</td>
<td>0.388: medium*</td>
</tr>
<tr>
<td>Height Jumped</td>
<td>0.863: v.large ***</td>
<td>0.134: small</td>
</tr>
<tr>
<td>CT</td>
<td>0.780: large ***</td>
<td>0.306: medium</td>
</tr>
<tr>
<td>RSI</td>
<td>0.826: v.large ***</td>
<td>0.004: trivial</td>
</tr>
<tr>
<td>$k_{vert}$</td>
<td>0.638: large **</td>
<td>0.347: medium</td>
</tr>
</tbody>
</table>

***p < 0.001; **p < 0.01; *p < 0.05

3. 4. 2. Rebound Jump Results

The mean GRF results are presented in figure 3.10. From this figure it can be seen that there was a significant reduction ($p = 0.002$) in peak GRF immediately following maximal fatigue, illustrated below as the “minimum”. There was also an increase in the peak GRF above baseline values through the recovery intervals. This increase, however, was not significant.
Figure 3.10: Mean (± 95% CI) GRF difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. **p < 0.01

Figure 3.11 illustrates the effect of maximal fatigue on the height jumped. There was a significant reduction (p = 0.004) in height jumped immediately following maximal fatigue and while there was some improvement in height jumped during the recovery intervals, this improvement was not significant.

Figure 3.11: Mean (± 95% CI) height jumped difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. **p < 0.01

The mean CT differences in the RBJ are presented in figure 3.12. Immediately following the maximal fatigue workout, there was a significant increase (p = 0.004) in ground CT. During the recovery intervals there was a decrease in CT below baseline levels but the decrease was not significant.
The RBJ RSI results are illustrated in figure 3.13. The maximal fatigue workout resulted in a significant decrease \((p = 0.002)\) in RSI. This decrease was reversed during the recovery intervals to a level above baseline values, but this increase above baseline was not significant.

Figure 3.13: Mean (± 95% CI) RSI difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. **\(p < 0.01\)

Figure 3.14 presents the results of \(k_{\text{vert}}\) for the RBJ. \(k_{\text{vert}}\) decreased significantly \((p = 0.003)\) following maximal fatigue. During the recovery intervals there was an increase in \(k_{\text{vert}}\), however, this increase was not significant.
Figure 3.14: Mean (± 95% CI) $k_{vert}$ difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. **p < 0.01

The actual percentage changes in performance for each of the dependent variables during the RBJ can be seen in Table 3.7. This percentage change represents the change from the mean baseline value to the minimum and maximum values achieved during the recovery intervals.

Table 3.7: Percentage change for the RBJ dependent variables from baseline values to minimum and maximum values achieved during recovery. **p < 0.01; *p < 0.05

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>% Change Baseline → Minimum</th>
<th>% Change Baseline → Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>GRF</td>
<td>10.4%**</td>
<td>7.3%</td>
</tr>
<tr>
<td>Height Jumped</td>
<td>16.5%**</td>
<td>0.7%</td>
</tr>
<tr>
<td>CT</td>
<td>18.9%**</td>
<td>3.7%</td>
</tr>
<tr>
<td>RSI</td>
<td>26.5%**</td>
<td>3.9%</td>
</tr>
<tr>
<td>$k_{vert}$</td>
<td>25.9%**</td>
<td>11.6%</td>
</tr>
</tbody>
</table>

**p < 0.01; *p < 0.05

The $\eta^2$ values for the RBJ can be seen in Table 3.8. The medium and large effect sizes indicate that the fatigue workout resulted in considerable change to the dependent variables.
Table 3.8: $\eta_p^2$ values for the RBJ dependent variables and classification of the magnitude of the effect size according to Hopkins (2002).

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Baseline → Minimum</th>
<th>Baseline → Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>GRF</td>
<td>0.659: large**</td>
<td>0.200: small</td>
</tr>
<tr>
<td>Height Jumped</td>
<td>0.619: large**</td>
<td>0.009: trivial</td>
</tr>
<tr>
<td>CT</td>
<td>0.551: medium**</td>
<td>0.095: small</td>
</tr>
<tr>
<td>RSI</td>
<td>0.680: large**</td>
<td>0.048: small</td>
</tr>
<tr>
<td>$k_{vert}$</td>
<td>0.645: large**</td>
<td>0.156: small</td>
</tr>
</tbody>
</table>

***p < 0.001; **p < 0.01; *p < 0.05

3.5. Discussion

The effect of maximal SSC fatigue on the mechanical properties of strength trained athletes was investigated in this study. The results indicate that a maximal SSC fatigue workout has a significant effect on both the performance outcome and the biomechanical performance of jumping. In addition, the study also provides an insight into the recovery process following a maximal SSC fatigue workout and indicates that the workout may have the potential to elicit a post activation potentiation (PAP) effect.

Following the maximal SSC fatigue workout there was a significant decrease in height jumped, GRF, RSI and $k_{vert}$ and a significant increase in CT in both the DJ and RBJ. The significant decrease in height jumped for both the DJ and RBJ indicated that the overall performance outcome was reduced while the GRF, CT, RSI and $k_{vert}$ variables provided information on the biomechanical performance of the jump. Performance in several sports, for example sprinting or rugby, can depend on the ability to develop power and the basis for power generation in many sporting contexts is the SSC. The SSC is vital for the effective execution of the DJ and RBJ and the dependent variables used in this study (height jumped, GRF, CT, RSI and $k_{vert}$) may indicate the effectiveness of the SSC.

Height jumped was the performance outcome of the DJ and RBJ. The height achieved in a vertical jumping action is said to be representative of the power production capabilities of an athlete (Carlock et al. 2004). Smilios (1998) has argued that the vertical jump is considered an effective evaluation of muscular power as the height of the jump correlates significantly with maximal power in units related to body mass. In addition to having an
ability to jump high, strength trained athletes must also possess the ability to maintain jumping capacity throughout a performance. A significant reduction in height jumped in both the DJ (p < 0.001, -21%) and RBJ (p < 0.01, -6.5%) following maximal SSC fatigue was observed in the present study. Comyns et al. (2011) reported similar findings with a significant reduction in flight time, from which height jumped is derived, in both the DJ and RBJ.

Maximal SSC fatigue may also affect the biomechanical jumping process. Minimal CT and a stiff leg action are required for an effective SSC. CT can be identified as the time difference between the initial foot contact and take-off and it has been observed that the shorter the transition period between the eccentric and concentric phases, the greater the potentiation effect in the concentric contraction (Cavagna et al. 1964). It has been demonstrated that the ability to produce short CT’s can improve anaerobic power characteristics (Houmard et al. 1994). Schmidtbleicher (1992) also reported that a short amortization phase is required for the subsequent concentric contraction to elicit the advantages of both the stored elastic energy and stretch reflex, two of the main mechanisms purported to explain the enhancement of force development associated with the SSC. If this time interval or phase is too long, the stored elastic energy can be wasted and the mechanical efficiency of the concentric phase will decrease. In this present study CT was significantly increased immediately following the fatigue workout in both the DJ (p < 0.001, 21.85%) and RBJ (p < 0.01, 18.87%). These results indicate that the increase in the amortization phase may have led to a decrease in performance due to an inability to exploit the advantages of the stored elastic energy and stretch reflex. Several submaximal SSC fatigue studies have reported similar findings of increased CT (Gollhofer et al. 1987b, Avela and Komi 1998, Horita et al. 2003) however, only Comyns et al. (2011) observed similar results following maximal SSC fatigue. Comyns et al. (2011) concluded that for an effective SSC function minimising ground CT is imperative.

An effective level of leg spring stiffness is necessary for optimal utilisation of the SSC as it allows for the efficient utilisation of the stored elastic energy in the musculoskeletal system that occurs during the pre-stretch phase of the SSC (Butler et al. 2003). Increased leg spring stiffness has been correlated with faster stride frequencies (Farley and Gonzalez 1996) and running velocity (Arampatzis et al. 1999, Bret et al. 2002) and an inverse relationship between stiffness and the metabolic cost of running has been established (Dalleau et al.
When performance is dependent on mechanical power output, for example in sprinting, it has been suggested that it is possible to produce a better performance through higher stiffness (Arampatzis et al. 2001). Arampatzis et al. (2001) also suggested that it is possible to maximise vertical take-off velocity in jumps through various leg stiffness values. Leg spring stiffness is an important component of mechanical efficiency for strength trained athletes and decreases in performance may result partly from alterations in stiffness regulation. Repeated SSC’s, both sub-maximal and maximal, have been reported to alter leg spring stiffness (Harrison and Gaffney 2004, Comyns et al. 2006, Nicol et al. 1991a). An association between reduced stretch reflex sensitivity and decreased stiffness following highly fatiguing SSC exercise has also been reported (Avela and Komi 1998). Komi et al. (1986) suggested that with the progress of fatigue, there was a change in stiffness characteristics, as did Comyns et al. (2011) who reported a significant reduction in leg spring stiffness immediately following maximal SSC fatigue. A similar effect was observed in the present study. A significant reduction in leg spring stiffness was observed in both the DJ (p < 0.01, 31.4%) and RBJ (p < 0.01, 25.9%) indicating an alteration in stiffness regulation and therefore the alteration of the effectiveness of the SSC.

The effect of fatigue on force production has been widely investigated. A reduction in force production in skeletal muscle may be a consequence of decreased activation, decreased sensitivity to calcium or decreased force capacity (MacIntosh and Allen 2000). During the SSC the force developed is largely dependent on the eccentric phase as maximal force during the concentric phase of the movement has been shown to be enhanced by prior eccentric muscle actions. However, the force potentiation associated with the SSC has been shown to significantly diminish following fatiguing SSC exercise (Comyns et al. 2011, Gollhofer et al. 1987b, Nicol et al. 1991b). Through detailed graphic analysis, Gollhofer et al. (1987a) was one of the first to reveal that force-time curves during contact on a force platform were influenced by SSC fatigue. They reported that the initial force peak became higher and the subsequent drop in force became more pronounced. Nicol et al. (1991b) reported similar findings when they performed a before-after marathon comparison of the GRF-time curves and revealed a clear drop in the vertical force after the impact peak. This may suggest that the neuromuscular system changed during the SSC fatigue with possible contractile failure or changes in neural activation whereby the nervous system may attempt to compensate for the loss in contractile capacity by changing muscle activation patterns, the duration of which would be dependent on the nature of the SSC fatigue (Komi and
Nicol 2000). This neuromuscular change may ultimately result in a reduction in overall impulse production with the potential to reduce performance. Following the maximal SSC fatigue workout in this present study, a significant decrease in GRF was observed in both the DJ (p < 0.01, 11.93%) and RBJ (p < 0.01, 10.42%) as recorded by an AMTI force platform.

The ability to change quickly from an eccentric to concentric contraction has been identified as the RSI (Young 1995) and may provide information regarding the effectiveness of the SSC. It involves the ability of the neuromuscular system to produce the greatest possible impulse in the shortest possible time period. RSI is often interpreted as an indicator of the ability of an athlete to withstand stretch loads during SSC activity (McClymont 2003). Few studies have assessed RSI following SSC fatigue. However, one study investigating the effect of maximal SSC fatigue on elite rugby players (Comyns et al. 2011) found a large significant reduction in RSI immediately following a maximal SSC fatigue workout and suggested that for efficient SSC behaviour, an improvement in RSI values should be observed post fatigue. The results of this present study demonstrate that maximal SSC fatigue significantly reduces an athlete’s ability to effectively utilise the SSC and change quickly from an eccentric to concentric contraction. A significant reduction in RSI was observed in both the DJ (p < 0.001, 31.8%) and RBJ (p < 0.01, 26.5%) immediately following the SSC fatigue workout.

Similar to previous SSC fatigue studies (Comyns et al. 2011, Avela and Komi 1998, Gollhofer et al. 1987a, Gollhofer et al. 1987b, Nicol et al. 1991a, Horita et al. 2003, Horita et al. 1996, Horita et al. 1999) the maximal SSC fatigue workout involved in this study evoked a significant change in both the performance measure (jump height) and biomechanical performance (GRF, CT, RSI and \(k_{\text{vert}}\)) of the DJ and RBJ. The results demonstrate that following maximal SSC fatigue there is reduction in force production in addition to a longer CT and reduced leg spring stiffness and RSI, all of which negatively impact on performance and ultimately indicate a loss in the effectiveness of the SSC.

Following SSC fatigue, recovery is said to occur in a bimodal fashion (Horita et al. 1999, Komi 2000), some characteristics of which are evident in this study whereby there was an initial dramatic decline in all variables in both the DJ and RBJ which was followed by a gradual recovery. However, some variables, including GRF, CT and \(k_{\text{vert}}\), not only
recovered to baseline values but were enhanced above baseline values, representing an enhancement which was superior to baseline values achieved prior to the fatigue workout. Only one previous study has demonstrated that a maximal SSC fatigue workout could enhance DJ and RBJ performance (Comyns et al. 2011). Comyns et al. (2011) attributed the enhancement in performance to PAP effect. However, there was no change in the performance outcome measure, height jumped, or RSI and therefore it may be suggested that, while maximal SSC fatigue may alter SSC behaviour, it does not have the ability to enhance performance through PAP.

PAP is an acute improvement in performance as a result of prior muscle activation (Hodgson et al. 2005). PAP may indeed be an important concept for strength trained athletes as it has been postulated that explosive movements may be enhanced if preceded by a biomechanically similar exercise. PAP is suggested to coexist with fatigue with optimal performance occurring when fatigue has subsided but the potentiated effect still exists (Hodgson et al. 2005). There appears to be an optimal time when the muscle has recovered from fatigue but is still potentiated and Hodgson et al. (2005) have cautioned that the optimal time appears to be variable among individuals. Therefore any PAP effect in group design studies may possibly be masked. For this reason, in this present study, each participants “maximum” post-fatigue score for each dependent variable, irrespective of time, was selected rather than focusing on one time period throughout the recovery. This allowed for identification of any potentiation effect, if indeed it does exist, within the group as it facilitated individual variation.

Comyns et al. (2011) concluded a PAP effect following significant improvements in CT, RSI, GRF and leg spring stiffness. In this present study no performance outcome improvement was seen for the DJ or RBJ, but a significant increase in GRF in the DJ was observed. While there was no other significant improvements in either the DJ or RBJ there were some notable changes which may indicate that the jumping process was altered during recovery from maximal SSC fatigue. In the DJ there was a particularly large increase in k<sub>vert</sub> (23.6%) while there was also an improvement in CT (7.9%). This type of alteration in the jumping process would result in the DJ being performed with a stiffer, shorter and more elastic leg spring action and a shorter time lag between the eccentric and concentric phase. Similarly, while there was no significant improvements in the RBJ results there were some noticeable differences. Leg spring stiffness showed the greatest improvement (11.6%),
while CT, GRF and RSI also showed evidence of improvement (3.7%, 7.3% and 3.9% respectively). While these improvements were not significant they do indicate, similar to Comyns et al. (2011), that the maximal SSC fatigue workout altered the manner in which the muscle tendon unit behaved, resulting in a more effective SSC behaviour. However, it is worth noting that the lack of statistical significance in this study may be due to the subjects participating in the study. The subjects used by Comyns et al. (2011) were elite rugby players while the present subjects were well-trained. Training status has been identified as one of the factors which may affect the elicitation of PAP. Duthie et al. (2002) found that stronger individuals improved vertical jump performance to a greater degree than weaker individuals through means of peak force and peak power. Young et al. (1998) reported similar findings of stronger individuals having a greater vertical jump increase following a potentiation activity than weaker subjects.

Several past SSC fatigue studies have failed to elicit a PAP effect (Horita et al. 2003, Horita et al. 1999, Komi 2000). This may be due to the intensity of the fatigue protocols utilised. Past studies (Horita et al. 2003, Horita et al. 1999, Komi 2000) have typically involved submaximal fatigue protocols in comparison to the maximal fatigue protocol utilised in the present study and by Comyns et al. (2011). It has been reported that repeated sub-maximal contractions have no effect on power output (Chiu et al. 2003) and Parry et al. (2008) concluded that the absence of potentiation after sub-maximal contractions could be due to the intensity of the stimulus being too low. This may be due to the theory that fewer motor units are activated during submaximal contractions, in particular type II motor units which have been purported to have the greatest potential magnitude to elicit potentiation (Gullich and Schmidtbleicher 1996). Therefore, it seems that protocols to elicit acute, transient enhancements in performance require previous, recent muscular contractions of maximal or near maximal intensity within the muscles that will perform the ensuing activity. However, it must be noted that in the present study although the maximal SSC fatigue workout resulted in subsequent SSC activities being performed with a more effective SSC, no improvements in the performance outcome measure, height jumped, were observed and therefore it may be difficult to attribute the changes to PAP.
3. 6. Conclusions

The SSC is critically important in the performance of many strength activities, such as sprinting and jumping, and is the basis of plyometrics a commonly used method of resistance training. This study illustrated the immediate, debilitating effect of maximal SSC fatigue on the performance of subsequent SSC activities, through both the performance outcome measures and the performance biomechanics. This was observed through significant reductions in height jumped, GRF, RSI, $k_{vert}$ and a significant increase in CT in both the DJ and RBJ. These changes indicate that the effectiveness of the SSC was greatly reduced ultimately leading to a decrease in performance.

In addition to the dramatic decline in performance observed there were indications that the maximal fatigue workout elicited a PAP effect. During recovery there was a significant increase in GRF in the DJ in addition to large percentage improvements in RSI, $k_{vert}$ and CT in both the DJ and RBJ. Although not significant, there was a definite enhancement with the jumps being performed with more force, a stiffer leg spring action and a more efficient CT, variables which have been associated with faster stride frequencies and running velocities. The results suggest that SSC fatigue may have the potential to evoke a PAP effect whereby subsequent SSC activities can be performed with a more effective SSC. However, there was no improvement in the performance measure of height jumped and given the changes in the biomechanical variables measured it may not be possible to enhance performance.

3. 7. Summary

In summary maximal SSC fatigue has an immediate, negative effect on performance but can also evoke a PAP effect in strength trained athletes. It has been proposed by several researchers than power-trained and strength-trained athletes are better able to evoke PAP than endurance trained athletes (Smith and Fry 2007, Chiu et al. 2003, Paasuke et al. 2007). Therefore the aim of Chapter 4 is to investigate the effect of maximal SSC fatigue on endurance trained athletes.
Chapter 4. The Effect of Maximal Stretch Shortening Cycle Fatigue on The Biomechanical Properties of Endurance Trained Athletes.
4. 1. Abstract

The aim of this study was to investigate the acute effects of maximal stretch shortening cycle (SSC) fatigue on the biomechanical performance of endurance trained athletes. Nine endurance athletes performed four initial sets of drop jumps (DJ) and rebound jumps (RBJ) in order establish baseline levels of performance. A maximal SSC fatigue protocol was then performed on sledge and force plate apparatus followed immediately by DJ/RBJ sets at 15, 45, 120, 300 and 600 seconds post fatigue. Peak ground reaction force (GRF), ground contact time (CT), height jumped, reactive strength index (RSI) and leg spring stiffness \( k_{vert} \) were calculated for each DJ and RBJ. The fatigue protocol resulted in a significant reduction in jump height \( p < 0.001 \) and RSI \( p < 0.01 \) in the DJ and a significant reduction in GRF \( p < 0.01 \), height jumped \( p < 0.01 \) and RSI \( p < 0.01 \) in the RBJ. The results also indicated that the maximal fatigue workout elicited a post activation potentiation effect whereby during recovery there was a significant improvement in performance in \( k_{vert} \) \( p < 0.05 \) in the DJ and in GRF \( p < 0.001 \) and \( k_{vert} \) \( p < 0.05 \) in the RBJ.

4. 2. Introduction

Traditionally, endurance athletes have focused almost exclusively on aerobic training with the belief that endurance performance is determined by maximal oxygen uptake, fractional utilisation of \( \dot{V}O_{2max} \) and work economy (Bassett and Howley 2000). However, successful endurance performance also requires muscular strength and anaerobic power, necessary for attacking, pace changing and final sprints and may be the difference between success and failure in athletes of similar endurance capabilities (Tanaka and Swensen 1998). Due to the fact that endurance athletes spend the majority of training time enhancing aerobic endurance one of the main challenges is to simultaneously achieve and maintain strength and anaerobic gains. Therefore, many competitive endurance athletes now perform concurrent strength and endurance training.

Concurrent training involves the performance of both strength and endurance training in order to achieve multiple training goals and ultimately improve performance. The body of scientific literature concerning the impact of resistance training on endurance performance is equivocal. It has been reported that endurance adaptation may be compromised by
concurrent strength and endurance training (Gravelle and Blessing 2000, Kyrolainen and Komi 1995, Nelson et al. 1990). In contrast, it has also been demonstrated that strength training does not interfere with the development of maximal oxygen uptake (Hickson et al. 1988, Hickson et al. 1980, Marcink et al. 1991, McCarthy et al. 1995, Hunter et al. 1987, Sale et al. 1990, Mikkola et al. 2007, Dudley and Djamil 1985, Chtara et al. 2005). Indeed, it may be possible that if the training mode, volume and intensity are appropriate and the training is programmed correctly, endurance athletes can avoid any interference effect (Mikkola et al. 2007).

Strength and power training based on the SSC can also be termed plyometric training which involves a high intensity eccentric contraction immediately followed by a rapid and powerful concentric contraction (Malisoux et al. 2006). The importance of the SSC to sprinting and jumping performance is well established (Kubo et al. 2000, Kyrolainen and Komi 1995). It has however also been suggested that the ability of endurance athletes to utilise the SSC is important and therefore endurance athletes may benefit from the inclusion of SSC-related activities in their training (Harrison et al. 2004). SSC training, or plyometrics, has been reported to improve performance in well-trained endurance athletes (Paavolainen et al. 1999, Spurrs et al. 2003) with the training effects mostly attributed to neuromuscular adaptations, such as neural control, muscle force and elasticity and running mechanics, which were transferred into improved muscle power and economy. Paavolainen et al. (1999) suggested that it was the improvements in neuromuscular characteristics that may be converted into improved economy, whereby improved muscle stiffness as a result of strength training enhances work economy as improved muscle stiffness may develop the body’s ability to store and utilise elastic energy, thereby reducing the energy cost of the movement.

Adaptation to training stimuli involves fatigue in response to appropriate stress in order to facilitate the stress-response-adaptation process otherwise known as the general adaptation syndrome (Brooks et al. 2000). Fatigue, defined as the inability of a muscle to generate force is an important factor in exercise performance and functional capacity (Mileva et al. 2009) and provides the opportunity to investigate the acute effects of training on physiological and biomechanical properties of athletes. Traditionally research investigating fatigue in endurance athletes has focused on factors related to $\dot{V}O_{max}$, however Noakes (1988) proposed that endurance performance may be limited not only by factors related to
oxygen uptake but also by factors affected by the interaction of neuromuscular and anaerobic characteristics.

The SSC has been identified as a unique model to study neuromuscular fatigue (Komi 2000). Repeated SSC muscle action can lead to acute and long-term impairments in neuromuscular performance (Kuitunen et al. 2004) and can potentially modify the mechanical properties of the muscle tendon unit (Nicol et al. 2006). During SSC exercise leg spring stiffness must be well regulated to meet external loading condition and decreases in leg spring stiffness have been observed with SSC fatigue (Horita et al. 1996, Avela and Komi 1998, Comyns et al. 2011). The stiffness of the musculotendinous system may determine the body’s ability to store and utilise the energy associated with the SSC (Wilson et al. 1991) and Kuitunen et al. (2002) have suggested that effective use of elastic energy during the SSC requires a high level of stiffness with proper adjustment in stiffness allowing for the absorption of high impact forces and the storage of elastic energy during the active braking phase.

Recovery following SSC fatigue has been observed to occur in a bimodal fashion whereby a dramatic, immediate, post-fatigue decline is followed by a short-lasting recovery and subsequent secondary decline, which may peak on the 2nd or 3rd day post-fatigue (Horita et al. 1999, Komi 2000). Recently Comyns et al. (2011) observed that following the initial, dramatic decline there was in fact a post activation potentiation (PAP) effect whereby there were significant improvements in CT, RSI, GRF and leg spring stiffness approximately 300 seconds post fatigue and concluded that the maximal fatigue workout resulted in a more efficient SSC during subsequent SSC activities. However, Comyns et al. (2011) elicited PAP with strength trained athletes and it has been proposed that power-trained and strength-trained athletes are better able to evoke PAP than endurance trained athletes (Smith and Fry 2007, Chiu et al. 2003, Paasuke et al. 2007) due largely to fibre type distribution (Hamada et al. 2000b, Vandervoort and McComas 1983). In contrast Morana and Perrey (2009) concluded that the enhanced fatigue resistance of endurance athletes does in fact allow the PAP effect to prevail longer over the fatigue effect associated with PAP.

There is a common misconception that the role of speed-strength capabilities is minor for endurance athletes with several factors of force production playing an important role in performance including muscle contractility, ability to utilise the potential energy stored in
muscle structures during the pre-stretch, force-time characteristics of the muscles and reactive ability (Tanaka and Swensen 1998, Noakes 1988, Noakes 2000, Paavolainen et al. 1999). The adaptation to explosive strength training in endurance athletes has been investigated with equivocal findings but it is also prudent to rigorously investigate the acute impact of maximal SSC fatigue on endurance athletes and the recovery process which follows. To date few SSC fatigue studies have investigated the acute effects of maximal SSC fatigue (Strojnik and Komi 1998, Comyns et al. 2011) with no reports of the acute effects of maximal SSC fatigue on endurance athletes. Consequently the aim of this study was to examine the acute effects of a maximal SSC fatigue workout on endurance athletes. The study also aimed to investigate the recovery of endurance athletes following maximal SSC fatigue in an attempt to identify if PAP could be elicited.

4.3. Method

4.3.1. Subjects

Nine highly trained endurance trained athletes (rowers) participated in this study. All participants had a minimum of 5 years endurance training. Six of the athletes were high level national athletes while three were international athletes. The physical characteristics of the athletes are presented in Table 4.1. All participants were fully informed verbally and in writing of the requirements, and the potential risks and benefits of participating in the study (Appendix B). An informed consent form (Appendix B) and pre-test questionnaire (Appendix C) were completed by each participant. All participants were required to undertake no more than 30 minutes light training and to refrain from any strength training or plyometrics during the 24 hours prior to the test. All experimental procedures were approved by the University of Limerick Ethics Committee. The methodology of Chapter 3 was replicated in this study and therefore the following sections were not duplicated; research design, instrumentation, test procedure, data collection, calculation of dependent variables and statistical analyses.

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>Mass (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>25 ± 6</td>
<td>187.7 ± 9.1</td>
<td>82.2 ± 11.4</td>
</tr>
</tbody>
</table>

Data expressed as mean ± SD
4. 3. 2. Research Design
Refer to Chapter 3 section 3.3.2

4. 3. 3. Instrumentation
Refer to Chapter 3 section 3.3.3

4. 3. 4. Test Procedure
Refer to Chapter 3 section 3.3.4

4. 3. 5. Data Collection
Refer to Chapter 3 section 3.3.5

4. 3. 6. Calculation of the Dependent Variables
Refer to Chapter 3 section 3.3.6

4. 3. 7. Statistical Analyses
Refer to Chapter 3 section 3.3.7

4. 4. Results

Table 4.2 presents the performance results of the maximal SSC fatigue workout.

Table 4.2: Performance scores of fatigue workout

<table>
<thead>
<tr>
<th>No. of jumps</th>
<th>Duration of workout(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>65 ± 29.3</td>
<td>84.6 ± 32.8</td>
</tr>
</tbody>
</table>

Data expressed as mean ± SD

The remaining results of this study are presented in two sections. The results of the DJ are presented in section 4.4.1 and the results of the RBJ are presented in section 4.4.2. Table 4.4 presents the baseline scores (mean (± SD)) for each of the dependent variables for both the DJ and RBJ jump.
Table 4.3: Mean baseline DJ and RBJ values for each dependent variable

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Drop Jump</th>
<th>Rebound Jump</th>
</tr>
</thead>
<tbody>
<tr>
<td>GRF (N)</td>
<td>1491.456 ± 296.269</td>
<td>1410.74 ± 300.375</td>
</tr>
<tr>
<td>Height jumped (cm)</td>
<td>33.44 ± 5.67</td>
<td>33.24 ± 5.25</td>
</tr>
<tr>
<td>CT (s)</td>
<td>0.62 ± 0.134</td>
<td>0.62 ± 0.134</td>
</tr>
<tr>
<td>RSI</td>
<td>0.588 ± 0.227</td>
<td>0.579 ± 0.227</td>
</tr>
<tr>
<td>$K_{\text{vert}}$ (kN.m$^{-1}$)</td>
<td>5.382 ± 2.306</td>
<td>6.187 ± 3.323</td>
</tr>
</tbody>
</table>

Data expressed as mean ± SD

The graphs in section 4.4.1 (DJ) and 4.4.2 (RBJ) illustrate the effect of maximal SSC fatigue on endurance trained athletes with respect to GRF, CT, height jumped, RSI and $K_{\text{vert}}$. The effect of fatigue on each dependent variable is represented by an individual graph, with each x-axis representing the baseline scores. The graphs display the change from the baseline scores to the minimum and maximum scores achieved in the post-fatigue recovery intervals. The minimum and maximum scores across all 5 recovery intervals (15, 45,120, 300 and 600 seconds) were selected in an attempt to identify the effects of fatigue and potentiation without the interference of individual variation across recovery time. In each graph the mean baseline score was subtracted from the minimum and maximum score achieved in order to show a representation of the actual change that occurred. The actual percentage changes and effect sizes of maximal fatigue on DJ and RBJ performance are also included.

4.4.1. Drop Jump Results

The effect of maximal SSC fatigue on peak GRF are illustrated in figure 4.1. Following the maximal fatigue workout there was an immediate reduction in GRF, however this reduction was not significant ($p = 0.06$). There was also an increase in GRF above baseline during the recovery intervals. This increase was not significant ($p = 0.075$).
Figure 4.1: Mean (± 95% CI) GRF difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue.

Figure 4.2 illustrates the results for height jumped. There was a significant reduction in performance immediately after the fatigue workout (p = 0.001). The height jumped recovered fully during the recovery intervals but did not show a significant improvement.

Figure 4.2: Mean (± 95% CI) height jumped difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. ***p < 0.001

The CT results are presented in figure 4.3. CT increased immediately following the maximal fatigue workout, however, the increase was not significant (p = 0.071). This was
followed by a reduction in CT during recovery but this improvement was not significant \((p = 0.078)\).

**Figure 4.3**: Mean (± 95% CI) CT difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue.

Figure 4.4 illustrates the effect of maximal fatigue on RSI. The results revealed a significant reduction in RSI immediately following the fatigue workout \((p = 0.007)\). During recovery RSI recovered to a level above baseline values, but this subsequent improvement in RSI was not significant \((p = 0.243)\).

**Figure 4.4**: Mean (± 95% CI) RSI difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. **\(p < 0.01\)**
The effect of the maximal fatigue workout on $k_{\text{vert}}$ is illustrated in figure 4.5. Despite there being a large decrease in $k_{\text{vert}}$ immediately following maximal SSC fatigue, the reduction was not significant ($p = 0.082$). There was, however, a significant increase in $k_{\text{vert}}$ above baseline values during the recovery ($p = 0.021$).

![Figure 4.5: Mean (± 95% CI) $k_{\text{vert}}$ difference between the baseline DJ and the minimum and maximum jumps post maximal fatigue. *p < 0.05](image)

The actual percentage changes in performance for each of the dependent variables during the DJ can be seen in Table 4.4. This percentage change represents the change from the mean baseline value to the minimum and maximum values achieved during the recovery intervals.

**Table 4.4: Percentage change for the DJ dependent variables from baseline values to minimum and maximum values achieved during recovery.**

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>% Change Baseline → Minimum</th>
<th>% Change Baseline → Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>GRF</td>
<td>5.3%</td>
<td>7.7%</td>
</tr>
<tr>
<td>Height Jumped</td>
<td>12.8%***</td>
<td>1.6%</td>
</tr>
<tr>
<td>CT</td>
<td>8.1%</td>
<td>6.1%</td>
</tr>
<tr>
<td>RSI</td>
<td>19.3%**</td>
<td>6.7%</td>
</tr>
<tr>
<td>$K_{\text{vert}}$</td>
<td>16.1%</td>
<td>22.9%*</td>
</tr>
</tbody>
</table>

***p < 0.001; **p < 0.01; *p < 0.05
The $\eta_p^2$ values for the DJ can be seen in Table 4.5. Measures of effect size in ANOVA are measures of the degree of association between an effect and the dependent variable (i.e. the effect of maximal fatigue workout on each of the dependent variables). If the value of the measure of association is squared it can be interpreted as the proportion of variance in the dependent variable that is attributable to each affect hence $\eta_p^2$ is used.

Table 4.5: $\eta_p^2$ values for the DJ dependent variables and classification of the magnitude of the effect size according to Hopkins (2002).

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Baseline $\rightarrow$ Minimum</th>
<th>Baseline $\rightarrow$ Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>GRF</td>
<td>0.374 : medium</td>
<td>0.343 : medium</td>
</tr>
<tr>
<td>Height Jumped</td>
<td>0.755 : large ***</td>
<td>0.012 : trivial</td>
</tr>
<tr>
<td>CT</td>
<td>0.352 : medium</td>
<td>0.337 : medium</td>
</tr>
<tr>
<td>RSI</td>
<td>0.620 : large **</td>
<td>0.166 : small</td>
</tr>
<tr>
<td>$K_{vert}$</td>
<td>0.331 : medium</td>
<td>0.504 : medium*</td>
</tr>
</tbody>
</table>

***$p < 0.001$; **$p < 0.01$; *$p < 0.05$

4. 4. 2. Rebound Jump Results

The effect of maximal SSC fatigue on GRF during the RBJ is illustrated in figure 4.6. Immediately following maximal SSC fatigue there was a significant reduction in GRF ($p = 0.007$) followed by a significant improvement in GRF during recovery ($p = 0.006$).

Figure 4.6: Mean (± 95% CI) GRF difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. ***$p < 0.001$; **$p < 0.01$
The results of the effect of maximal fatigue on height jumped are presented in figure 4.7. There was a significant reduction in performance immediately following the fatigue workout \( (p = 0.006) \). During recovery phases height jumped returned to baseline levels, with no significant improvement \( (p = 0.956) \).

**Figure 4.7:** Mean (± 95% CI) height jumped difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. **p < 0.01

Figure 4.8 presents the results of CT following maximal SSC fatigue. There fatigue workout resulted in an increase in CT which was not significant \( (p = 0.085) \). During the recovery, while there was a trend towards an improvement in CT the improvement was not significant \( (p = 0.122) \).

**Figure 4.8:** Mean (± 95% CI) CT difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue.
The results of RSI are present in figure 4.9. RSI was significantly reduced immediately following maximal SSC fatigue ($p = 0.005$). During recovery despite an improvement in RSI above baseline values being observed, this improvement was not significant ($p = 0.176$).

![Figure 4.9: Mean (± 95% CI) RSI difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. **$p < 0.01$](image)

Figure 4.9 illustrates the effect of maximal SSC fatigue on $k_{\text{vert}}$. Maximal SSC fatigue did not have a significant effect on $k_{\text{vert}}$ immediately following SSC fatigue ($p = 0.456$). $K_{\text{vert}}$ however was significantly improved during recovery ($p = 0.049$).

![Figure 4.10: Mean (± 95% CI) $k_{\text{vert}}$ difference between the baseline RBJ and the minimum and maximum jumps post maximal fatigue. *$p < 0.05$](image)
The actual percentage changes for each dependent variable during the RBJ can be seen in Table 4.6. This percentage change represents the change from the mean baseline value to the minimum and maximum values achieved during the recovery intervals.

Table 4.6: Percentage change for the RBJ dependent variables from baseline values to minimum and maximum values achieved during recovery.

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>% Change Baseline → Minimum</th>
<th>% Change Baseline → Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>GRF</td>
<td>7.3%</td>
<td>8.4%</td>
</tr>
<tr>
<td>Height Jumped</td>
<td>10.4%</td>
<td>0.3%</td>
</tr>
<tr>
<td>CT</td>
<td>6.6%</td>
<td>6.7%</td>
</tr>
<tr>
<td>RSI</td>
<td>13.5%</td>
<td>3.9%</td>
</tr>
<tr>
<td>K&lt;sub&gt;vert&lt;/sub&gt;</td>
<td>6.5%</td>
<td>23.7%</td>
</tr>
</tbody>
</table>

**p < 0.01; *p < 0.05

The η<sup>p2</sup> values for the RBJ can be seen in Table 4.7. The medium and large effect sizes indicate that the fatigue workout resulted in considerable changes to the dependent variables.

Table 4.7: η<sup>p2</sup> values for the RBJ dependent variables and classification of the magnitude of the effect size according to Hopkins (2002).

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Baseline → Minimum</th>
<th>Baseline → Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>GRF</td>
<td>0.622 : large**</td>
<td>0.757 : large***</td>
</tr>
<tr>
<td>Height Jumped</td>
<td>0.630 : large**</td>
<td>0.000 : trivial</td>
</tr>
<tr>
<td>CT</td>
<td>0.325 : medium</td>
<td>0.272 : medium</td>
</tr>
<tr>
<td>RSI</td>
<td>0.644 : large**</td>
<td>0.216 : small</td>
</tr>
<tr>
<td>K&lt;sub&gt;vert&lt;/sub&gt;</td>
<td>0.071 : small</td>
<td>0.402 : medium*</td>
</tr>
</tbody>
</table>

***p < 0.001; **p < 0.01; *p < 0.05

4. 5. Discussion

The aim of this study was to investigate the acute effects of maximal SSC fatigue on endurance athletes. It is well documented that the main physiological prerequisites for
success in endurance events include $\dot{V}O_{2\text{max}}$, exercise economy, fractional utilisation of $\dot{V}O_{2\text{max}}$ and lactate threshold (Jones 2006). It has however been suggested that other factors involving the interaction of neuromuscular and anaerobic characteristic may also limit endurance performance (Noakes 1988, Paavolainen et al. 1999) and the ability of endurance athletes to resist fatigue is essential for successful endurance performance. The results revealed that the maximal SSC fatigue workout had a significant impact on the effectiveness of the SSC with significant decreases in height jumped and RSI in the DJ and significant decreases in GRF, height jumped and RSI in the RBJ. In addition, the study also indicated that the endurance athletes experienced a post-activation potentiation (PAP) effect during recovery from maximal SSC fatigue, observed through significant increases in $k_{\text{vec}}$ in the DJ and GRF and $k_{\text{vert}}$ in the RBJ.

The maximal SSC fatigue workout had a significant impact on subsequent performance as observed through the significant decrease in height jumped in the DJ ($p < 0.001$) and RBJ ($p < 0.01$) immediately post fatigue. The results indicate that fatigue could not be compensated for in order to maintain performance. It is possible that this decrement in performance resulted from an impairment in neuromuscular performance whereby there was a reduction in stretch reflex sensitivity due to reduced tolerance to impact following maximal fatigue or there may have been a loss in the effective reutilisation of elastic energy due to a longer amortization phase. Komi and Nicol (2000) concluded from the results of several studies that both short-term and long-duration SSC fatiguing exercise leads to a deterioration in neuromuscular function and has a considerable influence on muscle mechanics, leg spring stiffness and reflex intervention.

Performance, in several sports, is determined by both the ability to generate force and the speed of force production. Similarly, prolonged endurance activities, which undoubtedly require specialized metabolic capacity, may also require high strength-endurance capacities and often involve a series of brief, explosive spikes in power output. It has been demonstrated that endurance athletes with the most powerful muscles are more likely to succeed and that the power and force production may be a major factor separating high and low ranked endurance athletes (Noakes 1988, Tanaka et al. 1993, Bulbulian et al. 1986, Stone et al. 2008). Successful endurance performance also requires the ability to resist fatigue in force production and to repeatedly produce adequate force and power over a period of time. During SSC’s the force developed is largely dependent on the eccentric
phase and SSC fatigue can cause considerable decreases in force production and ground reaction forces (Nicol et al. 1991a, Avela et al. 1999, Comyns et al. 2011). The results of the present study indicate that a maximal SSC fatigue workout altered force production capabilities with a significant decrease in GRF ($p = 0.007; 7.3\%$) in the RBJ and a $5.3\%$ decrease in the DJ ($p = 0.075$). Comyns et al. (2011) also observe a significant decrease in GRF however, this change was observed in both the DJ and RBJ. This conflict in results may well be expected given individuals with higher initial power levels usually experience the greatest power decrements during fatiguing exercise (Bishop et al. 2003, Bishop et al. 2004, Hamilton et al. 1991, Mendez-Villanueva et al. 2008) and Comyns et al. (2011) made the observation with elite rugby players with higher initial force levels than the endurance athletes in the present study.

RSI was also significantly affected by the maximal SSC fatigue workout. RSI is an index derived from the height jumped and the time spent developing the forces required to make the jump and has been described as an individual’s ability to change quickly from an eccentric to concentric contraction (Young 1995). The effectiveness is partly influenced by the time lag between the completion of the stretch and the initiation of the concentric contraction. In order to optimise the benefits of the SSC a short transition phase between the eccentric to concentric phase is necessary. If the time interval between stretching and shortening is too long, the stored elastic energy can be wasted and the mechanical efficiency of the concentric contraction will decrease (Kyrolainen and Komi 1995). It has been noted that less economical distance runners possessed a more compliant running style with a longer transition from the eccentric to concentric phase (Heise and Martin 1998). Paavolainen et al. (1999) also observed that shortening of the length of the transition phase and ground contact time occurred concurrently with a reduction in the energy cost of running. While RSI has recently been used as a practical means of quantifying plyometric or SSC performance (Comyns et al. 2011, Flanagan and Harrison 2007) there is a paucity of research regarding the effect of fatigue on RSI. Comyns et al. (2011) found that SSC fatigue had a significant impact on RSI, reporting a significant decrease in RSI in both the DJ ($p < 0.001$) and RBJ ($p < 0.001$). In the present study similar results were revealed for endurance athletes with significant drops in RSI in the DJ ($p < 0.01; 19.3\%$) and RBJ ($p < 0.01; 13.5\%$). The results highlight the impact the maximal SSC fatigue workout had on the ability of the athletes to effectively change from an eccentric to concentric contraction.
indicating that stored elastic energy may have been wasted ultimately leading to a decrease in the mechanical efficiency of the concentric phase.

Maximal SSC fatigue did not have an immediate significant effect on CT or $k_{vert}$. These results are inconsistent with previous SSC fatigue studies where SSC fatigue has commonly been characterized by an increase in contact time (Gollhofer et al. 1987b, Avela and Komi 1998, Horita et al. 2003, Comyns et al. 2011). Comyns et al. (2011) recently investigated the effect of maximal SSC fatigue on strength trained athletes and also reported a significant increase in CT in both the DJ ($p < 0.001$) and RBJ ($p < 0.01$) following maximal SSC fatigue. CT corresponds to the amortization phase of the SSC and a short amortization phase is required for the subsequent concentric contraction to elicit the advantages of both the stored elastic energy and stretch reflex (Schmidtbleicher 1992). The conflicting results of this study and previous SSC fatigue studies may be due to the ability of the endurance athletes to resist the fatiguing effects of the maximal workout to a greater degree than the strength trained athletes with Comyns et al. (2011). It may also be due to the fact that the CT was noticeably larger for the participants in the present study and therefore the smaller percentage changes observed with the endurance athletes may be due to the larger baseline CT observed. This difference in CT between strength and endurance athletes would be expected and may indicate that the two groups of athletes use different ground CT strategies when jumping. In addition, previous SSC fatigue studies involving endurance athletes have investigated long-duration or submaximal SSC fatigue (Avela and Komi 1998, Nicol et al. 1991a, Nicol et al. 1991b). This present study is the first, to our knowledge, to investigate the effect of maximal SSC fatigue on endurance athletes. However, it is worth noting that although there was not a significant change in CT in the DJ ($p = 0.071$) or RBJ ($p = 0.078$) there was a trend towards an increase in CT with changes of 8.1% and 6.7% observed in the DJ and RBJ respectively.

SSC fatigue is reported to result in an alteration in $k_{vert}$ (Nicol et al. 1991a, Harrison and Gaffney 2004, Comyns et al. 2011) and Komi et al. (1986) observed that with the progress of fatigue, there is a change in stiffness characteristics. Despite a reduction in $k_{vert}$ in the present study it was not significantly altered in the DJ ($p = 0.082$) or RBJ ($p = 0.456$) following maximal SSC fatigue. The control of $k_{vert}$ has been associated with the performance of SSC activities and the ability to control $k_{vert}$ allows for a more effective storage and utilisation of elastic energy thereby maximising performance (Saunders et al.
2006). Despite the fact that the decrease in $k_{\text{ven}}$ in the present study was not significant, the results do suggest it is possible that there was reduced stretch reflex sensitivity, as an association between reduced stretch reflex sensitivity and decreased stiffness following highly fatiguing SSC exercise has been established (Avela and Komi 1998) and several studies have supported the view that muscle damage, as apparent after SSC fatigue, alters stiffness regulation through a change in the afferent input from the muscle (Millet et al. 2003, Girard et al. 2008, Avela et al. 1999).

The dramatic decline in performance following the maximal SSC fatigue protocol conforms to the immediate effects of SSC fatigue observed in previous SSC fatigue studies (Horita et al. 1999, Komi 2000). This initial dramatic decline in performance has typically been demonstrated to be followed by a short term recovery to baseline levels and a secondary reduction with a longer lasting recovery. However, the results of this study revealed that the initial dramatic decline in DJ and RBJ performance was in fact followed by a gradual improvement in performance, which for some variables represented an enhancement in performance which was superior to baseline values achieved prior to the fatigue workout.

Enhancement in performance due to prior contractile activity has been termed post-activation potentiation (PAP). Performance enhancements can manifest in the form of applied performance (e.g. jumping, throwing, squatting), contractile property output (peak force, peak twitch force, rate of force development, contraction time) and/or neural function (neural conduction velocity, motor unit recruitment patterns). However, research investigating the ability of endurance athletes to elicit PAP is equivocal. Muscle fibre type is one of the most important muscular characteristics affecting the magnitude of PAP. It has been demonstrated that individuals with a greater percentage and cross-sectional area of type II muscle fibres can benefit to a greater degree (Hamada et al. 2000b, Vandervoort and McComas 1983) and the histochemical differences between strength and endurance athletes has long been established with endurance athletes typically possessing more type I than type II fibres. It is postulated that type II fibres elicit a greater PAP effect due to the fact that type II fibres undergo greater myosin light chain phosphorylation in addition to type II fibres experiencing the greatest neural excitation associated with resistance training (Hamada et al. 2000b, Sale 2004). It has been suggested that individuals with greater type II fibre expression exhibit greater and longer-lasting potentiation effects (Hamada et al.
but endurance athletes have demonstrated the ability to elicit a PAP effect (Hamada et al. 2000a, Morana and Perrey 2009, Boullosa and Tuimil 2009). Hamada et al. (2000a) proposed that the enhanced fatigue resistance of endurance athletes’ muscles and the ability of endurance training to increase the maximum shortening velocity of type I fibres may facilitate potentiation in endurance athletes, despite a low percentage of type II fibres.

PAP coexists with fatigue with optimal performance occurring when fatigue has subsided but the potentiation effect still exists (Hodgson et al. 2005). Hodgson et al. (2005) advocate that there is an optimal time when the muscle has recovered from fatigue and PAP prevails but have cautioned that this “optimal time” appears to be variable among individuals. Therefore any PAP effect in group design studies may possibly be masked, and indeed it may not exist at all. For this reason, in this present study, each participant’s best post-fatigue score for each dependent variable, irrespective of time, was selected allowing for identification of any PAP effect within the group as it facilitated individual variation. This method of analysis revealed some evidence of a PAP effect. During recovery there was a significant increase in $k_{\text{vert}}$ ($p < 0.05$) in the DJ and in GRF ($p < 0.001$) and $k_{\text{vert}}$ ($p < 0.05$) in the RBJ. While there was no performance outcome improvement, these results indicate that the maximal fatigue workout altered the way the muscle tendon unit behaved, resulting in a more effective SSC behaviour. In particular, the control of $k_{\text{vert}}$ plays an important role in exploiting the full benefits of the SSC and increased SSC utilisation has been observed with increases in $k_{\text{vert}}$ (Harrison and Gaffney 2004).

Typically, SSC fatigue studies have reported short-term recoveries involving either partial recoveries or complete recoveries (Nicol et al. 2006, Horita et al. 1999, Horita et al. 2003) but to date only one published study has identified a PAP effect similar to that evident in the present study following SSC fatigue (Comyns et al. 2011). Comyns et al. (2011) reported significant changes in the biomechanical process of jumping in strength trained athletes with significant improvements observed in RSI, GRF and $k_{\text{vert}}$ in both the DJ and the RBJ and in CT in the DJ. Comyns et al. (2011) concluded that the results indicated an enhancement in the effectiveness of the SSC and credited the PAP effect to the intensity of the maximal SSC fatigue protocol utilised. The results of the present study established that well-trained endurance athletes may also have the capacity to elicit a PAP effect through maximal SSC fatigue, similar in intensity to that used by Comyns et al. (2011). However, no
significant improvement was observed in the true performance measure, height jumped and therefore it may difficult to attribute the changes in the biomechanical variables to PAP alone. It may be possible that given the alterations observed in the biomechanical variables, GRF and $k_{\text{vert}}$, an enhancement in performance may be impossible.

4. 6. Conclusions

Concurrent strength and endurance training has the potential to evoke improvements in performance given the correct training mode, volume and intensity with improvements in force, economy and neuromuscular performance being observed in well-trained endurance athletes (Hoff et al. 1999, Spurrs et al. 2003, Osteras et al. 2002, Paavolainen et al. 1999, Mikkola et al. 2007, Paavolainen et al. 1991). The SSC is the basis for plyometrics, a popular method of strength training and the aim of this study was to investigate the acute effects of maximal SSC fatigue on endurance athletes. The exhausting SSC protocol induced a clear and immediate decrease in performance in addition to alterations in the biomechanical process of the jumps suggesting there was a decrease in neuromuscular performance through a decrease in stretch reflex sensitivity and a loss in the effective utilisation of stored elastic energy ultimately indicating a loss in SSC effectiveness.

The maximal SSC fatigue also resulted in a significant increase in $k_{\text{vert}}$ in both the DJ and RBJ and a significant increase in GRF in the RBJ. These changes facilitate a more effective storage and utilisation of elastic energy, reducing the energy cost of movement and potentially improving work economy and also suggest an improvement in stretch reflex sensitivity.

From a practical perspective this study demonstrates that well-trained endurance athletes may have the potential to elicit a PAP effect from maximal SSC fatigue. However, caution should be taken to individualise training sessions as the time course of PAP varies greatly between individuals and may in fact not be evident in some individuals (Gullich and Schmidtbleicher 1996). Indeed no improvement was observed in the performance outcome measure, height jumped. Further research is required to investigate if the PAP effect can be manipulated to produce chronic adaptations in endurance athletes.
4. 7. Summary

In summary maximal SSC fatigue has an immediate, debilitating effect on the performance of endurance athletes but it also has the potential to elicit a PAP effect. While resistance training, such as plyometrics, has the potential to improve performance, endurance athletes may also benefit from other methods of training. Therefore the aim of Chapter 5 is to identify the physiological parameters required for successful rowing performance in order to determine alternative training methods to elicit improvements in performance.
Chapter 5. The Physiological Determinants of 2000 m Rowing Ergometer Performance.
5. 1. Abstract

The aim of this study was to determine the relationship between 2000 m rowing ergometer performance and selected physiological parameters in well-trained rowers. 18 male and 9 female well-trained rowers participated in this study. A 2000 m time trial was performed, from which mean rowing velocity (m.s\(^{-1}\)), the performance outcome, was calculated. On a separate occasion each subject completed an incremental step test, consisting of seven discontinuous 4 minute bouts at fixed exercise intensities, which was used to determine maximum oxygen uptake (\(\dot{V}O_{2\text{max}}\)), power at \(\dot{V}O_{2\text{max}}\) (\(\dot{W}\dot{V}O_{2\text{max}}\)), peak power output (PPO), economy of movement (ECON), the power output at lactate threshold (\(\dot{W}_{LT}\)) and anaerobic threshold (\(\dot{W}_{AT}\)), maximum heart rate (HR\(_{\text{max}}\)) peak lactate (peak La), the power outputs associated with blood lactate levels of 2 (\(\dot{W}_{2\text{mmol/l}}\)) and 4 mmol.l\(^{-1}\) (\(\dot{W}_{4\text{mmol/l}}\)) and the oxygen consumption at lactate threshold (\(\dot{V}O_{2LT}\)) and anaerobic threshold (\(\dot{V}O_{2AT}\)). A seven stroke power test was used to assess maximal power (\(\dot{W}_{\text{max}}\)) and maximal force (\(F_{\text{max}}\)). Percentage body fat (BF\%), percentage of lean body mass (Lean\%) and percentage of fat free mass (FF\%) were estimated using the skinfold method and DXA. Simple linear regression identified \(\dot{V}O_{2\text{max}}\) as the strongest correlate of performance (\(r = 0.98, p < 0.001\)). Strong correlations were also observed for PPO (\(r = 0.93, p < 0.001\)), \(\dot{W}_{\text{max}}\) (\(r = 0.93, p < 0.001\)), \(\dot{W}_{4\text{mmol/l}}\) (\(r = 0.91, p < 0.001\)), \(\dot{V}O_{2\text{max}}\) (\(r = 0.84, p < 0.001\)), and \(\dot{W}_{2\text{mmol/l}}\) (\(r = 0.80, p < 0.001\)). A backward, stepwise multiple regression indicated that \(\dot{V}O_{2\text{max}}\) was the best single predictor of 2000 m performance with the model incorporating \(\dot{V}O_{2\text{max}}\) explaining 96.5% of the variance in 2000 m performance. \(\dot{V}O_{2\text{max}}\) was also identified as the variable with most influence on rowing performance (\(\beta = .982\)). The results suggest that in well-trained rowers, 2000 m rowing performance is almost exclusively determined by \(\dot{V}O_{2\text{max}}\).

5. 2. Introduction

The physiological capacities of rowers have been reported to be among the highest recorded (Hagerman et al. 1979). There are, however, numerous difficulties associated with physiological assessment during on-water rowing. Therefore, physiological assessment and monitoring of rowers typically involves simulating rowing on a rowing ergometer. Despite rowing ergometer performance not requiring the same skill level as on-water rowing it has
been shown to simulate the biomechanical and metabolic demands of on-water rowing (Lamb 1989, Hagerman 1984, Hagerman et al. 1979, Smith and Spinks 1995).

Rowing performance over the international standard distance of 2000 m involves a near maximal energy delivery for the duration of a race and is dependent on both the aerobic and anaerobic pathways. Traditionally the relative contribution from aerobic and anaerobic energy pathways was reported to be 70-75% and 25-30% respectively (Hagerman 1984, Secher 1990, Hagerman et al. 1978). However, more recent studies estimate the contribution from aerobic metabolism to be as high as 84-88% (Pripstein et al. 1999, Russell et al. 1998, de Campos Mello et al. 2009), indicating that elite rowers have a highly developed aerobic capacity facilitating the ability to work at a very high percentage (96-98%) of their maximal aerobic capacity (Hagerman et al. 1979).

Rowing has long been considered one of the most demanding endurance sports, with exceedingly high demands placed on the aerobic and anaerobic systems during a 2000 m race (McKenzie and Rhodes 1982, Hagerman 1984). It differs from other endurance sports with similar energy component relationships due to the fact that rowers are required to generate much higher forces for the duration of a 2000 m race which typically lasts between five and a half and eight minutes depending on boat type and conditions (Babraj and Volianitis 2007, McNeely 2011b). Research into the physiological predictors of rowing performance typically identified $\dot{V}O_{2max}$ as the best predictor of 2000 m rowing performance, accounting for 49-81% of performance variance (Cosgrove et al. 1999, Ingham et al. 2002, Kramer et al. 1994). Elite rowers have been observed to produce some of the highest absolute $\dot{V}O_{2max}$ values ever recorded with several researchers reporting values of over 6 l\cdot min$^{-1}$ in male elite rowers (Lacour et al. 2009, Hagerman 1984, Di Prampero et al. 1971, Mikulic 2011, Godfrey et al. 2005, Mikulic 2012). $\dot{V}O_{2max}$ has consistently been correlated with rowing performance, however, it has been noted that $\dot{V}O_{2max}$ does not explain all of the variance in rowing performance and several other predictors of rowing performance have emerged including power at $\dot{V}O_{2max}$, peak power output, $\dot{V}O_2$ at lactate threshold, percentage of slow twitch fibres, body mass, peak blood lactate, strength and power output at a blood lactate concentration of 4 mmol\cdot l$^{-1}$ (Ingham et al. 2002, Nevill et al. 2011, Secher et al. 1983, Kramer et al. 1994, Cosgrove et al. 1999, Womack et al. 1996, Warrington et al. 2003, Jensen et al. 1996). Indeed, Nevill et al. (2011) proposed rowing performance determinant models should consider a submaximal
parameter, a maximal parameter and an indicator of anaerobic/maximal power capability in order to successfully identify the key determinants of 2000 m performance. The aim of this study therefore was to examine the relationship between submaximal, maximal and anaerobic/maximal power parameters and 2000 m rowing performance.

5. 3. Method

5. 3. 1. Subjects

Twenty seven national level rowers, 18 male and 9 female, participated in this study. The physical characteristics of the rowers, grouped according to gender, can be seen in Table 5.1. All participants were proficient in both sculling and sweep rowing. All participants were fully informed verbally and in writing of the requirements and potential risks and benefits of participating (Appendix D). An informed consent form (Appendix D) and pre-test questionnaire (Appendix C and Appendix D) were completed by each participant. All participants were given a pre-test protocol (Appendix D) which was required to be followed. All experimental procedures were approved by the University of Limerick Ethics Committee.

Table 5.1: Physical characteristics of the subjects

<table>
<thead>
<tr>
<th></th>
<th>All Subjects</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n=27)</td>
<td>(n=18)</td>
<td>(n=9)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>22 ± 3</td>
<td>21 ± 3</td>
<td>22 ± 4</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>184.1 ± 8.8</td>
<td>188.3 ± 5.8</td>
<td>175.6 ± 7.9</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>84.9 ± 12.5</td>
<td>88.1 ± 11.4</td>
<td>78.5 ± 12.8</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>21.2 ± 6.9</td>
<td>17.4 ± 3.7</td>
<td>29.6 ± 4.6</td>
</tr>
</tbody>
</table>

Data expressed as mean ± SD

5. 3. 2. Research Design

The athletes attended the laboratory on three occasions over a 6 day period. Participants were requested to follow strict protocols in the 24 hours prior to any test days which included maintaining a normal diet, optimizing hydration and refraining from alcohol. Participants were also required to undertake no more than 30 minutes light training during
the 24 hours prior to the test. A training and food diary for the 24 hours prior to all testing days was requested from all participants. The following is a schematic summary of testing design (figure 5.1).

Figure 5.1: Schematic representation of the experimental design

5. 3. 2. 1. Body Mass and Stature

Body mass was assessed in minimal clothing using a Seca 799 digital column scale (Seca, Hamburg, Germany) and height was measured using a Seca 217 stadiometer (Seca, Hamburg, Germany).

5. 3. 2. 2. Body Composition

Body composition was measured using skinfold thickness and dual-energy X-ray absorptiometry (DXA). Skinfold thickness was measured in accordance with the International Standards for Anthropometric Assessment (ISAK) protocol. Repeated skinfold measurements were taken, using a Harpenden skinfold callipers (Baty International, West Sussex, UK), at seven sites: biceps, triceps, subscapular, supraspinale, abdominal, frontal thigh and medial calf. The sum of these seven sites was then calculated. The DXA was used to calculate percentage body fat (BF%), percentage lean body mass (lean%) and percentage fat free mass (FF%). A Lunar iDXA™ scanner (GE Healthcare, Chalfont St Giles, Bucks., UK) with enCORE™ 2011 v.13.6 software was used to capture total body composition scans. Calibration with use of a phantom spine was carried out daily.
5. 3. 2. 3. 2000 m Time Trial

The 2000 m time trial was performed on the first day of testing. The test-retest reliability of a 2000 m time trial on the Concept II model C has previously been examined with well-trained rowers, with a coefficient of variation of 0.6% being reported (Schabort et al. 1999). All participants performed a self-selected warm-up which was the normal pre-performance warm-up of each participant and was a minimum of 15 minutes in duration. An earlobe blood sample (5µL) was taken to determine blood lactate concentration (Lactate Pro, Arkray Factory Inc, Shiga, Japan) pre and post warm-up and 5 minutes post the 2000 m time trial. Heart rate was measured continuously (RS400, Polar Electro OY Finland) during the performance.

5. 3. 2. 4. Incremental Step Test

The incremental step test was completed a minimum of 48 hours after the 2000 m time trial and all athletes were instructed to follow strict pre-test protocols (Appendix D). The test was performed according to the Australian physiological assessment of rowing guidelines (Gore, 2000) (Appendix F) and was used to determine maximum oxygen uptake ($\dot{V}O_{2\text{max}}$), power at $\dot{V}O_{2\text{max}}$ ($\dot{W}V_{O_{2\text{max}}}$), 4 minute all-out power output (PPO), oxygen cost of movement (ECON), the power output at lactate threshold ($W_{LT}$) and anaerobic threshold ($W_{AT}$), maximum heart rate, peak lactate (peak La), and the power outputs associated with blood lactate levels of 2 ($W_{2\text{mmol/L}}$) and 4 mmol.L$^{-1}$ ($W_{4\text{mmol/L}}$).

On completion of a standardised warm-up, consisting of ten minutes rowing at 100 Watts for females and 130 Watts for males, the step test commenced. The test consisted of seven discontinuous 4 minute bouts at fixed exercise intensities with each bout followed by a one minute recovery. The subjects were instructed to begin the test with a stroke rate of 18 and to increase stroke rate by 2 strokes per minute for each stage. The test commenced with a work load and increment based purely on each rowers own best 2000 m time trial time from the previous year. The workloads and increments have been designed to elicit a blood lactate concentration in the range of 5-8 mmol.L$^{-1}$ in the 6th stage, with the 7th stage being a maximum all-out effort.
Heart rate was recorded throughout the test using a Polar heart rate monitor (RS400, Polar Electro OY Finland) and submaximal heart rates were identified as the average heart rate over the final 30 seconds of each workload. Maximum heart rate was the highest recorded value over a 5 second sampling period during the entire test. The mean power output achieved during the final 4 minute bout was identified as the PPO. The power associated with $\dot{V}O_{2\text{max}}$ ($\dot{W}O_{2\text{max}}$) was calculated by solving the regression equation describing $\dot{V}O_{2\text{max}}$ and power for the six incremental stages. The oxygen cost of movement (ECON) was assessed through calculation of the mean oxygen uptake per watt (ml/W) of the submaximal stages below lactate threshold as described by Nevill et al. (2011).

5.3.2.5. Gas Analysis

During the incremental step test expired air was continuously analysed for O$_2$ and CO$_2$ concentrations using an online gas collection system (Moxus modular oxygen uptake system, AEI technologies, Pittsburgh, PA) which was averaged over 30 second intervals. Prior to each test the analysers were calibrated with gases of known concentration (15.99% O$_2$ and 4.04% CO$_2$) and the pneumotach was calibrated with a 3l syringe. This was also verified after each test. Submaximal oxygen uptakes were calculated by averaging the reading recorded during the final two minutes of each submaximal workload. $\dot{V}O_{2\text{max}}$ was identified from the mean of the highest two consecutive readings in the final 4 minute bout.

5.3.2.6. Blood Lactate Analysis

Following the standardised warm-up and prior to the incremental test an earlobe blood sample (5µL) was taken to determine pre-test blood lactate concentration using the Lactate Pro®. The Lactate Pro® has a high data precision coefficient of variance of 3% and has been shown to correlate highly ($r = 0.99$) with traditional methods of laboratory assessment of blood lactate (Pyne et al. 2000). In order to begin the test the pre-test lactate measurement was required to be less that 1.2 mmol.l$^{-1}$. In the case where this value was greater the 1.2 mmol.l$^{-1}$ the participant was instructed to continue the warm-up for a specific length of time based upon the requirements of each individual. The duration for which the warm-up was extended depended upon the individual’s blood lactate level at the
end of the ten minute warm-up and once a blood lactate level of 1.2 or lower was achieved the warm-up was ceased.

Once the incremental test commenced a blood lactate sample was taken immediately at the end of each 4 minute bout. A sample was also taken 2 minutes and 5 minutes post the final 4 minute maximal bout.

All blood lactate plots were examined both manually and with the use of Lactate-E, software for blood lactate endurance markers (Newell et al. 2007), to identify the lactate threshold, anaerobic threshold and the powers associated with 2 and 4 mmol.l\(^{-1}\). The lactate threshold and anaerobic threshold were identified using ADAPT (1995) methods with the lactate threshold recorded as the highest workload completed before a rise in blood lactate of greater than 0.4 mmol.l\(^{-1}\) and the anaerobic threshold was calculated using modified \(D_{\text{max}}\) (Bourdon 2000).

5. 3. 2. 7. Seven Stroke Power Test

The seven stroke power test was completed on the final day of testing. For the purpose of this test a force transducer was attached to the handle of the rowing ergometer and a PowerLab Data Acquisition Systems (ADInstruments, Oxford, UK) was incorporated in order to evaluate force profiles and power output measurements from each stroke.

Participants were given a specific warm-up which included 5 minutes rowing at 60% of power at \(\dot{V}O_{2\text{max}}\), 5 minutes stretching and a further 10 minutes of rowing comprised of maximum efforts of 1, 2, 3 and 4 strokes. The participants then performed a seven stroke maximum power test, the first two of which were not recorded as the rower overcame the inertia of the flywheel. The rowers were required to maintain a stroke rate of 30 strokes per minute. If the stroke rate of 30 was not adhered to the participant was required to return to the lab to repeat the test a minimum of three hours later. Maximum force \((F_{\text{max}})\), maximum power \((W'_{\text{max}})\) and stroke rate were expressed as the mean value over the five recorded strokes.
5.3.3. Statistical Analyses

All statistical analyses were performed using the statistical package SPSS (Release 20.0). Means and standard deviations were calculated for all measured variables for all subjects and by gender. Normality of the data was verified using the Shapiro-Wilk test. For variables which were normally distributed Pearson’s product-moment correlation was used to examine the relationship between individual physiological variables and 2000 m rowing speed for all subjects and by gender. For variables which were not normally distributed Spearman’s correlation was used. The interpretation of correlation moments was: $r = 0.0$ to 0.09 (trivial); 0.1 to 0.29 (small); 0.3 to 0.49 (moderate); 0.5 to 0.69 (strong); 0.7 to 0.89 (very strong); 0.9 to 0.99 (nearly perfect) and 1.0 (perfect) (Hopkins 2000). 2000 m rowing speed (meters per second), calculated from the 2000 m time trial, was chosen to be the criterion variable in order to avoid reporting an implausible regression model that would predict a negative performance time. Significant correlations between 2000 m rowing speed and outcome variables were established using an alpha level of $p \leq 0.05$.

Stepwise (backward) multiple regression analysis, with 2000 m rowing ergometer velocity the dependent variable, was used to obtain a model to predict performance. In order to determine which variables to include in the stepwise regression, Pearson’s product-moment correlation and Spearman’s correlation were used to examine the relationship between all variables in a correlation matrix to identify any multi-collinearity. To ensure multi-collinearity was eliminated, variance inflation factor and tolerance statistics were utilised whereby all variables were required to have a variance inflation factor of less than 10 and a tolerance value of greater than .1 (Field 2005). In the case of two variables showing high inter-correlation ($r > .80$) (Field 2005) only one was entered into the regression analysis. Where multi-collinearity occurred the decision of which variable to enter into the regression analysis was based upon both theoretical knowledge and the variable which made the most impact on the predictive model in terms of both $R^2$ and the standardised coefficients $\beta$. The probability of the change in explained variance ($R^2$ change) F-value for removal of a variable was $\geq 0.10$. The strength of each model fit to the data was assessed using an ANOVA, and considered satisfactory where $p \leq 0.05$. Each predictor in the model was also assessed to ensure that a significant contribution was being made to the model and again was considered satisfactory where $p \leq 0.05$. 

121
5. 4. Results

Table 5.2 presents the mean (± standard deviation) of all the variables measured for all subjects and by gender.

Table 5.2: Mean values for all variables measured for all subjects and by gender.

<table>
<thead>
<tr>
<th></th>
<th>All Subjects (n=27)</th>
<th>Male (n=18)</th>
<th>Female (n=9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2000 m time (s)</td>
<td>414.3 ± 34.3</td>
<td>391.9 ± 11.1</td>
<td>459.0 ± 13.9</td>
</tr>
<tr>
<td>Speed (m.s⁻¹)</td>
<td>4.86 ± 0.38</td>
<td>5.10 ± 0.14</td>
<td>4.36 ± 0.13</td>
</tr>
<tr>
<td>BF%</td>
<td>21.2 ± 6.9</td>
<td>17.4 ± 3.7</td>
<td>29.6 ± 4.6</td>
</tr>
<tr>
<td>Lean%</td>
<td>75.6 ± 7.4</td>
<td>79.6 ± 4.2</td>
<td>66.7 ± 4.7</td>
</tr>
<tr>
<td>FF%</td>
<td>79.6 ± 7.4</td>
<td>83.5 ± 4.2</td>
<td>70.7 ± 4.9</td>
</tr>
<tr>
<td>Skinfold (mm)</td>
<td>98.0 ± 44.4</td>
<td>75.8 ± 24.2</td>
<td>142.3 ± 43.0</td>
</tr>
<tr>
<td>PPO (W)</td>
<td>319.1 ± 69.0</td>
<td>363.1 ± 31.3</td>
<td>231.2 ± 18.3</td>
</tr>
<tr>
<td>VO₂max (l/min⁻¹)</td>
<td>4.59 ± 0.83</td>
<td>5.10 ± 0.39</td>
<td>3.55 ± 0.34</td>
</tr>
<tr>
<td>VO₂max (ml·kg·min⁻¹)</td>
<td>54.3 ± 8.7</td>
<td>58.6 ± 6.9</td>
<td>45.7 ± 4.4</td>
</tr>
<tr>
<td>WO₂max (W)</td>
<td>297.6 ± 58.9</td>
<td>336.0 ± 21.6</td>
<td>220.9 ± 18.5</td>
</tr>
<tr>
<td>ECON (ml/W)</td>
<td>12.96 ± 1.79</td>
<td>12.28 ± 1.53</td>
<td>14.3 ± 1.6</td>
</tr>
<tr>
<td>2k Post La (mmol·l⁻¹)</td>
<td>13.0 ± 2.2</td>
<td>13.2 ± 2.0</td>
<td>12.8 ± 2.5</td>
</tr>
<tr>
<td>VO₂LT (l/min⁻¹)</td>
<td>2.75 ± 0.52</td>
<td>3.03 ± 0.38</td>
<td>2.19 ± 0.16</td>
</tr>
<tr>
<td>VO₂AT (l/min⁻¹)</td>
<td>3.80 ± 0.64</td>
<td>4.19 ± 0.30</td>
<td>3.01 ± 0.29</td>
</tr>
<tr>
<td>LT₅₀VO₂max</td>
<td>60.3 ± 5.4</td>
<td>59.4 ± 5.7</td>
<td>61.9 ± 4.9</td>
</tr>
<tr>
<td>AT₅₀VO₂max</td>
<td>83.1 ± 3.0</td>
<td>82.1 ± 2.7</td>
<td>84.9 ± 3.1</td>
</tr>
<tr>
<td>WLT (W)</td>
<td>154.9 ± 33.2</td>
<td>172.7 ± 25.1</td>
<td>119.1 ± 9.2</td>
</tr>
<tr>
<td>WAT (W)</td>
<td>237.8 ± 45.8</td>
<td>266.5 ± 21.9</td>
<td>180.3 ± 15.1</td>
</tr>
<tr>
<td>W₂mmol/l (W)</td>
<td>182.6 ± 42.7</td>
<td>207.6 ± 26.8</td>
<td>132.6 ± 13.2</td>
</tr>
<tr>
<td>W₄mmol/l (W)</td>
<td>232.5 ± 50.2</td>
<td>264.2 ± 23.6</td>
<td>169.1 ± 14.6</td>
</tr>
<tr>
<td>Peak La (mmol·l⁻¹)</td>
<td>13.3 ± 1.9</td>
<td>13.4 ± 2.1</td>
<td>13.1 ± 1.7</td>
</tr>
<tr>
<td>Fmax (N)</td>
<td>1136.28 ± 182.56</td>
<td>1245.22 ± 108.45</td>
<td>918.38 ± 56.64</td>
</tr>
<tr>
<td>Wmax (W)</td>
<td>463.0 ± 104.1</td>
<td>523.5 ± 63.3</td>
<td>341.9 ± 44.6</td>
</tr>
<tr>
<td>HRmax (bpm)</td>
<td>194 ± 8</td>
<td>195 ± 8</td>
<td>192 ± 7</td>
</tr>
</tbody>
</table>

Data expressed as mean ± SD
The correlation coefficients for all measured variables and 2000 m rowing velocity are presented in Table 5.3. Significant correlations were observed in submaximal \( W_4 \), \( \dot{V}O_{2AT} \), \( W'_2 \), \( \dot{V}O_{2LT} \), \( W_{LT} \), \( \dot{V}O_2 \), \( \text{ECON} \), \( \dot{V}O_{2max} \), maximal \( \dot{V}O_{2max} \), \( \dot{V}O_{2max} \), anaerobic/maximal power \( W_{max} \), \( F_{max} \) and morphological parameters (height, body mass, BF%, FF%, Lean% and skinfold).

Table 5.3: Correlation coefficients for determinants of 2000 m rowing ergometer speed.

<table>
<thead>
<tr>
<th></th>
<th>All Subjects (n=27)</th>
<th>Male (n=18)</th>
<th>Female (n=9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>( W\dot{V}O_{2max} ) (W)</td>
<td>0.98***</td>
<td>0.82***</td>
<td>0.96***</td>
</tr>
<tr>
<td>PPO (W)</td>
<td>0.93***</td>
<td>0.77***</td>
<td>0.95***</td>
</tr>
<tr>
<td>( W_{max} ) (W)</td>
<td>0.93***</td>
<td>0.82***</td>
<td>0.49</td>
</tr>
<tr>
<td>( W_2 ) mmol/l (W)</td>
<td>0.91***</td>
<td>0.68**</td>
<td>0.83**</td>
</tr>
<tr>
<td>( \dot{V}O_{2AT} ) (l·min(^{-1}))</td>
<td>0.86***</td>
<td>0.55*</td>
<td>0.91**</td>
</tr>
<tr>
<td>( F_{max} ) (N)</td>
<td>0.86***</td>
<td>0.63**</td>
<td>0.45</td>
</tr>
<tr>
<td>( \dot{V}O_{2AT} ) (l·min(^{-1}))</td>
<td>0.85***</td>
<td>0.51*</td>
<td>0.71*</td>
</tr>
<tr>
<td>( \dot{V}O_{2max} ) (l·min(^{-1}))</td>
<td>0.84***</td>
<td>0.49*</td>
<td>0.72*</td>
</tr>
<tr>
<td>( W_2 ) mmol/l (W)</td>
<td>0.80***</td>
<td>0.27</td>
<td>0.37</td>
</tr>
<tr>
<td>( \dot{V}O_{2LT} ) (l·min(^{-1}))</td>
<td>0.76***</td>
<td>0.35</td>
<td>0.41</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>0.75***</td>
<td>0.54**</td>
<td>0.62</td>
</tr>
<tr>
<td>( W_{LT} ) (W)</td>
<td>0.75***</td>
<td>0.29</td>
<td>0.17</td>
</tr>
<tr>
<td>ECON (ml/W)</td>
<td>-0.72***</td>
<td>-0.50*</td>
<td>-0.48</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>0.58**</td>
<td>0.54*</td>
<td>0.44</td>
</tr>
<tr>
<td>( \dot{V}O_{2max} ) (ml·kg·min(^{-1}))</td>
<td>0.54**</td>
<td>-0.26</td>
<td>-0.01</td>
</tr>
<tr>
<td>BF%</td>
<td>-0.53**</td>
<td>-0.30</td>
<td>-0.36</td>
</tr>
<tr>
<td>FF%</td>
<td>0.52**</td>
<td>0.32</td>
<td>0.36</td>
</tr>
<tr>
<td>Lean%</td>
<td>0.51**</td>
<td>0.30</td>
<td>0.34</td>
</tr>
<tr>
<td>( AT_{30} \dot{V}O_{2max} )</td>
<td>-0.44*</td>
<td>-0.05</td>
<td>0.27</td>
</tr>
<tr>
<td>Skinfold (mm)</td>
<td>-0.39*</td>
<td>0.43</td>
<td>-0.2</td>
</tr>
<tr>
<td>( LT_{30} \dot{V}O_{2max} )</td>
<td>-0.25</td>
<td>0.44</td>
<td>-0.45</td>
</tr>
<tr>
<td>2k Post La (mmol·l(^{-1}))</td>
<td>0.34</td>
<td>0.01</td>
<td>0.05</td>
</tr>
<tr>
<td>Peak La (mmol·l(^{-1}))</td>
<td>0.27</td>
<td>-0.11</td>
<td>0.62</td>
</tr>
<tr>
<td>( HR_{max} ) (bpm)</td>
<td>0.09</td>
<td>-0.04</td>
<td>0.11</td>
</tr>
</tbody>
</table>

***p <0.001; **p < 0.01; *p < 0.05
Following the identification of multi-collinearity several parameters, including some variables highly correlated with 2000 m rowing speed, were removed from the regression analysis including $\dot{V}O_{2\text{max}}$, PPO, $W_{\text{max}}$, $F_{\text{max}}$, $W_{\text{4mmol/l}}$ and $W_{\text{AT}}$. Multiple regression analysis was performed with the remaining variables with 2000 m rowing ergometer speed the dependent variable. The results of the stepwise (backward) linear regression are presented in Table 5.4. $\dot{W}V_{O_{2\text{max}}}$ was identified as the single best predictor of 2000 m rowing velocity, accounting for 96.5% of the variance:

$$\text{Velocity} = 2.955 + (0.006 \times \dot{W}V_{O_{2\text{max}}})$$

**Equation 5.1: Equation predicting 2000m rowing ergometer velocity**

The analysis also identified $\dot{W}V_{O_{2\text{max}}}$ as the parameter with the greatest influence on 2000 m rowing speed with a standardised coefficient $\beta$ of .982. Further explanatory variables, in addition to $\dot{W}V_{O_{2\text{max}}}$ were considered and included in the model. However, the addition of other variables to the model did not significantly increase the predictive power of the model, with a negligible increase in $R^2$. The influence of additional variables on 2000 m rowing was also negligible with standardised coefficient $\beta$ values of .085, .019, .024 and -.076 for $W_{\text{2mmol/l}}$, $\dot{V}O_{2\text{LT}}$, ECON and BF% respectively, noticeable smaller than that of $\dot{W}V_{O_{2\text{max}}}$ (.982) alone.

**Table 5.4: Multiple regression to predict 2000 m rowing velocity and the influence of parameters on 2000 m rowing speed for all subjects**

<table>
<thead>
<tr>
<th>Independent Variables</th>
<th>Unstandardised Coefficients</th>
<th>Standardised Coefficients</th>
<th>Standard Error</th>
<th>$\beta$</th>
<th>Pvalue</th>
</tr>
</thead>
<tbody>
<tr>
<td>R$^2 = 0.965$, F = 691.074, p &lt; 0.001</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Constant</td>
<td>2.955</td>
<td>0.074</td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>$\dot{W}V_{O_{2\text{max}}}$</td>
<td>0.006</td>
<td>&lt; 0.001</td>
<td>0.982</td>
<td></td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Multiple regression analysis was performed for male subjects only. The results of the stepwise (backward) linear regression are presented in Table 5.5. The results indicate that for male subjects $\dot{W}V_{O_{2\text{max}}}$ alone is not the best predictor of 2000 m rowing velocity,
accounting for only 66.9% of the variance. The results identified both $\dot{W}\dot{V}O_{2\text{max}}$ and $W_{\text{max}}$ as the key determinants of 2000 m rowing speed, accounting for 76.9% of the variance for this group of subjects:

$$\text{Velocity} = 3.515 + (0.003 \times \dot{W}\dot{V}O_{2\text{max}}) + (0.001 \times W_{\text{max}})$$

**Equation 5.2: Equation predicting 2000 m rowing ergometer velocity for male rowers**

The multiple regression analysis also indicated through the standardised coefficient $\beta$, that $\dot{W}\dot{V}O_{2\text{max}}$ and $W_{\text{max}}$ have a similar influence on 2000 m rowing velocity in male well-trained rowers with values of .467 and .473 respectively.

**Table 5.5: Multiple regression to predict 2000 m rowing speed and the influence of parameters on 2000 m rowing velocity for male subjects**

<table>
<thead>
<tr>
<th>Model</th>
<th>Independent Variables</th>
<th>Unstandardised Coefficients</th>
<th>Standardised Coefficients</th>
<th>Pvalue</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>B</td>
<td>Standard Error</td>
<td>$\beta$</td>
</tr>
<tr>
<td>1.</td>
<td>Constant</td>
<td>3.515</td>
<td>0.289</td>
<td></td>
</tr>
<tr>
<td></td>
<td>$\dot{W}\dot{V}O_{2\text{max}}$</td>
<td>0.003</td>
<td>0.001</td>
<td>.467</td>
</tr>
<tr>
<td></td>
<td>$W_{\text{max}}$</td>
<td>0.001</td>
<td>&lt;0.001</td>
<td>.473</td>
</tr>
</tbody>
</table>

$R^2 = 0.769, F = 25.009, p < 0.001$

<table>
<thead>
<tr>
<th>Model</th>
<th>Independent Variables</th>
<th>Unstandardised Coefficients</th>
<th>Standardised Coefficients</th>
<th>Pvalue</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>B</td>
<td>Standard Error</td>
<td>$\beta$</td>
</tr>
<tr>
<td>2.</td>
<td>Constant</td>
<td>3.294</td>
<td>0.319</td>
<td></td>
</tr>
<tr>
<td></td>
<td>$\dot{W}\dot{V}O_{2\text{max}}$</td>
<td>0.005</td>
<td>0.001</td>
<td>.818</td>
</tr>
</tbody>
</table>

$R^2 = 0.669, F = 32.345, p < 0.001$

Model 1: Predictors in model: $\dot{W}\dot{V}O_{2\text{max}}, W_{\text{max}}$

Model 2: Predictor in model: $\dot{W}\dot{V}O_{2\text{max}}$

Table 5.6 presents results from the multiple regression analysis for female rowers. The results identify $\dot{W}\dot{V}O_{2\text{max}}$ as the most important determinant of 2000 m rowing performance accounting for 92.9% of the variance:
Velocity = 2.841 + (0.007 × \(\hat{\dot{W}O}_{2\text{max}}\))

**Equation 5.3: Equation predicting 2000m rowing ergometer velocity for female rowers**

The results also indicate that \(\hat{\dot{W}O}_{2\text{max}}\) has the greatest influence on 2000 m rowing performance with a standardised coefficient \(\beta\) of .964.

**Table 5.6: Multiple regression to predict 2000 m rowing velocity and the influence of parameters on 2000 m rowing velocity for female subjects**

<table>
<thead>
<tr>
<th>Independent Variables</th>
<th>Unstandardised Coefficients</th>
<th>Standardised Coefficients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>Standard Error</td>
</tr>
<tr>
<td><strong>Constant</strong></td>
<td>2.841</td>
<td>0.159</td>
</tr>
<tr>
<td>(\hat{\dot{W}O}_{2\text{max}})</td>
<td>0.007</td>
<td>0.001</td>
</tr>
</tbody>
</table>

**5.5. Discussion**

Rowing is a strength-endurance sport demanding a high degree of muscular strength and endurance while also relying on both the aerobic and anaerobic energy pathways (Kyrolainen and Smith 1999, Hagerman 1984, Jackson and Secher 1976). This study examined aerobic, anaerobic and strength/power indices of performance through investigating the relationship between submaximal, maximal and anaerobic/maximal power parameters and 2000 m rowing performance. The main finding of this study was that, of the measured variables, \(\hat{\dot{W}O}_{2\text{max}}\) was the best single predictor of 2000 m rowing performance.

\(\hat{\dot{W}O}_{2\text{max}}\) was identified as the strongest correlate of performance (\(r = 0.98, p < 0.001\)) in this group of well-trained, well-trained rowers. This is in agreement with several studies which have identified \(\hat{\dot{W}O}_{2\text{max}}\) as an important parameter in predicting 2000 m rowing ergometer performance (Nevill et al. 2011, Ingham et al. 2002, Cosgrove et al. 1999, Jurimae et al. 2000). Most recently Nevill et al. (2011) identified \(\hat{\dot{W}O}_{2\text{max}}\) as the best single
predictor of performance, explaining 95.3% of the variance in rowing speed in elite rowers. Similar findings have been reported for $\dot{V}O_{2\text{max}}$ and distance running performance (Noakes et al. 1990, Morgan et al. 1989, Jones and Doust 1998). However, other studies which have investigated $W\dot{V}O_{2\text{max}}$ as a predictor of 2000 m rowing performance have involved either elite rowers (Nevill et al. 2011, Ingham et al. 2002) or heterogeneous groups of rowers indicated by the 2000 m performance times of the rowers (Cosgrove et al. 1999). The rowers in the present study represent a homogenous group of well-trained rowers, a group which the determinants of 2000 m rowing performance have not recently been investigated.

Traditionally, $\dot{V}O_{2\text{max}}$ has been regarded as the best single predictor or measure of endurance performance. Several studies have identified $\dot{V}O_{2\text{max}}$ as the best predictor of 2000 m rowing performance (Cosgrove et al. 1999, Ingham et al. 2002, Kramer et al. 1994, Secher et al. 1982) with Secher et al. (1982) reporting a direct relationship between placing in an international regatta and the average absolute $\dot{V}O_{2\text{max}}$ for the crew ($r = 0.87$). In the present study however, while $\dot{V}O_{2\text{max}}$ was highly correlated with performance ($r = 0.84, p < 0.001$), there were several parameters with higher correlations to performance namely $W\dot{V}O_{2\text{max}}$, PPO, $W\dot{\text{max}}$, $W_{4\text{mmol/l}}$, $W_{\text{AT}}$, $F_{\text{max}}$ and $\dot{V}O_{2\text{AT}}$. In addition, when $\dot{V}O_{2\text{max}}$ was expressed relative to body weight its correlation with 2000 m rowing performance dropped dramatically ($r = 0.54, p < 0.01$), an observation which has been made in several previous studies (Secher et al. 1982, Jurimae et al. 2002) and reflects the non-weight bearing nature of rowing.

These results support earlier observations that a high relative $\dot{V}O_{2\text{max}}$ is required for success in rowing (Hagerman 1984, Hagerman et al. 1978, Hagerman et al. 1979, McKenzie and Rhodes 1982, Secher et al. 1983, Secher et al. 1982) but they also indicate that $W\dot{V}O_{2\text{max}}$ is more highly correlated with performance. Indeed it has recently been observed that $W\dot{V}O_{2\text{max}}$ may be a more valuable tool for monitoring performance than $\dot{V}O_{2\text{max}}$ due to the apparent plateau in $\dot{V}O_{2\text{max}}$ in elite endurance athletes (Legaz Arrese et al. 2005, Mikulic 2011). Mikulic (2011) found that during the final two years of a six year physiological study of a world champion crew, $\dot{V}O_{2\text{max}}$ exhibited a plateau while $W\dot{V}O_{2\text{max}}$ during the same period increased by 5%.
In addition to $\dot{W}\dot{V}O_{2\text{max}}$ and $\dot{V}O_{2\text{max}}$ other parameters, both submaximal and maximal, of aerobic function were correlated with performance. PPO, the mean power output achieved during the final 4 minute stage of the incremental step test, was very strongly correlated with performance ($r = 0.93$, $p < 0.001$). The use of this maximal aerobic parameter in the monitoring of performance has a practical advantage for the coach and athlete as its measurement does not require any specialised metabolic equipment. In addition PPO has been used in the prescription of intervals for high intensity interval training for well-trained rowers. Driller et al. (2009) prescribed intervals of 90% PPO and concluded that high intensity interval sessions at or close to PPO may be the most effective means of eliciting additional improvements in $\dot{V}O_{2\text{max}}$ in already-well trained athletes. Therefore, in addition to its high correlation with performance, this parameter can facilitate effective training prescription.

In accordance with several studies (Cosgrove et al. 1999, Ingham et al. 2002, Messonnier et al. 1997, Riechman et al. 2002, Faff et al. 1993, Womack et al. 1996, Womack et al. 1992, Klusiewicz 1993, Bourdin et al. 2004), indices of the blood lactate response to submaximal exercise demonstrated a high correlation with performance including $W_{4\text{mmol/l}}$ ($r = 0.91$, $p < 0.001$), $W_{AT}$ ($r = 0.86$, $p < 0.001$), $W_{2\text{mmol/l}}$ ($r = 0.80$, $p < 0.001$), $W_{LT}$ ($r = 0.75$, $p < 0.001$). $W_{4\text{mmol/l}}$ has been identified as being closely related to 2000 m rowing performance (Ingham et al. 2002, Womack et al. 1996, Faff et al. 1993, Klusiewicz 1993, Cosgrove et al. 1999). However, in contrast to the present study several studies have reported that $W_{4\text{mmol/l}}$ is the best parameter for determining rowing performance (Womack et al. 1992, Faff et al. 1993, Klusiewicz 1993). Steinacker (1993) observed that $W_{4\text{mmol/l}}$ corresponds to approximately 75-85% of the power at $\dot{V}O_{2\text{max}}$ in successful rowers. The results of the present study, in which $W_{4\text{mmol/l}}$ corresponds to 78% of the power at $\dot{V}O_{2\text{max}}$, are in line with this. It is worth noting, however, that the $W\dot{V}O_{2\text{max}}$ observed in this study (297.6 ± 58.9W) was not as high as that observed in studies with elite rowers (Nevill et al. 2011, Mikulic 2011). Interestingly, the physiological significance of $W_{4\text{mmol/l}}$ has been questioned by some due to fact that is does not take into account the individual kinetics of the blood lactate concentration curve (Maestu et al. 2005, Coyle 1995, Stegmann et al. 1981) but there is limited information available regarding rowing performance and other blood lactate indices including $W_{AT}$, $W_{2\text{mmol/l}}$ and $W_{LT}$. Ingham et al. (2002) and Nevill (2011) both reported higher correlations for $W_{2\text{mmol/l}}$ and 2000 m rowing performance ($r = 0.92$, $p < 0.001$) than that observed in the present study. This may be due to the higher calibre of athletes used in these two studies,
with all rowers reaching World Championship finals, compared to the well-trained rowers in the present study.

It has been suggested that success in endurance sports is dependent on the economical utilisation of a high aerobic capacity and the ability to employ a large fraction of that capacity during competition (Costill et al. 1973, Jones 2006, Noakes 2003). Economy has been associated with success in distance running (Di Prampero et al. 1993, Conley and Krahenbuhl 1980, Noakes 1988), particularly where differences in $\dot{V}O_{2\text{max}}$ values between subjects are small (Noakes 2003, Noakes 1988, Conley and Krahenbuhl 1980). Rowing economy (ECON), assessed through the calculation of mean oxygen uptake per Watt, has received little attention resulting in a paucity of information regarding the importance of economy to rowing performance. Cosgrove et al. (1999) speculated that if rowers could reduce the oxygen cost of rowing at a given power, they could probably enhance performance. A strong negative relationship between ECON and 2000 m rowing performance was identified in this study ($r = -0.72$, $p < 0.001$), a finding which has not been previously reported with Nevill et al. (2011) observing a weak relationship between the two variables ($r = -0.33$). This finding suggests that in well-trained rowers, with lower aerobic and anaerobic capacities than elite rowers, the economy of movement may be an important determinant of performance. However, the concept of the economy requires further examination in order to critically interpret the findings.

Rowing differs from other endurance sports with similar energy demands due to the fact that rowers are required to continually generate much higher forces for the duration of competition. Despite the source of energy in a 2000 m rowing race being predominantly aerobic, anaerobic parameters such as maximal strength and muscle power are also suggested to have an impact on elite rowing performance (Ingham et al. 2002, de Campos Mello et al. 2009, Secher 1993) as performance depends on the functional capacity of both aerobic and anaerobic pathways. Short duration rowing power tests have been found to be strongly correlated with 2000 m rowing performance (Ingham et al. 2002, Riechman et al. 2002, Nevill et al. 2011) and Nevill et al. (2011) have suggested that the maximum power and force output ($W_{\text{max}}$, $F_{\text{max}}$) expressed as the mean over five maximal strokes provides an estimate of anaerobic capability. Similar to previous studies (Nevill et al. 2011, Ingham et al. 2002) $W_{\text{max}}$ and $F_{\text{max}}$ were highly correlated to 2000 m rowing performance ($r = 0.93$, $p < 0.001$ and $r = 0.86$, $p < 0.001$ respectively), however unlike the present study, Ingham
et al. (2002) reported that $W_{\text{max}}$ and $F_{\text{max}}$ were the strongest correlates of performance ($r = 0.95$, $p < 0.001$). This difference may again be the result of the calibre of athletes used in the two studies as Ingham et al. (2002) also observed that elite rowers could maintain, on average, 77% of $W_{\text{max}}$ during the 2000 m time trial compared to 70% in the present study illustrating that during a 2000 m race elite athletes can work at a higher percentage of maximum power when compared to well-trained rowers.

The results of the current study revealed that several parameters were identified as being highly correlated and significantly associated with 2000 m rowing performance. However, when multiple regression was performed $W\dot{V}O_{2\text{max}}$ emerged as the best predictor of performance accounting for 96.5% of the variance in performance ($R^2 = 0.965$, $p < 0.001$). The inclusion of other parameters did not lead to any further significant increase in $R^2$ and offered no advantage over the model with $W\dot{V}O_{2\text{max}}$ exclusively. Furthermore, $W\dot{V}O_{2\text{max}}$ was also identified as having the greatest influence on 2000 m rowing speed ($\beta = .982$) with all other parameters exerting a minimal, non-significant influence on 2000 m performance, as examined through the standardised coefficients $\beta$.

The model for the prediction of 2000 m rowing performance, which emerged from the multiple regression analysis, did not entirely agree with models which have previously been proposed. It has previously been suggested that one exclusive parameter cannot explain all of the variance in rowing performance and that by incorporating variables, such as muscle mass (Jurimae et al. 2002), peak blood lactate (Warrington et al. 2003), body composition (Purge et al. 2004) or velocity at a blood lactate of 4mmol$\text{l}^{-1}$ (Cosgrove et al. 1999), with $\dot{V}O_{2\text{max}}$ and placing them in regression equations increases the amount of variance that can be accounted for. While $W\dot{V}O_{2\text{max}}$ has been identified as a component in other 2000 m rowing predictive models (Ingham et al. 2002, Nevill et al. 2011), it has not been reported to be an exclusive parameter. Ingham et al. (2002) found that $W\dot{V}O_{2\text{max}}$, $W_{\text{max}}$, $W_{4\text{mmol/l}}$ and $\dot{V}O_{2\text{LT,LSS}}$ accounted for 98% of performance while Nevill et al. (2011) identified $W\dot{V}O_{2\text{max}}$, $W_{\text{max}}$ and $\dot{V}O_{2\text{LT}}$ as the key determinants of performance. Nevill et al. (2011) proposed that in order to successfully identify the key determinants of 2000 m performance, performance determinant models should consider a contribution from a low-intensity metabolic threshold, a maximal and/or functional aerobic capacity, and an indicator of anaerobic/maximal power capability. This may be due to the fact that elite rowers are likely to have similar aerobic power and rely on extremely developed aerobic and anaerobic
capacities, but in the case of well-trained rowers $W\dot{V}O_{2\text{max}}$ appears to be the most important variable in predicting 2000 m rowing performance in addition to being the variable with the most influence on performance.

A large number of variables were significantly correlated with performance but further investigation revealed a high degree of multi-collinearity between these variables and $W\dot{V}O_{2\text{max}}$. These variables included, but were not limited to, $W_{\text{max}}$, $F_{\text{max}}$ and $W_{4\text{mmol}/\text{l}}$. This multi-collinearity resulted in the removal of these variables from the predictive model and it makes it difficult to understand the individual contribution of each variable. However, it also suggests that these variables, in some way, interact with $W\dot{V}O_{2\text{max}}$ during 2000 m rowing performance. Interestingly, other rowing studies have not mentioned the concept of multi-collinearity and given the high degree of multi-collinearity in the present study it is reasonable to suggest that it may also have existed in these other studies. The interpretation of data can be problematic where multi-collinearity among predictor variables is unreported.

When the group was divided by gender it is interesting to note that $W\dot{V}O_{2\text{max}}$ remained the most important determinant of 2000 m rowing performance in well-trained female rowers, accounting for 92.9% of the variance while also exerting the greatest influence on performance ($\beta = .964$). However, $W\dot{V}O_{2\text{max}}$ alone accounted for only 66.9% of the variance for well-trained male rowers. The addition of $W_{\text{max}}$ improved the model to account for 76.9% of the variance with both $W\dot{V}O_{2\text{max}}$ and $W_{\text{max}}$ exerting a similar influence on performance ($\beta = .467$ and $\beta = .473$ respectively). The inclusion of $W_{\text{max}}$ may be expected as it has previously been reported that stronger rowers, particularly those with high leg strength perform better (Hagerman et al. 1972) and this model for well-trained male rowers is more in line with previous models which display the interaction between aerobic and anaerobic energy pathways when performing a 2000 m time trial (Nevill et al. 2011). From a practical perspective, the results of the present study suggest that rowing coaches and rowers should implement training strategies focusing primarily on the development of $W\dot{V}O_{2\text{max}}$ while also incorporating elements to develop $W_{\text{max}}$ for males in particular. The force production required to complete one repetition in rowing is higher than that observed in other endurance sports such as cycling and therefore high levels of strength may be necessary to obtain high levels of power during rowing which may explain the
importance of $W_{max}$ for males. Jensen et al. (1996) made a similar suggestion with regard to training, that rowers should emphasize training that increases leg strength.

5. 6. Conclusion

The rowing ergometer is a valuable tool for rowing training, the monitoring of training effects and the evaluation of rowing performance. In contrast to other endurance sports, however, there is a paucity of research available regarding physiological predictors of rowing performance. The findings of the present study revealed that of the 24 variables measured $\dot{W}V_{O2max}$ was the single best predictor of 2000 m rowing ergometer performance in well-trained rowers. $W_{max}$ was also identified as an important predictor of performance, for male well-trained rowers in particular, with both $\dot{W}V_{O2max}$ and $W_{max}$ having a similar influence on performance. As a result coaches should focus on the improvement of both $\dot{W}V_{O2max}$ and $W_{max}$ in order to enhance performance. A number of other physiological variables were significantly related to performance, but when these variables were included in the predictor model, the additional increase in $R^2$ was negligible.

5. 7. Summary

In summary, $\dot{W}V_{O2max}$ is the single best predictor of rowing performance in well-trained rowers and there are several other parameters which are highly correlated with performance. Aerobic training is traditionally the focus of rowers. However, high intensity interval training has been shown to induce improvements in endurance performance, $\dot{W}V_{O2max}$ and associated physiological variables in well-trained athletes (Driller et al. 2009, Laursen et al. 2005, Laursen and Jenkins 2002, Billat et al. 2002a, Smith et al. 1999, Westgarth-Taylor et al. 1997, Stepto et al. 1999). Therefore the aim of Chapter 6 is to monitor and evaluate changes in rowing performance and associated physiological variables in response to traditional rowing training and high intensity interval training.
Chapter 6. The Effect of High Intensity Interval Training on Well-Trained Rowers.
6. 1. Abstract

The aim of this study was to compare the effects of traditional long, slow, distance rowing training (LSD) with high intensity interval training (HIIT) in a group of well-trained rowers (n=19). Before and after the eight week training intervention each subject performed 4 tests in the human performance laboratory: 1) a 2000 m time trial; 2) a seven stage incremental step test to determine maximum oxygen uptake ($\dot{V}O_{2\text{max}}$), power at $\dot{V}O_{2\text{max}}$ ($W\dot{V}O_{2\text{max}}$), peak power output (PPO), economy of movement (ECON) and blood lactate indices; 3) a seven stroke power test to determine maximal power ($W_{\text{max}}$) and maximal force ($F_{\text{max}}$) and 4) a time to exhaustion test ($T_{\text{max}}$) at $W\dot{V}O_{2\text{max}}$. Body composition was also assessed pre and post training through percentage body fat (BF%), percentage of lean body mass (Lean%) and percentage of fat free mass (FF%) using the skinfold method (7 sites) and DXA. Following the baseline testing subjects were randomly assigned to either the HIIT or LSD training group. The LSD group continued with normal training of ten aerobic sessions per week including eight extensive aerobic sessions greater than 60 minutes in duration and two intensive aerobic sessions less than 60 minutes in duration. HIIT also involved ten sessions per week, eight of which were low intensity, aerobic sessions and two of which were HIIT sessions. The HIIT sessions involved 6-8 x 2.5 minute intervals at 100% PPO with recovery time based on heart rate (HR) returning to 70% HR$_{\text{max}}$. HIIT resulted in a significantly greater improvement in $\dot{V}O_{2\text{max}}$ ($p = 0.035$) and $W_{\text{LT}}$ ($p = 0.005$) than LSD. There was also a significant improvement in 2000 m time trial performance following 8 weeks of HIIT ($p = 0.011$) and although a significant improvement was not observed in the LSD group ($p = 0.237$), the improvement with HIIT was not significantly different from the LSD group ($p = 0.595$). In addition no significant improvement was observed in buffering capacity in either group. The results indicate that eight weeks of HIIT performed at 100% PPO is a more effective means to elicit improvements in $\dot{V}O_{2\text{max}}$ and $W_{\text{LT}}$ than traditional training in well-trained rowers.

6. 2. Introduction

The physiological attributes of rowers are amongst the highest recorded for any sport (Hagerman et al. 1979) and rowing presents the unique challenge of balancing the high development of the physiological abilities of endurance and strength (Babraj and Volianitis 2007). During a 2000 m rowing race exceptionally high demands are placed on both the aerobic and anaerobic energy systems (Kyrolainen and Smith 1999, Hagerman 1984, Jackson and Secher
1976) and during competition, rowers have been found to exercise at a severe steady state with the majority of work performed at 95-98% of maximal rowing capacity (Hagerman et al. 1978, McKenzie and Rhodes 1982). However, despite the high percentage of maximum rowing capacity rowers perform at during competition and the importance of the anaerobic system, the majority of training completed by international rowers is completed below the blood lactate threshold (approximately 90% during the preparation period) (Secher 1993, Steinacker 1993). It therefore appears that little overall emphasis is placed on training in the range of 80-100% $\dot{V}O_{2\text{max}}$, which could be achieved through high intensity interval training (HIIT).

There is an increasing body of evidence to suggest that HIIT is effective in improving performance and aerobic capacity in well-trained athletes from a variety of endurance sports (Wenger and Bell 1986, Robinson et al. 1991). However, much of the research to date has focused on endurance runners (Smith et al. 1999, Smith et al. 2003) and cyclists (Lindsay et al. 1996, Westgarth-Taylor et al. 1997, Weston et al. 1997) and as a consequence there is a dearth of information on the effects of HIIT on well-trained rowers where the traditional use of long slow distance training still predominates. Driller et al. (2009) examined the effects of HIIT in well trained rowers and reported that four weeks of HIIT was associated with significantly greater improvements in time-trial performance ($1.9 \pm 0.9\%, P = .02$) and relative $\dot{V}O_{2\text{peak}} (7 \pm 6.4\%, P = .03)$ when compared with traditional rowing training.

The mechanisms responsible for the reported performance improvements with HIIT in well-trained athletes remain somewhat unclear. Several mechanisms have been purported including increases in the maximal activities of mitochondrial enzymes (Henriksson and Reitman 1976, Burgomaster et al. 2005, Saltin et al. 1976), a reduction in glycogen utilisation and lactate accumulation during exercise of similar workloads (Harmer et al. 2000, Clark et al. 2004, Burgomaster et al. 2006), improvements in the ventilatory threshold (Laursen et al. 2005, Acevedo and Goldfarb 1989, Laursen and Jenkins 2002), enhanced fat oxidation (Westgarth-Taylor et al. 1997), increases in anaerobic capacity (Laursen et al. 2005) and increased buffering capacity (Weston et al. 1997). It has been suggested that the ability to buffer H$^+$ accumulation may be an important determinant of high-intensity exercise performance (Edge et al. 2006c). While some studies have reported no improvements in buffering capacity (Harmer et al. 2000, Nevill et al. 1989), several studies have reported an improvement in muscle buffering capacity with HIIT (Bishop et al. 2009, Edge et al. 2006a, Edge et al. 2006b, Weston et al. 1997, Gibala et al. 2006, Bell and Wenger 1988, Juel 1998, Sharp et al. 1986). Edge et al. (2006c) proposed that a
greater buffering capacity may improve high intensity exercise performance by allowing anaerobic glycolysis to continue during maximal exercise, resulting in a larger lactate production without an associated increase in H⁺ accumulation.

Seiler and Tonnessen (2009) have suggested that combining large volumes of low-intensity training with cautious use of HIIT may be the best-practice model for the development of endurance performance potential. However, despite this, there are few studies that have investigated the use of HIIT in well-trained rowers (Driller et al. 2009). While the literature suggests that performance of HIIT in addition to traditional, long, slow distance training is beneficial for well-trained endurance athletes there is a need for a controlled training intervention study with well-trained rowers. The aim of this study therefore was to compare the effects of HIIT to traditional long slow distance training in well-trained rowers. Blood pH and muscle buffering capacity have not previously been researched in relation to rowing and HIIT and therefore the study also aimed to examine the role of blood buffering capacity as an underlying mechanism to any physiological or performance enhancements observed.

6.3. Method

6.3.1. Subjects

Nineteen national level rowers, 14 male and 5 female, participated in this study. The physical characteristics of the athletes are presented in Table 6.1. All participants were proficient in both sculling and sweep rowing. All participants were fully informed verbally and in writing of the requirements and the potential risks and benefits of participating (Appendix D). An informed consent (Appendix D) form and pre-test questionnaire (Appendix C and Appendix D) were completed by each participant. All participants were given strict pre-test protocol to be followed. All experimental procedures were approved by the University of Limerick Ethics Committee.

<table>
<thead>
<tr>
<th></th>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>Mass (kg)</th>
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<tbody>
<tr>
<td>Male (n=14)</td>
<td>21 ± 4</td>
<td>186.7 ± 5.1</td>
<td>85.6 ± 9.7</td>
</tr>
<tr>
<td>Female (n=5)</td>
<td>23 ± 5</td>
<td>180.2 ± 7.5</td>
<td>79.2 ± 9.6</td>
</tr>
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Data presented as mean ± SD
6.3.2. Overview of Research Design

Participants were randomly divided into two groups, a long, slow distance group (LSD) and a high intensity interval group (HIIT). All subjects completed a series of baseline tests including anthropometric testing, a 2000 m time trial, an incremental step test, a time to exhaustion test ($T_{\text{max}}$) and a seven stroke power test. All tests were performed on a Concept II model C air braked rowing ergometer (Concept II, Nottingham, England) using a drag factor of 130 for males and 120 for females. Testing was completed over the period of one week, with a minimum of 48 hours between tests and all tests were completed at the same time of the day to avoid any diurnal variation. Post-training tests were conducted in the same order as the pre-training tests and pre and post-training tests were also performed at the same time of the day. Participants were requested to follow strict protocols in the 24 hours prior to any test days which included maintaining a normal diet, optimizing hydration and refraining from alcohol. Participants were also required to undertake no more than 30 minutes light training during the 24 hours prior to the test. A training and food diary for the 24 hours prior to all testing days was requested from all participants. An eight week training block was completed, with the LSD continuing with normal training comprised of low-intensity, long duration training while the HIIT group replaced two low intensity, long duration sessions with two high intensity interval sessions. Throughout the duration of the study all subjects were required to keep a detailed training diary, containing information on all training performed over the eight week period (Appendix E). The following is a schematic summary of testing design (figure 6.1).
6. 3. 2. 1. Body Mass and Stature

Body mass was assessed in minimal clothing using a Seca 799 digital column scale (Seca, Hamburg, Germany) and height was measured using a Seca 217 stadiometer (Seca, Hamburg, Germany).

6. 3. 2. 2. Body Composition

Body composition was measured using skinfold thickness and dual-energy X-ray absorptiometry (DXA). Skinfold thickness was measured in accordance with the International Standards for Anthropometric Assessment (ISAK) protocol. Repeated skinfold measurements were taken, using a Harpenden skinfold callipers (Baty International, West Sussex, UK), at seven sites, biceps, triceps, subscapular, supraspinale, abdominal, frontal thigh and medial calf. The sum of these seven sites was then calculated. The DXA was used to calculate percentage body fat (BF%), percentage lean body mass (lean%) and percentage fat free mass (FF%). A Lunar iDXA™ scanner (GE Healthcare, Chalfont St Giles, Bucks., UK) with enCORE™ 2011 v.13.6
software was used to capture total body composition scans. Calibration with use of a phantom spine was carried out daily.

6. 3. 2. 3. 2000 m Time Trial

The 2000 m time trial was performed on the first day of baseline testing. The test-retest reliability of a 2000 m time trial on the Concept II model C has previously been examined with well-trained rowers, with a coefficient of variation of 0.6% being reported (Schabort et al. 1999). Participants were requested to record a training and food diary for the 24 hours prior to testing which would be replicated for the 2000 m time trial at the end of the eight week training block. All athletes were fully familiar with the test having previously completed the test on a number of occasions. Participants performed a self-selected warm up which would be repeated for the post test 2000 m time trial. An earlobe blood sample (5µL) was taken to determine blood lactate concentration (Lactate Pro, Arkray Factory Inc, Shiga, Japan) pre and post warm-up and 5 minutes post the 2000 m time trial. A finger prick blood sample (100 µL) was taken pre and 5 minutes post the time trial to determine pH and HCO₃⁻ using a blood gas analyser (i-Stat, Abbott Point of Care, Princeton, NJ). Heart rate was measured continuously (RS400, Polar Electro OY Finland) during the performance.

6. 3. 2. 4. Incremental Step Test

The incremental step test was completed a minimum of 48 hours after the 2000 m time trial and all athletes were instructed to follow strict pre-test protocols (Appendix D). The test was performed according to the Australian physiological assessment of rowing guidelines (Gore, 2000) (Appendix F) and was used to determine maximum oxygen uptake (\( \tilde{V}O_{2\text{max}} \)), power at \( \tilde{V}O_{2\text{max}} \) (\( \tilde{W}V\tilde{O}_{2\text{max}} \)), 4 minute all-out power output (PPO), oxygen cost of movement (ECON), the power output at lactate threshold (\( \tilde{W}_{LT} \)) and anaerobic threshold (\( \tilde{W}_{AT} \)), maximum heart rate, peak lactate (peak La), and the power outputs associated with blood lactate levels of 2 (\( \tilde{W}_{2\text{mmol/L}} \)) and 4 mmol.L⁻¹ (\( \tilde{W}_{4\text{mmol/L}} \)).

On completion of a standardised warm-up, consisting of ten minutes rowing at 100 Watts for females and 130 Watts for males, the step test commenced. The test consisted of seven discontinuous 4 minute bouts at a fixed exercise intensity and stroke rate and with each bout followed by a one minute recovery. The subjects were instructed to begin the test with a stroke
rate of 18 and to increase stroke rate by 2 strokes per minute for each stage. The test commenced with a work load and increment based purely on each rower's own best 2000 m time trial time from the previous year (Appendix F). The workloads and increments have been designed to elicit a blood lactate concentration in the range of 5-8 mmol.l\(^{-1}\) in the 6\(^{th}\) stage, with the 7\(^{th}\) stage being a maximum all-out effort.

Heart rate (RS400, Polar Electro OY Finland) was recorded throughout the test and submaximal heart rates were identified over the final 30 seconds of each workload. Maximum heart rate was the highest recorded value over a 5 second sampling period during the entire test. The mean power output achieved during the final 4 minute bout was identified as the PPO. The power associated with \(\dot{V}O_{2\text{max}}\) \((\dot{W} \cdot \dot{V}O_{2\text{max}})\) was calculated by using the regression equation describing \(\dot{V}O_2\) and power for the six incremental stages. The oxygen cost of movement (ECON) was assessed through calculation of the mean oxygen uptake per watt (ml/W) of the submaximal stages below lactate threshold as described by Nevill et al. (2011).

6.3.2.5. Gas Analysis

During the incremental step test expired air was continuously analysed for \(O_2\) and \(CO_2\) concentrations using an online gas collection system (Moxus modular oxygen uptake system, AEI technologies, Pittsburgh, PA) which was averaged over 30 second intervals. Prior to each test the analysers were calibrated with gases of known concentration (15.99% \(O_2\) and 4.04% \(CO_2\)) and the pneumotach was calibrated with a 3-l syringe. This was also verified after each test. Submaximal oxygen uptakes were calculated by averaging the reading recorded during the final two minutes of each submaximal workload. \(\dot{V}O_2\text{max}\) was identified from the mean of the highest two consecutive readings in the final 4 minute bout.

6.3.2.6. Blood Lactate Analysis

Following the standardised warm-up and prior to the incremental test an earlobe blood sample (5µL) was taken to determine pre-test blood lactate concentration using the Lactate Pro. The Lactate Pro® has a high data precision coefficient of variance of 3% and has been shown to correlate highly \((r = 0.99)\) with traditional methods of laboratory assessment of blood lactate (Pyne et al. 2000). In order to begin the test a pre-test lactate measurement of 1.2 mmol.l\(^{-1}\) was required. In the case where this value was greater the 1.2 mmol.l\(^{-1}\) the participant was instructed
to continue the warm-up for a specific length of time based upon the requirements of the individual.

For the duration of the test a blood lactate sample was taken immediately at the end of each 4 minute bout. A sample was also taken 2 minutes and 5 minutes post the final 4 minute maximal bout.

All blood lactate plots were examined both manually and with the use of Lactate-E, software for blood lactate endurance markers (Newell et al. 2007), to identify the lactate threshold, anaerobic threshold and the powers associated with 2 and 4 mmol\textsuperscript{l}\textsuperscript{-1}. The lactate threshold and anaerobic threshold were identified using ADAPT (1995) methods with lactate threshold recorded as the highest workload completed before a rise in blood lactate of greater than 0.4 mmol\textsuperscript{l}\textsuperscript{-1} and anaerobic threshold calculated using modified D\textsubscript{max} (Bourdon 2000). The analysis of the blood lactate response using Lactate-E also allowed for the identification of individualised training zones for each subject. Five training zones were identified, UT3 (recovery), UT2 (extensive aerobic), UT1 (intensive aerobic), AT (threshold) and Max (Max/anaerobic) and were used in the prescription of intensity for training sessions for both training groups.

6. 3. 2. 7. Blood Gas Analysis

Prior to and 5 minutes post the incremental test a finger prick blood sample (100 µL) was taken to determine pH and HCO\textsubscript{3}, using a blood gas analyser (i-Stat, Abbott Point of Care, Princeton, NJ).

6. 3. 2. 8. Rate of Perceived Exertion

Participants rated their perceived exertion using the Borg Scale. The Borg Scale is a 15 point scale (starting at 6 and ending at 20) which can be used to gauge the level of intensity of training. The subjects were requested to choose a number on the scale which best described their level of exertion. The scale was verbally anchored and the subjects were informed that the number should reflect how heavy and strenuous the exercise felt to them, where 6 means “no exertion at all” and 20 means “maximal exertion”.

141
6. 3. 2. 9. Time to Exhaustion

The time to exhaustion test was completed a minimum of 48 hours post the incremental step test in order to identify $T_{\text{max}}$, the length of time exercise at $\dot{W}\hat{V}O_{2\text{max}}$ could be sustained. The power at which each subject performed $T_{\text{max}}$ (100% $\dot{W}\hat{V}O_{2\text{max}}$) was calculated using the regression equation describing $\dot{V}O_2$ and power for the first six stages of the incremental step test. Following a warm-up consisting of 5 minutes rowing at 60% of power at $\dot{V}O_{2\text{max}}$, 5 minute stretching and a further 5 minutes rowing at 60% of power at $\dot{V}O_{2\text{max}}$, the participants performed a time to exhaustion test at 100% power at $\dot{V}O_{2\text{max}}$. The test began when the rower reached the required power, as displayed on the Concept II monitor, and was terminated when power dropped below 90% of power at $\dot{V}O_{2\text{max}}$ for three consecutive strokes. Throughout the test power and time elapsed were visible to the participants on the Concept II monitor. The time to exhaustion post test was performed at the same power output and stroke rate as the pre test.

6. 3. 2. 10. Seven Stroke Power Test

The seven stroke power test was completed on the final day of baseline testing. For the purpose of this test a force transducer was attached to the handle of the rowing ergometer and a PowerLab Data Acquisition Systems (ADInstruments, Oxford, UK) was incorporated in order to evaluate force profiles and power output measurements from each stroke.

Participants were given a specific warm-up which included 5 minutes rowing at 60% of power at $\dot{V}O_{2\text{max}}$, 5 minutes stretching and a further 10 minutes of rowing comprised of maximum efforts of 1, 2, 3 and 4 strokes. The participants then performed a seven stroke maximum power test, the first two of which were not recorded as the rower overcame the inertia of the flywheel. The rowers were required to maintain a stroke rate of 30 strokes per minute. Maximum force ($F_{\text{max}}$), maximum power ($W'_{\text{max}}$) and stroke rate was expressed as the mean value over the five recorded strokes.

6. 3. 2. 11. Eight Week Training Intervention

The study was conducted during the preparation phase of the participants yearly training program. The participants were randomly divided in one of two training groups, the high
intensity interval training group, or the long slow distance group for the duration of the study. For the duration of the eight weeks all subjects completed daily training diaries. For each training session completed type of training, duration, heart rate, distance and mean power were recorded (Appendix E).

6. 3. 2. 11. 1. Long, Slow Distance Group Training

For the duration of the eight week training period all participants in the LSD group were requested to maintain their current training which consisted of ten sessions per week. Using the results of the incremental step test individual training zones were prescribed for each participant based on lactate responses. Participants in the LSD group completed eight sessions in the extensive aerobic zone (UT2) and two sessions in the intensive aerobic zone (UT1). All extensive aerobic sessions were longer than 60 minutes in duration while the intensive aerobic sessions were less than 60 minutes.

6. 3. 2. 11. 2. High Intensity Interval Training Group

Participants in the high intensity interval training group (HIIT), similar to the LSD, completed ten training sessions per week. However, two extensive aerobic (UT2) sessions per week were replaced with two HIIT sessions. Several studies have reported improvements in performance and associated physiological parameters with two HIIT sessions per week (Smith 2003, Esfarjani and Laursen 2007, Laursen et al. 2005, Driller et al. 2009) and Billat et al. (1999) concluded that three sessions per week did not result in any further increases in physiological parameters but could in fact induce overtraining. These sessions were at least 48 hours apart and were performed at 100% PPO, as identified in the final four minute bout of the incremental step test. These sessions took place twice each week over the eight week period with 16 HIIT sessions being completed in total. During week one and two 6 intervals were completed in each session, during week three and four 7 intervals were completed and from week five to eight, eight intervals were completed in each session. The intervals were 2.5 minutes in duration and recovery between each interval was similar to that used in previous studies (Kubukeli et al. 2002, Driller et al. 2009). During the recovery participants continued to row at 40% PPO until their heart rate returned to ≤70% of its maximum as identified during the incremental step test. Once this target heart rate was reached the next interval commenced immediately. However, in the case that 5 minutes rowing at 40% PPO failed to reduce heart rate the participant ceased rowing.
and waited until the heart rate dropped to the target value upon which the next interval commenced immediately.

6. 3. 3. Statistical Analyses

All statistical analyses were performed using the statistical package SPSS (Release 20.0). Means and standard deviations were calculated for all measured variables for all subjects by group and by sex. Normality of the data was verified using the Shapiro-Wilk test. An independent t-test was run to ensure there was no statistical difference between the two groups prior to the training intervention. A paired samples t-test was run separately in each group in order to determine the effect each training program had on the dependent variables. A two-way mixed analysis of variance (ANOVA) with repeated measures was then used to compare changes in the independent variables over time (between the pre and post test results) and between groups. Statistical significance was set at $p < 0.05$ for all analyses.

6. 4. Results

Descriptive statistics for all baseline physiological and performance variables measured are summarised in Table 6.2. No statistically significant physiological or performance differences existed between groups prior to training.
Table 6.2: Mean baseline values for all variables measured.

<table>
<thead>
<tr>
<th>Variable</th>
<th>LSD</th>
<th>HITT</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Descriptive Variables</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>22 ± 4</td>
<td>22 ± 4</td>
</tr>
<tr>
<td>Body Mass (kg)</td>
<td>83.0 ± 11.7</td>
<td>85.0 ± 7.9</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>184.1 ± 7.6</td>
<td>186.0 ± 4.8</td>
</tr>
<tr>
<td>Skinfold (mm)</td>
<td>84.9 ± 41.2</td>
<td>85.4 ± 35.2</td>
</tr>
<tr>
<td>BF (%)</td>
<td>20.1 ± 7.9</td>
<td>20.3 ± 6.8</td>
</tr>
<tr>
<td>Lean (%)</td>
<td>76.7 ± 8.5</td>
<td>76.6 ± 7.3</td>
</tr>
<tr>
<td>FF (%)</td>
<td>80.7 ± 8.5</td>
<td>80.6 ± 7.3</td>
</tr>
<tr>
<td><strong>Performance Variables</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2000 m TT (s)</td>
<td>415.6 ± 38.1</td>
<td>407.2 ± 23.2</td>
</tr>
<tr>
<td>(T_{\max}) (s)</td>
<td>609.3 ± 200.9</td>
<td>688.8 ± 160.5</td>
</tr>
<tr>
<td><strong>Physiological Variables</strong></td>
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<td></td>
</tr>
<tr>
<td>(\dot{V}O_{2\max}) (l.min(^{-1}))</td>
<td>4.62 ± 0.95</td>
<td>4.71 ± 0.61</td>
</tr>
<tr>
<td>(\dot{V}O_{2\max}) (ml.kg.min(^{-1}))</td>
<td>55.6 ± 8.9</td>
<td>56.0 ± 9.9</td>
</tr>
<tr>
<td>(W\dot{VO}_{2\max}) (W)</td>
<td>297.5 ± 64.0</td>
<td>305.2 ± 40.7</td>
</tr>
<tr>
<td>ECON (ml/W)</td>
<td>13.4 ± 1.8</td>
<td>12.7 ± 1.2</td>
</tr>
<tr>
<td>(W_{\text{LT}}) (W)</td>
<td>156.1 ± 34.3</td>
<td>162.3 ± 35.6</td>
</tr>
<tr>
<td>(W_{\text{AT}}) (W)</td>
<td>234.4 ± 46.3</td>
<td>252.6 ± 39.0</td>
</tr>
<tr>
<td>(W_{2\text{mmol/l}}) (W)</td>
<td>180.2 ± 41.6</td>
<td>197.7 ± 39.7</td>
</tr>
<tr>
<td>(W_{4\text{mmol/l}}) (W)</td>
<td>226.8 ± 48.5</td>
<td>245.2 ± 38.7</td>
</tr>
<tr>
<td>PPO (W)</td>
<td>311.0 ± 72.0</td>
<td>337.6 ± 53.6</td>
</tr>
<tr>
<td>(W_{\max}) (W)</td>
<td>452.7 ± 96.9</td>
<td>475.4 ± 86.8</td>
</tr>
<tr>
<td>(F_{\max}) (N)</td>
<td>1124.3 ± 176.3</td>
<td>1142.4 ± 165.7</td>
</tr>
<tr>
<td>Peak La (mmol.l(^{-1}))</td>
<td>13.3 ± 2.4</td>
<td>13.7 ± 1.8</td>
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<tr>
<td>2k Post La (mmol.l(^{-1}))</td>
<td>13.1 ± 2.6</td>
<td>12.4 ± 2.3</td>
</tr>
<tr>
<td><strong>Blood Gas Variables</strong></td>
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<td></td>
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<tr>
<td>2k pH</td>
<td>7.43 ± 0.02</td>
<td>7.40 ± 0.02</td>
</tr>
<tr>
<td>2k HCO(_3) (mmol.l(^{-1}))</td>
<td>24.40 ± 1.40</td>
<td>24.96 ± 2.55</td>
</tr>
<tr>
<td>Step pH</td>
<td>7.41 ± 0.02</td>
<td>7.41 ± 0.02</td>
</tr>
<tr>
<td>Step HCO(_3) (mmol.l(^{-1}))</td>
<td>25.41 ± 1.92</td>
<td>25.6 ± 1.58</td>
</tr>
</tbody>
</table>

Date presented as mean ± SD
Table 6.3 presents an overview of changes in all measured variables from pre to post testing in both the LSD and HIIT group. As displayed in figure 6.2 HIIT was associated with a significant improvement in 2000 m time trial performance ($p = 0.011$). However, this change was not significantly different compared with the change in 2000 m time trial performance in the LSD ($p = 0.595$). Absolute and relative $\dot{V}O_{2\text{max}}$ did not significantly change in either group although HIIT resulted in significantly greater improvements in both absolute $\dot{V}O_{2\text{max}}$ ($p = 0.035$) and relative $\dot{V}O_{2\text{max}}$ ($p = 0.032$) when compared with LSD. HIIT resulted in a significant improvement in $W'_{LT}$ ($p = 0.005$) and the change in $W'_{LT}$ was also significantly different between the groups ($p = 0.008$). HIIT was also associated with significant changes in PPO ($p = 0.017$) and BF% ($p = 0.004$), however these were not significant when compared to LSD. $W'_{2\text{mmol/l}}$ and $W'_{4\text{mmol/l}}$ increased significantly in both the HIIT ($p = 0.001$ and $p = 0.002$ respectively) and LSD ($p = 0.006$ and $p = 0.001$ respectively) groups although the changes were not significantly different between the groups ($p = 0.262$ and $p = 0.378$ respectively).

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{figure6.2.png}
\caption{Mean ($\pm$ 95% CI) percentage change in measured variables from pre to post testing. ***$p < 0.001$; **$p < 0.01$; *$p < 0.05$ Significant change from pre to post training within groups. † $p < 0.05$ Significant change from pre to post training between groups.}
\end{figure}
Table 6.3: Changes in all measured variable from pre to post testing.

<table>
<thead>
<tr>
<th>Variable</th>
<th>LSD</th>
<th>HUUT</th>
<th>%Δ</th>
<th>LSD</th>
<th>HUUT</th>
<th>%Δ</th>
</tr>
</thead>
<tbody>
<tr>
<td>2k TT (s)</td>
<td>415.6 ± 38.1</td>
<td>411.0 ± 31.2</td>
<td>-1.12</td>
<td>407.2 ± 23.2</td>
<td>400.2 ± 22.6</td>
<td>-1.72*</td>
</tr>
<tr>
<td>Body Mass (kg)</td>
<td>83.0 ± 11.7</td>
<td>82.8 ± 11.0</td>
<td>-0.24</td>
<td>85.0 ± 7.9</td>
<td>85.0 ± 7.5</td>
<td>-0.01</td>
</tr>
<tr>
<td>Skinfold (mm)</td>
<td>84.9 ± 41.2</td>
<td>77.2 ± 32.0</td>
<td>-8.6</td>
<td>85.4 ± 35.2</td>
<td>78.0 ± 31.0</td>
<td>-9.1**</td>
</tr>
<tr>
<td>BF (%)</td>
<td>20.1 ± 7.9</td>
<td>19.3 ± 7.3</td>
<td>-3.69</td>
<td>20.3 ± 6.8</td>
<td>18.8 ± 7.4</td>
<td>-7.35**</td>
</tr>
<tr>
<td>Lean (%)</td>
<td>76.7 ± 8.5</td>
<td>77.8 ± 7.9</td>
<td>+1.39</td>
<td>76.6 ± 7.3</td>
<td>78.1 ± 7.7</td>
<td>+1.91**</td>
</tr>
<tr>
<td>FF (%)</td>
<td>80.7 ± 8.5</td>
<td>81.8 ± 7.9</td>
<td>+1.38</td>
<td>80.6 ± 7.3</td>
<td>82.1 ± 7.8</td>
<td>+1.83**</td>
</tr>
<tr>
<td>VO2max (L.min⁻¹)</td>
<td>4.62 ± 0.95</td>
<td>4.54 ± 0.83</td>
<td>-1.69</td>
<td>4.71 ± 0.61</td>
<td>5.01 ± 0.67</td>
<td>+6.42†</td>
</tr>
<tr>
<td>V̇O2max (ml.kg.min⁻¹)</td>
<td>55.6 ± 8.9</td>
<td>54.8 ± 7.7</td>
<td>-1.42</td>
<td>56.0 ± 9.9</td>
<td>59.4 ± 9.5</td>
<td>+6.12†</td>
</tr>
<tr>
<td>ẆO2max (W)</td>
<td>297.5 ± 64.0</td>
<td>301.1 ± 57.4</td>
<td>+1.21</td>
<td>305.2 ± 40.7</td>
<td>320.9 ± 44.8</td>
<td>+5.13</td>
</tr>
<tr>
<td>ECON (ml/W)</td>
<td>13.4 ± 1.8</td>
<td>12.96 ± 1.86</td>
<td>-3.14</td>
<td>12.7 ± 1.2</td>
<td>12.7 ± 1.1</td>
<td>+0.08</td>
</tr>
<tr>
<td>ẆAT (W)</td>
<td>156.1 ± 34.3</td>
<td>157.9 ± 31.4</td>
<td>+1.15</td>
<td>162.3 ± 35.6</td>
<td>190.3 ± 37.1</td>
<td>+17.25**†</td>
</tr>
<tr>
<td>Ẇ2mmol/l (W)</td>
<td>234.4 ± 46.3</td>
<td>233.5 ± 44.6</td>
<td>-0.38</td>
<td>252.6 ± 39.0</td>
<td>261.9 ± 46.8</td>
<td>+3.70</td>
</tr>
<tr>
<td>Ẇ4mmol/l (W)</td>
<td>180.2 ± 41.6</td>
<td>199.4 ± 43.7</td>
<td>+10.65**</td>
<td>197.7 ± 39.7</td>
<td>225.78 ± 41.1</td>
<td>+14.22**</td>
</tr>
<tr>
<td>Ẇmax (W)</td>
<td>226.8 ± 48.5</td>
<td>243.8 ± 50.7</td>
<td>+7.50***</td>
<td>245.2 ± 38.7</td>
<td>267.4 ± 44.0</td>
<td>+9.06**</td>
</tr>
<tr>
<td>Ḟmax (N)</td>
<td>452.7 ± 96.9</td>
<td>451.5 ± 88.5</td>
<td>-0.27</td>
<td>475.4 ± 86.8</td>
<td>488.2 ± 72.2</td>
<td>+2.69</td>
</tr>
<tr>
<td>PPO (W)</td>
<td>311.0 ± 72.0</td>
<td>325.3 ± 67.3</td>
<td>+4.60</td>
<td>337.6 ± 53.6</td>
<td>358.8 ± 56.4</td>
<td>+6.29*</td>
</tr>
<tr>
<td>Ṫmax (s)</td>
<td>609.3 ± 200.9</td>
<td>615.1 ± 209.3</td>
<td>+0.95</td>
<td>688.8 ± 160.5</td>
<td>873.9 ± 337.2</td>
<td>+26.88</td>
</tr>
<tr>
<td>Peak La (mmol.l⁻¹)</td>
<td>13.3 ± 2.4</td>
<td>12.8 ± 2.0</td>
<td>-3.92</td>
<td>13.7 ± 1.8</td>
<td>12.6 ± 1.9</td>
<td>-8.00</td>
</tr>
<tr>
<td>2k Post La (mmol.l⁻¹)</td>
<td>13.1 ± 2.6</td>
<td>12.1 ± 2.1</td>
<td>-8.19</td>
<td>12.4 ± 2.3</td>
<td>12.2 ± 2.3</td>
<td>-0.09</td>
</tr>
<tr>
<td>2k pH</td>
<td>7.03 ± 0.08</td>
<td>7.03 ± 0.08</td>
<td>-0.02</td>
<td>7.06 ± 0.11</td>
<td>7.06 ± 0.09</td>
<td>-0.02</td>
</tr>
<tr>
<td>2k HCO3 (mmol.l⁻¹)</td>
<td>7.03 ± 1.11</td>
<td>7.87 ± 1.88</td>
<td>+11.95</td>
<td>7.52 ± 2.69</td>
<td>8.11 ± 1.87</td>
<td>+7.83</td>
</tr>
<tr>
<td>Step pH</td>
<td>7.11 ± 0.09</td>
<td>7.11 ± 0.09</td>
<td>-0.03</td>
<td>7.10 ± 0.75</td>
<td>7.09 ± 0.07</td>
<td>-0.09</td>
</tr>
<tr>
<td>Step HCO3 (mmol.l⁻¹)</td>
<td>9.47 ± 1.97</td>
<td>9.35 ± 2.19</td>
<td>-1.27</td>
<td>8.93 ± 2.01</td>
<td>8.87 ± 2.26</td>
<td>-0.75</td>
</tr>
</tbody>
</table>

***p < 0.001; **p < 0.01; *p < 0.05 Significant change from pre to post training within groups. † p < 0.05 Significant change from pre to post training between groups.
There was no significant change in resting buffering capacity, as identified through resting pH and HCO$_3^-$ levels and presented in figure 6.3.

Figure 6.3: Baseline pH and HCO$_3^-$ levels pre and post training.

Figure 6.4 displays the changes in peak La and post exercise pH and HCO$_3^-$, measured following the 2000 m TT, pre and post training. There were no significant changes in peak La or post exercise pH or HCO$_3^-$ following HIIT or LSD.

Figure 6.4: Peak blood lactate, post exercise pH and HCO$_3^-$ following a 2000 m TT pre and post training.
6.5. Discussion

The aim of this study was to compare the effects of HIIT and traditional, low intensity training on well-trained rowers. In addition, blood HCO$_3$, pH and lactate levels were measured to elucidate possible mechanisms underlying any performance differences between the two training groups. The main findings of this study were that HIIT resulted in significantly greater improvements in absolute and relative $\dot{V}O_{2\text{max}}$ and $W_L$ than LSD.

Aerobic training has traditionally been the training focus for well-trained rowers whom, similar to other endurance athletes, perform ~75% of training at intensities below the lactate threshold, despite competing at much higher intensities (Esteve-Lanao et al. 2007). Rowers have in fact been observed to work at 96-98% of their maximal aerobic capacity for the majority of a race (Hagerman et al. 1979). However, improvements in performance become difficult to attain for well-trained athletes and Billat et al. (2001) have suggested that additional increases in aerobic training do not further improve endurance performance or associated physiological variables such as $\dot{V}O_{2\text{max}}$, economy, anaerobic threshold or oxidative muscle enzymes. It has therefore been suggested that the combination of traditional endurance training and HIIT may optimise the development of aerobic muscle characteristics and enhance performance (Laursen 2010, Laursen and Jenkins 2002).

Several studies have reported improvements in performance in running (Acevedo and Goldfarb 1989, Smith et al. 2003, Smith et al. 1999, Houmard et al. 1994) and cycling (Laursen et al. 2002b, Westgarth-Taylor et al. 1997, Lindsay et al. 1996) following HIIT and Paton and Hopkins (2004) have reported that HIIT involving maximal and supramaximal intervals is associated with improvements in endurance performance of 3 - 8.3%. There is, however, a paucity of research regarding the effect of HIIT on 2000 m rowing performance. The results of the present study revealed a significant improvement in 2000 m time trial performance following 8 weeks of HIIT ($p = 0.011$) and although a significant improvement was not observed in the LSD group ($p = 0.237$), the improvement with HIIT was not significantly different from the LSD group ($p = 0.595$). It would therefore appear that both methods of training were successful in eliciting an improvement in performance. It is worth highlighting however that the performance improvement following HIIT was greater than following LSD despite baseline performance times being faster for the HIIT group. From a practical perspective HIIT was associated with a mean improvement of 7
seconds (-1.72%), comparable to 4.25 boat lengths in a typical single scull 2000 m race, while LSD resulted in a mean improvement of 4.6 seconds (-1.12%) which is comparable to 2.75 boat lengths in the same 2000 m race. This improvement following HIIT was similar to that reported by Driller et al. (2009) who observed an 8 second improvement in 2000 m time-trial performance with well-trained rowers following HIIT. In contrast to the present study, Driller et al. (2009) found that this improvement in performance was significantly greater than the improvements in the control group. The main difference between these studies was the duration over which they were completed. The present study involved HIIT training over an eight week period while Driller et al. (2009) utilised a shorter time period of four weeks. This may indicate that a shorter time period may be sufficient to elicit an improvement in 2000 m rowing performance through the use of HIIT and it is possible that the LSD group in the present study had adequate time over the eight week period to catch up with HIIT group. Little information is available regarding the rate at which endurance performance improves following HIIT. Studies have observed improvements in endurance running and cycling performance following three weeks (Stepto et al. 1999), four weeks (Smith et al. 1999, Lindsay et al. 1996, Laursen et al. 2002b, Smith et al. 2003, Laursen et al. 2005), six weeks (Westgarth-Taylor et al. 1997) and eight weeks (Acevedo and Goldfarb 1989) of HIIT. However, control groups were not utilised in all HIIT studies making improvements in performance difficult to interpret and given the dearth of research investigating the effects of HIIT on well-trained rowers, further research investigating the optimal time frame to perform HIIT is required. Indeed, Laursen and Jenkins (2002) suggest that in order to maximise the improvements in endurance performance associated with HIIT, regular assessments of training status and subsequent adjustments to HIIT programmes may be required. In the present study it may have been appropriate to retest the participants following four weeks of HIIT to re-evaluate PPO and adjust the HIIT sessions accordingly in order to observe greater between group differences in 2000 m time trial performance.

$\dot{V}O_{2\text{max}}$ has been identified as an important predictor of 2000 m rowing performance (Cosgrove et al. 1999, Ingham et al. 2002, Kramer et al. 1994, Secher et al. 1982) and a direct relationship between placing in an international regatta and absolute $\dot{V}O_{2\text{max}}$ has been observed (Secher et al. 1982). HIIT has been shown to be a very effective means of increasing $\dot{V}O_{2\text{max}}$ in untrained subjects (Laursen and Jenkins 2002, Hickson et al. 1977, MacDougall et al. 1998, Tabata et al. 1996). However, the effect of HIIT on $\dot{V}O_{2\text{max}}$ in well-
trained athletes is equivocal. Acevedo and Goldfarb (1989) and Daniels et al. (1978) both reported improvements in performance following HIIT independent of improvements in $\dot{V}O_{2\text{max}}$ in well-trained participants while other studies have reported an improvement in $\dot{V}O_{2\text{max}}$ following HIIT (Driller et al. 2009, Brooks et al. 2000, Billat et al. 2002a, Laursen et al. 2005, Smith et al. 1999). Improvements in $\dot{V}O_{2\text{max}}$ may occur through increases in oxygen delivery and/or oxygen utilisation by active muscles (Holloszy and Coyle 1984). In the present study absolute $\dot{V}O_{2\text{max}}$ improved significantly more following HIIT than LSD ($p = 0.032$). HIIT resulted in a 6.42% mean improvement in $\dot{V}O_{2\text{max}}$ while LSD resulted in a mean reduction in $\dot{V}O_{2\text{max}}$ of -1.69%. The magnitude of this improvement in $\dot{V}O_{2\text{max}}$ following HIIT is similar to that observed in other studies (Driller et al. 2009, Laursen et al. 2002b, Billat et al. 2002a, Smith et al. 1999). Billat et al. (2002a) reported an improvement in $\dot{V}O_{2\text{max}}$ of 5.4% ($p < 0.01$) in elite marathon runners following eight weeks of HIIT while Laursen et al. (2002b) found an 8% improvement in $\dot{V}O_{2\text{max}}$ ($p < 0.05$) following four weeks of HIIT. Furthermore, Driller et al. (2009) observed a 7% ($p < 0.05$) increase in $\dot{V}O_{2\text{max}}$ in well-trained rowers following four weeks of HIIT and concluded that this increase in $\dot{V}O_{2\text{max}}$ was likely to be the main contributing factor to the improved 2000 m time trial performance following HIIT. The improvement of $\dot{V}O_{2\text{max}}$ observed in the present study supports the view that the benefit of HIIT on $\dot{V}O_{2\text{max}}$ is dependent on the intensity of the intervals performed with HIIT sessions at or close to the velocity or power corresponding to $\dot{V}O_{2\text{max}}$ the most effective at eliciting additional improvements in $\dot{V}O_{2\text{max}}$ in already well-trained athletes.

The accumulation of blood lactate during incremental exercise tests is a measure commonly used to evaluate the effects of training, to establish individualised training intensities and to predict performance and the way in which blood lactate levels change with increased exercise intensity can provide valuable information about how an athlete is adapting to training (Bourdon 2000). The blood lactate response to exercise has been suggested to be a more sensitive estimate of endurance performance capability than $\dot{V}O_{2\text{max}}$ (Weltman 1995) and it has been demonstrated that indices of blood lactate during submaximal exercise and the ability to elicit less lactate for a given power output are highly correlated with rowing performance (Cosgrove et al. 1999, Ingham et al. 2002, Messonnier et al. 1997, Riechman et al. 2002, Faff et al. 1993, Womack et al. 1996, Womack et al. 1992, Klusiewicz 1993). The results of the present study revealed a significant improvement in $W_{LT}$ following eight
weeks of HIIT compared to LSD (p = 0.008). A 17.25% significant mean improvement in \( W_{LT} \) was observed following HIIT (p = 0.005, 28 W) compared to a 1.15% improvement following LSD (p = 0.731, 1.8 W). Training below the lactate threshold typically predominates rowing training programs. However, this result suggests that training above the lactate threshold is necessary to stimulate the development of the lactate response in well-trained athletes as previously suggested by Ingham et al. (2008). This is a significant finding in terms of training prescription as it indicates that HIIT does not compromise aerobic adaptations and has in fact the potential to stimulate aerobic adaptations in well-trained rowers. It has previously been shown that running speed at the lactate threshold was highly correlated with the improvement in performance following HIIT (Billat et al. 2002b). It has been suggested that HIIT could delay the accumulation of lactate in the blood to a greater extent than low intensity continuous training by increasing the oxidative capacity of a greater number of muscle fibres (Poole and Gaesser 1985) or HIIT may afford the ability to tolerate the presence of lactate, enhancing lactate removal and allowing better tolerance of high-intensity activity (Brooks et al. 2000).

The results of the study indicate that HIIT can improve \( \dot{V}O_{2\text{max}} \) and \( W_{LT} \) significantly more than LSD. However, there were also some non-statistical trends in the results of the study. The fact that these trends were non-statistical may be due to the fact that the rowers participating were already well-trained and the elicitation of further adaptations in well-trained athletes is difficult, particularly over short time periods such as eight weeks. HIIT resulted in an improvement in \( W\dot{O}_{2\text{max}} \) of 5.13% (15.7 W) while the LSD group experienced a noticeable smaller improvement of 1.21% (3.6 W). Several studies have observed \( W\dot{O}_{2\text{max}} \) to be highly correlated with 2000 m rowing ergometer performance (Nevill et al. 2011, Ingham et al. 2002, Cosgrove et al. 1999, Jurimae et al. 2000) and it has been suggested that \( W\dot{O}_{2\text{max}} \) may be a valuable tool for monitoring endurance performance (Legaz Arrese et al. 2005, Mikulic 2011). Indeed, Nevill et al. (2011) identified \( W\dot{O}_{2\text{max}} \) as the best single predictor of performance, explaining 95.3% of the variance in rowing speed in elite rowers while Chapter 5 of this thesis had similar results with \( W\dot{O}_{2\text{max}} \) emerging as the best predictor of performance accounting for 96.5% of the variance in performance (\( R^2 = 0.965, p < 0.001 \)). The trend towards an increase in \( W\dot{O}_{2\text{max}} \) following HIIT is similar to that observed in previous studies (Smith et al. 1999, Billat et al. 1999, Smith et al. 2003, Esfarjani and Laursen 2007) and suggests that HIIT provides a better
stimulus than LSD to elicit improvements in $\dot{W}VO_{2\text{max}}$. Improvements in $\dot{W}VO_{2\text{max}}$ may prove vital to athletes who have reached a plateau in $\dot{VO}_{2\text{max}}$, whereby no further improvements in $\dot{VO}_{2\text{max}}$ may be attained but the power which is produced at $\dot{VO}_{2\text{max}}$ may increase. This was recently observed by Mikulic (2011) who noted that while $\dot{VO}_{2\text{max}}$ reached a plateau in elite rowers, $\dot{W}VO_{2\text{max}}$ continued to increase.

There was also a non-statistical trend in $T_{\text{max}}$. $T_{\text{max}}$ is the length of time exercise at $\dot{V}O_{2\text{max}}$ can be sustained for and studies have shown that increasing $T_{\text{max}}$ through HIIT may result in substantial improvements in performance (Billat et al. 2000b, Billat 2001a). The results of this present study revealed a 26.88% improvement in $T_{\text{max}}$ following HIIT compared to a 0.95% improvement following LSD. This increase in $T_{\text{max}}$, although not significant, is similar in magnitude to the improvement observed in other studies (Smith et al. 2003, Esfarjani and Laursen 2007). The fact that a large improvement was observed without any statistical significance may be explained by that fact that $T_{\text{max}}$ is a highly individualised measure even between individuals with similar $\dot{V}O_{2\text{max}}$ values (Billat et al. 1994b). Although little work has been done to investigate $T_{\text{max}}$ in rowers, Billat et al. (1994b) demonstrated that $T_{\text{max}}$ was reproducible in well-trained runners ($r = 0.86; p < 0.05$) and concluded that $T_{\text{max}}$ was a reliable group measure which could be used to study the effects of training. $T_{\text{max}}$ has also been reported to be an important factor in optimising the prescription of HIIT (Smith et al. 2003, Hill and Rowell 1996, Laursen and Jenkins 2002, Billat 2001a). However, while $T_{\text{max}}$ has been developed as a practical method for determining the appropriate duration of interval bouts in well-trained runners, and to some extent well-trained cyclists, this concept has not been explored with rowers and further investigation is required.

In order to elucidate possible mechanisms underlying performance differences following eight weeks of HIIT and LSD, blood HCO$_3^-$, pH and lactate levels were measured. The mechanisms responsible for performance changes in well-trained athletes following HIIT are equivocal and an increase in buffering capacity is one such adaptation which has been purported to be responsible for the improvement in endurance performance associated with HIIT (Weston et al. 1997, Gibala et al. 2006). Buffering capacity is the ability of the tissue to resist pH changes during an acid load experienced with high intensity exercise (Juel 1998). Training at intensities above the lactate threshold, as observed with HIIT, causes the production of lactate and H$^+$ to exceed their removal resulting in an accumulation of H$^+$ and a decrease in pH, placing stress on the mechanisms involved in
pH regulation in addition to potentially impairing exercise performance (Bishop et al. 2009) and causing a reduction in power output (Bishop et al. 2003). The ability of muscle to buffer increased concentrations of H\(^+\) is an important factor during intense exercise and therefore those with a greater ability to regulate H\(^+\) during intense exercise may be better able to maintain intense muscle contractions and Edge et al. (2006c) have suggested that regular high intensity exercise training may result in an elevated buffering capacity. An improvement in blood buffering capacity can be identified through an increase in post-exercise blood pH in addition to a greater decrease in blood HCO\(_3\), measured by blood gas analysis, and a greater increase in blood lactate concentration. In the present study there was no significant change in resting buffering capacity following eight weeks of HIIT or LSD training. In addition there was no significant change in post-exercise blood pH, HCO\(_3\) or lactate for either group. While several studies have reported that HIIT is responsible for increasing resting muscle buffering capacity (Bishop et al. 2009, Edge et al. 2006a, Edge et al. 2006b, Weston et al. 1997, Gibala et al. 2006) the results of other studies are in agreement with the present study, whereby no change in buffering capacity was observed following HIIT (Harmer et al. 2000, Nevill et al. 1989). Differences in the results of studies investigating the effect of HIIT on buffering capacity may be attributed to differences in exercise intensity, recovery periods or mode of training utilised or basal bicarbonate levels, all of which have the potential to affect H\(^+\) accumulation (Edge et al. 2006c). In this present study it would be reasonable to suggest that the length of recovery periods used between intervals may have inhibited an improvement in buffering capacity. Studies which have reported improvements in buffering capacity have employed short recovery periods of less than 1 minute between intervals (Bell and Wenger 1988, Weston et al. 1997, Edge et al. 2006b) while studies which did not observe significant improvements in buffering capacity (Harmer et al. 2000, Nevill et al. 1989) predominately involved longer recovery periods similar to the individualised recovery periods utilised in the present study. A longer recovery period may alter the training stimulus necessary to improve buffering capacity as a longer recovery allows for greater removal of H\(^+\) prior to subsequent intervals (Sahlin et al. 1976). A large accumulation of H\(^+\) during HIIT sessions is suggested to be an important stimulus in the improvement of buffering capacity and the removal of H\(^+\) during long recovery periods would reduce H\(^+\) accumulation during training therefore reducing the potential to increase buffering capacity.
6. 6. Summary and Conclusion

HIIT has been observed to be an effective means for improving performance and associated physiological variables in well-trained endurance runners (Acevedo and Goldfarb 1989, Smith et al. 2003, Smith et al. 1999, Houmard et al. 1994) and cyclists (Laursen et al. 2002b, Westgarth-Taylor et al. 1997, Lindsay et al. 1996). There is however a dearth of research investigating the effects of HIIT on well-trained rowers. The results of the present study revealed that 8 weeks of HIIT in well-trained rowers resulted in significantly greater improvements in $\dot{V}O_{2\text{max}}$ and $\dot{W}_{\text{LT}}$ than traditional long, slow, distance training. This improvement in $\dot{W}_{\text{LT}}$ following HIIT may have significant implications for training prescription as HIIT resulted in large changes in this aerobic parameter. The results also showed a significant improvement in 2000 m rowing performance following HIIT, although this improvement was not significantly greater than the improvement observed with LSD. An investigation into blood buffering capacity as a mechanism underlying changes associated with HIIT revealed that blood buffering capacity was not altered following eight weeks of training. Further research is required in order to optimise HIIT for well-trained rowers, with regard to frequency, intensity, recovery period and duration of both the intervals and HIIT program. Further research is also required to investigate the underlying mechanisms responsible for performance and physiological changes following HIIT.
Chapter 7. Thesis Summary, Conclusions and Future Recommendations
7. 1. Introduction

As outlined in Chapter 1 successful endurance performance in sports such as rowing, running and cycling requires not only a large aerobic capacity but also muscular strength and anaerobic power necessary for attacking, pace changing and final sprints. Due to the importance of the aerobic capacity for prolonged endurance performance, endurance athletes (cyclists, rowers, cross-country skiers, runners) spend a large proportion of training time training at intensities below the lactate threshold despite competing at much higher intensities (Esteve-Lanao et al. 2007, Steinacker et al. 1998). However, despite the high aerobic contribution and the importance of the aerobic capacity to endurance performance, \( \dot{V}O_{2\text{max}} \) plateaux in elite endurance athletes with further increases in training volumes failing to yield any further improvements (Billat 2001a). In addition, the increase in aerobic training hours which has been observed over the past thirty years in endurance athletes has resulted in less variation in aerobic endurance and an increased importance on the anaerobic contribution to performance (McNeely 2011a). It is therefore apparent the improvements in endurance performance must be realised through means other than traditional, long, slow distance (LSD) training.

The aim of this thesis was to investigate the impact of alternative methods of training on well-trained endurance athletes in order to maximise training adaptations of the key biomechanical and physiological determinants associated with endurance performance. To address this aim a review of literature (Chapter 2) and four original research studies (Chapter 3-6) were completed. Chapter 3 investigated the effects of maximal stretch shortening cycle (SSC) fatigue on the biomechanical performance of well-trained strength athletes. Fatigue, the inability of the muscle to generate force, has been shown to be an important factor in exercise performance and functional capacity (Mileva et al. 2009) and provides the opportunity to investigate the acute effects of training on physiological and biomechanical properties of athletes. The recovery process following maximal SSC fatigue was also investigated to establish if a maximal SSC fatigue workout could elicit a potential post activation potentiation (PAP) effect in well-trained strength athletes. Based on the results of Chapter 3 and the suggestion that power-trained and strength-trained athletes are better able to elicit PAP than endurance athletes (Smith and Fry 2007, Chiu et al. 2003), the aim of Chapter 4 was to investigate the acute effects of maximal SSC fatigue on endurance athletes and to identify any PAP effect. The SSC is the basis of plyometrics, a popular
method of strength and power training. Concurrent training, the performance of both strength and endurance training, has been reported to improve performance in well-trained endurance athletes (Paavolainen et al. 1999, Spurrs et al. 2003). As such endurance athletes may also benefit from other training methods. The aim of Chapter 5 was therefore to identify the key physiological determinants of 2000 m rowing ergometer performance in order to establish which parameters if targeted in training program design may result in an improvement in performance.

High intensity interval training (HIIT) has been observed to induce improvements in endurance performance and associated physiological variables in well-trained endurance runners (Acevedo and Goldfarb 1989, Smith et al. 2003, Smith et al. 1999, Houmard et al. 1994) and cyclist (Laursen et al. 2002b, Westgarth-Taylor et al. 1997, Lindsay et al. 1996). The aim of Chapter 6 was to address a significant gap in the existing literature and investigate the effect of HIIT on well-trained rowers. While the results of each study have been discussed individually, the aim of this chapter (Chapter 7) is to combine all the findings and explore the overall conclusions from the body of work in this thesis. Limitations of the work will also be considered and recommendations for future research will be suggested.

7.2. Thesis Summary and Main Findings

Basic human muscle function typically involves the SSC, a phenomenon whereby an eccentric contraction precedes a concentric contraction with minimum delay between the eccentric and concentric phase (Komi 2000). The SSC is a form of muscle function in which all of the key components of performance sources, mechanical, neural and metabolic, are stressed and therefore the SSC provides a unique model to investigate the effects of neuromuscular fatigue on performance (Komi 2000). Chapter 3 revealed the immediate, impeding effect of maximal SSC fatigue on the performance of subsequent SSC activities. This effect was realised through not only the performance outcome measure (height jumped) but also through the biomechanics of performance (GRF, CT, RSI, k_ver) indicating that the effectiveness of the SSC was significantly reduced resulting in a decrease in performance. Minimal CT and a stiff leg action are required for an effective SSC both of which were significantly altered immediately following maximal SSC fatigue. A short CT, or amortization phase, is essential for the subsequent concentric contraction to elicit the
advantages of both the stored elastic energy and the stretch reflex (Schmidtbleicher 1992). An effective level of leg spring stiffness is also necessary for effective utilisation of the stored elastic energy (Butler et al. 2003) and an association between reduced stretch reflex sensitivity and decreased stiffness following fatiguing SSC exercise has been observed (Avela and Komi 1998). The significant changes in CT and leg spring stiffness in addition to significant reductions in GRF, RSI and height jump indicate that effectiveness of the SSC was reduced. It is possible that this was a result of a loss of storage and reutilisation of elastic energy or a reduction in stretch reflex sensitivity.

Typical recovery following SSC fatigue is purported to occur in a bimodal fashion (Komi 2000). The dramatic decline in performance following the maximal SSC protocol utilised in Chapter 3 conforms with the immediate effects of SSC fatigue observed in previous SSC fatigue studies (Horita et al. 1999, Komi 2000). However, while a recovery to baseline values was expected, further inspection of the recovery revealed an enhancement in the performance of the variables examined above baseline values. This enhancement may be attributable to PAP, an acute improvement in performance as a result of prior muscle activation which can be elicited in the form of applied performance, contractile property output and/or neural function (Hodgson et al. 2005). During recovery there was a significant increase in GRF in the DJ and large percentage improvements in RSI, $k_{ven}$, and CT in both the DJ and RBJ indicating that subsequent jumps were performed with more force, a stiffer leg spring action, a shortened CT and a greater ability to change quickly from the eccentric to the concentric contraction. These results suggest that although a maximal SSC fatigue workout results in an immediate, dramatic decline in performance it may have the potential to evoke a PAP effect whereby subsequent SSC activities can be performed with a more effective SSC. However, it must also be noted that the performance measure of height jumped was not altered and it may therefore be difficult to attribute the results to PAP.

Chapter 4 employed a methodology analogous to Chapter 3 in order to investigate if a maximal SSC fatigue workout evoked a similar response in endurance athletes as that observed in strength trained athletes. The importance of the SSC to strength trained athletes for sprinting and jumping is well established (Kubo et al. 2000, Kyrolainen and Komi 1995) but it is also suggested that it is important for endurance athletes to utilise the SSC effectively and endurance athletes may benefit from the inclusion of SSC-related
activities in training (Harrison et al. 2004). This may be realised through improvements in neuromuscular characteristics whereby improved leg spring stiffness as a result of strength training may develop the body’s ability to store and utilise elastic energy (Paavolainen et al. 1999). The exhausting SSC fatigue protocol employed evoked a similar response in the endurance athletes as with the strength athletes, with a clear and immediate decrease in performance and biomechanical process of jumping observed indicating a loss in SSC effectiveness. Further examination of the recovery process revealed that the maximal SSC fatigue protocol employed may have the potential to elicit a PAP effect in endurance athletes, similar to that observed with the strength trained athletes. It has been proposed that power-trained and strength-trained athletes are better able to evoke PAP than endurance trained athletes (Smith and Fry 2007, Chiu et al. 2003, Paasuke et al. 2007) due largely to fibre type distribution (Hamada et al. 2000b, Vandervoort and McComas 1983). However, Chapter 4 indicates that given the appropriate stimulus endurance athletes may experience a PAP effect as demonstrated through significant increases in k_{vent} and GRF. This change facilitates a more effective storage and utilisation of elastic energy and suggests an improvement in stretch reflex sensitivity was experienced. However, similar to the strength athletes, it must be noted that any changes observed in height jumped, the performance measure, were trivial which brings into question the presence of a PAP effect.

Despite several studies reporting an interference effect with concurrent training (Bell et al. 2000, Craig et al. 1991, Dudley and Djamil 1985, Sale et al. 1990), concurrent strength and endurance training has also been reported to result in improvements in performance, force, economy and neuromuscular performance in well-trained endurance athletes (runners, cyclists, cross country skiers) given the correct training mode, volume and intensity (Hoff et al. 1999, Spurrs et al. 2003, Osteras et al. 2002, Paavolainen et al. 1999, Mikkola et al. 2007, Paavolainen et al. 1991). The results of Chapter 3 and Chapter 4 examined the acute effects of a maximal SSC fatigue workout and demonstrated that a maximal SSC fatigue workout elicits a similar result in strength and endurance athletes. However, the chronic effects of training should also be investigated and given the fact that \( \dot{V}O_{2\text{max}} \) is known to plateau in well-trained endurance athletes (Billat 2001a, Lacour et al. 2009, Rusko 1987, Messonnier et al. 1998) the impact of alternative methods of training for endurance athletes should be explored. The difficulty to elicit further training gains in well-trained athletes has led many to experiment with alternative training methods. To successfully prescribe
training for well-trained endurance athletes an in-depth knowledge of the specific demands of the sport is pertinent.

The aim of Chapter 5 was to investigate the physiological determinants of 2000 m rowing ergometer performance. The rowing ergometer is a valuable tool for rowing training, the monitoring of training effects and the evaluation of rowing performance (Maestu et al. 2005, Smith and Spinks 1995). In contrast to other endurance sports, such as running and cycling, there is a dearth of information concerning the physiological predictors of rowing performance. The main finding of this study was that $\dot{V}O_{2\text{max}}$ was the strongest correlate of performance, the best predictor of 2000 m rowing performance and was the variable with the most influence on performance.

Once the key physiological parameters were identified, a natural progression was to identify how improvements could be elicited in these parameters and how they varied in response to training. Additional increases in aerobic training are reported to result in no further improvements in endurance performance or associated physiological variables including $\dot{V}O_{2\text{max}}$, $\dot{V}O_{2\text{max}}$ economy, anaerobic capacity or oxidative muscle enzymes in well-trained endurance athletes (Billat et al. 2001). The cautious use of HIIT in addition to traditional long, slow distance endurance training has therefore been suggested to optimise the development of aerobic muscle characteristics and enhance performance (Laursen 2010, Laursen and Jenkins 2002). Chapter 6 investigated the effect of HIIT on well-trained rowers and revealed that eight weeks of HIIT performed at 100% peak power output was a more effective means than traditional endurance training to elicit improvements in $\dot{V}O_{2\text{max}}$ and power output associated with the lactate threshold. HIIT was also associated with a mean improvement of 7 seconds in 2000 m time trial performance compared to a mean improvement of 4.6 seconds following eight weeks of traditional rowing training. Similar to previous studies (Nevill et al. 2011, Ingham et al. 2002, Cosgrove et al. 1999, Jurimae et al. 2000) Chapter 5 identified $\dot{V}O_{2\text{max}}$ as important determinant in 2000 m rowing performance indicating that training strategies with the ability to elicit improvements in $\dot{V}O_{2\text{max}}$ should be implemented. HIIT resulted in a 5.13% improvement in $\dot{V}O_{2\text{max}}$ while traditional training resulted in an improvement of 1.21%.
7.3. Thesis Conclusions

The following conclusions can be drawn based on the aforementioned work:

- Maximal SSC fatigue results in an immediate decline in the performance of subsequent SSC activities in both strength and endurance athletes due to a decrease in the effectiveness of the SSC with a loss in the ability to store and reutilise stored elastic energy or a reduction in stretch reflex sensitivity. Based on this conclusion the null hypotheses for Study 1 and Study 2 were rejected.

- Maximal SSC fatigue can elicit a PAP effect in biomechanical variables, including ground reaction force and leg spring stiffness, in both strength and endurance athletes.

- PAP is an extremely individualised phenomena and the optimal time when fatigue has subsided and PAP prevails is variable among individuals and caution should be taken to individualise training sessions and indeed PAP may not be evident in all athletes.

- Plyometric activities should not be performed immediately post fatigue as an individualised recovery of up to 300 seconds is required, at which time jumps can be performed with a more effective SSC.

- $\dot{W}O_{2\text{max}}$ is the best predictor of 2000 m rowing ergometer performance in well-trained female rowers.

- $\dot{W}O_{2\text{max}}$ and $W_{\text{max}}$ are the best predictors of 2000 m rowing ergometer performance in well-trained male rowers.

- HIIT is a more effective means than LSD training at eliciting improvements in $\dot{V}O_{2\text{max}}$ in well-trained rowers.

- HIIT is a more effective means than LSD training at eliciting improvements in the power output associated with the lactate threshold.
HIIT is as effective as LSD training at improving 2000 m rowing ergometer performance and \( \dot{W}O_{2\text{max}} \). Based on this finding the first null hypothesis of Study 4 was accepted.

HIIT should be performed at, or close to, peak power output to elicit improvements in performance and associated physiological parameters.

7. 4. Thesis Limitations

- The small sample size in the studies completed must be acknowledged. Given the calibre of athlete required the sample size in each study is relatively small. However, small sample sizes are relatively common when dealing with well-trained athletes.

- The endurance athletes in Chapter 4 were well-trained rowers. Due to the fact that SSC is not utilised during rowing, these athletes were not an ideal selection.

- Despite the fact that rowing ergometers have previously been shown to replicate the biomechanical and physiological demands of actual rowing, evaluation of rowers in laboratory settings does not allow a direct prediction of performance during on-water rowing.

- The statistical analysis in Chapter 5 involved a linear model to investigate the determinants of 2000 m rowing performance. A linear model assumes that for a similar increase in power, the same improvement in rowing velocity will result irrespective of the athlete’s level of performance. An allometic model, as proposed by Nevill et al. (2011), may be useful but given the homogenous nature of the athletes in the study this approach was not used.

- Due to the fact that Chapter 6 involved an eight week training intervention with ten training sessions per week, the reliability of the study depended on the honesty of the subjects to complete all training sessions as requested and to complete the daily training diary. This factor was minimised as much as possible with the help of coaches who were present at all training sessions.
Due to the incremental step test protocol and method used to identify $\dot{W}\dot{VO}_{2\text{max}}$, $T_{\text{max}}$ could not be incorporated into the interval prescription in Chapter 6. Instead a non-individualised interval duration of 2.5 minutes was prescribed.

### 7.5. Recommendations for Future Research

- A maximal SSC fatigue workout may have the potential to elicit a PAP effect in certain biomechanical variables, such as leg spring stiffness and ground reaction force, in both strength and endurance athletes. However, further research investigating this response is required.

- It is recommended that future studies should seek to verify if the results of this study can be replicated for other endurance athletes such as distance runners.

- Future research should investigate if the PAP effect can be manipulated to produce chronic adaptations in endurance athletes.

- Laboratory analysis should be extended to on-water rowing to establish whether a similar relationship exists between actual rowing performance and the various physiological parameters measured.

- Future research is required to determine whether a similar relationship exists between the various physiological parameters measured and rowing performance in lightweight rowers.

- Future research is required in order to optimise HIIT for well-trained rowers, with regard to frequency, intensity, recovery period and duration of both the intervals and the HIIT program.

- Further research should be completed to establish if $T_{\text{max}}$ can be incorporated into the interval prescription for rowers.

- Research is required to investigate the underlying mechanisms responsible for performance and physiological improvements following HIIT.
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190


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Appendices
Volunteer Information Sheet

Supervisor:  Dr. Drew Harrison

Investigator:  Niamh Ni Cheilleachair  9932275

Purpose
The aim of this study is to investigate the effects of sub-maximal exercise on the biomechanical properties and performance of strength trained athletes.

What is required of you?
Prior to inclusion in this study you will be required to undergo screening by completing a pre-test questionnaire in order to ensure you meet the requirements necessary for participation in the study. You will also be asked to submit a copy of your training program for the two weeks prior to testing.

Experimentation
- You will be asked to follow a pre-lactate-test protocol (which is included on the following page)
- You will be required to attend the Biomechanics Laboratory in the PESS department on three occasions.
- One the first day you will become familiar with the jumping technique required and the apparatus being used.
- There will then be two days of testing with a rest of 24 hours between tests.
- You will be requested to refrain from any high intensity exercise in the 48 hours prior to the testing day.
- On the testing day you will undertake a warm up of 5 minutes jogging and each volunteer will also perform 3-4 practice rebound jumps.
- Following the warm up and practice rebound jumps blood lactate will be taken from your earlobe using a “lactate pro” to establish your pre-test blood lactate level.
- The pre-test will consist of 4 drop jump rebound jump sets in order to establish a baseline jump height. All jumps will be performed on the sledge apparatus.
- Each jump must be performed with maximum effort in order to achieve a maximal jump height.
Appendix A: Information Sheet and Consent forms: Study 1

- You will then perform a “Bleep Test”, a 20m multistage fitness test.
- 1 minute after the termination of the fatigue workout you will perform 1 drop jump rebound jumpset. This will be repeated at 2 minutes, 5 minutes and 10 minutes after the termination.
- At 2, 5 and 10 minutes after the fatigue workout your lactate will be measured from a blood sample taken from the ear lobe.
- Following the final assessment at 10 minutes you will undertake a cool-down.
- You will be requested to return to the biomechanics laboratory 24 hours after the initial test in order to evaluate your recovery. You will be required to perform 4 drop jump rebound jump sets.

Potential Risks and Benefits

Risks

1. All blood sampling carries a risk of infection to the volunteer and the experimenter. The technique will take place in a designated area of the teaching laboratory, which will be kept clear for this procedure, and cleaned appropriately.
2. There is a possibility of slight muscle soreness following the completion of an exercise session. This soreness is a natural response to training.
3. Following the test you will experience the discomfort of fatiguing for at least 5 minutes post-exercise. Some embarrassment may be experienced by you by the distress caused by fatiguing exercise.

Benefits

It has been suggested that strength athletes may benefit from aerobic training. Among the benefits is the ability of aerobic training to assist in the achieving or maintenance of an optimal body weight which may be necessary for a successful performance in some strength based sports. In addition to this, those advocating endurance training for strength athletes believe that aerobic training may enhance a strength trained athlete’s ability to recover. This study will investigate the effect of sub-maximal fatigue on strength trained athletes and investigate recovery from such exercise.

Confidentiality

The results and information received in this study are regarded as confidential and will be used by the investigating team only. You will also receive a copy of your own personal results and in addition your coach will also receive a copy. Your data will be kept anonymous through your personal ID code. Your data will be destroyed 7-10 years after publication of the study.

Freedom of Withdrawal

Participation is voluntary. Therefore you are free to withdraw from the study at any time without prejudice or reason. If you have any queries prior to contesting participation to this study please ask any of the investigating team.
Appendix A: Information Sheet and Consent forms: Study 1

You can contact the researchers at any time should you have any questions.
Contact Information:

Drew Harrison
PESS Department
University of Limerick
Limerick
Drew.Harrison@ul.ie
061202809

Niamh Ni Cheilleachair
PESS Department
University of Limerick
Limerick
niamh.nicheilleachair@ul.ie
061204781

If you have any concerns about this study and wish to contact someone independent, you may contact:
The Chairman of the University of Limerick Research Ethics Committee
C/o Vice President Academic and Registrar’s Office
University of Limerick
Limerick
Tel: (061) 202022
Appendix A: Information Sheet and Consent forms: Study 1

Written Informed Consent Form

An investigation into the effects of sub-maximal exercise on the biomechanical properties and performance of strength trained athletes.

- I have read and understood the volunteer information sheet.
- I understand what the project is about and what the results will be used for.
- I have completed the pre-test questionnaire.
- I am fully aware of all of the procedures involving myself and of any of the risks and benefits associated with the study.
- I know that my participation is voluntary and that I can withdraw from the project at any stage without giving any reason.
- I understand that the results of the research may be published but that my identity will not be revealed.
- I am aware that my results will be kept confidential.
- I understand that any questions I have concerning the research study before and after my consent will be answered by Niamh Ni Cheilleachair, PESS Department, University of Limerick; Tel:061-213172 or Dr. Drew Harrison, PESS Department, University of Limerick; Tel: 061-202809

Volunteer’s name ..................................................

Volunteer’s signature ..................................................

Date .................................................................

Experimenter’s signature ...........................................
Volunteer Information Sheet

**Supervisor:**
Dr. Drew Harrison

**Investigator:**
Niamh Ni Cheilleachair 9932275

**Purpose**
The aim of this study is to investigate the effects of sub-maximal exercise on the biomechanical properties and performance of endurance trained athletes.

**What is required of you?**
Prior to inclusion in this study you will be required to undergo screening by completing a pre-test questionnaire in order to ensure you meet the requirements necessary for participation in the study. You will also be asked to submit a copy of your training program for the two weeks prior to testing.

**Experimentation**
- You will be asked to follow a pre-lactate-test protocol (which is included on the following page).
- You will be required to attend the Biomechanics Laboratory in the PESS department on three occasions.
- One the first day you will become familiar with the jumping technique required and the apparatus being used.
- There will then be two days of testing with a rest of 24 hours between tests.
- You will be requested to refrain from any high intensity exercise in the 48 hours prior to the testing day.
- On the testing day you will undertake a warm up of 5 minutes jogging and each volunteer will also perform 3-4 practice rebound jumps.
- Following the warm up and practice rebound jumps blood lactate will be taken from your earlobe using a “lactate pro” to establish your pre-test blood lactate level.
- The pre-test will consist of 4 drop jump rebound jump sets in order to establish a baseline jump height. All jumps will be performed on the sledge apparatus.
- Each jump must be performed with maximum effort in order to achieve a maximal jump height.
You will then perform a “Bleep Test”, a 20m multistage fitness test.

1 minute after the termination of the fatigue workout you will perform 1 drop jump rebound jumpset. This will be repeated at 2 minutes, 5 minutes and 10 minutes after the termination.

At 2, 5 and 10 minutes after the fatigue workout your lactate will be measured from a blood sample taken from the ear lobe.

Following the final assessment at 10 minutes you will undertake a cool-down.

You will be requested to return to the biomechanics laboratory 24 hours after the initial test in order to evaluate your recovery. You will be required to perform 4 drop jump rebound jump sets.

**Potential Risks and Benefits**

**Risks**

4. All blood sampling carries a risk of infection to the volunteer and the experimenter. The technique will take place in a designated area of the teaching laboratory, which will be kept clear for this procedure, and cleaned appropriately.

5. There is a possibility of slight muscle soreness following the completion of an exercise session. This soreness is a natural response to training.

6. Following the test you will experience the discomfort of fatiguing for at least 5 minutes post-exercise. Some embarrassment may be experienced by you by the distress caused by fatiguing exercise.

**Benefits**

The stretch shortening cycle (SSC) involves the stretching of an active muscle followed immediately by a concentric contraction. The nature of endurance sports calls for repeated SSC muscle action over a prolonged period of time. The ability of endurance athletes to effectively utilise the SSC, and avail of its benefits is vital for performance. This study will investigate your ability to resist fatigue of the SSC and will also investigate the pattern of recovery of your SSC to its original capacity.

**Confidentiality**

The results and information received in this study are regarded as confidential and will be used by the investigating team only. You will also receive a copy of your own personal results and in addition your coach will also receive a copy. Your data will be kept anonymous through your personal ID code. Your data will be destroyed 7-10 years after publication of the study.

**Freedom of Withdrawal**

Participation is voluntary. Therefore you are free to withdraw from the study at any time without prejudice or reason. If you have any queries prior to contesting participation to this study please ask any of the investigating team.
Appendix B: Information Sheet and Consent forms: Study 1

You can contact the researchers at any time should you have any questions.

Contact Information:

Drew Harrison       Niamh Ni Cheilleachair
PESS Department     PESS Department
University of Limerick University of Limerick
Limerick            Limerick
Drew.Harrison@ul.ie niamh.nicheilleachair@ul.ie
061202809           061204781

If you have any concerns about this study and wish to contact someone independent, you may contact:

The Chairman of the University of Limerick Research Ethics Committee
C/o Vice President Academic and Registrar’s Office
University of Limerick
Limerick
Tel: (061) 202022
An investigation into the effects of sub-maximal exercise on the biomechanical properties and performance of endurance trained athletes.

- I have read and understood the volunteer information sheet.
- I understand what the project is about and what the results will be used for.
- I have completed the pre-test questionnaire.
- I am fully aware of all of the procedures involving myself and of any of the risks and benefits associated with the study.
- I know that my participation is voluntary and that I can withdraw from the project at any stage without giving any reason.
- I understand that the results of the research may be published but that my identity will not be revealed.
- I am aware that my results will be kept confidential.
- I understand that any questions I have concerning the research study before and after my consent will be answered by Niamh Ni Cheilleachair, PESS Department, University of Limerick; Tel: 061-213172 or Dr. Drew Harrison, PESS Department, University of Limerick; Tel: 061-202809

Volunteer’s name ..................................................

Volunteer’s signature ............................................

Date ...............................................................  

Experimenter’s signature ......................................

205
Appendix C: Pre-Test Questionnaire: Study 1, 2, 3 and 4

Department of Physical Education & Sport Sciences

PRE-TEST QUESTIONNAIRE

Name …………………………………. Ref. No. ……………………
Date of Birth …………………………… Age: ……………………
Test procedure …………………………

As you are to be a subject in this laboratory/project, would you please complete the following questionnaire. Your cooperation in this is greatly appreciated.

Please tick appropriate box

YES  NO

Any information contained herein will be treated as confidential

Has the test procedure been fully explained to you?

1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?

2. Do you feel pain in your chest when you do physical activity?

3. In the past month, have you had chest pain when you were not doing physical activity?

4. Do you lose your balance because of dizziness or do you ever lose consciousness?
Appendix B: Information Sheet and Consent forms: Study 1

5. Do you have a bone or joint problem that could be made worse by a change in your physical activity? □ □

6. Is your doctor currently prescribing drugs for your blood pressure or heart condition? □ □

7. Do you know of any other reasons why you should not undergo physical activity? This might include severe asthma, diabetes, a recent sports injury, or serious illness. □ □

8. Have you any blood disorders or infectious diseases that may prevent you from providing blood for experimental procedures? □ □

If you have answered NO honestly to all questions then you can be reasonably sure that you can take part in the physical activity requirement of the test procedure.

I ………………………………. declare that the above information is correct at the time of completing this questionnaire Date ……/……/…….

Please Note: If your health changes so that you can then answer YES to any of the above questions, tell the experimenter/laboratory supervisor. Consult with your doctor regarding the level of physical activity you can conduct.

If you have answered YES to one or more questions:
Talk with your doctor in person discussing with him/her those questions you answered yes. Ask your doctor if you are able to conduct the physical activity requirements.

Doctor’s signature ………………………………… Date ……/……/……
Signature of Experimenter…………………………………… Date ……/…
Volunteer Information Sheet

Supervisor: Dr. Drew Harrison

Investigator: Niamh Ni Cheilleachair 9932275

Purpose
The aim of this study is to investigate and compare the effects of high intensity interval training and continuous training on the performance of endurance trained athletes.

What is required of you?
Prior to inclusion in this study you will be required to undergo screening by completing a pre-test questionnaire in order to ensure you meet the requirements necessary for participation in the study. You will be required to participate for a period of eight weeks, one week to complete pre-testing to establish your baseline fitness levels, six weeks of training and one week to complete post tests identical to those you performed in the first week. You will also be asked to submit a copy of your training program for the eight weeks period of testing.

Experimentation
- You will be asked to follow a pre-lactate-test protocol (which is included on the following page)

- You will be required to attend the Physiology Laboratory in the PESS department on eight occasions.

- You will be requested to refrain from any high intensity exercise in the 48 hours prior to the testing days.

- Session 1: One the first day you will become familiar with all equipment that will be used and you will perform a 2000m ergometer test on the Concept II. This session will take approximately 1 hour.

- Session 2: On the second day you will perform a VO2 maximum test to establish your maximal oxygen uptake. This test will involve a series of incremental tests until you reach a maximal level.
Appendix D: Information Sheet and Consent forms Study 3 & 4

- During this session you will wear a mouth piece and you expired air will be analysed. You will be introduced to this piece of equipment during the familiarisation session and will be given the opportunity to experience it.

- Prior to this test you will be asked to follow a pre-lactate-test protocol (which is included on the following page).

- During each stage of the incremental test blood lactate will be taken from your earlobe using a “lactate pro” to establish your blood lactate levels.

- A lactate sample will also be taken 2 and 5 minutes after the test is completed.

- This session will last approximately 1 hour.

- **Session 3:** During the third session you will perform a 30 minute submaximal ergometer session at 60%-70% of your VO2max. During this session you will also wear a mouth piece and you expired air will be analysed. This session including a warm-up and cool-down will take approximately 1 hour

- **Session 4:** During this session your height, weight and skin-fold thickness will be measured to assess your body composition. You will also receive a DXA scan.

- Following the initial four sessions you will assigned to a high intensity interval training group or a continuous training group.

- If you are assigned to the high intensity training group you will perform two training sessions per week, over a six week period, involving ten 500m pieces at 100% of your VO2 max. Prior to and after each of these sessions you will perform a 20 minute warm-up and cool-down respectively. This session will replace a normal long distance steady state training session from your normal training program so will require no extra time on top of your regular training program.

- If you are assigned to the continuous training group you will perform your regular long distance steady state sessions, two of which will be monitored per week over a six week period.

- At the end of the eight weeks you will repeat session 1 – 4.

**Potential Risks and Benefits**

**Risks**

7. All blood sampling carries a risk of infection to the volunteer and the experimenter. The technique will take place in a designated area of the teaching laboratory, which will be kept clear for this procedure, and cleaned appropriately.
8. There is a possibility of slight muscle soreness following the completion of an exercise session. This soreness is a natural response to training. Following the test you will experience the discomfort of fatiguing for at least 5 minutes post-exercise. Some embarrassment may be experienced by you by the distress caused by fatiguing exercise. However, you will be well accustomed with all aspects of the procedures from your daily training program and would not be greater than that which you would experience on a regular basis.

**Benefits**

Following the pre-test and post-test you will receive detailed physiological feedback regarding your performance. This will include a lactate profile and VO2 profile which is valuable information for both you and your coach regarding your current training status and your strengths and weaknesses as an endurance athlete. You will also receive a detailed description of your body composition.

**Confidentiality**

The results and information received in this study are regarded as confidential and will be used by the investigating team only. You will also receive feedback and a copy of your own personal results and in addition your coach with your consent. Your data will be kept anonymous through your personal ID code. Your data will be destroyed 7-10 years after publication of the study.

**Freedom of Withdrawal**

Participation is voluntary. Therefore you are free to withdraw from the study at any time without prejudice or reason. If you have any queries prior to contesting participation to this study please ask any of the investigating team.

You can contact the researchers at any time should you have any questions.

Contact Information:

Drew Harrison  
PES Department  
University of Limerick  
Limerick  
Drew.Harrison@ul.ie  
061202809

Niamh Ni Cheilleachair  
PES Department  
University of Limerick  
Limerick  
niamh.nicheilleachair@ul.ie  
061204726

If you have any concerns about this study and wish to contact someone independent, you may contact:  
*The Chairman of the University of Limerick Research Ethics Committee*  
C/o Vice President Academic and Registrar’s Office  
University of Limerick  
Limerick  
Tel: (061) 20202
Written Informed Consent Form

An investigation into the effects of sub-maximal exercise on the biomechanical properties and performance of endurance trained athletes.

- I have read and understood the volunteer information sheet.
- I understand what the project is about and what the results will be used for.
- I have completed the pre-test questionnaire.
- I am fully aware of all of the procedures involving myself and of any of the risks and benefits associated with the study.
- I know that my participation is voluntary and that I can withdraw from the project at any stage without giving any reason.
- I understand that the results of the research may be published but that my identity will not be revealed.
- I am aware that my results will be kept confidential.
- I understand that any questions I have concerning the research study before and after my consent will be answered by Niamh Ni Cheilleachair, PESS Department, University of Limerick; Tel:061-204726 or Dr. Drew Harrison, PESS Department, University of Limerick; Tel: 061-202809

Volunteer’s name ................................................

Volunteer’s signature ...........................................

Date ..............................................................

Experimenter’s signature .....................................
Pre-Lactate-Test Protocol

Supervisor: Drew Harrison
Investigator: Niamh Ni Cheilleachair 9932275

Subjects participating in the study:

Prior to testing the following is requested of you:

- **Nutritional status:** It is important you maintain a normal diet prior to the test (i.e. try and keep your intake of carbohydrate, proteins and fats normal).

- **Training:** No heavy training should be completed the day before the test. It is preferable that arrive at the lab fully rested.

- **Alcohol:** No alcohol should be consumed within 24 hours of the test.

- **Caffeine:** Maintain normal levels of caffeine

- **Sleep:** A normal night’s sleep should also be attained on the night before the test.

- **Hydration:** Make sure you are properly hydrated when arriving for the test.
**Pre-participation Questionnaire for Physiological Assessment**

<table>
<thead>
<tr>
<th>TEST LOCATION:</th>
<th>PERSONAL</th>
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<tbody>
<tr>
<td>NAME</td>
<td>DATE / /</td>
</tr>
<tr>
<td>DATE OF BIRTH</td>
<td>SPORT EVENT</td>
</tr>
<tr>
<td>ADDRESS</td>
<td>CONTACT TEL NO</td>
</tr>
<tr>
<td>DOCTOR</td>
<td>EMAIL</td>
</tr>
<tr>
<td>IN CASE OF EMERGENCY PLEASE NOTIFY:</td>
<td></td>
</tr>
</tbody>
</table>

**FAMILY HISTORY**

Please identify any health problems that have occurred in your immediate family.

________________________________________________________

Has someone in your family died suddenly (before the age of 50 years)?

- NO    □  YES    □

Do you know why?

- □

Is there any family history of:

- □ High Blood Pressure
- □ Heart problems
- □ Cancer or tumour
- □ Migraine headache
- □ Emotional problems
- □ Diabetes
- □ Bowel disorder
- □ Problem with pregnancy
- □ Anaemia
- □ Epilepsy
- □ Arthritis
- □ Kidney/Bladder disorder
- □ Stomach disorder
- □ Allergies/Asthma
- □ Genetic disorder
- □ Other – please name:

**CARDIAC**

1. Have you had a sudden blackout where you have lost consciousness and fallen to the ground for no good reason particularly in association with exercise?

   - NO    □  YES    □

2. Have you been diagnosed with a heart condition?

   - NO    □  YES    □

3. Do you develop front of chest tightness with exercise that prevents you continuing?

   - NO    □  YES    □

4. Do you get sudden onset very rapid heart beating that occurs for no obvious reason and which makes you feel unwell?

   - NO    □  YES    □

**ILLNESS**

1. Are you currently suffering from any type of illness?

   - NO □

   - YES □

2. Have you had any type of illness of health problem for the last two weeks?

   - NO □

   - YES □
### Appendix D: Information Sheet and Consent forms Study 3 & 4

#### INJURY

1. **Do you currently have any injuries?**
   - NO ☐
   - YES ☐
   - If yes, provide details (type, severity):

2. **Have you had injuries for the past two weeks?**
   - NO ☐
   - YES ☐
   - If yes, provide details (type, severity):

#### MEDICATION/SUPPLEMENTS

1. **Are you currently taking any medication?**
   - NO ☐
   - YES ☐
   - If yes, please provide details (type, dosage):

2. **Have you taken any medication over the last two weeks?**
   - NO ☐
   - YES ☐
   - If yes, please provide details (type, dosage):

Please list any supplements you are currently taking

<table>
<thead>
<tr>
<th>Supplement</th>
<th>Daily Dose</th>
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</table>

#### MOTIVATION

1. **Evaluate your motivation for training today**
   - POOR ☐
   - OK ☐
   - GOOD ☐
   - EXCELLENT ☐

2. **Evaluate your motivation for testing today**
   - POOR ☐
   - OK ☐
   - GOOD ☐
   - EXCELLENT ☐

#### TRAINING

1. **Evaluate your last week of physical training.**
   - EASY ☐
   - MODERATE ☐
   - HARD ☐
   - VERY HARD ☐

2. **How fatigued are you today? (0 = not at all; 5 = extremely)**
   - 0 ☐
   - 1 ☐
   - 2 ☐
   - 3 ☐
   - 4 ☐
   - 5 ☐

3. **How many hours ago did you last exercise?**

4. **Describe your last 3 training sessions.**

<table>
<thead>
<tr>
<th>Time Today</th>
<th>Training Session</th>
<th>Difficulty (easy, moderate, hard)</th>
</tr>
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<tbody>
<tr>
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Appendix D: Information Sheet and Consent forms Study 3 & 4

<table>
<thead>
<tr>
<th>Yesterday</th>
<th></th>
<th>Two days ago</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td></td>
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</tbody>
</table>

TRAVEL
Have you had to travel over the last seven days? Yes ☐ No ☐

If yes, provide details (eg., Plane, car; duration of trip)

MISCELLANEOUS
Please provide any additional information that you believe may influence your fitness test results

FOR WOMEN ONLY
1. Are you pregnant? NO ☐ YES ☐
2. Please indicate your current menstrual status:
   3. ☐ No menstruation ☐ Irregular  ☐ Regular menstruation
4. How many days since your last menstruation?____________________
5. Do you currently take an oral contraceptive? NO ☐ YES ☐

Adapted from ©Australian Sports Commission
### Appendix E: Training Diary Study 4

<table>
<thead>
<tr>
<th>Week no.</th>
<th>What week in the study are you</th>
</tr>
</thead>
<tbody>
<tr>
<td>Date</td>
<td>Todays date</td>
</tr>
<tr>
<td>Day</td>
<td>Day of week</td>
</tr>
<tr>
<td>Morning HR</td>
<td>HR as soon as you wake before getting out of bed</td>
</tr>
<tr>
<td>Weight</td>
<td>Morning weight</td>
</tr>
</tbody>
</table>

<table>
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<tr>
<th>Sleep</th>
<th>Fatigue</th>
<th>Stress</th>
<th>Muscle Soreness</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Very very good</td>
<td>Very very low</td>
<td>Very very low</td>
</tr>
<tr>
<td>2</td>
<td>Very good</td>
<td>Very low</td>
<td>Very low</td>
</tr>
<tr>
<td>3</td>
<td>Good</td>
<td>Low</td>
<td>Low</td>
</tr>
<tr>
<td>4</td>
<td>Average</td>
<td>Average</td>
<td>Average</td>
</tr>
<tr>
<td>5</td>
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<td>High</td>
<td>High</td>
</tr>
<tr>
<td>6</td>
<td>Very bad</td>
<td>Very high</td>
<td>Very high</td>
</tr>
<tr>
<td>7</td>
<td>Very very bad</td>
<td>Very very high</td>
<td>Very very high</td>
</tr>
</tbody>
</table>

### Training Details

#### Session no of wk

#### Explanation of session
Prior to the session explain the goals of the session.

#### Warm up
Give exact details of your warm-up. Type of warm up/Duration/Intensity/Distance covered/Average HR

#### Actual session
Include a *detailed description* of the session.

- **Session**: Water/erg/weights/cross-training
- **Duration**
- **Distance**
- **Average split**
- **Heart rate**: average HR for session

#### Cool down
Give exact details of your cool-down. Type of cool down/Duration/Intensity/Distance covered/Average HR

### Additional Information

#### Illness

#### Injury

#### Menstrual cycle

#### Causes of stress

*Comments*: Please supply any additional information regarding your day that is important. For example, how the training session went, how you felt during the training session, how you felt after the training session. If you're stressed/tired/fatigue can you suggest why. The more information you can give us the better.
## Appendix E: Training Diary Study 4

<table>
<thead>
<tr>
<th>HITT Session no.</th>
<th>Date</th>
<th>Day</th>
<th>Duration of interval</th>
<th>Warm up</th>
<th>Duration</th>
<th>Distance</th>
<th>HR range</th>
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<table>
<thead>
<tr>
<th>Session</th>
<th>Target Split</th>
<th>Target Watt</th>
<th>Actual Split</th>
<th>Actual Watt</th>
<th>Distance</th>
<th>HR</th>
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<tbody>
<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Interval 2</td>
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<td>Interval 8</td>
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<table>
<thead>
<tr>
<th>Cool down</th>
<th>Duration</th>
<th>Distance</th>
<th>HR range</th>
<th>Description</th>
</tr>
</thead>
</table>

*Comments:*
### Determination of Test Protocol

Table 2: Ergometer work loads (W and mm:ss.s) categorised with respect to the athlete’s best 2000m time in the previous year.

<table>
<thead>
<tr>
<th>Work Loads Increments (W)</th>
<th>Previous Years Selection Ergo Time</th>
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<tr>
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<td>5:50 0</td>
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<tr>
<td>Work Load 1 (W)</td>
<td>200</td>
</tr>
<tr>
<td>Work Load 2 (W)</td>
<td>245</td>
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<tr>
<td>Work Load 3 (W)</td>
<td>290</td>
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<td>Work Load 4 (W)</td>
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<td>Work Load 5 (W)</td>
<td>380</td>
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<td>Work Load 6 (W)</td>
<td>425</td>
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<td>Work Load 7 (W)</td>
<td>MAX</td>
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<td>Work Load 1 (mm:ss.s)</td>
<td>02:00.7</td>
</tr>
<tr>
<td>Work Load 2 (mm:ss.s)</td>
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<tr>
<td>Work Load 3 (mm:ss.s)</td>
<td>01:46.6</td>
</tr>
<tr>
<td>Work Load 4 (mm:ss.s)</td>
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<td>Work Load 5 (mm:ss.s)</td>
<td>01:37.4</td>
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<tr>
<td>Work Load 6 (mm:ss.s)</td>
<td>01:33.8</td>
</tr>
<tr>
<td>Work Load 7 (mm:ss.s)</td>
<td>MAX</td>
</tr>
</tbody>
</table>
Abstract submitted and presented at the American College of Sports Medicine, Baltimore 2010. Abstract awarded Biomechanics Interest Group Travel Award.

Abstract Title: The Effect of Maximal Fatigue on Skeletal Muscle in Strength & Endurance Athletes.
Ni Cheilleachair N. and Harrison A.J.
University of Limerick, Limerick, Ireland.

Background
Fatigue is a complex, multidimensional phenomenon and is a reflection of the type of work that has been done. Differences in fatigue between strength and endurance athletes have been documented (Skurvydas et al, 2002) but are usually attributed to muscle metabolism. A number of studies, however, have reported that decreases in muscle performance may be the result of the impaired utilisation of muscle stiffness-mediated elastic energy (Avela & Komi, 1998) which is associated with the stretch-shortening cycle (SSC) phenomenon. In coexistence with fatigue is the concept of potentiation. Potentiation initially exhibits a depression, most likely the result of acute fatigue, followed by a rapid rise in muscle function which can often result in an improvement in performance. Purpose: The aim of this study was to investigate and compare the affect of maximum SSC fatigue on endurance and strength trained athletes and to also investigate the path of recovery with any subsequent potentiation.

Methods: The study was approved by the UL Research Ethics Committee, and volunteers completed informed consent documents prior to participation. Ten strength trained athletes (STA) and nine endurance trained athletes (EDA) performed sets of drop (DJ) and rebound jumps (RBJ) prior to a maximal fatigue workout and 15, 45, 120, 300 and 600s after. All jumps were performed on a sledge and force plate apparatus and dependable variables of peak GRF, contact time, height jumped and leg spring stiffness were calculated.

Results: The statistical results (GLM ANOVA) revealed a significant reduction in peak GRF (p<0.01) and height jumped (p<0.01) in both groups of athletes following the fatigue workout. The STA also showed a significant reduction in $k_{vert}$ (p<0.01), and while the ETA had a reduction of 6.38% in $k_{vert}$ it was not a significant change. The difference between the baseline jumps and the maximum scores achieved during the 600 second recovery showed a significant increase in peak GRF (p = 0.001) and $k_{vert}$ (p = 0.049) for the ETA. The STA also showed increases in these dependent variables, a change in GRF of 7.34% and a change in $k_{vert}$ of 11.62%, but neither was statistically significant. Conclusion: The results of the study indicate that ETA and STA fatigue significantly in GRF generation and height jumped following a maximal fatigue workout. STA also show a significant reduction in $k_{vert}$, while ETA, despite showing a significant reduction in all other areas, appeared to resist a significant reduction in this dependent variable. The results also indicate that during the recovery after a maximal fatigue workout, ETA have an ability to potentiate. This
was evident from a significant increase in peak GRF and $k_{cen}$ above baseline values. These changes indicated that there is an enhancement in RBJ performance shortly after maximal fatigue as the RBJ is performed with a stiffer and more elastic leg spring action.

Reference


The effect of maximal fatigue on the biomechanical properties of skeletal muscle in strength & endurance athletes.

Niamh Ni Cheilleachair and A.J. Harrison

Biomechanics Research Unit, University of Limerick, Limerick, Ireland.

The purpose of this study was to compare the effects of maximal fatigue on the mechanical performance of strength and endurance athletes. Ten strength trained athletes and nine endurance athletes performed a maximum fatigue protocol on a sledge and force plate apparatus followed by drop and rebound jumps at 15, 45, 120, 300 and 600 seconds post fatigue. Measurements of peak force, ground contact time, leg spring stiffness and height jumped were calculated prior to the fatigue protocol to establish baseline values, and also for each jump following the fatigue protocol. The fatigue protocol resulted in a significant reduction in peak force (p<0.01) and height jumped (p<0.01) in both groups while leg spring stiffness was also reduced in the strength athletes (p<0.01). In addition the endurance athletes indicated a potentiation effect with a significant increase in peak force (p<0.01) and leg spring stiffness (p<0.05) during the post fatigue jumps.

KEY WORDS: fatigue, potentiation, SSC, strength athletes, endurance athletes

INTRODUCTION: Fatigue is a complex, multidimensional phenomenon and is a reflection of the type of work that has been done. It has been described as “the transient decrease in performance capacity of muscles when they have been active for a certain time, usually evidenced by a failure to maintain or develop a certain force or power” (Asmussen, 1979). The nature and cause of fatigue depends on the type of exercise being performed. Differences in fatigue between strength and endurance athletes have been documented (Skurvydas et al, 2002; Edwards et al, 1977; Sahlin et al 1998) but are usually attributed to muscle metabolism. A number of studies, however, have reported that muscle fatigue is not always associated with metabolic changes (Edwards et al, 1977; Sahlin et al 1998). Decreases in muscle performance following exercise may be somewhat the result of the impaired utilisation of muscle stiffness-mediated elastic energy (Avela & Komi, 1998). Komi et al (1986) reported similar findings when they concluded that repetitive impact loads may decrease the ability of the leg extensor muscles to maintain the necessary load and subsequently the muscle may lose its recoil ability. When active muscle is stretched, or when passively stretched muscle is suddenly activated, the muscle increases its tension and stores potential elastic energy in its series elastic component, which can then reappear during a subsequent shortening of the muscle.
This phenomenon involving eccentric and concentric contractions is known as the stretch shortening cycle (SSC).

In coexistence with fatigue is the concept of potentiation. Post-activation potentiation (PAP) is an acute transient improvement in performance as a result of prior muscle activation. PAP initially exhibits a depression, most likely the result of acute fatigue, followed by a rapid rise in muscle function. Few studies have examined the effect of maximal SSC fatigue on the performance of subsequent SSC activities. Consequently the aim of this study was to investigate and compare the effect of maximum SSC fatigue on endurance and strength trained athletes and to also investigate the path of recovery with any subsequent PAP.

**METHOD:** Ten strength trained athletes (STA) (rugby players) and nine endurance trained athletes (ETA) (rowers) participated in this study. All 10 STA and 6 of the ETA were high level national athletes while 3 ETA were international athletes.

**Table 1: Physical Characteristics of the Subjects.**

<table>
<thead>
<tr>
<th></th>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>Mass (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ETA</td>
<td>25.9±6.3</td>
<td>187.7±9.1</td>
<td>82.2±11.4</td>
</tr>
<tr>
<td>STA</td>
<td>20.0±0.8</td>
<td>180.9±4.9</td>
<td>89.8±12.8</td>
</tr>
</tbody>
</table>

The nature of the study was explained to each participant and written informed consent was obtained. The protocol was approved by the University of Limerick research ethics committee. All participants attended the laboratory for one session to complete the testing. On arrival at the laboratory the drop jump (DJ) and rebound jump (RBJ) technique was explained and demonstrated to the participants and following a general warm up of jogging and stretching the participants performed practice jumps. The jumps were performed on a sledge and force plate apparatus with the sledge inclined at 30º and the AMTI OR5-6 force platform mounted at right angles to the sledge apparatus. For all jumps the participants were secured to the chair with a harness and waist belt. Instruction was given to the participants to keep both arms folded across the shoulders in order to minimise upper body movement during the jumps. The participants were also instructed to perform each jump maximally while attempting to minimise ground contact time (CT) and maximise jump height (JH). Prior to the fatigue workout sets of jumps comprised of one DJ followed immediately by a RBJ were performed to establish the participant’s baseline values for each of the dependent variables: peak ground reaction force (pGRF), CT, JH and leg spring stiffness ($k_{\text{vert}}$). Testing commenced with four sets of DJ and RBJ with the participants being dropped from a predetermined height of 30 cm. Each set of jumps was immediately analysed using AMTI Bioanalysis software. Through use of the AMTI force plate instants of initial foot contact, take off and subsequent landing were obtained. From these ground reaction force traces, each of
Appendix G: Conference Publications

the dependent variables were calculated. Peak GRF was identified as the maximum force reading recorded from the ground reaction force traces from the force plate which was sampling at 1000Hz. CT was calculated as the difference between the time of initial foot contact and the time of take off. JH was calculated from the flight time (time difference between the take off and landing for jumps) and the use of the equation for linear motion \( s=ut+0.5at^2 \). The calculation of \( k_{vert} \) involved the use of SVHS video recordings which were digitised using Peak Motus (Peak Performance Technologies, Colorado, USA) to identify the displacement from landing to full crouch. The pGRF was then divided by this displacement to calculate \( k_{vert} \) which is defined as the ratio of GRF to the displacement of a spring.

Following the analysis of each set of baseline jumps, the jump with the highest recorded JH was selected for further analysis. 90% of this maximum jump was calculated and this value was marked on sledge rails from a position where the participant was seated in the chair with the dominant leg fully extended. The fatigue workout then began with the participant being dropped from a height of 30cm for 1 DJ followed by repeated RBJ until the 90% mark was not reached on three consecutive jumps. 15, 45, 120, 300 and 600 seconds following the termination of the fatigue workout the participants were dropped from 30cm to perform one set of DJ and RBJ. From these recovery intervals each subject’s minimum and maximum score for each dependent variable, irrespective of time, was identified. This allowed for identification of fatigue and any possible PAP without the interference of individual variation across recovery times.

**Statistical Analysis:** The software package SPSS (Version 16) was used to conduct all statistical analysis. A mixed effect split plot analysis of variance (SPANOVA) with repeated measures was used to evaluate differences between the average of the baseline scores and the minimum and maximum scores achieved during each recovery interval. The SPANOVA had 1 within-subjects factor namely Condition with 3 levels (baseline, minimum and maximum) and one between-subjects factor namely Group with 2 levels (ETA and STA).

**RESULTS:** Table 2 shows the mean ±SD of the number of jumps performed during the fatigue workout and the duration of the workout.

<table>
<thead>
<tr>
<th></th>
<th>No of jumps</th>
<th>Duration of workout (s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ETA</td>
<td>65.3±29.3</td>
<td>84.6±32.8</td>
</tr>
<tr>
<td>STA</td>
<td>55±24.9</td>
<td>69.3±21.2</td>
</tr>
</tbody>
</table>

The mean dependent variable scores for the baseline jumps were subtracted from the maximum and minimum scores for the jumps done after the fatigue workout and the results can be seen in figure 1. In this figure the baseline value is represented by the x-axis. The statistical results (GLM
ANOVA) revealed a significant reduction in pGRF (p<0.01) and JH (p<0.01) in both groups of athletes following the fatigue workout and is illustrated by the “error” bars in the figure below. The STA also showed a significant reduction in k_{vert} (p<0.01), and while the ETA had a reduction of 6.38% in k_{vert} it was not a significant change. The difference between the baseline jumps and the maximum scores achieved during the 600 second recovery showed a significant increase in pGRF (p = 0.001), an increase of 8.44% and k_{vert} (p = 0.049), an increase of 23.75% for the ETA. The STA also showed increases in these dependent variables, a change in GRF of 7.34% and a change in k_{vert} of 11.62%, but neither was statistically significant.

**DISCUSSION:** The maximal fatigue workout resulted in significant reductions in pGRF and JH for both ETA and STA. STA also showed a significant reduction in k_{vert}. These findings are similar.
Appendix G: Conference Publications

to studies which utilised submaximal SSC fatigue workouts (Avela & Komi, 1998; Gollhofer et al. 1987 a,b; Nicol et al. 1991) and to a study which utilised maximal SSC fatigue workouts (Comyns, 2006). Comyns (2006) found a loss of efficacy in the SSC in a group of strength trained rugby players through a significant reduction in flight time and pGRF and an increase in ground contact time. Comyns (2006) also identified a reduction in k<sub>vert</sub>, however it was not statistically significant.

Potentiation is known to co-exist with fatigue, whereby after an initial decline in performance following fatigue, there is a rapid rise in performance. While both ETA and STA in this study demonstrated an increase in performance following their initial decline, only the ETA demonstrated a significant improvement. The ETA showed a significant improvement in both pGRF (8.44% improvement) and k<sub>vert</sub> (23.75% improvement). While ETA have been reported to withstand fatigue better and recovery quicker than strength athletes, (Hakkinen & Myllyla, 1990), there is a dearth of information surrounding the potential for ETA to benefit from the phenomenon of potentiation and several studies have suggested that ETA are indeed not capable of receiving potential benefits of potentiation. Comyns et al. (2005) reported a similar potentiation effect, to that found in this study, on the biomechanics of performance of a fast SSC exercise due to a prior contractile activity, except with strength trained athletes. The results of the present study, however, indicate that ETA have the ability to alter the biomechanics of rebound jumps and perform the jump with a stiffer leg action.

**CONCLUSION:** The results of the study indicate that ETA and STA fatigue significantly in pGRF generation and JH following a maximal fatigue workout. STA also show a significant reduction in k<sub>vert</sub>, while ETA, despite showing a significant reduction in all other areas, appear to resist a significant reduction in k<sub>vert</sub>. The results also indicate that during the recovery after a maximal fatigue workout, ETA have an ability to potentiate. This was evident from a significant increase in pGRF and k<sub>vert</sub> above baseline values. These changes indicate that there is an enhancement in RBJ performance shortly after maximal fatigue as the RBJ is performed with a stiffer and more elastic leg spring action.

**REFERENCES:**
Appendix G: Conference Publications


Acknowledgement

The researchers would like to thank IRCSET for providing funding for this research.