Fluid manipulation strategies and running economy during prolonged high-intensity exercise

This item was submitted to Loughborough University's Institutional Repository by the/an author.

Additional Information:

- A Doctoral Thesis. Submitted in partial fulfilment of the requirements for the award of Doctor of Philosophy of Loughborough University.

Metadata Record: https://dspace.lboro.ac.uk/2134/25587

Publisher: © J.S. Sproule

Rights: This work is made available according to the conditions of the Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International (CC BY-NC-ND 4.0) licence. Full details of this licence are available at: https://creativecommons.org/licenses/by-nc-nd/4.0/

Please cite the published version.
<table>
<thead>
<tr>
<th>Author/Filing Title</th>
<th>Sprague, J.S.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accession/Copy No.</td>
<td>040129878</td>
</tr>
<tr>
<td>Vol. No.</td>
<td></td>
</tr>
<tr>
<td>Class Mark</td>
<td></td>
</tr>
</tbody>
</table>

LOAN COPY
Fluid Manipulation Strategies and Running Economy during Prolonged, High-Intensity Exercise

John Service Sproule

A Doctoral Thesis

Submitted in partial fulfilment of the requirements for the award of Doctor of Philosophy of Loughborough University

September 1996

© J S Sproule 1996
Summary

Running economy (RE) is defined as the rate of oxygen consumption at a given submaximal running velocity. Whilst the concept of running economy is well documented, little information is available about the daily variation in RE, variation in RE within prolonged running bouts and the effects on RE of exercise-induced dehydration. Thus, the principal aim of this research was to investigate these aspects and attempt to contribute further knowledge and understanding of RE.

The purpose of the first study (Chapter 4), was to investigate the daily variation in RE in twenty one habitually active Singaporean men ($VO_{2max} = 51.6 \pm 5.8$ ml.kg\(^{-1}\) min\(^{-1}\)). The RE was measured over three consecutive days during treadmill running at 3.33 m.s\(^{-1}\), and at running speeds representing relative exercise intensities of 60% and 85% $VO_{2max}$ (randomly assigned in a counterbalanced design). The running bouts were of 10 min duration at each speed, with 5 min rest between each running bout. The group mean ($\pm$SD) $VO_2$ during submaximal running at 3.33 m.s\(^{-1}\) (82 $\pm$5.7% $VO_{2max}$) was 44.5 ($\pm$2.1), 43.8 ($\pm$2) and 44.2 ($\pm$2) ml.kg\(^{-1}\) min\(^{-1}\) for days one, two and three respectively. The mean coefficient of variation (CV) for RE at 3.33 m.s\(^{-1}\) was 2.54 % (range = 0.24 - 5.41%). The group mean $VO_2$ during submaximal running at 60% and 85% $VO_{2max}$ was similarly invariant over the three consecutive days. The results showed no differences in daily RE for the group ($p > 0.05$). The results suggest that for the individuals in this study RE does not change on a daily basis.

An increase in $VO_2$ during prolonged constant pace running has been linked to increasing heart rate (HR), decreasing lung function and increasing ventilation ($V_{E\cdot}$). The second study
(Chapter 5), investigated the effects of prolonged moderate intensity running bouts on RE. Five Singaporean men (VO$_{2\text{max}}$ = 52.8 ±4.8 ml.kg$^{-1}$ min$^{-1}$) performed three prolonged runs (60 min) at 65% VO$_{2\text{max}}$ (test 1), 70% VO$_{2\text{max}}$ (test 2) and 75% VO$_{2\text{max}}$ (test3), with each test 7 days apart. Within each 60 min running bout the subjects ran at the common speed of 3 m.s$^{-1}$ (75% VO$_{2\text{max}}$) for the following periods 6 - 10, 31 - 35 and 56 - 60 min. The RE at 3 m.s$^{-1}$ was measured during minutes 9 - 10 (A), 34 - 35 (B) and 59 - 60 minutes (C). A lung function test (FVC & FEV$_{1}$) was performed and administered before and after each 60 min running bout. At A, B and C the RE did not change during the three tests. HR drifted upwards and approached significance (T(4) = -2.59; p = 0.06) during the 75% VO$_{2\text{max}}$ run, FVC decreased significantly for main effect time (F(1,3) = 26.62; p = 0.014), as did FEV$_{1}$ (F(1,3) = 36.28; p = 0.009), and V$_{E}$ increased during all of the prolonged runs with a significant difference for main effect time (F(1,4) = 16.75; p = 0.015). These findings suggest that increasing HR, decreasing lung function and increasing V$_{E}$ do not manifest in deteriorating RE during 60 minute running bouts at intensities up to 75% VO$_{2\text{max}}$.

As a result of the findings of the previous study a third study (Chapter 6), was designed to investigate the effects of higher intensity prolonged running on intraindividual variation in RE. Fifteen Singaporean men (VO$_{2\text{max}}$ = 56.6 ±4.7 ml.kg$^{-1}$ min$^{-1}$) performed three prolonged runs each in a counter-balanced design and the runs varied in time (40 or 60 min) and intensity (70% or 80% VO$_{2\text{max}}$). Each prolonged run was separated by seven days. The RE at 3 m.s$^{-1}$ was measured pre (pre RE) and post (post RE) each prolonged run and a control study involved monitoring RE at 10.8 3 m.s$^{-1}$ pre and post 60 min rest. There were no differences between pre RE and post RE values during the control run. However, there were differences between pre RE and post RE values when separated by a prolonged run. The mean absolute
changes in VO₂ (ml.kg⁻¹ min⁻¹) values were 38.4 ±2.5 vs 40.1 ±2.6 (80% VO₂max/40 min), 38.9 ±2.8 vs 41.5 ±2.6 (70% VO₂max/60 min) and 39.0 ±3.1 vs 42.7 ±2.9 (80% VO₂max/60 min; p < 0.01). The results of this investigation support that RE deteriorates during prolonged running, and the magnitude of the deterioration in RE increases with both increasing exercise intensity and with increasing time.

The third study found that RE deteriorated (p < 0.01) during a single bout of prolonged, high-intensity running (60 min at 80% VO₂max). Because of this finding and the possible effects of exercise-induced dehydration on RE, a fourth study (Chapter 7) investigated the effects on RE of ingesting either no fluid or an electrolyte solution with or without 6% carbohydrate (counterbalanced design) during 60 minute running bouts at 80% VO₂max, in either a thermoneutral (22-23°C; 56-62% RH) or a hot and humid environment (25-35°C; 66-77% RH). The subjects were 15 Singaporean men who had been subjects in the previous three studies (VO₂max = 55.5 ±4.4 ml.kg⁻¹ min⁻¹). The RE was measured from 9-10 min (pre 80%) and from 59-60 min (post 80%) during the prolonged running bouts, and at 3 m.s⁻¹ pre (pre RE) and post (post RE) each prolonged run. Fluids were administered every 2 min at an individual rate determined from pre tests to replace body weight (group mean = 17 4 ml.min⁻¹). The VO₂ for the post RE was higher (p < 0.05) than during pre RE test for all treatments and there were no differences between treatments (ANOVA). The mean increase in VO₂ from pre RE to post RE ranged from 3.4 to 4.8 ml.kg⁻¹ min⁻¹ across treatments. The deterioration in RE during prolonged running at 80% VO₂max (pre 80% vs post 80%) was less (mean increase range = 1.5 to 2.6 ml.kg⁻¹ min⁻¹) than the decrease observed during running at the common speed of 3 m.s⁻¹ (65 ±6% VO₂max). The deterioration in RE observed between pre RE and post RE was accompanied by changes (p < 0.05) in the following: increased V̇E; decreased R-
values; elevated HR; higher plasma lactate/potassium/ammonia concentrations and higher $T_{RE}$. In conclusion, the deterioration in RE at 3 m.s$^{-1}$ (65% $VO_{2\text{max}}$) after 60 min running at 80% $VO_{2\text{max}}$ appears to occur independent of whether fluid is ingested and regardless of whether the fluid contains carbohydrates or electrolytes, in both thermoneutral and in hot, humid environments. In summary the results of these studies demonstrate that RE is stable on a daily basis during submaximal, short duration (10 min) exercise at intensities up to 85% $VO_{2\text{max}}$. Also, RE remains stable throughout prolonged (60 min) exercise at 65% - 75% $VO_{2\text{max}}$, but RE did worsen significantly during a 60 minute running bout at 80% $VO_{2\text{max}}$. Finally, I addressed whether exercise dehydration or substrate reduction might be primary links in the observed deterioration in RE during 60 minute running bouts at 80% $VO_{2\text{max}}$, but the results were inconclusive.

*±SD
Acknowledgements

This work was completed as an external student of the Department of Physical Education, Sports Science and Recreation Management at Loughborough University, and was supported by research funding from Nanyang Technological University (Singapore) and sponsorship from NIKE (Singapore) and Quaker Oats (Singapore).

I am indebted to Professor Clyde Williams, my supervisor. He challenged me to ‘climb the mountain’ and was an inspirational expedition leader.

I also wish to thank Mike MacNeill for his deep friendship and support as I struggled to meet the demands of a full-time occupation with part-time study, Dr. Peter Horton for his ‘artistry’ and stimulating debate, Dr. David Stensel for his constructive criticism, Mr. Kunalan for ‘being there’, and Angus Fraser (Haematology Department, National University Hospital, Singapore) for sharing some of his knowledge of blood sampling procedures.

I am grateful to my former students from the School of Physical Education (Singapore). Thank you for your friendship and youthful enthusiasm. You gave freely of your time and your commitment as subjects in demanding experimental work was excellent.

Thank-you all.
To my wife Bee Leng, my son Sean and daughter Laura-Beth
## List of Contents

<table>
<thead>
<tr>
<th>Chapter 1</th>
<th>Introduction</th>
<th>1</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.1</td>
<td>Running efficiency</td>
<td>2</td>
</tr>
<tr>
<td>1.2</td>
<td>Running economy</td>
<td>3</td>
</tr>
<tr>
<td>1.3</td>
<td>Thesis overview</td>
<td>5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Chapter 2</th>
<th>Review of Literature</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.1</td>
<td>Overview</td>
<td>8</td>
</tr>
<tr>
<td>2.2</td>
<td>Physiological and environmental factors and running economy</td>
<td>9</td>
</tr>
<tr>
<td>2.2.1</td>
<td>Age</td>
<td>9</td>
</tr>
<tr>
<td>2.2.2</td>
<td>Gender</td>
<td>12</td>
</tr>
<tr>
<td>2.2.3</td>
<td>Vertical displacement</td>
<td>14</td>
</tr>
<tr>
<td>2.2.4</td>
<td>Body composition</td>
<td>15</td>
</tr>
<tr>
<td>2.2.5</td>
<td>Body temperature</td>
<td>17</td>
</tr>
<tr>
<td>2.2.6</td>
<td>Heart rate and ventilation</td>
<td>18</td>
</tr>
<tr>
<td>2.2.7</td>
<td>Air and wind resistance</td>
<td>20</td>
</tr>
<tr>
<td>2.2.8</td>
<td>Drafting</td>
<td>22</td>
</tr>
<tr>
<td>2.2.9</td>
<td>Altitude</td>
<td>21</td>
</tr>
<tr>
<td>2.2.10</td>
<td>Surface</td>
<td>22</td>
</tr>
<tr>
<td>2.2.11</td>
<td>Fatigue</td>
<td>22</td>
</tr>
<tr>
<td>2.2.12</td>
<td>Training</td>
<td>24</td>
</tr>
<tr>
<td>2.2.13</td>
<td>Running velocity and VO₂</td>
<td>27</td>
</tr>
<tr>
<td>2.2.14</td>
<td>Effectiveness</td>
<td>29</td>
</tr>
<tr>
<td>2.2.15</td>
<td>Lactate</td>
<td>29</td>
</tr>
<tr>
<td>2.2.16</td>
<td>Muscle fibre type</td>
<td>31</td>
</tr>
<tr>
<td>2.2.17</td>
<td>Genotype</td>
<td>32</td>
</tr>
<tr>
<td>2.2.18</td>
<td>Efficiency and fibre type</td>
<td>33</td>
</tr>
<tr>
<td>2.2.19</td>
<td>Excess post-exercise oxygen consumption (EPOC)</td>
<td>37</td>
</tr>
</tbody>
</table>
2.2.20 Fibre type, EPOC and overtraining 38
2.3 Biomechanical factors affecting running economy 41
2.3.1 Body mass 41
2.3.2 Body mass distribution 42
2.3.3 Running kinematics and economy 46
2.3.4 Kinetic factors 54
2.4 Psychological state and running economy 57
2.5 Conclusion 65

Chapter 3 General Methods 66

3.1 Apparatus and instrumentation 66
3.2 Subjects and laboratory procedures 67
3.2.1 Height and mass 67
3.2.2 Heart rate 67
3.2.3 Expired air collection and analysis 68
3.2.4 Measurement of body temperature 68
3.2.5 Subjective ratings of exertion 69
3.2.6 Environmental considerations 69
3.3 Preliminary testing 70
3.3.1 Familiarisation 70
3.3.2 Establishing a speed-VO₂ relationship 70
3.3.3 Determination of maximal oxygen uptake 71
3.3.4 Running economy 71
3.4 Standardised test procedure 72
3.5 The collection and analysis of blood samples 72
3.6 Statistical analysis 73

Chapter 4 Daily Stability in Running Economy 74

4.1 Introduction 74
4.2 Methods 76
Chapter 5  Running Economy, Ventilation and Heart Rate during Prolonged Running Bouts at 65%, 70% and 75% VO$_{2\text{max}}$

5.1  Introduction  95  
5.2  Methods  95  
5.3  Results  99  
5.4  Discussion  105  

Chapter 6  The Influence of Exercise Duration and Intensity on Running Economy

6.1  Introduction  106  
6.2  Methods  107  
6.3  Results  113  
6.4  Discussion  121  

Chapter 7  The Influence of either No Fluid or Carbohydrate-Electrolyte Fluid Ingestion and Environment on Running Economy post Prolonged, High-Intensity Running

7.1  Introduction  130  
7.2  Methods  134  
7.3  Results  140  
7.4  Discussion  160  

Chapter 8  General Discussion  183  

References  188
List of Figures

4.1 Experimental design of the level treadmill running study 78
4.2 Intraindividual VO₂ during steady state running at 3.33 m.s⁻¹ 89

5.1 Experimental design of the level treadmill running study 97
5.2 Heart rate when running at 10.8 km.h⁻¹ (75% VO₂max) during min 6-10, 31-35 and 56-60 of 60 min running bouts at 65%, 70% and 75% VO₂max (mean ±SD) 102

6.1 Experimental design of the level treadmill study 110

7.1 Schematic representation of the experimental procedures 136
7.2 Ventilation during running at 10.8 km.h⁻¹ pre (RE1) and post (RE2) prolonged running bouts (60 min 80% VO₂max), whilst ingesting no fluid, glucose-electrolyte or placebo-electrolyte solutions (mean ±SD) 144
7.3 Ventilation during 9-10 min (80%RE1) and 59-60 min (80%RE2) of prolonged running bouts (60 min 80% VO₂max), whilst ingesting no fluid, glucose-electrolyte or placebo-electrolyte solutions (mean ±SD) 145
7.4 Plasma lactic acid concentration during running at 10.8 km.h⁻¹ pre (RE1) and post (RE2) 60 min running bouts at 80% VO₂max, whilst ingesting no fluid, glucose-electrolyte or placebo-electrolyte solutions (mean ±SD) 153
7.5 Plasma potassium concentration during running at 10.8 km.h⁻¹ pre (RE1) and post (RE2) prolonged running bouts (60 min 80% VO₂max), whilst ingesting no fluid, glucose-electrolyte or placebo-electrolyte solutions (mean ±SD) 157
Plasma ammonia concentration during running at 10.8 km.h\(^{-1}\) pre (RE1) and post (RE2) prolonged running bouts (60 min 80% VO\(_{2\text{max}}\)), whilst ingesting no fluid, glucose-electrolyte or placebo-electrolyte solutions (mean ±SD)
List of Tables

4.1 Descriptive characteristics of the subjects 82
4.2 Running economy over three consecutive days at 3.33 m.s⁻¹ (82% VO₂max), and at treadmill speeds reflecting 60% VO₂max and 85% VO₂max (mean ±SD) 83
4.3 Ventilation, heart rate and plasma lactate concentration for three consecutive days of 10 min treadmill running bouts at 60% VO₂max, 85% VO₂max and 3.33 m.s⁻¹ each day (mean ±SD) 85

5.1 Descriptive characteristics of the subjects 100
5.2 Oxygen uptake when running at 10.8 km.h⁻¹ (75% VO₂max) during min 6-10, 31-35 and 55-60 of 60 min running bouts at 65% (T1), 70% (T2) and 75% VO₂max (T3) (mean ±SD) 101
5.3 Pre and post exercise lung function after 60 min running bouts at 65%, 70% and 75% VO₂max (mean ±SD) 103

6.1 Selected physiological and metabolic characteristics of the subjects 114
6.2 Oxygen uptake during 10 min level treadmill running at 10.8 km.h⁻¹ (68% VO₂max) pre (RE1) and post (RE2) three prolonged running bouts (mean ±SD) 115
6.3 Ventilation and respiratory exchange ratio during 10 min treadmill running at 10.8 km.h⁻¹ (68% VO₂max) pre (RE1) and post (RE2) three prolonged running bouts (mean ±SD) 117
6.4 Heart rate and rectal temperature during 10 min level treadmill running at 10.8 km.h⁻¹ (68% VO₂max) pre (RE1) and post (RE2) three prolonged running bouts (mean ±SD) 118
6.5 Plasma glucose and lactic acid concentrations during treadmill running at 10.8 km.h⁻¹ (68% VO₂max), pre (RE1) and post (RE2) three prolonged running bouts (mean ±SD) 119
6.6 Plasma ammonia concentration during submaximal running at 10.8 km.h$^{-1}$ (68% $VO_2_{max}$) pre (RE1) and post (RE2) three prolonged running bouts (mean ±SD)

7.1 Physical characteristics and maximal physiological data of the subjects

7.2 Oxygen uptake during treadmill running at 10.8 km.h$^{-1}$ pre (RE1) and post (RE2) prolonged high-intensity running (80% $VO_2_{max}$ 60 min), whilst ingesting either no fluid, a 6% glucose-electrolyte or a placebo-electrolyte solution (mean ±SD)

7.3 Oxygen uptake during treadmill running at 80% $VO_2_{max}$ for 60 min at min 9-10 (80% RE1) and at min 59-60 (80% RE2), whilst ingesting either no fluid, 6% glucose-electrolyte or placebo-electrolyte solutions (mean ±SD)

7.4 The R-values during treadmill running at 10.8 km.h$^{-1}$ pre (RE1) and post (RE2) prolonged high-intensity running bouts (80% $VO_2_{max}$ 60 min), whilst ingesting either no fluid, 6% glucose-electrolyte or placebo-electrolyte solutions (mean ±SD)

7.5 The R-values during min 9-10 (80% RE1) and min 59-60 (80% RE2) of prolonged high-intensity running bouts (80% $VO_2_{max}$ 60 min), whilst ingesting either no fluid, 6% glucose-electrolyte or placebo-electrolyte solutions (mean ±SD)

7.6 Heart rate during running at 10.8 km.h$^{-1}$ pre (RE1) and post (RE2) prolonged high-intensity running bouts (80% $VO_2_{max}$ 60 min) and at 9-10 min (80%RE1) and 59-60 min (RE2), whilst ingesting either no fluid, 6% glucose-electrolyte or placebo-electrolyte solutions (mean ±SD)
The rate of perceived exertion pre, during and post prolonged running bouts (60 min 80% VO\(_{2\text{max}}\)) whilst ingesting no fluid, glucose-electrolyte or placebo-electrolyte solutions (mean ±SD)

Plasma concentration of glucose during treadmill running at 10.8 km.h\(^{-1}\) (65% VO\(_{2\text{max}}\)) pre (RE1) and post (RE2) 60 min at 80% VO\(_{2\text{max}}\), whilst ingesting either no fluid, 6% glucose-electrolyte or placebo-electrolyte solutions (mean ±SD)

Plasma concentrations of sodium during treadmill running at 10.8 km.h\(^{-1}\) (65% VO\(_{2\text{max}}\)) pre (RE1) and post (RE2) 60 min running bouts at 80% VO\(_{2\text{max}}\), whilst ingesting either no fluid, 6% glucose-electrolyte or placebo-electrolyte solutions (mean ±SD)

Rectal temperature during treadmill running at 10.8 km.h\(^{-1}\) (65% VO\(_{2\text{max}}\)) pre (RE1) and post (RE2) prolonged high-intensity running bouts (80% VO\(_{2\text{max}}\) 60 min), whilst ingesting either no fluid, 6% glucose-electrolyte or placebo-electrolyte solutions (mean ±SD)
## Apendices

<table>
<thead>
<tr>
<th></th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Ethical review and informed consent</td>
<td>A1</td>
</tr>
<tr>
<td>B</td>
<td>Lactate analysis</td>
<td>A4</td>
</tr>
<tr>
<td>C</td>
<td>Glucose analysis</td>
<td>A6</td>
</tr>
<tr>
<td>D</td>
<td>Ammonia analysis</td>
<td>A8</td>
</tr>
<tr>
<td>E</td>
<td>Potassium analysis</td>
<td>A9</td>
</tr>
<tr>
<td>F</td>
<td>Sodium analysis</td>
<td>A10</td>
</tr>
<tr>
<td>G</td>
<td>Haemoglobin analysis</td>
<td>A11</td>
</tr>
</tbody>
</table>
Chapter 1

INTRODUCTION

Running has received considerable attention from researchers in physiology, biomechanics and psychology since the 1920's and 1930's (Berstein, 1984; Elftaman, 1940; Norman, 1984). Present day researchers are still engaged in attempting to satisfy the curiosity of recreational and competitive runners by addressing issues, such as what criteria dictate optimal performance? (McMahon, 1984). It is important however to be aware that it may not be possible to fully understand the fundamental mechanisms of running without undertaking research of a multidisciplinary nature (Morgan, 1992).

In attempting to answer questions of what governs optimal running performance, one must give consideration to the complex mechanisms underlying the concept of efficiency (Williams, 1985). Thereby initially enhancing our understanding of running by trying to explain why one form of running is more efficient than another.

During the 1960's the term 'mechanical efficiency' was commonly used in the discussion of work physiology to define the energy expended to do a given amount of work (Astrand, 1960; Michael, Hutton and Horvath, 1961; Wasserman, Van Kessel, and Burton, 1967) and these description of the concept were based on thermodynamic considerations (Whipp and Wasserman, 1969). The muscular efficiency of an individual was expressed in 1975 as the ratio of work accomplished to energy expended (Gaesser and Brooks, 1975), and in 1980 as the ratio of mechanical power to metabolic energy expenditure. (Stainsby, Gladden, Barclay, and Wilson, 1980). Presently, efficiency of gross human movement has been defined as the relationship between work output and energy consumption and this relationship is used for
describing the economy of human muscular work. (Oksa, Rintamaki, Hassi, and Rissanen, 1993). This relationship has been reported as being influenced by many factors, including gender (Auro and Komi, 1986a), thermodynamics of muscle (Huxley, 1974), work intensity (Auro and Komi, 1986b), the biomechanical nature of the work (Auro and Komi, 1986c), weight and friction of clothing (Amor, Vogel and Worsley, 1973) ambient conditions (Oksa, Rintamaki, Hassi and Rissanen, 1993), mechanisms of metabolism (Whipp and Wasserman, 1969) and muscle fibre composition (Auro and Komi, 1987). Isolated muscle efficiency is markedly different and has been defined as the efficiency of phosphorylative coupling (Whipp and Wasserman, 1969). To put it more simply: "the efficiency for the whole process of converting foodstuff into tension" (Cavanagh and Kram, 1985).

1.1 Running efficiency

Accurately quantifying running efficiency and interpreting measures of running efficiency is difficult as there are many unresolved problems (Bosco et al, 1987). For example, to what extent does multi-joint muscle involvement influence the relationship between metabolic energy expended and measures of mechanical work in running (Aleshinsky, 1986)? Other examples include the problem of specifying relative energy costs of positive and negative work (Cavanagh and Kram, 1985), and to what extent does the storage and utilisation of elastic energy (muscle, tendon, ligament) influence running efficiency (Cavagna, 1978)? It is possible that the search for the variables which reflect the least cost to metabolism could be based on muscular properties rather than say, running style, and this warrants serious consideration. Additionally, it is questionable whether the most efficient style of running is the most effective, and this has led some researchers to consider the metabolic cost of running a certain distance (i.e. running economy) rather than running efficiency (Daniels, 1985).
may be that one adopts certain styles of running on the basis of economy rather than efficiency, but this is speculative.

1.2 Running economy

In 1977-80 there was conflicting use of terminology to define 'economy' (Conley and Krahenbuhl, 1980; Goldspink, 1977). Since fractional utilisation of VO_{2max} has been found to be an important determinant of endurance performance (Costill, Thomason, and Roberts, 1973), it is generally agreed that, regardless of the subject's VO_{2max}, the lower the submaximal oxygen uptake for a given workload the better. Note that while this allows for both different activities and different individuals performing the same activity to be compared, it cannot make any statement with respect to efficiency. One individual may be less economical than another, but may actually be more efficient because he is actually performing considerably more work (Ingen Schenau and Cavanagh, 1990).

Inter-subject variation has been found to be in the range of 12% (trained runners) to 17% in recreational runners (Costill, Thomason, and Roberts, 1973). Successful performance in endurance events is highly related to the energy cost, but is independent of the actual work done as stated by Cavanagh and Kram (1985): "A runner with a high VO_{2submax} at a given speed may indeed be performing more mechanical work than one with a lower oxygen cost, and both may therefore be operating at precisely the same muscular efficiency". Daniels (1985) stated: "Although it is possible to describe the energy demand of running at any particular velocity in terms of caloric expenditure, it is more appropriate to refer to the 'aerobic demand' of a particular pace". Hence, the submaximal oxygen uptake per unit body weight (VO_{2submax}) required to perform a given task has been accepted as the physiological
criterion for 'efficient' performance and is termed 'economy'. Relating steady state aerobic rate of submaximal oxygen consumption to a velocity of running is not entirely acceptable as a means of describing running economy. This is because it "does not indicate what portion of that VO₂ is a function of good or bad mechanics as opposed to being related to differences in metabolism which may exist in different people or under different conditions" (Daniels, 1985). However, a definition of running economy which is now generally accepted is 'VO₂ measured during submaximal running' (Cavanagh and Kram, 1985; Daniels, 1985; Morgan, 1985). It is possible that future refinement of this definition might include scaling for the VO₂-to-body size relationship (Nevill et al, 1992; Rogers et al, 1995).

There appear to be many factors that may affect running economy, such as: gender, (Daniels and Daniels, 1992) age, (Daniels, Oldridge, Nagle, and White, 1978), training (Womack et al, 1995) stride rate and frequency (Hogberg, 1952; Morgan, Martin, Krahenbuhl, and Baldini, 1991), shoe weight (Frederick, Daniels, and Hayes, 1984), wind (Pugh, 1970; Daniels, Daniels, Baldwin, and Bradley, 1986), air resistance and altitude (Daniels, Bradley, Scardina, Van Handel, and Troup, 1985), private self-consciousness (Martin et al, 1995), electrolyte concentrations (Lindinger and Sjogaard, 1991), fluid ingestion (Wilber and Moffat, 1992), changes in the recruitment pattern of motor units (Shinohara and Moritani, 1992), plasma ammonia concentration (Broberg and Sahlin, 1989) and so forth. Other possible additional energy expenditure factors could be linked to clothing, footing, terrain, fatigue (Gregor and Costill, 1973; Pivarnik and Sherman, 1990; Staab, Agnew, and Scinolfi 1992) and even the position of the temporomandibular joint (Jakush, 1982; McArdle, Goldstein, and Last, 1984; Schwartz and Novich, 1980). Intra and interindividual differences in running economy do exist but "the reasons for these differences, to what extent changes can be elicited, and how
best to bring about desirable changes in running economy are not so clear" (Daniels, 1985). However, it is accepted that a wide variety of factors may influence running economy (Williams, 1990). These factors include psychological, biomechanical and physiological variables (Williams, 1990; Crews, 1992; Morgan and Craib, 1992).

The principal aim of this present thesis was to further understand running economy within prolonged running bouts. An experimental procedure was used for quantitatively assessing running economy and was subsequently employed pre and post prolonged bouts of constant pace running. The Review of Literature (Chapter 2) addresses the interdisciplinary nature of running economy (Morgan, 1992). This is initially from a consideration of the influence of psychological state and running economy, and continues with an examination of the biomechanical factors and environmental variables and their influence.

1.3 Thesis overview

The studies described in this thesis are presented in an order that progresses from a consideration of daily variation in running economy, to the effects of exercise duration and intensity on running, and attempts to attenuate deteriorating running economy with fluid ingestion during prolonged intensive running bouts. The Review of Literature (Chapter 2) addresses the interdisciplinary nature of running economy (Morgan, 1992). This is initially from a consideration of the influence of psychological state and running economy, and continues with an examination of the biomechanical factors and physiological and environmental variables and their influence on running economy. Possible mechanisms underlying the expression of economical running patterns are identified and the multidisciplinary complexity is presented. The major part of the review concentrates on our
current understanding of the physiology of running economy - either on a day-to-day basis or within single prolonged exercise sessions - had received little attention from researchers (Williams et al., 1991). This was surprising considering that oxygen drift during prolonged exercise has been well documented.

The first study (Chapter 4) investigated the daily variation in running economy over three consecutive days of treadmill running. There is a need to know if individuals are economically different from day to day because a lower running economy is associated with deterioration in distance running performance (Morgan et al., 1994). Thus, the aim of this study was to establish the extent to which running economy varies on a day to day basis at 60% and 85% VO$_{2\text{max}}$ running speeds.

Respiratory muscle fatigue and increasing heart rate have been suggested as the primary causes of increasing oxygen consumption during prolonged exercise (Bye et al., 1984; Pate et al., 1992). The second study therefore (Chapter 5) focused on the possible links between running economy, respiratory muscle fatigue and heart rate drift during 60 min running bouts at 65% - 75% VO$_{2\text{max}}$. Although physiological disturbances in lung function and heart rate were evident, no change was demonstrated in the RE.

The third study (Chapter 6) examined the influence of duration and intensity of exercise on running economy. This determined that running economy deteriorated significantly following a 60 min run at 80% VO$_{2\text{max}}$ but the mechanisms remained elusive. It is known that prolonged high intensity exercise results in decreases in muscle glycogen stores and potential imbalances in both intracellular and extracellular electrolytes (Powers et al., 1990). Thus,
several variables including increased fat metabolism and loss of body fluids leading to electrolyte imbalances were considered to be the key to further understanding of the running economy template.

The fourth study (Chapter 7) examined the functional advantage of repeated ingestion of either glucose-electrolyte or electrolyte solutions (electrolytes held constant) on running economy following 60 min running bouts at 80% \( \text{VO}_2\text{max} \). Providing fluid to maintain subjects at or close to euhydration should prevent the adverse effect dehydration has on cardiovascular and thermodynamics (Sawka and Pandolf, 1990). Additionally, maintaining blood glucose levels could ensure a high rate of carbohydrate oxidation (Maughan, 1991), and this would be more economical in terms of the ATP produced per unit of oxygen consumed (Lamb, 1984).

Finally, the general conclusion (Chapter 8) attempts to move towards a speculative multidisciplinary perspective by drawing together the findings of the studies reported with supporting studies in the literature. The question of which fluid ingestion strategy is best to help maintain running economy during prolonged intensive exercise sessions is addressed, but remains unanswered.
Chapter 2

Review of Literature

2.1 Overview
A high VO\text{\textsubscript{2max}} is considered an important attribute for distance running performance (Costill, 1967) and among distance runners with a similar VO\text{\textsubscript{2max}} it has been suggested that more than 50% of the variation in performance could be explained by differences in running economy (Daniels, 1985; Conley and Krahenbuhl, 1980). Cureton and Sparling (1980) have shown that trained runners with comparable performance may show as much as 30% between-subject variation in running economy. Recent investigations suggest that there may be daily variations in running economy within subjects (Morgan, Martin, Krahenbuhl, and Baldini, 1991) and further study is recommended. Also, it is well documented that the ability to sustain prolonged exercise is limited by several factors, including dehydration (Armstrong et al, 1985) and carbohydrate availability (Hultman, 1971). However, there is scant information on the influence of prolonged running bouts on running economy, either in thermoneutral or in hot and humid environments. This thesis examines daily variation in running economy, identifies an intensity and duration of exercise at which running economy significantly deteriorates, and whether the provision of carbohydrate - electrolyte or electrolyte solutions during exercise can help maintain running economy. In so doing an attempt is made to identify the mechanisms associated with deteriorating running economy during prolonged, high-intensity exercise.

Initially, the physiological and environmental factors are reviewed with particular emphasis on trying to explain why individuals have different running economy, and to grasp the current understanding of why oxygen kinetics change during prolonged exercise. Following this the
review considers the main kinematic and kinetic factors associated with the running economy template, and finally considers psychological state and running economy.

2.2 Physiological and environmental factors and running economy

2.2.1 Age

The available data, including longitudinal and cross-sectional descriptive data are conclusive in some of their findings. Children are less economical in running than adults (Astrand, 1952; Krahenbuhl and Pangrazi, 1983) and running economy improves with age up to adulthood (Daniels and Oldridge, 1971), but older adults are less economical than young adults (Sidney and Shephard, 1977). Leger and Mercier (1984) examined studies and their review indicated that the gross energy cost of running increases 2% per year from 18 down to 8 years old. A 28% higher VO_{max} for young subjects compared with adults was reported by Krahenbuhl and Pangrazi (1983). Explanations offered for these differences include a lower 'efficiency' of children and differences in leg length, stride length, basal metabolic rate, body surface area to body mass ratio, reduced glycolytic capacity, training, and growth-related factors (Bar-Or, 1983; MacDougall, Roche, Bar-Or, and Moroz, 1983; Rowland and Green, 1988). Possible reasons for reduction in economy at the other end of the age continuum include decreased hip flexibility, reduced muscle elasticity and antagonistic muscle relaxation, increased body fat mass, and increased cardiac and respiratory demands (Larish, Martin, and Mungiole, 1987; Walters, Hislop, Perry, Thomas, and Campbell, 1983).

There have been numerous investigations of running economy in children following the early work of Astrand (1952), involving low speeds within the range of 1.92 - 3.37 m.s^{-1}
Astrand (1952) observed that there was a steadily reduced demand for oxygen at any given submaximal running speed (range: 2.22 - 3.61 m.s\(^{-1}\)) as children grew older (age: 4 - 18 years, male; 4 - 17 years, female), suggesting that their running economy improved with age.

An important finding during the 1970's was that with males aged 10 - 18 years (Daniels and Oldridge, 1971) \(V_O_{2\text{max}}\) (l.min\(^{-1}\)) and weight increase steadily with age but when \(V_O_{2\text{max}}\) was expressed in ml.kg\(^{-1}\) min\(^{-1}\) to reflect a correction for the increase in body mass, \(V_O_{2\text{max}}\) remained unchanged over the 8 year period, although running performance in one and two mile runs improved steadily. The investigators in these two studies utilised three different age groups who were involved in training to study the eight year span, whereas a more recent study (Krahenbuhl, Morgan and Pangrazi, 1989) tested the same male subjects (who were not engaged in training) at 10 and again at 17 years. These studies confirmed that running economy improved with age in normally active children, irrespective of whether the subjects were involved in running training. However, the subjects who were involved in running training evidenced slightly better improvements in performance and it could be that running training during childhood and adolescence may enhance performance.

An experiment was set up to try and address the question of whether children can be taught to run more economically, and involved 10 year old male subjects. The 12 week experimental design incorporated two control groups, a group that received running training, a group that received technical instruction on running and a group that undertook both running training and technical instruction on running (Krahenbuhl, Pangrazi, Stone, Morgan, and Williams, 1989). There were no changes in running economy among any of the groups studied. Krahenbuhl and Williams (1992) considered this with reference to the slightly better
improvements in performance of those subjects involved in running training, evidenced in the earlier studies of Daniels et al (1971 and 1978) and stated:

"it appears that the practice of running may be more important than the teaching of running, at least during later childhood".

Several studies in the 1980's that focussed on the question of why children are less economical than adults have indicated possible reasons. For example, children have exhibited both disadvantageous stride rates and stride lengths (imposed by shorter limbs) and higher resting metabolic rates (MacDougall et al, 1983), with age-related variations in oxygen demand simply reflecting decreases in basal metabolic rate that occur with age. However, only a small percentage of the change in submaximal and gross oxygen demands of running that occurs with age was attributed to these factors. Thorstensson (1986) hypothesised that children are less efficient than adults at utilising elastic energy in running, but his methods have been criticised. Others have compared the oxygen cost per stride during running of prepubertal boys and adult men and observed that the oxygen demand per stride for the two groups was similar, but at every speed the boys exhibited shorter stride lengths and higher stride frequencies, concluding that there are no differences in muscular efficiency of boys and adult males, and suggesting a basis for explaining age-related differences in oxygen demand (Unithan and Eston, 1990; Rowland, 1989). Also, these studies noted that younger subjects have a higher ventilatory equivalent for oxygen during submaximal running compared to adult males, suggesting another possible contributory factor for age-related differences in running economy. Presently, we do not seem to be able to fully explain the differences observed in running economy between children and adults.
2.2.2 Gender

There are conflicting data and mixed findings on gender comparisons of running economy among trained and untrained subjects. For instance, some report no gender difference (Bunc and Heller, 1989; Hopkins and Powers, 1982; Maughan and Leiper, 1983), whereas others suggest that males are more economical (Bhambini and Sigh, 1985; Bransford and Howley, 1977; Cureton and Sparling, 1980). Mayhew et al (1979) assessed 25 subjects (twelve members of the Northeast Missouri State University cross country team: 7 male, 5 female) and thirteen subjects with no formal training for the previous six months (7 male, 6 female). This study found no differences in the relative VO$_2$ between males and females at low running speeds of 135, 150, 165, and 180 m.min$^{-1}$, regardless of their training state, and this was in agreement with the findings of previous studies (Daniels, Krahenbuhl, Foster, Gilbert, and Daniels, 1977; Gehlsen and Dill, 1977; Leitch and Clancy, 1976).

Pate et al (1985) used recorded times for a 24.2 km road race to match male (n = 8) and female (n = 8) adult non-elite distance runners. Also, the groups were essentially at the same levels of training intensity and they did not differ in percentage of body fat or in VO$_{2max}$ ($p > 0.05$), although percent fat was more variable in the males (range: 10.3 - 22.3%) than females (range: 14 - 21.6%). Various physiological measurements were recorded while subjects ran submaximally on a level treadmill at speeds between 2.68 - 3.80 m.s$^{-1}$ and this revealed that the sexes did not differ in VO$_2$ (ml.kg$^{-1}$ min$^{-1}$), heart rate, respiratory exchange ratio, or ventilatory equivalent of oxygen during submaximal running (or at maximal exercise). The protocol used in this study is questionable, as speed was increased every two minutes and it is unlikely that a steady state was attained within two minutes. However, identical protocols were used with males and females and it could be argued that the two groups were compared
in terms of the oxygen costs of running observed under the same conditions. The respiratory exchange ratios indicate that comparably trained non-elite males and females demonstrate similar abilities to metabolise fat during submaximal running and this supports the conclusion of Costill et al (1979).

Past studies have been criticised as some methodological problems in matching male and female subjects ($VO_{2\text{max}}$, training background, running performance) were highlighted. For example, Wells et al (1981) utilised male and female marathon runners with equal $VO_{2\text{max}}$ values and reported that female subjects worked at a higher percentage of their $VO_{2\text{max}}$ (during marathon-pace running), appeared to be about equal in running economy, and outperformed their male counterparts in a marathon. A valid criticism of this particular study was that the women subjects had been running longer and trained harder than the male subjects (Daniels, 1985).

Another study of the running economy of elite male and elite female middle and long distance runners with similar characteristics to those reported in other studies of elite runners (Pollock, 1977) included twenty Olympians (1984 Olympic Games: 8 - female; 12 - male), and found gender differences (Daniels and Daniels, 1992). At three common submaximal running velocities (4.47, 4.83 and 5.17 m.s$^{-1}$), the male runners in their study consumed less oxygen than their female counterparts and this supported the earlier findings for trained and untrained males and females of Brandsford and Howley (1977) and Howley and Glover (1974). Additionally, when two of the marathon runners (one male/one female; both American record holders) with almost identical $VO_{2\text{max}}$ were compared, the male was more economical, both at common velocities and at common relative intensities of running (83.1%
This is in general agreement with an earlier study of highly trained athletes (Conley and Krahenbuhl, 1980). The average VO$_{2\text{max}}$ was different, with the males having the higher VO$_{2\text{max}}$ (66.2 versus 75.4 ml.kg$^{-1}$ min$^{-1}$). When the eight women who had the highest VO$_{2\text{max}}$ were closely matched with eight male runners of equal VO$_{2\text{max}}$, the resulting vVO$_{2\text{max}}$ data gave the males a 7-8% performance advantage were both to run at VO$_{2\text{max}}$. Additionally, when the eight most economical female runners were matched with eight males of equal running economy, at comparable absolute running velocities, this revealed that the males had a 14% VO$_{2\text{max}}$ advantage and a 14% vVO$_{2\text{max}}$ advantage. It appears that having equal VO$_{2\text{max}}$ or equal economy does not guarantee equal performance, even in a purely aerobic sense. Another important finding was that when elite males and elite females both run at a similar intensity (male: 87.9% VO$_{2\text{max}}$; female: 85.5% VO$_{2\text{max}}$; p > 0.05) when at a blood lactate concentration of 4.0 mM, there were no gender differences in running economy at relative (%VO$_{2\text{max}}$) race intensities. The authors recommend:

"when runners are tested for comparative purposes, VO$_{2\text{submax}}$ (economy) data should be collected at speeds ranging up to 95% VO$_{2\text{max}}$, as well as VO$_{2\text{max}}$ and vVO$_{2\text{max}}$ data", if one wants the clearest possible individual 'aerobic profile'.

2.2.3 Vertical displacement

It has been suggested that females exhibit greater vertical displacement of the body when running (Bransford and Howley, 1977). A study to compare the metabolic costs and gait patterns of running (and walking) at self-selected, comfortable speeds in twenty four physical education students (12 female, 12 male), none of whom were competitive runners, found the
gross and net metabolic costs in kcal.kg\(^{-1}\) min\(^{-1}\) were higher (by 14%; p < 0.05) in females than in males (Bhamhani and Singh, 1985). It is worth noting that the females in this study had a 7.9% greater body fat content than the males, which is almost identical to the mean sex difference in percentage fat of the subjects in the Cureton and Sparling study (1980). A comparison of the vertical lift per stride revealed no differences between the sexes for running (and walking). This is in agreement with Bransford and Howley (1977), who observed a no sex difference in vertical lift per stride and in the vertical lift per km of a distance run, with subjects running at similar selected slow speeds. It should be noted that the displacement of the centre of gravity was not measured in the Bhamhani and Singh study (1985), but rather the vertical displacement of the fifth lumbar vertebra and this may not reflect accurately the vertical displacement of the whole body. The vertical lift per running stride in this study was 8.8 cm in males (speed range: 2.38-2.79 m.s\(^{-1}\)) and 8.89 cm in females (speed range: 2.28-2.51 m.s\(^{-1}\)). Cavanagh et al (1977) reported lower vertical lift per stride in good (8 cm) and elite (7.6 cm) long-distance runners when running at 4.96 m.s\(^{-1}\) and Williams and Cavanagh (1987) found a consistent trend in the vertical oscillation of actively training, non-elite distance runners. Although nonsignificant, it is worth noting that Williams and Cavanagh (1987) reported average vertical lift kinematics of 9.1 cm, 9.3 cm and 9.6 cm for the higher, medium and lower economy runners when running at 3.6 m.s\(^{-1}\). However, one must question whether such small absolute differences are really meaningful.

2.2.4 Body composition

Females in general are not expected to compete in distance running events against males as it has been observed that average performances are 20 - 30\% greater for men than women on distance running tests (Cureton, Hensley, and Tiburzi, 1979; Katch, Pechar, McArdle, and
Weltman, 1973). One factor that appears to account for sex difference in distance running performance is the greater mean percentage fat of females compared with males (Katch and McArdle, 1977; Wilmore, Brown, and Davis, 1977). It is well founded that body fat influences aerobic capacity by lowering the oxidative energy that can be made available to move each kilogram of body weight (Brooks, Hittleman, Faulkner, and Beyer, 1970). There have been several studies which have employed the addition of external weight to the body to investigate the effect of excess body weight (fat) on metabolic responses to human locomotion (Goldman and Lamoietro, 1962; Hanson, 1973; Miller and Blyth, 1955). An interesting study by Cureton and Sparling (1980) involved young, experienced competitive distance runners (10 male, 10 female) who were similar in physical condition (nonsignificant differences in mean VO_{2max} values, expressed as ml.kg^{-1} min^{-1} fat free weight), who were in training (mean weekly distances: 62 km - females; 40 km - males). Methods included an added weight condition for the males, so that each male's total percentage excess weight, defined as the fat weight plus added weight divided by the total weight, was equal to the percentage fat of a female with whom he was arbitrarily paired. The average increase in percentage excess weight was 7.5% (i.e. the mean sex difference in percentage fat). The main finding of this study was that equating the percent excess weight of the groups of male and female runners reduced the sex difference in treadmill run time and 12-minute performance by approximately 30%. Assuming the added weight had a similar effect on performance as carrying additional body fat, these results indicate that approximately one-third of the sex difference on these measures of distance running performance was due to the difference between the men and women in percent body fat. Thus, it would appear that the average young, trained female distance runner will experience a greater physiological stress when
running at submaximal speeds as this involves running at a higher percentage of a lower 
VO_{2max} expressed relative to body weight.

2.2.5 Body temperature

Some studies have suggested a positive association between body temperature and VO₂ 
during prolonged, constant load and submaximal exercise under hyperthermic conditions in 
humans (MacDougall, Reddan, Layton, and Dempsey, 1974; Saltin and Stenberg, 1964). 
Saltin and Stenberg (1964) reported a 5% rise in VO₂ during 3 hours of constant-load exercise 
under normal conditions. MacDougall et al (1974) also observed that VO₂ was higher in 
subjects who exercised at 70% VO_{2max} under hyperthermic conditions compared to normal or 
hypothermic conditions. This slight increase in VO₂ could be linked to increased peripheral 
blood flow and sweating demands, a rise in ventilatory rate and a decrease in oxidative 
phosphorylation efficiency (Gaesser and Brooks, 1984). It has been demonstrated that when 
rat skeletal muscle and liver mitochondria are incubated at high physiological temperatures, 
more oxygen is required to synthesise a given amount of ATP (Brooks, Hittleman, Faulkner, 
and Beyer, 1971). However, other studies have found little or no change in VO₂ during 
hyperthermic exercise (Rowell, Brengelmann, Murray, Kraning, and Kusumi, 1969). They 
suggest that the number, type or force-generating characteristics of muscle fibres may change 
as internal temperatures increase. This apparently enhanced muscular efficiency produces a 
constant aerobic demand (Rall, 1985). Data from some studies which have shown a reduction 
in VO₂ during the latter portion of a prolonged run also support the possibility of increased 
muscular efficiency with elevated muscle temperatures (Maron, Horvath, Wilkerson, and 
Gliner, 1976).
Oksa et al (1973) designed a study to evaluate how ambient temperatures -15°C and 21°C affect the efficiency of muscular work in 12 young sedentary males during incremental step exercise. Their results demonstrated that gross efficiency is modified by various temperatures and work loads during step exercise and the difference seemed to be related to changes in rectal and skin temperatures, with poorer efficiency in light work at -15°C probably due to the need to warm up the muscles, and at 21°C the activation of heat dissipation systems was probably responsible for the lower efficiency in heavy work. Interestingly, their highest efficiency values were reflected as rather similar rectal temperatures (37.4 - 37.7°C) and identical mean skin temperatures (32.8°C) with a 'slightly warm' thermal sensation in the legs. Others have suggested that the extent of the thermoregulatory response may be weakened as a result of training-induced adaptations to exercise in a hot environment. Increased plasma volumes may help spare energy demands during hyperthermic exercise (Bailey and Pate, 1991). It is important to note that several recent studies (Bahr, 1992; Elliot et al, 1992; Neary et al, 1993) have cautioned against causal quantification of excess VO₂ due to temperature alone. For example, muscle temperature was shown to change independent of rectal temperature.

2.2.6 Heart rate and ventilation

Respiratory muscles have been estimated to use only a small fraction (approximately 1.5%) of total oxygen consumption or cardiac output in healthy individuals at rest (Daniels, Foster, Daniels, and Krahenbuhl, 1977). During the 1980's some estimates of one or both of these factors during exercise were made (Anholm, Johnson, and Ramanathan, 1987; Collett, Perry, and Engel, 1985; Olgiati, Atchou, and Cerratelli, 1986) and it was suggested that increases in both these variables might limit blood flow to, and thus oxygen consumption by other
muscles during exercise (Bye, Farkas, and Roussos, 1983). Implicit in these earlier and recent research projects (Coast and Krause, 1993) has been the assumption that, during increased work of breathing, all of the difference between the resting and working total oxygen consumption and cardiac output is accounted for by the demands of the respiratory muscles, and this may be incorrect (Coast and Loy, 1991; Rusch, Shepherd, Webb, and Vanhoutte, 1981). However, earlier studies found that ventilation and heart rate contribute to whole body oxygen cost during exercise (Kitamura, Jorgensen, Gobel, and Taylor, 1972; Millic-Emili, Petit, and Deroanne, 1962). Recently Pate et al (1992) found both variables were positively correlated with oxygen consumption, indicating that better running economy was associated with a lower heart rate and ventilation.

In another study Daniels and Daniels (1992) reported that elite males and elite female middle and long distance runners both run at the same relative intensity of effort eliciting a 4 mM.l\textsuperscript{-1} blood lactate accumulation. The women were at 90.8\% of HR$_{\text{max}}$, the men at 89.3\% of their HR$_{\text{max}}$ and this difference was nonsignificant. However, at any common absolute running velocity, the women were always at a higher HR and higher blood lactate concentration than were the men, just as they were at a higher VO$_2$. Results confirming or refuting potential interrelationships among heart rate, ventilation and running economy during the course of an endurance or ventilatory training program are lacking. However, recent research findings preclude a dominant role for factors such as ventilatory or cardiac work (Poole et al, 1991; Womack et al, 1995).
2.2.7 Air and wind resistance

The treadmill is now commonly used to simulate overground running, as this facilitates the collection of data pertaining to physiological responses of the exercising subject, such as oxygen consumption during running. There have been several investigations to try and establish whether differences exist between the energy demands of treadmill and overground situations (Williams, 1986). Although treadmill running has been shown to be more economical (i.e. lower $V_{O_2}$) than overground running, there appears to be some agreement that there are no differences in oxygen demands for level running on the treadmill and overground at low to moderate speeds (McMiken and Daniels, 1976; Bassett, Giese, Nagle, Ward, and Raab, 1985). However, the similarity between treadmill running and overground running remains a controversial topic (Morgan and Craib, 1992). At faster speeds (4.47 - 5.37 m.s⁻¹) level overground running in calm air increased the aerobic demand of running by 7.1% compared with the energy requirement of level treadmill running (Morgan and Craib, 1992). Pugh's studies (1970 & 1971) involving treadmill and track running indicated an 8% greater energy cost for track running at almost 6 m.s⁻¹, and after calculating the energy 'cost' of overcoming air resistance on the track to be up to 7.5% (middle distance) and 13% (sprints) of the total energy cost, Pugh (1971) concluded that track running demands a higher $V_{O_2_{submax}}$ than does the treadmill at the same speed, due to the effects of air resistance rather than mechanical differences between overground and treadmill locomotion. Maksud et al (1971) and Davies (1980) support the view that track running involves greater energy demands compared to treadmill running.

Frishberg (1983) suggested that there were differences in metabolic demands between overground and treadmill level running, after he found that oxygen debt following a 100-yd
sprint was 36\% higher than on the treadmill. However, the validity of assessing total energy expenditure by measuring oxygen debt is questionable. Additionally, Frishberg based his value of a 3\% contribution of air resistance to the total energy expenditure during sprinting on the past findings of Hill (1927), but research in the early 1970's has shown this figure to be too low (Pugh, 1971). Bassett et al (1985) studied the energy requirements of inclined running (treadmill and overground) and supported the view of Ingen Schenau (1980) that treadmill running (5.7\% grade) and overground running (uphill) require the same amount of energy.

The American Academy of Sports Medicine (1980) presented theoretically derived prediction formula for overground hill running as it was believed to involve greater metabolic costs than inclined treadmill running. It could be argued that the treadmill belt moves by under the feet while the body is in flight and that runners do not undergo a net change in potential energy during treadmill running because they are running 'in place'. But these arguments appear to be invalid as the same change in potential energy occurs for treadmill and overground running (Ingen Schenau, 1980). According to Bassett et al (1985) the ACSM prediction formula for overground hill running may over-estimate VO\(_2\). However, it may be that further research is warranted on the effects of wind resistance, perceptual difficulties associated with treadmill running, and variation in the belt speed with each footplant (Cavanagh and Williams, 1982).

2.2.8 Drafting

There appears to be little experimental data available on 'drafting', with some research suggesting this 'strategy' could result in approximately 3\% -6\% energy savings (Pugh, 1971; Kyle, 1979). This strategy appears worthy of future consideration.
2.2.9 Altitude

Limited research indicates VO₂ demands are lower at altitude. A potential explanation for this finding is that a decreased energy demand is required to move through the less dense air of altitude (Hagerman, Addington and Gaensler, 1975; Daniels et al, 1977). This adds support to the effects of air resistance on VO₂max, with resulting higher aerobic demands during overground running compared to treadmill running. If it is true that overground running is more demanding than is treadmill running, then it could be that many reported data from laboratory treadmill testing are under-estimations of energy demands during race situations, if high running speeds are utilised.

2.2.10 Surface

McMahon and Greene (1978) have shown that surfaces with appropriate mechanical characteristics (about three times the stiffness of the body) have the property of passively increasing step length which may allow subjects to maintain speed with a lower expenditure of energy. They calculated that this increase in coefficient of performance could be in the order of 2% (MacMahon and Greene, 1979).

2.2.11 Fatigue

The association between running economy and fatigue is unclear. Early research focussed on determining the metabolic costs of adopting different race pace strategies to achieve minimal oxygen costs during short term, maximal runs (Adams and Bernauer, 1968; Ariyoshi, Yamaji, and Shepherd, 1979; Robinson, Robinson, Mountjoy, and Bullard, 1958). Relatively recent research examining the metabolic and biomechanical consequences of longer, high-intensity runs produced conflicting results, with one study reporting greater oxygen demand (i.e.
worsened economy) in two elite male runners within a few days of a competitive distance race (Cavanagh, Andrew, Kram, Rodgers, Sanderson, and Henig, 1985). However, another study demonstrated no change in economy in eight non-elite male runners one day after a hard training session (Martin, Fernhall, and Krahenbuhl, 1987).

Morgan et al (1990) extended this work by quantifying short-term changes in running economy and running mechanics following a prolonged maximal run. They found no difference in the running economy (range = 42.3 - 42.6 ml.kg⁻¹ min⁻¹) of 16 male distance runners one, two and four days following a 30-min level run (3.33 m.s⁻¹) at a mean exercise intensity of 89% VO₂max. This metabolic response mirrored that obtained by Martin et al (1987) and was accompanied by an identical decrease in RER, suggesting that the post-run increase in the percentage of fat kilocalories associated with the reduction in RER was not sufficient to produce a rise in the aerobic demand of running. Morgan et al (1990) did not quantify free fatty acid concentration and muscle glycogen depletion or exercise dietary control. However, they speculated that muscle glycogen levels were not depleted based on the findings of Costill et al (1971). Also, Kirwan et al (1988) have demonstrated reductions in muscle glycogen concentration, running economy and RER in trained male subjects who performed 5 days of high-intensity distance running (20 km per day at approximately 80% VO₂max), while on a low carbohydrate diet, suggesting a potential influence of dietary intake on RER. Several studies have produced corroborating data indicating no differences in selected lower body mechanics after a hard training session (Martin et al, 1987), speed-controlled fatigue runs (Williams, Snow, and Agruss, 1988) and prolonged maximal runs (Morgan et al, 1990). This suggests that observed reductions in RER do not appear to be counterbalanced by a mechanical shift towards a more economical gait pattern.
2.2.12 Training

Previous studies have indicated that endurance exercise training can induce large increases in VO2max, maximum work capacity and endurance, due in part to a greater arterio-venous difference (Holloszy and Booth, 1976) and reported possibly as a result of an increase in the respiratory capacity of skeletal muscle (Holloszy, 1967). A high positive relationship has been demonstrated between VO2max and the volume density of mitochondria in biopsies from the vastus lateralis muscle in both trained and untrained men and women (Hoppeler, Luthi, Claussen, Weibel, and Howald, 1973). However, presently there appears to be a lack of consensus regarding the effects of training on running economy.

Some studies have revealed no change (or a slight increase) in the aerobic demand of running following relatively short training periods of 6 - 11 weeks (Daniels, Yarbrough, and Foster, 1978; Lake and Cavanagh, 1990). Wilcox and Bulbulian (1984) reported no difference in economy over an 8-week cross-country training session in 7 collegiate runners who averaged 60 to 70 miles of running and 2 high-intensity workouts per week. Other investigators have reported a wide range of improvement (1% - 18%) in running economy following training over time spans of 14 weeks to 5 years (Conley, Krahnenbuhl, Burkett, and Millar, 1984; Patton and Vogel, 1977; Svedenhag and Sjodin, 1985). Sjodin et al (1982) reported that supplementing regular training with a weekly 20 min run performed at the velocity eliciting a 4mM.L^-1 blood lactate concentration (vOBLA) improved the economy of 8 well-trained middle and long-distance runners. This exercise intensity was conceived as representing a maximal threshold for the maintenance of a steady state and therefore as an optimal training intensity (Kindermann, Simon, and Keul, 1979; Mader, 1980). Also, as muscle metabolic characteristics related to OBLA had been shown to include muscle fibre type composition,
capillary density, and the balance between glycolytic and oxidative enzyme activities (Jacobs, 1981), Sjodin et al (1982) examined muscle metabolic adaptations with training as well. Their observed change in VOBLA correlated positively to the percentage of type 1 fibres in the vastus lateralis ($r = 0.83$, $p < 0.01$). Additionally, the activity of phosphofructokinase (PFK) and the ratio of PFK/citrate synthase (CS) decreased after the 14 weeks steady state training intensity (VOBLA), combined with an increased relative activity of the H-LDH (lactate dehydrogenase) isozyme. This suggested the rate of glycogenolysis during exercise would be relatively slower and the potential to oxidise pyruvate and/or lactate increased. Of importance was their finding that the change in absolute VO$_2$ running at 15 km per hour was correlated with the increase in relative activity of H-LDH after VOBLA training ($r = -0.75$, $p < 0.05$). This indicated that the improved running economy could be due to an improved intracellular oxidative capacity, which would increase the rate of lactate oxidation.

A limitation of Sjodin et al's study (1982) was the lack of a control group of subjects. Nonetheless, their conclusion that muscle fibre type composition may be an indicator of the trainability of the musculature is of importance. Particularly as others have reported that type 1 fibres have a higher respiratory capacity than type 2 fibres, both qualitatively (Gollnick, Armstrong, Saubert, Piehl, and Saltin, 1972) and quantitatively (Essen, Jansson, Henriksson, Taylor, and Saltin, 1975; Henriksson and Reitman, 1976). Also, correlations of 0.75 and 0.74 have been found between percentage type 1 fibres and VO$_{max}$, and between percentage type 1 fibres and muscle respiratory capacity respectively (Ivy, Costill, and Maxwell, 1980), in accordance with findings from previous research (Berg, Thorstensson, Sjodin, Hulten, Piehl, and Karlsson, 1978; Foster, Costill, Daniels, and Fink, 1978; Rusko, Havu, and Karvinen, 1978). Svendenhag and Sjodin (1985) also observed improvements in
running economy over a mean period of 22 months in 16 elite male distance runners who performed long distance, uphill, and interval running.

One outcome of running training may be an improved running economy but the level of fitness at the start of a study could be a factor in whether changes in economy will be found. A preponderance of data indicates that trained subjects are more economical than their untrained or less trained counterparts (Dolgener, 1982; Mayers and Gutin, 1979; Pollock, Jackson, and Pate, 1980). However, running economy has been found to vary among trained runners, with correlations between oxygen uptake at a specified velocity and running time for a particular racing distance ranging from 0.08 to 0.83 (Powers, Dodd, Deason, Byrd, and McNight, 1983). It is possible that training exerts an influence on economy and/or that economical runners are endowed with an anatomical or genetic makeup that produces an economical running style and favours success in longer events (Daniels, 1985). Thus, long distance runners may gravitate naturally towards endurance events and differences in the aerobic demand of running among various types of runners might reflect differences in the expression of a genetic predisposition towards worse or better economy (Morgan, Martin, and Krahenbuhl, 1989).

It has been suggested that running economy becomes more important in longer races (Fay, Londereec, Lafontaine, and Volek, 1989) and middle distance elite runners exhibit better economy at faster speeds whereas elite marathon runners tend to be more economical at slower running speeds (Daniels and Daniels, 1992). Daniels and Daniels (1992) concluded that due to a flatter regression curve (relating VO₂ and running economy), 800m and 1500m are more economical at their specialist race pace, but not at marathon pace. Additionally, they
found the elite male marathoners to be more economical at a comparatively slower speeds compared to the middle distance athletes. There have been mixed findings on the effects of both detraining and overtraining and this appears to be an area that warrants further research (Kuipers and Keizer, 1988; Houmard, Kirwan, Flynn, and Mitchell, 1989; Houmard, Costill, Mitchell, Spark, Hickner, and Roemmich, 1990).

2.2.13 Running velocity and $VO_2$

The concept of a linear relationship between a range of running speeds and $VO_2$ seems to hold during submaximal running where energy demands are predominantly met aerobically (Daniels, Fitts, and Sheehan, 1978), with regression lines varying considerably (flatter at slower speeds), depending upon the speeds chosen for comparison (Daniels et al, 1977). At higher running intensities where energy demands are met primarily through anaerobic metabolism the relationship is less clear, as blood lactate accumulates and total energy expenditure appears to rise exponentially as a function of running velocity, at least beyond 80% $VO_{2\text{max}}$ (Daniels et al, 1977). There is evidence that runners utilise approximately 85% $VO_{2\text{max}}$ during a 10 km race (Costill, 1970), 82-86% $VO_{2\text{max}}$ for 10 mile races (Farrell, Wilmore, Coyle, Billing, and Costill, 1979), and 75% $VO_{2\text{max}}$ or more during a marathon (Fox and Costill, 1972). A strong relationship has been found between the pace at which most distance runners race and the pace where large amounts of lactate start to accumulate in the blood (Acevedo and Golgfarb, 1989; LaFontaine, Londeree, and Spath, 1981; Morgan, Baldini, Martin, and Kohrt, 1989). Running at a pace corresponding to a physiological steady state has been thought to be the most efficient race strategy as high lactate accumulation has been associated with fatigue (MacClaren, Gibson, Parry-Billings, and Edwards, 1989).
Fay et al (1989) reported a strong association between treadmill velocity at predetermined lactate accumulation points (2.0 and 4.0 mM.L\(^{-1}\)) and race pace (5km - 16.09km), with correlation coefficients ranged between 0.84 and 0.94, whereas the oxygen cost of running at each of three submaximal paces (3.26 - 4.01 m.s\(^{-1}\)) only correlated moderately with each race pace (r = -0.40 [ns] to - 0.63, p < 0.05). In this study the plasma lactate 4.0 mM pace was a significantly better predictor of 5 km performance than either VO\(_{2\text{max}}\) or plasma lactate 2.0 mM pace, and the mean treadmill speed at plasma lactate 4 mM.L\(^{-1}\) of 4.29 m.s\(^{-1}\) was almost identical to the average 5 km race pace (4.31 m.s\(^{-1}\)). Another study that found a similar modest correlation between performance time and VO\(_{2\text{submax}}\) was by Farrel et al (1979) for a 9.7 km distance. Also, Conley and Krahenbuhl (1980) reported the relationships between steady state VO\(_2\) at 241, 268, and 295 m.min\(^{-1}\) and 10 km time were 0.83, 0.82, and 0.79 respectively. In contrast, Williams and Cavanagh (1987) observed no significant relationship between performance (10 km) and VO\(_{2\text{submax}}\) and this finding has been supported by others (Foster, Daniels, and Yarborough, 1977).

Performance times have subjects running at different speeds, which is different from comparing data for which all subjects run at the same constant speed. This may lead to problems as it could be that faster runners may be uneconomical when running at speeds that are slower than their race pace or vice versa. For example, the findings of Pate et al (1992) in their unique study of the determinants of running economy (2.68 m.s\(^{-1}\)) in habitual distance runners (n = 119 male; 69 female), indicated that running economy tended to be poorer in the subjects with a higher VO\(_{2\text{max}}\). They suggested that the runners with higher VO\(_{2\text{max}}\) values may have been less comfortable with running at this low pace, and therefore were less economical due to mechanical and/or neuromuscular factors. Perhaps 'optimising processes' may involve
achieving fine tuning to balance between minimising total body energy costs and avoiding peripheral muscle fatigue, even though this may result in higher oxygen uptake at certain workloads, as has been found for cycling (Patterson and Moreno, 1990).

### 2.2.14 Effectiveness

Most muscles acting across human joints work at a mechanical disadvantage (Hamill and Knutzen, 1995). There is little quantitative information in the literature concerning individual differences in muscle and joint geometry, and this may be an important source of variation in economy between different individuals. For example, with short muscle fibres and a long compliant tendon the positive work (from re-utilisation of elastic energy) can be considerable with little metabolic cost, during a 'stretch-shortening cycle' of a muscle tendon complex (Haan, Ingen Schenau, Ettema, Huying, and Lodder, 1989).

### 2.2.15 Lactate

Blood lactate has been measured during economy run studies to provide confirmatory evidence of a steady-state energy condition that aerobic metabolism was the predominant energy source during submaximal running bouts, and to supplement other physiological measures (Heck, Mader, Hess, Mucke, Muller, and Hollman, 1985; Sjodin and Jacobs, 1981). An important finding of the study by Farrell et al (1979) was that the onset of plasma lactate accumulation appeared to be closely related to the race pace for long distance races. The authors suggest two interpretations of this finding. Firstly, large accumulations of lactate should be avoided during distance races due to the possible inhibition of fat utilisation with the consequence of a greater dependence on carbohydrates, leading to glycogen depletion which has been associated with exhaustion during prolonged exercise (Costill, Gollnick, Jansson, Saltin, and Stein, 1973; Hermansen, Hultman, and Saltin, 1967). Secondly, a small
amount of plasma lactate is necessary to maintain free fatty acid levels at an optimal level (Boyd, Giamber, Mager, and Lebovitz, 1974).

Daniels and Daniels (1992) when discussing 'relative intensity', reported that eight of their subjects (elite marathoners) performed at an average of 85% \( \text{VO}_{2\text{max}} \). Also, they found elite male and female middle and long-distance runners both run at the same intensity (approximately 86% \( \text{VO}_{2\text{max}} \)) when at a blood lactate accumulation of 4.0 mM.l\(^{-1}\). On this topic Coyle et al (1991) have reported that elite cyclists work at a higher percentage of \( \text{VO}_{2\text{max}} \) at blood lactate threshold compared to highly trained non-elite cyclists (79% versus 75%; \( p < 0.05 \)) who had identical \( \text{VO}_{2\text{max}} \) values but significantly different performance times over 40 km, indicating that the measurement of lactate threshold can distinguish between well-trained and elite cyclists.

Conley and Krahenbuhl (1980) speculated that when performers are of comparable ability (10 km performance time) with similar maximal aerobic capacities that running economy is important to distance running because the relative intensity of a given pace would be lower for an economical runner than it would be for a less economical runner with a comparable \( \text{VO}_{2\text{max}} \). They suggested that variation in performance not accounted for by the economy of the subjects may be due to interindividual differences in muscle fibre composition, anaerobic threshold, and peak muscle and blood lactate tolerance. Williams and Cavanagh (1987) found that the percent of slow twitch muscle fibres and \( \text{VO}_{2\text{max}} \) correlated highly with 10 km performance times \( (r = -0.88 \) and \( r = -0.76, \) respectively).
2.2.16 Muscle fibre type

Until the early 1970's most attempts to explain the physiological variables for success in distance running were limited to measurements of metabolic and circulatory responses during exercise (Costill, 1969 & 1970). It has been suggested that the percentage of ST fibres may be a factor governing success in distance running (Gollnick et al, 1972). Several studies have reported a large percentage of ST fibres in the skeletal leg muscles of distance runners compared to athletes participating in non-endurance sports or untrained subjects (Bergh, Thorstensson, Sjodin, Hulten, Piechtl, and Karlsson, 1976). In one study muscle samples (gastrocnemius) were obtained from 14 elite distance runners, 18 trained middle distance runners, and 19 untrained males (Costill, Fink, and Pollock, 1976). This study found on the average, the elite runners possessed more ST fibres (79%) than either the untrained men (57%) or the middle distance runners (61%).

Many of these early studies and the more recent research on skeletal fibre composition also examined muscle enzyme activities, for a variety of sound reasons. For instance, muscular adaptation to exercise includes an enhanced capacity to produce ATP partly via quantitative increases in selected glycolytic and Krebs cycle enzymes (Holloszy, 1967; Baldwin, Winde, Terjung and Holloszy, 1973). Also, comparative studies have shown a positive relationship between the ability of a muscle to perform prolonged exercise and the activity of its respiratory enzymes (Costill et al, 1976). For example, succinate dehydrogenase (SDH) participates in the Krebs cycle and its histochemical detection is a useful index of the activity of this cycle and therefore the oxidative capacity of muscle (Holloszy, Oscai, Don, and Mole, 1970). The levels of SDH activity correlate highly with VO2max (Costill, Daniels, Evans, Fink, Krahenbuhl, and Saltin, 1976), and in the muscle of elite runners has been found to be
greater than that of middle distance runners and untrained men (Costill et al., 1976). Additionally, numerous studies have shown that endurance training increases muscle SDH activity 1.6 - 3.4 times the level of untrained muscle (Eriksson, Gollnick, and Saltin, 1972; Gollnick, Armstrong, Saltin, Saubert, Sembrowich, and Shephard, 1973).

Another study by Simoneau and Bouchard (1989) involving sedentary and active but non-elite young men and women reported no difference in activities of aerobic-oxidative enzyme markers between genders, whereas levels of glycolytic enzyme markers (hexokinase, phosphofructokinase, lactate dehydrogenase) were higher in male than in female skeletal muscle. Simoneau and Bouchard (1989) suggested this was a possible explanation for some of the metabolic differences that others (Chen and Bouchard, 1986) have observed between men and women.

### 2.2.17 Genotype

There have been few studies that have attempted to specifically examine the influence of fibre type on running economy, whereas the influence of muscle fibre type on work efficiency of isolated muscle has been widely studied (Suzuki, 1979). Some studies have reported that training increases the number of fibres with high oxidative capacities (Barnard, Edgerton, and Peter, 1970; Morgan, Cobb, Short, Ross, and Gunn, 1971). Others have suggested that there was no change by training in the contractile properties of ST and FT fibres as measured by the mATPase stain (Gollnick et al., 1972). Some investigations have demonstrated that fibre distribution is unchanged as a result of 4 to 6 months of training in young boys (11 to 13 years) and adult males (Eriksson et al., 1972; Gollnick et al., 1973). Costill et al (1976) obtained muscle samples from the lateral head of the gastrocnemius and examined muscle enzymes and the fibre composition of international-caliber, male and female track athletes.
(sprinters, middle distance & distance runners, field athletes) and untrained men and women. They found that the distance runners had a high percentage ST fibres (range; 63 - 74%) and the sprinters had a low range of ST fibres (21 - 28%). Also, Costill et al (1976) reported that male and female track athletes are similar in terms of fibre composition and the main distinction between the sexes was the significantly larger fibre areas of the male athletes, but it should be noted that there were no female distance runners in this study. On the basis of their findings Costill et al agreed with the earlier suggestions of Saltin (1973) that the athlete's success in strength, speed, and/or endurance events results, in part, from his or her genetic endowment. Some researchers of sedentary and active (non-elite), male and female, and unrelated or related (brothers, dizygotic and monzygotic twins) subjects have concluded from their observations that various environmental and other nongenetic conditions are responsible for a large fraction of the human variation in the skeletal muscle fibre-type proportion, fibre-type areas, and enzyme markers of energy metabolism (Bouchard, Simoneau, Lortie, Bouly, Marcotte, and Thibault, 1986; Simoneau and Bouchard, 1989). Alternatively, it has been reported that the proportion of type 1 muscle fibre type in human skeletal muscle was genetically fixed (Komi, Vitasalo, Havu, Thorstensson, Sjodin, and Karlsson, 1977). Importantly, no difference was observed in muscle fibre type among 31 trained male runners who exhibited good, medium and poor running economy (Williams and Cavanagh, 1987).

2.2.18 Efficiency and fibre type

Coyle et al (1992) estimated efficiency in 19 male competitive cyclists from both measurement of whole body VO₂ and the RER during steady state cycling below blood lactate threshold, and during two-legged knee extension (an exercise that primarily recruits only the quadriceps muscles). Multiple biopsies of the vastus lateralis were obtained to
improve accuracy. Elder et al (1982) had examined the accuracy of predicting fibre type distributions and recommended sampling between three and five sites, depending on the muscle, to reduce the between-site standard deviation to 5%. The distribution of muscle fibres was determined as averages and were as follows: type 1 = 56% (3), type 2a = 41% (3) and type 2b = 3% (1). Almost all of the type 2 fibres in these subjects were type 2a, as would be expected in well-trained endurance athletes (Coyle et al, 1985). They reported a strong direct relationship between muscular efficiency (the ratio of the change in work accomplished per minute and the change in energy expended per minute) and fibre type (type 1) \(r = 0.85; p < 0.001\) and concluded that most of the variability in muscular efficiency in highly enduranced-trained cyclists is related to differences in percentage of type 1 muscle fibres. It was speculated that this greater efficiency of type 1 over type 2 fibres could be possibly as a result of a lower rate of ATP turnover as reflected by a lower VO\(_2\) while performing exercise at a given power output (Coyle et al, 1992).

Suzuki (1979) investigated whether delta efficiency (delta work/delta energy) during bicycling at the same workload was influenced by different distributions of ST and FT fibres. Efficiencies of 20 - 27% have been reported for bicycling at frequencies between 40 and 100 rpm, and some studies have illustrated that ST and FT fibres have different mechanical efficiencies. For example, according to Goldspink (1978) the most economical shortening velocity of ST fibres is at low velocities. Suzuki (1979) sampled from the vastus lateralis and compared three subjects who were found to have predominantly ST fibres (range of ST = 69 - 90%) with three subjects who classified FT (range of FT = 69-85%). This study concluded that delta efficiency during bicycling below 80% VO\(_{2\text{max}}\) at 60 rpm is unaffected by the difference in muscle fibre composition of the vastus lateralis, but at 100 rpm the efficiency of
the ST group was significantly lower than that of the FT group (20% vs 29%, p < 0.01). Suzuki (1979) presented a possible explanation linked to the longer cross-bridge engagement time of ST fibres as follows:

"If the velocity of shortening is so high that engaged bridges in the muscle filaments do not have sufficient time to disengage before new bridges form, they may offer resistance to continued shortening (Woledge, 1968). Furthermore, when ST fibres are utilised at a high shortening speed, the energy required to overcome this increased resistance should increase with greater work loads, since there would be a greater number of cross-bridges engaged".

Suzuki (1979) also speculated that an explanation for the results may be related to the differences in the glycogen utilisation of different muscle fibres during exercise.

Cavagna and Citterio (1974) have shown that prestretching active muscle causes the muscle to work more efficiently in the subsequent positive work phase and this potentiation has been attributed to the storage and re-use of elastic energy. The amount of elastic energy re-used has been shown to be a function of both muscle length and rate of prestretch (Bosco et al, 1981). Bosco (1982) suggested that the elastic behaviour of muscle is related to its mechanical and morphological structure. Therefore, it was thought that the muscle coupling time (eccentric-concentric transition period) and the stretching speed of a muscle may affect the re-use of elastic energy differently according to whether type 1 or type 2 fibres are activated (Bosco, 1982).
Bosco et al (1987) examined how running economy was related to different muscle fibre composition. A significant relationship between percent fast twitch fibres (vastus lateralis) and net oxygen uptake per unit distance travelled during submaximal running at a low speed (3.3 m.s\(^{-1}\)) was observed in 17 young adult, track and field, non-elite athletes. Coupling time during running at speeds similar to that used in this study have been found to be approximately 8 ms (Ito et al, 1983), which is probably too long a cross-bridge time in some of the type 2 fibres (Lannergren, 1976), and some of the cross-bridges in the type 2 fibres could have been detached. Thus, Bosco et al (1987) hypothesised that slow twitch fibres may have retained stored elastic energy longer without crossbridge detachment, reducing reliance on energy generated from oxidative phosphorylation as evidenced by the net energetic cost during running for the subjects with a lower proportion (33%) of type 2 fibres compared to those with a higher proportion (54%) of type 2 fibres.

These findings support the research of Kaneko et al (1983) who noted that the mechanical efficiency of distance runners was higher (72%) than that observed in sprinters (47%) when running slowly (3.7 m.s\(^{-1}\)), as distance runners have been shown to have a larger percentage of type 1 fibres than do participants in sprint events. They reported an association (r = -0.66; p < 0.01) between the net aerobic demand of running and the ratio of efficiency of muscular work performed during prestretch jumps compared with a no prestretch condition in 13 of the same 17 subjects, implying that data on the elastic behaviour of leg extensor muscles during jumping might have applicability to running. However, knee bends and jumping do not accurately simulate timing characteristics and muscle loading conditions peculiar to running. For example, the knee bends in this study were executed with knee angular displacements of
approximately 90 degrees, which is larger than one would observe during slow distance running.

2.2.19 Excess post-exercise $O_2$ consumption (EPOC)

There are two components involved in the total energy cost of physical activity: the energy expended during the activity itself, which accounts for the majority of the caloric expenditure; and the second component is the energy expenditure during the postexercise period (EPOC), when oxygen consumption declines exponentially whilst the metabolic rate remains elevated above the preexercise level (Gore and Withers, 1990). Numerous factors have been associated with the elevated postexercise metabolic rate, but the relative contribution of possible mechanisms for the prolonged EPOC component remains unknown (Roth, Stanley, and Brooks, 1988). In an attempt to further understand the mechanisms of rapid and prolonged EPOC components Bahr (1992) utilised a cycle ergometer and examined the effects of submaximal exercise duration (including to exhaustion) and intensity, and the effects of supramaximal exercise (including to exhaustion) on the magnitude and duration of EPOC, in 15 physically active young male subjects. Unlike the Gore and Withers study (1990) the subjects in this study were not endurance trained. They reported some significant findings from these well controlled series of experiments. For instance, after prolonged submaximal exercise (71-80 min at 69-78% $V_{O_2\text{max}}$) they observed a prolonged EPOC amounting to a mean increase of 14% oxygen consumption above the 12 hour control level. This would seem to be in agreement with other cycle ergometer studies that have reported a prolonged EPOC for 7.5 hours (Chad and Wenger, 1989), 12-16 hours (Devlin and Horton, 1986) and 24 hours
(Maehlum, Grandmontagen, Newsholme, and Sejersted, 1986) following 60 min exercise at 67% \( VO_{2\text{max}} \), 71 min of intermittent exercise at 85% \( VO_{2\text{max}} \) and 65-90 min of exercise at 60-87% \( VO_{2\text{max}} \), respectively. These findings highlight the important role of exercise intensity in the magnitude of EPOC and also demonstrates that sufficiently intense and prolonged exercise leads to an increase in oxygen consumption which outlasts the exercise period by many hours. Additionally, Bahr (1992) observed a linear increase in EPOC with increasing duration (20 to 80 min) at exercise intensities at or above 50% of \( VO_{2\text{max}} \) and an exponential increase in EPOC with increasing exercise intensity (26 to 78% \( VO_{2\text{max}} \)) in their untrained subjects, supporting the research findings on the separate effects of exercise intensity and duration on EPOC in trained subjects (Gore and Withers, 1990).

Other important findings during the 12 hour recovery period of Bahr's study (1992) are that the respiratory quotient fell rapidly and remained below control levels after exercise, plasma glucose was lower than controls, there was increased availability of FFA postexercise and increased levels of plasma glycerol. These findings indicate that during recovery from exercise, there is an increased availability of fat and a shift in substrate oxidation from carbohydrates towards fat, probably associated with an increased uptake of blood glucose for muscle glycogen repletion. This substrate shift could possibly explain 10-15% of the observed EPOC (Bahr, 1992).

2.2.20 Fibre type, epoc and overtraining

Overtraining can be described as an imbalance between training and recovery and is commonly observed in those athletes who train the hardest. The phenomena has been described by a multitude of descriptive terms including staleness, overreaching, overwork,
burnout, chronic fatigue and overstress (Verde, Thomas, and Shephard, 1992). If recovery time is extended there is always the concern that detraining might occur leading to the loss of temporary training effects (Coyle et al., 1985). However, when there are excessive increases in the general overload of training and the time for recovery and adaptation is insufficient, performance capabilities deteriorate and indications of overtraining become apparent (Verde et al., 1992). These become clearly visible as primarily physiological and/or generally as both physiological-psychological, and/or collectively as physiological-psychological-biomechanical in nature (Rushall and Pyke, 1990). For instance, it has been suggested that an increase in rate of perceived exertion (RPE) indicates a greater recruitment of type 2 muscle fibres with increased afferent activity from peripheral pain receptors to the reticular activating system reflected in subjective feelings of fatigue (Pandolf et al., 1975; Skinner et al., 1973). In addition it appears that the nature of the exercise affects recovery time (Kuipers and Keizer, 1988). It has been reported that the time it takes to recover from exercise with a substantial component of eccentric work, such as distance running, is slower than that following the predominantly concentric work experienced in cycling (Rushall and Pyke, 1990). Examples of physiological symptoms that have been used as an indication of overtraining would be increased resting heart rate, greater than 'normal' elevation in heart rate during submaximal exercise, and retarded return of heart rate after exercise (Kuipers and Keizer, 1988). Thus, it would seem that the principle of recovery should be considered to be of similar importance to the principle of overload when training for sports and fitness, mainly because an athlete's improvement is dependent upon the provision of adequate recovery so that training effects can be maximised (Rushall and Pyke, 1990).
It has been established that there is an increase in resting metabolism after exercise (Bahr, Opstad, Medbo, and Sjersted, 1991). Additionally, there is evidence that exercise metabolism is increased during submaximal exercise when recovering from consecutive days of prolonged intense exercise (Kirwan et al, 1988). The reasons for this reduction in mechanical efficiency and concomitant reduction in performance in overtrained athletes are not fully understood, but glycogen depletion with a shift in metabolism from glycogenolysis to lipolysis, [fat metabolism requires more oxygen per unit of energy expenditure than does carbohydrate metabolism (Costill et al, 1979)], changes in hormonal levels (Opstad, Falch, Oktedalen, Fonnum, and Wergeland, 1984), fibre type (Vollestad and Blom, 1985) and cellular metabolic efficiency (Pate et al, 1992) appear to be some of the factors involved. For instance, it has been speculated if the ATP requirements of the myosin ATPase were increased for a given force and speed of contraction, this would lead to a decrease in mechanical efficiency (Bahr et al, 1991). Thus, as force increases and more type 2 fibres are activated, it has been suggested that similar force output requires a higher rate of ATP hydrolysis in type 2 fibres compared to type 1 (Goldspink, 1978).
2.3 Biomechanical factors affecting running economy

Several studies have indicated that biomechanical factors contribute to the determination of economy of motion (Fedak, Hegland, and Taylor, 1982). For example, a considerable degree of intersubject variability for competitive distance runners during stepping, walking and running has been reported (i.e. 43.2 - 53.8 ml.kg\(^{-1}\) min\(^{-1}\) for 4.13 m.s\(^{-1}\) running at 0% grade (Daniels, Scardina, and Foley, 1984). In another study (Williams and Cavanagh, 1987) biomechanical variables were identified which showed differences or consistent trends between groups separated on the basis of VO\(_{2\text{submax}}\), establishing the importance of biomechanical influences on running economy. However, it has been suggested that no single variable or small subset of variables can explain differences in economy between individuals but rather that economy is related to a weighted sum of the influences of many variables (Williams and Cavanagh, 1987).

2.3.1 Body mass

Numerous 'animal' studies suggest the assumption that aerobic demand is independent of body mass once it has been normalised is not justified when large variations in body mass exist (Garland, 1983; Taylor, Heglund, and Maloy, 1982; Taylor, Schmidt-Nielson, and Raab, 1970). Adult humans have a more narrow range of body mass. Some studies (Davies and Thompson, 1979; Skinner, Hustler, Bergteinova, and Buskirk, 1973) have shown no difference in mass-specific aerobic demand for adults of different body mass (lean/obese/weighted individuals). Other studies suggest that with elite runners and non-elite diverse habitual runners, heavier individuals (male and female) had lower mass-specific demands (Williams and Kavanagh, 1986; Williams, Cavanagh, and Ziff, 1987; Pate, Macera, Bailey, Bartoli, and Powell, 1992). Bergh et al (1991) found that higher body mass is
associated with lower aerobic demand per kilogram of body mass. However the authors caution that body mass may correlate with a number of other variables, for example, body surface area and stride frequency, which may affect the energy demand of running and this is consistent with comments by Taylor et al (1980).

It has been suggested that the metabolic demands of generating muscle force are proportional to the rate at which actin-myosin cross-bridges cycle (Huxley, 1974). It is possible that larger individuals are more economical per unit of body mass than smaller individuals because it is speculated that smaller animals with higher movement cycle rates also have higher intrinsic muscle velocities (Fedak et al, 1982). Bergh et al (1991) noted that the endurance-trained runners in their study weighed less than the other groups. Hence, there might be a covariation between running skill and body mass which might obscure the effect of body mass. Nonetheless, their findings indicate that submaximal oxygen uptake during running does not increase proportionally to body mass. This is in agreement with results from studies in rowing (Secher, 1983), cross-country skiing (Bergh, 1987), and bicycling (Swain, Coast, Clifford, Milliken, and Stray-Gunderson, 1987). Additionally, Bergh et al (1991) reported that maximal oxygen uptake did not increase in proportion to body mass. Thus, they suggest that dividing oxygen uptake by body mass may induce erroneous interpretations when comparing individuals or groups which differ in body mass. Their data, combined with previous studies on humans (Astrand and Rodahl, 1986; Dobelin, 1956) indicate that suitable units in this context might be ml.min⁻¹kg⁻⁰·⁷⁵.

2.3.2 Body mass distribution

It has been hypothesised, based on fundamental mechanical principles associated with
accelerating and decelerating the limbs with each stride and assuming all other factors (e.g., speed, total body mass, running style) are reasonably similar, that an individual whose extremities present a smaller inertial load to the musculature for these accelerations should be more economical (Taylor, Shkolnik, Dmi’el, Baharav, and Borut, 1974). Many functional morphologists have found this hypothesis - that for a given body mass, speed and gait, the smaller and more proximally distributed a limb mass, the less kinetic energy required to accelerate/ decelerate the limbs - intuitively appealing (Gray, 1968). No direct support for the hypothesis has been found in terrestrial animals (Taylor et al, 1982). This may be due to intra-animal error in oxygen consumption data, making any differences more difficult to detect, or to differences in stride length/frequency between animals (Taylor, 1986). However, in humans it has been shown that a lighter individual tends to possess a greater percentage of body mass in the extremities (Williams and Cavanagh, 1987). Additionally, Myers and Stuedel (1985) have hypothesised that an individual possessing a relatively greater amount of body mass in the extremities would have to perform a relatively greater amount of work in movement of the limbs. In support of this view has been the criticism of assumptions associated with normalising VO₂ data to allow comparisons across individuals of different weights (Nevill et al, 1992) and the speculation that peripheral oxygen consumption may vary between individuals (Pate et al, 1992). Thus, scaling for the VO₂-to-body size relationship warrants further study (Rogers et al, 1995).

Some investigators have measured the increased metabolic cost of carrying additional loads on the back during running and reported that oxygen consumption increased in direct proportion to the mass carried (Cureton, Sparling, Evans, Johnson, Kong, and Purvis, 1978;
Taylor et al., 1980). Several studies have modified (artificially) segment inertia properties via the addition of mass to segments and appear to have demonstrated a clear effect of limb mass and mass distribution on the cost of locomotion (Carlin and Dressendorfer, 1979; Jones, Toner, Daniels, and Knapik, 1984; Martin, 1985). It seems that the increase in metabolic cost is more extreme as the weights are placed more distally on the leg and it has been suggested that it is metabolically about 6 times as expensive to carry a given mass on the feet or ankles as it is on the back (Frederick, 1987). Human studies of limb loads report that adding 100 g (50 g per leg) to the feet increased oxygen uptake by about 1% over a fairly wide range of running velocities (Myers and Steudal, 1985). Myers and Steudal (1985) tried to ensure a constant stride frequency by their subjects and found that

"the cost of adding a given mass to the limbs is significantly greater than adding it to the centre of mass and that this effect becomes more pronounced as the limb loads are moved distally".

However, in most of these studies, the artificial load was concentrated in a small area in a segment and are not representative of normal variations in segment mass distribution. Therefore, some caution is advised when interpreting results.

Russell and Belding (1946) and Mathews and Wooten (1963) found differences in VO\(_2\) during walking that could not be explained by weight alone and suggested that some of the variation in VO\(_2\) could be attributable to shoe style (hip boots/combat boots/high-heeled shoes). Adding inserts to running shoes appears to accompany increases in oxygen uptake and this oxygen uptake shift could be explained by the added weight (Berg and Sady, 1985;
Burkett, Kohort, and Buchbinder, 1985; Hayes, Smith and Santopeitro, 1983). Others (Bosco and Rusko, 1983) have compared conventional running shoes with soft-soled, nonresilient shoes (same weight) and found an increase in the oxygen demands of running while running in the soft and nonresilient shoes. It has been speculated (Snel, Delleman, Heerkens, and Ingen schenau, 1985) that with a more resilient shoe cushioning system it might be possible to improve the economy of running. Other authors have examined the influence of different cushioning characteristics of shoes and running surfaces and they have presented data that suggest this might alter the mechanical work performed and produce changes in the patterns of movement and muscle activity which might, in turn, affect the economy of movement (Bates, Osternig, Sawhill, and James, 1983; Clarke, Frederick, and Cooper, 1983; Nigg, 1986). Frederick et al (1982) demonstrated that systematically adding weight to the shoes of their subjects increased VO₂ at speeds of 3.83 - 4.9 m.s⁻¹. Additionally, Frederick et al (1986) compared a conventional running shoe with a softer but more compliant and resilient shoe and found that the oxygen requirement of treadmill running at moderate speeds (3.65 - 4.55 m.s⁻¹) was lower when wearing a softer-soled shoe. A coefficient of variability of 2.48% for within subject day-to-day variations in submaximal VO₂ for treadmill running at speeds close to the treadmill speeds used in Frederick et al's study (1986) has been reported by Armstrong and Costill (1985). However, it is noted that the validity of this day-to-day variation has been questioned (Frederick et al, 1986).

The adverse affects of ageing and lower extremity orthopedic pathologies on the aerobic demand of walking and running are well documented in adults (Auro and Komi, 1986; Pivarnik and Sherman, 1990). Martin and Morgan (1992) highlight the problem of declining musculoskeletal flexibility with the elderly population and speculate that there may be a link
between increased aerobic demand and reduced flexibility. They speculate that increased resistance to motion near the extremes of the range of motion and/or modified gait patterns, such as shorter stride length, could be the result of flexibility declines. There is a need for research on the potential links between flexibility and RE.

2.3.3 Running kinematics and economy

When oxygen consumption is expressed relative to distance travelled (ml.kg⁻¹.meter⁻¹) differences in the speed-economy relationship become apparent. A U-shaped speed-economy relationship is visible for walking with a minimum aerobic demand to walk a given distance (Inman, Ralston, and Todd, 1981; Pearce, Cunningham, Donner, Rechnitzer, Fullerton, and Howard, 1983). In contrast to walking, the energy cost to run a given distance is similar regardless of the speed. Research done over the past 30 years generally supports the concept of a linear relationship between running speed and aerobic demand (Kram and Taylor, 1990).

Stride length is defined as the distance between successive contacts of the same foot, whereas half a stride is called a step or two successive contacts of opposite feet. Some investigators have used the terms 'stride' and 'step' to mean the same thing, but even Shakspeare (Merchant of Venice) differentiated between both units:

"I'll..... turn two mincing steps into a manly stride..." (act 111, scene 1v).

The individual choice combination of SL/SF is made from a wide continuum of possible combinations and it has been reported that runners tend naturally to adopt a stride length
which is within 4.2cm of the stride length at which energy cost is minimised (Cavanagh and Williams, 1982). The choice combination, usually subconscious, may be influenced by a number of factors including: anthropometric dimensions (Van Der Walt and Wyndham, 1972), surface properties (McMahon and Greene, P.R., 1979), velocity (Nilsson, Thorstensson, and Halbertsma, 1985), grade (Davies, Sargeant, and Smith, 1974), footwear (Clarke, Frederick, and Cooper, 1983), developmental status (Amano, Hoshikawa, Toyoshima, and Matsui, 1987), longitudinal influences (Nelson and Gregor, 1976), state of fatigue (Elliot and Roberts, 1980) and muscle fibre composition (Armstrong, Costill, and Gehlsen, 1984). For example, several investigators (Kram et al, 1985; Siler and Martin, 1991; Williams et al, 1991) found that fatigued runners tend to adopt longer stride lengths for a given running speed suggesting that the naturally chosen stride lengths may be influenced by changes in muscle function brought on by fatigue.

SL has been shown to affect the economy of distance running (Cavanagh and Williams, 1982) and it has been suggested that the preferred selection may be related to minimising metabolic energy cost (Keneko, Matsumoto, Ito and Fuchimoto, 1987; Plyley, Tidus, and Pierrynowski, 1985). Also, it is interesting to note that although it is generally thought that taller individuals inevitably take longer strides at a given velocity of running, there is little evidence to support this idea, and SL is not nearly as well correlated with either stature or leg length as is generally believed (Elliot and Blanksby, 1979; Frederick, Robinson, and Hamil, 1987). Additionally, there is evidence that factors other than limb segment mass or inertia are the primary determinants of SL (Cavanagh and Kram, 1989). Interestingly, Cavanagh and Kram (1989) suggested that:
"Individuals who appear externally to be physically identical may have internal differences in, for example, their muscle fibre populations, recruitment patterns, or mechanical advantage at important muscle insertions, as well as more subtle differences."

It has been established that humans increase both SL (curvilinearly) and SF (more at higher velocities) as running velocity increases (Dillman, 1975). The SL reaches a plateau at velocities which are much greater than those encountered in distance running (Cavanagh and Kram, 1989). It has been suggested that a SF of 1.7 to 1.8 hertz may represent the maximum stride rate achievable at any speed (Nilsson and Thorstensson, 1985) and that there may be a characteristic preferred 'narrow range' of SF to run economically at distance running speeds (2.5 - 6.0 m.s⁻¹).

The better runner has been reported to have less vertical oscillation (Gregor and Kirkendall, 1978), longer strides and greater lifting of the thigh (Hoshikawa, Miyashita, and Matsui, 1971), less time in support and more time in flight (Kurakin, 1972) and less change in velocity during contact with the ground (Kaneko, Fuchimoto, Ito and Toyooka, 1983). Nicol et al (1991) and Williams and Cavanagh (1986) have shown longer support times to be associated with lower (worse) economy. However, others Williams and Cavanagh (1987) have reported no significant differences between elite and good distance runners in angular kinematics, vertical oscillation and step length. Also, Cavanagh and Williams (1982) found no clear relationships between step length, leg length, and VO₂max when 10 well-trained runners ran at freely chosen step lengths.
Numerous studies have consistently demonstrated a U-shaped stride length/rate-economy response for the aerobic demand of walking and running. That is, there are individual optimal stride length/rate combinations at which aerobic demand is minimised (Heinert, Serfass, and Stull, 1988; Morgan and Martin, 1986; Powers and Ragsdale, 1982). It is possible that the mechanisms underlying the economy response may be associated with muscle force and power generating capabilities. Hill (1922) demonstrated that muscle efficiency varies with shortening velocity such that a most efficient velocity exists. Changes in stride length and rate require concomitant changes in the rates of muscle lengthening and shortening and rate of force development, which ultimately affect aerobic demand (Heglund, Fedak and Taylor, 1982; Taylor, 1985). In running at a given speed an optimum stride length to minimise oxygen uptake has been reported (Cavanagh & Williams, 1982; Hogberg, 1952). Kaneko et al (1987) examined whether or not the most efficient step frequency would exist in level running performed at a given speed by measuring not only the energy cost but also the mechanical power and mechanical efficiency under various combinations of step frequency and stride length. They used a force platform implanted in an outdoor running track to determine the external power of the body due to the kinetic and potential energy changes of the centre of mass. The internal mechanical power to accelerate the limbs relative to the centre of mass of the body was determined by a cinematographic technique. The total power output was taken as the sum of external power and internal mechanical power as in Fenn (1930). Oxygen uptake values were measured during steady state treadmill running using the same running speeds as in the track experiment. Thus, mechanical efficiency was calculated as total power output divided by net energy expenditure. Kaneko et al (1987) found when power increases more energy is required, and the mechanical efficiency calculated was not constant. At the extreme conditions of low and high stride rates, external mechanical power
and internal mechanical power were high respectively. This implies that even at a given speed
the efficiency varies with the working conditions, namely step frequency and stride length
and they speculated that one of the underlying factors may exist in the recruitment of
different types of muscle fibre as reported by Komi (1979). Goldspink (1977) reported a
lower efficiency of fast-twitch muscle fibre compared to slow-twitch fibre suggesting that
fibre recruitment might play a role in determining aerobic demand. In support of this Katz et
al (1986) concluded that a major determinant of the ATP turnover rate is the muscle fibre
composition (a higher turnover rate in fast-twitch fibres). Interestingly, Kaneko et al (1987)
found that the most efficient step frequency was virtually consistent with that in natural
running. This implies that the human machinery can automatically select an optimum step
frequency so that it can work more efficiently. Holt et al (1990) conducted a study to
determine whether the preferred frequency of locomotion (walking) was predictable as:

"the least amount of energy required to drive a harmonic oscillator."

Previous research findings on walking (Wilke, 1970; Fisher and Gullickson, 1978; Workman
and Armstrong, 1986) appear to support the notion that humans may be self-optimising
machines such that bipedal gait may have developed kinetically and structurally to become a
metabolically and mechanically efficient system of transportation. Thus, humans may
develop a 'freely' chosen, preferred stride frequency that is directly associated with
anthropometric and inertial characteristics of the legs (Zarrugh and Radcliffe, 1978). This
self-selection stride length/rate seems to occur at a point on the U-shaped stride length/rate
speed-economy curve where the metabolic cost (oxygen consumption) is minimal. Modelling
locomotion as a pendicular activity has been used in human gait, as walking fulfills the definition of an oscillator:

"rhythmic with temporal consistency" (Winter, 1980).

Holt et al (1990) stated that in a 'force-driven harmonic oscillator' there is a particular frequency, the resonant frequency, which requires the minimum force to maintain its oscillations and overcome the diminishing forces of, for example, gravity. If one considers walking as the biological equivalent of a force-driven harmonic oscillator, resonance may be the manner in which muscular effort is minimised, resulting in a decreased need for oxygen. However, although the idea that human behaviours can be considered as oscillatory processes is attractive, an element of caution is necessary in interpreting the findings of the study by Holte et al (1990). The lower limb should not be considered a rigid lever throughout the walking phase and the protocol of the study could be described as ecologically invalid (i.e the wearing of weights on one's ankle is neither a normal event nor is it a well learned skill).

Also, whilst the force-driven harmonic oscillator was modelled for movement in one plane only, the lower limb moves in a near sagittal plane. However, the suggestion that self-optimisation, in the form of minimal metabolic cost, underlies locomotion systems is very interesting. In particular, the notion that motor control parameters emerge from the physical attributes of the system is exciting. Workman and Armstrong (1986) proposed that the metabolic cost of walking may most conveniently be held to consist of three compartments:

1. Basal metabolic rate;
2. Other metabolic costs of walking, for example, the oxygen cost of breathing/the work of the heart;
3. The metabolic cost of the walking movement, which is exponentially related to ground speed.

It is debatable whether height is a significant determinant of metabolic cost of walking (Jankowski, Ferguson, Langelier, Chaniotis, and Choquette, 1972). Workman & Armstrong (1986) found that body height has a small but consistent effect on the predicted value of walking oxygen uptake. According to their predictive equations short people use more oxygen than do tall people of the same body weight at all speeds of walking. They question whether "efficient" walking and "comfortable speed of walking" are definable in terms of least metabolic cost per unit of distance travelled. Pearce et al (1983) suggested that comfortable rates of walking may relate to problems of maintaining balance. It is surmised that it is the need to maintain balance while walking that determines step frequency (Zarrugh and Radcliffe, 1978). This appears to lend some support to the above mentioned 'motor control parameters' notion and it would be interesting to examine whether there is a transfer to running, as one might expect.

It is reasonable to postulate that in studies of running economy where the inertial characteristics were artificially varied, one of the factors which might affect locomotor energetics was not held constant - the problem of maintaining balance. Previous research has established that the movement pattern exhibited by the upper body while running and the motion of the lower body are inextricably linked (Cappoza, 1981; Jakush, 1982). Studies on distance runners suggest that the arm swing is crucial to the transfer of angular momentum between upper and lower body about the vertical axis (Hinrichs, 1987). Case studies of asymmetrical arm action in running suggest that: "The arms must generate enough vertical
angular momentum in their combined forward and backward swings, along with the twisting back and forth of the upper trunk, to offset an approximately equal and opposite vertical angular momentum in the lower trunk and legs" (Hinrichs 1992). The implications of any artificial interference with the vertical angular momentum component are obvious and further adds to the 'motor control parameters' notion. Additionally, Myers and Steudel (1985) expressed reservations about their experimental design when pointing out that in keeping stride frequency artificially constant, any findings will have excluded the possible synergistic effects of gait and limb morphology on locomotor energetics.

A limited number of studies in the past have examined the relationship between mechanical and metabolic variables in runners (Luhtanen and Komi, 1978; Shorten, Wooton, and Williams, 1981). The study by Williams and Cavanagh (1987) provided the first substantial support for a relationship, showing that more economical runners tend to have identifiable patterns in their running mechanics. The biomechanical variables that were found to have significant differences included for example, trunk angle, with the more economical runners having a more pronounced forward lean (p < 0.05). However, although other variables highlighted differences, such as the more economical runners having less vertical oscillation, the differences were non-significant. Also, there were individual exceptions to the general trends found. For example, there were individuals who had low VO_{2submax} values and very little forward lean, and ones who had high VO_{2submax} values and a great deal of lean. These individual exceptions suggest that there are a number of mechanical factors that have potential influence on metabolic energy cost, with no one factor being of critical importance and:
"each individual might adopt specific movement patterns that are best suited to his or her own anatomical and physiological constraints" (Williams and Cavanagh, 1987).

### 2.3.4 Kinetic factors

Little attention has been given to the potential link between ground reaction force (GRF) descriptors and gait economy. Studies involving elite and recreational runners report moderate to weak relationships between GRF and economy (Williams and Cavanagh, 1986 & 1987). An interesting finding was that the more economical runners tended to have more of a rearfoot striking pattern, which may affect economy because of the need to provide cushioning during early ground contact. Thus, there may be an increased demand placed on the support phase muscles by individuals who strike the ground more forward on the foot as they:

"may have to rely more heavily on the musculature to assist with cushioning than rearfoot strikers who perhaps can rely more on footwear and skeletal structures to cushion and support the contact force" (Williams and Cavanagh, 1987).

Williams and Cavanagh (1987) found that the vertical component of the GRF as a function of time revealed that the first peak (Fz1) was significantly smaller in the low VO$_{2_{submax}}$ group than in the high VO$_{2_{submax}}$ group of subjects. This is interesting when one considers the paper by Dick and Cavanagh (1987). The purpose of their study was to quantify and propose an explanation for upward drift in oxygen uptake (UDO) during an activity involving eccentric muscle action - downhill running (3.83 m.s$^{-1}$). Dick and Cavanagh (1987) found significant increases in oxygen consumption (10%) during prolonged downhill running. They
hypothesised that UDO and increasing integrated electromyography during downhill running reflected increased motor unit recruitment within the eccentrically acting muscles, caused by an ongoing combination of muscle damage, connective tissue damage, and local muscle fatigue. There could be a link here when one considers that the phase from first peak (Fz1) to knee flexion involves eccentric contraction of the quadriceps, and Williams and Cavanagh found that those with a low VO$_{2\text{submax}}$ had a smaller force to control eccentrically (initial peak to knee flexion) when running at 3.57 m.s$^{-1}$.

Mechanical power reflects an indirect way of expressing muscular effort, if one assumes that a considerable portion of the metabolic demands of gait (walking/running) is attributed to muscles actively contracting concentrically or eccentrically, and should hence be a useful predictor of running economy. As both aerobic demand and mechanical power output are both speed dependent it is not surprising that Burdett et al (1983) and Shorten et al (1981) reported correlations exceeding 0.79 between both, for walking and running. This does not help explain whether interindividual differences in oxygen consumption for identical running speeds can be accounted for by mechanical power descriptors and there is disagreement amongst researchers, with some reporting several power-related descriptors (Dick and Cavanagh, 1987) and others observing that mechanical power does not explain economy variations among animals with wide variations in body size (Heglund, Cavagna, and Taylor, 1982). It should be noted that an accurate method for computing mechanical power is still elusive, due to issues such as storage and recovery of strain energy, the inability of computations to account completely for isometric contributions of muscles (Heise and Martin, 1990; Martin, Heise, and Morgan, 1991) and the differing energy costs of concentric and eccentric muscle actions during gait (Ingen Schenau and Cavanagh, 1990).
Many studies on calculating mechanical muscle power have inclined to disregard power dissipated to the environment, because this was considered such a small fraction of power output. However, wind tunnel (Kyle, 1979) and foot and shoe deformation research involving a calorimetric study where mechanical muscle power was compared with heat production (Webb, Saris, Schoffelen, Ingen Schenau, and Hoor, 1988) suggest that this component can be at least 10% of power output and, therefore, should not be ignored. The problems associated with these methods for calculating mechanical power have been comprehensively discussed before (Williams, 1985). Other approaches in running have used the product of the propulsive force and the velocity of the center of mass of the system but validity is questionable since the velocity of the point of application of the propelling force is not equal to the velocity of the center of mass. In running the velocity of the point of application of this force (foot on ground) is zero (Fukunaga, Matsuo, and Ichikawa, 1981). Ingen Schenau and Cavanagh (1990) attempted to clarify the formulation of power equations, applicable to endurance activities such as running, based on the application of conventional Newtonian mechanics, to a rigid segment model of the body. They too encountered problems, for example, in trying to assign positive or negative joint power to the actions of muscles which cross a joint:

"the bi-articular gastrocnemius can oppose the knee extensors in such a way that no, or even negative, power is generated in the knee joint, since the net moment is zero (or negative). In reality, the knee extensors may lift the calcaneus via the gastrocnemius muscle (Bobbert, Huijing and Ingen Schenau, 1986). In other words, the joint power
that appears computationally to have its source at the ankle can, to a large extent, be liberated in the knee muscles" (Aleshinsky, 1986).

It seems that the problem of power output calculation in running has not been entirely solved so far and no definitive relationships are obvious between running economy and mechanical power from the available data (Williams and Cavanagh, 1983).

Elastic properties of muscle and running economy may be important but are difficult to assess during running (Luhtanen amd Komi, 1978). Nigg and Anton (1995) suggested that although:

"total mechanical energy content in systems composed exclusively of masses and springs remains constant over time",

this may not be the case for shoe-heel material (resonance/timing effect) and the recoil characteristics of muscles. Siler and Martin (1991) and Nicol et al (1991) have suggested that a loss in the recoil characteristics of muscles is associated with significant changes in metabolic data, reflecting deteriorating RE following prolonged running.

2.4 Psychological state and running economy

Although there is a sparsity of studies on the influence of psychological state and running economy (Crews, 1992), there is some evidence that factors of a psychogenic nature, such as an individual's thoughts (cognitions), feelings (affect) and sensations (perception) can influence resting and exercise metabolism, either independently or in concert (Morgan, 1983a; Morgan, 1983b; Suess, Alexander, Smith, Sweeney, and Marion, 1980).
Investigations involving hypnotic suggestions of work, excitations or hypnotically perceived graded exercise ('light' - 'heavy') have reported increases of oxygen uptake in the range of 92 - 409 ml.min\(^{-1}\), suggesting that metabolic responses can be influenced not only by perception, but also by perceptions of intensity. (Fulde, 1937; Levin and Egolinski, 1936; Nemtzova and Shatenstein, 1936).

The cardiovascular effects of hypnotically suggested exercise have been compared with graded treadmill walking (5% at 3mph), and imagined exercise produced an increase in heart rate that was 57% of that following actual exercise (Berman, Simonson, and Heron, 1954). In a similar vein, subjects under hypnosis have been instructed to relive a specific exercise in which they had actively participated (Dudlye, Holmes, Martin, and Ripley, 1964). One of Dudlye et al's (1964) findings was that oxygen uptake increased by 106 ml.min\(^{-1}\) following suggested exercise. This supported earlier findings (Whitehorn, Lundholm, and Gardiner, 1930) and was corroborated a year later by Agosti and Camerota (1965), who reported that respiratory frequency during hypnotically suggested imagined exercise was greater than a hypnotically suggested relaxation condition (p < 0.01). There has been a variety of similar research suggesting that metabolic responses are elevated above the expected physiological cost under 'resting' conditions such as hypnotic suggestion (Cobb, Ripley and Jones, 1967; Bevegard, Arvidsson, Astrom, and Jonsson, 1968; Adlercreutz, Kuoppasalmi, Narvanen, Kosunen, and Heikkinen, 1982). However, caution is advised here as during hypnosis the individual is passive rather than active in controlling cognitions and/or emotions, and this may affect physiological responses during exercise (Crews, 1992).
There have been studies that incorporated similar methods for data collection on the influence of one's perception of the exercise cost on exercise metabolism, but whose findings are in disagreement (Morgan, 1970; Nemtzova and Shatenstein, 1936). Some studies have involved subjects exercising on a bicycle ergometer for either 5 minutes or 20 minutes at constant load of 100 watts with results indicating higher ventilatory minute volumes associated with hypnotic suggestions of heavy exercise (Morgan, Raven, Drinkwater, and Horvath, 1973; Morgan, Hirota, Weitz, and Balke, 1976). This has particular relevance to populations who are required to exercise in an extreme environment, as highlighted by Morgan (1985):

"absolute exercise in an extreme environment (e.g., higher temperature and humidity) would have a greater metabolic cost for all individuals, and the added cost for some 'types' (Morgan, 1983a & b) of individuals could be substantial".

Benson et al (Benson, Dryer, and Hartley, 1978) have reported decreases in oxygen consumption (4% reduction: p < 0.05) when subjects employ relaxation techniques to reduce anxiety while exercising at a fixed submaximal work intensity on a bicycle ergometer. However, it should be noted that the workload was very light as reflected in the mean reported heart rate during exercise (98 b.min⁻¹). These findings were not supported by Cadarette et al (1982) who failed to observe a decrease in oxygen consumption during similar exercise when practising the same relaxation response, although respiration rate, tidal volume, and ventilatory minute volume were reduced. Others have utilised relaxation strategies whilst subjects undertook an exhaustive endurance walking task (80% VO₂max) on a treadmill and found no differences in oxygen uptake, but their findings suggest that the
relaxation response enabled the experimental subjects to tolerate a greater amount of discomfort for a longer period of time (Morgan, Horstman, Cymerman, and Stokes, 1983). Elite distance runners have been observed to employ a different mental strategy compared to less capable runners, and the elite distance runners were observed to use less ($p < 0.002$) oxygen during treadmill running at speeds of $4.44 \text{ m.s}^{-1}$ than did elite middle-distance or non-elite college runners (Morgan and Pollack, 1977). The elite distance runners used more associative techniques compared to the other two groups, whilst the nonelite runners used more dissociative strategies. Thus, the elite distance runners in this study tended to consciously monitor their physiological efforts during training and competition while the term dissociation, in this context, implies engaging in various forms of distraction. Hatfield et al (1992) suggested the possibility of an association analogue (i.e. feedback) in the promotion of physiological efficiency when stating:

"It may be that the adoption of association promotes the restraint of any exaggerated physiological response to work stimuli, and this would seem particularly important to the performance of athletes engaged in endurance events for which economy of effort is important".

Interestingly, later studies involving non-elite subjects walking (Morgan et al, 1983), running (Okwumabua, Meyers, Schleser, and Cooke, 1983; Fillingham and Fine, 1986) or performing an unfamiliar endurance task (Weinberg, Jackson, and Gould, 1984) suggested that whilst experienced subjects benefit from associative strategies, inexperienced subjects benefit from dissociative techniques. If specific cognitive strategies (e.g. coping) are more effective in reducing the oxygen cost of exercise then there could be positive practical consequences, as
the average exerciser would probably be able to learn and implement a cognitive strategy
easier than learning hypnosis or having access to a hypnotist for each exercise session
(Crews, 1992). Many of the papers referred to discuss how cognition and perception at rest
and during exercise can influence heart rate, cardiac output, blood flow, ventilatory minute
volume, and oxygen consumption. The available evidence is indicative

"that variables of a psychogenic nature influence resting and exercise metabolism, and
efficiency of movement is dependent in part on cognition (thoughts), perception
(sensations), and affect (feelings)" (Morgan, 1985).

Ketai (1975) suggested that there may be differences between mood and affect in relation to
duration and intensity, and Crews (1992) made a distinction between mood and affect as
follows:

"Mood represents feelings of a longer duration and lower intensity, while affect refers to
shorter duration and higher intensity emotions".

Williams et al (1991) examined the relationship between certain mood states and within-
subject variation in running economy in moderately trained runners (VO$_{2\text{max}}$ range: 55 - 66
ml.kg$^{-1}$ min$^{-1}$; 10 km race time range: 38-45 min). Williams et al (1991) determined oxygen
consumption values five days from each week during each of three running paces (2.68, 3.13
and 3.58 m.s$^{-1}$; corresponding approximately to 50, 60 and 70% of VO$_{2\text{max}}$) and recorded a
weekly profile of mood states (POMS) scores over a four week period. The POMS is a
psychological inventory used to measure six affective states and each subject's POMS total
mood disturbance (TMD) score was determined by adding the scores from tension, depression, anger, fatigue, and confusion subscales, subtracting the vigor score and adding a constant of 100 to avoid negative numbers (McNair, Lorr, and Dropleman, 1971). The higher the TMD score, the worse the overall mood state. The POMS has been reported as the best single marker of disturbed function, indicating increased fatigue and decreased vigour in overtrained distance runners (Cockerill, Nevill, and Byrne, 1992). However, the one disadvantage of POMS is that the intent of the questions is fairly obvious to the respondent.

The within-subject group correlation between TMD scores and running economy was $r = 0.88 \ (p < 0.01)$, indicating that the more economical running economy values were associated with more positive mental health profiles. Also, the subscale illustrating the strongest relationship with running economy was tension ($r = 0.81, \ p < 0.01$). This supports the finding of Ziegler et al (1982), who demonstrated that reduced stress and reduced tension also reduce the oxygen cost of exercise at a given workload (50% $\text{VO}_{2\text{max}}$). However, Ziegler et al (1982) failed to explain how oxygen consumption was measured. However, the following were acknowledged by Williams et al (1991) and should be noted:

1. Most of their subjects distance events are run at paces above 85% $\text{VO}_{2\text{max}}$;

2. Day-to-day variations of other factors (e.g. water retention, diet, running mechanics) could be confounding variables;

3. Resting VO₂ values (baseline correction) were not obtained so it is not possible to determine whether changes in economy were due to an effect occurring during rest, exercise, or some combination of both.
Additionally, although there does appear to be a relationship between the influence of affect (a more immediate change in emotion) on rest and on the physiological response to exercise (Kavanagh and Hausfield, 1986), we cannot assume that more positive emotion produces greater economy,

"since both positive and negative emotions alter physiological responses to exercise" (Crews, 1992).

However, a more stable measure of emotion (mood) could improve economy, as long as the mood is positive rather than negative.

There have been numerous studies indicating that physiological responses can be increased or decreased during exercise using biofeedback (Fredrikson and Engel, 1985; Hatfield, Spalding, Mahon, Brody, and Vaccaro, 1986; Talen and Engel, 1986), but no resultant change in running economy has been reported. Bhambhani and Singh (1985) were of the opinion that the ventilatory equivalent for oxygen (\(V_{\text{E}}/\text{VO}_2\)) is an index of submaximal ventilatory efficiency. For this reason Hatfield et al (1992) designed a study where the ventilatory equivalent of oxygen was chosen as one target response of feedback control, since individuals can exert voluntary control upon \(V_{\text{E}}\) (Dempsey, Pellegrino, Aggarwal, and Olson, 1979). Hatfield et al (1992) found that competitive cross-country runners could use behavioural intervention during steady state treadmill running (at just below ventilatory threshold - 71% \(\text{VO}_{2\text{max}}\)) to exert a measurable reduction in ventilation. It is suggested that their subjects were able to maintain a similar oxygen consumption level while breathing a reduced volume of air
during 'feedback' conditions (digital display of $V_e$). They report that this appears to be achieved by greater extraction of the available oxygen in the inspired air on a per breath basis, as shown by the reduced value of the pressure of end-tidal oxygen upon expiration during feedback, whilst 'dumping' more carbon dioxide per breath, as indicated by the elevated pressure of end-tidal carbon dioxide. Thus, although a decreased oxygen consumption while maintaining absolute workload was not observed, the respiratory cost ($V_J$) of delivering the oxygen to the tissues did. This study supports the notion that an associative strategy may play a role in ventilatory efficiency.

A recent study by Martin et al (1995) examined private self-consciousness (PSC) which can be described as a measure of the degree to which people habitually direct attention inwards. Apparently individuals who score high on PSC are able to self-regulate their behaviour more effectively. For example, be more sensitive to muscle tension and subsequently relax, with reduced muscle activity resulting in reduced $O_2$ demand and greater economy. They found that PSC and RE were linked suggesting that runners who habitually direct attention inwards were also the most economical. Future research should attempt to substantiate these findings.
2.5 Conclusion

The physiological, biomechanical and psychological factors that may influence running economy, and the relationship between running economy and performance, have been extensively studied. Although several variables have been identified as determinants of running economy, a comprehensive model for running economy has yet to be established. In conclusion, this review highlights that, with regards to the economy of human movement, there is a hiatus, and further research appears justified. The following studies have attempted to further clarify our understanding of running economy from a physiological perspective, whilst recognising the inherent complexity of the interdisciplinary processes of running economy.
General Methods

The specific procedures followed in each study are briefly described in the experimental chapters, and common methods are reported below.

All subjects were fully informed of the nature and purpose of the study before giving formal consent (Appendix A) and were aware that they could withdraw from the study at any time without disclosing their reasons. The study was conducted in accordance with the approval and guidelines laid down by the University's Ethical Advisory Committee (School of Physical Education, Nanyang Technological University, Singapore). Subjects with a medical condition potentiating an undue personal risk were excluded.

3.1 Apparatus and instrumentation

Studies were performed on a motorised treadmill (Quinton Q65 series 90). The treadmill speed range and elevation range (range: 0 - 25%) fulfilled the requirements of all experimental protocols. However, it is recognised that there may be some differences between treadmill and overground running, especially during prolonged exercise (Nigg et al., 1995). For example, more vertical displacement during overground running has been recorded when compared with treadmill running (Pink et al., 1994).

Before commencing and during each study the treadmill speed calibration was validated (range: 2.4 - 24 km.h⁻¹) and the reliability of the speedometer confirmed. The treadmill belt length was measured and time recorded for the belt to complete fifty revolutions at various speeds spanning the experimental range, both without and with subjects who covered a wide
range of body mass ('light' - 'heavy'). The total distance covered by the belt during each set of fifty revolutions was determined and the actual treadmill speed calculated. No adjustment was necessary to achieve the intended speeds. The treadmill was linked to a microcomputer (ACER 910) and a printer (NEC Pinwriter P3300)). Using software (Sensormedics), performance data from the treadmill was continuously monitored and recorded.

3.2 Subjects and laboratory procedures

The subjects were male students from the School of Physical Education in Singapore. The three races (Chinese, Malay, Indian) were represented. They were all active, competing sportsmen and some of them were endurance athletes. The same subjects were employed for all of the studies.

3.2.1 Height and mass

Height was measured using a wall mounted stadiometer with a maximum range of 200 cm and accurate to ±0.01 cm (Harpenden Stadiometer). Subjects were weighed on each visit to the laboratory using balance scales (Detecto Ltd) with a capacity of 160 kg and accurate to ±0.05 kg. Before each preliminary test subjects were weighed in light-weight running attire (NIKE Ltd) and bare feet. Nude body mass was measured before and after each long run, subjects having been previously towelled down to remove all surface moisture.

3.2.2 Heart rate

Heart rate (HR) was recorded using a heart rate monitor via short-range telemetry (Sport Tester Polar Vantage XL) throughout the preliminary tests and experimental trials. Similar telemetric systems have been verified valid (Karvonen et al, 1984).
3.2.3 Expired air collection and analysis

Expired air was collected and analysed for oxygen and carbon dioxide content using a Sensormedics 2900 Metabolic Cart (Zirconium oxygen analyser/ infrared carbon analyser - NDIR). Ventilation, oxygen consumption, carbon dioxide production and respiratory exchange ratios were calculated as 6 consecutive 20-second averages.

Subjects were presented with a noseclip (Harvard Equipment) and mouthpiece (Hans Rudolph Inc) at least 60 seconds before each collection was due to be taken. This ensured evacuation of 'dead-space' with expiratory air. The mouthpiece communicated with the metabolic cart via a lightweight two-way non-rebreathing valve (Hans Rudolph Inc) and a 1.5 m length of wide-bore (30 mm) lightweight tubing (Sensormedics Equipment). Thus, a closed circuit was formed when the nose-clip and mouthpiece were correctly worn, allowing expired air to be collected over a measured time interval.

Both analysers were calibrated with gases of known concentrations prior to and during each test session. Flowmetre calibration was performed prior to each test using a 3-litre calibrated syringe (Sensormedics - D).

Respiratory values were standardised for temperature, atmospheric pressure and water vapour content using software developed by Sensormedics Ltd.

3.2.4 Measurement of body temperature

Rectal temperature ($T_{re}$) was measured with a rectal thermistor probe (YSI Re-usable Temperature Probe) inserted 10 cm beyond the external anal sphincter muscle (Nielsen and
Nielsen, 1962). A rectal probe was used as this is considered to be a sound representative indicator of changes in deep body temperature (Nadel and Horvarth, 1970). Thus, core temperature was estimated from rectal temperature (Gisolfi and Wenger, 1984).

3.2.5 Subjective ratings of exertion
Perceptions of fatigue during the preliminary tests and the experimental trials were measured using the fourteen point Borg Scale (Borg, 1973). The Borg Scale is a linear rating scale graded from 6 to 20, where 7 is anchored by the expression ‘Very, very light’ and 19 is anchored by the expression ‘Very, very hard’. Responses to the Borg Scale have been shown to correlate with relative exercise intensity and heart rate (Borg and Noble, 1974).

3.2.6 Environmental conditions
Ambient conditions in the laboratory were carefully monitored during all experimental trials. The laboratory was well ventilated and air conditioned. Ambient temperature (Stockburger thermometer) and humidity levels (Brannan Whirling Hygrometer) were recorded before and during every data collection in close proximity to the treadmill. During all preliminary and experimental testing the laboratory was maintained at 22 to 23°C within a relative humidity range of 59 to 72%. Barometric pressure was measured using a wall mounted barometer (Griffen and George).
3.3 Preliminary testing

Subjects were initially familiarised in the laboratory for 3 to 4 weeks. During this time they were introduced to running on a motorised treadmill, the laboratory setting and the experimental protocols. Prior to experimental trials, preliminary tests were completed to determine the oxygen cost of submaximal running and the maximal oxygen capacity.

3.3.1 Familiarisation

All subjects completed a minimum of four 15-min treadmill familiarisation runs, as recommended by previous research (Schieb, 1986). This was in the form of entry onto a moving treadmill, progression from a walk extending into a jog, and eventually into a run, including both level and inclined treadmill running, without and with a mouthpiece. Stopping procedures and signal systems when the subjects had a mouthpiece were explained and practised such that the subjects felt confident, safe and relaxed.

Subjects were instructed in the standard laboratory methods for sampling expired air, monitoring heart rate and collecting fingertip blood samples. Also, they were introduced to the Borg Scale of perceived exertion (Borg 1973, cited in Borg and Noble 1974).

3.3.2 Establishing a speed-VO\textsubscript{2} relationship

The first performance test determined the oxygen cost of running over a range of submaximal speeds (speed-VO\textsubscript{2} test). The speeds were selected with reference to each subject’s running ability and lay between 60 and 90% VO\textsubscript{2}\textsubscript{max}. Actual speeds ranged from 2.22 to 4.44 m.s\textsuperscript{-1}. This was a continuous test with subjects running for 4 min at four different speeds. Expired
air collections were made over the last min of each stage (ie. 3-4, 7-8, 11-12, and 15-16 min) and analysed as described previously. Percentage oxygen and carbon dioxide concentrations were measured in each sample as was the total volume of expired air collected. Thus, rates of oxygen consumption (VO$_2$) and carbon dioxide production (VCO$_2$) were determined using the Haldane transformation, and the minute ventilation volume (VE) was calculated. Responses to the Borg (1973) scale were recorded during each collection. By applying linear regression to the four co-ordinate values of VO$_2$ and running speed, individual relationships were established for each subject.

3.3.3 Determination of maximal oxygen uptake (VO$_{2\max}$)

Maximal oxygen uptake was determined during continuous, incremental-grade uphill running. The method used was a modification of the Taylor treadmill test (Taylor et al, 1955). The treadmill was initially set at a grade of 3.5%, and was subsequently increased by 2.5% every 3 min. Subjects ran at a constant submaximal speed throughout the test. Subjects aimed to run for as long as possible. An expired air collection was taken when subjects felt that they could only maintain the required exercise intensity for one more min. From this collection VO$_{2\max}$ was calculated. Criteria establishing a valid VO$_{2\max}$ test include: an R value greater than 1.15; plateauing of VO$_2$ and VE values; and a heart rate which approximated a predicted maximal value (Astrand and Rodahl, 1986).

3.3.4 Running economy (RE)

The RE was determined during 10 min running bouts (Rusko et al, 1986), with expired air analysed and VO$_2$ quantified over the last 2 min of each 10 min running bout.
3.4 Standardised test procedure

Subjects were asked to maintain their normal diets and activity levels prior to and during testing, but were requested to refrain from exercise for 24 hours pre-testing and report to the laboratory at 7 am after an overnight fast. The influence of circadian variation (Hill and Moore, 1994) and footwear (Martin, 1985; Frederick et al, 1986; Morgan and Craib, 1992) on VO\textsubscript{2} variability was eliminated by having subjects perform test sessions at the same time of day and in the same brand of running shoes (NIKE Ltd). They emptied their bladders before any pre-test measurements were made. Subjects were weighed nude before and after each prolonged run. A standard 5 min warm-up at 60% VO\textsubscript{2max} was performed before each test. Encouragement was given to all subjects during the prolonged running bouts, but varied according to the individually perceived response during the run.

Expired air collections were taken during each exercise bout from which \( V_E \), \( VO_2 \) and \( VCO_2 \) were determined and the respiratory exchange ratio (R) value was calculated. Also, the subjects were cooled whilst running on the treadmill by an electric fan.

3.5 The collection and analysis of blood samples

The characteristics of the commercial kit (Kodak Ektachem) used in the biochemical analysis of blood samples were initially examined in the laboratory over a physiological range of standard concentrations, and reliability and validity of methods were ascertained under the supervision of a haematologist from the National University Hospital (Singapore). Calibration with known standards was performed immediately before each experimental test session.
Fingertip samples of arterialised capillary blood were obtained with a use 'once only lancet' (Microtainer Brand Safety Flow), post a pre-lancing sterile swab (Mediswab: 70% v/v isopropyl alcohol). The capillary blood sampling continued by collecting 0.3 ml of blood in a capillary vessel (Sarstedt Microvette CB 300). Blood flow was improved upon by keeping the finger positioned lower than the hand, arm and heart (Bishop and Martino, 1993). The sample was immediately centrifuged (Kodak Ektachem Microcentrifuge) to separate the plasma. Standard pipetting techniques (Kodak Ektachem DTE pipette) were applied before slide (Kodak Ektachem DT) spotting (10μl) and analysis (Kodak Ektachem DT60 11). The assays are detailed in appendices B - G.

3.6 Statistical analysis

Data are presented as means (± SD). The physiological response data were analysed by parametric statistical methods, where values at the 0.05 level were accepted as being statistically significant. Details of the statistical tests are given in the respective chapters.
Daily Stability in Running Economy

4.1 Introduction

Maximal oxygen uptake ($VO_{2max}$) is considered a major determinant of distance running success (Davies & Thompson, 1979; Shephard, 1984; Morgan & Craib, 1992). However, this does not explain why individuals with comparable $VO_{2max}$ values demonstrate differences in running performance. Probably the main reasons for differences in endurance performance/capacity of runners with similar $VO_{2max}$ values are differences in training status, followed by differences in nutritional status (Coyle et al, 1988; Morgan et al, 1995). Some investigators have suggested that performance differences within homogeneous groupings can be explained by differences in running economy. Running economy (RE) is defined as the rate of oxygen consumption at a given submaximal running velocity (Cavanagh & Williams, 1982). It has to be considered that RE may play a part because a higher $VO_2$ relates to an increased relative exercise intensity (% $VO_{2max}$) when running at the same speed as a competitor. A number of authors have demonstrated that RE is strongly correlated to endurance running performance (Costill & Fox, 1969; Bransford & Howley, 1977; Gregor & Kirkendall, 1978), and contributes to the variation in performance found among individuals displaying comparable $VO_{2max}$ values (Daniels, 1985; Krahenbuhl, Morgan & Pangrazi, 1989; Morgan, 1989; Morgan et al, 1989).

There are a multitude of psychological (Morgan and Craib, 1992), biomechanical (Martin and Morgan, 1992) and physiological (Williams et al, 1991) factors that potentially affect aerobic demand, and many extrinsic and intrinsic factors - some immutable- have been associated with interindividual variation in economy of movement. However, the question of why one
individual with similar VO_{2max} and endurance performance characteristics demonstrates markedly better RE (ie. lower VO2) when compared with another remains elusive (Williams & Cavanagh, 1987) and continues to be the focus of many research studies (Pate et al, 1992). It is important to know why some people use less metabolic energy in performing a given movement than others and if individuals are economically different from day to day because humans have "limited energy to assign to the task of producing movement" (Frederick, 1985). This is particularly important in endurance events, such as distance running where glycogen depletion is associated with a deterioration in performance (Hagerman, 1992; Karlsson & Saltin, 1971; Mason et al, 1993).

Few studies have examined within-subject variation in RE, probably because moderate group mean fluctuations in economy have obscured large ranges of individual differences (Daniels et al, 1984; Armstrong & Costill, 1985; Morgan et al, 1987). This intriguing question of intraindividual variability has recently received some attention in homogeneous groupings (Morgan et al, 1990 & 1991; Williams et al, 1991), but there is a limited information base on the range of intraindividual oxygen consumption (VO_{2}) at the same relative exercise intensity over several consecutive days. The purposes of this present study were to establish the extent to which RE varies on a day to day basis (3 consecutive days) in a group of habitually active subjects heterogeneous in terms of VO_{2max}, and to further investigate the relationship between RE and endurance running performance.
4.2 Methods

4.2.1 Subjects:
Twenty one Singaporean male physical education students (15 Chinese, 3 Malay and 3 Indian) from the School of Physical Education in Singapore volunteered to participate in the study.

4.2.2 Protocol
Subjects were instructed to adhere to their normal diets throughout the duration of the study and were advised to refrain from eating or drinking, with the exception of a normal ingestion of water (Skinner, 1987), during the 3-hour period prior to each data collection.

After familiarisation, three preliminary tests were completed to determine the oxygen cost of resting metabolism and submaximal running, and VO₂max.

Running economy: Beginning two days after familiarisation RE data were obtained over three consecutive days while subjects performed 10 min level treadmill running bouts at speeds equivalent to 60% and 85% VO₂max, and at a common speed of 3.33 m.s⁻¹ (Figure 4.1). In each session the subject performed a 5 minute warm-up at 60% VO₂max speed on the treadmill. Following this the subjects were randomly assigned an order of level treadmill running sessions, with expired air analysed during the last five minutes of each run and VO₂ quantified over the last two minute collection period. For example, day one: 10 minutes at 60% VO₂max - five minutes rest - 10 minutes at 85% VO₂max - five minutes rest -10 minutes at 3.33 m.s⁻¹; day two: 10 minutes at 85% VO₂max - five minutes rest - 10 minutes at 3.33 m.s⁻¹ -
five minutes rest - 10 minutes at 60% VO$_{2\text{max}}$; day three: 10 minutes at 3.33 m.s$^{-1}$ - five minutes rest - 10 minutes at 60% VO$_{2\text{max}}$ - five minutes rest - 10 minutes at 85% VO$_{2\text{max}}$.

During each five minute rest period subjects sat on a chair placed on the treadmill, and were encouraged to drink approximately 200 ml of cold water to try and maintain euhydration (Maughan, 1989). The heart rate of subjects was recorded throughout each submaximal session.
Figure 4.1. Experimental design of the level treadmill running study. T1, T2 and T3 were at 60% $\dot{V}O_{2\text{max}}$, 85% $\dot{V}O_{2\text{max}}$ and at 3.33 m.s$^{-1}$ respectively. Speeds were randomly assigned in a counterbalanced design over three consecutive days.
**Blood sampling:** During two of the consecutive days of data collection left hand fingertip blood samples were taken as follows. Pre-exercise samples after twenty minutes sitting rest (Hagan et al, 1978), and exercise samples during the last 90 seconds of each 10 minute exercise period. The sample was immediately centrifuged to separate the plasma and analysed for plasma lactate.

**Body composition:** Body fat was assessed by an experienced nurse (Singapore Sports Medicine Research Centre) using caliper skinfold thickness (Harpenden instrument) and the Siri formula (1961) as recommended by Jackson and Pollock (1985).

Laboratory environment was controlled at 22 - 23°C within a relative humidity range of 62 - 69% by an air conditioning system.

**Performance:** Twelve of the twenty one subjects participated in a 5 km time trial on an outdoor artificial running track. The environmental temperature was 28°C and relative humidity was 72% during the time trial.

**Statistics:** Statistical analysis were carried out using SYSTAT (Evanston, Illinois, USA). Relationships were examined using Pearson product moment correlation and the $t$ - test for correlated data was employed to detect differences between two data sets.
4.3 Results

Paired samples t-tests indicated no significant differences between resting VO$_2$ and VO$_{2\text{max}}$ test-retest values. This confirmed the repeatability for the direct measurement of resting VO$_2$ and VO$_{2\text{max}}$, and associated physiological parameters. The descriptive physiological characteristics and 5 km performance time of the subjects in the present study are shown in Table 4.1.

Paired samples t-tests comparing either day to day VO$_2$ (ml.kg$^{-1}$ min$^{-1}$) or exercise VO$_2$ (VO$_2$ minus resting metabolism) at the common running speed of 3.33 m.s$^{-1}$ showed no significant differences over the three consecutive days. The mean VO$_2$ (ml.kg$^{-1}$ min$^{-1}$) at 3.33 m.s$^{-1}$ was 44.2 ±2.0 (range = 40.1 - 47.5) and this corresponded to a relative exercise intensity of 82% (±5.7) VO$_{2\text{max}}$. The relative exercise intensity experienced at the common speed of 3.33 m.s$^{-1}$ was found to have a high negative correlation with VO$_{2\text{max}}$ ($r = -0.90$; $p < 0.05$). The mean daily VO$_2$ (ml.kg$^{-1}$ min$^{-1}$) during submaximal running at 3.33 m.s$^{-1}$ was 44.5 (±2.1), 43.8 (±2.0) and 44.2 (±2.0) for days 1, 2 and 3 respectively ($n = 21$). The VO$_2$ over three consecutive days at 60% and 85% VO$_{2\text{max}}$ (and at 3.33 m.s$^{-1}$) is shown in Table 4.2.

The range of intraindividual VO$_2$ variation which existed for the running speeds at relative exercise intensities of 60% and 85% VO$_{2\text{max}}$ and the common running speed of 3.33 m.s$^{-1}$ were determined. Expressed as a percentage of mean VO$_2$, average day to day variation in RE
at 3.33 m.s\(^{-1}\) was 3.7\%. The mean coefficient of variation (CV) for RE at 3.33 m.s\(^{-1}\), derived by averaging individual CV values, was 2.5\% (range = 0.2 - 5.4\%).
Table 4.1. Descriptive characteristics of the subjects (n = 21).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>22.9 ± 3.2</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>61.4 ± 6.9</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>170 ± 5.5</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>9.6 ± 4.2</td>
</tr>
<tr>
<td>Resting $\dot{V}O_2$ (ml.kg$^{-1}$min$^{-1}$)</td>
<td>3.9 ± 0.4</td>
</tr>
<tr>
<td>$\dot{V}O_2$max (ml.kg$^{-1}$min$^{-1}$)</td>
<td>51.6 ± 5.8</td>
</tr>
<tr>
<td>$\dot{V}E$max (l.min$^{-1}$)</td>
<td>110.5 ± 18.2</td>
</tr>
<tr>
<td>HRmax (b.min$^{-1}$)</td>
<td>187 ± 9</td>
</tr>
<tr>
<td>5 km time (min)</td>
<td>22.2 ± 1.9</td>
</tr>
</tbody>
</table>
Table 4.2. Running economy ($\dot{V}O_2$) over three consecutive days at 3.33 m.s$^{-1}$ (82% $\dot{V}O_2_{max}$), and at treadmill speeds reflecting 60% $\dot{V}O_2_{max}$ and 85% $\dot{V}O_2_{max}$ ($n = 21$).

<table>
<thead>
<tr>
<th></th>
<th>$\dot{V}O_2$ (ml.kg$^{-1}$min$^{-1}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>at 3.33 m.s$^{-1}$</td>
</tr>
<tr>
<td>Day 1</td>
<td>44.5 ± 2.1</td>
</tr>
<tr>
<td>Day 2</td>
<td>43.8 ± 2.0</td>
</tr>
<tr>
<td>Day 3</td>
<td>44.2 ± 2.0</td>
</tr>
</tbody>
</table>

Values = mean ± SD
Individual linear regression equations were computed from the VO$_2$ and heart rate data recorded during the 16 minute continuous level submaximal treadmill run and used to predict VO$_{2\text{max}}$. Pearson product moment correlation was calculated and disclosed a significant correlation with directly determined values ($r = 0.84$). No differences (NS) were found between day-to-day measurements (3 consecutive days) of heart rate, lung ventilation and plasma lactate (Table 4.3).

The relative exercise intensity expressed as a percentage of both VO$_{2\text{max}}$ and HR$_{\text{max}}$, PLA at 3.33 m.s$^{-1}$ and 5 km performance time were examined. Both percentage of VO$_{2\text{max}}$ ($r = 0.88$) and PLA ($r = 0.65$) were significantly correlated with 5 km performance time.
Table 4.3. Ventilation ($\dot{V}_E$), heart rate and plasma lactate concentration ($n = 21$) for three consecutive days of 10 minute treadmill running bouts at 60% $\dot{V}O_{2\text{max}}$, 85% $\dot{V}O_{2\text{max}}$ and 3.33 m.s$^{-1}$ each day (counterbalanced design).

<table>
<thead>
<tr>
<th></th>
<th>60% $\dot{V}O_{2\text{max}}$</th>
<th>85% $\dot{V}O_{2\text{max}}$</th>
<th>3.33 m.s$^{-1}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Speed (m.s$^{-1}$)</td>
<td>$2.29 \pm 0.23$</td>
<td>$3.38 \pm 0.4$</td>
<td>$3.33 \pm 0.0$</td>
</tr>
<tr>
<td>Actual % $\dot{V}O_{2\text{max}}$</td>
<td>$61.7 \pm 3.9$</td>
<td>$85.6 \pm 3.8$</td>
<td>$82 \pm 5.7$</td>
</tr>
<tr>
<td>$\dot{V}E$ (l.min$^{-1}$)</td>
<td>$55.5 \pm 8.2$</td>
<td>$85.5 \pm 16.8$</td>
<td>$79.6 \pm 13.1$</td>
</tr>
<tr>
<td>Heart rate (b.min$^{-1}$)</td>
<td>$144 \pm 12$</td>
<td>$172 \pm 13$</td>
<td>$170 \pm 13$</td>
</tr>
<tr>
<td>Plasma lactate (mmol.l$^{-1}$)</td>
<td>$2.8 \pm 0.8$</td>
<td>$6.5 \pm 1.8$</td>
<td>$5.7 \pm 2.3$</td>
</tr>
</tbody>
</table>

Values = mean $\pm$ SD
4.4 Discussion

No significant difference in the mean $VO_2_{\text{max}}$ of different ethnic male Singaporean groups has been reported (Ong, 1992). The subjects in this present study were found to have higher $VO_2_{\text{max}}$ values (51.6 ±5.8 ml.kg⁻¹ min⁻¹) compared to those of the young male group of regular exercisers in Ong's study (45.2 ±6.8 ml.kg⁻¹ min⁻¹). However, both compare less favourably to either the moderately trained runners in the recent RE study by Williams et al (1991) or the high values that have been reported for elite male middle and long distance runners (Svedenhag & Sjodin, 1984).

The daily group mean oxygen cost of running at 3.33 m.s⁻¹ (range over 3 days = 43.8 - 44.5 ml. kg⁻¹ min⁻¹) is slightly higher than that of trained runners (range over 2 days = 42.2 - 42.6 ml.kg⁻¹ min⁻¹) at the same speed (Morgan et al, 1991). The mean $VO_2$ at 3.33 m.s⁻¹ was 44.2 ml.kg⁻¹ min⁻¹, which is almost identical to the mean $VO_2$ of the subjects who were running at 3.35 m.s⁻¹ in another recent study (Martin et al, 1993). Subjects in Morgan et al's study had higher $VO_2_{\text{max}}$ values, supporting the earlier research of Bransford and Howley (1977), who concluded that trained distance runners ($VO_2_{\text{max}} = 69.3$ ml.kg⁻¹ min⁻¹) were more economical than untrained runners ($VO_2_{\text{max}} = 51.4$ ml.kg⁻¹ min⁻¹). The subjects in this present study could be described as untrained runners, if categorisation was based on group mean $VO_2_{\text{max}}$ values. However, it should be noted that all of the subjects in this current study are habitual exercisers and some ($n = 6$) of them are endurance trained athletes.

Expressed as a percentage of mean $VO_2$, average day-to-day variation in running economy at 3.33 m.s⁻¹ was 3.69%. This mean daily variation range appears to support those that suggest
group measures remain stable across daily treadmill running sessions, as demonstrated by Morgan et al (1991). The mean coefficient of variation (CV) for economy, derived by averaging individual CV values, was 2.54% (range = 0.24 - 5.41%). This CV was lower than the 4.06% finding of Armstrong and Costill (1985) and 7.4% at the slightly higher speed of 3.35 m.s⁻¹ reported by Martin et al (1993). However, it was higher than the mean CV of 1.32% described by Morgan et al (1991), at the same running speed of 3.33 m.s⁻¹.

This raises an important question: to what extent does the moderate mean group fluctuation conceal larger intra-individual variation in the energy cost? For example, the range of intraindividual VO₂ variation at the two relative intensity speeds (60% & 85% VO₂max) and the common speed of 3.33 m.s⁻¹ over the three consecutive days was 0.3 - 4.8, 0.1 - 6.5 and 0.2 - 4.5 (ml.kg⁻¹ min⁻¹) respectively. Thus, most individuals appear to have a stable RE, that is daily variation was less than 2 ml.kg⁻¹ min⁻¹, whereas a few subjects (n = 3) seem to vary considerably from one day to another. These findings demonstrate that a few subjects may be experiencing significant intraindividual daily VO₂ variability when performing the same submaximal running task. For example, consider two subjects in this study with similar VO₂max values (A = 54.1 and B = 55.3 ml.kg⁻¹ min⁻¹) running at 3.33 m.s⁻¹. Over the three consecutive days subject A (distance runner) displays almost no variance (range = 42 - 42.2 ml.kg⁻¹ min⁻¹), whereas subject B (triathlete) showed much more variance (range = 41.5 - 45.5 ml.kg⁻¹ min⁻¹) when running at the same speed. Additionally, there were some individuals who displayed stability in VO₂ over the three days, yet had markedly different running economy at 3.33 m.s⁻¹. As an example, subject A referred to earlier had a better running economy (range = 42.0 - 42.2 ml.kg⁻¹ min⁻¹) compared to subject C (range = 46.8 - 48.6 ml.kg⁻¹ min⁻¹).
as shown in Figure 4.2. Also, consider another individual -subject D- who had a mean VO$_2$ of 44.0 ml.kg$^{-1}$ min$^{-1}$ over the three days. This was almost identical to the group mean VO$_2$ of 44.2 ml.kg$^{-1}$ min$^{-1}$, but subject D displayed large variance on a day to day basis. On day one his running economy was 44.4 ml.kg$^{-1}$ min$^{-1}$ and on day two his running economy was poorer (46.0 ml.kg$^{-1}$ min$^{-1}$). However, on day three he demonstrated a markedly improved running economy (41.7 ml.kg$^{-1}$ min$^{-1}$) compared to the group mean. Interestingly, subject D had both the highest VO$_{2\text{max}}$ and the best performance time for the 5 km run.
Figure 4.2. Intraindividual $\dot{V}O_2$ during steady state running at 3.33 m.s$^{-1}$. 
The cause(s) of this apparent intraindividual daily variation is an open question. The present study design could account for some of the variation. The relative workload associated with non-steady state aerobic demands is 85% \( \overline{\text{VO}_2} \) and above in trained runners (Sjodin & Svendenhag, 1985). Several of the subjects in this present study would meet this criteria of training status, but as a group they would not. Further, the common speed of 3.33 m.s\(^{-1}\) was close to this upper limit for steady state aerobic running (group mean = 82% ±5.7 \( \overline{\text{VO}_2} \)). It seems certain that some of the subjects exceeded the threshold for steady state aerobic running and this was evident in the group mean respiratory exchange ratios (1.03 ±0.04) and plasma lactate values (Table 4.3). However, this does not seem to be the complete answer. An example of two of the current subjects confounds the issue. A trained distance runner was the subject with the highest \( \overline{\text{VO}_2} \) (64.1 ml.kg\(^{-1}\) min\(^{-1}\)) and was found to have an intraindividual range of daily variance (expressed as a percentage of \( \overline{\text{VO}_2} \)) of 7.5%, 9.2% and 6.7% for the three exercise intensities, namely 60% and 85% \( \overline{\text{VO}_2} \) and 3.33 m.s\(^{-1}\) respectively. The relative exercise intensity at 3.33 m.s\(^{-1}\) for this subject was 69% \( \overline{\text{VO}_2} \). Compare this with the much lower variance of a subject who was a habitual exerciser but not a trained distance runner (\( \overline{\text{VO}_2} = 55.1 \) ml.kg\(^{-1}\) min\(^{-1}\)). The comparable intraindividual range of daily variance was 3.8%, 0.2 and 2.9% and this subject was running at a relative exercise intensity of 83% \( \overline{\text{VO}_2} \) at 3.33 m.s\(^{-1}\).

At this point it is worth considering that wide inter and intra subject daily variation in \( \overline{\text{VO}_2} \) has been reported for another exercise mode (stepping) by Thomas et al (1993). Additionally, great inter and intraindividual variation has been reported for the mechanical efficiency of pure negative work (eccentric contraction) by Aura & Komi (1986). As it is well documented that eccentric contractions play an important stabilising role during the support phase of the
Chapter 4

running cycle (Cavanagh, 1990), this could be linked, perhaps tenuously, to the theory of
general motor control (Schmidt, 1975). In support of this theory it has been stated that it
would be 'uneconomical' to develop a specific motor programme for every variation of a
movement (Schmidt, 1975). However, it has been suggested that the gross motor skill of
running may not adhere to strict temporal invariance, but this is considered to be a large part
of generalised motor programme theory (Maraj et al, 1993). As an associated factor it is
interesting to note that within subject difference in the proportion of the support phase during
running has been reported as significant (Gentner, 1987). The initial support phase involves
considerable eccentric contraction, particularly from the leg extensors. Two studies have
shown longer support times to be associated with lower economy (Williams & Cavanagh,
1986 & 1987), but others have suggested the opposite (Cavanagh, 1990). There are
implications here for the mechanical efficiency of muscular work and the variability of the
energetics of running with specific reference to elastic behaviour of and energy return from
leg extensor muscles in distance running (Bosco et al, 1987; Martin et al, 1993). Thus,
considering the many mechanisms at work in the human body, perhaps one should not be
surprised if significant variations in submaximal VO$_2$ for the same exercise intensity do exist
for some individuals, even if the causes are not fully understood.

Thomas et al (1993) have suggested that intraindividual variations in oxygen demand for a
submaximal stepping task may be an important contributor to inaccuracies in predicting
VO$_{2\text{max}}$ from such submaximal tests. This study also examined the relationship between the
direct measurement of VO$_{2\text{max}}$ and predicted VO$_{2\text{max}}$ based on the linear relationship between
heart rate and oxygen uptake, using the data obtained from the submaximal speed-oxygen
uptake running task. One accepts that some of the source of variability using this approach
could be attributed to measurement inaccuracy, deviations from expected maximal heart rate and deviations from the linearity of the stated relationship. However, as maximal heart rate had already been determined one might have been expected to find a stronger relationship between the direct and predicted VO$_{2\text{max}}$ values ($r = 0.84$). This observation lends support to the idea that variability in oxygen demand during submaximal exercise may be large enough to considerably affect the predictive accuracy of VO$_{2\text{max}}$ from submaximal heart rate oxygen uptake data. The following two examples highlight this potential inaccuracy. Inferring from the heart rate-VO$_2$ relationship, the inaccuracy ranged from a VO$_{2\text{max}}$ overprediction of 6 ml.kg$^{-1}$ min$^{-1}$ compared to the directly determined value for one subject (63 vs. 57 ml.kg$^{-1}$ min$^{-1}$), to another subject’s VO$_{2\text{max}}$ which was underpredicted by 5.8 ml.kg$^{-1}$ min$^{-1}$.

The pre-exercise fingertip plasma lactate concentrations (1.5 ±0.3 mmol.L$^{-1}$) were higher than anticipated but were similar to the findings of 1.7 mmol.L$^{-1}$ (±0.5) in a recent study (El-Sayed et al, 1993). El-Sayed et al’s results indicate that fingertip blood lactate concentrations appear to be higher than venous blood lactate concentrations, both pre-exercise (1.0 ±0.3 mmol.L$^{-1}$) and in response to running at varying intensity. This difference should be taken into account when comparing the findings of this present study.

It was not unexpected to find that relative intensity (% of VO$_{2\text{max}}$) and the plasma lactate accumulation at 3.33 m.s$^{-1}$ seem to have related important and significant ($p < 0.05$) roles in 5 km performance time ($r = 0.88$ and 0.65 respectively). The average speed during the 5 km time trial was 3.75 m.s$^{-1}$ which was quite close to the common speed of 3.33 m.s$^{-1}$ employed in this present study to measure RE. Differences in distance running performance times have been explained previously in terms of the fractional utilisation of VO$_{2\text{max}}$ (Costill et al, 1973).
and the rate of accumulation of plasma lactate (Farrell et al., 1979; Yoshida et al., 1993). In agreement with others (Hurley et al., 1984; Favier et al., 1986) we found that the subjects who were specifically involved in endurance training were able to function at a lower relative intensity and with a reduced lactate accumulation at 3.33 m.s⁻¹. For example the subject (distance runner) who ran the fastest time for 5 km was also operating at the lowest (predicted) relative exercise intensity (69% \( VO_2_{\text{max}} \)) of the group at 3.33 m.s⁻¹. Another comparative example involves two subjects with similar \( VO_2_{\text{max}} \) values. One was an endurance trained subject (triathlete) who had the lowest PLA value (2.7 mmol.l⁻¹) for the group at 3.33 m.s⁻¹. This subject ran a faster 5 km time (21.9 vs 23.3 min) than another subject (volleyball player) who accumulated a PLA of 4.3 mmol.l⁻¹ when running at exactly the same relative exercise intensity (both were at 87% \( VO_2_{\text{max}} \)) at 3.33 m.s⁻¹. This appears to support the findings of previous research that observed reduced circulating levels of lactate after a period of endurance training (Gladden, 1989; McDermott & Bonen, 1993). In agreement with others (Favier et al., 1986), PLA at a high relative exercise intensity seems to be a good predictor of distance running performance.

In conclusion, the major findings of this present study are as follows. The majority of individuals appear to be invariant in \( VO_2 \) during submaximal running (60% to 85% \( VO_2_{\text{max}} \)) over three consecutive days. However, a few subjects displayed daily intraindividual variance in \( VO_2 \) (> 2 ml.kg⁻¹ min⁻¹). Thus, daily intraindividual variance in \( VO_2 \) may be obscured if one considers variance relative to group mean values. It was speculated that if variability in the performance of gross motor skills is an inescapable feature of the human motor system, then daily intraindividual variance in metabolic processes during submaximal running is inevitable to a greater or lesser extent, and this may affect performance on a given day. Also, it was suggested that the daily variability in oxygen demand during submaximal running may
account for some of the error in predicting $V\text{O}_{\text{max}}$ based on the linearity in the heart rate-oxygen uptake relationship. Finally, the findings concur with the well documented research in that relative exercise intensity and plasma lactate at a given submaximal running speed play important and significant roles in distance running performance.
Running Economy, Ventilation and Heart Rate during Prolonged Running Bouts at 65%, 70% and 75% VO_{2max}

5.1 Introduction.

Running economy (RE) appears to be stable on a day-to-day basis for most individuals (Morgan et al, 1991; Chapter 4 - present thesis). However, there is scant information available in the literature on RE during prolonged exercise (Williams and Cavanagh, 1987). Several studies have suggested that respiratory muscle fatigue (Bye et al, 1984; Loke et al, 1982) and increasing heart rate (Pate et al, 1992) are mediators of increasing VO_{2} (deteriorating RE) during prolonged running. The purpose of this study was to investigate the effects of prolonged running bouts at 65% to 75% VO_{2max} on running economy, lung function and heart rate when running at the common speed of 10.8 km.h^{-1}.

5.2 Methods

5.2.1 Subjects

Five Singaporean male physical education students (Chinese) from the School of Physical Education in Singapore volunteered to participate.

5.2.2 Protocol

Subjects were instructed to adhere to their normal diets throughout the duration of the study and were advised to refrain from eating or drinking, with the exception of water during the 3-hour period prior to each data collection. All subjects were experienced treadmill runners,
having been involved in a recent investigation on daily variation in running economy in this laboratory (Chapter 4).

After two preliminary tests to determine the oxygen cost of submaximal running and VO_{2max}, the RE data were obtained over a three week period. In each session the subject performed a 5 minute warm-up at 60% VO_{2max} speed on the treadmill and 2 to 3 minutes of individual stretching exercises. Following this the subjects ran for 60 minutes at 65% VO_{2max} (T1), 70% VO_{2max} (T2) and 75% VO_{2max} (T3), with exactly seven days between each prolonged running bout.

Within each running bout the subjects ran at the common speed of 10.8 km.h^{-1} (3 m.s^{-1} ) during minutes 6 to 10, 31 to 35 and 56 to 60 minutes of each 60 minute running bout (Figure 5.1).

Expired air was collected for analysis of oxygen and carbon dioxide content during minutes 9 to 10, 34 to 35 and 59 to 60 minutes of each prolonged running bout. The heart rate of subjects was recorded during the same time periods.

Lung function (FVC and FEV_{1}) was measured immediately pre and post the 60 minute runs (Pony Spirometer).

The subjects ingested 150 ml of water every 15 minutes during each prolonged running bout.
Figure 5.1. Experimental design of this level treadmill running study. RI is speed at 65% \( \dot{V}O_2 \max \) (Test 1), or at 70% \( \dot{V}O_2 \max \) (Test 2) or at 75% \( \dot{V}O_2 \max \) (Test 3).
Laboratory environment was controlled at 22 - 23°C within a relative humidity range of 61% - 68%.

Statistics. Two way ANOVA with two repeated measures was used for statistical analysis (SPSS).
5.3 Results

The VO\textsubscript{2max} of the subjects was 52.8 (±4.8) ml.kg\textsuperscript{-1} min\textsuperscript{-1} (Table 5.1). The relative exercise intensity when running at 10.8 km.h\textsuperscript{-1} (3 m.s\textsuperscript{-1}) was 75% ±8.2% VO\textsubscript{2max}. The actual relative intensities experienced by the subjects during each 60 minute running bout (during minutes 19-20) were 65.6% VO\textsubscript{2max} (± 2.6%), 71.1% VO\textsubscript{2max} (± 2.6%) and 75.7% VO\textsubscript{2max} (±1%).

Running economy (VO\textsubscript{2}) at 10.8 km.h\textsuperscript{-1} (75% VO\textsubscript{2max}) at minutes 9-10, 34-35 and 59-60 minutes did not change during the three 60 minute running bouts (Table 5.2).

There was evidence of heart rate drift during the prolonged run at 75% VO\textsubscript{2max} (figure 5.2), but only slight heart drift occurring during the two lower intensity runs (65% and 70% VO\textsubscript{2max}). This increase in heart rate from 154 b.min\textsuperscript{-1} to 160 b.min\textsuperscript{-1} when running at 10.8 km.h\textsuperscript{-1} (75% VO\textsubscript{2max}) during the 75% VO\textsubscript{2max} run was approaching significance (T(4) = -2.59; p = 0.06).

Post-exercise lung function (FVC and FEV\textsubscript{1}) decreased during all test sessions when compared with pre exercise lung function data (Table 5.3). A significant difference was found in FVC (pre vs post) for main effect time (F(1,3) = 26.62; p = 0.014), with measure of power indicating a 91% effectiveness. There was no difference in main effect intensity (F(2,6) = 0.15; p = 0.861). A significant difference was also found in FEV\textsubscript{1} (pre vs post) for main effect time (F(1,3) = 36.28; p = 0.009) with measure of power indicating 45% effectiveness, but no difference in main effect intensity (F(2,6) = 1.98; p = 0.218).
Table 5.1. Descriptive characteristics of the subjects (n = 5)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>22.7 ± 1.3</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>57.4 ± 6.6</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>168 ± 2</td>
</tr>
<tr>
<td>$\dot{V}O_2_{\text{max}}$ (ml.kg$^{-1}$.m$^{-1}$)</td>
<td>52.8 ± 4.8</td>
</tr>
<tr>
<td>$\dot{V}<em>E</em>{\text{max}}$ (l.min$^{-1}$)</td>
<td>106.2 ± 12.6</td>
</tr>
<tr>
<td>HR$_{\text{max}}$ (b.min$^{-1}$)</td>
<td>188 ± 7</td>
</tr>
</tbody>
</table>
Table 5.2. Oxygen uptake (ml.kg\(^{-1}\)min\(^{-1}\)) when running at 10.8 km.h\(^{-1}\) (75% \(\dot{V}O_{2max}\)) during minutes 6-10, 31-35 and 56-60 of 60 minute running bouts at 65% (T1), 70% (T2) and 75% \(\dot{V}O_{2max}\) (T 3).

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>T1</th>
<th>T2</th>
<th>T3</th>
</tr>
</thead>
<tbody>
<tr>
<td>9 - 10</td>
<td>39.5 ± 0.5</td>
<td>39.4 ± 0.7</td>
<td>39.5 ± 1.5</td>
</tr>
<tr>
<td>34 - 35</td>
<td>39.6 ± 0.7</td>
<td>38.4 ± 1.0</td>
<td>39.5 ± 0.6</td>
</tr>
<tr>
<td>59 - 60</td>
<td>38.9 ± 0.2</td>
<td>38.7 ± 1.0</td>
<td>39.1 ± 1.0</td>
</tr>
</tbody>
</table>

Values are mean ± SD (n = 5)
Figure 5.2. Heart rate (n = 5) when running at 10.8 km.h\(^{-1}\) (75% \(\dot{V}O_{2\text{max}}\)) during min 6-10, 31-35 and 56-60 of 60 min running bouts at 65% (T1), 70% (T2) and 75% (T3) of \(\dot{V}O_{2\text{max}}\). Values are mean ± SD.
Table 5.3. Pre and post exercise lung function (litres) after 60 min running bouts at 65% \( \dot{V}O_{2\text{max}} \), 70% \( \dot{V}O_{2\text{max}} \), and 75% \( \dot{V}O_{2\text{max}} \).

<table>
<thead>
<tr>
<th>%( \dot{V}O_{2\text{max}} )</th>
<th>Pre-exercise</th>
<th>Post-exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FVC</td>
<td>FEV1</td>
</tr>
<tr>
<td>65</td>
<td>3.63 ± 0.34</td>
<td>3.18 ± 0.25</td>
</tr>
<tr>
<td>70</td>
<td>3.31 ± 0.44</td>
<td>3.02 ± 0.45</td>
</tr>
<tr>
<td>75</td>
<td>3.36 ± 0.51</td>
<td>2.89 ± 0.48</td>
</tr>
</tbody>
</table>

Values are mean ± SD (n = 5)
The $V_e$ increased slightly (range = 2 to 3 l.min$^{-1}$) during all of the prolonged running bouts, and a significant difference was found in main effect time ($F(1,4) = 16.75; p = 0.015$), with measure of power indicating 85.8% effectiveness. The actual increase in $V_e$ at 10.8 km.h$^{-1}$ was from 64.5 to 67.6 l.min$^{-1}$ during T1, from 65.6 to 67.4 l.min$^{-1}$ during T2, and from 65.7 to 67.8 l.min$^{-1}$ during T3.
5.4 Discussion

The main finding of this study was that running economy at 10.8 km.h\(^{-1}\) (75% VO\(_{2\text{max}}\)) remains stable throughout 60 minute running bouts at 65% to 75% VO\(_{2\text{max}}\).

This display of unchanging running economy occurred whilst all of the subjects experienced cardiovascular drift (154 - 160 b.min\(^{-1}\)), a significantly declining lung function (range = 7.9% to 15.7%) and a small (2 - 3 l.min\(^{-1}\)) but significantly increasing V\(_E\). Thus it would seem that the subjects in this study were able to maintain their running economy (VO\(_2\)) at 10.8 km.h\(^{-1}\) (75% VO\(_{2\text{max}}\)) whilst experiencing noticeable increases in heart rate, a decrease in lung function and a slight increase in V\(_E\) during 60 minute running bouts at intensities up to 75% VO\(_{2\text{max}}\). Similarly, the study by Vollestad et al (1984), also involving subjects exercising at 75% VO\(_{2\text{max}}\), revealed only very slight increases (NS) in VO\(_2\) after 60 minutes.

These findings seem to support the research of others (Aaron et al, 1992; Poole et al, 1991; Womack et al, 1995) who concluded that changing O\(_2\) kinetics is primarily attributable to mediators other than the increasing ventilatory or cardiac work.

In light of this present study it was decided to consider investigating running economy during prolonged running bouts either at an exercise intensity above 75% VO\(_{2\text{max}}\) or beyond 60 minutes duration, or both.

Finally, it was recognised that a limitation of this study was the low number (n = 5) of subjects involved.
The Influence of Exercise Duration and Intensity on Running Economy

6.1 Introduction

It has been demonstrated that running economy (RE) is an important correlate of successful distance-running performance in individuals with similar maximal oxygen consumptions (Morgan, 1989). Also, daily variability in running economy among moderately and well-trained distance runners has been examined previously (Williams et al. 1991; Morgan et al. 1994; Chapter 4 - present thesis) and found to be stable (ie. $< 2 \text{ ml. kg}^{-1} \text{ min}^{-1}$) for the majority of individuals. Additionally, short-term changes in RE have been quantified one, two and four days following a 30 min run at 89% of maximal oxygen consumption (Morgan et al. 1990). Results from Morgan et al.'s study indicated that RE (and related mechanics) were stable for up to 4 days following the run.

Research literature indicates that at least two measurements should be taken to provide a description of RE (Morgan et al. 1991; Williams et al. 1991) and it has been demonstrated by Morgan and co-workers that intraindividual daily variation in oxygen consumption ($\text{VO}_2$) during steady state treadmill running is small (ie. $< 2 \text{ ml.kg}^{-1} \text{ min}^{-1}$). Also, oxygen drift has been examined during prolonged treadmill running (Davies & Thompson, 1986), but the mediators of $\text{VO}_2$ upward drift have not been identified with certainty, although several factors would seem to contribute to a greater or lesser degree, such as: increased energy expenditure associated with dissipation of heat generated during exercise (Hagberg et al. 1978); increased catecholamine (epinephrine/norepinephrine) and growth hormone levels (Kaciuba-Uscilko et al. 1992); increased ventilation (Casaburi et al. 1986); rise in lactate catabolism (Armon et al. 1991); increased fat metabolism (Lamb, 1984); increased heart rate
(Westerland et al. 1992), or as a consequence of voluntary muscle weakness and reflecting an increased muscle fibre recruitment (Davies and Thompson, 1986).

Hagberg and colleagues (1978) investigated oxygen consumption during 20 min at 65% and at 80% $\text{VO}_2\text{max}$ (constant-load exercise) on the bicycle ergometer. They reported small but continuous changes in $\text{VO}_2$, lung ventilation ($\text{Ve}$), rectal temperature ($T_r$) and respiratory exchange ratio (R) and questioned whether what people have called 'steady state' exercise is really a period of non steady-state exercise. In other words metabolic variables are not invariable with time and a true steady-state of physiological variables does not seem to be maintained when performing prolonged exercise (Nielsen et al. 1984). Additionally, in tasks requiring prolonged effort, the effect on $\text{VO}_2$ of associated factors tends to increase with greater exercise intensities (Brooks & Fahey, 1984). However, to our knowledge, RE has not been examined immediately pre and immediately post prolonged treadmill running. Therefore, the purpose of this study was to examine the effects of prolonged running on intraindividual variability in RE within single exercise sessions.

6.2 Methods

6.2.1 Subjects

Fifteen Singaporean male physical education students from the School of Physical Education in Singapore volunteered to participate. The same subjects were used as controls in this study.
6.2.2 Protocol

Subjects were requested to rest the day immediately before a test session and report to the laboratory at 7 am after an overnight fast.

Three preliminary tests were completed to determine resting VO$_2$, the oxygen cost of submaximal running and VO$_{2\text{max}}$. Both resting VO$_2$ and VO$_{2\text{max}}$ data collections were retested 3 days after the initial tests.

Although the optimum training intensity for improving endurance performance remains to be established (Robinson et al. 1991), endurance training around an intensity specified by the individual anaerobic threshold has been shown to elicit a training response (Keith et al. 1992). For example, by increasing VO$_{2\text{max}}$ and substantially reducing VO$_2$ drift during prolonged exercise (Casaburi et al. 1987). Therefore VO$_{2\text{max}}$ was tested pre and post, to evaluate whether variability in VO$_2$ might be related to changes in VO$_{2\text{max}}$ occurring across the 4-week testing period (Morgan et al. 1994).

The RE data was obtained both pre (RE1) and post (RE2) a prolonged run (Figure 6.1). In each session the subject performed a 5 minute warm-up at 60% VO$_{2\text{max}}$ speed on the treadmill and 2 to 3 minutes of individual stretching exercises. Following this the subjects performed 10 minutes (Rusko et al. 1986) treadmill running at 10.8 km.h$^{-1}$ (RE1), with expired air analysed during the last four minutes of each run and VO$_2$ quantified over the last two minute collection period. After this the subject had a rest period of 5 minutes sitting on a chair placed on the treadmill. The subject then performed one of the following prolonged treadmill runs:
80% VO$_{2\text{max}}$ speed for 40 minutes (T1);

or

70% VO$_{2\text{max}}$ speed for 60 minutes (T2);

or

80% VO$_{2\text{max}}$ speed for 60 minutes (T3).
Legend

RE1 = 10 min running at 10.8 km.h\(^{-1}\)
D = 2 min data collection
REST = 5 min sitting rest
T1 = Running at 80% VO\(_{2\text{max}}\) for 40 min
T2 = Running at 70% VO\(_{2\text{max}}\) for 60 min
T3 = Running at 80% VO\(_{2\text{max}}\) for 60 min
RE2 = 10 min running at 10.8 km.h\(^{-1}\)

Figure 6.1. Experimental design of the level treadmill running study.
Following the prolonged run the subject again rested for 5 minutes as above, before performing a second 10 minutes treadmill run at 10.8 km·h⁻¹ (RE2) with expired air analysed and VO₂ quantified as for RE1. This procedure was repeated every 7 days, with a randomly assigned order of prolonged runs until all three treatments had been completed.

Change in VO₂ and the other variables were calculated as the difference between the last two minutes of RE1 and RE2 runs. Mean individual variability and the overall range of variability are expressed as both an absolute value of the difference between RE1 and RE2 measurements taken and as a percentage of the total mean response. To obtain the percentage expression of individual variability, the absolute values of the difference between the measurement obtained during RE1 and RE2 for each subject were divided by that individual's mean for an individual percentage variation. These individual values were then averaged to express mean within-subject variation (Morgan et al. 1991).

Fingertip samples of arterialised capillary blood were obtained and immediately centrifuged and analysed for plasma lactate, ammonia and glucose. Pre-exercise samples were taken after 20 minutes seated rest (Hagan et al. 1978), and exercise samples during the last 60 seconds of every RE1 and RE2 exercise period.

The blood-chemistry analysis equipment was calibrated once on every day of data collection. Calibration was performed close to, just above and just below the values of levels/concentrations anticipated.
Internal body temperature for seven of the subjects during testing was measured with a rectal thermistor probe inserted 10 cm beyond the external anal sphincter and left in place for 3 minutes immediately following RE1 and RE2 runs.

Fluid intake. On the basis of previous findings, subjects were encouraged to drink at least 200 ml of water (9 - 10°C) within every 20 minute period during treadmill running (Noakes et al. 1988). This amount of fluid intake has been shown to prevent significant dehydration (and minimise hyperthermia and cardiovascular drift) in recreational distance runners (Noakes et al. 1991) with a similar \( \text{VO}_2\max \) (58.3 ml·kg\(^{-1}\)·min\(^{-1}\)) to the subjects in this present study. Body weight was recorded immediately preceding exercise (RE1) and immediately post exercise (RE2), in order to make a correction factor for normalised VO\(_2\) and to give an indication of fluid losses. Percent mass loss was calculated by the difference between the initial and final weight divided by the initial weight expressed as a percentage (Noakes et al. 1988).

During all testing the laboratory was maintained at 22 - 23°C within a relative humidity range of 59 - 72%.

Control study. The same 15 subjects acted as their own controls. For these testing sessions the subjects performed RE1 followed by 60 minutes seated rest in the laboratory, before completing RE2. Data collection procedures were as outlined above.

Statistics. Statistical analysis was carried out using STATVIEW on a Macintosh computer. Relationships between variables were examined using Pearson product moment correlation and the t-test for correlated data was employed to detect differences between two data sets.
6.3 Results

Descriptive physiological and metabolic characteristics of the subjects are presented in Table 6.1. There was no significant difference in the \( \text{VO}_2 \text{max} \) (56.6 ± 4.7 ml.kg\(^{-1}\) min\(^{-1}\)) across the testing period.

There were no significant differences between the REI and RE2 physiological and metabolic variables during the control study. There were no significant differences between control REI and T1, T2 and T3 REI physiological and metabolic data. There was no significant difference between control RE2 \( \text{VO}_2 \) and T1 RE2 VO\(_2\) (Table 6.2). There were differences (\( p < 0.01 \)) between control RE2 \( \text{VO}_2 \) and either T2 or T3 RE2 \( \text{VO}_2 \) (Table 6.2). If one assumes that differences in \( \text{VO}_2 \) up to 2 ml.kg\(^{-1}\) min\(^{-1}\) could occur as a result of daily biological variance, then there was no difference in the REI and RE2 \( \text{VO}_2 \) values during T1 (Table 6.2). There were significant increases (\( p < 0.01 \)) in the \( \text{VO}_2 \) between the REI and RE2 values during both T2 and T3. The mean absolute differences in \( \text{VO}_2 \) during T2 and T3 were 2.6 and 3.7 ml.kg\(^{-1}\) min\(^{-1}\) respectively (Table 6.2). Mean within-subject variations of \( \text{VO}_2 \) between REI and RE2 for T1, T2 and T3 were 4.1% (range = 0.2 - 10.7%), 6.7% (range = 1.1 - 13.7%) and 9.1% (range = 4.8 - 13%) respectively.
Table 6.1. Selected physiological and metabolic characteristics of the subjects (n = 15).

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>23 ± 2</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>60.5 ± 0.6</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>170 ± 4</td>
</tr>
<tr>
<td>( \dot{V}O_{2\text{max}} ) (ml.kg(^{-1})min(^{-1}))</td>
<td>56.6 ± 4.7</td>
</tr>
<tr>
<td>( \dot{V}_{\text{Emax}} ) (l.min(^{-1}))</td>
<td>117.1 ± 14.2</td>
</tr>
<tr>
<td>HR(_{\text{max}}) (b.min(^{-1}))</td>
<td>188 ± 10</td>
</tr>
</tbody>
</table>
Table 6.2. Oxygen uptake (ml.kg\(^{-1}\)min\(^{-1}\)) during 10 min level treadmill running at 10.8 km.h\(^{-1}\) (68% \(\dot{V}O_{2\text{max}}\)) pre (RE1) and post (RE2) three prolonged running bouts (T1, T2 and T3).

<table>
<thead>
<tr>
<th></th>
<th>RE1</th>
<th>RE2</th>
<th>% change</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>38.8 ± 2.7</td>
<td>38.9 ± 2.6</td>
<td>0.2</td>
</tr>
<tr>
<td>T1 (80% (V\dot{O}_{2\text{max}}) 40 min)</td>
<td>38.4 ± 2.5</td>
<td>40.1 ± 2.6</td>
<td>4.4</td>
</tr>
<tr>
<td>T2 (70% (V\dot{O}_{2\text{max}}) 60 min)</td>
<td>38.9 ± 2.9</td>
<td>*41.5 ± 2.6</td>
<td>6.6</td>
</tr>
<tr>
<td>T3 (80% (V\dot{O}_{2\text{max}}) 60 min)</td>
<td>39.0 ± 3.1</td>
<td>*42.7 ± 2.9</td>
<td>9.5</td>
</tr>
</tbody>
</table>

Values are mean ± SD * (p < 0.01) n = 15
The $V_e$ during RE2 vs RE1 was higher ($p < 0.05$) by 4.3% to 6.4%, with the greatest increase (6.4%) post the 60 minute run at 80% $VO_{2\text{max}}$ (Table 6.3).

On all trials HR was higher ($p < 0.01$) during RE2 compared to RE1 (Table 6.4). Also, on all trials $V_e$ was higher ($p < 0.05$) between RE1 and RE2 data collections (Table 6.3) with the greatest change for T3 (4.2 l.min$^{-1}$), and an identical increase for T1 and T2 of 2.7 l.min$^{-1}$. The R-value decreased ($p < 0.01$) during RE2 compared to RE1 by a similar margin (0.06) following T1 and T2, but showed the greatest decrease of 0.08 during RE2 post T3 (Table 6.3). The $T_r$ increased between RE1 and RE2 treatments ($p < 0.01$) with the greatest increase for T3 (37.4 to 38.5$^\circ$C), and an identical increase (37.4 to 38$^\circ$C) for T1 and T2 (Table 6.4).

Plasma concentrations of lactate (LA) decreased ($p < 0.01$) between RE1 and RE2 tests for T1, T2 and T3 (Table 6.5). Plasma NH$_3$ was identical for control RE1 and RE2 (163 µmol.l$^{-1}$), but increased ($p < 0.01$) between RE1 and RE2 tests for all treatments (Table 6.6). The mean absolute increases were 59, 99 and 126 µmol.l$^{-1}$ for T1, T2 and T3 respectively. The plasma glucose concentrations during RE1 and RE2 tests were similar for T1 and T2 (NS) respectively, but decreased from 4.9 to 4.5 mmol.l$^{-1}$ during T3 ($p < 0.01$), as shown in Table 6.5.
Table 6.3. Ventilation ($\dot{V}_E$) and respiratory exchange ratio (R) during 10 min level treadmill running at 10.8 km.h$^{-1}$ (68% $\dot{V}O_{2\text{max}}$) pre (RE1) and post (RE2) three prolonged running bouts (T1, T2, and T3).

<table>
<thead>
<tr>
<th></th>
<th>$V_E$ (l.min$^{-1}$)</th>
<th>R - value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RE1</td>
<td>RE2</td>
</tr>
<tr>
<td><strong>T1 (80% $\dot{V}O_{2\text{max}}$ 40 min)</strong></td>
<td>63.0 ± 13.7</td>
<td>*65.7 ± 14.7</td>
</tr>
<tr>
<td><strong>T2 (70% $\dot{V}O_{2\text{max}}$ 60 min)</strong></td>
<td>65.8 ± 15.2</td>
<td>*68.5 ± 16.1</td>
</tr>
<tr>
<td><strong>T3 (80% $\dot{V}O_{2\text{max}}$ 60 min)</strong></td>
<td>65.2 ± 13.2</td>
<td>*69.4 ± 16.8</td>
</tr>
</tbody>
</table>

Values are mean ± SD  
* higher ($p < 0.05$)  
** Lower ($p < 0.01$)
Table 6.4. Heart rate and rectal temperature (T<sub>re</sub>) during 10 min level treadmill running at 10.8 km.h<sup>-1</sup> (68% VO<sub>2max</sub>) pre (RE1) and post (RE2) three prolonged running bouts (T1, T2 and T3).

<table>
<thead>
<tr>
<th></th>
<th>Heart rate (b.min&lt;sup&gt;-1&lt;/sup&gt;)</th>
<th>T&lt;sub&gt;re&lt;/sub&gt; (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RE1</td>
<td>RE2</td>
</tr>
<tr>
<td>T1 (80% VO&lt;sub&gt;2max&lt;/sub&gt;, 40 min)</td>
<td>146 ± 12</td>
<td>*156 ± 14</td>
</tr>
<tr>
<td>T2 (70% VO&lt;sub&gt;2max&lt;/sub&gt;, 60 min)</td>
<td>145 ± 14</td>
<td>*153 ± 15</td>
</tr>
<tr>
<td>T3 (80% VO&lt;sub&gt;2max&lt;/sub&gt;, 60 min)</td>
<td>148 ± 14</td>
<td>*159 ± 16</td>
</tr>
</tbody>
</table>

Values = mean ± SD  
* Higher (p < 0.01) n = 15
Table 6.5. Plasma glucose (GL) and lactic acid (LA) concentrations during treadmill running at 10.8 km.h\(^{-1}\) (68\% \(V_{O_{2\max}}\)), pre (RE1) and post (RE2) three prolonged running bouts (T1, T2 and T3).

<table>
<thead>
<tr>
<th></th>
<th>GL (m.mol.L(^{-1}))</th>
<th>LA (mmol.L(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RE1</td>
<td>RE2</td>
</tr>
<tr>
<td>T1 (80% (V_{O_{2\max}}), 40 min)</td>
<td>4.6 ± 0.4</td>
<td>4.6 ± 0.3</td>
</tr>
<tr>
<td>T2 (70% (V_{O_{2\max}}), 60 min)</td>
<td>4.7 ± 0.4</td>
<td>4.6 ± 0.4</td>
</tr>
<tr>
<td>T3 (80% (V_{O_{2\max}}), 60 min)</td>
<td>4.9 ± 0.5</td>
<td>4.5 ± 0.3*</td>
</tr>
</tbody>
</table>

Values = mean ± SD  * lower (p < 0.01) n = 15
Table 6.6. Plasma ammonia (NH₃) during submaximal running at 10.8 km.h⁻¹ pre (RE1) and post (RE2) three prolonged running bouts (T1, T2 and T3).

<table>
<thead>
<tr>
<th></th>
<th>NH₃ (µmol.L⁻¹)</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RE1</td>
<td>RE2</td>
<td>% change</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>163 ± 52</td>
<td>163 ± 35</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>T1 (80% VO₂max 40 min)</td>
<td>180 ± 64</td>
<td>*239 ± 73</td>
<td>32.7</td>
<td></td>
</tr>
<tr>
<td>T2 (70% VO₂max 60 min)</td>
<td>218 ± 77</td>
<td>*317 ± 113</td>
<td>45.4</td>
<td></td>
</tr>
<tr>
<td>T3 (80% VO₂max 60 min)</td>
<td>192 ± 64</td>
<td>*318 ± 117</td>
<td>65.6</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SD * higher (p < 0.01) n = 15
During testing the subjects ingested 698 (± 234), 896 (± 229) and 932 (± 331) ml of water for T1, T2 and T3 respectively. Mean BW loss ranged from 1.1% to 2.2% (p < 0.01), but did not differ significantly between trials.

6.4 Discussion

The absence of change in VO\textsubscript{2max} (56.6 ± 4.7 ml.kg\textsuperscript{-1} min\textsuperscript{-1}) across time (4-weeks) suggests that intraindividual variability in RE post prolonged running was not influenced by changes in VO\textsubscript{2max}. The mean relative exercise intensity (%VO\textsubscript{2max}) for the RE1 was 68.4% (± 8.4%). During RE2 the VO\textsubscript{2} increased (p < 0.01) following T2 and T3. The change in VO\textsubscript{2} was equivalent to a rise in relative intensity of 3%, 4.6% and 6.6% for T1, T2 & T3 respectively.

During the prolonged runs (T1, T2 and T3), the subjects ingested 698, 896 and 932 ml of plain water respectively. Unfortunately, the different volumes of water ingestion did not completely prevent dehydration (BW loss = 1.1% to 2.2%; p < 0.01). Even small amounts of dehydration (1% to 4.2% reduction in BW) can lead to an increase in core body temperature (Montain and Coyle, 1992), influence cardiovascular-respiratory dynamics (Montain & Coyle, 1992) and negatively influence endurance performance (Davies and Thompson, 1986). The BW loss in this present study slightly accentuated the rise in VO\textsubscript{2}. When expressed in terms of ml.kg\textsuperscript{-1} min\textsuperscript{-1}, the absolute increases being 1.7, 2.6 and 3.7 ml.kg\textsuperscript{-1} min\textsuperscript{-1} for T1, T2 and T3 respectively. The weight loss would be expected to reduce the workload, but as VO\textsubscript{2} increased, this suggests that a tendency towards a dehydrative state may constitute a part of the change in VO\textsubscript{2} found in this present study.
The absence of significant change ( > 2 ml.kg⁻¹ min⁻¹) in \( \text{VO}_2 \) during RE2 post T1 supports the findings of a previous study revealing no short-term change in RE following 30 min of running at 89% \( \text{VO}_{2\text{max}} \) (Morgan et al. 1990). This is in agreement with the findings of Dick and Cavanagh (1987), involving experienced distance runners (\( \text{VO}_{2\text{max}} = 65 \text{ ml.kg}^{-1} \text{ min}^{-1} \)) running for 40 minutes at 66% \( \text{VO}_{2\text{max}} \), who displayed a mean increase in \( \text{VO}_2 \) of 0.7 ml.kg⁻¹ min⁻¹. However, the change in \( \text{VO}_2 \) during RE2 following both T2 and T3 reported in the present study suggests that RE is significantly (\( p < 0.01 \)) and negatively affected during prolonged running beyond 60 minutes. There may be differences between the change in economy for running compared to cycling during prolonged exercise. Hamilton and co-workers (1991) reported a similar percentage of \( \text{VO}_2 \) increase of 6% to the 6.6% increase found during T3, after their subjects had cycled for 120 minutes at 70% \( \text{VO}_{2\text{max}} \) with no fluid replacement. McLellan and Cheung (1992) also found a similar increase (7.2%) in \( \text{VO}_2 \) during cycling for 30 minutes at 72.4% \( \text{VO}_{2\text{max}} \). Thus it seems that subjects become less economical during cycling for 30 minutes but not during running for corresponding time periods and within a similar range of relative intensity.

Mean within-subject variation of \( \text{VO}_2 \) between RE1 and RE2 for T1, T2 and T3 was 4.1%, 6.7% and 9.1% respectively. This suggests that intra-individual variability of \( \text{VO}_2 \) increases both with increasing time (T1 vrs T3; \( p < 0.01 \)) and with increasing exercise intensity (T2 vrs T3; \( p < 0.01 \)). However, there did not appear to be any trend with inter-individual variation of \( \text{VO}_2 \). For instance, subjects who were more economical during RE1 (T1, T2 or T3) did not display a smaller \( \text{VO}_2 \) increase during RE2 when compared to the less economical subjects in RE1. Thus, several of the least invariant runners (ie. < 1 ml. kg⁻¹ min⁻¹) in RE1 were the most variant in RE2 and several of the most invariant in RE1 (ie. (> 2 ml.kg⁻¹ min⁻¹) were the least
invariant in RE2. Also, some subjects demonstrated invariance with increasing exercise intensity but not with increasing time, and vice versa. Such subjects were equally likely to be one of the most or one of the least economical of subjects.

The individual anaerobic threshold (IAT) is the highest exercise intensity that can be maintained for prolonged periods. Average speed for the prolonged run in T2 (11.1 km.h⁻¹) and T1 and T3 (12.8 km.h⁻¹) was greater than RE speed tested (10.8 km.h⁻¹). The mean relative intensity during RE1 was 68.4% and during RE2 it was 71.4% (T1), 73% (T2) and 75% (T3). Mean RE1 plasma lactate concentrations were 3.9 (T1), 3.9 (T2) and 3.7 (T3) mmol.l⁻¹. Therefore, it is likely that the plasma lactate concentrations were higher during the prolonged runs in T1 and T3, as the RE testing speed was lower than the T1 and T3 prolonged runs speed. This finding appears to be in conflict with those who have suggested that maximal steady state achievable for about 60 min is around the 2 mmol.l⁻¹ level (Fay et al. 1989), but tends to support others (Stegmann & Kindermann 1982) who have reported that the IAT can range from lactate concentrations of 2 - 7.5 mmol.l⁻¹. Jenkins and Quigley (1990) have shown that highly trained endurance cyclists can tolerate high blood lactate concentrations (8.9 ±1.6 mmol.l⁻¹) during 30 minute exercise at their critical power.

The drop in plasma lactate found in this present study during the RE2 post T1, T2 and T3, but not found during the control run, is in agreement with previously reported data during prolonged exercise suggesting a larger blood lactate removal compared to the release by working muscles (Hermansen and Stensvold, 1972). According to Henry (1951), Mazzeo and co-workers (1986) and Bouissou and colleagues (1987) the rise in VO₂ with exercise might be due to the oxidative removal of lactate which appears in the blood during exercise. In this
present study the RE2 plasma lactate concentrations (ie. post prolonged run) were lower compared to RE1 concentrations for all three treatments, indicating that plasma lactate accumulation was not a limiting factor. This conforms with findings from other studies of prolonged exercise that have shown plasma and muscle lactate may be used as a fuel during prolonged exercise in the presence of low glycogen levels (Jacobs, 1981).

In this present study the upward drift of HR was evident during all RE2 testing compared to RE1 (Table 6.4). The mean HR values (RE1 = 146 b.min\(^{-1}\) and RE2 = 156 b.min\(^{-1}\); p < 0.01) suggest that the subjects in this present study were exercising just below the anaerobic threshold (Maffulli et al. 1987; Brettoni et al. 1989; Maffulli et al. 1991). This apparent change in central hemodynamics has been observed many times before (Johnson, 1987; Norman et al. 1987). The magnitude of the increase in HR has been shown to be both influenced by increased circulating catecholamines (Kalis et al. 1988) and directly related (r = 0.99, p < 0.01) to the amount of weight loss after prolonged (62-67% VO\(_{2\text{max}}\)) steady state exercise (Montain and Coyle. 1992).

Hamilton and co-workers (1991) have reported that when euhydration is maintained during prolonged submaximal exercise (120 min at 70% VO\(_{2\text{max}}\)), cardiovascular drift of HR is prevented. As the subjects in this present study did not maintain euhydration, evidenced by their BW loss, it is likely that the fluid loss contributed towards the increased heart rates.

The \(V_E\) increased by a small but significant margin during RE2 vs RE1 in this present study, with the greatest increase (6.4%) experienced post the 60 minute run at 80% VO\(_{2\text{max}}\) (Table 6.3). Hagberg and co-workers (1978) suggested that the cause of the increase in VO\(_2\) during 20 min constant load exercise (65% and 80% VO\(_{2\text{max}}\)) is primarily a temperature effect, both
directly (Q10 effect) and indirectly by playing a predominant role in increasing $V_e$, for instance via increased breathing frequency. Increased breathing frequency would increase the $VO_2$ of the respiratory muscles. Others have stated that a causal relationship between LA accumulation and increases in $V_e$ appears to be the most plausible explanation for the increases in $V_e$ during heavy exercise (Loat and Rhodes, 1993). Davies & Thompson (1986) and others (Casaburi et al. 1987; Kalis et al. 1988; McLellan and Cheung, 1992) have all provided evidence that a contribution to $VO_2$ drift, associated with an increased $V_e$ from the oxygen cost of breathing, seems plausible.

In this present study absolute change in $VO_2$ correlated highly with absolute change in $T_{re}$ between RE1 and RE2 of T3 ($r = 0.77; p < 0.01; n = 7$). Additionally, the absolute change in $T_{re}$ and HR between RE1 and RE2 of T3 was even more strongly associated ($r = 0.89; p < 0.01; n = 7$). Others have also found a strong relationship ($r = 0.69; p < 0.001$) between absolute changes in HR and in $T_{re}$ (Graetzer et al, 1994), especially during the latter stages ($r = 0.99$) of very prolonged submaximal running (Davies and Thompson, 1986). Westerland and co-workers (1992) suggested that their increase in $T_{re}$ (0.8°C) accounts for all of the increase in $VO_2$ during prolonged (30 minutes) low intensity (40% $VO_{2max}$) level running. However, as other variables also change during prolonged endurance exercise, such as HR, $V_e$ and $R$, this would seem to be an inappropriate conclusion. In this present study for example, $T_{re}$ during RE2 of T1 and T2 were identical (38°C ie. an increase of 0.6°C for both from the RE1 value), but the increase in $VO_2$ (1.7 vrs 2.6 ml.kg$^{-1}$ min$^{-1}$ respectively) was different ($p < 0.01$). Nonetheless, the findings of others such as Armon and colleagues (1991) provide supportive evidence that increased body temperature does appear to play a major role in oxygen drift. However, at this point it is important to refer to the excellent study by Poole
et al (1991). They concluded for the subjects in their study (VO$_{2\text{max}}$ = 55.7 ml kg$^{-1}$ min$^{-1}$), the slow component of VO$_2$ kinetics reflects predominantly increased leg VO$_2$, which precludes a dominant role for factors such as ventilatory, cardiac, or auxiliary muscle work and metabolic stimulation at sites outside the exercising limbs.

During prolonged exercise at 60-80% VO$_{2\text{max}}$, fatigue is known to coincide with impaired neuromuscular transmission (Wilson and Maughan, 1992), accumulation of lactate in muscle (Sahlin, 1978), changes within the central nervous system (Bigland-Ritchie et al. 1986), depletion of the muscle glycogen stores (Norman et al. 1987) and selective glycogen depletion of muscle fibres (type 1 fibres in particular - Costill et al. 1974). In this present study it is likely that the muscle glycogen content of the muscles involved in running was low during the RE2 runs (Bergstrom and Hultman, 1967; Hultman, 1971; Davies and Thompson, 1986). However, plasma glucose was well maintained throughout testing (Table 6.5). Thus, there is no reason to suspect that plasma glucose concentrations (nor muscle glycogen) were a limiting factor (Hultman & Greenhaff, 1992).

If one accepts in this present study that the subjects were low on muscle glycogen content and combined this information with the lower R values (P < 0.01) and increased V$\text{E}$ values (P < 0.01) found, then this suggests that part of the increased VO$_2$ could be accounted for, especially as the greatest change in these variables was for T3 (Table 6.3) which also had the greatest increase in VO$_2$. However, this is not as simple as it seems. For instance, Gass and colleagues (1983) reported a decrease in R with no increase in VO$_2$ or V$\text{E}$ in highly trained females during prolonged treadmill exercise at 65 - 75% VO$_{2\text{max}}$. Also, Sahlin and co-workers (1990) found significant (p < 0.05) increases in VO$_2$, V$\text{E}$ (and HR) during cycling exercise to
fatigue at 74% VO_{2\text{max}}, but no significant decrease in the R value between the 20th min and time of fatigue (75 min; range 61 - 96 min). Indeed the R value (0.96) at the 40th minute, 60th minute and end of exercise was the same. It is difficult to reconcile these findings from the above studies.

In this present study the pre-exercise plasma ammonia (NH₃) level was 82 ± 12 μmol.l⁻¹, which is comparable with the levels found in normal resting man (Mutch and Banister, 1983). The reports of exercise on plasma NH₃ in humans are scarce and not well understood. Mean plasma NH₃ concentration increased during the first RE1 to >160 mmol.l⁻¹ and was identical during RE1 and RE2 control runs, but showed a pronounced increase (p < 0.01) during RE2 to 239 μmol.l⁻¹ (T1), 317 μmol.l⁻¹ (T2) and 318 μmol.l⁻¹ (T3). Previous studies have also shown pronounced increases in plasma NH₃ during submaximal exercise (Eriksson et al. 1985; Graham et al. 1987) and during prolonged submaximal exercise to fatigue (Broberg and Sahlin, 1988; Sahlin et al. 1990). Broberg & Sahlin (1988) and others (Norman et al. 1988) have suggested that a low muscle glycogen content of both type 1 and type 2 muscle fibres may be associated with an increased degradation of adenine nucleotides. The main source of plasma NH₃ appears to be the AMP deaminase reaction to inosine monophosphate (IMP) and NH₃ in contracting skeletal muscle (Broberg and Sahlin, 1988), possibly with a small contribution from the reductive deamination of glutamate (Lowenstein, 1972). The low muscle glycogen content may limit the rate of glycogenolysis and impair resynthesis of ATP in glycogen-depleted fibres (Norman et al. 1987), resulting in an increase of ADP and AMP (Broberg and Sahlin, 1989; Spencer et al. 1991).
It has been suggested that muscles poor in mitochondria produce more NH₃ during exercise than muscles rich in mitochondria (Raggi et al. 1969; Meyer and Terjung, 1979). This suggests that changes in fibre type recruitment could also be an alternative explanation for enhanced IMP and NH₃ formation. However, a recent study by Norman and colleagues (1994) reported no significant correlation between AMP deaminase activity and either the proportion of different fibre types or the oxidative capacity and contractile property of muscle. This suggests that factors other than fibre type might determine the individual variation in AMP deaminase activity, but does not exclude the possibility of an intra-individual difference in AMP deaminase activity between type 1 and type 2 fibres within the same muscle. The most striking finding in a study by Norman and co-workers (1987), which involved subjects cycling at 68% VO₂max for 80 ± 13 minutes, was a pronounced increase in muscle IMP (during the latter part of exercise) combined with a strong inverse relationship between IMP and muscle glycogen content (r = -0.86). Also of interest is the recent finding that IMP formation has been shown to be attenuated when carbohydrates are supplied during exercise with low initial glycogen levels (Spencer et al. 1991). In this present study the biggest RE1 and RE2 plasma NH₃ difference was 126 mmol.l⁻¹ for the T3 run and the smallest difference was 59 mmol.l⁻¹ for the T1 run (Table 6.6). Thus, although the significance of NH₃ production is not fully understood it cannot be overlooked (Worceł and Ereksinska, 1962; Lo and Dudley, 1987).
In conclusion, the results of this investigation demonstrate that RE does become worse during prolonged runs and the magnitude of the deterioration in RE increases with both increasing exercise intensity and with increasing time. Also, differences in RE between some individuals become accentuated with exercise intensity and/or time, but there does not seem to be any clear pattern to this. In other words, variance and invariance in RE during prolonged exercise was demonstrated, but this variance or invariance can occur both with more economical and with less economical subjects.
The Influence of either No Fluid or Carbohydrate-Electrolyte Fluid Ingestion and Environment (thermoneutral vs hot and humid) on Running Economy post Prolonged, High-Intensity Running.

7.1 Introduction

Morgan et al (1995) support the findings of others (Chapter 6 - present thesis; Williams and Cavanagh, 1987) that variability in running economy (BETWEEN running bouts) is independent of performance ability, training status or familiarity with running. Thus, although some runners can display variance in running economy (NS) between exercise bouts, most runners appear to demonstrate daily stability in running economy (Chapter 4 - present thesis; Hunt et al, 1995).

Variance (ie. > 2 ml.kg⁻¹ min⁻¹) in running economy during a single bout of prolonged exercise has been found in a previous study (Chapter 6 - present thesis), and this variance can occur with both more economical and with less economical subjects. The previous finding of significant changes in running economy following a 60 minute run at 80% VO₂ max (Chapter 6 - present thesis) was not surprising considering, for example, that stride length changes have been shown to occur with fatigue during prolonged running (Williams et al, 1991; Siler and Martin, 1991) and temporal differences in neuromuscular patterns as stride length changes have recently been reported (Bates and Schneider, 1995). Also, when one has low muscle glycogen in the fibres then those fibres are not recruited, and yet they may still be using oxygen while one is recruiting other fibres.
The large number of physiological, biomechanical and psychological factors associated with running economy may explain the interindividual and large intraindividual differences observed for running economy in the literature (Bailey and Pate, 1991; Joyner, 1991; Pate et al, 1992). However, the presence of great inter and intraindividual variations in running economy makes it more difficult to examine how fatigue may influence running economy. Therefore, the relationships between distance running pattern and running economy have been usually examined in nonfatiguing situations (Cavanagh and Williams, 1982; Kaneko et al, 1987).

Numerous studies of moderate-intensity exercise (bench stepping, walking, cycling and running) have shown that carbohydrate (CHO) ingestion can improve performance and delay fatigue (Ladell, 1955; Pitts, 1944; Wright et al, 1991; Tsintzas et al, 1994). Williams et al (1992) confirmed the finding that dietary CHO loading improves endurance running performance. Also, Tsintzas et al's (1993) study showed that performance time for a 30-km road race is improved after ingesting a 5% CHO solution just prior to and during (every 5-km) the race. Tsintzas et al (1993) and Nicholas et al (1994) have demonstrated that carbohydrate-electrolyte ingestion spares muscle glycogen during prolonged submaximal running and during intermittent, high intensity shuttle running (90 minutes) respectively. This differs from the previous finding of Coyle et al (1986) who found that the pattern of muscle glycogen utilisation was not different during the first 3 hours of cycling at 71% $\text{VO}_2\text{max}$ when ingesting either a placebo (flavoured water) or a glucose polymer solution. Importantly however, Below et al (1995) correctly highlight that very few studies have been conducted to determine the influence of different types of CHO-electrolyte fluid replacement on performance during shorter duration (around 60 min) higher intensity exercise (80% $\text{VO}_2\text{max}$).
or more). Wilber and Moffat (1992) have demonstrated endurance capacity performance enhancement during treadmill running at 80% VO$_{2\text{max}}$ and Below et al (1995) have shown a significantly faster endurance performance time after prolonged cycling at 80% VO$_{2\text{max}}$ as a result of CHO fluid ingestion immediately before and during testing. Thus, there does seem to be a wealth of information concerning endurance exercise up to and around 70% - 75% VO$_{2\text{max}}$ but a scarcity of information on higher intensity endurance exercise and,

"this lack of information is unfortunate since many competitive athletic events are completed under these conditions" (Below et al, 1995).

Others have highlighted that there is a limited understanding of the extent to which locomotor energy requirements can be perturbed within a high-intensity exercise bout (Morgan and Craib, 1992). As stated above, much of the previous research has centred around prolonged work at low to moderate relative exercise intensity (ie. < 75% VO$_{2\text{max}}$). Presently, there is little information about the effects of fluid replacement beverages on VO$_2$ kinetics, blood homeostasis and performance during high-intensity exercise. Also, the questions of whether running economy deteriorates at certain intensities and/or durations of exercise (Chapter 6 - present thesis) demand further supportive evidence.

Prolonged high-intensity exercise results in increases in body temperature and plasma osmolality, a decrease in plasma volume and muscle glycogen stores, and potential imbalances in both intracellular and extracellular electrolytes (Powers et al, 1990). It is possible that one or some combination of such homeostatic imbalances might lead to diminished performance. The challenge for the sports scientist and athlete would be to limit
exercise-induced disturbances in homeostasis and one such intervention might be the ingestion of solutions during exercise.

The beneficial effects of CHO ingestion are due to its ability to maintain blood glucose levels and a high rate of CHO oxidation, at a time when liver and muscle glycogen levels are low (Coggan and Coyle, 1987; Coyle et al, 1986). This begs the question: is it possible to attenuate deteriorating running economy (or maintain/enhance RE) via enhanced/maintained blood glucose levels and/or an increased contribution of carbohydrate to oxidative metabolism (Maughan, 1991), ie. an increase in the efficiency of the oxidative energy system (Lamb, 1984). Additionally, although it is well documented that dehydration has an adverse effect on cardiovascular and thermoregulatory function and reduces exercise performance (Sawka and Pandolf, 1990), it is still not completely clear how dehydration influences running performance (Coyle and Montain, 1992). Therefore, this study was also conducted to document the effects of prolonged running with no fluid replacement on running economy. Further, the possibility that repeated drinking during exercise might prevent deterioration in running economy during prolonged high-intensity running has not been investigated to my knowledge. Hence, another purpose of this study was to determine whether CHO ingestion with repeated drinking (with electrolyte levels held constant) during exercise can help maintain or improve running economy, following high-intensity exercise lasting 1 h. Finally, fluid replacement during prolonged exercise in the heat has been shown to be important for enhancing endurance performance while maintaining thermoregulatory and circulatory function (Wyndham and Strydom, 1969; Millard-Stafford et al, 1992). Singapore is a hot and humid environment and the majority of Singaporean runners exercise outdoors. Hence, this study was also conducted to investigate the environmental influence (thermoneutral vs hot
and humid) on running economy when ingesting CHO-electrolyte fluid (repeated drinking design) during prolonged high-intensity exercise. Thus, in addition to the laboratory thermoneutral running bouts the subjects would have their RE measured pre and post an outdoor run under real environmental conditions for those resident in Singapore.

7.2 Methods

7.2.1 Subjects

Fifteen Singaporean male physical education students from the School of Physical Education in Singapore volunteered to participate.

7.2.2 Protocol

Subjects were asked to maintain their normal diets and activity levels prior to and during testing, but were requested to refrain from exercise for 24 hours pre-testing and report to the laboratory at 7 am after an overnight fast.

This present study involved subjects who had participated in earlier studies (Chapters 4, 5 and 6) of running economy at our laboratory in Singapore.

Two preliminary tests were completed to determine the oxygen cost of submaximal running and \( VO_{2\max} \). The RE data was obtained both pre (RE1) and post (RE2) a prolonged run (Figure 7.1). In each session the subject performed a 5 minute warm-up at 60% \( VO_{2\max} \) speed on the treadmill and 2 to 3 minutes of individual stretching exercises. Following this the subjects performed 10 minutes (Rusko et al. 1986) treadmill running at 10.8 km.h\(^{-1}\) (RE1),
with expired air analysed during the last four minutes of each run and VO₂ quantified over the last two minute collection period. The common running speed of 10.8 km.h⁻¹ was chosen so that none of the subjects would experience a relative intensity greater than 80% VO₂ max. After this the subject had a rest period of 5 minutes sitting on a chair placed on the treadmill.
Legend

EA = Expired air collection
CS = Capillary sample
HR = Heart rate
T = Rectal Temperature

↓ = Fluid Intake
■ = 10 min run at 10.8 km.h⁻¹
□ = 60 min run at 80% $\bar{VO}_2_{max}$

Figure 7.1. Schematic representation of the experimental procedures.
During this resting period a bolus of 500 ml (Hawley et al, 1992) of the treatment fluid was ingested before starting the prolonged run. Gisolfi and Duchman (1992) recommend up to 500 ml of water pre-event for events between 1 - 3 hours. This bolus was an attempt to hold constant gastric volume prior to the prolonged run (Maughan and Noakes, 1991; Mitchell and Voss, 1991; Noakes et al, 1991; Rehrer et al, 1989). The subject then performed an initial 60 minute run at 80% VO$_2$ max with no fluid intake (D). During the 60-minute running bout at 80%VO$_2$ max expired air was analysed during minutes 8 - 10 (80%RE1) and 58 - 60 (80%RE2). This initial exercise bout involving a dehydration run facilitated the determination of fluid needed to try and maintain body weight based on individual sweat rate data. Thus, information concerning fluid balance was monitored by changes in nude body weight before and post exercise. Each kilogram of weight loss corresponded to 990 ml (33 fluid ounces) of dehydration (Coyle and Montain, 1992). Additionally, change in body weight enabled a correction factor for normalised VO$_2$. Immediately and continuously following the prolonged running bout the subject performed a second 10 minutes treadmill run at 10.8 km.h$^{-1}$ (RE2), with expired air analysed and VO$_2$ quantified as for RE1. This procedure (Figure 7.1) was repeated on another three occasions every 7 days with the addition that the subjects received one of the following fluids via a repeated ingestion (group mean = 17 ml.min$^{-1}$) feeding schedule (every 2 minutes) during the 60 minute 80% VO$_2$ max running bout:

**Indoor laboratory**

Either a 6% CHO-electrolyte (21 Na$^+$ mEq; 3 mEq K$^+$ ) solution (G-E) or an artificially sweetened/coloured water-electrolyte (21 Na$^+$ mEq; 3 mEq K$^+$ ) placebo (P-E), with electrolytes held constant in both fluids (Gatorade, Quaker Oats Co.).
Outdoor track

On one occasion subjects performed the 60 minute run outdoors on the university 400 metre artificial running track whilst attempting to run at the same speed as per 80% $\text{VO}_2\text{ max}$ treadmill running speed, whilst receiving repeated ingestion of the 6% CHO-electrolyte solution (G-E-O), as above. The RE data (10.8 km.h$^{-1}$) was obtained in the thermoneutral laboratory immediately pre and post the outdoor run.

Fluid ingestion (18°C - 19°C) was taken orally from a plastic 60 cc syringe (Becton Dickinson & Co). Treatment order was randomised and counterbalanced and test solutions were administered double-blind. A 7-day recovery period between treatments was chosen to ensure sufficient recovery time between running bouts and also because, according to Williams et al (1992), this is often the maximum time an endurance athlete/participant has between races/events during the height of the athletics season. The exercise intensity during the 60 minute period was chosen based on findings from a recent study indicating a relative intensity of 80% $\text{VO}_2\text{ max}$ for 60 minutes resulted in a significant deterioration of RE (Chapter 6). Others (Xu and Montgomery, 1994) have also reported a significant deterioration in RE following prolonged running at 80% $\text{VO}_2\text{ max}$.

Encouragement was given to all subjects during the 60 minute running bouts, but varied according to the individually perceived response during the run. However, standardised encouragement was given to all subjects during RE2 by an experimenter who was blind to the treatment administration.
Steady state was determined from 6 consecutive 20-s values taken during the last 2 minutes of RE1 and RE2 and also during minutes 9 + 10 (80%RE1) and minutes 59 + 60 (80%RE2) of the prolonged running bout. Both analysers were calibrated with gases of known concentrations prior to each subject's RE1 and also 30 minutes before each subject's RE2 (ie. during the prolonged run) for every data collection throughout the study. I chose to quantitate the change in \( V_O^2 \) and the other variables as the difference between the last two minutes of RE1 and RE2 runs, and likewise for the two minute samples taken during 80%RE1 and 80%RE2.

Fingertip samples of arterialised capillary blood were obtained and the sample was immediately centrifuged (Micro Haematocrit Mk. 5, Hawksley and Sons Ltd) to separate the plasma and analysis for plasma lactate, ammonia, glucose, potassium and sodium. Plasma volume changes were calculated from changes in hematocrit and hemoglobin according to Dill and Costill (1974). Exercise samples were taken during the last 60 seconds of every RE1 and RE2 exercise period.

Internal body temperature for seven of the subjects during testing was measured with a rectal thermistor probe inserted 10 cm beyond the external anal sphincter and left in place for 2 minutes immediately following RE1 and RE2 runs.

All the measurements were made in a well-ventilated, air conditioned laboratory. During all testing the laboratory was maintained at 22 - 23°C within a relative humidity range of 59 - 72%. During the outdoor run the temperature and humidity ranged from 25° - 35°C and 66% - 77% respectively.
Statistics. Statistical analysis was carried out using Statview on a Macintosh computer. Relationships between variables were examined using Pearson product moment correlation and the t-test for correlated data was employed to detect differences between two data sets. Two way analysis of variance with repeated measures was used to identify trends between trials over time.

### 7.3 Results

Descriptive physiological and metabolic characteristics of the subjects are presented in Table 7.1. There was no significant difference in the VO_{2}\text{max} (55.5 \pm 4.4 \, \text{ml.kg}^{-1} \, \text{min}^{-1}) across the testing period. There were no significant differences in the REI physiological and metabolic variables between treatments.

The RE2 was significantly higher than RE1 for all treatments, with a mean group absolute range of 3.4 - 4.7 ml.kg^{-1} min^{-1}. There were no significant differences (ANOVA) between treatments for RE2 (Table 7.2).

The average running speed for the subjects running at 80\% VO_{2}\text{max} was 12.7 \pm 1.2 \, \text{km.h}^{-1}. There were no significant differences between treatments for both 80\%RE1 and 80\%RE2 (ANOVA), but the 80\%RE2 was significantly higher than 80\%RE1 across treatments (Table 7.3).

The V_{E} was significantly higher during both RE2 and 80\%RE2 compared to RE1 and 80\%RE1 respectively for all treatments (Figures 7.2 and 7.3). There were no differences (NS) between treatments for either RE2 or 80\%RE2 (ANOVA).
Table 7.1. Physical characteristics and maximal physiological data of subjects (n = 15)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (±S.D.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>23.5 ± 1.4</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>171 ± 3.4</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>62.6 ± 7.4</td>
</tr>
<tr>
<td>( \dot{V}O_2 \text{max} ) (ml.kg(^{-1}).min(^{-1}))</td>
<td>55.5 ± 4.4</td>
</tr>
<tr>
<td>( \dot{H}R \text{max} ) (b.min(^{-1}))</td>
<td>192 ± 7</td>
</tr>
<tr>
<td>( \dot{V}E \text{max} ) (l.min(^{-1}))</td>
<td>118.1 ± 16.2</td>
</tr>
</tbody>
</table>
Table 7.2. Oxygen uptake (\(\dot{\text{VO}}_2\)) during treadmill running at 10.8 km.h\(^{-1}\) (65% \(\dot{\text{VO}}_{2\text{max}}\)), pre (RE1) and post (RE2) prolonged high-intensity running (80% \(\dot{\text{VO}}_{2\text{max}}\) 60 min), whilst ingesting either no fluid, a 6% glucose-electrolyte or a placebo-electrolyte solution (n = 15).

<table>
<thead>
<tr>
<th></th>
<th>VO(_2) (mL.kg(^{-1})min(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RE1</td>
</tr>
<tr>
<td>No fluid</td>
<td>36.6 ± 2.4</td>
</tr>
<tr>
<td>Glucose-electrolyte</td>
<td>36.3 ± 2.5</td>
</tr>
<tr>
<td>Placebo-electrolyte</td>
<td>36.7 ± 2.1</td>
</tr>
<tr>
<td><strong>Glucose-electrolyte (outdoor)</strong></td>
<td>35.9 ± 2.6</td>
</tr>
</tbody>
</table>

Values are mean ± SD  * Higher (p < 0.05)  ** (n = 10)
Table 7.3. Oxygen uptake during treadmill running at 80% $\dot{V}O_2_{max}$ for 60 min, at min 9 - 10 (80% RE1) and at min 59 - 60 (80% RE2), whilst ingesting either no fluid, 6% glucose-electrolyte or placebo electrolyte solutions (n = 15).

<table>
<thead>
<tr>
<th></th>
<th>VO2 (ml.kg⁻¹.min⁻¹)</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RE1</td>
<td>RE2</td>
</tr>
<tr>
<td>No fluid</td>
<td>43.7 ± 2.8</td>
<td>*45.2 ± 2.5</td>
</tr>
<tr>
<td>Glucose-electrolyte</td>
<td>42.5 ± 2.4</td>
<td>*45.1 ± 3.3</td>
</tr>
<tr>
<td>Placebo-electrolyte</td>
<td>43.7 ± 3.8</td>
<td>*45.6 ± 4.4</td>
</tr>
</tbody>
</table>

Values are mean ± SD * Higher (p < 0.05)
Figure 7.2. Lung ventilation ($\dot{V}_E$) during running at 10.8 km.h$^{-1}$ pre (RE1) and post (RE2) prolonged running bouts (60 min 80% $\dot{V}O_2_{\text{max}}$), whilst ingesting no fluid, glucose-electrolyte or placebo-electrolyte solutions ($n=15$). Values are mean ± SD. * Higher ($p < 0.05$)
Figure 7.3. Lung Ventilation ($\dot{V}_E$) during minutes 9-10 (80%RE1) and 59-60 (80%RE2) of prolonged running bouts (60 min 80% $\dot{V}O_{2max}$), whilst ingesting no fluid, glucose-electrolyte or placebo-electrolyte solutions ($n$ = 15). Values are mean ± SD. * Higher ($p < 0.05$)
The R-value decreased significantly during RE2 for all treatments. The 80%RE2 R-value was lower (NS) during the D run and significantly lower in the G-E and P-E running bouts. The margin of the decrease in R-values was significantly greater for the RE2 compared to the 80%RE2 runs (Tables 7.4 and 7.5).

Heart rates (HR) were significantly higher during both 80%RE2 and RE2 when compared to RE1 and 80%RE1 (Table 7.6). Verification that the stress was similar in the G-E and P-E treatments can be deduced from the fact that both mean HR and VO$_2$ were almost identical. However, both the D and the G-E-O treatments resulted in significantly higher HR during RE2 and 80%RE2 than the G-E and P-E treatments, suggesting that there was some difference in the physiological stress being experienced.

The rate of perceived exertion (RPE) increased significantly during both 80%RE2 (from 11 to 17) and RE2 (from 9 to 13) compared to 80%RE1 and RE1 respectively, with no difference (NS) between treatments (Table 7.7).

There were no differences (NS) in plasma glucose concentration between RE1 and RE2 during the D and P-E exercise bouts. The plasma glucose concentration was significantly higher during RE2 for both the G-E and G-E-O treatments. The outdoor treatment resulted in the highest plasma glucose concentrations (Table 7.8).
Plasma lactate concentration was higher during RE2 for all treatments and was found to be significantly higher during both G-E treatments (Figure 7.4). The highest mean group concentration of plasma lactate (4 ±1.4 mmol.l\(^{-1}\)) was post the outdoor run (hot, humid environment).
Table 7.4. The R-values during treadmill running at 10.8 km.h⁻¹ (65% \( \dot{V}O_{2\text{max}} \)) pre (RE1) and post (RE2) prolonged high-intensity running bouts (80% \( \dot{V}O_{2\text{max}} \) 60 min), whilst ingesting either no fluid, 6% glucose-electrolyte or placebo-electrolyte solutions (n = 15).

<table>
<thead>
<tr>
<th>No fluid</th>
<th>RE1</th>
<th>*0.90 ± 0.05</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose-electrolyte</td>
<td>0.96 ± 0.04</td>
<td>*0.91 ± 0.04</td>
</tr>
<tr>
<td>Placebo-electrolyte</td>
<td>0.95 ± 0.04</td>
<td>*0.89 ± 0.04</td>
</tr>
<tr>
<td><strong>Glucose-electrolyte (outdoor)</strong></td>
<td>0.95 ± 0.07</td>
<td>*0.90 ± 0.05</td>
</tr>
</tbody>
</table>

Values are mean ± SD

* Lower (p < 0.05) ** n = 10
Table 7.5. The R-values during min 9-10 (80%RE1) and min 59-60 (80%RE2) of prolonged high-intensity running bouts (80% $\dot{V}O_{2\text{max}}$ 60 min), whilst ingesting either no fluid, 6% glucose-electrolyte or placebo-electrolyte solutions (n = 15).

<table>
<thead>
<tr>
<th></th>
<th>80%RE1</th>
<th>80%RE2</th>
</tr>
</thead>
<tbody>
<tr>
<td>No fluid</td>
<td>0.96 ± 0.02</td>
<td>0.95 ± 0.03</td>
</tr>
<tr>
<td>Glucose-electrolyte</td>
<td>0.98 ± 0.03</td>
<td>*0.95 ± 0.03</td>
</tr>
<tr>
<td>Placebo-electrolyte</td>
<td>0.97 ± 0.03</td>
<td>*0.94 ± 0.03</td>
</tr>
</tbody>
</table>

Values are mean ± SD  * Lower (p < 0.05)
Table 7.6. Heart rate (HR) during running at 10.8 km.h\(^{-1}\) pre (RE1) and post (RE2) prolonged high-intensity running bouts (80% \(\dot{V}O_2\text{max}\) 60 min) and at 9-10 min (80%RE1) and 59-60 min (80%RE2), whilst ingesting either no fluid, 6% glucose-electrolyte or placebo-electrolyte solutions (n = 15).

<table>
<thead>
<tr>
<th></th>
<th>HR (b.min(^{-1}))</th>
<th>RE1</th>
<th>RE2</th>
<th>80%RE1</th>
<th>80%RE2</th>
</tr>
</thead>
<tbody>
<tr>
<td>No fluid</td>
<td>148 ± 13</td>
<td>*164 ± 12</td>
<td>167 ± 6</td>
<td>*175 ± 6</td>
<td></td>
</tr>
<tr>
<td>Glucose-electrolyte</td>
<td>147 ± 11</td>
<td>*160 ± 11</td>
<td>164 ± 8</td>
<td>*171 ± 8</td>
<td></td>
</tr>
<tr>
<td>Placebo-electrolyte</td>
<td>147 ± 11</td>
<td>*161 ± 12</td>
<td>161 ± 9</td>
<td>*170 ± 11</td>
<td></td>
</tr>
<tr>
<td><strong>Glucose-electrolyte outdoors</strong></td>
<td>146 ± 12</td>
<td>*168 ± 12</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values are mean ± SD  
* Higher (p < 0.05) ** n = 10
Table 7.7. The rate of perceived exertion pre, during and post prolonged running bouts (60 min 80% $\dot{V}O_{2\text{max}}$) whilst ingesting no fluid, glucose-electrolyte (G-E) or placebo-electrolyte (P-E) solutions ($n = 15$).

<table>
<thead>
<tr>
<th></th>
<th>RE1</th>
<th>RE2</th>
<th>80%RE1</th>
<th>80%RE2</th>
</tr>
</thead>
<tbody>
<tr>
<td>No fluid</td>
<td>9.8 ± 1.8</td>
<td>13.3 ± 2.2*</td>
<td>11 ± 2.1</td>
<td>17.3 ± 2.4*</td>
</tr>
<tr>
<td>G-E</td>
<td>9.9 ± 1.9</td>
<td>12.7 ± 2.3*</td>
<td>10.9 ± 1.8</td>
<td>17.2 ± 2.1*</td>
</tr>
<tr>
<td>P-E</td>
<td>10 ± 1.4</td>
<td>13.2 ± 2.7*</td>
<td>11.2 ± 1.9</td>
<td>17.3 ± 2.3*</td>
</tr>
<tr>
<td>G-E outdoor</td>
<td>9.5 ± 2.2</td>
<td>13 ± 2.6*</td>
<td>10.8 ± 2.0</td>
<td>17.5 ± 2.1*</td>
</tr>
</tbody>
</table>

Values = mean ± SD
RE1 = 10 min at 10.8 km.h$^{-1}$ (65% $VO_{2\text{max}}$) pre the prolonged running bout.
RE2 = 10 min at 10.8 km.h$^{-1}$ post the prolonged running bout.
80%RE1 = during min 9-10 of the prolonged running bout.
80%RE2 = during min 59-60 of the prolonged running bout.
* Higher ($p < 0.01$)
Table 7.8. Plasma concentration of glucose (GL) during treadmill running at 10.8 km.h\(^{-1}\) (65% \(\dot{V}O_2_{max}\)) pre (RE1) and post (RE2) 60 min at 80% \(\dot{V}O_2_{max}\), whilst ingesting either no fluid, 6% glucose-electrolyte or placebo-electrolyte solutions (n = 15).

<table>
<thead>
<tr>
<th></th>
<th>GL (m.mol.l(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RE1</td>
</tr>
<tr>
<td>No fluid</td>
<td>5.1 ± 0.5</td>
</tr>
<tr>
<td>Glucose-electrolyte</td>
<td>5.0 ± 0.8</td>
</tr>
<tr>
<td>Placebo-electrolyte</td>
<td>4.7 ± 0.6</td>
</tr>
<tr>
<td><strong>Glucose-electrolyte outdoor</strong></td>
<td>5.1 ± 0.4</td>
</tr>
</tbody>
</table>

Values are mean ± SD  
* higher (p < 0.05)  
** n = 10
Figure 7.4. Plasma lactic acid concentration during running at 10.8 km.h⁻¹ pre (RE1) and post (RE2) 60 min running bouts at 80% VO₂max, whilst ingesting no fluid, glucose-electrolyte or placebo-electrolyte solutions (n = 15). Outdoor run n = 10. (mean ± SD) * Higher (p < 0.05)
Plasma sodium concentration during RE2 was significantly higher for the D and G-E-O treatments, with no change during the G-E treatment, but was significantly lower for the P-E treatment (Table 7.9). Plasma sodium concentration was significantly higher following the D treatment compared to the G-E and P-E treatments (ANOVA).

Plasma potassium concentrations were significantly higher during RE2 for all treatments, with no differences found between treatments (NS). The increase was by an identical margin for the D, G-E and P-E treatments and the highest mean group concentration during RE2 was following the G-E-O treatment (Figure 7.5).

The plasma ammonia concentration increased by a significant amount for all treatments during RE2, with no differences (NS) between treatments (Figure 7.6).

There were no measured changes (NS) in the concentration of haemoglobin and haematocrit either between RE1 and RE2 or between treatments. Plasma volume reduced slightly during the D trial (-1.4%) and expanded by similar margins during the G-E (+0.9%), P-E (+0.9%) and G-E-O (+1.4%) trials.

The rectal temperature was significantly higher during RE2 for all treatments (n = 7). There was no difference between indoor treatments (NS) but the rectal temperature was observed to be significantly the highest following the G-E-O treatment (Table 7.10).

During the 60 minute running bouts (80%VO2max) the subjects ingested identical individual amounts of fluid (group mean = 1048 ± 257 ml) during G-E, P-E and G-E-O treatments. Post
exercise body weight was significantly lower for all treatments, but mean body weight loss was only 0.8% - 0.9% post the G-E, P-E and the G-E-O treatments. The largest loss in body weight (2.7%) was following the D treatment and this was significantly lower (ANOVA) than body weight loss for the other three treatments.
Table 7.9. Plasma concentration of sodium (Na⁺) during treadmill running at 10.8 km.h⁻¹ (65% VO₂max) pre (RE1) and post (RE2) 60 min running bouts at 80% VO₂max, whilst ingesting either no fluid, 6% glucose-electrolyte or placebo-electrolyte solutions (n = 15).

<table>
<thead>
<tr>
<th></th>
<th>Na⁺ (mmol.l⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RE1</td>
</tr>
<tr>
<td>No fluid</td>
<td>138.3 ± 1.8</td>
</tr>
<tr>
<td>Glucose-electrolyte</td>
<td>138.2 ± 2.1</td>
</tr>
<tr>
<td>Placebo-electrolyte</td>
<td>138.2 ± 2.2</td>
</tr>
<tr>
<td>**Glucose-electrolyte</td>
<td>138.1 ± 2.0</td>
</tr>
</tbody>
</table>

Values are mean ± SD  
* Higher (p < 0.05)  
** Lower (p < 0.05)  
*** n = 10
Figure 7.5. Plasma potassium during running at 10.8 km.h⁻¹, pre (RE1) and post (RE2) 60 min running bouts at 80% \( \dot{V}O_{2\text{max}} \) (n = 15).

Legend
- **RE1**
- **RE2**
- NF = No fluid
- G-E = Glucose - electrolyte
- P-E = Placebo - electrolyte
- G-E-O = Glucose electrolyte (Outdoors)
Figure 7.6 Plasma ammonia (NH₃) during running at 10.8 km.h⁻¹, pre (RE1) and post (RE2) 60 min running bouts at 80% $\dot{V}O_{2\text{max}}$ (N = 15).

Legend

- ■ RE1
- ○ RE2

NF = No Fluid
G-E = Glucose - electrolyte
P-E = Placebo - electrolyte
G-E-O = Glucose - electrolyte (Outdoors)
Table 7.10. Rectal temperature ($T_{rc}$) during treadmill running at 10.8 km.h\(^{-1}\) (65\% $\dot{V}O_2_{max}$) pre (RE1) and post (RE2) prolonged high-intensity running bouts (80\% $\dot{V}O_2_{max}$, 60 min), whilst ingesting either no fluid, 6\% glucose-electrolyte or placebo-electrolyte solutions (n = 15).

<table>
<thead>
<tr>
<th></th>
<th>RE1</th>
<th>RE2</th>
</tr>
</thead>
<tbody>
<tr>
<td>No fluid</td>
<td>37.4 ± 0.3</td>
<td>*38.3 ± 0.6</td>
</tr>
<tr>
<td>Glucose-electrolyte</td>
<td>37.3 ± 0.2</td>
<td>*38.1 ± 0.6</td>
</tr>
<tr>
<td>Placebo-electrolyte</td>
<td>37.4 ± 0.4</td>
<td>*38.2 ± 0.7</td>
</tr>
<tr>
<td><strong>Glucose-electrolyte outdoor</strong></td>
<td>37.3 ± 0.3</td>
<td>*38.8 ± 0.7</td>
</tr>
</tbody>
</table>

Values are mean ± SD  
* Higher (p < 0.05)  
** n = 10
7.4 Discussion

The lack of change in \( \text{VO}_2 \text{max} \), pre-treatment \( \text{VO}_2 \) (RE1) and submaximal HR during RE1 implies that no significant training effect had occurred in the cardiovascular-respiratory system across the short duration testing period (5 weeks for each subject). The subjects had been requested not to train but to remain active.

The \( \text{VO}_2 \) during RE2 was significantly higher than during RE1 for all treatments (relative intensity: 66% vs 73% \( \text{VO}_2 \text{max} \)), but there were no significant differences in \( \text{VO}_2 \) between treatments during either RE1 or during RE2 in this present study. The mean group \( \text{VO}_2 \) during RE2 increased by 3.4 to 4.7 ml.kg\(^{-1}\) min\(^{-1}\) (Table 7.2), with the greatest deterioration in running economy experienced following the D running bout (mean increase in \( \text{VO}_2 = 4.8 \text{ ml.kg}^{-1}\text{ min}^{-1} \)).

Chryssanthopoulos et al (1994) found evidence of oxygen drift and reported similar increases in relative exercise intensity (from 69% to 76% \( \text{VO}_2 \text{max} \)) during prolonged running (121 minutes). Additionally, Nicol et al (1991a) examined the influence of the fatiguing effects of running a marathon on running economy and also evidenced large decrements in running economy (pre versus post marathon running economy = \( 35.8 \pm 2.3 \) vrs \( 45.2 \pm 6.8 \text{ ml.kg}^{-1}\text{ min}^{-1} \)) at similar running speeds to the 10.8 km.h\(^{-1}\) investigated in this present study.

The \( \text{VO}_2 \) during RE1 (10.8 km. h\(^{-1}\)) for all treatments expressed as ml. min\(^{-1}\) (approximately 2230) was similar to the range of \( \text{VO}_2 \) (2220 - 2460) reported by Lees et al (1995) for experienced runners during treadmill running at 10.5 km. h\(^{-1}\). However, the \( \text{VO}_2 \) during RE1
(65.7% VO$_{2\text{max}}$) was lower (36 vs 39 ml.kg$^{-1}$ min$^{-1}$) than the VO$_2$ during treadmill exercise at 10.3 km. h$^{-1}$ (62% VO$_{2\text{max}}$) reported by Tarnoposky et al (1990) for trained male subjects (VO$_{2\text{max}}$ = 62 ml.kg$^{-1}$ min$^{-1}$).

As already stated, I found no differences between treatments in VO$_2$ (during RE2) in this present study. This is in agreement with the findings of others (Hargreaves and Briggs, 1988; Snow et al, 1994; McConell et al, 1994), who reported no difference in VO$_2$ between CHO-electrolyte and placebo trials during prolonged exercise (65% - 70% VO$_{2\text{max}}$). In a study involving prolonged cycling exercise for similar duration and intensity, Below et al (1995) also found no difference in VO$_2$ between CHO or no CHO fluid ingestion treatments. Likewise, Mitchell et al (1989) found no differences between trials (water placebo vs 6% CHO vs 12% CHO vs 18% CHO) in VO$_2$ during cycling for 105 minutes at 70% VO$_{2\text{max}}$.

Additionally, Blomstrand et al (1995) found no difference between conditions (6% CHO+BCAA or 6% CHO or flavoured water) in the VO$_2$ during 60 min cycling exercise at 75% VO$_{2\text{max}}$.

In this present study I found that $V_E$ was significantly higher during both RE2 and 80%RE2 compared to RE1 and 80%RE1 respectively for all treatments (Figures 7.2 and 7.3), but there were no differences (NS) between treatments for either. Franch et al (1994) conducted a study involving 36 trained runners (VO$_{2\text{max}}$ = 54.8 ml.kg$^{-1}$ min$^{-1}$) completing a run to exhaustion (36.1 ±1.4 min) at 86.5% VO$_{2\text{max}}$, and found VO$_2$ increased gradually from the 10th minute until exhaustion. They also reported a positive correlation for VO$_2$ drift with the increase in $V_E$ ($r = 0.73; p < 0.001$), whereas in this present study I found significant differences but low correlations between changes in VO$_2$ and $V_E$ (RE1 vs RE2). Pate et al
(1992) found that $V_E$ was an important variable when estimating RE in average to good runners. Pederson et al (1994) conducted a study to examine the effects of training on the RE (RE at 85% $VO_2\text{max}$) of trained athletes (same subjects as Franch et al, 1994). They found that RE improved significantly ($p < 0.01$) with training by as much as 3.3% and this coincided with a significantly reduced $V_E$ ($r = 0.64; p < 0.0001$). The above findings, including this present study, indicate that $V_E$ may be an important variable in the running economy template. However, in some conflict with this is the recent finding that diminution of the $VO_2$ slow component with training was attributable to factors other than $V_E$ (Womack et al, 1995), but a role for $V_E$ in the RE template cannot be ruled out.

In this present study the R-values decreased significantly during RE2 for all treatments when compared to RE1 (Table 7.4). Unlike Wilber and Moffat (1992), who found significant differences in mean respiratory exchange ratio to be associated with differences in blood glucose concentration with CHO ingestion during running at 80% $VO_2\text{max}$, I found no differences (NS) between treatments in the R-value either at 80% $VO_2\text{max}$ or during the determination of RE1 and RE2, even although there were significant differences in blood glucose concentrations between treatments in this present study. The higher R-value during RE2 following CHO ingestion compared to the placebo drink (Tables 7.4 and 7.5) suggest a slightly greater reliance on CHO as an energy source. The findings of Wilber and Moffat (1992) would support this, but others would disagree (Williams et al, 1990). However, it is noted that the Williams et al study (1990) involved subjects under racing conditions which can be quite different from constant pace running. Below et al (1995) and Blomstrand et al (1995) also found no difference in R-value for CHO and no CHO fluid ingestion during prolonged exercise (50 and 60 min) at 80% $VO_2\text{max}$ and 75% $VO_2\text{max}$ respectively. However, I
did find that the R-values were maintained at a higher value during the 80% \( \text{VO}_2 \text{max} \) relative intensity run compared to the significant decline observed at 65% \( \text{VO}_2 \text{max} \) between RE1 and RE2, suggesting a greater rate of CHO oxidation (Lamb, 1984) when running at the higher relative intensity. Nevertheless the R-values have to be interpreted with some caution. For instance, there is the uncertainty of the contribution of amino acid oxidation to \( \text{VO}_2 \) and \( \text{VCO}_2 \) during prolonged exercise (Hawley et al., 1992). Also, respiratory exchange ratio gives only an indirect estimation of muscle substrate metabolism, since the gas exchange over the lungs represents a mean for the whole body (Jansson, 1982). It was interesting to find that the deterioration in RE was less during running at 80% \( \text{VO}_2 \text{max} \) compared to the deterioration found during running at the common speed of 10.8 km.h\(^{-1}\) (65% \( \text{VO}_2 \text{max} \)). The R-values (tables 7.4 and 7.5) suggest a higher CHO oxidation during running to be associated with a better maintenance of RE during prolonged running, because the margin of decrease in the R-values was significantly greater for the RE2 compared to the 80%RE2 running bouts.

As anticipated the RPE was higher (\( p < 0.01 \)) during RE2 vs RE1 and 80%RE2 vs 80%RE1, but there were no differences between treatments. Williams et al (1990) also reported that RPE during CHO and Placebo trials was not significantly different. This conflicts with the findings of Wilber and Moffat (1992) who reported the mean RPE during prolonged running (\( > 60 \) min) at 80% \( \text{VO}_2 \text{max} \) was less pronounced (14.5 vs 15.4; \( p < 0.05 \)) when ingesting CHO compared to a placebo (artificially flavoured water).

As expected, the HR were significantly higher during RE2 and 80%RE2 compared to RE1 and 80%RE1 respectively. In a similar type of study, monitoring RE pre and post prolonged running at 80% \( \text{VO}_2 \text{max} \), Xu and Montgomery (1994) reported slightly lower HR (165 b.min\(^{-1}\))
than those found in this present study (Table 7.6) during the latter stages of their prolonged run (90 min). The difference between findings could be partly attributed to the cooler environment in which their testing was undertaken (1.3°C - 3.9°C) compared to this present study (laboratory: 22°C - 23°C / outdoor: 25°C -35°C).

In agreement with others I found no difference in HR between G-E and P-E treatments throughout testing. Snow et al (1994), McConell et al (1994) and Blomstrand et al 1995) also found no difference in HR between trials (CHO-electrolyte vs placebo) during prolonged submaximal exercise (65% - 75% VO₂ max). This is consistent with the earlier findings during 80 - 90 minutes of cycling at 70% VO₂ max (Hargreaves and Briggs, 1988; Rehrer et al, 1992).

The findings of this present study are in agreement with the studies by Hamilton et al (1991) and Below et al (1995) in that fluid replacement significantly attenuates the increase in HR during prolonged exercise when compared to dehydrative exercise (Table 7.6). Significantly higher HR were recorded during RE2 in this present study during the D run compared to both the G-E and P-E bouts of running. Additionally, the cardiovascular problems associated with exercise in the heat and humidity were further evidenced, as the highest HR during RE2 was recorded post the outdoor run (25 - 35°C; 66% -77% humidity).

In this present study the rectal temperatures were significantly higher during RE2 for all treatments, with no difference between treatments tested in the laboratory (thermoneutral environment). However, the rectal temperature was observed to be significantly higher during RE2 post the G-E-O run compared to after the G-E run. Fluid consumption during exercise is known to reduce the rise in core temperature during physical exercise (Pitts et al, 1944;
Bacharach et al, 1994). For example, Gisolfi and Copping (1974) have reported a beneficial effect of fluid replacement on attenuating hyperthermia during prolonged moderate intensity exercise. This present study did not find significant differences in rectal temperature as a result of either fluid or no fluid replacement for the treatments in the thermoneutral environment, although rectal temperature was slightly higher as a result of dehydrative exercise (Table 7.10). However, Gisolfi and Copping conducted their treadmill exercise in the heat and this may explain differences in findings. This present study found that rectal temperature was significantly higher with G-E fluid replacement during RE2 following exercise in the hot and humid environment compared to the thermoneutral laboratory (38.8 vs 38.1°C). The two treatments with the highest \( T_r \) (no fluid & G-E outdoors) also produced the highest plasma Na\(^+\) concentrations (Table 7.9). This increase in Na\(^+\) concentration is thought to alter skin blood flow and to retard sweating, which can therefore reduce heat loss from the body (Fortney et al, 1985). It is unfortunate that the present study did not also investigate the influence of no fluid replacement with fluid replacement on rectal temperature and RE following exercise in a hot and humid environment.

Neary et al (1993) designed a study to determine whether core temperature was related to the elevated post-exercise oxygen consumption. Their subjects had a similar physiological profile \( (\text{VO}_2 \text{max} = 57.5 \text{ ml.kg}^{-1} \text{ min}^{-1}) \) to the subjects in this present study, and the study design likewise included 60 minutes of exercise (cycling/swimming) but at a relative intensity of 63% \( \text{VO}_2 \text{max} \). They reported a post exercise (cycling) rectal temperature of 38°C which corresponds to the rectal temperature following the three thermoneutral treatments (D, G-E and P-E) in this present study (Table 7.10). Their results suggested that "the difference in core temperature changes were related to the metabolic processes occurring in the body for the
exercise being performed”. However, Neary et al (1993) caution against the causal quantification of excess VO₂ due to temperature alone, as others (Bahr, 1992; Elliot, Glodberg and Keuhl, 1992) have questioned the validity of such estimates because, for example, muscle temperature has been shown to change independent of rectal temperature (Saltin and Hermansen, 1966). In this present study significantly higher rectal temperatures were recorded during RE2 after the outdoor run (38.8 ±0.7°C) compared to the indoor run (38.1 ±0.7°C), but both conditions resulted in identical worsening of running economy. This finding adds weight to the above recommended caution by Neary et al (1993).

It is well known that blood volume can be altered in response to physical exercise (Convertino, 1991). In this present study the mean plasma volume did not change (NS) during RE2 compared to RE1 (-1.4% for the D treatment; +0.9%, +0.9% and +1.4% for the G-E, P-E and G-E-O respectively). These changes are very close to those found in a study by Chryssanthopoulos et al (1994) involving more than 2 hours of treadmill running with 6.9% CHO fluid ingestion (2 ml.kg⁻¹BW every 5 km), when the mean change in plasma volume was 0.9% (±1.7%) to -0.5% (±2%) in their study. For this present study plasma volume changes were calculated on the basis of measurements of hematocrit and hemoglobin (Dill and Costill, 1974). For these calculations to be accurate, minimal red cell destruction or loss must have taken place during the exercise (and the mean cell volume must not have altered). That there is minimal red cell destruction even during very prolonged exercise has been suggested by several authors (Dickson et al, 1982; Steenkamp et al, 1986). In a study (Fallowfield, 1995) at similar exercise intensities and durations to this present study, plasma volume did not change (decrease by 1% - 3.5%; NS). Also, Below et al (1995) have provided some evidence to indicate that the influence of a large fluid replacement (1330 ±60 ml) on
maintaining plasma volume during prolonged high-intensity exercise is dependent on its CHO content, fluid with CHO (6%) was effective and fluid without CHO was not effective, but the mechanism of this response is unclear in their paper. This present study found no difference in plasma volume (+0.9%) and body weight (-0.9%) during RE2 between G-E and P-E trials and therefore does not support an influence of CHO content on better maintaining plasma volume compared to no CHO, because electrolyte content and fluid volume were constant.

Previous studies have shown that intensive exercise is associated with alterations in plasma (and muscle) electrolyte concentrations, as during prolonged exercise there is a continuous release of K⁺ ions from working muscle (Sahlin and Broberg, 1989). Consistent with other investigations (Green et al., 1993; Lindinger and Sjogaard, 1991; McKenna et al., 1993) this present study has demonstrated that prolonged exercise results in a significant increase (10 - 16%) in plasma K⁺ concentration. Chryssanthopoulos et al (1994) also found similar increases (4.7 to 5.4 mmol.l⁻¹) during prolonged running at a similar exercise intensity.

In this present study the mean group plasma K⁺ concentrations were significantly and identically higher during RE2 (5.3 mmol.l⁻¹) compared to RE1 (4.8 mmol.l⁻¹) for all treatments in the thermoneutral environment (Figure 7.5). Robinson et al (1995) similarly found no significant differences in the increases in K⁺ concentrations in no fluid vs fluid (water) trials (60 min cycling at 80% VO₂max). The rise in plasma K⁺ concentration was greatest during RE2 (5.6 mmol.l⁻¹) following the outdoor run (G-E-O), suggesting that environmental factors may play a role in plasma K⁺ concentrations. Others have also reported large increases ( > 20%) in plasma levels of K⁺ during prolonged submaximal exercise.
(Fallowfield et al., 1995; Rama et al., 1994) and during short-term maximal exercise (Boulay et al., 1995). This rise of K⁺ concentration is both well documented and closely related to the intensity of the exercise (Hazeyama and Sparks, 1979; Medbo and Sejersted, 1990; Wilkerson et al., 1982), suggesting that contracting muscles are the main source of raised plasma K⁺ concentration due to the increased electrical activity in the muscle cells (Sjogaard et al., 1985). Two other possible mechanisms responsible for the rise in plasma K⁺ concentrations with exercise are a decrease in plasma volume and a release of K⁺ from erythrocytes (Lindinger and Sjogaard, 1991). This efflux is opposed by cellular reuptake mediated by the sodium-potassium pump (Gullestad et al., 1995) and the consequences of the K⁺ efflux from the muscle cells may be numerous. Some of the consequences are discussed in the following paragraphs. Interestingly, a rise in K⁺ concentration in the circulation has been shown to facilitate respiratory drive during exercise (Band et al., 1985), contribute to vasodilation within the muscle (Scott et al., 1970) and stimulate heart rate (Lindinger and Sjogaard, 1991). Busse et al. (1992) found that VE and K⁺ changes were almost equal during incremental cycling, further supporting the hypothesis that K⁺ may act as a stimulus for exercise VE. It would seem reasonable to assume that such effects may lead to both increased oxygen demand and availability.

It has been repeatedly observed that muscle activity may lead to a net loss of cellular K⁺ in working muscles (Sjogaard, 1983; Everts et al., 1993; Everts and Clausen, 1994) and it is also well documented that during exercise, plasma K⁺ rises (Fenn, 1940; Sjogaard et al., 1985; McKenna et al., 1993). The progressive loss of K⁺ from working muscle cells - and distribution in the extracellular compartment - appears to be one of the factors causing the intensity of contraction to decrease (Fenn, 1940) and has recently been linked with an
impairment of the propagation of action potentials into the T-tubules (Clausen and Nielsen, 1994) and might constitute an important cause of fatigue (Lindinger and Sjogaard, 1991). A reduction in the membrane potential leads to a decrease in the amplitude of the action potential (Sandercock et al, 1985), which may result in a reduction in Ca\(^{+}\) release by the sarcoplasmic reticulum (Abramcheck and Best, 1989) and a decrease in the force of muscle contraction (Juel, 1988). In this context muscle K\(^{+}\) release may function as a fatigue safety mechanism which prevents the muscle cell from complete homeostatic failure (Lindinger and Sjogaard, 1991). Additionally, it is tempting to speculate that if the optimisation of motor unit recruitment patterns has been interfered with, this might account for some of the increase in exercise aerobic demand. It would be interesting to investigate the upper physiological limit of extracellular K\(^{+}\) concentration compatible with normal motor activity and the relationship between K\(^{+}\) accumulation in the extracellular space and specific alterations in muscle metabolic pathways (Marcos and Ribas, 1995). For instance, Tsintzas et al (1993) stated:

Moritani (1980) demonstrated that an abrupt increase in integrated EMG, representing changes in motor unit recruitment and/or motor unit firing frequency, correlated significantly with oxygen consumption \( (r = 0.97; n = 36) \). Matsumoto et al (1991) speculated that a progressive recruitment of additional motor units might take place to compensate for the deficit in fatigued motor units. In favour of ion perturbations playing a crucial role was the finding of a very quick normalisation in recovery, which had a time course similar to the return of maximal force of the implicated muscles (Allen et al, 1989). Nicol et al (1991a) evidenced a muscular force impairment, confirmed by a deterioration in resistance to impact and an increase of energy expenditure when comparing pre and post marathon kinematic (e.g. braking/push-off;contact/flight time) and electromyographic running data. Thus, a loss in the
efficiency of the contractile mechanism (Nicol et al, 1991b; Nicol et al, 1991c) resulting from, for example, interference with membrane depolarisation and/or recruitment patterns of muscles or resulting from repeated stretching loads imposed on the leg muscles during prolonged runs seems viable. Carsten (1988) has shown that the action potential propagation velocity is decreased by an increased extracellular K\(^+\) concentration (animal study). The effect of K\(^+\) is due to the increased concentration on the extracellular side of the membrane, was associated with a considerable broadening of the action potential, and resulted in an increased number of inexcitable cells. Of interest was Carsten's finding that type 2 muscle fibres showed a higher sensitivity to K\(^+\) than type 1 muscle fibres. Further, the action potential propagation velocity was nearly independent of moderate changes in the Na\(^+\) gradient - but it must be remembered that this was an animal study. The observed change in force production with fatigue (Nicol et al, 1991a) warrants further research, considering the findings of another recent study by Johnston et al (1995) which found that strength training can improve RE. Thus, it would be interesting to investigate whether strength training can attenuate the loss in efficiency of the muscle contractile mechanism (Nicol et al, 1991b+c) and result in positive benefits to RE during prolonged high-intensity running.

At a given level of dehydration the Sodium (Na\(^+\)) in sweat (assuming water loss due to respiration is negligible - Morimoto et al, 1981) determines the volume of fluid mobilised from the intra-cellular fluid compartment, thereby determining the effective maintenance of circulating blood volume (Nose et al, 1988). This emphasises the importance of producing a more dilute sweat in the heat adaptation process. It can be assumed that the subjects in this present study, having lived and exercise in a hot and humid environment, were acclimatised and likely to have a more dilute sweat. Fallowfield et al (1995) found that plasma
concentrations of $N^+$ tended to remain constant during prolonged (70 - 115 min) endurance running both with and without fluid replacement. However, I found that plasma $Na^+$ concentration was significantly higher for the D and G-E-O treatments, with no change (ie. constant) during the G-E treatment, but significantly lower for the P-E treatment (Table 7.9). Below et al (1995) found that a small CHO fluid replacement (200 ±10 ml) during prolonged exercise (50 min at 80% $VO_2\text{max}$) resulted in significantly higher serum $N^+$ concentration when compared with a large fluid replacement (1330± 60 ml).

Different mechanisms have been proposed regarding the ergogenic effect of CHO ingestion. One possibility is that CHO feeding during prolonged exercise acts to spare muscle glycogen, thereby delaying fatigue (Hargreaves et al, 1984). Conversely, it has been suggested that exogenous CHO does not affect muscle glycogen utilisation (Fielding et al, 1985), but instead may improve performance by maintaining blood glucose at a critical point in endurance exercise when liver and muscle glycogen levels are low and the uptake of glucose by skeletal muscle is increased (Coyle et al, 1986). Prolonged exercise at 60% to 80% $VO_2\text{max}$ will reduce muscle glycogen, and this is believed to be a major contributor to the onset of fatigue (Bergstrom and Hultman, 1967; Hermansen et al, 1967). For example, Vollestad et al (1984) investigated glycogen depletion patterns during exercise (cycling) at 75% $VO_2\text{max}$ and found large depletion patterns after both 40 and 60 minutes. However, the subjects in this present study never became hypoglycaemic during any of the treatments (Table 7.8). This suggests that hypoglycaemia per se was not a cause of the deteriorating RE we observed. This is important because it has previously been demonstrated that low blood glucose concentration during prolonged exercise induces CNS distress (Wolfe et al, 1986), and may partly explain why RPE responses were similar with either G-E or P-E solutions.
Prolonged running performance at 80% VO$_{2\text{max}}$ has been shown to be enhanced (time to exhaustion: 115 vs 92 minutes; p < 0.05) when ingesting CHO compared to a placebo solution (Wilber and Moffat, 1992). Others have also found similar performance benefits when running at 80% VO$_{2\text{max}}$ by ingesting CHO solution vs placebo (McMurray et al, 1983; Sasaki et al, 1987). In contrast, some have failed to detect significant performance differences between CHO vs placebo ingestion during prolonged running (Riley et al, 1988; Powers et al, 1990; Williams et al, 1990). However, closer examination of the Williams et al (1990) study reveals that the overall performance was actually enhanced as a result of CHO consumption, because mean running speed was higher with CHO vs placebo throughout testing. Additionally, the study by Riley et al (1988) had a long duration of pre-exercise fast (21 hours) and this may partly explain why the endurance performance was greater (NS) with CHO vs placebo (106 vs 102 minutes).

The benefits of CHO ingestion (delayed fatigue + improved performance) become more apparent when muscle glycogen is low and reliance on blood glucose increases (Coggan and Coyle, 1988 & 1991; El Sayed et al, 1995). Significant (and important) results from a recent study by Blomstrand et al (1995) show that type 1-fibres were low in glycogen after 80 min cycling at 75% VO$_{2\text{max}}$, whereas most of the type 2-fibres contained high or medium amounts of glycogen. Interestingly, they suggest that their subjects were not able to recruit their type 2-fibres to any great extent, despite the fact that the type 1-fibres were low in glycogen and presumably affected by fatigue. Interestingly, Jansson ((1986) and Tarnopolsky et al (1990) have found that males engaged in prolonged submaximal (65% VO$_{2\text{max}}$) exercise showed a greater muscle glycogen utilisation (and a higher exercise RER) than equally trained and
nourished females. This gender difference in muscle glycogen utilisation is likely to be associated with a greater utilisation of lipids by the females (Holloszy and Coyle, 1984) and a greater exercise catecholamine response in males than females, because epinephrine enhances muscle glycogenolysis (Graham et al., 1986; Nygaard, 1986; Sanchez et al., 1980). An appealing further study could involve an investigation of RE during prolonged running bouts using equally matched male and female subjects (VO2 max, training and performance history, fibre type).

Fallowfield et al (1995) reported that glucose levels remain unchanged during no fluid and water ingestion trials during running at 70% VO2 max for 70 - 115 min. Fallowfield et al's data also indicate that abstaining from fluid (water) ingestion seems to enhance CHO oxidation and suppress fat oxidation. Additionally, Wright et al (1991) found that CHO consumption during prolonged exercise (70% VO2 max) also resulted in higher rates of CHO oxidation. In this present study I found no evidence of improved rates of CHO oxidation between treatments, possibly because of the shorter duration compared to Wright et al (80 vrs 200+ min). I found plasma glucose concentration was significantly higher for both the G-E treatments compared to P-E or D. This is in agreement with Blomstrand et al (1995), who found that concentrations of glucose during similar exercise duration (80 min) and intensity (75% VO2 max) were higher (p < 0.05) when CHO was supplied compared to no CHO. During high-intensity exercise a CHO solution containing up to 10% glucose could have a gastric emptying rate similar to that of plain water (Owen et al, 1986). Additionally, the net absorption of water through the small intestine epithelial cells is significantly accelerated by the active transport of glucose and sodium (Davies et al, 1987), both of which were constituents of the CHO solution used in this present study. Therefore, it was not surprising
that blood glucose concentration was significantly greater during the CHO trials compared to
the placebo trial. However, in this present study the higher plasma glucose concentrations (G-
E and G-E-O trials) were not associated with an attenuation of the deteriorating running
economy I observed after prolonged exercise.

The plasma lactate concentrations I observed during RE2 (Figure 7.4) did not exceed values
above 4 mmol.l⁻¹. However, during the 60 minute bouts of running at 80% VO₂ max subjects
are likely to have been above or close to their lactate thresholds. Recently, Below et al (1995)
found 75 ±1% VO₂ max to be a lactate threshold for the subjects in their study. This further
supports that the individual anaerobic threshold can range above lactate concentrations of 4
mmol.l⁻¹ (Stegmann and Kindermann, 1982). The present study also found no difference in
plasma lactate between treatments (CHO-electrolyte vs placebo) and this finding supports
other recent research (McConnell et al, 1994; Snow et al, 1994).

Snow et al's study (1994) on the effect of CHO-electrolyte ingestion on ammonia metabolism
demonstrated that CHO ingestion may attenuate plasma and muscle ammonia (NH₃)
accumulation during prolonged cycling exercise at 65% VO₂ max. This was in contrast to the
findings of this present study. Although I observed plasma NH₃ concentration increasing by a
significant amount for all treatments during RE2 (Figure 7.6), I found no difference in plasma
NH₃ between treatments (D; G-E; P-E; G-E-O). A possible reason for the difference may be
the difference in exercise intensity between the studies. However, some researchers (Tsintzas
et al, 1993; Davis et al, 1997) have observed a decrease in plasma free fatty acids with CHO
ingestion during prolonged running and cycling. Plasma free fatty acids compete for the same
carrier with plasma tryptophan, therefore a decreased plasma FFA would decrease plasma
free tryptophan and thus the free-tryptophan-to-branched chain amino acid (BCAA) ratio. This in turn would decrease 5-Hydroxytryptamine (5-HT) synthesis in the brain and could delay initiation of central mechanisms of fatigue. The 'central fatigue' hypothesis proposes that central (mental) fatigue occurs when there is an increase in the concentration of the neurotransmitter serotonin (5-HT) in the brain. Brain 5-HT is known to promote sleepiness, lethargy, and mood alterations, and there is some evidence that altered brain 5-HT can affect endurance performance (Davis, 1994). Brain 5-HT increases when the concentration of free tryptophan (f-TRP), an amino acid precursor of 5-HT, increases relative to the total concentration of BCAA in plasma. An increased ratio of f-TRP:BCAA facilitates transport of TRP into the brain that results in an increased synthesis of brain 5-HT (Davis, 1994). It is hypothesised that this will contribute to fatigue via effects on the CNS (Blomstrand et al, 1991), but others (Coggan and Swanson, 1992) have suggested that there is abundant data clearly indicating that fatigue during prolonged exercise is of peripheral, not central, origin. The more recent research by Van Hall et al (1994) also suggests that performance cannot be influenced by changes in the plasma tryptophan:BCAA concentration ratio. The findings of this present study indicate a possible link between deteriorating running economy and increasing plasma NH₃ concentration during prolonged exercise (Figure 7.6) and this warrants further investigation.

Coyle and Montain (1992) recommended that the exercising endurance athlete can meet both his/her CHO (i.e. 30-60 g.h⁻¹) and fluid needs by drinking 625-1250 ml.h⁻¹ of beverages containing 4-8% CHO. The subjects in this present study demonstrated a large fluid with 6% CHO intake (mean = 1048 ml during 60 min at 80% VO₂ max) and there were large individual differences (range = 750 - 1500 ml.h⁻¹). This is very close to the recommendations of Gisolfi
and Duchman (1992), who recommended 800 to 1600 ml.h⁻¹ of a 6 to 8% CHO solution (with 10 - 20 mEq Na⁺) during exercise. This suggests that the subjects could have maintained a large (around 800 ml) gastric volume (Noakes et al, 1991). Others have found that the capacity for fluid ingestion during running has tolerable limits (Williams et al, 1990; Chryssanthopoulos et al; 1994). Feelings of abdominal fullness were a problem for all of the subjects in this present study. The high ventilatory level of the subjects during the prolonged high-intensity running bouts (60 min at 80% VO₂max) might have impeded the absorption of some of the ingested fluid, which could be responsible for some digestive tract discomfort encountered (Mitchell et al, 1989). Recent research has demonstrated that trying to replace more than one litre per hour sweat losses during high-intensity exercise (60 min at 85% VO₂max) in a moderate environment (20°C, 60% RH) would not appear to induce beneficial physiological effects, and may impair exercise performance (Robinson et al, 1995). Future studies could focus on optimal rates of fluid replacement for maintaining or reducing the worsening running economy evidenced in this present study. Part of the problem could be that most runners do not train with frequent ingestion of fluids and serial administration to maintain volume has been highlighted as having importance in optimising fluid delivery (Vist and Maughan, 1994). Hence, other questions arise: Can runners be trained to consume larger amounts of fluid (ie. close to the rate that matches sweating)? Is it possible to remove the discomfort caused by high rates of fluid replacement via training with a repeated drinking design? As Millard-Stafford et al (1992) stated:

"Although scientific evidence is lacking, we believe that runners can improve their ability to consume larger fluid intakes during running."
Gastric emptying of fluids has been studied using techniques such as aspiration of gastric content after exercise (Neufer et al, 1989; Rehrer et al, 1990). It is possible that gastric emptying was delayed during the 60 min at 80%VO\textsubscript{2max} as more vigorous exercise (> 70 - 75% VO\textsubscript{2max}) slows fluid transit (Murray, 1987). Some of the factors that influence gastric emptying of fluids include volume, temperature and composition of the beverage (osmolality, pH and caloric content). Gisolfi and Duchman (1992) recommended a cool (5 - 15°C) fluid ingestion, but unfortunately the range of fluid temperature in this present study was 18 - 19°C and it is recognised that this should be improved upon. Water absorption from the small intestine is facilitated by glucose and Na\textsuperscript{+} (Murray, 1987). Recently Beckers et al (1994) observed that water and glucose intestinal absorption was high (5.3 ±2.2 ml.cm\textsuperscript{-1} hr\textsuperscript{-1} and 1.91 ±0.23 mmol.cm\textsuperscript{-1} hr\textsuperscript{-1} respectively) during prolonged cycling at 65% VO\textsubscript{2max}. However, I do acknowledge that it is difficult to know how much fluid to prescribe for ingestion during prolonged running to optimise performance, and this question begs further study. The recommendations of the American College of Sports medicine (100-200 ml per 2-3 km) have been criticised (Coyle and Montain, 1992), because according to this recommendation slow runners would drink too little and fast runners too much! In general most runners drink only about 500 ml of fluid per hour (Noakes et al, 1988; 1991).

The subjects in this present study experienced a 2.7% loss in body weight during the D treatment, probably mostly the result of sweating (Mitchel et al, 1972), reflecting a fluid loss in excess of the threshold beyond which exercise performance is impaired. It is well documented that fluid losses of as little as 2% of body mass can cause impairment of thermoregulation and performance (Armstrong et al, 1985; Williams, 1985; Jones et al, 1995). It is therefore important to ingest fluids as frequently and in volumes as large as
possible but without causing gastrointestinal distress (Burke and Read, 1989), impairment to performance (Hawley et al, 1994) or hyponatraemia (Noakes et al, 1994). Fluids should be cooled to hasten gastric emptying, and perhaps to improve body temperature control and to enhance palatability (Costill and Saltin, 1974). However, the finding in this present study that running economy deteriorated identically with or without fluid ingestion (Tables 7.2 and 7.3) was surprising and demands further investigation.

In this present study I observed significantly different physiological responses between prolonged running in a thermoneutral laboratory (22 - 23°C) compared to running outdoors in the heat and humidity (25 -35°C), but found no difference in VO₂ (RE2) between these very different environmental treatments. It is interesting that Decombaz et al (1994) reported that VO₂ was significantly lower (1.97 vs 2.09 l.min⁻¹;p < 0.05) during treadmill running (60% VO₂ max) in the heat (31°C) after CHO loading (82% for 2 days) compared to a low CHO (14%) diet. A possible future study could investigate the influence of CHO loading on RE during the latter stages of prolonged high-intensity running under different environmental conditions (heat and humidity versus the thermoneutral laboratory).

Coyle et al (1992) have shown that efficiency (oxygen cost) of cycling (50 - 70% VO₂ max) is positively correlated with the percentage of type 1 muscle fibres. However, Franch et al (1994) found no relationship between fibre type composition and VO₂ drift when subjects were running at a higher exercise intensity (86.5% VO₂ max). They suggested that lower exercise intensities might 'favour' a relationship between type 1 fibre content and the oxygen cost of running. Tsintzas et al (1994) found that a CHO-electrolyte fluid ingested immediately before and during a 70% VO₂ max run to exhaustion resulted in a sparing of
muscle glycogen in type I fibres and improved endurance capacity (27%), when compared with a placebo solution. In support of this, others have demonstrated that the ingestion of a CHO-electrolyte solution during 4h of recovery from prolonged running improves subsequent endurance capacity (Fallowfield et al: in Wong et al, 1995) and drinking a prescribed volume (more than twice ad libitum) of G-E solution (calculated to replace body fluid losses) during recovery after prolonged running restores endurance running capacity to a greater extent than ad libitum rehydration during 4h of recovery (Wong et al, 1995).

Below et al (1995) demonstrated that both large volume fluid replacement and CHO ingestion (6%), individually improved high-intensity cycling endurance performance - but the mechanism by which it does so was unclear. Fallowfield and Williams (1993) have shown that a high CHO diet restored endurance capacity within 22.5 hours of a prolonged run (70% VO$_{2\text{max}}$), whereas an isocaloric diet without additional CHO did not. It would be interesting to conduct a similar experimental protocol to that of Below et al (1995) to test both the effects of large and small dosage CHO and fluid repeated drinking replacement on RE during high-intensity exercise, and also to investigate RE 24 hours later employing similar methods of dietary manipulation (rapid glycogen resynthesis) to those of Fallowfield and Williams (1993). To the best of our knowledge there is little information on the return of RE during recovery from prolonged exercise, ie. facilitative recovery processes. If, to facilitate rapid muscle glycogen synthesis after prolonged submaximal exercise, CHO ingestion should commence immediately post-exercise (Sherman, 1992), why not commence replenishment during exercise itself using a very frequent ingestion of liquid CHO? The findings of Doyle and Sherman (1991) suggest that the rate of muscle glycogen synthesis might be further increased using such a design. However, it is worth noting that factors other than glycogen
depletion contribute to muscular fatigue and nutritional treatment is only part of the template for recovery of muscle function (Sherman and Wimer, 1991; Young and Davies, 1984).

This present study concurs with the findings of others (Coggan and Coyle, 1988; 1991; 1995) that CHO ingestion does not seem to enhance CHO oxidation during the first 60 - 70 minutes of exercise as evidenced by the R-values in tables 7.4 and 7.5. It is interesting that improvement in endurance performance can occur with CHO ingestion (Neufer et al, 1987) without an apparent maintenance in economy. It is accepted that supplementation with CHO solutions during intense endurance exercise to replace fluid losses, and to provide additional CHO in situations where endogenous reserves may become limiting, can improve both endurance capacity and performance. However, it was surprising to find that these performance benefits do not seem to be associated with maintaining running economy, as evidenced by the significantly worse running economy during RE2 for all treatments in this present study (Tables 7.2 and 7.3). If one could maintain economy, perhaps there would be further enhancement of performance.

A recent study (Nigg et al, 1995) has demonstrated kinematic differences between overground and treadmill running. However, the finding of no difference in VO2 during RE2 when comparing the G-E treatments (outdoors vs indoors) appears to lend some support to the conclusions of Dillman (1970) and Williams (1985). Dillman reviewed the literature on the energy cost (and kinematic) differences between overground and treadmill running and concluded that there were no outstanding differences between the two modes of running. Also, importantly for this present study involving both treadmill and outdoor running, Williams (1985) concluded that when significant differences were reported, they have
generally been for speeds greater than 5 m.s\(^{-1}\). In this present study the common RE speed was 3 m.s\(^{-1}\) and the mean group 80% VO\(_{2\text{max}}\) relative intensity speed was 3.5 m.s\(^{-1}\).

Another thought provoking factor - beyond the realms of this present investigation - is speculated, as follows. One could infer that the conservation and return of energy may play a role in the deteriorating RE evidenced in this present investigation. For example, although the total mechanical energy content in systems composed exclusively of masses and springs remains constant over time (Nigg and Anton, 1995), this may not be the case for the shoe-heel material during the course of a prolonged run. Consideration of a changing resonance effect on RE during the time course of a prolonged run lasting around 80 minutes - and the implications for both the conservation of and the return of energy would seem to be worthy of future investigation (McNeill et al, 1986; Denoth, 1986; Komi and Bosco, 1978). Indeed, the data from a study by Nicol et al (1991a) suggest that one effect of fatigue is a loss in the recoil characteristics of muscles, and this was associated with significant changes in metabolic data reflecting deteriorating RE following prolonged running in their study.

The findings of this present study provide further supportive evidence that after prolonged running at 80% VO\(_{2\text{max}}\) for 60 minutes a significant deterioration ( \(> 3 \text{ ml.kg}^{-1} \text{ min}^{-1}\)) in RE occurs at the common speed of 10.8 km.h\(^{-1}\) (65% VO\(_{2\text{max}}\)). This negative change in RE following prolonged high-intensity running appears to happen to the same extent irrespective of no fluid or fluid ingestion, regardless of whether the fluid contains CHO (6%) and electrolytes or electrolytes, and without regard for environment (thermoneutral vs hot and humid). However, although RE (80%RE2) did deteriorate during the latter stage of the 60 minute runs at 80% VO\(_{2\text{max}}\), the decline was less than 3 ml.kg\(^{-1}\) min\(^{-1}\) for all treatments (D, G-
E and P-E). This indicates that higher relative intensity running (80% VO$_2$ max), which demands an increased contribution from CHO oxidative metabolism, may result in a very slight attenuation of the decline in RE more than during lower relative intensity running (65% VO$_2$ max). This observation is supported by the finding that the margin of the decrease in the R-values was greater for the RE2 compared to the 80%RE2 running bouts.

The significant deterioration in RE observed during running at 10.8 km.h$^{-1}$ (65% VO$_2$ max) post prolonged high-intensity running was accompanied by significant changes in the following: increased $V_r$; decreased R-values; greater heart rates; higher lactate, potassium and ammonia concentrations and higher rectal temperature.

It was speculated that part of the template responsible for deteriorating RE during prolonged high-intensity exercise could be associated with muscle force impairment and/or loss in the elastic recoil characteristics of muscles, resulting in a combined negative interference with the sequencing and efficiency of the contractile mechanism. Another possibility might be the serial recruitment of lower efficiency fast-twitch fibres demanding progressively greater O$_2$ consumption (Crow and Kushmerick, 1982; Wendt and Gibbs, 1973). Thus, this present study seems to have resulted in more questions than answers in my attempts to further understand running economy.
General Discussion

The principal aim of this present thesis was to examine the influence of either no fluid or repeated ingestion of an electrolyte solution with or without carbohydrate on the running economy - within prolonged running bouts - of young adult male Singaporeans. This information was important because energy limitations during prolonged exercise are associated with a deterioration in performance (Frederick, 1985; Mason et al, 1993). From the Review of Literature there is a general agreement that information on running economy within prolonged exercise sessions was both scant and limited (Daniels, 1985; Morgan, 1992; Wilber and Moffat, 1992). Whilst it was known that oxygen consumption increased (ie. reducing running economy) during prolonged running at a constant pace, the mechanisms remained unclear. Thus, the possibility of attenuating a worsening running economy was deemed worthy of study.

Within subject daily variation over three consecutive days was initially reported in Chapter 4. The daily variation range supported those who have previously and recently found day-to-day stability in running economy (Morgan et al, 1991; Hunt et al, 1995). However, a few of the subjects demonstrated daily variance and they were subsequently excluded from further studies. This was considered important in not confounding any change that may occur within single prolonged exercise bouts as a result of the duration or intensity of the exercise. Others have reported wide inter and intra subject daily variation in VO₂ during different modes of exercise (Thomas et al, 1993). The findings of this present study were cautiously speculated to be associated with the mechanical efficiency of negative work, temporal variance in the support phase of running and the ‘economics’ of generalised motor

Previous studies have suggested that respiratory muscle fatigue and increasing heart rate were mediators for increasing VO\textsubscript{2} (ie. deteriorating running economy) during prolonged exercise (Bye et al, 1984; Pate et al, 1992). The findings reported in Chapter 5 of the present study did not support this during prolonged running (60 min) at intensities up to 75\% VO\textsubscript{2}\text{max} in agreement with Vollestad et al (1984). Similarly, Womack et al (1995) and others (Aaron et al, 1992; Poole et al, 1991) have concluded that changing oxygen kinetics during prolonged exercise was not primarily attributable to increasing ventilatory or cardiac work.

Following on from this, the study described in Chapter 6 established that running economy did significantly deteriorate following 60 min of running at 80\% VO\textsubscript{2}\text{max}. This increase in oxygen consumption was accompanied by significantly lower R-values, indicating an increase in fat oxidation. In conflict with this interpretation were the findings of Gass et al (1983) and Sahlin et al (1990), who respectively reported a decrease in the R-value with no increase in VO\textsubscript{2} and an increase in VO\textsubscript{2} with no significant decrease in the R-value. However, both of these studies were at lower exercise intensities ( 75\% VO\textsubscript{2}\text{max}) and it was likely that there was a greater decrease in muscle glycogen concentration in the present study. A reduction in running economy has been shown to accompany lower muscle glycogen concentration (Kirwan et al, 1988).

It was interesting to record significantly higher plasma NH\textsubscript{3} concentrations post 60 min running at 80\% VO\textsubscript{2}\text{max} in study three (Chapter 6), because both a lower muscle glycogen
content and increasing systemic FFA concentrations have been speculated to elicit increased 5-HT concentrations resulting in a central fatigue effect (Broberg and Sahlin, 1988; Curzon et al, 1973; Parry-B billings et al, 1989). With this in mind, and in light of recent studies demonstrating that carbohydrate-electrolyte ingestion both spares muscle glycogen during prolonged and moderate to high-intensity running (Nicholas et al, 1994; Tsintzas et al, 1993 and 1994), and enhances endurance capacity performance during prolonged exercise at 80% \( VO_{2\text{max}} \) (Below et al, 1995; Wilber and Moffat, 1992), it was decided to explore the possibility of helping maintain running economy within prolonged high-intensity running bouts by maintaining levels of carbohydrate oxidation and suppressing fat oxidation (Coggan and Coyle, 1987; Maughan, 1991; Spencer et al, 1991).

The finding that running economy worsened significantly by a similar margin - after either no fluid or repeated ingestion of either glucose-electrolyte or electrolyte solutions, following 60 min running bouts at 80% \( VO_{2\text{max}} \) (Chapter 7) - was consistent with the findings of other recent research (Blomstrand et al, 1995; McConnell et al, 1994). Additionally, this present study resulted in significant increases (10% - 16%) in plasma \( K^+ \) following 60 min running at 80% \( VO_{2\text{max}} \), in agreement with other recent findings (Green et al, 1993; Chryssanthopoulos et al, 1994; Robinson et al, 1995).
Conclusion

Thus, as an overall thesis conclusion the following combination of metabolic and mechanical factors are suggested as underlying mechanisms for the deterioration in RE after 60 minute running bouts at 80% $\text{VO}_{2\text{max}}$. Some would demand either a central or a peripheral influencing component. My view - based on my findings and the review of literature - is that both central and peripheral mechanisms have a role to play when RE deteriorates during prolonged, high-intensity exercise. Both changes in muscle fibre recruitment and the sequencing and firing of motor units associated with excitation/contraction coupling interference from the increased extracellular K+ that I found seem plausible. Changes in both force generating capacity and gait timing characteristics (braking, support, propulsion) have been shown by others (Nicol et al, 1991a, b & c) to be worsened by a declining elastic return of energy from tissues. Add to this the possibility of some impairment of central motor drive as evidenced by the increased NH$_3$ concentration I found (Chapters 6 and 7). Also, variance in kinematics (gait cycle timing) associated with fatigue that has been demonstrated by others (Nicol et al, 1991a, b & c). I was unable to prevent or attenuate the deteriorating RE following the prolonged bouts of high-intensity running. The ingestion of glucose-electrolyte solutions appears to have made no difference to the deteriorating RE in my studies, that is the increase in oxygen consumption toward the end of prolonged heavy exercise. Despite the tenability of the findings of this thesis, the association between different fluid ingestions and running economy during prolonged intense running bouts should be interpreted cautiously due to the small sample size. However, the fifteen subjects involved in the final study (Chapter 7) were also involved in studies one, two and three (Chapters 4, 5 and 6), and this should be viewed as a strength of the overall study. More interdisciplinary research is required to clarify the mechanisms responsible for a deteriorating running economy during
prolonged high-intensity exercise, and this could be addressed by investigating both muscle function and running economy pre and post prolonged, high-intensity exercise bouts.
References


Referenced 196


References


References


References


Elftman H (1940) The work done by muscles in running. American Journal of Physiology 129: 672


Gisolfi CV and Duchman SM (1992) Guidelines for optimal replacement beverages for


17: 115-155.

Physiology 17: 675-676.

Goldspink G (Eds) Mechanics and Energetics of Animal Locomotion. London:
Chapman and Hall, Ltd.

Goldspink G (1978) Energy turnover during contraction of different types of muscle. In:
Asmussen E, Jorgensen K (Eds) Biomechanics VI-A. Baltimore: University Park

Gollnick PD, Armstrong RB, Saltin B, Saubert CW, Sembrowich WL, Shephard RE (1973)
Effect of training on enzyme activity and fiber composition of human skeletal muscle.


References


References


References 233


   Pp 53-54.

Lannergren A (1976) Force-velocity relation of isolated twitch and slow muscle fibres. XV
   Scandinavian Congress of Physiology and Pharmacology, Arhus. Acta Physiologica
   Scandinavica (Supplement) 404: 87.


Levin SL, Egolinski IA (1936) The effect of cortical functions upon energy changes in basal
   421-422.


References


Nicholas CW, Williams C, Boobis LH, Little N (1994) Effect of ingesting a carbohydrate-electrolyte beverage on muscle glycogen utilisation during high intensity, intermittent


References


Sanchez JJ, Peuquignot JM, Peyrin L, Monod H (1980) Sex differences in the sympa-


Appendix A: Ethical review and informed consent.

Subjects were presented with a general statement of the background of the assessment procedures to be employed and the objectives of the assessment;

Subjects were given a clear explanation of the procedures to be followed and their purposes, and description of any and all risks attendant to the procedures;

An offer to answer any queries of the subject concerning procedures or other aspects of the project;

An instruction that the subject was free to withdraw consent and to discontinue participation in the project or activity at any time without prejudice to the subject;

An explanation of the procedures which were to be taken to ensure the confidentiality of the data and information to be derived from the subject;

Subjects were familiarised fully with all test procedures before each formal test.
Consent Form

I the undersigned do freely consent to be a subject in a series of studies conducted by Mr John Sproule to investigate aspects of running economy.

I declare that the purposes of the study have been fully explained to me and that I understand them. I am aware that I have to report to the laboratory on different occasions as required by the investigator.

The procedures to be followed and their purposes, with a description of risks attendant to the procedures, have been explained to my satisfaction.

I know that if I have any queries concerning procedures or other aspects of these studies that I am encouraged to ask the investigator.

There are no medical reasons preventing my participation as a subject in these studies.

I understand and am aware that my body composition will be assessed, maximum aerobic power will be assessed, and fatiguing submaximal running will be required. Gas analysis, fingertip blood sampling and heart rate monitoring will occur during testing. I understand that there is possible risk in physical exercise at this level and some discomfort may be experienced. I understand that this may occur though the investigator will take all proper care in the conduct of the testing and I will fully assume that risk.
I agree that the research data gathered for the study may be published and used for statistical and scientific research provided my name or other identifying information is not used.

I hereby agree that I will present myself for testing in a suitable condition having abided by the requirements for diet and activity prescribed to me by the investigator.

Participant’s signature : ________________________________

Date : ________________________________

Investigator’s signature : ________________________________

Date : ________________________________

Witnessed by : ________________________________

Date : ________________________________
Appendix B: Lactate analysis

The *Kodak Ektachem* DT Slide (lac) is a dry, multilayered, film in a plastic support. It contains all the reagents necessary to determine lactate concentration in 10 μL of plasma. The analysis is based on the lactate oxidase catalysed conversion of L(+) lactate to pyruvate and hydrogen peroxide in the presence of oxygen. The generated hydrogen peroxide oxidises the 4-aminoantipyrine, 1,7-dihydroxynaphthalene chromogen system in a horseradish peroxidase catalysed reaction. The intensity of the colour is proportional to the amount of lactate in the specimen and is measured by reflectance spectrophotometry.

Reactive ingredients include lactate oxidase, peroxidase, 1,7-dihydroxynaphthalene, 4-aminoantipyrine hydrochloride. Other ingredients include pigment, binders, buffer, antioxidants, polymer cross-linking agents, and surfactants.

A 10 μL drop of sample is deposited on the slide and evenly distributed by the spreading layer. Lactate in the sample is oxidised by lactate oxidase to pyruvate and hydrogen peroxide. The hydrogen peroxide generated oxidises the 4-aminoantipyrine, 1,7-dihydroxynaphthalene chromogen system in a horseradish peroxidase catalysed reaction and results in a dye complex. Each mole of lactate oxidised results in 0.5 mole of the dye complex. The sample is incubated at 37°C for approximately 5 minutes and the intensity of the dye complex is measured by reflectance spectrophotometry.
L(+) Lactic acid + O₂ → Lactate oxidase → pyruvate + H₂O₂

2H₂O₂ + 4-aminoantipyrine + 1,7-dihydroxynaphthalene → peroxidase → red dye
Appendix C: Glucose analysis

The Kodak Ektachem DT Slide (GLU) contains all the reagents necessary to determine glucose concentrations in 10 µL of plasma. The analysis is based on the enzyme-catalysed reaction of glucose with molecular oxygen, followed by a second reaction that produces a highly coloured red dye. The intensity of the colour is proportional to the amount of glucose in the sample.

Reactive ingredients include glucose oxidase (source: aspergillus niger), peroxidase (source: horseradish), 1,7-dihydroxynaphthalene, and 4-aminoantipyrine hydrochloride. Other ingredients include pigment, binders, buffer, surfactants, antioxidants, and a polymer crosslinking agent.

The spreading layer distributes the 10 µL sample evenly on the slide and the aqueous portion diffuses into the reagent layer. Glucose from the sample reacts with molecular oxygen in the presence of the enzyme glucose oxidase, as shown in the reaction sequence. The second reaction leads to formation of a highly coloured complex. By measuring the amount of light reflected from the coloured layer after a fixed incubation period, the analyser can calculate the amount of glucose that is present in the sample.
\[ \beta-D \text{ Glucose} + O_2 + H_2O \xrightarrow{\text{glucose oxidase}} D-\text{gluconic acid} + H_2O_2 \]

\[ 2H_2O_2 + 4\text{-aminoantipyrine} + 1,7\text{-dihydroxynaphthalene} \xrightarrow{\text{peroxidase}} \text{red dye} \]
Appendix D: Ammonia analysis

The *Kodak Ektachem* DT Slide (NH₃) contains all the reagents necessary to determine ammonia concentrations in 10 µL of plasma. The reaction is based on the selective migration of ammonia through a semipermeable membrane into a layer containing an indicator dye. The ammonia reacts with an indicator to produce a highly coloured dye. The intensity of the colour is proportional to the amount of ammonia in the sample.

Reactive ingredients include bromphenol blue. Other ingredients include pigment, binders, surfactants, buffer, and humectant.

A 10 µL drop of specimen is deposited on the slide and evenly distributed on the spreading layer. The ammonia in the sample then diffuses through the semipermeable membrane to react with the indicator dye in the second reagent layer (indicator layer). By measuring the amount of light reflected from the dyed layer after a fixed incubation period, the analyser calculates the amount of ammonia present in the sample.

\[
\text{NH}_3 + \text{Bromphenol blue (ammonia indicator)} \rightarrow \text{blue dye}
\]
Appendix E: Potassium analysis

The *Kodak Ektachem* DT Slide (K\(^+\)) - when used with electrolyte reference fluid - contains all the reagents necessary to determine potassium concentrations in 10 µL of plasma. A 10 µL drop each of specimen and reference fluid are deposited simultaneously on the slide by a dual-tip pipette. An electrical potential develops in proportion to the concentration of potassium in the specimen and is measured by a sensitive voltmeter.

Slide ingredients include silver, silver chloride, potassium chloride, and valinomyocin. Other ingredients include binders, plasticisers, and surfactants.

Electrolyte analysis uses a procedure called differential potentiometry. The slide contains two identical ion-selective electrodes. Each consists of a silver and a silver chloride layer over which an ion-selective membrane has been added to make the electrodes selective for potassium ions. A 10 µL drop of sample fluid is dispensed on the sample electrode and a 10 µL drop of reference fluid is simultaneously dispensed on the reference electrode. The two fluids spread toward each other through a paper bridge, forming a liquid junction in the centre. Each electrode develops an electrical potential (voltage) in response to the concentration of potassium applied to it. The difference in potential between the two electrodes is proportional to the logarithm of the potassium concentration in the sample.
Appendix F: Sodium analysis

The *Kodak Ektachem* DT Slide (Na⁺) - when used with electrolyte reference fluid - contains all the reagents necessary to determine sodium concentrations in 10 μL of plasma. A 10 μL drop each of specimen and reference fluid are deposited simultaneously on the slide by a dual-tip pipette. An electrical potential develops in proportion to the concentration of sodium in the specimen and is measured by a sensitive voltmeter.

Slide ingredients include silver, silver chloride, sodium chloride, and methylmonensin. Other ingredients include binders, plasticisers, and surfactants.

Electrolyte analysis uses a procedure called differential potentiometry. The slide contains two identical ion-selective electrodes. Each consists of a silver and a silver chloride layer over which an ion-selective membrane has been added to make the electrodes selective for sodium ions. A 10 μL drop of sample fluid is dispensed on the sample electrode and a 10 μL drop of reference fluid is simultaneously dispensed on the reference electrode. The two fluids spread towards each other through a paper bridge, forming a liquid junction in the centre. Each electrode develops an electrical potential (voltage) in response to the concentration of sodium applied to it. The difference in potential between the two electrodes is proportional to the logarithm of the sodium concentration in the sample.
Appendix G: Haemoglobin analysis

The **Kodak Ektachem** DT Slide (Hb) contains all the reagents necessary to determine haemoglobin levels in whole blood. A 10 μL drop of whole blood is deposited on the slide. The sample spreads evenly and haemoglobin is released from the red blood cells. Haemoglobin in the sample undergoes a series of reactions in the slide to produce a coloured compound. The intensity of the colour is proportional to the amount of haemoglobin in the sample.

Reactive ingredients include potassium ferricyanide \((\text{K}_3\text{Fe(CN)}_6)\) and potassium thiocyanate (KSCN). Other ingredients include pigment, binders, buffer, surfactants, and polymer beads.

A surfactant in the spreading layer breaks the membrane of the red blood cell which results in the release of haemoglobin into the spreading layer. The haemoglobin is oxidised to methemoglobin which is then converted to a coloured compound as shown in the reaction sequence. By measuring the amount of light reflected from the dye layer after a fixed incubation period the analyser can calculate the amount of haemoglobin present in the sample.

\[
\text{Haemoglobin (Fe}^{2+}\text{)} \rightarrow \text{K}_3\text{Fe(CN)}_6 \rightarrow \text{methemoglobin (Fe}^{3+}\text{)}
\]

\[
\text{methemoglobin} \rightarrow \text{KSCN} \rightarrow \text{isothiocyanmethemoglobin}
\]