INVESTIGATION OF THE METABOLIC RESPONSES DURING THE CYCLING PHASE OF AN ULTRAENDURANCE TRIATHLON

By

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A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF MASTER OF SCIENCE in THE FACULTY OF GRADUATE STUDIES School of Human Kinetics

We accept this thesis as conforming to the required standards

THE UNIVERSITY OF BRITISH COLUMBIA

December, 1999.

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Department of Human Kinetics

The University of British Columbia
Vancouver, Canada

Date November 24, 1999.
ABSTRACT

The purpose of this study was to investigate the premise that ultraendurance triathletes perform the cycling portion of the Ironman Triathlon at an intensity near ventilatory threshold ($T_{vent}$). Eleven highly-trained ultraendurance triathletes ($\bar{x} \pm$ SEM: age = $35.8 \pm 1.6$ yrs; body fat = $11.7\%$; $\dot{V}O_2_{max} = 67.5 \pm 1.0$ ml·kg$^{-1}$·min$^{-1}$) completed two randomly assigned trials. The first trial consisted of a ride to volitional fatigue ($BiT_{vent}$) at the subjects $T_{vent}$ power output ($PO_{Tvent}$) calculated from a preliminary cycling $\dot{V}O_2_{max}$ test. The second trial was a 5 h bike time trial at a self-selected pace ($Bi_{SSP}$). The endurance time for the $BiT_{vent}$ was $130.7 \pm 18.6$ min. Significant differences between trials existed on the measures: power output = $273.6 \pm 8.6$ vs. $188.0 \pm 8.6$ W; $\dot{V}O_2 = 3.61 \pm 0.15$ vs. $2.64 \pm 0.09$ L·min$^{-1}$; $\%T_{vent} \dot{V}O_2 = 110.7 \pm 2.6$ vs. $80.5 \pm 3.3\%$; HR = $159 \pm 3$ vs. $130 \pm 4$ bpm; RER = $0.95 \pm 0.01$ vs. $0.91 \pm 0.01$; and $\dot{V}E_{max} = 103.4 \pm 4.7$ vs. $66.7 \pm 2.8$ L·min$^{-1}$; $BiT_{vent}$ vs. $Bi_{SSP}$ respectively. While these results suggest that ultraendurance triathletes do not perform at their $PO_{Tvent}$ during the cycling phase, mean HR data ($146 \pm 2$ bpm) from seven of these subjects during the bike portion of the Canadian Ironman Triathlon (IMC) demonstrate that ultraendurance triathletes do perform at an intensity near their $T_{vent}$ HR ($150 \pm 4$ bpm; $r = .873; p < 0.05$). A treadmill run $\dot{V}O_2_{max}$ subsequent to $Bi_{SSP}$ significantly reduced $T_{vent}$ speed ($8.9 \pm 0.3$ mph vs. $8.4 \pm 0.2$ mph; $p < .05$), however, did not improve the ability to predict IMC marathon run pace (IMC marathon = $5.9 \pm 0.3$ mph; $r = .425; p > .05$).
TABLE OF CONTENTS

ABSTRACT .................................................................................................................. ii

TABLE OF CONTENTS .............................................................................................. iii

LIST OF TABLES ........................................................................................................ v

LIST OF FIGURES ...................................................................................................... vi

ACKNOWLEDGEMENT ............................................................................................... vii

CHAPTER 1: INTRODUCTION TO THE PROBLEM ..................................................... 1
  1.1 Introduction to the Problem ................................................................................ 1
  1.2 Statement of the Problem .................................................................................. 3
    1.2.1 Subproblems .............................................................................................. 3
  1.3 Definitions .......................................................................................................... 3
  1.4 Delimitations ...................................................................................................... 4
  1.5 Limitations ......................................................................................................... 4
  1.6 General Hypothesis .......................................................................................... 5
    1.6.1 Secondary hypothesis ............................................................................... 5
  1.7 Significance of the Study .................................................................................. 6

CHAPTER 2: LITERATURE REVIEW: Thresholds and considerations for ultraendurance triathlon performance .................................................. 7
  2.1 Introduction ........................................................................................................ 7
  2.2 Historical Development .................................................................................... 7
  2.3 Physiological Profile of Triathletes ................................................................... 8
    2.3.1 Triathlon Performance Prediction from Physiological Assessments .......... 10
  2.4 Considerations for Ultraendurance Triathlon Performance ............................. 11
    2.4.1 Substrate Utilization .................................................................................. 11
      2.4.1.1 Carbohydrate (CHO) ....................................................................... 11
      2.4.1.2 Lipid Metabolism ........................................................................... 13
      2.4.1.3 Protein Requirements .................................................................... 15
    2.4.2 Fluid and Electrolyte Homeostasis ......................................................... 16
    2.4.3 Cardiovascular Drift .................................................................................. 17
      2.4.3.1 Cardiovascular Drift in Triathletes ................................................... 19
  2.5 The Anaerobic Threshold .................................................................................. 20
    2.5.1 Lactate Thresholds .................................................................................... 21
    2.5.2 Ventilatory Thresholds ............................................................................... 21
      2.5.2.1 Relationship between Lactate and Ventilatory Thresholds .......... 22
    2.5.2.2 Performance at Ventilatory and Lactate Thresholds .......................... 22
    2.5.3 Time to Exhausition at the Anaerobic Threshold ..................................... 23
  2.6 Thresholds for Long-Distance Performance .................................................... 24
    2.6.1 Thresholds for Swimming ........................................................................ 24
      2.6.1.1 Effects of Swimming on Cycling ..................................................... 25
LIST OF TABLES

1. Compilation of physiological attributes of triathletes .................................................. 9

2. Physiological effects of 800m swimming on 75 min cycling from Kreider et al. (1988a) ................................................................. 26

3. Physiological effects of prior swimming and cycling on 10km running from Kreider et al. (1988b) ......................................................... 28

4. Timeline for testing ........................................................................................................ 44

5. Descriptive data for all subjects ..................................................................................... 45

6. Primary variables during $\dot{V}O_2_{\text{max}}$ tests ................................................................ 47

7. Cycle ergometry $\dot{V}O_2_{\text{max}}$ variable comparisons ...................................................... 48

8. Treadmill running $\ddot{V}O_2_{\text{max}}$ variable comparisons .................................................. 48

9. Comparison of primary variables between trials ($B_{i_{\text{vent}}} \text{ vs. } B_{i_{\text{SSP}}}$) ............... 50

10. Comparison of secondary variables between trials ($B_{i_{\text{vent}}} \text{ vs. } B_{i_{\text{SSP}}}$) ............ 51

11. Estimated and actual run times and paces for the marathon run portion of the IMC ........................................................................ 52

12. Comparison of heart rate at $T_{\text{vent}}$ between trials ...................................................... 54
LIST OF FIGURES

1. Hypothetical plot of heart rate over time during an ultraendurance event as proposed by O'Toole et al. (1998) ................................................................. 37
2. Timeline of testing procedures for Bi_{Tvent} and Bi_{SSP} ................................................. 44
3. Comparison of power output over time for Bi_{Tvent} and Bi_{SSP} .......................... 54
4. Comparison of pedaling rate over time for Bi_{Tvent} and Bi_{SSP} .......................... 55
5. Comparison of heart rate over time for Bi_{Tvent}, Bi_{SSP}, and IMC ....................... 55
6. Comparison of heart rate over time for Bi_{SSP}, and IMC .................................. 56
7. Comparison of power output, heart rate, and pedaling rate over time for Bi_{SSP} ........ 56
8. Hematocrit over time during Bi_{SSP} ................................................................. 57
9. Respiratory exchange ratio over time during Bi_{SSP} ........................................... 57
10. 1999 IMC bike course profile ........................................................................ 58
11. Heart rate over time during 1999 IMC .......................................................... 58
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CHAPTER 1: INTRODUCTION TO THE PROBLEM

1.1 Introduction to the Problem

Over the years, researchers have endeavored to describe the factors that contribute to human performance during prolonged endurance exercise. Physiological variables that highly correlate with endurance performance included maximal oxygen uptake ($\dot{V}O_{2\text{max}}$), anaerobic threshold (AT), and economy of motion (Sleivert & Rowlands, 1996; Dengel et al., 1989). However, in activities beyond typical endurance events such as the marathon, termed 'ultraendurance' (i.e. > 4 hours; Hawley & Hopkins, 1995), the relationship between laboratory measured physiological variables fail to correlate strongly with ultraendurance triathlon (UET) performance (O'Toole et al., 1989; Langill & Rhodes, 1993; Miura et al., 1997).

The "endurance triathlon", or Ironman Triathlon, is a three-sport event consisting of a 3.8km swim, a 180km cycle, followed by a 42.2km marathon run. The duration is longer than four hours, and can be considered 'ultraendurance' (Kreider, 1991; Hawley & Hopkins, 1995). Therefore, a more appropriate name for the race may be an "ultraendurance triathlon (UET)" and will be referred to throughout this thesis accordingly.

The UET is a grueling event that takes between 8 and 17 hours to complete, depending on the calibre of athlete. The successful triathlete is one who has the ability to perform each sequential event at an optimal pace without creating fatigue that will hinder performance in the next event (O'Toole et al., 1989). However, the duration, intensity, and extreme environmental conditions encountered during UET combine to produce physiological stresses not seen in shorter races such as shorter triathlons and marathons (Hiller, 1989). While the sport of triathlon has received much research
attention, an optimum performance intensity has not been established that takes into
consideration the specific UET problems. Therefore, competition exercise intensities
during UET have been extremely variable (O'Toole et al., 1987a).

It is well established that there are physiological measures of endurance
performance we can measure in the laboratory, such as \( \dot{V}O_{2\text{max}} \), the AT, and economy
of movement (Wasserman, 1984). Although these physiological measures have been
proven reliable predictors of race success in endurance exercise (Petit et al., 1997;
Rhodes & McKenzie, 1984; Barlow et al., 1985), they have also been shown to be
different than an athlete’s performance threshold (Nichols et al., 1997; Hoogeveen &
Schep, 1997). Hence, there are limitations to the threshold concept. Nevertheless, this
reasoning has lead many authors to develop UET field predictions based upon
laboratory assessments (Langill and Rhodes, 1993; Miura et al., 1997). However, these
studies reveal that physiological measurements in the laboratory are not related to
performance during UETs, as other factors encountered such as hydration and energy
homeostasis must first be taken into consideration.

A detailed laboratory assessment of highly-trained ultraendurance triathletes,
coupled with field considerations will provide further insight as to the optimum intensity
that can be maintained for prolonged periods. If the ultraendurance triathlete could
identify a heart rate (HR) corresponding to a percentage of AT from a laboratory
physiological assessment, then theoretically, performing at this intensity could maximize
performance. Other "field" factors must also be considered including; the ability of the
triathlete to exercise at a high percentage of \( \dot{V}O_{2\text{max}} \) for a prolonged period of time, the
effect that a preceding event has on a subsequent event, cardiovascular (CV) drift, fluid
homeostasis, and energy balance (O'Toole et al., 1987a; Kreider et al., 1988; Coyle,
1998; Coyle & Montain, 1992). Coupling these factors along with the appropriate HR effect could theoretically maximize performance.

1.2 Statement of the Problem

The purpose of this investigation was to determine the intensity (\%VO_2max, \%T_{vent}, \%HR_{max}) that a group of highly-trained ultraendurance triathletes perform at during ultraendurance laboratory trials and during the cycling portion of the Canadian Ironman Triathlon.

1.2.1 Subproblems

1) to determine how cardiac drift affects this intensity.

2) to examine substrate utilization and its affects on HR.

3) to question whether cycling intensity can be optimized to allow subsequently for an optimal marathon run performance.

4) to question if the velocity at T_{vent} (determined via excess CO_2) during running subsequent to a 5 h cycle time trial can estimate race pace (time) for the marathon run phase of the Ironman Triathlon.

1.3 Definitions

1) Ventilatory threshold - (T_{vent}) the point where the aerobic energy response is of insufficient magnitude to supply the tissues energy requirement and there is an increased reliance on anaerobic processes with an accompanying abrupt increase in excess CO_2.
2) Excess CO₂ - nonmetabolic CO₂ (EXCO₂) formed as a result of the hydrogen ions of lactic acid being buffered by bicarbonate in the following reactions:

$$\text{HLa} + \text{NaHCO}_3 \rightleftharpoons \text{NaLa} + \text{H}_2\text{CO}_3 \rightleftharpoons \text{CO}_2 + \text{H}_2\text{O} + \text{NaLa}$$

The calculation EXCO₂ will be based on the formula of Volkov et al. (1975) where:

$$\text{EXCO}_2 = \dot{\text{VCO}}_2 - (\text{RER}_{\text{rest}} \times \dot{\text{V}}\text{O}_2)$$

1.4 Delimitations

This study was delimited by:

1) a sample of triathletes from the Province of British Columbia between the ages of 29 and 44 with a minimum 12 h previous best Ironman Triathlon time.

2) A respiratory gas-sampling rate set at 20-s intervals during ramped protocols, and measured for 5 min every 30 min during prolonged testing.

3) A heart rate sampling rate set at 5 s intervals during ramped protocols, and set at 60 s intervals during prolonged testing.

1.5 Limitations

This study will be limited by:

1) The data collection capabilities of the Vmax Metabolic Measurement Cart (V6200, SensorMedics Corporation, Yorba Linda, CA), and the Polar Vantage NV Heart Rate Monitor System (Polar Electro, Finland).

2) The individual metabolic response to the exercise protocols.

3) Race day conditions (terrain, weather, equipment problems, fuels, fluids, etc.)
4) Athlete self-selection of ultraendurance workload versus using a calculated workload. [Reasoning: if the workload were set by the evaluator then the athlete runs the risk of either not finishing the trial, or not being appropriately challenged].

1.6 General Hypothesis

This study will provide information for which to calculate an optimal critical ultraendurance intensity, which hereafter is referred to as the ultraendurance threshold (UT). This will be accomplished by observing physiological variables during an UET cycle performance. More specifically, this UT, as determined by a homogenous group of elite ultraendurance triathletes, will correlate significantly with some percentage of $T_{vent}$.

1.6.1 Secondary Hypotheses

1) The AT is too great of an intensity to ride at during the UET bike course.

2) An increase in HR (cardiovascular drift) will occur during the initial portion of the prolonged tests.

3) Substrate utilization (decrease in ratio of CHO:FAT), as reflected by respiratory exchange ratio (RER), will cause for significant increases in oxygen consumption ($\dot{V}O_2$) as well as decreases in HR, yet power output (PO) will be maintained throughout the prolonged tests.

4) A significant difference will exist between treadmill run $\dot{V}O_2_{max}$ measured variables rested versus post 5 h cycle time trial.

5) A significant difference will exist between treadmill run $T_{vent}$ measured variables rested versus post 5 h cycle time trial.
6) Treadmill run velocity at $T_{\text{vent}}$ subsequent to a 5 h cycling time trial will correlate highly with actual performance time for the marathon phase of the Ironman Triathlon.

1.7 Significance of the Study

The 3.8km swim portion of an UET represents only 10% of the total duration of the UET (Butts et al., 1991) and is therefore a relatively small feat compared with the remainder of the event. But thereafter, the Ironman triathlete is troubled with deciding on an intensity to be maintained throughout the cycle portion in an attempt to achieve peak performance. During the bike portion of the UET, athletes may be performing at too great of an intensity in the beginning of their 180km cycle race for many reasons. These include the excitement of the day they have trained many months for, on top of the fact that they are rested, tapered, and therefore psychologically “feeling strong”. Unfortunately, this surge of energy in the beginning of the cycling event could prove detrimental to their overall performance. It has been demonstrated that once blood lactate levels have risen above manageable levels due to exceeding the optimum PO it affects the athlete’s ability to continue generating the original PO (Firth, 1998; Wasserman, 1987), and can lead to exhaustion (Urhausen et al., 1994). Therefore, identifying an ‘ultraendurance threshold’, or a critical intensity, could optimize the performance of the ultraendurance triathlete.
CHAPTER 2: Literature Review: Thresholds and Considerations for
Ultraendurance Triathlon Performance

2.1 Introduction

This review is an examination of the literature pertaining to the physiological factors affecting the second phase of an UET. The review will examine the physiological attributes of triathletes, fluid and substrate requirements of prolonged exercise, as well as the anaerobic threshold (AT) concept relating to triathlon and its component sports of swimming, cycling, and running. The review will also identify principles by which HR readings during an ultraendurance event could provide useful information by which the ultraendurance triathlete could maximize their performance. These concepts will be combined to form a new paradigm, which could be called the “ultraendurance threshold (UT)”.

2.2 Historical Development

Triathlon is an endurance contest where participants compete consecutively in three sports, usually swimming, cycling, and running. It is still a relatively new sport (22 years old) and research studies are needed to identify the physiological mechanisms involved with prolonged and variable event exercise. The UET began in 1978 when the Waikiki Rough Water Swim (3.8km), the Around Oahu Bike Race (180.2 km), and the Honolulu Marathon (42.2 km) were combined to form the Hawaiian Ironman (O'Toole et al., 1989). This race was previously looked upon as the ultimate in endurance sports in the early 1980's and perhaps thought of as an upper limit to an endurance athlete's capacity (Laursen & Rhodes, 1999). The contest's early years saw very few athletes attempt this endeavor. However, with the media's exposure of the Hawaiian Ironman, shorter triathlon distances also began to grow in popularity as people aspired to one day
compete at the event. This growth has popularized the sport to a large extent and has helped in leading the Olympic distance triathlon (1.5-km swim, 40-km cycle, 10-km run) to make its debut in the Sydney 2000 Summer Olympic Games. As well, over 10,000 triathletes compete in Ironman Triathlons around the world yearly (Docherty, 1998). This increase in the popularity of UET has also lead to an increase into the scientific inquiry of ultraendurance exercise.

2.3 Physiological Profile of Triathletes

Physiological characteristics of triathletes encompass a wide range of endurance performance characteristics that are similar to that of cyclists (O'Toole et al., 1987). Elite triathletes are generally tall, of average to light weight and have low levels of body fat, and high maximum oxygen uptake (\(V\dot{O}_{2\max}\)) values (see Table 1). It has been suggested that the absolute aerobic capacity in triathletes is the critical component to maintain high exercise intensity, especially during the running portion of the triathlon (Zhou et al., 1997). However, \(V\dot{O}_{2\max}\) may be, on average, marginally lower than values previously observed in single event endurance specialists (Sleivert & Rowlands, 1996). \(V\dot{O}_{2\max}\) in triathletes has been shown to be significantly greater during treadmill running versus cycle ergometry, and lower during tethered swimming than both cycle ergometry and treadmill running (Schneider et al., 1990; Schneider & Pollack, 1991; Kohrt et al., 1989; O'Toole et al., 1987). Nevertheless, elite triathletes have significantly higher \(V\dot{O}_{2\max}\) values than sub-elite triathletes and high \(V\dot{O}_{2\max}\) levels are required for success in triathlons (Sleivert & Rowlands, 1996).

The ability of the triathlete to exercise at a lower percentage of \(V\dot{O}_{2\max}\) for a given submaximal workload might be the most important factor to triathlon success (Burke,
1995). This is influenced not only by $\dot{V}O_{2\text{max}}$ itself, but also by AT and economy of movement (Sleivert & Rowlands, 1996).

**Table 1.** Compilation of physiological attributes of triathletes.

<table>
<thead>
<tr>
<th>Author</th>
<th>Sex</th>
<th>N</th>
<th>Age (years)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
<th>$VO_{2\text{max}}$ (ml·kg$^{-1}$·min$^{-1}$)</th>
<th>AT (% of $VO_{2\text{max}}$)</th>
<th>% Body Fat</th>
</tr>
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<tbody>
<tr>
<td>Bunc et al. (1996)</td>
<td>F</td>
<td>13</td>
<td>17.1 ± 1.4</td>
<td>168.4 ± 2.0</td>
<td>58.8 ± 4.7</td>
<td>56.1 ± 2.4</td>
<td>83.1 ± 1.7</td>
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</tr>
<tr>
<td></td>
<td>M</td>
<td>23</td>
<td>17.7 ± 2.2</td>
<td>176.5 ± 5.1</td>
<td>66.7 ± 7.1</td>
<td>67.9 ± 5.9</td>
<td>82.4 ± 2.1</td>
<td>8.2 ± 2.3</td>
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<td>181 ± 0.06</td>
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<td>91.1 ± 1.0</td>
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<td>28.3 ± 2.3</td>
<td>166.4 ± 2.1</td>
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<td>85.0 ± 2.1</td>
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</tr>
<tr>
<td></td>
<td>M</td>
<td>18</td>
<td>27.7 ± 1.3</td>
<td>180.0 ± 1.5</td>
<td>76.2 ± 2.1</td>
<td>63.7 ± 1.6</td>
<td>85.0 ± 1.3</td>
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<td>Kohrt et al. (1989)</td>
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<td></td>
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<td>58.4 ± 1.4</td>
<td>≈ 85</td>
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<td>71.1 ± 2.5</td>
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<td></td>
<td>11.6 ± 0.9</td>
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<td>O'Toole et al. (1987a)</td>
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<td>8</td>
<td>30.5 ± 8.8</td>
<td>178.8 ± 6.6</td>
<td>74.7 ± 10.0</td>
<td>68.8 ± 10.4</td>
<td>9.9 ± 3.5</td>
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<td></td>
<td>F</td>
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<td>31.3 ± 5.6</td>
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<td>179.0 ± 5.7</td>
<td>74.3 ± 2.3</td>
<td>57.9 ± 1.8</td>
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<td>Kreider et al. (1988a)</td>
<td>M</td>
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<td>179.4 ± 6.3</td>
<td>72.8 ± 6.7</td>
<td>68.1 ± 11.9</td>
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<td>Kohrt et al. (1987)</td>
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<td>13</td>
<td>29.5 ± 4.8</td>
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<td>69.8 ± 5.6</td>
<td>60.5 ± 5.6</td>
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<td>40 ± 11</td>
<td>180.2 ± 8</td>
<td>73.7 ± 8</td>
<td>57.4 ± 7.5</td>
<td>61 ± 2.2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>10</td>
<td>31 ± 8</td>
<td>171.0 ± 9</td>
<td>58.0 ± 7</td>
<td>57.5 ± 5.6</td>
<td>61 ± 2.2</td>
<td></td>
</tr>
<tr>
<td>Dengel et al. (1989)</td>
<td>M</td>
<td>11</td>
<td>31.4 ± 1.8</td>
<td>179.9 ± 1.6</td>
<td>74.5 ± 2.3</td>
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<td>65.6 ± 1.7</td>
<td>8.6 ± 0.7</td>
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<td>M</td>
<td>16</td>
<td>22.1 ± 3.2</td>
<td>179.0 ± 6.4</td>
<td>73.5 ± 10.6</td>
<td>62.0 ± 8.4</td>
<td>12.3 ± 4.0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>7</td>
<td>21.0 ± 1.0</td>
<td>165.7 ± 3.6</td>
<td>59.8 ± 4.1</td>
<td>50.7 ± 2.6</td>
<td>19.0 ± 4.6</td>
<td></td>
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<tr>
<td>Schneider &amp; Pollack (1991)</td>
<td>F</td>
<td>10</td>
<td>25.5 ± 0.8</td>
<td>167.6 ± 1.7</td>
<td>59.0 ± 1.8</td>
<td>63.6 ± 1.2</td>
<td>74.0 ± 2.0</td>
<td></td>
</tr>
</tbody>
</table>
2.3.1 Triathlon Performance Prediction from Physiological Assessments

Many authors have attempted to predict triathlon race performance from physiological assessments. Some authors have shown significant correlation with $\dot{V}O_2_{\text{max}}$ and performance times in moderately trained triathletes (Butts et al., 1991; Kohrt et al., 1987; Zhou et al., 1997; O'Toole et al., 1989; Dengel et al., 1989; Langill & Rhodes, 1993). However, as the length of the triathlon increases, other parameters begin to play a large influence on ultraendurance performance, such as thermal regulation and fluid homeostasis, as well as energy balance (Kreider, 1991). Nevertheless, the AT has proven to be the greatest predictor of race performance in endurance cycling (Coyle et al., 1991; Coyle, 1995; Bishop et al., 1998) and endurance running events (Rhodes & McKenzie, 1984; Petit et al., 1997; Farrel et al., 1979; Perronnet et al., 1987). Olympic distance triathlon performance times have also been shown to be highly correlated with the AT measured during cycle ergometry (Zhou et al., 1997). While the $\dot{V}O_2_{\text{max}}$ of the athlete will set the upper limit, the ability of the triathlete to exercise at a higher fractional utilization of $\dot{V}O_2_{\text{max}}$ is crucial to race success (Zhou et al., 1997; Coyle, 1995).

The AT is defined as the $\dot{V}O_2$ during exercise above which there is a sharp increase in anaerobic energy production resulting in a significant increase in lactic acid levels (Wasserman, 1984). Theoretically then, the AT could be the ultimate intensity at which an ultraendurance triathlete could perform at during the Ironman Triathlon. However, ultraendurance considerations must first be identified to determine the ultraendurance triathlete's true limitations.
2.4 Considerations for Ultraendurance Triathlon (UET) Performance

The Ironman Triathlon can take anywhere from 8 to 17 hours depending on the calibre of the athlete (Docherty, 1998). This long duration, moderate intensity exercise in the heat creates unique physiological challenges including energy balance, and fluid and electrolyte homeostasis. Caloric expenditure for the UET can range from 8,500 to 11,500 kcals (Kreider, 1991), while sweat rates can reach up to two L·hr\(^{-1}\) in the heat, creating fluid and electrolyte disturbances (Coyle, 1994). Therefore, during ultraendurance competition, athletes require fluid and energy replacement to control body temperature and sustain endurance (Laursen & Rhodes, 1999). It is important to understand the influence these factors have on the ultraendurance triathlete as they affect variables measured in a laboratory assessment such as HR and AT (De Vito et al., 1995).

2.4.1 Substrate Utilization

Substrate utilization during an UET is of great interest to exercise physiologists. The extreme caloric expenditures of these athletes requires significant fueling contribution from all sources including carbohydrate (CHO), fat and protein (Applegate, 1989).

2.4.1.1 Carbohydrate (CHO)

Ultraendurance competition requires enormous energy expenditure and the ability to maintain a steady performance depends on an adequate supply of metabolic fuels (Kreider, 1991; Laursen & Rhodes, 1999). Intense exercise (i.e. above 60% \(\dot{V}O_2\text{max}\)) can be maintained for prolonged periods, provided that sufficient CHO is available for energy (Coyle & Montain, 1992). Therefore, CHO should be the main fuel consumed during ultraendurance events to maintain blood glucose levels (Anderson et
CHO must be replaced because it has a limited storage capacity. The total CHO availability from muscle, liver, and blood glucose yields approximately 2000-2500 kcal, or one to two hours aerobic glycolytic exercise (Sherman & Wimer, 1991; Hawley & Hopkins, 1995; Sherman, 1996). Intense exercise performed beyond this time period without a CHO source will cause blood glucose levels to decline and fatigue to occur (Singh et al., 1994). CHO might also reduce fatigue in the central nervous system. This is known as the central fatigue hypothesis (Davis, 1995; Davis et al., 1992; Walberg-Rankin, 1995). Hence, the goal for the ultraendurance athlete then remains in obtaining enough energy throughout the diet in the form of CHO to balance the constant energy output. Due to the importance of CHO, numerous authors have investigated its intake immediately prior to or during exercise and concluded that CHO ingestion improves performance during long duration exercise (Applegate, 1989; Lamb et al., 1991; Tarnopolsky et al., 1996; Tsintzas & Williams, 1998; Sherman et al., 1989; Walton & Rhodes, 1997).

CHO loading is a well-established and well-practiced ergogenic aid to promote endurance performance (Millard-Stafford et al., 1988; 1990; Brewer et al., 1988; Applegate, 1991; Lindeman, 1992; Singh et al., 1993; Jeukendrup et al., 1996). CHO is also equally important during the event itself. During prolonged exercise (>1-2 hrs), CHO should be ingested to prevent fatigue due to inadequate supply of blood glucose. Peters and colleagues (1995) demonstrated in 32 male triathletes that a liquid CHO feeding during exercise (75% $\dot{V}O_2\max$) resulted in significantly higher times to exhaustion versus a placebo. This may occur through the maintenance of blood glucose, or through the sparing of muscle glycogen (Tsintzas & Williams, 1998). It is also possible
that CHO ingestion may lead to a resynthesis of muscle glycogen in non-active muscle fibres during exercise (Walton & Rhodes, 1997).

The addition of CHO to fluid replacement beverages is important because it provides CHO late in exercise when there is often an inadequate supply of endogenous CHO to meet the energy requirements of the exercise task (Coyle & Montain, 1992). Maltodextrin has been shown to result in increased endurance cycling performance versus placebo (Langenfeld et al., 1994), and is a commonly used CHO source in commercial sport drinks. Fructose supplementation has been demonstrated to cause gastrointestinal discomfort in large doses during exercise and therefore should be limited (Craig, 1995). Ingestion of approximately 30-70g CHO·h⁻¹ (Coyle & Montain, 1992) or 0.2-0.6 g CHO·kg⁻¹·h⁻¹ (Applegate, 1989) in the form of a 5%-10% CHO solution will generally be sufficient to maintain blood glucose oxidation late in exercise and delay fatigue (Walberg-Rankin, 1995).

### 2.4.1.2 Lipid Metabolism:

Although CHO is the most important fuel to be consumed during ultraendurance exercise, it is important to understand the contribution that lipid makes as an energy substrate during prolonged exercise. Fat is the fuel of choice during exercise bouts of long duration, low intensity exercise (Okano et al., 1996; Romallo & Rhodes, 1998). As intensity increases, sustained exercise at a high PO requires the utilization of both fat and CHO simultaneously (Newsholme, 1981). But even at high work intensities (i.e. 85% \( \dot{V}O_2_{max} \)), a substantial amount of energy is still derived from fat oxidation, usually between 25 and 30% (Ranallo & Rhodes, 1998). Because CHO stores are limited, fat becomes a major energy substrate during an ultraendurance event (Kreider, 1991). Theoretically, adipose tissue triglyceride provides sufficient energy for about five days of
continuous marathon running (Newsholme, 1981). The use of this energy system has been labeled the aerobic lipolytic energy system (Hawley & Hopkins, 1995). Evidence for the major use of this system is evident from ultraendurance studies. Blood samples drawn immediately following completion of endurance races up to the length of a marathon do not demonstrate a decrease in serum triglycerides (Dufaux et al., 1986; Thompson et al., 1980). Ultraendurance events, however, have demonstrated this (Farber et al., 1991; Nagel et al., 1992; Ginsburg et al., 1996). Hence, as exercise increases in duration, athletes depend more on lipid as a major energy substrate.

Endurance training increases the size and number of mitochondria to greatly enhance aerobic metabolism and the ability of muscle to use oxygen to metabolize fat and CHO for energy (Hopkins et al., 1996). The ability then to oxidize fat at a very high rate for prolonged periods of time is very advantageous to the ultraendurance triathlete. This premise has lead to a hypothesis whereby "fat loading" prior to an ultraendurance event might increase performance (Hopkins, 1996). Performance, however, is more likely to be impaired by eating fat at the expense of CHO, as fat oxidation cannot support exercise intensities greater than 60% to 65% $\dot{V}O_2$ max (Sherman & Leenders, 1995; Hopkins et al., 1996; Clark et al., 1992). Digestive problems and the lowered preference for this type of fuel lead to a low intake despite the advantage of a higher energy content (Saris et al., 1989). Consequently, the complete reliance on fat as the fuel for ultradistance exercise would significantly compromise performance capacity (Kreider, 1991).

Nevertheless, fat is a necessary part of the diet in that it provides essential fatty acids and aids in the absorption of fat-soluble vitamins (Lindeman, 1992). Although fat consumption during competition should be limited, it is recommended that
ultraendurance athletes consume 20% to 30% of their total daily energy in the form of fat as desired during a regular training day (Saris et al., 1989; Clark et al., 1992).

2.4.1.2 Protein Requirements:

Protein requirements of ultraendurance athletes have not been critically evaluated, but evidence indicates that protein needs of endurance athletes may depend on the intensity and volume of training (Singh et al., 1994). Energy demands from dietary protein increase if glycogen becomes depleted (Lindeman, 1992). Ultraendurance studies indicate that athletes consume 5% to 15% of their calories from protein (Clark et al., 1992; Saris et al., 1989). The contribution of protein to total energy expenditure results from a negative energy balance. Kreider (1991) suggests five potential reasons for this occurrence:

1) Suppression of appetite and/or boredom of frequent eating
2) the inability to completely replenish muscle and liver glycogen stores within short periods of time
3) the high fiber content of complex CHO providing a sensation of fullness, yet caloric needs may not be met
4) inappropriate food choices
5) the difficulty of consuming 8,000-15,000 kcal·d⁻¹

In a recent review, Lemon (1995) states that endurance athletes would benefit from diets containing more protein than the current RDA of 0.8 g·kg⁻¹·day⁻¹. Current recommendations for endurance athletes range from 1.0 to 1.5 g·kg⁻¹·day⁻¹ (Clark et al., 1992, Lemon, 1995). This protein increase covers the increased loss of amino acids oxidized during exercise (greater than 60% \( \dot{V}O_2_{\text{max}} \)), and may provide additional raw materials to replace exercise-induced muscle damage which has been shown to occur during ultraendurance racing (Lemon, 1995; Case et al., 1995; Armstrong, 1986).
Markers such as blood urea nitrogen, potassium, and serum muscle enzyme levels have been demonstrated to increase during an UET, reflecting skeletal muscle permeability or disruption (Farber et al., 1991). In a study of a 1000km running race, Raschka and associates (1991) suggest the absolute protein intake of 1.7-g·kg⁻¹·day⁻¹ during the race should be increased in order to diminish the loss of musculature they found during ultraendurance racing. Therefore, prolonged ultraendurance exercise in itself can lead to ultrastructural damage in muscle, which could contribute to the body’s ability to replenish its glycogen stores. Protein requirements may be more important in ultraendurance events than previously thought.

2.4.2 Fluid and Electrolyte Homeostasis

The second major consideration resulting from the prolonged nature of triathlon is the maintenance of fluid and electrolyte homeostasis. Heat produced during exercise is primarily dissipated by the evaporation of sweat (Coyle & Montain, 1992; Brouns et al., 1992). Fluids are also lost through the respiratory tract, gastrointestinal tract, and kidneys (Greenleaf, 1992). Therefore, the proper replacement of fluids and electrolytes during prolonged exercise is essential (Applegate, 1989). Dehydration will result without fluid replenishment. Dehydration is a consequence of body water loss due to sweating, which impairs the process of heat dissipation, resulting in elevated deep body (core) temperatures and reduced endurance performance (Coyle & Montain, 1992). Environmental heat exposure will amplify this process. For dehydrated athletes, the continuation of exercise heat stress can result in severe hyperthermia, and potentially a life-threatening thermal injury (Sawka, 1992). Cognitive performance can also be adversely affected by body water deficit (Schoene, 1984). Dehydration is the most common reason for a triathlete in the Hawaii Ironman Triathlon to need medical
assistance, while hyponatremia, an extracellular sodium imbalance, is the predominant electrolyte disturbance (Hiller, 1989; Laird, 1989). Hence, sodium consumption during an UET is also of particular concern (O'Toole & Douglas, 1995).

Water losses can be as high as two l·h⁻¹ in hot weather, especially when the exercise intensity is greater than 70% \( \bar{V}O_2 \text{max} \) (Coyle; 1994). Since the average rates of gastric emptying and intestinal absorption can reach 1 l·h⁻¹ for water and solutions containing up to 8% CHO, athletes can be supplemented with both CHO and fluids at relatively high rates without compromising either fluid replacement or CHO absorption (over 60 g·h⁻¹ of CHO and 1 l·h⁻¹ of fluid) [Coyle & Montain, 1992].

2.4.3 Cardiovascular Drift

If the objective of this thesis is to determine a HR threshold intensity that would optimize performance during an UET, then it is important to observe the effects that long-duration performance have on HR. A slow but steady increase in HR is witnessed during prolonged endurance exercise at a constant work-rate. For example, Grant and associates (1997) showed HR to increase from 147 to 173 bpm during a two hour ride at the work rate corresponding to 46% \( \bar{V}O_2 \text{max} \) in eight untrained individuals. This phenomenon is known as cardiovascular (CV) drift. CV drift is caused by a progressive decrease in stroke volume (SV), resulting in an increase in HR required to maintain cardiac output (\( \dot{Q} \)) during prolonged exercise (Shaffrath & Adams, 1984; Grant et al., 1997). A rise in plasma catecholamine concentration parallel this rise in HR (Nielsen et al., 1984). CV drift occurs even more profoundly in conditions that combine high metabolic and thermal circulatory demands (Heaps et al., 1994). CV drift is augmented by dehydration (Coyle & Montain, 1992b; Heaps et al., 1994), and reduced by fluid replacement (Hamilton et al., 1991). Although not yet scientifically proven, CV drift may
occur less in trained individuals versus untrained (Lambert et al., 1998).

The mechanisms used to explain CV drift are still being investigated. The examination of CV drift in horses indicates that CV drift might be due to increases in core body temperature and lactate levels (Thomas & Fregin, 1990; Coyle, 1998). Human research indicates that reduced blood glucose levels may be related to CV drift. Hamilton and co-workers (1991) discovered that the infusion of an 18% glucose/saline solution during exercise, sufficient to maintain hyperglycemia and prevent dehydration, resulted in the complete elimination of CV drift during two hours of cycling at 70% \( \dot{VO}_2_{max} \). This study amplifies the importance of both fluid and CHO replacement simultaneously during prolonged exercise (Coyle, 1994).

The change in SV during prolonged exercise is equivocal. Although some studies have demonstrated SV to be maintained during prolonged exercise with fluid ingestion (Thomas & Fregin, 1990; Hamilton et al., 1991), the majority of author have shown SV to steadily decrease (Shaffrath & Adams, 1984; Coyle & Montain, 1992b; Grant et al., 1997; Coyle, 1998; Gonzalez-Alonso et al., 1998). Approximately one-half of this SV reduction is probably due to the reduced blood volume from dehydration during exercise that produces hyperthermia, while the remaining reduction in SV appears to be related to additional factors such as hyperthermia and the interaction with factors that further reduce ventricular filling, such as heart rate acceleration (Coyle, 1998).

Although it was previously felt that an increase in skin blood flow was the direct cause of a CV drift (Freud et al., 1987; Montain & Coyle, 1992a; 1992b), recent evidence has demonstrated that reductions in SV due to dehydration and concomitant hyperthermia are due to reductions in muscle blood flow (Coyle, 1998; Gonzalez-Alonso
et al., 1998). In fact, skin blood flow actually declines as skin and systemic vascular resistance increases as the CV system attempts to cope with the severe challenge of large reductions in $\dot{Q}$ (Coyle, 1998). Sweat rate becomes a problem.

CV drift occurs during triathlon events (Guezennec et al., 1996), therefore HR during an UET may be an overestimation of the work rate (O'Toole et al., 1989). Consequently, attempting to maintain euhydration could theoretically reduce the degree of CV drift during an UET. However, fluid compartment shifts also take place during exercise, even after 10 min (Nielsen et al., 1984). Farber and colleagues (1991) studied the metabolic changes in 11 subjects participating in the Hawaiian Ironman Triathlon. The authors noted an increase in serum albumin without a significant change in body weight to represent a decrease in plasma volume. This might represent the movement of fluid from the extracellular compartment to the skeletal muscle cells. This effect would happen secondarily to the development of an osmotic gradient caused by the breakdown of muscle glycogen to lactate during the swim resulting in a lactate concentration that is higher within muscle than in blood (Farber et al., 1991; Armstrong, 1986). Hence, the ultraendurance triathlete can prevent a proportion of the CV drift due to dehydration, but confounding fluid compartment shifts seem uncontrollable at this time.

2.4.3.1 Cardiovascular Drift in Triathletes

The scientific literature contains little direct data regarding the extent to which fluid replacement during exercise should match sweat rate to offset CV drift and hyperthermia (Coyle & Montain, 1992). CV drift has been observed during prolonged swimming (Nielsen et al., 1984), cycling (Shaffrath & Adams, 1984; Freund et al., 1987; Hamilton et al., 1991; Montain & Coyle, 1992a; 1992b), running (Westerlind et al., 1992;
1994) and during a triathlon (O'Toole et al., 1987a; 1989; Guezennec et al., 1996). However, swimming CV drift has been found to be not as profound as cycling CV drift, due to both the horizontal and hydrostatic conditions during swimming (Shaffrath & Adams, 1984; Nielsen et al., 1984). Nevertheless, CV drift will already have begun after prolonged swimming during an UET, possibly due to a decrease in plasma volume (McMurray; 1983; Guezennec et al., 1996; Hausswirth et al., 1996). This decrease in plasma volume might not only be due to dehydration (O'Toole et al., 1987a), but also due to a redistribution of blood volume (Farber et al., 1991). Even if blood volume remained constant, a decrease in venous return due to this redistribution would result in a CV drift. Hence, CV drift has implications for judging UET intensity on the basis of HR.

2.5 The Anaerobic Threshold

The AT is defined as the $\dot{V}O_2$ during exercise above which there is a sharp increase in anaerobiosis where lactate accumulation exceeds removal (Wasserman, 1984). Thus, the AT represents the upper limit of PO where lactate production and removal may attain equilibrium during constant load exercise (Rusko et al., 1986). Higher ATs have been associated with increased aerobic enzyme activity, lower muscle glycogen usage and lower blood lactate concentrations at the same relative workload, resulting in increased performance (Holloszy & Coyle, 1984; Coyle et al., 1988; Coyle et al., 1991; Loftin & Warren, 1994). The AT has proven useful for predicting the ability of athletes to sustain a given work rate for a prolonged period (Rhodes & McKenzie, 1984; Peronnet et al., 1987) and for determining the $\dot{V}O_2$ above which there is cardiovascular insufficiency in meeting tissue $O_2$ requirements (Wasserman, 1984). Hypothetically then, the AT, as defined by either the lactate threshold ($T_{lac}$), or the ventilatory threshold
21

(T_{vent}) [Weltman, 1995] could be the ultimate intensity at which an ultraendurance triathlete could perform at during the Ironman Triathlon.

2.5.1 Lactate Thresholds

The lactate concentration is approximately the same at rest in relatively fit adults, in normal sedentary subjects, and in adult patients with heart disease (Wasserman et al., 1994). But during exercise, the increase of lactate is inversely related to the physical fitness of the individual (Barlow et al., 1985). During incremental work, the lactate concentration increases initially very little until a distinct metabolic rate is reached at which lactate starts to increase steeply. This point is termed the lactate threshold (T_{lac}). The two prevalent theories underlying the T_{lac} are the fixed 4.0 mmol/L threshold and the interindividual lactate threshold, which occurs at different points for each individual. The reader is referred to Sjodin & Jakobs (1981) and Stegmann et al. (1981) respectively for an in depth review on each T_{lac}.

2.5.2 Ventilatory Thresholds

Above the T_{lac}, accelerated glycolysis increases muscle lactic acidosis. This acidosis is buffered primarily by bicarbonate which causes an increased alveolar CO\(_2\) output relative to O\(_2\) uptake (Wasserman et al., 1994). Davis et al. (1976) determined that gas exchange AT was a valid indirect method for the detection of the development of lactic acidosis during incremental exercise. Several ventilatory parameters have since been used to assess T_{vent}. These include \(\dot{V}_O_2_{max}\), ventilation (\(\dot{V}_E\)), excretion of carbon dioxide (\(\dot{V}CO_2\)), and the ratio of ventilation to maximum oxygen consumption (\(\dot{V}_E/\dot{V}O_2\)) [Davis et al., 1976; Black et al., 1998; Schneider et al., 1993; Loat & Rhodes, 1993; 1996; Anderson & Rhodes, 1989]. T_{vent} using the V-slope method (Schneider et
al., 1993) is determined as the point at which $\dot{V}CO_2$ departs from a line of linearity compared to that of $\dot{V}O_2$ and can be calculated using visual inspection (Frangolias & Rhodes, 1996) or computerized calculation (Black et al., 1998). Similarly, the $\dot{V}E/\dot{V}O_2$ breakaway point $T_{vent}$ calculation is determined as the point at which $\dot{V}E$ departs from a line of linearity compared to that of $\dot{V}O_2$ (Takano et al., 1991). This has also been proven to be a reliable measure of the AT (Caiozzo et al., 1982).

2.5.2.1 Relationship between Lactate and Ventilatory Thresholds

It is equivocal as to whether $T_{lac}$ and $T_{vent}$ occur at the same exercise intensities (Weltman, 1995). While $T_{lac}$ and $T_{vent}$ may occur at the same intensity (Simon et al., 1986), $T_{lac}$ has also been shown to occur at a higher relative exercise intensity to that of $T_{vent}$ (O'Toole et al., 1989). Therefore these two concepts might not necessarily be related by cause and effect (Simon et al., 1986; Tanaka et al., 1986).

Although there are many firm believers in the cause-and-effect relationship of $T_{vent}$ and $T_{lac}$ (Davis, 1985; Wasserman et al., 1994; Wasserman, 1984) some reviewers question the phenomena (Brooks, 1985; Stainsby, 1986; Walsh & Banister, 1988). Nevertheless, both $T_{lac}$ and $T_{vent}$ are widely used and generally accepted measures of endurance performance ability (Loat & Rhodes, 1993; 1997). At the very least, $T_{vent}$ provides important information concerning the relative level of $T_{lac}$.

2.5.2.2 Performance at Ventilatory and Lactate Thresholds

AT as indicated by either $T_{vent}$ or $T_{lac}$ improves with training (Hill et al., 1987) and when measured in the appropriate exercise mode has been related to swim, cycle and run performance in the triathlon (Sleivert & Rowlands, 1996). Hence, the ability of the triathlete to exercise at a higher percentage of $\dot{V}O_2_{max}$ for a given submaximal workload
is essential for triathlon success. This is influenced not only by \( \dot{V}O_2_{\text{max}} \) itself, but also by AT and economy of movement (Sleivert & Rowlands, 1996).

To illustrate the importance of training specificity and how it relates to the AT, Mazzeo & Marshall (1989) compared \( T_{\text{lac}} \) and \( T_{\text{vent}} \) in highly trained runners \((n=6)\) and cyclists \((n=6)\) during both treadmill running and cycle ergometry. Although \( \dot{V}O_2_{\text{max}} \) did not differ significantly for the cyclists during treadmill running and cycling, both \( T_{\text{lac}} \) and \( T_{\text{vent}} \) occurred at a relatively earlier workload during the treadmill run. They found the opposite was true for the runners (Mazzeo & Marshall, 1989). Triathlon is a unique sporting event requiring high relative ATs in both running and cycling. Therefore, training in all three disciplines is important to influence specific muscle groups causing increases in aerobic enzymes, subsequent increases in AT, and improved performance (Coyle, 1995). Strength training programs have also demonstrated significant improvements to both \( T_{\text{lac}} \) and cycling time to exhaustion at a constant work rate and are therefore recommended to ultraendurance triathletes as supplemental training (Marcinik et al., 1991, Tanaka & Swensen, 1998).

2.5.3 Time to Exhaustion at the Anaerobic Threshold

Laboratory assessments of cycling time to exhaustion at a subjects' AT have been reported to range from 48 to 255 minutes depending on the fitness of the athletes, the supply of exogenous fluid/CHO, and whether \( T_{\text{lac}} \) or \( T_{\text{vent}} \) was used to determine AT (Aunola et al., 1990; Davis et al., 1992; Loat & Rhodes, 1996). These studies indicate that the AT is too high of an intensity to be maintained during an UET, and that factors mentioned earlier (caloric balance, fluid homeostasis, and substrate shifts) cause detriment in prolonged performance. Thus, an optimum ultraendurance intensity,
different than the AT intensity, represented as a percentage of $\dot{V}O_2_{\text{max}}$ or AT, is needed to establish ultraendurance intensity guidelines.

2.6 Thresholds for Long-Distance Performance

It is equivocal whether or not $\dot{V}O_2_{\text{max}}$ and $T_{\text{vent}}$ are good predictors of triathlon performance. While some researchers have demonstrated a relationship (Zhou et al. 1997; Butts et al., 1991; Kohrt et al., 1987; De Vito et al., 1995), others have not (Dengel et al., 1989; O'Toole et al., 1989; 1996 Langill et al., 1993, unpublished thesis). These mixed findings may be due to the variability of athlete fitness, athlete experience, as well as uncontrollable environmental factors. This could also be due to the apparent effect that prior exercise has on each subsequent event during a triathlon. For example, it has been demonstrated previously that the AT can be reduced by the glycogen content of the muscles (Friedmann, 1998), substrate manipulation (Ivy et al., 1981; Glass et al., 1997), prior exercise (Black et al., 1984; Neary & Wenger, 1985) and extreme fatigue (Rowbottom et al., 1998). Likewise, rest and tapering have been shown to increase oxidative enzymes and muscle glycogen levels, and subsequently increase PO at $T_{\text{vent}}$ (Neary et al., 1992). Nevertheless, the threshold concept has been well researched and applied in each facet of the triathlon event; swimming, cycling, running, and the triathlon race as a whole.

2.6.1 Thresholds for Swimming

Swim times during a triathlon are not related to the physiological variables measured in maximal cycling and running tests (Zhou et al., 1997). Furthermore, resistance training does not appear to enhance swimming performance in untrained or competitive swimmers, despite substantial increases in upper body strength (Tanaka & Swensen, 1998). This is largely due to the specific skills and the efficient economy of
movement required for swimming (Sleivert & Rowlands, 1996). Swimming demonstrates lower values of $\dot{V}O_2^{\text{peak}}$, and peak $\dot{V}E$ versus cycling (Spinnewijn et al., 1996), which is most likely due to a decreased muscle mass utilization relative to cycling (McArdle, 1985; Medelli et al., 1993). Nevertheless, physical effort during the swim portion of a triathlon is very much required. Rating of perceived exertion (RPE) (Borg, 1982) has been shown to be reached at a lower percent of $\dot{V}O_2^{\text{max}}$ during swimming versus cycling (Spinnewijn et al., 1996) while blood lactate levels have been demonstrated to be highest in a triathlon during the swim portion (Lavoie, 1982; Pages et al., 1994; Farber et al., 1991).

### 2.6.1.1 Effects of Swimming on Cycling

Performance in swimming does not play the most important role in triathlon performance (Dengel et al., 1989; Margaritis, 1996). Swimming does however elicit a higher relative CHO oxidation rate than cycling during prolonged exercise at the same relative work intensity (Lavoie, 1982). This is primarily due to the larger percentage of fast-twitch muscle fibres that exist in the upper extremities relative to the lower extremities (Farber et al., 1991). Consequently, the preferred energy pathway of fast-twitch muscle fibres is that of glycolysis, which has a byproduct of lactic acid. Thus, it is logical to assume that an increased lactate concentration produced during the swim portion is utilized as substrate during the cycle portion of a triathlon (Farber et al., 1991; Brooks, 1986).

Nevertheless, swimming must still be considered to affect subsequent cycling and running bioenergetics (Hausswirth et al., 1996; 1997; Guezennec et al., 1996). Kreider and co-workers (1988) demonstrated that swimming significantly decreased triathlon cycling PO ($191 \pm 4.2$ to $159 \pm 7.6$ W) compared to control trials in nine male
triathletes during a simulated Olympic distance triathlon (see table 2). However, the mean PO of the group indicates that this triathlon population was a moderately trained group. Nevertheless, swimming prior to cycling produced significant differences \( (p < .05) \) in most of the measured physiological variables in this moderately trained group (Kreider et al., 1988). Interestingly, no significant difference in HR was observed between the control and triathlon cycling trials.

The effects of swimming on ensuing cycling performance in elite ultraendurance triathletes has not been published. However, we (Laursen et al., 1999) recently demonstrated that 3000m of swimming does not affect 3 h cycling performance (in terms of PO) in eight highly trained ultraendurance triathletes when a CHO/electrolyte beverage was consumed. Presumably the administration of this beverage offset some of the thermoregulatory differences that were apparent in Kreider et al. (1988a), enabling our subjects to maintain a similar PO compared to a control trial during a 3 h self-selected pace cycling time-trial.

Table 2. Physiological effects of 800m swimming prior to 75 min cycling during a simulated Olympic distance triathlon (Modified from Kreider et al., 1988a).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Cycling</th>
<th>Swimming prior to Cycling</th>
<th>Mean Difference</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cycling work output (W)</td>
<td>191 ± 4.2</td>
<td>159 ± 7.6</td>
<td>- 32</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>( \dot{V}O_2 ) (L-min(^{-1}))</td>
<td>3.18 ± 0.10</td>
<td>3.01 ± 0.11</td>
<td>- 0.17</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>( \dot{V}_E ) (L-min(^{-1}))</td>
<td>84.7 ± 4.4</td>
<td>80.4 ± 4.21</td>
<td>- 4.30</td>
<td>= 0.003</td>
</tr>
<tr>
<td>( \dot{Q} ) (L-min(^{-1}))</td>
<td>20.7 ± 1.2</td>
<td>18.9 ± 0.8</td>
<td>- 1.8</td>
<td>= 0.002</td>
</tr>
<tr>
<td>mean arterial pressure (mm Hg)</td>
<td>105 ± 3.8</td>
<td>96 ± 7.9</td>
<td>- 9</td>
<td>= 0.0001</td>
</tr>
<tr>
<td>Rectal temperature (°C)</td>
<td>38.2 ± 0.2</td>
<td>38.4 ± 0.3</td>
<td>+ 0.2</td>
<td>= 0.01</td>
</tr>
</tbody>
</table>
2.6.2 Thresholds for Cycling

The cyclist's ability to maintain an extremely high rate of energy expenditure for a long duration at a high economy of effort is dependent upon such factors as the individual's AT, muscle fibre type, muscle capillary density and certain anthropometric dimensions (Faria, 1992; Bishop et al., 1988; Coyle, 1995). While cycling time trial distances of 13.5 km, and 20 km have been reported to be performed at levels above the AT (Nichols et al., 1997), distances beyond 40-km have been demonstrated to be performed below the AT (O'Toole et al., 1987a; Hoogeveen & Schep, 1997). This evidence demonstrates that limitations to the AT concept exist and suggests the need to consider a concept that allows homeostatic exercise demands to be met optimally for prolonged periods at exercise intensities below the AT.

2.6.2.1 Effects of Cycling on Running

The effects that both the swimming and cycling segments of the triathlon have on subsequent running performance may be the most important factor to consider when looking at the overall triathlon performance. Marathon times during an UET are extremely variable and can range from approximately 2.5 hours, to over 6 hours (Docherty, 1998). Hence, the ability to run after prolonged swimming and cycling is important to overall triathlon performance, and depends on many factors. These include the effects of the prior exercises, as well as the fuel, hydration, and electrolyte status during the marathon. Kreider and co-workers (1988b) demonstrated that triathlon running performed at an identical control PO elicited significant changes in many physiological variables in nine moderately trained triathletes (Table 3).
**Table 3.** Physiological effects of 800m swimming, and 75 min cycling prior to a 40 min run during a simulated Olympic distance triathlon. Both control and triathlon runs were performed at identical workloads. (Data from Kreider et al., 1988b).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Running</th>
<th>Swimming and Cycling prior to Running</th>
<th>Mean Difference</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Rate (bpm)</td>
<td>161 ± 3.1</td>
<td>174 ± 3.6</td>
<td>+13</td>
<td>0.0001</td>
</tr>
<tr>
<td>$\dot{V}O_2$ (L·min$^{-1}$)</td>
<td>3.41 ± 0.1</td>
<td>3.85 ± 0.1</td>
<td>+0.44</td>
<td>0.001</td>
</tr>
<tr>
<td>$\dot{V}E$ (L·min$^{-1}$)</td>
<td>91.3 ± 3.3</td>
<td>104.2 ± 2.8</td>
<td>+12.9</td>
<td>0.0001</td>
</tr>
<tr>
<td>Arteriovenous $O_2$ Difference (ml·100ml$^{-1}$)</td>
<td>15.3 ± 0.2</td>
<td>17.2 ± 0.3</td>
<td>+1.9</td>
<td>0.002</td>
</tr>
<tr>
<td>Rectal temperature (°C)</td>
<td>38.3 ± 0.2</td>
<td>39.2 ± 0.3</td>
<td>+0.9</td>
<td>0.005</td>
</tr>
<tr>
<td>Stroke Volume (ml·min$^{-1}$)</td>
<td>138 ± 2.4</td>
<td>129 ± 3.6</td>
<td>-9</td>
<td>0.02</td>
</tr>
<tr>
<td>Mean arterial pressure (mm Hg)</td>
<td>102 ± 11.2</td>
<td>89 ± 5.5</td>
<td>-13</td>
<td>0.0003</td>
</tr>
</tbody>
</table>

Hence, prior swimming and cycling caused increases in HR, $\dot{V}O_2$, $\dot{V}E$, a-VO$_2$ difference, and rectal temperature, with decreases in SV and arterial pressure. De Vito and colleagues (1995) demonstrated $\dot{V}O_2$peak and $T_{vent}$ to be reduced by the first two segments of a triathlon (1.5-km swim, 32-km bike). $\dot{V}O_2$peak decreased from 69 to 64 ml·kg$^{-1}$·min$^{-1}$ while $T_{vent}$ decreased from 58 to 51 ml·kg$^{-1}$·min$^{-1}$. As well, prior swimming and cycling were reported to create physiological differences in subsequent running performance during simulated laboratory triathlons (Hausswirth et al., 1996; 1997; Guezennec et al., 1996). The explanation of these physiological effects include dehydration, fuel availability (glycogen), substrate utilization, muscle fatigue, cardiovascular fatigue and fluid compartment shifts (O'Toole & Douglas, 1995). Hence, the impairment of endurance performance induced by the first two segments of a triathlon is well established and is an important aspect to consider when observing triathlon run intensity. Ultimately, the ability to minimize the effects of the
aforementioned proposed mechanisms will hypothetically elicit an optimal triathlon run performance. These factors will be important elements to consider when establishing an ultraendurance threshold (UT).

2.6.3 Thresholds for Running

Numerous researchers have confirmed that the AT is related to endurance performance in running events ranging from 10 km to the marathon (Petit et al., 1997; Roecker et al., 1998; Rhodes & McKenzie, 1984; Perronnet et al., 1987). Farrel and associates (1979) demonstrated that runners set a race pace that allows the largest possible $\dot{V}O_2$ that just avoids the exponential rise in plasma lactate. Sjodin & Svedenhag (1985) provide further evidence showing that elite marathon runners run their races at an average pace equal to 86% $\dot{V}O_2_{\text{max}}$. This pace represented approximately 93% of their 4 mmol·L⁻¹ $T_{\text{lac}}$ (O’Toole & Douglas, 1995). Slower marathon runners (finishing times >3hrs) reportedly ran at 65% $\dot{V}O_2_{\text{max}}$. Therefore, elite marathon runners were able to sustain higher relative and absolute energy outputs leading to faster finish times. Hence, the capacity to sustain a high fractional utilization of $\dot{V}O_2_{\text{max}}$ during distance running is related to endurance running performance.

2.6.4 Thresholds for Triathlon

When assessing the AT in triathletes, it is necessary to take into consideration the type of ergometer used as different protocols have been reported to generate different values, usually running $\dot{V}O_2_{\text{max}}$ greater than cycling $\dot{V}O_2_{\text{max}}$ (Schneider & Pollack, 1991). The amount of muscle mass, the type and the distribution of active motor units involved in each exercise test might be at the origin of these differences (Medelli et al., 1993). AT also occurs at a lower percent of $\dot{V}O_2_{\text{max}}$ in cycling versus...
running, however, this may only suggest that triathletes have a greater potential for improvement in cycling. Improvement in event specific AT may be the most important factor to improved performance. Kohrt and co-workers (1989) reported increases of 6% and 10% in PO at $T_{\text{lac}}$ for cycling and running respectively without an improvement in $\dot{V}O_2$max. Hence, improvement in triathlon performance is dependent on the ability of the triathlete to utilize a greater percentage of $\dot{V}O_2$max.

Studies on longer duration triathlons have not demonstrated the same results. O'Toole and colleagues (1989) studied the relationship between exercise test variables and bike race times in 24 participants (14 men, 10 women) entered in the 1985 Hawaiian Ironman Triathlon. The researchers found that $\dot{V}O_2$ values at ATs were not highly related to bike finish times ($r = -0.26$ to $-0.58$). Furthermore, $\%\dot{V}O_2_{\text{peak}}$, HR, and $\%HR_{\text{max}}$ at thresholds were not related to bike finish times ($r = -0.01$ to $0.06$). This indicates the importance that other factors such as fuel and hydration homeostasis have on ultraendurance success. The sport of triathlon, however, has grown immensely since 1985 and the top triathletes are now more educated in their race strategy. Most triathletes understand the need for proper pacing, as well as the importance of hydration and refueling schedules during an ultraendurance event (Laursen & Rhodes, 1999). This has lead to increased Ironman triathlon performance times over the years (Docherty, 1998). Therefore, it would be valuable to repeat this research with today's ultraendurance triathlete.

### 2.6.5 The Use of Heart Rate Monitors to Measure Intensity in Ultraendurance Athletes

Because little scientific information is available for the use of HR monitors during performance, athletes fail to fully take advantage of physiologic information (O'Toole et
al., 1998). Although HR parameters measured in the laboratory might underestimate those in the field (Kenny et al., 1995), HR measured during a triathlon has been reported to be similar to HR at T_{vent} during a physiological assessment (Zhou et al., 1997; O'Toole et al., 1987a; Roalstad et al., 1987). Thus, HR monitoring may allow for a more precise control of intensity than that of subjective monitoring alone.

However, estimated maximal HRs are too variable to be of use to precisely guide training pace during cycling or running (Saldanha et al., 1997). Therefore, a laboratory physiological assessment is important for the ultraendurance triathlete to establish parameters such as HR_{max} and HR at T_{vent}. This provides the athlete with knowledge regarding the approximate HR intensities (or percent of HR_{max}) at which to train at, as well as limitations to long-duration race performance. O'Toole and associates (1998) suggests that during prolonged (> 6 h) cycling or running, a highly-trained athlete may expect to exercise at an average intensity close to 80% HR_{max}, but should also expect intensity to decline by 6-7% during the cycle ride or run. This decreased HR near the end of prolonged exercise suggests that intensity declines during ultraendurance events. This may occur due to substrate depletion, altered muscle efficiency, fluid and electrolyte imbalances, thermoregulatory problems, cardiac fatigue, as well as psychological factors (O'Toole & Douglas, 1995). Hence, the ability of the ultraendurance athlete to identify specific reasons for this intensity decline may be important for success in future ultraendurance endeavors (O'Toole et al., 1998).

Roalstad and colleagues (1987) reported that ultraendurance triathletes maintained average HRs of approximately 75% of their HR_{max} during the bike and run portions of the Hawaii Ironman. Furthermore, the better finishing times were found in triathletes where HRs fluctuated the least during the race. Nevertheless, much remains
to be clarified about the HR responses of ultraendurance athletes, particularly in relation to performance and the attainment of individual maximal potential.

2.7 A New Paradigm: The Ultraendurance Threshold (UT)

Indeed, the AT has been demonstrated to be a reliable measure to predict the ability of athletes to sustain a given work rate for a prolonged period (Roecker et al., 1998; Rhodes & McKenzie, 1984; Wasserman, 1984). The AT represents the upper limit of PO where lactate production and removal may attain equilibrium during constant load exercise (Rusko et al., 1986). Hypothetically then, the AT could be the optimum intensity at which an ultraendurance triathlete could perform at during the Ironman Triathlon. But this is truly not the case. Athletes have been shown to perform for up to 255 minutes at the AT when supplied intermittently with a CHO/electrolyte beverage (Davis et al., 1992). O'Toole and co-workers (1987a) demonstrated that ultraendurance triathletes perform at a pace below their AT. Consequently, other factors come into play then that inhibit the ability of the athlete to maintain their AT intensity. These include substrate utilization shifts, plasma volume decline, and muscle recruitment pattern shifts (Farber et al., 1991; O'Toole et al., 1987a).

As exercise increases in duration, CHO supply from glycogen stores decline and caloric demands require a greater contribution from fat and protein stores (Kreider, 1991). Subsequently, this decreases exercise intensity as fat oxidation reduces the availability of ATP relative to the oxidation of CHO (Gollnick, 1988). Evidence for this is shown as an increased $\dot{V}O_2$ during prolonged exercise. For example, O'Toole and associates (1987b) reported in 5 of 7 world class female ultraendurance triathletes that $\dot{V}O_2$ increased significantly during the first 2.5 hours of a 5-hour cycle. Because fat metabolism requires approximately 5% more oxygen to produce the same external
work, and the increase in $\dot{V}O_2$ was found to be 8%, this most likely accounts for some of the increase (O'Toole et al., 1987b). Further evidence for this substrate shift is shown from RER data (0.93 – 0.5hrs, 0.85 – 2.5 hrs, 0.82 – 5hrs).

Intensities near the AT require a significant contribution from CHO stores (Gollnick, 1988). It stands to reason that when CHO stores begin to diminish, fat oxidation increases to meet the body's energy demands, and subsequently intensity declines (O'Toole et al., 1987a). Furthermore, an increased intensity will produce higher lactate levels early on in performance. This racing technique has been demonstrated to reduce overall endurance performance (Firth, 1998; Wasserman, 1987). Hence, attempting to complete an UET at the AT would be detrimental to the overall performance. Although intensities at the AT represent the upper limit of PO where lactate production and removal obtain an equilibrium (Rusko et al., 1986), substrate equilibrium is out of balance (CHO oxidation > fat oxidation) for ultraendurance exercise. Therefore, the UT must take into consideration the limited glycogen storage capacity and limited provision of exogenous CHO due to finite gastric emptying rates (Tsintzas & Williams, 1998; Coyle & Montain, 1992), in an attempt to maintain a more balanced fuel utilization. Certainly, CHO utilization during the first two hours will be out of proportion, as glycogen stores are initially high, and glycogen promotes its own usage at high concentrations (Gollnick, 1988). However, beyond this two to three hour period of exercise, fueling contribution obtains more of an equilibrium (O'Toole et al., 1987b). Hence, the UT is an exercise intensity that maintains a more balanced fuel contribution leading to increased performance.

Dehydration decreases exercise intensity due to decreases in $\dot{Q}$. This can be prevented by fluid ingestion. Nevertheless, it seems that fluid compartment shifts also
occur during ultraendurance events resulting in decreases in SV (Farber et al., 1991). However, \( \dot{Q} \) might still be maintained by an increase in HR (O'Toole et al., 1987b). Hence, the UT might be affected by these compartment shifts even under homeostatic hydration conditions. Therefore, HR intensity would have to be adjusted accordingly to the individual's plasma volume response to prolonged exercise. More research is required to understand the fluid compartment shifts during prolonged ultraendurance exercise.

It has been shown that the neuromuscular system adapts during prolonged exercise by recruiting different muscle groups (O'Toole et al., 1987b). As the muscles begin to fatigue, technique and recruitment patterns can be altered to accomplish the same work output (Sjogaard et al., 1986). This adaptation changes the economy of motion requiring greater \( \dot{V}O_2 \) at the same PO (Sleivert & Rowlands, 1996; O'Toole et al., 1987b). These changes in muscle recruitment patterns also happen naturally during triathlons as exercise mode changes from swimming, to cycling, to running. The influence that the preceding event has on subsequent events has been demonstrated (Kreider et al., 1988; Laursen et al., 1999). Therefore, the UT intensity must take the preceding event into consideration. For example, running after prolonged cycling might change HR parameters due to the increased use of relatively rested muscle fibres during the initial stages of the marathon. Hence, just as \( \dot{V}O_2 \text{max} \) and AT are specific to the event protocol, so would be the UT. For example, Dengel and colleagues (1989) calculated event-specific %\( \dot{V}O_2 \text{max} \) for swimming, cycling, and running and found the percent \( \dot{V}O_2 \text{max} \) used during the submaximal tests was significantly related to swimming \((r=0.91)\), cycling \((r=0.78)\), and running \((r=0.86)\) times during a Half-Ironman triathlon. Hence, the UT is specific to each event.
When the majority of these possible hypotheses for the demise of the intensity at the AT are taken into account, a new intensity can be reestablished. This new intensity would have to take into consideration the aforementioned factors allowing for adequate substrate, fluid and electrolyte, and neuromuscular homeostasis to be met. Accordingly, I offer that a unique physiological paradigm be proposed for the ultraendurance athlete that accompanies the aforementioned considerations that the ultraendurance athlete encounters during prolonged ultra-distance exercise. This paradigm would be called the ultraendurance threshold, and would be defined as the optimum intensity that an ultraendurance athlete can perform at during an ultra-distance event.

2.8 Conclusions

This review has outlined considerations for the development of an UT where the UT could be defined as the optimum maintainable exercise intensity during ultraendurance performance. It has been outlined that the UT can be monitored by HR readings, and that after HR reaches a steady state, a slow but steady increase occurs representing the effects of CV drift. Over time, intensity may decline due to substrate shifts and neuromuscular fatigue by 6% - 7% (O'Toole et al., 1998). Hence, the graph of mean HR over time for ultraendurance athletes may appear similar to that of Figure 1.

The UT is a unique paradigm to other threshold concepts in that it must take into consideration the unique complications of prolonged ultraendurance exercise. These complications include substrate utilization, fluid and electrolyte homeostasis and the effects of dehydration and plasma volume shifts on cardiovascular function. Other considerations include the effect that the preceding event has on subsequent events, as
well as neuromuscular fatigue. These considerations will all affect the ability to maintain performance at the AT. Although, the UT will exist at an intensity below the AT, it may prove to be proportionate. For example, subjects with higher ATs may have higher UTs. Evidence for this concept has been previously provided. O'Toole and associates (1987a) demonstrated that a metabolic rate exists below the AT that defines an individual's maximal ultraendurance steady state for the cardiorespiratory and metabolic response to exercise. The study reported a moderate correlation ($r = 0.61; p < .05$) between percent $\dot{V}O_2_{peak}$ at 10W below mean $T_{vent}$ work rate and bike finish times in the Hawaiian Ironman. Hence, here lies evidence that an UT exists at a relative rate below the AT.

Consequently, further research is required to determine adequately whether a critical intensity exists relative to $\dot{V}O_2_{max}$ and the AT in ultraendurance triathletes. This intensity will also take into account the unique considerations of ultraendurance events. This relative intensity could be defined as the ultraendurance threshold.
Figure 1. A hypothetical plot of HR over time during an ultraendurance event (according to data suggested by O'Toole et al., 1998).

Hypothetical Plot of HR over Time During Ultraendurance Exercise

- Initial Rise in HR as the result of CV Drift
- Gradual decrease in HR due to substrate shift (increased fat oxidation relative to CHO)
- HR at AT
CHAPTER 3: METHODS AND PROCEDURES

3.1 Subjects

Eleven male ultraendurance triathletes were selected from the province of British Columbia to participate in this study. This sample size is similar to previous UET studies (O'Toole et al., 1987a; 1989; Farber et al., 1991). Subjects were solicited through an advertisement placed on the Ironman Canada web page (http://www.ironman.ca) and in the British Columbia Triathlon Association newsletter (TriBC Word). The inclusion criteria included a cycling $\dot{V}O_2\text{max}$ of score of 60 ml·kg$^{-1}$·min$^{-1}$, and having completed a minimum of one prior Ironman Triathlon distance event in under 12 h. Approximately the top 20% of all male finishers finish within this time period (Docherty, 1989). The exclusion criteria were (1) not being able to complete any of the testing procedures, (2) not being able to complete the bike portion of the Ironman Triathlon in less than six hours, or (3) not being able to complete the Ironman race. Before testing, all risks and benefits were thoroughly explained to the subjects and written informed consent was obtained in accordance with the Guidelines for Ethical Review at the University of British Columbia.

3.2 Testing Procedures

All laboratory testing was performed in the J.M. Buchanan Exercise Science Laboratory at the University of British Columbia. Subjects were asked to prepare for all testing in the same manner they would prepare for an ultraendurance race. Subjects were instructed to refrain from heavy exercise 24 hours prior to all tests. Individuals were informed of the intent of the study, hypotheses and experimental protocols. Prior to the first session, consent forms were signed, followed by baseline measures of height, weight and percent body fat (Going, 1996). During session #1 ($BiVO_2\text{max}$), a
maximal oxygen consumption test (\(\dot{V}O_{2\text{max}}\)) was performed on a cycle ergometer to determine whether or not athletes were to be included in the study, and also to establish threshold calculations.

Session #2 (Tr\(\dot{V}O_{2\text{max}}\)1) consisted of a \(\dot{V}O_{2\text{max}}\) test on the treadmill. A minimum of 48 hrs separated the \(\dot{V}O_{2\text{max}}\) tests from any subsequent test in an attempt to prevent any carry-over effects. Session #3 (Bi\(T_{\text{vent}}\)) consisted of a cycling ride to volitional fatigue at the athletes calculated PO at \(T_{\text{vent}}\) (PO\(_{T\text{vent}}\)). The main purpose of this test was to establish whether or not the AT is a reasonable intensity to be maintained during the cycle portion of the UET.

Session #4 (Bi\(\text{SSP}\)) consisted of an ultraendurance cycling 5 h ride at the athletes' self-selected PO performed on the cycle ergometer. Athletes were asked to mimic the pace that they would choose to ride at during the Ironman Triathlon bike course. PO was blinded to the athlete during both experimental sessions. A second treadmill run \(\dot{V}O_{2\text{max}}\) test (Session #5; Tr\(\dot{V}O_{2\text{max}}\)2) immediately followed Bi\(\text{SSP}\). The rationale for this test was to determine if running speed at \(T_{\text{vent}}\) could better predict marathon run performance during the Ironman compared to that of Tr\(\dot{V}O_{2\text{max}}\)1. The Bi\(T_{\text{vent}}\) trial and Bi\(\text{SSP}\) trial were completed in random order so as to avoid training effects.

Session #6 (Bi\(\dot{V}O_{2\text{max}}\)2), a cycle \(\dot{V}O_{2\text{max}}\) test, consisted of an identical procedure to that of Bi\(\dot{V}O_{2\text{max}}\)1 and took place approximately two weeks pre-event to ensure that \(\dot{V}O_{2\text{max}}\), \(T_{\text{vent}}\) and baseline measures did not change significantly compared to those of Bi\(\dot{V}O_{2\text{max}}\)1. During this test, hematological status was determined through measured hemoglobin (Hb) and hematocrit (Hct) levels.
Session #7 (IMC) consisted of HR measurements during the Canadian Ironman Triathlon, in Penticton, British Columbia, on August 29th, 1999. This was accomplished using computer downloadable portable HR monitors (Polar Vantage NV, Polar Electro, Finland).

3.3 Testing Protocols

During all laboratory testing sessions, expired gases were collected and analyzed using the Vmax metabolic cart (V6200, SensorMedics Corporation, Yorba Linda, CA). HR was measured using a Polar Vantage NV HR monitor (Polar Electro, Finland), and cycling PO was calculated using an electronically braked cycle ergometer (SensorMedics 800 Ergometer, SensorMedics Corporation, Yorba Linda, CA). All capillary blood samples were collected from fingertips in 25-μL heparinized capillary tubes. All sessions were preceded with an explanation of the testing procedures followed by a physiological warm-up with all the equipment in place against a light resistance load. This provided the subjects with an opportunity to become familiar with the equipment and the actual process for gathering data during the test. During the $\dot{V}O_2_{max}$ tests, volitional fatigue was defined by the following criteria:

1. The oxygen consumption ceases to increase linearly with rising workload and approaches a plateau or drops slightly, the last two values agreeing within $\pm 2 \text{ ml·kg}^{-1}·\text{min}^{-1}$.

2. Attainment of 90% of age predicted HR$_{max}$

3. Respiratory exchange ratio (RER) is greater than 1.10

$T_{vent}$ was calculated using the excess CO$_2$ (EXCO$_2$) elimination curve (Frangolias & Rhodes, 1996) by visual inspection by two independent reviewers for identification of
the PO and running speed at which CO\textsubscript{2} showed a sudden and sustained increase (i.e. \(T_{\text{vent}}\)). In the event of a disagreement, the \(\dot{V}_E/\dot{V}O_2\) curve (Takano et al., 1991) was used to clarify the actual threshold point.

**Session #1 – Bike \(1\) \(V_{O_2\text{max}}\) Test (Bi\(V_{O_2\text{max}}\))**

The electronically braked cycle ergometer was used with the protocol starting at an initial workload of 100W and having progressive increases of 30W each minute. Volitional fatigue was defined by either the inability to maintain a minimum cadence of 40rpm or the subject's own termination of the test.

**Session #2 - Treadmill \(V_{O_2\text{max}}\) Test (Tr\(V_{O_2\text{max}}\))**

This procedure involved a continuous (zero grade) treadmill run protocol started at an initial velocity of 3.5 mph and increased 0.5 mph each minute. After the attainment of 10 mph, the grade was increased by 2% each minute. Volitional fatigue was defined by the athlete no longer being able to maintain the treadmill velocity. This protocol created difficulties in representing peak speed numerically for statistical analysis. Therefore, time on the treadmill was used as a maximum speed/power value, called maximum treadmill time (MTT). From that, a percentage of maximum treadmill time (\%MTT) was calculated from the speed at which \(T_{\text{vent}}\) occurred (\%MTT-\(T_{\text{vent}}\)).

**Session #3 – Ventilatory Threshold Ride to Volitional Fatigue (Bi\(T_{\text{vent}}\))**

Baseline levels of weight, blood lactate (Lac') (Accusport, Boehringer Mannheim), blood glucose (Glu) (One Touch II, Lifescan Canada Ltd., Burnaby, B.C.), and hematocrit (Hct) were recorded. Hct determinations were made by centrifugation for 5 min using a microcapillary centrifuge (Drummond Scientific Co., Broomall, PA.). After warming up and becoming familiar with the testing procedures, the subject began riding
at PO_{Tvent} that was calculated during BiVO_{2max1}. After 5 min riding to obtain steady state, expired gases were collected for 5 min, and thereafter for 5 min at the top and bottom of each hour thereafter. When the athlete felt he could maintain his cadence for less than 5 min, a final gas collection procedure occurred until volitional fatigue. Lac−, Glu, and Hct were also recorded at the top of each hour of exercise, and at exhaustion. Borg's rating of perceived exertion (RPE) (Borg, 1982) was inquired every 30 min throughout. Subjects received unlimited access to water, and a CHO/electrolyte beverage (Shaklee Performance™) during this test to maximize their time to exhaustion (Davis et al., 1992). Quantities of each were recorded. Volitional fatigue was defined as the athlete unable to maintain a cadence of 40 rpm or the subject's own termination of the test. Weight was measured post-test to determine fluid loss/gain.

Session #4 – Five-Hour Cycling Time Trial at Self-Selected Pace (Bi_{SSP})

A minimum of one week separated the experimental trials. Bi_{SSP} protocol was identical to that used during Bi_{Tvent} with one exception; that being the ability to increase or decrease PO according to what the subject felt was a pace they would ride at during the Ironman Triathlon bike course. Although PO during the ride could be either increased or decreased at the request of the subject, the subject was encouraged to choose a PO that they would be challenged by, while at the same time being able to maintain for the 5 h duration. Because of the duration of Bi_{SSP}, and potentially Bi_{Tvent}, athletes were permitted to use the toilet as required. The required time off the bike was recorded and adjusted so that the subject would have to complete the full 5 h of cycling during Bi_{SSP}. Immediately following Bi_{SSP}, athletes performed a transition (standardized at 5 min) into run apparel to complete session #5 (TrVO_{2max2}), which was identical in procedure to that of BiVO_{2max1}. 
Session #6 – Bike VO_{2max} Test (BiVO_{2max}2)

A \dot{V}O_{2max} test, identical to the BiVO_{2max}1 occurred approximately two-weeks prior to IMC to ensure that \dot{V}O_{2max} and T_{vent} did not change significantly from BiVO_{2max}1 due to training effects. At this point, descriptive hematological parameters were determined in duplicate. Hemoglobin (Hb) concentrations were measured colorimetrically (540nm) after conversion to cyanmethemoglobin, using Sigma Diagnostics test reagents. (Sigma Diagnostic assay kit #525), while Hct determinations were made by centrifugation for 5 min. Descriptive measures of weight and body composition were measured again.

Session #7 – The Canadian Ironman Triathlon, Penticton, B.C. August 29, 1999 (IMC).

HR was recorded at 60-second intervals during the race using downloadable Polar HR monitors. Watches were removed from the athletes and information was downloaded at the earliest possible convenience. All timing during the race was done electronically by SportStats, the official timers for IMC.

3.4 Experimental Design and Data Analysis

A repeated measure analysis of variance (ANOVA) was used to compare the dependent variables, \dot{V}O_{2}, \%\dot{V}O_{2max}, \%T_{vent}, \%P_{O2vent}, RER, \dot{V}_{E}, PO, Lac\textsuperscript{-}, Glu, Hct, RPE, and pedaling rate (PR) during Bi_{Tvent} and Bi_{SSP}. The comparison of individual variables between trials was conducted using Hotelling T^2. HR during IMC was compared with HR during both Bi_{Tvent} and Bi_{SSP} using trend analysis to compare the pattern of change over time. A one-way ANOVA was used to compare the dependent variables \dot{V}O_{2max}, T_{vent}, HR at T_{vent} (HR_{Tvent}), percent of HR_{max} at T_{vent} (%HR_{max}T_{vent}),
\( \dot{V}_{E, \text{max}} \), and \( \text{RER}_{\text{peak}} \) for BiVO2max1, BiVO2max2, TrVO2max1 and TrVO2max2. When the ANOVA revealed a significant difference, Tukey's Post Hoc test was used to specify where the difference occurred. Hotelling \( T^2 \) was used to examine differences between the two run \( \dot{V}O_2_{\text{max}} \) tests (MTT, \( T_{\text{vent}} \) speed, %MTT-\( T_{\text{vent}} \)), and the two cycling \( \dot{V}O_2_{\text{max}} \) tests; peak PO (PPO), percent of PPO at \( T_{\text{vent}} \) (%PPO-\( T_{\text{vent}} \)), and PO at \( T_{\text{vent}} \) (PO\( T_{\text{vent}} \)). Pearson Product correlation examined the relationship between TrVO2max1 and TrVO2max2 predictions of IMC marathon run time, and actual IMC marathon run time. Pearson Product correlation matrix was also used to examine the relationship between BiVO2max1 and BiVO2max2 HR\( T_{\text{vent}} \) and mean HR during Bi\( T_{\text{vent}} \), BiSSP, and IMC. All statistics were run on SPSS 8.0 for Windows. The alpha level was set at .05. Results are expressed as a mean ± the standard error of the mean ( \( \bar{x} \pm \text{SEM} \)).

3.5 Diagram of Design

**Table 4. Time Line For Testing.**

<table>
<thead>
<tr>
<th>June 1 – 15</th>
<th>June 15 – 30</th>
<th>July 1 – 31</th>
<th>August 10 - 20</th>
<th>August 29</th>
</tr>
</thead>
<tbody>
<tr>
<td>BiVO2max1</td>
<td>Bi( T_{\text{vent}} )</td>
<td>BiSSP</td>
<td>BiVO2max2</td>
<td>IMC</td>
</tr>
<tr>
<td>TrVO2max1</td>
<td></td>
<td>TrVO2max2</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Figure 2. Timeline of Testing Procedure for Bi\( T_{\text{vent}} \) and BiSSP.**

*Unlimited access to water, CHO/electrolyte beverage, CHO bars throughout*
CHAPTER 4: RESULTS AND DISCUSSION

4.1 Results

4.1.1 Descriptive Measures

Ten of the eleven subjects in the study were focusing on competition at the 1999 Ironman Canada Triathlon (August 29th), while one subject, on a similar training regimen was focussed on competition in the 1999 Florida Ironman Triathlon (November 4th). This single subject's test data was only included for comparison of $\dot{V}O_2\text{max}$ results and experimental testing, however, when comparisons were made for IMC, his data was omitted from the analysis. Descriptive data of the subject pool is listed in Table 5. Hematological measures of oxygen carrying capacity (Hb and Hct) were within normal ranges (Bodary et al., 1999). Age was higher than in most Olympic distance triathlon studies (Bunc et al., 1996; Zhou et al., 1997; Sleivert & Wenger, 1993; Kreider et al., 1988a; 1988b) however, was typical of UET studies (O'Toole et al., 1987a; 1989). Training status during the testing period and prior to the 1999 Canadian Ironman Triathlon was $16.3 \pm 1.0 \text{ hr-wk}^{-1}$ ($2.7 \pm 0.5 \text{ hr-wk}^{-1}$ swimming, $8.5 \pm 0.6 \text{ hr-wk}^{-1}$ cycling, $4.5 \pm 0.5 \text{ hr-wk}^{-1}$ running). All subjects satisfied the criteria of having previously completed an Ironman distance race in less than 12 hr (mean time = $10.73 \pm 0.17$ hr).

Table 5. Descriptive measures: age, height (Ht), weight (Wt), percent body fat (%BF), hemoglobin (Hb), and Hematocrit (Hct).

<table>
<thead>
<tr>
<th>age (yr)</th>
<th>Ht (cm)</th>
<th>Wt (kg)</th>
<th>%BF (%)</th>
<th>Hb (g.dL$^{-1}$)</th>
<th>Hct (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>x</td>
<td>35.8</td>
<td>176.7</td>
<td>68.9</td>
<td>11.7</td>
<td>16.9</td>
</tr>
<tr>
<td>SEM</td>
<td>1.6</td>
<td>2.3</td>
<td>2.2</td>
<td>1.2</td>
<td>0.2</td>
</tr>
</tbody>
</table>
4.1.2. $\dot{V}O_{2\text{max}}$ Comparisons

The variables of HR, $\dot{V}O_2$, and PO were measured as an absolute value at $T_{vent}$ and as a relative percentage of their maximum (i.e. $T_{vent}/\dot{V}O_{2\text{max}}$) for $BiVO_{2\text{max}1}$, $BiVO_{2\text{max}2}$, $TrVO_{2\text{max}1}$, and $TrVO_{2\text{max}2}$. No significant differences were found during the $\dot{V}O_{2\text{max}}$ tests on the following variables: $\dot{V}O_{2\text{max}}$ absolute, $\dot{V}O_{2\text{max}}$ relative, $HR_{\text{max}}$, $HR_{T\text{vent}}$, $%HR_{\text{max}-T\text{vent}}$, $T_{\text{vent}}$ absolute, $T_{\text{vent}}$ relative (all $p > .05$; Table 6). However, the ANOVA revealed significant differences on $\dot{V}E_{\text{max}}$, and $RER_{\text{Peak}}$ ($p < .05$; Table 6). Tukey's post hoc test clarified the differences on $\dot{V}E_{\text{max}}$ to exist between $BiVO_{2\text{max}2}$ and both treadmill running $\dot{V}O_{2\text{max}}$ tests ($p < .05$), while the significant differences in $RER_{\text{Peak}}$ were between $TrVO_{2\text{max}2}$ and both cycling $\dot{V}O_{2\text{max}}$ tests ($p < .05$).
Table 6. Primary Variables. Comparison of the following variables over time between the maximal tests: absolute and relative oxygen uptake ($\dot{V}O_{2\text{max}}$), absolute and relative ventilatory threshold ($T_{\text{vent}}$), maximum heart rate ($HR_{\text{max}}$), heart rate at ventilatory threshold ($HR_{T_{\text{vent}}}$), percent of maximum heart rate at $T_{\text{vent}}$ ($\%HR_{\text{max}}$-$T_{\text{vent}}$), maximum ventilation ($\dot{V}_E_{\text{max}}$), and peak respiratory exchange ratio ($RER_{\text{peak}}$). (* = Significant difference between tests; $p < .05$).

| $\bar{x} \pm \text{SEM, N=11}$ | BiVO$_{2\text{max}}$ | BiVO$_{2\text{max}}$ | TrVO$_{2\text{max}}$ | BiSSP followed by TrVO$_{2\text{max}}$
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>$\dot{V}O_{2\text{max}}$ (L-min$^{-1}$)</td>
<td>4.57 ± 0.16</td>
<td>4.68 ± 0.16</td>
<td>4.74 ± 0.17</td>
<td>4.45 ± 0.19</td>
</tr>
<tr>
<td>$\dot{V}O_{2\text{max}}$ (ml·kg$^{-1}$·min$^{-1}$)</td>
<td>64.4 ± 1.0</td>
<td>67.5 ± 1.0</td>
<td>67.0 ± 1.4</td>
<td>63.3 ± 1.5</td>
</tr>
<tr>
<td>$T_{\text{vent}}$ (L-min$^{-1}$)</td>
<td>3.20 ± 0.12</td>
<td>3.30 ± 0.08</td>
<td>3.43 ± 0.13</td>
<td>3.24 ± 0.14</td>
</tr>
<tr>
<td>$T_{\text{vent}}$ (%$VO_{2\text{max}}$)</td>
<td>70.0 ± 1.4</td>
<td>70.8 ± 1.4</td>
<td>72.4 ± 1.1</td>
<td>73.2 ± 1.9</td>
</tr>
<tr>
<td>$HR_{\text{max}}$ (beats·min$^{-1}$)</td>
<td>179.2 ± 3.7</td>
<td>176.2 ± 3.3</td>
<td>185.3 ± 3.2</td>
<td>177.5 ± 2.2</td>
</tr>
<tr>
<td>$HR_{T_{\text{vent}}}$ (beats·min$^{-1}$)</td>
<td>150.3 ± 3.7</td>
<td>148.7 ± 3.4</td>
<td>151.4 ± 3.2</td>
<td>144.9 ± 2.6</td>
</tr>
<tr>
<td>$%HR_{\text{max}}$-$T_{\text{vent}}$</td>
<td>83.9 ± 1.6</td>
<td>84.5 ± 1.4</td>
<td>81.7 ± 1.2</td>
<td>81.7 ± 1.1</td>
</tr>
<tr>
<td>$\dot{V}<em>E</em>{\text{max}}$ (L·min$^{-1}$) *</td>
<td>171.1 ± 6.3</td>
<td>188.4 ± 6.4</td>
<td>161.4 ± 5.2</td>
<td>153.6 ± 4.9</td>
</tr>
<tr>
<td>$RER_{\text{peak}}$ *</td>
<td>1.15 ± 0.02</td>
<td>1.15 ± 0.02</td>
<td>1.10 ± 0.02</td>
<td>1.08 ± 0.02</td>
</tr>
</tbody>
</table>
No significant difference was found between PPO, $POT_{vent}$, and $\%PPO-T_{vent}$ between $BiVO_{2max1}$ and $BiVO_{2max2}$ ($p > .05$; Table 7). Furthermore, no significant difference was found between $\%MTT-T_{vent}$ ($p > .05$; Table 8). However, $TrVO_{2max2}$ $T_{vent}$ speed and MTT was significantly less than $TrVO_{2max1}$ ($p < .05$; Table 8).

Table 7. Within protocol comparison of measures during maximal cycle ergometer tests ($BiVO_{2max1}$ vs. $BiVO_{2max2}$): peak power output ($PPO$), power output at $T_{vent}$ ($POT_{vent}$), percent of peak power output at $T_{vent}$ ($\%PPO-T_{vent}$) (All $p > .05$).

<table>
<thead>
<tr>
<th>x ± SEM, N=11</th>
<th>$BiVO_{2max1}$</th>
<th>$BiVO_{2max2}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>PPO (W)</td>
<td>420 ± 18</td>
<td>437 ± 18</td>
</tr>
<tr>
<td>$T_{vent}PO$ (W)</td>
<td>274 ± 9</td>
<td>287 ± 9</td>
</tr>
<tr>
<td>$%PPO-T_{vent}$</td>
<td>65.7 ± 1.1</td>
<td>66.1 ± 1.4</td>
</tr>
</tbody>
</table>

Table 8. Within protocol comparison of measures during maximal treadmill run tests ($TrVO_{2max1}$ vs. $TrVO_{2max2}$): maximum treadmill time (MTT), speed at $T_{vent}$ ($T_{vent}$ Speed), $T_{vent}$ expressed as a percent of maximum treadmill time ($\%MTT-T_{vent}$). ($\dagger$ = Significant difference between tests; $p < .05$).

<table>
<thead>
<tr>
<th>x ± SEM, N=11</th>
<th>$TrVO_{2max1}$</th>
<th>$TrVO_{2max2}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>MTT (min) $\dagger$</td>
<td>16.25 ± 0.44</td>
<td>15.21 ± 0.44</td>
</tr>
<tr>
<td>$T_{vent}$ Speed (mph) $\dagger$</td>
<td>8.9 ± 0.3</td>
<td>8.4 ± 0.2</td>
</tr>
<tr>
<td>$%MTT-T_{vent}$ (%)</td>
<td>65.8 ± 2.1</td>
<td>64.2 ± 1.6</td>
</tr>
</tbody>
</table>
4.1.3. Experimental Findings (Bi\textsubscript{vent} vs. Bi\textsubscript{SSP})

During Bi\textsubscript{vent}, subjects #1, #5, and #11, could not perform at their PO\textsubscript{vent} for longer than one hour (13, 38, and 45 min respectively). Therefore, their results were omitted for the statistical comparison between Bi\textsubscript{vent} and Bi\textsubscript{SSP} (N=8). The endurance time (ET) for the Bi\textsubscript{vent} trial equated to 2 h 10.7 ± 18.6 min, whereas all subjects completed the 5 h Bi\textsubscript{SSP} trial.

In an attempt to decipher this apparent dichotomy (those that cycled for a long versus a short duration), the subject pool (N=11) was divided into those that performed less than 2 h (ET\textsubscript{low} = 49.1 ± 6.7 min; N=6), and those that performed greater than 2 h (ET\textsubscript{high} = 2 h 49.4 ± 12.1 min; N=5). Although a trend towards a higher CV and metabolic rate relative to T\textsubscript{vent} did exist in all parameters (ET\textsubscript{low} vs. ET\textsubscript{high}), the differences were not statistically significant (p > .05). Pearson Product Correlation Matrix identified %PO\textsubscript{vent} to have a significant relationship with ET (r = -.570; p < .05).

The submaximal rides were completed at an average PO of 273.6 ± 8.6 W and 188.0 ± 8.6 W which corresponded to 65.7 ± 1.1% and 45.3 ± 2.0% PPO for Bi\textsubscript{vent} and Bi\textsubscript{SSP} respectively (p < .05; Figure 3). Significant differences between trials existed on the following variables averaged over time: HR (Figure 5), \( \dot{V}O_2 \) absolute, %\( \dot{V}O_{2\text{max}} \), %T\textsubscript{vent}, %PO\textsubscript{Tvent}, RER, \( V_E \) (p ≤ .05; Table 9). \( \dot{V}O_2 \) did not significantly increase over time during Bi\textsubscript{SSP} (p > .05; Figure 8), however, RER significantly decreased over time (p < .05; Figure 9).
Table 9. Comparison of selected variables over time between the ventilatory threshold to volitional fatigue trial (Bi\text{vent}) and the five hour self-selected pace time trial (Bi\text{SSP}): oxygen uptake ($\dot{V}O_2$), percent of maximum oxygen uptake (%\(\dot{V}O_2\text{max}\)), percent of ventilatory threshold (%\text{T}vent), percent of \text{T}vent power output (%\text{POT}vent), percent of maximum heart rate (%HR\text{max}), and minute ventilation (\(V_E\)). (All \(p < .05\) between trials)\(\uparrow\ = \)significantly different over time; \(p < .05\); \(N = 8\)).

<table>
<thead>
<tr>
<th>Trial</th>
<th>5</th>
<th>30</th>
<th>60</th>
<th>Final</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\dot{V}O_2$ (L\cdot min(^{-1}))</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bi\text{vent}</td>
<td>$3.61 \pm 0.15$</td>
<td>$3.62 \pm 0.15$</td>
<td>$3.60 \pm 0.15$</td>
<td>$3.52 \pm 0.14$</td>
<td>$3.61 \pm 0.15$</td>
</tr>
<tr>
<td>Bi\text{SSP}</td>
<td>$2.46 \pm 0.12$</td>
<td>$2.64 \pm 0.12$</td>
<td>$2.74 \pm 0.12$</td>
<td>$2.73 \pm 0.06$</td>
<td>$2.64 \pm 0.09$</td>
</tr>
<tr>
<td>%(\dot{V}O_2\text{max})</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bi\text{vent}</td>
<td>$75.6 \pm 4.1$</td>
<td>$75.8 \pm 4.1$</td>
<td>$75.5 \pm 4.0$</td>
<td>$73.8 \pm 3.9$</td>
<td>$75.6 \pm 4.1$</td>
</tr>
<tr>
<td>Bi\text{SSP}</td>
<td>$51.6 \pm 5.0$</td>
<td>$55.2 \pm 4.6$</td>
<td>$57.5 \pm 4.3$</td>
<td>$57.1 \pm 2.2$</td>
<td>$55.4 \pm 3.5$</td>
</tr>
<tr>
<td>%\text{T}vent</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bi\text{vent}</td>
<td>$110.7 \pm 2.9$</td>
<td>$111.0 \pm 2.6$</td>
<td>$110.4 \pm 2.4$</td>
<td>$108.2 \pm 2.4$</td>
<td>$110.7 \pm 2.6$</td>
</tr>
<tr>
<td>Bi\text{SSP}</td>
<td>$75.9 \pm 3.9$</td>
<td>$81.2 \pm 3.5$</td>
<td>$84.3 \pm 3.0$</td>
<td>$84.8 \pm 4.6$</td>
<td>$80.5 \pm 3.3$</td>
</tr>
<tr>
<td>%\text{POT}vent</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bi\text{vent}</td>
<td>$100.0 \pm 0.0$</td>
<td>$100.0 \pm 0.0$</td>
<td>$100.0 \pm 0.0$</td>
<td>$100.0 \pm 0.0$</td>
<td>$100.0 \pm 0.0$</td>
</tr>
<tr>
<td>Bi\text{SSP}</td>
<td>$60.3 \pm 3.2$</td>
<td>$66.7 \pm 3.6$</td>
<td>$70.9 \pm 3.7$</td>
<td>$69.3 \pm 3.5$</td>
<td>$67.1 \pm 3.2$</td>
</tr>
<tr>
<td>RER $\uparrow$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bi\text{vent}</td>
<td>$0.98 \pm 0.01$</td>
<td>$0.95 \pm 0.01$</td>
<td>$0.95 \pm 0.01$</td>
<td>$0.91 \pm 0.02$</td>
<td>$0.95 \pm 0.01$</td>
</tr>
<tr>
<td>Bi\text{SSP}</td>
<td>$0.93 \pm 0.01$</td>
<td>$0.92 \pm 0.01$</td>
<td>$0.91 \pm 0.01$</td>
<td>$0.89 \pm 0.00$</td>
<td>$0.91 \pm 0.01$</td>
</tr>
<tr>
<td>$V_E$ (L\cdot min(^{-1})) $\uparrow$</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bi\text{vent}</td>
<td>$97.8 \pm 3.9$</td>
<td>$103.5 \pm 4.6$</td>
<td>$108.4 \pm 5.9$</td>
<td>$104.0 \pm 4.5$</td>
<td>$103.4 \pm 4.7$</td>
</tr>
<tr>
<td>Bi\text{SSP}</td>
<td>$60.9 \pm 2.9$</td>
<td>$66.4 \pm 3.2$</td>
<td>$69.2 \pm 2.6$</td>
<td>$70.1 \pm 2.5$</td>
<td>$66.7 \pm 2.8$</td>
</tr>
</tbody>
</table>
Hct and Lac were significantly different between BiTv and BiSSP over time (p ≤ .05; Table 10). However, Glu was not significantly different between trials or over time (p > .05; Table 10).

Table 10. Comparison of blood glucose (Glu), blood lactate (Lac), and hematocrit (Hct) levels over time during the ventilatory threshold to volitional fatigue trial (BiTv) and the five hour self-selected pace time trial (BiSSP): († = significantly different between trials; p < .05; N = 8).

<table>
<thead>
<tr>
<th>Trial</th>
<th>Rest</th>
<th>60</th>
<th>Final</th>
<th>Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glu (mmol·L⁻¹)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BiTv</td>
<td>5.9 ± 0.6</td>
<td>5.2 ± 0.2</td>
<td>5.6 ± 0.4</td>
<td>5.6 ± 0.4</td>
</tr>
<tr>
<td>BiSSP</td>
<td>6.3 ± 0.4</td>
<td>5.6 ± 0.3</td>
<td>5.3 ± 0.2</td>
<td>5.7 ± 0.3</td>
</tr>
<tr>
<td>Lac (mmol·L⁻¹) †</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BiTv</td>
<td>2.2 ± 0.3</td>
<td>6.7 ± 0.8</td>
<td>4.9 ± 0.4</td>
<td>4.6 ± 0.5</td>
</tr>
<tr>
<td>BiSSP</td>
<td>2.4 ± 0.4</td>
<td>2.8 ± 0.4</td>
<td>4.0 ± 0.9</td>
<td>3.1 ± 0.6</td>
</tr>
<tr>
<td>Hct (%) †</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BiTv</td>
<td>43.3 ± 0.4</td>
<td>44.0 ± 0.7</td>
<td>44.4 ± 0.8</td>
<td>43.9 ± 0.6</td>
</tr>
<tr>
<td>BiSSP</td>
<td>42.8 ± 0.5</td>
<td>43.1 ± 0.7</td>
<td>40.9 ± 0.6</td>
<td>42.3 ± 0.6</td>
</tr>
</tbody>
</table>

Significant differences between trials also existed on the following variables averaged over time: RPE = 16.6 ± 0.4 vs. 13.5 ± 0.5; pedaling rate (PR) = 70.7 ± 3.7 vs. 82.5 ± 2.0 rpm for BiTv vs. BiSSP respectively (p ≤ .05; figure 4). Trend analysis revealed RPE, HR, V̇E, and Lac to increase significantly over time, while PR and RER
decreased significantly over time (p < .05). Furthermore, PR, $\dot{V}O_2$, Lac', and Hct demonstrated significant interaction effects (p < .05), therefore displaying different patterns of change over time.

Subjects lost 0.5 ± 0.2 and 0.3 ± 0.3 kg during Bi$_{Tvent}$ and Bi$_{SSP}$ respectively which was not significantly different between trials or from baseline measurements (p > .05). Subjects ingested 1.99 ± 0.52 L and 4.92 ± 0.33 L of fluids which provided 175.7 ± 42.5 g and 498.8 ± 25.0 g of CHO, or in relative terms, 14.9 ± 1.6 and 3.5 ± 0.2 ml·kg$^{-1}$·hr$^{-1}$, and 1.4 ± 0.1 and 14.1 ± 0.4 g·kg$^{-1}$·hr$^{-1}$ CHO for Bi$_{Tvent}$ and Bi$_{SSP}$ respectively (p < .05).

4.1.4. Ironman Canada (IMC) Performance Results

Subjects completed the Ironman Canada (IMC) performance in a time of 677.2 ± 15.9 min (swim = 67.3 ± 1.7 min, bike = 331.3 ± 3.7 min, run = 270.4 ± 12.2 min). Of all the physiological measures, only PPO during BiVO$_{2max2}$ correlated significantly with IMC performance ($r = -.567$, p < .05). Using the treadmill velocity at $T_{vent}$ during TrVO$_{2max1}$ and TrVO$_{2max2}$, an estimated time (min) and pace (mph) for the marathon component was developed and compared to the actual time and pace for the triathlon (Table 11). An explanation of the development of the run estimation is presented in Appendix A.

Table 11. Estimated and actual run times and paces for the marathon run portion of the Ironman Canada Triathlon (IMC).

<table>
<thead>
<tr>
<th>Estimated from TrVO$_{2max1}$</th>
<th>Estimated from TrVO$_{2max2}$</th>
<th>Actual IMC Marathon Run Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time (min)</td>
<td>Pace (mph)</td>
<td>Time (min)</td>
</tr>
<tr>
<td>178.0 ± 5.3</td>
<td>8.9 ± 0.3</td>
<td>189.2 ± 4.7</td>
</tr>
</tbody>
</table>
4.1.5. Heart Rate Comparisons between Trials

In order to compare the HR responses between Bi_Tvent, Bi_SSP, and IMC, the sample size was reduced to N = 5 due to either the subject not completing a minimum of 60 min on the Bi_Tvent trial, or loss of HR data due to HR monitor malfunction. As before, comparison between the three performance trials could only be made for 60 min. HR showed significant differences between trials, over time, and interactively (trial x time) (p < .05; Figure 5).

HR_Tvent during both cycling VO_{2max} tests (BiVO_{2max1} and BiVO_{2max2}) demonstrated significant relationships with mean IMC HR, Bi_SSP HR, and Bi_Tvent HR (Table 12). However, non-significant relationships were demonstrated in amongst the three performance trials (p > .05). Table 12 displays the comparison of HR_Tvent between the mean HR during each of the three performance trials. Interestingly, HR during IMC most closely matched HR_Tvent during BiVO_{2max2} (%diff = -1.1 ± 1.5%), and correlated strongly (r = .866; p < .05).

Figure 6 compares the HR response between Bi_SSP and five hours of the 180-km IMC cycle portion. Because of the loss of data due to HR monitor malfunction, the sample size for HR comparison between the trials was reduced to N = 7. HR was significantly different between the trials (p < .05; Figure 6), however, not over time (p > .05). Furthermore, there was a significant interaction effect (trial x time; p < .05).
Table 12. Comparison of mean heart rate (HR), mean percent of maximum heart rate (%HR\text{max}), mean difference (diff) and mean percent difference (%diff) during the performance trials in relation to the graded cycling \( \dot{V}O_2 \text{max test} \) (Bi\( \dot{V}O_2 \text{max} \)). The correlation \((r)\) to Bi\( \dot{V}O_2 \text{max}1 \) and Bi\( \dot{V}O_2 \text{max}2 \) are also presented. (\( \dagger \) = Significant correlation; \( p < .05 \)).

<table>
<thead>
<tr>
<th></th>
<th>IMC (N=7)</th>
<th>Bi\text{T}vent (N=11)</th>
<th>Bi\text{SSP} (N=11)</th>
</tr>
</thead>
<tbody>
<tr>
<td>x HR</td>
<td>146.3 ± 2.4</td>
<td>159.2 ± 2.9</td>
<td>129.9 ± 3.7</td>
</tr>
<tr>
<td>x %HR\text{max}</td>
<td>84.6 ± 1.3%</td>
<td>88.9 ± 0.7%</td>
<td>72.5 ± 1.3%</td>
</tr>
<tr>
<td>x diff (bpm)</td>
<td>-2.2 ± 2.7</td>
<td>+8.9 ± 2.9</td>
<td>-20.3 ± 2.7</td>
</tr>
<tr>
<td>x %diff</td>
<td>-1.1 ± 1.5%</td>
<td>+5.0 ± 1.6%</td>
<td>-11.4 ± 1.5%</td>
</tr>
<tr>
<td>Bi( \dot{V}O_2 \text{max}1 )</td>
<td>( r = .873 \dagger )</td>
<td>( r = .825 \dagger )</td>
<td>( r = .770 \dagger )</td>
</tr>
<tr>
<td>Bi( \dot{V}O_2 \text{max}2 )</td>
<td>( r = .866 \dagger )</td>
<td>( r = .662 \dagger )</td>
<td>( r = .799 \dagger )</td>
</tr>
</tbody>
</table>

Figure 3. Comparison of power output (PO) over time for the ventilatory threshold ride to volitional fatigue trial (Bi\text{T}vent) versus the five hour self-selected pace cycling time trial (Bi\text{SSP}). Significantly different between trials (\( p < .05 \)).

![PO over Time Graph](image-url)
Figure 4. Comparison of pedaling rate (PR) over time for the ventilatory threshold ride to volitional fatigue trial (BiTv) versus the five hour self-selected pace cycling time trial (BiSSP). Significantly different between trials and over time (p < .05).

Figure 5. Comparison of heart rate (HR) over time for the ventilatory threshold ride to volitional fatigue trial (BiTv), the five hour self-selected pace cycling time trial (BiSSP), and the cycling portion of the Ironman Canada (IMC). All significantly different between trials and over time (p < .05).
Figure 6. Comparison of heart rate (HR) over time for the five hour self-selected pace cycling time trial (BiSSP), and the first 300 min of the cycling portion of the Ironman Canada (IMC). Significantly different between trials ($p < .05; N = 7$).

![IMC Cycle Portion vs. BiSSP over Time](image)

Figure 7. Comparison of power output (PO), heart rate (HR), and pedaling rate (PR) over time for the five hour self-selected pace cycling time trial (BiSSP), ($N = 11$).

![Comparison of PO, HR, and PR over Time for BiSSP trial](image)
Figure 8. Hct over time during BiSSP (significant decrease over time; p < .05).

Figure 9. RER over time during BiSSP (significant decrease over time; p < .05).
Figure 10. 1999 Ironman Canada Bike Course Profile (Wolf Infosystems, 1999).

Figure 11. HR over time during the 1999 Ironman Canada (N=7).
4.2 Discussion

Ultraendurance performance is an endeavor in which the athlete attempts to adjust the factional utilization of \( \dot{V}O_2 \) during performance in order to obtain maximal benefits (i.e. the greatest speed) without exceeding a critical PO level that cannot be maintained for the duration of the event. Therefore, it is not surprising that athletes, coaches, and exercise physiologists have investigated various strategies to try to establish an optimal exercise intensity sustainable during training and race performances. Although controversy does exist with respect to detection, nomenclature, interpretation, and mechanisms (Brooks, 1985; Davis, 1985) strong relationships have been demonstrated between AT and endurance performance. The AT has demonstrated a relationship with endurance cycling (Coyle et al., 1991; Coyle, 1995; Bishop et al., 1998) endurance running (Rhodes & McKenzie, 1984; Petit et al., 1997; Farrel et al., 1979; Perronnet et al., 1987), as well as Olympic distance triathlon performance (Zhou et al., 1997). However, in longer duration events, this strong relationship between the AT and performance begins to decline (O'Toole et al., 1989; Langill and Rhodes, 1993; Miura et al., 1997). Nevertheless, ultraendurance triathletes will still strive to perform at some critical intensity, which might be relative to the AT (O'Toole et al., 1987a).

4.2.1 Descriptive Measures

The subjects in this study appear to be similar to other groups of triathletes (Zhou et al., 1997; Sleivert & Wenger, 1993; O'Toole et al., 1987a; 1989; Kreider et al., 1988a; 1988b). The subjects were slightly older than in most Olympic distance triathlon studies (Bunc et al., 1996; Zhou et al., 1997; Sleivert & Wenger, 1993; Kreider et al., 1988a; 1988b), however, they were comparable to UET studies (O'Toole et al., 1987a; 1989).
This may reflect the physiological and psychological experience it takes to compete over longer, more demanding distances. Height, weight, and percent body fat were also within expected ranges (O'Toole et al, 1987b). The training status of the triathletes prior to IMC was $8 \pm 2 \text{ km-wk}^{-1}$ swimming, $270 \pm 18 \text{ km-wk}^{-1}$ cycling, and $50 \pm 5 \text{ km-wk}^{-1}$ running. This represented $2.7 \pm 0.5 \text{ hr-wk}^{-1}$ swimming, $8.5 \pm 0.6 \text{ hr-wk}^{-1}$ cycling, and $4.5 \pm 0.5 \text{ hr-wk}^{-1}$ running, respectively, which is consistent with other UET studies (O'Toole, 1989; O'Toole et al., 1989).

### 4.2.2 $\dot{V}O_2\text{max}$ Comparisons

The AT has been demonstrated to be reduced by the glycogen content of the muscles (Friedmann, 1998), substrate manipulation (Ivy et al., 1981; Glass et al., 1997), prior exercise (Black et al., 1984; Neary & Wenger, 1985) and extreme fatigue (Rowbottom et al., 1998). Likewise, rest and tapering have been shown to increase oxidative enzymes and muscle glycogen levels, and subsequently increase $PO$ at $T_{\text{vent}}$ (Neary et al., 1992). Hence, our findings of non-significant differences in $\dot{V}O_2\text{max}$ and $T_{\text{vent}}$ between $\text{TrVO}_2\text{max}1$ and $\text{TrVO}_2\text{max}2$ (both relative and absolute) were surprising. These findings disagree with those of De Vito et al. (1995) where both $\dot{V}O_2\text{peak}$ and $T_{\text{vent}}$ were significantly reduced by the first two segments of a short-course triathlon (1.5-km swim, 32-km bike). This disparity may be related to differences in cycling intensity prior to the treadmill $\dot{V}O_2\text{max}$ test. In all likelihood, the BiSSP trial would be performed at a lower exercise intensity than in De Vito et al.'s (1995) 32-km bike time trial due to differences in duration. Hence, it is possible that the lower intensity during BiSSP in the present study coupled with CHO ingestion during the ride may have lead to glycogen synthesis in some active (and inactive) muscle fibers (Palmer et al., 1999). This would
allow for the availability of adequate glycogen stores to produce similar excess CO₂ for
the detection of T₉vent. A non-significant RERₚₑᵃᵏ (p > .05) between TrVO₂max₁ and
TrVO₂max₂ further supports this hypothesis. Nevertheless, a declining trend did exist
(Table 6). VO₂max declined (although not statistically) from 4.74 ± 0.17 to 4.45 ± 0.19
L·min⁻¹, while T₉vent declined (although not statistically) from 3.43 ± 0.13 to 3.24 ± 0.14
L·min⁻¹. TrVO₂max₁ to TrVO₂max₂ respectively. Furthermore, TrVO₂max₂ MTT and
T₉ventSpeed were significantly reduced relative to TrVO₂max₁ (p < .05), indicating that the
BiSSP trial did have some effect. TrVO₂max₂ T₉vent estimation subsequent to BiSSP
significantly reduced T₉ventSpeed, however, only marginally (TrVO₂max₁ T₉vent = 8.9 ± 0.3
mph, TrVO₂max₂ T₉vent = 8.4 ± 0.2 mph; p < .05). This reduction did not improve the
ability to predict IMC marathon run pace (IMC marathon = 5.9 ± 0.3 mph; r = .425; p > .05). One limitation to this finding, however, is that TrVO₂max₁ occurred first in time
order in comparison to TrVO₂max₂. This was to ensure that subjects were familiar with a
running treadmill VO₂max test prior to engaging in that activity subsequent to BiSSP.
Hence, there exists the potential for a training effect to have occurred, TrVO₂max₁ to
TrVO₂max₂, that could have lessened the magnitude of these results.

Treadmill running will generally elicit a greater VO₂max score than cycle ergometry
due to an increase in active muscle mass (McArdle et al., 1986; Medelli et al., 1993).
While VO₂max and T₉vent were not significantly different between cycling and running (p > .05), there was a percent difference in VO₂max equal to 3.9% (BiVO₂max₁ vs. TrVO₂max₁),
which is consistent with other triathlon study's (Kohrt et al., 1987; O'Toole et al., 1987b;
Kreider et al., 1988a; Schneider et al., 1990). Hence, our finding of a modest decline, in
comparison to single sport athletes, where the maximums are 9 to 11% less (Roalstad, 1989; Faulkner et al., 1971), is likely due to training specificity (O'Toole, 1989).

4.2.3 Relationship of Threshold to $Bi_{Tvent}$ and $Bi_{SSP}$

The AT is defined as the $\dot{V}O_2$ during exercise above which there is a sharp increase in anaerobiosis where lactate accumulation exceeds removal (Wasserman, 1984). This may represent the upper limit of PO during constant load exercise (Rusko et al., 1986). Strong relationships have been demonstrated between AT and endurance performance. For example, Rhodes & McKenzie (1984) demonstrated a strong correlation ($r = 0.94$) between run velocity at $T_{vent}$ and marathon performance time. However, in longer duration events, this strong relationship between the AT and performance begins to decline (O'Toole et al., 1989; Langill and Rhodes, 1993; Miura et al., 1997). For example, Langill and Rhodes (1993) demonstrated a moderate correlation ($r = 0.76$) between run velocity at $T_{vent}$ and IMC marathon performance using the identical protocol. As well, O'Toole et al. (1989) determined that the relationship between exercise test variables and bike race times in the 1985 Hawaiian Ironman Triathlon were not highly related to bike finish times ($r = -0.26$ to -0.58). $\%\dot{V}O_2{}_{peak}$, HR, and $\%HR_{max}$ at thresholds were also not related to bike finish times ($r = -0.01$ to 0.06). This decreased relationship may be due to the apparent effect that prior exercise has on each subsequent event during a triathlon (Kreider et al., 1989a; Laursen et al., 1999). It may also be due to the variability of athlete fitness, athlete experience, as well as uncontrollable environmental factors. Nevertheless, it makes sense to assume that the ultraendurance triathlete will still strive to perform at some critical intensity, which might be relative to the AT.
4.2.3.1 BiTv̇ent Trial

The purpose of the experimental trials was to test the hypothesis that ultraendurance triathletes perform at Tv̇ent during the bike portion of the Ironman triathlon. This hypothesis seemed questionable as previous AT cycles to exhaustion have demonstrated times to volitional fatigue ranging from 48 to 255 min at a PO corresponding to AT (Aunola et al., 1990; Davis et al., 1992; Loat & Rhodes, 1996). Hence, this seemed too great an intensity to be maintained during the bike portion of an UET. Fuel requirements and thermoregulatory considerations (Kreider, 1991; Laursen and Rhodes, 1999) would likely prevent most athletes from performing at this PO intensity for the duration necessary.

Of the 11 athletes, three subjects could not perform the BiTv̇ent trial for longer than 60 min (32 ± 5.1 min) and were subsequently removed from the statistical analysis. The remaining subjects (N=8) performed at their POTv̇ent for 2 h, 10.7 ± 18.6 min, whereas all subjects completed the 5 h Bissp trial. With the exception of Glu, significant differences existed on all measured variables when comparing BiTv̇ent to Bissp (Table 9, 10, Figure 3-5). Hence, this data suggests that ultraendurance triathletes do not choose to perform at the PO that corresponds to Tv̇ent on a ramped cycling protocol.

In retrospect, it may have been more appropriate to use a fixed HR or V̇O₂ instead of a fixed PO. V̇O₂ and HR during BiTv̇ent were both higher than V̇O₂Tv̇ent (+298 ± 62ml) and HRTv̇ent (+9 ± 3 bpm) during BiVO₂maẋ1. Hence, the actual intensity during BiTv̇ent corresponded to 110.7 ± 2.6% VO₂Tv̇ent, and 104.8 ± 2.1% HDTv̇ent, or 7.3 ± 1.5% above V̇O₂Tv̇ent, and 3.8 ± 1.7% above HRTv̇ent. These results bring to question the ability of a 30 W-min⁻¹ ramped cycling protocol to accurately calculate the PO that corresponds to whole body Tv̇ent. It might be suggested that a slower ramped PO
protocol would result in a more accurate determination, however, Hoogeveen & Schlep (1997) recently performed this (beginning at 2.5 W·kg\(^{-1}\) and increasing by 40W every 4 min) and the predictive value of the plasma lactate response in relation to 40km cycling performance was negligible. Conversely, running studies have shown excellent relationships between AT and performance (Petit et al., 1997; Roecker et al., 1998; Rhodes & McKenzie, 1984; Perronnet et al., 1987; Roecker et al., 1998). These physiological disparities are unexplainable, but it has been suggested that a ramp protocol during cycling might alter, in some unknown way, the classical mechanism of diffusion of Lac\(^-\) and/or H\(^+\) through the sarcolemma (Chicharro et al., 1997) causing a lag time in Lac\(^-\) or EXCO\(_2\) detection. In any case, \(P_{O_{T_{vent}}}\) was clearly above the levels of the metabolic and CV \(T_{vent}\) markers. When exercise occurs above \(T_{vent}\), \(\dot{V}O_2\) does not level off, but continues to rise for several minutes until either a delayed but elevated steady state is attained, or exhaustion occurs (Jones et al., 1999). This additional increase in \(\dot{V}O_2\) has been referred to as the \(\dot{V}O_2\) slow component (Barstow and Mole, 1991; Xu and Rhodes, 1999), and may be the result of an increased recruitment of Type II motor units during heavy exercise (Barstow et al., 1996). Hence, questions exist regarding the specific meaning of "exercise intensity". If exercise intensity refers to whole-body physiological stress, this might be reflected metabolically in \(\dot{V}O_2\), and cardiovascularly in HR (Jeukendrup & Van Diemen, 1998). Conversely, exercise intensity could also refer to the mechanical PO of the athlete (McLellan & Cheung, 1992). However, as has been demonstrated, these two concepts do not necessarily run parallel during a ramped cycle ergometry \(\dot{V}O_2\)\(_{max}\) test (Boulay et al., 1997). While ultraendurance athletes may perform at a metabolic or CV intensity that corresponds to \(T_{vent}\), it is unlikely based on the present findings and that of others (Aunola et al., 1990;
Davis et al., 1992; Loat & Rhodes, 1996) that ultraendurance triathletes perform at their 
$P_{O_{vent}}$

Boulay et al. (1997) recently demonstrated decreases in $P_{O}$, $\dot{V}E$, $\dot{V}CO_{2}$ and Lac when HR was kept constant at 5 bpm below $HR_{vent}$ for 90 min. Hence, these findings and ours, appear to refute the critical power concept. Critical power represents the highest metabolic rate at which a steady state response can be achieved during prolonged exercise (Monod & Schierrer, 1965), and may be similar to the $P_{O_{vent}}$ (Moritani et al., 1981). This may explain why three subjects could not perform past 60 min at $P_{O_{vent}}$, and why three others could not perform past 80 min. Hence, important interindividual differences must exist when performing to volitional fatigue at $P_{O_{vent}}$. Furthermore, the large variability subject to subject (range = 12.0 to 3 h 39.6 min) demonstrates the limitation of the $EXCO_{2} T_{vent}$ determination during a graded cycling $\dot{V}O_{2\text{,max}}$ test to predict a homogenous ET (at least in ultraendurance triathletes). A trend did exist towards a higher CV and metabolic rate relative to $T_{vent}$ in $ET_{low}$ versus $ET_{high}$, although this was not statistically significant ($p > .05$). It is tempting to speculate that athletes in the $ET_{low}$ group may have been overtrained at that particular time in the testing, as testing took place during training for an UET. Urhausen et al. (1998) noted a 27% decrease in time to volitional fatigue at 110% of the IAT in athletes that were in an overtrained state. Furthermore, it has been reported that 65% of all endurance athletes develop symptoms of overtraining at some time in their competitive career (Margan et al., 1987; see also review by McKenzie, 1999). Nevertheless, the potentiality of overtraining to explain some of the variance in ET is purely speculative, and this variance is likely related also to other factors.
4.2.3.2 BiSSP Trial and the Ultraendurance Threshold (UT) Hypothesis

The BiSSP trial was performed at an intensity equating to 55.4 ± 3.5% \(\dot{V}O_{2\text{max}}\) which compares almost identically to the lab based 5 h ultraendurance cycling rides of O'Toole et al. (1987a), but that their cycle was followed by an identical self-selected pace 3 h treadmill run. Interestingly, the self-chosen pace of O'Toole et al.'s (1987a) male triathletes (N=8) was 6 mph, which resembles the 5.9 ± 0.3 mph IMC marathon pace in the present study. Perhaps one limitation to this method of self-selected pace is that adjustments of intensity do not rely on physiological measures, but on the athlete's perception of the effort. Hence, we cannot clearly ascertain that the intensity chosen during BiSSP would be representative of the cycle portion of IMC. Nevertheless, in a performance setting, athletes will chose to perform according to their perception of the effort. This perception of effort during BiSSP equated to a RPE score of 15.0 ± 0.4 during the final measurement, indicating that the athletes perceived this effort to be considerable.

Athletes chose a significantly higher PR when cycling during BiSSP (82.5 ± 2.3 rpm) than when cycling at the PO\(T\text{vent}\) (62.9 ± 3.7 rpm) (p < .05; Figure 4). This increased PR in BiSSP may be a result of a reduction in the activation of the vastus lateralis muscle (Takaishi et al., 1994) due to the decrease in force application and connected to a reduction in \(\dot{V}O_2\) (Hagberg et al., 1981). In the present study, PR was increased 31.1% in BiSSP vs. Bi\(T\text{vent}\) (p < .05), whereas \(\dot{V}O_2\) was reduced by 26.8% (p < .05). Therefore the higher observed PR may be associated with a reduction in the forces applied on the pedals, the latter eliciting the recruitment of a higher percentage of slow-twitch fibers which have a lower recruitment threshold, a larger oxidative capacity, and a better mechanical efficiency for contraction (Takaishi et al., 1994; Coyle et al.,
1991). Hence, in the BiTvent trial, the progressive recruitment of additional motor units and/or the higher firing rate of recruited motor units was required to compensate for the deficit in contractility provoked by the alteration of fatigued motor units (Takaishi et al., 1994). This would help explain the lower chosen PO intensity during the BiSSP versus BiTvent. Lower Lac- values obtained during BiSSP (p < .05; Table 10) also suggest that the UT pace requires the majority of metabolism in its aerobic component, enabling triathletes to save energy to continue their prolonged effort either cycling or running. By reducing exertion in the cycling phase, triathletes may be more willing to push themselves in the running segment. This has been recently demonstrated in short-course triathletes, where drafting during the cycle enabled increased performances during the final run phase (Hausswirth et al., 1999). This was demonstrated in the present study as TrVO₂max2 was not significantly different than TrVO₂max1 in terms of VO₂max (p > .05), indicating that the UT pace still enabled a significant contribution from glycolytic metabolism to produce subsequent Lac- (as indicated by EXCO₂) during TrVO₂max2.

RER data further supports the UT hypothesis whereby fuel oxidation (CHO relative to fat) is more in equilibrium. During earlier ultraendurance laboratory studies (O'Toole et al., 1987b), RER demonstrated a large decline (0.93 to 0.82). Our study demonstrated a modest decline (Figure 9). Presumably, the ingestion of adequate quantities of fuels and fluids would aid this balance (Palmer et al., 1999). Most notably, O'Toole et al. (1987b) allowed fluids only during their 5 h cycles, whereas in the present study, triathletes were permitted any types of fuels or fluids. High CHO "sport gels" were used, in addition to solid CHO and CHO/electrolyte beverages. The comparison of O'Toole et al.'s (1987a) RER data to the present study suggests that the large quantities of these high CHO sources (14.1 ± 0.4 g·kg⁻¹·hr⁻¹ CHO) allowed for an
increase in CHO oxidation, presumably from blood glucose (Palmer et al., 1999). This further helps to explain the non-significant $\text{VO}_2\text{max}$ scores in $\text{TrVO}_2\text{max}2$ versus $\text{TrVO}_2\text{max}1$. This finding may represent the increase of applied physiological knowledge in ultraendurance triathletes.

4.2.3.3 Hematological Comparisons

Resting hematological measures of Hb and Hct were both higher than previously reported in UET studies. Hb levels (16.9 ± 0.2 g-dL$^{-1}$) were higher than in sedentary norms (Berne and Levy, 1983), which is unusual in ultraendurance triathletes. In fact, O'Toole et al. (1988) reported anemia (Hb < 14g-dL$^{-1}$) in 39% of male Ironman triathletes. Hct levels in the present study (43.9 ± 0.4%) were within normal ranges but were somewhat higher than the 42.5% reported in a large sample size by O'Toole et al. (1999). Normally, ultraendurance triathletes exhibit a pseudoanemia, which is generally attributed to a training-induced increase in plasma volume which is greater than that in the red cell volume (Convertino, 1991). The reasons for these hematological differences is unknown. Hct levels significantly declined during BiSSP yet increased significantly during BiVent, thus demonstrating a significant interaction effect ($p < .05$; Figure 8, Table 10). These findings fall in line with O'Toole et al. (1999) where although Hct generally increases due to a decrease in plasma volume with prolonged exercise, the variability is quite large (range = -7.1 to +10.0%). Hence, it appears that some individuals hemoconcentrate during triathlons while others hemodilute (O'Toole et al., 1999). Our findings of a significant interaction suggests that intensity and duration may play a factor in hemodynamics (Wells et al., 1987). Fluid dynamics, fluid intake, sodium loss, and intra/extravascular proteins may also play a role (Neutians and Gaehgten, 1994).
4.2.4 IMC Performance Results and Comparisons to Laboratory Trials

The mean finishing time of subjects in IMC was 11.3 ± 0.3 h, which was slower than the mean of their personal best times of 10.7 ± 0.2 h. Of the ten subjects entered in IMC, two achieved personal best times, one was 15 min off his personal best, and seven were greater than 30 min off their personal best. This increase was mainly attributable to the run where the average time was 32.1 ± 11.0 min greater than their IMC marathon personal best, while previous swim and bike times were nearly identical to their personal bests. Large variability occurred during the latter portion of the marathon run (Figure 11) where successful triathletes were able to maintain a high HR intensity, while less successful triathletes were not. Subjective comments for the decline in marathon time included gastrointestinal complaints, extreme leg cramping and even an anaphylactic reaction from a bee sting. IMC marathon run times strongly correlated with IMC overall times ($r = .982; p < .05$), suggesting that the run phase of the UET is critical to successful performance in experienced ultraendurance triathletes. Nevertheless, overexertion during the swim and cycle phases has the potential to create problems during the run phase (Kreider et al., 1989a; Laursen et al., 1999).

HR response during the triathlon (Figure 6 and 11) supports O'Toole et al.'s (1987a) finding that ultraendurance triathletes perform the bike portion of the Ironman at the HR corresponding to $T_{vent}$. Although CV drift was evident during $B_{SSP}$, it was not during IMC (Figure 6 and 11), as HR obtained its peak during the swim portion only 30 min into the race. This is most likely due to psychological factors due to the excitement of the day (Selley et al., 1995). This finding, however, is contrary to previous studies that have demonstrated the horizontal position in swimming combined with water immersion to elicit lower HRs compared to cycling and running at the same relative
intensity (Shaffrath & Adams, 1984; Nielsen et al., 1984). Nevertheless, HR during IMC began its slow decline after 30 min, and by the cycle portion, fluctuated around $BiVO_{2\text{max}}$ $HR_{T\text{vent}}$. These HR fluctuations tracked the IMC elevation chart (Figure 10), with the two distinguishable peaks in HR (Figure 6, 11) matching the two major elevation climbs (Richter Pass and Yellow Lake). Furthermore, as the race progressed, HR readings declined ($p < .05$). Mean HR during the run segment declined 5.5% compared to the cycle phase, similar to the 6 to 7% decline in HR intensity suggested by O'Toole et al. (1998) during ultraendurance exercise performed greater than 6 h. As previously mentioned, this decline may reflect substrate depletion, altered muscle efficiency, fluid and electrolyte imbalances, thermoregulatory problems, cardiac fatigue, as well as psychological factors (O'Toole & Douglas, 1995). The longer the competition distance, the more substrate availability and glycogen storage quantity are considered to be performance-determining factors (Roecker et al., 1998). Substrate utilization was altered significantly during BiSSP ($p < .05$) and may have been further altered had a swim preceded it (Laursen et al., 1999). Declining thermoregulatory and CV efficiencies while running attributable to prior swimming and cycling has also been documented (Kreider et al., 1988b).

Both Roalstad et al. (1987) and O'Toole et al. (1987a) independently reported that the cycling portion of the Hawaii Ironman is performed at an exercise intensity of approximately 75% of $HR_{\text{max}}$. O'Toole et al. (1987a) also found that 75% $HR_{\text{max}}$ corresponded to $T_{\text{vent}}$ suggesting that athletes perform the cycling portion of the Ironman at an intensity which approximates $HR_{T\text{vent}}$. Our subjects performed at $84.6 \pm 1.3%$ $HR_{\text{max}}$ which closely matched $HR_{T\text{vent}}$ during $BiVO_{2\text{max}}$ ($\text{%diff} = -1.1 \pm 1.5%$), and showed a strong relationship ($r = .866; P < .05$). Hence, over the course of the IMC bike phase, our triathletes performed at the HR intensity that corresponds to their
Therefore, although the PO_{vent} did not match the physiological T_{vent} measures, HR_{vent} does seem to be a realistic average HR to perform at during the cycle portion of the Ironman triathlon. Unfortunately, we can not conclude that this is an "optimal" HR intensity to maintain during an UET as only two of ten subjects achieved a personal best time. It is interesting to note, however, that one of these subjects achieved the highest relative \( \dot{V}O_{2max} \) score, suggesting that a high \( \dot{V}O_{2max} \) is required to maintain a high exercise intensity for prolonged periods (Sleivert & Rowlands, 1996). However, this field based HR intensity may not be representative of a lab-based intensity. HR has been demonstrated to be higher during a race when compared to HR at the same running speed under non-competitive laboratory controlled conditions (Selley et al., 1995). Hence, the psychological fact of being in a race environment versus a controlled lab environment can alter physiological responses to exercise. In the current study, HR during Bissp was significantly less than HR during the cycle portion of the IMC (\( p < .05; \) Figure 6), and subjects were asked to mimic their Ironman cycle intensity. Furthermore, it has been estimated that for every 1% loss in body mass due to dehydration, HR increases by 7 bpm (Coyle and Montain, 1992). Therefore, in a hot environment (i.e. 22.4 to 30.2 °C during the bike portion of IMC), dehydration/plasma volume shifts can change the HR-intensity relationship drastically. This would explain further the difference between these two trials. Hence, if athletes use HR monitors as a gauge of their racing pace, they may perform at a lesser PO than expected during a race, should a racing target HR be calculated on a HR determined during training or a physiological assessment (Lambert et al., 1998).

CV drift occurs with increasing exercise duration (Lambert et al., 1998). Therefore, HR during the later stages of the IMC cycle portion might not be indicative of the expected PO. If indeed HR remained relatively constant during the IMC bike phase,
we might expect the PO to decline. This decrease in PO was recently demonstrated by Boulay et al. (1997) where HR was kept constant at 5 bpm below $T_{vent}$ during a 90 min performance trial. This trend was demonstrated during Bi_{SSP} as HR increased significantly over time ($p < .05$), while PO was shown to decline somewhat after 2h 10 min (Figure 7). Another difference that exists between IMC and Bi_{SSP} is the relatively steady PO during Bi_{SSP} versus the undulating PO during IMC due to terrain and wind. However, Palmer et al. (1999) recently demonstrated non-significant metabolic and CV differences in an undulating PO cycle versus a steady state PO ride; each eliciting identical mean POs. Nevertheless, certain factors have been shown to change submaximal HRs during cycling. These include bike setup (Jeukendrup & Van Diemen, 1998) and the aerodynamic (aerobar) cycling position (Sheel et al., 1996).

4.2.5 Practical Implications

One of the purposes of this thesis was to provide some practical information for ultraendurance triathletes. It has been demonstrated previously (O'Toole et al., 1987a; Roalstad et al., 1987), and now in an experienced and well-trained group that ultraendurance triathletes perform at a mean HR intensity which is close to $HR_{Tvent}$. Therefore, it may be valuable for an ultraendurance triathlete to obtain a physiological assessment including a cycling $\dot{V}O_{2\text{max}}$ test in order to establish $HR_{Tvent}$. Hence, if uncertainties in HR intensity exist during the cycle portion of the race, an observation of a portable HR monitor may provide some confidence that the athlete is performing near optimal physiological performance. In this sense, HR monitors may provide a more accurate index of exercise intensity than speed (Jeukendrup & Van Diemen, 1998). Environmental and physiological factors such as wind, air temperature, air density, humidity and terrain have a large impact on cycling speed (Jeukendrup & Van Diemen, 1998). In fact, HR monitors may even represent 'ergogenic aids' (Lambert et al., 1998).
as they can motivate athletes to work at high intensities at or above $T_{\text{vent}}$ (Jeukendrup & Van Diemen, 1998).

Finally, this study has demonstrated the importance of the run portion on overall triathlon performance. Hence, training for UETs should focus on the run phase, and specifically on running subsequent to cycling. It is hoped this study will provide important information to the ultraendurance triathlete, coach, and exercise physiologist intent on improving UET performance.
CHAPTER 5: SUMMARY AND CONCLUSION

5.1 Summary

Physiological measurements such as economy of motion, $\dot{V}O_2\text{max}$, and the AT have been demonstrated to predict endurance performance. However, when these concepts are extended to ultraendurance sports (i.e. > 4 h), predictability of these measurements declines (O'Toole et al., 1989; Langill and Rhodes, 1993; Miura et al., 1997). The potential reasons for this decline are numerous and include: fuel utilization, fluid and electrolyte homeostasis, the effects of dehydration and plasma volume shifts on CV function, the effect that the preceding component has on subsequent components, as well as neuromuscular fatigue. (Farber et al., 1991; O'Toole et al., 1987a; 1987b; Kreider, 1991). Nevertheless, it is logical to assume that ultraendurance triathletes will attempt to maintain some critical intensity during the UET, which may similarly be related to $T_{vent}$ (O'Toole et al., 1987a). The findings from the present study demonstrate that ultraendurance triathletes do not perform at their $POT_{vent}$. ET during $Bi_{Tvent}$ was 2 h 10.7 ± 18.6 min, with great variability (range = 12.0 min to 3 h, 39.6 min). This variability creates questions about the ability of a 30 W-min$^{-1}$ ramped protocol to estimate the AT as defined as the upper limit of PO where Lac$^-$ production and removal attain equilibrium during constant load exercise (Rusko et al., 1986). $Bi_{Tvent}$ was performed at 7.3 ± 1.5% above $\dot{V}O_2\ T_{vent}$, and 3.8 ± 1.7% above $HR_{Tvent}$, suggesting limitations of the EXCO$_2$ elimination curve to estimate a $POT_{Tvent}$ equating to metabolic and CV $T_{vent}$. Conversely, $Bi_{SSP}$ was performed at 18.5 ± 2.9% below $\dot{V}O_2\ T_{vent}$ and 16.4 ± 2.0% below $HR_{Tvent}$. Hence, the critical intensity chosen by ultraendurance triathletes during a five hour cycling time trial is performed so that metabolism is largely in its aerobic component. Nevertheless, the ramped protocol did find considerable
agreement between the HR_{Tvent} during BiVO_{2max2} and the mean HR performed at during the cycle portion of IMC (-1.1 ± 1.5% difference, r = .866; p < .05). This relationship has been previously demonstrated in other UET studies (O'Toole et al. 1987a; Roalstad et al. 1987a).

The present study has also demonstrated the limitation of a lab based UET cycling simulation to resemble a similar effort in the field (IMC). The significant difference in HR measurement (129.9 ± 3.7 vs. 146.3 ± 2.4 bpm; Bi_{SSP} vs. IMC respectively; p < .05) is most likely due to the psychological effects of a race environment (Selley et al., 1995), potential effects of the swim prior to cycling (Kreider et al., 1988a; Laursen et al., 1999), as well as the increased effects of dehydration and plasma volume shifts due to increasing temperature resulting in a CV drift (Coyle & Montain, 1992). Nevertheless, the final RPE measurement during the Bi_{SSP} trial was 15.0 ± 0.4, indicating that the athletes perceived this effort to be considerable. TrVO_{2max2} T_{vent} estimation subsequent to Bi_{SSP} significantly reduced T_{vent} Speed, however, only marginally (TrVO_{2max1} T_{vent} = 8.9 ± 0.3 mph, TrVO_{2max2} T_{vent} = 8.4 ± 0.2 mph; p < .05). This did not significantly improve the ability to predict IMC marathon run pace (IMC marathon = 5.9 ± 0.3 mph; r = .425; p > .05). These findings are in opposition to those demonstrated in an Olympic distance triathlon (De Vito et al., 1995), and are likely related to differences in intensity and duration (Roecker et al., 1998).

From a CV standpoint, ultraendurance triathletes perform the second phase of an UET at their HR_{Tvent}. Metabolically (in terms of the fractional utilization of VO_{2}) and mechanically (in terms of PO), it appears they do not. A number of confounding variables serve to increase HR to the level of HR_{Tvent} during IMC, however, it is unlikely that the fractional utilization of VO_{2} and subsequent PO, follow a similar pattern.
5.2 Conclusions

1. The ET for Bi_{vent} trial equated to 2 h 10.7 ± 18.6 min, whereas all subjects completed the 5 h Bi_{SSP} trial indicating that ultraendurance triathletes do not perform the Ironman bike phase at their PO_{vent}. As well, significant differences between Bi_{vent} and Bi_{SSP} existed on all physiological measures except Glu.

2. HR_{vent} during BiVO$_2$max-2 was similar to IMC mean HR (-1.1 ± 1.5% difference) and showed a strong relationship (r = .866; p < .05).

3. A treadmill run \(\text{\hat{V}O}_2\text{max} (\text{Tr}VO_{2}\text{max}2)\) subsequent to Bi_{SSP} significantly reduced T_{vent}Speed (8.9 ± 0.3 mph vs. 8.4 ± 0.2 mph; p < .05), however, did not improve the ability to predict IMC marathon run pace (IMC marathon = 5.9 ± 0.3 mph; r = .425; p > .05).

5.3 Recommendations

It would be valuable to repeat the Bi_{vent} portion of this study by having subjects perform to volitional fatigue at their \(\text{\hat{V}O}_2\text{T}_{\text{vent}}\), and at their HR_{vent}, thus adjusting PO accordingly, similar to the study protocol of Boulay et al. (1997). This would determine if indeed ultraendurance triathletes are able perform metabolically at a level of intensity corresponding to T_{vent} during the second phase of an UET.
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APPENDIX A

Sample calculation for the estimation of $T_{vent}$ in either the running or cycling $V_{O_{2\max}}$ tests.

The corresponding time at the breakaway point in the graph of excess CO$_2$ vs. time was used to determine $T_{vent}$ point. This point in time would correspond to a specific cycling power output, or a specific treadmill velocity.

For example, in this case: minute 13 = 10.0 mph