PHYSIOLOGICAL AND METABOLIC RESPONSES TO CONSTANT AND VARIABLE LOAD CYCLING PERFORMANCE

Thesis submitted for the degree of Doctor of Philosophy (Medical Physiology)

by

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GARRY STANLEY PALMER

DEDICATION

For Dad

I wanted you to see this

and for Liz

I love you

Shoestring **By Rob and Dr Jack**

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DECLARATION

I, ***Garry Stanley Palmer,*** do hereby declare that the experiments presented in this thesis were conceived and executed by myself and, apart from the normal guidance from my supervisors, I have received no assistance.

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

This thesis is presented in fulfilment of the requirements for the degree of PhD.

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Date: 2/2/99

ABSTRACT

The experiments described in this thesis comprise a series of related, yet independent investigations examining the physiological and metabolic responses of well-trained amateur cyclists under conditions designed to mimic actual competitive situations, during individual and mass start races. In Section A the physiological responses to constant load and steady state exercise are determined. In Section B, the metabolic factors associated with constant and variable load cycling performance are examined.

The aim of the first investigation in Section A was to monitor the physiological responses of seven competitive cyclists (peak oxygen uptake [$\dot{V}O_{2peak}$] 5.16 ±0.32 L/min; peak power output [PPO] 398 ±22 W: values mean ±SD) during a four day (d) stage race, consisting of a 15 km time-trial (TT), a mass start 110 km road race (RR1), an individual 5.5 km hill climb (HC), and a second mass start road race over 105 km (RR2). Within 10 d of the final race, cyclists underwent laboratory testing for the determination of $\dot{V}O_{2peak}$, PPO, and peak heart rate (HR_{peak}). Comparisons of the HR responses to each race revealed that the individual events were performed at a relatively high and constant work rate (91.1 ±2.5% and 93.2 ±4.7% of HR_{peak} measured in the field for the TT and HC respectively). In contrast, despite similar racing speeds (42.2 ±1.0, 39.9 ±0.2, and 40.6 ±0.5 km/h for the TT, RR1, and RR2, respectively), the HR responses to the longer mass-start races were more random in both frequency and amplitude, and were reduced to 81.9 ±9.6% and 78.6 ±8.9% of HR_{peak}. Such stochastic changes in HR were seemingly unrelated to course terrain but were more related

to the group dynamics of the cyclists riding in the bunch. The results of this study reveal the stochastic nature of bunch cycle racing and show that the HR responses of competitive cyclists are more a function of tactical bunch riding than of the prevailing terrain.

The second study of this thesis determined the reliability of the performance of well-trained cyclists to laboratory simulated race conditions. Six subjects (PPO 437.2 ±31.0 W) performed a random order of three 20 km and three 40 km TT on their own bikes mounted on a Kingcycle ergometer. The time taken for the laboratory simulated 20 km and 40 km TT rides were highly reproducible (coefficient of variation [CV] 1.1 ±0.9% and 1.0 ±0.5%, respectively). However, the mean power output and heart rate were significantly different ($P<0.0001$) between the 20 km and 40 km TT (327.5 ±16.9 versus 303.9 ±14.9 W and 171.4 ±5.1 versus 168.3 ±4.4 beats/min, respectively). A strong relationship ($r^2 = 0.995$ and 0.996, $p < 0.001$, for the 20 km and 40 km TT respectively) was observed between the mean cycling time and the average sustained power output. The results of this investigation show that simulated laboratory TT over 20 and 40 km undertaken by trained cyclists are highly reproducible, and that race conditions can be reliably simulated under laboratory conditions.

Although reliability of a laboratory test is an important factor in deciding the utility of a test, the validity of that measure is also important. Accordingly, the third study of this thesis compared laboratory TT performances and actual race performances. Eight well-trained cyclists (PPO 450.0 ±37.8 W) performed three simulated 40 km TT in the laboratory and two 40 km TT races under competitive conditions. A significant correlation ($r^2 = 0.96$, $p < 0.001$) was found between laboratory and road race TT, with the road race being on average 8% slower

(group mean times: 56:24 ±3:59 (n=8); 61:26 ±4:25 (n=7); and 60:12 ±2:49 min:s (n=6) for the laboratory and two road TT respectively). These results indicate that the Kingcycle ergometry system can be used by sports scientists as both a valid and reliable method of assessing the physiological responses of well-trained cyclists during 20 km and 40 km time trials under controlled laboratory conditions.

Although simulation of TT events in the laboratory setting now makes it possible to determine the metabolic responses to such exercise, there have been no attempts to replicate the stochastic physiological responses of riders participating in mass start cycle races. Accordingly, in Chapter Seven, of this thesis, a laboratory based protocol was designed in an attempt to replicate the physiological responses to the stochastic exercise pattern utilised by cyclists in mass-start road races, and subsequently assess the effects of stochastic versus steady-state exercise on subsequent cycling TT performance. Six competitive cyclists ($\dot{V}O_{2peak}$ 4.83 ±0.42 L/min; PPO 432 ±39 W) undertook a random order two 150 min paced rides which were either constant load (SS) (58 % of PPO) or variable intensity (VI) (58 ± 12.2 % of PPO). These rides were immediately followed by a 20 km TT performance on the Kingcycle ergometer. Although HR during VI mirrored that previously reported in mass start road races in the field, the *mean* HR responses throughout the 150 min paced rides were not significantly different between the two conditions (153 ± 6 and 150 ± 11 beats/min; 79.6 ± 3.2% and 77.3 ± 5.9% of HR_{peak}, respectively). However, the time taken for the subsequent 20 km TT was significantly faster (26:32 ±1:30 vs. 28:08 ±1:47 min:s, $P<0.05$), and the mean power output was significantly greater (340.3 ± 44.2 vs. 302.5 ± 42.3 W; 77.8 ±10.2 vs. 70.0 ±9.8 % of PPO, $P<0.05$) following 150 min of SS compared to the VI cycling.

Having established the reliability and validity of a laboratory based testing ergometer in Section A, the two studies in Section B of this thesis examine the metabolic factors associated with constant and variable load cycling performance. In the first study in Section B, the efficacy of carbohydrate (CHO) ingestion on the performance of a 20 km TT was investigated in 14 well-trained cyclists ($\dot{V}O_{2peak}$ 4.52 ±0.60 L/min; PPO 400.4 ±46.6 W) who performed a random order of two 20 km TT consuming a bolus of 8 ml/kg BM of either a CHO (6.8 g/100 ml CHO) or placebo (PLA) beverage, immediately before the timed ride.

The ingestion of CHO did not improve performance times for the 20 km compared to the placebo (27:41 ±1:39 vs. 27:41 ±1:39 min:s). As would be expected from identical TT times, the HR responses (170.7 ±6.5 vs. 171.1 ±5.1 beats/min) and mean power outputs (311.7 ±40.1 vs. 311.5 ±38.1 W) for CHO and PLA respectively were also similar. The results of this study indicate that CHO availability does not limit high intensity cycling performance lasting ~30 min.

As no metabolic measurements were taken in any of the previously described studies, it was not possible to elucidate the potential mechanism(s) that might have been responsible for the observed differences in TT performance. Accordingly, the final investigation of this thesis compared the effects of prolonged (140 min) SS and VI exercise on metabolism and subsequent 20 km TT performance in 6 well-trained cyclists ($\dot{V}O_{2peak}$ 4.48 ±0.48 L/min; PPO 401 ±44.7 W). The *average* power output throughout SS was maintained at 58% of PPO, while VI was 58 ±12% of PPO. Throughout the rides, which were conducted in a random order, subjects ingested a total of 14 g/kg of a 5 g/100 ml U-^{14}C labelled glucose solution for the determination of the rates of plasma glucose oxidation

(Glu_{ox}). Muscle biopsies were obtained from the vastus lateralis before and after 140 min of exercise. Average $\dot{V}O_2$ (3.08 ±0.06 vs. 3.15 ±0.08 L/min) and HR (156 ±3.3 vs. 160 ±2.7 beats/min) were similar between trials as were the subjective ratings of perceived exertion (RPE) (12.6 ±1.7 vs. 12.7 ±1.5 units or SS and VI, respectively). Plasma [glucose] was well maintained throughout both trials (SS 5.6 ±0.2, VI 5.7 ±0.5 mM), despite a gradual decline in [insulin] from 34.3 ±13.8 and 37.8 ±16.7 after the first 10 min to 20.3 ±7.3 and 18.9 ±7.7 μU/ml during the final 10 min of exercise for SS and VI respectively ($P<0.05$). [FFA] increased significantly ($P<0.05$) during the 140 min ride from 0.13 ±0.11 to 0.35 ±0.18 in SS and 0.10 ±0.04 to 0.37 ±0.16 mM in VI. The area under the plasma [lactate] vs. time curve was significantly greater during VI than SS (29.1 ±9.6 vs. 24.6 ±9.0 mM/140 min, $P=0.03$). Muscle glycogen utilisation was reduced 27% during VI compared to SS (74 ±42 vs. 102 ±24 mmol/kg w.w.), while total Glu_{ox} as higher (99.2 ±13.1 vs. 83.9 ±12.7 g/140 min). The number of type I muscle fibres PAS staining darkly at the end of 140 min was 2% for SS and 40% following VI. Conversely, the number of type II fibres showing a reduced PAS staining was ten fold higher after VI compared to SS (10 vs. 1%). Despite differences in pre-load exercise, subsequent TT performance was not significantly different (29.14 ±2.24 vs. 30.50 ±2.23 min for SS and VI respectively). These results show that whole body metabolic and cardiovascular responses to 140 min of either SS or VI exercise of the same *average* intensity are similar, despite differences in skeletal muscle carbohydrate metabolism and recruitment.

In conclusion, the results from the studies reported in this thesis indicate that there are substantial differences in the physiological responses of well trained cyclists to individual TT and mass start races. Utilising a new ergometer, it has been shown

that it is possible to replicate the physiological demands of TT racing in the laboratory setting. Furthermore, the results obtained in the laboratory have excellent validity when compared to actual competitive races. The physiological demands of mass start cycle racing are also reproducible in the laboratory. In possibly the first investigation to examine the metabolic and performance responses to variable intensity or constant load exercise of the same average intensity, it was possible to show that whole body responses (i.e. $\dot{V}O_2$, HR, RPE, energy expenditure) to the two different riding strategies were remarkably similar. Furthermore, analysis of muscle glycogen depletion patterns revealed that differences in skeletal muscle carbohydrate metabolism and recruitment were relatively small under the conditions evaluated in this study, and did not affect performance of a subsequent bout of high-intensity exercise.

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The work in this thesis has been (or will be) published in the following journals:

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ABBREVIATIONS USED THROUGHOUT THIS THESIS

BM	Body mass
dpm	Disintegrations per minute
CHO	Carbohydrate
CHO_{ox}	Total carbohydrate oxidation (Equation 5)
CV	Coefficient of variation
d	Day
EXP	Experimental trial (Chapter Nine)
FFA	Free fatty acids
FAT_{ox}	Total fat oxidation (Equation 5)
Glu_{ox}	Plasma glucose oxidation
HC	5.5 km Hill climb (Chapter Four)
HR	Heart rate
h	hours
HR_{peak}	Peak heart race (recorded during either a maximal test or field performance)
Kg	Kilograms

min	minutes
mM	millimoles
PAS	Periodic Acid Schiffs stain (Section 3.3.7)
P/W	Power to body mass ratio (PPO/BM)
PERF	Performance trial (Chapter Nine)
PLA	Placebo
PPO	Peak power output
RR1	110.0 km road race (Chapter Four)
RR2	105.0 km road race (Chapter Four)
RPE	Rating of perceived exertion
RPS	Revolutions per second of the Kingcycle flywheel (Equation 1)
s	seconds
$SACO_2$	Specific (radio) activity of expired CO_2 in dpm/mmol (Equation 6)
Sa_{glu}	Specific (radio) activity of plasma glucose in dpm/mmol (Equation 6)
SD	Standard deviation
SS	Steady state or constant load
TT	Time trial
$\dot{V}CO_2$	Volume of expired carbon dioxide

VI	Variable intensity
$\dot{V}O_2$	Oxygen uptake
$\dot{V}O_{2max}$	Maximal oxygen uptake (reported by other authors)
$\dot{V}O_{2peak}$	Peak oxygen uptake (recorded during a maximal test)
W	Watt
W_{final}	The last completed workload in a maximal test on the Lode ergometer (Equation 2)
w.w.	Wet weight

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SECTION B

METABOLIC FACTORS ASSOCIATED WITH CONSTANT AND VARIABLE LOAD CYCLING PERFORMANCE

CHAPTER ONE

INTRODUCTION AND OVERVIEW

1.1 Introduction

Cycling is a sport encompassing many different disciplines: competition can last from as little as a few seconds, as in the case of 200 m track sprinting, to as long as several weeks for some of the major stage races (e.g. the Tour de France). The terrain can vary from cross-country (mountain biking), to light off-road tracks (cyclocross), open highways (road racing and time trials) to the use of specially constructed wooden or concrete (indoor and outdoor) banked tracks. Each of these categories of cycling may then be divided further into separate races within each discipline.

The experiments conducted for this thesis focus on the mass start "road races" and the individual races against the clock, commonly referred to as "time trials". A typical event would be extremely difficult to describe as a road race could have as few as 20, or as many as 200 competitors all starting a race at the same time. Events may be a short "criterium" around a tight street circuit, with a typical duration of 1 hour (h) plus a number of laps, or be on a longer loop circuit or a point to point course over distances of 50 to 250 km. Further to this, stage races may cover a number of days, some with more than one race per day, or as in the case of some major tours, racing may occur daily for several weeks. The Tour de France race in 1998 for example, covered 3,877 km in 22 stages over 23 days, including climbs to altitudes of 2,645 m. The entire event was covered at a record average speed of 39.983 km/h.

In the case of time trial events, each athlete starts at a predetermined staggered time interval, and has to race against the clock to cover the race distance in as short a time as possible, with no assistance from any other rider. Whilst there are

no set distances for time trial events, 40 km is the international standard distance for most amateur events, although races are frequently held over distances ranging from 10 miles (16.1 km), or shorter in the case of prologue time trials, to events of 24 h. There are also a few races that use time trial regulations for multi-day events (e.g. the Race Across America).

It has been suggested that the one hour record is possibly the most physiologically demanding of all cycling events (Keen 1998). The cyclist must cover as much distance as possible on a track with no assistance from other competitors. The only outcomes are success or failure. The current hour record is 56.375 km set by Chris Boardman of the UK. It has been estimated that Boardman was required to maintain 442 W for the 60 min, which equates to 6.4 W/kg body mass (BM), or 238 W/m^2 body surface area. Laboratory assessment of Boardman indicated that such a workrate would require a $\dot{V}O_2$ of 5.6 L/min or 81 ml/kg/min for the duration of the effort (Keen 1998).

Currently there exists a wealth of information regarding the physiological and metabolic responses to fixed intensity endurance cycling. However, until recently there have been few investigations examining the responses of trained cyclists to actual competition in the field (Hopkins and Hawley 1989; Jeukendrup and van Diemen 1998). Although those data provided some indication of the nature of road race cycling, the number of subjects and the data were of limited scope.

1.2 Aims and overview of this thesis

The overall aim of the current thesis is to investigate the physiological and metabolic responses of well-trained cyclists to constant load and variable intensity cycling during both actual competition and simulated laboratory situations.

The first half of the thesis investigates the physiological responses of cyclists to both individual time trial races and mass start road races. The second part of the thesis examines some of the metabolic factors that might contribute to the performance outcome of constant load and variable intensity cycling.

The first study, described in Chapter Four, describes the physiological responses of a group of well trained cyclist to a four day cycle stage race, which comprised an individual "prologue" time trial, two mass start road races, and an individual hill climb. This is the first study in the literature to detail the different physiological responses of trained cyclists to mass start races and individual time trials during actual competition.

The investigation described in Chapter Five determines the reliability of a novel laboratory based method for assessing the performance of highly trained cyclists. Using an air-braked ergometer, which accommodates the rider's own bicycle, athletes are able to self select, and change, the intensity at which they ride for a fixed distance, in the same way they would for an individual time trial in actual competition. In Chapter Six the physiological and performance responses of cyclists to time trial competition in the field are compared to laboratory based performance time trials of the same distance. The results of these two studies show the specific ergometer used to be the most valid and reliable method currently available for the performance testing of well-trained cyclists. This was

essential as the final studies of this thesis were laboratory based simulations of mass start and time trial competition.

Indeed, the study described in Chapter Seven may be the first of its kind to attempt to replicate the physiological demands of mass-start road races in the laboratory. Specifically, this investigation determined whether a prolonged bout of constant load cycling of the same *average* intensity as a variable load work bout would affect the performance of a subsequent time-trial.

Having previously established the reliability and validity of the testing ergometer, the second part of this thesis investigated some of the metabolic factors which might affect cycling performance. The first study in Section Two (Chapter Eight) examines whether the common practice of carbohydrate ingestion improves the short term (~30 min) time trial performances of well trained cyclists.

The final investigation (Chapter Nine) of this thesis was undertaken specifically to examine the metabolic and hormonal responses of well trained individuals to prolonged cycling while performing constant load and variable intensity exercise which was of the same *average* intensity. By measuring whole body (heart rate and oxygen consumption), metabolic (rates of substrate utilisation) and muscle (glycogen loss in type I and type II fibres) responses, it was hoped to provide a more complete picture of the requirements of constant load and variable intensity cycling events.

In summary, this thesis evaluates some of the physiological and metabolic factors associated with the performance of well-trained cyclist competing in mass-start and individual cycle time trial races.

CHAPTER TWO

REVIEW OF LITERATURE

2 REVIEW OF LITERATURE

The aim and scope of this thesis is to investigate various physiological and metabolic responses to mass start and individual cycling competition. However, a review of the literature reveals that currently there exists no studies which have characterised either the type of competition an elite cyclist undertakes, or the responses of such athletes to actual competition.

Accordingly, in order to gain a background into the sport of cycling, and the athletes that partake of the sport, this review will focus on the history of cycling, and the physiological characteristics of elite cyclists.

2.1 HISTORY OF CYCLING

2.1.1 History of the Bicycle

The exact origins of the bicycle are difficult to determine. There are some who propose that illustrations of "contraptions" resembling descendants of today's bicycle date back to 1,300 BC, whilst the first incarnations of the modern bicycle are suggested to have taken shape in 1690 (Le Mond and Gordis 1987). However, it wasn't until 1816 that Baron Carl Drais zu Sauerbronn constructed the first manoeuvrable, two wheeled man powered machine (Van der Plas, 1983). This machine, called the "Draisine" was a wooden structure where the rider straddled the frame across a lightly padded seat, and propelled the bike forwards by pushing one leg off the ground after the other. Although this bike could be steered, it was extremely heavy, and required significant effort to propel.

Technical improvements to this first bicycle followed slowly, introduced by small time experimenters, ingenious craftsmen, and enterprising industrialists. First, a steel version of the Draisine was made. This was followed by a modification which attached pedals to the front wheels, and then in around 1839, a system which allowed for driving the bike with treadles attached to the rear wheel was added.

In 1861 the Michaux velocipede, or "bone shaker" was released. It had a short frame consisting of a "backbone" that ran from the steering head to the real wheel axle, and carried a long "leaf-spring" which supported the saddle, the rider again propelling the machine by pedals attached directly to the front axle. Over the next ten years, production of this cycle increased, however, more importantly, so did the technical innovations and refinements it included. In 1868 the first ever track race, over a distance of 1,200 m, was won at the St. Cloud race track, Paris on a Michaux velocipede. One year later a team of Michaux riders took all the prizes in the first long distance road race, the Paris-Rouen, which covered a distance of 123 km and had a field of 198 participants. The event was eventually won in a time of 10 h and 40 min which is an average speed of eight miles per hour!

The advent of the Franco-Prussian war in 1871 marked the end of the French cycling boom, and also developments of the bicycle industry in Great Britain. However, the industrial revolution and advent of mass production allowed further technical and commercial developments which characterised the bicycle in the second half of the nineteenth century. As the size of the wheel limited the gear ratio of the bicycle (one pedal revolution equalled one turn of the wheel) many of the better cyclists found that their speed was limited by their leg cadence with the average wheel size of the Michaux velocipede. In order to overcome this problem,

a directly driven wheel with the greatest size possible was required. The high wheel or "ordinary" bicycle was born.

In 1871 British craftsman, James Starley used the first tension-spoked wheel in which the rim and hub were connected by looped wire spokes. The front, drive wheel was much larger than the rear wheel and, in one particular model this drive wheel was over 2 m tall. Amazingly this design prevailed for the next 15 years. However, it was not the shape of the bicycle alone that evolved during those years, there were also advances in materials, construction methods, and mechanical design. Further, tubular steel frames, hollow metal rims, and solid rubber tyres were but a few of the improvements that allowed manufacturers to build lighter, stronger, cheaper, faster and more comfortable bicycles each year.

The major drawback of the "ordinary" was that the equilibrium of the rider could be easily upset, and many a cyclist ended up taking a fall. A different design was therefore needed to increase the safety of the bicycle. The next stages of development included the "Star safety bicycle" in 1882 which had a small front wheel and large rear drive wheel, thereby placing the riders weight, and centre of gravity, further back. Alternative methods of achieving a safer design were to include the use of indirect gearing to reduce the size of the front wheel, without a loss in the speed of travel.

Although various methods of indirect gearing were used, the chain was being rapidly developed, and ultimately proved to be the only practical device. Further advances in safety were made in various models up until 1885 when a vastly improved version of the "safety" was released: the Rover safety bicycle. This new bike was a true "high performance" machine with a simple, smooth chain drive, a

sturdy diamond frame, and two standard-sized wheels. By 1887 the design of this bike could hardly be distinguished from it's much more modern counterparts, and by 1889, it had virtually replaced the "ordinary".

The final stage in the evolution of the bicycle was completed in 1887 with the introduction of the first pneumatic tyre. By the early 1890's the tyre had been refined and all top cycle racers were using air-filled tyres. Whilst this may be seen as the final stage in bicycle development, technological advances are continually being made in the manufacture of bikes, particularly in the use of new light weight materials and aerodynamically efficient designs which are aimed at improving either the maximum speed of the submaximal economy of the rider.

2.1.2 History of Cycle Racing

Cycle racing on the track and road had a strictly commercial origin. Bicycle manufacturers employed professional riders to promote the superiority of their machines (Woodforde 1970). During the 1870's and 1880's some of the most popular cycle events organised were "six-day races". The aim of these events was to see how many miles an individual rider could cover in six days. The first of these was held in 1875 in Newcastle. The first *indoor* "six" took place in the United States in 1886 with the winner covering a total distance of 1,401 miles (~2,250 km) in 12 h of riding per day. The first truly six day race in which competitors could ride for 24 h/d was held in New York in 1891, with the winner covering an eventual 1,466 miles (~2,360 km) in 142 hrs (10.3 miles per hour or 16.6 km/h). After 1886 the new safety bicycle allowed greater distances to be covered, with the record reaching over 2,000 miles (3,220 km) (13.9 miles per hour or 22.4 km/h) in 1897,

and 2,754 miles (~4,430 km) (19.1 miles per hour or 30.8 km/h) with two man team racing in 1899. The popularity of the six day races was enhanced by the spectacle of half dead riders, crawling around the track, punch-drunk with fatigue, some hallucinating or mumbling wildly, the crowd ever expectant of seeing a crash or collapse in the middle of the track.

As well as the format of the six day race, speed was also considered important. The first rider to break 20 miles per hour (~32.2 km/h) was H. L. Cortis in 1882. This feat was undertaken on a penny farthing. By 1899 the first individual had broken 1 min for the mile by following just cm behind a speeding train to obtain the benefits of drafting (although this nearly ended in disaster when the train braked suddenly at the end of its track!).

Whilst the Americans were focused on the six day track events, in Europe the popularity of road racing continued to grow, and so did the commercialism of the sport. With riders being required to use certain cycles, racing became an outlet for the cycling industry. Cycling also became a publicity wagon for enterprising newspapers. Interestingly, Henri Desgrange, the holder of the first one hour record (set in 1893 covering 35.325 km), was later to go on to become editor of a new daily paper, and by 1903 had started the Tour de France as a publicity event for his newspaper. The first Tour de France had sixty riders and covered approximately 1,550 miles (~2,500 km) in six stages over 18 days. The Tour de France soon became such a spectacular and interesting event to the public that the concept of multi-stage road races spread to other European countries.

From this point on cycling has developed in several directions. The current professional riders still compete in the major tours, and "classic" road races of

yesteryear. Track racing is a pursuit which has virtually ceased to exist in the United States, but still has a large following during the European Winter. On the other hand, the challenge of the individual events which have largely developed from the World one hour record, have mostly been taken up by amateur riders looking to quantify performance improvements.

2.2 Physiology of the elite cyclist

Despite the fact that most of the major developments in design of the bicycle had been made by the early 1890's, there have still been considerable improvements in the record performances of cyclists. These are largely due to an improvement in the physiology and training practices of cyclists, although dramatic reductions in drag as a result of better aerodynamics of the bicycle components, and riders clothing, are also important. Whilst considerable emphasis had been placed on determining the physiological characteristics of endurance runners, until recently there was a lack of such data for top American road cyclists (Hagberg et al. 1979b). As such, Hagberg et al. (1979b) studied nine national class American cyclists who were assessed for physical and physiological characteristics. Mean (±SEM) age, height, weight and % body fat was 25.1 ±1.6 yr, 180.3 ±2.3 cm, 72.0 ±2.2 kg and 7.6 ±0.2 % respectively. The group trained an average of 18.8 ±0.9 h/wk, with 74 ±6% of their training time based on distance and 20 ±6% undertaken as interval work. The mean maximal oxygen uptake ($\dot{V}O_{2max}$) of the group using a one min incremental protocol previously described by Hagberg et al. (1979a) was 70.3 ±2.0 ml/kg/min, with no differentiation in $\dot{V}O_{2max}$ being able to be made between the cyclists later selected for world championships and non-team

members for that event (n=4 vs n=5). In conclusion Hagberg et al. (1979b) stated that the $\dot{V}O_{2max}$ of these athletes was similar to that previously reported in top European cyclists (Israel and Weber 1972) and therefore other factors may contribute to producing high level cycling performances.

Vrijens et al. (1982) also considered that the physiological profile of competitive road cyclists may shed light on factors contributing to success in international competition. These researchers aimed to determine age related norms for physiological characteristics. A total of 406 cyclists from the age of 13 were subdivided into age groups and profiles for each group determined. Not surprisingly, there were no differences between height and body weight of the different aged amateur (over 19 yr) riders, amateurs selected for the world championships and professionals (Table 2.1), however, % body fat reduced with riding ability (Table 2.1).

It is interesting to note that the data of Vrijens et al. (1982) shows little difference in any of the physiological characteristics despite the range in abilities of the cyclists. Further, despite the slightly higher values, it was not noted whether the differences in $\dot{V}O_{2max}$ and "Watt max" were significantly different between the groups. Additionally, the $\dot{V}O_{2max}$ data of Vrijens et al. (1982) was, on average, between 2.7 and 4.6 ml/kg/min lower than that previously reported for top American cyclists (Hagberg et al. 1979b).

Table 2.1 Physiological characteristics of amateur, and well trained Belgian cyclists. (Adapted from Vrijens et al. 1982).

Category	Age (yr:mnth)	Height (cm)	Weight (kg)	% Body fat	$\dot{V}O_{2max}$ (L/min)	$\dot{V}O_{2max}$ (ml/kg/min)	Watt max (W)
Amateur (n=56)	19:6	177.3 ±5.1	71.6 ±6.4	13.4 ±2.7	4.54 ±0.45	63.8 ±6.7	390 ±42.0
Amateur (n=41)	20:5	178.6 ±4.7	72.1 ±5.3	12.9 ±2.7	4.69 ±0.47	64.6 ±7.1	412 ±41.3
World Team (n=17)	21:1	178.9 ±4.6	71.4 ±4.6	11.6 ±2.1	4.81 ±0.29	67.6 ±5.9	448 ±30.0
Professional (n=40)	26:5	175.2 ±6.7	71.3 ±6.1	11.6 ±2.6	4.71 ±0.40	65.7 ±6.1	419 ±47.8

These authors also reported that physical work capacity was significantly greater in high level amateurs and professionals, and suggested this enhanced endurance capacity, which in turn would lead to improved performance.

Faria et al. (1989) measured the physiological responses of elite junior riders and the compared these to mature cyclists in order to try and identify the physiological requirements for competitive success. Fifteen male junior (15 ±1.7 yr) cyclists (height 179.2 ±1.1 cm, weight 69.0 ±8.4 kg, body fat 7.4 ±2.0%) underwent incremental testing for the determination of $\dot{V}O_{2max}$ and estimation of ventilatory

break point (T_{vent}). The mean $\dot{V}O_{2max}$ of 5.2 ±0.8 L/min or 75.5 ±3.1 ml/kg/min was high in comparison to values previously reported for other elite endurance cyclist (Table 2.2).

Although there is no comparative performance data for the athletes in the various studies reported in Table 2.2, and other physiological measures such as peak power are difficult to relate due to differing test protocols, a general assumption can be made that no one team, or Nation, performed significantly better than any other. Hence, the $\dot{V}O_{2max}$ data illustrated in Table 2.2 raises an interesting question: why are such large differences observed in $\dot{V}O_{2max}$ data of the teams (range 67.6 ml/kg/min to 77.4 ml/kg/min) yet the performances of the athletes similar?

Analysis of the results of the study by Faria et al. (1989) show that T_{vent} occurred at ~83% of the mean $\dot{V}O_2$. These results suggest that this group of cyclists was able to compete at a high level not only as a result of their high $\dot{V}O_{2max}$, but because of the high T_{vent}, they had the capacity to sustain a high percentage of $\dot{V}O_{2max}$ for sustained periods (Faria et al. 1989). The ability to sustain a high steady state $\dot{V}O_2$ is an important characteristic of top cyclists and has been investigated further by Coyle et al. (1988).

Table 2.2 Comparison of maximal oxygen uptake among elite cyclists. Adapted from Faria et al. (1989).

Author	Team	Oxygen Uptake (ml/kg/min)
White et al. 1982	G.B. Olympic team	77.4
Israel & Weber 1972	East Germany national team	75.5
Faria et al. 1989	US Junior national team	75.5
Malhotra et al. 1984	Indian team	75.1
Burke 1982	US national team	74.0
Hermansen 1973	Norwegian team	73.0
Joussellin et al. 1984	French team	71.1
Wilber et al. 1997	US National road team	70.3
Hahn et al. 1986	Australian national team	70.0
Lopatequi et al. 1986	US Class I, II	69.6
Strømme et al. 1977	US Elite	69.1
Strømme et al. 1977	Swedish national team	69.1
Burke 1982	Swedish team	69.0
Faria et al. 1968	Danish team	68.0
Bonjer, 1979	Elite Dutch national team	67.6

In that investigation, Coyle et al. (1988) divided fourteen well-trained male cyclists into two equal groups according to the relationship between their blood lactate threshold (LT) and $\dot{V}O_2$. Group H (n=7, age 24.7 ±1.4 yr, weight 71.1 ±1.4 kg, $\dot{V}O_{2max}$ 4.87 ±0.04 L/min or 68.6 ±1.2 ml/kg/min) achieved LT at 81.5 ±1.8% of $\dot{V}O_{2max}$, whilst group L (n=7, age 25.1 ±0.8 yr, weight 72.1 ±1.5 kg, $\dot{V}O_{2max}$ 4.75 ±0.03 L/min or 66.0 ±1.2 ml/kg/min) achieved LT at 65.8 ±1.7% of $\dot{V}O_{2max}$. Each subject was required to undertake a performance task which required them to exercise until fatigue at an intensity of ~88% of $\dot{V}O_{2max}$. A strong relationship was found between time to fatigue and % $\dot{V}O_{2max}$ at LT ($r = 0.90$, $P<0.001$) with group H exercising for 60.8 ±3.1 min and group L for only 29.1 ±5.0 min. These results reveal that despite similar $\dot{V}O_{2max}$ values, significant performance differences can occur as a result of submaximal responses to exercise.

Examination of the submaximal responses to exercise in these two groups determined that group L displayed a significantly ($P < 0.001$) higher respiratory exchange ratio (RER) than group H (0.94 ±0.01 vs 0.85 ±0.01), and hence greater calculated CHO oxidation (605 ±20 vs 358 ±24 mmol). In addition muscle glycogen utilisation was also significantly ($P < 0.001$) higher in group L (65.4 ±5.6 vs 27.9 ±3.0 mmol/kg).

Histological examination of muscle biopsies of several of the subjects in the investigation by Coyle et al. (1988) revealed that cyclists in group H had a higher percentage of Type I muscle fibres (66.7 ±5.2 vs 46.9 ±3.8%, $P < 0.01$), and a 15% lower mean fibre area (6,930 ±412 vs 8,132 ±426 μm^2, $P < 0.08$). These authors suggest that this accounts for the 24% greater capillary density of the subjects in group H (405 ±20 vs 327 ±36 capillaries/mm^2, $P < 0.08$). These data

demonstrate that the endurance performances of cyclists can be significantly different even though maximal values (i.e. $\dot{V}O_{2max}$ or PPO) are similar.

Coyle et al. (1991) repeated these investigations using a group of "elite" national class athletes (n = 9) and a group of "good-state class" athletes (n = 6). Whilst there where no significant differences in the physiological characteristics of these athletes, nor any differences in the riders $\dot{V}O_{2max}$, the elite cyclists reached lactate threshold at a higher percentage of $\dot{V}O_{2max}$ (79.2 ±1.1 vs 75.3 ±1.5 % $\dot{V}O_{2max}$, $P < 0.05$) and where able to produce a higher average workrate (346 ±7 vs 311 ±12 W, $P < 0.05$) during a 1 h laboratory performance test. Again the elite athletes in this trial possessed a greater percentage of Type I fibres (66.5 ±3.7 vs 52.9 ±5.7% Type I, $P < 0.05$) and a higher muscle capillary density (464 ±25 vs 377 ±22 capillaries/mm^2, $P < 0.05$) than the "good-state class" cyclists. Of additional note however, was that in this investigation, the average power output during the simulated 1 h performance ride was highly correlated to $\dot{V}O_2$ at lactate threshold ($r = 0.93$, $P < 0.001$).

More recently, several articles have reviewed the relationships between various physiological factors and endurance cycling performance (Burke 1994; Burke et al. 1990; Coyle 1995; Faria 1992). These authors all support the notion that a high $\dot{V}O_{2max}$, a high proportion of Type I fibres, and a high capillary density will all assist in improving submaximal economy, and hence improve endurance performance. The major limitation of many of these investigations is that whilst it can be assumed that elite cyclists require a high $\dot{V}O_{2max}$, and a high submaximal efficiency, there have been no investigations to date that have attempted to use these measures to predict endurance cycling performance during actual

competition. Further, whilst a wealth of information currently exists regarding the physiological characteristics of cyclists and other elite athletes, and their metabolic responses to laboratory based steady-state exercise, there are no data available regarding the responses of these athletes to actual competition. Accordingly, the studies undertaken for this thesis describe a series of related, yet independent investigations to determine the physiological responses of well-trained cyclists during actual competition, and also examine the metabolic responses of these athletes to simulated competition in the laboratory setting.

CHAPTER THREE

METHODOLOGY

3.1 APPARATUS

3.1.1 The Lode cycle ergometer

The Lode cycle ergometer (Lode, Groningen, The Netherlands) is an electrically braked ergometer. Power output on this ergometer is independent of pedal frequency between 60-120 revolutions/min. For all testing, the ergometer was modified with both standard racing handlebars, clip-on triathlon style handlebars, and clip-in pedals. The cyclist was also able to adjust the vertical height and perpendicular distance from the bottom bracket of both the saddle and handlebars to match their specific size requirements. On the first visit to the laboratory these measurements were recorded, and replicated for each cyclist during all subsequent testing sessions.

During the experimental trials described in the following chapters, power output on the Lode ergometer was adjusted either manually by the investigator, or pre-programmed to the specific requirements of that experiment. During programming of the Lode ergometer for the non steady-state work bouts (described in detail in the studies in Chapters Seven and Nine), the investigator was required to input delta power and delta time for each work and time interval for the total duration of that test. This was a simple process whereby the change in power required for each work step was entered into the Lode terminal, followed by duration of each individual workstep (60 seconds). The changes in workload were, in all cases immediate, and were different for each individual athlete.

3.1.2 The Kingcycle ergometer

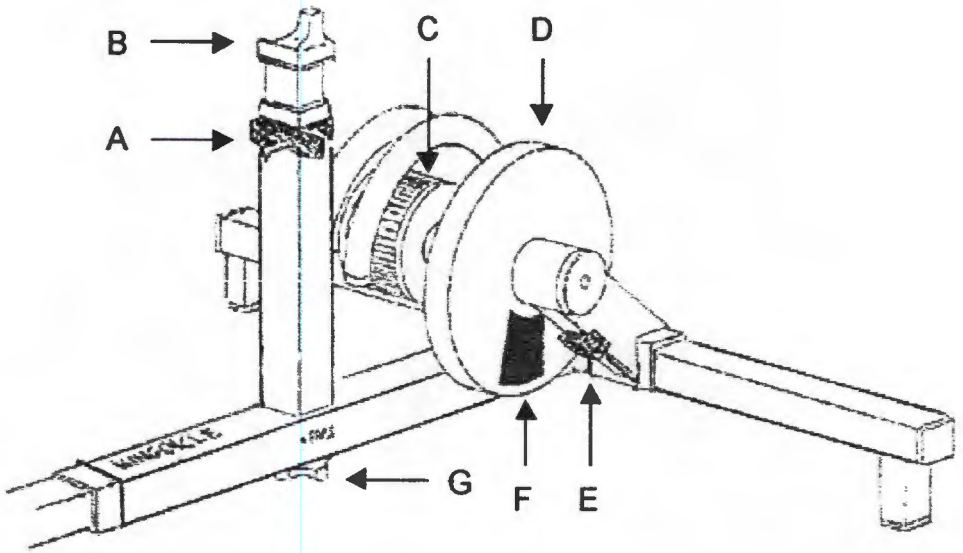
The Kingcycle ergometry system (Kingcycle Ltd., High Wycombe, Buckinghamshire, UK) is an air-braked ergometer that allows cyclists to ride their own racing bicycles in the laboratory setting (Figure 3.1). After removal of the front wheel, the subject's bicycle was attached to the ergometry system by the front fork and supported by an adjustable pillar under the bottom bracket (Figure 3.2). The bottom bracket support (B in Figure 3.2) was used to adjust the rolling resistance of the rear tyre on an air-braked flywheel. From the output of a photo-optic sensor monitoring the velocity of the flywheel, (diameter 190 mm and mass 4.6326 kg [E & F in Figure 3.2]) in revolutions/s (RPS), an IBM compatible computer calculated the power output (W) that would be generated by a cyclist riding at that speed on level terrain, using the following equation:

$$\mathbf{W = 0.000136 \cdot RPS^3 + 1.09 \cdot RPS} \qquad \textbf{(Equation 1)}$$

Where W is the power output in Watts (W), and RPS is the velocity of the flywheel in revolutions/s.

Figure 3.1 Cyclist riding their own bike mounted on the Kingcycle Ergometry System.

Figure 3.2 Diagrammatic representation of the Kingcycle ergometer showing: (A) optical crank sensor; (B) bottom bracket support; (C) air-brake/fan; (D) flywheel; (E) optical flywheel sensor; (F) flywheel marker, and (G) bottom bracket support pillar adjuster.



Prior to each test, subjects performed a series of 'run down' calibrations, in which they accelerated to a work rate of ~400 W and then immediately stopped pedalling, while remaining seated in their normal riding position. During this calibration the bottom bracket support was adjusted (G in Figure 3.2) until the necessary rolling resistance was achieved, as indicated by the computer display showing that the slowing of the flywheel matched a pre-determined reference power decay curve. The total resistance provided by the ergometer is equivalent to that experienced by a cyclist of approximately 65 kg riding on a flat road (Personal Communication, Kingcycle Ltd).

During all experimental trials, the Kingcycle ergometer was interfaced with an IBM compatible computer which continually monitored and stored, power output (W) and pedal cadence (revolutions/min). During maximal tests the computer provided information to the rider as to the power output required for that trial or time period. The power output was presented to the rider in the form of the blue "kite" indicating whether they should ride either slower or faster (Figure 3.3, right panel). The cyclist is required to maintain a power output such that the "kite" remains on the correct line for the duration of the test. However, should a cyclist's power output either increase or decrease to the point that the "kite" enters the red hatched zone (a 5% deviation from the desired power) an "out of range" warning is flashed on the screen. If the cyclist is not able to return to the necessary power output within ~10 s, or should they continually enter the out of range zone, the test was terminated.

The information regarding cadence, power output, heart rate (HR), time, elapsed distance and speed (shown on the left panel of Figure 3.3) was made available to the cyclist for the first five minutes of the maximal test. After this time, the information was covered from the cyclists view.

During all time trials (TT), the rider was presented with information regarding the percentage distance still to ride (Figure 3.4). This was illustrated in the form of a countdown from 100% to 1% (shown on the top left panel of the computer screen), as well as the blue "kite" moving from start to finish. Additional information regarding the cadence, power output, HR, test time, and speed was obstructed from the athletes view in order to blind the performance trials.

Figure 3.3 Computer screen showing the data displayed to the cyclist during a Kingcycle maximal test (left side of screen), and the blue "kite". Note that the display data were covered from the cyclist's view after five minutes of a test.

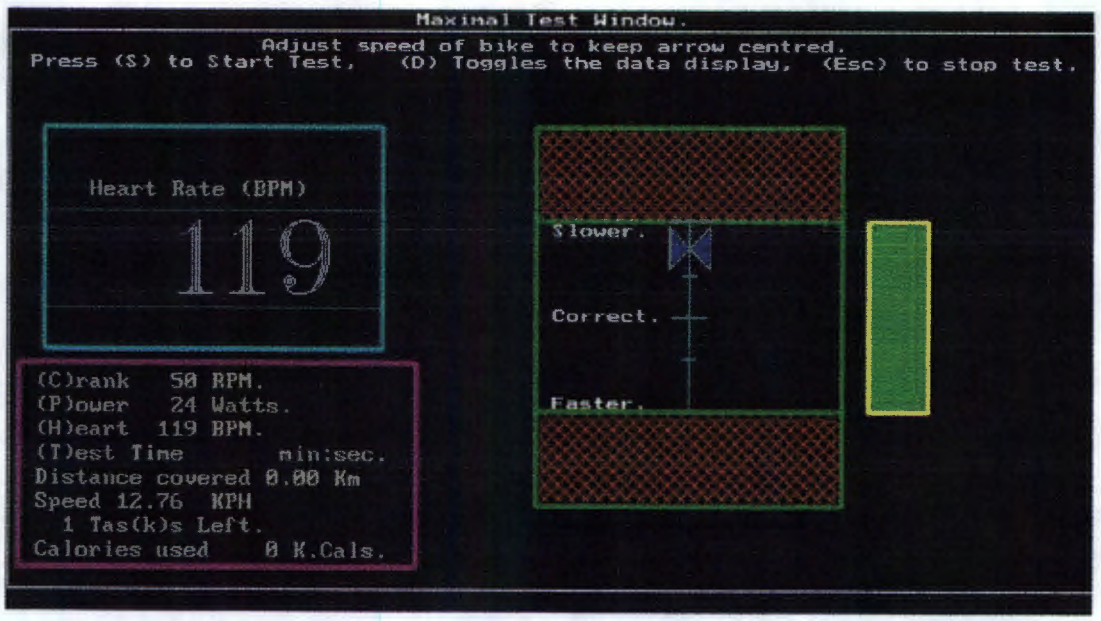
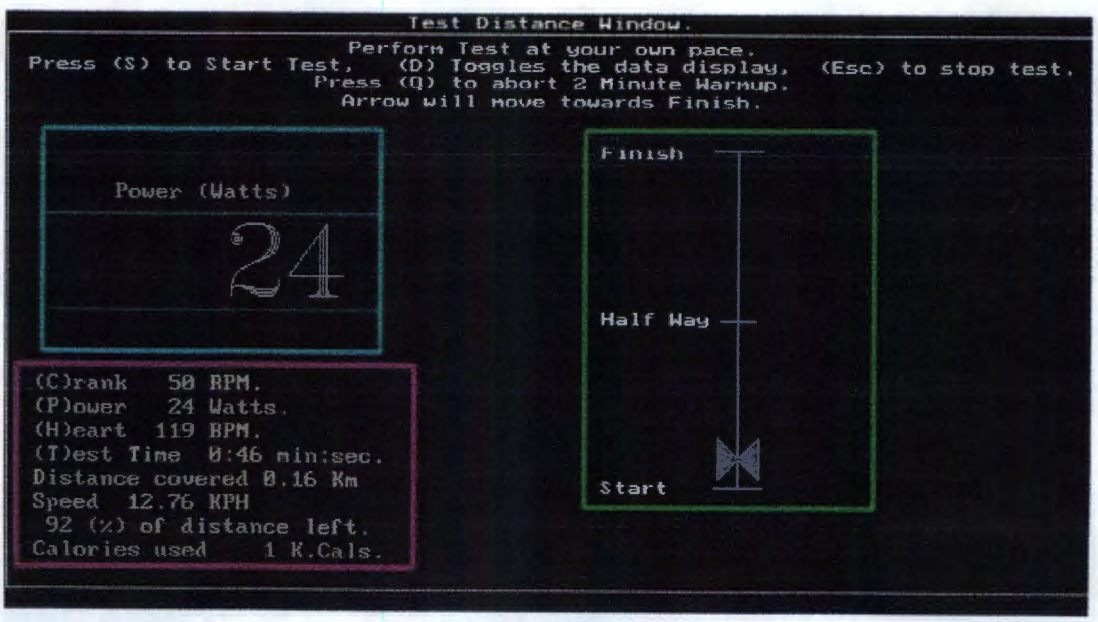


Figure 3.4 Computer screen showing information presented to the cyclist during a time trial. Note that the display data (purple box) were covered from the cyclist's view during all time trials, and the highlighted option (light blue box) was given as a percentage of the total distance remaining.



3.1.3 Heart rate (HR) monitoring

During all experimental trials, HR was monitored using a Polar Sports Tester HR monitor (Polar Electro, OY, Kempele, Finland). This monitor uses a short wave telemetry system and consists of a transmitter (an electrode belt worn around the chest) and a wrist mounted receiver. The receiver records and stores the momentary HR at predetermined intervals of 5, 15 or 60 s, which can subsequently be "down-loaded" into an IBM compatible computer for subsequent analysis.

Although the Polar HR monitor has previously been shown to be both valid and reliable (Bar-Or et al. 1996; Leger and Thivierge 1988; MacFarlane et al. 1989), and has its own in built error filter (Personal Communication, Polar Electro), if HR values collected during an experiment were outside the expected physiological ranges during moderate to intense exercise (<50 beats/min, or >220 beats/min) those particular data points were excluded from all subsequent analyses. This problem only arose when group responses to exercise were measured, and subjects were riding close together in a bunch (Chapter Four). In this situation, any subject whose HR trace was effected for greater than 5% of the exercise period was excluded from subsequent data analysis under that particular condition.

3.2 SUBJECTS AND PRELIMINARY TESTING

3.2.1 Subject characteristics

During all the experimental trials described in this thesis, a total of 34 cyclists acted as subjects. Of these, eight individuals participated in two or more trials. All of these subjects were highly trained cyclists or triathletes, who were, at the time of an investigation, participating in regular endurance training (>300 km/wk) and competition and had been for a minimum of the three previous consecutive years. Subjects ranged in ability from good club level cyclists to Provincial and National representatives. Apart from three females who participated in the study described in Chapter Eight, all subjects were male. The specific characteristics of the subjects who participated in an experiment are shown in each chapter.

Prior to participating in any investigation, all subjects were fully informed of the nature and risks of a study. Each cyclist gave their written informed consent in accordance with the guidelines outlined by the American College of Sports Medicine (1995). All subjects were also fully habituated to the methods and procedures involved prior to participation in an experimental trial. This was undertaken by ensuring that subjects had either participated in a investigation of a similar nature in our laboratory, or by having subjects report to the laboratory for several practice or familiarisation sessions prior to an experiment.

3.2.2 Determination of peak oxygen uptake and peak power output

All subjects were required to undergo a progressive incremental test for the determination of peak oxygen uptake ($\dot{V}O_{2peak}$) and peak sustained power output

(PPO). On reporting to the laboratory, subjects were required to void and/or urinate, before being weighed in their cycle shorts. Body mass (BM) was recorded to the nearest 0.1 kg on a Seca 701 balance beam scale (Seca Ltd, Birmingham, UK).

Each test was conducted after the subjects had refrained from all heavy physical activity for a minimum of 24 h. Subjects first completed a warm-up lasting 15-20 min at a self-selected intensity. It has been my experience that the higher the ability level of a cyclist, the longer they prefer to warm-up prior to a maximal effort. Subjects remained seated for the duration of the maximal ride and received strong verbal encouragement from the principle investigator.

Where the Lode ergometer was used for a maximal test, subjects undertook a protocol previously validated and described in detail by Hawley and Noakes (1992). Briefly, a test commenced at a workload of 3.33 W/kg BM with a 50 W increase after 150 s and 25 W increments after each subsequent 150 s workload, until subjects reached volitional fatigue. This coincided with either a drop in cadence of >10 revolutions/min, or a respiratory exchange ratio (RER) of >1.10, or both. Peak sustained power output was determined from the equation of Kuipers et al. (1985):

$$\mathbf{PPO = W_{final} + ((t/150) \cdot 25\ W)} \qquad \textbf{(Equation 2)}$$

where PPO is the peak power output, W_{final} is the last completed workload, and t is the duration for which the final, uncompleted workload was maintained.

Where the Kingcycle ergometer was used for maximal testing, subjects commenced the test at a power output of 200, 250 or 300 W depending on their BM and riding ability. Subjects were then required to increase their pace to follow a continuous ramp of 1 W/3 s, (20 W/min) until volitional fatigue. Fatigue corresponded with the inability of the cyclist to produce the desired power output despite strong verbal encouragement, and in all cases occurred between 8 and 12 min of a test. The computer determined PPO as the highest average power output produced during any 60 s period of a test. During maximal testing, expired air was collected for the determination of $\dot{V}O_{2peak}$ (described subsequently in section 3.3.1).

3.2.3 Experimental trials

Most subjects agreed to participate in several experimental trials. A typical experiment consisted of a performance TT (Chapters Five, Six and Eight) or a variable intensity (VI) endurance ride followed by a TT (Chapters Seven and Nine). All trials were conducted in a random order and separated by a minimum of four days and a maximum of 14 d. In order to ensure subjects commenced each experimental trial in a similar nutritional and physical state, rides were conducted at the same time of day, a minimum of 24 h post exercise and 3 h post prandial. All subjects were also required to maintain a full training diary and dietary record for the 72 h prior to their first trial and were then instructed to maintain the same diet and training regimen in the 72 h preceding the subsequent trial(s).

In the case of the study described in Chapter Nine, in which measurements of muscle glycogen concentration were made, subjects were provided with pre-

prepared food (Nutrifit, Cape Town, RSA) of the same total energy content and composition as their habitual diets. They were also requested to maintain the same training pattern for this 72 h pre-trial period. A registered dietician using a commercial computer program (Food Finder Diet Analysis, Medtech, Tygerberg, Cape Town, South Africa) determined the energy content and nutritional composition of a four day dietary record of subject's habitual diets. Subjects were given precise written and verbal instructions of how to record all food and fluid consumed during the four day period, which included one day of the weekend. Compliance to the diets subsequently provided was facilitated by instructing subjects to return all pre-prepared food that they had not consumed and to record any additional fluid and foods they ingested.

To further ensure the same training was undertaken in the period immediately prior to a trial, subjects were requested to maintain a diary for each three-day period prior to an experiment. It has been a personal observation that well-trained subjects will still ride moderately hard the day before a laboratory trial, even when instructed to the contrary. Therefore, subjects were instructed to refrain from all heavy exercise for the 24 h preceding a testing day and were given a HR monitor to wear during this period to record all activity. If these HR records showed a subject had trained, or had been involved in vigorous physical activity, he or she was not allowed to participate in that experiment.

All laboratory based performance TT efforts were undertaken on the Kingcycle ergometer. Subjects were instructed to cover an assigned distance, usually 20 km, in "the shortest possible time". During these rides, the only feedback subjects received was the elapsed distance. Subjects were allowed to consume either water or a carbohydrate (CHO) beverage *ad libitum* throughout the first TT, but the

drink chosen and the volume consumed by each subject remained the same for all subsequent tests in that particular experiment.

Throughout each trial, power output and cadence were monitored continuously, averaged each 60 s, and subsequently stored by the IBM computer which was interfaced with the Kingcycle ergometry system. Momentary HR was also monitored by a HR monitor (described previously in section 3.1.3), and recorded at 15 s intervals throughout the TT.

3.3 COLLECTION AND ANALYSIS OF EXPIRED AIR SAMPLES

3.3.1 Gaseous exchange ($\dot{V}E$, $\dot{V}O_2$ $\dot{V}CO_2$, RER)

During gas collection, subjects wore a noseclip and breathed through a mouthpiece or facemask attached to an Oxycon Alpha automated gas analyser (Erich Jaeger Ltd., Hoechberg, Germany). This system operates on a breath-by-breath analysis basis, has a maximum flow rate of 20 L/s and flow resistance of <0.1 kPa/L/s. The oxygen analyser is based on the differential-paramagnetic measurement principle, and carbon dioxide analysers on the infrared absorption method. Both analysers are accurate to 0.01% (Personal Communication, Erich Jaeger Ltd).

Before each test, the gas analyser was calibrated with a Hans Rudolph 5530, 3 L syringe and a 5% CO_2: 95% N_2 gas mixture. Analyser outputs were processed by an IBM compatible computer that calculated L/min rates of ventilation ($\dot{V}E$),

oxygen consumption ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), and respiratory exchange ratio (RER) using conventional equations (Jones and Campbell 1982).

3.3.2 Calculation of carbohydrate (CHO) and fat oxidation, and energy consumption

At the same time as expired gas was sampled, whole body rates of instantaneous CHO and fat oxidation (g/min) were calculated by indirect calorimetry using stoichiometric equations (Frayn 1983) assuming a non-protein RER.

$$\textbf{CHO oxidation} = 4.585 \cdot \dot{V}CO_2 - 3.226 \cdot \dot{V}O_2 \quad \textbf{(Equation 3)}$$

$$\textbf{Fat oxidation} = 1.695 \cdot \dot{V}O_2 - 1.701 \cdot \dot{V}CO_2 \quad \textbf{(Equation 4)}$$

Such equations are based on the assumption that $\dot{V}O_2$ and $\dot{V}CO_2$ accurately reflect tissue O_2 consumption and CO_2 production. In well trained subjects similar to those employed in the current investigation, indirect calorimetry has previously been shown to be a valid method for quantifying rates of substrate oxidation during strenuous exercise at intensities of 80-85 % of $\dot{V}O_{2max}$ (Romijn et al. 1992).

Furthermore, a pilot study showed that rates of ventilation ($\dot{V}_E$) during the experiment using variable intensity exercise (described in Chapter Nine) were relatively constant. This would suggest that respiratory compensation for increasing metabolic acidosis was negligible. Assuming a non-steady-state lactate distribution volume of 100 ml/kg BM (Stanley et al. 1988) the resultant loss of HCO3 to CO_2 would be expected to increase $\dot{V}CO_2$ values by less than 0.08 L/min. Indeed, even the most rapid (~1.5 mM) increases in plasma lactate

concentrations during the variable workloads would be expected to increase $\dot{V}CO_2$ by, at most, 2% (MacRae et al. 1995).

Subsequent estimation of the total energy (kcal) utilised during an exercise bout was determined from the area under the CHO and fat oxidation versus time curve for each subject, and calculated from the following equation:

$$\textbf{Total energy (kcal)} = \textbf{4.1}\bullet CHO_{ox}\,\textbf{(g) + 9.3}\bullet FAT_{ox}\,\textbf{(g)} \quad \textbf{(Equation 5)}$$

Where CHO_{ox} is the total carbohydrate oxidation as determined by the area under the carbohydrate oxidation curve (g) and FAT_{ox} is the total fat oxidation as determined by the area under the fat oxidation curve.

3.3.3 Determination of metabolite concentrations in the plasma

During those experiments in which blood was sampled, a Jelco 18 gauge cannula (Critikon, Halfway House, Transvaal, SA) was inserted into a forearm vein of the subject. At predetermined intervals, normally every ten min, ~15 ml of blood was drawn, of which equal quantities were placed into pre-chilled vacutainers for the subsequent determination of plasma glucose, lactate, insulin, free fatty acids (FFA), and glucagon concentrations. After each sample was drawn, the cannula was flushed with 2-3 ml of sterile saline.

Samples were then placed on ice until after the trial, at which time they were centrifuged at 2,500 revolutions/min at 5°C for 10 min. The plasma from the samples was stored at -20°C until subsequent analysis using the following techniques.

Plasma Glucose

Plasma glucose concentrations were determined in duplicate by the glucose oxidase method of Hyvarinen and Nikkilia (1962) using a glucose analyser (Glucose Analyser 2, Beckman Instruments Inc., Fullerton, California, USA). The intra-assay variation of this technique in our laboratory is less than four percent.

Plasma Lactate

Plasma lactate concentration was measured by spectrophotometric (Beckman Model 35, Beckman Instruments Inc., Fullerton, California, USA) enzymatic assays (Lactate PAP, boiMerieux, Lyon, France) as previously described in detail by Gutman and Wahlefeld (1984). The intra-assay variation of this technique in our laboratory is less than three percent.

Plasma Insulin and Glucagon

Plasma insulin and glucagon concentrations were determined using radioimmunoassay techniques (Coat-a Count Insulin; Double Antibody Glucagon, both Diagnostic Products, Los Angeles, USA). The intra-assay variation for the determination of plasma insulin concentration was less than three percent.

Plasma Free Fatty Acids

Serum free fatty acids (FFA) were measured using an enzymatic colorimeter assay (Shimizu et al. 1980).

3.3.4 Plasma glucose and lactate specific activity, and rates of plasma glucose oxidation

In order to calculate the rates of plasma glucose oxidation in the experiment described in Chapter Nine, subjects ingested a 300 ml pre-trial bolus solution containing tracer amounts (0.004% 0.19 μCi/g) of a uniformly labelled U-^{14}C glucose tracer (Amersham International, Buckinghamshire, UK). This same solution was then ingested during the trial at a rate of 150 ml every 15 min. During exercise expired CO_2 was collected at 10 min intervals in a 2 L anaesthetic gas bag using a Hans Rudolph 2700 one-way valve, and subsequently passed through a solution containing 1 ml of hyamine hydroxide (United Technologies, Packard, Illinois, USA), 1 ml of 96% ethanol (SAARCHEM, Krugersdorp, RSA), and 1-2 drops of phenolphthalein (SAARCHEM, Krugersdorp, RSA). The expired CO_2 was passed through this solution until exactly 1 mmol of CO_2 was trapped, as indicated by the changing of the phenolphthalein indicator from pink to clear (Scherrer et al. 1978).

Approximately 10 ml of liquid scintillation cocktail (Ready Gel, Beckman Instruments, Fullerton, California, USA) was added to this solution which was then stored in a dark area for 12 h. Samples were subsequently counted in an Insorb 460C Automatic Liquid Scintillation counter (United Technologies, Packard, Illinois, USA) for $^{14}CO_2$ radioactivity in disintergrations/min (dpm). All counts were corrected for differences in quench and background.

A 1 ml sample of plasma that had been collected for determination of plasma glucose was utilised for this assay. Initially 70 μl of 3.5 M $HClO_4$ was added to deproteinise each sample and drive off any ^{14}C-bicarbonate as $^{14}CO_2$. The

samples were then centrifuged at 5,000 revolutions/min for 10 min at 4 °C and the protein-free supernatant removed and kept refrigerated. The precipitate was then resuspended in 0.76 ml of 0.13 M $HClO_4$, re-centrifuged and the supernatant added to that previously saved. This step was repeated a further time. The pH of the combined supernatant of each sample was then neutralised with the addition of 136 µl of 3 M K_2CO_3 in 0.01 M Tris-HCl buffer, and centrifuged again at 5,000 revolutions/min for 20 min to remove the precipitate. The supernatant was then passed through an anion exchange column (Extra-Sep RC SAX, chromatography Research Supplies, Addison, Illinois, USA) which had been conditioned with 2 x 10 ml washes of ethanol and 2 x 10 ml washes of distilled H_2O. The void volume, which contained some glucose, was collected as the remaining glucose was eluted with distilled H_2O (3 x 1 ml). Lactate was then eluted with 2 x 1ml of 2 pH 1 M $CaCl_2$.

Collected samples were then evaporated to near dryness at 60°C for ~20 h, and after cooling, mixed with 15 ml of scintillation cocktail. ^{14}C radioactivity was measured in an Insorb 460C Automatic Liquid Scintillation counter (United Technologies, Packard, Illinois, USA). After scintillation counting it was found that the specific activity of the lactate samples were just above background levels and so the subsequent rates of plasma glucose oxidation did not need to be corrected for any lactate oxidation that might have occurred.

Losses in radioactivity during preparation of the sample were calculated from a control plasma sample that had been spiked with a known amount of U-^{14}C glucose and run concurrently with the test samples. Such recoveries averaged 90.1 ±0.7%. After the corrections for losses of radioactivity had been made, the

specific activity in dpm/mmol glucose could be calculated. Further, since the 1 ml aliquot of plasma used for radiation counting was from the same plasma sample as previously used for the determination of glucose concentration, total blood glucose oxidation was calculated from:

$$\mathbf{Glu_{ox} = (SACO_2 / Sa_{glu}) \cdot \dot{V}CO_2} \quad \textbf{(Equation 6)}$$

In this equation Glu_{ox} is the rate of plasma glucose oxidation in mmol/min; $SACO_2$ is the specific (radio) activity of the expired CO_2 in dpm/mmol; Sa_{glu} is the corresponding specific (radio) activity of the plasma glucose in dpm/mmol; and $\dot{V}CO_2$ is the volume of expired CO_2 mmol/min, calculated from the L/min $\dot{V}CO_2$ and the 22.4 ml/mmol gas volume. Since the complete conversion of one molecule of U-^{14}C glucose to six molecules of $^{14}CO_2$ decreases the dpm/mmol specific radioactivity by a factor of six, the $\dot{V}CO_2$ values did not need to be divided by six to allow for six CO_2 molecules arising from the oxidation of one glucose molecule.

3.3.5 Muscle biopsy technique

In the experiment described in Chapter Nine, each subject was required to undergo four muscle biopsies. While subjects rested in a supine position, a percutanous muscle biopsy sample was taken from the vastus lateralis using the technique of Bergström (1962) as modified by Evans et al. (1982). Pre and post exercise samples were taken from contra-lateral legs, with post exercise samples being obtained within 60 s of the completion of the exercise task.

The biopsy technique involved using a 50 ml disposable syringe which was connected to a 5 mm biopsy needle (Evans et al. 1982). The area above the belly of the vastus lateralis (approximately 15-20 cm above the knee) of both legs was shaved, cleaned with betadine antiseptic solution, and the skin and outer layer of the subcutaneous tissue were locally anaesthetised with 2 ml of Lignocaine (Labethica Pty. Ltd., Bethlehem, South Africa). Five minutes after the administration of the anaesthetic, or when the area was no longer sensitive to touch, a sterile surgical blade was used to make an incision of approximately one cm in length through the skin and underlying subcutaneous tissue and muscle fascia. The incision made for the post-exercise biopsy was then closed with skin closures and dressed.

The muscle biopsy was made by placing the tip of the biopsy needle, with its inner cylinder closed, approximately 2.5 cm deep into the muscle. The inner cylinder was then retracted a few centimetres while the outer cylinder remained in place. As the inner was retracted, the plunger of the 50 ml syringe was retracted causing a suction force which pulled the muscle tissue into the "window" of the hollow needle. While suction was maintained, the central cylinder was swiftly closed, cutting the muscle tissue that had protruded into the central bore of the biopsy needle. As the cut was completed, suction of the syringe was released and the needle withdrawn from the muscle. The section of biopsied muscle was removed from the biopsy needle using sterile forceps. A sample of approximately 50-100 mg was collected in all cases.

After collection, the muscle biopsy sample was divided into two equal pieces. One piece was immediately frozen in liquid nitrogen, placed in an Eppendorf tube and stored at -70°C for subsequent determination of glycogen content. The second

piece was mounted for cutting before being frozen in liquid N_2 and stored for later staining (described subsequently, in section 3.3.7).

3.3.6 Total muscle glycogen

The glycogen content of each muscle sample was calculated using the method of Passonneau and Lauderdale (1974) which measures glycogen as glucose equivalents after alkaline digestion of the muscle, precipitation and washing of the glycogen and subsequent acid hydrolysis to glucose. The glucose is then measured using a conventional glucose analyser after neutralisation of the hydrolyslate.

Specifically, frozen muscle samples (20-30 mg) were dissected free of blood, connective tissue and fat, and then weighed to within 0.1 mg, without defrosting, in pre-weighed 1.5 ml reaction vials containing 200 µl of cold 40% KOH. Tubes were then heated at 100 °C for 30 min in a heating block with occasional mixing to dissolve the tissue. The vials were then removed and allowed to cool to room temperature.

The glycogen was then precipitated by adding 0.8 ml of absolute ethanol to the vials, mixed, and allowed to stand overnight at 4 °C. The vials were then centrifuged at 8,500 revolutions/min for 10 min in a micro centrifuge. The supernatant was then carefully aspirated with a syringe and needle without disturbing the glycogen pellet, and 0.2 ml absolute ethanol added to wash the pellet. The vials were then centrifuged for a further two min and the supernatant removed as before.

The glycogen pellet was then hydrolysed to glucose by adding 0.2 ml of 2N HCl^- to the vials and heating in the heating block at 100 °C for three h with occasional mixing. The vials were then removed and 0.1 ml of Tris buffer (0.2 M pH 7.5) and 4 µl Universal Indicator added. The pH was then adjusted to 7.5 using 2 M NaOH (approximately 160 µl) and the vials weighed to determine final volume. Glucose concentration of the samples were then determined on a Beckman Glucose analyser and muscle glycogen content (as glucose equivalents) determined as follows:

$$\textbf{Muscle glycogen} = \textbf{[glucose]/18} \bullet \textbf{(F - T)/S} \qquad \textbf{(Equation 7)}$$

where: muscle glycogen (mmol/kg) is glycogen present in the sample; glucose is the measured blood glucose concentration (mg/dL) following hydrolysis of the sample; F - T is final weight of the sample and vial minus vial weight (g), and; S is the weight of the initial sample (g).

The coefficient of variation (CV) for this assay in our laboratory is <5% for duplicate glycogen assays of a single piece of muscle and <7% for assays of the glycogen content of separate pieces of the same muscle biopsy (Hawley et al. 1997).

3.3.7 Muscle fibre typing and histochemical analysis

The second piece of muscle was cleaned of blood and connective tissue, orientated on cork, and embedded in OCT compound (Tissue Tek, Miles Scientific, Illinois, USA). The sample was then frozen by lowering it into a beaker containing liquid nitrogen and agitating it repeatedly for several seconds. The frozen sample

was then wrapped in parafilm, placed in a labelled plastic bag, and stored at -70°C until it was sectioned.

Cryostat sections (~ 10 μm) of each muscle sample were cut at -20 °C using a SLEE-HRM cryostat (South London Electrical Equipment Co., London, UK). Serial sections of each sample were stained for determination of slow twitch (type I), and fast twitch (type II) fibres using ATP-ase (pH 4.3, 4.6, and 9.3) (Brooke and Kaiser 1970) and glycogen content using Periodic Acid Schiffs (PAS) and PAS-D. (Pearse 1961).

Myosin ATP-ase Stain

Following cutting, slides were allowed to dry in air for 5-10 min. The first slide was stained for pH 9.4. This was incubated in a rinse buffer (1) containing: 5 ml of 2% sodium barbitone; 2.5 ml of 2% calcium chloride, and; 17.5 ml distilled H_2O. After 10 min a further two slides were incubated at either pH 4.3 or 4.6 in rinse buffer (2) (5 ml veronal acetate buffer [1.94 g sodium acetate and 2.94 g sodium barbitone in 100 ml distilled H_20], 10 ml 0.1 N HCl and 8 ml distilled H_2O, adjusted to the desired pH with 0.1 N HCl) for a further five min.

All three slides were then rinsed with rinse buffer (1) as described above, drained, and incubated for 45 min in ATP substrate (containing 5 ml 2% sodium barbitone, 2.5 ml 2% calcium chloride, 0.02 g ATP [di sodium salt] in 17.5 ml distilled H_20).

Slides were then rinsed three times in 1% $CaCl_2$ (total 10 min), three times in 2% cobalt chloride (total six min), and three times in 0.01 M sodium barbitone (total 10 min). Next slides were washed with tap and distilled H_20, placed into a minimal

amount of 1% ammonium sulphide, washed again, and finally dehydrated, cleared and mounted.

At pH 9.4 type II fibres stain dark, while type I remain light. Under pH 4.3 the reverse occurs, whilst at pH 4.6 type I stain dark, type IIa stain lightly and type IIb stain dark. Hence, with a comparison of stains for pH 4.3, 4.6 and 9.4 the fibre type can be determined.

Periodic Acid Schiffs (PAS) Stain

In order to estimate the glycogen content of each muscle fibre, sections of the muscle were stained using the periodic acid Schiffs (PAS) stain. The section was incubated in 0.5 % periodic acid for five min, and then rinsed. The slide was then incubated in Schiffs solution (1 g basic fuchsin, 2 g sodium metabisulphate, 2 ml concentrated HCl, and 0.2 g decolourising charcoal in 200 ml distilled H_20) for 15 min before being washed in running H_20 for a further 10 min. The slide was then incubated in haematoxylin for one min, blue in Scotts T.W.S. and washed in running water. Finally, the slide was dehydrated, cleared and mounted.

A second slide was intubated in a 1% amylase solution for 30 min at 37 °C prior to being stained for PAS. This stain was undertaken to assess how much of the reaction to PAS was a reaction to capillaries, cytoplasm and other cellular materials. This staining allowed for visualisation of glycogen in the muscle cell: the darker the stain, the greater the concentration of glycogen present.

Stained samples were marked and presented double-blind to an independent pathologist for determination/explanation of staining patterns through fibre type.

Further, sections from each biopsy sample were magnified using a Leica DRA microscope (Leica Technology B.V., Rijswijk, The Netherlands) and digitised with a Leica Quantimed 500 Image system. The intensity of the PAS staining in each individual muscle fibre, of each slide was automatically rated by a grey scale value of 0 to 250 (dark to light) using Adobe Photoshop Version 4.0 (Adobe Systems Inc., Seattle, Washington). These values were then scaled into a rating of 1 to 5 (light to dark) to allow for the estimation of glycogen content in each individual fibre. These sections were then visually cross matched for individual fibre type using the ATP-ase stained sections to allow for determination of glycogen concentration through fibre type. Each section contained an average of 98 ±5 fibres.

3.3.8 Rating of Perceived Exertion (RPE)

In the experiment described in Chapter Nine, ratings of perceived exertion (RPE) were obtained using the 20 point Borg scale (Borg 1975) every 10 min for the duration of both the paced steady state and variable intensity rides.

SECTION A

PHYSIOLOGICAL FACTORS ASSOCIATED WITH CONSTANT AND VARIABLE LOAD CYCLING PERFORMANCE

CHAPTER FOUR

HEART RATE RESPONSES DURING A FOUR DAY CYCLE STAGE RACE

4.1 Introduction

While the physiological responses to endurance exercise in a laboratory setting under steady state conditions have been well documented, there is little information currently available concerning the physiological responses of well trained cyclists during either training or actual competition (see 2.2). Therefore, the aim of the first experiment of this thesis was to monitor heart rate (HR), and assess the physiological responses of a group of top amateur cyclists during a four-day stage race which is held annually in Cape Town, South Africa in early March (summer).

4.2 Experimental design

The seven subjects in this investigation (Table 4.1) were recruited from a total of 126 competitors in the 'Giro del Capo' cycle race. The 'Giro' is by invitation only, and is open to top local and international amateur and professional riders only. The race consists of four consecutive days of racing, comprising a 16.0 km individual time trial on the first day, and a mass start 110.0 km road race on day two. On the third day competitors completed a 5.5 km hill climb, and on the final day they rode in a mass start 105.0 km road race. During the event, the average climatic conditions during the races were 21.4 °C and 73% relative humidity, with a mean barometric pressure of 762.5 mm Hg (101.7 K Pa).

Table 4.1 Physiological characteristics of subjects.

Subject	Mass (kg)	$\dot{V}O_{2peak}$ (L/min)	Lab HR_{peak} (beats/min)	Field HR_{peak} (beats/min)	PPO (W)	P/W (W/kg)
1	67.0	5.09	191	194	352	5.25
2	74.0	4.69	190	192	403	5.45
3	62.5	5.54	192	202	392	6.27
4	81.5	5.30	175	187	415	5.09
5	86.5	5.55	194	194	417	4.82
6	70.0	4.99	201	190	406	5.80
7	80.0	4.98	194	196	403	5.04
Mean	74.5	5.16	191	194	398	5.39
SD	± 8.5	± 0.32	± 8	± 5	± 22	± 0.50

$\dot{V}O_{2peak}$, peak oxygen uptake; Lab HR_{peak}, the peak heart rate recorded during the maximal laboratory test; Field HR_{peak}, the peak heart rate recorded during any race; PPO, peak sustained power output measured during the maximal laboratory test; P/W, power-to-body mass ratio (PPO/BM).
All values are mean ± SD.

During each day's competition subjects were required to wear a HR monitor (see 3.1.3) which recorded HR at 60 s intervals, except during the shorter hill-climb (stage three) during which 15 s intervals were chosen. As described previously (Section 3.1.3), if HR data recorded were found to be outside the normal

physiological range for exercise (i.e. >220 or <50 beats/min) for more than five percent of any subject's total race time, they were not used in the sample for that day's racing. This meant that for statistical purposes each stage of the race had an 'n' of five subjects.

In order to estimate $\dot{V}O_2$ from the data recorded in the field, subjects were also required to report to the laboratory and perform a maximal incremental test to exhaustion on the Lode ergometer (as previously described in section 3.1.1 and 3.2.2) within ten days of completing the 'Giro'.

4.3 Results

Figure 4.1 shows the relationship between each subjects $\dot{V}O_2$ and HR during the maximal incremental test. This linear regression of:

$$\%HR_{peak} = (0.64 \pm 0.04) \bullet \%\dot{V}O_{2peak} + (36.7 \pm 3.3) \qquad \text{(Eq. 8)}$$

obtained from all subjects (N=7) was subsequently used to determine the relationship between the HR recorded in the race and the estimated $\dot{V}O_2$.

Figure 4.1 The regression data from the maximal laboratory test for the percentage of peak heart rate versus oxygen uptake. The 95 % confidence intervals are illustrated by the red dashed lines.

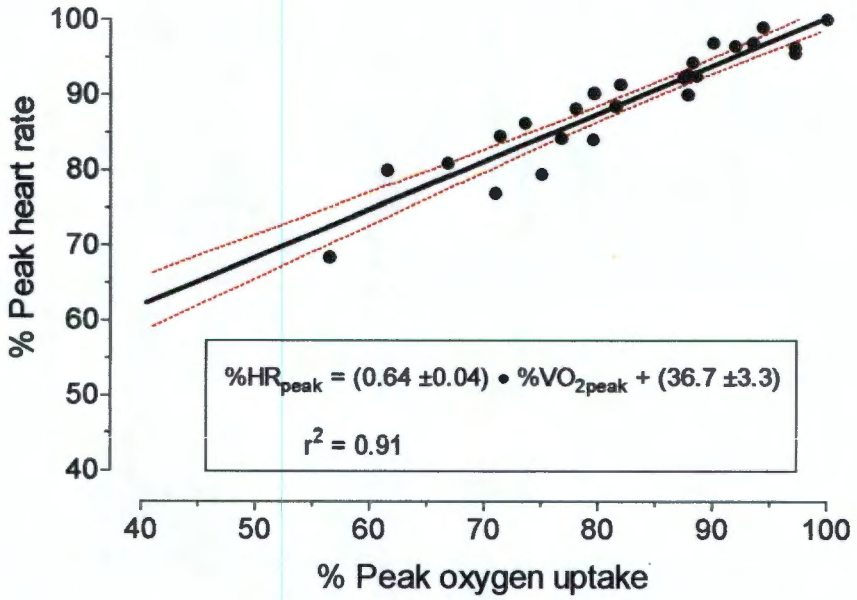


Table 4.2 shows the distances, mean times and placings, average velocities, and the percentage of HR_{peak} and percentage of estimated $\dot{V}O_{2peak}$ attained during the four races. The duration of the two mass start races (Day two, 110.0 km in ~2:45 h:min; Day four 105.0 km in ~2:35 h:min) were very similar. On the other hand, despite the time trial (day one) being six times shorter than the group races (16.0 km in ~23 min), it was ridden at a similar speed (42.2 ±1.0 km/h for the time trial and 39.9 ±0.2 and 40.6 ±0.5 km/h for the 110.0 and 105.0 km road races respectively).

Table 4.2 Race characteristics and subject performances.

Race Type	Time Trial	Road Race	Hill Climb	Road Race
Distance (km)	16.0	110.0	5.5	105.0
Race Time (h:min:s)	0:22:47 ± 0:00:33	2:45:15 ± 0:00:44	0:14:38 ± 0:01:14	2:35:10 ± 0:02:02
Range	0:22:20 - 0:23:41	2:44:30 - 2:45:57	0:13:01 – 0:15:53	2:33:51 – 2:38:27
Race Position	49.2 ±24.6	50.8 ±35.7	65.4 ±51.0	52.2 ±45.1
Range	30 - 91	3 – 84	2 - 116	5 - 107
Velocity (km/h)	42.1 ±1.0	39.9 ±0.2	22.7 ±2.0	40.6 ±0.5
% Field HR_{peak}	94.11 ±2.48	81.91 ±9.62	93.18 ±4.74	78.65 ±8.87
% $\dot{V}O_{2peak}$	90.5	71.2	89.0	66.1

h:min:s, hours:minutes:seconds; % Field HR_{peak}, the percentage of maximal heart rate recorded during any race; % $\dot{V}O_{2peak}$, the approximate percentage of maximal oxygen uptake measured during the maximal laboratory test.

All values are mean ± SD. n = 5 per race.

The average HR's recorded during the 16.0 km individual time trial (day one) are displayed in Figure 4.2A. After only two min, the mean HR had increased to 182 ±5 beats/min (93.8% of HR_{max} or 89% of estimated $\dot{V}O_{2peak}$), where, apart from a slight decline between 11 and 12 min which was a result of a downhill section of the course, it remained for the duration of the ride.

The HR responses of five subjects during the 5.5 km hill climb (day three) are displayed in Figure 4.2B. HR increased to 173 ±5 beats/min after only 2 min of the

climb and thereafter remained between 171 and 191 beats/min for the remainder of the ride. The mean intensity of the entire ride was ~89% of estimated $\dot{V}O_{2peak}$, which is the same as was observed during the 16.0 km time trial.

The responses during the two mass start road races were also similar: HR's were 81.9 ±9.6% of peak field HR on day two versus 78.6 ±8.9% of peak field HR on day four, whilst the mean velocities recorded were 39.9 and 40.6 km/h for days two and four respectively. Because of these similarities, only the data obtained during the final days race (105.0 km) are shown (Figure 4.3). As can be seen, the HR response (Upper Panel) *appears* to mirrors the course profile (Lower Panel) such that any increase in gradient is accompanied by an increase in HR and vice versa. The mean HR for the entire race was 151 ±17 beats/min, which corresponds to ~66% of $\dot{V}O_{2peak}$. In the 110.0 km race (day two) the mean HR was 157 ±18 beats/min, or ~71% of $\dot{V}O_{2peak}$.

Figure 4.2 Individual subjects (n=5) heart rate data recorded during: (A) the 16.0 km time trial and (B) the 5.5 km hill climb event.

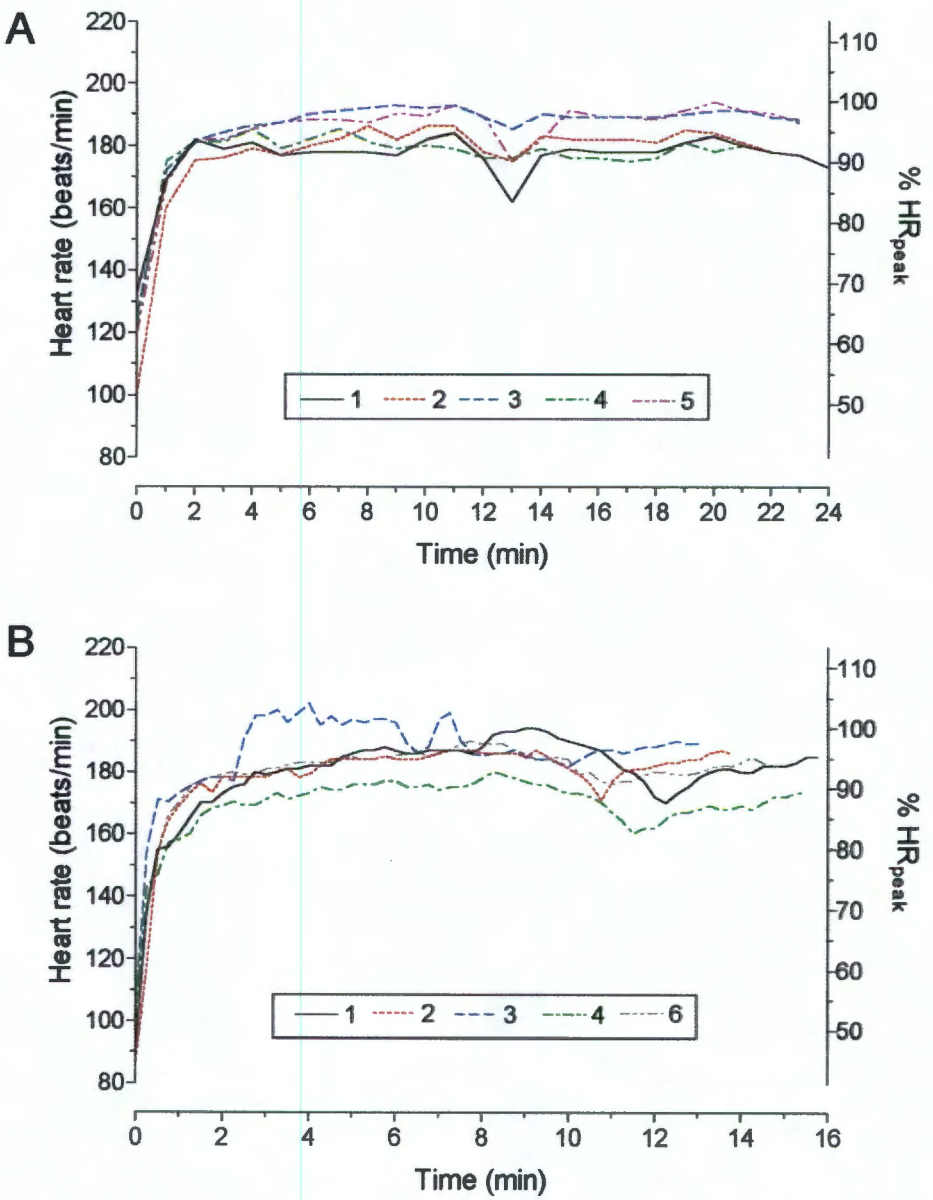


Figure 4.3 The individual subjects (n=5) HR data recorded during that race (Upper panel) shown as raw values and a percentage of HR_{peak} and the course profile of the 105.0 km mass start road race (Lower panel).

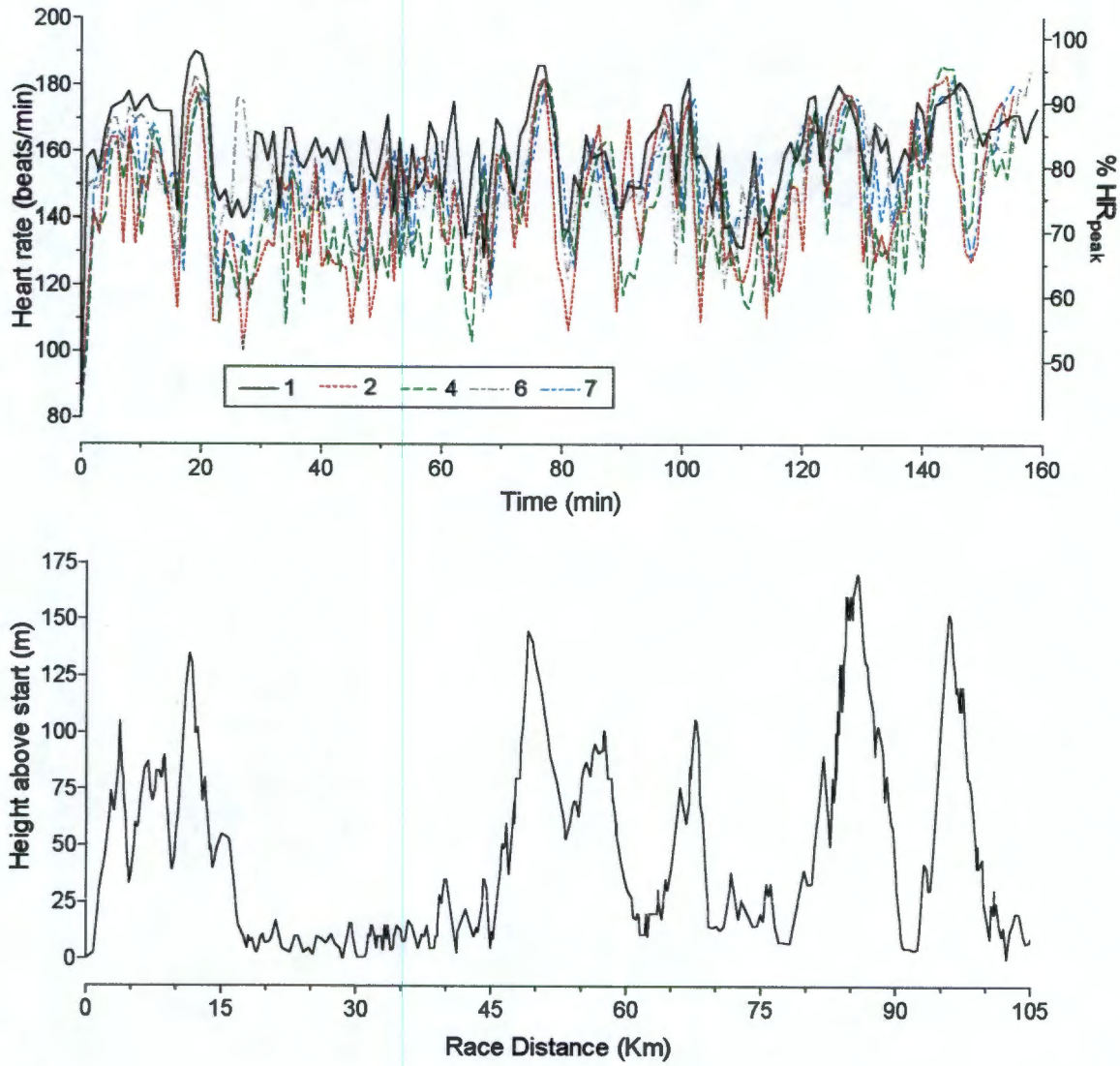


Figure 4.4 shows the percent of race time spent at various percentages of HR_{peak}, for the 5.5 km hill-climb (Panel A) and the 105.0 km mass start road race (Panel B). The difference in distribution of HR and time between individual and group races are clearly highlighted by the mean and the standard deviation values of 93.2 ± 4.7% and 78.6 ± 8.9% for the individual hill climb, and the mass start 105.0 km road race respectively.

The difference between the individual events and the bunch events is again illustrated by the distribution of percentage of time spent at different race intensities which is shown for all four stages in Table 4.3. As can be clearly seen in Table 4.3, the majority of the race is spent at HR between 91-100% of HR_{peak} or 81-100% of estimated $\dot{V}O_{2peak}$ in the individual events (TT and hill climb). In contrast, the majority of the longer group races are spent between 71-90% of HR_{peak} or 51-90% of $\dot{V}O_{2peak}$.

Figure 4.4 Percentage of total race time spent at specific exercise intensities in: (A) 5.5 km hill climb and (B) 105.0 km group race.

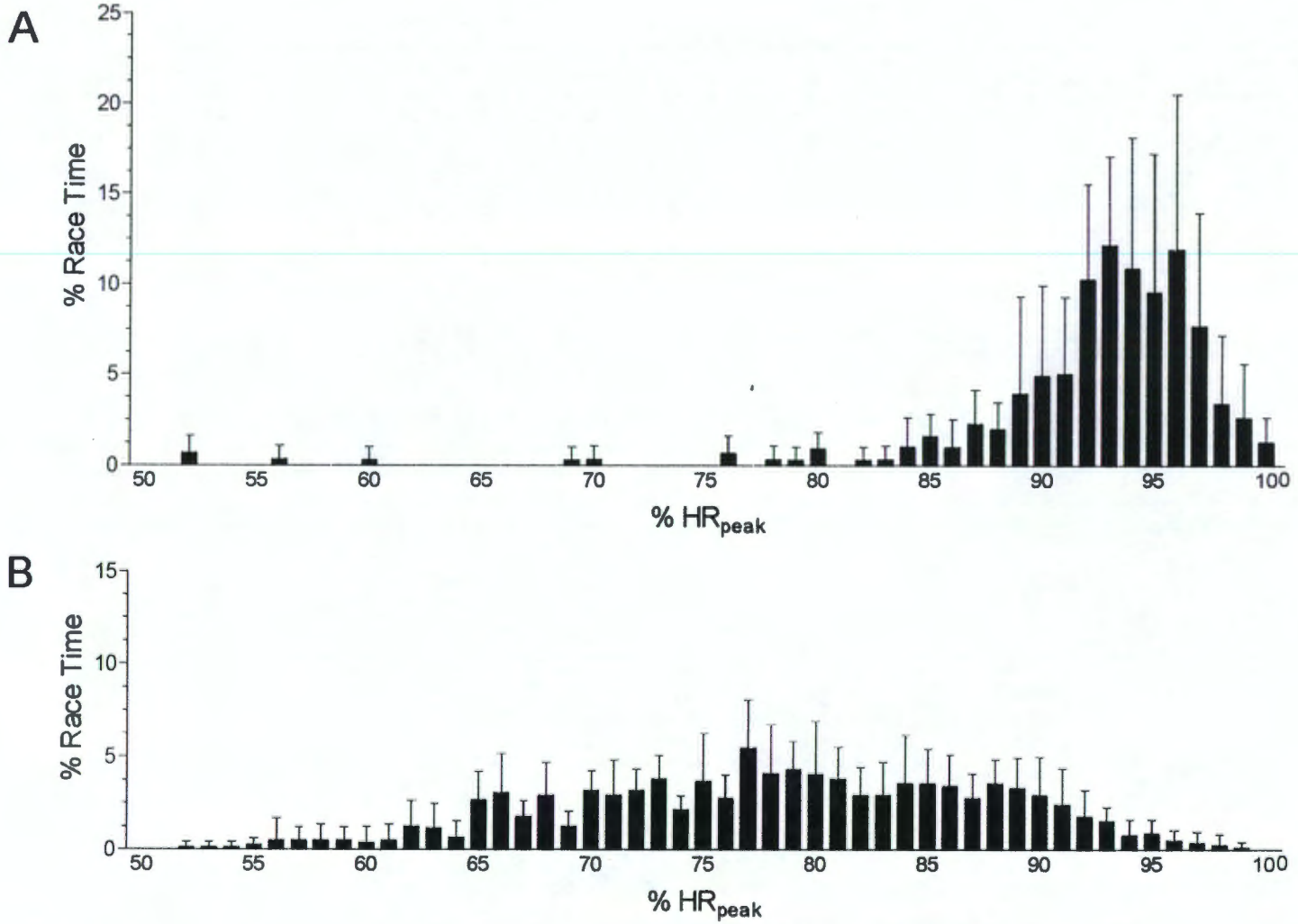


Table 4.3 The percentage of total race time spent at differing exercise intensities.

% Field HR_{peak}	Stage 1	Stage 2	Stage 3	Stage 4
51-60	0.9 ±2.0	3.6 ±3.6	1.3 ±0.8	3.1 ±3.2
61-70	3.5 ±2.0	8.8 ±3.5	0.6 ±0.9	18.6 ±9.3
71-80	0.0 ±0.0	25.3 ±2.8	2.3 ± 0.9	36.7 ±7.7
81-90	7.7 ±6.2	44.3 ±5.8	17.6 ±14.8	32.9 ±11.1
91-100	92.3 ±6.2	17.8 ±6.5	75.0 ±11.8	8.7 ±4.8

% $\dot{V}O_{2peak}$	Stage 1	Stage 2	Stage 3	Stage 4
31-40	1.7 ±2.4	2.5 ±2.2	0.3 ±0.7	3.7 ±.3.4
41-50	1.7 ±2.4	6.6 ±4.0	0.3 ±0.7	12.3 ±7.4
51-60	0.0 ±0.0	11.2 ±1.9	0.6 ±0.9	16.6 ±2.1
61-70	0.0 ±0.0	18.8 ±3.2	2.3 ±0.9	24.6 ±6.5
71-80	3.5 ±2.0	21.7 ±3.9	4.4 ±4.1	20.3 ±6.2
81-90	40.5 ±31.2	29.3 ±8.9	40.7 ±20.3	18.4 ±6.6
91-100	56.0 ±32.2	7.5 ±3.7	47.5 ±21.7	2.9 ±2.1

%Field HR_{peak}, the percentage of peak heart rate recorded during any race; % $\dot{V}O_{2peak}$, the approximate percentage of peak oxygen uptake as recorded in the maximal laboratory test. All values are mean ± SD. n = 5 per race.

4.4 Discussion

The major finding of this study was the markedly different HR responses observed during the individual TT stages on days one and three (Figure 4.2) compared to the mass-start bunch races on days two and four (Figure 4.3). During the shorter, individual races in which the cyclists are self paced, they race against the clock to record the fastest speed and the shortest possible time. The result is that riders produce near maximal efforts from the start of the race, rapidly attaining high (~93% of peak) HR which are then maintained for the remainder of the race (Figure 4.2).

While Foster et al. (1993b) have previously shown high and constant HR responses to short distance (2,000m) laboratory simulated TT, the maintenance of a constant HR for the duration of longer distance TT events also appears to hold true. Observations of an Olympic gold medal cyclist during a 50 mile (80 km) TT on an undulating course (total elevation > 400 m) revealed a HR of 178 ± 5 beats/min for the total duration of 1:44:49 h:min:s. (G. S. Palmer and L. Passfield, unpublished observations, 1992).

In contrast to the observations made during the individual time trial races, HR during both mass-start road-races were more random with variable changes in the frequency and amplitude of the responses. Although it is tempting to speculate that this stochastic physiological response is merely a reflection of, and directly related to, the terrain of the course (Figure 4.3), closer examination of the data reveals that is not the case. Whilst large climbs resulted in an expected increase in HR (Figure 4.3 upper and lower panels), the HR responses of the cyclists remained highly stochastic, albeit slightly lower, during a relatively flat section of

the 105.0 km race, which occurred between 20 km and 40 km of the race. This suggests that the major factor which is responsible for the stochastic nature of the HR responses in the mass-start races is not the course profile *per se* but rather, a result of the differing exercise intensities caused by group dynamics of the cyclists. Further confirmation that group dynamics exert the major influence on energy expenditure during mass start cycle races are the personal observations of several practitioners working with elite cyclists. In professional mass start road races, HR's are maintained within a very narrow range despite considerable variations in course terrain (N. Terrados; I. Mujika, Personal Communications, 1998). These researchers both suggest that this may be due to control of the pace of the race by a strong team, or teams.

Jeukendrup and van Diemen (1998) warn, however, that it is important to determine whether HR is a reflection of exercise intensity *per se*, or more a measure of whole body stress. These authors note that HR can be affected by riding position, "cardiac drift", or environmental conditions such as heat or altitude. Jeukendrup (Personal Communication, 1998) has also noted that as the ability of the rider increases the range of the HR response decreases. Although this may be because these riders are less affected by their environment, and HR is more a reflection of actual exercise intensity, rather than whole body stress, it could also be due to more control of the race pace by stronger teams in the peleton.

The stochastic responses in HR observed in well-trained amateur cyclists in this study may be explained by reductions in the $\dot{V}O_2$ when "drafting" behind another competitor's wheel, which frequently occurs during group racing. For example McCole et al. (1990) have shown that at speeds similar to those measured during

two of the stages of the races in this study (i.e. ~40 km/h), a cyclist riding at the back of an eight-man pack can reduce his $\dot{V}O_2$ by up to 39% compared to when he rides alone. This energy saving allows riders working as a group to ride up to 5 km/h faster than a single rider (Kyle 1988; Sjogaard et al. 1986; Whitt and Wilson 1974). Thus, whilst it is viewed to be advantageous to follow, or 'draft', another cyclist to reduce energy expenditure, this riding strategy probably accounts for the differences in exercise intensity as riders move within, or in and out of the main bunch.

The poor relationship between race time and overall position also highlights the effect of 'drafting' and tactics within the main group of riders. In the shorter 16.0 km TT and 5.5 km hill climb the range of finishing times was 6% and 20% of the total race time respectively, while the range of race time is less than three percent of total race time in the longer duration mass-start events (Table 4.2). This suggests that the pace is dictated by the bunch, or possibly the break-away group, as opposed to the individual cyclist. More to the point, the finishing time of a rider in a group event is dictated by the first rider across the line in a particular bunch. In this respect some riders conserve energy for the final sprint for first place, whilst others choose to record the same overall time as the race winner and finish lower in the placings. Thus, two riders may record an identical race time and yet be separated by 20 or more places. Further, since in large groups it may take the whole field several minutes to cross the finish, the large spread in race position and relatively small spread in race time may be somewhat misleading. This will allow some athletes an 'easy' ride as they are content to maintain the pace of the group whereas in the individual event they must produce a continuous effort close to the maximum of their ability.

Another factor which might explain the stochastic nature of the HR response during mass-start events is that large cyclists are at an advantage while cycling on level roads when compared to their smaller counterparts, owing to a greater reduction in wind resistance and hence higher power to frontal area when they adopt a "low profile" racing position (Swain et al. 1987). Again, the reduction in wind resistance conferred by bunch riding may be a cause of the decreased HR observed in the group events, with changes in exercise intensity being caused as the rider moves within the bunch whilst still maintaining a constant speed. However, these results need to be interpreted with caution as Swain et al. (1987) only tested cyclists at speeds of up to 20 miles/h (32 km/h), a velocity which is far below that experienced in top amateur and professional racing, and at which wind resistance has less of an impact on riding velocity.

The second finding of this study was the observation of differences between the peak HR measured in the maximal laboratory test and that recorded in the field (Table 4.1). Although not *statistically* significant, this difference may be of *physiological* significance when trying to identify the optimal training intensities for endurance athletes based on the results of laboratory tests. Differences in peak HR determined in the laboratory compared to the field setting may be due to a number of reasons, including (i) differences in the mechanical efficiency of the cyclist on their own bikes compared to the laboratory ergometer, (ii) a failure of the progressive, incremental maximal test employed to elicit a true peak HR, due to the fact that subjects remained seated in the test, whereas they frequently ride out of the saddle for long periods in the field and (iii) the psychological motivation of the athlete to really push himself to his maximum capacity in the laboratory.

Greater regard for the difference in peak HR must also be made when it is considered that the number of subjects was small (n=7) and the range in peak HR values was fairly large (26 beats/min in the case of Lab HR_{max}).

In conclusion, this is the first study to report the physiological responses of competitive cyclists to a multi-day stage race. The results highlight the stochastic nature of group/bunch cycling in well-trained amateur riders and show that the energy expenditures of these elite racing cyclists are probably a function of bunch riding. This finding, and the differences in peak HR recorded in the field and laboratory, raises the question of the validity of "steady-state" laboratory testing to prescribe training intensities, predict performance, or evaluate metabolic responses to events which are largely stochastic in nature. It also highlights the need for sports scientists to develop reliable and valid laboratory based testing protocols which accurately simulate competitive conditions.

CHAPTER FIVE

ASSESSMENT OF THE REPRODUCIBILITY OF PERFORMANCE TESTING ON AN AIR-BRAKED CYCLE ERGOMETER

5.1 Introduction

The results of the study described in Chapter Four illustrate that during time trial (TT) and individual events, cyclists maintain high and constant heart rate (HR) responses even when perturbations in the course profile occur. Furthermore, it was shown that cyclists are often capable of attaining higher HR when racing, compared to when they are required to undertake a "maximal" test in the laboratory. Accordingly, the first aim of the second study in this thesis was to measure the physiological responses of cyclists in a laboratory simulated TT and to determine if such responses were similar to those previously recorded in the field (Chapter Four). A second aim was to assess the reliability of a laboratory ergometer during repeated rides over 20 and 40 km. The issue of reliability is important because, to date, the vast majority of investigations which have measured "performance" in the laboratory have utilised either the exercise time to exhaustion at a fixed work rate, or the change a selected submaximal physiological variable as a measure of exercise capacity. While these are perfectly valid measures in themselves they do not reproduce the metabolic or physiological demands of either TT or mass start road races.

5.2 Experimental Design

In this investigation, six well-trained subjects (Table 5.1) were required to perform, a random order of three 20 km and three 40 km TT on the Kingcycle ergometry system (described in detail in section 3.1.2). Pre-experimental control was maintained as described previously (section 3.2.3). During all laboratory trials,

environmental conditions were standardised at 20 °C, while relative humidity ranged from 65 - 75% and barometric pressure from 1019 to 1024 mmHb.

During the first ride, subjects were allowed to consume either water or a carbohydrate (CHO) beverage *ad libitum*, but subsequently the drink chosen and the volume consumed by each subject remained the same for all tests.

Table 5.1 Subject Characteristics.

Subject	Mass (kg)	Height (m)	Age (yr)	HR_{peak} (beats/min)	PPO (W)	Power/Weight (W/Kg)
1	69.0	1.82	23	191	396	5.74
2	77.0	1.70	29	178	437	5.68
3	74.0	1.78	21	198	404	5.46
4	84.0	1.87	27	189	466	5.55
5	81.0	1.85	21	186	468	5.78
6	79.0	1.87	24	187	452	5.72
Mean	*77.3*	*1.81*	*24*	*188.2*	*437.2*	*5.65*
SD	*5.3*	*0.07*	*3*	*6.6*	*31.0*	*0.12*

Determination of peak heart rate (HR_{peak}), and peak power output (PPO) are described in the text (3.2.2). All values are presented as mean ± SD.

5.3 Results

Table 5.2a and Table 5.2b show the results of each cyclists performance in the simulated 20 km and 40 km TT respectively. The group CV for the 20 km TT was 1.1 ± 0.9%, and for the 40 km TT, it was 1.0 ± 0.5%.

Table 5.2a The times (min:s) for the 20 km Laboratory Time Trial

	1	2	3	4	5	6	**Group Mean**	**Group SD**
Trial 1	28:35	26:37	28:25	25:52	25:39	26:39	**26:58**	**01:15**
Trial 2	28:45	28:10	28:01	25:41	25:13	26:53	**27:07**	**01:26**
Trial 3	28:33	27:37	28:17	25:56	24:55	26:29	**26:58**	**01:26**
Mean	28:38	27:28	28:14	25:50	25:16	26:40	**27:01**	**01:20**
SD	00:06	00:47	00:12	00:08	00:22	00:12		
CV	0.37	2.86	0.72	0.50	1.46	0.75	**1.11**	**0.94**

All times represented are min:s. CV, coefficient of variation.

Table 5.2b The times (min:s) for the 40 km Laboratory Time Trial

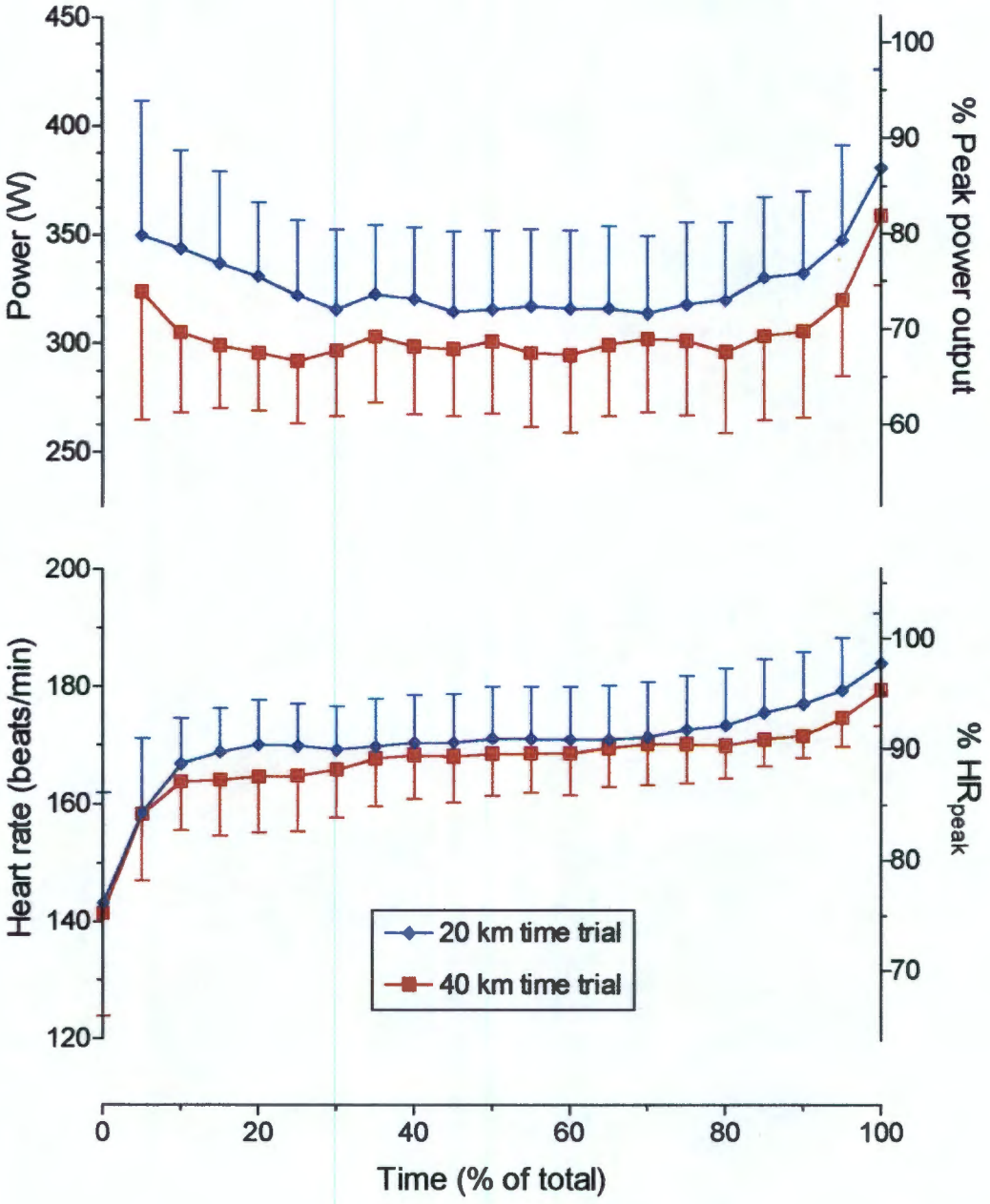
	1	2	3	4	5	6	**Group Mean**	**Group SD**
Trial 1	59:34	54:55	59:44	53:17	53:35	55:18	**56:04**	**02:53**
Trial 2	58:31	55:27	58:36	53:32	52:41	54:55	**55:37**	**02:29**
Trial 3	58:50	56:36	58:49	53:19	52:01	55:36	**55:52**	**02:49**
Mean	58:58	55:39	59:03	53:23	52:46	55:16	**55:51**	**02:41**
SD	00:32	00:52	00:36	00:08	00:47	00:21		
CV	0.91	1.55	1.02	0.25	1.49	0.62	**0.97**	**0.50**

All times represented are min:s. CV, coefficient of variation.

Figure 5.1 illustrates the power output and HR attained during the 20 km and 40 km simulated TT. The data presented are the group means (± SD) for each 5% interval of each subjects TT for all three trials (n=18).

As might be expected, the average power output for the 20 km TT was significantly greater than that during the 40 km TT (327.5 ± 16.9 vs. 303.9 ± 14.9 W; P<0.0001; 73.9 ± 3.8 vs. 68.6 ± 3.4% of PPO). The average HR during the 20 km TT was also higher than during the 40 km TT (171.4 ± 5.1 vs. 168.3 ± 4.4 beats/min; P<0.0001; 92.0 ± 2.7 vs. 90.3 ± 2.4% of HR_{peak}).

Figure 5.1 The group mean values for power output (W) (Upper panel) and heart rate (beats/min) (Lower panel) for each five percent section of the 20 km and 40 km laboratory time trials, (n = 6).



The relationships between the average power output of the six subjects and their cycling time (min) over the 20 km and 40 km laboratory TT are shown in Figure 5.2A and Figure 5.2B, respectively. A regression of the average power output and times for the 20 km TT yielded the following equation:

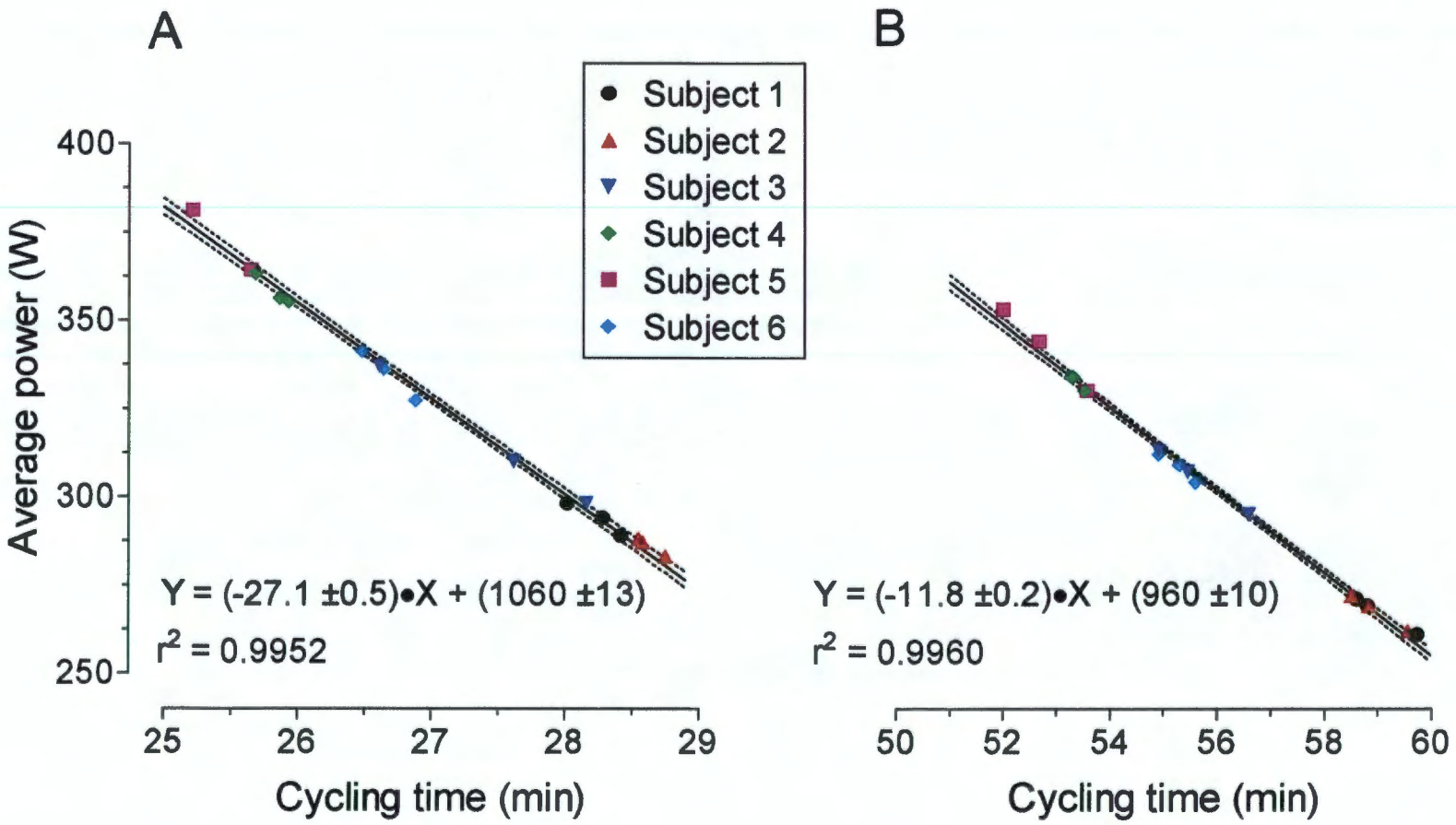
$$\mathbf{Y = (-27.1 \pm 0.5) \cdot X + (1060 \pm 13)} \qquad \textbf{(Equation 9)}$$

while the regression between the average 40 km power output and TT time resulted in:

$$\mathbf{Y = (-11.8 \pm 0.2) \cdot X + (960 \pm 10)} \qquad \textbf{(Equation. 10)}$$

These data show that for a 65 kg cyclist to ride a 20 km in a time of 26:40 or a 40 km in a time of 53:20 (i.e. a 45 km/h) an average power output of approximately 320 W must be maintained for the duration of the ride.

Figure 5.2 The relationship between the average power output (W) and cycling time for each 20 km (panel A) and each 40 km (panel B) time trial distance for each subject (n=6). The dashed lines illustrate the 95% confidence intervals.



5.4 Discussion

The major finding of this study was that laboratory simulated performance rides on the Kingcycle ergometer were highly reproducible, as indicated by the small CV between individuals performances over both 20 km and 40 km distances (Table 5.2). Such reliability is important because the Kingcycle ergometry system can now be employed for laboratory based testing of well-trained cyclists on their own bicycles.

As previously noted, most investigations that have assessed exercise capacity have usually relied upon changes in physiological function during exercise, or on the time to exhaustion at a fixed workload as a measure of *performance* (Maughan 1991). Such measures often fail to provide the sports scientist with the mechanisms underlying the true measure of performance. Furthermore, the test-retest reproducibility of several other commonly used testing protocols is relatively low (Jeukendrup et al. 1996; McLellan et al. 1995). Therefore, a major advantage of the Kingcycle system compared to traditional ergometry systems is that performance over a fixed distance or time can be accurately determined while the cyclist rides his/her own bike.

Jeukendrup et al. (1996) reported a "new validated performance test". They compared the results of several performance rides, each of approximately one hour duration. Ten different subjects completed six rides for each protocol. Protocol A involved subjects exercising continuously at ~75% of maximal power output until fatigue. The CV from the test was 26.6% with a range of 17.4 to 39.5%. Protocol B started with 45 min of exercise at 70% maximal power output, followed by a 15 min "time trial" where subjects were asked to produce as much

work as possible. CV for this trial was 3.5%, with a range of 1.2 to 5.8%. Protocol C required the subjects to perform a set amount of work, which would usually fatigue the subject in ~1 h, as quickly as possible. The CV for this trial was 3.4% with a range of 0.8 to 5.8%. Of importance is that the CV reported for all of these trials, including the most reliable protocol reported by Jeukendrup et al. (1996) are all much higher than that found in the current study for rides lasting ~30 min to 1 h.

Of interest in the present study are the observed steady-state HR responses throughout both the 20 km and 40 km TT, when compared to large (~60 W) perturbations in power output (Figure 5.1). This finding highlights the variability of the relationship between HR and power when cyclists ride in the laboratory, an observation that has previously been reported during competitive road races (Chapter Four; Jeukendrup and van Diemen 1998). The Kingcycle ergometry system was also sensitive enough to detect the rapid but small changes in power output that occurred during the 20 km and 40 km TT (Figure 5.1). Although the difference in mean HR during the ride was only ~3 beats/min, there was an average difference in power output of ~23 W between the 20 km and 40 km TT.

The HR data recorded in the laboratory TT are comparable to the HR data recorded in shorter distance TT in actual competition and in other laboratory studies (Chapter Four; Foster et al. 1993a). In Chapter Four HR responses of 94.1 ±2.5% HR_{peak} during a 16.0 km TT in the field were reported. Similarly high HR responses were recorded in the present investigation during the simulated TT (~90% and ~89% of peak HR for the 20 km and 40 km respectively). In addition, during competitive TT events, athletes produce near maximal efforts from the

onset of the race, rapidly attaining a high HR, which was then maintained for the duration of the ride (Chapter Four). This relationship was observed for both power and HR responses recorded during simulated competition on the Kingcycle ergometer, a finding which is in agreement with the data on 5 km TT performance of elite and sub-elite athletes reported by Foster et al. (1993a).

In conclusion, the results of this study demonstrate that the laboratory simulated TT performances undertaken on the Kingcycle ergometry system over 20 km and 40 km were highly reproducible. This indicates that the Kingcycle ergometry system can be used by sport scientists as a reliable method of assessing short-term (< 60 min) endurance performance in the laboratory. However, it remains to be established whether the physiological responses of such simulated TT accurately reflect the performance capabilities of cyclists during actual competition.

CHAPTER SIX

ASSESSMENT OF THE VALIDITY OF PERFORMANCE TESTING ON AN AIR-BRAKED CYCLE ERGOMETER

6.1 Introduction

The results of the investigation described in Chapter Five show that performance rides undertaken by trained subjects in the laboratory over 20 km and 40 km on the Kingcycle ergometer are highly reproducible, having a CV of ~1%. Others (Jeukendrup et al. 1997; McLellan et al. 1995) have previously reported CV's ranging from 3.4% to 26 % for a variety of performance tests lasting approximately one hour. However, although the reliability of a test is an important pre-requisite for the sports scientist, the validity of a measure is also essential if laboratory results are to be used to monitor or predict actual race times during competition.

In addition to this, a further problem associated with many of the standard laboratory ergometers is that experienced cyclists often encounter difficulty in assuming their normal riding position during testing (Firth 1981). Accordingly, a number of air-braked cycle simulators have been adapted for use by trained cyclists in the laboratory (Argentieri et al. 1988; Clifford et al. 1988; Coast et al. 1988; Davies 1980, Dengel et al. 1990; Firth 1981; Foster et al. 1993a, 1993b; Keen et al. 1991; Lamont et al. 1992; La Voie et al. 1988, Oude Vrielink et al. 1984; Seifert and Langenfeld 1988; Telford 1982a, 1982b). Although such simulators allow cyclists to ride their own bikes in the laboratory, to date, they have been largely used to measure (Coast et al. 1988; La Voie et al. 1988, Oude Vrielink et al. 1984; Seifert and Langenfeld 1988) or predict (Dengel et al. 1990; Keen et al. 1991; Lamont et al. 1992) oxygen uptake during exercise. There has been little research which has evaluated the physiological and metabolic responses during simulated competition. In part this is because the validity of most ergometers has not been established.

Hence, the aim of this investigation was to compare time trial (TT) performances measured in the laboratory on the Kingcycle ergometer system with actual race performances over the same distance in competition on the road.

6.2 Experimental Design

Eight subjects (Table 6.1) performed three 40 km TT on the Kingcycle ergometer (3.1.2, 3.2.2 and 3.2.3) and also competed in both a provincial 40 km TT championship (Western Province, Cape Town, South Africa [WP-TT] championships) and a club 40 km TT race (City [C-TT]). Environmental conditions during the races were (values are range): temperature 14.8 - 15.7 °C vs. 8.8 - 15.2 °C; barometric pressure 1020 mmHb vs. 1029 mmHb; relative humidity, 51 - 54% vs. 82 - 100%, and; wind speed, 9.9 - 10.1 m/s vs. 2.2 - 2.3 m/s for the WP-TT and C-TT events respectively.

The two races were separated by a period of seven days. For the duration of each race, HR was recorded at 15 s intervals. Unfortunately, subjects 7 and 8 (Table 6.1) competed in the WP-TT only. Due to a malfunction in the HR monitoring device no HR data were recorded for subject 8, whilst subject 2's cycle developed mechanical problems during one of the races; thus a total of five athletes completed both road TT.

Table 6.1: Subject Characteristics.

Subject	Mass (kg)	Height (m)	Age (yr)	HR_{peak} (beats/min)	PPO (W)	Power/Weight (W/Kg)
1	74.0	1.78	21	198	404	5.46
2	84.0	1.87	27	189	466	5.55
3	81.0	1.85	21	186	468	5.78
4	79.0	1.87	24	187	452	5.72
5	69.0	1.75	22	181	411	5.96
6	82.0	1.80	27	172	458	5.59
7	86.0	1.86	25	190	520	6.05
8	75.0	1.79	20	191	421	5.61
Mean	78.5	1.82	23	186.7	450	5.71
SD	5.7	0.05	3	7.7	37.8	0.21

Determination of peak heart rate (HR_{peak}), and peak power output (PPO) are described in Chapter Three (3.2.2). All values are presented as mean ± SD.

6.3 Results

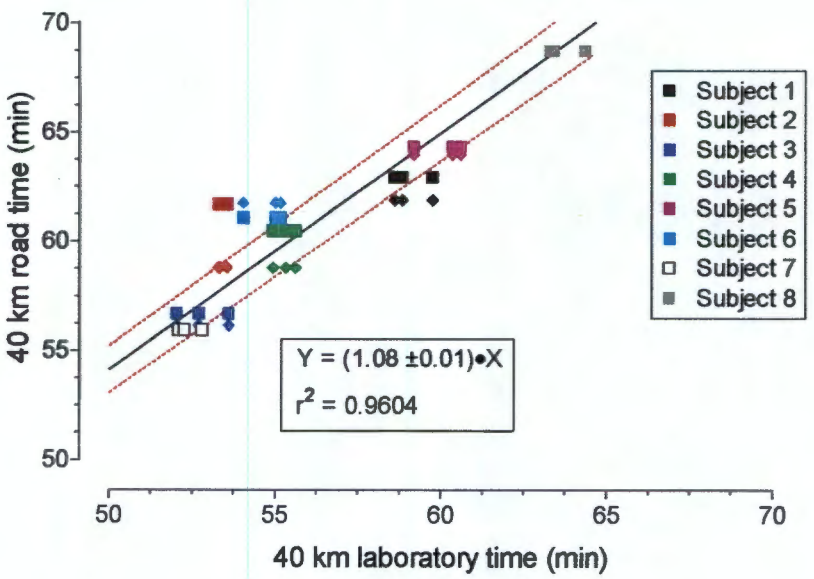
The relationship between the mean laboratory simulated 40 km time and two 40 km race times for each subject are displayed in Figure 6.1. Group means times were 56:24 ± 3:59 (n=8), 61:26 ± 4:25 (n=7), and 60:12 ± 2:49 min:s (n=6) for the laboratory, WP-TT and C-TT, respectively. A regression of the laboratory and field times yielded the following equation:

$$\mathbf{Y = (1.08 \pm 0.01) \cdot X} \qquad \textbf{(Equation 11)}$$

where Y is TT time recorded in the field, and where X is the 40 km TT time recorded in the laboratory.

It was calculated that, on average, the road TT races took approximately 8% longer than the laboratory based efforts of the same distance.

Figure 6.1: The relationship between the 40 km time recorded in the laboratory and two 40 km road TT times, with 95% confidence intervals. Each subjects performance in the WP-TT is indicated by the ■ symbol, the C-TT performance is indicated by the ◆ symbol, all three laboratory trials are shown for each subject.

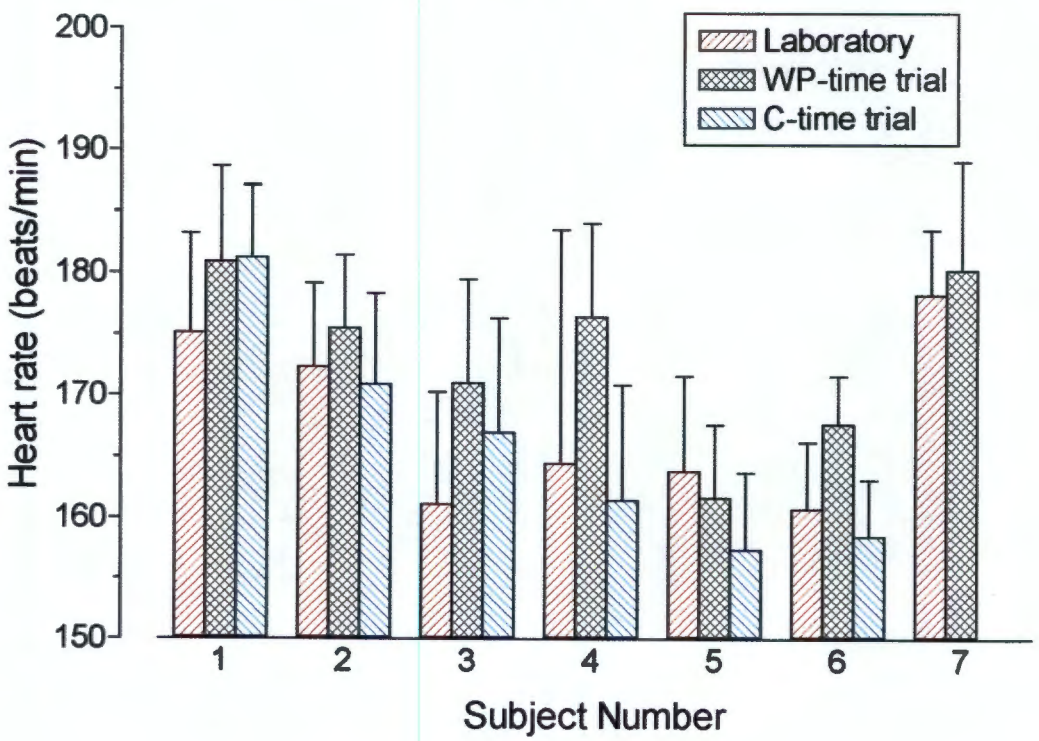


As the flywheel of the airbraked Kingcycle ergometer system is calibrated to approximate the resistance experienced by a rider of 65 kg on a flat road, a comparison of each riders average field TT time minus their average laboratory TT time (delta time) versus their weight minus 65 kg (the rider mass equating to the

total resistance offered by the Kingcycle system) was undertaken. Such analysis revealed that there was no relationship (r=0.039, p=0.48).

Figure 6.2 displays the average HR response of each subject for the three laboratory rides and for the competitive races. Group mean HR's were 167.9 ± 7.9 (n=8), 173.3 ± 7.0 (n=7), and 166.1 ± 9.0 (n=6) beats/min for the laboratory, WP-TT and C-TT, respectively. There were no differences in HR response between the laboratory TT and the C-TT. However, there were significant ($P<0.05$) differences in the HR response between the laboratory TT and WP-TT (n=7) and also between the C-TT and WP-TT (n=6) ($P<0.05$).

Figure 6.2: The average heart rate response (beats/min) for each subject for the 40 km laboratory time trials and for the two 40 km road time trials.



6.4 Discussion

The first important finding of this investigation was that the times recorded in the laboratory simulated TT were, on average, 8% faster than the TT performances in the field over the same distance (Figure 6.1). The slower road based TT compared to the simulated laboratory performances may result from a variety of factors including environmental conditions, route, terrain, road surface, and rolling resistance, all of which may cause a cyclist to ride slower in the field.

Swain et al. (1988) have previously suggested that larger riders may be at an advantage compared to smaller riders when cycling on flat roads by virtue of their greater $\dot{V}O_2$ relative to their frontal area. Indeed, in the current study there was a significant correlation between both road TT times and the BM of the rider ($r = -0.69$ and $r = -0.71$ for the WP-TT and C-TT respectively, $P < 0.05$). In addition there was a highly significant relationship between the average 40 km laboratory TT time and the riders BM ($r = -0.83$, $p < 0.001$). Of importance, however, was the finding that despite differences in rider BM from the equivalent resistance offered by the Kingcycle ergometer (range 4 – 21 kg, which is the difference between the total resistance provided by the ergometer [estimated to equate to the equivalent wind and rolling resistance experienced by a 65 kg cyclist] and the riders BM, see section 3.1.2), there was no differences between their laboratory and road TT times. This may have been the result of riders using a variety of methods to reduce their frontal area and/or drag coefficient, such as low profile cycles, aerodynamic handlebars, or disc wheels which serve to reduce the effects of mass and size on aerodynamics (Kyle 1994; McCole et al. 1990). Further, in races such as those used in this investigation, the course is often not a

completely straight line. Consequently, the prevailing wind will not act directly on to the rider, but will often be a crosswind. This may negate any possible advantage conferred by the superior $\dot{V}O_2$ to frontal area of the larger riders. Swain (1997) further suggests that if a rider maintains a constant power on a course with equal length segments of uphill and downhill, or direct headwind and tailwind, their overall performance time will be reduced compared to if they had increased power into uphill or headwind sections and reduced power into downhill or tailwind sections. This improvement would be in the region of 40 s for a rider in a 40 km TT who is able to sustain an average $\dot{V}O_2$ of 5 L/min, with a 10% variation from this average.

In addition the road surface for the majority of public highways will not be as smooth as the flywheel surface of the Kingcycle ergometer, thus increasing rolling resistance due to friction. Furthermore, in actual competition riders often have to cycle with their head raised in order to take care to avoid drain covers, or pot holes, in the road ahead. In the laboratory, however, the riders have little concern as to looking ahead, and are able to drop their head, thereby alter their riding position, saving energy and ensuring maximal power delivery. One further factor yet to be determined is the effect of deceleration into, and acceleration out of a corner in the road on the riders average power output.

The differences found between the HR recorded during the laboratory simulated TT and C-TT in comparison with the WP-TT also highlights that environmental factors are likely to be responsible for the differences in observed HR. Indeed, both road TT races were ridden on different courses and under differing environmental conditions (temperature 14.8 - 15.7 °C vs. 8.8 - 15.2 °C; barometric

pressure 1020 mmHb vs. 1029 mmHb; relative humidity, 51 - 54% vs. 82 - 100%, and; wind speed, 9.9 - 10.1 m/s vs. 2.2 - 2.3 m/s for the WP-TT and C-TT events respectively). In addition, the WP-TT was the Provincial Championship event, which may have caused greater anxiety in some subjects compared to the laboratory simulated rides, or the Club championship event.

In conclusion the results of this study show that although the laboratory TT were, on average, 8% faster than the road based time trials over the same distance, the physiological responses were similar. This is an important finding as it allows exercise physiologists to simulate the physiological responses of TT competitions in the laboratory setting. Accordingly the second half of this thesis will focus on some of the physiological and metabolic determinates of cycling performance during both constant (i.e. individual time trials) and variable load (i.e. mass start road races) exercise.

CHAPTER SEVEN

EFFECTS OF CONSTANT LOAD VERSUS VARIABLE INTENSITY EXERCISE ON SUBSEQUENT CYCLING PERFORMANCE

7.1 Introduction

Different pacing strategies would be expected to have profound effects on both metabolism and subsequent cycling performance. Yet for the most part, studies which have attempted to examine the performance capacities of well trained individuals lack ecological validity in that they do not permit the athlete to choose their own pacing strategy. The results of the study described in Chapter Four clearly show that during group cycle races the responses to competition are stochastic, and vary in a random fashion. However, to date, almost all laboratory testing of cyclists has been undertaken using constant-load work, under steady-state conditions (Chapter Two). Such conditions, while often deemed necessary for valid metabolic measurements are often not a true reflection of field conditions, especially in mass start races.

Accordingly, the aims of the study described in this chapter were, firstly to evaluate the physiological responses of well-trained cyclists to laboratory based variable intensity exercise and, secondly, to assess the effects of prolonged, sub-maximal steady-state and variable intensity cycling on subsequent cycling time-trial performance. It was hypothesised that cyclists may choose to race stochastically because it confers a performance advantage.

7.2 Experimental Design

Each of six well-trained subjects (Table 7.1) was required to undertake two experimental rides. Each trial commenced with calibration of the Kingcycle ergometry system (described in Chapter Three), and was followed by a self

selected, standardised warm-up. Subjects then completed 150 min of cycling on the Lode ergometer. The rides consisted of either constant load (SS) or variable intensity (VI) work. During one paced ride the average power output was 58% of PPO (SS), while for the other trial, the same *average* exercise intensity for each cyclist, as defined by the area under the power versus time curve, was varied within one standard deviation of 12.2% of PPO (VI) (Figure 7.1). Both these paced efforts were immediately followed by a 20 km performance time trial (TT) on the Kingcycle ergometer.

Table 7.1 Subject Characteristics.

Subject	Mass (kg)	Height (m)	Age (yr)	$\dot{V}O_{2peak}$ (L/min)	HR_{peak} (beats/min)	PPO (W)
1	77.0	1.87	22	4.05	187	361
2	88.5	1.86	28	5.22	186	468
3	66.5	1.79	19	4.84	210	433
4	78.0	1.86	19	4.72	196	422
5	91.5	1.78	40	5.12	194	459
6	83.0	1.86	22	5.01	182	449
Mean	80.75	1.84	25	4.83	192.5	432.0
SD.	9.0	0.04	8	0.42	10.0	38.6

Determination of peak heart rate (HR_{peak}), peak power output (PPO) and peak oxygen uptake ($\dot{V}O_{2peak}$), are described in Chapter Three. All values are mean ± SD.

The range in power outputs during the VI ride were from 35.8 to 82.3% of PPO, or from ~155 to ~355 W. Such a range of power outputs were designed to mimic the average workrates of cyclists during the previously investigated 105 km mass start cycle race during which had been found to have a random variation in the frequency and amplitude of exercise over time (Chapter Four).

Such variable intensity exercise was reproduced in the present study from the HR measured previously during competition and the previously reported %HRpeak = 0.64•%$\dot{V}O_{2peak}$ + 36.7 **(Equation 8)** regression measured in the laboratory (Chapter Four). $\dot{V}O_2$ values were subsequently converted to power output using the equation of Keen et al. (1991).

Exactly 60 s after the subjects had completed the 150 min paced effort they commenced a 20 km performance TT on the Kingcycle ergometer.

In order to prevent the onset of hypoglycaemia and minimise cardiovascular drift during the 150 min rides, subjects consumed a carbohydrate (CHO) solution (5% maltodextrin [MAXIM™, AMS Ltd., Goole, UK]) at a rate of 10 ml/kg/h, while during the TT subjects were allowed access to water *ad libitum*.

7.3 Results

Figure 7.1 illustrates the group mean power outputs (W) and HR (beats/min) responses during the two 150 min rides. As described previously, the power output during this section of the trial was 58% PPO for the constant load ride and 58 ± 12.2% of PPO for the variable intensity effort. The mean HR recorded during

the constant load and variable intensity 150 min paced effort were not significantly different (153 ± 6 and 150 ± 11 beats/min; 79.6 ± 3.2% and 77.3 ± 5.9% of HR_{peak}, respectively).

Figure 7.1 The group mean values for (A) power output (W) and (B) heart rate (beats/min) for each min of the 150 min effort during the constant load and variable intensity rides. The green line indicates the mean heart rate response (n=6) from the 105 km mass start cycle race described in Chapter Four.

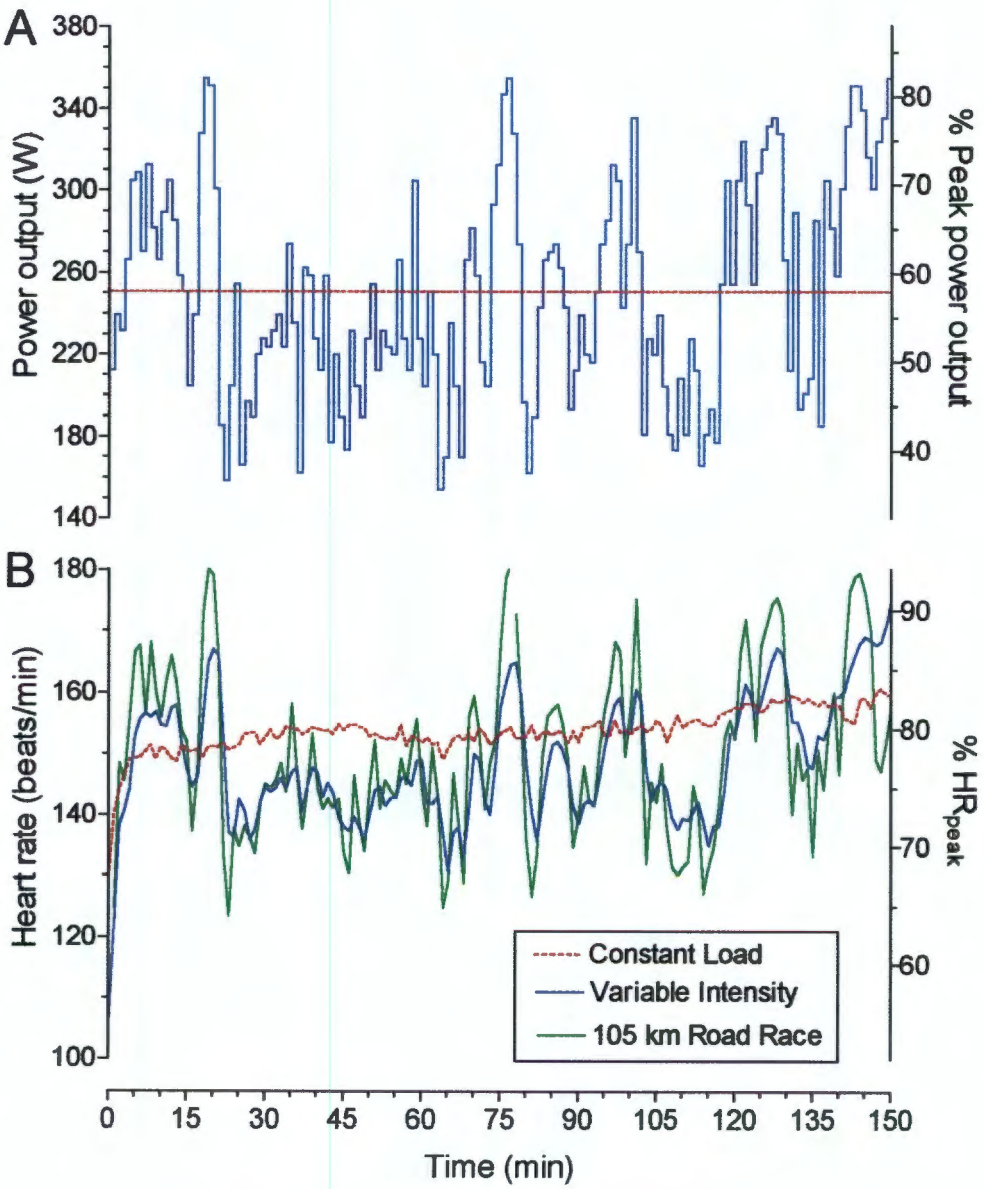


Table 7.2 shows the percentage of time spent exercising at different intensities during the 150 min variable intensity ride. Riders spent the majority of the time (49.5 ±15.6% and 31.7 ±18.5% of total time respectively) between 71-80 and 81-90% of HR_{peak}, with only 18.7% of total time spent outside this range (0.5 ±0.8%, 14.3 ±24.0% and 3.9 ±3.9% of total time between 51-60, 61-70 and 91-100% of HR_{peak} respectively).

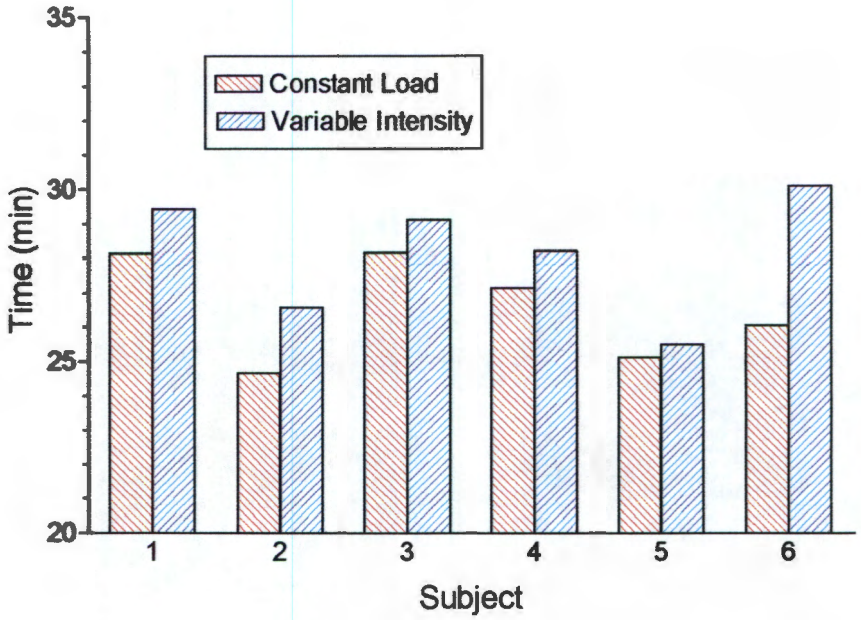
Table 7.2 The proportion of time spent at different percentages of peak heart rate during the 150 min variable intensity ride.

% HR_{peak}	51-60	61-70	71-80	81-90	91-100
Mean	0.5	14.3	49.5	31.7	3.9
S.D.	± 0.8	± 24.0	± 15.6	± 18.5	± 3.9

% HR_{peak} is the percentage of the peak HR recorded during the maximal exercise test.

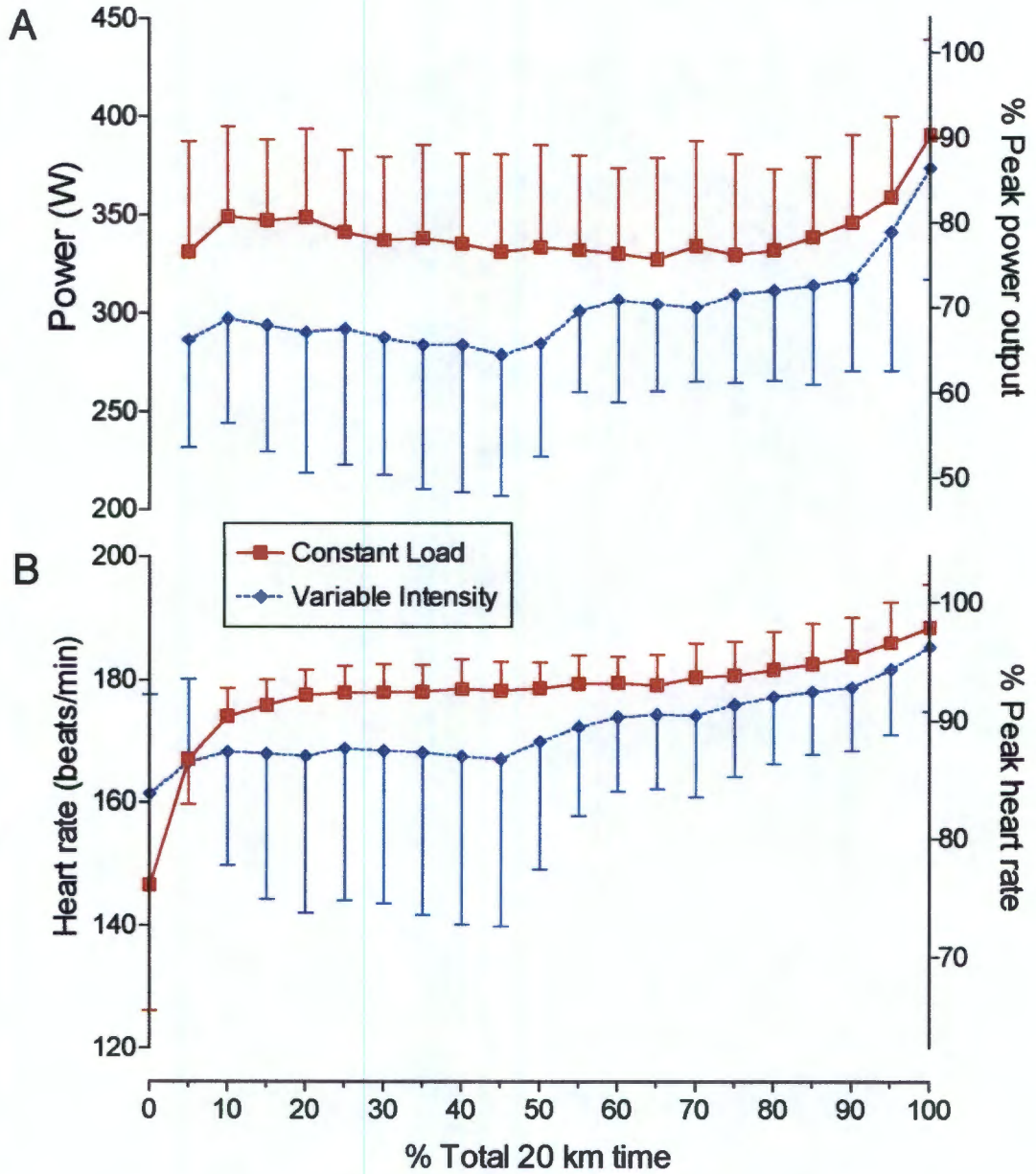
Figure 7.2 illustrates the time each subject took to complete the 20 km performance ride which followed the 150 min pre-load. The group mean time for the TT following the constant load ride was significantly faster than the times recorded following the variable intensity ride (26:32 ± 1:30 min versus 28:08 ± 1:47 respectively, $P<0.05$). All subjects improved their performance following the constant load ride by 1:36 ±1:18 min or an average of ~6%.

Figure 7.2 The time to complete the 20 km performance time trial.



As would be expected from the faster performance rides, the group mean power output (Figure 8.3A) following the constant load ride was also significantly greater than that following the variable intensity ride (340.3 ± 44.2 vs. 302.5 ± 42.3 W; 77.8 ± 10.2 vs. 70.0 ± 9.8% of PPO, $P<0.05$). The mean HR of the six subjects for each five percent section of the TT is illustrated in figure 7.3B. Despite the significantly higher power outputs associated with the constant load ride compared to the variable intensity ride, the HR recorded during the two subsequent TT were not significantly different (178 ± 5.0 vs. 172 ± 16.3 beats/min; 92.5 ± 2.6 vs. 89.4 ± 8.5% of HR_{peak}).

Figure 7.3 The group mean ± SD values for (A) power output (W) and (B) heart rate (beats/min) for each five percent section of the 20 km performance time trial.



7.4 Discussion

The primary aims of this chapter were, firstly to reproduce the responses of well-trained cyclists that had previously been determined in the field (Chapter Four), under standardised laboratory conditions. Secondly, to determine if such riding strategies would be more advantageous to subsequent cycling performance than constant load exercise. As can be seen from Figure 7.1, the first goal was achieved, as highlighted by the similar HR responses recorded during the 150 min variable intensity ride in the laboratory compared to that recorded previously during an actual competition of approximately the same duration. Such similarities indicate that the variable intensity workload employed in the current investigation closely mimicked the physiological demands of bunch, or mass start cycle racing.

Hence, the first important finding of this study was that, despite the identical mean power outputs and HR during the initial 150 min of exercise (Figure 7.1), there was a significant improvement in the time to complete the 20 km TT following the 150 min constant load ride versus the variable intensity ride. The average improvement was 1:36 ±1:18 min (5.5 ±4.6%), which was statistically significant ($P < 0.05$). Indeed, all six riders improved their TT performance after the 150 min constant load ride compared to the variable intensity effort (Figure 7.2). As might be expected, this improvement in cycling performance was also reflected in a greater mean power output following the constant load ride (340.3 ± 44.2 vs. 302.5 ± 42.3 W, $P < 0.05$; Figure 7.3A).

Of interest was the finding that both the HR and power outputs recorded for cyclists during the 20 km TT were similar to previous field and laboratory performance rides (Chapters Four, Five and Six; Foster et al. 1993a, 1993b),

suggesting that the athletes were working at approximately the same maximal constant load that they sustain for a competitive time trial event. As the present investigation was concerned solely with mimicking the stochastic responses of group cycle racing and assessing the effects of such varying intensities on performance, this study did not undertake any metabolic measurements. Hence, one can only speculate about the mechanisms concerned with reducing performance following variable intensity exercise. The suspicion is that the repeated work jumps during the variable intensity ride may have been associated with an increased muscle glycogen utilisation particularly in the type II fibres. However, muscle biopsies would be needed to confirm such a theory.

Whatever the mechanism, the results of the current investigation and that reported previously by Foster et al. (1993b) emphasise that exercise physiologists may need to consider the best way to ride a given distance (variable intensity versus constant load) in order to optimise performance. Given the importance of appropriate pacing in events lasting longer than 80 –100 s (Foster et al. 1994; van Ingen Schenau et al. 1994), there has been surprisingly little research in this area. Indeed, the advice to athletes that "even pace" is the most appropriate strategy to adopt is based largely on the results of a single investigation undertaken over 40 years ago (Robinson et al. 1958). In that study, three well trained men ran 1,200 m with three different pacing strategies: a fast start; a slow start, and; an even pace. With the fast start $\dot{V}O_2$ lactate and RPE were higher that the other two strategies. Robinson et al. (1958) concluded that the best strategy was an even pace, delaying maximal effort "as late as possible".

In conclusion, this is the first study to replicate the stochastic nature of competitive bunch cycle racing under standard laboratory conditions and assess its subsequent effects on performance. The finding that 20 km TT performance was enhanced after 150 min of steady-state exercise rather than variable intensity cycling raises the question of why riders choose not to maintain an "even-pace" strategy during *all* races.

Section B of this thesis attempts to evaluate the metabolic differences during the different forms of exercise will be undertaken in order to try and understand why constant load exercise appears to be more beneficial to performance compared to the current practices of cyclists in competitive situations.

SECTION B

METABOLIC FACTORS ASSOCIATED WITH CONSTANT AND VARIABLE LOAD CYCLING PERFORMANCE

CHAPTER EIGHT

THE EFFECTS OF CARBOHYDRATE INGESTION ON 20 KM TIME TRIAL PERFORMANCE IN WELL-TRAINED CYCLISTS

8.1 Introduction

The studies described in the first half of this thesis have determined the physiological responses of well trained cyclists to constant load (i.e. individual time trial) and variable intensity (i.e. mass start road races) exercise. The results of these studies have established that 1) all cycling races are not performed at a constant work rate but, in the case of mass-start road races, continually vary in intensity, and 2) that these conditions can be reproduced in the laboratory. In the second half of this thesis, several metabolic interventions that could potentially have a major impact on the performance of both constant-load and variable intensity cycling will be examined.

One widely accepted practice of endurance athletes is the consumption of carbohydrate during prolonged (>90 min) exercise. Indeed, carbohydrate ingestion has been shown to delay the onset of fatigue during moderate intensity (70-75 % $\dot{V}O_{2max}$), constant load cycling (for review see Coggan and Coyle 1991). Furthermore, it has recently been shown that carbohydrate ingestion can also improve the performance of high intensity time trial cycling lasting ~1 h in both moderate (El Sayed et al. 1997; Jeukendrup et al. 1997) and warm (Below et al. 1995) environmental conditions. However, whether carbohydrate ingestion improves cycling performances in events lasting ~25-30 min, in which the athlete freely selects their own pace (such as that previously described in Chapter Four) has yet to be determined.

Accordingly, using the Kingcycle ergometry system, which has been validated in the studies described in the first section of this thesis, (Chapters Four and Five) this investigation determines whether CHO ingestion might also improve cycling

time trial (TT) performance lasting <30 min compared to the consumption of plain water.

8.2 Experimental design

Fourteen endurance trained cyclists (11 males, 3 females: Table 8.1) performed a random order of two 20 km TT on the Kingcycle ergometer (described in sections 3.1.2 and 3.2.3). Pre-experimental dietary and training was control was maintained as previously described (Section 3.2.3).

Table 8.1 Subject characteristics

Subject	Mass (kg)	Height (m)	Age (yr)	Sex	PPO (W)	$\dot{V}O_{2peak}$ (l/min)	HR_{peak} (beats/min)	P/W (W/kg)
1	75.0	1.93	22	M	449	5.02	197	5.99
2	72.5	1.75	24	M	377	3.94	189	5.20
3	75.0	1.79	23	F	385	4.32	180	5.13
4	62.0	1.78	23	F	365	4.10	188	5.88
5	65.0	1.82	25	M	418	4.42	194	6.43
6	63.5	1.74	24	M	417	4.84	183	6.65
7	78.0	1.80	23	M	364	4.08	190	4.67
8	78.0	1.87	28	M	487	5.44	193	6.24
9	76.0	1.68	22	M	362	4.06	188	4.76
10	63.0	1.72	21	F	295	3.33	187	4.68
11	65.9	1.80	29	M	420	4.58	188	6.37
12	67.0	1.81	22	M	419	4.69	191	6.25
13	75.0	1.88	16	M	425	5.49	194	5.66
14	82.6	1.93	23	M	423	4.94	195	5.12
Mean	*71.3*	*1.81*	*23.2*	---	*400.4*	*4.52*	*189.8*	*5.65*
SD	*6.7*	*0.07*	*3.1*	---	*46.6*	*0.60*	*4.7*	*0.70*

P/W, power-to-body mass ratio (PPO/BM)

As previously stated, the original intention of this study was to investigate the effect of carbohydrate ingestion *during* exercise on 20 km TT performance. However, pilot work revealed that it was impractical for subjects to drink while cycling at such a high intensity as it interfered with their ability to maintain the desired workrate. More to the point, riders indicated that during actual competition they rarely, if ever, consumed any fluid during a maximal effort lasting ≤ 30 min. Accordingly, it was decided that subjects would consume test solutions immediately before exercise in one bolus feeding. Specifically, they ingested an 8 ml/kg body mass solution of either a commercial carbohydrate-electrolyte (CHO) beverage (Energade™, Bromor Foods, Salt River, Cape Town) or a placebo (PLA). The concentration of CHO beverage was 6.8 g/100 ml and contained 14.7 mEq/L of sodium and 0.5 mEq/L of potassium.. The placebo was coloured and flavoured with a sugar-free additive. Subjects ingested an average of 570 ±54 ml of the test solutions, both of which were indistinguishable in taste to the subjects. During the CHO trial, subjects ingested 39 ±4 g of carbohydrate. Precisely 10 min after finishing a drink, subjects mounted the Kingcycle ergometer and started a 5 min warm-up at a work rate of 150 W. On completion of this warm-up, subjects then undertook a 20 km performance TT on the Kingcycle ergometer.

8.3 Results

Table 8.2 shows the individual performance time, power output and HR for the 20 km TT under the two experimental conditions for all subjects. Eight subjects rode slightly faster on the placebo trial, while seven performed marginally better with CHO ingestion. However, such differences were small, and overall, there were no

significant differences in 20 km performance between the two trials (27:41 ±1:39 min:s for CHO and placebo; Figure 8.1). Similarly, both mean heart rate (171 ±6 vs. 171 ±5 beats/min; Figure 8.2, top panel) and mean power output (312 ±40 vs. 311 ±38 W for CHO and placebo, respectively; Figure 8.2, lower panel) were similar during the two rides. There was no order effect of the two experimental conditions.

Figure 8.1 The individual performance times for the 20 km time trials following the ingestion of 8 ml/kg BM of either a 6.8 g/100 ml carbohydrate-electrolyte solution, or a coloured, flavoured placebo. The horizontal lines indicate the group mean values.

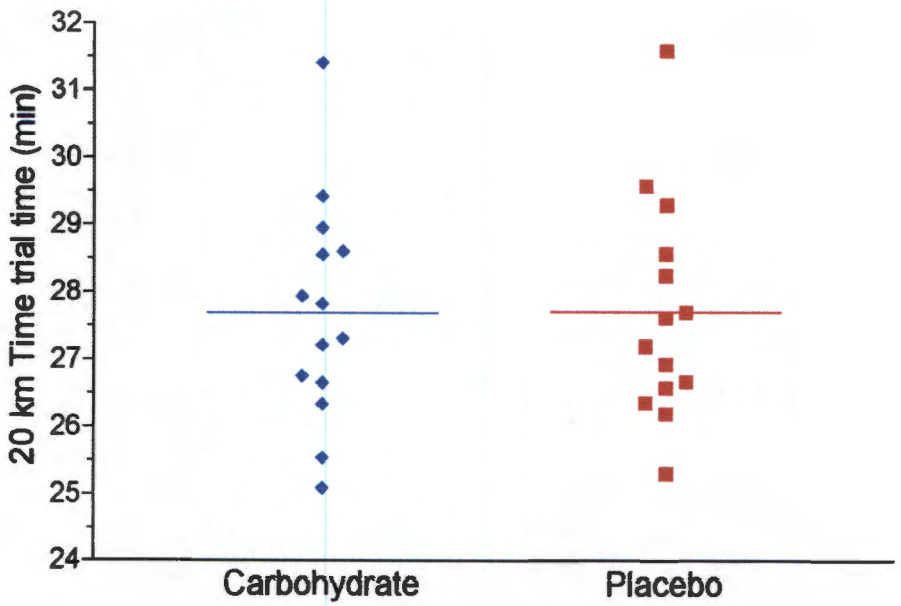
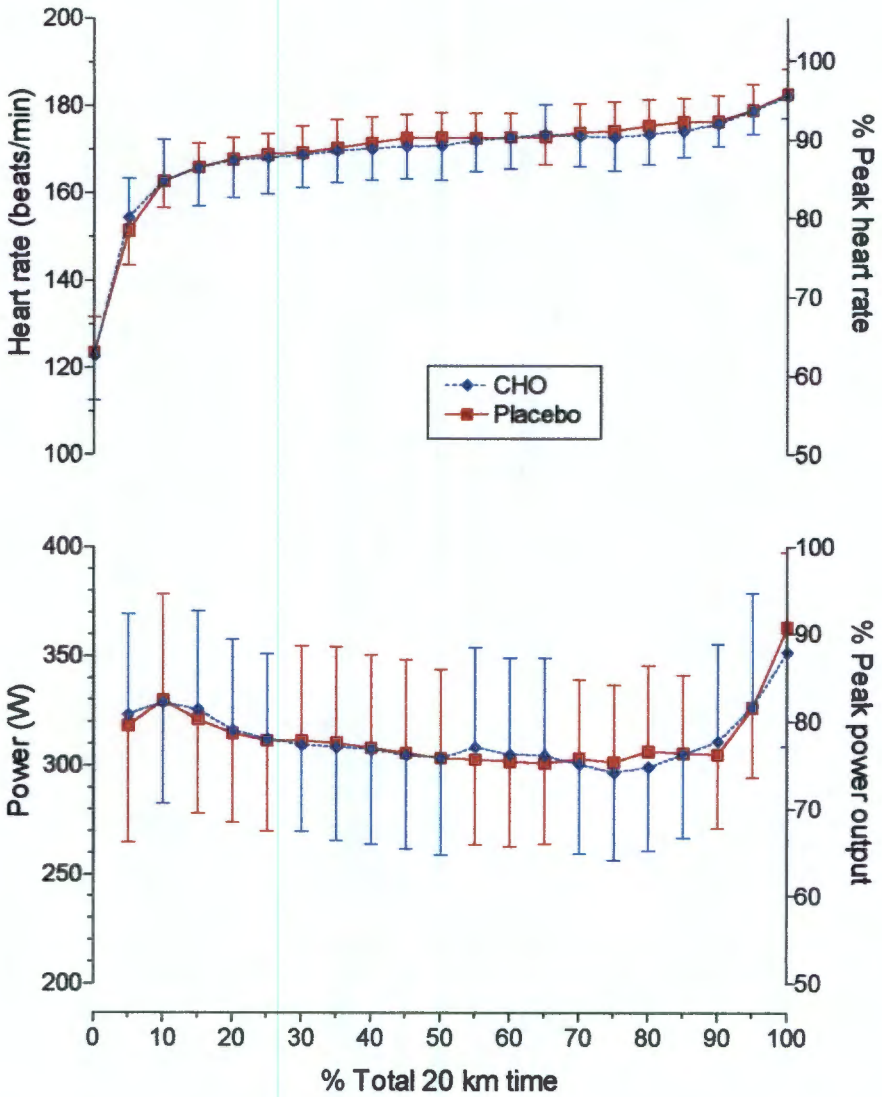


Table 8.2 Performance time (min:s), mean power output (W) and mean heart rate (beats/min) for each subject during carbohydrate (CHO) and placebo (PLA) trials.

Subject	Time (min:s)			Power (W)			Heart Rate (beats/min)		
	CHO	PLA	Δ	CHO	PLA	Δ	CHO	PLA	Δ
1	26:20	26:55	-00:35	345.5	327.8	17.7	163.4	166.3	-2.9
2	27:49	27:11	00:38	305.2	320.6	-15.4	162.9	169.7	-6.8
3	27:56	28:14	-00:18	301.6	294.4	7.1	167.2	171.1	-3.9
4	28:33	28:33	00:00	286.8	286.9	-0.1	180.9	177.7	3.2
5	26:39	26:21	00:18	335.0	342.7	-7.7	173.5	173.8	-0.4
6	27:18	27:41	-00:23	315.4	308.3	7.1	n/a	n/a	n/a
7	28:57	29:17	-00:20	279.3	273.8	5.5	n/a	n/a	n/a
8	25:05	25:18	-00:13	385.1	377.9	7.2	166.8	168.3	-1.5
9	29:25	29:34	-00:09	269.1	268.1	1.0	167.6	162.1	5.5
10	31:24	31:35	-00:11	234.5	231.9	2.6	n/a	n/a	n/a
11	26:45	26:11	00:34	332.2	348.5	-16.3	166.8	166.5	0.2
12	27:12	26:40	00:32	320.3	334.1	-13.8	169.7	173.9	-4.2
13	28:36	27:36	01:00	286.7	309.3	-22.6	178.2	174.3	3.9
14	25:32	26:34	-01:02	367.6	337.2	30.4	180.3	178.5	1.8
Mean	*27:41*	*27:41*	*00:00*	*311.7*	*311.5*	*0.2*	*170.7*	*171.1*	*-0.5*
SD	*01:39*	*01:39*	*00:33*	*40.1*	*38.1*	*14.4*	*6.5*	*5.1*	*3.8*

Δ Represents the difference between CHO and PLA trials. n/a indicates data for this subject are not available.

Figure 8.2 The absolute and relative values for heart rate (top panel) and power output (bottom panel) for each 5% segment of the 20 km performance time trials.



8.4 Discussion

The main finding of the present study was that the ingestion of ~40 g of carbohydrate 15 min prior to exercise did not improve 20 km cycle time-trial performance in 14 well-trained subjects. In contrast, El-Sayed et al. (1997) and Jeukendrup et al. (1997) have recently reported that carbohydrate ingestion

improved cycling performance lasting ~1 h. Jeukendrup et al. (1997) fed 19 subjects an 8 ml/kg bolus before and 2 ml/kg at regular intervals during an exercise task requiring subjects to complete a set amount of work as fast as possible. The ingestion of ~130 g of carbohydrate improved performance time by 2.3% compared to a placebo (58.7 vs. 60.1 min; $P<0.001$). El-Sayed et al. (1997) had 8 cyclists ingest ~40 g of carbohydrate 25 min before a 1 h simulated TT. They found that carbohydrate ingestion enabled subjects to ride at a significantly higher average power output (277 vs. 269 W; $P<0.05$) and cover a greater distance (41.5 vs. 41.0 km; $P<0.05$) compared to placebo.

Unfortunately, the precise mechanism(s) by which carbohydrate ingestion improved performance in these two studies was not elucidated. However, it has previously been suggested that the key to improved performance during sustained high-intensity exercise is the relative carbohydrate availability in the form of circulating blood glucose (Coggan and Coyle 1991). Specifically, carbohydrate ingestion maintains euglycemia and high rates of carbohydrate oxidation late in exercise. Alternatively, it may well be that carbohydrate ingestion before and during maximal exercise lasting ~1 h exerts a positive effect on the central nervous system, possibly by reducing the athletes perception of effort, thereby enabling them to tolerate higher workloads for longer periods. Unfortunately, a role for central fatigue during exercise is often accepted only by default when the investigator is unable to find support for the hypothesis of a peripheral (muscle) dysfunction (for review see Davis 1995). The fact remains that central fatigue may limit both short-term high-intensity work capacity as well as more prolonged, moderate-intensity exercise.

One hypothesised disadvantage of pre-exercise carbohydrate feedings is the elevation of plasma insulin concentrations which would be expected to suppress fat metabolism while concomitantly accelerating carbohydrate oxidation and causing a decline in plasma glucose concentration during subsequent exercise. Unfortunately, warnings to avoid carbohydrate intake in the hour before exercise have become part of sports nutrition dogma. However, a recent review of the investigations examining the effect of carbohydrate feedings during the hour immediately before exercise on subsequent performance reveals that 10 studies found a performance enhancement of between 7-20%, or no negative effect of the feedings, while only one study found that performance was impaired (for review see Hawley and Burke 1997). Although plasma glucose or insulin was not determined in the present study, no subjects complained of fatigue or manifested any symptoms of hypoglycemia.

During the current investigation it was decided to determine only performance related parameters as other ancillary measurements (i.e. blood and gas sampling) interfere with an athletes ability to concentrate during high-intensity exercise (Coyle and Hamilton 1990; Jeukendrup et al. 1997; Robinson et al. 1995). But the fact that performance times during the 20 km TT were identical after CHO and placebo ingestion supports the notion that maximal exercise lasting ~30 min is not likely to be limited by either endogenous substrate availability, or central nervous system fatigue. There is confidence in these findings since the reliability of the criterion performance task has previously been shown to be very high (Chapter Five).

Furthermore, Hawley et al. (1997) recently reported that carbohydrate-loading and the concomitant elevation in muscle glycogen stores failed to improve 1 h cycle TT

performance in well-trained subjects compared to when they consumed their normal diet. Taken collectively, these findings indicate the CHO availability per se does not limit high-intensity cycling time trial performance lasting ~30 min, such as that seen in the 16.0 km TT in Chapter Four.

CHAPTER NINE

PHYSIOLOGICAL AND METABOLIC RESPONSES TO CONSTANT AND VARIABLE LOAD CYCLING PERFORMANCE

9.1 Introduction

The results of the study described in Chapter Four highlighted the variable shifts in the frequency and amplitude of the heart-rate (HR) responses to mass-start endurance cycle races. Furthermore, Chapter Seven revealed that 20 km time-trial (TT) performance which followed 150 min of either SS or VI cycling undertaken at the same *average* power output was 6% faster after SS. In that study it was speculated that the repeated work jumps during VI may have been associated with an increased muscle glycogen utilisation compared to SS exercise. However, the absence of any metabolic measurements precluded an attempt at elucidating the mechanism(s) associated with the reduction in TT performance following VI exercise.

Accordingly, the aims of the final chapter of this thesis were, firstly, to evaluate the metabolic and hormonal responses to prolonged (140 min) cycling in well-trained men ingesting CHO throughout both SS (~70% of $\dot{V}O_{2peak}$) and VI (40 to 85% of $\dot{V}O_{2peak}$) cycling of the same *average* intensity. Secondly, it was wished to determine the effects of these two different exercise pre-loads on subsequent 20 km TT performance.

9.2 Experimental Design

Six well-trained male cyclists (Table 9.1) completed a random crossover of four experimental trials that were separated by exactly 7 d, and conducted at the same time of day. Pre-trial control was ensured by providing subjects with pre-prepared food (as described in 3.2.3) that matched their habitual energy intake and

composition (Table 9.2), and by monitoring pre-trial HR and training (3.2.3). Further, subjects reported to the laboratory 3 h after a standardised breakfast for each ride.

Table 9.1 Subject characteristics

Subject	Age (yr)	Mass (kg)	Height (m)	PPO (W)	$\dot{V}O_{2peak}$ (L/min)	HR_{peak} (beats/min)
1	17	65	1.81	363	4.07	196
2	26	79	1.76	347	3.90	182
3	19	60	1.78	387	4.34	198
4	41	90	1.78	457	5.06	197
5	19	82	1.81	449	5.01	195
6	25	70	1.81	402	4.52	193
Mean	24.5	74.3	1.79	401	4.48	193
SD	8.8	11.3	0.02	44.7	0.48	5.89

Table 9.2 Energy intake, and breakdown of subject's habitual diets

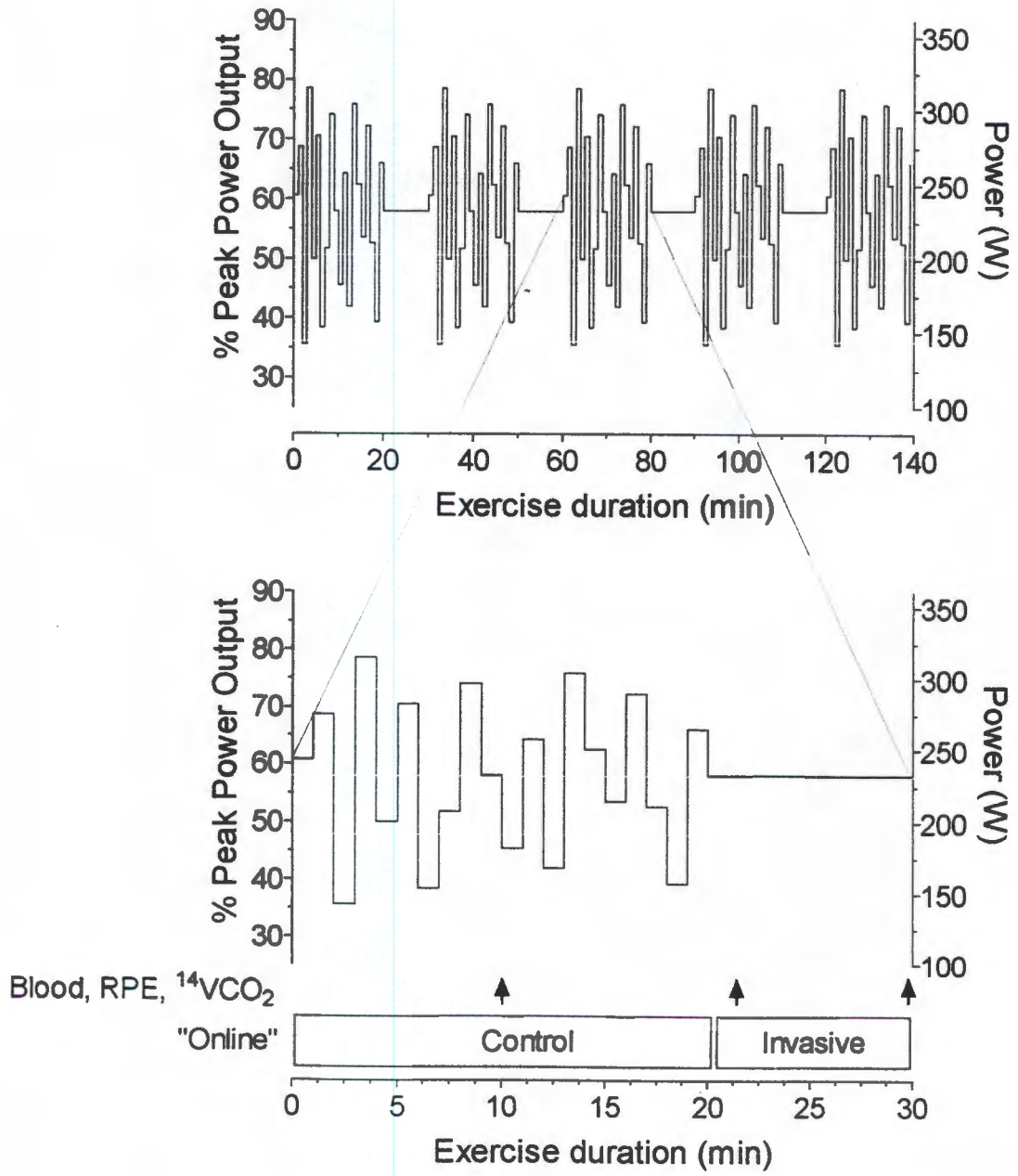
Subject	Energy (kJ)	CHO (g)	CHO (%)	Protein (g)	Protein (%)	Fat (g)	Fat (%)
1	18 769	548	52	162	14	184	37
2	11 176	296	47	148	22	99	33
3	14 769	443	54	139	16	127	32
4	16 038	369	40	131	14	193	45
5	12 222	433	64	98	13	91	28
6	10 653	297	47	119	19	81	29
Mean	13 938	398	50.7	133	16.3	129	34.0
SD	3 156	97	8.1	22	3.5	49	6.3

On arrival at the laboratory, the subject's cycle was placed on the Kingcycle ergometer, and the calibration process was conducted (as previously described in detail in section 3.1.2). Immediately prior to each experimental trial subjects ingested 4 ml/kg BM of a 5 g/100 ml carbohydrate (CHO) solution and then underwent a five min incremental warm-up on the Lode ergometer (3.1.1). The warm-up commenced at an intensity of 29% of PPO (~116 W) and was increased at a rate of ~6% of PPO (~24 W) every min until the desired intensity for that trial was reached.

Figure 9.1 shows a schematic of the protocols with the exercise intensity represented as a percentage of PPO, and as a mean power output (W). The first 140 min of each ride consisted of either a constant load work (SS), or VI (experimental). During VI subjects rode five 20 min bouts of variable intensity exercise interspersed with 4 x 10 min periods of work at a constant power output (58% of PPO). The average work rate during each 20 min period was 58 ±13.3% of PPO with a range in power of between 35 and 77% of PPO (~40 and 85% of $\dot{V}O_{2peak}$, Figure 9.1). By design, the mean power output throughout the two rides, as calculated by the area under the power versus time curve for each subject, was the same: 58 ±11% PPO or 232 ±44 W. Such a range in VI was chosen because it was similar to that previously observed in the field during mass start road races (Chapter Four), and the same as a previous study that examined the effects of stochastic exercise on subsequent exercise performance (Chapter Seven).

As this investigation aimed to determine both the metabolic and subsequent performance responses to 140 min of SS and VI, two of the 140 min experimental rides (control) were followed by a 20 km "performance" TT undertaken on the Kingcycle ergometer, which commenced exactly 60 s after completion of the 140 min experimental control exercise bouts. These TT were conducted as previously described (3.1.2 and 3.2.3). During the other SS and VI 140 min experimental trials all invasive metabolic data was collected.

Figure 9.1 A schematic of the protocols employed during the experimental trials. The open boxes denote the period during the control and invasive trials when "on-line" gas analysis was undertaken. The arrows indicate the collection of blood, the subjects ratings of perceived exertion (RPE) and $^{14}\dot{V}CO_2$ during EXP. See experimental design (9.2) for details.



During the two control experimental trials, subjects breathed for five 20 min periods through the previously described gas analysis system (3.3.1). The gas collection periods during these two control experimental rides were between 0-20, 30-50, 60-80, 90-110 and 120-140 min. During the two invasive experimental rides expired gas was collected for 10 min periods after 20, 50, 80, and 110 min of the ride. Using such a design, subjects were 'on-line' for an entire (although not the same) 140 min VI or SS experimental ride (Figure 9.1). Immediately before all experimental trials, subjects ingested 4 ml/kg BM of a 5 g/100 ml CHO solution. During the two invasive experimental rides the CHO solution contained 18μCi ^{14}C labelled glucose for the subsequent determination of the rates of plasma glucose oxidation. After the first 15 min of each 140 min invasive experimental ride and at subsequent 15 min intervals thereafter, subjects ingested a 5 g/100 ml of the same solution at a rate of 10 ml/kg BM/min. During the 140 min control rides, the CHO beverage was consumed in the 10 min period between gas collections. Throughout the TT subjects had access to water *ad libitum*. $^{14}\dot{V}CO_2$ was collected for 2-3 min periods after 20, 50, 80, and 110 min during the invasive experimental rides by having subjects breathe through a Hans Rudolph one way value to fill a 2 L anaesthetic bag with expired air (section 3.3.4) for the subsequent determination of the rates of plasma glucose oxidation.

Prior to the two invasive experimental rides subjects rested in a supine position and a muscle biopsy was taken from the vastus lateralis muscle (described in detail in section 3.3.5). At the same time an incision was made in the contralateral leg for a post exercise biopsy, while a Jelco 18 gauge cannula was inserted in a forearm vein for blood sampling. All post exercise muscle biopsy samples were collected within 60-120 s of completion of the 140 min invasive rides. Samples

were subsequently analysed for total muscle glycogen (section 3.3.6) while histochemical analysis was also undertaken (section 3.3.7). No TT was performed following the SS and VI invasive trials.

Ratings of perceived exertion (RPE) were recorded (see 3.3.8), and venous blood samples (20 ml) drawn at min 10, 21, 30 and after each 10 min period thereafter during the 140 min invasive experimental rides for subsequent analysis (described previously in 3.3.3 and 3.3.4).

9.3 RESULTS

9.3.1 Oxygen uptake, HR, RPE, rates of substrate oxidation, and energy expenditure

Table 9.3 displays the $\dot{V}O_2$, HR and RPE, whilst Table 9.4 displays the rates of CHO and fat oxidation, averaged for each successive 10 min time period during the two 140 min experimental rides. During SS $\dot{V}O_2$ remained relatively constant throughout the 140 min of exercise at ~3.0 L/min. Despite the five bouts of stochastic work during VI which totalled 100 of the 140 min of exercise (~70%), $\dot{V}O_2$ also remained steady at ~3.1 L/min and was only significantly higher than SS between 111-120 min (3.22 ±0.39 vs 3.13 ±0.38 L/min, $P<0.05$). There was a gradual drift in HR during both trials so that during the last 10 min of exercise, HR for both SS and VI were ~25 beats/min higher than after the first 10 min (144 ±7 to 167 ±7 and 145 ±6 to 169 ±13 beats/min for SS and VI respectively, $P<0.001$). However, there were no differences in HR between the two experimental conditions at any time point.

RPE (Table 9.3) rose progressively from 9.3 ±2.0 and 9.7 ±1.9 units after the first 10 min to 13.0 ±2.2 and 14.0 ±1.7 units during the last 10 min of exercise for SS and VI respectively ($P<0.05$). However, there were no differences in RPE between the two experimental conditions at any time point during exercise, nor a difference in the average RPE throughout the entire 140 min bout (12.6 ±1.7 vs 12.6 ±1.5 for SS and VI respectively).

As would be expected with the higher workrate, CHO oxidation was significantly elevated during the initial 10 min of VI compared to SS (3.42 ±0.84 vs 2.89 ±0.60 g/min, $P=0.03$). CHO oxidation was still higher during the second bout of VI exercise (from 31-40 min) compared to SS (3.43 ±0.63 vs 2.94 ±0.62 g/min, $P=0.007$), but thereafter, there were no differences between the two experimental conditions. Accordingly, the average rate of CHO oxidation for the entire 140 min of exercise for SS and VI were similar (2.90 ±0.09 vs 3.08 ±0.26 g/min).

Accompanying the accelerated glycogenolysis during the early stages of exercise, there was a concomitant reduction in the rate of fat oxidation during the first portion of VI (0.28 ±0.11 vs 0.41 ±0.19 g/min, $P<0.05$). Thereafter, fat oxidation between the two trials was not significantly different, averaging 0.46 ±0.05 and 0.42 ±0.10 g/min for SS and VI respectively. The overall rate of total energy expenditure for the two experimental conditions was also similar (SS 901 ±98, VI 904 ±142 J/kg/min).

Table 9.3 Oxygen uptake, heart rate and ratings of perceived exertion during 140 min of constant load (SS) or variable-intensity (VI) cycling.

	Time (min)													
	0-10	11-20	21-30	31-40	41-50	51-60	61-70	71-80	81-90	91-100	101-110	111-120	121-130	131-140
	Oxygen uptake (L/min)													
SS	2.97 ±0.24	3.00 ±0.25	3.04 ±0.27	3.05 ±0.27	3.04 ±0.25	3.07 ±0.33	3.07 ±0.30	3.11 ±0.33	3.10 ±0.37	3.12 ±0.36	3.14 ±0.36	3.13 ±0.38	3.16 ±0.39	3.15 ±0.39
VI	3.06 ±0.33	3.08 ±0.35	2.99 ±0.25	3.11 ±0.30	3.11 ±0.34	3.08 ±0.26	3.19 ±0.37	3.16 ±0.40	3.19 ±0.33	3.24 ±0.39	3.19 ±0.40	3.22 ±0.39*	3.23 ±0.36	3.21 ±0.35
	Heart rate (beats/min)													
SS	144 ±7	150 ±9	154 ±10	156 ±10	158 ±9	160 ±9	161 ±10	160 ±10	163 ±11	163 ±10	165 ±10	167 ±10	168 ±10	167 ±7
VI	145 ±6	149 ±9	154 ±9	156 ±9	155 ±9	158 ±10	158 ±9	158 ±10	162 ±11	163 ±10	163 ±11	166 ±12	168 ±11	169 ±13
	Rating of Perceived Exertion													
SS	9.3 ±2.0	10.3 ±1.5	10.7 ±1.2	11.0 ±0.9	11.5 ±0.5	12.3 ±0.5	12.8 ±0.8	13.0 ±1.3	14.0 ±1.1	13.7 ±1.9	14.2 ±1.7	15.3 ±2.3	15.0 ±2.2	13.0 ±2.2
VI	9.7 ±1.9	10.2 ±1.8	11.0 ±1.8	11.5 ±1.6	11.5 ±1.4	12.3 ±1.2	12.7 ±1.4	12.7 ±1.8	13.3 ±1.0	13.7 ±1.9	13.8 ±1.5	14.7 ±1.9	15.0 ±2.2	14.0 ±1.7

Values are mean ±SEM of n=6 subjects. *VI significantly greater than SS, $P < 0.01$; VI significantly greater than SS, $^{+}$ $P < 0.05$

Table 9.4 The calculated rates of substrate oxidation during 140 min of constant load (SS) or variable-intensity (VI) cycling.

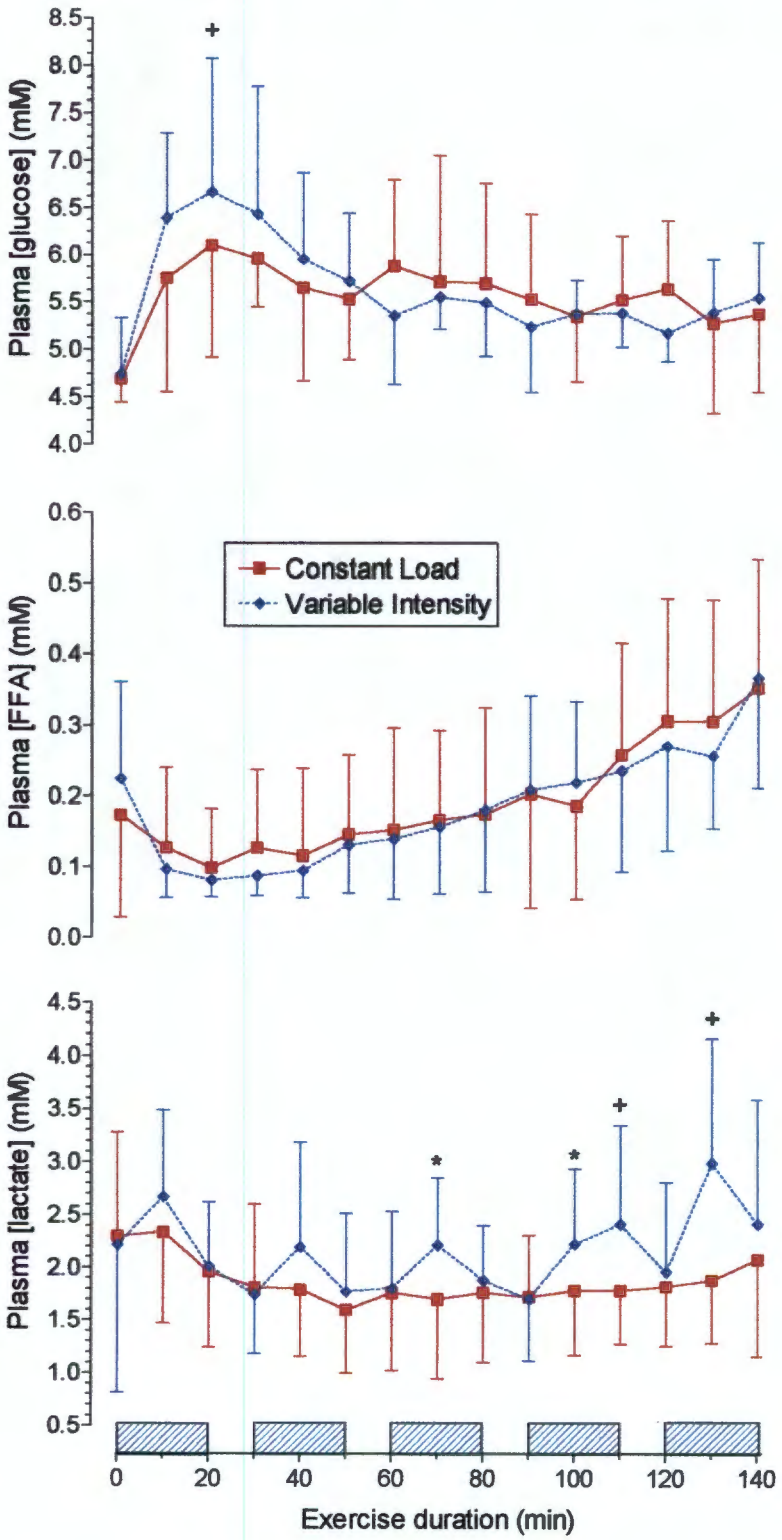
	Time (min)													
	0-10	11-20	21-30	31-40	41-50	51-60	61-70	71-80	81-90	91-100	101-110	111-120	121-130	131-140
	Carbohydrate oxidation (g/min)													
SS	2.89	2.98	3.08	2.94	2.90	3.00	2.93	2.94	2.85	2.89	2.86	2.82	2.79	2.74
	±0.60	±0.59	±0.43	±0.62	±0.58	±0.55	±0.60	±0.62	±0.45	±0.63	±0.61	±0.44	±0.65	±0.55
VI	3.42	3.39	2.67	3.43	3.29	2.92	3.27	3.17	2.86	3.17	2.98	2.70	2.94	2.93
	±0.84^{+}	±0.68	±0.40	±0.63*	±0.55	±0.34	±0.38	±0.45	±0.26	±0.45	±0.48	±0.29	±0.41	±0.52
	Fat oxidation (g/min)													
SS	0.41	0.41	0.37	0.43	0.44	0.42	0.44	0.46	0.49	0.48	0.51	0.51	0.54	0.55
	±0.15	±0.19	±0.16	±0.20	±0.20	±0.17	±0.19	±0.19	±0.16	±0.20	±0.20	±0.15	±0.19	±0.18
VI	0.28	0.28	0.39	0.31	0.34	0.45	0.38	0.40	0.53	0.44	0.49	0.60	0.52	0.51
	±0.14^{+}	±0.11	±0.07	±0.10	±0.08	±0.06	±0.08	±0.06	±0.08	±0.10	±0.09	±0.11	±0.09	±0.09

Values are mean ±SEM of n=6 subjects. *VI significantly greater than SS, $P < 0.01$; VI significantly greater than SS, $^{+}$ $P < 0.05$

9.3.2 *Circulating metabolites*

Figure 9.2 shows the plasma glucose, FFA and lactate concentrations during the two experimental conditions. Resting plasma glucose concentrations were the same for SS and VI (4.7 ± 0.2 vs 4.7 ± 0.6 mM; Figure 9.2, top panel). With the ingestion of carbohydrate plasma glucose concentration rose progressively and after 20 min of exercise it was significantly higher in VI than SS (6.7 ±1.4 vs 6.1 ±1.2 mM, $P<0.05$). From 20 to 60 min of exercise, plasma glucose concentration declined in to 5.3 ±0.7 mmol/L in VI, although euglycemia (>5 mM) was well maintained throughout the entire 140 min ride (5.7 ±0.5 mM). During SS, blood glucose concentration averaged 5.6 ±0.2 mM and was relatively constant for the whole exercise bout (Figure 9.2, top panel). Plasma FFA concentrations were similar before exercise (0.18 ±0.17 vs 0.22 ±0.14 mM) and rose progressively throughout both trials so that by the end of 140 min they had reached ~0.35 mM for both VI and SS (Figure 9.2, middle panel). As might be expected during SS, plasma lactate concentration remained relatively stable averaging 1.8 ±0.2 mM for the entire ride (Figure 9.2, lower panel). On the other hand, plasma lactate concentration during VI mirrored the changes in exercise intensity: with the work jumps, lactate concentration increased by ~1 mM from ~1.6 to 2.5 mM. After the first hour of exercise, lactate concentration in VI rose progressively, and was significantly higher than SS after 70, 100 and 110 min, reaching a peak of 3.0 ±1.2 mM after 130 min (all $P<0.05$, Figure 9.2 lower panel). The area under the lactate vs time curves was significantly greater for VI compared to SS (29.1 ±9.6 vs 24.6 ±9.0 mM/140 min; $P=0.03$).

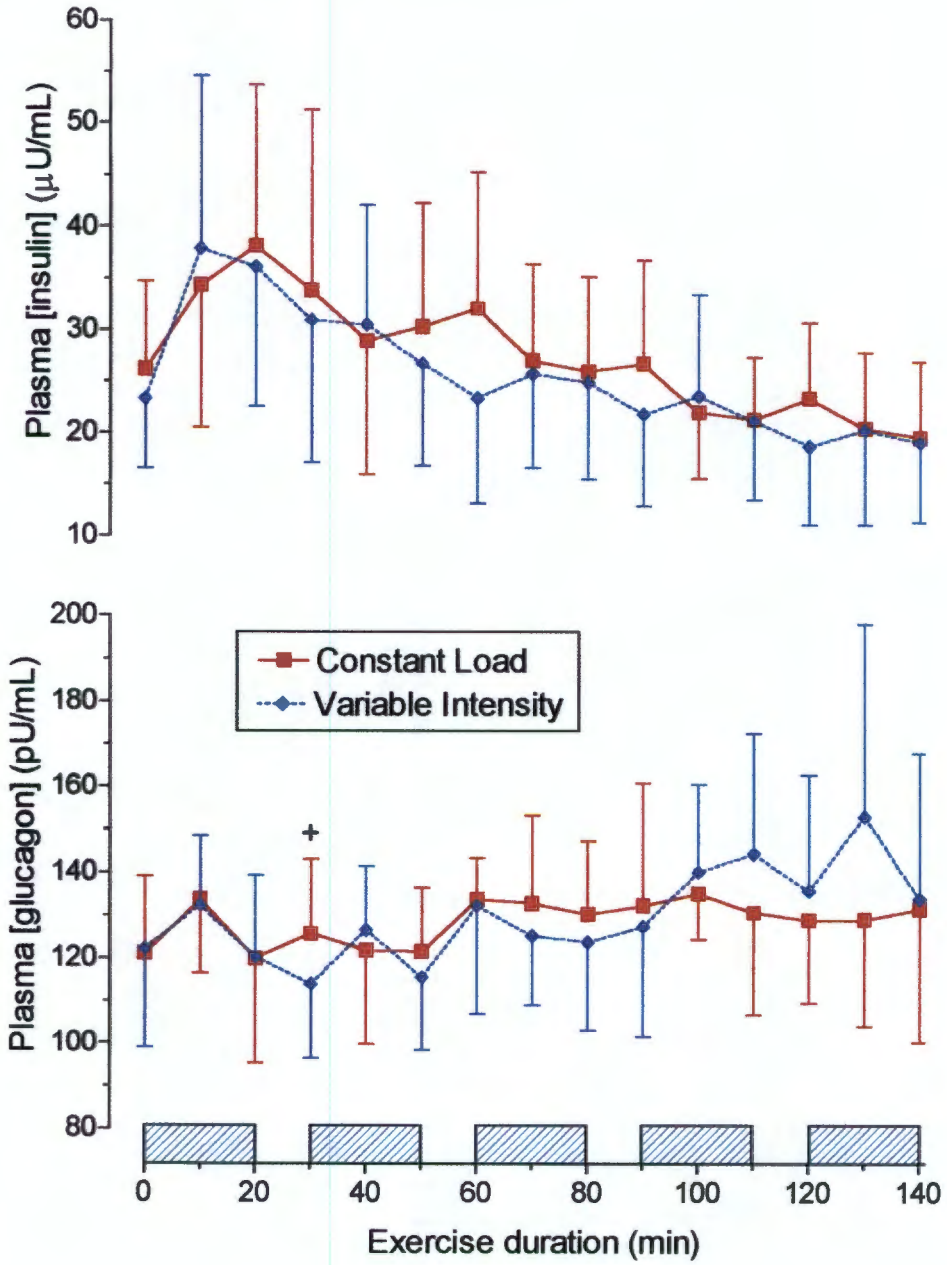
Figure 9.2 Plasma glucose (upper panel), free fatty acid (FFA, middle panel) and lactate concentrations (lower panel) during 140 min of either steady-state or variable intensity exercise. The periods of variable intensity exercise are shown by the shaded boxes. * Variable intensity significantly greater than steady-state, P<0.001; + Variable intensity significantly greater than steady-state, P<0.05



9.3.3 *Hormonal responses*

Figure 9.3 shows the concentrations of the circulating hormones, insulin and glucagon, in response to the two different experimental trials. Plasma insulin concentrations were similar at rest for the two experimental conditions (26 ±9 vs 23 ±7 μU/ml for SS and VI), rose to between 35-40 μU/ml after 30 min of exercise, and then declined progressively throughout the remainder of the work bout so that by the end of 140 min of either VI or SS they were ~20 μU/ml (Figure 9.3, upper panel). Plasma glucagon concentrations were the same at rest for the two trials (121 ±18 vs 122 ±23 pU/ml for SS and VI respectively) and apart from the 30 min time-point (SS, 126 ±17; VI, 114 ±17 pU/ml, P=0.02), were not significantly different between treatments (average 128 ±5 vs 130 ±11 pU/ml for SS and VI). There were no statistically significant differences in the area under the plasma insulin or plasma glucagon curves (368 ±141 vs 351 ±130 μU/ml/140 min and 1724 ±298 vs 1754 ±264 pU/ml/140 min for SS and VI respectively).

Figure 9.3 Plasma insulin (upper panel) and glucagon concentrations (lower panel) during 140 min of either steady-state or variable intensity exercise. The periods of variable intensity exercise are shown by the shaded boxes. + Variable intensity significantly greater than steady-state, $P<0.05$



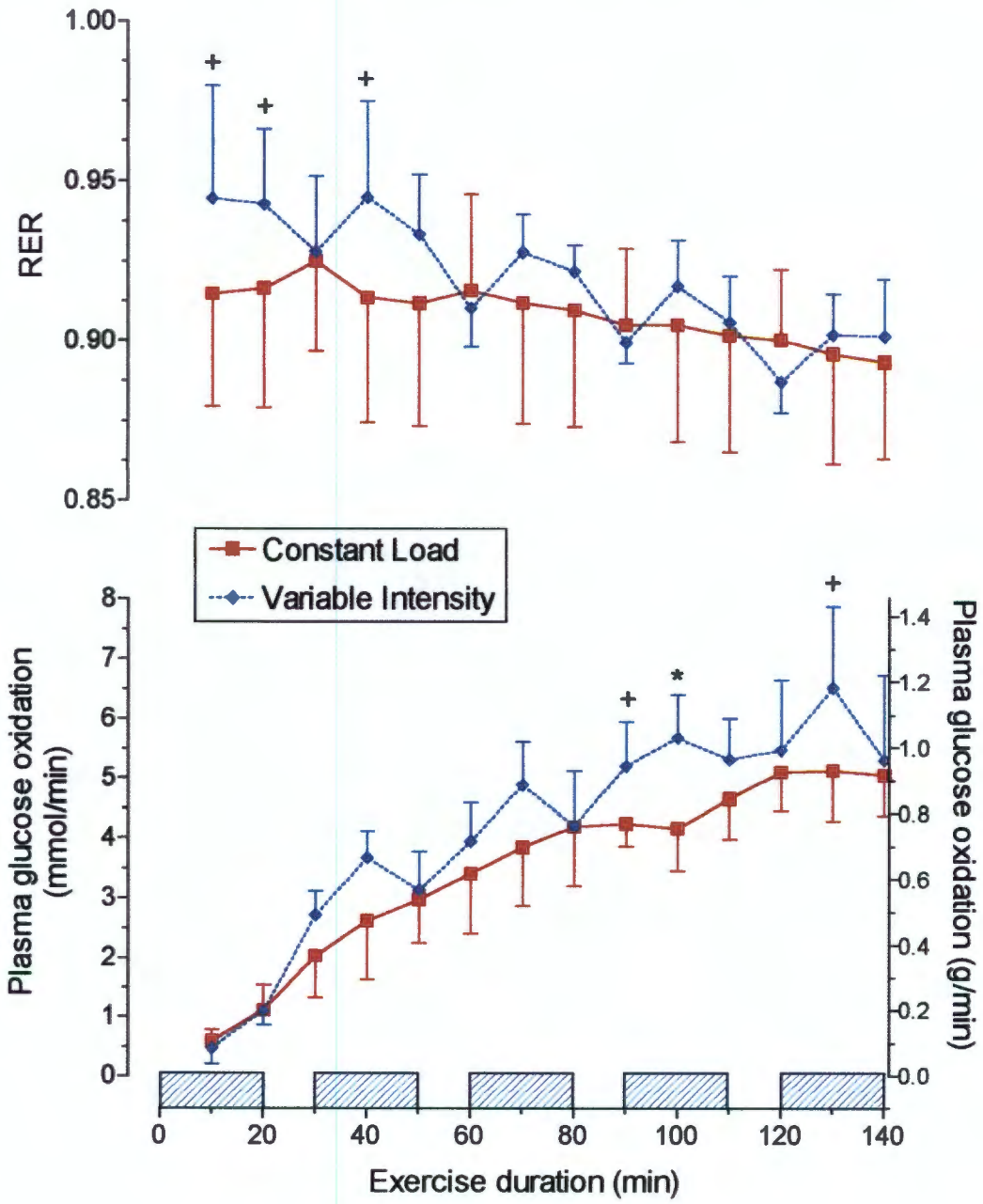
9.3.4 *Rates of plasma glucose oxidation*

The rates of plasma glucose oxidation and the RER values for the two experimental conditions are displayed in Figure 9.4. During the first 30 min of the ride, the initial work jumps during VI were associated with a significantly ($P < 0.05$) elevated RER (0.94 vs 0.91, 0.94 vs 0.92, and 0.94 vs 0.91 for VI and SS at 10, 20 and 40 min respectively). However, after the initial 40 min, RER declined progressively throughout both rides from 0.93 to 0.90 and from 0.91 to 0.89 for VI and SS respectively.

During SS the rate of plasma glucose oxidation rose progressively throughout exercise from 0.58 ±0.19 mmol/min (0.10 ±0.03 g/min) at 10 min and peaked at 5.11 ±0.85 mmol/min (0.93 ±0.15 g/min) after 130 min of the workbout. Rates of plasma glucose oxidation also rose over time during VI, with such increases being directly related to the changes in exercise intensity, particularly during the latter stages of the ride. The rate of plasma glucose oxidation was significantly higher in VI than in SS at 90 (5.12 ±0.75 vs 4.23 ±0.38 mmol/min; 0.95 ±0.14 vs 0.77 ±0.07 g/min, P=0.03), 100 (5.67 ±0.72 vs 4.15 ±0.71 mmol/min; 1.03 ±0.13 vs 0.75 ±0.13 g/min, P= 0.005) and after 130 min (6.50 ±1.35 vs 5.11 ±0.85 mmol/min; 1.19 ±0.25 vs 0.93 ±0.15 g/min) of exercise (Figure 9.4).

The average rate of plasma glucose oxidation was 0.73 ±0.33 vs 0.61 ±0.26 g/min for VI and SS, respectively. The total plasma glucose oxidised during the entire exercise bout (as calculated from the area under each subjects curve) was greater in VI than SS (99.2 ±13.1 vs 83.9 ±12.7 g/140 min, $P<0.05$).

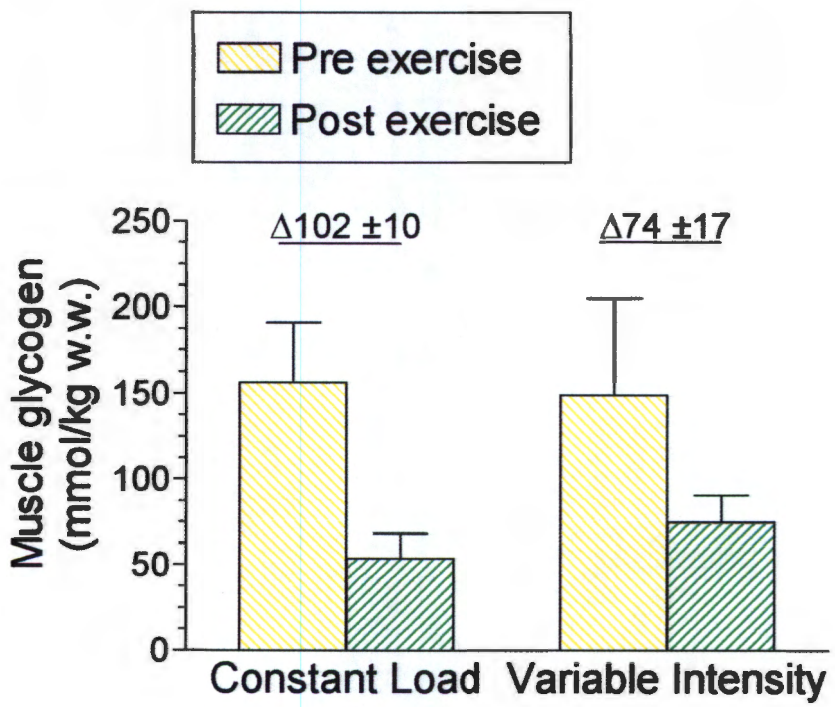
Figure 9.4. Respiratory Exchange Ratio (RER, upper panel) and the rates of plasma glucose oxidation (lower panel) during 140 min of either steady-state or variable intensity exercise. The periods of variable intensity exercise are shown by the shaded boxes. * Variable intensity significantly greater than steady-state, P<0.001; + Variable intensity significantly greater than steady-state, P<0.05



9.3.5 *Muscle glycogen utilisation*

The muscle glycogen concentration of the vastus lateralis muscle before and after 140 min of either SS or VI exercise are shown in Figure 9.5. As intended, muscle glycogen content did not differ between SS or VI before exercise (156 ±34 vs 148 ±56 mmol/kg w.w.). Neither were there any differences in glycogen content after 140 min of exercise (54 ±35 vs 75 ±16 mmol/kg w.w. for SS and VI respectively). Although glycogen utilisation was reduced by 27% during VI compared to SS, this was not statistically significant (VI 74 ±42, SS 102 ±24 mmol/kg/w.w.).

Figure 9.5 Muscle glycogen concentration in the vastus lateralis before and after 140 min of either steady-state or variable intensity exercise.



9.3.6 *Muscle fibre type, and PAS staining*

The average muscle fibre composition of the vastus lateralis muscle of the subjects was 53.6 ±2.9% type I and 46.4 ±2.9% type II fibres.

Figure 9.8 shows the percentage of fibres stained for glycogen with periodic acid-Schiffs reagent after 140 min of either SS or VI exercise. All muscle fibres stained dark (4-5) for glycogen before exercise. However, there was a marked disappearance of glycogen from the type I fibres (as indicated by a low grey scale score) after SS compared to VI (98% vs 59% of fibres scoring 0-2 for SS and VI respectively). Conversely, the density of type I fibres darkly stained (3-5) at the end of 140 min of exercise was only 2% after SS versus ~40% following VI (Figure 9.8). The number of type II fibres showing a negative grey scale score (0-1) was ten fold higher after VI compared to SS (Figure 9.8). However, for both SS and VI, approximately two thirds of type II fibres stained darkly for glycogen (3-5) at the end of the 140 min pre-load exercise bout.

Figure 9.6 A typical section of muscle stained for PAS, pre-trial (A), post 140 min of constant load exercise (B), and (C) the corresponding slide stained for ATP-ase at pH 4.3.

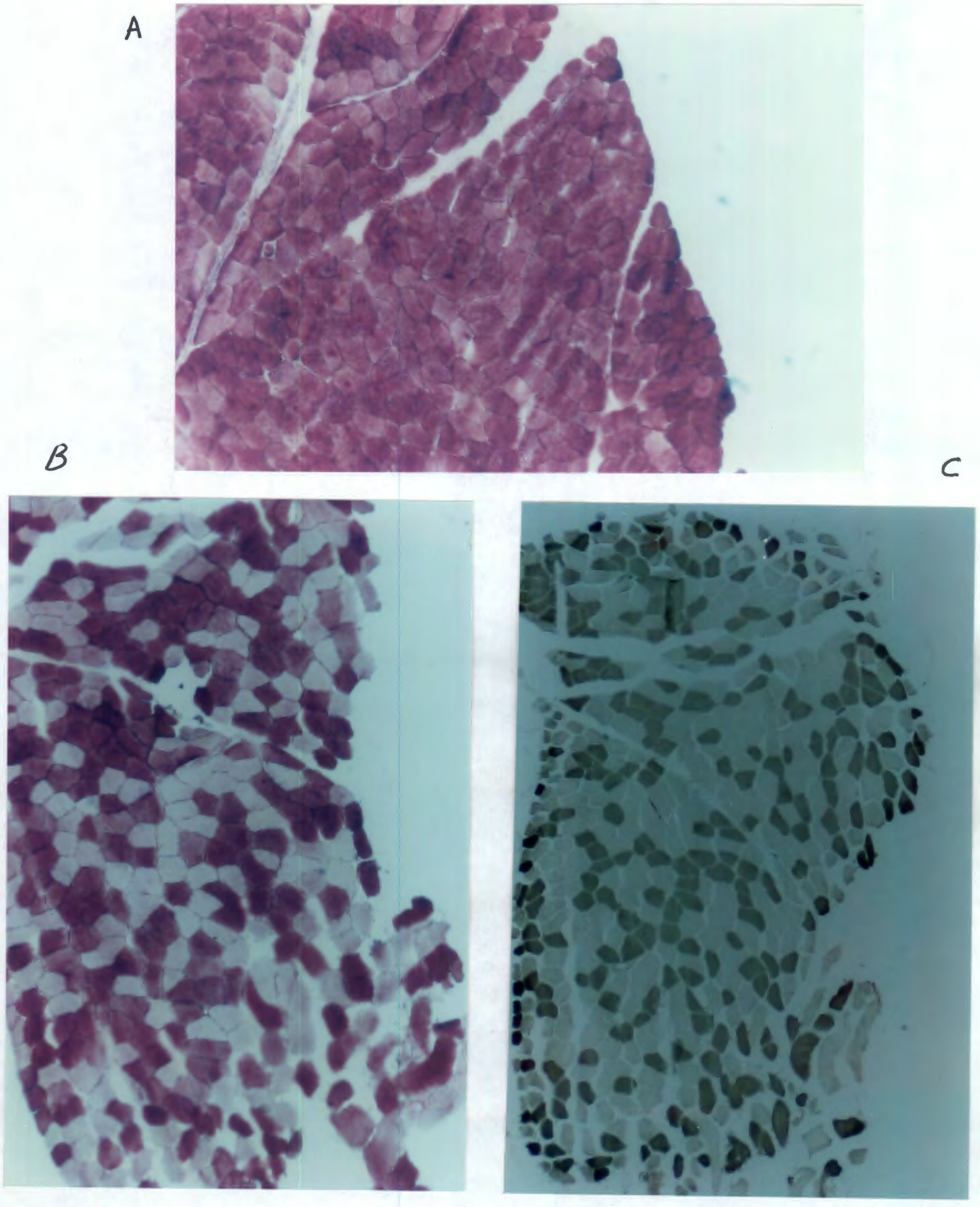


Figure 9.7 A typical section of muscle stained for PAS, pre-trial (A), post 140 min of variable intensity exercise (B), and (C) the corresponding slide stained for ATP-ase at pH 4.3.

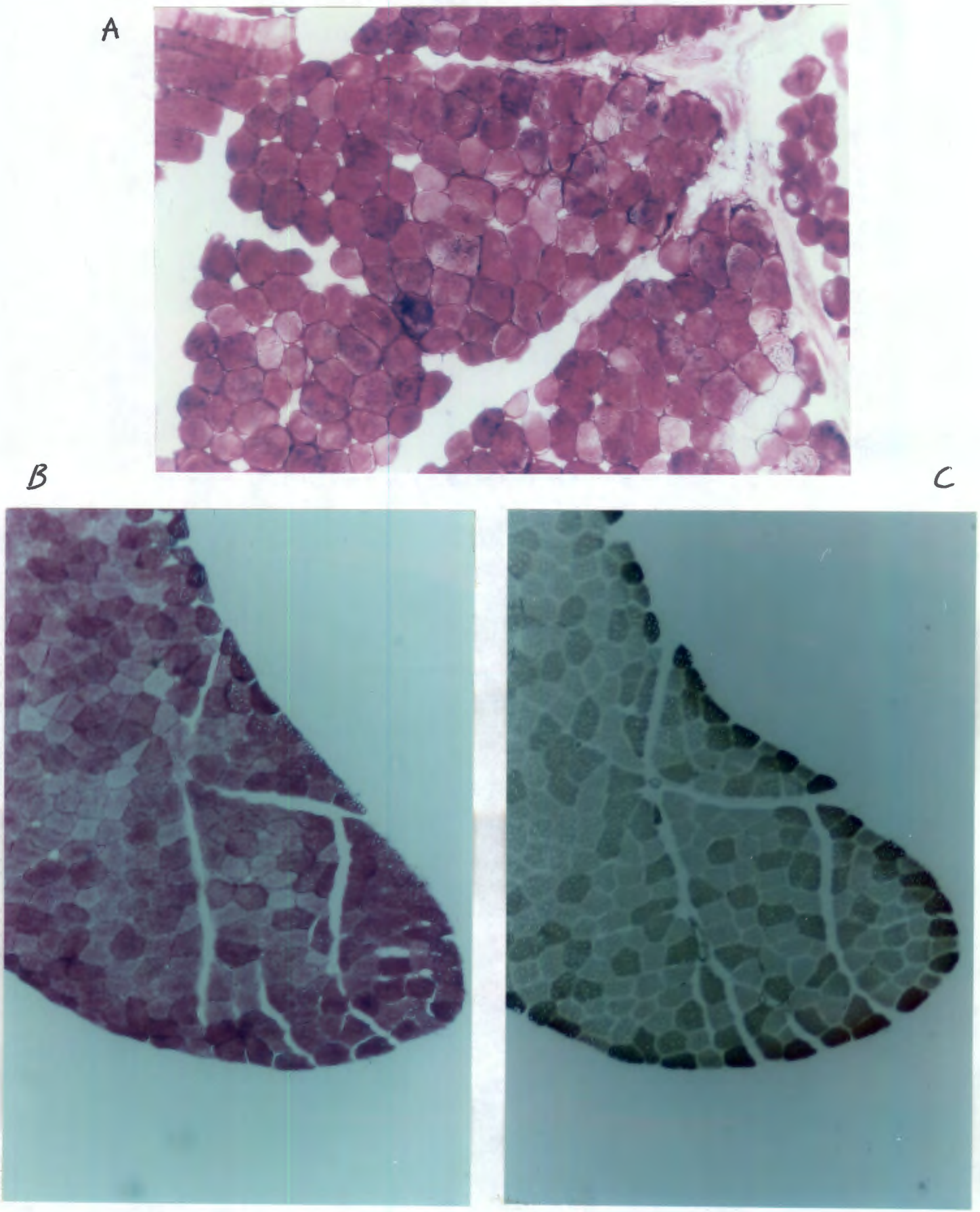
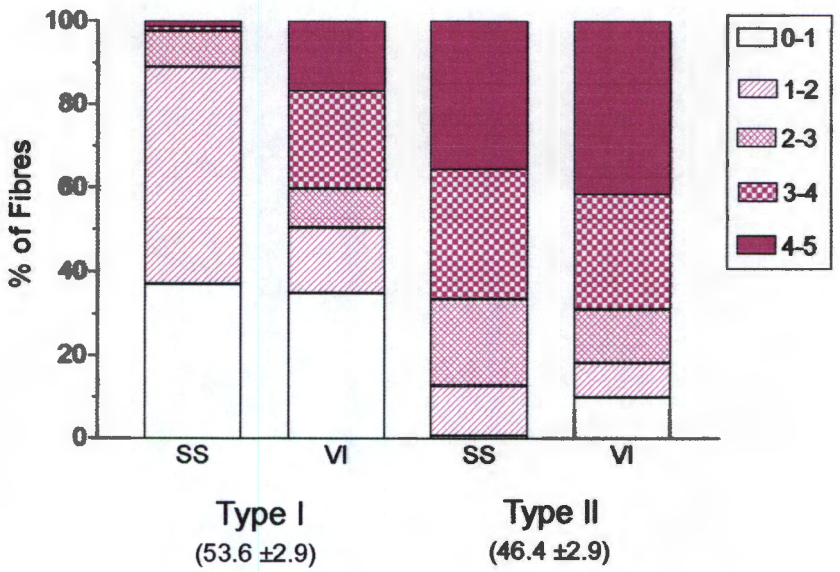


Figure 9.8 The post trial intensity of glycogen staining of the muscle biopsy samples obtained from the vastus lateralis after 140 min of either constant-load or variable-intensity cycling and stained for glycogen content using periodic acid-Schiffs reagent. The pattern of staining is displayed for both type I and type II muscle fibres, and was automatically rated on a grey scale by computer with 0-1 (negative) to 4-5 (darkly stained).



9.3.7 *Time-trial performance*

Figure 9.9 shows the individual performances in the 20 km TT. Despite the intended differences in the 140 min pre-load exercise bout, the subsequent 20 km TT performances were not significantly different (29:08 ±2:15 vs 30:30 ±2:14 min:s for SS and VI respectively). As would be expected with similar performance times, there were no differences between average power output between trials (283 ±49 vs 256 ±42 W for SS and VI respectively, Figure 9.10A). Figure 9.10B also shows

that the mean HR responses were similar between the two rides (171 ±9 vs 172 ±10 beats/min for SS and VI respectively).

Figure 9.9 Individual performance times recorded in the 20 km time trial following either 140 min of constant or variable intensity exercise. Individual subjects are linked. The horizontal line denotes the mean performance time for the group (n=6).

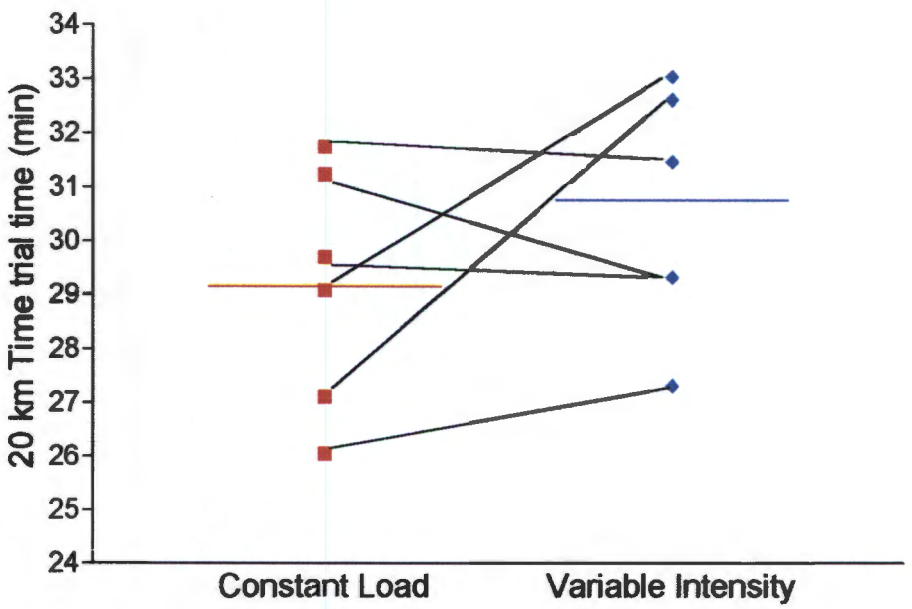
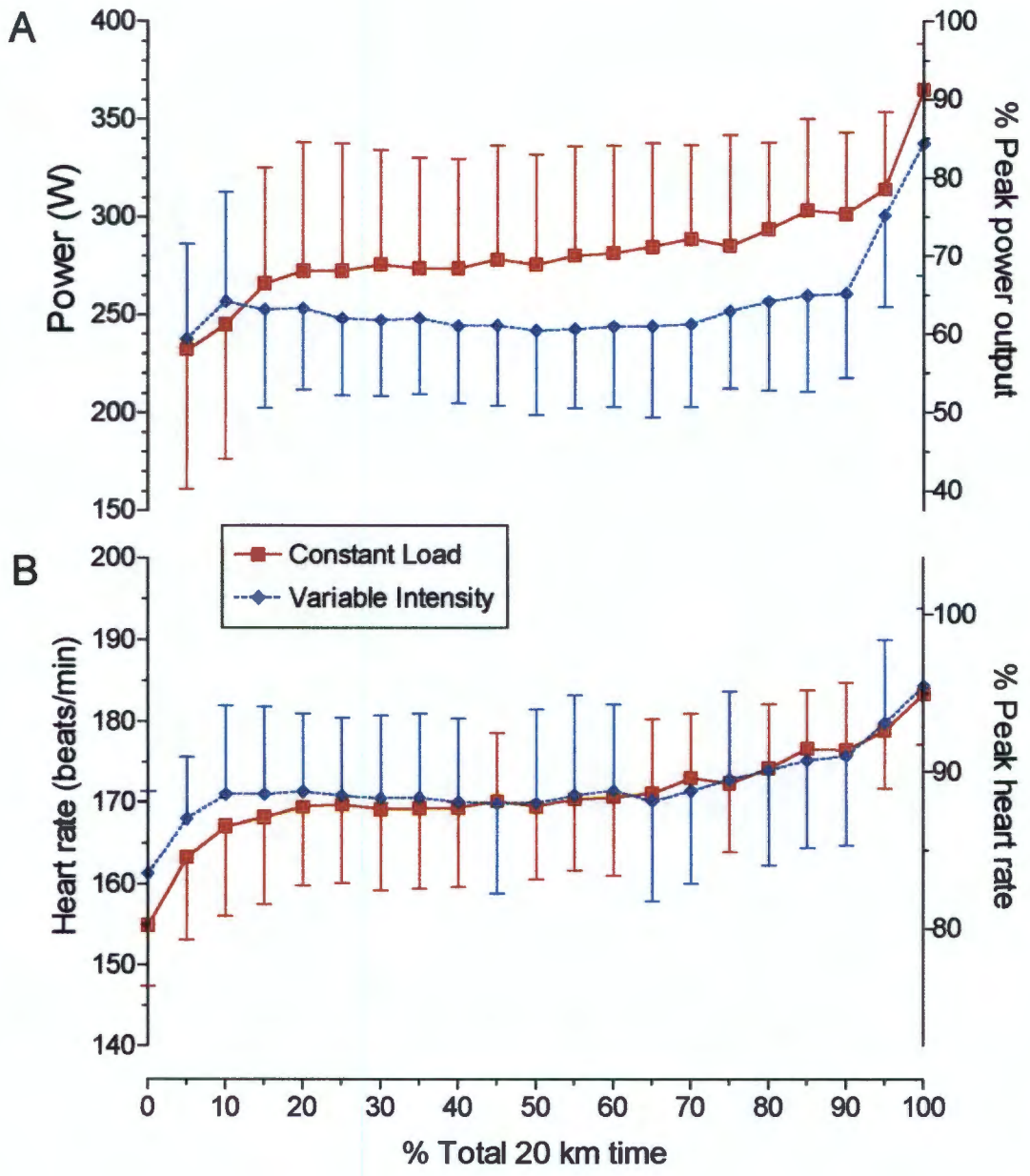


Figure 9.10 The group mean ±SD values for (A) power output (W), and (B) heart rate (beats/min) for each five percent section of the 20 km performance time trial following 140 min of either constant load (■), or variable intensity (◆) exercise.



9.4 Discussion

The first finding of this chapter was that despite five 20-min bouts of "stochastic" exercise, totalling ~70% of the entire workbout, the average $\dot{V}O_2$ was remarkably steady throughout both the constant load and variable intensity work (Table 9.3). Neither did the subjects perceive any differences in average effort during the two workbouts, or at any time point during the 140 min rides. Yaspelkis et al. (1993) reported that $\dot{V}O_2$ was elevated ~40% (from ~2.1 to 3.45 l/min) when their well-trained subjects increased their workrate from low (45% of $\dot{V}O_{2max}$) to moderate (75% of $\dot{V}O_{2max}$) intensity and that their subjects' ratings of perceived effort reflected the alterations in exercise intensity: during their low-intensity (45% of $\dot{V}O_{2max}$) workbouts, subjects found exercise to be relatively easy (9-12 units), but during the more intense exercise (75% of $\dot{V}O_{2max}$), perception of effort was higher (13-15 units). A possible reason for the discrepant findings between the results of the present study and that of Yaspelkis et al. (1993) could be that the variable-intensity exercise model employed in this study alternated rapidly between short bouts of low and high intensity work. This model was chosen because it is a more accurate simulation of real conditions in competition (Chapter Four; Jeukendrup and van Diemen 1998).

In contrast, Yaspelkis et al. (1993) employed a protocol in which subjects cycled for 30 min at 45% of $\dot{V}O_{2max}$ followed by six repeated 16 min periods of alternate cycling at 75% and 45% of $\dot{V}O_{2max}$ (8 min each) followed by a rest period, then a further period of alternate intervals (3 min at 45% $\dot{V}O_{2max}$, 3 min at 75% of $\dot{V}O_{2max}$).

As might be expected from similar $\dot{V}O_2$ values, the average HR responses to the variable-intensity and constant load trials were almost identical (Table 9.3). This finding emphasises the difficulties of attempting to monitor exercise intensity by HR data alone. In cycling, for example, HR cannot be considered an accurate indicator of workrate (power output) or speed in situations in which a cyclist is riding in a pack, or is free to choose their own pace. The results of the study in Chapter Four, and other researchers (Jeukendrup and van Diemen 1998) have previously reported that in mass start cycling races, HR response is random, with frequent changes in amplitude and frequency, and that such perturbations are not related to either speed, power output or course profile (Chapter Four; Jeukendrup and van Diemen 1998). More to the point, when the duration of a workload is short (<2 min) the cardiovascular response will lag behind any changes in muscle power output.

Despite the similar whole body responses ($\dot{V}O_2$, HR, energy cost) to the two different experimental protocols, there were differences in the lactate profiles between trials (Figure 9.2), with plasma lactate levels reflecting the variable intensity exercise. After the variable workbouts, lactate concentrations were ~1.5 mM higher than values at the same time during the constant-load ride. Despite the fact that there were periods of low-intensity exercise during the variable-intensity ride, plasma lactate concentrations tended to remain slightly elevated above those observed during the constant-load trial, particularly during the latter stages of the 140 min ride, resulting in a greater area under the lactate versus time curve for variable-intensity exercise. The lactate concentrations measured in the present study are similar to those reported by Yaspelkis et al. (1993). They are, however, somewhat lower than those measured by Coggan and Coyle (1988)

during intense cycling. These workers reported values of ~5 mM when their highly-trained subjects alternated every 15 min between 60% and 85% of $\dot{V}O_{2max}$ (Coggan and Coyle 1988).

Yaspelkis et al. (1993) have previously reported that during variable intensity cycling, CHO supplementation spared muscle glycogen compared to when subjects ingested only water. In that study, the glycogen sparing (~20%) was confined mainly to the type I muscle fibres, with little or no difference in glycogen use occurring in the type II fibres (Yaspelkis et al. 1993). Glycogen sparing in the type I muscle fibres with CHO ingestion has also been reported by Tsintzas et al. (1995) at the end of 60 min of constant-speed running at 70% of $\dot{V}O_{2max}$, and during submaximal running to exhaustion (Tsintzas et al. 1996). In both these studies, the glycogen sparing in the type I muscle fibres was remarkably consistent, and was between 25-28% when CHO was ingested, compared to a water control. Others (Bosch et al. 1994; Coyle et al. 1986), however, have observed no differences in glycogen utilisation after several hours of submaximal constant-load cycling when subjects were fed either CHO or water.

The second finding of the present investigation was the 27% reduction in total muscle glycogen utilisation during 140 min of variable-intensity cycling, compared to when subjects completed the same amount of work as constant-load exercise (Figure 9.5). The amount of glycogen remaining in the muscle (~80 mmol/kg w.w.) after 140 min is in close agreement with the value reported by Yaspelkis et al. (1993) after ~130 min of variable-intensity cycling (~90 mmol/kg w.w.). The difference in whole muscle glycogen utilisation between trials just failed to reach statistical significance. Although the total plasma glucose oxidised during the 140

min experimental rides was greater in VI than SS, such a difference cannot explain the reduction in calculated glycogen degradation . On the assumption of an active muscle mass of 8 kg during cycling (Kuipers et al. 1987) the ~15 g greater glucose oxidation in the VI compared to the SS trial would explain only a small proportion of the glycogen sparing.

However, the true rate of glycogen utilisation by contracting fibres cannot be accurately assessed by measuring changes in the total glycogen of muscle samples (Gollnick et al. 1974). Accordingly, PAS staining was performed to determine if there were similar patterns of glycogen depletion in the different fibre types (Figure 9.8). Such analysis revealed that less than 5% of the total number of type I fibres stained dark (3-5) for glycogen at the end of 140 min of constant load cycling, compared to over 40% at the end of the variable intensity exercise. Accordingly, almost >95% of type I fibres stained negatively or light (0-2) for glycogen after constant load exercise, compared to ~60% in the variable-intensity trial. On the other hand, there was a marked loss of glycogen from the type II fibres (those staining 0-1) after variable-intensity exercise (~10%) with little or no loss occurring after the constant load workbout.

In previous studies (Coyle et al. 1986; Essen 1978; Gollnick et al. 1973, 1974; Vollestad et al. 1984; Yaspelkis et al. 1993) the intensity of the PAS staining in individual fibres was rated visually by one or more of the investigators. The objectivity and reliability of the PAS rating procedure has been questioned (Gollnick et al. 1974). It has been noted that a considerable decline in glycogen levels may be necessary before any change in stain becomes apparent (Gollnick et al. 1974). In the present study an automated computer system scored the muscle samples, thus removing an element of observer bias. Furthermore, these

results are in excellent agreement with previous studies of muscle glycogen depletion patterns during both prolonged, continuous, constant load (Essen 1978; Gollnick et al. 1973; Vollestad et al. 1984) and "severe" (>80% $\dot{V}O_{2max}$) intermittent cycling (Essen 1978). Those studies show that during moderate-intensity (<70% $\dot{V}O_{2max}$), constant load exercise, type I fibres are the first to display reduced PAS staining, whereas intense, variable intensity exercise at close to 100% of $\dot{V}O_{2max}$ recruits both type I and type II fibres load (Essen 1978; Gollnick et al. 1973, 1974; Vollestad et al. 1984).

Although it has been suggested that muscle glycogen synthesis can occur during low-intensity (40% of $\dot{V}O_{2max}$) exercise when CHO is ingested and the glycogen content of the muscle is low (Kuipers et al. 1987), such synthesis is probably restricted to nonactive (type II) muscle fibres and is unique to the conditions of that experiment. Instead, based on both the biopsy data and histochemical analyses, the results of this study would suggest it is more likely that the 27% reduction in glycogen utilisation observed during exercise of variable intensity was due to a sparing of glycogen, probably in the type I fibres, as has previously been reported during variable intensity cycling by Yaspelkis et al. (1993) and in running by Tsintzas et al. (1995, 1996). Further support for this contention is the finding that the total blood glucose oxidised during variable intensity exercise was significantly greater than during the constant load work (Figure 9.4). The peak rates of blood glucose oxidation in the current investigation are in good agreement with previous studies which have used either arteriovenous sampling or isotopic tracer techniques (Broberg and Sahlin 1989; Coggan and Coyle 1991; Katz et al. 1986; Stein et al. 1989). However, they are much lower than the estimated ~2 g/min rates estimated by Coyle et al. (1986) at the end of intense, constant load

exhaustive cycling. Such differences are hard to explain, particularly as plasma insulin concentrations have been shown to be almost two-fold higher in variable intensity (Yaspelkis et al. 1993) than constant load cycling (Coyle et al. 1988).

Yaspelkis et al. (1993) found that, when compared to water ingestion, CHO supplementation reduced muscle glycogen use during variable intensity cycling. In that study, the glycogen sparing (~20%) was confined mainly to the type I muscle fibres, with little or no difference in glycogen use occurring in the type II fibres (Yaspelkis et al. 1993). Glycogen sparing in the type I muscle fibres with CHO ingestion has also been reported by Tsintzas et al. (1995) at the end of 60 min of constant-speed running at 70% of $\dot{V}O_{2max}$, and during submaximal running to exhaustion (Tsintzas et al. 1996). In both these studies, the glycogen sparing in the type I muscle fibres was remarkably consistent, and was between 25-28% when CHO was ingested, compared to a water control. Although others (Bosch et al. 1994; Coyle et al. 1986) have not observed any differences in glycogen utilisation after several hours of submaximal constant-load cycling when subjects were fed either CHO or water (for review see Tsintzas and Williams 1998). To the best of our knowledge, there are no reports in the literature comparing muscle glycogen utilisation during VI and SS exercise of the same average power output when subjects ingest CHO. However, the possibility remains that in the current study, CHO ingestion resulted in a net glycogen synthesis in some active (and inactive) muscle fibres during the VI ride. In support of this hypothesis, Kuipers et al. (1987) have previously reported that following a ride to exhaustion designed to result in glycogen depletion, net glycogen synthesis occurred in the non-active muscles of well-trained cyclists who ingested large (~500 g) amounts of CHO during a subsequent bout of prolonged (3 h) low-intensity (~50% of VO_{2max})

cycling. These workers found that muscle glycogen content was increased by an average of ~30% after the low-intensity workbout compared to the value at exhaustion (199 vs. 136 mmol/kg d.w.). However, the amount of CHO incorporated into muscle was likely to be much higher since these workers could not account for the fate of a large proportion of the CHO ingested by their subjects (~275 g). Although it is tempting to speculate that glycogen synthesis could explain the tendency for attenuated loss of glycogen during the VI ride in the current investigation, there was insufficient muscle biopsy tissue left to quantify if there had been any incorporation of ^{14}C into glycogen during both experimental rides.

The third finding of this study was that despite marked differences in the 140 min pre-load exercise bout, subsequent 20 km TT performance was not statistically different between the two experimental trials (Figure 9.10). This result differs from the study described in Chapter Seven in which performance in a similar TT was improved by 6% after well-trained cyclists had completed 150 min of constant load cycling at ~250 W (65% of $\dot{V}O_{2max}$) compared to when the same amount of work was undertaken as stochastic exercise in which the power output varied between 155-355 W (Chapter Seven). During the final 10 min of the 150 min "stochastic" ride, subjects sustained high work rates (>300 W) during the last 10 min of the ride, and finished with a bout of high (~340 W, >90% of $\dot{V}O_{2max}$) intensity cycling (Figure 7.1). Although no metabolic measures were taken in that study, such an intense bout of exercise could have resulted in cyclists commencing the TT with high blood (and muscle) lactate concentrations compared to the constant load

exercise. Evidence for this contention comes from an analysis of the power outputs during the first three-quarters of the 20 km TT. After the "stochastic" ride, power was consistently lower than constant load exercise, although riders were able to increase their speed during the latter stages of both TT, finishing at similar (~400 W) workloads. In contrast, during the final 10 min of the variable intensity workbout in the current study, power outputs only exceeded 300 W for brief periods (see Figure 9.1). More to the point, lactate concentration actually fell during the last 10 min of the variable intensity ride and were only marginally higher than at the end of the constant load ride (2.1 versus 2.4 mM, respectively). This small difference is unlikely to be of any physiological importance. Unfortunately, a third muscle biopsy was not taken at the end of the 20 km TT, largely because the *a priori* hypothesis was that there would be a performance enhancement after the constant load rather than the variable intensity workbout. Hence, it would be difficult to interpret any changes in glycogen utilisation if the exercise bouts were of different intensity and duration.

In conclusion, the final experimental chapter of this chapter aimed to examine the metabolic and performance responses to prolonged, variable intensity or constant load exercise of the same *average* intensity. Despite similar whole body responses to the two different exercise bouts (i.e. $\dot{V}O_2$, HR, RPE, energy expenditure), lactate concentrations were consistently higher throughout the variable-intensity compared to the constant load exercise. There was also evidence to suggest that variable-intensity exercise may result in glycogen sparing in the type I muscle fibres compared to when the same work is performed as constant load exercise and carbohydrate is ingested throughout exercise. Further support for this interpretation was the finding that plasma glucose oxidation during

variable-intensity exercise was significantly greater than during the constant-load work. Such differences, however, did not impact on subsequent high-intensity exercise performance. It is concluded that when well-trained subjects perform prolonged, variable-intensity exercise, or constant load exercise of the same average intensity, there are only small differences in skeletal muscle carbohydrate metabolism and recruitment, and that such differences do not affect the performance of a subsequent bout of high-intensity cycling. It is clear that further research is necessary in a variety of locomotor sports (cycling, running, swimming, rowing etc.) to determine the pacing strategies that optimise performance.

CHAPTER TEN

SUMMARY

10.1 Summary

The studies described in this thesis evaluated selected aspects of competitive bicycle racing. Despite the popularity of the sport, there are relatively few studies that have examined either the physiological or metabolic responses to performance of well trained cyclists to either training or competitive situations. Furthermore, whilst there currently exists a wealth of information regarding the physiological and metabolic responses to fixed intensity endurance cycling, the results of such studies may not be ecologically valid when comparisons to actual competition are to be drawn.

The finding of the first study (Chapter Four), was that well-trained amateur cyclists produced markedly different HR responses during the individual TT stages when compared to the mass-start bunch races. During the individual TT stages the riders produced near maximal efforts from the start of the race, rapidly attaining high (~93% of peak) HR which were then maintained for the remainder of the race, in contrast, during the mass start road race stages, HR's were random with variable changes in the frequency and amplitude of the responses. This is a finding of fundamental importance as it highlights the stochastic nature of group cycling in well-trained amateur riders and show that the energy expenditures of these elite racing cyclists are probably a function of bunch riding. It also raises the question of whether the current practice of assessing physiological and metabolic responses to cycling under steady state conditions is of valid for cyclists who will compete in events where there are continual changes in exercise intensity.

While measures of either the exercise time to exhaustion at a fixed work rate, or the change in a selected submaximal physiological variable are perfectly valid

measures of exercise capacity in themselves, they do not reproduce the metabolic or physiological demands of either TT or mass start road races. Accordingly, the second study of this thesis described in Chapter Five, measured the physiological responses of well-trained cyclists in a laboratory simulated TT and to determine if such responses were similar to those previously recorded in the actual competition (Chapter Five). To be of further scientific value, it was also necessary to assess the reliability of a laboratory ergometer during repeated TT rides over 20 and 40 km (Chapter Five), durations that often matched those often used during competition.

The first important finding of the study described in Chapter Five was that the simulated performance TT rides on the Kingcycle ergometer were highly reproducible. This was indicated by a small CV between individuals performances over both 20 km and 40 km distances. The second major finding of this study was that the HR data recorded in the laboratory TT were comparable to the HR data recorded in shorter distance TT in actual competition and in other laboratory studies (Chapter Four; Foster et al. 1993a). These findings are of fundamental importance as they show that the Kingcycle ergometry system can now be employed for laboratory based testing of well-trained cyclists on their own bicycles, which will be both highly reliable, and produce physiological responses similar to those previously observed in the field.

Although the reliability of a measure of performance is an important pre-requisite for the sports scientist, the validity of that measure is also essential if laboratory results are to be used to monitor or predict actual race times during competition. Hence, the aim of study described in Chapter six was to determine the validity of the Kingcycle ergometer system by comparing self paced TT performances

measured in the laboratory on the Kingcycle ergometer system with actual race performances over the same distance in competition on the road. The results of this study showed that although, on average, the laboratory TT were 8% faster than the road based competition TT times, the physiological responses to both laboratory and "road" were very similar. This too is an important finding as it allows exercise physiologists to simulate the physiological responses of TT competitions in the laboratory setting, giving further weight to the ability to reproduce the physiological responses to actual individual or self paced competition under standardised laboratory conditions.

The results of the investigations described in Chapters Five and Six illustrate that self paced, performance testing on the cyclists own bicycle is not only possible, but also highly valid and reliable. However, the results of the study described in Chapter Four clearly show that during group cycle races the responses to competition are far from steady state, being variable intensity in nature.

Accordingly, the primary aim of the study described in Chapter Seven was to evaluate the physiological responses of well-trained cyclists to laboratory based bout of variable intensity exercise designed to mimic the stresses of a mass start cycle race that had previously been observed during actual competition (Chapter Four). A secondary aim was to assess the effects of prolonged, sub-maximal steady-state and variable intensity cycling on subsequent cycling TT performance in an effort to try and determine which riding strategy would be more advantageous to the athlete.

The first major finding of this investigation was that the HR responses recorded during the 150 min variable intensity ride in the laboratory compared to that

recorded previously during an actual competition of approximately the same duration (Chapter Four). Such similarities in HR responses indicate that the variable intensity workload employed in Chapter Seven closely mimicked the physiological demands of the mass start cycle racing (Chapter Four). This is a crucial finding as it allows the sports scientist to replicate the variable physiological responses to mass start cycle racing under standardised laboratory conditions. The second important finding from the study described in Chapter Seven was that the athlete's ability to perform a self paced 20 km TT was reduced following the 150 min bout of variable work, despite the same average intensity. This observation may have a significant bearing on the recommendations for performance enhancement in such group races.

In conclusion, Section A of this thesis showed that during actual competition, well-trained amateur cyclists maintained a constant workrate during an individual TT event, whilst, due to the group dynamics, competitive bunch cycle racing results in a variable intensity workload for the same athletes. Further, it was shown that it is possible to replicate the physiological demands of both the individual TT events and mass start cycle competitions under standardised laboratory conditions. Accordingly the second half of this thesis used these models to focus on some of the physiological and metabolic determinates of cycling performance during both constant load (i.e. individual TT) and variable load (i.e. mass start road races) exercise.

One such intervention frequently practised by cyclists is carbohydrate ingestion, which has been shown to delay the onset of fatigue during prolonged (>90 min) moderate intensity (70-75% $\dot{V}O_{2max}$), constant load cycling (for review see Coggan

and Coyle 1991). Recently it was also shown that carbohydrate ingestion can also improve the performance of high intensity TT cycling lasting ~1 h in both moderate (El Sayed et al. 1997; Jeukendrup et al. 1997) and warm (Below et al. 1995) environmental conditions. However, whether carbohydrate ingestion improves cycling TT performances in events lasting ~25-30 min, (such as that previously described in Chapter Four) has yet to be determined. Accordingly, using the Kingcycle ergometry system, which was validated in the studies described in Section A of this thesis, (Chapters Four and Five) the aim of Chapter Eight was to determine whether CHO ingestion would improve cycling TT performance lasting <30 min. In this study 14 well-trained cyclists performed two 20 km TT after ingestion of ~600 ml of fluid containing either ~40g CHO or a placebo.

The results of this study showed that CHO ingestion did not improve 20 km cycling TT performance. This finding has practical relevance to cyclists as either they cannot, or usually choose not to consume any type of beverage in a race of this duration.

In contrast to the high intensity, steady state responses that prevailed during the TT events in Chapter Four, the responses to the mass-start endurance cycle races were variable, as highlighted by the random shifts in the frequency and amplitude of the heart-rate (HR) responses of the cyclists. Furthermore, Chapter Seven revealed that 20 km TT performance which followed 150 min of either SS or VI cycling undertaken at the same *average* power output was 6% faster after SS. Whilst little is known regarding the metabolic responses to VI exercise in which the workrate fluctuates in a random fashion, it was speculated in Chapter Seven that the repeated work jumps during VI may have impaired subsequent exercise as a result of an increased muscle glycogen utilisation compared to SS exercise.

However, the absence of any metabolic measurements precluded an attempt at elucidating the exact mechanism(s) associated with the reduction in TT performance following VI exercise.

To date, relatively few studies have examined intense intermittent exercise, or variable-intensity work in which power output or speed changes in a random or stochastic nature. Reasons for this may include (i) the lack of appropriate equipment; (ii) concerns that non steady-state conditions do not permit valid or reliable estimates of substrate metabolism or (iii) the belief that steady-state conditions are common in most sports.

Whilst Chapter Four clearly showed that steady-state conditions do not prevail during mass start cycle racing, the results from the study described in Chapter Seven indicated that it is possible to replicate such variable intensity conditions in the laboratory setting.

Accordingly, the final study described in Chapter Nine utilised a similar approach to compare the metabolic and hormonal responses to prolonged (140 min) cycling at either constant load (232.5 ±10.6 W, ~70% of $\dot{V}O_{2peak}$) or at variable-intensity (143.1 ±6.5 to 314.7 ±14.3 W, ~40 to 85% of $\dot{V}O_{2peak}$) of the same *average* intensity. The aims of this study were, firstly, to evaluate the metabolic and hormonal responses to prolonged (140 min) cycling in well-trained men ingesting CHO throughout both SS (~70% of $\dot{V}O_{2peak}$) and VI (40 to 85% of $\dot{V}O_{2peak}$) cycling of the same *average* intensity. A second aim was to determine the effects of these two different exercise pre-loads on subsequent 20 km TT performance. Despite similar whole body responses to the two different exercise bouts (i.e. $\dot{V}O_2$, HR, RPE, energy expenditure), lactate concentrations were consistently higher

throughout the variable-intensity compared to the constant load exercise. There was also some evidence to suggest that variable-intensity exercise may result in glycogen sparing in the type I muscle fibres compared to when the same work is performed as constant load exercise and carbohydrate is ingested throughout exercise. However, this sparing was small, and not statistically significant. Indeed, such differences in muscle carbohydrate metabolism did not impact on subsequent high-intensity exercise performance. The results of this study are in direct contrast to those reported in Chapter Seven. The reasons for these differences are not immediately obvious. However, during the last 10 min of the variable intensity ride utilised in Chapter Seven, subjects sustained high work rates (>300 W) and finished with a bout of high (~340 W, >90% of $\dot{V}O_{2max}$) intensity cycling.

It is concluded that when well-trained subjects perform prolonged, variable-intensity exercise, or constant load exercise of the same average intensity under the conditions evaluated in this study, there are small differences in skeletal muscle carbohydrate metabolism and recruitment, but that such differences do not affect the performance of a subsequent bout of high-intensity cycling. Whether such a finding would apply to a short duration (e.g. a track points race) or very prolonged exercise bouts (for example, the Tour de France) remains to be established.

In summary, the studies conducted in this thesis have examined the responses of well-trained cyclists to individual and mass start cycle racing. The physiological responses of both the steady state, self paced performance of the individual events, and the variable intensity responses to group events were replicated under

standardised laboratory conditions. These methods of testing were then used to determine metabolic responses to constant load and variable intensity exercise. During these metabolic studies, it was shown that CHO ingestion did not enhance 20 km individual TT performance. Further, there were similar whole body responses to VI and SS exercise during 140 min of exercise in well-trained cyclist, despite differences in lactate response, and evidence to suggest that variable-intensity exercise may result in a small glycogen sparing effect in the type I muscle fibres when compared to the same total constant workload.

The results from the studies conducted for this thesis suggest that in well-trained cyclists, when the total work performed (for approximately 2 h) is of the same average power output, there are only minor physiological and metabolic differences between steady state and variable intensity exercise. Although it would appear that during major competitions more work is done during the second half of an event, this may simply be a function of the riders level of ability, and the initial pacing control of the race.

In conclusion, the study described in Chapter Four, revealing the stochastic nature of group cycle racing, has unlocked a feature of cycle racing previously unrecognised by the exercise physiologist. This thesis then partially answers one question raised by that finding, namely that steady state and variable intensity exercise at the same average power output produces very similar physiological and metabolic responses. Taken collectively, the results of the series of studies conducted for this thesis highlight the need for exercise physiologists to evaluate the physiological responses of athletes to actual competition in the field, and then replicate them under laboratory conditions. Such a strategy will allow for the determination of those metabolic variables which could provide an insight into the

factors limiting or predicting performance. This in turn will lead to them to the development of the most appropriate training methods for performance enhancement in these individuals.

CHAPTER ELEVEN

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