Kent Academic Repository

Coleman, Damian Alan (2001) *The energetics of competitive road race cycling.* Doctor of Philosophy (PhD) thesis, University of Kent.

The Energetics of Competitive Road Race Cycling

By

Damian Alan Coleman

Canterbury Christ Church University College

Thesis submitted to the University of Kent at Canterbury for the Degree of
Doctor of Philosophy

October 2001

Abstract

One of the roles of the sport scientist is to ascertain the demands of a competitive event, and establish training regimes for success in the chosen event. The aim of this thesis was to establish the relative demands, and establish the physiological responses to road race cycling.

Study 1 - Physiological Responses of Competitive Road Race Cyclists.

Research has shown that the Kingcycle (EDS Portaprompt Ltd, High Wycombe, UK.) maximal minute power (MMP) test correlates strongly to performance during endurance time trial cycling events (Davison *et al.*, 1997). The aim of this study was to evaluate the test as a method of ascertaining a relative exercise intensity in a homogenous population of racing cyclists.

Eleven male trained cyclists consented to participate in this study. Following familiarisation, subjects underwent two MMP tests, followed by a further six 10-minute submaximal Kingcycle trials at relative exercise intensities (60, 65, 70, 75, 80, 85% MMP).

The coefficient of variation for maximal minute power output between MMP tests was 1.72% (SEE 2.6%). Submaximal test data revealed that subjects had similar lactate inflection point (68.9 ± 2.2% MMP). Thus it appears the figures derived from MMP are reliable, and the figures attained reflect lactate kinetics thus explaining the high correlation between MMP and performance.

Study 2 - Data Collection from Competitive British Cycling Federation Events.

Previous research has used heart rate data to assess the demands of road cycling by establishing a heart rate:power relationship in the laboratory and extrapolating the laboratory data to heart rate data recorded in the field (Palmer *et al.*, 1994). The development of a portable powermeter (SRM, Julich, Welldorf, Germany.) allows for the simultaneous recording of power and heart rate in the field. The aim of this study was to use the SRM powermeter to evaluate this method of heart rate analysis during competitive cycling competition.

Nine male subjects participated in this study. Following familiarisation, subjects underwent MMP testing ± 7 days of a road race in which subjects competed and recorded the race with an SRM powermeter. Power output, pedal cadence, heart rate and speed/distance were recorded from the competitive races at 1 second resolution. Five subjects also underwent Wingate testing in the laboratory during the same time period.

Mean power output from the races was 264 watts, mean heart rate was 169 beats min^{-1}, which was significantly higher (11 beats.min^{-1}, p<0.05) than the predicted heart rate from laboratory tests similar to Palmer *et al.* (1994). The data also revealed road race cycling to be highly intermittent in nature, with high power outputs (>MMP) recorded during hill climbing. The findings therefore question i) the methods used by Palmer *et al.*, (1994), and, ii) the ecological validity of the previous studies that have attempted to simulate road race cycling (Brouns *et al.*, 1989, Palmer *et al.*, 1997).

Study 3 - Correlates of Cycling Hill Climb Ability.

Study 2 revealed the importance of hill climbing to the road race cyclist. Hill climbing during road race cycling generally involves seated and standing cycling, the aim of this study was to evaluate the simulated hill climbing ability of competitive cyclists relative to standard stationary seated ergometer testing (Kingcycle).

Following familiarisation, eight trained cyclists undertook Kingcycle MMP, Wingate and simulated climbing (Woodway treadmill, Guhb, Germany.) tests. Two simulated climbs were conducted, the first at a 6% gradient for 6 km, and the second at a 12% gradient for 1 km.

Subjects sustained 92.6 % of MMP during the 6 km climb, despite recording the same % of maximum heart rate, and lower blood lactate concentration (B[Lac$^-$]) than when exercising at 85% in study 1. MMP test data and Wingate mean power both significantly correlated to performance time when data were expressed relative to body mass (r^2 >0.79). These findings indicate that seated stationary ergometer testing can predict hill climb ability, and that when riding on a gradient the relationship between power, heart rate, and B[Lac$^-$] is different when compared to level cycling.

Study 4 - The Effect of Gradient on Cardio Respiratory Measures during Submaximal Cycling.

Study 3 revealed that riders can sustain higher power outputs on a gradient than when compared to level cycling, this could be attributed to the changes in cycling position during hill climb performance testing. The aim of this study was to evaluate the submaximal cardio respiratory responses to level and graded seated cycling.

Six trained cyclists undertook MMP testing followed by two submaximal trials at (60% of MMP) on a motorised treadmill (Woodway). Submaximal trials consisted of a level (0%) and graded (6%) condition.

There was no significant difference for power output, heart rate, RER and gross mechanical efficiency between level and graded cycling (p>0.05). Significantly lower oxygen consumption (VO_2), Ventilation (VE), and carbon dioxide production (VCO_2) values were recorded during graded cycling. These results indicate that respiratory measures during submaximal cycling are lower during graded cycling, possibly explaining the high power outputs sustained during field and laboratory hill climb performance.

Study 5 - The Effect of Glucose Polymer Supplementation on Simulated Road Race and Even Power Cycling.

The data from study 2 indicates potential limitations with the method of evaluation of road cycling by Palmer *et al.* (1994), and the previous attempts to simulate road race cycling (Brouns *et al.*, 1989, Palmer *et al.*, 1997). The aim of this study was to construct a valid road race laboratory simulation protocol, and compare the effects of glucose polymer ingestion and the physiological responses to even power and simulated road race cycling.

Six male trained cyclists undertook MMP testing followed by two even power, and two simulated road race trials. Each condition consisted of a glucose polymer (Tekno fuel, Medway, UK) and a placebo trial. The trials were two hours in duration. The mean power output equated to 60% of MMP. The trials were followed by a 2 km hill climb performance test at a 6% gradient. A fresh performance test was also completed by all subjects.

There were no significant differences between even power and simulated road race cycling for mean power, heart rate, VO_2, ammonia (NH_3), urea, economy, or efficiency values during the trials (p>0.05). Significantly higher B[Lac⁻], and VCO_2 production were observed during simulated road race cycling (p<0.05). Glucose polymer ingestion improved post even power (5.3%) and post simulated road race (6.01%) cycling performance. Power output sustained during the performance test was significantly lower following the simulated road race trial when compared to fresh and even power trials. Better relative performance following simulated road race cycling was found in riders with lower plasma NH_3 and urea concentrations (r>0.81), and in those riders with the higher B[Lac⁻] following the performance test (r=-0.81). These factors did not significantly correlate to post even power performance.

These results indicate there are specific physiological demands of intermittent road race cycling when compared to performance following even power cycling.

The findings from this study highlight the specific demands of road race cycling, the potential problems of interpreting heart rate data in the field, and the physiological responses of trained cyclists during hill climbing in the field or simulated in the laboratory.

Contents

List of Figures

competitive road race events.

Chapter 14. The Effect of Glucose Polymer Supplementation on Simulated Road Race and Even Power Cycling.

List of Tables

List of Appendices

Acknowledgements

Glossary of terms

Term	Definition
ADH	Anti diuretic hormone
ADP	Adenosine diphosphate
AMP	Adenosine monophosphate
AMPD	Adenosine monophosphate dehydrogenase
ATP	Adenosine triphosphate
B[Lac⁻]	Blood lactate concentration
BCAA	Branched chain amino acid
BCAAT	Branched chain amino acid amino transferase
BCOA	Branched chain oxo acid
BCOADH	Branched chain oxo acid dehydrogenase
beats min⁻¹	Beats per minute
BLac⁻	Blood lactate
Ca²⁺	Calcium ion
CHO	Carbohydrate
CHO_D	Carbohydrate depleted
CHO_L	Carbohydrate loaded
Cl⁻	Chloride ion
CONT	Continuous
Cr	Creatine
CS	Citrate synthase
EMG	Electromyogram
EP	Even power trial
FFA	Free fatty acids
G	Glucose trial
GC	General classification
GME	Gross mechanical efficiency
H+	Hydrogen ions
HPO₄⁻²	Phosphate ion
IMP	Inosine mono phosphate
INT	Intermittent
K⁺	Potassium ion
kg	kilogram
KJ	Kilojoules
l min⁻¹	Litres per minute
LDH	Lactate dehydrogenase
MAOD	Maximal accumulated oxygen deficit
Mg²⁻	Magnesium ion
ml kg⁻¹ min⁻¹	Millilitres per kilogram per minute
MMP	Maximal minute power
MMP kg⁻¹	Maximal minute power per kg per minute
MP	Mean power (Wingate cycling test)
Na⁻	Sodium ion
NAD⁻	Nicotinamide adenine dinucleotide ion
NH₃	Ammonia
V̇CO₂ l min⁻¹	Volume of carbon dioxide litres per minute
V̇O₂ l min⁻¹	Volume of oxygen litres per minute
V̇O₂ ml kg⁻¹ min⁻¹	Volume of oxygen millilitres per kilogram per minute
V̇O₂max l min⁻¹	Maximal volume of oxygen litres per minute
P	Placebo trial
PCr	Phosphocreatine
Pi	Inorganic phosphate
PNC	Purine nucleotide cycle
PO₂	Pressure of oxygen
PP	Peak power (Wingate cycling test)
RER	Respiratory exchange ratio
Revs min⁻¹	Revolutions per minute
RR	Road race simulation trial
SRM	Schoberer Rad Messtechnik
TCA	Tricarboxylic acid cycle
TOTN	Tour of the north
W	Watts
Wmax	Maximum power required to achieve V̇O₂max

Chapter 1. Introduction

There are two distinct disciplines in the world of competitive road cycling, massed start road race cycling, and individual time trial cycling. Within each discipline there are variations in the distance of each event, the type of course/track where competition takes place, and the category of the competitive riders taking part in the races.

One of the roles of the sport scientist is to ascertain the demands of a competitive event, and establish training regimes for success in that event. Olds *et al.* (1995) indicated that the physical requirements of a rider undertaking competitive cycling are going to be dependent upon the specific discipline involved in the competition.

The major difference between these two cycling disciplines is the use of drafting. During time trial cycling the riders start alone at timed intervals, with no drafting of other competitors allowed. In massed start road race cycling drafting is a key component of the sport. Drafting causes reductions in the energy cost of cycling for a given speed (McCole *et al.*, 1990) thus performance outcomes involve both physical and tactical ability.

Cycling exercise is a popular method of assessment in the laboratory. Many studies have specifically looked at laboratory simulated time trials, or have used laboratory simulated time trials as a performance test following intervention (Balmer *et al.*, 2000, Coleman *et al.*, 1995, Davison *et al.*, 1997, Lindsay *et al.*, 1996, Loftin and Warren, 1994). However, there is limited published research on road race cycling, giving the sport scientist limited reviewed evidence to base training regimes, and appropriate laboratory assessment of this discipline.

The majority of published research on road race cycling has documented descriptive characteristics of the road race cyclist (Burke *et al.*, 1977, Sjogaard, 1984, Tanaka *et al.*, 1993). There has been limited research on the simulation of this discipline in the laboratory (Brouns *et al.*, 1989, Palmer *et al.*, 1997). Burke *et al.* (1977) speculated that the demands of road racing involved sprinting, hill climbing and breaking away from other drafting riders. Incorporating these activities into laboratory simulations would be important in the study of the sport. The papers that have attempted to simulate road race cycling have not incorporated these demands into their protocols, therefore a degree of caution is required when interpreting the results of these studies.

The present study aimed to assess the demands of competitive road race cycling, thus providing data for the construction of an appropriate laboratory simulation of this discipline, which would further the current body of knowledge that exists on road race cycling.

Chapter 2. The Energy Demands of Cycling

The aim of this chapter is to review the current research on the energy demands of cycling and the factors associated with the fatigue accompanying this type of exercise. According to Kyle (1991), the energy requirements of cycling are to overcome external frictional forces; which are predominantly wind resistance and equipment inefficiencies. Kyle (1991) also indicated that decreases in the energy required for a given speed during cycling were possible through modification of the riders' technique, position, equipment and physiology.

During this chapter it is intended to focus upon the demands of (i) time trial/even power cycling, and, (ii) the contrasting requirements of variable intensity road race cycling. The major difference between these two cycling disciplines is that during time trial cycling the riders start alone at timed intervals, with no drafting of other riders allowed. Drafting is allowed in massed start road race cycling, which causes reductions in the energy cost of cycling for a given speed (McCole et al., 1990) thus performance outcomes involve both physical and tactical ability.Coyle et al. (1991) reported some specific physiological factors that predicted performance in time trial events. In comparing two groups of riders they reported that 'elite national class' riders had a larger proportion of slow twitch muscle fibres, increased muscle capillary density, higher citrate synthase activity and lower lactate dehydrogenase (LDH) activity compared to the 'good state class rider'. Time trial cycling in terms of power output is essentially a steady state discipline, with mathematical calculations by Swain (1987) indicating that the fastest rides on flat courses (with no wind) would be rides at an even power, with small deviations $\pm$ 5% on climbs and descents on hillier courses. Both road race cycling and time trialing events are predominantly endurance based, with time trial efforts ranging from 10 miles (17-35 minutes in duration) to 12 and 24 hour rides, and road races ranging from 45 minutes in British criterium races to over 7 hours in European 'Classics'. Therefore aerobic metabolism would appear critical in the outcome of all of these events.

According to McArdle et al. (1991), the three major requirements for aerobic energy metabolism are

i) To have substrate available to donate electrons.

ii) To have an adequate Oxygen (O_2) supply. iii) To have available the enzymes and 'metabolic machinery' to enable reactions to occur.

2.1 Substrate availability

Fatigue during steady state cycling lasting over one hour in duration has often been shown to coincide with the depletion of muscle glycogen stores, and a fall in blood glucose supply (Bergstrom and Hultman, 1967). Studies have shown dependence upon muscle, and blood borne glucose concentrations at intensities varying from 50% $\dot{V}O_2max$ to 85% $\dot{V}O_2max$ during cycling exercise (Massicote et al., 1985. Coggan and Coyle, 1989). It appears that adequate glucose availability is critical to the performance of the time trial cyclist even for races as short as one hour in duration (Below et al., 1995). As muscle glycogen levels become

2

depleted, there is a gradual shift to blood borne glucose as substrate for metabolism. Glycogenolysis in the liver maintains blood glucose levels if the rate of extraction by the exercising muscle mass is equal to the rate of glucose production by the liver (Coggan and Coyle, 1989). Flynn *et al.* (1987) reported a critical blood glucose level <3.5mmol.l^{-1}, below which exercise will be significantly impeded with fatigue approaching. Glucose supplementation has been shown to prolong endurance during steady state cycling. Supplementation provides an additional source of carbohydrate (CHO), which allows CHO metabolism to continue at a higher rate (compared to water trials) later in exercise (Coggan and Coyle, 1988). Another potential source of CHO to fuel muscle contraction is the utilisation of blood lactate (BLac$^-$) as a substrate. During brief intense contractions, and during steady state cycling, under conditions of low CHO availability, B[Lac$^-$] has been reported to be lower when compared to a CHO loaded state (Neville *et al.*, 1993). The lower B[Lac$^-$] under conditions of low CHO availability could be explained by the inhibition of glycolysis due to the obvious reduction in available substrate, plus it has also been speculated that the reduced B[Lac$^-$] are the result of lactate (Lac$^-$) oxidisation within the muscle (Anderson and Rhodes, 1991). Boulay *et al.* (1997), indicated a further possible reason for reduced B[Lac$^-$] (which they reported during 90 minutes of endurance cycling), could be due to muscles shifting from being net releasers of Lac$^-$, to removers of Lac$^-$ from the blood therefore net uptake by that muscle. The oxidative slow twitch fibres are believed to have the greatest affinity for the conversion of Lac$^-$ back into pyruvic acid. It is also believed that Lac$^-$ uptake by the resting fibres will also occur during periods of Lac$^-$ production, and they use it as substrate for general cellular function (Anderson and Rhodes, 1991). BLac$^-$ is also believed to contribute to the production of liver glycogen, or directly converted into glucose in the liver and released into the blood stream (MacDougall *et al.*, 1977). Bergstrom and Hultman (1967) observed this to be the case where not only was there a large uptake of BLac$^-$ by the liver, but also a larger O_2 extraction required in this region to oxidise the BLac$^-$. This extra metabolism required because of the elevated B[Lac$^-$] could result in increased aerobic demand, placing more demands upon the cardiovascular system. This has important implications during variable intensity exercise. Bergstrom and Hultman (1967) reported that resting fibres were still glycogen loaded at fatigue, indicating that resting muscle fibres cannot donate glucose to the active muscles. Therefore, the oxidisable substrate that has been converted into lactate in the working muscle cell has been transported away from the active muscle mass, into a cell that has ample glycogen reserves and oxidised.

Depletion of muscle glycogen will lead to a reduced rate of CHO oxidisation, if blood glucose supply is not adequate to maintain the rate of CHO utilisation. If this occurs, a reduction in blood glucose concentrations occurs which has been seen in many studies. Coggan and Coyle (1987) have speculated on the effects of reduced adenosine triphosphate (ATP) availability and fatigue during prolonged exercise. Decreased levels of blood glucose have been shown to stimulate the release of glucagon from the alpha cells of the pancreas, (thought to be due to reduced glucose perfusion of the pancreas. Galbo, 1975). Glucagon is believed to have a gluconeogenic effect (converting amino acids and glycerol into glucose), thereby increasing the utilisation of these substrates for oxidisation in contrast to glycogen. Lemon and Mullin (1980) reported the effects of

3

depleted CHO stores on substrate utilisation during exercise. They compared a carbohydrate-depleted trial (CHO$_D$) to a CHO loaded condition (CHO$_L$). During the CHO$_D$ trial there was an increase in energy derived from protein sources during a 1-hour ride at 61% $\dot{V}O_2$max, (CHO$_D$ 10.4%, CHO$_L$ 4.4% of energy from protein). The reduced rates of ATP availability during CHO$_D$ according to Lemon and Mullin (1980) hindered the cell's ability to synthesise phospholipids, causing a reduction in the cell's ability to maintain the cell membrane. The authors speculated that increased enzyme efflux into the blood may have occurred; these enzymes were then degraded down into amino acids and then deaminated, increasing plasma levels of ammonia and serum urea. This has also been reported by Brouns et al. (1990), where under conditions of CHO availability, less ammonia accumulation occurred. Costill and Hargreaves (1992), speculated that increased ammonia and Inosine monophosphate (IMP) concentrations were due to CHO depletion and the breakdown into component parts of adenosine monophosphate (AMP) in the donation of its phosphate to adenosine diphosphate (ADP) in the rephosphorylation of ATP. Brouns et al. (1990) also reported progressive elevations in ammonia concentrations as glycogen stores became depleted and a correlation between incidents of muscle cramp and high ammonia levels. Ammonia has also been associated with peripheral fatigue and central nervous system dysfunction (Urhausen and Kindermann, 1992). The implications of ammonia accumulation are discussed in chapter four. Muscle, liver and blood glucose sources appear critical to the endurance athlete's performance during sub maximal exercise. However the relationship during high intensity exercise is less clear. During road race cycling it is likely that there will be periods of increased intensity where tactical attacks will be made, and/or breakaway groups will form at the front of the race, with sprints and hill climbing (Burke et al., 1977). To be a successful road race cyclist it is important to be able to react and have the physical capacity to respond to such moves. Jenkins et al. (1994) noted that at intensities above the anaerobic threshold, electrolyte displacement, intramuscular acidosis, and failure of the calcium pumps to return calcium to the sarcoplasmic recticulum have been indicated as major factors in fatigue. Hargreaves et al. (1984) reported that glycogen depletion significantly reduced anaerobic performance, however, Symonds and Jacobs, (1989) found no significant difference between glycogen reserves and high intensity performance (50 maximal knee extensions on an isokinetic bench), indicating that factors other than energy reserves may influence the rider's ability during intense exercise. Jacobs (1981) reported that there is a lower muscle glycogen limit (40 mmol.kg^{-1}) below which anaerobic performance will be hindered thus possibly explaining the equivocal findings. Some of the studies looking into high intensity performance have not depleted muscle glycogen to this extent, and therefore it would be unlikely to affect the anaerobic performer. This was acknowledged by Symonds and Jacobs (1989). Their experimental group (low CHO) had muscle glycogen reserves of 153 mmol.kg^{-1} d.w remaining at the onset of anaerobic exercise. This was compared to muscle glycogen levels of 427 mmol.kg^{-1} d.w for the control group. This has further been illustrated by Drabbs and Maud (1997) who reported improved peak power output during the Wingate cycling test post (compared to pre) $\dot{V}O_2$max testing (5 minute recuperation between tests) where some glycogen depletion would have occurred, but not

to extreme levels. However, due to the associated endurance component of road cycling also taxing limited glycogen reserves, plus the implications of high demands upon muscle glycogen which have been reported during high intensity sprinting (Boobis *et al*, 1982), glycogen availability may be a limiting factor in both sustained aerobic, and the intense sprinting requirements of road race cycling.

During repeated sprint type exercise, PCr has been implicated as a major contributor to the maintenance of power output, (Balsom *et al*., 1992). Tzintzas *et al*. (1998) reported that reductions in muscle glycogen at the end of an endurance run have also been shown to correlate significantly with reductions in phosphocreatine (PCr). They concluded that carbohydrate availability spared PCr, reflecting a greater supply of energy from the oxidative system, rather than depleting this 'alactic' phosphagen system to supply energy requirements. Therefore reductions in muscle glycogen could limit road race cycling performance due to an inability to sustain work output anaerobically. The relationship between glycogen depletion and time has been studied by Bergstrom and Hultman (1967), they revealed a non linear response, with glycogen depletion following a 'semi-logarithmic' pattern. They noted the greatest depletion occurring during the first 15 minutes of cycling (intensity 155 watts), followed by a tailing off in depletion when the residues of muscle glycogen fell to low values (0.1g per 100g wet muscle). They noted that it was at this point that blood borne glucose began to play a greater role in CHO oxidisation within the muscle. Due to the limited stores of CHO, research into altering blood glucose levels with CHO feeding have been extensive, with the majority showing increased endurance and/or performance improvements, although the mechanisms by which CHO feeding improves performance have yet to be fully documented. Nassis *et al*. (1998) suggested that possible improvements in performance were due to the prevention of hypoglycaemia, and also the ability to maintain a high rate of CHO oxidisation. CHO ingestion before endurance cycling at 'anaerobic threshold' intensities for one hour has also been shown to improve performance (Below *et al*., 1995), however, in events of shorter duration (26-30 minutes), pre exercise carbohydrate feeding has been shown to have no beneficial effect on performance (Palmer *et al*., 1998); this is understandable as it would be unlikely that carbohydrate stores would be inadequate over this shorter time period.

The majority of research in this area has been carried out on lower intensity endurance exercise. Coggan and Coyle (1989) fed subjects 3g.kg^{-1} CHO (or a placebo) 30 minutes before the anticipated time of fatigue at 70 % $\dot{V}O_2$max, and showed an increase in endurance time by 21% following CHO ingestion. It is unlikely that any glycogen sparing would have occurred up to the point of ingestion as both groups were fed only water up until CHO or placebo ingestion. This confirms the findings of Bergstrom and Hultman (1967) who reported a gradual shift from muscle glycogen degradation when these stores were low. They reported a switch to blood borne CHO, which improved performance by enabling continued CHO oxidisation. It is generally believed that the major benefits from CHO feeding occur by blood glucose providing and alternative source of energy for contraction in the latter stages of exercise (Hargreaves and Briggs, 1988). Nassis *et al*. (1998), also indicated that glycogen sparing due to CHO feeding was another possible reason for improved performance during CHO feeding, but data from muscle biopsy studies were

5

not conclusive. Two studies that have found spared muscle glycogen with CHO feeding throughout exercise are Hargreaves *et al.* (1984) and Yaspelkis *et al.* (1993) who both used intermittent protocols for their studies. These authors speculated that during periods of lower intensity, glycogen resynthesis could have occurred in previously recruited fibres (fibres that were utilised during the intense periods of the exercise protocols), (Hargreaves and Briggs, 1988). It is possible therefore that glucose feeding during an intermittent trial could promote the maintenance of muscle glycogen through the resynthesis during recovery periods, in which case there are possible endurance/performance benefits.

2.2 Enzyme activity, muscle structure and function

It has been reported by Coyle *et al.* (1991) that elite class time trial riders in the USA had a higher proportion of slow twitch muscle fibres, compared to lower ranked riders. Increased proportions of slow twitch muscle fibre could potentially be beneficial to the time trial rider. Coyle *et al.* (1991), suggested that those riders with particularly high slow twitch fibre composition would be activating highly oxidative fibre types, which are more efficient during endurance exercise. This would support the findings of Jenkins and Quigley (1990) that endurance athletes use a large proportion of their capacity without lactic acidosis occurring, hinting that they have the 'metabolic machinery' available to prevent acidosis (McArdle *et al.*, 1991).

The laboratory measurement of lactic acidosis during incremental testing has been shown to predict endurance performance in trained cyclists by Davison *et al,* (1997) who reported elevations in B[Lac⁻] at an exercise intensity of $61.4 \pm 3.9\%$ of maximum minute power recorded on a Kingcycle ergometer. This exercise intensity is slightly higher than the 50% of $\dot{V}O_2$max indicated by Spurway, (1992). The observed increases in B[Lac⁻] during incremental exercise are due to metabolic adjustments required to maintain ATP production (Spurway, 1992). It is believed that the appearance of BLac⁻ is due to reductions in PO_2, which results in elevated intramitochondrial levels of NADH in order to maintain the required electron flux (ATP production) (Spurway, 1992). With elevations in intramitochondrial NADH, the concentrations within the cytoplasm also rise as NADH is shuttled across the mitochondrial membrane, subsequently pyruvate is then converted into Lac⁻ in the presence of NADH and lactate dehydrogenase (LDH).

However, the initial and subsequent reductions in PO_2 do not limit ATP production, as ATP production continues to rise as exercise intensity increases. Indeed it has been indicated by Connett *et al.* (1990) that the PO_2 would have to drop to below 10% of the observed PO_2 in maximally working muscle for it to limit ATP production.

A further cause of the rises in B[Lac⁻] has been attributed to the recruitment of type 2B muscle fibres. Activation of 2B muscle fibre is likely to result in the production of Lac⁻ due to the sparse number of mitochondria within this particular type of fibre (Spurway, 1992).

Therefore those riders with particularly high percentages of slow twitch muscle fibres are less likely to produce and accumulate Lac⁻, which is a key determinant of elite performance (Coyle *et al.* 1991) .

Essen *et al.* (1977) reported only slow twitch fibres recruited during a 60 minute trial at 55% $\dot{V}O_2$max. During an intermittent trial (15 seconds 100% $\dot{V}O_2$max, 15 seconds rest), both fast twitch and slow twitch fibres were recruited, consequently only 28% of energy was derived from FFA during this trial, compared to 40% during a continuous trial, indicating a greater reliance on CHO for metabolism. As it appears that resting fibres play no part in glycogen donation to active fibres, thus the types of fibres recruited for a given activity seem likely to be critical due to their specific metabolic capabilities, and fuel utilisation patterns.

For the road race cyclist, it may be beneficial to possess a significant proportion of fast twitch muscle fibre, as it is generally the intense anaerobic efforts where important tactical moves are made in the race. Fatigue, and the ability to continue these types of efforts may be linked to a variety of factors. High intensity efforts have been shown to place large demands upon muscle glycogen stores, with one six-second cycle sprint recording a decrease in muscle glycogen of 14% (Boobis *et al.*, 1982)

According to Essen *et al.* (1977), during very brief intense exercise, the energy is derived from ATP and PCr stores (34%), lactate and myoglobin, (22% and 44%) depending upon training status, muscle groups used and the exact profile of power output.

Hultman *et al.* (1987) list four possible metabolic limitations during intense contraction, with fatigue being linked to:-

1) Decreased pH.

2) Increased Inorganic Phosphate (P_i).

3) Increased Magnesium (Mg^{2+}).

4) Inhibition of sodium (Na^+) and Potassium (K^+) transporting ATPase.

It has been observed that intramuscular pH tends to be lower than blood pH, (Hermansen and Osnes, 1972) and it is low muscle pH that is cause of fatigue rather than blood pH during intense contraction (Brookes, 1987). Decreased pH has been shown to decrease maximal force production when muscle pH falls from 7.0 to 6.2 in electrically stimulated frog skeletal muscle fibres (Robertson and Kerrick, 1979). A pH value of 6.6 has been reported in the muscle fibres extracted from the *quadriceps femoris* immediately following a cycle test to exhaustion in man (Sahlin, 1978).

The hydrolysis of ATP and the release of P_i is also believed to play a part in the fatigue process during intense contraction (Hultman *et al.*, 1987). Increases in P_i have been associated with decreases in tension within the muscle (White and Thorson, 1972).

The above review of factors that have been associated with fatigue during high intensity exercise suggests that they are likely to be the result of insufficient ATP production rather than reduced fuel availability. Burke *et al.* (1977), reported the need for the road race cyclist to cope with periods of high intensity

exercise, during climbing, breaking away, and during sprinting. Previous data has been reported in the literature of this variable pattern during road race cycling (Palmer *et al.*, 1994), using heart rate recordings, and single case study of 'smoothed' data reports by Jeukendrup and VanDieman (1998) using SRM power crank technology. Both of these studies have their limitations due to 'smoothed' data (heart rate or power) possibly not indicating the true demands of competition, to date there has been no power data reported from road racing with a higher resolution of 10 seconds per data point. Therefore the demands placed upon different energy systems is open to speculation.

Chapter Three. Euhydration During Exercise.

Euhydration is the term used when the body contains the required amounts of water and it is distributed to the various water compartments according to their needs (Tortora and Agnostakos, 1988).

Water plays a critical role in bodily functions, dissolving the majority of substrates ingested or produced, it is involved in the transportation of dissolved molecules, temperature regulation, and provides a medium for cellular reactions to occur, water also plays a major role in the shock absorption and cushioning of joints and organs of the body (Pivarnik and Palmer, 1994). Therefore the availability of water is crucial to the intracellular and the extracellular functioning of the human body.

The regulatory control of euhydration is a function of hormonal control (primarily aldosterone and anti diuretic hormone (ADH)), hydrostatic pressures within compartments, and osmotic pressures influenced by the concentrations of electrolyte solutes within the various fluid compartments. According to Pivarnik and Palmer, (1994), exercise, and in particular endurance exercise pushes the regulatory mechanisms to their limits. This chapter aims to review literature investigating endurance events, as well as high intensity exercise, that could give an indication of the implications for the road race cyclist.

3.1 The Movement Of Body Fluids

The major body fluid compartments in the body are the intracellular compartment, the intravascular compartment, and between them the interstitial compartment. Separating the three compartments there are semi-permeable membranes that allow the passage of fluids between them, depending upon the pressures between compartments.

The volumes of the intracellular and the intravascular compartments are kept to within very precise limits (Scanlon and Sanders, 1991). For the intracellular compartment, it is crucial that there is enough water available for essential cellular function, but it must also have sufficient control over the upper limit of water entering the cell to prevent the rupturing of the cell membrane (Carlsson, 1991). The major regulation of the intracellular compartment is controlled by the tonicity of the interstitial compartment (Carlsson, 1991), and under normal resting conditions the intracellular electrolyte concentrations are isotonic (in terms of osmotic forces) with the interstitial compartment.

The intravascular volume must also be regulated to maintain blood pressure within the vascular system, to prevent the pooling of blood, and ensure oxygenation for the extremities via normal circulation. The intravascular compartment must also regulate against high blood pressures, and its associated capillary damage.

The regulatory control mechanism for the vascular system enables the vasodilation/vasoconstriction of small veins and arteries that can increase or decrease the space through which the blood is flowing (Carlsson, 1991). However, this is a limited facility, and to compensate for this, fluid exchange can occur

across the semi permeable membranes of the capillaries, depending upon the requirements of the vascular volume.

Although the volume of the interstitial fluid is not closely regulated, the solute concentrations of this compartment are closely controlled, as its concentration will depend on whether the other compartments have a net gain (or loss) of fluid.

3.2 Exercise and Fluid Shifts

The majority of research has concentrated upon fluids in the vascular volume, due to the difficulty in measuring other fluid compartments within the exercise setting.

At the onset of exercise, capillary dilation occurs as blood pressure increases and the demand increases for the blood supply to the working muscle (Hudlika *et al.*, 1982). According to Senay and Pivarnik (1985), the dilation not only aids in the maintenance of blood pressure but also increases the surface area for diffusion through the capillary wall.

The normal exchange that occurs during exercise is the driving out of fluids from the vascular volume by filtration pressures into the interstitial spaces. According to Greenleaf *et al.* (1977), as exercise commences, the initial fluid losses from this compartment are isotonic to the vascular fluid. The volume of vascular fluid has been shown to decrease proportionally to work load, due to the proportional increase in blood pressure with workload (Pivarnik and Palmer, 1994). However Wilkerson *et al.* (1977) found that this was not the case for all exercise intensities. Exercise intensity up to 65% $\dot{V}O_2$max showed the classic linear reduction in plasma volume, however at intensities of 65% and above, plasma volume losses were elevated and disproportional to the exercise intensity. Wilkerson *et al.* (1977) concluded that this was because 65% $\dot{V}O_2$max was the point at which significant B[Lac⁻] accumulation first occurred. Collins *et al.* (1989) reported that BLac⁻ reductions post exercise mirrored the plasma volume replenishment, and concluded it is possible that BLac⁻ levels are at least part responsible for the decrease in plasma volume. However, during submaximal efforts, with a steady B[Lac⁻], Van Beaumont *et al.* (1981) reported an 8-15% reduction in plasma volume depending upon the 30 minute submaximal cycle intensity (40-70% $\dot{V}O_2$max), and M^{C}Murray (1983), found a 17% decrease in plasma volume during steady state exercise at 70% of maximum heart rate). The majority of plasma volume decrements were seen during the first 10 minutes of exercise, therefore, it is possibly filtration pressures, and muscle lactate levels that exert influence over the removal of fluid out of the vascular space due to initial rises in B[Lac⁻] at the onset of exercise.

Plasma volume losses usually occur at the onset of exercise, after approximately 15 minutes, an equilibrium is met where the pressures in the interstitium matches the capillary pressures (Smith *et al.*, 1976). Greenleaf *et al.* (1977) also reported this initial plasma volume loss during the first 10 minutes of exercise, however they reported a gradual replacement back into the vascular volume for the remaining 50 minutes of a 60 minute effort (although not back up to initial plasma volume concentrations), at exercise intensities corresponding to 23%, 43% and 63% $\dot{V}O_2$max. The initial loss of plasma volume as stated before was

10

isotonic with the vascular volume, but as exercise progressed, Greenleaf *et al.* (1977) reported a gradual hypotonic shift leaving the vascular volume with a greater concentration of solutes remaining. The gradual fluid shift back into the vascular volume could be the reason for some of the equivocal results found during ultra endurance events. If a progressive shift back into the vascular volume occurred, then the difference in plasma volume for pre race and at the end of a 56 km foot race (Irving *et al.*, 1990) may not be statistically significant. Other studies on endurance events have noted significant changes during the early stages of events, Myhre *et al.* (1977) noted that plasma volume changes had occurred after 6 km of a 42.2 km marathon, although Irving *et al.* (1990) concluded that plasma volume remained stable, a progressive return may have occurred. Greenleaf *et al.* (1977) concluded that once the reduction in outward filtration occurred, greater concentrations of solutes, in particular vascular proteins maintained, or attracted fluid back into this compartment.

Although during exercise it has been shown that plasma proteins have increased in number (Maughan, 1985), decreased in number (Harrison *et al.*, 1975), and remained the same (Ekblom *et al.*, 1970) it is generally accepted that protein concentrations in the vascular volume are negatively correlated to the volume of fluid that leaves this compartment, and the equivocal findings in the above studies may be due to variations in the dehydration status of subjects, the subject training status, and the temperature of the exercise setting. According to Senay *et al.* (1977) the route taken by the interstitial proteins into the vascular volume is via the lymphatic system. Senay and Pivarnik (1985) reported increased lymph flow during exercise, which with the massaging action of the muscles aided the passage of protein, and in particular albumin (Maughan, 1985) into the vascular space. The increased protein concentration of the vascular space will aid in attracting water back into the vascular volume, and will also enhance the buffering capabilities of the blood (Astrand and Rodahl, 1986).

During maximal cycling exercise, plasma volume has been shown to decrease, Poortmans, (1971) 11.9%, Wilkerson *et al.* (1977) 14%, Senay (1980) 15-18%, Van Beaumont *et al.* (1981) 15.6%, although again equivocal results for changes in plasma protein concentrations have been reported, Poortmans (1971) reported decreased concentrations during steady state exercise.

Depending upon the intensity of the exercise, the ambient temperatures and the dehydration status of subjects, it appears that plasma volume decreases during exercise. Once the fluid has left the vascular space it enters the interstitial compartment, diluting the solutes within this compartment. As the fluid initially does not appear to move back into the vascular volume due to filtration pressures, some fluid will move via osmosis into the intracellular compartment (Sjogaard and Saltin, 1982), with the increase in B[Lac$^-$] this will increase the osmotic forces aiding the flow of water to this compartment. Sjogaard and Saltin (1982) extracted fluid samples from the muscle cells of subjects during exercise, their results showed that the decrease in plasma volume was almost fully accounted for by a 15% increase in muscle fluid content if all of the active muscle mass was accounted for, the authors did concede however, that as muscle fibres/groups

were working to different intensities, this could influence the amounts of intracellular fluid expansion. The concentration of other electrolytes within the body fluid compartments is also of importance for the fluid balance during exercise, as with proteins and Lac⁻ they have the ability to draw water between compartments through osmotic pressures. Hence another important feature of fluid balance is the regulation of electrolyte concentrations, which are monitored, and depending upon their concentrations, will conserve or excrete water via the renal system (Scanlon and Sanders, 1991). At rest the three major fluid compartments are isotonic, each compartment contains a similar fluid to solute ratio (Scanlon and Sanders, 1991). Although the solute numbers are the same, the individual electrolyte numbers vary between compartments, although a Cation-Anion balance does exist (Herbert. 1983). The major cation of the intracellular environment is potassium (K^+), and anion phosphate (HPO_4^{-2}). In the extracellular fluid, sodium (Na^+) is the major cation and chloride (Cl^-) is the major anion, the major difference between the interstitial volume and the intravascular compartment is that the vascular volume contains a greater proportion of cations in the form of protein (Scanlon and Sanders, 1991). The major reason for the difference between intra and extracellular concentrations of electrolytes is to maintain the required membrane potential, this is required for nerve impulse transmission, this is achieved via the sodium - potassium ATPase pumps, and a semi permeable membrane. At rest the membrane potential is -88mv (Cunningham *et al.* 1971), depolarisation requires a shift of potassium extracellularly, and sodium intracellularly, reversing the potential and sending the electrical charge throughout the cell. If electrolyte concentrations (intra or extracellularly) are not maintained any further shift of solutes may inhibit the passage of an action potential. As previously mentioned, the solute concentration of the electrolytes will exert osmotic forces, so it is necessary to retain the solutes within the compartments to prevent the loss of fluids. A decrease in electrolyte concentrations will decrease the cell's ability to hold water, and will therefore decrease its ability to perform routine cellular function (Pivarnik and Palmer, 1994).

As exercise will exert further pressures upon the electrolyte balance, with increased nerve and muscle impulse transmission, the buffering of H^+ ions, transportation of glucose across membranes, storage of glycogen (muscle and liver), protein synthesis, elimination of electrolytes in sweat, (Lehninger, 1975), the balance of electrolytes is crucial for exercise continuation.

Research into electrolyte balance and exercise has again centred upon the plasma concentrations for accessible data collection. Greenleaf *et al.* (1977) reported that during 60 minutes (63% $\dot{V}O_2$max) cycling, increased plasma concentrations of Na^+, Cl^- and Ca^{2+} were found, with less change in K^+, and plasma proteins. For longer term endurance events, (Hillier *et al.*, 1985, Irving *et al.*, 1990, Rocker *et al.*, 1989), similar findings have been reported, with increased Na^+, Cl^-, but it appears that K^+ is also increased extracellularly following exhaustive ultra endurance running. Spurway (1992) noted that elevations in plasma K^+ occur following a 'barrage' of electrical activity, and also under conditions where elevated ADP and Pi inhibit Na/K-ATPase. . During exercise, elevations in aldosterone levels (associated with Na^+

12

retention), are believed to increase the renal secretion of K^+, via the blood stream, to the kidneys, which in turn helps maintain the membrane potential. Endurance exercise is thought to increase the extracellular concentrations of K^+, and if concentrations are pushed to their limit, (extracellular concentrations of 8.0 $mEq \cdot l^{-1}$) it inhibits the maintenance of a resting potential across the cell membrane, and is a factor that is believed to have been responsible for fatalities in endurance events (Dennis 1992).

The loss of fluids from the body via sweating occurs due to the heat dissipation requirements of the body. Maughan (1991), reported that the volume of fluid lost from the plasma will depend upon training/acclimatisation status of individuals. Thermal dehydration has been shown to decrease plasma volume by approximately twice the fraction of body fluid loss (Dill and Costill, 1974), this however is a smaller contribution in terms of fluid volume than losses from interstitial and intracellular compartments. Upsetting the fluid balance of the plasma volume (i.e. dehydration) will affect performance. During exercise the blood flow to the muscles must be maintained, there is also a need to remove heat at the skin's surface by convection (Herbert, 1983) thus there is competition for the blood supply, which is less of a problem during euhydration. Therefore maintenance of the plasma volume (and intracellular hydration) is required for peak performance. The benefits of fluid intake has been shown in a thermoneutral environment to enhance the endurance performance of athletes exercising at 70% $\dot{V}O_2$max to volitional exhaustion, (Fallowfield et al., 1994). Interestingly, the authors reported increased glycogen oxidisation, suppressed fat oxidisation, and elevated $B[Lac^-]$, as a consequence of restricted fluid intake. Moquin and Mazzeo (2000) reported elevated epinephrine concentrations during mild dehydration, which has been associated with elevated muscle glycogenolysis (Arnall et al., 1986). These studies further highlight the need for fluid intake for optimum fluid balance.

3.3 Water Addition During Exercise

Apart from the obvious intake of fluid through drinking during events, other activities occur that add fluid to the body. The addition of metabolic water, (formed from hydrogen and oxygen during exercise) has been shown to increase with the intensity of exercise, however, this is offset by respiratory fluid losses which account for losses of approximately 75% of the metabolic water added (Pivarnik and Palmer, 1994), but there is still an addition to the body fluid.

The other addition to the body fluid levels is the addition of water liberated through the splitting of glycogen, approximately 3-4g of water is stored with each gram of glycogen, (Olsson and Saltin, 1970), this again increases in proportion to the intensity of exercise.

3.4 Recovery after Exercise

The shift of fluid is reversed when exercise is ended, where filtration pressures were initially increased by the raised hydrostatic pressure, the drop in filtration pressures that accompanies the cessation of exercise will allow the filtration of fluid back into the vascular volume (Irving et al., 1990). The osmotic forces will also act in a manner that will raise the plasma volume. As the plasma fluid loss was hypotonic to the

13

concentration of the vascular volume the solute concentration of the blood would have been raised during exercise, and if an influx of proteins into the vascular space occurs, then the ability to attract volumes of fluids back into the vascular volume will be increased by osmotic forces (Irving *et al.*, 1990).

During a longer recovery period, the plasma volume has been shown to expand, Irving *et al.* (1990) showed a progressive plasma volume increase 1-3 days after exercise, (peak 12.5%), this was attributed to an increased protein concentration and concluded that muscle damage was responsible for the increased vascular protein concentration). Brouns *et al.* (1989) also found significant plasma volume increases during rest days of a 'Tour de France' simulation study. Kargotich *et al.* (1998) indicated that plasma volume expansion post exercise were beneficial, as the expansion increases stroke volume, increases thermoregulatory efficiency, and decreases blood viscosity.

3.5 Fluid Balance During a Road Race Effort

No study has been carried out looking specifically at fluid balance during a road race type effort. However, using the current literature available, it is possible to make a few assumptions about the fluid movement during this type of exercise.

The intensity varies during road race cycling, previous simulations by Palmer *et al.* 1997, Schabort *et al.* (1998) and Brouns *et al.* (1990) have recognised this in their laboratory road race simulation protocols. They speculated that these increased bursts might utilise more glycogen, therefore the metabolic water released might be greater during the road race cycling compared to steady state cycling.

The filtration pressures out of the vascular volume are influenced by hydrostatic pressure, it has been shown by Palmer *et al.* (1994) that the road race type effort involves fluctuations in heart rate response. This would indicate a possible rise in filtration during the high intensity efforts, and a reduction in filtration and vascular return of fluids during the recovery/less intense periods of the race. During steady state cycling (Duration 1 hour), Greanleaf *et al.* (1977) showed an initial decrease in plasma volume, followed by a trend of repletion of this volume, in their study, the majority of depletion occurred during the first 10 minutes of exercise, and the degree of depletion has been shown to increase as the intensity rises (Wilkerson *et al.*, 1977). During a road race changes in intensity could be a limiting step via the control and the adjustment of the vascular volume during the switch from low to higher intensity exercise, and likewise the return to lower intensity exercise.

Intracellularly the build up of Lac^- needs also to be addressed, during the brief periods of intense effort, H^+ build up will occur, which will be buffered by electrolytes, the build up of Lac^- will increase the osmolality of the intracellular environment, which will increase the osmotic pressure to take fluid into the cell, and the removal of lactate from the cell.

Fluid intake has been shown to influence substrate metabolism during exercise (Fallowfield *et al.*, 1994). This is of considerable importance to the road race cyclist, with large demands upon glycogen stores

14

(Palmer *et al.*, 1997). Fallowfield *et al.* (1994) reported glycogen sparing (both liver and muscle) due to fluid intake, making glycogen available for the latter stages of exercise.

Chapter Four. Ammonia Accumulation During Exercise

The aim of this chapter is to outline the pathways of ammonia (NH_3) production, the causes of accumulation during exercise, and the possible consequences of NH_3 accumulation and relate them to previous findings to the current literature on road race cycling.

Ammonia can be formed either through the break down of amino acids as fuel for exercise, or through the purine nucleotide cycle.

4.1 Protein utilisation

During exercise the substrates for muscle contraction are primarily fat and carbohydrate. Many investigators have shown a relatively small contribution of protein to the supply of energy to the working muscle, which has previously been assumed to play a minor role in energy provision (Astrand and Rodahl, 1986). Although relative to the energy derived from fat and carbohydrate the supply of ATP from protein catabolism may be lower, during endurance exercise where substrate is potentially a limiting factor, the supply of 5-10% (Lemon and Mullin, 1980) of energy from protein could have a significant effect on performance. However, it is not only the relatively small utilisation of protein which is implicated with possible performance outcomes, but the associated metabolic bi-products that have been associated with fatigue (Brouns *et al.*, 1990).

4.1.1 The Source of Protein Utilised during Exercise

Dohm *et al.* (1987) reported that by monitoring markers of protein catabolism it appears that it is non-contractile protein that is available for substrate for muscle contraction, and contractile proteins are not utilised as substrate during exercise. It appears that liver and intramuscular protein sources provide the amino acids that are degraded during exercise. Kasperek *et al.* (1982) reported that during 2-3 hours of treadmill running, a reduction in overall liver protein of 25% occurred in rodents.

As the intramuscular free pool of amino acids is very small when compared to the large efflux of amino acids from the muscle during exercise, this provides evidence that intramuscular protein sources contribute to protein utilisation during muscle contraction. As much as 75% of the total pool of free amino acids are found intramuscularly, however this pool is relatively small and can only account for approximately 1% of protein catabolised during exercise. Therefore endogenous protein breakdown must largely account for the increased amino acid utilisation during exercise.

Research has raised questions about the role and supply of amino acids to working muscles, and especially the branched chain amino acids (Leucine, isoleucine, valine,) which are the predominant group of amino acids that can be oxidised by skeletal muscle, (Graham *et al.*, 1995). The branched chain amino acids (BCAA) supply tricarboxylic acid cycle (TCA cycle) intermediates acetyl CoA (from leucine) and Succinyl CoA (from valine and isoleucine). However, the pathway of BCAA oxidisation by skeletal muscle has NH_3 as a potential bi-product.

16

4.1.1.1 Branched Chain Amino Acid Metabolism

During exercise the BCAA's are released from the splanchic bed and are transported via the bloodstream to the working muscles. The working muscles transaminate the BCAA to glutamate and can be converted further to alanine, aspartate or glutamine. Deamination is also a fate of glutamate with the production of NH_3 in the working muscle (figure 4.1).

Figure 4.2 (From Graham *et al.*, 1995) shows the primary pathway of deamination of BCAA in more detail.

BCAA's are supplied either from the small intramuscular pool or delivered via the blood supply to the working muscle. Coupled with alpha ketogluterate, the NH_3 group from the BCAA is converted into glutamate and branched chain oxo acid (BCOA) in the presence of branched chain amino acid amino transferase (BCAAT). BCOA can efflux out of the cell for oxidation in the liver, however the predominant fate is the degradation of the carbon skeleton by branched chain oxo acid dehydrogenase (BCOADH) into TCA intermediates. In the presence of Glutamate dehydrogenase (GDH), glutamate can be deaminated with the release of NH_3 and alpha ketogluterate. In the presence of alanine aminotransferase, glutamate can also be converted into alanine, and likewise in the presence of aspartate aminotransferase, and glutamine aminotransferase, aspartate and glutamine are produced (Figure 4.1). As GDH and BCOADH are both located within the mitochondria, oxidative slow twitch muscle fibres with the highest mitochondrial content have the greatest potential to oxidise BCAA's.

Figure 4.1- Schematic representation of transaminase and transdeaminase reactions within skeletal muscle.

17
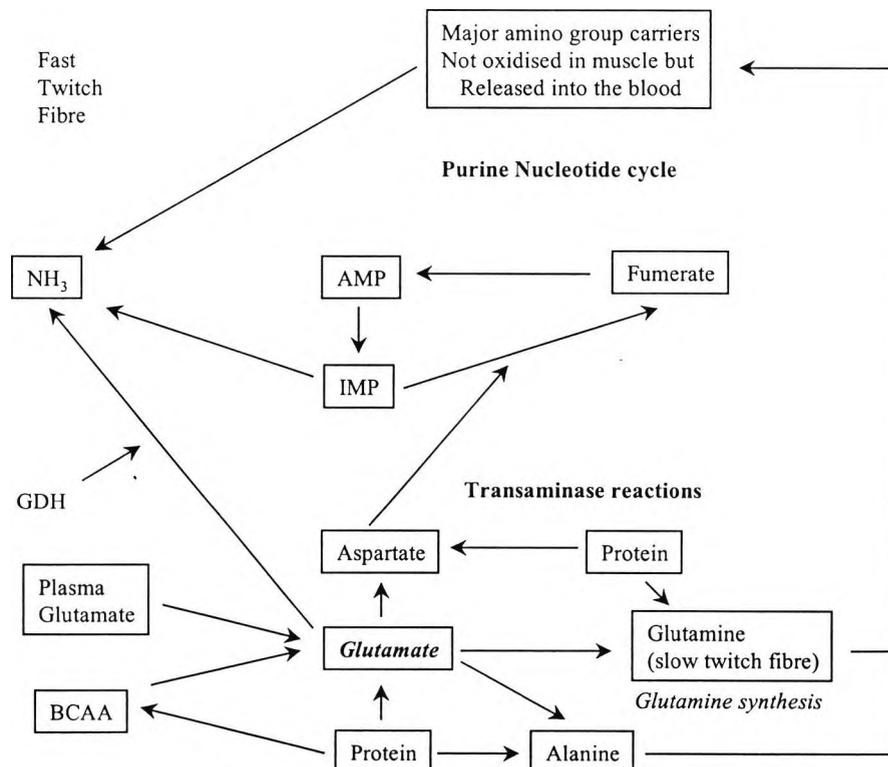
Figure 4.2 – Transamination and transdeamination of BCAA
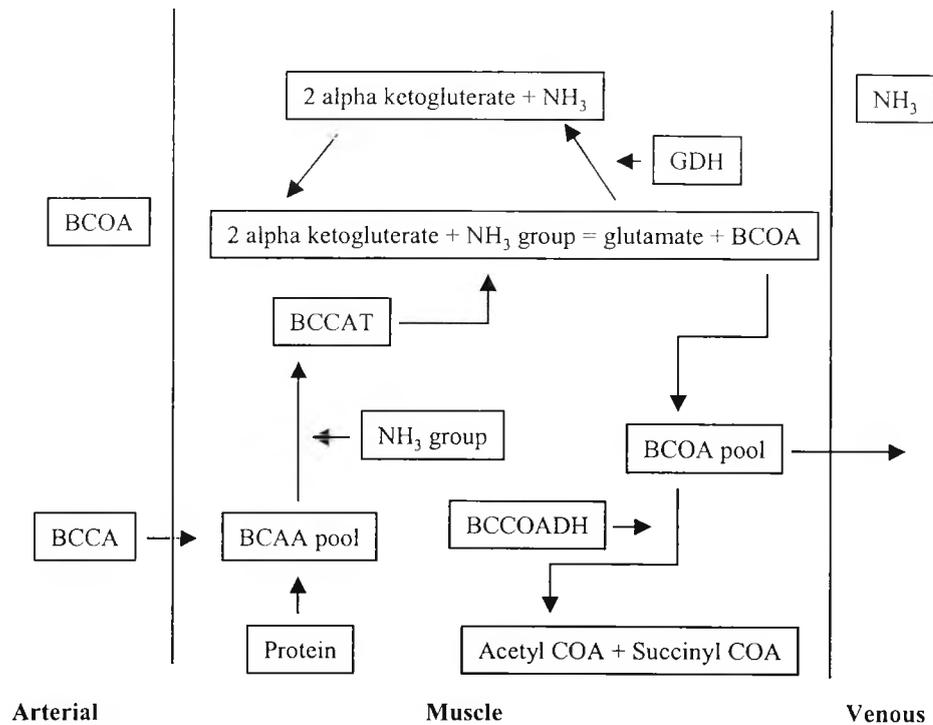
4.1.1.2 Bi-Products of BCAA Metabolism

The fate of the carbon skeleton is oxidisation through the TCA cycle, the amino groups released however, have different fates.

4.1.1.2.1 Aspartate

Aspartate is an essential component of the purine nucleotide cycle (PNC). (Figure 4.3). The principle role of the PNC is to prevent excessive accumulation of AMP. Under conditions where ATP cannot be resynthesised by aerobic respiration, there is an accumulation of ADP. In the presence of adenylate kinase, some of the ADP is used to form ATP and AMP. AMP can be degraded down into Inosine monophosphate (IMP) and NH_3. If aspartate is available, it will convert IMP into adenylosuccinate. In the conversion of adenylosuccinate to AMP, fumarate is produced, which is used to replenish the TCA cycle.

If aspartate is not available, IMP is converted into inosine and eventually hypoxanthine, which will have the consequence of net purine nucleotide loss from the cell. As *de novo* nucleotide synthesis is a time consuming energetic process, (Graham *et al.*, 1995), maintaining nucleotide status could possibly reduce energetic availability within the cell.

Previous road cycling simulations by Brouns *et al.* (1990) and Palmer *et al.* (1997) have simulated race intensities to vary between 30 and 90% of maximum aerobic power. Aside from the acceleration required

18

to match higher aerobic workloads, the role of IMP accumulation and removal may not be an issue to consider. The Brouns *et al.* (1990), and Palmer *et al.* (1997) papers studied road cycling for 2+ hours in duration, and it may be that the mechanism of the purine nucleotide cycle has a more predominant role in preserving adenosine nucleotide status in shorter, more intense events.

Figure 4.3 – The purine nucleotide cycle
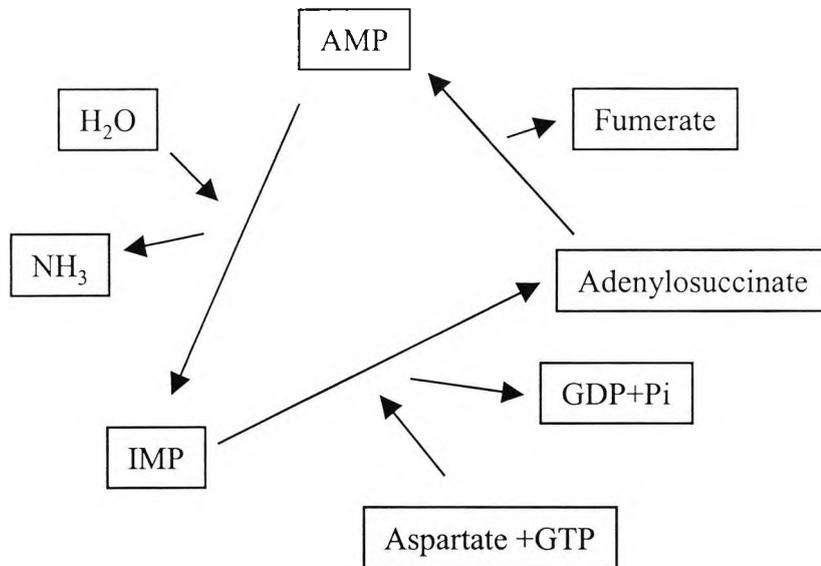
4.1.1.2.2 Glutamine

Both glutamine and alanine are amino carriers that leave the muscle cell for deamination in the liver and gut. Essentially glutamine and alanine are temporary stores of nitrogen with reduced toxicity compared to NH_3 (Houston, 1995). Glutamine is formed if glutamate is not oxidised within the muscle. At rest glutamine is released from the muscle, Eriksson *et al.* (1985) reported resting plasma concentrations of 538 $\mu mol.l^{-1}$ in healthy subjects. During three 15 minute bouts of cycling they also reported a near linear increase with exercise intensity measured up to 80% $\dot{V}O_2max$ (Figure 4.4). Upon deamination of glutamine, the TCA cycle intermediate alpha ketoglutaric acid is formed. Glutamine formation is believed to play a role in minimising the formation of NH_3 within the cell, preventing a toxic accumulation, and allowing transport to the liver for removal.

This raises the issue of possible differences in limitation depending upon the duration of races. The ability to continually supply TCA intermediates without the NH_3 accumulation in the muscle could be implicated in fatigue. Longer races where energy substrate availability may become taxed may reduce the rate of glutamine release due to an ATP requirement in the process of glutamate deamination. If this occurs there is a possibility that greater NH_3 will be produced with the associated toxicity.

4.1.1.2.3 Alanine

Alanine (and alpha ketogluterate) is formed through the conversion of glutamate in the presence of alanine aminotransferase and pyruvate. Once transported to the liver via the bloodstream it is deaminated forming pyruvic acid. Pyruvic acid is then used as a gluconeogenic precursor, and once converted to glucose, is released by the liver and taken up by the exercising muscle. This is known as the glucose-alanine cycle. Eriksson *et al.* (1985) reported a rise in alanine with an increase in exercise intensity (figure 4.4). Wahren *et al.* (1973) reported that during recovery 9% of the gluconeogenic precursor came from alanine compared to 6% in the resting state. These are similar figures to Hultman (1978) who reported that 8% of the glucose production in the liver is due to alanine, which actually reduces to 1% during heavier workloads. Like glutamine, the release of alanine essentially reduces the volume of NH_3 produced within the muscle, by passing it to the liver for deamination.

Figure 4.4 - Net release of alanine, glutamine and NH_3 at different exercise intensities. (Eriksson *et al.* 1985).
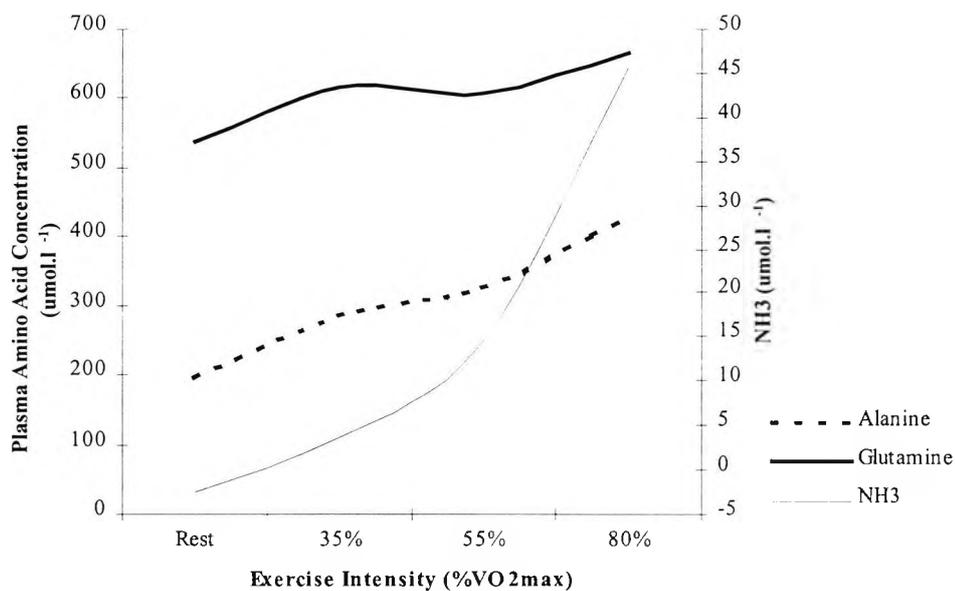
The major role of alanine is not as a precursor for glucose however, its major role is as an amino carrier (Hultman, 1978). Like the formation of glutamine, alanine formation requires energy (in the form of pyruvate). Again if substrate was limited, alanine formation may be reduced causing an increase in NH_3 and toxicity. If NH_3 concentrations are indicative of a lack of mainstream fuel (CHO and fat) for exercise, then an endurance athlete's ability to utilise fat as substrate instead of utilising CHO would be beneficial. Reductions of CHO utilisation have been shown to increase amino acid metabolism (Haralambie and Berg, 1976), if CHO is spared there is a possible sparing of the amino carriers of Nitrogen for the later stages of exercise, possibly reducing the toxicity of NH_3 within the muscle cell.

4.1.1.2.4 Ammonia Release

Ammonia plays a specific role in the cell as a buffer for H^+. The formation of ammonia comes from the non-permeable ion ammonium that has consumed H^+ and can leave the muscle cell via concentration gradients (Graham *et al.* 1997). NH_3 is produced under conditions that activate the purine nucleotide cycle and also BCAA degradation.

The pattern of NH_3 release during exercise depends upon the exercise duration and intensity, which essentially governs the direction of ammonia flow due to the concentration gradient between tissue and blood. Conditions that favour NH_3 release by the purine nucleotide cycle are possibly going to be different to those that favour BCAA breakdown.

The production of NH_3 through the purine nucleotide cycle is regulated by AMPD activity, during exercise AMPD binds to myosin, which maintains the production of ATP under conditions of intense muscle contraction. Activity of AMPD is highest in fast twitch muscle, and is possibly due to the fact that reduced inhibition of AMPD occurs in the presence of H^+, which is more likely to occur in fast twitch fibres compared to the more oxidative slow twitch fibres. Likewise the production of NH_3 through the breakdown of BCAA is likely to be predominantly a slow twitch mitochondrial rich fibre process due to the mitochondrial bound GDH. Pages *et al.* (1994) reported that during high intensity exercise $[NH_3]$ and $B[Lac^-]$ significantly correlated. This is possibly due to activation of the pathway of the purine nucleotide cycle (NH_3 production) and glycolysis being activated during intense muscle contraction. In contrast during endurance exercise they reported no correlation between NH_3 and $B[Lac^-]$, and during a triathlon, decreases in $B[Lac^-]$ over time were reported despite rises in NH_3. Due to reduced acidosis, it is unlikely that activation of fast twitch fibre and purine nucleotide cycling were responsible for the rise in NH_3 therefore the source of production would be likely to come from BCAA degradation. This could also be due to reductions in other N carriers that require energy for formation, leaving toxic NH_3 to leave the cell.

In terms of exercise intensity and endurance performance, Itoh and Ohkowa (1990) reported that during steady state treadmill running at 50, 70, and 100% $\dot{V}O_2max$, and at a supra maximal cycling intensity (Maximal for 60 seconds) there was a correlation between $B[Lac^-]$ and $[NH_3]$. Using all of the $B[Lac^-]$ and $[NH_3]$ across all of the exercise intensities measured, significant correlation coefficients for both sprint athletes (r=0.78), and endurance athletes (r=0.82) were attained. Interestingly there were quite dramatic differences at different individual exercise intensities between the two types of athletes, that possibly reflect their training status/muscle fibre type make up. At the two higher work rates there were weaker correlations for the sprint athletes compared to the endurance athletes, possibly reflecting reductions in NH_3 accumulation with sprint training, whereas maximal $B[Lac^-]$ can increase with sprint training. In the endurance trained athlete the increased anaerobic metabolism appears to have been reflected by increased $B[Lac^-]$ and NH_3. At the 75% maximum heart rate workload (which would be likely to be activating slow twitch fibre), any NH_3 formation from the endurance-trained athlete is possibly coming from BCAA

21

utilisation, with minimal Lac⁻ formation. The sprint athlete with a larger cross section of fast twitch fibre would possibly have to activate these fibres at this lower work rate (although possibly not the lowest work rate), causing small rises in B[Lac⁻] and [NH_3]. In theory the subjects in this study were working at the same relative exercise intensity, however depending upon training status there appears to be differences in not only the plasma concentrations of metabolites, but also the relationship between these metabolites.

Within the literature there are authors who appear to limit their views on NH_3 production to simply being a factor of the purine nucleotide cycle without any mention of BCAA catabolism (e.g. Lutoslawska *et al.*, 1992). This is despite research monitoring endurance exercise and supporting the findings of Pages *et al.* (1994) of uncoupled kinetics between plasma Lac⁻ and NH_3 concentrations. Head *et al.* (1996) is another study that ignores the potential source of NH_3 from BCAA breakdown, they concluded that at an exercise intensity of 55% $\dot{V}O_2$max the purine nucleotide cycle was in action. Using beta blockade of fat as a fuel for exercise (Metoprolol and Propranolol) significant increases in plasma NH_3 were reported (Head *et al.*, 1996). However without isolating the potential source of NH_3 from BCAA breakdown these conclusions have their limitations.

4.2 Ammonia release during endurance exercise.

A methodological concern is that by sampling plasma concentrations the pattern of increase may be simply a reflection of changes in blood flow to the liver for removal of NH_3 and concurrent conversion to urea. Eriksson *et al.* (1985) reported that despite reductions in liver blood flow during exercise there were similar rates of removal during exercise compared to resting values, therefore the rises seen during exercise were representative of NH_3 release by the muscle. Broberg and Saltin (1989) reported release of ammonia from the working muscles was gradually increased in the first few minutes of exercise. Their calculations were based on the arterial venous difference of the working muscle group. Brooks (1987b) reported no increased rate of urea formation during exercise, and Eriksson *et al.* (1985) concluded that small changes in plasma urea during exercise reflected this process, as essentially there was no increase in NH_3 removal for the conversion to urea during exercise. Graham *et al.* (1995) reported resting concentrations of NH_3 to be between 20-60 μmol.l⁻¹. This is due to the ongoing process of degradation and synthesis of skeletal muscle. During endurance exercise concentrations of over 250 μmol.l⁻¹ have been reported in the literature (Brouns *et al.*, 1990), although values of ~180 μmol.l⁻¹ are more typically seen at exhaustion. Brouns *et al.* (1990) have reported the consequences to be a potential instigator of cramp. During endurance exercise one of the major considerations is the availability of substrate to supply energy for repeated muscle contraction. Rises in NH_3 were reported by Brouns *et al.* (1990) at fixed work intensities (70% of power required to achieve $\dot{V}O_2$max), and intermittent intensities (30-90% of power required to achieve $\dot{V}O_2$max). (Figure 4.5). They concluded that due to greater activation of fast twitch muscle fibres during exhaustive endurance exercise the purine nucleotide cycle produced the NH_3 in the latter stages of the trials.

Data from highly trained cyclists reported by Wagenmakers *et al.* (1991) would question Brouns *et al.* (1990) conclusions, as they reported no breakdown of AMP or hypoxanthine in either a carbohydrate depleted or loaded state. It is likely therefore that during steady state endurance exercise the predominant source of NH_3 is likely to come from protein catabolism, with the synthesis of TCA intermediates for oxidisation.

Despite the possible consequences of increased NH_3 formation that have been linked to fatigue, altered central nervous system function, lethargy, convulsions, changes in intracellular pH, and alterations in intra and extra cellular electrolytes (Graham *et al.*, 1995) the specific role and implications for fatigue during exercise are not fully understood. Ahlborn *et al.* (1992) administered NH_3 to rats prior to prolonged submaximal treadmill running, and found a significant dose dependant reduction in time to fatigue in his population, elsewhere however elevations of NH_3 through the administration of BCAA's has produced equivocal results. Madsen *et al.* (1996) and Calders *et al.* (1997) have not shown negative consequences from this procedure. Madsen *et al.* (1996) reported that during laboratory simulated 100km cycling, performance was not significantly altered ($p>0.05$), despite plasma NH_3 levels being significantly higher during the BCAA trial compared to the control trial. Calders *et al.* (1997) reported that during a treadmill run to exhaustion, with increased plasma NH_3 concentrations due to BCAA supplementation, endurance time improved in rats, suggesting NH_3 did not play a role in fatigue, with the task lasting 99 minutes in the BCAA trial and 76 minutes in the control trial.

Figure 4.5 - NH_3 concentrations during intermittent and steady state exercise (Brouns *et al.*, 1990).
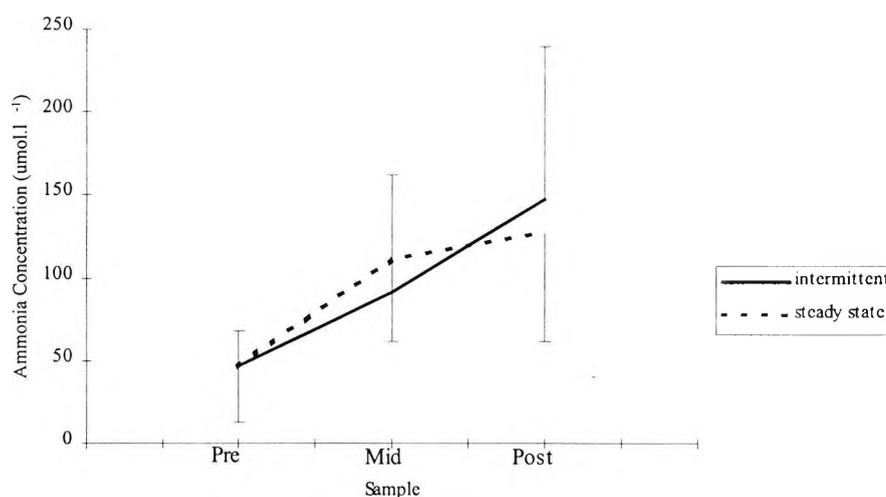
If performance is improved with increased levels of plasma NH_3 during BCAA supplementation, then possibly another explanation is that elevated NH_3 during normal endurance exercise represents depleted substrate as amino carriers. Despite elevations in NH_3, BCAA supplementation will possibly continue to supply intermediates for the TCA cycle, whereas under normal circumstances there is possibly a carbon drain on the TCA cycle intermediates.

4.2.1 The Role of Ammonia in Lactate formation and endurance exercise.

Pages *et al.* (1994) reported that Lac⁻ and NH_3 kinetics were not coupled during endurance exercise, however, this is possibly due to the different processes of NH_3 formation during endurance compared to high intensity exercise. During exercise of increasing intensity the process of both protein catabolism and AMP deamination may be the source of NH_3 released by the muscle. Banister *et al.* (1983) reported that during a ramp test to exhaustion all subjects in their study showed significant rises in NH_3 at 40-50% $\dot{V}O_2$max, and hinted that NH_3 may be a primary toxin in exhaustive exercise. They hypothesised that rapid NH_3 accumulation in the muscle could in theory induce glycolysis via early excessive pyruvate accumulation, as NH_3 affected oxidative pathways. Early pyruvate accumulation would in theory then lead to the formation of Lac⁻. Banister *et al.* (1983) reported NH_3 accumulation at a lower intensity of exercise than had previously been reported. Wilkerson *et al.* (1977) reported significant elevations only above 70% $\dot{V}O_2$max, Meyer (1980) reported increases at intensities above 80% $\dot{V}O_2$max. Lowenstein (1972) indicated that possibly NH_3 interfered with oxidative metabolism at the mitochondrial level, promoting pyruvate and associated B[Lac⁻] accumulation.

In terms of monitoring the physiological markers of endurance training, the onset of blood lactic acid accumulation test is possibly the best predictor of endurance performance, with a rightward shift in the accumulation of BLac⁻ with training (Coyle *et al.* 1991). If NH_3 is implicated in the stimulation of Lac⁻ production then what are the possible reasons behind a rightward shift? Firstly endurance training increases the relative cross sectional area of slow twitch muscle fibres, reducing the size of fast twitch fibres that have been associated with greater NH_3 releases due to increased AMPD activity in fast twitch muscle (Itoh and Ohkuwa, 1990). The development of slow twitch fibres with endurance training may also increase the size and number of mitochondria, essentially reducing the load upon individual mitochondria (Holloszy and Coyle 1984) and reducing the need to rely upon the purine nucleotide cycle to maintain cellular energy charge. Associated with an increased concentration of mitochondria is an increase in the enzymes BCAADH, glutamate dehydrogenase and alanine aminotransferase which have been reported with training (Graham *et al.*, 1985). This opens the possibility of an increase in amino group carriers without the deamination within the muscle, but passed via the bloodstream to the liver. Einspar and Tharp (1989) reported increased plasma alanine concentrations with training after intense exercise. Other authors have speculated that the major role of alanine is to serve as an amino carrier (Felig, 1975), thereby reducing nitrogen concentrations and possibly [Lac⁻] (Holloszy 1975). Wagenmakers *et al.* (1991) reported that the efflux of alanine and glutamine are dependant upon substrate supply within the muscle. Using 'highly' trained subjects (maximum aerobic power 411 watts) a 2-hour intermittent test protocol was employed analysing plasma concentrations of alanine and glutamine, during two trials carbohydrate loaded (CHO_L), and carbohydrate depleted (CHO_D). Due to increased concentrations of plasma glutamine and alanine during CHO_L they concluded that substrate is of 'primary' importance in amino carrier transfer to the liver. This is because alanine requires substrate for formation (pyruvate) and glutamine requires ATP for

formation. Lemon and Mullin (1980) reported that due to the energy requirements to maintain the phospholipid barrier of the cell membrane, reduced substrate availability could promote leakage, resulting in degradation and eventually NH_3 formation. It could also be possible that the pathway of pyruvate to alanine reduced the process of pyruvate conversion to Lac^- (Poortmans 1988). Wagenmakers *et al.* (1991) also reported that two subjects who fatigued early (prior to 2 hours) they had the lowest concentrations of plasma glutamine, possibly associating reduced glutamine levels with fatigue. Raubottom *et al.* (1997) also indicated that glutamine (amino carrier) release may possibly influence performance and reported in trained triathletes that lower glutamine concentrations were indicative of overtraining, and increased performance was seen in this longitudinal study with increased plasma glutamine levels. However Green *et al.* (1991) were unsure whether the drop in NH_3 formation was due to decreased release and/or increased removal rates of NH_3, alanine and glutamine. Alanine and glutamine concentrations have also been studied by Babji *et al.* (1983). During cycling exercise that consisted of 10 minute bouts at 25, 50, 75 and 100% $\dot{V}O_2max$ they reported similarities in the exponential rises of both NH_3 and Lac^- in healthy subjects. However the NH_3 concentrations in the plasma only rose three fold compared to a ten-fold rise in Lac^-. They reported a linear increase in glutamine (similar to the findings of Eriksson *et al.*, 1985) with concentrations rising to 734 $\mu mol.l^{-1}$. Graham and McLean (1998) reported that although glutamine differences were highly variable amongst individuals, the pattern of release into the plasma (calculated from arterial - venous differences) progressively increased with exercise intensity. Babji *et al.* (1983) reported a gentle exponential rise in alanine as work intensity increased, which is a different pattern compared to the linear efflux rate with exercise intensity reported by Eriksson *et al.* (1985).

Therefore at intensities that increase $B[Lac^-]$, amino acid metabolism is also possibly induced, with the requirement of transporting NH_3 out of the cell. Therefore N carrier availability could play a major role if intensity increases in the latter stages of a race, the cyclist requires a buffering capacity to maintain homeostasis.

4.2.2 Ammonia formation during high intensity exercise.

The predominant source of NH_3 during high intensity exercise is likely to come from the purine nucleotide cycle. However there is some evidence that increased protein catabolism occurs during acidosis, and may contribute to the rise in NH_3 during repeated intense muscle contraction (England and Ross-Price, 1995).

Research on athletes with differing backgrounds by Itoh and Ohkuwa (1990) reported significantly higher blood NH_3 concentrations in sprinters compared to endurance athletes. They concluded that athletes with reduced fast twitch muscle fibres and significantly higher mitochondrial content may not posses the NH_3 generating steps from the purine nucleotide cycle. This possible limitation of the endurance athlete may have negative consequences upon competitive performance. For example, decreased ability to produce ATP through the purine nucleotide cycle may inhibit performance if a sprint finish or attacks are required. A lower [AMPD] may also facilitate net loss of adenosine from the muscle. In trained subject groups

participating in intense exercise Balsom *et al.* (1992) also reported net adenosine loss post 15 x 40 metre sprints and during sprints of differing duration. Balsom *et al.* (1992) reported minimal increases in hypoxanthine with the shorter sprints suggesting that the ATP formation was achieved from other sources possibly phosphocreatine availability. Creatine supplementation ($20g.day^{-1}$ for 5 days) has been shown to increase the rates of ADP rephosphorylation, and reduce adenosine deaminase activity, therefore reducing the accumulation of hypoxanthine and IMP, in other words enabling ATP supply to be maintained through an alternate source rather than AMP and IMP formation. The results reported by Balsom *et al.* (1992) where longer sprints generated greater plasma hypoxanthine concentrations led them to conclude the acidosis that occurs during longer sprints requiring the activation of glycolysis may interfere with creatine rephosphorylation resulting in the need for longer recovery periods between longer sprints, and, due to increased rates of fatigue during longer sprints, it is a possibility that the products of AMP degradation possibly influence repeated sprint ability.

Again previous research has not acknowledged the need for intense sprinting during simulated road cycling, however, the requirement to continually change work rates may induce acidosis and affect rates of recovery during variable intensity activity. Bangsbo (1994) reported higher plasma concentrations of NH_3, IMP, hypoxanthine and uric acid during competition. One could hypothesise that during this type of repeated sprint that the removal and disposal of these products could also influence the ability to produce further work of a high intensity nature. Therefore during repeated sprint type activity, recovery periods to remove these bi products could significantly influence the condition that players are in at the end of competition.

Lutoslawska *et al.* (1992) reported that rowing for five minutes at 50, 70, 85% of maximum power (calculated from a 2 km maximal performance test) with rest periods of 5 minutes between sessions, significantly elevated NH_3 over steady state exercise. Lutoslawska concluded that there are different metabolic demands during intermittent exercise, and referenced the findings of Essen *et al.* (1977) who concluded that a greater muscle mass was utilised during an intermittent cycling exercise protocol compared to a steady state condition, possibly due to the increased demands of accelerating up to the desired power outputs, during an intermittent condition.

Exercise of a high intensity nature but with an endurance component has also been shown to produce a significant correlation between $B[Lac^-]$ and plasma NH_3 (r=0.66). Rowing performance (2 km) lasting 405 seconds for male athletes caused significant rises in plasma NH_3 from 44.5 to 215 $\mu mol.l^{-1}$, and $B[Lac^-]$ 1.8 to 15.1 $mmol.l^{-1}$, and according to Lutoslowska (1996) indicative of greater reliance on anaerobic pathways during the test protocol.

This again raises questions about an 'ideal' muscle fibre type, or their training practice to develop an ideal profile. The trade off is between the ability to produce higher powers when sprinting or climbing hills (Burke *et al.*, 1977) with its associated bi product accumulation, verses the less powerful but more oxidative slow twitch muscle fibre.

4.3 Removal of Ammonia

Due to its possible implications for performance, the ability to remove NH_3 from the plasma, convert it to urea in the liver and the remove it in sweat or urine (Lemon, (1987) reported 85% of the nitrogen from protein catabolism is excreted in the urine as urea) could also play a role in reducing the toxicity of this compound during exercise. Again questions need to be asked regarding an actual increase in urea concentration as opposed to apparent rises due to haemoconcentration, or reduced removal rates. Indeed the largest increases in serum urea are found during recovery post exercise. This is thought to be due to a significant reduction in sweating post exercise, resulting in the prevention of urea excretion by this method (Lemon and Mullin 1980). The increases in serum urea that have been reported are relatively small, and is not a major mechanism that operates during exercise, this is reportedly due to relatively small changes in the removal rates of NH_3 during exercise that have been reported by Eriksson *et al.* (1985). Therefore if there is only a small increase in NH_3 uptake during exercise then it is possible that there will only be small increases in the conversion to urea, and the increases seen post exercise may in part be due to both the decrease in sweat rates and the increase in blood flow to the liver post exercise delivering more NH_3 for conversion.

Typically reported resting urea concentrations are in the region of 6 mmol.l^{-1} (Rauwbottom *et al.*, 1997). Some exercise studies however have found significant changes in the urea concentration in both plasma and sweat. Cerny (1975) reported a 20% rise in plasma urea concentration during exercise at 60-65% $\dot{V}O_2$max after one hour in duration. Decombaz (1979) reported a 44% increase in serum urea during a 70-90 km cross-country skiing event, but not all studies have found this. Lemon and Mullin (1980) reported that with increased exercise intensity there is an increase in urea excretion, which is possibly linked to increased sweat rates at higher exercise intensities. Rattu *et al.* (1996) reported a significant reduction in serum urea during a laboratory simulated 25-mile time trial using experienced cyclists as subjects. They also reported that during glucose supplementation there was an even greater reduction in serum urea and concluded that there was less reliance on amino acids during the glucose trial. The absolute reductions in plasma urea concentrations could be due to high sweat rates removing urea. As water supplementation has been shown to significantly affect performance in a one hour endurance time trial, it might be the result of maintaining high sweat production and hence urea removal. Lemon and Mullin (1980) reported findings along similar lines, they reported a more significant rise in urine and sweat urea concentrations in a carbohydrate depleted trial compared to a loaded trial (18.97 vs 10.25 mmol.l^{-1} - a 154.2 fold increase in the depleted state verses 65.6 fold increase in the loaded state). Lemon and Mullin then used this data and calculated the rate of energy derived from protein catabolism during their exercise protocol which consisted of 60 minutes at 75% $\dot{V}O_2$max (10.4% in the depleted condition verses 4.4% in the loaded trial).

Lemon *et al.* (1989) reported increases in serum urea (252% 4.6-11.6 mmol.l^{-1}) during a 62-minute swim at an even paced intensity (~70% $\dot{V}O_2$max). They calculated that decreases in blood flow to the kidneys could only account for the 34% of the rise in serum urea. Therefore the amino acid contribution to energy supply resulted in the observed increase in serum urea. Haralambie and Berg (1976) reported that serum urea

27

increased linearly with exercise duration after an initial plateau for 60-70 minutes. They monitored a variety of athletes competing in running, skiing, marching, or cycling events. The tasks also varied in duration and lasted from 15-765 minutes. They concluded that the rise in serum urea was indicative of liver glycogen depletion and also occurs at a point where muscle glycogen is becoming depleted, with the theory that accelerated protein utilisation will occur at this juncture. Novak (1986) reported findings of an initial rise, an apparent drop followed by a second rise. However the exercise was of 24 hours in duration. Novak (1986) also reported a similar pattern of response of serum uric acid, which represents the oxidisation of hypoxanthine for disposal. Uric acid has been used as a marker of training status by Rauwbottom *et al.* (1997). They reported a negative correlation coefficient between uric acid and performance, during triathlon exercise.

Like Novak (1986), Irving *et al.* (1989) investigated sustained endurance activity of a 24 hour run. They concluded that the increase in urea levels from resting values of 5 $\mu mol.l^{-1}$ to 8-9 $mmol.l^{-1}$ was the result of increased amino acid utilisation as a fuel for gluconeogenisis during exercise. Likewise Farber *et al.* (1987) concluded that inadequate carbohydrate intake during an ironman triathlon (over 9 hours in duration) resulted in increased serum urea in their study.

From these findings CHO supplementation appears to reduce the amount of energy derived from protein during steady state exercise (Lemon and Mullin 1980). However variable intensity exercise could elicit different metabolic demands. Higher intensity bursts may increase sweat rates, which is one of the major elimination pathways for urea, however lower intensity periods could possibly do the reverse. During steady state exercise it has been hypothesised that the rises in urea may simply reflect fuel status within the muscle, however the pattern of urea formation during variable intensity exercise has yet to be fully documented.

Chapter Five. Efficiency during Cycling.

During steady state endurance laboratory 'performance' tests, physiological factors have been shown to correlate with maximum work rate for that given period of time, and have been shown to have a close relationship with field performance (Coyle *et al.,* 1991). However, the energy cost for that sustained work rate, (or efficiency of movement) may also have a bearing upon success in endurance events (Cavanagh and Kram, 1995). In simplified terms, if an athlete has used less energy for the same work accomplished than an opponent with similar energy stores, then discrepancies in endurance performance between two athletes matched with maximum physiological variables could possibly be explained. The aim of this chapter is to analyse the findings from research into the economy of movement, and efficiency during steady state exercise, and attempt to apply it to the demands of variable intensity road race cycling.

5.1 Definitions of Efficiency

Efficiency equations are used to calculate the amount of energy required to perform a given amount of external work. Calculations require steady state exercise for accuracy in gaining accurate calculations of energy expenditure. There are four main calculations that are used widely in the literature, (Cavanagh and Kram, 1985, Coyle *et al.,* 1992) each have their limitations, and affect the final percentage of energy consumed: work done calculation. The most commonly used equation is gross mechanical efficiency (equation 5.1).

Equation 5.1 - Gross mechanical efficiency.

$$\frac{\text{Work Accomplished}}{\text{Energy Expended}} \times 100$$

The calculation of gross mechanical efficiency is the division of work accomplished by total energy cost. Calculation of gross mechanical efficiency according to Cavanagh and Kram (1985), is limited as it fails to account for the energy required to maintain the body at rest (respiration, circulation, ion transport etc.). As this energy requirement is not deducted the figures for gross mechanical efficiency are believed to underestimate the efficiency of movement.Net efficiency calculation subtracts a resting value of energy expenditure (equation 5.2)

Equation 5.2 - Net efficiency

$$\frac{\text{Work Accomplished}}{\begin{array}{c}\text{Energy Expended}\\\text{Above that at Rest}\end{array}} \times 100$$

In analysing this method of calculating efficiency, Cavanagh and Kram, (1985), concluded further deductions need to be made from the energy cost of movement. They reported that the energy cost of movement in an unloaded state has to be taken into account for calculations to be valid. For example a subject can pedal on an unloaded cycle ergometer with the work output being recorded as zero. As this will cause an increase in energy cost over resting values, according to Cavanagh and Kram (1985), it should be taken into account during efficiency calculations. In other words, the expenditure of limb movement with no load must be deducted as a requirement before load is applied to the ergometer. The calculation of work efficiency takes this into account (5.3). Work efficiency specifically calculates the energy cost of overcoming the resistive forces applied by the ergometer, by deducting resting and zero load cycling. However, the calculation of the zero work load is difficult, Gasser and Brookes (1975) tried to gain zero baseline by switching their electronically braked cycle ergometer off, but failed to get a satisfactory baseline measure. If the zero load energy cost figure is wrong then it will distort the results from the loaded cycling protocol. Another reported way to calculate the energy cost of limb movement is to plot a regression line through the data points if it is assumed that the relationship between calorific expenditure and work load is linear (Gaesser and Brookes, 1975).

Equation 5.3 - Work efficiency

$$\frac{\text{Work Accomplished}}{\substack{\text{Energy Expended} \\ \text{Above that in Cycling} \\ \text{Without a load}}} \times 100$$

To overcome the problems of calculating a zero limb movement, the delta efficiency equation, gives a floating baseline measure from the physiological and external energy cost of the exercise (Gaesser and Brookes, 1975). The measurement of increased work accomplished over a previous lower work rate, divided by the increased energy cost minus the previous energy cost at the lower work rate (equation 5.4).

Equation 5.4 - Delta efficiency

$$\frac{\text{Delta Work Accomplished}}{\text{Delta Energy Expended}} \times 100$$

According to Coyle *et al.* (1992) delta efficiency is the most valid estimate of efficiency as the calculation eliminates energy expenditure that does not contribute to the work output.

5.2 Factors Affecting Efficiency

Gross mechanical efficiencies during cycling have been reported to be between 18 and 26% in 19 well trained male competitive cyclists ($\dot{V}O_2$max ~69ml kg^{-1} min^{-1}), (Coyle *et al.*, 1992). However, for a given work rate, factors that raise or lower physiological stress upon an athlete will affect efficiency calculations. The majority of studies into efficiency and cycling have centred their research around the energetic minimum of movement.

5.2.1 Effect of Work Rate

A major criticism of many studies into efficiency and cycling is the low absolute work rates that the subjects are required to sustain for a period of time. Gaesser and Brookes (1975), reported that $\dot{V}O_2$ at higher work rates to be proportionally less than at lower work rates. Therefore gross mechanical efficiency, net efficiency, and work efficiency all increase as the workload increases. They reported that at relatively low work rates between 31-123W there was initially a very rapid rise in gross mechanical efficiency and net efficiency as work rate increased the percentage efficiency began to plateau. These are low values for work rates for trained cyclists, and would probably play a minor role in the outcome of road race cycling. Coast *et al.* (1996) reported gross mechanical efficiency calculations to range from 19.4%, to 21.2% at an exercise intensity of 85% $\dot{V}O_2$max (~240 W). Another major criticism of efficiency studies is that they have not used competitive trained cyclists as subjects for the studies (Davison and Flynn, 1997). Nickleberry and Brooks (1996) concluded that there were no significant differences between competitive and recreational cyclists at work rates from rest to 250 W, for energy input ($\dot{V}O_2$), gross mechanical efficiency, or delta efficiency. If this is the case, and training status is not an important factor determining efficiency, then there is possibly a genetic link determining a cyclist's efficiency during cycling ergometer exercise. Coyle *et al.* (1992) reported a significant correlation between % slow twitch muscle fibre and % gross and delta efficiency, discussed later in this chapter.

If conclusions to be drawn here are that the elite cyclist has no advantage over sub elite riders in terms of energy input then the findings from running exercise studies (intersubject differences of 12-15%) appear not to follow the same trend during cycling exercise. If this is the case, elite cyclists performing at higher work outputs than sub elite riders, must possess mechanisms capable of producing higher forces, and/or are less susceptible to fatigue.

5.2.2 Effect of Pedal Cadence

Pedal cadence has been widely researched in the literature as a factor that can significantly alter physiological stress upon a cyclist under laboratory conditions. Coast *et al.* (1986) reported that a rider could be disadvantaged by 5-10% by selecting the wrong combination of pedal cadence and work rate. Numerous studies analysing pedal cadence in cycling have been performed, with the aim of using efficiency calculation to determine an optimal pedal cadence, as minimal rate of energy expenditure for a given work rate (Sanderson, 1991). Cycling is different from many other sports in that there can be a large difference in

rate of limb movements to create the same external power output (Hagberg *et al.*, 1981), and it appears that there is a possible trade off between efficiency and power output. Trained cyclists typically use pedal cadences in excess of 90 revolutions per minute (rev min^{-1}). Marsh and Martin (1995), reported preferred cadence in experience cyclists to be 95.3 rev min^{-1}, however the literature related to cadence and efficiency has appeared equivocal in terms of physiological and metabolic stress. For the same power output, a cadence of 90 rev min^{-1} has been shown to cause significantly greater rises in ventilatory and heart rate responses than a cadence at 60 rev min^{-1} (Hagan *et al.*, 1992). Suzuki *et al.* (1979), and Jordan *et al.* (1979) reported similar findings in that delta efficiency was maximised at 60 rev min^{-1} versus higher cadences (100-135 rpm).Coast *et al.* (1986) reported an optimal cadence of between 60 and 80 rev min^{-1}, as gross mechanical efficiency calculations above and below that figure produced an inverted U type pattern for physiological responses. Hagberg *et al.* (1981) adopted a slightly different approach to collecting and analysing their data. Using seven highly trained competitive cyclists (mean $\dot{V}O_2$max ~67ml kg^{-1} min^{-1}), Hagberg *et al.* (1981) reported the physiological responses relative to the riders' preferred cycling cadence. The B[Lac⁻] revealed a 'U' shape response with the lowest point equating to ~20 rev min^{-1} lower than preferred cadence. This is a similar finding to Buchanan and Weltman (1985), who found the power output for fixed B[Lac⁻] criteria (4 mmol.l^{-1}) for threshold determination to be higher at 60 rpm than at any of the higher cadences measured (90 and 120 rev min^{-1}) i.e. a shift to the left with higher cadences. Buchanan and Weltman (1985), also reported significantly higher B[Lac⁻] values at 120 rev min^{-1} at work rates above the lactate threshold. However, as $\dot{V}O_2$ values were higher for the same power output, it is feasible that the elevated $\dot{V}O_2$ (relative exercise intensity) would be responsible for the elevated lactate response. The other variables measured followed a similar pattern in that optimal cadence appeared to be below the preferred choice of the riders. Davison and Flynn (1997), using a Kingcycle test rig and trained (maximum minute power 394 ± 45 W) experienced (8.4 ± 6.5 years) cyclists, reported that gross mechanical efficiency was maximised at 90 rev min^{-1}, as opposed to 80, 100 and 120 rev min^{-1} conditions. However, conflicting findings have been reported by Jordan *et al.* (1979) on national/world class cyclists using a Monark ergometer. Their results showed the lowest oxygen cost of cycling on a Monark ergometer occurring at 60 rev min^{-1}, and a 5 % increase in $\dot{V}O_2$ from 85 rev min^{-1} (the preferred cadence of the road time triallists) to 115 rev min^{-1} (the preferred cadence of the track pursuiters). This conflict may be due top the type of ergometer used since the Kingcycle test rig is claimed to mimic the rolling characteristics of a cyclist on the road, by incorporating high inertial loads (Keen *at al.*, 1991).

Merrill and White (1984), offered a possible explanation for competitive riders selecting high cadences due to peer group and coaching pressure, with cadence depending upon cycling discipline (track, road, time trial).These findings have implications for the road race cyclist. As previously mentioned preferred cadence has been documented to be around 95 rev min^{-1} (Marsh and Martin, 1995). This cadence has been shown to result in an elevated B[Lac⁻], indicating increased production, and/or decreased removal, and/or increased

efflux rates. This is also magnified by cyclists having to change cadence with alterations in movement of the peleton, which again would alter B[Lac]. If prolonged periods are spent at inefficient cadences, inducing a higher relative exercise intensity, increased glycogen would be used for glycolitic energy production. However the time spent at 'efficient' and/or 'inefficient' cadences, and mean cadences of road race cyclists have yet to be reported. In the literature changes in B[Lac] have been attributed to changes in blood flow with cadence. Coast et al. (1986) concluded that as higher force contractions have been shown to occlude blood flow, differences in B[Lac] during 60 versus 120 rev min^{-1} conditions could therefore be attributed to the facilitation of venous return by the muscle pump mechanism. This may indicate that the skeletal muscle pump (venous return) is more effective at higher cadences, resulting in increased perfusion (Gotshall et al., 1996). The resulting increase in blood flow may account for an increased washout of Lac (from the muscle into the blood), which could possibly explain the choice of higher cadence amongst competitive cyclists despite a higher oxygen cost of exercise (Gotshall et al., 1996).

5.2.2.1 Pedal Cadence and Muscle Fibre Type

Suzuki et al. (1979) reported that subjects with a high percentage of slow twitch muscle fibre content of the *quadriceps femoris* were more delta efficient at 60 rev min^{-1} compared to 100 rev min^{-1} (23.3 vs 19.6%), and subjects with a higher fast twitch muscle fibre content of the *quadriceps femoris* were more delta efficient at 100 rev min^{-1} compared to 60 rev min^{-1} (25.3 vs 28.8%). The conclusion was that more resistance was offered to cross bridge cycling at 100 rev min^{-1} for the slow twitch group as the shortening velocity was too high, hence using more energy for contraction (Barany, 1967). Suzuki et al. (1979), concluded that as RER values were similar between groups at 100 rev min^{-1} this suggests that the slow twitch fibre group recruited less economical fast twitch fibres, possibly resulting in higher glycogen utilisation. Crowley et al. (1996) reported that due to an increased Lac and epinephrine response, more fast twitch fibres were recruited at 95 vs 55 rev min^{-1}. In contrast to Suzuki (1979) they concluded that increases in delta efficiency at 95 rev min^{-1}, were due to contraction velocities which were better matched to the optimal efficiency of recruited fibres.

Coyle et al. (1991) reported significant differences between % slow twitch muscle fibres of elite and sub-elite riders (66.5 vs 59.9%). However they did not report any findings on pedal cadence during a 40 km laboratory performance trial where subjects were able to alter their pedalling dynamics. Theoretically the elite cyclists would have ridden at a lower cadence maintaining the efficiency of their higher slow twitch fibre content (nearer to the force velocity optimum for slow twitch fibres) than the sub elite cyclist who possessed a higher % fast twitch muscle fibre content. In theory, this could result in a difference in B[Lac], with the sub elite riders recruiting fast twitch fibres with the tendency for higher glycolitic capacity, however no significant differences in B[Lac] were observed during this study (7.1 vs 7.4 mmol.l^{-1} in elite and sub-elite athletes). Coyle et al. (1992) in following up the 1991 study, calculated gross mechanical and delta efficiency during 5 minute work bouts at 50, 60 and 70% $\dot{V}O_2$max. Significant correlations were found for % slow twitch muscle fibre and gross mechanical efficiency (r=0.75, P<0.001) and for % slow twitch muscle fibre and delta efficiency (r=0.85, P<0.001).

33

Takaishi *et al.* (1996) reported similar patterns of metabolic stress during 15 minutes at 85% $\dot{V}O_2$max at cadences from 40-100 rev min^{-1} in collegiate cyclists. Different patterns of response to pedal cadence were found regarding neuromuscular fatigue at different pedal rates, they found for normalised data the minimal values of EMG (muscle recruitment) occurred between 70 and 80 rev min^{-1} for trained and untrained subjects. Increasing the pedal cadence however showed a different response pattern between the two subject groups. Trained cyclists had a much smaller increase of EMG activity at the higher cadences, compared to the untrained subjects who showed a steep rise in EMG activity. Takaishi *et al.* (1996) speculated that the increased EMG activity was in response to a reduction in force production caused by peripheral fatiguing factors, such as Lac$^-$ increase, potassium homeostasis, and changes in calcium removal from the contractile material. Takaishi *et al.* (1996), concluded that experience/training status could affect neuromuscular fatigue, and speculated that the cadence at which minimal EMG may be higher in elite national class cyclists, compared to the riders of 3-4 years experience that were used in their study.

It appears therefore that the muscle fibre type of an individual plays a role in the selection of pedal cadence, their individual efficiency, and consequently their metabolic response to the exercise. This has major implications for the assessment of cyclists in the laboratory, which is discussed later in this chapter.

5.2.2.2 Pedal Cadence and Ratings of Perceived Exertion

Ratings of perceived exertion (RPE) could possibly explain the reasons why cyclists choose to ride at a higher cadence than appears to be physiologically appropriate. The adjustment of cadence is believed to be linked to RPE (Cavanagh and Kram, 1985), and maybe due to muscle fibre type, and possibly work load (Coyle *et al.,* 1991). Perceptions of effort during cycle exercise have been reported to be affected by pedal cadence, however, studies have shown increased RPE with increasing pedal cadence (Coast *et al.*, 1986) with the increased metabolic costs of a fixed external work output. Pivarnik *et al.* (1988) found no significant differences in RPE at 50 or 90 rev min^{-1} however non-cyclists were used in this study. Therefore why do trained cyclists choose a cadence that feels harder than a slower one? The competitive cyclist appears to be riding at pedal cadences above what is believed to be 'efficient'. This could be simply a laboratory phenomenon linked to the methods of testing, and the specificity of exercise. It is possible that ergometer testing is not specific enough to cycling on the road. During road cycling there is a need to match the dynamics of the rider(s) in front to maintain drafting advantages. Therefore, a higher cadence would allow for faster acceleration, shorter time span in the wind and therefore maximising the effectiveness of drafting that would play a dominant role in the outcome of cycle races, McCole *et al.* (1990) reported a 28% decrease in $\dot{V}O_2$ for cyclists drafting behind another rider. Yet this does not explain why a rider who has attacked alone and is not drafting during a road race fails to ride at cadences linked to RPE, and also the time trialist who has no benefits from drafting again chooses a cadence that is above what is believed to be perceived as minimal load. It could be possible that any changes in gradient and or road surface may require significant input by the athlete whilst time trialing in the field. Consequently a higher pedal cadence is required to minimise stress upon the body in the field, maintaining rhythm and speed. The

34

true physiological costs of riding in the field have yet to be documented, and could affect efficiency calculation, which may be the result of differences between riding on a smooth roller/flywheel, compared to undulating rough road surfaces. Research in this area by Marsh and Martin (1998) analysing cadences from 50-110 rev min^{-1} at powers from 75-250W led them to conclude that RPE may not be a critical factor in cadence selection during submaximal cycling.

5.2.3 Effect of Body Mass

A study by Berry *et al.* (1993) reported net $\dot{V}O_2$ and gross $\dot{V}O_2$ data to be positively correlated to body mass, and accordingly net and gross efficiency were found to significantly negatively correlate with body mass during cycle ergometer exercise. This study incorporated a wide variation in body mass to specifically look into this area, and is possibly supported by findings from Wahlund (1948), who found similar $\dot{V}O_2$ values for a group of individuals homogenous in terms of body mass. Berry *et al.* (1993) concluded that the increases in $\dot{V}O_2$ observed may be attributed to an increase in work required to move the legs. The authors concluded that the use of gross and net measures of efficiency were inappropriate, and that delta and work efficiencies would be a more appropriate measure of efficiency due to taking baseline values into account when making the calculations.

5.3 Inefficiency During Cycling

Inefficiency during exercise is the term used to describe wasted effort, or a non productive force produced, which is stressing energy systems further than is theoretically required for a given movement. Patterson and Moreno (1990), studied force effectiveness as a measure of wasted energy during cycling exercise. They revealed that a significant amount of force produced (~25%) during the down stroke of the pedal action was used to overcome negative forces from the trailing leg, they speculated that training may possibly increase motor control, increasing propulsive work output with training status (Faria, 1992). Nickleberry and Brooks (1996), found no significant differences for gross mechanical efficiency or delta efficiency between competitive and non-competitive cyclists. In theory if the untrained subjects were less co-ordinated to the pedal action, compared to the trained cyclists, there would be increased negative forces acting during the pedal stroke, causing the inexperienced cyclist to achieve more work during the propulsive phase of the pedal action, to maintain a given power output measured at the flywheel of an ergometer. In other words, a higher $\dot{V}O_2$ would have been recorded, decreasing efficiency. The competitive subjects used in this study had only 4.3 ±1.2 years experience from which it might be assumed they were not elite cyclists, but were reasonably powerful athletes (75% $\dot{V}O_2$max equated to 258 ±10 watts). Coyle *et al.* (1991) made a comparison of force production during a Lac$^-$ threshold test between elite and sub elite cyclists. They found the negative forces on the upstroke of the pedal action to be similar in both groups, and the performance differences (53.9 ±0.5 vs 60 ±1.1 min/40 km) were attributed to a higher peak torque produced by the elite riders. As gross mechanical efficiency was not significantly different between groups, it indicates that for a given workload the energy cost was similar. As the elite subjects were able to sustain a higher work rate

and a higher ratio of propulsive to negative forces, you would expect a higher $\dot{V}O_2$submax value, however, the propulsive forces acting on the cranks were not measured during the performance ride, and any increase in negative forces, may require increases in propulsive forces to maintain a power output on an ergometer measuring flywheel velocity. Sanderson (1991), concluded that experienced cyclists (USA Cat II) had similar characteristics to recreational cyclists for index of effectiveness (proportion of overall force perpendicular to the cranks), but relatively low work rates (100-235 watts) may have failed to make any distinction between subject groups. Sanderson (1991) did report however a reduction in index of effectiveness as cadence increased from 60-100 rpm. During 70 minute laboratory simulated time trial rides it has been shown there is a steady increase in $\dot{V}O_2$, $\dot{V}E$, B[Lac$^-$] and heart rate (Balmer et al., 1998). Despite no increases in power output, it appears that subjects are under considerably more physiological stress in the latter stages due to the upward drift in these variables. It is speculation, but it is possible that due to fatigue there may be a gradual increase in negative forces acting on the crank, making the rider work harder to maintain the work output measured at the rear wheel of the bicycle. However according to Patterson and Moreno (1990), ineffective force production that increased as pedal cadence increased from 60-120 rpm, resulted in relatively small increases in metabolic cost to compensate for the non quantifiable work output. Nevertheless, the effects on muscle fatigue of forces acting against the pedal crank motion have yet to be documented, especially with regard to endurance performance. Hagan et al. (1992) concluded that during prolonged exercise the drift upwards in $\dot{V}O_2$ at exercise intensities over 50% $\dot{V}O_2$max was a result of increased muscle fibre recruitment, after estimating 13-15% of the drift could be attributed to increased core temperature, and 6-15% could be attributed to the increase in the O_2 demand of the respiratory muscle whose work rate increased due to increases in ventilation. Supporting their findings were Poole et al. (1991) who found 86% of the rise in $\dot{V}O_2$ could be accounted by an increased leg O_2 uptake. They also concluded due to a reduction of B[Lac$^-$] with time, the drift up in $\dot{V}O_2$ could not be attributed to the B[Lac$^-$].

5.4 Implications for the Physiological Assessment of Cyclists

These findings have implications for the laboratory testing of cyclists. Many protocols have used fixed criteria for pedal cadence (e.g. 60 rpm) to determine fatigue at maximal or submaximal exercise protocols which have remained popular as they enable relatively unfit subjects to participate in exercise testing (Pivarnik et al., 1988). Disparity between performance results and laboratory tests could therefore be explained by 'inefficient' cadences chosen by experimenters. Cadence appears to be an individual phenomenon, and any cadence chosen as a set protocol will cause inefficient losses amongst at least some subjects. In theory this may cause elevations in physiological responses, which might be interpreted to suggest weaker performance in the field, and disparity between laboratory and field performance. There may also be differences in the process of loading upon muscles during ergometer testing as opposed to track or road cycling, and possibly variations between ergometer types (Kenny et al., 1995), which again could

influence physiological responses to submaximal exercise, and altering efficiency calculations between laboratories.

In terms of laboratory simulation of cycling, the cyclist should be able to manipulate cadence as if they were cycling on the road or in competition. Their behaviour in the peleton should be matched, so that the physiological responses recorded can be applied to their actual race behaviour. The reduction in pedal cadence that has been observed in the field (Davison and Coleman unpublished observation) could possibly be a factor that may alter performance. A laboratory protocol that attempts to simulate cycling using a fixed cadence criterion, may fail to match the physiological stress a competitive cyclist endures in the field, reducing the applicability of findings to the competitive cyclist.

A further point to consider when interpreting laboratory based efficiency data is the temperature and humidity of the environment in which the cyclists are participating. According to Daniels (1985), these factors will have a significant effect on the efficiency of movement. Increased energy cost of circulation, sweating, and ventilation under hyperthermal conditions will reduce efficiency as the work accomplished will remain unchanged. This again could be an influencing factor for variation of inter and intra subject differences, and cause variations in reported efficiencies between laboratories.

Chapter Six. Physiological Characteristics of Road Racing Cyclists

During steady state cycling, the energy supply depends upon and individual's aerobic capacity, their maximal aerobic power, the proportion of this which is sustainable, and an individual's mechanical efficiency (Olds, 1992). Olds (1992), speculated that with alterations in race duration, and speed changes, the relative importance of different factors may change. For the road cyclist, with events that last for a minimum of 45 minutes, riders are possibly going to require different attributes to the track sprinter (whose performance lasts ~10-11 seconds), however very little literature has been published on the road race cyclist.

6.1 Aerobic Power of Road Race Cyclists

Previous studies that have simulated road race cycling have focused on using exercise intensities relative to aerobic power, with intensities ranging from 30-90% of maximum power output derived from a $\dot{V}O_2max$ test (Brouns *et al.*, 1990, Palmer *et al.*, 1997). Therefore highly oxidative muscle fibres would be a potential advantage, along with the ability to produce high power outputs without the build up of BLac⁻. The oxidative nature of road cycling is further highlighted by a recent publication in the popular cycling press (Cycling Plus, August 1998) which showed a mean work output of 269 watts for a ride of nearly seven hours in duration (figure 6.1).

Figure 6.1 SRM data published in the popular cycling press from the Amstel Gold World cup race; The SRM system was recording the work output of race winner Bjarne Riis
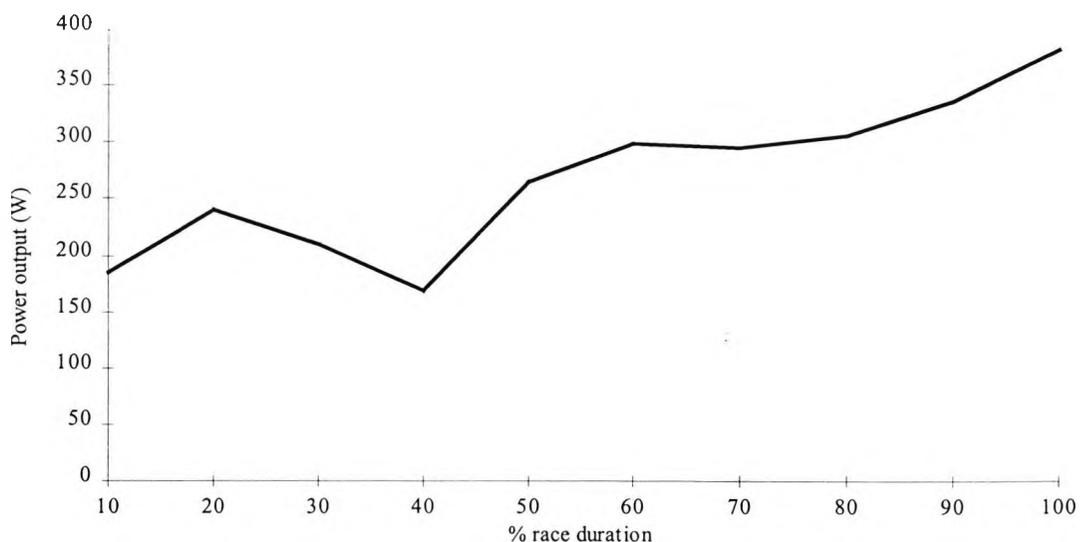
Values reported for $\dot{V}O_2max$ in elite road race riders were reported by Vrijens *et al.* (1982), with Belgium professional riders attaining figures of 4.71 lmin⁻¹ (65.7 ml kg⁻¹ min⁻¹). Faria *et al.* (1989), reported previous studies findings of values of 77.4 ml kg⁻¹ min⁻¹ in British elite riders and 67.6 ml kg⁻¹ min⁻¹ in Dutch elite

riders (in Bonjer, 1979). Brennan *et al.* (1994) reported Kingcycle maximal minute power figures (MMP) for three groups of riders, including the Northern Ireland commonwealth games squad (Table 6.1). The testing protocol for maximal minute power is described in 7.2.2.1. Previous research by Keen *et al.* (1991), reported a significant relationship between $\dot{V}O_2$max and MMP output achieved on the Kingcycle rig. Table 6.1 below is the predicted $\dot{V}O_2$max values from the Brennan *et al.* (1994) study. The Brennan *et al.* (1994) findings of a higher predicted $\dot{V}O_2$max in Northern Irelands' elite versus sub elite development riders is a similar finding to Malhotra *et al.* (1985), who reported that in Indian cyclists who attained higher absolute ($1\,min^{-1}$) and relative to body mass ($ml\,kg^{-1}\,min^{-1}$) values were the selected riders for international duty. Selection was gained through previous competition results with the laboratory tests conducted after selection decisions were made.

Table 6.1 Maximum minute power values and predicted $\dot{V}O_2$max values from (Brennan *et al.* 1994).

Rider Status	MMP (W)	MMP·kg^{-1} (W·kg^{-1})	Predicted $\dot{V}O_2$max (l·min^{-1})	Predicted $\dot{V}O_2$max (ml·kg^{-1}·min^{-1})
Commonwealth Games Squad	486	7.09	5.376	78.4
Development Squad	455	6.22	5.085	69.5
Club Level	423	6.11	4.733	69.2

Sjogaard (1984), reported $\dot{V}O_2$max values again in professional continental riders of 4.96 $l\,min^{-1}$ or 71 $ml\,kg^{-1}\,min^{-1}$, compared to riders competing at a lower level of 3.79 $l\,min^{-1}$ and 56 $ml\,kg^{-1}\,min^{-1}$. Pfeiffer *et al.* (1993) reported the potential dangers of using absolute laboratory markers as performance indicators during road race cycling. This was because team tactics, crashes, and differing terrain will all influence finishing positions, and are not taken into consideration with laboratory testing.

When the demands during cycling change from lower power outputs to the higher power outputs, O_2 kinetics may also play a major role in a rider's ability during road cycling. Craig *et al.* (1993) reported in track cycling (4000 metres pursuit) not only is there a requirement for a high $\dot{V}O_2$max, but also the ability to achieve it quickly as this could also significantly influence results. This has been speculated by Olds (1992), who stated that during shorter distance events it is not necessarily the $\dot{V}O_2$max that is important, but the anaerobic capacity and $\dot{V}O_2$ kinetics that play a large role. Fast $\dot{V}O_2$ kinetics could cause a reduction in the reliance upon anaerobic sources during the initial stages of exercise, sparing muscle fuel, and reducing acidosis within the muscle. To produce high forces, and to continually change work rates in a variable fashion (Palmer *et al.*, 1994), the reliance on anaerobic pathways may be essential. Therefore a high $\dot{V}O_2$max may not indicate the true performance potential of an individual. However the anaerobic

component of the sport has not been emphasised during the previous attempts to simulate the sport in the laboratory. This is possibly due to the comparison of heart rate recordings in the field to a heart rate and power regression equation derived from testing in the laboratory. Using this method no exercise intensity will exceed maximum heart rate and hence $\dot{V}O_2$max. The recording of power during the field on one rider during a mountain stage (6.5 hours in duration) of the Tour de France by Jeukendrup and VanDieman (1998) again did not highlight the importance of 'anaerobic' powers, but the smoothed data (each data point consisting of a mean power of the previous 60 seconds) did have data points in excess of 400 watts for sustained periods. In the same paper Jeukendrup and VanDieman (1998) also showed a power trace of a 170 km race smoothed over 10 seconds was a lot more variable than the heart rate trace, with data points in excess of 700 watts. As the SRM system is a relatively new tool in the field of cycling there is little data published on actual field cycling, and no studies to date have reported further resolution (1 second sampling is available using the SRM system) than the Jeukendrup and VanDieman (1998) paper during competitive cycling events of any ranking. Therefore assumptions about the relative importance of the anaerobic and aerobic systems and their contribution to successful road race performance appear in conflict from the sport science simulations, compared to the coaching perspective of individuals working in the sport.

During steady state exercise at the previously simulated intensities (Brouns et al., 1990), exercising at 90% $\dot{V}O_2$max requires a significant contribution from the anaerobic system. Potteiger et al. (1995) concluded that although this intensity of exercise relies heavily on aerobic indices, there are significantly elevated levels of BLac⁻ resulting in decreased muscle and blood pH. Burke et al. (1977) hypothesised that due to competitive road cyclists requiring oxidative potential for long periods and the ability to break away, climb and sprint, a mixture of 50% slow twitch, and 50% fast twitch muscle fibre would be ideal for both the sprinting and endurance requirements in the sport. Evidence from elite professional road riders suggests this is not the case, and, as reported previously, higher values for $\dot{V}O_2$max in professional riders indicates that a high oxidative capacity is essential, therefore even in athletes that compete in variable intensity exercise a high percentage of slow twitch muscle fibre is essential. Sjogaard (1984), reported that for elite riders 55-85% of muscle fibres were slow twitch (compared to 15-75% in riders of a lower category), they also reported higher capillary density in elite riders, per cross sectional area (509 vs 380 capillaries per mm²), and per fibre (3.3 vs 2.1 capillaries per fibre), compared to lower category riders. Sjogaard (1984), measured the 'aerobic potential' of these athletes using the ratio of citrate synthase:lactate dehydrogenase as a marker. She reported that as the cycling season progressed, (where emphasis is upon racing and recovery, and having to do less 'training') aerobic potential increased through a reduction in lactate dehydrogenase and an increase in citrate synthase activity.

6.1.1 Oxygen Consumption and Relative Exercise Intensity

Previous studies have used maximal 'aerobic' power (watts), and $\dot{V}O_2$max as parameters to establish relative exercise intensity during laboratory tests (e.g. Coggan and Coyle, 1989). Keen et al. (1991) reported a significant relationship between maximum minute power on a continuous ramp test to volitional

exhaustion, and $\dot{V}O_2$max in a heterogeneous population of cyclists (r=0.98). Likewise using a slightly different protocol (25 watts increases in work rate every 150 seconds), Hawley and Noakes, (1992) reported a significant correlation coefficient r= 0.97 (p<0.0001) between these two variables in a heterogeneous population of cyclists. Hawley and Noakes (1992) acknowledged that they were unsure if this relationship would hold for a homogenous population of racing cyclists. There has been no data published on a more homogenous group of racing cyclists from these authors. However Coyle *et al.* (1992) reported that elite riders and sub elite riders had no differences in $\dot{V}O_2$max, yet their sustainable percentage of $\dot{V}O_2$max, and mechanical power output were significantly different, resulting in the performance differences. Loftin and Warren (1994) reported $\dot{V}O_2$max to be a poor predictor of 16 km performance (r=-0.31), and concluded that markers at the ventilatory threshold (absolute $\dot{V}O_2$ at ventilatory threshold, and percentage of $\dot{V}O_2$max) were better predictors than absolute $\dot{V}O_2$max. Coyle (1998) has since concluded that to normalise exercise intensity, the use of the Lac⁻ threshold should be employed rather than a fixed percentage of $\dot{V}O_2$max during assessment of endurance exercise. These findings are echoed by Bishop *et al.* (1998) who suggested that the reason 'lactate threshold' markers correlated to endurance performance was because lactate simply reflected a rate of muscle glycogen use, therefore in theory, speed or power (at lactate threshold) would correlate to endurance performance (rate of fuel utilisation). The findings by Coyle *et al.* (1991) and Loftin and Warren (1994), are not supported by the findings of Dobbins *et al.* (1998) who reported strong correlations between performance and measures of $\dot{V}O_2$peak and maximal minute power at $\dot{V}O_2$peak. This is possibly due to differences in the $\dot{V}O_2$peak testing protocols. The Dobbins (1998), studies used the Kingcycle test rig for their data collection for both performance and maximal testing protocols. Bishop *et al.* (1998) also reported a significant correlation (p<0.001) between maximal values and performance. If the conclusions by Bishop *et al.* (1998) (performance prediction from lactate threshold tests simply reflect a rate of glycogen use), are taken into consideration with the Dobbins (1998) study, where Kingcycle maximum minute power was a significant performance predictor, the two would be linked theoretically. Previous studies have used percentages of Kingcycle maximum minute power to 'normalise' relative exercise intensity (Passfield *et al.*, 1995) however to date, no data has been published on the physiological responses at different relative exercise intensities relative to Kingcycle maximum minute power. The relatively fast, continuous ramp ~ 20 watts per minute (employed by the Kingcycle software) compared to stepped protocols (3 - 4 minute work stages, increasing 25 watts per increment) could possibly account for the differences observed. It is possible that this test, and the subsequent powers reached, or predicted and measured $\dot{V}O_2$max values are heavily influenced by Lac⁻ kinetics and therefore produce high correlation coefficients to performance in the laboratory. In other words the percentages of maximum minute power sustained is very similar between individuals (Bishop *et al.*, 1998). The stepped ramp protocol used by Coyle *et al.* (1991) may yield different (lower) values for maximal power output. Coyle *et al.* (1991) used a protocol that consisted of increasing work rate every 2 minutes to exhaustion. Dobbins *et al.* (1998) reported similar findings when altering continuous ramp rates during maximal testing protocols. They

41

reported significantly lower $\dot{V}O_2$peak values during a 5 watts per minute ramp protocol compared to faster ramps of 20 watts, and 35 watts, per minute (Table 6.2).

Dobbins *et al.* (1998) controlled the duration of the 35 and 20 watts trials (8-12 minutes). In their conclusions they made the assumption that if anaerobic capacity could be expressed as a fixed amount of time that could be sustained above a work load (that can be sustained by the aerobic system), the faster work rates will elicit higher maximum powers due to the increased ramp per minute of exercise. Likewise during the slower ramp rate of 5 watts they concluded that the extended time spent above the aerobically sustainable intensity exhausted the anaerobic system, via the build up of metabolites before $\dot{V}O_2$peak was elicited. The 5 watts trial lasted 30.36 minutes compared to 10.43 and 11.15 minutes in the 20 watts and 35 watts trials.

Table 6.2 Effect of ramp rate during maximal testing (Dobbins *et al.* 1998).

Protocol	$\dot{V}O_2$peak ($l\cdot min^{-1}$)	Maximal Minute Power (W)
35 watts	4.32 ± 0.63	393 ± 50
20 watts`	4.18 ± 0.54	$367 \pm 47*$
5 watts	$3.93 \pm 0.48**$	$305 \pm 30**$

* Denotes Significant difference from the 35 watts protocol. ($p<0.05$)

** Denotes Significant difference from 20 watts and 35 watts protocols. ($p<0.05$)

If there is a fixed amount of time that an individual can sustain above an aerobic work load then stepped protocols are likely to yield lower maximal power figures than fast ramp protocols, this may explain differences in performance prediction from different test protocols both Coyle *et al.* (1991) and Loftin and Warren (1994), used step type protocols. One further point to note is that the results from the Dobbins *et al.* (1998) study, is that there are greater standard deviations for both $\dot{V}O_2$peak, and maximum minute power during the higher versus the lower ramp rates, which could possibly indicate a greater differentiation between riders' abilities. Therefore, during studies that are trying to achieve a standard exercise intensity for athletes, the testing protocol for $\dot{V}O_2$max, and power at $\dot{V}O_2$max appear critical in determining a relative exercise intensity.

6.1.2 $\dot{V}O_2$max and Performance Prediction

Unsurprisingly due to the time involved in the endurance events of cycling, $\dot{V}O_2$max has become the common method of predicting performance. During field performance, the results will also depend upon the terrain. This is likely to influence the correlation coefficient depending upon whether the raw data is expressed relative to body mass ($ml\cdot kg^{-1}\cdot min^{-1}$), or in absolute terms ($l\cdot min^{-1}$). Table 6.3 below shows a collection of endurance cycling tasks and their correlation to $\dot{V}O_2$max. The highest correlation coefficient reported in Table 6.3 comes from the study by Malhortra *et al.* (1985), who concluded that stronger correlations to absolute $\dot{V}O_2$max are achieved with the longer duration of a performance trial

Table 6.3   Predicting performance from $\dot{V}O_2$max values.

Author	Performance trial	Correlation Coefficient. (time: $\dot{V}O_2$max)
Malhortra *et al.* (1985). $l\cdot min^{-1}$	84 km (undulating)	r=-0.87
Dobbins (1996). $l\cdot min^{-1}$	16 km (undulating)	r=-0.86
Rollings *et al.* (1995). $l\cdot min^{-1}$	2 km (climb)	r=-0.60
Dobbins (1996). $ml\cdot kg^{-1}\cdot min^{-1}$	16 km (undulating)	r=-0.59
Debold *et al.* (1996) $ml\cdot kg^{-1}\cdot min^{-1}$	14.73 km (climb)	r=-0.79

6.2 Pacing Strategy

The use of physiological variables to predict performance in road race cycling is going to have its limitations due to the effects of drafting, team tactics employed and the terrain involved.   To add to those variables Hickey *et al.* (1992) reported that the knowledge of an appropriate pacing strategy will alter time trial performance significantly.   It is unclear in many studies how the pacing strategy of individuals or a group was controlled for. However, during repeated performance trials altering the pacing strategy could significantly influence performance.   Foster *et al.* (1993) reported that during track cycling there is a wide variation in pacing strategies in short events lasting from 1-4 minutes. During the 1980 Olympic 1 km cycling event (~65 seconds) there was a trend toward slowing down in the latter stages. During the 4 km event there was a trend toward even paced strategy throughout the trial. Ingen Scherer *et al.* (1992) also reported data from these two events, and then employed 'all out' and 'constant power' trials over these distances.   They reported that the all out strategy employed during the 1 km event produced the fastest results, and the actual performance of the riders during the 4 km event produced the fastest times compared to their intervention trials (Figures 6.2 and 6.3). These are in contrast to the findings of Foster *et al.* (1993), who reported that during 2 km track cycling the even paced trial appeared to yield the fastest times compared to four other pacing strategies (Figure 6.4).

Foster *et al.* (1993) reported that these changes in time did not attain statistical significance, but went on to conclude that the findings were meaningful in the sporting context in terms of selecting the correct pacing strategy.   They also reported no significant correlations to $\dot{V}O_2$ uptake kinetics, the percentage of work derived through oxidative metabolism, or accumulated oxygen deficit values.   They concluded that differences in the rates of muscle Lac⁻ accumulation in relation to pacing strategy may be responsible for the changes in performance seen in their study. It appears for cycling disciplines of 2 km and further, even power strategy appears to yield the fastest times in those events.

Previous findings by Coleman *et al.* (1994) showed that cyclists who were given no feedback on pacing, produced a 'U' shape pattern of power production in terms of their pacing strategy (Figure 6.5).

Figure 6.2 Pacing strategy and finishing time for 1 km cycling (Ingen Scherer *et al.*, 1992).
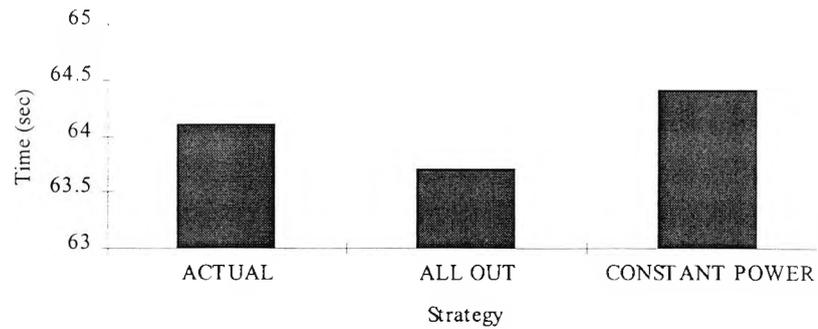
This pattern was reproduced for all four conditions in a study looking at the effects of creatine supplementation on 16 km cycling performance. An initial hard start was followed by a reduction in power, and a rise in pace again in the final minutes of the 16 km event.

Figure 6.3 Pacing strategy and finishing time for 4km cycling (Ingen Scherer *et al.*, 1992).
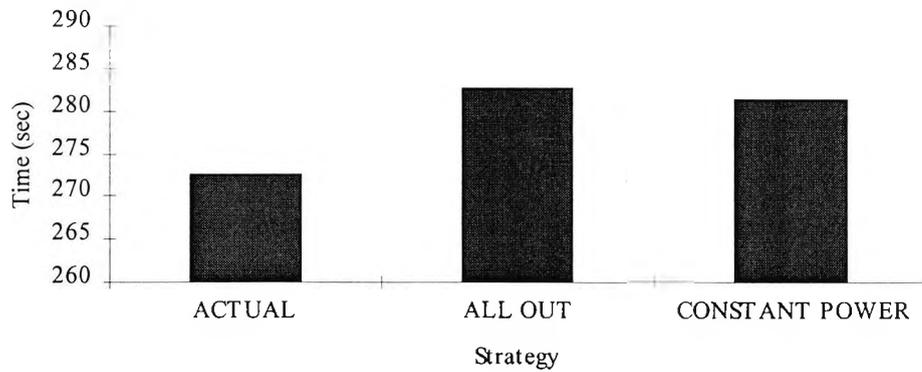
Figure 6.4 Starting pacing strategy and finishing time for 2km cycling (Foster *et al.*, 1993).

Time (minutes)
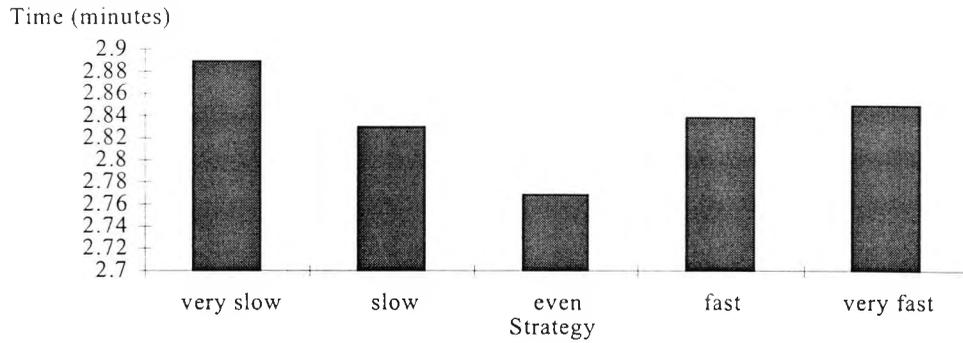
Figure 6.5 'U' shape pattern of power output during 4 trials (mean of eight rides per trial). (Coleman *et al.*, 1995).
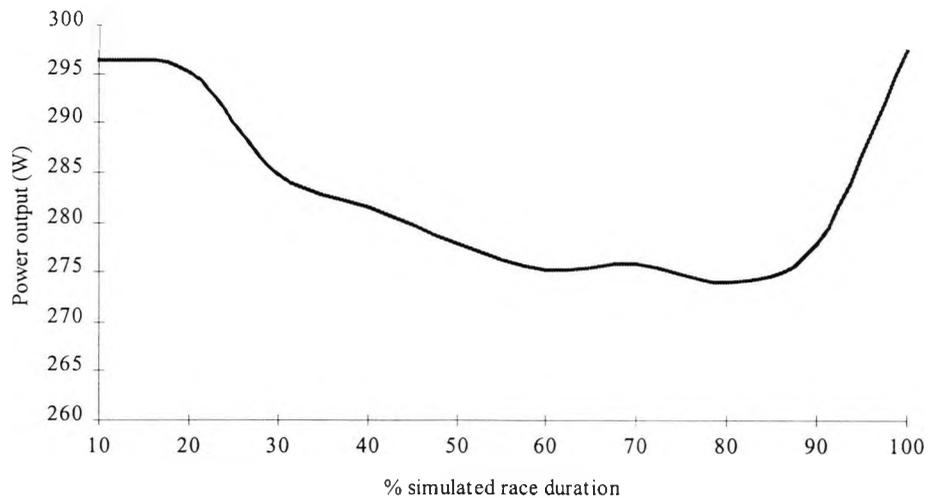
This 'U' shape pattern of pacing has also been shown to exist in the field (Balmer unpublished observations) and during other laboratory trials by Lindsay *et al.* (1996), in a study evaluating performance over 40 km, the subjects were again blinded to time elapsed, and in both trials this pattern was observed with approximately 50 watts decrease in power from the initial fast start to the trough of the 'U'. During a second trial after an interval training intervention, the pacing still ultimately followed the 'U' shape, but at approximately half distance an increase in power output for approximately 10 minutes was observed before returning to a lower power. This pattern has not been seen in the field, and has not been previously reported

in the literature. A change in pacing would have influenced the overall results, with the only measured physiological marker of heart rate which was elevated due to the increase in power.

Figure 6.6 shows the B[Lac⁻] measured at 5 minute intervals throughout the performance trial in the study by Coleman *et al.* (1994). The B[Lac⁻] showed an initial steep rise, linked to the fast start of these athletes.

Figure 6.6 B[Lac⁻] during 16km performance. (Coleman *et al.*, 1995).
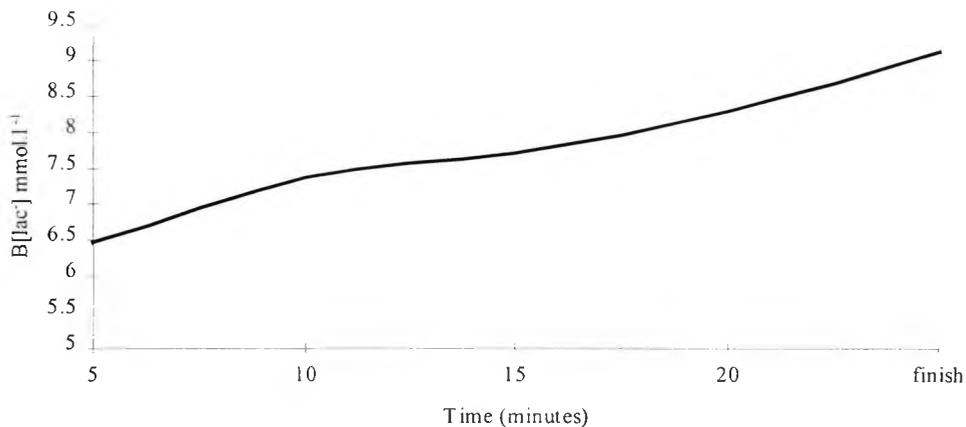
The period of adjustment by the athletes (reducing power output) still resulted in an increase in B[Lac⁻]. It would be interesting to speculate on the effects of reducing the fast start by these riders, which might decrease initial B[Lac⁻], resulting in the ability to maintain a higher work output during the middle phase of the 16 km performance trial. Therefore, if there are changes in pacing between trials for repeated performance testing it may not only effect the time to compete the trials (Foster *et al.*, 1993), but also the metabolic responses that are recorded during such testing. These findings therefore need to be applied to repeated measures testing, and in terms of performance testing need to be standardised to ascertain if differences in performance are due to physiological or pacing phenomena.

Hickey *et al.* (1992) reported the coefficient of variation for performance (time) over three different durations of cycling exercise, ~105 minutes, ~12 minutes, and ~0.55 minutes. Blinding their subjects to time elapsed, and only allowing them to view work completed (kJ), they reported that there was low variation for the two longer trials (Table 6.4). During the shorter trial there was greater variation compared to the other two trials.

Despite not indicating the pacing strategy of these athletes, the findings of Hickey *et al.* (1992), indicate there is minimal variation in trained cyclists performance (time and work rate). However, there were larger variations in physiological response in the Hickey *et al.* (1992) study (table 6.4).

Table 6.4 Coefficient of variation for experienced trained cyclists (US category 2/3) over three different duration trials. (Hickey *et al.*, 1992).

Variable	~105 minutes	~12.03 minutes	~0.55 minutes
Time	1.01%	0.95%	2.43%
kJ	0.5%	0.53%	1.35%
Watts	1.25%	1.53%	3.09%
$\dot{V}O_2$ l min^{-1}	3.02%		
$\dot{V}O_2$ ml kg^{-1} min^{-1}	3.64%		
RER	3.53%		

6.3 Effects of Body Mass; Hill Climbing Ability

A high $\dot{V}O_2$max relative to body mass may also be a prerequisite of a successful road cyclist due to the terrain involved in many races. The importance of hill climbing ability has been recognised by Rollings *et al.* (1995) as an important component of cycle racing that can influence the of results in cycle road races. Swain and Wilcox, (1992) reported that during uphill cycling most of the power output achieved goes into opposing the forces of gravity, leaving little for the development of forward force. This suggests that the effect of body and cycle mass will significantly influence performance. A study by Stovall *et al.* (1993) reported that during the Tour Du Pont (America's top international stage race), significant correlations were observed between the body mass of 55 male competitors and the finishing times on the three stages of the race with the most amount of climbing. The most significant correlation (r=0.41) between these two values was observed on stage 8, which had 6000m of climbing during the 158 km stage. Pfeiffer *et al.* (1993) also observed a significant correlation between finishing time and relative $\dot{V}O_2$ ml.kg^{-1}min^{-1} on 11 stages of an elite female stage race. The results from these studies are interesting in that because of crashes, mechanical problems and team tactics it often means that stronger riders are left back in the peleton, therefore the correlation may actually underestimate the true significance of the relationship. These intervening variables would also be implicated in one day racing, however no data has been reported on single one-day races, or body mass relative to laboratory power output and finishing race position. Jeukendrup and VanDieman (1998) showed the importance of sustained high power outputs in excess of 350 watts during a mountain climb lasting approximately one hour during the Tour de France. During one day racing this may not necessarily be the case, as sustained one hour climbing is not generally a characteristic of one day racing. Rollings *et al.* (1985) found a significant correlation between laboratory 2 km climbing performance (time) and relative $\dot{V}O_2$max (r=-0.71) as the highest predictor of performance. They concluded that an increase in $\dot{V}O_2$max, and optimising body mass would improve hill climb ability in trained cyclists.

According to Swain *et al.* (1994) the relationship between climbing ability and relative measures is not directionally proportional to body mass. They reported that during uphill treadmill cycling the mass

exponent was calculated to be 0.79. A direct relationship with body mass would have revealed an exponent of 1, however, as the 0.79 value is closer to 1 than the mass exponent for $\dot{V}O_2$max (0.67 which is also observed for surface area), then small cyclists should have better performances during hill climbing because their relative advantage in $\dot{V}O_2$max is greater than their relative disadvantage in energy cost. The disadvantage to smaller riders is that the mass of the bicycle is relatively heavier when compared to the heavier cyclists. Swain *et al.* (1994) reported that the added cost of the bicycle to be 12% in the larger cyclists as compared to 17% in the smaller riders. This resulted in a lower relative $\dot{V}O_2$ among heavier cyclists on a 10% gradient at 11.3 km.h^{-1} when expressed in ml.kg^{-1}min^{-1}.Changes in the physiological response during uphill cycling are further complicated by the alteration in cycling position, with riders sitting in the saddle, and standing on the pedals. Swain and Wilcox (1992) reported an increased $\dot{V}O_2$ during standing cycling was due to the use of accessory muscles recruited during the rocking motion of the bicycle. They concluded that increased upper body muscular activity was involved in torso stabilisation, and that the legs needed to provide support during standing cycling. The question of why trained cyclists adopt the standing position on the bicycle for an increased metabolic cost therefore needs to be addressed. Swain and Wilcox (1992), concluded that the shift to standing on the pedals brings the riders mass over the pedals which may enhance power production, and may relieve muscle fatigue by changes in recruitment patterns. Ryschon and Stray Gundersen (1991), concluded that the reasons cyclists choose the standing position is to reduce perceived exertion. However, the nature of physiological measurement in the laboratory during these studies has meant that cyclists have been required to spend extended periods of time in the standing position. It is debatable whether this would be the normal requirement in a road race where they might put in short bursts in the standing position. It is possible that this may not necessarily elevate the physiological responses to standing cycling that prolonged periods in the standing position have shown, however this has yet to be investigated. Anecdotal data from experienced cyclists also suggests that a shift to standing cycling may also be partly due to relieving contact points with the saddle and avoiding saddle soreness.

Any event that increases the need for a rider to complete the task standing out of the saddle may therefore require an increase in energy supply. The selection of when to alter position on the bicycle and altering muscle fibre recruitment could be an important factor in cycle races. Therefore the reasons may be peripheral fatigue, and the rider changes position to relieve these sensations. This results in increasing the energetic demand, (some of which is due to upper body exercise), which may increase the cost of movement centrally ($\dot{V}O_2$ and heart rate), but may however momentarily relieve the peripheral effects of fatigue. Despite possible changes in the position of a rider, the movement on the bicycle whilst climbing, and the importance of climbing ability in any major cycle race (e.g. Tour de France) there has been no data published on how graded cycling compares to the stationary position associated with laboratory ergometer type testing (e.g. Kingcycle or Monarch cycling).This line of investigation has implications for the evaluation of competitive cyclists in the field. Palmer *et al.* (1994) reported the heart rate responses of an elite group of cyclists during the Giro del Capo a 4 day stage race in South Africa. Any evaluation with

regard to matching the heart rate to a measured laboratory power output may be limited by any changes in seated and standing cycling. This could also be said for power measurement in the field with regard to metabolic stress. A set power output may have increased metabolic demands if the rider stands on the pedals increasing $\dot{V}O_2$ and heart rate compared to the seated position, therefore increasing the demands for a given power output. In this example the interpretation of the heart rate:power regression equation determined in the laboratory possibly has its limitations when extrapolating to field performance where cycling in and out of the saddle has occurred. The measurement of both power output and heart rate simultaneously in the field may allow for the analysis of the heart rate power relationship with regard to the true cost (power) of the event.

To summarise, many studies have evaluated cyclists with specific attention to aerobic power, which in the laboratory setting appears reproducible although the absolute values attained may vary according to the methods involved. The two major previous studies that have attempted to simulate road race cycling have also emphasised the importance of $\dot{V}O_2$max and assumed that a high oxidative capacity appears to be a prerequisite of the elite road race cyclist (Brouns *et al.*, 1990, Palmer *et al.*, 1997) However these studies do not appear to acknowledge that there are many road races that end in sprint finishes, and there are also many intermediate sprint 'primes'. Consequently the capacity to produce anaerobic power may also play an important role in the performance outcomes of the road race cyclist.

6.4 Anaerobic Responses of Trained Cyclists

The measurement and applicability of anaerobic power to racing cyclists has been relatively ignored when compared to the volume of literature regarding aerobic power. This is probably not surprising considering that road races last from 1-8 hours in duration, indicating the sport has an important endurance component. However, it is not necessarily the rider who possesses the highest aerobic power who will be the first rider over the line. Burke *et al.* (1977) indicated that anaerobic power is also an important requirement for competitive road racing cyclists. These athletes need the ability to be able to break away, climb steep hills and sprint at the end of the race, Burke *et al.* (1977) hypothesised that a fibre type mix of 50% fast twitch, 50% slow twitch slow twitch could possibly be ideal for these athletes, as the races are a mixture of both sprinting and endurance demands, although as previously indicated, studies have not shown this to be the case (Sjogaard, 1984). Palmer *et al.* (1994) reported that road racing was stochastic, with the dynamics of the peleton responsible for the changes in intensity (heart rate response). Possible implications of a higher anaerobic power could mean that it would take less time, and hence less overall effort to bridge gaps that appear when coming out of corners, with pace changes, and matching the efforts of other riders. This would aid in keeping the cyclist in the slipstream of other riders to far greater effect, reducing the oxygen cost of cycling (McCole *et al.*, 1990) and hence the overall energetic demand of the event.

6.4.1 Measurement of Anaerobic Power in Trained Cyclists

Relatively few studies have analysed the anaerobic power of competitive racing cyclists through single anaerobic test protocols. Tanaka *et al.* (1993), reported data from 30 second Wingate tests on a selection of American based riders. They concluded that as riders moved through the category system of the USCF there was an increase in peak power, mean power, and peak and mean power relative to body mass (Table 6.5). This could be interpreted as the quality of races increases as you move up the category system, anaerobic power needs to be increased to participate in those races. Indeed, Vrijens *et al.* (1982) reported no significant differences between professional riders and development squad riders for maximal aerobic power, therefore differences in race performance and results could possibly be explained by differences in anaerobic power, however no reference was made to issues of the percentage of sustained power and efficiency of these cyclists. The categorising (licensing) of individual riders however may favour those with higher relative anaerobic power. During many races, there will be whole peleton sprint finishes, for first place, and nearly always for the lower placing where points to move up the category system would be available. By drafting and staying with the main peleton those riders with the greater anaerobic capacity can regularly pick up points to upgrade their category. Whereas those riders' who possess a lower anaerobic capacity would have to escape from the peleton to secure points. This depends upon their tactics, teamwork, and other riders' tactics, to collect points from the category system.

Table 6.5 Wingate Anaerobic Power of Category Ranked Cyclists. Tanaka *et al.* (1993)

CAT	2 (n=7) male	3 (n=11) male	4 (n=12) male	2-4 (n=6) female
Peak Power (PP) (watts)	994.07	985.17	923.41	783.67
PP/kg (watts.kg^{-1})	13.86	13.55	12.8	12.17
Mean Power (MP) (watts)	804.05	805.16	749.45	614.8
MP/kg (watts.kg^{-1})	11.22	11.06	10.4	9.56
% Fatigue	34.25	33.46	36.65	37.8

Therefore the findings from Tanaka *et al.* (1993), (table 6.5) could possibly be explained from a different perspective. However, the importance of a high anaerobic capacity are highlighted from the coaching perspective.

Shearn *et al.* (1996) also analysed anaerobic capacity in the form of Wingate tests on 12 regional cyclists, who recorded lower figures than that of Tanaka *et al.* (1993). Peak power (PP) values were 704watts, (10.1 watts.kg^{-1}) Mean power (MP) 592.4watts (8.56 watts.kg^{-1}). The authors also gave their subjects a 7.1 mile time trial task on relatively flat roads, the subjects recorded a mean time of 1057 seconds for the time trial. Correlation analysis was performed, and time to complete the time trial significantly correlated with PP.kg^{-1} (r=-0.58), mean power.kg^{-1} (r=-0.61), and total work.kg^{-1} (r=-0.61). This data suggests that 7.1-mile time

trial is linked to anaerobic power and body mass indices, or that aerobic power and anaerobic power correlates in these athletes. As the time trial task lasted over 17 minutes in duration, the major contribution of energy supply would be expected to come from oxidative processes. The findings by Shearn *et al.* (1996) possibly imply two things, firstly, there is possibly a high contribution of anaerobic energy release during the time trial task, according to Balmer *et al.* (unpublished observations) during a laboratory simulated 16 kilometre time trial, subjects were exercising at 84% $\dot{V}O_2$max. As the 7.1-mile test is shorter you would expect the intensity to be slightly higher, Potteiger *et al.* (1995), concluded that exercising at 90% $\dot{V}O_2$max relies heavily on aerobic indices, there are however large amounts of lactate produced, indicating a significant anaerobic contribution to overall energy production.

The second possible explanation of the Shearn *et al.* (1996) findings could suggest that the Wingate testing protocol is not a test purely of anaerobic energy supply, but there is a significant aerobic contribution (Medbo *et al.*, 1989, reported a 40% aerobic energy contribution to a 30 second sprint). This has been speculated by Withers *et al.* (1991) who, concluded that during 30 second sprints the processes of (ATP) replenishment is primarily supplied by phosphocreatine (PCr), however some ATP would be synthesised from oxidative metabolism by the O_2 stores in the body bound to haemoglobin and myoglobin, and from O_2 dissolved in body fluids and in the lungs. During longer sprint tasks (60 and 90 seconds) Withers *et al.*, (1991) reported significant correlations between $\dot{V}O_2$max and average power ($r= 0.81$, and $r=0.97$), and highlighted that aerobic metabolism plays a major role during anaerobic capacity testing in endurance athletes (in this case road cyclists).

Withers *et al.* (1991) also speculated there may be individual variation in response to 30 second sprint tests. They hypothesised that two subjects can perform exactly the same amount of work during such a test, but depending upon $\dot{V}O_2$max, and the $\dot{V}O_2$ increase at the onset of such a test, there are possible differences in anaerobic yield from the test. Therefore two riders competing in the same sprint finish may produce the same amount of anaerobic energy for contraction, but depending upon aerobic parameters and oxygen kinetics, the winner of the sprint could theoretically be the rider who is able to supplement the anaerobic energy release via oxidative energy metabolism, and perform more work. These authors also reported that the percentage of fast twitch fibre, and index of fatigue both significantly correlated with the peak B[Lac$^-$] ($r=0.81$ and $r=0.85$ respectively). Withers *et al.* (1991) speculated that a high $\dot{V}O_2$max, and fast oxygen kinetics would reduce glycogen depletion, and reduce lactate yield from a sprint, which would have implications for endurance exercise.

Withers *et al.* (1991), reported that duration of the anaerobic test will also alter the physiological responses during this type of exercise. They reported similar peaks in B[Lac$^-$] response time, in terms of time to peak (7.5 minutes post test) after separate all out sprint tests lasting 30, 60 and 90 seconds. However differences were seen for the absolute values of BLac$^-$. Significantly lower values were seen for BLac$^-$ after 30 seconds compared to the other sprints of longer duration, possibly indicating a greater reliance of PCr to supplement

51

the energy for contraction rather than glycolysis, plus according to Medbo *et al.* (1989), an aerobic contribution of 40% of the energy supplied during a 30 second sprint effort. The highest B[Lac⁻] were seen after 60 seconds sprinting, and although there is a higher percentage of aerobic energy supply of approximately 50% (Medbo *et al.*, 1989), PCr stores would be exhausted rapidly, and glycolysis would be the major supply of anaerobic energy production during sprinting of this duration. During the longer sprint of 90 seconds, less BLac⁻ accumulation occurred, this was according to Medbo *et al.* (1989) due to the inhibition of glycolysis by H⁻, reducing the muscle cell's ability to process glycogen in this manner, and any further Lac⁻ production would be dependant upon the individual's ability to transfer lactate to other compartments.

Sprints during the road race event may possibly vary in duration, however, it could be speculated from previous simulation studies by Brouns *et al.* (1990) and Palmer *et al.* (1997) that anaerobic capacity does not play a major role in this type of exercise because they have not simulated intense anaerobic demands.

Terrandos *et al.* (1994), concluded that the anaerobic capacity of professional road racing cyclists was not limiting when they compared values of Maximal Accumulated Oxygen Deficit (MAOD) to short distance Kayak racers on sport specific ergometers. The Kayak racers were classed as highly anaerobic athletes due to the nature of their short distance event (500m), and as the results showed no significant differences between the athletes for MAOD, they concluded that anaerobic capacity in elite cyclists was not a limiting factor to performance. This supports the conclusions of Tanaka *et al.* (1993) who summarised that although aerobic power is a major determinant of success in endurance events the anaerobic power in their athletes was very high, and higher than have been reported in some anaerobic athletes.

Sjogaard (1984), took muscle biopsy samples taken from the *vastus lateralis* muscle to analyse for anaerobic and aerobic enzymes. She measured concentrations of Lac⁻ dehydrogenase (LDH) as a marker of glycolytic activity within the muscle. Concentrations of citrate synthase (CS) were used to represent the aerobic potential of the muscle. Comparisons were made for these enzymes between elite 'Tour de France' cyclists, and competitive road racing cyclists of a lower standard. The study also measured these variables in the close season (February) and in July when the riders would be expected to be in peak condition for the Tour de France cycle race. The elite riders had significantly lower LDH concentrations compared to the competitive riders (628 mmol.kg.d.w⁻¹ vs 1100 mmol.kg.d.w⁻¹ respectively), but the elite riders had significantly higher CS a concentration compared to the competitive riders (69 mmol.kg.d.w⁻¹ Vs 41 mmol.kg.d.w⁻¹). The ratio of aerobic:anaerobic potential CS:LDH ratio was higher in the elite riders compared to the competitive riders (CS:LDH 0.1 Vs 0.04 respectively). From the beginning of the racing season (February) to the end of the Tour de France, (July) the concentration of LDH decreased (612-518 mmol.kg.d.w⁻¹), in the elite riders, where as CS concentration increased (65-112 mmol..kg.d.w⁻¹) which increase the ratio of CS:LDH to 0.2 in July. There are a couple of explanations for these findings, the first is the nature of training and racing that the elite riders participate in. The elite riders participating in the Tour de France would be expected to cover 3,500-4000 kilometres in a 3-week period. The other races on the

elite calendar are also often over 250 kilometres in length, incorporating the need for aerobic endurance in such events. The competitive riders would be competing over shorter distances, typically races lasting 100-160 kilometres in length. With these races being considerably shorter, in theory they could be more intense, requiring a larger anaerobic capacity to cope with the demands of the event, compared to a 250 km race requiring greater aerobic endurance. This could also help explain why the sub elite riders who compete in shorter races spend a higher percentage of time at higher intensities hence success is determined by fibre type make up or the training/racing patterns of these riders. The results from Sjogaard (1984) study may help to explain why riders struggle to make the transformation from amateur to professional cycling, as the demands placed upon a successful elite professional rider may well be different from racing at an amateur level.

Sjogaard (1984) also reported BLac⁻ values recorded after a maximal aerobic power test, the results indicated that the competitive cyclists relied to a greater extent on anaerobic energy sources as B[Lac⁻] were significantly higher than the elite riders (elite 8.2 mmol.l⁻¹ vs competitive 15.7 mmol.l⁻¹). The values for the elite riders are similar to those reported by Mendoza *et al.* (1994) in competitive cyclists (8.2 ± 2.6 mmol.l⁻¹). Barlow *et al.* (1985) reported that non-competitive cyclists recruited more fatigueable fast twitch fibre during maximal aerobic power testing, which resulted in greater BLac⁻ accumulation, despite producing less work than competitive cyclists. With the results from Terrandos *et al.* (1994) and Tanaka *et al.* (1993), indicating that anaerobic capacity in elite level cyclists is high, coupled with the results from Sjogaard (1984), of reduced LDH activity, and higher CS activity it may indicate that top level road cyclists are able to produce more work through the activation of slow twitch fibre than untrained or less conditioned individuals. Coyle *et al.* (1992) reported that despite a greater percentage of slow twitch muscle fibre, elite time trial cyclists produced higher peak torque at the crank arm than non elite time trial cyclists. With reduced LDH activity in slow twitch muscle fibre this would then lead to a reduction in lactate production until higher relative intensities.

The road race cyclist has the ability to produce high values for anaerobic power (Tanaka *et al.*, 1993), yet does not possess the intermediate enzymes for glycolytic energy production in high concentrations (Sjogaard, 1984). Therefore it appears that the road cyclist has the ability to achieve high powers without the classic muscle fibre type or glycolytic capacity generally associated with anaerobic performance. To date no studies have looked at the interaction between the aerobic and anaerobic capacities of these athletes.

6.4.2 Repeated Sprint Activity

A major criticism of single tests to analyse anaerobic capacity in relation to race performance is that they are not specific enough to road racing cyclists. With track sprinters, there is a single effort made lasting between 10-12 seconds, however, it appears according to Palmer *et al.* (1997) that the road race cycling is made up of many changes in intensity. Therefore the looking at a single sprint may not give us the information that is required when looking at differences between elite riders and sub elite riders. Unfortunately data on repeated sprint ability is only available on team game players.

53

Research by Hamilton *et al.* (1991) reported that after completing thirty, six second running sprints (54 seconds recovery) the post exercise B[Lac⁻] showed a strong relationship with peak speed (r=0.9), peak power (r=0.92), and mean speed (r=0.83). This research was carried out on team game players whose muscle morphology is likely to be different from the more homogenous slow twitch morphology of road racing cyclists. However, Hamilton *et al.* (1991) speculated on the effects of a higher slow twitch aerobic athletic component. They hypothesised that the greater O_2 supply might aid in the replenishment of PCr which has been shown to be directly related to capillary density (Tesch and Wright, 1983). Therefore despite glycolysis being activated in the first second of contraction, (Balsom *et al.*, 1992, Hultman and Sjoholm, 1983) athletes with a higher slow twitch muscle fibre make up may rely to a greater extent upon PCr, and replenished PCr during repeated sprint activity reducing the reliance on glycolysis and the LDH reaction, resulting in lower Lac⁻ production. This again has implications for endurance with decreased glycogenolysis, and increasing the availability of glycogen during the later stages of exercise. The pattern of force production during repeated sprints has been shown to change during repeated sprint activity. McCartney *et al.* (1984) reported that during a set of 4 x 30 second sprints on a bicycle ergometer, peak power was attained in the first 2 seconds of the first two sprints. During the final two sprints, the peak power was not generated until the 10th second of the protocol. (There was a decrement of peak and mean power from tests 1-4, but there was a similar rate (slope) of fatigue during all tests). These findings were supported by Spriet *et al.* (1989) who reported that during three repeated Wingate tests, peak power was achieved in the first 1-2 pedal revolutions in the first two tests, but delayed until the 9th - 10th pedal revolution in the third test. Both protocols used similar 4-minute rest periods between sprints. The authors hypothesised that despite the availability of PCr, the rapid rate of PCr utilisation stimulated glycolysis during the first two sprints. Glycolysis then appeared to be inhibited during 3rd and 4th sprints as there was no detectable reduction in glycogen or rise in muscle Lac⁻.The pattern of force application in road cyclists has not previously been reported, however, it is interesting to speculate as it may help to explain why elite road race cyclists have a high slow twitch fibre content, yet the demands appear to have an important anaerobic component. If during repeated sprinting there is a tendency for the peak power to be applied to the crank after 10 seconds during later efforts, this also has implications for the tactical nature and training of road race cyclists. The ability to produce high forces instantly would increase the riders' ability to close gaps, and accelerate away from other riders who may take longer to generate the forces. This is a possible difference between cyclists of different standards, where it is not necessarily the peak power produced, but the time to peak power that is important, and influencing performance as described by Burke *et al.* (1977).

6.4.3 Physiological Response to Intermittent Endurance Exercise

Many team and individual sports are made up of bursts of high intensity activity, followed by periods of recovery. Many of these activities last well over 60 minutes in duration, implying that physiologically there is an important aerobic component, as well as an essential anaerobic capacity, to compete in such sports. This has been acknowledged by Schabort *et al.* (1998) reported high reproducibility during an intermittent

exercise protocol (coefficient of variation for time 1.7%) on three repeated trials of 100km at an even power with four 1 km and four 4 km maximal efforts incorporated.

One of the major studies investigating intermittent exercise was carried out by Essen *et al.* (1977). Using an intermittent protocol (INT) of 15 seconds of work followed by 15 seconds of rest, to produce a mean power of 157 watts (55% $\dot{V}O_2$max), the intermittent protocol was compared to continuous exercise (CONT) at the same intensity and duration (60 minutes). During their study the heart rate response differed between the two conditions. During the continuous protocol the heart rate was initially 11 beats min[-1] lower when compared to the intermittent trial, which supports the conclusions of Kenny *et al.* (1995), and Vanhelder *et al.* (1985) who reported that the linearity of heart rate versus power is influenced by intermittent exercise, causing elevations in heart rate for a given power. These findings are in contradiction to those of Palmer *et al.* (1997) who found during a similar matched intermittent and continuous experimental protocol, heart rates were actually higher on average during the continuous protocol compared to the intermittent ride (153 Vs 150 beats min[-1]). However, as the exercise progressed during the Essen *et al.* (1977) study, a drift of 20 beats min[-1] during INT and 27 beats min[-1] during CONT, resulted in no significant differences between the two trials at the end of the exercise protocol. The findings from Essen *et al.* (1977) could be linked to the $\dot{V}O_2$ response where initially the $\dot{V}O_2$ was higher in the intermittent and then the drift up in $\dot{V}O_2$ from the CONT exercise matched the $\dot{V}O_2$ response from the INT protocol. The lower initial heart rate during CONT and the greater drift up (27 beats min[-1]) could be explained by alterations in substrate use. Although there were no significant differences in glycogen depletion between the two protocols or free fatty acid oxidisation, slightly higher triglyceride utilisation was observed during the CONT trial. The greater drift up in heart rate (and O_2 cost) during CONT may have been due to the higher O_2 cost of triglyceride utilisation later in exercise. Vanhelder *et al.* (1985) reported that free fatty acid oxidisation was significantly higher during the continuous task as opposed to the intermittent protocol, (although contradictory to the findings of Essen *et al.*, 1977), could also lead in an increase in $\dot{V}O_2$ to oxidise free fatty acids as substrate. Palmer *et al.* (1997) reported less drift up in the heart rate response during their 150 minute protocol at 58% power output at $\dot{V}O_2$max (10 beats min[-1]), this study used trained cyclists, and it is possible that the subjects were able to metabolise a greater proportion of triglycerides from the beginning of both the continuous and the intermittent protocols. A $\dot{V}O_2$:power output comparison over time of trained cyclists has not previously been reported, but it has implications for the measurement of heart rates from the road race event (and other sports) and trying to predict demands in the laboratory from establishing a $\dot{V}O_2$:power response, which will possibly be altered due to the individual intermittent nature of each race. These findings also have implications for riders that use a pulse meter to gauge their efforts during road race events, with not only elevations in heart rate during the race compared to steady state rides, but also possibly a drift up over time in the heart rate response, compared to power.

The Essen *et al.* (1977) study also revealed differences in leg blood flow between the two protocols. The CONT protocol elicited a higher overall leg blood flow, whereas the INT exercise had higher blood flow

55

during the recovery periods compared to the intense intervals. This has the implications for the supply of O_2 and the removal of bi-products, which could be a key factor in competitive road race cycling. The repeated occlusion of blood flow to the exercising limbs could result in significant local fatigue due to the inability to supply substantial amounts of O_2 to the working muscles. No significant differences were seen for muscle glycogen depletion due to the changes in blood flow, a greater reliance on local glycogen supply might be expected, however, a greater use of blood glucose was observed for the CONT protocol (23% Vs 17%), which is possibly due to the occlusion during the intermittent trial reducing blood flow, and transit time for the supply of blood glucose. Vanhelder et al. (1985) reported that during intermittent exercise of 20 minutes duration (1 minute work 1 minute rest), there were no differences in blood glucose concentrations when compared to a continuous task of the same duration. This possibly indicates that as time increases, blood glucose could play an increasing role in continuous compared to intermittent exercise.

Due to the greater blood glucose and triglyceride utilisation during the CONT protocol of the Essen et al. (1977) study, and the similarities for glycogen and free fatty acid oxidisation, there appears to be a small discrepancy for energy utilisation between the two protocols. It is possible that Lac⁻ utilisation could have been used by active tissues. Essen et al. (1977) reported the production of 1.5 mole min⁻¹ of Lac⁻ throughout the INT protocol, compared to the CONT trial, where apart from the first 5 minutes of exercise there was no net production or removal of Lac⁻. Kozaris and Mongomery (1991), also reported changes in the Lac⁻ response with an intermittent versus continuous protocol. They also concluded that sustained muscular contraction restricted blood flow, but immediately post exercise there was an increase in blood flow. They concluded that increased blood flow during recovery increased the Lac⁻ efflux, as opposed to a continuous test where there would be minimal changes in blood flow. Due to the occlusion during short work periods there is a greater rate of glycolysis which could also be accounted for by the increased Lac⁻ values seen during intermittent protocols. For the road race cyclist it is important that sufficient recovery is permitted between bursts if tactics allow. This would enable recovery periods to remove Lac⁻ and H^+, and increase blood flow to the previously active muscle fibres. The release of Lac⁻ may also reflect a larger recruitment of fast twitch fibres during an intermittent protocol (Essen et al., 1977). It has been speculated that both fast twitch and slow twitch fibres are recruited during intermittent exercise (Spriet et al., 1989, Linossier et al., 1993). During repeated Wingate sprinting, Spriet et al. (1989) reported that the work rate during the third test (4 min recovery between tests) was 82% of the previous bout, but only 32% of glycogenolysis occurred compared to the previous bout. They concluded that it was possible through the activation of and slow twitch fibre for a greater ATP yield to come via the oxidative metabolism of glycogen. Linossier et al. (1993) reported an increase in the percentage of slow twitch fibres following a 7 week intensive 5 second sprint training program, however, the fibre size appeared to decrease, resulting in an unchanged percentage and slow twitch area. This could possibly explain the findings of Sjogaard (1984), who found homogenous fibre type amongst elite professional riders 55-85% and slow twitch fibre, compared to the lower level competitive cyclists 15-75%. Linossier et al. (1993) concluded that slow twitch fibres becomes more

involved during the recovery periods from intense exercise, repleting PCr, and oxidating Lac⁻ within the muscle. Further evidence of both fast twitch and slow twitch fibre activation during intermittent exercise comes from the reductions in muscle glycogen in both fibre types during this mode of exercise (Greenhaff *et al.*, 1992). Linossier *et al.* (1993) reported at the end of an intermittent training protocol there was a significant increase in glycolytic activity, and no change in the concentrations of PCr. Glycolytic activity during this study significantly correlated with the production of high powers (r=0.87). As anaerobic power is believed to be involved in sprinting, climbing and breaking away during road race cycling, then potentially the glycolytic capacity of the rider could play an important role in performance and race results of an individual. The effects on performance of variable intensity versus continuous exercise have been reported by Palmer *et al.*, (1997). During a 150 minute variable intensity road race simulation protocol at 58% Wmax, subjects performed a 20 km time trial in the fastest time possible. The variable intensity protocol increased the mean time to complete the task by 1 minute 36 seconds, and reduced the mean power output sustained by 37 watts, and the mean heart rate by 6 beats min⁻¹. Palmer *et al.* (1997) concluded that this was due to greater reliance upon glycogen during the variable intensity protocol, reducing the substrate availability during the performance test. No data has been published by these authors on the speculation of greater glycogen utilisation during the variable intensity task compared to an even power trial. However, there is a possibility because the activation of both fast twitch and slow twitch fibre during variable intensity exercise would cause glycogen depletion in both fibre types (Greenhaff *et al.*, 1992), whereas during the continuous protocol there would be less fast twitch fibre activity, enabling greater fast twitch recruitment during the intense 20 km performance test. The results from these studies suggest an increased energy cost of performing variable intensity exercise compared to a continuous work output, with the results indicating that performance is significantly affected. There are issues regarding quantifying the energy cost of accelerating up to higher powers due to the inertial effects of the ergometer flywheel during variable intensity exercise trials. There has been no detail included regarding this issue within the methods sections of these papers so it is unclear whether the inertial cost of acceleration has been discounted, and the subjects in fact produced more work during intermittent trials. Although there are some issues surrounding the methods associated with changes in pace during variable intensity exercise, it still appears that continuous trials are less demanding and maintaining a continuous power output would increase performance in the latter stages of races (Palmer *et al.*, 1997). A steady state ride appears an impossibility during a road due to the peleton dynamics (Palmer *et al.*, 1994) therefore if variation in intensity occurs, selecting when to make the bursts of effort, and recognising the detrimental effects of wasted attacks can be incorporated into race tactics and will have implications for performance.

6.5 The Specificity of Exercise; The Ergometer

In evaluating aerobic and anaerobic power, the use of cycle ergometers alongside treadmill testing is probably the most popular format for the testing of athletes in the sport science laboratory. These tests appear sport specific to the racing cyclist, however research has begun to question the assumptions behind

the extrapolation of laboratory findings to field performance. Simulating cycling in the laboratory may not be as simple as it appears. A study by Padilla et al. (1996) reported changes in the energetic demand between simulated laboratory cycling and actual velodrome cycling. They reported significantly higher B[Lac⁻] in the field tests compared to the laboratory tests at the higher work rates. There are a couple of limitations to this study, firstly, exercise intensity was calculated using the equations from Di Prampero who used track cyclists for the $\dot{V}O_2$ - speed calculation, where as Padilla et al. (1996) used road cyclists. This could affect the energetic demands of the track tests by inexperienced riders failing to maintain the shortest distance around the velodrome, which experienced riders would be following. The second limitation is the lower pedal cadence at which the laboratory test were calculated (70 rpm), as opposed to the self-selected ratios that would be higher in the field. Further possible explanations for these findings are documented by Kenny et al. (1995). These authors compared the submaximal exercise responses to riding on a velodrome surface, and laboratory exercise tests on a Monarch type stationary ergometer, and cycling on a motorised treadmill. They used heart rate as an intensity guide and found significant differences in forces at the crank between field and laboratory tests. Kenny et al. (1995) reported higher peak and average crank torque values during treadmill cycling, which would be likely to elevate the demands, and increase B[Lac⁻] as described by Padilla et al. (1996). Kenny et al. (1995) concluded that this was possibly down to differences in maintaining balance on the track compared to maintaining balance on a monarch ergometer or cycling on a treadmill. Kenny et al. (1995) speculated that the inertial characteristics of the laboratory testing ergometers could account for the differences in physiological responses, although no mention was made about the energetics of leaning around the curves of the velodrome compared to upright cycling on the ergometry systems. This is a valid consideration when testing athletes and trying to make comparisons to field conditions. For example Loftin and Warren (1994), reported that competitive cyclists (USA category 3 and 4) with a laboratory measured $\dot{V}O_2$max of 4.0 l·min⁻¹, (55.4 ml·kg⁻¹·min⁻¹) were able to complete a 16 kilometre task on Velodyne trainer in an average time of 16 minutes and 18 seconds, riding at a mean pedal cadence of 118 rpm, for a sample of six subjects. The average speed for such a ride is 58.9 km.h⁻¹ which is substantially faster than the current world hour record, held by Chris Boardman, (a top professional rider), who completed the record ride at a predicted mean power output of 442 watts (Keen, unpublished observations). It is unlikely that the category 3 and 4 riders could perform such high work rates considering their $\dot{V}O_2$max values. It is possible that the inertial characteristics, and loading upon the riders were different when compared to those experienced in true racing conditions.

The work of Loftin and Warren (1994) was designed to test riders in their sport specific positions, Svaren et al. (1994), concluded that the use of racing cycles attached to test ergometers, can yield differences in $\dot{V}O_2$max due to alterations in motor unit recruitment patterns. This could possibly be an area of variation amongst tests performed on elite athletes. Many studies have attempted to evaluate elite and sub elite riders in respect to physiological and metabolic variables, however, the ability to perform in the laboratory may be concealed by changes in such things as muscle fibre recruitment patterns, altering physiological and

metabolic demands. Padilla *et al.* (1996), reported that in their experience of laboratory testing, elite cyclists preferred sport specific tests, which where performed with equipment with which they are familiar. This does however pose a few problems for the experimenter. The developments of sport specific cycling ergometers that allow the cyclist to mount their own bicycles are a significant progression from the Monarch type system previously used. However, there are other considerations that need to be made before expressing the potential for an ergometer to simulate field conditions. Evangelisti *et al.* (1994) investigated changes in the riding position of moderately trained cyclists ($\dot{V}O_2$max = 56 ml.kg^{-1}min^{-1}) and their physiological cost of movement. They manipulated the handlebar position of the riders from the standard drop position to using an aerodynamic handlebar position. The authors found that the drop handlebar position yielded a significantly lower heart rate (2 beats min^{-1}) and $\dot{V}O_2$ response (2 ml.kg^{-1}min^{-1}), indicating changes in position can alter the physiological cost of cycling. They concluded that it is essential to test riders in their cycling position in the laboratory for accuracy. During road races cyclists tend to move around on their bicycles to an extent. Standing for sprinting and hill climbing, moving forward on the saddle in an aerodynamic tuck on the flat etc. The ergometer chosen must have the ability to cope with these changes, as the reasons cyclists move are possibly due to decrease the sensation of effort in the legs (Tanaka *et al.*, 1996), or according to Swain and Wilcox (1992) riders move to enhance power production, and relieve muscle fatigue by changing recruitment patterns. In terms of testing cyclists, the ability to move around is essential when looking at endurance tasks. If Swain and Wilcox (1992) are correct, and that fatigue can be relieved by a change in position (from seated to standing for example) then any ergometer that requires an elite cyclist to remain in the same position could cause premature fatigue, and show a poorer reflection of an individual's capability. In terms of endurance, any change in muscle fibre recruitment may also allow for the use of glycogen that would otherwise remain inside the (un) recruited muscle cell. It appears that the ergometer choice could potentially affect physiological responses to exercise. Therefore the selection of ergometer (and protocol) must replicate the sporting conditions in order for cyclists to perform to their racing potential.

Chapter Seven. Rationale for studies described in the thesis

7.1 Chapter 9. (study 1).

In the simulation of cycling, there have been many different methods of ascertaining a relative exercise intensity to standardise procedures. Previous research has used percentages of $\dot{V}O_2$max to determine a relative exercise intensity (e.g. Coggan and Coyle, 1987). The rationale behind using this method is that performance can be predicted from maximal values. Strong correlations have been found between performance and $\dot{V}O_2$max, the values shown previously in table 6.3 display results from a variety of studies ($r=-0.59$ to 0.87). However, $\dot{V}O_2$max has also been shown to be a poor predictor of performance, Loftin and Warren (1994) reported a correlation coefficient $r=-0.31$. This has led to other methods being utilised to standardise relative exercise intensity. Passfield et al. (1995) used maximum minute power (derived from Kingcycle testing protocols) to identify a relative exercise intensity in highly trained cyclists. Maximal minute power has subsequently been found to strongly correlate to performance, Davison et al. (1997) reported $r>0.9$ between maximum minute power and 16.1 km laboratory simulated trials. In terms of constructing a road race simulation protocol from power output data, a measure of power output would be required to achieve this. Keen et al, (1991) reported a strong correlation between maximal minute power and $\dot{V}O_2$ max, however, no studies have evaluated the maximal minute power test for reliability, and the physiological responses of cyclists at different percentages of this maximal intensity. Therefore one of the initial aims of this study was to evaluate this test, for reliability and note the physiological responses of well trained cyclists riding at different percentages of maximum minute power output.

7.2 Chapters 10 & 13. (Study 2 and 5)

Previous research has estimated the energetics of road race cycling from the heart rate and power relationship in the laboratory (Palmer et al., 1994). However, there are some discrepancies between the perceptions of the demands of the sport (e.g. Burke et al., 1977) and the findings of those who have attempted to simulate road race cycling in the sport science laboratory (Brouns et al., 1989. Palmer et al., 1997). With the development of a portable power measuring device (SRM powermeter), one of the aims of this study is to clarify the energetics of actual field competition.

Burke et al. (1977) speculated that sprinting, like hill climbing, was a key factor associated with road racing performance. Compared to the volume of literature on cyclists aerobic power, there is limited data on the anaerobic responses of well trained road race cyclists. With the recording of power output during competitive races, the aim was to also record standard laboratory tests of anaerobic power/capacity (Wingate) to compare field and laboratory based responses.

The aim of this study is to record field data on competing cyclists, and, using this data, create a simulation of road race cycling in the laboratory that replicates the demands that these riders face in the field.

The simulation of road race cycling by Palmer et al, (1997) included a 20 km performance time trial at the end of the simulation. There were however minimal physiological variables presented, therefore predicting

performance following a period of simulated road cycling was not possible. The benefits of constructing a valid simulation of road race cycling and performance test would allow for the measurement of physiological variables, which might give some insight into the factors that influence road race cycling performance. Previous research has indicated that B[Lac⁻] could be linked to performance outcomes (Sjogaard, 1994), likewise NH_3 (Brouns *et al.*, 1990), percentage of $\dot{V}O_2$max (Coyle *et al*, 1992), and efficiency (Cavanagh and Kram, 1995). Palmer *et al.*, (1997) speculated that during their simulation of road cycling there was more muscle glycogen used compared to an even power trial, and, this resulted in reductions in performance following simulated road cycling compared to even power cycling. Previous research has indicated that carbohydrate availability (Coggan and Coyle, 1989) is critical to endurance performance. Yaspelkis *et al*, (1993) and Hargreaves *et al*, (1984) reported muscle glycogen sparing during an intermittent exercise protocol with glucose feeding. During even power cycling this has not been reported, however performance benefits have been reported (Coggan and Coyle, 1989). The performance benefits have been indicated to come from the ability of blood glucose to support exercise intensity, however this is limited to 75% $\dot{V}O_2$max late in exercise (Coggan and Coyle, 1988). With the speculation of Palmer *et al*, (1997) and the findings of Yaspelkis *et al*, (1993) and Hargreaves *et al* (1984), it could be speculated that glucose supplementation may have a greater impact upon intense performance (~100% $\dot{V}O_2$max) following intermittent exercise than compared to even power cycling due to the resynthesis of muscle glycogen during intermittent protocols. Therefore a comparison of responses (and performance) to glucose feeding for even power and intermittent cycling was a further aim of this study.

7.3 Chapters 11 and 12. (Studies 3 and 4).

Burke *et al.* (1977) also speculated that climbing was a very important components of road race cycling. Stovall *et al.* (1993) reported that significant correlations were observed between body mass and time to complete mountainous stages of a professional stage race, and Pfeiffer *et al*, (1993) reported that $\dot{V}O_2$max ($ml\,kg^{-1}\,min^{-1}$) correlated to the finishing time of elite female road cyclists during similar hilly days of a stage race. The studies by Stovall *et al.* (1993), and Pfeiffer *et al.* (1993) would have had their data influenced by team tactics, crashes, mechanical trouble, which could result in stronger or lighter riders left behind weaker/heavier riders. No research has been conducted on the measurement of power output and the prediction of this key component of road race cycling, therefore the aim is to analyse the role of hill climbing in road races, and report the key factors associated with hill climbing performance.

Chapter Eight. General Methodology

The aim of this chapter is to outline general methods of physiological testing (equipment and protocols used) that were common throughout the various studies. All laboratory-based studies were carried out in the Canterbury Christ Church University College Sport Science laboratory. This laboratory holds British Association of Sport and Exercise Sciences (BASES) accreditation status.

8.1 Subject Recruitment

All studies gained ethical committee approval prior to the onset of physiological testing. Subjects were recruited on a voluntary basis, initially with the help of a British Cycling Federation Senior coach. All subjects completed a medical questionnaire, and gave written informed consent. All subjects were informed that they could withdraw from the testing protocol at any time.

8.2 Testing Procedures

8.2.1 Initial Testing Procedures

Prior to all physiological assessment in the laboratory, laboratory temperature and humidity were recorded (RS TH07, SLS. Nottingham, UK) and barometric pressure (F.D & Co. Ltd. Watford, UK).

Upon arrival at the laboratory general subject details were recorded. Subjects' age, height and body mass were recorded to the nearest year, 0.01 m, and 0.1 kg (Balance beam scales, Kent & Sussex scales, Wadhurst, UK) respectively.

8.2.2 Equipment and Testing Protocols

Equipment that was commonly used throughout the studies is outlined below, with a brief introduction, protocols, and validation/reliability practices.

8.2.2.1 The Kingcycle Testing Rig

8.2.2.1.1 Introduction

The Kingcycle testing rig (EDS Portaprompt, High Wycombe, UK) has previously been used by sport scientists and coaches as a sport specific method of exercise assessment, and has been used to evaluate elite cyclists by the British Cycling Federation.

The Kingcycle was developed to simulate the inertial characteristics of rolling resistance that a cyclist would experience on the road. The rig allows for the attachment of the subject's own bicycle with the front wheel removed and the forks clamped onto a support by a quick release skewer. The bicycle is supported underneath the bottom bracket shell by an adjustable rubber support, which is used to raise or lower the subject's rear wheel onto a roller. The roller is air-braked to create the resistance with a fan on one side of the roller, and a 4.6 kg flywheel on the opposite side of the roller.

During use the Kingcycle rig relays information via an optical sensor adjacent to the flywheel to a PC (DAN Technology PLC) to display power output continuously by analysing the velocity of the flywheel using equation 8.1.

Equation 8.1 Calculation of power output by the Kingcycle test rig.

$$watts = 0.000136 \, (RPS)^3 + 1.09 \, RPS$$

$(RPS = revs \, s^{-1})$

8.2.2.1.2 Calibration Procedures

The calibration procedure of the Kingcycle relies on a deceleration curve stored in the computer's memory. Before calibration can commence however, the operator must ensure that the pressure in the rear bicycle tyre is standardised (100 psi), and that the subject doesn't move from the calibrated position on the bicycle. The operator selects the power value to which the rig is to be calibrated, and asks the subject to pedal up the flywheel to that power. Upon reaching that power the subject is instructed by the PC to stop pedalling and hold their position on the bicycle. The computer then matches the current deceleration curve with that of the ideal curve stored in its memory. If the current curve differs from the stored curve the PC will instruct the operator to raise or lower the subject's bicycle to match the decay curve. The rig is then deemed to be calibrated as long as the subject maintains the same position on the bicycle.

8.2.2.1.3 Kingcycle Maximum Minute Power Test.

The Kingcycle maximum minute power (MMP) test was used during these studies to determine a maximal power value at $\dot{V}O_2$max, and to simultaneously predict/measure $\dot{V}O_2$max. Previous research by Keen *et al.* (1991) produced a significant correlation coefficient (r=0.98, p<0.0001) between maximal minute power and $\dot{V}O_2$max.

Once calibration procedures have been completed, subject details of name, age, height, body mass, category and gender are entered into the programme. The starting power and ramp rate for an individual are selected on the basis of ability, gender, and body mass, with the aim of achieving volitional fatigue within 8-12 minutes. The computer displays a gradually increasing power output that the subject must match for a valid test to occur. When the subject cannot maintain the desired work rate, the test is terminated. The highest average power over any 60-second period during the test is termed maximum minute power. A schematic diagram of this test is shown in figure 8.1. This has been shown to be reproducible by Keen *et al.* (1991) who reported significant Test-retest reliability (r=0.98). Palmer *et al.* (1996) also reported reproducible figures for the Kingcycle testing rig for submaximal performance testing (coefficient of variation for time, 1.1 ± 0.9%). Keen *et al.* (1991) concluded that this method of testing was a valid method of measuring aerobic power of cyclists.

Figure 8.1 Schematic representation of the Kingcycle maximal minute power test. Maximal minute power is calculated from the highest work rate in any 60 second period of the test.
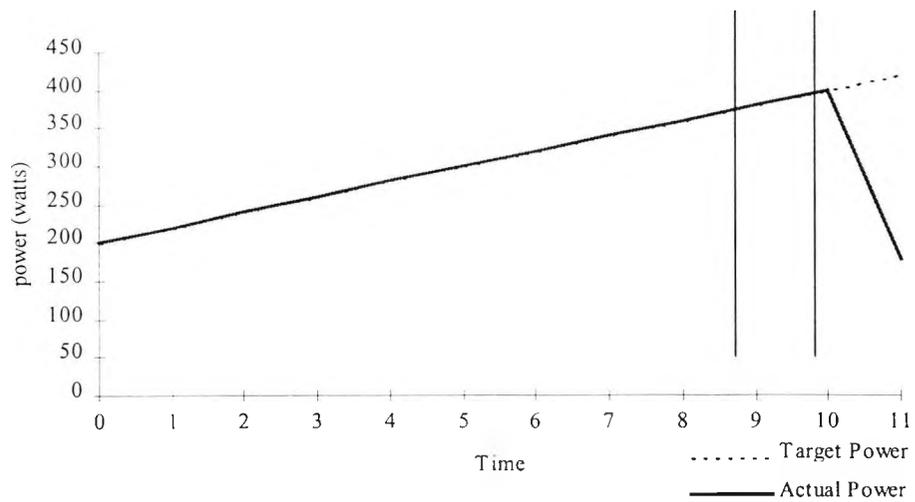
During the Kingcycle MMP tests carried out during these studies, heart rate was recorded during all tests (Polar, Kempele, Finland), and subjects were cooled by a large fan (Grants Electrical, Ballymena, UK).

Wind speed of the fan has previously been measured (Department of Geography, Canterbury Christ Church University College) using an annometer on repeated occasions. (Figure 8.2).

Figure 8.2 Relationship between wind speed and speed settings of the laboratory fan (n=6).
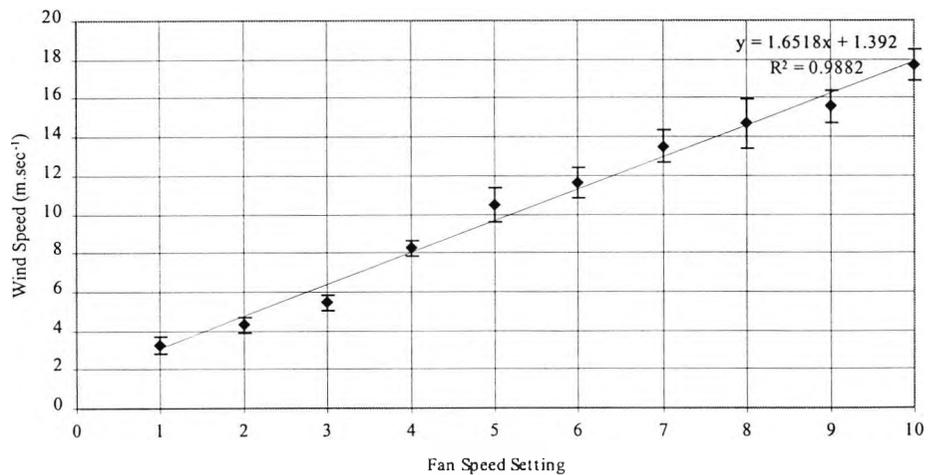
8.2.2.2 The SRM Powercrank System

8.2.2.1 Introduction

To test the validity and reliability of the Kingcycle system the measurement of power output was also recorded in conjunction with the SRM powermeter system.

The SRM powercrank system (Professional, Welldorf, Germany) has been designed to measure, record and evaluate power output during laboratory or field cycling exercise. During these studies both the 'Professional' and 'Research' crank were used. This system compromises a standard Shimano Dura-ace chainset and crank arm 172.5 mm in length. The chainset spider has been removed and a deformable alloy disc inserted containing 4 strain gauges (20 gauges in the research model). The chainset has two standard Shimano chainrings attached (53 and 39 teeth). The SRM Powercrank is mounted to a standard bottom bracket fitting and can be fitted on almost all bicycles.

The SRM system measures work output via the deformation of the alloy surrounding the strain gauges. The data is relayed telemetrically to a handlebar-mounted computer by a sensor placed perpendicular to the disc. The computer also stores heart rate (Polar technology), pedal cadence. During road cycling speed and distance is also recorded by the system by an additional front fork mounted sensor and a magnet attached to the spokes of the front wheel. The computer stores mean data over the sample period. Data can be stored at 1, 2, 5, 10, 20, 30, 60, 120 second intervals for all parameters. During this study the data was stored at 1 second intervals; the computer can store data for 7 hours 23 minutes at this sample rate. The data is then downloaded onto a PC and analysed using the SRM software package.

8.2.2.2.2 Calibration of the SRM Powercrank

The SRM Powercrank system undergoes two stages of calibration. The first is a dynamic calibration carried out before purchase, and during service work by Ingenieurburo Schoberer. A motorised dynamic calibration is carried out to determine the slope of increased deformation to the strain gauges. Accuracy of this calibration procedure has been confirmed by Keen (unpublished observations) using static methods of calibration. However, the manufacturer claims this method could lead to inaccurate data recordings. Jones and Passfield (1998) designed and built a rig from a Monark exercise cycle and a motorised treadmill to test the manufacturers accuracy claims, they reported figures that exceeded the claims of the SRM company (95% limits of agreement = 0.3, 1.1, 1.0 % on 3 separate units).

The operator carries out the second phase of calibration. Once the SRM Powercrank has been fitted to the bottom bracket axle of the bicycle by a crank bolt the zero position of the system has to be recorded. The SRM Powercrank has to be turned with no resistance so the chain is removed. The cranks are then spun forward. The handlebar mounted computer then reads the strain gauge reading (Hz) as the zero position. The zero position is then stored by the computer. The zero position is dependant upon the strain created when the SRM Powercrank is attached to the bottom bracket axle of the bike, and by the tension of the chain ring bolts that are fitted to the system. Once data has been downloaded into the PC corrections for the zero position can be performed.

8.2.2.3 Gas analysis; Covox Microlab

The Covox online gas analysis system was calibrated to known gas mixtures (Oxygen 21%, Carbon dioxide 5%, Balance nitrogen, Cryoservice Ltd, Worcester, England), and the pneumotachograph was calibrated

using a 3 litre syringe (Hans Rudolf Inc. Kansas, USA). Ventilation was converted to STPD, and data was displayed on a computer monitor at 30 second intervals during testing procedures. Variability of the Covox microlab system is displayed in table 8.1.

Table 8.1 Coefficient of variation during repeated trials using the Covox microlab.

Measure	Coefficient of Variation		
	$60 \, l \, min^{-1}$	$90 \, l \, min^{-1}$	$120 \, l \, min^{-1}$
% O_2	0.28%	0.33%	0.38%
% CO_2	4.6 %	3.03%	2.4%
VE	1.83%	1.77%	1.07%

Chapter Nine. Physiological Responses of Competitive Road Race Cyclist

9.1 Introduction

Determining relative exercise intensity for endurance tasks is a common practice in Sport Science for analysing the physiological responses of athletes, or for developing protocols for intervention studies. However, the criteria for relative exercise intensity are varied in the literature, with percentages of $\dot{V}O_2$max, heart rate, maximal power, lactate threshold, or ventilatory threshold being used. In terms of correlation to performance, previous research has highlighted the importance of $\dot{V}O_2$max and Kingcycle maximal minute power as key determinants (e.g. Malhortra *et al*., 1985. Davison *et al*., 1997). Other research has focused upon the importance of the BLac⁻ response (Bishop *et al*., 1998), and the percentage of $\dot{V}O_2$max sustained (as opposed to absolute $\dot{V}O_2$max) as a key determinant of performance (Coyle *et al* 1991). Coyle presenting data at the 1998 ACSM conference indicated that relative exercise intensity ought to be derived from a Lac⁻ threshold test. A further possible determinant of performance outcomes is the efficiency of movement and the energy cost of a given task (Cavanagh and Kram, 1995). As the MMP test is a good predictor of performance, the aim of this study was to evaluate the Kingcycle MMP test, and record the physiological responses during submaximal exercise intensities relative to an individual's maximal data, and to evaluate the test as a method of ascertaining a relative exercise intensity in a homogenous population of racing cyclists.

9.2 Methodology

Eleven trained male competitive cyclists were recruited on the basis of previous expertise at road racing to participate in this study (8.1). Prior to any physiological assessment laboratory and subject details were recorded (8.2.1). Following a period of familiarisation to the testing procedures subjects were required to return to the laboratory on five separate occasions (one visit per week) for a series of two maximal, and six submaximal exercise tests. All subjects had previously undergone Kingcycle evaluation for coaching purposes.

9.2.1 Familiarisation

During familiarisation (and data collection) subject's entered the laboratory having refrained from training during the previous 24 hours, they were also instructed to maintain their normal training and dietary routines during the testing period.

Familiarisation consisted of a Kingcycle MMP test (8.2.2.1) on the subject's own bicycle equipment. Following a warm up to their own requirements, subjects were fitted with a face mask (Hans Rudolf. Kansas city, USA), and connected to an online gas analysis system (Covox, Exeter, UK). Subjects indicated when they were ready to start the test, and were instructed to match the desired power output displayed by the PC. They were verbally encouraged during the test until the required power output could no longer be matched. Three minutes post test, a thumb prick blood sample was taken (Unilet lancets, Owen Mumford, Oxford, England) and analysed for Lac⁻ and glucose (Yellow Springs Instrument, 2300 Stat Plus, Ohio USA).

9.2.2 Experimental procedures

9.2.2.1 Tests 1 and 2. Kingcycle MMP tests.

Following dietary and training control as described above, subjects underwent the same protocol as described above during familiarisation on two consecutive visits to the laboratory 1 week apart. Measurement of MMP, maximum heart rate, $\dot{V}O_2$max, and 3 minute post B[Lac⁻] were recorded. Two consecutive tests were completed for test/retest analysis.

9.2.2.2 Tests 3, 4 and 5. Kingcycle Submaximal tests.

Following identical preparation to the previous tests, subjects underwent six randomised submaximal tests at intensities corresponding to 60%, 65%, 70%, 75%, 80% and 85% of the highest MMP recorded from tests 1 and 2. Two tests were completed during each visit to the laboratory with the lowest intensity condition performed first, with a 30-minute break between tests. Intensities for each visit to the laboratory for submaximal testing were paired at the following percentages of MMP (60% and 85%, 65% and 75%, 70 and 80%). Subjects were ramped up to the desired work output over a 5 minute period and on reaching the desired work rate were instructed to maintain their work output to ± 5 watts for a 10 minute period for a valid test. Online gas analysis (Covox) was conducted throughout all of the submaximal tests. Heart rate was also recorded throughout the test (Polar). Thumb prick capillary blood samples were taken at 4 and 9 minutes during each submaximal test and analysed for B[Lac⁻]. On completion of all tests subjects were allowed to warm down at their own desired intensity.

9.2.3 Statistical analysis

A one-way repeated measures ANOVA was used to establish if there were any differences between trials for the repeated MMP data, and the efficiency and economy data from all exercise intensities. The coefficient of variation was also calculated on repeated MMP trials. Power output was calculated at B[Lac⁻] of 4 mmol.l⁻¹, and the power output corresponding to the data point preceding an increase in B[Lac⁻] of 1 mmol.l⁻¹. The power output was also calculated where the blood lactate response deviated from baseline values using a visual interpretation of the lactate curve data.

9.3 Results

All 11 subjects completed the maximal testing phase of this study, however, only 10 subjects completed all of the submaximal trials. All 11-subject baseline details are reported in table 9.1.

Values for the maximal minute power tests are shown in figure 9.1, trial 1 was conducted without gas analysis and trial 2 with gas analysis sampled. No significant differences were observed between the two trials (P=0.96). The coefficient of variation between the two trials for maximum minute power was 1.72% (95% CI - 1.01-2.85%), SEE 2.60 (95% CI - 1.51-4.26).

Table 9.1 Baseline subject details for 11 subjects.

(mean ± S.D)

Subjects	n=11
Age	29 ± 9 years
Height	1.75 ± 0.05 m
Mass	72.6 ± 5.4 kg
MMP	test 1 380.6 ± 35.3 watts
	(test 2 381.2 ± 28.5 watts)
$\dot{V}O_2$max l min^{-1}	4.97 ± 0.57
$\dot{V}O_2$max ml kg^{-1} min	68.43 ± 6.02

Figure 9.1 Comparison between two Kingcycle maximum minute power tests (one week apart). Test two also included gas analysis.
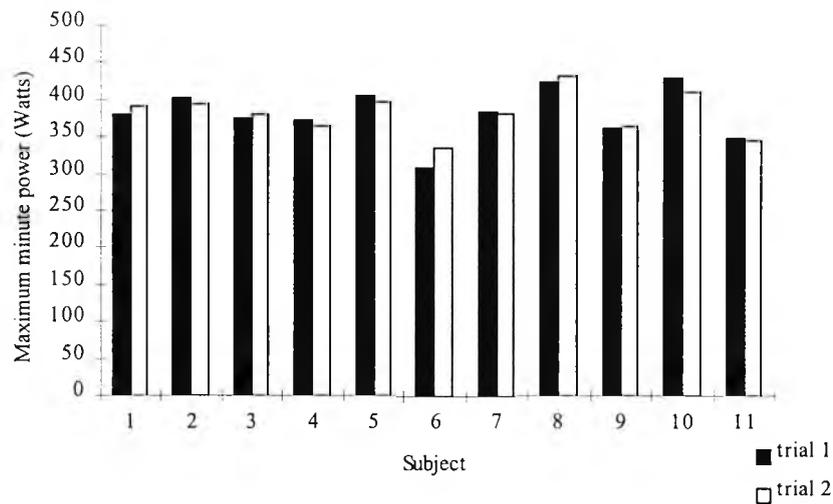
Table 9.2 below shows the heart rate and B[Lac$^-$] during the maximal minute power test, and during the 10-minute submaximal work bouts.

Failure to establish a baseline B[Lac$^-$], and missing data points resulted in the loss of one subjects data using two of the methods of lactate curve analysis. Strong positive correlations were found between MMP and; power output corresponding to B[Lac$^-$] at 4mmol.l^{-1} (r=0.92, n=10); power output corresponding to the data point preceding and increase in B[Lac$^-$] of 1 mmol.l^{-1} (r=0.88, n=9); power output at a visual interpretation of lactate threshold (r=0.94, n=9). The mean data for the percentage of MMP at the various blood lactate concentrations are shown in table 9.3. The lactate power relationship for each subject are presented in appendix 1.

Table 9.2 Heart rate and Blood lactate responses to exercise at different relative exercise intensities.

(mean ± S.D.)

Relative Work Load	Heart Rate (beats·min⁻¹)	Relative Heart Rate (%)	Blood Lactate Concentration (mmol.l⁻¹)
60% MMP	145.6 ± 7.3	76.4 ± 4.7	2.51 ± 0.56
65% MMP	151.9 ± 7.8	79.7 ± 4.7	2.57 ± 0.70
70% MMP	160.5 ± 11.4	84.2 ± 5.6	2.87 ± 0.77
75% MMP	168.7 ± 10.9	88.5 ± 5.6	5.06 ± 1.05
80% MMP	173.3 ± 10.9	90.9 ± 5.5	7.11 ± 1.65
85% MMP	179.7 ± 10.2	94.2 ± 5.2	11.93 ± 2.09
100% - MMP	190.9 ± 10.0	100	12.13 ± 2.76

Table 9.3 Power output at various blood lactate concentrations. (mean ± SD)

Method	% MMP
B[Lac⁻] 4mmol.l⁻¹	72.4 ± 2.6 %
Data point preceding a 1 mmol.l⁻¹ increase in B[Lac⁻]	70.6 ± 3.9 %
Visual interpretation	68.9 ± 2.2 %

The three methods of analysing the BLac⁻ response to incremental exercise also correlated significantly (table 9.4)

Table 9.4 Relationship between 3 different methods of assessment of the BLac⁻ response during incremental work (n=9).

Method	Correlation coefficient	95% CI
B[Lac⁻] 4mmol.l⁻¹ vs Data point preceding a 1 mmol.l⁻¹ increase in B[Lac⁻]	r= 0.67*	0.01-0.92
B[Lac⁻] 4mmol.l⁻¹ vs Visual interpretation	r= 0.91#	0.66-0.98
Visual interpretation ¹ vs Data point preceding a 1 mmol.l⁻¹ increase in B[Lac⁻]	r= 0.77*	0.22-0.95

*p<0.05

#p<0.01

Respiratory, gross mechanical efficiency and economy values are shown in Table 9.5. Figure 9.2 shows the $\dot{V}O_2$, $\dot{V}CO_2$, RER, responses at the submaximal work bouts. There were no significant differences for

economy (p=0.29), or gross mechanical efficiency (p=0.57), between different exercise intensities (Figures 9.3 and 9.4).

Table 9.5 Respiratory, economy and gross mechanical efficiency data from exercise trials at different exercise intensities. (mean ± SD).

Relative Work Load	$\dot{V}O_2$ l·min^{-1}	$\dot{V}CO_2$ l·min^{-1}	RER	Gross Mechanical Efficiency (%)	Economy (%)
60% MMP	3.39 ± 1.86	2.94 ± 0.34	0.86 ± 0.02	19.54 ± 1.58	66.07 ± 5.39
65% MMP	3.71 ± 0.39	3.23 ± 0.39	0.87 ± 0.03	19.93 ± 0.97	67.92 ± 3.03
70% MMP	3.91 ± 0.35	3.59 ± 0.33	0.92 ± 0.03	20.18 ± 1.00	68.28 ± 2.36
75% MMP	4.25 ± 0.34	3.95 ± 0.31	0.93 ± 0.03	19.63 ± 0.67	68.00 ± 2.66
80% MMP	4.50 ± 0.25	4.41 ± 0.36	0.98 ± 0.06	20.6 ± 1.4	71.56 ± 5.05
85% MMP	4.69 ± 0.51	4.64 ± 0..56	0.99 ± 0.05	19.9 ± 1.56	69.51 ± 5.75

Figure 9.2 Mean respiratory responses during 10-minute submaximal exercise intensities at different percentages of maximum minute power.
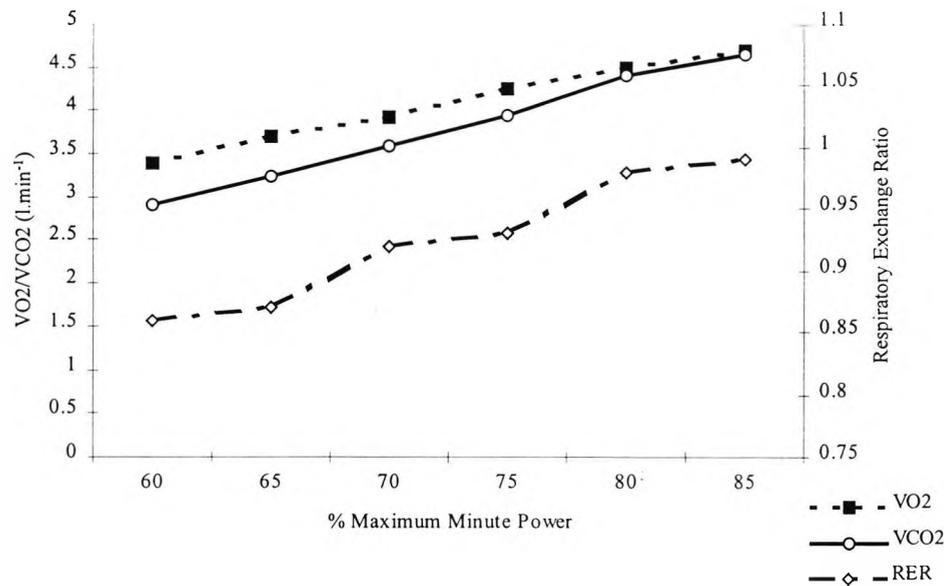
Figure 9.3 Mean economy (watts.l^{-1}) during 10 minute submaximal exercise intensities at different percentages of maximum minute power.
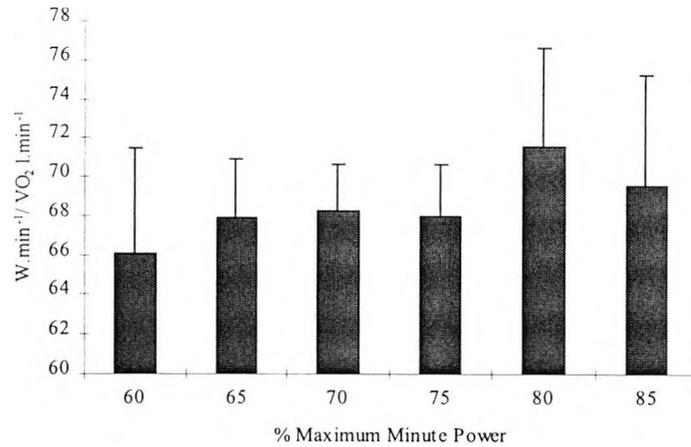
Figure 9.4 Mean gross mechanical efficiency during 10-minute submaximal exercise intensities at different percentages of maximal minute power.
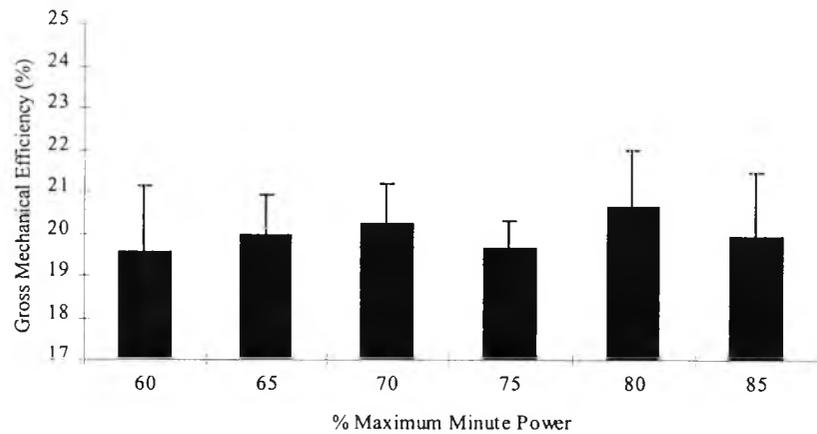
9.4 Discussion

9.4.1 Characteristics of subjects

The mean measured $\dot{V}O_2$max group data from this study (~68 ml.kg^{-1}min^{-1}) compared favourably to previous values reported in the literature by Vrijens *et al.* (1982) of 65.7 ml.kg^{-1}min^{-1} in Belgium professional riders, and also the findings by Bonjer (1979) (67.6 ml.kg^{-1}min^{-1} in Dutch elite riders). However, they do not reach the maximal figures recorded by White *et al.* (1982) (77.4 ml.kg^{-1}min^{-1} in British elite riders) or predicted values by Brennan *et al.* (1994) who reported values and 78.4 ml.kg^{-1}min^{-1} in Northern Irish Commonwealth Games squad. Although the laboratory measures do compare between the subjects used in this study and those of Vrijens *et al.* (1982) and Bonjer (1979) the subjects used in this study do not compete at an elite level. This could possibly be attributed to differences in testing protocol for

72

the attainment of $\dot{V}O_2$max. Two subjects hold a British 'elite' ranked licence (although it is generally a lower grade than continental counterparts).

The differences in performance relative to the similar laboratory tests could simply be explained by the pitfalls of comparing data from different laboratories using different measurement systems, and different methods to ascertain $\dot{V}O_2$max. If we assumed cross comparison could be applied, club level cyclists (from this study, and Brennan et al., 1994) are attaining similar figures to Professional/elite riders competing 15-20 years ago. It could also indicate that factors other than $\dot{V}O_2$max differentiate between some very ordinary (in terms of performance ability - British 3rd category) riders when compared to elite professional riders. This has been considered by Vrijens et al. (1982) who concluded that maximal figures did not differ between sub elite and elite riders, but power/$\dot{V}O_2$ at threshold levels was the differentiating factor.

9.4.2 Reliability of Kingcycle MMP testing

The figures for maximum power output recorded by the Kingcycle ergometer showed no significant difference between two tests, conducted seven days apart. The coefficient of variation between tests was 1.72 % for power. This figure is similar to reported figures for submaximal performance cycling (for time) reported by Palmer et al. (1996), who reported coefficient of variations for time (derived from power) of 1.1 % for simulated 20 km performance testing, and 1.0 % for 40 km performance testing. On a longer endurance test Schabort et al. (1998) reported a coefficient of variation (time) of 0.93% for a 100 km Kingcycle laboratory performance test. This suggests that both the Kingcycle performance and MMP data is reliable. A further conclusion from this data is that the method of 'controlling' subjects during the first study by asking them to prepare for each test in a similar fashion to competition racing, eating well, and no training on the day preceding the test. This appears to be sufficient in minimising any variation in maximum power. Data on the effects of prior training on Kingcycle maximal test performance has been collected by Balmer (unpublished observations) during a study on repeated maximal and 70 km performance testing. They reported significant reductions in Kingcycle maximum minute power with hard training/racing the day before or even 2 days before the test procedures.

9.4.3 Physiological responses during submaximal testing

9.4.3.1 BLac⁻ response to submaximal exercise

All three methods of blood lactate assessment in this study all correlated to MMP strongly (r= 0.88-0.94). However, only the 4 mmol.l⁻¹ method allowed for power output detection in all subjects. The mean % MMP derived from these methods showed similarity (~69 to 72.5% of MMP), however smaller variability was around the mean was recorded using the visual method of blood lactate interpretation (table 9.3). This therefore explains the high correlations obtained by Davison et al. (1997) between endurance performance (16 km) and MMP. From these results, it is apparent that in well trained cyclists the results obtained from the MMP test could simply be a reflection of BLac⁻ kinetics. As $\dot{V}O_2$max correlates significantly (r=0.98. Keen et al., 1991) with MMP using the 20 watts per minute protocol, it is therefore not surprising that

$\dot{V}O_2$max obtained from Kingcycle testing also significantly correlates with endurance performance (r=0.86. Dobbins *et al.*, 1996).

As exercise intensity increased, there was also an increase in the standard deviation for the group data for B[Lac⁻]. This suggests that although there was a similar inflection point for BLac⁻ between subjects, there are smaller differences in the BLac⁻ at lower as opposed to higher work rates. Bishop *et al* (1998) concluded that the B[Lac⁻] was a reflection of glycogen utilisation, in which case at higher exercise intensities rates of glycogen utilisation may differ, which may have implications for endurance performance. Data collected using SRM technology by Davison *et al.*, (1999) however suggests that during endurance tasks lasting from one hour to twelve hours cyclists maintain a very similar percentage of their maximum power, the exact percentages sustained being governed by the duration of the event.

During the highest exercise intensity (85% MMP) a mean B[Lac⁻] of 11.28 mmol.l⁻¹ was reached. Submaximal values at 85% of MMP actually produced a higher standard deviation than the maximal B[Lac⁻] recorded post MMP test (table 9.2). This data shows that well trained cyclists can sustain exercise intensities for a ten-minute period in substantial excess of the BLac⁻ threshold, and can tolerate significant B[Lac⁻] for a sustained period of time. This confirms previously presented data by Coleman *et al.* (1995) who reported B[Lac⁻] rising to over 9 mmol.l⁻¹ during simulated 16 km performance testing (lasting approximately 23 minutes).

No significant difference (p>0.05) was found between the B[Lac⁻] recorded post MMP test and the values at 85% MMP. There is a possibility that the amount of energy derived from the anaerobic breakdown of glycogen may have an upper limit, despite differences in exercise intensity (85% MMP vs 100% MMP) and duration (10 minutes vs ~4 minutes above B[Lac⁻] threshold during MMP). Spreit, (1995) reported that although the glycolytic flux is not believed to be limited by typical metabolic changes during heavy exercise (e.g. pH) the contribution of ATP from glycolysis during exhausting exercise was dependant upon the lactate and associated ion movements into open circulation. Spriet (1995) indicated that circulating lactate concentrations of over 9 mmol.l⁻¹ may alter lactate efflux, and reported a 30% decrease in the contribution of glycolysis to ATP production with closed circulation. Coyle, presenting data at the American College of Sports Medicine conference (Orlando, 1998) indicated that work output at the lactate threshold was the most effective method of establishing a relative exercise intensity, (in terms of metabolic response). It appears that the Kingcycle maximum minute power test is a good indicator of power output at the blood lactate threshold, although the complexity of physiological responses at different relative intensities warrants further investigation. Each submaximal work stage during this study had a duration of 10 minutes. The implications of longer work stages (>60 minutes) at the various percentages of MMP may further substantiate the findings of Bishop *et al*, (1998), and further investigate rates of substrate depletion at fixed percentages of MMP (lactate threshold) during endurance exercise.

A further conclusion from these findings is the critical importance of Kingcycle maximum minute power output. If similar percentages of power output can be sustained during the steady state condition, plus the relatively normalised BLac⁻ response during set percentages of maximum minute power, then the ability to achieve high figures is possibly going to determine time trial performance. In theory a high maximum power, coupled with a reduced frontal area (Wright *et al.*, 1994) would produce a good time trialist, with body mass being a factor during hillier races (Rollings *et al.* 1985).

9.4.3.2 Efficiency calculations from submaximal exercise intensities

During the submaximal study, efficiency values revealed similar figures (18-23%) to those previously reported by Coyle *et al.*(1992) who recorded figures from 18-26% in a group of well-trained cyclists. During the tests at the different relative exercise intensities, there were minor fluctuations of the mean group data of ~1% during these trials, supporting the findings of Nickleberry and Brooks (1996). Therefore the efficiency calculations of trained cyclists possibly do not play such an important role in the outcome of events when compared to the 12-15% differences that have been postulated in runners (Cavanagh and Kram, 1995). The nature of road cycling however has a significantly longer duration than 10 minutes, previous studies have not reported if these values change over longer time periods, where in theory even the small differences may have an impact upon performance.

9.4.3.3 Heart rate and $\dot{V}O_2$max responses to Submaximal exercise

The relationship between percentage maximum heart rate and the corresponding percentage of $\dot{V}O_2$max have previously been plotted by McArdle *et al.* (1996) using four other studies to construct their table (table 9.6). The data from this study shows a similar pattern during the lower exercise intensities. However, during the last two work rates of 80 and 85% of MMP the percentage of $\dot{V}O_2$max higher for a given heart rate. From this study, equation 9.1 was derived.

Table 9.6 The relationship between heart rate and $\dot{V}O_2$ (from McArdle *et al.*, 1996)

% Maximum Heart Rate	% $\dot{V}O_2$max
50	28
60	40
70	58
80	70
90	83
100	100

Equation 9.1 $\dot{V}O_2$ and heart rate relationship during submaximal exercise

$$\%\dot{V}O_2max= 1.4494 \text{ x (\% maximum heart rate) - 42.289} \qquad (r=0.996, p<0.001).$$

Compared to the McArdle *et al.* (1996) data, these cyclists would be utilising a higher proportion of $\dot{V}O_2max$ during steady state cycling for a given percentage of maximum heart rate. From the data presented therefore, to elicit training adaptation in the region of the BLac⁻ inflection point, (occurring between 70-75% of MMP), training at heart rates between 84.2 and 88.5% of maximum will elicit these changes. This corresponds to a $\dot{V}O_2$ of between 78.7-85.5% of maximum values.

Chapter Ten. Data Collection From Competitive British Cycling Federation Cycling Events

10.1 Introduction

One of the major roles of the coach is to prepare athletes for competition. This is achieved by establishing the demands of the sport, and developing training methods to enhance athletic ability, to match the demands of the sport. For the coach to be successful, a detailed knowledge of the competitive demands is required. During road race cycling, heart rate data has previously been recorded to ascertain the physiological demands of the sport (Palmer *et al.*, 1994), with Sport Scientists using this data to replicate the sporting demands in the laboratory (Palmer *et al.*, 1997). Telemetric heart rate recording and the subsequent playback of the data have enabled subjects to compete without data collection interfering with their performance. The development of SRM powercrank technology also has the same benefits, and allows the recording of not only heart rates, but also of power output, pedal cadence, speed, time and distance covered. The aim of this study was to record these variables during competition (SRM), and compare them to maximal values recorded in the laboratory (Kingcycle MMP test. 8.2.2.1). An initial study was required to investigate the relationship between SRM and Kingcycle measurement of power output.

10.2 Comparison between Kingcycle and SRM power measurement.

The aim of this study was to evaluate the relationship between two methods of calculating power output, with SRM technology being used for the collection of field data and the Kingcycle ergometer used in the laboratory setting. The relationship between the two systems would be important when comparing field and laboratory data. Previous unpublished research by Keen indicated there might be a discrepancy between the two measures of power output.

10.2.1 Method

Thirteen undergraduate students gave informed consent (8.2) and had initial details recorded (Table 10.1).A bicycle fitted with the SRM powercrank system was attached to the Kingcycle test rig, and calibration procedures were followed for both systems (8.2.2.1.2, 8.2.2.2.2). Once calibrated, subjects were required to exercise on the bicycle at five different intensities with each work stage lasting one minute. The subjects rode to Kingcycle power output values displayed on the PC screen. A recovery period of approximately two minutes was allowed between each work stage, with subjects instructed to be exercising at the required intensity ten seconds before that work stage commenced. The selection of work stage intensity was dependant upon the individual subject's ability. If at any stage during the work interval the power output dropped from the desired figure, the work interval was eliminated from the analysis. The mean power for each work stage was recorded by the two respective systems.

10.2.2 Statistical Analysis

The data for each 60-second work period were compared using a Pearsons Product Moment correlation. The data was checked for heteroscedasticity by correlating the absolute residuals and the fits (Neville, 2000). A repeated measures ANOVA was used to establish if there were any differences between the data from the two methods of measuring power output. Ratio limits of agreement were also calculated using the methods described by Bland and Altman (1986).

10.2.3 Results

Table 10.1 Subject details. (mean ± SD).

Subjects	n=13
Age	25 ± 7 years
Height	1.76 ± 0.10m
Mass	70.2 ± 10.4 kg

The mean work rate undertaken by the subjects was 183.8 ±106.8 Kingcycle watts, which equated to 167.0 ± 97.8 SRM watts. There was evidence of heteroscedasticity within the data therefore logarithmic transformation was employed. The ratio limits of agreement between Kingcycle and SRM power were 0.90 (95% CI = 0.90-0.91) ×/÷ 1.04.

Figure 10.1 The relationship between Kingcycle and SRM measures of power output. (dotted line represents the line of identity)
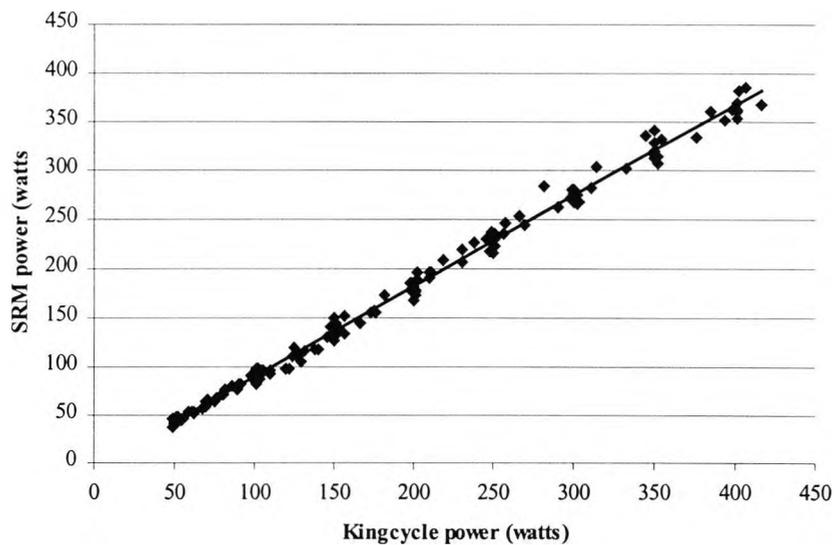
$y= 0.9145x-1.0674$

$r2=0.9957$

There was a significant difference between the two systems for the logarithmically transformed data (p=0.035). The Kingcycle measure of power output was 9.62 ± 4.22% higher than the SRM system. Figure 10.1 shows the relationship between Kingcycle and SRM watts over the range of powers sampled for each minute. The correlation coefficient revealed a significant relationship (r=0.99, p<0.01) between the two measures of power output.

10.2.4 Discussion

Power output during these studies was monitored by two independent systems, the Kingcycle test rig, and the SRM power meter. Data recorded by both systems showed a significant linear relationship (r=0.99, p<0.05. 95% CI - 1.6-32 W) at power outputs ranging from 50 - 500 watts, however there was a significant difference between the two systems for the absolute values attained for power output. The mean difference between SRM and Kingcycle power was 9.6%, a correction factor that has now been employed by the latest Kingcycle software. The accuracy of both systems has previously been investigated, data collected by Jones and Passfield (1998) using dynamic methods to calibrate the SRM system have shown that the SRM system is reading 'true' watts (validity 1%) and the Kingcycle appears to be the piece of apparatus that is over reading power output by 9.6%. The results of Jones and Passfield (1998) indicated greater accuracy than the claims made by the SRM Company. These figures compare favourably with the unpublished observations by Keen, who reported similar (1%) deviations using a static method of calibration. The SRM company do however state a dynamic calibration should be performed to ensure validity. Therefore the SRM system was returned at regular intervals to Germany for dynamic calibration. The calibration values remained stable with only slight modification during servicing.

10.3 Data collection from road cycling events

10.3.1 Method

Nine competitive male cyclists of mixed ability were recruited to participate in this study. All subjects had previously been involved in physiological testing in the Laboratory. Following the collection of laboratory and subject details (8.2), Subjects underwent a Kingcycle MMP test (8.2.2.1.3). Once the bicycle was attached to the Kingcycle rig, warm up began at a self-paced intensity, with subject's indicating when they were ready to begin the test. Maximal minute power, predicted $\dot{V}O_2$max and maximal heart rate were recorded.

Within ± 7 days of the Kingcycle MMP test, subjects had pre-entered a competitive British Cycling Federation race. Race duration varied, as did the standard of the competition (relative to the category of racing licence held by the individual). SRM powercranks were fitted to the subject's bicycle two days prior to the race, and all calibration procedures were completed (8.2.2.2.2). Just prior to the race, a further calibration was performed.

During the event subjects were able to view power, heart rate, cadence, speed, and time elapsed with the only instruction that their aim was to win that race. Some subjects recorded more than one competitive race

during the ± 7-day period. Measured parameters were stored by the handlebar-mounted computer at one-second intervals, and downloaded following the race.

For three of the local courses, the profile of the course was determined using Ordnance Survey Land ranger maps (188, 189, 197).

For comparison with the Palmer *et al.* (1994) study, SRM data were also collected during two-stage race cycling events with a smaller subject group (n=4).

10.3.1.1 Kingcycle Power Test (Wingate).

Five of the subjects returned to the laboratory to undergo a Kingcycle anaerobic power test. The Kingcycle power test is a 30 second sprint test designed to measure anaerobic capacity based upon the classic Wingate protocol. Upon arrival at the laboratory, details of the subject were recorded and SRM Powercranks were mounted on the subject's bicycle and calibration procedures were followed. The subject's bicycle was then attached to the Kingcycle test rig and the additional sprint rig attached. The sprint rig consists of increased supports extending from the rear of the Kingcycle, connecting a nylon band from the rig to the subject's bicycle saddle rails. This is used to prevent any slippage of the rear wheel of the bicycle on the Kingcycle roller. Following calibration, and warm up, subjects selected their chosen gear ratio (previously selected from familiarisation trials) with which they felt they could achieve the highest work output for the test period. Subjects were then instructed to reduce their power output to 30 watts. The test began when subjects indicated they were ready to start the test. The subjects were instructed to accelerate as fast as possible and to cover the furthest distance during the 30-second test. Subjects were only allowed to view time expired during the test. Power output was recorded by the Kingcycle rig and the SRM system throughout the test. Values for peak power, mean power, and distance covered were recorded during the test.

10.3.2 Statistical analysis

Laboratory and field heart rate and power data were compared, using a one-way ANOVA. The laboratory data was also correlated to the finishing positions of all of the one-day races using a Spearmans rank correlation.

10.4 Results

All 13 subjects completed the laboratory testing protocol, however, during the races, two subjects failed to record the complete race duration during field data collection. One subject punctured during the road race, the other crashed out of their race, both riders failed to finish. Subject details for the 11 subjects are listed in table 10.2. Predicted $\dot{V}O_2$max values are calculated from Keen *et al.* (1991). A total of 16 races were recorded during this study. Raw data, and smoothed data from these races are shown in appendix 2.

The mean race duration was 111.3 ± 64.9 minutes. Finishing positions from the field data ranged from 1st place to 53rd place. No significant correlations between laboratory test indices and finishing position were recorded (p>0.05).

Table 10.2 Subject details from the laboratory visit ± 7 days from the competitive cycle race. (mean ± S.D.).

Subjects	n = 11
Age (years)	29 ± 10
Height (m)	1.78 ± 0.04
Mass (kg)	72.9 ± 3.8
Maximum Minute Power (SRM) (W)	394 ± 36
Predicted $\dot{V}O_{2max}$ ($l\cdot min^{-1}$)	4.86 ± 0.42
Predicted $\dot{V}O_{2max}$ ($ml\cdot kg^{-1}\cdot min^{-1}$)	67 ± 5
Maximum Heart Rate (beats·min^{-1})	190 ± 5

Peak work rate (PWR) during the races are shown in Figure 10.2, peak values for sampling rates of 1, 5, 10, 30, 60, 90, 120 seconds are reported. The relationship between peak power output values and the race duration are shown in figure 10.3.

All subjects (n=5) recorded a higher power output in the field compared to the laboratory sprint test (1186.2 ± 57.5 vs 937 ± 105 watts) for peak power (p=0.001).

Figure 10.4 shows a comparison between data recorded by Bjarne Riis (Tour de France winner 1996) during the Amstel Gold World Cup race (>6 hours duration), and the data collected from British races.

Figure 10.2 (Mean) peak work rates for seven sampling periods during competitive road race events. (n=16).
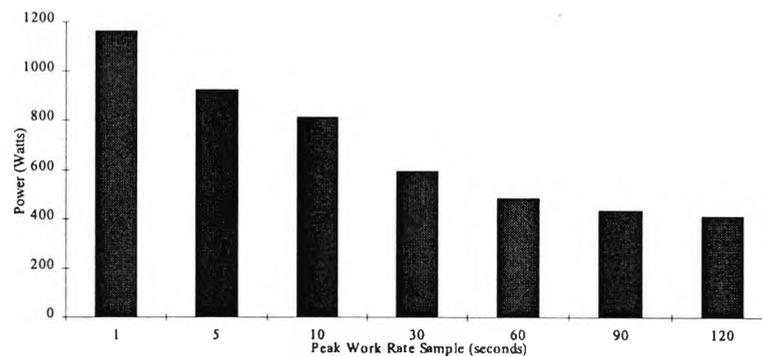
Figure 10.3 (Mean) Relative peak work rates over the duration of competitive road race cycling events.
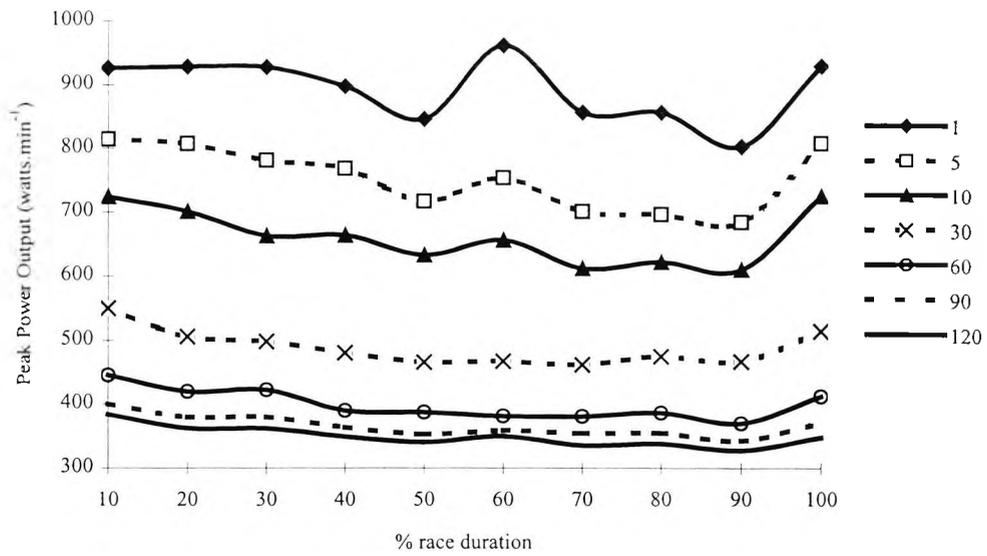
Figure 10.4 A comparison between mean power from road race data (GB races, n=16) and one elite performance
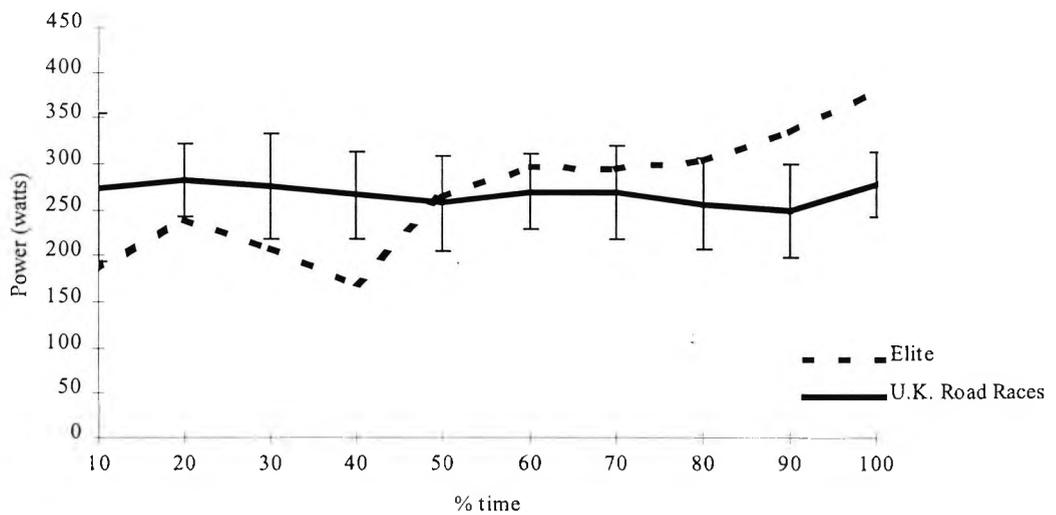
10.4.1.1 Relative exercise intensity

Mean data from the British cycling races is shown in table 10.3. Subjects averaged 67.2 % of maximum minute power during the races. During 10 of the 16 recorded races, heart rate was simultaneously recorded,

this equated to 88.5 % of maximum heart rate. Mean race pedal cadence was 76.8 ± 4.0 revolutions per minute.

Table 10.3 Mean (absolute and relative) data from the field for Power output, Heart rate and pedal cadence. (mean $\pm s$)

Absolute Power	264 ± 8.8 watts
% Maximum minute power	67.2 ± 7.6 %
Heart Rate	169 ± 7 beats min[-1]
% Heart Rate$_{max}$	88.5 ± 2.9 %
Pedal Cadence	74.5 ± 5.2 revolutions per minute

The distribution of power output is shown in figure 10.5. The results showed that $39.1 \pm 9.2\%$ of the race duration was spent above maximum minute power. During competition, $12.7 \pm 6.1\%$ of the race duration was spent at zero watts, where the bicyclists were 'free wheeling'. Deducting the time spent at zero power output and zero cadence (12.7% of race duration) results in a mean pedal cadence of 84.9 revolutions per minute, and likewise a power output of 302 watts - 80% of maximum minute power for the time working.

Appendix 2 shows the traces from three races (smoothed over 60 seconds) relative to the course profile over which those races took place, Forestside, Frant, and Godmersham.

Figure 10.6 shows the distribution of heart rate response during the ten races. To allow direct comparison, the distribution categories selected are the same as those selected by Palmer *et al.*, (1994). Subjects spent $6.0 \pm 7.9\%$ of the race duration between 71-80% of maximum heart rate, $57.2 \pm 23.7\%$ of race duration between 81-90% maximum heart rate, and $34.9 \pm 28.0\%$ of race duration between 91-100% of maximum heart rate.

For the 10 races with heart rate being recorded, the mean power output during the races was 267 ± 34 watts, and the mean heart rate was 169 ± 7 beats min[-1]. From the laboratory maximum minute power test, a heart rate: power relationship was calculated for each rider, (one subject's data was omitted from these calculations Subject 3, due to the race being stopped for a period of 5 minutes - table 10.4).

Actual mean heart rate was significantly higher in the field (p=0.006) compared to the maximal minute power test (169 vs 158 beats min[-1] for actual and predicted heart rate). Correlation analysis revealed only a moderate correlation between duration of the race and the difference between actual and predicted mean heart rate (r=0.40).

Figure 10.5 Relative distribution of exercise intensity during competitive road race cycling events
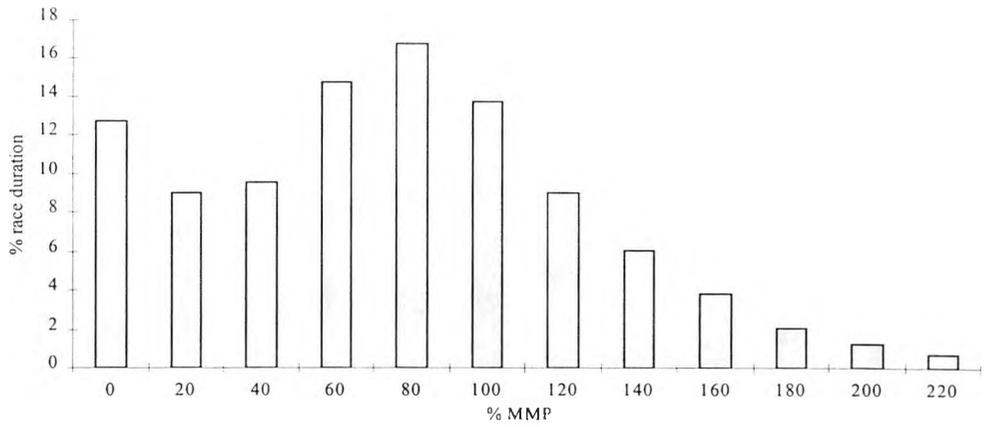
Figure 10.6 The relative distribution of heart rates during competitive road race cycling events.
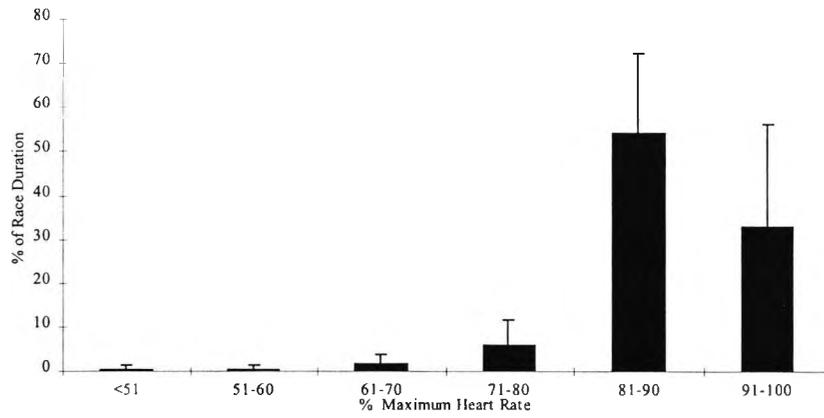
Figure 10.7 Heart rate response during each 10% portion of the races; comparison to the laboratory predicted heart rate. (* denotes p<0.05).

84
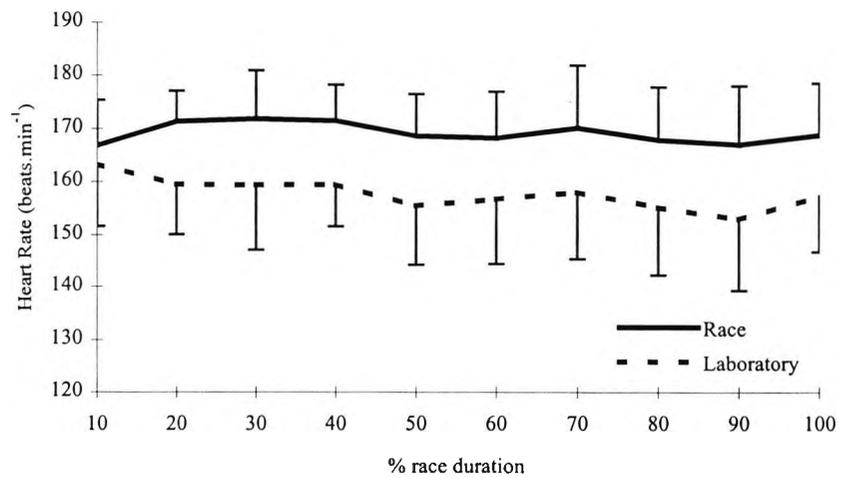
Table 10.4 Actual and predicted power output from laboratory and field data.

Subject	Actual Power (watts)	Actual Heart Rate (beats min^{-1})	Laboratory Predicted Heart Rate (beats min^{-1})
1	343	169	155
2	271	185	181
3	224	173	134
4	233	159	146
5	286	166	159
6	244	169	155
7	254	172	157
8	257	169	158
9	291	163	158
10	273	160	153
Mean	267	169	156
S.D.	34	7	11

The differences between actual and 'laboratory predicted' heart rate response are shown in figure 10.7. There were significant differences between these two methods, in all but two of the portions of the race - the first 10% and the 70% portions.

10.4.1.2 Energy demands of stage race cycling; FBD Milk Ras

Further field (SRM) data were collected without laboratory-based data on one rider from an international 'amateur' stage race (FBD Milk Ras - Ireland). During the 9-stage race the rider finished 4th overall on general classification, and recorded one stage win (stage 3). The mean power output was 235 ± 16.35 watts, heart rate 144 ± 8 beats min^{-1}, and pedal cadence 80.79 ± 4.5 rev min. The mean mechanical energy expenditure was 2823 ± 887 kj.day^{-1}. Figure 10.8 shows the stage-by-stage breakdown of the power output and heart rate response during the 9-stage race.

Using the SRM analysis software, a 4-minute smoothing factor was run over the heart rate and power output data. By shifting the heart rate response forward (due to a time lag between power output and heart rate response), the software then calculates the highest correlation coefficient between the two variables. From this a physical work capacity (PWC) at heart rate 150 is calculated.

Figure 10.9 below shows the calculated PWC 150 values for the nine stages during the FBD Milk Ras. There is a significant linear increase over time ($r=0.891$, $p<0.05$).

Figure 10.8 Mean Power output and heart rate responses from a competitive stage race cycling event. (n=1).
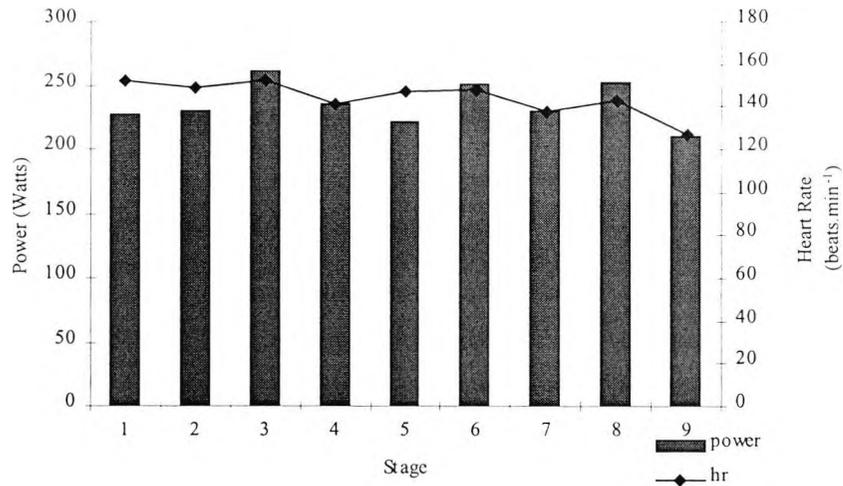
Figure 10.9 Predicted physical work capacity at heart rate 150 beats·min^{-1} during competitive stage race road cycling. (n=1).

$$y = 10.223x + 212.43$$
$$R^2 = 0.7942$$
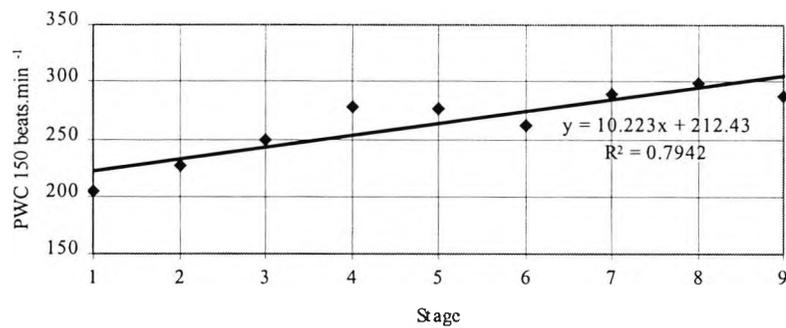
10.4.1.3 Energy demands of stage race cycling; P&O Tour of the North (TOTN)

Three riders completed a five-day (6 stage) Tour of Northern Ireland, however one stage was not fully recorded by one subject. All subjects rode for the same racing team. Subject data are in table 10.5.

Table 10.5 Subject details from the TOTN

Subject	Age (years)	Height (m)	Mass (kg)
A	23	1.83	74.0
B	27	1.77	70.0
C	31	1.78	67.0

The race was made up of 4 one-day stages and one split stage (am & pm), incorporating four mass start road race stages and two short time trials. Stage details are in table 10.6. Overall finishing position and mean data from the race are recorded in table 10.7. Heart rate response was not recorded on one of the riders.

Heart rate responses of subjects A and B are shown in figures 10.10 - 10.14. Although riding for the same team, the team role for these riders was different. Rider A was classed as a domestic, Rider B as joint leader with another member of the team (not included in this report). Roles of the riders did not become apparent until after stage 4 where rider B made the decisive split of the race, rider A did not.

Table 10.6 stage details from the TOTN

Stage	Discipline	Distance
1	Time trial	5 miles
2	Road race	80 miles
3**	Time trial	6 miles
4**	Road race	55 miles
5	Road race	75 miles
6	Road race	75 miles

Table 10.7 Race data from the TOTN

Subject		A			B			C	
	W	fC	rpm	W	fC	rpm	W	fC	rpm
stage 1	349	174	84	353	182	82	373		95
stage 2	282	158	66	239	167	73	287		79
stage 3**	281	166	72	325	180	84	353		93
stage 4**	228	147	75	264	171	75	241		73
stage 5	230	144	60	280	165	79	240		72
stage 6	237	147	68	261	160	73	256		75
GC finish		35			7			25	

** denotes split stages

Figure 10.10 Heart rate response during time trial and road race stages of the TOTN.
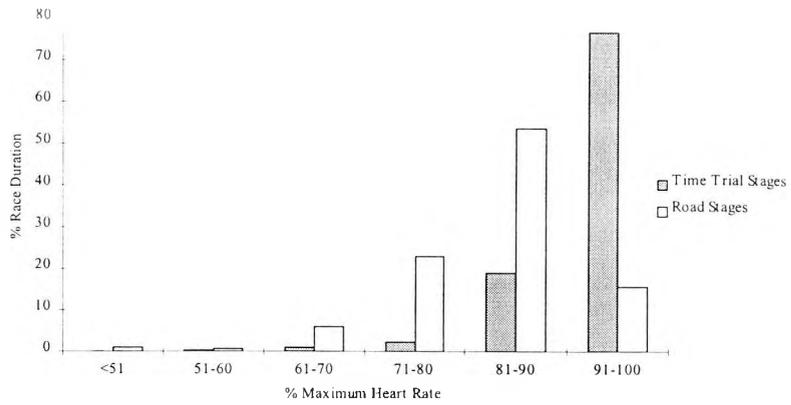
Figure 10.11 Heart rate response during stage 1 of the TOTN
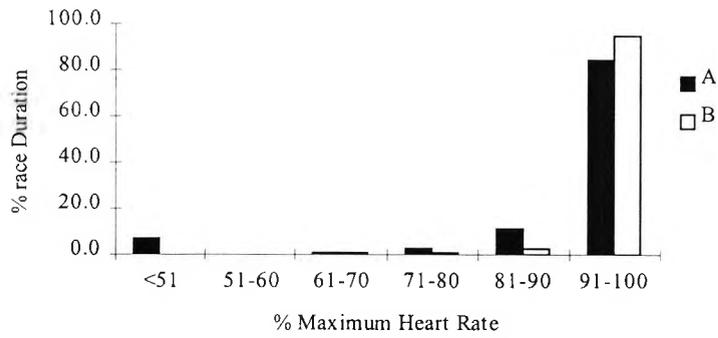
Figure 10.12 Heart rate response during stage 2 of the TOTN
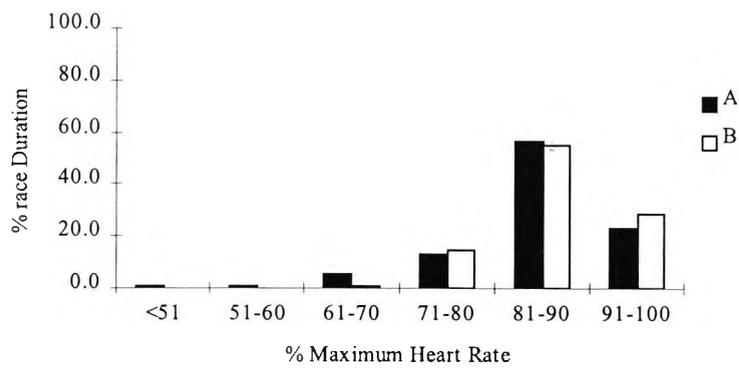
Figure 10.13 Heart rate response during stage 3 of the TOTN
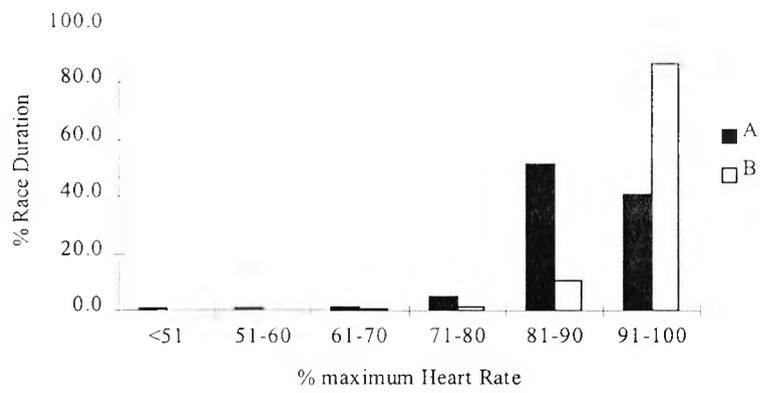
Figure 10.14 Heart rate response during stage 4 of the TOTN
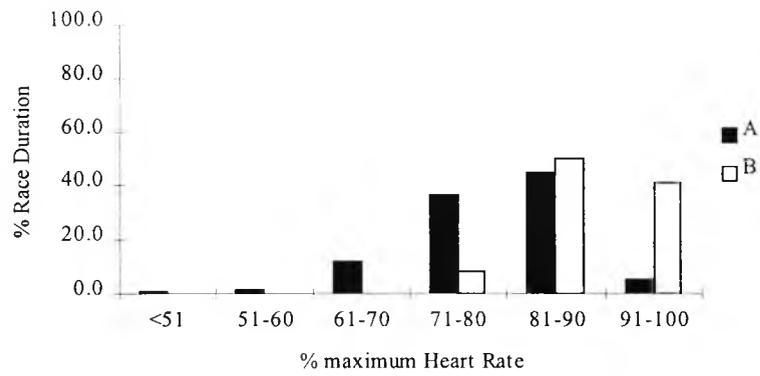
Figure 10.15 Heart rate response during stage 5 of the TOTN
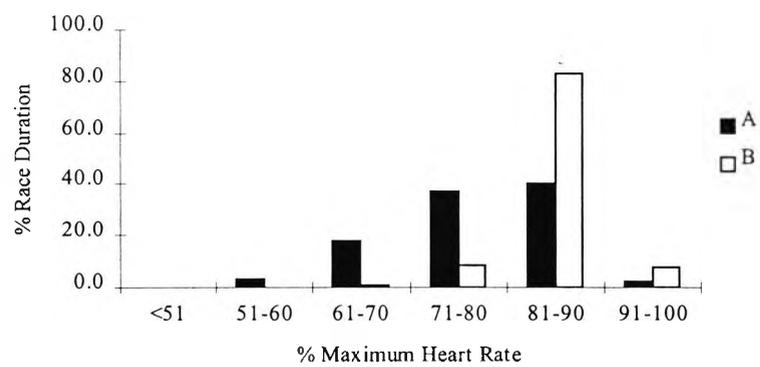
Figure 10.16 Heart rate response during stage 6 of the TOTN
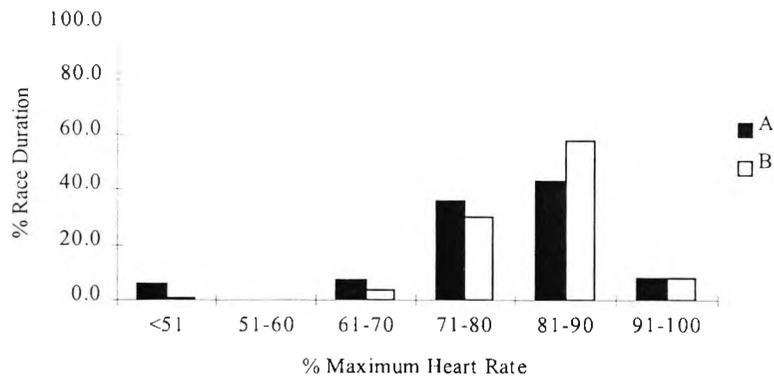
10.5 Discussion

10.5.1 Power output during road cycling

The data collected from the competitive road cycling events is the first data presented with the resolution of 1 Hz. Recent studies have reported SRM data, however not with this resolution (e.g. Jeukendrup *et al.*, 1998), and the data has been a 'case study' of an individual ride, compared to this sample of races. SRM data has also been published in the popular cycling press from a ride in a European 'Classic' world cup race with the rider being former Tour de France winner Bjarne Riis.

Appendix 2 shows the SRM race traces for Power, pedal cadence, and depending upon rider choice, heart rate and speed. From the data, it is apparent that the power output is highly variable, and intermittent in nature. The traces reveal that the road race effort is made up of many repeated bursts, interspersed with recovery periods of inactivity (in terms of power output). For those riders that opted to wear a heart rate transmitter, it is also apparent that the power output is far more variable than heart rate during competition.

Previous studies by Brouns *et al.* (1991) and Palmer *et al.* (1997) have tried to simulate road race cycling at relative exercise intensities fluctuating between 30 and 90% of maximal power output derived from a $\dot{V}O_2$max test. The data collected during this study indicates >20% of data points falling below 30% of MMP, and >39% of data points above 90% of MMP. Palmer *et al*, (1997) constructed a simulation from heart rate traces from races. The data from this study suggests that the heart rate is not as variable as the power output trace, and the heart rate recordings do not represent the rapid alterations that are seen in the power output trace. With data points in excess of 100% of MMP not reflected in the heart rate response (as heart rate doesn't exceed 100% of the maximum value recorded during a MMP test) this indicates how the Palmer *et al.* (1997) protocol which was derived from heart rate was limited to ~85% MMP. Likewise, the heart rate never drops to zero when the power output of the subject drops to zero. Therefore in the Palmer *et al.* (1997) study there was no recovery periods during the protocol. With the use of heart rate (with power

90

or $\dot{V}O_2$) it is important therefore that the baseline data is recorded at the start of the test. Baseline heart rate values may have been recorded during field data collection but failure to associate this with a zero power output may have resulted in some inaccuracies in the prediction of power output from the heart rate response recorded in the field. Because of the lack of recovery periods (zero watts), the laboratory simulation of Palmer et al. (1997) should be described as a 'variable intensity' protocol when in reality road race cycling is intermittent.

The mean distribution of power output during the races follows a relatively normal distribution, except for a significant portion of the data at zero watts (12.7%). The data at zero watts significantly influences the mean data from the races, and as mentioned previously, is substantially different from any study that previously has tried to simulate this type of exercise. The mean power output during the measured races was 269 watts, which equated to ~67% of maximum minute power output and ~89% of maximum heart rate. This mean heart rate value is higher than in the Palmer et al. (1996) study who used the heart rate data collected from a South African 4 day stage race the Giro del Capo to develop their road race simulation study. The stage race heart rate responses recorded during this study indicate that the use of heart rate data recorded in the later stages of the race may not be representative of a 'fresh' heart rate response recorded during one day races. Data collected from the FBD milk Ras (the PWC 150 data) shows that heart rate relative to power output falls during stage race cycling. This is further complicated by the Tour of the North data. The heart rate and power data shows a more varied response from day to day. For example rider A (who was used for Domestique purposes by rider B) on stage 2 averaged 282 watts, stage 3, 281 watts, for a difference of 8 beats min[-1] in heart rate response. Likewise the same rider averaged the same heart rate on stages 4 and five with a 9 watts difference in power. Rider B shows a similar response to the FBD rider, chasing a position on the overall classification (like the FBD rider) resulted in a drop in heart rate relative to the power.

The heart rate power relationship is important as it has previously been used to quantify work done during road cycling, by using the heart rate data and extrapolating to laboratory work output. Simultaneous recording of power output has highlighted that this method is clearly flawed. Further research could analyse these responses in relation to tactics and successive days of cycling with a demand in excess of 3000kj.

10.5.2 Anaerobic power during road cycling

The data sampled at different time intervals showed similar patterns for data at 1 second through 5, 10, 30, 60, 90, 120 seconds and for the mean power during each 10% portion of the race. Although non significant, there was an overall trend for a drift down in the peak powers for each of these sampling intervals with the lowest at the 90% portion of the race and an increase back up in the last 10% of the race duration. Peak power of these athletes recorded in the field was in excess of 1150 watts, which is higher than has previously been reported during laboratory testing (Tanaka et al., 1993), and significantly different from laboratory Wingate testing carried out on five of the subjects in this study. This could indicate differences

in our subject selection compared to the 2nd and 3rd category (USA) riders of Tanaka *et al.* (1993). This data could also possibly imply that the nature of anaerobic testing via a Wingate type protocol may not enable trained cyclists to elicit the kind of power outputs that they can achieve in the field.

Appendix 2 shows the sustained high power outputs (>100% MMP) that were recorded when climbing during this study. For sustained power output illustrated by the smoothed data, the peaks during each race where profiles were included came on the climbs during those races. During climbing in competition subjects were able to stand up on their bicycles, therefore the differences could be due to the body posture and force application, during a Wingate type test subjects are required to remain in the seated position, whereas riders stand up during competition. If this is a factor in the generation of power output during anaerobic exercise, it may also have implications for the measurement and evaluation of aerobic capacity (MMP) measured in the laboratory setting. If peak powers achieved during climbing were substantially higher (~300 watts) than previously reported during laboratory tests, then standard stationary (essentially level graded) ergometry testing may not be representative of an essential component of road cycling. This therefore was the rationale behind measuring aerobic capacity, anaerobic capacity and simulated hill climb performance in the hill climb study (chapter 11).

10.5.3 Power output and heart rate response during road cycling

Previous studies have used the heart rate responses during laboratory ramp type tests to calculate energy demands of road race cyclists (e.g. Palmer *et al.* 1996). The simultaneous recording of power output and heart rate by the SRM system allowed for analysis of the relationship between these two variables in the field. The data indicates that heart rate at a given power is significantly elevated relative to that of the same subject in the laboratory, for a power output of 272 watts, the heart rate response was 158 beats·min^{-1} in the laboratory compared to 169 beats·min^{-1} during competition. Essen *et al.* (1977) reported an 11 beats·min^{-1} increase in heart rate during the initial stages of an intermittent trial compared to a steady state condition. The laboratory heart rate:$\dot{V}O_2$ response therefore overestimates the power demands of an event, and the use of field heart rates to compare to laboratory responses (e.g. blood lactate threshold responses) may have limitations. Differences between laboratory predicted and actual race heart rate were observed after 20% of the race duration was completed (figure 10.7), with heart rate differences at 70% of race duration failing to reach significance (p=0.06). Steady state cycling during the study described in chapter 8 revealed a heart rate (at 70% MMP) of 84% of the maximum value recorded during the MMP test. That compares to the ~67% of MMP recorded in the field at a heart rate apparently 'inflated' to 89.5% of maximum heart rate.

Using the same method of establishing a heart rate power relationship in the laboratory, and comparing it to simulated performance in the laboratory, and race performance in the field, data on time trial (~steady state) cycling shows a different response. The relationship did not show significantly higher heart rates in the field or indoor performance relative to the laboratory ramp test relationship (p=0.12) for a 12-subject sample (Balmer unpublished observation).

Kenny *et al.* (1995) and Vanhelder *et al.* (1985) reported that the linearity of the power : heart rate relationship is influenced by intermittent exercise. Two possible explanations are the adrenaline response of competition not being matched in the laboratory setting, or secondly the continual change in intensity elevated the heart rate response during road cycling. Work rates of varied intensity could cause changes in the response of metabolites in the blood and the blood pressure. If increased work periods drove fluid out of the vascular volume, then the increased heart rate is a possible the result. Wilkerson *et al.* (1977) reported that greater losses of plasma volume were seen with the appearance of BLac⁻, possibly reducing plasma volume, resulting in an increased cardiac frequency for a given volume. If this is a possible influence on heart rate then the amount of sprinting and repetitive intermittent activity that is likely to elevate B[Lac⁻], may cause greater changes in the power:heart rate ratio.

A further complication may be the posture adopted by the cyclists during competition compared to laboratory tests. During road cycling there are many times when a rider will ride out of the saddle in a standing posture. Swain and Wilcox (1992) reported an increased $\dot{V}O_2$ observed during standing cycling due to 'accessory muscles' being utilised in the rocking motion of the bicycle. These alterations in energetic cost have also been reported by Ryschon and Stray Gundersen (1991). They reported a 12% increase in $\dot{V}O_2max$ and an 8% elevation in heart rate response during standing versus seated cycling. Therefore this is possibly a factor that has influenced the difference between seated laboratory and standing/seated field performance.

This data suggests that there is a change in the energetic cost of cycling during standing cycling. This has implications for the physiological assessment of competitive road race cyclists, if the standing posture is an important component of a race - during sprinting, climbing and breaking away (i.e. when power outputs are extremely high), plus for the comfort factor of altering muscle fibre recruitment for a few seconds, the assessment of road cyclists using a seated posture may not allow for the true reflection of the cyclists' capacity to be recorded in the laboratory.

This also has major implications for the previous studies carried out comparing heart rate data to laboratory responses to determine the physiological demands of sports. Palmer *et al.* (1994) reported the heart rate responses of an elite group of cyclists during the Giro del Capo a 4-day stage race in South Africa, with comparison to laboratory heart rate:power measures to ascertain the demands of the race. The findings of this current study indicate that in all riders the laboratory relationship is likely to overestimate the power output predicted from a field heart rate response. These findings do highlight the limitations however of converting heart rate data from the field into other physiological responses recorded in the laboratory, although the exact cause remains speculative.

10.5.4 Pedal cadence during road cycling

It has been reported that trained cyclists have a preferred pedal cadence of 95.3 rpm (Marsh and Martin, 1995), this appears in contrast to optimal cadences reported in the literature (Jordan *et al.*, 1979 reported

optimal cadence of 60 rpm, Coast *et al.*, 1986 reported optimal cadence between 60 and 80 rpm). Mean cadence during the road races was 74.5 rpm, apparently agreeing with the findings of Coast *et al.* (1986). However if the data at zero watts (i.e. when the pedals are not turning) is removed, the mean pedal cadence for time pedalling is ~85 rpm, thus closer to Marsh and Martins' (1995) findings. Our study however does not attempt to solve this apparent disagreement, and the added explanation by Merrill and White (1994) that race cadences selected are due to peer and coaching pressure complicates things further. Another of their arguments is that due to group dynamics of the peleton cyclists must be able to change pace almost instantly, thus being caught in a high gear would have detrimental effects on the acceleration of an individual. Changing gears and pedal cadence to a preferred level can only happen when there are lower forces acting on the crank, thus the behaviour of other individuals is possibly going to influence any mean figures that are generated from this type of data collection.

10.5.5 Correlation to finishing position during road cycling

Rollings *et al.* (1995) have identified body mass as an important component of cycle racing that can influence results in cycle road races. Stovall *et al.* (1993) reported that during the American stage race 'Tour Dupont', there were significant correlations between body mass and finishing position of the 55 male competitors, with the strongest correlation on the hilliest stage. Likewise Pfeiffer *et al.* (1993) reported significant correlations between finishing time and $\dot{V}O_2$ ml·kg^{-1}·min^{-1} in 11 female elite riders, and this despite the individual tactical nature, and possible team tactics and mechanical problems influencing the results. This study showed no significant correlations between finishing position and power: body mass ratio. Without measuring the body mass of the other competitors in the race it is difficult to assess this relationship with more confidence. However, riders with a higher power to weight ratio failed to win lower class races than those with lower power to weight ratios in races of a better (licence) standard. The subjects were instructed to try to win the races, some riders opted to break away early (e.g. one rider was away in a break for 85 miles and was caught ending up with the worst position (53rd) yet has a power to weight ratio of over 6.1 watts.kg^{-1}), whereas others left their efforts until a sprint finish. Likewise the terrain was not necessarily hilly, and races were on different courses. The correlation between finishing position and laboratory measures is also going to be influenced by riders that recorded more than one race with different finishing positions despite the same laboratory measures. Rollings *et al.* (1985) concluded that increases in $\dot{V}O_2$max and optimising body mass would improve road performance in trained cyclists. By reducing body mass, the energetic cost of riding the course would reduce physiological responses, thus possibly reducing acidosis, and the reliance on muscle glycogen, both of which could influence performance in the latter stages of a race. The findings from this study cannot support these findings, as the nature of the sport is not necessarily just a test of physiological capacity. In terms of gaining a high finishing position, this study highlights the importance of tactical awareness as a major component of this sport, and possibly as important as the variables (MMP, MMP.kg^{-1}) measured in the laboratory during one day cycle races on relatively flat terrain (compared to 6000m of climbing in the toughest stages of the Tour de France).

Chapter Eleven. Correlates of Cycling Hill Climb Ability

11.1 Introduction

The rationale for this study comes from the findings in chapter 10. Road race data (appendix 2) when smoothed over show high power outputs on the climbs during a race. The power outputs sustained were substantially higher than MMP in many cases indicating that the climbs may place significant physiological demands upon the competitive road cyclists. Chapter 8 showed significant physiological stress at 85% of MMP, (94% maximum heart rate, B[Lac⁻] 11.28 mmol.l⁻¹). Therefore an aim of this study was to evaluate the power, heart rate and BLac⁻ response to simulated hill climbing.

Due to the high power demands during climbing, it appears that the hills during any road race are potentially critical to the outcome of a race. Observations from the field show that riders during climbing often switch position from seated to standing riding, to maintain speed on the hills. During laboratory evaluation, the rider is forced to adopt a seated position due to the calibration procedures of the Kingcycle (8.2.2.1.2). Thus a further the aim of this study was to establish if standard seated ergometry testing (Kingcycle) could predict competitive cyclists' performance on a gradient (motorised treadmill) with freedom to change their posture during treadmill trials.

Due to differences in hill climb length during different races, two different simulated climbs were used for this study (1km at a 12% gradient and 6km at a 6% gradient). Anecdotal evidence from professional cycling suggests that during the World cup (1 day races) that have many short climbs (0.4-2 km), true climbers (riders with a high aerobic power to weight ratio) do not achieve the results and generally larger riders win these races. Whereas during the longer climbs (5-20 km) during the national tours of Italy, France and Spain, those riders with a high aerobic power to weight ratio do exceptionally well. Therefore another aim of this study was to see if there was any difference in the demands of the two different simulated climbs.

11.2 Method

Eight trained cyclists were recruited (8.1) to participate in this study. Laboratory and subject details were recorded (8.2.1), along with standardising pressure in the subjects' bicycle tyres. The mass of the subject in full kit plus the mass of the bicycle were also recorded during preliminary measures. Subjects were familiarised to the testing procedures (11.2.1) and were required to visit the laboratory on six further occasions (one per week) for a series of tests. The testing protocol consisted of a Kingcycle MMP test (8.2.2.1.3), a Kingcycle power 'Wingate' test (10.3.1.1) and four trials riding on a large motorised treadmill graded at 6% and 12% to simulate hill climbing. All tests were conducted in a random order. Prior to all laboratory visits, subjects refrained from training the previous day, and maintained their normal dietary practices.

11.2.1 Familiarisation

Seven of the subjects had previously undergone physiological testing in the Sport Science laboratory, however, all subjects were (re) familiarised with the Kingcycle (MMP and sprint protocol), and the thumb prick blood sampling technique to be used in this study. Subjects were also given a minimum of 2 hours familiarisation to treadmill cycling, with practice runs at the gradients/distances that were to be used during this study.

11.2.2 Experimental procedures

All tests were carried out in random order, subjects were only informed of which test they were completing 30 minutes prior to that test.

11.2.2.1 Test 1. Kingcycle MMP .

A Kingcycle MMP test was completed by all riders as described previously (8.2.2.1.3).

11.2.2.2 Test 2. Kingcycle Power Test.

Subjects underwent a Kingcycle power test as described in 10.3.1.1.

11.2.2.3 Tests 3 and 4. 6 km (6%) Treadmill Performance Tests

Subjects performed two 6 km treadmill performance tests at a 6% gradient on two separate occasions. Upon arrival, laboratory details were recorded and SRM Power cranks were attached to the subjects' bicycles and calibrated. Subject details were recorded and bike plus body mass value was compared to the subjects' previous data. During initial weighing of the subject's bicycle a 750g water bottle was added. If the subject had an increase in body mass water was removed from the bottle. Any loss in body mass was matched by placing weights in the subject's racing jersey pockets. Subjects warmed up on the treadmill and indicated to the experimenter when they were ready to start. The experimenter gave instructions to the riders on maintaining an even paced strategy for the fastest ride possible. Oral instructions were given by the subjects to influence starting speed, and speed during the test. These were adjusted by the experimenter accordingly. The starting speed of the second test was calculated as the average speed of the first test. The aim was to complete the test in the shortest time possible. Subjects were allowed to view power, heart rate, cadence, time elapsed, distance covered and speed during the test.

11.2.2.4 Tests 5 and 6, 1km (12%) Treadmill Performance Tests.

Subjects performed two 1 km performance rides at a 12 % gradient on a motorised Woodway treadmill, with the protocol identical to tests 3 and 4 except for the distance and gradient of the tests.

11.2.3 Statistical Analysis

Performance (time) was analysed in relation to the standard laboratory measures recorded from Kingcycle testing. Pearson's product moment correlation analysis was then used to establish the relationship between independent variables. Stepwise multiple regression analysis was also used to construct prediction equations for each of the climbs. The coefficient of variation was also calculated on repeated trials.

Significant differences between the treadmill tests were calculated using a one-way analysis of variance test. The data was analysed using absolute values and allometric scaling was also employed based upon the work of Swain *et al.* (1994).

11.3 Results

11.3.1 Standard stationary ergometer results

Table 11.1 shows the subject details from the MMP test for the 8 subjects.

Table 11.1 Subject details and maximum minute power test results (mean ± S.D.)

Subjects	n=8
Age (years)	27 ± 7
Height (m)	1.77 ± 0.06
Mass (kg)	72.98 ± 5.47
Predicted $\dot{V}O_{2max}$ ($l \cdot min^{-1}$)	4.01 ± 0.3
Predicted $\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	55 ± 4
Maximum Heart Rate (beats·min^{-1})	192 ± 5
Maximum Minute Power (SRM) (W)	357 ± 19.6
Maximum Minute Power·kg^{-1} (W·kg^{-1})	4.89

Results for the eight subjects from the 30-second sprint test are shown in table 11.2.

Table 11.2 Individual responses to 30-second sprint test protocol. Values for peak power and % fatigue are for a 1 second sample.

	Peak Power (watts)	Mean Power (watts)	Time to Peak Power (seconds)	% Fatigue
1	796 (782)	635	12	37.6
2	818 (786)	571	8	45.8
3	918 (901)	643	12	40.5
4	732 (715)	558	9	41.8
5	777 (772)	632	10	54.2
6	794 (722)	641	5	25.2
7	974 (966)	768	9	35.5
8	905 (893)	673	9	49.9
Mean ± S.D.	839 ± 83.2	640.2 ± 64.4	9.25 ± 2.2	41.3 ± 9.0%

11.3.2 Treadmill test results

Using the methods of detecting heteroscedasticity outlined by Neville (2000), there was no evidence of heteroscedasticity within the data.

The times recorded for the 6 kilometre climb at a 6% gradient, and the 1 kilometre climb at a 12% gradient were 16.3 ± 1.08 and 4.19 ± 0.3 minutes respectively. These equated to $92.6 \pm 4.2\%$ and $115.38 \pm 6.1\%$ of maximum minute power. Significantly higher power outputs were sustained during the 1 kilometre climb for absolute ($p<0.001$), and relative power outputs ($p<0.001$). There was also a significant difference between B[Lac⁻] 5 minutes post exercise for the 1 km (11.68 ± 1.9 mmol.l⁻¹) and the 6 km climb (9.65 ± 1.59 mmol.l⁻¹), ($p=0.036$). No significant differences were observed for heart rate (184 ± 6 vs 181 ± 4 beats min⁻¹) between 1 km and 6 km performance ($p=0.204$), which equated to 96% and 94% of maximum heart rate.

11.3.2.1 Performance Trial 1 and Performance Trial 2

There were no significant differences between performance trials 1 and 2, for both the 1 km (4.2 ± 0.17 vs 4.19 ± 0.27 minutes, $p=0.937$) and the 6 km (16.79 ± 0.91 vs 16.31 ± 1.08 minutes, $p=0.348$) trials. The coefficient of variation (time) for the two 6 km trials was 2.03% (95% CI= 1.32-4.4%) $r=0.91$ (95% CI= 0.57-0.98), SEE 2.24 (95% CI= 1.38-4.2). For the two 1 km trials the coefficient of variation was 2.28% (95% CI= 0.99-5.42%), $r=0.68$ (95% CI= -0.81-0.99), SEE 3.47 (95% CI=1.51-8.24). To record the faster times in the second trial higher power outputs were also recorded. For the 29 second improvement in time for the 6 km climb subjects increased power output (321 ± 17 vs 331 ± 17 watts for the first and second trial), but this was also not significantly different between trials ($p=0.286$). Coefficient of variation for power was 2.29% (95% CI - 1.24-4.13%), $r=0.82$ (95% CI= 0.26-0.97) SEE 3.28 (95% CI=1.78-5.93) for the 6 km trials. For the 1-second improvement during the 1 km trials a non significant ($p=0.35$) increase in power output was recorded (408 ± 32 vs 411 ± 24 watts for the first and second trials respectively). Coefficient of variation for power for the 1 km trials was 4.04% (95% CI - 1.6-9.58%), $r= 0.75$ (95% CI= 0.1-0.95), SEE 6.43 (95% CI= 2.8-15.26). The heart rate response followed a similar pattern for the 6 km climb (179 ± 7 vs 181 ± 4 beats min⁻¹ for the first and second trial) ($p=0.48$), and the 1 km climb (182 ± 6 vs 184 ± 6 beats min⁻¹) ($p=0.51$).

11.3.2.2 Performance Prediction

Performance predictions from the maximal minute test and the 30-second sprint test are shown in table 11.3.

The highest correlation coefficient recorded was that of one climb versus the other ($r=0.97$, $p<0.01$). Power output sustained during the 1 km and the 6 km did not yield significant correlations to performance time ($r=-0.157$, and $r=-0.428$ respectively).

Where MMP is maximal minute power output, SPP is the sprint test peak (1 second) power, and SMP is the sprint test mean power (30 seconds).

Stepwise multiple regression yielded the following two equations.

6 km speed $(km.h^{-1})$ = 0.788 SMP kg^{-1} + 1.884 MMP $(kg + bicycle\ mass)$ + 7.295

$(r=0.96, p=0.0016)$.

1 km speed $(km.h^{-1})$ = 0.521 SMP kg^{-1} + 1.201 MMP $(kg + bicycle\ mass)$ + 4.72

$(r=0.98, p=0.0004)$.

Table 11.3 Correlation coefficients for time versus laboratory measures.

	1 km Climb at 12 % grade	6 km Climb at 6% grade
MMP (watts.kg^{-1})	-0.89	-0.87
MMP $kg^{0.67}$	-0.88	-0.89
MMP $kg^{0.79}$	-0.92	-0.87
MMP (kg + bicycle mass)	-0.90	-0.89
SPP (watts kg^{-1})	-0.65	-0.56
SPP $kg^{0.67}$	-0.60	-0.51
SPP $kg^{0.79}$	-0.53	-0.63
SPP (kg + bicycle mass)	-0.64	-0.55
SMP (watts kg^{-1})	-0.89	-0.85
SMP $kg^{0.67}$	-0.89	-0.86
SMP $kg^{0.79}$	-0.87	-0.84

11.4 Discussion

11.4.1 Power output during simulated hill climbing

Power output sustained for the 6 km hill climb study was significantly higher than had previously been reported during cycling for this duration (~16 minutes). Fairbarn *et al.* (1991) reported that well trained cyclists (~ 60 $ml.kg^{-1}min^{-1}$) could only sustain 90% of maximum power output for around six minutes on a standard stationary ergometer. Evidence from Chapter 8 indicated that B[Lac⁻] was in excess of 11 $mmol.l^{-1}$ at 85% of maximum minute power output, at the end of the 6% climb B[Lac⁻] was lower than this at a value of 9.65 $mmol.l^{-1}$, despite a higher percentage of MMP sustained (>92%) and although these subjects were different from the subjects who completed the study in chapter 8, if anything, the subjects who completed this study were a lower standard of athlete ($\dot{V}O_2max$ = 55 vs 68 $ml.kg^{-1}min^{-1}$. For the 16 minute climb at over 90% of MMP the heart rate recorded equated to 94% of maximum heart rate, an identical figure to that recorded at 85% of MMP in the results from chapter 9.

From the results from this study, and from the results from the study described in chapter 10, it appears that riders are able to sustain very high powers when cycling on a gradient. Possible explanations may be an increased upper body component of climbing, or it may be that allowing the rider to change position from seated to standing allows for a higher power output to be achieved. In an investigation into the demands of standing and seated cycling, Swain and Wilcox (1992) introduced the topic and speculated that the shift to standing on the pedals allows a rider to bring his mass over the pedals enhancing power production, and may also relieve muscle fatigue by changes in recruitment patterns. This could help to explain the increased power sustained by the cyclists in this current study for an apparent lower physiological cost. A reduced physiological cost during standing cycling however was not supported by their results. Swain and Wilcox, (1992) reported no significant difference between standing and seated conditions, and Ryschon and Stray-Gundersen (1991) reported an increase in the $\dot{V}O_2$ cost of standing cycling. Ryschon and Stray-Gundersen (1991) suggest that the biomechanical advantage of standing cycling is overshadowed by the cost of the upper body exercise. These authors indicated that standing (compared to seated cycling) involves a greater musculature, with the upper torso being more involved for guiding of the rocking motion of the bicycle. They also suggest that standing cycling requires greater support, and the result of simply increased grip on the handlebars would elevate $\dot{V}O_2$. There is also indirect evidence that standing cycling also requires increased power from the *quadriceps femoris* to support the body mass (Tanaka *et al*. 1987). Tanaka *et al*. (1987) reported elevated EMG activity during standing cycling compared to seated cycling. As there is always a degree of flexion at the knee joint during standing cycling, the *quadriceps femoris* must be recruited to not only to maintain power output, but to also support the body weight of the rider.

Ryschon and Stray-Gundersen (1991) concluded that the reason why cyclists adopt the standing position during climbing is to distribute the workload over a greater muscle mass, which despite the elevated $\dot{V}O_2$ cost, is perceived by the cyclist to reduce the intensity.

Chapter 11 investigates the cardio respiratory responses of riding on graded and level cycling in the seated position at a lower exercise intensity.

A positive correlation was recorded between performance and MMP.kg^{-1}, which supports the findings by Rollings *et al*. (1985) who concluded that increases in $\dot{V}O_2$max and optimising body mass would improve performance in trained cyclists. Likewise the weak to moderate correlations of absolute power sustained during these hill climb trials again highlight the importance of body mass during simulated climbing. These physiological adaptations would allow for faster predicted simulated hill climb performance, however, again tactics during road cycling can possibly mask this ability, even during hillier races, if a rider does not go with a break, even if they have the highest $\dot{V}O_2$max relative to body mass (ml.kg^{-1}min^{-1}) it would not necessarily be a winning attribute.

11.4.2 Physiological responses to simulated hill climb performance

Despite differences in the physiological responses during level and graded cycling, during the hill climb study the prediction of performance was still possible from the level stationary ergometer. Relative to body mass the power outputs recorded during the 'Wingate' type tests correlated significantly to performance results during the hill climb tests. The B[Lac⁻] suggests a significant contribution of the anaerobic system during both of the hill climb tests. However, with mean exercise intensity lasting over 16 minutes during the 6 km climb, it is surprising that mean (30 second) power and even peak (1 second), power correlate to performance time. Previous authors have indicated however that a significant portion of energy is being derived via the aerobic system during exercise of this short duration. These findings support the work of Withers *et al.*, (1991) who found strong correlations between markers of aerobic metabolism ($\dot{V}O_2$max) and sprint performance during laboratory testing in trained road cyclists. Therefore aerobic capacity is possibly influencing both test results. Using an off line system of gas analysis (Douglas bags) Keen (unpublished observation) has presented data indicating $\dot{V}O_2$max can be achieved rapidly during a 90 second sprint effort. This therefore possibly explains the findings during the hill climb study. Previous data on well-trained cyclists (Sjogaard, 1984) has shown significantly high proportions of slow twitch muscle fibre present, with some fast twitch (IIa), and minimal IIb fibres present. During the road race cycling it appears that the ability to continually fluctuate between high and low powers for over four hours is the general pattern of demands in this sport. Even though previous simulation studies have not really recognised the high power demands, authors such as Burke *et al.* (1977) have recognised the importance of sprinting ability. However, as the duration of the cycle road races is generally 45 minutes plus, the endurance component and relatively high force production may possibly be a result of slow twitch muscle fibre activation. Despite what one might expect to perceive as a sport specific test, athletes trained at other disciplines achieve higher peak powers than the competitive cyclists during the Kingcycle anaerobic test (team game player peak power (1 second) = 1770 watts, 26 watts.kg⁻¹, unpublished observation).

This study also further indicates that the heart rate response during exercise has limitations when assuming it is associated with a specific power value. There was no significant difference between the heart rates recorded during the 6 km climb and the 1 km climb, yet the power differences were 92.6 and 115.4% of MMP for the two climbs which equates to 330 vs. 412 watts for no significant difference in heart rate. There was a slightly higher heart rate but only 1.6% (p>0.05), for the production of 20% more power. The work of Palmer *et al.* (1994) matching heart rate to a power (or $\dot{V}O_2$) value therefore has its limitations.

Chapter Twelve. The Effect of Gradient on Cardio Respiratory Measures During Submaximal Cycling.

12.1 Introduction

The results from chapter 11 indicate that well trained cyclists can sustain higher percentages of MMP during graded cycling compared to previous research on level cycling. When achieving this higher sustained work rate, a lower mean B[Lac⁻] was observed compared to a previous group of subjects riding at a lower percentage of MMP. This could be due to a decrease in Lac⁻ production or an increased Lac⁻ removal, either of which would possibly increase the $\dot{V}O_2$ for a given work rate. However, these findings could simply be a function of an increased efficiency - mechanical advantage, allowing more work for less effort during graded as opposed to level cycling. The aim of this study was to evaluate the cardio respiratory demands of level and graded cycling on the same subjects, and compare these responses to a short hill climb performance test.

12.2 Method

Six well-trained male cyclists were recruited to participate in this study (8.1). Initial details were recorded (8.2), and in common with the methodology in the previous study, tyre pressure was standardised (100 psi), plus the mass of the full kit (bike plus rider) was also recorded. Likewise a 2-hour familiarisation to treadmill riding, and refamiliarisation to Kingcycle procedures was undertaken (11.2.1). All subjects had previously undergone physiological assessment in the Sport Science laboratory.

Following familiarisation, subjects were required to make four further visits to the laboratory. Test 1 consisted of a Kingcycle MMP test, tests 2 and 3 involved riding on a motorised treadmill at either a gradient, or on the level. Test 4 consisted of a treadmill performance test (2 km at a 6% gradient). Tests 2, 3 and 4 were carried out in random order. During treadmill familiarisation, this simulated climb was attempted on at least two occasions to ensure that subjects knew the demands of the performance test.

Prior to all tests subjects refrained from training the previous day, and were instructed to maintain their normal dietary practices.

12.2.1 Experimental procedures

12.2.1.1 Test 1

Test 1 consisted of a Kingcycle MMP test as described previously (8.2.2.1.3). Data was also simultaneously collected using the SRM powercrank system previously attached to the subject's bicycle.

12.2.1.2 Tests 2 and 3

Tests 2 and 3 consisted of 6-minute trials riding on the motorised treadmill. Following a thorough warm up, subjects' indicated when they were ready to begin. Subjects' were then fitted with a facemask (Hans Rudolf. Kansas city, USA), and connected to an online gas analysis system (Covox. Exeter, UK). A further 2 minute warm up was then followed by the adjustment of speed to elicit 60% of the individual's MMP. Test 2 consisted of subjects' riding at 60% of MMP on a level gradient. Test 3 only differed from test 2 in

that the 60% MMP was elicited on a 6% gradient. Throughout the 6-minute trials $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, and heart rate were recorded. During trials 2 and 3 pedal cadence was matched between trials, and riders remained seated during all trials.

12.2.1.3 Test 4

Test 4 comprised of a treadmill performance test of 2 km in duration at a 6% grade. Following warm up to their own requirements, subjects indicated to the operator when they were ready to begin the testing protocol. The treadmill was elevated up to 6% and on adjustment of the treadmill speed to the fastest average speed recorded during familiarisation, the test commenced. Subjects orally communicated with the experimenter to adjust the speed of the treadmill belt. The subjects were instructed to complete the test in the shortest time possible.

12.2.2 Statistical analysis

Differences in the physiological responses were ascertained using an Analysis of covariance test (covariate treadmill power).

12.3 Results

All six subjects (details in table 12.1) completed the testing protocol. Table 12.2 shows the cardio-respiratory response to level and graded cycling. Due to slight differences in the mean power (3 W) ANCOVA was used to analyse the data.

Significant differences in $\dot{V}O_2$ (0.17 $lmin^{-1}$, p=0.018) (Figure 12.1), $\dot{V}CO_2$ (0.32 $lmin^{-1}$, p=0.032), and $\dot{V}E$ (4.64 $lmin^{-1}$, p=0.035), (figure 12.2) were recorded. No significant differences were found for RER values (p=0.273) (figure 12.3) and heart rate (p=0.461) on the level compared to graded cycling. Gross mechanical efficiency was also similar (p=0.408) between trials.

Table 12.1 Subject details (mean ± S.D.)

Subjects	n=6
Age (years)	26 ± 7
Height (m)	1.785 ± 0.05
Mass (kg)	72.7 ± 6.5
$\dot{V}O_{2max}$ ($lmin^{-1}$)	5.23 ± 0.52
Maximum minute power (watts) (SRM)	413.2 ± 42.2

Table 12.2 Cardio-respiratory response during level and graded treadmill cycling (n=6). (mean ± SD).

Treadmill Gradient	0%	6%
Power Output	249 ± 23.5 watts	252 ± 25.3 watts
$\dot{V}O_2$	3.08 ± 0.39 l min^{-1}	2.91 ± 0.19 l min^{-1}*
$\dot{V}CO_2$	2.94 ± 0.36 l min^{-1}	2.62 ± 0.24 l min^{-1}*
$\dot{V}E$	59.34 ± 8.57 l min^{-1}	54.7 ± 6.55 l min^{-1}*
RER	0.94 ± 0.02	0.90 ± 0.03
Gross Mechanical Efficiency	23.47 ± 2.41 %	24.79 ± 0.76 %
Heart Rate	146 ± 11 beats min^{-1}	143 ± 8 beats min^{-1}

*denotes significantly different from level (0%) treadmill cycling (p<0.05).

Figure 12.1 Oxygen consumption during submaximal exercise at the same power output during level and graded cycling.
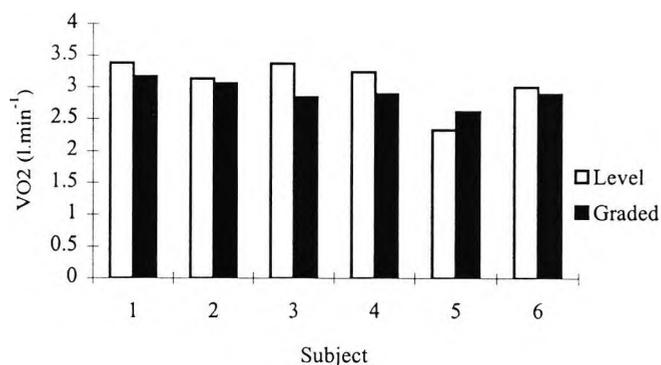
Figure 12.2 Ventilatory response during submaximal exercise at the same power output during level and graded cycling.
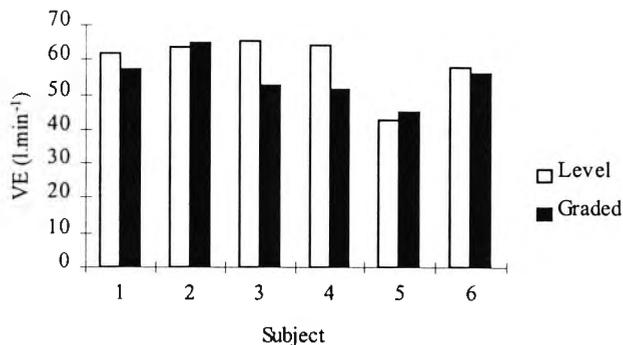
Figure 12.3 Respiratory exchange ratio during submaximal exercise at the same power output during level and graded cycling.
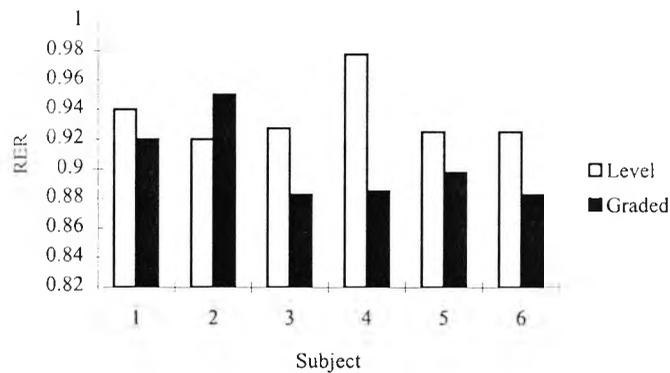
Figure 12.4 Gross mechanical efficiency during submaximal exercise at the same power output during level and graded cycling.
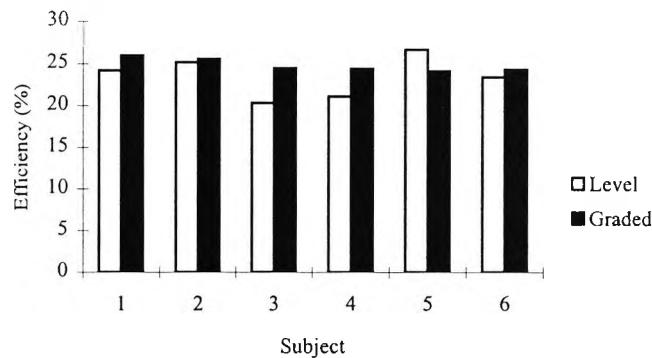
12.3.1 Performance test

The results of the performance test of 2 km at a 6% indicated that riders sustained 113.8 ± 8.91% of maximum power during the test. The time taken to complete the performance test was 4.70 ± 0.33 minutes, which equated to a speed of 25.65 ± 1.8 km.hr^{-1}. Gross mechanical efficiency calculations from the submaximal rides significantly correlated to performance time for the graded trial (r=-0.87, p<0.05), but not for the gross mechanical efficiency calculations from the level trial (r=-0.07 p>0.05). The MMP.kg^{-1}min^{-1} was again a strong predictor of hill climb performance time (r= -0.84).

12.4 Discussion

12.4.1 Cardio respiratory measures

The findings from this study indicate that there are physiological changes between riding on the level as opposed to a 6% gradient. Despite similar power outputs, differences in $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}E$ were

105

observed. This would then appear to support the findings in chapter 11. Trained cyclists could sustain ~93% MMP during a 16+ minute performance test on a gradient during the study described in chapter 11, however previous research on more aerobically trained cyclists revealed that on a stationary ergometer on the level 90% of MMP could only be sustained for ~ 6 minutes (Fairbarn *et al.*, 1991). The chapter 11study also found lower B[Lac⁻] when compared to the highest intensity (85% MMP) completed in chapter 9. Therefore the major finding is that these changes in physiological response are likely contributors to the increase in overall performance seen in chapter 11.

The combination of differences between the two conditions (~8% $\dot{V}E$, ~6% $\dot{V}O_2$, ~11% $\dot{V}CO_2$) indicate lower physiological stress on a gradient compared to on the level. This is the first data of this kind to highlight these differences, however, the exact cause remains speculative.

The reductions in $\dot{V}O_2$ indicate an enhanced economy of movement working on a gradient compared to the level condition. Previously alterations in the $\dot{V}O_2$ response have been attributed to changes in the riders position on the bicycle (Ryschon and Stray-Gundersen, 1991), however the cyclists did not change position between the level and graded conditions. Matching the pedal cadences between the two conditions may provide the answer. Previous research by Balmer *et al.*, (2000) suggests that cyclists opt for higher cadences in the laboratory than in the field. This is believed to be due to differences in the inertial load upon the ergometer, or the resistance in the field. If graded cycling changes the inertial load (compared to level cycling) it may alter the optimal cadence, and in turn the $\dot{V}O_2$ response. The pedal cadence averaged 80.6 ± 0.85 revs min⁻¹ in the graded trial and 81.2 ± 1.1 revs min⁻¹ during the level trial. Previous research has indicated that a cadence of 84 revs min⁻¹ during treadmill cycling at 10% is more efficient than a lower cadence (Swain and Wilcox, 1992), however, during level (ergometer) cycling it does appear that the majority of the literature suggests lower oxygen costs at pedal cadences around 60 rev min⁻¹ (Suzuki *et al.*, 1979, Jordan *et al.*, 1979, Coast *et al.*, 1986). Conflicting findings have been reported by Davison and Flynn (1997), with optimal cadences around 90 rev min⁻¹ however Balmer *et al.*, (2000) reported higher cadences during Kingcycle simulated performance than outdoor performance. This could suggest therefore that the inertial load could be different between graded and level cycling, and also between different ergometry systems. This then may influence the optimal cadence and the overall $\dot{V}O_2$ response during exercise.

Failure to ride at an optimal cadence however does not explain the performance results of the study described in chapter 11. The results indicated that high power outputs could be sustained on a gradient compared to what had previously been sustained during level cycling, and, the lower B[Lac⁻] riding at 92% MMP on a gradient compared to the B[Lac⁻] in the trials at 85% MMP in the study described in chapter 9. The high power outputs recorded during actual races indicate that whilst climbing the cyclist has to sustain very high powers, therefore these results could possibly be attributed to the training status of recruited

muscle fibres during climbing. Further study investigating this phenomenon could compare non cyclists to trained cyclists to isolate this variable.

The limitation of using heart rate like the previous study is again shown here. The extrapolation of a heart rate to a specific $\dot{V}O_2$ value appears to be affected by the gradient of the treadmill. Thus road data with changes in gradient are going to affect the heart rate, $\dot{V}O_2$ and the power response (chapter 11) all of which were variables not considered by the Palmer *et al.* (1994) study.

12.4.2 Performance test

The performance test data from this study corroborates the findings in the previous two studies of well-trained cyclists being capable of maintaining high percentages of MMP during such performance tests. During this study, the performance test time was ~31 seconds slower than the 1 km climb in chapter 11 with ~2% reduction in relative intensity (% MMP) that could be sustained.

Gross mechanical efficiency calculations of the gradient appear also to predict the performance during the 2 km climb, whereas the level efficiency calculation did not. This is possibly due to the effectiveness of specific muscles being recruited during climbing, thus the more efficient the cyclist at submaximal intensities, the better the performance outcomes.

Chapter Thirteen. Laboratory Simulation of Road Race Cycling

13.1 Introduction

Previous research on competitive road cyclists has either used maximal measures of aerobic capacity, or steady state 'performance' tests to evaluate riders' capacities in the laboratory. The data from chapter 10 highlights that road race cycling is very intermittent, and this type of testing may not give us a true reflection of the road race cyclists' capacity. The findings discussed in chapter 9 also showed that no parameters from the Kingcycle MMP test correlated to performance outcome during road cycling. It would appear that either this marker of physical capacity does not play a role in the endurance road race performance (which it does in time trial performance), or the influence of tactics and the use of drafting skew the importance of the physiological responses in the laboratory.

One of the aims of this study is to remove the tactical nature of road racing (by ensuring all subjects complete the same relative amount of work) prior to a performance test. This would therefore give some indication of whether tests of maximal aerobic capacity are relevant for this type of athlete who is regularly utilising anaerobic power and anaerobic capacity to compete.

Previous attempts to simulate road race cycling in the laboratory have used 'variable intensity' (intensity that changes from lower to higher work loads) as opposed to 'intermittent' protocols (where the intensity is variable, but there are also intermittent rest periods) (Palmer *et al.*, 1997, Brouns *et al.*, 1991). These studies varied intensity from 30% maximum power output to 90% maximum power output during a performance ride to exhaustion. From the data in chapter 9, competitive races shows a rather different pattern, with ~13% of race duration at zero watts, and ~30% of race duration above 100% MMP. Therefore another aim of this study was to examine the physiological responses to a more intermittent protocol than has previously been conducted.

Palmer *et al.* (1997), concluded from their study on variable intensity cycling that performance reductions following the variable intensity trial (compared to an even power trial) were due to increased muscle glycogen use during the variable intensity trial. Previous research has shown that with glucose feeding, muscle glycogen can be spared during variable intensity exercise (Yaspelkis *et al.*, 1993). Therefore, if muscle glycogen is a limiting factor influencing performance trials post variable intensity exercise, glucose feeding may enhance performance. It could be speculated that glucose supplementation during variable intensity exercise may therefore have greater effects than when compared to a placebo trial, and be able to supply substrate for this type of exercise. In theory therefore, glucose feeding during an intermittent trial, versus a placebo intermittent trial might have greater effects than when compared to glucose and placebo steady state trials. Therefore a further aim of this study was to test this hypothesis.

13.2 Preliminary study

13.2.1 Introduction

The problems of sport specificity when testing the competitive cyclist have been discussed in chapter 7. The discussion centred on the true replication of road conditions within the laboratory. The actual race data from chapter 10 was used to create the road race simulation protocol (discussed later). During races subjects were able to ride in and out of the saddle, move around on the bicycle, and race with as little intervention as possible. To use this data to create a simulation, and to keep the protocol as specific to the competitive situation as possible, the subject must be able to shift their position, move on the bicycle and opt for an out of the saddle position at their own discretion. The design of the Kingcycle test rig does not allow for the shifting of the cyclist's position, due to the requirement of keeping the mass on the roller constant for a valid reading of power output.

The results from chapter 11 and 12 also reveal possible changes in physiological responses, and performance (power output) during graded as opposed to level ergometry testing. Therefore, the motorised treadmill was selected as the 'ergometer' to be used during this study in an attempt to replicate field conditions.

13.2.2 Treadmill data

13.2.2.1 Introduction

The aim of this preliminary study was to investigate the relationship between power output, body mass, treadmill speed and treadmill gradient. Previous research has used motorised treadmills as a means of eliciting relative exercise intensities (e.g. % $\dot{V}O_2$max), to date however, no research has been published regarding power output whilst cycling on a gradient. Previously work by Jakeman (1989) utilised a pulley system, connected to a basket of weights to increase the exercise intensity during treadmill cycling. This method of increasing resistance was used during this study to uniformly increase or decrease the power output that needed to be attained by the subjects. The pulley type system allows for the instant loading, as opposed to subjects winding up to a given power, which might be a source of variation in total work achieved at the end of the trials. Therefore, another variable the weight basket, and the addition of weights, with treadmill gradient, speed, and power output were also investigated in this preliminary study.

13.2.2.2 Methods

Three male experienced cyclists (details table 13.1) undertook a series of treadmill calibration rides, at submaximal/maximal and supramaximal intensities, with the aim of investigating the relationship between power, speed and gradient of treadmill cycling.

Following the recording of laboratory and subject details, subjects were given a minimum of two hours familiarisation to treadmill cycling on their own bicycle before the study commenced.

Prior to data collection, subjects warmed up and indicated to the experimenter when they were ready to begin the testing protocol. Each trial consisted of a 2-minute work stage with either treadmill speed, treadmill gradient, mass of the rider, or weight on the pulley system being manipulated.

13.2.2.2.1 Power output and treadmill speed/gradient.

Subjects were required to ride for repeated two-minute work stages at treadmill gradients from 0% to 10% in 1% increments, at two speeds, 13.6 and 27.2 kph.

13.2.2.2.2 Power output and mass during graded cycling.

Subjects were required to ride for repeated two-minute work stages on treadmill gradients from 0% to 5% in 1% increments at three different speeds 12.8, 19.2 and 25.6 kph. Two random trials were completed. One trial was conducted with the subject wearing normal racing clothing. The second trial was conducted with the subject carrying 7.8 kg in their racing jersey pockets. Both trials were conducted during the same visit to the laboratory.

13.2.2.2.3 Power output and the effects of increasing weights on the pulley basket during treadmill cycling.

Subjects were required to ride at repeated treadmill speeds (12-36kph) for two-minute work stages. Repeated trials consisted of riding with and without the addition of 1 kg to the pulley system.

13.2.2.3 Results

Table 13.1 shows the details (n=3) of the participants in this study.

Table 13.1 Subject details. (mean ± S.D.).

Subjects	n=3
Age (years)	21 ± 3
Height (m)	1.77 ± 0.08
Mass (kg)	69.2 ± 8.8

Figure 13.1 shows the effects of two different treadmill speeds and varying treadmill gradient on the power output during treadmill cycling. The correlation coefficient revealed a significant linear relationship between these variables (r=0.999, p<0.001) for both treadmill speeds.

Figure 13.2 shows the interaction between body mass (plus cycle mass) and treadmill speed, gradient, and power output. As before, correlation coefficients showed a linear response to increasing speed and/or gradient, (r=0.999, p<0.001) for all conditions. The increase in body mass of 9.1% resulted in an increase in power output of 8.6% across all trials. Data for the effect of body mass at the different gradients is shown in table 13.2.

Figure 13.1 The relationship between power output, speed and gradient during treadmill cycling.
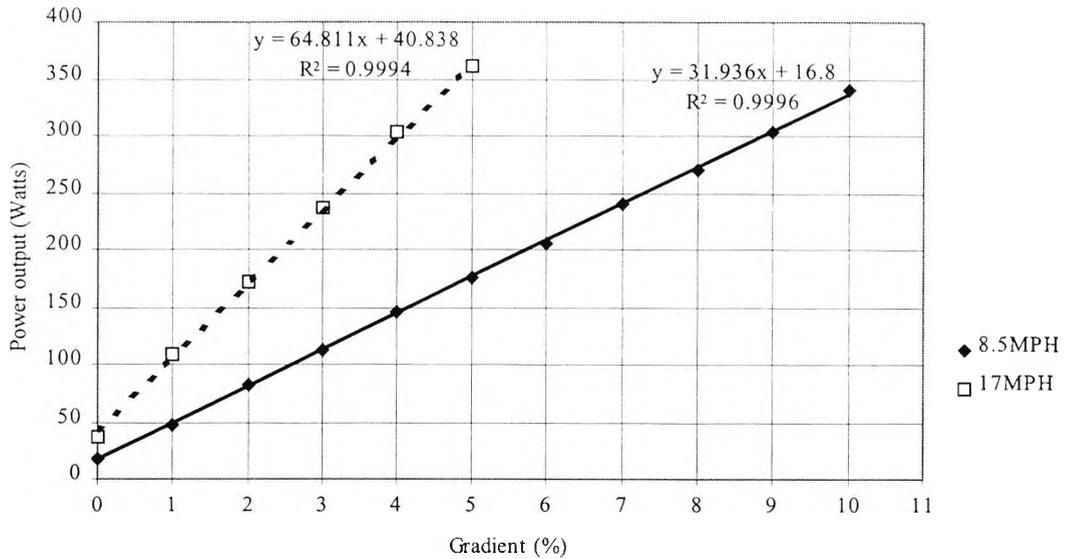
Figure 13.3 shows the effect of adding 1 kg in weight to the pulley system basket on power output during treadmill cycling. The mean data at each speed shows a significant relationship between treadmill speed and the effects of adding 1 kg in weight to the basket (r=0.97, p<0.05). Individual data for each trial (2 minutes) at each speed for each subject also resulted in a significant correlation coefficient (r=0.94, p<0.05).

Table 13.2 The effect of 9.1% increase in body mass on power output during climbing. (mean ± SD)

Gradient (%)	% increase in power output
0	7.5 ± 2.2%
1	9.31 ± 2.98 %
2	7.77 ± 2.16 %
3	10.01 ± 2.72 %
4	8.3 ± 0.73 %
5	8.27 ± 1.03 %

Figure 13.2 The relationship between power output, body mass, speed and gradient during treadmill cycling.
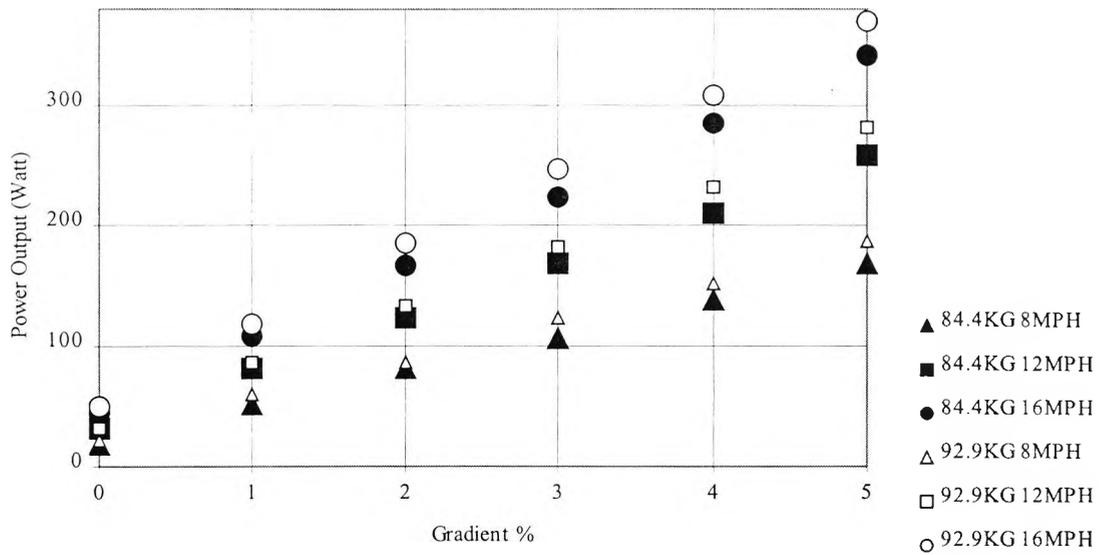
84.4kg, 8 mph. y = 63.743x + 53.476, r² = 0.9995

84.4kg, 12 mph. y = 59.311x + 46.505, r² = 0.9998

84.4kg, 16 mph. y = 49.337x + 34.29, r² = 0.9996

92.9kg, 8 mph. y = 44.837x + 33.524, r² = 0.0.9995

92.9kg, 12 mph. y = 32.2x + 24, r² = 0.9986

92.9kg, 16 mph. y = 29429x + 20.829, r² = 0.9989

Figure 13.3 The effect of adding 1 kilogram to the weights cradle at different treadmill speeds.
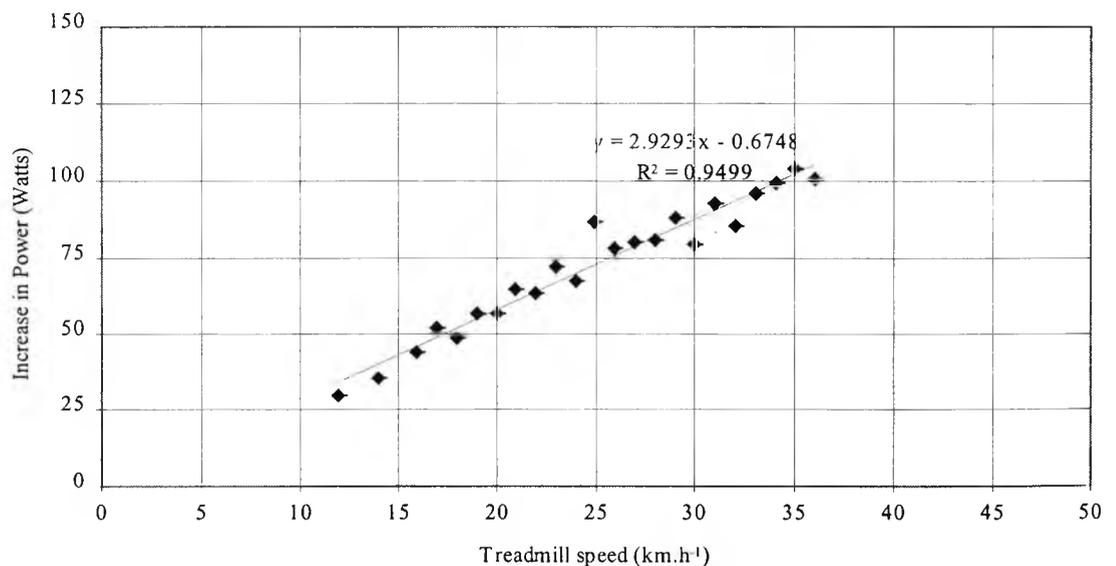
13.2.2.4 Discussion

The aim of this study was to investigate the relationship between treadmill speed, treadmill gradient, body mass, and the effect of using the pulley system on power output during treadmill cycling.

Figure 13.1 shows the effect of increasing the treadmill gradient upon the power demands of treadmill cycling. For two different treadmill speeds, the results showed a significant linear correlation (r=0.999). Any increases in treadmill speed also increase the power output of treadmill cycling (figures 13.1, 13.2), likewise did body mass, as demonstrated by artificially altering body mass (table 13.2, figure 13.2).

These results indicate that the body mass of a rider (and bicycle) will influence the energy demands of riding on a treadmill. In terms of performance outcomes, then minimising both bicycle and body mass will reduce the energetic demand of climbing which appears almost a direct relationship (9.1% increase in mass = 8.6% increase in power output).

A further aim of this study was to view these results with regard to creating a road race simulation on the running treadmill. Body mass appears critical in the formula for creating a protocol of relative exercise intensity. If a subject rides on the treadmill at 25 km.h^{-1}, at gradients from 0% to 5% in 1% increments a relationship between these variables can be established for that rider as an individual. Any changes in treadmill gradient can be accounted for by the slope of a regression line plotted through the data points. Figure 13.1 shows the proportional nature of increased (or decreased) speed, therefore any changes in speed can be accounted for by and initial calibration ride. Finally, figure 13.3 shows the effect of adding weights to the basket/pulley system during treadmill cycling. Again a strong linear correlation was recorded, leading to the use of such a system being quantifiable.

Therefore to conclude, if the subject performed an initial calibration ride, using the pulley system, and manipulating treadmill speed and gradient, calculations of exercise intensity (power output) could be made, therefore the utilisation of the treadmill testing in developing a road race simulation could be possible.

13.3 The development of a protocol to simulate road race cycling

13.3.1 Introduction

The aim of this study was to use the data collected in chapter 10 to create a laboratory simulation of road cycling. The data in chapter 10 revealed distinct differences between the distribution of power output in previous studies, compared to this data. Therefore the aim was to create a simulation that was more 'intermittent' as opposed to variable intensity.

A further difference was the utilisation of the motorised treadmill to create the simulation. Previous studies have utilised standard stationary ergometers to create simulated road racing. However, chapters 11 and 12 have highlighted possible differences between level and graded cycling. Possibly the most distinct difference recorded was the higher work outputs sustained by the subjects on a graded treadmill trial. As the road race data was collected during level and graded cycling, some of the sustained high work rates

recorded could possibly not be achieved on the level on a stationary ergometer, therefore the use of the motorised treadmill for this study was imperative.

13.3.2 Treadmill gradients

In creating a road race simulation, the initial stages involved the development of varying gradients for the protocol. The majority of cycle races in this country take place over a series of 'laps' of a circuit, using this kind of repetitiveness would allow for comparison over time within the physiology laboratory. The profile of a local road race circuit (Godmersham, Canterbury, Kent) was taken, and was scaled to create a 30-minute 'lap' to be repeated four times over. (Figure 13.4 shows the actual profile from maps of the Godmersham circuit).

Figure 13.4 Profile of the four 'laps' of the Godmersham road race circuit
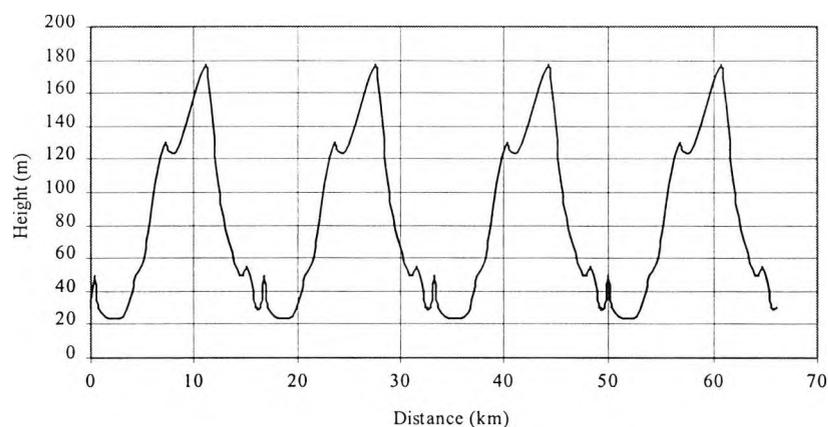
As the motorised treadmill had a maximal speed of 36.6 kph, to simulate road race type pedal cadences (distribution), subjects were limited to their inner chain ring on their bicycle, but could select any rear sprocket gearing at any time.

13.3.3 Power Outputs

The spread of power outputs was created using the data from road races (chapter 10). However, due to each race being very different in terms of timing of attacks, hills, sprints and duration, (mean data is just a smooth line over time), manipulation of typical patterns was attempted.

The aim was to increase the spread of power output compared to previous studies investigating this sport. Therefore high intensity intervals were interspersed with periods of recovery, on the gradients high-sustained power outputs were achieved, similar to the patterns observed during road races.

Due to safety reasons, the 'high' intensity periods were limited to 150% of MMP. The mean overall intensity for the pilot study was 64% MMP plus the performance test, (~65% MMP). However, due to

114

problems with fatigue, and cramp (2 subjects failed to reach the final performance test) this was reduced to 58% MMP plus the performance test (~60% MMP) for the actual study. The aim thereafter was to make the simulation as variable as possible within these limits (0 and 150% MMP).

To simulate descending, and to simulate the amount of time spent at zero power output, 2-minute periods of 'freewheeling' were achieved by having an experimenter hold the bicycle stationary, (blood samples were also drawn at this time). Further periods of zero watts were achieved by having subjects pedal up to the front of the treadmill and stop pedalling and drift back to the back of the treadmill. During periods of light exercise the feeding took place (glucose or placebo), plus the mouthpiece was handed up for gas analysis. For comparison over time, blood samples, gas samples, and feeding were conducted at exactly the same point during each lap.

13.3.4 Performance Test.

At the end of the Godmersham road race course is a 3 km climb to the finish line. To try to simulate the finish, a 2 km climb (6% gradient) was used as the finishing performance test, preceded by a 3 minute period at a fixed work intensity (70% maximum minute power) at a gradient of 6%. This was done to allow subjects to re familiarise themselves with the gradient during this pre performance test period. Subjects were instructed to complete the performance test as quickly as possible, and were allowed to view elapsed distance and time during the test. The sustained gradient of 6% was chosen to match other studies conducted in our laboratory (chapters 11 and 12), and to enable subjects to pace their efforts, which might be more difficult if the gradient constantly changed.

13.3.5 The Protocol

The protocol as mentioned previously was designed to be 'intermittent' as opposed to a variable protocol, the aim was to try to match the histogram pattern of the road race collected data, with that of the simulation protocol (figures 13.5, 13.6).

The protocol had two periods of intense intervals per 'lap' with light recovery periods between efforts. Sustained high work rates were generated during some of the graded sections of the protocol, to try to simulate the similar patterns from the road races.

13.3.6 Sampling

During the protocol continuous measures of heart rate and SRM power output (1 second sampling) were taken throughout the 2-hour simulation. During 'simulated' descending, blood samples were drawn with the mid lap sample analysed for B[Lac⁻] and glucose, and the end of lap sample for B[Lac⁻], glucose, NH_3, and urea. (figure 12.5). Expired gas was analysed for two periods during the simulation, again indicated on figure 13.5, during two periods of fixed intensity exercise (60% MMP).

Figure 13.5 Schematic representation of one 'lap' of the road race simulation protocol (30 minutes)
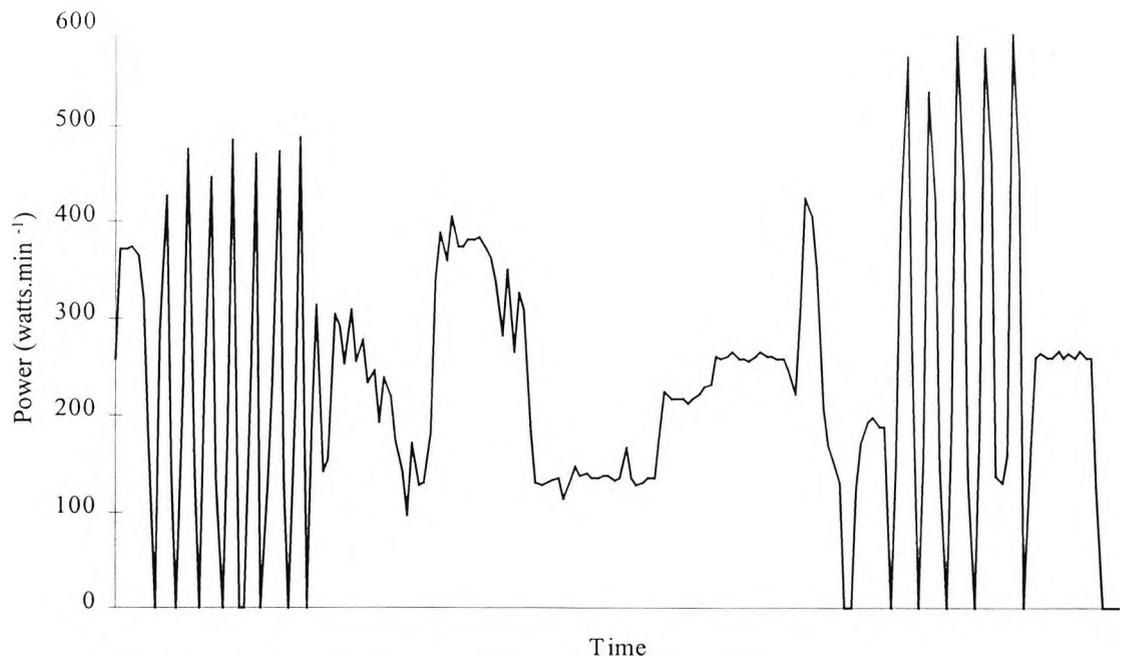
Figure 13.6 Distribution of power output from actual race data and the simulation protocol
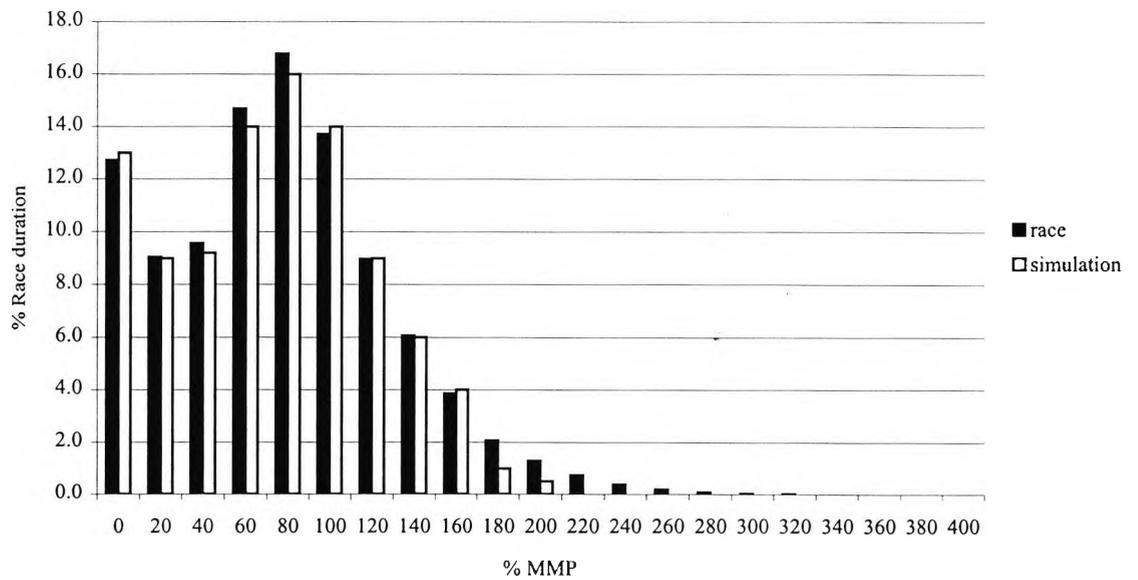
Chapter Fourteen. The Effects of Glucose Polymer Supplementation on Simulated Road Race and Even Power Cycling.

14.1 Introduction

Previous study into the demands of competitive road race cycling concluded that glycogen depletion was possibly the major factor contributing to a deterioration in performance following a variable intensity trial as compared to a steady state trial (Palmer et al., 1997). However, during their study they did not measure any markers of fuel utilisation. The aim of this study was again to compare steady state versus simulated road race cycling. Previous research has shown muscle glycogen sparing through the ingestion of glucose polymer (Yaspelkis *et al.*, 1993). Studies on steady state cycling have not shown muscle glycogen sparing during endurance exercise through glucose feeding, therefore, it is possible that the ingestion of glucose polymer during an intermittent road race simulation may have more of an effect than when compared to even power cycling. The aim of this study was to test this theory.

14.2 Methods

Subjects were recruited as previously described (8.1), and seven male competitive road race cyclists were selected to participate in this study. Subjects were physically active and engaged in training at the time of study.

14.2.1 Pre testing regime

Subjects reported to the laboratory prior to all tests having rested from training the during the previous 24 hour period, otherwise subjects maintained their usual training and dietary routine throughout the testing period. This method of controlling subjects had previously been shown to minimise variation between repeated MMP tests (chapter 9).

Prior to arrival on all laboratory visits subjects were instructed to consume only water during the preceding 2-hour period. Subject details were recorded (8.2.2.1), along with the weight of all of the cycle equipment to be used during the study. Upon arrival to the laboratory on subsequent visits, subjects body mass was recorded, and subjects cycle equipment remained unchanged.

14.2.2 Familiarisation

All subjects had previously cycled on a motorised treadmill (Woodway, Gubh, Germany) during previous research studies or during pilot data collection for this study. However, initial familiarisation sessions on treadmill cycling were completed to ensure that subjects were comfortable with, i) changes in treadmill speed and gradient, ii) riding in and out of the saddle, iii) changing gears on the bicycle, iv) high intensity loads with weights placed on the pulley system, v) maintaining a position at the front of the treadmill. Other familiarisation of feeding, blood sampling and gas analysis on the treadmill were carried out during this period. Subjects also completed on at least two occasions the performance test to be carried out at the end of each condition on the treadmill.

14.2.3 Calibration rides

All subjects completed a series of calibration rides on the treadmill in order to calculate the power demands for each individual on the treadmill (as discussed in 13.2.2.4). The SRM power cranks were fitted to the subject's bicycle and calibrated (8.2.2.2.2). Tyre pressure was standardised at 100psi, and the value for subject plus cycle mass was recorded on a calibrated beam scale (Seca) with subjects barefooted and wearing just cycling shorts. As body mass changes could influence the work rates during the study, a strategy was employed to ensure the combined weight of the bicycle and subject was the same during all trials. This was achieved by placing a large, full water bottle (750g), in the bottle cage of the bicycle during weighing, which could be emptied if the subject increased body mass during the study. Weights could be placed in the pockets of subjects racing jersey if their body mass decreased during the study. Subjects were otherwise clothed in cycling shorts and socks, with optional use of a protective crash helmet.

Subjects then warmed up on the treadmill and the pulley line was attached to the subjects' saddle rail during this period. Subjects then rode at set gradients and speeds (25km.h^{-1} at 0%, 2.5% and 5%, plus 36.6km.h^{-1} at 0% gradient). During the calibration period, riders were cooled by a large fan (Grants Electrical Services, Ballymena, Northern Ireland) with wind speed kept constant (10 m.sec^{-1}) throughout all tests. The power demands were recorded during this period. A regression line was drawn through the 25 km.h^{-1} data points, and used to calculate the power demands at varying gradients and speeds on the treadmill (the slope of the line is an individual equation based upon body mass, cycle mass, and efficiency of cycle equipment).

14.2.4 Experimental procedures

Following familiarisation, subjects were required to complete a further six visits to the laboratory. A cross over design was employed for this study, and subjects were scheduled for two sessions per week for three weeks, with a minimum of 48 hours separating tests. Following the completion of tests 1 and 2, the remaining tests were completed in random order. Supplementation during the protocol was conducted in a single blind fashion.

14.2.4.1 Test 1. Kingcycle maximum minute power (MMP) test.

Following subject measurements, subjects' bicycles were fitted with SRM powercranks, and were mounted on the Kingcycle test rig and calibration procedures were followed (8.2.2.1.2). Subjects were then allowed to warm up and indicated to the experimenter when they were ready for the test to begin.

Subjects underwent a Kingcycle MMP test as described in 8.2.2.1.2. Prior to testing subjects were fitted with a face mask (Hans Rudolf Inc. Kansas, USA) for online gas analysis (Covox Microlab, Exeter, England).

Heart rate (Polar Sports Tester, Kempele, Finland) and power (SRM) were also recorded throughout the test. Subjects were cooled by a large fan during the test (Grants Electrical Services, Ballymena, Northern Ireland). All subjects were verbally encouraged throughout the test.

14.2.4.2 Test 2. Individual subject data

Speed and weight to be added to the pulley system was calculated from an individual calibration ride on the treadmill (14.2.3). Power output was recorded at various treadmill speeds and gradients of 0 and 5%. From this data the protocol could be created with weights for the basket, and speeds for the treadmill being individual to each rider.

14.2.4.3 Tests 3 and 4. Road race simulation

After weighing and correcting for body mass differences, subjects mounted their bicycle on the treadmill and the pulley system was attached to the saddle rails of the subject's bicycle. Subjects then completed a 10-minute warm up at their own requested intensity and were allowed to drink water ad libitum. On completion of the warm up subjects' bicycles were held enabling subjects to freewheel and a thumb prick capillary blood sample was taken (50 µl) (Unilet lancets, Owen Mumford, Oxford, England) and analysed for B[Lac⁻] and blood glucose concentration (Yellow Springs Instrument, Stat Plus 2300, Ohio USA). A further sample was taken (150 µl) and immediately centrifuged (Hawksley and Son Ltd. Lancing, England) and analysed for plasma urea and Ammonia (NH_3) concentrations (Eastman Kodak Company. New York, USA).

Subjects then began the simulation which consisted of 4 x 30 minute 'laps' at varying power outputs (figure 13.5) developed from actual road race data (SRM) equating to an average of 60% MMP.

Changes in power were achieved by altering speed and gradient of the treadmill, and by placing weights on the basket. At set intervals subjects were also instructed to drift up and down the treadmill during the protocol to increase the intermittent nature of the test, and match the demands from data recorded in the field. During the trial subjects were fed 8 x 94ml (750ml total) of an artificially sweetened (aspartamine) water placebo or an 11.7% glucose polymer solution (Teknofuel, Medway, UK). Subjects were fed the assigned fluid at 3 and 22 minutes of each 30-minute lap. Subjects were passed up a nose clip and mouthpiece (Hans Rudolf) two minutes prior to Online gas analysis (Covox) at 13-21 minutes and 24-28 minutes of each lap. Blood samples were taken with identical methods to the post warm up procedure, a 50 µl sample was analysed for [Lac⁻] and glucose at 21 minutes into each lap. On the completion of each lap, a further 200 µl blood sample was taken and analysed for [Lac⁻], glucose, urea and NH_3. Heart rate was monitored throughout the trial (Polar, Kempele, Finland). Power was recorded by the SRM Powercrank on 4 subjects during the four trials. During the trial subjects could select any gear ratio, and ride in or out of the saddle as would be their choice during an actual race. On completion of the trial subjects immediately underwent the performance test on the treadmill (14.2.4.5).

14.2.4.4 Tests 5 and 6. Even Power Trials.

Following preparation and warm up procedures identical to the simulated road race trials, subjects performed two steady state trials two hours in duration on the motorised treadmill. One trial was conducted with subjects fed a glucose polymer supplement, and one trial on the placebo drink (both drinks were identical to the simulated road race trial for volume and concentration of the supplements).

119

The steady state trials were conducted over the same treadmill gradients as the simulated road race protocol, however, by manipulating the speed of the treadmill, and by placing weights on the weights basket attached to the subjects bicycle saddle rails, the power output was maintained at a constant 60% MMP. The data collection procedures for power, heart rate, blood sampling and gas analysis were identical to the simulated road race trial. On completion of each 2-hour trial the performance test was carried out on the treadmill.

14.2.4.5 Test 7. The Performance Test

On completion of the 4 laps (2 hours), the subjects were required to finish the trial with a performance test. Subjects rode into the performance test without a break from the end of their trial. The treadmill gradient was taken up to 6% gradient and the speed of the treadmill adjusted to elicit 70% MMP. This pace was held for 3 minutes. On completion of the 3-minute period the clock and distance were reset and subjects were instructed to complete 2 km as fast as possible. The subject orally instructed the experimenter how the speed of the treadmill was to be adjusted. Previous attempts at the climb gave subjects an indication of duration of the test, and all subjects were instructed that the most efficient pacing strategy was even paced. Heart rate was monitored throughout the performance test and power was also recorded. Subjects were allowed to view time and distance covered during the performance test. This information was also orally communicated to the subject by an additional member of laboratory staff. On completion of the test subjects spent 3 minutes of active recovery on the treadmill before a final blood sample was taken and analysed for B[Lac⁻] and glucose.

14.2.4.6 Test 8. Fresh Performance Test

Subjects arrived at the laboratory having prepared for the test as in the steady state and road race trials. Subjects warmed up and indicated to the experimenter when they were ready to start the test. The protocol was identical to the performance test carried out after each of the trials, but without the 2-hour trial beforehand. Data collection for heart rate, power, and B[Lac⁻] were identical to the performance tests after each trial.

14.2.5 Statistical Analysis

Data from the four laps and the performance test was evaluated using a two-way (glucose/placebo and intermittent/even power) repeated measures Analysis of Variance test, with Tukey post hoc analysis for significant differences (Statisticamac). The coefficient of variation was calculated for the power output recorded by the four subjects that rode the SRM powercrank during their trials.

14.3 Results (pilot study)

14.3.1 Introduction

In order to assess the practicality of using the pulley system, the motorised treadmill, and the planned sampling techniques, a pilot study was conducted prior to experimental procedures. The protocol for the pilot study elicited 62% of MMP.

14.3.2 Results and Discussion

The subject details of the five male trained cyclists who took part in this study are shown in table 14.1.

Table 14.1 Subject details for the pilot study. (mean ± S.D.).

Subjects	n=5
Age	28 ± 8 years
Height	1.74 ± 0.03 m
Mass	66.4 ± 6.2 kg
Maximum Minute Power (SRM)	377.4 ± 22.4 watts

Only 3 subjects managed to complete the testing protocol, and each of those had a period where they needed to stop during the 2-hour testing period.   Premature exhaustion during the 2-hour simulation for the remaining 3 riders was seen during this study, with exercise induced muscle cramps, and the inability to ride at 70% of maximum minute power prior to the performance test being the main problem. Therefore the protocol was revised for actual study with the % MMP being reduced to 58% (figure 13.5). Data on 3 subjects who completed all four trials of the 2-hour simulation are shown in table 14.2.

Table 14.2 Pilot study results on three subjects

	Even Power (glucose)	Even Power (placebo)	Road Race Simulation (glucose)	Road Race Simulation (placebo)
Power Output (watts)	234 ± 14.8	234 ± 14.5	233 ± 9.5	232 ± 10.7
Blood Glucose ($mmol.l^{-1}$)	4.64 ± 0.60	4.3 ± 0.71	5.49 ± 0.85	4.4 ± 0.64
B[Lac⁻] ($mmol.l^{-1}$)	2.62 ± 0.58	2.17 ± 0.51	5.17 ± 0.81	4.58 ± 0.71
$\dot{V}O_2$ $l.min^{-1}$	3.16 ± 0.23	3.07 ± 0.32	3.22 ± 0.33	2.98 ± 0.37
$\dot{V}O_2$ $ml.kg^{-1}.min$	45 ± 2.52	44 ± 4.16	46 ± 4.04	43 ± 5.13
RER	0.89 ± 0.01	0.87 ± 0.02	0.90 ± 0.01	0.91 ± 0.02
Heart Rate ($beats.min^{-1}$)	149 ± 8	146 ± 9	157 ± 8	147 ± 11

The power output recorded during these trials appears reproducible. The coefficient of variation for the road race simulation trials was 0.649% and for the even power trials 0.479%. Sources of error are

likely to come from the motorised treadmill controls (sometimes flicking between treadmill speeds (±0.2 kph), plus the duration of blood sampling sometimes varying due to experimental procedures.

14.4 Results (main study)

Subject details of the six competitive road riders are shown in table 12.1. All subjects were tested during a set training phase and complete the testing protocol within a 10-day period.

14.4.1 Power

Power output was measured on four riders during the protocol. Mean absolute power and relative power output (% maximum minute power) during the trials are shown in table 14.3. There was no significant difference between trials for power (p=0.79). The coefficient of variation for the road race simulation trials was 1.35%, and for the even power trials was 1.86%.

Table 14.3 Mean power output during all trials (mean ± S.D.).

	Even Power (glucose trial)	Even Power (placebo trial)	Road race (glucose trial)	Road race (placebo trial)
Power output (watts)	253.6 ±10.3	250.1 ±11.0	259.2 ± 18.3	260.5 ±16.7
% maximum minute power	61.4 ± 2.0	60.6 ±1.6	62.8 ± 0.7	63.0 ± 0.7

Figures 14.1 and 14.2 Show the even power and road race simulation protocols. Figure 13.6 shows the distribution of power output during the road race simulation study.

Figure 14.1 Power output during the even power trial (n=1)

122
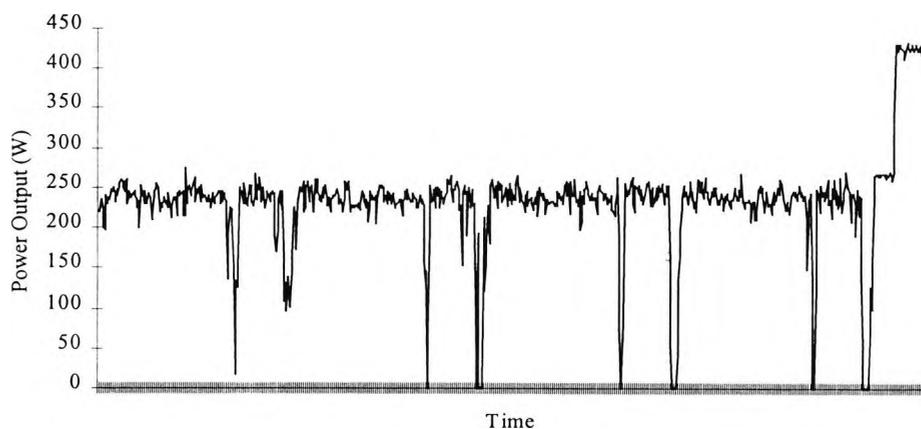
Figure 14.2 Power output during the simulated cycling trial
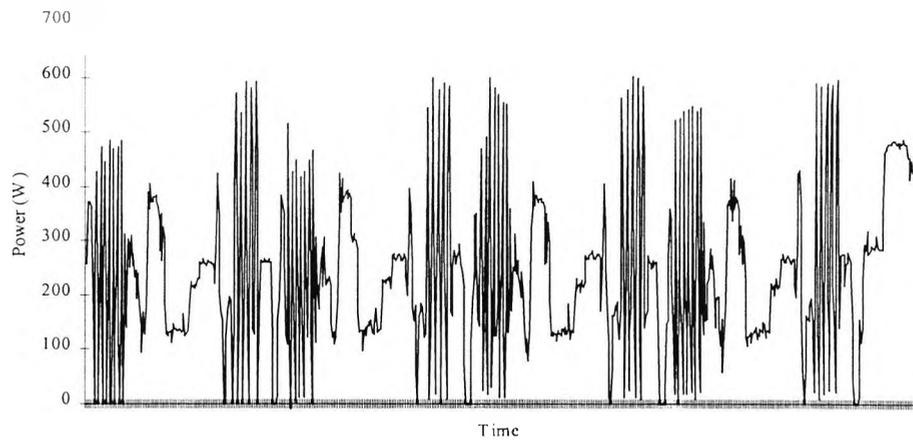
14.4.2 Blood measures

Data analysis was carried out on the mean of the two samples taken per 'lap' during both the road race simulation trials, and the even power trials for measures of $BLac^-$ and blood glucose. The data analysis of plasma NH_3 and plasma urea were from the single sample taken during each 'lap' of the protocol.

14.4.2.1 Blood Lactate

The mean $B[Lac^-]$ was significantly elevated during the road race trials compared to the even power trials (3.25 ±1.24 vs 1.78 ±0.92 mmol.l^{-1}, p=0.014). Figure 14.3.

There was no significant difference in $B[Lac^-]$ between glucose supplement and placebo trials (2.55 ± 1.38 vs. 2.48 ±1.20 mmol.l^{-1}, p=0.71). There was also no significant change over time for $B[Lac^-]$ during any trial (p=0.94).

Figure 14.3 Blood lactate concentration during the even power and road race trials
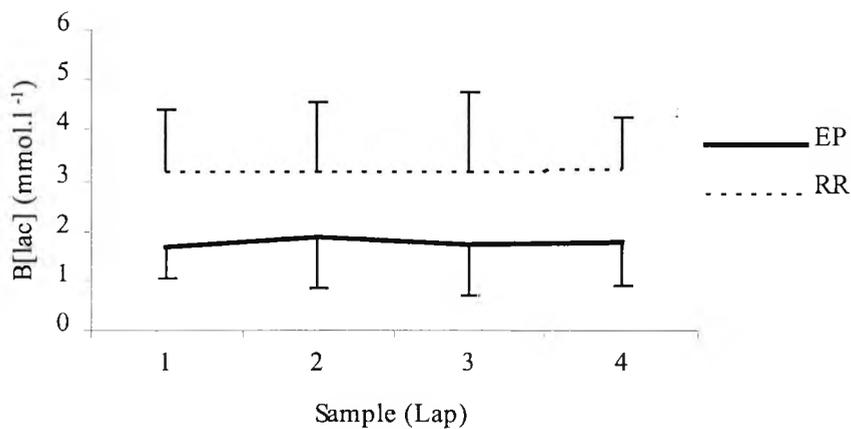
Figure 14.4 Blood lactate concentration during glucose and placebo trials
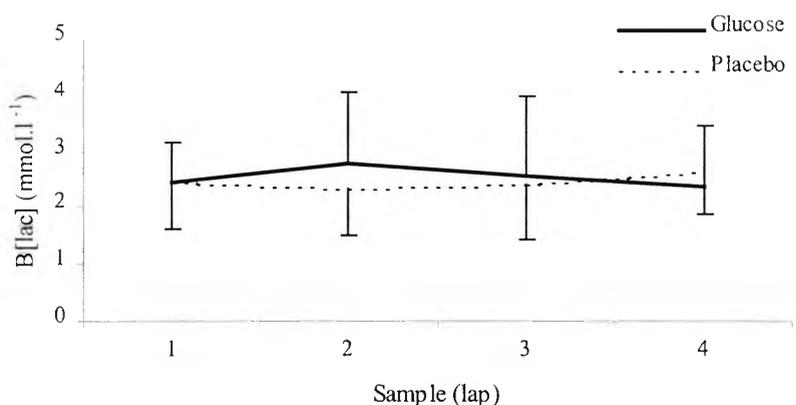
14.4.2.2 Blood Glucose

There was no significant difference for the mean values between the even power and the road race trials $(4.53 \pm 0.67$ vs 4.61 ± 0.74 mmol.l^{-1}, p=0.81).

Figure 14.5 Blood glucose concentration during even power and road race trials.
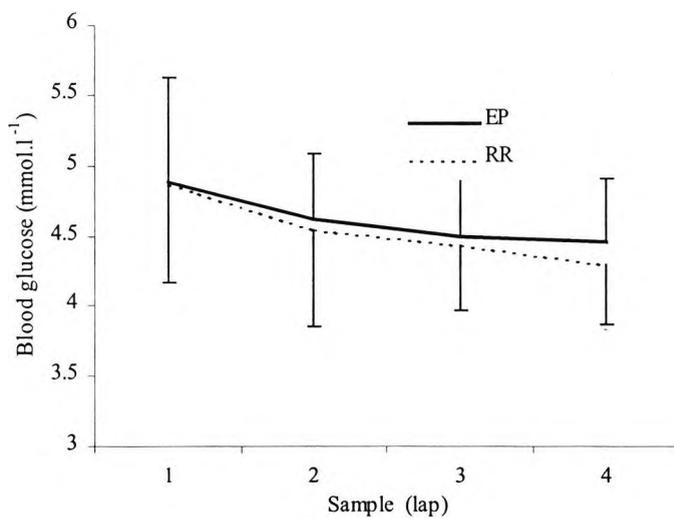
The mean blood glucose concentration was significantly higher in the glucose compared to the placebo trials $(4.93 \pm 0.73$ vs 4.22 ± 0.43 mmol.l^{-1}, p=0.046). During both trials there were significant

reductions in blood glucose over time (p=0.0009). Post hoc analysis revealed blood glucose concentrations during lap 1 were significantly higher than laps 3 (p=0.009), and lap 4 (p=0.0003). There was also a significant reduction in blood glucose between laps 2 and lap 4 (p=0.013).

Figure 14.6 Blood glucose concentration between glucose and placebo trials.
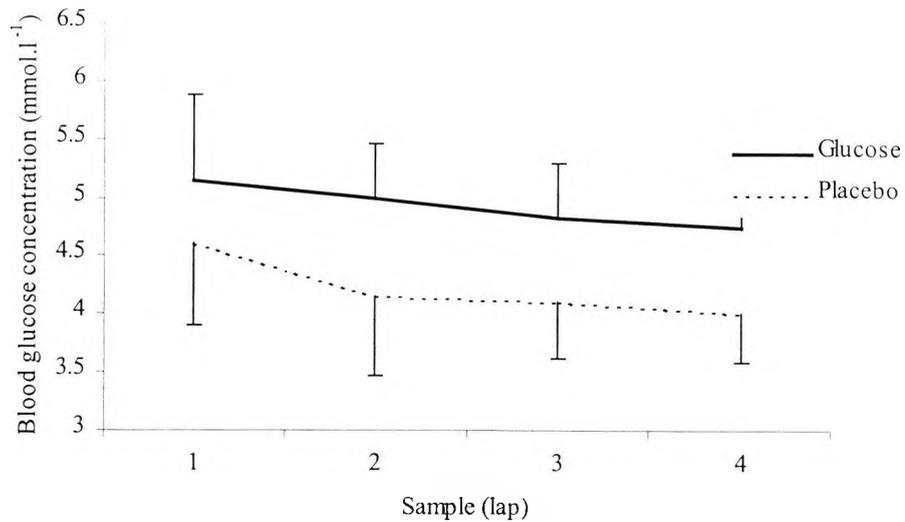
14.4.2.3 Plasma Ammonia

The post warm up plasma ammonia concentration was 116.3 ± 9.3 $\mu mol.l^{-1}$ There were no significant differences between even power and road race simulation trials for post warm up values (p=0.769). There were no significant differences in plasma $[NH_3]$ between even power and road race trials (p=0.26), and likewise between glucose and placebo trials (p=0.2).

Figure 14.7 Plasma ammonia concentrations during all trials.
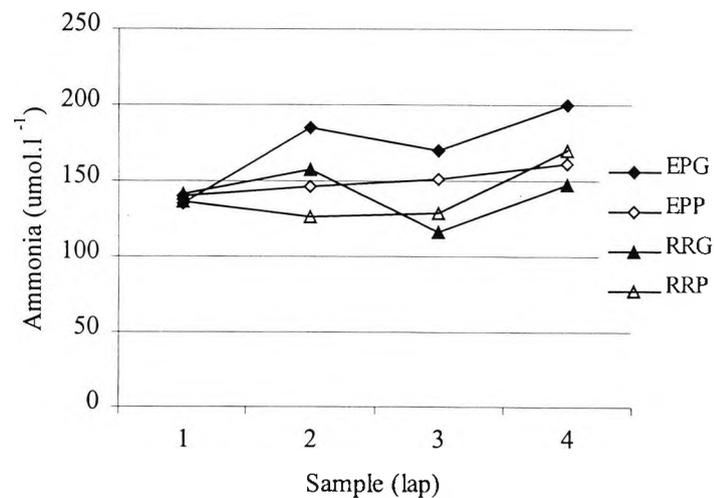
Table 14.4 Lap by lap plasma ammonia (μmol.l·min^{-1}) concentrations during the four trials.
(mean ± SD). EP = even power, RR = road race, G = glucose, p = Placebo.

Lap	EPG	EPP	RRG	RRP
1	134.8 ± 41.9	139.7 ± 41.8	140.8 ± 34.1	135.7 ± 39.3
2	185.5 ± 69.5	146.0 ± 52.3	157.5 ± 82.3	126.4 ± 39.9
3	169.7 ± 66.3	150.8 ± 62.1	166.2 ± 40.0	129.2 ± 53.3
4	200.0 ± 78.2	161.3 ± 53.0	147.0 ± 48.5	170 ± 84.9
Mean ± s	172.4 ± 55.5	149.5 ± 48.2	145.5 ± 50.9	135.4 ± 49.7

No significant changes in plasma [NH$_3$] were seen over time (p=0.053), Figure 14.7, table 14.4. Figures 14.8 and 14.9 show the relationship between B[Lac⁻] and NH$_3$ samples during the even power and the road race simulation trials. A significant correlation was found between B[Lac⁻] and plasma [NH$_3$] during simulated road race cycling, but not during even power cycling.

Figure 14.8 The relationship between blood lactate and plasma ammonia concentrations during even power cycling.
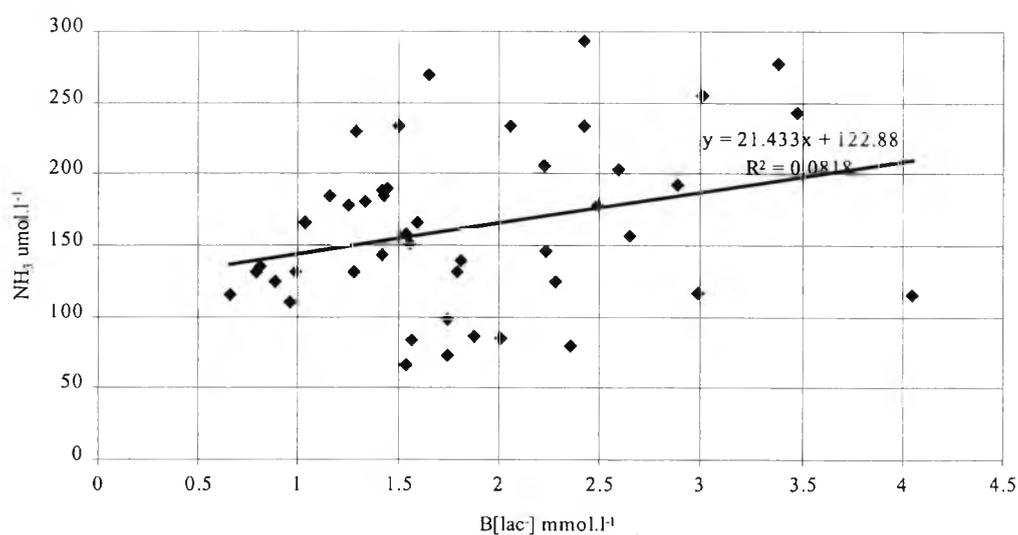
Figure 14.9 The relationship between blood lactate and plasma ammonia concentrations during simulated road race cycling.
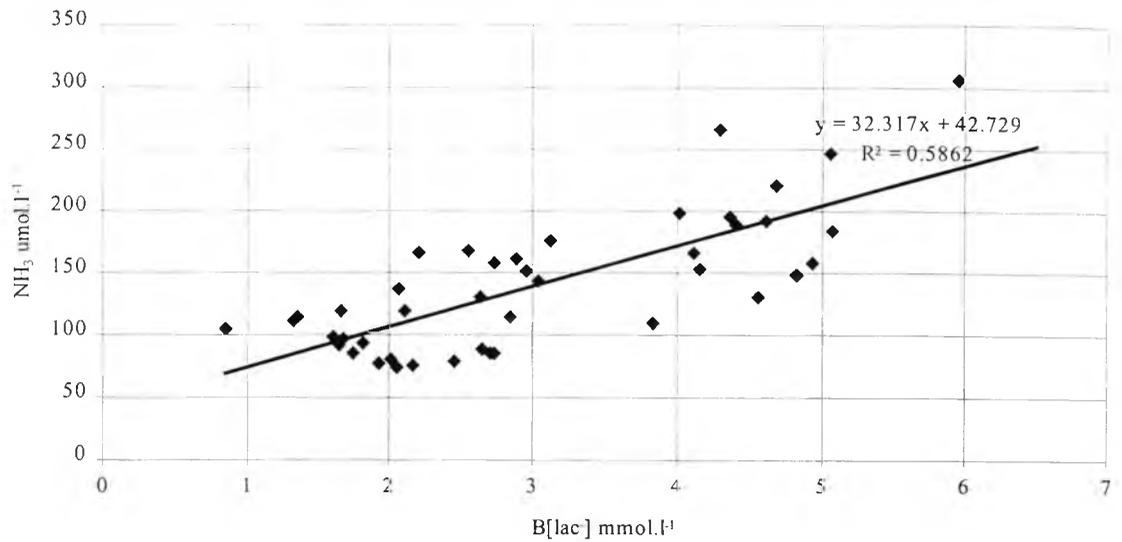
14.4.2.4 Plasma Urea

Mean post warm-up values for plasma urea were 7.37 ± 1.62 mmol.l^{-1}. There were no significant differences between trials for post warm up values (p=0.98). (figure 14.10).

The during trial samples were not significantly different between the even power and the road race trials (p=0.65), or between glucose and placebo conditions (p=0.46). Mean concentrations for the four conditions are shown in table 14.5.

There were no statistically significant changes in plasma urea over time (p=0.129).

Table 14.5 Plasma urea concentrations (mmol.l^{-1}min^{-1}) during the four trials. (mean $\pm$ S.D.).

Lap	EPG	EPP	RRG	RRP
1	7.58 ± 2.16	7.2 ± 1.49	7.17 ± 1.38	7.5 ± 1.91
2	8.10 ± 2.79	7.5 ± 1.71	7.58 ± 1.54	7.8 ± 1.14
3	7.98 ± 2.33	7.28 ± 1.71	7.88 ± 2.61	7.7 ± 1.15
4	8.74 ± 1.79	7.87 ± 1.94	7.85 ± 1.8	7.3 ± 0.99
Mean $\pm$ S.D.	8.1 ± 0.48	7.46 ± 0.30	7.62 ± 0.33	7.57 ± 0.22

Figure 14.10 Plasma urea concentration during all trials.
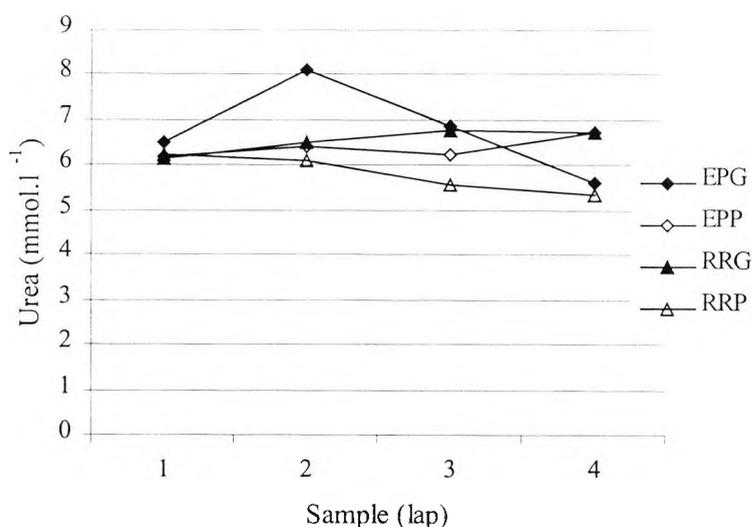
14.4.3 Respiratory data

Respiratory data was collected for two periods during each 'lap' of the trials. A mid lap sample, and an end of lap sample.

During the mid lap sample, a period of matched work rate between the even power trials and the road race simulation trial was recorded. Prior to the matched period, the subjects wore the mouthpiece and gas data collected for six minutes during the mid lap sample where the workloads were not matched between even power and the road race simulation trials.

The results presented therefore represent the 'mid lap matched' value plus the 'total mid lap' sample and an 'end of lap' sample. The data was analysed as absolute $\dot{V}O_2$ values, relative to body mass ($ml\,kg^{-1}min^{-1}$), and as a percentage of $\dot{V}O_2max$.

14.4.3.1 Oxygen Consumption ($\dot{V}O_2$)

For the matched work rates between the even power and the road race trials, there was no significant difference for the $\dot{V}O_2$ for the mean values during this sampling periods (p=0.31) (figure 14.11). There were no significant differences between the glucose and placebo trials (p=0.352).

For the total mid lap gas sample there were significant differences for the total $\dot{V}O_2$ between the even power and the road race trials (3.13 vs 2.94 $l\,min^{-1}$, p=0.00004). There were significant increases over time for this sampling period; lap 1 was significantly lower than lap 2 (p=0.023), lap 3 (0.0004), and lap 4 (p=0.0012). For the sampling period at the end of each 'lap' there were no significant differences between even power and road race trials (p=0.104), or between glucose and placebo trials (p=0.52). There were significant differences between laps for the $\dot{V}O_2$ sample at the end of the lap (p=0.006). Post hoc analysis revealed lap 1 was significantly lower than lap 4 (p=0.036), and lap 2 was also significantly lower than lap 4 (p=0.007)

Figure 14.11 Oxygen consumption (l min^{-1}) during all trials.
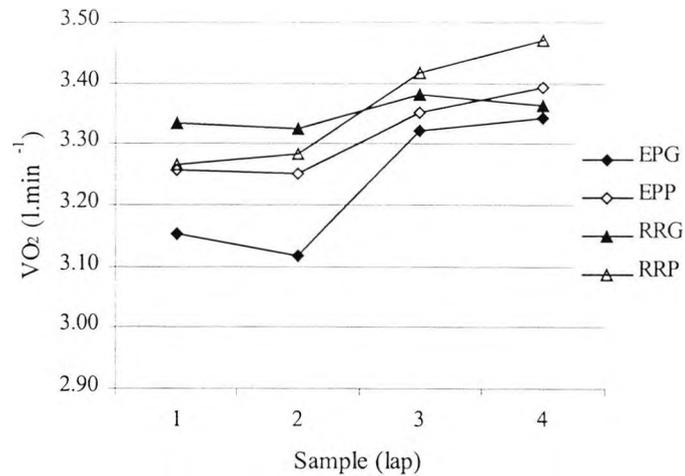
Table 14.6 Total oxygen consumption data from three samples during all trials (mean ± S.D.)

Trial	Mid Lap Matched Sample	Mid Lap Total Sample	End of Lap Matched Sample
Even Power (glucose Trial)	3.24 ± 0.40	3.13 ± 0.46	3.26 ± 0.41
Even Power (placebo Trial)	3.29 ± 0.36	3.18 ± 0.45	3.31 ± 0.39
Road Race Simulation (glucose trial)	3.38 ± 0.40	2.98 ± 0.43*#	3.35 ± 0.35
Road Race Simulation (placebo trial)	3.28 ± 0.40	2.89 ± 0.42*#	3.36 ± 0.38

* denotes significant difference from even power glucose trial

# denotes significant difference from even power placebo trial

Table 14.7 Changes in oxygen consumption over time during all conditions mean of total sampling periods (l·min^{-1}, mean ± S.D.)

Trial	Lap 1	Lap 2	Lap 3	Lap 4	$\dot{V}O_2$ 'Drift'
Even Power (glucose trial)	3.05 ± 0.29	3.00 ± 0.35	3.21 ± 0.36	3.21 ± 0.37	0.16
Even Power (placebo trial)	3.24 ± 0.21	3.21 ± 0.25	3.29 ± 0.26	3.32 ± 0.26	0.08
Road Race Simulation (glucose trial)	3.12 ± 0.50	3.12 ± 0.49	3.12 ± 0.51	3.13 ± 0.55	0.01
Road Race Simulation (placebo trial)	3.02 ± 0.53	3.06 ± 0.49	3.16 ± 0.56	3.20 ± 0.57	0.18

14.4.3.2 Carbon dioxide concentration in expired air

Carbon dioxide production was significantly higher for the total mean data for the road race simulation trials compared to the even power trial for the gas sample at the end of the lap sample (3.42 ± 0.36 vs 2.88 ± 0.09 $lmin^{-1}$, p=0.0001) (table 14.8). During the total mid lap sample there were significantly higher concentrations of carbon dioxide in the expired air during the even power trial compared to the road race simulation trials (2.94 ± 0.12 vs 2.31 ± 0.56 $lmin^{-1}$, p=0.0001). There was also a significant difference between the matched mid lap sample between the even power and road race conditions (p=0.028).

Table 14.8 Carbon dioxide concentration of expired air during each 'lap' of all trials (end of lap sample). (mean ± S.D.)

Trial	Lap 1	Lap 2	Lap 3	Lap 4
Even Power (glucose trial)	2.92 ± 0.34	2.85 ± 0.38	2.94 ± 0.52	2.78 ± 0.45
Even Power (placebo trial)	2.99 ± 0.35	2.88 ± 0.44	2.85 ± 0.56	2.83 ± 0.52
Road Race Simulation (glucose trial)	3.53 ± 0.38	3.45 ± 0.39	3.36 ± 0.54	3.43 ± 0.54
Road Race Simulation (placebo trial)	3.43 ± 0.35	3.32 ± 0.41	3.47 ± 0.58	3.33 ± 0.51

Figure 14.12 Carbon dioxide production during all trials (matched samples).
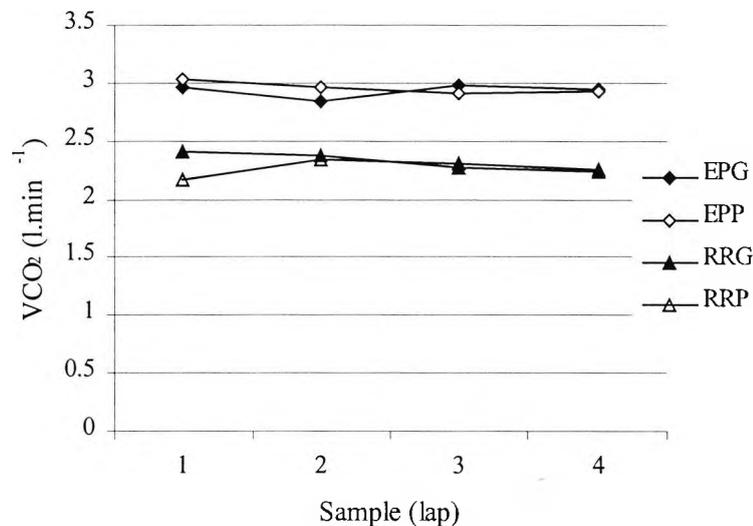
Glucose polymer ingestion had no influence on $\dot{V}CO_2$ production for the mid lap matched sample (p=0.84), for the total mid lap sample (p=0.11) or for the end of lap sample (p=0.53).

14.4.3.3 Respiratory exchange ratio

The RER values for the matched sampling period were not significantly different between the even power and the road race simulation trials (p=0.11) (figure 14.12). For the same sampling period there were also no

differences between glucose and placebo trials (p=0.37). There were however significant differences over time (p=0.0002). Post hoc analysis revealed lap 1 RER values were significantly higher than laps 3 (p=0.0004) and lap 4 (p=0.0006). Lap 2 RER values were also significantly higher than laps 3 (p=0.024) and lap 4 (p=0.0018).

An identical pattern was observed for the total mid lap sample. There were no significant differences between even power and road race simulated trials (p=0.206), or between glucose and placebo trials (p=0.371). There were however changes over time (p=0.00001). Post hoc analysis indicated that RER values on lap 1 were significantly higher than laps 3 (p=0.0002), and 4 (p=0.0001). Lap 2 RER values were also significantly higher than laps 3 (p=0.006), and 4 (p=.0002).

There were also significant differences between even power and simulated road race cycling for the end of lap sample (p=0.004), there were no significant differences between glucose and placebo trials for this period (p=0.26). There were significant changes during the trials over time for this sampling period (p=0.0005). Post hoc analysis showed lap 1 RER values were significantly higher than laps 3 (p=0.0005) and 4 (p=0.0001). Lap 2 RER values were also significantly higher during lap 2 compared to lap 4 (p=0.0005).

Figure 14.13 Respiratory exchange ratio during the even power and road race trials (matched samples).
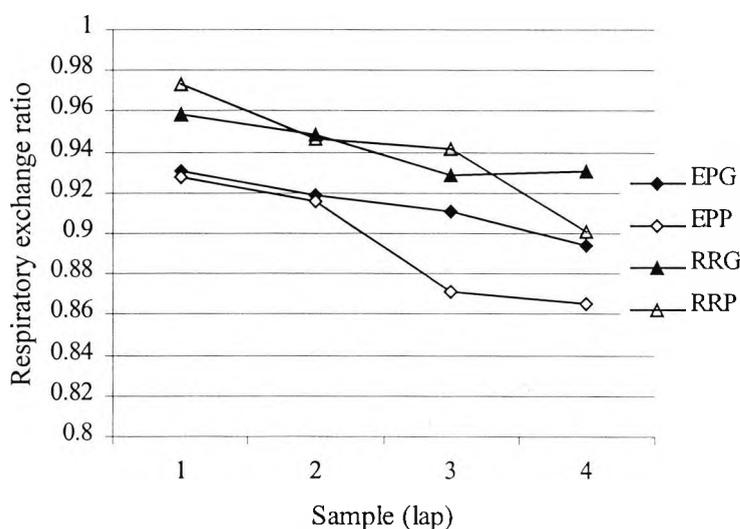
Mean RER values for each lap are shown in table 14.9.

Table 14.9 Mid Lap matched RER samples over each lap of all trials. (mean ± S.D.)

Trial	Lap 1	Lap 2	Lap 3	Lap 4	Mean ± s
Even Power (glucose trial)	0.92 ± 0.04	0.90 ± 0.04	0.90 ± 0.03	0.89 ± 0.03	0.90 ± 0.01
Even Power (placebo trial	0.92 ± 0.05	0.91 ± 0.05	0.87 ± 0.05	0.87 ± 0.04	0.89 ± 0.03
Road Race Simulation (glucose trial)	0.97 ± 0.04	0.95 ± 0.04	0.93 ± 0.04	0.93 ± 0.04	0.95 ± 0.02
Road Race Simulation (placebo trial)	0.96 ± 0.03	0.96 ± 0.03	0.94 ± 0.04	0.91 ± 0.03	0.94 ± 0.02

14.4.4 Economy and Efficiency data

14.4.4.1 Gross Mechanical Efficiency

Gross mechanical efficiency calculations were made for the total mid lap sample and at the matched work rate during this period.

No efficiency calculations were made during the end of lap sample due to RER values exceeding 1.0 in some subjects.

14.4.4.1.1 Matched work rate sample

There were no significant differences between the matched gross mechanical efficiency values between even power and simulated road race trials (23.25 ± 1.74 vs 22.82 ± 1.53 %, p=0.09). There were also no significant differences between the glucose and placebo trials for this sampling period (p=0.317) or for the total mid lap sampling period (p=0.249). For the matched sampling period there was a significant drop in efficiency over time (p=0.009). Post hoc analysis indicated that lap 1 efficiency values were significantly higher than lap 3 (p=0.009) and lap 4 (p=0.029) figure 14.13. For the total mid lap sampling period, even power trials were significantly lower than during simulated road race cycling (p=0.0005).

Figure 14.14 Gross mechanical efficiency during all trials (matched samples).
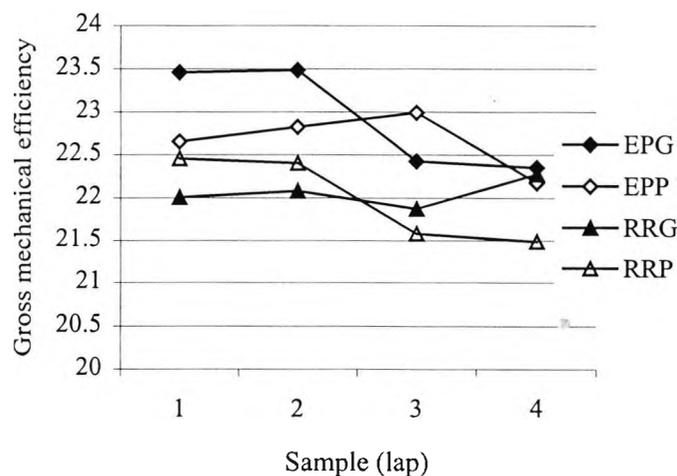
14.4.4.2 Economy

Economy was measured for the total mid lap sample, the matched work rate during the mid lap sample, and during the end of lap gas sample.

There were significant differences between economy values for the matched mid lap sample between the even power and road race trial, $(84.25 \pm 9.56$ vs 78.61 ± 4.99 W.$(l.O_2^{-1})$, p=0.001). There were no significant differences between glucose and placebo trials $(81.46 \pm 7.63$ vs 81.40 ± 6.92 W.$(l.O_2^{-1})$, p=0.57). Over time, significant reductions in economy were observed (p=0.000). Post hoc analysis revealed significant reductions in economy occurred only in the road race simulation trials, with lap 1 values were significantly higher than lap 3 (p=0.00018) and lap 4 (p=0.0034).

For the total mid lap sample there were also significant differences between even power and simulated road race trials (p=0.0007). There were no significant differences between glucose and placebo trials (p=0.43). Significant reductions in economy over time were also recorded for this sampling period (p=0.0019). Post hoc analysis indicated that lap 1 economy values were significantly higher than lap 2 (p=0.026), lap 3 (p=0.0018) and lap 4 (p=0.009).

The end of lap sample showed no significant differences in economy between even power and simulated road race trials (p=0.288), or glucose and placebo trials (p=0.601). There were changes in economy over time for this sampling period (p=0.005). Post hoc analysis indicated that lap 1 economy values were significantly higher than lap 4 (p=0.039) Lap 2 economy values were also significantly higher than lap 4 (p=0.005).

14.4.4.2.1 End of 'Lap' Sample

There were no significant differences between the end of lap samples between the even power and road race trials $(76.29 \pm 3.33$ vs 79.40 ± 5.42 W.$(l.O_2^{-1})$, p=0.288), or between the glucose and placebo trials (p=0.601). There were significant reductions in economy between lap 1 and lap 4 (p=0.003), and lap 2 and lap 4 (p=0.005) for this sampling period.

14.4.5 Heart Rate

There were no significant differences between the even power trials and the road race simulation trials for heart rate $(147 \pm 7$ vs 151 ± 7 beats.min^{-1}, p=0.19). There were also no significant differences between glucose and placebo trials $(149 \pm 7$ vs 149 ± 7 beats.min^{-1}, p=0.99). Over time, there were however significant rises in heart rate (p=0.0000) (table 14.10). Lap 1 heart rates were significantly lower than lap 2 (p=0.04), lap 3 (p=0.026) and lap 4 (p=0.001). Heart rates during lap 4 were also significantly higher than lap 2 (p=0.0009) and lap 3 (p=0.001).

Figure 14.15 Relative heart rate during all

trials.
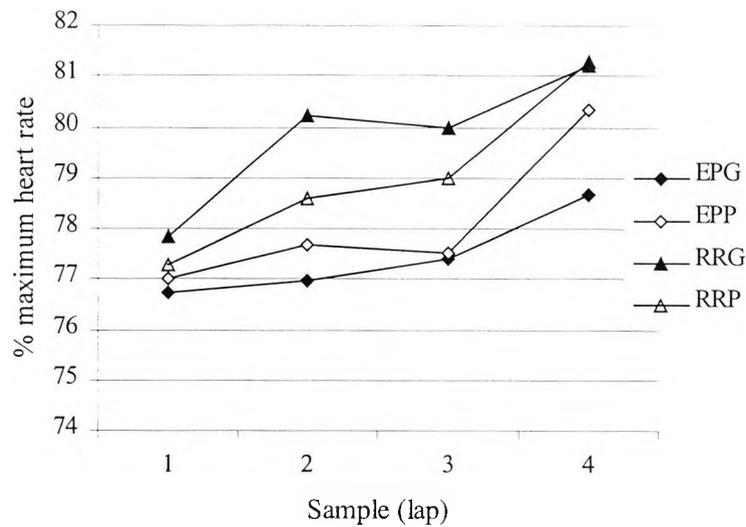
Table 14.10 Heart rate response during the four trials (mean ± S.D.).

	Lap 1	Lap 2	Lap 3	Lap 4
Even power (glucose trial)	145 ± 7	146 ± 7	147 ± 7	149 ± 7
Even Power (placebo trial)	146 ± 6	147 ± 8	147 ± 7	152 ± 7
Road race simulation (glucose trial)	147 ± 6	152 ± 8	152 ± 6	154 ± 8
Road race simulation (placebo trial)	147 ± 6	149 ± 9	150 ± 8	154 ± 8

14.4.6 One Subject Data

Online gas analysis was performed on one subject for 24 minutes during each lap of each of the four trials (total 98 minutes of the 120 minute protocol).   Mean values for each condition are shown in table 14.11.

14.4.6.1 Oxygen consumption

Figure 14.15 shows the pattern of oxygen consumption recorded on one subject during the even power and the road race simulation trials. The breaks in the data collection were due to consume the experimental drinks during the trials.

Lap by lap breakdown of the oxygen consumption data are shown in table 14.12.

Table 14.11 Respiratory data from all four conditions (n=1). (mean ± S.D.).

	Even Power (glucose trial)	Even Power (placebo trial)	Road Race Simulation (glucose trial)	Road Race Simulation (placebo trial)
$\dot{V}O_2$ l·min^{-1}	2.91 ± 0.20	3.28 ± 0.19	3.07 ± 0.80	2.98 ± 0.83
$\dot{V}O_2$ ml·kg^{-1}·min^{-1}	40.42 ± 2.71	45.15 ± 4.53	42.52 ±11.02	41.3 ± 11.38
% $\dot{V}O_{2max}$	53.8 ± 3.6	60.1 ± 6.0	56.6 ±14.7	55.0 ± 15.1
$\dot{V}CO_2$ l·min^{-1}	2.68 ± 0.19	2.74 ± 0.16	2.97 ± 0.87	2.83 ± 0.89
Respiratory exchange ratio	0.92 ± 0.04	0.83 ± 0.02	0.96 ± 0.07	0.95 ± 0.07

14.4.6.2 Carbon dioxide production

Mean values for carbon dioxide production (n=1) are shown in table 14.11. The $\dot{V}CO_2$ response is shown lap by lap in figure 14.16, and in table 14.13.

Table 14.12 Oxygen consumption during each lap for all trials (n=1). (mean ± S.D.).

	Lap 1	Lap 2	Lap 3	Lap 4
Even power (glucose)	2.85 ± 0.22	2.89 ± 0.21	2.84 ± 0.16	2.99 ± 0.16
Even power (placebo)	3.27 ± 0.22	3.28 ± 0.17	3.28 ± 0.08	3.28 ± 0.17
Road race simulation (glucose)	3.07 ± 0.81	3.09 ± 0.83	3.03 ± 0.76	3.06 ± 0.81
Road race simulation (placebo)	2.64 ± 0.98	3.14 ± 0.67	2.99 ± 0.79	3.04 ± 0.82

Figure 14.16 Oxygen consumption during even power and simulated road race cycling (n=1).
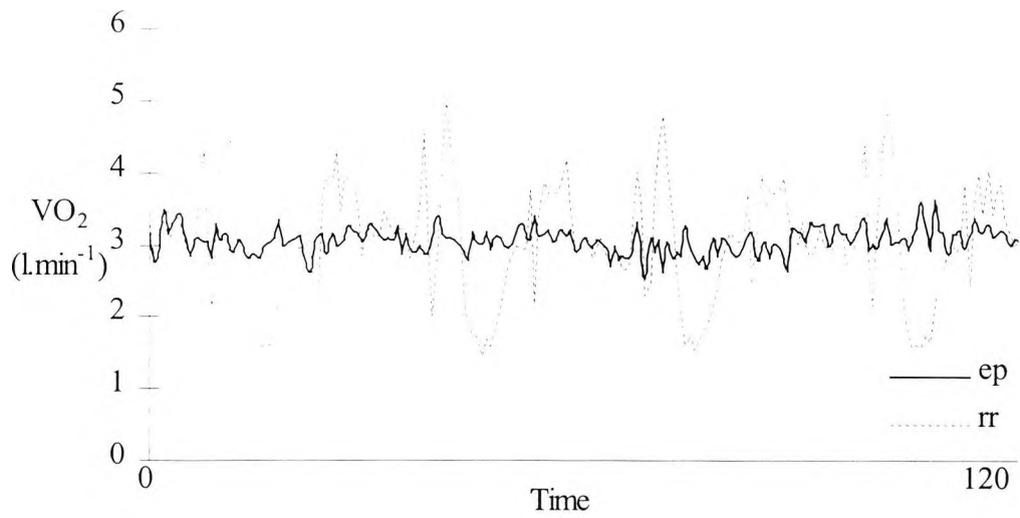
Figure 14.17 Carbon dioxide production during even power and simulated road race cycling (n=1)
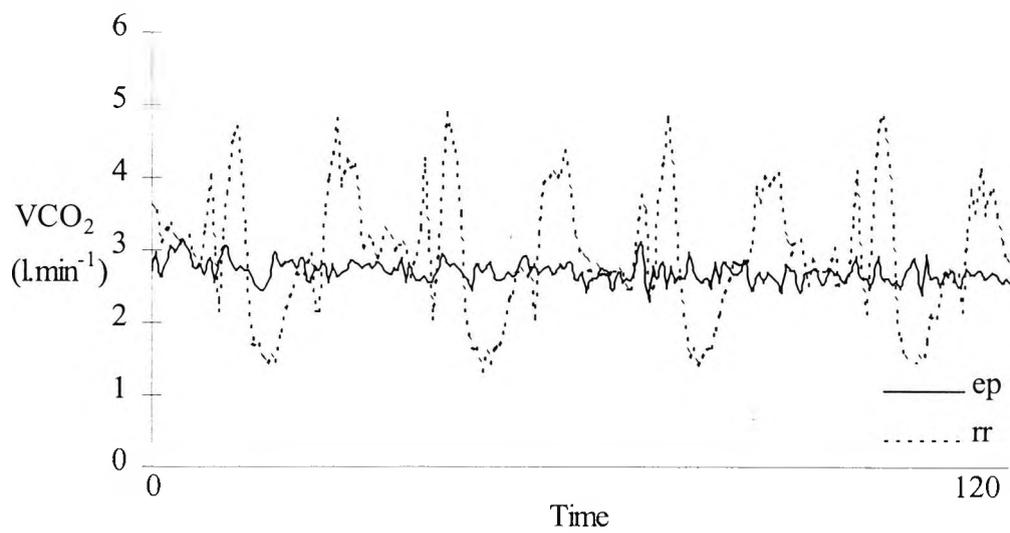
Figure 14.18 Respiratory exchange ratio during even power and simulated road race cycling (n=1).
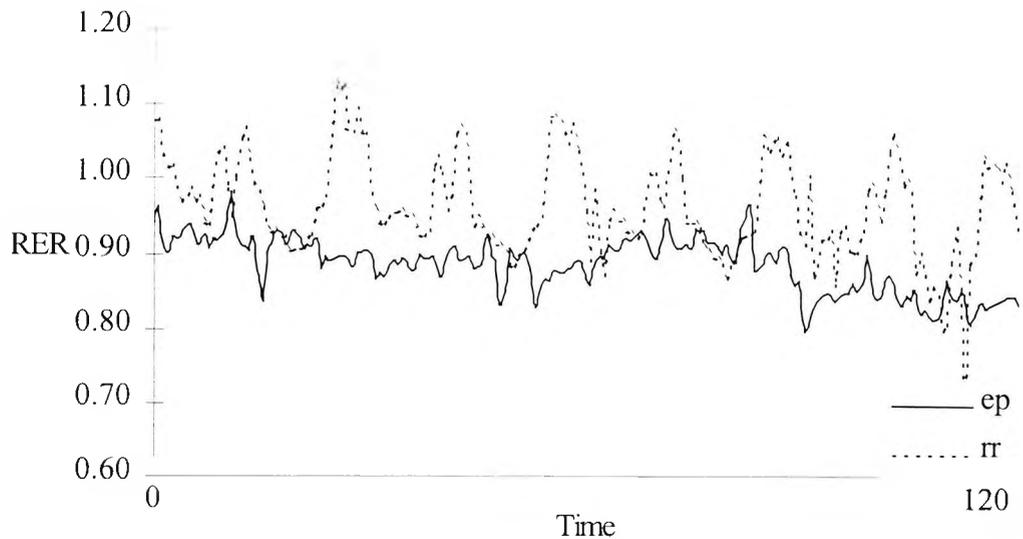
Table 14.13 Carbon dioxide concentration during each lap of all trials (n=1). (mean ± S.D.)

	Lap 1	Lap 2	Lap 3	Lap 4
Even power (glucose)	2.75 ±0.24	2.69 ± 0.17	2.65 ± 0.15	2.62 ±0.13
Even power (placebo)	2.81 ±0.19	2.73 ± 0.14	2.77 ± 0.14	2.68 ± 0.14
Road race simulation (glucose)	3.10 ± 0.91	3.00 ± 0.89	2.89 ± 0.83	2.87 ± 0.85
Road race simulation (placebo)	2.51 ± 1.00	3.00 ± 0.84	2.86 ± 0.83	2.86 ± 0.87

14.4.6.3 Respiratory exchange ratio

The RER values are shown in table 14.11. The 4-lap breakdown for each trial is shown in table 14.14, and in figure 14.17.

Table 14.14 Respiratory exchange ratio during each lap for each trial (n=1). (mean ± S.D.)

	Lap 1	Lap 2	Lap 3	Lap 4
Even power (glucose)	0.96 ± 0.03	0.93 ± 0.03	0.91 ± 0.02	0.87 ± 0.02
Even power (placebo)	0.86 ± 0.02	0.83 ± 0.01	0.84 ± 0.0	0.82 ± 0.02
Road race simulation (glucose)	1.00 ± 0.06	0.97 ± 0.06	0.95 ± 0.06	0.93 ± 0.07
Road race simulation (placebo)	0.95 ± 0.05	0.98 ± 0.08	0.96 ± 0.06	0.93 ± 0.09

14.4.7 Performance data

14.4.7.1 Time

There was a significant differences between glucose and placebo trials (p=0.03), and between the even power and road race simulation trials (p=0.042) for time to complete the performance tests at the end of each trial. Table 14.15 shows the mean times for all conditions.

Table 14.15 performance times for all conditions. (mean ± S.D.).

Condition	'Fresh'	Even power (glucose trial)	Even power (placebo trial)	Road race simulation (glucose trial)	Road race simulation (placebo trial)
Time (mins)	4.70± 0.33	5.02 ±0.44	5.26 ±0.51	5.16± 0.36	5.49 ±0.34
Power (W)	469.5 ±56.2	440.2± 62.4	420.3± 57.4	427.3 ±52.0	400.2± 35.8

Figure 14.19 Performance times recorded following the four trials, and the 'fresh' performance trial.
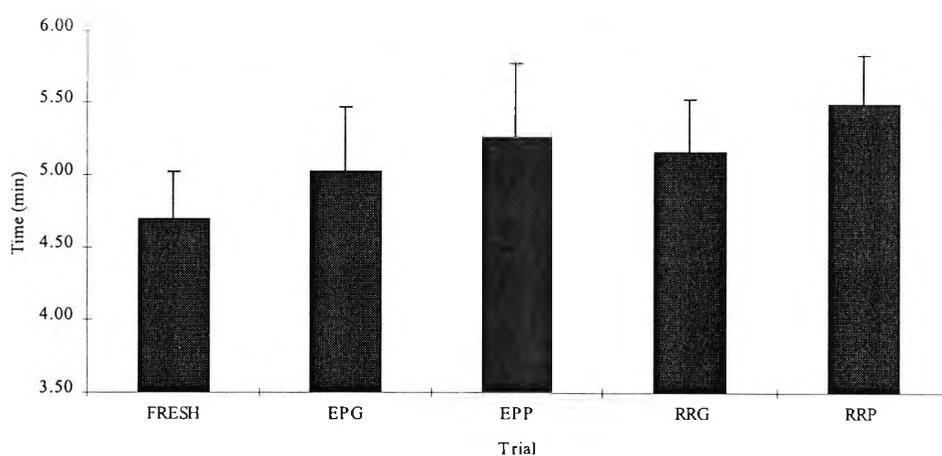
14.4.7.2 Power

Compared to the fresh performance trial in study 4 there was a significant drop in power output during the road race simulation trial versus the even power trial (-55.8 ± 25.8 vs -47.5 ± 23.4 watts, p=0.045), and a significant drop in the placebo compared to the glucose trials (-59.3 ±24.9 vs -35.7 ±14.75 watts, p=0.006).

Figure 14.20 Reductions in power output during the performance tests following all four trials – comparison to the 'fresh' performance trial.
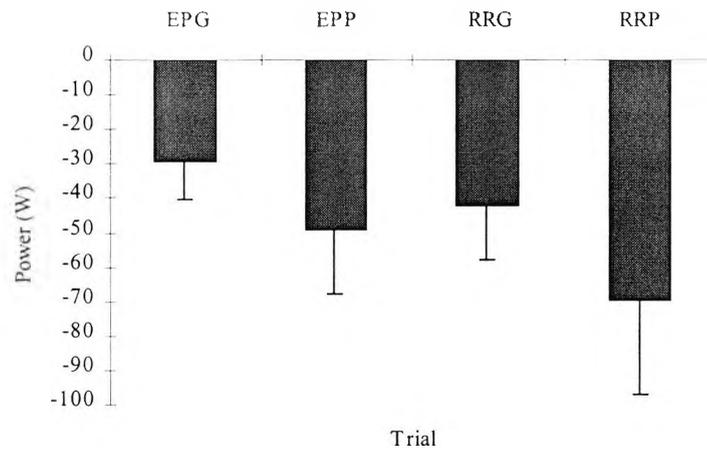
14.4.7.3 Performance Test Heart rate

No significant differences were observed between glucose and placebo (0.68) or between even power and road race simulation (p=0.44) for heart rate at 70% maximum minute power prior to the performance test. There were also no significant differences between heart rate values during the performance test, for even power versus simulated road race trials, (p=0.51), or for glucose versus placebo trials (p=0.73).

A significantly higher heart rate values were observed during the last minute of the road race simulation performance trials versus even power trial (p=0.000).

14.4.7.4 Post exercise BLac⁻ and blood glucose concentrations

Simulated road race or even power cycling had no effect on B[Lac⁻] response 3 minutes post performance test (8.9 ± 2.75 vs 10.75 ± 3.28 mmol.l⁻¹, p=0.207), however B[Lac⁻] was significantly elevated post glucose compared to placebo (11.11 ± 3.07 vs 8.55 ± 2.67 mmol.l⁻¹, p=0.02). Figure 14.20 shows the B[Lac⁻] during trials and post performance test.

Figure 14.21 Blood lactate concentrations during the 2 hour trials and post performance tests
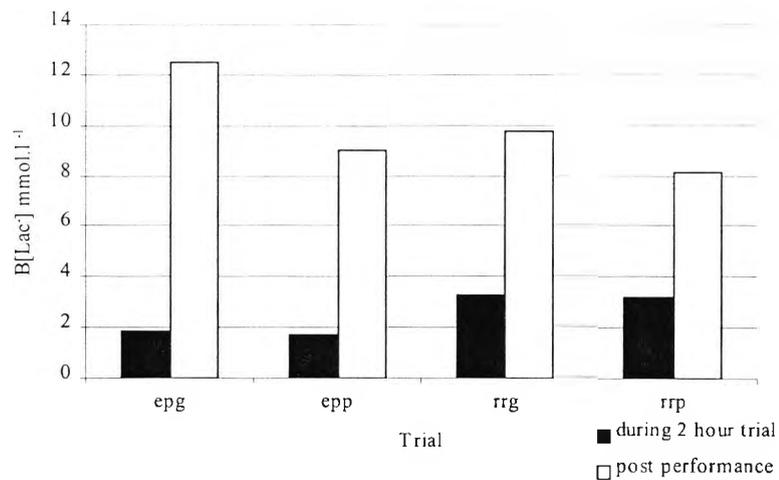
The same relationship was observed for blood glucose concentration measured 3 minutes post performance test, there was no significant difference between even power and road race (5.93 ± 0.95 vs 6.49 ± 1.48 mmol.l^{-1}, p=0.20), but a significant difference between glucose and placebo trials (6.92 ±1.39 vs 5.49 ± 0.46 mmol.l^{-1}, p=0.003).

14.4.7.5 One subject performance data

Gas analysis was performed on one subject during all of the performance tests. Mean power outputs for the performance tests are shown in table 14.16.

14.4.7.5.1 One subject performance respiratory data

Power output, and respiratory data for each trial is shown in table 14.16.

14.4.7.6 Performance Correlation analysis

Performance predictions from maximum minute test data are shown in table 14.17.

The highest correlation coefficients for predicting performance came from the other climbs (table 14.18). Appendix 4 shows the correlation coefficients between measured variables during the road race simulation and the even power trial correlated to reductions in performance relative to the individual rider's fresh performance test.

Table 14.16 Power, respiratory and economy data from the four performance trials (n=1). (mean ± S.D.).

	Fresh trial	Even power (glucose trial)	Even power (placebo trial)	Road race simulation (glucose trial)	Road race simulation (placebo trial)
Power (W)	475	438.4	401	425	411
$\dot{V}O_2$ ($l \cdot min^{-1}$)	5.34	4.76	5.05	4.68	4.69
% $\dot{V}O_{2max}$	98.5	87.8	93.2	86.3	86.5
Peak recorded $\dot{V}O_2$ $l \cdot min^{-1}$ (60 sec)	5.47	5.16	5.14	5.00	5.04
% $\dot{V}O_{2max}$ (of peak recorded values)	101%	95.2	94.8	92.3	93.0
$\dot{V}CO_2$ ($l \cdot min^{-1}$)	6.18	4.81	4.70	4.90	4.82
RER	1.16	1.01	0.93	1.04	1.03
RER $_{peak}$	1.18	1.11	0.97	1.14	1.09
Economy ($W \cdot l \cdot O_2^{-1}$)	89.0	92.1	79.4	90.9	87.6

Table 14.17 Correlation coefficients for time for the 2 km climb performance test for the 'fresh', post road race simulation, and post even power trials (n=6).

	Fresh trial	Even power (glucose trial)	Even power (placebo trial)	Road race simulation (glucose trial)	Road race simulation (placebo trial)
Maximum minute power (MMP) ($W \cdot min^{-1}$)	-0.90*	-0.94*	-0.81	-0.73	-0.57
$MMP \cdot kg^{-1}$	-0.70	-0.56	-0.54	-0.50	-0.69
$MMP \cdot kg^{0.67}$	-0.87*	-0.79	-0.72	-0.65	-0.71
$MMP \cdot kg^{0.79}$	-0.82*	-0.72	-0.66	-0.61	-0.72
$MMP \cdot kg^{0.33}$	-0.92*	-0.91*	-0.80	-0.72	-0.66
$\dot{V}O_2max$ $l \cdot min^{-1}$	-0.53	-0.59	-0.53	-0.58	-0.71
$\dot{V}O_2max$ $ml \cdot kg^{-1} \cdot min^{-1}$	-0.33	-0.27	-0.31	-0.40	-0.84*
$\dot{V}O_2max$ $ml \cdot kg^{0.67} \cdot min^{-1}$	-0.42	-0.41	-0.41	-0.49	-0.83*
$\dot{V}O_2max$ $ml \cdot kg^{0.79} \cdot min^{-1}$	-0.39	-0.36	-0.38	-0.46	-0.84*
$\dot{V}O_2max$ $ml \cdot kg^{0.33} \cdot min^{-1}$	-0.46	-0.50	-0.48	-0.53	-0.77

*denotes p<0.05

141

Table 14.18 Correlation coefficients for the 2 km performance test (time) for all trials (n=6).

	Fresh	Even power (glucose)	Even power (placebo)	Road race simulation (glucose)	Road race simulation (placebo)
Fresh	1.00				
Even power (g)	0.94*	1.00			
Even power (p)	0.89*	0.96*	1.00		
Road Race Simulation (g)	0.89*	0.78	0.75	1.00	
Road Race Simulation (p)	0.71	0.61	0.68	0.78	1.00

*denotes p<0.05

14.5 Discussion

14.5.1 Power output during the even power and road race simulation study

The motorised running treadmill was used during this study to elicit not only the required powers, but to elicit them uniformly, and instantly as would happen in the field. The only difference was that the subject was warned about the increase in load, rather than self-inducing the changes in work rates during the 2-hour road race simulation. A possible limitation to the simulation study is the uniform nature of the increases in power output that were elicited by placing weights on the pulley system. Previous research into repeated sprint activity by McCartney et al. (1986) and supported by Spriet et al. (1989) showed that the pattern of force application changed during repeated sprint type testing. The highest peak powers were attained during the first 2 seconds initially, however during subsequent 30 second Wingate cycling tests, peak power was achieved later during the 10th second of the test. During the road race simulation protocol, when the weights were dropped on the basket for the 12 sprints per lap, instantaneous work was required to prevent the subject being pulled from the treadmill (i.e. the extra power was required within 1 second). The implications of this are discussed later in this chapter.

The motorised treadmill also required subjects to balance and handle their bicycles, and allowed riders to ride out of the saddle, change posture at their own volition, allowing for recruitment changes and easing tired muscles, and saddle soreness if required. This is what would happen in the field, and therefore improves the specificity of the exercise testing procedures. The last, and possibly most beneficial reason for using the Woodway treadmill is to allow for the simulation of hills, with the associated changes in respiratory, and efficiency factors. If the motor recruitment patterns change with alteration in posture during climbing, then, treadmill testing again increases the validity of assessment of these athletes.

Figures 14.1 and 14.2 show the SRM traces from the even power and the road race simulation study. During the even power trials, the treadmill gradient and speed altered and followed the same gradient pattern as the road race simulation trial. This was due to the differences observed from previous hill climb studies and the

142

effects of gradient on performance would not influence results, therefore, any possible differences observed would be due to the intermittent versus the even power protocol. Calculating the mean power for the trials, the power output for the even power trials was 61.4% and 60.6% of maximum minute power for the glucose and placebo trials respectively. During the road race trials, the mean work rates again incorporating the last two phases was 62.8% and 63.0% of maximum minute power during the glucose and placebo rides. This compares to an estimated intensity of 58% for the Palmer *et al.*, (1997) study. The variation in the actual road race simulation (1.35%) and the even power (1.86%) during the study were higher than the pilot study data. This variation is still lower than the manufacturer claims of the system (2%). The increased variation is likely to be due to the longer blood sampling required during the actual study, as NH_3 and urea assays required larger blood samples.

14.5.1.1 Even power trace

The even power trace shows some variation in power output during the two-hour simulation. The blood sampling required subjects to ease off and be held (therefore zero power output) which caused most of the deviation from the mean power recorded. The other 'noise' appears even during steady state ergometry testing with the SRM (Balmer and Coleman unpublished observations), and is far less stochastic than an attempted even trace recorded in the field.

14.5.1.2 Road race power trace

The road race simulation trace is shown in graph represents the work rates achieved during different periods of the road race simulation. Power outputs of 150% of maximum minute power were achieved. One limitation of this study was that it did not elicit work rates higher than 150% of maximum minute power output for safety reasons on the motorised running treadmill. In the recorded traces from competition, there were short periods in excess of 150% MMP, however even attaining 150% MMP was arguably more realistic than previous studies that have attempted to simulate road cycling. These studies had their power outputs ranging from 30-90 % of maximum minute power (Brouns *et al.*, 1990. Palmer *et al.*, 1997), which is a much narrower distribution than the data in this study. The distribution of the data somewhat matches the pattern recorded in the field, with what would be an almost normal distribution if it were not for the data recorded at zero watts of power.

14.5.2 Blood parameters during simulated cycling

14.5.2.1 BLac⁻ response

The mean B[Lac⁻] values observed during the road race compared to the even power trials during all four laps of the trials were 3.25 vs 1.78 mmol.l⁻¹. This compares to 2.51 mmol.l⁻¹ at a steady state intensity 60% MMP in the study presented in chapter 9. The B[Lac⁻] are indicative of metabolic adjustments required to maintain ATP production (Spurway, 1982). Essen *et al.* (1977) reported that only slow twitch muscle fibres were recruited during a steady state trial for 60 minutes at 55% $\dot{V}O_2$max, whereas both slow twitch and fast twitch muscle fibres were recruited during an intermittent trial. The exercise intensities in this study were

similar (60-65% VO₂max), differences in B[Lac⁻] could be attributed to the different metabolic capabilities of muscle fibre. The recruitment of fast twitch muscle fibre to generate the instant forces applied at 150% of maximum minute power for the session of five intervals, plus the small changes in exercise intensity throughout the protocol that would require anaerobic energy production as adjustments in aerobic metabolism take place. The production of Lac⁻ from glycogen would have resulted in increased CHO use during the road race trial, during this present intermittent simulation study the only marker of CHO utilisation recorded was the respiratory exchange ratio, and higher values were observed during the road race simulation compared to the even power trials as speculated by Palmer *et al.* (1997).

The B[Lac⁻] values during the road race simulation do however appear low bearing in mind the high powers achieved during the interval sessions of this protocol. It is possible that these cyclists have the ability to either produce these powers, i) without the production of Lac⁻, ii) without the accumulation of B[Lac⁻].

During repeated Wingate testing, (4 x 30 seconds) McCartney *et al.* (1986) reported no detectable changes in muscle Lac⁻ production, or glycogen depletion during the latter tests. There were reductions in force however, and they concluded that increased slow twitch muscle fibre recruitment would have reduced the force, the Lac⁻ accumulation, and glycogen depletion. Spreit *et al.* (1989) reported that during a third Wingate test (3 out of 3), there was 82% of the work achieved from the previous test, yet only 32% of energy that was derived from glycogenolysis during the Wingate number 2 occurred during the third trial. They concluded that H⁺ concentrations inhibited phosphorylase b to a transformation, thereby reducing glycogenolysis. The reductions in work were then offset by the recruitment of more slow twitch muscle fibre enabling higher ATP yield through oxidative metabolism. Essen *et al.* (1977) also reported lipid metabolism during repeated high intensity bursts of exercise. The studies by McCartney *et al.* (1986), and Spriet *et al.* (1989) worked subjects maximally during the repeated bouts of exercise, Essen *et al.* (1977) worked subjects for 60 minutes at the power output required to achieve V̇O₂max for 15 seconds with 15 seconds recovery. The present study did not maximally work subjects, therefore the work intervals were not as intense as the McCartney *et al.* (1986) or the Spriet *et al.*(1989) studies so it would be less likely that H⁺ accumulation would inhibit glycogenolysis. However, the longer recovery to work ratio in this current study compared to the Essen *et al.* (1977) study, and the lower intensity than the McCartney *et al.* (1986) and the Spriet *et al.* (1989) studies might lead to enhanced slow twitch fibre recruitment and possible lipid metabolism to cover the ATP demand. Muscle biopsy sampling could identify the likely substrate use during future studies that attempt to simulate road race cycling.

McCartney *et al.* (1986) reported that this pattern of possible slow twitch fibre recruitment elicited changes in the pattern of peak force production with initial (supposedly fast twitch generated) sprints recording peak powers during the first 1-2 seconds, and the second two sprints (supposedly slow twitch fibre generated) taking 9-10 seconds to generate peak power. During the anaerobic power testing of the hill climb study (chapter 10), mean time to peak power was 8.8 seconds for the competitive cyclists, possibly fitting the

theory that slow twitch muscle is predominantly developed in road cyclists, and is recruited during Wingate type testing. However without further investigation using the muscle biopsy analysis technique this remains speculative. The recruitment patterns during intense exercise in endurance athletes to our knowledge has not been documented. In cycling with high forces produced yet the typical morphology being one of predominant slow twitch fibre it is tempting to speculate that these athletes can produce these high powers through slow twitch fibre.

During the road race simulation protocol the riders needed to generate peak force instantly to prevent themselves being dragged off the treadmill. This possibly required the regeneration of ATP through 'alactic' mechanisms. Phosphocreatine could have covered the energy cost for some of the sprints involved, and replenishment achieved during the recovery periods (Hamilton *et al.,* 1991). Therefore this could reduce the reliance upon glycolysis and the LDH reaction, resulting in lower Lac⁻ production, and lower Lac⁻ appearance in the blood stream. From the SRM race traces it is somewhat difficult to ascertain if there are any changes in the rate at which an athlete can generate peak power, however it is interesting to note the late peak powers recorded during the Wingate test for the hill climb study (9 seconds) resulting in high levels of BLac⁻ accumulation.

Testing designed to evaluate anaerobic capacity (Wingate) has previously correlated to aerobic sustained performance, Tanaka *et al.* (1993) reported mean and peak power indices relative to body mass correlated with time during a hill climb task lasting a mean duration of 17 minutes 37 seconds. Likewise the hill climb study (chapter 11) found anaerobic power measures correlating to sustained performance lasting 16.31 minutes. It could be implied therefore that the work rates sustained were achieved through highly oxidative muscle fibre, reducing the BLac⁻ responses during this type of effort.

Withers *et al.* (1991) indicated reliance upon aerobic sources during intense exercise, and, speculated that a high $\dot{V}O_2$max, and fast oxygen kinetics would reduce muscle glycogen use, and the Lac⁻ yield from a sprint.

Increased utilisation of slow twitch muscle fibre would also enable the removal and re-conversion of Lac⁻ to pyruvate during periods of recovery. This process could also be activated in the liver, via the Cori cycle during periods of reduced exercise intensity, thereby reducing the B[Lac⁻] despite the high work rate intervals during the simulation study.

Essen *et al.* (1977) reported production of 1.5 mmol.l⁻¹ throughout an intermittent exercise protocol compared to a continuous trial. During the continuous trial, where, after that there was no net production or removal from the working limbs (except for the first five minutes of exercise). Koziris and Montgomery (1991), also reported variation in the Lac⁻ response when comparing intermittent and continuous trials. They indicated that leg blood flow could alter the response to intermittent and continuous tests of anaerobic capacity. Koziris and Montgomery (1991) analysed the response to three tests; an all out continuous test (90 seconds in duration), an all out intermittent test (6 x 15 seconds all out with 15 seconds recovery), and a continuous paced trial at the mean power of the continuous all out trial. They hypothesised that intermittent

exercise would result in higher B[Lac⁻] compared to the other two trials due to the continuous trials requiring sustained muscular contractions which would restrict blood flow through the working muscles. They speculated that this would result in initially higher intramuscular [Lac⁻], elevated H⁺ which would then inhibit glycolysis, with the result of reducing anaerobic contribution during continuous trials. Koziris and Montgomery, (1991) indicated that the recovery periods during an intermittent bout would allow for the wash out of lactate into the blood therefore more energy would be derived through anaerobic energy sources without the inhibition of glycolysis. This discussion was based upon high B[Lac⁻] (12.4-14.2 mmol.l⁻¹), and the authors reported significantly higher B[Lac⁻] during both all out trials compared to the continuous paced trial. Occlusion during low intensity exercise (the continuous trial at 60% MMP in this study) would not have resulted in limited blood flow due to occlusion, and therefore would not have initiated the mechanisms associated with reductions in glycolysis, as this would have led to premature fatigue. However, due to the intense nature of the work interval bouts during the road race simulation trials, and the reductions in blood flow that occur during intense muscle contractions, this could enhance the reliance upon anaerobic metabolism during these periods, with higher B[Lac⁻] following the extended recovery periods after each work interval. This would result in higher B[Lac⁻] during the road race simulation compared to the even power trial.

Following the glucose trials, power output was significantly higher (6%) during the 2 km performance tests. The post trial B[Lac⁻] was also significantly higher following the glucose trials (27%). This has previously been reported following exercise lasting approximately 1 minute in duration by Jacobs (1981). She reported a drop in muscle force during intense contractions following a period of prolonged exercise, and, like the present study this was accompanied by far greater metabolic changes. Jacobs (1981) reported that muscle lactate levels were reduced, and speculated that due to prolonged exercise, glycogen depletion would decrease the available substrate available for enzymatic reactions; in other words prolonged exercise would reduce the flux through glycolysis. Jacobs (1981) also indicated that as the performance changes were not of the same magnitude as the fall in the lactate concentrations, and that lipid utilisation may have contributed to offset some of the deficit in energy demand. Lipid utilisation has been reported during high intensity exercise (Essen *et al.*, 1977).

Chapter 6 highlighted the limited data on the anaerobic responses of competitive cyclists, further research should firstly focus on the importance of this component to road cyclists. This current study simulated road race cycling with a relative exercise intensity derived from a MMP test. The analysis of the responses of riders with similar MMP and different anaerobic power/capacity capabilities would highlight the influence of enhanced anaerobic capabilities on road race performance.

The second direction for future research in this area should focus the laboratory assessment of anaerobic power of road cyclists. The data presented from chapter 9 indicates that riders in the field can produce higher peak powers than when they undergo Wingate testing in the laboratory. With the presentation of

highly intermittent data from actual races, it has become apparent that we may not necessarily be able to measure the anaerobic power of trained cyclists in the laboratory using current protocols.

A further avenue for investigation centres around the discussion within this chapter of the mechanisms capable of eliciting the high power outputs in athletes that have been shown to have predominantly slow twitch muscle fibre characteristics. The relative contribution of energy systems to repeated sprinting and the fibre recruitment patterns could be determined if the muscle biopsy studies could be conducted following intermittent exercise.

14.5.2.2 Blood glucose concentrations

There were no significant differences between road race simulation and the even power trials for blood glucose concentrations (4.53 vs 4.61 mmol.l^{-1}). This supports the findings of Vanhelder et al. (1985) who reported no difference in blood glucose concentrations during intermittent versus a continuous trial.

The higher RER values during the road race simulation trials indicate higher rates of carbohydrate utilisation and B[Lac$^-$] during this trial. The blood glucose concentrations significantly reduced during the trials between laps 1-4, this is a similar response to the reductions in RER values for the same time periods. The similarity in blood glucose concentrations (between even power and simulated cycling trials) implies that either the reliance upon this blood borne substrate was the same between trials (therefore greater reliance upon muscle glycogen during the road race trial), or, the supply and maintenance utilised greater liver glycogen and the process of gluconeogenisis to maintain blood glucose concentrations during the road race simulation trial, compared to the even power trial. Again further study using the muscle biopsy technique would give further detail in the investigation of substrate utilisation during this form of exercise. Likewise instigating a protocol to deplete muscle glycogen (induced via dietary and exercise manipulation), followed by an intermittent or continuous trial might indicate the relative ability of blood glucose to sustain intensity during these two different forms of exercise.

14.5.2.3 NH$_3$ response to exercise.

Mean post warm up values for NH$_3$ and urea were higher than resting values reported by Brouns et al. (1990), however there were no significant differences between the trials for the post warm up value. There were also no significant increases in plasma NH$_3$ over time during the trials.

This data is in contrast to research published by Lemon and Mullin (1980). They reported that reduced substrate availability during endurance exercise with limited substrate supply induced protein breakdown, with an associated NH$_3$ accumulation. During a carbohydrate depleted trial increased dependence upon protein was observed, indicating utilisation to cover the energy demands of the task. Palmer et al. (1997) concluded that possible reductions in performance after intermittent activity were the result of an increased utilisation of muscle glycogen. If this were the case during this study an increased contribution of protein breakdown to cover the energy requirement in theory would have been recorded. The road race simulation trials, and the even power trials did not induce conditions of hypoglycaemia as might be expected under

conditions of severe muscle glycogen depletion. Therefore it is possible that this is the reason why significant increases of plasma NH_3 accumulation were not observed during this study.

Another possible explanation is the coupling of $B[Lac^-]$ and NH_3 kinetics. Correlation analysis revealed that during the road race simulation trials, the absolute plasma NH_3 and $B[Lac^-]$ concentrations correlated significantly (r=0.77). During the even power trials, the absolute plasma NH_3 concentrations did not significantly correlate with $B[Lac^-]$, (r=0.29). These values are for the mean of each subject during each condition. This therefore suggests that in the road race simulation trial the Lac^- and ammonia kinetics are matched, whereas during the even power trial they were not. Lutoslowska (1996), reported similar correlation coefficient for $B[Lac^-]$ and plasma $[NH_3]$ for intense 'endurance' exercise (r=0.66). This could also be linked to the findings of Pages *et al.* (1994) who reported coupled kinetics between Lac^- and NH_3 during high intensity exercise, however, during endurance exercise the kinetics were uncoupled. It appears from the road race simulation results and previous literature that during high intensity exercise the production of NH_3 and $B[Lac^-]$ are linked thus it is likely that the PNC is involved in these processes. It also appears that during steady state exercise the NH_3 and $BLac^-$ responses are not linked this would be due to the oxidisation of branched chain amino acids during steady state exercise. Although amino acid metabolism can increase under conditions of increased acidosis, highly oxidative muscle has a greater capacity to oxidise branched chain amino acids (Graham *et al.,* 1995). Therefore under conditions of slow twitch muscle fibre recruitment amino acid metabolism may occur, with minimal $BLac^-$ accumulation, therefore resulting in uncoupled characteristics between the two metabolites.

Even though there was no significant rise in the accumulation of plasma NH_3, it could be that the purine nucleotide cycle was 'cycling' during intermittent exercise. Breakdown of AMP with the production of IMP occurs under conditions of intense muscle contraction. NH_3 is produced during this process, however, if aspartate is available IMP can be converted into adenylosuccinate, and back into AMP. However, during these conditions of anaerobic exercise, and aspartate (GTP unavailable) is not available, IMP is further degraded into hypoxanthine and uric acid. It is likely therefore that under conditions of intense intermittent exercise NH_3 is released, likewise some Lac^- is produced, hence the coupled kinetics during the road race simulation trials.

The pattern of NH_3 formation during all trials is possibly another indication of substrate not being under conditions of depletion during the trials. The less toxic amino carriers alanine and glutamine both transport the nitrogen compound formed within the muscle cell to the liver for deamination. These two compounds are formed within the cell to minimise toxicity. The formation of these two nitrogen 'carriers' however requires ATP (glutamine) and pyruvate (alanine). It has been speculated that during endurance exercise where substrate is limited there will be increases in NH_3 due to reduced substrate availability to fuel the formation of alanine and glutamine. As there was no significant rise in plasma $[NH_3]$ during any trial, (which has been reported as a consequence of a reduction in substrate availability, Haralambie and Berg,

1976), it is another indication that the subjects were not totally glycogen depleted during the four 'laps' of the protocol.

14.5.2.4 Plasma urea response to exercise

The post warm up plasma urea concentrations were not significantly different between trials, however the post warm up values were higher than had been previously reported by Rauwbottom *et al.* (1997). There was a mean increase of 12.3% in plasma urea during the even power trials, compared to a mean rise of 3.4% in the road race trials. These values are lower than has been previously reported by Cerny (1975) who reported a significant 20% increase during steady state exercise at similar intensity (60-65% $\dot{V}O_2$max) for one hour in duration.

Decombaz (1979) also reported significant rises in plasma urea (44%) during endurance (70-90 kilometres) cross-country skiing. Lemon *et al.* (1989) reported a 252% increase in plasma urea during a 62 minute swim at approximately 70% of $\dot{V}O_2$max, they calculated that changes in blood flow to the liver could only account for 34% of the rise so the rest of the plasma accumulation was due to increased protein breakdown. Haralambie and Berg (1976), monitored serum urea levels of athletes throughout a variety of endurance activities, and concluded that serum urea levels increased linearly with exercise duration after an initial plateau for 60-70 minutes. However, Rattu *et al.* (1996) reported reductions in plasma urea concentrations during a 25 mile laboratory simulated cycling time trial performance test, with subjects ingesting glucose polymer during one trial and a placebo substance during the other. The drop during the Rattu *et al.* (1996) study was approximately 10-15% from the resting value, between the two trials. There was a greater reduction during the glucose trial compared to the placebo trial during the Rattu *et al.* (1996) study, and they concluded that there was less reliance upon amino acids for energy production compared to the placebo trial. There were no significant differences between the glucose and placebo trials during this study however. Haralambie and Berg (1976) concluded from their findings that the rise in serum urea was indicative of muscle glycogen levels becoming depleted. Studies on ultra endurance tasks have concluded that the rises in serum urea observed during an iron man triathlon (Farber *et al.*, 1987), and during a 24-hour run (Irving *et al.*, 1989) were due to inadequate carbohydrate sources, resulting in increased protein utilisation, during this type of endurance exercise. Therefore again, it is possible that the simulated road race did not deplete muscle glycogen to extreme levels whereby increased rates of protein utilisation would induce significant increases in serum urea.

The pattern of increased serum urea concentrations typically occurs during the hours, and possibly the days after exercise has ceased. Previous authors have speculated that due to reduced sweat rates at the cessation of exercise, serum urea increases due to this being a major removal mechanism for urea. Lemon and Mullin (1980) reported that with increased exercise intensities, there would be increased sweat rates, leading to the increased removal of urea through the sweating mechanism, thereby reducing the concentrations of serum urea. This possibly explains the findings by Rattu *et al.* (1996) who reported reduced levels of serum

urea during the 25-mile laboratory time trial. Previous studies by Coyle *et al.* (1991) reported that cyclists can maintain exercise intensity in excess of 80% during this type of task indicating that sweat rates are likely to be high to maintain core temperature during exercise that is likely to last approximately one hour.

The findings of no significant increases in urea are possibly linked to the similar responses in NH_3, however, the breakdown and utilisation of protein, and its pathway to excretion are implicated in performance outcomes of these athletes discussed later in this chapter.

14.5.3 Cardiovascular responses to simulated road and even power cycling

14.5.3.1 Oxygen consumption and carbon dioxide production

The 'one subject' data shows wide variations in $\dot{V}O_2$ throughout the simulation trial, however, when the matched samples during each trial are compared there are no significant differences between the $\dot{V}O_2$ values (absolute or relative). It appears therefore that adjustments to repay any oxygen debt incurred during the interval sessions is repaid quickly and efficiently by these athletes, and hence within a couple of minutes of an interval session the $\dot{V}O_2$ has returned to a value reflecting the current exercise intensity. Due to the similarities in $\dot{V}O_2$, there were no significant differences for gross mechanical efficiency, or economy during matched periods of work. The end of lap sample showed higher $\dot{V}CO_2$ values during the post sprint recovery period, it appears therefore that possibly the 'longer term' (2-5 minutes) compensation for an intense interval session is elevated $\dot{V}CO_2$ production compared to increased $\dot{V}O_2$. As the B[Lac⁻] also appear relatively low compared to what might be expected post repeat sprint activity, carbon dioxide excretion appears to be a major central compensatory mechanism post intense intermittent sprinting. Further research into the recovery patterns from intense exercise on these athletes is warranted.

14.5.3.2 Comparative cardio respiratory response to Kingcycle and Treadmill cycling

Table 14.19 below shows a summary of results from chapters 9 (physiological responses of competitive road race cyclists), 12 (effect of gradient on cardio respiratory measures during submaximal cycling) and 14 (effect of glucose polymer supplementation on simulated road race and even power cycling). The data selected is for steady state rides at a relative intensity of 60% MMP. The results are possibly more comparable between chapter 9 (physiological responses of road cyclists) and chapter 12 (effect of gradient on cardio respiratory measures during submaximal cycling) due to the short nature of both protocols (10 and 6 minutes respectively) whereas chapter 14 was the mean data over the sampling periods during the 2-hour protocol.

In comparing the two different ergometers, the results show similarities for B[Lac⁻], and % maximum heart rate. However $\dot{V}O_2$ data shows a 9% difference in relative exercise intensity, when comparing the data in chapters 8 and 11, and thus affects the other figures (economy and efficiency calculations) associated with the $\dot{V}O_2$ values. Kenny *et al.* (1995) reported higher average crank torque during treadmill as opposed to track cycling. This they concluded would elevate the demands of riding on a treadmill compared to the track. With lower demands on the treadmill compared to the Kingcycle ($\dot{V}O_2$), it may be that for the same

150

relative power output, the treadmill still requires a higher $\dot{V}O_2$ than the track, and the Kingcycle requires a higher $\dot{V}O_2$ than the treadmill. Further research is warranted into the reasons behind the changes in $\dot{V}O_2$ between Kingcycle and treadmill bicycling, and possibly more importantly, how these compare to the cycling demands on the road.

Table 14.19 comparison of treadmill and Kingcycle cycling at 60% MMP

	Chapter 9	Chapter 12	Chapter 14
B[Lac] mmol.l^{-1}	2.51		1.78
% Maximum Heart Rate	76.4%	77.2%	78%
% $\dot{V}O_2$max	68%	59%	62%
$\dot{V}CO_2$ (l·min^{-1})	2.94	2.94	2.88
RER	0.86	0.94	0.89
Gross Mechanical Efficiency (%)	19.54	23.47	22.82
Economy (watts.l.O$_2$)	66.07	80.84	84.25

14.5.4 Post exercise performance

The 2 km performance test was repeated on 2-3 occasions during familiarisation prior to the testing procedures commencing. Previous research by Foster *et al.* (1993) reported faster trials during an even paced strategy during a study that included a range of pacing strategies. The findings were not statistically significant, however, in terms of ensuring differences were due to true physiological considerations (as opposed to pacing strategy) optimal pacing was encouraged, and subjects were instructed that their fastest ride would be their most even paced.

14.5.4.2 Even power versus road race simulated cycling (performance tests)

The mean exercise intensity ridden for the final 2 km performance test was above 400 watts in all trials (table 14.15).

The performance test post even power cycling was 3.7% faster (p<0.05) than post road race simulated cycling. One possible explanation for the relatively better performance in the even power compared to the road race simulation trials is the recruitment pattern of muscle fibres. Essen *et al.* (1977) reported slow twitch recruitment only in a steady state trial (55% $\dot{V}O_2$max), compared to slow twitch and fast twitch recruitment during an intermittent trial. According to Bergstrom and Hultman (1967), resting fibres cannot donate glycogen to other active fibres, in which case the utilisation of fast twitch fibres to work at higher intensities during the even power trials may explain the results. However the post performance B[Lac] were not significantly different between trials, therefore it might be premature to make these assumptions.

There were no significant differences for mean heart rates between even power and simulated road race trials. Despite worse performance post road race simulation trials, heart rate during the last minute of the test was significantly higher during this trial (for lower power) compared to the even power trial. This again indicates problems when comparing to what was observed during the road race data collection from actual races. For a given power output, (derived from the maximum minute test data) heart rate was significantly elevated on the road. This occurred for only the final minute of the trials lasting nearly two hours and ten minutes, compared to the actual road races where from 20% of race distance covered, there was an elevated heart rate response compared to laboratory predicted data.

14.5.4.3 Road race trial

Following the 2-hour road race trial, performance power was lower during the 2 km performance test relative to fresh performance (mean - 55.8 watts) over the 2 km trial, this was a significantly greater drop than the even power trial (p<0.05).

Table 14.17 shows the correlations between MMP test data and the five 2 km performance tests (4 trials and the rested fresh trial). Following the road race simulation glucose trial none of the MMP and $\dot{V}O_2$ variables correlated significantly to performance (time), with scaled $\dot{V}O_2$max correlating to performance in the placebo trial.

This compares to significant correlations for the Fresh performance to absolute MMP values and relative MMP values. This indicates that reductions in performance were not the same for each subject.

Appendix 4 shows the correlations for the performance test and physiological responses during the road race protocol.

In this study higher post performance B[Lac⁻] were indicative of improved relative performance during the 2 km climb. This again possibly indicates a greater fast twitch fibre utilisation resulted in better performance and increased Lac⁻ production, if fast twitch fibre was already fatigued (glycogen depleted), there would possibly be an inability to recruit them, and produce the desired force during the performance test resulting in decreased BLac⁻ levels.

Elevated relative plasma urea and NH_3 concentrations during the road race trials were also related to poorer relative performance (r=0.82 and r=0.81 respectively). Previous research by Brouns *et al.*, (1990) reported a wide range of NH_3 responses to endurance exercise. In theory elevated glycogen use would result in the use of protein sources as an alternative fuel, likewise PNC activity during anaerobic bursts resulting in the accumulation of NH_3, and the subsequent elevations in urea concentrations. Thus as previously indicated by Brouns *et al.*, (1990) these mechanisms are implicated in fatigue during endurance based intermittent activity.

The mean respiratory measures taken during the road race simulation trials failed to produce any significant correlates of performance. However the upward drift in $\dot{V}O_2$ from lap 1 to lap 4 correlated positively to

improved relative performance at the end of the road race trials. During even power exercise it has been speculated that the oxygen slow component is due to increased recruitment of fast twitch fibres resulting in inefficiency during the latter stages of exercise (Xu and Rhodes, 1999) It is likely however that fast twitch fibres would be recruited from the onset of exercise during the highly intensive sprints. The ability to utilise or switch to greater fat metabolism during exercise of a high intensity nature could be indicative of greater slow twitch muscle fibre recruitment as the simulation progressed. This might also reduce the recruitment of fast twitch fibres, that might then be required to sustain the high powers reported in the performance tests. Due to the reflection of anaerobic metabolism in the RER measurements taken during intermittent exercise, other methods of substrate utilisation analysis need to be undertaken to investigate these findings further.

To summarise, it appears that to produce a high relative performance following a road race simulation trial, riders need to minimise plasma concentrations of NH_3 and urea, and following the performance test have high B[Lac⁻] following the trial. These factors in the literature have been linked to the ability to minimise carbohydrate use during exercise, reducing NH_3 and urea formation, and having muscle glycogen available to produce lactate, thus producing higher work rates with a larger anaerobic component in the latter stages of road race cycling.

14.5.4.4 Even Power trial

Relative performance decrements were also seen following the 2-hour even power trials compared to the fresh performance tests, a reduction in power of 47.5 watts.

Interestingly the respiratory responses during even power trial showed the opposite responses compared to what has been previously reported in the literature. Increased relative $\dot{V}O_2$, $\dot{V}CO_2$, and RER correlated to improved performance during the even power performance trials, however only the RER value attained statistical significance in this study with small subject numbers. In the calculation of efficiency from the even power data those riders with high efficiencies performed worse than those with lower efficiency values relative to their previous fresh performance trials. This is in direct contrast to the work of Passfield *et al.* (1995) who reported smaller reductions in MMP following a 3-hour even power trial. In this current study the subjects were working at a higher exercise intensity compared to the Passfield *et al.* (1995) study. Like the Passfield study the early measures (lap 1) gave the indication of these results (r=-0.65 and r=-0.86) during the matched work rate phase of the protocol. The final samples (end of lap sample on lap 4) correlated strongly to performance (r=-0.91 and r=-0.88) with lower RER values again indicating poorer relative performance. This during the later stages of exercise may link to adequate CHO availability. Appendix 4 shows the lap-by-lap correlation data for chapter 14. Coyle *et al.* (1991) reported that glucose availability late in exercise enabled subjects to sustain higher power outputs during performance testing, this appears to be the case and is reflected in the RER values in the latter stages of the even power trial.

14.5.4.5 One subject performance data

Figures 14.25, 14.26 and 14.27 show the respiratory responses of the one individual who completed the majority of the trials with constant gas analysis. Table 14.17 shows the respiratory responses during the performance trial for this individual. Using the five performance trials (power) and comparing them to the respiratory data, relative performance correlated to the ability to achieve high $\dot{V}O_2$ (r=0.58), to produce $\dot{V}CO_2$ (r=0.90) and produce high RER values (r=0.90). Therefore, reductions in performance seen during these four trials are possibly linked to the ability to achieve these values. The fresh performance test elicited a mean $\dot{V}O_2$ of 5.34 $1\,min^{-1}$ (98.5% of $\dot{V}O_2$max), with reductions down to 86.5% $\dot{V}O_2$max during the road race placebo performance trial. $\dot{V}CO_2$ values during the post trial performance rides equated to 76-79% of the fresh performance value, likewise mean RER data ranged between 80-90% of what was achieved during the fresh performance test. These figures are similar to the percentage of fresh performance (power) sustained by this rider (92% even power (glucose trial), 84% even power (placebo trial), 89% road race simulation trial (glucose trial), 86% road race simulation (placebo trial).

Substrate, and anaerobic metabolism appear important, likewise the ability to achieve a high percentage of $\dot{V}O_2$max at the end of the trials. During the road race simulation trials the respiratory data shows wide fluctuations (high standard deviations) during the trial for $\dot{V}O_2$, $\dot{V}CO_2$, and RER. This may influence the ability to achieve a high $\dot{V}O_2$ value at the end of competition, and possibly indicates that central fatigue could be implicated in the reductions in performance at the end of the 2-hour trials. The economy data from these trials shows that this individual had an economy of 90 $W.l.O_2$, during all but the even power placebo trial (79 $W.l.O_2$). This could suggest that the anaerobic contribution to the production of power during 3 out of the 4 trials was very similar, i.e. if greater energy production had come from anaerobic sources with the same $\dot{V}O_2$ a greater economy would be the result. The values for economy and B[Lac⁻] for this individual showed a positive correlation (r=0.88).

These findings cannot differentiate between central and peripheral factors influencing performance. Is it the ability to produce the higher relative power outputs that require the higher respiratory values? or is it the inability to achieve these respiratory values that limits the ability to produce the power outputs at this stage of the trials?. As the $\dot{V}CO_2$ and RER values are stronger correlates to the power produced by this one subject it may again suggest that the availability of fuel will be a determining factor associated with anaerobic energy release during intensive exercise lasting approximately 5 minutes.

14.5.5 The effect of glucose polymer ingestion on simulated cycling

The absolute values for blood glucose concentration during the forth lap of all trials indicated that no trial induced hypoglycaemia. The 120 minute (plus performance test) length of these trials was possibly not

long enough to reduce blood glucose concentrations to the extent seen previously by Flynn *et al.* (1987), who reported at the concentration of 3.5 mmol.l[-1] exercise would be impeded with fatigue approaching

Confirming the findings of many other studies, the supplementation of glucose polymer improved performance. Compared to the fresh 2 km performance trial all trials had a reduction in power, however glucose polymer supplementation during even power trial was reduced less than when compared to the placebo trial. Performance following the glucose-supplemented trial was enhanced 5.3% (time) compared to the placebo trial. The same pattern was observed for the simulated road race trials with glucose polymer significantly improving results (6.01%). There was no significant difference between the effect of glucose on even power compared to simulated road race cycling. Previous studies have shown improvements with glucose supplementation during steady state cycling exercise at varied intensities (50-85% $\dot{V}O_2$max Massicote *et al.*, 1985, Coggan and Coyle, 1988) and during exercise as short as 1 hour in duration (Below *et al.*, 1995). Coggan and Coyle (1989) reported performance improvements following CHO ingestion of 21% at an exercise intensity of 70% $\dot{V}O_2$max (~5% higher than during this study) on trained subjects who had previously fasted. Their protocol consisted of feeding subjects 30 minutes before the anticipated time to fatigue (derived from previous tests), thereby 'eliminating' the possibility of the sparing of muscle glycogen. This indicates that the blood borne substrate can play a major role in the outcome of cycling events over a wide spectrum of times/distances. The findings from this study also indicate that there are performance improvements following CHO ingestion even when the subjects have only abstained from food for just two hours (as would occur with typical race preparation), which is more likely to occur in competing athletes than longer periods of fasting.

These improvements in performance were accompanied by a 14% higher blood glucose concentration in the glucose trials, with significant differences reached on 'lap' 2, with statistical significance increasing during the two remaining 'laps'.

During intermittent exercise improved performance with glucose supplementation has previously been observed by Yaspelkis *et al.* (1993) and by Hargreaves *et al.* (1984). They reported that spared muscle glycogen during the carbohydrate feeding trials improved performance during these trials. They concluded that possible muscle glycogen resynthesis occurred during the periods of reduced energy requirement, and the availability of exogenous substrate repleted muscle glycogen. Similarities in blood glucose between the road race and even power trials suggests that if muscle glycogen sparing / resynthesis occurred then either the precursor was not glucose (i.e. possibly BLac[-]) or alternatively, liver glucose production maintained blood glucose. Other studies have previously reported that during steady state exercise performance improvements occur without sparing of muscle glycogen (Coggan and Coyle, 1989).

The manifestation in performance improvements during this current study could be indicated by the blood samples taken during the latter stages of exercise between the two trials. Despite significantly elevated blood glucose concentrations during the glucose supplemented trials, significant differences in the RER

were not apparent during the protocols until 'lap' four. This possibly indicates that rates of CHO use were not significantly different for these athletes despite greater carbohydrate availability during the early laps. Alternatively the process of supplying blood glucose and maintaining euglycaemia were not taxed until the last 30 minutes of the trials. Therefore the CHO supplemented trial enabled the cyclists to utilise more glucose, for aerobic and anaerobic energy release, and therefore maintain higher work rates at the work intensities greater than 85%$\dot{V}O_2$max. The performance improvements during the glucose trials compared to the placebo trials could possibly be explained by the post performance blood samples.

Following the glucose trials, power output was significantly higher (6%) during the 2 km performance tests. The post trial B[Lac⁻] was also significantly higher following the glucose trials (27%). This has previously been reported following exercise lasting approximately 1 minute in duration by Jacobs (1981). She reported a drop in muscle force during intense contractions following a period of prolonged exercise, and, like the present study this was accompanied by far greater metabolic changes. Jacobs (1981) reported that muscle lactate levels were reduced, and speculated that due to prolonged exercise, glycogen depletion would decrease the available substrate available for enzymatic reactions; in other words prolonged exercise would reduce the flux through glycolysis. Jacobs (1981) also indicated that as the performance changes were not of the same magnitude as the fall in the lactate concentrations, and that lipid utilisation may have contributed to offset some of the deficit in energy demand. Lipid utilisation has been reported during high intensity exercise (Essen *et al.*, 1977). Evidence of reduced carbohydrate use is also supported by the one subject respiratory data, where $\dot{V}CO_2$ and RER values indicative of substrate use were lower during the placebo trials compared to the glucose supplemented trials.

Palmer *et al.* (1997) speculated that during a post trial performance test, glycogen depletion was the likely factor causing fatigue following an intermittent exercise trial. They speculated that improved performance following an even power trial was due to reduced demands upon glycogen reserves during steady state exercise. Palmer *et al.* (1997) reported no markers of substrate utilisation during these trials. Interpretations of the results from this current study come from a variety of responses that have implications for fuel utilisation during exercise. During the simulation protocols the respiratory data showed elevated RER values during the road race simulation trials at the points sampled during the protocol. This could simply demonstrate that there was not true steady state under the conditions measured during the road race simulation trial at the sampling points. The higher RER values for the road race simulation trials (which are confirmed by the one subject whole simulation trials) show that there is continual adjustment of $\dot{V}O_2$, $\dot{V}CO_2$, and hence RER throughout the protocol. This continual adjustment is due to the continual changes in power output that is characteristic of road cycling. This continual adjustment will not be covered initially by the consumption of O_2, thus anaerobic sources will be required. The elevated B[Lac⁻] and RER values are thus indicators of an elevated carbohydrate requirement for this type of exercise.

The adjustment to match the power demands is likely to involve fast twitch muscle fibre. Likewise during the performance trials of the hill climb study and the simulation studies the relatively high power outputs, plus the high B[Lac⁻] recorded, indicate that fast twitch fibre are contributing to the maintenance of these work rates. Repeated use of fast twitch fibres during the road race simulation protocol possibly induced a degree of glycogen depletion within these fibres. This would reduce the effectiveness of this muscle fibre type during the performance test at the end of the road race simulation study, and possibly contribute to the reductions in power output during the performance tests of these trials. The B[Lac⁻] 3 minutes post performance tests were lower for the road race versus the even power trials, but this did not reach statistical significance.

If as speculated by Palmer *et al.* (1997) variable intensity exercise causes greater reductions in muscle glycogen than even power exercise then following placebo road race trial you would expect the poorest performance. This was confirmed by the slowest times to complete the 2 km performance test. $\dot{V}O_2$max values, absolute ($l\,min^{-1}$) or relative (kg^{-1}, $kg^{-0.33/0.67/0.79}$) failed to correlate significantly to fresh performance, or to the performances during both even power trials and the road race glucose trial. $\dot{V}O_2$ indices correlated significantly to the performance of the simulated road race placebo trial. If fast twitch muscle fibre was glycogen depleted, more emphasis would be placed upon the slow twitch aerobic fibres. As it appears that fast twitch fibres and slow twitch fibres (high $\dot{V}O_2$ values recorded during the performance testing) are recruited during the fresh performance testing, a reduction in the ability to use fast twitch muscle fibres would reduce performance, and, place power production from slow twitch muscle thus performance being derived from oxidative sources. Thus the $\dot{V}O_2$max values correlate strongly to performance for the post road race placebo trial.

This again highlights the importance of tactical awareness during road cycling as if selective glycogen depletion is a determinant of reductions in performance during the latter stages of road race cycling, opting when to use precious carbohydrate stores, and maintaining force production for the latter stages of the race is important.

Chapter Fifteen. Conclusion

The results from these studies have highlighted some specific areas for future research into the energy demands of road and time trial cycling.

15.1 Laboratory Equipment and Methodology

The use of laboratory-based ergometers has been previously highlighted as an area of concern in terms of specifically testing road cyclists. This study has highlighted the possible differences between not only level and graded cycling (in terms of physiological responses), but also potential differences between Kingcycle and treadmill cycling in terms of relative physiological responses. The SRM technology is now available to assess the breakdown of the pedal revolution during all types of cycling (analysis and sampling at 50 Hz), therefore a comparison of torque produced through the pedal action may start to answer some of the questions posed during this study on an altered physiological response during cycling on different ergometry systems and at different gradients.

The hill climb study data (chapter 10) highlighted that trained cyclists could sustain very high power outputs during simulated climbing. These are higher than has previously been reported (Fairbarn *et al.*, 1991) and for a lower physiological cost than in previous stationary (level) ergometer testing. Possible explanation of these findings comes from chapter 11, where reduced $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}E$ on a gradient indicates that there is reduced central overload for the same external work output during simulated hill climb cycling. As hill climbing is such an important component of road cycling, further investigation should focus on the possible causes of this phenomenon. We have speculated that enhanced fibre recruitment when riding in the upright climbing position could be the key factor in enhancing power output. EMG evaluation of key muscle groups involved in cycling would establish if this is the cause of the higher work rates on the gradient. More frequent sampling using the latest SRM system also tell us if the force generating characteristics are different when riding on a gradient compared to level cycling. These potential future studies have implications for the competitive cyclist because if they can be manipulated for level cycling (position change of the bicycle for example) then they may significantly enhance performance. However, despite the changes in physiological responses during level and graded cycling it still appears that stationary ergometer testing can predict simulated hill climb performance in the laboratory.

Other possible differences between the laboratory and the field conditions lie with the measurement of anaerobic power output. It appears that the use of the Wingate protocol also has limitations in terms of the analysis of the road cyclist. Data from the BCF races show that riders can elicit higher peak powers on the road rather than the laboratory setting. Further research into more specific protocol/testing procedures that provide a valid representation of field results is required in order to assess the cyclists. It is probably likely that peak powers are achieved during road cycling when the riders are out of the saddle, therefore this may cast doubt on the use of the seated Wingate testing protocol in the assessment of anaerobic power in these athletes. A direct comparison of seated and standing Wingate testing compared to field data may offer

further insight into the likely cause of the discrepancy between field and laboratory recorded data in this study.

15.1.1 Competitive cyclists

The aim of this study was to evaluate competitive cyclists in a sport specific laboratory environment, and thus try to highlight some of the key components that make up a successful road race cyclist. The subjects in the road race study had equivalent maximum power outputs to the elite professional riders that have previously been reported (Lucia *et al.*, 1998). However again, the protocols used possibly give different results (speculated to be 10% lower in step protocols, Lucia *et al.*, 1998). This is highlighted by the vast difference in the performance abilities of the riders in these studies compared to the continental professional riders who compete at the top of the world rankings. For a true comparison similar protocols and equipment have to be used. The use of the Kingcycle MMP protocol appears to give the sport scientist a lot of information regarding the lactate inflection point, which has been indicated as a major predictor of (steady state) endurance performance (Coyle *et al.* 1991). This data, and the findings from Davison *et al.* (1997) confirm that the smooth ramp protocol of the Kingcycle MMP test enables a good non-invasive prediction of the lactate inflection point.

MMP also appears reliable (CV 1.72%) in subjects who have prepared in a manner equivalent to their pre race preparation. i.e. a full day off prior to the MMP test, plus a following of their pre race feeding and drinking pattern has shown to provide reproducible results. As this figure appears vital in the sustained power that these endurance athletes can sustain, then further research should focus on methods of training (interval/continuous), volume of training, and intensity of training that might be implemented to try to improve the MMP figure for the competitive cyclist.

15.2 Road Race cycling performance

One of the major findings of this study is that road race cycling is far more variable than has previously been envisaged by sport scientists. The variability is also greater than has previously been used in a simulation by Brouns *et al.* (1989) and Palmer *et al.* (1997), however the mean power output during the races is not particularly high.

The importance of tactical awareness during the road race is important to ensure the rider does not waste energy, but expends the required energy in creating breaks and marking those other riders who are likely to play a major role in the outcome of the races. The Stage race data on the 3 riders competing in the same race further highlights the importance of tactics and rider role in the collection of physiological data from road cycling events.

Not only were the powers recorded higher than had previously been envisaged, there was a significant proportion of the data (~12%) at zero watts. This again has not previously been simulated in other studies into this sport, where although the mean values for the simulations may have been similar (Palmer *et al.*, 1997, had a mean relative exercise intensity of 58%) to the overall road race data average, the spectrum of

159

power outputs covered by the road race cyclist is a lot wider than has previously been reported. This of course would not be highlighted by the heart rate response to exercise (zero watts does not give you zero heart rate), and it is simply the availability of SRM technology that has allowed this data to become available.

Evidence of the importance of tactics comes from the initial road race data collection. There were no strong predictors from the laboratory data indicating what were the main attributes that would indicate a high finishing position during road race cycling. However, once the tactics were removed in the laboratory simulation study, by each rider performing the same relative amount of work, the prediction of simulated performance from physiological factors could be made from the standard stationary ergometer measures (Kingcycle). The requirement for riders to have some kind of 'race plan' and to be aware of what is happening during a race appear crucial to the outcome, and further confirms the requirement of coaching from a physiological and psychological background.

15.3 Physiological responses to simulated road race cycling

To our knowledge, this is the first study to use an intermittent pattern of power outputs to simulate road cycling. These powers have been derived from actual race performances, this coupled with differences in physiological responses during simulated climbing, and the incorporation of this into the simulation, make the results more sport specific than previous studies in this area. This therefore makes the physiological response data very interesting to the cyclist and sport scientist. The results from the road race simulation trials indicate that optimising carbohydrate use, minimising the appearance of NH_3 and urea, and having adequate substrate to produce higher B[Lac$^-$] at the end of exercise will benefit performance in these athletes.

15.3.1 Blood lactate response to exercise

One of the most obvious points of discussion and further research comes from the B[Lac$^-$] data recorded during the simulation study. Despite a series of 5 intense bursts, the B[Lac$^-$] data showed only a small increase over the even power trial ($p<0.05$). There could be a few possible explanations for this metabolic pattern. 1) the riders did not produce Lac$^-$ during these sprints, 2) The riders' [Lac$^-$] did not appear in the blood after these sprints, 3) the riders could clear the B[Lac$^-$] very quickly after the sprints. The single sample of B[Lac$^-$] does not give us the information to be certain on the reason behind the relatively low B[Lac$^-$].

Despite the low variability in B[Lac$^-$] at the sampling points, $\dot{V}O_2$ and $\dot{V}CO_2$ showed wide variations during the simulated trials depending upon the exercise intensity. Further investigation into the BLac$^-$ response with blood and intramuscular biopsy sampling, plus establishing patterns of production and removal of BLac$^-$ during this type of exercise would give further detail, and enable explanations to be proposed for the relatively low B[Lac$^-$].

Currently in coaching circles the emphasis on training is centred around aerobic power, the road race data has shown high peak anaerobic powers, despite the relatively low B[Lac⁻] values, further study into the effects of anaerobic power on road race cycling could highlight an optimum way of preparing these athletes for competition. If a simulation study was conducted on two groups of subjects with similar MMP values but different anaerobic power and anaerobic capacities, the effect on the performance outcome may enlighten the coach and the sport scientist to the role of anaerobic power and capacity on road race performance.

15.3.2 Heart rate response to exercise

Previous studies have reported high heart rate drifts during laboratory tests (Essen *et al.*, 1977), and we also have evidence that this is not simply a laboratory phenomenon with heart rate drift relative to steady state power output being recorded during time trial cycling. During this current investigation, the riders who had higher heart rate drift had a poorer relative performance following the two-hour even power and the simulated road trials. The drift in heart rate, plus the apparent 'inflation' in heart rate during road cycling make the interpretation of heart rate more complicated for the athlete and the sport scientist. Previous research by Palmer *et al.*, (1994) used heart rate data from road race cycling to establish a power output at which these riders were working. The data presented here shows fundamental problems with this method, problems associated with laboratory and field differences for the same external workload, heart rate drift, and changes during stage race cycling. Further research is required into the mechanisms associated with the inflated heart rate response in the field. The laboratory simulation did not initiate this response, so further field investigation of the cyclists' performance/response may be required to determine the cause of this change in the heart rate power relationship.

15.3.3 Substrate utilisation

Glucose supplementation significantly elevated blood glucose concentrations during the experimental trials. There was no difference between road race and even power trials, however there does appear from other data that there are differences in carbohydrate utilisation between these trials. Confirmation of this would require muscle biopsy sampling, and the analysis of different fibre types to support the findings from this study.

This study demonstrated a significant improvement in performance post intermittent and even power cycling with glucose polymer supplementation compared to the placebo trials. The supplementation enabled the cyclists to produce power outputs closer to those achieved during the fresh trials.

This is further evidence that substrate availability is vitally important to the cyclist when competing in either even power or intermittent road race cycling events.

References

Ahlborn, E.N., Davis, J.M. and Bailey, S.P. (1992). Effects of ammonia on endurance performance in the rat. *Medicine and Science in Sports and Exercise*, **24**, s50.

Anderson, G., and Rhodes, E. (1991).Relationship between blood lactate and excess CO_2 in elite cyclists. *Journal of Sports Sciences,* **9**, 173-181.

Arnall, DA., Marker, JC., Conlee, RK., Winder, WW. (1986). Effects of infusing epinephrine on liver and muscle glycogenolysis during exercise in rats. *American Journal of Physiology*, **250**, e641-e649.

Astrand, P.O. and Rodahl, K. (1986). *Textbook of Work Physiology - Physiological Bases of Exercise.* 3rd edition. Singapore: McGraw-Hill.

Babji, P., Matthews, S.M. and Rennie, M.J. (1983). Changes in blood ammonia, lactate and amino acids in relation to workload during bicycle ergometer exercise in man. *European Journal of Applied Physiology*, **50**, 405-411.

Balmer, J., Davison, R.C.R., Coleman D.A., Bird, S.R. (2000). The validity of power output recorded during exercise performance tests using a kingcycle air braked cycle ergometer when compared with an srm powermeter. *International Journal of Sports Medicine*. **21**, 195-199.

Balmer, J., Coleman, D.A., Davison R.C.R., Theakston, S. and Bird, S.R. (1998). Blood lactate and cardio-respiratory responses when cycling for 70 min at lactate minimum power output. *Medicine and Science in Sports and Exercise*, **30,** S306.

Balsom, P.D., Seger, J.Y., Sjodin, B. and Ekblom, B. (1992). Physiological responses to maximal intensity intermittent exercise. *European Journal of Applied Physiology*, **65**, 144-149.

Bangsbo, J. (1994). Energy demands in competitive soccer. *Journal of Sports Sciences,* **12,** s5-s12.

Banister, E.W., Allen, M.E., Mekjavic, I.B., Singh, A.K., Legge, B. and Mutch, B.J.C. (1983). The time course of ammonia and lactate accumulation in blood during bicycle exercise. *European Journal of Applied Physiology*, **51**, 195-202.

Barany, M. (1967). ATPase activity of myosin correlated with speed of muscle shortening. *Journal General Physiology,* **24**, 284-290.

Barlow, K., Weltman, A., Schurrer, R. and Henritze, J. (1985). Prediction of maximal effort bicycle ergometer endurance performance. *International Journal of Sports Medicine,* **6**, 190-196.

Below, P., Mora-Rodriguez, R., Gonzalez-Alonso, J. and Coyle, E. (1995). Fluid and CHO ingestion independently improve performance during 1hr of intense exercise. *Medicine and Science in Sports & Exercise*, **27**, 200-210.

Bergstrom, J. and Hultman, E. (1967). A study of the glycogen metabolism during exercise in man. *Scand J Clin Lab Invest,* **19**, 218-228.

Berry, M., Storsteen, J. and Woodard, M. (1993). Effects of body mass on exercise efficiency and VO_2 during steady state cycling. *Medicine and Science in Sport and Exercise,* **25**, 1031-1037.

Bishop, D., Jenkins, D. and MacKennon, L. (1998). The relationship between plasma lactate parameters, peak power, and one hour cycling performance in women. *Medicine and Science in Sport and Exercise,* **8**, 1270-1275.

Boobis, L., Williams, C. and Wootton, S. (1982). Human muscle metabolism during brief maximal exercise. *Journal of Physiology,* **338**, 21-22.

Boulay, M., Simoneau, J., Lortie, G. and Bouchard, C. (1997). Monitoring high intensity endurance exercise with heart rate and thresholds. *Medicine and Science in Sport and Exercise,* **29**, 125-132.

Brennan, J.H., Wallace, E.S. and White, J.A. (1994). Physiological characteristics of commonwealth games cycling squad. *Journal of Sports Sciences, 12, s130.*

Broberg, S. and Sahlin, K. (1989). Adenine nucleotide degradation in human skeletal muscle during prolonged exercise. *Journal of Applied Physiology*, **67**, 116-122.

Brooks, G. (1987a) Lactate production during exercise: oxidizable substrate vs fatigue agent. In *Exercise: Benefits, Limits and Adaptations*. (edited by D. Macleod, R. Maughan, M. Nimmo, T. Reilly and C. Williams). pp.144-159. London :E & FN Spon

Brooks, G.A. (1987b). Amino acid and protein metabolism during exercise and recovery. *Medicine and Science in Sports and Exercise*, **19**, s150-s156.

Brouns, F., Beckers, E., Wagenmakers, A.J.M. and Saris, W.H.M. (1990). Ammonia accumulation during highly intensive long lasting cycling: individual observations. *International Journal of Sports Medicine*, **11**, s78-s84.

Brouns, F., Saris, W., Stroeken, J., Beckers, E., Thijessen, R., Rehrer, N. and Hoor, F. (1989). Eating drinking and cycling, a controlled Tour de France simulation. *International Journal of Sports Medicine*, **10**, 32-40.

Buchanan, M. and Weltman, A. (1985). Effect of pedal frequency on VO$_2$ and work output at lactate threshold, fixed blood lactate concentrations of $_2$mM and 4 mM, and max in competitive cyclists. *International Journal of Sports Medicine,* **6**, 163-168.

Burke, E., Cerny, F., Costill, D. and Fink, W. (1977). Characteristics of skeletal muscle in competitive cyclists. *Medicine and Science in Sport and Exercise,* **9**, 109-112.

Calders, P., Pannier, J.L., Matthys, D.M. and Lacroix, E.M. (1997). Pre exercise branched chain amino acid administration increases endurance performance in rats. *Medicine and Science in Sports and Exercise*, **9**, 1182-1186.

Carlson, N. (1991). *Physiology of Behaviour.* pp 379-400. Massachusetts: Allyn & Bacon Press.

Cavanagh, P. and Kram, R. (1985). Mechanical and muscular factors affecting the efficiency of human movement. *Medicine and Science in Sport and Exercise,* **17**, 326-331.

Cavanagh, P. and Kram, R. (1985). The efficiency of human movement a statement of the problem. *Medicine and Science in Sport & Exercise,* **17**, 304-308.

Cerny, F.J. (1975). Protein metabolism during two hour ergometer exercise. *In Metabolic adaptations to prolonged physical exercise.* (edited by H. Howard and J.R. Poortmans). pp. 232-237. Basel: Birkauser Verlag.

Coast, R., Cox, R. and Welch, H. (1986). Optimal pedalling rate in prolonged bouts of cycle ergometry. *Medicine and Science in Sport and Exercise,* **18**, 225-230.

Coggan, A. and Coyle, E. (1987). Reversal of fatigue during prolonged exercise by CHO infusion or ingestion. *Journal of Applied Physiology,* **63**, 2388-2395.

Coggan, A. and Coyle, E. (1989). Metabolism and performance following CHO ingestion late in exercise. *Medicine and Science Sport & Exercise,* **21**, 59-65.

Coggan, AR., Coyle, EF. (1987). Reversal of fatigue during prolonged exercise by carbohydrate infusion or ingestion. *Journal of Applied Physiology*, **63**, 2388-2395.

Coggan, AR., Coyle, EF. (1988). Effect of carbohydrate feeding during high intensity exercise. *Journal of Applied Physiology*, **65**, 1703-1709.

Coleman, D., Passfield, L., Hales, T. and Wright, M. (1995) The effect of creatine supplementation on endurance performance. *Journal of Sports Sciences*, **13**, 23-24.

Collins, M., Cureton, K., Hill, D. and Roy, C. (1989). Relation of plasma volume change to intensity of weightlifting. *Medicine and Science in Sport and Exercise*, **21**, 178-185.

Costil, D. and Hargreaves, M. (1992). Nutrition and fatigue. *Sports Medicine*, **13**, 86-92.

Coyle, E., Sidossis, L., Horowitz, J. and Beltz, J. (1992) Cycling efficiency is related to % slow twitch muscle fibres. *Medicine and Science in Sport and Exercise,* **24**, 782-788.

Coyle, E., Feltner, M., Kautz, S., Hamilton, M., Montain, S., Baylor, A., Abraham, L. and Petrek, G. (1991). Physiological and biomechanical factors associated with elite endurance cycling performance. *Medicine and Science in Sport and Exercise,* **23**, 93-107.

Craig, N., Norton, K., Bourdan, D., Woolford, S., Stonef, T., Squires, B., Olds, T., Conyers, R. and Walsh, C. (1993). Aerobic and anaerobic indices contributing to track endurance cycling performance. *European Journal of Applied Physiology,* **67**, 150-158.

Crowley, M., Alterink, C., Matt, K. and Willis W. (1996). Effects of cycling cadence on VO$_2$ kinetics and delta efficiency. *Medicine and Science in Sport and Exercise,* **28**, S21.

Cunningham, J., Carter, N., Rector, F. and Seldin, D. (1971). Resting transmembrane potential differences of skeletal muscle in normal subjects and severely ill patients. *Journal of Clinical Investigation*, **50**, 49-59.

Daniels, J. (1985). A physiologists view of running economy. *Medicine and Science in Sport and Exercise,* **17**, 332-338.

Davison, R.C.R. and Flynn, S. (1997) Optimal pedalling cadences for racing cyclists. *Journal of Sports Sciences,* **15**, 45.

Davison, R.C.R., Coleman, D. and Bird, S. (1997). Determination of the Lactate Minimum points in Cyclists (conference abstract), *Journal of Sports Sciences* **15**(1), 45 - 46.

Decombaz, J., Reinhardt, K., Anantharaman, G., Von Glutz, G. and Poortmans, J.R. (1979). Biochemical changes in a 100km run: free amino acids, urea and creatinine. *European Journal of Applied Physiology*, **41**, 61-72.

Dennis, K. and Renney, K. (1992). Marathon Tragedy raises questions. *Inside Texas Running*, **16**, 7.

Dill, D. and Costill, D. (1974). Calculations of percentage change in volumes of blood plasma, red cells in dehydration. *Journal of Applied Physiology*, **37**, 247-248.

Dobbins, T., Morris, K. and Fallowfield, J.L. (1998). The effect of test protocol on $\dot{V}O_{2peak}$ and maximum power output during sport specific testing of experienced cyclists. *Journal of Sports Sciences*, **16**, 26-27.

Dohm, G.L., Tapscott, E.B. and Kasperek, G.J. (1987). Protein degradation during endurance exercise and recovery. *Medicine and Science in Sports and Exercise*, **19**, s166-s171.

Drabbs, M. and Maud, P. (1997). A single test for assessing maximal oxygen consumption and anaerobic performance capacity of competitive cyclists. *Journal of Strength and Conditioning Research,* **11**, 125-128.

Einspahr, K.J., and Tharp, G. (1989). Influence of endurance training on plasma amino acid concentrations in humans at rest and after intense exercise. *International Journal of Sports Medicine*, **10**, 233-236.

Ekblom, B. (1970). Effect of physical training on circulation during prolonged severe exercise. *Acta. Physiol. Scand*, **78**, 145.

England, B.K. and Russ Price, S. (1995). Acidosis and glucocorticoids interact to provoke muscle protein and amino acid catabolism. *Blood purification*, **13**, 147-152.

Eriksson, L.S., Broberg, S., Bjorkman, O. and Wahren, J. (1985). Ammonia metabolism during exercise in man. *Clinical Physiology*, **5**, 325-336.

Essen, B., Hagenfeldt, L. and Kaijser, L. (1977). Utilisation of blood borne and intramuscular substrates during continuous and intermittent exercise in man. *Journal of Physiology*, **265**, 489-506.

Evangelisti, M.I., Verde, T.J., Andres, F.F. and Flynn, M.G. (1994). Effects of handlebar position on physiological responses to prolonged cycling. *Medicine and Science in Sport and Exercise*, **26**, s64.

Fairburn, M.S., Coutts, K.C., Pardy, R.L., McKenzie, D.C. (1991). Improved respiratory muscle endurance of highly trained cyclists and the effects on maximal exercise performance. *International Journal of Sports Medicine*, **12**, 66-70.

Fallowfield, J., Williams, C., Wilson, W., Booth, J., Growns, S. and Choo, B. (1994). Effect of water ingestion on endurance capacity during prolonged constant pace running. *Presented at the B.A.S.E.S. Conference*, Aberdeen University July 18-21.

Farber, H., Arbetter, J., Schaefer, E., Hill, S., Dallal, G., Grimaldi, R. and Hill, N. (1987). Acute metabolic effects of an endurance triathlon. *Annals of Sports Medicine*, **3**, 131-138.

Faria, I. (1992). Energy expenditure, aerodynamics and medical problems in cycling. *Sports Medicine*, **14**, 43-63.

Faria, I., Faria, C., Roberts, S. and Yoshimura, D. (1989). Comparison of physical and physiological characteristics in elite young and mature cyclists. *Research Quarterly*, **60**, 388-395.

Felig, P. (1975). Amino acid metabolism in man. *Ann rev biochem*, **44**, 260-266.

Flynn, M.G., Costill, D.L., Hawley, J.A., Neufer P.D., Fielding R.A., Sleeper, M.D. (1987). Influence of selected carbohydrate drinks on cycling performance and glycogen use. *Medicine and Science in Sport and Exercises*, **19**, 37-40.

Foster, C., Snyder, A.C., Thompson, N.N., Green, M.A., Foley, M. and Schrager, M. (1993). Effect of pacing strategy on cycle time trial performance. *Medicine and Science in Sport and Exercise*, **25**, 383-388.

Gaesser, G. and Brookes, G. (1985). Muscular efficiency during steady rate exercise : effects of speed

and work rate. *Journal of Applied Physiology* **38**, 1132-1139.

Galbo, H., Holst, J.J., Christensen N.J. (1975). Glucagon and plasma responses to graded and prolonged exercise in man. *Journal of Applied Physiology,* **38**, 70-76..

Gollnick, P., Karlsson, J., Piehl, K. and Saltin, B. (1978). Phosphorylase a in human skeletal muscle during exercise and electrical stimulation. *Journal of Applied Physiology,* **45**, 852-857.

Gotshall, R., Bauer, T. and Fahrner, S. (1996) Cycling cadence alters exercise hemodynamics. *International Journal of Sports Medicine,* **17**, 17-21.

Graham, T.E. and MacLean, D.A. (1998) Ammonia and amino acid metabolism in skeletal muscle: human, rodent and canine models. *Medicine and Science in Sports and Exercise,* **30**, 34-46.

Graham, T.E., Rush, J.W.E. and MacLean, D.A. (1995). Skeletal muscle amino acid metabolism and ammonia production during exercise. In *Exercise Metabolism.* (edited by M. Hargreaves). pp.131-176. Champaign, Illinois: Human Kinetics.

Graham, T.E., Turcotte, L.P., Kiens, B. and Richter, E.A. (1997). Effect of endurance training on ammonia and amino acid metabolism in humans. *Medicine and Science in Sports and Exercise,* **29**, 646-653.

Green, H.J., Jones, S., Ball-Burnett, M. and Fraser, I. (1991). Early adaptations in blood substrates, metabolites, and hormones to prolonged exercise training in man. *Can.J.Physiol.Pharmacol,* **69**, 1222-1229.

Greenhaff, P.L., Casey, A., Short, A., Harris, R., Soderlund, K. and Hultman, E. (1992) Influence of oral creatine supplementation of muscle torque during repeated bouts of maximal voluntary exercise in man. *Clinical Science,* **84**, 565-571.

Greenleaf, H., Convertino, V., Stremel, E., Bernauser, E., Adams, W., Vignow, S. and Brock, P. (1977). Plasma sodium, Calcium, volume shifts and thermoregulation during exercise in man. *Journal of Applied Physiology,* **2**, 1026-1032.

Hagan, D., Weis, S. and Raven, P. (1992). Effect of pedal rate on cardiorespiratory responses during continuous exercise. *Medicine and Science in Sport and Exercise,* **24**, 1088-1095.

Hagberg, J., Mullin, J., Giese, M. and Spitznagel, E. (1981). Effect of pedalling rate on submaximal responses of competitive cyclists. *Journal of Applied Physiology,* **51**, 447-451.

Hamiliton, A., Neville, M., Brooks, S. and Williams, C. (1991). Physiological responses to maximal intermittent exercise: Differences between endurance trained runners and games players. *Journal of Sports Sciences,* **9**, 371-382.

Haralambie, G. and Berg, A. (1976). Serum urea and amino nitrogen changes with exercise duration. *European Journal of Applied Physiology,* **36**, 39-48.

Hargreaves, M. and Briggs, C. (1988). Effect of CHO ingestion on exercise metabolism. *Journal of Applied Physiology,* **65**, 1553-1555.

Hargreaves, M., Costil, D., Coggan, A., Fink, W. and Nishibata, L. (1984). Effect of carbohydrate feedings on muscle glycogen utilisation and exercise performance. *Medicine and Science Sport & Exercise,* **16**, 219-222.

Harrison, M., Edwards, R. and Leitch, D. (1975). Effect of exercise and thermal stress on plasma volume., *Journal of Applied Physiology,* **39**, 925.

Hawley, J.A. and Noakes, T.D. (1992). Peak power output predicts maximal oxygen uptake and performance time in trained cyclists. *European Journal of Applied Physiology,* **65**, 79-83.

Head, A., Jakeman, P.M. and Kendall, M.J. (1996). Ammonia and hypoxanthine production during exercise on ß1selective and non selective beta blockade. *Journal of Sports Sciences,* **14**, 87-88.

Herbert, W. (1983). Water and electrolytes. *Ergogenic Aids in Sport.* (edited by M. Williams). pp 56-95. Champaign, Illinois: Human Kinetics.

Hermansen, L. and Osnes, J. (1972) Blood and muscle pH after maximal exercise in man. *Journal of Applied Physiology,* **32**, 304-308.

Hickey, M.S., Costil, D.L., McConnell, G.K., Widrick, J.J. and Tanaka, H. (1992). Day to Day variation in time trial cycling performance. *International Journal of Sports Medicine,* **13**, 467-470.

Hillier, W., OToole, M. and Massimo, R. (1985). Electrolyte and glucose changes during the Hawiian Ironman triathalon. *Medicine and Science in Sport and Exercise, Medicine and Science in*

Sport and Exercise, **17**, 215.

Holloszy, J.O. (1975). Adaptation of skeletal muscle to endurance exercise. *Medicine and Science in Sport and Exercise*, **7**, 155-164.

Holloszy, J.O. and Coyle, E.F. (1984). Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *Journal of Applied Physiology*, **56**, 831-838.

Houston, M.E. (1995). Amino acid metabolism. In *Biochemistry Primer of Exercise Science*. Pp117-122. Human Kinetics, Champaign, Illinois.

Hudlika, O., Zweifach, B. and Tyler, K. (1982). Capillary recruitment and flow velocity in skeletal muscle after contraction. *Microvasc*. Res, **23**, 201-213. Cited in Senay & Pivarnik (1985).

Hultman, E. (1978). Regulation of carbohydrate metabolism in the liver during rest and exercise with special reference to diet. *In Landry, Orban, 3rd international symposium on the Biochemistry of Exercise*. pp 99-126. Miami: Symposium Specialists.

Hultman, E. and Sjoholm, H. (1983). Substrate availability. In *Biochemistry of exercise* (edited by H. Knuttgen, J. Vogel and J. Poortmans). pp 63-75. Champaign, Illinois: Human Kinetics.

Hultman, E., Spriet, L. and Sodelund, K. (1987) Energy metabolism and fatigue in working muscle. In *Exercise: Benefits, Limits and Adaptations* 63-85. Edited by Macleod D, Maughan R, Nimmo M, Reilly T, Williams C, E & FN Spon. London.

Ingen Schenau, G.J., Koning, J.J. de. and de Groot, G. (1992). The distribution of anaerobic energy in 1000m and 4000m cycling bouts. *International Journal of Sports Medicine*, **13**, 447-451.

Irving, R., Noakes, T.D., Burger, S., MyBurgh, K., Queirdo, D. and Van Zyl Smit, R. (1990). Plasma volume and renal function during and after ultra marathon running. *Medicine and Science in Sport and Exercise*, **22**, 581-587.

Irving, R.A., Noakes, T.D. and Van Zyl Smit, R. (1989). Metabolic and renal changes in two athletes during a world 24 hour relay record performance. *British Journal of Sports Medicine*, **23**, 227-232.

Itoh, H. and Ohkuwa, T. (1990). Peak blood ammonia and lactate after submaximal, maximal and supramaximal exercise in sprinters and long distance runners. *European Journal of Applied Physiology*, **60**, 271-276.

Ivy, J., Chi, M., Hintz, C., Sherman, W., Hellendall, R. and Lawry, H. (1987). Progressive metabolite changes in individual muscle fibres with increasing work rates. *American Journal of Physiology*, **252**, c630-c639.

Jacobs, I. (1981). Lactate concentrations after short, maximal exercise at various glycogen levels. *Acta Physiol Scand*, **111**, 465-469.

Jenkins, D. and Quigley, B. (1990).Blood lactate in trained cyclists during cycle ergometry at critical power. *European Journal of Applied Physiology*, **61**, 278-283.

Jenkins, D., Hutchins, C. and Spillman, D. (1994) The influence of dietary CHO and pre exercise glucose consumption on supramaximal intermittent exercise performance. *British Journal of Sports Medicine*, **28**, 266-271

Jeukendrup, A., and Van Dieman, A. (1998). Heart rate monitoring during training and competition in cyclists. *Journal of Sports Sciences*, **16**, 591-599.

Jones, S. and Passfield, L. (1998). The dynamic calibration of bicycle power measuring cranks. In *The engineering of Sport*. (edited by S. Haake). pp 265-274. Oxford: Blackwell Science.

Jordan, L. and Merrill, E. (1979). Relative efficiency A function of pedalling rate for racing cyclists. *Proceedings of the Physiological Society conference*, 49-50.

Kargotich, S., Goodman, C., Keast, D. and Morton, A. (1998). The influence of plasma volume changes on the interpretation of biochemical parameters used for monitoring exercise, training and sports. *Sports Medicine*, **26**, 101-117.

Kasperek, G.J., Dohm, G.L, Barakat, H.A., Srausbauch, P.H., Barnes, D.W. and Snider, R.D. (1982). The role of lysomes in exercise induced hepatic protein loss. *Biochem. J*, 202, 281-288.

Keen, P.S., Passfield, L. and Hale, T. (1991). Indirect determination of $\dot{V}O_{2max}$ using a sport specific (cycling) ergometry system. *Journal of Sports Sciences*. **9**, 420.

Kenny, G.P., Reardon, F.D., Marion, A. and Thoden, J.S. (1995). A comparative analysis of physiological responses at submaximal workloads during different laboratory simulations of field cycling. *European Journal of Applied Physiology*, **71**, 409-415.

Kozaris, L.P. and Montgomery, D.L. (1991). Blood lactate concentration following intermittent and continuous cycling test of anaerobic capacity. *European Journal of Applied Physiology*, **63**, 273-277.

Kyle, C. (1991). Ergogenics for bicycling. In *Perspectives in exercise science and sports medicine, Volume 4 Ergogenics: enhancement of performance in exercise and sport.* (edited by D. Lamb and M. Williams). USA: WMC Brown.

Lehninger, A. (1975). *Biochemistry*. Worth Press, New York.

Lemon, P.W.R. (1987). Protein and exercise: update 1987. *Medicine and Science in Sports and Exercise*, **19**, s179-s190.

Lemon, P.W.R. and Mullin, J.P. (1980). Effect of initial glycogen levels on protein catabolism during exercise. *Journal of Applied Physiology*, **48**, 624-629.

Lemon, P.W.R., Deutsch, D.T. and Payne, W.R. (1989). Urea production during prolonged swimming. *Journal of Sports Sciences*, **7**, 241-246.

Lindsay, F.H., Hawley, J.A., Myburgh, K.H., Schomer, H.H., Noakes, T.D. and Dennis, S.C. (1996). Improved athletic performance in highly trained cyclists after interval training. *Medicine and Science in Sport and Exercise*, **28**, 1427-1434.

Linossier, M.T., Denis, C., Dormis, D., Geyssant, A. and Lacour, J.R. (1993) Ergometric and metabolic adaptation to a 5 second sprint training programme. *European Journal of Applied Physiology*, **67,** 408-414.

Loftin, M. and Warren, B. (1994). Comparison of a simulated 16.1 km time trial, $\dot{V}O_{2max}$ and related factors in cyclists with different ventilatory thresholds. *International Journal of Sports Medicine*, **15**, 498-503.

Lowenstein, J.M. (1972). Ammonia production in muscle and other tissues. The purine nucleotide cycle. *Science*, **171**, 387-400.

Lutoslawska, G., Klusiewicz, A., Sitkowskid, W., Krawczyk, B. (1996). Effect of simulated 2 km laboratory rowing on blood lactate, plasma inorganic phosphate and ammonia in male and female junior rowers. *Biology of Sport*, **13**, 31-38.

Lutoslawska, G., Ladyga, M., Klusiewicz, A. and Krawczyk, B. (1992). Plasma ammonia and blood lactate in male and female athletes following an intermittent rowing exercise. *Biology of Sport*, **9**, 175-182.

MacDougall, J., Ward, G., Sale, D. and Sutton, J. (1977). Muscle glycogen repletion after high intensity intermittent exercise. *Journal of Applied Physiology*, **42**, 129-132.

Madsen, K., MacLean, D.A., Kiens, B. and Christensen, D. (1996). Effects of glucose, glucose plus branched chain amino acids, or placebo on bike performance over 100 km. *Journal of Applied Physiology*, **81**, 2644-2650.

Malhotra, M.S., Gupta, R.K. and Khanna, G.L. (1985). Selection criteria for competitive road cyclists - a physiological approach. *Snipes Journal*, **8**, 20-29.

Marsh, A.P., Martin, P.E. (1997). Effect of cycling experience, aerobic power and power output on preferred and most economical cadence. *Medicine and Science in Sport and Exercises*, **29**, 1225-1232.

Marsh, A.P., Martin, P.E. (1998). Perceived exertion and preferred cycling cadence. *Medicine and Science in Sport and Exercises*, **30,** 942-948.

Massicote, D., Peronnet, C., Brisson, G., Hillaire, M. and Ledoux, M. (1985). Substrate utilisation and insulin response to glucose and fructose ingestion during exercise in men. *Medicine and Science in Sport and Exercise*, **17,** 280.

Maughan, R. (1991). Fluid and Electrolyte loss and replacement in exercise. *Journal of Sports Science*, **9**, 117-142.

Maughan, R. And Whiting, P. (1985). Factors influencing plasma glucose concentrations during marathon running. *Exercise Physiology*. 1 87-89, edited by Dotson C, Humphrey J, New York AMS Press.

McArdle, W., Katch, F. and Katch, V. (1991*) Exercise physiology, energy nutrition and human performance* 3rd edition. London:Lea & Febinger.

McCartney, M., Spreit, L., Heigenhauser, G., Kowalchuk, J., Sutton, J. and Jones, N. (1984). Muscle power and metabolism in maximal intermittent exercise. *Journal of Applied Physiology*, **60**,

1164-1169.

McCole, S., Claney, K., Conte, J., Anderson, R. and Hagberg, J. (1990) Energy expenditure during bicycling. *Journal of Applied Physiology*, **68**, 748-753.

McMurray, R. (1983). Plasma volume changes during submaximal swimming. *European Journal of Applied Physiology*, **51**, 347-356.

Medbo, J. and Tabataq, I. (1989). Relative importance of aerobic and anaerobic energy release during short lasting exhaustive bicycle exercise. *Journal of Applied Physiology*, **67**, 1881-1886.

Merrill, E. and White, J. (1984). Physiological efficiency of constant power output at varying pedal rates. *Journal of Sports Sciences*, **2**, 25-34.

Meyer, R.A., Dudley, G.A. and Terjung, R.L. (1980) Ammonia and IMP in different skeletal muscle fibres after exercise in rates. Journal of Applied Physiology, **49**, 1037-1041.

Myhre, L., Hartung, G. and Tucker, D. (1982). Plasma volume and blood metabolites in middle aged runners during a warm weather marathon. *European Journal of Applied Physiology*, **48**, 227-240

Nassis, G., Williams, C. and Chisnall, P. (1998). Effect of carbohydrate electrolyte drink on endurance capacity during prolonged high intensity running. *British Journal of Sports Medicine*, **32**, 248-252.

Neville, A. (2000). Just how confident are you when publishing the results of your research? *Journal of Sport Science*, **18**, 569-570.

Mendoza, W., Carey, D.G., Serfass, R.C. (1994). Validity of the conconi heart rate method for determining lactate threshold in competitive cyclists. *Medicine and Science in Sport and Exercises*, **26**, s64.

Neville, M., Williams, C., Roper, D., Slater, C. and Neville, A. (1993). Effect of diet on performance during recovery from intermittent sprint exercise. *Journal of Sports Sciences*, **11**, 119-126.

Nickleberry, B. and Brooks, G. (1996). No effect of cycling experience on leg cycle ergometer efficiency. *Medicine and Science in Sport and Exercise*, **28**, 1396-1401.

Novak, J. (1986). Blood changes during and after 24 hours swimming attempt. *Hungarian Review of Sports Medicine*, **27**, 275-285.

Olds, T., Norton, K., Craig, N., Olive, S. and Lowe, E. (1995). The limits of the possible - models of power supply and demand in cycling. *The Australian Journal of Science and Medicine in Sport*, **27**, 29-33.

Olsson, K. and Saltin, B. (1970). Variation in total body water with muscle glycogen changes in man. *Acta. Physiol. Scand*, **80**, 11.

Padilla, J., Mujika, I., Cuesta, G., Marie, J. and Chatard, J.C. (1996) Validity of a velodrome test for competitive road cyclists. *European Journal of Applied Physiology*, **73**, 446-451.

Pages, T., Murtra, B., Ibanez, J., Rama, R., Callis, A. and Palacios, L. (1994). Changes in blood ammonia and lactate levels during a triathlon race. *Journal of Sports Medicine and Physical Fitness*, **34**, 351-356.

Palmer, G., Hawley, J., Dennis, S. and Noakes, T. (1994). Heart rate responses during a 4-d cycle stage race. *Medicine and Science in Sport and Exercise*, **26**, 1278-1283.

Palmer, G.S., Noakes, T.D. and Hawley, J.A. (1997). Effect of steady state verses stochastic exercise on subsequent cycling performance. *Medicine and Science in Sport and Exercise*, **29**, 684-687.

Palmer. G., Hawley, J., Rodger, I., Burke, L. and Noakes, T. (1998). Carbohydrate ingestion immediately before exercise does not improve 20 km time trial performance in well trained cyclists. *International Journal of Sports Medicine*, **19**, 415-418.

Passfield, L., Hale, T. (1995). Endurance capacity in elite cyclists. *Journal of Sport Science*, **13**, 40.

Patterson, R.P., Moreno, M.L. (1990). Bicycling pedalling forces as a function of pedalling rate and power output. *Medicine and Science in Sport and Exercises*, **22**, 512-516.

Pfeiffer, R., Harder, B., Londis, D., Barber, D. and Harper, K. (1993). Correlating indices of aerobic capacity with performance in elite women road cyclists. *Journal of Strength and Conditioning Research*, **7**, 201-205.

Pivarnik, J. and Palmer, R. (1994). Water and electrolyte balance during rest and exercise. in, *Nutrition*

in Exercise and Sport 2nd edition. Edited by Wolinsky L, Hickson J, CRC press London.

Pivarnik, J., Montain, J., Graves, J. and Pollock, M. (1988). Effects of pedal speed during incremental cycle ergometer exercise. *Research Quarterly for Exercise and Sport,* **59**, 73-77.

Poole, D.C., Schaffartzik, W., Knight, D.C., Derion, T., Guy, H.J., Preduletto, R., Wagner, P.D. (1991). Contribution of the exercising legs to the slow component of oxygen uptake kinetics in humans. *Journal of Applied Physiology,* **18**, 1245-53.

Poortmans, J.R. (1971). Serum protein deamination during short exhaustive activity. *Journal of Applied Physiology,* **30**, 190-192.

Poortmans, J.R. (1988). Protein metabolism. In *Principles of Exercise Biochemistry.* (edited by J.R. Poortmans). pp 164-193. Basel, Karger: Med Sport Sci,.

Potteiger, J., Nickel, G., Webster, M., Haub, M. and Palmer, R. (1996). Sodium citrate ingestion enhances 30km cycling performance. *International Journal of Sports Medicine,* **17**, 7-11.

Rattu, J.A.M., Lin, X. and El-Sayed, M.S. (1996). The effect of carbohydrate ingestion on plasma urea and creatinine levels in response to cycling exercise in trained athletes. *Journal of Sports Sciences,* **14**, 98-99.

Rauwbottom, D.G., Keast, D., Garcia-Webb, P. and Morton, A.R. (1997). Training adaptation and biological changes among well trained male triathletes. *Medicine and Science in Sports and Exercise,* **29**, 1233-1239.

Robertson, S. and Kerrick, W. (1979). The effects of pH on Ca+ activated force in skeletal muscle fibres. *Pflugers Arch,.* **380**, 41-45.

Rocker, L., Kirsch, K., Heyduck, B. and Altenkirck, U. (1989). Influence of prolongued physical exercise on plasma volume, plasma protein, electrolytes, and fluid regulating hormones. *International Journal of Sports Medicine,* **10**, 270-274

Rollings, A.T., Porcari, J.P. and Floyd, W. (1995). Predictors of uphill riding performance in trained cyclists. *Medicine and Science in Sport and Exercise,* **26**, s238.

Ryschon, T.W. and Stray-Gunnersen, J. (1991). Effect of body position on the energy cost of cycling. *Medicine and Science in Sport and Exercise,* **23**, 949-953.

Ryschon, TW., Stray-Gundersen, J. (1991). Effect of body position on the energy cost of cycling. *Medicine and Science in Sports and Exercise,* **23**, 949-953.

Sahlin, K., Palskog, G. and Hultman, E. (1978). Adenine accumulation in skeletal muscle of the quadrecep muscle in men after exercise. *Pflugers Arch,* **367**, 143-149.

Sanderson, D. (1991). The influence of cadence and power output on the biomechanics of force application during steady rate cycling in competitive and recreational cyclists. *Journal of Sports Sciences,* **9**, 191-203.

Scanlon, E. and Saunders, P. (1991). *Anatomy and Physiology.* pp 441-453. USA: FA Davis Company.

Schabort, E.J., Hawley, J.A., Hopkins, W.G., Mujika, I. and Noakes, T.D. (1997). A novel reliable laboratory test of endurance performance for road cyclists. *Medicine and Science in Sport and Exercise,* **30**, 1744-1751.

Senay, L. and Kok, R. (1977). Effects of training and heat acclimatisation on blood plasma content of exercising men. *Journal of Applied Physiology,* **43**, 591-599.

Senay, L. and Pivarnik, J. (1985). Fluid shifts during exercise. *Exercise and Sport Science Reviews,* **13**, 335-358.

Senay, L., Rogers, G. and Jooste, P. (1980). Changes in blood plasma during progressive treadmill and cycle exercise. *Journal of Applied Physiology.* **49**, 59.

Shearn, W., Szmedra, L., Mookerjee, S. and Weatherford, J. (1996) Anaerobic power as a predictor of short time trial performance. *Medicine and Science in Sport and Exercise,* **13**, s156.

Sjogaard, G. (1984). Muscle morphology and metabolic potential in elite road cyclists during a season. *International Journal of Sports Medicine,* **5**, 250-254.

Sjogaard, G. and Saltin, B. (1982). Extra and intracellular spaces in muscles of man at rest and with dynamic exercise. *American Journal of Applied Physiology,* **240**, 271-280

Smith, E., Guyton, A., Manning, R. and White, R. (1976). Integrated mechanisms of Cardiovascular response and control during exercise in normal humans. *Prog. Cardiovascular Disease,* **18**,

169

421-443.

Spriet, L., Lindinger, M., McKelvie, R., Heigenhauser, G. and Jones, N. (1989). Muscle glycogenolysis and H+ concentration during maximal intermittent cycling. *Journal of Applied Physiology*, **66**, 8-13.

Spurway, NC. (1992). Aerobic exercise, anaerobic exercise and the lactate threshold. *British Medical Bulletin*. **48**, 569-596.

Stoval, K., Swain, D., Debendetti, K., Pruitt, A. and Burke, E. (1993). Body mass and performance in the tour dupont. *Medicine and Science in Sport and Exercise,* **25**, s169.

Suzuki, Y. (1979). Mechanical efficiency of fast and slow twitch muscle fibres in man during cycling. *Journal of Applied Physiology,* **47**, 263-267.

Svaren, S, Potteiger, J., Hopkins, D. and Evans, B. (1994). Validity and reliability of a cycle ergometer protocol using a racing cycle and indoor trainer. *Medicine and Science in Sport and Exercise*, **26**, s42.

Swain, D. (1994). Influence of body mass in endurance bicycling. *Medicine and Science in Sport and Exercises*, **26**, 58-63.

Swain, D.P. (1987). A model for optimising cycling performance by varying power on hills and in the wind. *Medicine and Science in Sport and Exercise*, **29**, 1104-1108.

Swain, D.P. and Wilcox, J. (1992). Effect of cadence on the economy of uphill cycling . *Medicine and Science in Sport and Exercise*, **24**, 1123-1127.

Symonds, D. and Jacobs, I.. (1989). High intensity exercise performance is not impaired by low intramuscular glycogen, *Medicine and Science in Sport & Exercise,* **21**, 550-557.

Takaishi, T., Yasuda, Y., Ono, T. and Moritani, T. (1996). Optimal pedalling rate estimated from neuromuscular fatigue for cyclists. *Medicine and Science in Sport and Exercise*, **25**, 1492-1497.

Tanaka, H., Bassett Jr, D., Swensen, T. and Sampedro, R. (1993). Aerobic and anaerobic power characteristics of competitive cyclists in the united states cycling federation. *International Journal of Sports Medicine,* **14**, 334-338.

Tanaka, K., Nakadomo, F., Maritani, T. (1987). Effects of standing cycling and the use of stirrups on maximal oxygen uptake. *European Journal of Applied Physiology*, **56**, 699-703.

Terrados, N., Fernandez, B. and Perez-Landaluce, J. (1994). Is anaerobic capacity limited in endurance athletes. *Medicine and Science in Sport and Exercises,* **26**, s183.

Tesch, P. and Wright, J. (1983). Recovery from short term intense exercise. Its relation to cappillary supply and blood lactate concentration. *European Journal of Applied Physiology,* **52**, 98-103.

Tortora, G. and Anagnostakos, N. (1989). *Principles of anatomy and physiology* 6th edition. Harper-Row, London.

Tsintzas, K., Williams, C., Constantin-Teodosiu, O., Hultman, E., Boobis, L. and Greenhaff, P. (1998). Carbohydrate ingestion improves oxidative ATP resynthesis in Slow twitch and Fast twitch muscle fibres during prolonged exercise. *Medicine and Science in Sport and Exercise,* **30**, s175.

Urhausen, A. and Kindermann, P (1992), Biochemical monitoring of training. *Clin J of Sports Medicine,* **2**, 52-61.

Van Beaumont, W., Underkofler, S. and Van Beaumont, S. (1981). Erythrocyte volume, plasma volume, and acid-base changes in exercise and heat dehydration. *Journal of Applied Physiology*, **50**, 1255-1262.

Vanhelder, W.P., Radomski, M.W., Goode, R.C. and Casey, K. (1985). Hormonal and metabolic responses to exercise of equal duration and external work output. *European Journal of Applied Physiology*, **54**, 337-342.

Vrijens, J., Pannier, J. and Bouckaert, J. (1982). Physiological profile of competitive road cyclists. *Journal of Sports Medicine,* **22**, 207-216.

Wagenmakers, A.J.M., Beckers, E.J., Brouns, F., Kuipers, H., Soeters, P.B., Van Der Vusse, G.J. and Saris, W.H.M. (1991). Carbohydrate supplementation, glycogen depletion, and amino acid metabolism during exercise. *American Journal of Physiology*, **260**, e833-e890.

Wahlund, J. (1948). Determination of the physical working capacity. *Acta Med Scand,* **132**, 215.

Wahren, J., Felig, P., Hendler, R. and Ahlborg, G. (1973). Glucose and amino acid metabolism during recovery after exercise. *Journal of Applied Physiology,* **34**, 838-845.

White, D. and Thorston, J. (1972). Phosphate starvation and the non linear dynamics of insect fibrillar flight muscle. *J Gen Physiol,* **60**, 307-336.

Wilkerson, J., Gutin, B. And Horvath, S. (1977). Exercise induced changes in red cell and plasma volume in man. *Medicine and Science in Sport and Exercise,* **9**, 155-158.

Withers, R.T., Sherman, W.M., Clarke, D.G., Esselbach, S.R., Nolan, S.R., Mackay, M.H. and Brinkman, M. (1991). Muscle metabolism during 30, 60, and 90 s of maximal cycling on an air braked ergometer. *European Journal of Applied Physiology,* **63**, 354-362.

Xu, F. and Rhodes, E. C. (1999) Oxygen Uptake Kinetics During Exercise. *Sports Medicine,* **27**, 313-327.

Yaspelkis, B., Patterson, J., Anderlan, P., Ding, Z. and Ivy, J. (1993). CHO Supplementation spares muscle glycogen during variable intensity exercise. *Journal of Applied Physiology,* **75**, 1477-1485.

Appendix 1. Raw data from Chapter 9
Appendix 1(i). MMP data

trial 1	trial 2	age (years)	mass (kg)	height (m)	category	VO2max	ml.kg
380	391	21	70.1	1.86	3	5.06	72.2
402	393	23	76.3	1.78	1	5.3	69.5
376	381	51	72.5	1.76	3	4.83	66.6
406	397	19	80.4	1.83	1	5.28	65.7
306	335	27	60	1.7	2	3.89	64.8
384	380	25	75.3	1.84	1	5.24	69.6
425	433	33	67.8	1.74	1	5.28	77.9
362	363	23	75	1.72	1	5.3	70.7
428	411	40	75.9	1.75	1	5.78	76.2
347	345	31	72.5	1.75	3	4.58	63.2

Appendix 1(ii). Power data

power	60	65	70	75	80	85	100
1	229	248	267	286	305	324	381
2	241	261	281	302	322	342	402
3	244	264	284	305	325	345	406
4	234	254	273	293	312	332	390
5	201	218	235	251	268	285	335
6	230	250	269	288	307	326	384
7	260	281	303	325	346	368	433
8	218	236	254	272	290	309	363
9	208	226	243	260	278	295	347
10	257	278	300	321	342	364	428

Appendix (iii). Heart rate data

heart rate	60	65	70	75	80	85	100
1	135	135	141	147	156	164	171
2	155	159	179	178	187	191	190
3	147	151	149	165	165	179	195
4	141	145	152	161	175	177	204
5	159	162	173	187	194	202	198
6	141	148	165	161	164	178	194
7	145	157	159	173	174	180	186
8	142	153	168	172	172	176	202
9	141	153	160	173	173	174	188
10	150	156	159	170	173	176	181

B[Lac⁻] analysis method	Power output
Visual Interpretation of threshold	235 watts
4 mmol.l⁻¹	239 watts
Data point preceding a 1 mmol.l⁻¹ rise in lactate concentration	235 watts
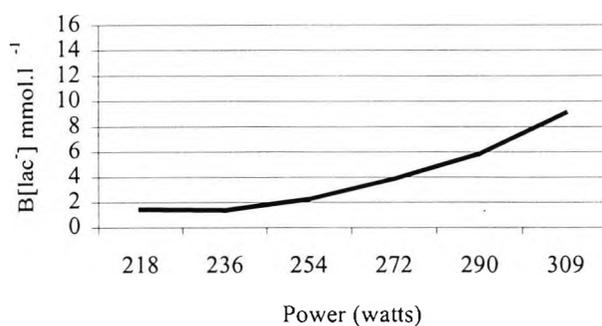
B[Lac⁻] analysis method	Power output
Visual Interpretation of threshold	236 watts
4 mmol.l⁻¹	272 watts
Data point preceding a 1 mmol.l⁻¹ rise in lactate concentration	236 watts
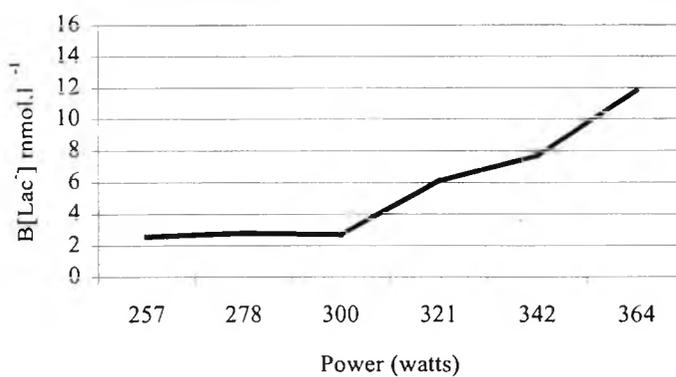
B[Lac⁻] analysis method	Power output
Visual Interpretation of threshold	300 watts
4 mmol.l⁻¹	310 watts
Data point preceding a 1 mmol.l⁻¹ rise in lactate concentration	300 watts
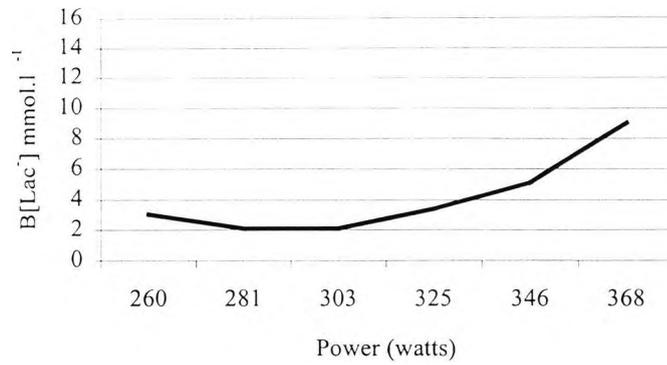
B[Lac⁻] analysis method	Power output
Visual Interpretation of threshold	303 watts
4 mmol.l⁻¹	333 watts
Data point preceding a 1 mmol.l⁻¹ rise in lactate concentration	303 watts
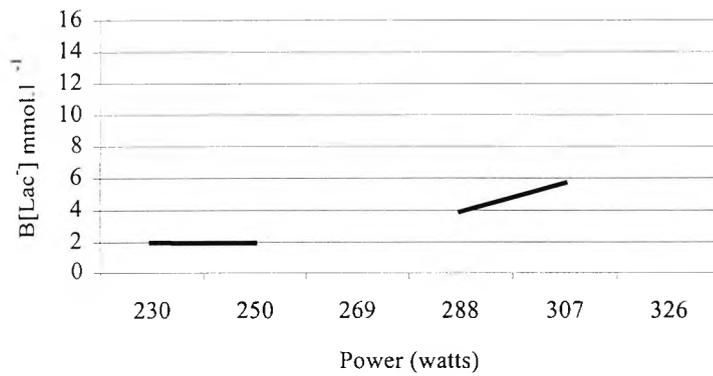
B[Lac⁻] analysis method	Power output
Visual Interpretation of threshold	Not determined
4 mmol.l⁻¹	288 watts
Data point preceding a 1 mmol.l⁻¹ rise in lactate concentration	Not determined
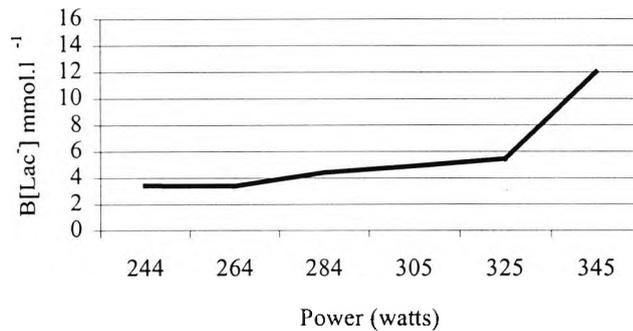
B[Lac⁻] analysis method	Power output
Visual Interpretation of threshold	264 watts
4 mmol.l⁻¹	274 watts
Data point preceding a 1 mmol.l⁻¹ rise in lactate concentration	325 watts

174
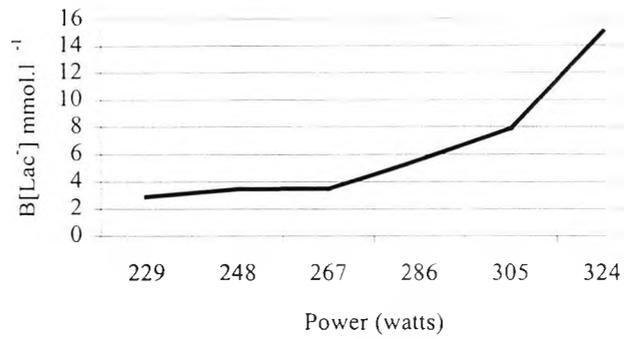
B[Lac⁻] analysis method	Power output
Visual Interpretation of threshold	267 watts
4 mmol.l^{-1}	272watts
Data point preceding a 1 mmol.l^{-1} rise in lactate concentration	267 watts

B[Lac⁻] analysis method	Power output
Visual Interpretation of threshold	235 watts
4 mmol.l^{-1}	239 watts
Data point preceding a 1 mmol.l^{-1} rise in lactate concentration	235 watts
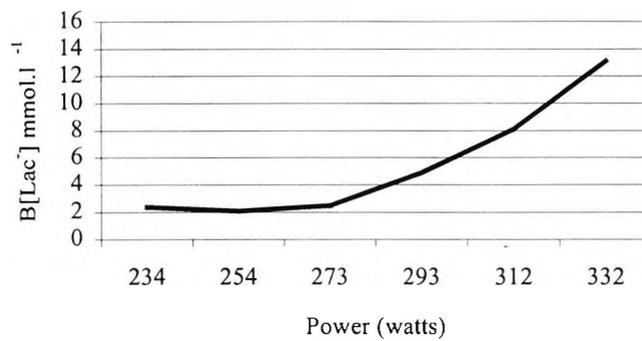
B[Lac⁻] analysis method	Power output
Visual Interpretation of threshold	273 watts
4 mmol.l^{-1}	283 watts
Data point preceding a 1 mmol.l^{-1} rise in lactate concentration	273 watts
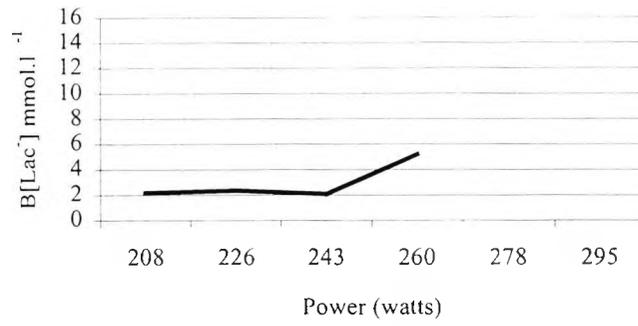
B[Lac⁻] analysis method	Power output
Visual Interpretation of threshold	243watts
4 mmol.l⁻¹	254watts
Data point preceding a 1 mmol.l⁻¹ rise in lactate concentration	243 watts

Appendix 2 Raw and smoothed data competitive cycle races

File   Edit   View   Powercontrol   Window   Help

Power [watts] Speed [km/h]   Training   Cadence [rpm] Heart rate [bpm]

1000
60.0

875
52.5

750
45.0

625
37.5

500
30.0

375
22.5

250
15.0

125
7.5

0
0.0

0:30:00   1:00:00 h

200
200

180
180

160
160

140
140

120
120

100
100

80
80

60
60

25/07/95 18:52-19:57 DAC/R 1:05h 1060kJ 41km DAMIAN COLEMAN LYDDEN MOTOR CURCUIT   Smoothing: 0%

\(0) File \(1) R

19:41:35 (+54:24) 279 watts 189 bpm 35.0 km/h 89 rpm

File   Edit   View   Powercontrol   Window   Help

Power [watts] Speed [km/h]   Training   Cadence [rpm] Heart rate [bpm]

1000
60.0

875
52.5

750
45.0

625
37.5

500
30.0

375
22.5

250
15.0

125
7.5

0
0.0

0:30:00   1:00:00 h

200
200

180
180

160
160

140
140

120
120

100
100

80
80

60
60

25/07/95 18:52-19:57 DAC/R 1:05h 1060kJ 41km DAMIAN COLEMAN LYDDEN MOTOR CURCUIT   Smoothing: 0:01:00

\(0) File \(1) R

19:47:08 (+59:57) 173 watts 188 bpm 39.1 km/h 94 rpm

184

File   Edit   View   Powercontrol   Window   Help

Power [watts] Speed [km/h]   Training   Cadence [rpm] Heart rate [bpm]

07/05/95 10:29-11:49 JN/H 1:20h 1263kJ 50km jason nind tour of lancs   Smoothing: 0%

[0] File \ (1) H \ (2) Y

11:36:14 [+1:09:06] 172 bpm 65.7 km/h

SRM Training System - [07/05/95 10:29-11:49 JN/H 1:20h 1263kJ 50km jason nind

File   Edit   View   Powercontrol   Window   Help

Power [watts] Speed [km/h]   Training   Cadence [rpm] Heart rate [bpm]

07/05/95 10:29-11:49 JN/H 1:20h 1263kJ 50km jason nind tour of lancs   Smoothing: 0:01:00

[0] File \ (1) H \ (2) Y

10:39:00 [+11:52] 371 watts 137 bpm 32.6 km/h 86 rpm

185

12/02/95 11:56-14:24 JN 2:14h 1597kJ 27km JASON NIND PERFS PEDAL RACE 45 MILES 5TH 12.2.95

12/02/95 11:56-14:24 JN 2:14h 1597kJ 27km JASON NIND PERFS PEDAL RACE 45 MILES 5TH 12.2.95

14:01:59 (+2:05:34) 195 watts 164 bpm 97 rpm

190

Appendix 2. Power output and course Profiles
Appendix 2(xxxv). Power output and course profile from one lap of the Forestside road race circuit
I.   Figure A

II. Figure B

Distance (km)
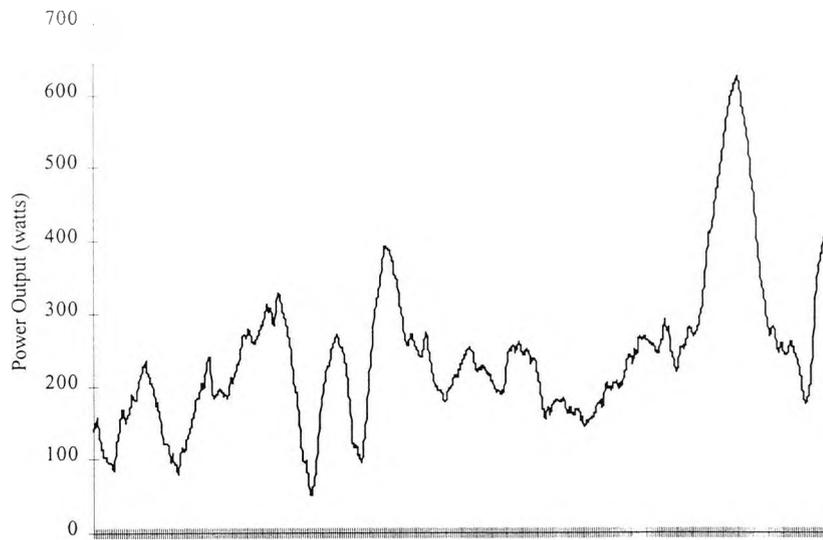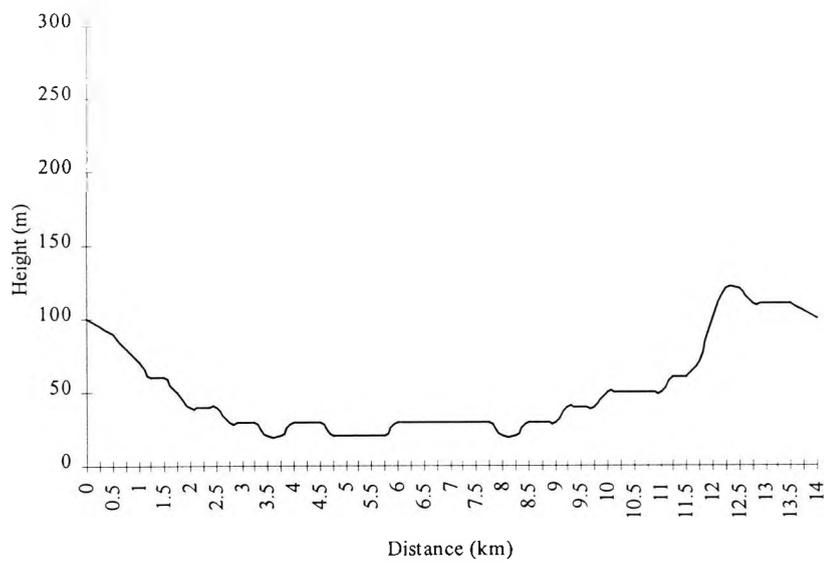
Appendix 2(xxxvi). Power output and course profile from one lap of the Godmersham road race circuit

I. Figure C
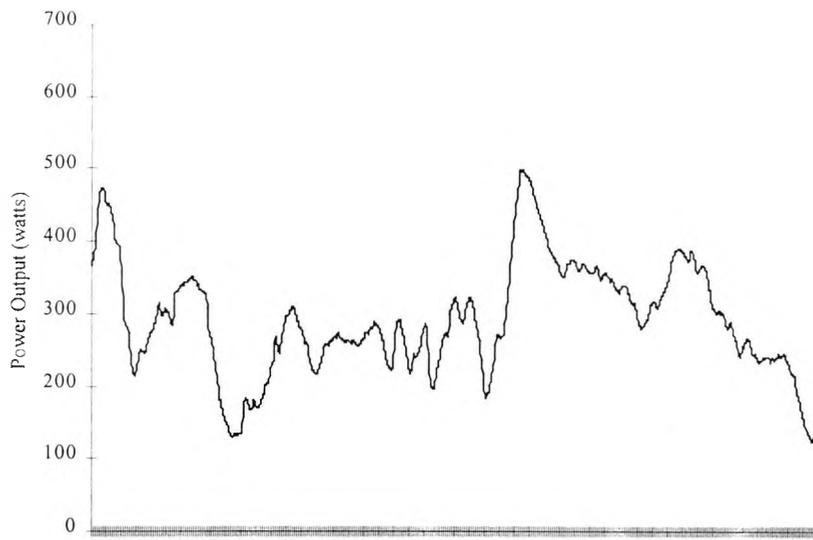
II. Figure D
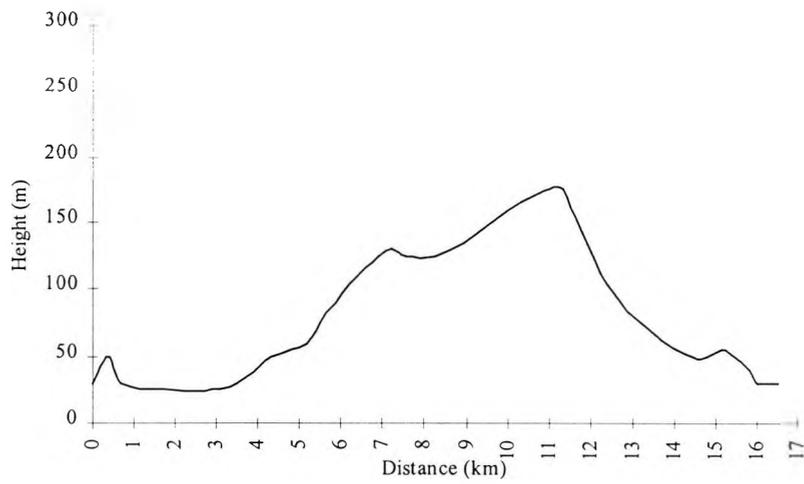
Appendix 2(xxxvii). Power output and course profile from one lap of the Frant road race circuit

I. Figure E
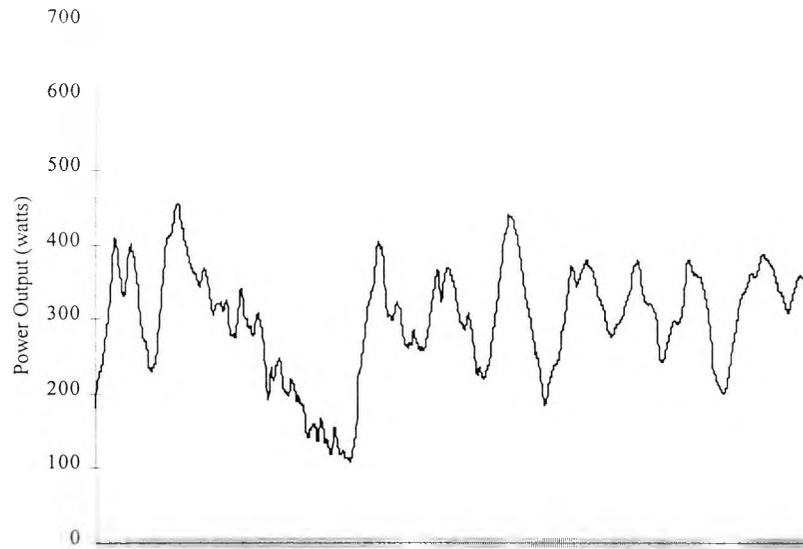
II. Figure F
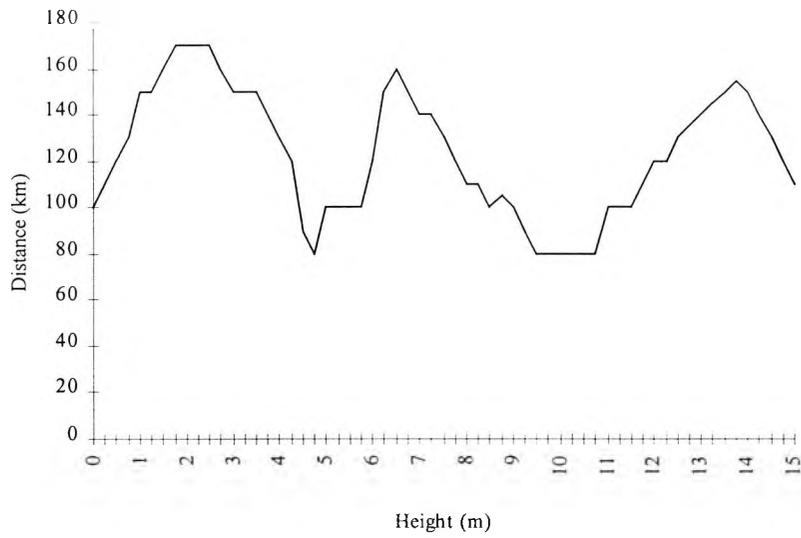
Appendix 3. Raw data from chapter 11
Appendix 3(i). MMP test data

Subject	MMP	HR MAX	B[Lac] T	B[Lac] 5POST
1	365	202	7.5	9.54
2	370	192	9.33	14.4
3	383	191	7.06	10.5
4	343	190	6.65	9.53
5	339	190	6.38	13.3
6	362	193	6.11	13.2
7	369	195	4.69	9.43
8	324	185	3.3	6.8

Appendix 3(ii). Hill climb results

Subject	time 6km	6km w·min^{-1}	time 1km	1km w·min^{-1}	% MMP	W·kg	W·kg$^{-0.67}$	W·(KG+C)$^{-1}$
1	15.08	330	3.85	391.4	90.41	5.45	21.82	4.72
2	15.38	329	4	403.1	88.92	5.48	22.01	4.69
3	15.63	368.4	4.07	464.1	96.19	4.77	20.28	4.19
4	15.88	337.6	4.15	423.9	98.43	4.76	19.54	4.12
5	16.18	308.9	4.05	393.8	91.12	4.93	19.91	4.24
6	16.83	321.7	4.25	415.2	88.87	4.87	20.19	4.19
7	16.95	328.3	4.42	408.2	88.97	4.66	19.74	4.1
8	18.43	317.5	4.72	390.9	97.99	4.13	17.43	3.6

Appendix 3(iii). Hill climb study correlation matrix

	MMP	time 6km	6km w·min^{-1}	time 1km	1km w·min^{-1}	% MMP	W·kg^{-1}	W·kg$^{-0.67}$	W·(kg+C)	HR max	B[Lac] T	B[Lac] 5POST
MMP	1.00											
time 6km	-0.61	1.00										
6km w·min^{-1}	0.66	-0.43	1.00									
time 1km	-0.52	0.97	-0.27	1.00								
1km w·min^{-1}	0.57	-0.27	0.90	-0.16	1.00							
% MMP	-0.46	0.27	0.36	0.35	0.35	1.00						
W·kg^{-1}	0.54	-0.87	0.06	-0.89	-0.11	-0.61	1.00					
W·kg-0.67	0.71	-0.89	0.21	-0.88	0.05	-0.63	0.98	1.00				
W·(kg+C)$^{-1}$	0.60	-0.89	0.12	-0.90	-0.07	-0.61	0.99	0.99	1.00			
HR max	0.58	-0.62	0.10	-0.63	-0.13	-0.60	0.71	0.74	0.75	1.00		
B[Lac] T	0.57	-0.91	0.31	-0.89	0.19	-0.35	0.91	0.90	0.89	0.42	1.00	
B[Lac] 5POST	0.37	-0.49	-0.14	-0.57	0.03	-0.64	0.64	0.63	0.60	0.13	0.70	1.00

Appendix 4. Raw data from chapter 12
Appendix 4(i). Physiological responses to level cycling

LEVEL	Watts· min⁻¹	hr	$\dot{V}O_2$ l·min⁻¹	$\dot{V}O_2$ ml·kg·min⁻¹	$\dot{V}CO_2$ l·min⁻¹	$\dot{V}E$ l·min⁻¹	rer	kcal·min⁻¹	GME
1	283	138.2	3.38	41.5	3.16	61.8	0.94	16.81	24.19
2	269	140	3.13	41.75	2.88	63.8	0.92	15.49	25.15
3	244	160	3.37	53.5	3.38	65.55	0.92	16.72	20.31
4	242	147.1	3.24	43.75	3.11	64.2	0.97	16.30	21.09
5	216	132.7	2.33	34.5	2.34	42.7	0.92	11.61	26.64
6	242	158	3	40	2.77	58	0.92	14.69	23.40

Appendix 4(ii). Physiological responses to graded cycling

GRADED	Watts· min⁻¹	hr	$\dot{V}O_2$ l·min⁻¹	$\dot{V}O_2$ ml.kg·min⁻¹	$\dot{V}CO_2$ l·min⁻¹	$\dot{V}E$ l·min⁻¹	rer	kcal·min⁻¹	GME
1	291	133.4	3.17	38.9	2.94	57.4	0.92	15.68	25.94
2	275	143.8	3.06	40.8	2.90	65	0.95	15.25	25.55
3	238	155	2.83	45	2.50	52.7	0.88	13.88	24.45
4	245	144.58	2.89	39	2.50	51.7	0.88	14.10	24.37
5	225	135.18	2.60	38.5	2.34	45.4	0.89	12.83	24.11
6	238	148	2.88	38.5	2.54	56	0.88	14.14	24.31

Appendix 5. Raw data from chapter 14.
Appendix 5(i). Subject achievements.

Subject	British Cycling Federation Category	Achievements (Road Racing)	Achievements (Time Trialing)
1	E	1st Tour of Warwickshire 1997. 2nd Tour of the North 1997, 6th Divisional Championships 1996, (5th 1997). Ridden for Divisional team and represented England in International stage races. Won many local road races and time trials.	National BBAR* Team 1995. Won many local time trials 10miles 20min 11sec 25miles 51min 24sec 50miles 1hr 47min sec 100miles 3hr 48min sec 12hour 275 miles
2	1	5th Tour of the Mendips 1997. 11th Tour of the North 1997. 13th Divisional Championships 1996. Top 10 finishes in other local road races (1 win).	Won local time trials, many top 10 finishes in local time trials. 10miles 20min 50sec 25miles 53min 50sec 50miles 1hr 48min 12sec 100miles 3hr 49min 14sec 12hours 270 miles Top 10 in BBAR 1998.
3	2	11th Divisional Championships 1997. Top 10 places in local road races.	Top 10 finishes in local time trials. 10miles 20min 42sec 25miles 53min 56sec 50miles 1hr 50min 50sec 100miles 4hr 27min sec
4	1	1st World Veterans road race championship 2000 2nd Divisional Championships 1996, (4th 1997). Won many local road races.	National 24 hour time trial champion 1996. Silver medalist 12hour championships 1994 & 1995. National BBAR* Team 1995. Won many local time trials. 10miles 20min sec 25miles 53min sec 50miles 1hr 45min sec 100miles 3hr 45min sec 12hour 274miles
5	2	9th Divisional Championships 1996 (7th 1997). Top 10 finishes in local road races.	Won many local time trials. 10miles 20min 56sec 25miles 53min 48sec 50miles 1hr 52min 30sec 100miles 4hr 24min sec
6	2	8th Divisional Championships 1996. Won Local road races, Top 10 finishes in many road races.	National 12hour champion (on three occasions) and national BBAR champion. Won many time trials.

Appendix 5(ii). Raw data from chapter 14.

	NH$_3$ (µmol.l^{-1})	NH$_3$ above warm up value (µmol.l.$^{-1}$)	Urea (µmol.l^{-1})	Urea above warm up value (µmol.l^{-1})	Heart rate (beats.min^{-1})	Relative heart rate (% maximum)	B[Lac$^-$] (mmol.l^{-1})	Blood glucose (mmol.l^{-1})
RRG								
1	120.75	-1.25	5.1	-3	141.7994	74.63127	2.82875	4.705
2	225.6667	155.6667	10.175	1.875	153.0198	84.07682	4.84	5.4975
3	91.5	20.5	6.4	0.3	155.4397	79.71264	2.4675	5.74375
4	153.6667	29.66667	7.625	0.425	147.3333	77.54386	2.7025	4.4025
5	178.75	38.75	8.575	-0.325	149.5136	81.70141	4.78375	4.68
6	102.5	-3.5	7.85	0.05	160.656	81.13942	2.0355	4.085
RRP								
1	96.66667	35.66667	6.8	1.2	143.4269	75.48782	2.445	3.4
2	160	28	6.566667	0.566667	149.6232	82.21056	4.3475	4.49125
3	83	-41	6.133333	0.133333	157.8202	80.93345	2.885	4.90125
4	153	22	9.475	-1.725	141.7194	74.58918	2.51375	3.73875
5	214.75	66.75	7.975	-0.425	147.3906	80.54131	5.0425	4.08875
6	105.25	3.25	7.6	0.4	159.3509	80.48024	2.10625	4.67875
EPG								
1	196.6667	49.66667	4.966667	0.266667	133.2069	70.10892	1.2415	4.52625
2	202.5	118.5	8.8	1.8	152.5755	83.83267	4.215	6.65375
3	93.5	5.5	6.475	0.475	146.9342	75.35085	1.49	5.1625
4	247.25	59.25	11.175	1.275	149.0284	78.43598	1.746375	4.95
5	167	3	9.675	0.275	148.5223	81.15973	1.35	4.335
6	127.25	29.25	6.575	0.175	150.0006	75.75787	0.906625	4.385
EPP								
1	128.75	6.75	4.975	0.175	137.1475	72.18288	1.60625	3.92875
2	156	58	7.875	0.875	149.0358	81.88781	2.76375	4.4175
3	85.5	32.5	5.95	0.15	155.2259	79.60301	1.571875	4.585
4	225.75	44.75	9.175	0.975	145.017	76.32473	2.04	3.915
5	175	50	9.075	-0.025	146.9467	80.29877	1.5825	4.09875
6	125.75	13.75	7.725	0.625	155.4685	78.51944	0.839125	4.37875

Appendix 5(ii). Raw data from chapter 14 (cont).

	$\dot{V}O_2$ (l.min^{-1}) mid lap	% $\dot{V}O_2$max mid lap	$\dot{V}O_2$ (l.min^{-1}) matched mid lap	% $\dot{V}O_2$max matched mid lap	$\dot{V}O_2$ (l.min^{-1}) end of lap	% $\dot{V}O_2$max end of lap
RRG						
1	3.105556	52.54747	3.784167	64.02989	3.9105	66.16751
2	2.920833	60.85069	3.66875	76.43229	3.449	71.85417
3	2.497292	44.99625	2.960625	53.34459	3.139333	56.56456
4	2.127708	52.66605	2.645	65.4703	2.977	73.68812
5	2.781875	52.38936	3.42125	64.43032	3.532	66.51601
6	2.400909	44.29722	2.930625	54.07057	3.389	62.52768
RRP						
1	2.899167	49.05527	3.6575	61.88663	2.9455	49.83926
2	2.926875	60.97656	3.775	78.64583	3.598	74.95833
3	2.28	41.08108	3.004167	54.12913	3.411	61.45946
4	2.161667	53.5066	2.67875	66.30569	2.9815	73.7995
5	2.461667	46.35907	3.44625	64.90113	3.4955	65.82863
6	2.382955	43.96595	2.905	53.59779	3.299333	60.87331
EPG						
1	3.564583	60.31444	3.5975	60.8714	3.4005	57.53807
2	3.7575	78.28125	3.8825	80.88542	3.564667	74.26389
3	3.068125	55.28153	2.995	53.96396	3.474667	62.60661
4	2.634167	65.20215	2.63375	65.19183	2.7095	67.06683
5	3.510417	66.10954	3.53625	66.59605	3.365333	63.37728
6	2.912818	53.74203	2.922083	53.91298	2.9445	54.32657
EPP						
1	3.644583	61.66808	3.69875	62.5846	3.4015	57.55499
2	3.675083	76.56424	3.743333	77.98611	3.515	73.22917
3	3.154167	56.83183	3.070625	55.32658	3.277	59.04505
4	2.622083	64.90305	2.64375	65.43936	2.59	64.10891
5	3.427139	64.54122	3.5	65.91337	3.35	63.08851
6	3.268864	60.31114	3.266875	60.27445	3.273167	60.39053

Appendix 5(ii). Raw data from chapter 14 (cont).

	$\dot{V}CO_2$ (l.min⁻¹) mid lap	$\dot{V}CO_2$ (l.min⁻¹) matched mid lap	$\dot{V}CO_2$ (l.min⁻¹) end of lap	RER mid lap	RER matched mid lap	RER end of lap
RRG						
1	2.1275	2.593125	3.814	0.912761	0.913924	0.975219
2	2.612917	3.314375	3.5185	0.894555	0.903417	1.020374
3	2.236875	2.57625	3.426	0.894942	0.868815	1.097578
4	1.811667	2.260625	2.82	0.851524	0.854863	0.947068
5	2.38	2.8825	3.642	0.855542	0.842517	1.030752
6	2.196042	2.7125	3.4285	0.915668	0.926064	1.011202
RRP						
1	2.623958	3.259375	3.575	0.90529	0.891939	0.951008
2	2.622083	3.45	3.7035	0.896183	0.914653	1.029365
3	2.035	2.639167	3.396	0.894575	0.878654	0.995848
4	1.82375	2.241875	2.837	0.844316	0.837592	0.952002
5	2.435417	2.945	3.6035	0.896505	0.854531	1.030976
6	2.144167	2.62	3.310667	0.887515	0.902932	1.003452
EPG						
1	3.317292	3.34625	3.156667	0.930982	0.931344	0.936346
2	3.496944	3.595	3.287	0.927668	0.922071	0.931347
3	2.787292	2.73625	2.883	0.908629	0.934948	0.825543
4	2.34625	2.350625	2.4045	0.890674	0.892486	0.887671
5	3.109896	3.164375	2.986	0.885887	0.894943	0.887271
6	2.694911	2.73125	2.6755	0.926149	0.935301	0.908925
EPP						
1	3.3375	3.420625	3.0815	0.915971	0.925324	0.906108
2	3.547292	3.613125	3.339	0.965313	0.965715	0.950288
3	2.842292	2.744375	2.895	0.901423	0.894388	0.886222
4	2.258958	2.280625	2.234	0.861553	0.86264	0.862723
5	3.049381	3.2	3.0335	0.88685	0.922656	0.906256
6	2.720625	2.705	2.734667	0.832267	0.829215	0.835309

Appendix 5(ii). Raw data from chapter 14 (cont).

	Economy mid lap	Economy matched mid lap	Economy end of lap	GME mid lap	GME matched mid lap	GME end of lap
RRG						
1	82.47909	75.19494	74.55693	23.95667	21.8	0
2	83.8643	79.03144	80.98195	24.4375	21.585	0
3	87.01333	84.32448	78.58066	25.3625	23.8825	0
4	91.31309	86.73381	74.36683	26.895	24.0625	0
5	83.39465	79.54721	74.86864	24.5225	22.2	0
6	97.29061	92.40839	78.40636	28.2	25.59	0
RRP						
1	88.23219	83.58529	74.49698	25.635	22.705	0
2	83.83737	77.26758	77.75301	24.4275	20.995	0
3	96.32046	260.0203	72.32159	28.07	23.46	0
4	89.917	85.6343	74.27259	26.5175	23.8425	0
5	98.20523	84.30236	75.67556	28.55	21.985	0
6	97.97776	94.15178	80.52854	27.97333	25.99	0
EPG						
1	79.88504	79.36834	83.69197	23.073	22.915	24.34667
2	72.49866	70.16843	76.44256	20.965	20.32	22.06
3	78.42301	80.33794	69.25354	22.7675	23.17333	20.53333
4	82.05385	82.06113	79.85413	23.92675	23.9275	23.31
5	73.51639	72.99711	76.69169	21.44	21.22	22.36
6	89.07795	88.74478	88.04048	25.7425	25.6	25.5725
EPP						
1	78.04236	76.9248	83.66283	22.6187	22.2525	24.3075
2	74.12712	72.77787	77.53702	21.2292	20.83333	22.2825
3	76.30017	78.40641	73.63899	22.19429	22.8475	21.49
4	82.38806	81.70659	83.44644	24.2035	24.0175	24.5125
5	75.33747	73.72198	77.11072	21.9456	21.28	22.365
6	79.29804	79.3997	79.25588	23.48998	23.56	23.4475

Appendix 5(ii). Raw data from chapter 14 (cont).

	% fresh time	Body mass (kg)	MMP (watts.min^{-1})	Performance Power (watts.min^{-1})	Decrement in power (watts.min^{-1})	Time (min)
RRG						
1	106.5814	89.9	473	522	-35	4.583
2	116.442	87.8	452	433	-71	5.21666
3	108.4337	86.8	397	423	-35	5.4
4	107.0957	80.4	360	388	-28	5.48333
5	108.9888	76	436	373	-34	4.85
6	111.6825	86.2	432	425	-50	5.416666
RRP						
1	122.4884	89.9	473	454	-103	5.267
2	126.1161	87.8	452	400	-104	5.65
3	110.4418	86.8	397	415	-43	5.5
4	116.5352	80.4	360	357	-59	5.96666
5	111.609	76	436	364	-43	4.9666
6	115.4639	86.2	432	411	-64	5.6
EPG						
1	105.4186	89.9	473	528	-29	4.53333
2	102.6786	87.8	452	491	-13	4.6
3	104.4177	86.8	397	439	-19	5.2
4	111.002	80.4	360	374	-42	5.6833
5	109.6629	76	436	371	-36	4.88
6	108.2474	86.2	432	438.4	-36.6	5.25
EPP						
1	112.0233	89.9	473	497	-60	4.817
2	106.7634	87.8	452	473	-31	4.783333
3	106.4257	86.8	397	431	-27	5.3
4	117.1875	80.4	360	355	-61	6
5	111.236	76	436	365	-42	4.95
6	118.2062	86.2	432	401	-74	5.733333

Appendix 5(ii). Raw data from chapter 14 (cont).

	% maximum heart rate @ 70% MMP	% maximum heart rate during performance test	Heart rate during last minute of performance test (beats^{-1})	B[Lac$^-$] post performance test (mmol.l^{-1})	Blood glucose post performance test (mmol.l^{-1})
RRG					
1	77.95666	92.92982	186.3333	8.25	4.12
2	91.47609	101.7879	188.9167	8.76	7.7
3	78.70445	92.86859	190	9.59	6.8
4	81.72515	98.50877	195.75	11.7	6.78
5	87.38616	96.05322	182.2787	12.9	6.65
6	85.0954	98.07526	201.3934	11	5.84
RRP					
1	79.56656	93.37461	191	6.55	5.41
2	89.47134	99.42631	185.6667	5.75	5.47
3	84.55678	93.03922	186.8333	13.5	6.17
4	78.0848	92.28801	183.0833	5.95	5.01
5	87.99636	96.714	181.377	10.6	5.53
6	89.46409	96.8543	197.1475	8.6	5.69
EPG					
1	75.82043	91.2218	97.36842	12.7	6.65
2	90.73359	100.4808	100.2289	7.98	9.74
3	76.97706	92.01243	96.5812	15.9	7.42
4	82.58772	98.25324	102.6754	15.8	7.02
5	86.77292	95.43021	97.61713	13.7	8.44
6	81.77048	91.68764	95.5539	12.1	5.9
EPP					
1	78.0805	92.0034	97.45614	8.28	4.8
2	87.45174	99.90361	101.6026	4.83	5.26
3	81.4305	95.33654	99.82906	10.5	6.29
4	79.57602	94.39798	98.90351	9.9	4.98
5	87.87796	97.36861	100.7256	9.73	5.92
6	83.43434	93.10008	95.94304	8.05	5.37

Appendix 5(iii). One subject data ($\dot{V}CO_2$)

Lap 1 — $\dot{V}CO_2$ ($l.min^{-1}$)

Even Power (g)	Even Power (p)	Road Race (g)	Road Race (p)
2.71		3.59	
2.98		3.54	
2.67	2.6	3.1	
2.6	3.08	3.27	
3.22	2.91	3.36	
2.9	3.02	3.17	
3.01	3.08	3.09	
3.09	3.19	3.15	
3.18	2.84	3.2	
2.94	2.64	2.91	
2.63	2.87	2.77	
2.7	2.92	2.89	
2.7	2.66	3.33	
2.91	2.88	4.03	
2.64	2.49	2.95	
2.72	2.97	2.16	
3.07	2.81	3.27	
2.98	3.15	4.16	3.44
2.88	2.75	4.85	4.34
2.78	2.65	4.58	4.79
2.73	2.81	4.41	4.04
2.78	2.67	3.53	3
2.79	2.65	2.22	2.03
2.44	2.79	1.7	1.76
2.2	2.75	1.78	1.63
2	2.9	1.49	1.51
2.14	2.93	1.48	1.42
2.45	2.84	1.58	1.49
2.79	3.15	1.51	1.44
2.55	3	1.92	1.84
3.12	2.56	2.43	1.96
2.96	2.7	2.49	2.21
2.91	2.84	2.6	2.39
2.78	2.73	2.73	2.65
2.89	2.87	2.72	2.6
2.8	2.38	2.82	2.87
2.75	2.38	3.06	2.82
2.83	2.63	2.17	
2.73	2.86	3.61	
2.53	2.59	4.04	
2.77	2.83	4.33	
2.62	2.68	4.8	
2.67	2.69	3.88	
2.65	2.81	4.21	
2.92	2.81	4.14	
2.62	3.06	4.18	
2.65	2.98	3.72	
2.75	2.76	3.03	
2.77	2.84	3.16	

Lap 2 — $\dot{V}CO_2$ ($l.min^{-1}$)

Even Power (g)	Even Power (p)	Road Race (g)	Road Race (p)
2.7	2.78	3.08	
2.61	3.07	2.9	
2.87	2.73	2.99	
2.65	2.78	3.27	
2.79	2.57	3.17	
2.56	2.92	3.01	
2.81	2.62	3.09	
2.77	3.01	3.07	
2.43	2.67	2.97	
2.93	2.63	2.65	
2.44	2.72	2.95	
2.33	2.83	3.33	
2.35	2.93	4.24	
2.46	2.67	3.16	
2.44	2.68	2.06	
2.84	2.6	3.02	
3.12	2.78	4.03	
2.92	2.75	4.89	
2.79	2.77	4.65	
2.72	2.88	4.36	
2.74	2.72	3.46	
2.62	2.67	2.16	
2.62	2.54	1.79	
2.47	2.43	1.67	
2.87	2.75	1.63	
2.69	2.68	1.34	
2.66	2.86	1.57	
2.82	2.84	1.44	
2.66	2.56	1.64	
2.5	2.68	2.07	1.31
2.52	2.71	2.2	1.76
2.64	2.76	2.55	2.1
2.6	2.68	2.55	2.29
2.72	2.63	2.77	2.61
2.72	2.96	2.84	2.63
2.79	3.02	2.74	2.54
2.62	2.75	2.77	2.74
2.79	2.84	2.53	1.57
2.67	2.67	3.26	3.24
2.77	2.68	4.47	3.38
2.83	2.72	3.74	4.27
2.68	2.65	3.95	4.17
2.66	2.71	4	4.01
3.02	2.61	3.66	4.48
2.84	2.65	4.66	4.06
2.9	2.78	3.51	4
2.77	2.45	3.22	3.09
2.59	2.58	3.02	2.99
2.69	2.69	3.03	2.78

Appendix 5(iii) One subject data ($\dot{V}CO_2$) (cont)

lap 3	$\dot{V}CO_2$ ($l.min^{-1}$)			Lap 4	$\dot{V}CO_2$ ($l.min^{-1}$)		
Even Power (g)	Even Power (p)	Road Race (g)	Road Race (p)	Even Power (g)	Even Power (p)	Road Race (g)	Road Race (p)
2.42		2.68	3.02	2.28	2.54	2.79	3.51
2.58		2.53	2.93	2.72	2.62	2.76	2.87
2.6		2.83	2.73	2.63	2.75	2.48	2.56
2.75		2.75	2.58	2.57	2.88	2.91	2.81
2.71		2.49	2.92	2.62	2.9	2.62	2.89
2.69		2.56	2.8	2.46	2.61	2.6	2.85
2.45		2.72	2.72	2.61	2.54	2.63	2.82
2.7		2.74	2.72	2.76	2.74	3.06	2.66
2.56		2.76	2.55	2.74	2.58	3.01	3.05
2.62		2.48	2.47	2.64	2.69	2.64	2.41
2.58		2.56	2.41	2.6	2.49	2.54	2.6
2.71		3.58	2.6	2.66	2.65	2.51	2.82
3.1		3.8	3.68	2.71	2.73	3.33	3.56
2.67		3.26	3.78	2.71	3.1	4.06	4.09
2.32		2.19	2.4	2.51	2.75	2.61	2.93
2.77		2.8	2.18	2.42	2.81	2.16	2.1
2.62		4.08	3.23	2.42	2.56	3.12	3.16
2.84		4.54	4	2.7	2.64	4.02	4.28
2.48		4.91	4.75	2.99	2.83	4.4	5.13
2.77		4.27	4.48	2.51	2.74	4.64	5.04
2.56		3.35	3.76	2.59	2.65	4.11	4.2
2.63		2.37	2.83	2.4	2.66	3.45	3.19
2.6		1.91	2	2.49	2.66	2.24	1.81
2.96		1.56	1.47	2.39	2.54	1.8	1.77
2.73		1.61	1.66	2.66	2.6	1.46	1.58
2.62		1.51	1.35	2.84	2.7	1.41	1.4
2.55		1.5	1.53	2.82	2.95	1.31	1.52
2.62		1.63	1.64		2.65	1.64	1.44
2.46		1.93	1.42		2.39	1.33	1.45
2.83		2	1.83		2.94	1.71	1.86
2.55		2.2	2.06		2.61	2.18	2.05
2.82		2.52	2.33		2.64	2.07	2.07
2.74		2.57	2.67	2.57	2.41	2.45	2.33
2.66		2.79	2.94	2.67	2.68	2.73	2.67
2.67		2.83	2.82	2.72	2.58	2.84	2.58
2.71		2.92	2.69	2.75	2.68	2.5	2.64
2.87		2.83	2.84	2.58	2.43	2.7	2.62
		2.39	2.03	2.48	2.62	2.25	2.06
2.44		2.99	3.21	2.67	2.84	3.64	2.93
2.62		3.67	4.08	2.49	2.84	4.08	3.59
2.69		3.65	3.66	2.67	2.65	3.78	3.28
2.55		4.15	3.85	2.72	2.72	4.38	3.84
2.7		4.07	3.7	2.59	2.61	3.44	3.58
2.64		3.83	4.11	2.64	2.63	3.93	3.64
2.56		4.11	4	2.71	2.67	4.1	3.59
2.41		3.54	3.56	2.67	2.66	3.41	3.52
2.69	2.93	2.89	3.32	2.39	2.7	3.28	2.94
2.74	2.71	2.96	2.92	2.54	2.66	2.7	3.03
2.76	2.66	3.1	3.06	2.54	2.57	2.81	2.8

Appendix 5(iv). One subject data ($\dot{V}O_2$)

Lap 1	$\dot{V}O_2$ (l.min^{-1})		
Even Power (g)	Even Power (p)	Road Race (g)	Road Race (p)
3.16		3.28	
2.77		2.87	
2.69	2.97	3.17	
3.44	3.55	3.31	
2.97	3.35	3.12	
3.1	3.48	3.13	
3.18	3.55	3.25	
3.22	3.65	3.28	
2.96	3.2	2.95	
2.68	2.99	2.86	
2.79	3.33	2.96	
2.78	3.4	3.54	
3.04	3.02	4.29	
2.71	3.36	2.94	
2.79	2.87	2.09	
3.15	3.45	3.14	
2.95	3.21	4.22	
2.73	3.58	4.86	3.82
2.81	3.03	4.3	4.6
2.8	2.99	4.07	4.66
2.85	3.31	3.29	3.8
2.89	3.16	2.2	2.88
2.48	3.14	1.69	2.07
2.5	3.24	1.8	1.8
2.46	3.25	1.55	1.7
2.29	3.36	1.59	1.61
2.6	3.38	1.71	1.52
2.84	3.22	1.6	1.62
2.58	3.61	2.07	1.57
3.26	3.45	2.63	2
3.02	2.95	2.72	2.2
2.89	3.16	2.83	2.49
2.82	3.32	2.99	2.65
3	3.17	3.01	2.91
2.88	3.38	3.06	2.83
2.84	2.79	3.28	3.12
2.48	2.79		2.97
3.03		2.27	
2.97	3.27	2.25	
2.81	3.49	3.7	
2.67	3.1	3.94	
2.91	3.39	3.84	
2.74	3.21	4.29	
2.8	3.21	3.45	
2.78	3.38	3.94	
3.12	3.39	3.9	
2.74	3.61	3.82	
2.74	3.57	3.5	
2.92	3.18	2.86	
2.92	3.35	3.23	

Lap 2	$\dot{V}O_2$ (l.min^{-1})		
Even Power (g)	Even Power (p)	Road Race (g)	Road Race (p)
2.83	3.78	3.22	
3.13	3.29	3.08	
2.9	3.37	3.18	
3	3.12	3.45	
2.67	3.54	3.32	
2.99	3.16	3.15	
2.88	3.65	3.23	
2.59	3.16	3.22	
3.18	3.15	3.13	
2.62	3.28	2.83	
2.44	3.36	3.19	
2.47	3.51	3.61	
2.63	3.14	4.56	
2.57	3.18	3.11	
3.02	3.18	2	
3.39	3.41	3	
3.03	3.29	4.21	
2.85	3.32	5.01	
2.81	3.38	4.39	
2.89	3.27	4.07	
2.74	3.21	3.26	
2.77	3.01	2.2	
2.64	2.94	1.91	
3.07	3.32	1.76	
2.84	3.25	1.74	
2.63	3.41	1.46	
2.94	3.4	1.7	
2.88	2.99	1.58	
3.02	3.2	1.8	
2.87	3.3	2.29	1.96
2.7	3.3	2.48	2.38
2.71	3.22	2.88	2.6
2.85	3.19	2.84	2.92
2.82	3.53	3.05	2.93
2.96	3.61	3.1	2.76
2.94	3.25	2.95	2.94
2.99	3.15	3.74	3.81
3.3	3.5	2.67	1.7
3	3.23	3.38	3.47
3.06	3.23	4.4	3.36
3.1	3.34	3.39	4.01
2.88	3.22	3.64	3.84
2.91	3.25	3.74	3.71
3.3	3.14	3.53	4.13
3.06	3.18	4.57	3.78
3.11	3.33	3.29	3.71
2.96	2.9	3.1	2.99
2.76	3.09	3.01	2.96
2.94	3.28	3.19	2.89

Appendix 5(iv) One subject data ($\dot{V}O_2$) (Cont)

lap 3	$\dot{V}O_2$ (l.min⁻¹)		
Even Power (g)	Even Power (p)	Road Race (g)	Road Race (p)
3.01		2.91	3.22
2.95		2.75	2.91
3.07		3.15	2.78
3.03		3.01	3.18
2.95		2.71	2.97
2.7		2.81	2.87
2.99		2.93	2.86
2.79		2.93	2.7
2.85		2.64	2.67
2.81		2.74	2.62
2.93		3.9	2.82
3.33		4.01	4.05
2.89		3.28	3.96
2.55		2.18	2.38
3.1		2.83	2.13
2.91		4.29	3.28
3.06		4.6	4.26
2.62		4.76	4.82
3.03		3.97	4.24
2.82		3.2	3.55
2.9		2.41	2.8
2.85		2.06	2.09
3.26		1.68	1.54
2.92		1.71	1.76
2.82		1.64	1.43
2.74		1.66	1.67
2.86		1.81	1.81
2.69		2.15	1.59
3.11		2.2	2.09
2.84		2.49	2.32
3.09		2.88	2.71
3.08		2.86	3.05
3		3.11	3.26
2.86		3.1	3.04
2.87		3.21	2.88
2.98		3.08	3.04
3.13		3.7	3.74
3.18		2.62	2.27
2.78		3.22	3.44
2.98		3.77	4.13
3.04		3.49	3.43
2.83		4.06	3.66
3.01		3.89	3.52
2.97		3.52	4.02
2.82		3.95	3.92
2.66		3.41	3.38
3.08	3.37	2.9	3.31
3.12	3.23	3.06	2.96
3.06	3.23	3.4	3.26

Lap 4	$\dot{V}O_2$ (l.min⁻¹)		
Even Power (g)	Even Power (p)	Road Race (g)	Road Race (p)
2.73	3.36	3.06	2.9
3.23	3.39	2.98	2.65
3.06	3.47	2.73	3.09
2.94	3.61	3.28	3.19
3.01	3.56	2.9	3.09
2.82	3.17	2.89	3.03
3	3.1	2.97	2.89
3.18	3.4	3.46	3.2
3.13	3.19	3.22	3.3
3	3.29	2.81	2.58
2.94	3.08	2.84	2.84
2.95	3.24	2.78	3.12
3.02	3.39	3.71	3.93
3.02	3.76	4.41	4.35
2.72	3.15	2.67	2.95
2.75	3.29	2.23	2.06
2.77	3.15	3.24	3.17
3.13	3.22	4.18	4.62
3.33	3.42	4.46	5.21
2.79	3.24	4.56	4.76
2.9	3.18	3.97	3.87
2.76	3.3	3.38	3.08
2.89	3.31	2.26	1.83
2.76	3.09	1.9	1.69
3.06	3.21	1.58	1.52
3.19	3.31	1.56	1.66
	3.6	1.5	1.64
	3.21	1.87	1.66
	2.92	1.52	1.8
	3.62	1.97	2.2
	3.2	2.57	2.43
	3.18	2.43	2.78
2.83	2.93	2.83	3.11
3.06	3.26	3.12	2.94
3.12	3.19	2.81	2.99
3.12	3.28	3.05	3
2.93	2.98	3.85	3.87
2.97	3.35	2.55	2.31
3.21	3.53	4.07	3.22
2.91	3.5	4.2	3.78
3.09	3.27	3.62	3.23
3.2	3.36	4.21	3.87
3.02	3.21	3.29	3.6
3.08	3.22	3.88	3.58
3.12	3.29	4.11	3.59
3.08	3.26	3.32	3.45
2.77	3.27	3.31	3.01
2.91	3.26	2.8	3.22
3	3.12	3.05	3.06

Appendix 5(v) One subject data (RER)

Lap 1	RER		
Even Power (g)	Even Power (p)	Road Race (g)	Road Race (p)
0.94		1.08	
0.96		1.08	
0.97	0.87	1.03	
0.94	0.87	1.02	
0.98	0.87	1.02	
0.97	0.87	0.99	
0.97	0.87	0.97	
0.99	0.89	0.98	
0.99	0.88	0.99	
0.98	0.86	0.97	
0.97	0.86	0.98	
0.97	0.88	0.94	
0.96	0.86	0.94	
0.97	0.87	1.00	
0.97	0.86	1.03	
0.97	0.88	1.04	
1.01	0.88	0.99	
1.05	0.91	1.00	0.90
0.99	0.89	1.07	0.94
0.98	0.85	1.08	1.03
0.98	0.84	1.07	1.06
0.97	0.84	1.01	1.04
0.98	0.86	1.01	0.98
0.88	0.85	0.99	0.98
0.81	0.86	0.96	0.96
0.93	0.87	0.93	0.94
0.94	0.88	0.92	0.93
0.98	0.87	0.94	0.92
0.99	0.87	0.93	0.92
0.96	0.87	0.92	0.92
0.98	0.85	0.92	0.89
1.01	0.86	0.92	0.89
0.99	0.86	0.91	0.90
0.96	0.85	0.90	0.91
0.97	0.85	0.92	0.92
0.97	0.85	0.93	0.92
0.95	0.80	0.96	
0.97	0.82	0.98	
0.95	0.84	1.03	
0.95	0.83	1.13	
0.96	0.83	1.12	
0.95	0.84	1.12	
0.95	0.83	1.07	
0.94	0.83	1.06	
0.96	0.85	1.09	
0.97	0.83	1.06	
0.94	0.87	1.06	
0.95	0.85	0.98	

Lap 2	RER		
Even Power (g)	Even Power (p)	Road Race (g)	Road Race (p)
0.92	0.81	0.96	
0.92	0.83	0.94	
0.91	0.82	0.94	
0.93	0.82	0.95	
0.96	0.82	0.95	
0.94	0.83	0.96	
0.96	0.82	0.96	
0.94	0.84	0.95	
0.92	0.83	0.95	
0.93	0.83	0.94	
0.95	0.84	0.92	
0.95	0.83	0.92	
0.94	0.85	0.93	
0.95	0.84	1.02	
0.94	0.82	1.03	
0.92	0.82	1.01	
0.96	0.84	0.96	
0.98	0.83	0.98	
0.97	0.85	1.06	
0.95	0.83	1.07	
0.96	0.83	1.06	
0.95	0.84	0.98	
0.94	0.83	0.94	
0.93	0.83	0.95	
0.95	0.82	0.94	
1.01	0.84	0.92	
0.96	0.84	0.92	
0.92	0.86	0.91	
0.83	0.84	0.91	
0.88	0.82	0.90	0.90
0.98	0.84	0.89	0.88
0.96	0.83	0.89	0.88
0.95	0.82	0.90	0.89
0.96	0.84	0.91	0.90
0.94	0.84	0.92	0.92
0.89	0.85	0.93	0.93
0.85	0.81	0.95	0.92
0.89	0.83	0.96	0.93
0.91	0.83	1.02	1.01
0.91	0.81	1.10	1.06
0.93	0.82	1.09	1.09
0.91	0.83	1.07	1.08
0.92	0.83	1.04	1.08
0.93	0.83	1.02	1.07
0.93	0.83	1.07	1.08
0.94	0.84	1.04	1.03
0.94	0.83	1.00	1.01
0.91	0.82	0.95	0.96

Appendix 5(v) One subject data (RER) (Cont)

lap 3	RER			Lap 4	RER		
Even Power (g)	Even Power (p)	Road Race (g)	Road Race (p)	Even Power (g)	Even Power (p)	Road Race (g)	Road Race (p)
0.92	0.81	0.96		0.84	0.77	0.93	1.08
0.92	0.83	0.94		0.86	0.79	0.91	0.83
0.91	0.82	0.94		0.87	0.80	0.89	0.88
0.93	0.82	0.95		0.87	0.81	0.90	0.94
0.96	0.82	0.95		0.87	0.82	0.90	0.94
0.94	0.83	0.96		0.87	0.82	0.89	0.98
0.96	0.82	0.96		0.87	0.81	0.88	0.83
0.94	0.84	0.95		0.88	0.81	0.93	0.92
0.92	0.83	0.95		0.88	0.82	0.94	0.93
0.93	0.83	0.94		0.88	0.81	0.89	0.92
0.95	0.84	0.92		0.90	0.82	0.90	0.90
0.95	0.83	0.92		0.90	0.81	0.90	0.91
0.94	0.85	0.93		0.90	0.82	0.92	0.94
0.95	0.84	1.02		0.92	0.87	0.98	0.99
0.94	0.82	1.03		0.88	0.85	0.97	1.02
0.92	0.82	1.01		0.87	0.81	0.96	1.00
0.96	0.84	0.96		0.86	0.82	0.96	0.93
0.98	0.83	0.98		0.90	0.83	0.99	0.98
0.97	0.85	1.06		0.90	0.85	1.02	1.06
0.95	0.83	1.07		0.89	0.83	1.04	1.09
0.96	0.83	1.06		0.87	0.81	1.02	1.04
0.95	0.84	0.98		0.86	0.80	0.99	0.99
0.94	0.83	0.94		0.87	0.82	0.95	1.05
0.93	0.83	0.95		0.87	0.81	0.92	1.04
0.95	0.82	0.94		0.89	0.82	0.90	0.84
1.01	0.84	0.92			0.82	0.87	0.93
0.96	0.84	0.92			0.83	0.88	0.87
0.92	0.86	0.91			0.82	0.88	0.81
0.83	0.84	0.91			0.81	0.87	0.85
0.88	0.82	0.90	0.90		0.82	0.85	0.84
0.98	0.84	0.89	0.88		0.83	0.85	0.74
0.96	0.83	0.89	0.88	0.91	0.82	0.87	0.75
0.95	0.82	0.90	0.89	0.87	0.82	0.88	0.91
0.96	0.84	0.91	0.90	0.87	0.81	1.01	0.86
0.94	0.84	0.92	0.92	0.88	0.82	0.82	0.88
0.89	0.85	0.93	0.93	0.88	0.82	0.70	0.68
0.85	0.81	0.95	0.92	0.84	0.78	0.88	0.89
0.89	0.83	0.96	0.93	0.83	0.80	0.89	0.91
0.91	0.83	1.02	1.01	0.86	0.81	0.97	0.95
0.91	0.81	1.10	1.06	0.86	0.81	1.04	1.02
0.93	0.82	1.09	1.09	0.85	0.81	1.04	0.99
0.91	0.83	1.07	1.08	0.86	0.81	1.05	0.99
0.92	0.83	1.04	1.08	0.86	0.82	1.01	1.02
0.93	0.83	1.02	1.07	0.87	0.81	1.00	1.00
0.93	0.83	1.07	1.08	0.87	0.82	1.03	1.02
0.94	0.84	1.04	1.03	0.86	0.83	0.99	0.98
0.94	0.83	1.00	1.01	0.87	0.82	0.96	0.94
0.91	0.82	0.95	0.96	0.85	0.82	0.92	0.92

Appendix 5(vi). - Correlation coefficients for measured variables verses reductions in performance.

	Even power (glucose)	Even power (placebo)	Road race simulation (glucose)	Road race simulation (placebo)
% Fresh time	1.00	1.00	1.00	1.00
Absolute time	0.66	0.73	0.25	0.25
Reduction in power	-0.96	-0.94	-0.97	-0.97
Performance power	-0.80	-0.52	-0.12	0.36
Heart rate	0.12	-0.08	0.55	-0.39
%maximum heart rate	0.01	-0.42	0.85	-0.12
% maximum heart rate @ 70%	0.00	-0.33	0.85	-0.04
% maximum heart rate during performance test	0.00	-0.61	0.77	0.39
% maximum heart rate during last minute of performance test	0.23	-0.74	0.13	0.15
Heart rate drift	0.75	-0.06	0.77	0.50
Blood lactate concentration	-0.58	-0.53	0.49	0.01
Blood lactate concentration post performance	0.56	0.23	-0.22	-0.84
Blood glucose concentration	-0.68	-0.51	0.29	-0.35
Blood glucose concentration post performance	-0.43	-0.49	0.58	-0.47
Plasma ammonia concentration	0.29	0.46	0.59	-0.02
Plasma ammonia concentration above warm up measure	-0.43	-0.39	0.80	0.30
Plasma urea concentration	0.55	0.37	0.82	-0.19
Plasma urea concentration above warm up measure	-0.27	0.35	0.71	0.43
$\dot{V}_{O_2}$ mid lap	-0.63	-0.51	0.21	0.82
% $\dot{V}_{O_{2max}}$ mid lap	-0.25	-0.26	0.45	0.82
$\dot{V}_{O_2}$ mid lap matched	-0.61	-0.46	0.28	0.62
% $\dot{V}_{O_{2max}}$ mid lap matched	-0.27	-0.22	0.47	0.70
$\dot{V}_{O_2}$ end lap	-0.81	-0.60	0.01	-0.10
% $\dot{V}_{O_{2max}}$ end lap	-0.28	-0.30	0.20	0.10
$\dot{V}_{CO_2}$ mid lap	-0.70	-0.66	0.78	0.61
$\dot{V}_{CO_2}$ mid lap matched	-0.66	-0.60	0.87	0.67
$\dot{V}_{CO_2}$ end lap	-0.78	-0.73	0.13	0.32
RER mid lap	-0.71	-0.84	0.57	0.15
RER mid lap matched	-0.70	-0.79	0.64	0.57
RER end lap	-0.11	-0.79	0.23	-0.11
Efficiency mid lap	0.44	0.88	0.30	-0.96
Efficiency mid lap matched	0.44	0.71	0.23	-0.43
Efficiency end lap	0.38	0.77	-	
Economy mid lap	0.39	0.84	0.32	-0.91
Economy mid lap matched	0.40	0.66	0.30	-0.57
Economy end lap	0.35	0.66	0.86	0.31

Appendix 5(vii) - Lap by lap correlation coefficients for measured variables verses reductions in performance for each lap of each condition.

	Even power (glucose trial)				Even power (placebo trial)				Road race simulation (glucose trial)				Road race simulation (placebo trial)			
	1	2	3	4	1	2	3	4	1	2	3	4	1	2	3	4
Ammonia	0.63	0.08	0.64	-0.14	0.66	0.65	0.33	0.13	0.19	0.73	0..04	0.35	0.2	-0.75	-0.29	0.43
Ammonia (AWU)	-0.33	-0.47	0.21	-0.66	-0.30	0.18	-0.24	-0.65	0.53	0.83	0.26	0.74	0.50	-0.79	-0.10	0.51
Urea	0.66	0.51	0.64	0.28	0.27	0.36	0.36	0.45	0.63	0.78	0.94	0.67	-0.05	-0.34	-0.27	-0.23
Urea (AWU)	-0.12	-0.03	0.03	-0.76	-0.20	0.21	0.31	0.65	0.42	0.59	0.94	0.58	0.58	0.59	0.30	0.32
Heart rate	-0.01	0.03	0.14	0.31	0.04	-0.25	-0.11	0.02	0.49	0.34	0.52	0.75	-0.46	-0.47	-0.48	-0.05
% max heart rate	-0.08	-0.06	0.04	0.16	-0.29	-0.54	-0.47	-0.33	0.74	0.72	0.82	0.95	-0.23	-0.31	-0.22	0.25
Blood lactate	-0.64	-0.52	-0.50	-0.63	-0.77	-0.60	-0.47	-0.03	-0.03	0.61	0.64	0.35	-0.23	-0.17	-0.01	0.53
Blood glucose	-0.46	-0.74	-0.70	-0.89	-0.47	-0.52	-0.41	-0.54	0.20	0.04	0.32	0.46	-0.50	-0.41	-0.25	0.14
$\dot{V}O_2$ mid lap total	-0.29	-0.47	-0.63	-0.62	-0.48	-0.47	-0.50	-0.57	0.14	0.17	0.71	0.26	0.95	0.53	0.64	0.77
% $\dot{V}O_{2max}$ (total)	0.65	-0.57	-0.25	-0.20	-0.21	-0.23	-0.28	-0.32	0.39	0.42	0.67	0.40	0.91	0.63	0.67	0.78
$\dot{V}O_2$ matched	-0.20	-0.37	-0.62	-0.80	-0.43	-0.37	-0.27	-0.56	0.39	0.16	0.80	0.23	0.96	0.60	0.69	0.83
% $\dot{V}O_{2max}$ (match)	0.74	-0.67	-0.27	-0.58	-0.18	-0.12	0.47	-0.35	0.56	0.38	0.61	0.40	0.76	0.73	0.73	0.81
$\dot{V}O_2$ end lap	-0.72	-0.84	-0.80	-0.95	-0.39	-0.62	-0.67	-0.56	-0.03	-0.02	-0.11	0.15	0.24	0.24	0.43	0.55
% $\dot{V}O_{2max}$ (end)	-0.17	0.04	-0.23	-0.25	-0.04	-0.32	-0.64	-0.22	0.14	0.19	0.07	0.32	0.47	0.78	0.52	0.74
$\dot{V}CO_2$ mid lap total	-0.66	-0.55	-0.72	-0.65	-0.66	-0.64	-0.63	-0.71	0.15	0.18	0.75	0.33	0.88	0.57	0.62	0.73
$\dot{V}CO_2$ matched	-0.63	-0.48	-0.68	-0.58	-0.62	-0.57	-0.52	-0.65	0.41	0.21	0.90	0.31	0.89	0.65	0.70	0.79
$\dot{V}CO_2$ end lap	-0.72	-0.74	-0.82	-0.98	-0.69	-0.79	-0.65	-0.72	0.00	0.28	0.05	0.16	0.14	0.28	0.23	0.35
RER mid lap total	-0.54	-0.78	-0.87	-0.43	-0.91	-0.84	-0.61	-0.83	0.17	0.21	0.68	0.48	-0.21	0.20	0.37	0.42
RER matched	-0.65	-0.67	-0.89	-0.61	-0.86	-0.77	0.08	-0.79	0.25	0.33	0.82	0.25	0.15	0.59	0.40	0.40
RER end lap	-0.09	0.41	-0.31	-0.91	-0.89	-0.89	-0.15	-0.88	0.04	0.85	0.32	0.20	-0.31	0.30	-0.15	0.21
GME mid lap total	-0.01	0.27	0.45	0.40	0.85	0.80	0.82	0.98	0.04	0.11	-0.30	-0.07	-0.89	-0.23	-0.46	-0.52
GME matched	-0.13	0.07	0.46	0.54	0.63	0.52	0.43	0.90	-0.32	0.14	-0.58	-0.07	-0.93	-0.39	-0.60	-0.68
GME end lap	-0.24	0.63	0.34	0.28	0.44	0.85	0.76	0.61								
Econ mid lap total	-0.06	0.2	0.41	0.39	0.79	0.71	0.74	0.97	-0.06	0.14 -	-0.25	-0.04	-0.88	-0.17	-0.44	-0.51
Econ matched	-0.19	0.01	0.43	0.52	0.50	0.40	0.44	0.90	-0.27	0.19	-0.25	-0.04	-0.88	-0.35	-0.44	-0.66
Econ end lap	0.19	0.60	0.31	0.15	0.22	0.76	0.67	0.54	0.74	0.97	0.92	0.59	0.83	0.48	0.42	-0.19

Appendix 5(viii). Correlation coefficients for road race and even power trial decrements in performance and mean physiological variables

	Simulated Road Race	Even Power
TIME	1.00	1.00
mass	0.50	-0.49
MAX	0.52	-0.40
perf power	0.32	-0.63
LACTATE	0.33	-0.58
% HR DURING	0.34	-0.27
%HR @70%	0.49	-0.17
% PERF HR	0.74	-0.31
hr last min	0.10	-0.22
HR DRIFT	0.84	0.70
AMMONIA	0.26	-0.03
AMMONIA ABOVE RESTING	0.82	-0.32
EFFICIENCY	0.04	0.87
VO2	0.50	-0.70
VO2.BM	0.44	-0.44
VO2TOTAL	0.50	-0.55
RER	0.47	-0.88
VCO2	0.42	-0.60
VE	0.55	-0.60
VE END OF LAP	0.30	-0.61
VE 2MIN	0.66	-0.61
UREA	0.25	0.42
UREA ABOVE RESTING	0.81	-0.08
GLUCOSE	0.10	-0.73
GLABW	0.06	-0.67
LACPERF	-0.81	0.39
GLUC PERF	0.08	-0.68
GLUC AB PERF	0.04	-0.59
eff0	0.44	0.44
eff5	0.58	-0.32
VO2DRIFT	-0.81	0.55
%MAX DRIFT	-0.86	0.61