

**AN INVESTIGATION OF EFFICIENCY WITHIN  
CYCLING**

**BY**

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## ABSTRACT

The effect of training on metabolic efficiency in cycling is an under researched area. Previous studies have not found significant differences in cycling efficiency between trained cyclists and untrained participants which has largely limited further research in this area. However upon closer examination of the literature problematic methods are apparent. The main aim of this thesis was to investigate the effects of training upon metabolic efficiency in cycling. Before this could be done, it was necessary to establish a testing protocol capable of producing reliable data for use in the calculation of metabolic efficiency. This enabled the calculation of an appropriate sample size to have a chance of detecting significant differences between groups of trained and recreational cyclists. Statistically significant differences were consequently found between these two populations. Training studies were therefore needed to establish whether cycling efficiency was affected by training and if so what type. The results of the subsequent training studies showed firstly, that alterations in training volume and intensity did result in changes in the metabolic efficiency of cycling. Secondly, using an intervention study the metabolic efficiency of cycling was specifically increased as a result of the addition of high intensity training. Training volume was shown to have little effect on metabolic efficiency. This thesis is the first to demonstrate that metabolic efficiency is directly influenced by training over a cycling season and is significantly increased as a result of high intensity training.



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# CHAPTER 1: INTRODUCTION AND REVIEW OF THE LITERATURE

## **1.1 Rationale for the work of this thesis**

Efficiency has been suggested to be one of the most important functional abilities of cyclists involved in road race competitions (Coast et al., 1986; Horowitz et al., 1994; Coyle, 1995; Olds et al., 1995; Coyle 1999; Mosley & Jeukendrup, 2001). The cost, in terms of energy expenditure, to maximise mechanical work during competition is likely to play a key role in the successful performance of an athlete (Coast et al., 1986; Coyle, 1995; Olds et al., 1995; Coyle 1999; Horowitz et al., 1994; Mosley & Jeukendrup, 2001). However, of the other proposed factors which determine performance;  $\dot{V}O_{2max}$  (Faria, 1992; Olds et al., 1995; Bassett & Howley, 2000; Atkinson et al., 2003a), Lactate Threshold (Faria, 1992; Coyle, 1995; Coyle 1999; Atkinson et al., 2003a) and Anaerobic Capacity (Joyner & Coyle, 2008), efficiency has received the least research attention. Although research has been conducted in this area, there is still considerable debate about whether efficiency changes in response to training (Coyle, 2005; Martin et al., 2005; Schumacher et al., 2005; Gore et al., 2008). Therefore this thesis will aim to investigate the influence of training on metabolic efficiency within cycling.

Measures of efficiency and economy have been extensively used to provide a convenient index of the effectiveness with which an individual can convert chemical energy into mechanical power (Gaesser & Brooks, 1975; Suzuki, 1979; Hagberg et al., 1981; Künstlinger et al., 1985; Barbeau et al., 1993;

Nickleberry & Brooks, 1996) and has been reported to be in the range of 17 – 23% (Coyle et al. 1992; Moseley & Jeukendrup, 2001). An improvement in efficiency implies an increase in mechanical power output for any specific metabolic cost (Olds, 1995). The 17 – 23% range would suggest that for the same rate of metabolic energy expenditure, an efficient cyclist could produce 35% more power than a less efficient cyclist  $((23-17)/17) \times 100 = 35\%$ ). Horowitz et al. (1994) suggested that an absolute difference in efficiency of 1.8% could result in a 9% difference in maximal sustained power during 1 hour cycling performance test. Similarly, Mosley and Jeukendrup (2001) predicted that a 1% increase in efficiency could result in a 63 second improvement in 40km time-trial performance. This could potentially be crucial in a race performance; for example, the 53km individual time trial of the 2008 Tour de France saw Carlos Sastre maintain an advantage of 65 seconds to his closest rival Cadel Evans in penultimate stage to hold on to the yellow jersey. Coyle (2005) found an 8% improvement in efficiency during a case study of one professional cyclist. He suggests this translates into an 18% improvement in steady state power output per kilogram of body weight when cycling at a given oxygen cost. Therefore it is evident that improving efficiency may afford real performance related benefits to competitive cyclists.

In this chapter of the thesis there will be a critical examination of the factors that influence cycling efficiency, data collection and the potential mechanisms by which efficiency could be increased with training. This

information will help to identify the current key issues within the efficiency debate and inform the subsequent investigations within this thesis.

## **1.2 The concept of efficiency**

### **1.2.1 The efficiency of human movement**

The concept of efficiency considers the relationship between energy input and output. This relates the amount of energy required to perform a particular task, to the external work accomplished. Stainsby et al. (1980) provide a definition of efficiency as

“the work accomplished divided by the energy expended to do that work”

(Stainsby et al., 1980, p518).

In most physiological investigations the work accomplished is measured as external work performed by a muscle or intact body and is normally expressed as a percentage.

The energy used in muscle contraction is derived mainly from the oxidation of carbohydrates and fat. During this process a portion of the energy released is conserved within the Adenosine Tri-phosphate (ATP) molecule. This process is termed phosphorylative coupling and has an efficiency of approximately 60% (Whipp & Wasserman, 1969). In muscle contractions, some energy available from ATP is used for performance of work, this is termed contraction coupling and has an efficiency of approximately 49% (Whipp & Wasserman, 1969). Phosphorylative and contraction coupling are

independent of each other and are in series with each other (Wilkie, 1974 & 1975). Therefore when viewing the overall efficiency of a muscle as the ratio of external work performed to the energy expended by the muscle, it is the product of these two processes. According to Whipp and Wasserman (1969) this would make the maximal overall efficiency, in isolated preparations, about 30% and set the ceiling for human efficiency measurements. Even so, values as low as -120% for downhill treadmill walking (Margaria, 1968) and +250% for level treadmill walking (Pierrynowski et al. 1980) have been reported.

Since the early 20<sup>th</sup> century much of the research into human muscular efficiency has employed cycle ergometry (Benedict & Cathcart, 1913), as it was thought to allow accurate quantification of energy output (however this was not necessarily the case as many such ergometers may never have been calibrated). Consequently recent research has tended to assume cycling efficiency measured by indirect calorimetry, equates with efficiency of the muscle (Whipp & Wasserman, 1969, Gaesser & Brooks, 1975, Nordeen-Snyder, 1977, Suzuki, 1979, Hagberg et al. 1981, Coyle et al, 1992). However, in most instances it is not possible to measure efficiency using in vitro muscle preparations due to the invasive nature of the techniques used. Instead caloric equivalent of substrates utilized are determined from gross sub-maximal oxygen uptake and respiratory quotient (RQ) (the ratio of metabolic gas exchange) and used to gauge a measure of the energy cost of the exercise. External work is measured via an exercise ergometer.

### **1.2.2 Efficiency definitions**

During steady state cycle ergometry efficiency has been used to provide a convenient index of how effectively a cyclist can convert chemical energy into mechanical power (Gaesser & Brooks, 1975, Suzuki 1979, Hagberg et al. 1981, Künstlinger et al. 1985, Barbeau et al, 1993, Capelli et al. 1993, Kenny et al, 1995, Nickleberry & Brooks, 1996). However, there are inherent problems in estimating muscular efficiency from cycle ergometry which will be further discussed below.

During cycling exercise the human body acts as an energy converting system and work done would be moving the human body (mass) against an external resistance (power output). Energy inflow into this system is therefore contained as food intake. Thus it is intuitive to assume that the energy consuming processes of digestion be considered in the calculation of efficiency. It could also be suggested that energy to sustain homeostasis also be considered within total energetic cost, as without this maintenance cost the cyclist would be unable to do any work. Many researchers have been interested in the human body as an energy converting system. They have therefore sought to subtract all of the energy costs not directly attributable to work production from the estimation of energy expenditure via oxygen uptake, referred to as base-line subtraction (Gaesser & Brooks, 1975; Stainsby et al., 1980). Various methods of base-line subtraction have been proposed, although all could be criticised due to their over simplification of the complex physiological systems that contribute to measured energy expenditure.

The simplest form of base-line subtractions is net efficiency. Net efficiency requires the energy expenditure at rest to be measured, and assigned to the performance of internal work (to maintain homeostasis). This is then subtracted from the total amount of energy expended for the external work accomplished. The reasoning behind this subtraction is that resting metabolic rate is needed to maintain homeostasis, irrespective of the work accomplished, and therefore is not associated with doing this work.

$$\frac{\text{Work Accomplished}}{\text{Energy Expended above that at rest}} \times 100$$

#### **Equation 1 – Net Efficiency**

However, it is very difficult to obtain valid and reliable resting energy expenditure measurements as there are many factors which could influence resting metabolism such as prior physical activity, travel to the laboratory, stress and ambient temperature. One would also presume that by performing such a subtraction it is assumed that resting metabolic rate is constant (which is clearly not the case [Stainsby et al., 1980]) and completely independent from the processes involved in performing work. Therefore the human body would be capable of having two energy flows (homeostasis and work production) which run in parallel and do not influence or rely on each other. For some bodily functions this may be the case, they may have little impact on work production (e.g. gastrointestinal metabolism), however to suggest that these functions have no impact on the energy cost of work production or that work does not affect them seems overly simplistic.

Experimental and theoretical analyses of mechanical work (Caldwell & Forrester, 1992), as well as simulation-based analyses (Neptune & van den Bogert, 1998), have demonstrated that the amount of mechanical work done by muscles during locomotor movements is incorrectly estimated when internal work is considered as an independent cost. This suggests that physiological processes do not stay stable with increasing exercise intensity.

The calculation of net efficiency does not take into account the cost of moving the involved limbs to perform movement (Cavanagh & Kram, 1985; Stainsby, et al., 1980). It is proposed that for a valid measurement of cycling efficiency this must be taken into account (Gaesser & Brooks, 1975). When cycling on an ergometer against zero load (unloaded pedalling) energy is expended above that at rest although no useful work is being done (Whipp & Wasserman, 1969). This could also be included within the term internal work (resting metabolic rate + energy required to move the legs/stabilise the body on the bike). In the calculation of Work Efficiency (WE) the internal work is subtracted from the total or gross energy expenditure during exercise at a measured power output.

$$\frac{\text{Work Accomplished}}{\text{Energy Expended above that during unloaded cycling}} \quad \times 100$$

**Equation 2 – Work Efficiency**

The calculation of unloaded cycling is problematic. The WE calculation suggests that the energetic cost of unloaded cycling is an energy loss (i.e.

always lost as heat). There is also the assumption that this cost is a constant independent energy flow, different from that of loaded cycling. When measured on an ergometer the total work done (energy expended) is zero however, this does not reflect what is happening within the body. If the legs are moving similarly to normal pedalling during unloaded pedalling, then the kinetic energy of the legs will have to be decreased by negative muscle work (to decelerate the leg). The energy of the legs can no longer be decreased by doing external work (Kautz & Neptune, 2002). Therefore energy is being lost by decreased mechanical effectiveness rather than heat. Consequently it could be argued that unloaded cycling would not provide a valid baseline to apply to a range of work intensities as it is dissimilar to normal pedalling.

Gaesser and Brooks (1975) proposed a floating baseline measure for the physiological and external energy cost of exercise, Delta Efficiency (DE). Energy expenditure at a lower power output is subtracted from the energy expenditure at a higher one. The advantage of this calculation is that the measurement of resting metabolic rate is not required, and it is less affected by potential changes in base-line energy cost caused by work rate (e.g. body temperature related increases in metabolic rate).

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

**Equation 3 – Delta Efficiency**



Coyle et al. (1992) therefore suggests that this is the most valid estimate of efficiency as the calculation estimates energy expenditure that only contributes to the work output. However Moseley and Jeukendrup (2001) have shown it to be an unreliable measurement and Stainsby et al. (1980) suggest that DE is only valid in situations where the unmeasured work remains constant across exercise conditions. Stainsby et al. further argue, since DE is necessarily calculated from data collected over two or more different power outputs, this assumption becomes untenable. This is because the delta efficiency calculation assumes that the energetic cost of delta power production at a given work rate is independent from that of the previous work rate. Therefore the assumption is that efficiency is independent of the work rate increments, i.e. it is a linear response. Moseley and Jeukendrup, (2001) have shown this not to be the case, especially at higher power outputs. They have shown that there is a disproportionate increase in efficiency at higher work rates due to the proportion of energy expenditure used to maintain homeostasis becoming smaller as the total energy expenditure increases. Thus for efficiency to increase at higher work rates, energy expenditure must increase non-linearly. As DE assumes a linear relationship between power output and energy expenditure it may over-estimate efficiency at higher power outputs.

The most commonly used measure of efficiency is Gross Efficiency (GE) (Coyle et al. 1992, Nickleberry & Brooks, 1996, Passfield & Doust 2000, Moseley et al. 2004). The calculation of GE divides the work accomplished by the total energy cost.

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

#### **Equation 4 – Gross Efficiency**

The GE work rate relationship is curvilinear as a consequence of proportion of energy expenditure used for maintenance of homeostasis becoming smaller as the total energy expenditure increases. Gaesser and Brooks (1975) therefore considered this calculation to be distorted by internal work and thus rejected the use of GE. Stainsby et al. (1980) would also argue that for these reasons GE does not resemble muscular efficiency. Even withstanding this, Poole et al. (1992) have demonstrated parallel increments in leg and whole body  $\dot{V}O_2$  in response to increments in power output over a range of 20-90%  $\dot{V}O_{2\max}$ . They established that estimates of muscular efficiency calculated from whole body  $\dot{V}O_2$  do in fact approximate efficiency of working limb muscles in humans during cycling. If one is attempting to identify the total energetic cost of cycling to the body, as the entire body is in action, then a whole body efficiency measure is preferable.

#### **1.2.3 Efficiency within the performance model**

The majority of the performance models of cycling have presented a physiological approach to outlining the determinants of cycling performance (Olds et al., 1995; Coyle, 1995 & 1999; Joyner & Coyle, 2008). This is also the case when cycling efficiency is considered; the majority of research has been physiological in nature. Therefore it is the intention of this thesis to focus primarily on physiological rather than on biomechanical aspects

related to cycling efficiency. One such model of performance is presented by Joyner and Coyle (2008) who propose an integrated model of the physiological determinants of endurance ability. This model is a refinement of the performance model which Coyle has previously presented (Coyle, 1995 & 1999), and suggests endurance performance is composed of 'performance related abilities', determined by 'functional abilities' and 'morphological components'. The refined model presented in Figure 1 has three main facets; lactate threshold  $\dot{V}O_2$ , anaerobic/buffering capacity and efficiency of movement which are all directly linked to key morphological characteristics. Alterations to any of the morphological components would therefore affect their relative contribution to factors within the cyclist's functional abilities (Performance  $\dot{V}O_2$  and Gross Mechanical Efficiency), and performance related responses.

As illustrated in Figure 1, Coyle and co-workers propose that a key determinant of efficiency in cycling is muscle fibre type. Their previous work has demonstrated that there is a positive relationship between the percentage Type I fibres and efficiency (Coyle et al., 1992; Horowitz et al., 1994). In respect to performance, cyclists with a high percentage of Type I fibres were able to maintain a 9% higher power output than those with a comparably low percentage of Type I fibres (Horowitz et al., 1994). Herein lies an unusual paradox in that research has demonstrated trained endurance athletes possess higher percentages of Type I fibres compared to an untrained individual (Tesch & Karlsson, 1985). Therefore it could be expected that calculated efficiency is different between trained and

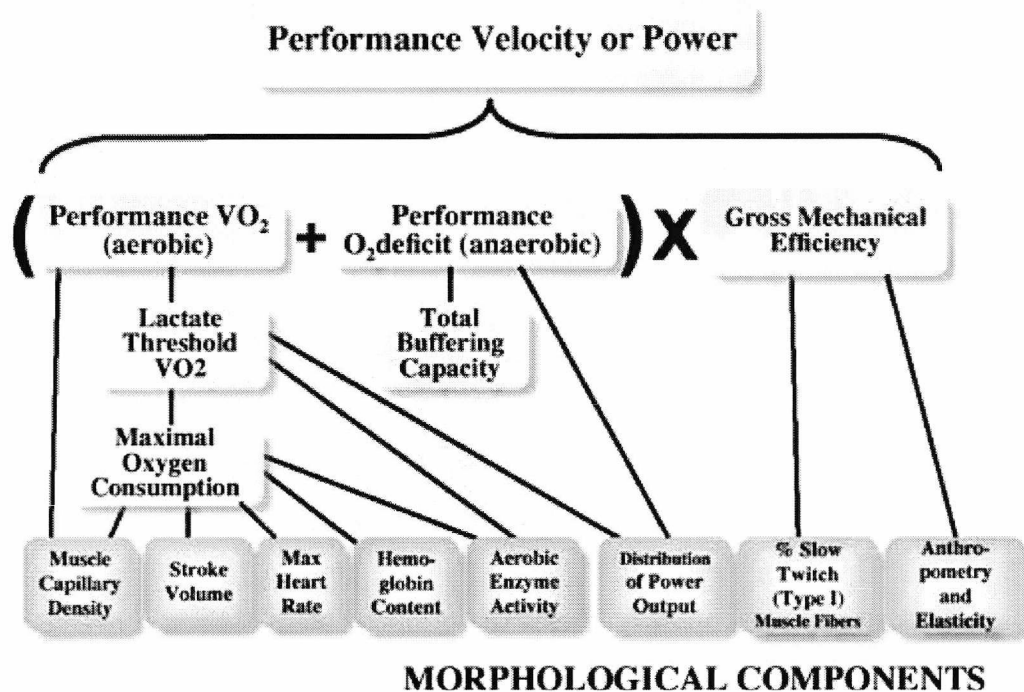


Figure 1. Integration of physiological factors determining endurance performance ability (Joyner & Coyle, 2008).

untrained individuals during cycling exercise. However this is not the case, the majority of past research using cross-sectional research designs has identified no significant differences in efficiency between trained and untrained participants (Böning et al., 1984; Marsh & Martin 1993; Nickleberry & Brooks 1996; Marsh et al., 2000; Moseley et al., 2004). Therefore, research in the field of training and its effect on efficiency has been limited.

Cycling performance involves integration of muscular, cardiovascular and neurological factors that function cooperatively to efficiently transfer the energy from aerobic and anaerobic ATP turnover into velocity and power.

Research has described the cardiovascular and muscular factors that govern oxygen delivery to active muscles (Bassett & Howley, 2000; Kanstrup & Ekblom, 1984; Saltin & Strange, 1992), oxidative ATP rephosphorylation (Balaban, 1990) and markers of metabolic stress (Holloszy & Coyle, 1984; Robergs et al., 2004). It has become increasingly apparent that metabolic efficiency is hugely important as a physiological determinant of cycling performance. In theory, if cyclists were able to improve their efficiency for work at the same power output, they would be working at a lower proportion of their  $\dot{V}O_{2\max}$ . This could therefore potentially increase the power output during endurance performance (Horowitz et al., 1994). However there is little conclusive evidence within the literature as to the determinants of efficiency and how they are affected by training. Therefore, not only does efficiency provide a fascinating topic for study, but it also has a potential to provide a significant performance advantage.

#### **1.2.4 Exercise Economy**

Due to the difficulties of obtaining accurate estimates of work done, the term 'economy' rather than efficiency is often used. In contrast to efficiency, economy refers to the energy required to maintain a constant velocity of movement or power output. Economy may therefore be defined as the submaximal oxygen uptake (per unit of bodyweight  $\dot{V}O_{2\text{submax}}$ ) required to perform a given task (Cavanagh & Kram, 1985). The lower the submaximal oxygen cost for a given work rate the more economical performance. Although it cannot be immediately assumed that this means an improved efficiency, as work done is not included in the calculation. Even

so it is often used as a physiological criterion for 'efficient' performance (Lucia et al. 1998 & 2002a).

A high cycling economy will require a lower percentage of  $\dot{V}O_{2max}$  required to sustain a given mechanical work (Lucia et al. 2002a). Professional cyclists have been shown to display a considerably higher cycling economy than amateur cyclists despite possessing similar  $\dot{V}O_{2max}$  values (Lucia et al. 1998 & 2002a). In runners with similar  $\dot{V}O_{2max}$  values, economy has been used to explain 65.4% of the variation in running performance (Conley & Krahenbuhl, 1980). Horowitz et al. (1994) investigated the effect of muscle fibre type on cycling economy and endurance performance. Cyclists with a high Type I percentage of fibres were able to produce an average of 9% greater power output for the same oxygen cost during a one hour performance trial compared to individuals high in Type II fibres. This finding was independent of the cyclists  $\dot{V}O_{2peak}$  and indices of metabolic strain.

### **1.2.5 Summary**

There are several different methods to calculate efficiency, each with a specific criticism. Baseline subtraction methods seem to come up against most criticism for the subtraction of an estimated amount of energy expenditure. What is clear from the literature is that whole body efficiency measured via gases expired at the mouth may not provide a valid measurement of energy expended by muscle. Thus the usage of the term 'gross mechanical efficiency' may be a misnomer. Throughout the rest of

this thesis the term 'gross efficiency' will therefore be used instead of 'gross mechanical efficiency' to indicate whole body energy expenditure. Gross efficiency may be a more relevant measure from a practical point of view as it provides a measure of general whole body efficiency. Patently DE is not a measure of 'muscular efficiency', however some might assume it is indicative. DE has also not been reported to be the most reliable measurement of efficiency.

## **1.3 FACTORS INFLUENCING EFFICIENCY**

### **1.3.1 Muscle Fibre Type**

Coyle et al. (1992) have suggested that most of the variability in efficiency observed in well-trained cyclists is due to differences in muscle fibre type composition. Coyle et al. (1992) have found the percentage of slow twitch fibres in the Vastus Lateralis muscle to be positively correlated with gross and delta efficiency, not only in cycling, but also in a novel two-legged extension task. From their data they developed a regression equation for estimating gross efficiency and delta efficiency from the measured percentage of Type I muscle fibres:

$$GE = 0.0671(\% \text{ Type I}) + 16.991 \quad (r=0.75, p<0.001)$$

Coyle et al. (1992) page 785.

**Equation 5 - The calculation of GE from a measured percentage of Type I muscle fibres**

$$DE = 0.1393(\% \text{ Type I}) + 13.49 \quad (r=0.85, p<0.001)$$

Coyle et al. (1992) page 785.

**Equation 6 - The calculation of DE from measured percentage of Type I muscle fibres**

Later research by Horowitz et al. (1994) divided participants into a high and low percentage Type I muscle fibre groups, subsequently endurance performance was evaluated using a one hour performance test. As predicted by their equations, cyclists with a higher percentage of Type I fibres also



had a significantly higher efficiency and were able to maintain a 9% higher power output. Jansson and Kaijser (1987) studied the metabolic responses of untrained and highly trained men to exercise at 65%  $\dot{V}O_{2peak}$ . They demonstrated that highly trained individuals had a greater percentage of Type I muscle fibres (70% vs 40%) and thus GE (22% vs 19%). If this data is then applied to equation 5 of Coyle et al. (1992) the calculated GE values of 22% and 20% are very similar to those reported above. This further provides evidence that GE and the percentage of Type I muscle fibres may be related. If highly trained participants possess a greater percentage of Type I muscle fibres which are correlated with a high GE, differences between trained and untrained participants should be evident in the efficiency literature.

The equations of Coyle et al. (1992) suggest that the maximal GE which could be achieved on the basis of the Vastus Lateralis muscle possessing 100% slow twitch fibres would be 23.7%, with maximal DE being 27.4%. Lucia et al. (2002a) demonstrate GE's of 24.5 – 28.1% which would suggest 111.9 and 165.6% slow twitch fibre compositions. This is clearly not possible and would suggest either measurement errors within Lucia et al's study or Coyle et al's regression equation. Similarly when using the regression equations on Coyle's own case study of the Grand Tour Champion (Coyle, 2005) the data are problematic. The 1999 data report a GE of 23.05% and a DE of 23.12%, these would equate to a percentage slow twitch composition within the Vastus Lateralis muscle of 90.3% and 69.1% respectively. This may suggest that there is not a linear relationship

between GE, DE and the percentage slow twitch fibres as Coyle et al's regression equations would expect.

Not all studies support the link between efficiency and muscle fibre type. Research by Suzuki (1979), Medbø et al. (1990) and Pedersen et al. (2002) have demonstrated no correlation between efficiency and the relative muscle compositions of Type I fibres. Medbø's study found  $O_2$  uptake to increase linearly with exercise intensity in 16 participants with no correlation between percentage Type I fibres and efficiency. They assumed a linear relationship between muscle fibre type, work rate and  $\dot{V}O_2$ , an assumption which is regarded as incorrect (Zoladz et al. 1995; Stuart et al. 1981). This assumption may mask a difference in efficiency of Type I and II fibres, as Barstow et al. (1996) have demonstrated an increased oxygen cost of high intensity exercise to be associated with a greater percentage of Type II fibres.

### **1.3.2 Aerobic enzyme capacity**

A fundamental adaptation to endurance-type exercise training is an increase in the oxidative capacity of the skeletal muscle (Holloszy & Coyle, 1984). This adaptation is evident by a general increase in the mitochondrial content with a corresponding increase in the potential for ATP provision by aerobic metabolism. Therefore there is a coordinated increase in the capacity for fatty acid, carbohydrate and amino acid oxidation and the enzymatic pathways required for handling this process (Holloszy & Booth, 1976). Gollnick et al. (1973) demonstrated increases in skeletal muscle oxidative

capacity and substrate utilisation following several weeks of moderate intensity exercise at  $\sim 65\% \dot{V}O_{2\text{peak}}$ . Similarly Green et al. (1992) have demonstrated that even a short period of endurance training (5-7 days) increases glycogen availability but reduces the rate of glycogen catabolism during matched-work exercise, resulting in increased endurance capacity (Green et al., 1995). These shifts in substrate utilisation are often attributed to the enhanced respiratory control sensitivity that results from increased mitochondrial density, as reflected by changes in the enzymatic activity in the tricarboxylic acid cycle and electron transport chain (Holloszy & Coyle, 1984).

Recently studies have shown that increases in maximal activities of mitochondrial enzymes reduces glycogen utilisation and lactate accumulation during matched-work exercise (Burgomaster et al., 2006), and improves performance during tasks that primarily rely on aerobic metabolism (Burgomaster et al., 2005 & 2006). Gibala et al. (2006) demonstrated that training related increases in muscle oxidative capacity and muscle buffering capacity contributed to an increase in performance, during a matched-work trial. They suggest this is primarily due to increases in the maximal activity of cytochrome c oxidase. These findings have also been demonstrated by Dudley et al. (1982) and Terada et al. (2001) using similar methodologies. More recently Iaia et al. (2008) have extended the findings of Gibala et al. (2006) by demonstrating reduced oxygen cost of fixed work intensities following high intensity training. Additionally, Burgomaster et al. (2008) report that skeletal muscle oxidative capacity is

increased, with net reductions in muscle glycogenolysis and phosphocreatine degradation following 6 weeks of high intensity training in previously untrained participants.

The time course required to demonstrate muscle fibre type adaptations (as discussed below in 1.4.1) is considerably longer than the 6 weeks of Burgomaster et al's study, and so may not be a factor in the results. However to the author's knowledge there is no study directly investigating the influence of aerobic enzyme capacity on efficiency in cycling. More work is required in this area to fully establish a relationship between aerobic enzyme capacity and cycling efficiency, as well as the potential mechanisms by which increases in aerobic enzyme capacity can increase efficiency in cycling.

### **1.3.3 Pedal Cadence**

#### a) Pedal cadence and efficiency

Of all of the factors that influence cycling efficiency, pedal cadence has received the most research attention (Bannister & Jackson, 1967; Jordan & Merrill, 1979; Hagberg et al., 1981; Faria et al., 1982; Croisant & Boileau, 1984; Merrill & White, 1984; Buchanan & Weltman, 1985; Coast et al., 1986; Hagen et al., 1992; Sidossis et al., 1992; Londree et al., 1997; Marsh & Martin, 1998; Takaishi et al., 1998; Chavarren & Calbet, 1999; Brisswalter et al., 2000; Lepers et al., 2001; Lucia et al., 2001b; Belli & Hintz, 2002; Foss & Hallen, 2004; Lucia et al., 2004a; Mora-Rodriguez & Aguado-Jimenez, 2006; Samozino et al., 2006). Most authors agree that

changes in muscle shortening velocity (i.e. pedal cadence) markedly affect efficiency, with the general consensus of opinion being that higher cadences result in decreased efficiency (Croisant & Boileau, 1984; Merrill & White, 1984; Coast, et al., 1986; Chavarren & Calbet, 1999; Samozino et al., 2006). The major stimulus for research has been the frequent finding that experienced cyclists often adopt a pedal cadence in excess of the most economical (Merrill & White, 1984, Patterson & Moreno, 1990, Nickleberry & Brooks, 1996, Marsh & Martin, 1997). In most of the studies cited above the optimal cadence lies between 60 and 80 rev.min<sup>-1</sup>.

Figure 2 illustrates the effect of increasing cadence on cycling efficiency using pooled data from previous research studies. Most research investigating the influence of cadence suggests that at a constant power output, efficiency decreases as cadence increases (Gaesser & Brooks, 1975; Suzuki, 1979; Jordan & Merill, 1979; Ferguson et al., 2001). However, as can be seen in Figure 2, the effect of cadence on efficiency is inconsistent. The inter-study variation is also much larger than any visible trend with some studies showing a positive effect. The inter-study variations could be explained by differences in the methods used to determine efficiency, possibly by variations in work rate. The greatest impact of cadence on efficiency seems to be at low work rates (Chavarren & Calbet, 1999; Samozino et al., 2006). This will be discussed in more detail below.

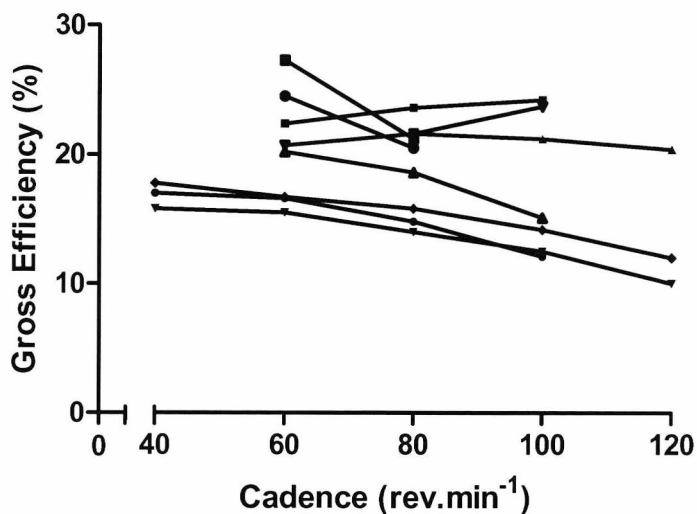


Figure 2. The effect of cadence on efficiency using pooled data from previous research studies (Data from Gaesser & Brooks, 1975; Nickleberry & Brooks, 1996; Sidossis et al., 1992; Chavarren & Calbet, 1999; Moseley & Jeukendrup, 2001; Foss & Hallen, 2004; Lucia et al., 2004; Moseley et al., 2004; Mora-Rodriguez & Aguado-Jimenez, 2006; Samozino et al., 2006)

Using computer simulations Umberger et al. (2006) found that efficiency is lowest at 40 rev.min<sup>-1</sup> and increases to a broad plateau between 60 and 100 rev.min<sup>-1</sup>, and decreases substantially at 120 rev.min<sup>-1</sup>. This is also supported by the findings of Woolford et al. (1999) who demonstrated that power output at lactate threshold was approximately 50W higher when elite junior cyclists used a pedal cadence of 90-100 rev.min<sup>-1</sup> compared to 120-130 rev.min<sup>-1</sup>. They also found metabolic cost to be higher for any submaximal work rate at 120-130 rev.min<sup>-1</sup> compared to 90-100 rev.min<sup>-1</sup>. Faria et al. (1982) established that at a low power output (140W) GE decreased from 18-14% as cadence increased from 68-132 rev.min<sup>-1</sup>, however at 290W it remained constant at 22%. It could be speculated that

their results may be due to the fact that they used trained cyclists where as previous studies used non-cyclists resulting in increased oxygen consumption without any increases in useful work.

Several factors are thought to contribute to the decreased efficiency at higher cadences. These include the increased cost of internal work (Whipp & Wasserman, 1969, Gaesser & Brooks, 1975, Hagberg et al. 1981), the reduced effectiveness of pedal force application (Lafortune & Cavanagh, 1980 Ericson & Nisell, 1988, Patterson & Moreno, 1990, Faria, 1992) and changes in muscle fibre recruitment (Barstow et al., 1996). Recently Samozino et al. (2006) found that cycling efficiency is decreased when cadence differed from the cadence which maximised efficiency (40 rev.min<sup>-1</sup>). They identified that when cadence increases and becomes higher than cadence at maximal efficiency, GE is decreased due to increased energy expenditure for the same power output.

It could be concluded from the literature above that the higher cadences used by elite level road race cyclists (range 89.3 – 92.4 ± 1.3 rev.min<sup>-1</sup>; Lucia et al., 2001b) do not appear to be beneficial. However the disadvantage of the higher aerobic demand at the preferred rate may be outweighed by the need to enhance muscular power production and minimise peripheral stress. Takaishi et al. (1996) have noticed that pedalling rates which produce minimal neuromuscular fatigue coincide with the cyclist's preferred rate. Cyclists may be recruiting fibres that they do not mind fatiguing, so that when required they can recruit the major muscle

mass which is still largely fatigue free. Faster cadences may be related to increasing muscular power and minimising fatigue rather than increasing metabolic efficiency. Similarly Kohler and Boutellier (2005) suggest that the freely chosen cadence may not follow the principle of minimising energy cost because of the differences between velocity giving maximal power and that of maximal efficiency. However their analysis do not account for other factors that are affected by cadence as well, such as activation-deactivation dynamics of the muscle.

Not all studies have found a decreasing efficiency as cadence increases. Hagberg et al. (1981) found experienced cyclists preferred cadence to also be their most economical with higher and lower cadences increasing the oxygen cost of the activity. They used blood lactate as a marker of stress, reporting that measured concentrations revealed a 'U' shaped response with the lowest point equating to a similar cadence as their preferred cadence during cycling at 80% of their  $\dot{V}O_{2max}$ . However the results of this study should be viewed with caution. Firstly, the efficiency values reported are related to work efficiency which has been criticised as a measure of efficiency (Gaesser & Brooks, 1975, Kautz & Neptune, 2002). Secondly, the chosen work rate of 80%  $\dot{V}O_{2peak}$  means the achievement of a metabolic steady state unlikely, especially since the blood lactate concentrations reported were  $6.0 \text{ mmol}^{-1}$ .

Davison and Flynn (1997) used a Kingcycle ergometer (Kingcycle Ltd., High Wycombe, Buckinghamshire, UK) to test trained cyclists and reported



GE was maximised at 90 rev.min<sup>-1</sup> as opposed to 80, 100 or 120 rev.min<sup>-1</sup>. Sidossis et al. (1992) found DE increases from 21 - 24.5% (24.5% is above 100% slow twitch fibre composition according to the regression equation of these authors. Coyle et al., 1992) as cadence increased from 60-100 rev.min<sup>-1</sup> also using trained cyclists. Other investigators also report that experienced cyclists are more efficient at higher pedal rates (Ahlquist et al., 1992, Takaishi et al., 1998, Lucia et al., 2004a). The studies of Sidossis et al. (1992), Faria et al. (1982), Ahlquist et al. (1992) and Lucia et al. (2004) used power outputs that were considerably higher than previous investigations, as a result their data may be more representative of competitive road race cycling and may be suggestive of cadence related training adaptations. This may also, to some degree, explain the choice of higher cadences in competitive cyclists and would question whether an increased oxygen cost is actually incurred by a trained population at these higher rates. It appears that the effect of cadence on O<sub>2</sub> cost may be population specific.

Several other factors have been shown to influence the efficiency/cadence relationship, including crank length, body position, velocities and acceleration of body segments, forces in muscles and joints, linear and angular displacement (Faria, 1992). Furthermore, cycling experience appears to influence efficiency at various cadences (Coast & Welsch, 1985, Faria et al. 1982, Hagberg et al. 1981). Marsh et al. (2000) asked experienced runners with no cycling experience (but of equal aerobic capacity to a group of cyclists), to pedal at their freely chosen cadence at a

constant power output of 200 watts. Surprisingly their average preferred cadence was 92 rev.min<sup>-1</sup> and their most economical cadence was 63 rev.min<sup>-1</sup>, essentially the same as the cadences recorded by the cyclists. This data contradicts previous research showing years of cycling experience are required to feel comfortable at higher cadences (Faria et al. 1982, Sidossis et al. 1992). Lucia et al. (2004a) showed mean GE to be significantly higher at 100 rev.min<sup>-1</sup> than at 60 rev.min<sup>-1</sup> in professional cyclists generating high power outputs (mean 366W). In contrast  $\dot{V}O_2$ , HR, Lactate, and muscle recruitment (Root Mean Squared-EMG in vastus lateralis and gluteus maximus) were significantly lower. This is reinforced by the observation that Lance Armstrong uses a high cadence (>90 rev.min<sup>-1</sup>) during road racing and time trialling regardless of terrain.

#### b) Pedal cadence and muscle fibre type

Slow twitch muscle fibres have been shown to possess a greater oxidative capacity and efficiency than fast twitch muscle fibres. Work by Goldspink (1978) demonstrated that during isometric muscle contractions Type I fibres were over three times more efficient than Type II fibres. Coyle et al. (1992) calculated GE and DE during 5 minute work bouts at 50, 60 and 70%

$\dot{V}O_{2max}$ . Significant correlations were found between percentage of slow twitch fibre and GE ( $r=0.75$ ,  $p<0.001$ ) and for percentage of slow twitch fibre and DE ( $r=0.85$ ,  $p<0.001$ ). However Coyle et al. (1992) did not take into account the variation between individuals in freely chosen pedal rate and the possible effect of this on the relationship between the proportion of Type I fibres and GE (their participants used a pre-set pedal rate of 80

rev.min<sup>-1</sup>). It has been suggested that cyclists with a high percentage of myosin heavy chain (MHC) Type I choose pedal rates close to the pedal rates at which maximum peak crank power occurs. Conversely participants with low percentages of MHC Type I may choose lower pedal rates more closely favouring high GE (Hansen et al., 2002). Therefore by fixing the cyclist's cadence at 80 rev.min<sup>-1</sup> Coyle et al. (1992) may have unwittingly affected their own results.

Suzuki (1979) collected muscle samples from the Quadriceps Femoris muscle of 6 participants (PE students and laboratory staff) and demonstrated that participants with a higher percentage of slow twitch fibres were more delta efficient at 60 compared to 100 rev.min<sup>-1</sup> (28.8 vs 23.3%). In contrast, participants with a higher percentage of fast twitch fibres were more delta efficient at 100 compared to 60 rev.min<sup>-1</sup> (28.8 vs 25.3%). However these results should be interpreted with caution due to the small sample size. Suzuki wrongly concluded that more resistance was offered to cross bridge cycling at 100 rev.min<sup>-1</sup> for slow twitch fibres as shortening velocity was too high. It is now known that the force demands of pedalling, rather than contraction velocity, determines muscle fibre recruitment (Ahlquist et al. 1992). Therefore a cadence of 100 rev.min<sup>-1</sup> is not too high for Type I muscle fibres to effectively contribute to cycling power output. In support of this Gollnick et al. (1974) found no significant differences in fibre type recruitment during different contraction frequencies (30-120 rev.min<sup>-1</sup>) at various intensities (30 – 150%  $\dot{V}O_{2max}$ ) using histochemical glycogen depletion patterns. Similarly Beelen et al. (1993) observed that both ST and

FT fibres were recruited during cycle exercise ( $90\% \dot{V}O_{2\max}$ ) at 60 and 120  $\text{rev}\cdot\text{min}^{-1}$ .

Higher pedal rates may require a greater oxygen uptake for a given power output because of an increase in internal work for repetitive limb movements (Boning et al. 1984, Gaesser & Brooks, 1975, Coast & Welch, 1985, Coast et al. 1986, Brisswalter et al. 2000). However, these higher pedalling rates may also decrease force demands per revolution (MacIntosh et al. 2000), shorten contraction time encouraging greater blood flow into the muscle (Takaishi et al. 1998, Gotshall et al. 1996) and enable greater recruitment of Type I muscle fibres (Takaishi et al. 1994, Ahlquist, 1992, Gotshall et al. 1996).

Crowley et al. (1996) report greater fast twitch fibre recruitment at cadences of 95 versus 55  $\text{rev}\cdot\text{min}^{-1}$  demonstrated by increases in blood lactate and epinephrine levels. They suggested that increases in DE at 95  $\text{rev}\cdot\text{min}^{-1}$  were due to contraction velocities being better matched to the optimal contraction velocities and thus efficiency of the recruited fibres. However, similar to Suzuki (1979), they wrongly concluded that this was due to fast twitch fibres. It is evident that at 80  $\text{rev}\cdot\text{min}^{-1}$ , Type I fibres are contracting closer to their peak efficiency contraction velocity rather than Type II's (Coyle et al., 1991; Zoldaz et al., 1995; Hansen et al. 2002). Consequently the cyclist with a higher percentage of Type I compared with Type II fibres will be more efficient, reflected by a lower  $\dot{V}O_2$  for a given power output. Maximal muscular efficiency occurs at approximately 1/3 of the maximal shortening

velocity in both Type I and Type II muscle fibres (Coyle et al., 1991). The maximal shortening velocity of Type II muscle fibres is however 3-5 times greater than that of Type I muscle fibres (Goldspink, 1978, Fitts et al., 1989). Thus a considerably faster cadence than  $80 \text{ rev}\cdot\text{min}^{-1}$  would be associated with optimising energy expenditure in actions dominated by Type II fibres. Studies have shown that maximum power during pedalling is achieved at rates near  $120 \text{ rev}\cdot\text{min}^{-1}$  (Hautier et al., 1996). In contrast, the freely chosen cadence of experienced cyclists is often  $\sim 90 \text{ rev}\cdot\text{min}^{-1}$  during submaximal pedalling (Hagberg et al., 1981; Marsh & Martin, 1993).

During submaximal pedalling, relatively more slow twitch fibres are recruited (Gollnick et al., 1974), and therefore the optimal power generation velocity would move to pedalling rates below  $120 \text{ rev}\cdot\text{min}^{-1}$  (Umberger et al., 2006). The preferred pedalling rate of  $90 \text{ rev}\cdot\text{min}^{-1}$  could be near optimal for producing muscle power during submaximal pedalling. Fibre type composition of the main power producing muscles is therefore likely to determine individual differences in preferred cadence. Where a cyclist is constrained to work at an externally fixed cadence (e.g. in a lab test or in a team time trial) they may be working at a cadence that is non-optimal. Therefore higher heart rates and oxygen consumption may occur than if they were using a preferred cadence at a fixed power output (Norden-Snyder, 1977; Hagberg, 1981). This again highlights problems of using methods with uniformly fixed, as opposed to individually selected cadences when investigating efficiency in cycling

### c) Summary

Cadence has been the most researched variable affecting efficiency in cycling. Studies have reported increases, decreases and unaltered efficiency values as a result of changes in pedal cadence. Trained cyclists tend to adopt pedal cadences in the range of 89-93 rev.min<sup>-1</sup> even though they are not necessarily the most efficient. Other factors may therefore influence the choice of pedal rate. Optimal muscle shortening velocity, increased muscle perfusion, fibre type recruitment, work rate and event duration have all been suggested as alternative reasons for pedal cadence selection.

### **1.3.4 Pedalling mechanics and muscle recruitment**

#### a) Pedalling dynamics

In a physiological study, Coyle et al. (1988) found that differences in performance between competitive cyclists were not entirely due to changes in the physiological parameters they studied ( $\dot{V} O_{2\max}$ , blood lactate threshold, muscle glycogen utilisation, fibre type composition, and muscle enzyme measures). From their findings they hypothesised that a cyclist's performance ability might in part be due to biomechanical factors related to individual pedalling technique. However, they did not collect biomechanical data to support their hypothesis.

Patterson and Moreno (1990) studied force effectiveness as a measure of wasted energy during cycling. They found that a significant amount of force produced (~25%) during the downstroke of the pedal action was used to overcome negative forces from the trailing leg. This additional work would

be associated with a greater metabolic cost compared to a situation whereby negative forces were reduced. Korff et al. (2007) have demonstrated that actively attempting to pull up during the upstroke was most mechanically efficient but least metabolically efficient. Recently force application during the upstroke phase has been shown to explain variations within GE in a group of recreational cyclists, i.e. those riders with an effective 'recovery' profile demonstrated a greater efficiency (Zameziati et al., 2006). Conversely Mornieux et al. (2006) found that the more effective their participants were in applying forces on the downstroke, the greater their efficiency, albeit again in a group of untrained cyclists. Thus if cyclists could focus on applying large downstroke forces this may result in increased efficiency.

Torque produced at the crank is not necessarily reflective of the work done by the muscles, and therefore the metabolic cost incurred (Kautz & Neptune, 2002). Energy increases and decreases in the leg are the result of redistribution of segmental energy by muscle forces. It is proposed that energy decreases resulting from leg deceleration are able to generate a pedal force tangential to the crank, and thus positive work (Kautz & Neptune, 2002). Therefore via the conservation of energy, external power at the crank can instantaneously exceed the total power produced by the muscle during deceleration of the between 95 and 170°. Assuming gases measured at the mouth reflect muscular energetic cost (Poole et al., 1992) it might be unlikely that relationships exist between efficiency and pedalling mechanics. This remains to be determined in a group of trained cyclists.

## b) Muscle recruitment

Cycling is characterised by cyclical movements that require muscles to generate mechanical power to overcome an external resistance (e.g. friction, gravity and inertia). Muscle power is the product of muscle force and contraction velocity, both of which are influenced by intrinsic muscle properties of length-tension and length-velocity relationships as well as the kinetics of muscle activation and de-activation (Neptune & Herzog, 2000). Although the complexity of these interactions is well described (Zajac, 1989), very little is known about the in vivo performance during cycling due to the difficulty of performing invasive evaluations of muscle force, length, velocity and activation.

Activation and deactivation dynamics have been shown to influence neural control and optimal performance albeit in an animal model (Josephson, 1993). Activation and deactivation dynamics are the processes that describe the delay between muscle force development (the delay between neural excitation arriving at the muscle and the muscle developing force) and relaxation (the delay between neural excitation finishing and muscle force falling to zero) (Zajac, 1989). These delays are therefore primarily due to the processes of calcium dynamics and cross-bridge attachment and detachment (Zajac, 1989). Using an animal model (rat soleus muscle) van Soest and Casius (2000) demonstrated that without the delay for activation and deactivation a single muscle could generate 60% greater muscle power during a high rate pedalling exercise. In turn this suggests that activation and deactivation dynamics (of the agonist and antagonists), rather than the



force-velocity relationship may be the limiting factor on muscle power at high contraction velocities ( $\sim 120 \text{ rev}\cdot\text{min}^{-1}$ ). It may be that through training at particular cadences, the nervous system develops patterns of excitation of individual muscles that accommodate the individual muscle's characteristics of activation and deactivation, thereby producing the most efficient performance of cycling movements. Consequently a greater coordination may be afforded between muscles that generate propulsive force during cycling. Efficient performance may depend on the ability of the nervous system to adapt to the influence of activation and deactivation dynamics to effectively develop power (i.e. for the same  $\text{O}_2$  cost more power could be generated). This suggestion is supported by mathematical modelling of muscle coordination during a bout of cycling exercise (Neptune and Herzog, 1999). Negative muscle work has been shown to increase at higher cadences ( $> 90 \text{ rev}\cdot\text{min}^{-1}$ ) as a result of activation and deactivation dynamics (i.e. the opposing muscle(s) 'switching off'). In Neptune and Herzog's model (Neptune & Herzog, 1992), virtually no negative muscle work (as the quadriceps provided downstroke torque, the opposing quadriceps were deactivated) was observed at  $90 \text{ rev}\cdot\text{min}^{-1}$  compared to higher pedal rates. Therefore minimal additional negative muscle work had to be overcome during the propulsive downstroke phase, resulting in a lower  $\text{O}_2$  cost. If as a result of training lower negative muscle forces were encountered due to more effective muscle coordination between the contracting muscles in one leg, between legs and in synergistic muscles (e.g. the gluteals), GE may increase.

Changing muscle recruitment has been shown to change GE. Cannon et al. (2007) demonstrated that pedalling with a dorsiflexed ankle position increased the activity of the gastrocnemius muscle, causing a decrease in GE when compared to a self-selected pedal stroke. The higher level of muscle recruitment (as measured via using EMG) in the dorsiflexed position, resulted in a greater  $\dot{V}O_2$  at the same work rate and cadence. However it could also be speculated that the demand of the experimental condition may have also resulted in greater trunk muscle and extraneous limb movement, which in turn would have also raised the  $\dot{V}O_2$  demand. As outlined in section 1.3.4a above Korff et al. (2007) investigated the effect of pedalling technique on efficiency in trained cyclists. Using instrumented pedals they discovered that attempting to pull up on the pedal was mechanically most efficient but metabolically least efficient. This is may be due to the increased muscle activity necessary to actively lift the leg during the recovery phase. Although it could be argued that asking participants to change pedalling style simply invokes an acute response of increasing  $\dot{V}O_2$  by using untrained recruitment patterns. If this action is trained, the cyclist may be able to decrease the oxidative cost however this speculation requires verification using an experimental study design.

### c) Summary

The extent to which forces measured at the pedal (using a conventional crank and pedal system) influence whole body efficiency calculated from gases collected at the mouth is unclear. More compelling evidence exists for the influence of changing muscle recruitment patterns. The possibility of

optimising the pedal cycle force dynamics to gain greater propulsive and less negative torques whilst, concurrently incurring the same relative metabolic cost from the exercising muscles offers a potential mechanism by which efficiency could be increased.

### **1.3.5 Anthropometric characteristics and body position**

Body size has been shown to affect efficiency whilst cycling. One early study by Wahlund (1948) found individuals with similar  $\dot{V}O_2$  values had similar body masses. A later study by Berry et al. (1993) reported net and gross  $\dot{V}O_2$  data to be positively correlated to body mass. Net and gross efficiency were also found to be significantly correlated with body mass during cycle ergometer exercise. The authors concluded that the increases in  $\dot{V}O_2$  observed may be attributed to an increase in work required to move the mass of the legs. This finding has also since been corroborated by Francescato et al. (1995). They used weights to increase the lower limb mass and concluded that a greater lower limb mass significantly increases the oxygen cost of pedalling. However they linearly extrapolated the metabolic cost of moving the legs from an unloaded pedalling condition. This has been criticized by Kautz and Neptune (2002) who suggest it is a flawed concept claiming that the metabolic cost of moving the legs during unloaded pedalling has no relationship to GE measured during loaded cycling.

The efficiency of transfer of power from the human body to the drive chain of the bicycle depends upon crank length (Inbar et al., 1983; Hull &

Gonzales 1988; Gonzales & Hull, 1989), longitudinal foot position on the pedal (Hull and Gonzales, 1988), seat height (Shennum & de Vries, 1976; Nordeen-Snyder, 1977; Gonzales & Hull, 1989), seat tube angle (Gonzales & Hull, 1989; Browning et al., 1992), and as already discussed, pedal cadence (Boning et al., 1984; Gonzales & Hull, 1989). Alterations in these bicycle parameters have been shown to influence metabolic efficiency measured during cycling.

The seat tube angle (STA) is measured as the position of the seat relative to the crank axis of the bicycle. Research on the effect of varying STA's on metabolic variables has demonstrated acute changes. Nordeen-Snyder (1977) and de Vries (1976) have shown that as seat height varied a setting exists at which  $\dot{V}O_{2\text{submax}}$  is minimised (100% trochanteric height) and power output is maximised, thus acutely optimising efficiency. Either side of this 100% trochanteric height efficiency was decreased. Similarly Heil et al. (1995) found that maximum values for  $\dot{V}O_2$ , HR and RPE varied with changes in STA. More specifically for a STA at 83° and 90°  $\dot{V}O_2$ , HR and RPE were significantly lower in a group of trained cyclists compared to 69°. Therefore this suggests that changes in mean hip angle acutely alters cardiovascular stress for a given power output (Heil et al., 1997). Price and Donne (1997) similarly found an energetic optimum for combinations of STA and seat height (70° STA; 100% trochanteric height) in a group of trained cyclists. However they suggest acute physiological alterations caused by changes in STA appear to be independent from changes in seat height. Alterations in the muscle force length relationships (quadriceps versus hamstrings) and

ankling patterns could account for the differences in efficiency they found with increased STAs. When the STA is shifted from shallow to steep angles (69° to 90°) a greater flexion at the ankle occurs which alters muscle fibre recruitment and there is a change in lower limb orientation that places the rotation of the legs more directly over the crank axis. Thus changing STA acutely changes metabolic efficiency.

Studies have also investigated alterations in handle bar position and height. Ryschon and Stray-Gundersen (1991) found no significant difference in  $\dot{V}O_{2\max}$  between dropped and upright seated positions. Although Origenes et al. (1993) did not detect any changes in  $\dot{V}O_2$  with changes in upper body posture in inexperienced cyclists. Specific chronic adaptations related to position (and muscle length tension relationships) may therefore only occur with years of training. Thus a trained cyclist may become adapted to the cycling position that they use over a period of time. Consequently it is intuitive to suggest that the trained cyclist must be assessed in the same position when performing testing in a laboratory environment. The ergometer used should be set according to the relative dimensions of the participant's own bicycle, any deviations from the cyclist's chronically adapted posture may acutely change their efficiency. These ergometer settings should then be replicated during all subsequent tests. It is also important for the cyclist to maintain a consistent position on the ergometer throughout the exercise test as changes in posture from a standing, to an upright seated, to a dropped handle bar position may acutely affect efficiency values.

### 1.3.6 Maximal Oxygen Uptake

Künstlinger et al. (1985) and Moseley and Jeukendrup (2001) found weak correlations between  $\dot{V}O_{2\max}$  and GE ( $r=0.49$ ,  $p<0.05$ ), suggesting that a high aerobic capacity may be linked to a high efficiency. In contrast Lucia et al. (2002a) suggest an inverse correlation exists between  $\dot{V}O_{2\max}$  and efficiency. They propose a high efficiency may compensate for a relatively low  $\dot{V}O_{2\max}$  in the homogenous group of 'world-class' cyclists they studied. Hunter et al. (2005) also support this inverse relationship in a group of untrained participants. They suggest the inverse relationship may be due to fibre type differences between individuals. Hunter et al., (2005) demonstrated a high  $\dot{V}O_{2\max}$  to be more strongly related to a high proportion of Type IIa muscle fibres than Type I. Conversely exercise economy/efficiency is more strongly related to a high proportion of Type I muscle fibres (Coyle et al., 1992; Horowitz et al., 1994; Mogensen et al., 2006). Thus given the training undertaken by elite cyclists it is possible that they develop a higher aerobic capacity in their Type IIa fibres, thereby increasing their efficiency, and decreasing their  $\dot{V}O_{2\max}$ .

Moseley et al. (2004) do not support the concept of an inverse relationship between  $\dot{V}O_{2\max}$  and GE. They found no correlation between GE at 165W and  $\dot{V}O_{2\max}$  across both trained and untrained cyclists. The reason for discord between these studies is not clear. However it may be related to the differences in participant's cycling experience. A greater range of abilities are evident in the participant population of Moseley et al. compared to that

of Lucia et al. (2002a). The inverse relationship may only be seen in highly trained cyclists. Atkinson et al. (2003b) have criticised Lucia's findings by suggesting that a spurious correlation could account for much of the relationship found. Lucia et al. (2002a) also used an automated breath-by-breath system (CPX/D; Medical Graphics: St. Paul, MN) which has been shown to significantly underestimate (10.7-12.0%)  $\dot{V}O_2$  at power outputs between 100 and 300W compared to an automated Douglas bag system (Gore et al., 2003). Thus equipment error could also account for the relationship seen. If absolute values for  $\dot{V}O_2$  decline due to equipment drift this would not only lower  $\dot{V}O_{2max}$  but also raise GE. Given the equivocal findings outlined above, the relationship between training status,  $\dot{V}O_{2max}$  and GE remains to be fully explored with a need for longitudinal study methodologies.

### **1.3.7 Cycling experience and the effect of training**

A question which has been addressed by several studies, but which is still inconclusive, is whether cycling efficiency can be increased by training. Lucia et al. (1998) demonstrated professional cyclists have a lower  $\dot{V}O_2$  than elite cyclists at a power output of 300W during an incremental test, although efficiency was not calculated. Similarly Künstlinger, et al. (1985) found that trained cyclists have a lower  $\dot{V}O_2$  than untrained at low to moderate work rates, during a prolonged endurance test. A significant difference in GE (22% vs. 19%) between 5 trained and 5 untrained participants was also observed by Jansson and Kaijser (1987). In this study large differences were observed in  $\dot{V}O_{2peak}$  (4.81 vs. 3.29 L·min<sup>-1</sup>), oxidative

enzyme activities (100% greater in trained) and muscle fibre composition (70% vs. 40% Type I fibres) of the 2 groups. This raises the possibility that the cumulative effects of training cause adaptations in muscle morphology which in turn increases GE.

Data from Lucia et al. (2002a) investigating “world class” road cyclists reported an average gross efficiency of 24.5% with a peak of 28.1%. This data is considerably higher than the range of 17-23% reported in most cycling efficiency research (Moseley & Jeukendrup, 2001; Coast et al. 1986, Coyle et al. 1992; Horowitz et al. 1994). Whether these differences are the result of training or a genetic natural selection process to succeed as a high level cyclist is unclear. However the study has come under some criticism due to the extreme values reported on the grounds of errors within the methods and gas analysis system used (Jeukendrup et al., 2003). Therefore it is important to develop experimental methods that are valid and reliable when attempting to calculate efficiency.

Gardner et al. (1989) and Gissane et al. (1991) have both shown training can improve exercise efficiency, albeit in previously untrained individuals. A more relevant population was studied by Barbeau et al. (1993) who investigated 7 elite cyclists physiological adaptations over a continuous 9 month period of training. They found increases in GE at power outputs of 200 and 250W, although interestingly no change at 150W. This may indicate that increases in efficiency may be specifically related to time spent at a particular power output, as most quality training would be conducted at



moderate to high intensities (200W or above). Unfortunately Barbeau et al. (1993) do not present any training data to support this speculation. More recently Sassi et al. (2008) have demonstrated a 1.1% increase in GE over the course of a 6 month period in 8 professional road cyclists and 5 mountain bikers. As with Barbeau et al. (1993), Sassi et al. (2008) do not present any training data. It is therefore unclear why GE increased as the cyclists were from quite different disciplines of the sport. The changes in efficiency that Sassi et al. (2008) found were not significant, possibly due to the low statistical power in their method of data collection. It is also problematic that the authors used a constant power test at a relative rather than absolute power. On subsequent analysis of their results it is therefore not possible to rule out that the changes in GE were due to decreases in the relative work rates during the constant power test when expressed as a percent of the cyclist's peak power output.

Coyle's (2005) case study on a Grand Tour champion documents an 8% improvement in efficiency over a seven year period. However this study has come under severe criticism by Schumacher et al. (2005), Martin et al. (2005) and Gore et al. (2008). These critiques point to limitations in the study design and method. In the first instance even though the athlete in question was tested five times over the 7-year period of study only the first and last test were completed at the same time of the cycling season. It is therefore difficult to distinguish seasonal and maturation effects. Martin et al. (2005) further criticise Coyle's paper by suggesting that just attributing improvements in performance to efficiency is speculation. Coyle attributes

much of the physiological reason for improvements in efficiency down to fibre type adaptations (speculating that the rider's fast twitch fibres took on more of a slow twitch role), even though no muscle biopsies were taken and only one participant was used. It could be suggested just as likely an explanation would be that these improvements occurred due to modifications in diet, chemotherapy (therefore loss of lower limb mass), and training for improvements in aerobic power, unfortunately there is no evidence in Coyle's manuscript to support any of these speculations.

In contrast to the studies above, others have failed to find any differences in efficiency between trained and untrained cyclists (Boning et al. 1984; Marsh et al., 2000). Nickleberry and Brooks (1996) and Moseley et al. (2004) have concluded that there are no significant differences in energy input ( $\dot{V}O_2$ ), GE and DE between competitive and recreational cyclists during a graded exercise test. Marsh and Martin (1993) have shown prior cycling experience to have no effect on either preferred or most economical pedalling cadence between trained and untrained cyclists. They conclude whole body muscular efficiencies must be similar between the groups studied. Interestingly however, no efficiency data was provided to support this assertion ( $\dot{V}O_2$  data only). Although if this is the case and training status is not an important factor determining efficiency, then efficiency is likely to be genetically determined.

A number of the papers listed above could be criticised based on the specific methods that have been employed to assess efficiency and failure to address

the risk of committing a type 2 statistical error; lack of sample size (Boning et al., 1984; Marsh & Martin, 1993; Nickleberry & Brooks, 1996; Marsh et al., 2000), use of low power outputs (Boning et al., 1984; Nickleberry & Brooks, 1996; Marsh et al., 2000), short stage protocols which do not ensure steady state conditions (Boning et al., 1984; Marsh & Martin, 1993; Nickleberry & Brooks, 1996; Marsh et al., 2000; Moseley et al., 2004) and artificially imposing cadences (Boning et al., 1984; Marsh & Martin, 1993; Nickleberry & Brooks, 1996; Marsh et al., 2000; Moseley et al., 2004).

### **Criticisms of past research**

#### a) Sample Size

Several of the studies outlined in Table 1 have few participants in their sample; this is further confounded by subdividing them into two or more groups. None of the studies presented in Table 1 report statistical power within their methods. It may be that many of their findings are due to statistical failings (i.e. committing a type 2 error). Given a large enough sample size and a small enough error variance, it may be possible to detect statistical differences between trained and untrained populations. From the modelling research outlined earlier in this chapter, a 1% difference in performance (Moseley & Jeukendrup, 2001) between trained and untrained cyclists reported in several of the studies in Table 1, is of practical significance and would account for some of the differences in performance between the groups. Taking the data of Nickleberry and Brooks (1996) as an example, it is possible to further illustrate the impact of a lack of statistical power on a research method and its subsequent conclusions. Nickleberry

and Brooks report no significant difference in cycling efficiency exists between trained and recreational cyclists (12 participants split into two groups; trained cyclists N=6; recreational cyclists N=6). At 250W they report a mean difference for GE of 1.7% between the groups, which was not significant. This would be seen as being practically significant in terms of a performance trial (9% power difference in an hour time trial using data from Horowitz et al. [1994]). Using the mean GE (19.8%) and Coefficient of Variation (upper 95% CI = 6.4%) from Moseley and Jeukendrup's (2001) investigation of the reliability of cycling efficiency, and the sample size calculation methods of Hopkins (1997) ( $n=64s^2/d^2$ , where  $s$  = the within-participant standard deviation, i.e. the CV from Moseley and Jeukendrup (2001);  $d$  = smallest worthwhile post-pre difference to detect) , it is possible to determine that Nickleberry and Brooks (1996) would have need 19 participants to determine a significant difference between the groups. Thus Nickleberry and Brooks could have inadvertently committed a type 2 statistical error. As illustrated in Table 1 it is clear that many of the non significant findings previously reported may not have come to the same conclusions had they calculated sample size based on prior reliability data for efficiency and/or the smallest effect worth detecting.

Author(s)	Sample Size	Stage Duration	Power Outputs used	Efficiency measure	Cadence imposed	Effect Size / Statistical Power	Efficiency finding
Nickleberry & Brooks (1996)	12 (6 competitive; 6 recreational)	4 mins	50-200W	DE	Yes (50 and 80rev.min <sup>-1</sup> )	0.71 / 0.20 at 200W	No significant difference (>1% difference between groups)
Marsh et al. (2000)	31 (11 competitive cyclists; 10 trained runner; 10 non-cyclists)	5 mins	Trained cyclists; 100, 150, 200W Untrained 75, 100, 150W	DE	Yes (50, 65, 80, 95, 110rev.min <sup>-1</sup> )	1.25 / 0.77 at 80 rev.min <sup>-1</sup>	No significant difference (~1% difference between groups at 80 & 95rev.min <sup>-1</sup> )
Boning et al. (1984)	15 (9 trained cyclists; 6 untrained)	3 mins	50, 100, 200W	GE & NE	Yes (40, 60, 70, 80, 100rev.min <sup>-1</sup> )	0.87 / 0.33 at 200W	GE ( $P<0.05$ ) & NE (both 1% mean difference)
Moseley et al., (2004)	69 trained cyclists (divided on $\dot{V}O_2$ peak, low, medium and high)	3 mins	95W increasing by 35W	GE & DE	Yes (80-90 rev.min <sup>-1</sup> )	2.33 / 1.00 between low and high $\dot{V}O_2$ peak groups	GE & DE no significant difference (GE 0.9% difference Med-High groups; DE 1.2% difference Low to High groups)
Mogensen et al. (2006)	18 (9 trained cyclists; 9 untrained)	5 mins	40, 80 & 120W	GE & DE	Yes (80rev.min <sup>-1</sup> )	3.33/0.99 for GE 0.44 / 0.93 for DE	No significant difference between groups ( $P<0.05$ )

Table 1. Methods used for the investigation of differences in efficiency between trained and untrained populations

b) Protocol stage durations

The ability to detect changes in cycling performance over time and/or study the effects of selected interventions/treatments require testing methods and equipment to be both valid and reliable (Atkinson & Nevill, 1998; Hopkins et al. 1999). In order to measure efficiency reliably all gas collections must take place under steady state exercising conditions, otherwise measured pulmonary  $\dot{V}O_2$  will not reflect muscle  $O_2$  consumption (Poole et al., 1992). At the onset of exercise or in the transition from one power output to another, during light to moderate intensities, pulmonary  $\dot{V}O_2$  increases in a mono-exponential manner to reach a steady state within 2-3 minutes (Whipp & Wasserman, 1972; Whipp, 1987; Whipp, 1994). Thus protocols with 3 minute stages or less would potentially not allow steady state  $\dot{V}O_2$  to occur (Wasserman et al., 2005). Heavy exercise intensities, characterised by a sustained metabolic acidosis, have been found to result in a delayed steady state or in severe exercise for  $\dot{V}O_2$  to continue rising to  $\dot{V}O_{2peak}$  and ensuing exhaustion (Whipp, 1987; Poole et al., 1988; Whipp, 1994). Consequently, the relationship between  $\dot{V}O_2$  and power output presents a marked deviation from linearity at higher intensities when examined carefully (Zoladz et al., 1995). The exercise intensities eliciting this additional  $O_2$  consumption, or  $\dot{V}O_2$  slow component, should not be used to determine efficiency values. This is because the calculated energy expenditure will be heavily dependent upon the timing of the expired gas collection and thus the extent of a  $\dot{V}O_2$  slow component.

The calculated energy equivalent for a given  $\dot{V}O_2$  depends upon the equivalence of RER and muscle RQ. During non-steady state exercise resulting in a metabolic acidosis, plasma bicarbonate buffering will result in “extra”  $CO_2$  production being measured in pulmonary gases. Whipp (1987) indicates that under such non-steady state conditions  $CO_2$  may be transiently stored in muscle and blood. Even where pulmonary gas exchange is measured under exercising steady state conditions, whole body RER does not necessarily reflect muscle RQ (Riley et al., 1996) as RER is an integrated response of all metabolically active tissues in the body, not just muscle. Surprisingly however, Romijn et al. (1992) have found indirect calorimetry to provide a valid means of estimating oxidised substrate even at relatively high exercise intensities ( $\sim 85\% \dot{V}O_{2peak}$ ). Even so any errors in RER measurement are likely to have an impact on the estimated energy expenditure. Decreasing RER by 0.05 reduces calculated energy expenditure by 1.3% typically increasing GE by 0.4%. As  $CO_2$  production is used with the calculation of RER it is important to ensure it’s stability prior to taking efficiency measurements. Slow kinetics of  $\dot{V}CO_2$  (in relation to  $\dot{V}O_2$ ), mean that it does not stabilise until  $\sim 3-4$  minutes of a constant load test (Whipp, 1987; Chuang et al., 1999; Wasserman et al., 2005). Therefore any measurement of efficiency should not commence until after 5 minutes of steady state exercise. Many of the protocols used in studies outlined in Table 1 do not allow time for this to occur.

### c) Power Outputs

Efficiency has been shown to increase with increasing power outputs (Gaesser & Brooks, 1975; Seabury et al., 1977; Stuart et al., 1981; Coast & Welch, 1985; Croisant & Boileau, 1984; Banister & Jackson, 1967; Chavarren & Calbert, 1999). Figure 3 illustrates a high consistency of the effect of work rate on cycling efficiency compared to the plot of cadence (Figure 2), using the same pool of data. McDaniel et al. (2002) suggest that 95% of all variation in metabolic cost (comparing cadence, work rate and movement speed by altering crank length), is explained by work rate.

As stated in section 1.2, the curved work rate-gross efficiency relationship (shown in Figure 3) is a consequence of the diminishing contribution of energy expended to sustain body position on the bike and other bodily processes to total energy expenditure at higher power outputs (Stainsby et al., 1980). Moseley and Jeukendrup (2001) suggest that a plateau occurs in the work rate-gross efficiency relationship at higher workloads (>200W) which are more functional for the trained cyclist.



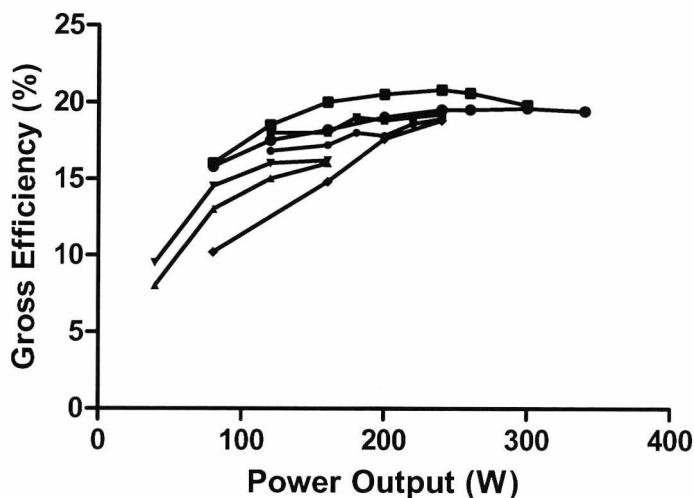


Figure 3. The effect of work rate on efficiency in cycling using pooled data from previous research studies (Gaesser & Brooks, 1975; Nickleberry & Brooks, 1996; Sidossis et al., 1992; Chavarren & Calbet, 1999; Moseley & Jeukendrup, 2001; Foss & Hallen, 2004; Lucia et al., 2004; Moseley et al., 2004; Mora-Rodriguez & Aguado-Jimenez, 2006; Samozino et al., 2006)

A particular criticism of past research is in relation to the exercise intensities used for assessing efficiency during cycling. Many previous research studies using cycle ergometry have focused researching the energetic optimum (Gaesser & Brooks, 1975, Hagberg et al., 1981; Seabury et al., 1977) and have used untrained participants. Therefore only low absolute power outputs (31 – 123W) have been required to be sustained for a period of time. These values are very low for the trained cyclist and thus are not reflective of powers commonly used by the road racing cyclists. As a consequence they could be considered invalid when considering this population.

#### d) Imposed Cadence

Many of the studies outlined in Table 1 have imposed artificial cadences on their cyclists. Hansen and co-workers (2002) have demonstrated that self-selected cadence closely matches the rate of peak efficiency within the muscle fibres used. As previously discussed, cadence could be considered as a highly individualised parameter and setting a standardised rate for all participants may therefore be problematic. If standardised across all participants some would be forced to ride using a cadence that is sub-optimal, thereby their decreasing efficiency. Interestingly this is evident in Coyle's (2005) case study on a Grand Tour champion. It is well known that this cyclist's cadence increased markedly (70-90 to  $>100 \text{ rev.min}^{-1}$  Armstrong & Carmichael, 2006) over the 7 year period of Coyle's study. However, he was forced to maintain a fixed cadence of  $85 \text{ rev.min}^{-1}$  by Coyle at all tests.

#### e) Summary

Given that cycling efficiency has been proposed as a key element of performance it is interesting to consider that research has found no benefit of training in cross-sectional studies. On closer examination of this work it is apparent that the majority of this work could be criticised on the grounds of the methods used. A couple of longitudinal studies have investigated the influence of training on efficiency of trained cyclists, although these too could be criticised on the basis of the methods used to assess efficiency.

## **1.4 Currently proposed mechanisms for training related changes in efficiency**

It has been speculated that metabolic efficiency during cycling increases with training (Barbeau et al., 1993; Coyle, 2005; Sassi et al., 2008), however there is little evidence that this is the case as these studies have tended to lack scientific rigor. Therefore there is no real direct evidence for mechanisms by which efficiency may increase with training. As previously outlined, investigative and cross-sectional study designs have largely been utilised to identify relationships between efficiency and muscle fibre type, mitochondrial adaptations and muscle recruitment. Based on these suggested factors which affect efficiency, the section below will discuss evidence presented in the research literature for potential mechanisms that may lead to chronic training related changes in the efficiency of cycling.

### **1.4.1 Muscle fibre type transformation**

Several authors (Coyle et al., 1992; Suzuki, 1979; Horowitz et al., 1994) have demonstrated that efficiency is correlated to the percentage of Type I muscle fibres. However, fibre type only explained 56-72% of the observed variations in gross and delta efficiencies found (Coyle et al., 1992). The discussion below will consider the potential for fast-to-slow twitch fibre type transformations which could be a beneficial adaptation that may increase efficiency in cycling.

Fibre type transformations have been shown to occur under the influence of various factors and conditions. Generally, increased neuromuscular

activity/overload elicits transformations from fast to slow, whereas reduced levels of neuromuscular activity/unloading lead to transitions in the opposite direction (Tipton, 1996). Several investigators have confirmed the plasticity of mammalian skeletal muscle utilising techniques such as cross-reinnervation (Barany & Close, 1971), chronic low frequency electrical stimulation (Maier et al., 1988; Jarvis et al., 1996; Windisch et al., 1998) and compensatory threshold (Ianuzzo et al., 1976; Roy et al., 1985). Such studies have shown that it is possible to modify the contractile and histochemical properties of muscle fibres by electrical stimulation with a new impulse pattern, although they do not rule out the possible influence of some unknown factor. For example, following 28 days of chronic low frequency electrical stimulation, an approximate 6-fold increase in mitochondrial content in the superficial portion of a rabbit tibialis anterior muscle has been found (Reichmann et al., 1985). These changes were accompanied by a 5 to 6 fold increase in mitochondrial key enzymes. Similarly Salmons and Sreter (1976) showed that when a fast twitch motor neuron was cross re-innervated with a slow twitch soleus muscle of a rabbit and was subjected to 24 hour sustained low frequency stimulation, the now fast contracting Soleus muscle once again took on slow-twitch characteristics.

Given the nature of endurance training in good amateur/professional cyclists (several hours per day at low force and movement speeds), it is suggested there could be the possibility for low frequency stimulation induced transitions from Type IIB to Type IIA and ultimately Type I as outlined in

the animal experiments above. Conversion of skeletal muscle fibres from IIB / IIX to Type IIA have been shown through extensive endurance training (Jansson & Kaijser, 1977; Saltin & Gollnick, 1983), although researchers have failed to find firm evidence of transition to Type I. Therefore the so-called 'glycolytic' fibres (Type IIB / IIX) will respond to enhanced neuromuscular contractile activity with increases in their aerobic-oxidative potential (transformed to IIA) by pronounced increases in mitochondrial content and elevated levels of enzymes involved in substrate oxidation (Holloszy & Coyle, 1984). This has led to the formulation of a concept called 'adaptive range', which demonstrates the adaptive possibilities for each muscle fibre types (Westgaard & Lømo, 1988; Gundersen, 1998; Windisch et al., 1998).

The majority of research on fibre type transformation has been conducted on animals with little corresponding research on humans following endurance type training (Saltin et al., 1977; Green et al., 1991; Houmard et al., 1993; Thayer et al., 2000). The majority of investigations that have been conducted on humans are cross-sectional in nature and so the influence of genetic differences between trained and untrained groups require verification. Recently Mogensen et al. (2006) found no differences in fibre type between trained ( $\dot{V}O_{2max} >55\text{ml.kg.min}$ ) and untrained participants ( $\dot{V}O_{2max} <45\text{ml.kg.min}$ ) using a cross-sectional approach, although GE was related to the percentage of Type I fibres. They also found no significant difference between the two groups in terms of GE at absolute power outputs. However the trained participants had a significantly higher GE (1.2%) at a

relative intensity of 80%  $\dot{V}O_{2peak}$ . Although it could be suggested that this may simply be due to a decrease in the relative significance of basal energy expenditure of the trained compared to the untrained participants as they were cycling at a higher absolute power output. There is also likely to have been significant metabolic acidosis (and therefore  $\dot{V}O_2$  slow component) in the untrained group when cycling at this high intensity.

Interestingly Mogensen et al., (2006) present a mean difference in absolute GE of 1% at 80 and 120W which research (Horowitz et al., 1994; Jeukendrup et al., 2000; Moseley & Jeukendrup, 2001) would suggest will account for large differences in performance between the two groups. These findings should also be interpreted with caution as their trained group were not competitive cyclists (mean  $\dot{V}O_{2max} >55\text{ml.kg.min}$ ), nor did they use cycling as a part of their training regime.

Only Simoneau et al. (1985) and Howald (1985) have reported the number of Type I fibres to significantly increase following 6 – 15 weeks of high intensity aerobic interval training. In one of the few longitudinal studies (28 males; 11 highly trained endurance athletes, 10 fitness trained and 7 untrained) Trappe et al. (1995) investigated changes in human gastrocnemius muscles over a 20 year period. They found significant increases in the percentage of Type I muscle fibres in untrained as well as trained participants, however it is hard to reconcile whether this is a direct result of endurance training undertaken as no supporting data or information is provided.

The effects of endurance training are not only on the contractile system of muscle fibres. Adaptations that might affect efficiency, which are detectable early in a training programme, are not of the myosin using ATP, but of the oxidative systems in supplying it. Mitochondrial volume and aerobic capacity increase greatly, especially in Type II fibres, and anaerobic capacity decreases (Holloszy, 1975; Jansson & Kaisjer, 1977; Saltin & Gollnick, 1983). Supply of oxygen to the muscle fibres is enhanced by increased capillarisation (Anderson & Henricksson, 1977; Ingjer, 1979) and increased myoglobin content (Hickson, 1981; Harms & Hickson, 1983). This would in turn suggest the potential for a shift towards a less wasteful ATP consumption by the myosins and thus the potential for enhanced metabolic efficiency whilst cycling. These parameters will be discussed in more detail in section 1.4.2 below.

### **Summary**

Previous work has demonstrated that there is a positive relationship between percentage Type I fibres and efficiency (Coyle et al., 1992; Horowitz et al., 1994; Mogensen et al., 2006). Thus it is intuitive to expect that the greater the proportion of these fibres within working muscles, the greater the efficiency. As demonstrated in the research above one would assume that efficiency is increased as a result of years of endurance training (Lucia et al., 2002a; Coyle, 2005), which has been shown in runners using an economy measurement (Conley et al., 1984; Morgan et al., 1995; Franch et al., 1998; Jones, 1998; Billat et al., 1999; Jones et al., 1999). It is therefore puzzling

that current research has consistently demonstrated no differences in efficiency between trained cyclists and untrained individuals. As previously discussed, examining the current efficiency literature in more detail identifies cases in which type 2 statistical errors may have been committed. It is therefore possible that differences between trained cyclists and untrained participants do exist, but the methods used may not have been able to detect them. If the trained cyclist has no efficiency advantage over the untrained cyclist then the findings of running exercise studies are contradictory (inter-participant differences of 12-15%; Bransford & Howley, 1977; Morgan et al., 1995; Carter et al., 1999).

On the premise that efficiency is linked to the percentage of Type I fibres (Coyle et al., 1992; Horowitz et al., 1994; Mogensen et al., 2006) a literature search demonstrates that it is clear that differences in efficiency should exist between trained and untrained populations. Cross-sectional studies have demonstrated fibre type differences exist between trained and untrained participants. Whether this is due to a process of training or natural selection is not clear. However, evidence from longitudinal training studies has shown the possibility for fibre type transformation of various degrees. The research outlined above demonstrates that if athletes train for a prolonged period of time (in excess of 12 weeks) they are likely to adapt their muscle fibre type according to the type of stressor applied.



#### **1.4.2 Aerobic enzyme capacity, UCP3 and PGC-1 $\alpha$ coactivators**

At the muscle level, O<sub>2</sub> utilisation depends on both capillary and mitochondrial status. It has been demonstrated that mitochondria are markedly affected by exercise training (Holloszy & Coyle, 1984). Endurance training at a submaximal intensity has been shown to increase mitochondrial content ranging from 50-100% within 6 weeks (Gollnick et al., 1973; Fitts et al., 1974). More specifically mitochondrial volume and aerobic capacity increase greatly, especially in Type II fibres, and anaerobic capacity decreases (Holloszy, 1975; Jansson & Kaisjer, 1977; Saltin & Gollnick, 1983). Similarly alterations in muscle redox potential and high energy phosphate flux due to muscle contraction experienced during exercise has been shown to induce mitochondrial activation and biogenesis in muscle cells (Hood, 2001). Significant increases in Cytochrome c concentration in slow twitch muscle fibres has been shown to be proportional to the frequency of endurance training (Hickson, 1981; Henriksson et al., 1986); with increases seen within 10 days of the commencement of training. The resultant increased capacity to generate energy via aerobic mechanisms may have the potential to decrease the submaximal exercise O<sub>2</sub> cost. However, Mogensen et al. (2006) found no differences in oxidative phosphorylation or mitochondrial volume comparing muscle biopsies taken from trained and untrained human individuals. They also found no differences in mitochondrial efficiency (P/O ratio) during state 3 respiration (maximal ADP stimulated respiration), and reported no correlation between mitochondrial efficiency and cycling efficiency. Incidentally they used isolated muscle preparations, and so it is

impossible to exclude the mitochondrion as a determining factor for the efficiency of muscular work during in vivo conditions. In vivo other factors which may vary between individuals could influence the mitochondrion such as fatty acids, free radicals and membrane potentials (Kadenbach, 2003). In turn this would thereby affect the efficiency of the muscular work on an individual basis.

Mitochondrial uncoupling protein 3 (UCP3) content within the muscle may also be a determining factor of metabolic efficiency. This protein has been suggested to be involved in thermogenesis by mediating mitochondrial proton leak. This means that energy is dissipated as heat as opposed to being conserved in the form of ATP (Boss et al., 2000). Cycling efficiency has been shown to be inversely related to UCP3 content (Schrauwen et al., 1999; Mogensen et al., 2006). Muscle content of UCP3 has also been shown to be reduced by endurance training (Russell et al., 2003; Fernstrom et al., 2004; Schrauwen et al., 2005) and is lower in Type I muscle fibres (Russell et al., 2003). Cross-sectional studies have demonstrated that, compared to untrained people, endurance trained participants exhibit lower UCP3 expression at mRNA (Schrauwen et al., 1999; Russell et al., 2002) and protein levels (Russell et al., 2003; Mogensen et al., 2006). Therefore UCP3 production may be an important determinant of cycling efficiency.

Iaia et al. (2009) and Daussin et al. (2008) found a reduced submaximal oxygen uptake at fixed work rates following intensive interval training (6-8 weeks). They speculate the decreases in submaximal oxygen uptake were

due to changes in the working muscle's oxidative capacity and metabolic processes. Burgomaster et al. (2008) further suggest that the production of adenosine monophosphate activated protein kinase released as a result of intensive training (Koullmann & Bigard, 2006; Gibala et al., 2006), may cause the up-regulation of the transcriptional coactivator peroxisome proliferator-activated receptor  $\gamma$  coactivator  $1\alpha$  (PGC- $1\alpha$ ) which in turn regulates mitochondrial biogenesis in Type I, IIa and IIx fibres (Russell et al., 2003; Taylor et al., 2005; Terade et al., 2005).

PGC- $1\alpha$  is at the nexus of the regulation of gene expression programs needed for skeletal muscle adaptations to increased work demands (i.e. training). In a novel study Handschin et al. (2007) investigated the role of PGC- $1\alpha$  by training mice deficient in PGC- $1\alpha$ . They found these mice exhibited a shift in fibre type from Type I and IIa towards Type IIb and IIx fibres, with corresponding decreases in endurance capacity. Similarly mice with hind leg motor denervation have been shown to maintain skeletal muscle function and tone in the absence of motor neuron signaling with ectopic expression of PGC- $1\alpha$  as a result of protein kinase injection (Sandri et al., 2006). This therefore suggests that the protein coactivator PGC- $1\alpha$  could have a key role to play in determining efficiency in cycling due to mitochondrial biogenesis and fibre type transformation as it is activated as a result of strenuous endurance training. Mitochondrial biogenesis and fibre type transformation towards Type I is likely to increase the oxidative potential of the muscle and in turn decrease the oxygen cost of submaximal exercise. However, research is needed to ratify this speculation.

Most of the research outlined above has been conducted on animals or low to moderately trained individuals. Whether comparable improvements would be seen in trained cyclists remains to be determined. If similar improvements were possible in trained cyclists as a result of their routine training (as high intensity training is commonly used), it would further add support to the argument that significant differences in efficiency should exist between trained cyclists and untrained individuals.

### **1.4.3 Cadence and muscle fibre type transformation**

During active muscle shortening, thermodynamic efficiency is the same (17–27%) in both Type I and Type II muscle fibres. However peak efficiency is reached at different velocities (at around 15% of maximum shortening velocity in both cell Types) (He et al., 2000). Thus improved efficiency at higher cadences may be a reflection of the fact that the lower extremity muscles responsible for meeting the power output demands of the task may be closer to their optimal shortening velocity (Sidossis et al., 1992).

At 80 rev.min<sup>-1</sup> Type I muscle fibres of the Vastus Lateralis muscle are closer to their peak efficiency contraction velocity than Type II (Coyle et al., 1991). Lucia et al. (2004a) demonstrated that when professional cyclists ride at 366 ± 37W (75%  $\dot{V}O_{2max}$ ) lactate concentration is higher when pedalling at 60 rev.min<sup>-1</sup> in comparison to 80 or 100 rev.min<sup>-1</sup>. They hypothesised that this high power output using a cadence of 60 rev.min<sup>-1</sup> produces a greater recruitment of Type II fibres (Ferguson et al., 2001), thus leading to higher

lactate production, increased oxygen cost and therefore lower efficiency. Thus the effect of fibre-type distribution on whole-body efficiency when cycling may not be due to differences in contractile efficiency between myosin isoforms. Rather it may be due to the shortening velocities during cycling (i.e. cadence) being closest to those associated with peak efficiency in Type I fibres. Trappe et al. (2006) have demonstrated increases in peak shortening velocity of Type I fibres as a result of marathon training. Consequently one would assume that this would also influence the shortening efficiency at which peak efficiency is attained. Therefore if the runners in Trappe's study were able to better match the new 'most efficient' shortening velocity with that actually used during running it could be assumed that running efficiency would be improved. Thus there could be a mechanism by which muscle fibres could provide improved contractile efficiency in response to training, without any change in metabolic efficiency. Given the findings of Trappe et al. it is further puzzling that no differences in whole body efficiency have been reported between trained cyclists and untrained individuals during cycling exercise.

## 1.5 Overall Summary

An interest in the efficiency of human movement can be traced to the antecedents of modern human physiological research. Since this time considerable data have been published on human efficiency. Of the many factors known to influence efficiency; cadence, work rate and cycling position have been demonstrated to exert a significant effect. These parameters must be strictly controlled throughout comparative work on efficiency.

The inability to find differences between trained cyclists and untrained persons has limited the progress of efficiency research in the literature over the past decade. However on close scrutiny of the literature, evidence is available to suggest that differences in efficiency should exist between trained cyclists and untrained individuals given its physiological determinants. Type I muscle fibres are thought to contract with a greater efficiency than Type II fibres at cadences typical of endurance cycling. This morphological characteristic of the active skeletal muscle mass has been established to be largely responsible for determining efficiency and the success in endurance performance of individuals with similar aerobic fitness. Despite this evidence it is strange that studies have not reported that efficiency is sensitive to training over a short-term period, or that there are differences between highly trained endurance athletes and untrained individuals.

The lack of longitudinal studies investigating efficiency has also limited understanding of the degree to which adaptations in efficiency occur with training. It is clear that the influence of training status on efficiency remains to be fully established using sound methods. There is also a need to conduct further research into the impact of training on efficiency using longitudinal study designs. Further research is warranted to ascertain whether efficiency is affected during a period of training/detraining similar to that found during a normal competitive season. Carefully planned studies, using repeated measures designs still need to be conducted before a measurable link between training and efficiency can be discounted. Many studies have also not appropriately selected from a large enough or representative sample group to effectively identify differences between trained and untrained populations. Muscle typology is unlikely to be the sole determinant of cycling efficiency. Several studies have found that the changes in cycling efficiency could be induced by alterations in muscle recruitment and pedalling strategies.

## **1.6 Research aims and Objectives**

The aim of this research was to investigate the effect of training on metabolic efficiency in cycling.

The objectives were identified as being:

- To develop a protocol capable of detecting small changes in efficiency that is based upon sound scientific and physiological principles.
- To compare the effect of training on cycling efficiency using a cross-sectional study design with adequate statistical power.
- To explore the changes in cycling efficiency that occur over a year's worth of training and racing in competitive cyclists, and to evaluate the relationship between efficiency and training.
- To evaluate the impact of two different intensities of training on efficiency in cycling.



## **CHAPTER 2: GENERAL METHODS**

### **2.1 Preamble**

This chapter will detail the general methods used throughout the thesis which include the measurement of; body mass and stature, energy expenditure via indirect calorimetry from the measurement of oxygen uptake and carbon dioxide production, power output, blood lactate, heart rate and environmental conditions. All studies and procedures employed were approved by the Canterbury Christ Church University Ethics Committee.

### **2.2 Pre-test information and requirements**

Prior to all testing each participant received a written explanation of the testing procedures (see appendices 1-4) which included any potential risks and discomforts associated with participation. Participants then completed a health and medical questionnaire (see appendix 6) and gave written informed consent to take part in the study (see appendix 5). They were also informed they were free to withdraw from the study at any point and for any reason should they desire to do so. Full familiarisation took place with all protocols prior to any testing, and the importance of strict compliance with pre-experimental procedures was stressed. Prior to each laboratory visit participants were requested to ensure that they were well rested and fully hydrated. In particular, they were asked to avoid training during the day before and to prepare for each test as if it were a race. No formal dietary control was exercised however.

### **2.3 Measurement of body mass and stature**

At each visit to the laboratory, body mass was measured to the nearest 0.1kg by means of beam balance scales (Seca, Germany). Pre-trial body mass was recorded with the participants barefoot, having removed all items of clothing except the cycling shorts. Stature was measured to the nearest 0.5cm by means of a Stadiometer (Seca, Germany).

### **2.4 Measurement of respiratory gases**

Expired air was measured using a Cosmed Quark b<sup>2</sup> breath-by-breath analysis system (Cosmed Srl, Rome, Italy). Measurements were made on a breath-by-breath basis via participants wearing a mask with a built in turbine connected via a sampling tube and electronic sensor cable to the analyser. During all tests expired air was analysed for the concentrations of O<sub>2</sub> and CO<sub>2</sub> by sampling through a Zirconia temperature controlled O<sub>2</sub> sensor and an infrared CO<sub>2</sub> analyser. Prior to the start of the data collection for this thesis the Cosmed was calibrated via the use of a metabolic pump to check the accuracy of measurement against known gas values, results of which are shown in Table 2.

Table 2. VacuMed data collected from the Cosmed Quark b<sup>2</sup>

VT	RF (breaths·min <sup>-1</sup> )	Flow (L·min <sup>-1</sup> )	% $\dot{V}O_2$ Error	% $\dot{V}CO_2$ Error
1.5L	20	6.95	2.16	2.95
1.5L	40	10.95	3.17	2.55
2L	50	15.50	2.87	3.39

The Cosmed online gas analysis system was also calibrated to known gas mixtures (Oxygen 16.01%, Carbon dioxide 4.99%, Balance nitrogen, Air Gas Inc. Tulsa, USA), and the turbine was calibrated using a 3-litre syringe

(Hans Rudolf Inc. Kansas, USA) in the laboratory. Repeated trials (x3) of the analyser were performed by the known gas concentrations being passed through the system to establish agreement and reliability coefficients. The coefficient of variation for %O<sub>2</sub> was 0.2% (95%CI 0.10 – 0.43%) with that for %CO<sub>2</sub> being 0.56% (95%CI 0.51 – 0.62). Prior to each test and periodically during extended use the analyser was calibrated with both room air (20.93% O<sub>2</sub> and 0.03% CO<sub>2</sub>) and a gas mixture (16.01% O<sub>2</sub> and 4.99% CO<sub>2</sub>). If either gas concentration varied from the expected value by > ± 0.01% the calibration procedure was repeated.

A calibration syringe was used to check the volume measurements recorded by the turbine. The range of measured values was 21-201 L·min<sup>-1</sup>. The coefficient of variation for repeat assessment (x3) of expiratory volume was 0.37% (95% CI -21.63 to 22.37%) and 0.16% (95% CI -9.08 to 9.40%) for inspiratory volume. There was no significant difference ( $P>0.05$ ) in the bias recorded between actual volume (pumped past the Cosmed turbine using a 3L syringe) and the Cosmed display volume, the SEE (95%) was calculated to be 0.83%. There was a significant relationship between the volume measured and the volume recorded ( $r=0.99$ ;  $P<0.05$ ). Prior to each test the bi-directional turbine was also calibrated against the gas syringe.

Energy expenditure, CHO and fat oxidation rates were estimated from steady-state  $\dot{V}O_2$  and RER using:

Work rate energy expenditure ( $\text{kcal}\cdot\text{min}^{-1}$ ) = Power output x 0.01433

Total Energy expenditure ( $\text{kcal}\cdot\text{min}^{-1}$ ) = kcal per litre  $O_2$  consumed x  $\dot{V}O_2$

Kcal per litre of  $O_2$  consumed was calculated from the thermal equivalents of oxygen for non-protein RQ (as represented by RER) using the data of Zuntz (1901). Gross efficiency was calculated as the percent ratio of power output to power input (Gaesser & Brooks, 1975). Power output was recorded using a Lode Excalibur Sport Ergometer and power input was estimated from energy expenditure. Delta efficiency was calculated as the ratio of delta power output to delta energy expenditure (Gaesser & Brooks, 1975). Delta power output was calculated as the difference in power output between two different exercise intensities, and delta energy expenditure equalled the difference in energy expenditure between the two intensities.

### **2.5 Cycle ergometry and the measurement of power output**

All exercise testing was completed using an electromagnetically braked ergometer (Lode Excalibur Sport, Lode, Groningen, NL). The participant's habitual riding position was replicated (saddle height, reach, handle bar height, crank lengths) and measurements were recorded and reproduced for each test. STA was kept constant for each individual and participants used their own pedal/shoe combination. Calibration of the ergometer was performed prior to the start and periodically throughout the time course of

the studies in this thesis using a dynamic calibration rig (Lode Portable Calibration Rig, Lode, Groningen, NL) and was found it to be within 1% of a true value (CV = 1%, CI = 0.7 – 1.2%) for power outputs of 25 – 1000W at cadences of 40, 60, 80, 100 and 120 rev.min<sup>-1</sup>. The reliability of the Lode Excalibur Sport ergometer has also been investigated by Earnest et al. (2005) using a “human calibration” model. They found the ergometer to be very reliable allowing for good repeatability of the variables measured (CV’s%: Time to exhaustion 8%;  $\dot{V}O_{2peak}$  7%; Peak power output 6%; Heart rate peak 6%; ventilatory threshold power output 10%; respiratory compensation point power output 8%). No significant differences were identified between any of the maximal or submaximal variables on repeated testing. In addition no significant difference was noted for the  $\dot{V}O_2$  response during the submaximal tests. Thus Earnest et al. (2005) concluded that although some variance exists, it could be accounted for by the day-to-day physiological differences of humans.

Prior to each of the tests in this thesis the ergometer’s cranks were zero adjusted according to the manufacturer’s recommendations. Crank forces for both right and left cranks were measured at two degree intervals during every revolution. Strain gauges in both right and left cranks were used to obtain crank torque measurements in two dimensions; these were then converted into a digital electrical signal and registered by the computer programme. The crank mounted strain gauges were also calibrated prior to the study and periodically throughout according to the manufacturers’ recommendations, as well as via a dynamic calibration using known

calibrated weights and a calibration rig (Lode Portable Calibration Rig, Lode, Groningen, NL). During this process the calibration rig drove the ergometer at 70 and 90 rev.min<sup>-1</sup> against resistances of 150 and 300 W. Crank forces recorded on the ergometer were within 7% of the true value (SEE = 6.6%; CV% 4.8; 95%CI -4.6 – 5.3). From the crank torque measurement, total torque applied on each crank, downstroke torque between 0 - 180° tangential to the crank displacement and upstroke torque between 180 - 360° were calculated. Crank torque every 2 degrees were averaged across the sampling period for both right and left cranks. Total torque was then calculated from the sum of the right and left crank torques. Net torque was calculated as the sum of only positive torques across both crank arms, with negative torque being the difference between total and net torques. Crank Torque Effectiveness was calculated from the division of net by total torque (CTE = net torque/total torque).

## **2.6 Methods of blood sampling**

The finger-prick capillary blood sample method was used for all blood collection. A capillary tube whole blood sample was taken for the determination of blood lactate concentration (Biosen C-Line, EKF Industrie, Elektronik GmbH, Barleben, Germany). Prior to sampling the fingertip was wiped with a medi-swab and the first drop of blood was discarded. Free-flow blood was collected in a 20-microlitre glass capillary tube and immediately mixed with a lysing stabilising agent in a safe-lock vial. This involved shaking the sealed vial for approximately 15-s. Samples were analysed within 10-min of completion of the test using a Biosen C-Line

analyser (EKF Industrie, Elektronik GmbH, Barleben, Germany). Before analysis, the analyser was calibrated with a standard  $12.0 \text{ mmol}\cdot\text{L}^{-1}$  solution. This technology has previously been shown to be reliable for the measurement of blood lactate by Davison et al. (2000).

### **2.7 Measurement of heart rate**

Heart rate was recorded during each test using a radio telemetry system (Polar, Kempe, Finland) recorded by the Cosmed Quark b<sup>2</sup> breath-by-breath analysis system. Prior to testing the participants were fitted with a heart rate monitor strap. To ensure good skin contact the strap was moistened and positioned on the chest according to manufacturer's recommendations.

### **2.8 Environmental Conditions**

According to Daniels (1985) and Stainsby et al. (1980), temperature and humidity of the exercise environment will have a significant effect on the efficiency of movement. Under hyperthermal conditions the energy cost of the exercise will increase due to greater circulation, sweating and ventilation which in turn will reduce efficiency as the work accomplished remains unchanged (Stainsby et al., 1980). Hettinga et al. (2007) have shown that GE is decreased when cycling in the heat ( $15.6$  vs  $35.5^{\circ}\text{C}$ ). Therefore laboratory temperature was strictly controlled using an air conditioning system within the range  $20\text{-}22^{\circ}\text{C}$  and humidity was monitored at each test. All participants were also cooled by using electric fans.

## **2.9 Submaximal testing procedures**

Initially 10 minute work stages were used for the gas collection from which energy expenditure was calculated. However upon analysis of the data collected (minute 5 vs minute 10) in the study presented in chapter 3 it was demonstrated that expired  $\dot{V}O_2$  and  $\dot{V}CO_2$  were stable following 5 minutes of steady state cycling across the power outputs 150-300W (mean difference;  $\dot{V} O_2 = 0.002 \text{ L}\cdot\text{min}^{-1}$ ;  $\dot{V} CO_2 0.043 \text{ L}\cdot\text{min}^{-1}$ ;  $P>0.05$ ). The duration of each workload was then subsequently shortened to 8 minutes. Expired gas measures were taken continuously with the last 3 minutes of each stage being used for the calculation of efficiency. Three as opposed to 1 minute were collected to allow for more data points to be recorded, thus affording greater consistency in the measurements.

For the studies detailed in chapters five and six a combined threshold and maximal test was used. This enabled both the collection of submaximal and maximal  $\dot{V}O_2$  data at one testing session as used by Jones and Doust (1996). The details of this test and other procedures in relation to submaximal testing are provided in the experimental chapters as appropriate. For all studies, it was decided that preferred cadence be used throughout. Discussions in Chapter 1 outlined that cyclists would naturally select a particular cadence. If they were forced to ride above or below this preferred rate then efficiency could be altered (Norden-Snyder, 1977 Cavanagh & Kram, 1985; Brisswater et al., 2000; Samozino et al., 2006). Hansen and Ohnstad (2008) have also demonstrated that freely chosen cadence during submaximal cycling is robust over a prolonged period (12 weeks). Thus



freely chosen pedal rate was established at the first test of each study and maintained at the same rate throughout subsequent tests.

### **2.10 Maximal testing procedures**

Maximum aerobic power ( $\dot{V}O_{2\max}$ ) was defined as the highest measured 60 second  $\dot{V}O_2$  determined using an incremental protocol to volitional exhaustion. Expired gas was collected throughout using a breath-by-breath approach. Similarly maximal power output ( $W_{\max}$ ) was calculated from the same incremental test protocol using the methods of Kuipers et al. (1985). Power output was measured via the Lode Excalibur Sport ergometer. The specific protocol used is explained within each of the experimental chapters.

## **CHAPTER 3: THE RELIABILITY OF THE MEASUREMENT OF EFFICIENCY**

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Aspects of the following chapter have been peer-reviewed and published in the following article:

Hopker, J.G. Coleman, D.A. Wiles, J. (2007) Differences in Gross Mechanical Efficiency between trained and recreational Cyclists. *Applied Physiology, Nutrition and Metabolism*. 32: 6, 1036-1042.

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### **3.1 Introduction**

The research conducted into cycling efficiency has reported a wide range of mean values (17 – 27%; Sidossis et al., 1992; Nickleberry & Brookes, 1996; Moseley & Jeukendrup, 2001; Lucia et al., 2002a; Moseley et al., 2004; Coyle 2005). Whether the considerable variation in efficiency values are real or just an artefact of ‘noise’ within the measurement is unclear. Without knowledge of the reliability of the methods of measurement and calculation of efficiency it is difficult to interpret the results of previous studies. In the current literature only one article has specifically addressed the issue of the reliability of the calculation of efficiency in cycling (Moseley & Jeukendrup, 2001).

When investigating any physiological parameter the ability to detect changes over time and/or study the effects of selected interventions/treatments requires testing methods and equipment to be both

reliable and valid (Hopkins et al., 1997; Atkinson & Nevill, 1998). To investigate the effects of selected interventions on performance, within participant variation (random variation of a participant's repeated measurement) expressed as a coefficient of variation percentage (CV) must be evaluated. The lower the within participant variation (CV), the better the accuracy of a single measurement and ability to track changes over a period of time. Knowing the reliability of a measurement will also allow for determination of a suitable sample size and establish acceptable confidence limits for subsequent experiments (Hopkins et al., 1997; Atkinson & Nevill, 1998).

Previous research by Moseley and Jeukendrup (2001) demonstrated a CV of 4.2% (95% CI 3.2-6.4%) for GE and 6.7% (95% CI 5-10%) for DE. Their data demonstrate a degree of intra-individual variation, meaning that the smallest change in GE that could be reliably detected from their study and its protocol would be an absolute value of 0.6%. However, criticisms could be levelled at their methods, specifically the use of 3 minute incremental protocols. It has been shown that  $\dot{V}O_2$  does not stabilise until at least 3 minutes at a constant power output (Wasserman et al., 2005), and the slower kinetics of  $\dot{V}CO_2$  mean that it does not stabilise until ~3-4 minutes (Wasserman et al., 2005). Secondly Moseley and Jeukendrup (2001) imposed a set cadence of 80 rev.min<sup>-1</sup> on participants. It has been shown that if cyclists ride at a cadence other than their freely chosen rate they may be less efficient (Nordeen-Snyder, 1977; Hagberg et al., 1981; Samozino et al., 2006). Thus, further work is needed to assess the reproducibility of GE

and DE in competitive cyclists, particularly using a constant load exercise test to ensure a true steady state at a preferred cadence.

Cavanagh and Kram (1985) suggest that how humans apply force to an ergometer may influence metabolic efficiency. Pedalling technique has been investigated by several researchers (Davis & Hull, 1981; Patterson & Moreno, 1990; Coyle et al., 1991; Zameziati et al., 2006), however, the possible link between pedalling mechanics and changes in efficiency during submaximal cycling remains to be fully established. Zameziati et al. (2006) simultaneously measured pedalling effectiveness and GE during submaximal cycling at different intensities with a fixed cadence of 80 rev.min<sup>-1</sup>. They found that the index of effectiveness measured over the total crank cycle and that of the upstroke phase were significantly correlated to GE. These results therefore suggest that pedalling effectiveness during a full revolution could play a determining role in efficiency changes at a constant pedal rate, at least in recreational cyclists. Even though the literature above provides some insight into pedalling mechanics as a factor affecting GE, to the author's knowledge there is no previous research investigating the reliability of pedalling effectiveness. This makes the results of studies investigating this variable difficult to interpret.

### **3.2 Aims of the investigation**

The purpose of this study was to evaluate the range of measurement error associated with the assessment of GE, DE and Crank Torque Effectiveness (CTE) from a constant load protocol at 50% and 60% of maximal power output using a preferred cadence method.

### **3.3 Methods**

#### **Participants**

Fifteen endurance-trained competitive cyclists were recruited from local clubs to take part in this investigation with physical characteristics of (mean  $\pm$  SD): 33  $\pm$  4.0 yr, 75.8  $\pm$  10.5 kg,  $W_{\max}$  421  $\pm$  37 W,  $\dot{V}O_{2\max}$  61.1  $\pm$  5.2 mL $\cdot$ kg<sup>-1</sup> $\cdot$ min<sup>-1</sup> and at least 2 years of training experience. Throughout the study, cyclists served as their own control following their normal diet and daily activity patterns. All were instructed not to train within the 24 hours prior to testing.

#### **Experimental Procedures**

Cyclists visited the testing laboratory on four separate occasions, each separated by a 7 day period. The first test was a progressive maximal aerobic power test followed by three repeated tests of cycling efficiency at 50% and 60%  $W_{\max}$  as calculated from their maximal test. Measures of  $\dot{V}O_2$ ,  $\dot{V}CO_2$ , RER and power output were made throughout all exercise tests.

### *Maximal Aerobic Power Test*

The purpose of the maximal aerobic power test was to assess maximal power output, maximal oxygen uptake, maximal heart rate and freely chosen pedalling cadence. Cyclists initially completed a 10 minute warm-up at the starting power for the test (100W). Once the warm-up had been completed participants cycled using their freely chosen cadence at each power output for 1 minute after which time the load increased by 25W/min. The cyclists were asked to keep their cadence constant at this preferred rate and were given visual feedback from a display unit to do so. All cyclists conducted the continuous test until volitional exhaustion. Maximal power output was calculated using the  $W_{\max}$  formula:

$$W_{\max} = WE + (25 \text{ W/t} \times tE)$$

(Kuipers et al., 1985)

where  $W_{\max}$  = maximal power output (Watts); WE = power output of last stage completed; 25 W = work rate increment; t = work rate duration (seconds); tE = duration of final stage (seconds).

### *Steady State Trials - Cycling Efficiency*

Prior to each trial cyclists completed a warm-up of 10 minutes at 100W using their preferred cadence. After a short rest participants were asked to cycle at a power output of 50% of their  $W_{\max}$  for 10 minutes using their preferred cadence. On completion of this ten minute bout the work rate was increased to 60% of their  $W_{\max}$  for a further 10 minutes. The test was

continuous, no rest was allowed between work rates.  $\dot{V}O_2$  and RER were averaged over the final three minutes of each 10 minute bout at 50% and 60%  $W_{max}$ . GE and DE were then calculated as outlined in the general methods, chapter 2. Crank Torque was also measured during the same sampling period across the three trials and calculated as in chapter 2.

### *Statistical Analysis*

Mean GE, DE and CTE data for each of the three tests were obtained. Due to measurement error increasing in proportion to the size of the measured value (heteroscedasticity), within participant variation was expressed as a coefficient of variation (CV). Briefly, this is calculated as the standard deviation expressed as a percentage of the mean. Confidence intervals (95% CI) of the CV were also calculated (Hopkins, 2000).

Data were checked for violations of normality using a Shapiro-Wilk test. Paired two-tailed Student's t-tests were then used to analyse differences in the data between 50% and 60%  $W_{max}$  trials. Pearson's Correlation Coefficient was used to establish if measures of GE and DE could be accounted for by changes in CTE. Statistical significance was set at  $P \leq 0.05$  for all analysis conducted. All values are expressed as mean and standard deviation (mean  $\pm$  SD) unless otherwise stated.

### **3.4 Results**

Group mean power output at 50% and 60% $W_{max}$  equalled 213W and 255W respectively. Mean GE at 50% $W_{max}$  was  $20.3\% \pm 1.0\%$  with GE at 60% $W_{max}$  being  $20.3\% \pm 0.8\%$ . There were no significant differences between efficiency values recorded at 50 and 60% $W_{max}$  ( $P=0.79$ ). Table 3 shows individual values and within-participant variation (expressed as a CV) for GE at 50% $W_{max}$ . Absolute change in the mean across the three trials was 0.3% (95%CI -1.16-0.73). Calculated error of measurement (CV) across the three trials was 4.93% (95%CI 2.94-6.93). At 60% $W_{max}$  (Table 4) absolute change in the mean was 0.17% (95%CI -0.58-1.67), whilst the mean CV for the three repeated trials was 4.15% (95%CI 2.51-5.79). Mean DE for the group was  $21.5\% \pm 2.3$ . The calculation of DE (Table 5) was the most unreliable efficiency measure with a change in the mean of 0.5% (95%CI -1.75-2.76) which equates to a mean CV for the three repeated trials of 10.9% (95%CI 6.40-15.38).

CTE proved to be a reliable measure (Tables 6 & 7) with a CV of 1.42% (95%CI: -0.81-3.64) and 1.20% (95%CI: -0.77-3.16) at 50% and 60% $W_{max}$  respectively. CTE at 50% $W_{max}$  was significantly lower than at 60% ( $80.7 \pm 1.1$  vs  $86.0 \pm 1.0$ ) ( $P<0.01$ ). No significant relationships between GE and CTE at 50 and 60% $W_{max}$  (50%  $r=-0.34$ ; 60%  $r=-0.49$ ), or DE and CTE at either 50% or 60% (50%  $r=-0.29$ ; 60%  $r=-0.28$ ) were observed. Mean cadence was  $94 \pm 6$  rev.min<sup>-1</sup>. No order effect was observed for either efficiency measure across the power outputs ( $P>0.05$ ), or CTE ( $P>0.05$ ).



Table 3. Gross Efficiency and CV across three steady state trials at 50%  $W_{\max}$ .

Participant	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	Mean
Mean GE1 (%)	22.5	18.3	18.6	24.1	20.9	19.9	19.0	19.5	18.2	22.8	18.2	18.9	20.0	19.5	23.8	
Mean GE2 (%)	22.3	17.6	18.2	21.7	21.0	21.9	21.3	20.8	17.2	22.8	18.7	20.8	20.3	19.3	22.6	
Mean GE3 (%)	21.1	19.4	19.4	21.7	19.1	20.1	19.4	24.3	17.1	20.6	18.3	20.8	21.3	18.4	21.3	
Mean GE (%)	22.0	18.5	18.8	22.5	20.4	20.6	19.9	21.6	17.5	22.0	18.4	20.2	20.6	19.1	22.6	20.3
SD (%)	0.78	0.87	0.59	1.41	1.07	1.08	1.2	2.46	0.60	1.25	0.27	1.10	0.68	0.61	1.24	1.02
CV (%)	3.56	4.74	3.18	6.29	5.26	5.24	6.05	11.4	3.48	5.68	1.51	5.49	3.33	3.23	5.51	4.93
95% CI	2.03- 5.09	3.03- 6.46	2.01- 4.35	3.52- 9.07	3.16- 7.36	3.12- 7.35	3.69- 8.41	6.59- 16.24	2.28- 4.67	3.23- 8.13	0.97- 2.06	3.32- 7.66	1.99- 4.67	2.02- 4.44	3.07- 7.94	2.94- 6.93

Table 4. Gross Efficiency and CV across three steady state trials at 60%  $W_{max}$

Participant	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	Mean	
Mean GE1 (%)	23.7	18.7	17.6	22.4	20.7	20.5	19.1	19.4	19.7	21.4	19.1	18.5	20.3	19.0	22.0		
Mean GE2 (%)	23.3	18.0	20.4	20.0	20.4	21.8	20.5	21.1	17.8	21.0	19.2	20.2	21.4	19.4	21.8		
Mean GE3 (%)	22.4	20.4	19.5	21.8	19.7	20.2	20.2	22.5	18.2	20.6	19.3	20.2	21.6	17.9	21.3		
Mean GE (%)	23.1	19.0	19.1	21.4	20.3	20.8	20.0	21.0	18.6	21.0	19.2	19.6	21.1	18.8	21.7	20.3	
SD (%)	0.68	1.24	1.41	1.24	0.50	0.84	0.75	1.55	1.01	0.36	0.11	0.95	0.74	0.74	0.37	0.84	
CV (%)	2.96	6.55	7.40	5.79	2.47	4.05	3.76	7.38	5.47	1.72	0.60	4.88	3.49	3.99	1.72	4.15	
95% CI	1.62- 4.31	4.11- 9.00	4.62- 10.18	3.36- 8.22	31.49- 3.45	2.40- 5.70	2.29- 5.24	4.34- 10.42	3.48- 7.46	1.01- 2.43	0.38- 0.84	3.00- 6.76	2.05- 4.94	2.52- 5.46	0.99- 2.45	2.51- 5.79	

Table 5. Delta Efficiency and CV across three steady state trials

<b>Participant</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5</b>	<b>6</b>	<b>7</b>	<b>8</b>	<b>9</b>	<b>10</b>	<b>11</b>	<b>12</b>	<b>13</b>	<b>14</b>	<b>15</b>	<b>Mean</b>
<b>Mean DE1 (%)</b>	31.4	20.7	19.7	16.5	19.4	24.1	19.4	18.6	33.6	16.2	25.1	16.9	21.5	16.9	16.0	
<b>Mean DE2 (%)</b>	30.2	22.7	19.8	14.5	17.9	21.3	19.4	22.8	23.5	15.1	26.4	17.5	29.6	19.7	18.6	
<b>Mean DE3 (%)</b>	31.8	22.9	19.9	22.4	19.2	20.6	25.7	16.4	26.3	21.0	27.0	17.7	23.1	16.1	21.3	
<b>Mean DE (%)</b>	31.1	22.1	19.8	17.8	18.8	22.0	21.5	19.3	27.8	17.4	26.2	17.4	24.8	17.6	18.6	21.5
<b>SD (%)</b>	0.86	1.22	0.11	4.09	0.78	1.85	3.63	3.28	5.23	3.11	0.96	0.39	4.29	1.87	2.67	2.29
<b>CV (%)</b>	2.76	5.52	0.55	23.03	4.17	8.41	16.91	16.99	18.84	17.82	3.66	2.26	17.34	10.71	14.32	10.89
<b>95% CI</b>	1.07- 4.44	3.12- 7.90	0.34- 0.77	14.99- 31.06	2.62- 5.71	4.78- 12.04	9.80- 24.02	10.57- 23.41	8.57- 29.10	11.73- 23.91	1.78- 5.54	1.49- 3.03	8.92- 25.75	7.02- 14.39	9.09- 19.56	6.40- 15.38

Table 6. Crank Torque Effectiveness and CV across three steady state trials at 50%W<sub>max</sub>

Participant	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	Mean
Mean CTE1 (%)	73.7	84.8	91.5	78.5	79.9	86.6	86.1	88.1	77.9	67.6	85.6	81.1	73.3	80.4	80.5	
Mean CTE2 (%)	74.8	83.7	88.2	79.5	80.3	87.4	84.1	84.4	77.8	68.1	83.7	77.5	71.0	78.7	81.9	
Mean CTE3 (%)	74.9	82.6	88.0	81.5	80.3	87.2	85.3	87.0	78.1	71.0	84.9	78.1	72.9	81.0	80.1	
Mean CTE (%)	74.5	83.7	89.2	79.8	80.2	87.0	85.2	86.5	77.9	68.9	84.7	78.9	72.4	80.0	80.8	80.7
SD (%)	0.65	1.08	1.98	1.52	0.21	0.39	1.01	1.89	0.16	1.85	0.96	1.97	1.23	1.20	0.94	1.14
CV (%)	0.88	1.28	2.22	1.90	0.26	0.45	1.18	2.19	0.21	2.68	1.13	2.49	1.70	1.50	1.16	1.42
95% CI	-0.4	-0.8	-1.7	-1.1	-0.2	-0.3	-0.8	-1.5	-0.1	-0.9	-0.8	-1.4	-0.7	-0.9	-0.7	-0.8
	-2.2	3.4	6.1	4.9	0.7	1.2	3.2	5.9	0.5	6.3	3.0	6.3	4.1	3.9	3.0	3.6

Table 7. Crank Torque Effectiveness and CV across three steady state trials at 60%W<sub>max</sub>

Participant	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	Mean
Mean CTE1 (%)	81.0	90.3	92.8	87.8	89.1	90.4	90.8	93.6	83.4	70.7	90.8	84.7	78.4	82.9	85.6	
Mean CTE2 (%)	81.9	88.7	93.0	86.9	85.6	90.6	89.2	90.3	83.4	71.4	90.7	85.2	76.8	83.2	88.2	
Mean CTE3 (%)	82.3	88.8	92.2	88.5	85.6	90.6	88.9	90.4	84.8	75.7	91.3	84.5	78.6	84.3	86.3	
Mean CTE (%)	81.8	89.3	92.6	87.7	86.8	90.5	89.6	91.4	83.9	72.6	90.9	84.8	77.9	83.5	86.7	86.0
SD (%)	0.69	0.92	0.40	0.78	2.06	0.13	1.03	1.88	0.78	2.69	0.35	0.33	0.97	0.71	1.33	1.00
CV (%)	0.84	1.03	0.43	0.88	2.37	0.14	1.15	2.06	0.93	3.71	0.38	0.38	1.25	0.86	1.54	1.20
95% CI	-0.5 – 2.0	-0.8 – 2.8	-0.4 – 1.2	-0.6 – 2.4	-1.7 – 6.4	-0.1 – 0.4	-0.9 – 3.2	-1.6 – 5.8	-0.6 – 2.5	-1.6 – 9.0	-0.3 – 1.1	-0.3 – 1.0	-0.7 – 3.2	-0.5 – 2.3	-1.1 – 4.2	-0.8 – 3.2

### **3.5 Discussion**

#### **Gross and Delta Efficiency**

Efficiency values obtained from this study (GE 17.6-23.7%, DE 17.4-31.1%) are comparable to those in the literature (Gasser & Brooks, 1975; Suzuki, 1979; Coyle et al, 1992; Sidossis et al., 1992; Horowitz et al., 1994, Nickleberry & Brooks, 1996; Moseley & Jeukendrup, 2001). Measures of GE were found to be more reliable than those of DE, a finding which is also in agreement with that of Moseley and Jeukendrup (2001). CTE was found to highly reliable at both 50 and 60% $W_{max}$ .

The coefficient of variation for GE from the new protocol was 4.9% (95% CI 2.9-6.9) at 50%  $W_{max}$  and 4.2 (95% CI 2.5-5.8) at 60%  $W_{max}$ . Therefore in absolute terms, the ‘noise’ in current protocol is ~0.9% for GE. The CV values obtained are comparable with those previously reported by Moseley and Jeukendrup (2001) ranging from 3.2-6.4%. The current research found that measures of DE were less reliable (than those for GE) with a CV of 10.9% (95% CI 6.4-15.4), again consistent with the findings of previously published data (Moseley & Jeukendrup, 2001). The variation of GE is approximately half of that for DE. Therefore if DE were used as the primary outcome variable for the rest of this thesis, large changes in efficiency would be needed (~2.2%) to detect significant differences with training. Similarly a large number of participants (65 in each group; power = 0.8; effect size  $[1/SD] = 0.44$ ) would be needed to possess the necessary statistical power to have a chance of detecting a difference between groups or in the same group with training.

Moseley and Jeukendrup (2001) attribute their reliability findings to what they suggest is a curvilinear (as opposed to linear; Gaesser & Brooks, 1975; Croisant & Boileau, 1984; Chavarren & Calbert, 1999; Samozino et al. 2006) relationship between efficiency and work rate. Findings of the current research appear to support this; there was no difference in GE between 50% and 60%  $W_{\max}$  (although only two power outputs were used). The current study's mean work rates at 50 and 60%  $W_{\max}$  (213W & 255W) were in the region where Moseley and Jeukendrup demonstrate a plateau occurs (200 - 275W) in the power output-efficiency curve. This distortion occurs due to the proportion of energy expenditure that is used to maintain homeostasis becoming smaller as total energy expenditure increases (Stainsby, 1980). To enable GE increases at higher power outputs, energy expenditure must therefore increase non-linearly. This suggests that the points on a power output versus energy expenditure plot cannot be a straight line (see Figure 4).

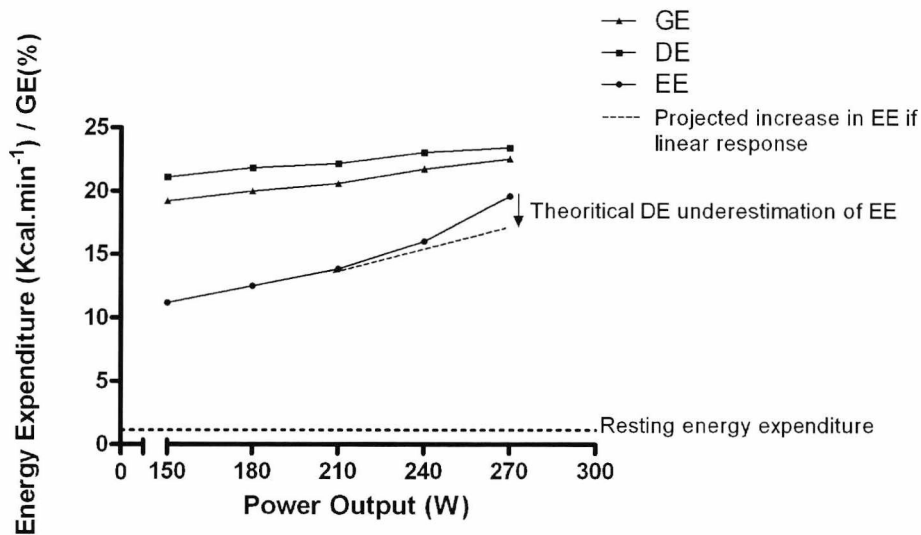


Figure 4. The theoretical relationship between GE, DE and Energy Expenditure

However the calculation of DE has been suggested to be a more valid measurement of muscle efficiency (Coyle et al., 1992) as unlike GE, resting energy metabolism is excluded from the calculation.

The theoretical maximum for efficiency of muscular work is computed as the product of phosphorylative and contraction coupling efficiencies (60% and 49% respectively). Thus muscular efficiency is calculated:

$$\text{Muscular efficiency} = (0.6 \times 0.49) \times 100$$

(Whipp & Wasserman, 1969)

This therefore sets the ceiling for the maximum efficiency of muscle work as 29%. The DE data from some of the riders in the current study may therefore be problematic as values in excess of 30% were recorded (see Table 5). These high values may be the result of inherent 'noise' within the



measurement. The curvilinear nature of the efficiency-work rate relationship would suggest that DE causes an overestimation of efficiency values.

The current study used a cadence fixed at the cyclist's preferred rate (mean 94 rev.min<sup>-1</sup>) and so cadence did vary amongst individuals (SD = 6 rev.min<sup>-1</sup>). Preferred rather than fixed uniform cadence was favoured as both Nordeen-Snyder (1977) and Hagberg et al. (1981) reported that well-trained competitive cyclists pedalling at their preferred rate (mean 91 rev.min<sup>-1</sup>) were significantly more economical than when they were asked to pedal slower or faster at the same power output. Similarly Rossato et al., (2008) have shown that the use of preferred cadence results in the most effective application of force during the downstroke phase of the crank cycle compared to cadences 20% higher and lower across work rates of 60 and 80% maximum power output. The standard deviation across all cyclists was only 6 rev.min<sup>-1</sup>, thus it is speculated that this would not significantly alter the calculated efficiency values anyway (see Figure 2). In addition, pedal cadence did not change between work rates or over test sessions.

### **Crank Torque Effectiveness**

Peddalling technique has been proposed as a factor which may in part determine performance (Coyle et al., 1988). CTE or pedalling effectiveness has been investigated by several authors (Lafortune et al., 1983; Patterson & Moreno, 1990; Kautz et al., 1991; Sanderson & Black, 2003; Zameziati et al., 2006; Mornieux, et al., 2006; Korff et al., 2007), however no previous study has reported the reliability of these measurements. The current study

found CTE to be a highly reliable measure (Tables 6 & 7) at both relative exercise intensities used (CV= 1.4% and 1.3% at 50 and 60%  $W_{\max}$  respectively).

Interestingly there was a significant increase in overall effectiveness with increases in power output ( $P<0.01$ ) which corroborate the work of other authors (Davis & Hull, 1981; Ericson & Nisell, 1988; Patterson & Moreno, 1990; Sanderson, 1991 and Zameziati et al., 2006). Although there was considerable inter-individual variation in CTE (68.9-92.6%), correlations suggest that it does not account for the variability of GE or DE in the trained cyclists used in the current study. As effectiveness increased at higher power outputs, the share of non-effective forces decreased; however this did not appear to contribute to increases in GE. In support of these findings, Zameziati et al. (2006) found no significant correlations between GE and pedalling effectiveness during the downstroke phase of the pedalling action. However, they did find significant, moderate, correlations between GE and the upstroke and total crank cycle ( $r=0.66$  &  $0.79$  respectively) which are not noticed in the current study. Possible reasons as to why the current study's results did not find any significant relationship between CTE and GE could be that the importance of generating propulsive force between  $0$  and  $180^\circ$  masked any inefficiency from the upstroke, or the computation of CTE during a full crank cycle is not sensitive enough to compare with a gross  $\dot{V}O_2$  measured at the mouth. In addition, torque measured at the crank might not be reflective of the work done by the muscles and therefore metabolic cost. Mathematical modelling has calculated that from  $350^\circ$  to  $95^\circ$ , the

muscles of the leg generate more power than is delivered to the crank, therefore accelerating the leg (Kautz & Neptune, 2002). From 95° to 170° the kinetic energy of the legs decrease associated with leg deceleration, although again more energy is delivered to the crank than is generated by the muscles. Therefore due to additional work performed by the muscles in terms of limb acceleration and deceleration a greater metabolic cost is incurred than could be suggested from crank torque data.

### **3.6 Conclusion**

This investigation provides a clear indication that the use of a long stage, preferred cadence protocol is reliable for the determination of GE in cycling. Even though DE is suggested to be the most valid measurement of muscular efficiency its considerable day-to-day variation makes it a worse measure of cycling efficiency when tracking performance change. It is recommended that if cycling efficiency measurements are to be performed then GE should be the preferred calculation that is used. GE will therefore be used as the primary outcome variable throughout the rest of this thesis. The absolute values of GE and DE as well as the reliability coefficients reported in the current study are comparable to those previously published. CTE was found to be highly reliable across the range of relative power outputs used. The test protocols and measurement equipment used in the present study provide a reliable assessment of both metabolic efficiency and crank torque that can be used in future investigations.

## **CHAPTER 4: DIFFERENCES IN EFFICIENCY BETWEEN TRAINED AND RECREATIONAL CYCLISTS**

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Aspects of the following chapter have been peer-reviewed for publication in the following article:

Hopker, J.G. Coleman, D.A. Wiles, J. (2007) Differences in Gross Mechanical Efficiency between Trained and Recreational Cyclists. *Applied Physiology, Nutrition and Metabolism*. 32: 6, 1036-1042.

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### **4.1 Introduction**

Most research investigating differences in cycling efficiency between trained and recreational cyclists have found no significant differences between the groups (Stuart et al., 1981; Marsh & Martin 1993; Nickleberry & Brooks 1996; Marsh et al., 2000; Moseley et al., 2004). However due to the physiological adaptations occurring over years of endurance training this may be surprising.

Several authors have previously demonstrated correlations between cycling efficiency and the percentage of Type I muscle fibres (Coyle et al., 1992; Horowitz et al., 1994; Mogensen et al., 2006), even though these are cross-sectional in nature. The scientific literature suggests that training has an influence on muscle fibre type. Differences in muscle fibre type have been demonstrated between trained and untrained participants (Fitzsimons et al.,

1990; Baldwin & Haddad 2001; Putman et al., 2004). Specifically, prolonged training at moderate to low intensities (as trained cyclists would commonly undertake as part of normal training) has been shown to stimulate muscle fibre transformation towards more slow-twitch Type I (Kadi & Thornell 1999; O'Neill et al. 1999). Given that Coyle et al. (1992), Horowitz et al. (1994) and Mogensen et al. (2006) have all shown that efficiency is significantly related to percentage of Type I muscle fibres, it is highly unlikely that differences between trained and untrained populations do not exist. With this in mind there could be other possible reasons why no significant differences in efficiency have previously been reported between trained cyclists and untrained individuals. One possible explanation could be the methods used in the previous research; for example GE has sometimes been compared between trained and untrained participants at a given absolute intensity (Stuart et al., 1981; Marsh et al., 2000; Moseley et al., 2004). However this same absolute intensity may represent a higher relative intensity for untrained versus trained participants due to their lower maximal tolerated power output. Since exercise efficiency is significantly influenced by exercise intensity (Gaesser & Brooks, 1975), the differences in efficiency, or lack of them in the published literature, could in part be explained by the absolute work intensity imposed.

Research by Nickleberry and Brooks (1996) concluded that no differences exist between trained and recreational cyclists in terms of delta efficiency. However, upon close examination of their data large mean differences between groups are present (~2% GE & ~7% DE). Indeed this is common

when the majority of the literature is scrutinized more closely. There appears to be 'physiologically relevant' but statistically non-significant differences in efficiency between trained and untrained participants (Stuart et al., 1981; Marsh & Martin 1993; Nickleberry & Brooks 1996; Marsh et al., 2000). General criticisms of these research studies are provided in chapter 1.

Despite much of the literature indicating there are no significant differences between trained cyclists and untrained individuals, other research comparing different cycling specific populations has indicated that professional cyclists have a very high efficiency. Lucia et al. (2002a) reported efficiency values of 28.1% for "world class professional cyclists"; which is considerably higher than any research has previously reported. However this particular investigation has recently come under criticism due to these high values (Jeukendrup et al., 2003). Künstlinger et al. (1985) have also demonstrated that oxygen uptake during a prolonged cycling test was significantly lower in a group of trained cyclists compared to non-cyclists. They attributed these changes to differences in the force distribution the pedalling cycle although estimations of efficiency were not made ( $\dot{V}O_2$  only).

Economy measures (the oxygen cost of cycling expressed as W.L.O<sub>2</sub>) have also been presented in the literature, with Jeukendrup et al. (2000) reporting trained cyclists values at 74 W.L.O<sub>2</sub>, compared to world class riders at >78 W.L.O<sub>2</sub>. Working back from these numbers for a power output of 150W the  $\dot{V}O_2$  would be 2.02 L.min<sup>-1</sup> for the trained compared to 1.92 L.min<sup>-1</sup> for the

world class cyclist. For similar efficiency values between these groups e.g. 22.5%, the kcal used per litre of oxygen consumed for the trained cyclists would be 4.73, this would require an RER of  $\sim 0.75$  based on the thermal equivalents of oxygen for a nonprotein RQ. This compares to the world class cyclist who would have a substantially higher RER  $\sim 0.94$  (kcal per litre of oxygen consumed  $\sim 4.98$ ). This clearly has not been reported in the literature, and if this data is reworked to match RER (at 0.94), the efficiency calculations for the world class cyclists would be 22.5 vs 21.4%.

The methods used in previous research have not been able to detect any differences between trained and untrained participants, many of which have specific limitations. Therefore the purpose of this study was to utilise a research design that had enough power to detect any differences which may exist between trained and recreational cyclists.

## **4.2 Aims of the investigation**

The aim of this study is to identify if there are differences in gross efficiency between trained and recreational cyclists at both relative and absolute power outputs.

## **4.3 Methods**

### **Participants**

A cross-sectional study design was utilized where 16 trained competitive cyclists (mean  $\pm$  standard deviation:  $33 \pm 4$  yr,  $1.76 \pm 0.05$  m,  $75 \pm 10$  kg,  $W_{\max} 421 \pm 38$  W,  $\dot{V}O_{2\max} 62.6 \pm 7.30$  mL $\cdot$ kg $^{-1}\cdot$ min $^{-1}$ ) with at least 2 years

of cycle training experience (3 - 19 yr), and 16 recreational cyclists (mean  $\pm$  standard deviation: 22  $\pm$  3 yr, 1.75  $\pm$  0.06 m, 76  $\pm$  10 kg,  $W_{\max}$  292  $\pm$  34 W,  $\dot{V}O_{2\max}$  42.6  $\pm$  7.80 mL $\cdot$ kg $^{-1}\cdot$ min $^{-1}$ ) who cycle regularly as part of a fitness regime, volunteered to take part in the investigation.

The results of the reliability study outlined in chapter 3, identified the variability of the measurement of GE using a long-stage duration, preferred cadence protocol. Consequently appropriate sample size based on sufficient statistical power can be estimated. The Coefficient of Variation used from the reliability study was 4.5% (average of CV at 50 and 60% $W_{\max}$ ). The methods used to calculate sample size are outlined by Baguley (2004) using the methods of Cohen (1988) and GPower software (Erdfelder et al., 1996);

$$N \text{ per group} = 2(\delta/d)^2$$

$d$  was the detected effect size, Power was set at 0.8, and therefore  $\delta$  (the value for non-centrality) could be calculated using GPower. The most appropriate selection of the likely change has attracted some debate in the literature with some authors selecting a meaningful change/difference in the parameter (Petersen et al., 2004), with others using the smallest worthwhile change of 0.2 of the between participant standard deviation (Cohen, 1988; Hopkins, 2000). For the current study the work of Nickleberry and Brookes (1996) was initially consulted, with the mean difference in GE observed between trained and recreational cyclists at 80 rev.min $^{-1}$  being 1.0%. This value has been suggested to have a worthwhile effect on performance (Moseley & Jeukendrup, 2001). The sample population standard deviation was derived from the reliability study (chapter 3), which yielded a value of



1.02% (highest SD across 50 and 60% $W_{max}$ ), this value is similar to the values (0.92%) presented by Moseley and Jeukendrup (2001). As recommended by Batterham and Atkinson (2005) effect size was then calculated based on this previously published data. The effect statistic was therefore calculated as  $1/1.02 = 0.98$ . Using GPower the value for non-centrality was calculated to be 2.59. Therefore the calculated sample size was 14 participants in each group.

### **Experimental Procedures**

All participants undertook a test of efficiency at two relative work rates (50% and 60% $W_{max}$ ) and at the same absolute power output (150W) using a preferred fixed cadence. The recreational cyclist's cycling position was determined on the first trial by adjusting the saddle height to ensure the arch of the foot was in contact with the pedal when it was at its lowest position. Handle bar height and reach were also adjusted to allow a comfortable position, toe straps and set crank lengths of 170mm were used. These measurements were then recorded and replicated for the subsequent test.

#### *Maximal Aerobic Power Test*

Participants completed a maximal aerobic power test as described in chapter 3. Throughout the test cardio-respiratory responses were assessed using the previously described procedures in this thesis.

### *Steady State Trials - Cycling Efficiency*

With at least seven days following the maximal aerobic power test participants completed one test of cycling efficiency as outlined in chapter 3 and one trial at a fixed absolute power output of 150W for 10 minutes.  $\dot{V}O_2$ ,  $\dot{V}CO_2$  and crank torque were measured. GE, DE and CTE were calculated for all trials as described in chapters 1 and 2 respectively.

### *Statistical Analysis*

Prior to all statistical analyses, data were checked for normality. Comparisons of the mean of measured variables between trained and recreational cyclists were assessed using a two-factor ANOVA, statistical significance was set at 95% confidence ( $P \leq 0.05$ ). Unadjusted Post Hoc analysis (Perneger, 1998), was then conducted to identify where any significant differences within the data existed. Differences in GE and CTE between the groups were assessed at 150W, 50 and 60% $W_{max}$ . Cadence differences between trained and recreational cyclists were identified using an independent Student's t-test. Results are expressed as mean  $\pm$  SD unless otherwise stated.

## **4.4 Results**

### **Gross and Delta Efficiency**

All participants completed all three tests. However prior to analysis of the results two trained cyclists were excluded, one due to injury sustained between testing sessions and the other due to his status as a trained competitive cyclist (he had not competed for 2 years).

GE did not significantly change across the work rates in the trained group ( $P=0.45$ ; see Table 8), indicating that the increase in oxygen consumption requirements was proportional to the increase in work completed over the sampling period. However in the recreational group the 60%  $W_{\max}$  trial was significantly lower in terms of efficiency than the 150 W fixed workload ( $P=0.01$ ) (see Table 8).

Trained cyclists had a significantly higher GE at 150 W ( $20.9 \pm 1.3\%$  vs.  $19.9 \pm 1.7\%$ ;  $P=0.03$ ), 50% $W_{\max}$  ( $20.6 \pm 1.4$  vs.  $18.9 \pm 1.3\%$ ;  $P=0.05$ ) and 60%  $W_{\max}$  ( $20.5 \pm 1.2$  vs.  $18.8 \pm 0.9\%$ ;  $P<0.01$ ) compared to the recreational group.

Table 8. Gross efficiency of trained and recreational cyclists.

	Trained Mean $W_{\max} = 425\text{W}$	Recreational Mean $W_{\max} = 292\text{W}$
GE fixed 150W	$20.9 \pm 1.3^*$	$19.9 \pm 1.3$
GE 50% $W_{\max}$	$20.6 \pm 1.4^*$	$18.9 \pm 1.7$
GE 60% $W_{\max}$	$20.5 \pm 1.2^*$	$18.8 \pm 0.9$

\* = Significantly higher than the recreational group.

DE calculated across the two relative power outputs was not significantly different between the two groups (trained cyclists =  $21.5 \pm 4.2\%$ ; recreational cyclists =  $19.1 \pm 4.2\%$ ;  $P=0.14$ ). Trained cyclists used a preferred cadence which was significantly higher than the recreational cyclists ( $94 \pm 6$  vs.  $69 \pm 4$  rev.min<sup>-1</sup>;  $P<0.01$ ).

### **Crank Torque Effectiveness**

No significant differences in mean CTE were found between trained and recreational cyclists at either 50% $W_{\max}$  ( $82.2 \pm 4.1$  vs  $82.9 \pm 4.4$ ;  $P=0.71$ ), or 60% $W_{\max}$  ( $87.6 \pm 3.3$  vs  $87.2 \pm 4.0$ ;  $P=0.79$ ). However, at a fixed work rate of 150W the trained group possessed a significantly lower effective crank torque than the recreational group ( $73.0 \pm 5.6$  vs  $82.3 \pm 1.4$ ;  $P<0.01$ ). CTE significantly increased with increasing exercise intensities in both trained and recreational groups ( $P<0.01$ ). CTE data is presented in Table 9 below.

Table 9. Crank Torque Effectiveness of trained and recreational cyclists.

	Trained	Recreational
CTE fixed 150W	73.0 ± 5.6*	82.3 ± 1.4
CTE 50%W <sub>max</sub>	82.2 ± 4.1	82.9 ± 4.4
CTE 60%W <sub>max</sub>	87.6 ± 3.3	87.2 ± 4.0

\*Significantly lower than the recreational group

There were no differences in positive or negative pedal force application between trained and recreational cyclists at 50 or 60% W<sub>max</sub> ( $P=0.69$ ). However at 150W the trained group used significantly lower pedal forces than the recreational group (44.5 vs 52.6 Nm;  $P<0.01$ ). The trained cyclist's negative torque was also significantly higher (14.5 vs 10.3 Nm;  $P<0.01$ ). There were no significant correlations between CTE and GE at 50 or 60% W<sub>max</sub> in either the trained or recreational group ( $P>0.05$ ).

#### **4.5 Discussion**

##### **Gross and Delta Efficiency**

Results demonstrated that trained competitive cyclists possess a greater GE than recreational cyclists at both relative and absolute exercise intensities. The enhanced GE is reflected by a lower energy expended, as calculated from oxygen cost, for a given amount of mechanical work (Coyle, 1995). GE has been suggested to be an important determinant for endurance performance combining with lactate threshold  $\dot{V}O_2$  to establish lactate threshold performance power and ultimately performance power (Coyle, 1999). However, as this is a cross-sectional study it should be pointed out



that it is difficult to speculate on why differences exist as there is no control for selection and adaptation.

Interestingly, the calculated DE did not show the same significant differences as GE. Results show a mean difference between the groups of 2.4%. However, due to the poor reliability of DE (as shown in chapter 3) and large variations in the within participant data ( $SD = 4.2\%$ ) it is unsurprising that a statistical difference was not found. It is estimated (based on GPower), that if DE were the primary outcome variable 102 participants would have been needed to conclude no difference between groups.

Based upon the GE differences found in the current study between trained and recreational cyclists these alone would account for approximately 81 seconds when cycling at  $60\% W_{max}$  over a 40km time trial distance (Jeukendrup & Martin, 2001).

The sample size calculations for the current study were based on the work of Nickleberry and Brookes (1996). This paper was used for two reasons, firstly it is a major publication in the field, and secondly a conservative approach was employed in that the differences reported between trained and recreational cyclists were probably likely to be smaller than the differences between trained and untrained participants. In fact their differences in GE between trained and untrained participants were similar to the current study's findings of 1.02%, 1.66%, and 1.68% at the three work rates undertaken. In the current study at 150 W the differences between trained

and recreational cyclists as a proportion of the recreational efficiency was 5.1%, and this rose to 8.8% and 8.9% for the relative work intensities.

Interestingly, the proportional differences found in GE in the current study agree with existing literature examining differences between trained and untrained participants (Nickleberry & Brooks, 1996; Marsh et al., 2000). However using a longer-stage duration, preferred cadence protocol and with sufficient participant numbers (given the 'noise' in primary outcome variable), different conclusions are reached. As previously discussed in chapter 1 many of the non significant findings of previous studies are primarily due to the experimental designs employed and lack of sufficient sample size. Nickleberry and Brooks (1996) only used 6 trained and 6 recreational cyclists. Their trained participants also had a low  $\dot{V}O_{2\text{peak}}$  value compared to those normally cited in the literature for competitive cyclists ( $48.6\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ). Based upon their data the achieved statistical power was 0.20 and so Nickleberry and Brooks would have actually needed 33 participants to identify differences between their two groups.

Moseley et al. (2004) used sixty-nine participants comprising of both trained and untrained participants and still failed to find significant differences between the groups. It is possible that limitations in their protocol could account for their findings. An incremental test was used to determine efficiency with stages of three minutes in duration. They present data from McDaniel et al. (2002) in support of their protocol, which suggests that metabolic cost is stable during minutes 3, 4 and 5 of a five minute

incremental protocol. However on closer examination of McDaniel's data the stability in  $\dot{V}O_2$  occurred between minute 3 and 4 whereas Moseley et al. were sampling between minute 2 and 3 due to the protocol increment occurring at minute 3. Wasserman et al. (2005) indicate it is likely to take considerably longer for  $\dot{V}CO_2$  to stabilise than  $\dot{V}O_2$ . This is potentially problematic as  $\dot{V}CO_2$  is a determinant of RER which in turn is used in the calculation of energy expenditure. In the current study long stage durations (10 minutes) were used to ensure steady state had been reached. Moseley et al. (2004) also imposed a cadence of 80-90 rev.min<sup>-1</sup> which was enforced for all participants regardless of cycling ability. This is problematic as cadence has been shown to affect oxygen cost (Faria et al., 1982; Merrill & White, 1984; McDaniel et al., 2002), which in turn affects efficiency (Davison & Flynn, 1997). If an untrained participant is forced to cycle at an unnatural pedalling rate, higher than their preferred (from the current study average was  $68 \pm 4$  rev.min<sup>-1</sup>) they will incur additional oxygen cost and thus efficiency will be artificially decreased (Brisswater et al., 2000; Samozino et al., 2006). However it should be noted that in the current study trained cyclists rode using a significantly higher cadence than the recreational cyclists (25 rev.min<sup>-1</sup>). Therefore they would have incurred a higher overall metabolic cost at the same exercise intensity due to the cost of non-propulsive work of simply moving their limbs, which will increase oxygen cost and thus depress GE values (Gaesser & Brooks, 1975; Francescato et al., 1995; Londeree et al., 1997). Had this been taken into account the differences may have been increased. Similarly if training affects pedal rate, trained cyclists would have to ride at their preferred



cadence. If forced to cycle at a slower rate inflated oxygen cost values would be recorded (Nordeen-Snyder, 1977; Hagberg et al., 1981). This study aimed to investigate the efficiency of the trained cyclists at a rate of movement at which they normally train and compete; other possible cadences were therefore irrelevant.

The current research findings support those of Lucia et al. (2002a) who suggest that due to either genetics and/or training adaptations; high level cyclists could possess a greater GE than a less endurance trained population. However, the current research does not support the high values (~28%) found by Lucia et al. (2002a). This may be due to the fact that “world class” cyclists were not assessed. Jeukendrup et al. (2003) have also criticised the work of Lucia et al. on the grounds of measurement error, causing inflated efficiency values. Although criticised by Martin et al. (2005), Schumacher et al. (2005) and Gore et al. (2008) increases in GE values due to training adaptations have been documented by Coyle (2005) in his study of a Grand Tour Champion. The cyclist’s GE values increased from 21.18% to 23.05% over a seven year period. Coyle proposes the main mechanism for this may be due to the cyclist increasing his percentage of Type I muscle fibres. Indeed the data from the current study would somewhat support the hypothesis that training causes adaptations to improve GE. However it cannot necessarily be totally attributed to fibre type transformations, as pedalling action (Coyle et al., 1991; Mornieux et al., 2006; Samozino et al., 2006; Zameziati et al., 2006), neuromuscular adaptation (Takaishi et al., 1998) and greater fat oxidation (Lucia et al., 1998) may significantly influence changes in GE.

### **Crank Torque Effectiveness**

The highly reliable measures of CTE (as demonstrated in chapter 3) were not shown to be significantly different between trained and recreational cyclists at relative work rates. However trained cyclists CTE was significantly lower at the fixed power output of 150W. It is speculated that as this workload was 35% of the mean  $W_{\max}$  of the trained cyclists compared to 51% $W_{\max}$  for the recreational cyclists, CTE for the trained cyclists was not such an important factor in determining power output as for the recreational group (see Figure 5 below). Similarly the differences in pedalling cadence and thus crank rate could account for the results. The higher cadence of the trained cyclists may have resulted in less force being generated per crank cycle (MacIntosh et al., 2000) compared to the recreational group. The importance of having a high CTE appears to become progressively more important as the intensity of exercise increases and efficient generation of power is required. This is demonstrated in the current data by significant increases in CTE as power output increased for both trained and recreational cyclists (see Figures 5-7 below). Indeed Kautz et al. (1991) demonstrated that crank torques became more effective when trained cyclists rode at higher power outputs (equivalent to 60-100%

$\dot{V}O_{2\max}$ ).

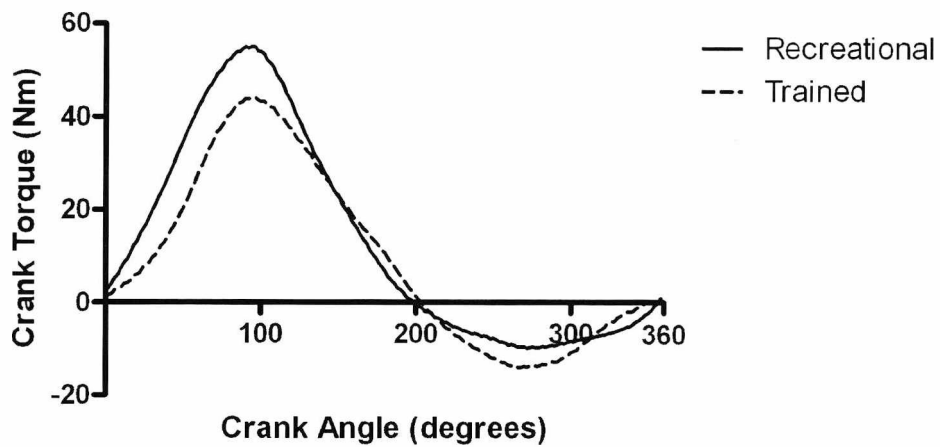


Figure 5. Mean Crank Torque at 150W.

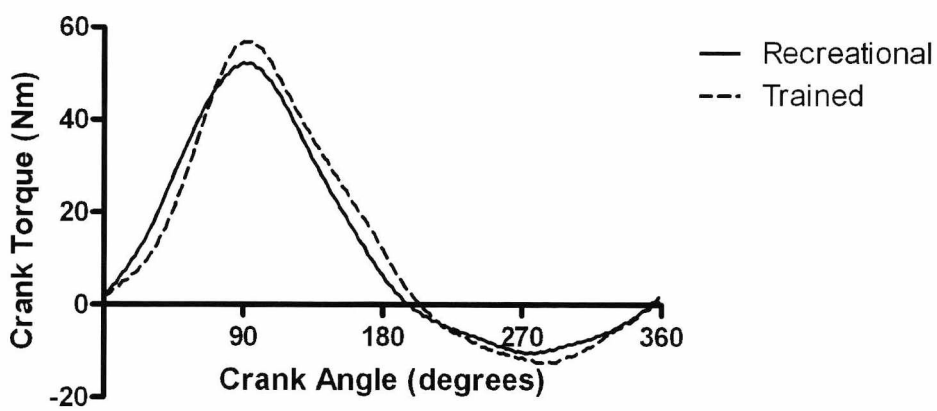


Figure 6. Mean Crank Torque at 50%W<sub>max</sub>.

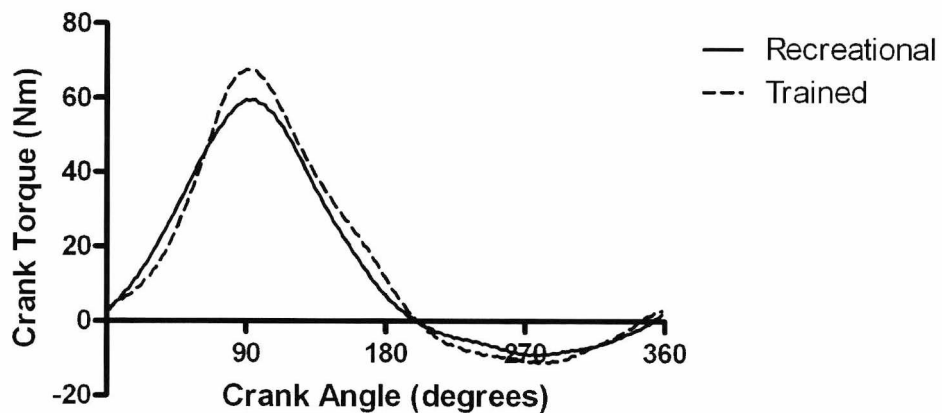


Figure 7. Mean Crank Torque at 60%W<sub>max</sub>.

Coyle et al. (1991) suggests that trained cyclists are able to produce more propulsive torque over the entire pedal stroke by maximising downstroke torque. There was some evidence to support this in the current study, but differences were not significant at either a low power output of 150W or at power outputs equivalent to 50 and 60% of maximum (Figures 5-7). It therefore appears that pedalling action may not be an important determinant of cycling ability. In fact at 150W the recreational cyclists produced greater propulsive (52.6 vs 44.5Nm) and less negative torque (-10.3 vs -14.5Nm) than the trained cyclists. Thus as speculated above, it could be assumed that as this was at a relatively higher percentage of the recreational cyclists  $W_{max}$ , they had to be more co-ordinated and efficient in maximising propulsive torque values.

Results of the current study suggest higher positive crank torque production seems not to be associated with an increased oxygen uptake and lower efficiency when cycling at a constant work rate and cadence. These findings, supported by others, failed to identify any significant relationships between efficiency and pedalling technique over the crank cycle (Lafortune & Cavanagh, 1983; Coyle et al., 1991; Mornieux et al., 2006). The lack of a relationship between GE and CTE could be explained by the fact that CTE was calculated over the whole crank cycle. This calculation is therefore influenced by both high and low pedal forces produced during the propulsive and recovery phases of the pedal cycle. Therefore, the ability to attribute changes in GE to just one phase is not possible. Metabolic cost on

the other hand is mainly incurred during the high pedal forces of the downstroke phase. This is especially true if cyclists have a passive recovery upstroke phase to their pedalling action and rely on the downstroke of the opposite leg to perform the action. Consequently the majority of energy will be spent during the downstroke phase. Thus it might be more relevant to only assess pedalling effectiveness during this phase (Coyle et al., 1991; Sanderson 1991; Sanderson & Black, 2003) in order to explain individual differences in GE. However, Kautz and Neptune (2002) and Neptune and van den Bogart (1998) have suggested that the amount of mechanical work done at the crank does not represent that of the muscles and therefore metabolic cost.

Recently Zameziati et al. (2006) have identified that analysis of the upstroke phase could account for small variations in GE in a group of untrained cyclists. They suggest that if the upstroke phase is an active process (i.e. by performing an active muscle contraction to raise the recovering leg), the rider would be able to reduce non-propulsive forces contrary to the downstroke phase and potentially improve GE. However as their study used untrained cyclists with a wide range of cycling abilities ( $60\%W_{\max}$  range = 80 - 260W), gross efficiencies (range = 15 - 24%) and pedalling strategies (% effective in upstroke phase range = -60 - +2%) it may have been more likely that they would establish a correlation in their data than the current study. The use of a more homogenous population of trained cyclists in the current study ( $60\%W_{\max}$  range = 240-296W; GE range = 18 - 21%; CTE = 77.6 -

91%) with a smaller dispersion of data could therefore account for the different findings.

If higher absolute work rates were used (+250W) in the current study, forcing a more efficient pedal stroke in the trained cyclists, differences between trained and recreational cyclists may have been seen. Figure 8 below illustrates the effect of power output on crank torque effectiveness.

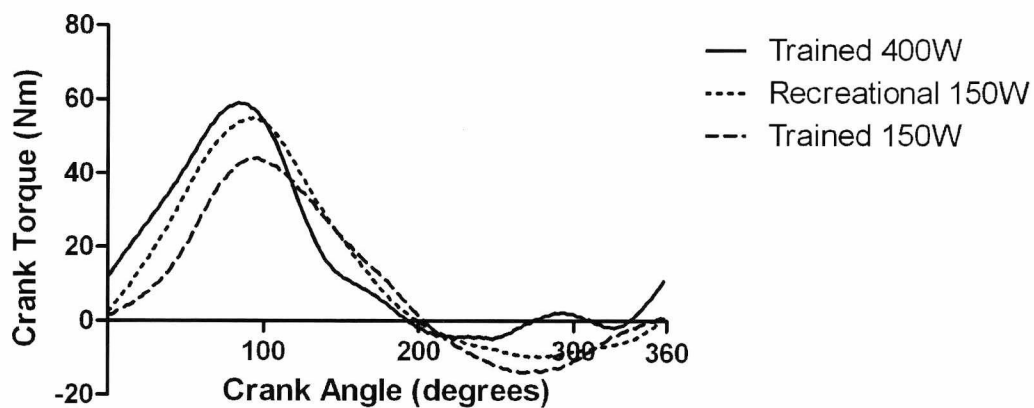


Figure 8. The effect of power output on crank torque.

Figure 8 illustrates one trained cyclist's crank torque at 150 and 400W in comparison with the mean recreational cyclist crank torque. It is possible to see that as the trained cyclist approached a higher percentage of his maximal power output the crank torque profile becomes more optimally orientated towards maximising positive torques and minimising negative ones. Similar peak torque values were found to the untrained group, however the trained cyclist appears to develop less negative peak torque. Further research is needed to investigate the relationship between the oxygen cost of cycling and crank torque data.

#### **4.6 Conclusion**

Using the methods developed in chapter 3, the results of the current study demonstrate that differences in GE exist between trained and recreational cyclists at both absolute and relative work rates. However, as the study employed a cross-sectional design, further research is needed to establish whether these differences are due to training, non-training related aspects (e.g. the mechanics of cycling, limb segment mass differences) or a genetic pre-disposition of the trained cyclists. No differences in CTE were found between trained and recreational cyclists at either of the relative power outputs used. Therefore crank torque effectiveness does not appear to be a factor which influences GE. Greater research is needed to verify this speculation. Further research should also seek to identify whether forces generated at the crank have an influence on GE measured via gases at the mouth and how they are affected by training.

## **CHAPTER 5: CHANGES IN CYCLING EFFICIENCY DURING A COMPETITIVE SEASON**

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Aspects of the following chapter have been peer-reviewed and published in the following article:

Hopker, J.G. Coleman, D.A. Passfield, L. (2009) Changes in cycling efficiency during a competitive season. *Medicine and Science in Sports and Exercise*. 41, 912-919.

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### **5.1 Introduction**

Coyle and colleagues have suggested that cycling GE is a key determinant of endurance cycling performance (Horowitz et al., 1994; Coyle, 1995). Horowitz and co-workers (1994) established the link between GE and cycling performance by comparing these parameters in two groups of cyclists. They found that the group with the higher GE were able to generate a greater power output for the same  $\dot{V}O_2$ . Recently, Coyle (2005) speculated that 7 years of endurance training was responsible for an 8% improvement in GE observed in a champion cyclist. However, the effect of chronic endurance training on GE in competitive cyclists has not been reported.

Further evidence that GE influences cycling performance was provided by Lucia et al. (1998). These authors found that professional riders were able to generate a greater power output than amateurs even though their  $\dot{V}O_{2\text{ max}}$  values were similar. Moreover, changing GE results in a corresponding



alteration in performance. Passfield and Doust (2000) found that a reduction in GE results in correlated changes during a 5 minute cycling performance trial. It has been calculated that a 1% improvement in GE will provide a 63 s advantage in a 40km time-trial (Moseley & Jeukendrup, 2001). Given these links between GE and cycling performance, it is surprising that the possible effect of training on GE has received little research attention.

There is a paucity of information concerning the change in GE in response to training and its variation during a full competitive season. Some studies have found an effect on exercising  $\dot{V}O_2$  which may imply a change in GE. Barbeau et al. (1993) and Norris and Petersen (1998) have both demonstrated that at a fixed moderate intensity exercise  $\dot{V}O_2$  decreases after a period of endurance training. Paton and Hopkins (2005) also found reductions in the  $O_2$  cost of cycling (-3%), but this was following 5 weeks of explosive, high-resistance interval training. Additionally, Bastiaans, et al. (2001) have enhanced DE by replacing 37% of endurance training with explosive strength training for 9 weeks. Only Hintzy et al. (2005) have examined the effects of endurance training on GE. These researchers found a six week training programme in 9 healthy untrained women resulted in significant gains in GE. The authors suggest this improvement in efficiency may be largely due to improved pedalling skill as the participants were previously unaccustomed to cycling. Whether this same improvement occurs in trained cyclists remains unclear.

Typically researchers have tended to use GE as the method for calculating efficiency in cycling, although there are two other calculations that appear in the literature, DE and WE. Both of these terms are defined in Chapter 1 as well as their relative criticisms of on grounds of validity and reliability (Stainsby et al., 1980; Moseley & Jeukendrup, 2001; Kautz and Neptune, 2002). Even so, the longitudinal effects of training on all three of these different methods for calculating efficiency have not been investigated.

## **5.2 Aims of the investigation**

The first requirement in an investigation of the potential training related changes in GE is to establish a reliable measurement which was completed in chapter 3. Following this it was necessary to establish group differences between trained and recreational cyclists which chapter 4 documents. Therefore, the next step is to establishing if GE changes with training in a group of trained cyclists. If efficiency is genetically determined there will be no changes observed with training and it will stay stable over time. If training does influence GE then establishing the type, intensity and duration required to bring about increases in this important performance parameter is crucial.

Accordingly, the aim of this study was to determine if endurance training results in a change in GE in competitive cyclists. A secondary aim of the study was to examine if any changes identified in GE over a training/racing season (pre-competition, competition, post competition and transition

phases when training volume, intensity and duration will tend to vary), were related to the training undertaken.

### **5.3 Methods**

#### **Participants**

Fourteen endurance-trained competitive male cyclists were selected from local clubs as well as professional teams to take part in this investigation with physical characteristics at the first test of (mean  $\pm$  SD): 34  $\pm$  8 yr, 74.3  $\pm$  7.4 kg,  $W_{\max}$  406  $\pm$  43 W,  $\dot{V}O_{2\max}$  59.5  $\pm$  3.8 mL $\cdot$ kg<sup>-1</sup> $\cdot$ min<sup>-1</sup>. The sample size was derived from work conducted in chapter 3. All cyclists had a minimum of 3 years competitive cycling experience and familiarity of laboratory-based testing.

#### **Procedures**

All participants visited the laboratory five times during the study. The assessment consisted of a combined threshold test and maximal aerobic power test using a continuous incremental protocol. Following an initial 8 minute period of unloaded cycling (for the determination of work efficiency) participants completed a continuous incremental exercise test. Starting at 150W, work rate increased by 30 W every 8 min. Blood lactate samples were taken every 4<sup>th</sup> and 8<sup>th</sup> minute of each work stage until the measured concentration of lactate in the blood reached 4 mmol $\cdot$ L. Power and heart rate were recorded continuously. Expired gases were collected over the final three minutes of each 8 minute bout of exercise completed (Quark b2, Cosmed, Italy) for the measurement of  $\dot{V}O_2$  and RER. These

data were used to calculate GE, DE and WE as described in chapters 1 and

2.

Lactate Threshold (LT) was determined as the power eliciting a 1 mmol·L increase in blood lactate above baseline (Coyle et al. 1984), and OBLA as a measured 4 mmol·L concentration of lactate in the blood (Heck, 1985). Once 4mmol·L was obtained cyclists were immediately switched to a fast ramp protocol to elicit  $\dot{V}O_{2\max}$ . This protocol used a 30W per minute ramp rate continued until volitional exhaustion. Maximal power output ( $W_{\max}$ ) was calculated using the  $W_{\max}$  formula (Kuipers et al. 1985) outlined in chapter 3.

### *The Training Period*

The initial testing (Test 1) took place in January. Subsequent tests were timed to correspond with the end of a training phase. Thus Test 2 was conducted at the end of 'pre-competition' period; Test 3 at the end of the 'competition' period; Test 4 at the end of the 'post competitive phase and finally Test 5 at the end of the 'transition phase' (see Figure 9 below). These phases were approximately 3 months in duration. All participants were instructed to refrain from strenuous exercise for 24 h immediately prior to each test and were not informed of their performance in any of the assessments until all phases had been completed.

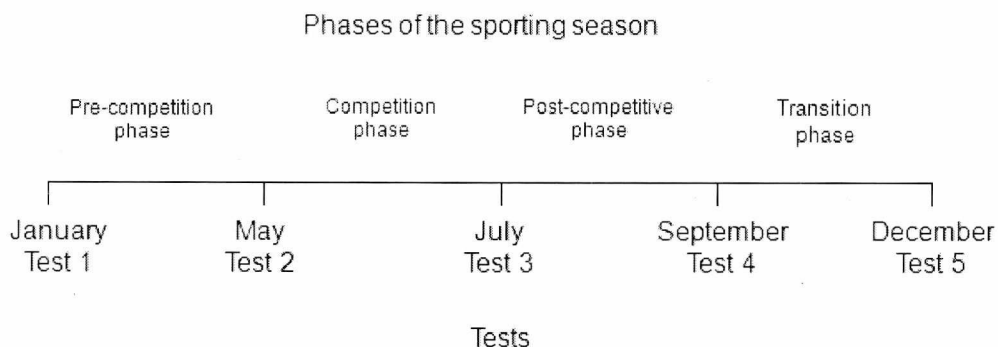


Figure 9. Schematic diagram illustrating the testing schedule and phases of the competitive cycling season.

Throughout the study, participants served as their own control maintaining their normal diet and training patterns. Training was not manipulated or altered and the cyclists were asked to train and compete in their normal training macrocycles. Cyclist's training session/race data was monitored and quantified in terms of duration and intensity using heart rate (Polar S725X, Polar, Kempele, Finland), with data sampling every 5 seconds. Total training time as well as percentage of time spent training below LT, between LT and OBLA and then time spent above OBLA was calculated.

#### *Data Analysis*

Prior to all statistical analyses, data were checked for violations of normality using a Shapiro-Wilk test. Comparisons of the mean of measured variables across the three tests were assessed using repeated measures Analysis of Variance. Unadjusted Post Hoc analysis (Perneger, 1998), was then conducted to identify where any significant differences within the data

existed. Relationships between training intensity and time recorded via heart rate and changes in physiological variables were analysed using Pearson's Correlation Coefficient and, where necessary a partial correlation to control for either training volume or intensity. Submaximal efficiency data were averaged to the highest common power output achieved across the Tests. Statistical significance was set at  $P \leq 0.05$  for all analysis conducted. All values are expressed as mean and standard deviation (mean  $\pm$  SD) unless otherwise stated.

#### **5.4 Results**

Twelve cyclists completed the study. Two cyclists withdrew part way through the year for personal reasons and so their data were excluded prior to analysis.

##### **Training Time**

Data were collected and analysed for the two month period immediately prior to each testing session. Between Test 1 and Test 2 total weekly training completed was  $11.4 \pm 6.5$  hours, between Tests 2 and 3 this time decreased to  $9.9 \pm 4.9$  hours, and rose between Test 3 and 4 to  $12.9 \pm 11.4$  hours. Finally, weekly total training time decreased again to  $6.8 \pm 5.2$  hours between Tests 4 and 5. Throughout the phases of the season there were no significant differences between amount of training per week below LT (sub LT) ( $P=0.17$ ) and between LT and OBLA (LT-OBLA) ( $P=0.39$ ). However the above OBLA training (above OBLA) significantly decreased between Tests 4 and 5 ( $P<0.01$ ) (Table 10 below).

Table 10. Cyclists' mean absolute time (minutes) spent training/racing at relative exercise intensities throughout the sporting season. Percentage of total time spent training/racing given in brackets.

Training Zone	'Pre-Comp'	'Comp'	'End-Comp'	'Post-Comp'
Sub LT	3992 (73)	3501 (74)	3959 (73)	2333 (72)
LT – OBLA	781 (14)	621 (13)	789 (14)	766 (24)
Above OBLA	678 (12)	619 (13)	697 (13)	164 (5)

### **Phase related changes in Efficiency**

Efficiency changed significantly ( $P<0.01$ ) across the training phases. GE was significantly higher at Tests 2, 3 and 4 (see Table 11 and Figures 10 & 11) compared to Test 1 and Test 5. There were no significant differences across Tests 2-4 ( $P>0.05$ ).

Table 11. Mean gross efficiency averaged across all power outputs to the highest common (achieved across all Tests) for each cyclist. Mean  $\pm$  SD.  
\*Significantly higher than Tests 1 and 5 ( $P<0.01$ )

	Gross Efficiency (%)
Test 1 (Jan.)	19.6 $\pm$ 0.7
Test 2 (April)	20.6 $\pm$ 1.0*
Test 3 (July)	20.6 $\pm$ 1.3*
Test 4 (Sept.)	20.3 $\pm$ 0.8*
Test 5 (Dec.)	19.4 $\pm$ 0.8

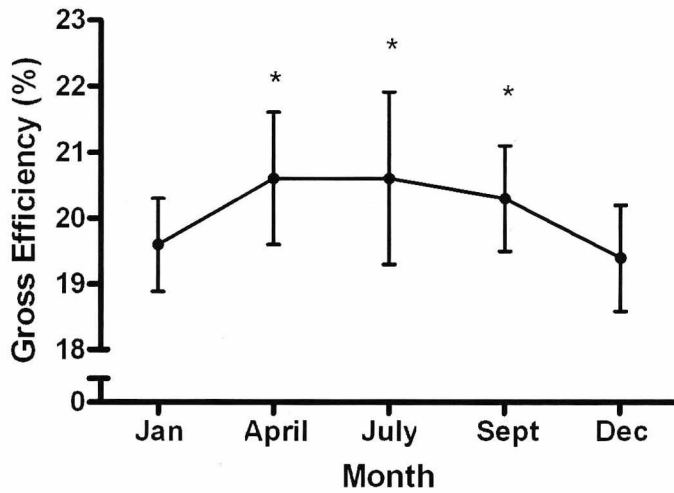


Figure 10. Illustration of the mean change in gross efficiency across the different phases of the season (group mean efficiency across all common power outputs  $\pm$  SD). \* Significantly higher than tests 1 and 5 ( $P < 0.01$ ).

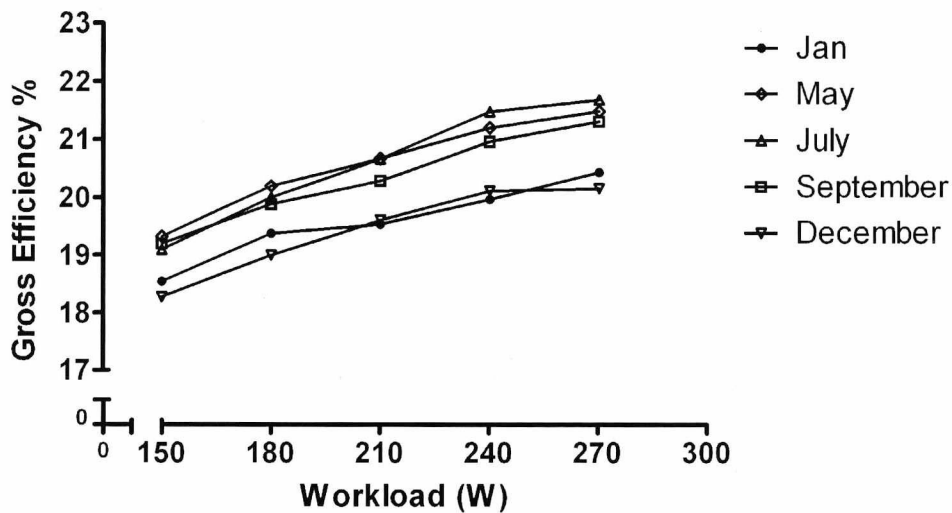


Figure 11. Illustration of gross efficiency measured across each common power output during the season

WE was significantly higher at Test 1 compared to Tests 3 and 5 ( $P = 0.02$ ) (see Table 12 and Figures 12 and 13). The WE recorded at Test 5 was significantly lower than all other tests ( $P < 0.05$ ).



Table 12. Mean work efficiency averaged across all power outputs to the highest common (achieved across all Tests) for each cyclist. Mean  $\pm$  SD.

\*Significantly higher than Tests 3 and 5. #Significantly lower than all other Tests.

	Work Efficiency (%)
Test 1 (Jan.)	33.3 $\pm$ 3.0*
Test 2 (April)	33.2 $\pm$ 2.6
Test 3 (July)	32.2 $\pm$ 2.4
Test 4 (Sept.)	32.9 $\pm$ 2.5
Test 5 (Dec.)	30.8 $\pm$ 1.6#

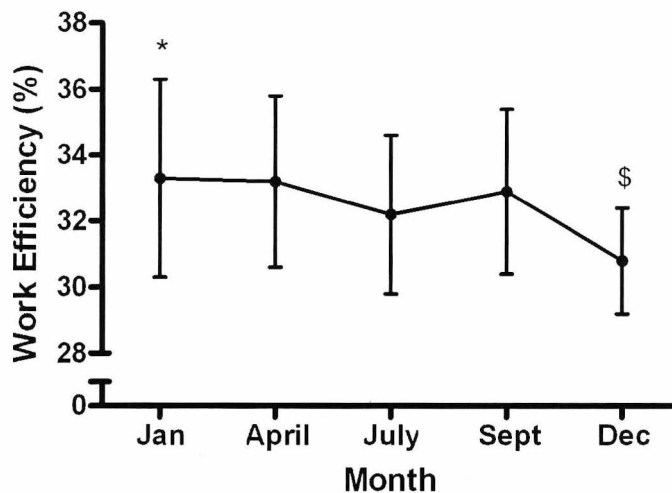


Figure 12. Illustration of the mean change in work efficiency across the different phases of the season (group mean efficiency across all common power outputs  $\pm$  SD). \*Significantly higher compared to Tests 3 and 5 ( $P=0.02$ ). \$ Significantly lower than all other Tests ( $P<0.05$ ).

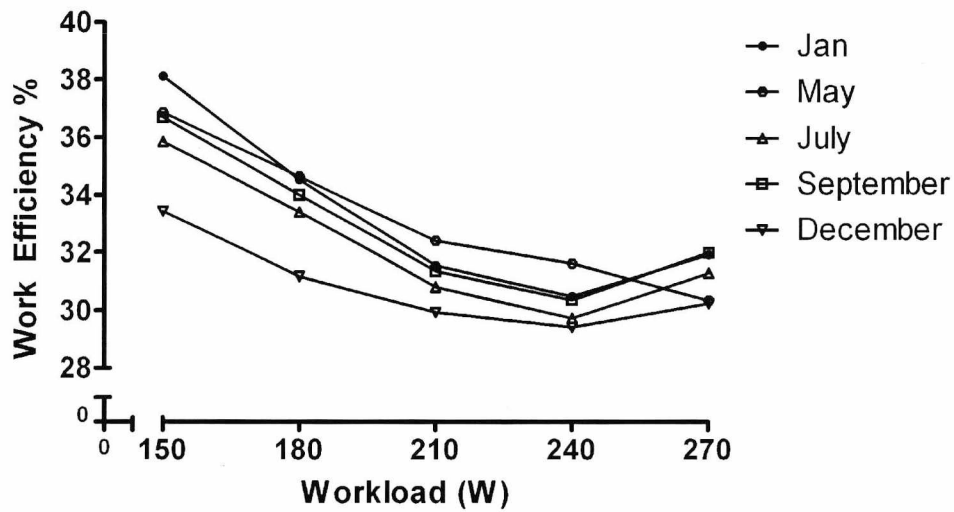


Figure 13. Illustration of Work Efficiency measured across each common power output during the season

There were no significant changes in DE throughout the course of the season ( $P > 0.05$ ) (see Table 13 and Figure 14).

Table 13. Mean Delta Efficiency averaged across all power outputs to the highest common (achieved across all Tests) for each cyclist. Mean  $\pm$  SD.

	Delta Efficiency (%)
Test 1 (Jan.)	26.0 $\pm$ 4.4
Test 2 (April)	26.7 $\pm$ 1.7
Test 3 (July)	29.6 $\pm$ 5.7
Test 4 (Sept.)	26.3 $\pm$ 3.9
Test 5 (Dec.)	25.4 $\pm$ 2.9

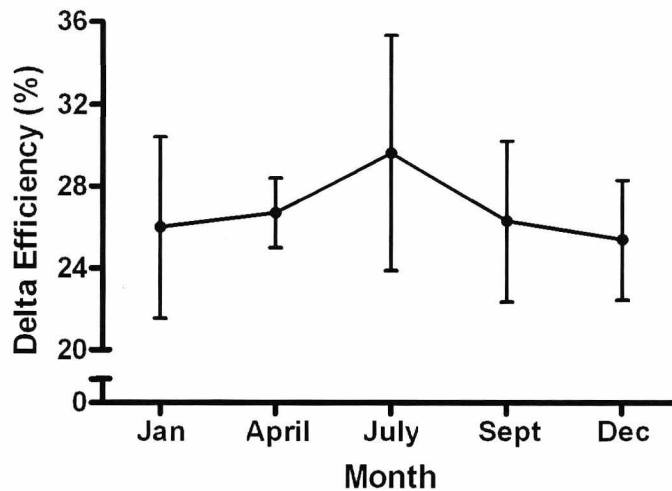


Figure 14. Illustration of the mean change in Delta Efficiency across the different phases of the season (group mean efficiency across all common power outputs  $\pm$  SD).

#### **Relationship between training and phase related changes in efficiency**

GE at Test 2 was significantly ( $r=0.84$ ;  $P<0.01$ ) related to the total time spent training over the pre-competitive phase (see Figure 15a). When using a partial correlation to control for volume of training, a significant relationship ( $r=0.80$ ;  $P=0.01$ ) was also identified between GE and the time spent above OBLA (see Figure 15b). The GE at Test 3 was significantly related to the total time spent training ( $r=0.80$ ;  $P=0.01$ ), and also time below LT ( $r=0.78$ ;  $P=0.01$ ) during the pre-competitive phase. When analysing the change in GE between Test 2 and 3 they were most strongly related to the total time spent training between LT and OBLA intensities ( $r=0.87$ ;  $P=0.02$ ) within this phase.

During the competitive phase a significant inverse relationship ( $r=-0.85$ ;  $P<0.01$ ) between the change in GE and percentage change in training sub LT was found (see Figure 15c). A significant positive relationship ( $r=0.76$ ,  $P=0.01$ ) was measured between the change in GE and the percentage of training completed between LT and OBLA intensities (see Figure 15d). Finally, a significant positive relationship existed between GE and total time spent training between LT and OBLA during the post competitive phase ( $r=0.70$ ;  $P = 0.03$ ). Those riders who maintained GE during this phase spent more time between LT and OBLA intensities. There were no significant relationships between GE and training time in the data collected over the transition phase.

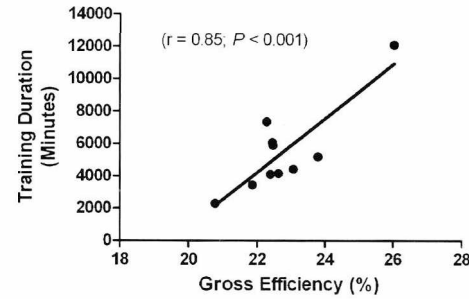


Figure a.

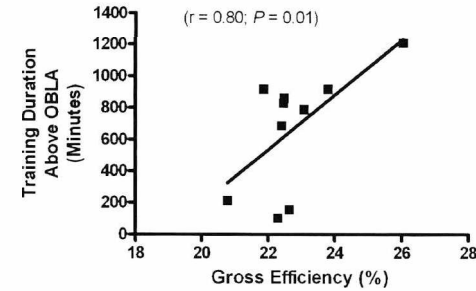


Figure b.

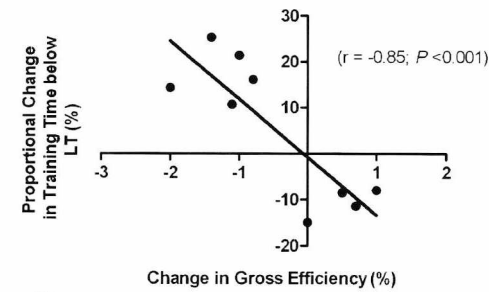


Figure c.

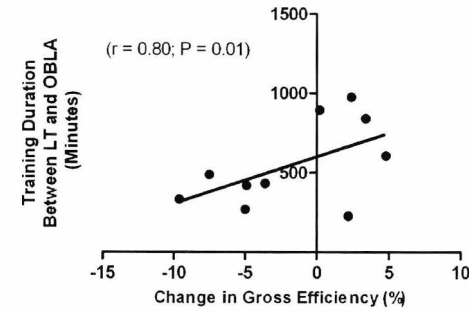


Figure d.

Figure 15. Correlations illustrating relationships between training and GE. Figure a. Relationship between GE at Test 2 and the total time spent training over the pre-competitive phase. Figure b. Relationship between GE and the time spent above OBLA. Figure c. Illustrates the inverse relationship between the change in GE and percentage change in training sub LT the competitive phase. Figure d. The relationship between the change in GE and the percentage of training completed between LT and OBLA intensities.

### Phase related changes in Crank Torque

There were no significant changes in positive crank torques over the phases of the season at set power outputs of 210W ( $P=0.41$ ) or 270W ( $P=0.56$ ) (Figures 16 and 17 below). Net (210W  $P=0.43$ ; 270W  $P=0.25$ ) and negative (210W  $P=0.10$ ; 270W  $P=0.19$ ) torques also showed no significant changes over the season. Although as demonstrated by Figure 16 below, both downstroke and upstroke torques were lower at Test 2 compared to the other Tests at 210W. There were no relationships present between GE and total, net or negative torque production ( $P>0.05$ ).

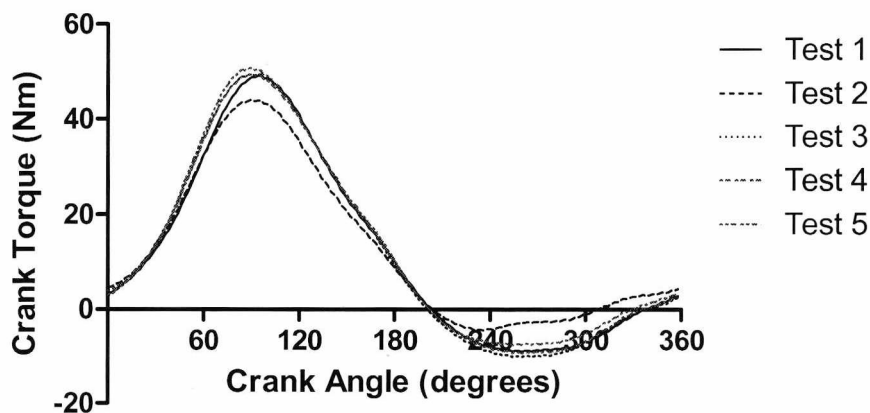


Figure 16. Mean Crank Torque averaged over both cranks across the phases of the season at 210W

Pedal cadence was not constrained during the final maximal part of the tests (once 4 mmol·L had been achieved). Pedal cadence over this phase of the test did not significantly change over the different phases of the cycling season (mean cadence range over the season = 87-88 rev.min<sup>-1</sup>;  $P>0.05$ ).

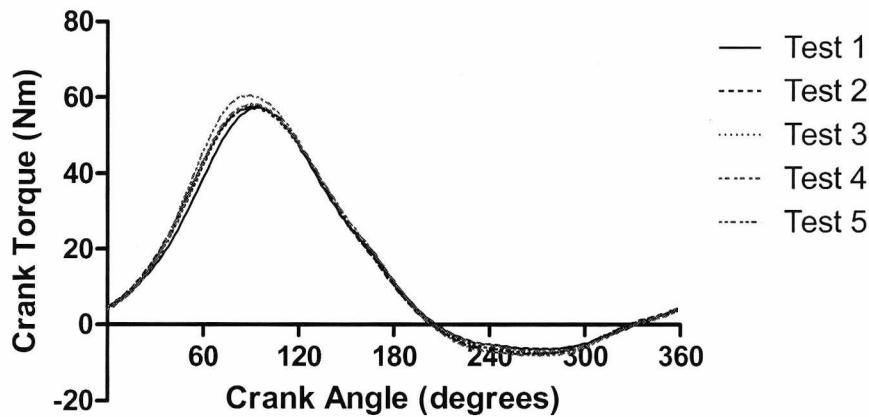


Figure 17. Mean Crank Torque averaged over both cranks across the phases of the season at 270W

### **Phase related changes in other physiological variables**

All physiological responses measured during the different phases of the sporting season are presented in Table 14. As shown in Table 14,  $W_{\max}$  was significantly higher across the Tests 1-3 compared to Tests 4 and 5 conducted in the later part of the season. There were no significant changes in measured mass, maximal heart rate or  $\dot{V}O_{2\max}$  ( $L \cdot \text{min}^{-1}$ ). When scaled to body mass,  $\dot{V}O_{2\max}$  was significantly higher at Test 2 compared to all other times of the season. Measured LT occurred at a significantly higher power output across the main competition phases of the season (Tests 2-4) as did the point of OBLA. Heart rate measured at 270W was significantly lower at Test 3 compared to all other phases of the season.  $RER_{\text{submax}}$  was significantly lower at Test 2 compared to all other phases of the season, and was significantly higher at Test 4 compared to all other phases.

Variable	Test 1	Test 2	Test 3	Test 4	Test 5
Mass (kg)	74.3 ± 7.4	74.4 ± 6.6	74.9 ± 6.5	72.1 ± 3.6	74.3 ± 6.3
W <sub>max</sub> (W)	406 ± 43	421 ± 46	410 ± 31	390 ± 27*	399 ± 37*
$\dot{V}O_{2max}$ (L·min <sup>-1</sup> )	4.4 ± 0.4	4.5 ± 0.4	4.2 ± 0.3	4.2 ± 0.3	4.4 ± 0.5
$\dot{V}O_{2max}$ (mL·kg <sup>-1</sup> ·min <sup>-1</sup> )	59.5 ± 3.8	60 ± 5.6 <sup>§</sup>	59.2 ± 4.9	57.5 ± 3.4	60.8 ± 4.4
LT (W)	257 ± 28 <sup>#</sup>	270 ± 26	281 ± 29	255 ± 16	241 ± 23 <sup>#</sup>
OBLA (W)	297 ± 35 <sup>~</sup>	308 ± 35	316 ± 27 <sup>&amp;</sup>	285 ± 16	275 ± 28 <sup>~</sup>
HR <sub>max</sub> (b·min <sup>-1</sup> )	184 ± 12	183 ± 10	181 ± 9	180 ± 9	185 ± 11
HR at 270W (b·min <sup>-1</sup> )	160 ± 10	158 ± 12	152 ± 10 <sup>^</sup>	159 ± 10	162 ± 10
RER <sub>submax</sub>	0.92 ± 0.00	0.87 ± 0.00 <sup>^</sup>	0.92 ± 0.00	0.96 ± 0.01 <sup>&amp;</sup>	0.93 ± 0.00

\*Significantly lower than Tests 1-3  $P < 0.05$ ; <sup>§</sup>Significantly higher than Tests 1 and 3-5  $P < 0.05$ ; <sup>#</sup>Significantly lower than Tests 2-4  $P < 0.05$ ;

<sup>~</sup>Significantly lower than Tests 2-4  $P < 0.05$ ; <sup>&</sup>Significantly higher than all other Tests  $P < 0.05$ ; <sup>^</sup>Significantly lower than all other Tests  $P < 0.05$

RER<sub>submax</sub> is calculated mean RER up to highest common power output across all Tests.

Table 14. Performance related changes measured during the continuous incremental test across the five Tests during the season



Finally ventilation ( $\text{L}\cdot\text{min}^{-1}$ ) decreased between Tests 1 and 2 (69.6 vs 63.9  $\text{L}\cdot\text{min}^{-1}$  respectively) however was statistically unchanged over the course of the season. Even so, two significant inverse correlations existed between changes in VE and GE (Figure 18), specifically between Tests 1 and 2 ( $r = -0.97$ ;  $P < 0.01$ ) and Test 4 to 5 ( $r = -0.78$ ,  $P = 0.01$ ).

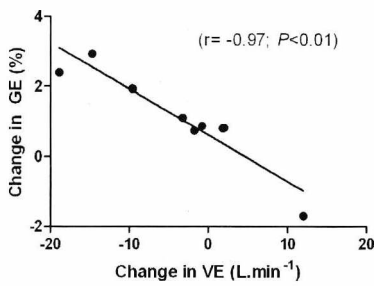


Figure a

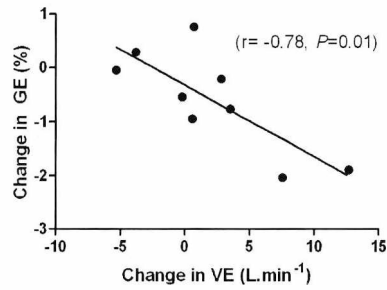


Figure b

Figure 18. Relationship between changes in GE (%) and changes in VE ( $\text{L}\cdot\text{min}^{-1}$ ), averaged to the highest common work rate. Figure a. Relationship over the pre-competition phase. Figure b. Relationship over the transition phase.

## **5.5 Discussion**

The main findings of this study were that GE significantly changed across the competitive cycling season in 12 well-trained cyclists. These changes were related to the volume and intensity of the training undertaken. The proportion of time that cyclists spent at intensities above their OBLA heart rate correlated to the changes in GE. Cyclists' WE significantly decreased over the competitive season, whilst DE did not significantly change.

### **Changes in Cycling Efficiency**

Previously the effect of long-term endurance training on GE in competitive cyclists has not been fully investigated. Coyle (2005) observed an 8% increase in GE over a 7 year period in one elite cyclist. Others (Gissane et al., 1991; Hintzy et al., 2005) have also demonstrated significant increases in GE as a result of training, albeit in untrained individuals. Consistent with the present study, Barbeau et al. (1993) found that elite cyclists'  $\dot{V}O_2$  was lower at 250W during the months of May and July, thus demonstrating an adaptation from the high volume phase of training. However, the protocol used during this study consisted of 2 minute increments which have been suggested to be inappropriate for measuring GE (Wasserman et al., 2005).

In contrast to the measures of GE, WE decreased over the course of the season (33.3-30.8%) and DE remained unchanged. The way in which WE is calculated may partly explain its apparent reduction. The measurement of WE involves a baseline subtraction of the metabolic cost of "unloaded

cycling” and its use has been criticised as a consequence (Stainsby et al., 1980; Kautz & Neptune, 2002). Figure 19 below illustrates the crank torque profile of one cyclist during both unloaded and loaded (150W) cycling conditions. It is clear from the profiles that an unloaded condition has minimal resemblance to the loaded situation. This may also suggest differences in the degree and timing of muscle recruitment between the two conditions. Thus the validity of subtracting the oxygen cost of an unloaded condition from loaded cycling as used in the WE calculation has to be questioned.

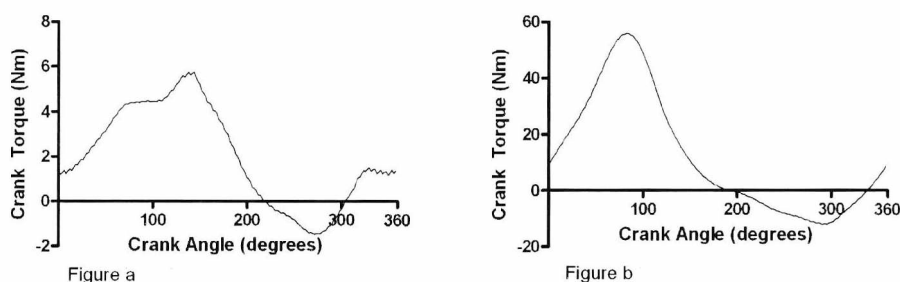


Figure 19. Crank torque profiles of one cyclist at two different power outputs. Figure a. The cyclist’s crank torque profile during unloaded cycling. Figure b. The crank torque profile at 150W.

Paradoxically, a decrease in WE could be apparent if the cost of the subtracted baseline measured during unloaded cycling is also diminished. In the present study, a higher  $\dot{V}O_2$  at 0W was observed in January, although it was not significantly different from subsequent measures ( $1.12 \text{ L}\cdot\text{min}^{-1}$  Test 1 vs  $1.06 \text{ L}\cdot\text{min}^{-1}$  Test 4 as the next highest measurement;  $P=0.89$ ). This resulted in a larger baseline being subtracted from the total  $O_2$  cost than for

subsequent tests. Such a decrease in the O<sub>2</sub> cost of moving the legs may be due to riders improving their ability to complete the action of unloaded cycling. When cycling at zero watts the energy generated in the legs would have to be decreased by additional negative muscle work as no external work is present to do so (Kautz & Neptune, 2002). As this is different to normal pedalling, the metabolic cost is likely to be different as well. Similarly to WE, changes in DE were not consistent with those of GE. DE has been suggested to be the most valid measurement of muscular efficiency (Whipp & Wasserman, 1969) and this may therefore indicate that changes outside the muscle are responsible for the changes in GE. However, it is possible that the inherent variability of this measure (~10%), as shown in chapter 3 and by Moseley and Jeukendrup (2001), may have masked any meaningful changes that occurred (see Figure 12; 7% change across the year,  $P=0.76$ ).

The current study found a 5% relative change across all cyclists GE from Test 1 to Test 2. Passfield and Doust (2000) demonstrate that such changes in GE can have a significant effect on cycling performance. The largest absolute mean change in GE occurring during the year was ~1%. A change of this magnitude has been suggested to result in a 63 s improvement for a 40 km time-trial (Moseley & Jeukendrup, 2001). However, the mean change in GE for the whole year was 0.02%. Thus, it appears that GE increases initially, but then falls back towards a baseline at the end of the year (see Figure 8). This finding may indicate that the changes observed are a physiological training response to the volume and intensity of training

undertaken. The changes found in current year long study are consistent with those reported by Coyle (2005) in that a 1-2% increase in efficiency is capable over a period of training and racing. Although the current study tends to support those that have suggested the changes observed in Coyle's case study may be the result of variations in the timing of testing rather than a chronic adaptation (Martin et al., 2005; Schumacher et al., 2005).

The current data suggest that if athletes are carefully controlled prior to testing, the usage of a long duration work stage protocol can be reliably used for identifying longitudinal changes in GE. To standardise GE measurements for tracking longitudinal changes, tests should be scheduled for the same periods of time year on year with prior training/racing being taken into consideration. In addition other factors such as ambient temperature, pedal cadence, bicycle set-up and testing intensities should all be standardised as they have been previously shown to acutely influence GE values (Marsh & Martin, 1997; Price & Donne, 1997; Hettinga et al., 2007). The current data also suggest that testing within the competition phase is of importance to identify maximal GE values.

### **Training related changes**

The percentage training time spent in the intensity bands used by cyclists in the present study was comparable to the world class cyclists measured by Lucia et al. (2000b). Although riders in the present study appear to have more of an emphasis on higher intensity training compared to the professional cyclists in Lucia's study. Cyclists in the current study spent a

greater percentage of their total time at intensities above OBLA (4.9 vs 12% in the pre-competition phase; 8.1 vs 13% during the competitive phase and 1.5 vs 5% in the transition phase). However when absolute time is considered, the largely amateur cyclists in the current study completed considerably less total time training than has been previously reported for professional cyclists (an average of 11 vs 22 hours per week during the pre-season phase [Hawley & Stepto, 2001]).

The main training related changes in GE occurred between Test 1 and 2 and were significantly related to the total time spent training over the two months prior to Test 2. Interestingly, a partial correlation to control for training volume revealed a significant correlation between the change in GE and the proportion of the total time spent at or above OBLA. This observation suggests that not only is training volume important, but also the amount of high intensity training completed during the period. These trends in training-related changes in GE were also observed between Tests 2 and 3. An inverse relationship was found between time spent at intensities below LT and GE. Therefore, the time spent training between LT and OBLA appeared to be important if GE was maintained during the competition phase of the season.

A putative mechanism for the increase in GE is a transformation of muscle fibre type characteristics (Coyle, 1995). Cyclists with a high percentage of Type I fibres are able to produce significantly more work for a given  $\dot{V}O_2$ . (Horowitz et al., 1994). Baumann et al. (1987) demonstrated that

adaptations in muscle isoform patterns could occur over an 8 week period of cycle ergometer training for 30 minutes, 5 days per week. Similarly Burgomaster et al. (2008) have shown that short term high intensity interval type training (>OBLA) can elicit changes consistent with fibre type transformation. However no measures of myosin type were conducted during their study to support this.

Muscle typology is unlikely to be the sole determinant of GE though. Several studies have found that changes in GE could be induced by alterations in muscle recruitment and pedalling strategies (Zameziati et al., 2006; Cannon et al., 2007; Korff et al., 2007) as discussed below. A reduction in ventilation (VE) could also contribute to an increase in GE. Ventilation rate at each work intensity decreased over the pre-competition and increased over the transition phase, albeit not significantly ( $67.5 \text{ L}\cdot\text{min}^{-1}$  at Test 1;  $66.7 \text{ L}\cdot\text{min}^{-1}$  at Test 3;  $71.3 \text{ L}\cdot\text{min}^{-1}$  at Test 5). However a significant inverse relationship between GE and VE was found across these phases (Figure 18 a&b). Reducing the metabolic demand of the respiratory muscles could lead to decreases the  $\text{O}_2$  cost of exercise. Harms et al. (2000) found that decreasing the work of breathing during strenuous exercise significantly increases the exercise tolerance. Therefore the changes seen in GE in the current study could be accounted for by a decreased demand for oxygen of the respiratory muscles. In turn this would allow redistribution of blood flow and  $\dot{V} \text{O}_2$  to the leg musculature (Harms et al., 2000). This is a potential adaptation which may have occurred due to the high ventilation rates encountered during high intensity exercise which formed a part of the

cyclist's training between Test 1 and 2. Alternatively, an improved capacity to buffer lactic acid in the exercising leg muscles as a result of training may have led to a lower ventilatory drive (Cassaburi et al., 1987). A lower rate of carbohydrate oxidation may have resulted in lower CO<sub>2</sub> production (RER 0.92 vs 0.87 Test 1-2) (Westgarth-Taylor et al., 1997) which, in turn, could have also caused a lower drive for ventilation at the same exercise power output. The metabolic demand of the respiratory muscles would therefore be decreased due to the lower ventilatory rate, thus allowing for a reduced total body oxygen cost of exercise.

The significant decrease in GE observed between Tests 4 and 5 was accompanied by a varied reduction in both training volume and intensity amongst individuals. The varied nature of the reduction in training amongst individual cyclists meant that no significant correlations were found between GE and training time over the transition period. The loss of training-induced physiological adaptations as seen in the current study are likely to be the result of an insufficient training stimulus (Lucia et al., 2000a) during the transition phase of a cyclist's season. This reversibility may indicate metabolic adaptations are the primary factor underpinning the changes in GE observed in this study. Further research may attempt to maintain the intensity and/or volume of training in cyclists over this transition period of the season to establish if GE can be sustained.



### **Changes in crank torque**

There is much debate about the most appropriate pedalling strategy for cyclists to adopt. Anecdotal reports from cyclists suggest that trying to pedal in circles is the most appropriate strategy. Lance Armstrong and his coach Chris Carmichael actively tried to change Armstrong's pedal action to one of pedalling in 'perfect circles' (Armstrong & Carmichael, 2006). However the literature does not tend to support this strategy as being most efficient. Actively attempting to pedal in circles has been shown to be no more beneficial than riding with a preferred pedalling action or focusing on pushing down on the pedal (Korff et al., 2007). Korff et al. (2007) demonstrated that actively attempting to pull up during the upstroke was more mechanically efficient but least metabolically efficient. They conclude that mechanical effectiveness is not indicative of GE. This suggests that muscle recruitment rather than mechanical effectiveness at the crank may be more of a determinant of GE. The results of the current study would tend to support this position. No relationship was found between total, net or negative torque at the crank and GE at any of the power outputs studied. Crank torque and pedal cadence (during the maximal part of the test) did not significantly change over the course of the year even though changes were seen in GE, training volume and intensity. Thus it could also be concluded that cyclist's preferred method of pedalling is relatively robust and does not alter over a prolonged period of time.

It is possible that the cyclists may have developed more coordinated muscle recruitment strategies during pedalling as their training volume and intensity

increased over the pre-competitive phase. Studies have demonstrated that when task dynamics of cycling are altered (e.g. via the usage of a non-circular chainring), the nervous system is able to adapt. It does this by changing activation dynamics associated with muscle force development (Brown et al., 1996). Neptune and Herzog (2000) identified that when using a novel oval chainring, cyclists advanced their muscle excitation earlier in the crank cycle (between 2-10°). Thus if individual muscle activation and deactivation dynamics are enhanced along with better coordination between agonists and antagonists there is the possibility that greater power could be generated during pedalling for the same oxygen cost. However as no electromyographic data was collected during the current study it is impossible to support this speculation.

### **Changes in other physiological variables**

Consistent with the findings of the present study, Lucia et al. (2000b; 2001a) and Chicharro et al. (2000) found no significant change in professional cyclist's  $\dot{V}O_{2\max}$  ( $L \cdot \min^{-1}$ ) during a racing season. These authors attribute this finding to the extensive training history of their participants and their already high values (mean =  $\sim 74 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ ). It is noted however, that the  $\dot{V}O_{2\max}$  values of the participants in the current study seem low considering the training status of the riders ( $4.5 \text{ L} \cdot \text{min}^{-1}$ ;  $62.0 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$  at Test 2). This may be due to the extended test protocol employed in the present study, which may also have reduced sensitivity to changes in this parameter. Interestingly though, an inverse relationship was identified between changes in GE and changes in  $\dot{V}O_{2\max}$  (Figures 20 a-d). Figure 20

below demonstrates inverse correlations between these parameters which were strongest during the pre-competitive, competitive and post competitive phases of the season ( $r=-0.51$ ,  $r=-0.67$  &  $r=-0.66$  respectively). Correlations weakened during the transition phase ( $r=0.30$ ). It is not clear whether the mechanisms for change in GE have a negative influence on  $\dot{V}O_{2max}$  or vice versa. The findings extend the work of Lucia et al. (2002a) who identified an inverse correlation between  $\dot{V}O_{2max}$  and economy by suggesting GE increased to compensate for riders who have a low  $\dot{V}O_{2max}$ . Results of the current study suggest that this inverse relationship may also occur as a result of training. Atkinson et al. (2003b) have suggested that a spurious correlation could account for much of Lucia et al's (2002a) relationship found. This is not the case in the current study as the same factor is not used in the calculation of both correlated variables. Interestingly this inverse relationship has also previously been reported in highly trained runners (Morgan & Daniels, 1994).

In agreement with the findings of Jeukendrup et al. (2000) and Hoogeveen (2000) the  $W_{max}$  of the cyclists changed significantly over the training year. Most notably,  $W_{max}$  was highest in the build up to, and during the competitive phases of the season. Viewed in conjunction the inverse correlation between  $\dot{V}O_{2max}$  and GE it could be suggested the increase in  $W_{max}$  is to some extent mediated by increases in GE.

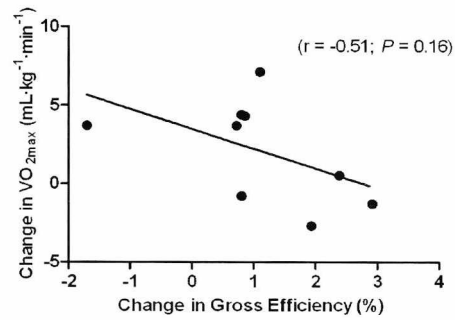


Figure a

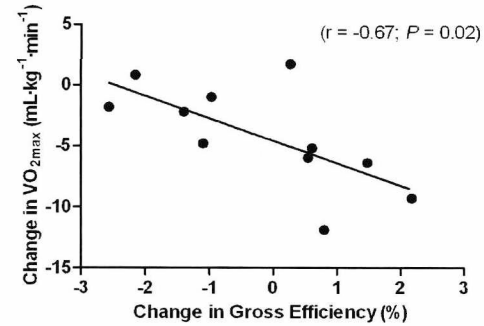


Figure b

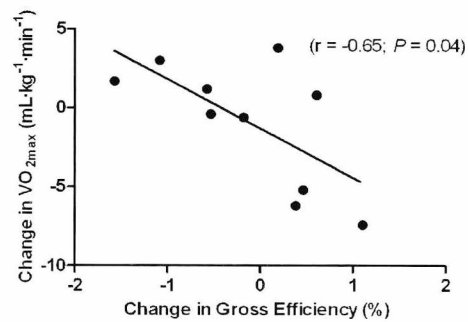


Figure c

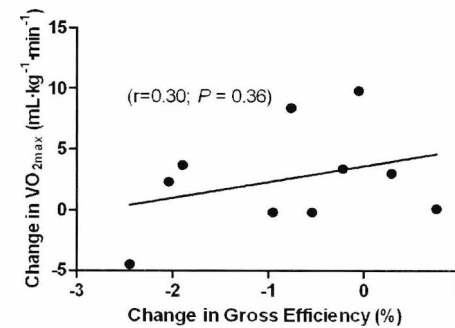


Figure d

Figure 20. Inverse relationship between changes in GE and changes in  $\dot{V}O_{2max}$  over the season. Figure a. Inverse relationship over the Pre-season phase. Figure b. Inverse relationship over the Competition phase. Figure c. Inverse relationship over the Post-competition phase. Figure d. Relationship over the Transition phase.

As shown in figure 20, adaptations manifested at sub-maximal intensities may be more important than maximal parameters. Lucia et al. (2000b) suggest that changes in the ability to sustain a high percentage of their  $\dot{V}O_{2max}$  may be more important than possessing a high peak power output. Thus routine monitoring of GE is critical due to its importance in determining percentage of  $\dot{V}O_{2max}$  that can be sustained. The results of the current study also demonstrate fluctuations in power output at LT and OBLA over the course of the competitive season. Similar changes were also seen by Lucia et al. (2000b) who found that blood lactate concentration at submaximal work rates were significantly lower during the competitive phase of the season compared to rest and pre-competition phases.

As shown in Table 14, power output at threshold intensities increased significantly over the course of the season which is in accordance with previous findings in endurance cyclists (Chicharro et al., 2000; Lucia et al., 2000b; Lucia et al., 2001c). LT and OBLA occurred at significantly higher power outputs in the competitive phases of the season (Test 2 and 3) when compared to pre- or post-competition phases (Tests 1 and 4). The agreement observed between these improvements and the time spent training in the higher intensity exercise domains, further highlights the possible importance of this type of training. Heart rate at a power output of 270W was significantly lower at Test 3 compared to all other phases but the changes do not coincide with the changes seen in GE.

## **5.6 Conclusion**

In conclusion this study demonstrates that GE in competitive cyclists changed significantly over the course of a racing season. This study found that GE was highest during the competitive phases of the season and was related to the proportion of high intensity training undertaken. However, the mechanisms for these changes in GE require further investigation. It is recommended that GE should be monitored as part of a routine testing programme for competitive cyclists as it is a key determinant of fractional utilisation of  $\dot{V}O_{2 \max}$ , which in turn determines performance. The findings of the study suggest that GE measurements should be taken at the same phase of the season if conducting year on year assessments.

## **CHAPTER 6: THE EFFECT OF TRAINING VOLUME AND INTENSITY ON GROSS EFFICIENCY**

### **6.1 Introduction**

Results of the previous investigation (as detailed in Chapter 5), demonstrated that changes in GE during a cycling season are related to the volume and intensity of training. However performing multiple correlations on the same data set may have increased the chances of committing a Type I statistical error. Also a study based on correlational analysis does not show direct 'cause-and-effect'. An intervention study is therefore necessary to examine the potential for the volume and intensity of training to influence GE in cycling.

The effect of endurance training on GE is unclear both from longitudinal (Gardner et al., 1989; Gissane et al., 1991; Barbeau et al., 1993; Coyle, 2005; Sassi et al., 2008) and cross-sectional studies (Marsh & Martin, 1993; Mogensen et al., 2006; Moseley et al., 2004; Nickleberry & Brookes, 1996). A training-induced change in GE has been suggested as the notable adaptation in the development of a multiple Tour de France champion (Coyle, 2005), sparking considerable interest and debate (Martin et al., 2005; Schumacher et al., 2005; Gore et al., 2008). Whilst changes in GE have been shown to affect endurance performance (Passfield & Doust, 2000) and chronic changes are known to occur during one or more cycling seasons (Barbeau et al., 1993; Sassi et al., 2008), the data discussed in Chapter 5 is the first to demonstrate that endurance training is associated

with the development of a higher GE in competitive cyclists. Since efficiency is acknowledged as a determinant of endurance cycling performance (Coyle, 1995; Jeukendrup & Martin, 2001; Passfield & Doust, 2000), research that systematically examines the effects of different training protocols on changes in GE is needed.

Historically cyclists have tended to use large volumes of training to induce an overload stimulus (Hawley & Stepto, 2001). Endurance exercise training involves adaptations that result in increases in both maximal aerobic power and the capacity for submaximal exercise (Hawley & Stepto, 2001). These studies have suggested the areas of skeletal muscle fibre, oxidative and glycolytic enzyme activity, capillary density, lean thigh volume, neuromuscular recruitment and pedalling mechanics are potential determinants of GE that could be influenced by training (Lafortune & Cavanagh, 1983; Coyle, 1995; Francescato et al., 1995; Takaishi et al., 1998; Coyle, 1999; Hawley & Stepto, 2001; Korff et al., 2007). Recently, Iaia et al. (2009) have found sub-maximal oxygen uptake at various fixed work rates to be reduced following intensive sprint interval training. However, the possibility that the impact of high intensity training on performance may be mediated by a change in GE has not been directly determined. The impact of interval training has been examined by several authors (MacDougall et al., 1998; Lindsay et al., 1996; Hawley et al., 1997; Westgrath-Taylor et al., 1997; Stepto et al., 1999; Stepto et al., 2001; Laursen et al., 2002). Typically interval training consists of a number of sustained aerobic exercise bouts lasting 3-15 min alternated with shorter rest



intervals (60-90 s) of activity at a slower pace. The objective of such training is to expose the physiological systems to sustained exercise at an intensity (or effort) which corresponds to the athlete's current highest "steady-state" or lactate threshold pace (Hawley et al., 1997). The benefits ascribed to aerobic interval training include enhanced lactate kinetics, stimulation of specific neurological patterns of muscle fibre recruitment needed at race pace, improved fatigue resistance, and enhanced athletic performance (Lindsay et al., 1996; Hawley et al., 1997; Westgarth-Taylor et al. 1997; Stepto et al., 1999).

Given the findings of the research in Chapter 5 (time spent >OBLA vs increases in GE), and those from the previous research presented above, it was felt that the impact of high intensity intervals on GE warranted further investigation. There has also been no systematic intervention study which has investigated the impact of training on GE in cycling.

## **6.2 Aim of the investigation**

The aim of the present study was to compare the effects of two different endurance training regimes on changes in GE, DE and WE in competitive cyclists.

## **6.3 Methods**

### **Participants**

A group of 29 endurance-trained competitive male cyclists were selected from local clubs and professional teams to take part in this investigation.

The cyclists' physical characteristics at the start of the study were (mean  $\pm$  SD): 35  $\pm$  8.5 yr, 72.9  $\pm$  4.6 kg,  $W_{\max}$  383  $\pm$  36 W,  $\dot{V}O_{2\max}$  60.8  $\pm$  8.3 mL $\cdot$ kg<sup>-1</sup> $\cdot$ min<sup>-1</sup>. The sample size was derived from work conducted in Chapter 3. All cyclists had a minimum of 3 years competitive cycling experience and familiarity of laboratory-based testing.

### **Procedures**

All cyclists visited the laboratory three times during the study. The cyclists' body mass and stature were measured by means of beam balance scales and stadiometer (Seca, Germany). Lower limb dimensions and skin fold thicknesses were measured prior to each cyclists test. This data enabled the calculation of total and lean leg volume according to the procedures of Winter et al. (1991).

Throughout each test, laboratory conditions were maintained in the range of 18-22°C and 45-55% humidity and cyclists were cooled with an electric fan. Cyclists were tested using an electronically braked ergometer (Lode Excalibur Sport, Lode, Groningen, NL) which was setup to replicate their normal riding position for all tests (i.e. saddle height, reach, handle bar height, crank length). The assessment consisted of a threshold test and a maximal aerobic power ramp test. Following an initial 8 minute period of unloaded cycling (for the determination of work efficiency) cyclists completed a continuous incremental exercise test. Cyclists initially selected their preferred pedal cadence which they were then required to hold constant throughout all testing sessions. Starting at 150W cyclists cycled for 4 min,

the power output was then increased to 180W for a further 4 min. From 210W onwards power output increased by 30 W every 8 min with blood lactate samples being taken every 4<sup>th</sup> and 8<sup>th</sup> minute. This continued until the measured concentration of 4 mmol·L was reached or exceeded at which point the test was terminated. Blood lactate was measured as detailed in Chapter 2. Crank torque was recorded continuously throughout the test, from which total force at the crank (sum of total forces normal and tangential to the crank arm), net force (sum of propulsive forces only) and negative force (sum of resistive forces only) were subsequently calculated (Lode Excalibur Sport, Lode, Groningen, NL). For each cyclist, crank torques were measured concomitantly with the gas collection.

Cyclists then rested for 5 minutes prior to the commencement of a ramp protocol to elicit  $\dot{V}O_{2max}$ . This protocol started at 150W using a 20W/min ramp rate which continued until volitional exhaustion. Maximal power output ( $W_{max}$ ) was calculated using the  $W_{max}$  formula (Kuipers et al., 1985) outlined in Chapter 3. All cyclists were instructed to refrain from strenuous exercise for 24 h immediately prior to each test. Throughout the study, cyclists were asked to prepare for each laboratory visit as if preparing for a race, and to follow the same training and nutritional regimes prior to each visit.

Expired gases were collected over the final three min of each 8 min bout of exercise completed (Quark b2, Cosmed, Italy) for the measurement of  $\dot{V}O_2$  and RER. The power,  $\dot{V}O_2$  and RER data collected from each 3 minute

sampling period were used to calculate GE, DE & WE using the equations outlined in Chapters 1 and 2.

*Training Period*

Cyclists were randomly divided into two groups. Group A were instructed to supplement their normal training with two specified high intensity training sessions per week. These sessions were individually set for each cyclist and are detailed in Table 15. Group B cyclists were asked to conduct all of their training under the heart rate equating to their 4mmol·L lactate point minus 5 beats.

Table 15. Summary of Group A and B training sessions during the first 6 weeks of the study. Both of Group A’s sessions were conducted each week in addition to normal base training.

	Group A (n = 16)	Group B (n = 13)
Training sessions	<p>Session 1: 3-5 x 4 minutes at OBLA HR + 5 beats with 4 minutes recovery between</p> <p>Session 2: 3 minute intervals with each interval starting with a maximal effort of 40s, the next 30s, followed by 20s and finally 10s seconds. The remainder of the 3 minutes was recovery. This is repeated 3-5 times.</p>	<p>Normal training mileage at HR = OBLA minus 5 beats.</p>

The study lasted for 12 training weeks consisting of two, 6 week training phases over the pre-season period (approximately January to April). Even though there is no data for the immediate period prior to the commencement of the study it is anticipated that cyclists were conducting predominantly low intensity training as this was the transition phase of their season. For the first 6 weeks of the study cyclists' training was controlled as outlined in Table 15. Cyclists were asked not to race and only engage in training during this period of time. During the second 6 week phase cyclists' training and racing were not controlled in either group, therefore this is not designed as a cross-over study. The second phase of training was used to see if the addition of more intensive training following restricted base training would increase efficiency during cycling in Group B. Heart rate was measured throughout every training session and race (Polar S725X, Polar, Kempele, Finland). Heart rate was sampled every 5 s and sessions were quantified as the percentage of time spent training below LT, between LT and OBLA and above OBLA.

#### *Data Analysis*

Prior to all statistical analyses data were checked for violation of the parametric assumptions. Comparisons of the changes in variables across the three tests were assessed using a two-way ANOVA (with two groups and three times) with repeated measures for time. Least significant difference unadjusted post hoc analysis (Perneger, 1998) was then conducted to identify where any significant differences within the data existed. Statistical significance was set at  $P \leq 0.05$ . All values are expressed as mean and

standard deviation (mean  $\pm$  SD) unless otherwise stated. Submaximal data were averaged to the highest common power output achieved across the three Tests.

## **6.4 Results**

### **Training Time**

Data were collected and analysed over each 6 week period between tests. Table 16 shows the mean training time in minutes conducted over the study. This data violated the assumptions for parametric assessments, consequently the non-parametric Kruskal-Wallis test was adopted. Between Tests 1 and 2 Group A spent significantly more time training at heart rates above OBLA compared to Group B ( $P=0.02$ ). There were no significant differences between the groups in time spent below LT ( $P=0.95$ ), between LT and OBLA ( $P=0.86$ ), or in total training time ( $P=0.73$ ). The time spent in the sub LT training zone was significantly lower ( $P=0.28$ ) for Group A riders between Tests 2 and 3. Overall training volume ( $P=0.18$ ) and time spent in the other training zones did not change between tests (LT-OBLA  $P=0.58$ ; above OBLA  $P=0.59$ ). Group B completed a significantly greater proportion of their training at intensities above OBLA heart rates between Tests 2 and 3 ( $P=0.03$ ). Overall training volume was not significantly different between the groups ( $P=0.80$ ).

Table 16. Mean time spent training/racing at relative exercise intensities throughout the study period.

Training Zone	Group A	Group A	Group B	Group B
	Test 1-2	Test 2-3	Test 1-2	Test 2-3
Sub LT	2138 ± 1841	1586 ± 1678	2262 ± 1836	2134 ± 1049
LT – OBLA	537 ± 377	631 ± 383	556 ± 381	670 ± 325
Above OBLA	288 ± 180	295 ± 220	136 ± 80	327 ± 286

### **Changes in Efficiency**

GE significantly changed in both groups over the study period. GE values are displayed in Table 17 and Figure 21 for both groups of cyclists. The Group A cyclists significantly improved their efficiency over the intervention period between Tests 1 and 2, both in terms of absolute and relative GE ( $P<0.01$ ). However, there were no significant changes in the GE of Group B over this training period when high intensity training was restricted ( $P=0.89$ ). Consequently, the change in GE for Group A between Test 1 and 2 was significantly greater than for Group B ( $P<0.01$ ).

Table 17. GE values across the 3 Tests in Groups A and B. \* indicates significant change in GE over baseline.

	Group A	Group B
	GE (%)	GE (%)
Test 1	19.9 ± 1.4	21.2 ± 2.1
Test 2	21.4 ± 1.4*	21.2 ± 2.5
Test 3	22.0 ± 1.7*	22.6 ± 0.8*

Subdividing the GE calculation into  $\dot{V}O_2$  and RER it is possible to identify that the significant increases between Test 1 and 2 in Group A were due to a decrease in submaximal  $\dot{V}O_2$  (-0.25L;  $P < 0.01$ ) as opposed to RER (0.02;  $P = 0.60$ ).

Between Tests 2 and 3 Group A demonstrated no significant change in GE, (21.4 vs 22.0%  $P = 0.16$ ), submaximal  $\dot{V}O_2$  (-0.07L;  $P = 0.23$ ) or RER (0.01;  $P = 0.40$ ). Group B significantly improved their absolute GE above baseline (21.2 vs 22.6%;  $P = 0.03$ ) and in relation to their changes between Test 1 and 2 (0.1 vs 7.7%;  $P = 0.04$ ). Over the total study period there was no significant difference in the changes in submaximal  $\dot{V}O_2$  (-0.22L vs -0.17L;  $P = 0.43$ ) or RER (0.02 vs 0.01;  $P = 0.91$ ) between Group A and B.



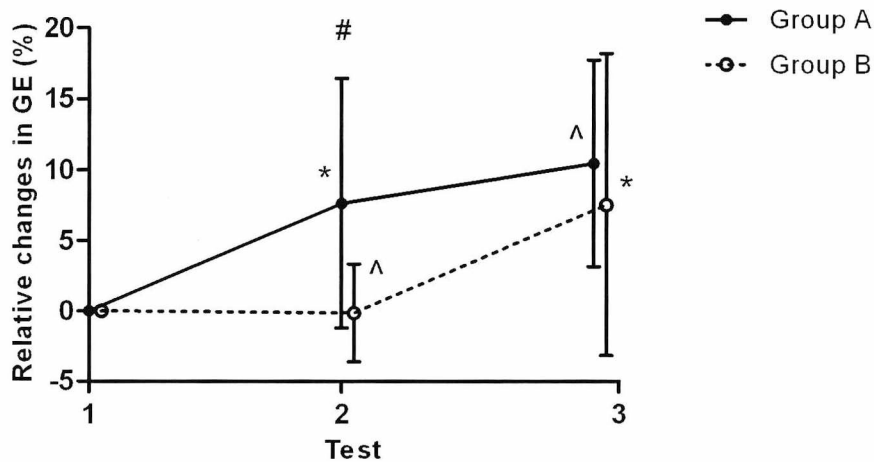


Figure 21. Relative changes in GE across the study period. \* = Significant increase above previous test ( $P < 0.05$ ); ^ = no significant change over previous test; # = Significant difference between the groups ( $P < 0.05$ ).

An interaction effect was found in WE between training group and test session ( $P = 0.04$ ). There was a significant increase in WE between Tests 1 and 2 (3.0%;  $P = 0.03$ ) in Group A (Figure 22), but not between Tests 2 and 3 (0.6%;  $P = 0.51$ ). Group B did not significantly increase their WE across the study ( $P > 0.05$ ). The oxygen cost of unloaded cycling did not significantly change across the Tests in either group (mean change = 0.08L & 0.06L;  $P > 0.05$ ).

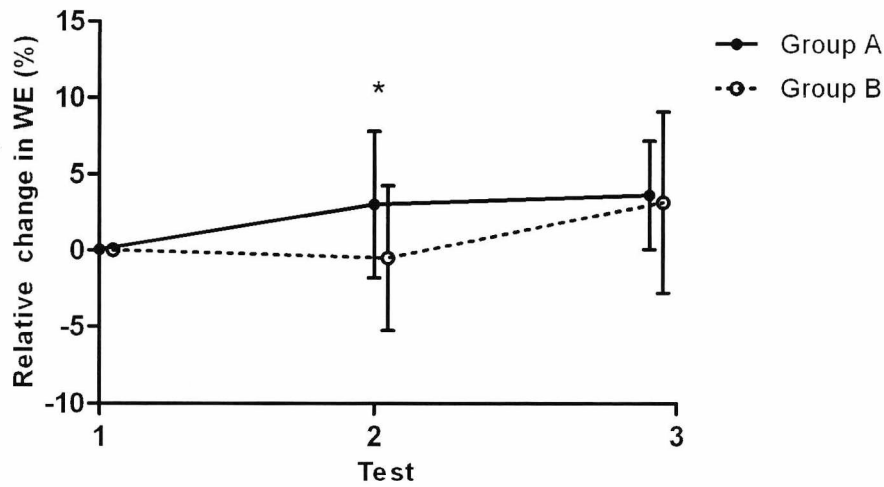


Figure 22. Relative changes in WE measured across the study period.  
Values are means + SD. \* = Significant increase in Group A above previous  
test ( $P < 0.05$ ).

DE mirrored the increases in GE over the study period (Test 1-3 Group A 5.8%; Group B 4.9%); however these changes were not significant ( $P = 0.77$ ) in either group (Figure 23).

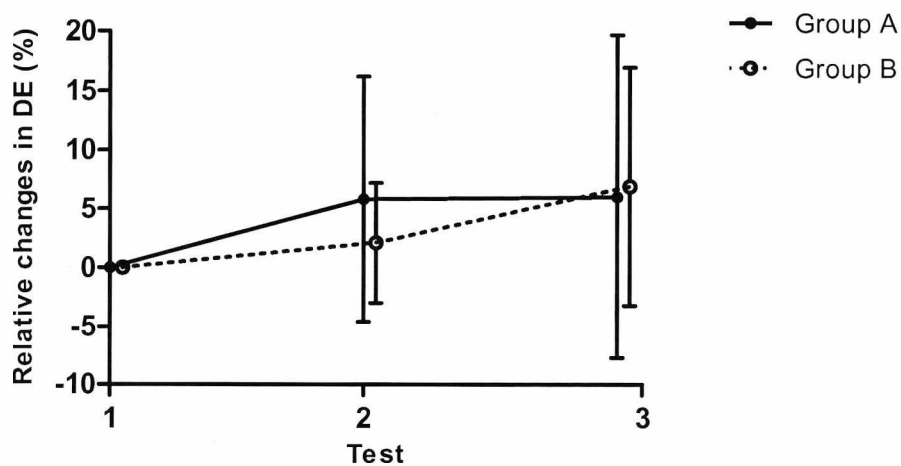


Figure 23. Relative changes in DE measured across the study period. Values  
are mean + SD.

## Changes in Crank Torque

There were no significant differences in total, net or negative crank torque over the course of the study in either group at 0 ( $P>0.05$ ), 210 ( $P>0.05$ ), or 270W ( $P>0.05$ ) as shown in Figures 24 and 25 or between groups ( $P>0.05$ ) as in Figure 26 below. However, significantly less negative work was completed by cyclists in both groups at 270W compared to 210W ( $P<0.01$ ). No significant correlations existed between GE and total, net or negative torques at 210W (total torque  $r=0.11$ ; net torque  $r=-0.17$ ; negative torque  $r=0.22$ ) or 270W (total torque  $r=-0.22$ ; net torque  $r=0.14$ ; negative torque  $r=-0.31$ ). There was also no significant correlation between  $\dot{V}O_2$  at 0W and total ( $r=0.10$ ), net ( $r=-0.22$ ) or negative torque ( $r=0.19$ ). The decreased negative crank torque between 210 and 270W could not account for the increase in GE seen between these two powers ( $R^2=0.32$ ).

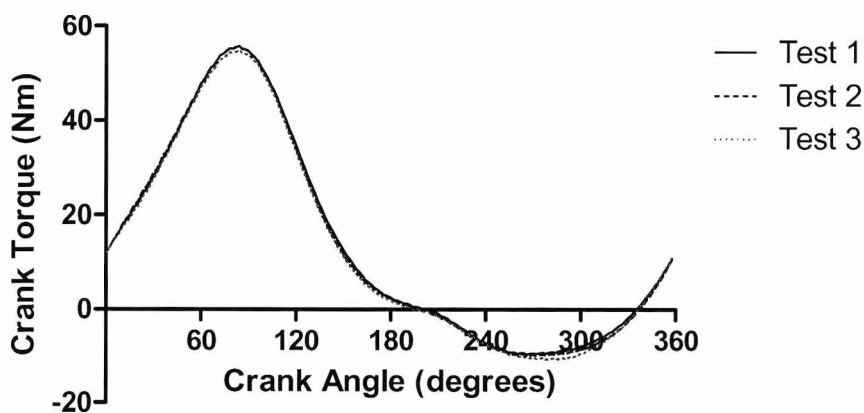


Figure 24. Mean crank torque at 210W from all participants across the three Tests.

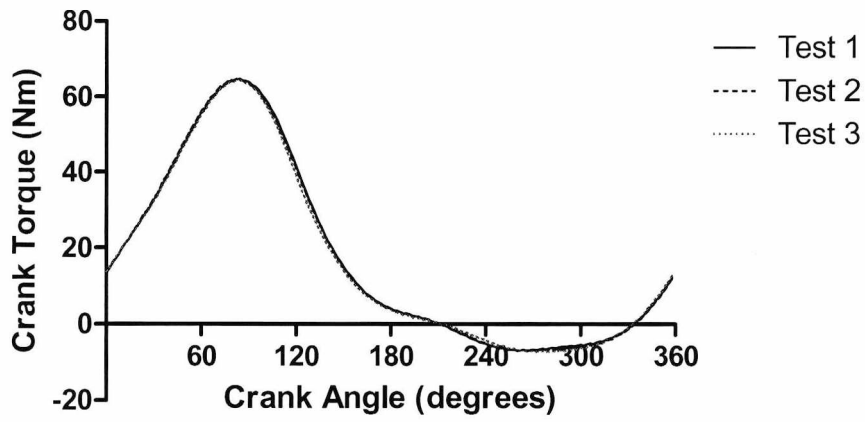


Figure 25. Mean crank torque at 270W from all participants across the three Tests.

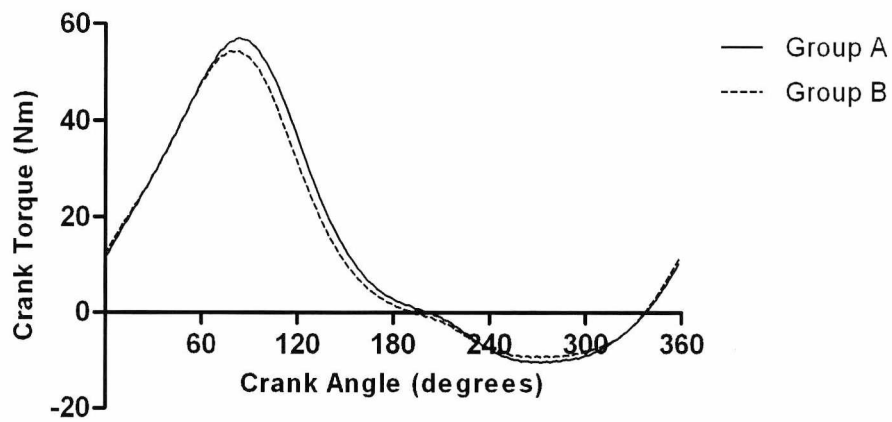


Figure 26. Mean crank torque at 210W from Group A and Group B cyclists at Test 2.

### **Changes in other variables**

Responses measured at the three tests are displayed in Table 18 below.  $W_{\max}$  significantly increased in Group A at Test 2 compared to baseline ( $P=0.01$ ) and was greater than the change in Group B ( $P=0.05$ ). Both absolute and relative  $\dot{V}O_{2\max}$  were significantly lower in Group A at Test 3 compared to Test 1 ( $P<0.01$ ). RER was significantly higher at Test 3 compared to Test 1 in Group A ( $P=0.05$ ). No other variables were different between groups or across tests.

Variable	Test 1		Test 2		Test 3	
	Group A	Group B	Group A	Group B	Group A	Group B
Mass (kg)	72.7 ± 3.9	73.1 ± 5.5	72.9 ± 4.0	73.7 ± 5.0	73.0 ± 4.4	73.2 ± 4.9
W <sub>max</sub> (W)	389 ± 34	375 ± 39	402 ± 37*	383 ± 42	400 ± 44	396 ± 31
$\dot{V}O_{2max}$ (L·min <sup>-1</sup> )	4.6 ± 0.5	4.3 ± 0.6	4.3 ± 0.5	4.3 ± 0.5	4.1 ± 0.4 <sup>^</sup>	4.2 ± 0.4
$\dot{V}O_{2max}$ (mL·kg <sup>-1</sup> ·min <sup>-1</sup> )	62.6 ± 7.6	58.6 ± 8.8	58.5 ± 6.4	59.0 ± 8.5	56.7 ± 5.2 <sup>^</sup>	57.4 ± 5.5
LT (W)	233 ± 25	231 ± 28	234 ± 27	228 ± 34	229 ± 29	230 ± 39
OBLA (W)	271 ± 33	275 ± 38	280 ± 39	276 ± 33	274 ± 39	278 ± 44
HR <sub>max</sub> (b·min <sup>-1</sup> )	186 ± 10	184 ± 8	185 ± 10	185 ± 8	184 ± 9	185 ± 11
VE (L·min <sup>-1</sup> )	88.5 ± 8.9	85.0 ± 8.4	85.0 ± 9.0	82.5 ± 10.4	83.6 ± 12.0	82.9 ± 5.8
Leg Volume (dm <sup>3</sup> )	8.5 ± 1.4	8.9 ± 1.5	8.6 ± 1.3	8.0 ± 2.4	8.5 ± 1.2	8.8 ± 1.2
Lean Leg Volume (dm <sup>3</sup> )	5.7 ± 0.9	5.9 ± 1.0	5.7 ± 8.6	5.9 ± 0.9	5.7 ± 0.8	5.8 ± 0.8

\*Significantly higher than baseline ( $P < 0.05$ ). <sup>^</sup>Significantly lower than baseline ( $P < 0.05$ ). Values are means + SD.

Table 18. Performance related responses measured at each test across Groups A and B.

## **6.5 Discussion**

### **General overview of findings**

The main finding of this study is that the addition of endurance training at intensities above OBLA results in an increase in GE. Group A significantly enhanced their GE during the high intensity training intervention period between Tests 1 and 2. This change was not observed in Group B who were restricted from such training over the same study period. During this period training volumes were not significantly different between groups A and B, but a greater training time spent at heart rates above OBLA was found for Group A. The conclusion that high intensity training was responsible for the increase in GE is further reinforced by the observation that an increase in GE was also measured in Group B once high intensity training was allowed. These results confirm and extend the results discussed in Chapter 5.

All indices of efficiency responded in a similar fashion, although not all results were significant. It is interesting to note that there were similar mean changes in DE (5.8% Group A; 4.9% Group B; see Figure 23). Although, given the poor reliability reported in Chapter 3 and by Moseley and Jeukendrup (2001), as well as the large variability observed in both groups (SD 2.9-7.4%), a non-significant finding is unsurprising ( $P=0.77$ ). WE increased by a slightly smaller amount (3.6% Group A; 3.1% Group B; see Figure 22), with increases in Group A being significant between Tests 1 and 2.

Using reliability data from Chapter 3 of this thesis it is possible to further highlight the impact this variability could have on the results of the study. If DE were used as the primary outcome variable 87 participants would have been required in each group to conclude no significant changes.

### **Changes in efficiency**

GE increased from 19.9% to 21.4% (1.5% absolute; 7.6% relative) in Group A over the initial 6 week period training between Tests 1 and 2. To the authors knowledge this is the first study to demonstrate that GE can be increased by 6 weeks' of training and to establish a relationship between introducing high intensity training and changes in GE. Cross-sectional studies have previously demonstrated similar absolute differences in GE of 1.2% between professional and elite cyclists (Lucia et al., 1998); and 1.4% between trained and untrained cyclists as illustrated in Chapter 4, but longitudinal studies are limited. However, the findings of Chapter 5 demonstrate increases in GE of 1% occur during the competitive phase of a cycling season that is then reversed in the off-season. Also, although controversial, Coyle (2005) reports a 1.9% improvement in GE over a 7 year period in a Grand Tour Champion. The interpretation of Coyle's data has been questioned by several authors (Martin et al., 2005; Schumacher et al., 2005; Gore et al., 2008).

The data indicates that the increase in Group A's GE of 1.5% equates to a ~24 W increase in power output able to be sustained during a 1 hour performance test. This is comparable to the findings of Horowitz et al.,



(1994) who reported a 1.8% higher GE would result in a 27W increase in 1 hour time-trial performance power output. Data from Moseley and Jeukendrup (2001) indicates that the reported change would also lead to ~88s improvement in 40km time-trial performance. Interestingly, these postulated improvements in time-trial time and performance power are consistent with those previously reported for interval training. For example, Westgarth-Taylor et al. (1997) found a 90 second improvement in 40km time-trial performance following 6 weeks of interval training. Similarly, Stepto et al. (1999) found that following high-intensity interval training (85% peak power) cyclists increased both absolute and relative sustained exercise intensity for a 40 km time-trial, resulting in a ~100s improvement. They suggest absolute intensity improved due to increases in  $W_{\text{peak}}$  as a result of the intervals, although the mechanisms for increases in relative exercise intensity are not so clear cut. It is possible that increases in efficiency could have allowed the cyclists to sustain a higher fraction of  $\dot{V}O_{2\text{max}}$  following training. Whether the changes seen in the current study would actually result in an improvement in time-trial performance remains to be established. Future research could include a time-trial type performance element to identify the impact changes in GE may have in a cyclist's performance.

Analysis of the training data suggests that supra-OBLA intensity training was the key stimulus for improving GE in cycling. Supra-OBLA interval training has been shown to decrease the oxygen cost of cycling (Barnett et al., 2004; Burgomaster et al., 2005; 2008) in recreationally active

participants. Burgomaster et al. (2005) have also demonstrated that sprint interval training (30s sprints, 4-8 x per session for 2 weeks) can double the length of time that intensive aerobic exercise can be maintained (26-51 min).

It is worth noting in the present study that Group A did not improve GE between Tests 2 and 3. This may be the result of removing the prescribed supra-OBLA interval sessions. The cyclists may have lacked structure and/or progressive overload in their training as total volume and time spent >OBLA were similar to that between Tests 1 and 2. Alternatively these cyclists may have been approaching the limit for such training-induced changes in GE. Therefore, if continued progressive overload were present no changes may have occurred anyway.

Absolute responses of WE and DE were comparable with those of GE, although generally not significant. WE increased significantly in Group A (3.0%) but not Group B (-0.5%) between Tests 1 and 2. As with measurements of GE, WE remained statistically unchanged between Tests 2 and 3 in Group A (+0.6%). However Group B improved their WE (+3.7%), albeit not significantly. It is speculated that this non-significant finding was again due to the large SD of the measurement (~6%). The WE calculation has previously been criticized because the cost of moving the legs has been shown to be dependent on the external workload (Kautz & Neptune, 2002). Therefore, the measurement of O<sub>2</sub> cost, VE and calculated RER during unloaded pedalling may not reflect a similar proportion of the total energetic

cost incurred at higher power outputs. The measurement of the O<sub>2</sub> cost of unloaded pedalling and the WE calculation is therefore problematic.

Changes in leg mass have been associated with a change cycling economy (Francescato et al., 1995). Indeed these authors suggest that the cost of moving the legs is a key determinant of efficiency in cycling. Leg volume, lean leg volume and body mass did not change during the current study period. Thus it seems that these variables were not responsible for the changes found in GE or WE.

#### **Possible mechanisms for changes in GE**

The current study demonstrates improvements in GE with high intensity interval training. This type of training has previously been shown to decrease the oxygen cost of cycling (Barnett et al., 2004; Burgomaster et al., 2005; 2006; 2008) in recreationally active participants. Indeed, Burgomaster et al. (2005) demonstrate that sprint interval training (30s sprints, 2-4 minutes per session for 2 weeks) can double the length of time that intensive aerobic exercise can be maintained (26-51 minutes). This is even more interesting given that peak oxygen uptake was not increased, although aerobic adaptations did occur within the active skeletal muscles. This was represented by a 38% increase in activity of the mitochondrial enzyme citrate synthase. From the perspective of biochemistry it has long been recognized that sprint interval training can increase aerobic enzyme activity in muscle with 6-8 weeks (Henriksson & Reitman, 1977; Dudley et al., 1982; Rodas et al., 2000). It is likely that the potency of high intensity

training comes from its' ability to cause a high level of motor unit recruitment. Consequently Type IIa muscle fibres that are equally as responsive as Type I fibres in their ability to increase mitochondrial enzyme activity will be recruited, stressed and stimulate to adapt to the demands imposed (Jansson & Kaijser, 1987). As efficiency in cycling has been linked to muscle fibre type (Coyle et al., 1992; Horowitz et al., 1994; Mogensen et al., 2007) it could be speculated that this increased aerobic enzyme activity may, in part, be a potential adaptive mechanism for the changes seen in GE during this study. It has been shown that this mitochondrial activity has the potential to be increased to equally high levels in both fast and slow twitch fibres following high intensity training (Chi et al., 1993). The low intensity exercise (as undertaken by Group B between Tests 1 and 2) may be less effective at increasing aerobic enzyme activity in Type IIa fibres.

Daussin et al. (2008) demonstrate that interval training is capable of stimulating improvements in skeletal muscle mitochondrial function in untrained individuals. Conversely they found no changes in mitochondrial function when the group trained at 60% of maximum power output. This suggests that the stimulus of 60% maximum power output is not sufficient to induce adaptations even in sedentary individuals. High intensity interval training therefore appears to cause a higher muscle oxidative potential after training. Daussin et al. (2008) matched mechanical work and training session duration between their interval and continuous training regimes. This suggests that the fluctuations of oxygen uptake and energy turnover caused by interval training are a major factor influencing mitochondrial

adaptations. Interestingly Daussin et al. (2008) also found a correlation between gain in mitochondrial function and increase in time to exhaustion at a pre-training set power output. This may be suggestive of an improvement in economy/efficiency.

Iaia et al. (2009) demonstrated a reduction in the oxygen cost at a range of fixed intensity work rates following sprint interval training. They speculate that this was due to changes in the working muscle's oxidative capacity and metabolic processes. Burgomaster et al. (2008) suggest that the production of adenosine monophosphate activated protein kinase released as a result of intensive training (Gibala et al., 2006; Koulmann & Bigard, 2006), may cause the up-regulation of the transcriptional coactivator peroxisome proliferator-activated receptor  $\gamma$  coactivator 1  $\alpha$  (PGC-1 $\alpha$ ) which in turn regulates mitochondrial biogenesis in Type I, IIa and IIx fibres (Russell et al., 2003; Taylor et al., 2005; Terade et al., 2005). Not only does PGC-1  $\alpha$  activate mitochondrial biogenesis in the skeletal muscle, but it also plays a role in gene encoding of myofibrillar proteins characteristics of oxidative fibres. Mice transgenically expressing PGC-1  $\alpha$  have been shown to increase the proportion of fibres expressing MHC I and MHC IIa characteristics (Lin et al., 2002). Burgomaster and coworkers found significant increases in PGC-1  $\alpha$  following training in both sprint interval training and traditional endurance training groups, but the effect was greater in the interval group. Thus there appears to be some evidence in the findings of Burgomaster et al. (2008) that might indicate processes are occurring

within the skeletal muscle which may ultimately result fibre type transformation towards MHC I and IIa.

Fibre type transformation has been shown in some fast myosin of endurance trained muscle, with shifts towards a more efficient isoform (Green et al., 1984). However it is unclear as to whether these adaptations would be seen within the 6 weeks of the current study. The majority of research suggests 8-12 weeks or longer is necessary to induce fibre type transformation (Gollnick et al., 1973; Costill et al., 1979; Baumann et al., 1987; Windisch et al., 1998; Williamson et al., 2001). It is therefore unclear whether the changes seen in GE in the current study were mediated by fibre type transformation. In contrast, it has been demonstrated that mitochondrial adaptations linked to increased oxidative potential of a muscle are evident within 10 days of starting endurance training (Spina et al., 1996). However participants were untrained, whether similar levels of adaptation are likely in trained cyclists is unclear. Nevertheless adaptations in these mechanisms are much quicker than those of muscle fibre transformation and thus have a greater potential to influence the changes in GE within the time scale of the current study.

Future research should look to advance the current knowledge on muscle fibre type and efficiency in cycling. The research of Coyle et al. (1992), Horowitz et al. (1994) and Mogensen et al. (2006) are based on correlational evidence between efficiency and % slow twitch fibres. Unfortunately this work has not been taken further in trying to establish a 'cause-and-effect'

relationship between fibre type and efficiency. If muscle fibre biopsies were taken pre and post a training intervention it would be possible to directly identify if changes in muscle fibre type and/or mitochondrial/enzymatic adaptations occur as a result of interval type training. It would then be possible to identify if these adaptations had an effect on efficiency measured via gases produced at the mouth.

### **Crank torque**

Total, net and negative crank torques did not significantly change over the study period. Although, significantly less negative work was conducted when cyclists were cycling at 210W compared to 270W. This did not explain changes in GE between these two power outputs.

There was no association between crank torques and GE, WE or DE. This is not unexpected as torque at the crank does not necessarily reflect work done by the active muscles (Kautz & Neptune, 2002). However, Mornieux et al. (2006) established that inter-individual differences in force during the downstroke phase of the crank cycle were related to variations in net efficiency. No relationships were found with GE. The usage of net efficiency to establish efficiencies whilst cycling is problematic. Net efficiency suffers from the same criticisms levelled at WE due to the baseline subtraction of resting  $\dot{V}O_2$  (Stainsby et al., 1980). Zameziati et al. (2006) have also reported a relationship between pedalling effectiveness and GE/net efficiency ( $r=0.66$  and  $0.54$  respectively). Using instrumented pedals they found that upstroke torque could explain small variations in efficiency

measured at the mouth. However Zameziati et al. (2006) used untrained cyclists who are likely to possess large variations in the effectiveness of their pedalling action and GE. Thus finding a correlation in this heterogeneous group of cyclists is more likely than with the current study's homogenous group of trained cyclists.

### **Changes in other variables**

Cyclists'  $W_{\max}$  increased in Group A as a result of the interval training programme. This finding is consistent with previous research on the effects of interval training in well-trained endurance cyclists (Hawley et al., 1997; Westgarth-Taylor et al., 1997; Stepto et al., 1998; Laursen et al., 2002). Although in contrast to Laursen et al. (2002)  $\dot{V}O_{2\max}$  and blood lactate responses did not increase following interval training. Therefore adaptations which influence GE appear not to cause corresponding increases in  $\dot{V}O_{2\max}$  or exercising blood lactate response.

To further examine the factors that may result in the changes seen in GE, cyclists were ranked based on their observed change in efficiency across Test 1-3 and divided them into equal high and low change groups. Using an independent T-test the high change group was found to have a significantly lower initial GE at Test 1 compared with the low change group (GE 20.0 vs 21.8%;  $P < 0.01$ ). These cyclists also had a significantly higher  $\dot{V}O_{2\max}$  at Test 1 ( $\dot{V}O_{2\max}$  4.6 vs 4.0 L·min<sup>-1</sup>;  $P < 0.01$ ). The high change GE group recorded a decrease in the  $\dot{V}O_{2\max}$  compared with the lower GE change group ( $\dot{V}O_{2\max}$  -0.44 vs 0.1 L·min<sup>-1</sup>; -5.9 vs 0.8 mL·kg<sup>-1</sup>·min<sup>-1</sup>  $P < 0.01$ ) over



the study period. To ensure a valid data set prior to the above analyses, three cyclists' were excluded as their data were not entirely consistent with the criteria for achieving  $\dot{V}O_{2\max}$  (a last minute rise of  $\leq 150\text{ml/min}$ ), as suggested by Midgeley et al. (2007). Recently Iaia et al., (2009) also report a decrease in  $\dot{V}O_{2\max}$  following a period of intensive endurance training (mean  $\dot{V}O_{2\max}$  54.8 vs 53.5  $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$  before and after 4 weeks of intensive interval training respectively), in previously endurance trained runners. Interestingly they also report significant increases in running economy and decreases in the submaximal energy cost of running at a range of running velocities. Unfortunately no relationships between  $\dot{V}O_{2\max}$  and running economy are presented in their results.

Using data from both Group A and B across the time course of the study significant inverse relationships (Figures 27a-d) were found between both absolute and relative changes in GE and  $\dot{V}O_{2\max}$  using Pearson's Correlation Coefficient. It appears that those cyclists with the largest improvement in GE experience the largest decrease in  $\dot{V}O_{2\max}$  (see Figures 27a-d. Data are presented over the specific intervention period [27a&c] and then over the whole 12 weeks of the study [27b&d]). Interestingly Group B did not demonstrate an inverse relationship between changes in GE and  $\dot{V}O_{2\max}$  when their training intensity was restricted between Tests 1 and 2 (Figure 27c).

Interpreting the current data with the findings of Hunter et al. (2005) it could be speculated that these changes may be due to adaptation within the

muscle cell. They have demonstrated that a high  $\dot{V}O_{2\max}$  is more strongly related to a high proportion of Type IIa fibres than Type I. Conversely exercise economy/efficiency is more strongly related to a high proportion of Type I fibres (Coyle et al., 1992; Horowitz et al., 1994; Mogensen et al., 2006). Thus over the 12 week training period of the current study it is possible that cyclists with an initially high proportion of Type II fibres experienced some form of transformation towards Type I, thereby increasing their GE and decreasing  $\dot{V}O_{2\max}$ . Those cyclists initially high in Type I fibres, also having a high GE at Test 1, subsequently experienced minimal fibre type transformation and so their  $\dot{V}O_{2\max}$  remained relatively stable. Whether these changes are due to fibre type transformation is speculative as no biopsies were taken in the current study. It would also be expected that these adaptations are temporary and linked to the stimulus of training undertaken as the findings of Chapter 5 have previously shown changes in GE to be reversible over the course of a cycling season.

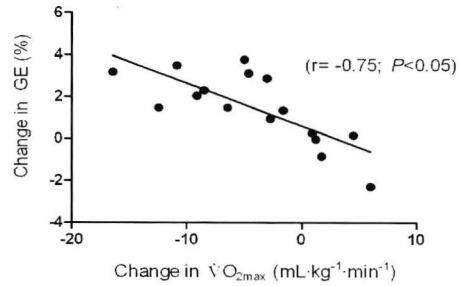


Figure a

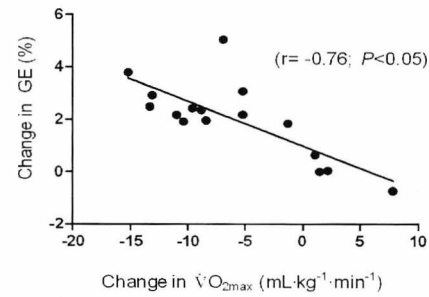


Figure b

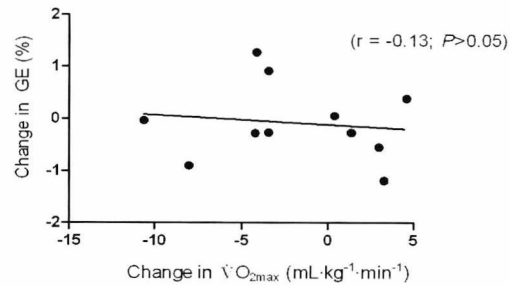


Figure c

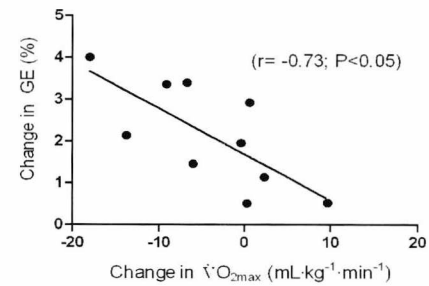


Figure d

Figure 27. Inverse correlations between changes in  $\dot{V}O_{2max}$  and changes in GE over 12 weeks of training in Groups A and B. Figure 27a Relationship between change in  $\dot{V}O_{2max}$  and change in GE in Group A Test 1-2. Figure 27b Relationship between change in  $\dot{V}O_{2max}$  and change in GE in Group A Test 1-3. Figure 27c Relationship between change in  $\dot{V}O_{2max}$  and change in GE in Group B Test 1-2. Figure 27d Relationship between change in  $\dot{V}O_{2max}$  and change in GE in Group B Test 1-3. 3 cyclists were excluded for not achieving a plateau in the VO<sub>2</sub> response during the last minute of the test.

The impact on performance of the training-induced changes in the present study warrants further investigation as a consequence of the inverse relationship shown above. These findings are similar to the findings of Lucia et al. (2002) who identified an inverse correlation between  $\dot{V}O_{2\max}$  and economy. They suggested GE increased to compensate for cyclists who have a low  $\dot{V}O_{2\max}$ . The current results extend those of Lucia et al. by suggesting that the inverse relationship they found may occur as a result of training. However, Atkinson et al. (2003b) have suggested that a spurious correlation could account for much of the relationship found, but cannot explain the relationship observed in the current study. As outlined in Chapter 5 this is not the case, as the same factor is not used in the calculation of both correlated variables. This relationship is also not evident due to gas measurement error as analyser was rigorously checked prior to each test. Similarly there is evidence that equipment drift (causing artificially low  $\dot{V}O_2$  values) has not occurred. If this were the case then both groups would have increased GE at all Tests.

### **Conclusion**

In conclusion, this study demonstrates that GE during cycling is increased in 6 weeks by the introduction of high intensity training. Training-induced changes in GE were found to be inversely related to changes in  $\dot{V}O_{2\max}$ . Competitive cyclists looking to enhance their GE should look to incorporate 6 weeks of high intensity training prior to the main competitive phase of their season. However, the underpinning mechanisms, and performance implications of these findings remain to be determined.

## **CHAPTER 7: GENERAL DISCUSSION AND CONCLUSIONS**

### **7.1 Executive summary of findings**

This thesis set out to investigate efficiency in competitive cycling. There are three main findings; 1. It is now possible to measure small changes in GE using a long stage duration, preferred cadence protocol; 2. Gross efficiency in cycling is affected by the volume and intensity of training undertaken over the course of a cycling season; 3. Gross efficiency is specifically increased as a result of high intensity training. The aim of this chapter is to summarise the findings of the work and to conclude on the data collected.

The four studies that provide the focus of this thesis allow for further insight into exercise efficiency in terms of current method, tracking longitudinal changes and the effects of training. It has been shown that GE can be reliably calculated using a long stage duration protocol to ensure steady state of  $\dot{V}O_2$  and  $\dot{V}CO_2$  responses. The results of the study in Chapter 3 showed that this protocol provided calculations of efficiency in close agreement with those previously reported in the literature (17-23%) (Coyle et al., 1992; Horowitz et al., 1994; Moseley & Jeukendrup, 2001; Moseley et al., 2004). Reliability coefficients obtained from repeated testing using the protocol also provided good agreement with those published by Moseley and Jeukendrup (2001). GE was shown to be more reliable than DE (CV = 4.5 vs 10.9% respectively) and was therefore used as the primary outcome variable. As pedalling mechanics have been suggested to influence efficiency during cycling (Coyle et al., 1998; Patterson & Moreno, 1990;

Mornieux et al., 2006; Zameziati et al., 2006) it was also decided to establish the reliability of the measure of crank torque. CTE was found to be a reliable measurement (CV = 1.2-1.4%), however no relationship was identified between CTE and GE ( $P>0.05$ ).

Having established a reliable protocol, the study in Chapter 4 sought to re-examine the research investigating differences between trained and recreational cyclists. The aim of conducting this cross-sectional work again was to broadly examine the effects of training on GE, using a reliable protocol and more powerful method than previously published work. The results of the study showed that differences did exist between trained and recreational cyclists at both the same absolute and relative power outputs. A 1% difference between the groups was established at an absolute power output of 150W. The differences increased to 1.7% at the relative intensity of  $60\%W_{\max}$ . No significant differences in crank torque effectiveness were found between trained and recreational cyclists at relative power outputs. However at an absolute power output of 150W recreational cyclists had a significantly more effective crank torque profile than the trained group. This is largely attributed to a work rate of 150W representing a higher relative exercise intensity in the recreational group. Thus the recreational cyclists were forced cycle with a greater optimisation of their pedalling strategy in comparison to the trained group.

As this study was cross-sectional in nature it could not be established whether the differences found in GE were due to differences in pedal rate,

training, genetic predisposition or natural selection processes in the trained group. Therefore the next stage in the research process was to establish if GE changed with training.

Chapter 5 documents the results of a longitudinal study designed to establish whether well-trained cyclists' GE changed over the course of a training/competitive year. GE showed the biggest increase at the end of the pre-season period (+1%). Having recorded each cyclist's training data it was possible to establish that increases in GE were related to the total training volume, as well as time spent at heart rates >OBLA. GE was then maintained by the cyclists over the competition phase of the season before decreasing significantly back to baseline level over the reduced training transition phase of the season. Crank Torque (total, net and negative torques) remained unchanged over the season.

As a result of these findings an intervention training study was implemented to further explore the influence of training on GE. The findings of Chapter 6 document that training at higher exercise intensities (>OBLA) causes significant increases in GE. However, training at low to moderate intensities has no effect on GE. This was demonstrated by Group A's significant increase in GE over the first half of the study (with specified high intensity sessions) and Group B's over the second half once training restrictions were removed. Interestingly Group A did not significantly increase their GE over this period (Tests 2-3) even though they completed similar amounts and proportions of training as they did between Tests 1 and 2. This suggests that

perhaps a key stimulus for adaptation linked to increases in GE is the change of training intensity, i.e. addition of the high intensity training. Another interesting and somewhat unexpected finding of this thesis was an inverse relationship between changes in GE and changes in  $\dot{V}O_{2\max}$ . Therefore at the same time as being a key stimulus for increasing GE, high intensity training appears to depress  $\dot{V}O_{2\max}$ . Conversely low to moderate intensity training enables maintenance of  $\dot{V}O_{2\max}$ , but affords no benefits for GE.

## **7.2 Contextualising changes in cycling efficiency**

There is an abundance of evidence within the literature that efficiency in cycling can be acutely changed via; altered cadence (Croisant & Boileau, 1984; Merrill & White, 1984; Coast et al., 1986; Patterson & Moreno, 1990; Nickleberry & Brooks, 1996; Marsh & Martin, 1997; Chavarren & Calbet, 1999; Samozino et al., 2006), bicycle set-up (Nordeen-Snyder, 1977; Heil et al., 1995; Price & Donne, 1997), muscle recruitment patterns (Cannon et al., 2007; Korff et al., 2007), pedalling mechanics (Patterson & Moreno, 1990; Coyle et al., 1998; Mornieux et al., 2006; Zameziati et al., 2006), leg mass changes (Francescato et al., 1995) and fatigue (Passfield & Doust, 2000). The magnitude of these acute changes ranges from 0 – ~2%. A confounding issue within much of this literature is that there are a number of different methods of efficiency calculations evident, each with its own relative criticisms (cf. Stainsby et al., 1980). In addition only one study has specifically investigated the reliability of any of these calculations (Moseley & Jeukendrup, 2001). The findings of past research should therefore be



viewed with these limitations in mind. Table 19 below contrasts the magnitude of changes in GE seen in Chapter 6 against some of the acute efficiency interventions documented in the literature. It is interesting to note that high intensity training is more effective at increasing GE during cycling than other forms of acute interventions.

The majority of previous literature published on efficiency in cycling has suggested that chronic changes as a result of training are not possible (Boning et al., 1984; Nickleberry & Brooks 1996; Marsh, et al., 2000; Moseley et al., 2004; Mogensen et al., 2006). There are only three previous studies in the literature which have demonstrated training related changes in efficiency with competitive cyclists (Barbeau et al., 1993; Coyle 2005; Sassi et al., 2008). Although statistically significant training mediated increases in GE have only been demonstrated by one of these studies. Barbeau et al. (1993) found a significant increase in GE at 250W over the pre-season period (Nov – Feb). GE then continued to increase up until the end of the season. Unfortunately, they do not provide numerical data to support this and so it is difficult to interpret the magnitude of changes in comparison to those of the current study. It is similarly disappointing that training was not closely monitored and so the significant increases in GE over the year could not be linked to a particular type of training and/or racing. These criticisms could also be levelled at the study of Sassi et al. (2008) who demonstrated a 1.1% increase in GE over a 6 month period. This increase proved to be non-significant, possibly due to a lack of statistical power within their method and the protocol implemented. Similar to the findings of the study in

Chapter 5 cyclists increased their GE over the pre-season and competitive phases. Although as no training data is presented it is not possible to identify whether this was an influencing factor in their GE results.

The mean increase of 1.5% found in the study in Chapter 6 is lower than that documented by Coyle (2005). However within the results of the Chapter 6 intervention study three cyclists did improve their GE by over 3%. Thus the findings of Coyle (2005) could be explained by short-term training adaptations and not long-term maturation as he suggests. It is unfortunate that like Barbeau et al. (1993) and Sassi et al. (2008), Coyle does not provide any training data that may be considered when interpreting his results (although publication of this data may have been guarded due to the professional rider(s) that were participants). In contrast to the short work-stage durations (2-5mins) of Barbeau et al., (1993) and Coyle (2005), the results of this thesis were achieved using a long work-stage method of determining cycling efficiency that has been shown to produce reliable steady state  $\dot{V}O_2$  and  $\dot{V}CO_2$  data. Sassi et al. (2008) did use a long duration test to measure GE, however this was in the form of a time to exhaustion trial at 78% of peak power output. Although as a result of using this type of approach it is possible that on re-testing the participants after a period of training, this power output may have decreased to a relative 71-73% of peak power output. This may therefore account for the improvements they found in GE.

Table 19. Interventions shown to result in changes in efficiency during cycling. Magnitude of changes shown in comparison to findings of Chapter 6 in this thesis. \* = acute changes seen in a single testing session rather than chronic adaptation resulting from training.

Authors	Study	Magnitude of efficiency change	Intervention
Current study	Interval training	+1.4% GE	Pre to post 6 weeks of training
Korff et al. (2007)*	Peddalling technique	-1.6% GE	Comparison of normal pedalling action and pulling up action on the upstroke to minimize negative torque
Cannon et al. (2007)*	Peddalling technique	+0.5% GE	Comparison of pedalling with dorsiflexed ankle position to normal action
Morineux et al. (2008)*	Pedal type	+0.5% Net Efficiency	Comparison between normal pedal and clipless pedal
Price & Donne (1997)*	Seat tube height	+1.1% GE	Cycling with seat tube height of 96 compared to 104% trochanteric height.
Berry et al. (1993)	Body mass	<1% GE	10 kg change in body mass
Francescato et al. (1995)*	Leg mass changes	+1% GE	Decreasing leg mass by 40%
Grappe et al. (1998)*	Body position	+0.1% GE	Cycling in an aero position compared to upright posture

Passfield and Doust (2000)*	Fatigue	-0.9% GE	Following 75mins cycling at 60% $\dot{V}O_{2max}$ .
Lucia et al. (2004b)*	Rotor Crank system	+0.3% GE	Using the Rotor Crank System compared to conventional crank system.

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### **7.3 Possible explanations for the discrepancies of findings related to training and efficiency from the current and previous studies**

Training has been shown to cause muscle fibre type transformations (Simoneau et al., 1985; Howard, 1985; Westgaard & Lomo, 1988; Tipton, 1996; Gundersen, 1998; Windisch et al., 1998), aerobic enzyme capacity (Henriksson & Reithman, 1977; Dudley et al., 1982; Rodas et al., 2000; Burgomaster et al., 2005; Burgomaster et al., 2008), mitochondrial adaptations (Chi et al., 1993; Russell et al., 2003; Taylor et al., 2005; Terade et al., 2005; Burgomaster et al., 2008; Daussin et al., 2008) and muscle recruitment patterns (Brown et al., 1996; Takaishi et al., 1998; Neptune & Herzog, 2000). Consequently it would be plausible that training should influence GE. The fact that the majority of research investigating the effect of training on GE has failed to find a significant effect indicates potential design and method problems.

The measurement of  $\dot{V} O_2$  and  $\dot{V} CO_2$  at the mouth and subsequent calculation of efficiency is a somewhat “noisy” measure (CV = 4.5%). Statistical power analysis is necessary prior to data collection to enable the optimal sample size to be estimated (Batterman & Atkinson, 2005). Based on reliability data from Chapter 3 it was identified that 14 participants were required to identify a main effect of differences in efficiency at  $P < 0.05$  with an effect size of 0.98. Therefore as none of the previous efficiency research investigating the effect of training on efficiency had investigated the reliability of the measurement prior to data collection, the majority did not recruit enough cyclists to identify an effect (Boning et al. 1984; Nickleberry

& Brooks, 1996; Marsh et al., 2000; Sassi et al., 2008). It is therefore unsurprising that the results of this thesis contradict those previously published.

Whipp (1987) and Wasserman et al. (2005) have both stated that at least 3 minutes are required to obtain steady state O<sub>2</sub> with a considerably longer time required for stable CO<sub>2</sub> measurements (~5 minutes). There are a wide range of protocols evident in the past literature for the collection of gases to enable subsequent efficiency calculations. Unfortunately many protocols of past research did not allow for steady state conditions prior to sampling (Boning et al., 1984; Nickleberry & Brooks, 1996; Marsh et al., 2000; Moseley et al., 2004). If steady state conditions are not ensured then it could be argued that efficiency calculations are invalid. The long work-stage durations of the protocols used within this thesis ensured for steady state conditions to be encountered prior to any gas collection. This may account for some of the different conclusions drawn between this thesis and previous work.

#### **7.4 The importance of high intensity training for increasing gross efficiency**

The study documented in Chapter 6 found no significant difference in the magnitude of GE gain between Groups A and B at the end of the 12 weeks of training. However over the first 6 weeks of training Group A significantly increased their GE with the addition of high intensity training. Group B were restricted to train below their OBLA heart rate and consequently did

not improve their efficiency. This suggests the intensity of cyclist's training is an important factor for increasing GE. However, there is likely to be a threshold of intensity below which limited adaptations will occur (as shown by Group B), and a ceiling to the magnitude of adaptation that is likely (as suggested by the limited increase in Group A Test 2-3).

Group B conducted their training below OBLA heart rate between Tests 1 and 2. With the addition of high intensity training (Test 2-3) they significantly increased their GE. Therefore a key stimulus for increasing GE could be the addition of more intensive interval training rather than the intensity of training itself. It could be assumed that Group A were conducting primarily low intensity training (below OBLA intensity) over the off-season (Nov & Dec) prior to the commencement of the study (although there is no data to support this assertion). The addition of intensive training in January subsequently improved their GE. This is also seen in the research study in Chapter 5, as cyclists increased intensity of their training over the pre-season period efficiency increased. GE was then maintained by the intensity of the training and racing during the competitive phase of the season. Future research should therefore investigate strategies to optimise training for maximum efficiency gains. Similarly it is important that future research investigates how to maintain efficiency during the transition phase of a cyclist's year so that the observed decreases in Chapter 5 are attenuated.

The results of this thesis also suggest that there might be a finite limit to the magnitude of efficiency gain in trained cyclists. It is interesting to consider why, in Chapter 6, Group A failed to continue to increase their GE during the second 6 weeks of training even though the time spent training at heart rates above OBLA was statistically unchanged. Maintaining continued progressive overload with high intensity training is difficult, but further research could look to identify if continued progressive overload is capable of producing continued increases in GE. Are cyclists able to continue increasing their efficiency until they reach the theoretical limit of efficiency (Whipp & Wasserman, 1969)?

### **7.5 Possible mechanisms associated with high intensity training-induced increases in GE**

Of five previous studies (Gardner et al., 1989; Gissane et al., 1991; Barbeau et al., 1993; Coyle, 2005; Sassi et al., 2008) to investigate the influence of training on exercise efficiency, all have demonstrated training related increases. The results of this thesis support the fact that training has a positive effect of increasing GE within competitive cyclists. A number of mechanisms have been proposed to explain the increased economy/efficiency observed following training. Whilst it has been suggested this is due to fibre type transformation (Coyle et al., 1992; Horowitz et al., 1994; Coyle, 2005), mitochondrial aerobic enzyme adaptation (Burgomaster et al., 2005; Burgomaster et al., 2008), pedalling mechanics and muscle recruitment (Cannon et al., 2007; Korff et al., 2007), the precise underlying mechanisms continue to remain elusive.



A popular theory in the literature is that efficiency is linked to muscle fibre type (Coyle et al., 1992; Horowitz et al., 1994; Mogensen et al., 2006). Thus, an improvement in efficiency would suggest an improved efficiency of ATP turnover within the muscle fibres during contraction (Coyle et al., 1992; Sidossis et al., 1992; Daussin et al., 2008). Coyle et al. (1992) have reported cross-sectional observations that GE and DE during cycling were correlated with the percentage of Type I fibres in the vastus lateralis muscle and a novel leg extension exercise. Using the regression equation from Coyle et al. (1992) the changes in GE of Group A cyclists observed in Chapter 6 would suggest the percentage of Type I muscle fibres in the force producing muscle may have increased from 43 to 66% over a 6 week period of training. However past research (Baumann et al., 1987; Fitts et al., 1989; Windisch et al., 1998; Kadi & Thornell, 1999) has shown the time course of adaptation in muscle fibres to be >8 weeks, and so muscle fibre type transformation is unlikely to be the cause of the increased GE seen. Although, if the full 12 weeks of the study are considered the predicted fibre type transformation using the formulae of Coyle et al. (1992) would be 43 to 75% in Group A cyclists and 63 to 84% in Group B cyclists. Interestingly the predicted magnitude of increase in percentage Type I fibres across this group of cyclists is similar to the Coyle et al. (1991) cross-sectional observations in competitive cyclists. However this would need to be verified using a longitudinal study design, with invasive measures.

Another potential adaptation that also requires verification is that of increases in the aerobic enzyme content of skeletal muscle with training. From the perspective of biochemistry it has long been recognized that increases in aerobic enzyme activity in muscle are possible with short-term endurance training (Henriksson & Reitman, 1977; Dudley et al., 1982; Rodas et al., 2000). This adaptation is evident by a general increase in the mitochondrial content with a corresponding increase in the potential for ATP provision by aerobic metabolism (Holloszy & Coyle, 1984). Recent studies by Burgomaster et al. (2005; 2006; 2008) and Spina et al. (1996) have demonstrated an up-regulation of oxidative enzymes that are linked with mitochondrial biogenesis with 2-6 weeks of interval training. This was linked with a decreased oxygen cost of exercise and ability to sustain exercise.

Gollnick et al. (1973) demonstrated increases in skeletal muscle oxidative capacity and substrate utilisation following several weeks of moderate intensity exercise at  $\sim 65\% \dot{V}O_{2\text{peak}}$ . Similarly Green et al. (1992) have demonstrated that even within 5-7 days of commencing endurance training, increases glycogen availability (and reduced rate of glycogen catabolism) during matched-work exercise result in increase endurance capacity (Green & Dawson, 1995). These shifts in substrate utilisation are often attributed to the enhanced respiratory control sensitivity that results from increased mitochondrial density. In turn this is reflected by changes in the enzymatic activity in the tricarboxylic acid cycle and electron transport chain (Holloszy & Coyle, 1984). Within the training related studies in this thesis

there is evidence of changes in substrate utilisation based on RER measurements, however this is not a consistent finding.

Increased mitochondrial efficiency has also been proposed as a mechanism by which cycling efficiency can be increased. Reduced coupling of respiration has been shown with increased UCP3 content within the muscle (Boss et al., 2000), leading to more energy being lost as heat rather than conserved as ATP within the mitochondria. Mogensen et al. (2006) provide the only study of muscle UCP3 content and efficiency in cyclists to date. They demonstrate that muscle UCP3 and work efficiency were negatively correlated in a cohort of mixed-ability cyclists. More recently Iaia et al., (2009) reported an increased submaximal running economy after a period of high intensity endurance training that was not associated with UCP3 protein expression in trained runners. On the contrary, their results showed a tendency for UCP3 content to actually increase as a result of training. Both results of Mogensen et al. (2006) and Iaia (2009) could however be explained by muscle fibre type. UCP3 is more abundant in Type II fibres (Russell et al., 2003), so relationships could have been secondary to the better-known correlation between fibre-type and efficiency/economy mentioned previously. Iaia et al. (2009) reported an increase in fast twitch fibres post their high intensity interval training. Of course, it may also be that Type II fibres are less efficient *in vivo* precisely because they express more UCP3.

It is likely that the potency of high intensity training comes from the high level of motor unit recruitment required. Consequently Type IIa muscle fibres, which are equally as responsive as Type I fibres in their ability to increase mitochondrial density and enzyme activity will be recruited, stressed and stimulated to adapt to the demands imposed (Jansson & Kaijser, 1997). Intensive endurance training performed for prolonged periods has also been shown to result in alterations in myosin ATPase activity whereby Type II fibres become more like Type I (Widrick et al., 1996). Type I fibres in turn increase ATPase activity, alter myosin type and increase muscle fibre shortening velocity. The increased percentage of Type I fibres contracting at shortening velocities closer to their peak efficiency (equivalent to 80-90 rev.min<sup>-1</sup> [Coyle et al., 1991]), coupled with the possible conversion of Type IIa towards Type I, would therefore mean that more of the muscle was contracting at shortening velocities reflective of the cadences used by the trained within this thesis. This would improve efficiency as the new, most efficient, shortening velocity would better match the actual shortening velocity used during cycling. In support of this speculation, Fitts et al., (1989) demonstrated that ten weeks of intense swimming training increased maximal shortening velocity of Type I fibres, whereas decreases were observed in Type II's. Thus, shortening velocity may be optimised to the rate of shortening experienced during the exercise training. Further research is needed to investigate the influence of muscle fibre shortening velocity.

The protein coactivator PGC-1 $\alpha$  has been shown to mediate adaptations in muscle fibre type and the mitochondria as a result of training (Handschin et al., 2007). Burgomaster et al. (2008) is one of the few studies that have shown PGC-1 $\alpha$  to be increased by training in humans. Whether PGC-1 $\alpha$  directly influenced the decreased oxygen cost of cycling presented within their results is unclear and warrants formal confirmation. Exercise training has been shown to activate PGC-1 $\alpha$  expression via the amplitude and frequency of calcium fluxes (Naya et al., 2000; Olson & Williams, 2000), adenosine monophosphate kinase production (Winder et al., 2006; Rockl et al., 2007; Jager et al., 2007) and production of reactive oxygen species and hypoxia (St-Pierre et al., 2006). PGC-1 $\alpha$  appears to represent an important nexus for translating extracellular information into changes in gene expression, ultimately resulting in phenotype changes including mitochondrial biogenesis and fibre type switching (Type IIb to Type IIa; Type IIa to Type I). These adaptations mean the muscle would be able to generate work more efficiently and may therefore result in a decreased oxygen cost during cycling exercise.

What is clear from the results of this thesis is that the improvements seen in GE are temporary, and sensitive to the intensity of training undertaken. It is therefore evident that the main mechanism driving changes in GE is a relatively plastic and responsive adaptation. This may suggest that the changes seen in the current study were unlikely to be mediated by muscle fibre transformation. Interestingly Fitts et al., (1989) found that training related changes in muscle fibre shortening velocity reverted to baseline level

after a period of detraining. It is speculated that this may be an influencing factor of the results seen in Chapter 5 during the transition period.

#### *$\dot{V}O_2/GE$ relationship*

The inverse relationship between changes in GE and  $\dot{V}O_{2max}$  as a result of training is an interesting finding of this work. Hunter et al. (2005) suggest that this inverse relationship may be related to the muscle fibre. Hunter et al. (2005) demonstrated a high  $\dot{V}O_{2max}$  is more strongly related to a high proportion of Type IIa fibres than Type I. Other research has suggested exercise efficiency is more strongly related to a high proportion of Type I fibres (Coyle et al., 1992; Horowitz et al., 1994; Mogensen et al., 2006). Thus over the 12 week training period of the study documented in Chapter 6, it is possible that cyclists with an initially high proportion of Type IIa fibres experienced some form of transformation towards Type I, thereby increasing their GE and decreasing  $\dot{V}O_{2max}$ . Those cyclists initially high in Type I fibres, also having a high GE at Test 1, subsequently experienced minimal fibre type transformation and so their  $\dot{V}O_{2max}$  remained relatively stable. However this does not help to explain the inverse relationship that is evident after the first 6 weeks of training in Group A cyclists. Although not as strong as that in Chapter 6, the inverse correlation is also evident during the year long study presented in Chapter 5. This finding further supports the negative relationship between GE and  $\dot{V}O_{2max}$  as it is seen both when cyclists conduct “normal” training, and when a specific intervention is implemented. Further studies are required to investigate this relationship and identify potential strategies to maintain  $\dot{V}O_{2max}$  whilst increasing GE.

### *Crank Torque/Muscle recruitment*

From the results presented in this thesis, pedalling mechanics and the cost of moving the legs do not seem to be important factors influencing training related changes in GE. Neither of these parameters demonstrated concomitant changes with GE during the course of a season of training and racing, or when a specific training intervention was implemented. However Korff et al. (2007) suggest that muscle recruitment, rather than mechanical effectiveness measured at the crank may be more of a determinant of GE. Additional torque produced at the crank may not be reflective of the work done by the muscles and therefore the metabolic cost incurred (Kautz & Neptune, 2002).

It is possible that adaptations may have occurred within the muscle cell as a result of training that altered its contraction properties. Muscle activation and deactivation properties could have then altered the amount of time required for muscle contraction and relaxation. Theoretically as muscle fibre shortening velocity and muscle activation and deactivation are better matched to the required rate during pedalling at a given cadence (i.e. the cyclist may have become more coordinated in their pedalling action within and between muscles), less negative muscle work may be encountered. Lower negative muscle work (in antagonistic muscles) would require comparably less positive muscle force (in agonist muscles) to overcome the resistance at any given power output, resulting in a lower submaximal O<sub>2</sub> cost and higher GE. However this is purely speculation due to the difficulty of performing invasive evaluations on muscle force, length, velocity and

activation. Currently no equipment is available to measure these parameters during cycling in vivo, although electromyography may give an indication of muscle timing.

It is not expected that cadence changes were a factor in the results. Cyclists were encouraged perform the majority of their training at the same preferred rate as used in the laboratory tests. When freely chosen cadence was monitored in the final stages of the efficiency test documented in Chapter 5, no significant differences were found over the course of the season. Therefore it is unlikely that cyclists had any cadence related adaptations that may have influenced GE.

### **7.6 Suggestions for future research**

The number of findings from past research that have suggested no significant differences exist between trained and untrained cyclists has limited the extent of research in the field of efficiency. Previous arguments in this thesis have suggested that this is largely due to method problems leading to type 2 statistical errors. Based upon the proposed physiological factors which influence efficiency, differences should be apparent. The research studies conducted within this work clearly demonstrate that with a sound methodological approach differences between trained and recreational cyclists are apparent. Furthermore, cycling efficiency is influenced in the short-term (6 weeks) by the intensity and volume of training. A comparison of training regimes confirms that high intensity training is a key stimulus for increasing efficiency. Further studies are



needed to identify the mechanisms influencing these changes. Findings are further complicated by the inverse relationship which was found between GE and  $\dot{V}O_{2\max}$  ( $r = -0.77-0.82$ ). It is possible that as Lucia (2002a) suggests, GE increases to compensate for  $\dot{V}O_{2\max}$ , but it is unlikely to be solely down to this factor. Hunter et al. (2005) propose that muscle fibre type transformation may account for this inverse relationship. Therefore more invasive studies are needed to examine the link between muscle related adaptation, changes in GE and its inverse relationship with  $\dot{V}O_{2\max}$ . Further studies are also required to identify training strategies which combine overload and progression stimuli to optimise GE gains, whilst at the same time protect maximal oxidative capacity ( $\dot{V}O_{2\max}$ ). Similarly future studies could consider the extent to which GE can continue to improve if the overload of training intensity is continually progressed. Finally, training studies might be implemented to seek to maintain efficiency during the transition period of the cycling season. Thus at the start of the following training year further training related increases may be possible. The use of cyclo-cross riders who road race during the summer months but then also maintain the intensity of their training and racing by competing over the winter months might be an appropriate group to study.

Past research investigating skeletal muscle's impact on efficiency have been cross-sectional in nature (Coyle et al., 1992; Horowitz et al., 1994; Mogensen et al., 2006). Longitudinal intervention studies are required to add more credibility. Muscle biopsies taken pre and post training interventions that increase GE would help to identify any link between

changes in efficiency and adaptations in the mitochondrion, aerobic enzyme concentration and/or muscle fibre type. Similarly the taking of muscle biopsies would enable the analysis of single muscle fibre function to identify changes in muscle fibre contraction velocity.

It is currently unclear what impact the magnitude of changes seen in GE within this thesis would have on performance. Predicted changes in performance based on mathematical modelling are possible, however this does not have ecological validity. No study has directly investigated the impact that increasing efficiency would have on cycling performance. The impact of changing efficiency has only been investigated by Passfield and Doust (2000) who demonstrated fatigue following prolonged exercise decreased GE. Horowitz et al. (1994) demonstrated differences in performance ability of cyclists high and low in GE using a cross-sectional approach. When considering the results of these studies it could be suggested that the improvement of efficiency seen within this thesis would translate into a performance advantage. However this study did not go as far as testing the cyclists in a time-trial setting.

## **7.7 Conclusions**

This thesis evaluated the impact of training on metabolic efficiency during cycling. Key issues concerning the reliability of the measurement of efficiency were addressed in order to establish appropriate and reproducible methods. Main findings were that GE was significantly higher in a trained population of cyclists irrespective of whether absolute or relative power

outputs were considered. Over the period of a training/detraining as occurs across a cycling season, GE increased and maintained by the volume and intensity of training that the cyclists undertook. When training volume and intensity was subsequently decreased GE also declined. Other efficiency measures of DE and WE proved not to be so informative due to the validity and reliability of their measurement. Both DE and WE demonstrated increases over the course of the season, however due to the “noise” in each measurement no “significant” changes were detected.

High intensity training was identified as a key stimulus for increasing GE by the implementation of a training intervention study. The supplementation of specific high intensity training sessions provided a significant increase in GE compared to volume only training. Interestingly when high intensity work was supplemented to volume only training comparable increases in GE were seen. The exact mechanisms for training related increases in GE remain to be fully understood as they were not a focus of the current thesis. However it is hoped that this thesis provides signposts for future research.

Unexpectedly an inverse relationship was identified between changes in GE and changes  $\dot{V}O_{2max}$  over the course of a season, as well as when specific training interventions were used. Therefore the increase in GE appears to be at the expense of maximal aerobic capacity. It is unclear why there should be an inverse relationship between GE and  $\dot{V}O_{2max}$  and the reasons for this. Further research is needed to examine the mechanisms of this finding and to

establish if it is possible to preserve  $\dot{V}O_{2\max}$  whilst at the same time maximising efficiency. It also remains to be determined what impact this inverse relationship has on cycling performance.

Crank torque data proved not to be related to efficiency measurements. This may be due to the fact that what is occurring at the crank, in terms of torque production, may not reflect total muscular work being done. Similarly total muscular work may not be the only physiological process that is measured by collecting gases breathed out at the mouth.

When considering the findings of this thesis in light of previous research investigating both acute and chronic changes in efficiency, it is evident that the magnitude of improvements found is considerable. Although it was not possible to identify the exact mechanisms for the increases seen, evidence of previous research suggests that those responsible may be a combination aerobic enzyme adaptations, muscle fibre adaptations, and enhanced coordination of muscle recruitment. Based upon the current findings, it can however be concluded that high intensity training is a key stimulus for improving gross efficiency in competitive cyclists.

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## APPENDIX 1

### THE RELIABILITY OF THE MEASUREMENT OF CYCLING EFFICIENCY

#### PARTICIPANT INFORMATION

A research study is being conducted at the University of Kent and Canterbury Christ Church University (CCCU) as part of a PhD thesis by James Hopker. Dr. Damian Coleman, Senior Lecturer at CCCU is a co-researcher.

#### Background

The aims of this study are to:

1. To identify the reliability of physiological responses from a constant load test protocol using a preferred cadence method
2. To assess the reliability of the mathematical methods for the calculation of gross and delta efficiency as well as crank torque effectiveness

#### What will you be required to do?

Participants in this study will be required to visit the laboratory on two occasions, once for a test of their maximal aerobic capacity and the second time for a test of their cycling efficiency.

To participate in this research you must:

- be either A competitive male cyclist for at least 2 years or recreational cyclist, aged 18 – 45 years
- be a non-smoker
- not be taking any medications (for high cholesterol, high blood pressure, etc.)
- have no known heart condition or diabetes.

#### Procedures

The study requires four visits (one per week for four weeks) to the Sport and Exercise Science laboratory at the University of Kent at Medway.

In every test your oxygen uptake will be measured. You will wear a face mask, attached to a gas analysis machine that will enable the measurement of the amount of oxygen you are breathing and using. You may feel a little uncomfortable, however, you will be given a chance to get to use it prior to testing. As a trained athlete, risks in testing are minimal. If however, you fell 'out of the ordinary' during any test you should let me know immediately so that the test can be terminated.

### **Visit 1**

Maximal Oxygen Uptake Test (VO<sub>2Max</sub>): This test involves a fast ramped protocol starting at 100 watts, progressing by 25 watts every minute until you are no longer able to continue. This test is likely to last from between 14 to 20 minutes, and involves exercising at a maximal level for a short period. You will experience some discomfort, comparable with riding slightly above 10 mile time trial pace.

### **Visit 2 – 4 (Separated by 3 – 7 days)**

Efficiency test: Each of these tests will involve a ride for 20 minutes at two submaximal power outputs (50% Maximal Minute Power (MMP) for 10 minutes, 60% MMP for 10 minutes), utilising your preferred cadence recorded in the VO<sub>2Max</sub> test. This will be performed against 50 and 60% of the maximum power output you achieved during your maximal test.

### **Feedback**

If required you will receive written feedback of your maximal test results outlining your training intensities. Included within this will be training advice and an offer of future testing if this is likely to benefit your training.

### **Confidentiality**

All measurements (data) and personal information will be stored securely within University of Kent premises in accordance with the Data Protection Act 1998 and the University's own data protection requirements. Data can only be accessed by James Hopker and Dr. Damian Coleman. After completion of the study, all data will be made anonymous (i.e. all personal information associated with the data will be removed).

### **Deciding Whether to Participate**

If you have any questions or concerns about the nature, procedures or requirements for participation do not hesitate to contact me. Should you decide to participate, you will be free to withdraw at any time without having to give a reason.

### **Any Questions?**

Please contact **James Hopker** on 01634 888814 or email [\*\*j.g.hopker@kent.ac.uk\*\*](mailto:j.g.hopker@kent.ac.uk)

Centre for Sport Studies, University of Kent at Medway, The Medway Building, Chatham Maritime, Chatham, ME4 4AG.

## APPENDIX 2

### DIFFERENCES IN EFFICIENCY BETWEEN TRAINED AND RECREATIONAL CYCLISTS

#### PARTICIPANT INFORMATION

A research study is being conducted at the University of Kent and Canterbury Christ Church University (CCCU) as part of a PhD thesis by James Hopker. Dr. Damian Coleman, Senior Lecturer at CCCU is a co-researcher.

#### Background

Previous research has demonstrated that metabolic efficiency is an important determinant of endurance performance. Within cycling this is measured as the amount of energy used for a given amount of physical work (i.e. watts of power). It has been suggested that if efficiency can be improved by as little as 1% there is the potential for a significantly enhanced cycling performance. However there few previous studies actually investigating it's trainability. Those that have researched this area found no differences between world class and recreational cyclists, suggesting that efficiency cannot be trained. However this does not sit well with the physiological components that determine metabolic efficiency. It is the intention of this research to investigate these findings further and identify if differences do actual exist.

#### What will you be required to do?

Participants in this study will be required to visit the laboratory on two occasions, once for a test of their maximal aerobic capacity and the second time for a test of their cycling efficiency.

#### To participate in this research you must:

- be either a fit recreationally active cyclist or a competitive male cyclist for at least 2 years or recreational cyclist, aged 18 – 45 years
- be a non-smoker
- not be taking any medications (for high cholesterol, high blood pressure, etc.)
- have no known heart condition or diabetes.

#### Procedures

##### **Visit 1**

You will be asked to complete a brief questionnaire about your current training regime. Weight and height will be measured prior to a 10 minute warm-up on the cycle ergometer. You will then undergo a test of your maximal aerobic exercise capacity. This test involves an incremental protocol starting at 100 watts, progressing by 25 watts every minute until you are no



longer able to continue. This test is likely to last from between 14 to 20 minutes, and involves you exercising at a maximal level for a short period. You will experience some discomfort, comparable with riding slightly above 10 mile time trial pace. Considerable self-motivation is required to achieve a true reflective measure of your maximal capacity.

### **Visit 2**

This test will involve either a ride for 20 minutes at two submaximal power outputs (50% Maximal Minute Power (MMP) for 10 minutes, 60% MMP for 10 minutes), utilising your preferred cadence recorded during visit 1 and a 10 minute ride at 150Watts.

- Drink ONLY WATER in the morning of testing
- No alcohol within 24 hours
- Not to be taking any medication for preceding 4 weeks
- Free from dietary supplementation (vitamins etc.) for the preceding 4 days
- Free from illness/infection during the preceding 2 weeks

### **Feedback**

If required you will receive written feedback of your maximal test results outlining your training intensities. Included within this will be training advice and an offer of future testing if this is likely to benefit your training.

### **Confidentiality**

All measurements (data) and personal information will be stored securely within University of Kent premises in accordance with the Data Protection Act 1998 and the University's own data protection requirements. Data can only be accessed by James Hopker and Dr. Damian Coleman. After completion of the study, all data will be made anonymous (i.e. all personal information associated with the data will be removed).

### **Deciding Whether to Participate**

If you have any questions or concerns about the nature, procedures or requirements for participation do not hesitate to contact me. Should you decide to participate, you will be free to withdraw at any time without having to give a reason.

### **Any Questions?**

Please contact **James Hopker** on 01634 888814 or email [\*\*j.g.hopker@kent.ac.uk\*\*](mailto:j.g.hopker@kent.ac.uk)

Centre for Sport Studies, University of Kent at Medway, The Medway Building, Chatham Maritime, Chatham, ME4 4AG.

## **APPENDIX 3**

### **CHANGES IN CYCLING EFFICIENCY DURING A COMPETITIVE SEASON**

#### **PARTICIPANT INFORMATION**

A research study is being conducted at the University of Kent at Medway and Canterbury Christ Church University (CCCU) as part of a PhD thesis by James Hopker. Dr. Damian Coleman, Senior Lecturer at CCCU is a co-researcher.

#### **Background**

Previous research has demonstrated that metabolic efficiency is an important determinant of endurance performance. Within cycling this is measured as the amount of energy used for a given amount of physical work (i.e. watts of power). It has been suggested that if efficiency can be improved by as little as 1% there is the potential for a significantly enhanced cycling performance. However there few previous studies actually investigating its trainability. Research conducted on runners has suggested that different types of training (e.g. low vs high intensity) would influence gross mechanical efficiency. It is therefore the intent of this research to track changes in gross mechanical efficiency of trained cyclists over the course of a competitive season whereby training intensities would vary depending on the phase of training.

#### **What will you be required to do?**

Participants in this study will be required to visit the laboratory on five occasions over the course of the season for a maximal test of maximal aerobic capacity. These would be scheduled in the training transition periods of January, March, July, October and December. Participants would also be required to train according to heart rate (a heart rate monitor will be provided). Heart rates will need to be recorded every training session and downloaded on a regular basis.

To participate in this research you must:

- be a competitive male cyclist for at least 2 years, aged 18 – 45 years
- be a non-smoker
- not be taking any medications (for high cholesterol, high blood pressure, etc.)
- have no known heart condition or diabetes.

#### **Testing Procedures**

You will be asked to complete a brief questionnaire about your current training regime. Weight and height will be measured prior to a 5 minute

warm-up on the cycle ergometer. You will then undergo a test of your maximal aerobic exercise capacity. This test involves an incremental protocol starting at 150 watts, progressing by 30 watts every eight minutes until you are no longer able to continue. This test is likely to last from between 60 to 90 minutes, and involves you exercising at a maximal level for a short period. Small finger prick blood samples will be taken throughout the test. You will experience some discomfort, comparable with riding slightly above 10 mile time trial pace. Considerable self-motivation is required to achieve a true reflective measure of your maximal capacity.

### **Feedback**

If required you will receive written feedback of your maximal test results outlining your training intensities. Included within this will be training advice and an offer of future testing if this is likely to benefit your training.

### **Confidentiality**

All measurements (data) and personal information will be stored securely within University of Kent premises in accordance with the Data Protection Act 1998 and the University's own data protection requirements. Data can only be accessed by James Hopker and Dr. Damian Coleman. After completion of the study, all data will be made anonymous (i.e. all personal information associated with the data will be removed).

### **Deciding Whether to Participate**

If you have any questions or concerns about the nature, procedures or requirements for participation do not hesitate to contact me. Should you decide to participate, you will be free to withdraw at any time without having to give a reason.

### **Any Questions?**

Please contact **James Hopker** on 01634 888814 or email [\*\*j.g.hopker@kent.ac.uk\*\*](mailto:j.g.hopker@kent.ac.uk)  
Centre for Sport Studies, University of Kent at Medway, The Medway Building, Chatham Maritime, Chatham, ME4 4AG.

## APPENDIX 4

### **THE EFFECT OF TRAINING VOLUME AND INTENSITY ON GROSS EFFICIENCY**

#### **PARTICIPANT INFORMATION**

A research study is being conducted at the University of Kent at Medway and Canterbury Christ Church University (CCCU) as part of a PhD thesis by James Hopker. Dr. Damian Coleman, Senior Lecturer at CCCU is a co-researcher.

#### **Background**

Previous research has demonstrated that metabolic efficiency is an important determinant of endurance performance. Within cycling this is measured as the amount of energy used for a given amount of physical work (i.e. watts of power). It has been suggested that if efficiency can be improved by as little as 1% there is the potential for a significantly enhanced cycling performance. However there few previous studies actually investigating its trainability. The results of a previous research study conducted within this series of investigations have demonstrated that gross efficiency increased over the course of a competitive cycling season. It has been found that greater the volume of low intensity training cyclists completed greater their increases in the gross mechanical efficiency. Similarly results indicated that the greater the volume of moderate intensity training those riders completed the higher their gross efficiency. It is therefore the intention of this study to identify which type of training leads to the greatest improvement in gross efficiency over the period of time from January to May.

#### **What will you be required to do?**

Participants in this study will be required to visit the laboratory on three occasions (January and May) for a test of cycling efficiency and a maximal test of maximal aerobic capacity. Participants would also be required to train at specific prescribed intensities according to heart rate (a heart rate monitor will be provided). Heart rates will need to be recorded every training session and downloaded on a regular basis.

To participate in this research you must:

- be a competitive male cyclist for at least 2 years, aged 18 – 55 years
- be a non-smoker
- not be taking any medications (for high cholesterol, high blood pressure, etc.)

- have no known heart condition or diabetes.

### Testing Procedures

You will be asked to complete a brief questionnaire about your current training regime. Weight and height will be measured prior to a 5 minute warm-up on the cycle ergometer. You will then undergo a test of your maximal aerobic exercise capacity. This test involves an incremental protocol starting at 150 watts, progressing by 30 watts every eight minutes until you are no longer able to continue. This test is likely to last from between 60 to 90 minutes, and involves you exercising at a maximal level for a short period. You will experience some discomfort, comparable with riding slightly above 10 mile time trial pace. Considerable self-motivation is required to achieve a true reflective measure of your maximal capacity.

### Feedback

If required you will receive written feedback of your maximal test results outlining your training intensities. Included within this will be training advice if this is likely to benefit your training.

### Confidentiality

All measurements (data) and personal information will be stored securely within University of Kent premises in accordance with the Data Protection Act 1998 and the University's own data protection requirements. Data can only be accessed by James Hopker and Dr. Damian Coleman. After completion of the study, all data will be made anonymous (i.e. all personal information associated with the data will be removed).

### Deciding Whether to Participate

If you have any questions or concerns about the nature, procedures or requirements for participation do not hesitate to contact me. Should you decide to participate, you will be free to withdraw at any time without having to give a reason.

### Any Questions?

Please contact **James Hopker** on 01634 888814 or email [j.g.hopker@kent.ac.uk](mailto:j.g.hopker@kent.ac.uk)  
Centre for Sport Studies, University of Kent at Medway, The Medway Building, Chatham Maritime, Chatham, ME4 4AG.

# CONSENT FORM

**Title of Project:**

**Name of Researcher:**

**Contact details:**

Address:

Tel:

Email:

**Please initial box**

1. I confirm that I have read and understand the information sheet for the above study and have had the opportunity to ask questions.
2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason.
3. I understand that any personal information that I provide to the researchers will be kept strictly confidential
4. To my knowledge I have not taken any substances that will adversely or otherwise affect this study
5. I agree to take part in the above study.

\_\_\_\_\_  
Name of Participant

\_\_\_\_\_  
Date

\_\_\_\_\_  
Signature

\_\_\_\_\_  
Name of Person taking consent  
(if different from researcher)

\_\_\_\_\_  
Date

\_\_\_\_\_  
Signature

\_\_\_\_\_  
Researcher

\_\_\_\_\_  
Date

\_\_\_\_\_  
Signature

PRE-TEST HEALTH QUESTIONNAIRE

Name.....

Date Of Birth..... Age.....

Please answer these questions truthfully and completely. The sole purpose of this questionnaire is to ensure that you are in a fit and healthy state to complete the exercise test.

1. How would you describe your present level of activity?

- Vigorous activity:            Less than once a month
- Once a month
- Once a week
- Two or three times a week
- Four or five times a week
- More than five times a week

2. Do you suffer, or have you ever suffered from any form of heart complaint?

Yes / No

3. Do you suffer, or have you ever suffered from:

- Asthma                            Yes / No
- Diabetes                           Yes / No
- Bronchitis                        Yes / No
- Epilepsy                           Yes / No
- High Blood Pressure          Yes / No

4. Have you had to consult your doctor in the last three months? If yes, please give brief details

.....  
.....

5. Are you currently taking any form of medication? If yes, please give brief details

.....  
.....

6. Do you have any form of muscle or joint injury?

Yes / No

7. Have you suffered from a bacterial or viral infection in the last two weeks?

Yes / No

8. Have you had cause to suspend your training in the last two weeks for a physical reason?

Yes / No

9. Is there any reason why you should not be able to successfully complete tests which require maximum effort?

Yes / No

If yes, please give brief details

.....  
.....

Signature of Participant.....

Date.....

Signature of Researcher.....