Fachhochschule Wiener Neustadt
Wirtschaft · Technik · Gesundheit · Sicherheit · Sport

Mechanical efficiency and oxygen uptake kinetics during flat and uphill cycling at different cadences in indoor cycling

Masterarbeit

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Wiener Neustadt, 01.10.2014

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Kurzzusammenfassung:

Die Parameter der Effizienz und Sauerstoffaufnahmekinetik beim Radfahren sind als wichtige Messparameter in der Leistungsdiagnostik postuliert. Die Einflüsse auf diese Parameter wurden bereits eingehend unter Laborbedingungen untersucht. Der Einfluss von Geländeneigung wurde aber bisher noch kaum erforscht. Darum ist das Ziel dieser Studie die Auswirkungen von Geländeneigung, Trittfrequenz und Intensität auf die Gesamteffizienz (GE) und Sauerstoffaufnahmekinetik unter Laborbedingungen zu untersuchen.

An der Studie nahmen dreizehn gut trainierte Radfahrer teil (Mittelwert ± Standardabweichung Alter: 23.0 ± 4.7 Jahre; Größe: 178.5 ± 5.2 cm; Gewicht: 69.0 ± 7.8 kg; $\dot{V}O_{2max}$: 68.2 ± 4.7 $mL \cdot min^{-1} \cdot kg^{-1}$). Die Studie bestand aus zwei Tests: einem Stufentest im Labor (GXT) und an einem separaten Tag, 8 Tests mit einer Dauer von jeweils 6 min. Der GXT diente zur Ermittlung der maximalen Sauerstoffaufnahme ($\dot{V}O_{2max}$), der maximalen Leistung (P_{max}) und der ventilatorischen Schwellen (VT und RCP). Um die Geländeneigung im Labor zu simulieren wurde das Test Rad auf einem Rollentrainer (TACX) montiert und am Laufband fixiert. Die Steigung wurde dann mithilfe des Laufbands verändert. Während der Labortests absolvierten die Probanden 4 Tests in der Ebene (Steigung 1.5%) und 4 Tests im simulierten Anstieg (Steigung 5%). Die Tests beinhalteten zwei Intensitäten (90%VT und Δ70) und zwei Trittfrequenzen (60 und 90 $rev \cdot min^{-1}$). Die Reihenfolge der 4 Tests in der Ebene und im Anstieg war: 90%VT mit 60 $rev \cdot min^{-1}$, Δ70 mit 60 $rev \cdot min^{-1}$, 90%VT mit 90 $rev \cdot min^{-1}$, Δ70 mit 90 $rev \cdot min^{-1}$.

Signifikante Einflüsse der simulierten Steigung gab es auf die Zeitkonstante (τ) (mittlere Differenz = 2.8 s; $F_{1,12}$ = 5.1; p = 0.043) und die Sauerstoffaufnahme am Ende der Tests (mittlere Differenz = 69 $mL \cdot min^{-1}$; $F_{1,12}$ = 6.3; p = 0.027) in der Phase II der Sauerstoffaufnahmekinetik. Die unterschiedlichen Trittfrequenzen beeinflussten signifikant die Zeitkonstante (mittlere Differenz = 3 s; $F_{1,12}$ = 7.1; p = 0.021) und die Amplitude (mittlere Differenz = 176 $mL \cdot min^{-1}$; $F_{1,12}$ = 14.8; p = 0.002). Signifikante Einflüsse auf alle Parameter der Sauerstoffaufnahmekinetik, mit Ausnahme des Verzögerungswerts (time delay) gab es durch die unterschiedlichen zwei Intensitäten.

Einfluss auf die Effizienz hatte die Trittfrequenz (21 ± 1.6 % bei 60 $rev \cdot min^{-1}$; 18.6 ± 1.1 % bei 90 $rev \cdot min^{-1}$; p < 0.001) nicht aber die Geländeneigung (19.7 ± 1.8 % in der Ebene; 19.7 ± 1.8 % in der Steigung; p = 0.81).

Schlagworte (mind. 3, max. 6):

Sauerstoffaufnahmekinetik, Gesamteffizienz, Geländeneigung, Laborbedingungen

Abstract:

One of the most meaningful parameters for performance measurement in cycling are oxygen uptake kinetics and gross efficiency. A number of studies investigated efficiency and oxygen uptake during cycling in the laboratory. Therefore, the aim of this study was to analyse the effect of gradient, cadence and exercise intensity on oxygen uptake kinetics and gross efficiency (GE) in laboratory conditions and to verify previous results. Thirteen well-trained cyclists participated in this study (mean ± SD age: 23.0 ± 4.7 years; stature: 178.5 ± 5.2 cm; body mass: 69.0 ± 7.8 kg; $\dot{V}O_{2max}$: 68.2 ± 4.7 $mL \cdot min^{-1} \cdot kg^{-1}$). The study consisted of two testing sessions: one incremental graded exercise test (GXT) to exhaustion and on a separate day 8 test-trials of 6 min duration. To simulate gradient the test bike was mounted on an indoor training roller and fixed on a treadmill. The GXT was performed to determine maximum oxygen uptake ($\dot{V}O_{2max}$), maximum power output (P_{max}) and gas exchange thresholds (VT and RCP). During the laboratory test the subjects performed 4 trials on level ground (1.5% inclination) and 4 uphill trials (5% inclination). The trials were performed at two intensities (90%VT and Δ70) and two cadences (60 and 90 $rev \cdot min^{-1}$). The order of the four level and uphill cycling trials was 90%VT at 60 $rev \cdot min^{-1}$, Δ70 at 60 $rev \cdot min^{-1}$, 90%VT at 90 $rev \cdot min^{-1}$, Δ70 at 90 $rev \cdot min^{-1}$.

Significant differences between uphill and level ground cycling were found for the time constant (τ) (mean difference = 2.8 s; $F_{1,12}$ = 5.1; p = 0.043) and end-exercise $\dot{V}O_2$ (mean difference = 69 $mL \cdot min^{-1}$; $F_{1,12}$ = 6.3; p = 0.027) of the phase II oxygen uptake response. Cadence significantly affected the τ (mean difference = 3 s; $F_{1,12}$ = 7.1; p = 0.021) and amplitude (mean difference = 176 $mL \cdot min^{-1}$; $F_{1,12}$ = 14.8; p = 0.002). Significant differences between moderate and high exercise intensities were found for all measured oxygen uptake kinetics parameters (i.e. τ, amplitude, slow component, end-exercise $\dot{V}O_2$ gain, phase II $\dot{V}O_2$ gain and end-exercise $\dot{V}O_2$) except time delay.

The GE was affected by cadence (21 ± 1.6 % at 60 $rev \cdot min^{-1}$; 18.6 ± 1.1 % at 90 $rev \cdot min^{-1}$; p < 0.001) but no significant effect of flat (19.7 ± 1.8 %) compared to uphill cycling (19.7 ± 1.8 %) was found (p = 0.81).

Keywords :

oxygen uptake kinetics, gross efficiency, inclination, indoor conditions

Contents

Introduction

Despite the importance of uphill cycling performance, there is limited research investigating this ability. The aims of the present study were to examine the effects of high and low cadences during simulated uphill conditions in the laboratory on mechanical efficiency and oxygen uptake kinetics. To enhance and measuring performance the mechanisms of the oxygen uptake kinetics and mechanical efficiency seems to be two important fields. For explanation of the mechanisms of the oxygen uptake kinetics and mechanical efficiency a short literature overview has been done.

1. Oxygen uptake kinetics

The human body needs a continuous supply of chemical energy which is stored in the adenosine tri-phosphate (ATP) molecule to maintain numerous functions. ATP is a special carrier molecule of free energy and powers all of the cell´s energy requiring processes. Cells contain only a small quantity of ATP and therefore it is crucial that ATP concentrations are maintained during exercise. For the maintenance of intramuscular ATP the molecule must be continually resynthesize via three energetic pathways.

The three energetic systems are the phosphocreatine (PCr) system, the glycolytic system and the aerobic system. The beginning of muscular movement rapidly activates the PCr system. The PCr system is dominant for the first ten seconds during a maximal effort. The anaerobic glycolytic system is also readily available and supplies ATP with a net formation of lactic acid. When the glycolytic flux is slower, the glycolytic reaction can be used with pyruvic acid oxidised and lactate equilibrium maintained. The immediate and anaerobic glycolytic systems have a finite capacity because they rely in phosphorylation at a level of limited substrates and are associated with the accumulation of metabolic by-products. As long as the glycolytic flux rate is not too fast and enough oxygen (O_2) can be delivered, oxidative phosphorylation is used. Oxidative phosphorylation has no such limitations like the glycolytic system (DiMenna, 2010). For this oxidative processes O_2 must be inspired via the ventilatory system, transported through the cardiovascular system to the muscles and consumed in the mitochondria to produce ATP (Figure 1). Factors such as highest attainable $\dot{V}O_2$ ($\dot{V}O_{2max}$) or the rate at which $\dot{V}O_2$ rises in the transition to an activity, will all influence an athlete's tolerance to physical activity (Jones & Poole, 2005a). If muscular work spontaneously increase, muscle O_2 uptake must also rise to support the ATP production. The ATP turnover

increases instantaneously, while the muscle oxygen uptake increases relatively slowly. This rise in muscle $\dot{V}O_{2max}$ can be described by an exponential function. Jones and Poole (2005b) proposed that the muscle and pulmonary $\dot{V}O_2$ responses at moderate work rates (e.g. where a steady state occurs) gives a close match to the muscle ATP turnover rate.

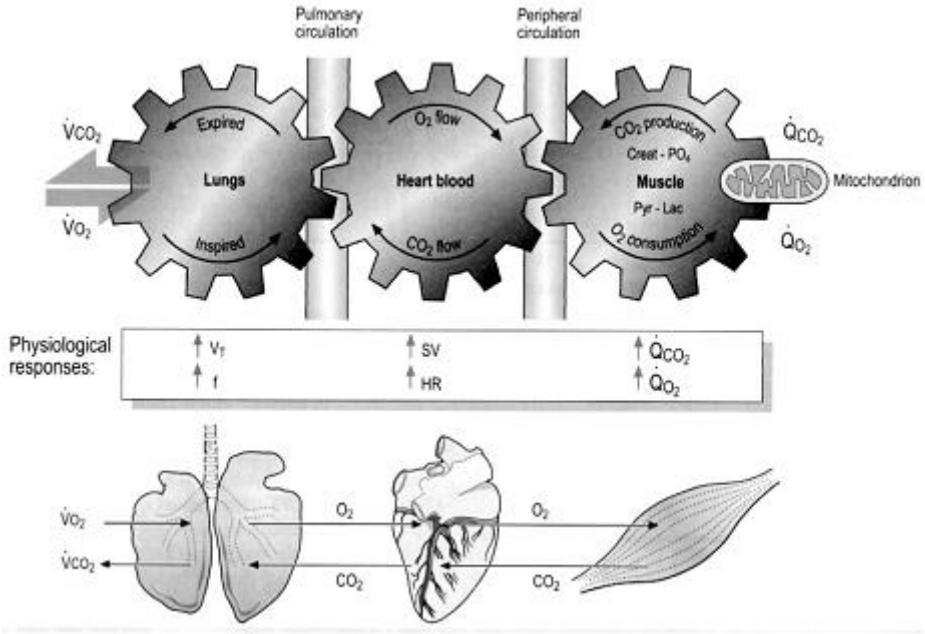

Figure 1: Illustration of the schematic relationship between muscle O_2 consumption and pulmonary O_2 uptake. V_T = tidal volume; f = breathing frequency; SV = stroke volume; HR = heart rate; Creat-PO_4 = creatine phosphate; Pyr-Lac = pyruvate-lactate; $\dot{Q}CO_2$ = muscle CO_2 production; $\dot{Q}O_2$= muscle O_2 utilization (Jones & Poole, 2005b).

1.1 Measuring O2 uptake kinetics

Ideally, the oxidative response in the active muscles would be assessed by direct measurement of mitochondrial O_2 consumption. Although direct measures of O_2 in the exercising muscle are theoretically the ideal method, this approach is highly invasive and technically complex. Therefore commonly non-invasive breath-by-breath measurements are used to measure $\dot{V}O_2$ to determine the physiological dynamic of muscle and pulmonary $\dot{V}O_2$ kinetics. The $\dot{V}O_2$ measured at the mouth during exercise reflects the elevated $\dot{V}O_2$ in the exercising muscles, although there is a degree of contamination in measurements of respiratory gas exchange at the mouth. Especially, the transition time delays and the interposition of O_2 stores between the sites of O_2 exchange and measurement have to take into account by determining muscle $\dot{V}O_2$ with

measurement via respired gas analysis (Behnke et al., 2005). The oxygen uptake response is associated with other physiological responses like increased heart rate (HR) and pulmonary ventilation. The dynamic responses of pulmonary gas exchange reflects the integrated response of the ventilatory, cardiovascular and neuromuscular systems to the exercise challenge (Jones & Poole, 2005a).

First studies concerning $\dot{V}O_2$ kinetics where carried out in 1913 by Krogh and Lindhard and later by Hill and Lupton. They were first recognizing the importance of the dynamic phase of $\dot{V}O_2$ and its role in determining the oxygen deficit at exercise onset. For measuring $\dot{V}O_2$ they used the Douglas bag technique. Expired gases were periodic collected in Douglas bags and analyzed afterwards (A. V. Hill et al., 1924; Krogh & Lindhard, 1913).

Nowadays, the open-circuit method is used for measurement of expired gases. Rapidly responding gas analysers and economical flow meters allow a measurement of $\dot{V}O_2$ parameters on breath-by-breath basis. Whole body O_2 consumption measured at the mouth closely reflects the kinetics of O_2 consumption in active muscles (DiMenna, 2010). Therefore, this instrument becomes a valuable tool for characterization of $\dot{V}O_2$ kinetics and many O_2 uptake kinetics researchers base their findings on pulmonary $\dot{V}O_2$ measurements (Koga et al., 2005).

1.2 Characteristics of the $\dot{V}O_2$ response at exercise onset

The pulmonary $\dot{V}O_2$ uptake following the start of exercise has been well described (Linnarsson, 1974; Whipp et al., 1982; Whipp & Wasserman, 1972). The first exponential models by Henry (1951) and subsequent ones (Margaria et al., 1965; Whipp, 1971) show already that an exponential process began immediately when work rate was increased.

At the onset of movement or dynamic exercise, pulmonary oxygen uptake continues, after a short delay representing the muscle to lung blood transit time, along a time course that can be modelled by a single-exponential function (Whipp et al., 1982). They developed the standard procedure for data conditioning for $\dot{V}O_2$ kinetics investigations. The breath-by-breath data points were transformed by interpolation into second-by-second data points so that the analysis was not biased by irregular distribution (Beaver et al., 1986).

The typical response of $\dot{V}O_2$ uptake during the transition from rest or unloaded exercise to moderate or heavy exercise with steady-state or slow-component (SC) can be divided into three distinct phases (Figure 2).

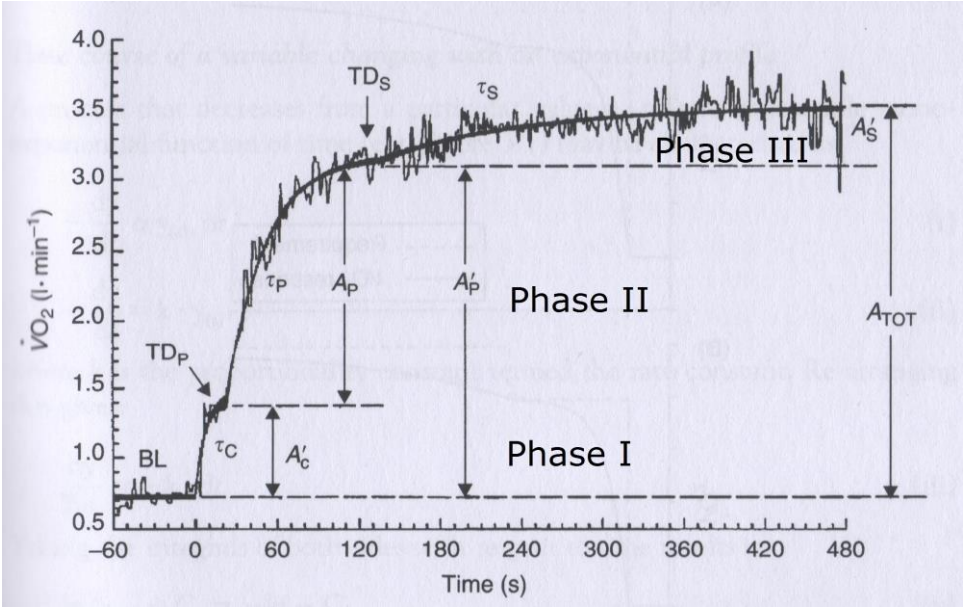

Figure 2: Typical response of O_2 uptake during heavy cycling exercise. BL = baseline; τ_C = time constant cardiac phase; A'_C = amplitude cardiac phase; TD_P = time delay primary (fundamental) phase; τ_P = time constant fundamental phase; A_P = amplitude fundamental phase; A'_P = Amplitude from BL fundamental phase; TD_S = time delay SC or steady state phase; τ_S = time constant SC; A'_S = amplitude SC phase; A_{TOT} = end-exercise amplitude over BL. Adapted from Whipp and Rossiter (2005).

Phase I - cardiodynamic phase

At the beginning of a constant work rate exercise, there is an early rapid initial response in the first 15-20 s. This first increase in $\dot{V}O_2$ is called cardiodynamic phase. It is associated with an increased blood flow through the lung consequent to the immediate higher cardiac output at the onset of exercise. This leads to an increase in $\dot{V}O_2$ which does not reflect an increased muscle O_2 consumption (Jones & Poole, 2005a). The phase I response can be described with the following equation (Koga et al., 2005; Mahler, 1985):

$$\dot{V}O_2(t) = \dot{V}O_{2baseline} + A_0 (1 - e^{-t/\tau 0})$$

Where $\dot{V}O_2(t)$ is the $\dot{V}O_2$ at any time t, $\dot{V}O_{2baseline}$ is the $\dot{V}O_2$ before the onset of exercise, A_o is the asymptotic amplitude and τ_o is the time constant.

The end of phase I and beginning of phase II is marked by a fall in the end-tidal pressure of O_2 ($PETO_2$), resulting from a higher muscle O_2 consumption (Jones & Poole, 2005b). This point of transition from phase I to phase II occurs at about 20 seconds after exercise onset and is determined by visual inspection (Murias et al., 2011). Visual inspection is important because a slow cardiovascular response to exercise might lead to a prolonged phase I (Poole & Jones, 2005).

Usually phase I response is removed from kinetic analysis in order to infer muscle $\dot{V}O_2$ from pulmonary $\dot{V}O_2$ (Koga et al., 2005).

Phase II – fundamental phase

Phase II or primary/fundamental phase is characterized by a rapid exponential increase in $\dot{V}O_2$ towards the expected steady state or beginning of SC. The exponential increase in $\dot{V}O_2$ is initiated when venous blood from exercising muscles arrives at the lungs. Therefore, pulmonary $\dot{V}O_2$ kinetics provides a close approximation of muscle $\dot{V}O_2$ kinetics (Bangsbo, 2000; Barstow et al., 1996; Grassi et al., 1996; Jones & Poole, 2005b; Krustrup et al., 2009). An accurate description of the exponential response profile of phase II forms the basis for $\dot{V}O_2$ kinetics analysis.

The phase II and initial rise in muscle $\dot{V}O_2$ can be described by the following equation:

$$\dot{V}O_2(t) = \dot{V}O_{2baseline} + A_1 (1 - e^{-(t - TD1/\tau 1)})$$

Where $\dot{V}O_2(t)$ is the $\dot{V}O_2$ at any time t, $\dot{V}O_{2baseline}$ is the $\dot{V}O_2$ before onset of exercise, A_1 is the asymptotic amplitude, TD_1 is the time delay preceding the increase in muscle $\dot{V}O_2$, and τ_1 is the time constant.

The time constant (τ) is a measure of the time required for $\dot{V}O_2$ to reach 63% of the final amplitude. At 2 τ $\dot{V}O_2$ attained 86% of the amplitude, at 3 τ 95% and at 4 τ $\dot{V}O_2$ will reach more than 98% of its final amplitude and the final response is completed. Therefore, for a τ of 20 s: 63% of the response amplitude is attained after 20 s; 86% of the response amplitude is attained after 40 s (2 x τ); 95% of the response amplitude is attained after 60 s (3 x τ) and 98% of the response amplitude is attained after 80 s (4 x τ) (Jones & Poole, 2005b), (Figure 3).

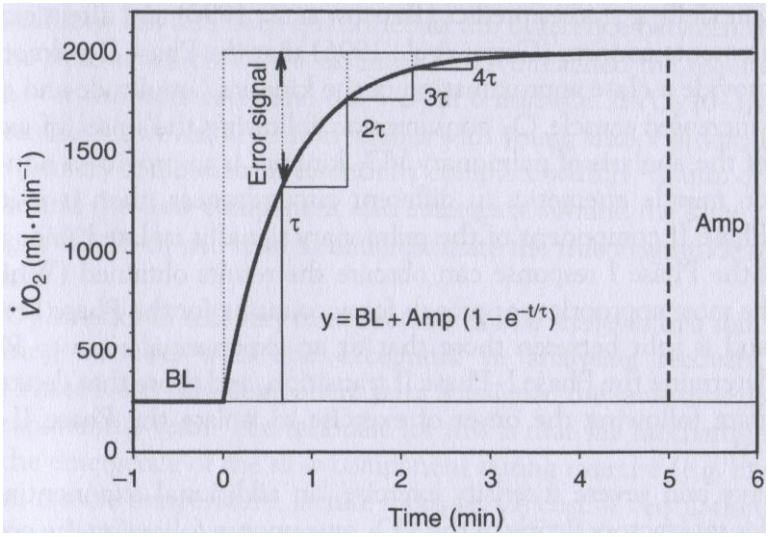

Figure 3: Schematic illustration of the exponential increase in muscle $\dot{V}O_2$ following the onset of exercise. BL = baseline; Amp = amplitude above BL; τ = time constant (Hughes, 2005; Jones & Poole, 2005a)

Lower values in τ (~20 s) represent fast $\dot{V}O_2$ response kinetics, whereas extremely unfit or unhealthy individuals will have a slower response (e.g. $\tau \sim 50$ s).

Phase III - steady state or slow component phase

Phase III is the final phase of $\dot{V}O_2$ kinetic analysis and corresponds to the point at which $\dot{V}O_2$ and cardiac output ($\dot{Q}$) plateaus and venous O_2 content reaches its lowest level (Jones & Poole, 2005a). Depending on health and fitness of the individuals and the exercise intensity a steady-state in $\dot{V}O_2$ will be reached between 2 and 15 or more min. Usually a steady-state at constant work rates with low intensities below gas exchange (GET) or lactate threshold (LT) occurs within 2-3 min. At muscle and pulmonary $\dot{V}O_2$ responses at low to moderate work rates where a steady-state in $\dot{V}O_2$ is reached rather rapidly a close match to the muscle ATP turnover rate is provided (Jones & Poole, 2005a, 2005b).

At higher work rates (above GET or LT) $\dot{V}O_2$ is further rising and leading to a delayed steady-state or $\dot{V}O_2$ is attaining to higher values even to its maximum. Whipp and Wasserman (1972) investigated that with increasing work rates above anaerobic threshold the time to achieve a steady state $\dot{V}O_2$ increases greatly. This $\dot{V}O_2$ increase is the so called SC (Carter et al., 2000a; Ozyener et al., 2001; Scheuermann & Barstow, 2003; Wilkerson et al., 2004). SC typically emerges typically after ~ 90-120 s at higher work rates (Jones & Poole, 2005a). For illustration see Figure 4.

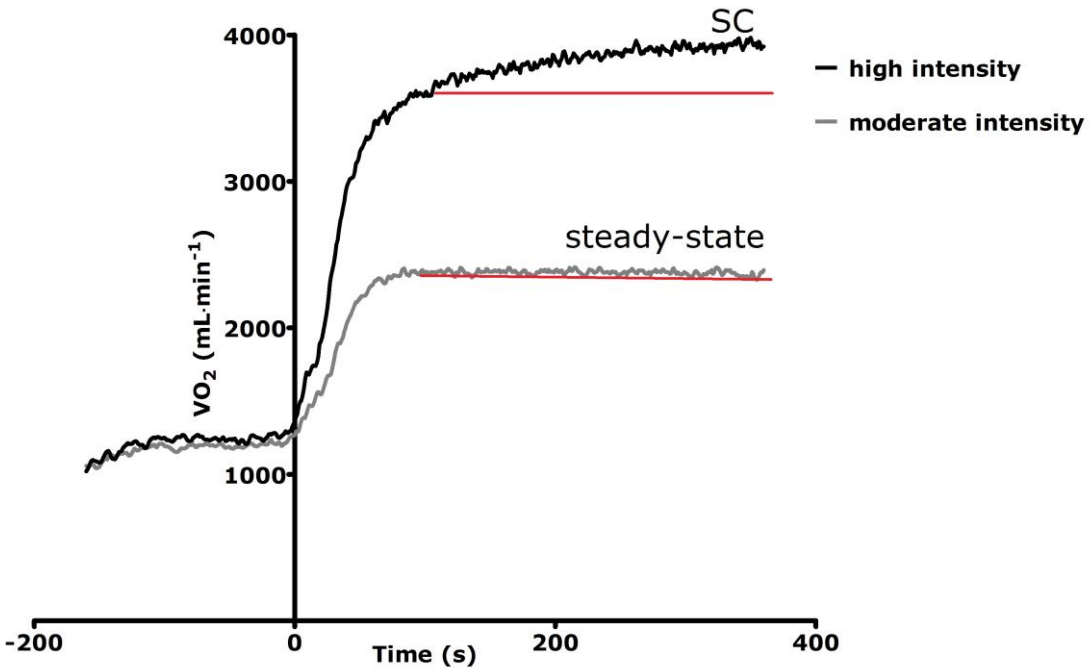

Figure 4: $\dot{V}O_2$ responses at high and moderate intensity constant work rate.

The phase III can be described by the following equation:

$$\dot{V}O_2(t) = \dot{V}O_{2baseline} + A_2 (1 - e^{-(t - TD2/\tau 2)})$$

The $\dot{V}O_2$ SC represents an increasing inefficiency accompanied by a fall in muscle [PCr] and higher glycogen utilization and metabolic accumulation (Bailey, 2010; Krustrup, Hellsten, et al., 2004; Rossiter et al., 2002). That is why faster $\dot{V}O_2$ kinetics or lower $\dot{V}O_2$ SC kinetics can determine the exercise tolerance and are a possible marking for fatigue (Burnley & Jones, 2007; Jones & Burnley, 2009). When exercise is performed between GET and critical power (CP) $\dot{V}O_2$ eventually stabilize at submaximal $\dot{V}O_2$ and the tolerable of exercise is in a range of 2-4 hours (Bailey, 2010; Barstow & Mole, 1991; Linnarsson, 1974; Paterson & Whipp, 1991; Whipp & Mahler, 1980; Whipp & Wasserman, 1972). When the work rate is increased 5% above CP the SC drives $\dot{V}O_2$ to the $\dot{V}O_{2max}$ and blood [lactate] increase (Astrand & Saltin, 1961; Gaesser & Poole, 1996; Poole et al., 1988; Wasserman & Whipp, 1975). As a result Poole et al. (1988) published that the tolerable duration was limited to ~ 18 min in their study.

To characterise the entire response including all three phases a three component exponential model is used (Whipp & Rossiter, 2005):

$$\dot{V}O_2(t) = \dot{V}O_{2baseline} + A_0 (1 - e^{-(t/\tau 0)}) + A_1 (1 - e^{-(t - TD1)/\tau 1}) + A_2 (1 - e^{-(t - TD2)/\tau 2})$$

A three component model describes the $\dot{V}O_2$ kinetics from moderate, heavy and very heavy intensity exercise. A_{TOT} is the end exercise amplitude above BL; A_1 corresponds to the amplitude above BL where phase II attains the onset of SC and A_2 is the difference between A_{TOT} and A_1 (Scheuermann et al., 2001).

The Oxygen-deficit

At the onset of exercise, work rate and also energetic (ATP) demand increases immediately in a square-wave or step-wise fashion, while $\dot{V}O_2$ increases more slowly with an exponential kinetic response (Barstow et al., 1994). A so called oxygen-deficit (Krogh & Lindhard, 1913) occurs at the beginning of exercise between the ATP requirement and the ATP derived from oxidative phosphorylation. The O_2 deficit is determined by τ and the amplitude (A) of $\dot{V}O_2$ response (Whipp & Rossiter, 2005). As a result, if the $\dot{V}O_2$ - work-rate relationship is linear and τ is constant the O_2 deficit can be calculated as:

$$O_2 \text{ deficit} = \text{amplitude} \times \tau$$

Therefore, the O_2 deficit can be determined if A and τ are both known precisely (Linnarsson, 1974; Whipp & Rossiter, 2005; Whipp & Wasserman, 1972).

In order to restore the energy demands in the contracting muscles, the O_2 deficit must be compensated by an increased ATP turnover rate through PCr degradation and anaerobic glycolysis with a small contribution from the O_2 stores in the muscle. Also there will be a small contribution from the O_2 bound to myoglobin at higher intensities. As a result, a bigger O_2 deficit is associated with greater depletion of muscle PCr and a higher production of lactic acid and protons through anaerobic glycolysis. Therefore, faster $\dot{V}O_2$ kinetics can be beneficial during high-intensity exercise and can delay the depletion of muscle high-energy phosphates and the point of fatigue. Likewise faster $\dot{V}O_2$ kinetics would raise the proportional energy contribution from oxidative phosphorylation during high intensive exercise (Bailey, 2010; Jones & Poole, 2005b).

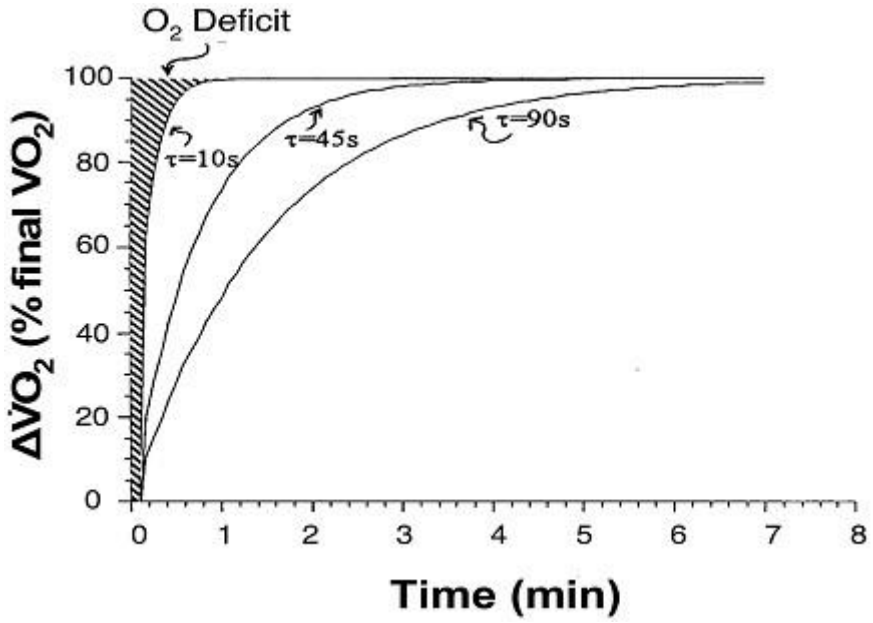

Figure 5: Schematic illustration of the effect of speeding $\dot{V}O_2$ kinetics on the O_2 deficit (Jones & Poole, 2005a).

Figure 5 shows different O_2 kinetics and their influence on the O_2 deficit. With the equation for O_2 deficit and the assumption of a steady state increase in $\dot{V}O_2$ of 1 $L \cdot min^{-1}$ an race horse with a τ of 10 s would gain a O_2 deficit of 0.17 L (1 x 10/60), a sedentary human with a τ of 45 s would gain a O_2 deficit of 0.75 L (1 x 45/60) and a cardiac patient with a τ of 90 s would gain a O_2 deficit of 1.5 L (1 x 90/60) at the same work rate (Prinz, 2014).

1.3 Factors affecting O_2 uptake kinetics and $\dot{V}O_2$ SC

The $\dot{V}O_2$ kinetics is principally influenced upon both O_2 delivery and peripheral O_2 extraction. Therefore, the limiting factors in the pathways of O_2 conductance to the mitochondrion will be determined by the exercise mode, the environment (e.g. altitude) and subject characteristics (e.g. age, training status, disease status) (Bassett & Howley, 2000; Wagner, 2000). Some studies reported that under normal conditions (e.g. normoxia, absence of pathology and no limitations to O_2 delivery) at moderate exercise intensities the $\dot{V}O_2$ response is determined not by O_2 delivery but by local factors within the working muscle cells (Grassi et al., 1996; Poole et al., 2008; Zoladz et al., 2006). Poole et al. (2008) proposed that there are O_2-delivery-dependent and O_2-delivery-independent zones (Koga et al., 2005) during the process of $\dot{V}O_2$ kinetics, whilst Hughes (2005) has argued that O_2 always regulates $\dot{V}O_2$ kinetics.

Poole et al. (1991) reported that the majority of the $\dot{V}O_2$ SC (> 85%) originates from the exercising muscle (Rossiter et al., 2002). In connection with $\dot{V}O_2$ SC a progressive rise in blood [lactate]/[pyruvate], muscle [inorganic phosphate] (P_i) and [H^+] occurs (Jones et al., 2008; Poole et al., 1988).

1.3.1 Influence of muscle fibre type

The skeletal muscle is composed of a mixture of three different main fibre types. Muscle fibres are defined as type I (slow twitch oxidative), type IIA (fast twitch oxidative glycolytic) and type IIX (fast twitch glycolytic). The different fibre types show different functions such as maximal shortening velocity, peak power and fatigue resistance (Bottinelli & Reggiani, 2000; Sieck & Regnier, 2001). Several studies have shown differences in force production, maximal shortening speed and in efficiency (Jones et al., 2005). Numerous studies examined the role of muscle fibre type and muscle activation on $\dot{V}O_2$ SC (Barstow et al., 1996; Cannon et al., 2011; Saunders et al., 2000; Scheuermann & Barstow, 2003; Scheuermann et al., 2001; Vanhatalo et al., 2011).

Studies to investigate the relationship between muscle fibre type and $\dot{V}O_2$ kinetics were carried out by Barstow et al. (1996) and Pringle, Doust, Carter, Tolfrey, Campbell, et al. (2003). The results showed a negative correlation between the percentage of type I fibres with the relative amplitude of the $\dot{V}O_2$ SC and τ during heavy and severe exercise (Figure 6). Pringle, Doust, Carter, Tolfrey, Campbell, et al. (2003) suggested that the correlation between percentage of type I fibres and τ could be linked to higher enzyme activity in type I compared to type II fibres.

Furthermore, Krustrup, Hellsten, et al. (2004) and Krustrup, Soderlund, et al. (2004) investigated that a strong involvement of type II fibres causes $\dot{V}O_2$ SC (Poole & Jones, 2005). The recruitment of fibres with low oxidative capacity (typ II fibres) at high work rates might also have an impact upon the $\dot{V}O_2$ kinetic response in phase II (Brittain et al., 2001; Jones et al., 2005; Koppo et al., 2004; Pringle, Doust, Carter, Tolfrey, Campbell, et al., 2003; Pringle, Doust, Carter, Tolfrey, & Jones, 2003). Additionally, one reason for slower $\dot{V}O_2$ kinetics in fast compared to slow muscle fibres might be slower blood flow dynamics in typ II than in typ I fibres (Behnke et al., 2003). Also the O_2 cost of recovery processes in fatigued fibres, could play a role in development of the $\dot{V}O_2$ SC (Vanhatalo et al., 2011).

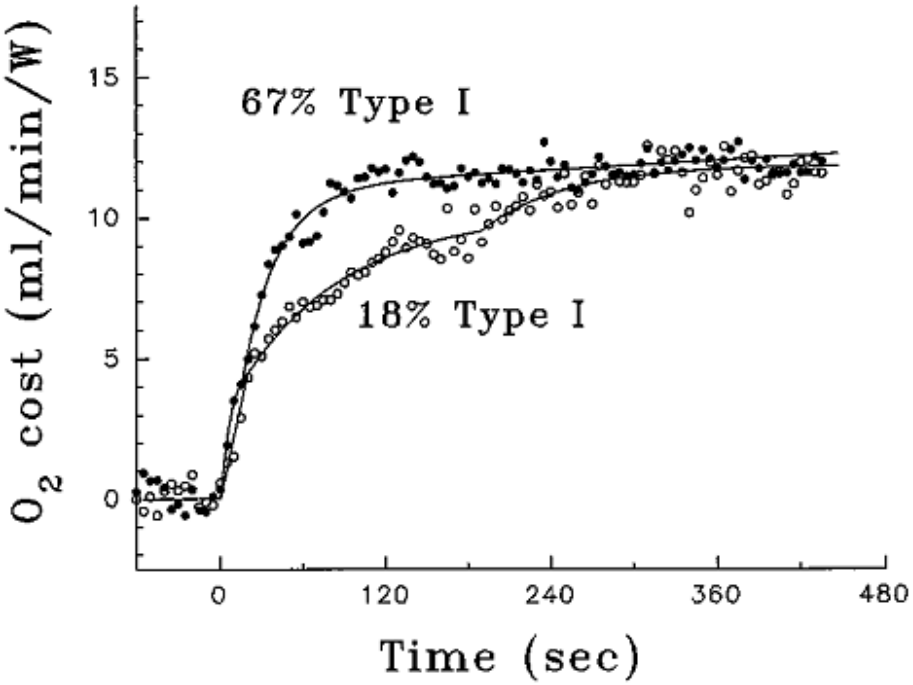

Figure 6: $\dot{V}O_2$ responses to heavy exercise in subjects with a high percentage and a low percentage of type I fibres (Barstow et al., 1996).

1.3.2 Influence of training

As discussed above, slower $\dot{V}O_2$ kinetic is associated with a greater depletion of [PCr], greater accumulation of blood [lactate] and hydrogen ions (H^+) and a greater O_2 deficit. Jones and Koppo (2005) examined the effect of training on $\dot{V}O_2$ kinetics using cross-sectional and longitudinal studies. They compared trained and untrained subjects and tried to explain physiological adaptions in response to training. Cross-sectional studies have investigated a faster $\dot{V}O_2$ response (i.e. smaller τ, faster steady-state attainment) in trained than in untrained subjects (Barstow & Mole, 1991; Borrani et al., 2001; Caputo et al., 2003; Carter et al., 2000a; Cerretelli et al., 1979; Cleuziou et al., 2003; Koppo et al., 2004; Marwood et al., 2010; Powers et al., 1985; Russell et al., 2002; Weltman & Katch, 1976; Zhang et al., 1991). For illustration, Figure 7 shows that the τ of trained subjects is significantly lower compared to untrained subjects.

Powers et al. (1985) examined that $\dot{V}O_{2max}$ is related to $\dot{V}O_2$ kinetics in athletes with similar training programs. Athletes with higher $\dot{V}O_{2max}$ values showed faster τ values (Jones & Koppo, 2005).

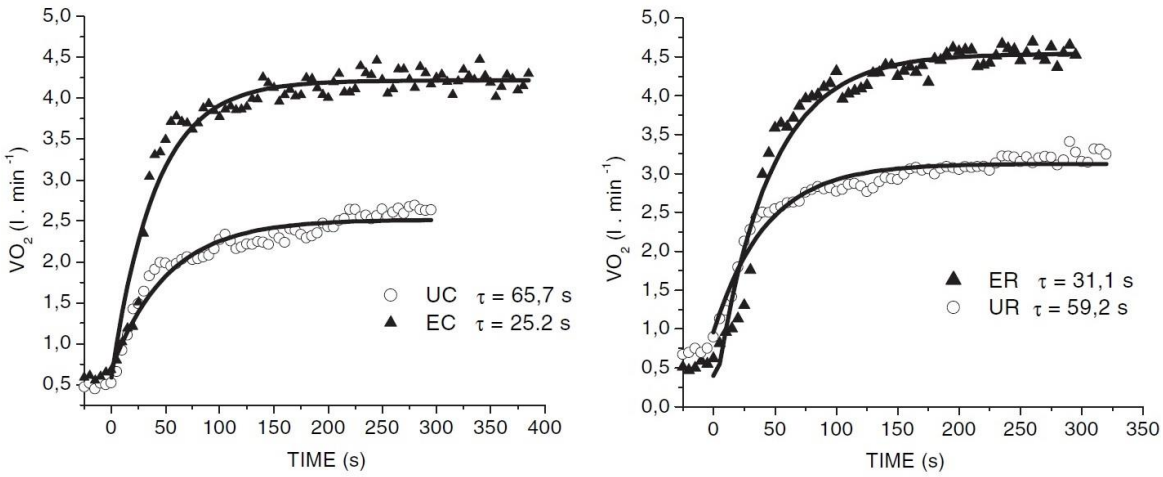

Figure 7: Comparison of untrained (UC and UR) and endurance-trained (EC and ER) cyclists and runners on $\dot{V}O_2$ kinetics (Caputo et al., 2003).

Longitudinal studies have shown training induced reductions in τ of phase II during both moderate- and heavy-intensity exercise (Babcock et al., 1994; Bell et al., 2001; Berger et al., 2006; Fukuoka et al., 2002; Hagberg et al., 1980; Hickson et al., 1978; Phillips et al., 1995; Yoshida et al., 1992). Endurance training leads to physiological adaptions of cardio-vascular and metabolic functions and will influence aerobic function parameters like $\dot{V}O_{2max}$, efficiency or exercise economy, CP, $\dot{V}O_2$ kinetics and lactate threshold (Burnley & Jones, 2007; Jones & Carter, 2000).

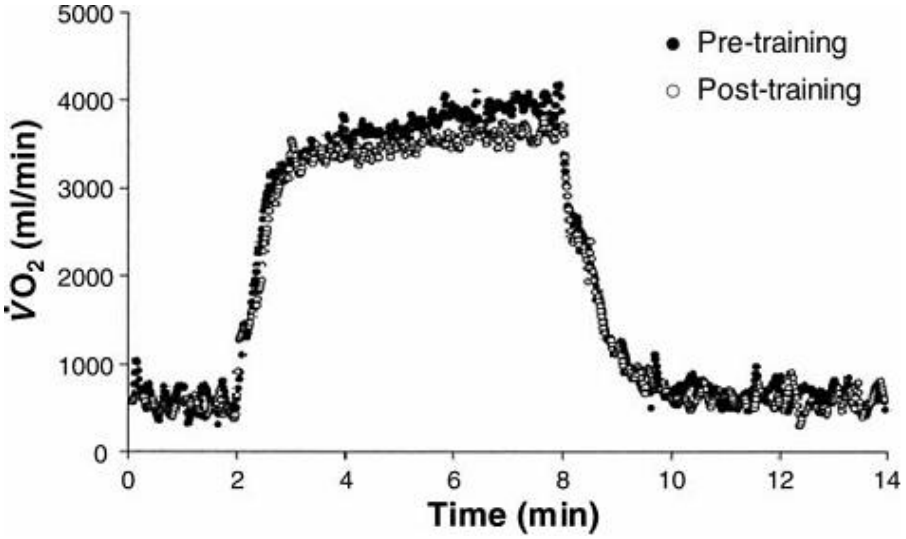

Figure 8: Effect of 6-week running training on $\dot{V}O_2$ kinetics (Carter et al., 2000a).

Phillips et al. (1995) reported a reduced τ from 37.2 s to 28.8 s (after 4 days training) and then further to 15.8 s (after 30 days training). In the study of Carter et al. (2000a) the amplitude of $\dot{V}O_2$ SC was significantly reduced with 6-week running training by ~

100 mL·min^{-1} (Figure 8). At the same time $\dot{V}O_{2max}$ and lactate threshold showed only small changes. These results illustrates that enhanced fitness levels leads to faster $\dot{V}O_2$ kinetics and a reduced O_2 deficit (Figure 5).

1.3.3 Influence of cadence

A number of studies demonstrated that the recruitment of typ II muscle fibres is enhanced with higher pedal rate for the same work rate (Beelen & Sargeant, 1993; MacIntosh et al., 2000; Sargeant, 1994, 1999). Furthermore, they reported that subjects tend to use higher pedal rates at heavy exercise compared to low work rates. Therefore, different pedal rates at the same power outputs have been used for studying the effects of fibre-type recruitment (Pringle, Doust, Carter, Tolfrey, & Jones, 2003). Studies have shown a faster τ at low pedal rates (35 – 75 rev·min^{-1}) compared to high pedal rates (90 – 115 rev·min^{-1}) at heavy intensities (Barstow et al., 1996; Zoladz et al., 1998). For illustration see Figure 9. Furthermore, the amplitude of $\dot{V}O_2$ SC and blood [lactate] was significantly higher at 115 compared to 35 rev·min^{-1} but there was no significant difference in $\dot{V}O_2$ gain (Barstow et al., 1996; Pringle, Doust, Carter, Tolfrey, & Jones, 2003) (Figure 10).

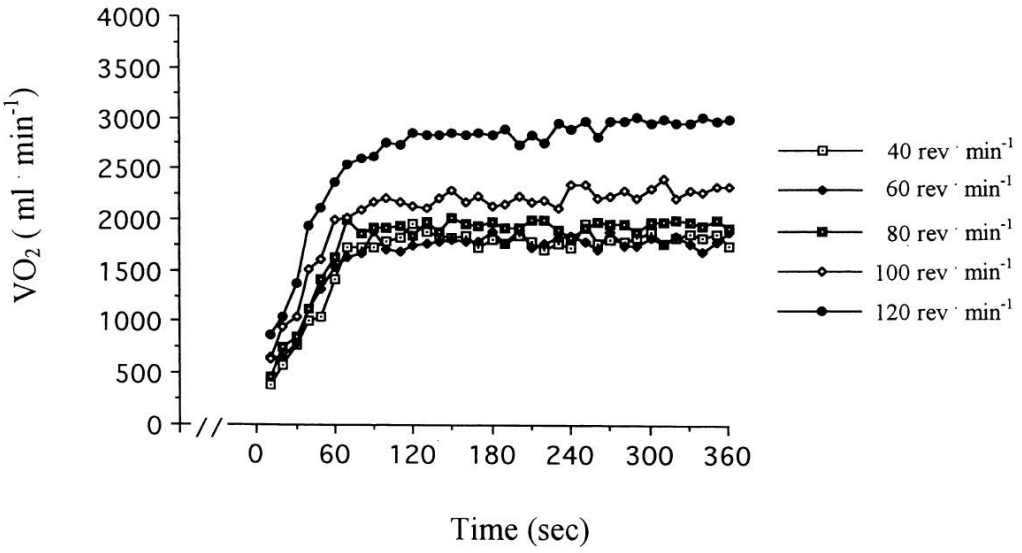

Figure 9: $\dot{V}O_2$ response during cycling at constant work rates (50% $\dot{V}O_{2max}$) (Zoladz et al., 1998).

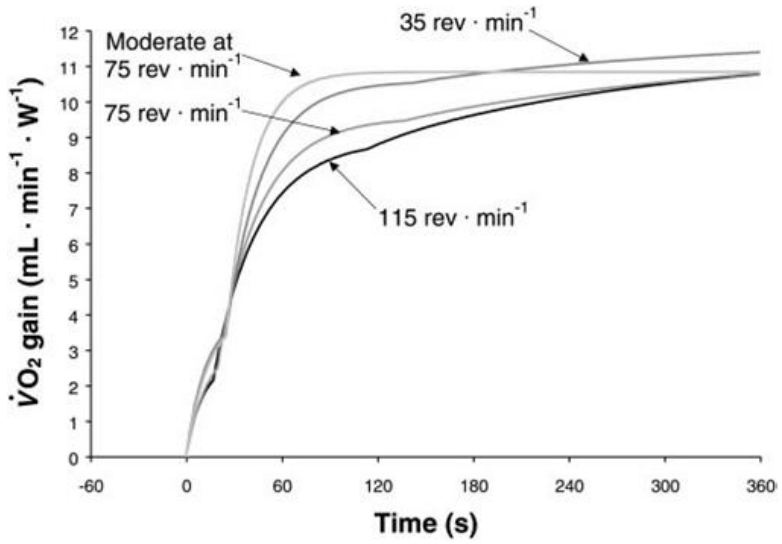

Figure 10: $\dot{V}O_2$ kinetics (primary gain) at heavy exercise at 35, 75 and 115 rev·min^{-1}. For comparison the $\dot{V}O_2$ response at moderate intensity at 75 rev·min^{-1} is shown (Pringle, Doust, Carter, Tolfrey, & Jones, 2003).

In some studies a reduction in the primary $\dot{V}O_2$ gain with higher pedal rates has been observed both at the same relative work rate or as the power output was increased (Barstow et al., 1996; Carter et al., 2000b; Carter et al., 2002; Jones et al., 2002; Ozyener et al., 2001).

1.4 Intensity domains

A number of exercise intensity domains have been identified (Jones & Poole, 2005a; Whipp & Rossiter, 2005). Discovery of intensity-specific differences in the metabolic and gas exchange responses during constant work rate exercise showed that the commonly used practice of defining work rates as a percentage of $\dot{V}O_{2max}$ was not appropriate for normalising intensities between subjects or under changing conditions within the same subject (DiMenna, 2010). Parameters used for determining intensity domains are gas exchange threshold (GET) or lactate threshold (LT), critical power (CP) (Moritani et al., 1981), primary response $\dot{V}O_{2peak}$ and $\dot{V}O_{2max}$. Whilst exercise intensities for moderate intensity are given as a percentage of GET (e.g. 90% GET), for heavy, severe or extreme intensity the delta intensity (Δ = difference between GET and $\dot{V}O_{2max}$) has been used (DiMenna, 2010). The most commonly used schemata are illustrated in Figure 11. Schema A is used throughout this thesis.

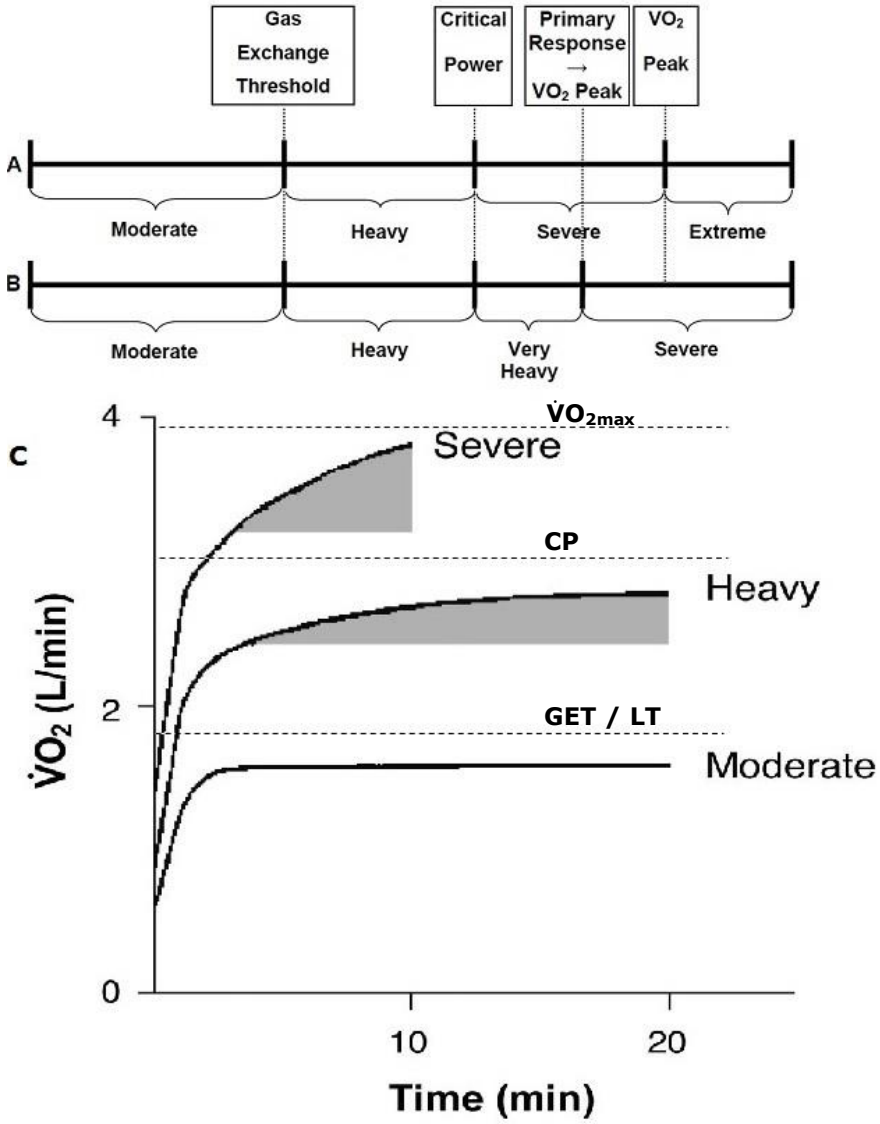

Figure 11: Three commonly used schemata to categorize exercise intensity domains. Schemata A and B (DiMenna, 2010) and scheme C adapted from Jones and Poole (2005b).

Moderate-intensity exercise

Moderate-intensity exercise defines all work rates that usually can be sustained for long periods with a modest sense of effort. Therefore, moderate-intensity work rates are below GET and accompanied by a steady state in $\dot{V}O_2$ attained within 3 minutes (J. E. Hansen et al., 1984; Wasserman & Whipp, 1975; Whipp & Mahler, 1980). At moderate intensity constant work rate exercises, blood [lactate] is not elevated and the typical cost in $\dot{V}O_2$ is 9-11 $mL \cdot min^{-1} \cdot W$ (Jones & Poole, 2005a).

Heavy-intensity exercise

Heavy-intensity exercise indicates work rates between the GET and below CP where the oxidative cost is greater (~13 $mL \cdot min^{-1} \cdot W$) and attainment of steady state $\dot{V}O_2$ is delayed due to $\dot{V}O_2$ SC (Barstow & Mole, 1991; Linnarsson, 1974; Paterson & Whipp, 1991; Roston et al., 1987; Whipp & Mahler, 1980; Whipp & Wasserman, 1972). Blood lactate and $\dot{V}O_2$ steady state will be attained at an elevated level above baseline after a considerable delay as much as 10-15 min or more in extreme (Whipp & Rossiter, 2005). The upper boundary (i.e. CP) for this domain is defined as the maximal intensity at which blood [lactate] and $\dot{V}O_2$ can be stabilized (i.e. maximal lactate steady state (MLSS)) (Pringle & Jones, 2002; C. G. Smith & Jones, 2001).

Severe-intensity exercise

Severe-intensity exercise work rates are lying between CP and $\dot{V}O_{2max}$ and can only be tolerated for a few minutes (~ 10-20 min). Furthermore this domain will also be characterized by the presence of a $\dot{V}O_2$ SC that drives $\dot{V}O_2$ to its maximum and blood [lactate] rises inexorably until the end of exercise (Astrand & Saltin, 1961; Gaesser & Poole, 1996; Poole et al., 1988; Whipp & Rossiter, 2005). To investigate exercise response in this domain intensities of 60-80% Δ are typically used (DiMenna, 2010).

During heavy- and severe-intensity exercise the primary $\dot{V}O_2$ kinetics might be slowed relative to that observed in moderate exercise (Jones et al., 2002; Jones & Poole, 2005a; Ozyener et al., 2001; Pringle, Doust, Carter, Tolfrey, Campbell, et al., 2003; Rossiter et al., 2002; Scheuermann & Barstow, 2003; Whipp & Rossiter, 2005).

Extreme-intensity exercise

During extreme-intensity exercise the ability to sustain exercise is so limited that fatigue intervenes before $\dot{V}O_{2max}$ can be achieved (Jones & Poole, 2005a). The tolerable duration of exercise is usually < 140 s (i.e. ~4 times a τ of ~30 s.) (D. W. Hill et al., 2002). There is also no $\dot{V}O_2$ SC that can be discerned.

2. Efficiency

The mechanical efficiency during steady-state cycling is defined as the ratio of mechanical work accomplished to the metabolic energy expenditure (EE) required to do that work (Gaesser & Brooks, 1975) and has been suggested to be a key determinant of cycling performance (Coast et al., 1986; Coyle, 1995, 1999; Horowitz et al., 1994; Joyner & Coyle, 2008; Moseley & Jeukendrup, 2001; Olds et al., 1995). Measuring the external work during cycling is an accurate method where energy consumption is calculated from O_2 uptake. Hopker, Jobson, et al. (2012) demonstrated that the Douglas bag method provides the most reliable measurement of cycling efficiency. Nevertheless in most studies that investigated mechanical efficiency in cycling online expired gas analysis systems were used (Ettema & Loras, 2009).

The mechanical efficiency provides an index of effectiveness with which a person can convert chemical energy into mechanical power (Barbeau et al., 1993; Gaesser & Brooks, 1975; Hagberg et al., 1981; Hopker, Passfield, et al., 2009; Kunstlinger et al., 1985; Nickleberry & Brooks, 1996). Commonly used indices of efficiency are net efficiency, work efficiency, delta efficiency and gross efficiency (Hopker, Passfield, et al., 2009; Nimmerichter, 2011).

2.1 Net efficiency

Net efficiency (NE) shows the energy consumption in the muscle contraction itself and therefore all energy cost that is not related to work production is subtracted from the total amount of EE (baseline-subtraction) (Gaesser & Brooks, 1975; Stainbsy et al., 1980).

The baseline subtraction assumes that all processes related to resting metabolism are independent, constant (Stainbsy et al., 1980) and isolated from the process of doing work. That suppose that two independent energy flows are working parallel, while not effecting each other. It has been criticised that NE assumes the baseline to be unaffected at higher work rates because various processes are altered at higher work rates (e.g. gastrointestinal, blood flow, cardiac output and ventilation) and thus will influence the baseline energy expenditure (Cavanagh & Kram, 1985; Stainbsy et al., 1980). The assumed resting metabolic rate is affected by changes in the energy consumption that are used to maintain homeostasis during exercise (Moseley & Jeukendrup, 2001).

NE is described by following equation:

$$\mathrm{NE} = \frac{\text{work rate}}{\text{metabolic rate} - \text{rest metabolic rate}} \times 100$$

2.2 Work efficiency

The definition of work efficiency (WE) is based on the assumption that the metabolic rate for unloaded cycling is not utilized for doing external work. It is defined as work rate divided by the difference in energy expenditure of unloaded exercise to that of the current exercise (Whipp & Wasserman, 1969). Where metabolic rate for unloaded cycling is the sum of the rest metabolic rate and the metabolic rate required for unloaded cycling.

$$\mathrm{WE} = \frac{\text{work rate}}{\text{metabolic rate} - \text{metabolic rate unloaded}} \times 100$$

Gaesser and Brooks (1975) proposed to replace the value of unloaded cycling with the y-intercept of the work rate –efficiency relationship and the general work rate –EE relationship, because of the deviation of $\dot{V}O_2$ for unloaded cycling.

$$\mathrm{WE} = \frac{\text{work rate}}{\text{metabolic rate} - \text{y intercept}} \times 100$$

The calculation of WE accounts the additional costs of moving legs but the measurement of unloaded cycling is criticized. Kautz and Neptune (2002) have debated that internal work is not related to doing external work and therefore unloaded cycling does not provide a valid baseline for reference to a range of work rates. There is always some energy loss because some energy is converted into heat. Also Ettema and Loras (2009) criticized the use of WE as a measure of muscular efficiency because a summation of internal and external work is impossible. The interaction at different time between the different energy deposits and work transitions and how they are linked to each other make it impossible to summarize internal (e.g. elastic energy) and external energy production costs. There is only information about one of the two deposits and furthermore no information about the direct transition of muscle work to external work Figure 12.

However, it seems difficult to experimentally determine the energy cost of true internal work (i.e. work that never appears as external) in loaded cycling (Ettema & Loras, 2009).

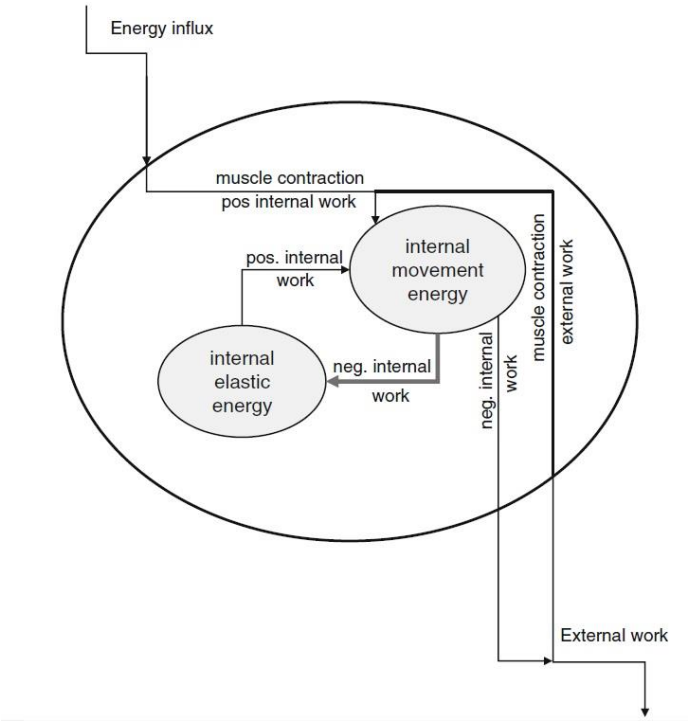

Figure 12: Model for energy flow during exercise. The terms positive (pos) and negative (neg) indicate the direction of energy flow (Ettema & Loras, 2009).

2.3 Delta efficiency

The calculation of delta efficiency (DE) is based on the assumed linear relationship between work rate and energy cost or the change in EE in relation to the change in work rate (Hopker, Passfield, et al., 2009).

$$\mathrm{DE} = \frac{\Delta \text{ work rate}}{\Delta \text{ metabolic rate}} \times 100$$

Coyle et al. (1992) assumed that DE is the most valid calculation of whole body efficiency. But in some other studies researchers recommended that the use of DE may be limited (Cavanagh & Kram, 1985; Kautz & Neptune, 2002; Stainbsy et al., 1980). Firstly the linear relationship between energy cost and work rate is not true and therefore, it does not mean that it provides a valid measure (Ettema & Loras, 2009;

Hopker, Passfield, et al., 2009). Furthermore, the use of DE may also be limited due to a greater day-by-day variability than gross efficiency (Hopker et al., 2007; Moseley & Jeukendrup, 2001).

NE, WE and DE calculation use baseline subtraction methods. Due to various criticisms it was conducted that for whole body movements definitions of efficiency involving baseline subtractions lead to invalid measures of efficiency. Therefore, gross efficiency calculation will be used during this master thesis.

2.4 Gross efficiency

Gross efficiency (GE) is the most commonly used measure of efficiency for whole body exercise and is expressed as percentage of EE. GE is defined as the ratio between the work accomplished and the EE (van Ingen Schenau & Cavanagh, 1990). GE is reported usually to be in a range of 15-25% during cycling (Coyle et al., 1992; Gaesser & Brooks, 1975).

$$\mathrm{GE} = \frac{\text{work accomplished}}{\text{energy expenditure}} \times 100$$

EE during exercise can be determined using the caloric equivalent from the measurement of steady-state $\dot{V}O_2$ and the respiratory exchange ratio (RER) (Peronnet & Massicotte, 1991), or from the calculation of $\dot{V}O_2$ and $\dot{V}CO_2$ (Brouwer (1957) in Moseley and Jeukendrup (2001)). Throughout this master thesis EE is estimated according to Brouwer (1957):

$$\mathrm{EE} = \left[\, 3.869\ \dot{V}\mathrm{O}_2 + 1.195\ \dot{V}\mathrm{CO}_2 \,\right] \times \left(\frac{4.186}{60}\right) \mathrm{x}\ 1000$$

2.5 Factors affecting gross efficiency

Hopker, Passfield, et al. (2009) considered several factors affecting GE like power output, muscle fibre type distribution, pedal rate, aging, gradient and training status. Furthermore, factors like riding position, alterations in seat tube angle and saddle height (Heil et al., 1995; Price & Donne, 1997), muscle damage from high intensity training (Kyrolainen et al., 2000; Twist & Eston, 2009), high ambient temperatures (35.5 °C)

(Hettinga et al., 2007) and the gradient of terrain (Arkesteijn et al., 2013). The most relevant factors for this master thesis will be discussed below.

2.5.1 Influence of test protocol

To measure GE accurately the whole gas collection has to take place under steady-state exercise conditions, otherwise measured pulmonary $\dot{V}O_2$ may not reflect muscle O_2 consumption (Hopker, Passfield, et al., 2009; Poole et al., 1992). A steady-state during light to moderate exercise (i.e. RER < 1.0) can be reached within 2-3 min (Whipp & Wasserman, 1972). Additional O_2 consumption or SC should not be used to calculate GE. During determination of GE it has also to take into account that calculated EE depends on the RER (i.e. a decrease of 0.05 in RER reduces EE by 1.3% and increases GE by ~0.4%). As a result as $\dot{V}CO_2$ affects RER it is important to ensure its stability before starting GE measurements and therefore it is important to take into account that a steady state in $\dot{V}CO_2$ occurs after about 4 min (Chuang et al., 1999; Hopker, Coleman, et al., 2009; Wasserman et al., 2005). For this reason, longer work stages (≥ 5 min) should be used to determine GE (Hopker, Passfield, et al., 2009).

Even though GE measures should be based on steady state conditions, de Koning et al. (2012) reported that some studies published efficiency results from incremental protocols with short stage durations. Therefore, they examined the effect of different stage durations on GE during an incremental test. Their results show significant higher GE at the 1 min stage compared to 3 and 6 min stages and GE is rising with higher work rates (Figure 13).

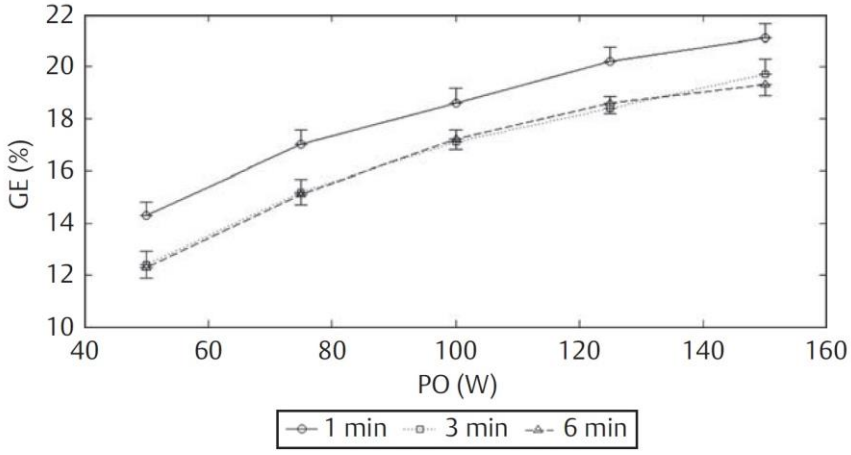

Figure 13: GE determined during stages of 1, 3, and 6 min. Also significant effects between different power outputs (PO) are visible (de Koning et al., 2012).

Further studies of shorter work stage durations of 2 min (Barbeau et al., 1993) and 3 min (Mora-Rodriguez & Aguado-Jimenez, 2006; Moseley et al., 2004; Samozino et al.,

2006) have been found in the literature. Longer stage durations have been reported by Cannon et al. (2007) and Lucia et al. (2004) with 6 min, Hopker, Coleman, et al. (2009) with 8 min and Hettinga et al. (2007) with 20 min.

2.5.2 Influence of training status

Some studies investigated the influence of the training status on efficiency and reported that GE is not responding to endurance training (Marsh & Martin, 1993; Marsh et al., 2000; Mogensen et al., 2006; Moseley et al., 2004; Nickleberry & Brooks, 1996). Contrary, Horowitz et al. (1994) investigated that cyclists with higher GE are able to produce higher power outputs during a 1-hour time trial (TT) performance than cyclists with a lower GE at the same $\dot{V}O_2$. These findings are similar to the results from Lucia et al. (1998), who reported higher power outputs from professional cyclists than amateur riders at similar $\dot{V}O_{2max}$ values.

Hopker et al. (2013) reported that a careful examination of previous studies suggests that a lack of statistical power may hide meaningful differences in GE between trained and untrained cyclists.

In their studies they investigated that cycling efficiency is higher in trained cyclists (Hopker, Coleman, et al., 2009; Hopker et al., 2010; Hopker et al., 2013; Hopker et al., 2007). An increase in GE is associated with a higher performance in endurance competitions because a lower percentage of $\dot{V}O_{2max}$ at any exercise intensity is needed and therefore a reduction in muscle glycogen utilisation occurs (Nimmerichter, 2011). Findings in longitudinal study designed researches showed that endurance training increases GE over short-term (Hopker et al., 2010), single (Hopker, Coleman, et al., 2009) or multiple (Santalla et al., 2009) cycling seasons. Figure 14 shows changes in GE across different phases of the season. Furthermore the study of Coyle (2005) demonstrated an 8% improvement in efficiency with no significant changes in $\dot{V}O_{2max}$ over seven years in a Tour de France winner. Hopker, Coleman, et al. (2012) concluded that in moderate-highly trained cyclists, GE increases with training but not $\dot{V}O_{2max}$. Furthermore, subjects with higher $\dot{V}O_{2max}$ seemed to be more responsive to training induced changes in GE compared to athletes with lower $\dot{V}O_{2max}$. With the help of an mathematical model Moseley and Jeukendrup (2001) calculated that an increase of 1% in GE will result in a 63 s advantage in a 40 km TT.

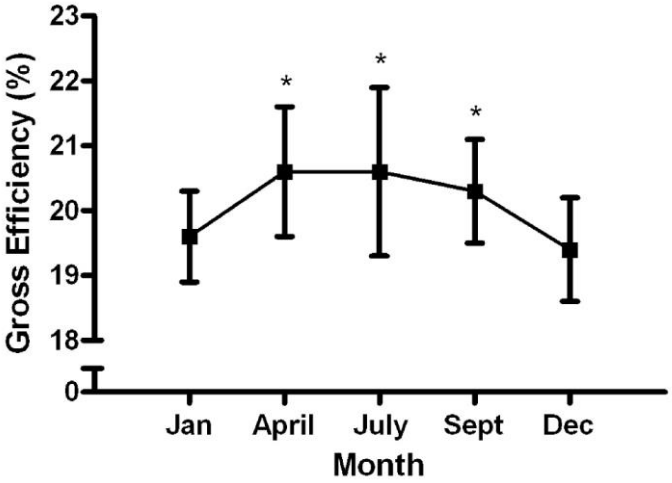

Figure 14: Graphic of the mean GE values across different phases of the season. *Significantly higher than test Jan and test Dec (Hopker, Coleman, et al., 2009).

The changes in GE strongly correlated with total training time and the amount of training at higher exercise intensities (Hopker, Coleman, et al., 2009; Nimmerichter, 2011). For illustration see Figure 15. Potential mechanisms which might be responsible for training induced increases in GE include typ I muscle fibre transformation (Coyle et al., 1992; Horowitz et al., 1994), adaptions in enzymatic profiles (Coyle et al., 1991), mitochondrial biogenesis (Holloszy & Coyle, 1984; Hood & Saleem, 2007) and capillary density (Zoladz et al., 2005).

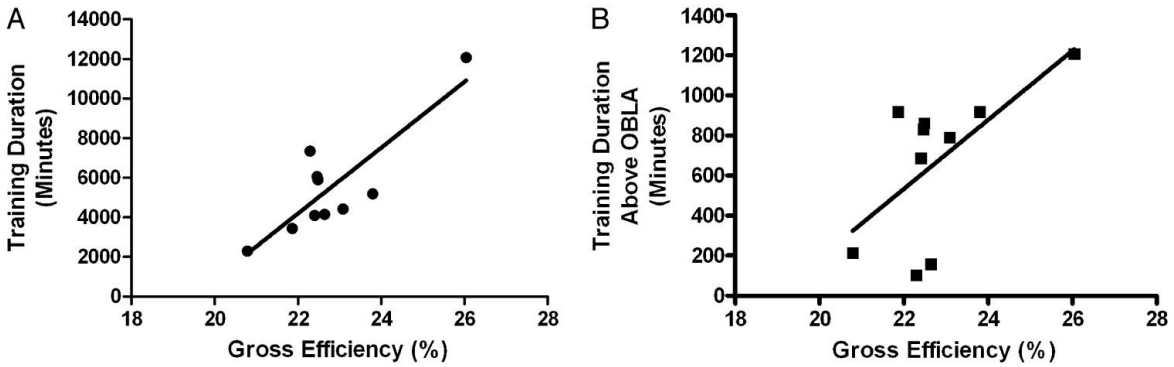

Figure 15: Correlations between GE and total training time (A) and training at higher intensities (B) (Hopker, Coleman, et al., 2009).

GE is a key determinant of fractional utilisation of $\dot{V}O_{2max}$ which determines performance therefore Hopker, Coleman, et al. (2009) suggest that GE should be part of the routine testing program for competitive cyclists.

2.5.3 Influence of work rate and cadence

Many studies have examined that GE is affected by work rate and cadence (Chavarren & Calbet, 1999; Ettema & Loras, 2009; Gaesser & Brooks, 1975; Samozino et al., 2006; Sidossis et al., 1992).

Figure 16 shows the decreases in GE at any given work rate with increasing cadence and increases in GE at any given cadence with increasing power output (Samozino et al., 2006). Based on this findings Chavarren and Calbet (1999), Gaesser and Brooks (1975) and Nickleberry and Brooks (1996) has been conclude that the optimal and most efficient cadence is around 50 rev·min^{-1}. This differs from the cadence that professional cyclists prefer during training and competitions (i.e. 80-100 rev·min^{-1}) (Lucia et al., 2001; Rossato et al., 2008; Sassi et al., 2009). It has been shown that the cadence choice is influenced by a few factors like age (Sacchetti et al., 2010), cycling experience (Chapman et al., 2008; Marsh & Martin, 1997), terrain (Lucia et al., 2001; Sassi et al., 2009) and exercise duration (Argentin et al., 2006). Another reason for choosing higher cadences is that cyclists try to reduce the forces applied to the cranks and minimise neuromuscular fatigue (i.e. is higher for pedalling with low cadences) instead of riding at energetically optimal cadences (Vercruyssen & Brisswalter, 2010).

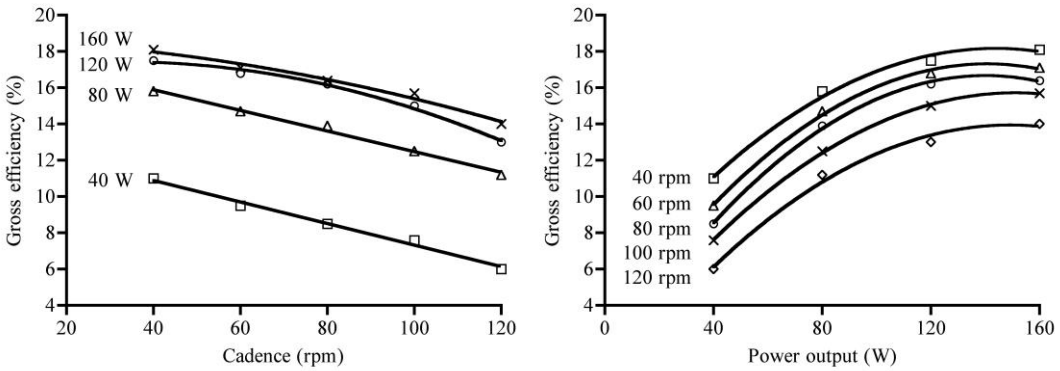

Figure 16: Illustration of GE at different power output and different cadence (Samozino et al., 2006).

In trained cyclist the most efficient cadence is increasing from 50 rev·min^{-1} to 80 rev·min^{-1} when power output is increasing from 100 W to 300 W (Coast & Welch, 1985). In addition Foss and Hallen (2004) examined similar results whereas the optimal cadence for low work rates (< 125 W) was found at 60 rev·min^{-1} and for higher power outputs (350 W) at 80 rev·min^{-1}.

2.5.4 Influence of gradient

Just a few studies investigated the influence of gradient on GE. Millet et al. (2002) and E. A. Hansen et al. (2002) report that GE does not change when comparing level ground and uphill cycling condition. Also Tanaka et al. (1996) measured GE during uphill cycling at different gradients (4% and 8%) at predetermined speeds. However, in all three studies the variations in cadence or power output were too high to get valid results (Arkesteijn et al., 2013). The main finding in the study of Arkesteijn et al. (2013) showed a decrease in GE during cycling on 8% compared to 0% and 4% gradients at the same cadence and power output. As a result GE was lower during uphill cycling compared to level ground cycling (Figure 17).

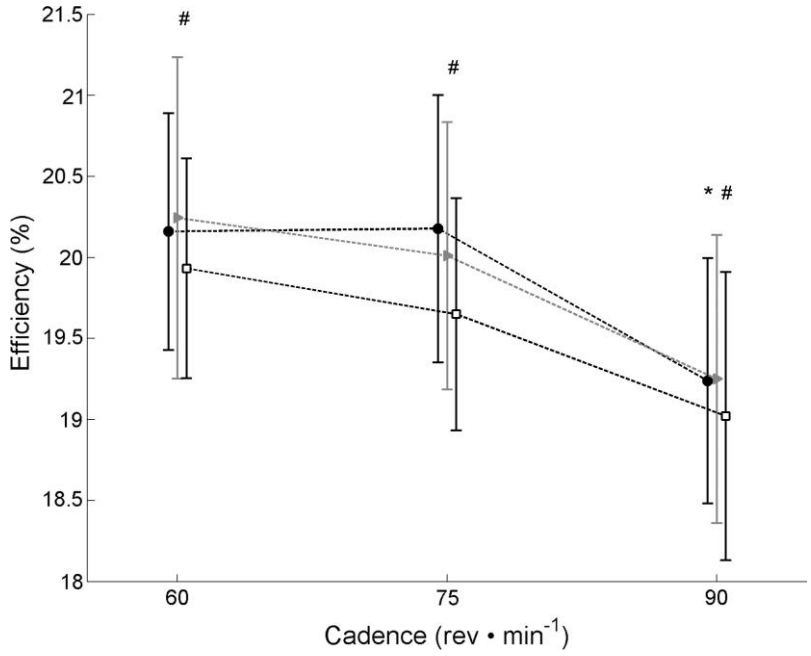

Figure 17: The effect of gradient and cadence on GE. GE at 0% = black circles; 4% = gray circles and 8% = white circles (Arkesteijn et al., 2013).

2.6 Summary and purpose of the study

This review has summarized the physiological responses of oxygen uptake at the onset of exercise. It has been shown that various factors such as cadence, exercise intensity, training status or the test protocol affect both oxygen uptake kinetic and efficiency. Consequently, GE and O_2 uptake kinetic should be determined in standardised conditions in this study. It has also been shown that the influence of gradient is currently rarely observed. Therefore, the general aim of this study was to examine the influence of cadence, exercise intensity and gradient on parameters of the $\dot{V}O_2$ uptake kinetics and GE in laboratory conditions with well-trained cyclists.

The following hypotheses have been addressed:

1. There will be significant differences in O_2 kinetics parameters between different intensities.
2. There will be significant differences in O_2 kinetics parameters between different cadences.
3. There will be significant differences in GE between different cadences.
4. There will be significant differences in O_2 kinetics parameters between level and simulated uphill cycling.
5. There will be significant differences in GE between level and simulated uphill cycling.

3. Material and Methods

3.1 Participants

A total of thirteen well-trained male competitive cyclists participated in this study (mean ± SD age 23.0 ± 4.7 years; height 178.5 ± 5.2 cm; body mass 69.0 ± 7.8 kg; $\dot{V}O_{2max}$ 68.2 ± 4.7 $mL \cdot min^{-1} \cdot kg^{-1}$). All athletes raced previous years in road races, mountain bike races or triathlons. All subjects followed their common training program and competed at races during the test period. Before testing, all participants were informed of the experimental procedures and completed a health questionnaire. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and by the institutional review board. The participants provided written informed consent before entering the study.

3.2 Study Design

Subjects visited the laboratory at two different occasions. The first test day participants performed, one laboratory incremental graded exercise test (GXT) to exhaustion for determination of ventilatory threshold (VT) and respiratory compensation point (RCP).

On a second day, the participants underwent 8 test-trials of 6 min duration at a constant power and given cadence. For simulating uphill condition the test bike was mounted on an indoor trainer was fixed on a treadmill.

Figure 18: Example for the test trials.

During the test-trials 4 level ground and 4 uphill trials were completed. The order of these trials was 90%VT at 60 $rev \cdot min^{-1}$, Δ70 at 60 $rev \cdot min^{-1}$, 90%VT at 90 $rev \cdot min^{-1}$, Δ70 at 90 $rev \cdot min^{-1}$. Between each test-trial a rest period of 45 minutes was set.

The participants were asked to refrain from caffeine, alcohol and from strenuous exercise the day before the tests and were instructed to follow a carbohydrate-rich diet and to drink at least 3 litres.

3.3 Laboratory Incremental Graded Exercise Test

For measuring maximum oxygen uptake ($\dot{V}O_{2max}$), power output (P_{max}), blood [lactate] and heart rate (HR_{max}) a GXT was performed on an electromagnetically braked ergometer (Lode Excalibur, Groningen, The Netherlands) at a cadence of 90-100 $rev \cdot min^{-1}$. Furthermore, the ventilatory threshold (VT) and the respiratory compensation point (RCP) were determined as sub-maximal performance correlates. The ergometer was equipped with a drop handlebar, a race saddle and the participants own pedals.

After a 3-min standardised warm up at 40 W the initial workload was increased by 20 $W \cdot min^{-1}$ until exhaustion. To calculate the maximal power the method of Kuipers et al. (1985) was used, if the last work rate was not completed:

$$P_{max} = PL + (t/60 \times 20)$$

where PL is the last completed work rate (W) and *t* is the time for the incomplete work rate (s).

Oxygen uptake was measured continuously throughout the test via breath-by-breath open circuit spirometry (MetaMax 3B, Cortex Biophysik, Leipzig, Germany). Before each test, gas analysers were calibrated with gases of known concentrations (4.99 Vol% CO_2, 15.99 Vol% O_2, Cortex Biophysik, Leipzig, Germany). Volume and flow were calibrated with a 3-L syringe (Type M 9474-C; Cortex Biophysik, Leipzig, Germany). The subjects wore a facemask and breathed through a low-resistance impeller turbine.

Achievement of $\dot{V}O_{2max}$ was assumed as the highest 30 seconds value attained before volitional exhaustion. Determination of VT the criteria of an increase of the ventilatory equivalent of O_2 ($VE/\dot{V}O_2$), without a concomitant increase of the ventilatory equivalent of CO_2 ($VE/\dot{V}CO_2$) and the first loss of linearity in pulmonary ventilation (VE) and carbon dioxide ventilation ($\dot{V}CO_2$) were used (Beaver et al., 1986). Respiratory compensation point (RCP) was determined by using the criteria of an increase in both $VE/\dot{V}O_2$ and $VE/\dot{V}CO_2$ and the second loss of linearity in VE and in $\dot{V}CO_2$ (Wasserman et al., 1999).

Every minute blood samples were taken from the hyperaemic earlobe (Anderson & Rhodes, 1991; E. W. Smith et al., 1997) to measure blood [lactate] (Biosen S – line,

EKF Diagnostic, Barleben, Germany). The analyser was calibrated with a standard solution of 12.0 mmol·L^{-1} and accuracy was verified by using control solutions with known concentrations of 1.6 mmol·L^{-1} and 3.6 mmol·L^{-1} (Precinorm – U, Precipath – U, Roche Diagnostics, Mannheim, Germany). Heart rate was measured continuously throughout the test using short-range radio-telemetry (Polar Vantage NV; Polar Electro, Kempele, Finland).

3.4 Laboratory test trials

The laboratory tests consisted of four trials at level-ground with a gradient of 1.5% and four uphill trials with a gradient of 5%. To simulate gradient the test bike was mounted on an indoor training roller (Tacx Blue Motion, BV Terneuzen, Netherland) and fixed on a treadmill (h/p/cosmos sports & medical GmbH, Nußdorf, Germany). Power output was measured at a rate of 1 Hz with a SRM professional power meter (Schoberer Rad-Messtechnik, Jülich, Germany) which was mounted on a 26-inch mountain bike. To ensure accurate measures a static calibration was applied before the study (Wooles et al., 2005). The bikes´ fork was locked and the tires were inflated to 4 bar. A special indoor training tire was used (Continental Hometrainer II MTB Folding Trainer Tyre, Hannover, Germany). Before each test, the seatpost was adjusted to reproduce the position of the subjects while riding their own bicycles. The participants were instructed to eat and drink during the test session to ensure high glycogen stores and full hydration. Throughout the trials, laboratory conditions remained stable.

The four trials in both conditions were performed at a cadence of 60 and 90 rev·min^{-1} and at a power output of 90% of VT (90%VT) and the work rate at 70% between VT and P_{max} (Δ70)($\Delta 70 = (P_{max} - P_{VT}) \times 0.7 + P_{VT}$). Between each cycling bouts the subjects had a resting period of at least 45 minutes.

After a 10-min warm up on the test-bike the trials started with a 3-min baseline exercise at a power output of 50 W and 60 rev·min^{-1} followed by 6 min cycling at the criterion work rate and cadence. Both, power output and cadence were self-controlled by the athletes during the trials (Nimmerichter et al., 2012).

Gas exchange and pulmonary ventilation were continuously measured throughout the trials as described above.

The $\dot{V}O_2$ breath-by-breath data were examined to exclude errant breaths from sighing, swallowing, coughing, etc., and breaths lying more than four standard deviations from the local mean of 5 data points were removed. The filtered data were linearly interpolated to provide second-by-second values and subsequently time aligned to the onset of the exercise (Whipp & Rossiter, 2005). The first 20 s of the transition from

baseline to Δ70 were deleted to most likely exclude the cardio-dynamic or phase I response (Murias et al., 2011), and a single-exponential algorithm was used to model the kinetics of the fundamental (phase II) response of $\dot{V}O_2$:

$$\dot{V}O_2(t) = \dot{V}O_{2baseline} + A\,(1 - e^{-(t-TD)/\tau})$$

where $\dot{V}O_2(t)$, $\dot{V}O_{2baseline}$, A, TD and τ represent the $\dot{V}O_2$ at any given time (t), the $\dot{V}O_2$ at baseline exercise, the amplitude from baseline to its asymptote, the time delay and the time constant of the response, respectively.

To identify the optimal fitting window of the fundamental parameter response a purpose-designed software was used (LabView 6.1, National Instruments, Newbury, UK) according to the methods of Rossiter et al. (2002). Beginning from the initial 60 s of exercise, the fitting window was increased iteratively by 5-s to end-exercise. For each fitting window the estimated τ was plotted against time and through visual inspection the onset of the $\dot{V}O_2$ SC was determined as the point at which the estimated τ progressively increased following an initial plateau. The parameter estimates were then resolved by least-squares non-linear regression (Graph Pad Prism 5.0, San Diego, USA). The amplitude of the $\dot{V}O_2$ SC was calculated from the difference between the mean of the final 30 s $\dot{V}O_2$ at end-exercise and the asymptote of the fundamental response. To calculate the end-exercise and the fundamental $\dot{V}O_2$ gain the respective amplitudes were divided by the increment in work rate above baseline ($\Delta VO_2/\Delta WR$) (Whipp & Rossiter, 2005).

A capillary blood sample was taken from the hyperaemic earlobe for the measurement of blood [lactate] 1 min post-exercise.

Heart rate was monitored continuously by short-range radio-telemetry (Garmin ANT+, Schaffhausen, Swiss).

Gross efficiency was calculated from measures of EE (J), $\dot{V}O_2$ ($mL \cdot min^{-1}$), $\dot{V}CO_2$ ($mL \cdot min^{-1}$) and power output (W) averaged over the last 60 s of the trials at 90%VT.

$$\mathrm{GE} = \frac{\text{work accomplished}}{\text{energy expenditure}} \cdot 100$$

With $EE = [3.869 \times \dot{V}O_2 + 1.195 \times \dot{V}CO_2] \times (4.186/60) \times 1000$

3.5 Data Analyses

Descriptive data are presented as mean ± standard deviation (SD). To determine differences across the test variables a three-factorial mixed ANOVA with cadence (60 vs. 90 rev·min^{-1}), intensity (90%VT vs. Δ70) and terrain (uphill vs. flat) as model factors was used. Significant main effects were followed up with the Bonferroni post-hoc procedure. The level of significance was set at $p < 0.05$ (2-tailed). The graphics were generated with the software Graph Pad Prism 5.0 (GraphPad Software Inc., San Diego). All statistics were performed using the software package SPSS statistics 20 (IBM Corporation, Armonk, NY).

4. Results

The group mean P_{max} from the incremental graded exercise test was 404 ± 43 W and mean VO_{2max} was 68 ± 4.7 $ml \cdot kg^{-1} \cdot min^{-1}$. The calculated mean power for the test trials were 159 W (59% of VO_{2max} and 39% of P_{max}) for 90%VT and 336 W (93% of VO_{2max} and 83% of P_{max}) for Δ70 intensities. Descriptive data for the participants and maximal measures of the GXT are presented in Table 1.

Table 1: Descriptive data and characteristic of performance from the incremental graded exercise test (mean ± SD)

Variables	Values (*N*=13)
Age (years)	23.0 ± 4.7
Stature (cm)	178.5 ± 5.2
Body mass (kg)	69.0 ± 7.8
P_{max} (W)	404 ± 43
P_{max} ($W \cdot kg^{-1}$)	5.9 ± 0.4
$\dot{V}O_{2max}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	68 ± 4.7
90%VT1 (W)	159 ± 20
Δ70 (W)	336 ± 35
HR_{max} ($b \cdot min^{-1}$)	190 ± 7
$Lactate_{max}$ ($mmol \cdot L^{-1}$)	12.5 ± 2

P = power output; $\dot{V}O_2$ = oxygen uptake; 90%VT1 = 90% from ventilatory threshold; Δ70 = work rate at 70% between VT and P_{max}; HR = heart rate

Table 2 shows the power characteristics during laboratory test-trials. There was no significant difference of cadence ($p = 0.469$) and terrain ($p = 0.816$) in power characteristics between the trials and the coefficient of variation were between 3.5 – 5%.

Table 2: Power characteristics during laboratory test-trials (mean ± SD)

Trial	Power test-trail (W)	CV (%)
90%VT_60_climb	159 ± 21	4.4 ± 1.2
Δ70_60_climb	335 ± 32	4.1 ± 0.9
90%VT_90_climb	158 ± 19	4.3 ± 1.7
Δ70_90_climb	336 ± 33	3.9 ± 1.1
90%VT_60_flat	160 ± 20	4.1 ± 0.8
Δ70_60_flat	335 ± 34	3.7 ± 1.2
90%VT_90_flat	159 ± 20	3.7 ± 0.9
Δ70_90_flat	337 ± 32	4.8 ± 1.6

CV = coefficients of variation

4.1 Heart rate and lactate

Table 3 presents results of the heart rate and [lactate] response for each test-trial.

The mean heart rate was significantly higher at 90 rev·min^{-1} compared to 60 rev·min^{-1} (mean difference = 9.9 b·min^{-1}; $F_{1,12}$ = 289.9; $p < 0.001$) (Figure 19). Mean heart rate was significantly different between 90%VT and the Δ70 intensity (mean difference = 35.3 b·min^{-1}; $F_{1,12}$ = 309.2; $p < 0.001$). The mean heart rate was significantly higher in uphill cycling than in flat cycling (mean difference = 3.4 b·min^{-1}; $F_{1,12}$ = 8.1 $p < 0.05$).

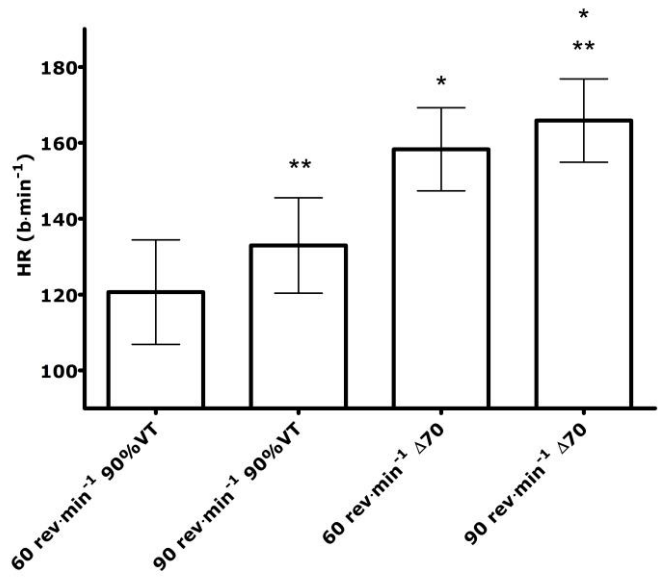

Figure 19: Heart rate at 60 rev·min^{-1} and 90 rev·min^{-1} at 90%VT and Δ70 intensity. **main effect of cadence ($p < 0.001$); *main effect of intensity ($p < 0.001$).

The mean [lactate] 1 min of end exercise was significantly higher at 90 rev·min^{-1} compared to 60 rev·min^{-1} at Δ70 intensities (mean difference = 0.7 mmol·L^{-1}; $F_{1,12}$ = 34.6; $p < 0.001$) (Figure 20). The mean [lactate] was significantly different at Δ70 compared to 90%VT intensity (mean difference = 3.4 mmol·L^{-1}; $F_{1,12}$ = 33.8; $p < 0.001$). There was no significant difference in [lactate] for terrain, flat vs. uphill (mean difference = 0.2 mmol·L^{-1}; $F1_{,12}$ = 0.8; $p = 0.39$).

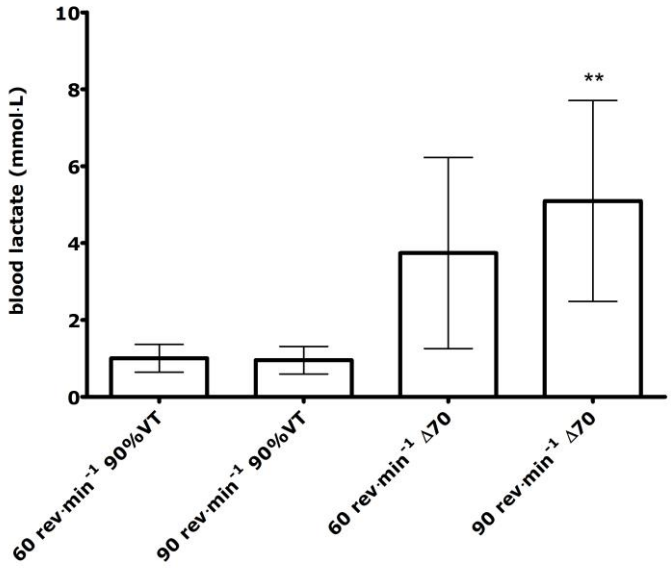

Figure 20: blood [lactate] at 60 rev·min^{-1} and 90 rev·min^{-1} at different intensities. **main effect of cadence ($p < 0.001$).

Table 3: Characteristics of heart rate and lactate (mean ± SD). **main effect of cadence ($p < 0.001$); *main effect of intensity ($p < 0.001$); $^{\$}$main effect of terrain ($p < 0.05$).

Trial	Heart rate ($b \cdot min^{-1}$)	[lactate] ($mmol \cdot L^{-1}$)
90%VT_60_uphill	124 ± 14	1.1 ± 0.4
Δ70_60_uphill	161 ± 10*	3.5 ± 1.9*
90%VT_90_uphill	133 ± 13**	0.9 ± 0.3**
Δ70_90_uphill	166 ± 11** *	4.8 ± 2.9** *
90%VT_60_flat	117 ± 13$^{\$}$	0.9 ± 0.2
Δ70_60_flat	156 ± 12* $^{\$}$	4.0 ± 3.0 *
90%VT_90_flat	133 ± 13** $^{\$}$	1.0 ± 0.4 **
Δ70_90_flat	166 ± 12** * $^{\$}$	5.3 ± 2.3 ** *

4.2 Oxygen uptake

A significant main effect of cadence (mean difference = 3 s; $F_{1,12} = 7.1$; $p = 0.021$), intensity (mean difference = 3.5 s; $F_{1,12} = 6.6$; $p = 0.025$) and terrain (mean difference = 2.8 s; $F_{1,12} = 5.1$; $p = 0.043$) on the time constant was found.

There was a significant difference between the 60 vs. 90 $rev \cdot min^{-1}$ cadence (mean difference = 175.8 $mL \cdot min^{-1}$; $F_{1,12} = 14.8$; $p = 0.002$) and intensity 90%VT vs. Δ70 (mean difference = 1251.2 $mL \cdot min^{-1}$; $F_{1,12} = 414.8$; $p < 0.001$).No significant main effect of terrain (mean difference = 16.8 $mL \cdot min^{-1}$; $F_{1,12} = 0.7$; $p = 0.416$) on the amplitude was found.

A significant main effect of cadence (mean difference = 4.4 s; $F_{1,12} = 10.6$; $p = 0.007$) on the time delay was found. There was no significant main effect of intensity (mean difference = 2.7 s; $F_{1,12} = 2.2$; $p = 0.166$) and terrain (mean difference = 0.2 s; $F_{1,12} = 0.07$; $p = 0.8$).

Slow component occurs only at severe intensity Δ70 trials. There was no significant main effect of cadence (mean difference = 78.5 mL; $F_{1,12} = 2.1$; $p = 0.176$) and terrain (mean difference = 6.9 mL; $F_{1,12} = 0.4$; $p = 0.843$) on SC.

A significant main effect of cadence 60 vs. 90 $rev \cdot min^{-1}$ (mean difference = 0.7 $mL \cdot min^{-1} \cdot W^{-1}$; $F_{1,12} = 9.8$; $p = 0.009$) on the end-exercise $\dot{V}O_2$ gain was found. There was no

significant main effect of terrain (mean difference = 0.2 $mL \cdot min^{-1} \cdot W^{-1}$; $F_{1,12} = 2.1$; $p = 0.171$).

There was a significant difference for the phase II $\dot{V}O_2$ gain between the 60 $rev \cdot min^{-1}$ and the 90 $rev \cdot min^{-1}$ cadence (mean difference = 1.2 $mL \cdot min^{-1} \cdot W^{-1}$; $F_{1,12} = 18.8$; $p < 0.001$) and between the 90%VT and the Δ70 intensity (mean difference = 2.6 $mL \cdot min^{-1} \cdot W^{-1}$; $F_{1,12} = 92.4$; $p < 0.001$). No significant main effect of terrain (mean difference = 0.004 $mL \cdot min^{-1} \cdot W^{-1}$; $F_{1,12} = 0.001$; $p = 0.979$) was found.

There was a significant difference between the 60 $rev \cdot min^{-1}$ and the 90 $rev \cdot min^{-1}$ cadence (mean difference = 275 $mL \cdot min^{-1}$; $F = 45.9$; $p < 0.001$), the 90%VT and the Δ70 intensity (mean difference = 1627 $mL \cdot min^{-1}$; $F_{1,12} = 625.9$; $p < 0.001$) and uphill or flat conditions (mean difference = 69 $mL \cdot min^{-1}$; $F_{1,12} = 6.3$; $p = 0.027$) for the end-exercise $\dot{V}O_2$.

Table 4 and Table 5 present the mean $\dot{V}O_2$ kinetic parameters for each trial. The $\dot{V}O_2$ kinetic responses for cadence, intensity and terrain are presented in Figure 21, Figure 22 and Figure 23.

Table 4: O2 kinetic responses to moderate intensity exercise (mean ± SD). *main effect of cadence ($p < 0.001$); $^{\$}$main effect of terrain ($p < 0.05$).

Measure	90%VT_60 uphill	90%VT_90 uphill	90%VT_60 flat	90%VT_90 flat
Baseline $\dot{V}O_2$ ($mL \cdot min^{-1}$)	1206 ± 96	1181 ± 119	1123 ± 100	1189 ± 141
Phase II $\dot{V}O_2$ time constant (s)	17 ± 6	12 ± 4*	23 ± 8$^{\$}$	17 ± 6*
Phase II $\dot{V}O_2$ time delay (s)	19 ± 12	18 ± 4*	13 ± 8	14 ± 7*
Phase II $\dot{V}O_2$ amplitude ($mL \cdot min^{-1}$)	1114 ± 206	1328 ± 251*	1152 ± 236	1346 ± 251*
Phase II $\dot{V}O_2$ gain ($mL \cdot min^{-1} \cdot W$)	10.3 ± 1.6	12.2 ± 0.9*	10.6 ± 2.1	12.4 ± 1.6*
End-exercise $\dot{V}O_2$ ($mL \cdot min^{-1}$)	2617 ± 289	2843 ± 351*	2543 ± 261$^{\$}$	2799 ± 307*$^{\$}$

No end-exercise gain occurs because of constant $\dot{V}O_2$ levels during the 90%VT trials.

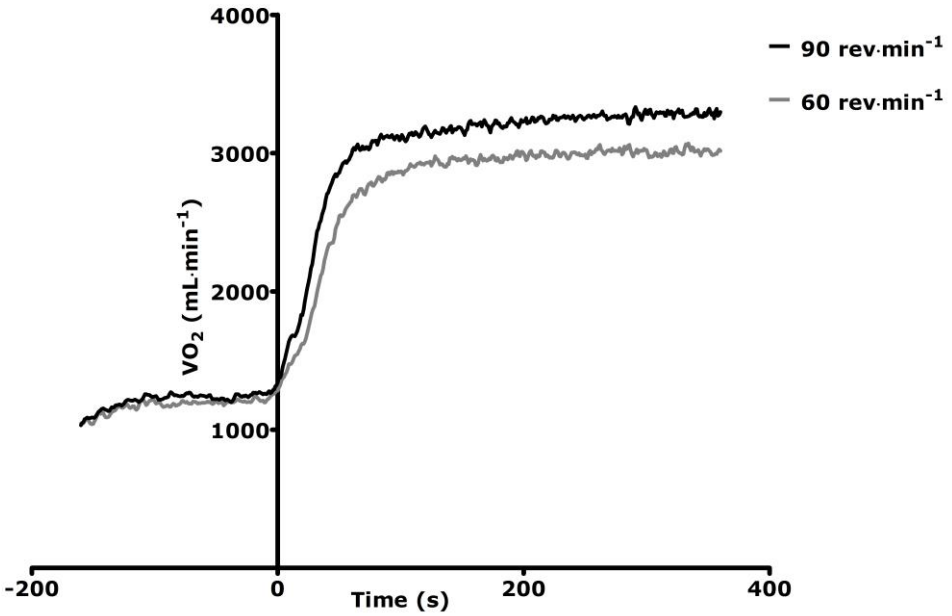

Figure 21: Oxygen uptake response to all trials for both cadences. Significance level for time constant ($p = 0.021$) and amplitude ($p = 0.002$).

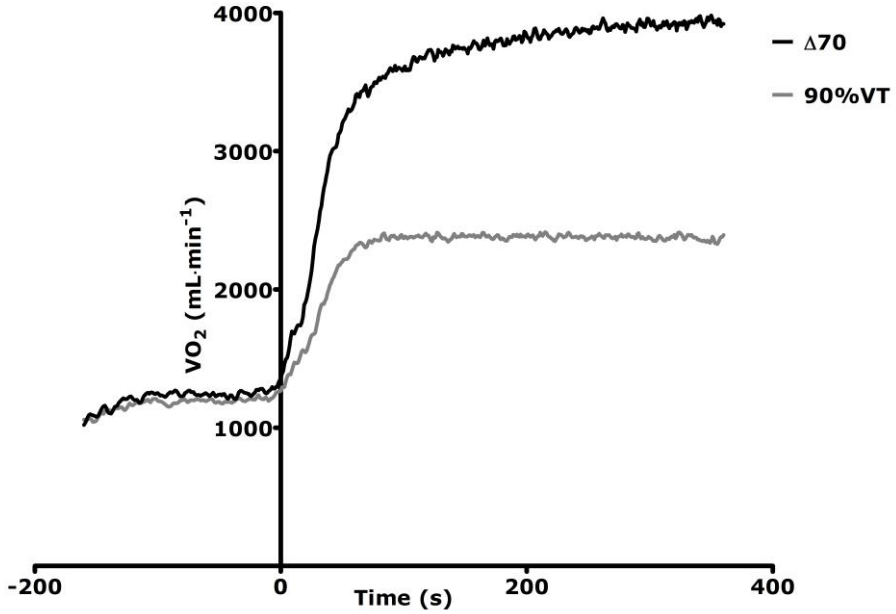

Figure 22: Oxygen uptake response to all trials for both intensities. Significance level for time constant ($p = 0.025$) and amplitude ($p < 0.001$).

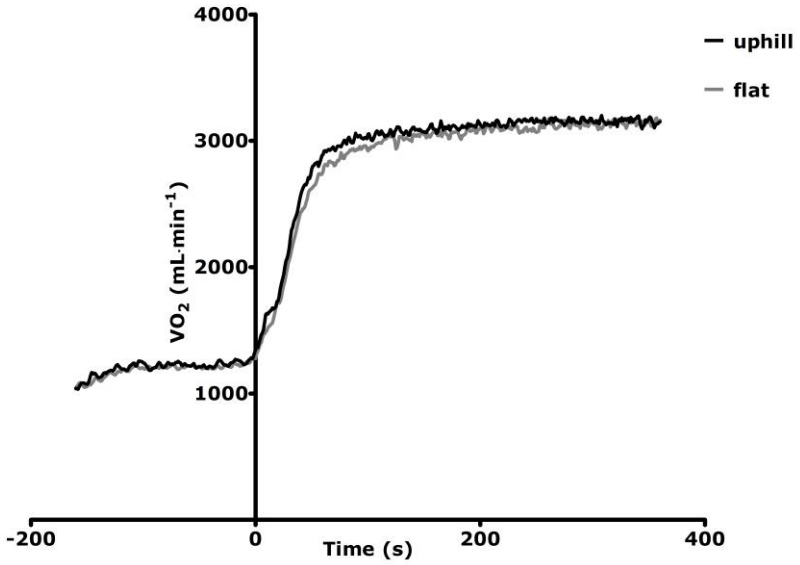

Figure 23: Oxygen uptake response to all trials for flat and uphill conditions. Significance level for time constant ($p = 0.043$) and amplitude ($p = 0.416$).

Table 5: O2 kinetic responses to severe intensity exercise (mean ± SD). *main effect of cadence ($p < 0.001$); $^{\$}$main effect of terrain ($p < 0.05$).

Measure	Δ70_60 uphill	Δ70_90 uphill	Δ70_60 flat	Δ70_90 flat
Baseline $\dot{V}O_2$ ($mL \cdot min^{-1}$)	1191 ± 116	1230 ± 101	1123 ± 100	1250 ± 135
Phase II $\dot{V}O_2$ time constant (s)	21 ± 5	20 ± 6*	21 ± 6	21 ± 5$^{\$}$
Phase II $\dot{V}O_2$ time delay (s)	18 ± 7	13 ± 5*	18 ± 8	14 ± 5*
Phase II $\dot{V}O_2$ amplitude ($mL \cdot min^{-1}$)	2455 ± 358	2580 ± 293*	2370 ± 299	2541 ± 317*
Phase II $\dot{V}O_2$ gain ($mL \cdot min^{-1} \cdot W^{-1}$)	8.7 ± 1.2	9.1 ± 1.1*	8.4 ± 0.8	8.9 ± 1.2*
$\dot{V}O_2$ slow component (mL)	610 ± 263	669 ± 336	597 ± 276	695 ± 388
End-exercise $\dot{V}O_2$ ($mL \cdot min^{-1}$)	4255 ± 463	4479 ± 416*	4090 ± 394$^{\$}$	4486 ± 484*$^{\$}$
End-exercise $\dot{V}O_2$ gain ($mL \cdot min^{-1} \cdot W^{-1}$)	9.9 ± 1.1	11 ± 1.7*	9.9 ± 1.3	10.7 ± 0.8*

4.3 Efficiency

There was a significant difference in GE at 60 (21 ± 1.6 %) and 90 rev·min^{-1} (18.6 ± 1.1 %) (mean difference = 2.2 %; $F_{1,12}$ = 28.1; $p < 0.001$). No significant effect of flat (19.7 ± 1.8 %) compared to uphill cycling (19.7 ± 1.8 %) (mean difference = 0.07 %; $F_{1,12}$ = 0.06; $p = 0.81$) was found. For illustration see Figure 24.

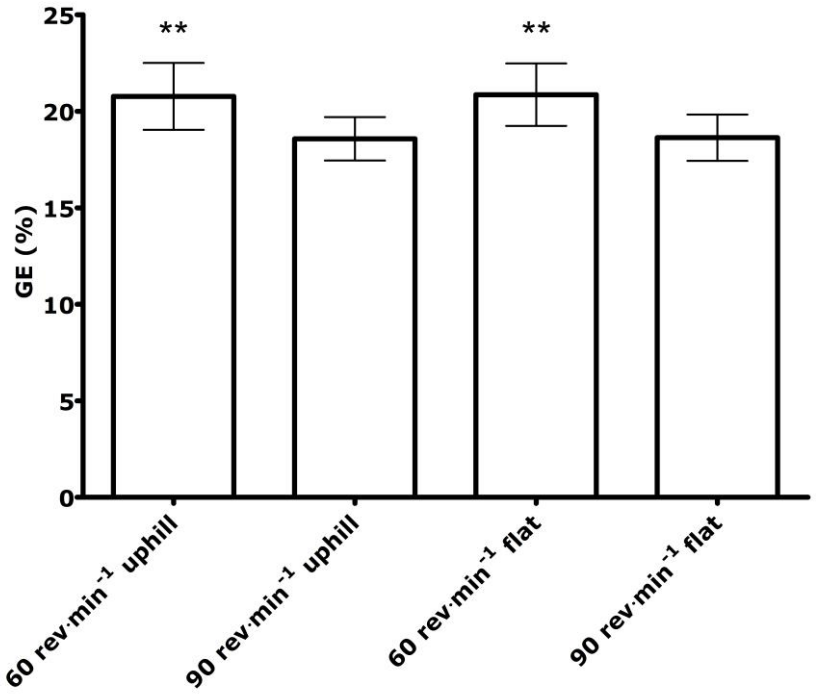

Figure 24: GE at 90%VT for flat and uphill cycling. **main effect of cadence ($p < 0.001$).

5. Discussion

The aim of the present study was to examine the influence of cadence, intensity and terrain on oxygen uptake kinetics and GE in laboratory conditions.

The main findings showed a small influence of gradient on O_2 uptake kinetics. Both time constant (mean difference = 2.8 s; $F_{1,12}$ = 5.1; p = 0.043) and end-exercise $\dot{V}O_2$ (mean difference = 69 $mL \cdot min^{-1}$; $F_{1,12}$ = 6.3; p = 0.027) were affected by inclination.

The exercise intensity influenced the time constant (mean difference = 3.5 s; $F_{1,12}$ = 6.6; p = 0.025), the amplitude (p < 0.001), the phase II $\dot{V}O_2$ gain (p < 0.001), the end-exercise $\dot{V}O_2$ (p < 0.001).

Cadence influenced the time constant (mean difference = 3 s; $F_{1,12}$ = 7.1; p = 0.021), amplitude (mean difference = 175.8 $mL \cdot min^{-1}$; $F_{1,12}$ = 14.8; p = 0.002), time delay (mean difference = 4.4 s; $F_{1,12}$ = 10.6; p = 0.007), end-exercise $\dot{V}O_2$ gain (mean difference = 0.7 $mL \cdot min^{-1} \cdot W^{-1}$; $F_{1,12}$ = 9.8; p = 0.009), phase II $\dot{V}O_2$ gain (mean difference = 1.2 $mL \cdot min^{-1} \cdot W^{-1}$; $F_{1,12}$ = 18.8; p < 0.001) and end-exercise $\dot{V}O_2$ (mean difference = 275 $mL \cdot min^{-1}$; $F_{1,12}$ = 45.9; p < 0.001).

Influence of gradient on O_2 uptake kinetics

To our knowledge, this is the first study to experimentally demonstrate the influence of gradient on oxygen uptake kinetics. Previous studies have examined oxygen uptake kinetics to determine influences of muscle activation (Burnley et al., 2002; Saunders et al., 2000; Scheuermann et al., 2001), muscle fibre type (Barstow et al., 1996; Carter et al., 2004; Jones et al., 2004; Pringle, Doust, Carter, Tolfrey, Campbell, et al., 2003; Vanhatalo et al., 2011), effects of priming exercise (Berger et al., 2006; Buono & Roby, 1982; DiMenna et al., 2009, 2010; DiMenna et al., 2008; Gerbino et al., 1996; Gutin et al., 1976; Inbar & Bar-Or, 1975; Ingjer & Stromme, 1979; Jones et al., 2006; Weltman & Katch, 1976; Whipp, 1994; Whipp & Mahler, 1980) and cadence (Barstow et al., 1996; Jones et al., 2004; Pringle, Doust, Carter, Tolfrey, & Jones, 2003; Zoladz et al., 1998).

In the present study we observed significant differences in oxygen uptake kinetics between flat and uphill cycling. More specifically, the time constant was significantly faster and the end-exercise $\dot{V}O_2$ was significantly higher in uphill (5 %) than in flat (1.5 %) cycling under laboratory conditions. The findings of Nimmerichter et al. (2012) and Tan and Aziz (2005) have shown that higher power outputs can be produced and sustained in uphill time trials compared to flat time trials. The faster time constant and higher end-exercise $\dot{V}O_2$ in uphill cycling subjects showed in our study can be an explanation for their findings.

Influence of exercise intensity on O_2 uptake kinetics

Consistent with the findings of the present work, Perrey et al. (2001), Koppo et al. (2004) and Scheuermann et al. (2001) found significant differences in a lengthening of time constant and a higher amplitude for moderate (90%VT) and severe (Δ70) exercise intensities. In contrast, other studies investigated no differences in time constant between moderate and severe exercise intensities (Barstow & Mole, 1991; Ozyener et al., 2001; Scheuermann & Barstow, 2003). Due to this equivocal findings Poole and Jones (2005) examined the results of 26 studies (i.e. running and cycling) carried out between 1991 and 2004 investigating the influence of exercise intensity on time constant. The analyses show that the time constant is usually slower for heavy/severe exercise (24.4 s) in contrast to moderate exercise (20.2 s).

Furthermore, the results of the present study are in agreement with reports demonstrating that the phase II $\dot{V}O_2$ gain systematically decreases as exercise intensity rises (Jones et al., 2002; Pringle, Doust, Carter, Tolfrey, Campbell, et al., 2003; Scheuermann & Barstow, 2003). A trend for phase II $\dot{V}O_2$ to become reduced at higher work rates is also investigated in several earlier studies in both cycling (Barstow et al., 1993; Carter et al., 2000b; Ozyener et al., 2001) and running (Carter et al., 2002). These findings are supported by the results of this study (Table 4 and Table 5). One factor that may influence the lower phase II $\dot{V}O_2$ gain is that type II muscle fibres have a lower $\dot{V}O_2$ gain than typ I fibres (Barstow et al., 1996; Jones et al., 2002; Pringle, Doust, Carter, Tolfrey, Campbell, et al., 2003; Scheuermann & Barstow, 2003).

Influence of cadence on O_2 uptake kinetics

The findings of this study showed that cadence had an significant influence on all examined O_2 kinetic parameters except the SC at Δ70 intensities. This result is in contrast to the findings of Barstow et al. (1996) and Pringle, Doust, Carter, Tolfrey, and Jones (2003) who reported a significantly higher $\dot{V}O_2$ SC amplitude at higher cadences compared to low pedal rates. Our findings showed a faster time constant and a higher amplitude at higher cadences. These results are supported by the findings of Barstow et al. (1996) and Zoladz et al. (1998). Furthermore, the increased $\dot{V}O_2$ gain at 90 rev·min^{-1} compared to 60 rev·min^{-1} is in contrast to the findings of several studies (Barstow et al., 1996; Carter et al., 2000b; Carter et al., 2002; Jones et al., 2002; Ozyener et al., 2001) that observed a reduction in primary $\dot{V}O_2$ gain with higher pedal rates.

As described in the literature review above several studies investigated the influence of factors like gradient and cadence on GE during cycling. The main findings of this study were that GE was significantly lower at higher cadences and no effect of gradient on GE was found.

Influence of gradient on GE

In this study no significant differences (mean difference = 0.07 %; $F_{1,12}$ = 0.06; p = 0.81) in GE between flat (19.7 ± 1.8 %) and uphill cycling (19.7 ± 1.8 %) were found. Our results are in contrast to the findings of Arkesteijn et al. (2013) who examined GE between a gradient of 0%, 4% and 8% during cycling on a treadmill under laboratory conditions. They found significant differences in GE between 0% and 8% and 4% and 8% gradient but not between 0% and 4% inclination. Contrary some previous studies found no significant differences in GE on the influence of gradient (E. A. Hansen et al., 2002; Harnish et al., 2007; Leirdal & Ettema, 2011; Millet et al., 2002). However, it should be noted that these studies used different exercise intensities and freely chosen cadence which may distort the interpretation (Arkesteijn et al., 2013).

Influence of cadence on GE

The results of this study are consistent to the literature where GE is reported to be lower with increasing pedal rates at same work rates. This findings showed that GE is 2.2 % lower at 60 rev·min^{-1} (21 ± 1.6 %) compared to 90 rev·min^{-1} (18.6 ± 1.1 %). Therefore, GE negatively correlates with cadence independent of exercise intensity (Arkesteijn et al., 2013; Camara et al., 2012; Samozino et al., 2006). In contrast, a higher GE at higher cadences (100 vs. 60 rev·min^{-1}) was reported when work rates increased up to 75% P_{max} and more (Lucia et al., 2004; Samozino et al., 2006). Several previous studies have shown that the most metabolic efficient cadence is between 60 and 70 rev·min^{-1} (Boning et al., 1984; Coast & Welch, 1985; Hagberg et al., 1981; Seabury et al., 1977; Sidossis et al., 1992) but biomechanical analyses of cycling investigated that the minimisation of muscular forces (i.e. muscular torques and muscular forces) and therefore the use of higher pedal rates (Marsh et al., 2000; Neptune & Hull, 1999; Redfield & Hull, 1986) is a priority of the nervous system. As a result, preferred cadences in cycling are between 90 and 100 rev·min^{-1} (Hagberg et al., 1981; Marsh & Martin, 1993, 1997; Marsh et al., 2000).

Cardiovascular and metabolic responses

The findings of the present study showed cardiovascular and metabolic responses for cycling at different gradient, at high or low exercise intensities and different cadences. Different heart rate (HR) and blood [lactate] responses were found between 90%VT intensities and Δ70 intensities and different inclination. The HR was significantly higher at uphill compared to flat cycling (mean difference = 3.4 b·min^{-1}; $F_{1,12}$ = 8.1 $p < 0.05$),

at severe exercise intensities compared to moderate exercise intensities (mean difference = 35.3 $b \cdot min^{-1}$; $F_{1,12}$ = 309.2; $p < 0.001$) and at high cadences compared to low cadences (mean difference = 9.9 $b \cdot min^{-1}$; $F_{1,12}$ = 289.9; $p < 0.001$). Blood [lactate] was significantly higher at severe exercise intensities (mean difference = 3.4 $mmol \cdot L^{-1}$; $F_{1,12}$ = 33.8; $p < 0.001$) and high cadences (mean difference = 0.7 $mmol \cdot L^{-1}$; $F_{1,12}$ = 34.6; $p < 0.001$). These findings are in contrast to the results reported by Lucia et al. (2004) where HR and blood [lactate] levels increased with lower cadences at high work rates. Our findings of the present study are supported by the results of Nimmerichter et al. (2012) and Tan and Aziz (2005) who reported higher HR and blood [lactate] values during uphill cycling compared to flat level cycling. Moreover, higher HR and blood [lactate] values at higher intensities were also investigated by Harnish et al. (2007).

6. Conclusion and directions for future research

In conclusion, this thesis has shown that oxygen uptake kinetics, GE and metabolic responses such as HR and blood [lactate] are affected by exercise intensity, cadence and gradient in laboratory conditions.

The study shows that GE is significantly higher in high compared to low pedal rates and not affected by inclination. Cycling at higher cadences and higher work rates induce higher physiological responses during moderate and high intensity exercises. Furthermore, oxygen uptake kinetics were faster during uphill- compared to flat-cycling and therefore can lead to higher exercise tolerance during climbing.

Accordingly our results, hypotheses 1, 2, 3 and 4 can therefore be accepted while hypothesis 5 is rejected.

A limitation of the study was that free treadmill riding was not possible. Therefore, future studies which investigate influences of gradient under laboratory conditions should try to implement free treadmill cycling in their experimental design. As result further research is required to investigate effects on GE and oxygen uptake kinetics at different inclination in laboratory and field conditions.

List of abbreviations

Δ70	70% difference between VT and P_{max}
A	exponential response amplitude
ATP	adenosine tri-phosphate
CIL	crank inertial load
CP	critical power
DE	delta efficiency
EE	energy expenditure
GE	gross efficiency
GET	gas exchange threshold
GXT	incremental graded exercise test
H^+	hydrogen ion/proton
HR	heart rate
MLSS	maximal lactate steady state
NE	net efficiency
PCr	phosphocreatine
P_i	inorganic phosphate
P_{max}	maximum power output
RCP	respiratory compensation point
RER	respiratory exchange rate
SC	slow component
SD	standard deviation
τ	time constant (time to reach 63% of an exponential response)
TD	exponential response time delay
$\dot{V}CO_2$	carbon dioxide ventilation
VE	pulmonary ventilation
$VE/\dot{V}CO_2$	ventilatory equivalent for carbon dioxide
$VE/\dot{V}O_2$	ventilatory equivalent for oxygen

$\dot{V}O_{2max}$	maximum oxygen uptake
VT	ventilatory threshold
W	watt
WE	work efficiency
WR	work rate

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