SINGLE- LEG CYCLING: A NEW TRAINING PARADIGM FOR IMPROVED BILATERAL CYCLING PERFORMANCE?

by

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III. Intellectual property rights

IV. Abstract

Purpose: A pilot study to examine the potential additive training stimulus of single-leg (SL) cycle training (4wk), upon double-leg (DL) cycling TT performance. **Methods:** 11 trained cyclists (2 female), with ≥4 yr experience (mean ± SD: 33 ± 10yr, 73 ± 5.6kg, W_{max} = 335 ± 33 W, VO_{2max} = 62 ± 5.9mL·kg $^{-1}$· min^{-1}) were recruited. Cyclists performed a SL and DL VO_{2max} test, plus a ~30min simulated DL TT, before they were randomly assigned to either the DL or SL training groups. Cyclists incorporated 12 x1h sessions (under laboratory supervision) to their normal training. DL participants performed 6 x 5min at ~70% $W_{max,}$ with both legs simultaneously (1:1 rest). SL participants performed 12 x 5min at ~35% W_{max}, alternating training leg every 5min (on modified cycle ergometers- the contralateral pedal loaded with a 10 kg counter-weight). All performance tests were then repeated. **Results:** Cycling time trial (TT) performance was improved similarly, regardless of the mode of cycle training intervention (both groups improved TT performance by ~4.1% ($p<0.05$)). DL VO_{2max} and W_{max} were only increased significantly in the DL trained group (both $p \leq 0.01$), by ~4.6% and ~3.0% respectively. No significant improvement in SL VO_{2max} was observed in either group. **Conclusion:** 4 wks of SL training did significantly improve DL TT performance in well trained cyclists, perhaps eliciting a differential training stimulus. Regardless, SL training remains to be proven significantly more effective than DL training of a similar duration and intensity.

Key words: Single-leg cycling, Training response, Time trial performance, Maximal aerobic power, Plasma lactate accumulation, Limitations to VO_{2max}, Peripheral adaptation.

V. Attestation of authorship

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, if contains no material previously published or written by another person, nor material to which the substantial extent has been accepted for the qualification of any degree or diploma of a university or other institution of higher learning, except where due acknowledgement is made.

Signed……

Date……

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IX. List of Abbreviations

SL	Single-legged
DL	Double-legged
$VO_{2\ max}$	Maximal oxygen consumption
W_{max}	Maximal sustained power output (wattage) at VO_{2max}
W_{peak}	Maximal power output (wattage) reached at VO_{2max}
HR_{max}	Maximal heart rate (beats.min^{-1}) reached at VO_{2max}
HR	Heart rate (beats.min^{-1})
TT	Time trial
TTC	Time to complete
PO	Power output
O_2	Oxygen
CO_2	Carbon dioxide
V CO_2	Volume of carbon dioxide
N_2	Nitrogen
VE	Minute ventilation
RER	Respiratory exchange ratio
CAD	Cadence
RPM	Revolution per minute
HIT	High intensity interval training
TRIMP	Training impulse
BLa	Blood lactate
MBC	Muscle buffering capacity
CV	Coefficient of variation
SD	Standard deviation
d	Day
wk	Week
y	Year
h	Hour
bw	Body weight
kg	Kilogram
W	Watt/ Wattage
Av.	Average
CW	Counter weight
NADH	Nicotinamide adenine dinucleotide hydrogenase
NAD^+	Nicotinamide adenine dinucleotide
LDH	Lactate dehydrogenase
H^+	Hydrogen
ECSS	European College of Sport Science
Q	Cardiac output
VL	Vastus lateralis

Chapter 1.0

Introduction

1.0 Introduction

The popularity of cycling as a competitive pursuit continues to grow exponentially, at both a national and global level. British cycling increasing their number of cyclists with race licenses by approximately 53% between 2005-2008 alone (Jones 2008- BC Press). As a result of this growing competitive interest, demand for literature regarding elite cycle training strategies for improved cycling fitness and competitive performance has increased within both the cycling profession and the public domain.

Evidently the type, volume, frequency and intensity of training performed by the individual athlete are key influential factors in improving cycling performance (Hawley 1997). However despite a plethora of scientific research to date investigating both high intensity and endurance training, specific successful cycle training paradigms for improving high performance cycling have yet to be fully optimized (Faria 2009).

Specifically scientific literature related to the unique effects of certain training interventions on the performance of well- trained athletes is limited, due to reluctance of such athletes to participate in invasive studies and to alter their already established training regimens (Stepto et al. 1999).

As a result coaches and researchers alike continue to build upon anecdotal evidence with varied attempts to explore the most effective of training strategies to maximize cardiovascular and local musculoskeletal adaptations that underlie improvements in performance; experimenting with new technological advances and forward thinking training models. The need

for more scientific research focussed specifically on the optimization of training strategies is clear. Emphasis should be given to the conception of new training paradigms, which involve the identification and implementation of alternative training stimuli with the potential to evoke novel adaptive cellular responses for elevated competitive cycling performance in both the elite and sub-elite.

1.1 Study aims and objectives

The aim of this study is to further develop the existing SL (single legged) exercise model (with the addition of a counterweight device) in order to determine if the incorporation of SL cycle training into a normal cycling training regime, further improves DL (double legged) cycling performance; in comparison to a normal DL cycle training of an equivalent intensity and duration. DL cycling performance will be compared pre- post two different 4 week cycle interval training interventions via the implementation of a DL VO_{2max} test, two independent SL $VO_{2\,max}$ tests and a ~30min laboratory based TT (set an intensity of 85% DL W_{max}).

1.2 Literature review

This review aims to evaluate the existing research surrounding implied differences in limitation to maximal aerobic consumption experienced during high intensity DL and SL cycling exercise. Within this review the rational supporting the implementation of SL cycling as an alternative training paradigm for improved DL cycling performance is explored, plus justification for the inclusion of an opposing counterweighted pedal within the existing SL cycling model is outlined.

1.2.1 Physiological limitations to Double-legged VO_{2max}

During prolonged high intensity physical work it is the ability of an individual to uptake, transport and deliver oxygen to the working muscles effectively and efficiently which is thought to greatly influence that individual's endurance performance capacity (Saltin and Astrand, 1967; Rusko 1978). Although VO_{2max} is not a complete indication of 'performance' *per se*, it clearly is one of the major characteristics that determine performance in endurance sport (Peronnet *et al.* 1991; di Prampero, 2003). Each step of the oxygen cascade from the air into the mitochondria is a potential restriction of VO_{2max}. If the oxidative phosphorylation is limited by the availability of mitochondrial oxygen, which is determined by how quickly the oxygen can be delivered to the muscle cell, a supply limitation is present. Equally, if the oxygen availability in the mitochondria outreaches the utilization by the oxidative phosphorylation, the system is demand limited (Wagner 2000).

Previously researchers have argued that the increase in VO_{2max} during DL cycling exercise was solely limited by peripheral (tissue) events within the working muscle; unrelated to cardiovascular transport and the capacity of the heart to increase its output (Kaijser et al. 1970). Although this is almost certainly the case during exercise where the effective exercising muscle mass is reduced and presumably cardiac output is not the predominant limiting factor (as in SL exercise) (Davies and Sargeant 1975). Contradictory evidence has accumulated with regard to normal bilateral (DL) exercise as maximal systemic O_2 delivery is considered the product of maximal cardiac output (Q_{max}), plus the arterial O_2 content (CaO_2) at peak exercise. Maximal cardiac output is determined by the stroke volume (SV) and heart rate (HR) at maximal or near-

maximal exercise (Q = SV × HR), with SV playing a predominant role (Saltin et al. 1968). Therefore, during DL exercise maximal oxygen uptake in athletic individuals appears to be limited by oxygen transport limitation (i.e. cardiac output) (Saltin et al. 1967; Wagner 2000; Liguzinksi & Korzeniewski 2007).

On a continuum from rest to maximal exercise, the cardiovascular system must adjust to the increasing metabolic demand by ensuring the delivery of O_2 and substrates to all body cells, especially the myocytes, without compromising arterial pressure. Currently, the prevailing theory is that the cardiovascular system responds to exercise of increasing intensity up to a certain maximal aerobic power (VO_{2max}) (Mortensen et al.2005). The cardiovascular system is able to accommodate a rise in O_2 demand from the musculoskeletal system by increasing systemic O_2 delivery to the contracting skeletal muscle mass, whilst also increasing the perfusion pressure (Holmgren 1956; Astrand et al. 1964; Mortensen 2005; 2008). However, when no longer able to respond in this manner, it may be considered a limitation to the cardiovascular system which leads to a limitation of maximal exercise performance (Neary and Wenger 1986; Saltin 1976).

Subsequent research comparing SL vs. DL exercise of both legs concurrently includes heavy conjecture that the limitation to VO_{2max} during exercise in which a large muscle mass is used (DL) could be principally central. Decrements in power production in this instance assumed to be the result of a limit to the maximal amount of oxygen that can be delivered to each exercising limb (whilst both legs are exercised simultaneously), as the maximal vasodilatory capacity of skeletal muscle clearly exceeds the ability of the heart to deliver blood and still maintain adequate arterial perfusion pressure (Secher et al. 1981; Richardson et al.1999). Specifically, central factors

considered to contribute to this limitation include cardiovascular fatigue, inadequate pulmonary diffusing capacity and an acute limitation of muscle perfusion in order to maintain whole body blood pressure (Davies & Sargeant 1975; Klausen et al. 1982, Neary and Wenger 1986) - see Figure 1.

Interestingly, in support of this theory that DL VO_{2max} is predominantly limited by the central component (i.e. cardiac output) several previous studies have shown that VO_{2max} measured during SL exertion can be up to ~70-85% of that measured during a regular bilateral incremental test protocol (Neary & Wenger 1986; Stamford et al. 1978; Klausen at al. 1982, Gleser 1973). However, on a per limb basis, the concurrent maximal double-legged oxygen consumption does not subsequently equal double the value accomplished by that on a single limb. In fact via scrutiny of previous data (mentioned above) and our own pilot data (un-published pilot 2009), we have established that a cyclist who is working maximally bilaterally sees a reduction of approximately 30% in the amount of power production (wattage) they are capable of per limb- each leg only able to work at ~70% of SL VO_{2max}. Saltin et al. (1976) suggested that just one third of the muscle mass of man can fully tax the capacity of the heart and consume the oxygen delivered by the heart, an evidence of supply limitation of VO_{2max} in healthy subjects during whole body work.

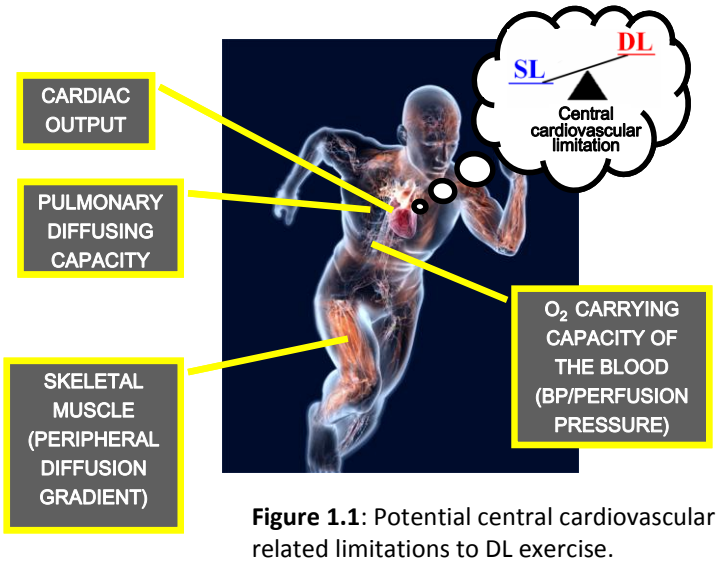

Figure 1.1: Potential central cardiovascular related limitations to DL exercise.

Furthermore, those assumptions related to a central limitation to exercise involving the activation of a large muscle mass have been supported by more recent work, which has examined the limitations to human VO_{2max} in detail (Mortensen et al. 2005, 2008; Levine 2008; Liguzinski and Korzeniewski 2007; Ogita et al. 2000). Primarily Mortensen and colleagues (2005), focused upon discerning the impact of limitations to systemic and locomotor muscle blood flow and O_2 transport (whilst exercising a large muscle mass-i.e. two large limbs, exercised simultaneously) and how they may impose a limitation to aerobic power and endurance capacity in trained individuals; via the comparison of DL cycling exercise and SL knee extensor exercise. During maximal DL cycling blood flow and O_2 delivery levelled off at ~73-88% of peak power, blunting leg VO_2 per unit work despite increasing O_2 extraction. In contrast to SL cycling, where leg blood flow and VO_2 increased until volitional exhaustion during one-legged knee-extensor exercise where O_2 transport was not limited (due to a smaller active muscle mass). Thus, these observations indicate that it is an inability of the circulatory system to sustain a linear increase in O_2 delivery to the locomotor muscles which may subsequently restrain aerobic power production and endurance capacity in humans during maximal DL exercise. Ogita et al. (2000) similarly concluded that the differential VO_2 response between SL and DL exercise could be attributed not only to the difference in force application throughout the exercise movement and to the effect of a postural component but also to the modified circulatory response caused by the multiple limb exercise.

Subsequently, Mortensen et al. (2008) further investigated the regulatory limits of cardiac output (*Q)* and muscle perfusion through the comparison of systemic and leg haemodynamic responses to maximal and supramaximal DL cycling *versus* maximal SL knee-extensor exercise.

During supramaximal DL cycling, *Q*, leg blood flow (LBF), and systemic and leg O_2 delivery and VO_2 reached peak values after 60–90 s and thereafter levelled off at values similar to or ~6% (P<0.05) below maximal cycling. Whereas, during SL knee-extensor exercise *Q* and LBF increased linearly until exhaustion, accompanied by increases in non-locomotor tissue perfusion (~12 l.min). Plus, post DL cycling compared to SL knee-extensor exercise, *Q*, LBF, leg vascular conductance, leg O_2 delivery and leg VO_2 for a given power were reduced by ~32–47% (P <0.05).

Collectively these findings indicated that the blunted muscle perfusion during exercise engaging a large compared to small muscle mass could be associated with enhanced vasoconstrictor activity and a plateau in *Q*. These data were considered to suggest that despite the higher metabolic energy demand, systemic and locomotor skeletal muscle perfusion, vascular conductance, O_2 delivery and aerobic metabolism become progressively more restricted during an intense whole body exercise such as high intensity DL cycling. This restriction suggested to be due to an inability of the circulatory system to meet the increasing metabolic demands of both the locomotor and non-locomotor muscles, indicative of the restriction of tissue and muscle perfusion regardless of the extreme exercise stimulus to vasodilate during supramaximal exercise.

Similarly, recent 'in silico' studies have also shown agreement with Saltin's (1976) previous preposition that it is a limitation of oxygen delivery to the muscle, rather than a limited capacity of the musculature to adapt to utilize the available oxygen that inhibits subsequent performance. One such static computer model quantifiably predicted that when all other parameters and system properties are kept constant, an increase in working muscle mass from

2.5kg (single quadricep) to 15kg (two legs) causes a restriction to whole body oxygen supply by blood; consequently limiting maximal O_2 consumption, oxidative ATP production and work rate (Liguzinski and Korzeniewski 2007). Similarly, by calculating the contribution of the individual steps of the respiratory cascade, di Prampero (1985) concluded that for healthy humans exercising in normoxia, about 75 % of VO_{2max} is set by central oxygen transport and 25 % by the periphery.

Furthermore, Levine (2008) has recently written an encompassing review, providing in depth discussion related to the limitations of whole body (DL) VO_{2max} as a determinant of performance. Within this review both the 'classical' viewpoints and 'contemporary' investigations into the mechanisms underlying peripheral muscle fatigue due to energetic supply/demand mismatch were clarified. The take home message was that VO_{2max} is still currently considered an important determinant of endurance performance which according to Levine (2008) represents a true parametric measure of cardiorespiratory capacity for an individual at a given degree of fitness and oxygen availability. However, recent controversy surrounding the continued relevance (or irrelevance) of VO_{2max} as an index of 'maximal' aerobic capacity is discussed in depth, plus the relevance of the alternative concepts including: exercise economy and lactate threshold. Levine (2008) concludes that athletes are proposed to stop exercising at VO_{2max} due to severe functional alterations at the local muscle level due to what is ultimately a limitation in convective oxygen transport (Amann and Calbert 2008). Thus, highlighting the peripheral contribution to VO_{2max} and peripheral component of oxidative fatigue as a potential area for future research. However, VO_{2max} is not an absolute concept; it is acutely changeable dependent on the trained status of the

individual, plus the active muscle mass that is engaged in the activity (Wagner 2000; Saltin et al. 1976). During dynamic endurance exercise (e.g. cycling) increased oxygen to be delivered to the working skeletal muscles in order to meet increased energy demands. The power that can be sustained by the muscles of each leg during endurance exercise is limited by the delivery and extraction of oxygenated blood. It is suggested that training improves O_2 delivery by enhancing mean transit time to maintain or expand O_2 extraction (a-v O_2 difference) even at high blood flow rates. Improved a-v O_2 difference is achieved by an increased mitochondria size and number, increased rate limiting enzymes and capillary density (Basset and Howley 2000). Previous research by Wagner et al. (2000) has suggested that in ambient air at sea level, maximal oxygen uptake in athletic individuals is considered primarily set by oxygen transport limitation (i.e. cardiac output), whereas in unfit subjects, it is set by mainly peripheral metabolic limitations. Therefore, it could be suggested that in trained subjects peripheral factors previously limiting $VO_{2\,max}$ are improved and a training strategy which may target these peripheral metabolic adaptations specifically could optimize training induced enhancements in both the trained and untrained; potentially impacting more so upon the untrained individual.

1.2.2 Training for improved endurance cycling performance

The purpose of exercise training is to alter physiological systems in such a way that physical work capacity is enhanced through improved homeostasis preservation during subsequent exercise sessions (Laursen, 2010). Principally endurance performance is governed by a multitude of peripheral and central physiological variables and parameters that are being constantly adjusted in response to repeated training bouts. Parameters such as VO_{2max}, cardiovascular

dynamics and sustainable power distribution (lactate threshold), along with their regulatory morphological components (i.e., aerobic enzyme activity, muscle buffering capacity, capillary density, skeletal muscle acid base status), are related to both peripheral and central regulatory metabolic pathways (Joyner & Coyle 2008; Hawley 2002; Hawley & Stepto 2001). Therefore, it is the way that certain training regimens impact upon those regulatory pathways that is of specific interest when targeting improved performance.

During a high intensity endurance event such as a cycling time trial, the focus is to maintain the highest level of speed or power production possible, a combination of which defines an individual's peak 'performance velocity' (Coyle 1999; Joyner & Coyle 2008). Endurance exercise training is noted to result in profound adaptations of the cardiorespiratory and neuromuscular systems that enhance the delivery of oxygen from the atmosphere to the mitochondria and enable a tighter regulation of muscle metabolism (Jones & Carter 2000). These adaptations affect an improvement in endurance performance via metabolic changes that is manifest as a rightward shift in the 'velocity-time curve' through either an increased muscle power production, increased ability to resist fatigue, or increased motor coordination and efficiency (Lambert et al. 2008). This shift enables athletes to exercise for longer at a given absolute exercise intensity, or to exercise at a higher exercise intensity for a given duration.

It is known that athletes who are already highly trained incorporate high-intensity interval training (HIT) into their training programs to improve their performance in preparation for competition (Hawley et al. 1997; Laursen & Jenkins 2002; Laursen 2010). In fact a number of recent studies, typically using cyclists, have found that substituting ~15% of the total weekly training volume with HIT completed at a variety of intensities (i.e., 80 – 150% W_{max}, for 2-4 wk),

can improve 40km cycling TT performance significantly, by up to 5%, in a matter of 2-4 weeks (Lindsay et al. 1996; Stepto et al. 1999; Westgarth-Taylor et al. 1997; Weston et al. 1997). Previously, the common hypothesis has been that a training modality which elicits a more intense training stimulus, such as repeated HIT training, might subsequently allow the athlete to sustain a higher fraction of VO_{2max} or power output for a longer period without the excessive accumulation of lactate in the active muscles (Stepto et al. 1998; Westgarth-Taylor et al. 1997). A hypothesis which is strongly reflected by the work of Weston et al. (1997), which demonstrated that after only six sessions of HIT (6-8 x 5 min at 80% W_{max}) in well trained cyclists a significant improvement in muscle buffering capacity was reported. However, the mechanism underpinning an improved muscle buffering capacity is not well understood. Edge et al. (2006) suggested that increased H^+ accumulation, potentiated via HIT above the lactate threshold is a potent stimulus for the up-regulation of muscle buffering capacity.

Evidence for the importance of peripheral adaptations for training induced improvements in endurance capacity suggests that training aimed at placing an increased stimulus or metabolic 'loading' on peripheral systems and mechanisms that may lead to increased adaptations to such peripheral factors could have a positive effect on endurance performance (Laursen et al. 2005; Laursen & Jenkins 2002). Laursen et al. (2002) demonstrating that the more traditional measure of endurance capacity -$VO_{2\ max}$ has previously been recorded as unchanged post high intensity interval training which improved both ventilatory threshold (VT) and peak power output (PPO), following only four HIT sessions in already highly trained cyclists.

Consistently, measures of alternative factors related to peripheral adaptation such as measures of W_{max} , economy and lactate buffering capacity/ threshold power output have been reported to

perhaps be superior indicators of subsequent endurance cycling performance (Bishop et al. 1998; Coyle et al.1991).

1.2.3 Proposed alternative enhancement potentiated by a SL training regime

An alternative cycle training approach, which focuses upon a reduction in the central limitations (i.e. oxygenated blood supply) which are prevalent when performing high intensity cycle training, could involve exercising each single smaller muscle mass (lower-limb) individually. The single leg cycling training model has been designed in order to train only one leg at a time, rather than train both legs simultaneously. During normal bilateral HIT cycling, each active limb reportedly consumes ~ 50% of the total oxygen uptake available (minus the O_2 cost of unloading cycling), as theoretically each leg is only responsible for producing half of the power required by the cycling action. As a result the musculature of each leg when training in the DL (synchronous) manner is able to maintain only the capacity to process ~50% of the total supply of the oxygenated blood available. Submaximal aerobic performance potential is characterized by measures such as ventilatory threshold (VT- the point at which CO_2 production exceeds O_2 consumption) and lactate threshold (LT- the point at which accumulation of peripheral metabolic by products in maintained at a consistent low level). Both of these measures relate to the metabolic energy cost of performing sustained high intensity aerobic exercise (economy of motion). Thus, increases in VT and LT reflect increases in respiratory capacity, in particular the muscle's ability to utilize oxygen whilst accruing minimal metabolic stress An improvement in VT and LT is synonymous with improved submaximal endurance performance, however training increases are challenging because the respiratory capacity of the muscle must be stressed (overloaded) in order to produce such positive adaptations. The premise behind SL cycling is that the reduced oxygen uptake of the inactive leg allows the active muscles to

be potentially supplied with significantly more oxygenated blood (Klausen et al. 1982), such an increase in oxygen availability should allow for greater work production in each leg, overloading mitochondria, and causing an increase in respiratory capacity (Abbiss et al. 2010). Therefore, single-leg cycling may allow for greater peripheral muscle respiratory adaptations than double-leg cycling (Klausen et al.1982). Specific molecular adaptations to regular SL training are thought to related to improvements in mitochondrial oxidative capacity (oxidative enzyme activity) and glucose transport with the muscle cell- particularly influx (Abbiss et al. 2010).

In summary, DL endurance cycle training is known to elicit both central and peripheral adaptations, alter neural recruitment patterns, and cause profound changes in bioenergetics and enhanced morphological, metabolic substrate and acid based status in skeletal muscle (Hawley & Stepto 2001; Hawley 2002). A combination of these factors allow an individual to increase their training status and the performance velocity they are able to sustain. However, the aim of this study was to implement a novel SL cycle training regimen, the idea behind which was to examine whether or not specifically targeting a local peripheral adaptation (via the intense activation of a small independent muscle mass), would elicit a similar or greater performance enhancement than that already demonstrated by traditional methods of DL endurance cycling which have been discussed above.

1.3. Single-legged Cycle Training: a new training model

1.3.1 Previous Investigation

The impetus for research using a SL exercise training model (on a cycle ergometer) was initially generated in the 1970's. Previous research was driven by specific interest in the influence of locomotor muscle oxygen delivery upon VO_{2max} in the trained and untrained limb musculature (Clausen el al. 1978; Gleser 1973; Hardman et al. 1987; Klausen 1982; Saltin 1976). However, very few studies investigated the physiological effect of SL cycling in trained participants, and even fewer involved a comparative training approach, Swenson and Howley (1993) the only researchers to previously do this. The majority of studies that have been conducted, have utilized different SL exercise models and include methodological factors that make interpretation and comparison of the findings difficult – to be discussed in more detailed below. Other limitations include the fact that the majority of previous studies neglected to include a 'control group', plus nearly all investigations engaged small sample sizes, which consequently resulted in low statistical power.

In one of the earliest related investigations, involving six sedentary subjects ($VO_{2max=}$ 46.6 $ml.kg^{-1}.min^{-1}$), Gleser (1973) reported a 16% improvement of SL peak aerobic power (VO_2) and a 13% enhancement of the SL peak cardiac output post four weeks of cycle training of just one leg (2 x exhaustive sessions.wk at ~75% VO_{2max}); the contralateral passive leg was used as a control. Despite this, neither DL VO_{2max} nor maximal cardiac output was demonstrated to increase after SL training when the same exercise test was performed in the normal DL mode (when exercising both limbs simultaneously). As a result, Gleser (1973) suggested that the increase in SL VO_{2max} was brought about via a training induced enhancement of the ability of the peripheral muscle

vasculature to dilate and accept a higher leg blood flow. However any enhancement observed during maximal SL cycling was not evident during DL exercise.

Nevertheless, despite the seeming clarity of previous conclusions by Gleser (1973), his findings have since been queried due to the proposed use of a reportedly 'invalid' SL training approach (Davies and Sargeant 1975). The SL training routine involved the simultaneous use of one bike between 2 subjects, each subject standing upright. Thus, no replicable or comparable measures were taken and each subject was potentially training at different levels of intensity; largely dependent on the effort generated by their partner. In addition, whilst standing, each subject could in effect utilize their opposing 'resting limb' to generate transferable power to the 'cycling limb'; potentially invalidating the measurement of power output generated by each limb independently. However, Gleser (1973) maintained that a comprehensive familiarisation to the SL partnership working model minimized any issue with compliance and equal power output.

Subsequently, within a more controlled design, Davies & Sargeant (1975) investigated the effects of SL cycle training on the physiological response to one- and two-leg work. During the study subjects trained each leg separately for 30min (3 sessions per wk for 5-6 wk, at ~80% SL VO_{2max}). Results were in agreement with Gleser (1973), in that post SL training of a limb they demonstrated a large increase in SL cycling VO_{2max} (~ 14%). Thus, suggesting that when each leg is trained independently, there could be a greater capacity for the independently trained limb to improve its oxidative capacity. Davies and Sargeant (1975) concluded that this may be due to an increased heamodynamic response (an increased blood flow to the active limb), evident during unilateral exercise, thus explaining the notable lack of a similar significant improvement in bilateral VO_{2max}.

In 1976 the nature of the SL training response was further investigated. Saltin et al. produced additional evidence which showed a significant increase in maximal aerobic power following SL endurance exercise; primarily considered due to a marked peripheral adaptation. In this study by Saltin et al. (1976), those subjects that had performed one-leg endurance training on a cycle ergometer improved their VO_{2max} by 24% during a subsequent incremental exercise to exhaustion with that trained leg. However, DL VO_{2max} was only improved by 11%. Thus, the improvement observed during two-leg exercise was less than expected if the limitation to VO_{2max} had been only of peripheral origin. Results therefore supporting aforementioned arguments suggesting that part of the limitation to VO_{2max} during bilateral exercise is due to insufficient perfusion of the working musculature and oxygen delivery by blood; a limitation that may be removed during SL exercise.

A successive training study by Klausen et al. (1982) added further evidence to this theory, with an in depth investigation of the physiological impact of SL training- including Leg blood flow (LBF) determined via intravenous tracer technique. Participants trained each leg individually (30 min LL; 30 min RL) on a cycle ergometer 3 x wk (8 weeks of endurance exercise in total). Results indicated that post SL training of SL peak leg VO_2 was increased by 19% (0.51 l.min), whereas the increase demonstrated during DL maximal exercise was only 11% (0.37 l.min). This was mainly considered due to a 23% higher peak leg blood flow in the SL mode. Conversely, prior to SL training, peak VO_2 per limb was the same during unilateral and bilateral cycling, despite the fact that leg blood flow was already 8% higher during SL exercise. This highlights the preposition that VO_{2max} may be augmented via specific training strategies which focus upon producing increases in muscle conductance and O_2 uptake.

In summary, none of the early research including reference to SL training (training one limb independently), has attempted to include quantifiable cycling performance related measures (i.e., W_{max} /maximal print performance/ TT performance). Previous research only focusing on the determination of VO_{2max} and the limitations to maximal, whole body exercise when a large muscle mass is recruited. Equally, no attempt to validate SL training as an alternative training mode has been completed and no thought to minimizing differential muscle recruitment during SL cycling in comparison to normal DL cycling has been made- making it hard to compare this preliminary data with the values relating to VO_{2max} and training intensity to those which we expect to demonstrate within this current study with the addition of a counterweight to the SL training mode.

1.3.2. Contemporary single- legged cycling research

As outlined in 2.3.1 above, previously SL cycling as a 'training technique' has only very briefly been touched upon as a research concept. More recently, researchers have attempted to focus more on the investigation of any potential performance enhancement in the DL mode as a result of additional SL training, rather than simply focusing on establishing a direct inference regarding central vs. peripheral limitation upon VO_{2max} using the two different exercise models.

To date, Swensen and Howley (1993) have implemented the only study which has demonstrated that 4 weeks of SL endurance cycle training produced a *significant* increase in maximal bilateral aerobic power ($p < 0.001$). Within this four week investigation SL trained subjects ($n = 9$), completed non-simultaneous training of both legs (each leg 15 min.day, 4 d.wk), whereas the DL participants completed double that amount (30 min.day) with both legs

simultaneously. Both groups trained at a heart rate equal to that measured at 75% of their DL $VO_{2max.}$ Gains in DL aerobic power were found to be significant regardless of whether both legs were trained individually (SL) or together (DL). However, by attempting to match training intensity between groups via the HR elicited during exercise, the SL group will have worked at a higher training intensity. In addition, any increase in $VO_{2\ max}$ noted in this study could be attributed to the low training status of the participants recruited- as they were 'healthy male varsity athletes' not highly trained cyclists ($VO_{2\ max}$ = 4.27 l.min).

More recently an increase of ~40% in the rate of O_2 uptake during SL cycling, compared to bilateral cycling ($p<0.05$) has been reported (Bundle et al. 2006); these findings support previous work which indicates that cardiovascular limitations imposed upon DL exercise do not apply to maximal SL exercise (Davies & Sargeant 1975; Klausen et al. 1982; Neary & Wenger 1986; Saltin et al. 1976; Stamford et al. 1978). Furthermore, such evidence adds to the preposition that in the SL training mode the reduced oxygen uptake of the inactive limb allows the active musculature to be potentially supplied with an optimal amount of oxygenated blood. Therefore, additive improvements in the potential mitochondrial oxidative capacity of skeletal muscle may be stimulated via increased blood flow and oxygen delivery to the exercised muscle (Abbiss et al. 2010).

In concurrence with these aforementioned considerations is the previous research generated by Dr James Martin and colleagues. Martin et al. (unpublished-patent pending) provided preliminary data which asserts that athletes may use SL cycling to increase the metabolic capacity of each leg. In one example Martin et al. revealed how a cyclist on a light SL training program (less than 8 hours per

week) achieved lactate thresholds comparable to those of elite cyclists - 77% VO_{2max}. Equally the most recent paper produced by Martin and Colleagues (Abbiss et al. 2010), has added further sub-cellular information to this line of ' SL training' research, with the analysis of muscle biopsies collected from the vastus lateralis (VL) after a consistent 3 wk (2 sessions per wk) high intensity period of SL vs. DL cycling training. These most recent data highlight a specific role for SL cycling regarding an upregulation of certain oxidative enzymes (cytochrome c oxidase subunits II (COX II) and IV (COX IV) and muscle protein concentration which are known markers for increased muscular oxidative capacity and mitochondrial biogenesis.

1.3.3. Proposed implications for a novel training stimulus

Manipulation of the intensity, duration and modality of a training session, changes the relative demands on particular metabolic pathways within muscle cells, as well as oxygen delivery to muscle. In response, different adaptations may occur in both central and peripheral metabolic systems. It is proposed that in comparison to normal DL interval cycle training, high intensity, SL cycle training of each limb independently may produce different and perhaps superior training adaptations (the underlying thoughts will be discussed in more detail below).

Competitive cyclists are usually able to train with a majority aerobic component up to their individual VO_{2max} (Joyner & Coyle 2008; Mortensen et al. 2005), which therefore sets the upper limit for the amount of oxygen delivered to the active muscle mass. However, as previously discussed, during exercise in the normal DL cycling mode oxygen is delivered to both legs simultaneously (perfusing neither leg maximally with oxygenated blood flow), therefore delivery

is thought to be limited by central factors related to insufficient blood flow and oxygen delivery to the active muscle mass (Klausen et al. 1982; Saltin et al. 1976; Secher et al. 1981). Previous research has focussed upon avoiding this central limitation via induction an increased central delivery of oxygenated blood through the alteration of the exercise environment (i.e. using hyperbaric chambers or hyperoxic therapies) (Perry et al. 2007). These methods are thought to induce an O_2 'overload' which is suggested to acutely increase VO_{2max} and therefore allow a higher exercise intensity to be performed. It is this enhanced exercise intensity which is thought to stimulate a number of positive metabolic/oxidative adaptations within the muscle cell which target enhanced mitochondrial oxidative capacity, cellular glucose transport proteins (Glut 4) and potential skeletal muscle blood delivery; thus enhancing peripheral muscular respiratory adaptation (Abbiss et al 2010).

Alternatively, during SL cycling at submaximal values of whole body VO_{2max}, central limitations are nonexistent and the cyclist is able to perfuse the musculature, capillaries and mitochondria of each independent leg with significantly more oxygenated blood at a higher rate without the relative cost implication of having to manipulate the training environment (Davies and Sargeant 1975; Saltin et al. 1976; Klausen 1982). Furthermore, previous training research using a reduced muscle mass has emphasised that in comparison to a large active muscle mass, the perfusion needs of a small active muscle mass can be accommodated by the cardiovascular system with relative ease. This decreased cardiovascular load is also accompanied by a reduction in ventilation, because the ventilatory system is not similarly less taxed by the comparatively small active muscle mass (Klausen et al. 1982; Richardson et al. 1995, 1998; Richardson and Saltin 1998). As a result of the reduced strain on the cardio-respiratory system during SL cycling, increased perfusion and oxygenation of each working limb (when exercised individually), can be

achieved compared to during high intensity DL cycling (Richardson et al. 1999; Harms et al. 1997). The VO_{2peak} of an isolated quadriceps muscle group, demonstrated to be 2-3 times higher than when measured in the same muscle group during whole-body work (Saltin et al. 1976; Davies & Sargeant 1975). Therefore this SL training model has potential to be useful not only in healthy, trained populations but also populations that suffer from cardio- respiratory disorders, which coincide with major central cardiovascular exercise limitations.

In summary, research to date surrounding limitations to bilateral cycling performance suggests that there may be potential for further improvement of oxidative capacity if each leg could be exercised independently, thus potentially eliciting a higher relative training intensity per limb (Abbiss et al. 2010; Neary & Wenger 1986; Stamford et al. 1978; Mortensen et al. 2005;2008). As a result, we propose that during a sustained high intensity cycling effort in the SL mode (the heightened intensity enabled via the addition of a counterweight), individuals may be able to exercise each leg separately at a higher percentage of individual leg VO_{2max} (Neary & Wenger 1986; Stamford et al. 1978; Klausen at al. 1982, Gleser 1973). Reduced muscle mass exercise (SL cycling) as a training modality may induce increased muscle fibre recruitment, plus increased metabolic loading of the peripheral oxidative sub structures within the active musculature. If so, increased targeted peripheral adaptation and potential further development of fatigue resistance and MBC above that demonstrated previously post high intensity DL interval training (Edge et al. 2006; Fitts 1977; Weston 1997; Westgarth-Taylor et al. 1997).

1.4. Modifying the SL Cycling Technique

1.4.1. Introduction of a Counter weight device

Within previous research researchers have either not utilized a form of device to return the 'passive pedal' during the SL cycling exercise or have not used a consistent method (two exceptions detailed below- Bundle et al. 2006; Sheppard 1988). Therefore the SL cycling technique employed in the majority of investigations involved a more powerful upstroke and a less powerful down-stroke; thus muscle recruitment for this type of SL cycling was very different from normal bilateral cycling. Some research groups did utilize a pedal fitting (i.e. a metal clip and shoe fastening/a plimsoll that was actually fitted to the pedal), which allowed participants trained in the SL model to exert more force on the pedal during the upward phase of the pedal stroke (Davies and Sargeant 1975; Gleser 1973). However, despite making the action a little smoother this addition only increased the discrepancy in muscle recruitment during SL cycling in comparison to DL cycling; the irregular pedalling action remained and forced flexion of the active independent leg was increased.

Gleser (1973) did acknowledge the need to support the active limb whilst completing the upstroke of the pedal and therefore piloted a spring device positioned on the underside of the active pedal to help return the pedal to the 'top' position, but this technique was later rejected due to concerns that participants cycling in this mode did not achieve a plateau in VO_{2max}. Finally, Gleser (1973) designed a SL cycling training model which involved two cyclists SL cycling in partnership, each participant acting as the contralateral limb for the other; the idea that they replicated each other's SL crank torque as closely as possible. This model was not very robust

and proved an invalid model to examine SL cycling, as the performance of each cyclist was highly influenced by the combined effort and co-ordination of each partnership.

The introduction of a counterweight device in order to facilitate smoother, more efficient unilateral (SL) ergometer operation was first utilized in a very basic form in preliminary studies investigating muscle mass as factor limiting physical work (Shephard et al. 1988). Subsequent to that a 9.4kg counterweight device was used by Bundle et al. (2006) within a study design which compared force production and neuromuscular compensation during SL cycling and DL sprint cycling. More recently, an increase in the commercial value of SL cycling with a counterweight (SLAM Pedals- Luescher Teknik, Victoria, Australia) has become evident, yet little scientific support for this product has been produced/ released into the public domain. Martin and colleagues have further developed this initial counterweight model in order to investigate whether SL cycling exercise could produce greater gains in DL ventilatory threshold, thus allowing endurance athletes to perform for prolonged periods at close to $VO_{2max,}$ minimizing metabolic stress experienced in the skeletal muscle (pilot data 2009- unpublished).

The addition of a counterweight device is theorized to allow SL cycling to be performed with a metabolic cost and biomechanics that are considered similar to those associated with normal DL cycling. Specifically, the counterweighted contralateral pedal is proposed to allow a smoother, unforced leg flexion more similar to that experienced during normal DL cycling, where both legs are activated simultaneously. This concept is key when comparing the two different training modalities, as previously the two have been incomparable due to previous concerns regarding vastly dissimilar muscle recruitment and neuromuscular co-ordination during SL

cycling without a counterweight device. Currently, we are in agreement with current conjecture highlighting the need of a counter weight (CW) of at least 9-10kg (Thomas 2009, Martin et al. unpublished 2008- Appendix 12) to effectively replicate DL muscle activation.

1.5. Hypotheses:

To our knowledge, no previous research comparing the training response to DL vs. SL cycle interval training (with a counter weight device), upon endurance performance has been implemented amongst well trained cyclists. Therefore, the purpose of this study is to test the hypothesis that incorporating high intensity SL cycling endurance training within an existing DL training regime (with a counter weight device), improves cycling TT performance more than equivalent (energy expenditure matched) interval training in the DL mode in well trained cyclists. We postulate that cyclists training in the DL mode (where both legs are working simultaneously) are unable to produce the maximal possible training intensity/ stimulus for positive aerobic adaptation and peripheral fatigue resistance, due to central limitation. Consequently, we hypothesise that aerobic high intensity SL interval cycling will maximally challenge the peripheral muscles without gaining a central limitation (due to the reduced active muscle mass), and thereby enable a significantly greater whole body maximal aerobic training response.

Hypothesised adaptations to SL cycle training include: a superior increase in power output recorded at VO_{2max} (W_{max}), plus an increased peripheral fatigue resistance evident via an increased average power output when performing a sustained high intensity effort and

potentially increased muscle lactate buffering capacity (decreased lactate appearance for a given sub-maximal exercise intensity). These proposed metabolic adaptations culminating in a superior improvement in cycling time trial (TT) performance (TTC) in the SL trained participants, than that demonstrated post aerobic high intensity DL interval cycling. These proposed SL training adaptations are independent of any improvement in DL VO_{2max}, however an increase in maximal oxygen consumption in the SL mode is expected.

Chapter 2.0

Methodology

2. Methodology

2.1. Participants

Fourteen trained cyclists (2 f), (mean ± SD: 33 ± 10y, 73 ± 5.6kg, W_{max} = 335 ± 33 W, VO_{2max} = 62 ± 5.9 ml·kg $^{-1}$· min^{-1}, cycling experience = 8± 5y), with prior TT experience were recruited within a prospective, non randomized comparative design and carefully assigned in equal number to either the SL or DL training group, respectively) training group. Unfortunately due to illness, four subjects were excluded from the final data set. Groups were matched for gender, body mass, training load and fitness levels. All participants had a high level of aerobic fitness (VO_{2max} > 55 ml.kg bw.min^{-1}); as well as a minimum of four years competitive cycling experience, plus a minimum cycling training load of ~5h.wk.

Participants were recruited via web advertisements and word of mouth in local cycling and triathlon clubs. All subjects were deemed healthy, as assessed by the General Health Questionnaire of the School of Sport and Exercise Sciences. Any potential participants demonstrating any of the contra-indications to exercise listed in the code of practice for exercise testing were not tested. Participants were also excluded if taking supplements considered to have the potential to impact on the training intervention (e.g. Anabolic steroids, B-alanine, nitrate and creatine). All subjects were given a subject information sheet explaining the aims, risks and protocol of the study. Written consent to participate in the study was provided by each subject prior to study commencement. Ethical approval was obtained from the [redacted]

2.2. Experimental design

Each participant completed four different performance tests pre and post a four week training intervention period comprising of 12x 1h training sessions (3 sessions per wk); alongside their habitual training. These four performance tests included: a DL cycling VO_{2max} test, a SL cycling VO_{2max} test with the right leg, a SL cycling VO_{2max} test with the left leg, and a laboratory cycling time trial (these tests will be described in more detail below).

Within the 'pre' test week (Week 1), participants completed 24h diet records prior to each preliminary performance testing session (all of which were completed in a fasted state), they were subsequently instructed to repeat this diet exactly prior to each of the concurrent post tests. In addition, throughout the study period participants were required to keep a detailed record of their additional personal training for subsequent analysis. The subjects were asked to maintain the average number of training hours and level of intensity (per week) both prior to and throughout the study period.

Preliminary testing, plus SL cycling familiarization sessions were all completed at least two days, but no more than four days prior to the first training session. In accordance post-testing followed a similar trend, all post tests commenced three days following the final training session performed within the laboratory.

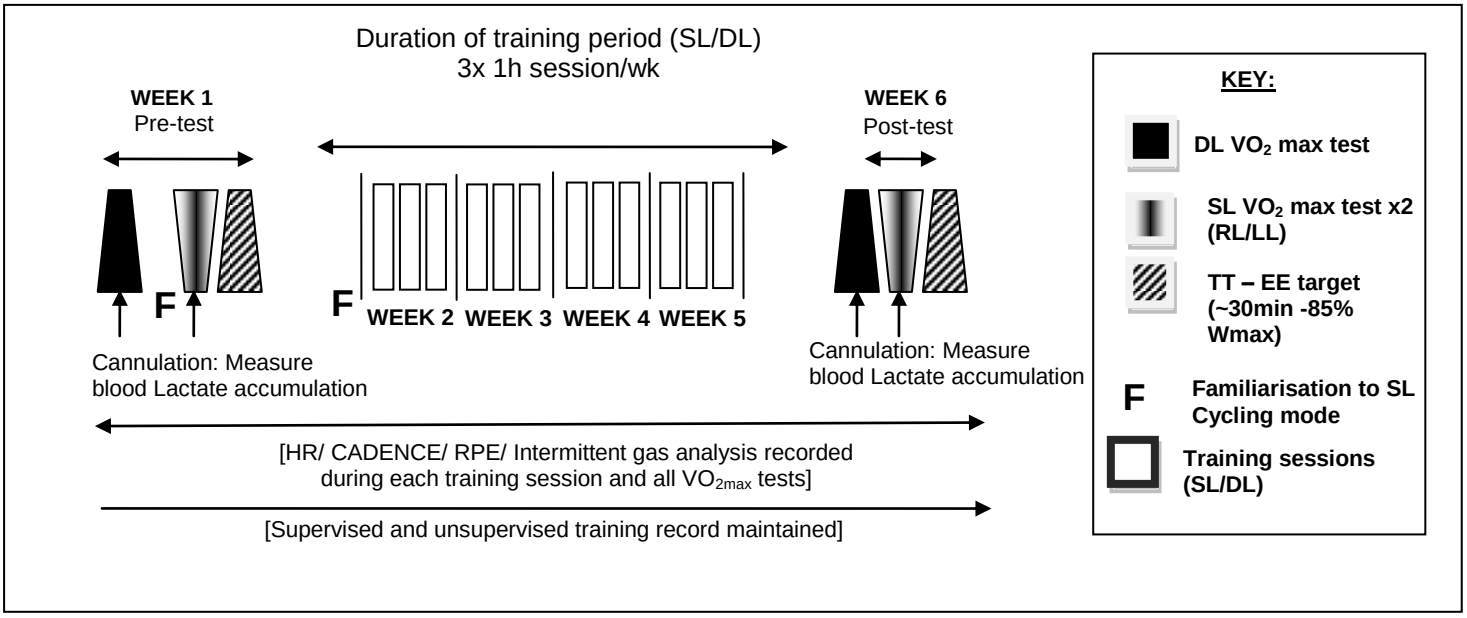

Figure 2.1: Main Study design- Double legged vs. Single legged cycle training study.

Participants completed the DL VO_{2max} test, followed by independent right leg (RL) and left leg (LL) single-legged tests (48h post). Each of the single legged VO_2 max tests (left leg and right leg) were separated by 15 min of recovery, plus the order that they were completed in randomized. Lastly (72hr post the SL max tests), participants each completed a simulated ~ 30min time trial (TT), within the laboratory under controlled and standardised conditions. Trials were performed in silence, in a small testing laboratory with a screen placed behind each participant to minimise distraction. Both the preferred bike set up and laboratory environment were recorded during the preliminary trial and closely replicated during the post trial (further details below- 2.3.2). Before the start of the experimental trials all participants were familiarized with the laboratory setting, test bike (used for all laboratory trials and training - Lode Excalibur Sport, Groningen, The Netherlands) and testing protocols. Specifically between the DL VO_{2max} and the SL VO_{2max} each participant was familiarized with the new modified SL

exercise bike and the SL cycling technique/sensation. All participants had prior competitive TT experience.

2.3. Main comparative performance measures

2.3.1. Incremental cycling exercise test to exhaustion (VO_{2max}):

Both prior to and post the four week training intervention each subject was required to complete three incremental cycle exercise tests to volitional exhaustion in order to determine the individual maximum power output (W_{max}) and maximal oxygen consumption (VO_{2max}) derived whilst cycling with both legs, plus RL and LL alone. Breath by breath analysis using an online automated gas-analysis system (Oxycon Pro, Jaeger, Wuerzberg, Germany) was implemented throughout all maximal tests and training protocols in order to ensure accurate gas analysis (see below: 'Respiratory breath analysis'). Oxygen consumption (VO_2) was considered to be maximal (VO_{2max}) when the two following criteria were met: 1) Oxygen uptake (a plateau in VO_2) (< 150 ml.min and < 2 ml.kg.min), 2) Respiratory Exchange Ratio (>= 1.10) and 3) Heart Rate (>= 95% HRmax) (Casajus et al. 2009; Robergs et al. 2010). VO_{2max} was calculated as the average oxygen uptake over the last 60 s of the test. W_{max} was calculated according to the following formula:

$$\mathbf{W_{max} = W_{final} + (t/T)*W}$$

Where W_{final} (W) is the final stage completed, t (s) is the amount of time reached in the final uncompleted stage, T (s) is the duration of each stage and W (W) is the workload increment (Jeukendrup et al. 1996).

All tests were performed on an electromagnetically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands), modified to the configuration of a racing bicycle with adjustable saddle height and handlebar position. Additional modification was employed to the two SL cycle ergometers, which were both fitted with specially adapted cranks and 10kg counterweights on contralateral pedal axis. Each modified cycle ergometer was set up in collaboration with a small stool (76cmH x 32cmW x 24cmD), complete with waterproof cushion and positioned behind the counterweighted pedal to the side of the bike in order to support the inactive limb. Care was taken to keep the resting leg as inactive as possible but, as pointed out by Ahlborg, Hagenfeldt & Wahren (1975), even with this precaution, some muscular activity and metabolic alterations in the non-exercising leg cannot be totally avoided. Prior to each test height and body mass (Seca Alpha, Hamburg, Germany) were recorded and bike set up was noted for comparison and reproducibility in the concurrent post test.

During all tests heart rate (HR) was recorded continuously by a radiotelemetry HR monitor (Polar Vantage NV, Kempele, Finland). RPE was recorded at the end of each completed stage. During each SL VO_{2max}test care was taken to clarify that instead of acknowledging whole body RPE, participants should focus on the working limb specifically. Also throughout each maximal test cadence (CAD) was observed and averaged over the last min of each stage. In addition, during each test it was required to collect a 2ml blood sample from each participant (via a plastic cannula inserted into the antecubital vein), both prior to plus immediately post test, as well as at the end of each complete stage in order to ascertain blood lactate (LA) accumulation values.

- ***Double legged Cycling VO_{2max} test:*** Participants started cycling at 95 W for 180 s, followed by sustained incremental steps of 35 W every 180 s until volitional exhaustion (i.e. when the athlete could no longer maintain a cadence >60 rpm).

  The $VO_{2\ max}$ and W_{max} achieved during the incremental exercise tests were 63 ± 5 ml·kg body wt^{-1}·min^{-1} and 338 ± 34 W, respectively. The maximal results from the DL VO_{2max} test were then used to determine the work rate corresponding to 35% and 70% W_{max}; values later employed in the experimental exercise trials.

- ***Single leg Cycling VO_{2max} test (RL/LL):*** Participants started SL cycling at 66 W for 120 s, followed by increments of 16 W every 60 s until volitional limb exhaustion (as defined above). During the SL VO_{2max} test the 'resting' leg was supported by a padded stool placed behind the counterweighted pedal (figure 3). Participants were required to rest there shin on top of the aforementioned stool, in a position that minimized the contribution of stabilitory musculature O_2 consumption and additional muscle recruitment for increased force production. Blood samples were collected every 2 minutes rather than at the end of each completed stage, until exhaustion. The $VO_{2\ max}$ and W_{max} achieved during the incremental SL exercise tests to exhaustion were 45 ± 5 ml· kg bw^{-1}·min^{-1} and 210 ± 20 W, respectively.

Figure 2.2: SL Subject completing a SL $VO_{2\ max}$ test on the specifically modified Lode cycle ergometer, complete with contralateral counterweight device.

2.3.2. Cycling time trial (TT):

During this study each participant performed two simulated TTs, pre and post a 4 week training intervention. Each TT was preceded by a 10 min warm-up period (5 min at 35% and 5min at 50% DL W_{max}). After the warm-up, the ergometer was set in the linear mode so that by increasing pedalling rate, the work rate increased, in accordance with the following formula:

$$W = L * (RPM)_2$$

In which RPM is the pedaling rate and L is a (constant) linear factor. Hence, the work rate (W) measured in watts was equal to the cadence^2 $(RPM)^2$ multiplied by the linear factor (L). Using the above equation L can be calculated by W/ $(RPM)^2$.

The linear factor is based on individual W_{max} and was calculated so that 85% W_{max} will be produced at a pedalling rate of 90rpm. Participants were asked to complete a set amount of work (energy expenditure based target) in as short a time as possible, consequently eliciting a maximal effort on the part of each cyclist. Total work to be completed was individualised, equivalent to working at 85% W_{max} for 30 min and was calculated according to the equation adapted from Jeukendrup *et al.* (1996):

Total amount of work (kJ) = 0.85 * Wmax (W) * 1800(s)

Where 0.85 refers to a factor equivalent to 70%, W_{max} (W) is the maximum wattage attained during the incremental test in watts and 1800 refers to the number of seconds that make up 30 min.

Participants completed the TT in a separate laboratory in complete silence, no encouragement, nor verbal or visual feedback related to performance of physiological variables

was communicated. In addition a screen was erected behind the participant to avoid any distraction. A computer was connected to the ergometer and work, power and time were recorded; however minimal information was displayed to the participants in order to ensure internally set self pacing. The only feedback conveyed to the athletes was an unlabelled computer generated visual model depicting the amount of work performed in relation to the total amount of work to be completed. Any changes in time trial performance in response to the training intervention will be determined by examining changes from baseline (pre TT) relating to average power output (PO) and time taken to complete (TTC) the set amount of work for each subject. All participants were required to be overnight fasted prior to each test. Also heart rate (HR) and cadence (CAD) was monitored continuously throughout all tests and averaged over the last minute of each 5min stage of the TT, and the last min of each incremental stage of the differing VO_{2max} tests. To minimise thermal stress, a floor-standing fan was positioned in front of cyclists and water was provided *ad libitum* during all performance tests and training sessions.

2.4. Training intervention

2.4.1. Protocol definition

Participants completed their regular base training, however on three days of each training week the subjects' normal training was replaced with 3x 1 h lab based cycle training in either the SL or DL condition. Participants were asked to use each laboratory based training session as a substitute for an existing session, rather than a supplement to their habitual training programme to ensure habitual training load was maintained. To monitor training outside of the laboratory, training diaries were completed by all participants. All laboratory-based training

was carried out, under supervision in the Human Performance Laboratory, within The School of Sport and Exercise Sciences.

DL and SL participants performed the first training week cycling at 35% and 70% of their predetermined DL W_{max} respectively. All training sessions were ~ 1h in duration and performed at a self-selected cadence. Exercise training intensity increased by 6% and 3% each week, for both the DL and SL groups, respectively. DL training group participants performed 6 x 5 minute intervals with both legs (including 5 min rest between each effort), whereas SL participants continuously alternated the exercising leg every 5 min (swapping between two Lode cycle ergometers, especially modified for SL cycling- the opposing pedal loaded with a 10kg counter weight).

Heart rate measurements, rate of individual perceived exertion, rate of perceived leg effort and cadence were recorded throughout the training period (always in the last 30 seconds of each interval). In addition at specified intervals during the training session (see Figure 4), 5 min gas samples were collected using an online gas analysis system (Oxycon Pro, Viasys, Wuerzberg, Germany), thus allowing determination and comparison of values of O_2 consumption. Comparisons of DL cycling performance both pre and post the training intervention were utilized in order to ascertain whether any performance improvement had been observed over the 4 week training intervention, in either group.

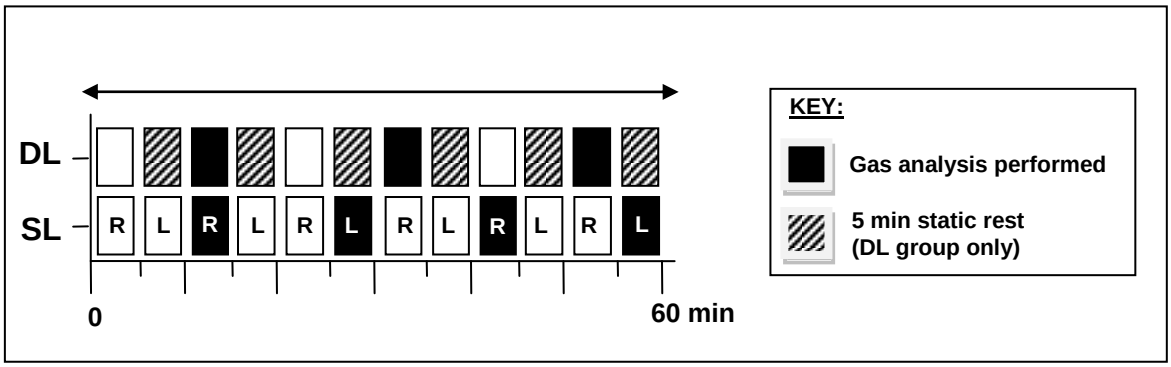

Figure 2.3: Plan of intermittent gas analysis during DL and SL training sessions respectively.

2.4.2. Relative training intensity

Within the SL (single legged cycling) condition, participants trained three times each week, each training session lasting one hour. Each session SL participants cycled twelve intervals in total comprising of 6 x 5 minutes right leg only (RL) and 6 x 5 minutes left leg only (LL). Training intensity was increased each week (refer to table below).

Training Week 1	5 min cycling RL / 5 min cycling LL	Training intensity: 35% pre DL W_{max}
Training Week 2	5 min cycling RL / 5 min cycling LL	Training intensity increased by 3%
Training Week 3	5 min cycling RL / 5 min cycling LL	Training intensity increased by 3%
Training Week 4	5 min cycling RL / 5 min cycling LL	Training intensity increased by 3%

Table 2.1: SL training protocol (over the four week training intervention). Describing the relative increases in training intensity per week.

Whereas, within the DL (double legged cycling) condition, participants trained three times each week, each training session lasting one hour. Each session DL participants cycled six

intervals in total comprising of 6 x 5 minutes double legged, between each interval they rested in a static position. The DL condition was matched with the SL condition for work done during the session, therefore training intensity was also increased each week during the training intervention (refer to table below).

Training Week 1	5 min continuous DL cycling (5 min rest)	Training intensity: 70 % pre DL W_{max}
Training Week 2	5 min continuous DL cycling (5 min rest)	Training intensity increased by 6%
Training Week 3	5 min continuous DL cycling (5 min rest)	Training intensity increased by 6%
Training Week 4	5 min continuous DL cycling (5 min rest)	Training intensity increased by 6%

Table 2.2: DL training protocol (over the four week training intervention). Describing the relative increases in training intensity per week.

2.5. Data Collection

2.5.1. Respiratory breath analysis

Breath-by-breath measurements were performed throughout exercise by using an online automated gas-analysis system (Oxycon Pro, Jaeger, Wuerzberg, Germany). During collections participants wore a nose clip and breathed through a specialized, lightweight mouth piece which was connected to a sample twin tube and volume transducer. Expired gas was delivered, via the twin tube, to the fast response paramagnectic and infrared analysers which determined $O_{2\ and}$ CO_2 concentrations respectively. The volume sensor was calibrated by using a 3-liter calibration syringe (Hans Rudolph 5530 3-l syringe), and the gas analyzers were calibrated by using certified reference gases (4.95% $CO_{2,}$ 95.05% N_2 BOC Gases, Surrey, UK). The Oxycon was used in order to

carry out an accurate and efficient analysis of a multiple of respiratory values; gaseous concentrations and airflow values synchronized via computer software. In analysis gaseous data was averaged every eight breaths, including: Minute ventilation (VE L.min), Volume of oxygen consumed (VO_2 ml.min), Volume of carbon dioxide produced (VCO_2 ml.min), Oxygen uptake (ml.min.kg body mass) and Respiratory exchange ratio (RER- the ratio of volume of carbon dioxide produced to the volume of oxygen utilized). Data collected was then further analyzed according to specific time intervals; aforementioned values calculated for the last minute in each progressive stage; until exhaustion was reached. From this data we were able to accurately calculate VO_{2max} and $VO_{2\ peak}$ specifically.

2.5.2. Blood data analysis

Blood was collected from a cannula inserted into an antecubital vein during both the SL and DL VO_{2max} tests. In the final 30 s of each stage (set exercise intensity) and at immediate cessation of the test, a blood sample (~2 ml) was collected in pre-chilled vacutainers containing 20mM K3 ethylenediamine tetraacetic acid (EDTA) (Sigma, Dorset, UK); for subsequent plasma lactate analysis. The samples were centrifuged immediately at 4°C for 10 min at 3,500 rpm and the supernatant split into aliquots related to time points within the test/training protocol and frozen at -20^{o}C until further analysis. Plasma lactate concentrations were determined in duplicate spectrophotometrically (Lactate reagent, ABX Diagnostics). This method allows lactate dehydrogenase (LDH) activity to be evaluated by monitoring the NAD^+ reduction at 340 nm, in accordance with the below reaction:

$$\textbf{Lactate} + \textbf{NAD}^{+} \xrightarrow{\text{LDH}} \textbf{Pyruvate} + \textbf{NADH} + \textbf{H}^{+}$$

The change in absorbance with time due to the conversion of NAD to NADH is directly proportional to LDH activity. In order to determine lactate concentration via this method we used an automated spectrophotometric analyzer (Cobas Mira Bio, Roche, Basel, Switzerland) with commercially available reagents (ABX Diagnostics Lactate Reagent Kit, cat. no. A11A01721). The reagents, standards and quality assurance materials were all prepared according to the manufacturer's instructions. Once determined, plasma lactate concentrations were then plotted against exercise intensity (W), Maximal values of lactate accumulation were, individually determined and compared within individual pre-post tests in order to ascertain whether or not any potential augmentation in blood lactate appearance was evident.

2.4.3. Training Records

The participants were asked to maintain a steady level of training from one week prior to the first familiarization session until completion of the study, participants replacing similar training sessions within their normal routine with laboratory based interval sessions. Participants were provided with daily logs in which they recorded their daily training information (frequency, intensity, duration and type). To compare the total training load among groups prior to and during the intervention. Subsequently the training impulse (TRIMP) (Foster et al. 2001) was calculated which was expressed as a product of stress (duration of activity) and strain (subjective rating of training intensity related to heart rate).

2.4.4. Statistical analysis

Before all statistical analyses, data were checked for violations of normality using a Shapiro–Wilk test. Statistical analyses were performed by using SPSS for Windows version 15.0 software package (Statistical Package for Social Science, Chicago, IL). Table values are expressed mean percentage change and data variability as standard deviation (SD). Statistical significance was set at $P < 0.05$. Differences between pre- and post- test mean values of the different variables were calculated and a Student's t-test for paired samples was used to test if significant changes occurred in time for each group separately. Also, the percentage change over time was calculated for each subject individually, and a one sample t-test was completed in order to test whether the mean values of the group differed significantly.

Chapter 3.0

Results

3. Results

3.1 Participants

3.1.1. Individual Characteristics

Table 1 below presents the physiological characteristics of the participants recorded prior to the four week training intervention. At baseline (i.e. pre-test phase) there was no significant difference in $VO_{2\,max}$ and W_{max} between groups. As closely as was possible both groups were matched for gender, $VO_{2\,max,}$ weight, level of cycling experience and age. Despite a loss of four participants due to illness (SL = -3, DL = -1), there were no significant differences between groups related to the physiological or performance variables measured.

Subjects	*Sex*	*Age*	*Weight*	*Cycling experience*	*VO_2 max*	*TT TTC*
	m/f	**(y)**	**(kg)**	**(y)**	**(ml/kg/min)**	**Time to Complete (sec)**
SL	1f, 4m	31.4 ± 7.5	76.4 ± 5.9	6.8 ± 2.4	63.9 ± 5.1	1839 ± 134.7
DL	1f, 5m	32.3 ± 8.0	69.3 ± 4.9	8.3 ± 7.3	62.3 ± 5.2	1802 ± 75.7

Table 3.1. Physiological characteristics of participants. Mean values ± SD.

3.1.2 Physiological parameters prior to training

The average $VO_{2\,max}$ during maximal SL cycling with a 10kg counterweight (3.27 $L.min^{-1}$) was lower than that recorded during maximal DL cycling (4.54 $l.min^{-1}$). The maximal HR recorded during SL cycling was on average lower (~18 $beats.min^{-1}$). W_{max} in the SL $VO_{2\,max}$ test (210 ± 4 W), was approximately 128 W less than that reached concurrently in the DL $VO_{2\,max}$ test (refer to the comparison of maximal SL vs. DL values below- Figure 1.1).

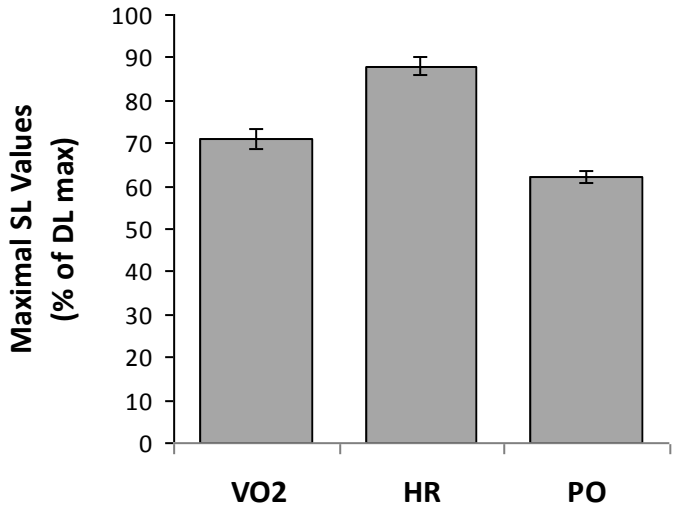

Figure 3.1. A comparison of maximal SL cycling values displayed as a percentage of maximal DL cycling values reached during corresponding VO_2 max test conditions prior to start of the training programme. Where SL is single leg cycling and DL is double leg cycling. Data are mean values ± SD.

Sub-maximal plasma lactate concentrations were higher (~ 17%) at 130W in the SL $VO_{2\ max}$ test condition (2.61 ± 0.2 mmol.l), in comparison to 130W in the DL $VO_{2\ max}$ test condition (1.37 ± 0.1 mmol.l). Peak lactates in the SL condition were found to be lower (~14%), in comparison to maximal lactates taken at the end of the DL $VO_{2\ max}$ test (10.6 ± 0.7 mmol.l and 12. 4 ± 0.7 mmol.l).

3.2. Training Intensity

The mean training intensity for each group over the training intervention period is displayed in Table 3.2 below. Data are present for the first and last training weeks (Wk 1 vs. 4), in order to give an indication and allow comparison between the increase in training load (~25%) for both training groups throughout the study. The values are represented as a percentage of maximal SL and DL performance values (VO_{2max} / W_{max}/ HR_{max}).

Training Intensity	*SL Group*					*DL Group*	
	L	R	AV	**% DL max**	**% SL max**	AV	**% DL max**
Training PO (Watts)							
W1	125 ± 13	125 ± 13	125 ± 13	**35 ± 0.0**	**58 ± 2.5**	225 ± 14	**70 ± 0.0 ***
W4	158 ± 17	158 ± 17	158 ± 17	**44 ± 0.0**	**73 ± 1.7**	282 ± 17	**88 ± 0.0 ***
Training VO_2 (l/min)							
W1	2.29 ±0.22	2.20 ± 0.20	2.25 ± 0.24	**47 ± 2.0**	**67 ± 7.5**	3.48 ± 0.32	**81 ± 3.7 ***
W4	2.88 ± 0.38	2.81 ± 0.37	2.85 ± 0.36	**59 ± 1.6**	**85 ± 8.4**	4.26 ± 0.41	**99 ± 3.9 ***
Training HR (bpm)							
W1	121 ± 17	119 ± 17	120 ± 16	**65 ±7.9**	**76 ± 10.2**	146 ± 6	**83 ± 2.8 ***
W4	138 ± 12	136 ± 14	137 ± 12	**74 ± 5.2**	**87 ± 7.7**	165 ± 11	**93 ± 2.9 ***
Training CAD (rpm)							
W1	96 ± 17	98 ± 15	97 ± 16	**104 ± 9**	**106 ± 6**	99 ± 7	**108 ± 7**
W4	97 ± 8	98 ± 6	97 ± 7	**105 ± 9**	**108 ± 12**	101 ± 6	**112 ± 13**
Training RPE							
W1	14 ± 1	14 ± 1	14 ± 1	-	-	12 ± 1	-
W4	16 ± 1	15 ± 1	15 ± 1	-	-	16 ± 1	-

Table 3.2. A comparison of group average measurements of training intensity achieved in week 1 and week 4 during the training each intervention; for both the SL and DL training groups. Raw data are also expressed as a percentage (%) of preliminary DL or SL maximal values for easier comparison between groups. Where VO_2 is oxygen consumption; HR is heart rate (beats.min^{-1}); CAD is cadence (revolutions.min^{-1}); and RPE is perceived (leg) effort. Data are expressed as mean values ± SD. *Significantly different from SL training.

There was no significant difference in absolute or perceived training intensity between limbs in the SL group. Oxygen consumption, heart rate (bpm), cadence (rpm) and RPE remained similar between limbs throughout the four week training intervention (see Table 3.2).

On average the training intensity in the DL vs. SL training group was significantly different. Throughout the training intervention the DL training group worked at a higher absolute level of oxygen consumption (VO_2) (t(1) = 1.943 , p <0.001) , power output (PO) (t(1) = 2.015, p <0.001) heart rate (HR) (t(1) = 1.780 , p <0.001) and cadence (CAD) (t(1) = 2.131, p = 0.021) (see Figure 3.2 below).

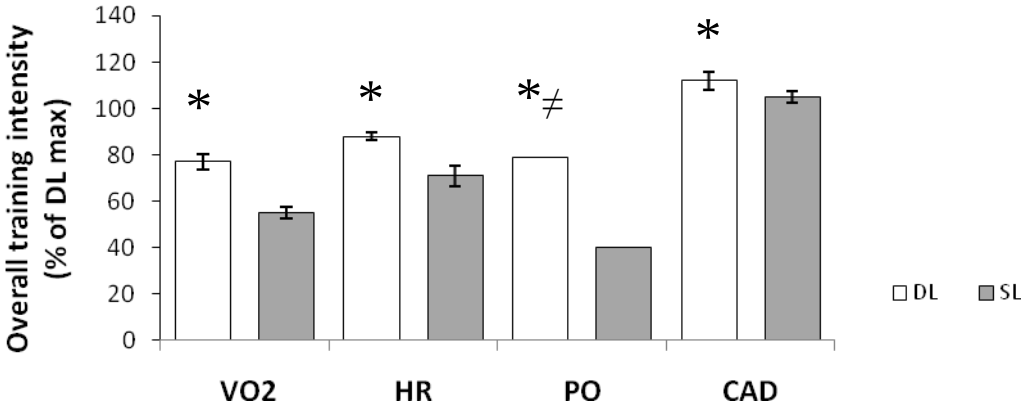

Figure 3.2. Average training intensity values for both SL and DL training groups. Data displayed for comparison as percentage of maximal DL cycling values recorded in the preliminary DL VO_{2max} test. Mean values ± SD. *Significantly different from SL training intensity. ≠No standard deviation [SD] reported as work rate in both trial interventions was set as a percentage of DL power output.

However, if the relative oxygen consumption per limb is considered (i.e., VO_2 values are halved for the DL training group, to VO_2 estimate muscle mass specific work) then it is clear that participants in the SL group worked at a significantly higher relative (muscle mass specific) work intensity throughout the training intervention (higher percentage preliminary DL VO_2max) (t (1) = 1.943 p = <0.01) (refer to Figure 3.3).

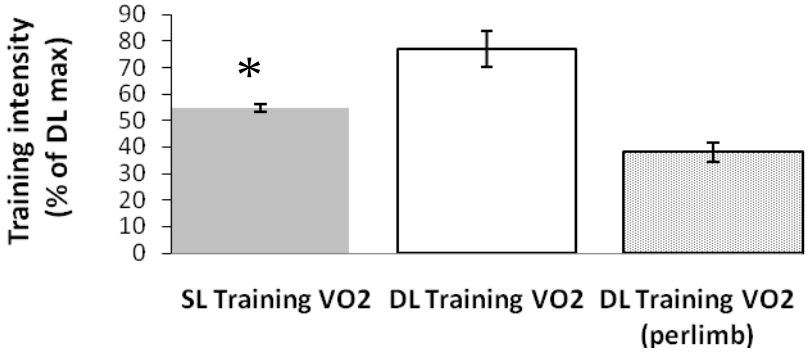

Figure 3.3. A comparison of average levels of oxygen consumption (VO_2) achieved during each training intervention. Actual data for SL and DL training are displayed against estimated DL (per limb) training oxygen consumption (VO_2). All data are presented as percentage (%) of preliminary DL VO_2max. Where SL is single leg trained; DL is double leg trained; and DL (per limb) is 50% DL training data. Data are mean values ± SD. * Significant difference from estimated (per limb) DL training oxygen consumption (VO_2 / limb).

Overall, the total personal training load per week was calculated via a measurement of training impulse 'TRIMP' data related to individual participant training regimes (refer to methodology- Foster et al .2001). The weekly training load both prior to and during the training intervention was calculated as similar within both groups. (SL training group: pre 1004 ±202, during 1049 ± 263; DL training group: pre 986 ± 168, during 1002 ± 242; mean ± SD). Participants maintained training load during the study at a similar level both prior to and during their study involvement, replacing a portion of their current base training with laboratory based interval training.

3.3 Training response

3.3.1 Double legged $VO_{2\ max}$ test

Performance Variables	SL Training group			DL Training Group		
	PRE	POST	% Change	PRE	POST	% Change
DL VO_2 max Performance						
VO_2max (l.min)	4.81 ± 0.53	4.95 ± 0.58	**2.9 ± 5.4%**	4.32 ± 0.54	4.53 ± 0.59 *	**4.6 ±1.6 %**
Wmax (Watts)	359 ± 38	363 ± 39	**1.3 ± 2.1%**	321 ± 20	330 ± 18 *	**3.0 ± 1.5%**
HR max (bpm)	182 ± 10	182 ± 12	**0.0 ± 2.3%**	177 ± 10	178 ± 10	**0.7 ± 1.7%**
Av CAD (rpm)	93 ± 8	93 ± 5	**0.0 ± 2.8%**	91 ± 4	95 ± 6 *	**4.4 ± 1.9 %**

Table 3.3. A comparison of physiological variables tested during the Double legged cycling $VO_{2\ max}$ test. Values are displayed as group averages and are compared pre and post training intervention. Percentage change (% Change) between values pre-post is displayed for each of the different training groups (Single leg (SL) trained v.s. Double leg (DL) trained). * Significant difference from pre training values.

Maximal oxygen consumption* (VO_{2max})*:

VO_{2max} improved significantly post training in the Double legged intervention group (DL) (t = -5.832, p = 0.001). A 2.9% improvement was observed in the SL group, which did not reach significance (t= -0.988, p = 0.189) (refer to Table 3.3). Despite the significant improvement observed in the DL group, an independent T-test showed no difference in the changes observed in $VO_{2\ max}$ (pre vs. post) between the two training groups (SL vs. DL) nor between groups at either time point (pre- or post-training).

Maximal power output (W_{max}):

Similarly to maximal oxygen consumption (VO_{2max}), only the DL training group achieved a significant improvement (pre-post training) in power output at $VO_{2\ max}$ (W_{max}) (t(1)= -4.758, p= < 0.01). A non-significant improvement was demonstrated in the SL trained group (t(1)=0.137, p= 0.136).

Maximal heart rate and cadence:

Maximal heart rate (HR_{max}) recorded during the DL VO_{2max} test was the similar both before and after training regardless of training intervention. Average cadence (av CAD) during the post training DL VO_{2max} test was only significantly increased in the DL training group ($t(1)= -5.477$, $p = <0.01$), whereas within the SL group there was no increase (pre – post training); thus displaying a significant difference in training response with regard to cadence (refer to Table 3.3).

3.3.2. Single legged VO_2 max test

Performance Variables	***SL Training group***			***DL Training Group***		
	PRE	POST	**% Change**	PRE	POST	**% Change**
SL VO_2 max Performance						
VO_2 max (l.min)	3.54 ± 0.43	3.63 ± 0.25	**3.1 ± 8.2 %**	3.12 ± 0.41	3.15 ± 0.32	**1.3 ± 5.6 %**
Wmax (W)	216 ± 26	227 ± 25 *	**4.9 ± 5.6 %**	204 ± 12	208 ± 8 *	**2.2 ± 3.4 %**
HR max (bpm)	162 ± 15	165 ± 10	**2.2 ± 4.6 %**	161 ± 11	159 ± 10	**-0.7 ± 3.7 %**
Av CAD (rpm)	91 ± 12	95 ± 6 *	**5.1 ± 0.1 %**	90 ± 7	89 ± 4	-2.6 ± 0.1 %

Table 3.4. A comparison of physiological variables tested during the Single legged $VO_{2\,max}$ test. Values are displayed as group averages and are compared pre and post training intervention. Percentage change (% Change) in values pre-post is displayed for each of the different training groups (Single leg trained v.s. DL trained). * Significant difference from pre training values.

Maximal single-legged oxygen consumption* (VO_{2max}*):

On average single legged VO_{2max} (right leg (RL) and left leg (LL) VO_{2max} averaged), was not improved significantly post either the DL training intervention ($t(2) = 0.2829$, $p = - 0.614$), or the SL training intervention ($t(2) = 0.3255$, $p = - 0.488$) (refer to Table 3.4). There was no significant difference in SL maximal oxygen consumption between groups prior to or post either training intervention. An overall trend for a higher preliminary RL VO_{2max} , compared to LL VO_{2max} ,was

evident in both training groups, which reflected all participants right leg dominance; however this trend failed to reach statistical significance (p = 0.12) (see Table 3.5). No statistically significant difference was reported between training groups pre or post intervention.

SL VO_2 max (l.min)	***Right Leg (RL)***		***Leg Leg (LL)***	
	Pre	**Post**	**Pre**	**Post**
SL Training Group	3.62 ± 0.35	3.61 ± 0.57	3.46 ± 0.54	3.64 ± 0.57
DL Training group	3.20 ± 0.45	3.19 ± 0.37	3.06 ± 0.37	3.09 ± 0.27

Table 3.5. A comparison of single leg values of maximal oxygen consumption ($VO_{2\,max}$) recorded during the right legged (RL) and left legged (LL) $VO_{2\,max}$ performance test. Values are displayed as group averages and are compared pre and post differing group training intervention. Mean values ± SD.

Maximal single- legged power output (W_{max}):

Within the single leg test condition, on average, both SL and DL training intervention groups respectively achieved a significant improvement (pre-post training) in maximal power output (W_{max}) ($t(1) = 2.258$, $p = 0.03$; $t(1) = -2.132$, $p < 0.05$). Within the SL trained cyclists there was a trend for a higher percentage increase in SL W_{max} (pre-post intervention) ($p = 0.239$) (refer to Table 3.4). However, there was no significant difference between groups either pre or post either training intervention.

When considering each leg separately, despite controlling for order effects, a trend (in both groups) for lower preliminary left leg (LL) W_{max} values was evident, however this trend failed to

reach significance (t (1) = 2.080, p = 0.064). No significant difference in preliminary W_{max} values was reported between groups. Post training intervention a significant increase in LL W_{max} was only identified within the SL training group (~ 11W) (t(1) = -2.048, p 0.05), a comparable trend for improvement within the DL training group was also evident however this trend failed to reach statistical significance (t(1) = -1.727, p = 0.072). Neither training group reported a significant improvement in RL W_{max} post training intervention.

Maximal heart rate and cadence:

Post the training intervention period maximal heart rate recorded during the single legged (SL) VO_{2max} test was significantly increased in neither the SL/ DL training group. A significant increase in cadence during the post SL VO_{2max} tests was observed only in the SL training intervention group (t (1) = 1.833, p = 0.01), there was no significant increase reported in the DL training group (p = 0.256). Cadence the only variable to again exhibit a significantly different training response between groups (refer to Figure 3.4).

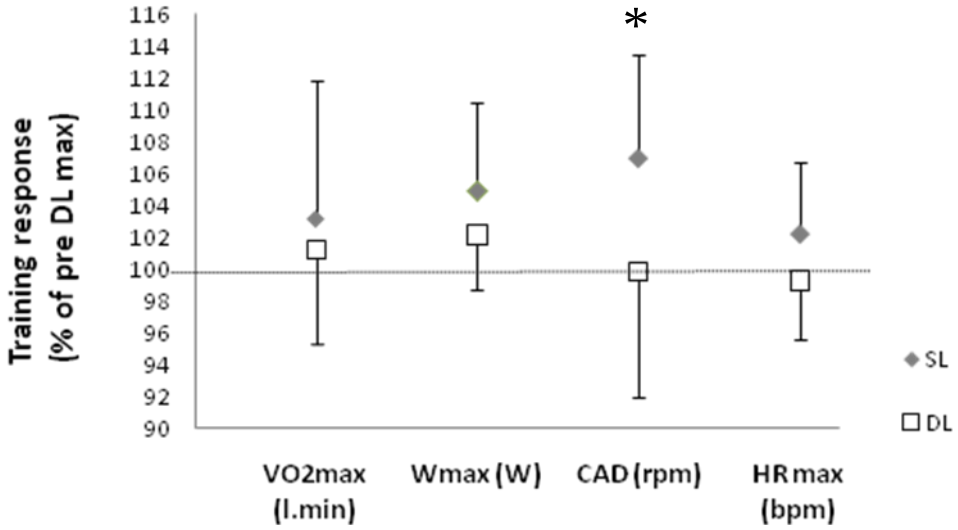

Figure 3.4. Variation in response to training for variables measured during the SL VO_2max test. Data points represent the mean training adaptation for each group (SL/DL), calculated from averaging individual participant post -training values as a percentage of their individual pre-training value (i.e., 90% represents a 10% decrease, 110% represents a 10% increase). Mean values ± SD. * Significant difference from DL group training response.

3.3.3 Plasma Lactate Concentrations

Performance Variables	*SL Training group*			*DL Training group*		
	PRE	POST	**% Change**	PRE	POST	**% Change**
DL VO_2 max Performance						
Max Lactate (mmol.l)	12.4 ± 1.7	13.1 ± 1.6	**5.4 ± 26 %**	12.5 ± 1.7	13.1 ± 2.4	**5.5 ± 21%**
SL VO_2 max Performance						
Max Lactate (mmol.l)	10.3 ± 2.4	11.3 ± 2.3	**9.8 ± 12% ***	11.0 ± 2.1	11.7 ± 1.6	**6.4 ± 16 %**

Table 3.6. A comparison of blood lactate values collected during the maximal incremental double legged/ single legged performance tests. Values are displayed as group averages and are compared pre and post differing training intervention. Percentage change (% Change) in values pre-post is displayed for each of the different training groups (Single leg trained v.s. Double legged trained). * Significant difference from pre training values.

The plasma lactate values obtained during the incremental DL and SL exercise test (VO_{2max}) both demonstrated a characteristic curvilinear increase with increasing intensity. Maximal plasma lactate concentrations were higher after maximal DL exercise, than after SL exercise (p= 0.02) (refer to Table 3.7). Conversely, a comparison of plasma lactate samples obtained during a sub-maximal stage (130 W) of both SL and DL incremental VO_{2max} tests indicated higher plasma lactate concentrations during sub-maximal SL cycling (p = <0.01). Post training intervention only the SL training group reported significantly increased plasma lactate concentrations immediately post maximal SL cycling exercise (p = 0.05), but no similar significant increase in DL maximal plasma lactate concentration post the DL or SL training intervention was observed (refer to Table 3.7).

3.3.4. Time trial performance

Performance Variables	*SL Training group*			*DL Training group*		
	PRE	POST	**% Change**	PRE	POST	**% Change**
TT Performance						
Av PO (W)	301 ± 23	313 ± 45 *	**4.2 ± 3.9 %**	273 ± 14	284 ± 10 *	**4.0 ± 2.1 %**
Av HR (bpm)	169 ± 14	169 ± 13	**0.5 ± 2.5 %**	164 ± 8	165 ± 10	**0.5 ± 1.7 %**
Av CAD (rpm)	90 ± 3	93 ± 2	**2.8 ± 2.7 %**	93 ± 2	93 ± 1	**0.1 ± 1.6 %**
TTC (sec)	1839 ± 135	1764 ± 86 *	**- 4.1 ± 3.2 %**	1803 ± 76	1729 ± 65*	**- 4.1 ± 1.9 %**

Table 3.7. A comparison of physiological variables tested during the laboratory based cycling time trial (TT). Values are displayed as group averages and are compared pre and post training intervention. Where Av PO is average power output (watts); Av HR is average heart rate (beats.min); Av CAD is average cadence; and TTC is time to complete (sec). Percentage change (% Change) in values pre-post is displayed for each of the different training groups (Single leg trained v.s. DL trained). * Significant difference from pre training values.

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There was no significant difference in time to completion (TTC) between groups at the start of the training intervention. Cycle time trial (TT) time to completion (TTC) improved significantly from baseline in both the SL and DL training groups (refer to Table 3.6). The SL and DL training groups both showed an identical 4.1% improvement in TT performance.

Equally, power output (PO) (watts) during the TT also increased significantly in both groups post training intervention (Table 3.8), irrelevant of the training mode implemented (SL training group- $t(1)= -3.066$, $p = 0.018$; DL training group- $t(1)= -4.932$, $p = 0.002$ (refer to Table 3.8); with no significant difference in the improvements between the groups. Average cadence (rpm) and heart rate (beats.min^{-1}) during the TT were the same in both groups, before and after training. The SL group showed a trend for increased CAD after training but this did not reach statistical significance.

Chapter 4.0

Discussion

4. Discussion

4.1 SL training compliment provides no additional enhancement in DL time trial performance

This study was one of the first to compare the impact of a four week SL and DL cycle training intervention on endurance cycling performance outcomes. Our findings show that both training interventions resulted in improved performance i.e., a decrease in TT time to completion of ~4.1 % (~1min 15sec) and an increase in TT average power output (4.2 ± 3.9 SL vs. 4.0 ±2.1 % DL). However, due a limited sample size, inadequate TT familiarisation and lack of a control group (minus any laboratory training compliment) we are unable to conclusively report a novel improvement in TT performance as a result of the two training methods (SL/DL).

The observed performance improvements are comparable to previous improvements in 40 km TT performance(2.1 – 4.5%), reported post other published DL HIT training regimens with intervals of a similar duration (~5mins) (Lindsay 1996; Westgarth-Taylor 1997).For example Stepto and colleagues (1999) reported that a 2.5 % increase in 40-km TT performance was achieved after a three week training programme consisting of six high intensity work out bouts (4-8 x 3-6min at 85 % W_{max} - ~90s rest). Similarly, Weston et al. (1997) found that skeletal muscle buffering capacity, 40km TT performance and time to fatigue at 150 % peak power output (PPO) were also significantly improved ($p < 0.05$), after six sessions of high intensity cycling exercise (6-8 x 5mins at ~80% PPO). In addition, recent research corroborates these previous studies, HIT intervals of a moderate duration (~3-5min) considered useful in enhancing performance during longer cycling events (e.g. 40 K TT) (Laursen et al. 2002; 2005). However, contention still exists regarding the exact metabolic mechanism(s) responsible, and the optimal training processes for facilitating maximal

enhancements in endurance capacity and sustainable power output in endurance athletes (Flueck and Eilers 2010).

Prior investigations have identified both peripheral and central limitations to performance velocity, i.e. the speed or power that may be sustained for a prolonged duration (4min - 4h) (Coyle 1991; Joyner and Coyle 2008; Laursen et al. 2005; Lucia et al 2004). As discussed above and reviewed extensively- incorporating periods of intensified training (from 70- ≥90 % DL VO_{2max}) has been reported to act upon both central and peripheral components, potentiating adaptations which result in enhanced endurance performance (Seiler & Tønnessen, 2009). However, SL training was proposed to specifically induce a larger, local muscular adaptation in oxidative capacity via deliberate overload of peripheral factors such as muscle mitochondrial density, capillary density, oxidative enzyme activity, plus muscle fibre recruitment and level of activation. This increased peripheral loading potentiated via a reduction in central limitation and an increased in local oxidative stress when exercising a smaller muscle mass independently (SL) (Abbiss et al. 2010; Anderson et al. 1985; Davies and Sargeant 1974; Saltin 1976; Neary and Wenger 1986).

However, despite the growing impetus for investigation of high intensity interval training regimens and the concurrent effect on peripheral physiological systems and mechanisms (Burgomaster 2006; Iaia and Bangsbo 2010; Laursen et al, 2005; Laursen & Jenkins, 2002). Within this study we failed to demonstrate any additional benefit of a training model designed to elicit an additional peripheral training load. The concurrent increase in SL W_{max} and sustained PO during the TT after SL training was in accordance with our hypothesis. The increase in SL W_{max} without a concomitant increase in DL W_{max} or VO_{2max} suggested that SL cycling enhanced TT performance via

peripheral mechanisms related to endurance performance. However, no additional increase in TT performance was demonstrated post SL training. Therefore we may conclude that any potential additive peripheral adaptation generated by this novel training regimen may have been nullified. The lack of any additional TT performance enhancement potentially due to a reduction in central loading (SL group trained at ~50% DL VO_{2max}) and therefore decreased central cardiovascular adaptation experienced as a result of SL cycling.

4.2 SL training did not improve any performance indices measured during DL $VO_{2\ max}$ test

DL training increased DL VO_{2max}, DL W_{max} and av DL CAD. However no increase in these DL performance variables was evident post the SL training intervention. SL training did however increase SL W_{max} and average CAD during the SL $VO_{2\ max}$ test. Our findings suggest that SL and DL exercise seems to result in differential training effects. These effects point towards the specificity of training principle as described by Muller et al. (2000).

The explanation for the lack of increase in DL VO_{2max} post SL training could be multifaceted. Our results suggest improvements in DL VO_{2max} are not indicative of a peripheral adaptation. Plus, any metabolic adaptations elicited via exercising a reduced muscle mass (a single leg) at ~ 49-57 % DL VO_{2max} may not prove pertinent enough to illicit meaningful alterations in central circulation and whole body VO_{2max}. This finding is perhaps due to a lack of specificity with regard to differential motor recruitment, or alternatively a reduced cardiovascular strain within the SL exercise model. Any increase in SL W_{max} perhaps a result of increased rates of oxygen uptake during SL cycling within each leg. Thus, allowing greater muscle and pedal forces to be sustained aerobically in the SL mode, resulting in decreased local oxidative fatigue at a given PO, when exercising in the SL mode. These

inferences are supported by previous work, as even when exercising maximally in the DL mode, each leg does not reach the same metabolic (oxidative), localised stress as when each leg is exercised individually (Stamford et al. 1978; Davies and Sargeant 1974). Davies and Sargeant (1975) demonstrated a ~40 % greater rates of oxygen uptake (on a per leg basis) within the SL cycling mode, in comparison with DL cycling, when cycling at a sustained maximal intensity.

However despite a significant increase SL W_{max} post SL training, which could infer an increase in local muscular fatigue resistance, we found no concurrent increase in DL W_{max}. Explanation for this lack of transference could be the predominant limitation behind DL VO_{2max} in highly trained athletes- which is related to a central cardiovascular limitation, and/ or a potential internal central nervous system control. The crucial point that the onset of these central limitations are thought to occur prior to any quantifiable local (peripheral) muscle fatigue (McMurray and Tenan, 2010).

Our results were similar to those produced by a prior investigation into the effects of non simultaneous endurance training of both legs by Davies and Sargeant (1975). Their findings demonstrating a similar minimal, non-significant increase in DL $VO_{2\ max}$ of ~0.14 $L.min^{-1}$ (an increase of ~4.5%). In comparison, Klausen et al. (1982) demonstrated a larger and significant increase of ~16% in SL VO_{2max}. Plus, a larger but non-significant increase of 11 % in DL VO_{2max} (~0.37 $L.min^{-1}$) post an eight week SL training regimen (3x 1h/wk). These data were similar to our own results, predominantly supporting the non significant increase in DL VO_{2max} post SL training. The training regimen is this study utilized a study intervention twice as long as our own (8wks), potentially allowing further time for notable adaptation. Therefore perhaps the duration of the training intervention in the present study was too short to evoke an improvement in DL VO_{2max}. However, notable increases in endurance capacity (VO_{2peak}) and 40km TT performance have been observed

after similarly short (4 wk) training programmes (Laursen et al.2002; 2005; Lindsay et al. 1996; Swenson and Howley 1992). Swenson and Howley 1992) provided the first evidence for a significant increased DL $VO_{2\ max}$ post a SL training regimen. Within this study, SL endurance training of both legs caused a large, significant increase in maximum DL aerobic power ($VO_{2\ max}$) as did DL training (0.45 l.min $^{-1}$and 0.39 l.min $^{-1}$ respectively) ($p < 0.0008$).

However, a more compelling argument for the discrepancy in results between these similar investigations relates to the training intensity of our current study (35- 44% DL W_{max} / ~ 58-73% SL W_{max}). Similarly to the prior study by Davies and Sargeant (1975) (~ 80% SL W_{max}) the SL training intensity we prescribed was significantly lower than that prescribed by Klausen et al. (1982) and Swensen and Howley (1993). The latter two studies involved training intensities equivalent to ~ 75% DL HR_{max} ,~ 35-50 beats.min^{-1} higher than in our own current SL training regimen. Therefore in order to illicit a greater increase in DL $VO_{2\ max}$ post SL, the SL training regimen utilised should perhaps be of a higher intensity than our own (≥ 80% SL W_{max}). However, it is unlikely that such an increase in SL training intensity would be sustainable for as long as 5min in duration. This is mainly due to increased recruitment of more glycolytic muscle fibers which are less fatigue resistant (discussed further below- 4.3.).

Earlier research supports these assertions. Bell and Wenger (1988) demonstrated an increase in both SL and DL $VO_{2\ max}$ (~5% and ~11% respectively p=<0.05), post a 7 week SL interval training regimen (4d/wk). However, much shorter and more intense SL intervals were utilised (15-20 x 20s at 150% SL $VO_{2\ max}$), with a work-rest interval of 1:3.

Equally, it should be noted, that the participants recruited by Klausen et al. (1982) and Swensen & Howley (1992) were not trained cyclists, but described as 'young, healthy, untrained males'. This untrained population perhaps allowing for the exhibited larger increase in improvement pre-post due to a lower pre-training fitness status. In contrast our participants were well trained, experienced cyclists; an athletic population which usually report VO_{2max} as stable, even when performance is reportedly increased (Basset et al. 2000; Bentley et al.2001; Vollard et al.2009).

In summary, the use of a SL training strategy similar to the one outlined in this study may be considered ineffectual if the athlete's focus is purely an enhancement in DL VO_{2max} and associated performance indices. However, it is not unprecedented to report an increase in endurance performance without a concomitant increase in VO_{2max} (Hawley et al. 1997; Hawley and Stepto, 2001). Endurance capacity is reported to correlate strongly with other physiological variables (i.e. lower levels of lactate appearance at given submaximal exercise intensity) reflecting an individuals' ability to tolerate prolonged (~60 min) bouts of submaximal workloads (70-90% VO_{2max}). Equally, previous research surrounding predictors of cycling TT performance has highlighted certain peripheral factors (W_{max}, PO at ventilatory threshold, lactate accumulation at a sustained submaximal power) as better indicators of endurance performance capacity than traditional measures, such as VO_{2max} (Coyle et al 1991; Balmer et al. 2000; Vollard et al. 2009; Lucia et al.2004). These data supporting previous findings which have illustrated that an increase in training intensity can improve endurance performance without a change in VO_{2max} (Acevedo and Goldfarb 1989). These authors (Acevedo and Goldfarb 1989), finding no change in VO_{2max} (65.3 ± 2.3 vs. 65.8 ± 2.4

$ml.kg^{-1}.min^{-1}$), despite a large reduction(63s)in 10-km run time post eight weeks of intensified run training (90-95% HR_{max}).

4.3 Increased lactate during SL exercise and post SL intervention could indicate differential muscle recruitment

Plasma lactate concentration during incremental maximal SL exercise demonstrated a characteristic curvilinear increase with increasing VO_2 ($l.min^{-1}$). However, submaximal blood lactate values were found to be considerably higher during SL cycle exercise, blood lactate averaging ~ 1.2 $mmol.l^{-1}$ higher (p 0.05) during SL cycling at 130 watts, in comparison to DL cycling at 130 watts These results consistent with previous data (Stamford et al. 1978; Davies and Sargeant 1975 and Neary and Wenger 1986). However, despite the congruent nature of the current data, the point must be made that as detailed above (2.3.1) the $VO_{2\ max}$ protocols did differ between the SL and DL mode in both duration and organisation of steps, thus a direct comparison is impossible.

Current research has clearly demonstrated an increased local mechanical and metabolic stress elicited via SL cycling compared with DL cycling (Grisham et al. 2010; Rakowski et al, 2010). Grisham et al. (2010) recently provided data demonstrating that high intensity SL exercise can produce a higher perturbation in the metabolic milieu, indicating rises in blood lactate concentrations equivalent to those produced via maximal DL cycling. The potential reasons for such an increase in blood lactate appearance whilst exercising in the SL mode are discussed below.

In a single contracting muscle fiber the frequency and duration of contractions determine energy demand (Adenosine Tri Phosphate- ATP production). Usually an athlete will recruit different

isoforms of muscle fiber according to the power or speed required for the activity (Talmadge et al. 1993). In endurance trained athletes the primary muscle fibers utilized are slow twitch (type I), which are highly oxidative and resistant to fatigue (Burke et al. 1973). However, these fibers are not considered able to generate high levels of force production for prolonged duration. Previous research has suggested that during intense whole body exercise 100% of the available muscle fibers may not be simultaneously recruited; in order to counter rapid fatigue (Sloninger et al. 1997). For example, St Clair Gibson et al. 2001 demonstrated that at any one point during a 100km cycling TT (as quantified by intra-muscular electromyography (IEMG)) activation of the vastus lateralis (VL) was considered reduced by ≤ 20% of a pre-exercise maximal voluntary contraction (MVC). However, in concurrence they also found large glycogen depletion values within the VL, thus indicating an alternating recruitment pattern, as opposed to a general low level of activation.

The differential activation pattern described above is proposed to maximise muscular recruitment, whilst avoiding premature fatigue during sub-maximal activity (Enoka and Stuart 1992; Westgaard and De Luca 1999).

During intense SL exercise, the reduction in muscle mass increases the need for an alternate fiber recruitment strategy. However due to the reduction in total number of motor units available, the duration and amount that the smaller muscle group may be able to effectively stave off fatigue via alternate patterns of recruitment may be limited in comparison to a larger active muscle mass. Therefore exercising with a smaller total muscle mass is proposed to lead to a premature fatigue of the predominant oxidative type I fibers recruited, plus additional and greater recruitment of more powerful, glycolytic type II fibers. Plus, rapid oxidation of the lactate by surrounding fibers and tissues has previously suggested to be reduced in the SL model (Neary and Wenger 1986). As a result a greater accumulation of hydrogens ions (H^+) and lactate occurs in the muscle due to a much

greater involvement of the glycolytic energy system providing ATP for the muscle contraction (Robergs et al. 2004).

Interestingly, post the training intervention period, only the SL trained group demonstrated an increased maximal plasma lactate immediately after the final stage in the SL incremental VO_{2max} test. The ability to continue exercising at a maximum intensity for an extended period, with high plasma lactate concentrations could be due to an increased capacity to utilise lactate for continued energy production and minimise the negative effect of H^+ accumulation within the muscle (Neilsen, 2001; Peronnet, 2010). This increased plasma accumulation perhaps the result of a training induced increased occurrence, oxidation and removal of lactate from the intra-muscular environment into the blood and surrounding tissues. The transport and uptake lactate and corresponding protons an important factor regulating the maintenance of sustained high levels of muscular contraction (Gladden 2004). Plus, one that is reportedly increased in a dose response manner in conjunction with high intensity (110-120% DL W_{max}) HIT training (Dalleck 2010).

In summary, these data suggest that SL training may induce greater tolerance to lactate accumulation during SL exercise; again highlighting a degree of training specificity. This proposed augmentation in total buffering capacity with SL training would also fit with previous research which has shown an increase in capacity to buffer H^+ through training (Bell & Wenger, 1988; Juel et al. 1999; 2004). Plus, improvement in buffering capacity correlates well with improvement in endurance capacity physical activity (Weston et al, 1997; Juel et al, 2004). Bell and Wenger (1988) demonstrated (using a similar SL training model to Saltin et al. 1976) that in comparison to an untrained leg, SL sprint cycle training (7 wk) elicited a markedly higher plasma lactate accumulation

in the trained leg, post a 60 s anaerobic power test. However, further similar studies are required to progress the findings of this current study and support the inferences made here. Specifically, examination of the relationship of muscle recruitment patterns (EMG) and force production in cycle specific muscle groups (e.g. VL) during both SL with a counterweight device (5, 7.5, 10, 12.5kg) and DL cycling exercise is needed. This type of investigation would be useful in order to clarify which counterweight produces a muscle activation and power output (amplitude and frequency) most similar to that produced throughout the normal bilateral (DL) pedal stroke. Also, muscular fatigue may be calculated during a longer simulated training effort (~5min). Equally, invasive arterio-venous measurement or the use of stable isotope infusion techniques may have to be implemented in order to clarify exact limb blood flow during SL cycling with a counter weight device. However most interesting would be the inclusion of muscle biopsy both prior and post a SL vs. DL training intervention, including an invasive measure such as this further in depth data related to muscle oxidative enzyme regulation and muscle protein content could be recorded. This level of investigation is needed to begin to clarify the metabolic and neuromuscular differences and physiological adaptations that stem from these two differing training modalities.

4.4 Training specificity is key when aiming to improve performance

Throughout our current results clearly highlight a specificity of training response to each of the respective training interventions used within this comparative study. A significant increase in W_{max} , VO_{2max} and average CAD within the DL $VO_{2\,max}$ test was only evident within the DL trained participants. Concurrently, SL trained participants were the only ones to exhibit a large significant

increase in SL W_{max} and av CAD maintained during the SL $VO_{2\ max}$ test. The principle of specificity dictates that the body will adapt to the demands imposed upon it. Adaptive responses to exercise are considered a function of the specific movement patterns executed during training. Therefore in order to maximise the training response, the training modality should allow optimal innervations of the specifically trained muscles (Withers et al. 1981). Plus the training prescribed should be closely matched to the required training outcome (i.e. the performance scenario), in order to cultivate the desired enhancement of performance.

Within our study the 5-min DL cycling intervals were carried out at a pace similar to that of the time trial (70-88% DL W_{max}). These DL intervals were expected to improve performance due to the growing validity for HIT training at similar intensities (Laursen et al. 2002; Lindsay et al. 1996; Stepto et al.1999; Westgarth-Taylor et al. 1997). Whereas the SL training intervals were set to 50% intensity of the DL intervals and only required the recruitment of one single leg at a time. With our current results in mind, we conclude that our SL training intervention, plus the supporting counter weight device, offered a good comparison to the aforementioned DL training regimen. However, the efficacy for the further implementation of the SL training regimen as a compliment to normal DL personal endurance training in the current format used here is minimal; the lack of specificity for an enhanced DL performance evident.

4.5 Inter-individual variability in training response is high

Initially, when participant recruitment was contemplated, a priori test to compute the required sample size suggested that ≥ 7 participants within each group would be sufficient to detect statistically significant differences in TT performance (TTC/ av PO). A required sample size of 16 (8

in each training group) was estimated to detect significant differences in performance data. Unfortunately within our sample group four individuals had to be excluded due to illness, only eleven of the fifteen endurance trained cyclists recruited successfully completed the study; thus minimizing the overall sample size and weakening the power of the subsequent research findings. Also the inter-individuality in training response recorded within these smaller groups was large.

Within this study the resultant small participant sample represents a significant limitation in the analysis of performance. Subsequent analysis of intra-individual differences in TT performance showed that in both groups training effect differed significantly between individuals. Within the SL training group particularly there was a significant difference between measurements in response to the SL training intervention. Future studies should aim to recruit larger participant numbers in order to account for high dropout rates and high levels of inter-individuality amongst the chosen sample, while providing a sufficient number of athletes to detect statistical significance in small potentially meaningful differences.

Equally a major factor within this current study is that our participants in general may well have been recruited at differing stages of personal training status- some participants using the study as a means to re-establish a regular training regimen; thus generally contributing to variability in response. Similarly, Lindsay et al. (1996) previously acknowledged the potential positive impact of any form of training intervention, highlighting athletes specifically as being particularly suggestive to novel training techniques. Thus, with a particularly ‘novel’ training paradigm such as SL cycle training the physiological vs. psychological impact may not be easily differentiated.

A further methodological issue which significantly affected the training intensity between individuals arose due to the chosen method used to standardize training intensity between groups. The problem which faced investigators at the time of protocol definition was how best to ensure that training intensity between groups was as closely matched as possible. Previous investigators have also struggled with this issue, Swenson and Howley (1992) failed to match the training intensity between groups, their protocol allowing the SL group to train at a far greater training volume and intensity per limb.

Within our investigation the training intensity was standardized via a measure of total work done per limb, in an attempt to allow an element of control, whilst maximizing the potential training effect to be seen in both groups. However, during the preliminary stage of the training intervention (Wk 1) the participants rated their perceived exertion (RPE) a lot higher in higher within the SL training group (SL= 14 ± 1; DL =12 ± 1). The difference in perceived exertion between groups could be indicative of the lack of previous familiarisation to the SL training modality. Alternatively, the workload prescribed to the DL training group in Wk 1 (77% W_{max}) may have been too much of a compromise with regard to intensity (i.e. not challenging enough to illicit a higher perception of effort, or even a substantial perfusion limitation).

Equally, when training intensity was expressed as percentage VO_{2max} it was proven to be varied between both individuals and training conditions. This discrepancy alone could account for an increased variability in training response. However, if we had chosen to match training intensity using a specific per limb percentage of VO_{2max}, this could have negated any potential benefit of the SL training model, as any potential to elicit a higher training effort (% VO_2) whilst exercising a smaller muscle mass would have been be lost.

4.6 Practical applications for SL cycle training research

The main premise of this current research was performance orientated, however research involving the application of a SL cycle training model has also been utilized in order to investigate possible clinical perspectives. Within a clinical physiological setting the SL cycling training model has been used to substantial positive effect within cardio-respiratory rehabilitation models (Bjørgen et al. 2009; Dolmage and Goldstein 2006; 2008). In the case of an 8 week training study with COPD patients (Bjørgen et al. 2009), SL cycle training for just 30min per weekday, reportedly improved DL VO_{2max} by ~12-14%. Furthermore, isolated small muscle mass exercise and SL cycling studies in COPD patients have previously demonstrated greater muscle mass specific power output and stimulus than comparative whole body exercise, concluding potential increases in training response (Richardson et al. 1999; 2004; and Dolmage and Goldstein 2006; 2008). Work utilizing the COPD model highlights SL cycling as a potential training model to allow maximal leg work, whilst minimizing the cost of ventilatory contribution to exercise induced fatigue, however further investigation is necessary in a larger cohort (Mador 2008). Equally, within a slightly different clinical model, unilateral exercise focused upon the hemiparetic limb has also recently been highlighted as a potential effective intervention within stroke rehabilitation (Billinger 2010).

Moving away from clinical examples of current application of the SL training model, additional development and optimization of the training protocol is needed if further invasive exploration is to be undertaken, in order to fully comprehend the validity of SL training for enhancement of DL cycling performance in highly trained athletes. Subsequent research should focus around the recent work by Grisham et al. (2010), within which SL cycling at supra-maximal DL intensities has been

identified as a possible tool for habituating afferent nocireceptors and potentially reducing perceived exertion associated with subsequent exercise.

4.7 Further unanswered questions surrounding the SL training model

The main aim of this current pilot study was to ascertain the potential usefulness of SL cycle training as a training strategy to improve DL endurance cycling performance. We did not include any invasive measurements, and therefore were unable to investigate potential mechanisms for the observed performance effects adaptation or changes at the molecular level. Unfortunately this means that changes observed in performance variables could not be attributed directly to changes resulting in adaptation of relevant physiological mechanisms. As a result conclusions generated via this study are still subject to the limitations of speculation; therefore future research should include an invasive element allowing further intracellular investigation such as muscle biopsy and subsequent immunohistochemical analysis of intramuscular factors of interest such as capillary density and potential markers for mitochondrial biogenesis. Plus, potentially altered plasticity in muscle fibre type as a result of endurance training in the SL cycle model is yet to have been investigated specifically. Similarly, particular emphasis should be put on ascertaining the exact limb blood flow kinetics during DL vs. SL cycling, along with a more specific measurement of arterio-venous lactate accumulation across the exercising limb specifically, in order to fully elucidate the training adaptations observed post SL cycling.

Further investigation along this vein of research should be employed, however emphasis must be put on controlling all other training and environmental variables if any potentially additive marginal gain in performance is to be highlighted. Future research should perhaps focus upon intervals of a higher intensity and shorter duration, the more recent research of this nature identifying a large metabolic disturbance when supra-maximal SL cycle efforts were attempted (Grisham et al. 2010). In relation, a measure that we failed to include within our current study is a measure related to the impact of a high intensity SL training regimen upon the anaerobic performance component; a potential benefit theorised as a result of current speculation for a reduced force sensation and fatigue resistance post SL training. The impact of SL cycle training upon initial peak power production could be tested via an inertial load test (Martin et al. 1977) or a 30 s Wingate protocol and would provide the missing data relating to any potential improvement in anaerobic capacity.

Equally, in relation to the continued application of the SL training modality, the exact weight of the counterweight to be utilized in such investigations has yet to have been conclusively optimized for different individuals. Further investigation involving EMG and simultaneous kinematic analysis of independent pedal strokes should be undertaken. Typical SL cycling pedal stroke should be analysed both with and without a counterweight, plus with different weightings of counterweight- in different sub groups related to age, sex and weight (also advised by Thomas et al. 2009) (Please see Appendix 4 for potential future study design).

4.8 Concluding remarks

In conclusion, despite significant changes pre-post in TT performance variables between individuals in both training groups, unfortunately due to limited familiarisation, overall sample size and lack of a control group who did not receive any laboratory led training compliment this study was unable to demonstrate definitely that either a DL or SL interval training compliment resulted in a significantly greater performance enhancement over time. Thus, it is still not clear what additive impact, if any, inclusion of the SL training stimulus within a normative cycling training regime has on subsequent DL cycling endurance performance. It is likely that when considering investigations regarding this novel SL training method, the measure of $VO_{2\ max}$ is not sufficiently sensitive to the adaptations predicted to occur. Thus, the emphasis usually placed upon improvements in $VO_{2\ max}$ as a direct inference of improved endurance performance may have been previously overemphasized and lack validity in this scenario.

Therefore, despite inferences for an additive benefit of SL training with a counterweighted opposing pedal we can only conclude that the SL training induced improvement in time trial cycling performance may be considered beneficial to endurance cyclists, but currently has not been proven to have any additive benefit to normal DL high intensity interval training (HIIT) or a normal self imposed training regimen. SL cycle training did induce enhancement in SL W_{max}, especially in the usually non-dominant left leg. Therefore SL cycling should be investigated further as a means to reducing any bilateral deficit in power production noted between limbs.

Without the use of further invasive procedures, the exact molecular mechanism supporting the observed training effects in either group can only be based on conjecture. Speculation surrounds the possibility that SL cycling exercise elicits an increased muscle mass specific exercise intensity, compared with conventional DL interval training. The premise being that by working each

leg separately at an increased muscle specific intensity a greater peripheral adaptation may be elicited; specifically targeting enhanced fatigue resistance and potential oxygen utilization at the muscular level. However the concurrent lack of additive performance benefit portrayed within this study and lack of any measure of muscular oxidative potential does place this assertion into question; plus the hypothesis of increased fatigue resistance remains to be proven conclusively. Further investigation is warranted, the main focus of which should be to optimize the weight of the counterweighted pedal and specifically target variability within the sample population. A 'training camp' scenario would be ideal for ensure increased control of confounding factors within subsequent similar trials.

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Appendices XI

Appendix I: Participant Information sheet

UNIVERSITY OF BIRMINGHAM

Participant Information Sheet

Study title: 'One legged cycling as a model for improving two legged cycling performance.'

Location: Human Performance Laboratory, University of Birmingham

Investigators: Mr. Adrian Holiday - 3rd yr. UG -Student
Miss Lucy Walton - 3rd yr. UG -Student
Miss Rachel Turner – MPhil Student

Supervisors: Professor Asker Jeukendrup, University of Birmingham
Dr Kevin Tipton, University of Birmingham
Dr James Martin, University of Utah, Salt Lake City, USA.

Purpose of the study:

In cycling, training one leg at a time may give different and perhaps superior training adaptations than training with two legs. Early studies were unable to demonstrate superior effects of single leg (SL) training on maximal double legged (DL) aerobic power (Davies & Sargeant, 1975; Klausen et al. 1982). However several more recent studies reported a significant increase in maximal aerobic power following single leg endurance exercise compared with double leg training (Bundle et al. 2006; Gevais & Ellis 2001; Neary & Wenger 1986; Saltin et al. 1976; Stamford et al. 1978; Swensen & Howley 1993). In previous studies a single leg was trained by pedalling on a bike or cycle ergometer with one leg. This involves a more powerful upstroke and a less powerful down-stroke and thus muscle recruitment of single leg cycling is different from two leg cycling. By using a counterweight on the opposing pedal, cycling motion of dual leg cycling may be mimicked, muscle fibre recruitment may be more similar or even identical and performance effects of single leg cycling may be more profound. To date no one has examined the effects of single leg training with a counterweight on dual leg time trial performance.

The main study aim:

1. Compare a training programme of single-legged cycling with a double-legged (normal) interval cycling programme of a similar intensity and duration.

Study Design:

If you agree to take part in this study, you will be required to complete ~3 hours of performance testing before and after a 4 week training period. During the training weeks you will be asked to replace three sessions of normal training with lab based training (1h sessions). This training will consist of either one- or two-legged cycling. All tests will be performed in the Human Performance laboratory of the School of Sport and Exercise Sciences, University of Birmingham and will be supervised by the investigators.

The quality of the data collected and the success of this research project will depend on you adhering to all the training, testing and dietary instructions. Therefore, we will provide you with an instruction booklet that will clearly states everything that you will need to do to help us carry out this important research project.

Procedures – What performance tests/training sessions will you be required to perform when?

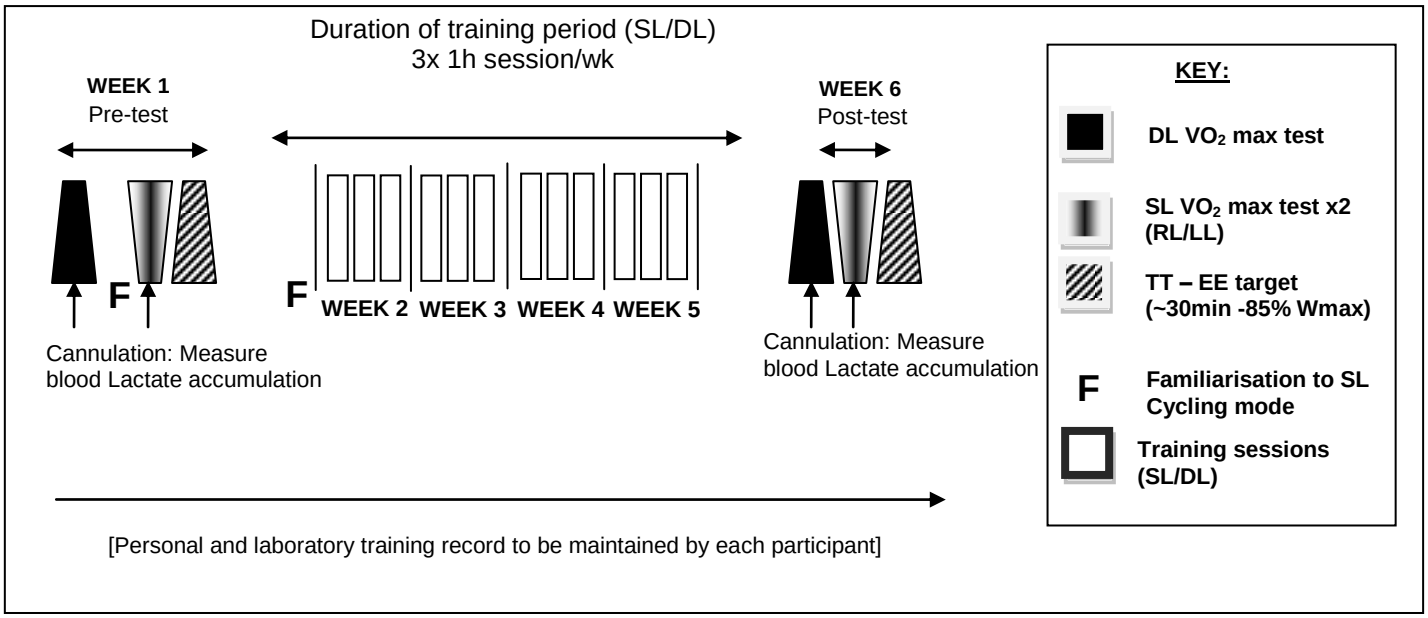


Figure 1: Study design

Study Logistics:

Each participant will complete four performance tests before and after a 4 week training period (figure 1). You will be required to record your diet prior to each preliminary performance testing session (which will be completed before you have eaten, preferably in the morning before breakfast). It is important that we keep a detailed record of what you eat prior to your pre-performance tests as we would like you to replicate what you ate prior to each of the concurrent post tests. Also you will be required to keep a detailed record of your own personal training throughout the study period for our subsequent analysis. This personal training should not replicate the study training session, nor should they be overly intense- leading to a detriment in performance/completion of your lab sessions.

Preliminary testing, including familiarization should be at least a day, but no more than 5 days prior to the first training session. Post-testing should not be any more than 3days post the last training session performed. If you agree to participate in this study you will be required to visit the laboratory on 20 separate occasions. These visits will last

approximately an hour or less and will involve in the majority (12/20 visits) monitored training sessions. The study will span over 6 weeks in total.

On the first day of the study you will complete the two-legged VO_2 max test, followed by a 10 min single legged (SL) cycling familiarisation in preparation for the SL VO_2 max tests (either straight after/ the next day. On day 3 you will complete both single-legged tests. Between the single-legged tests there will be a break of 15 minutes to allow for recovery. Lastly no longer than 2 days later you will each complete a simulated ~ 30min time trial (TT), within the laboratory under controlled conditions. Post the final performance measure you will be allocated to your training group (single leg/double leg) and will be required to complete a familiarization session, identical to your study training protocol, but only 45mins in duration. Also prior to you starting your laboratory training sessions, you will be required to complete one more preliminary session, which involves: a measurement of maximum SL power output during a single pedal revolution, plus taking measures of leg volume (these measures will be taken either the day before the TT or the same day as your final familarisation session depending on your levels of recovery between each test).

After initial baseline testing and familiarization you will start the four weeks of lab based training (3x 1hr sessions/week); completing the lab training on days 1, 3 and 5 of each week. You will still be able to complete your regular training; however on three days a week a lab session will replace your personal training. The lab sessions will consist of either one or two legged cycling. Post training testing will occur no longer than three days following your final bout of lab training.

Preliminary Procedures

Preliminary body composition/ individual leg strength measures include:

- Measures of height and weight.
- Measure of individual leg volume/composition.
- Measure of individual leg peak power output during pedalling action.

Performance measures (Pre/Post)

VO_2 max tests:

These tests involve incremental cycling until exhaustion. A 2ml blood sample blood will be collected from a plastic cannula inserted into the antecubital vein both prior to the test and at the end of each complete stage. Gas analysis using an Oxycon Pro (online breath analysis system), with a fitted oxycon mouth piece system, will occur throughout all test and training protocols in order to ensure accurate gas analysis.

- *Two-legged* – Participants start at 95W and this is increased by 35W every three minutes. The present protocol is a compromise between different protocols and is believed to give both accurate submaximal as well as maximal values for VO_2 and peak power output.
- *Single-legged* – In this case participants start at 66W for a total of 2mins and thereafter this is increased by 16W every 1minute.

Time trial (TT):

During the study you will perform two TTs, pre and post 4 weeks of training. Each TT will be preceded by a 10 min warm-up period (5mins at 35%Wmax, immediately followed by 5mins at 50% Wmax). After the warm-up you will perform a simulated TT in which a target amount of work will have to be completed in as short a time as possible. The energy target will be set so that if you cycle at 85% Wmax it will take 30 minutes to complete. If you cycle at a lower intensity it will take longer, however if you cycle at a higher intensity it will be completed in a shorter amount of time.

You will be required to not to eat prior to each *performance test*; which will preferably be performed in the morning before breakfast. Heart rate, cadence and perceived exercise intensity will be monitored and recorded continuously throughout the tests.

Training protocol

Throughout the study you will be allowed to complete your regular training, however on three days of each training week the subjects' normal training will be replaced with lab based training of either one- or two-legged cycling (3h/wk). As a result, you will therefore be required to keep a detailed record of your personal training, within a format we will provide you when you commence the study.

As this a training study aimed at assessing differences over time it is requested that you adhere to the requirements of your training group. It is important that no new exercise or activities is commenced during the duration of the study as this may influence the results. We also ask that you do not replicate the lab training sessions in your personal training and that you don't over exert yourself prior to a lab training session; as it is important performance levels are maintained and that you are able to complete each lab training session. The lab training will be completed a minimum of two hours after a meal and you will be required to record what you eat the meal before and for three hrs after the exercise bout (dietary record sheets will be provided). All laboratory based training will be carried out in the Human Performance Lab, within The School of Sport and Exercise Sciences.

Training sessions:

SL participants will cycle SL at 35% of their predetermined DL Wmax for one hour, alternating the exercising limb every 5 min (on a Lode bike especially modified for SL cycling- the opposing pedal loaded with a 10kg counter weight). Whereas DL participants will exercise using both legs (utilising a normal cycling technique), alternating between 5 min cycling at 70% of their predetermined Wmax and 5 min rest, for a total of one hour.

Heart rate measurements; rate of individual perceived exertion; rate of perceived leg effort; cycling economy; energy expenditure; plus gas analysis -using an Oxycon Pro (an online breath analysis computer system) will be taken throughout both the training protocol and all performance tests. Each week the workload in both groups will be increased by 3% of each individual's Wmax in order avoid a performance plateau (a training effect), as you become more accustomed to the protocol and are able to tolerate higher workloads.

Comparisons of performance procedures pre and post the training intervention will be utilized in order to ascertain whether any performance improvement has been observed.

Single Legged:

Week 1	5 min right, 5 min left.	35% DLWmax
Week 2	5 min right, 5 min left.	Increase by 3%Wmax
Week 3	5 min right, 5 min left.	Increase by 3%Wmax
Week 4	5 min right, 5 min left.	Increase by 3%Wmax

Double legged:

Week 1	5 min cycling, 5 min rest.	70% DLWmax
Week 2	5 min cycling, 5 min rest.	Increase by 3%Wmax
Week 3	5 min cycling, 5 min rest.	Increase by 3%Wmax
Week 4	5 min cycling, 5 min rest.	Increase by 3%Wmax

Dietary and Activity controls:

You will be asked to record what you eat for the day before each preliminary performance test and be asked to repeat this before the post testing sessions. During the training period there will be no diet intervention/manipulation, however you must not have eaten for a minimum of 2hrs prior to training.

Risks:

As a result of maximal exercise tests and the training sessions you may well experience fatigue. This will be fairly short lived with a full recovery within 24 hours.

Blood samples will be taken during four visits (in total) via an indwelling venous catheter inserted into an antecubital vein. Insertion of the catheter may involve a very small amount of pain due to the initial needle insertion. Occasionally this procedure may result in a small bruise on the arm at the site of the needle insertion. This can usually be prevented by applying pressure on the arm when the cannula has been removed. This procedure is necessary in order to obtain lactate concentrations. Trained personnel will draw the blood in order to minimize these risks. Risks will be minimized by safe practice and conduct by trained members of the research team. If at any point during the protocol you feel uncomfortable or unable to continue, testing will be ceased immediately.

Confidentiality:

All data obtained will be dealt with in a confidential manner. Your name will not be used in any publications resulting from this study, making it impossible to identify you in the report. No additional measurements to those described above will be made and all samples will be discarded after use.

Rights:

It is your choice whether or not you wish to take part in this study. If you wish to take part in this study, you will be given this information sheet to read and be asked to sign a consent form. You are reminded that if you decide to take part in the study, you are still free to withdraw from the study at anytime without provision of reason. Requests for a copy of the results attained will be honoured following study completion and publication in a peer-reviewed scientific journal.

Incentives:

You will be given £50 to cover travel and time costs.

Benefits:

The results of the VO_2 max test will give you an indication of your performance capacity and training status. You will also get information about sport nutrition in general and there is an opportunity to ask the experiment leaders anything about sport nutrition and training.

Contacts:

[redacted]

Appendix 2: Participant Consent Form

UNIVERSITY OF BIRMINGHAM

Consent form

Investigation: One legged cycling as a model for improving two legged cycling performance

Investigators: Professor Asker Jeukendrup,
Dr Kevin Tipton,
Dr James Martin,
Miss Rachel Turner

Subject Number:

Name:

Address:

DOB:

I have read the attached information sheet and discussed the investigation with ……. ……who has explained the procedures to my satisfaction. I am willing to undergo the investigation but understand that I am free to withdraw at any time without having to give an explanation and that doing so will not affect my treatment or care I receive

Signed……..

Witnessed……...

Date…….

Appendix 4: Participant food diary instructions

Keeping a food diary

- The more information you provide the more accurate the feedback can be.

- Do not alter your diet because you know someone is going to be looking at it. This needs to be a reflection of your normal diet.

- Keep your form with you all day, and write down everything you eat or drink as and when you eat and drink it. Do not rely on your memory at the end of the day. Record your eating as you go.

How to record your food and drink

1. Start a new sheet for each new day, putting the date and the day at the top of the page.

2. In the first column record the time that you are eating and/or drinking.

3. In the second column describe the food/drink you are consuming trying to give as much detail as possible, giving the brand name, the food and how it was cooked, for example
   - Hovis, wholemeal bread, toasted
   - Asda, chicken breast, grilled
   - Chip shop chips OR McCain oven chips
   - Walkers salt and vinegar crisps
   - Robinsons no added sugar orange squash
   - McDonald's Big Mac
   - Or if it is home-made i.e. a meal that you share with your partner or family- please detail the recipe (and amounts- eg. 400g lean beef, 4x tinned tomatoes, etc) and estimate what %age of the meal you had.

4. In the third column record how much of each food item you ate. Where possible try and weigh the food, making sure you indicate whether it was cooked or not when you weighed it. For solid foods, the food should be placed on the scale on a plate or container. The plate or container must be weighed empty first and the scales can then be zeroed. Each item of food can then be added to the plate and weighted individually, returning the scales to zero between each item.

5. If there is any food left over, try and estimate how much you did not eat and record it in column 4 (*not strictly necessary if you have detailed what you think you have eaten in detail).*

- Remember to record any extras, such as mayonnaise, gravy, sugar etc, any snacks/drinks you ate/drank throughout the day. If you ate in a restaurant, please indicate this as well as recording what you ate and drank.

Appendix 5: Participant Food Diary- Exemplar

DAY OF THE WEEK ..Sunday..		DATE:...25/01/09.....			
TIME	DESCRIPTION OF FOOD OR DRINK CONSUMED	WEIGHT	WEIGHT LEFT OVER	PLEASE LEAVE THESE COLUMNS BLANK	
7.00 am Breakfast:	2x a slice of Warburtons white, thick sliced bread				
	2x a thin spread of flora original				
	2x a thick spread of tesco's own raspberry jam				
	1x a cup of milky tea – with Marybelle skimmed milk + 1x tsp of soft brown sugar				
8.30- 12 During training:	1litre weak diluted squash – orange tesco's own				
	1 go ahead cereal bar	15g	0g		
12.30 post training snack:	2x a slice of Warburtons white, thick sliced bread				
	2x a thin spread of flora original				
	2x a thick spread of tesco's own raspberry jam				
	1x a cup of milky tea – same as above				
1pm Lunch:	Really big bowl of porridge! Made with:				
	Co-op's own medium porridge oats				
	Marrybelle Skimmed milk	80g			
	1x large tablespoon of raspberry jam (same as above)	~ 250ml			
3pm Snack:	1x braeburn apple (large)		0g		
	7x dried apricots- Whitworths	~ 65g	0g		
	2x ginger biscuits co-op's own	~ 60g	0g		
6pm During training:	1 litre of plain water				
8pm Dinner	Spag bol: lean beef mince (co-op)	500g	250g		
	2x can of chopped tomatoes (co-op)	800g	400g		
	1x large courgette	200g	100g		
	1x tin of green giant sweetcorn	60g	30g		
	Spaghetti (co-op)	450g	225g		
	1xtps oregano, 2xgarlic cloves + a large squirt of tomato ketchup! 1.5 litres of dilute squash				
Pudding!	Rice pudding (1 tin)	400g	0g!		

Appendix 6: Participant Exercise Diary Template

DAY AND TIME	DESCRIPTION of training (please include activity and type e.g. continuous/interval)	If possible include			DURATION of training
		Max heart rate	Average Heart rate	Distance covered	

Appendix 7: SL Participant Training Record- Exemplar

SL- Training session no. 10

Study:	SL CYCLING
Name:	X
DOB:	04/05/1989
Age:	19
Weight:	81.53
Height:	184.5
Date:	20/03/2009
Time:	4:30pm
Barometric pressure:	33%
Temperature:	21.2 °C
Wmax:	387

Time (mm:ss)	Training leg	Actual Pow W	BIKE A Adjusted Power W	BIKE B Adjusted Power W	Oxycon measurem (mm:ss)	Heart Rate (bpm)	Cadence (rpm)	RPE
0						86		
5	R	158	155		0-5	134	92	13
10	L	158		153	5-10	140	89	14
15	R	158	155		10-15	146	91	13
20	L	158		153	15-20	143	85	15
25	R	158	155		20-25	141	90	13
30	L	158		153	25-30	139	89	15
35	R	158	155		30-35	138	89	15
40	L	158		153	35-40	138	80	15
45	R	158	155		40-45	140	84	15
50	L	158		153	45-50	140	83	17
55	R	158	155		50-55	139	77	17
60	L	158		153	55-60	144	85	17

Training sessions:

Week 1	35%	125	121	122
Week 2	38%	136	132	132
Week 3	41%	147	143	143
Week 4	44%	158	155	153

Appendix 8: Participant Final Report- Exemplar

Sport and Exercise Sciences
Performance Evaluation

University of Birmingham
Sport Science Dept.
Edgbaston
B15 2TT Birmingham

THE UNIVERSITY OF BIRMINGHAM

Name	x	Age (yrs)	19
Date	22/03/2009	Weight (kg)	81.53
Test	Cycle Ergometer	Heigth (cm)	184.5

Sport scientist R Turner

MAXIMAL PERFORMANCE

	16/02/2009	22/03/2009
Wmax	358	375
VO2max	4943	5239.2
PVO2max	5079	5388
Wmax/kg	4.39	4.60
VO_2max/kg	59.1	64.3
PVO2max/kg	60.8	66.1

Pre Test 16/02/2009 ♦

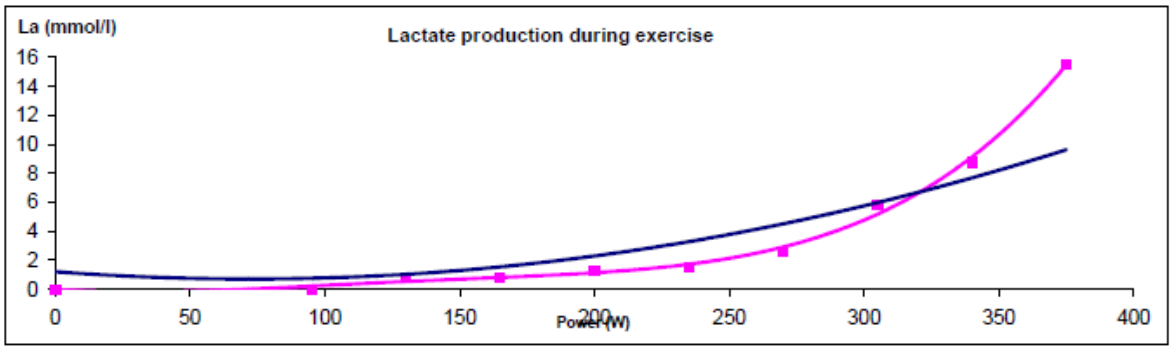
Stage	Power Output (Watts)	Lactate (Analox) (mmol/l)	Heart Rate (b/min)	VO2 (ml/kg/min)	RPE (Perceived Exertion)	Minute Ventilation (l/min)	Cadence (rpm) (s/min)	Watts/ kg
0	0	1.14						
1	95	1.05	113	21.1	7	49.25	80	1.37
2	130	1.47	118	25.4	7	42	79	1.88
3	165	1.39	131	31.4	9	53.25	85	2.38
4	200	1.69	144	36.4	11	65.25	85	2.89
5	235	2.25	159	42.1	11	81.75	85	3.39
6	270	4.02	166	46.7	14	93.8	83	3.90
7	305	8.01	177	51.8	15	125	90	4.40
8	340	8.44	183	56.5	19	151	88	4.91
9	375	8.45	189	59.1	20	174	72	5.42

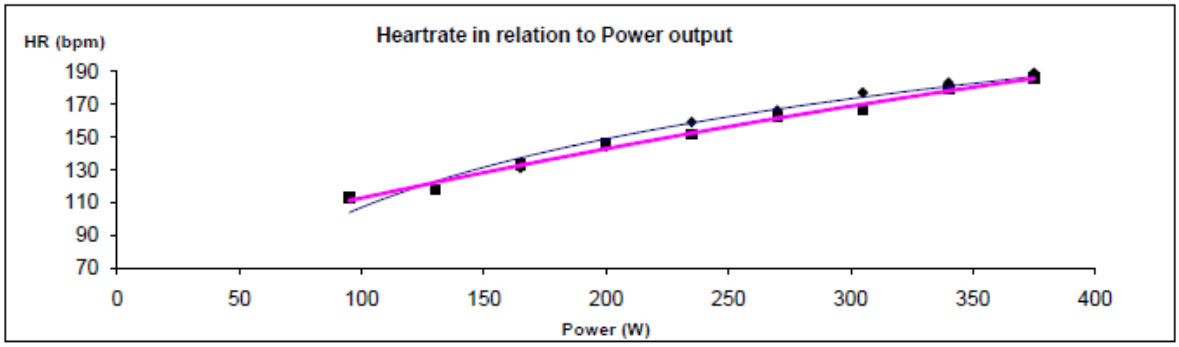
Post Test 22/03/2009 ■

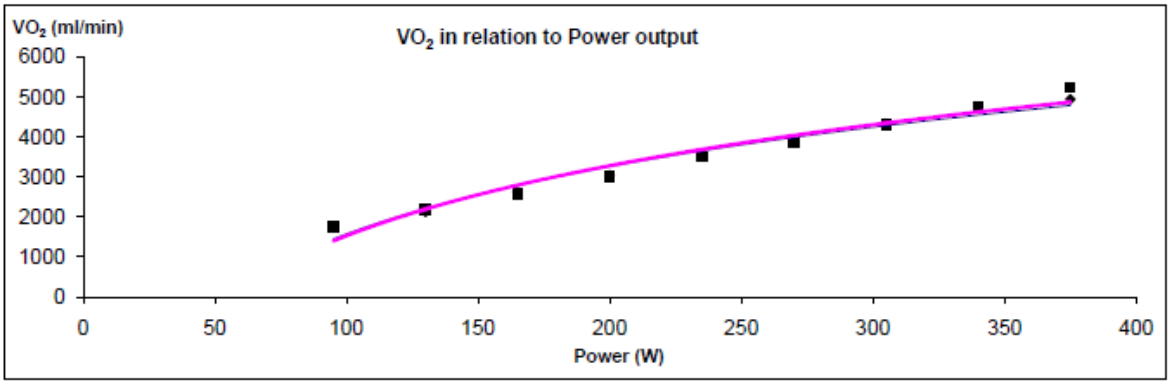
Stage	Power Output (Watts)	Lactate (mmol/l)	Heart Rate (b/min)	VO2 (ml/kg/min)	RPE (Perceived Exertion)	Minute Ventilation (l/min)	Cadence (rpm)	Watts/ kg
0	0	<0.79						
1	95	<0.79	113	21.5	9	39.0	81	1.17
2	130	0.82	118	26.9	9	45.3	83	1.59
3	165	0.79	133	31.6	9	57.0	84	2.02
4	200	1.24	146	37.0	9	64.8	89	2.45
5	235	1.5	152	43.1	11	75.5	87	2.88
6	270	2.6	162	47.4	11	89.5	87	3.31
7	305	5.85	167	52.9	13	109.2	89	3.74
8	340	8.76	179	58.5	15	144.4	93	4.17
9	375	15.54	186	64.3	20	173.7	83	4.60

Aerobic Fitness at submaximal intensities:

	22/03/2009
Cadence at Lactate Threshold (rpm)	87
Cadence at Lactate Turnpoint (rpm)	89
Power at Lactate Threshold (W)	235
Power at Lactate Turnpoint (W)	305
Heart Rate at Lactate Threshold (bpm)	152
Heart Rate at Lactate Turnpoint (bpm)	167
VO2 at Lactate Threshold (ml/min)	3511
VO2 at Lactate Turnpoint (ml/min)	4314.5
% of VO_2 max at Lactate Threshold	67.0
% of VO_2 max at Lactate Turnpoint	82.4
% of Wmax at Lactate Threshold	63
% of Wmax at Lactate Turnpoint	81
Power at Fatmax (W)	235
VO2 at Fatmax (ml/min)	3511
% of VO2 at Fatmax	67.0
HR at Fatmax (bpm)	152






VO2 (ml/min)
VO2 in relation to Power output
6000
5000
4000
3000
2000
1000
0
0
50
100
150
200
250
300
350
400
Power (W)

Appendix 9: Results- Raw data

							DL VO2 max test					TT		
PRE		Sex m/f	Age (yrs)	Weight (kg)	Height (cm)	Cycling Exp (y)	PVO2 (ml/min)	VO2 max (ml/min)	VO2 max (ml/kg/min)	Wmax (W)	Max HR	EE TC kJ	TTC secs	TT av PO
SL	1	f	31	69.3	172.7	5	4031	3915	56.5	294	168	449.8	1952	227.0
	4	m	20	83.6	184.5	7	5079	4943	59.1	358	189	547.7	1996	274.4
	5	m	41	78.8	181.5	8	5326	4965	63.0	387	186	592.1	1674	355.3
	10	m	32	79.2	193.5	4	5408	5323	67.2	375	192	573.8	1819	315.4
	14	m	33	71.4	184.5	10	4990	4897	68.6	379	174	579.1	1753	330.4
DL	6	m	28	76.9	181.5	5	5331	5121	66.6	352	174	538.6	1920	283.3
	7	m	31	67.2	180.0	5	4381	4192	62.4	319	178	488.7	1812	269.8
	8	m	37	73.8	175.5	23	4408	4356	59.0	325	179	496.6	1720	289.5
	11	m	46	64.8	165.0	8	4233	4192	64.7	313	189	479.4	1857	258.2
	12	f	28	65.9	172.5	4	3644	3530	53.5	291	158	444.9	1743	255.3
	15	m	24	67.5	175.2	5	4598	4542	67.3	324	182	496.3	1765	281.2
	Av		32	72.6	178.8	8	4675.4	4543.2	62.6	338	179	517.01	1819	285.4
	SD		7.4	6.3	7.7	5	590.8	557.4	4.9	34	10	51.9	102.3	36.4
	SEM		2	1.9	2.3	1.6	178.1	168	1.5	10	3	16	31	11
	n		11	11	11	11	11	11	11	11	11	11	11	11

							DL VO2 max test					TT		
POST		Sex m/f	Age (yrs)	Weight (kg)	Height (cm)	Cycling Exp (y)	PVO2 (ml/min)	VO2 max (ml/min)	VO2 max (ml/kg/min)	Wmax (W)	Max HR	EE TC kJ	TTC secs	TT av PO
SL	1	f	31	69.5	172.7	5	4011	3967	57.0	294	170	449.8	1809	248.7
	4	m	20	81.5	184.5	7	5388	5239	64.3	375	186	547.7	1849	296.2
	5	m	41	75.5	181.5	8	5610	5488	72.7	393	186	592.1	1627	368.4
	10	m	32	79.5	193.5	4	5152	5012	63.1	377	198	573.8	1795	319.6
	14	m	33	71.5	184.5	10	5060	5020	70.2	376	169	579.1	1740	332.8
DL	6	m	28	77.6	181.5	5	5503	5421	69.9	354	171	538.6	1847	291.1
	7	m	31	66.6	180.0	5	4834	4477	67.3	330	181	488.7	1712	285.2
	8	m	37	74.7	175.5	23	4668	4523	60.6	342	179	496.6	1679	295.8
	11	m	46	63.8	165.0	8	4464	4415	69.3	321	188	479.4	1734	276.5
	12	f	28	63.6	172.5	4	3753	3595	56.6	301	162	444.9	1664	267.4
	15	m	24	67.6	175.2	5	4843	4732	70.0	333	186	496.3	1735	286.1
	Av		32	71.9	178.8	8	4844.2	4717.2	65.5	345	180	517.0	1745	297.1
	SD		7.4	6.3	7.7	5	593.1	595.7	5.6	33	11	51.9	73.5	32.9
	SEM		2	1.9	2.3	1.6	178.8	180	1.7	10	3	16	22	10
	n		11	11	11	11	11	11	11	11	11	11	11	11

	PRE	RL VO2 max					LL VO2 max				
		PVO2 (ml/min)	VO2 max (ml/min)	VO2 max (ml/kg/min)	% pre DL VO2 max	Wmax (W)	PVO2 (ml/min)	VO2 max (ml/min)	VO2 max (ml/kg/min)	% pre DL VO2 max	Wmax (W)
SL	1					175					176
	4	3301.0	3105.3	37.4	63	219	3092	2988	36.0	61	205
	5	4185.0	3864.0	50.7	81	245	4247	4143	54.4	86	249
	10	3893.0	3728.0	46.5	69	234	3749	3643	45.4	68	221
	14	3984.0	3802.2	53.9	79	228	3217	3074	43.6	64	212
DL	6	3876.0	3708.6	48.4	73	215	3929	3745	48.9	73	210
	7	3489.0	3169.8	46.9	75	200	3098	2980	44.1	71	196
	8	3228.0	3143.4	42.1	71	214	3220	3161	42.4	72	219
	11					205	3177	2781	43.0	66	197
	12	2657.0	2514.3	38.6	72	182	2880	2507	38.4	72	188
	15	3536.0	3459.5	51.8	77	218	3281	3180	47.6	71	205
	Av	3572.1	3388.4	46.2	73	212	3389.0	3220.2	44.4	70	207
	SD	471.5	443.3	5.8	5.3	20.9	435.1	488.4	5.2	6.9	19.3
	SEM	157	148	2	2	6	138	154	2	2	6
	n	9	9	9	9	11	10	10	10	10	11

	POST	RL VO2 max					LL VO2 max				
		PVO2 (ml/min)	VO2 max (ml/min)	VO2 max (ml/kg/min)	% pre DL VO2 max	Wmax (W)	PVO2 (ml/min)	VO2 max (ml/min)	VO2 max (ml/kg/min)	% pre DL VO2 max	Wmax (W)
SL	1	2856	2713	39.22	69	197	2667	2563	37	65	183
	4	3977	3513	43.37	73	233	3616	3517	43	73	227
	5	3952	3808	50.49	80	247	4273	4152	55	87	270
	10	3635	3553	44.48	66	225	3852	3583	45	67	213
	14	3696	3570	50.17	73	243	3448	3321	47	68	226
DL	6	3838	3641	47.20	71	213	3644	3469	45	68	209
	7	3747	3429	52.48	84	205	3313	3102	47	76	203
	8	3147	3065	41.30	70	220	3101	2949	40	67	217
	11	3123	3065	47.28	73	213	3201	3059	48	74	199
	12	2713	2603	40.87	76	202	2830	2669	42	78	191
	15	3502	3362	50.39	75	214	3389	3276	49	73	212
	Av	3471.5	3301.8	46.1	74	219	3394.00	3242	45.3	72	214
	SD	442.2	389.6	4.5	5.1	16.3	455.3	447.9	4.9	6.6	23.0
	SEM	133	117	1	2	5	137	135	1	2	7
	n	11	11	11	11	11	11	11	11	11	11

: Missing data due to Oxycon malfunction

Appendix 10: Proposed future study design: Optimization of the SL counterweight device

Laboratory set up will require careful alignment all thirteen Vicon cameras (Vicon Motion Analysis System-Oxford Metrics Ltd), surrounding a single, specifically modified, electromagnetically-braked cycle ergometer (Lode Excalibur Sport, Groningen, Netherlands).

Participants will report to the laboratory at specific allocated time slots (~90min each), participants will be tested separately. Once the skin is prepared correctly, four electromyography (EMG) electrodes will be positioned over the belly of the tibialis anterior, vastus lateralis, biceps femoris and gastrocnemius, plus 42 reflective markers will be placed at the appropriate anatomical points.

Prior to data collection participants will be allowed a DL cycling warm up (5min) at a ~ 60% W_{max}, followed by a short familiarization trial in the single leg cycling mode, with the right leg only.

The main data capture trial will consist of participants cycling in two different conditions (DL/SL). Primarily participants will cycle normally in the DL mode at four different workloads (90, 130, 170, 210W). Consecutively, once the cranks had been changed to accommodate SL cycling, each participant will then continue to complete SL cycling with four different counterweights (0, 5, 10, 12.5kg), pedalling with each at three different workloads (90, 130, 170W). Half the group will complete each condition in this order the other vice versa, in both conditions each participant will complete each set of workloads in a randomized order.

Each separate trial (DL/SL) will involve cycling for 90 s at a constant cadence of 80 rpm. Once cadence had stabilized (use of a metronome could help), a 30 s data caption will be implemented via the Vicon motion analysis system (kinematics sampled at a rate of 250Hz, EMG data sampled at 1000Hz); through which muscle activity and limb kinematics can be established. Participant heart rate (HR) and perceived exertion (RPE) will be recorded at the end of each data capture. Each data capture trial will be separated by one minute and different conditions separated by ~ 4 minutes whilst the counterweight or pedal is changed. Please refer to Figure 1 below.

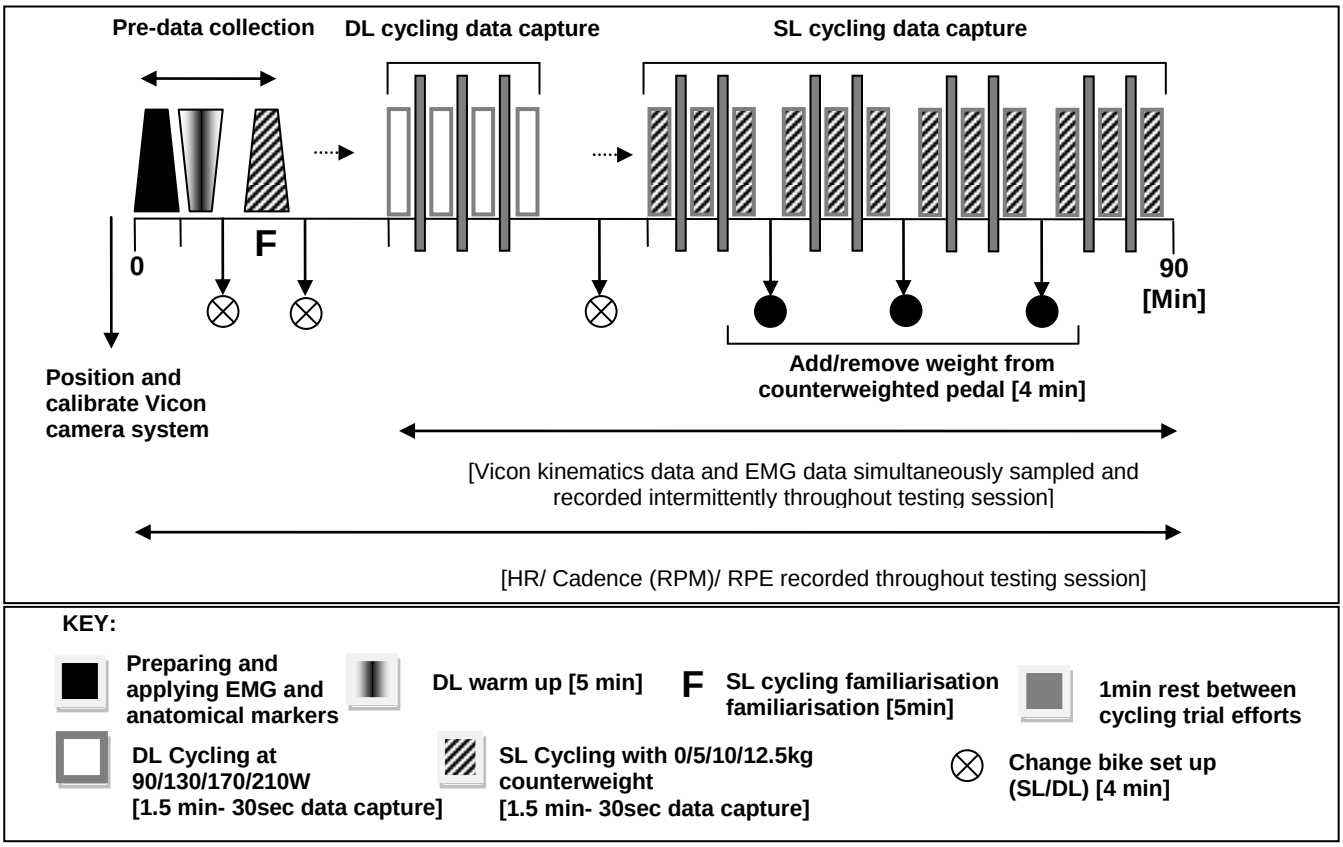


Figure 5.1: Counter weight optimization pilot study design- An investigation comparing differences in muscle activity and limb kinematics between DL cycling and SL cycling with different counterweights.

Data Analysis: Both kinematic and EMG data will be captured simultaneously via the Vicon camera system. Post testing session raw individual motion analysis data and EMG data will be labelled and transformed to .csv files in preparation for exporting into the computer software programme SciLab (INRIA. Le Chesnay, Cedex, France). Thus, allowing the trace of muscle activity (referring to our four chosen muscles) throughout the pedal stroke to be generated and comparisons drawn related to similarities between normal DLcycling and SL cycling with differing counterweights.

Appendix 11: Anecdotal inference derived from counterweighted SL cycle training practice

Despite a demonstrated lack of significant additive training impact of SL cycling, those participants recruited within the SL training group did identify some interesting anecdotal inferences pertaining to the usefulness of this alternative form of training. During the training sessions participants found the incremental training intensity necessary as at first the SL training regimen proved very quickly fatiguing. Anecdotally accessory muscle recruitment (i.e. increased activation of both gluteal and hip flexor muscles) was increased during the SL training in comparison to DL cycling exercise. Familiarizing all participants with the counterweight device prior to training and testing was very important, as participants did find the novel SL cycling technique plus the different seated position awkward at first. The most difficult aspect of the SL training regimen was reported to be the initial phase (10 -20 sec) of each 5min SL interval, within which inertia of the 10kg counterweight had to be overcome using just the single limb; a slight allowance was made for this via incremental loading of wattage during the primary 5- 10 pedal cycles.

Those who completed the SL training regimen reported feeling a decreased force sensation or an increased perception of strength in each individual limb when cycling normally at a given PO. This decreased force sensation post endurance training has been reported previously (Carafelli et al. 1995). Plus, two highly experienced cyclists within the SL training group felt they were able to initiate the downward phase of the pedal stroke earlier in the pedal cycle and with more initial force. Equally, the more familiar each participant became to the SL training modality the higher the cadence they were able to generate for the same power output. However, despite anecdotal inference for an increased sensation of power output, all SL trained participants perceived SL cycle training with a counterweight device as a lot harder than normal DL training of a similar intensity. This is despite similar average ratings for perceived limb exertion between groups. This anecdotal finding is in direct opposition of research completed recently by Abbis et al. (2011). Who described the counterweighted SL cycle training as a method which produces greater individual leg power output than DL cycling, with lower reported levels of perceived exertion. Such discrepancies in perceived exertion should be further investigated in a larger cohort, with a close comparison of the exact intensity of the SL training and detailed information on each athletes pre-training status and current training load.