

# **Hemodynamic changes in recreational cyclists following a long and a short interval high intensity cycling intervention**

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## Summary

The incorporation of high intensity interval training (HIIT) sessions into training plan is becoming increasingly popular due to the multitude of physiological benefits and time efficient nature of the training method. The physiological changes and specific adaptation pathways which follow the different variations of HIIT programmes is not fully understood. It was expected that the short interval HIT programme would be more successful in the improvement of hemodynamics in comparison to the long interval HIT. In addition that the short interval HIT would also improve aerobic and anaerobic ([La] buffering) metabolic performance parameters to a greater extent.

The present study used a similar HIIT programme as previously used (Rønnestad *et al.*, 2015). The previously used training programme was adapted to ensure the recreational cyclists (current study) were able to complete the programme. The two HIIT programmes were performed for the same work duration and the same sRPE, to ensure an equal training load (Inoue *et al.*, 2016). In addition, the use of a high RPE simulates the typical intensity that majority athletes train in real world settings (Seiler *et al.*, 2013).

The sixteen recreational cyclists who participated in the study were 21.9 ( $\pm$  2.75) years old, the range was 18 - 27 years. On average, the participants had 5.1 ( $\pm$  2.92) years cycling experience. The participants trained on average of 7.3 ( $\pm$  1.94) hours per week between both groups and ranged from four to ten hours a week before the start of the HIIT programme. In the pre-test phase before the HIIT programme, the average participant's mass was 77.3  $\pm$  7.79kg and the average %BF was 11.0  $\pm$  2.86% at the beginning of the trial.

The changes following the two HIIT programmes were assessed through the peripheral blood content ( $\Delta$ [O<sub>2</sub>Hb] and  $\Delta$ [HHb]), the aerobic performance parameters (VO<sub>2max</sub>, O<sub>2</sub> pulse, PPO and PO at OBLA) and lastly the anaerobic parameters were assessed during a repeated Wingate test ([La]<sub>max</sub>, PPO and %PR). The measurements of the hemodynamic changes were done with the use of near infrared spectroscopy (NIRS). The short interval HIT programme was more effective in the improvement of eight out of the eleven aerobic performance markers in comparison to the long interval HIT programme. The short interval HIT programme improved the  $\Delta$ [O<sub>2</sub>Hb] in the periphery during exercise. In contrast, the long interval HIT was more effective in the improvement of increased blood [La] accumulation following training.

It was found that the short interval HIT programme showed improvements in the increased O<sub>2</sub> availability in the periphery, the changes were suggested to be associated with central adaptations. The long interval HIT showed increased extraction of O<sub>2</sub> at OBLA, the improvements were suggested as mostly peripheral adaptations. The findings of the present study suggest that two similar HIIT programmes (which were matched for training load) showed different adaptation pathways provides an explanation for the differences in magnitude of changes seen. The changes associated to central adaptations were more pronounced following the six week training programme than the peripheral adaptations, as peripheral changes require a longer duration of training.

## Opsomming

Die toevoeging van hoë intensiteitsinterval inoefening (HIIT) sessies tot 'n inoefeningsplan groei in populariteit as gevolg van die veelvuldige fisiologiese voordele en tyddoeltreffende aard van die oefenmetode. Die fisiologiese veranderinge en spesifieke aanpassings van verskillende variasies van die HIIT-programme, word nie ten volle verstaan nie. Die verwagting sal wees dat 'n kort interval HIT-program meer suksesvol sal wees as 'n lang interval HIT-program om hemodinamiese verbetering tot volg te hê. Daarbenewens sal die kort interval HIT ook tot 'n groter mate aërobiese en anaërobiese ([La] buffer) metaboliese prestasieveranderlikes verbeter.

Die huidige studie gebruik 'n soortgelyke HIIT-program soos voorheen gebruik (Rønnestad *et al.*, 2015). Die voorheen gebruikte inoefeningsprogram is aangepas om te verseker dat die rekreasiefietsryers (huidige studie) die program kon voltooi. Die twee HIIT-programme is teen dieselfde werkdsuur en dieselfde sRPE uitgevoer om 'n gelyke oefenlading te verseker (Inoue *et al.*, 2016). Daarbenewens simuleer die gebruik van 'n hoë RPE (8-10) die tipiese intensiteit waarby meerderheidsatlete oefen (Seiler *et al.*, 2013).

Die sestien rekreasiefietsryers wat aan die studie deelgeneem het, was 21.9 ( $\pm$  2.75) jaar oud, die reikwydte was 18-27 jaar. Die deelnemers het gemiddeld 5.1 ( $\pm$  2.92) jaar fietsryervaring gehad. Beide inoefenings groepe het gemiddeld 7.3 ( $\pm$  1.94) uur per week geoefen, met vier tot tien uur per week voor die aanvang van die HIIT-program. Voor die aanvang van die HIIT-program was die gemiddelde massa van die deelnemers 77.3  $\pm$  7.79kg en die gemiddelde % liggaamsvet 11.0  $\pm$  2.86%.

Die veranderinge na aanvang van die twee HIIT-programme was geassesseer deur die perifere bloedinhoud ( $\Delta$ [O<sub>2</sub>Hb] en  $\Delta$ [HHb]), die aërobiese prestasieveranderlikes (VO<sub>2max</sub>, O<sub>2</sub> pulse, PPO en PO by OBLA) en laastens is die anaërobiese veranderlikes geassesseer tydens 'n herhaalde Wingate toets ([La] maksimum, PPO en % PR). Naby infrarooi spektroskopie (NIRS) was gebruik om die hemodinamiese veranderlikes te meet. Die kort interval HIT-program was meer effektief in die verbetering van agt uit die elf aërobiese prestasie merkers in vergelyking met die lang interval HIT-program. 'n Toename in O<sub>2</sub>-aflewering was gevind tydens oefening en herstel in die kort interval HIT-program. In teenstelling hiermee was die lang interval HIT meer effektief in die verbetering van verhoogde bloed [La] ophoping na inoefening.

Daar is bevind dat die kort interval-HIT-program verbeteringe toon met verhoogde O<sub>2</sub>-beskikbaarheid in die periferie. Die veranderinge word geassosieer met sentrale aanpassings. Die lang interval HIT het verhoogde ekstraksie van O<sub>2</sub> by OBLA getoon, wat geassosieer word met perifere aanpassings. Die bevindings van die huidige studie dui dat die aanpassings van twee soortgelyke HIIT-programme (ooreenstemde oefenlading) deur verskillende meganismes verduidelike kan word. Na die ses weke inoefenings program was die grootste verandering gesien in die sentrale aanpassings as die perifere aanpassings, aangesien perifere veranderinge 'n langer inoefenings tydperk verg.

# Glossary

%	: percentage
%PPO	: percentage of peak power output
%VO <sub>2max</sub>	: percentage of maximum oxygen consumption
[La]	: blood lactate concentration
[La] <sub>max</sub>	: maximal lactate concentration
Abs. VO <sub>2max</sub>	: absolute maximal oxygen consumption
ACSM	: American College of Sports Medicine
ADP	: adenosine diphosphate
ATP	: adenosine triphosphate
ATT	: adipose tissue thickness
BF%	: body fat percentage
BM	: body mass
bpm	: beats per minute
BSME	: Biering-Sørensen test of static muscular endurance
CHO	: carbohydrate
CI	: confidence interval
cm	: centimetre
CO <sub>2</sub>	: carbon dioxide
CON	: continuous exercise
CTCT	: Cape Town Cycle Tour
CV	: coefficient of variation
EMG	: electromyography
ES	: effect size
etc.	: etcetera
FA	: fatty acids
FFA	: free fatty acids
Fig.	: figure

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gr·dL <sup>-1</sup>	: grams per decilitre
H <sup>+</sup>	: hydrogen ion
Hb	: hemoglobin
HCO <sub>3</sub> <sup>-</sup>	: bicarbonate
HHb	: deoxygenated hemoglobin
HIIT	: high intensity interval training
HIT	: high intensity training
HR	: heart rate
HR <sub>max</sub>	: maximum heart rate
ICC	: inter-class correlation
k	: constant for scattering
kg	: kilogram
km	: kilometer
L·min <sup>-1</sup>	: litres per min
L·min <sup>-1</sup> ·kg <sup>-1</sup>	: litres per min per kilogram body mass
LI	: long interval
max	: maximum
Mb	: myoglobin
MICT	: moderate intensity continuous training
min	: minute
ml	: millilitre
ml·min <sup>-1</sup>	: millilitre per minute
ml·min <sup>-1</sup> ·kg <sup>-1</sup>	: millilitre per minute per kilogram body mass
mm	: millimeter
mmol·L <sup>-1</sup>	: millimoles per litre
%MMS	: percentage maximal muscle saturation
mVO <sub>2</sub>	: muscle oxygen consumption
N <sub>2</sub>	: nitrogen
NIR	: near infrared

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NIRS	: near infrared spectroscopy
nm	: nanometer
NO	: nitric oxide
nTHI	: total hemoglobin index
O <sub>2</sub>	: oxygen
OBLA	: onset of blood lactate accumulation
O <sub>2</sub> Hb	: oxygenated hemoglobin
O <sub>2</sub> Pulse	: oxygen pulse
P	: probability
PaCO <sub>2</sub>	: partial pressure of carbon dioxide
PGC-1 $\alpha$	: peroxisome proliferator-activated receptor gamma coactivator 1alpha
pH	: potential of hydrogen
PO	: power output
PPO	: peak power output
PPO: BM	: relative peak power output
PO@OBLA	: power output at the onset of blood lactate accumulation
PR%	: power of reproducibility percentage
Q <sub>max</sub>	: maximal cardiac output
<i>r</i>	: correlation coefficient
<i>r</i> <sup>2</sup>	: coefficient of determination
RBC	: red blood cells
R- value	: respiratory quotient
Rel. VO <sub>2max</sub>	: relative maximal oxygen consumption
ROS	: radioactive oxygen species
RPE	: rate of perceived exertion
RPM	: revolutions per minute
R <sub>s</sub>	: Spearman's rank-order correlation
s	: second
SD	: standard deviation

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SI	: short interval
TT	: time trial
VL	: vastus lateralis
VO <sub>2</sub>	: oxygen consumption
VO <sub>2max</sub>	: maximal oxygen consumption
vs.	: versus
w	: watts
W1	: first Wingate
W2	: second Wingate
yrs	: years
Z1	: zone 1
Z2	: zone 2
Δ[La]	: change in lactate concentration
Δ[HHb]	: change in deoxygenated hemoglobin concentration
Δ[O <sub>2</sub> Hb]	: change in oxygenated hemoglobin concentration
ΔW	: change in power
μL	: microlitre
μMol	: micromole



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# Chapter 1

## 1.1 Introduction

High intensity interval training (HIIT) or high intensity training (HIT) has been identified as an effective training method in various sports, however, a comprehensive understanding of the physiological adaptations in response to varying formats of HIIT is still lacking. Various athletic and health related benefits are associated with participation in a HIIT programme. Adaptations to the cardiorespiratory system, also referred to as central adaptations, have been well studied and have been documented as the main contributing factor to an increase in maximal aerobic capacity ( $VO_{2max}$ ). It has been found that HIIT elicits greater improvements in  $VO_{2max}$  than conventional endurance training (moderate intensity continuous exercise: MICT), especially in untrained and less fit individuals (Weston *et al.*, 2014). Nevertheless, only few studies have dealt with the peripheral adaptations associated with HIIT, among other, neuromuscular factors, blood buffering capacity, mitochondrial density and capillarization (Hoppeler *et al.*, 1985; Costes *et al.*, 2001; Neary *et al.*, 2002; Gliemann, 2016).

The secondary advantageous aspect to HIIT training is that an increased training stimulus is achieved in a shorter period of time. The shorter time spent training is advantageous for the majority of the population who do not have sufficient time to perform long training sessions. One such group is the recreational cycling community, identified as cyclists who participate in the sport for enjoyment or the maintenance of a healthy active lifestyle. The majority of recreational cyclists do not have access to coaches who tailor their individual cycling programs, yet they participate regularly in very demanding cycling events. The recreational cyclists will need to read up on various training methods to achieve their set goals and will most likely not have a full understanding of all the processes involved in the physiological adaptations they are aiming for. Therefore, further enlightenment into the topic of HIIT would be of great importance so that sport scientists can provide more definitive advice to cyclists while keeping their specific training goals in mind.

HIIT contains nine adjustable aspects and each alteration influences the stimulus that is applied. Aspects include exercise modality, interval length, interval intensity, recovery length, recovery intensity, series length, series number, recovery length between series and recovery intensity between series (Buchheit and Laursen, 2013a). Long interval HIT makes use of moderate exercise



intensity (threshold power/pace) over a longer interval period (greater than 2 min). It has been previously shown that long interval HIT at least maintains a high  $VO_{2max}$  in cyclists, but may also provide a stimulus for further  $VO_{2max}$  improvements (Neary *et al.*, 2002; Buchheit and Laursen, 2013a). Long interval HIT has also been used to increase the body's capability to endure high power outputs, as the body is placed under high stress for a prolonged period (Neary *et al.*, 2002; Daussin *et al.*, 2008; Buchheit and Laursen, 2013a).

Short interval HIT is recognized by the short period of high intensity exercise (at/above  $VO_{2max}$  power/pace). The general protocol used for short interval HIT programmes is usually an effort lasting between 15 to 45 s, and performed at an intensity higher than the longer intervals (Tschakert and Hofmann, 2013). The integration of short interval HIT into training programmes increases the body's capability to handle very high power outputs, as well as the accelerations associated with reaching these power values. Adaptations usually associated with this form of HIIT is improved Wingate performances (i.e. anaerobic exercise capacity), increased peak power output and increased tolerance for high levels of blood lactate (Creer *et al.*, 2004). Nevertheless, it is unclear whether the cardiovascular changes associated with a short interval HIT protocol are the result of central adaptation (i.e. stroke volume) and/or peripheral adaptations (i.e. increased  $O_2$  extraction) (Sloth *et al.*, 2013).

Another variable in HIIT programmes is rest length and intensity between exercise intervals, as this will ensure that a certain physiological benefit is elicited (i.e. increased  $VO_{2max}$ ). The short and long HIIT protocols of Rønnestad *et al.* (2015) makes use of a high work to rest ratio (2:1) with half the interval intensity. This methodology ensures that oxygen uptake remains above 90% of  $VO_{2max}$ . Rønnestad *et al.* (2015) found superior performance adaptations ( $VO_{2max}$ , power at  $VO_{2max}$  and an improved blood lactate profile) after a short interval HIT than a long interval HIT programme in highly trained cyclists. It was hypothesized that more favourable adaptations occurred after the short interval HIT, due to the higher training stimulus. However, local (muscle) adaptations could not be described, as muscle tissue analysis in the exercising muscle was not performed. It was speculated that the short interval HIT lead to improved lactate tolerance, however, lactate measurements were not obtained regularly enough to confirm this. Therefore, more research is required to examine the specific effects of various HIIT strategies on the localized hemodynamics of the active muscles (Neary, 2004).

Adaptation to training can be detected in the hemodynamics of the periphery through near-infrared spectrometry (NIRS). NIRS is a non-invasive technique that allows quantitative analysis of oxygenation in the tissue. NIRS uses the light oxygen-dependant absorbency of NIR light, sent off by the probe to hemoglobin/myoglobin (Hb/Mb, binding site of oxygen in blood); these values are used for the monitoring the oxygenation potential and efficiency of oxygen utilization. These results can be utilized to further individualize or periodize an athlete's training plan (Foster *et al.*, 1999).

Whole body measurement with NIRS is not currently feasible and only a small sample site is measurable at any one time. In cycling, the primary active muscles are the *vastus lateralis* (VL), *vastus medialis*, *gluteus maximus*, *semimembranosus*, *biceps femoris*, *gastrocnemius*, *soleus* and *tibialis anterior*. It was reported previously that the VL is one of the best representations of whole body cycling exercise due to its location under the participant's cycling shorts (less irritation and clear signal with minimal outside signal noise) and it is one of the main muscles involved in the pedalling movement (Zorgati *et al.*, 2015). NIRS has also been shown to be sensitive to whole body oxygen kinetic changes during various workloads from a small sample site (Bhambhani, 2004).

Muscle oxygenation is a measurement of how oxygen is delivered to the active skeletal muscles and how much of this is used in the aerobic metabolic energy production process. Adaptions that will lead to an increase in delivery of O<sub>2</sub> includes; increased oxygen carrying capacity of the blood, increased delivery of blood to the active tissue (cardiovascular adaptations) and capillarization of the tissue, to name a few. Adaptations that lead to an increased utilization of oxygen in the active muscle tissue include, among other, increased mitochondrial density and oxidative metabolic enzymes. Maintaining the balance of supply and demand for O<sub>2</sub> is essential for maintaining homeostasis in the active muscles and prolonging endurance capacity. Muscle oxygenation has yet to be explored with relation to HIIT intervention studies (Neal *et al.*, 2013; Rønnestad *et al.*, 2015). With the use of the non-invasive NIRS technique it is possible to map the local adaptations in the active muscles associated with these two training methods.

## 1.2 Motivation

From the literature it is clear that HIIT is an effective form of training to elicit favourable performance benefits. Previous studies have found differences in the performance outcomes and central adaptations of long interval HIT and short interval HIT. However, it is unknown how the duration of the intervals affects the peripheral adaptations. The best modality and variation of HIIT for each phase in a periodized training programme is unknown, as the peripheral adaptations first need comprehensive investigation. Similar central adaptations are seen with long interval HIT and short interval HIT, however, it is not known whether these two approaches may lead to different peripheral adaptations. It has been observed that the kinetics of peripheral muscle oxygenation (oxyhemoglobin and deoxyhemoglobin) have a marked correlation to systemic oxygen intake ( $\text{VO}_2$ ) (Kawagunchi *et al.*, 2001). However, the majority of the literature cannot comment as to whether improvements in various cardiovascular parameters can be attributed to either central or peripheral factors (Sloth *et al.*, 2013). With regard to peripheral adaptations, the influences of exercise duration and intensity has not been established due to a lack of data (MacInnis and Gibala, 2017).

Muscle oxygenation adaptation data can be utilized in the individualization of training programmes. This includes the phase (period) of training during which a specific HIIT would be recommended, thus ensuring the optimization of specific physiological adaptations.

## 1.3 Problem statement

### 1.3.1 Purpose of the study

The purpose of the study is to broaden our understanding of the physiological adaptations to HIIT in cycling. This will specifically be beneficial in the prescription of HIIT as part of a yearly periodization programme.

### 1.3.2 Research aims

1. To examine the hemodynamic changes (change in oxygenated hemoglobin concentration ( $\Delta[\text{O}_2\text{Hb}]$ ) and change in deoxygenated hemoglobin concentration ( $\Delta[\text{HHb}]$ ) following a short interval (SI) and long interval (LI) high intensity intervention (HIT) over 12 sessions in recreationally active cyclists.
2. To determine the cycling performance benefits following a short interval and long interval HIT intervention over 12 sessions.

### 1.3.3 Hypothesis

1. *A short interval HIT programme will illicit greater muscle oxygenation adaptations than a long interval HIT programme.* Changes in performance have been attributed to mostly central adaptations. However, peripheral adaptations should also be present as they have a causal link to the enhancement of the performance improvements (Costes *et al.*, 2001; Neary *et al.*, 2002; Daussin *et al.*, 2008).
2. *A short interval HIIT programme will elicit greater aerobic capacity changes in comparison to a long interval HIT.* Rønnestad *et al.* (2015) found significantly greater performance parameter improvements after a short interval HIT than a long interval HIT.
3. *Due to the increased number of accelerations from rest in the short interval HIT programme, there will be a greater improvement in performance parameters and higher levels of accumulated blood [La] during the repeated Wingate 30 s test compared with the long interval HIT programme.* An increased buffering capacity was previously identified by Sharp *et al.* (1986) following a similar short interval HIT (30 s format). The buffering capacity was identified with an increase in blood [La] (pre to post-testing) after a maximal intensity exercise test, with no change of pH.

## Chapter 2

### High Intensity Interval Training

#### 2.1 Introduction

High intensity interval training is a well-established exercise training method for the improvement of cardiorespiratory and metabolic function, with the added benefit of being time efficient. Interval based training makes use of periods of intense exercise, at an intensity which cannot be maintained for an extended period of time, separated by periods of rest, at either lowered intensity to promote active recovery, or complete rest/passive recovery.

There are a multitude of adjustable factors in HIIT protocols and these alterations are made in order to attain a certain stimulus and to adapt a particular physiological feature. Parameters which can be altered include; modality of exercise; work intensity, work duration, rest intensity, rest duration, number of repetitions in the set, the number of sets repeated and the duration and intensity of recovery in between sets (Buchheit and Laursen, 2013a). When the intensity and duration are altered in the periods of work and rest, the key factors in HIIT program design (Buchheit and Laursen, 2013a), there are nearly limitless combinations of interval training sessions and each of these adjusted factors can elicit various physiological benefits (Kilpatrick *et al.*, 2015). Appropriate manipulation of HIIT is very important, as one has to be aware of the middle to long term physiological adaptations, as well as the day to day and weekly training periodization considerations of each individual athlete. The micro cycle periodization is needed to avoid overload of the stimulus and to optimize physiological adaptations, as the possibility of a high training volume accumulation at high intensity is present. Thus the aim is to maximize the training stimulus and minimize the musculoskeletal injury risk (Buchheit and Laursen, 2013b).

Interval training has become one of the most popular training methods to improve various physiological and fitness parameters (Sindiani *et al.*, 2017). The major advantage of this training method for the vast majority of athletes is the capability to improve anaerobic and aerobic metabolic systems simultaneously, due to the significant load placed on both the neuromuscular and

cardiovascular system in a typical HIIT session (Sindiani *et al.*, 2017). In the last decade this training method has become an emerging trend not only among athletes, but also in individuals seeking health and fitness benefits, as it has been shown to increase training status and provide health benefits within a relatively short amount of time, compared with conventional training methods (Dunstan *et al.*, 2002; Kubukeli *et al.*, 2002; Guiraud *et al.*, 2010; Gibala *et al.*, 2012; Arena *et al.*, 2013; Milanovic *et al.*, 2015; MacInnis and Gabala, 2017).

Interval training has been cited in many ways in recent literature, of which the most common terms are high intensity training (HIT) and high intensity interval training (HIIT). Both of these variations refer to the same training methodology, however, HIT does not indicate that the intense periods are separated into intervals with periods of relief.

Two common HIT variations include the short interval HIT and long interval HIT. Short interval HIT refers to intervals between 15 - 45 s dispersed by various periods of lowered intensity exercise or complete rest. Long interval HIT refers to intervals between three to twenty min, and up to an hour in some studies (Buchheit and Laursen, 2013a).

The intensity at which these protocols are performed can be well managed with the use of the rate of perceived exertion (RPE) scale, due to its simplicity and versatility. The exertion for each session is selected as the maximal intensity of exercise which can be maintained for the required duration (Dishman *et al.*, 1987). RPE has been strongly correlated with heart rate ( $r = 0.74$ ;  $P < 0.001$ ) and blood lactate concentration ( $r = 0.83$ ;  $P < 0.001$ ), during simultaneous measurement at the end of each work load during an incremental step test. Scherr *et al.* (2013) showed that the fixed lactate thresholds of 3 and 4 mmol·L<sup>-1</sup> corresponded to an RPE of  $12.8 \pm 2.1$  and  $14.1 \pm 2.0$  on the 6 – 20 point Borg scale, respectively, in both an athletic and a leisure sport population.

Relatively little is known about the influence of exercise intensity, duration and frequency on the integrative physiological responses (central and peripheral) to interval training (MacInnis and Gabala, 2017).

## 2.2 Central Adaptations to Training

Physiological adaptations resulting in an improvement in oxygen delivery to the active muscle are regarded central adaptations and are primarily the result of prolonged aerobic training.  $VO_{2max}$ , reflecting the individual's maximal rate of aerobic energy generation, is associated with success in sport of an endurance nature. In whole body exercise such as running, cycling and rowing, it is widely accepted that  $VO_{2max}$  is usually limited because of the rate of oxygen delivery to the active muscle and not due to the muscle's utilization and extraction of oxygen from the localized blood (Jones and Carter, 2000).

$VO_{2max}$  is strongly related to the maximal cardiac output ( $Q_{max}$ ). High  $VO_{2max}$  and  $Q_{max}$  values are usually expressed in elite athletes and in turn related to high stroke volumes at maximal exercise. This is because maximal heart rate is often similar in sedentary individuals and elite athletes of the same age and somatotype. Following a training period, the exercising muscle will possibly require less blood flow at the same absolute submaximal intensity as before training because of a greater oxygen extraction by the active muscles. A higher  $Q_{max}$  is achieved by the increase in the left ventricle size and thickness of the myocardium, leading to an increase in the stroke volume and resulting in a larger blood volume output. These adaptations lead to a lower heart rate during submaximal exercise. The increased cardiac output and the enhanced extraction potential of oxygen by the active muscles during maximal exercise, results in higher values of  $VO_{2max}$  (Daussin *et al.*, 2008; Bækkerud *et al.*, 2016).

In addition to increased  $Q_{max}$ , the carrying capacity of oxygen in the blood is also greater following a specific endurance training intervention, due to the increase in total blood hemoglobin concentration ([Hb]). It is speculated that the increase in blood plasma volume following endurance training causes an increase in red blood cells, which probably offsets an anticipated reduction in [Hb] caused by prolonged weight-bearing exercise (Jones and Carter, 2000).

HIIT protocols, which elicit a greater demand of near-maximal or maximal oxygen intake have been linked to the greatest adaptation in  $VO_{2max}$ , due to the increased stress which is placed on the oxygen delivery and oxygen utilization systems (Buchheit and Laursen, 2013a). For an oxidative stimulus at an optimal level, the athlete should spend several min at an intensity close to  $VO_{2max}$  to allow large

motor unit recruitment and a near maximal cardiac output. This in turn signals the mechanisms responsible for oxidative adaptations in the skeletal muscle, as well as an enlargement of the myocardium (Burgomaster *et al.*, 2008; Gibala *et al.*, 2012).

Similar increases in  $VO_{2max}$  have been observed following short (15 - 45 s) and long (3 - 4 min) intervals when comparable volumes of high intensity training have been performed. An average improvement of 24% in  $VO_{2max}$  was reported for the short interval and a 16% improvement for the long interval programme in recreationally active, young, males after 20 sessions (Knuttgén *et al.*, 1973). Similar improvements in  $VO_{2max}$  were identified by Helgerud *et al.* (2007), reported a 6% improvement in the short interval and 9% improvement in the long interval programme in recreationally, young, active males after 24 sessions.

The exact mechanism(s) as to how the specific central and peripheral adaptations occur are still not known. However, it is speculated that with training at or near  $VO_{2max}$ , the relative high level of muscle fiber recruitment and alternate activation of the cardiovascular and cellular signalling pathways are major causal factors in the potent increase and adaptations in multiple endurance capacity markers (Kilpatrick *et al.*, 2015).

A faster reoxygenation rate following exercise has been marked as a potential benefit following HIIT, with a resultant improved metabolic waste removal. The main factor leading to the enhanced reoxygenation rate is an increase in oxygen delivery capacity to the active muscles. An improvement in  $O_2$  delivery capacity has been attributed to a multitude of training methodologies, from traditional endurance training to HIIT (Burgomaster *et al.*, 2008).



## 2.3 Peripheral Adaptions to Training

### 2.3.1 Aerobic adaptations

Skeletal muscle is a highly adaptable tissue and is capable of adaptations in both a metabolic and morphologic response to contractile activity, specifically with endurance training. Two important factors of aerobic exercise capacity include; skeletal muscle mitochondrion function and capillary density (Daussin *et al.*, 2008). Qualitative changes associated with mitochondrial adaptation include greater capacity to oxidize fuel substrates at varying exercise intensities, including carbohydrates (CHO) and fatty acids (FA). It was also found that a higher fitness status leads to less dependency on CHO and higher levels of FA oxidation during submaximal exercise. The greater energy contribution from FA leads to a maintenance of glycogen stores and increased exercise duration during submaximal exercise (Daussin *et al.*, 2008).

Mitochondrial biogenesis in the muscle has a 'master regulator', which is known as peroxisome-proliferator activated receptor  $\gamma$  coactivator (PGC)-1 $\alpha$  (Wu *et al.*, 1999). Low volume HIIT has been shown to activate the master regulator, possibly via reactive oxygen species (ROS) (Kang *et al.*, 2009). As little as six sessions over a 2 week period resulted in the increase of skeletal muscle oxidation capacity, which can be identified through the increase in maximal activity and protein content of the mitochondrial enzymes (Burgomaster *et al.*, 2006; Gibala *et al.*, 2006).

Skeletal muscle mitochondrial adaptation and  $VO_{2max}$  are mediated by the exercise intensity and regulate the specific response to the prescribed training. Physiological adaptations appear to be similar when low volume HIIT was compared to high volume, moderate intensity continuous training (MICT), even though the MICT had five times the training duration of the HIIT programme (MacInnis and Gabala, 2017).

An increase in capillary density enhances the ability of the muscles to extract more oxygen from the carriers present in the blood. Hoppeler *et al.* (1985) stated that changes in skeletal muscle capillarization requires weeks or months of training and changes appear to be less pronounced when exercise is performed at higher intensities. A greater expression of vascular endothelial growth factor

concentration is more readily associated with MICT, however, MacInnis and Gibala (2017) suggested that HIIT can be equally effective to increase vascular endothelial factor concentration.

Neary *et al.* (2002) studied the peripheral adaptations in response to HIIT by using NIRS. An improvement in a 20 km time trial performance was attributed to peripheral adaptations, as the maximum level of muscle deoxygenation was significantly enhanced post-training; however, the study compared HIIT to a conventional continuous endurance training method. Rønnestad *et al.* (2015) compared the adaptations between a short and long interval HIT, however, no peripheral measures were included. Therefore, no comments could be made on the localized adaptations and its associated performance related benefits after the two HIIT protocols.

Thus, it is concluded that further research is required to examine the changes in muscle oxygenation and deoxygenation measured by NIRS during maximal exercise in untrained and trained subjects. With the use of peripheral measurements, our understanding of the peripheral adaptations to training and fitness status of individuals can be broadened (Neary *et al.*, 2002; Rønnestad *et al.*, 2015).

### **2.3.2 Anaerobic adaptations**

Adaptations to the anaerobic metabolic system occur in multiple ways, one being improved skeletal muscle ion handling. This is achieved by the disturbance of muscle ion homeostasis, leading to an adaptation in the muscle ion transport proteins. A reduced muscle ion presence relates to a conducive energy production environment, as H<sup>+</sup> ions present (i.e. metabolic acidosis) in the muscle lead to a 'shutdown of energy producing machinery' in the muscle (Mohr *et al.*, 2007).

Buffering capacity is a rapid defence mechanism to prevent the disturbance of homeostasis caused by an increase in H<sup>+</sup> ion accumulation (Boning *et al.*, 1991). Blood buffering capacity is associated mainly with bicarbonate ion concentration [HCO<sub>3</sub><sup>-</sup>], plasmatic protein concentration (total blood base) and arterial pressure of CO<sub>2</sub> (PaCO<sub>2</sub>) (Ratel *et al.*, 2002). In practice, it means an athlete can exercise for longer and tolerate higher blood lactate concentrations, while maintaining blood pH levels. Improvements in muscle buffering capacity have been observed with both low and high intensity exercise.

A gold standard measurement has not yet been developed to directly assess the anaerobic energy contribution during high intensity exercise. The preferred methods for measurement are the accumulation of O<sub>2</sub> deficit at the onset of exercise and muscle lactate concentrations during exercise (Beneke *et al.*, 2011). The O<sub>2</sub> deficit has only been reported in a few HIIT studies (Tabata *et al.*, 1997; Vuorimaa *et al.*, 2000). It is speculated that HIIT protocols which elicit a higher post exercise blood lactate level will most likely stress the anaerobic metabolic system, as well as the lactate clearance pathway (Buchheit and Laursen, 2013a).

### **2.3.3 Neuromuscular adaptations**

Neuromuscular adaptations occur due to the high intensity (above 85% of power required to induce VO<sub>2max</sub>) and relatively low volume nature of HIIT. Improvements to the neuromuscular system will be predominantly present in higher intensity exercise, because low intensity exercise cannot induce the required stress in order for adaptation to occur (Buchheit and Laursen, 2013b). Bonachci *et al.* (2009) reported adaptations in the neuromuscular and musculoskeletal system in response to HIIT, whereby the tension in the locomotor muscles, tendons, joints and bone lead to remodelling and improvement in performance, as a function of the neural adjustments and improvements in the muscle's force generating capacity. These adaptations will improve locomotion characteristics (efficiency) and theoretically also improve the fatigue resistance of the lower limbs through the neuromuscular learning effect. Usually, low cadence HIIT is performed in cycling as a strength specific session for fatigue resistance benefits (Paton *et al.*, 2009).

Quantifying the neuromuscular load elicited by a HIIT training session is problematic, with limited studies available on the effect of cycling HIIT on the neuromuscular adaptations. The large variation in training status of participants in previous literature adds to the uncertainty of effects of different HIIT protocols on neuromuscular and musculoskeletal loading (Buchheit and Laursen, 2013b).

## 2.4 Short Interval High Intensity Training (short interval HIT)

Short interval high intensity training has been found to elicit similar physiological changes in comparison to traditional endurance training, as seen by selected markers of whole body and muscular adaptations (Rønnestad *et al.*, 2015).

A high level of muscle fiber recruitment and stress to type 2 muscle fibers has been shown during short interval HIT, because of the high exercise intensity (and greater stress) throughout the short interval. Gibala and McGee (2008) postulated that the increased recruitment of type 2 motor units may lead to adaptations in muscle oxidative capacity following a sprint HIIT (a short supramaximal effort of 5 – 30 s, separated by long rest periods of 4 min or more) (Buchheit and Laursen, 2013b). The higher stress levels are manifested in the increased presence of reactive oxygen species (ROS) (MacInnis and Gibala, 2017).

The main differences between a sprint HIIT and short interval HIT is the longer rest periods in the sprint interval programme. It is speculated that skeletal muscle remodelling after sprint HIIT seems to be mediated through the same signalling pathways involved in the promotion of oxidative capacity adaptations as traditional endurance training (Burgomaster *et al.*, 2008). Adaptations in oxidative capacity have been observed through marked increases in oxidative enzyme expression and endurance capacity during HIIT (Gibala and McGee, 2008).

Improvements in muscle glycogen content have been found in short interval HIT protocols, due to the large stress placed on anaerobic fuel production (Gibala *et al.*, 2006; Mohr *et al.*, 2007). Gibala and McGee (2008) found a marked improvement in glycogen stores, a lower rate of muscle catabolism and increased endurance capacity in healthy recreationally active individuals after 6 sessions sprint training over 2 weeks. Cycle time to exhaustion at 80% of PPO in the sprint group showed a statistically significant improvement ( $P < 0.05$ ), whereas the endurance training group showed no difference after two weeks. It was further deduced that short interval HIT caused an improvement in muscle buffering capacity, as the participants were able to exercise for longer at higher levels of blood lactate concentrations.

Short interval HIT has been found to be more effective than long interval HIT in already well-trained individuals, over the course of a 10 week training phase, where HIIT was performed twice a week (Rønnestad *et al.*, 2015).  $VO_{2max}$  was improved by 8.7% ( $P < 0.05$ ) in the short interval HIT group, whereas the long interval HIT improved by 2.6% ( $P > 0.05$ ). There was also a significant increase in PPO and  $PO_{OBLA}$  (8.5%;  $P < 0.05$  and 12.9%;  $P < 0.01$ ), compared with the long interval HIT group who improved by 1.6% ( $P = 0.33$ ) and 5% ( $P > 0.05$ ), respectively. In the same study, the mean power output during a 30 s Wingate test was significantly higher in the short interval HIT group (5%;  $P < 0.01$ ), compared with the long interval HIT group (1.4%;  $P = 0.3$ ).

Gibala and McGee (2008) are of the opinion that sprint interval training is more effective than endurance training to elicit aerobic and anaerobic training adaptations. The performance benefits with short interval HIT exceed those with low intensity endurance training and are far more time effective in achieving the desired results (Gibala and McGee, 2008).

## 2.5 Long Interval High Intensity Training (long interval HIT)

In the past it was thought that the only manner to improve the aerobic capacity of an athlete was to perform long sessions at relatively low exercise intensity. However, it has been shown that long interval HIT is more effective than the conventional low intensity, high volume training model (continuous training model) to bring about adaptations in aerobic capacity in recreationally trained cyclists (Bacon *et al.*, 2013; Seiler *et al.*, 2013).

Bacon *et al.* (2013) found that interval training programs which lasted on average 9 weeks resulted in a statistically significant increase in absolute  $VO_{2max}$  of  $0.85 \text{ L}\cdot\text{min}^{-1}$ . This finding was consistent across multiple studies (Knuttgen *et al.*, 1973; Winder *et al.*, 1979; Cox *et al.*, 1986; Hurley *et al.*, 1986; Roca *et al.*, 1992; Coggan *et al.*, 1993; Hickson *et al.*, 1997; Proctor *et al.*, 2001; Warburton *et al.*, 2004). It was concluded that HIIT, with intervals lasting between 3 - 5 min, caused significantly larger increases in  $VO_{2max}$  compared to moderate intensity continuous training (MICT) ( $P < 0.001$ ).

Neary *et al.* (2002) reported a 6% increase in  $VO_{2max}$  and a 4% increase in PPO in well-trained cyclists ( $P < 0.05$ ) over a three week long interval HIT program, while untrained individuals (Costes *et al.*, 2001) improved by 8% ( $P > 0.05$ ) following 4 weeks of intense training. Neary *et al.* (2002) also

showed a strong correlation between changes in  $VO_{2max}$  and PPO after training ( $r = 0.89$ ;  $P \leq 0.05$ ), as well as a significant correlation between improvement in 20 km TT performance and increase in tissue deoxygenation ( $r = -0.75$ ;  $P < 0.05$ ).

From the available evidence it seems that long interval HIT is an effective training method to improve various performance related indicators and is a more powerful training tool at improving endurance capacity in comparison to the MICT method.

## 2.6 Practical considerations

There are a few practical considerations to be kept in mind when dealing with HIIT and the associated training load which is placed on the athlete's physiological systems (Buchheit and Laursen, 2013b).

Training status needs to be considered when planning the introduction of HIIT into a training programme. The training status refers to the base fitness level of the athlete entering a HIIT program. An aerobic base platform (base fitness) is needed to facilitate the specific adaptations that are associated with HIT sessions (Laursen, 2010). Well-trained athletes are regarded more trainable and HIIT is also more effective in trained individuals. Untrained individuals also have a lower gross mechanical efficiency when compared to recreationally trained individuals (Hopker *et al.*, 2007). Furthermore, the same modality of training should be used for HIT than any other training. It is also recommended that lesser trained individuals perform HIIT at a lower intensity level and for longer interval time periods (Buchheit and Laursen, 2013b).

Another aspect which needs to be taken into consideration is the phase of training, which refers to the implementation of specific training dose during a specific phase in the periodization plan. This is done to ensure progression in training and reaching peak performance for a certain event (Buchheit and Laursen, 2013b).

The acute load placed on the neuromuscular system needs to be closely regulated to prevent acute and chronic injury risk. With respect to long term physiological development, the possibility of interference among the various physiological adaptations when other training needs are also addressed (i.e. concurrent training, demands of the specific sport, etc.) must be carefully planned and managed. Fatigue associated with post-HIIT sessions may lead to a reduction in force production

capacity and the rate at which the force can be applied in subsequent sessions. This will in turn lower the stimuli for optimal neuromuscular adaptations. Buchheit and Laursen (2013b) stressed that a happy medium needs to be found, with regard to neuromuscular and cardiovascular stress.

Lastly, the assessment of neuromuscular fatigue in response to HIIT should be carefully considered as muscle fatigue is very task specific. Countermovement jumps (CMJ) and sprint speed over a short distance are often used as measures of fatigue in runners. CMJ are a better reflection of the efficiency of the muscle contractile and the activation potential, while sprint speed can be affected by many confounding factors (Buchheit and Laursen, 2013b). In cycling, it would be preferable to use Wingate sprints to assess adaptation and performance changes in cycling.

## 2.7 Summary

Literature suggests that long and short exercise intervals with a work to rest ratio of less than one will allow the athlete to work for longer at an intensity equal to  $VO_{2max}$ , or at least greater than 90%  $VO_{2max}$ . Nevertheless, long term maximization of  $VO_{2max}$  development with associated performance benefits with the use of different HIIT sessions, as well as the most efficient way of accumulating time spent at  $VO_{2max}$  in a HIIT session still needs to be determined (Buchheit and Laursen, 2013a).

HIIT sessions with long intervals are probably best to ensure adaptations in cardiopulmonary function (i.e. central physiological factors) (Buchheit and Laursen, 2013a). It was suggested that the relationship between the intensity of the HIIT session and the acute neuromuscular performance probably follows a Gaussian distribution; high enough to induce post-activation potentiation leading to long term structural adaptations for fatigue resistance to high intense exercise. However, if the intensity is too high, there are associated impairments due to residual fatigue (carry-over effect) in subsequent sessions (Buchheit and Laursen, 2013b). Various programming methods are available to achieve progression in the training plan, which leads to the optimal performance of an athlete and assists in the prevention of injury; thus, in terms of risk/reward, HIIT is likely the best approach.

## Chapter 3

### Near-infrared Spectroscopy

#### 3.1 Introduction

The oxidative capacity of muscle is an important measurement of the peripheral or localized capacity of tissue to consume oxygen. In the case of skeletal muscle, exercise training causes adaptations in the size and number of mitochondria, oxidative enzymes, arterial blood flow and capillary network. Thus, the measurement of the oxidative capacity of muscle is an indicator of peripheral changes to exercise, opposed to central changes that are reflected by cardiovascular, respiratory and neuromuscular adaptations.

The measurement of muscle oxidative capacity was previously only possible via small samples of muscle being extracted for biopsy. Muscle biopsy is considered the gold standard in muscle oxidative capacity measurement; however, it is inconvenient and cumbersome due to the invasive nature of the procedure (Kime *et al.*, 2003). Local tissue inflammation and deoxygenation can alter the metabolic kinematics in needle biopsy specimens prepared on a later day, if taken from the same site. Van Thienen *et al.* (2014) reported a 10% reduction ( $P < 0.05$ ) in proximal total oxygenation index (TOI) after repeated muscle biopsies. A muscle biopsy also holds physical discomfort and potential risk for the individual.

Near-infrared spectrometry (NIRS) has been found a viable substitute for a muscle biopsy, with the advantage that a wide variety of hemodynamic kinetics can be measured non-invasively in various tissue (Quaresima *et al.*, 2003; Neary, 2004). NIRS has been used for many years to measure continuous changes in oxygenation in the tissue under the probe (Millikan, 1937), however, its application in a sport science setting is relatively new. At first, the measurements were only done during an acute exercise session; however, developments in NIRS technology with regard to the improvement of the hardware and the algorithms in the image processing of the light absorption signal (Quaresima *et al.*, 2003) have paved the way for more applications during exercise and training, as



well as the monitoring of changes in oxidative metabolic responses to various stimuli in both cerebral and skeletal muscle tissue (Neary, 2004).

NIRS is an instrument which can be used in the sports industry to provide an analysis of the muscle physiology when monitoring training adaptations (Neary, 2004). The first measurements reported outside of a laboratory setting was in 1992 (Chance *et al.*, 1992). The analysis of reoxygenation rates was performed on the quadriceps muscles of competitive elite rowers, after a state of maximal deoxygenation in the muscle tissue was generated through an exhaustive physical rowing exercise bout. The reoxygenation rate provided a non-invasive indication of oxygen delivery stress caused by exhaustive exercise and could be used to estimate the fitness status of the rower (faster reoxygenation rates would suggest higher fitness levels). Thus, reoxygenation data, in combination with a measure of muscle power output, can provide an indication of peripheral muscle adaptations in response to exercise training (Quaresima *et al.*, 2003).

NIRS can objectively evaluate the muscle oxidative metabolism in athletes following a singular training session. Thus, various therapeutic strategies (massage, compression garments, active recovery, ice bath therapy, etc.) can be compared and specific training session benefits can be identified in the hemodynamic data (Quaresima *et al.*, 2003). Specifically, recovery strategies can also be assessed for efficacy through muscle oxygenation recovery rate measurements.

Christmass *et al.* (1999) investigated the fuel utilization differences in physically active subjects during high intensity (HIIT) and submaximal, continuous (CON) exercise on separate days. The most important finding was a decrease in *vastus lateralis* oxygenation during work periods of the intermittent protocol. The results showed a difference in O<sub>2</sub> availability (TOI) in the muscle, as well as a difference in substrate utilization, even though similar overall muscle oxygen uptake (mVO<sub>2</sub>) and energy expenditure was observed. This type of information can be used when tailoring an athlete's diet around training, or when specifically targeting a particular fuel substrate during training.

## 3.2 Principle of measurements

NIRS utilizes near-infrared (NIR) light with wavelengths varying between 650 nm and 950 nm to penetrate the tissue (subcutaneous fat, muscle, brain, etc.). The light is absorbed by oxygen-dependant chromophores, either scattered in the tissue below the probe or lost to reflectance (Ferrari *et al.*, 2004). Chromophores are light absorbing complexes which selectively absorb light of a specific frequency. They are present in a metal (iron) complex at the centre of a tetrapyrrole macrocycle ring, which is found in the heme group of hemoglobin (Liu *et al.*, 1995). Oxygenated hemoglobin has a peak wavelength absorbency of approximately 850 nm and deoxygenated hemoglobin of approximately 760 nm. This variation of absorbencies allows for a clear determination of the deoxygenated hemoglobin level in comparison to the oxygenated hemoglobin level (Pereira, 2007).

The detector probe of the NIRS obtains a returned signal from the NIR light propagated tissue and the software applies the modified Beer-Lamberts law (Delpy and Cope, 1997) in the deconvolution of the received signal. Once the data has been processed, information is provided on a monitor that relates to tissue oxygen kinetics and metabolism. The NIRS signal is mainly derived from smaller arterioles, capillaries and venules. This can be attributed to the fact that larger blood vessels contain higher volumes of blood and in turn cause an almost complete absorption of the NIR light signal (Mancini *et al.*, 1994). The absorption of constantly dynamic variable chromophore concentrations (changes from oxyhemoglobin to deoxyhemoglobin) leads to the possibility of kinetic oxygen mapping potential, whereas the fixed concentrations of chromophores (melatonin in skin, etc.) or light scattering are theoretically considered constant rates (Ferrari *et al.*, 2004).

## 3.3 Variables

There are various outcome variables which can be measured with the use of NIRS. The most important variables, in the context of the current study, include: oxygenated hemoglobin ( $O_2Hb$ ) and deoxygenated hemoglobin (HHb).

**Oxygenated Hemoglobin ( $O_2Hb$ )** gives an indication of the presence of oxygen in the localized muscle and it is usually represented as a change in  $O_2Hb$  concentration ( $\Delta O_2Hb$ ). Conversion from  $O_2Hb$  to deoxygenated hemoglobin (HHb) indicates the  $O_2$  utilization of the resting or active muscle. A

positive linear relationship has been found between the conversion rate of [O<sub>2</sub>Hb] to [HHb] and intensity level of exercise (Colier *et al.*, 2001; Boone *et al.*, 2015). The main limiting factor of utilization of the O<sub>2</sub> attached to the Hb complex is the mitochondrial oxidative capacity; mitochondria's capacity for oxidative phosphorylation is the localized muscular potential for oxidative energy production during exercise performance (Ryan *et al.*, 2014). Recent studies found that  $\Delta$ [O<sub>2</sub>Hb] measurements may not only be useful in the measurement of local hemodynamic differences, but also in the characterization of changes in different muscle groups over time as a result of long-term training (Quaresima *et al.*, 2003; Neary, 2004; Buchheit and Ufland, 2011).

A comparison between athletes and controls (aged 19 - 23 yrs) during an incremental exercise test found that recovery time of  $\Delta$ [O<sub>2</sub>Hb] was statistically significantly faster in male athletes compared to healthy male controls ( $P < 0.05$ ). The effective rate at which the  $\Delta$ [O<sub>2</sub>Hb] decreases was also significantly different for the two groups ( $P < 0.05$ ). It was suggested that the recovery rate of  $\Delta$ [O<sub>2</sub>Hb] and the effective decrease in muscle  $\Delta$ [O<sub>2</sub>Hb] can be used as distinctive variables to characterise muscle O<sub>2</sub> metabolism during human movement (Ding *et al.*, 2001).

**Deoxygenated Hemoglobin (HHb)** indicates the deoxygenation level in the localized muscle. It can be used as an indication of O<sub>2</sub> extraction by the local tissue in response to an increase in exercise intensity and thus an increased utilization of O<sub>2</sub> (Usaj, 2001). It is usually represented as a change in concentration of HHb ( $\Delta$ [HHb]).

Grassi *et al.* (1999) found the inflection point (a breaking point in the linear increase to increased workload/threshold) of muscle deoxygenation closely correlates to point of inflection of blood [La] vs. the workload (watts) ( $r^2 = 0.95$ ;  $P = 0.0045$ ). The increased deoxygenation in the capillary bed of the exercising muscle leads to a rise in lactate concentration. Once a breaking point (i.e. the lactate threshold) has been surpassed, clearance is not able to match production rate and accumulation occurs. Boone *et al.* (2015) found a strong positive correlation between  $VO_{2max}$  and the breaking point in [HHb] ( $r = 0.76$ ;  $P < 0.001$ ) during a  $VO_{2max}$  step incremental cycle test with healthy participants of varying fitness levels. Therefore, a link exists between fitness status of an individual and muscle hemodynamic kinetics; better aerobically conditioned individuals possess a higher level of deoxygenation potential of Hb (at least 20% higher) compared to recreationally trained athletes.

Usaj (2001) studied the peripheral changes in the forearm muscle of healthy men (aged  $22 \pm 2$  years) following a continuous isometric training program over four weeks, five times a week, with the use of a hand grip dynamometer. The training intensity was higher than the maximum intensity obtained during the isometric muscle test and training time was increased throughout the training period. The isometric training led to increased isometric contraction time of the flexor digitorum in comparison to the pretest values. A statistically significant and strong correlation ( $r = 0.87$ ;  $P < 0.05$ ) was found between the time to exhaustion and relative concentration of HHb. Increased time to exhaustion was linked to a greater potential of muscle deoxygenation, rather than increased contraction potential.

### 3.4 Validity and reliability

There is sufficient evidence that NIRS is an objective measurement tool in the evaluation of localized muscle oxidative metabolism and hemodynamics (Neary, 2004).

NIRS has been validated in vivo against multiple testing modalities (Ferrari *et al.*, 2004), including the gold standard of muscle energy kinetics, namely high-resolution respirometry performed in permeabilized muscle fibers prepared from a muscle biopsy. Ryan *et al.* (2014) compared NIRS recovery kinetics of  $m\dot{V}O_2$  to the maximal ADP-stimulated mitochondrial respiration in muscle fiber bundles following a 10 - 20 s isometric contraction of the *vastus lateralis* in 21 active and healthy participants. A strong correlation ( $r = 0.61 - 0.74$ ;  $P < 0.01$ ) was found between the two different measurement techniques. Thus, it was concluded that NIRS provides a non-invasive, cost effective means of mitochondrial respiratory capacity measurement.

Sako *et al.* (2001) found a strong correlation between  $m\dot{V}O_2$  and magnetic resonance spectroscopy measurement of phosphate utilization ( $r = 0.97$ ;  $P < 0.001$ ) in the forearm muscle during a maximal voluntary handgrip exercise performed by twelve healthy men.

Miura *et al.* (2000) found a strong negative correlation ( $r = -0.93 - -0.99$ ;  $P = 0.01$ ) between TOI in the *vastus lateralis* and  $\dot{V}O_2$  measured by respiratory gas analysis in seven healthy men during 5 repeats of 6 min cycling at different workloads. A strong negative correlation was also found between TOI and blood [La] ( $r = -0.89$  to  $-0.99$   $P < 0.05$ ), as well as surface myoelectric activity measured with surface electrodes (EMG) ( $r = -0.95$  to  $-0.99$ ;  $P < 0.01$ ).

Repeated NIRS measurements were done in healthy men and within one week on the erector spinae muscle, using the Biering-Sørensen test of static muscular endurance (BSME) (Biering-Sørensen, 1983). The test-retest reliability for  $\Delta[\text{O}_2\text{Hb}]$  was statistically significant ( $ICC = 0.69 - 0.84$ ) (Kell *et al.*, 2004).

Pereira *et al.* (2005) investigated the reproducibility of  $\Delta[\text{O}_2\text{Hb}]$  in healthy men (aged  $37.2 \pm 10.2$  yrs). The men performed a maximum number of knee extensions at slow and fast movement rates and oxygenation was recorded in the *vastus lateralis*. Statistically significant reproducibility of the NIRS data was reported with the performance of knee extensions at a slow movement rate ( $r = 0.73 - 0.76$ ), as well as the fast movement rate ( $r = 0.85 - 0.97$ ). Tanimoto and Ishii (2006) reported similar reproducibility in  $\Delta[\text{O}_2\text{Hb}]$  ( $r = 0.85$ ) in young healthy men while performing isotonic knee extensions. Furthermore, the coefficient of variation (CV) for the rate of decrease in  $\Delta[\text{O}_2\text{Hb}]$  during the tests, which was performed on separate days (at the same time of day), was consistent throughout various intensities of rhythmic, isometric exercise of the flexor digitorum ( $16 - 25\%$ ) (van Beekvelt *et al.*, 2001). McCully *et al.* (1994) found the exponential time constant of muscle oxygenation recovery following strength exercise to have a CV of 15% between tests on different days and 5.7% between tests on the same day.

### 3.5 Factors affecting NIRS measurements

There are a number of limitations to the measurement capabilities of NIRS. One drawback involves the interference of adipose tissue thickness (ATT) with the NIRS signal. This occurs when ATT is too thick for the NIR light to penetrate enough to reach the muscle fiber. Because the NIR light cannot reach the muscle fiber bed, the measurement of oxidative metabolism will not be possible, or values will be lower than predicted (depending on the thickness of ATT). An ATT of greater than 15 mm has been found to influence the NIR light propagation significantly. A negative correlation ( $r = -0.70$ ) has been found between  $\Delta[\text{O}_2\text{Hb}]$  and ATT by van Beekvelt *et al.* (2001). When the ATT does not exceed 15 mm the NIR light can penetrate shallow regions of the muscle (Matsushita *et al.*, 1998).

Recently an algorithm has been formulated in order to adjust for the influence of ATT, however, it is limited to a specific thickness of ATT and the algorithm is not provided with commercial units (Niwayama *et al.*, 2000; Quaresima *et al.*, 2003). Therefore, exclusion of participants with an ATT

greater than 15 mm is recommended.

NIRS is unable to differentiate whether O<sub>2</sub> is released from myoglobin (Mb) or from hemoglobin (Hb). This occurs due to the fact that the two chromophores of Hb and Mb overlap in the infrared wavelength range. However, due to the small contribution of Mb in the oxygen transport system it is regarded negligible. Thus, it is assumed that most of the NIRS data represent a change in oxygenated hemoglobin and deoxygenated hemoglobin (Mancini *et al.*, 1994; Quaresima *et al.*, 2003).

It has been found that the age of participants influences NIRS readings. Costes *et al.* (1999) found that older persons ( $67 \pm 5$  yrs) have lower muscle O<sub>2</sub> saturation levels in the *vastus lateralis* when compared to younger persons ( $27 \pm 4$  yrs). This was explained by the age-related decrease in muscle blood flow which is a limiting factor in O<sub>2</sub> supply to the active muscle. Although aerobic muscle metabolism (conversion rate of O<sub>2</sub>Hb to HHb) was not affected by the aging process, the  $\Delta$ [O<sub>2</sub>Hb] and  $\Delta$ [HHb] recovery rates were significantly slower in the older persons due to impaired O<sub>2</sub> supply.

Pharmacological drugs or dietary supplementation (vitamins and minerals) has also been found to influence localized O<sub>2</sub> utilization. Thompson *et al.* (1996) found that nifedipine (an oral hypertension/angina drug) counteracted the O<sub>2</sub> deficit during recovery of muscle oxygenation following exercise. Nifedipine is classed as a calcium channel blocker helping to lower blood pressure by relaxing the blood vessels throughout the body. This leads to increased blood flow, providing a potential to match the required oxygen demands of exercise.

An increased level (higher than resting levels) of epinephrine and lactate has been found to lead to an increase in O<sub>2</sub> utilization, as investigated by Murakami *et al.* (2000). Maximal voluntary contractions were performed with one hand, while NIRS measurements were taken from the non-exercising flexor digitorum. This was done to ensure changes associated with NIRS variables were due to the epinephrine and lactate and not due to the fact that the muscle was exercising. Significantly strong correlations were found between increase of O<sub>2</sub> utilization and increase in blood lactate ( $r = 0.64$ ;  $P < 0.01$ ), as well as the increased level of O<sub>2</sub> utilization and blood epinephrine concentrations ( $r = 0.81$ ;  $P < 0.05$ ) of the non-exercising muscle. This indicates that if supplements or pharmaceutical drugs are taken which can lead to an increase in epinephrine or lactate concentration, the peripheral O<sub>2</sub>

utilization will be artificially high.

Minor variations in the NIRS-derived  $\Delta[\text{O}_2\text{Hb}]$  measurements of the *vastus lateralis* have been identified during repeated resistance exercise trials in healthy men, which were separated by six to seven days (Scott *et al.*, 2014). The authors speculated that these changes may be caused by minor changes in ambient or intramuscular temperature, as well as body posture during exercise.

### 3.6 Summary

The advantages of the NIRS technique is that the measurement is non-invasive, rapid and in real-time and at a fairly low running cost, whilst providing a high resolution image. The use of NIRS in sport has recently become exponentially prevalent due to the rapid development of technology. Literature has confirmed that NIRS is a valid, reliable and objective tool for the evaluation of localized muscle hemodynamics and oxidative metabolism. Measurements can either be taken during or after exercise and the test-retest reproducibility is sufficiently high to study both acute and chronic exercise training adaptations. NIRS can be used to assess changes in endurance status of an athlete and determine whether alterations need to be made to training prescription.

# Chapter 4

## Methodology

### 4.1 Study design

The study followed a pre-post experimental design with a random assignment of the sample of convenience into two experimental groups (Fig. 4.1). Following the familiarisation testing, all participants continued with their build-up to the Cape Town Cycle Tour. Pre testing followed the Cape Town Cycle Tour (CTCT) and then random allocation to the two training groups for the HIIT interventions was done. Post-testing followed directly after the completion of the HIIT interventions.

### 4.2 Participants

Twenty one healthy, recreationally active men were personally recruited from the Stellenbosch area and asked to volunteer for the study. The required sample size was calculated with G\*Power 3 (Faul *et al.*, 2007) and based on the results of Rønnestad *et al.* (2015). It was indicated that a total of 20 participants would be sufficient to detect a large practically significant change in maximal oxygen uptake ( $VO_{2max}$ ), with a power of 0.95 and a 5% level of significance.

All participants completed a health questionnaire (Appendix A) and training history questionnaire (Appendix B) for screening, an informed consent form (Appendix C) and dietary consumption form (Appendix D).

### 4.3 Inclusion criteria

Participants were included if:

- they were male and between the ages of 18-29 years old.
- they have competed in at least one CTCT or similar distance event in the past two years.
- they had entered into the 2017 edition of the CTCT.
- they performed endurance physical exercise, two to three times per week of at least 60 - 90 min per session, at the time of recruitment.
- they have two or more years of riding experience on a social or club level.



- they were studying/working on a full time basis during the study period, as this would classify the participant as recreational.

#### 4.4 Exclusion criteria

Participants were excluded if:

- they suffered from a recurring injury which had not fully recovered or at the time of testing, took medication which could have led to anomalies in the training responses (recommendation letter from a physician was required in the case of a recurring injury to be considered for the study).
- their skinfold thickness on the belly of the *vastus lateralis* exceeded 15 mm. Measurements of sedentary individuals have been reported at  $4.6 \pm 1.2$  mm for VL adipose tissue thickness (Cardinale *et al.*, 2007).
- they performed strenuous exercise 24 hours before each laboratory test visit. On the day of each test the participant was required to abstain from alcohol and caffeine 12 hours pre-test and not consume food three to four hours before. Water could be consumed in moderation.

#### 4.5 Place of study

All physiological performance tests were conducted in the Sport Physiology Laboratory at the Department of Sport Science, Stellenbosch University. The HIIT intervention phase was performed under supervision of the researcher.



**Figure 4.1**

Computrainer which was used for the study (Photograph taken by Kyle Basson)

# Study Design Layout:

**Recreational Cyclists**  
 -21 recreationally active cyclists were recruited from the Stellenbosch area and asked to volunteer for the study.

- ## Testing Battery
- Visit 1:
- Anthropometry & Hb level.
  - VO<sub>2Max</sub> Test (Aerobic) with Muscle Oxygenation using NIRS
- Visit 2:
- 30 s Wingate Protocol with [La] Recovery. (Anaerobic and Neuromuscular)

6 week HIIT  
 2 sessions per week

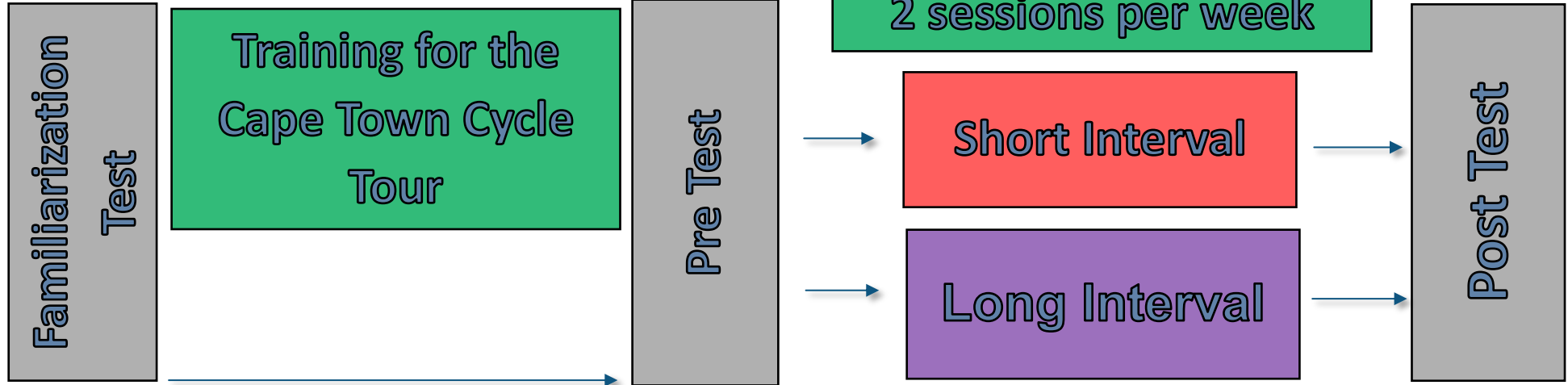


Figure 4.2 Study design layout

## 4.6 Procedures

The experimental period lasted approximately 15 weeks from the initial familiarization test to the final post-tests. It consisted of one week of familiarization testing in the laboratory; the CTCT training period; one week of pre-testing; six weeks HIIT intervention protocol (long intervals or short intervals) and one week of post-testing.

Testing was only done once an individual was found eligible for the study according to the health (Appendix A) and training history questionnaires (Appendix B), signed the informed consent form (Appendix C) and completed the 24 hour dietary list (Appendix D). The three sets of testing (familiarization test, pre-test and post-test) consisted of six laboratory visits in total.

During visit one the participants completed all the questionnaires, all testing procedures were explained and anthropometric measures were taken. Participants then performed a maximal aerobic capacity assessment ( $VO_{2max}$ ) with concurrent NIRS measurements. Participants returned to the laboratory a minimum of 48 hours later for Visit two, which consisted of the dietary questionnaire and maximal anaerobic (Wingate) test.

Following the familiarization test, the participants followed their own personal training program in the lead up to the CTCT. The 2017 edition of the CTCT was unfortunately cancelled, due to weather, fire and political unrest all occurring on the same weekend. The pre-test was performed following the date of the CTCT using the identical testing protocol as the familiarization test and dietary control diary.

The group was randomly divided into either the Long Interval High Intensity Training (long interval HIT) group or the Short Interval High Intensity Training (short interval HIT) group by means of an online randomizer programme ([www.randomizer.org](http://www.randomizer.org)). The HIIT interventions lasted approximately six weeks, with two HIIT sessions per week in the laboratory (Appendix E and F). The training zones for the interventions were individualized and based on the pre-test results. All HIIT sessions were performed in the laboratory on a Computrainer cycle ergometer with the participant's own bicycle, or on the Velotron cycle ergometer. Post-tests were performed following the specific HIIT intervention period and the same testing protocol was performed as the familiarization test. The HIIT intervention commenced a week after the CTCT for all participants, so that no races could confound the cyclists' responses to the HIIT programme.

Laboratory testing of cycling performance was performed at the same time of day for each participant, as circadian rhythms can significantly alter their performance and physiological responses. Diet was kept the same for each testing visit to exclude food and drink intake as confounding factors. The training performed (laboratory and other training) was recorded for the study duration in a monitoring questionnaire (Appendix H).

## 4.7 Blood [Hb] measurements

A finger stick blood sample was taken prior to the anthropometry measurements. The finger was cleaned with an alcohol swab and then pricked with an Accucheck Soft Clicks (Roche diagnostics, Mannheim, Germany). The first blood droplet was discarded and the second droplet was drawn into the capillary tube of the HemoCue Hb201+ (2016 HemoCue AB, Ängelholm, Sweden). The reading was taken at the end of the countdown and recorded. The normal range of hemoglobin is 13.5 – 17.5 gr·dL<sup>-1</sup> (ACSM guidelines, 2010; Nicoll *et al.*, 2012).

## 4.8 Anthropometric measurements

The anthropometrical measurements consisted of stature (height), body mass and skinfolds (Hosand Technologies srl, Verbania). The information was used to determine the participant's percentage body fat, using the BodyMetrix BX2000 software.

### 4.8.1 Stature

Stature is defined as the perpendicular distance between the transverse planes of the vertex and the inferior aspects of the feet (Marfell-Jones *et al.*, 2006). The stretch stature method was used and measurements were done with a sliding stadiometer (Seca, Germany). The participants were asked to stand barefoot on the scale with their heels together. The heels, buttocks and upper part of the back touched the scale. The head of the participant was in a Frankfort position. This is when the orbital (lower edge of the eye socket) and the tragon (the notch superior to the tragus of the ear) are horizontally aligned. The participant was then asked to take a deep breath and the head board was firmly placed down the vertex compressing the hair as much as possible. The measurement was then taken to the nearest 0.1 centimeter (cm).

## 4.8.2 Body mass

Body mass was measured using a calibrated electronic scale (UWE BW-150, 1997 model Brisbane Australia) recorded to the nearest 0.1kg. Participants were asked to stand barefoot in the middle of the scale, distributing their weight evenly on both legs. They were clothed in minimal and light weight cycling attire.

## 4.8.3 Body metrics

The BodyMetrix™ BX2000 (Hosand Technologies srl, Verbania) ultrasound system is a reliable measure for estimating %BF, FM and FFM, with intra-class correlations ranging from 0.84 - 0.98 (Smith-Ryan *et al.*, 2014). The device utilizes the Jackson and Pollock 7-site protocol to assess body composition (Jackson and Pollock, 1980). The measurements were taken at the following anatomical locations according to the manufacturer's recommendations:

- Chest • midway between the anterior axillary line and the nipple for men
- Scapula • the lateral side just below the bottom tip of the scapula
- Axilla • the mid-axillary line, level with the bottom of the sternum
- Triceps • midway between the acromion and radiale, on the mid-line of the posterior surface of the arm
- Waist • 5 cm lateral to the umbilicus
- Hip • most lateral aspect above the iliac crest
- Thigh • the anterior midline of the thigh midway between the patella and the crease of the hip

A pencil-eraser sized ultrasound gel was applied to the device and reapplied during the assessment when necessary. All of the measurements were taken on the right side of the body. Each site was measured 2 - 3 times to ensure reliability. When the 1<sup>st</sup> and 2<sup>nd</sup> measurement did not differ by more than 1 mm, the 2<sup>nd</sup> measurement was recorded. When the difference was larger, a 3<sup>rd</sup> measurement was taken and recorded.

#### 4.8.4 Subcutaneous fat layer of the *vastus lateralis*

The subcutaneous fat layer of muscles influences the optical path length of the NIRS measurements (Niwayama, *et al.*, 2000). Since NIRS probes generally have a penetration depth of around 50% of the inter-opted distance, it is recommended that adipose tissue thickness (ATT) be less than 25 mm to ensure photon penetration of the muscle tissue (Bhambhani, 2004). The BodyMetrix BX2000 was used to ensure that no participant had a measurement greater than 15 mm on the VL. Skin and adipose thickness were measured at the probe attachment site after the area was shaved to remove hair and cleaned using alcohol swabs to ensure no interference.

#### 4.9 Maximal exercise capacity test ( $VO_{2Max}$ )

Participants performed a maximal cycling test to determine their  $VO_{2max}$ . This  $VO_{2max}$  test result was used to determine the participant's exercise intensity for the HIIT intervention. A step incremental exercise test to exhaustion was performed on either the Computrainer (RacerMate, Seattle, USA) with their own bicycle or on the Velotron Dynafit Pro (RacerMate, Seattle, USA). The Cosmed Quark CPET (Rome, Italy) metabolic analyser was used for breath by breath analysis to calculate and record exercise intensity and selected cardio-respiratory parameters continuously throughout each test. Heart rate was measured through telemetry (COSMED wireless HR monitor, Italy) interfaced with the metabolic system. Gas analysers were calibrated to 16%  $O_2$ , 4%  $CO_2$  and balance  $N_2$  and the turbine flow meter was calibrated with a 3 L calibration syringe before each test.

The participants performed a 10 min warm-up at 80 w and a cadence of their choice. Participants were allowed to drink water after the warm-up and then the face mask and heart rate monitor were fitted. Participants started the test at 120 w and increased to 150 w after 60 s, thereafter the workload increased by 30 w every 150 s. The participants were asked to keep the cadence between 80 - 100 rpm throughout the test. In the last 30 s of each stage, a capillary blood sample (0.3  $\mu$ l) was taken from the finger and immediately analysed for blood lactate concentration (Lactate Pro 2 LT-1730, Japan). The exercise intensity increased until the participant reached exhaustion and was unable to maintain the cadence at or above 80 rpm, at which point the test was terminated. Peak power output (PPO) was calculated at this point of test cessation (Kuipers *et al.*, 1985).

The American College of Sports Medicine has described the test termination criteria to ensure maximal responses are recorded from all participants (McArdle *et al.*, 2010). These criteria include; (1) the  $\text{VO}_2$  does not increase by more than 150 ml per successive workload; (2) a respiratory quotient (R- value) equal or above 1.15 is reached; (3) the heart rate is more than 90% of the age predicted maximal heart rate; (4) the rating of perceived exertion is above 9 on the 1-10 Borg scale (Appendix G). Anyone of these criteria in combination with the participant indicating he was exhausted constituted a maximal test.



**Figure 4.3**

*Step incremental exercise test setup (Photograph taken by Kyle Basson)*

## 4.10 Blood [lactate] measurements

A finger prick blood sample was taken before the warm-up of the  $VO_{2max}$  test commenced. The finger was cleaned with an alcohol swab and then pricked with an Accucheck Soft Clicks (Roche diagnostics, Manhein, Germany). The first droplet of blood was wiped away and the second was drawn into the capillary tube of the Lactate Pro 2 meter (ARKRAY, Inc. Kyoto, Japan). The reading was taken at the end of the countdown and recorded. This was done 30 s before the end of each completed work load. The samples were continuously taken until the lactate concentration reached  $4 \text{ mmol}\cdot\text{L}^{-1}$ . The last sample was taken at the termination of the test.

## 4.11 Oxygen pulse

Stroke volume (SV) can be estimated through the calculation of the oxygen pulse ( $O_2$  pulse), which is a function of  $VO_2$  divided by heart rate (Bhambhani *et al.*, 1994; Neary *et al.*, 2002). A strong correlation ( $r = 0.85$ ) between  $O_2$  pulse and SV was found in a previous study involving men who performed lower body exercise (Bhambhani *et al.*, 1994).

## 4.12 Anaerobic Power measurements

The testing protocol consisted of two all-out 30 s sprints on the Wingate protocol and performed on an electrically braked cycle ergometer (Velotron Cycle Ergometer with RacerMate Wingate software, Seattle, USA). The all-out sprints were separated by 4 min recovery (Creer *et al.*, 2004). The cycle ergometer was adjusted to personal preferences of the participant to replicate their own bike setup as much as possible. Resting [La] was measured prior to the 10 min warm-up (Lactate Pro 2 Blood Lactate Test Meter, ARKRAY, Japan), 3 min into the first recovery phase, as well as 3, 6 and 9 min after the second Wingate test. For each of the sprints, peak power, mean power and power of reproducibility were recorded as outcome variables.

The mean and maximum Wingate power outputs were used to calculate the power of reproducibility (Hazell *et al.*, 2010). This variable indicates the consistency of the repeated bouts, which is affected by fatigue and the utilization of short-term energy systems (i.e. phosphocreatine-ATP synthesis).

$$\text{Power of Reproducibility: } ((PO_1 + PO_2)/2)/\text{best PO} \times 100$$

PO is power output (either mean or maximum)



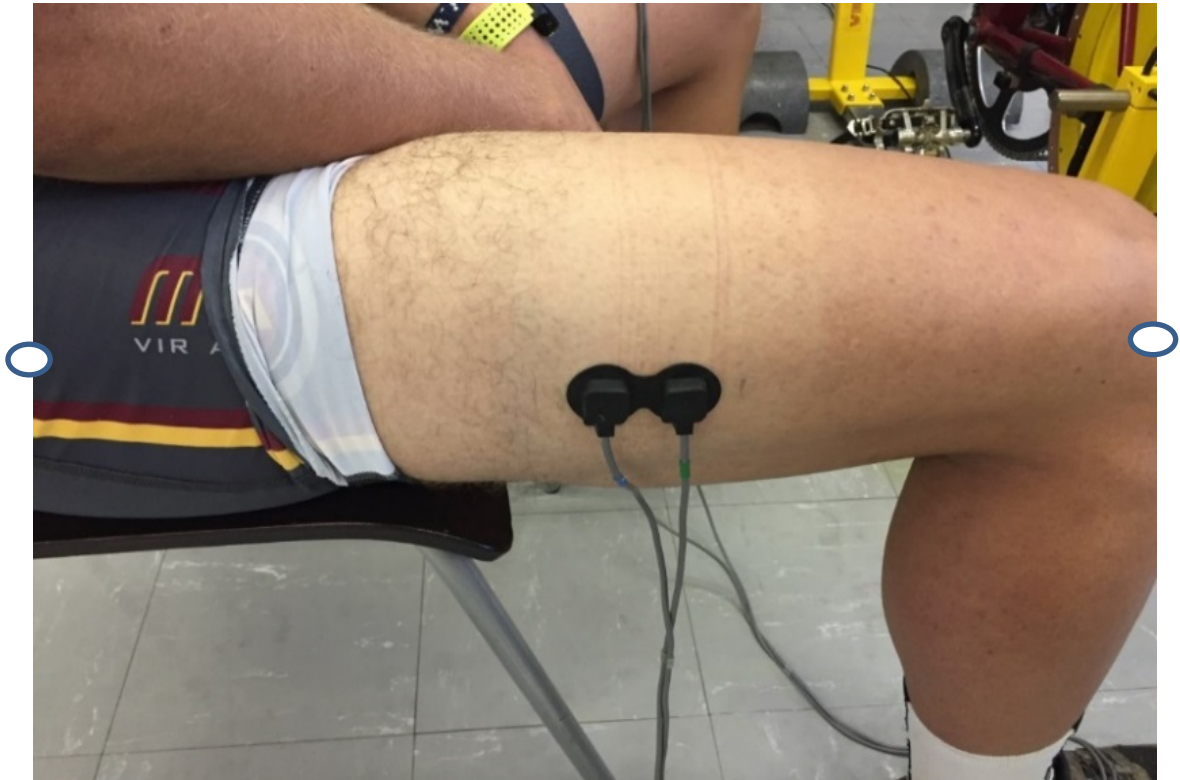
## 4.13 Muscle oxygenation measurements

Near Infra-Red Spectrometry (NIRO-200NX, Hamamatsu Photonics K.K. Germany) spatially resolved device was used concurrently during the incremental exercise test for the measurement of concentration of oxygenated hemoglobin ([O<sub>2</sub>Hb]) and deoxygenated hemoglobin ([HHb]). The Modified Beer-Lambert law was used by the NIRS hardware to calculate these parameters from the light absorption magnitude.

The NIRS probe was positioned on the belly of the *vastus lateralis* (VL) in a longitudinal direction on the midpoint between the head of the greater trochanter and the patella. The distance between the light emitter and detector probe was set at 35mm, ensuring the light penetration depth would be greater than 17.5 mm (Hamaoka *et al.*, 2007). A photo was taken of the placement to ensure the exact location of the NIRS probe was achieved with every test. The NIRS probes were secured with IV3000 stickers to keep the probe on the skin and eliminate light interference. The probes were also covered by a Velcro neoprene strap around the thigh. The NIRS device was calibrated to manufacturer's guidelines before each test.

The laboratory temperature was carefully controlled to 18 °C by the air conditioner to ensure the room temperature did not affect the readings. Data was collected from the start of the incremental exercise test until two min after the participant reached exhaustion. The initial baseline value of the NIRS variables prior to the start of the test was arbitrarily set to zero. The data were used for the assessment of the magnitude of changes from baseline (i.e. relative changes) in the outcome variables.

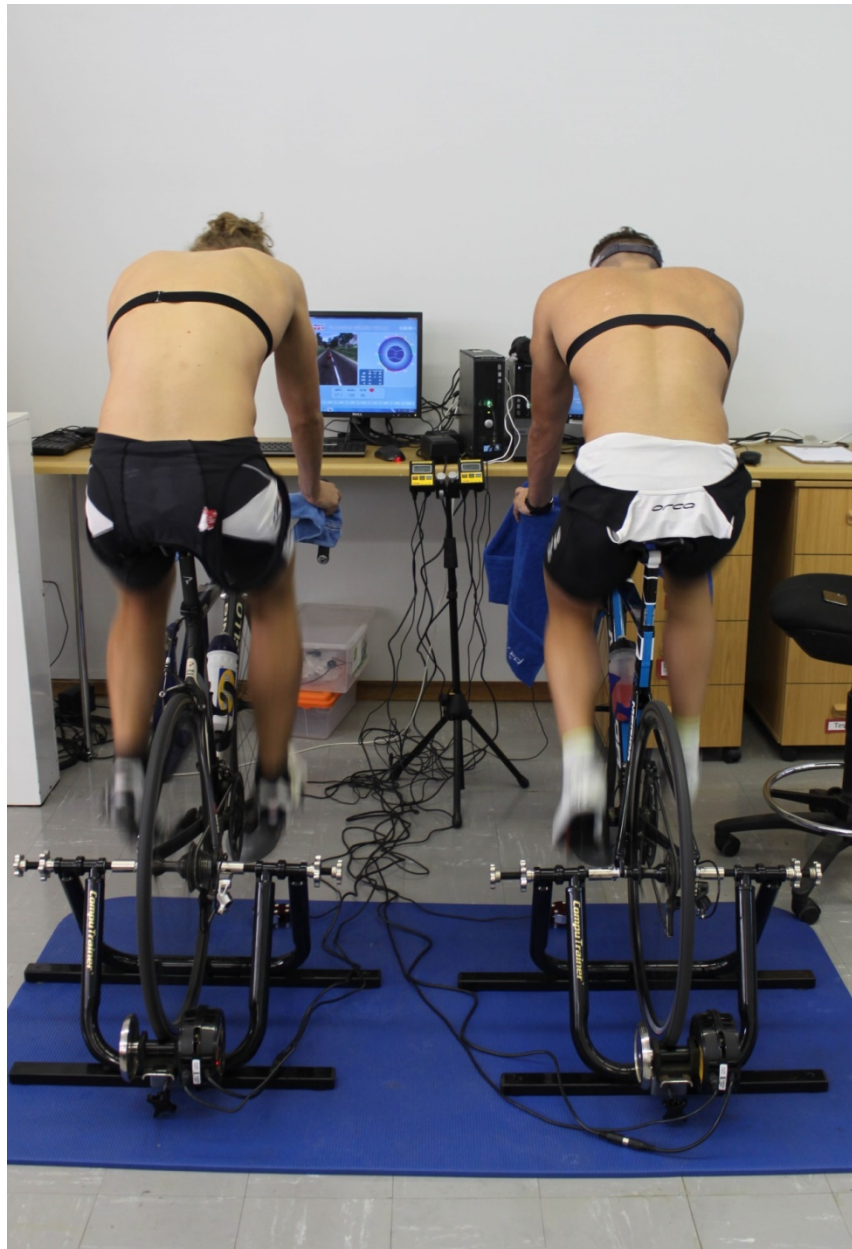
The NIRS data was separated into two 30 s zones, with the mean and standard deviation of the measurements calculated using Microsoft Excel (Microsoft Office 2010). The zones were defined as: Z1: the point at which the onset of blood lactate accumulation occurred and Z2: the point at peak power output (point of cessation of the test).



**Figure 4.4** NIRS probe attachment to vastus lateralis; the dots indicate the anatomic positions used to find the midpoint where the NIRS probe is attached (Photograph taken by Kyle Basson)

#### 4.14 HIIT intervention (Long and Short)

A detailed explanation of the HIIT protocol for the short interval HIT is provided in Appendix E and the long interval HIT protocol provided in Appendix F. The intervals were individualized for each participant; ensuring an optimal training stimulus was achieved for each session. Both HIIT protocols have been used in a previous intervention study (Rønnestad *et al.*, 2015) and even though the previous study used highly trained cyclists as participants, the training zones were specific to the participants' fitness level ensuring that the participant was able to complete the desired workloads. All training sessions were completed in the Sport Physiology Laboratory, in the department of Sport Science.



**Figure 4.5** HIIT session setup on the Computrainer (Photograph taken by Kyle Basson)

## 4.15 Statistical analysis

Statistical analyses were performed using Statistica version 13.2 (Dell 1984 - 2016) and Excel (Microsoft Office 2010). A custom built spreadsheet was used to perform the MBI analysis (Hopkins, 2017). The data was inspected for normality with normal probability plots. The present study's data followed a Gaussian distribution, as there were no statistically significant outcomes during the Levene's test for Homogeneity (Hill & Lewicki, 2006). Descriptive statistics are presented as either mean  $\pm$  SD or effect size (ES)  $\pm$  95 confidence interval (CI).

The NIRS data were averaged into a mean value for each phase and the baseline reading was subtracted from the mean of each phase to calculate the relative change in NIRS. A relative measurement was taken in order to make comparisons within and between groups possible. Baseline values refer to the NIRS data recorded before warm-up.

Two-by-two ANOVA's were used to determine a statistically significant difference as expressed by the test and time interaction (within-group changes) as well as the group and time interaction (between-group changes). Results of  $P < 0.05$  were considered statistically significant.

Cohen's effect sizes (ES) and 95% CI were calculated to compare the magnitude of differences within the groups and between the groups. The threshold for a difference to be considered practically important (the smallest worthwhile change, SWC) was set at 0.2 x between subject SD (Hopkins *et al.*, 2009). Quantitative interpretation of the threshold values was based on the guidelines of Hopkins *et al.* (2009): 0-0.19, trivial/negligible;  $\geq 0.2$  and  $< 0.6$ , small;  $\geq 0.6$  and  $< 1.2$ , moderate;  $\geq 1.2$  and  $< 2$ , large. The probability that the magnitude of change within the groups (pre- to post-testing) and the magnitude of differences between groups was greater than the practically important threshold (SWC) was rated as  $< 0.5\%$ , almost certainly not; 0.5-4.9%, very unlikely; 5-24.9%, unlikely; 25 - 74.9%, possibly; 75 - 94.9%, likely; 95 - 99.5%, very likely;  $> 99.5\%$ , almost certainly (Hopkins *et al.*, 2009). If the 95% CI crossed both the upper and lower boundaries of the practically important threshold (ES  $\pm$  0.2), the magnitude of change/difference was described as unclear.

Between-group comparisons in all outcome variables were summarized in forest plots (ES  $\pm$  95% CI). A positive effect size reflects a favouring towards the short interval HIT programme and a negative effect size reflects a favouring towards the long interval HIT programme.

The Spearman rank-order correlation coefficient was calculated to determine the relationship between the change in Wingate power output and the change in blood [La]. The 95 % confidence interval was calculated (<http://vassarstats.net/rho.html>) and tabulated together with the correlation coefficient. Correlation magnitudes and the effects of the training interventions were compared to standardized thresholds of 0.10 (small), 0.30 (moderate), 0.50 (large), 0.70 (very large) and 0.90 (extremely large). The chances of the true effect being at least that of the observed magnitude was interpreted using the probabilistic terms of Hopkins *et al.* (2009).

#### 4.16 Ethical aspects

Ethical clearance was obtained from the Ethics Committee for Human Research (Humanoria) at Stellenbosch University (SU-HSD-003945). All testing and laboratory procedures were performed in accordance with the *Declaration of Helsinki*. The tests and measurements in the study were standard cycle performance tests that are routinely done in high performance laboratories. The reliability and validity, as well as the safety of the tests have been determined (Gore, 2000).

The participants were informed that their participation was completely voluntary. Therefore, participants could withdraw from the project at any point in time. An informed consent form was completed by participants and they were given a clear explanation of the protocols and procedures that were used and were encouraged to ask questions. There were no serious risks involved in the study as all participants were healthy and physically fit; nonetheless participants may have experienced dizziness, fainting and discomfort during the exercise tests or HIIT sessions. The potential risks were minimized as much as possible by thoroughly explaining the procedure to the participants and carefully monitoring changes in the physiological variables. Participants were phoned 6 hours post-test for confirmation of wellbeing.

Blood samples were obtained non-invasively via a finger prick and did not exceed 2mL per test. Gloves, alcohol swabs and hermitically sterilized needles were used and all were sent for incineration post-testing in a biohazard collection bin.

HIIT has been shown to be safe and improve quality of life in patients who have coronary heart disease (Guiraud *et al.*, 2010; Kemi & Wisløf, 2010; Kessler *et al.*, 2012; Arena *et al.*, 2013) and it has been shown to improve the diseased states of these patients (Dunstan *et al.*, 2002; Gibala *et al.*,

2012; Weston *et al.*, 2014). HIIT has also been shown to be safe in elderly patients and has the ability to improve cognitive function following the exercise protocol (Heyn *et al.*, 2004; Casanova *et al.*, 2009; Murias *et al.*, 2010). Therefore, HIIT posed minimal to no risk for healthy, active individuals. Training was done under supervision of the researcher in the laboratory and if a participant appeared in distress, the training session was terminated. During the present study four sessions were terminated, by two participants, one from each HIIT programme. The sessions were repeated 48 hours later when the participant could complete the session properly.

## Chapter 5

### Results

#### 5.1 Descriptive characteristics

Twenty-one recreational cyclists volunteered to participate in the study. The participants were randomly split into a short interval (SI) high intensity training and a long interval (LI) high intensity training group. Five of the participants were excluded (SI = 2; LI = 3) from the final data set due to non-compliance (personal time constraints or health issues). Sixteen participants completed the study (SI = 8; LI = 8). The participants who completed the study all attended the training sessions in the laboratory over a period of 6 - 7 weeks. On average, the participants had 5.1 ( $\pm$  2.92) years cycling experience (Table 5.1a).

**Table 5.1a: Descriptive characteristics of the participants (mean  $\pm$  SD).**

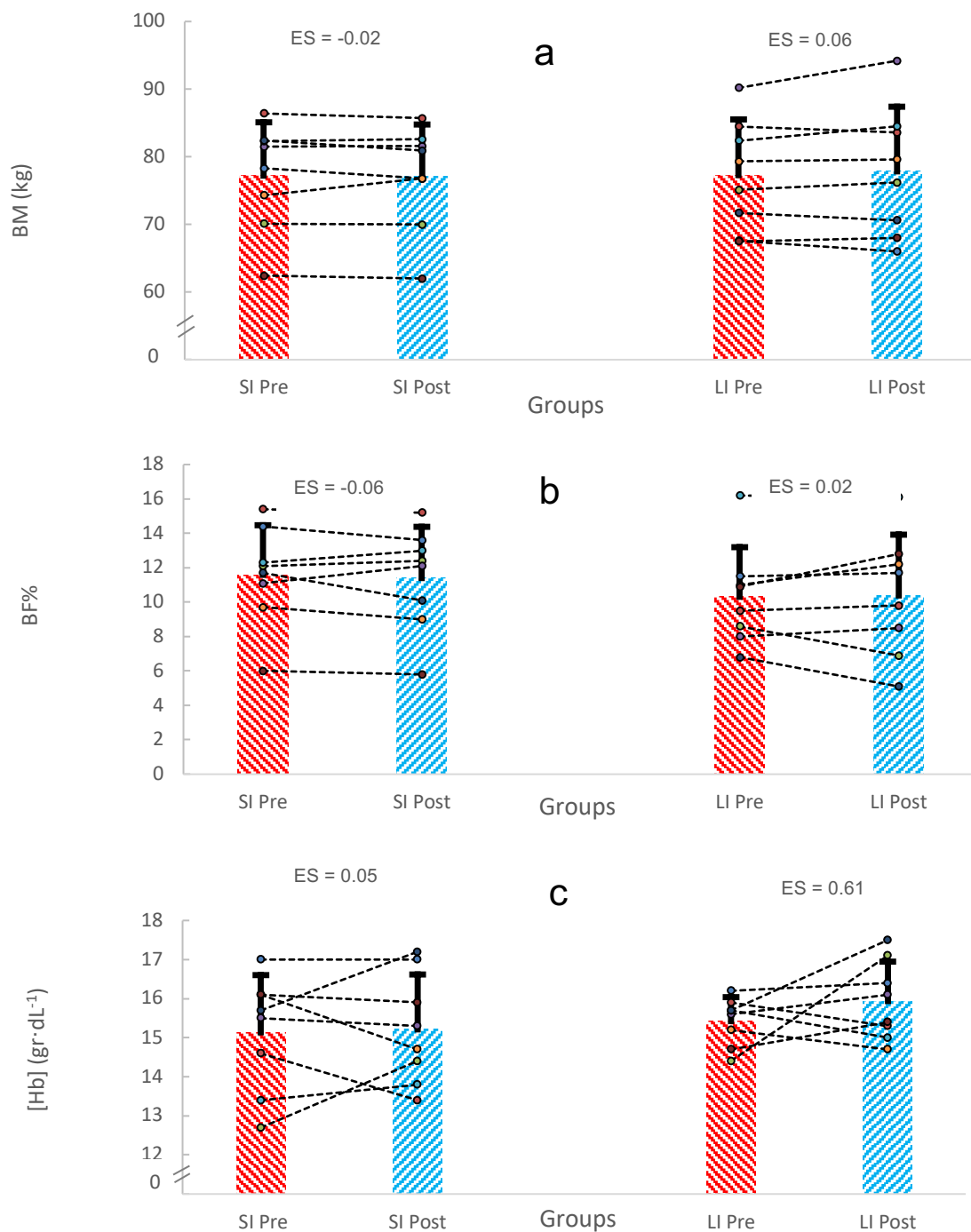
Variable	SI	LI	Total	ES (95% CI)	Qualitative outcome
n	8	8	16		
Age (yrs)	22.1 $\pm$ 2.80	21.6 $\pm$ 2.88	21.9 $\pm$ 2.75	0.18 (-0.82; 1.15)	Negligible
Height (cm)	183.2 $\pm$ 6.25	180.6 $\pm$ 4.50	181.9 $\pm$ 5.43	0.49 (-0.53; 1.45)	Small
Body mass (kg)	77.2 $\pm$ 7.89	77.3 $\pm$ 8.23	77.3 $\pm$ 7.79	-0.01 (-0.99; 0.97)	Negligible
% Body fat	11.6 $\pm$ 2.88	10.3 $\pm$ 2.88	11.0 $\pm$ 2.86	0.45 (-0.56; 1.42)	Small
[Hb] (gr·dL <sup>-1</sup> )	15.1 $\pm$ 1.46	15.4 $\pm$ 0.61	15.3 $\pm$ 1.09	-0.26 (-1.23; 0.74)	Small
Training age (years)	4.8 $\pm$ 3.18	5.4 $\pm$ 2.83	5.1 $\pm$ 2.92	-0.20 (-1.17; 0.79)	Small

[Hb]: hemoglobin concentration, kg: kilogram, gr: gram, dL: decilitre, SI: short interval and LI: long interval.

At baseline, there were no statistically significant differences in physical characteristics between the two groups ( $P > 0.05$ ) (Table 5.1a). The age range of the participants was 18 - 27 years with a mean age of 21.9 ( $\pm$  2.75) years. All, but one, of the participants had [Hb] values which were within the expected range of 13.5 and 17.5 gr·dL<sup>-1</sup> (ACSM, 2010; Nicoll *et al.*, 2012). One of the participants had a pre-test [Hb] of 12.7 gr·dL<sup>-1</sup>, however, at post-testing he had a higher [Hb] (14.4 gr·dL<sup>-1</sup>), which was above the minimum normative value. None of the participants were above the maximum value for [Hb]. None of the participants' thigh skinfold thickness exceeded 25 mm.

Figure 5.1a represents the within-group body mass (BM) changes from pre- to post-test. There was no statistically significant ( $P > 0.05$ ) or practically meaningful changes ( $ES < 0.20$ ) in either group. One cyclist in each group experienced a notable increase in BM; the SI participant increased his BM

by 2.5 kg and the LI participant increased his BM by 4.0 kg. Figure 5.1b and 5.1c illustrate that no statistically significant ( $P > 0.05$ ) or practically important changes ( $ES < 0.20$ ) occurred in the body fat percentage during post-testing. However, the mean [Hb] of the LI group ( $+0.5 \text{ gr}\cdot\text{dL}^{-1} \pm 1.21$ ;  $ES = 0.61$ ) were likely higher during post-testing, whereas the change in the SI group was negligible ( $P > 0.05$ ;  $ES < 0.20$ ).



**Figure 5.1**

Descriptive data for within-group changes in (a) body mass, (b) percent body fat and (c) hemoglobin concentration. The columns denote group means  $\pm$  SD and lines indicate the responses of the individuals. BM: body mass in kilograms, BF%: body fat percentage, [Hb]: hemoglobin concentration, gr·dL<sup>-1</sup>: grams per decilitre, SI: short interval and LI: long interval.



There were no statistically significant between-group differences in the personal characteristics of the groups ( $P > 0.05$ ) (Table 5.1b). The chance that the LI group experienced truly larger changes in BM (0.6 kg more), BF% (0.1% more) and [Hb] ( $0.5 \text{ gr}\cdot\text{dL}^{-1}$  higher) following the intervention is likely small.

**Table 5.1b: Between-group differences in personal characteristics (mean  $\pm$  SD).**

	Changes between groups				
	SI mean difference	LI mean difference	ES (95% CI)	Qualitative outcome	% chances of greater/trivial/smaller effect
Body mass (kg)	$-0.2 \pm 1.27$	$0.6 \pm 1.90$	$-0.43 (-1.40; 0.58)$	Small	11/21/69
Body fat (%)	$-0.2 \pm 0.85$	$0.1 \pm 1.26$	$-0.28 (-1.25; 0.72)$	Small	19/27/53
[Hb] ( $\text{gr}\cdot\text{dL}^{-1}$ )	$0.1 \pm 1.12$	$0.5 \pm 1.21$	$-0.34 (-1.31; 0.66)$	Small	13/23/63

[Hb]: hemoglobin concentration, kg: kilogram, gr: gram, dL: decilitre, SI: short interval and LI: long interval

## 5.2 Training characteristics

Table 5.2a depicts the cyclists' training time, outside of the laboratory, before and after the intervention. Pre-testing training hours were calculated as the average of the previous six weeks' training. The training hours ranged from four to ten hours a week, with an average of  $7.3 (\pm 1.94)$  hours per week between both groups.

The groups decreased their training hours during the intervention by 32% (SI) and 14% (LI), however, the decrease was only statistically significant for the SI group ( $P = 0.04$ ). As a result of the moderate decrease in training time of the SI group, the between-group difference in training hours suggested a possibly moderate decreasing effect (ES =  $-0.61$ ). There was neither a statistically, nor a practically meaningful between-group difference in mean HIIT work periods (ES =  $0.04$ ) and mean RPE (ES =  $-0.07$ ) during the interventions in the laboratory (Table 5.2b).

**Table 5.2a: Within-group changes following HIIT intervention (mean  $\pm$  SD).**

	Pre	Post	ES (95% CI)	Qualitative outcome	% chances of increase/trivial/decrease effect
SI training hours	$7.5 \pm 1.75$	$5.1 \pm 2.50$	$-1.11 (-2.10; -0.01)$	Moderate	1/3/96
LI training hours	$7.0 \pm 2.20$	$6.0 \pm 2.57$	$-0.42 (-1.38; 0.59)$	Small	12/21/67

SI: short interval and LI: long interval

**Table 5.2b: Between-group comparison in training time and intensity level during the training interventions (mean  $\pm$  SD).**

	SI	LI	ES (95% CI)	Qualitative outcome	% chances of increase/trivial/decrease effect
HIIT session work period (min)	12.3 $\pm$ 2.23	12.2 $\pm$ 2.86	0.04 (-0.94; 1.02)	Negligible	38/30/32
Mean session RPE	7.9 $\pm$ 0.81	7.9 $\pm$ 0.74	-0.07 (-1.05; 0.91)	Negligible	29/30/41

RPE: rate of perceived exertion, SI: short interval and LI: long interval

### 5.3 Maximal aerobic capacity

The cycle incremental exercise test to exhaustion was used to assess the aerobic capacity of the participants before and after the intervention. All the tests (pre- and post-intervention) met the ACSM criteria for a valid maximal exercise test (McArdle *et al.*, 2010).

#### 5.3.1 Within-group changes

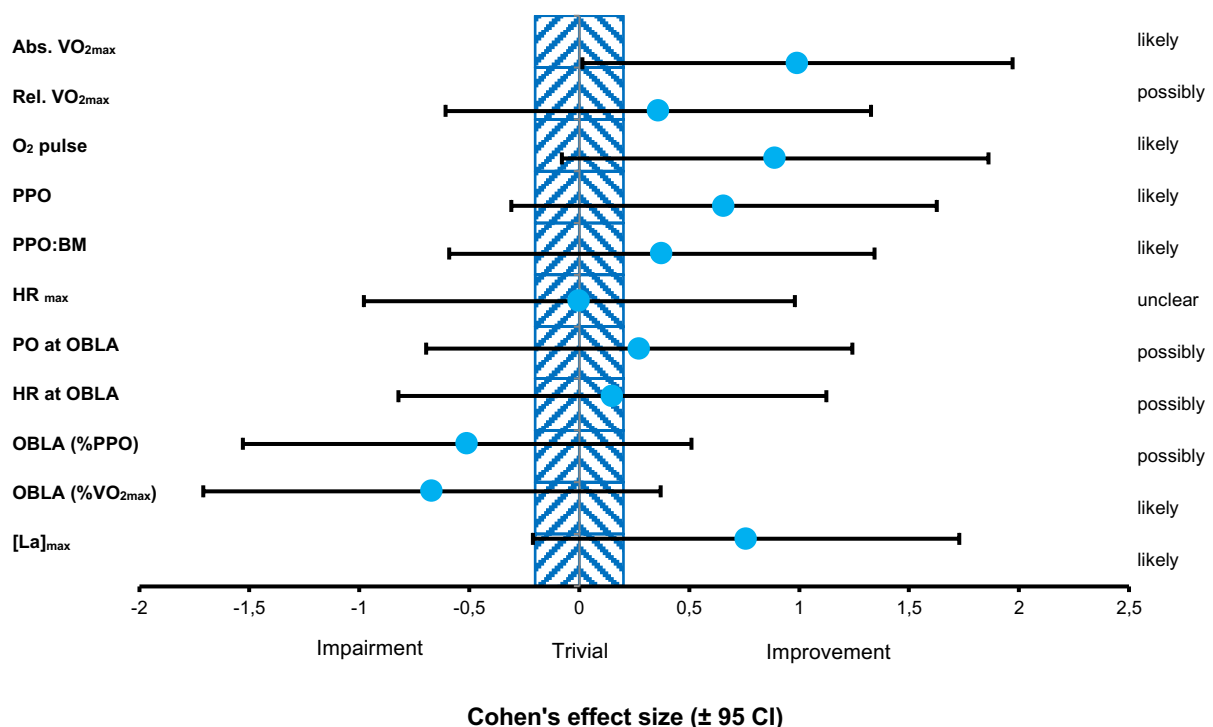
Table 5.3a (SI training group) and table 5.3b (LI training group) present the within-group changes in aerobic capacity measurements in response to the intervention. The results show no statistically significant changes in maximal aerobic capacity in either the SI group (Table 5.3a), or the LI group (Table 5.3b). However, with the exception of HR<sub>max</sub> and HR at OBLA, the SI training intervention likely caused small to moderate improvements in maximal aerobic capacity (Fig. 5.2).

The SI training group experienced a moderate increase in absolute VO<sub>2max</sub> (+6.4%; ES = 0.99), while peak power output (+ 6.9%; ES = 0.66) and peak blood lactate concentration (+22.5%; ES = 0.76) were moderately higher. In contrast, OBLA, expressed as %PPO (-2.3%; ES = -0.30) and %VO<sub>2max</sub> (-4.1%; ES = -0.65) were moderately lower after the intervention.

**Table 5.3a: Within-group changes (pre- to post-intervention) in maximal exercise capacity for the short interval HIT group (mean  $\pm$  SD).**

Short Interval					
	Pre	Post	ES (95% CI)	Qualitative outcome	% chances of better/trivial/poorer effect
VO <sub>2max</sub> (ml·min <sup>-1</sup> )	4061.5 $\pm$ 204.79	4322.9 $\pm$ 311.60	0.99 (-0.10; 1.97)	Moderate	94/5/2
VO <sub>2max</sub> (ml·min <sup>-1</sup> ·kg <sup>-1</sup> )	53.3 $\pm$ 8.20	56.2 $\pm$ 7.97	0.36 (-0.65; 1.33)	Small	74/3/23
O <sub>2</sub> pulse (ml·min <sup>-1</sup> ·bpm <sup>-1</sup> )	20.7 $\pm$ 1.26	22.0 $\pm$ 1.72	0.89 (-0.18; 1.86)	Moderate	90/7/3
PPO (w)	319.5 $\pm$ 31.88	341.6 $\pm$ 35.25	0.66 (-0.38; 1.62)	Moderate	87/5/8
PPO:BM (w·kg <sup>-1</sup> )	4.2 $\pm$ 0.78	4.5 $\pm$ 0.82	0.37 (-0.63; 1.34)	Small	75/3/22
HR <sub>max</sub> (bpm)	196.4 $\pm$ 6.21	196.4 $\pm$ 6.37	0.00 (-0.98; 0.98)	Negligible	50/0/50
PO at OBLA (w)	252.1 $\pm$ 28.64	260.6 $\pm$ 33.63	0.27 (-0.73; 1.24)	Small	69/2/29
HR at OBLA (bpm)	174.9 $\pm$ 8.87	176.0 $\pm$ 5.81	0.15 (-0.84; 1.12)	Negligible	61/1/38
OBLA (%PPO)	78.9 $\pm$ 4.61	76.3 $\pm$ 5.51	-0.51 (-1.48; 0.51)	Small	9/18/73
OBLA (%VO <sub>2max</sub> )	82.2 $\pm$ 5.38	78.8 $\pm$ 4.97	-0.67 (-1.63; 0.37)	Moderate	6/12/82
[La] <sub>max</sub> (mmol·L <sup>-1</sup> )	15.1 $\pm$ 3.76	18.5 $\pm$ 5.11	0.76 (-0.29; 1.73)	Moderate	89/5/5

*Abs. VO<sub>2max</sub>*: absolute maximal oxygen uptake in millilitres per minute, *Rel. VO<sub>2max</sub>*: relative maximal oxygen uptake, *O<sub>2</sub> pulse*: Oxygen pulse, *PPO*: peak power output, *PPO:BM*: peak power output per kilogram body mass, *HR<sub>max</sub>*: maximal heart rate, *PO at OBLA*: power output at onset of blood lactate accumulation, *HR at OBLA*: heart rate at onset of blood lactate accumulation, *OBLA (%PPO)*: percentage of peak power output at onset of blood lactate accumulation, *OBLA (%VO<sub>2max</sub>)*: percentage of maximal oxygen uptake at onset of blood lactate accumulation and *[La]<sub>max</sub>*: maximal lactate concentration.

**Figure 5.2**

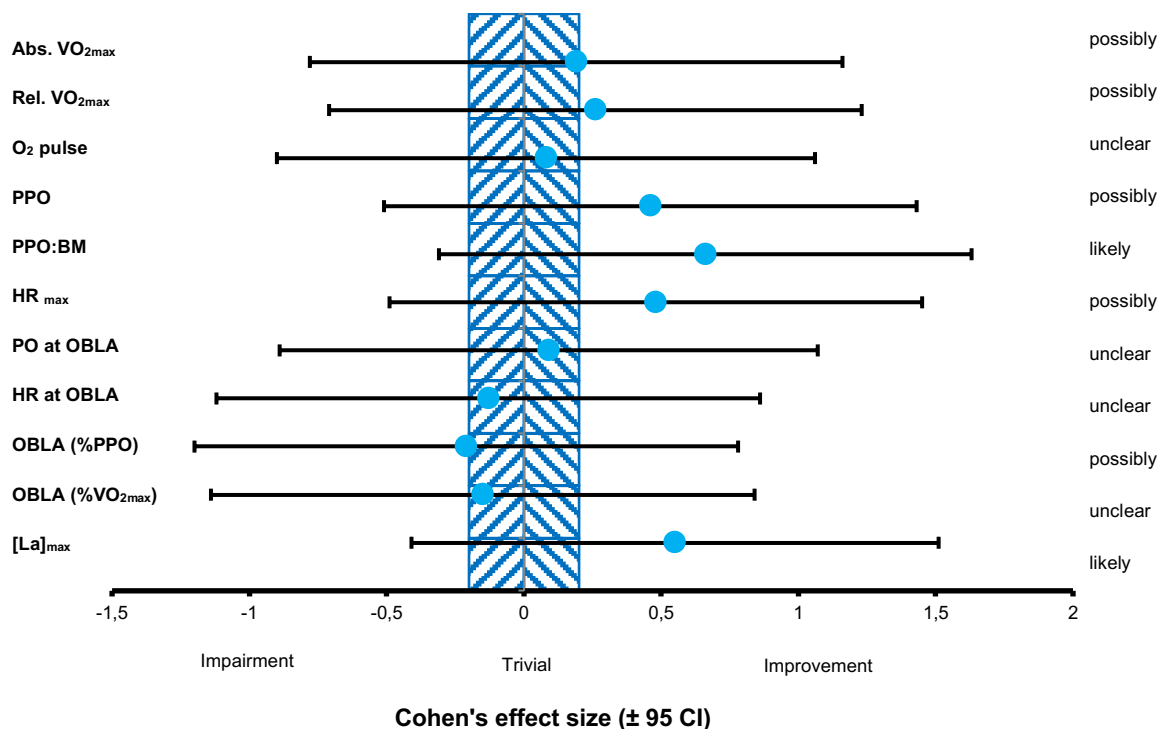
Forest plot showing the effect sizes for the changes in aerobic capacity measures of the SI group. The smallest worthwhile change threshold was set at an effect size greater than 0.2 or smaller than -0.2 shown on the graph with the shaded column. *Abs. VO<sub>2max</sub>*: absolute maximal oxygen uptake in millilitres per minute, *Rel. VO<sub>2max</sub>*: relative maximal oxygen uptake, *O<sub>2</sub> pulse*: Oxygen pulse, *PPO*: peak power output, *PPO:BM*: peak power output per kilogram body mass, *HR<sub>max</sub>*: maximal heart rate, *PO at OBLA*: power output at onset of blood lactate accumulation, *HR at OBLA*: heart rate at onset of blood lactate accumulation, *OBLA (%PPO)*: percentage of peak power output at onset of blood lactate accumulation, *OBLA (%VO<sub>2max</sub>)*: percentage of maximal oxygen uptake at onset of blood lactate, *[La]<sub>max</sub>*: maximal lactate concentration accumulation at cessation of VO<sub>2max</sub> test, *SI*: short interval and *LI*: long interval.

Most of the changes in maximal aerobic capacity in the LI training group were negligible or small (Table 5.3b). The exceptions were, PPO:BM (+4.9%; ES = 0.66, likely improved).

**Table 5.3b: Within-group changes (pre- to post-intervention) in maximal exercise capacity for the long interval HIT group (mean  $\pm$  SD).**

		Long Interval				
	Pre	Post	ES (95% CI)	Qualitative outcome	% chances of better/trivial/poorer effect	
VO <sub>2max</sub> (ml·min <sup>-1</sup> )	3906.9 $\pm$ 594.10	4011.4 $\pm$ 529.66	0.19 (-0.81; 1.16)	Negligible	49/28/23	
VO <sub>2max</sub> (ml·min <sup>-1</sup> ·kg <sup>-1</sup> )	50.1 $\pm$ 5.09	51.3 $\pm$ 4.16	0.26 (-0.74; 1.23)	Small	55/26/19	
O <sub>2</sub> pulse (ml·min <sup>-1</sup> ·bpm <sup>-1</sup> )	20.8 $\pm$ 3.40	21.1 $\pm$ 3.20	0.08 (-0.90; 1.06)	Negligible	41/30/29	
PPO (w)	315.3 $\pm$ 34.35	330.0 $\pm$ 29.16	0.46 (-0.56; 1.43)	Small	70/20/11	
PPO:BM (w·kg <sup>-1</sup> )	4.1 $\pm$ 0.15	4.3 $\pm$ 0.40	0.66 (-0.38; 1.63)	Moderate	82/12/6	
HR <sub>max</sub> (bpm)	188.1 $\pm$ 5.91	190.8 $\pm$ 5.28	0.48 (-0.54; 1.45)	Small	71/19/10	
PO at OBLA (w)	233.6 $\pm$ 46.19	238.0 $\pm$ 48.08	0.09 (-0.89; 1.07)	Negligible	42/30/28	
HR at OBLA (bpm)	164.5 $\pm$ 10.85	163.1 $\pm$ 10.78	-0.13 (-1.10; 0.86)	Negligible	26/29/44	
OBLA (%PPO)	73.9 $\pm$ 10.55	71.7 $\pm$ 10.30	-0.21 (-1.18; 0.78)	Small	21/28/51	
OBLA (%VO <sub>2Max</sub> )	80.7 $\pm$ 8.23	79.2 $\pm$ 11.39	-0.15 (-1.12; 0.84)	Negligible	25/29/46	
[La] <sub>max</sub> (mmol·L <sup>-1</sup> )	16.1 $\pm$ 4.87	18.4 $\pm$ 3.45	0.55 (-0.48; 1.51)	Small	75/17/8	

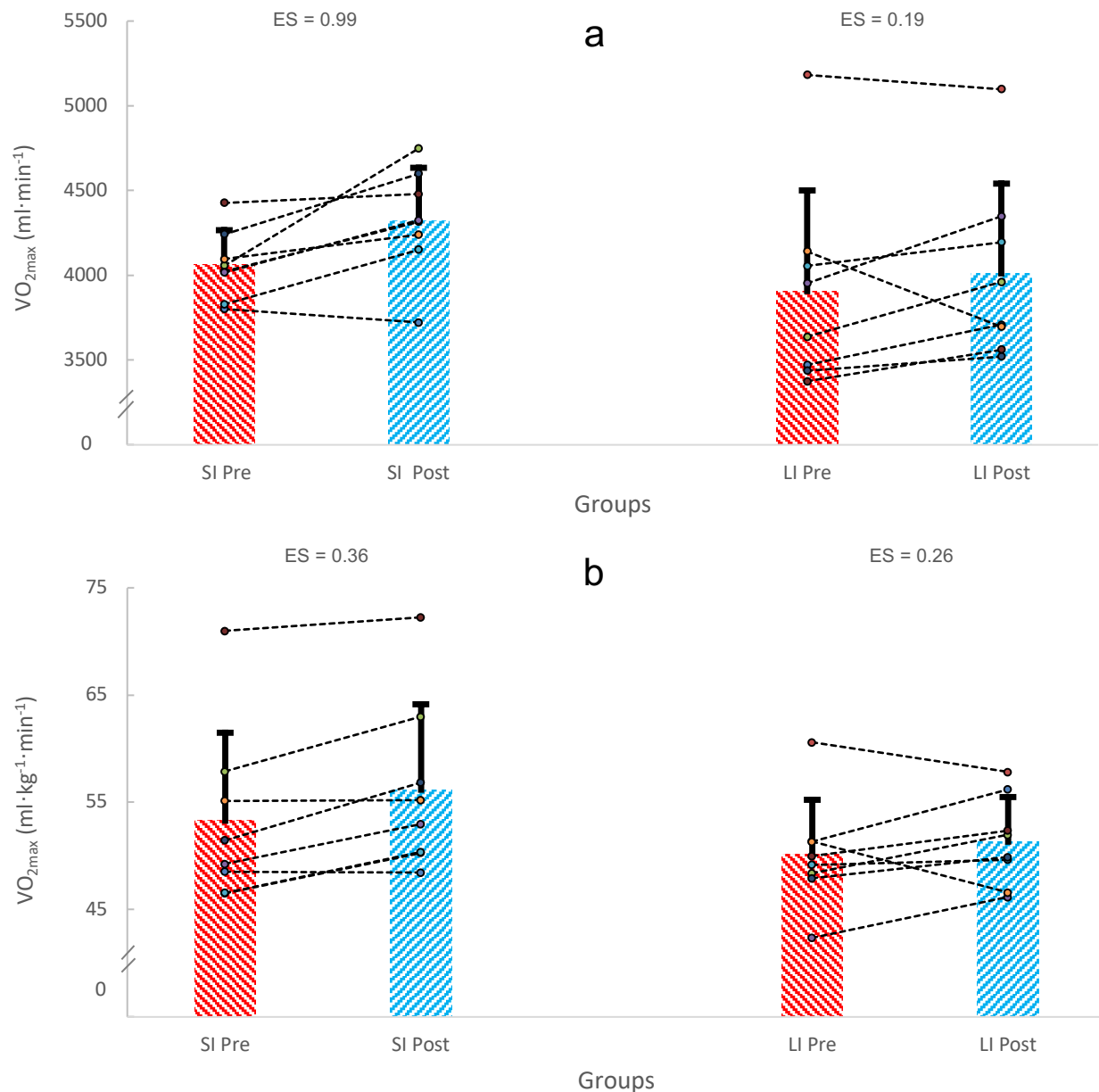
*Abs. VO<sub>2max</sub>*: absolute maximal oxygen uptake in millilitres per minute, *Rel. VO<sub>2max</sub>*: relative maximal oxygen uptake, *O<sub>2</sub> pulse*: Oxygen pulse, *PPO*: peak power output, *PPO:BW*: peak power output per kilogram body mass, *HR<sub>max</sub>*: maximal heart rate, *PO at OBLA*: power output at onset of blood lactate accumulation, *HR at OBLA*: heart rate at onset of blood accumulation, *OBLA (%PPO)*: percentage of peak power output at onset of blood lactate accumulation, *OBLA (%VO<sub>2max</sub>)*: percentage of maximal oxygen uptake at onset of blood lactate accumulation and *[La]<sub>max</sub>*: maximal lactate concentration.



**Figure 5.3**

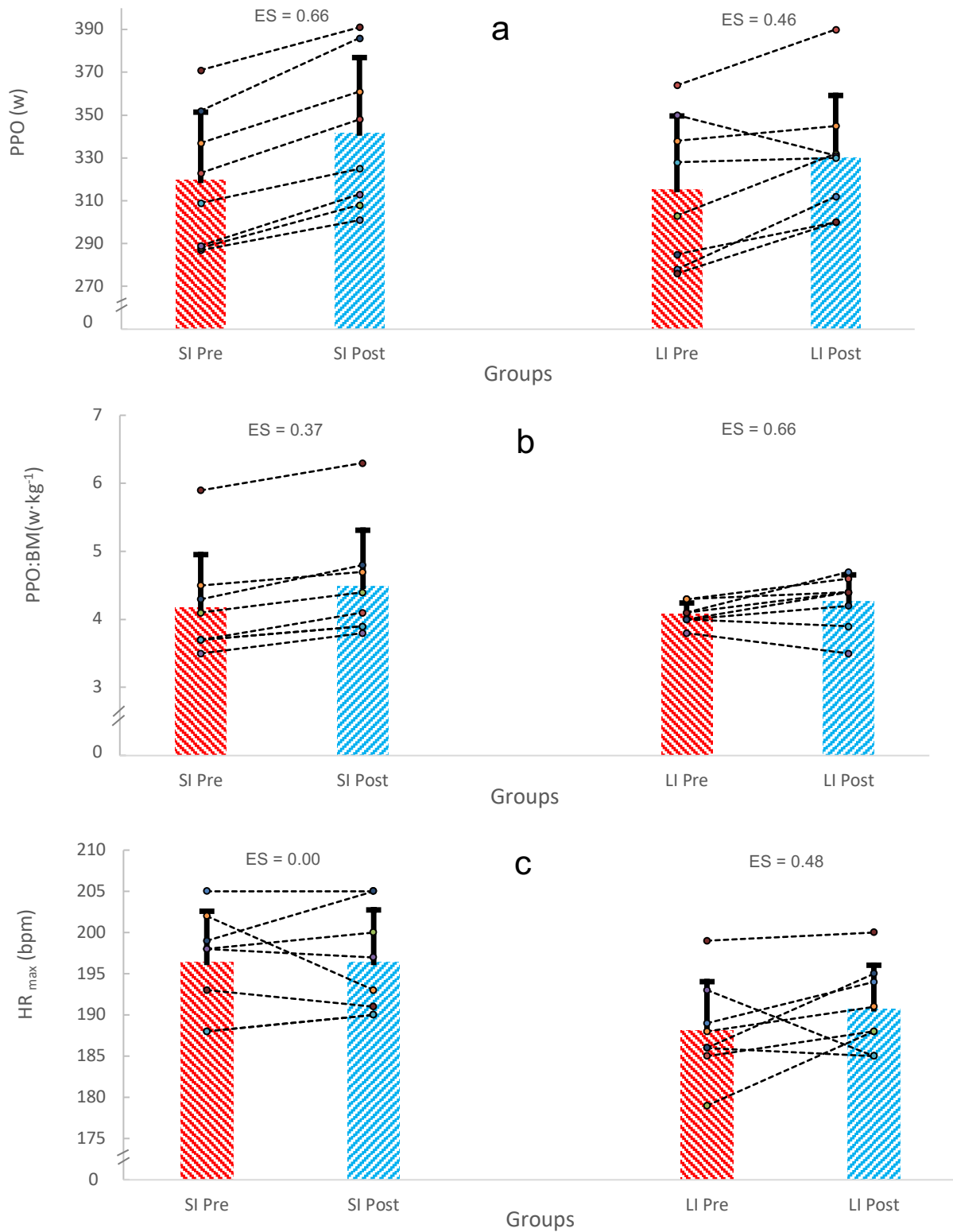
Forest plot showing the effect sizes for the changes in aerobic capacity measures of the LI group. The smallest worthwhile change threshold was set at an effect size greater than 0.2 or smaller than -0.2 shown on the graph with the shaded column. *Abs. VO<sub>2max</sub>*: absolute maximal oxygen uptake in millilitres per minute, *Rel. VO<sub>2max</sub>*: relative maximal oxygen uptake, *O<sub>2</sub> pulse*: Oxygen pulse, *PPO*: peak power output, *PPO:BM*: peak power output per kilogram body mass, *HR<sub>max</sub>*: maximal heart rate, *PO at OBLA*: power output at onset of blood lactate accumulation, *HR at OBLA*: heart rate at onset of blood accumulation, *OBLA (%PPO)*: percentage of peak power output at onset of blood lactate accumulation, *OBLA (%VO<sub>2max</sub>)*: percentage of maximal oxygen uptake at onset of blood lactate, *[La]<sub>max</sub>*: maximal lactate concentration accumulation at cessation of VO<sub>2max</sub> test, *SI*: short interval and *LI*: long interval.

Figures 5.4, 5.5 and 5.6 illustrate the within-group changes in the various maximal exercise capacity parameters, as well as the individual responses. Figure 5.4 shows that only one cyclist in the SI group and two cyclists in the LI training group did not improve their absolute  $VO_{2max}$ . All cyclists in the SI training group improved their PPO, while only one participant in the LI training group did not improve his PPO (Fig. 5.5a). Five cyclists in the SI training group and six in the LI group achieved higher  $HR_{max}$  values during post-testing (Fig. 5.5c).

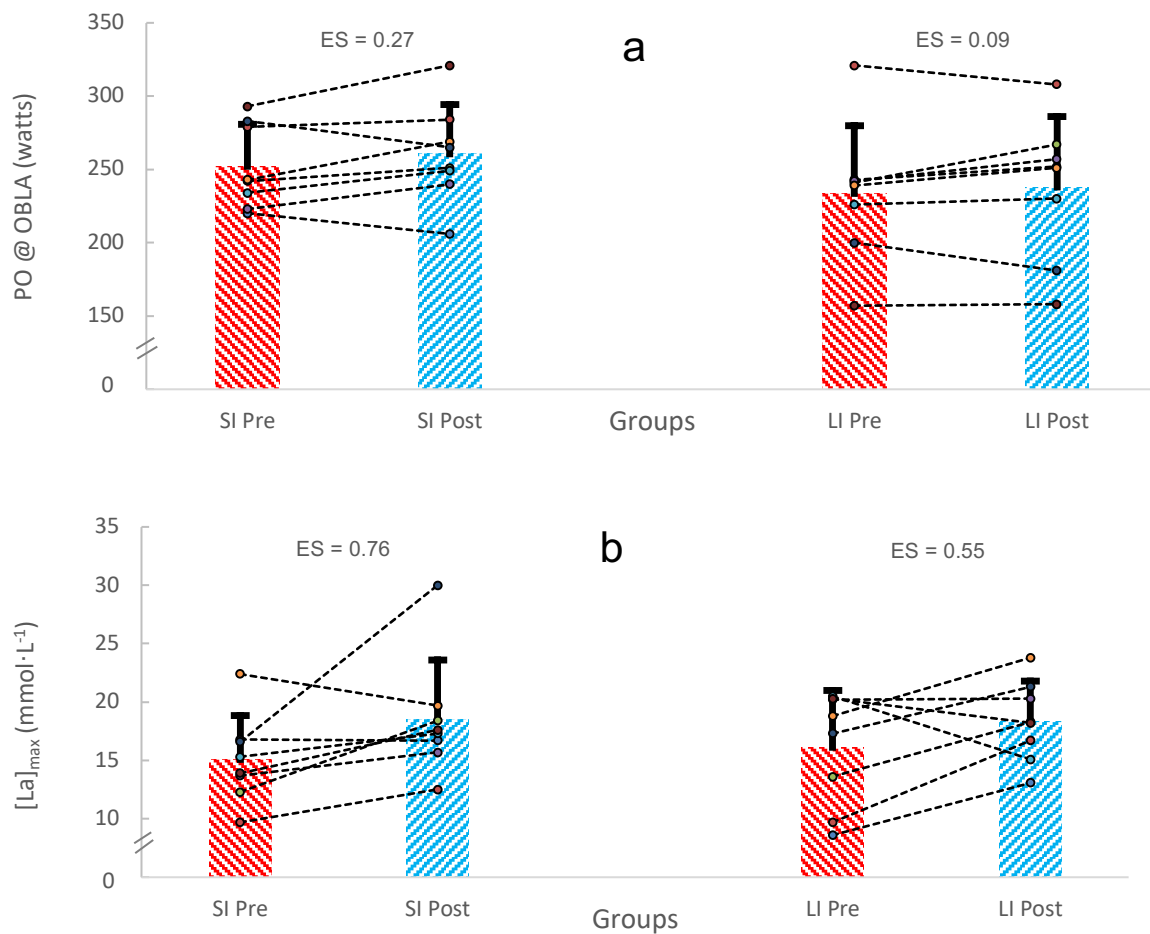


**Figure 5.4**

Within-group changes in (a) absolute  $VO_{2max}$  and (b) relative  $VO_{2max}$  for the short interval HIT and long interval HIT groups over 12 interval sessions. The columns denote group means  $\pm$  SD and lines indicate the responses of the individuals.  $VO_{2max}$ : maximal oxygen uptake, ml: millilitre, min: minute, kg: kilogram, SI: short interval training and LI: long interval training.



**Figure 5.5** Within-group changes in (a) PPO, (b) PPO: BM and (c) HR<sub>max</sub> for the short interval HIT and long interval HIT groups over 12 interval sessions. The columns denote group means ± SD and lines indicate the responses of the individuals. VO<sub>2max</sub>: maximal volume of oxygen uptake, w: watts, ml: millilitre, min: minute, kg: kilogram, SI: short interval and LI: long interval.

**Figure 5.6**

Within-group changes in (a) PO @ OBLA and (b) Peak [La] for the short interval HIT and long interval HIT groups over 12 interval sessions. The columns denote group means  $\pm$  SD and lines indicate the responses of the individuals. PO @ OBLA power output at the onset of blood lactate accumulation, [La]<sub>max</sub> maximal lactate concentration at cessation of VO<sub>2max</sub> test, SI short interval training and LI long interval training.

### 5.3.2 Between-group changes

Table 5.3c depicts the between-group differences in maximal aerobic capacity. None of the changes in the parameters following training were statistically significantly different between the groups ( $P > 0.05$ ). Nevertheless, the SI group showed moderately larger increases in absolute and relative VO<sub>2max</sub> (ES = 0.63 and 0.60, respectively) and small improvement in peak power output (ES = 0.56) over the LI group. There were only small differences between the groups in the OBLA variables, while the increase in maximal blood [La] were possibly larger in the SI group than the LI group.

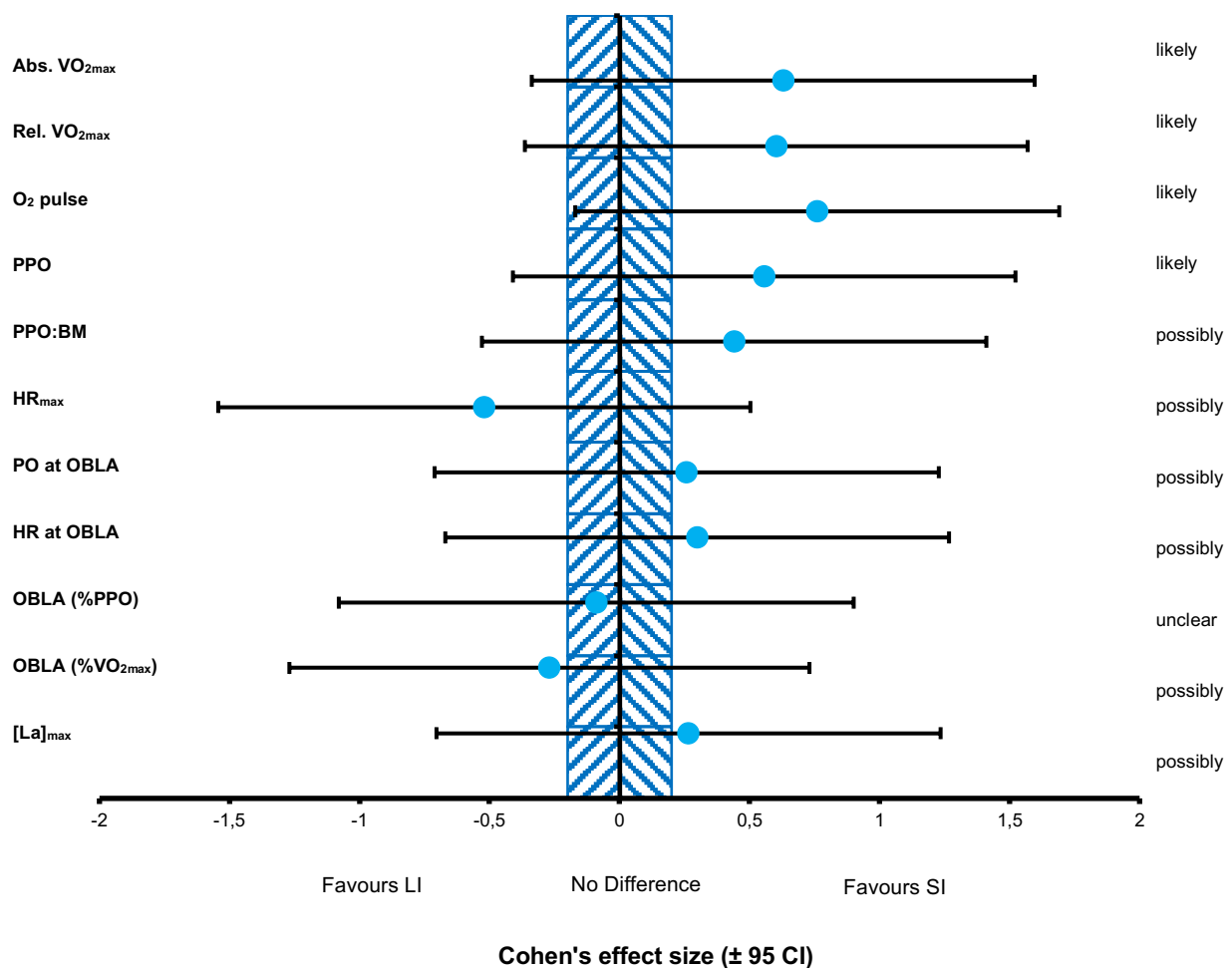
**Table 5.3c: Between-group comparisons of the changes in maximal exercise capacity in response to the training intervention (mean  $\pm$  SD).**

Differences between groups					
	SI mean difference	LI mean difference	ES (95% CI)	Qualitative outcome	% chances of better/trivial/poorer effect
$VO_{2max}$ (ml·min <sup>-1</sup> )	261.4 $\pm$ 231.48	104.5 $\pm$ 266.48	0.63 (-0.41; 1.60)	Moderate	80/14/6
$VO_{2max}$ (ml·min <sup>-1</sup> ·kg <sup>-1</sup> )	2.9 $\pm$ 2.16	1.2 $\pm$ 3.36	0.60 (-0.43; 1.57)	Moderate	78/15/7
$O_2$ pulse (ml·min <sup>-1</sup> ·bpm <sup>-1</sup> )	1.3 $\pm$ 1.04	0.3 $\pm$ 1.66	0.76 (-0.33; 1.69)	Moderate	84/11/4
PPO (w)	22.1 $\pm$ 6.20	14.8 $\pm$ 17.52	0.56 (-0.47; 1.52)	Small	76/17/8
PPO:BM (w·kg <sup>-1</sup> )	0.3 $\pm$ 0.11	0.2 $\pm$ 0.30	0.44 (-0.57; 1.41)	Small	68/21/11
$HR_{max}$ (bpm)	0.0 $\pm$ 4.38	2.6 $\pm$ 5.55	-0.52 (-1.49; 0.50)	Small	9/18/73
PO at OBLA (w)	8.5 $\pm$ 17.00	4.4 $\pm$ 14.74	0.26 (-0.74; 1.23)	Small	55/27/19
HR at OBLA (bpm)	1.1 $\pm$ 8.04	-1.4 $\pm$ 8.73	0.30 (-0.70; 1.27)	Small	58/26/17
OBLA (%PPO)	-2.7 $\pm$ 5.27	-2.2 $\pm$ 6.35	-0.09 (-1.06; 0.90)	Negligible	29/30/41
OBLA (% $VO_{2Max}$ )	-3.4 $\pm$ 7.90	-1.4 $\pm$ 6.85	-0.27 (-1.24; 0.73)	Small	18/26/56
$[La]_{max}$ (mmol·L <sup>-1</sup> )	3.4 $\pm$ 4.80	2.2 $\pm$ 4.25	0.26 (-0.73; 1.23)	Small	55/26/18

*Abs.*  $VO_{2max}$  absolute maximal oxygen uptake in millilitres per minute, *Rel.*  $VO_{2max}$  relative maximal oxygen uptake, *O<sub>2</sub> pulse*: Oxygen pulse, *PPO* peak power output, *PPO:BM* peak power output per kilogram body mass, *HR<sub>max</sub>* maximal heart rate, *PO at OBLA* power output at onset of blood lactate accumulation, *HR at OBLA* heart rate at onset of blood accumulation, *OBLA (%PPO)* percentage of peak power output at onset of blood lactate accumulation, *OBLA (% $VO_{2max}$ )* percentage of maximal oxygen uptake at onset of blood lactate accumulation, *[La]<sub>max</sub>* maximal lactate concentration.



Figure 5.7 summarizes the effect sizes ( $\pm$  95% CI) for the between-group differences of eleven aerobic metabolic markers measured. Eight of the eleven in performance markers favoured improvements by the SI training group over the LI training group. The group\*time analysis indicated that there was no statistically significant interaction effect ( $P > 0.05$ ) in any of the measured aerobic performance variables.



**Figure 5.7**

Forest plot showing the effect sizes for the between-group mean differences in aerobic capacity measures. A positive effect size favours the short interval intervention and a negative effect size favours the long interval intervention. The smallest worthwhile change threshold was set at an effect size greater than 0.2 or smaller than -0.2 shown on the graph with the shaded column. Abs.  $VO_{2max}$ : absolute maximal oxygen uptake in millilitres per minute, Rel.  $VO_{2max}$ : relative maximal oxygen uptake,  $O_2$  pulse: Oxygen pulse PPO: peak power output, PPO:BM: peak power output per kilogram body mass,  $HR_{max}$ : maximal heart rate, PO at OBLA: power output at onset of blood lactate accumulation, HR at OBLA: heart rate at onset of blood accumulation, OBLA (%PPO) : percentage of peak power output at onset of blood lactate accumulation, OBLA (% $VO_{2max}$ ): percentage of maximal oxygen uptake at onset of blood lactate,  $[La]_{max}$ : maximal lactate concentration accumulation at cessation of  $VO_{2max}$  test, SI: short interval and LI: long interval.

## 5.4. Maximal anaerobic capacity

The Wingate Anaerobic Test was used to assess the participants' anaerobic capacity before and after the intervention. Two consecutive 30 s Wingate tests were performed, with a four min recovery period in between. The power output and blood [La] measures were used to assess anaerobic changes.

### 5.4.1 Anaerobic Power

Power output measurements of the peak and 30 s mean power for the first and second Wingate were recorded. These values were used to calculate the power of reproducibility for each cyclist as a percentage. A value of 100% means a cyclist was able to perfectly replicate his performance in the two Wingate tests.

#### 5.4.1.1 Within-group changes

Table 5.4a (SI training group) and table 5.4b (LI training group) present the within-group changes in anaerobic capacity from pre- to post-intervention. None of the variables changed statistically significantly ( $P > 0.05$ ) in either group. No statistically significant interaction effects ( $P > 0.05$ ) were revealed for group\*time (pre to post), group\*repeat (W1 and W2) or repeat\*time\*group.

The SI group showed a moderate improvement in their ability to replicate their performance in the two Wingate tests ( $PR\%_{max}$ : + 2.0%;  $ES = 1.09$ ;  $PR\%_{mean}$ : +1.6%;  $ES = 0.90$ ) during post-testing, while improvement in the LI group was small ( $PR\%_{max}$ : +1,2%;  $ES = 0.56$ ;  $PR\%_{mean}$ : +0.7%;  $ES = 0.31$ ). Although the SI group did not improve their mean or maximum power output for W1, there were small improvements in both mean and maximum power output for W2. Although the changes were small, the LI group made improvement in both mean and maximum power output for both Wingate tests.

**Table 5.4a: Within-group changes (pre- to post-intervention) in anaerobic capacity for the short interval HIT programme (mean  $\pm$  SD).**

Short Interval					
	Pre	Post	ES (95% CI)	Qualitative outcome	% chances of better/trivial/poorer effect
$W1_{max}$ (w)	848.1 $\pm$ 100.57	849.4 $\pm$ 90.19	0.01 (-0.97; 0.99)	Negligible	36/30/34
$W2_{max}$ (w)	791.4 $\pm$ 98.03	833.0 $\pm$ 87.38	0.45 (-0.57; 1.41)	Small	69/20/11
$W1_{mean}$ (w)	746.0 $\pm$ 68.68	746.9 $\pm$ 65.78	0.01 (-0.97; 0.99)	Negligible	36/30/34
$W2_{mean}$ (w)	675.5 $\pm$ 62.71	698.1 $\pm$ 50.43	0.40 (-0.61; 1.36)	Small	65/22/13
$PR\%_{max}$	96.7 $\pm$ 2.29	98.6 $\pm$ 0.88	1.09 (-0.01; 2.08)	Moderate	96/3/1
$PR\%_{mean}$	95.3 $\pm$ 1.90	96.8 $\pm$ 1.40	0.90 (-0.17; 1.87)	Moderate	91/6/2

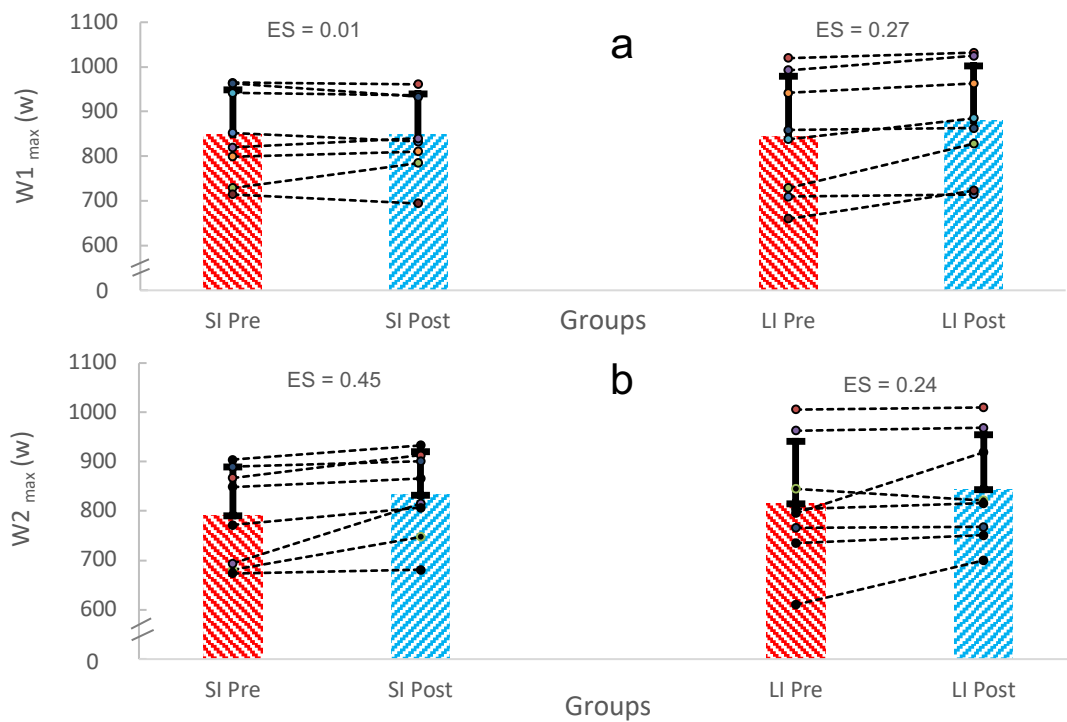
Power output measured in Watts. *SI*: short interval,  $W1_{max}$ : maximum power output for first Wingate test,  $W2_{max}$ : maximum power output for second Wingate test,  $W1_{mean}$ : mean power output for first Wingate test,  $W2_{mean}$ : mean power output for second Wingate test,  $PR\%_{max}$ : maximum power output percentage of reproducibility and  $PR\%_{mean}$ : mean power output percentage of reproducibility.

**Table 5.4b: Within-group changes (pre- to post-intervention) in anaerobic capacity for the long interval HIT programme (mean  $\pm$  SD).**

Long Interval					
	Pre	Post	ES (95% CI)	Qualitative outcome	% chances of better/trivial/poorer effect
$W1_{max}$ (w)	843.9 $\pm$ 135.27	879.3 $\pm$ 123.00	0.27 (-0.73; 1.24)	Small	56/26/18
$W2_{max}$ (w)	815.6 $\pm$ 125.69	844.4 $\pm$ 110.35	0.24 (-0.75; 1.21)	Small	53/27/20
$W1_{mean}$ (w)	747.1 $\pm$ 103.68	773.3 $\pm$ 84.33	0.28 (-0.72; 1.25)	Small	56/26/18
$W2_{mean}$ (w)	700.9 $\pm$ 93.27	718.1 $\pm$ 72.31	0.21 (-0.79; 1.18)	Small	51/28/22
$PR\%_{max}$	96.3 $\pm$ 2.67	97.5 $\pm$ 1.63	0.56 (-0.47; 1.53)	Small	76/16/8
$PR\%_{mean}$	95.8 $\pm$ 2.28	96.5 $\pm$ 2.05	0.31 (-0.69; 1.28)	Small	58/25/17

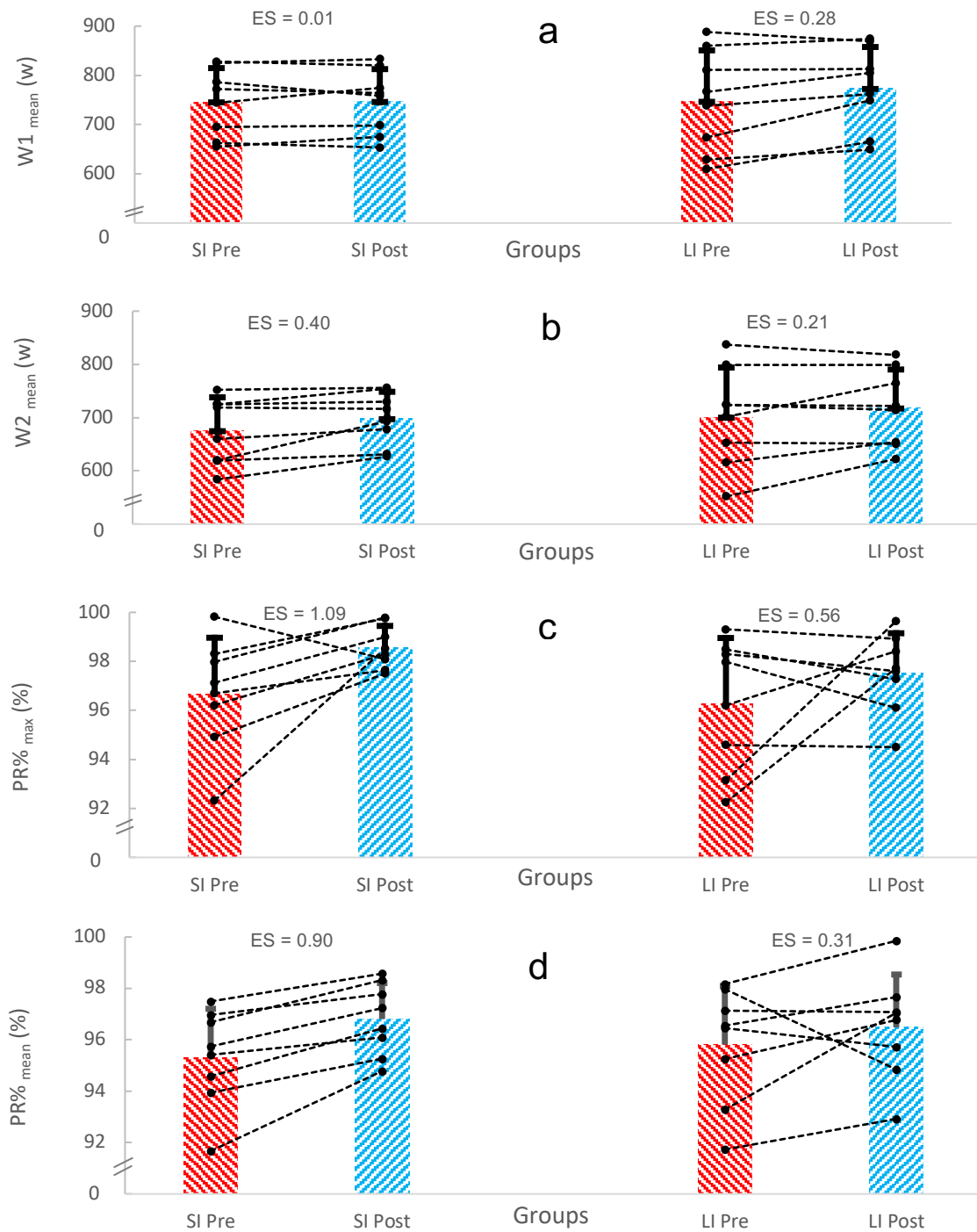
Power measured in Watts. *LI*: long interval group,  $W1_{max}$ : maximum power output for first Wingate test,  $W2_{max}$ : maximum power output for second Wingate test,  $W1_{mean}$ : mean power output for first Wingate test,  $W2_{mean}$ : mean power output for second Wingate test,  $PR\%_{max}$ : maximum power output percentage of reproducibility and  $PR\%_{mean}$ : mean power output percentage of reproducibility.

Figure 5.8 illustrates the within-group changes in maximum power output during the first and second Wingate performances, as well as the individual responses. Three participants in the SI group performed worse in  $W1$  (Fig. 5.6a), but only one performed worse in  $W2$  (Fig. 5.6b) during follow-up testing. In the LI group there was only one participant who did not perform better in the two Wingate tests after the intervention (Fig. 5.6a and b).



**Figure 5.8** Within-group changes in (a)  $W1_{max}$ : first Wingate peak power and (b)  $W2_{max}$ : second Wingate peak power for the SI group and LI group over 12 interval sessions. The columns denote group means  $\pm$  SD and lines indicate the responses of the individuals. w: watts, SI: short interval and LI: long interval.

Figure 5.9 depicts the within-group changes for anaerobic capacity and the individual responses. Seven participants in the SI group improved their ability to reproduce their max PO during post-testing, while the whole group improved their mean PO reproducibility. Three participants in the LI group performed worse during post-testing in terms of max PO reproducibility and two in terms of mean PO reproducibility.



**Figure 5.9**

Within - group changes in (a)  $W1_{mean}$ : first Wingate mean power, (b)  $W2_{mean}$ : second Wingate mean power output, (c)  $PR\%_{max}$ : percentage reproducibility in maximum power and (d)  $PR\%_{mean}$ : percentage of reproducibility in mean power output for the SI and LI training groups over 12 interval sessions. The columns denote group means  $\pm$  SD and lines indicate the responses of the individuals. SI: short interval and LI: long interval.

### 5.4.1.2 Between-group changes

Table 5.4c depicts the between-group differences for anaerobic capacity. The improvement in W1 maximum PO was statistically significantly greater in the LI group than the SI group ( $P = 0.04$ ). However, there were no other statistically significant differences between the groups in response to the interventions ( $P > 0.05$ ).

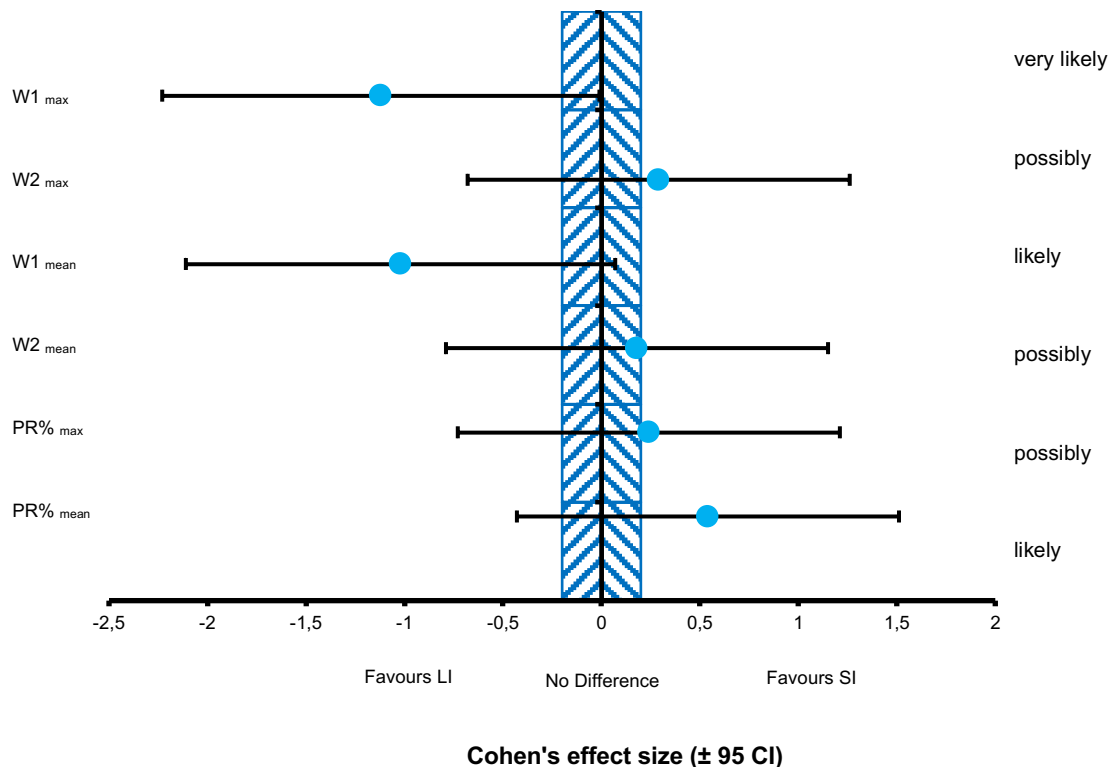
The chances that the LI group improved their performance more than the SI group during W1 is very likely, while the difference in performance was less pronounced during W2. Small to moderately better performances were observed for the SI group in terms of the reproducibility of W1 and W2 compared with the LI group.

**Table 5.4c: Between-group comparisons (pre- to post-intervention) in anaerobic power capacity (mean  $\pm$  SD).**

	Differences between groups				
	SI mean difference	LI mean difference	ES (95% CI)	Qualitative outcome	% chances of better/trivial/poorer effect
W1 <sub>max</sub> (w)	1.3 $\pm$ 27.70	33.0 $\pm$ 35.38	-1.12 (-2.11; -0.01)	Moderate	1/3/96
W2 <sub>max</sub> (w)	41.6 $\pm$ 37.63	28.8 $\pm$ 50.14	0.29 (-0.71; 1.26)	Small	57/26/17
W1 <sub>mean</sub> (w)	0.9 $\pm$ 18.43	26.1 $\pm$ 29.69	-1.02 (-2.00; 0.07)	Moderate	1/5/94
W2 <sub>mean</sub> (w)	22.6 $\pm$ 25.19	17.3 $\pm$ 34.64	0.18 (-0.81; 1.15)	Negligible	48/29/23
PR% <sub>max</sub>	1.9 $\pm$ 2.18	1.2 $\pm$ 3.17	0.24 (-0.75; 1.21)	Small	53/27/20
PR% <sub>mean</sub>	1.5 $\pm$ 0.76	0.7 $\pm$ 2.03	0.54 (-0.48; 1.51)	Small	75/17/8

SI: short interval, LI: long interval, W1<sub>max</sub>: maximum power output for first Wingate test, W2<sub>max</sub>: maximum power output for second Wingate test, W1<sub>mean</sub>: mean power output for first Wingate test, W2<sub>mean</sub>: mean power output for second Wingate test, PR%<sub>max</sub>: maximum power output percentage of reproducibility, PR%<sub>mean</sub>: mean power output percentage of reproducibility. Power measured in Watts.

Figure 5.10 summarizes the effect sizes for the between-group comparisons in anaerobic capacity measures. In three of the six variables, greater improvements were observed in the SI group compared with the LI group.



**Figure 5.10** Forest plot showing the effect sizes for the between-group mean differences in anaerobic capacity measures. A positive effect size favours the short interval intervention and a negative effect size favours the long interval intervention. The smallest worthwhile change threshold was set at an effect size greater than 0.2 or smaller than -0.2 shown on the graph with the shaded column. W1<sub>max</sub>: first Wingate peak power, W2<sub>max</sub>: second Wingate peak power, W1<sub>mean</sub>: first Wingate mean power, W2<sub>mean</sub>: second Wingate mean power output, PR %<sub>max</sub>: percentage of reproducibility for peak power, PR%<sub>mean</sub>: percentage of reproducibility for mean power.

## 5.4.2 Blood lactate concentration profiles

Blood lactate readings were taken three min after the first and second Wingate test, with two additional readings at six and nine min after the second Wingate to depict blood lactate recovery after supra-maximal exercise.

### 5.4.2.1 Within-group changes

Within-group changes in blood lactate concentrations are summarized in Table 5.5a (SI training group) and Table 5.5b (LI training group). The group\*time (pre to post), group\*repeat (Wingate 1 to Wingate 2) and repeat\*time\*group analysis showed no statistically significant interactions for the

blood [La] measures. Although the SI group had a statistically significant lower pre-training [La], there were no other statistically significant changes in either group.

The SI group showed a large practical reduction in resting lactate levels (-36.8%; ES = 1.29), but mainly negligible changes in the remaining measures. Similarly, the LI group demonstrated a small reduction in resting blood lactate concentration (-22.2%; ES = 0.55), but small to negligible changes in [La] during recovery.

**Table 5.5a: Within-group changes (pre- to post-testing) in the blood lactate profiles of the short interval HIT programme (mean  $\pm$  SD).**

Short Interval					
	Pre	Post	ES (95% CI)	Qualitative outcome	% chances of better/trivial/poorer effect
Pre [La]	1.9 $\pm$ 0.77	1.2 $\pm$ 0.24	-1.29 (-2.28; -0.15)	Large	1/2/98
W1 [La]	15.7 $\pm$ 2.56	16.3 $\pm$ 2.58	0.22 (-0.78; 1.19)	Small	51/27/21
W2 [La]	19.7 $\pm$ 3.08	20.3 $\pm$ 4.38	0.15 (-0.84; 1.12)	Negligible	46/29/25
R1 [La]	18.8 $\pm$ 2.46	18.7 $\pm$ 2.65	-0.04 (-1.02; 0.94)	Negligible	32/30/38
R2 [La]	17.0 $\pm$ 3.80	17.5 $\pm$ 3.89	0.14 (-0.85; 1.11)	Negligible	45/29/26

*Pre [La]* lactate concentration before Wingate test, *W1 [La]* lactate concentration three min after the first Wingate, *W2 [La]* lactate concentration three min after the second Wingate, *R1 [La]* lactate concentration six min after the second Wingate, *R2 [La]* lactate concentration nine min after the second Wingate. Blood lactate concentrations in mmol·L<sup>-1</sup>

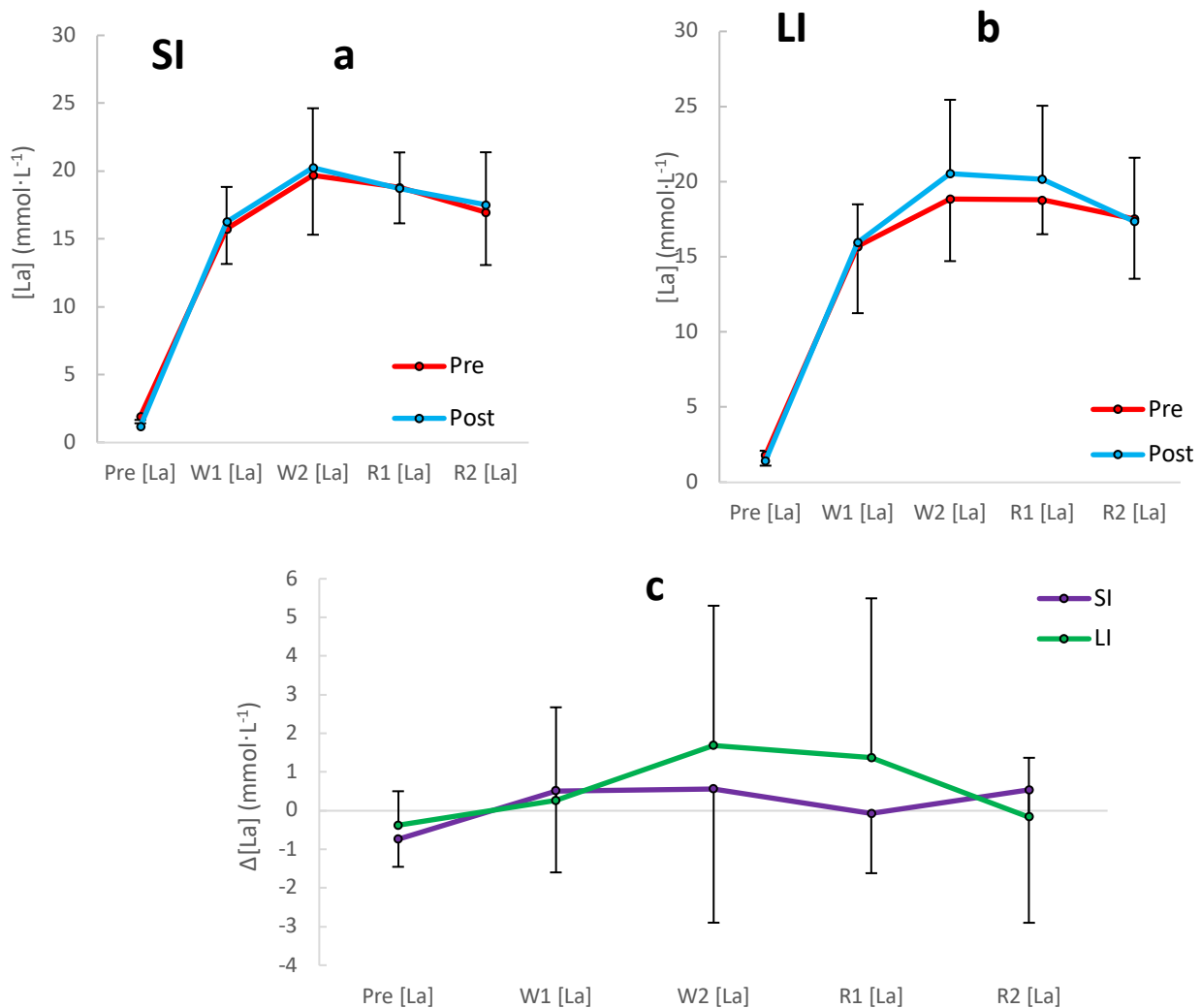
**Table 5.5b: Within-group changes (pre- to post-intervention) in the blood lactate profiles of the long interval HIT programme (mean  $\pm$  SD).**

Long Interval					
	Pre	Post	ES (95% CI)	Qualitative outcome	% chances of better/trivial/poorer effect
Pre [La]	1.8 $\pm$ 0.71	1.5 $\pm$ 0.66	-0.55 (-1.51; 0.48)	Small	8/16/75
W1 [La]	15.7 $\pm$ 4.45	16.0 $\pm$ 2.53	0.07 (-0.91; 1.05)	Negligible	40/30/30
W2 [La]	18.9 $\pm$ 4.13	20.5 $\pm$ 4.91	0.37 (-0.64; 1.34)	Small	63/23/14
R1 [La]	18.8 $\pm$ 2.28	20.2 $\pm$ 4.89	0.36 (-0.65; 1.33)	Small	62/23/14
R2 [La]	17.5 $\pm$ 3.98	17.4 $\pm$ 4.23	-0.04 (-1.02; -0.94)	Negligible	32/30/38

*Pre [La]* lactate concentration before Wingate test, *W1 [La]* lactate concentration three min after the first Wingate, *W2 [La]* lactate concentration three min after the second Wingate, *R1 [La]* lactate concentration six min after the second Wingate, *R2 [La]* lactate concentration nine min after the second Wingate. Blood lactate concentrations in mmol·L<sup>-1</sup>



Fig. 5.11a and b depicts the likely meaningful reduction in resting [La] in both groups following the training interventions. Fig. 5.11c shows the small to negligible changes in blood [La] during recovery for the two groups.



**Figure 5.11** Blood [La] during the repeated Wingate test and recovery for (a) the short interval training group, (b) the long interval training group and (c) changes in blood [La] for the SI and LI training group following 12 HIIT sessions. Pre [La]: lactate concentration before Wingate test, W1 [La]: lactate concentration three min after the first Wingate, W2 [La]: lactate concentration three min after the second Wingate, R1 [La]: lactate concentration six min after the second Wingate, R2 [La]: lactate concentration nine min after the second Wingate. Blood lactate concentrations in mmol·L<sup>-1</sup>.

#### 5.4.2.2 Between-group changes

The between-group differences for the changes in blood [La] in response to the interventions are shown in Table 5.5c. There were no statistically significant differences ( $P > 0.05$ ) in the between-group changes from pre to post, however, small to moderate practical differences were observed in three of the five measures. In particular, the SI group was more effective in lowering their resting [La],

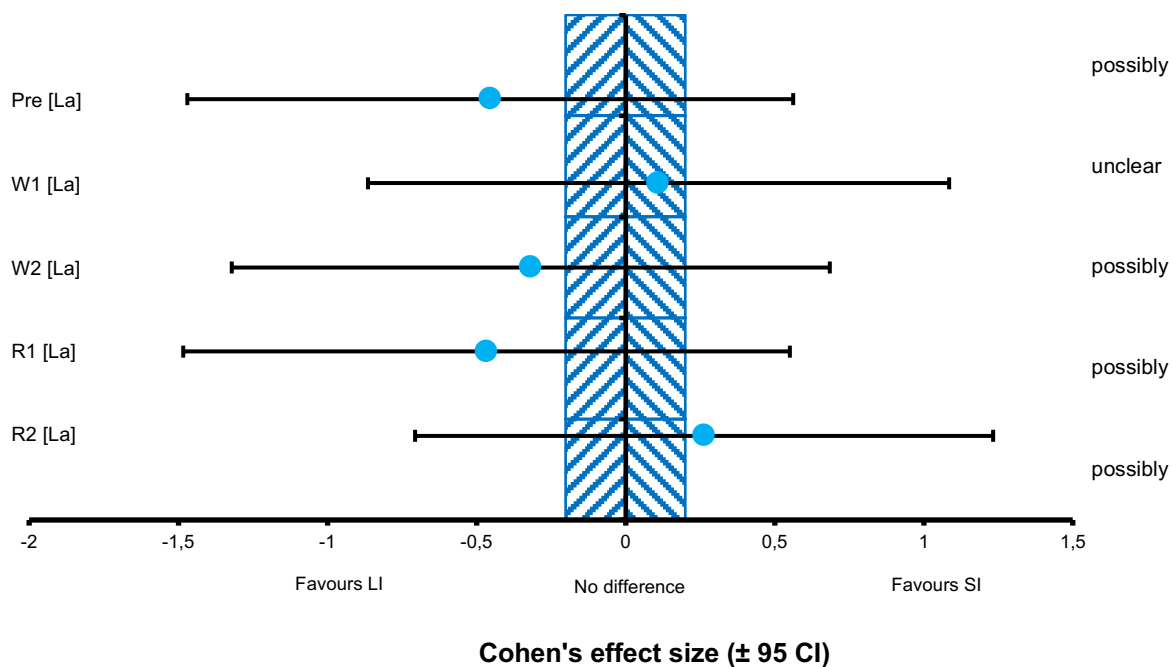
however, the chances for true differences in the recovery profiles between the two groups are possibly small to negligible.

**Table 5.5c: Between-group comparisons (pre- to post-intervention) in blood lactate profile (mean  $\pm$  SD).**

	Differences between groups				% chances of better/trivial/poorer effect
	SI mean difference	LI mean difference	ES (95% CI)	Qualitative outcome	
Pre [La]	-0.7 $\pm$ 0.71	-0.4 $\pm$ 0.88	-0.45 (-1.42; 0.56)	Small	11/20/69
W1 [La]	0.5 $\pm$ 2.11	0.3 $\pm$ 2.41	0.11 (-0.88; 1.08)	Negligible	43/30/27
W2 [La]	0.6 $\pm$ 3.46	1.7 $\pm$ 3.62	-0.32 (-1.29; 0.68)	Small	16/25/59
R1 [La]	-0.1 $\pm$ 1.54	1.4 $\pm$ 4.12	-0.47 (-1.43; 0.55)	Small	10/20/70
R2 [La]	0.5 $\pm$ 3.44	-0.2 $\pm$ 1.53	0.26 (-0.74; 1.23)	Small	55/27/18

*Pre [La]:* lactate concentration before Wingate test, *W1 [La]:* lactate concentration three min after the first Wingate, *W2 [La]:* lactate concentration three min after the second Wingate, *R1 [La]:* lactate concentration six min after the second Wingate, *R2 [La]:* lactate concentration nine min after the second Wingate. Blood lactate concentrations measured in mmol·L<sup>-1</sup>.

Figure 5.12 summarizes the between-group differences in blood lactate profiles in response to the two interventions. The results show that in most cases LI training led to greater practically meaningful changes compared with SI training.



**Figure 5.12**

Forest plot showing the effect sizes for the between-group mean differences in lactate profile measures. A positive effect size favours the short interval intervention and a negative effect size favours the long interval intervention. The smallest worthwhile change threshold was set at an effect size greater than 0.2 or smaller than -0.2 shown on the graph with the shaded column. *Pre [La]:* lactate concentration before Wingate test, *W1 [La]:* lactate concentration three min after the first Wingate, *W2 [La]:* lactate concentration three min after the second Wingate, *R1 [La]:* lactate concentration six min after the second Wingate, *R2 [La]:* lactate concentration nine min after the second Wingate.

### 5.4.3 Correlation between anaerobic changes

Table 5.5d presents the Spearman rank-order correlations between the changes in power output and changes in peak blood [La] from pre- to post-testing in both groups. There were no statistically significant correlations ( $P > 0.05$ ) between any of the anaerobic measures for neither the first, nor the second Wingate test. The chances for true relationships between pairwise comparisons were likely small to large for the SI group, but mostly negligible for the LI group.

**Table 5.5d: Spearman rank-order correlations between anaerobic capacity measures in the short interval and long interval HIT programme.**

Variable		SI		LI	
Wingate $\Delta$ PO	Peak $\Delta$ [La]	$R_s$ (95% CI)	% chance of small likely positive/trivial/negative effect	$R_s$ (95% CI)	% chance of small likely positive/trivial/negative effect
$W1_{max}$ vs	W1	-0.52 (-0.90; 0.29)	7/8/86	0.10 (-0.65; 0.75)	50/17/33
$W1_{mean}$ vs	W1	-0.55 (-0.90; 0.25)	5/7/88	-0.02 (-0.71; 0.69)	39/18/43
$W2_{max}$ vs	W2	-0.22 (-0.80; 0.57)	24/16/60	-0.40 (-0.86; 0.42)	12/11/77
$W2_{mean}$ vs	W2	-0.48 (-0.89; 0.34)	8/9/83	-0.14 (-0.77; 0.63)	50/17/33

No statistically significant correlations between changes in anaerobic measures ( $P > 0.05$ ).  $\Delta W$ : Change of Wingate power,  $W1_{max}$ : peak power of the first Wingate,  $W1_{mean}$ : mean power of the first Wingate,  $W2_{max}$ : peak power of the second Wingate,  $W2_{mean}$ : mean power of the second Wingate,  $\Delta$ [La] change in peak lactate concentration,  $W1$ : first Wingate performance,  $W2$ : second Wingate performance and  $R_s$ : Spearman rank order correlation.

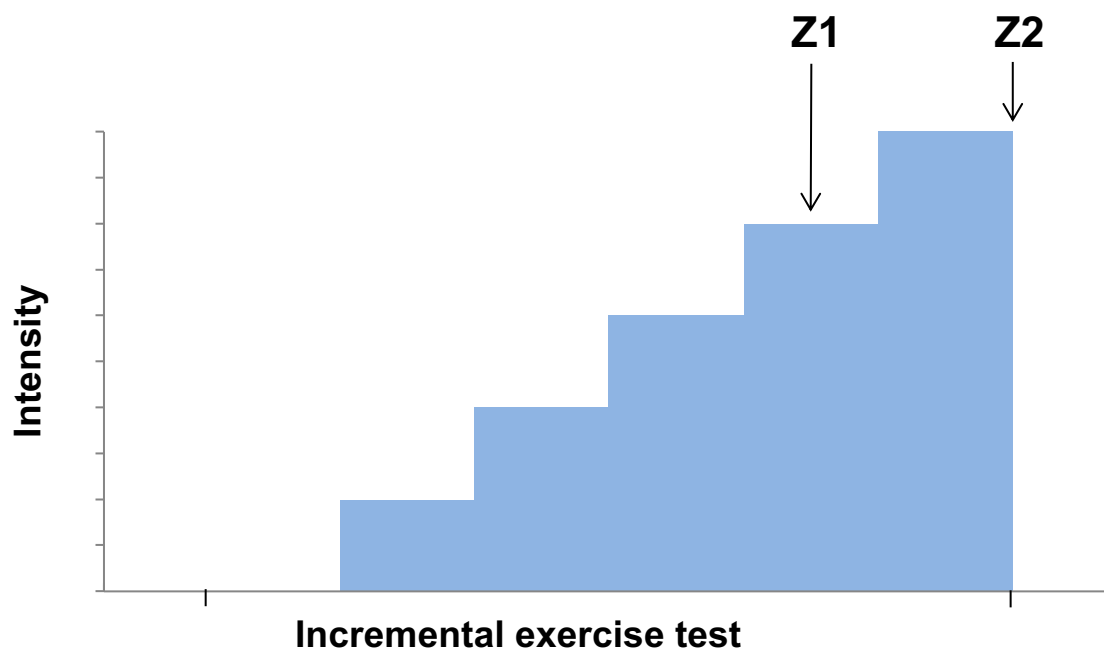
## 5.5. Changes in near infrared spectroscopy (NIRS) measures

The changes in oxyhemoglobin ( $\Delta[\text{O}_2\text{Hb}]$ ) and deoxyhemoglobin ( $\Delta[\text{HHb}]$ ) of the *vastus lateralis* (VL) are reported as means and standard deviations. The results are depicted in Tables 5.6 - 5.7 and Figures 5.14 - 5.17. The figures represent the relative changes to the baseline reading taken before the warm-up phase.

The incremental exercise test was divided into two zones for within-group and between-group comparisons following the HIIT programme (Fig 5.13):

Z1: zone 1 is the 30 s period at the onset of blood lactate accumulation (OBLA).

Z2: zone 2 is the final 30 s of the incremental exercise test (PPO).



**Figure 5.13**

Graphic representation of the incremental exercise test. Z1: zone 1 the 30 s period at the onset of blood lactate accumulation (OBLA) and Z2: zone 2 the final 30 s of the incremental exercise test (PPO).

### 5.5.1 Oxygenated hemoglobin ( $\Delta[\text{O}_2\text{Hb}]$ )

The  $\Delta[\text{O}_2\text{Hb}]$  is an indication of the presence of oxygen in the localized muscle, attached to the hemoglobin complex of the blood. A decrease in the relative change is usually an indication of an increasing workload when  $\text{O}_2$  dissociates from hemoglobin.

#### 5.5.1.1 Within-group changes

The within-group  $\Delta[\text{O}_2\text{Hb}]$  in response to the interventions are presented in Table 5.6a (short interval HIT programme) and Table 5.6b (long interval HIT programme). There were no statistically significant differences in the  $\Delta[\text{O}_2\text{Hb}]$  in either group ( $P > 0.05$ ). The group\*time (pre to post), group\*repeat (zones 1 and 2) and the repeat\*time\*group interactions were not statistically significant ( $P > 0.05$ ) (Fig. 5.12).

There were small practically meaningful increases in relative  $\Delta[\text{O}_2\text{Hb}]$  during submaximal and peak exercise in the SI group (Table 5.6a), while the changes in the LI group were negligible ( $ES < 0.20$ ).

**Table 5.6a: Within-group changes (pre- to post- intervention) in  $\Delta[\text{O}_2\text{Hb}]$  for the short interval HIT programme (mean  $\pm$  SD) during the incremental exercise test.**

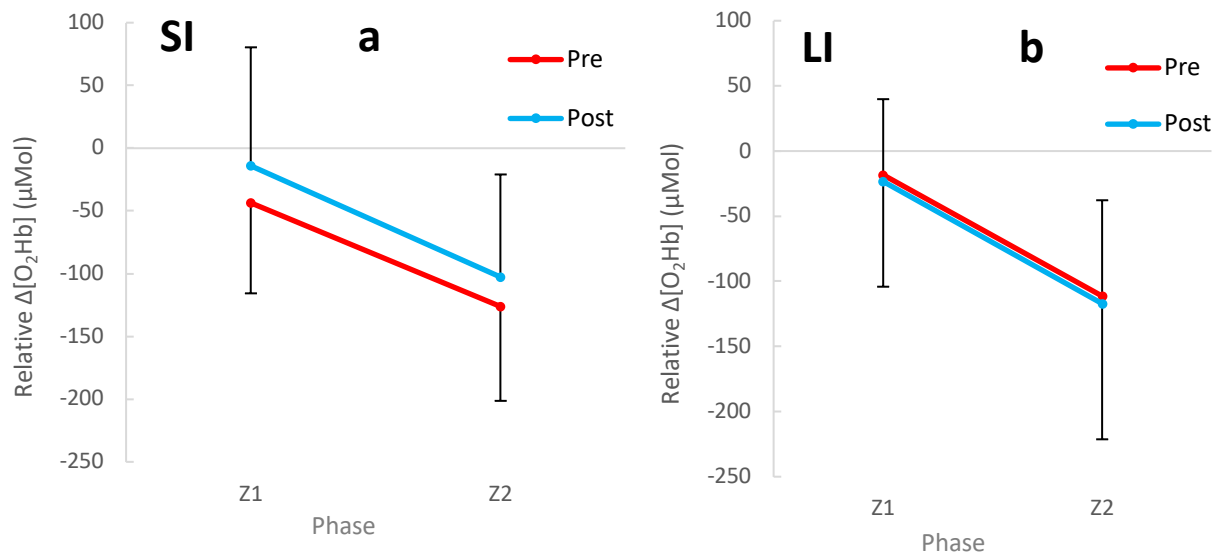
Short Interval					
	Pre	Post	ES (95% CI)	Qualitative outcome	% chance of increase/trivial/decrease effect
Z1	-43.6 $\pm$ 94.42	-14.0 $\pm$ 72.05	0.35 (-0.65; 1.32)	Small	70/26/4
Z2	-126.1 $\pm$ 81.78	-102.7 $\pm$ 75.13	0.30 (-0.70; 1.27)	Small	61/30/10

Z1: onset of blood lactate accumulation and Z2: peak power. All mean values represent a 30 s average of  $\Delta[\text{O}_2\text{Hb}]$  relative to the baseline reading.

**Table 5.6b: Within-group changes (pre- to post- intervention) in  $\Delta[\text{O}_2\text{Hb}]$  for the long interval HIT programme (mean  $\pm$  SD) during the incremental exercise test.**

Long Interval					
	Pre	Post	ES (95% CI)	Qualitative outcome	% chance of increase/trivial/decrease effect
Z1	-18.3 $\pm$ 63.14	-23.3 $\pm$ 85.86	-0.07 (-1.04; 0.92)	Negligible	30/30/40
Z2	-111.3 $\pm$ 79.32	-117.1 $\pm$ 109.92	-0.06 (-1.04; 0.92)	Negligible	31/30/39

Z1: onset of blood lactate accumulation and Z2: peak power output. All mean values represent a 30 s average of  $\Delta[\text{O}_2\text{Hb}]$  relative to the baseline reading.



**Figure 5.14** Relative  $\Delta[\text{O}_2\text{Hb}]$  for (a) the SI training group and (b) the LI training group during the incremental cycling test to exhaustion. Values are means  $\pm$  SD. Z1: onset of blood lactate accumulation and Z2: peak power output.

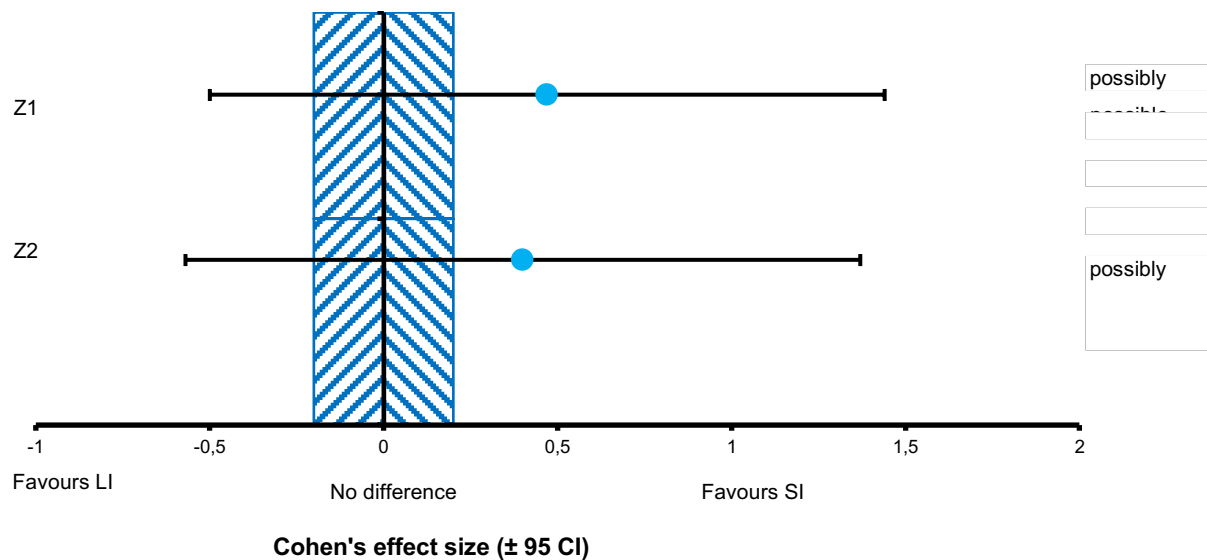
### 5.5.1.2 Between-group changes

The between-group differences in  $\Delta[\text{O}_2\text{Hb}]$  are shown in Table 5.6c for the SI and LI training groups, with Figure 5.15 providing a graphical illustration of the mean differences between the groups. There were no statistically significant differences ( $P > 0.05$ ) in the between-group changes. The likelihood that the observed differences were practically meaningful was also likely small.

**Table 5.6c: Between-group differences (pre- to post- intervention) in  $\Delta[\text{O}_2\text{Hb}]$  (mean  $\pm$  SD) during the incremental exercise test.**

	SI mean difference	LI mean difference	ES (95% CI)	Qualitative outcome	% chance of increase/trivial/decrease effect
Z1	29.5 $\pm$ 82.90	-5.1 $\pm$ 62.89	0.47 (-0.55; 1.44)	Small	70/20/10
Z2	23.5 $\pm$ 58.91	-5.8 $\pm$ 84.03	0.40 (-0.61; 1.37)	Small	65/22/12

Z1: onset of blood lactate accumulation and Z2: peak power output. All mean values represent a 30 s average of  $\Delta[\text{O}_2\text{Hb}]$  relative to the baseline reading.



**Figure 5.15** Forest plot representing the effect sizes for the mean differences between groups in  $\Delta[O_2Hb]$  measures. A positive effect favours the short interval and a negative effect favours the long interval intervention. The smallest worthwhile change threshold was set at an effect size greater than 0.2 or smaller than -0.2 shown on the graph with the shaded column. Z1: onset of blood lactate accumulation, Z2: peak power output,  $O_2Hb$ : oxygenated hemoglobin, SI: short interval and LI: long interval.

## 5.5.2 Deoxygenated hemoglobin ( $\Delta[HHb]$ )

The change in deoxygenated hemoglobin ( $\Delta[HHb]$ ) is an indicator of the removal of  $O_2$  from the  $O_2Hb$  complex in the localized muscle, through the increased utilization of  $O_2$  during exercise of increasing workloads (Usaj, 2001).

### 5.5.2.1 Within-group changes

Table 5.7a (SI training group) and Table 5.7b (LI training group) present the within-group changes in  $\Delta[HHb]$  before and after the interventions. There were no statistically significant differences ( $P > 0.05$ ) in these changes in either group. The analysis of group\*time (pre to post), group\*repeat (zones) and repeat\*time\*group also showed no statistically significant interactions (Fig 5.16).

The SI training group experienced practically small meaningful decreases in  $\Delta[HHb]$  during the exercise phases, with a likely small decrease at OBLA (ES = 0.56) (Table 5.7a). There was also a likely moderate decrease in  $\Delta[HHb]$  during submaximal exercise in the LI group (ES = 0.56), however, no meaningful change at peak exercise (Table 5.7b).

**Table 5.7a: Within-group changes (pre- to post-intervention) in  $\Delta$ [HHb] for the short interval HIT programme (mean  $\pm$  SD) during the incremental exercise test.**

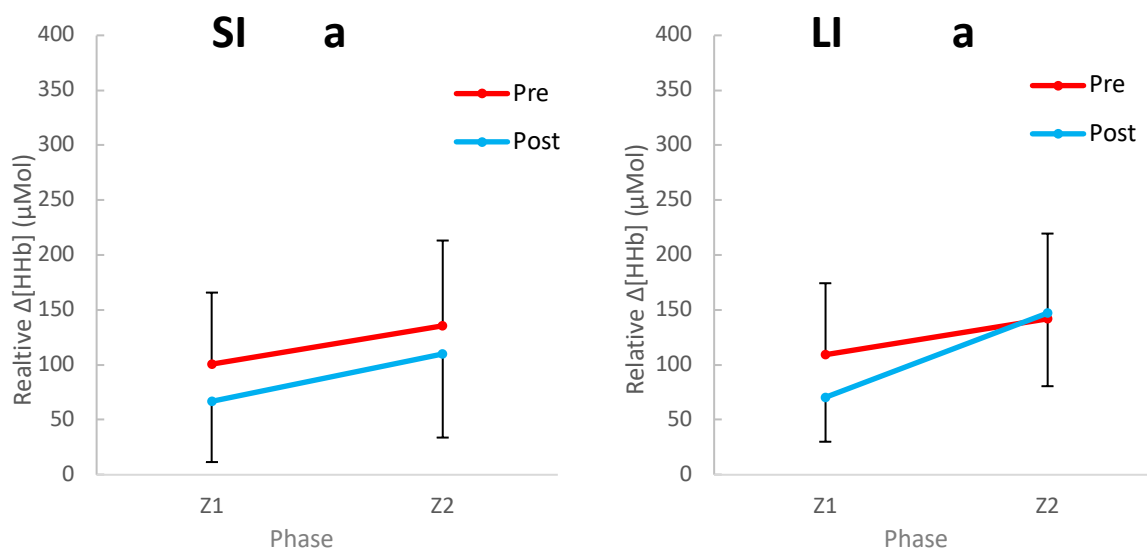
Short Interval					
	Pre	Post	ES (95% CI)	Qualitative outcome	% chance of increase/trivial/decrease effect
Z1	100.7 $\pm$ 55.33	66.9 $\pm$ 65.11	-0.56 (-1.53; 0.47)	Small	8/16/76
Z2	135.6 $\pm$ 76.13	109.9 $\pm$ 77.64	-0.33 (-1.30; 0.67)	Small	15/24/60

Z1: onset of blood lactate accumulation and Z2: peak power output. All mean values represent a 30 s average of  $\Delta$ [HHb] relative to the baseline reading.

**Table 5.7b: Within-group changes (pre- to post-intervention) in  $\Delta$ [HHb] for the long interval HIT programme (mean  $\pm$  SD) during the incremental exercise test.**

Long Interval					
	Pre	Post	ES (95% CI)	Qualitative outcome	% chance of increase/trivial/decrease effect
Z1	109.3 $\pm$ 40.36	70.4 $\pm$ 74.89	-0.65 (-1.61; 0.39)	Moderate	6/13/81
Z2	141.9 $\pm$ 66.52	147.1 $\pm$ 67.21	0.08 (-0.91; 1.05)	Negligible	41/30/29

Z1: onset of blood lactate accumulation and Z2: peak power output. All mean values represent a 30 s average of  $\Delta$ [HHb] relative to the baseline reading.

**Figure 5.16**

Relative  $\Delta$ [HHb] for (a) the SI training group and (b) the LI training group during the incremental cycling test to exhaustion with recovery phases. Values are means  $\pm$  SD. Z1: onset of blood lactate accumulation, Z2: peak power output, R1: 30 s recovery, R2: 60 s recovery, R3: 90 s recovery and R4: 120 s recovery.

### 5.5.2.2 Between-group changes

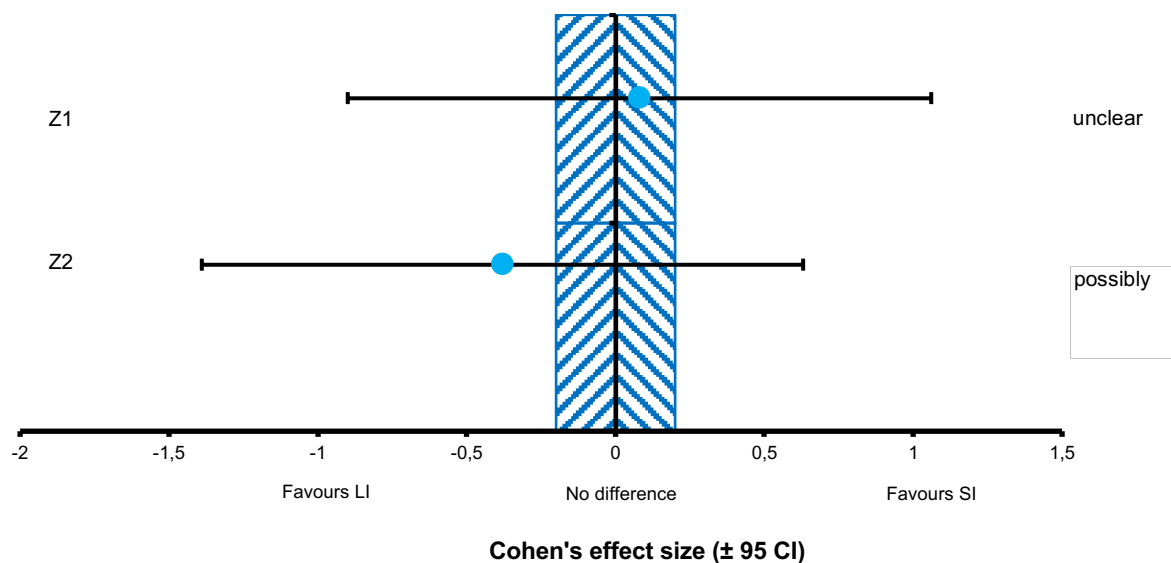
The between-group comparisons for  $\Delta$ [HHb] are depicted in Table 5.7c. No statistically significant differences ( $P > 0.05$ ) were observed between the groups in response to the interventions. At most, a possible small difference in  $\Delta$ [HHb] was observed at peak exercise in favour of the LI group, however, there was no clear benefit for either programme during submaximal exercise (Fig. 5.17).



**Table 5.7c: Between-group differences (pre- to post-intervention) in  $\Delta$ [HHb] (mean  $\pm$  SD) during the incremental exercise test.**

	SI mean difference	LI mean difference	ES (95% CI)	Qualitative outcome	% chance of increase/trivial/decrease effect
Z1	-33.9 $\pm$ 60.62	-38.8 $\pm$ 57.54	0.08 (-0.90; 1.06)	Negligible	41/30/29
Z2	-25.6 $\pm$ 64.40	5.2 $\pm$ 96.43	-0.38 (-1.34; 0.63)	Small	13/23/63

Z1: onset of blood lactate accumulation and Z2: peak power output. All mean values represent a 30 s average of  $\Delta$ [HHb] relative to the baseline reading.

**Figure 5.17**

Forest plot representing the effect sizes for the mean differences between groups in  $\Delta$ [HHb] measures. A positive effect favours the short interval and a negative effect favours the long interval intervention. The smallest worthwhile change threshold was set at an effect size greater than 0.2 or smaller than -0.2 shown on the graph with the shaded column. Z1: onset of blood lactate accumulation, HHb: deoxygenated hemoglobin, SI: short interval and LI: long interval.

### 5.5.3 Percentage maximal muscle saturation (%MMS) of O<sub>2</sub>

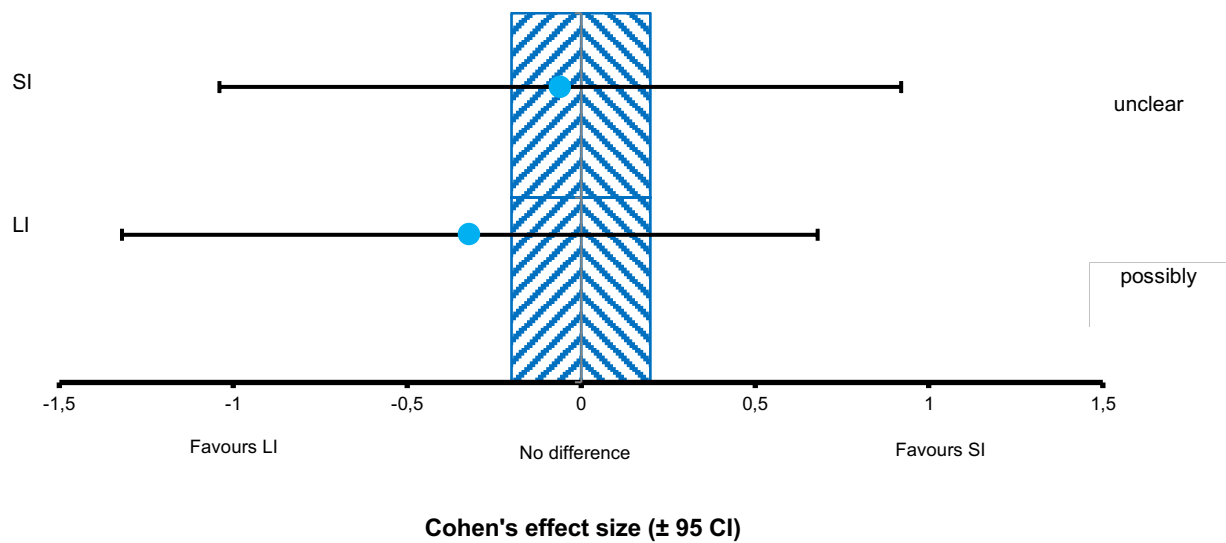
The %MMS of O<sub>2</sub> at the point of OBLA provides an indication of the desaturation of O<sub>2</sub> in the tissue. The relative change in this percentage can be used to indicate whether there was an increase in extraction/utilization of O<sub>2</sub> at the point of OBLA following training.

Table 5.8 and Fig 5.18 show that the LI training possibly caused a small meaningful increase in O<sub>2</sub> extraction/utilization at OBLA (Z1), but that the change following SI training was negligible.

**Table 5.8: Within-group changes in %MMS of O<sub>2</sub> at OBLA following the HIIT interventions (mean ± SD).**

	Pre (%)	Post (%)	ES (95% CI)	Qualitative outcome	% chance of increase/trivial/decrease effect
SI	32.6 ± 35.13	30.9 ± 16.97	-0.06 (-1.04; 0.92)	Negligible	31/30/39
LI	30.5 ± 16.71	23.4 ± 26.73	-0.32 (-1.29; 0.68)	Small	16/25/59

%MMS: percentage maximal muscle saturation; SI: short interval and LI: long interval



**Figure 5.18**

Forest plot representing the effect sizes for the mean differences in %MMS of O<sub>2</sub> measures. The smallest worthwhile change threshold was set at an effect size greater than 0.2 or smaller than -0.2 shown on the graph with the shaded column. Z1: onset of blood lactate accumulation, %MMS: percentage of maximal muscle saturation, SI: short interval and LI: long interval.

# Chapter 6

## Discussion

### 6.1 Introduction

High intensity interval training (HIIT) is an effective method of training in multiple sporting codes. The benefits are not limited to peak sport performance. It is also a time efficient method of training which leads to similar, and sometimes better, physiological adaptations than traditional moderate intensity continuous training (MICT). Therefore, this training methodology is very advantageous for recreational cyclists, as they generally have limited training time and need to use their available time as effectively as possible.

The hemodynamic changes during an incremental exercise test to exhaustion in response to two types of HIT programmes have not been reported previously. The alteration of one of the nine features of a HIIT programme (Buchheit and Laursen, 2013a) could lead to diverse physiological adaptations (Kilpatrick *et al.*, 2015). MacInnis and Gibala (2017) also highlighted that more research is needed into the integrated physiological responses to HIIT. The current study is the first to describe the hemodynamic adaptations in muscles in response to high intensity training that differed with respect to interval duration. Thus, the purpose of the study was to broaden our understanding of the physiological adaptations to HIIT in recreationally active cyclists, with specific focus on the peripheral hemodynamic changes.

The primary aim of the study was to assess the hemodynamic changes ( $\Delta[\text{O}_2\text{Hb}]$  and  $\Delta[\text{HHb}]$ ) in the *vastus lateralis* muscles following a brief HIIT programme consisting of either short intervals or long intervals. A secondary aim was to compare the cycling performance benefits (aerobic and anaerobic) of the two HIIT programmes. The knowledge gained from this study is beneficial for the prescription of HIIT as part of the annual periodized training programme of recreational cyclists.

In this discussion emphasis will be placed on the results obtained from effect size statistics and magnitude based inferences, in other words, the chance of finding better, unclear or poorer responses in the outcome variables relative to the smallest worthwhile difference, also known as the practically important threshold. The reasons are twofold: firstly, the findings are based on a small sample size

( $n = 16$ ), which constrains the external validity of the conclusions according to traditional statistics. However, effect size statistics are independent of sample size; unlike statistical significance tests where P values are confounded by sample size (Sullivan & Feinn, 2012). Secondly, absolute changes in sport performance parameters are usually very small; however, even the smallest changes (i.e. improvements) may mean the difference between winning and losing for the athlete. Thus, effect sizes and magnitude-based inferences are a much better indicator of the real-world (i.e. practical) significance of an outcome (Batterham & Hopkins, 2005).

### 6.1.1 Characteristics of the study sample

The participants in the study were of similar age and body mass than participants in previous HIIT studies (Costes *et al.*, 2001; Neary *et al.*, 2002; Burgomaster *et al.*, 2008; Rønnestad *et al.*, 2012; Rønnestad *et al.*, 2015;). There were no age, body composition and cycling performance differences at baseline between the short interval and long interval HIT groups, which allow for fair comparisons.

Even though the participants in the current study are described as recreational cyclists, they trained a fair amount of hours per week ( $7.3 \pm 1.94$ ) and they participated regularly in cycling races over various distances. This study was conducted at the end of the official cycling season and the participants reported a reduced cycling training volume during the study intervention. Participants were also instructed to limit their training outside the laboratory to low intensity sessions with the aim to provide active recovery in aid of the physiological responses following the HIT sessions. Thus, it is assumed that the physiological and performance changes that were observed in this study can primarily be attributed to the HIIT programmes.

The mean percentage body fat for both groups (5 - 22%) fell within the health/fitness range for men (Powers and Howley, 2012). Therefore, it is unlikely that adipose tissue thickness interfered with the NIRS signal. Additionally skin pigmentation was ruled out as an interfering factor, as all participants were of fair complexion. To minimize the effect of dietary intake, or changes in nutrition on the cyclists' performances in the laboratory tests (Jones and Burnley, 2009), the participants' nutritional consumption was recorded (with portion sizes) for the 24 hour period prior to pre-testing and they were requested to repeat this nutrition plan for the post-testing (Bogdanis *et al.*, 1996).

According to the ACSM guidelines (2010), the normative range for [Hb] in healthy men is 13.5 - 17.5 gr·dl<sup>-1</sup>. The baseline measure for one of the participants in the short interval HIT programme was below the expected minimum (12.7 gr·dl<sup>-1</sup>), however, his post-test [Hb] was within the normative range (14.4 gr·dl<sup>-1</sup>). Although the possibility cannot be excluded that this low baseline value could have affected his physiological and performance responses, Bækkerud *et al.* (2015) found that [Hb] is not a direct reflection of the benefits associated with O<sub>2</sub> carrying capacity. Furthermore, this participant's observed hemodynamic responses fitted the trend for all the cyclists in the short interval HIT and on this basis his data were not excluded from the data set.

### 6.1.2 Overview of findings

The primary findings of the current study were that the short interval HIT programme was likely more effective in the enhancement of O<sub>2</sub> delivery to the periphery which lead to an increase in  $\Delta[\text{O}_2\text{Hb}]$  in the muscles. The long interval HIT programme was possibly more effective in improving the extraction and utilization of O<sub>2</sub> in the periphery. Therefore it is postulated that a short interval HIT probably leads to mostly central aerobic adaptations and a long interval HIT programme probably mostly peripheral aerobic changes.

The secondary finding suggest that a short interval HIT programme is possibly more useful in improving a larger spectrum of both aerobic and anaerobic cycling performance parameters. In this study the short interval HIT resulted in larger improvements in eight out of the eleven selected maximal aerobic performance markers, in comparison to the five out of eleven that were improved by the long interval HIT programme. Similarly, four out of the six anaerobic power performance markers were improved after the short interval HIT, while the long interval HIT resulted in small, but better performances in all the anaerobic performance markers. Both HIIT programmes led to moderately lower resting blood [La], while changes in the lactate profiles were possibly more pronounced after the long interval HIT programme. Notably, the differences in performance benefits for the two effort-matched HIIT programmes were evident after only 12 sessions (6 weeks).

## 6.2 Research Hypothesis

### 1<sup>st</sup> Hypothesis

*A short interval HIT programme will illicit greater muscle oxygenation adaptations than a long interval HIT programme. Rønnestad et al. (2015) found significantly greater performance parameter improvements due to short interval HIT than long interval HIT. These performance benefits have been attributed to mostly central adaptations. However, peripheral adaptations should also be present as they have a causal link to the enhancement of the performance improvements (Costes et al., 2001; Neary et al., 2002; Daussin et al., 2008).*

Exercise training adaptations are generally categorised as either centrally mediated, through the heart, circulation and brain, or peripherally mediated, through muscle specific changes (Neary et al., 2002; Guiraud et al., 2010; Murais et al., 2010; Rozenek et al., 2016). For example, it is generally accepted that maximal oxygen uptake and utilization during exercise ( $VO_{2max}$ ) is dependent on the delivery capacity of the heart (i.e. maximal cardiac output ( $Q_{max}$ )), as well as peripheral (muscle) factors that enhances oxygen extraction (i.e. arterial-venous oxygen difference) (Powers and Howley, 2012). In the current study, measures of oxyhemoglobin and deoxyhemoglobin were recorded via NIRS to assess whether short-term HIT with different interval durations and the accompanying changes in cycling performance can be attributed to either central or peripheral training adaptations.

The hemodynamic changes associated with the short interval and long interval HIT programme can be found in Chapter 5 (section 5.5). Figures 6.1 and 6.2 illustrate the hypothesized adaptation pathways for both interventions.

#### 6.2.1 Hemodynamic changes during exercise

The results of the present study seem to indicate that a short interval HIT intervention possibly facilitated higher levels of  $\Delta[O_2Hb]$  and lower levels of  $\Delta[HHb]$  in the muscles during both submaximal and peak exercise (Table 5.6a and Table 5.7a). On the other hand, these changes in hemodynamics were negligible after the long interval HIT intervention.

It is suggested that the training responses in the short interval HIT group reflects enhanced arterial blood flow, resulting in more  $O_2$  availability to the active muscles. The improved  $\Delta[O_2Hb]$  presence

may be due to an increase in blood volume, stroke volume or increased O<sub>2</sub> carrying capacity of blood Hb. Both groups presented with higher blood [Hb] during post-testing, which must be assumed is a consequence of the training interventions, as was previously observed by Jones and Carter (2000) and Choudhary *et al.* (2012). However, the long interval HIT group had a slightly larger increase in [Hb] than the short interval HIT group, which implies that their O<sub>2</sub> carrying capacity could have been higher. Therefore, it is more likely that O<sub>2</sub> availability to the active muscles were enhanced through changes in blood volume and/or stroke volume. This study did not include the measurement of either of these variables. However, O<sub>2</sub> pulse, which is a predictor of the stroke volume of the heart (Bhambhani *et al.*, 1994; Neary *et al.*, 2002), was calculated from the available data. The short interval HIT group had a clinically significant increase in O<sub>2</sub> pulse following the programme, while the increase in the long interval HIT group was negligible. This evidence supports the hypothesis that the short interval HIT programme elicited more pronounced central adaptations than the long interval HIT programme.

Typically, peripheral adaptations to exercise training include skeletal muscle capillarization (Hoppeler *et al.*, 1985), increased mitochondrial density (Holloszy and Coyle, 1984) and increased oxidative enzyme function (Neary *et al.*, 2001). Skeletal muscle capillarization through HIIT is mediated through vascular endothelial growth factor which is released following exercise training. MacInnis and Gibala (2017) observed that exercise intensity is an important factor in the adaptation of mitochondria, with longer formats of HIT (i.e. long interval HIT) being more beneficial than shorter formats of HIT.

The hemodynamic changes following the long interval HIT programme in this study are consistent with previous findings that suggest increased O<sub>2</sub> utilization and extraction at sub-maximal exercise intensities. Cyclists in the short interval HIT programme showed minimal peripheral adaptations after the twelve training sessions. This was illustrated in the negligible changes in maximal muscle O<sub>2</sub> saturation (%MMS) at the onset of blood lactate accumulation (OBLA). In contrast, the %MMS of O<sub>2</sub> at OBLA were possibly lower after the long interval HIT than at baseline (Table 5.8). Neary *et al.* (2002) suggested that increased deoxygenation is mediated by an increased release of O<sub>2</sub> from the O<sub>2</sub>Hb complex through the Bohr effect. They proposed that this is brought about by adaptations around and within muscle fibers which will enhance the (a-v)O<sub>2</sub>diff (i.e. oxygen extraction) by active muscle fibers, such as increased capillarization, mitochondrial density and improved activity of oxidative enzymes in the mitochondria. These findings are in line with those of Costes *et al.* (2001),

who showed that  $\Delta[\text{O}_2\text{Hb}]$  in the active muscles is lower during an incremental exercise test following an endurance training programme. They deduced that the reduction in  $\Delta[\text{O}_2\text{Hb}]$  during submaximal exercise was the result of increased  $\text{O}_2$  utilization by the muscles. In addition, Zwaard *et al.* (2016) compared the hemodynamic responses during exercise of individuals with and without cycling experience. They found that participants with cycling experience were able to reach a %MMS of  $\text{O}_2$  at OBLA that was 15% lower than the participants with no experience. The authors proposed that the muscles of the experienced group had an enhanced ability to extract and utilize  $\text{O}_2$ .

It is concluded that consistently longer durations of high intensity exercise, as with a long interval HIT programme are more conducive to peripheral adaptations in the active muscles. This deduction stems from the practically meaningful enhancement in  $\text{O}_2$  extraction and utilization during submaximal exercise in response to the long interval HIT compared to the short interval HIT programme.

## 2<sup>nd</sup> Hypothesis

*A short interval HIT programme will elicit greater changes in aerobic capacity in comparison to a long interval HIT programme.*

### 6.2.2 Changes in aerobic capacity

It was found in the present study that eight of the eleven cardiometabolic markers ( $\text{VO}_{2\text{max}}$ ,  $\text{O}_2$  pulse, PPO, PPO: BM, PO at OBLA, HR at OBLA and  $[\text{La}]_{\text{max}}$ ) were likely more improved by the short interval HIT programme compared to the long interval HIT programme. The increase in  $\text{VO}_{2\text{max}}$  by the cyclists in the short interval HIT programme was roughly two-fold higher than that of the long interval HIT group (absolute  $\text{VO}_{2\text{max}}$ : +6% vs +3% and relative  $\text{VO}_{2\text{max}}$ : +5% vs +2%, respectively). Similar findings were seen for PPO and PO at OBLA (PPO: + 7% vs +5%; PO at OBLA: +3 vs +2%, respectively), which suggests that the short interval HIT programme was more favourable in enhancing aerobic capacity in comparison to the long interval HIT programme.

The improvement in  $\text{VO}_{2\text{max}}$  is a common finding in HIIT studies (Creer *et al.*, 2004; Perry *et al.*, 2008; Walter *et al.*, 2010; Rønnestad *et al.*, 2015; Zinner *et al.*, 2016). These previous studies reported increases in  $\text{VO}_{2\text{max}}$  of between 2.6% to 10%, in response to similar HIT programmes (short interval and long interval HIT) and over similar durations (9 - 20 sessions) than the current study. The cyclists



in these studies were either recreationally active (Creer *et al.*, 2004; Perry *et al.*, 2008; Walter *et al.*, 2010; Zinner *et al.*, 2016) or highly trained (Rønnestad *et al.*, 2015). Thus, the responses of the cyclists in the current study compares well with those in the literature and should not be disregarded because of a lack of statistical significance.

Buchheit and Laursen (2013a) proposed that the improvements in  $VO_{2max}$  with short interval HIT programmes which is associated with higher exercise intensities during the intervals are due to the near maximal repeated ventricular filling at high rates, as well as higher heart rates, which is associated with cardiovascular training adaptations. This provides a feasible explanation as to why the short interval HIT intervention in the present study resulted in greater improvements in  $VO_{2max}$  than the long interval HIT intervention.

In contrast to the findings of previous and the current study, Bækkerud *et al.* (2015) reported greater central adaptations ( $O_2$  delivery) after a 4 x 4 min HIT intervention (similar to the long interval HIT in this study) compared a 10 x 1 min HIIT (+9% vs +3%, respectively) intervention (similar to short interval HIT in this study). However, Bækkerud *et al.* (2015) failed to control the work to recovery ratio between the two HIT groups; the 4 x 4 min HIT programme had a larger work to rest ratio (4:3) than the 10 x 1 min HIT group (1:3). The work duration was also longer for the 4 x 4 min HIT indicating that the training stimulus was not the same for the two groups. Nonetheless, both training groups showed greater aerobic improvements ( $VO_{2max}$ ,  $O_2$  pulse and time to exhaustion) in comparison to the MICT programme, thus supporting the notion that HIIT is more effective than MICT.

In the current study, the cyclists in the short interval HIT group improved their PPO during the incremental exercise test to exhaustion by 7%, which is in accordance with previous studies who reported improvements of 4% to 9% (Creer *et al.*, 2004; Rønnestad *et al.*, 2015; Zinner *et al.*, 2016). Thus, it seems that PPO during an incremental exercise test is more likely to increase following a 30 s short interval format (as used in the present and previous studies) than with longer interval HIT programmes. It is postulated that this performance improvement can be attributed to neuromuscular adaptations and the associated changes in motor recruitment patterns, which are more pronounced at higher power outputs ( $PO > 85\% VO_{2max}$ ) (Esbjörnsson-Lijedahl *et al.*, 1985; Powers and Howley, 2012; Buchheit and Laursen, 2013b). Thus, it is suggested that the short interval HIT led to greater improvements in PPO because of the higher work outputs during training. In addition, there are an

increased number of accelerations from rest with the short interval HIT. The accelerations provided an additional neuromuscular stimulus as there would be increased motor unit activation from the explosive strength (torque) movement pattern to overcome the inertia of the rest periods (Powers and Howley, 2012; Rønnestad *et al.*, 2015).

Similar to the findings of the current study, Rønnestad *et al.* (2015) and Inoue *et al.* (2016) also found improvements in submaximal work capacity (PO at OBLA). The previous studies found improvements favouring a short interval (30 s) HIT (+4% to +9%) in comparison to a long interval (4 min) HIT (+2% to +4%). Of note is that the participants in the study of Rønnestad *et al.* (2015) were well-trained cyclists, while the current study involved recreational cyclists. Nevertheless, the magnitude of change in PO at OBLA was in the same region.

In the current study, the shorter interval period allowed the participants to reach higher PO in comparison to the longer intervals where participants had to maintain the highest possible PO for an extended period of time (4 min). It is therefore unlikely that the long interval HIT group achieved the same PO during their sessions as the short interval HIT group. Importantly, however, is that these two programmes were matched for training load and effort, based on session duration and session RPE, as was done by Rønnestad *et al.* (2015). In the latter study, the effort matched groups showed no differences in sRPE or [La] after each HIT session and it was therefore assumed that the workloads in the two HIT programs were matched.

It is thus put forward that a short interval HIT programme is more conducive to changes in aerobic capacity than a long interval HIT programme, specifically in recreational cyclists. As indicated in Fig 6.1., it is proposed that the improvements in performance parameters are mainly attributed to central (cardiovascular) physiological adaptations, as well as the higher exercise intensities that characterised the short interval HIT programme. As submitted by MacInnis and Gibala (2017), exercise intensity is an important mediating factor in the performance changes that can be expected in response to endurance training, irrespective of training format (MICT or HIIT)

### 3<sup>rd</sup> Hypothesis

*Due to the increased number of accelerations from rest in the short interval HIT programme, there will be a greater improvement in anaerobic performance parameters and higher levels of accumulated blood [La] during the Wingate 30 s test compared with the long interval HIT programme.*

#### 6.2.3 Changes in anaerobic capacity

The effects of interval duration on anaerobic capacity and blood lactate recovery were equivocal. The LI group very likely outperformed the SI group during the first Wingate test after the intervention, while there was a small chance that the SI group performed better during the second Wingate test. Both groups experience clinically significant decreases in resting blood [La] during the follow-up testing. No clear and meaningful changes were observed in the lactate profiles of the SI group, while the small changes in lactate accumulation and recovery in the LI group were possibly meaningful.

Contrary to our hypothesis, the cyclists in the long interval HIT programme increased their PPO in the first Wingate and peak blood [La] accumulation to a greater magnitude than the short interval HIT programme. Although two previous studies found similar findings (Creer *et al.*, 2004; Rønnestad *et al.*, 2015), the methodologies of these studies were different from the current study. For instance, participants in the study by Rønnestad *et al.* (2015) only performed a single Wingate test. Nevertheless, these authors explained that the longer intervals cause more neural adaptations in the muscles, as well as an increased glycolytic metabolic output (anaerobic metabolism) and thus higher blood [La], compared to the short interval programmes.

Perry *et al.* (2008) observed similar blood lactate responses following a long interval HIT intervention as in the current study. They used a similar long interval HIT programme and over the same study duration in recreationally active participant group and they also observed an increased blood [La] of  $+2.1 \text{ mmol}\cdot\text{L}^{-1}$  following a Wingate test. The increased lactate accumulation was possibly due to either an increase in buffering capacity or an increased activity of lactate carrier proteins (MCT1 and MCT4), which are both typical endurance training adaptations. Both these adaptations would facilitate increases in lactate tolerance during high intensity exercise Perry *et al.* (2008). Unfortunately, the previous study did not include a comparative group, however, it does suggest that the findings in the current study are feasible and worthy of further investigation.

There was an initial delay in lactate recovery for the long interval HIT group following the second Wingate test in the present study. The initial [La] clearance delay may be as a result of the unchanged O<sub>2</sub> delivery during exercise. The metabolism of lactate is largely an oxygen dependant process (Emhoff *et al.*, 2013), which occurs either directly through utilization by the active cells or brain, or indirectly through the conversion to glycogen in the liver (glycogenesis). The lack of a concomitant increase in O<sub>2</sub> supply following training, in the wake of a higher work output and therefore higher metabolic demand (resulting in increased [La]) could explain the elevated [La] during recovery.

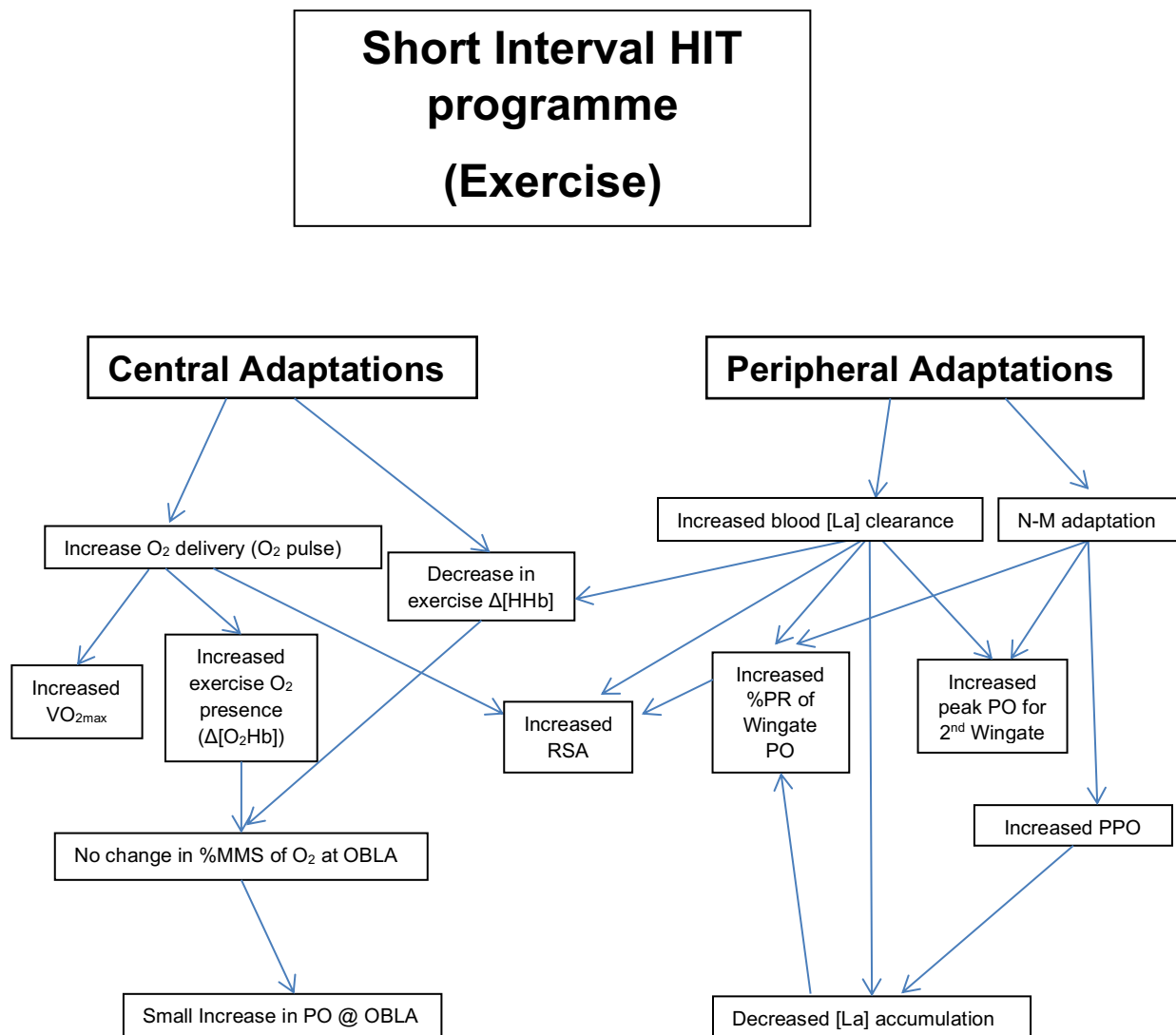
McGawley and Bishop (2015) attributed their findings on repeated sprint performance after a short interval HIT intervention to an increased contribution of aerobic metabolism (45% difference from 1<sup>st</sup> to last sprint performance). This finding is in agreement with the current study which found that the SI group had a better ability to maintain PO (RSA) in the successive Wingate tests in comparison to the LI group, which suggests that they possibly recovered at a quicker rate after the first all-out effort, which is in line with the suggestion of Bogdanis *et al.* (1996). Thus, the improvement in maximal aerobic capacity in the SI group likely contributed to a higher aerobic contribution and better fatigue resistance during the Wingate tests. This would not only justify their higher PO during the all-out exercise, but also the minimal changes in their blood [La] profiles.

The weak correlations between the changes in Wingate PO and blood [La] for both interventions (Table 5.5d) suggest that maximal anaerobic capacity was not only improved through anaerobic adaptations (e.g. increase in muscle strength and power), but that the aerobic changes contributed to the improved anaerobic performances. In addition, Brooks *et al.* (2000) observed that a decreased [La] following a training programme was a result of improved lactate oxidation during recovery (O<sub>2</sub> dependant process).

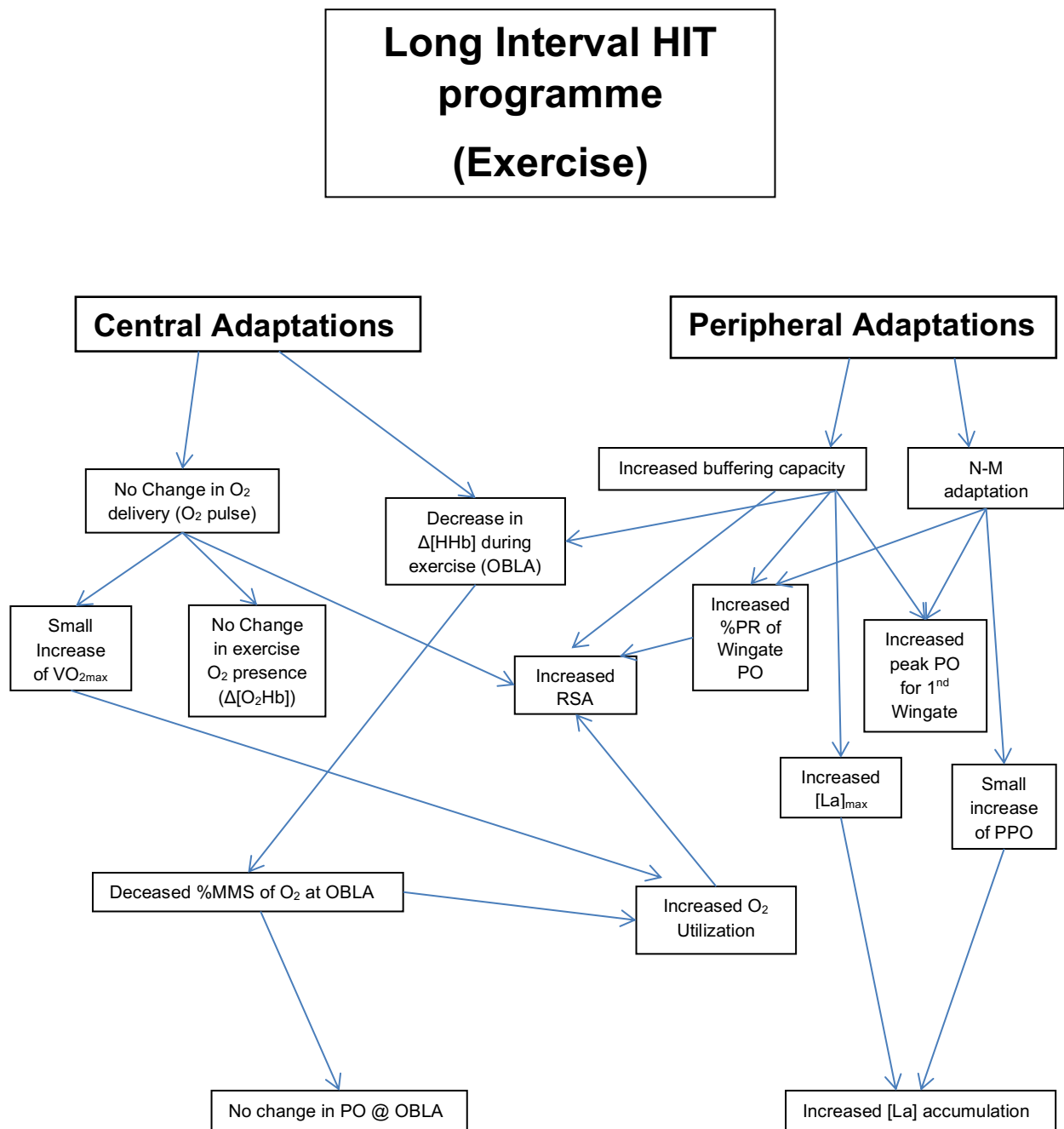
The repeated sprint ability (RSA) of the cyclists following the short interval HIT programme in the present study improved more than the group in the long interval HIT. The maximum and mean PO increased by 1.9% and 1.5% in the short interval HIT group, compared to the 1.2% and 0.7% change in the long interval HIT group. Thus, the short interval HIT group was able to achieve more consistent mean and maximum PO during the repeat Wingate test. This improvement in RSA has previously been attributed to a pacing strategy (subconsciously attained). Ansley *et al.* (2004) showed that cyclists employ a pacing to maintain sprint power in successive sprints. However, this explanation can

be ruled out in the current study as the blood [La] profiles indicate that the lactate values of the short interval HIT group increased to a greater extent following W1 than W2. This suggests that the short interval HIT group did not pace themselves to maintain a more consistent repeated Wingate performance.

The findings of this study on the effects of different types of HIIT interventions is inconclusive and should be the focus of future studies. There are only slim evidence to suggest that a long interval HIT is more favourable in improving anaerobic capacity than a short interval HIT. If we assume this to be true, we propose that the likely physiological mechanisms behind these anaerobic adaptations relate to the peripheral adaptations, which enable the muscle to tolerate higher levels of blood [La]. The present findings are in contrast with most other studies who measured anaerobic capacity (Creer *et al.*, 2004; Rønnestad *et al.*, 2015), however, the methodological differences in the training prescription probably account for the contrasting results.

**Figure 6.1**

Hypothesized model for the exercise physiological and performance adaptations to a short interval programme.  $VO_{2max}$ : maximal oxygen uptake;  $O_2Hb$ : oxygenated hemoglobin;  $HHb$ : deoxygenated hemoglobin; %MMS: percentage of maximal muscle saturation; OBLA: onset of blood lactate accumulation; %PR: percentage power of reproducibility; PO: power output;  $O_2$  pulse: oxygen pulse; RSA: repeated sprint ability; N-M: neuromuscular; PPO: peak power output and  $[La]$ : concentration of lactate.

**Figure 6.2**

Hypothesized model for the exercise physiological and performance adaptations to a long interval programme.  $VO_{2max}$ : maximal oxygen uptake;  $O_2Hb$ : oxygenated hemoglobin;  $HHb$ : deoxygenated hemoglobin; %MMS: percentage of maximal muscle saturation; OBLA: onset of blood lactate accumulation; %PR: percentage power of reproducibility; PO: power output;  $O_2$  pulse: oxygen pulse; RSA: repeated sprint ability; N-M: neuromuscular; PPO: peak power output and  $[La]$ : concentration of lactate.

### 6.3 Overview of findings

Hypothesis 1: *A short interval HIT programme will illicit greater muscle oxygenation adaptations than a long interval HIT programme.*

The muscle oxygenation changes associated with the short interval HIT programme were increased more than the long interval HIT programme. The increased presence of  $\Delta[\text{O}_2\text{Hb}]$  associated with the short interval HIT programme is indicative of central adaptations taking place with this training modality. Based on the findings, this hypothesis is **accepted**.

Hypothesis 2: *A short interval HIT programme will elicit greater changes in aerobic capacity benefits in comparison to a long interval HIT programme.*

The current study identified improvements in eight out of the eleven selected aerobic metabolic markers by the short interval HIT programme in comparison to the long interval HIT programme. The present findings suggest that the hypothesis must be **accepted**.

Hypothesis 3: *Due to the increased number of accelerations from rest in the short interval HIT programme, there will be a greater improvement in performance parameters and higher levels of accumulated blood [La] during the Wingate 30 s test compared with the long interval HIT programme.*

The long interval HIT showed an increased blood [La] accumulation following the performance of two 30 s Wingate tests. This could suggest that an improved buffer capacity and an increased [La] tolerance may have been attained. Based on these findings the hypothesis is **rejected**, however, with serious reservations due to the absence of clear differences between the interventions.



## 6.4 Practical Implications

The findings of the present study indicate that the short interval HIT stimulate a likely increased rate of adaptation over 6 weeks for the majority of endurance performance predictor outcomes in comparison to the long interval HIT. In contrast, the long interval HIT programme points toward a very likely performance benefit if the competition demands were anaerobic in nature, such as a track sprint event, lasting roughly 30 s in one isolated performance (i.e. no repetitions without a full recovery).

The postulated link between the performance parameters and the hemodynamic changes can be used to non-invasively measure benefits following a training programme. The efficacy of a training program can also be evaluated with the NIRS measures and used to identify required training prescription adjustments.

In conclusion, the performance outcomes from the present study can be used in the tailoring and periodization of training. The training session selection can be further personalized and based on specific training needs of the recreationally active athlete. The training sessions can either be based off of the competition demands or the identified weaknesses of the athlete. The specialization of the training session selection will ensure the athlete is able to achieve the best possible competition performance.

## 6.5 Limitations

- The small sample size of the present study limited the statistical power of the study.
- The study only included recreationally active cyclists and all were men. Therefore the findings cannot be extrapolated to well-trained, competitive cyclists and women. Physiological responses to training have various underlying causal factors, which are attributed to (not only) training status and genetic factors.
- Like most HIIT studies, the interventions in this study only lasted 12 sessions over 6 weeks. Therefore, it is not known whether a longer duration may have provided additional benefits to either HIIT programme, or may have resulted in different findings.

## 6.6 Recommendations for future research

- Future research should provide a better understanding to the submaximal adaptations (training adaptations associated around the point of OBLA/MLSS) following various HIIT models. This knowledge can be used to further specify the prescription of training.

- Future research should also be aimed at the effect that HIIT has on the mechanical efficiency/muscle activation improvements related to a training programme. The software used in this study (Racermate, Seattle, USA) has the capability to measure the pedalling efficiency, however, this data was unavailable during the exercise tests and interval training sessions. This was possibly due to the electromagnetic brake of the hardware to provide a specific resistance (power output). The pedalling efficiency could potentially be combined with an EMG to track the muscle activation improvements.

## 6.7 Conclusion

This study is the first to compare the hemodynamic changes between two different HIIT prescription formats, which were matched for duration of work, work-to-rest ratio and effort of exercise intensity (RPE). The findings provide feasible explanations for the different adaptation pathways for the two methods of HIIT prescription.

The increased utilization of O<sub>2</sub> at submaximal exercise intensity as seen in the long interval HIIT has been previously reported following endurance training. The improvements are attributed to peripheral adaptations. In addition, the findings of improved aerobic performance parameters which favour the short interval HIT programme have also been identified. The improvements identified following the short interval HIT programme were mostly related to central adaptations.

The hemodynamic findings associated with the short interval HIT programme have not yet been reported previously. The present study identified an increased  $\Delta[\text{O}_2\text{Hb}]$  during an incremental exercise test following the short interval HIT programme and support previous literature that this intervention resulted in mostly central physiological adaptations.

The findings of the current study support the previous theory of Buchheit and Laursen (2013a) that if one of the nine facets of HIIT programme design is manipulated, the resulting stimulus for adaptation

is different. This can be seen in the present study, whereby the only factor which was adjusted (interval length), with all other factors controlled. Therefore, the differences in interval length plays an important role in the adaptation pathways.

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# Appendix A

## Health Status Questionnaire

On this questionnaire, a number of questions regarding your physical health are to be answered. Please answer every question as accurately as possible so that a correct assessment can be made. Please mark the space to the left of the question to answer "yes". Leave blank if your answer is "no". Please ask if you have any questions. Your response will be treated in a confidential manner.

Name: \_\_\_\_\_ Date: \_\_\_\_\_

### Medical Screening – ACSM Medical Screening Questionnaire

- Do you have any personal history of heart disease?
- Do you have any personal history of metabolic disease (thyroid, renal, liver)?
- Have you had diabetes for less than 15 years?
- Have you had diabetes for 15 years or more?
- Have you experienced pain or discomfort in your chest apparently due to blood flow deficiency?
- Any unaccustomed shortness of breath (perhaps during light exercise)?
- Have you had any problems with dizziness or fainting?
- Do you have difficulty breathing while standing or sudden breathing problems at night?
- Do you suffer from ankle oedema (swelling of the ankles)?
- Have you experienced a rapid throbbing or fluttering of the heart?
- Have you experienced severe pain in leg muscles during walking?
- Do you have a known heart murmur?
- Do you have any family history of cardiac or pulmonary disease prior to age 55?
- Have you been assessed as hypertensive on at least 2 occasions?
- Has your serum cholesterol been measured at greater than 5.4mmol/l?
- Are you a cigarette smoker?
- Would you characterize your lifestyle as "sedentary"?

### Medical History

Are you currently being treated for high blood pressure?

If you know your average blood pressure, please enter: \_\_\_\_\_/\_\_\_\_\_

Please Check All That Apply.

- |   |   |  |
|---|---|--|
| <input type="checkbox"/> has doctor ever found an abnormal ECG? | <input type="checkbox"/> Limited Range of Motion?   | <input type="checkbox"/> Stroke?                         |
| <input type="checkbox"/> Abnormal Chest X-Ray?                  | <input type="checkbox"/> Recently Broken Bones?     | <input type="checkbox"/> Epilepsy or Seizures?           |
| <input type="checkbox"/> Rheumatic Fever?                       | <input type="checkbox"/> Arthritis?                 | <input type="checkbox"/> chronic Headaches or Migraines? |
| <input type="checkbox"/> Low Blood Pressure?                    | <input type="checkbox"/> Bursitis?                  | <input type="checkbox"/> Persistent Fatigue?             |
| <input type="checkbox"/> Asthma?                                | <input type="checkbox"/> Swollen or Painful Joints? | <input type="checkbox"/> Stomach Problems?               |
| <input type="checkbox"/> Bronchitis?                            | <input type="checkbox"/> Foot Problems?             | <input type="checkbox"/> Hernia?                         |
| <input type="checkbox"/> Emphysema?                             | <input type="checkbox"/> Knee Problems?             | <input type="checkbox"/> Anemia?                         |
| <input type="checkbox"/> Other Lung Problems?                   | <input type="checkbox"/> Back Problems?             | <input type="checkbox"/> Are You Pregnant?               |
|   | <input type="checkbox"/> Shoulder Problems?         |  |

Has a doctor imposed any activity restrictions? If so, please describe:

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## Family History

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Have your mother, father, or siblings suffered from (please select all that apply):

- Heart attack or surgery prior to age 55
- Stroke prior to age 50
- Congenital heart disease or left ventricular hypertrophy
- High cholesterol
- Diabetes
- Obesity
- Hypertension
- Osteoporosis
- Asthma
- Leukemia or cancer prior to age 60

## Medications

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Please Select Any Medications You Are Currently Using

- |   |   |
|---|---|
| <input type="checkbox"/> Diuretics                | <input type="checkbox"/> Other Cardiovascular                       |
| <input type="checkbox"/> Beta Blockers            | <input type="checkbox"/> NSAIDS/Anti-inflammatories (Motrin, Advil) |
| <input type="checkbox"/> Vasodilators             | <input type="checkbox"/> Cholesterol                                |
| <input type="checkbox"/> Alpha Blockers           | <input type="checkbox"/> Diabetes/Insulin                           |
| <input type="checkbox"/> Calcium Channel Blockers | <input type="checkbox"/> Other Drugs (record below).                |

Please list the specific medications that you currently take:

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## Emergency Contacts

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Please list your general practitioner and person to be contacted in case of emergency

Doctor: \_\_\_\_\_ Phone: \_\_\_\_\_

Contact: \_\_\_\_\_ Phone: \_\_\_\_\_

## Activities and Goals

On average, how many times do you exercise per week? \_\_\_\_\_

On average, how long do you exercise? \_\_\_\_\_ minutes

On a scale from 1 to 10, how intense is your typical workout (circle one):

Very Easy **1 2 3 4 5 6 7 8 9 10** Very Intense

For each activity that you participate in, indicate your typical exercise time in minutes per session:

Running/Jogging: _____	Weight Training: _____	Skiing/Boarding: _____
Walking: _____	Aerobics Classes: _____	Yoga/Martial Arts: _____
Stair Climbing: _____	Swimming: _____	Other: _____
Bicycle/Spinning: _____	Racquet Sports: _____	

## Lifestyle

Are you a cigarette smoker? \_\_\_\_\_ If so, how many per day? \_\_\_\_\_

Previously a cigarette smoker? \_\_\_\_\_ If so, when did you quit? \_\_\_\_\_

How many years have you smoked or did you smoke before quitting? \_\_\_\_\_

Do you/did you smoke: \_\_\_\_\_cigarettes? \_\_\_\_\_cigars? \_\_\_\_\_pipe?

Please rate your daily stress levels (circle one):

Low	Moderate	High: I enjoy the challenge	High: sometimes difficult to handle	High: often difficult to handle
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Do you drink alcoholic beverages? \_\_\_\_\_

How many units of alcohol do you consume per week: \_\_\_\_\_ (see Alcohol Units Calculator below)

**Alcohol Units Calculator**

Type of Drink	Units
1 glass of wine	1
1 pub measure of spirits (Gin, Vodka etc.)	1
1 can of beer	1.5
1 bottle of strong lager	2.5
1 can of strong lager	4
1 bottle of wine	7
1 litre bottle of wine	10
1 bottle of fortified wine (port, sherry etc.)	14
1 bottle of spirits	30

Dietary Habits: Please select all that apply

- I seldom consume red or high fat meats
- I pursue a low-fat diet
- I eat at least 5 servings of fruits/vegetables per day
- I almost always eat a full, healthy breakfast
- My diet includes many high-fiber foods
- I rarely eat sugar or high-fat dessert



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## Other

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Please indicate any other medical conditions or activity restrictions that you may have. It is important that this information be as accurate and complete as possible.

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Is any of this information critical to understanding your readiness for exercise? Are there any other restrictions on activity that we should know about?

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Thank you for taking the time to complete this questionnaire!

## Appendix B

### Training History Questionnaire

How long have you been cycling for?	Years	Months
How many Races have you competed in?	10-100km	100km+
Are you currently being coached?	<input type="checkbox"/> Yes	<input type="checkbox"/> No
Please describe a typical week of your current training:	Duration (hours)	Intensity (RPE out of 10)
Monday		
Tuesday		
Wednesday		
Thursday		
Friday		
Saturday		
Sunday		
On average, how many training hours per week (past 6 weeks)?		
On average, how many training hours per week (past 2 weeks)?		
What intensity has your training been over the last 6 weeks?		

(self-developed)

# Appendix C



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## STELLENBOSCH UNIVERSITY CONSENT TO PARTICIPATE IN RESEARCH

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### **Hemodynamic changes following a long and a short interval high intensity cycling intervention.**

You are asked to participate in a research study conducted by Kyle Basson BSc Hons Sports Science (High Performance Sport), from the Sports Science Department at Stellenbosch University. Results will be contributed thesis. You were selected as a possible participant in this study because you are an 18-29 year old male recreational cyclist.

#### **1. PURPOSE OF THE STUDY**

The purpose of the study is to broaden the understanding of the physiological adaptations to HIIT in cycling. This is beneficial for the prescription of specific training.

#### **2. PROCEDURES**

If you volunteer to participate in this study, we would ask you to do the following things:

The study will consist of one week of familiarization testing in the laboratory; six weeks personal training program in the lead up to the Cape Town Cycle Tour (CTCT) period; one week of pre testing; six weeks HIT intervention protocol (Long Intervals or Short Intervals) and one week of post testing.

Testing protocol will include:

- Anthropometric measurements
- Maximal aerobic assessment ( $VO_{2max}$ ) with concurrent NIRS measurements
- Maximal anaerobic test (Wingate 30sec sprint)

Once testing procedures have been completed, the six-week personal program will proceed as follows:

- Sessions to be performed under participants' own discretion
- This phase will be a build up to the Cape Town Cycle Tour

Pre-test will follow the CTCT and the testing protocol will be repeated.

Randomization of the group will occur to assign either the long or short interval training protocol

The six-week long and short interval training intervention will include:

- Two HIIT sessions per week will occur in the lab.
- Training plan will be provided with a training diary for monitoring purposes.

Following HIIT intervention period, Post-test will occur and repeat the testing protocol.

The study on a whole will last around 15 weeks.

### **3. POTENTIAL RISKS AND DISCOMFORTS**

There will be no serious risks involved in the study as all participants will be healthy and physically fit; nonetheless participants may experience dizziness, fainting and discomfort during the exercise tests or HIIT sessions.

The potential risks will be minimized as much as possible by thoroughly explaining the procedure to the participants and carefully monitoring changes in the physiological variables.

Participants will be phoned 6 hours post-test for confirmation of wellbeing.

Blood samples will be obtained non-invasively via a finger prick and will not exceed 2mL per test. Gloves, alcohol swabs and hermitically sterilized needles will be used, all sent for incineration post testing in a biohazard collection bin.

If an injury or adverse event occurs during testing or training the session will be terminated immediately and the participant will receive specific supervision from the researcher who is qualified to perform basic medical aid, as well as a qualified Biokineticist who is on the premises. Mr. Rainsford is also qualified in basic life support (BLS) (0761763292 HPCSA registration no. BK 0019747) and he is able to perform cardiopulmonary resuscitation (CPR) and use an Automated External Defibrillator (AED) (the latter device is in the Sport Physiology Laboratory).

Should any emergency arise, the participant will be stabilized and then immediately transported to the emergency room at Stellenbosch Medi-Clinic.

### **4. POTENTIAL BENEFITS TO SUBJECTS AND/OR TO SOCIETY**

The participant will receive three sets of physiological test protocols for free.

The participant will receive free training and monitoring for the duration of the study, which will benefit his cycling performance.

Expansion of the body of knowledge with regards to the peripheral adaptations associated with two different modalities of HIIT cycling interventions. This knowledge can be used in the tailoring of training programs.

### **5. PAYMENT FOR PARTICIPATION**

The participant will not receive payment for participation in the study.

### **6. CONFIDENTIALITY**

Any information that is obtained in connection with this study and that can be identified with you will remain confidential and will be disclosed only with your permission or as required by law. Confidentiality will be maintained by means of storage on a personal computer under password protection, only Kyle Basson and Elmarie Terblanche will have access to the raw data.

Confidentiality will be kept in research thesis by removal of names associated to the data results and the majority of the results being represented as an average in the group of participants.

## 7. PARTICIPATION AND WITHDRAWAL

You can choose whether to be in this study or not. If you volunteer to be in this study, you may withdraw at any time without consequences of any kind. You may also refuse to answer any questions you don't want to answer and still remain in the study. The investigator may withdraw you from this research if circumstances arise which warrant doing so. Participant will be removed from the study if proposed training is not met, if the subject has abnormalities in hemoglobin readings throughout testing protocol, if the subject attains an injury preventing participation.

## 8. IDENTIFICATION OF INVESTIGATORS

If you have any questions or concerns about the research, please feel free to contact:

Kyle Basson – 0614125473 - 16484258@sun.ac.za

Elmarie Terblanche - 021 913 4217 - et2@sun.ac.za

## 9. RIGHTS OF RESEARCH SUBJECTS

You may withdraw your consent at any time and discontinue participation without penalty. You are not waiving any legal claims, rights or remedies because of your participation in this research study. If you have questions regarding your rights as a research subject, contact Ms Maléne Fouché [mfouche@sun.ac.za; 021 808 4622] at the Division for Research Development.

### SIGNATURE OF RESEARCH SUBJECT OR LEGAL REPRESENTATIVE

The information above was described to me by Kyle Basson in Afrikaans/English and I am in command of this language or it was satisfactorily translated to me. I was given the opportunity to ask questions and these questions were answered to my satisfaction.

I hereby consent voluntarily to participate in this study. I have been given a copy of this form.

\_\_\_\_\_  
**Name of Subject/Participant**

\_\_\_\_\_  
**Name of Legal Representative (if applicable)**

\_\_\_\_\_  
**Signature of Subject/Participant or Legal Representative**

\_\_\_\_\_  
**Date**

### SIGNATURE OF INVESTIGATOR

I declare that I explained the information given in this document to \_\_\_\_\_. He was encouraged and given ample time to ask me any questions. This conversation was conducted in English/Afrikaans.

\_\_\_\_\_  
**Signature of Investigator**

\_\_\_\_\_  
**Date**



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**Hemodinamiese veranderinge in rekreasie fietsryers na 'n lang- en kort-interval hoë intensiteit fietsry intervensie.**

Jy word gevra om deel te neem aan 'n navorsingstudie wat uitgevoer word deur Kyle Basson (BSc Hons Sportwetenskap), in die Departement Sportwetenskap by die Universiteit Stellenbosch. Die resultate sal deel wees van 'n MSc tesis. Jy is as moontlike deelnemer vir die studie gekies omdat jy 'n 18-29 jarige manlike rekreasie fietser is.

**1. DOEL VAN DIE STUDIE**

Die doel van die studie is om ons kennis uit te brei van die fisiologiese aanpassings in fietsryers na 'n hoë intensiteit interval oefenprogram (HIIT). Dit sal afrigters en fietsryers help om hulle oefenprogramme te verbeter.

**2. PROSEDURES**

Indien jy instem om aan die studie deel te neem, vra ons jou om die volgende te doen:

1. Vraelyste oor jou persoonlike inligting en gesondheid te voltooi sodat ons kan vasstel of jy kwalifiseer om aan die studie deel te neem.
2. 'n Ses weke HIIT intervensie protokol te voltooi, wat twee keer per week in die Sportfisiologie Laboratorium onder toesig sal plaasvind. Jy sal in een van twee groepe verdeel word, naamlik 'n langinterval of 'n kort intervalprogram. Elke oefensessie in die laboratorium sal 45 min duur.
3. Die Sportfisiologie Laboratorium op drie geleenthede te besoek vir oefentoetse (voor, tydens en na die oefenfases). Twee oefentoetse sal oor twee dae gedoen word, naamlik 'n maksimale uithouvermoë toets en 'n maksimale anaërobiese toets. Beide toetse sal op 'n fietsergometer in die laboratorium afgelê word. gedurende hierdie oefentoetse sal Klein bloedmonsters via a vingerprik geneem word om bloedlaktaatkonsentrasie te bepaal. Omtrent 15 bloedmonsters sal tydens die twee toetse geneem word en die totale volume van bloed sal nie meer as 2 mL wees nie. Elektrodes sal ook op jou bobene geplak word gedurende die maksimale uithouvermoë toets end it sal gekoppel word aan 'n oppervlak-infrarooi spektrometer. Dit sal ons toelaat om veranderinge in bloedvloei duer jou spiere te meet. Elke besoek aan die laboratorium sal tussen 30 -60 min duur.
4. Aan die Kaapstad Fietstoer deel te neem.
5. Die duur van die studie is 15 weke.

### **3. MOONTLIKE RISIKO'S EN ONGEMAKLIKHEID**

Daar is geen ernstige risiko's in die studie nie. Die oefentoetse en HIIT wat jy gaan aflê mag ongemak, naarheid of duiseligheid veroorsaak, maar dit sal nie anders wees as wat jy tydens 'n harde oefensessie ervaar nie.

Die potensiële risiko's sal soveel as moontlik beperk word deur vir jou 'n deeglik verduideliking van die prosedures te gee en deur veranderinge in jou fisiologiese response tydens die oefentoetse en oefensessies noukeurig te monitor. Jy sal ook 'n telefoonoproep 6 ure na 'n toets ontvang om uit te vind hoe jy voel.

Bloedmonsters sal via 'n vingerprik geneem word en nie meer as 2 mL bloed sal per toets geneem word nie. Handskoene, alkohollappies en nuwe lugdigte gesteriliseerde naalde sal gebruik word vir elke bloedmonster. Hierdie verbruikbare materiaal sal in 'n spesiale "biohazard" blik geplaas word en gestuur word vir verbranding.

As 'n besering of ongewone noodsituasie tydens die toets of die oefening plaasvind sal die sessie onmiddellik beëindig word en jy sal spesifieke behandeling van die navorser ontvang wat bevoeg is om basiese noodhulp uit te voer, asook van 'n gekwalifiseerde biokinetikus (Mnr Rainsford) wat op die perseel beskikbaar is. Mnr Rainsford is ook gekwalifiseerd in die basiese lewensondersteuning (BLS) (0761763292 HPCSA registrasie nr. BK 0019747) en hy is in staat om kardiopulmonêre resussitasie (KPR) uit te voer en 'n Outomatiese Eksterne Defibrillator (AED) te gebruik. (Laasgenoemde toestel is in die Sportfisiologie Laboratorium).

Indien enige noodsituasie opduik, sal jy gestabiliseer word en dan onmiddellik na die ongevalle-afdeling by Stellenbosch Medi-Kliniek geneem word.

### **4. MOONTLIKE VOORDELE VIR PROEFPERSONE EN/OF VIR DIE SAMELEWING**

Jy sal drie stelle fisiologiese toetse gratis ontvang. Jy sal ook 'n gratis oefenprogram ontvang vir die duur van die studie wat jou fietsryprestasie sal verbeter.

Die resultate van hierdie studie sal ons kennis verbreed oor die inoefeningaanpassings wat geassosieer word met die twee verskillende HIIT fietsry intervensies. Hierdie kennis kan gebruik word in die verfyning van inoefeningsprogramme.

### **5. VERGOEDING VIR DEELNAME**

Jy sal nie enige vorm van betaling ontvang vir deelname aan die studie nie.

### **6. VERTROULIKHEID**

Enige inligting wat verkry word in hierdie studie en wat geïdentifiseer kan word met jou sal vertroulik bly en sal slegs bekend gemaak word met jou toestemming of soos deur die wet vereis.

Vertroulikheid sal behou word deur die data op 'n rekenaar met 'n vertroulike wagwoord te stoor. Slegs die navorser en die studieleier sal toegang tot die data hê. Die data sal op die studieleier se rekenaar (met 'n wagwoord) vir 3 jaar na die studie bewaar word. Slegs die studieleier het toegang tot hierdie rekenaar. Indien 'n artikel van die resultate gepubliseer word, sal daar geen melding van name van deelnemers wees nie. Slegs groepresultate sal beskikbaar gestel word.

## 7. DEELNAME EN ONTTREKKING

Jy kan kies of jy wil deel wees van hierdie studie of nie. As jy vrywillig aan die studie deelneem, kan jy enige tyd onttrek sonder gevolge van enige aard. Jy kan weier om sekere vrae te beantwoord en nogsteeds aan die studie deelneem. Die navorser kan jou onttrek van hierdie navorsing indien omstandighede dit vereis. Die volgende omstandighede sal tot beëindiging van jou deelname lei; As jy nie aan die regulasies van die toetse en oefensessies voldoen nie, as jy 'n hemoglobienwaarde het wat buite die normale grense vir gesonde persone is, en as jy 'n besering opdoen wat veroorsaak dat jy nie aan die toetse en oefensessies kan deelneem nie.

## 8. IDENTIFIKASIE VAN ONDERSOEKERS

Indien jy enige vrae of besorgdhede het omtrent die navorsing, staan dit jou vry om in verbinding te tree met:

Kyle Basson – 0614125473 - 16484258@sun.ac.za

Elmarie Terblanche - 021 913 4217 - et2@sun.ac.za

## 9. REGTE VAN PROEFPERSONE

Jy kan te eniger tyd jou inwilliging terugtrek en jou deelname beëindig, sonder enige nadelige gevolge vir jou. Deur deel te neem aan die navorsing doen jy geensins afstand van enige wetlike regte, eise of regsmiddel nie. Indien jy vrae het oor jou regte as proefpersoon by navorsing, skakel met Me Maléne Fouché [mfouche@sun.ac.za; 021 808 4622] van die Afdeling Navorsingsontwikkeling.

### VERKLARING DEUR PROEFPERSOON OF SY/HAAR REGSVERTENWOORDIGER

Die bostaande inligting is aan my, \_\_\_\_\_ gegee en verduidelik deur Kyle Basson in Afrikaans/English en ek is dié taal magtig. Ek is die geleentheid gebied om vrae te stel en my vrae is tot my bevrediging beantwoord.

Ek bevestig dat ek hiermee vrywillig aan die studie deelneem. 'n Afskrif van hierdie vorm is aan my gegee.

\_\_\_\_\_  
**Naam van proefpersoon/deelnemer**

\_\_\_\_\_  
**Naam van regsverteenvoordiger (indien van toepassing)**

\_\_\_\_\_  
**Handtekening van proefpersoon/deelnemer of regsverteenvoordiger**      **Datum**

### VERKLARING DEUR ONDERSOEKER

Ek verklaar dat ek die inligting in hierdie dokument vervaar verduidelik het aan \_\_\_\_\_ . Hy is aangemoedig en oorgenoeg tyd gegee om vrae aan my te stel. Dié gesprek is in Afrikaans/Engels gevoer.

\_\_\_\_\_  
**Handtekening van ondersoeker**

\_\_\_\_\_  
**Datum**





## Appendix E

<b>Short Interval HIT session</b>			
	<b>10 min warm up</b>	<b>4-5 RPE</b>	
	<b>30 s</b>	<b>8-10 RPE</b>	<b>x 11</b>
	<b>15 s</b>	<b>6-7 RPE</b>	<b>x 11</b>
	<b>3 min</b>	<b>1-2 RPE</b>	
	<b>30 s</b>	<b>8-10 RPE</b>	<b>x 11</b>
	<b>15 s</b>	<b>6-7 RPE</b>	<b>x 11</b>
	<b>3 min</b>	<b>1-2 RPE</b>	
	<b>30 s</b>	<b>8-10 RPE</b>	<b>x 10</b>
	<b>15 s</b>	<b>6-7 RPE</b>	<b>x 10</b>
	<b>10 min cool down</b>	<b>4-5 RPE</b>	

Adapted short interval HIT (Rønnestad *et al.*, 2015)

## Appendix F

<b>Long Interval HIT session</b>		
	<b>10 min warm up</b>	<b>4-5 RPE</b>
	<b>4 min</b>	<b>8-10 RPE</b>
	<b>2 min</b>	<b>6-7 RPE</b>
	<b>4 min</b>	<b>8-10 RPE</b>
	<b>2 min</b>	<b>6-7 RPE</b>
	<b>4 min</b>	<b>8-10 RPE</b>
	<b>2 min</b>	<b>6-7 RPE</b>
	<b>4 min</b>	<b>8-10 RPE</b>
	<b>10 min cool down</b>	<b>4-5 RPE</b>

Adapted long interval HIT (Rønnestad *et al.*, 2015)

## Appendix G

<b>1 – 10 RPE Scale</b>		
0	Rest	
1	Extremely Easy	Restful breathing, can sing
2	Very Easy	Can talk in complete sentences
3	Easy	Can maintain for hours
4	Moderate	Talking first becomes broken
5	Somewhat Hard	Heavier breathing begins
6	Moderately Hard	Deep breaths, talking is avoided
7 (~LT)	Hard	Deep forceful breathing (but still sustainable)
8	Very Hard	Labored, cannot talk, cannot maintain beyond a few minutes
9	Very, very hard	Very labored, breathless, can only hold ~1 min
10	<b>Extremely Hard – max!</b>	<b>Gasping for air, 5-20 second maximum</b>

1-10 RPE Borg Scale (Indoor Cycling Association, 2015)



