The impact of recovery interval duration and intensity on the acute physiological responses to interval training during cycling exercise

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A dissertation submitted in partial fulfillment of the requirements for the degree of Master of Science

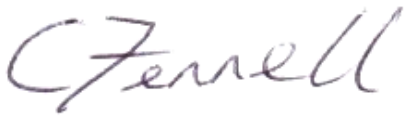
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Declaration

'No part of this thesis has been submitted in support of an application for any degree or other qualification of the University of Kent, or any other University or Institution of learning'.

Signed 

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List of Abbreviations and Symbols

ACT	Active recovery intensity
ANOVA	Analysis of variance
ATP	Adenosine triphosphate
B[La]	Blood lactate concentration ($mmol^{-1}$)
Bf	Breaths per minute ($L.min^{-1}$)
BP	Blood pressure
CV	Coefficient of variation (%)
CO_2	Carbon dioxide
CONT	Continuous low intensity training
H^+	Hydrogen ions
HHb	Deoxyhaemoglobin
HIIT	High intensity interval training
HR	Heart rate ($beats.min^{-1}$)
HRmax	Maximal minute heart rate ($beats.min^{-1}$)
IND	Individualised recovery duration
LT	Lactate threshold (W)
LTP	Lactate turnpoint (W)
M[La]	Muscle lactate concentration ($mmol^{-1}$)

MMP	Maximal minute power (W)
$m\dot{V}O_2$	Muscle oxygen consumption ($ml.O_2.min^{-1}.100g^{-1}$)
NIRS	Near-infrared spectroscopy
O_2	Oxygen
O_2Hb	Oxyhaemoglobin
O_2Hbmax	100% muscle oxygenation after arterial occlusion
O_2Hbmin	0% muscle oxygenation during arterial occlusion
PA	Passive recovery intensity
PCr	Phosphocreatine
PO	Power output (W)
$p\dot{V}O_{2max}$	Power output at $\dot{V}O_{2max}$ (W)
RCT	Respiratory compensation threshold
RER	Respiratory exchange ratio
RPE	Rating of perceived exertion (Borg 6 – 20)
sRPE	Session rating of perceived exertion (0 – 10)
STD	Standardised recovery duration
T@HRmax	Time at maximal minute heart rate
$T@\dot{V}O_{2max}$	Time at maximal oxygen consumption
tHb	Total haemoglobin
TSI %	Tissue saturation index percentage

$\dot{V}CO_2$	Volume of expired carbon dioxide ($L.min^{-1}$)
$\dot{V}_E$	Pulmonary ventilation ($L.min^{-1}$)
VL	Vastus lateralis muscle
$\dot{V}O_2$	Pulmonary oxygen uptake ($L.min^{-1}$)
$\dot{V}O_{2max}$	Maximal oxygen consumption ($L.min^{-1}$ / $ml.kg.min^{-1}$)
$\dot{V}O_{2peak}$	Peak oxygen consumption ($L.min^{-1}$ / $ml.kg.min^{-1}$)
$v\dot{V}O_{2max}$	Velocity at $\dot{V}O_{2max}$
80A	Active recovery at 80% of power output at the LT
110A	Active recovery at 110% of power output at the LT

Abstract

The current thesis aimed to investigate the impact of the recovery interval duration and intensity on the acute physiological and perceptual responses to high intensity interval training (HIIT) during cycling exercise. Two studies were completed to examine the effects of the recovery interval duration and intensity in isolation. In *Study One*, sixteen participants completed a 6 x 4-min and 3 x 8-min HIIT session twice, with a standardised (STD) and individualised (IND) recovery duration based upon a resolution of muscle oxygen consumption ($m\dot{V}O_2$) to pre-exercise levels. In *Study Two*, fourteen participants completed a 6 x 4-min and 3 x 8-min HIIT session three times, with a passive (PA) and two active (ACT) recovery intensities. Results of *Study One* found there were no significant differences between the IND and STD recovery durations for any of the physiological or performance parameters assessed. *Study Two* results demonstrated that ACT recovery intensities increased the overall accumulation of central and peripheral physiological stress, without increasing the total training time commitment of the HIIT session, when compared to PA recovery intensity. Recovery intensity did not affect the time spent above 90 and 95% of $\dot{V}O_{2max}$ during the HIIT sessions. In conclusion, full recovery of $m\dot{V}O_2$ and a return of the exercising muscle to metabolic homeostasis may *not* be required to maintain work interval performance and to generate the desired acute physiological responses during HIIT. Moreover, evidence within this thesis highlights the importance of the optimisation of the recovery interval components to the specific individual and HIIT protocol when seeking to maximising the training stimulus, and time efficiency of the training session. However, at present, the 2:1 work recovery ratio and a moderate ACT recovery intensity appear to be the most practical recovery component prescription when programming the recovery intervals during long work interval HIIT across a broad range of individuals.

I. Introduction

HIIT forms an integral part of a successful endurance athletes training programme and has therefore been the focus of many exercise physiologists research since the late 1950s. HIIT can be simply described as short periods of high intensity work, separated by brief periods of low intensity work or complete inactivity (Laursen & Jenkins, 2002). Its discontinuous nature, by design, allows for the accumulation of a greater amount of time exercising in the heavy to severe intensity domains (i.e. above critical power, the lactate steady state or ≥ 90% of maximal oxygen consumption [$\dot{V}O_{2max}$], also known as the 'red zone'; Poole et al., 2016; Buchheit & Laursen, 2013), than could be achieved during a single bout of continuous exercise at the same intensity until exhaustion (MacDougall & Sale, 1981).

HIIT has been shown to produce a potent stimulus for driving central and peripheral endurance adaptations (MacInnis & Gibala, 2017). The performance benefits of HIIT alone are particularly powerful in untrained and recreationally active individuals (Milanovic et al., 2016), while highly trained athletes can also further enhance endurance performance by undertaking relatively short periods of HIIT (Hawley et al., 1997; Iaia & Bangsbo, 2010; Laursen, 2010). However, there has been a long-standing debate surrounding the superiority of HIIT versus continuous low intensity training (CONT). While HIIT is known to be a highly effective and more time efficient, both training methods have been shown to result in similar physiological adaptations and are likely interdependent (MacInnis & Gibala. 2017: Seiler, 2010).

The ultimate aim of endurance training is to elicit an overload stimulus that generates specific molecular responses which enhance the adaptive phenotype (Coffey & Hawley, 2007). The training stimulus is the sum of three key components: volume (duration), intensity and frequency of sessions (Hawley, 2002). While HIIT fulfils the intensity component, CONT increases the volume of training that can be performed in a certain training cycle. Undertaking large volumes of HIIT would likely result in excessive fatigue, which in turn would reduce the intensity and effectiveness of the HIIT sessions being performed. Hence the importance of CONT, which not only provides a training stimulus but also allows for recovery between HIIT sessions.

The multivariate equation of HIIT programming contains six main components: work interval intensity, work interval duration, number of work intervals, recovery interval intensity, recovery interval duration, and overall session load (overall session load being determined by the five preceding HIIT components; Tschakert & Hofmann, 2013). Each component can be individually or simultaneously manipulated to alter the acute physiological response of the HIIT session. This ability to almost infinitely adjust HIIT has resulted in a large increase in the diversity of HIIT protocols applied in the scientific literature and has made the acute physiological responses difficult to predict (Buchheit & Laursen, 2013). None the less, the main concerted goal of researchers has been to optimise HIIT prescription, in order to maximise the acute training stimulus and time efficiency of the specific session.

The manipulation of the work interval components has been the predominant focus of HIIT research, as this is where the training stimulus is ultimately generated (Buchheit & Laursen, 2013; Buchheit & Laursen, 2013b; Laursen & Jenkins, 2002; MacInnis & Gibala, 2017;

Tschakert & Hofmann, 2013). However, the understanding of how the recovery interval components effects HIIT performance is also important when looking to programme an effective HIIT session. It is therefore surprising that the recovery interval components have received comparatively limited attention in sports science research, despite forming a significant element of HIIT programming. Although the work interval is of great importance to the overall training stress produced by a HIIT protocol, optimal work interval performance can only be achieved if separated by adequate recovery (Schoenmakers et al., 2019). There is considerable diversity in recovery interval research (*Tables 2.1 & 2.2*), however it is clear that the recovery interval is integral to HIIT performance, as inadequate recovery will negatively affect the performance of the work intervals. While an excessive recovery will negatively affect the time efficiency of the HIIT session.

Through reviewing the available research (see II. Literature Review), it was evident that there were several significant gaps in the literature. Firstly, there are currently no effective methods based on physiological rationale, that allows for the individualisation of the recovery interval duration (see section II.IV – The recovery interval: Duration). Secondly, many of the studies which have examined recovery interval intensity during cycling based HIIT using long work intervals (≥ 1-min) have used experimental designs which are not reflective of the type of HIIT sessions used in practice (see section II.V – The recovery interval: Intensity). Finally, there has been paucity in recovery interval research investigating cycling-based exercise using long work interval HIIT protocols, similar to training sessions used by coaches and athletes (i.e. ecologically valid HIIT protocols). The overall aim of this thesis was to address the aforementioned gaps in the literature and in doing so investigate the effect of the recovery interval duration and intensity on the acute physiological and perceptual responses to interval training during cycling exercise.

II. Literature Review

II.I – History of HIIT

HIIT is not a recent phenomenon, with athletes incorporating some form of HIIT into their training programmes since the early 20th century (Billat, 2001). Olympians, Hannes Kolehmainen (3 x Olympic Champion 1912) and Pavoo Nurmi (9 x Olympic Champion 1920 – 1928) both incorporated interval training into their programmes, allowing them to train at velocities near their competition velocity. Triple Olympic Champion (1952), the famous Czechoslovakian runner Emil Zatopek popularised interval training and was reported to undertake training sessions such as 100 x 400m runs with 200m recoveries per day (Billat, 2001). Interval training was first reported in a scientific journal by Reindell & Roskamm, (1959). Then in 1960, Swedish researchers Astrand and colleagues published several papers on the acute physiological responses during interval training and continuous exercise (Astrand et al., 1960), following on from the pioneering work of A.V. Hill in the 1920s (Hill & Lutpon, 1923).

Over recent decades, HIIT research has been increasingly applied to a broad population of recreationally active individuals, diseased / rehabilitation patients, endurance athletes and team sport players (Gibala et al., 2012; Burgomaster et al., 2005; Spencer et al., 2005; Glaister, 2005; Buchheit & Laursen, 2013; Helgerud et al., 2007; Wisloff et al., 2007). As the interest in HIIT research has grown, so too has the diversity of the HIIT regimes applied. This has in turn increased the complexity of how each HIIT component impacts the acute physiological responses of the specific HIIT protocols, while simultaneously making acute

physiological responses more difficult to predict (Buchheit & Laursen, 2013). Throughout the following literature review all forms of interval training will be referred to as 'HIIT' regardless of the work interval duration used in the HIIT protocol. The duration of the work intervals within the study being discussed will be made clear if relevant (i.e. long HIIT ≥ 1-min; short HIIT ≥ 30-s to < 1-min; sprint 4-s to < 30-s).

II.II – Purpose of HIIT

HIIT is an intermittent mode of endurance training, characterised by short high intensity work intervals (i.e. 4-s to ≥ 10-min) generally performed in the severe intensity domain or above the anaerobic threshold (Buchheit & Laursen, 2013). The work intervals are separated by brief periods of low intensity work or complete inactivity that allows for partial, but not necessarily full recovery (Laursen & Jenkins, 2002). The reason HIIT is an effective training method is because it increases the duration of high intensity exercise performed in a single session, then could be achieved during a single bout of continuous exercise at the same intensity until exhaustion (MacDougall & Sale, 1981). This is important because there is strong evidence that the performance of exercise at higher intensities elicits a greater activation of the signalling pathways associated with mitochondrial biogenesis (such as: phosphorylation of AMPK and p38 MAPK and the expression of PGC-1α mRNA; Gibala et al., 2009; Little et al., 2011; Metcalfe et al., 2015), when compared with low to moderate intensity exercise (MacInnis & Gibala, 2017). Chronic activation of these pathways leads to an increase in mitochondrial density (Coffey & Hawley, 2007), a key physiological adaptation for improving aerobic energy metabolism.

Research has also shown HIIT to be more effective than CONT for improving $\dot{V}O_{2max}$ in healthy adults, regardless of whether training volume was matched or not (Milanovic et al., 2016). The meta-analyses from Bacon et al., (2013) and Weston et al., (2014) also report HIIT to be more effective than work matched CONT for improving $\dot{V}O_{2max.}$ It should be noted that the magnitude of improvement in $\dot{V}O_{2max}$ from undertaking HIIT is highly variable and population dependent (i.e. training status, age, gender; Bouchard & Rankinen, 2001; Vollaard, 2009). While HIIT has been shown to be a powerful stimulus for eliciting

improvements in mitochondrial content and $\dot{V}O_{2max}$, its importance has not been established for many other key physiological endurance adaptations such as: increase in skeletal muscle capillary density, maximum stroke volume, cardiac output and total blood volume (MacInnis & Gibala, 2017).

The comparison of HIIT and CONT has been the focus of many studies, due to findings suggesting HIIT provides a superior training stimulus when compared to CONT (MacInnis & Gibala, 2017). However, the superiority of HIIT over CONT is largely associated with the training status of the individuals. Short term HIIT alone offers untrained and recreationally active individuals a potent stimulus for improving endurance performance, with a considerably lower training volume and time commitment (Milanovic et al., 2016). While there is also evidence to suggest that highly trained athletes can further enhance endurance performance by undertaking relatively short periods of HIIT, within their current training programmes (Hawley et al., 1997; Iaia & Bangsbo, 2010; Laursen, 2010).

A detailed discussion about the ongoing debate surrounding HIIT versus CONT is beyond the scope of the current literature review. However, the idea that HIIT and CONT have a dichotomous physiological effect is likely exaggerated, as both endurance training methods have been shown to result in similar physiological adaptations and are likely interdependent (MacInnis & Gibala, 2017). One proposed method for training intensity distribution is a ratio of approximately 80:20 of CONT to HIIT, which has been shown to be an effective training periodisation model for endurance athletes (Seiler, 2010). Moreover, research examining the training characteristics of elite endurance athletes appears to converge on the 80:20 (CONT:HIIT) training intensity distribution (Seiler, 2010).

The future direction of endurance training and more specifically HIIT research should focus on how to maximise the acute physiological and adaptive signalling response of HIIT. Research has shown that a diverse range of HIIT protocols are able to induce beneficial training effects, and therefore it could be assumed that an accurate or individualised prescription of HIIT may not be relevant (Tschakert & Hofmann, 2013). While a laissez-faire approach to HIIT prescription may work for untrained or recreationally active individuals, such an approach would be unlikely to prove beneficial to highly trained endurance athletes. The path to be able to fully optimise HIIT to a specific individual will require innovative thinking and progressive research techniques. The following section (II.III – Components of HIIT) briefly outlines the multiple interconnected components of the multivariate equation that is HIIT programming.

II.III – Components of HIIT

There are six main components of HIIT programming: work interval intensity, work interval duration, number of work intervals, recovery interval intensity, recovery interval duration, and overall session load (overall session load being determined by the five work and recovery components; Tschakert & Hofmann, 2013). However, in some cases HIIT sessions can also be broken down further into nine components. These nine components incorporate the six main components in addition to: number of series of repetitions, between series recovery duration and between series recovery intensity (Buchheit & Laursen, 2013). The specific prescription of the multiple components strongly influences the acute physiological response of the HIIT session. Moreover, all of the components are interrelated and as such the manipulation of any one component impacts on the other components and consequently the overall session outcome.

By manipulating each component, researchers have sought to optimise HIIT prescription, in order to maximise the acute training stimulus of the session (Buchheit & Laursen, 2013; Buchheit & Laursen, 2013b; Tschakert & Hofmann, 2013). While our understanding of HIIT programming and prescription has improved, very little is known about how HIIT can be individualised to a specific athlete to maximise their acute physiological responses to the session. It is easy to prescribe the optimal HIIT session to an athlete, based the mean response of a large study cohort. However, it is unlikely that the athlete in question will produce the same session response as the group mean, due to individual differences in training response (Mann et al., 2014).

The predominant focus of HIIT research has been on the manipulation of the work interval components, as this is where the majority of the training stimulus is generated (See the following reviews for a full overview: Buchheit & Laursen, 2013; Buchheit & Laursen, 2013b; Laursen & Jenkins, 2002; MacInnis & Gibala, 2017; Tschakert & Hofmann, 2013). Research investigating HIIT reports a broad range of prescriptions for work interval duration, with durations from 4-s to ≥ 10-min. The duration and number of work intervals within a session ultimately influences the highest sustainable work interval intensity. Researchers use several methods for prescribing work interval intensity: fixed percentages of the power at $\dot{V}O_{2max}$ ($p\dot{V}O_{2max}$), velocity at $\dot{V}O_{2max}$ ($v\dot{V}O_{2max}$) or maximal minute heart rate (HRmax; Buchheit & Laursen, 2013), percentage of time to exhaustion (50 to 70%) while exercising at $v\dot{V}O_{2max}$ or $p\dot{V}O_{2max}$ (Billat, 2001; Laursen & Jenkins, 2002; Laursen et al., 2004; Smith et al., 1999; Smith et al., 2003), power output (PO) zones based on functional threshold power (Allen & Coggan, 2010) and self-paced 'maximal session effort' or maximal sustainable velocity (Laurent et al., 2014; Seiler & Hetlelid, 2005; Seiler et al., 2013; Smilios et al., 2017). Combined the work interval components determine most of the physiological stress produced during the HIIT session.

The work interval components maybe important in facilitating the training stimulus, however optimal HIIT session performance (i.e. achieving the greatest training stimulus for the specific HIIT session) can only be achieved if adequate recovery separates the work intervals. If there is an imbalance between the demands of the work interval and the recovery provided, this can lead to HIIT sessions that are too hard to complete (Laursen et al., 2002), or HIIT sessions that are too easy (Smilios et al., 2017). Therefore, when designing HIIT protocols, it is important that all the components are considered to ensure the greatest training stimulus is achieved for the specific HIIT protocol.

The recovery interval components have been a relatively neglected part of HIIT research, in comparison to the work interval components. The aim of the two studies contained within this thesis (V. Experimental Chapter – Study One and VI. Experimental Chapter – Study Two) is to advance the available scientific knowledge regarding the recovery interval components. Therefore, the following sections (II.IV and II.V) and the remainder of the literature review, will provide a comprehensive review of the current literature surrounding the areas of recovery interval duration and intensity.

II.IV – The recovery interval: Duration

The effects of the recovery interval duration on subsequent work interval performance has received limited attention in sports science research. Despite the recovery interval forming a significant element in the multivariate equation of HIIT programming, the predominant focus of HIIT research has been on the manipulation and optimisation of the work interval (see relevant review papers for further insight: Buchheit & Laursen, 2013; Laursen & Jenkins, 2002; MacInnis & Gibala, 2016; Tschakert & Hofmann, 2013). None the less, the duration of the recovery interval has been the interest of several studies using different HIIT protocols and exercise modalities (*Table 2.1*). Table 2.1 contains a comprehensive list of research papers which have examined the acute physiological effects of recovery interval duration on HIIT performance.

II.IV.1 – Literature search strategy

Electronic database searches were performed using PubMed, Google Scholar and ResearchGate. The search terms covered the areas of high intensity interval training and recovery interval duration, using a combination of the following key words: high-intensity interval training, interval training, sprint training, recovery interval, recovery duration, intermittent exercise. Relevant papers were collated in Mendeley. Reference lists of collated papers were examined for other eligible papers. Electronic searches of the papers which had cited relevant papers were also made to identify other eligible papers.

II.IV.2 – Literature inclusion criteria

1) Examining the acute physiological effects of recovery interval duration on interval training performance.

2) Protocols applied must be formed of ≥ 2 repeated intervals.

3) Studies must be investigating between work interval recovery intensity, NOT recovery intensity after a training session.

4) Study participants must be healthy individuals, free from injury or disease.

Twenty-nine papers met the inclusion criteria.

Table 2.1 – Summary of studies which have investigated the acute physiological effects of recovery interval duration.

Study	Sample size Age (years) VO2max (ml.kg.min^{-1})	Exercise modality	HIIT protocol (work INT intensity)	Recovery duration (recovery intensity)	Optimum	Key study findings
Long HIIT (≥ 1-min)						
Seiler & Hetlelid, (2005)	N = 9 30 ± 4 72 ± 5	Running	6 x 4-min (SP)	1-min; 2-min; 4-min; SS (ACT - SP)	2:1	Higher running velocity in 2-min (85% v$\dot{V}O_{2max}$) and 4-min (84% v$\dot{V}O_{2max}$) compared to 1-min (83% v$\dot{V}O_{2max}$). Work $\dot{V}O_2$ higher in 2-min condition, compared to 1-min and 4-min. End RPE higher in 2-min condition versus 4-min condition. No difference in peak B[La] and peak HR responses between conditions.
Smilios et al., (2017)	N = 11 22 ± 1 52 ± 4	Running	4 x 4-min (90% of MAV)	2-min; 3-min; 4-min (ACT - 35% of MAV)	2:1	Time above 80 and 90% of HRmax sig. longer with 2-min and 3-min recovery, compared to 4-min. B[La] and RPE sig. higher after 2-min, when compared to 4-min. No sig, difference in B[La] and RPE between 3-min and 4-min. No sig. in percentage of $\dot{V}O_{2max}$ attained and total exercise time above 80, 90 and 95% $\dot{V}O_{2max}$.
Laurent et al., (2014)	N = 16 21 ± 2 61 ± 5	Running	6 x 4-min (SP)	1-min; 2-min; 4-min (ACT 4.8-kph)	2:1	Extending recovery duration increased the SS running velocity. No sig. difference in $\dot{V}O_2$, HR, B[La] and RPE between all recovery durations.
Schoenmakers & Reed, (2018)	N = 12 34 ± 11 53 ± 7	Running	6 x 4-min (SP)	1-min; 2-min; 3-min, SS (ACT - SP)	4:3	Sig. higher running velocity in 3-min, compared with all other conditions, and higher in SS versus 2-min. No sig. difference between all conditions in RPE reported and time spent ≥ 90 and 95% $\dot{V}O_{2max}$ or HRmax.
Zavorsky et al., (1998)	N = 12 25 ± 5 73 ± 4	Running	10 x 400m runs (96% v$\dot{V}O_{2max}$)	60-s; 120-s; & 180-s (ACT – N/S)	1-min	Mean HR sig. higher in 1-min, but no sig. differences in peak HR and $\dot{V}O_2$ between conditions. Sig. increases in RPE with decreasing recovery duration.
Edwards et al., (2011)	N = 11 27 ± 7 64 ± 4	Running	5 x 1000m runs (RPE 17)	2 x SS-PR; HR130; 1:1 W:R (Steady walking pace)	HR130	Sig. shorter recovery duration in the HR130 condition, resulting in a sig. slower running velocity and greater fatigue index, when compared to all other conditions. No sig. difference in HR and end B[La] across all conditions.

Table 2.1 – continued

Study	Sample size Age (years) VO2max ($ml.kg.min^{-1}$)	Exercise modality	HIIT protocol (work INT intensity)	Recovery duration (recovery intensity)	Optimum	Key study findings
Edge et al., (2013)	N = 5 21 ± 2	Cycling	6 x 2-min (92% of P@$\dot{V}O_{2max}$)	1-min; 3-min	1-min	Mean INT HR higher in 1-min, compared to 3-min. Sig. higher end B[La], H^+, and M[La] content during 1-min, then 3-min. Muscle PCr lower after 1-min.
Mavrommataki et al., (2006)	N = 9 16 ± 1	Rowing	2 x 1000m rows (all-out max effort)	1.5-min; 3-min; 6-min (PA)	6-min	Mean PO of the 2nd INT was sig. higher in the 6-min, compared to the 1.5-min, but not 3-min. Mean HR of 2nd INT sig. higher in 1.5-min, compared to 3-min and 6-min. No sig. in peak HR across INT and recovery conditions.
McLean et al., (2016)	N = 12 21 ± 3 64 ± 7	Running (Field based)	6 x 2-min (SSG format)	30-s; 120-s	30-s	120-s resulted in a sig. greater decrease in HHb and HR during recovery INT, when compared to 30-s. No sig. difference in HHb, HR, RPE, running velocity and distance, between conditions during the 2-min exercise bouts.
Koklu et al., (2015)	N = 12 15 ± 0.5	Running (Field based)	4 x 4-min (SSG format)	1-min; 2-min; 3-min; 4-min (PA)	1-min	%HRmax achieved during the session was sig. higher in the 1-min condition, when compared to 3-min and 4-min. No sig, difference in B[La] and RPE across all recovery conditions. No sig. difference in total distance (m) covered during the SSG across all recovery conditions.
Short HIIT (≥ 30-s to < 1-min)						
McEwan et al., (2018)	N = 14 30 ± 7 54 ± 8	Running	12 x 30-s (105% of MAS)	30-s; SS [51 ± 15-s] (PA)	SS	Mean recovery duration sig. longer in SS condition. Relative time ≥ 105% MAS and men running velocity sig. greater in SS. Time ≥ 90% HRmax higher in 30-s, compared to SS. No sig. differences in end B[La] and RPE between conditions.
Gosselin et al., (2012)	N = 8 23 ± 2	Running	60-s work intervals (Target intensity of 90% of $\dot{V}O_{2max}$)	30-s; 1-min	2:1	Mean and peak $\dot{V}O_2$ and HR sig. higher in 30-s compared to 1-min. No sig. differences in RPE between conditions. Both recovery conditions failed to achieve the target intensity of 90% of $\dot{V}O_{2ma}$.

Table 2.1 – continued

Study	Sample size Age (years) VO2max ($ml.kg.min^{-1}$)	Exercise modality	HIIT protocol (work INT intensity)	Recovery duration (recovery intensity)	Optimum	Key study findings
Hazell et al., (2010)	N = 48 24 ± 3	Cycling	G1 = 4 to 6 x 30-s G2 & G3 = 4 to 6 x 10-s (maximal effort)	G1 = 4-min; G2 = 4-min; G3 = 2-min	4-min	Mean and PPO in sprints higher in G2 and G3. G1 performed more total work.
Ainsworth et al., (1993)	N = 16 25 ± 5 68 ± 4	Cycling	2 x 45-s (maximal effort)	6-min; 9-min; 12-min (80rpm at 9.8-N resistance)	6-min	Mean PO of 2nd 45-s sprint sig. lower after 6-min, when compared to 9-min and 12-min conditions. No sig. differences in 5-s PPO achieved between all conditions. Decrease in PO from 1st to 2nd sprint sig. greater after 6-min and 9-min, when compared to 12-min. No sig. in end B[La] between all conditions.
Toubekis et al., (2005)	N = 16 21 ± 1	Swimming	8 x 25-m sprints (maximal effort)	45-s; 2-min (PA & ACT60 60% of 100m velocity)	2-min	Mean swimming velocity sig. faster in 2-min, compared to 45-s. No sig. in end B[La]. 50-m sprint times 2.4% faster in 2-min condition versus 45-s.
Sprint interval training (4-s to < 30-s)						
Brownstein et al., (2018)	N = 14 12 ± 0.4	Running	10 x 30-m (5-s) sprints (maximal effort)	30-s; SS	30-s	SS recovery duration sig. shorter (12s) than the fixed 30-s condition. Mean sprint time sig. faster in 30-s. Smaller performance decrement in the 30-s condition compared to SS. Mean and peak HR higher in SS.
Gibson et al., (2017)	N = 11 14 ± 1	Running	10 x 30-m (5-s) sprints (maximal effort)	30-s; SS	30-s	HIIT protocol shorter in SS, as SS recovery duration sig. shorter (10-s) than 30-s. Mean sprint time sig. faster in 30-s condition, compared to SS. No sig. differences in peak HR, B[La] and RPE.
Padulo et al., (2015)	N = 17 16 ± 0	Running	6 x 40-m (6-s) sprints (maximal effort)	15-s; 20-s; 25-s	25-s	Total sprint times were 3% faster in 25-s condition compared to 15-s and 1.3% faster compared to 20-s. B[La] and fatigue index were highest in 15-s and lowest in the 25-s.

Table 2.1 – continued

Study	Sample size Age (years) VO2max ($ml.kg.min^{-1}$)	Exercise modality	HIIT protocol (work INT intensity)	Recovery duration (recovery intensity)	Optimum	Key study findings
Iaia et al., (2015)	N = 13 19 ± 1	Running	6 to 8 x 20-s sprints (maximal effort)	40-s; 2-min	2-min	Mean running velocity were higher in 2-min, compared to 40-s. There was a smaller decrement in speed across subsequent sprints in the 2-min, compared to 40-s.
Baker et al., (2007)	N = 8 27 ± 8	Cycling	8 x 6-s sprints (maximal effort)	30-s; 60-s (PA)	30-s & 60-s	PPO sig. higher in 60-s, compared to 30-s. Sig. higher HR during the sprints in the 30-s compared to 60-s. No sig. in RPE and end B[La] between 30-s and 60-s.
Billaut et al, (2003)	N = 20 23 ± 2	Cycling	4 sets of 2 x 8-s sprints (maximal effort)	15-s; 30-s; 60-s; 120-s (PA)	≥ 30-s	Decrease in peak PO and total work of 2nd sprint only sig. in 15-s condition. Sprint performance maintained when 30-s, 60-s and 120-s recovery applied.
Lee et al., (2012)	N = 14 19 ± 1	Cycling	12 x 4-s sprints (maximal effort)	20-s; 90-s (ACT 50W, 60 – 70rpm)	90-s	PPO and mean PO of the sprints were sig. higher in 90-s, compared to 20-s, with a lower RPE and fatigue index. End B[La] higher in 20-s.
Ohya et al., (2013)	N = 8 26 ± 3 51 ± 6	Cycling	10 x 5-s sprints (maximal effort)	25-s, 50-s; 100-s (PA; ACT 40% $\dot{V}O_{2max}$)	≥ 25-s	PPO and mean PO decrement over the sprints lowest in 100-s. Mean $\dot{V}O_2$ and B[La] were highest in 25-s and 50-s, compared to the 100-s. Muscle reoxygenation lower in 25-s when compared to 50-s and 100-s.
Phillips et al., (2014)	N = 14 25 ± 5	Cycling	10 x 6-s sprints (SP)	SS; RR (10% less than SS time)	RR	No sig. difference in mean PO, PPO, HR, fatigue index score and RPE between recovery conditions.

Table 2.1 – continued

Study	Sample size Age (years) VO2max ($ml.kg.min^{-1}$)	Exercise modality	HIIT protocol (work INT intensity)	Recovery duration (recovery intensity)	Optimum	Key study findings
Glaister et al., (2005)	N = 25 21 ± 2	Cycling	20 x 5-s sprints (maximal effort)	10-s; 30-s	10-s	PPO and mean PO of the sprints were higher in the 30-s condition, compared to 10-s. Fatigue index, RPE and end B[La] were lower in the 30-s, compared to 10-s. Work and recovery INT, $\dot{V}O_2$, RER and HR were higher in the 10-s, compared to 30-s.
Kavaliauskas et al., (2015)	N = 32 39 ± 8	Cycling	6 x 10-s (maximal efforts) 2 weeks, 6 sessions	*G1* = 30-s *G2* = 80-s *G3* = 120-s	30-s	Mean HR was higher in G1, when compared to G3 for all training sessions. Mean HR was higher in G2, when compared to G3 for sessions 1 and 2.
Cooke & Barnes, (1997)	N = 11 24 ± 3	Cycling	2 x maximal sprints to exhaustion (9 to 15-s)	30-s; 60-s; 90-s; 120-s (PA)	120-s	PPO of 2nd sprints were sig. lower after 30-s, 60-s and 90-s recovery. Only 120-s recovery allowed for similar PPO across both sprints.
Shi et al., (2018)	N = 13 26 ± 6 61 ± 9	Cycling	40 x 6-s sprints (maximal effort, resistance 7.5% body weight)	15-s; 30-s; 60-s (PA)	15-s	PPO and mean PO were higher in 60-s, compared to 15-s and 30-s. RPE sig. higher in 15-s, compared to 60-s. Accumulated exercise time ≥ 80, 85, 90, 95 and 100% $\dot{V}O_{2max}$ sig. greater in 15-s, compared to all other conditions. Accumulated exercise time ≥ 90, 95 and 100% HRmax sig. greater in 15-s, compared to 60-s.
Monks et al., (2017)	N = 10 24 ± 5		10 x 10-s sprints (maximal effort)	30-s; 180-s	180-s	Repeated sprint ability decreased to a greater extent with 30-s compared to 180-s recovery. 30-s recovery increased perceived pain compared to 180-s recovery. Time course and extent of neuromuscular fatigue of knee extensors similar between conditions.

Note. Age and $\dot{V}O_{2max}$ are presented as Mean ± SD. *Abbreviations:* SP = Self-paced maximal effort, SS = Self-selected recovery duration, ACT – active recovery intensity, PA – passive recovery intensity, RER = respiratory exchange ratio, $\dot{V}O_2$ = oxygen uptake ($L.min^{-1}$), $\dot{V}O_{2max/peak}$ = maximal oxygen uptake, $v\dot{V}O_{2max}$ = velocity that elicits $\dot{V}O_{2max}$, INT = interval, W:R = work recovery ratio, B[La] = blood lactate concentrations, M[La] = muscle lactate concentration, PCr = phosphocreatine, HR = heart rate (bpm), HRmax/peak = maximal heart rate, HR130 = recovery duration based on HR recovery to 130bpm, PPO = peak power output, PO = power output (W), P@$\dot{V}O_{2max}$ – Power output at $\dot{V}O_{2max}$, HHb = deoxyhaemoglobin, MAS = maximal aerobic speed, MAV = maximal aerobic velocity, G = study group, Sig. = significant, SSG – Small sided football game, RR – reduced recovery time, N/S – Not specified, SS-PR – Self-selected recovery duration based on perceived readiness, RPE = rating of perceived exertion, HIIT = high-intensity interval training.

Table adapted from the review of Schoenmakers et al., (2019).

To date the foremost study on the impact of recovery interval duration during long work interval HIIT (≥ 1-min) is from Seiler & Hetlelid, (2005). Well trained runners completed a 6 x 4-min HIIT session on three occasions with different recovery interval durations: 1, 2 or 4-min. Participants were instructed to achieve the highest average running speed during the work intervals (i.e. self-paced maximal effort). The key finding was that doubling the recovery duration from 1 to 2-min only lead to a 2% increase in running velocity. Further increasing the recovery duration to 4-min lead to no additional increase in achieved work intensity. It was concluded that the 2-min (or 2:1 work recovery ratio) was sufficient to preserve work interval performance during HIIT. In physiological terms, the 2-min recovery duration is consistent with the rapid time course of several acute intracellular recovery processes that occur after exercise cessation, such as the rapid component of phosphocreatine (PCr) recovery (Harris et al., 1976; Taylor et al., 1983), changes in potassium concentration (Lindinger, 1995; Medbo & Sejersted, 1990) and recovery of specific intracellular ions (i.e. inorganic phosphate [Pi] and dihydrogen phosphate [H_2PO_4]) which are linked to muscle contractile function (Boska et al., 1990; Degroot et al., 1993). The completion of the aforementioned recovery processes within 2-min, has been suggested to explain why the extension of recovery interval duration beyond 2-min had no further benefit to work interval performance.

More recently, two studies of similar design to Seiler & Hetlelid, (2005) also examined the effect of recovery interval duration on the acute physiological responses of HIIT (Smilios et al., 2017; Schoenmakers & Reed, 2018). Smilios et al., (2017), asked moderately trained runners to perform 4 x 4-min HIIT sessions at 90% of maximal aerobic velocity, with different recovery interval durations: 2, 3, or 4-min (ACT recovery at 35% of maximal aerobic velocity). While recovery interval duration had no effect on the total exercise time

spent at 80, 90 or 95% of $\dot{V}O_{2max}$, the 2-min recovery duration significantly increased the time spent exercising > 90% HRmax and resulted in higher rating of perceived exertion (RPE) and blood lactate concentration (B[La]) values. Likewise, in the study of Schoenmakers & Reed, (2018), well trained runners completed a 6 x 4-min HIIT protocol, with either a 1-min, 2-min or 3-min fixed recovery interval duration. While mean running velocity was higher in the 3-min HIIT protocol, there were no significant differences found in the time spent ≥ 90 and 95% of $\dot{V}O_{2max}$ and HRmax between recovery durations. The findings of Smilios et al., (2017) and Schoenmakers & Reed, (2018) corroborate those of Seiler & Hetlelid, (2005), by demonstrating that there is an optimum recovery duration for specific HIIT protocols, beyond which there is no further increase in the performance and acute physiological response of the work intervals.

Laurent et al., (2014), also investigated the effect of recovery interval durations on well trained runners, using the established 6 x 4-min HIIT session. Work intervals were self-paced on a maximal effort basis, with 1, 2, or 4-min recovery interval durations. The three recovery conditions demonstrated similar physiological (% HRmax, % Peak oxygen consumption [$\dot{V}O_{2peak}$], B[La]) and perceptual (RPE and session RPE [sRPE]) responses, supporting the findings of Seiler & Hetlelid, (2005). Moreover these results provide further efficacy for the 2:1 work recovery ratio as the most practical recovery duration prescription while concomitantly reducing the total time spent exercising.

Overall the findings of the literature suggest there is an optimum recovery duration for the specific HIIT protocol; with the optimum duration being the shortest time necessary to allow the individual to maintain work interval performance without compromising the acute

physiological responses, thereby maximising the time efficiency of the HIIT session. Extension of the recovery interval beyond the optimum duration appears to have no further benefit to HIIT session performance but increases the overall training time commitment. Unfortunately, the overwhelming majority of studies have used fixed recovery durations and/or work recovery ratios (i.e. 1:1 or 2:1) when investigating the acute effects of recovery interval duration, making it difficult to elucidate the optimum recovery duration for specific HIIT protocols. While fixed durations and work recovery ratios maybe the most common and practical approach to prescribing recovery interval duration, it is based on the assumption that every individual requires the same recovery duration during HIIT sessions. On the contrary, the optimal recovery interval duration is most likely highly individual and dependent on training status (Schoenmakers et al., 2019).

Although the attempts have been sparse, researchers have sought to individualise the recovery interval duration to try and find the optimum recovery duration for the individual during a specific HIIT protocol. The most common method of individualisation is the use of self-selected recovery duration, whereby participants are asked to recommence exercise when they perceive themselves to be able to complete the next work interval at the desired intensity. In the study of Seiler & Hetlelid, (2005) participants were provided with no feedback (time elapsed or heart rate [HR) and were asked to take as long as necessary during the recovery period to ensure they could complete the next work interval at the desired running velocity. The mean self-selected recovery duration was 118 ± 23-s, very close to the 2-min recovery duration (or 2:1 ratio) suggested to be physiologically optimal for the specific HIIT session. As expected, there was no difference in HIIT session performance between the self-selected recovery duration and the fixed recovery durations (Seiler & Hetlelid, 2005).

More recently, researchers have been further exploring the use of self-selected recovery duration as a method to individualise HIIT (Edwards et al., 2011; Gibson et al., 2017; Phillips et al., 2014; McEwan et al., 2018). McEwan et al., (2018) asked recreationally active runners to complete 12 x 30-s intervals at 105% of maximal aerobic velocity, with either 30-s recovery or a self-selected recovery duration (instructed to provide themselves with enough recovery to complete all 12 intervals at the target intensity). The participants mean self-selected recovery duration was significantly longer (51 ± 15-s) than the pre-set 30-s recovery. The longer self-selected recovery resulted in a greater change in HR during the recovery intervals (19 ± 9 bpm versus 8 ± 5 bpm). The relative time spent at > 105% of maximal aerobic velocity was greater during the longer self-selected recovery duration (90 ± 6% versus 74 ± 20%). However, the absolute time spent at > 90% of HRmax during the work intervals was lower with the self-selected recovery duration when compared to the 30-s recovery. In the study of Edwards et al., (2011), the participants self-selected recovery duration was significantly shorter than the work recovery ratio condition but resulted in similar 1000m running velocities and physiological response, making for a more time efficient HIIT session. Current research shows that self-selected recovery durations can be an effective method of individualisation, when participants are well familiarized with the procedures and physical demands of the HIIT protocol (McEwan et al., 2018; Edwards et al., 2011).

There are benefits of self-selected recovery duration prescription, as it considers the day-to-day variation in the individuals environmental and/or psychological state (McEwan et al., 2018; Edwards et al., 2011; Edwards & Noakes, 2009). Indeed, a well-trained and/or experienced athlete maybe able to more accurately perceive their readiness to commence the next work interval based on past experience, however it is less likely an inexperienced and/or

recreationally active individual will know when best to recommence exercise (Brownstein et al., 2018; Gibson et al., 2017). Ultimately there is one major limitation of the self-selected recovery prescription, it does not take into account the individuals' physiological readiness to recommence exercise. Which if not considered could lead to inadequate or excessive recovery between work intervals potentially compromising the training session.

To the authors knowledge there has only been one study which has used a physiological measure to individualise the duration of the recovery interval. Edwards et al., (2011) individualised the recovery duration by using the time taken for the participants HR to return to 130bpm between five, 1000m track runs. The HR recovery method produced close to a 2:1 work recovery ratio (113.8 ± 48-s), significantly shorter than both attempts of the self-selected recovery condition (162.3 ± 31.3-s and 158.2 ± 45.5-s) and 1:1 work recovery ratio condition (198.1 ± 8.2-s). However, there were no significant differences between recovery conditions for: running velocity, B[La], mean work interval HR or the mean intensity of the runs. In line with previous research (Billaut et al., 2003; Laurent et al., 2014; Seiler & Hetlelid, 2005; Smilios et al., 2017), the findings of Edwards et al., (2011) demonstrate that at a certain point further extension of the recovery interval duration has no effect on the physiological response of the work intervals.

Based on current understandings, the recovery of HR may not be appropriate in the prescription of recovery duration (Tocco et al., 2015). Firstly, it has been suggested that the fast phase of HR recovery is not influenced by cellular conditions around the active muscle at the cessation of exercise but is primarily mediated by central control mechanisms (Seiler & Hetlelid, 2005; Seiler & Sjursen, 2004). Therefore, assuming HR recovery is not related to

skeletal muscle recovery, muscular energy turnover or systemic oxygen (O_2) demand (Buchheit et al., 2012; Wu et al., 2005), the evidence for its usefulness as a mechanism for determining recovery interval duration is questionable. Secondly, using a fixed HR value for all participants does not take in account the interindividual and intraindividual differences/variation in HR responses (Achten & Jeukendrup, 2003).

At present most of the research investigating the acute physiological effects of recovery duration on HIIT performance have commonly used fixed recovery durations and/or work recovery ratios (i.e. 1:1 or 2:1) to prescribe recovery interval duration (Seiler & Hetlelid, 2005; Smilios et al., 2017; Schoenmakers et al., 2018; Laurent et al., 2014). Methods such as self-selected recovery durations based on perceived readiness offer a promising direction for HIIT individualisation, however it does not account for the individual's physiological readiness to recommence exercise. The use of HR recovery has been the only physiological based method used to individualise recovery interval duration. However, the method has received limited research attention, likely due to the inherent limitations of using HR to prescribe recovery duration, as described previously. There is currently no other method based on physiological rationale, that allows for the individualisation of the recovery interval duration.

Near infrared spectroscopy (NIRS), is a well-known non-invasive method used to measure muscle oxygenation, which reflects the ratio of O_2 delivery to the working muscle and $m\dot{V}O_2$ in the capillary beds (Hamaoka et al., 1996; Jones et al., 2016). The recovery of $m\dot{V}O_2$ considers the condition of the exercising muscle, as measurements are derived directly from the muscle body. Moreover, the recovery of $m\dot{V}O_2$ indicates an equilibrium in O_2 delivery

and consumption, thus no competition and/or inhibition of available O_2 supplies at the start of exercise (Buchheit et al., 2011). It has been suggested that the recovery duration of $m\dot{V}O_2$ after high intensity exercise is likely related to a greater depletion of adenosine triphosphate (ATP), PCr and/or myoglobin O_2 stores, which logically take longer to be restored. In addition, it is possible that $m\dot{V}O_2$ remains elevated above baseline values after high intensity exercise to compensate for the detrimental effect of a decreased muscle pH on PCr recovery (van den Broek et al., 2007; McMahon & Jenkins, 2002). Therefore, it is possible that $m\dot{V}O_2$ recovery coincides with the return of the exercising muscle to a state of metabolic homeostasis. The recovery rate of $m\dot{V}O_2$ also takes into account the intensity of the prior exercise (Buchheit et al., 2011), the individuals training status (Chance et al., 1992; Ding et al., 2001; Ichimura et al., 2006; Kounalakis et al., 2009) and age (Kutsuzawa et al., 2001). Based on current knowledge the recovery duration of $m\dot{V}O_2$ may provide a method to individualise the recovery interval duration during HIIT. With the IND recovery interval duration theoretically being the optimal recovery duration for the specific individual and HIIT protocol (optimal being defined as *the shortest time necessary to allow the individual to maintain work interval performance without compromising the acute physiological responses, thereby maximising the time efficiency of the HIIT session*).

As shown in table 2.1, the majority of cycling based research has focused on short work interval (≥ 30-s to < 1-min) and sprint interval (4-s to < 30-s) HIIT protocols. To date only one cycling based study has examined recovery interval duration during long work interval (≥ 1-min) HIIT (Edge et al., 2013). Participants were required to complete 6 x 2-min work intervals with either 1-min or 3-min recovery intervals. The 1-min recovery duration resulted in significantly higher B[La], muscle lactate concentration (M[La]), and hydrogen ion (H^+) content at the end of the HIIT session, while significantly lowering muscle PCr, when

compared to the 3-min recovery duration. Unfortunately, the study only had a cohort of 5 participants, limiting the power and applicability of the findings. Long work interval HIIT is regularly incorporated into the training programmes of endurance cyclists. As such, more research is required to improve the current understandings of the effects of the recovery interval duration on the performance of long work interval HIIT during cycling exercise.

As the preceding literature review shows, the recovery interval duration is an important component of the multivariate equation that is HIIT programming. While an inadequate recovery duration will negatively affect the performance of the work intervals, an excessive recovery duration will negatively affect the time efficiency of HIIT. The optimal recovery duration must therefore be a compromise, to maximise the training stimulus, while reducing the total session duration. Future research should seek to investigate whether the recovery duration of $m\dot{V}O_2$ would provide a method of optimising the recovery interval. In addition, while running has received the majority of recovery duration research attention, the effect of recovery duration on HIIT during cycling exercise has not yet been properly established and should be at the forefront of any future research.

II.V – The recovery interval: Intensity

There is a sizable body of literature which has investigated the effects of recovery interval intensity, using different HIIT protocols across various sports (*Table 2.2*). Table 2.2 contains a comprehensive list of research papers which have examined the acute physiological effects of recovery interval intensity on HIIT performance.

II.V.1 – Literature search strategy

Electronic database searches were performed using PubMed, Google Scholar and ResearchGate. The search terms covered the areas of high intensity interval training and recovery interval intensity, using a combination of the following key words: high-intensity interval training, interval training, sprint training, recovery interval, recovery intensity. Relevant papers were collated in Mendeley. Reference lists of collated papers were examined for other eligible papers. Electronic searches of the papers which had cited relevant papers were also made to identify other eligible papers.

II.V.2 – Literature inclusion criteria

1) Examining the acute physiological effects of recovery interval intensity on interval training performance.

2) Protocols applied must be formed of ≥ 2 repeated intervals.

3) Studies must be investigating between work interval recovery intensity, NOT recovery intensity after a training session.

4) Study participants must be healthy individuals, free from injury or disease.

Thirty-two papers met the inclusion criteria.

Table 2.2 – Summary of studies which have investigated the acute physiological effects of recovery interval intensity.

Study	Sample size Age (years) VO2max (ml.kg.min^{-1})	Exercise modality	HIIT protocol (work INT intensity)	Recovery Intensity (recovery duration)	Optimum	Key study findings
Long HIIT (≥ 1-min)						
Barbosa et al., (2016)	N = 18 25 ± 4 41 ± 3	Cycling	Long INT: half duration of $\dot{V}O_2SC$ Short INT: Onset of VO_2SC (95% I$\dot{V}O_{2max}$)	PA; ACT 50% I$\dot{V}$O2max (2:1 W:R)	PA & ACT	TTE was significantly longer during the PA condition (1523 ± 411-s) when compared to the ACT condition (902 ± 239-s), during the short INT sessions. In contrast, there were no significant differences in the TTE between the PA (984 ± 260-s) and ACT (886 ± 254-s) recovery conditions, during the long INT sessions. Recovery interval intensity also had no effect on end exercise $\dot{V}O_2$, between all HIIT sessions.
Coso et al., (2010)	N = 11 22 ± 3 52 ± 6	Cycling	4 x 1.5-min (163% of RCT)	4.5-min ACT (24% RCT); 6-min ACT (18% RCT); 9-min ACT (12% RCT)	9-min ACT	9-min ACT increased plasma pH and reduced B[La] to a greater extent than the 4-min and 6-min ACT. Similar work INT $\dot{V}_E$ and HR between HIIT sessions. $\dot{V}_E$ and HR dropped to a sig. lower level as recovery duration increased.
Dorado et al., (2004)	N = 10 24 ± 2 58 ± 5	Cycling	4 x bouts to exhaustion (approx. 2-min) (110% Wmax)	ACT (20% $\dot{V}O_{2max}$); Stretching; PA (5-min)	ACT	Work performed (kJ) sig. greater during ACT. Sig. higher aerobic energy yield during ACT, then stretching or PA. ACT recovery accelerated $\dot{V}O_2$ kinetics and increased $\dot{V}O_{2peak}$ attained during work INT. Similar peak B[La] between conditions.
Monedero & Donne, (2000)	N = 18 25 ± 1 68 ± 2	Cycling	2 x 5-km (maximal effort)	PA; ACT (50% $\dot{V}O_{2max}$); Massage; 50/50 ACT/PA (15-min)	50/50 ACT/PA	The ACT/PA recovery condition was found to be most effective at B[La] clearance and maintenance of 5-km performance time. Higher recovery HR during ACT and ACT/PA conditions.
McAinch et al., (2004)	N = 7 22 ± 4 58 ± 9	Cycling	2 x 20-min (maximal effort)	PA; ACT (40% $\dot{V}O_{2peak}$) (15-min)	PA & ACT	No difference between the ACT and PA conditions in work performed (kJ) during the work INT. No sig. difference in B[La], muscle glycogen content or ATP and PCr concentrations between ACT and PA conditions.

Table 2.2 - continued

Study	Sample size Age (years) VO2max (ml.kg.min^{-1})	Exercise modality	HIIT protocol (work INT intensity)	Recovery Intensity (recovery duration)	Optimum	Key study findings
Siegler et al., (2006)	N = 10 30 ± 7 56 ± 7	Cycling	3 x bouts to exhaustion (110% max workload)	PA; ACT (20% max workload) (12-min)	PA & ACT	ACT demonstrated enhanced metabolic waste removal. No sig. differences in TTE or $\dot{V}O_{2peak}$ of work INT.
Stanley & Buchheit, (2014)	N = 14 25 ± 4 67 ± 4	Cycling	3 x 3-min (90% p$\dot{V}O_{2max}$)	ACT (30% p$\dot{V}O_{2max}$); ACT (60% p$\dot{V}O_{2max}$) (2-min)	ACT	$\dot{V}O_2$, HR and cardiac output sig. higher during the 60% ACT condition when compared to the 30% ACT condition. No difference in stroke volume between recovery conditions. TSI% sig. lower during 60% ACT, compared to 30% ACT.
Mandroukas et al., (2011)	N = 15 22 ± 3	Running	4 x 4-min (12-km.h^{-1})	PA; ACT (8-km.h^{-1}) (4-min)	PA & ACT	No sig. difference in work INT HR and $\dot{V}O_2$ between PA and ACT. B[La] sig. higher in PA when compared to ACT. Recovery INT HR and $\dot{V}O_2$ sig higher during ACT.
Losnegard et al., (2015)	N = 10 22 ± 3	Cross-county skiing	2 x 800m (maximal effort)	PA; ACT (58% of $\dot{V}O_{2peak}$) (21-min)	PA & ACT	No sig. difference in performance of the 800m work bouts between recovery conditions. No sig. difference in work interval $\dot{V}O_2$, $\dot{V}O_{2peak}$, HRpeak and B[La] between PA and ACT recovery conditions.
Short HIIT (≥ 30-s to < 1-min)						
Bogdanis et al., (1996)	N = 13 25 ± 3 55 ± 2	Cycling	2 x 30-s (maximal effort)	PA; ACT (40% $\dot{V}O_{2max}$) (4-min)	ACT	PO and $\dot{V}O_2$ of 2nd INT was sig. higher in ACT when compared to PA. No differences in venous B[La] or pH. Recovery INT HR was sig higher during ACT.
Spierer et al., (2004)	G1 (Sedentary) N = 6 32 ± 1 37 ± 6 G2 (Moderately Trained) N = 9 22 ± 1 46 ± 5	Cycling	30-s sprints to exhaustion (or peak < 70% of 1st sprint) (maximal effort)	PA; ACT (28% $\dot{V}O_{2max}$) (4-min)	ACT	Mean number of 30-s sprints completed was the same between ACT and PA for G1 and G2. Peak PO of sprints were not sig. different between PA and ACT for G1 and G2. Mean PO was sig. higher during ACT compared to PA for G1. No sig. difference in mean PO between ACT and PA for G2. TW achieved during the session were sig. higher during the ACT compared to PA for G1 and G2. Peak HR and B[La] were not sig. different between PA and ACT for G1 and G2.

Table 2.2 - continued

Study	Sample size Age (years) VO2max ($ml.kg.min^{-1}$)	Exercise modality	HIIT protocol (work INT intensity)	Recovery Intensity (recovery duration)	Optimum	Key study findings
Dupont et al., (2007)	N = 12 23 ± 5	Cycling	1 x 15-s followed by 1 x 30-s INT (maximal effort)	PA; ACT (20% MAP); ACT (40% MAP) (15-s)	PA	Mean PO and peak PO of 30-s INT sig. higher after PA, then after both ACT.
Koizumi et al., (2011)	N = 10 20 ± 1	Cycling	2 x 30-s (maximal effort)	PA; ACT (30% of $\dot{V}O_2$ at LT) (20-min)	ACT	TW and peak PO of the 2nd 30-s sprint were sig. higher after the ACT, when compared to the PA. Greater decrease in B[La] during ACT 20-min recovery. During ACT 20-min recovery, mean $\dot{V}O_2$, HR and change in tHb were sig. higher than the PA 20-min recovery.
Kriel et al., (2016)	N = 12 23 ± 3	Cycling	4 x 30-s (maximal effort)	PA; ACT (60W) (2-min)	PA	Mean PO was sig. higher during the PA (374 ± 70W) when compared to the ACT (340 ± 73W). No sig. difference in mean $\dot{V}O_2$ and HR between PA and ACT. HHb of VL sig. higher during ACT when compared to PA.
Yamagishi & Babrai, (2019)	N = 7 23 ± 5 48 ± 5	Cycling	4 x 30-s sprints (maximal effort)	PA; ACT (20% $\dot{V}O_{2max}$); ACT (30% $\dot{V}O_{2max}$); ACT (40% $\dot{V}O_{2max}$) (4-min)	ACT	No main effect of recovery intensity on peak PO, mean PO or reduction in PO across sprints. Work INT $\dot{V}O_2$ was sig. higher during all ACT conditions when compared to the PA condition. Work INT HR was sig. higher during the 30% and 40% ACT conditions when compared to PA and ACT 20% conditions. Recovery $\dot{V}O_2$ sig. increased during all ACT conditions when compared to PA condition. No sig. in B[La] between all recovery intensities.
Miladi et al., (2011)	N =10 26 ± 2 56 ± 7	Cycling	2 sets of 4 x 30-s followed by TTE test (120% MAP)	PA; ACT (30% MAP); DS (4-min between work INT set recovery) (30-s between INT recoveries were passive)	DS	TTE was sig. longer after DS, when compared to ACT and PA. B[La] sig. higher at end of both work INT sets during the PA, compared to ACT and DS. HR and $\dot{V}O_2$ recovered to a greater extent during the recovery INT in the PA, compared to ACT and DS. No sig. differences in end work INT HR and $\dot{V}O_2$ between all conditions.

Table 2.2 - continued

Study	Sample size Age (years) VO2max ($ml.kg.min^{-1}$)	Exercise modality	HIIT protocol (work INT intensity)	Recovery Intensity (recovery duration)	Optimum	Key study findings
Lopez et al., (2014)	N = 15 29 ± 8	Cycling	6 x 30-s (maximal effort)	PA; ACT (1.1 $W.kg^{-1}$) (4-min)	ACT	Mean PO sig. higher in sprints 5 & 6 of ACT condition, compared to PA. No sig. difference in mean PO for sprints 1 to 4 between conditions. No sig. difference in mean HR and total work (kJ) of 30-s sprints between PA and ACT. HR of ACT recovery INT sig. higher than PA recovery INT.
Wahl et al., (2013)	N = 12 25 ± 3 64 ± 10	Cycling	4 x 30-s (maximal effort)	PA; ACT (40% PPO) (7-min 30-s)	PA & ACT	No sig. differences in mean PO and PPO between the PA and ACT recovery conditions. Total work (kJ), total energy expenditure (kJ), mean $\dot{V}O_2$ and mean HR sig, higher in ACT condition, compared to PA. B[La] sig. higher throughout PA session, compared to ACT.
Thevenet et al., (2007)	N = 8 16 ± 1 57 ± 6	Running	30-s to exhaustion (10% MAV)	PA; ACT (50% MAV) (30-s)	PA & ACT	TTE was sig. longer during PA (2145 ± 829-s) compared to ACT (1072 ± 388-s). No sig. differences in absolute time at spent above 90% and 95% $\dot{V}O_{2max}$ between PA and ACT. Time spent above 90% and 95% $\dot{V}O_{2max}$ as a percentage of TTE was sig. higher for ACT than PA.
Toubekis et al., (2006)	N = 9 19 ± 1 65 ± 1	Swimming	8 x 25-m sprints (maximal effort)	PA; ACT50 (50% of 100m velocity); ACT60 (60% of 100m velocity) (45-s)	PA	Mean performance time of the 25-m sprints was faster during the PA, compared to ACT50 and ACT60. B[La] was sig. higher during PA, compared to ACT50 and ACT60. Peak HR after each sprint was not sig. different between conditions.
Toubekis et al., (2005)	N = 16 21 ± 1	Swimming	8 x 25-m sprints (maximal effort)	PA; ACT60 (60% of 100m velocity) (45-s; 2-min)	PA	Mean swimming velocity sig. faster in PA compared to ACT for both recovery durations. No sig. in end B[La]. 50-m sprint times 2.4% faster in 2-min compared to 45-s, in both ACT and PA conditions.

Table 2.2 - continued

Study	Sample size Age (years) VO2max ($ml.kg.min^{-1}$)	Exercise modality	HIIT protocol (work INT intensity)	Recovery Intensity (recovery duration)	Optimum	Key study findings
Jougla et al., (2010)	N = 7 21 ± 0.5	Rugby	6 x 4 consecutive actions (Scrummaging, agility sprinting, tackling & straight sprinting)	PA; ACT (50% MAS) (30-s)	PA	Scrum forces were lower in the ACT than PA condition. Total sprint time were sig. longer in the ACT, compared to PA. PA enabled better performance of the Narbonne test, when compared to ACT. No sig. difference in mean HR during the 6 x 4 actions between ACT and PA.
Sprint interval training (4-s to < 30-s)						
Dupont et al., (2004)	N = 12 24 ± 4	Cycling	15-s INT to exhaustion (maximal effort)	PA; ACT (40% $\dot{V}O_{2max}$) (15-s)	PA	TTE sig. longer with PA (962 ± 314-s), then ACT (427 ± 118-s). Mean metabolic power sig. lower during PA, then ACT. Mean rate of O_2Hb decrease sig. slower during PA, then ACT. No sig. differences in $\dot{V}O_{2peak,}$ mean $\dot{V}O_2$, HRpeak, mean HR and B[La] between PA and ACT.
Spencer et al., (2008)	N = 9 19 ± 2 53 ± 7	Cycling	6 x 4-s sprints every 25-s (maximal effort)	PA; ACT (35% $\dot{V}O_{2max}$ / 60W); ACT (20% $\dot{V}O_{2max}$ / 20W) (21-s)	PA	Peak PO sig. lower in sprints 2, 3, 4 & 6 ACT (35% $\dot{V}O_{2max}$) and sprints 4 & 6 ACT (20% $\dot{V}O_{2max}$) when compared to PA. No sig. difference in peak PO between the two ACT conditions. No sig. differences in ATP, PCr and M[La] between the two ACT conditions.
Spencer et al., (2006)	N = 9 25 ± 7 55 ± 6	Cycling	6 x 4-s sprints every 25-s (maximal effort)	PA; ACT (32% $\dot{V}O_{2max}$) (21-s)	PA	ACT resulted in a sig. greater reduction in PO and lower final peak PO, when compared to PA. PCr was sig. after 21-s of recovery and post session during ACT, when compared to PA. No sig. difference between ACT and PA in M[La] after 21-s of recovery.

Table 2.2 - continued

Study	Sample size Age (years) VO2max (ml.kg.min^{-1})	Exercise modality	HIIT protocol (work INT intensity)	Recovery Intensity (recovery duration)	Optimum	Key study findings
Ohya et al., (2013)	N = 8 26 ± 3 51 ± 6	Cycling	10 x 5-s sprints (maximal effort)	PA; ACT (40% $\dot{V}O_{2max}$) (25-s, 50-s, and 100-s)	PA	Peak PO values sig. higher during PA than ACT for 25-s recovery condition (sprints 2 to 9) and 50-s recovery condition (sprints 2 to 6, 9 and 10). No difference in peak PO between PA and ACT during 100-s recovery condition. ACT associated with lower muscle reoxygenation. Recovery intensity had no effect on performance over 100-s recovery condition.
Signorile et al., (1993)	N = 6 27 ± 9	Cycling	8 x 6-s sprints (maximal effort)	PA; ACT (60rpm, 1-kg resistance) (30-s)	ACT	Mean peak PO and TW of all sprints were sig. higher during ACT condition, when compared to PA condition.
Bishop et al., (2009)	N = 8 21 ± 2 51 ± 5	Cycling	18 x 4-s sprints (maximal effort)	PA; ACT (35% $\dot{V}O_{2max}$) (120-s)	PA & ACT	No sig. difference in mean work per sprint (J) or mean peak PO per sprint, between the ACT and PA conditions. HR sig. higher during ACT protocol than PA protocol. No sig. differences in plasma lactate concentration and RPE between ACT and PA conditions.
Ahmaidi et al., (1996)	N = 10 27 ± 2	Cycling	4 to 5 x 6-s sprints against increasing braking forces (1kg, 2kg, 4kg and 6kg)	PA; ACT (32% MAP) (5-min)	ACT	No sig. difference in mean sprint PO between PA and ACT at 2kg and 4kg braking forces. Mean sprint PO were sig. higher with ACT than PA at 6kg braking forces. Plasma lactate concentrations were sig. lower with ACT than PA.
Connolly et al., (2015)	N = 7 22 ± 3	Cycling	6 x 15-s sprints (maximal effort)	PA; ACT (80rpm, 1kg resistance) (3-min)	PA & ACT	Sig. greater decrease in sprint PPO with PA recovery, compared to ACT. No sig. difference in mean PPO between recovery conditions. No sig. difference in B[La] between recovery conditions

Table 2.2 - continued

Study	Sample size Age (years) VO2max ($ml.kg.min^{-1}$)	Exercise modality	HIIT protocol (work INT intensity)	Recovery Intensity (recovery duration)	Optimum	Key study findings
Dupont & Berthoin, (2004)	N = 12 24 ± 4 58 ± 7	Running	15-s runs to exhaustion (120% of MAS)	PA; ACT (50% MAS) (15-s)	ACT	TTE was sig. shorter for ACT (445 ± 79-s) then PA (745 ± 171-s). T@$\dot{V}O_{2max}$ and above 90% $\dot{V}O_{2max}$ was not sig. different between ACT and PA. Percentage of exercise time spent at $\dot{V}O_{2max}$ and above 90% $\dot{V}O_{2max}$ was sig. higher for ACT (41.2 ± 26.8%) than PA (25.4 ± 16.2%). No sig. difference in B[La], mean or Peak HR between ACT and PA.
Buchheit et al., (2009)	N = 10 27 ± 4 55 ± 8	Running	6 x 4-s sprints every 25-s (maximal effort)	PA; ACT (2-$m.s^{-1}$) (21-s)	PA	Mean running speed was sig. lower and percent speed decrement sig. higher for ACT compared to PA. Mean session HR and $\dot{V}O_2$ were sig. higher for ACT compared to PA. No sig. difference in HRmax attained between ACT and PA. Mean HHb level was sig. higher during ACT condition, compared to PA condition. RPE sig. higher in ACT compared to PA.

Note. Age and $\dot{V}O_{2max}$ are presented as Mean ± SD. *Abbreviations:* PA = passive recovery intensity, ACT = active recovery intensity, TTE = time to exhaustion, $\dot{V}O_2$ = oxygen uptake ($L.min^{-1}$), $\dot{V}_E$ = pulmonary ventilation, $\dot{V}O_2SC$ = $\dot{V}O_2$ slow component, $\dot{V}O_{2max/peak}$ = maximal oxygen uptake, I$\dot{V}O_{2max}$ = intensity that elicits $\dot{V}O_{2max}$, INT = interval, W:R = work recovery ratio, RCT = respiratory compensation threshold, B[La] = blood lactate concentrations, M[La] = muscle lactate concentration, PCr = phosphocreatine, ATP = adenosine triphosphate, HR = heart rate (bpm), HRmax/peak = maximal heart rate, PPO = peak power output, PO = power output (W), HHb = deoxyhaemoglobin, tHb = total haemoglobin, MAS = maximal aerobic speed, MAV = maximal aerobic velocity, p$\dot{V}O_{2max}$ = power at $\dot{V}O_{2max}$ T@$\dot{V}O_{2max}$ = time at $\dot{V}O_{2max}$, VL = vastus lateralis muscle, TW = total work (kJ), LT = lactate threshold, G = study group, DS = dynamic stretching, Sig. = significant, RPE = rating of perceived exertion, TSI (%) = Tissue saturation index.

The majority of cycling based research has focused on short HIIT (work intervals ≥ 30-s to < 1-min) and sprint interval training (work intervals 4-s to < 30-s; see *table 2.2*). This is perhaps due to the continued interest in maximising the time efficiency of HIIT for recreationally active individuals (Burgomaster et al., 2008; Gibala et al., 2012). In the studies of Spencer et al., (2006 & 2008) 6 x 4-s sprints with 25-s recovery intervals were prescribed. In both studies, low intensity ACT recovery (< 35% $\dot{V}O_{2max}$) lead to a significantly greater decrement in PO and reductions in peak PO when compared to PA recovery intervals. However, there were no significant differences in total work produced across the work intervals, between recovery conditions. PCr was also measured, with lower PCr levels following 21-s of recovery during the ACT recovery conditions. PCr is a key energy store for anaerobic sprint performance, and as Spencer et al., (2006 & 2008) show low to moderate levels of muscle activation produced during ACT recovery attenuates the resynthesis of PCr effecting the performance PO of subsequent intervals.

In accordance, Dupont et al., (2004 & 2007) also found ACT recovery to impair repeated sprint performance, when compared to PA recovery. When performing 15-s sprints with 15-s recoveries, it was found that PA recovery increased the time to exhaustion when compared to ACT recovery at 40% of $\dot{V}O_{2max}$. The PA recovery resulted in a lower metabolic power when compared to the ACT recovery, which could explain the increased time to exhaustion (Dupont et al., 2004). By extending time to exhaustion athletes would be able to accumulate a greater training load from the given session. PA recovery may also allow for a greater reoxygenation of myoglobin and consequently a greater resynthesis of PCr (Dupont et al., 2004; Spencer et al., 2006 & 2008), hence the ability to perform a greater number of repeated sprints before exhaustion. Several other studies have also shown repeated short/sprint intervals to be negatively affected by ACT recovery (Kriel et al., 2016; Ohya et al., 2013).

When performing all-out sprint intervals, PA recovery appears to be the most appropriate recovery intensity for maintaining work interval performance (Spencer et al., 2006 & 2008; Dupont et al., 2004 & 2007; Kriel et al., 2016; Ohya et al., 2013).

However, there are a proportionate number of studies which have shown ACT recovery to be beneficial to repeated short/sprint interval performance. Signorile et al., (1993), prescribed 8 x 6-s sprints interspersed with 30-s recovery. Peak PO and total work were found to be significantly higher during the ACT recovery condition when compared to the PA recovery condition. The authors therefore concluded that ACT recovery was superior for maximising the performance of repeated sprints when compared to PA recovery. In agreement, Ahmaidis et al., (1996) also showed an increase in 6-s sprint PO when 5-min ACT recoveries (32% of maximal aerobic power) were applied between sprints, when compared to 5-min PA recoveries.

The research of Bogdanis et al., (1996), Spierer et al., (2004) and Yamagishi & Babraj, (2019), focused on 30-s sprint (Wingate) performance with 4-min ACT or PA recovery intervals. In the study of Bogdanis et al., (1996), 2 x 30-s sprints were performed with a 4-min ACT recovery (40% of $\dot{V}O_{2max}$) or PA recovery. The ACT recovery resulted in a significantly higher mean PO and $\dot{V}O_2$ during the 2nd 30-s sprint, when compared to the PA recovery. The findings of Spierer et al., (2004) corroborate those of Bodganis et al., (1996), demonstrating that a 4-min ACT recovery at 28% of $\dot{V}O_{2max}$ increases the mean 30-s sprint PO, in addition to increasing the total work achieved during the repeated sprints, when compared to a PA recovery. It has been suggested that ACT recovery promotes O_2 delivery through increased blood flow to the working muscle, which in turn improves PCr resynthesis

(Sahlin et al., 1979) and accelerates the removal of lactate and H^+, providing the recovery interval is of an adequate duration. Finally, ACT recovery (40% of $\dot{V}O_{2peak}$) has been shown to increase the mean $\dot{V}O_2$ and HR of the HIIT session (6 x 30-s sprint with 4-min recovery) when compared to PA recovery. The greater training load of the ACT recovery condition, resulted in an greater increase in the participants endurance capacity after 6 HIIT sessions (Yamagishi & Babraj, 2019).

As the above review of literature shows, the most effective recovery intensity appears to be influenced by the duration of the recovery interval provided. When short/sprint work intervals are separated by short (i.e. < 30-s) recovery intervals, PA recoveries appear to allow for the maximum performance of subsequent intervals (Spencer et al., 2006 & 2008; Dupont et al., 2004 & 2007; Kriel et al., 2016; Ohya et al., 2013). However, when long recovery intervals (i.e. 4-min) separate the work intervals, ACT recovery results in the greatest performance of the subsequent intervals (Ahmaidi et al., 1996; Bogdanis et al., 1996; Spierer et al., 2004; Lopez et al., 2014; Yamagishi & Babraj, 2019). Moreover, recovery intensity programming also appears to be dependent on the desired outcome of the session, with ACT recovery between short/sprint intervals shown to induce a greater training stimulus and endurance adaptations, albeit in untrained adults (Yamagishi & Babraj, 2019). The growing diversity of HIIT protocols, aligned with recovery intensity being dependent on the design of the HIIT protocol and desired session outcome, makes determining the optimum recovery intensity for a specific HIIT protocol difficult for athletes and coaches.

There has been a comparatively limited number of studies which have investigated the effects of recovery interval intensity during cycling HIIT sessions with long work intervals (≥ 1-min;

Barbosa et al., 2016; Coso et al., 2010; Dorado et al., 2004; Monedero et al., 2000; McAinch et al., 2004; Siegler et al., 2006; Stanley & Buchheit, 2014), when compared to short and sprint HIIT. This absence of research is surprising as long work interval sessions provide a potent stimulus for driving central and peripheral endurance adaptations (MacInnis & Gibala, 2017; Tschakert & Hofmann, 2013) and are therefore frequently incorporated into endurance athletes training programmes.

Coso et al., (2010) recruited moderately trained cyclists to perform 4 x 1.5-min intervals at 163% of their respiratory compensation threshold (RCT). Recovery intervals were either 4.5-min at 24% of RCT, 6-min at 18% of RCT or 9-min at 12% of RCT. The researchers found the 9-min recovery at 12% of RCT resulted in lower B[La] and H^+ concentrations, when compared to the shorter more intense recovery periods. The greater metabolic muscle recovery afforded by longer less intense recovery intervals, may be important as intramuscular acidosis has been shown to produce muscle fatigue by hampering the excitation contraction coupling (Favero et al., 1995) or by slowing glycogenolysis and therefore energy provision (Spriet et al., 1989). However, it is questionable whether the consideration of these protracted recovery processes are crucial when looking to maximise the physiological response and time efficiency of HIIT.

In the recent study of Barbosa et al., (2016) physically active males performed repeated work intervals of two different lengths (either long or short) at 95% of $\dot{V}O_{2max}$ until exhaustion. Recovery intervals were prescribed on a 2:1 work recovery ratio and were either, ACT (50% of intensity that elicits $\dot{V}O_{2max}$) or PA intensity. The participants time to exhaustion was significantly longer during the PA condition (1523 ± 411-s) when compared to the ACT

condition (902 ± 239-s), during the short interval sessions. However, there were no significant differences in the time to exhaustion between the PA (984 ± 260-s) and ACT (886 ± 254-s) recovery conditions during the long interval sessions, with recovery interval intensity having no effect on end exercise $\dot{V}O_2$, between all HIIT sessions. An earlier study using repeated work intervals to exhaustion found ACT recovery to be beneficial to work interval performance (Dorado et al., 2004). When performing repeated work intervals to exhaustion (4 x work intervals at 110% max PO), 5-min ACT recovery (20% of $\dot{V}O_{2max}$) was found to increase the total work performed in the 2nd, 3rd and 4th work intervals by 13% and 9% when compared to two modes of PA recovery. ACT recovery between work intervals also increased the mean $\dot{V}O_2$ and $\dot{V}O_{2peak}$ of the subsequent work intervals, in addition to accelerating $\dot{V}O_2$ kinetics when compared to PA recovery (Dorado et al., 2004).

In a study of similar design to Dorado et al., (2004), participants were required to perform 3 x work intervals to exhaustion at 110% of maximum workload, with either 12-min of ACT recovery (20% maximum workload) or PA recovery. While ACT recovery demonstrated enhanced metabolic waste removal, there were no differences in time to exhaustion or the $\dot{V}O_{2peak}$ attained during the work intervals, between recovery conditions (Siegler et al., 2006). Interestingly another study found combined recovery (50/50 ACT/PA) to be the most effective for B[La] clearance and maintenance of work interval performance (5km maximal efforts with 15-min recovery), when compared to a solely ACT recovery (50% of $\dot{V}O_{2max}$) or PA recovery (Monedero & Donne, 2000).

Finally, in the study of McAinch et al., (2004), participants completed 2 x 20-min cycling bouts with 15-min recovery, which were either an ACT (40% of $\dot{V}O_{2peak}$) or PA intensity.

There was no difference in the work performed during the 20-min cycling bouts between the two recovery conditions. Despite there being no difference in work interval performance, ACT recovery did accelerate the decline in plasma lactate levels.

Many of the studies which have examined recovery interval intensity during cycling based HIIT using long work intervals (≥ 1-min) have used experimental designs which are not reflective of the type of HIIT sessions used in practice. Specifically, the use of long recovery durations (relative to the work interval duration; Siegler et al., 2006; Monedero & Donne, 2000), a limited number of work intervals (Monedero & Donne, 200), limited overall work interval duration (Stanley & Buchheit, 2014) and time to exhaustion work intervals (Dorado et al., 2004; Siegler et al., 2006). As such the constraints of these experimental designs preclude the practical application of their research findings. To the authors knowledge there has not been a study investigating the acute physiological and perceptual effects of recovery interval intensity during cycling exercise, using HIIT sessions similar to those currently used by athletes and in HIIT research (Billat et al., 2001; Fiskerstrand & Seiler, 2004; Seiler & Sjursen, 2004; Steinacker et al., 1998; Stepto et al., 2001).

Overall, the variation in findings between studies are most likely due to differences in HIIT protocol design and differences in recovery intensities applied (Barbosa et al., 2016; Coso et al., 2010; Dorado et al., 2004; Monedero et al., 2000; McAinch et al., 2004; Siegler et al., 2006). Ultimately this also constrains the practical application of study findings, as the recovery intensity found to be best for a specific HIIT protocol may not be optimal when applied to another HIIT protocol design. Therefore, it is important there is continued research

utilising different HIIT protocol designs and recovery intensities, to broaden the understanding of the acute effects of recovery interval intensity across HIIT protocols.

An overriding limitation of the cycling research is the heterogeneity in how each study quantifies the effect of recovery interval intensity on work interval performance and overall HIIT session performance. While simply reporting mean PO, peak PO, metabolite clearance or time to exhaustion gives an insight into the effect of the manipulated variable, these measures alone fail show how effective the HIIT session was as an acute training stimulus. It has been suggested that the time at $\dot{V}O_{2max}$ (T@$\dot{V}O_{2max}$) provides an easily measurable indication of the acute training stimulus generated by the HIIT session (Buchheit & Laursen, 2013). When compared to PA recoveries, intermittent runs to exhaustion using ACT recoveries have been reported to be 40 to 80% shorter in duration (Thevent et al., 2007; Dupont et al., 2003; Dupont et al., 2004; Dupont & Berthoin, 2004). However, despite the decreased time to exhaustion when using ACT recovery, the absolute T@$\dot{V}O_{2max}$ may not differ to a PA recovery condition; moreover, the T@$\dot{V}O_{2max}$ relative to the total exercise duration may be substantially greater when ACT recovery is implemented (Dupont & Berthoin, 2004). In addition, increasing ACT recovery exercise intensity from 50% to 67% of $\dot{V}O_{2max}$ was associated with increases in T@$\dot{V}O_{2max}$ and T@$\dot{V}O_{2max}$ relative to the total exercise duration. However, increasing recovery intensity further to 87% of $\dot{V}O_{2max,}$ T@ $\dot{V}O_{2max}$ was reduced but T@ $\dot{V}O_{2max}$ relative to total exercise duration was increased (Thevenet et al., 2007). As the studies of Dupont & Berthoin, (2004) and Thevenet et al., (2007) show, the use of T@$\dot{V}O_{2max}$ clearly defines the acute physiological effect of manipulating recovery intensity. Future research investigating the manipulation of any HIIT variable should consider measuring and reporting T@$\dot{V}O_{2max}$. This would make elucidating

the acute physiological effects easier, in addition to allowing for clearer comparisons between the different recovery intensities and HIIT protocols.

As shown in table 2.2 and discussed in the review above, there is considerable diversity in the current body of literature investigating the acute effects of recovery interval intensity. The heterogeneity in HIIT protocols and methodologies used, in addition to the results presented makes the practical application of the research findings difficult. Recovery intensity appears to be highly dependent on HIIT protocol design and the desired training session outcome. The lack of consensus in the research regarding the optimum recovery intensity, makes it clear there is not a one size fits all approach to prescribing recovery intensity. Hence the importance of continued research into the acute effects of recovery interval intensity using different HIIT protocol designs to address the gaps in current understandings.

II.VI – Thesis Statement of Purpose

Through reviewing the available research which has investigated the acute effects of the recovery interval components on HIIT, it was evident that there were several significant gaps in the literature (for overview of literature see *Tables 2.1 & 2.2*). Firstly, there are currently no effective methods based on physiological rationale, that allows for the individualisation of the recovery interval duration. It was proposed that the recovery duration of $m\dot{V}O_2$ may provide a method of individualising the recovery interval. Secondly, more research is required to improve current understandings of the effects of the recovery interval duration on the performance of long work interval HIIT during cycling based exercise. Study one therefore sought to investigate whether individualising the duration of the recovery interval based on the participants $m\dot{V}O_2$ recovery duration would maximise the performance and acute physiological response to long work interval HIIT sessions when compared to a STD recovery duration (2:1 work recovery ratio).

Recovery interval intensity is highly dependent on the design of the HIIT protocol, limiting the practical application of research findings to the specific HIIT protocols used in the study. In addition, many of the studies which have examined recovery interval intensity during cycling based HIIT using long work intervals (≥ 1-min) have used experimental designs which are not reflective of the type of HIIT sessions used in practice. Study two therefore sought to investigate the acute physiological and perceptual effects of a PA and two ACT recovery intensities during cycling based HIIT using long work intervals (≥ 1-min). While adding to the current understanding of how the manipulation of recovery interval intensity effects work interval and overall HIIT session performance.

II.VII – Statement of Research Hypothesis

Study One – The impact of recovery interval duration on the acute physiological responses to interval training during cycling exercise.

Null Hypothesis: The IND recovery duration will have no effect on work interval PO and will not result in a greater acute physiological response, when compared to the STD recovery duration. As measured by mean work interval and HIIT session: HR, B[La], RPE, sRPE, $\dot{V}O_2$, T@$\dot{V}O_{2max}$, T@HRmax, HHb, O_2Hb and TSI %.

Alternative Hypothesis: The IND recovery duration will produce a higher work interval PO and will therefore result in a greater acute physiological response, when compared to the STD recovery duration (2:1 work recovery ratio). As measured by mean work interval and HIIT session: HR, B[La], RPE, sRPE, $\dot{V}O_2$, T@$\dot{V}O_{2max}$, T@HRmax, HHb, O_2Hb and TSI %.

Study Two – The impact of recovery interval intensity on the acute physiological responses to interval training during cycling exercise.

Null Hypothesis: Increasing ACT recovery intensity will have no effect on work interval PO and will not reduce the acute physiological responses, when compared to a PA recovery intensity. As measured by mean work interval and HIIT session: HR, B[La], RPE, sRPE, $\dot{V}O_2$, T@$\dot{V}O_{2max}$, T@HRmax, HHb, O_2Hb and TSI %.

Alternative Hypothesis: Increasing ACT recovery intensity will reduce work interval PO and will therefore reduce the acute physiological responses, when compared to a PA recovery intensity. As measured by mean work interval and HIIT session: HR, B[La], RPE, sRPE, $\dot{V}O_2$, T@$\dot{V}O_{2max}$, T@HRmax, HHb, O_2Hb and TSI %.

III. General Methods

III.I – Equipment List

- Respiratory gas exchange data were assessed using an Metalyzer 3B Cortex, online breath by breath gas analyser (Metalyzer 3B; CORTEX Biophysik GmbH, Leipzig, Germany). Prior to all testing the Cortex analyser was calibrated with ambient air and known concentrations of O_2 (17%) and carbon dioxide (CO_2; 5%). The bidirectional turbine (flow meter) was calibrated with a 3-litre calibration syringe.

- The Cyclus2 ergometer (Leipzig, Germany), was pre-programmed to run the test protocols, and to collect the PO and HR data. Participants own bikes were fixed to the Cylus2 for all the test protocols.

- Garmin HR monitors (Garmin, Kansas, USA) were used to collect HR data via the Cyclus2 and then exported for analysis.

- The portable NIRS device (Portamon, Artinis Medical Systems, The Netherlands) used in all cases was a 3-wavelength continuous wave system, which simultaneously uses the Beer-Lambert and spatially resolved spectroscopy method. Changes in tissue O_2Hb, HHb and tHb were measured using the differences in absorption characteristics at 770, 850 and 905 nm (corresponding to the absorption wavelengths of O_2Hb and HHb).

- Hokanson rapid inflation system (Hokanson E20; Bellevue, WA, USA) was used to inflate the BP cuff (Hokanson SC12D; Bellevue, WA, USA) to a pressure of 300mmHg for all occlusions.

- B[La] samples were analysed using a Biosen C-Line (EKF Diagnostic, London, UK) and then safely disposed of in accordance with the Human Tissue Act.

- RPE measurements were taken using the Borg 6–20 scale (Borg, 1998). sPRE measurements were taken using the scale of 0-10 as proposed by Foster et al., (2001).

III.II – Determination of $\dot{V}O_{2max}$

Introduction

$\dot{V}O_{2max}$ is the highest physiological rate at which O_2 can be utilised by the body during aerobic exercise. An individual's $\dot{V}O_{2max}$ establishes the upper boundary of their aerobic capacity and has long been a prerequisite for endurance performance (Saltin & Astrand, 1967). Alongside, the lactate threshold (LT), performance O_2 deficit and gross efficiency, the $\dot{V}O_{2max}$ determines an athlete's performance velocity or PO (Joyner & Coyle, 2008). It is also currently accepted as the gold standard for assessing cardiorespiratory fitness (Beltz et al., 2016). The first test of $\dot{V}O_{2max}$ was conducted by Hill & Lupton, (1923), and has since been widely used in exercise physiology laboratories around the world to classify the fitness levels of study participants and athletes (Paton & Hopkins, 2001). $\dot{V}O_{2max}$ can be reported as an absolute value; litres of O_2 per minute ($L.min^{-1}$) or as a value relative to the individuals body mass; millilitres of O_2 per kilogram of body mass per minute ($ml.kg.min^{-1}$).

Methods

The $\dot{V}O_{2max}$ test protocol started with a 10-min warm-up at 100 W, after which the required cycling PO was increased by 20 W every 1-min until the participant reached volitional exhaustion (operationally defined as a cadence of < 60 revolutions/min for > 5-s, despite strong verbal encouragement). After volitional exhaustion participants were allowed to cool down for 10-min at a self-selected PO.

PO and HR were measured continuously throughout the test, with RPE measurements asked in the last 10-s of each 1-min stage of the test, using the Borg 6-20-point scale (Borg, 1982).

Data Analysis

The participant's $\dot{V}O_{2max}$ were assessed as the highest pulmonary O_2 uptake ($\dot{V}O_2$; $L.min^{-1}$) that was attained during a 60-s period in the test and reported as absolute ($L.min^{-1}$) and relative ($ml.kg.min^{-1}$) values. In the absence of a plateau in $\dot{V}O_2$, secondary criteria were used (Poole et al., 2008).

Maximal minute power (MMP) was assessed as the highest 1-min PO achieved during the test. HRmax was assessed as the highest 1-min HR achieved during the test.

III.III – NIRS data collection during the HIIT sessions

Introduction

NIRS, is a well-known non-invasive method used to measure muscle oxygenation which reflects the ratio of O_2 delivery to the working muscle and $m\dot{V}O_2$ in the capillary beds (Hamaoka et al., 1996; Jones et al., 2016). NIRS devices provide information on relative changes in tissue oxyhaemoglobin (O_2Hb), deoxyhaemoglobin (HHb) and total haemoglobin (tHb) at rest and during exercise (Ferrari et al., 2011). However, only through using muscle occlusion techniques can the rate $m\dot{V}O_2$ (i.e. the muscles oxidative capacity) be estimated (Ryan et al., 2012). NIRS works on the basis that O_2Hb and HHb have different light absorption characteristics and measurements can be made depending on the changes in the absorption of the light received by the probe (Jones et al., 2016).

The small size of the NIRS device has allowed researchers to investigate a wide range of skeletal muscles, in particular those of the upper and lower limbs (Ferrari et al., 2011). The vastus lateralis (VL) muscle has often been used in exercise physiology research, due to its large size and importance in lower limb locomotion (Buchheit & Ufland, 2011; Puente-Maestu et al., 2003; Ichimura et al., 2006). NIRS was incorporated into the current studies as it provides a practical non-invasive method to assess the acute peripheral responses to HIIT and has been used effectively in previous HIIT research investigating the recovery interval components (Christmass et al., 1999; Stanley & Buchheit, 2014; McLean et al., 2016; Kriel et al., 2016; Ohya et al., 2013; Buchheit et al., 2009). Moreover, the VL muscle was considered to be the most appropriate muscle from which to collect NIRS data in the current studies, due to the use of cycling exercise and arterial occlusions.

Methods

The NIRS optode was placed over the right VL muscle, approximately 8 cm from the knee joint on the vertical axis. The NIRS optode was affixed using kinesio tape (Kinesio Precut, Albuquerque, NM, USA) and a velcro strap to prevent movement. The optode was covered with a soft black cloth to prevent signal contamination from external light sources. Skinfold thickness at the site of application of the NIRS optode was determined before the testing sessions using Harpenden skinfold callipers (British indicators Ltd, Burgess Hill, UK).

Prior to the commencement of each HIIT session participants adopted a standardised resting position, seated with the knee flexed at 90^{o} for a 2 min period, during which baseline NIRS parameters were established.

The BP cuff was placed at the top of the right thigh to obstruct the femoral artery, proximal to the NIRS device. To normalise the NIRS signal, a 5-min arterial occlusion was applied using the BP cuff (Hokanson SC12D; Bellevue, WA, USA) connected to a rapid-inflation system (Hokanson E20; Bellevue, WA, USA), to completely deoxygenate the tissue under the NIRS optode (i.e., 0% oxygenation; O_2Hbmin and HHbmax). The peak hyperemic response upon release of the BP cuff indicated 100% oxygenation (O_2Hbmax). Participants remained seated for 5-min after the arterial occlusion. The BP cuff was then removed before the HIIT sessions began.

During the HIIT sessions NIRS data were acquisitioned via Bluetooth connection to a personal laptop and then exported at 10 Hz.

Data Analysis

NIRS data were analysed using a custom written excel spreadsheet. O_2Hb and HHb were converted to percentages using the ischemic calibration data from the 5-min occlusion (formulas 1 and 2).

$$[1]\ \%\ \mathrm{O2Hb} = \frac{(\mathrm{O2Hb} + \mathrm{HHbmax})}{(\mathrm{O2Hbmax} - \mathrm{O2Hbmin})}\ x\ 100$$

$$[2]\ \%\ \mathrm{HHb} = \frac{(\mathrm{HHb} + \mathrm{O2Hbmax})}{(\mathrm{O2Hbmax} - \mathrm{O2Hbmin})}\ x\ 100$$

The following measures: % O_2Hb, % HHb, muscle tissue saturation % (TSI%) were then averaged over each work and recovery interval of the HIIT session. The minimum and maximum 30-s average for each NIRS measure were calculated. The change in % O_2Hb, % HHb and TSI%, from the last 30-s of the work interval to the last 30-s of following recovery interval were calculated across the HIIT sessions.

III.IV – HIIT session protocols and data collection methods

Introduction

The field of HIIT research has produced a diverse range of HIIT protocols. Work interval durations can range from 4-s to ≥ 10-min, with work interval intensity often based on a fixed percentage of $p\dot{V}O_{2max}$ or $v\dot{V}O_{2max}$ (Buchheit & Laursen, 2013). It has been suggested that longer work intervals ≥ 2-min allow for a greater amount of accumulated $T@\dot{V}O_{2max}$ than shorter work interval durations (< 1-min; Buchheit & Laursen, 2013; Seiler & Sjursen, 2004; Vuorimaa et al., 2000). Moreover, many studies have utilised HIIT protocols with longer work interval durations, as they are reported to be consistent with HIIT sessions performed by endurance athletes (Buchheit et al., 2012; Buchheit & Laursen, 2013; Demarie et al., 2000; Millet et al., 2003; Seiler & Hetlelid, 2005). Hence the selection of the 6 x 4-min and 3 x 8-min HIIT sessions used in the current studies, which are matched for total work interval duration.

While many studies continue to prescribe work interval intensity based on a fixed PO or velocity, there have been a growing number of studies investigating the acute physiological effects of HIIT using a 'maximal session effort' prescription approach (Laurent et al., 2014; Seiler & Hetlelid, 2005; Seiler et al., 2013; Smilios et al., 2017). In line with the aforementioned HIIT research, the work interval intensity for the current studies were also prescribed on a 'maximal session effort' basis.

Methods

Prior to starting the HIIT sessions participants were fitted with the NIRS device, which was worn for the full duration of the HIIT session. Full methods for NIRS data collection during the HIIT sessions can be found in General methods, Section III.III - NIRS data collection during the HIIT sessions.

All HIIT sessions started with a 10-min warm-up at 100W, at the participants self-selected cadence. The face mask used to collect respiratory gas data was fitted to the participant 2-min before the end of the warm-up and was worn until the final work interval of the session was completed. All HIIT sessions finished with a 10-min cool down at 100W.

The HIIT sessions used were 6 x 4-min and 3 x 8-min work intervals. HIIT session schematics are shown in figures 3.1 and 3.2. Both HIIT sessions have an equal work duration of 24-min. Work intervals were self-paced on a 'maximal session effort' basis. Participants were instructed to ride as hard as possible during the work intervals and to achieve the highest PO possible. Participants could self-select their cadence during all HIIT sessions. Consistent verbal encouragement was given throughout every session. The number of HIIT sessions completed in each study are detailed in the respective experimental chapters.

PO and HR were measured continuously throughout the HIIT sessions.

B[La] samples were taken via the fingertip capillary. Samples were collected pre warm-up and during the last 30-s of each work interval.

RPE measurements were asked during the last 15-s of each work interval. sRPE measurements were asked at the end of the 10-min cool down. Participants were instructed to NOT include the warm-up and cool down in their sRPE rating.

The details of how the recovery interval components were manipulated is described in the relevant experimental chapters. Recovery interval duration – V. Experimental Chapter – Study One, recovery interval intensity – VI. Experimental Chapter – Study Two.

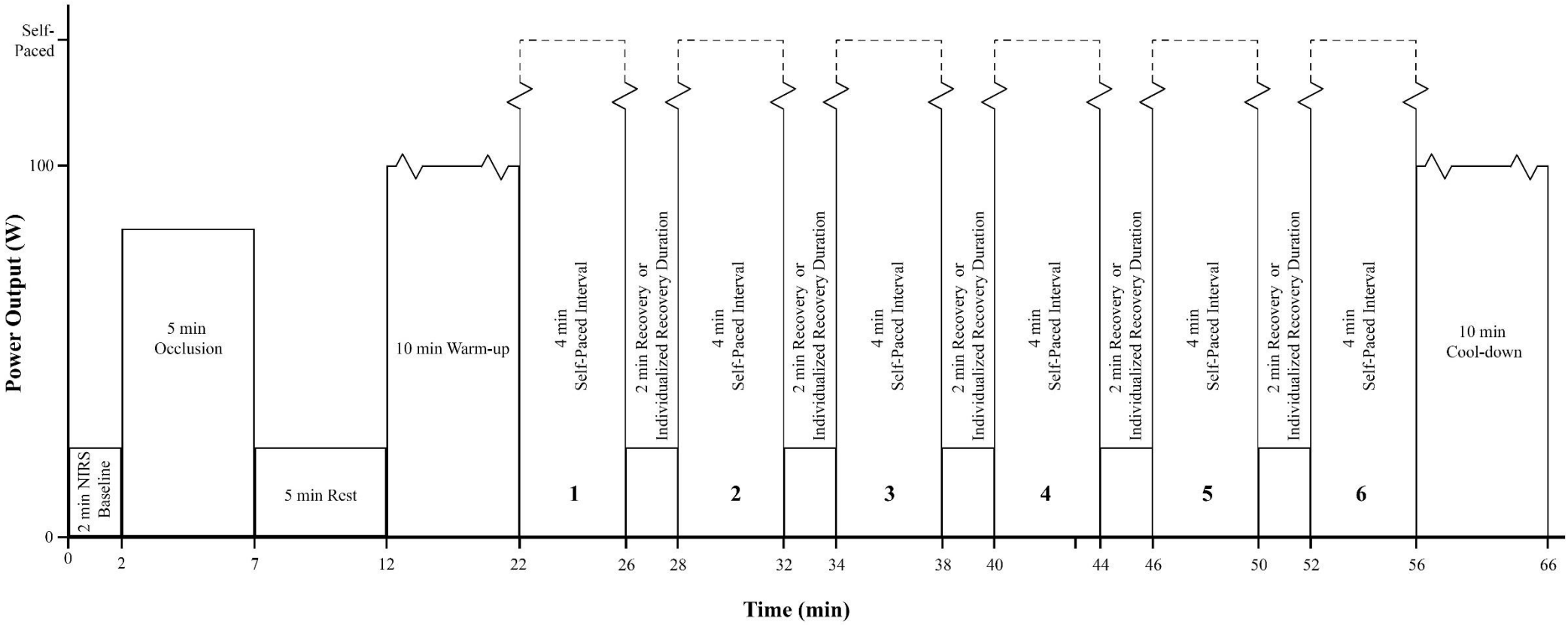


Figure 3.1 – Schematic for the 6 x 4-min HIIT session.

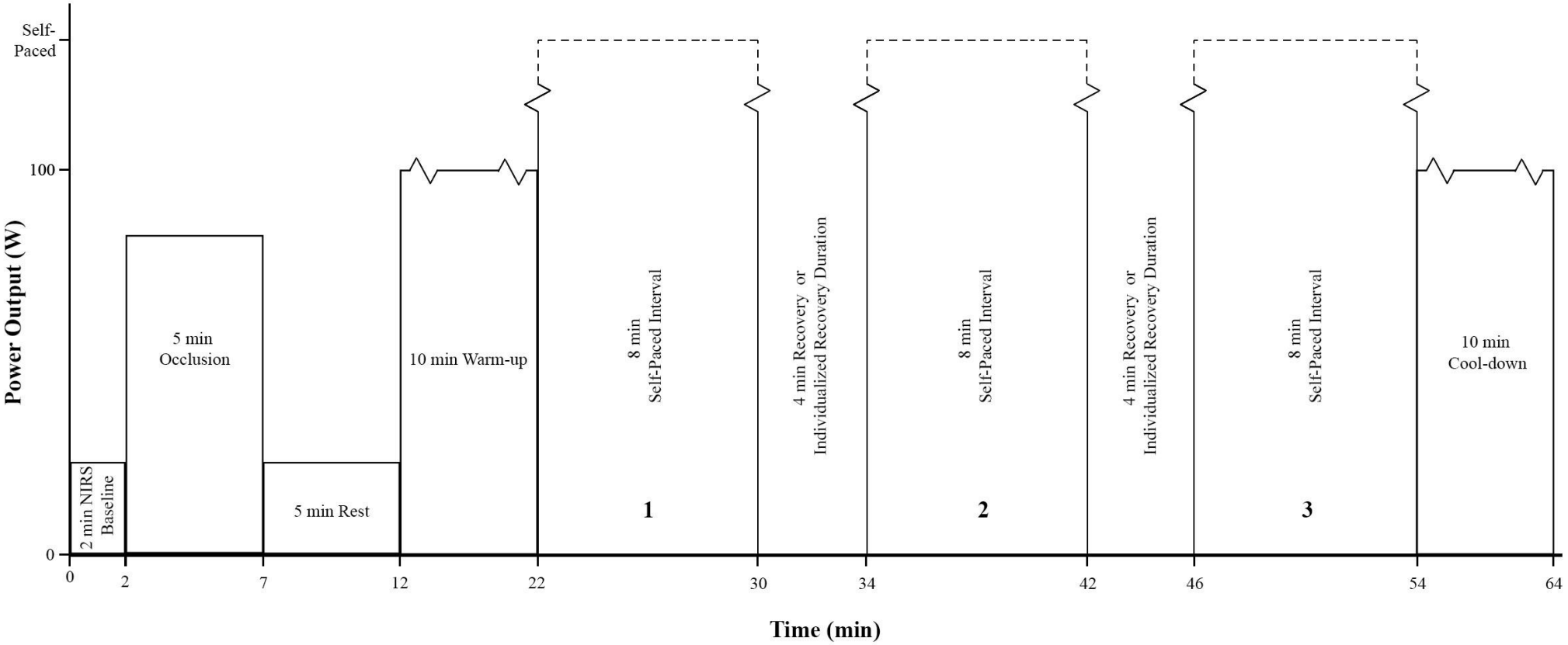


Figure 3.2 – Schematic for the 3 x 8-min HIIT session.

Data Analysis

PO data were averaged over each work interval and the whole session. HR data were averaged over each work and recovery interval and the whole session. The minimum and maximum average 30-s HR from the work and recovery intervals were calculated. In addition, the average HR from the last 30-s of the work and recovery intervals were calculated.

Work and recovery interval averages for respiratory gas data ($\dot{V}O_2$, Relative $\dot{V}O_2$, Pulmonary ventilation [$\dot{V}_E$] and Bf) were calculated. The minimum and maximum 30-s average attained during all work and recovery intervals for each respiratory gas measure were calculated. In addition, a 30-s average taken at the end of the work and recovery intervals were calculated for each respiratory gas measure.

The time spent above 60, 70, 80, 90 and 95% of MMP, $\dot{V}O_{2max}$ and HRmax were calculated across the total duration of the work intervals and the total duration of the HIIT session.

NIRS data were analysed as described in III. General methods, Section III.III - NIRS data collection during the HIIT session.

IV. Methods for the determination of $m\dot{V}O_2$ recovery duration

IV.I – Introduction

The use of NIRS to assess muscle reoxygenation rates after dynamic exercise has been the interest of several studies over previous years (Buchheit et al., 2011; Buchheit & Ufland, 2011; Costes et al., 2001; Neary et al., 2001, 2002 & 2005; Puente-Maestu et al., 2003; Ichimura et al., 2006). However, reoxygenation rates using the raw NIRS signal only reflects the ratio of O_2 delivery and O_2 consumption in the working muscles, consequently the influence of the two factors on muscle reoxygenation kinetics cannot be deciphered (Hampson & Piantadosi, 1988; Belardinelli et al., 1995). Therefore, to examine the recovery of $m\dot{V}O_2$ after exercise, a method based on repeated transient arterial occlusions was proposed (Motobe et al., 2004). The rate of muscle deoxygenation during the arterial occlusions can be used to estimate $m\dot{V}O_2$. Through plotting the changes in $m\dot{V}O_2$ across repeated occlusions, the time course of $m\dot{V}O_2$ recovery after a specific exercise bout can be estimated (Buchheit et al., 2011). During arterial occlusions blood volume changes have been suggested to confound the slope measurements for O_2 consumption (De Blasi et al., 1997; Van Beekvelt et al., 2001). To ensure the metabolic exchange between O_2Hb and HHb is not masked by blood changes under the NIRS probe, Ryan et al., (2012) proposed a method to correct for blood volume changes. The application of the blood volume correction to the NIRS data provides consistent and reliable signals for O_2Hb and HHb during the arterial occlusions (Ryan et al., 2012).

The reliability of $m\dot{V}O_2$ recovery after whole body exercise has been shown to have a coefficient of variation (CV) ranging from 6 to 16% (Buchheit et al., 2011). To limit the

potential for day-to-day variation in the measurement of m$\dot{V}O_2$ recovery it is important that participants, maintain the same posture during the occlusion process (Bringard et al., 2006) and there is consistent placement of the NIRS probe on the selected muscle body.

The following methods were used to determine each participant's m$\dot{V}O_2$ recovery duration which will be used as the IND recovery duration, to be applied to the HIIT sessions in study one (V. Experimental Chapter – Study One).

IV.III – Methods

The NIRS optode was placed over the right VL muscle, approximately 8 cm from the knee joint on the vertical axis. The NIRS optode was affixed using kinesio tape (Kinesio Precut, Albuquerque, NM, USA) and a velcro strap to prevent movement. The optode was covered with a soft black cloth to prevent signal contamination from external light sources. Skinfold thickness at the site of application of the NIRS optode was determined before the testing sessions using Harpenden skinfold callipers (British indicators Ltd, Burgess Hill, UK).

Prior to the commencement of exercise participants adopted a standardised resting position, seated with the knee flexed at 90^{o} for a 2 min period, during which baseline NIRS parameters were established.

The BP cuff was placed at the top of the right thigh to obstruct the femoral artery, proximal to the NIRS device. To normalise the NIRS signal, a 5-min arterial occlusion (Ischemic calibration) was applied using the BP cuff (Hokanson SC12D; Bellevue, WA, USA) connected to a rapid-inflation system (Hokanson E20; Bellevue, WA, USA), to completely deoxygenate the tissue under the NIRS optode (O_2Hbmin and HHbmax). The peak hyperemic response upon release of the BP cuff indicated O_2Hbmax. Participants remained seated for 5-min after the arterial occlusion. To ensure the BP cuff remained in place during the exercise portion of the tests the participants were instructed to roll the leg of their cycling shorts over the deflated BP cuff, which was then also affixed with adhesive tape.

After warming up at 100 W for 10-min the participants completed a single self-paced 4-min interval (on a consistent 'maximal effort basis'). Immediately following the interval, a series of 20 brief (10s) arterial occlusions were applied to measure $m\dot{V}O_2$ recovery back to resting levels. To minimise the discomfort to participants, the duration between arterial occlusions began at 10s and extend to 20s by the end of the repeated occlusions (i.e. 10s for occlusions *1-10*, 15s for occlusions *11-15*, 20s for occlusions *16-20)* as recommended by Ryan et al., (2012). Participants were instructed to keep the leg under occlusion at the bottom of the pedal stroke, remaining completely still and to hold the same posture throughout the occlusion procedure. Figure 4.1 displays the schematic for the repeated occlusion protocol.

After cooling down at 100 W for 10-min, participants then completed a seated rest for 20-min before repeating the above protocol, this time completing a single self-paced 8-min interval (on a consistent 'maximal effort basis').

NIRS data were acquisitioned via Bluetooth connection to a personal laptop and then exported at 10 Hz. Expired gas data, PO and HR were continuously measured throughout the single self-paced work intervals (Data presented in *Table 4.1*).

The time taken for $m\dot{V}O_2$ to recover to 95% of baseline levels, after the 4-min and 8-min intervals were used as the participants IND recovery duration during the respective HIIT sessions in visits 3 to 6 (see *Calculation of individual $m\dot{V}O_2$ recovery duration*; for description of how $m\dot{V}O_2$ recovery duration was calculated).

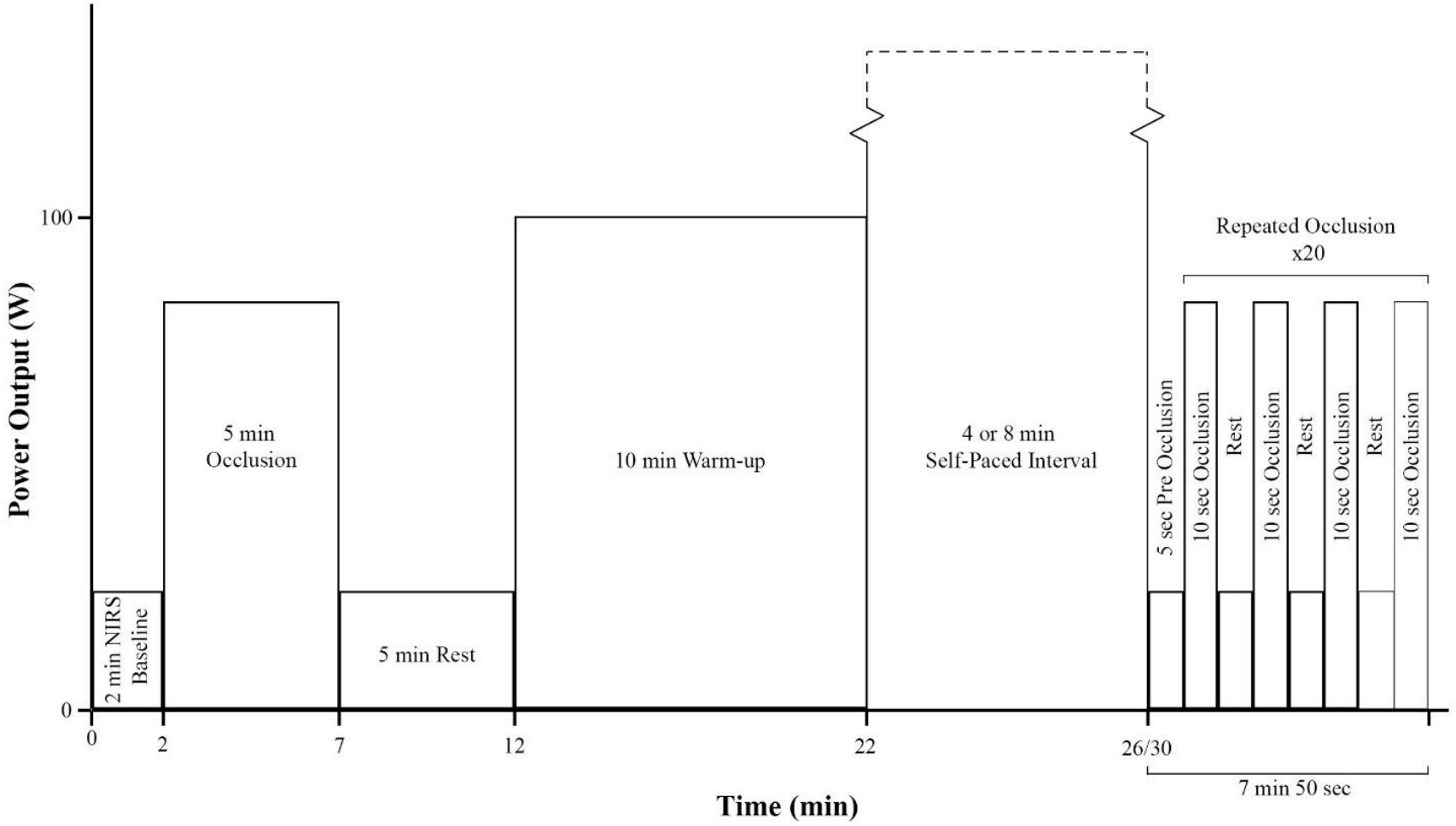


Figure 4.1 – Schematic for the repeated occlusion protocol.

IV.IV – Data Analysis

Correction of blood volume

NIRS data were analysed using a custom written excel spreadsheet. The method of blood volume correction as previously described by Ryan et al., (2012) were applied to the NIRS data. The application of blood volume correction factor assumes that during an arterial occlusion, the changes in O_2Hb and HHb occur with a 1:1 ratio that represents mitochondrial O_2 consumption only, making the area under the NIRS optode a closed system (Ryan et al., 2012).

Equation. 1 below describes the calculation of the blood volume correction factor (β: which corrects the NIRS signal for changes in blood volume, proportioned into oxygenated and deoxygenated sources):

$$[1]\ \beta(t) = \frac{\mathrm{O2Hb(t)}}{(\mathrm{O2Hb(t)+HHb}(t))}$$

β = blood volume correction factor, *t* = time, O_2Hb = oxygenated haemoglobin/myoglobin signal, HHb = deoxygenated haemoglobin/myoglobin signal.

The β was calculated for each data point to account for small changes in the proportionality of the blood volume change throughout a cuff. Each data point was then corrected using the corresponding β according to equations 2 and 3 below:

[2] Corrected $O_2Hb = O_2Hb – [tHb \times (1 – \beta)]$

[3] Corrected $HHb = HHb – (tHb \times \beta)$

Figure 4.2 displays an example of NIRS data collected during the repeated occlusion protocol, which has been corrected for blood volume.

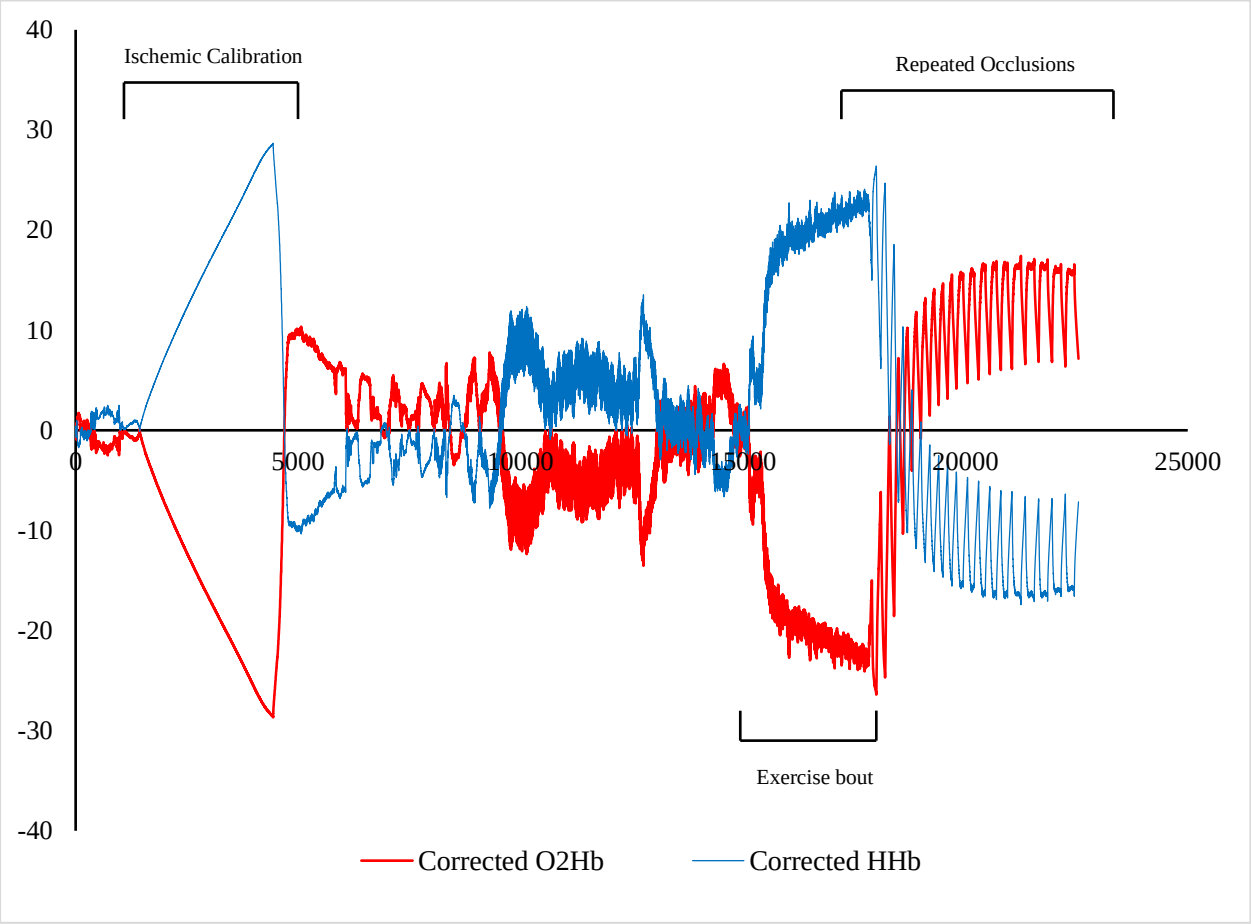


Figure 4.2 – Example of NIRS data corrected for blood volume, from the determination of $m\dot{V}O_2$ recovery duration protocol.

Calculation of $m\dot{V}O_2$

$m\dot{V}O_2$ was calculated as the initial slope of change in corrected HHb during the arterial occlusion using simple linear regression (Ryan et al., 2012). To calculate the initial slope of change in corrected HHb, 50 data points from seconds 1 to 6 of the occlusion were used. Muscle density was assumed as 1.04 kg 1^{-1} (Van Beekvelt et al., 2001), the linear slope of increase in corrected HHb expressed in micromolar units ($\mu M\ s^{-1}$) was converted to millilitres O_2 per minute per 100g tissue ($ml.O_2.min^{-1}.100g^{-1}$) using the following equation (Equation. 4; Van Beekvelt et al., 2002):

[4] $m\dot{V}O_2 = ((HHb \times 60) / (10 \times 1.04) \times 4) \times 22.4 / 1000$

Data derived from the repeated arterial occlusions were then plotted versus recovery time to show the time course of $m\dot{V}O_2$ recovery after the 4-min and 8-min intervals (*Figure 4.3*). The first and second $m\dot{V}O_2$ values were systematically discarded as they did not provide accurate $m\dot{V}O_2$ values (This was likely due to O_2 stores being too low, confounding the assessment of an accurate $m\dot{V}O_2$).

Calculation of individual $m\dot{V}O_2$ recovery duration

The following method was used to calculate the participants $m\dot{V}O_2$ recovery duration. This method takes into account the differences in the rate of the participants $m\dot{V}O_2$ recovery, in addition to providing a consistent method of prescribing the IND recovery duration.

The participants IND recovery duration was calculated as the time at which the $m\dot{V}O_2$ recovery curve intercepts the 95% $m\dot{V}O_2$ value (for example see *Figure 4.3*). The 95% $m\dot{V}O_2$ value was calculated as 95% of the difference between the peak $m\dot{V}O_2$ value and the end $m\dot{V}O_2$ value. Half (50%) $m\dot{V}O_2$ recovery duration was calculated in the same way.

The slope between each $m\dot{V}O_2$ data point on the $m\dot{V}O_2$ curve was calculated. This allowed for an estimation of the rate of change in $m\dot{V}O_2$ per second across the $m\dot{V}O_2$ curve.

Formula [5] was used to calculate the $m\dot{V}O_2$ value at 95% of the $m\dot{V}O_2$ curve. The IND recovery durations were calculated as the time at which the $m\dot{V}O_2$ recovery curve intercepted the $m\dot{V}O_2$ value output from formula 5.

$$[5]\ m\dot{V}O2\ \text{value} = \left(\left(m\dot{V}O2\text{peak} - m\dot{V}O2\text{end}\right) - \left(\frac{(m\dot{V}O2\text{peak} - m\dot{V}O2\text{end})}{100} \times 95\right)\right) + m\dot{V}O2\text{end}$$

$m\dot{V}O2peak$ = first $m\dot{V}O_2$ value, $m\dot{V}O2end$ = last $m\dot{V}O_2$ value.

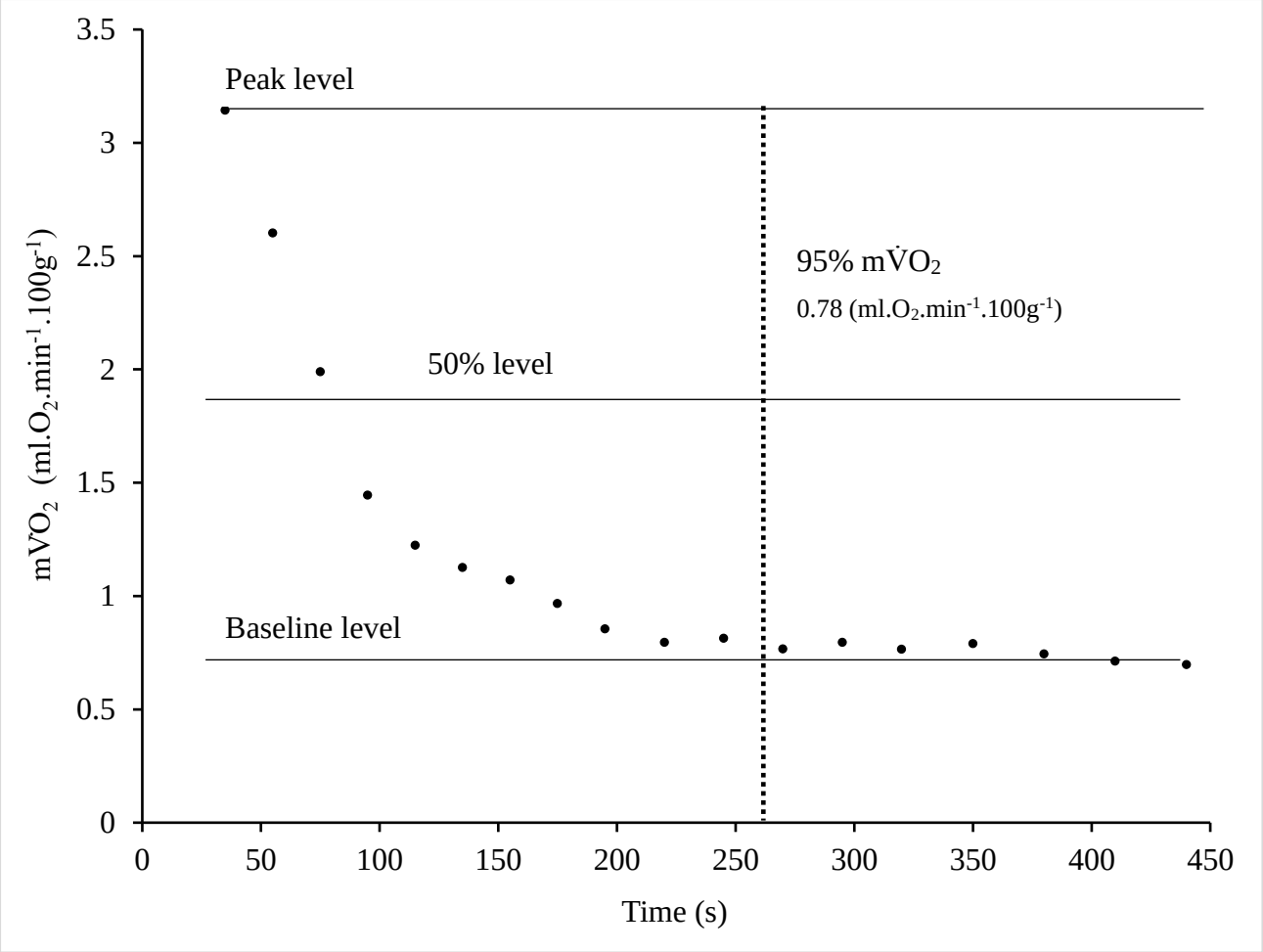


Figure 4.3 – Example of $m\dot{V}O_2$ recovery curve.

In this example the 95% $m\dot{V}O_2$ value output from Formula [5] was 0.78 ($ml.O_2.min^{-1}.100g^{-1}$). The time point at which the $m\dot{V}O_2$ curve intercepted 0.78 ($ml.O_2.min^{-1}.100g^{-1}$) provides the IND recovery duration (i.e. 260-s).

Table 4.1 displays the recovery duration and physiological data collected during the determination of $m\dot{V}O_2$ recovery duration protocols.

Table 4.1 – Determination of $m\dot{V}O_2$ recovery duration data (Mean ± SD).

Measurement	**Mean ± SD (CV%)**	
	4-min Interval	**8-min Interval**
$m\dot{V}O_2$ 95% Recovery duration (s)	205 ± 79 (39)	200 ± 81 (41)
$m\dot{V}O_2$ 50% Recovery duration (s)	69 ± 10 (14)	82 ± 20 (24)
Max post interval $m\dot{V}O_2$ ($ml.O_2.min^{-1}.100g^{-1}$)	2.77 ± 0.74 (26.7)	2.65 ± 0.61 (23.1)
Min post interval $m\dot{V}O_2$ ($ml.O_2.min^{-1}.100g^{-1}$)	0.55 ± 0.21 (37.8)	0.6 ± 0.32 (53.4)
PO (W)	330 ± 62 (19)	297 ± 56 (19)
Relative PO ($W.kg^{-1}$)	4.6 ± 0.7 (15.5)	4.1 ± 0.7 (16.3)
% MMP	88.2 ± 5.4 (6.1)	79.3 ± 4 (5)
Heart Rate (bpm)	166 ± 10 (6)	171 ± 10 (6)
% HRmax	88.3 ± 2.7 (3)	91.4 ± 3.2 (3.5)
$\dot{V}O_2$ ($L.min^{-1}$)	3.6 ± 0.6 (18.2)	3.7 ± 0.7 (18.6)
Relative $\dot{V}O_2$ ($ml.kg.min^{-1}$)	49 ± 8 (15)	51 ± 8 (16)
% $\dot{V}O_{2max}$	82.5 ± 6.3 (7.6)	85 ± 6.6 (7.8)
$\dot{V}_E$ ($L.min^{-1}$)	127.3 ± 31.2 (24.5)	127.7 ± 33.2 (26)
Bf ($breaths.min^{-1}$)	44.4 ± 9.4 (21.2)	45.6 ± 10 (21.9)

The $m\dot{V}O_2$ values measured immediately after the cessation of exercise were 2.77 and 2.65 $ml.O_2.min^{-1}.100g^{-1}$ for the 4-min and 8-min intervals respectively (*Table 4.1*). The minimum $m\dot{V}O_2$ values recorded during the measurement of $m\dot{V}O_2$ recovery duration were: 0.55 and 0.6 $ml.O_2.min^{-1}.100g^{-1}$ for the 4-min and 8-min intervals respectively (*Table 4.1*). While previous researchers have measured the reoxygenation response of the VL muscle (Puente-Maestu et al., 2003; Ichimura et al., 2006; Buchheit & Ufland, 2011), they did not report the $m\dot{V}O_2$ values in $ml.O_2.min^{-1}.100g^{-1}$, making comparisons to the current data difficult. However, in the study of Buchheit et al., (2011) the $m\dot{V}O_2$ of the gastrocnemius muscle was measured. They reported $m\dot{V}O_2$ values from 1.2 to 2.3 $ml.O_2.min^{-1}.100g^{-1}$ immediately after the cessation of exercise, which are comparable to the current $m\dot{V}O_2$ values. The current $m\dot{V}O_2$ values are also comparable to those reported using the same technique on the flexor digitorum superficialis muscle (van Beekvelt et al., 2002).

In the study of Buchheit et al., (2011), $m\dot{V}O_2$ was measured after exercise of varying intensities. As expected, $m\dot{V}O_2$ immediately post exercise was significantly greater after exercising at a higher intensity, when compared to a lower exercise intensity. Exercise intensity for the measurement of $m\dot{V}O_2$ recovery duration in the current study was prescribed as 'self-paced maximal effort'. The 'maximal effort' prescription resulted in high percentages of $\dot{V}O_{2max}$ and HRmax being attained during the 4-min and 8-min intervals (> 80% of max for both measurements; *Table 4.1*). The greater exercise intensity may explain the higher post exercise $m\dot{V}O_2$ in the current study when compared to previous research (Buchheit et al., 2011; van Beekvelt et al., 2002). While Ryan et al., (2012), found there was no difference in resting $m\dot{V}O_2$ between the VL and gastrocnemius muscles, to the authors knowledge the difference between $m\dot{V}O_2$ of the VL and gastrocnemius muscles immediately after high intensity exercise has not yet been established.

Furthermore, $m\dot{V}O_2$ has been shown to be up to 6 times greater after exercise, when compared to baseline/resting levels (Buchheit et al., 2011). In accordance, the current study shows $m\dot{V}O_2$ to be 4.5 to 5 times greater immediately post exercise, when compared to the baseline/resting $m\dot{V}O_2$ values measured during the recovery period.

Table 4.2 - Determination of $m\dot{V}O_2$ recovery duration NIRS data (Mean ± SD).

Measurement	**Mean ± SD (CV%)**	
	4-min Interval	**8-min Interval**
% O_2Hb	26.9 ± 9.4 (35.1)	26.2 ± 8.8 (33.7)
Change % O_2Hb	15.1 ± 7.2 (27.5)	24 ± 12.2 (51)
% HHb	73.1 ± 9.4 (12.9)	73.8 ± 8.8 (12)
Change % HHb	15 ± 7.2 (48)	25.1 ± 12.8 (51)
tHb	4.1 ± 7.7 (186.8)	4.1 ± 6.9 (168)
Change tHb	5.2 ± 4.3 (81.8)	5.3 ± 2.8 (54.1)
TSI %	56.9 ± 10.2 (18)	58.6 ± 8.3 (14.1)
Change TSI %	7.4 ± 5.1 (69.4)	11.7 ± 10.3 (88.2)

In the current study we present large CVs for m$\dot{V}O_2$ 95% recovery duration (39% and 41% for the 4-min and 8-min intervals respectively; *Table 4.1*). These large CVs are likely due to the variation in participant training status which has been shown to have an influence on muscle reoxygenation rates (Chance et al., 1992; Ding et al., 2001; Ichimura et al., 2006; Kounalakis et al., 2009). While inclusion criteria to the study stipulated the requirement of being a 'well trained cyclist' with > 2 years training and racing experience, this still allowed for meaningful differences in exercise capacity between individual participants ($\dot{V}O_{2max}$: 60 ± 7 ml.kg.min^{-1} and MMP 373 ± 57 W; see *Table 5.1*). Reoxygenation rates can be accelerated after training in line with changes which improve endurance performance; increases in muscle oxidative enzymes (Puente-Maestu et al., 2003), blood flow and capillarization (Costes et al., 2001; Kime et al., 2003). In addition, age also effects m$\dot{V}O_2$ recovery rate (Kutsuzawa et al., 2001), with the current studies participants aged from 18 to 55 years. It is therefore possible that age also effected the CV of m$\dot{V}O_2$ recovery duration in the current study.

V. Experimental Chapter – Study One

The impact of recovery interval duration on the acute physiological and perceptual responses to interval training during cycling exercise

V.I – Introduction

Interval training prescription comprises of six main components: work interval intensity, work interval duration, number of work intervals, recovery interval intensity, recovery interval duration, and overall session load (Buchheit & Laursen, 2013; Tschakert & Hofmann, 2013). The work interval components have received the greatest research attention as they ultimately facilitate the training stimulus produced by the HIIT protocol (Buchheit & Laursen, 2013; Tschakert & Hofmann, 2013). Interestingly, even though recovery interval duration is an important aspect of the overall HIIT prescription, there has been a paucity of research investigating this component of HIIT programming.

Optimal HIIT performance can only be achieved if adequate recovery is provided between work intervals, therefore understanding the acute responses to manipulating recovery durations are important when designing HIIT sessions (Schoenmakers & Reed, 2019). The prescription of an inadequate recovery duration will negatively affect the performance of the work intervals, while an excessive recovery duration will negatively affect the time efficiency of HIIT. The optimal recovery duration must therefore be a compromise, to maximise the training stimulus achieved during the work intervals, while reducing the total session duration.

At present researchers investigating the acute effects of recovery interval duration on HIIT performance have commonly used fixed recovery durations and/or work recovery ratios (i.e. 1:1 or 2:1) to prescribe recovery interval duration (Seiler & Hetlelid, 2005; Smilios et al., 2017; Schoenmakers & Reed, 2018; Laurent et al., 2014). In an attempt to individualise recovery interval duration, researchers have used the return of HR to a set value (Edwards et al., 2011) and self-selected recovery durations (Schoenmakers & Reed, 2018; Seiler & Hetlelid, 2005; McEwan et al., 2018; Brownstein et al., 2018; Gibson et al., 2017). However, it has been suggested that these methods are not appropriate for individualising recovery interval duration (for explanation see V. Literature review, section II.IV – The recovery interval: Duration).

There is currently no robust physiological method for individualising HIIT recovery interval duration. The current study therefore sought assess whether the recovery duration of $m\dot{V}O_2$ would provide a method of individualising the duration of the recovery intervals within a HIIT session. With the IND recovery interval duration theoretically being the optimal recovery duration for the specific individual and HIIT protocol. Measurements of $m\dot{V}O_2$ are derived directly from the exercising muscle, with recovery of $m\dot{V}O_2$ indicating an equilibrium in O_2 delivery and consumption, thus no competition and/or inhibition of available O_2 supplies at the start of exercise (Buchheit et al., 2011). It has been suggested that the recovery duration of $m\dot{V}O_2$ after high intensity exercise is likely related to a greater depletion ATP, PCr and/or myoglobin O_2 stores, which logically take longer to be restored. In addition, it is possible that $m\dot{V}O_2$ remains elevated above baseline values after high intensity exercise to compensate for the detrimental effect of a decreased muscle pH on PCr recovery (Graaf et al., 2007; McMahon & Jenkins, 2002). The recovery rate of $m\dot{V}O_2$ also takes into account the intensity of the prior exercise (Buchheit et al., 2011), the individuals training status (Chance

et al., 1992; Ding et al., 2001; Ichimura et al., 2006; Kounalakis et al., 2009) and age (Kutsuzawa et al., 2001). Therefore, by using the recovery duration of $m\dot{V}O_2$ to individualise the recovery interval duration, participants should be commencing the subsequent work intervals with the muscles required for exercise close to a state of metabolic homeostasis.

Statement of Purpose

The main aim of the study was to investigate whether individualising the duration of the recovery interval based on the participants $m\dot{V}O_2$ recovery duration would maximise the performance of the work intervals and the acute physiological response to long work interval HIIT sessions, when compared to a STD recovery duration (2:1 work recovery ratio).

Statement of Hypothesis

Null Hypothesis: The IND recovery duration will have no effect on work interval PO and will not result in a greater acute physiological response, when compared to the STD recovery duration. As measured by mean work interval and HIIT session: HR, B[La], RPE, sRPE, $\dot{V}O_2$, T@$\dot{V}O_{2max}$, T@HRmax, HHb, O_2Hb and TSI %.

Alternative Hypothesis: The IND recovery duration will produce a higher work interval PO and will therefore result in a greater acute physiological response, when compared to the STD recovery duration (2:1 work recovery ratio). As measured by mean work interval and HIIT session: HR, B[La], RPE, sRPE, $\dot{V}O_2$, T@$\dot{V}O_{2max}$, T@HRmax, HHb, O_2Hb and TSI %.

V.II – Methods

Participants

Sixteen trained cyclists were recruited to take part in the study. The participants characteristics and anthropometrics are presented in table 5.1.

All participants had a minimum of 2 years competitive racing experience and were in training for the next competitive season. In addition, all participants were using HIIT in their current training programmes (see *Table 5.1*).

Participants were required to complete a cycling experience questionnaire to ensure they met the inclusion criteria. The study was completed with full ethical approval from the University of Kent ethics committee, according to the Declaration of Helsinki standards. All participants provided signed informed consent and completed a health questionnaire, prior to testing to ensure they were in full health and able to deal with the exercise demands of the study.

Table 5.1 – Participant characteristics/anthropometrics, $\dot{V}O_{2max}$ test and cycling history questionnaire results.

N = 16	Mean ± SD
Age (yrs.)	32 ± 13
Height (cm)	177.9 ± 5.2
Mass (kg)	72.4 ± 9.1
VL Skin Fold (mm)	8.8 ± 2.1
Thigh Circumference (cm)	53 ± 6.6
$\dot{V}O_{2max}$ ($L.min^{-1}$)	4.3 ± 0.6
Relative $\dot{V}O_{2max}$ ($ml.kg.min^{-1}$)	60 ± 7
MMP (W)	373 ± 57
Relative MMP ($W.kg^{-1}$)	5.2 ± 0.7
HRmax (bpm)	188 ± 12
Years training	5.6 ± 4.4
Years competing	5.3 ± 3.5
Mean weekly training hours	10.1 ± 4.4
Hours of HIIT per Week	4.8 ± 1.7

Study Design

Each participant completed six visits to the laboratory: Visit 1 being an incremental exercise test to identify $\dot{V}O_{2max}$ and familiarise the participants with the laboratory environment and equipment. Visit 2, was the determination of the participants IND recovery duration. Visits 3 to 6 were the 6 x 4-min and 3 x 8-min HIIT sessions, two with the STD recovery duration and two with the participants IND recovery duration.

Visits were conducted on non-concurrent days and participants were instructed to refrain from any exercise in the day prior to testing and intense exercise in the two days prior. Participants were instructed to arrive euhydrated for each visit as they would be unable to drink for the duration of the exercise portion of the visit (due to wearing the face-mask to collect expired gases). Participants were advised to arrive in a post-prandial state, having eaten at least 4-hours prior to testing and were told to not consume caffeine within 4-hours and alcohol within 24-hours of testing.

Each participant completed all their visits to the laboratory at the same time of day to avoid any circadian variance. At each visit room temperature, humidity and pressure (mmHg) were recorded. An electric fan was placed 2 m in front of the participants to provide cooling during all tests if requested.

Visit 1: $\dot{V}O_{2max}$ Test

At the first visit, participants were measured for anthropometric values: height and mass. The participants then completed the $\dot{V}O_{2max}$ test, procedures for which are described in chapter III. General methods, section III.II – Determination of $\dot{V}O_{2max}$. Following the $\dot{V}O_{2max}$ test, participants were briefed on the procedures to be used in visits 2 to 6. Participants were also familiarised with the arterial occlusions, to ensure they were comfortable with the procedure. The circumference of the top of the right thigh was measured to make sure the participants were fitted with the correct sized BP cuff, to ensure the fullest occlusion could be applied.

Visit 2: The determination of m$\dot{V}O_2$ recovery duration

At visit 2 the participants IND recovery duration was measured. See chapter *IV. Methods for the determination of m$\dot{V}O_2$ recovery duration,* for full description of the procedures used to determine each participants IND recovery duration for the 6 x 4-min and 3 x 8-min HIIT sessions.

Visits 3 to 6: HIIT sessions

Participants completed both the 6 x 4-min and 3 x 8-min HIIT sessions twice (4 HIIT sessions in total), once with the STD recovery duration and once with the IND recovery duration. HIIT session methods and schematics can be found in chapter III. General Methods, section III.IV - HIIT session protocols and data collection methods.

Full methods for NIRS data collection during the HIIT sessions can be found in chapter III. General methods, section III.III - NIRS data collection during the HIIT sessions.

The STD recovery durations used were 120-s and 240-s for the 6 x 4-min and 3 x 8-min HIIT sessions respectively (i.e. the commonly prescribed 2:1 work recovery ratio; Seiler & Hetlelid, 2005). The participants IND recovery durations were 205 ± 79-s and 200 ± 81-s for the 6 x 4-min and 3 x 8-min HIIT sessions respectively (*Table 4.1*), as measured in visit 2 (see chapter *IV. Methods for the determination of m$\dot{V}O_2$ recovery duration).*

All recovery intervals were PA intensity. The HIIT sessions were randomised and completed on non-consecutive days.

Statistical Analysis

Data were presented as individual values or mean ± SD (unless specified otherwise). Statistical analyses were conducted using IBM SPSS Statistics 24 (IBM, Armonk, New York, USA). Visual inspection of Q-Q plots and Shapiro-Wilk statistics were used to check whether data were normally distributed. Three separate two-way repeated measure analysis of variance (ANOVA), 1) two HIIT protocols (6 x 4-min vs 3 x 8-min) X two recovery durations (STD vs IND); 2) two recovery durations (STD vs IND) X number of work intervals; 3) two recovery durations (STD vs IND) X number of recovery intervals were used to determine between and within condition effects for all dependent variables. Bonferroni *post hoc* comparisons were used when a main effect or interaction was significant. The criteria of $P < 0.05$ was used for the detection of significance in all cases.

V.III – Results

V.III.1 – Key physiological HIIT session results

Power Output Results

There was no interaction between HIIT protocol and recovery duration for mean PO (main effect $F < 0.001$, $P = 0.985$). Mean PO was significant higher during the 6 x 4-min HIIT protocols when compared to the 3 x 8-min HIIT protocols (main effect of protocol $F = 58.296$, $P < 0.001$). There was no effect of recovery duration (main effect of duration $F = 4.086$, $P = 0.061$), showing that there was no significant difference between the STD and IND recovery durations for the 6 x 4-min and 3 x 8-min HIIT protocols (*Figure 5.1*).

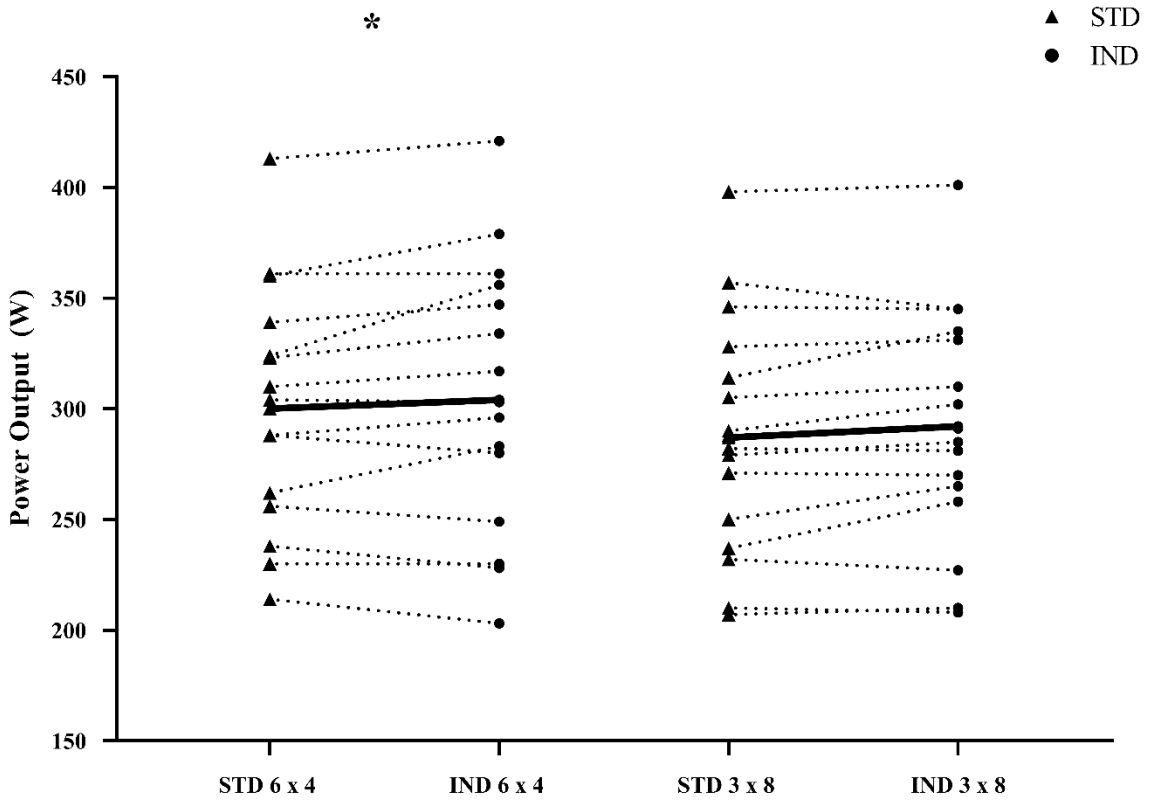


Figure 5.1 – Mean PO during the 6 x 4-min and 3 x 8-min HIIT sessions (Solid line = Mean), *** = *P* < 0.05**.

There was a significant effect of interval during the 6 x 4-min HIIT sessions (main effect of interval $F = 7.333$; $P < 0.001$), showing that there was a significant difference in the mean PO between work intervals (*Figure 5.2a*). There was no effect of interval during the 3 x 8-min HIIT sessions (main effect of interval $F = 2.528$, $P = 0.096$), showing that there was no difference in mean PO between work intervals (*Figure 5.2b*).

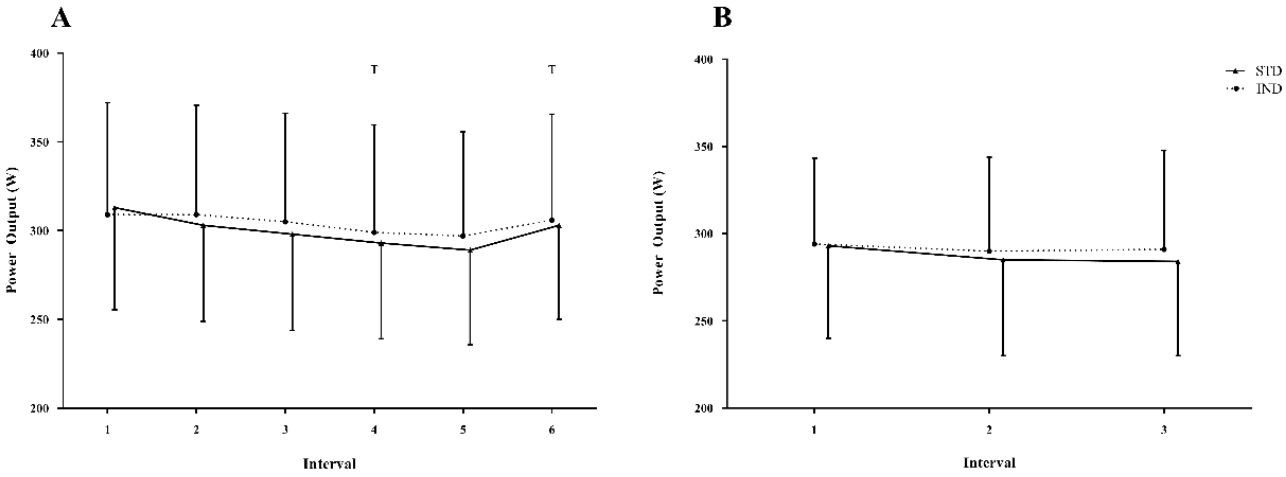


Figure 5.2 – A = Mean work interval PO during the 6 x 4-min HIIT sessions, **B** = Mean work interval PO during the 3 x 8-min HIIT sessions (Mean ± SD). T = Significant difference from previous interval.

Heart Rate Results

There was a significant interaction between HIIT protocol and recovery duration for mean session HR (main effect $F = 4.944$, $P = 0.046$). There was no significant difference in mean session HR between the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of protocol $F = 0.339$, $P = 0.571$). There was no effect of recovery duration (main effect of duration $F = 3.698$, $P = 0.079$). *Post hoc* tests revealed that mean session HR was significantly higher during the STD 6 x 4-min HIIT sessions, compared to the IND 6 x 4-min HIIT sessions ($P < 0.05$; *Figure 5.3*).

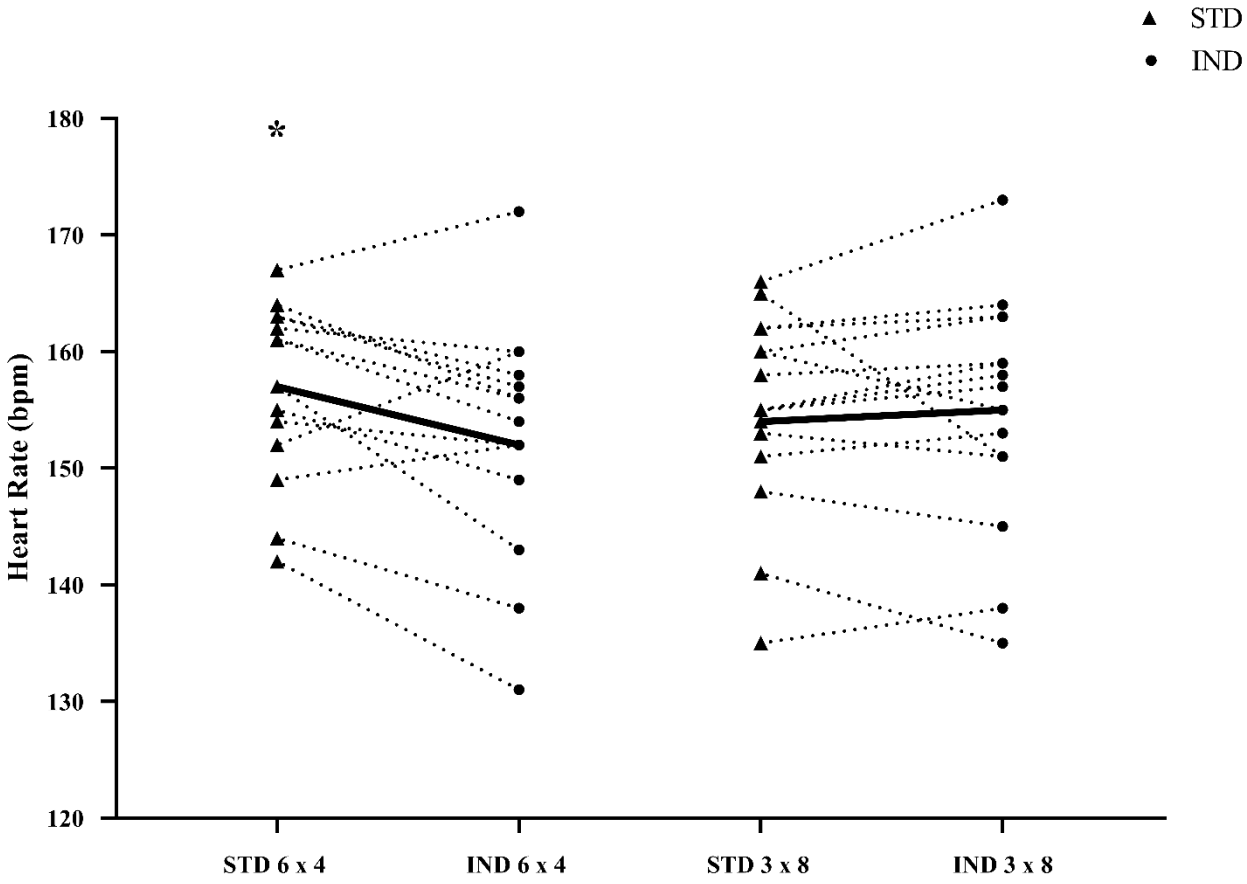


Figure 5.3 – Mean session HR during the 6 x 4-min and 3 x 8-min HIIT sessions (Solid line = Mean). *** = *P* < 0.05**.

There was no interaction between recovery duration and work interval during the 6 x 4-min (main effect $F = 0.774$, $P = 0.572$; *Figure 5.4a*) and 3 x 8-min HIIT protocols (main effect $F = 3.249$, $P = 0.054$; *Figure 5.4b*). There was an effect of interval during the 6 x 4-min (main effect of interval $F = 43.291$, $P < 0.001$; *Figure 5.4a*) and 3 x 8-min HIIT protocols (main effect of interval $F = 40.044$, $P < 0.001$; *Figure 5.4b*), showing that there was a significant increase in work interval HR across the HIIT sessions during the STD and IND recovery durations.

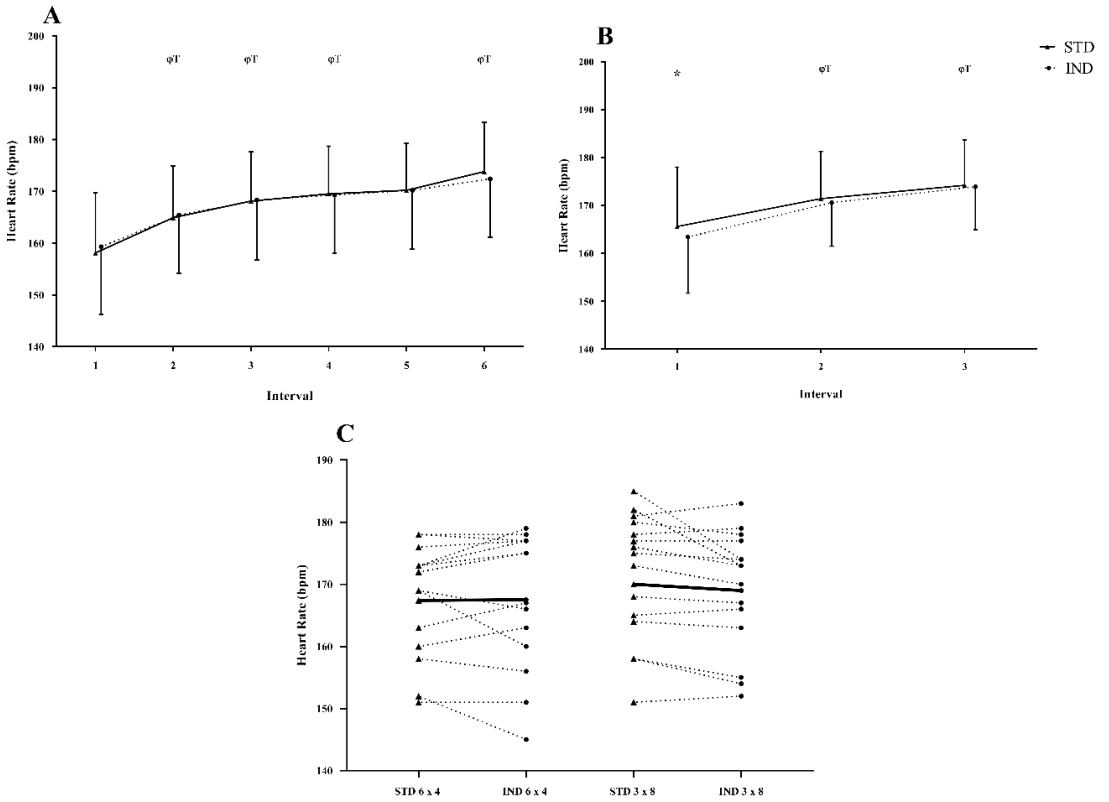


Figure 5.4 – A = Work interval HR during the 6 x 4-min HIIT sessions, **B** = Work interval HR during the 3 x 8-min HIIT sessions (Mean ± SD), **C** = Mean work interval HR during the 6 x 4-min and 3 x 8-min HIIT sessions (Solid line = Mean). φ = Significant difference from interval 1, T = Significant difference from previous interval, * = significant difference between recovery conditions (***P* < 0.05**).

There was no interaction between HIIT protocol and recovery duration for mean work interval HR (main effect $F = 2.022$, $P = 0.18$; *Figure 5.4c*). Mean work interval HR was significantly higher during the 3 x 8-min HIIT protocols when compared to the 6 x 4-min HIIT protocols (main effect of protocol $F = 11.975$, $P = 0.005$; *Figure 5.4c*). Recovery duration had no effect on mean work interval HR, with no significant differences found between the STD and IND recovery durations, for both the 6 x 4-min and 3 x 8-min HIIT session (main effect of duration $F = 1.279$, $P = 0.28$; *Figure 5.4c*).

B[La] Results

There was no interaction between HIIT protocol and recovery duration for the mean B[La] response (main effect $F = 0.001$, $P = 0.982$). There was no significant difference in mean B[La] response between the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of protocol $F = 0.062$, $P = 0.807$; *Figure 5.5*). Recovery duration had no significant effect on the mean B[La] response during both the 6 x 4-min and 3 x 8-min HIIT sessions (main effect of duration $F = 0.243$, $P = 0.630$; *Figure 5.5*).

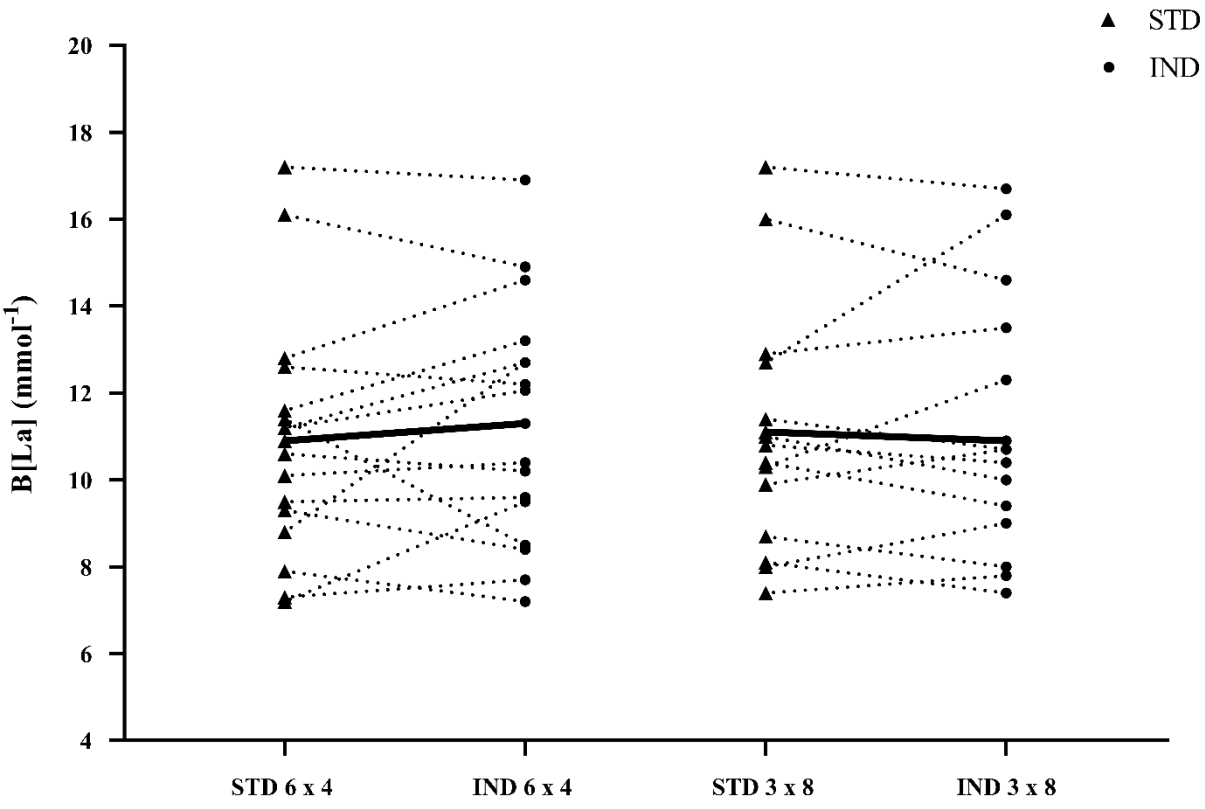


Figure 5.5 – Mean B[La] response during the 6 x 4-min and 3 x 8-min HIIT sessions (Solid line = Mean).

There was a significant interaction between recovery duration and work interval during the 6 x 4-min HIIT protocols (main effect $F = 4.409$, $P = 0.001$; *Figure 5.6a*), demonstrating that the pattern of change in B[La] across work intervals was different between recovery durations. There was no interaction between recovery duration and work interval during the 3 x 8-min HIIT protocols (main effect $F = 1.446$, $P = 0.254$; *Figure 5.6b*), demonstrating that the pattern of change in B[La] across work intervals was not different between recovery durations.

There was an effect of interval during the 6 x 4-min (main effect of interval $F = 22.905$, $P < 0.001$; *Figure 5.6a*) and 3 x 8-min HIIT protocols (main effect of interval $F = 13.137$, $P < 0.001$; *Figure 5.6b*), showing that there was a significant change in work interval B[La] response across the HIIT sessions during the STD and IND recovery durations.

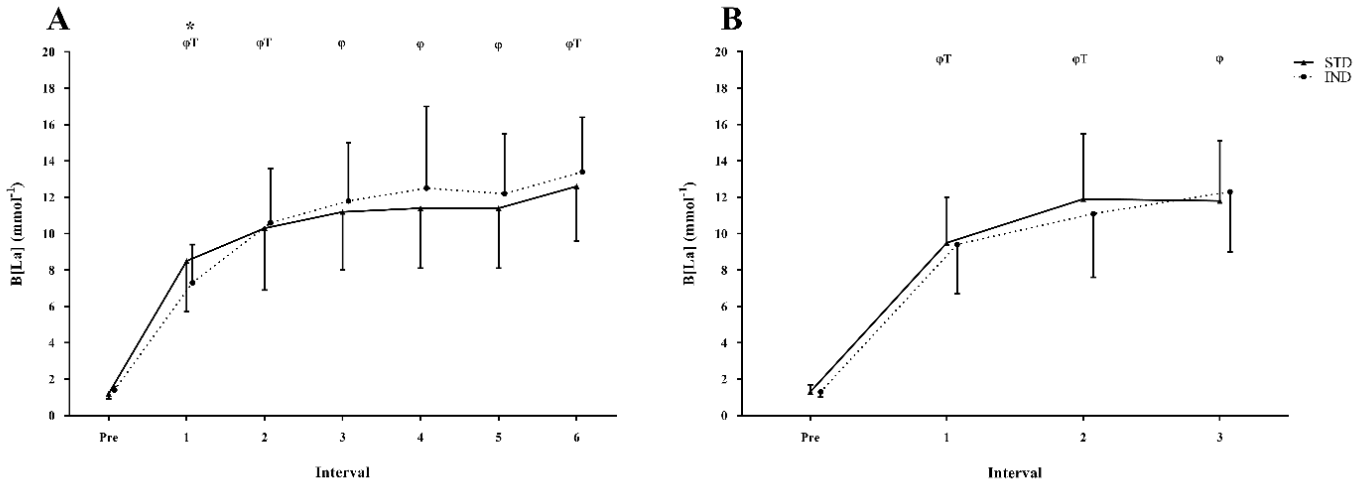


Figure 5.6 – A = Mean work interval B[La] during the 6 x 4-min HIIT sessions, **B** = Mean work interval B[La] during the 3 x 8-min HIIT sessions (Mean ± SD). φ = Significant difference from interval 1, T = Significant difference from previous interval, * = significant difference between recovery conditions (***P* < 0.05**).

V̇O₂ Results

There was a significant interaction between HIIT protocol and recovery duration for mean session $\dot{V}O_2$ (main effect $F = 6.199$, $P = 0.025$). There was no significant difference in mean session $\dot{V}O_2$ between the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of protocol $F < 0.001$, $P = 0.984$). Recovery duration had a significant effect on mean session $\dot{V}O_2$ (main effect of duration $F = 8.829$, $P = 0.01$). *Post hoc* tests revealed mean session $\dot{V}O_2$ was significantly greater during the STD 6 x 4-min HIIT session, when compared to the IND 6 x 4-min HIIT session ($P < 0.05$; *Figure 5.7*).

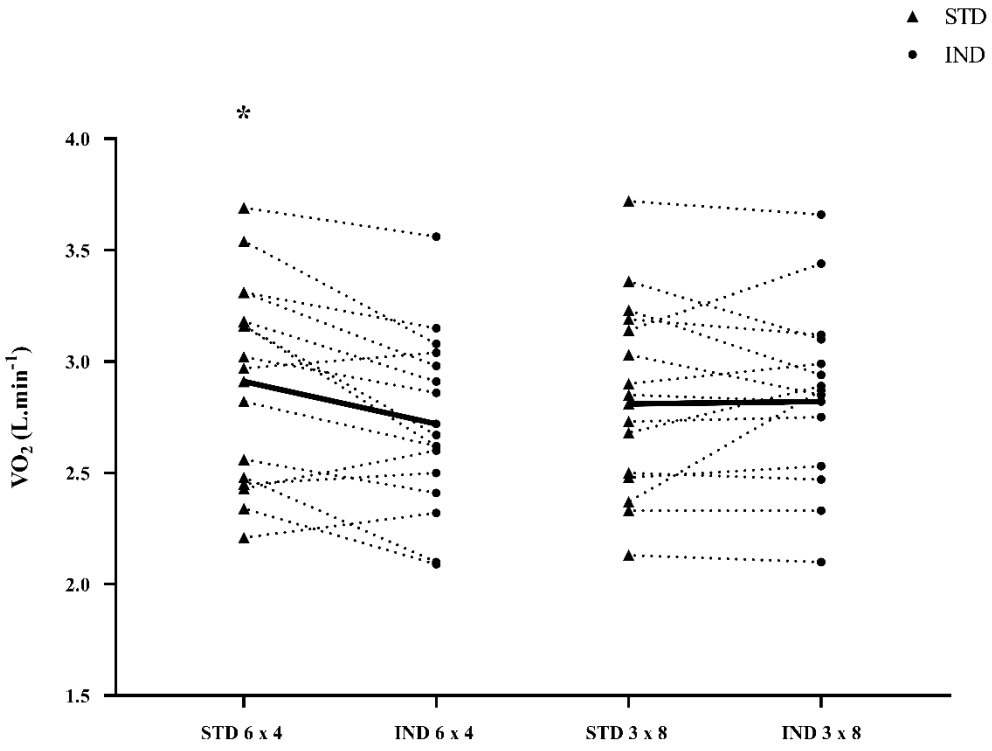


Figure 5.7 - Mean session $\dot{V}O_2$ during the 6 x 4-min and 3 x 8-min HIIT sessions (Solid line = Mean). *** = *P* < 0.05**.

There was no interaction between recovery duration and work interval during the 6 x 4-min (main effect $F = 2.045$, $P = 0.082$; *Figure 5.8a*) and 3 x 8-min HIIT protocols (main effect $F = 0.064$, $P = 0.938$; *Figure 5.8b*), demonstrating that the pattern of change in work interval $\dot{V}O_2$ was not different between the STD and IND recovery durations.

Recovery duration had no effect on work interval $\dot{V}O_2$ during the 6 x 4-min (main effect of duration $F = 1.159$, $P = 0.299$; *Figure 5.8a*) and 3 x 8-min HIIT protocols (main effect of duration $F = 0.754$, $P = 0.399$; *Figure 5.8b*).

There was a significant effect of interval during the 6 x 4-min (main effect of interval F = 12.94, P < 0.001; *Figure 5.8a*) and 3 x 8-min HIIT protocols (main effect of interval F = 17.42, P < 0.001; *Figure 5.8b*), showing that there was a similar upward trend in work interval $\dot{V}O_2$ across the HIIT sessions during the STD and IND recovery durations.

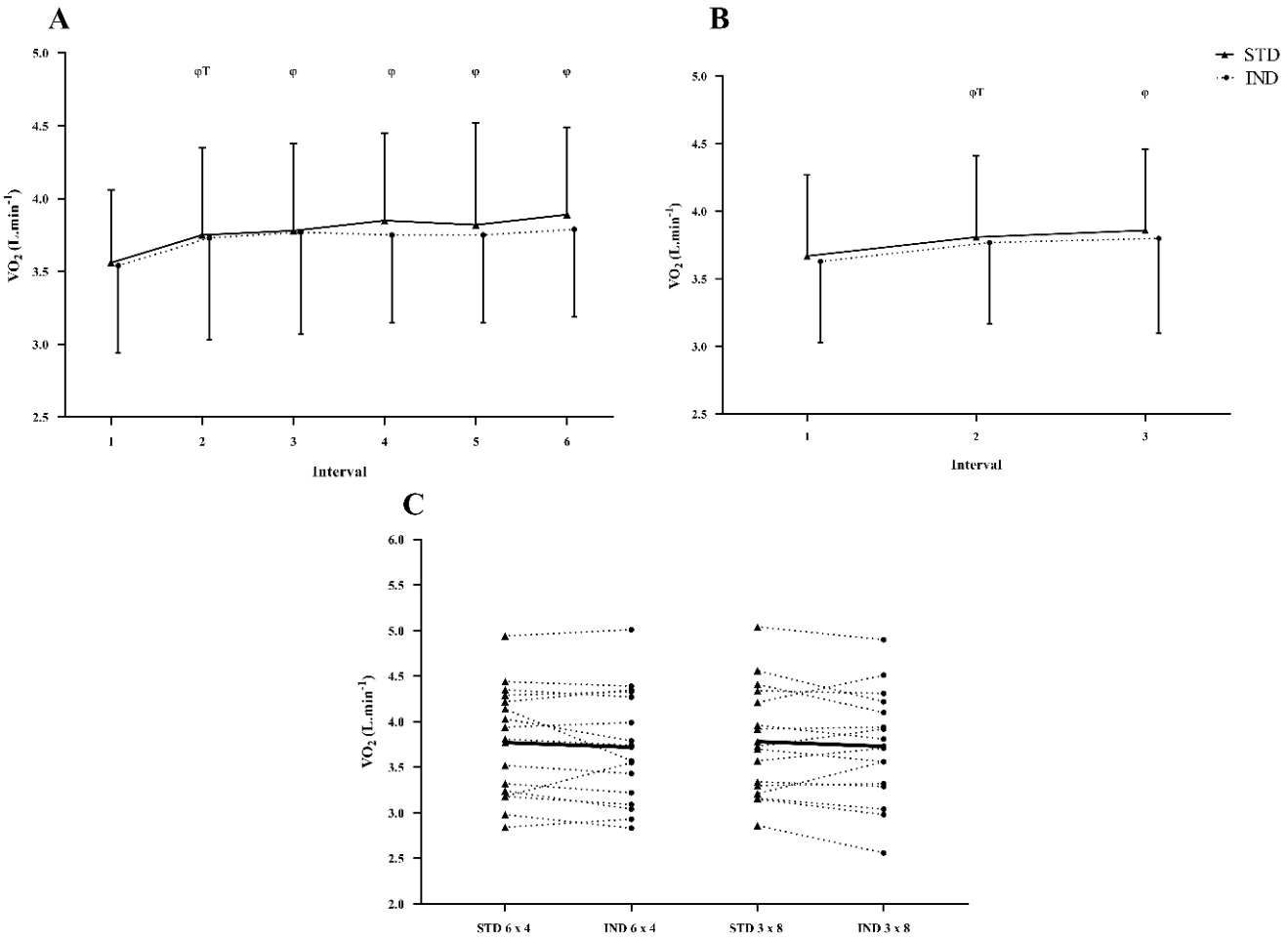


Figure 5.8 – A = Work interval $\dot{V}O_2$ during the 6 x 4-min HIIT sessions, **B** = Work interval $\dot{V}O_2$ during the 3 x 8-min HIIT sessions (Mean ± SD), **C** = Mean work interval $\dot{V}O_2$ during the 6 x 4-min and 3 x 8-min HIIT sessions (Solid line = Mean). φ = Significant difference from interval 1, T = Significant difference from previous interval.

There was no interaction between HIIT protocol and recovery duration for mean work interval $\dot{V}O_2$ (main effect F = 0.017, P = 0.898). There was no significant difference in mean work interval $\dot{V}O_2$ between the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of protocol F = 0.058, P = 0.813). Recovery duration had no effect on work interval $\dot{V}O_2$ during the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of duration F = 2.225, P = 0.156; *Figure 5.8c*).

V.III.2 – Key perceptual HIIT session results

There was no interaction between HIIT protocol and recovery duration for RPE (main effect $F = 0.72$, $P = 0.409$; *Figure 5.9a*) and sRPE (main effect $F = 0.408$, $P = 0.533$; *Figure 5.9b*). There was no significant difference in RPE (main effect of protocol $F = 2.456$, $P = 0.138$; *Figure 5.9a*) and sRPE (main effect of protocol $F = 0.01$, $P = 0.92$; *Figure 5.9b*) between the 6 x 4-min and 3 x 8-min HIIT protocols. Recovery duration had no effect on the participants perceptual response to the HIIT sessions with no significant differences in reported RPE (main effect of duration $F = 1.959$, $P = 0.182$; *Figure 5.9a*) and sRPE (main effect of duration $F = 1.36$, $P = 0.262$; *Figure 5.9b*).

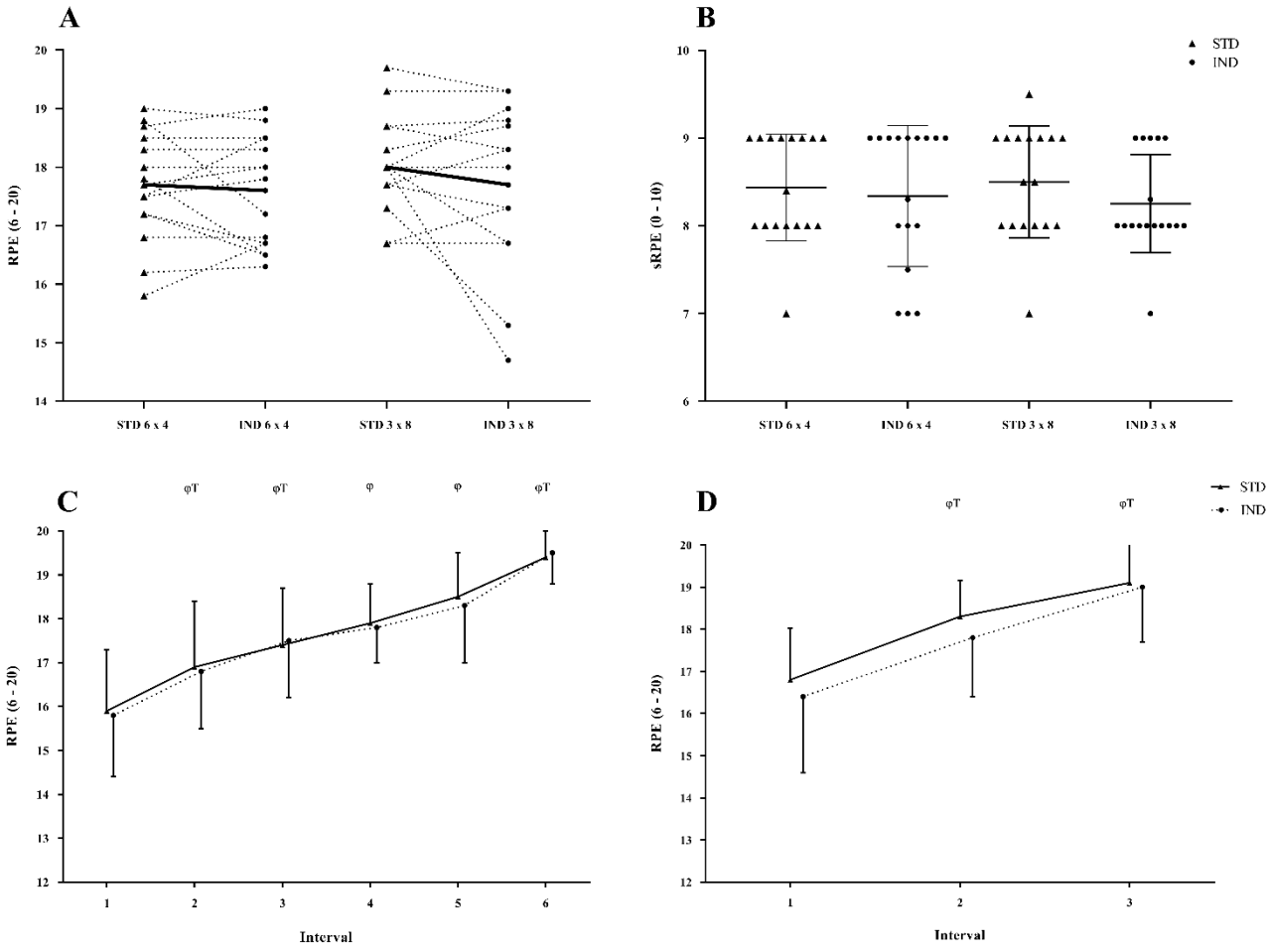


Figure 5.9 – A = Mean RPE during the 6 x 4-min and 3 x 8-min HIIT sessions (Solid line = Mean), **B** = sRPE during the 6 x 4-min and 3 x 8-min HIIT sessions (Mean ± SD), **C** = Work interval RPE during the 6 x 4-min HIIT sessions, **D** = Work interval RPE during the 3 x 8-min HIIT sessions (Mean ± SD). φ = Significant difference from interval 1, T = Significant difference from previous interval.

There was no interaction between recovery duration and work interval during the 6 x 4-min (main effect $F = 0.579$, $P = 0.716$; *Figure 5.9c*) and 3 x 8-min HIIT protocol (main effect $F = 1.255$, $P = 0.3$; *Figure 5.9d*), demonstrating that the pattern of change in work interval RPE was not different between the STD and IND recovery durations.

Recovery duration had no effect on work interval RPE during the 6 x 4-min (main effect of duration $F = 0.231$, $P = 0.638$; *Figure 5.9c*) and 3 x 8-min HIIT protocol (main effect of duration $F = 1.613$, $P = 0.223$; *Figure 5.9d*).

There was a significant effect of interval during the 6 x 4-min (main effect of interval $F = 55.222$, $P < 0.001$; *Figure 5.9c*) and 3 x 8-min HIIT protocol (main effect of interval $F = 50.853$, $P < 0.001$; *Figure 5.9d*), showing that there was a significant increase in work interval RPE across the HIIT sessions during the STD and IND recovery durations.

V.III.3 – Key recovery interval results

Heart Rate Results

There was no interaction between recovery duration and recovery interval during the 6 x 4-min (main effect $F = 1.472$, $P = 0.224$; *Figure 5.10a*) and 3 x 8-min HIIT protocols (main effect $F = 3.257$, $P = 0.093$; *Figure 5.10b*), demonstrating the pattern of change in HR across the recovery intervals was not different between the STD and IND recovery durations.

Recovery interval HR was significantly higher during the STD 6 x 4-min HIIT session, when compared to the IND 6 x 4-min HIIT session (main effect of duration $F = 11.584$, $P = 0.005$; *Figure 5.10a*). Recovery duration had no effect on recovery interval HR during the 3 x 8-min HIIT protocols (main effect of duration $F = 1.192$, $P = 0.293$; *Figure 5.10b*).

There was a significant effect of interval during the 6 x 4-min (main effect of interval $F = 41.814$, $P < 0.001$; *Figure 5.10a*) and 3 x 8-min HIIT protocol (main effect of interval $F = 12.381$, $P = 0.003$; *Figure 5.10b*), showing that there was a similar upward trend in recovery interval HR across the HIIT sessions during the STD and IND recovery durations.

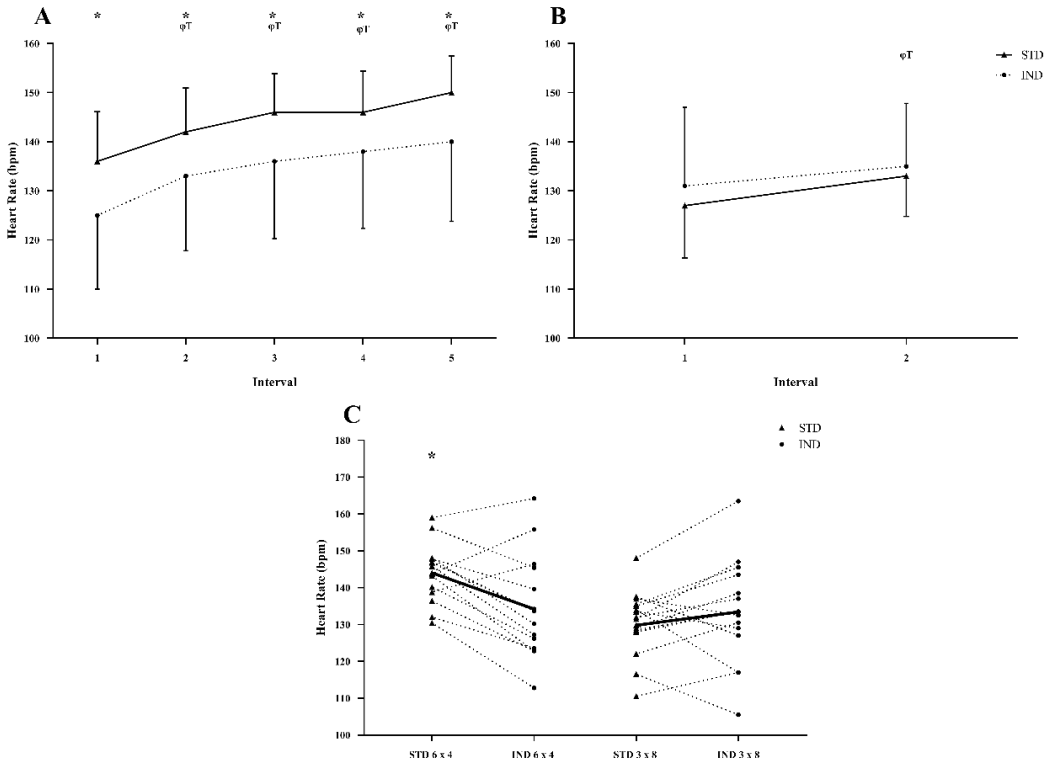


Figure 5.10 – A = Recovery interval HR during the 6 x 4-min HIIT sessions, **B** = Recovery interval HR during the 3 x 8-min HIIT sessions (Mean ± SD), **C** = Mean recovery interval HR during the 6 x 4-min and 3 x 8-min HIIT sessions (Solid line = Mean). φ = Significant difference from interval 1, T = Significant difference from previous interval, *** = *P* < 0.05**.

There was a significant interaction between HIIT protocol and recovery duration for mean recovery interval HR (main effect $F = 14.395$, $P = 0.003$; *Figure 5.10c*). Mean recovery interval HR was significantly higher during the 6 x 4-min HIIT protocols when compared to the 3 x 8-min HIIT protocols (main effect of protocol $F = 9.533$, $P = 0.009$; *Figure 5.10c*). Recovery duration had no effect on mean recovery interval HR (main effect of duration $F = 3.572$, $P = 0.003$). *Post hoc* tests revealed a significantly higher mean recovery interval HR during the STD 6 x 4-min HIIT session, when compared to the IND 6 x 4-min HIIT session ($P < 0.05$; *Figure 5.10c*).

V̇O₂ Results

There was no interaction between recovery duration and recovery interval during the 6 x 4-min (main effect $F = 1.125$, $P = 0.353$; *Figure 5.11a*) and 3 x 8-min HIIT protocol (main effect $F = 0.339$, $P = 0.569$; *Figure 5.11b*), demonstrating that the pattern of change in recovery interval $\dot{V}O_2$ across the recovery intervals was not different between the STD and IND recovery durations.

Recovery interval $\dot{V}O_2$ was significantly higher during the STD 6 x 4-min HIIT session, when compared to the IND 6 x 4-min HIIT session (main effect of duration $F = 14.474$, $P = 0.002$; *Figure 5.11a*). Recovery duration had no effect on recovery interval $\dot{V}O_2$ during the 3 x 8-min HIIT protocols (main effect of duration $F = 2.058$, $P = 0.172$; *Figure 5.11b*).

There was a significant effect of recovery interval during the 6 x 4-min (main effect of interval $F = 15.345$, $P < 0.001$; *Figure 5.11a*) and 3 x 8-min HIIT protocol (main effect of interval $F = 5.31$, $P = 0.036$; *Figure 5.11b*), showing that there was a similar upward trend in recovery interval $\dot{V}O_2$ across the HIIT sessions in both STD and IND recovery durations.

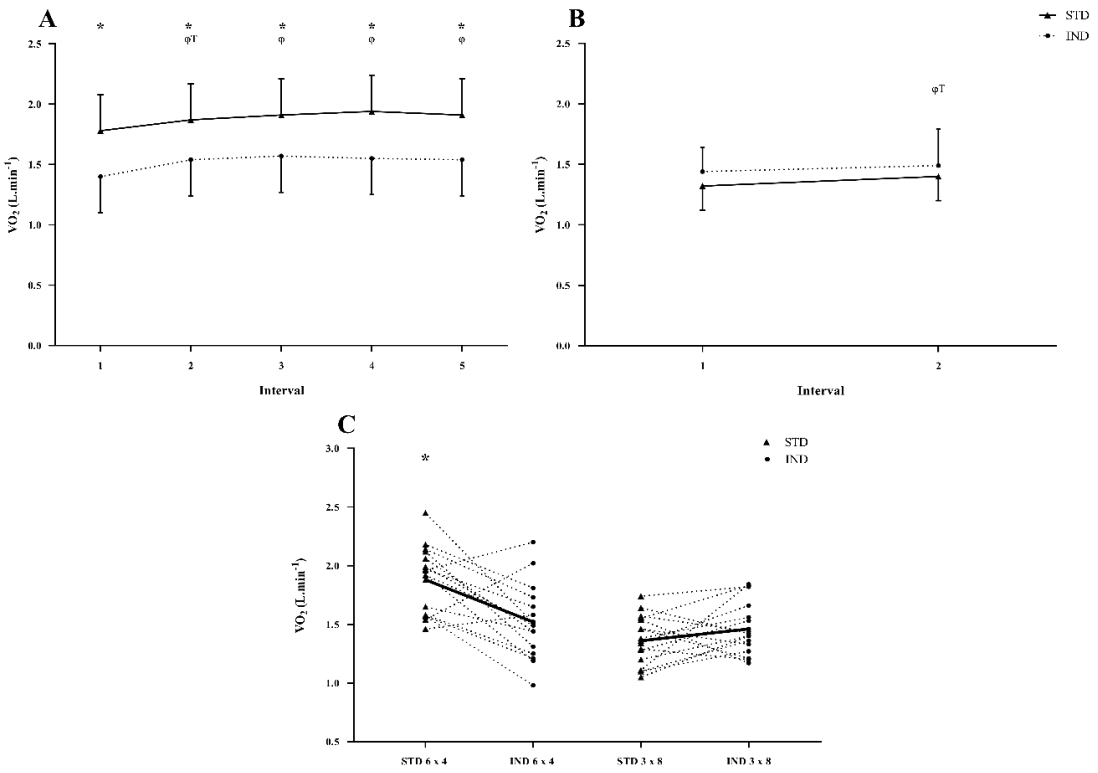


Figure 5.11 – A = Recovery interval $\dot{V}O_2$ during the 6 x 4-min HIIT sessions, **B** = Recovery interval $\dot{V}O_2$ during the 3 x 8-min HIIT sessions (Mean ± SD), **C** = Mean recovery interval $\dot{V}O_2$ during the 6 x 4-min and 3 x 8-min HIIT sessions (Solid line = Mean). φ = Significant difference from interval 1, T = Significant difference from previous interval, *** = *P* < 0.05**.

There was a significant interaction between HIIT protocol and recovery duration for mean recovery interval $\dot{V}O_2$ (main effect $F = 16.624$, $P = 0.001$). Mean recovery interval $\dot{V}O_2$ was significantly higher during the 6 x 4-min HIIT protocols, when compared to the 3 x 8-min HIIT protocols (main effect of protocol $F = 39.174$, $P < 0.001$). There was no main effect of recovery duration on mean recovery interval $\dot{V}O_2$ (main effect of duration $F = 4.443$, $P = 0.052$). *Post hoc* tests revealed a significantly higher mean recovery interval $\dot{V}O_2$ during the STD 6 x 4-min HIIT session, when compared to the IND 6 x 4-min HIIT session ($P < 0.05$; *Figure 5.11c*)

V.III.4 – Time at % of $\dot{V}O_{2max}$, % of MMP and % of HRmax results

Recovery duration had no effect on the percentage of the work intervals spent above 90 and 95% of $\dot{V}O_{2max,}$ for both the 6 x 4-min and 3 x 8-min HIIT sessions ($P > 0.05$). The 6 x 4-min HIIT sessions resulted in a significantly greater percentage of the work intervals spent above 95% of $\dot{V}O_{2max}$ when compared to the 3 x 8-min HIIT sessions ($P = 0.018$; *Table 5.2*).

The STD recovery duration resulted in a significantly greater percentage of the total HIIT session spent above 90% of $\dot{V}O_{2max}$ when compared to the IND recovery duration (main effect $P = 0.027$). *Post hoc* tests revealed that the participants spent a significantly greater percentage of the STD 6 x 4-min HIIT session above 90% of $\dot{V}O_{2max}$ when compared to the IND 6 x 4-min HIIT session ($P < 0.05$). In contrast, there were no differences found in the percentage of the 3 x 8-min HIIT sessions spent above 90% of $\dot{V}O_{2max}$ between the STD and IND recovery durations ($P > 0.05$). Recovery duration had no effect on the percentage of the total session spent above 95% of $\dot{V}O_{2max,}$ for both the 6 x 4-min and 3 x 8-min HIIT sessions ($P = 0.074$). There was no significant difference in the percentage of the total session spent above 95% of $\dot{V}O_{2max}$, between the 6 x 4-min and 3 x 8-min HIIT protocols ($P > 0.05$; *Table 5.2*).

Recovery duration had no effect on the percentage of the work intervals and total session spent above 90 and 95% of MMP, for both the 6 x 4-min and 3 x 8-min HIIT sessions ($P > 0.05$). The 6 x 4-min HIIT sessions resulted in a significantly greater percentage of the work intervals and total session spent above 90 and 95% of MMP, when compared to the 3 x 8-min HIIT sessions ($P < 0.05$; *Table 5.2*).

Recovery duration had no effect on the percentage of the work intervals spent above 90 and 95% of HRmax, for both the 6 x 4-min and 3 x 8-min HIIT sessions ($P > 0.05$). There were no significant differences between the 6 x 4-min and 3 x 8-min HIIT sessions in the percentage of the work intervals spent above 90 and 95% of HRmax ($P > 0.05$; *Table 5.2*).

The 3 x 8-min HIIT sessions resulted in a significantly greater percentage of the total session spent above 90% and 95% of HRmax, when compared to the 6 x 4-min HIIT session ($P < 0.05$). The STD recovery duration resulted in a significantly greater percentage of the total session spent above 90%, but not 95% of HRmax, when compared to the IND recovery duration ($P < 0.05$; *Table 5.2*).

Complete tables of all percentage results including time participants spent at 60, 70, 80, 90 and 95% of MMP, HRmax, $\dot{V}O_{2max,}$ $\dot{V}_{Emax}$ and Bfmax during the HIIT sessions can be found in IX. Appendix, section IX.I – Additional results from study one.

Table 5.2 – Percentage of the work intervals and HIIT sessions spent above 90 and 95% of $\dot{V}O_{2max}$, MMP and HRmax.

		Time at % $\dot{V}O_{2max}$		Time at % MMP		Time at % HRmax	
		90	**95**	**90**	**95**	**90**	**95**
% of work intervals	**STD. 6 x 4**	57.1 ± 21.6 (37.8)	34.8 ± 23.1 (66.2)	6.7 ± 5.2 (77.6)	3.3 ± 3 (91.2)	60.4 ± 19.4 (32.2)	32.6 ± 18.8 (57.7)
	IND. 6 x 4	52 ± 25.3 (48.6)	31.3 ± 27.1 (86.5)	8.7 ± 7.4 (85.2)	2.8 ± 2.6 (91.5)	58.4 ± 19.6 (33.6)	27.9 ± 19.6 (70.1)
	STD. 3 x 8	52.4 ± 27.5 (52.6)	27.6 ± 22.9 (83)	3.3 ± 2.4 (74.9)	1.7 ± 1.8 (108.1)	65.5 ± 22.6 (34.5)	38.2 ± 20.9 (54.6)*****
	IND. 3 x 8	45.4 ± 24.4 (53.8)	20 ± 19 (95)	3.5 ± 1.6 (45.4)	1.6 ± 1.3 (78.8)	60.6 ± 23 (37.9)	29.5 ± 18.5 (62.8)
% of HIIT Session	**STD. 6 x 4**	41.3 ± 15.9 (38.6)*****	25.3 ± 17.2 (68)	4.7 ± 3.6 (77.3)	2.4 ± 2.3 (94.1)	48.4 ± 15.5 (32.1)*****	25.7 ± 14.7 (57.2)
	IND. 6 x 4	30.8 ± 14 (45.5)	17.7 ± 14.9 (84.2)	4.9 ± 3.6 (73.9)	1.6 ± 1.4 (84.3)	40.2 ± 14.7 (36.6)	19 ± 13.6 (71.6)
	STD. 3 x 8	40.1 ± 20.9 (52.2)	21.2 ± 17.4 (82.1)	2.4 ± 1.7 (72.6)	1.2 ± 1.3 (103.3)	52.2 ± 17.8 (34.1)	30.3 ± 16.3 (53.8)
	IND. 3 x 8	35.3 ± 18.4 (52)	14.8 ± 13.2 (89.2)	2.7 ± 1.3 (47.3)	1.3 ± 1 (78.6)	50.2 ± 19.2 (38.2)	25 ± 16.8 (67.2)

Values are Means ± SD, * = $P < 0.05$ significant difference between recovery conditions.

(To normalise the data due to differences in HIIT session duration, resulting from differences between IND and STD recovery durations, the data were statistically analysed and presented as percentages).

V.III.5 – Key NIRS results

The following figures present NIRS results: % HHb, % O_2Hb and TSI %, during the work and recovery intervals of the 6 x 4-min and 3 x 8-min HIIT sessions.

There was a significant interaction between HIIT protocol and recovery duration for % HHb at the end of the work interval (main effect $F = 7.435$, $P = 0.016$; *Figure 5.12a*). There was no difference between the 6 x 4-min and 3 x 8-min HIIT protocols in % HHb at the end of the work intervals (main effect of protocol $F = 2.493$, $P = 0.137$; *Figure 5.12a*). There was no main effect of recovery duration on % HHb at the end of the work intervals (main effect of duration $F = 3.105$, $P = 0.100$; *Figure 5.12a*). *Post hoc* tests revealed that % HHb was significantly higher at the end of the work intervals of the STD 6 x 4-min HIIT session, when compared to the IND 6 x 4-min HIIT session ($P < 0.05$; *Figure 5.12a*).

There was a significant interaction between HIIT protocol and recovery duration for % HHb at the end of the recovery interval (main effect $F = 14.063$, $P = 0.002$; *Figure 5.12b*). There was no difference between the 6 x 4-min and 3 x 8-min HIIT protocols in % HHb at the end of the recovery intervals (main effect of protocol $F = 0.844$, $P = 0.374$; *Figure 5.12b*). There was no main effect of recovery duration on % HHb at the end of the recovery intervals (main effect of duration $F = 3.905$, $P = 0.068$; *Figure 5.12b*). *Post hoc* tests revealed that % HHb was significantly higher at the end of the recovery intervals of the IND 6 x 4-min HIIT session, when compared to the STD 6 x 4-min HIIT session ($P < 0.05$; *Figure 5.12b*).

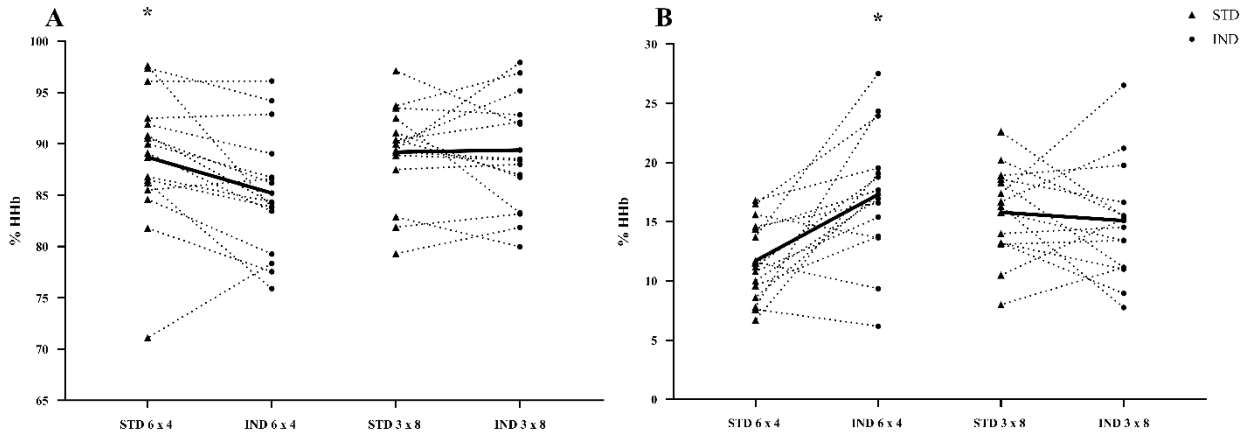


Figure 5.12 – A = Mean % HHb at the end of the work intervals during the 6 x 4-min and 3 x 8-min HIIT sessions, **B** = Mean % HHb at the end of the recovery intervals during the 6 x 4-min and 3 x 8-min HIIT sessions (Solid line = Mean). *** = *P* < 0.05.**

There was a significant interaction between HIIT protocol and recovery duration for % O_2Hb change during the recovery intervals (main effect F = 14.444, P = 0.002; *Figures 5.13a & 5.13b*). There was a significant interaction between recovery duration and recovery interval during the 6 x 4-min HIIT protocols (main effect F = 3.094, P = 0.022; *Figure 5.13a*), but not during the 3 x 8-min HIIT protocols (main effect F = 0.125, P = 0.729; *Figure 5.13b*).

There was no significant difference in % O_2Hb change between the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of protocol F = 0.439, P = 0.519; *Figures 5.13a & 5.13b*).

There was a significant effect of recovery duration during the 6 x 4-min HIIT sessions (main effect of duration F = 18.448, P = 0.001; *Figure 5.13a*), with % O_2Hb recovering to a greater extent during the STD recovery duration when compared to the IND recovery duration. Recovery duration had no effect on the % O_2Hb change during the 3 x 8-min HIIT protocols (main effect of duration F = 0.008, P = 0.928; *Figure 5.13b*).

There was no effect of interval during the 6 x 4-min (main effect of interval F = 1.427, P = 0.236; *Figure 5.13a*) and 3 x 8-min HIIT protocols (main effect of interval F = 3.819, P = 0.071; *Figure 5.13b*), showing that there was no difference in the magnitude of change in % O_2Hb between the recovery intervals, throughout each HIIT session.

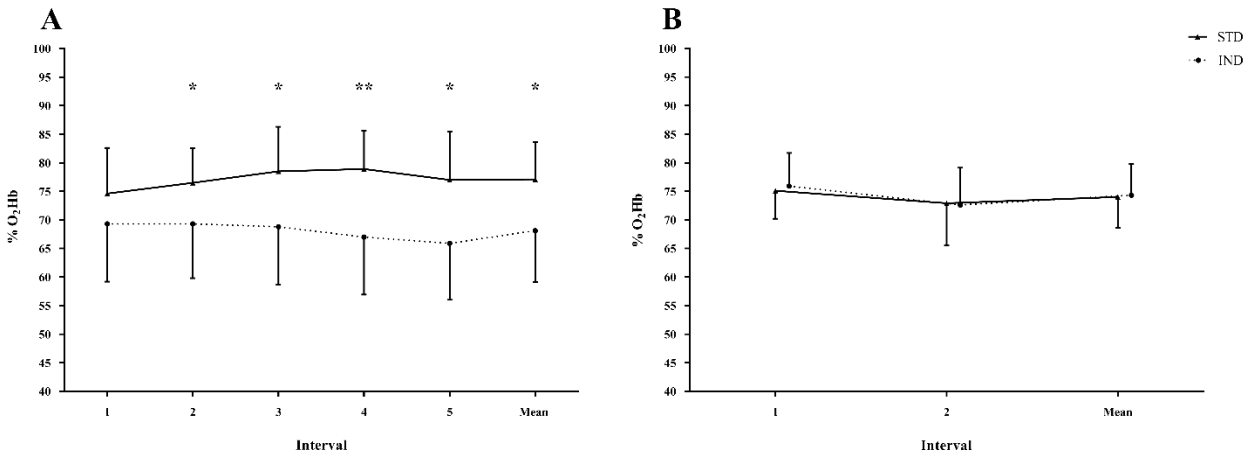


Figure 13 – A = % O_2Hb change during the recovery intervals throughout the 6 x 4-min HIIT sessions, **B** = % O_2Hb change during the recovery intervals throughout the 3 x 8-min HIIT sessions (Mean ± SD). ***** = P < 0.05, ****** = P < 0.001**.

There was no interaction between HIIT protocol and recovery duration for mean TSI % at the end of the work intervals (main effect F = 1.038, P = 0.326; *Figure 5.14a*). There was no significant difference in TSI % at the end of the work intervals between the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of protocol F = 0.173, P = 0.684; *Figure 5.14a*). Recovery duration had no effect on TSI % at the end of the work intervals (main effect of duration F = 1.975, P = 0.182; *Figure 5.14a*).

There was no interaction between HIIT protocol and recovery duration for mean TSI % at the end of the recovery intervals (main effect F = 0.157, P = 0.698; *Figure 5.14b*). There was no significant difference in TSI % at the end of the recovery intervals between the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of protocol F = 2.169, P = 0.163; *Figure 5.14b*). Recovery duration had no effect on TSI % at the end of the recovery intervals (main effect of duration F = 0.594, P = 0.454; *Figure 5.14b*).

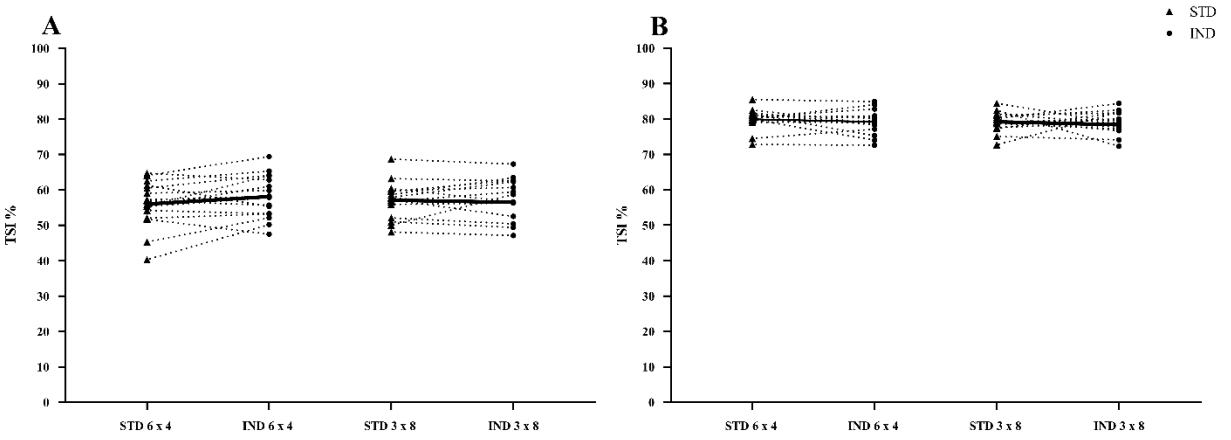


Figure 5.14 – A = Mean TSI % at the end of the work intervals during the 6 x 4-min and 3 x 8-min HIIT sessions, **B** = Mean TSI % at the end of the recovery intervals during the 6 x 4-min and 3 x 8-min HIIT sessions (Mean ± SD).

V.IV – Discussion

V.IV.1 – Key study findings

The main purpose of this study was to investigate whether individualising the duration of the recovery interval based on the recovery of $m\dot{V}O_2$ (i.e. IND recovery duration), would increase the performance of the work intervals and the acute physiological response to a HIIT session, compared to a STD recovery duration (2:1 work recovery ratio). Previous research has used work to recovery ratios (i.e. 2:1 and 1:1) to identify the effect of the recovery interval duration on the acute physiological responses of HIIT sessions (Seiler & Hetlelid, 2005; Schoenmakers & Reed, 2018; Smilios et al., 2017; Laurent et al., 2013; Edge et al., 2013). However, this has been the first study to measure and then apply the participants $m\dot{V}O_2$ recovery duration to HIIT sessions. The IND 4-min recovery duration (205-s) was significantly longer than the STD 4-min recovery duration (120-s). However, there were no significant differences found between the STD (240-s) and IND (200-s) 8-min recovery durations (*Table 4.1*).

The key finding of this study was that the IND recovery duration, did not improve the performance of the work intervals or the acute physiological response to the HIIT session, when compared to the STD recovery duration in well-trained cyclists (see IV.III.1 – Key physiological HIIT results). Specifically, mean POs were not significantly different between the STD and IND recovery conditions, for both the 6 x 4-min and 3 x 8-min HIIT sessions (*Figure 5.1*). These results demonstrate that increasing the recovery interval duration beyond 120-s (or 2:1 work recovery ratio) during a 6 x 4-min HIIT session, provides no additional performance benefit to the subsequent work intervals (*Figure 5.1*). The current studies results

corroborate those of Seiler & Hetlelid (2005), Smilios et al., (2017) and Schoenmakers & Reed, (2018), who found that increasing the recovery interval duration beyond 120-s, during a 6 x 4-min HIIT session did not induce any additional increase in achieved work intensity.

During the 3 x 8-min HIIT session, it was found that decreasing the recovery interval duration below the 2:1 work recovery ratio (or 240-s), did not affect the performance of the subsequent work intervals (*Figure 5.1*). These results suggest that the recovery interval duration maybe determined by the duration of the work intervals within the HIIT session, providing further evidence to support the assumption that the optimal recovery duration is dependent on HIIT protocol design. Based on the current study's findings it appears that the optimal recovery duration may decrease relative to the work interval duration (i.e. longer intervals may require less than a 2:1 work recovery ratio).

In the current study the STD and IND recovery interval durations of both the 6 x 4-min and 3 x 8-min HIIT sessions were ≥ 120-s, providing enough time for key intracellular recovery processes to occur. Such as the rapid component of PCr recovery which occurs within 90-s of exercise cessation (Harris et al., 1976; Taylor et al., 1983), and changes in potassium concentration which are resolved within 60-s (Lindinger, 1995; Medbo & Sejersted, 1990). Intracellular recovery of specific ions (i.e. Pi and H_2PO_4) which are linked to muscle contractile function (Boska et al., 1990; Degroot et al., 1993) also follow a rapid time course of recovery, taking 1 to 2-min to re-establish low intracellular concentrations. The aforementioned recovery processes have been identified as a potential explanation for why the extension of the recovery interval beyond 120-s has no further benefit to work interval performance and acute physiological responses to HIIT (Seiler & Hetlelid, 2005). However,

research has shown the recovery kinetics of $m\dot{V}O_2$ to be well-correlated with the recovery kinetics of PCr (Ryan et al., 2013). Findings of the current study show that $m\dot{V}O_2$ takes ≥ 120-s to recover at the cessation of maximal high intensity cycling exercise (*Table 4.1*). Consequently, it can be assumed that a full recovery of PCr would not have been achieved within 120-s. Current evidence would therefore indicate that full metabolic homeostasis of the exercising muscle is not achieved within 120-s.

Based on the $m\dot{V}O_2$ findings of the current study (*Table 4.1*), it is clear that the exercising muscle would not have recovered to the same extent during the 120-s recovery of the STD 6 x 4-min HIIT session, when compared to the 205-s recovery of the IND 6 x 4-min HIIT session. Despite the incomplete recovery provided during the STD 6 x 4-min HIIT session the performance of the work intervals were not affected (*Figure 5.1*). This suggests that full metabolic recovery of the exercising muscle may not be required to maintain work interval performance during HIIT. However, the same inference cannot be made for the 3 x 8-min HIIT sessions as the STD recovery duration (240-s) was longer than the IND recovery duration (200-s). Therefore, it would be assumed full metabolic recovery was attained in both cases, hence the similar work interval performance (*Figure 5.1*).

The current study adds to the current body of literature on the effect of recovery interval duration on HIIT, providing evidence that a full recovery of $m\dot{V}O_2$ at the exercising muscle may not be required to maintain work interval performance and to generate the desired acute physiological responses (Seiler & Hetlelid, 2005; Smilios et al., 2017; Schoenmakers & Reed, 2018; Laurent et al., 2014). Therefore, based on current findings the optimal recovery interval duration would likely be the shortest time necessary to allow the individual to

maintain work interval performance, thereby maximising the time efficiency of the HIIT session. However, as the findings from the current study show, the full recovery time of $m\dot{V}O_2$ at the exercising muscle does not provide the optimal recovery interval duration. Unfortunately, the findings of the current study do not allow for the elucidation of the shortest (i.e. optimal) recovery duration required for the maintenance of work interval performance. Future research should therefore seek to establish the point at which further decreasing recovery interval duration negatively effects work interval performance and/or results in the failure to complete the intervals or HIIT session. Through establishing the shortest recovery duration required for a given HIIT protocol, research would be a step closer to fully maximising the time efficiency of HIIT.

V.IV.2 – Acute physiological responses to the HIIT sessions

In the current study, the participants physiological responses to the HIIT sessions were measured. As recovery duration had no effect on the mean work interval POs participants were able to produce (*Figures 5.2a & 5.2b*), it is not surprising that there were no significant differences found in mean work interval HR (*Figure 5.4c*), mean B[La] response (*Figure 5.5*) and mean work interval $\dot{V}O_2$ (*Figure 5.8c*), between the STD and IND recovery conditions, for both the 6 x 4-min and 3 x 8-min HIIT sessions.

Recovery duration did affect HR and $\dot{V}O_2$ responses during the recovery intervals (V.III.3 – Key recovery interval results). The significantly longer recovery interval duration (205-s IND vs 120-s STD) of the IND 6 x 4-min HIIT sessions allowed for a greater reduction in HR and $\dot{V}O_2$ during the recovery interval, when compared to the STD 6 x 4-min HIIT session (*Figures 5.10a & 5.11a*). This in turn resulted in a significantly lower mean recovery interval HR and $\dot{V}O_2$ (*Figures 5.10c & 5.11c*) which explains the lower mean session HR and $\dot{V}O_2$ (*Figures 5.3 & 5.7*), when compared to the STD 6 x 4-min HIIT session. Despite the greater decrease in HR and $\dot{V}O_2$ during the recovery intervals of the IND 6 x 4-min HIIT session, there was no effect on the participants ability to attain high percentages of HRmax and $\dot{V}O_{2max}$ in the subsequent work intervals (*Table 5.2*).

There were no significant differences found in the time participants spent at 90 and 95% of HRmax between the STD and IND recovery conditions, during the work intervals of the 6 x 4-min HIIT session (*Table 5.2*). Current HR response findings corroborate those of Schoenmakers & Reed, (2018), who also investigated the effect of recovery duration on self-paced 6 x 4-min HIIT performance using runners. They found that 1-min, 2-min and 4-min

recovery durations had no effect on the time spent exercising above 90 and 95% of HRmax (Schoenmakers & Reed, 2018). However, in the current study the shorter duration of the STD 6 x 4-min HIIT session (2040-s versus 2465 ± 396-s IND session duration) resulted in participants spending a greater percentage of the whole session above 90 and 95% of HRmax when compared to the IND 6 x 4-min HIIT session, albeit only significant at 90% of HRmax (*Table 5.2*).

The STD and IND 3 x 8-min HIIT sessions produced similar HR responses, with no significant differences in mean session HR (*Figure 5.3*), mean work interval HR (*Figure 5.4c*) and mean recovery interval HR (*Figure 5.10c*). There were also no significant differences between the STD and IND recovery conditions in the percentage of the whole 3 x 8-min HIIT session participants spent above 90 and 95% of HRmax (*Table 5.2*). These findings are most likely due to there being no significant difference in the time of the STD (240-s) and IND (200 ± 81-s) recovery durations. Moreover, these findings also support the use of a 2:1 work recovery ratio for longer work intervals (≥ 8-min), adding to previous research which as shown the 2:1 ratio to be sufficient for maintenance of 4-min work interval performance (Seiler & Hetlelid, 2005; Smilios et al., 2017; Laurent et al., 2014).

Seiler & Hetlelid (2005), used a similar 6 x 4-min HIIT protocol (in well trained runners) to the current study and observed an upward drift in recovery interval HR as the session progressed across all three of the recovery interval duration conditions (1-min, 2-min and 4-min). In the current study, the upward drift in mean recovery interval HR was observed during all HIIT sessions (*Figures 5.10a & 5.10b*). Mean work interval HR also exhibited an upward drift across the HIIT sessions (*Figures 5.4a & 5.4b*). However, recovery interval

duration did not attenuate the magnitude of HR drift across work intervals. Although HR did not recover to the same extent throughout the HIIT sessions, this did not significantly affect the PO produced during the work intervals (*Figures 5.2a & 5.2b*).

The time spent at high percentages of $\dot{V}O_{2max}$ (≥ 90 and 95%) is often used to quantify the effectiveness of a HIIT protocol (Buchheit & Laursen, 2013). When exercising close to $\dot{V}O_{2max}$ the O_2 delivery and utilisation systems are maximally stressed, which has been suggested to be an effective stimulus for improving $\dot{V}O_{2max}$ and endurance performance (Buchheit & Laursen, 2013; Midgley et al., 2006; Laursen, 2002). During the current study, participants spent 45 to 57% of the work intervals above 90% of $\dot{V}O_{2max}$ and 20 to 34% of the work intervals above 95% of $\dot{V}O_{2max}$ (*Table 5.2*). These findings are comparable to those of Schoenmakers & Reed, (2018) who reported their participants to be spending around 57% of the exercise time above 90% of $\dot{V}O_{2max}$ and 37% above 95% of $\dot{V}O_{2max,}$ during a HIIT session of similar design to the current study (6 x 4-min). While research on the optimal time spent at high percentages of $\dot{V}O_{2max}$ per session is limited, it is argued that to achieve the optimal stimulus from a HIIT session athletes should be spending several minutes above 90% of $\dot{V}O_{2max}$ per session (Billat, 2001; Laursen & Jenkins, 2002; Midgley et al., 2006). The accumulated exercise time above 90 and 95% of $\dot{V}O_{2max}$ in the current study and that of Schoenmakers & Reed, (2018), fall within the range suggested to the optimal for endurance adaptations (Billat, 2001; Laursen & Jenkins, 2002; Midgley et al., 2006; Buchheit & Laursen, 2013).

In the current study, the IND recovery duration did not lead to an increase in the time participants spent above 90 and 95% of $\dot{V}O_{2max}$ when compared to the STD recovery

duration, for both the 6 x 4-min and 3 x 8-min HIIT sessions (*Table 5.2*). In agreement, Schoenmakers & Reed, (2018) and Smilios et al., (2017) also found recovery interval duration to have no effect on the time participants spent exercising above 90 and 95% of $\dot{V}O_{2max,}$ despite subsequent work intervals starting from a lower $\dot{V}O_2$ after the longer recovery intervals. Furthermore, in line with the HR findings, the percentage of the total session participants spent at > 90 and 95% of $\dot{V}O_{2max,}$ was greater during STD 6 x 4-min HIIT session, when compared to the longer IND 6 x 4-min HIIT session (*Table 5.2*).

Schoenmakers & Reed, (2018) reported that shorter recovery intervals (1-min) resulted in an increased metabolic rate at the start of the next work interval, which lengthened the time needed to reach a $\dot{V}O_2$ plateau. Furthermore, the mean response time of $\dot{V}O_2$ and HR was found to be faster after longer recovery intervals (≥ 3-min) and was accompanied by higher $\dot{V}O_2$ and HR amplitude (Schoenmakers & Reed, 2018; Smilios et al., 2017). This explains why similar times spent at 90 and 95% of $\dot{V}O_{2max}$ and HRmax were found between recovery durations even though the work intervals started from a lower $\dot{V}O_2$ and HR after the longer recovery intervals. These findings suggest that $\dot{V}O_2$ kinetics adjust to regulate the O_2 supply that corresponds to the metabolic requirements of the exercise stimulus (Schoenmakers & Reed, 2018).

The HR and $\dot{V}O_2$ results of the current study do not support the implementation of the IND recovery duration, over the STD 2:1 work recovery ratio. None the less, the current study results and those of Schoenmakers & Reed, (2018) and Smilios et al., (2017) show that shorter recovery intervals (≤ 2-min) allow for a greater accumulation of physiological stress relative to the total time spent training, making for a more time efficient HIIT session.

However, if the time efficiency of the HIIT session is not important, longer recovery intervals can still be implemented in the knowledge that the physiological stress achieved during the work intervals will not be reduced. As demonstrated by the findings of the current study which found similar work interval HR (*Figures 5.4a & 5.4b*), $\dot{V}O_2$ (*Figures 5.8a & 5.8b*) and B[La] (*Figures 5.6a & 5.6b*) values, in addition to similar durations spent above 90 and 95% of HRmax and $\dot{V}O_{2max}$ (*Table 5.2*), regardless of recovery interval duration.

However, there remains a limited understanding of the dose-response relationship between training stress (i.e. time at high percentages of $\dot{V}O_{2max}$) and training induced changes in endurance performance, with large inter-individual responses present (Bouchard & Rankinen, 2001; Vollaard et al., 2009). While it is evident that the time spent at high percentages of $\dot{V}O_{2max}$ can provide valuable insight within a study to the understanding of how to optimise HIIT programming. There are numerous methodological limitations which need to be considered when interpreting and comparing $\dot{V}O_2$ findings from different studies such as differences in: determining $\dot{V}O_{2max}$, the reliability level of analysers, $\dot{V}O_2$ kinetics and intra-day variation in participant $\dot{V}O_{2max}$, all of which make comparisons between studies difficult (Dupont et al., 2003; Midgley et al., 2007; Midgley et al., 2007b).

The recovery interval duration had no effect on the B[La] values achieved during the work intervals (*Figure 5.5*). Similarly, several other studies have also reported recovery duration to have no effect on the B[La] response during HIIT (Seiler & Hetlelid, 2005; Laurent et al., 2014; Edwards et al., 2011). In the current study B[La] samples were taken at the end of the work intervals, the lack of difference in work interval PO between recovery conditions (*Figures 5.2a & 5.2b*) may therefore explain the similar levels of metabolic stress produced

(*Figures 5.6a & 5.6b*). These findings demonstrate that through decreasing the recovery duration, it is possible to increase the metabolic stress produced relative to the time spent training.

The acute physiological responses to the HIIT sessions show that the exercise intensity was not significantly different between recovery conditions. Previous research has found that cellular stress occurs in proportion to exercise intensity (Egan & Zierath, 2013), with strong evidence suggesting higher exercise intensities promote greater metabolic signalling than lower intensities of exercise (McInnis & Gibala, 2017). Furthermore, downstream to the multiple metabolic signals, mitochondrial protein synthesis was found to be greater during exercise performed at a higher intensity relative to work matched exercise at a lower intensity (DiDonato et al., 2014), suggesting a greater rate of mitochondrial biogenesis when a fixed volume of exercise is performed at a higher intensity. Therefore, as the recovery interval duration did not significantly alter the exercise intensity of the work intervals, it can be assumed that the HIIT sessions provided a similar acute stimulus for driving endurance adaptations, with the shorter recovery durations producing a more time efficient stimulus.

NIRS findings

In addition to measuring the central responses to the HIIT sessions, NIRS was used throughout all HIIT sessions to investigate any potential effects of recovery interval duration on the peripheral responses (HHb, O_2Hb & TSI %). There have been very few studies which have used NIRS to assess the peripheral responses to HIIT when investigating the manipulation of recovery interval duration (Christmass et al., 1999; McLean et al., 2016). To

the authors knowledge this is the first study to present NIRS data on the effect on recovery interval duration during cycling based HIIT.

There was no significant difference in % HHb at the end of the work intervals between the STD and IND 3 x 8-min HIIT sessions, suggesting similar levels of muscle de-oxygenation (*Figure 5.12a*). McLean et al., (2016) also found recovery interval duration to have no effect on HHb measured during the work interval, despite a greater decrease in HHb during the longer recovery interval. There was also no significant difference in % HHb at the end of the recovery intervals during the STD and IND 3 x 8-min HIIT sessions (*Figure 5.12b*), indicating similar levels of O_2Hb recovery (*Figure 5.13b*). In comparison, % HHb was significantly higher at the end of the work intervals during the STD 6 x 4-min HIIT session, when compared to the IND 6 x 4-min HIIT session (*Figure 5.12a*). The increased level of muscle de-oxygenation during the work intervals of STD 6 x 4-min HIIT session may have been expected, due to the significantly shorter recovery interval duration not allowing for the fullest recovery of O_2Hb. On the contrary, % HHb was significantly lower at the end of the recovery intervals during the STD 6 x 4-min HIIT session, when compared to the IND 6 x 4-min HIIT session (*Figure 5.12b*), indicating a greater recovery of O_2Hb (*Figure 5.13a*). At present we cannot provide an explanation for this finding, however it is possible that the differences found were simply an artefact of measurement error.

Furthermore, there were no significant differences in the TSI % at the end of the work intervals between recovery conditions, for both the 6 x 4-min and 3 x 8-min HIIT sessions (*Figure 5.14a*). Likewise, there were no significant differences in the TSI % at the end of the recovery intervals between recovery conditions, for both the 6 x 4-min and 3 x 8-min HIIT

sessions (*Figure 5.14b*). The recovery intervals were prescribed as PA intensity, which allowed for a rapid recovery of TSI % immediately after work interval cessation. TSI % during complete PA rest was around 80% with very little variation between participants (CVs between 3.9 and 7.4). In contrast, during exercise there was a greater variation in TSI % between participants with CVs between 8.8 and 13.3, indicating potential effects of exercise intensity.

The above findings notwithstanding, all HIIT sessions increased muscle de-oxygenation in the exercising muscle suggesting a discrepancy in O_2 delivery and utilization (Belfry et al., 2012). These results are in accordance with previous research which has shown increased muscle de-oxygenation during HIIT using longer work intervals (Zafeiridis et al., 2015). Several studies have highlighted local hypoxia as a stimulus for increasing the activity of mitochondrial enzymes, capillary proliferation, and mitochondrial biogenesis (Fluck, 2006; Prior et al., 2004; Terrados et al., 1990). Therefore, the level of muscle de-oxygenation achieved during exercise may dictate the extent of adaptations in the exercising muscle. As the current study shows HIIT provides an effective method for inducing increased levels of muscle de-oxygenation. However, it has been found that different exercise modalities (continuous, short interval [30-s] and long interval [2-min]), when performed under isoeffort conditions result in a similar peripheral hemodynamic response (Zafeiridis et al., 2015).

V.IV.3 – Acute perceptual response to the HIIT sessions

Recovery interval duration had no effect on reported RPE or sRPE values, during both the 6 x 4-min and 3 x 8-min HIIT sessions (*Figures 5.9a & 5.9b*). Throughout all four HIIT sessions there was a linear increase in work interval RPE, with reported values reaching between 18 and 19 (Very hard to Extremely hard) at the last work interval (*Figures 5.9c & 5.9d*). This linear increase in RPE occurred despite mean PO being relatively consistent across the work intervals (*Figures 5.2a & 5.2b*). Similar increases in RPE have been observed in previous HIIT studies involving well trained runners, despite the participants maintaining a relative constant running velocity across the work intervals (Schoenmakers & Reed, 2018; Seiler & Sjursen, 2004; Seiler & Hetlelid, 2005). The high RPE values reported across these studies are not surprising, as participants were asked to self-pace their efforts during the HIIT session on a 'maximal session effort' basis (Schoenmakers & Reed, 2018; Seiler & Sjursen, 2004; Seiler & Hetlelid, 2005). The upward drift in RPE can be attributed to the increasing physiological, biomechanical, and psychological stress the participants experienced as the HIIT sessions progressed (Marcora et al., 2009; Ulmer, 1996).

Seiler & Hetlelid, (2005) and Schoenmakers & Reed, (2018) also reported near maximal RPE values (≥ 17) for the final interval of a 6 x 4-min HIIT session. Contrary, to current findings, Seiler & Hetlelid, (2005) and Smilios et al., (2017), found 2-min recovery intervals led to a significantly higher final interval RPE when compared to a 4-min recovery interval. The reason no such differences in RPE were found in the current study may be due to the recovery intervals being closer in duration.

For all HIIT sessions mean sRPE was > 8 (*Figure 5.9b*), suggesting that most participants perceived the sessions to be near maximal efforts despite the intermittent PA recoveries. In agreement with Seiler & Hetlelid (2005) and based on the RPE values reported, between 24 to 32-min of total work duration would appear to represent the upper limit for long work interval HIIT sessions (≥ 4-min work intervals) before voluntary exhaustion occurs. Extending work duration further would likely reduce the exercise intensity of work intervals, thereby potentially reducing the effectiveness of the session. Interestingly, research has also shown the accumulation of 32-min of work during HIIT (at a "maximal effort" intensity prescription) to induce greater physiological adaptations, when compared to accumulating 64-min of work at a lower intensity or 16-min of work at a higher intensity. Importantly, the 32-min HIIT session resulted in lower peak RPE values, compared to the 64-min and 16-min HIIT sessions (Seiler et al., 2013).

V.IV.4 – Observation of individual participants responses

The group mean results of the current study show that there were no differences in the performance of the work intervals between the IND and STD recovery durations. However, closer inspection of individual data reveals that several of the participants had a higher mean PO during the IND recovery duration prescription, when compared to the STD recovery duration (*Figure 5.15*). 8 of the 16 participants had a higher mean PO during the IND 6 x 4-min HIIT session (341 ± 45W), when compared to the STD 6 x 4-min HIIT session (326 ± 46W; *Figure 15-A*). While 10 of the 16 participants had a higher mean PO during the IND 3 x 8-min HIIT session (299 ± 52W), when compared to the STD 3 x 8-min HIIT session (290 ± 53W; *Figure 15-A*). There were 6 participants who had a higher mean PO in both the 6 x 4-min and 3 x 8-min IND HIIT sessions, when compared to the STD 6 x 4-min and 3 x 8-min HIIT sessions.

The higher mean PO did not necessarily translate to an increased level of physiological stress produced during the work intervals. Only 4 of the 8 participants with a higher mean PO during the IND 6 x 4-min HIIT session, also had a higher mean work interval $\dot{V}O_2$ (*Figure 5.15-B*) and mean work interval HR (*Figure 5.15-C*), when compared to the STD 6 x 4-min HIIT session. Of the 10 participants with a higher mean PO during the IND 3 x 8-min HIIT session, 6 had a higher mean work interval $\dot{V}O_2$ (*Figure 5.15-B*) and 2 had a higher mean work interval HR (*Figure 5.15-C*), when compared to the STD 3 x 8-min HIIT session.

Interestingly, 7 of the 8 participants with a higher mean PO during the IND 6 x 4-min HIIT session had an IND recovery duration longer (274 ± 49-s), than the STD 120-s recovery duration. Whereas, 7 of the 10 participants with a higher mean PO during the IND 3 x 8-min

HIIT session had an IND recovery duration shorter (158 ± 31-s), than the STD 240-s recovery duration. These results indicate that the optimum recovery duration is highly individual, dependent on the HIIT protocol design and participant training status (Schoenmakers et al., 2019). Moreover, current results suggest that the work recovery ratio prescription may not be optimal for everyone but remains the most practical and suitable method for prescribing recovery interval duration across a broad range of individuals.

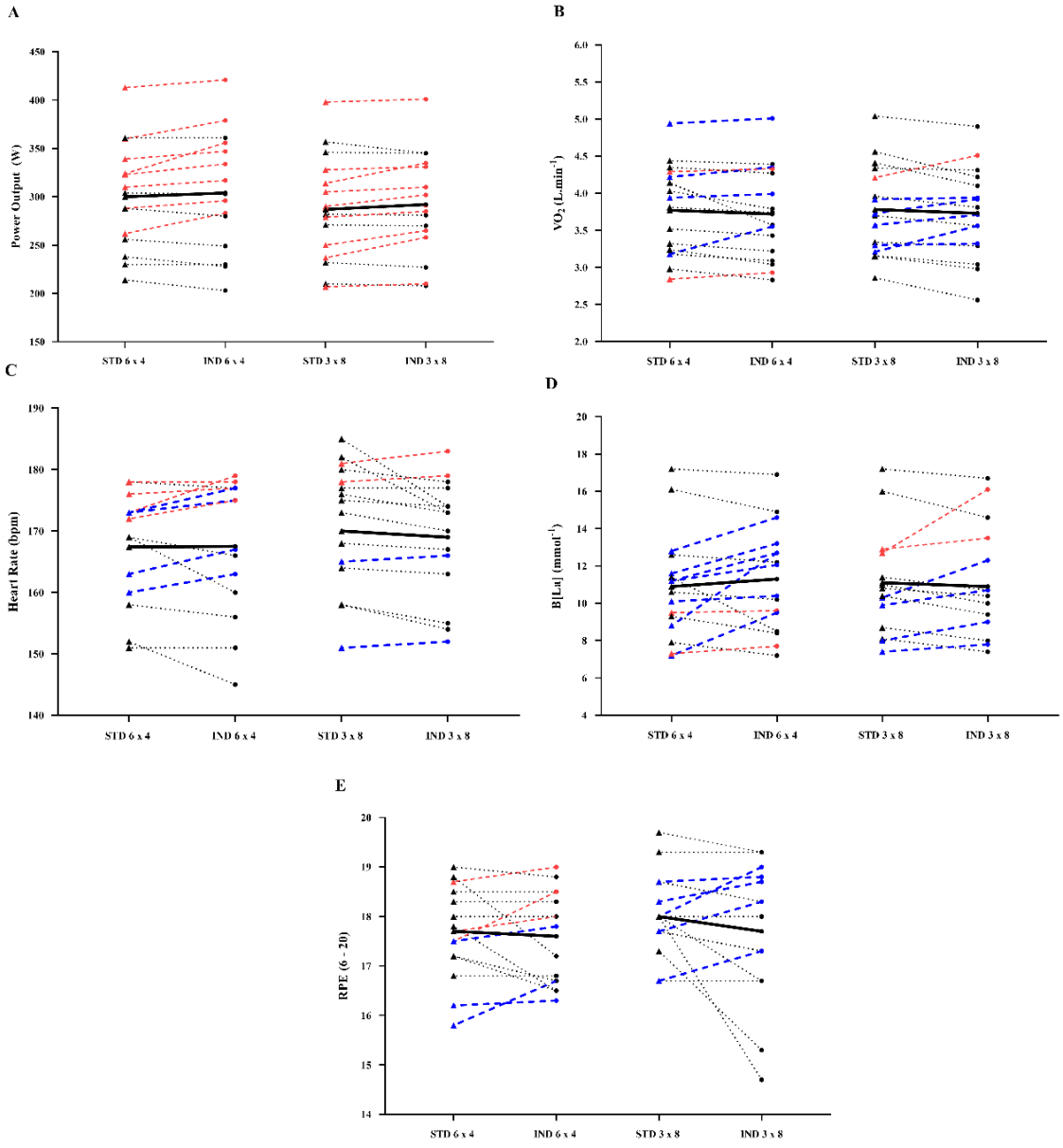


Figure 5.15 – 6 x 4-min and 3 x 8-min HIIT session results: **A)** Mean PO, **B)** Mean work interval $\dot{V}O_2$, **C)** Mean work interval HR, **D)** Mean B[La] response, **E)** Mean RPE. Solid black line = Mean, Red dashed line = participants with higher values in the IND condition, Blue dashed line = participants with higher value in the IND condition and a higher IND mean PO.

V.IV.5 – Study Limitations

An initial delimitation of the study was the measurement of $m\dot{V}O_2$ recovery duration after a single 4-min or 8-min high intensity work interval, which was then applied to a HIIT session of multiple high intensity work intervals. While there is currently no research investigating how $m\dot{V}O_2$ recovery duration changes across repeated work intervals, it can be assumed that $m\dot{V}O_2$ recovery duration will not be the same after repeated work intervals when compared to a single work interval. However, the same limitation can be applied to a standardised recovery duration, as it is likely the optimal recovery duration required between work intervals changes across a HIIT session. Indeed, Chidnok et al., (2013) has shown that a key aspect of skeletal muscle recovery, which is closely linked to time constant for $m\dot{V}O_2$ recovery, the restoration of PCr takes longer as HIIT sessions progress. This highlights the idea that the optimal recovery interval duration is likely to change throughout a HIIT session. However, it must be questioned whether the complexity of individualising recovery intervals to such an extent is necessary for maximising HIIT session outcomes.

In the current study, participants could self-select their work interval exercise intensity, although they were instructed to complete each HIIT session on a 'maximal session effort' basis. This form of exercise prescription relies on the motivation of the participant to truly push themselves 'maximally' during each session. Therein lies the limitation, each participants perception of 'maximal session effort' varies and could therefore result in participants achieving different levels of physiological stress during each session as it does not allow for the precise manipulation of the physiological responses to a given HIIT session (RPE responses reflect the sensation of how strenuous the exercise is, relative to the combined physiological, biomechanical, psychological stress placed on the body during

exercise; Marcora et al., 2009; Ulmer, 1996). Through not controlling work interval intensity the effect of recovery interval duration becomes more difficult to assess. On the other hand, allowing participants to self-pace ensures they complete the session while also removing any limitations of pre-set exercise intensities. This allows the participants to push themselves to what they perceive to be 'maximal' at each session and in doing so should maximise the training stimulus of the prescribed HIIT session. Moreover, RPE is a universal exercise intensity regulator irrespective of exercise mode and is therefore a practical method for coaches to prescribe exercise intensity.

In the current study only two recovery interval durations were compared (STD and IND). Although this was the intention from the conception of the study design, this does limit the studies ability to assess the effect of a broader range of recovery interval durations.

Finally, the main limitation of the study was not matching the exercise intensity of the work intervals during the measurement of m$\dot{V}O_2$ recovery duration and the HIIT sessions. As previous research has shown, m$\dot{V}O_2$ recovery is affected by exercise intensity (Buchheit et al., 2011; Krustrup et al., 2009). The 4-min (330W) and 8-min (297W) interval PO during the measurement of m$\dot{V}O_2$ recovery duration was significantly higher than the mean PO during the experimental 6 x 4-min (302W) and 3 x 8-min HIIT sessions (289W; $P < 0.05$). This raises the question: if exercise intensity had been controlled during the measurement of m$\dot{V}O_2$ recovery duration, with the same exercise intensity then applied to the HIIT sessions, would there have been differences found in the acute physiological responses? Future research looking to further investigate m$\dot{V}O_2$ recovery duration as a way of individualising HIIT recovery interval duration should: control for exercise intensity and assess the reliability

and reproducibility of NIRS measurements of m$\dot{V}O_2$ recovery duration of the VL muscle after a single and multiple bouts of intense cycling exercise.

V.IV.6 – Practical applications

As this study has demonstrated, the 2:1 work recovery ratio appears to sit in a "*sweet spot"* of recovery interval duration. By increasing or decreasing the recovery interval duration within the range of the 2:1 work recovery ratio, we have found there to be no significant effect on the performance of subsequent work intervals and the acute physiological response to the HIIT session (when using PA recoveries). Therefore, when programming HIIT sessions coaches and athletes should consider utilising the 2:1 work recovery ratio. In doing so, they can be reasonably confident they are achieving adequate recovery between work intervals, while maximising the time spent training.

V.IV.7 - Conclusion

In conclusion, individualising HIIT recovery duration based on m$\dot{V}O_2$ recovery duration, does not maximise the performance of the work intervals and the acute physiological response of the HIIT session, when compared to a STD recovery duration (2:1 work recovery ratio). The current study findings demonstrate that a full recovery of m$\dot{V}O_2$ at the exercising muscle may not be required to maintain work interval performance and to generate the desired acute physiological responses. Consequently, the time taken for a full recovery m$\dot{V}O_2$ to occur does not provide the optimal recovery interval duration for the specific individual and HIIT protocol. However, current evidence further highlights the efficacy for continued use of the 2:1 work recovery ratio.

The question of how to individualise the recovery interval duration for specific HIIT sessions remains unanswered. However, it stands to question whether a test to individualise recovery interval duration is required. Moreover, if such a test is devised through future research, its usefulness will remain limited if the equipment needed constrains the test to a physiology laboratory, as was the case with the measurement of m$\dot{V}O_2$ recovery duration.

VI. Experimental Chapter – Study Two

The impact of recovery interval intensity on the acute physiological and perceptual responses to interval training during cycling exercise

VI.I – Introduction

Recovery interval intensity is an important component in HIIT programming and has received a sizeable amount of research attention (*Table 2.2*). The majority of the cycling based research has focused on short HIIT (work intervals ≥ 30-s to < 1-min) and sprint interval training (work intervals 4-s to < 30-s; see *Table 2.2*), likely due to the continued interest in maximising the time efficiency of HIIT for recreationally active individuals (Burgomaster et al., 2008; Gibala et al., 2012). However, there have been comparatively few studies investigating the effects of recovery interval intensity during cycling based HIIT using long work intervals (≥ 1-min; Barbosa et al., 2016; Coso et al., 2010; Dorado et al., 2004; Monedero & Donne, 2000; McAinch et al., 2004; Siegler et al., 2006; Stanley & Buchheit, 2014). The absence of such research is surprising as long work interval sessions are frequently incorporated into the training programmes of endurance athletes (Buchheit & Laursen, 2013).

Many of the studies which have examined recovery interval intensity during cycling based HIIT using long work intervals (≥ 1-min) have used experimental designs which are not reflective of the type of HIIT sessions currently used by coaches and athletes in training programmes. Specifically, the use of long recovery durations (relative to the work interval duration; Siegler et al., 2006), a limited number of work intervals (Monedero & Donne, 200),

limited overall work interval duration (Stanley & Buchheit, 2014) and time to exhaustion work intervals (Dorado et al., 2004; Siegler et al., 2006). The constraints of these experimental designs preclude the practical application of their research findings.

As shown in table 2.2 there is considerable diversity in the HIIT protocols and participants examined, in addition to heterogeneity in methodologies and results presented in the current body of literature investigating recovery interval intensity. However, despite the diversity in the research, it is evident that there is not an optimal or one size fits all approach to prescribing recovery intensity. Recovery intensity appears to be dependent of HIIT protocol design and the desired training session outcome, hence the importance of further research to address the gaps in the understanding of the effect of recovery interval intensity on HIIT. To the authors knowledge there has not been a study investigating the acute physiological and perceptual effects of recovery interval intensity during cycling exercise, using HIIT sessions similar to those currently used by athletes and in HIIT research (Billat et al., 2001; Fiskerstrand & Seiler, 2004; Seiler et al., 2004; Steinacker et al., 1998; Stepto et al., 2001).

Statement of Purpose

The main aim of the study was to investigate the acute physiological and perceptual effects of a PA and two ACT recovery intensities during cycling based HIIT using long work intervals (≥ 1-min). The study also sought to add to the current understanding of how the manipulation of recovery interval intensity effects work interval and overall HIIT session performance.

Statement of Hypothesis

Null Hypothesis: Increasing ACT recovery intensity will have no effect on work interval PO and will not reduce the acute physiological responses, when compared to a PA recovery intensity. As measured by mean work interval and HIIT session: HR, B[La], RPE, sRPE, $\dot{V}O_2$, T@$\dot{V}O_{2max}$, T@HRmax, HHb, O_2Hb and TSI %.

Alternative Hypothesis: Increasing ACT recovery intensity will reduce work interval PO and will therefore reduce the acute physiological responses, when compared to a PA recovery intensity. As measured by mean work interval and HIIT session: HR, B[La], RPE, sRPE, $\dot{V}O_2$, T@$\dot{V}O_{2max}$, T@HRmax, HHb, O_2Hb and TSI %.

VI.II – Methods

Participants

Fourteen well trained cyclists were recruited to take part in the study. The participants characteristics and anthropometrics are presented in table 6.1.

All participants had a minimum of 2 years competitive racing experience and were in training for the next competitive season. In addition, all participants were using HIIT in their current training programmes (see *Table 6.1*).

Participants were required to complete a cycling experience questionnaire to ensure they met the inclusion criteria. The study was completed with full ethical approval from the University of Kent ethics committee, according to the Declaration of Helsinki standards. All participants provided signed informed consent and completed a health questionnaire prior to testing to ensure they were in full health and able to deal with the exercise demands of the study.

Table 6.1 – Participant characteristics/anthropometrics, $\dot{V}O_{2max}$ test, LT test and cycling history questionnaire results.

N = 14	Mean ± SD
Age (yrs.)	33 ± 13
Height (cm)	176.6 ± 5.9
Mass (kg)	70.6 ± 8.1
VL Skin Fold (mm)	9.5 ± 2.7
$\dot{V}O_{2max}$ ($L.min^{-1}$)	4.3 ± 0.6
Relative $\dot{V}O_{2max}$ ($ml.kg.min^{-1}$)	62 ± 9
MMP (W)	370 ± 56
Relative MMP ($W.kg^{-1}$)	5.2 ± 0.8
HRmax (bpm)	187 ± 11
PO at LT (W)	205 ± 44
PO at LTP (W)	273 ± 48
Thigh Circumference (cm)	55.1 ± 6.4
Years riding	10.6 ± 10.2
Years training	6.8 ± 6
Years competing	6.3 ± 5.4
Mean weekly training hours	9.1 ± 2.9
Hours of HIIT per Week	5.2 ± 1.5

Study Design

Each participant completed seven visits to the laboratory: Visit 1 being tests to identify the LT and $\dot{V}O_{2max}$, in addition to familiarising the participants with the laboratory environment and equipment. Visit 2 to 7 were the 6 x 4-min and 3 x 8-min HIIT sessions, with the recovery intervals completed at three different exercise intensities: PA, ACT recovery at 80% of PO at the LT (80A) and ACT recovery at 110% of PO at the LT (110A).

Visits were conducted on non-concurrent days and participants were instructed to refrain from any exercise in the day prior to testing and intense exercise in the two days prior. Participants were instructed to arrive euhydrated for each visit as they would be unable to drink for the duration of the exercise portion of the visit (due to wearing the face-mask to collect expired gases). Participants were advised to arrive in a post-prandial state, having eaten at least 4-hours prior to testing and were told to not consume caffeine within 4-hours and alcohol within 24-hours of testing.

Each participant completed all their visits to the laboratory at the same time of day to avoid any circadian variance. At each visit room temperature, humidity and pressure (mmHg) were recorded. An electric fan was placed 2 m in front of the participants to provide cooling during all tests if requested.

Visit 1: Lactate threshold and $\dot{V}O_{2max}$ tests

At the first visit, participants were measured for anthropometric values: height and mass.

Prior to starting the LT test resting B[La] samples were taken. The participants then completed a 10-min warm-up at 50 W. The test then commenced, with required PO set at 80 W for 4-min, after which the required PO increased by 20 W every 4 min, to allow time for the lactate to diffuse into the blood (Bentley et al., 2007). The 4-min increments continued until B[La] samples were reading > 4 mmol. Participants completed a cool down for 10-min at 50 W, after which they completed seated rest for 10-min, before commencing the $\dot{V}O_{2max}$ test protocol.

During the LT test B[La] samples were collected using fingertip capillary blood 30-s before the end of each stage. Blood samples were analysed using a Biosen C-Line (EKF Diagnostic, London, UK) and then safely disposed of in accordance with the Human Tissue Act. PO and HR were continuously measured throughout the test, and RPE measurements were asked at the end of each stage (Borg, 1982).

The first LT were assessed as the point at which B[La] breaks from linearity (Yoshida et al., 1987). The lactate turn-point (LTP) were assessed as the second break point after which B[La] begins to rise exponentially above 4 mmol (Faude et al., 2009).

The participants then completed the $\dot{V}O_{2max}$ test, procedures for which are described in the chapter III. General methods, section III.II – Determination of $\dot{V}O_{2max}$. Key results from of the LT test and $\dot{V}O_{2max}$ test are contained within Table 6.1.

Following the LT and $\dot{V}O_{2max}$ tests, participants were briefed on the procedures to be used in visits 2 to 7. Participants were also familiarised with the arterial occlusions, to ensure they were comfortable with the procedure. The circumference of the top of the right thigh was measured to make sure participants were fitted with the correct sized BP cuff, to ensure the fullest occlusion could be applied.

Visits 2 to 7: HIIT sessions

Participants completed both the 6 x 4-min and 3 x 8-min HIIT sessions three times (6 HIIT sessions in total), once with each of the three recovery interval intensities: PA, 80A and 110A. HIIT session methods and schematics can be found in chapter III. General Methods, section III.IV - HIIT session protocols and data collection methods.

Full methods for NIRS data collection during the HIIT sessions can be found in chapter III. General methods, section III.III - NIRS data collection during the HIIT sessions.

The ACT recovery intensities were calculated as 80% and 110% of the participants PO at the LT (*Table 6.2*). During the PA recovery intensity HIIT sessions participants were instructed to remain on the bike but to not turn the pedals.

Table 6.2 – 80A and 110A recovery intensities.

Measurement	Mean ± SD (CV%)
80A recovery intensity (W)	164 ± 35 (21)
110A recovery intensity (W)	225 ± 48 (21)
80A recovery intensity % of MMP	44 ± 4 (9)
110A recovery intensity % of MMP	60 ± 5 (9)

All recovery interval durations were a standardised 2:1 work recovery ratio (120-s recovery duration and 240-s recovery duration, for the 6 x 4-min and 3 x 8-min HIIT sessions respectively). The HIIT sessions were randomised and completed on non-consecutive days.

Statistical Analysis

Data were presented as individual values or mean ± SD (unless specified otherwise). Statistical analyses were conducted using IBM SPSS Statistics 24 (IBM, Armonk, New York, USA). Visual inspection of Q-Q plots and Shapiro-Wilk statistics were used to check whether data were normally distributed. Three separate two-way repeated measures ANOVA, 1) two HIIT protocols (6 x 4-min vs 3 x 8-min) X three recovery intensities (PA, 80A and 110A); 2) three recovery intensities (PA, 80A and 110A) X number of work intervals; 3) three recovery intensities (PA, 80A and 110A) X number of recovery intervals were used to determine between and within condition effects for all dependent variables. Bonferroni *post hoc* comparisons were used when a main effect or interaction was significant. The criteria of $P < 0.05$ was used for the detection of significance in all cases.

VI.III – Results

VI.III.1 – Key physiological HIIT session results

Power Output Results

There was a significant interaction between HIIT protocol and recovery intensity for mean session PO (main effect $F = 21.791$, $P < 0.001$). Mean session POs were significantly higher during the 6 x 4-min HIIT protocols when compared to the 3 x 8-min HIIT protocols (main effect of protocol $F = 10.623$, $P = 0.006$). There was a significant effect of recovery intensity on mean session PO during the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of intensity $F = 329.292$, $P < 0.001$). *Post hoc* tests revealed that the mean session PO was significantly higher during the 110A intensity compared to the 80A and PA intensities, during the 6 x 4-min and 3 x 8-min HIIT protocols ($P < 0.001$). Mean session PO was significantly higher during the 80A intensity compared to the PA intensity, during the 6 x 4-min and 3 x 8-min HIIT protocols ($P < 0.001$; *Figures 6.1a & 6.1b*).

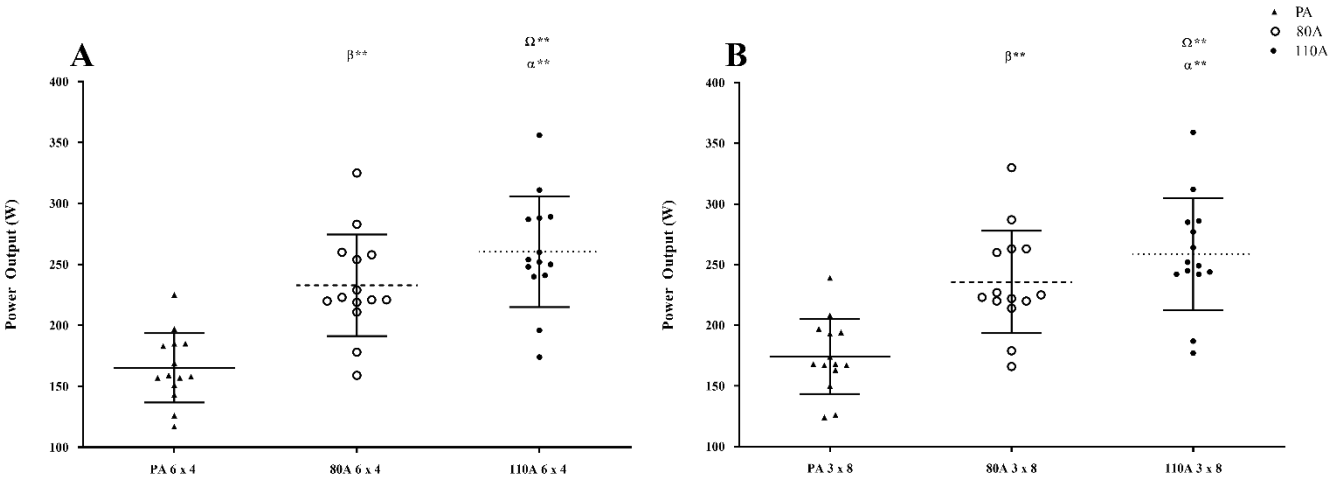


Figure 6.1 – A = Mean session PO during the 6 x 4-min HIIT sessions, **B** = Mean session PO during the 3 x 8-min HIIT sessions (Mean ± SD). **** = *P* < 0.001**, **Ω** = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

There was no interaction between HIIT protocol and recovery intensity for work interval PO (main effect $F = 2.181$, $P = 0.133$). There was no significant interaction between recovery intensity and work interval during the 6 x 4-min (main effect $F = 1.612$, $P = 0.110$; *Figure 6.2a*) and 3 x 8-min HIIT protocols (main effect $F = 2.729$, $P = 0.039$; *Figure 6.2b*), demonstrating that the pattern of change in PO across the work intervals was not different between recovery intensities.

Work interval POs were significantly higher during the 6 x 4-min HIIT protocols when compared to the 3 x 8-min HIIT protocols (main effect of protocol $F = 27.275$, $P < 0.001$). There was a significant effect of recovery intensity on work interval PO during the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of intensity $F = 9.137$, $P = 0.001$). *Post hoc* tests revealed that work interval PO was significantly higher during the PA intensity compared to the 80A and 110A intensities, during the 6 x 4-min and 3 x 8-min HIIT protocols ($P < 0.05$). There were no significant differences in work interval PO between the 80A and 110A intensities, during the 6 x 4-min and 3 x 8-min HIIT protocols ($P > 0.05$; *Figures 6.2a & 6.2b*).

There was an effect of interval during the 6 x 4-min HIIT protocols (main effect of interval $F = 4.473$, $P = 0.001$; *Figure 6.2a*) and but not the 3 x 8-min HIIT protocols (main effect of interval $F = 0.371$, $P = 0.694$; *Figure 6.2b*).

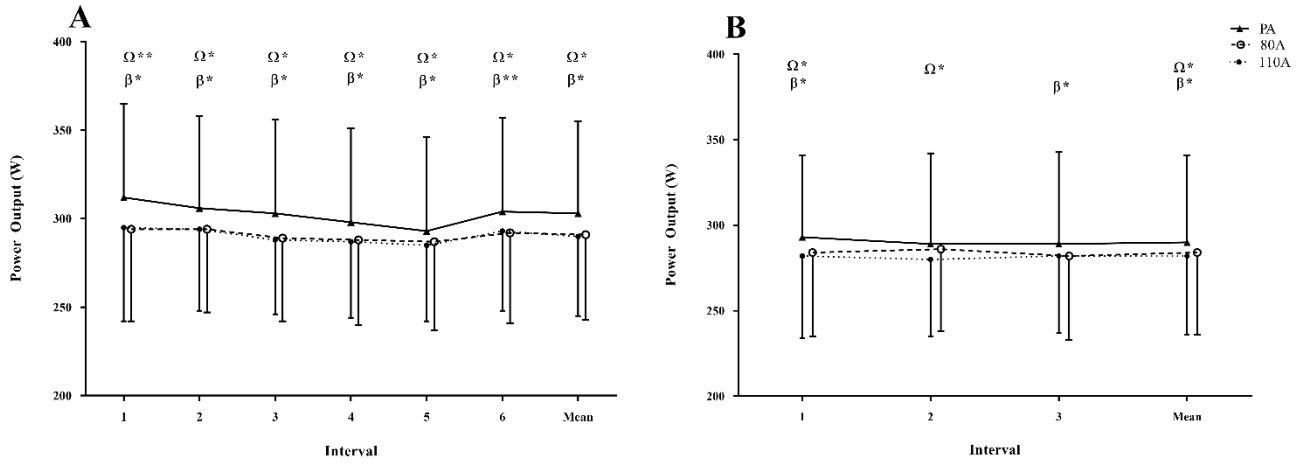


Figure 6.2 – A = Work interval PO during the 6 x 4-min HIIT sessions, **B** = Work interval PO during the 3 x 8-min HIIT sessions (Mean ± SD). *** = *P* < 0.05, ** = *P* < 0.001**, **Ω** = Significant difference between PA & 110A, β = Significant difference between PA & 80A.

Heart Rate Results

There was no interaction between HIIT protocol and recovery intensity for mean session HR (main effect $F = 1.752$, $P = 0.197$). Mean session HRs were significantly higher during the 6 x 4-min HIIT protocols when compared to the 3 x 8-min HIIT protocols (main effect of protocol $F = 12.917$, $P = 0.004$). There was a significant effect of recovery intensity on mean session HR during the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of intensity $F = 49.903$, $P < 0.001$). *Post hoc* tests revealed that mean session HR was significantly higher during the 110A intensity compared to the 80A intensity ($P < 0.05$) and PA intensity ($P < 0.001$), during the 6 x 4-min and 3 x 8-min HIIT protocols. Mean session HR was significantly higher during the 80A intensity compared to the PA intensity, during the 6 x 4-min and 3 x 8-min HIIT protocols ($P < 0.05$; *Figures 6.3a & 6.3b*).

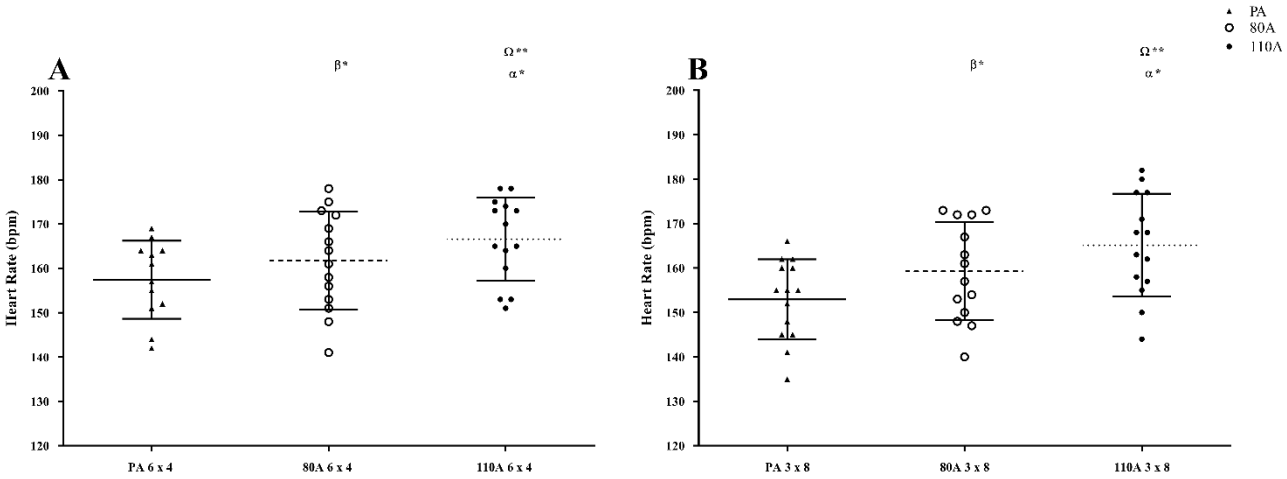


Figure 6.3 – A = Mean session HR during the 6 x 4-min HIIT sessions, **B** = Mean session HR during the 3 x 8-min HIIT sessions (Mean ± SD). *** = *P* < 0.05, ** = *P* < 0.001**, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

There was no interaction between HIIT protocol and recovery intensity for work interval HR (main effect $F = 1.910$, $P = 0.172$; *Figures 6.4a & 6.4b*). There was a significant interaction between recovery intensity and work interval during the 6 x 4-min (main effect $F = 8.147$, $P < 0.001$; *Figure 6.4a*) and 3 x 8-min HIIT protocols (main effect $F = 12.993$, $P < 0.001$; *Figure 6.4b*), demonstrating that the pattern of change in HR across the work intervals was different between recovery intensities.

There was no significant difference in work interval HR between the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of protocol $F = 1.745$, $P = 0.213$). There was no effect of recovery intensity on the mean HR of the work intervals during the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of intensity $F = 2.758$, $P = 0.085$; *Figures 6.4a & 6.4b*).

There was an effect of interval during the 6 x 4-min (main effect of interval $F = 86.818$, $P < 0.001$; *Figure 6.4a*) and 3 x 8-min HIIT protocols (main effect of interval $F = 64.214$, $P < 0.001$; *Figure 6.4b*), showing that there was significant increase in work interval HR across the HIIT sessions.

Post hoc tests revealed the HR of work intervals 3 to 6 of the 6 x 4-min HIIT protocol to be significantly higher during the 110A intensity compared to the 80A intensity ($P < 0.05$). HR of work intervals 1, 5 and 6 of the 6 x 4-min HIIT protocol were significantly higher during the 110A intensity compared to the PA intensity ($P < 0.05$; *Figure 6.4a*). HR of work interval 1 of the 3 x 8-min HIIT protocol was significantly higher during the PA intensity compared to the 80A ($P < 0.001$) and 110A ($P < 0.05$; *Figure 6.4b*) intensities.

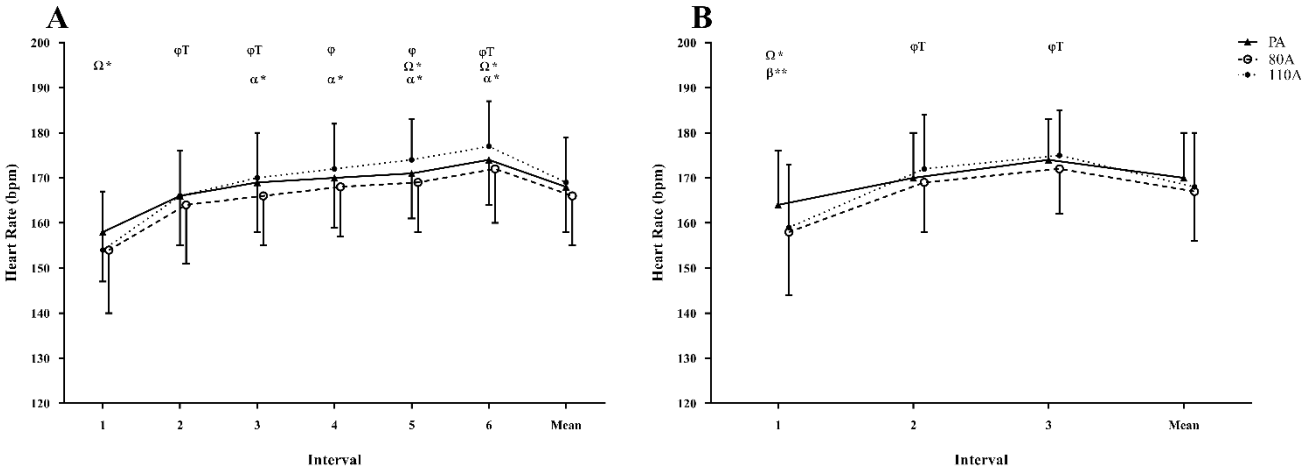


Figure 6.4 – A = Work interval HR during the 6 x 4-min HIIT sessions, **B** = Work interval HR during the 3 x 8-min HIIT sessions (Mean ± SD). φ = Significant difference from interval 1, T = Significant difference from previous interval, *** = *P* < 0.05, ** = *P* < 0.001**, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

B[La] Results

There was a significant interaction between HIIT protocol and recovery intensity for the mean B[La] response (main effect $F = 5.3$, $P = 0.024$). There were no significant differences in mean B[La] response between the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of protocol $F = 1.385$, $P = 0.262$; *Figures 6.5a & 6.5b*). There was a significant effect of recovery intensity on mean B[La] response during the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of intensity $F = 13.632$, $P = 0.001$). *Post hoc* tests revealed that mean B[La] was significantly higher during the PA 6 x 4-min HIIT session, compared to the 80A and 110A 6 x 4-min HIIT sessions ($P < 0.05$; *Figure 6.5a*).

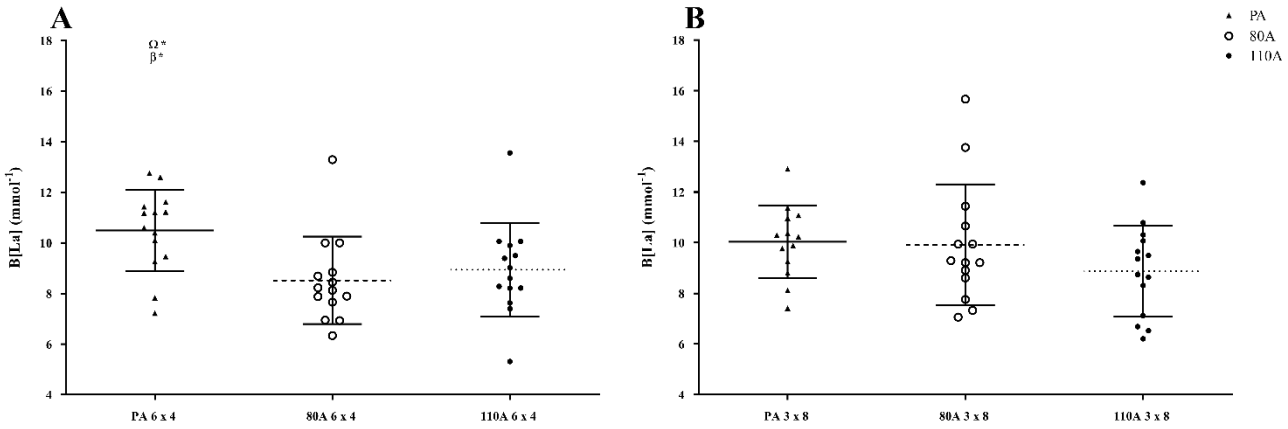


Figure 6.5 – A = Mean B[La] response during the 6 x 4-min HIIT sessions, **B** = Mean B[La] response during the 3 x 8-min HIIT sessions (Mean ± SD). ***** = ***P*** **< 0.05**, **Ω** = Significant difference between PA & 110A, β = Significant difference between PA & 80A.

There was no significant interaction between recovery intensity and work interval during the 6 x 4-min (main effect $F = 1.744$, $P = 0.077$; *Figure 6.6a*) and 3 x 8-min (main effect $F = 2.055$, $P = 0.102$; *Figure 6.6b*) HIIT protocols, demonstrating that the pattern of change in B[La] across the work intervals was not different between recovery intensities. There was an effect of interval during the 6 x 4-min (main effect of interval $F = 18.845$, $P < 0.001$; *Figure 6.6a*) and 3 x 8-min HIIT protocols (main effect of interval $F = 13.461$, $P < 0.001$; *Figure 6.6b*), showing that there was significant increase in the work interval B[La] across the HIIT sessions.

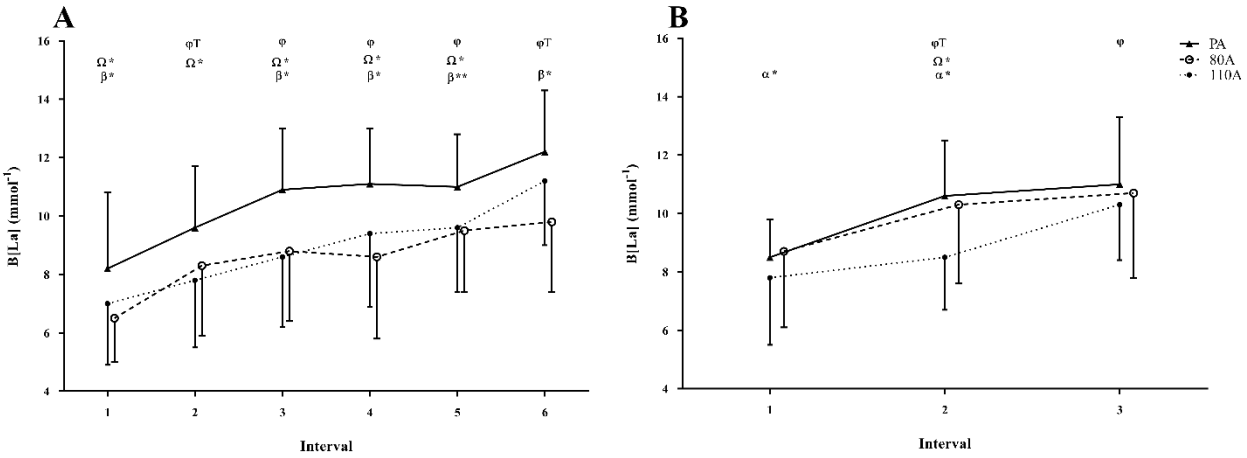


Figure 6.6 – A = Work interval B[La] response during the 6 x 4-min HIIT sessions, **B** = Work interval B[La] response during the 3 x 8-min HIIT sessions (Mean ± SD). φ = Significant difference from interval 1, T = Significant difference from previous interval, *** = *P* < 0.05, ** = *P* < 0.001**, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

V̇O₂ Results

There was no interaction between HIIT protocol and recovery intensity for mean session $\dot{V}O_2$ (main effect $F = 1.991$, $P = 0.157$). Mean session $\dot{V}O_2$ was significantly higher during the 6 x 4-min HIIT protocols when compared to the 3 x 8-min HIIT protocols (main effect of protocol $F = 8.619$, $P = 0.012$). There was a significant effect of recovery intensity on mean session $\dot{V}O_2$ during the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of intensity $F = 54.645$, $P < 0.001$). *Post hoc* tests revealed that mean session $\dot{V}O_2$ was significantly higher during the 110A intensity compared to the 80A and PA intensities, during the 6 x 4-min and 3 x 8-min HIIT protocols ($P < 0.001$). Mean session $\dot{V}O_2$ was significantly higher during the 80A intensity compared to the PA intensity, during the 6 x 4-min and 3 x 8-min HIIT protocols ($P < 0.05$; *Figures 6.7a & 6.7b*).

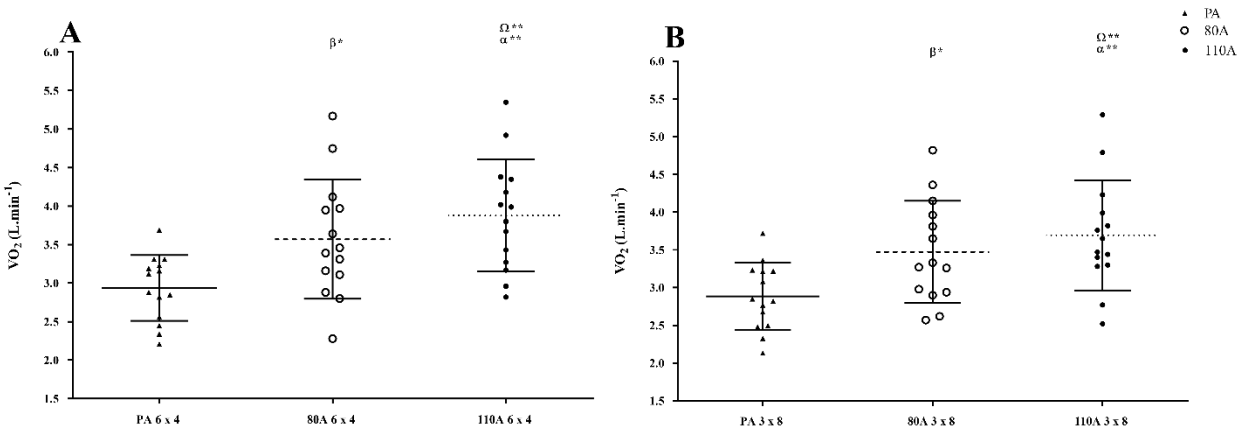


Figure 6.7 – A = Mean session $\dot{V}O_2$ during the 6 x 4-min HIIT sessions, **B** = Mean session $\dot{V}O_2$ during the 3 x 8-min HIIT sessions (Mean ± SD). *** = *P* < 0.05, ** = *P* < 0.001**, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

There was a significant interaction between HIIT protocol and recovery intensity for work interval $\dot{V}O_2$ (main effect $F = 6.002$, $P = 0.007$). There was a significant interaction between recovery intensity and work interval during the 6 x 4-min (main effect $F = 6.053$, $P < 0.001$; *Figure 6.8a*) and 3 x 8-min HIIT protocols (main effect $F = 4.106$, $P = 0.006$; *Figure 6.8b*), demonstrating that the pattern of change in $\dot{V}O_2$ across the work intervals was different between recovery intensities.

There was no significant difference in work interval $\dot{V}O_2$ between the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of protocol $F = 0.04$, $P = 0.844$). There was no effect of recovery intensity on work interval $\dot{V}O_2$ during the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of intensity $F = 1.346$, $P = 0.278$).

There was an effect of interval during the 6 x 4-min (main effect of interval $F = 33.477$, $P < 0.001$; *Figure 6.8a*) and 3 x 8-min HIIT protocols (main effect of interval $F = 37.31$, $P < 0.001$; *Figure 6.8b*), showing that there was significant increase in the work interval $\dot{V}O_2$ across the HIIT sessions.

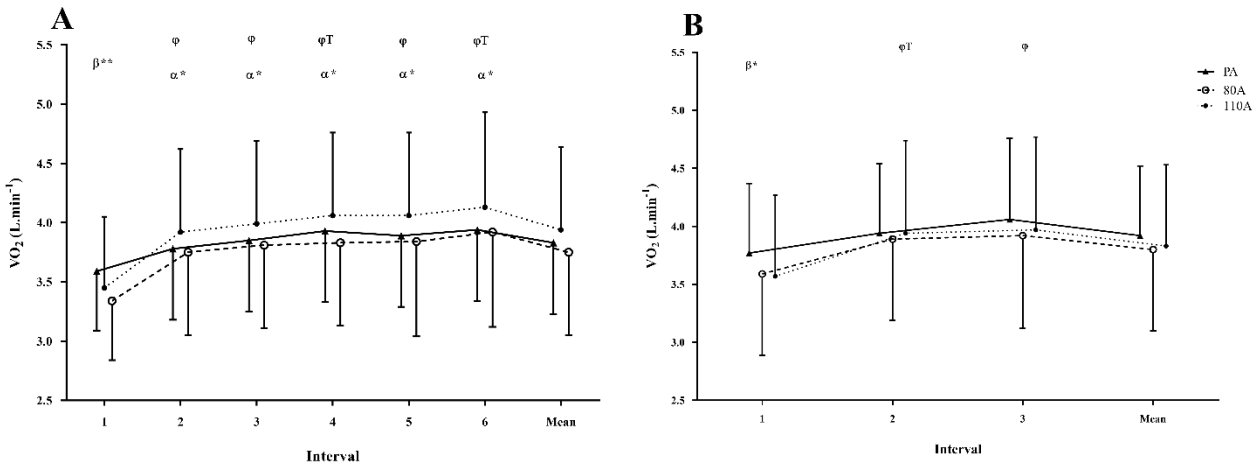


Figure 6.8 – A = Work interval $\dot{V}O_2$ during the 6 x 4-min HIIT sessions, **B** = Work interval $\dot{V}O_2$ during the 3 x 8-min HIIT sessions (Mean ± SD). φ = Significant difference from interval 1, T = Significant difference from previous interval, *** = *P* < 0.05, ** = *P* < 0.001,** β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

VI.III.2 – Key perceptual HIIT session results

Mean RPE was significantly higher in the PA recovery condition, when compared to the 80A recovery condition of the 3 x 8-min HIIT session ($P < 0.05$). There was no significant difference between the other recovery conditions ($P > 0.05$; *Figure 6.9b*). There were no significant differences in mean RPE between the three recovery intensities of the 6 x 4-min HIIT session ($P > 0.05$; *Figure 6.9a*). There was no significant difference in the RPE values reported between the 6 x 4-min and 3 x 8-min HIIT protocols ($P > 0.05$).

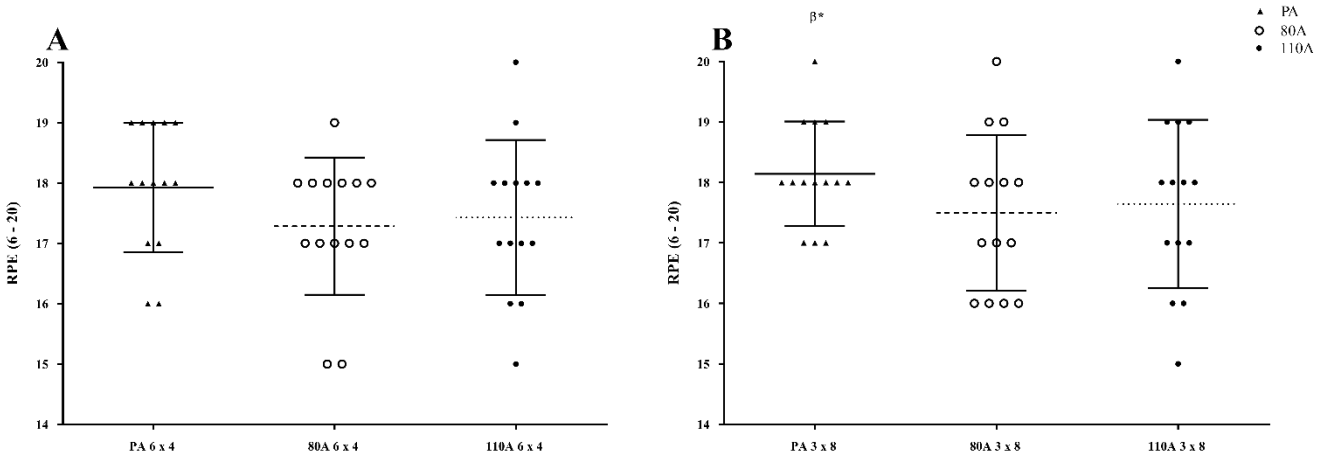


Figure 6.9 – A = Mean RPE during the 6 x 4-min HIIT sessions, **B** = Mean RPE during the 3 x 8-min HIIT sessions (Mean ± SD). *** = *P* < 0.05**, β = Significant difference between PA & 80A.

There was no significant interaction between recovery intensity and work interval during the 6 x 4-min (main effect $F = 1.814$, $P = 0.064$; *Figure 6.10a*) and 3 x 8-min HIIT protocols (main effect $F = 1.591$, $P = 0.191$; *Figure 6.10b*), demonstrating that the pattern of change in RPE across the work intervals was not different between recovery intensities. There was an effect of interval during the 6 x 4-min (main effect of interval $F = 85.108$, $P < 0.001$; *Figure 6.10a*) and 3 x 8-min HIIT protocols (main effect of interval $F = 79.751$, $P < 0.001$; *Figure 6.10b*), showing that there was significant increase in the RPE values reported after each subsequent work interval.

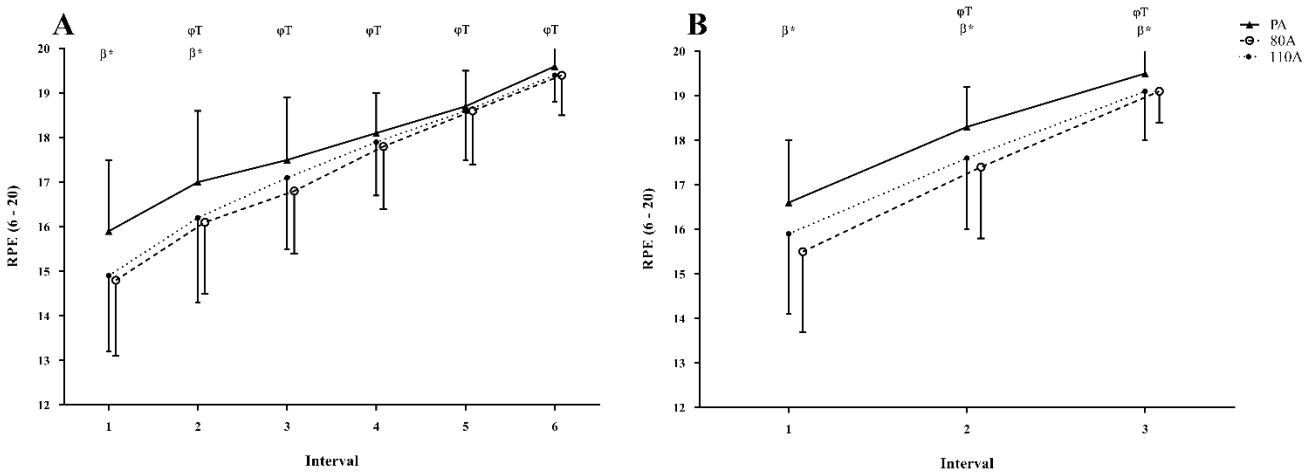


Figure 6.10 – A = Mean work interval RPE during the 6 x 4-min HIIT sessions, **B** = Mean work interval RPE during the 3 x 8-min HIIT sessions (Mean ± SD). φ = Significant difference from interval 1, T = Significant difference from previous interval, *** = *P* < 0.05**, β = Significant difference between PA & 80A.

sRPE was significantly higher after the 110A recovery condition, when compared to the PA and 80A recovery conditions ($P < 0.05$), there was no significant difference in sRPE between the PA and 80A recovery conditions ($P > 0.05$), in both the 6 x 4-min and 3 x 8-min HIIT sessions (*Figures 6.11a & 6.11b*). There was no significant difference in the sRPE values reported between the 6 x 4-min and 3 x 8-min HIIT protocols ($P > 0.05$).

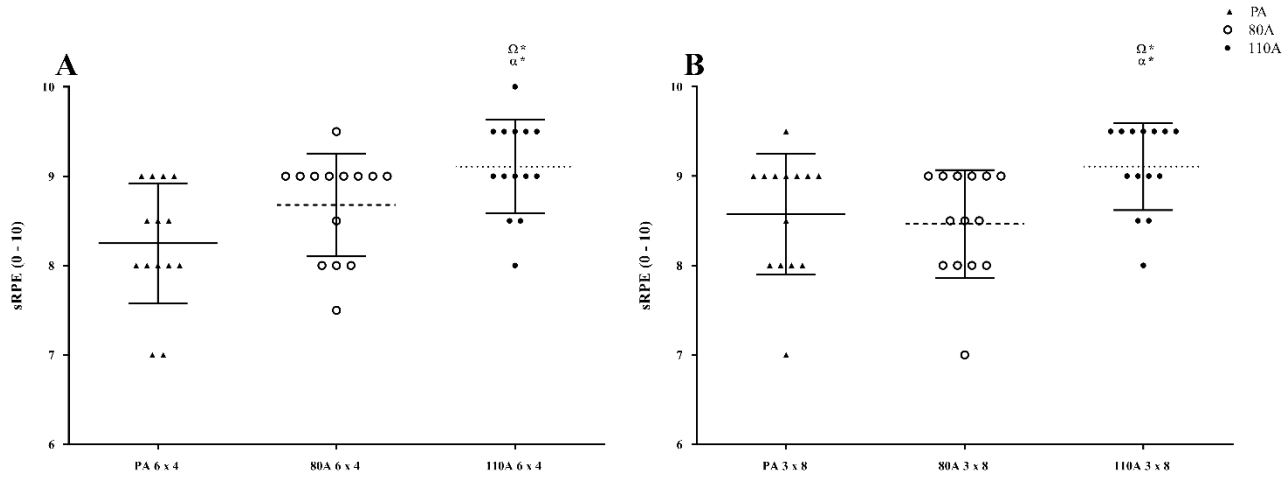


Figure 6.11 – A = sRPE of the 6 x 4-min HIIT sessions, **B** = sRPE of the 3 x 8-min HIIT sessions (Mean ± SD). *** = *P* < 0.05**, Ω = Significant difference between PA & 110A, α = Significant difference between 80A & 110A.

VI.III.3 – Key recovery interval results

Heart Rate Results

Mean recovery interval HR was significantly higher during the 110A recovery intensity, when compared to the PA and 80A recovery intensities, during the 6 x 4-min ($P < 0.05$; *Figure 6.12a*) and 3 x 8-min HIIT protocols ($P < 0.001$; *Figure 6.12b*). Mean recovery interval HR was significant higher during the 80A recovery intensity, when compared to the PA recovery intensity, during the 6 x 4-min and 3 x 8-min HIIT protocols ($P < 0.001$; *Figures 6.12a & 6.12b*).

The 6 x 4-min HIIT protocols resulted in significantly higher mean recovery interval HRs, when compared to the 3 x 8-min HIIT protocols (main effect $P < 0.001$). *Post hoc* pairwise comparisons revealed that recovery interval HR was significantly higher in the PA and 80A 6 x 4-min HIIT sessions, when compared to the PA and 80A 3 x 8-min HIIT sessions ($P < 0.001$). No significant differences in mean recovery interval HR were found between the 110A 6 x 4-min and 110A 3 x 8-min HIIT sessions ($P > 0.05$; *Figures 6.12a & 6.12b*).

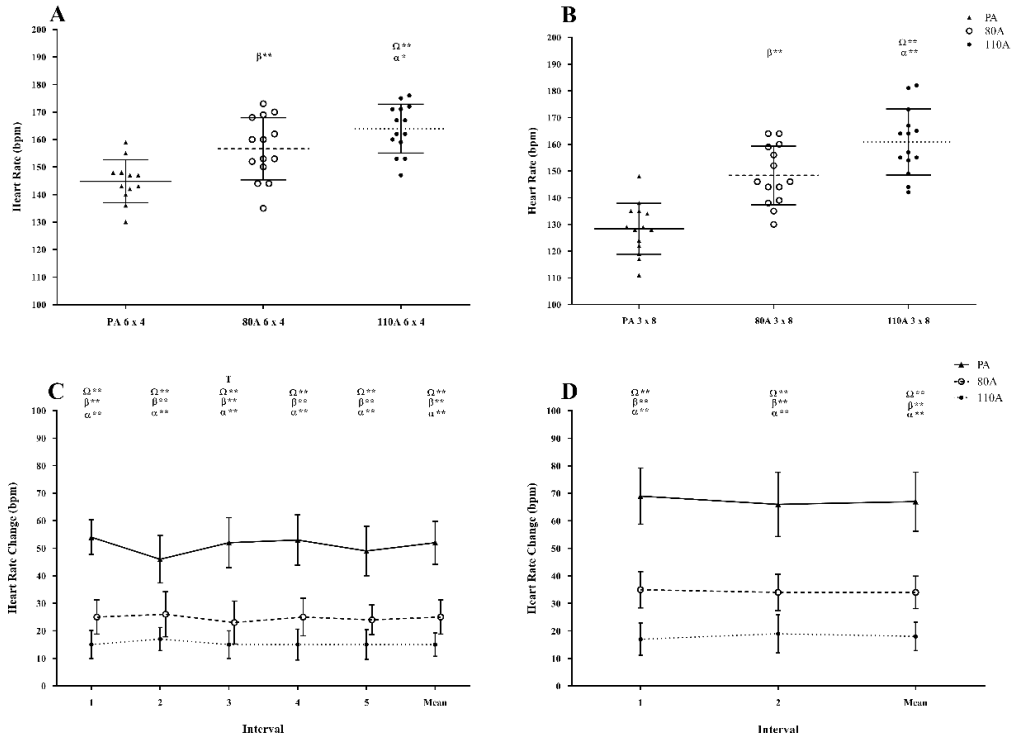


Figure 6.12 – A = Mean recovery interval HR during the 6 x 4-min HIIT session, **B** = Mean recovery interval HR during the 3 x 8-min HIIT sessions, **C** = HR change during the recovery intervals of the 6 x 4-min HIIT sessions, **D** = HR change during the recovery intervals of the 3 x 8-min HIIT sessions (Mean ± SD). T = Significant difference from previous interval, *** = *P* < 0.05, ** = *P* < 0.001**, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

There was a significant interaction between HIIT protocol and recovery intensity (main effect $F = 30.22$, $P < 0.001$). There was no significant interaction between recovery intensity and interval during the 6 x 4-min HIIT protocols (main effect $F = 1.929$; $P = 0.065$), demonstrating that the pattern of change in HR across the recovery intervals was not different between recovery intensities (*Figure 6.12c*). There was a significant interaction between recovery intensity and recovery interval during the 3 x 8-min HIIT protocols ($F = 3.728$; $P = 0.038$), demonstrating that the pattern of change in HR across the recovery intervals was different between recovery intensities (*Figure 6.12d*).

HR recovered to a greater extent during the 3 x 8-min HIIT protocols, when compared to the 6 x 4-min HIIT protocols (main effect of protocol $F = 98.446$, $P < 0.001$; *Figures 6.12c & 6.12d*)

There was a significant effect of recovery intensity (main effect of intensity $P < 0.001$), with HR recovering to a greater extent during the PA recovery intensity, compared to the 80A and 110A recovery intensities during both the 6 x 4-min and 3 x 8-min HIIT sessions (*Figures 6.12c & 6.12d*).

There was no effect of interval for the 6 x 4-min (main effect of interval $F = 1.812$; $P = 0.144$; *Figure 6.12c*) and 3 x 8-min HIIT protocols (main effect of interval $F = 0.095$; $P = 0.763$; *Figure 6.12d*), showing that there was no significant difference in the magnitude of change in HR between recovery intervals, throughout each HIIT session.

V̇O₂ Results

Mean recovery interval V̇O₂ was significantly higher during the 110A recovery intensity, when compared to the PA and 80A recovery intensities, during the 6 x 4-min ($P < 0.001$; *Figure 6.13a*) and 3 x 8-min HIIT protocols ($P < 0.001$; *Figure 6.13b*). Mean recovery interval V̇O₂ was significant higher during the 80A recovery intensity, when compared to the PA recovery intensity during the 6 x 4-min and 3 x 8-min HIIT protocols ($P < 0.001$; *Figures 6.13a & 6.13b*).

The 6 x 4-min HIIT protocols resulted in significantly higher mean recovery interval V̇O₂ values, when compared to the 3 x 8-min HIIT protocols (main effect $P < 0.001$). *Post hoc* pairwise comparisons revealed that mean recovery interval V̇O₂ of the 6 x 4-min HIIT sessions was significantly higher at all three recovery intensities, when compared to the 3 x 8-min HIIT sessions ($P < 0.001$; *Figures 6.13a & 6.13b*).

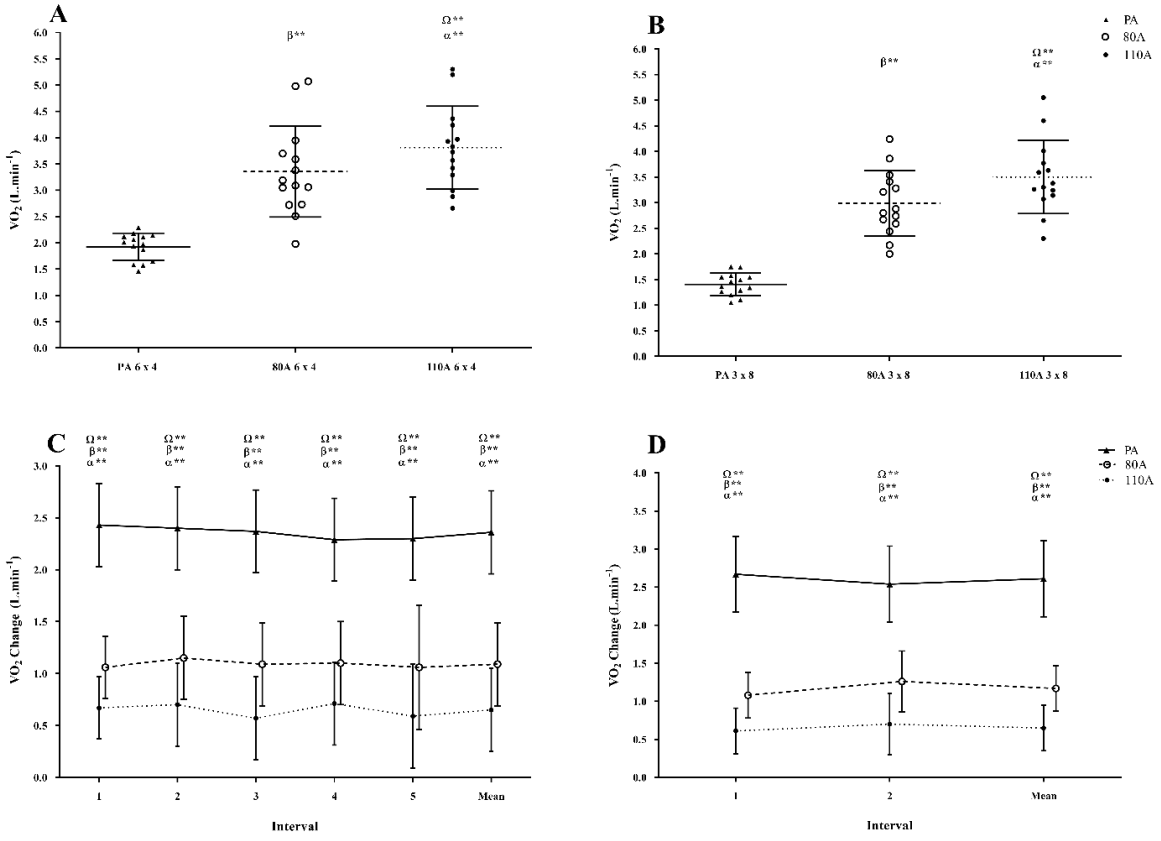


Figure 6.13 – A = Mean recovery interval V̇O₂ during the 6 x 4-min HIIT sessions, **B** = Mean recovery interval V̇O₂ during the 3 x 8-min HIIT sessions, **C** = V̇O₂ change during the recovery intervals of the 6 x 4-min HIIT sessions, **D** = V̇O₂ change during the recovery intervals of the 3 x 8-min HIIT sessions (Mean ± SD). **** = *P* < 0.001**, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

There was no significant interaction between recovery intensity and interval during the 6 x 4-min HIIT protocols (main effect $F = 1.17$; $P = 0.324$), demonstrating that the pattern of change in $\dot{V}O_2$ across the recovery intervals was not different between recovery intensities (*Figure 6.13c*). There was a significant interaction between recovery intensity and interval during the 3 x 8-min HIIT protocols (main effect $F = 7.685$; $P = 0.002$), demonstrating that the pattern of change in $\dot{V}O_2$ across the recovery intervals was different between recovery intensities (*Figure 6.13d*).

There was no significant difference in the recovery of $\dot{V}O_2$ between the 6 x 4-min and 3 x 8-min HIIT protocols (main effect of protocol $F = 2.865$, $P = 0.114$; *Figures 6.13c & 6.13d*).

There was a significant effect of recovery intensity (main effect of intensity $P < 0.001$), with $\dot{V}O_2$ recovering to a greater extent during the PA recovery intensity compared to the 80A and 110A recovery intensities, during the 6 x 4-min and 3 x 8-min HIIT protocols (*Figures 6.13c & 6.13d*).

There was no effect of interval for the 6 x 4-min (main effect of interval $F = 0.98$; $P = 0.427$; *Figure 6.13c*) and 3 x 8-min HIIT protocols (main effect of interval $F = 0.464$; $P = 0.508$; *Figure 6.13d*), showing that there was no significant difference in the magnitude of change in $\dot{V}O_2$ between recovery intervals, throughout each HIIT session.

VI.III.4 – Time at % of $\dot{V}O_{2max}$, % of MMP and % of HRmax results

ACT recovery intensities (80A and 110A) significantly reduced the time participants spent above 90 and 95% of MMP, when compared to the PA recovery intensity during the 6 x 4-min HIIT sessions ($P < 0.05$). Participants spent significantly longer above 90% of MMP during the PA 3 x 8-min HIIT session, when compared to the 80A and 110A 3 x 8-min HIIT sessions ($P < 0.05$). The PA 3 x 8-min HIIT session resulted in a significantly longer time spent above 95% of MMP, when compared to the 110A 3 x 8-min HIIT session ($P < 0.05$), but not the 80A 3 x 8-min HIIT session ($P > 0.05$). The 6 x 4-min HIIT protocols produced significantly longer times spent above 90 and 95% of MMP, when compared to the 3 x 8-min HIIT protocols ($P < 0.05$; *Table 6.3*).

There were no significant differences in the time participants spent above 90 and 95% of $\dot{V}O_{2max}$ during the work intervals, between all three recovery intensity conditions of the 6 x 4-min HIIT sessions ($P > 0.05$). Participants spent significantly longer above 90% of $\dot{V}O_{2max}$ during the PA 3 x 8-min HIIT session, when compared to the 80A 3 x 8-min HIIT session ($P < 0.05$), but not the 110A 3 x 8-min HIIT session. There were no significant differences in the time spent above 95% of $\dot{V}O_{2max}$ during the work intervals, between all three recovery intensity conditions of the 3 x 8-min HIIT sessions ($P > 0.05$). Both the 6 x 4-min and 3 x 8-min HIIT protocols produced similar times spent above 90 and 95% of $\dot{V}O_{2max}$ during the work intervals ($P > 0.05$; *Table 6.3*).

Participants spent significantly longer above 95% of HRmax during the PA 6 x 4-min HIIT session, when compared to the 80A and 110A 6 x 4-min HIIT sessions ($P < 0.05$). There were no significant differences in the time spent above 90% of HRmax between all three recovery intensities during the 6 x 4-min HIIT session ($P > 0.05$). The PA 3 x 8-min HIIT session resulted in a significantly longer time spent above 90% of HRmax, when compared to the 80A 3 x 8-min HIIT session ($P < 0.05$), but not the 110A 3 x 8-min HIIT session ($P > 0.05$). There were no significant differences in the time spent above 95% of HRmax between all three recovery intensities during the 3 x 8-min HIIT session ($P > 0.05$). Both the 6 x 4-min and 3 x 8-min HIIT protocols produced similar times spent above 90 and 95% of HRmax during the work intervals ($P > 0.05$; *Table 6.3*).

Table 6.3 – Time (s) and percentage of the work intervals spent above 90 and 95% of $\dot{V}O_{2max}$, MMP and HRmax (Mean ± SD).

		Time at % $\dot{V}O_{2max}$		Time at % MMP		Time at % HRmax	
		90	**95**	**90**	**95**	**90**	**95**
Time (s) and % of work intervals	**PA. 6 x 4**	806 ± 266 56 ± 18.4%	516 ± 263 35.8 ± 18.3%	89 ± 76 6.2 ± 5.3% **Ω*β***	52 ± 50 3.5 ± 3.4% **Ω*β***	954 ± 145 66.2 ± 10.1%	591 ± 221 41 ± 15.3% **Ω*β***
	80A. 6 x 4	669 ± 392 46.4 ± 27.2%	444 ± 328 30.9 ± 22.8%	19 ± 28 1.3 ± 1.9%	15 ± 25 1 ± 1.8%	734 ± 267 50.9 ± 18.5%	254 ± 251 17.6 ± 17.4%
	110A. 6 x 4	749 ± 417 52 ± 29%	523 ± 384 36.3 ± 26.6%	26 ± 32 1.8 ± 2.2%	15 ± 23 1.1 ± 1.6%	902 ± 165 62.7 ± 11.4%	333 ± 236 23.1 ± 16.4%
	PA. 3 x 8	841 ± 321 58.4 ± 22.2 % **β***	499 ± 301 34.7 ± 20.9%	48 ± 39 3.4 ± 2.8% **Ω*β***	27 ± 29 1.9 ± 2% **Ω***	962 ± 218 66.8 ± 15.1% **β***	539 ± 268 37.4 ± 18.6%
	80A. 3 x 8	686 ± 320 47.6 ± 22.2%	383 ± 274 26.6 ± 19%	19 ± 28 1.3 ± 1.9%	14 ± 24 1 ± 1.7%	817 ± 299 56.8 ± 20.7%	363 ± 288 25.2 ± 20%
	110A. 3 x 8	640 ± 373 44.4 ± 25.9%	377 ± 332 26.2 ± 23.1%	17 ± 25 1.2 ± 1.7%	10 ± 14 0.7 ± 1%	887 ± 215 61.6 ± 15%	350 ± 220 24.3 ± 15.3%

* = $P < 0.05$, ** = $P < 0.001$, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

The 90 and 95% of MMP results are the same for work intervals and whole HIIT session (*Tables 6.3 & 6.4*), as the PO of the recovery intervals was < 90% of MMP.

Recovery intensity had no effect on the percentage of the whole HIIT session spent 90 and 95% of $\dot{V}O_{2max}$ during the 6 x 4-min protocols ($P > 0.05$). The PA condition resulted in a significantly greater percentage of the whole 3 x 8-min HIIT session spent above 90% of $\dot{V}O_{2max,}$ when compared to the 80A condition ($P < 0.05$), but not the 110A condition ($P > 0.05$). Recovery intensity had no effect on the percentage of the whole HIIT session spent above 95% $\dot{V}O_{2max}$ during the 3 x 8-min HIIT protocols ($P > 0.05$). There was no significant difference between the 6 x 4-min and 3 x 8-min HIIT protocols in the percentage of the whole session spent above 90 and 95% of $\dot{V}O_{2max}$ ($P > 0.05$; *Table 6.4*).

Participants spent significantly greater percentage of the whole 6 x 4-min HIIT session above 90% of HRmax during the 110A condition, when compared to the 80A ($P < 0.05$), but not the PA condition ($P > 0.05$). Participants spent significantly greater percentage of the whole 6 x 4-min HIIT session above 95% of HRmax during the PA condition, when compared to the 80A and 110A conditions ($P < 0.05$). The PA 3 x 8-min HIIT session resulted in significantly greater percentage of the whole HIIT session spent above 90% of HRmax, when compared to the 80A ($P < 0.05$), but not the 110A 3 x 8-min HIIT session ($P > 0.05$). There were no significant differences in the percentage of the whole 3 x 8-min HIIT sessions spent above 95% of HRmax between all three recovery intensities ($P > 0.05$). There was no significant difference between the 6 x 4-min and 3 x 8-min HIIT protocols in the percentage of the whole session spent above 90 and 95% of HRmax ($P > 0.05$; *Table 6.4*).

(In order to take into account differences in HIIT session duration between the 6 x 4-min and 3 x 8-min HIIT protocols, data were analysed as percentages of the whole HIIT session; *Table 6.4*)

Complete tables of all percentage results including time participants spent at 60, 70, 80, 90 and 95% of MMP, HRmax and $\dot{V}O_{2max}$ during the HIIT sessions can be found in IX. Appendix, section IX.II – Additional results from study two.

Table 6.4 – Time (s) and percentage of the whole HIIT session spent above 90 and 95% of $\dot{V}O_{2max}$, MMP and HRmax (Mean ± SD).

		Time at % $\dot{V}O_{2max}$		Time at % MMP		Time at % HRmax	
		90	**95**	**90**	**95**	**90**	**95**
Time (s) and % of HIIT Session	**PA. 6 x 4**	834 ± 280 40.9 ± 13.7%	536 ± 279 26.3 ± 13.7%	89 ± 76 4.3 ± 3.7% **Ω*β***	52 ± 50 2.6 ± 2.5% **Ω*β***	1091 ± 154 53.5 ± 7.6%	678 ± 248 33 ± 12% **Ω*β***
	80A. 6 x 4	786 ± 485 38.6 ± 23.8%	504 ± 380 24.7 ± 18.6%	19 ± 28 0.9 ± 1.4%	15 ± 25 0.7 ± 1.2%	883 ± 348 43.3 ± 17%	317 ± 299 15.5 ± 14.7%
	110A. 6 x 4	973 ± 558 47.6 ± 27.2%	659 ± 492 32.3 ± 24.1	26 ± 32 1.3 ± 1.6%	15 ± 23 0.7 ± 1.1%	1183 ± 240 58 ± 11.8% **α***	385 ± 281 18.8 ± 13.8%
	PA. 3 x 8	861 ± 325 44.8 ± 16.9% **β***	513 ± 306 26.7 ± 16%	48 ± 39 2.5 ± 2% **Ω*β***	27 ± 29 1.4 ± 1.4% **Ω***	1026 ± 228 53.4 ± 11.9% **β***	569 ± 276 29.7 ± 14.4%
	80A. 3 x 8	716 ± 331 37.3 ± 17.2%	399 ± 284 20.8 ± 14.8%	19 ± 28 1 ± 1.4%	14 ± 24 0.7 ± 1.3%	884 ± 329 46.1 ± 17.2%	383 ± 308 20 ± 16%
	110A. 3 x 8	719 ± 434 37.5 ± 22.6%	413 ± 372 21.5 ± 19.4%	17 ± 25 0.9 ± 1.3%	10 ± 14 0.5 ± 0.7%	1014 ± 288 52.9 ± 15%	415 ± 280 21.6 ± 14.6%

* = $P < 0.05$, ** = $P < 0.001$, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

VI.III.5 – Key NIRS results

The following figures present NIRS results: % HHb, % O_2Hb and TSI %, during the work and recovery intervals of the 6 x 4-min and 3 x 8-min HIIT sessions.

Recovery intensity had no effect on the mean % HHb levels at the end of the work intervals, during the 6 x 4-min and 3 x 8-min HIIT sessions ($P > 0.05$). There was a significant effect of HIIT protocol (main effect $P = 0.032$), with a higher mean % HHb at the end of the 80A 3 x 8-min HIIT session, when compared to the 80A 6 x 4-min HIIT session ($P < 0.05$). No significant differences were found between 6 x 4-min and 3 x 8-min HIIT protocols for the PA and 110A recovery conditions ($P > 0.05$; *Figures 6.14a & 6.14b*).

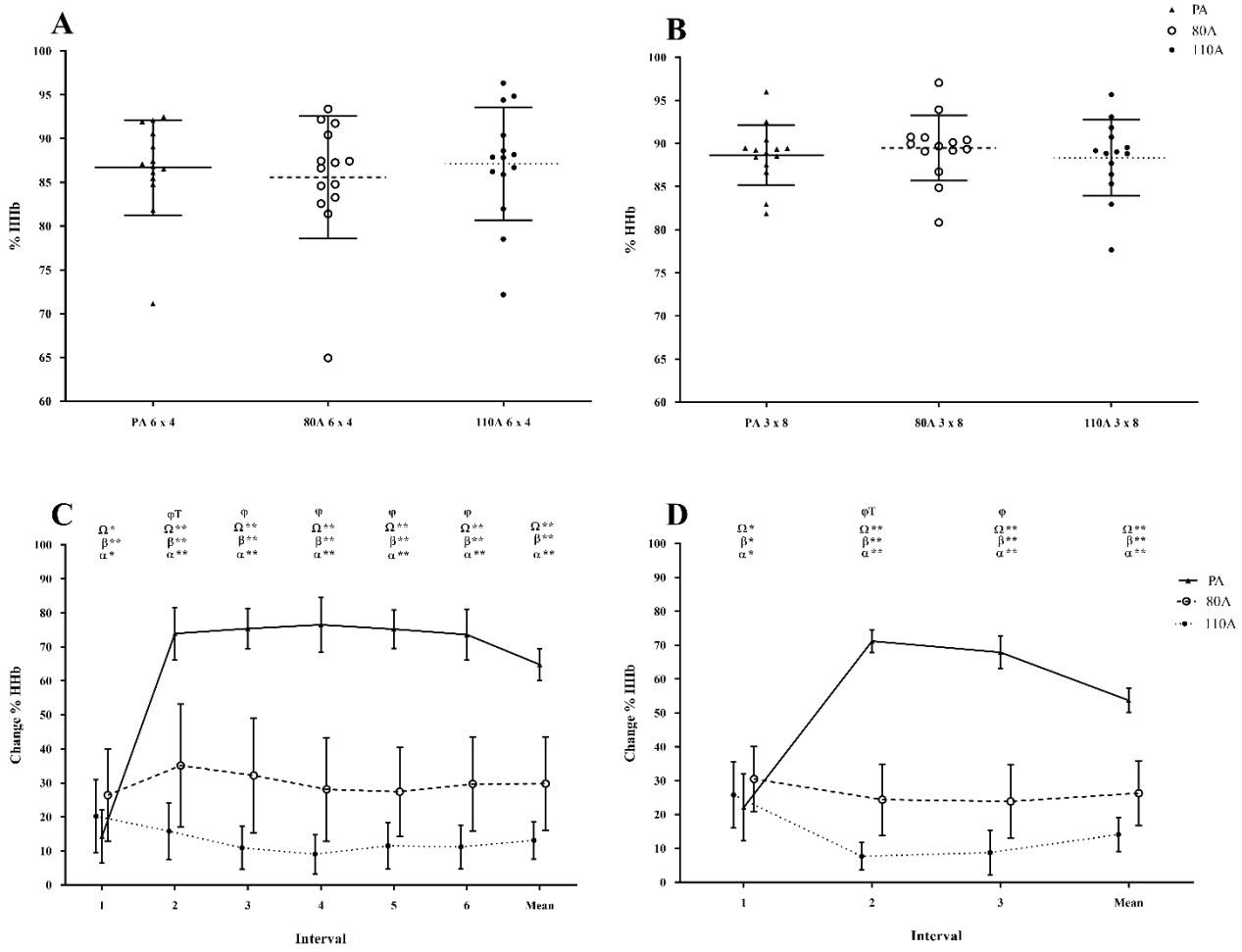


Figure 6.14 – A = Mean % HHb at the end of the work intervals during the 6 x 4-min HIIT sessions, **B** = Mean % HHb at the end of the work intervals during the 3 x 8-min HIIT sessions, **C** = Change in % HHb during the work intervals throughout the 6 x 4-min HIIT sessions, **D** = Change in % HHb during the work intervals throughout the 3 x 8-min HIIT sessions (Mean ± SD). φ = Significant difference from interval 1, T = Significant difference from previous interval, *** = *P* < 0.05, ** = *P* < 0.001**, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

There was a significant interaction between recovery intensity and work interval during the 6 x 4-min (main effect $F = 163.3$, $P < 0.001$; *Figure 6.14c*) and 3 x 8-min (main effect $F = 171.3$, $P < 0.001$; *Figure 6.14d*) HIIT protocols, demonstrating that the pattern of change in % HHb across work intervals was different between recovery conditions.

There was a significant effect of recovery intensity on the change in % HHb during the work intervals of the 6 x 4-min ($P < 0.001$) and 3 x 8-min ($P < 0.001$) HIIT sessions. There was a greater change in % HHb during the PA recovery intensity, compared to the 80A and 110A recovery intensities. There was a greater change in % HHb during the 80A recovery intensity, compared to the 110A recovery intensity (*Figures 6.14c & 6.14d*).

There was an effect of interval during the 6 x 4-min (main effect of interval $F = 33.49$, $P < 0.001$; *Figure 6.14c*) and 3 x 8-min HIIT protocols (main effect of interval $F = 20.948$, $P < 0.001$; *Figure 6.14d*), showing that there was a significant difference in the magnitude of change in % HHb between work intervals, within each HIIT session.

Mean % HHb at the end of the recovery intervals was significantly higher during the 110A recovery condition, when compared to the PA and 80A recovery conditions, during the 6 x 4-min ($P < 0.001$; *Figure 6.15a*) and 3 x 8-min ($P < 0.001$; *Figure 6.15b*) HIIT sessions. Mean % HHb at the end of the recovery intervals was significant higher during the 80A recovery condition, when compared to the PA recovery condition during the 6 x 4-min and 3 x 8-min HIIT sessions ($P < 0.001$; *Figures 6.15a & 6.15b*).

The 6 x 4-min HIIT protocols resulted in significantly lower mean % HHb at the end of the recovery intervals, when compared to the 3 x 8-min HIIT protocols (main effect $P = 0.001$). *Post hoc* pairwise comparisons revealed that mean % HHb at the end of the recovery intervals of the 6 x 4-min HIIT sessions was significantly lower at all three recovery intensities, when compared to the 3 x 8-min HIIT sessions ($P < 0.001$; *Figures 6.15a & 6.15b*).

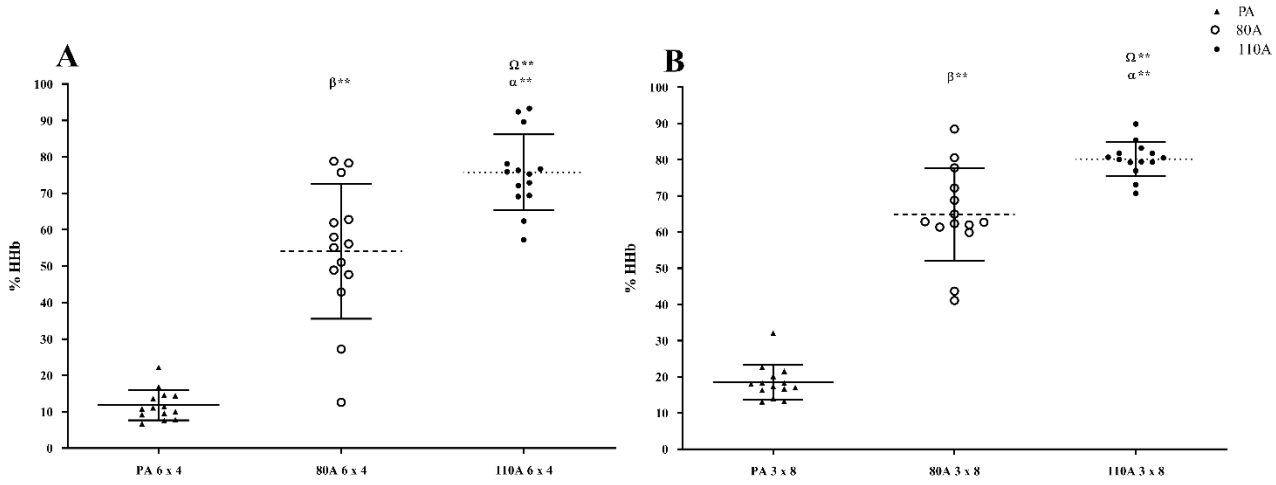


Figure 6.15 – A = Mean % HHb at the end of the recovery intervals during the 6 x 4-min HIIT sessions, **B** = Mean % HHb at the end of the recovery intervals during the 3 x 8-min HIIT sessions (Mean ± SD). ****** = ***P* < 0.001**, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

There was a significant interaction between recovery intensity and interval during the 6 x 4-min (main effect $F = 10.048$; $P < 0.001$; *Figure 6.16a*) and 3 x 8-min HIIT protocols (main effect $F = 4.602$; $P = 0.019$; *Figure 6.16b*), demonstrating that the pattern of change in % O_2Hb across the recovery intervals was different between recovery intensities.

% O_2Hb recovered to a greater extent during the 6 x 4-min HIIT protocols, when compared to the 3 x 8-min HIIT protocols (main effect of protocol $F = 14.289$, $P = 0.002$; *Figures 6.16a & 6.16b*)

There was a significant effect of recovery intensity (main effect of intensity $P < 0.001$), with % O_2Hb recovering to a greater extent during the PA recovery intensity, compared to the 80A and 110A recovery intensities during the 6 x 4-min and 3 x 8-min HIIT sessions (*Figures 6.16a & 6.16b*).

There was an effect of interval during the 6 x 4-min HIIT protocols (main effect $F = 8.014$; $P < 0.001$), showing that there was a significant difference in the magnitude of change in % O_2Hb between recovery intervals, throughout each HIIT session (*Figure 6.16a*). There was no effect of interval during the 3 x 8-min HIIT protocols (main effect $F = 1.482$; $P = 0.245$), showing that there was no significant difference in the magnitude of change in % O_2Hb between recovery intervals, throughout each HIIT session (*Figure 6.16b*).

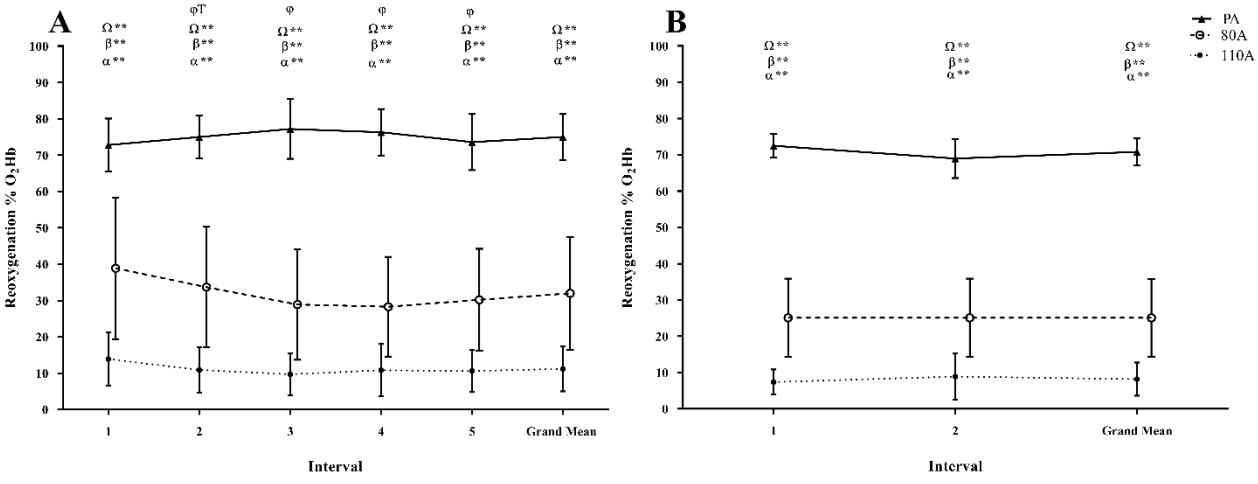


Figure 6.16 – A = % O_2Hb change during the recovery intervals throughout the 6 x 4-min HIIT sessions, **B** = % O_2Hb change during the recovery intervals throughout the 3 x 8-min HIIT sessions (Mean ± SD). φ = Significant difference from interval 1, T = Significant difference from previous interval, **** = *P* < 0.001**, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

Recovery intensity had no effect on the mean TSI % levels at the end of the work intervals, during the 6 x 4-min HIIT protocols ($P > 0.05$; *Figure 6.17a*). Mean TSI % at the end of the work intervals was significantly higher during the PA 3 x 8-min HIIT session, when compared to the 80A and 110A 3 x 8-min HIIT sessions ($P < 0.05$; *Figure 6.17b*).

There was no effect of HIIT protocol ($P = 0.775$), with similar TSI % levels at the end of the work intervals between the 6 x 4-min and 3 x 8-min HIIT protocols across all three recovery conditions (*Figures 6.17a & 6.17b*).

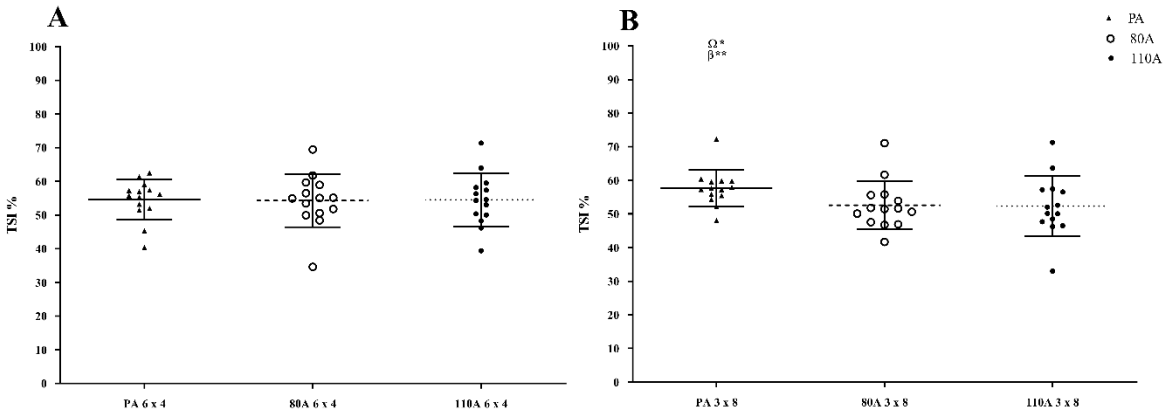


Figure 6.17 – A = Mean TSI % at the end of the work intervals during the 6 x 4-min HIIT sessions, **B** = Mean TSI % at the end of the work intervals during the 3 x 8-min HIIT sessions (Mean ± SD). ***** = $P < 0.05$, ****** = $P < 0.001$**, **Ω** = Significant difference between PA & 110A, β = Significant difference between PA & 80A.

Mean TSI % recovered to a greater extent during the PA recovery intensity, when compared to the 80A and 110A recovery intensities, during the 6 x 4-min ($P < 0.001$; *Figure 6.18a*) and 3 x 8-min HIIT protocols ($P < 0.001$; *Figure 6.18b*). Mean TSI % recovered to a greater extent during the 80A recovery intensity, when compared to the 110A recovery intensity, during the 6 x 4-min ($P < 0.05$; *Figure 6.18a*) and 3 x 8-min HIIT protocols ($P < 0.001$; *Figure 6.18b*).

There was an effect of HIIT protocol (main effect $P = 0.009$), with TSI % recovering to a greater extent during the 110A 3 x 8-min HIIT session, when compared to the 110A 6 x 4-min HIIT session ($P < 0.001$; *Figures 6.18a & 6.18b*).

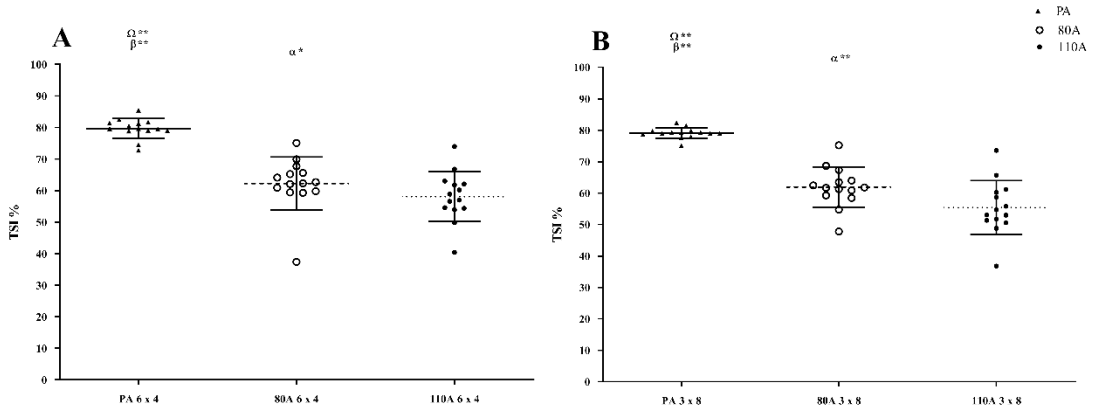


Figure 6.18 – A = Mean TSI % at the end of the recovery intervals during the 6 x 4-min HIIT sessions, **B** = Mean TSI % at the end of the recovery intervals during the 3 x 8-min HIIT sessions (Mean ± SD). *** = *P* < 0.05, ** = *P* < 0.001, Ω** = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

VI.IV – Discussion

VI.IV.1 – Key study findings

The main purpose of this study was to investigate the effects of three different intensity recovery intervals (PA, 80A and 110A) on HIIT session performance. It was hypothesised that increasing recovery interval intensity would reduce the performance of the subsequent work intervals, thereby reducing the acute physiological response of the work intervals and HIIT session.

The key finding of this study was that increasing recovery interval intensity resulted in a significant reduction in work interval PO (*Figures 6.2a & 6.2b*), but did not affect the physiological stress produced during the work intervals as hypothesised (*Figures 6.4a, 6.4b, 6.8a & 6.8b*). However, ACT recovery resulted in a significantly greater physiological stress produced during the recovery intervals (*Figures 6.12a, 6.12b, 6.13a & 6.13b*). By increasing the ACT recovery intensity participants were able to accumulate a greater training load without increasing the total training time commitment of the HIIT session, when compared to PA recovery (*Figures 6.1a & 6.1b*). The current study demonstrates that the role of the recovery interval may go beyond simply providing the adequate recovery between the work intervals (Schoenmakers et al., 2019), to having a direct influence on the training stimulus the HIIT session generates.

The PA recovery condition significantly increased mean work interval PO (*Figures 6.2a & 6.2b*) and the time participants spent above 90 and 95% of MMP, when compared to both ACT recovery conditions (*Table 6.6*). The lower POs measured during ACT recovery HIIT

sessions are likely attributable to greater culminative fatigue, incomplete ATP repletion, inhibition of muscle reoxygenation of myoglobin/haemoglobin and PCr resynthesis. In comparison to the PA recovery which allows for a fuller metabolic recovery of the exercising muscles (Dupont et al., 2004 & 2007; Davis & Green, 2009; Spencer et al., 2006 & 2008; Lopez et al., 2014; Wahl et al., 2013).

Previous research which has focused on the effect of recovery intensity on short (≥ 30-s to < 1-min) repeated work interval performance and has produced conflicting results. Yamagishi & Babrai, (2019) reported recovery intensity to have no effect on mean work interval PO, while Dupont et al., (2007) and Kriel et al., (2016) found mean work interval PO to be significantly higher using PA recovery, compared to ACT recovery. Conversely, Bogdanis et al., (1996), Koizumi et al., (2011) and Spierer et al., (2004) found mean work interval PO to be higher when ACT recoveries were applied and compared to a PA recovery. The heterogeneity in study design (differences in the number and duration of work intervals, differences in ACT recovery intensities and difference in participant fitness levels) will most likely explain the conflicting findings. Current findings and those of the aforementioned studies (Yamagishi & Babraj, 2019; Dupont et al., 2007; Kriel et al., 2016; Bogdanis et al., 1996; Koizumi et al., 2011; Spierer et al., 2004) highlight that the optimal recovery interval intensity is likely dependent on HIIT protocol design and supports the assessment that there is not a one size fits all approach to prescribing HIIT recovery interval intensity.

VI.IV.2 – Acute physiological responses to the HIIT sessions

In the current study, the participants physiological responses to the HIIT sessions were measured. As would be expected, increasing recovery intensity led to a significantly higher mean recovery interval HR (*Figures 6.12a & 6.12b*) and $\dot{V}O_2$ (*Figures 6.13a & 6.13b*). However, there was no effect on mean work interval HR (*Figures 6.4a & 6.4b*) and $\dot{V}O_2$ (*Figures 6.8a & 6.8b*), despite mean work interval PO being significantly lower when recovery intensity was increased (*Figures 6.2a & 6.2b*). The maintenance of elevated HR and $\dot{V}O_2$ during the recovery intervals, combined with unaffected work interval HR and $\dot{V}O_2$, resulted in a significantly higher mean session HR (*Figures 6.3a & 6.3b*) and $\dot{V}O_2$ (*Figures 6.7a & 6.7b*) during the ACT conditions, when compared to PA recovery.

In line with the present findings, Mandroukas et al., (2011) also found recovery intensity to have no effect on 4-min work interval HR and $\dot{V}O_2$ values, despite mean recovery interval HR and $\dot{V}O_2$ being significantly lower during the PA condition in comparison to the ACT condition. In agreement, Barbosa et al., (2016), Coso et al., (2010), Kriel et al., (2016) and Yamagishi & Babraj, (2019) all found increasing recovery intensity to have no effect on work interval $\dot{V}O_2$ and/or HR, but significantly increased the $\dot{V}O_2$ and HR of the recovery intervals. The previously discussed findings demonstrate that through increasing recovery intensity it is possible to increase the accumulated physiological stress of a given HIIT session and therefore potentially induce greater endurance performance adaptations without increasing the overall training time commitment (MacInnis & Gibala, 2017). In support, Yamagishi & Babraj, (2019), found that greater endurance adaptations were achieved over a 2-week training period, when untrained individuals performed HIIT sessions with ACT recovery, compared to PA recovery. It should be noted that the findings of Yamagishi & Babraj,

(2019), are limited due to the untrained status of the participants, where even the smallest increase in training stress during a training intervention could potentially improve endurance performance. However, it is unknown whether the same performance benefit would be seen in trained individuals, who would likely require a greater increase in training stress to improve performance, than simply applying ACT recovery to HIIT sessions during a short training intervention.

The three recovery intensities (PA, 80A and 110A) produced similar times spent at 90 and 95% of $\dot{V}O_{2max}$ during the 6 x 4-min and 3 x 8-min HIIT sessions (*Tables 6.3 & 6.4*). There was only a significant difference found between the PA 3 x 8-min and 80A 3 x 8-min HIIT sessions in time spent above 90% of $\dot{V}O_{2max}$ (*Tables 6.3 & 6.4*). Thevenet et al., (2007), also found the time spent above 90 and 95% of $\dot{V}O_{2max}$ to be similar between ACT and PA recovery conditions. While Dupont & Berthoin, (2004) also found the T@$\dot{V}O_{2max}$ and the time spent above 90% of $\dot{V}O_{2max}$ to be similar between ACT and PA recovery conditions, during 15-s repeated sprints to exhaustion. Additionally, several studies investigating a variety of HIIT protocols (12-min to 30-min of work interval exercise time) have shown that well trained athletes are able to accumulate above 10-min at > 90% of $\dot{V}O_{2max}$ (Buchheit et al., 2012; Millet et al., 2003) and 4-min to 10-min > 95% of $\dot{V}O_{2max}$ during HIIT sessions (Demarie et al., 2000; Millet et al., 2003). These findings are comparable to those of the current study, highlighting the effectiveness of long work interval HIIT as an endurance training stimulus.

Current findings, those of Thevenet et al., (2007) and Dupont & Berthoin, (2004) differ from previous research which has shown faster $\dot{V}O_2$ kinetics during intermittent exercise with ACT

recovery, compared with PA recovery (Dorado et al., 2004). Research to the contrary has observed a slowing of $\dot{V}O_2$ kinetics, specifically phase II $\dot{V}O_2$ kinetics, when high intensity exercise is initiated from a higher metabolic rate (i.e. work intervals preceded by moderate to heavy exercise; Brittain et al., 2001; DiMenna et al., 2008; Hughson & Morrissey, 1982; Hughson & Morrissey, 1983; MacPhee et al., 2005). Consequently, the slowing of $\dot{V}O_2$ kinetics and lower work interval PO limited the time participants spent at 90 and 95% of $\dot{V}O_{2max}$ during the ACT recovery conditions, despite starting the work intervals from an elevated $\dot{V}O_2$.

However, as discussed in study one (V. Experimental Chapter - Study One, section V.IV.2 – Acute physiological response to the HIIT sessions), PA recovery lowers the metabolic rate from which the work intervals commence, resulting in faster $\dot{V}O_2$ kinetics when compared to ACT recovery (Schoenmakers & Reed, 2018; Smilios et al., 2017). Moreover, the magnitude of the $\dot{V}O_2$ response during exercise is largely driven by the external workload, measured as PO. During the PA recovery sessions participants were able to spend significantly longer at 90 and 95% of MMP (*Tables 6.3 & 6.4*) and had significantly higher mean work interval POs (*Figures 6.2a & 6.2b*), when compared to the ACT intensities. The combination of the higher POs and faster $\dot{V}O_2$ kinetics, resulted in participants achieving similar times at 90 and 95% of $\dot{V}O_{2max}$ when compared to the ACT recovery conditions, despite starting the work intervals from a significantly lower $\dot{V}O_2$ during the PA recovery condition. The findings of the current study demonstrate that recovery interval intensity may not be important for achieving high percentages of $\dot{V}O_{2max}$ during HIIT sessions. In accordance with study one current evidence would indicate that $\dot{V}O_2$ kinetics adjust to regulate the O_2 supply that corresponds to the metabolic requirements of the exercise stimulus (Schoenmakers & Reed, 2018). In addition, current findings would also suggest that an ACT recovery may be the best recovery intensity

prescription when compared to PA recovery, as it increases the overall physiological stress of the training session, without compromising the time spent at higher percentages of $\dot{V}O_{2max.}$

A novel finding of the current study is that maximising the PO produced during the work interval may *not* be necessary for achieving a greater accumulation of central physiological stress during a HIIT session. The ACT conditions allowed participants to achieve significantly higher mean session HR (*Figures 6.3a & 6.3b*) and $\dot{V}O_2$ (*Figures 6.7a & 6.7b*), when compared to PA recovery. The greater physiological stress was achieved during the ACT recovery conditions, despite significantly lower work interval POs (*Figures 6.2a & 6.2b*) and significantly shorter durations spent above 90 and 95% of MMP (*Tables 6.3 & 6.4*), in comparison to the PA recovery condition. These findings suggest that the purpose of the recovery interval should not simply be to maximise the performance PO (or velocity) of the work interval. But should also be to maintain a level of physiological stress, thereby increasing the overall training stimulus and time efficiency of the HIIT session. Moreover, as shown in study one (V. Experimental Chapter – Study One), current findings indicate that commencing the work intervals with the exercising muscles in a heightened metabolic state maybe beneficial to HIIT performance, and reduces the PO required to achieve the desired physiological stress.

Low intensity ACT recovery between work intervals has been shown to be more effective in the removal of B[La] than PA recovery (Bogdanis et al., 1996; Coso et al., 2010; Siegler et al., 2006; Mandroukas et al., 2011). During the 6 x 4-min HIIT sessions, both ACT recovery conditions (80A and 110A) resulted in significantly lower B[La] values when compared to the PA recovery conditions (*Figures 6.5a & 6.6a*). The PA 3 x 8-min HIIT session also

produced higher B[La] values when compared to the ACT conditions, although the differences were not statistically significant (*Figures 6.5b & 6.6b*). However, as the B[La] measurements were taken at the end of the work intervals it is possible that the lower B[La] values were simply due to the lower work interval PO of the ACT conditions. Instead of the ACT recovery intervals facilitating a greater removal of B[La] when compared to the PA condition. Dorado et al., (2004) and McAinch et al., (2004) also found there to be no difference in B[La] achieved at the end of the work intervals between ACT and PA recovery conditions.

Past research has linked B[La] accumulation to muscle fatigue and diminished endurance exercise performance (Cairns, 2006), although there is growing body of evidence showing B[La] does not inhibit exercise performance (Hall et al., 2016). The current study's findings show that the accumulation of B[La] does not affect the performance of subsequent work intervals. Figures 6.6a and 6.6b show a linear increase in B[La] throughout all HIIT sessions, however this is not accompanied by a concurrent decrease in work interval PO, which remains relatively stable throughout all HIIT sessions (*Figures 6.2a & 6.2b*).

NIRS findings

As in study one, NIRS was used throughout all HIIT sessions to investigate any potential effects of recovery interval intensity on peripheral responses (HHb, O_2Hb & TSI %). NIRS has been used to investigate the effects of recovery intensity on muscle deoxygenation during long work interval HIIT (≥ 1-min; Stanley & Buchheit, 2014), short work interval HIIT (≥ 30-s to < 1-min; Dupont et al., 2007; Kriel et al., 2016) and repeated sprint (4-s to < 30-s;

Buchheit et al., 2009; Ohya et al., 2013) HIIT performance. The current study adds to this limited body of literature, providing further insight into the peripheral responses during HIIT.

Recovery interval % HHb was significantly increased as recovery intensity was increased (*Figures 6.15a & 6.15b*). In addition, figures 6.16a and 6.16b show that PA recovery allows for the fullest recovery of O_2Hb, with increasing recovery intensity resulting in a concurrent decrease in the magnitude of O_2Hb recovery (*Figures 6.16a & 6.16b*). In agreement, Buchheit et al., (2009) found ACT recovery resulted in a higher level of muscle deoxygenation at the VL during the recovery intervals, when compared to PA recovery. The increased deoxygenation of the VL muscle (an important locomotor muscle during cycling performance) would potentially impair key recovery processes, such as ATP and PCr resynthesis, and M[La] clearance which require the availability of O_2 (Spencer et al., 2006). Moreover, insufficient O_2 availability (i.e. local hypoxia) has been suggested to affect muscular performance and exaggerate the rate of development of both central and peripheral fatigue (Amann & Calbet, 2008). Therefore, the incomplete recovery provided by the ACT conditions may explain the lower work interval POs, when compared to the PA recovery conditions (*Figures 6.2a & 6.2b*). Buchheit et al., (2009), Kriel et al., (2016) and Ohya et al., (2013) support the findings of the current study by showing the increased deoxygenation of the VL muscle during ACT recovery leads to a reduction in work interval performance.

Recovery intensity had no effect on the % HHb level at the end of the work intervals, during both the 6 x 4-min and 3 x 8-min HIIT sessions, suggesting similar levels of muscle de-oxygenation (*Figures 6.14a & 6.14b*). In support of current findings, Kriel et al., (2016), found higher POs did not necessarily result in the greatest increases in HHb, while the ΔHHb

response remained unchanged across work intervals as mean PO decreased. Kriel et al., (2016), therefore suggested that the level of peripheral/local O_2 consumption may not simply be a demand driven system. However, figures 6.14c and 6.14d provide evidence to the contrary, showing that the higher work interval PO of the PA recovery condition resulted in a greater change in % HHb, when compared to the 80A and 110A recovery conditions. The magnitude of change in % HHb during the work intervals of each recovery condition, effectively offset the differences in % HHb observed at the end of the recovery intervals (*Figures 6.15a & 6.15b*). These results provide an explanation for the similar levels of % HHb at the end of the work intervals during the 6 x 4-min and 3 x 8-min HIIT sessions (*Figures 6.14a & 6.14b*), while demonstrating that magnitude of change in HHb is linked to the demands of the exercise. The acute % HHb response to different recovery intensities is similar to the acute HR and $\dot{V}O_2$ responses discussed earlier. Current findings provide further evidence to suggest that maximising work interval PO may *not* be required to achieve the desired levels of physiological stress during the work intervals, when utilising ACT recovery intensities.

The mean % HHb level at the end of the work intervals were between 85 and 90%, regardless of recovery intensity or HIIT protocol (*Figures 6.14a & 6.14b*), with a small CVs present across participants (3.9 to 8.2%). Study one (V. Experimental Chapter – Study One) presents comparable mean % HHb levels at the end of the work intervals, despite different recovery durations. The level of deoxygenation that can be achieved at the VL muscle has been linked to the individuals training status (Jacobs et al., 2013). Similar mean % HHb levels found in the current study are therefore not surprising, as all participants recruited to the current study were of a similar training status (*Table 6.1*). In addition, with participants attaining close or equal to their $\dot{V}O_{2max}$ at the end of the work intervals (*Tables 9.17 & 9.18*), it would stand to

reason that participants were also reaching the upper limit of O_2 delivery and utilisation at the VL muscle. This would also provide an explanation for the similar % HHb levels found at the end of the work interval across all HIIT protocols (*Figures 6.14a & 6.14b*). Reaching an upper O_2 delivery and utilisation limit at the active muscle site during exercise has been reported previously during HIIT (Kriel et al., 2016) and repeated Wingate testing (Dupont et al., 2007).

Recovery intensity had no effect on the TSI % at the end of the work intervals (*Figures 6.17a & 6.17b*). However, increasing recovery intensity resulted in significantly lower TSI % at the end of the recovery intervals (*Figures 6.18a & 6.18b*). Therefore, it can be inferred that increasing recovery intensity decreases the overall TSI % of the HIIT session. In agreement, Stanley & Buchheit, (2014), found mean TSI % during a 3 x 3-min cycling based HIIT session to be significantly decreased as recovery intensity was increased.

In line with previously discussed findings showing ACT results in a greater accumulation of central physiological stress during a HIIT session, NIRS data provides evidence that ACT recovery also results in a greater accumulation of peripheral stress during a HIIT session (i.e. greater time spent with high levels of muscle de-oxygenation). As discussed in study one (V. Experimental Chapter - Study One, section V.IV.2 – Acute physiological responses to the HIIT sessions), the level of muscle de-oxygenation (i.e. local hypoxia) achieved during exercise may dictate the extent of adaptations in exercising muscle (Fluck, 2006; Prior et al., 2004; Terrados et al., 1990). Therefore, based on the current study's findings the prescription of ACT recovery intervals during HIIT would provide a greater stimulus for driving adaptations at the exercising muscle, compared to PA recovery intervals.

It should be noted that NIRS only provides a limited insight into the oxygenation and hemodynamics of one small portion of exercising muscle, therefore care should be taken when extrapolating the findings beyond the scope of the measurements and study. In addition, there is currently no standard method for analysing and presenting NIRS data, therefore caution is advised when comparing and interpreting NIRS data of different studies.

VI.IV.3 – Acute perceptual response to the HIIT sessions

To the authors knowledge this is the first study to present the perceptual responses to long work interval HIIT (≥ 1-min) with different recovery interval intensities. Buchheit et al., (2008) found that ACT recovery between 4-s sprints significantly increased RPE values, when compared to PA recovery intervals. In contrast, during the current study PA recovery resulted in a higher mean RPE being reported at the of end the work intervals, when compared to the ACT recovery conditions (*Figures 6.9a & 6.9b*). However, mean RPE was only significantly higher during the PA 3 x 8-min HIIT session when compared to the 80A 3 x 8-min HIIT session (*Figure 6.9b*). The higher mean RPE values reported during the PA condition maybe linked to the significantly higher work interval POs achieved during the PA condition (*Figures 6.2a & 6.2b*). The contrasting findings of the current study and Buchheit et al., (2008) demonstrates that the acute perceptual responses to a HIIT session maybe influenced by HIIT protocol design when recovery intensity is manipulated.

In accordance with V. Experimental Chapter - Study One, section V.III.2 – Key perceptual HIIT session results, there was a linear increase in mean work interval RPE throughout all HIIT sessions, with reported RPE values reaching ≥ 18 (Very hard) at the last work interval (*Figures 6.10a & 6.10b*). Reported RPE values were significantly higher after each

subsequent work interval, moreover there was no effect of recovery interval intensity on the increase in work interval RPE (*Figures 6.10a & 6.10b*). The linear increase in RPE occurred alongside increases in work interval HR (*Figures 6.4a & 6.4b*), $\dot{V}O_2$ (*Figures 6.8a & 6.8b*) and B[La] (*Figures 6.6a & 6.6b*). The upward drift in physiological stress throughout the HIIT sessions provides an explanation for the linear increase in RPE, however it is highly likely that biomechanical and psychological processes also effected the participants RPE (Marcora et al., 2009; Ulmer, 1996).

Participants reported significantly higher sRPE values at the end of the 110A recovery condition when compared to the 80A and PA recovery conditions, during both the 6 x 4-min and 3 x 8-min HIIT sessions (*Figures 6.11a & 6.11b*). The higher sRPE of the 110A recovery condition is likely the result of the greater accumulated training load (i.e. higher mean session PO; *Figures 6.1a & 6.1b*) and greater overall physiological stress (i.e. higher mean session HR and $\dot{V}O_2$; *Figures 6.3a, 3.b, 6.7a & 6.7b*), in comparison to the 80A and PA recovery conditions.

VI.IV.5 – Study Limitations

The current study was limited to investigating two ACT recovery intensities, a lower intensity 80% of the participants LT and a higher intensity 110% of the participants LT. With only two ACT recovery intensities having been examined, there is a limit to the conclusions which can be made as to the optimal recovery intensity. However, based on current findings it appears that an intensity between 80 and 110% of LT allows for enough recovery to ensure the work intervals can be completed, while increasing the overall training stress of the HIIT session.

An additional delimitation of the current study is the use of the self-paced 'maximal session effort' intensity prescription. For full discussion regarding this limitation see V. Experimental Chapter - Study One, section V.IV.6 – Study Limitations.

VI.IV.6 – Practical applications

As this study demonstrates, ACT recovery at an intensity between 80 and 110% of the LT results in the greatest overall training stress without compromising the time spent at high percentages of $\dot{V}O_{2max}$, during self-paced long interval HIIT (≥ 1-min). Therefore, if the goal of the HIIT session is to generate the greatest training stimulus, coaches and athletes should consider using ACT recovery.

VI.IV.7 - Conclusion

In conclusion, current findings indicate that ACT recovery does not affect the physiological stress produced during the work intervals or time spent above 90 and 95% of $\dot{V}O_{2max}$, but increases the physiological stress produced during the recovery intervals. As a result, ACT recovery increases the overall accumulation of central and peripheral physiological stress, without increasing the total training time commitment of a specific HIIT session, when compared to PA recovery. Overall, the results suggest that an ACT recovery may be the best intensity prescription during long work interval cycling based HIIT, as it increases the overall physiological stress and time efficiency of the HIIT session, when compared to PA recovery.

VII. General Discussion

VII.I – Thesis overview and main findings

This thesis aimed to investigate the impact of the recovery interval components on the acute physiological and perceptual responses to HIIT during cycling exercise. Two studies were completed to examine the effects of the recovery interval duration and intensity in isolation.

Study one (V. Experiemental Chapter – Study One) sought to investigate whether individualising the duration of the recovery interval based on the participants $m\dot{V}O_2$ recovery duration (i.e. the IND recovery duration) would maximise the performance of the work intervals and the acute physiological response to a HIIT session when compared to a STD recovery duration (2:1 work recovery ratio). The main finding of study one was the IND recovery duration did not improve the performance of the work intervals or the acute physiological response to the HIIT sessions, when compared to the STD recovery duration in well-trained cyclists. With no significant differences found between the IND and STD recovery duration for any of the physiological or performance parameters assessed. This has been the first study to demonstrate that a full recovery of $m\dot{V}O_2$ at the exercising muscle (or a full metabolic recovery) may not be required to maintain work interval performance and to generate the desired acute physiological responses during HIIT.

Study two (VI. Experimental Chapter -Study Two) sought to investigate the acute physiological and perceptual effects of a PA and two ACT recovery intensities during cycling based HIIT using long work intervals (≥ 1-min). The main findings of study two were: 1) As

hypothesised ACT recovery reduced work interval PO but did not affect the physiological stress produced during the work intervals as was initially predicted. However, ACT recovery did increase the physiological stress produced during the recovery intervals. As a result, ACT recovery increased the overall accumulation of central and peripheral physiological stress, without increasing the total training time commitment of the HIIT session, when compared to PA recovery. 2) Recovery intensity did not affect the time spent above 90 and 95% of $\dot{V}O_{2max}$ during HIIT, extending the findings of Thevenet et al., (2007) and Dupont & Berthoin, (2004).

The third and novel main finding of the study indicates that maximising the work interval PO may *not* be necessary for achieving the greatest accumulation of central and peripheral physiological stress during the work intervals and whole HIIT session when using ACT recovery. In support of study one, the findings of study two suggest that commencing the work intervals with the exercising muscles in a heightened metabolic state is beneficial to HIIT performance, and reduces the PO required to achieve the desired physiological stress.

Overall, the current thesis provides greater clarity to current understandings of the acute effects of the recovery interval components on HIIT performance, adding to the diverse body of literature presented in tables 2.1 and 2.2.

VII.II – Optimisation of the recovery interval, key to maximising the performance and time efficiency of HIIT.

The six main components of HIIT are: work interval intensity, work interval duration, number of work intervals, recovery interval intensity, recovery interval duration, and overall session load (Tschakert & Hofmann, 2013). In the past research attention has been directed towards the optimisation of the work interval components as they are fundamental to driving the training stimulus of the HIIT session (Buchheit & Laursen, 2013; Tschakert & Hofmann, 2013). While there was no debate as to the importance of the recovery interval components as they are intrinsic to HIIT programme design (Buchheit & Laursen, 2013; Tschakert & Hofmann, 2013), there remained the question of how important the 'optimisation' of the recovery components was to HIIT protocol design and the overall session outcome. However, the experimental chapters within this thesis have demonstrated that the optimisation of the recovery interval components may be of greater importance to determining the overall training stimulus and time efficiency of the HIIT session, than perhaps first thought.

Schoenmakers et al., (2019), stated in their review that to achieve the required exercise intensity during subsequent work intervals, the recovery intervals must accommodate the return of metabolic homeostasis to the exercising muscles. An imbalance between the demands of the work intervals and the recovery provided, could lead to premature fatigue and consequently a reduction in the number of work intervals performed and failure to complete the training session. As shown previously, incorrect programming of the HIIT components has resulted in training sessions that are too hard to complete (Laursen et al., 2002). Indeed, a full metabolic recovery of the exercising muscles between work intervals will guarantee the maintenance of work interval performance and completion of the HIIT session. Although

allowing for a full recovery is unlikely to be the most time efficient recovery interval prescription.

The author of the current thesis agrees with Schoenmakers et al., (2019), appropriate recovery must be provided between work intervals to ensure the completion of the HIIT session. However, the experimental chapters contained herein, provide evidence that metabolic homeostasis and/or a full recovery of the exercising muscle may *not* be required to maintain work interval performance and generate the greatest training stimulus for the specific HIIT session. Moreover, evidence within this thesis indicates that the role of the recovery interval components goes beyond simply providing adequate recovery between work intervals, to having a direct influence on the training stimulus the HIIT session generates. The research of Schoenmakers and colleagues focused primarily on assessing the acute effect of recovery interval duration on HIIT performance (Schoenmakers et al., 2019; Schoenmakers & Reed, 2018). The current thesis extends upon the work of Schoenmakers and colleagues, by investigating the acute effect of recovery interval intensity, in addition to the acute effect of recovery duration on HIIT performance. By taking into consideration recovery interval duration and intensity, the current thesis presents the reader with a fuller understanding of the effect of the recovery interval components and the importance of optimisation when programming HIIT sessions.

Optimisation of the recovery interval components to the specific HIIT protocol is therefore important when the goal is maximising the performance and time efficiency of training session. In the context of the HIIT protocols used within this thesis (long work intervals ≥ 1-min performed on a self-paced "maximal effort" prescription), the optimal composition of the

recovery components would be the shortest recovery duration at the highest possible recovery intensity. However, the recovery components should allow for the maintenance of work interval performance (i.e. power output or velocity) at a level that does not compromise the acute physiological response. The overall result being the accumulation of the greatest training stress within the shortest training time commitment possible.

The optimal recovery duration and intensity are highly dependent on the design of the HIIT protocol, in addition to the goal of the session. There are infinite possible combinations of HIIT protocol design, therefore the recovery duration and intensity recommended herein, are unlikely to be applicable to other HIIT designs and session goals. While the research can be used to guide HIIT design, coaches and athletes are advised to be cautious when extrapolating the finding beyond the scope of the HIIT protocols used in the studies they are referencing. Without a standardised method for calculating the optimal recovery durations and intensities for specific HIIT protocols, there will remain an element of trial and error to optimising the recovery components to a specific individual.

VII.II – Practical applications of study findings: A guide for coaches and athletes.

The current thesis adds to the available literature coaches and athletes can use to inform the programming of HIIT sessions, specifically long work interval HIIT during cycling exercise (a comprehensive list of the available literature on the acute effects of the recovery interval components can be found in *Tables 2.1 & 2.2*). Unfortunately, from current findings it is not possible for coaches and athletes to determine the optimum recovery interval component composition for specific individuals and HIIT protocols. Nonetheless, there are several practical applications which can be drawn from the findings of the two studies within this thesis.

Firstly, as demonstrated in study one (V. Experimental Chapter – Study One), the 2:1 work recovery ratio appears to sit in a "*sweet spot"* of recovery interval duration. By increasing or decreasing the recovery interval duration within the range of the 2:1 work recovery ratio, this study has found there to be no significant effect on the performance of subsequent work intervals and the acute physiological response to the HIIT session (when using PA recoveries). At present the majority of research indicates the 2:1 work recovery ratio is the most practical method for prescribing recovery duration across a broad range of individuals (Seiler & Hetlelid, 2005; Smilios et al., 2017; Laurent et al., 2014). Until a method for determining the optimal recovery duration for a specific individual is established, coaches and athletes should consider utilising the 2:1 work recovery ratio when programming HIIT sessions. In doing so, they can be reasonably confident they are achieving adequate recovery between work intervals, while maximising the time spent training.

Secondly, based on the findings of study two (VI. Experiemental Chapter – Study Two) coaches and athletes should consider using ACT recovery at an intensity between 80 and 110% of the LT, which current results have shown to produce the greatest overall training stress without compromising the time spent at high percentages of $\dot{V}O_{2max}$, during self-paced long work interval HIIT (≥ 1-min).

Overall, the combination of the 2:1 work recovery ratio and an ACT recovery intensity appears to be best recovery interval prescription when programming the recovery intervals during long work interval HIIT across a broad range of individuals. However, as highlighted throughout the thesis the optimum recovery interval duration and intensity is highly individual and dependent on the design of the HIIT protocol. Therefore, the aforementioned practical recommendations should be treated as a guide by coaches and athletes and not the 'gold standard' for recovery interval prescription.

VII.VI - Future Research

The current thesis has highlighted several directions for future research. Firstly, future research should endeavour to establish a standardised method for measuring and/or modelling the optimum recovery duration and intensity for a specific individual and HIIT protocol design. Through establishing the optimum recovery interval component composition required for a specific individual and HIIT protocol design, we would be a step closer to fully maximising the performance and time efficiency of HIIT.

Secondly, following on from study one future research looking to further investigate $m\dot{V}O_2$ recovery duration as a way of individualising HIIT recovery interval duration should: control for exercise intensity and assess the reliability and reproducibility of NIRS measurements of $m\dot{V}O_2$ recovery duration of the VL muscle after a single and multiple bouts of intense cycling exercise.

Finally, theoretically there must be a point at which there is no further increase in the training stimulus that can be produced in a single training session (i.e. a training stimulus threshold). Similar hypothesis of diminishing returns with increased duration of HIIT, have been proposed previously (see review, MacInnis & Gibala, 2017). The ‘gold standard’ or ‘optimum’ HIIT session would therefore be the shortest session required to reach the training stimulus threshold for the specific individual. Achieving this currently theoretical ‘gold standard’ HIIT session, would undoubtedly require the individualisation and optimisation of all HIIT components and in the current authors opinion should be the overall goal of future HIIT research.

VII.V – Thesis Conclusion

The current thesis has shown that the full recovery of $m\dot{V}O_2$ and a return of the exercising muscle to metabolic homeostasis may *not* be required to maintain work interval performance and to generate the desired acute physiological responses during HIIT. Moreover, evidence within this thesis reinforces the importance of the optimisation of the recovery interval components to the specific individual and HIIT protocol, when seeking to maximising the training stimulus and time efficiency of the training session. While the current thesis is unable to determine the optimum recovery interval component composition for specific individuals and HIIT protocols, it would appear that the combination of the 2:1 work recovery ratio and an ACT recovery intensity (between 80 and 110% of LT) may be the best recovery interval prescription when programming the recovery intervals during long work interval HIIT across a broad range of individuals. Future research should endeavour to establish a standardised method for measuring and/or modelling the optimum recovery duration and intensity for a specific individual and HIIT protocol design.

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IX. Appendix

IX.I – Additional results from study one

The following section contains additional results from V. Experimental Chapter – Study One.

Table 9.1 – Full 6 x 4-min HIIT session time at % of MMP and HRmax results.

		Time at % MMP Mean ± SD (CV%)					Time at % HRmax Mean ± SD (CV%)				
		60	**70**	**80**	**90**	**95**	**60**	**70**	**80**	**90**	**95**
Total Work (s)	**STD.**	1430 ± 16 (1)	1337 ± 105 (8)	790 ± 380 (48)	96 ± 75 (78)	48 ± 45 (93)	1436 ± 7 (1)	1379 ± 27 (2)	1248 ± 67 (5)	869 ± 280 (32)	470 ± 271 (58)
	IND.	1432 ± 13 (1)	1328 ± 164 (12)	880 ± 458 (52)	125 ± 106 (85)	40 ± 37 (92)	1433 ± 8 (1)	1376 ± 35 (3)	1244 ± 82 (7)	841 ± 282 (34)	402 ± 282 (70)
Total Work (%)	**STD.**	99.2 ± 1.1 (1.1)	92.9 ± 7.2 (7.8)	54.9 ± 26.4 (48.1)	6.7 ± 5.2 (77.6)	3.3 ± 3 (91.2)	99.7 ± 0.5 (0.5)	95.8 ± 1.9 (1.9)	86.6 ± 4.6 (5.3)	60.4 ± 19.4 (32.2)	32.6 ± 18.8 (57.7)
	IND.	99.5 ± 0.9 (0.9)	92.2 ± 11.4 (12.4)	61.1 ± 31.8 (52.1)	8.7 ± 7.4 (85.2)	2.8 ± 2.6 (91.5)	99.5 ± 0.6 (0.6)	95.6 ± 2.4 (2.5)	86.4 ± 5.7 (6.6)	58.4 ± 19.6 (33.6)	27.9 ± 19.6 (70.1)
Total Session (s)	**STD.**	1430 ± 16 (1)	1337 ± 105 (8)	790 ± 380 (48)	96 ± 75 (78)	48 ± 45 (93)	1979 ± 82 (4)	1792 ± 114 (6)	1405 ± 375 (27)	986 ± 317 (32)	527 ± 302 (57)
	IND.	1432 ± 13 (1)	1328 ± 164 (12)	880 ± 458 (52)	125 ± 106 (85)	40 ± 37 (92)	2154 ± 334 (16)	1795 ± 141 (8)	1515 ± 146 (10)	983 ± 346 (35)	475 ± 335 (71)
Total Session (%)	**STD.**	70.1 ± 0.7 (1) *	65.5 ± 5.2 (8) **	38.7 ± 18.6 (48.1)	4.7 ± 3.6 (77.3)	2.4 ± 2.3 (94.1)	97 ± 4.1 (4.2) **	87.9 ± 5.6 (6.4) *	73.3 ± 5.1 (7) *	48.4 ± 15.5 (32.1) *	25.7 ± 14.7 (57.2)
	IND.	59.6 ± 9.8 (16.5)	54.8 ± 9.2 (16.8)	35.3 ± 18 (50.9)	4.9 ± 3.6 (73.9)	1.6 ± 1.4 (84.3)	88.2 ± 10.3 (11.7)	74.5 ± 13 (17.4)	62.8 ± 11.4 (18.1)	40.2 ± 14.7 (36.6)	19 ± 13.6 (71.6)

* = $P < 0.05$, ** = $P < 0.001$.

Table 9.2 – Full 3 x 8-min HIIT session time at % of MMP and HRmax results.

		Time at % MMP Mean ± SD (CV%)					Time at % HRmax Mean ± SD (CV%)				
		60	**70**	**80**	**90**	**95**	**60**	**70**	**80**	**90**	**95**
Total Work (s)	**STD.**	1424 ± 20 (1)	1181 ± 228 (19)	489 ± 317 (65)	46 ± 34 (74)	24 ± 26 (108)	1430 ± 13 (1)	1391 ± 26 (2)	1301 ± 67 (5)	943 ± 325 (34)	550 ± 301 (55)
	IND.	1424 ± 22 (2)	1278 ± 177 (14)	563 ± 313 (56)	50 ± 22 (45)	23 ± 18 (79)	1433 ± 9 (1)	1392 ± 24 (2)	1295 ± 66 (5)	875 ± 333 (38)	437 ± 286 (66)
Total Work (%)	**STD.**	98.9 ± 1.4 (1.4)	82 ± 15.9 (19.4)	33.9 ± 22.1 (65)	3.3 ± 2.4 (74.9)	1.7 ± 1.8 (108.1)	99.3 ± 0.9 (0.9)	96.6 ± 1.8 (1.9)	90.4 ± 4.7 (5.2)	65.5 ± 22.6 (34.5)	38.2 ± 20.9 (54.6) *
	IND.	98.9 ± 1.6 (1.6)	88.8 ± 12.3 (13.8) *	39.1 ± 21.7 (55.5)	3.5 ± 1.6 (45.4)	1.6 ± 1.3 (78.8)	99.5 ± 0.6 (0.6)	96.7 ± 1.7 (1.7)	90 ± 4.6 (5.1)	60.6 ± 23 (37.9)	29.5 ± 18.5 (62.8)
Total Session (s)	**STD.**	1424 ± 20 (1)	1181 ± 228 (19)	489 ± 317 (65)	46 ± 34 (74)	24 ± 26 (108)	1789 ± 107 (6)	1581 ± 82 (5)	1416 ± 84 (6)	1002 ± 342 (34)	580 ± 312 (54) *
	IND.	1424 ± 22 (2)	1278 ± 177 (14)	563 ± 313 (56)	50 ± 22 (45)	23 ± 18 (79)	1729 ± 145 (8)	1561 ± 76 (5)	1403 ± 87 (6)	926 ± 350 (38)	460 ± 303 (66)
Total Session (%)	**STD.**	74.2 ± 1.1 (1.5)	61.5 ± 11.9 (19.3)	25.4 ± 16.5 (64.9)	2.4 ± 1.7 (72.6)	1.2 ± 1.3 (103.3)	93.2 ± 5.5 (5.9)	82.4 ± 4.3 (5.3)	73.7 ± 4.3 (5.9)	52.2 ± 17.8 (34.1)	30.3 ± 16.3 (53.8)
	IND.	77.9 ± 5.7 (7.3) *	69.6 ± 9.3 (13.4) *	30.2 ± 15.8 (52.4)	2.7 ± 1.3 (47.3)	1.3 ± 1 (78.6)	94.1 ± 6.5 (7)	85.2 ± 7.5 (8.8)	76.5 ± 6.4 (8.4)	50.2 ± 19.2 (38.2)	25 ± 16.8 (67.2)

* = $P < 0.05$.

Table 9.3 – Full 6 x 4-min HIIT session time at % of $\dot{V}O_{2max}$, $\dot{V}_E$ max and Bfmax results.

		Time at % $\dot{V}O_{2max}$ Mean ± SD (CV%)					**Time at % $\dot{V}_E$ max Mean ± SD (CV%)**					**Time at % Bfmax Mean ± SD (CV%)**				
		60	**70**	**80**	**90**	**95**	**60**	**70**	**80**	**90**	**95**	**60**	**70**	**80**	**90**	**95**
Total Work (s)	**STD.**	1356 ± 38 (3)	1304 ± 55 (4)	1178 ± 139 (12)	821 ± 311 (38)	502 ± 332 (66)	1210 ± 189 (16)	1009 ± 318 (32)	769 ± 333 (43)	411 ± 283 (69)	201 ± 200 (100)	1318 ± 270 (20)	1145 ± 349 (30)	906 ± 365 (40)*	530 ± 381 (72)	368 ± 331 (90)
	IND.	1348 ± 46 (3)	1294 ± 60 (5)	1156 ± 153 (13)	749 ± 364 (49)	451 ± 390 (86)	1192 ± 145 (12)	955 ± 320 (34)	709 ± 362 (51)	375 ± 287 (76)	188 ± 193 (103)	1329 ± 174 (13)	1096 ± 331 (30)	798 ± 393 (49)	463 ± 345 (74)	303 ± 308 (102)
Total Work (%)	**STD.**	94.1 ± 2.6 (2.8)	90.6 ± 3.8 (4.2)	81.8 ± 9.6 (11.7)	57.1 ± 21.6 (37.8)	34.8 ± 23.1 (66.2)	84 ± 13.2 (15.7)	70.1 ± 22.1 (31.5)	53.4 ± 23.1 (43.2)	28.6 ± 19.6 (68.8)	14 ± 13.9 (99.4)	91.5 ± 18.7 (20.5)	79.5 ± 24.2 (30.5)	62.9 ± 25.3 (40.2)*	36.8 ± 26.4 (71.7)	25.6 ± 22.9 (89.5)
	IND.	93.6 ± 3.2 (3.4)	89.9 ± 4.2 (4.6)	80.3 ± 10.6 (13.2)	52 ± 25.3 (48.6)	31.3 ± 27.1 (86.5)	82.8 ± 10.1 (12.2)	66.3 ± 22.2 (33.5)	49.3 ± 25.2 (51.1)	26 ± 19.9 (76.4)	13.1 ± 13.4 (102.5)	92.3 ± 12.1 (13.1)	76.1 ± 23 (30.2)	55.4 ± 27.3 (49.3)	32.2 ± 23.9 (74.4)	21.1 ± 21.4 (101.5)
Total Session (s)	**STD.**	1495 ± 36 (2)	1403 ± 63 (4)	1234 ± 154 (12)	842 ± 325 (39)	516 ± 350 (68)	1366 ± 255 (19)	1107 ± 360 (33)	813 ± 359 (44)	428 ± 298 (70)	209 ± 208 (99)	1693 ± 405 (24)	1401 ± 466 (33)	1044 ± 448 (43)	588 ± 425 (72)	399 ± 350 (88)
	IND.	1483 ± 36 (2)	1387 ± 61 (4)	1208 ± 154 (13)	763 ± 374 (49)	459 ± 399 (87)	1360 ± 205 (15)	1053 ± 360 (34)	757 ± 393 (52)	393 ± 308 (78)	199 ± 208 (105)	1779 ± 341 (19)	1358 ± 434 (32)	941 ± 470 (50)	528 ± 389 (74)	347 ± 334 (96)
Total Session (%)	**STD.**	73.3 ± 1.8 (2.4)*	68.8 ± 3.1 (4.5)**	60.5 ± 7.6 (12.5)**	41.3 ± 15.9 (38.6)*	25.3 ± 17.2 (68)	67 ± 12.5 (18.7)*	54.3 ± 17.7 (32.6)*	39.9 ± 17.6 (44.1)*	21 ± 14.6 (69.8)	10.2 ± 10.2 (99.8)	83 ± 19.9 (23.9)*	68.7 ± 22.9 (33.3)*	51.2 ± 21.9 (42.8)*	28.8 ± 20.9 (72.5)*	19.6 ± 17.2 (87.7)*
	IND.	61.7 ± 10.4 (16.8)	57.6 ± 9 (15.7)	50 ± 9.2 (18.4)	30.8 ± 14 (45.5)	17.7 ± 14.9 (84.2)	56.6 ± 13.1 (23.1)	43.7 ± 17 (38.9)	31.2 ± 16.8 (53.9)	15.9 ± 12.7 (80)	7.9 ± 8.8 (111.1)	73.8 ± 16.6 (22.5)	56.4 ± 19.3 (34.3)	39 ± 20.9 (53.5)	22 ± 16.8 (76.5)	14.4 ± 14.3 (99.3)

* = $P < 0.05$, ** = $P < 0.001$.

Table 9.4 - Full 3 x 8-min HIIT session time at % of $\dot{V}O_{2max,}$ $\dot{V}_E$ max and Bfmax results.

		Time at % $\dot{V}O_{2max}$ Mean ± SD (CV%)					Time at % $\dot{V}_E$ max Mean ± SD (CV%)					Time at % Bfmax Mean ± SD (CV%)				
		60	**70**	**80**	**90**	**95**	**60**	**70**	**80**	**90**	**95**	**60**	**70**	**80**	**90**	**95**
Total Work (s)	**STD.**	1383 ± 22 (2)	1336 ± 47 (3)	1202 ±126 (11)	753 ± 396 (53)	398 ± 330 (83)	1198 ± 209 (17)	991 ± 327 (33)	701 ± 419 (60)	356 ± 343 (96)*****	180 ± 242 (135)	1288 ± 222 (17)	1074 ± 349 (32)	778 ± 380 (49)	455 ± 354 (78)	312 ± 316 (101)
	IND.	1388 ± 23 (2)	1341 ± 39 (3)	1176 ± 176 (15)	649 ± 345 (53)	278 ± 258 (93)	1196 ± 182 (15)	956 ± 314 (33)	602 ± 330 (55)	220 ± 244 (111)	108 ± 182 (170)	1283 ± 208 (16)	1041 ± 378 (36)	748 ± 418 (56)	426 ± 381 (89)	323 ± 353 (110)
Total Work (%)	**STD.**	96 ± 1.5 (1.6)	92.8 ± 3.3 (3.5)	83.4 ± 8.8 (10.6)	52.4 ± 27.5 (52.6)	27.6 ± 22.9 (83)	83.1 ± 14.5 (17.4)	68.8 ± 22.8 (33.1)	48.6 ± 29.1 (59.8)	24.7 ± 23.8 (96.5)*****	12.5 ± 16.8 (134.5)	89.5 ± 15.4 (17.3)	74.6 ± 24.2 (32.5)	54 ± 26.4 (48.8)	31.6 ± 24.6 (77.9)	21.7 ± 22 (101.2)
	IND.	96.3 ± 1.6 (1.7)	93.1 ± 2.7 (2.9)	81.5 ± 12 (14.7)	45.4 ± 24.4 (53.8)	20 ± 19 (95)	83 ± 12.7 (15.3)	66.4 ± 21.8 (32.9)	41.8 ± 22.9 (54.9)	15.3 ± 17 (111)	7.5 ± 12.7 (169.4)	89.1 ± 14.5 (16.2)	72.3 ± 26.2 (36.3)	52 ± 29 (55.9)	29.6 ± 26.4 (89.4)	22.4 ± 24.5 (109.5)
Total Session (s)	**STD.**	1448 ± 24 (2)	1387 ± 50 (4)	1234 ± 128 (10)	769 ± 402 (52)	407 ± 334 (82)	1278 ± 227 (18)	1042 ± 343 (33)	734 ± 432 (59)	368 ± 354 (96)*****	184 ± 254 (138)	1539 ± 315 (20)	1229 ± 405 (33)	851 ± 411 (48)	489 ± 373 (76)	337 ± 329 (97)
	IND.	1446 ± 20 (1)	1384 ± 39 (3)	1196 ± 178 (15)	654 ± 348 (53)	279 ± 260 (93)	1263 ± 190 (15)	993 ± 319 (32)	618 ± 341 (55)	224 ± 248 (110)	110 ± 185 (168)	1455 ± 252 (17)	1149 ± 406 (35)	803 ± 442 (55)	448 ± 395 (88)	336 ± 361 (108)
Total Session (%)	**STD.**	75.4 ± 1.3 (1.7)	72.3 ± 2.6 (3.6)	64.3 ± 6.6 (10.3)	40.1 ± 20.9 (52.2)	21.2 ± 17.4 (82.1)	66.5 ± 11.8 (17.7)	54.3 ± 17.9 (32.9)	38.9 ± 23 (59.2)	19.2 ± 18.4 (95.7)*****	9.6 ± 13.2 (137.3)	80.2 ± 16.4 (20.5)	64 ± 21.1 (33)	44.3 ± 21.4 (48.3)	25.5 ± 19.4 (76.3)	17.6 ± 17.1 (97.3)
	IND.	79.1 ± 6.4 (8)*****	75.7 ± 6.2 (8.1)	65.3 ± 10.3 (15.8)	35.3 ± 18.4 (52)	14.8 ± 13.2 (89.2)	69.1 ± 11.8 (67.9)	54.4 ± 18 (33.2)	33.8 ± 18.9 (56)	12.3 ± 13.5 (110)	6.1 ± 10.1 (167)	80.2 ± 17.2 (21.4)	63.6 ± 24 (37.8)	44.8 ± 25.8 (57.6)	25.4 ± 23 (90.6)	19.1 ± 20.9 (109.4)

* = $P < 0.05$.

Table 9.5 - Additional work interval PO and HR results from the 6 x 4-min HIIT sessions.

		Work Interval PO and HR results Mean ± SD (CV%)						
	Int. No.	**1**	**2**	**3**	**4**	**5**	**6**	**Mean**
PO as % MMP	**STD.**	83.6 ± 5.5 (6.6)	81 ± 3.9 (4.9)	79.8 ± 4 (4.9)	78.4 ± 3.5 (4.5)	77.4 ± 3.6 (4.6)	81.1 ± 4.1 (5.1)	80.2 ± 3.1 (3.9)
	IND.	82.5 ± 6.3 (7.6)	82.5 ± 5.5 (6.6)	81.3 ± 5.3 (6.5)	79.6 ± 5.7 (7.2)	79.2 ± 6 (7.6)	81.7 ± 4.3 (5.3)	81.1 ± 5 (6.1)
Relative PO ($W.kg^{-1}$)	**STD.**	4.3 ± 0.7 (15.2)	4.2 ± 0.7 (15.7)	4.1 ± 0.7 (16)	4.1 ± 0.7 (16)	4 ± 0.6 (16)	4.2 ± 0.7 (15.9)	4.2 ± 0.6 (15.4)
	IND.	4.3 ± 0.7 (16)	4.3 ± 0.7 (15.8)	4.2 ± 0.7 (16.5)	4.1 ± 0.7 (16.6)	4.1 ± 0.7 (15.9)	4.2 ± 0.7 (16.3)	4.2 ± 0.7 (15.9)
HR as % Max	**STD.**	84.2 ± 3.6 (4.3)	87.9 ± 2.4 (2.7)	89.6 ± 2.6 (2.9)	90.3 ± 2.7 (3)	90.7 ± 2.8 (3)	92.6 ± 3.3 (3.5)	89.2 ± 2.4 (2.7)
	IND.	84.8 ± 3.5 (4.1)	88.1 ± 3.2 (3.6)	89.6 ± 3.4 (3.8)	90.2 ± 3.5 (3.8)	90.7 ± 4.1 (4.5)	91.8 ± 3.7 (4)	89.2 ± 3.2 (3.6)

Table 9.6 - Additional work interval PO and HR results from the 3 x 8-min HIIT sessions

		Work Interval PO and HR results Mean ± SD (CV%)			
	Int. No.	**1**	**2**	**3**	**Mean**
PO as % MMP	**STD.**	78.3 ± 3.8 (4.8)	76 ± 4 (5.2)	75.9 ± 4.1 (5.4)	76.7 ± 3.5 (4.5)
	IND.	78.6 ± 3.2 (4.1)	77.6 ± 3.7 (4.7)	77.6 ± 4.3 (5.5)	77.9 ± 3 (3.9)
Relative PO ($W.kg^{-1}$)	**STD.**	4.1 ± 0.6 (15.2)	3.9 ± 0.7 (16.5)	3.9 ± 0.6 (16.5)	4 ± 0.6 (15.9)
	IND.	4.1 ± 0.5 (12.8)	4 ± 0.6 (15)	4 ± 0.7 (16.7)	4 ± 0.6 (14.6)
HR as % Max	**STD.**	88.2 ± 3.7 (4.2)	91.4 ± 3.3 (3.6)	92.8 ± 3.2 (3.4)	90.8 ± 3.1 (3.5)
	IND.	86.8 ± 3.7 (4.2)	90.7 ± 3.2 (3.5)	92.4 ± 3.6 (3.9)	89.9 ± 3.1 (3.5)

Table 9.7 – Additional work interval $\dot{V}O_2$ results from the 6 x 4-min HIIT sessions.

		$\dot{V}O_2$ (L.min^{-1}) Mean ± SD (CV%)					
	Int. No.	**1**	**2**	**3**	**4**	**5**	**6**
Int. Mean	**STD.**	3.56 ± 0.5 (15.4)	3.75 ± 0.6 (16.3)	3.78 ± 0.6 (17.2)	3.85 ± 0.6 (16.6)	3.82 ± 0.7 (17.5)	3.89 ± 0.6 (16.2)
	IND.	3.54 ± 0.6 (17)	3.73 ± 0.7 (17.7)	3.77 ± 0.7 (17.4)	3.75 ± 0.6 (16.9)	3.75 ± 0.6 (16.8)	3.79 ± 0.6 (16.7)
Int. Mean % of max	**STD.**	82.8 ± 5.8 (7)	86.9 ± 6.3 (7.3)	87.6 ± 6.4 (7.3)	89.2 ± 5.3 (5.9)	88.3 ± 5.3 (6)	90.2 ± 5.9 (6.5)
	IND.	82 ± 5.8 (7.1)	86.4 ± 6.2 (7.2)	87.3 ± 6.1 (6.9)	86.8 ± 5.7 (6.5)	87 ± 6.4 (7.3)	87.8 ± 5.9 (6.7)
Int. Max (30s)	**STD.**	3.98 ± 0.6 (14.9)	4.16 ± 0.6 (15.3)	4.18 ± 0.7 (16)	4.28 ± 0.6 (23.6)	4.24 ± 0.7 (15.9)	4.34 ± 0.7 (15.5)
	IND.	4.01 ± 0.6 (16)	4.18 ± 0.7 (16.8)	4.21 ± 0.7 (16.8)	4.19 ± 0.7 (16.1)	4.18 ± 0.7 (16.7)	4.26 ± 0.7 (15.6)
Int. Max % of max	**STD.**	92.5 ± 6.5 (7.1)	96.6 ± 6.9 (7.2)	97 ± 6.2 (6.3)	99.3 ± 5 (5.1)	98.3 ± 5.4 (5.5)	100.8 ± 6.4 (6.4)
	IND.	93.1 ± 6.3 (6.8)	97 ± 6.9 (7.1)	97.6 ± 6.8 (7)	97.2 ± 6.7 (6.9)	97 ± 7.2 (7.5)	99 ± 6.6 (6.6)

Table 9.8 – Additional work interval $\dot{V}O_2$ results from the 3 x 8-min HIIT sessions.

		$\dot{V}O_2$ (L.min^{-1}) Mean ± SD (CV%)		
	Int. No.	**1**	**2**	**3**
Int. Mean	**STD.**	3.67 ± 0.6 (16.3)	3.81 ± 0.6 (16.3)	3.86 ± 0.6 (16.2)
	IND.	3.63 ± 0.6 (15.4)	3.77 ± 0.6 (16.7)	3.8 ± 0.7 (17.4)
Int. Mean % of max	**STD.**	85.1 ± 5.3 (6.2)	88.3 ± 4.8 (5.4)	89.4 ± 4.8 (5.4)
	IND.	84.2 ± 5.1 (6)	87.2 ± 4.9 (5.7)	87.9 ± 4 (4.5)
Int. Max (30s)	**STD.**	4.14 ± 0.6 (14.9)	4.22 ± 0.6 (15.10)	4.27 ± 0.7 (16)
	IND.	4.09 ± 0.6 (13.9)	4.11 ± 0.6 (15.6)	4.22 ± 0.7 (16.4)
Int. Max % of max	**STD.**	96.1 ± 5.2 (5.4)	98 ± 5.8 (6)	99.1 ± 7 (7.1)
	IND.	95.2 ± 6.4 (6.7)	95.4 ± 5.6 (5.8)	97.8 ± 4.1 (4.2)

IX.II – Additional results from study two

The following section contains additional results from VI. Experimental Chapter – Study Two.

Table 9.9 – Full 6 x 4-min HIIT session time at % of MMP results.

		Time at % MMP Mean ± SD (CV%)				
		60	**70**	**80**	**90**	**95**
Total Work (s)	**PA**	1430 ± 17 (1)	1375 ± 93 (7)	940 ± 386 (41) **Ω*β***	89 ± 76 (86) **Ω**β***	52 ± 50 (97) **Ω*β***
	80A	1423 ± 38 (3)	1336 ± 166 (12)	625 ± 506 (81)	19 ± 28 (144)	15 ± 25 (170)
	110A	1440 ± 1 (0)	1417 ± 42 (3) **α***	465 ± 470 (101)	26 ± 32 (123)	15 ± 23 (150)
Total Work (%)	**PA**	99.3 ± 1.2 (1.2)	95.5 ± 6.4 (6.7)	65.3 ± 26.8 (41) **Ω*β***	6.2 ± 5.3 (85) **Ω**β***	3.5 ± 3.4 (96.2) **Ω*β***
	80A	98.8 ± 2.6 (2.7)	92.8 ± 11.5 (12.4)	43.4 ± 35.1 (80.9)	1.3 ± 1.9 (143)	1 ± 1.8 (170.5)
	110A	100 ± 0.1 (0.1)	98.4 ± 2.9 (3) **α***	32.3 ± 32.7 (101.1)	1.8 ± 2.2 (123.2)	1.1 ± 1.6 (149.4)
Total Session (s)	**PA**	1430 ± 17 (1)	1375 ± 93 (7)	940 ± 386 (41) **Ω*β***	89 ± 76 (86) **Ω**β***	52 ± 50 (97) **Ω*β***
	80A	1423 ± 38 (3)	1336 ± 166 (12)	625 ± 506 (81)	19 ± 28 (144)	15 ± 25 (170)
	110A	1825 ± 299 (16) **Ω**α****	1417 ± 42 (3) **α***	465 ± 470 (101)	26 ± 32 (123)	15 ± 23 (150)
Total Session (%)	**PA**	70.1 ± 0.8 (1.1)	67.4 ± 4.6 (6.8)	46.1 ± 18.9 (41) **Ω*β***	4.3 ± 3.7 (85.4) **Ω**β***	2.6 ± 2.5 (97.4) **Ω*β***
	80A	69.8 ± 1.9 (2.7)	65.5 ± 8.1 (12.4)	30.7 ± 24.8 (80.9)	0.9 ± 1.4 (144.7)	0.7 ± 1.2 (170.7)
	110A	89.5 ± 14.6 (16.4) **Ω**α****	69.5 ± 2.1 (3) **α***	22.8 ± 23.1 (101.1)	1.3 ± 1.6 (123.3)	0.7 ± 1.1 (149.9)

* = $P < 0.05$, ** = $P < 0.001$, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

Table 9.10 – Full 3 x 8-min HIIT session time at % of MMP results.

		Time at % MMP Mean ± SD (CV%)				
		60	**70**	**80**	**90**	**95**
Total Work (s)	**PA**	1428 ± 20 (1)	1248 ± 218 (17)	654 ± 372 (57) **Ω**β***	48 ± 39 (82) **Ω*β***	27 ± 29 (106) **Ω***
	80A	1433 ± 12 (1)	1211 ± 297 (25)	362 ± 362 (100)	19 ± 28 (145)	14 ± 24 (169)
	110A	1440 ± 1 (0) **Ω***	1303 ± 285 (22)	209 ± 215 (103)	17 ± 25 (145)	10 ± 14 (140)
Total Work (%)	**PA**	99.1 ± 1.4 (1.4)	86.6 ± 15.2 (17.6)	45.4 ± 25.9 (57.1) **Ω**β***	3.4 ± 2.8 (82.9) **Ω*β***	1.9 ± 2 (105.9) **Ω***
	80A	99.5 ± 0.9 (0.9)	84.1 ± 20.6 (24.5)	25.2 ± 25.1 (99.9)	1.3 ± 1.9 (145.1)	1 ± 1.7 (169.8)
	110A	100 ± 0.1 (0.1) **Ω***	90.5 ± 19.8 (21.9)	14.5 ± 15 (103.3)	1.2 ± 1.7 (146.2)	0.7 ± 1 (140.8)
Total Session (s)	**PA**	1428 ± 20 (1)	1248 ± 218 (17)	654 ± 372 (57) **Ω**β***	48 ± 39 (82) **Ω*β***	27 ± 29 (106) **Ω***
	80A	1433 ± 12 (1)	1211 ± 297 (25)	362 ± 362 (100)	19 ± 28 (145)	14 ± 24 (169)
	110A	1748 ± 239 (14) **Ω**α****	1303 ± 285 (22)	209 ± 215 (103)	17 ± 25 (145)	10 ± 14 (140)
Total Session (%)	**PA**	74.3 ± 1.1 (1.5)	65 ± 11.4 (17.5)	34 ± 19.4 (57.1) **Ω**β***	2.5 ± 2 (80.4) **Ω*β***	1.4 ± 1.4 (102.8) **Ω***
	80A	74.6 ± 0.6 (0.9)	63.1 ± 15.5 (24.5)	18.9 ± 18.9 (99.9)	1 ± 1.4 (144.1)	0.7 ± 1.3 (168.8)
	110A	91.1 ± 12.5 (13.7) **Ω**α****	67.9 ± 14.9 (21.9)	10.9 ± 11.2 (103.3)	0.9 ± 1.3 (144.7)	0.5 ± 0.7 (141.4)

* = $P < 0.05$, ** = $P < 0.001$, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

Table 9.11 – Full 6 x 4-min HIIT session time at % of HRmax results.

		Time at % HRmax Mean ± SD (CV%)				
		60	**70**	**80**	**90**	**95**
Total Work (s)	**PA**	1436 ± 7 (1) **Ω*β***	1388 ± 32 (2)	1265 ± 63 (5)	954 ± 145 (15) **β***	591 ± 221 (37) **Ω*β***
	80A	1428 ± 13 (1)	1403 ± 33 (2) **β***	1272 ± 96 (8)	734 ± 267 (36)	254 ± 251 (99)
	110A	1430 ± 14 (1)	1409 ± 34 (2)	1327 ± 99 (7) **Ω***	902 ± 165 (18) **α***	333 ± 236 (71)
Total Work (%)	**PA**	99.7 ± 0.5 (0.5) **Ω*β***	96.4 ± 2.2 (2.3)	87.8 ± 4.3 (4.9)	66.2 ± 10.1 (15.2) **β***	41 ± 15.3 (37.4) **Ω*β***
	80A	99.2 ± 0.9 (0.9)	97.4 ± 2.3 (2.4) **β***	88.3 ± 6.6 (7.5)	50.9 ± 18.5 (36.4)	17.6 ± 17.4 (99)
	110A	99.3 ± 1 (1)	97.8 ± 2.4 (2.4)	92.1 ± 6.9 (7.5) **Ω***	62.7 ± 11.4 (18.3) **α***	23.1 ± 16.4 (70.9)
Total Session (s)	**PA**	2006 ± 36 (2)	1812 ± 91 (5)	1527 ± 94 (6)	1091 ± 154 (14)	678 ± 248 (37) **Ω*β***
	80A	2026 ± 18 (1) **β***	1973 ± 93 (5) **β****	1677 ± 222 (13) **β***	883 ± 348 (39)	317 ± 299 (94)
	110A	2028 ± 20 (1) **Ω***	2008 ± 36 (2) **Ω****	1875 ± 150 (8) **Ω**α***	1183 ± 240 (20) **Ω* α****	385 ± 281 (73)
Total Session (%)	**PA**	98.4 ± 1.7 (1.8)	88.8 ± 4.4 (5)	74.9 ± 4.7 (6.3)	53.5 ± 7.6 (14.1)	33 ± 12 (36.4) **Ω*β***
	80A	99.3 ± 0.9 (0.9) **β***	96.7 ± 4.6 (4.7) **β****	82.2 ± 10.9 (13.2) **β***	43.3 ± 17 (39.4)	15.5 ± 14.7 (94.3)
	110A	99.4 ± 1 (1) **Ω***	98.4 ± 1.7 (1.8) **Ω****	91.9 ± 7.4 (8) **Ω**α***	58 ± 11.8 (20.3) **Ω* α****	18.8 ± 13.8 (73.1)

* = $P < 0.05$, ** = $P < 0.001$, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

Table 9.12 – Full 3 x 8-min HIIT session time at % of HRmax results.

		Time at % HRmax Mean ± SD (CV%)				
		60	**70**	**80**	**90**	**95**
Total Work (s)	**PA**	1431 ± 12 (1)	1396 ± 26 (2)	1313 ± 59 (5)	962 ± 218 (23) **β***	539 ± 268 (5) **Ω***
	80A	1426 ± 13 (1)	1405 ± 28 (2) **β***	1301 ± 84 (6)	817 ± 299 (37)	363 ± 288 (80)
	110A	1431 ± 11 (1)	1410 ± 22 (2) **Ω***	1337 ± 54 (4)	887 ± 215 (24)	350 ± 220 (63)
Total Work (%)	**PA**	99.4 ± 0.9 (0.9)	97 ± 1.8 (1.9)	91.2 ± 4.1 (4.5)	66.8 ± 15.1 (22.7) **β***	37.4 ± 18.6 (49.7) **Ω***
	80A	99 ± 0.9 (0.9)	97.5 ± 2 (2) **β***	90.4 ± 5.8 (6.4)	56.8 ± 20.7 (36.5)	25.2 ± 20 (79.5)
	110A	99.4 ± 0.7 (0.7)	97.9 ± 1.5 (1.5) **Ω***	92.8 ± 3.7 (4)	61.6 ± 15 (24.3)	24.3 ± 15.3 (63)
Total Session (s)	**PA**	1773 ± 109 (6)	1587 ± 85 (5)	1427 ± 77 (5)	1026 ± 228 (22) **β***	569 ± 276 (49)
	80A	1907 ± 12 (1) **β****	1811 ± 112 (6) **β****	1515 ± 164 (11) **β***	884 ± 329 (37)	383 ± 308 (80)
	110A	1911 ± 11 (1) **Ω****	1889 ± 22 (1) **Ω****	1732 ± 140 (8) **Ω****α****	1014 ± 288 (28)	415 ± 280 (68)
Total Session (%)	**PA**	92.4 ± 5.7 (6.1)	82.7 ± 4.5 (5.4)	74.3 ± 3.9 (5.3)	53.4 ± 11.9 (22.3) **β***	29.7 ± 14.4 (48.7)
	80A	99.4 ± 0.6 (0.7) **β****	94.4 ± 5.8 (6.2) **β****	78.9 ± 8.5 (10.8) **β***	46.1 ± 17.2 (37.3)	20 ± 16 (80.2)
	110A	99.5 ± 0.6 (0.6) **Ω****	98.4 ± 1.1 (1.1) **Ω****	90.2 ± 7.3 (8.1) **Ω****α****	52.9 ± 15 (28.3)	21.6 ± 14.6 (67.6)

* = $P < 0.05$, ** = $P < 0.001$, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

Table 9.13 – Full 6 x 4-min HIIT session time at % of $\dot{V}O_{2max}$ results.

		Time at % $\dot{V}O_{2max}$ Mean ± SD (CV%)				
		60	**70**	**80**	**90**	**95**
Total Work (s)	**PA**	1353 ± 39 (3)	1296 ± 55 (4)	1168 ± 141 (12)	806 ± 266 (33)	516 ± 263 (51)
	80A	1379 ± 67 (5)	1263 ± 205 (16)	1034 ± 358 (35)	669 ± 392 (59)	444 ± 328 (74)
	110A	1406 ± 38 (3) **Ω**α***	1328 ± 203 (15) **α****	1161 ± 372 (32) **α***	749 ± 417 (56)	523 ± 384 (73)
Total Work (%)	**PA**	93.9 ± 2.7 (2.9)	90.1 ± 3.8 (4.2)	81.1 ± 9.8 (12.1)	56 ± 18.4 (32.8)	35.8 ± 18.3 (51)
	80A	95.8 ± 4.7 (4.9)	87.7 ± 14.2 (16.2)	71.8 ± 24.8 (34.6)	46.4 ± 27.2 (58.7)	30.9 ± 22.8 (73.8)
	110A	97.7 ± 2.6 (2.7) **Ω**α***	92.2 ± 14.1 (15.3) **α****	80.7 ± 25.8 (32) **α***	52 ± 29 (55.7)	36.3 ± 26.6 (73.4)
Total Session (s)	**PA**	1499 ± 39 (3)	1404 ± 59 (4)	1229 ± 156 (13)	834 ± 280 (34)	536 ± 279 (52)
	80A	1889 ± 174 (9) **β****	1631 ± 329 (20) **β***	1284 ± 498 (39)	786 ± 485 (62)	504 ± 380 (75)
	110A	1984 ± 112 (6) **Ω**α***	1879 ± 336 (18) **Ω**α****	1597 ± 535 (33) **Ω* α***	973 ± 558 (57)	659 ± 492 (75)
Total Session (%)	**PA**	73.5 ± 1.9 (2.6)	68.8 ± 2.9 (4.2)	60.2 ± 7.6 (12.7)	40.9 ± 13.7 (33.6)	26.3 ± 13.7 (52.3)
	80A	92.6 ± 8.5 (9.2) **β****	80 ± 16.1 (20.2) **β***	62.9 ± 24.4 (38.8)	38.6 ± 23.8 (61.7)	24.7 ± 18.6 (75.4)
	110A	97.3 ± 5.5 (5.7) **Ω**α***	92.1 ± 16.4 (17.9) **Ω**α****	78.3 ± 26.2 (33.5) **Ω* α***	47.6 ± 27.2 (57.2)	32.3 ± 24.1 (74.7)

* = $P < 0.05$, ** = $P < 0.001$, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

Table 9.14 – Full 3 x 8-min HIIT session time at % of $\dot{V}O_{2max}$ results.

		Time at % $\dot{V}O_{2max}$ Mean ± SD (CV%)				
		60	**70**	**80**	**90**	**95**
Total Work (s)	**PA**	1389 ± 22 (2)	1347 ± 45 (3)	1217 ± 131 (11)	841 ± 321 (38) **β***	499 ± 301 (60)
	80A	1399 ± 44 (3)	1322 ± 154 (12)	1116 ± 334 (30)	686 ± 320 (47)	383 ± 274 (72)
	110A	1407 ± 15 (1) **Ω****	1364 ± 60 (4)	1101 ± 323 (29)	640 ± 373 (58)	377 ± 332 (88)
Total Work (%)	**PA**	96.4 ± 1.5 (1.6)	93.5 ± 3.1 (3.3)	84.5 ± 9.1 (10.8)	58.4 ± 22.2 (38) **β***	34.7 ± 20.9 (60.4)
	80A	97.2 ± 3.1 (3.2)	91.8 ± 10.7 (11.7)	77.5 ± 23.2 (29.9)	47.6 ± 22.2 (46.6)	26.6 ± 19 (71.6)
	110A	97.7 ± 1 (1.1) **Ω****	94.7 ± 4.1 (4.4)	76.5 ± 22.4 (29.3)	44.4 ± 25.9 (58.2)	26.2 ± 23.1 (88.1)
Total Session (s)	**PA**	1462 ± 27 (2)	1403 ± 48 (3)	1254 ± 131 (10)	861 ± 325 (38) **β***	513 ± 306 (60)
	80A	1736 ± 155 (9) **β****	1514 ± 220 (15) **β***	1216 ± 369 (30)	716 ± 331 (46)	399 ± 284 (71)
	110A	1894 ± 16 (1) **Ω**α***	1769 ± 152 (9) **Ω**α****	1339 ± 449 (34)	719 ± 434 (60)	413 ± 372 (90)
Total Session (%)	**PA**	76.2 ± 1.4 (1.9)	73.1 ± 2.5 (3.4)	65.3 ± 6.8 (10.4)	44.8 ± 16.9 (37.6) **β***	26.7 ± 16 (59.9)
	80A	90.4 ± 8.1 (8.9) **β****	78.8 ± 11.4 (14.5) **β***	63.3 ± 19.2 (30.3)	37.3 ± 17.2 (46.2)	20.8 ± 14.8 (71.3)
	110A	98.6 ± 0.8 (0.9) **Ω**α***	92.2 ± 7.9 (8.6) **Ω**α****	69.8 ± 23.4 (33.5)	37.5 ± 22.6 (60.3)	21.5 ± 19.4 (90)

* = $P < 0.05$, ** = $P < 0.001$, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A.

Table 9.15 – Additional work interval PO and HR results from the 6 x 4-min HIIT sessions.

		Work Interval PO and HR results Mean ± SD (CV%)						
	Int. No.	**1**	**2**	**3**	**4**	**5**	**6**	**Mean**
PO as % MMP	**PA**	84.2 ± 4.4 (5.2)	82.5 ± 3 (3.6)	81.7 ± 3.4 (4.1)	80.2 ± 3.6 (4.5)	79 ± 3.9 (5)	82 ± 3.4 (4.1)	81.6 ± 3 (3.6)
	80A	79.3 ± 6 (7.6)	79.4 ± 4 (5)	78.2 ± 2.8 (3.6)	77.9 ± 3 (3.9)	77.4 ± 3.8 (4.9)	78.8 ± 4.1 (5.2)	78.5 ± 3.3 (4.2)
	110A	79.6 ± 5.9 (7.4)	79.4 ± 3.8 (4.8)	78 ± 3 (3.8)	77.8 ± 3.4 (4.4)	77 ± 3 (3.9)	79.2 ± 3.2 (4.1)	78.5 ± 3 (3.8)
Relative PO ($W.kg^{-1}$)	**PA**	4.5 ± 0.8 (18.5)	4.4 ± 0.8 (17.8)	4.3 ± 0.8 (17.8)	4.2 ± 0.8 (17.8)	4.2 ± 0.7 (17.5)	4.3 ± 0.8 (17.8)	4.3 ± 0.8 (17.7)
	80A	4.2 ± 0.8 (19.3)	4.2 ± 0.7 (17.4)	4.1 ± 0.7 (16.4)	4.1 ± 0.7 (16.5)	4.1 ± 0.7 (17)	4.2 ± 0.7 (16.9)	4.1 ± 0.7 (17)
	110A	4.2 ± 0.8 (20)	4.2 ± 0.7 (17.4)	4.1 ± 0.7 (15.9)	4.1 ± 0.6 (15.8)	4.1 ± 0.6 (15.9)	4.2 ± 0.7 (16.8)	4.1 ± 0.7 (16.8)
HR as % Max	**PA**	84.6 ± 3.5 (4.2)	88.8 ± 2.2 (2.4)	90.6 ± 2.1 (2.4)	91 ± 2.2 (2.4)	91.6 ± 1.9 (2)	93.4 ± 2.5 (2.6)	90 ± 1.9 (2.1)
	80A	82.4 ± 3.8 (4.7)	87.8 ± 2.7 (3)	88.9 ± 3.1 (3.4)	89.8 ± 3.3 (3.6)	90.7 ± 3.8 (4.2)	92.3 ± 4.1 (4.4)	88.7 ± 3 (3.4)
	110A	82.2 ± 3.6 (4.4)	89 ± 1.7 (1.9)	90.9 ± 2.1 (2.3)	91.9 ± 2.2 (2.4)	93 ± 2.5 (2.7)	94.9 ± 2.3 (2.4)	90.3 ± 1.9 (2.1)

Table 9.16 - Additional work interval PO and HR results from the 3 x 8-min HIIT sessions.

		Work Interval PO and HR results Mean ± SD (CV%)			
	Int. No.	**1**	**2**	**3**	**Mean**
PO as % MMP	**PA**	79.1 ± 2.6 (3.3)	77.9 ± 3.9 (5)	77.7 ± 4.4 (5.7)	78.2 ± 3.3 (4.2)
	80A	76.6 ± 3.8 (5)	77.1 ± 3 (3.9)	76.2 ± 4.1 (5.3)	76.7 ± 3.2 (4.2)
	110A	76.3 ± 4.5 (5.8)	75.7 ± 2.7 (3.5)	76.2 ± 2.8 (3.6)	76.1 ± 3.1 (4)
Relative PO ($W.kg^{-1}$)	**PA**	4.2 ± 0.7 (17.2)	4.1 ± 0.7 (17.9)	4.1 ± 0.7 (18.1)	4.1 ± 0.7 (17.6)
	80A	4.1 ± 0.8 (18.6)	4.1 ± 0.7 (17)	4 ± 0.7 (16.7)	4 ± 0.7 (17.3)
	110A	4 ± 0.8 (18.6)	4 ± 0.7 (16.4)	4 ± 0.7 (16.5)	4 ± 0.7 (17.1)
HR as % Max	**PA**	88.1 ± 3.3 (3.8)	91.3 ± 2.4 (2.6)	93.2 ± 2.2 (2.4)	90.8 ± 2.3 (2.6)
	80A	84.7 ± 3.9 (4.6)	90.7 ± 3.2 (3.5)	92.4 ± 3.6 (3.9)	89.3 ± 3.1 (3.5)
	110A	84.8 ± 3.4 (4.1)	91.8 ± 2.2 (2.4)	93.6 ± 2.4 (2.6)	90.1 ± 1.9 (2.1)

Table 9.17 – Additional work interval $\dot{V}O_2$ results from the 6 x 4-min HIIT sessions.

		$\dot{V}O_2$ (L.min^{-1}) Mean ± SD (CV%)					
		1	**2**	**3**	**4**	**5**	6
Int. Mean	**PA**	3.54 ± 0.5 (15.1) **β****	**φT** 3.73 ± 0.6 (15.6)	**φ** 3.8 ± 0.6 (16.6)	**φT** 3.88 ± 0.6 (15.8)	**φ** 3.84 ± 0.6 (16.7)	**φT** 3.89 ± 0.6 (15.5)
	80A	3.34 ± 0.5 (16.4)	3.75 ± 0.7 (18.6)	3.81 ± 0.7 (19.1)	3.83 ± 0.7 (19.5)	3.84 ± 0.8 (20.1)	3.92 ± 0.8 (21.6) **β***
	110A	3.45 ± 0.6 (16.3)	3.92 ± 0.7 (17.6) **α***	3.99 ± 0.7 (18.1) **α***	4.06 ± 0.7 (18.3) **α***	4.06 ± 0.7 (18.4) **α***	4.13 ± 0.8 (18.3) **α***
Int. Mean % of max	**PA**	81.9 ± 5.6 (6.9) **β****	86.2 ± 5.4 (6.3)	87.5 ± 5.5 (6.2)	89.6 ± 3.8 (4.2)	88.4 ± 4.5 (5)	89.9 ± 5.5 (6.1)
	80A	77.3 ± 6.9 (8.9)	86.5 ± 8.5 (9.8)	87.6 ± 7.3 (8.4)	88.2 ± 8.1 (9.2)	88.2 ± 8.7 (9.9)	90 ± 10.5 (11.6)
	110A	80 ± 9.5 (11.9)	90.7 ± 10.1 (11.1) **α***	92.1 ± 8.8 (9.5) **α***	93.6 ± 9.2 (9.9) **α***	93.6 ± 8.9 (9.5) **α***	95.3 ± 9.3 (9.8) **α***
Int. Max (30s)	**PA**	3.94 ± 0.6 (14.4)	**φT** 4.15 ± 0.6 (14.5)	**φ** 4.22 ± 0.6 (15.3)	**φT** 4.33 ± 0.6 (13.9)	**φ** 4.28 ± 0.7 (15.5)	**φT** 4.36 ± 0.6 (14.8)
	80A	3.91 ± 0.7 (18.6)	4.11 ± 0.8 (18.5)	4.16 ± 0.8 (19.2)	4.2 ± 0.8 (19.8)	4.18 ± 0.9 (20.8)	4.32 ± 0.9 (21.7)
	110A	4.04 ± 0.7 (17.4)	4.19 ± 0.8 (18.1)	4.28 ± 0.8 (18)	4.33 ± 0.8 (17.4)	4.34 ± 0.8 (18.7)	4.44 ± 0.7 (16.4)
Int. Max % of max	**PA**	91.2 ± 6.1 (6.7)	96 ± 6.5 (6.7)	97.4 ± 5.5 (5.6)	100.1 ± 4 (4)	98.7 ± 5.1 (5.2)	100.8 ± 6.1 (6.1)
	80A	90.2 ± 9.1 (10.1)	94.9 ± 9.9 (10.5)	95.6 ± 8.7 (9.1)	96.6 ± 9.8 (10.2)	96.1 ± 10.4 (10.8)	99.2 ± 12.6 (12.7)
	110A	93.4 ± 11 (11.7)	96.8 ± 10.9 (11.3)	98.8 ± 9.6 (9.7)	100 ± 10 (10)	100 ± 10.1 (10.1)	102.7 ± 9.7 (9.5)

* = $P < 0.05$, ** = $P < 0.001$, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A, φ = Significant increase from interval 1, T = Significant increase from previous interval.

Table 9.18 – Additional work interval $\dot{V}O_2$ results from the 3 x 8-min HIIT sessions.

		$\dot{V}O_2$ (L.min^{-1}) Mean ± SD (CV%)		
		1	**2**	**3**
Int. Mean	**PA**	3.72 ± 0.6 (15.3) **β***	**φT** 3.89 ± 0.6 (16)	**φ** 4.01 ± 0.7 (16.5)
	80A	3.59 ± 0.7 (18.8)	3.89 ± 0.7 (18.5)	3.92 ± 0.8 (19.8)
	110A	3.57 ± 0.7 (19.1)	3.94 ± 0.8 (20)	3.97 ± 0.8 (19.7)
Int. Mean % of max	**PA**	85.9 ± 4.9 (5.7) **β***	89.7 ± 4.6 (5.2)	92.5 ± 5.6 (6)
	80A	82.8 ± 7.9 (9.5)	89.4 ± 6.6 (7.3)	90.2 ± 7.8 (8.6)
	110A	82.1 ± 7.3 (8.8)	90.5 ± 8 (8.9)	91.3 ± 7.6 (8.3)
Int. Max (30s)	**PA**	4.18 ± 0.6 (14.5)	**φT** 4.36 ± 0.6 (14.8)	**φT** 4.49 ± 0.7 (16.1) **β***
	80A	4.07 ± 0.7 (17.9)	4.25 ± 0.7 (16.8)	4.31 ± 0.8 (19.4)
	110A	4.04 ± 0.8 (19.6)	4.21 ± 0.8 (19.8)	4.33 ± 0.8 (18.4)
Int. Max % of max	**PA**	96.6 ± 5.1 (5.3)	100.7 ± 5.2 (5.2)	103.6 ± 6.8 (6.6) **β***
	80A	93.9 ± 8.3 (8.8)	98.1 ± 7.3 (7.4)	99.2 ± 8.9 (8.9)
	110A	92.9 ± 8.7 (9.3)	96.9 ± 8.5 (8.7)	99.9 ± 8.3 (8.3)

* = $P < 0.05$, ** = $P < 0.001$, Ω = Significant difference between PA & 110A, β = Significant difference between PA & 80A, α = Significant difference between 80A & 110A, φ = Significant increase from interval 1, T = Significant increase from previous interval.