Enhancing Team Pursuit Performance using Blood Flow Restriction.

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Abstract

The studies in this thesis investigated the physiological determinants of 4-km team-pursuit (TP) track cycling performance and critically evaluated the use of modelling finite work capacity (W′) and its dynamic balance (W'_{BAL}) during the TP. This thesis also examined the integration of blood flow restriction (BFR) into high-intensity interval training (HIIT) as an intervention to improve factors related to TP performance. A series of related investigations were conducted with trained cyclists up to the Olympic level.

Study One recruited male TP squads from International, National, and Regional performance levels. The TP squads were assessed for their critical power (CP) and W′. Maximal 4-km TP efforts confirmed different performance times of 3:49.9, 3:56.7, and 4:05.4 (minutes:s) for International, National, and Regional, respectively. Four TP simulation trials quantified W′ reconstitution from 0 to 100 W below CP. Results showed that the International squad were differentiated from National and Regional performance levels with greater CP ($p < 0.05$), likely preserving W′ for leading efforts. Furthermore, the International team possessed the fastest rates of W′ reconstitution at recovery intensities within 50 W of CP ($p < 0.05$), demonstrating the importance of W′ reconstitution at intensities near CP for recovery in the TP. The International team also expended a greater total quantity of W′ than its initial size (104 ± 5%), further demonstrating the capacity to utilise the reconstituted W′. In conclusion, we found that the TP relies on high aerobic capacity and rapid metabolic recovery abilities.

An intervention was conceived based on the demands of the TP and the existing training sessions of elite TP cyclists. The training intervention included principles of TP training philosophy where cyclists repeatedly practice competition demands, at their TP lead intensity. As elite TP cyclists engage in substantial training volumes, it was important not to substantially exceed current training workloads. Based on previous BFR research with trained cyclists, an intervention integrating BFR into the recovery between TP efforts was devised. The intervention was performed on an ergometer to enable greater control over conditions and intensity.

To evaluate the metabolic demands of the BFR intervention, the Study Two assessed the acute physiological responses in 11 male and female highly-trained cyclists ($\dot{V}O_{2PEAK}$ 65 ± 9 $mL \cdot kg^{-1} \cdot minute^{-1}$). Using a within-subject design, participants performed two work- and duration-matched HIIT sessions. The HIIT consisted of six high-intensity repetitions with BFR occlusion between work bouts at 200 mmHg for 2-minutes applied proximally on the thighs (BFR) or HIIT alone without BFR (CON). Work intensity was set as 85% of the mean power output of a maximal 30-s test to simulate TP lead intensity. Cardiopulmonary variables (O_2 uptake, $\dot{V}O_2$; carbon dioxide production $\dot{V}CO_2$; and ventilation, $\dot{V}E$) and muscle oxygenation responses were measured during the HIIT, and vascular endothelial growth factor (VEGF) was measured pre- and 3-hours post-HIIT. Results demonstrated that BFR increased $\dot{V}CO_2$ and $\dot{V}E$ (both $p < 0.05$) during work bouts but did not affect $\dot{V}O_2$ and TSI (both $p > 0.05$). Compared to CON, the BFR intervention significantly decreased $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, and TSI during BFR occlusion (all $p < 0.05$). Following cuff release, there were significantly higher values of $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}E$, whereas TSI was suppressed (all $p < 0.05$). There were significant enhancements of serum VEGF concentration at 3-hours post-HIIT after BFR when compared to CON. As BFR appeared to delay recovery, it was hypothesised that BFR may increase metabolic and oxidative stress by delaying recovery processes. The delay in recovery may enhance the adaptations to HIIT without increasing training workload.

After demonstrating that applying BFR during recovery in high-intensity work bouts increased markers of physiological stress, Study Three assessed the performance and physiological effects of the training as a chronic intervention. Using a between-subject design, ten performance-matched male trained cyclists (weekly volume >6-hours$\cdot week^{-1}$) were assigned to BFR or CON conditions. Participants performed pre- and post-intervention tests to determine lactate thresholds, 30-s maximal sprint cycling performance, and an intermittent test designed with high-intensity bouts comparable to the TP. Work bouts were performed at 85% of the mean power output of the maximal 30-s test. Muscle oxygenation and cardiopulmonary measures were continually assessed throughout the intermittent test. Participants performed four-weeks of work- and duration-matched HIIT either with 2-minutes of 200 mmHg thigh BFR between work bouts or HIIT alone (CON). Following BFR intervention, there were significant

improvements in intermittent test time to exhaustion, 30-s mean power output, and submaximal lactate thresholds compared to CON (all $p < 0.05$). Furthermore, BFR led to significant intermittent test improvements for $\dot{V}O_{2PEAK}$ and the rate of muscle tissue reoxygenation (all $p < 0.05$). There were no significant changes over the intervention period for CON, indicating that HIIT was ineffective in this cohort when BFR was not incorporated. Therefore, it was demonstrated that the integration of BFR between HIIT work bouts improves intermittent performance and a range of physiological factors associated with performance in trained cyclists.

Finally, the BFR intervention was integrated into two HIIT sessions within a training camp of an elite TP squad preparing for the Olympic Games to test its potential efficacy and feasibility. As in the previous BFR studies, this case-study (Study Four) applied 2-minutes of 200 mmHg thigh BFR between high-intensity bouts. Work intensities were set at the individual cyclists' TP lead intensity. A questionnaire was developed to assess the pain, tolerance, enjoyment, and compare the intervention to other training modalities. Questionnaire responses indicated that the elite cyclists enjoyed and positively perceived the intervention, appreciating the variety and efficiency of the training stimulus. All but one elite cyclist tolerated that intervention. Further investigation in conjunction with medical staff indicated that the intolerant cyclist had a pre-existing undiagnosed cardiovascular condition and presented with femoral artery claudication (discussed in the addendum). Thus, integrating BFR into HIIT for elite track cyclists was feasible and tolerable when no contraindications existed.

In summary, elite TP performance relies on high sustained aerobic power output and rapid W′ recovery between efforts. This thesis showed integrating BFR between HIIT work bouts provides an additional training stimulus and can improve factors related to aerobic capacity and high-intensity intermittent performance in trained cyclists. The BFR intervention is tolerable within an elite cohort and may improve TP performance without increasing training workload.

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Time for a ride.

Publications and presentations arising from this work

Journal articles:

Chapter Three: Pugh, C. F., Beaven C. M., Ferguson R. A., Driller M. W., Palmer C. D., C. D. Paton (2022). "Critical Power, Work Capacity, and Recovery Characteristics of Team-Pursuit Cyclists." International Journal of Sports Physiology and Performance **17**(11): 1-8.10.1123/ijspp.2021-0478

Chapter Four: Pugh, C. F., C. D. Paton, R. A. Ferguson, M. W. Driller, C. M. Beaven, (2023). "Acute physiological responses of blood flow restriction between high-intensity interval repetitions in trained cyclists." Under review at European Journal of Sport Science

Conference presentations:

Oral Presentations:

Pugh C., Ferguson R., Driller M. FTP is an estimate of CP. Annual Conference of Sport and Exercise Science New Zealand. 2019. Palmerston North, New Zealand.

Pugh C., Driller M., Ferguson R., Paton C., Beaven M. Team Pursuit: Fitter is Faster. Annual Conference of Sport and Exercise Science New Zealand. 2022. Auckland, New Zealand. * ***, Won *Best Oral Presentation***

Welburn A., Bailey S., Pugh C., Ferguson R. Physiological Characteristics Associated with W′ and W'_{BAL} Used During Intermittent Exercise Task to Failure. Journal of Science and Cycling. 2023, **12**:2 Bilbao, Spain.

Pugh, C. F., C. D. Paton, R. A. Ferguson, M. W. Driller, C. M. Beaven, (2023). Acute Physiological Responses of Blood Flow Restriction During Recovery in High-Intensity Interval Training in Trained Cyclists. Annual Conference of Sport and Exercise Science New Zealand. 2023. Wellington, New Zealand.

Poster Presentations:

Pugh C., Driller M., Ferguson R., Paton C., Beaven M. Understanding Work Restitution. Annual Conference of Sport and Exercise Science New Zealand. 2020. Christchurch, New Zealand.

Inclusion in media:

Cheung, S. (2023, September 6). Repeatability: How Many Matches to Burn? PEZ Cycling News. https://pezcyclingnews.com/toolbox/repeatability-how-many-matches-to-burn/

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Abbreviations and Symbols

ANOVA	Analysis of variance
ATP	Adenosine triphosphate
BFR	Blood flow restriction
°C	Degrees Celsius
CON	Control
CO_2	Carbon dioxide
CP	Critical power
D_{CP}	Absolute difference of recovery work rate and critical power
ELISA	Enzyme-linked immunosorbent assay
EPOC	Excess post-exercise oxygen consumption
H^+	Hydrogen ion
HIF-1α	Hypoxia-inducible factor 1-alpha
HIIT	High-intensity interval training
HR	Heart rate
kJ	Kilojoule
LT1	Lactate threshold 1
MAP	Maximal aerobic power
MPO	Mean power output
mRNA	Messenger RNA
O_2	Oxygen
PCr	Phosphocreatine
PGC-1α	Peroxisome proliferator-activated receptor gamma coactivator 1-alpha
pH	Power of hydrogen
Pi	Inorganic phosphate ion
PPO	Peak power output
s	Seconds
SIT	Sprint interval training
$\tau_{W'}$	Time constant of W′ reconstitution, *Tau*
TP	Team pursuit
TSI	Tissue saturation index
TT	Time trial
TTE	Time to exhaustion at a given power output
$\dot{V}CO_2$	Rate of carbon dioxide production
$\dot{V}E$	Rate of ventilation
VEGF	Vascular endothelial growth factor
$\dot{V}O_2$	Oxygen utilisation rate
$\dot{V}O_{2MAX}$	Maximal oxygen utilisation rate
$\dot{V}O_{2PEAK}$	Peak oxygen utilisation rate
$\dot{V}O_{2PEAK\text{-}PERF}$	Peak oxygen utilisation rate during the intermittent performance test
$\dot{V}O_{2PEAK\text{-}STEP}$	Peak oxygen utilisation rate during the stepped incremental test
W	Watts
W′	Work capacity above critical power
W'_{ERG}	Size of the W′ during ergometer cycling
W'_{EXP}	Quantity of expended W′
$W'_{TRAINING}$	Size of the W′ during training

Chapter One – Thesis overview

General introduction

The team-pursuit (TP) is a track cycling event where teams of 4 riders compete to complete 4000-m in the fastest time. Performances in International level male TP competitions like the Olympic Games have improved steadily in recent decades, with the current world record standing at 3:42.032 (mean velocity: 64.85-km·hr^{-1}) set at the Tokyo 2020 Olympic Games. This progression can partly be attributed to advancements in engineering and aerodynamics leading to improved cycling efficiency (Lukes *et al.* 2005).

Tracking or simulating the dynamic balance of high-intensity work capacity (W'_{BAL}) during the TP allows for more precise pacing strategy individualisation and has also contributed to faster times (Bartram *et al.* 2021). Understanding how the W'_{BAL} model parameters of critical power (CP), work prime (W'), and ability to reconstitute W' differ across TP performance levels may provide insight into methods to enhance TP performance. Previous research demonstrated that TP cyclists have endurance-orientated characteristics with similarities to road and time-trial cyclists (Van Der Zwaard *et al.* 2018). However, few studies have reported the CP, W', and the rate of W' reconstitution in contemporary International level teams (Bartram *et al.* 2018), and no previous research has compared these factors across performance levels.

Intermittent exercise performance, characterised by periods of high-intensity work followed by low-intensity recovery periods, is limited by the attainment of critical maximum/minimum values of intracellular and extracellular metabolites and substrate concentrations (Ferguson *et al.* 2010, Poole *et al.* 2016). Relief periods allow these fatigue-related metabolites to recover towards baseline values, with the magnitude of recovery proportional to the intensity and duration of the recovery (Skiba *et al.* 2012, Chidnok *et al.* 2013a). Moreover, the reserve oxidative capacity of the active muscle group helps restore homeostasis following metabolite accumulation and substrate depletion (Thoden 1991).

Endurance training of sufficient intensity and duration triggers adaptations that enhance the performance of prolonged workouts. These adaptations include mitochondrial biogenesis, angiogenesis, muscle fibre type conversion from type IIx to IIa, increased muscle glycogen content, and alterations to substrate metabolism at submaximal workloads (Joyner and Coyle 2008). These changes improve endurance performance by increasing substrate availability, facilitating the removal of fatigue-related metabolites, and enhancing oxygen (O_2) delivery ($\dot{Q}O_2$) and uptake ($\dot{V}O_2$) to active tissue (Van Der Zwaard *et al.* 2018). However, as training status improves, the physiological signals driving these adaptations decrease in magnitude (Laursen and Jenkins 2002a), and innovative training methods may be necessary for athletes to drive continued adaptation given the already high levels of fitness.

High intensity interval training (HIIT) has demonstrated efficacy in enhancing adaptations in highly-trained individuals (Rønnestad *et al.* 2020). When low-volume HIIT and medium-volume low-intensity training (LIT) are periodised within a training programme, high-performance athletes can gain benefits of both training modalities while mitigating excessive sympathetic stress that may lead to non-functional overreaching or performance plateaus (Guellich *et al.* 2009). Some of the most successful endurance athletes are reported to maintain the largest weekly volume of low-intensity training, which is achieved by reducing the proportion of HIIT work (Foster *et al.* 2022). Thus, HIIT develops high-intensity performance for competition (Schumacher and Mueller 2002), whereas LIT targets the key endurance physiological characteristics identified in elite TP cyclists (Van Der Zwaard *et al.* 2018).

Given that even minor performance improvements can be critical for success in elite competitions (Hopkins *et al.* 1999), developing effective training methods is crucial for those seeking optimal performance. While increasing training load or intensity can maintain adaptive signals, there is an attenuation in performance adaptions following a 3+ week HIIT programme (Paton and Hopkins 2005, Norrbom *et al.* 2022), with a raised risk of non-functional overreaching and maladaptation (Midgley *et al.* 2006). Understanding the training strategies that produce sustained performance enhancements in W'_{BAL} parameters will be key for improving performance in elite TP cyclists.

Blood flow restriction (BFR) training, which limits blood flow in and/or out of active limbs, has emerged as one strategy that enhances metabolic stress and adaptive signals compared to unrestricted

exercise (Fujita *et al.* 2007). While most research has focused on BFR with low-load resistance training (de Oliveira *et al.* 2016), there is potential for BFR to augment high-intensity training (Taylor 2016, Mitchell 2019, Ienaga *et al.* 2022). Applying BFR specifically during recovery periods of HIIT could provide an additional stimulus for highly-trained athletes without increased mechanical workload. However, the feasibility and impacts of this approach are yet to be examined in an elite TP population.

Therefore, the overall aim of this thesis is two-fold: 1) To characterise key physiological determinants of TP performance including CP, W′ and W′ reconstitution kinetics; 2) To investigate BFR during HIIT as a method to enhance training adaptations and performance in highly-trained endurance cyclists. The findings will address knowledge gaps regarding TP physiology and the potential of BFR to progress elite athletes beyond a training plateau.

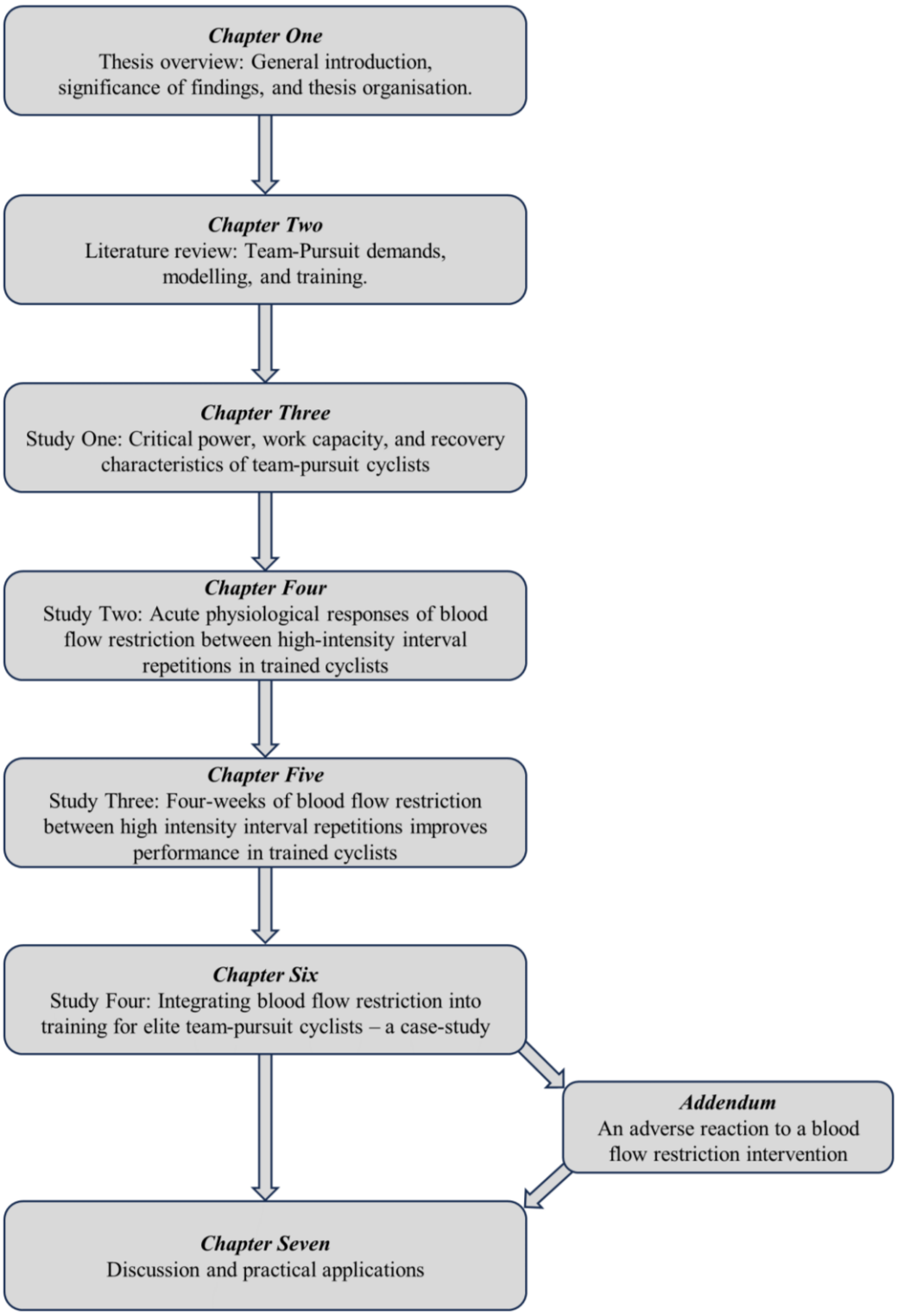
Significance of findings

This research makes important contributions to our understanding of elite team-pursuit cycling performance. To remain competitive at the Olympic and World Championship level, national sporting bodies invest substantially in podium finishes by optimising the various factors underpinning team-pursuit success. Recently, the emergence of innovative modelling techniques and training methods may offer a potential advantage to team-pursuit squads. Leveraging these advancements, this research was the first to present the physiological factors that distinguish team-pursuit performance levels and demonstrate the application of individualised modelling to inform pacing strategies and training principles. A novel blood flow restriction training intervention was integrated within an existing team-pursuit training modality to enhance training stimulus. Through controlled experimental studies, the intervention demonstrated preliminary findings of enhanced intermittent performance and provided mechanistic insights into the origins of adaptions. The feasibility and tolerance of the training method was then verified in an elite team preparing for the Olympics Games. Collectively, the research serves as a valuable resource for enhancing team-pursuit performance through the implementation of innovative assessment and training techniques tailored to elite cyclists.

Chapter organisation

This thesis is comprised of seven chapters (see Schematic of thesis structure). Chapter One provides a concise introduction to the topic and delves into the significance of the findings presented in this thesis. Chapter Two provides a review of existing literature on the physiological demands of the team-pursuit event and identifies opportunities for novel training methods and optimisations to further enhance performance capabilities. Chapters Three through Five present experimental studies, each written as a stand-alone paper either published, under review, or prepared for submission in peer-reviewed journals. Specifically, Chapter Three employs individualised modelling to identify key W'_{BAL} parameters that differentiate performance levels in team-pursuit. Chapter Four assesses the acute physiological effects of a unique training intervention within highly-trained cyclists. Chapter Five, informed by the previous chapters, explores the potential of this intervention to induce chronic adaptations that may enhance team-pursuit performance. Chapter Six provides an insight into the practical application of a novel training intervention within an elite team-pursuit squad as they prepared for the Olympic Games. An addendum chapter elaborates on an adverse reaction to the intervention also intended to be submitted for peer-review in a journal following thesis submission. For consistency, these chapters follow a standard format (Abstract, Introduction, Methods, Results, Discussion), though these articles may appear in different formats when presented in their respective journals, according to journal specifications. Given the structure of self-contained chapters, some repetition is unavoidable. To aid readability, all citations are consolidated into a single bibliography at the end of the thesis.

Schematic of thesis structure



Chapter Two – Literature review

Introduction

This literature review begins by providing background on the team-pursuit (TP) cycling event, including the format, historical performance trends, physiological demands, drafting positions, and resistive forces. It then explores current knowledge on physiological determinants of endurance performance, such as exercise intensity domains and the critical power model. Adaptations of trained athletes to endurance and high-intensity interval training are examined.

Emerging research suggests that blood flow restriction (BFR) training may augment the training stimulus and accelerate adaptations. Therefore, a dedicated section reviews potential mechanisms and applications of pairing BFR with endurance training, analysing effects on angiogenesis, mitochondrial biogenesis, and post-exercise recovery kinetics.

This review synthesises current research on the TP event, determinants of endurance performance like the critical power model, and evidence-based training interventions like BFR. The scope enables making targeted recommendations for training practices to optimise TP performance in highly-trained cyclists. Overall, this provides context and justification for studying how BFR may enhance TP performance determinants like aerobic capacity and recovery.

The team-pursuit event

Format and rules

The team-pursuit (TP) is a 4000-meter track cycling event where teams of four cyclists compete to record the fastest time over the distance. The TP takes place on velodromes, beginning from a standing start. Cyclists use fixed-gear bicycles where pedalling cadence is directly proportional velocity on a given gear-ratio.

In major competitions like the Olympics, teams first race solo in a qualifying round. The top qualifying teams then advance to head-to-head races in the first round and finals. If one team catches the other, they automatically win (hence "team-*pursuit*"). The finishing time is recorded from the third cyclist crossing the line, meaning only three need to complete the full distance. Strategic utilisation of a fourth 'sacrificial' cyclist may conserve energy in teammates.

Historical TP performances

The track cycling world record progression for the TP has steadily improved over time. The current record of 3:42.032 (minutes:s.ms), set at the 2020 Tokyo Olympics, equates to an average velocity of ~64.9-km·h^{-1}. Major milestones include surpassing the 4-minute barrier (60.0-km·h^{-1} average) at the 2000 Sydney Olympics and breaking the 3:50.000 mark (62.6-km·h^{-1} average) at the 2018 Gold Coast Commonwealth Games.

Though track cycling events occur throughout the year via World/Nations Cup meets, TP world records are frequently set at high-profile competitions like the Olympics, World Championships, or Commonwealth Games. International federations often target these events for peak performance, as they offer the most prestige and financial rewards. Figure 1 demonstrates the progression of world record performances for the male individual pursuit (for comparison), TP, as well as a modelled prediction of the male TP world record by Schumacher *et al.* (2002). The Schumaker and Mueller model used a simple linear equation relating record mean velocity to the year:

$$\textit{TP Mean Velocity (km·hr}^{-1}\textit{)} = 0.2833\textit{(Date; Year)} - 506.6 \qquad \text{Equation 1}$$

$$\textit{4-km TP Time (s)} = (4 / (0.2833\textit{(Date; Year)} - 506.6)) * 3600 \qquad \text{Equation 2}$$

Using the Schumacher *et al.* (2002) model, the predicted Tokyo 2020 Olympics TP time would be 3:38.350. However, the fastest performance in Tokyo was slower than this predicted time, showing the rate of progression in TP performance has been below that predicted by the Schumaker and Mueller model over the past 20 years.

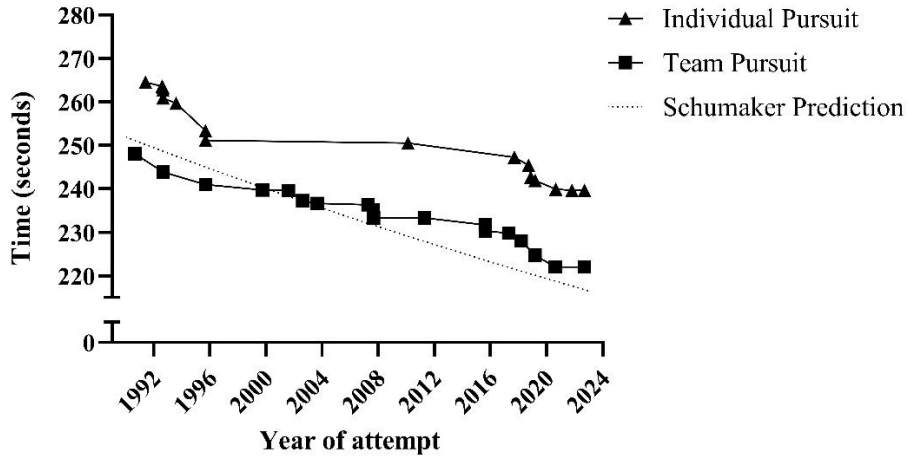


Figure 1. Time evolution of male individual pursuit and team-pursuit world record performances in cycling from 1992 to 2023.

To predict a TP performance with greater accuracy, more sophisticated models would need to utilise various approaches to quantify the performance potential, such as air resistance, power output, rider positioning, aerodynamics of rider and bicycle (Chabroux *et al.* 2012, Chowdhury and Alam 2014, Terra *et al.* 2018, Bigham 2021), track geometry (Lukes *et al.* 2012), technical ability (Guellich *et al.* 2009), and pacing strategies (Wagner *et al.* 2013). For example, Broker *et al.* (Broker *et al.* 1999) developed a mathematical model to estimate the power requirements during TP racing. Their model was based on direct power measurements obtained during competitive events. It incorporates factors related to rolling resistance, aerodynamics, and the combined mass of the rider and bicycle. Additional parameters also accounted for altitude, track conditions, velocity, estimated frontal area, and position on the bike.

There have been many occasions where technological innovations have led to significantly faster performances, and in some cases, these were deemed unfair or unsafe by the international cycling regulatory body, Union Cycliste Internationale (UCI) (Kyle 2000). For example, Graeme Obree's pioneering "tucked" and "superman" positions were demonstrated to reduce resistant drag forces in comparison to the standard positioning adopted at the time (Grappe *et al.* 1997), leading to the UCI restricting handlebar extensions to a 75 cm frontal projection from the bottom bracket in 2000 (UCI 1.03.023).

Resistive forces in track cycling

A cyclist's velocity can be described by Newton's second law and is determined by the interaction of propulsive and resistive forces acting upon them. The main propulsive forces while cycling are mechanical power output and losing gravitational potential energy. In contrast, the most influential resistive forces are aerodynamic drag, inertia, and gaining gravitational potential energy (Martin *et al.* 1998).

Velodrome cycling represents a highly standardised environment. Indoor velodromes permit control of ambient conditions to reduce air resistance by increasing temperature to reduce air density. Tracks that are used in international competitions must comply with regulations that define the range of acceptable track dimensions and geometries, although some variance is permitted that lead to velodromes with different length straight and corner sections. Velodromes have no change in altitude when travelling along the track's datum line. Therefore, the key forces determining track cycling velocity include mechanical power output, aerodynamic drag, rolling resistance, and centripetal forces in the corners (Martin *et al.*, 2006). In simplified terms, the fundamental equation for track cycling velocity as discussed by Lukes *et al.* (2012) is

$$Resultant\ Force = Propulsive\ Force - Aerodynamic\ Drag - Rolling\ Resistance. \quad \text{Equation 3}$$

Rider geometry of team-pursuit cyclists

Air resistance (aerodynamic drag) increases with the square of speed (Kyle and Burke 1984, Olds *et al.* 1993). At velocities above 40-km·h^{-1}, approximately 90% of a cyclist's power output is used to overcome aerodynamic drag forces (Kyle *et al.* 1984). The cyclist's body and wearable components, like helmets and skinsuits, are responsible for 64%–82% of the total system drag (Defraeye *et al.* 2010, Barry *et al.* 2015), whereas the bicycle has a reported 21-39% contribution on aerodynamic drag (Kyle *et al.* 1984, Malizia and Blocken 2021). While cycling on a velodrome at speeds greater than 30-km·h^{-1}, adopting an aerodynamically optimised position, all else being equal, would lead to reduced aerodynamic drag and an increased resultant velocity compared to an upright position (Grappe *et al.*

1997, Defraeye *et al.* 2010). Consequently, national sporting bodies invest substantially into aerodynamic materials, technology, and wind tunnel testing to optimise the ratio between power output and power demand. A position of horizontal torso and low head reduces frontal surface area and the coefficient of drag (Malizia *et al.* 2021), but maintaining this position has biomechanical limits affecting pedalling efficiency (Broker 2002) and muscle fibre recruitment patterns (Savelberg *et al.* 2003), decreasing comfort, range of motion, blood flow (Schep *et al.* 2001), and visibility. Thus, balancing power production versus aerodynamic drag savings remains critical to overall track cycling performance.

Physiological demands of the team-pursuit

In the USA's preparation for the 1996 Atlanta Olympic Games, when the men's world TP record stood at 4:00.958, Broker *et al*. (1999) reported that travelling at 60-km·h^{-1} required a lead power output to be 607 W, and a mean positional power output of ~450 W. However, the now-banned 'Superman' aerodynamic position was still legal at the time (Grappe *et al.* 1997). More recently, Heimans *et al.* (2017) studied TP cyclists using modern positioning, though not necessarily more aerodynamic, and reported that lead cyclists at a velocity of approximately 56.7-km·h^{-1} (equivalent to a TP time of around 4:14 min:ss) produced a mean power output of ~507 W. The lead cyclist experiences the full effect of resistive drag forces, however drafting riders provide a slight reduction in drag force for the lead cyclist compared to riding alone (Íñiguez-De-La Torre and Íñiguez 2009). The lead rider provides drafting riders with a wake of low-pressure air, lowering air resistance and the power demands of the drafting cyclists. Fitton *et al*. (2018) demonstrated that aerodynamic drag was reduced by 4%, 42%, 48%, and 47% for positions 1-4 compared to riding alone, respectively. However, the reduction in aerodynamic drag is not directly proportional to the reduction in power demands, as inertia and technique also impact the power requirement for a given rider's velocity. Several studies have reported the power demands for TP cyclists in each position (Broker *et al.* 1999, Schumacher *et al.* 2002, Heimans *et al.* 2017, Blocken *et al.* 2018). Comparing several TP datasets, that mean power demands as a percentage of the lead rider were 64-72% at position 2, 55-64% at position 3, and 54-64% at position 4. However, these data were

collected across varying velodromes, power meters, distance between riders, rider abilities, equipment, and positioning.

The TP cyclist designated as the lead cyclist for the first lap begins from a stationary position in a timing gate that releases the wheel in response to a start signal. In elite level TP, the lead rider must produce a near-maximal effort in the first lap (first 250-m) for rapid acceleration as inertia presents the greatest resistive force initially (Martin *et al.* 2006). The other three cyclists are supported while stationary by officials. The gate starter can shift their centre of gravity to gain momentum off the start. While the elevated riders cannot shift their weight as effectively as the gate starter, they benefit from the drop in elevation from the inside to outside of the track. In subsequent laps, TP cyclists may require lead power outputs exceeding 650 W (Broker *et al.* 1999, Schumacher *et al.* 2002). Thus, competitive TP cyclists require highly adapted physiologies to produce the power outputs necessary for elite performance (Craig *et al.* 1993, Van Der Zwaard *et al.* 2018).

Team-pursuit cyclists may utilise a variety of cadences and gear ratios. Maximum reported cadences were as high as 139 RPM in 1996 (Broker *et al.* 1999), whereas in a recent 2023 UCI World Championships upload to Strava (Gee 2023) mean cadence was 113 RPM, though initial pedal strokes appeared unrecorded which would further reduce the mean cadence. The ratio between the number of teeth on the front and rear gears describes the number of rear wheel revolutions for each crank revolution. Multiplying the gear ratio by the rear wheel circumference provides meters travelled per pedal revolution. A larger gear ratio requires more torque to accelerate to a given speed from a standing start compared to a smaller gear ratio (Flyger *et al.* 2013). High torque cycling contractions, associated with larger gear ratios, rely on muscular strength (Douglas *et al.* 2021) which accelerates neuromuscular fatigue (Burnley *et al.* 2012). As the gear ratio cannot be adjusted during a TP, as would be possible on a road bicycle with derailleurs and multiple chain rings, a compromise must be made between a gear that is both tolerable from stationary and once at high racing speeds. For example, high cadence cycling requires trained muscle recruitment pattern firing and increased activation of fast-twitch muscle fibres (Wakeling *et al.* 2006), which can influence the maximum sustainable cycling intensity (Broxterman *et al.* 2015). A torque-velocity relationship that describes the optimal cadence for power production over

a duration can be used to identify appropriate gearing based on biomechanical, phenotypic, and physiological factors (Figure 2) (Douglas *et al.* 2021).

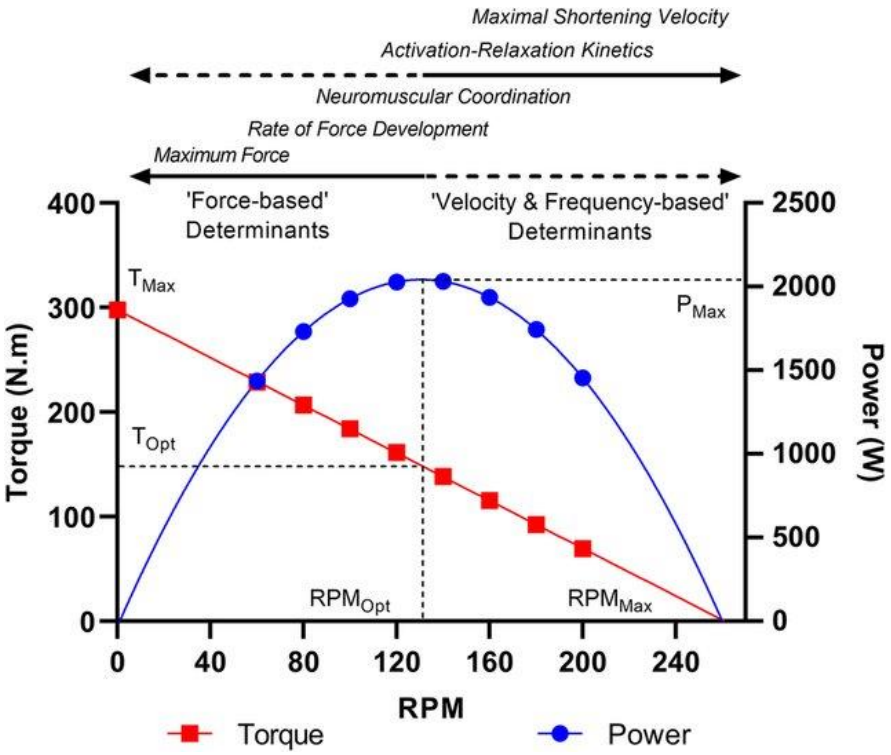


Figure 2. Torque-velocity relationship.

Abbreviations and definitions: T_{Opt}, optimal torque; T_{Max}, maximum torque; RPM_{Opt}, optimal cadence; RPM_{Max}, maximum cadence; P_{Max}, maximum power; N.m, newton meters; W, Watts. Douglas et al. 2021.

Physiological determinants of endurance performance

Exercise domains concept

Distinct metabolic domains across the intensity spectrum are bounded by key physiological thresholds that mark shifts in the predominant energy systems and metabolites (Whipp 1996, Black *et al.* 2017). These thresholds are identified via changes in physiological metrics during incremental exercise testing (Jones *et al.* 2008), and mark the transitions between moderate, heavy, severe and extreme exercise intensities. The point at which a person is unable to continue an exercise task, known as task failure, exhaustion, or fatigue, follows a characteristic curvilinear pattern across intensities (Burnley and Jones 2018). Maximal power can only be sustained for seconds (Bundle and Weyand 2012), while lower intensity exercise can be endured for hours (Davies and Thompson 1986, Lepers *et al.* 2002).

Mathematical models, especially hyperbolic ones, can accurately describe the relationship between power output and time to exhaustion (TTE) (Jones *et al.* 2010). These models demonstrate the distinct exercise intensity domains (Gaesser and Poole 1996) and a theoretical asymptotic limit to human endurance capacity (Monod and Scherrer 1965).

Moderate domain

The first domain encountered during incremental exercise is the moderate domain, which describes an "easy" relative effort or "light-intensity" training where ventilation is unlaboured (Whipp and Wasserman 1972). The physiological responses to the moderate domain are a balance in O_2 delivery and supply, baseline blood lactate and hydrogen ion (H^+) levels, indicating a surplus of aerobic and metabolic capacity (Galán-Rioja *et al.* 2020). Consequently, the muscle tissue O_2 saturation index (TSI) stabilises, reflecting an equilibrium in the ratio of oxygenated to deoxygenated haemoglobin (Ferreira *et al.* 2016, Matthews *et al.* 2023). Although the mechanisms limiting exercise in this domain are unclear due to a lack of exhaustive exercise data, studies of prolonged ultra-endurance exercise provide some insights (Davies *et al.* 1986, Lepers *et al.* 2002, Martin *et al.* 2010). Davies *et al.* (1986) showed a gradual increase in $\dot{V}O_2$ over many hours of exercise, suggesting alterations in muscle energetics and motor unit recruitment are necessary to sustain submaximal efforts. Central fatigue appears to predominate, with declines in maximal voluntary contraction force but maintained peripheral muscle function (Davies *et al.* 1986). More recent work by Black *et al.* (2017) indicates that moderate-intensity exercise can be sustained for prolonged periods with minimal changes in muscle metabolites. Research by MacPhee and colleagues (2005) demonstrated that exercise in the upper, compared to lower, region of the moderate exercise domain led to slower $\dot{V}O_2$ kinetics and a greater gain in $\dot{V}O_2$ for an increase in work rate ($\Delta\dot{V}O_2/\Delta$ work rate).

At the boundary between moderate and heavy exercise domains, there is an increased reliance on glycolytic metabolism to generate ATP (adenosine triphosphate). This transition can be defined by a specific criterion, such as a 1 $mmol \cdot L^{-1}$ increase in blood lactate concentration above baseline levels, known as lactate threshold 1 (LT1) (Coyle *et al.* 1988). Alternatively, the boundary can be identified

by the gas exchange threshold (GET), which is characterised by a disproportional increase in the ventilatory equivalents for O_2 ($\dot{V}E/\dot{V}O_2$) and CO_2 ($\dot{V}E/\dot{V}CO_2$). While O_2 uptake ($\dot{V}O_2$) increases linearly with exercise intensity, a disproportional increase in carbon dioxide output ($\dot{V}CO_2$) or ventilation ($\dot{V}E$) indicates a decrease in blood pH as bicarbonate buffers hydrogen ions. Both LT1 and GET serve as key indicators of an individual's metabolic fitness and endurance training status, as outlined by Jones (2006).

Heavy domain

The heavy-intensity exercise domain occurs between the LT1 and critical power, with exercise tolerance ranging from 40-minutes to 3-hours (Coyle *et al.* 1988). The heavy domain is characterised by sustainable homeostasis of $\dot{V}O_2$, blood lactate and pH, glycogen stores, and inorganic phosphate ion concentration [Pi] that can be maintained for up to ~30-minutes (Poole *et al.* 1988). After which, muscular efficiency decreases and there is a progressive recruitment of type II fibres, as is reflected in the $\dot{V}O_2$ slow component (Jones *et al.* 2008). Accordingly, muscle TSI can reach a steady state equilibrium in the heavy domain (Ferreira *et al.* 2016, Mathews *et al.* 2023). At the onset of heavy intensity exercise there is a transient reduction of microvascular PO_2, reflecting a greater muscle O_2 utilisation relative to muscle blood flow (McDonough *et al.* 2005).

Both central and peripheral fatigue contribute to exercise intolerance in the heavy domain. Peripheral fatigue stems from processes such as reactive O_2 species production, potassium and hydrogen ion accumulation, and glycogen depletion, which disrupt excitation-contraction coupling (Allen 2009). Glycogen availability may be especially important since depletion impairs local ATP supplies critical for calcium release and contraction (Ørtenblad *et al.* 2013). Central fatigue arises from reduced neural drive to muscles or decreased motoneuron responsiveness (McNeil *et al.* 2011), though neurotransmitters like serotonin and dopamine are unlikely involved (Nybo and Secher 2004, Meeusen *et al.* 2006). The cumulative effects of peripheral and central fatigue processes impair force production and motivate task termination, rather than a single dominant mechanism (Noakes and St Clair Gibson 2004).

The second significant threshold defines the boundary between the discrete 'heavy' and 'severe' domains and is the highest work rate permitting steady-state O_2 uptake ($\dot{V}O_2$), blood lactate, and hydrogen ions (H^+) (Whipp 1996). This critical power (CP) is derived from the power-duration relationship asymptote (Moritani *et al.* 1981). The limit of tolerance at CP is typically 25-60-minutes (Hill 1993, Poole *et al.* 2016). Given similarities, CP correlates with anaerobic threshold, the respiratory compensation point, and functional threshold power, although the measures are not equivalent (Morgan *et al.* 2019, Karsten *et al.* 2021, Poole *et al.* 2021). Kirby *et al.* (2021) demonstrated that the directionality of the TSI signal reflects sustainable versus unsustainable exercise. Specifically, a plateau or zero-slope in TSI occurs at intensities below CP, while a negative TSI slope occurs during severe-domain exercise. Current research knowledge supports the conclusion that the greatest intensity which elicits a TSI zero-slope estimates the transition from heavy to severe exercise intensity domains and the highest steady-state metabolic rate (Matthews *et al.* 2023).

Severe domain

The severe exercise domain occurs at intensities above CP, where $\dot{V}O_2$, muscle metabolism, and acid-base balance fail to reach a steady state (Poole *et al.* 1988, Jones *et al.* 2008). Exercise tolerance is less than ~60-minutes and governed by the power-duration relationship (Poole *et al.* 2016). The $\dot{V}O_2$ rises to a maximal value ($\dot{V}O_{2MAX}$) at task failure, even if the power output is below that eliciting $\dot{V}O_{2MAX}$ in an incremental test (Murgatroyd *et al.* 2011). Peripheral fatigue also develops rapidly and plateaus at task failure (Burnley *et al.* 2012).

Exercise at intensities greater than the CP results in progressive reductions of muscle PCr, pH, and glycogen stores, with increasing [Pi] (which affects sarcoplasmic reticulum calcium ion handling), lactate ion concentrations, and $\dot{V}O_2$ (Poole *et al.* 1988). These measures reliably reach their respective minimum or maximum values at the limit of tolerance (Jones *et al.* 2011), irrespective of whether the bout duration was short or long.

The progressive development of fatigue during severe-intensity exercise may be linked to the relationships between the recruitment of type II muscle fibres, loss of muscular efficiency, and changes in muscle metabolites and substrates (Jones and Vanhatalo 2017). These factors combine to produce an impaired contractile function and stimulate mitochondrial respiration; leading to the development of the $\dot{V}O_2$ slow component and attainment of the $\dot{V}O_{2MAX}$ (Burnley and Jones 2007). Attainment of $\dot{V}O_{2MAX}$ makes failure inevitable as oxidative phosphorylation plateaus. Additional motor unit recruitment then relies on finite O_2 supplies and further compounds fatigue (Burnley *et al.* 2007). Moreover, reduced motoneuron gain requires greater excitation to maintain discharge (McNeil *et al.* 2011). Ultimately, failure occurs when increased motor drive no longer compensates for loss of power and excitability (Martinez-Valdes *et al.* 2020).

Severe exercise also stresses respiratory and cardiovascular systems. Hyperventilation increases the O_2 cost of breathing and can cause respiratory muscle fatigue, while cardiovascular constraints may exacerbate muscle metabolic stress (Romer and Polkey 2008). Muscle TSI decreases consistently during severe-intensity exercise (Mathews *et al.* 2023), reaching minimum values at exhaustion (Kirby *et al.* 2021). Of note, the TP is expected to be performed predominantly at power outputs within the severe domain.

The boundary between the severe and extreme exercise domains is not well researched, yet it is typically thought of to coincide with the maximal aerobic power (MAP). The MAP indicates maximum aerobic capacity, observed with peak $\dot{V}O_2$ ($\dot{V}O_{2PEAK}$) during graded exercise (Bassett and Howley 2000). Although MAP is usually tolerable for 3-4-minutes, MAP correlates with 16.1-km TT endurance performance (Balmer *et al.* 2000).

Extreme domain

The extreme domain represents an intensity which, during constant-load cycling, leads to exhaustion before a true maximal $\dot{V}O_2$ has been achieved. It is typical for the power-duration relationship determined from performance tests in the severe domain to overpredict performance in the extreme

domain (Alexander *et al.* 2019). Similar metabolic disturbances occur in the extreme domain as the severe domain. For instance, 80% of 1 repetition maximal intensity exercise to failure decreases PCr and glycogen while increasing intramuscular lactate (Macdougall *et al.* 1999). No study has reported metabolic responses to multiple extreme exercise intensities in the same subjects, so it is unclear if PCr, Pi, and pH reach common values across extreme work rates as in the severe domain (Grassi *et al.* 2015). In the extreme exercise domain, the relationship between the inverse of TTE and intensity is linear but with a reduced slope compared to the severe domain. This observation suggests that exercise tolerance in the extreme domain may be limited by mechanisms specific to this intensity range, akin to the severe domain. Whether this represents a clear breakpoint between underlying mechanisms is unclear. More research on metabolic responses across extreme intensities is needed, particularly in the context of the TP event, since a significant portion of the TP is expected to be performed at power outputs greater than the MAP.

Work capacity above critical power (W')

The hyperbolic relationship between severe-intensity exercise and TTE has been identified and validated (Monod *et al.* 1965, Poole *et al.* 1988, Jones *et al.* 2017). An accurate power-duration relationship can be obtained by performing three to five fixed intensity trials on separate days that result in exhaustion within 2–15-minutes. The TTE for each power output is recorded and plotted to form the hyperbolic power-duration curve. It is observed that the maximum tolerable duration increases as power decreases, until an asymptote is reached (Figure 3) (Jones *et al.* 2017). This asymptote is the CP and measured in Watts (W), while the curvature constant of the power-duration relationship represents the work capacity above CP (denoted W′ and pronounced "W prime"; measured in kilojoules, kJ). Therefore, the work completed above CP during various severe-intensity exhaustive constant-work rate trials would be approximately equal when modelled (Figure 3). The relationship can also be plotted as a work-done vs time model, which presents a linear $y=mx+c$ regression equation, where y is the total work done, the slope (m) represents the CP, x is the time in s, and the intercept (c) is the W′ size. As CP reflects the maximum rate of oxidative metabolism which, when exceeded, draws upon the finite W′, it

might seem instinctive to think of the W′ as the ‘anaerobic’ capacity. However, given the close relationship between the $\dot{V}O_2$ slow component, maximal accumulated O_2 deficit (Moritani *et al.* 1981, Hill 1993), and the W′, this is likely an oversimplification (Vanhatalo *et al.* 2011). Instead, current literature does not consider CP and W′ as ‘aerobic’ and ‘anaerobic’ units, but as components of an integrated bioenergetic system (Poole *et al.* 2016).

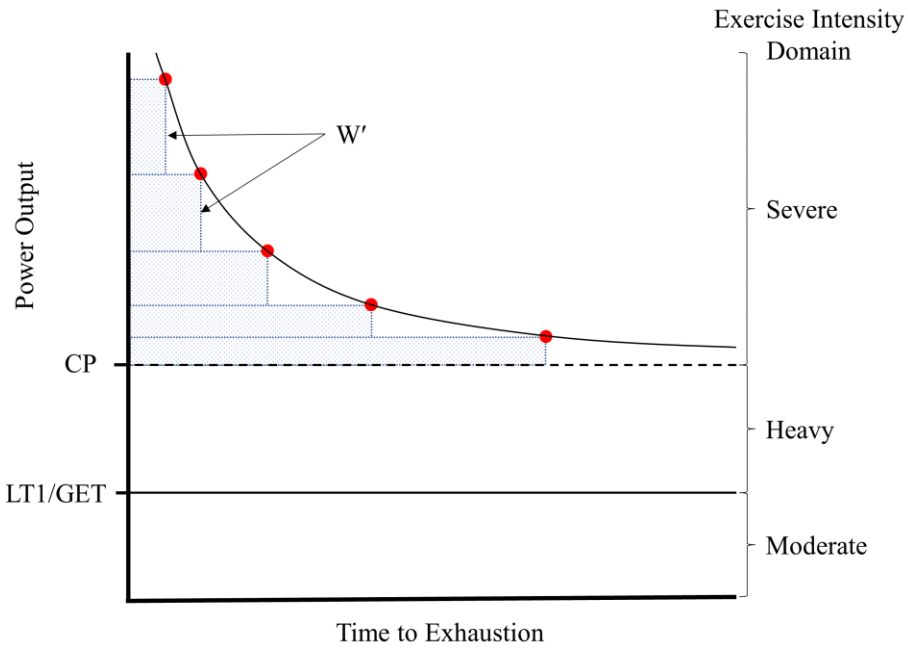


Figure 3. The hyperbolic power-duration relationship with associated exercise intensity domains.

Shaded boxes are of approximately equal area. Abbreviations and definitions: CP, critical power; W', work prime; LT1, lactate threshold 1; GET, gas exchange threshold. Adapted from Poole et al. (2016).

The underlaying physiology supports the mathematical modelling of performance. Fundamentally, the CP represents a critical metabolic rate that is provided by ‘wholly-oxidative’ energy pathways without the progressive accumulation of blood lactate or breakdown of intramuscular PCr (Poole *et al.* 2016). Therefore, it may be more appropriate to term this maximum metabolic steady state rate as ‘critical $\dot{V}O_2$', as the mechanical output at CP is a reflection of the physiological environment (Poole *et al.* 2016). For example, muscular efficiency decreases with increased pedalling cadence (Barker *et al.* 2006), such that, when power output is kept constant but cadence increases, the internal load and metabolic cost of cycling increases, and CP is reduced (Jones *et al.* 2017).

Methods of determining CP & W′

The quantification of key physiological metrics such as CP and LT1 are dependent on the specific testing protocols and conditions employed. For example, Broxterman *et al.* (2015) demonstrated that while the critical $\dot{V}O_2$ did not change when determined from either 60 RPM or 100 RPM cycling, CP was significantly different when determined using the two cadences. Valenzuela and colleagues (2022) demonstrated that the relationship between cycling performance and environmental conditions, such as temperature, follows an inverse 'U' shaped curve, with greatest performances attained in the range of ~10 to 25 °C.

Kordi *et al.* (2019) presented data suggesting that CP values are greater when determined from an upright cycling position compared to a time trial (TT) 'aero' position. However, it is important to note that this study had a relatively small sample size of just 7 participants, and 2 of these individuals exhibited higher CP estimates in the TT position compared to upright cycling. Additionally, for one participant, the CP value differed by only 4 W between positions. Therefore, while body position can influence power output and physiological metrics, the effect appears inconsistent across individuals. Moreover, there are some study design limitations that warrant consideration when interpreting the findings. The trial order was not randomised (upright cycling was always conducted first), and residual fatigue may not have been adequately controlled between tests. There was also a lack of standardisation in participant bike set-up, and flexibility or ability to rotate the hips anteriorly was not assessed. Consequently, factors such as saddle height, handlebar reach, and restricted movement may have influenced results (Holliday and Swart 2021), given that the angle of hip flexion can influence cycling metabolic cost (Faulkner and Jobling 2020). Thus, while the Kordi *et al.* study provides preliminary evidence that body position can impact CP values, the small sample size and methodological considerations suggest that further research is needed to quantify these effects more rigorously and draw more definitive conclusions.

Factors such as exercise mode, intensity, body position, and environment can all impact the parameter estimates of CP and W′. Consequently, if any of these factors vary substantially during a test or

competitive event, the resulting CP and W′ estimates may be inaccurate. For example, previous studies have shown reasonably consistent CP values derived from laboratory and field-based cycling tests of 2-15-minutes duration (Karsten *et al.* 2014, Triska *et al.* 2015). However, poor agreement exists between laboratory and field measures of W′, with higher W′ reported during laboratory tests in one study (Triska *et al.* 2015) and higher W′ reported during field-based tests in another (Karsten *et al.* 2014). Therefore field-based testing may provide more accurate estimates of W′ for cycling competitions, whereas CP appears more robust to differences between laboratory and field environments. It should be noted that both the Karsten *et al.* (2014) and Triska *et al.* (2015) studies utilised a TTE trial format in laboratory conditions but TT format in field conditions. As the authors were also essentially measuring the similarity of TTE and TT format trials, this may have influenced the results of CP and W′, where W′ may have been significantly influenced by the trial format. Interestingly, the influence of terrain for outdoor cycling demonstrates no difference between 4- and 10-minute tests for uphill or flat cycling, but significantly greater power output was produced during the uphill 1-minute test than a flat terrain 1-minute test (Hovorka *et al.* 2018).

Generally, CP testing protocols should closely match the format of an athlete's typical training and racing to enhance ecological validity and predictive ability. Some research groups have compared CP values obtained from the 'maximum mean power output' values in training and racing over a range of durations (Quod *et al.* 2010, Leo *et al.* 2020, Nimmerichter *et al.* 2020, Leo *et al.* 2021, Leo *et al.* 2022) and reported similar CP estimates to laboratory testing. However, such use of training and racing data is potentially insufficient for accurate parameter estimates as the mid-ride efforts were unlikely to be true tests to exhaustion which would be otherwise disruptive to racing or training. Use of training and racing data to estimate CP and W′ is likely to lead to underestimation of CP, W′, or both. Indeed, W′ has not shown high agreement between training and racing and laboratory estimates (Karsten *et al.* 2015, Leo *et al.* 2020).

There are several methods of estimating CP and W′, and the protocols which produce the most accurate estimates may not be practical or relevant in specific circumstances. Traditional recommendations advocate a minimum trial duration of 2-minutes in untrained subjects to ensure attainment of $\dot{V}O_{2MAX}$,

with a maximum trial duration of 10-15-minutes (Hill *et al.* 2002). However, faster $\dot{V}O_2$ kinetics in trained cyclists enables $\dot{V}O_{2MAX}$ within 90-s of maximal exercise (Withers *et al.* 1991, Caputo and Denadai 2008), suggesting the 2-minute lower limit may not apply universally. Moreover, test durations of 1-, 4-, and 10-minutes align with recent practices in high-performance track cycling literature (Bartram *et al.* 2017, Bartram *et al.* 2018, Bartram *et al.* 2021).

To reduce athlete burden, a 3-minute 'all-out' test estimates CP and W′ from a single trial (Burnley *et al.* 2006). However, 3-minute 'all-out' tests are most accurate with isokinetic ergometers, and suffer from inherent pacing impeding maximal effort and affecting parameter accuracy compared to CP and W′ derived from multiple trials (Wright *et al.* 2017). A 3-parameter CP model adds a maximal instantaneous power parameter (Hugh Morton 1996), which extends the CP and W′ model application from the upper boundary of the severe domain to 1-s at the expense of added complexity. The 3-parameter model tends to lead to lower CP estimates and higher W′ estimates compared to the 2-parameter model but produces the most accurate parameter estimates in TTE format trials (Maturana *et al.* 2018). The power-law relationship proposes that power output decays as a function of duration, avoiding fixed asymptotes (Drake *et al.* 2023); however, the power-law model currently cannot model recovery of W′ which is an important consideration for cycling performance and discussed below. An omni-domain power-duration model has been proposed that combines multiple functions to predict maximal power from seconds to hours (Puchowicz *et al.* 2020); however, its applicability to intermittent exercise remains untested. In summary, various models demonstrate strengths and limitations that warrant consideration when selecting a CP testing protocol for specific sports contexts. Further research should continue to improve approaches to balance accuracy, practicality, and application across exercise modes and contexts.

W′ reconstitution

Fatigue mechanisms are complex, and exhaustion (depleted W′) involves multiple intramuscular and systemic factors reaching critical thresholds. The physiological basis for W′ was originally thought to be substrate availability (Poole *et al.* 1988), especially glycogen and ATP, which decline by ~30% at exhaustion (Gaitanos *et al.* 1993, Rossiter *et al.* 2002). However, this hypothesis was challenged by the

finding that artificially lowering glycogen by 60-70% only decreases W′ by ~20% (Miura *et al.* 2000). In contrast, phosphocreatine (PCr) depletion is significant following high-intensity exercise (Bogdanis *et al.* 1996), and both W′ recovery and PCr resynthesis only occur at intensities less than CP (Ferguson *et al.* 2010). Indeed, the accumulation of metabolites such as Pi and H^+ during severe-intensity exercise have also been associated with fatigue and W′ depletion (Coats *et al.* 2003, Ferguson *et al.* 2010, Chidnok *et al.* 2013b, Cè *et al.* 2020, Chorley *et al.* 2020). However, W′ reconstitution is faster than both pH and PCr recovery (Tesch *et al.* 1989, Dawson *et al.* 1997, Ferguson *et al.* 2010).

Literature suggests that the key factor in W′ reconstitution is oxidative capacity, correlating with citrate synthase activity (Gaitanos *et al.* 1993, McCully *et al.* 1993) and capillary density (Tesch *et al.* 1985). As such, $\dot{V}O_2$ remains elevated post-exercise (EPOC) to assist metabolic recovery (Laforgia *et al.* 2006). The W′ reconstitution kinetics provide valuable insight into supra-CP exercise tolerance and have become a key research focus for modelling intermittent exercise capacity in sports (Lievens *et al.* 2021, Weigend *et al.* 2021, Bourgois *et al.* 2023). However, the precise physiology of W′ remains elusive and challenging to quantify. Therefore, further research is essential to refine the conceptual model relating W′ dynamics to exercise tolerance and fatigue (Skiba and Clarke 2021a).

Early CP models accounting for intermittent exercise.

In 2004, Morton and Billat introduced a theoretical model that extended the continuous CP model to intermittent protocols. Their model quantified the expenditure of W′ during work intervals above CP and the reconstitution of W′ during recovery phases below CP. The model calculated the total endurance time as the sum of complete work-rest cycles until W′ depletion. Morton and Billat's intermittent CP model avoids unrealistic infinite endurance times by respecting mathematical constraints:

$$Recovery\ Power < CP < Work\ Bout\ Power < \left(CP + \left(\frac{W'}{Work\ Bout\ Duration}\right)\right) \qquad \text{Equation 4}$$

Morton *et al.* (2004) demonstrated their model's effectiveness by achieving a good model fit to empirical running data from six athletes. Significantly lower critical velocity estimates emerged from intermittent

versus continuous runs, suggesting underlying physiological differences between intermittent and continuous testing modalities. Overall, this seminal work by Morton and Billat provided an innovative theoretical framework tailored specifically for intermittent exercise that laid the foundation for applying the concept to diverse athletic and training scenarios.

Ferguson *et al.* (2010) investigated how the duration of recovery after exhaustive exercise above CP affected the parameters of the power-duration relationship. Participants performed constant-load exercise to exhaustion at different intensities. Then, after a conditioning bout at the intensity predicted to cause exhaustion in 6-minutes, they completed additional efforts above CP following recovery periods of 2-, 6-, and 15-minutes. Ferguson and colleagues found that the extent of W′ reconstitution highly depended on the recovery duration. Shorter recovery led to smaller W′ reconstitution. For instance, W′ recovered to only 37±5% and 65±6% of control values after 2- and 6-minutes of recovery, respectively. Even after 15-minutes, W′ was still only 86±4% replenished. Therefore, Ferguson *et al.* (2010) concluded that the rate and completeness of W′ recovery are critical factors influencing intermittent exercise tolerance above CP across repeated efforts, with a depleted W′ contributing to earlier onset of fatigue.

W′ balance models

The hyperbolic relationship that describes the limit of tolerance in the severe domain is valid during efforts that are consistently within the intensity of the severe domain. However, to accurately calculate the remaining W′ (W′ balance, W'_{BAL}) at any point during a variable power workout or race, it is important to estimate how quickly W′ reconstitutes during periods when power output is below CP. Without knowing the rate that W′ recovers while exercising below CP, it becomes challenging to determine the limit of tolerance in an intermittent exercise session or competition featuring intensities both above and below CP. By estimating W′ recovery rate below CP, athletes and practitioners can better predict the total available W′, and the duration of high-intensity exercise that can be sustained before complete W′ depletion.

There is a growing body of literature investigating how the reconstitution of W′ depends on the duration and workload during recovery phases. To study the effect of recovery phase characteristics between intervals of high-intensity exercise intervals on W'_{BAL}, researchers require participants to perform intervals above CP with varying recovery periods, followed by an open-ended trial to assess the recovered work capacity (Ferguson *et al.* 2010, Chidnok *et al.* 2013a, Skiba *et al.* 2015, Bartram *et al.* 2018). The first model for tracking the depletion and reconstitution of W′ was developed by Skiba *et al.* (2012), and followed by their second model in Skiba *et al.* (2015), which proved more applicable in the context of the TP. Both Skiba's models feature an exponential recovery rate with a time constant ($\tau_{W'}$; *Tau*) describing the recovery rate. All *Tau* estimation methods rely on the premise that a change in available W′ between a conditioning bout and the open-ended trial must be due to recovery between the efforts (Skiba *et al.* 2015). Indeed, research has demonstrated the exponential recovery kinetics of W′ (Ferguson *et al.* 2010, Bartram *et al.* 2018). Chidnok *et al.* (2013a) modelled the metabolic recovery kinetics using ^{31}P-magnetic resonance spectroscopy and reported that increased recovery duration enabled a delay in attaining critical values of PCr, ADP, Pi, and H^+, thus prolonging TTE. In other words, work done above CP increased as recovery periods were extended (Chidnok *et al.* 2013a). This work supports earlier suggestions that W′ is surrogate measure for physiological measures such as metabolite accumulation and substrate depletion (Ferguson *et al.* 2010).

Skiba 1

The initial W'_{BAL} equation by Skiba *et al.* (2012) used an integral method to calculate the remaining W′ at any moment. The calculation was based on the cumulative W′ expended and an assumed exponential W′ reconstitution kinetics,

$$W'_{bal} = W' - \int_0^t \left(W'_{exp}\right)\left(e^{-\frac{t-u}{\tau W'}}\right) \qquad \text{Equation 5}$$

Where W'_{exp} is equal to the expended W′, $t - u$ is equal to the time in s between measurement segments (usually 1 Hz), and $\tau W'$ is the time constant of W′ reconstitution. The magnitude of W′ remaining at any timepoint is equal to the difference between the known W′ and the total sum of W′ expended before

time *t* in the workout, each Joule of which is being recharged exponentially during recovery below CP. Specifically, the integral term summed the W′ expended in above CP efforts up to a time point, with each term decaying exponentially during any below CP recovery periods. The exponential decay was governed by a time constant *Tau* (τW′) that dictated the recovery rate. While innovative, Skiba's original integral model was limited because W'_{BAL} could only be resolved retrospectively, and the analysis was highly specific to the combination of work and recovery intervals performed.

Skiba 2

In 2015, Skiba *et al.* published a differential model for W'_{BAL}

$$W'_{BAL} = \begin{cases} W'_{BAL,i-1} - \left((P_i - CP) \cdot \Delta u_i\right), P_i > CP \\ W'_0 - W'_{expended} \cdot \left(e^{-\frac{\Delta u_i}{\tau W'}}\right), P_i < CP \end{cases} \quad \text{Equation 6}$$

where *i* is the i^{th} segment of the total time subdivided into n segments at 1 Hz (Δu), and P_i is the mean power output for segment *i.* Thus, W'_{BAL} is calculated sequentially and $W'_{BAL,i-1}$ represents a preceding estimation of W'_{BAL}. $W'_{expended}$ is the quantity of depleted W′ at *i*-1.

In Skiba 2, the recovery rate *Tau* is calculated as an exponential relative to the recovering work rate and the overall W′ size (Skiba *et al.* 2015) (Equation 6). Meaning, the further one exercises beneath their CP, the faster the recovery time constant, and the greater the rate of W′ reconstitution. The W′ reconstitution time constant (τW′) showed a curvilinear relationship with the recovery intensity, which was determined in absolute terms as the delta to CP (D_{CP})

$$\tau W' = \frac{W'}{D_{CP}} \quad \text{Equation 7}$$

When incorporated into the differential W'_{BAL} equation, the Skiba 2 *Tau* adjustment permitted a prediction of W′ depletion during intermittent efforts of varying intensities and durations within a group of ten recreational participants (Skiba *et al.* 2015). However, as aerobic adaptations improve oxidative

capacity and metabolite clearance, the recovery rate *Tau* is expected to be relative to the aerobic capacity and individual (Skiba *et al.* 2014a), not just the size of the W′ as the Skiba 2 *Tau* adjustment would suggest.

Bartram adjustments

Bartram *et al*. (2018) evaluated the Skiba W'_{BAL} model in elite track cyclists during a training camp. They introduced ten sessions consisting of three high-intensity intervals. The first two intervals were designed to deplete W′ by ~30%, with 60-s recovery periods at various predetermined power outputs ranging from CP (D_{CP} 0) to 200 W below CP (D_{CP} 200) (Figure 4). In the third interval, a TTE trial was completed. Instead of using CP and W′ values from laboratory tests, the authors utilised the athletes' individual CP and W′ values derived from training and racing data. At D_{CP} 0, W′ would theoretically remain stable during recovery at CP. The authors created a metric, $W'_{TRAINING}$, as the W'_{BAL} value at exhaustion during the D_{CP} 0 trial, indicating the usable W′ capacity under the specific trial conditions. They reasoned this was necessary since the trials were completed during a training camp rather than typical exhaustive tests.

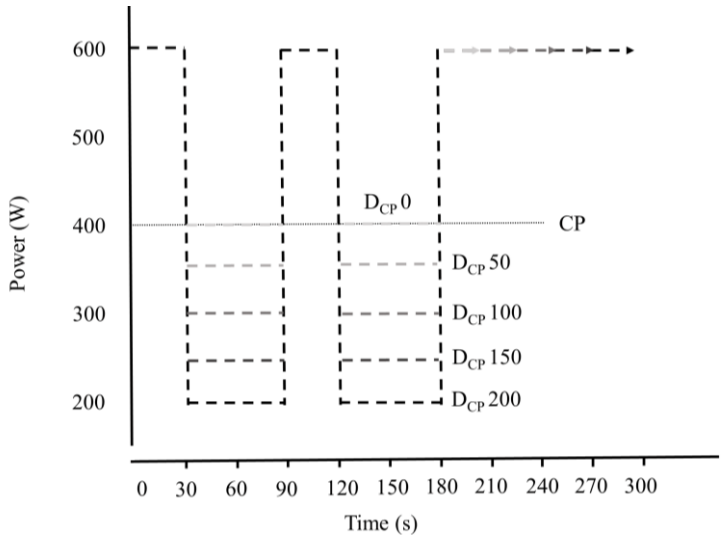


Figure 4. Protocol to determine rates of W′ reconstitution in TP cyclists.

The final interval is an open-ended trial to volitional exhaustion.
Abbreviations and definitions: W, Watts; s, seconds; D_{CP} = Absolute Watts beneath critical power (CP). Adapted from Bartram et al. (2018)

With $W'_{TRAINING}$ representing the limit of tolerance, the time constant of W′ reconstitution (*Tau*) was adjusted for each trial so that W'_{BAL} at exhaustion equalled $W'_{TRAINING}$. The regression of *Tau* across several intensities enables prediction of rate of W′ reconstitution at any intensity below CP. Bartram *et al*. (2018) determined a population-specific equation for *Tau* in elite Australian track cyclists

$$\tau W' = 2287.2 * D_{CP}{}^{-0.688} \quad \text{Equation 8}$$

and compared this to the original Skiba *et al*. (2015) model (Figure 5). They concluded the Skiba *et al*. model substantially underestimates W′ reconstitution rates in elite cyclists, indicating recovery kinetics are highly individualised and dependent on training status. Both Skiba *et al*. (2015) and Bartram *et al*. (2018) models predict that *Tau* increases as intensity approached CP. In a follow-up study, Bartram *et al*. (2021) applied generalised *Tau* values to TP W'_{BAL} tracking. They reported mean recovery intensity of ~79 W below CP (D_{CP} 79). Although races typically feature recoveries within 50 W of CP, no protocols to date have assessed recovery within 50 W of CP (i.e., D_{CP} <50). Yet, Figure 5 illustrates that recovery at D_{CP} <50 is an area where a small change in D_{CP} can have a large effect on the time constant due to the exponential nature of the relationship. Therefore, whilst research has demonstrated the importance of customised W′ recovery protocols, W′ reconstitution kinetics near CP remain insufficiently explored for track cycling race modelling and may constitute a key performance determinant. The unexplored regions of D_{CP} <50 feature the exponential aspect of the *Tau* relationship, and thus, should be examined in greater detail in future research. Lievens *et al*. (2021) presented data demonstrating that the exponential region may occur within the heavy intensity domain, with further reductions in intensity within the moderate domain resulting in diminishing improvements in recovery rate.

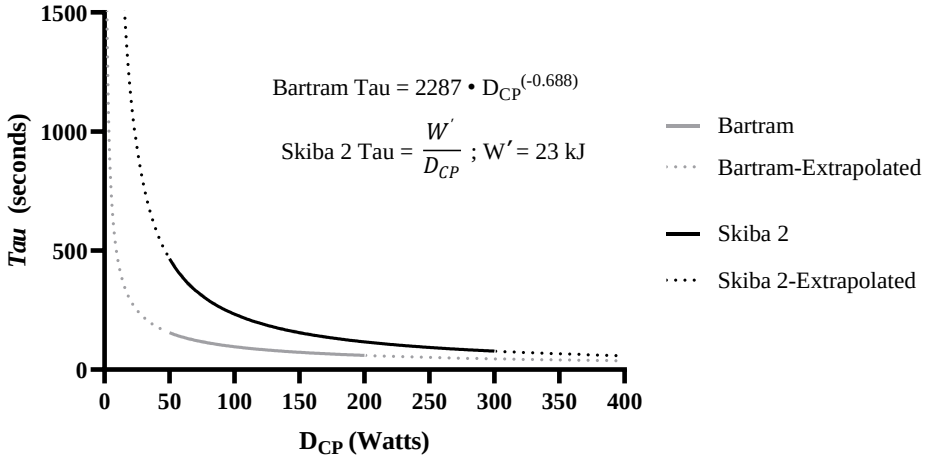


Figure 5. Comparison of recovery rate time constant adjustments.

Note that the W′ value of 23 kJ for the Skiba 2 Tau is the mean W′ size reported by Bartram et al. (2018) in elite TP cyclists. Abbreviations and definitions: W′, finite work capacity; Tau, time constant of the rate of W′ reconstitution; D_{CP}, difference between recovery intensity and critical power (CP); W, Watts; s, seconds

A 2-component model of recovery

Recent studies by Chorley *et al.* (2023) and Caen *et al.* (2021) modelled W′ reconstitution kinetics following exhaustive exercise across a range of recovery durations. Both studies recruited trained male cyclists to perform repeated bouts of high-intensity cycling with recovery phases ranging from 30-s to 15-minutes. In both studies, W′ reconstitution was calculated after each recovery duration and mathematically modelled using exponential functions. The biexponential model provided the best fit to the data in both cases, exhibiting a fast initial recovery phase and slower second phase. The rapid initial phase was attributed to enhanced aerobic contribution from changing $\dot{V}O_2$ kinetics and phosphocreatine resynthesis by Caen *et al.* (2021). Chorley *et al.* (2023), related the biexponential time constants to the recovery intensity relative to CP. Both studies found W′ reconstitution was strongly influenced by aerobic fitness, with higher $\dot{V}O_{2PEAK}$ associated with greater W′ reconstitution over time. Therefore, these two studies demonstrated that W′ recovery follows a biphasic exponential time course dependent on recovery duration and aerobic capacity. Additionally, the reconstitution of the W′ slows with repeated maximal exercise (Caen *et al.* 2019, Chorley *et al.* 2019, Lievens *et al.* 2021).

Limitations of current W'_{BAL} models

Though the W'_{BAL} model shows promise for understanding pacing strategies and performance in endurance cycling, there remain several key limitations in current approaches that warrant further investigation. A primary constraint is the reliance on fixed CP and W′ estimates, which fail to account for within-athlete fluctuations in these parameters that may arise from factors like pedalling frequency (cadence) (Barker *et al.* 2006), positional shifts on the bike (Faulkner *et al.* 2020), and fatigue effects over the course of a race (Stevenson *et al.* 2022, Stevenson *et al.* 2023). Dynamically updating CP and W′ could provide greater precision but requires developing methods to continually assess these values in real-time.

While measuring W'_{BAL} during exercise has not been demonstrated in literature, some evidence exists to suggest that proxies may be used to indicate proximity to exhaustion which may be suitable for use within competition, such as muscular tissue O_2 saturation index (TSI). A recent study of the tibialis anterior muscle found an increase in oxidative capacity and O_2 supply from distal to proximal, suggesting gradients in mitochondrial function drive phosphocreatine recovery rather than fibre type or exercise mode (Heskamp *et al.* 2021). However, more research into the specifics of how TSI can reflect W'_{BAL} values in real time or the proximity to exhaustion is warranted. There is also evidence that heart rate variability (Fleitas-Paniagua *et al.* 2022) and TSI can be used to denote the boundary between heavy and severe exercise domains (Kaufmann *et al.* 2023), but needs further evaluation to demonstrate the precision necessary for fluctuations in domain boundary determination during exercise.

Adaptations to endurance training

Endurance training of sufficient volume and intensity can induce several systemic and cellular adaptations that enable exceptional aerobic capacities in elite athletes (Furrer *et al.* 2023). Centrally, respiratory muscles increase their strength and endurance (Wagner 2000), permitting greater maximal ventilation. Cardiac remodelling leads to increased left ventricular internal diameter, wall thickness, and mass (Degens *et al.* 2019), which manifests in heightened resting and maximal stroke volumes, owing to greater end-diastolic volume and contractility (Zhou *et al.* 2001). During intense exercise,

stroke volume continues increasing until exhaustion, enabling maximal cardiac outputs reaching 40 L·minute^{-1}, compared to 20 L·minute^{-1} in untrained individuals (Ekblom and Hermansen 1968). Total blood and haemoglobin mass can be trained to increase by up to 20% (Garvican *et al.* 2010), enhancing O_2 carrying capacity. Plasma volume also increases, expanding the vascular space (Convertino 1991).

Within active muscles, endurance training can increase capillary density by up to 50% (Andersen and Henriksson 1977), reducing diffusion distances while permitting greater blood flow. The enhanced vascularisation through angiogenesis enables the increased systemic O_2 extraction observed in endurance athletes (Richardson *et al.* 1999). Angiogenesis is the synthesis of new capillaries from existing vasculature (Olfert *et al.* 2016). The capillaries form an integral part of the peripheral microvasculature, which deliver O_2 and remove metabolites from tissues (Olfert *et al.* 2016). Therefore, skeletal muscle capillary density is well correlated with improved endurance and aerobic capacity (Hudlicka *et al.* 1992, Mitchell *et al.* 2018, Hellsten and Gliemann 2023). Generally, a greater capillary surface area permits increased O_2 delivery and greater clearance of metabolites.

Angiogenesis is stimulated by several factors arising during exercise, including reduced O_2 tension in local tissue (hypoxia), metabolite accumulation (Olesen *et al.* 2010), vascular sheer stress (Prior *et al.* 2004), and muscle stretch (Egginton 2011). These conditions stimulate endothelial and muscle cells to upregulate gene expression for capillary growth (Olfert *et al.* 2016). The primary mechanism for angiogenesis is the upregulation of vascular endothelial growth factor (VEGF), mediated by nitric oxide (NO) (Benoit *et al.* 1999) and hypoxia inducible factor-1α (HIF-1α) (Egginton 2009). Inhibiting HIF-1α prevents hypoxic signalling in vitro, demonstrating its significance (Toffoli *et al.* 2009). During exercise, tissue perfusion increases 100-fold over resting levels (Andersen and Saltin 1985), and the resulting shear stress on capillary endothelial cells stimulates angiogenesis (Hudlicka *et al.* 1992).

Because of the association between endurance performance and capillary density, it is unsurprising that inducing angiogenesis may be a target for those aiming to enhance endurance performance. Indeed, a range of exercise training modalities have been shown to stimulate angiogenesis, including LIT (Cocks *et al.* 2013, Hoier *et al.* 2013), HIIT (Jensen *et al.* 2004), and SIT (Cocks *et al.* 2013, Bonafiglia *et al.* 2017). A recent meta-analysis demonstrated that exercise training increased skeletal muscle capillary

density (Liu *et al.* 2022). Compared to LIT (<50% $\dot{V}O_{2MAX}$), moderate-intensity continuous training (50-80% $\dot{V}O_{2MAX}$) resulted in a 21% greater increase in capillaries per muscle fibre, while HIIT (80-100% $\dot{V}O_{2MAX}$) elicited a 54% greater increase in sedentary individuals. However, Liu and colleagues found that well-trained subjects did not exhibit further increases in capillarisation with various types of training interventions. Therefore, the literature suggests that, while exercise training can potently stimulate angiogenesis in untrained skeletal muscle, this adaptation may plateau in already well-trained populations, potentially requiring supplementary stimuli to elicit an angiogenic response.

Further adaptations occur within the muscle fibres, including a shift towards slow-twitch oxidative type I fibres (Howald *et al.* 1985) and robust mitochondrial biogenesis (Holloszy 1967). Mitochondrial volume density can reach ~9% in endurance athletes, double that of untrained individuals (Hoppeler *et al.* 1973), while mitochondrial oxidative capacity and efficiency are also improved in endurance athletes (Larsen *et al.* 2012). The greater mitochondrial content, alongside elevated myoglobin stores, provides an expanded capacity for aerobic ATP production. Glycogen and intramuscular lipid levels also increase, enhancing substrate availability (Goodpaster *et al.* 2001). Additionally, endurance training has been shown to increase total lipid oxidation during exercise by 24%, accompanied by upregulated gene expression of FAT/CD36 and CPT1 involved in cellular fat uptake and oxidation (Tunstall *et al.* 2002).

Additional muscle changes include tendon collagen protein synthesis (Miller *et al.* 2005), which improves elastic energy storage and return. Resistance to fatigue is also heightened through changes in membrane excitability and ion handling (Overgaard *et al.* 2004). Together, the systemic, vascular, metabolic, and mechanical adaptations within O_2 transport, delivery, and utilisation pathways permit the high $\dot{V}O_{2MAX}$ of elite endurance athletes, reaching 80-96 $mL \cdot kg^{-1} \cdot minute^{-1}$ (Joyner *et al.* 2008). This comprehensive restructuring and remodelling may culminate to an exceptional aerobic capacity in highly-trained endurance athletes.

High-intensity interval training

High-intensity interval training (HIIT) involves repeated bouts of high-intensity exercise interspersed with recovery periods. The high-intensity work bouts range from 80-100% of maximal heart rate or over 90% of maximal O_2 uptake ($\dot{V}O_{2MAX}$), while recovery periods allow partial recovery (Laursen *et al.* 2002a, Buchheit and Laursen 2013a). This intense stop-and-go format provides a potent physiological stimulus. During high-intensity exercise, ATP is primarily derived from anaerobic glycolysis, resulting in rapid glycogen depletion, intramuscular acidosis from lactate and H^+ ion accumulation, and PCr depletion (Wadley *et al.* 2016). There is also increased generation of reactive O_2 species and oxidative stress (Gibala *et al.* 2012). The repeated perturbations to homeostasis during HIIT drive mitochondrial biogenesis, up-regulation of muscle antioxidant defence, enhanced microvascular filtration capacity, and cardiovascular adaptations (Daussin *et al.* 2008a, Little *et al.* 2010, Milanović *et al.* 2015, MacInnis and Gibala 2017). Specifically, signalling through pathways involving AMPK, p38 MAPK, PGC-1α and p53 are robustly activated by HIIT, triggering increased mitochondrial proteins, oxidative enzymes like citrate synthase and cytochrome oxidase (COX), and upregulation of antioxidant enzymes (Gibala *et al.* 2006, Little *et al.* 2011). Peripheral vascular adaptations also occur, with increased arteriovenous O_2 difference and capillary density after a period of HIIT (Daussin *et al.* 2008a, Daussin *et al.* 2008b).

For recreational athletes and individuals looking to improve fitness with limited time, HIIT is an effective and time-efficient strategy to rapidly enhance $\dot{V}O_{2MAX}$ and aerobic performance compared to moderate-intensity continuous training (Helgerud *et al.* 2007, Buchheit *et al.* 2013a). Typical HIIT protocols for recreationally active or untrained populations generally utilise work intervals of 30-s to 4-minutes repeated 4-6 times, with intensities over 90% of heart rate max or $\dot{V}O_{2MAX}$ and work:recovery ratios of 1:1 to 1:3 (Buchheit *et al.* 2013a). This prescription allows individuals to gain rapid fitness benefits and cardiometabolic health improvements with a low total training volume and time commitment. For example, HIIT is often less than 25% of the training duration compared to traditional continuous endurance training (Buchheit *et al.* 2013a, Jung *et al.* 2014).

For trained athletes ranging from highly competitive recreational athletes to elite professionals, HIIT provides an efficient training stimulus for enhancing $\dot{V}O_{2MAX}$, economy, lactate threshold, and sustainable power output (Laursen *et al.* 2002a). In trained runners and cyclists, improvements averaging 5-15% in $\dot{V}O_{2MAX}$ have been documented over interventions lasting 2-8-weeks (Laursen *et al.* 2002a). The high intensity exercise bouts stimulate muscular and cardiovascular adaptations that increase $\dot{V}O_{2MAX}$, the fraction of $\dot{V}O_{2MAX}$ that can be sustained, and the lactate threshold which translates to improved endurance performance (Laursen *et al.* 2002a). For athletes involved in competitive events, sport-specific HIIT designs utilising maximal sprint, power, or speed intervals can provide further event-specific conditioning and performance benefits (Buchheit *et al.* 2012). However, the extreme demands of HIIT training cycles can lead athletes to non-functional overreaching if sufficient recovery periods are not built into the periodised training program (Billat *et al.* 1999). Athletes focused on HIIT must balance it appropriately with other training priorities such as skills, tactics, strength, and speed development within a periodised structure.

For elite athletes who are already highly-trained and approaching their physiological 'ceilings', intensifying HIIT protocols can be an effective way to continue providing a sufficient training stimulus for further adaptations (Laursen 2010). While traditional HIIT sessions with 3-5 minute intervals still have their place, as athletes reach elite levels, incorporating shorter intervals of 10-60 seconds, uphill sprints, heavy resistance, or hypoxic exposure into HIIT protocols can make the demands more challenging (Laursen 2010, Rønnestad *et al.* 2014a, Rønnestad *et al.* 2020). The key is to employ a periodised approach, strategically varying the HIIT protocols to continually apply overload stimuli, while accounting for the principle of specificity (van Erp *et al.* 2021). However, the line between optimal adaptation and non-functional overreaching becomes finer for elite athletes. Small potential performance gains from highly demanding HIIT must be carefully weighed against the accompanying residual fatigue, increased injury risk, risk of overreaching, and motivational burnout that can arise from excessively strenuous intervals (Solli *et al.* 2017).

Periodisation

Periodisation structures sequential training phases or blocks to maximise adaptation and performance (Stone *et al.* 2021). The traditional model of periodisation builds aerobic capacity through an initial period of high-volume, low-intensity training before shifting emphasis towards race-specific abilities through more high-intensity training (Figure 6) (Krüger 2016, González-Ravé *et al.* 2021). Traditional periodisation has been widely used in endurance sports like swimming, running, and cycling (Casado *et al.* 2022). However, block models often utilise concentrated training doses by alternating focused blocks of low-, moderate-, and high-intensity exercise (Issurin 2010).

For cycling specifically, a recent review (Galán-Rioja *et al.* 2023) found traditional models over 8-12 weeks improved $\dot{V}O_{2MAX}$, lactate threshold and TT performance in trained cyclists. However, block models lasting 1-8 weeks led to similar or slightly greater improvements in these measures (Rønnestad *et al.* 2014b, Hebisz *et al.* 2021). The concentrated overload of block periodisation may provide greater benefit for already well-trained athletes by optimising adaptation between concentrated training doses (Mølmen *et al.* 2019). Nevertheless, evidence suggests both approaches can benefit endurance athletes if properly integrated within the training programme (Kiely 2018). The optimal model likely depends on the training phase, cycling discipline, and individual athlete. Frequent testing and adjustments are key to determine ideal sequencing of training intensities through sound program design. While periodisation can enhance performance in cyclists, continued research is needed on integrating periodisation principles into long-term training progression.

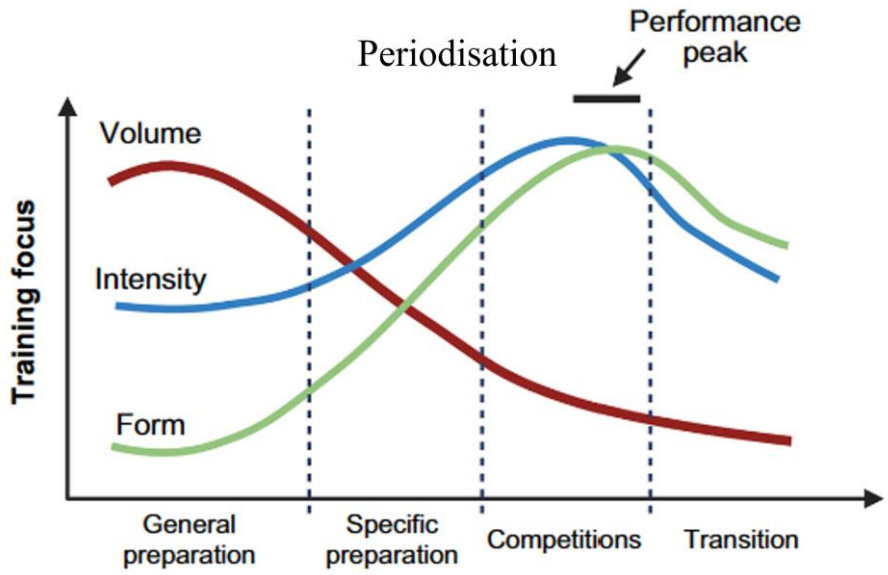


Figure 6. Periodisation through a training cycle.

Adapted from Furrer et al. (2023).

Polarised training

The polarised model distributes most training time (~80-90%) at low intensities below the first ventilatory or lactate threshold, with the remaining time near maximal intensities (Seiler 2010). Proponents argue this approach provides an optimal stimulus-recovery balance by emphasising recovery while including intense intervals for $\dot{V}O_{2MAX}$ and economy (Foster *et al.* 2022). Evidence shows polarised programs can improve aerobic capacity and endurance performance compared to threshold-focused models in trained runners, cyclists, and triathletes (Neal *et al.* 2013, Muñoz *et al.* 2014). The proposed benefits include enhanced mitochondrial biogenesis and capillary density from extensive low-intensity training, along with improved $\dot{V}O_{2MAX}$, economy, and lactate tolerance through optimised high-intensity sessions (Seiler and Tønnessen 2009).

Research indicates successful high-performance athletes tend to adopt a polarised '75-5-20' training structure, with ~75% of total volume at intensities below lactate threshold, ~20% near $\dot{V}O_{2MAX}$, and just ~5% at moderate intensities near lactate threshold (Figure 7) (Fiskerstrand and Seiler 2004). However, critics contend increasing duration at intensities near $\dot{V}O_{2MAX}$ and lactate threshold through a pyramidal approach may better increase oxidative capacity and endurance-specific fitness (Burnley *et al.* 2022).

The critics' position is that lactate threshold training offers a key overload stimulus, so underemphasising this intensity zone may hinder adaptation (Esteve-Lanao *et al.* 2007).

The ideal approach likely depends on the individual athlete, discipline, training phase, and competition demands (Kiely 2018). While polarised models have proven effective for various endurance athletes, integrating polarised concepts within properly sequenced training cycles seems most beneficial. More research is still needed comparing polarised and alternative intensity distributions across diverse athletic populations.

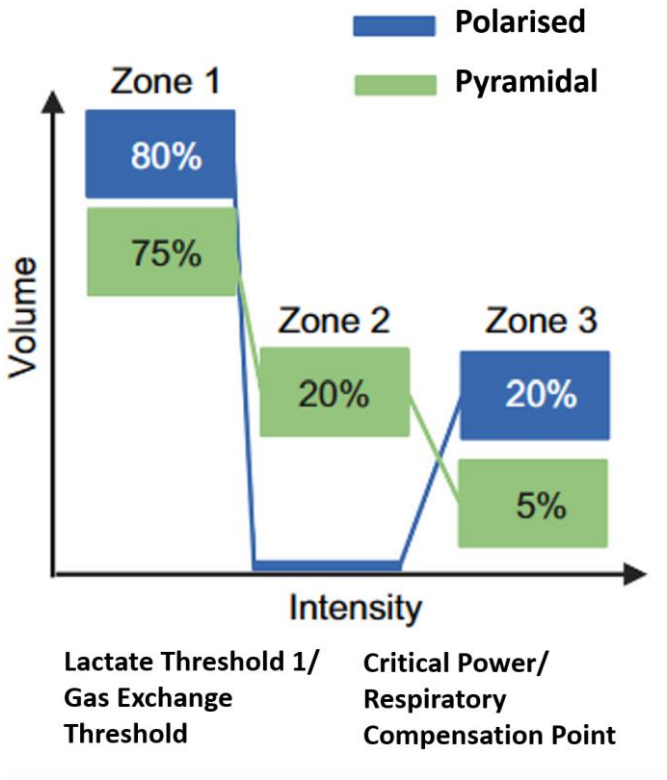


Figure 7. The principle of a polarised vs pyramidal training programme in respect to volume and intensity. Adapted from Furrer et al. (2023).

Reduced adaptive response with increased training status

Despite the previous evidence demonstrating the effects of exercise interventions to augment the factors of performance, once an individual possesses a highly adapted physiology it is challenging to gain additional adaptations (Laursen *et al.* 2002a). Indeed, there is evidence demonstrating decreased

magnitude of physiological adaptations with increased training status (Hoppeler *et al.* 1985). For example, Saltin *et al.* (1977) presented evidence to suggest that large improvements in $\dot{V}O_{2MAX}$, capillarisation, and mitochondrial content observed within the first few weeks of an exercise intervention are gradually attenuated as non-progressive exercise continues for further months. Performance adaptations from high-intensity interval training (HIIT) can reach a plateau after 8-12 sessions (Paton *et al.* 2005) or ~3-weeks of training (Norrbom *et al.* 2022) without progressive overload of intensity or duration.

A reduced amplitude of adaptive remodelling is also demonstrated at the molecular level, where Perry *et al.* (2010) demonstrated that a decreased magnitude of PGC-1α mRNA expression followed subsequent training sessions in a 14-day period (Figure 8). A similar blunting of adaptative responses has been observed in angiogenic factors. Where Richardson *et al.* (2000) observed a reduced expression of VEGF mRNA following 8-weeks of endurance training. In addition, increases in traditional endurance training volume have been demonstrated to be inadequate in producing further aerobic capacity enhancements in trained runners (Daniels *et al.* 1978). Moreover, while HIIT is known to stimulate mitochondrial biogenesis in healthy untrained individuals (Laursen *et al.* 2002a), 2 weeks of HIIT was shown to be insufficient to enhance mitochondrial enzyme activity in well-trained cyclists ($\dot{V}O_{2PEAK}$ >60 $mL \cdot kg^{-1} \cdot minute^{-1}$) (Perry *et al.* 2010).

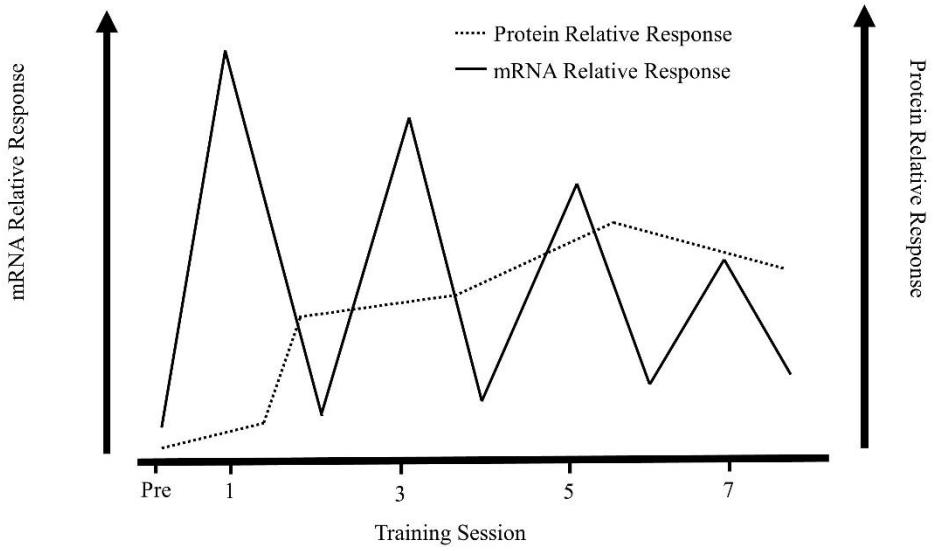


Figure 8. The responses and relationship of PGC-1α mRNA and PGC-1α protein activity throughout 2-weeks of high-intensity interval training.

Adapted from Perry et al. (2010)

Therefore, it is necessary to develop training methods for highly adapted athletes that continue to stimulate the driving mechanisms of endurance adaptation. Highly trained athletes often have high training volumes where additional submaximal training has little effective on improving performance (Laursen *et al.* 2002a). However, small improvements for these athletes can lead to success in competition (Paton and Hopkins 2001, Midgley *et al.* 2006). Previous studies have demonstrated that training prescriptions that include intervals at $\dot{V}O_{2MAX}$ can enhance the $\dot{V}O_{2MAX}$ in elite runners (Billat *et al.* 2002, Smith *et al.* 2003); whereas, Rønnestad (2020) failed to improve measures of aerobic capacity in elite cyclists with 5-minute intervals of ~79% MAP. Thus, literature supports training at sufficient intensity for continued aerobic capacity enhancements in elite athletes.

Although endurance performance may increase following HIIT interventions in endurance athletes, previous literature often demonstrates that not all of the classic determinants of endurance capacity improve concurrently, such as $\dot{V}O_{2MAX}$ (Laursen *et al.* 2002b), work economy (Rønnestad *et al.* 2014a), fractional utilisation of $\dot{V}O_{2MAX}$ at lactate threshold (LT) (Helgerud *et al.* 2007), and maximal lactate steady state (MLSS) intensity (Laursen *et al.* 2005). It has been previously demonstrated that more intense intervals improve 40-minute cycling performance more so than longer intervals in well-trained

(Rønnestad *et al.* 2014a) and elite cyclists (Rønnestad *et al.* 2020), demonstrating aerobic adaptations to at least some of the endurance determinants. To test the effects of high-intensity, short duration intervals in comparison to moderate-intensity, long duration intervals, Rønnestad *et al*. (2020) recruited elite road and mountain bike cyclists and assigned them to either a SIT or moderate-intensity training (MIT) group. In the three weeks prior to the commencement of the study, all participants focussed on low-intensity, high-volume training (LIT), with less than 8% of this training above low-intensity. The participants were assigned to either a short (30-s) or long (5-minute) interval group. A work:recovery ratio of 2:1 and total work duration of ~20-minutes was employed within both groups. Participants performed the highest sustainable effort possible for each interval, and recovery power was set at 50% of the working power output. The results demonstrated that while the perceived effort of each interval was similar (~17.5 RPE: 6-20 Borg scale), the absolute intensity of each HIIT interval was ~20% higher in the short interval group (441 ± 31 W vs 368 ± 35 W; ~94% vs ~79% of MAP). In terms of performance, Rønnestad *et al*. (2020) reported greater improvements in: $\dot{V}O_{2MAX}$, MAP, power output at blood [lactate] = 4 mmol, fractional utilisation of $\dot{V}O_{2MAX}$ at blood [lactate] = 4 mmol, and 20-minute TT performance within the short interval group compared to the long interval group. The authors concluded that short intervals enhanced endurance performance and its determining factors by a greater degree than long intervals, despite similar work durations. In contrast to similar studies, which matched total work done between conditions of differing duration and intensity, Rønnestad *et al.* (2020) used perceived effort to control for differences in the training protocol, which is suggested to be more realistic in terms of real-world training (Seiler *et al.* 2013).

Stepto and colleagues (1999) explored the effect of different work bout intensities and work:recovery ratios in HIIT affect performance adaptations. They randomly assigned 20 male endurance trained cyclists to one of five HIIT groups with varying interval durations (30-s to 8-minutes) and intensities (80-175% MAP). The 30-s (175% MAP; 1:9 work:recovery ratio) and 4-minute (85% MAP, 4:1.5 ratio) groups improved 40-km TT speed, but the other groups did not. The 1-minute (100% MAP, 1:4 ratio) and 4-minute (85% MAP, 4:1.5 ratio) HIIT formats increased MAP, while the other groups showed no improvements. These results highlight that multiple mechanisms influence how HIIT enhances

performance and that various HIIT formats can elicit similar outcomes. However, comparisons between studies are problematic due to diverse work:recovery ratios and work bout intensities used.

Given the positive findings for 30-s work bouts and the principle of specificity, HIIT designed for enhancing elite TP performance should feature short duration (30-s), high-intensity work bouts (at lead TP intensity) to have the greatest potential for enhancing TP performance. Furthermore, considering individual variability in recovery rate between HIIT bouts, a range of work:recovery ratios may lead to enhanced performance depending on the physiological characteristics of the individual. However, the literature demonstrates endurance performance enhancements from HIIT work:recovery ratios between 1:1 to 1:9.

Blood flow restriction

Blood flow restriction (BFR) training involves exercising with partially restricted blood flow, which is typically achieved by applying a pneumatic cuff or tourniquet to the proximal portion of a limb (Patterson *et al.* 2019). The degree of occlusion from BFR is dependent on several factors including cuff pressure, cuff width, and limb diameter (Loenneke *et al.* 2012). While there is evidence suggesting beneficial performance adaptations in healthy males from applying BFR without engaging in exercise (Jones *et al.* 2015), applying BFR within an exercise session creates a unique stress environment in the active muscles that appears to augment adaptations to resistance and endurance exercise versus normal blood flow conditions (Pignanelli *et al.* 2021). The mechanisms likely involve increased fast-twitch fibre recruitment, metabolite accumulation, fluctuations in oxygenation, and vascular shear stress during BFR exercise (Ferguson *et al.* 2021).

During BFR exercise, the restricted blood flow and O_2 delivery to the working muscles is proposed to create an acute state of metabolic stress. This increased metabolic stress is thought to increase the reliance on anaerobic metabolism to maintain ATP production, accelerating glycolysis, phosphocreatine breakdown, and accumulation of metabolites like lactate, H^+, and Pi (Pignanelli *et al.* 2021). The heightened fast-twitch fibre recruitment with BFR resistance exercise is demonstrated by

increased electromyography (EMG) signal of fast-twitch fibres during low-load resistance exercise with BFR compared to normal blood flow (Takarada *et al.* 2000, Moore *et al.* 2004). Other acute studies indicate increased fast-twitch fibre activation through heightened glycogen depletion (Suga *et al.* 2009) and greater PCr breakdown (Suga *et al.* 2010) in type II fibres after BFR exercise.

The metabolite accumulation is evidenced by greater blood lactate levels following low-load BFR resistance exercise versus low-load normal blood flow resistance exercise (Kim *et al.* 2017). Upon release of the tourniquet during recovery intervals of BFR exercise, reactive hyperaemia occurs leading to rapid increases in limb blood flow and a state of high shear stress acting on the endothelium (Pignanelli *et al.* 2021). The oscillations of muscle hypoxia during BFR exercise and normoxic reperfusion upon tourniquet release provides a distinct pattern of stress and recovery, which may function as mechanotransductive signals (Burkholder 2007). For example, Ferguson *et al.* (2018) demonstrated an increase of VEGF and PGC-1α mRNA expression following low-load resistance exercise with BFR, which may be induced by ischaemic and shear stress stimuli.

A proposed mechanism for muscle hypertrophy with BFR training is that the accentuated metabolic stress and local hypoxia during BFR exercise augments the typical anabolic pathways stimulated by resistance exercise (Scott *et al.* 2014). Furthermore, studies show increased early phase mTORC1 signalling (Fry *et al.* 2010), satellite cell activation (Nielsen *et al.* 2020), and muscle protein synthesis (Fujita *et al.* 2007) following acute BFR resistance exercise compared to normal blood flow exercise. With chronic BFR resistance training, these heightened cellular responses are proposed to drive enhanced increases in muscle cross-sectional area, fibre size, and capillarisation comparable to traditional high-load training (Lixandrão *et al.* 2018).

Blood flow restriction and endurance training

Integrating BFR into training sessions may help overcome plateaus in performance progression by enhancing adaptive effects without increasing mechanical workload (Ross *et al.* 2023). When BFR is employed during endurance training, the resulting effects include enhanced vasculature sheer stress

(Paiva *et al.* 2016), local tissue hypoxia (Corvino *et al.* 2017), increased metabolic stress (Fujita *et al.* 2007), and increased reactive O_2 species (Christiansen *et al.* 2018). Therefore, endurance exercise with BFR elicits a more stressful muscular environment, which may provide an additional adaptive stimulus (Suga *et al.* 2009). Naturally, the tolerable training intensity decreases during BFR (Ganesan *et al.* 2015), similar to the effects of simulated hypoxia (Townsend *et al.* 2017). While early studies demonstrated the potency of BFR as a stimulus for resistance training (Loenneke *et al.* 2012), relatively fewer studies have explored BFR's effects on endurance training.

For endurance adaptations, hypoxia and a reliance on anaerobic glycolysis during BFR exercise is proposed to increase activation of cell signalling involved in mitochondrial biogenesis (Burgomaster *et al.* 2003, Hardie *et al.* 2006). Preobrazenski *et al.* (2020) reported greater increases in PGC-1α mRNA following 30-minutes of aerobic exercise under gravity-induced BFR compared to normal blood flow. However, Conceição *et al.* (2016) reported attenuated expression of PGC-1α isoforms from 15-minutes of cycling at 40% $\dot{V}O_{2PEAK}$ with BFR compared to 30-minutes at 70% $\dot{V}O_{2PEAK}$ unrestricted. Both the Preobrazenski *et al*. and Conceição. studies recruited a similar level of participants, however reduced BFR training volume may introduce methodological bias (Bennett and Slattery 2019), which may reduce the pertinence of Conceição *et al.* (2016) findings. Further investigations have demonstrated acutely enhanced VEGF, HIF-1α, and PGC-1α mRNA expression when participants performed repeated ~20-40% 1 RM knee-extension with BFR at 220 mmHg (Larkin *et al.* 2012) and 110 mmHg (Ferguson *et al.* 2018). Accordingly, most of the evidence indicates improved muscle mitochondrial respiration, capillarity, and O_2 extraction following BFR endurance training versus normal blood flow (Sundberg *et al.* 1993, Christiansen *et al.* 2019).

The mechanisms by which BFR enhances endurance adaptations likely involve increased vascular shear stress (Sundberg *et al.* 1993), increased citrate synthase activity and local tissue hypoxia (Kaijser *et al.* 1990), and metabolic stress during exercise (Loenneke *et al.* 2011). Christiansen *et al.* (2018) reported enhanced PGC-1α and Na^+, K^+-ATPase complex subunit ancillary protein phospholemman-1 (FXYD1) mRNA expression. FXYD1 has an important role in maintaining the sodium and potassium ion gradients, which are crucial in preserving skeletal muscle membrane excitability to resist fatigue

(McKenna *et al.* 2008). A summary of the potential adaptation effects of endurance BFR training is presented in Figure 9.

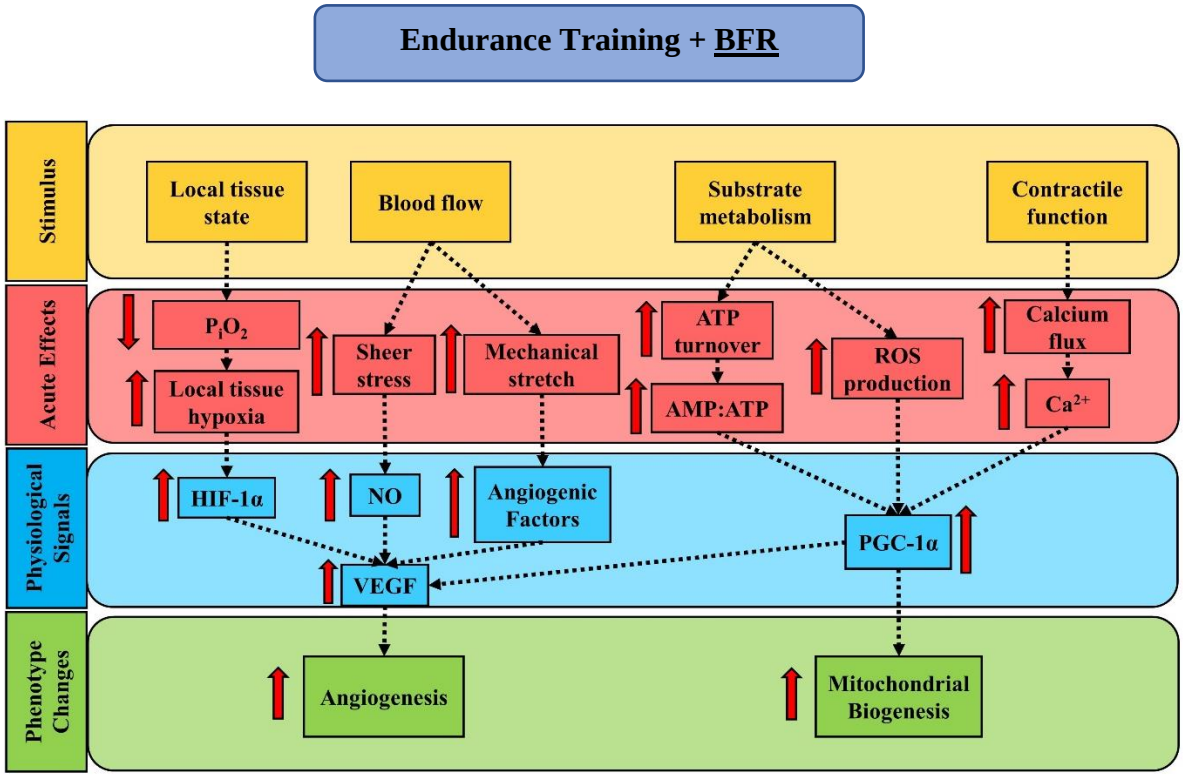


Figure 9. Hypothesised signalling effects of blood flow restriction (red arrows) on angiogenesis and mitochondrial biogenesis.

Abbreviations and definitions: AMP, adenosine monophosphate; ATP, adenosine triphosphate; Ca^{2+}, calcium ion; HIF-1α, hypoxia-inducible factor 1-alpha; NO, nitric oxide; PGC-1α, peroxisome proliferator-activated receptor gamma coactivator 1-alpha; P_iO_2, inspired partial pressure of oxygen; ROS, reactive oxygen species; VEGF, vascular endothelial growth factor. Adapted from Mitchell (2019)

Along with mitochondrial adaptations, the oscillations in shear stress with BFR exercise may optimise activation of molecular regulators of angiogenesis and capillary growth. Studies report increased limb blood flow, vascular conductance, and capillary contacts following BFR aerobic training (Esbjörnsson *et al.* 1993), which may improve O_2 delivery and extraction. Over weeks of BFR training, the repetitive pattern of oscillating shear stress provides a stronger stimulus for vascular remodelling compared to normal blood flow exercise. The mechanisms for BFR-induced improvements in $\dot{V}O_{2MAX}$ likely involve increased cardiovascular stress and adaptations in highly-trained athletes (Smith *et al.* 2021). During BFR exercise, venous pooling leads to reduced stroke volume, so heart rate must increase to maintain cardiac output (Ozaki *et al.* 2010). This induces greater cardiac mechanical stress over multiple training

sessions, which can improve stroke volume (Park *et al.* 2010) and subsequently $\dot{V}O_{2MAX}$ (Abe *et al.* 2010). Some evidence also indicates BFR aerobic training may increase left ventricular hypertrophy and femoral artery diameter, enhancing O_2 delivery (Christiansen *et al.* 2020).

Besides the cardiovascular adaptations, BFR exercise can improve the LT to a greater extent than $\dot{V}O_{2MAX}$. At a given workload, BFR increases relative exercise intensity (Thomas *et al.* 2018), which is key for LT improvements (Londeree 1997, Edge *et al.* 2006). Both continuous and intermittent BFR can stimulate angiogenesis through local hypoxia and shear stress (Hudlicka and Brown 2009, Egginton 2011, Hunt *et al.* 2013), enhancing O_2 extraction and metabolite removal. Increased intracellular buffering capacity may also play a role, as one study found improved CP without angiogenesis after sprint BFR training (Mitchell *et al.* 2019). The exact mechanisms are unclear but likely involve the hypoxic and metabolic stress of BFR exercise.

Current research applying BFR within endurance training interventions has reported mixed results. For example, de Oliveira *et al.* (2016) reported an enhanced aerobic capacity following BFR that was similar to training at a much higher intensity. Whereas, Keramidas *et al.* (2012) reported no significant differences between the BFR and a control group in the improvement of the $\dot{V}O_{2MAX}$ following 6 weeks of HIIT (~90% $\dot{V}O_{2MAX}$). This variation of the reported effects of BFR and endurance exercise could be partly attributed to inconsistency of protocols (Paton *et al.* 2017). Indeed, de Oliveira *et al.* and Keramidas *et al.* prescribed different BFR pressures, and Keramidas *et al.* did not report the width of the restriction cuff, both of which have an impact on tissue blood flow (Loenneke *et al.* 2012).

While previous evidence shows BFR can provide an adaptive stimulus like higher intensity training, few studies have explored BFR in trained individuals where performance improvements are often blunted. Highly trained athletes also demonstrated limited capillary adaptations to additional HIIT, with no change or declines in capillarity reported (MacInnis *et al.* 2017). Thus, novel strategies to provide an augmented stimulus may help surmount limited capillary growth in trained athletes. As training at or near $\dot{V}O_{2MAX}$ appears important for continued improvements in highly-trained individuals (Bangsbo 2015), applying BFR during recovery from HIIT may provide additional stimulus while maintaining normal training intensity (Ross *et al.* 2023).

Blood flow restriction applied during recovery phases of high-intensity interval training

Applying BFR during high-intensity exercise has been reported to provoke intolerable discomfort (Willis *et al.* 2018) and hinder achieving the high-intensity mechanical stimulus thought to be necessary for developing high-intensity performance (Bangsbo 2015). Instead, applying BFR during recovery between high-intensity intervals enables maintaining competition intensity while potentially maintaining benefits of accentuated metabolic disturbance. Applying occlusion after high-intensity exercise elicits a state of 'metabolic freeze' (Harris *et al.* 1976b) in which there is constrained recovery of PCr, pH (Okita *et al.* 2019), and muscle TSI (McManus *et al.* 2018, Mitchell 2019, Ienaga *et al.* 2022), contributing to enhanced metabolic and oxidative stress. For example, Giovanna and colleagues (2022) found a 2-week training program with BFR during recovery between 10-s sprints improved relative but not absolute $\dot{V}O_{2PEAK}$ during an anaerobic cycling test (105% MAP) in endurance-trained athletes. The lack of change in absolute $\dot{V}O_{2PEAK}$ implies limited central cardiovascular adaptations to increase O_2 delivery, but the improved relative $\dot{V}O_{2PEAK}$ suggests there were likely peripheral muscle adaptations that enhanced O_2 extraction and utilisation, such as increased capillarisation and mitochondrial density (Skattebo *et al.* 2020).

In contrast, Taylor *et al.* (2016) observed significant $\dot{V}O_{2MAX}$ improvements in trained cyclists following 4 weeks of incorporating ~76% arterial occlusion (BFR) for 2-minutes during the recovery between 30-s all-out sprints. A 1:9 work:recovery ratio was implemented during HIIT and the program progressed from 4 to 7 sprints over the 4-week intervention. Significant $\dot{V}O_{2MAX}$ and HIF-1α mRNA increases occurred with BFR versus non-BFR controls after training; however, no performance improvement occurred in a 15-km TT. Providing a taper to dissipate residual fatigue, as well as using a TTE performance test at constant power may better reveal adaptations without the impact of pacing strategy (Coakley and Passfield 2018). A subsequent training study utilising a progressive number of 30-s sprint repetitions with BFR during recovery over 4-weeks (Mitchell *et al.* 2019), suggested a trend toward enhanced angiogenesis signalling with chronic BFR application that may have reached statistical significance with a larger sample size.

The contrasting findings between Giovanna et al. (2022) and Taylor *et al*. (2016) highlight the variable responses that can occur with BFR training interventions. The balance between driving central cardiovascular adaptations versus peripheral muscle adaptations may depend on factors such as the specific exercise stimuli, the training status of the subjects, the details of the BFR protocol used, and the duration of the intervention. However, the overall premise is that applying BFR during the recovery periods has the potential to stimulate both central adaptations to enhance O_2 delivery and peripheral adaptations to improve O_2 extraction and utilisation within the active muscles.

In a similar intervention to Taylor et al. (2016) and Mitchell et al. (2019), Solsona and colleagues (2021) examined a repeated sprint protocol (5 x 30-s sprints with 4-minutes recovery) with BFR at 60% arterial occlusion pressure (~115 mmHg) for 2-minutes during recovery. While the authors demonstrated that BFR elicited a decrease in $\dot{V}O_2$ and $\dot{V}CO_2$ during the recovery from sprints, indicating reduced venous return, there was no significant decrease in vastus lateralis muscle TSI, suggesting limited additional oxidative stress. In contrast, Mitchell (2019) demonstrated that modest (~11%) reductions in vastus lateralis TSI can be achieved at 120 mmHg despite using similar BFR pressures (~115 vs 120 mmHg) and cuff widths (13 vs 13.5 cm). Furthermore, Mitchell (2019) demonstrated that an increased BFR pressure of 160 mmHg can further decrease rectus femoris muscle TSI, yet had no significant effect on vastus lateralis TSI. Mitchell (2019) suggested that increased concentration of deoxygenated haemoglobin at greater occlusion pressures signifies a greater imbalance between muscle O_2 delivery and utilisation, indicative of an increased hypoxic stimulus. An overview of the studies investigating the acute effects of applying BFR following HIIT or SIT is summarised in Table 1.

Table 1. Overview of studies investigating the acute application of blood flow restriction between high-intensity or sprint interval training.

Reference	Participants	HIIT Protocol	BFR Protocol	Key Findings
Taylor et al. 2016	8 moderately trained male cyclists. 32±7 yrs. $\dot{V}O_{2MAX}$, 4.3± 0.4 $L \cdot min^{-1}$	4 x 30-s maximal cycling sprints, 4.5-min rest between intervals.	130 mmHg BFR during 2-min recovery between intervals. Cuff width not reported.	BFR increased HIF-1α mRNA expression. PGC-1α and VEGF mRNA increased similarly in BFR and control.
Mitchell 2019	6 endurance trained males. Age and $\dot{V}O_{2PEAK}$ not reported.	4 x 30-s maximal cycling sprints, 4.5-min rest between intervals.	120 and 160 mmHg BFR during recovery. 13.5 cm cuff width.	Decrease in muscle oxygenation at 120 mmHg, further decrease at 160 mmHg.
Okita et al. 2019	7 untrained males. 21.6 ± 1.6 yrs. $\dot{V}O_{2PEAK}$ not reported.	3 x 1-min plantarflexion, at 20/40% 1-RM every 2-sec. 1-min rest between intervals.	1.3x resting BP (142.0 ± 17.7 mmHg). Continuous and intermittent BFR (during exercise or recovery). 18.5 cm wide thigh cuff.	40% 1-RM with recovery BFR produces similar metabolic stress to other conditions but with reduced discomfort.
Solsona et al. 2021	13 moderately trained males. 24 ± 3 yrs. $\dot{V}O_{2PEAK}$ not reported.	5 x 30-s maximal cycling sprints, 4-min rest between intervals.	60% AOP (114.6 ± 8.3 mmHg). during initial 2-min of recovery. 13 cm cuff width.	No change in vastus lateralis muscle oxygenation. Decreased cardiorespiratory measurements in BFR.
Ienaga et al. 2022	11 healthy males. 22.5 ± 0.3 yrs. $\dot{V}O_{2PEAK}$ not reported.	3 sets of 3 x 6-s maximal cycling sprints, 24-s rest between sprints, 5-min rest between sets.	100-120 mmHg occlusion for 2-min during recovery between sets. 11 cm cuff width.	Augmented muscle deoxygenation without affecting power output

Abbreviations and definitions: 1RM, one repetition maximum; AOP, arterial occlusion pressure; BFR, blood flow restriction; BP, blood pressure; HIF-1a, hypoxia-inducible factor 1-alpha; PGC-1a, peroxisome proliferator-activated receptor gamma coactivator 1-alpha; mRNA, messenger ribonucleic acid; VEGF, vascular endothelial growth factor; $\dot{V}O_{2MAX}$, maximum rate of oxygen utilisation

As the combination of sprint interval training and BFR applied during the recovery between work bouts has the potential to increase $\dot{V}O_{2MAX}$ in trained cyclists (Taylor *et al.* 2016, Mitchell *et al.* 2019) and improve CP and W′ in an elite cyclist (Taylor 2016), integrating BFR into high-performance TP training could further extend HIIT benefits. Of note, enhanced $\dot{V}O_{2MAX}$, power output, capillary density, and oxidative capacity are known to specifically relate to TP performance (van der Zwaard *et al.* 2016). If tolerable, BFR could improve TP performance by enhancing these factors. Faster W′ reconstitution from adaptations like improved $\dot{V}O_{2MAX}$, CP, capillary density or mitochondria may also improve recovery between TP efforts (Chorley *et al.* 2020). An overview of the studies investigating the chronic effects of applying BFR following HIIT or SIT is summarised in Table 2.

Table 2. Overview of studies investigating the chronic application of blood flow restriction between high-intensity or sprint interval training.

Study	Participants	HIIT Protocol	BFR Protocol	Key Findings
Taylor et al. 2016	20 well-trained male cyclists. 27±6 yrs. 4.6±0.4 $L \cdot min^{-1}$	4-7x 30-s cycling sprints, 4.5-min rest between intervals. Progressive increase in reps per week over 4 weeks. Two sessions per week	130 mmHg BFR during 2-min recovery between intervals. 13 cm cuff width.	Increased $\dot{V}O_{2MAX}$ in BFR vs control.
Mitchell et al. 2019	21 competitive male cyclists and triathletes. 23±5 yrs. 4.6 ± 0.5 $L \cdot min^{-1}$	4-7x 30-s cycling sprints, 4.5-min rest between intervals. Progressive increase in reps per week over 4 weeks. Two sessions per week	120 mmHg BFR during 2-min recovery between intervals. 13.5 cm cuff width.	$\dot{V}O_{2MAX}$ increased in BFR. No effect on CP, capillary density, or mitochondrial protein content.
Giovanna et al. 2022	39 endurance-trained men. 25.6 ± 5.7 yrs. ~4.2 $L \cdot min^{-1}$	4 sets of 5 x 10-s cycling sprints with 20-s active recovery, 2-weeks training. Two sessions per week	45% AOP (88±10 mmHg). 11 cm cuff width. Four conditions: Continuous BFR, exercise BFR, recovery BFR, hypoxia.	Peak aerobic power increased in all groups. Relative $\dot{V}O_{2PEAK}$ during test at 105% $\dot{V}O_{2PEAK}$ increased in recovery BFR group.

Abbreviations and definitions: AOP, arterial occlusion pressure; BFR, blood flow restriction; CP, critical power; VEGF, vascular endothelial growth factor; $\dot{V}O_{2MAX}$, maximum rate of oxygen utilisation; $\dot{V}O_{2PEAK}$, peak rate of oxygen utilisation

While the potential benefits of BFR training are highlighted throughout this review, it is important to also consider the potential negative outcomes and risks, especially for athletic populations. The occlusive nature of the BFR stimulus raises concerns about ischemia-reperfusion injury, which may increase the production of reactive O_2 species and can lead to oxidative stress, inflammation, and muscle damage (Cheng *et al.* 2003, Garten *et al.* 2015). Case studies have reported instances of rhabdomyolysis occurring with BFR training (Iversen and Røstad 2010, Tabata *et al.* 2016, Clark and Manini 2017), though the overall incidence rate appears low (0.1 to 0.2%) (Thompson *et al.* 2018). Prolonged ischemia from overly restrictive cuff pressures could potentially exacerbate this effect. Additionally, excessive external compression from the cuffs can lead to bruising, petechiae, nerve damage, and discomfort (Nakajima *et al.* 2006, Nakajima *et al.* 2011). Athletes may be at particular risk due to higher training volumes and intensities. More research is warranted on the incidence of adverse effects in highly-trained populations. Careful monitoring of cuff pressures (Loenneke *et al.* 2012), occlusion duration, and indirect muscle damage markers (Sieljacks *et al.* 2016, Nielsen *et al.* 2017) is recommended, especially when implementing more strenuous high-frequency BFR protocols. Screening for individual risk factors and contraindications is also advisable (Kacin *et al.* 2015).

Hastening the rate of recovery

While the trainability of the recovery rate time constant has not been directly explored, there is indirect evidence suggesting aerobic training can enhance recovery from high-intensity intermittent exercise. Previous research has shown that aerobic training improves the rate of recovery following intense efforts (Thoden 1991). Tomlin and Wenger (2001) proposed that increased aerobic capacity provides greater oxidative energy during passive recovery, while improved blood flow facilitates removal of metabolic byproducts like lactate and H^+ ions. Indeed, Tesch and Wright (1983) found that higher capillary density, which can be increased through upregulation of VEGF expression (Hoier and Hellsten 2014), has been associated with greater blood lactate clearance after intense exercise (Oyono-Enguelle *et al.* 1990, Tomlin *et al.* 2001). Aerobic adaptations such as enhanced $\dot{V}O_2$ kinetics also reduce early reliance on non-oxidative metabolism, delaying metabolite accumulation (Tomlin *et al.* 2001).

Together, these circulatory and metabolic improvements likely facilitate recovery from repeated high-intensity bouts.

Supporting the theory that aerobic fitness influences recovery capacity, Hamilton *et al*. (1991) found that aerobically trained individuals consumed more O_2 during repeated sprints compared to less fit controls. Similarly, impaired O_2 delivery during hypobaric exposure increased lactate accumulation and reduced performance (Balsom *et al.* 1994). Links between aerobic fitness and intermittent performance are further evidenced in soccer, where elite players demonstrate excellent repeated sprinting skills and high aerobic capacity (Krustrup *et al.* 2003).

Previous research has demonstrated that mountain biking (Stapelfeldt *et al.* 2004, Impellizzeri and Marcora 2007) and cross-country skiing (Gløersen *et al.* 2020) often involve repeated high-intensity efforts that cumulatively exceed an athlete's initial W′ at the start of a race. This 'repeatability' indicates that the ability to repeatedly generate high power output, and recover during lower intensity periods within a race, is a key feature of some racing formats (Maunder *et al.* 2021). Menaspà *et al.* (2017) reported that female cyclists able to attain a higher racing performance (top-5) were also those who can perform a greater number of short high-intensity efforts (≥ 10-s at > 7.5 $W \cdot kg^{-1}$) during 1-day races. However, Muriel *et al.* (2022) did not find that the number of high-intensity efforts during the 'La Vuelta 2020' grand tour stage race differed between professional cyclists of eight World Tour and seven ProTeam cyclists. It should be noted though, that Muriel *et al.* (2022) assessed cyclists with different roles within teams and not necessarily cyclists who performed well, which limits the applicability of their results to determine whether repeatability is a key performance factor in male grand tour stage racing.

Van Der Zwaard *et al*. (2018) identified enhanced physiological characteristics in Dutch national-level pursuit cyclists, including higher mean $\dot{V}O_2$ during a 15-km TT, VO_2 at LT1 and LT2, MAP, type I muscle fibres, capillary density, and capillaries per fibre. Despite assessing these fundamental physiological determinants of endurance, the rate of recovery was not evaluated. Given the intermittent nature of the TP event, one might expect superior recovery rates in TP cyclists if relief periods permit a return towards pre-exercise homeostasis. While Bartram *et al.* (2018) demonstrated that TP cyclists

possessed a greater rate of W′ reconstitution than predicted by Skiba 2, no studies have explored recovery rate as a TP performance factor. This is surprising, as the TP presents a standardised and controlled environment to apply the W'_{BAL} concept.

Applying BFR during the recovery phases between high-intensity intervals has the potential to enhance both central cardiovascular adaptations and peripheral muscle adaptations, which could improve TP performance. Studies by Taylor et al. (2016) and Mitchell et al. (2019) demonstrate that incorporating BFR during the recovery phases of SIT can increase $\dot{V}O_{2MAX}$ in trained cyclists, suggesting improvements in O_2 delivery and/or extraction. Furthermore, Giovanna et al. (2022) found that BFR during recovery between sprints improved relative $\dot{V}O_{2PEAK}$ during a test at 105% MAP, implying peripheral muscle adaptations that enhance O_2 utilisation, such as increased capillarisation and mitochondrial density (Skattebo *et al.* 2020). As van der Zwaard et al. (2018) highlight, enhanced $\dot{V}O_{2MAX}$, TT power output, capillary density, and oxidative capacity are related to TP performance. Additionally, faster W′ reconstitution from these adaptations could improve recovery between TP lead turns. Given the intermittent nature of the TP event, superior recovery rates are likely a key performance factor (Bartram *et al.* 2018). Therefore, integrating BFR into the recovery periods of HIIT for TP cyclists could potentially enhance both the central and peripheral factors that contribute to improved recovery and overall TP performance.

Literature review summary

The TP is an Olympic event in endurance track cycling that requires cyclists to produce repeated near-maximal efforts with minimal recovery. To record a competitive time, a TP team must precisely execute a practiced effort to attain maximal performance capabilities. As world records have rapidly progressed in recent years, the extreme physiological and biomechanical demands have heightened. While basic physiological attributes of elite TP cyclists are established, the factors differentiating performance level remain unexplored. Examining determinants of TP performance may reveal key aspects to inform training and racing strategies for optimising capabilities.

Specifically, elite TP cyclists possess excellent CP and rate of W′ reconstitution. However, the degree to which these factors influence TP performance is unknown. Enhanced understanding of how W′ balance model parameters impact TP outcomes could refine training and racing approaches.

Traditionally, varied periodised training, including low- and high-intensity, develops key aspects of TP performance such as endurance and high-intensity repeatability. However, further optimisations face challenges from diminishing returns and non-functional overreaching risks. Thus, novel stimuli augmenting capabilities without disrupting training are appealing.

There is potential for BFR training to provide an additional stimulus between efforts at TP intensities. Such an intervention may enhance TP-specific tolerance and parameters of the W'_{BAL} model, helping cyclists improve repeated high-intensity performance without increasing workload. However, research integrating BFR with current training and evaluating feasibility and tolerability is needed, as BFR often induces discomfort.

In summary, while the TP represents an extreme endurance challenge, further research on training enhancement and recovery practices for elite track cyclists is needed to push the boundaries of human performance in this event.

Research questions

Research Question One

What are the key physiological determinants from the W'_{BAL} model that can differentiate performance levels in the TP event in track cycling? Additionally, how can the W'_{BAL} model be used to improve the understanding of the physiological demands and training requirements of the TP?

Research Question Two

What are the acute physiological responses when BFR is integrated between high-intensity intervals for highly-trained cyclists? Are these acute physiological responses sufficient to indicate a potential adaptative stimulus if the BFR intervention is applied chronically?

Research Question Three

How does a chronic intervention of applying BFR between high-intensity work bouts impact aspects related to TP performance in trained cyclists?

Research Question Four

If the integration of BFR into HIIT indicates a sufficient training stimulus for significant adaptations, can this proposed BFR training intervention be integrated into an elite TP training programme? Furthermore, are the acute effects observed in elite TP cyclists similar to those in non-elite cyclists, and what are the elite TP cyclists' subjective perception of the intervention?

Chapter Three – Critical power, work capacity, and recovery characteristics of team-pursuit cyclists

Pugh, C. F., C. M. Beaven, R. A. Ferguson, M. W. Driller, C. D. Palmer, and C. D. Paton (2022). "Critical power, work capacity, and recovery characteristics of team-pursuit cyclists." International Journal of Sports Physiology and Performance **17**(11): 1606-1613.

Prelude

To identify physiological factors that differentiate elite from sub-elite TP cyclists, we applied novel W'_{BAL} modelling using individualised parameters. Quantifying these individual determinants may reveal opportunities to optimise TP performance. The W'_{BAL} model has previously been applied in TP cyclists using generalised parameters of W′ reconstitution rates. However, the extent to which individualised parameters of W′ reconstitution can improve the accuracy and validity of W'_{BAL} modelling in the TP is currently unexplored.

Abstract

Purpose: Leading a 4-km team-pursuit (TP) requires high-intensity efforts above critical power (CP) that depletes rider's finite work capacity (W′), whereas riders following in the aerodynamic draft may experience some recovery due to reduced power demands. This study aimed to determine how rider ability and CP and W′ measures impact TP performance and the extent to which W′ can reconstitute during recovery positions in a TP race.

Methods: Three TP teams, each consisting of 4 males, completed individual performance tests to determine their CP and W′. Teams were classified based on their performance level as International (INT), National (NAT), or Regional (REG). Each team performed a TP on an indoor velodrome (INT: 3:49.9; NAT: 3:56.7; REG: 4:05.4; minute:sec). Ergometer-based TP simulations with an open-ended interval to exhaustion were performed to measure individual ability to reconstitute W′ at 25-100 W below CP.

Results: The INT team possessed higher CP (407 ± 4 W) than both NAT (381 ± 13 W) and REG (376 ± 15 W) ($p < .05$), whereas W′ was similar between teams (INT: 27.2 ± 2.8 kJ; NAT: 29.3 ± 2.4 kJ; REG: 28.8 ± 1.6 kJ; $p > .05$). The INT team expended 104 ± 5% of their initial W′ during the TP and possessed faster rates of recovery than NAT and REG at 25 and 50 W below CP ($p < .05$).

Conclusions: CP and rate of W′ reconstitution have a greater impact on TP performance than W′ magnitude and can differentiate TP performance level.

Introduction

The men's team-pursuit (TP) is a track cycling event in which four riders work together to complete 4-km in the fastest time possible. Team members rotate positions between leading the team and riding closely behind to share the work and permit recovery before their next lead effort. A TP team travelling at a speed of ~60-km·hr^{-1} requires the lead rider to produce 550-650 W (Broker *et al.* 1999, Jeukendrup *et al.* 2000), whilst the mean team power output across all positions has been reported to be between 425-460 W (Broker *et al.* 1999, Jeukendrup *et al.* 2000). Given the TP requirement for repeated high-intensity efforts, it is unsurprising that oxidative aerobic pathways contribute the majority (~75%) of a TP's energy demands (Jeukendrup *et al.* 2000, Tomlin *et al.* 2001). TP cyclists thus possess endurance-orientated characteristics such as superior power output at maximal and submaximal intensities (Bartram *et al.* 2018, Van Der Zwaard *et al.* 2018).

The maximum sustainable rate of oxidative metabolism and the corresponding power output has been termed the critical power (CP; Watts), whilst the finite work capacity and thus tolerance to exercise above CP can be defined as the 'work-prime' (W′; Joules). Together, the CP and W′ are parameters that describe the power-duration relationship of cycling performance. Male TP cyclists competing at world-cup level have been reported to have a CP of ~391 W, meaning a rider leading the TP will expend W′. A drafting rider has the opportunity to cycle at power outputs below CP (Bartram *et al.* 2021), however it is yet to be fully established if meaningful recovery occurs during periods of below CP cycling in the TP.

Cycling at intensities below CP reduces a rider's metabolic demands (Kirby *et al.* 2021), and permits recovery (Chorley *et al.* 2020). Skiba *et al.* (2012, 2015) published a model for intermittent exercise that assumes W′ is expended when power > CP, and predicts the extent of W′ reconstitution (Chidnok *et al.* 2012) when power < CP. The rate of W′ reconstitution correlates with an individual's $\dot{V}O_{2MAX}$ and CP (Chorley *et al.* 2020) and depends on both the duration and intensity of work and relief periods (Caen *et al.* 2019). Therefore, the rate of W′ reconstitution is activity-specific and may represent an important metric for TP performance. Moreover, the rate of W′ reconstitution is reportedly faster in well-trained cyclists (Bartram *et al.* 2018) compared to recreational participants (Skiba *et al.* 2015).

The balance between W′ expenditure and reconstitution (W'_{BAL}) predicts the remaining W′ at any point during intermittent exercise and indicates the proximity of individual riders to task failure. The W'_{BAL} model can be employed within the TP if the rate of W′ reconstitution is assessed in conditions that reflect the duration and intensity of the TP. While Skiba *et al.* (2012, 2015) determined recovery rates ranging 50-300 W below CP, the protocol's work/relief characteristics do not reflect actual power demands of the TP. Bartram *et al. (2018)* developed a protocol to assess the rate of W′ reconstitution that is physiologically relevant to the TP and published generalised recovery rates at 50-200 W below CP in high-performance endurance track cyclists. Bartram *et al.* (2021) subsequently applied their population-specific recovery model to a TP team of similarly well-trained cyclists, and concluded that whilst their recovery rates produced reasonable W'_{BAL} predictions, individually derived recovery models are necessary for optimal W'_{BAL} tracking. Furthermore, Bartram *et al.* (2021) reported the mean power during periods of W′ reconstitution was ~79 W below CP, yet the stochastic power demands of the TP suggest there are recovery periods within 50 W of CP. Therefore, whilst previous literature has investigated the importance of protocol design for determining the rate of W′ reconstitution and demonstrated that TP races feature periods below CP, the rate of W′ reconstitution may not yet have been assessed at sufficient intensities to model W'_{BAL} in a TP. Furthermore, the degree to which the rate of W′ reconstitution and periods spent below CP are performance determinants of the TP is unclear.

To our knowledge, there are currently no studies that have applied W'_{BAL} modelling to different levels of TP performance using individualised parameters. Therefore, the primary aim of this study was to compare the CP, W′, and rates of W′ reconstitution across three TP performance levels (International, National and Regional). A secondary aim was to evaluate the association of recovery ability with the magnitude of recovery and end TP W'_{BAL} values. It was hypothesised that CP, W′, and rate of W′ reconstitution would differ between performance levels, and that W'_{BAL} values would be influenced by the recovery models employed.

Methods

Subjects

Twelve competitive male track cyclists participated in the study (age: 22 ± 3 y, height: 181 ± 4 cm, body mass: 77.6 ± 5.9 kg). All cyclists had high-performance training histories, and nine had previously competed in international competitions at junior or senior World Championships. Participants were grouped by their current respective TP performance level as either International (n = 4, age: 24 ± 3 y, height: 179 ± 4 cm, body mass: 74.2 ± 4.9 kg), National (n = 4, age: 21 ± 2 y, height: 184 ± 3 cm, body mass: 76.5 ± 3.6 kg), or Regional (n = 4, age: 22 ± 3 y, height: 181 ± 3 cm, body mass: 82.1 ± 5.7 kg). All participants gave their written informed consent to complete the study, which was approved by the University of Waikato Human Research Ethics Committee and performed in accordance with the declaration of Helsinki.

Experimental design

In a cross-sectional design, participants performed eight experimental trials over four weeks (Figure 10). Participants initially completed a cycling CP assessment protocol consisting of three time-trials (TT) over 1-, 4-, and 10-minute durations to determine CP and W′. These test durations have been applied by high-performance Australian cyclists (Bartram *et al.* 2017, Bartram *et al.* 2018, Bartram *et al.* 2021) and are consistent with recommendations by Poole (1986). Participants then performed a maximal effort TP within their respective teams on an international standard 250-m indoor velodrome. Finally, participants performed four high-intensity interval sessions to assess rate of W′ reconstitution at various TP relief intensities. Intervals were completed at individual TP lead power with the relief intensities being performed at 0, 25, 50, and 100 W below CP; the relief intensity at CP (0) was performed first, and the order of remaining trials was randomised. Prior to each testing session, participants were requested to not ingest any potential performance enhancing ergogenic aids (e.g., sodium bicarbonate).

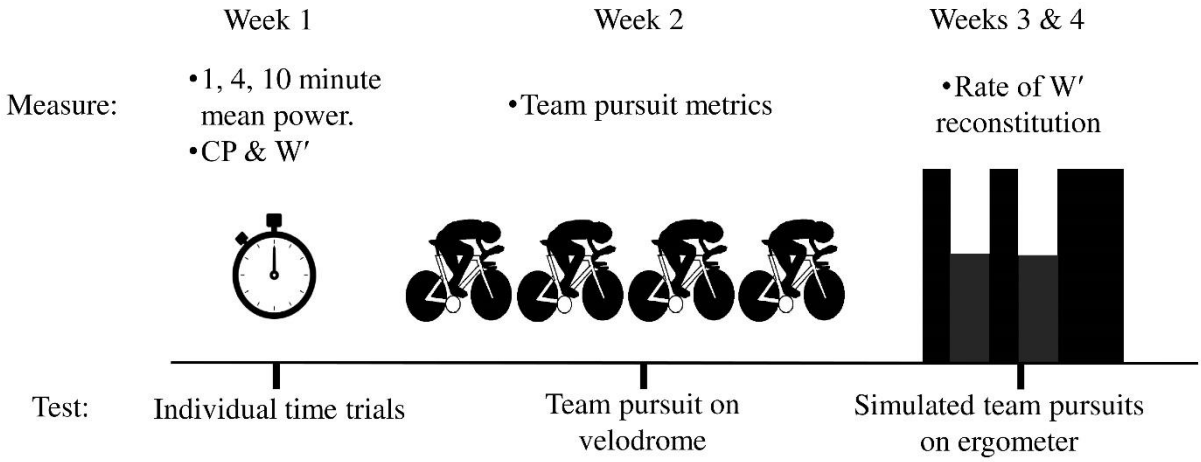


Figure 10. Study timeline.

The first week of the protocol prescribed performance tests to determine CP and W′. The second week included a 4-km team-pursuit. In the final two weeks, participants completed 4 ergometer-based interval sessions to determine the rate of W′ reconstitution at the intensities observed in the team-pursuit.
Abbreviations and definitions: CP: critical power. W′: work capacity above CP.

Performance measures

Critical power and W′

Field-based TTs were used to derive estimates of rider CP and W′. Performance tests of 1-, 4-, and 10-minute duration were conducted in a randomised order and separated by at least 24-hours. Participants performed a self-selected 20-minute warmup before each trial and were instructed to produce their "greatest average power possible" for each TT. Trials began from a stationary start, and participants were required to remain seated and maintain a cadence of 100-120 RPM using self-selected gear ratios. Participant's personal bicycles were fitted with an SRM (Schoberer Rad Meßtechnik, Jülich, Germany) crankset calibrated according to Wooles and colleagues (2005).

Team-pursuit

Each team performed a TP in simulated competition conditions against similarly matched teams that did not participate in the study, starting on opposite sides of the track. Participants used their personal fixed-gear bicycles fitted with the power meters from field-based TTs. The first rider began from a starting gate, and a Tissot timing system (Corgémont, Switzerland) provided a finishing time for the 3rd rider to

complete 4-km. The mean power exerted when the rider was in the lead position was defined as the lead power.

W′ reconstitution trials

The reconstitution rate of W′ ($\tau_{W'}$) was estimated using data from four simulated TP races performed over two consecutive days (with 4-hours rest between trials on the same day). These trials took place at an indoor venue, and the conditions were 20-24 °C, 30-45% relative humidity. Tests were performed on the same bicycles and power meters as the field-based TTs mounted on a stationary cycle ergometer (Wahoo KICKR v5, Atlanta, GA, USA), calibrated according to the manufacturer's instructions. The ergometer's resistance was controlled via software (TrainerRoad, Reno, Nevada, USA) using an isokinetic function set to predefined power requirements. All trials were preceded by a standardised 20-minute warmup featuring a 10-minute ramp from 35% to 85% CP and 1.5-minutes constant-load cycling at 100% CP, followed by 15-s at the participant's TP lead power and finally 8.25-minutes at 35% CP.

The trial format featured two 30-s intervals at the participant's TP lead power interspersed by 60-s periods at each prescribed TP relief intensity below CP (D_{CP}). Relief conditions were CP (D_{CP} 0), 25 W below CP (D_{CP} 25), 50 W below CP (D_{CP} 50), and 100 W below CP (D_{CP} 100). Immediately following the 2nd relief interval, participants performed a time to exhaustion interval at their TP lead power. Participants completed a familiarisation trial of the D_{CP} 0 condition in the week preceding data collection. Participants were instructed to maintain a pedal cadence of 110-120 RPM.

Data analysis

Power data were recorded at 1 Hz and was downloaded to Golden Cheetah (GC, version 3.5, open-source, https://www.goldencheetah.org/) before being processed in Excel (Redmond, WA, USA).

Three separate CP models were employed to determine the lowest standard error of the estimates (SEE) for CP and W′:

linear work-time model:

$$Work = (CP \cdot T) + W' \quad \text{Equation 9}$$

linear power-time model:

$$P = \frac{W'}{T} + CP \quad \text{Equation 10}$$

nonlinear power-time model:

$$T = \frac{W'}{(P - CP)} \quad \text{Equation 11}$$

where *Work* represents the cumulative mechanical energy output (Joules), and *P* represents the mean power output for a given duration (*T*, in s). The nonlinear power-time model produced lower SEE than the linear work-time and linear power-time^{-1} models for CP (4 ± 2 W vs 7 ± 2 W and 10 ± 3 W, respectively) and was used to estimate CP and W′ for all participants.

Power data from each TP race was analysed for mean power output, work performed above CP relative to the initial amount of the W′ (%), mean power output during lead efforts, and mean relief power output while in positions 2, 3, and 4.

Power data from W′ reconstitution trials and CP and W′ parameters were processed to estimate W'_{BAL} using the following formula:

$$W'_{BAL} = \begin{cases} W'_{BAL,i-1} - \left((P_i - CP) \cdot \Delta u_i\right), P_i > CP \\ W'_0 - W'_{expended} \cdot \left(e^{-\frac{\Delta u_i}{tau}}\right), P_i < CP \end{cases} \quad \text{Equation 12}$$

where *i* = the *i*th segment of the total time subdivided into n segments at 1 Hz (Δu), P_i = mean power output for segment *i*. Thus, W'_{BAL} is calculated sequentially and $W'_{BAL,i-1}$ represents a preceding estimation of W'_{BAL}. $W'_{expended}$ is the quantity of depleted W′ at *i*-1 and is calculated as:

$$W'_{expended} = W'_0 - W'_{BAL,i-1}$$ Equation 13

see Skiba & Clarke (2021) for review (2021a). The W′ reconstitution time constant (*Tau,* in s) is provided by the power function of D_{CP}-*Tau*:

$$Tau = A \cdot {D_{CP}}^B$$ Equation 14

where *A* represents a scaling factor and *B* represents the rate of decay.

Previous literature demonstrates that there is a high-level of agreement for CP between laboratory and field testing yet W′ is environment-specific (Karsten *et al.* 2014, Triska *et al.* 2017). Therefore, a parameter (W'_{ERG}) was calculated from the D_{CP} 0 trial to standardise the magnitude of W′ on a stationary ergometer when no recovery would occur. The participant's W'_{ERG} was defined as the final W'_{BAL} value of the D_{CP} 0 trial. A single *Tau* value was set for each of D_{CP} 25, 50, and 100 W conditions and determined using Excel's iterative Solver function, which fit end W'_{BAL} values to W'_{ERG}. D_{CP}-*Tau* power regressions were derived for each participant to predict *Tau* at any intensity below CP (Skiba *et al.* 2015, Bartram *et al.* 2018).

Power data from the TP were processed using the parameters of CP, W′, and individualised $\tau_{W'}$ to produce W'_{BAL} traces during TP races. To explore the effect of different D_{CP}-*Tau* models on TP W'_{BAL} values, races were also processed using the equations from Skiba *et al.* (2015) and Bartram *et al.* (2018).

Generalised D_{CP}-*Tau* power functions were calculated for each team from mean $\tau_{W'}$ at D_{CP} 25, D_{CP} 50, and D_{CP} 100. Individualised equations cannot be reported because of an agreed-upon embargo with the national federation.

Statistical analysis

Data were analysed in SPSS (IBM, Armonk, NY, USA) using one-way ANOVA between the three performance levels and dependent variables: CP, W′, 1-, 4-, 10-minute TT mean power output, TP mean

power output, TP mean power output in all positions, TP W′ work done, and $\tau_{W'}$ at D_{CP} 25, D_{CP} 50, and D_{CP} 100. Post-hoc analyses were performed to compare between groups with Tukey's HSD test. The assumption of normality was verified using the Shapiro-Wilk test. A repeated-measures ANOVA was performed to compare final TP W'_{BAL} predictions from individualised D_{CP}-*Tau* models with previously published models (Skiba *et al.* 2015, Bartram *et al.* 2018). Bivariate correlations (r) were performed between CP and D_{CP}-*Tau* (D_{CP}: 25, 50, and 100) and the strength of r was determined using the following criteria: ≤ 0.1, trivial; > 0.1 to 0.3, small; > 0.3 to 0.5, moderate; > 0.5 to 0.7, large; > 0.7 to 0.9, very large; and > 0.9 to 1.0, almost perfect (Hopkins *et al.* 2009). Statistical significance was set at $p < 0.05$.

Results

CP and W′

The mean CP and W′ estimates and 1-, 4-, and 10-minute average power outputs are presented in Table 3. The International team had a greater CP compared to National (p = .047) and Regional teams (p = .021), whereas the International team's W′ magnitude was not different to National (p = .519) or Regional (p = .667) teams. The mean power output of the International team's 1- and 4-minute TT was not different to National (p = .900, p = .273) and Regional teams (p = .793, p = .087), respectively. In contrast, mean power output in the 10-minute TT was greater in the International team than National (p = .040) and Regional teams (p = .020).

Table 3. Absolute CP and W′ estimates and performance test average power outputs for International, National, and Regional TP performance levels.

	1-minute (W)	4-minutes (W)	10-minutes (W)	CP (W)	CP SEE range (W)	W′ (kJ)	W′ SEE range (kJ)
International	777 ± 42	523 ± 13	452 ± 6 [a,b]	407 ± 4 [a,b]	3-8	27.2 ± 2.8	1.7-2.8
National	791 ± 26	506 ± 8	430 ± 9	381 ± 13	3-6	29.3 ± 2.4	1.8-2.7
Regional	798 ± 50	498 ± 17	424 ± 16	376 ± 15	2-5	28.8 ± 1.6	1.0-2.9

Abbreviations: CP, critical power. SEE, standard error of the estimate. W′, work capacity above CP. [a] indicates a significant difference ($p < .05$) between International and National squads. [b] indicates a significant difference ($p < .05$) between International and Regional squads. Values presented as mean ± SD, where applicable.

Team-pursuit races

Team pursuit data are presented in Table 4. The International team produced a greater mean TP power output compared to Regional (p = .034) but not National teams (p = .072). The International team produced a greater mean power output while leading the TP than both National (p = .016) and Regional teams (p = .015). No differences were observed between the International team and National or Regional teams for the mean power output of positions 2 (p = .809, p = .813), 3 (p = .108, p = .088), and 4 (p = .294, p = .140), respectively.

Table 4. Team pursuit data for International, National, and Regional performance levels.

	International	National	Regional
Finishing time (minute:s)	3:49.9	3:56.7	4:05.4
Mean power (W)	501 ± 26 [b]	471 ± 34	463 ± 12
Lead rider power (W)	658 ± 13 [a,b]	609 ± 23	613 ± 19
Mean power at position 2 (W)	446 ± 31	422 ± 53	423 ± 50
Mean power at position 3 (W)	405 ± 30	372 ± 27	370 ± 16
Mean power at position 4 (W)	426 ± 21	392 ± 35	381 ± 19

[a] indicates a significant difference ($p < .05$) between International and National squads. [b] indicates a significant difference ($p < .05$) between International and Regional squads. Values presented as mean ± SD, where applicable.

W′ reconstitution

The mean relief intensity of the D_{CP} 0 trial was 6 W above CP, whereas the mean relief intensity of the D_{CP} 25, D_{CP} 50, and D_{CP} 100 trials were 20, 43, and 91 W below CP, respectively. The mean duration of W′ reconstitution trials for International, National, and Regional teams were 224 ± 13, 243 ± 11, and 216 ± 10 s, respectively. There were large, significant negative correlations between CP and $\tau_{W'}$ at D_{CP} 25 (r = -.819, p = .001) and D_{CP} 50 (r = -.714, p = .009), but no significant correlation at D_{CP} 100 (r = -.510, p = .091) (Figure 11). Individualised D_{CP}-*Tau* data are presented in Table 5 along with the Bartram *et al.* (2018) and Skiba *et al.* (2015) D_{CP}-*Tau* values for comparison of models. Generalised D_{CP}-*Tau*

functions for International, National, and Regional teams are presented in Figure 11. The mean standard error of *A* and *B* D_{CP}-*Tau* parameters are 19% and 15% in the International team, 20% and 12% in the National team, and 13% and 9% in the Regional team, respectively. The International team possessed lower *Tau* values compared with National and Regional teams at D_{CP} 25 (p = .034 and p = .009) and D_{CP} 50 (p = .032 and p = .017), respectively. There was no difference in $\tau_{W'}$ at D_{CP} 100 between the International team and National (p = .158) or Regional (p = .119) teams.

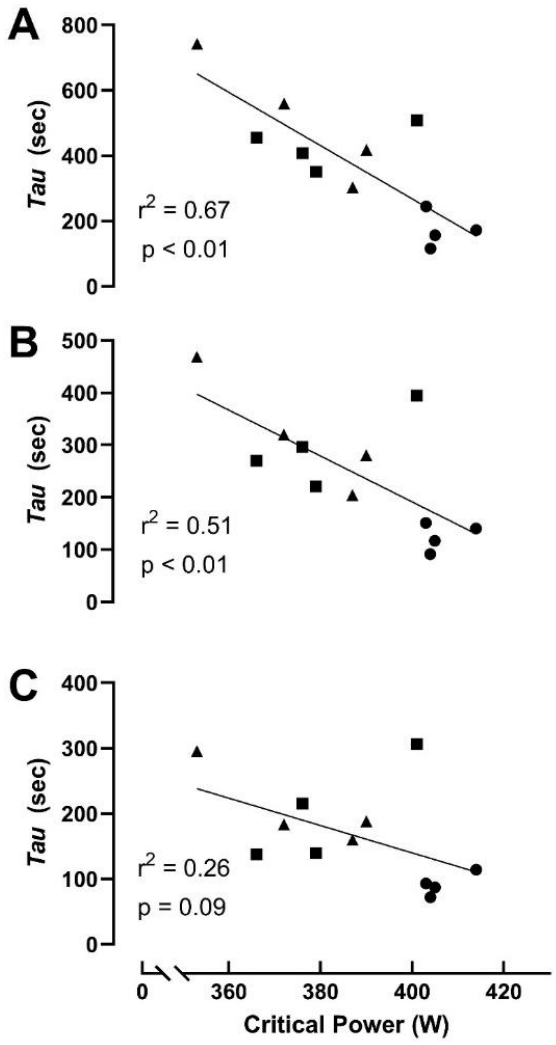


Figure 11. Relationship of critical power and rate of recovery in team-pursuit cyclists.

Panels: A) Rate of recovery 25 W below CP; B) Rate of recovery 50 W below CP; C) Rate of recovery 100 W below CP. International—Circles; National—Triangles; Regional-Squares.
Abbreviations and definitions: Tau, the time constant of W' reconstitution; CP, critical power; sec, seconds; W, Watts.

Table 5. W′ reconstitution individualised Tau data with the Bartram and Skiba 2 relationships.

	International	National	Regional	Bartram	Skiba International	Skiba National	Skiba Regional
Tau equation	$789 \cdot D_{CP}{}^{-0.468}$	$1883 \cdot D_{CP}{}^{-0.487}$	$5184 \cdot D_{CP}{}^{-0.700}$	$2287 \cdot D_{CP}{}^{-0.688}$	W′ / D_{CP}	W′ / D_{CP}	W′ / D_{CP}
Interval power (W)	658 ± 13	609 ± 23	613 ± 19				
W'_{ERG} (kJ)	10.7 ± 1.8	7.0 ± 3.6	7.7 ± 1.5				
W′ reconstitution time constant (s)							
D_{CP} 25	176 ± 47 [a,b]	393 ± 77	546 ± 124	250	1088 ± 110	1173 ± 96	1154 ± 62
D_{CP} 50	125 ± 23 [a,b]	279 ± 75	335 ± 79	155	544 ± 55	586 ± 48	577 ± 31
D_{CP} 100	92 ± 15	200 ± 69	207 ± 53	96	272 ± 28	293 ± 24	288 ± 15

Generalised equations are reported instead of individualised equations because of an agreed-upon embargo with the national federation. Tau equations are presented in the format: $A.D_{CP}{}^{B}$, where A is a scaling factor and B is the rate of decay. Abbreviations and definitions: D_{CP}, difference between relief intensity and critical power; Tau, W′ reconstitution time constant; W′, work capacity above critical power; W'_{ERG}, final W'_{BAL} value of the D_{CP} 0 trial. [a] indicates a significant difference ($p < .05$) between International and National squads. [b] indicates a significant difference ($p < .05$) between International and Regional squads. Values presented as mean ± SD, where applicable.

Team-pursuit W'_{BAL}

Figure 12 shows TP W'_{BAL} traces using the individualised, Bartram (2018), and Skiba (2015) D_{CP}-*Tau* models, and Table 6 presents the final W'_{BAL} values from all models. The W′ expended relative to its initial amount was similar in the International (104 ± 5%) to the National (75 ± 18%; p = .117) and Regional teams (99 ± 7%; p = .754). Bartram's D_{CP}-*Tau* model produced similar final W'_{BAL} values to individualised D_{CP}-*Tau* models in the International (p = .333) and National (p = .295) teams, but greater final W'_{BAL} values in the Regional team (p = .006). Skiba's D_{CP}-*Tau* function produced significantly lower final W′ values in the International team (p = .002) but had no effect with the National (p = .165) and Regional (p = .267) teams.

Table 6. Comparison of the individualised recovery rate relationship on the W'_{BAL} values of TP races with the Bartram and Skiba relationships.

	International	National	Regional
End W'_{BAL} (kJ)			
Individualised	6.8 ± 3.6	6.4 ± 5.3	4.3 ± 4.0
Bartram	6.2 ± 4.1	9.0 ± 6.0	8.5 ± 3.4 *
Skiba 2	3.0 ± 3.7 *	5.3 ± 5.2	2.9 ± 3.4

** indicates a significant difference to individualised recovery rate relationship ($p < .05$). Values presented as mean ± SD.*

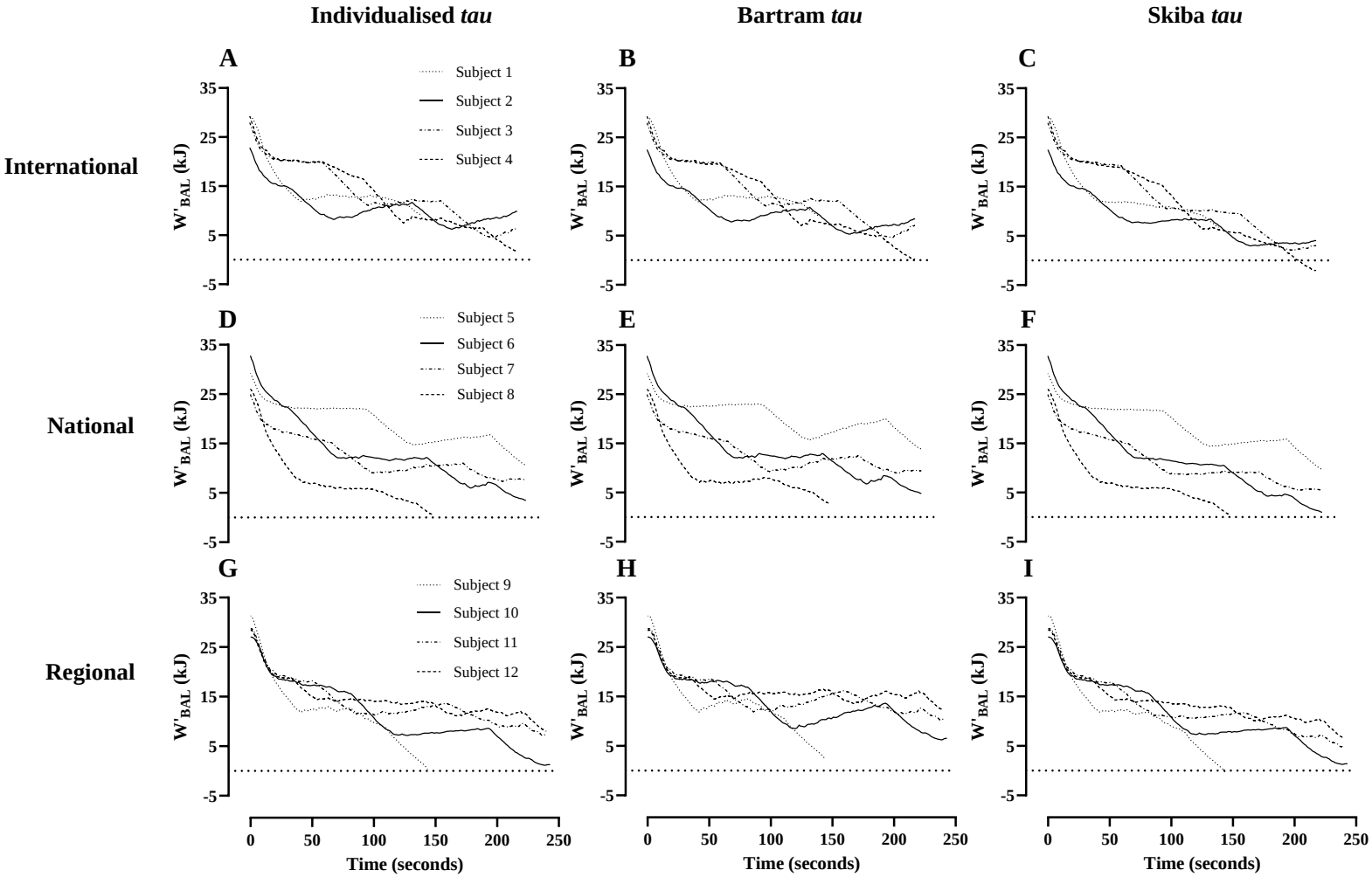


Figure 12. Modelling W'BAL during a team-pursuit using individualised, Bartram, and Skiba Tau recovery rate models.

Leading TP efforts can be identified by sharp reductions in W'BAL.
A) International TP with individualised Tau, B) International TP with Bartram Tau, C) International TP with Skiba Tau, D) National TP with individualised Tau, E) National TP with Bartram Tau, F) National TP with Skiba Tau, G) Regional TP with individualised Tau, H) National TP with Bartram Tau, I) National TP with Skiba Tau. Abbreviations and definitions: Tau: the time constant of W' reconstitution. Abbreviations and definitions: kJ, kilojoules; Tau, time constant of W' reconstitution; W'BAL: remaining capacity to perform work above critical power.

Discussion

The main aim of this study was to determine how rider ability and individual CP and W′ measures impact TP performance and to what extent W′ can reconstitute during recovery positions in a TP race. The present data demonstrate that the International TP team is distinguished from lower-performing teams by enhanced CP and $\tau_{W'}$, but not W′ magnitude. In addition, whilst W'_{BAL} values were influenced by the employed D_{CP}-*Tau* model, similar W'_{BAL} values were observed between the individualised, Skiba, and Bartram model forms with respect to performance level.

The main finding that CP and $\tau_{W'}$ were greatest in the International TP team is not surprising given that high-performance pursuit cyclists have highly adapted aerobic physiological characteristics including an enhanced $\dot{V}O_{2MAX}$, proportion of type I muscle fibres, capillary density, mitochondrial content (Van Der Zwaard *et al.* 2018), and $\dot{V}O_2$ uptake kinetics (Craig *et al.* 1993). Whilst CP (Vanhatalo *et al.* 2016, Mitchell *et al.* 2018), and intense exercise performance (Iaia *et al.* 2011) are known to be highly dependent on muscle capillary supply, the physiological underpinnings of $\tau_{W'}$ have yet to be established. An enhanced muscle capillary supply may facilitate the clearing of fatigue-inducing metabolites (Iaia *et al.* 2011) and increase O_2 availability in the muscles (Tomlin *et al.* 2001), both of which likely accelerates $\tau_{W'}$ as indicated by the relationship between muscle reoxygenation rate and $\tau_{W'}$ (Kirby *et al.* 2021).

Leading a TP demands a power output greater than CP which results in expenditure of W′ and contributes to the high levels of fatigue typical of TP events. Despite a ~50% reduction in aerodynamic drag (Fitton *et al.* 2018), the power requirements of drafting riders are still substantial, and there could be opportunities to ride below CP. Recovering in non-lead positions is critical as it reduces the metabolic requirements (McCole *et al.* 1990), which can preserve W′. To our knowledge, the current study is the first to implement W'_{BAL} modelling in high-level TP teams using individualised estimates of CP, W′, and $\tau_{W'}$ at TP relief intensities. The International team recovered faster than the National and Regional teams at D_{CP} 25 and D_{CP} 50, which indicates the importance of recovery at intensities just below CP to TP performance. Moreover, in the International team, the overall expended quantity of W′ was greater than its initial magnitude and indicates there were periods of meaningful recovery despite the high-

intensity. Models from Skiba *et al.* (2015) and Bartram *et al.* (2018) permit extrapolation of D_{CP}-*Tau* to D_{CP} 25, yet overpredicted *Tau* at D_{CP} 25 in the International team of the present study (1138-s vs 250-s vs 176-s, respectively). This difference in D_{CP}-*Tau* between the current International team and published models emphasise the importance of individualised $\tau_{W'}$ testing for sports requiring maximal efforts with periods above and below CP.

The present study compared the individualised, Skiba *et al.* (2015), and Bartram *et al.* (2018) D_{CP}-*Tau* relationships using TP W'_{BAL} values. Our findings suggest that $\tau_{W'}$ of the International and National teams were similar to Bartram's model, whereas, the National and Regional teams were similar to Skiba's model (2015). As Bartram's participants were high-performing and Skiba's were recreational, these results indicate that published models may provide approximate estimates of $\tau_{W'}$ with respect to performance level. Indeed, we report significant correlations between CP and $\tau_{W'}$ at D_{CP} 25 and D_{CP} 50 (both $R^2 > .5$), which supports previous literature findings (Chorley *et al.* 2020). We observed that National level cyclists possessed higher *Tau* values compared to elite cyclists in Bartram *et al.* (2018) despite similar CP estimates. This observation supports previous literature and indicates that $\tau_{W'}$ is multifaceted and likely influenced by protocol design, CP, $\dot{V}O_{2MAX}$, and EPOC (Chorley *et al.* 2020, Lievens *et al.* 2021).

As a consequence of a greater CP and faster W′ reconstitution, the International team were able to produce a higher lead rider power output than the National and Regional teams, which likely contributed to the International team finishing 7- and 15-s faster than National and Regional teams, respectively. The power outputs generated by the International team were greater than previously published data from international competition (Jeukendrup *et al.* 2000) (mean power: 501 vs 461 W; lead power: 658 vs 581 W, respectively), whilst the ~25-s faster finishing time indicates a remarkable improvement in performance over the past ~20-years. Although the higher mean speed produced by the International team in the present study partially explains the increase in power demand, the time disparity may also be attributed to developments in reduction of aerodynamic drag (Kyle *et al.* 1984) or increased technical ability.

A TP team which achieves full W′ depletion by the race's finish has been suggested to be optimal for performance (Bartram *et al.* 2018). However, a lower final W'_{BAL} value does not exclusively belong to an optimally paced team as poor strategic and technical decisions may reduce final W'_{BAL} values without a corresponding improvement in TP finishing time. The comparison data presented in Figure 12 and Table 6 demonstrate that final TP W'_{BAL} values were often greater than 0 kJ with no differences between performance levels and suggests that a TP team's ability to fully deplete W′ was not a decisive TP performance factor. Yet, Subject 1 withdrew from their race with a W'_{BAL} of ~9 kJ, and the National TP team finished with 25% of their W′ remaining, which implies sub-optimal pacing strategies and thus, faster performances are theoretically possible.

This study features the following limitations. Cardio-metabolic data during CP and W′ estimation trials were not obtained which does not allow the verification of the attainment of $\dot{V}O_{2MAX}$. While the well-trained participants were likely to have attained $\dot{V}O_{2MAX}$ during 4- and 10-minute performance tests, we cannot verify the 1-minute performance test elicited $\dot{V}O_{2MAX}$. However, test durations less than 90-s are adequate to determine maximal accumulated O_2 deficit (Withers *et al.* 1991), and endurance training is reported to hasten $\dot{V}O_2$ uptake kinetics to permit attainment of $\dot{V}O_{2MAX}$ in <2-minutes (Caputo *et al.* 2008). Additionally, the 1-, 4-, 10-minutes trial format is not considered the "gold-standard" CP protocol, which advocates test durations of 2-15-minutes. Yet, the test durations are within the recommendations by Poole (1986) and are employed by the Australian high-performance track endurance program (Bartram *et al.* 2017, Bartram *et al.* 2018, Bartram *et al.* 2021) and, anecdotally, in New Zealand's equivalent male track endurance program. Recent literature (Caen *et al.* 2021) indicates that W′ reconstitution exhibits biexponential recovery kinetics unlike the mono-exponential model applied in the present study. Presumably, an International TP team's superior endurance capacity may lead to faster W′ reconstitution in both the "slow-" and "fast-phases". However, it could be speculated that a TP team would benefit most from enhanced "fast-phase" kinetics, given periods of below-CP intensity are transient.

Practical applications

This study utilised performance tests that are readily replicable in a practical setting and provided acceptable error to allow confidence in estimating CP and W′. The D_{CP}-*Tau* models presented offer a generalised estimate of $\tau_{W'}$ from International to Regional performance levels and permit practitioners to implement approximate W'_{BAL} modelling in other TP cyclists. While improvements in CP and/or W′ may lead to enhanced TP performance, our data indicates that overall endurance capacity is the most important physiological factor of TP performance.

Conclusions

Field-based performance tests for CP, W′, and W′ reconstitution in International, National, and Regional TP cyclists revealed that the International team is distinguished by an enhanced CP and faster W′ reconstitution at TP relief intensities. These findings suggest that CP and rate of recovery have a greater impact on TP performance than W′ magnitude and demonstrate the importance of high-intensity aerobic fitness to the TP.

Chapter Four – Acute physiological responses of blood flow restriction between high-intensity interval repetitions in trained cyclists: a randomised crossover trial

Pugh, C. F., C. D. Paton, R. A. Ferguson, M. W. Driller, C. M. Beaven (2024) "Acute physiological responses of blood flow restriction between high-intensity interval repetitions in trained cyclists." European Journal of Sport Science. *In review*

Prelude

The key findings from Study One highlight the importance of an elevated CP and faster rate of W′ reconstitution for elite TP performance in track cycling. To target improvements in these parameters in highly-trained cyclists, interventions that acutely impose physiological stress may stimulate beneficial adaptations. Specifically, interventions that induce metabolic disturbances, hypoxia, angiogenic signalling, and/or oxidative stress could potentially enhance mitochondrial function, increase capillary density, improve metabolite buffering, and accelerate phosphocreatine resynthesis, thereby increasing CP and/or accelerating W′ reconstitution rate.

With this rationale, a BFR protocol was designed to integrate occlusion between the high-intensity intervals typically performed by TP cyclists. However, before implementing this BFR intervention long-term, it is prudent to first examine its acute potency to elicit sufficient physiological strain under controlled conditions. Demonstrating that this integrated BFR protocol acutely stresses the physiological systems governing CP and W′ reconstitution would provide justification for longer-term implementation as a strategy to enhance TP performance. Existing literature suggests that BFR training can potentially improve oxidative capacity, buffering capacity, and angiogenesis, which are key determinants of CP and W′ reconstitution rate. Therefore, Study Two aimed to determine the acute impacts of integrating BFR into a TP-specific interval session on markers of metabolic stress, muscle oxygenation, and angiogenic signalling.

Abstract

Blood flow restriction (BFR) is increasingly being used as a training technique to enhance aerobic performance in endurance athletes. This study the examined physiological responses when BFR was applied during the recovery phases between intervals in a high-intensity interval training (HIIT) session in trained cyclists. Eleven competitive road cyclists (mean ± SD, age: 28 ± 7 y, body mass: 69 ± 6 kg, peak oxygen uptake: 65 ± 9 $mL \cdot kg^{-1} \cdot min^{-1}$) completed two randomised crossover conditions: HIIT with (BFR) and without (CON) BFR applied during recovery. HIIT consisted of six 30-s cycling bouts at an intensity equivalent to 85% of maximal 30-s power (523 ± 93 W), interspersed with 4.5-min recovery. Blood flow restriction (200 mmHg, 12 cm cuff width) was applied for 2-min in the early recovery phase between each interval. Pulmonary gas exchange ($\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}E$), tissue oxygen saturation index (TSI), heart rate (HR), and serum vascular endothelial growth factor concentration (VEGF) were measured. Compared to CON, BFR increased $\dot{V}CO_2$ and $\dot{V}E$ during work bouts (both $p<0.05$, $d<0.5$), but there was no effect on $\dot{V}O_2$, TSI, or HR ($p>0.05$). In early recovery, BFR decreased TSI, $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}E$ (all $p<0.05$, $d>0.8$) versus CON, with no change in HR ($p>0.05$). In late recovery, when BFR was released, $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, and HR increased, but TSI decreased versus CON (all $p<0.05$, $d>0.8$). There was a greater increase in VEGF at 3-hours post-exercise in the BFR condition compared to CON ($p<0.05$, $d>0.8$). Incorporating BFR into HIIT recovery phases altered physiological responses compared to exercise alone and thus has the potential to enhance training adaptations over time.

Introduction

The volume, intensity, and frequency of exercise performed during a training program are key determinants of the degree of physiological and performance adaptation (Galán-Rioja *et al.* 2023). Incorporating diverse training modalities is especially important in endurance sports like competitive cycling (van Erp *et al.* 2021) as cycling often involves variable power output demands. These demands include exercise efforts at both maximal and submaximal intensities, which stress both oxidative and glycolytic energy pathways (Jeukendrup *et al.* 2000). While high volumes of low-intensity training promote muscular conditioning and attenuate fatigue-related power output decline during prolonged exercise (Maunder *et al.* 2021), high-intensity interval training (HIIT) in lower volumes stimulates greater and more effective adaptation to higher intensities than lower-intensity training alone (Seiler *et al.* 2009).

The integration of HIIT into training regimens, usually prior to competition, has been shown to enhance both endurance and high-intensity performance (Laursen *et al.* 2002a). Specifically, HIIT can enhance key physiological performance markers such as the peak rate of O_2 uptake ($\dot{V}O_{2PEAK}$), muscle buffering capacity, and muscle oxidative capacity (Laursen *et al.* 2002a, Gibala *et al.* 2009). Despite the physiological benefits, performance adaptations from HIIT can reach a plateau after 8-12 sessions (Paton *et al.* 2005) or ~3-weeks of training (Norrbom *et al.* 2022) without progressive overload of intensity or duration. This plateau is potentially attributed to a decline in the activity of transcription factors HIF1-α and PGC-1α (Perry *et al.* 2010, Norrbom *et al.* 2022), both of which are key activators of vascular endothelial growth factor (VEGF), a principal driver of angiogenesis (Lee *et al.* 2004, Chinsomboon *et al.* 2009, Olfert *et al.* 2010).

Integrating blood flow restriction (BFR) into HIIT sessions may help overcome plateaus in performance progression by enhancing adaptive effects without increasing mechanical workload (Ross *et al.* 2023). The use of BFR involves applying pneumatic cuffs or tourniquets to limbs to manipulate blood flow and occlude venous return. This altered blood flow profile facilitates physiological stressors such as reduced muscle oxygenation (McManus *et al.* 2018), enhanced vascular shear stress (Hudlicka *et al.* 2009, Preobrazenski *et al.* 2020), metabolite accumulation (Loenneke *et al.* 2011, Sakamaki-Sunaga *et*

al. 2012), and increased oxidative stress (Christiansen *et al.* 2018). The intensified physiological and metabolic stress associated with BFR exercise stimulate adaptive responses in skeletal muscle and the microvasculature (Ferguson *et al.* 2021). For example, greater upregulation of skeletal muscle PGC-1α mRNA was observed following BFR cycling compared to work-matched exercise without BFR (Preobrazenski *et al.* 2020). Similarly, Larkin *et al*. (2012) showed low-load knee extension exercise with BFR increased VEGF and HIF-1α mRNA 4-hours post-exercise compared to the same exercise without BFR.

Applying BFR intermittently during the recovery phase of high-intensity exercise sustains intramuscular metabolite concentrations (Okita *et al.* 2019) and reduces muscle oxygenation (McManus *et al.* 2018, Mitchell 2019, Solsona *et al.* 2021, Ienaga *et al.* 2022). Although a session of 4 x 30-s sprints with BFR during recovery did not amplify the increase in PGC-1α or VEGF mRNA expression compared to sprinting alone, there was an augmented HIF-1α mRNA response with BFR (Taylor *et al.* 2016). A subsequent training study utilising a progressive number (4 to 7) of 30-s sprint repetitions with intermittent BFR during recovery over 4-weeks (Mitchell *et al.* 2019), suggested a trend toward enhanced angiogenesis signalling with chronic BFR application. While BFR did not significantly enhance sprint training adaptation in trained individuals, it may provide added stimulus in trained athletes who have plateaued in their adaptation to traditional HIIT protocols. Therefore, the heightened physiological and metabolic stresses elicited by integrating BFR into the recovery phases of HIIT may potentiate physiological adaptations.

Using BFR during the recovery phase of high-intensity exercise sustains intramuscular metabolite concentrations (Okita *et al.* 2019) and reduces muscle oxygenation (McManus *et al.* 2018, Mitchell 2019, Ienaga *et al.* 2022). Although a single session of 4 x 30-s sprints with BFR during recovery did not amplify the increase in PGC-1α or VEGF mRNA expression compared to sprinting alone, there was an augmented HIF-1α mRNA response with BFR (Taylor *et al.* 2016). A subsequent training study utilising a progressive number of 30-s sprint repetitions with BFR during recovery over 4-weeks (Mitchell *et al.* 2019), suggested a trend toward enhanced angiogenesis signalling with chronic BFR application that may have reached statistical significance with a larger sample size. Therefore, the

heightened metabolic and oxidative stresses elicited by integrating BFR into the recovery phases of HIIT may potentiate physiological adaptations, providing a plausible pathway to further extend the training benefits of HIIT.

Few studies to date have investigated the magnitude of acute physiological perturbations experienced during the application of BFR in the recovery phases during HIIT (Taylor *et al.* 2016, Mitchell 2019, Okita *et al.* 2019, Solsona *et al.* 2021, Ienaga *et al.* 2022). Therefore, the objective of this study is to investigate the acute physiological effects of a six-repetition HIIT session either with BFR applied during the recovery phases, or with an unrestricted recovery in trained cyclists. It was hypothesised that, compared to a standard HIIT session without BFR, applying BFR during the recovery phases would reduce cardio-pulmonary and muscle O_2 saturation measures, adding to the overall physiological stress of the training stimulus, and increase serum VEGF concentration post-exercise.

Methods

Participants

Eleven competitive road cyclists (male n = 9, female n = 2) training for >6-hours·week^{-1} volunteered for the study (age: 28 ± 7 y, height: 175 ± 7 cm, body mass: 69 ± 6 kg, $\dot{V}O_{2PEAK}$: 4.5 ± 0.7 L·minute^{-1}; 65 ± 9 mL·kg^{-1}·minute^{-1}). Inclusion criteria for females required uninterrupted hormonal contraception use throughout the study to control for potential hormonal fluctuations. Participants' abilities were categorised as trained (tier three, n = 2), highly-trained (tier four, n = 6), and professional (tier five, n = 3) based on established nomenclature (De Pauw *et al.* 2013). Prior to testing, participants completed a medical screening questionnaire to ensure they had no cardiovascular or haematological contraindications to BFR. Participants were screened prior to recruitment (Kacin *et al.* 2015) and provided written informed consent. The study was approved by the participating institution's human research ethics committee [ethics approval number: HREC(Health)2021#22] and performed in accordance with the Declaration of Helsinki.

Experimental design

This study employed a randomised crossover design with two experimental conditions: HIIT with (BFR) or without (CON) BFR applied during recovery phases. Participants attended the laboratory on three occasions over a ~21-day period. During the first visit, participants completed an incremental ramp test to determine their $\dot{V}O_{2PEAK}$, and a maximal 30-s sprint test. Participants also completed a partial familiarisation of the experimental test protocol, which included two repetitions of the personalised CON and BFR sessions with NIRS and occlusion but without cardiopulmonary or blood sampling. In visits two and three, participants performed a HIIT protocol consisting of six, 30-s work bouts with either the experimental (BFR) or control (CON) condition applied during the recovery between work bouts. The order of the BFR and CON conditions was randomised using a coin toss for the first participant. The subsequent participants then performed the conditions in an alternating order, resulting in six participants performing the BFR condition first and five performing the CON condition first.

All trials took place in an environmentally controlled laboratory (temperature 19 ± 1 ºC; relative humidity 45 ± 5%). Experimental sessions were conducted at the same time of day (±1-hour) for each participant to control for diurnal variation were separated by a 3- to 7-day washout period. This washout duration was chosen in accordance with the time course of physiological responses to BFR during recovery between high-intensity work bouts, as reported by Mitchell (2019). Participants performed all trials on their personal bicycles mounted to a stationary ergometer (Kickr V5, Wahoo, Atlanta, GA, USA), with resistance controlled via software (TrainerRoad, Reno, Nevada, USA). Power output in Watts (W) was recorded at 1 Hz by the cycle ergometer.

Participants were instructed to avoid strenuous physical activity and replicate any light training performed in the 24-hours prior to each testing session. Participants recorded their dietary intake for the 24-hours preceding the first experimental trial and replicated this as closely as possible for each subsequent trial. Participants abstained from caffeine-containing products 12-hours before the tests and refrained from eating, consuming only water for the 3-hours before each testing session.

Preliminary testing procedures

Incremental ramp test

Participants completed an incremental ramp exercise protocol starting at 150 W and increasing at 25 $W \cdot minute^{-1}$ (~0.4 $W \cdot s^{-1}$). Participants self-selected a pedalling cadence between 75-100 revolutions per minute (RPM) and maintained this cadence throughout the duration of the test. Exercise continued until volitional exhaustion or when cadence fell 10% below the chosen rate for more than 5-s, despite strong verbal encouragement. Pulmonary gas exchange was measured continuously throughout exercise with a metabolic cart (Parvo metabolic cart, Medics TrueOne 2400, Salt Lake City, Utah, USA), which was calibrated according to the manufacturer's instructions. This experimental set-up allowed for breath-by-breath analysis of O_2 uptake ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), and minute ventilation ($\dot{V}E$). The highest mean $\dot{V}O_2$ measured over any continuous 30-s period during the ramp test was defined as $\dot{V}O_{2PEAK}$. Maximal aerobic power (MAP) was defined as the mean power output during the final 5-s of the test (Buchfuhrer *et al.* 1983). The mean MAP achieved by participants in the study was 409 ± 58 W and 5.91 ± 0.77 W/kg in relative terms.

Maximal 30-s test

Following a 10-minte active-recovery period after the incremental ramp test, participants completed a 10-s familiarisation effort of the 30-s maximal sprint test to establish appropriate gearing for a cadence of 80-120 RPM. The incremental test and 10-s familiarisation functioned as a warm-up for the 30-s test. The maximal 30-s test was performed while seated with the ergometer set in an isoinertial mode, 5-minutes after the familiarisation. The procedure required participants to pedal at a constant cadence of 110 RPM before ergometer resistance was applied. Participants were provided strong verbal encouragement to produce a maximal effort throughout the 30-s test. The test was deemed valid if the participant maintained their cadence within an 80-120 RPM range. The mean power output attained during the maximal 30-s test defined the metric, P30s. Peak power output was recorded as the greatest 1-s power output value. On average, participants attained a P30s of 625 ± 117 W and a peak power output of 894 ± 250 W.

Main experimental protocols

Upon arrival at the laboratory, participants rested in a seated position for 10-minutes while they were fitted with two near-infrared spectroscopy (NIRS) sensors to measure tissue saturation index (TSI; %), recorded at 1 Hz (Moxy 3, Fortiori Design LLC, Minnesota, USA). The Moxy 3 has previously demonstrated good test-retest reliability (CV: 5.7-6.2%) (McManus *et al.* 2018), and accuracy for assessing tissue oxygenation during occlusion protocols (Feldmann *et al.* 2019, Yogev *et al.* 2023), thus supporting its suitability for the current study. The NIRS sensors were secured with a compression bandage to the left and right vastus-lateralis muscle bellies at 40% of the distance between the greater trochanter and the lateral epicondyle of the femur. Heart rate (HR) was measured continuously via telemetry (H10, Polar Electro, Kempele, Finland). A venous blood sample was obtained 10-minutes before the warm-up. Participants then performed a 20-minute individualised warm-up which consisted of 5-minutes at 35% of MAP, 3-minutes at 50% of MAP, 1-minute at 60% of MAP, 1-minute at 75% of MAP, and 10-minutes at 30% of MAP.

Experimental HIIT exercise trials began immediately after the warm-up following a 5-s countdown. Pilot studies informed that team-pursuit lead power output was approximately 85% of an individual's mean power output during the maximal 30-s test (85% of P30s). Therefore, the HIIT exercise trial involved six, 30-s work bouts at 85% of P30s (523 ± 94 W), interspersed with ~4.5-minutes of passive recovery in a supine position on an adjacent bed (work-to-recovery ratio=1:9). With 30-s remaining before their next work bout, participants remounted their bicycles to continue the subsequent repetition. At this point, they could cycle freely with zero resistance applied to the ergometer and were instructed to increase their pedal cadence to 100-120 RPM just before the start of each work bout. Breath-by-breath pulmonary gas exchange was measured throughout the exercise session. After completing the exercise session, participants were allowed to leave the laboratory but required to fast (except drinking water) and avoid exercise, before returning to the laboratory 3-hours later to provide a post-exercise venous blood sample.

BFR protocol

In the BFR condition, once the participant laid semi-supine, 11 cm wide occlusion cuffs (Occlude, Aarhus, Denmark) were applied to the uppermost portion of both thighs. The cuffs were then inflated to 200 mmHg using hand-operated sphygmomanometers. This pressure was determined following pilot studies conducted in a similar cohort. The cuff application and inflation process took approximately 30-s from the end of the work bouts. Pressure was maintained for 2-minutes before deflation and cuff removal to provide participants with 1.5-minutes of unoccluded supine recovery. The BFR and CON conditions were workload- and duration-matched.

Blood sampling and analysis

Blood samples were taken by venepuncture (21G, Vacutainer, PrecisionGlide, BD) from antecubital veins with the participant seated. Blood samples for serum (SST II Advance Gold, 8.5 mL, Vacutainer, BD) were immediately inverted five times following collection, then rested at room temperature for 10-minutes before being placed inside an ice-cooled, insulated container. Blood samples were centrifuged within 2-hours of collection at 1500 G for 10-minutes before serum was aliquoted and stored at -20 ℃. Serum samples were analysed, in triplicate, using human VEGF-A ELISA plates (Invitrogen, Thermo Fisher Scientific, Waltham, MA, USA) with automated plate washing and reading. All samples for a given individual were analysed on a single plate. The intra-assay coefficient of variation of ELISA analyses was 5.1%, and the inter-assay CV was 6.7% and concentrations reported in $pg \cdot mL^{-1}$.

Data handling

A cycling computer (Edge 520, Garmin, Olathe, Kansas, USA) was used to capture the raw power output from the ergometer, NIRS devices, and HR monitor. The raw data file was downloaded to Golden Cheetah (version 3.5, open source, https://www.goldencheetah.org/) for subsequent analysis. These data were then imported into an Excel spreadsheet (Microsoft, Redmond, WA, USA). Similarly, raw metabolic data were incorporated into the same spreadsheet and synchronised with the exercise onset using event markers from both the cycling computer and metabolic cart. Mean power output was

calculated for each 30-s work bout. Mean values for TSI, HR, $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}E$ were calculated over entire HIIT sessions, and separately categorised into three phases: the work bouts (0- to 30-s), the early recovery phase (occluded: 1- to 3-minutes), and the late recovery phase (unoccluded: 3- to 4.5-minutes). For analysis, mean values for TSI, $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, and HR were calculated for each phase across all six repetitions and participants. Mean values for each variable and condition were also determined over entire recovery phases (combined early and late recovery phases). In addition, traces from all participants were averaged into 5-s bins for each variable to create a single visual representation.

Statistical analysis

Separate one-way repeated-measures ANOVA were conducted to assess the effect of condition on entire recovery periods and whole sessions. A two-way 2x6 repeated-measures ANOVA was conducted with 'condition' (BFR and CON) and ‘repetition' (1-6) (each divided into time phases of work bouts, early recovery, and late recovery) as factors, comparing power output, TSI, $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, and HR means. The interaction effects between condition and repetition, as well as condition and HIIT phase, were examined. To determine the effect of BFR on angiogenic signalling, a two-way repeated-measures ANOVA was run on serum VEGF concentration with two within-subject factors: ‘time’ (pre-exercise, 3-hours post-exercise) and ‘condition’ (BFR, CON). Where significant interaction effects were found, Bonferroni-corrected *post-hoc* analyses were performed. Residual normality was confirmed by the Shapiro-Wilk test. Mauchly’s test indicated that the assumption of sphericity was violated for the main effects of repetition ($p<0.05$); therefore, degrees of freedom were corrected using Greenhouse-Geisser estimates. The statistical analyses were performed using SPSS (IBM, SPSS for Windows, Version 29.0, Armonk, NY), with statistical significance set at $p\leq0.05$. Graphs were produced in GraphPad Prism 9.5.1 for Windows (GraphPad Software, San Diego, California, USA). If an effect was significant, the magnitude of effect sizes were interpreted using thresholds of 0.2, 0.5, and 0.8 for *small*, *moderate*, and *large* (Cohen 1988). An effect size of $d<0.2$ was considered *trivial*. Data are presented as mean ±95% confidence intervals constructed by the normal approximation method.

Results

5-s trace interpretation

All participants completed the full protocol in CON and BFR, and there are no instances of missing data. Figure 13 shows 5-s averaged traces representing the mean across all participants for each experimental condition and variable. During the early recovery phase of the BFR condition, there was a reduction in TSI, $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}E$ compared to CON. However, during the late recovery phase, BFR cuff release led to TSI returning towards CON levels, while $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}E$ rise above CON levels. The effect of BFR is maintained for TSI, $\dot{V}O_2$, and $\dot{V}CO_2$ across all repetitions. The BFR condition led to an elevated HR during recovery compared to CON and led to an increase in $\dot{V}E$ across repetitions.

Power output

There was no significant main effect of condition (BFR vs. CON) on power output ($p=0.597$; mean ± SD: BFR 523 ± 94 W, CON 523 ± 92 W). Power output for BFR and CON, respectively, were: repetition 1, 521 ± 95 W and 523 ± 94 W; repetition 2, 526 ± 95 W and 524 ± 94 W; repetition 3, 525 ± 95 W and 524 ± 93 W; repetition 4, 527 ± 96 W and 524 ± 93 W; repetition 5, 526 ± 94 W and 523 ± 92 W; repetition 6, 516 ± 93 W and 517 ± 89 W. The condition by repetition interaction on power output was not statistically significant ($p=0.237$). There was no main effect of repetition on power output ($p=0.630$).

Figure 14 shows the mean data for all variables across all sets and phases from all participants. Table 7 shows the data for the measured physiological variables during the different phases of the HIIT session.

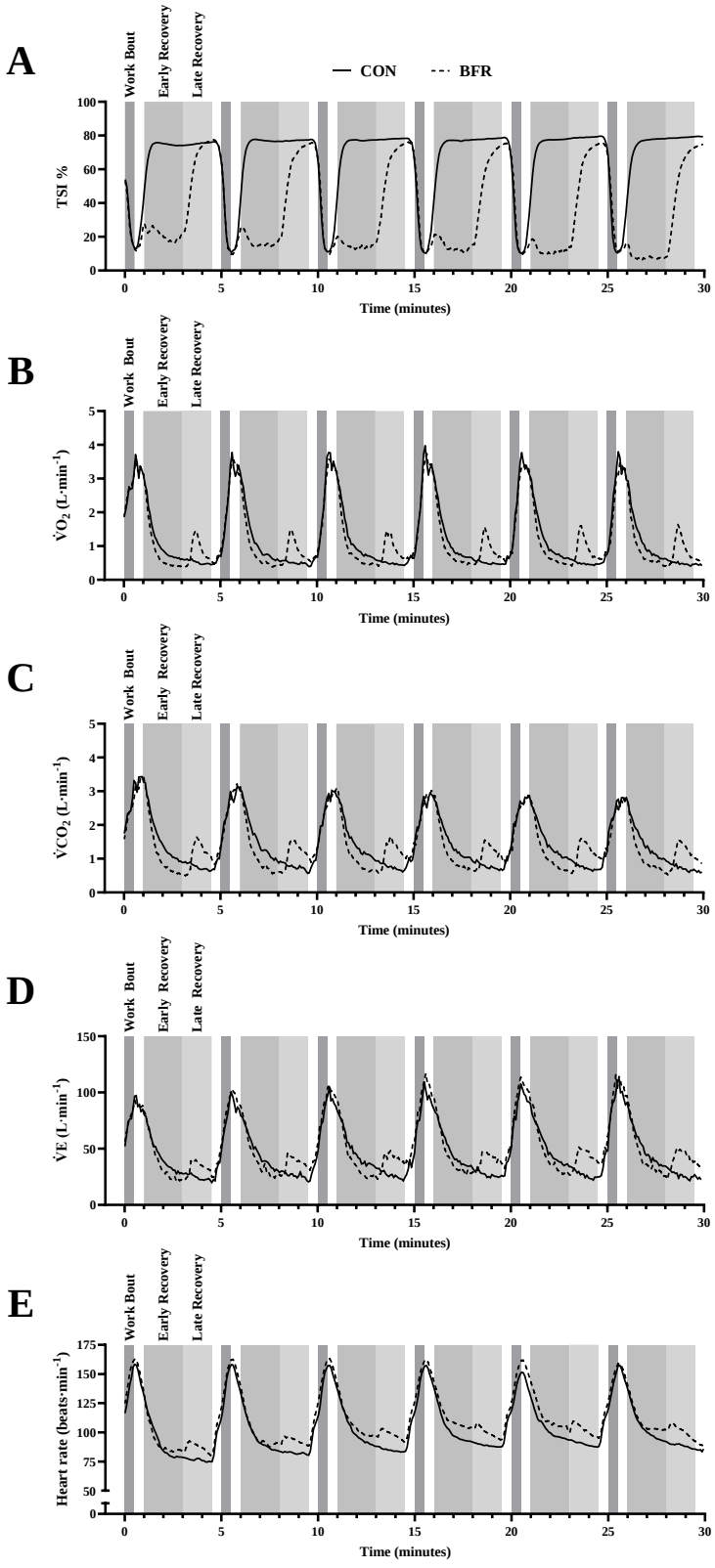


Figure 13. Mean 5-second traces of physiological parameters for BFR and CON interventions for an individual participant.

*Phases are labeled in the first repetition, with consistent shading thereafter. Panels: **A)** tissue saturation index (TSI); **B)** oxygen uptake ($\dot{V}O_2$); **C)** carbon dioxide production ($\dot{V}CO_2$); **D)** minute ventilation ($\dot{V}E$); and **E)** heart rate (HR) for blood flow restriction (BFR) and control (CON) conditions.*

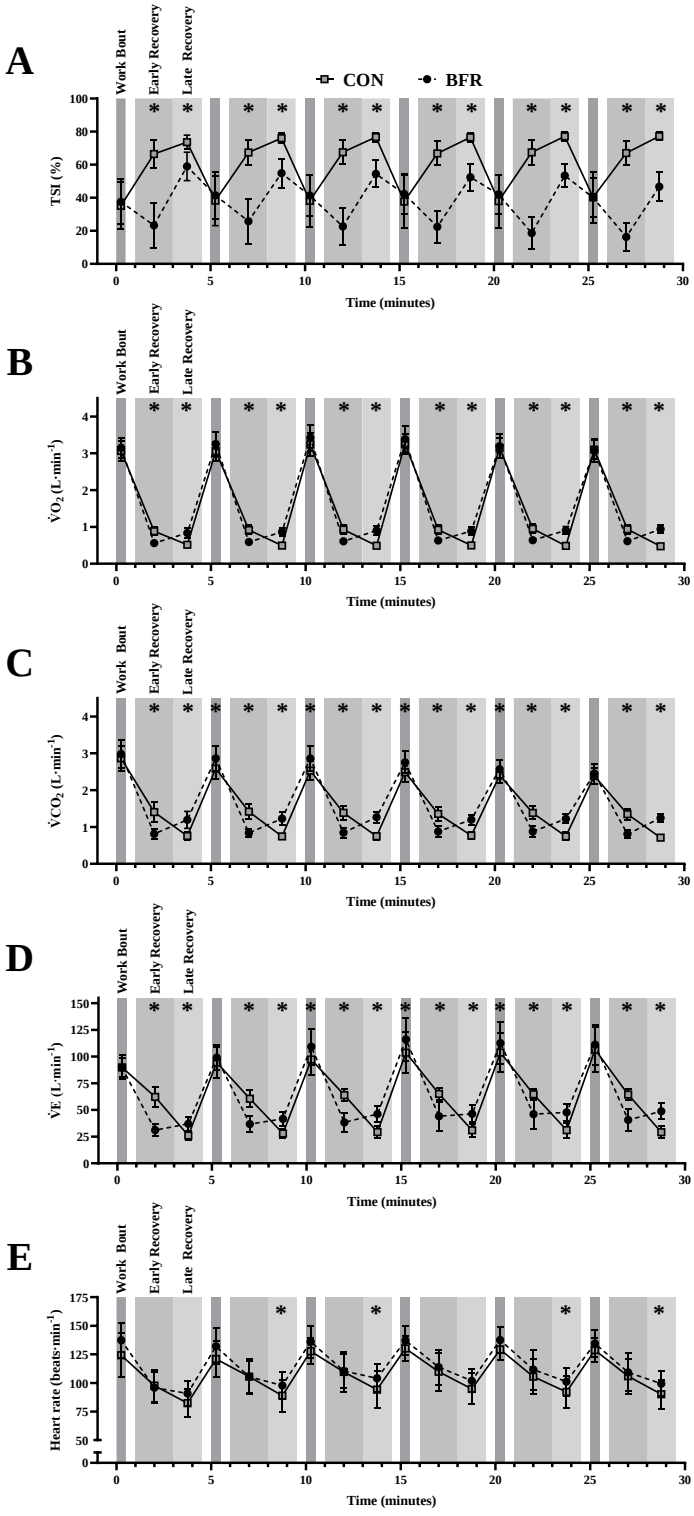


Figure 14. Physiological responses during BFR and CON interventions.

*Phases are labeled in the first repetition, with consistent shading thereafter. Panels: **A**) tissue saturation index (TSI); **B**) oxygen uptake ($\dot{V}O_2$); **C**) carbon dioxide production ($\dot{V}CO_2$); **D**) minute ventilation ($\dot{V}E$); and **E**) heart rate (HR) for blood flow restriction (BFR) and control (CON) conditions. Values are presented as mean ±95% confidence intervals. An asterisk (*) indicates a significant ($p<0.05$) post-hoc result between conditions.*

Table 7. Comparison of physiological parameters between BFR and CON interventions.

Measure	Averaging Period	Condition		Cohen's *d* for Condition Means
		BFR	CON	
TSI (%)	Entire Session	35.6 ±5.4*	64.0 ±2.4	**2.58 ±0.60**
	Entire Recovery	35.2 ±7.9*	71.0 ±4.7	**2.00 ±0.59**
	Work Bout	37.5 ±9.9	32.5 ±14.1	0.26 ±0.39
	Early Recovery	21.5 ±10.6*	67.1 ±7.5	**6.23 ±2.28**
	Late Recovery	53.5 ±7.5*	76.2 ±2.9	**5.77 ±2.58**
$\dot{V}O_2$ (L·minute^{-1})	Entire Session	1.21 ±0.10	1.22 ±0.10	0.05 ±0.21
	Entire Recovery	0.73 ±0.07	0.74 ±0.09	0.07 ±0.28
	Work Bout	3.35 ±0.35	3.25 ±0.30	0.24 ±0.32
	Early Recovery	0.61 ±0.07*	0.92 ±0.11	**2.11 ±0.37**
	Late Recovery	0.89 ±0.11*	0.49 ±0.06	**2.98 ±0.39**
$\dot{V}CO_2$ (L·minute^{-1})	Entire Session	1.40 ±0.14	1.43 ±0.15	0.12 ±0.20
	Entire Recovery	1.01 ±0.12*	1.11 ±0.15	**0.47 ±0.23**
	Work Bout	2.61 ±0.26*	2.80 ±0.32	**0.44 ±0.25**
	Early Recovery	0.84 ±0.12*	1.38 ±0.19	**2.22 ±0.28**
	Late Recovery	1.23 ±0.14*	0.74 ±0.10	**2.38 ±0.42**
$\dot{V}E$ (L·minute^{-1})	Entire Session	55.7 ±9.1	51.9 ±8.0	0.25 ±0.26
	Entire Recovery	41.7 ±7.9*	48.7 ±5.5	**0.60 ±0.40**
	Work Bout	109.0 ±17.1*	101.7 ±17.5	**0.29 ±0.21**
	Early Recovery	39.5 ±9.1*	63.4 ±6.2	**3.07 ±1.15**
	Late Recovery	44.6 ±6.8*	29.1 ±5.3	**1.50 ±0.43**
HR (beats·minute^{-1})	Entire Session	114 ±10*	106 ±11	**0.48 ±0.28**
	Entire Recovery	104 ±12	99 ±14	0.29 ±0.41
	Work Bout	134 ±12	127 ±10	0.34 ±0.60
	Early Recovery	108 ±14	106 ±15	0.10 ±0.32
	Late Recovery	99 ±10*	90 ±13	**0.42 ±0.34**

Mean ±95% CI of tissue saturation index (TSI), oxygen uptake ($\dot{V}O_2$), carbon dioxide output ($\dot{V}CO_2$), minute ventilation ($\dot{V}E$), and heart rate (HR) at each time phase. Asterisks (*) indicate significant difference between blood flow restriction (BFR) and control (CON) conditions at that time point ($p \leq 0.05$). Effect sizes for significant effects are presented in ***bold.***

Tissue saturation index (TSI)

Mean TSI was significantly reduced with BFR compared to CON ($p<0.001$), with a *large* ($d=2.58$) effect size. During work bouts, TSI did not differ between conditions ($p=0.496$). However, TSI was significantly reduced with BFR during early ($p<0.001$) and late ($p<0.001$) recovery compared to CON, with *large* ($d=6.23$ and 5.77, respectively) effect sizes. There was a *large* ($d=2.00$) and significant reduction from BFR during the entire recovery TSI ($p<0.001$), compared to CON. The interaction between condition and repetition on TSI was non-significant ($p=0.093$). There was no main effect of repetition on TSI ($p=0.396$).

$\dot{V}O_2$

$\dot{V}O_2$ during the work bout was not significantly affected by conditions (p=0.193). However, BFR lead to a significant and *large* (*d*=2.11) reduction in early recovery $\dot{V}O_2$ (p<0.001), and a significant and *large* (*d*=2.98) increase in late recovery $\dot{V}O_2$ (p<0.001), compared to CON. Total recovery $\dot{V}O_2$ did not differ between conditions (p=0.469). There was a significant main effect of repetition on $\dot{V}O_2$ (p=0.040). *Post-hoc* analysis demonstrated that the 4th repetition was greater than the 2nd (p=0.014). There was no significant interaction between condition and repetition on $\dot{V}O_2$ (p=0.274). Mean session $\dot{V}O_2$ did not differ between BFR and CON (p=0.540).

$\dot{V}CO_2$

There was a *small* (*d*=0.44) but significant increase in work bout $\dot{V}CO_2$ with BFR compared to CON (p=0.010). With BFR, there was a *large* (*d*=2.22), significant decrease in early recovery $\dot{V}CO_2$ (p<0.001) and a *large* (*d*=2.38), significant increase in late recovery $\dot{V}CO_2$ (p<0.001), compared to CON. There was a *small* (*d*=0.47) but significant reduction in entire recovery $\dot{V}CO_2$ with BFR (p=0.001). There was a significant main effect of repetition on $\dot{V}CO_2$ (p=0.013). *Post-hoc* analysis demonstrated that $\dot{V}CO_2$ in the 5th repetition was less than the 3rd (p=0.018). There was no significant interaction between condition and repetition on $\dot{V}CO_2$ (p=0.192). Mean session $\dot{V}CO_2$ did not differ between conditions (p=0.478).

$\dot{V}E$

There was a *small* (*d*=0.29) but significant increase in work bout $\dot{V}E$ with BFR compared to CON (p=0.044). Compared to CON, there was a significant and *large* (*d*=3.07) reduction in $\dot{V}E$ during early recovery with BFR (p<0.001), whereas late recovery $\dot{V}E$ was significantly increased in BFR (p<0.001) with a *large* (*d*=1.50) effect size. An interaction between condition and repetition (p=0.012) indicated that with BFR, $\dot{V}E$ was greater in the 3rd compared to 1st repetition (p=0.044), and in the 3rd compared to 2nd repetition (p=0.049). In CON, $\dot{V}E$ was greater in the 4th compared to 2nd repetition (p=0.009), and

in the 5th compared to the 2nd (p=0.031). *Post-hoc* tests for the simple main effect of condition at each repetition showed V̇E was greater in CON compared to BFR during the 1st repetition (p<0.001). There was a significant main effect of repetition on V̇E (p=0.004), with *post-hoc* tests showing V̇E was greater in the 3rd than the 2nd repetition (p=0.031). During the entire recovery, there was a significant and *moderate* (*d*=0.60) reduction in V̇E for BFR (p=0.008), compared to CON. Mean session V̇E did not differ between conditions (p=0.164).

HR

There was no significant effect of condition on work bout HR (p=0.127) or early recovery HR (p=0.600). Compared to CON, BFR led to a *small* (*d*=0.42) but significant increase in HR during late recovery (p=0.028). During the entire recovery, there was no significant differences in HR between conditions (p=0.192). There was no significant interaction between condition and repetition on HR (p=0.873). There was a *small* (*d*=0.48) but significant increase in mean session HR for BFR compared to CON (p=0.017). There was no main effect of repetition on HR (p=0.054).

VEGF

There was a significant interaction effect between condition and time on serum VEGF concentration (p=0.040). There was a *small* (*d*=0.31 ± 0.18) but significant increase in serum VEGF concentration from baseline to 3-hours post-HIIT in the BFR condition (Figure 15, Panel A). *Post-hoc* analyses of change revealed a greater baseline to post-intervention increase in the BFR condition (19 ± 11 pg·mL^{-1}) compared to the CON condition (-1.9 ± 14 pg·mL^{-1}) with a *large* effect size (*d*=0.91 ± 0.86) (Figure 15, Panel B).

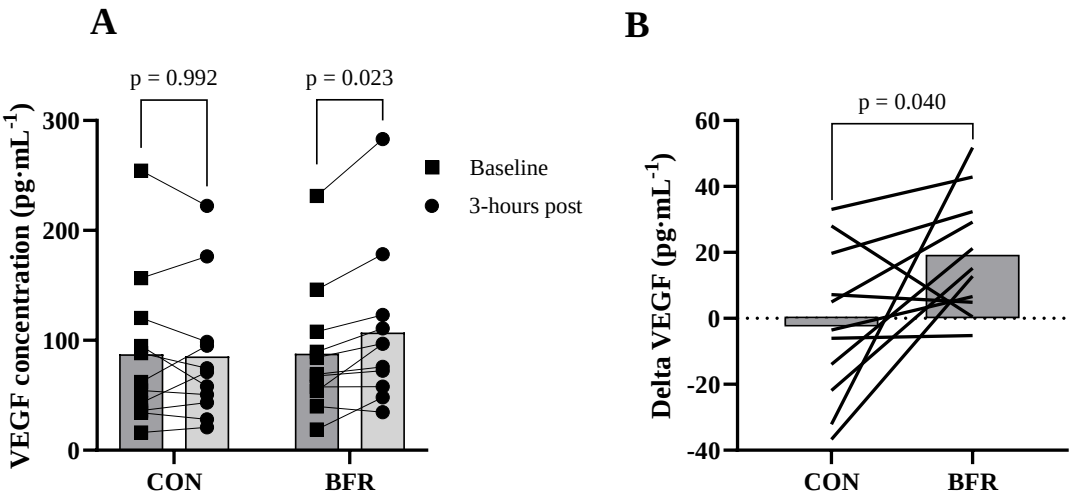


Figure 15. Acute vascular endothelial growth factor responses for BFR and CON.

*Panels: **(A)** Serum vascular endothelial growth factor (VEGF) at baseline and 3-hours post-exercise for blood flow restriction (BFR) and control (CON) conditions. **(B)** Change in VEGF (post minus baseline) for BFR and CON. Bars are means. Brackets with p-values show between-condition comparisons at each timepoint in **A**. The p-value in **B** indicates the condition × time interaction.*

Discussion

Here we demonstrate that acute application of BFR during the early recovery phases of HIIT elicits significant reductions in TSI, $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}E$ during the BFR occlusion phase. Serum VEGF concentration was increased to a greater extent following HIIT with BFR compared to control. Upon reperfusion in the BFR condition during the late recovery phase, $\dot{V}O_2$, $\dot{V}CO_2$, $\dot{V}E$, and HR rose beyond CON levels, while TSI remained reduced. The HIIT work bouts were unaffected by BFR in terms of power output, TSI, $\dot{V}O_2$, and HR. Although, BFR increased $\dot{V}CO_2$ and $\dot{V}E$ during the work bouts compared to CON. Over the entire HIIT protocol, BFR decreased TSI and increased HR, with no changes in $\dot{V}O_2$, $\dot{V}CO_2$, or $\dot{V}E$. A progressive increase in $\dot{V}E$ with successive work bouts was observed with BFR. Collectively, these findings confirm our hypothesis by demonstrating BFR can acutely manipulate physiological responses when BFR is integrated into HIIT recovery phases.

In the early recovery phase, the substantial TSI reduction in the BFR condition aligns with previous studies applying BFR during recovery from high-intensity exercise (McManus *et al.* 2018, Ienaga *et al.* 2022). The ~68% decrease in TSI during the early recovery phase exceeded the ~11% reduction

reported by Mitchell (2019) despite the present study using a lower relative intensity, potentially reflecting Mitchell's lower occlusion pressure compared to the present study (~125 vs 200 mmHg, respectively). The BFR-induced attenuation of TSI and $\dot{V}O_2$ imply constrained oxidative metabolism while diminished $\dot{V}CO_2$ implies reduced metabolite clearance from isolated peripheral circulation (Loenneke *et al.* 2010), and is consistent with the 'metabolic freeze' phenomenon (Okita *et al.* 2019). Further, the BFR-induced reduction in TSI and $\dot{V}O_2$ following high-intensity cycling suggests local hypoxia, which may stimulate the angiogenic response and is mediated, in part, by an increase in the activity of HIF1-α (Rey and Semenza 2010). Previous research shows that intramuscular stress elicited by moderate-load resistance exercise with subsequent occlusion is caused by restricted phosphocreatine resynthesis and proton accumulation (Okita *et al.* 2019). These homeostatic perturbations may activate the AMP-activated protein kinase (AMPK), which in turn triggers the phosphorylation of PGC-1α (Cantó *et al.* 2010), a key step in promoting mitochondrial biogenesis (Coffey and Hawley 2007). Thus, rapidly inducing ischemia via BFR after high-intensity exercise restricts recovery processes, and may augment both physiological and metabolic stress, and downstream angiogenic and mitochondrial adaptive signalling. Herein, we provide evidence of an enhanced physiological stress in comparison to exercise alone.

The marked increases in $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}E$ during reperfusion in the BFR late recovery phase indicate re-established peripheral and systemic circulation. The rapid TSI increase reflects restored O_2 delivery, enabling oxidative phosphorylation and phosphocreatine resynthesis, as demonstrated by indices of muscle oxidative capacity when TSI is high (Pilotto *et al.* 2022). As phosphocreatine recovery kinetics align with $\dot{V}O_2$ after exercise (Hargreaves and Spriet 2020), rephosphorylation likely contributes to the elevated $\dot{V}O_2$ in the present study. The elevated $\dot{V}CO_2$ during reperfusion reflects the requirement for bicarbonate buffering of hydrogen ions accumulated during ischemia, as demonstrated previously (Okita *et al.* 2019). Therefore, the elevated $\dot{V}O_2$, $\dot{V}CO_2$, and $\dot{V}E$ following reperfusion facilitate normalisation of intramuscular pH while enabling metabolic and oxidative recovery prior to subsequent work bouts. Repeated HIIT bouts with BFR can exacerbate metabolic accumulation and fatigue (McClean *et al.* 2023), evidenced here by a higher $\dot{V}CO_2$ and $\dot{V}E$ with BFR in subsequent bouts.

However, there was evidence of greater physiological stress but not decreased exercise tolerance, as power output was similar between conditions through all repetitions.

The elevated serum VEGF concentration 3-hours following BFR provides putative evidence of an augmented angiogenic response (Hoier *et al.* 2014). While skeletal muscle cells contain vesicular stores and secrete substantial amounts of VEGF in response to muscle contractions (Høier *et al.* 2010), the significant increase in circulating VEGF following BFR is an encouraging finding. The elevated VEGF with BFR exercise may result from adaptive responses to reduced TSI caused by vascular occlusion as well as shear stress on the endothelium induced by compression of blood vessels (Hudlicka *et al.* 2009). Provided the acute elevation in serum VEGF also manifests as a chronic elevation with repeated BFR sessions, a sustained increase in circulating VEGF may induce long-term adaptations in skeletal muscle O_2 delivery and metabolite clearance (Tesch *et al.* 1983). Specifically, previous research has demonstrated that 3 weeks of daily resistance training (20% 1RM) performed to failure with concurrent BFR (100 mmHg) augments muscle O_2 delivery and capillarisation compared to work-matched unoccluded exercise (Nielsen *et al.* 2020). Furthermore, Mitchell et al. (2019) demonstrated that BFR (120 mmHg) applied during the recovery phases of a 4 week maximal sprint interval training intervention increased $\dot{V}O_{2max}$ to a greater extent than sprint interval training alone. The submaximal HIIT protocol used in the present study elicited an unchanged VEGF response in CON. Thus, BFR appears necessary to augment the VEGF response resulting from the current HIIT protocol in the present trained cyclist cohort.

A limitation of the present study is the lack of a sham condition where low, non-occlusive pressure BFR would have been applied, which would have controlled for response bias impacting the results. Additionally, the BFR cuff pressure was uniformly set at 200 mmHg rather than adjusted per individual using arterial occlusion pressure assessments, meaning that the degree of arterial occlusion varied between participants (Loenneke *et al.* 2012, Hunt *et al.* 2016). The increase in circulating VEGF cannot be directly interpreted as enhanced VEGF protein expression in the exercised muscle. It is also acknowledged that serum VEGF exhibits substantial individual variation, and that the pre-post blood sampling at two timepoints provides limited insight on the sustainability of the observed changes (Kraus

et al. 2004). Additionally, serum VEGF may not accurately reflect muscle VEGF mRNA expression, as skeletal muscle cells increase VEGF secretion in response to contractile activity (Høier *et al.* 2010). Furthermore, while a 3–7-day washout period was implemented between experimental trials, residual effects from the previous trial cannot be entirely ruled out. Finally, a single pair of BFR cuffs were used across all participants, leading to relatively different coverage of the upper thigh and overlap of cuffs between participants, which may have influenced the degree of occlusion (Bielitzki *et al.* 2021).

Our findings indicate that incorporating BFR into a HIIT training regimen resulted in acute physiological changes while preserving a high-intensity exercise stimulus. Importantly, participants could tolerate the BFR intervention, and BFR did not impact the completion of their workouts. In fact, BFR occlusion only affected work bout power output, $\dot{V}E$ and $\dot{V}CO_2$ with no impact on work bout $\dot{V}O_2$, TSI, or HR. Our use of a standardised 200 mmHg BFR pressure allows for replication of this study in the field with minimal equipment, despite potential differences in the degree of occlusion between individuals. Coaches and athletes may consider integrating BFR into the recovery phases of HIIT to strategically modulate responses and induce targeted adaptations, depending on their goals and individual training programs. Given the endurance-based nature of intermittent high-intensity cycling events (Pugh *et al.* 2022), the augmented physiological and metabolic stress responses from the present intervention may translate to additional muscular and cardiovascular adaptations with consistent training. Longitudinal intervention studies are necessary to substantiate the efficacy of the present study's intervention over a full training cycle to elucidate benefits for endurance athletes.

Conclusions

In conclusion, BFR during HIIT recovery intervals induced greater acute physiological perturbations compared to HIIT with unoccluded recovery in trained cyclists. Specifically, BFR reduced TSI, $\dot{V}O_2$, and $\dot{V}CO_2$ during the early recovery phase, while increasing $\dot{V}O_2$ and $\dot{V}CO_2$ in late recovery. There was also a greater concentration of serum VEGF 3-hours post HIIT with BFR compared to CON. These greater physiological stressors resulting from BFR have the potential to augment adaptive mechanisms

that could benefit high-intensity exercise performance and endurance. Further research is warranted to investigate whether the acute effects of adding BFR to HIIT translate to measurable long-term performance improvements in trained cyclists across a training period.

Chapter Five – Four-weeks of blood flow restriction between high-intensity interval repetitions improves performance in trained cyclists: a randomised, between-subject trial

Prelude

The findings from Chapter Four demonstrated the potential benefits of integrating BFR between high-intensity intervals to augment the physiological stress and enhance the training stimulus. Building on those promising acute findings, this chapter aims to assess the efficacy of applying an integrated BFR and HIIT intervention over a prolonged period. Specifically, it examines the chronic effects of this training intervention on key physiological factors related to TP performance, similar to those identified as important in Chapter Three.

Abstract

Applying blood flow restriction (BFR) during the recovery phases between intervals in high-intensity interval training (HIIT) increases acute metabolic and physiological stress. However, the chronic effects of this integrated BFR and HIIT protocol on intermittent exercise performance are unclear. This study examined whether applying BFR during HIIT recovery phases over a 4-week training period enhances physiological determinants and intermittent cycling performance compared to HIIT alone in trained cyclists.

Ten trained male cyclists (age: 27 ± 8 y, body mass: 72.6 ± 13.4 kg, peak O_2 uptake, $\dot{V}O_{2PEAK}$: 59 ± 7 $mL \cdot kg^{-1} \cdot minute^{-1}$) completed eight HIIT sessions (6x30-s efforts, 4.5-minutes recovery) over four-weeks, either with (BFR) or without (CON) thigh occlusion at 200 mmHg applied for 2-minutes between each HIIT repetition. Pre- and post-intervention, cyclists completed assessments of lactate threshold, 30-s maximal power output, and an intermittent cycling test.

Two-way ANOVAs revealed significant condition-by-time interactions, indicating greater improvements with BFR compared to CON for lactate threshold, 30-s mean power output, accumulated intermittent work, muscle reoxygenation half-time, and $\dot{V}O_{2PEAK}$ during the intermittent test (all $p<0.05$). From pre- to post-intervention, BFR increased power output at baseline blood [lactate] +1 $mmol \cdot L^{-1}$ (7 ±5%), accumulated work in the intermittent test (55 ±44%) and $\dot{V}O_{2PEAK}$ in the intermittent test (7 ±2%), while reducing muscle reoxygenation half-time (26 ±25%) (all $p<0.05$). In contrast, the CON condition showed no significant performance changes.

These findings suggest that incorporating BFR into HIIT recovery phases may improve key physiological determinants of intense intermittent cycling performance, such as aerobic capacity, glycolytic power, and indices of recovery, in trained cyclists. However, given the small sample size, these results should be considered preliminary. Further research with larger samples is warranted to confirm if strategically implementing BFR with HIIT further augmented adaptations and high-intensity intermittent cycling performance beyond HIIT alone.

Introduction

Endurance track cycling events, such as the team-pursuit, demand exceptional aerobic power and the ability to repeatedly produce high-intensity efforts (Pugh *et al.* 2022). Specifically, team-pursuit cyclists require an excellent peak O_2 uptake ($\dot{V}O_{2PEAK}$) to sustain power output over 4-km, sufficient glycolytic power to meet high power requirements, and an elevated lactate threshold to better tolerate metabolite accumulation (Van Der Zwaard *et al.* 2018). Therefore, team-pursuit cyclists may employ a combination of submaximal training and high-intensity interval training (HIIT) (Schumacher *et al.* 2002) to develop aerobic and high-intensity capacities (Christensen *et al.* 2023).

While high-volume, low-intensity training can enhance endurance and fatigue resistance in highly-trained endurance athletes (Seiler and Kjerland 2006, Maunder *et al.* 2021), increasing low-intensity training volume does not necessarily lead to improved endurance performance (Costill *et al.* 1991). In contrast, although performed in lower volumes, HIIT can be more effective than low-intensity training for enhancing $\dot{V}O_{2PEAK}$, muscle oxidative capacity, and muscle buffering capacity (Laursen *et al.* 2002a, Gibala *et al.* 2009). However, the magnitude of physiological adaptations to a given HIIT stimulus may plateau after 8-12 sessions (Paton *et al.* 2005) or 3-weeks (Norrbom *et al.* 2022) without progression in volume or intensity.

Augmenting HIIT sessions with an additional stimulus, such as blood flow restriction (BFR), is a proposed strategy to progress chronic physiological adaptations without progressive mechanical overload. By applying a pneumatic cuff or tourniquet proximally on a limb, BFR restricts venous blood flow and exacerbates intramuscular metabolites perturbations (Suga *et al.* 2012). Integrating BFR into exercise can enhance metabolic stress (Okita *et al.* 2019) and has demonstrated effectiveness in promoting muscular hypertrophy and strength gains during low-load resistance exercises (Vissing *et al.* 2020, Pignanelli *et al.* 2021). Studies also suggest a positive effect of BFR in promoting microvascular and mitochondrial adaptations, even when training loads are reduced (Groennebaek *et al.* 2018, Nielsen *et al.* 2020). Consequently, strategically combining BFR with HIIT may escalate metabolic and oxidative stress to facilitate progressive adaptations beyond what can be achieved with HIIT alone (Ross *et al.* 2023).

However, applying BFR during high-intensity exercise may provoke intolerable discomfort (Willis *et al.* 2018) and hinder the high-intensity mechanical stimulus thought necessary for developing high-intensity performance (Bangsbo 2015). Instead, cyclists training for repeated high-intensity efforts, like team-pursuit, may utilise BFR during recovery between high-intensity intervals. This approach may increase metabolic and physiological stress while maintaining competition-specific intensity during work intervals. Previous studies in endurance trained athletes have shown that applying BFR between repeated 30-s sprints for eight sessions improved incremental test $\dot{V}O_{2PEAK}$ (Taylor *et al.* 2016, Mitchell *et al.* 2019), suggesting increased maximal cardiac output, peripheral O_2 extraction, or both (Ferguson *et al.* 2021). Additionally, six sessions of BFR applied between repeated 10-s sprints improved relative $\dot{V}O_{2PEAK}$ during a ~2.5-minute time to exhaustion test (Giovanna *et al.* 2022), implying peripheral rather than central adaptations (Skattebo *et al.* 2020).

While the existing literature indicates that BFR applied during recovery may improve measures of aerobic capacity, the effects on intermittent high-intensity cycling performance remain unclear. Therefore, the present study aimed to investigate the chronic physiological and performance adaptations from a four-week HIIT program integrating BFR during recovery phases. We hypothesised that this training approach would improve lactate threshold parameters, 30-s maximal sprint performance, and intermittent high-intensity performance compared to HIIT alone.

Methods

Participants

Ten trained male cyclists volunteered for the study (age: 27 ± 8 y, height: 179 ± 7 cm, body mass: 72.6 ± 13.4 kg, $\dot{V}O_{2PEAK}$: 59 ± 7 $mL \cdot kg^{-1} \cdot minute^{-1}$, 4.5 ± 0.6 $L \cdot minute^{-1}$). While a priori sample size calculations are ideal, we recruited the maximum feasible sample of trained cyclists within our budget and timeline constraints. All participants regularly trained >6-$hours \cdot week^{-1}$ and competed at club or national level. Prior to testing, participants completed medical and BFR screening questionnaires to ensure good health and the absence of injury (Patterson *et al.* 2019). All participants gave their written

informed consent to complete the study, which was approved by the participating institutions' human research ethics committee (ethics approval code: HREC(HECS)2021#40) and conformed to the Declaration of Helsinki.

Experimental design

A parallel-group controlled trial compared the effects of a four-week, twice-weekly HIIT program performed with or without BFR during HIIT recovery periods. Participants were pair-matched based on cycling proficiency standards defined by De Pauw *et al.* (2013) to evenly distribute cycling ability between the BFR (BFR, n=5) and control (CON, n=5) conditions. The matching process resulted in the following distribution of proficiency tiers across conditions: tier 3, n=3; tier 4, n=2.

All tests and training sessions were performed in a temperature (19 ± 2 °C) and humidity (45 ± 5% RH) controlled laboratory at the same time of day for each participant to minimise diurnal variation. Visits were separated by ≥48-hours. Participants used their personal bicycles mounted on a calibrated stationary ergometer (Kickr V5, Wahoo, Atlanta, GA, USA), with resistance controlled by commercial software (TrainerRoad, Reno, NV, USA). To standardise dietary intake, participants recorded nutrition in food diaries and were instructed to consume the same diet in the 24-hours before each visit.

Performance tests

Participants completed pre- and post-intervention tests on two separate days. On the first test day, a lactate threshold test was conducted, followed by a 15-minute rest period and a maximal 30-s sprint test. On the second test day, an intermittent high-intensity test was performed (details below).

Stepped test

Participants performed an incremental stepped cycling test starting at 120 W and increasing by 30 W every 4-minutes until exhaustion. The test was performed at a pedal cadence of 80-100 RPM, which participants were required to maintain throughout. Exhaustion was defined as failing to sustain ≥80 RPM for >3-s despite verbal motivation to increase cadence. Capillary blood samples were collected from the earlobe in the last 30-s of each stage and analysed immediately for lactate concentration (Lactate Pro 2, Arkray, Kyoto, Japan). A lactate-power curve was produced for each participant with lactate threshold parameters defined as the power output corresponding to an increase of 1 $mmol \cdot L^{-1}$ (LT1; (Thoden 1991)), and at a fixed blood [lactate] of 4 $mmol \cdot L^{-1}$ (FBLA4; (Faude *et al.* 2009)). These thresholds were all determined objectively using Lactate-E software (Newell *et al.* 2007). Additionally, breath-by-breath pulmonary gas exchange was measured continuously throughout the test. The highest continuous 30-s $\dot{V}O_2$ during the stepped threshold test defined $\dot{V}O_{2PEAK\text{-}STEP}$. Cycling efficiency was calculated using $\dot{V}O_2$ and $\dot{V}CO_2$ measures.

Maximal 30-s test

Participants performed a 30-s maximal sprint test with the ergometer set to an isoinertial mode. A 10-s sprint trial 5-minutes prior established appropriate resistance and gear settings for each participant's subsequent 30-s maximal effort. Participants were required to pedal with a cadence of 120 RPM before resistance was applied at the start of the test. Power output was recorded at 1 Hz with peak power output (highest 1-s power output: PPO) and mean power output (average 30-s power output: MPO) recorded. Fatigue index (FI) was calculated as the rate of power output decline from PPO to minimum power relative to the 30-s duration expressed in $W \cdot sec^{-1}$. Pilot testing found the power output during lead positions of a team-pursuit approximates 85% of the MPO during a maximal 30-s test. Thus, an individual's 30-s test MPO was multiplied by 0.85 to establish training intensity for the experimental trials. Data from our laboratory (unpublished observations) showed that most cyclists could complete at least six repetitions at 85% of their 30-s MPO within 30-minutes, which informed the training protocol.

Intermittent exercise test

An intermittent cycling test was designed to simulate the physiological demands of a team-pursuit event. The test included work bouts at a similar duration and intensity to the leading phases of a team-pursuit, with recovery phases of comparable duration but standardised at an intensity equivalent to LT1. The intermittent test began with a 20-minute standardised warm-up phase, consisting of a progressive intensity protocol starting at 50% of LT1 power and incrementally increasing to 90% of the power output at FBLA4 over 10-minutes. This stage was followed by 2-minutes at FBLA4 intensity, 10-s at 85% of 30-s test MPO, and the remaining warm-up at 50% LT1. Immediately following the warm-up, the test phase began, which involved repeated 30-s high-intensity intervals at 85% of 30-s test MPO, interspersed with 1-minute recovery periods at LT1 intensity. The test continued until volitional exhaustion or cadence dropped below 80 RPM for >3-s despite verbal motivation to increase.

Performance in the intermittent test was assessed by measuring the total work (in kJ) accomplished during the test and the time-to-exhaustion (TTE, in s). Breath-by-breath pulmonary gas exchange was measured continuously throughout the test. Performance peak $\dot{V}O_2$ ($\dot{V}O_{2PEAK\text{-}PERF}$) was defined as the highest continuous 15-s $\dot{V}O_2$ during the intermittent test. Vastus lateralis muscle O_2 saturation data were measured continuously throughout the test. The post-intervention intermittent test used identical intensities in the warm-up, work, and recovery phases as the pre-intervention test.

Physiological measurements and data handling

Breath-by-breath expired respiratory gases were collected during the stepped test using a metabolic cart (Parvo Medics TrueOne 2400, Salt Lake City, Utah, USA), calibrated to the manufacturer's instructions. Gas calibration was performed with an Alpha standard 16.0% O_2 and 4.0% CO_2 gas mixture. The data were transferred to Excel (Microsoft, Redmond, VA, USA) for analysis and time-alignment to the test start. Energy expenditure was estimated via the Péronnet and Massicotte (1991) equation using the final 2-minutes of respiratory data from each stage where the respiratory exchange ratio was <1.0. Gross efficiency (GE) was defined as the proportion of external mechanical work done relative to overall energy expenditure. Delta efficiency (DE) was computed as the reciprocal of the

slope from the regression line between external mechanical work and energy expenditure (Coyle *et al.* 1992).

Near-infrared spectroscopy (NIRS) measurements were taken during the intermittent test using two NIR Moxy 3 sensors (Moxy 3, Fortiori Design LLC, Minnesota, USA). Each sensor was fastened with a compression bandage on the left and right vastus lateralis muscles at 40% of the distance from the hip's greater trochanter to the knee's lateral epicondyle. Raw 1 Hz muscle tissue saturation index (TSI) data were recorded using a cycling computer (Edge 520, Garmin, Olathe, Kansas, USA). Muscle oxygenation data were downloaded to Golden Cheetah (version 3.5, open source, https://www.goldencheetah.org/) and exported to Excel for anomaly correction and averaging across both legs. Ergometer power output (in Watts, W) data were collected at 1 Hz and aligned from exercise onset using event markers.

To assess the half-time of TSI recovery, we plotted TSI data against time for each intermittent test recovery phase and evaluated the relationships with a monoexponential regression. The exponential relation used for this purpose was:

$$TSI(t) = Ae^{-kt} + C \qquad \text{Equation 15}$$

Where *t* is time (in s), *A* is the initial TSI difference from baseline, *k* is the rate constant (slope), and *C* is the asymptotic baseline. The time constant (τ) was 1/*k*. Half-time was defined as ln(2)*τ. The correlation coefficients (r) of the time-TSI relationships for each recovery period varied from 0.99 to 0.84 (0.94 ± 0.05).

Training intervention

Participants were instructed to refrain from HIIT and resistance training outside of the supervised experimental sessions. After the four-week intervention, a recovery week was prescribed to all participants, in which participants would continue with their personal training but without HIIT or resistance training. To ensure adherence to the prescribed exercise protocols, participants' personal

training activities were monitored, and compliance was verified through review of the participants' training logs.

Training sessions began with a self-selected warm-up of at least 10-minutes. Participants then completed six 30-s high-intensity intervals at 85% of the 30-s test MPO, as demonstrated in Figure 16. A 4.5-minute recovery period followed each interval, during which participants dismounted and rested supine on a nearby bed.

In the experimental condition, BFR was applied by inflating 11 cm wide occlusion cuffs (Occlude, Aarhus, Denmark) to 200 mmHg proximally on the thighs. Cuffs were inflated using hand-operated sphygmomanometers within 30-s post-interval for 2-minutes, followed by passive recovery without cuffs for the remaining 1.5-minutes. The control condition did not receive BFR during recovery. Participants remounted the bicycle 30-s before the next interval.

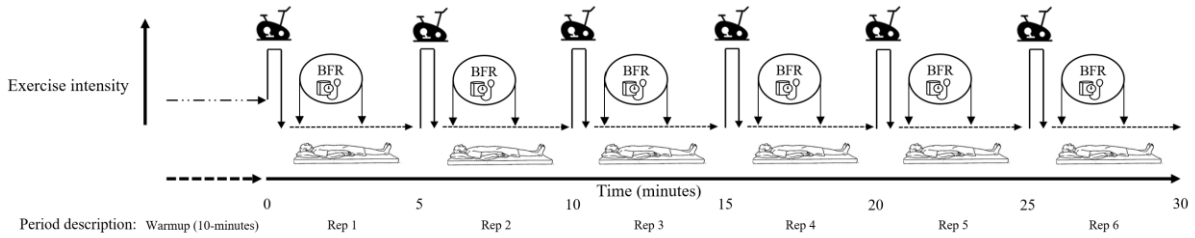


Figure 16. Timeline of training sessions.

The ergometer icon indicates when participants were mounted and exercising. The arrows adjoined to the BFR (blood flow restriction) oval indicate the start and finish of application during the BFR condition; the CON (control) condition rested supine without cuffs.

Statistical analysis

Performance variables were analysed using a two-way repeated-measures analysis of variance (RM-ANOVA), with time (pre- vs. post-intervention) and condition (BFR vs. CON) as the main factors. Normality was assessed using Shapiro-Wilk tests and sphericity was assessed with Mauchly's test, with both assumptions confirmed to not be violated ($p > 0.05$). The performance variables assessed were LT1, FBLA4, GE, DE, and $\dot{V}O_{2PEAK\text{-}STEP}$ from the threshold test; PPO, MPO, and FI from the 30-s test; and accumulated work, TTE, TSI recovery half-time, and $\dot{V}O_{2PEAK\text{-}PERF}$ from the intermittent test. Where

significant interaction effects were observed, Bonferroni-corrected *post-hoc* paired t-tests were used to locate differences. To assess the magnitude of the interaction effect (condition x time) for each variable, we calculated individual percentage change scores from pre- to post-intervention. The mean and standard deviation of these change scores were determined for each condition and variable. Where appropriate, we calculated the mean effect size (Cohen's d) between BFR and CON change scores to demonstrate the magnitude of the condition x time interaction for each variable. We also computed the effect sizes of any significant main effects of time or condition. Effect sizes were interpreted using the thresholds of 0.2, 0.5, and 0.8 for *small*, *moderate*, and *large*, respectively (Cohen 1988). An effect size of <0.2 was considered *trivial*. For all effect sizes, 95% confidence intervals were calculated to quantify the precision of the estimates. Two Pearson correlation analyses were utilised to assess the relationship between $\dot{V}O_{2PEAK\text{-}STEP}$ and $\dot{V}O_{2PEAK\text{-}PERF}$ in absolute and relative terms at pre- and post-intervention timepoints. The statistical analyses were performed using SPSS (Version 29.0, IBM, Armonk, NY), with statistical significance set at $p \leq 0.05$. Data are presented as mean ± SD unless otherwise stated. Graphs were produced in GraphPad Prism 9.5.1 for Windows (GraphPad Software, San Diego, California, USA).

Results

There were no significant differences between conditions in pre-intervention performance measures ($p>0.05$). Over the four-week intervention period, the training loads for the supervised experimental sessions were comparable between the BFR and CON conditions, with 842 ± 182 kJ and 777 ± 71 kJ, respectively ($p=0.479$). The weekly training duration, including both the supervised experimental sessions and personal training, was not significantly different between BFR (10.5 ± 4.1-hours) and CON (10.8 ± 3.0-hours) conditions ($p=0.918$). Physiological and performance outcome comparisons for BFR and the CON conditions are summarised in Table 8.

Table 8. Physiological and performance variables pre and post blood flow restricted (BFR) and control (CON) training interventions.

	BFR		CON		Condition x Time Interaction	
	Pre	Post	Pre	Post	p-value	Effect Size *d* ±95%CI
Variable	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD		
LT1 (W)	270 ± 33	290 ± 40	267 ± 45	265 ± 38	**0.025***	1.98 ±1.51
FBLA4 (W)	298 ± 34	316 ± 45	291 ± 47	297 ± 39	0.152	1.06 ±1.32
Gross efficiency (%)	19.9 ± 1.6	20.6 ± 0.9	21.3 ± 1.4	21.7 ± 1.0	0.242	0.51 ±1.26
Delta efficiency (%)	23.0 ± 1.2	23.7 ± 1.5	22.6 ± 1.6	23.7 ± 2.9	0.620	0.20 ±1.24
$\dot{V}O_{2PEAK\text{-}STEP}$ ($L \cdot minute^{-1}$)	4.48 ± 0.55	4.72 ± 0.72	4.53 ± 0.53	4.57 ± 0.52	0.223	0.87 ±1.30
$\dot{V}O_{2PEAK\text{-}STEP}$ ($mL \cdot kg \cdot minute^{-1}$)	56.9 ± 7.1	59.8 ± 5.2	62.6 ± 4.9	63.3 ± 4.6	0.220	1.00 ±1.31
PPO (W)	799 ± 185	896 ± 239	707 ± 71	735 ± 121	0.591	0.96 ±1.31
30-s MPO (W)	688 ± 133	738 ± 138	635 ± 52	645 ± 64	**0.036***	1.61 ±1.43
30-s FI ($W \cdot s^{-1}$)	8 ± 2	11 ± 5	6 ± 1	8 ± 2	0.921	0.34 ±1.25
Accumulated work (kJ)	96.0 ± 37.8	137.5 ± 40.7	124.4 ± 60.6	129.4 ± 79.5	**0.029***	1.74 ±1.46
TTE (s)	271 ± 150	381 ± 173	341 ± 142	347 ± 192	**0.025***	1.58 ±1.42
TSI recovery half-time (s)	83.6 ± 63.3	51.8 ± 29.0	38.6 ± 20.4	45.0 ± 29.2	**0.045***	2.01 ±1.52
$\dot{V}O_{2PEAK\text{-}PERF}$ ($L \cdot minute^{-1}$)	4.82 ± 0.74	5.16 ± 0.85	4.81 ± 0.64	4.88 ± 0.79	**0.031***	2.27 ±1.59
$\dot{V}O_{2PEAK\text{-}PERF}$ ($mL \cdot kg \cdot minute^{-1}$)	61.0 ± 7.5	65.4 ± 7.8	66.3 ± 6.3	67.6 ± 7.8	**0.020***	2.34 ±1.61

Abbreviations: LT1, Initial rise of 1 $mmol \cdot L^{-1}$; FBLA4, power output at fixed blood lactate accumulation of 4 $mmol \cdot L^{-1}$; $\dot{V}O_{2PEAK\text{-}STEP}$, peak rate of oxygen uptake during the step test; PPO, peak power output; MPO, mean power output; FI, fatigue index; TTE, time to exhaustion; TSI, muscle tissue oxygen saturation index; $\dot{V}O_{2PEAK\text{-}PERF}$, peak rate of oxygen uptake during the intermittent test. Significant 'condition x time' interaction effects are presented in ***bold*** *with an asterisk (*).*

Lactate measurements

Significant interactions between condition and time was observed for LT1. *Post-hoc* analyses revealed LT1 demonstrated a greater increase from pre- to post-intervention in BFR (7.4 ± 4.7%) compared to the CON condition (-0.5 ± 3.1%), with a *large* effect size (d=1.30 ±0.88), as shown in Figure 17 (Panel A). For FBLA4 (Figure 17, Panel B), no significant interactions between condition and time or between-condition differences were found (p=0.637). However, a significant main effect of time was observed, indicating a *large* increase in FBLA4 from pre- to post-intervention across both BFR (5.9 ± 4.2%) and CON conditions (2.5 ± 2.9%) (p=0.013, d=0.93 ±0.64).

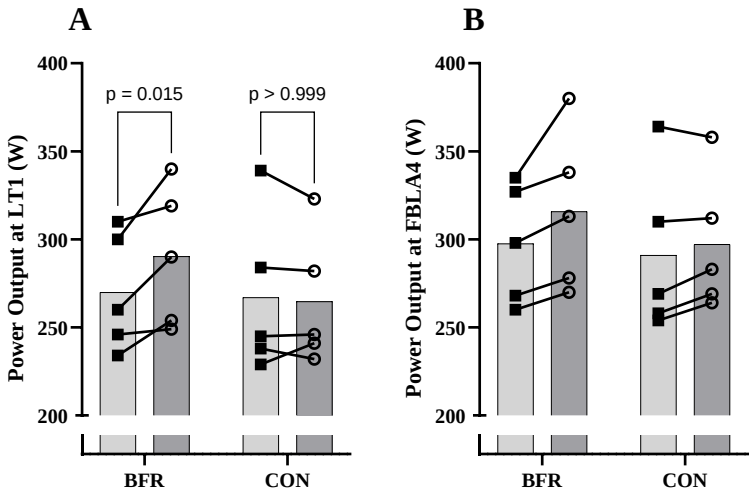


Figure 17. Lactate threshold parameters pre and post blood flow restricted (BFR) and control (CON) training interventions.

Panels: A) power output corresponding to a 1.0 mmol·L^{-1} rise in blood lactate concentration above baseline, LT1; B) power output at a fixed blood lactate concentration of 4.0 mmol·L^{-1}, FBLA4.

Efficiency

There were no significant interactions between condition and time for GE or DE, nor significant main effect of condition for GE or DE (all p>0.050). Additionally, there was no significant main effect of time for DE, yet there was a significant main effect of time for GE. *Post-hoc* analyses showed a *moderate* increase in GE from pre- to post-intervention (p=0.035, d=0.71 ±0.40), irrespective of condition.

$\dot{V}O_{2PEAK-STEP}$

For absolute and relative $\dot{V}O_{2PEAK-STEP}$, there were no significant interactions between condition and time, nor main effects of condition or time (all $p>0.05$).

Maximal 30-s test

For PPO, there was no significant interaction between condition and time, nor a significant main effect for condition or time (all $p>0.05$) (Figure 18, Panel A).

For MPO, there was a significant interaction between condition and time and a significant main effect of time (Figure 18, Panel B). *Post-hoc* tests revealed a *large* increase in MPO from pre- to post-intervention in the BFR condition (d=2.20 ±1.37) but no significant change in the CON condition. *Post-hoc* analyses for the main effect of time showed a *large* increase in MPO from pre- to post-intervention, irrespective of condition ($p=0.006$, d=0.94 ±0.44).

For FI, there was no significant interaction between condition and time, nor a significant main effect for condition or time (all $p>0.05$) (Figure 18, Panel C).

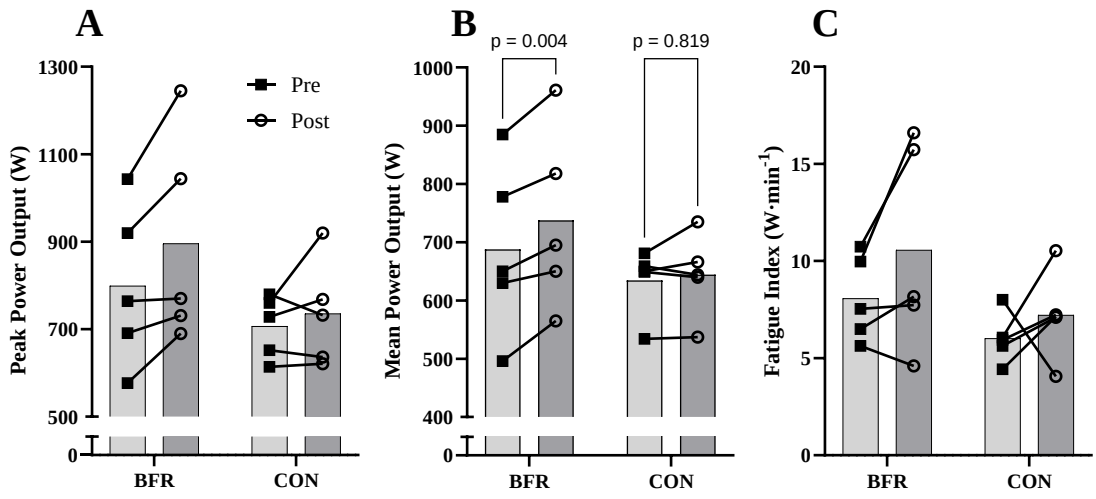


Figure 18. Maximal 30-s sprint parameters pre and post blood flow restricted (BFR) and control (CON) training interventions. Panels: A) peak power output (PPO); B) mean power output (MPO); C) Fatigue index (FI).

Intermittent test

For accumulated work, there was a significant interaction between condition and time, and a significant main effect of time (Figure 19, Panel A). *Post-hoc* tests revealed a *large* increase in accumulated work from pre- to post-intervention in the BFR condition (p=0.003, *d*=1.89 ±1.26), but no significant change for the CON condition (p=0.638). Irrespective of condition, there was a *large* increase in accumulated work from pre- to post-intervention (p=0.010, *d*=0.82 ±0.45).

For TTE, there was a significant interaction between condition and time and a significant main effect of time (Figure 19, Panel B). *Post-hoc* analyses revealed a *large* increase in TTE from pre- to post-intervention in the BFR condition (*d*=1.82 ±1.17), but no significant change for CON. Irrespective of condition, there was a *moderate* increase in TTE from pre- to post-intervention (p=0.016, *d*=0.73 ±0.42).

For TSI half-time, there was a significant interaction between condition and time (Figure 19, Panel C). *Post-hoc* tests revealed a *large* decrease in TSI half-time from pre- to post-intervention in the BFR condition (*d*=1.01 ±0.64), but no significant change for CON. Representative TSI traces during the intermittent test for BFR and CON are depicted in Figure 20.

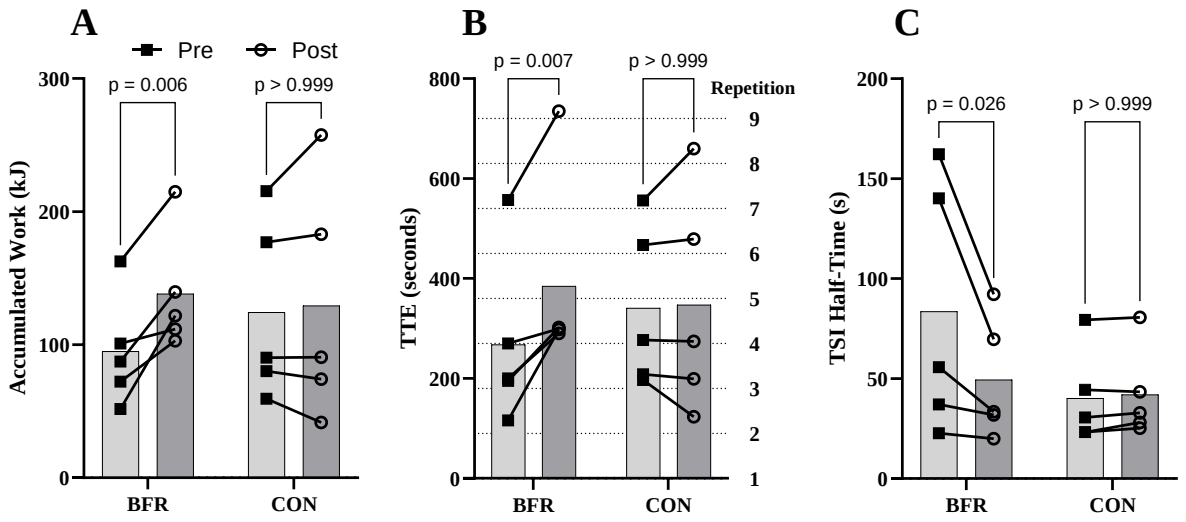


Figure 19. Intermittent trial parameters pre and post blood flow restricted (BFR) and control (CON) training interventions.

Panels: A) accumulated work; B) time to exhaustion (TTE) with repetitions completed indicated; C) muscle tissue oxygen saturation index (TSI) half-time.

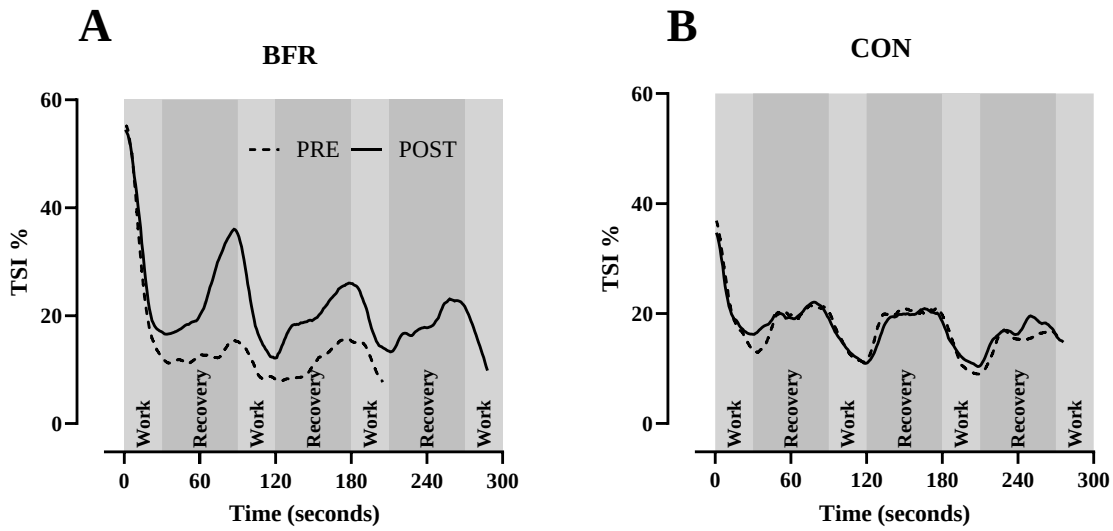


Figure 20. Individual traces of a representative participant's muscle tissue oxygen saturation index (TSI) during the intermittent test pre and post blood flow restricted (BFR) or control (CON) training interventions.

$\dot{V}O_{2PEAK\text{-}PERF}$

For absolute $\dot{V}O_{2PEAK\text{-}PERF}$, there was a significant interaction between condition and time and a significant main effect of time. *Post-hoc* tests showed a *large* increase in absolute $\dot{V}O_{2PEAK\text{-}PERF}$ from

pre- to post-intervention in the BFR condition (*d*=2.44 ±1.52), but not in the CON condition (Figure 21, Panel A). Irrespective of condition, there was a *large* increase in absolute $\dot{V}O_{2PEAK\text{-}PERF}$ from pre- to post-intervention (p=0.004, *d*=0.98 ±0.45). Absolute $\dot{V}O_{2PEAK\text{-}PERF}$ was significantly and positively correlated with absolute $\dot{V}O_{2PEAK\text{-}STEP}$ at both pre (p<0.001, R^2=0.96) and post-intervention (p<0.001, R^2=0.96).

For relative $\dot{V}O_{2PEAK\text{-}PERF}$, there was a significant interaction between condition and time and a significant main effect of time. *Post-hoc* tests revealed a *large* increase in relative $\dot{V}O_{2PEAK\text{-}PERF}$ from pre- to post-intervention in the BFR condition (*d*=3.78 ±2.35), but not in CON (Figure 21, Panel B). Irrespective of condition, there was a *large* increase in relative $\dot{V}O_{2PEAK\text{-}PERF}$ from pre- to post-intervention (p<0.001, *d*=1.23 ±0.57). Relative $\dot{V}O_{2PEAK\text{-}PERF}$ was significantly and positively correlated with relative $\dot{V}O_{2PEAK\text{-}STEP}$ at both pre- (p<0.001, R^2=0.93) and post-intervention (p<0.001, R^2=0.91). Note that absolute and relative values of $\dot{V}O_{2PEAK\text{-}PERF}$ were greater than $\dot{V}O_{2PEAK\text{-}STEP}$, reflecting the reduced test duration and $\dot{V}O_2$ averaging period in $\dot{V}O_{2PEAK\text{-}PERF}$ compared to $\dot{V}O_{2PEAK\text{-}STEP}$.

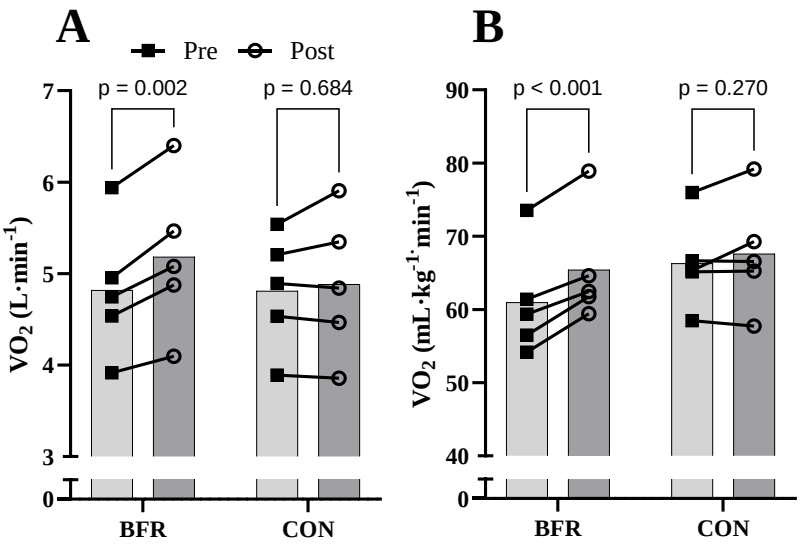


Figure 21. Performance $\dot{V}O_{2PEAK}$ during the intermittent test for blood flow restriction (BFR) and control (CON) conditions, pre- and post-intervention.

Panels: A) absolute $\dot{V}O_{2PEAK\text{-}PERF}$; B) relative $\dot{V}O_{2PEAK\text{-}PERF}$.

Discussion

The main finding of this study is that incorporating BFR during the recovery intervals of HIIT elicited preliminary evidence of improving several physiological determinants and performance markers related to intense intermittent cycling ability. Specifically, BFR significantly improved power output at LT1 by ~7%, indicating enhanced submaximal endurance capacity. Additionally, BFR led to a ~7% increase in 30-s MPO, suggesting improved glycolytic power. The BFR condition also augmented intermittent high-intensity cycling performance, with significant increases of ~55% in accumulated intermittent work and ~7% in $\dot{V}O_{2PEAK\text{-}PERF}$. In contrast, the CON condition showed no significant performance improvements, corroborating previous research on plateaus in adaptations to HIIT alone (Paton *et al.* 2005, Norrbom *et al.* 2022). Consistent with our hypothesis, these preliminary findings demonstrate that incorporating BFR during HIIT recovery phases can potentially induce adaptations that enhance determinants of high-intensity intermittent cycling performance.

The increased power output at LT1 with BFR indicates an improved ability to sustain submaximal efforts, which may facilitate a greater contribution from aerobic energy pathways during the intermittent cycling (Brooks 2018). Additionally, an enhanced LT1 suggests a greater capacity to buffer and clear muscular metabolites, such as hydrogen ions, which can aid in recovery between high-intensity intervals by normalising pH (Sahlin 2014). Although both BFR (~5.9%) and CON (~2.5%) showed improvements in FBLA4 from pre- to post-intervention, BFR did not significantly augment these improvements beyond CON. Previous studies (Taylor *et al.* 2016, Mitchell *et al.* 2019) have reported comparable magnitudes of change in critical power and time trial performance following BFR during repeated sprint training recovery phases to the FBLA4 effects observed here. Despite the large but non-significant effect size of the condition-by-time interaction for FBLA4, a larger sample size may have revealed significant differences between conditions. Unlike LT1, which decreased on average from pre- to post-intervention in CON, mean FBLA4 increased in the CON condition. This contrast likely arose as FBLA4 represents an absolute lactate value, while LT1 indicates relative lactate levels influenced by baseline values.

The augmented work accumulated, $\dot{V}O_{2PEAK\text{-}PERF}$, and hastened muscle TSI recovery with BFR suggest enhanced intermittent high-intensity recovery capabilities (Chorley *et al.* 2020, Kirby *et al.* 2021). Given the reliance of team-pursuit cyclists on rapid recovery between intense efforts (Pugh *et al.* 2022), these provisional enhancements may improve performance through facilitated recovery between leading phases. Specifically, the improved $\dot{V}O_{2PEAK\text{-}PERF}$ but not $\dot{V}O_{2PEAK\text{-}STEP}$ with BFR indicates modality-specific rather than generalised aerobic adaptations. These findings align with previous research by Giovanna *et al.* (2022) showing $\dot{V}O_{2PEAK}$ improvements during a TTE trial at 105% of maximal aerobic power but not during an incremental test following chronic BFR between repeated 10-s sprint training. In contrast, Taylor *et al*. (2016) and Mitchell *et al*. (2019) demonstrated improved $\dot{V}O_{2PEAK}$ through incremental exercise after integrating BFR between repeated 30-s sprints. Thus, adaptation specificity likely depends on training characteristics. The increased $\dot{V}O_{2PEAK\text{-}PERF}$ but not $\dot{V}O_{2PEAK\text{-}STEP}$ implies peripheral rather than central physiological improvements, potentially through enhanced skeletal muscle O_2 extraction capabilities (Skattebo *et al.* 2020, Hellsten *et al.* 2023). However, variability in the rate of TSI recovery decreases confidence in the measured changes, despite TSI recovery showing significant augmentation from pre- to post-intervention with BFR. Additionally, though non-significantly different at baseline, the 20% lower pre-intervention intermittent TTE for BFR compared to CON could partially explain the improved $\dot{V}O_{2PEAK\text{-}PERF}$. Nonetheless, similar baseline absolute $\dot{V}O_{2PEAK\text{-}PERF}$ between groups and consistently greater $\dot{V}O_{2PEAK\text{-}PERF}$ than $\dot{V}O_{2PEAK\text{-}STEP}$ suggests pre-intervention function did not limit observed effects.

Our preliminary findings demonstrated an enhanced 30-s test MPO in the BFR condition, indicating an improved glycolytic capacity. This adaptation likely enabled greater tolerance for the initial high-intensity bout of the intermittent cycling test when glycolysis is the primary source of ATP (Buchheit and Laursen 2013b). However, the benefit of an enhanced glycolytic capacity diminishes during successive high-intensity intervals within a HIIT session due to accumulating metabolites (Bishop *et al.* 2011) and exercise-induced acidosis (Fitts 2016), which can impair glycolytic performance. Notably, the improved 30-s MPO was accompanied by increased power output at the LT1, which may enhance

lactate shuttling and buffering capacity (Poole *et al.* 2021), thereby attenuating acidosis during the recovery phases.

While these findings are encouraging, they should be interpreted with caution due to the small sample size (n=5 per group), which increases the risk of both Type I and Type II errors by reducing statistical power and increasing the chance that true effects may not reach significance or positive responses observed in this cohort may not generalise. We note that aspects of the study design could also be improved in future work. Specifically, the HIIT protocol employed a fixed number of repetitions rather than progressing this parameter throughout the intervention. Although the tapering week was included to reduce fatigue for post-intervention testing and may have facilitated the realisation of performance adaptations in the BFR condition, the taper may have been unnecessary in the CON condition. We also uniformly set the BFR cuff pressure at 200 mmHg rather than standardising the degree of BFR for each participant using arterial occlusion pressure (Mattocks *et al.* 2017). While our decision not to use a graded exercise test to assess $\dot{V}O_{2PEAK}$ decreased participant workload, an incremental maximal exercise test may have allowed more definitive comparisons of aerobic fitness outcomes to existing literature. Other limitations include the absence of a normal training control or BFR sham condition and limited control over personal training intensities. Additional research is still required to validate performance effects within team-pursuit cyclists.

Our results suggest that incorporating BFR into HIIT sessions could be a beneficial strategy for trained cyclists aiming to improve measures of submaximal, high-intensity, and intermittent performance without increasing the mechanical workload. Cyclists in the BFR condition showed preliminary evidence of augmented work done in the intermittent test by ~41.5 kJ (~55%), submaximal lactate threshold by ~20 W (~7%), and 30-s MPO by ~50 W (~7%). Since events like the track cycling team-pursuit require exceptional aerobic capacity and rapid recovery between intense efforts (Pugh *et al.* 2022), integrating BFR training may particularly benefit such intermittent high-intensity sports. While the post-taper performance gains highlight usefulness for peaking before a competition, the present BFR intervention could also be applied during general preparation phases of periodised training. Beyond

cycling, BFR implementation may benefit other intermittent sports, such as ice-skating team-pursuit, soccer, or rugby, through similar mechanisms.

Conclusions

In conclusion, this study provides preliminary evidence that incorporating BFR during HIIT recovery phases over a four-week intervention can improve several physiological determinants and performance outcomes related to intense intermittent cycling. The improvements appear to be mediated through augmented peripheral adaptations that improved aerobic capacity and metabolic recovery between high-intensity work bouts. Overall, strategically implementing BFR into HIIT protocols holds potential to further enhance high-intensity intermittent cycling performance beyond HIIT alone. However, these findings should be interpreted cautiously given the small sample size, and further research with larger samples is warranted to confirm the performance effects, particularly in team-pursuit cyclists.

Chapter Six – Integrating blood flow restriction into training for elite team-pursuit cyclists – a case-study

Prelude

Having established in previous chapters that BFR applied between HIIT work bouts can elicit significant physiological responses and improve intermittent performance in trained cyclists, this chapter aims to examine the feasibility and tolerability of this intervention for world-class athletes preparing for the Olympics. This chapter provides essential insight into the real-world application of this emerging training method for elite TP cyclists.

Introduction

Elite team-pursuit (TP) cyclists typically periodise their training, emphasising high volumes of aerobic endurance work during the general preparation phase before shifting to focused high-intensity and event-specific skills training in the pre-competition phase (Schumacher *et al.* 2002). Aerobic conditioning builds the foundation for endurance performance (Joyner 1991, Broker *et al.* 1999) by enhancing the ability to sustain power output during prolonged endurance exercise or repeated high-intensity efforts (Maunder *et al.* 2021). High-intensity training is considered necessary to develop high-intensity performance capabilities (Bangsbo 2015) and can be structured as high-intensity interval training (HIIT), whereby bouts of high-intensity exercise are interspersed with periods of rest or recovery. Additionally, despite lower training volumes, HIIT can be more effective than low-intensity training at enhancing $\dot{V}O_{2PEAK}$, muscle oxidative capacity, and muscle buffering capacity (Laursen *et al.* 2002a, Gibala *et al.* 2009). However, the adaptive response to HIIT may plateau after 8-12 sessions (Paton *et al.* 2005) or 3-4 weeks (Norrbom *et al.* 2022) in highly-trained athletes without progression of intensity or duration.

Incorporating novel stimuli, like blood flow restriction (BFR), could augment adaptations to normal training sessions by applying a passive stimulus without increasing external training load. By inflating pneumatic cuffs proximally on the limbs, BFR reduces venous return and can be applied before (Patterson *et al.* 2015), during (Conceição *et al.* 2019, Christiansen *et al.* 2020), or after exercise (Taylor *et al.* 2016, Okita *et al.* 2019). While applying BFR during high-intensity intervals decreases exercise tolerance (Willis *et al.* 2018); applying BFR during recovery between work bouts has been shown to maintain competition-specific intensity while potentially constraining metabolite clearance and reoxygenation and contribute to enhanced physiological strain (Okita *et al.* 2019). For example, applying BFR during recovery between sprints in endurance athletes improved $\dot{V}O_{2PEAK}$ compared to control groups after 2- to 4-weeks (Taylor *et al.* 2016, Mitchell *et al.* 2019, Giovanna *et al.* 2022). As this approach can increase aerobic capacity in trained cyclists, integrating BFR into team-pursuit HIIT could further extend benefits relevant to performance. Elite TP cyclists often perform HIIT sessions to practice the high-intensity requirements of leading the TP, and integrating short BFR occlusion into

their HIIT recovery phases could similarly produce beneficial adaptations if tolerated amongst their overall training. However, negative outcomes like ischemia-reperfusion injury, oxidative stress, rhabdomyolysis, and discomfort have been reported, warranting careful implementation in athletes.

To evaluate the feasibility of incorporating BFR into HIIT recovery phases, we introduced a BFR protocol to an international TP squad during a training camp. We examined muscle oxygenation responses to validate previous findings on BFR in elite cyclists. Based on evidence that trained populations tolerated similar protocols, we hypothesised that the cyclists would successfully integrate BFR into their HIIT sessions.

Methods

Participants

Five elite male TP cyclists (age: 25 ± 4 years, height: 179.3 ± 4.5 cm, body mass: 75.5 ± 5.7 kg, mean ± SD) volunteered for this observational study. All cyclists were training for an upcoming Olympic Games taking place 12 weeks after the present study. All participants were screened before recruitment according to BFR safety guidelines (Kacin *et al.* 2015). Participants provided written informed consent, and the study was approved by the participating institution's human research ethics committee [ethics approval number: HREC(HECS)2021#04] and performed in accordance with the Declaration of Helsinki.

Study Design

BFR was introduced into two HIIT sessions of a TP training camp for five TP cyclists. Cycling training load throughout the camp was evaluated from an individual participant (Cyclist 1). Muscle oxygenation and power output data were collected for two of the five cyclists (referred to as Cyclist 2 and 3). Data collection was limited to these participants due to equipment availability. All cyclists were provided

with questionnaires to report pain, enjoyment, and tolerability of the BFR intervention. Four cyclists returned completed questionnaires (see Appendix 1 for questionnaire format).

Questionnaire

A questionnaire was developed to assess cyclists' perceptions of the cycling intervention, with a focus on evaluating pain, enjoyment, tolerability, and comparisons to other training modalities. The questionnaire contained two closed ended questions utilising the Borg CR10 scale (Borg 1998) to rate pain levels and progression, and six open ended questions eliciting free responses on general thoughts, enjoyment, comparisons to other training, and tolerability feelings after the session and the next day. The closed ended 0-10 Borg CR10 scale has demonstrated reliability and validity in measuring perceived pain in cycling (Monnier-Benoit *et al.* 2006).

Cyclists were familiarised to the questionnaire and Borg CR10 scale in-person prior to the intervention. After completing the final BFR session, cyclists were provided with an electronic questionnaire link to complete independently within 24-hours.

Training schedule

The cyclists participated in a five-day training camp with daily morning road cycling sessions. On days 2 and 5 of the camp, the road cycling continued into the afternoon. HIIT was performed in the afternoons on days 1 and 4, during which the BFR intervention was integrated (Table 9). All cycling activities utilised the cyclists' personal bicycles, configured for either standard road or TP positions. Time trial bicycles, replicating the positions used in the TP, were used for two sessions. Strength training was performed on day 3 to maintain muscle strength but was not quantified. Training data files of Cyclist 1 were analysed, and power output values were categorised into bins of 50 watts, except for 0 W values which had their own bin.

Table 9. Training schedule of an elite team-pursuit squad with blood flow restriction intervention.

	Day 1	Day 2	Day 3	Day 4	Day 5
Morning	Road ride. 115-km, 2500-m vertical gain.	Road ride. 200-km, 1500-m vertical gain. Structured intensity included.	Road ride on TT bikes. Low intensity	Road rode on TT bikes. 115-km, 1000-m vertical gain. Structured intensity included.	Road ride. 200-km, 1500-m vertical gain. Structured intensity included.
Afternoon	HIIT (6x 30-s at TP lead power) with BFR for 2-minutes between reps.		Resistance training: strength maintenance	HIIT (6x 30-s at TP lead power) with BFR for 2-minutes between reps.	

On day 1,3, and 4 of the training camp, participants trained twice per day. On day 2 and 5, participants trained once per day. Abbreviations and definitions: HIIT, high-intensity interval training; TP, team-pursuit; TT, time trial.

BFR exercise protocol

Cyclists performed a HIIT protocol on their personal bicycles mounted to air-braked trainers (LeMond Revolution, USA). The protocol consisted of 6 x 30-s intervals at an intensity equivalent to their TP lead power, interspersed by 4.5-minute recovery periods. During recovery, cyclists wore 11 cm wide, pneumatic cuffs (Occlude, Aarhus, Denmark) bilaterally on the upper thighs inflated as soon as possible after exercise to 200 mmHg. Pressure was maintained for a 2-minute duration before deflation and cuff removal to provide participants with 1.5-minutes of unoccluded supine recovery. With 30-s remaining before their next interval, participants remounted their bicycles to perform the subsequent interval.

Power output

Workload was quantified using power meters (Shimano SRM 9000, Schoberer Rad Meßtechnik, Jülich, Germany) installed on two bicycles. The power meter strain gauges were statically calibrated prior to testing according to Wooles and colleagues (2005). Some variability in cadence and resistance occurred with the bicycle trainers, leading to fluctuations in power output around the prescribed target values. To maintain the desired intensity, verbal feedback was provided to instruct cyclists to sustain the

specified power output. Mean high-intensity power output was defined as the average power generated specifically during each high-intensity work bout.

Muscle oxygenation

Muscle O_2 saturation was determined with near-infrared spectroscopy (NIRS) sensors (Moxy 3, Fortiori Design LLC, Minnesota, USA). Two cyclists (Cyclists 2 and 3) completed their HIIT sessions with NIRS sensors secured with a compression bandage to the left and right vastus-lateralis muscle bellies at 40% of the distance between the greater trochanter and the lateral epicondyle of the femur. Mean tissue O_2 saturation index (TSI; %) data was computed for the entire session, as well as during each occluded period. A 90-s resting period while seated on bicycles before the first HIIT work bout provided baseline values of TSI for each of the two observed cyclists.

The half-time of TSI recovery (in s) was calculated for the initial 30-s following each release of BFR cuff pressure and evaluated with the following monoexponential regression:

$$TSI(t) = Ae^{-kt} + C \qquad \text{Equation 16}$$

Where t is time (in s), A is the initial TSI difference from baseline, k is the rate constant (curve slope), and C is the asymptotic baseline. The time constant (τ) was $1/k$. Half-time was defined as $\ln(2)*\tau$. The correlation coefficients (r) of the time-TSI relationships for each recovery period varied from 0.96 to 0.80 (0.90 ± 0.05). The mean and standard deviation of the six recovery phases were calculated for each of the two cyclists that were assessed for TSI.

Data handling and statistical analysis

Power output and NIRS data were continuously collected at 1 Hz using a cycling computer (Edge 1030, Garmin Ltd., USA). The raw data files were downloaded to Golden Cheetah software (version 3.5, open-source) for initial processing before being imported into Excel (Microsoft Corporation, USA) for analysis. Figures were constructed using GraphPad Prism (version 9.5.1, GraphPad Software, San

Diego, CA, USA) to illustrate raw power output, TSI traces during high-intensity interval training sessions, as well as the power output distribution throughout the training camp.

Results

A total of distance of 585-km was completed during the 24-hours total training duration. During this training, Cyclist 1 gained 5654-m of vertical elevation and demonstrated a mean power output of 172 W, resulting in a total work completed of 14,399 kJ. The distribution of power output across the five days of the training camp is demonstrated in Figure 22, not including resistance training.

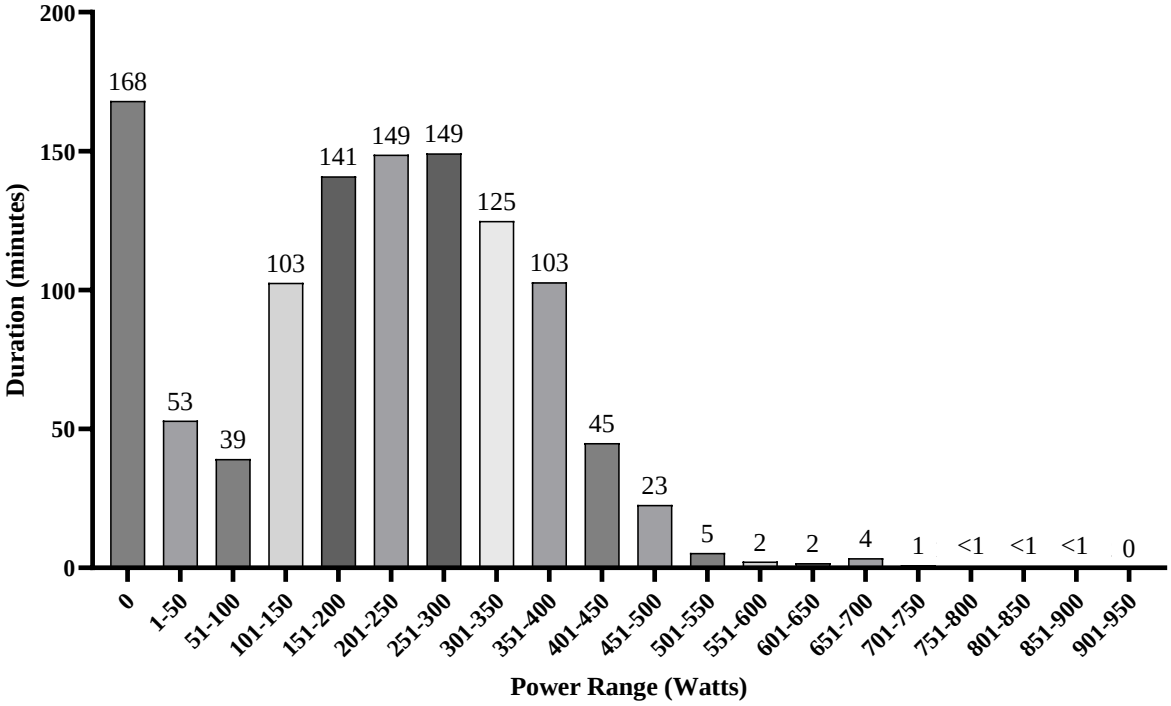


Figure 22. Distribution of power output during an elite team-pursuit training camp.

During the initial BFR application, Cyclist 5 experienced significant discomfort, which we classified as an adverse response. To ensure their safety, we immediately discontinued the BFR protocol for this individual and promptly removed the occlusion. It is important to note that this participant had no known pre-existing cardiovascular conditions prior to their involvement in our study. However, the participant had reported a long-standing issue of what they described as severe lactate accumulation in their left

leg, particularly during high-intensity exercise. This condition and follow up assessment are further detailed in the addendum.

Responses to questionnaires indicated that the BFR intervention was painful (pain ratings up to 10 out of 11); however, it was well tolerated and perceived positively by participants (Table 10). The cyclists found the BFR sensations novel and unlike other training modalities. Overall, they enjoyed the variety and challenge of BFR within HIIT recovery and felt it provided a beneficial training stimulus despite fatigue immediately after sessions.

Raw power output and TSI data from HIIT sessions for two cyclists are presented in Figure 23. Data from HIIT sessions for Cyclists 2 and 3 revealed that the power output during high-intensity work bouts was 628 ± 14 W and 640 ± 23 W, respectively, which closely aligned with the target intensities of 635 W and 650 W set for each cyclist. Mean power output for Cyclists 2 and 3 for each repetition, respectively, was: repetition 1, 621 W and 603 W; repetition 2, 622 W and 634 W; repetition 3, 611 W and 638 W; repetition 4, 628 W and 651 W; repetition 5, 640 W and 644 W; repetition 6, 650 W and 674 W.

TSI values across the entire HIIT session were 29.4 ± 31.3% and 23.5 ± 27.7% for Cyclists 2 and 3, respectively. During BFR application, TSI was 4.0 ± 2.6% and 5.5 ± 3.5%, while during the reperfusion phase of recovery, TSI was 66.5 ± 8.3% and 49.8 ± 8.1%. During the entire recovery, TSI was 31.3 ± 5.6% and 23.4 ± 4.5%, respectively. The half-time of TSI recovery was 9 ± 3-s and 17 ± 12-s for Cyclists 2 and 3, respectively.

Table 10. Responses from Team Pursuit cyclists for an intervention where blood flow restriction is applied to high-intensity interval training recovery periods.

Cyclist	General Thoughts	Pain Levels (Borg CR10)	Progression of Pain Through Reps	Tolerance Over Time	Comparison to Other Training Modalities	Enjoyment	Feelings After Session	Feelings the Next Day
1	Good, different variety, big medicine in a short session	Very painful (8) Especially the first couple of sessions. With increased intensity in the 30 second efforts came a lot more pain in the cuffed period.	Rep 4 it would really come on. This could be influenced by the effort put into the 30 s.	More tolerable. Was an unusual feeling for the first session but when you learn what to expect it seems less uncomfortable.	Comparable to no other. The last lap of a TP!	I enjoyed the variety and different challenge. Get in, get it done and get out.	I would always feel really good immediately after that session	Not too bad, hard to say due to hard training block
2	I quite like it, but this could be a quite biased opinion as I've used it historically. I like that it's different to other modalities of training and like the theory behind it.	Some days excruciating 10. Some days maybe like an 8. Pain ramps through the cuffed 2-mins.	Sometimes you are hurting really bad after the first rep and sometimes it starts to hurt a bit more each session.	Still as painful each time. I think it's dictated by how hard you go on the bike.	A very strong throbbing ache.	I like the session.	Some days really sore afterwards, some days no pain at all afterwards.	Generally fine
3	It was a good session which hurt at the time but wasn't super taxing.	6.5. It was increasingly painful while the cuffs are on, but the pain left quickly after it’s removed.	It started pretty tolerable and ended up quite painful on the last.	I seemed to get a similar feeling across all the sessions.	Nothing I've ever done	I liked what I got from the session	Body was slightly fatigued but was more of just pain in the legs	Fine
4	It was a good top-up to a long day on the bike with no intensity I thought it kept the body in check with the high-power load, and it came at very little cost to affecting the following days training.	8. Painful but tolerable. The pain got worse through the first minute and then was steady until removed.	Pain decreased through the session from an 8 at the start to a 6 at the end.	I feel like the body did start to tolerate it more by the final session where you could just push harder on the bike.	I wouldn’t compare it to anything I think it has its own unique sensations.	I enjoyed it.	I felt fatigued immediately post-session.	Felt fine the next day and was able to train to my max.

Abbreviations and definitions: BFR, blood flow restriction; TP, team-pursuit.

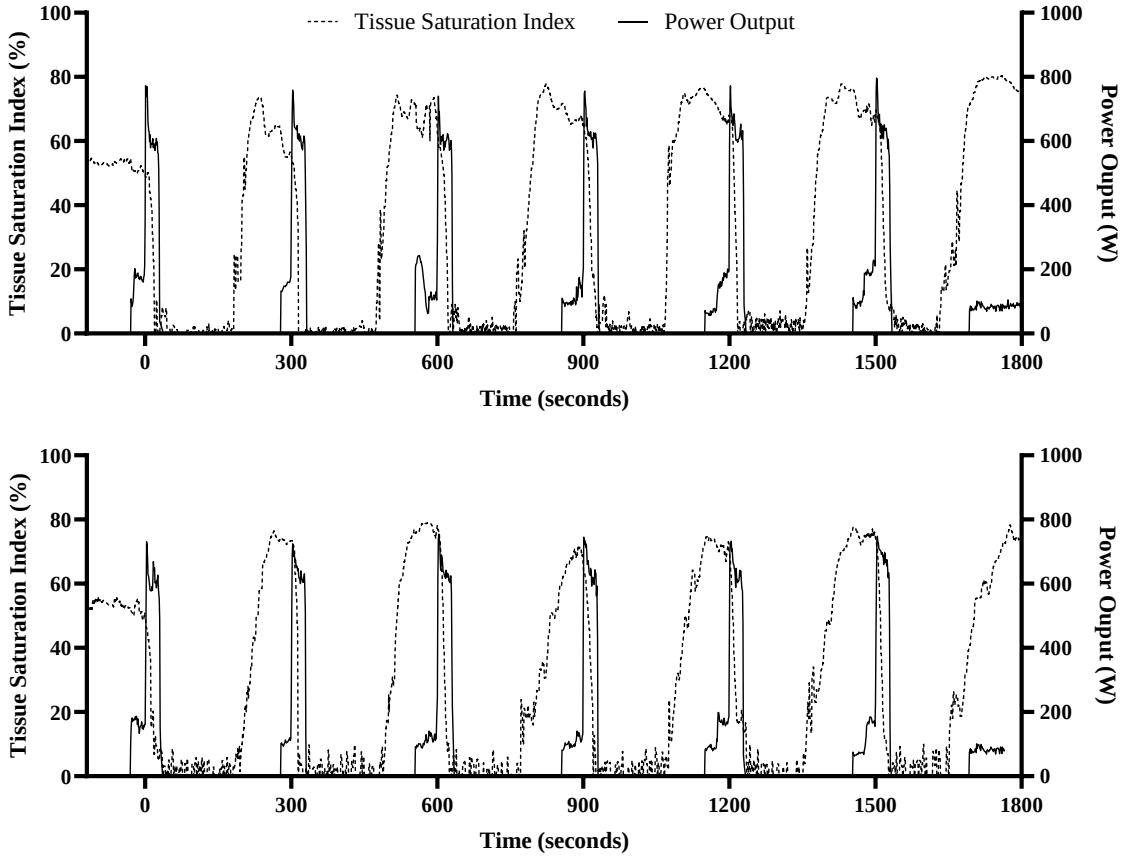


Figure 23. Raw power output and muscle oxygenation data during a high-intensity interval training session with blood flow restriction applied during recovery phases.

Top and bottom panes present Cyclist 2 and 3, respectively. Tissue oxygen saturation index values at time <-30-s are included as a baseline indication.

Discussion

This case-study provides initial evidence that BFR can be incorporated into HIIT for elite male TP cyclists. A post-intervention questionnaire revealed that the cyclists enjoyed and tolerated adding BFR to HIIT, despite the pain involved. The reduced muscle TSI during occlusion confirms previous acute studies investigating similar BFR interventions in controlled laboratory environments. Despite the occlusion during recovery phases, power output did not decrease, as both cyclists whose data were measured achieved their greatest mean power output in their final work bout. Furthermore, the final three work bouts were consistently performed at a greater intensity than the initial three. Over the 5-day training camp, cyclists performed a total of 24-hours of training at an average intensity of 172 W,

including 4-minutes at 650-700 W. This training programme aligns with established TP training principles (Schumacher *et al.* 2002) of high-volume, low-intensity training combined with race-specific, high-intensity efforts, despite the relatively short ~4-minute event duration. Overall, the results and direct insights from elite cyclists support the feasibility of combining BFR and HIIT in an elite training setting.

Participants reported moderate to severe pain during BFR application, ranging from 6.5 to 10 on a 0-11 scale, using descriptor such as "uncomfortable", "throbbing", and "excruciating" to characterise the sensation. This intense pain may be attributed to metabolic acidosis resulting from the accumulation of hydrogen ions (Pope *et al.* 2013, Okita *et al.* 2019). Edge and colleagues (2006) suggested that intramuscular acidosis induced by high-intensity exercise can stimulate adaptations to enhance muscle buffering capacity for hydrogen ions. Such adaptations may be beneficial for TP cyclists, given the high-intensity and rapid recovery demands of the TP event.

The peak pain reported towards the end of sessions indicates a cumulative fatigue effect. Some participants felt acutely fatigued and sore immediately after each session but reported minimal residual effects the following day. Some participants indicated that their pain levels were related to the intensity of the preceding exercise effort, implying a connection between exercise intensity and subsequent BFR-induced pain. As cyclists completed 115-km rides earlier in the day to the HIIT sessions, some residual fatigue likely remained from the morning training. However, this did not negatively impact the completion of the subsequent HIIT and BFR sessions during the training camp, demonstrating the cyclists' high level of fitness. Future research should examine the impact of this intervention on subsequent performance and muscle fatigue, such as using interpolated twitch techniques to assess peripheral fatigue.

The NIRS data demonstrate consistent TSI decreases during BFR for the two observed cyclists. Compared to the trained cyclists in Chapter Three, the elite TP cyclists in the present study had lower TSI values during entire recovery phase (~ 27.4% vs ~35.2%), potentially as a result of the greater intensity work bouts sustained by the elite TP cyclists. The reduced muscular TSI provides evidence for the potential effectiveness of BFR application in elite athletes. Previous research, including work

by Christiansen *et al*. (2020), has highlighted the role of intramuscular hypoxia as a stimulus for vascular adaptations and enhanced O_2 extraction. Such adaptations may prove advantageous for TP cyclists.

Practical applications

This study provides initial evidence that integrating BFR into HIIT is feasible for diversifying the training stimulus for elite male TP cyclists. The pain ratings and reduced muscle oxygenation data help set expectations for BFR use. Although definitive conclusions about performance impact require controlled experimental designs, the cyclists' post-intervention Olympic performance provides preliminary indications of a positive impact.

Limitations

This study has several limitations that warrant consideration. First, the LeMond Revolution ergometer used for the HIIT sessions did not allow for electronic control and precise regulation of power output. As a result, cyclists had to manually gauge their effort levels, which may have introduced some variability in the actual intensity achieved across sessions and participants. Second, the questionnaire assessing subjective perceptions was only completed once by each participant, within 24 hours after the final BFR session. This retrospective self-report may have been subject to poor recall or response biases. Having participants complete the questionnaire immediately after each session could improve accuracy. Additionally, the single questionnaire administration provided insufficient data for statistical analysis of these perceptual measures. Third, given the variation in perceived pain amongst cyclists and the standardised BFR cuff pressure of 200 mmHg, the degree of occlusion likely differed amongst cyclists. Although the consistently reduced TSI indicates that some level of occlusion was achieved, personalised BFR pressures based on arterial occlusion pressure may help achieve more consistent occlusion in future trials.

Conclusions

This study demonstrates that elite TP cyclists can incorporate BFR into HIIT sessions during a training camp. Cyclists enjoyed the BFR sessions, indicating the variety, novelty, and efficiency of the intervention potentially offset the pain involved. The reduced TSI and mounting pain induced by BFR imply localised hypoxia and metabolite accumulation, which could promote beneficial adaptations beyond traditional HIIT. Importantly, the BFR did not negatively impact achieving target work bout intensity. Optimal BFR frequency and timing within a training cycle also require investigation to maximise benefits and minimise risk of non-functional overreaching.

Chapter Seven – General discussion

Thesis aims

This thesis aimed to critically evaluate the efficacy of BFR training and modelling of W'_{BAL} to enhance various performance factors associated with TP performance. The overall goal was to contribute to the advancement of knowledge regarding evidence-based training methods and periodisation strategies specifically tailored for the TP event. To achieve this goal, a series of interconnected studies were undertaken. The initial study explored the physiological determinants of TP performance, with a particular focus on the parameters of the W'_{BAL} model, such as CP, W', and W' reconstitution. Building upon these insights into the demands of TP, a novel BFR intervention was conceptualised and designed to specifically target the identified key performance factors. The second study focused on assessing the acute physiological effects of this BFR protocol when applied to highly-trained cyclists. The aim was to gauge its potential as a valuable training stimulus. In the third study, the BFR intervention was implemented longitudinally over a training cycle with trained cyclists to investigate how it influenced relevant parameters that impact TP performance. The final case-study extended the application of the BFR training intervention to an Olympic TP squad during their routine training activities. This was conducted to explore the feasibility and practicality of integrating this specialised training method into the routines of elite athletes.

W'_{BAL} modelling insights for team-pursuit cyclists

In Chapter Three, a study was conducted involving three different performance levels of TP cyclists. The primary objective of this study was to determine the key W'_{BAL} parameters distinguishing performance across these different levels. Given the established utility of the W'_{BAL} model in estimating work capacity availability during cycling (Skiba *et al.* 2015, Skiba and Clarke 2021b), and TP events

(Bartram *et al.* 2018, Bartram *et al.* 2021), the model's parameters were well-suited for assessing the TP event's distinct demands.

The findings highlighted CP and W′ reconstitution rate as critical differentiators of TP performance. These findings reaffirm the importance of aerobic fitness and energy availability in intermittent exercise (Tomlin *et al.* 2001) and the TP specifically (Broker *et al.* 1999). Higher CP enabled cyclists to exercise within the heavy domain at higher power outputs, preserving finite W′ reserves for the intense leading phases. Moreover, faster rates of W′ reconstitution during recovery periods below CP allowed superior intermittent exercise capability by enabling greater recovery between leading phases.

Remarkably, International TP cyclists expended over 100% of their initial W′. This capacity is only feasible due to their enhanced ability to reconstitute depleted W′ stores over the TP's duration, highlighting W′ reconstitution kinetics as a key factor distinguishing elite from sub-elite abilities. The significance of accelerating this parameter was further reinforced by identifying the "hyperbolic" region slightly below CP as a sensitive intensity region greatly impacting reconstitution dynamics. While the difference in W′ expended at the TP finish between International and National teams was not statistically significant (104 ± 5% vs 75 ± 18%; $p = 0.117$), the lower W′ expenditure by the National team suggests their pacing strategy may have been sub-optimal, resulting in an underestimation of their true performance capacity.

Figure 24 serves as an updated iteration of Figure 5, originally presented in Chapter Two and illustrates how the findings regarding W′ reconstitution in this study can be aligned with and applied to the existing body of literature. For TP, the hyperbolic region of the D_{CP}-*Tau* relationship is particularly important given the substantial disparity between models in this specific region. The International team exhibited faster W′ reconstitution rates than lower levels when recovering within 50 W of CP, but not at intensities 100 W below CP. Since TP recovery phases frequently occur near CP, this hyperbolic W′ reconstitution rate significantly impacted recovery effectiveness. Even slight changes in intensity within the hyperbolic region considerably impact *Tau*; therefore, enhancing *Tau* in this hyperbolic region emerges as a key target for improving TP performance capabilities. In line with the observations by Lievens *et al.* (2021), the hyperbolic region denotes recovery in the heavy exercise domain. Reducing the absolute

difference in power output between CP and the LT1/GET would shift the hyperbolic region of the D_{CP}-*Tau* curve to the left, leading to an improvement in the rate of recovery.

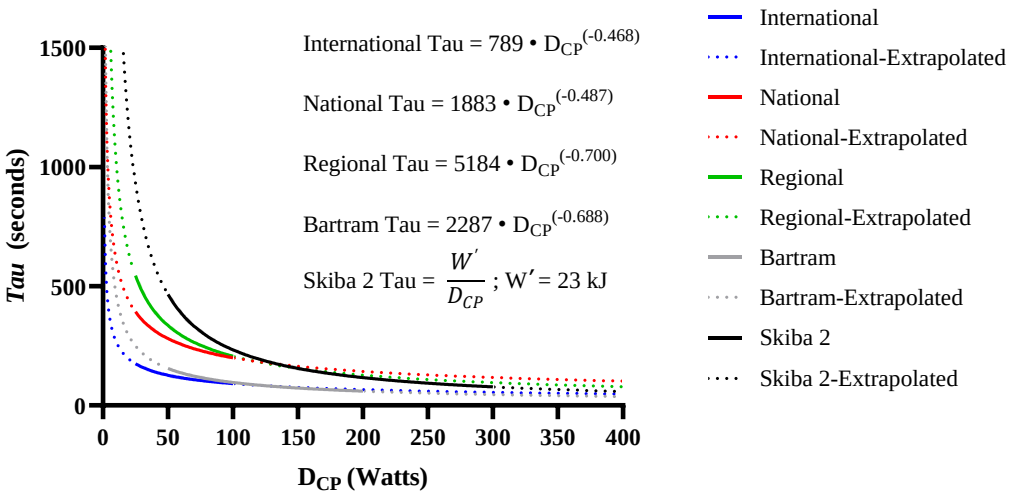


Figure 24. Comparison of existing and new W′ reconstitution adjustments in the D_{CP}-Tau form.

Note that the W′ value of 23 kJ for the Skiba 2 Tau is the mean W′ size reported by Bartram et al. (2018) in elite TP cyclists. Abbreviations and definitions: W′, finite work capacity; Tau, time constant of the rate of W′ reconstitution; D_{CP}, difference between recovery intensity and critical power (CP); W, Watts; s, seconds

Surprisingly, there were no significant differences in W′ magnitude or 1-minute power outputs across TP performance levels. A plausible explanation is that once TP cyclists attain a sufficient W′ to meet the event's intense power demands, further increasing W′ yields minimal additional benefits. As such, excessively inflating W′ at the expense of reduced CP could prove counterproductive (Poole *et al.* 2016). Similarly, overdeveloped CP and W′ reconstitution rates may hinder initial acceleration or leading phases. The findings therefore suggest that for 'optimal' TP performance, training should prioritise elevating CP and enhancing W′ reconstitution kinetics, while maintaining an adequate W′ level for the intense start and lead efforts.

Though the mean International W′ magnitude (28.5 ± 2.6 kJ) exceeded that of elite TP cyclists in prior studies (~23.5 kJ) (Bartram *et al.* 2018, Bartram *et al.* 2021), this is likely as a result of different modelling approaches rather than true physiological discrepancies. Bartram and colleagues utilised the linear work-time model which tends to produce higher CP but lower W′ values compared to the

nonlinear power-time approach used presently (Maturana *et al.* 2018). Considering the modest 1-3 kJ standard errors here versus ~1-10 kJ errors reported previously, the differences in estimated W′ magnitudes across studies are likely negligible.

While a weak negative correlation was found between CP and W′ (R^2 = 0.191, p = 0.156) (Figure 25), the relationship between these parameters remains inconsistent across studies. Some report strong positive associations (Chorley *et al.* 2020), while others show no significant link (Bourgois *et al.* 2023). The negative trend observed herein may reflect elite TP cyclists trending towards higher CP but slightly lower W′ capacities (Poole *et al.* 2016). However, the low R^2 value indicates CP and W′ largely represent distinct physiological capacities.

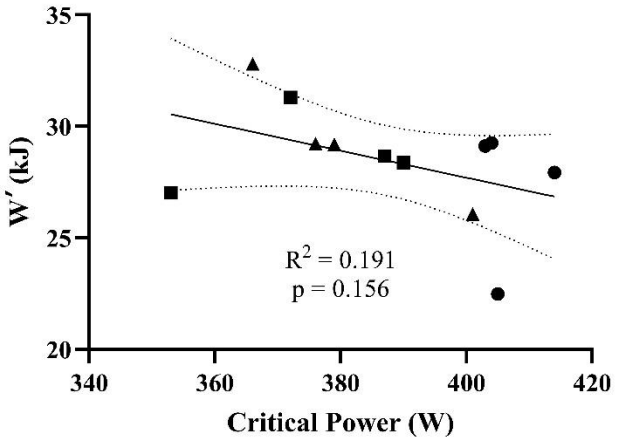


Figure 25. Relationship between critical power and W′ in team-pursuit cyclists.

Dotted lines show 95% confidence bands. International—Circles; National—Triangles; Regional-Squares. Abbreviations and definitions: W′: work capacity above critical power.

In contrast, significant positive correlations emerged between CP and W′ reconstitution rates at 25 W (R^2 = 0.725) and 50 W (R^2 = 0.559) below CP. These observations align with CP representing the aerobic capacity governing W′ reconstitution. Interestingly, no significant correlations manifested between W′ magnitude itself and reconstitution rates at any intensity level (Figure 26). This dissociation between W′ size and its reconstitution kinetics contrasts findings by Chorley et al. (2020), where larger W′ associated with greater absolute W′ recovery, though the effect disappeared when expressed as percentages in trained cyclists. The relationship between W′ magnitude and reconstitution rates

therefore appears complex. While models like Skiba's differential formula (Equation 6 and 7) inherently link W′ expenditure to reconstitution rate, the precise interdependencies require further clarification. Methodological differences, such as the lack of a prior exhaustive bout and use of higher recovery intensities in the present study compared to Chorley et al. (2020), may impact measured reconstitution kinetics (Skiba *et al.* 2014b). Clearly, additional research is warranted to definitively map the relationship between W′ magnitude and its rate of reconstitution across different intensity domains.

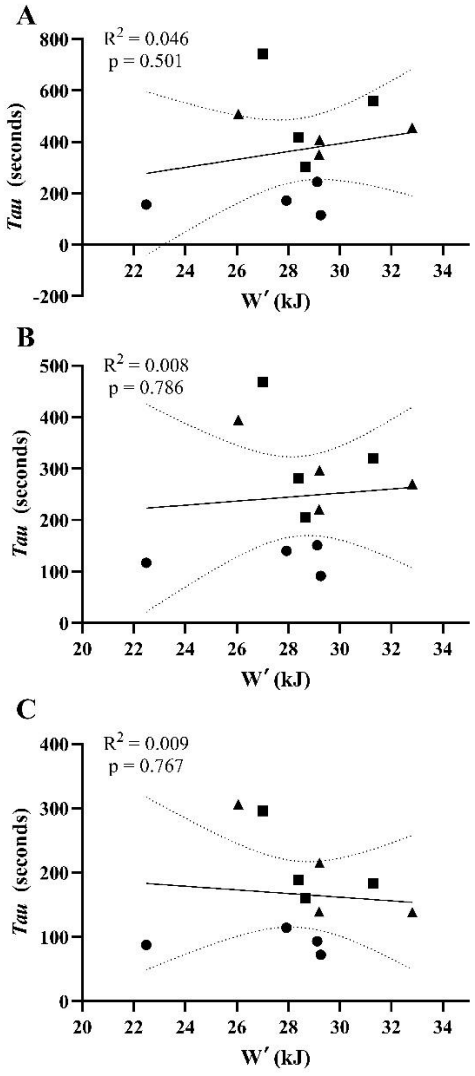


Figure 26. Relationship between W' and Tau of reconstitution.

Panel A shows Tau at D_{CP} 25, Panel B shows Tau at D_{CP} 50, and Panel C shows Tau at D_{CP} 100. Dotted lines show 95% confidence bands. International—Circles; National—Triangles; Regional-Squares. Abbreviations and definitions: D_{CP}: the absolute difference in power output between the critical power and recovery intensity below critical power; Tau: the rate of W' reconstitution; W': work capacity above critical power.

These W'_{BAL} modelling insights collectively informed the design and rationale for incorporating a novel BFR intervention into the training of elite TP cyclists, as demonstrated in Chapter Six. Elite TP cyclists undertake substantial training volumes when preparing for the major events such as the Olympics. To minimise disruption, the BFR protocol was integrated into existing HIIT sessions that replicated TP demands before competition (Schumacher *et al.* 2002). Applying BFR between work bouts at TP lead intensities aimed to accentuate the training stimulus stress and accelerate specific adaptations like CP, W′ reconstitution rate, and intermittent capabilities without increasing the mechanical workload. Previous research also supports implementing BFR during recovery intervals between high-intensity efforts to promote favourable physiological and performance adaptations (Taylor *et al.* 2016, Mitchell 2019, Mitchell *et al.* 2019, Okita *et al.* 2019, Giovanna *et al.* 2022).

Acute physiological responses and mechanistic insights of blood flow restriction between high-intensity intervals

The acute study in Chapter Four investigated the physiological impact of incorporating BFR into the recovery phases of HIIT sessions in trained cyclists. Consistent with previous studies, applying BFR between HIIT work intervals hindered typical recovery processes like muscle reoxygenation (TSI recovery), $\dot{V}O_2$, and $\dot{V}CO_2$ compared to control HIIT sessions (Harris *et al.* 1976a, McManus *et al.* 2018, Okita *et al.* 2019, Solsona *et al.* 2021, Ienaga *et al.* 2022). These impaired physiological responses play a central role in reestablishing homeostasis after severe-intensity exercise. The restricted blood flow led to metabolite accumulation and local muscle deoxygenation that prolonged the metabolic and physiological stress.

Notably, the elite TP cyclists in the case study (Chapter Six) also exhibited reduced muscle TSI levels when BFR was integrated into their HIIT sessions, corroborating the acute muscle deoxygenation effects observed in Chapter Four. This local hypoxic state created by BFR is proposed to amplify specific adaptations relevant to TP performance, such as improving O_2 delivery and metabolic recovery capacities.

Furthermore, Chapter Four revealed a novel finding with increased circulating serum VEGF concentration following the acute BFR intervention compared to control HIIT. In contrast to previous studies that examined VEGF at the mRNA level and did not detect increases with BFR (Taylor *et al.* 2016, Mitchell 2019), the current study measured circulating VEGF protein and found significant elevations following acute BFR application. As VEGF plays a central role in promoting angiogenesis (Lee *et al.* 2004, Chinsomboon *et al.* 2009, Olfert *et al.* 2010), this demonstrates BFR's potency to stimulate adaptive responses that may enhance metabolic recovery. Although an acute circulating VEGF increase does not necessarily indicate chronic VEGF gene expression, it demonstrates BFR's potential to induce pro-angiogenic signals that may facilitate peripheral vascular remodelling and O_2 transport adaptations over time, that would support improved intermittent exercise performance.

The acute physiological responses in Chapter Four, coupled with the elite TP case study observations, provided key mechanistic insights underlying BFR's ability to amplify adaptations to HIIT. Specifically, by restricting blood flow and O_2 delivery during recovery periods, BFR accentuates local muscle hypoxia and metabolite accumulation, and coincided with increases in circulating VEGF, a putatively angiogenic factor. These effects likely contributed to the enhanced intermittent abilities, muscle reoxygenation, and improved O_2 uptake observed after chronic BFR training in Chapter Five, which may ultimately translate to better W′ reconstitution capabilities for TP performance as identified in Chapter Three.

Chronic physiological effects of blood flow restriction between high-intensity intervals

To investigate whether these acute physiological indicators translated into chronic performance adaptations, Chapter Five implemented an four-week BFR training intervention in trained cyclists. Eight BFR sessions were incorporated into HIIT, with BFR applied specifically during the recovery periods between intense work intervals. Pre- and post-intervention testing batteries evaluated key performance indicators including lactate threshold, 30-s sprint ability, and an intermittent cycling test designed to simulate TP demands.

Remarkably, BFR training led to significant, albeit preliminary, improvements in intermittent performance and related physiological indicators, despite the absence of a progressive overload in mechanical workload. These included improvements in submaximal lactate threshold, mean power output during a 30-s sprint, and $\dot{V}O_{2PEAK}$ during the intermittent test. These enhancements are likely to be advantageous for TP cyclists, aligning with the Chapter Three findings that identified CP, W′ reconstitution rate, and intermittent capabilities as key performance determinants.

Notably, $\dot{V}O_{2PEAK}$ improved during the intermittent test but not incremental exercise, a pattern consistent with Giovanna *et al.* (2022). In contrast, Taylor *et al.* (2016) and Mitchell *et al.* (2019) integrated BFR between 30-s sprints and observed gains in $\dot{V}O_{2PEAK}$ during incremental tests. If BFR had primarily improved $\dot{V}O_{2PEAK}$ by improving central cardiovascular factors like cardiac output, $\dot{V}O_{2PEAK}$ increases would be expected during ramp exercise as well. Instead, the discrepancy suggests BFR may elicit peripheral adaptations that enhance O_2 extraction and utilisation capabilities specifically for intermittent exercise, rather than elevating maximum cardiac output per se. This potential for enhanced peripheral O_2 delivery and extraction with BFR could mechanistically link to the acute increase in circulating VEGF observed following BFR in Chapter Four.

The control group demonstrated limited physiological and performance adaptations, indicating that the cohort of cyclists was, as anticipated for well-trained individuals, experiencing a reduced adaptive response to the training stimulus (Paton *et al.* 2005, Norrbom *et al.* 2022). However, when BFR was introduced in the absence of a progressive increase in mechanical load, it led to substantial improvements in intermittent performance. These findings suggest that BFR may enhance performance in well-trained athletes, although the small sample size means the results should be interpreted with caution, as another group of cyclists may respond differently. Though efforts were made to match the condition cohorts, the performance of the BFR group in the pre-intervention testing suggested that the BFR group entered the study with less intermittent fitness than CON. Despite starting at a lower fitness level, the BFR group finished with a greater intermittent fitness level, demonstrating the potential of the intervention.

In contrast to the substantial 55% improvement in intermittent test performance, BFR did not significantly impact FBLA4, a parameter strongly correlated with CP (Valenzuela *et al.* 2021), compared to control. However, the ~5% improvement in FBLA4 with BFR versus only ~2% for control, while not statistically significant, could still be meaningfully beneficial based on Paton and colleagues' (2001) findings that even 1-2% gains are impactful for trained athletes. Overall, the findings suggest BFR's potential to enhance CP capabilities, although further research explicitly evaluating BFR's impact on CP is needed.

The intermittent performance improvement exceeded improvements in individual physiological components like 30-s power or muscle reoxygenation rate. These data indicate the intermittent test captured the cumulative, synergistic effects of BFR training on multiple parameters underpinning the W'_{BAL} model and TP performance, as outlined in Chapter Three - including CP, W′ reconstitution kinetics, and intermittent exercise capabilities.

Mechanistically, the observation that BFR increased the submaximal lactate threshold (LT1) suggests BFR training can enhance the power output separating moderate from heavy exercise intensity domains. By increasing LT1/GET, BFR may have shifted this critical reconstitution intensity region closer to CP, consequently accelerating W′ reconstitution during intermittent recovery phases and improving tolerance, which was identified as a key TP determinant in Chapter Three. However, without directly assessing CP before and after BFR training, the precise impact on the reconstitution dynamics and power-tau relationship remains speculative. A summary of the potential adaptation effects of endurance BFR training is presented in Figure 27, with the proposed interaction with W'_{BAL} parameters included.

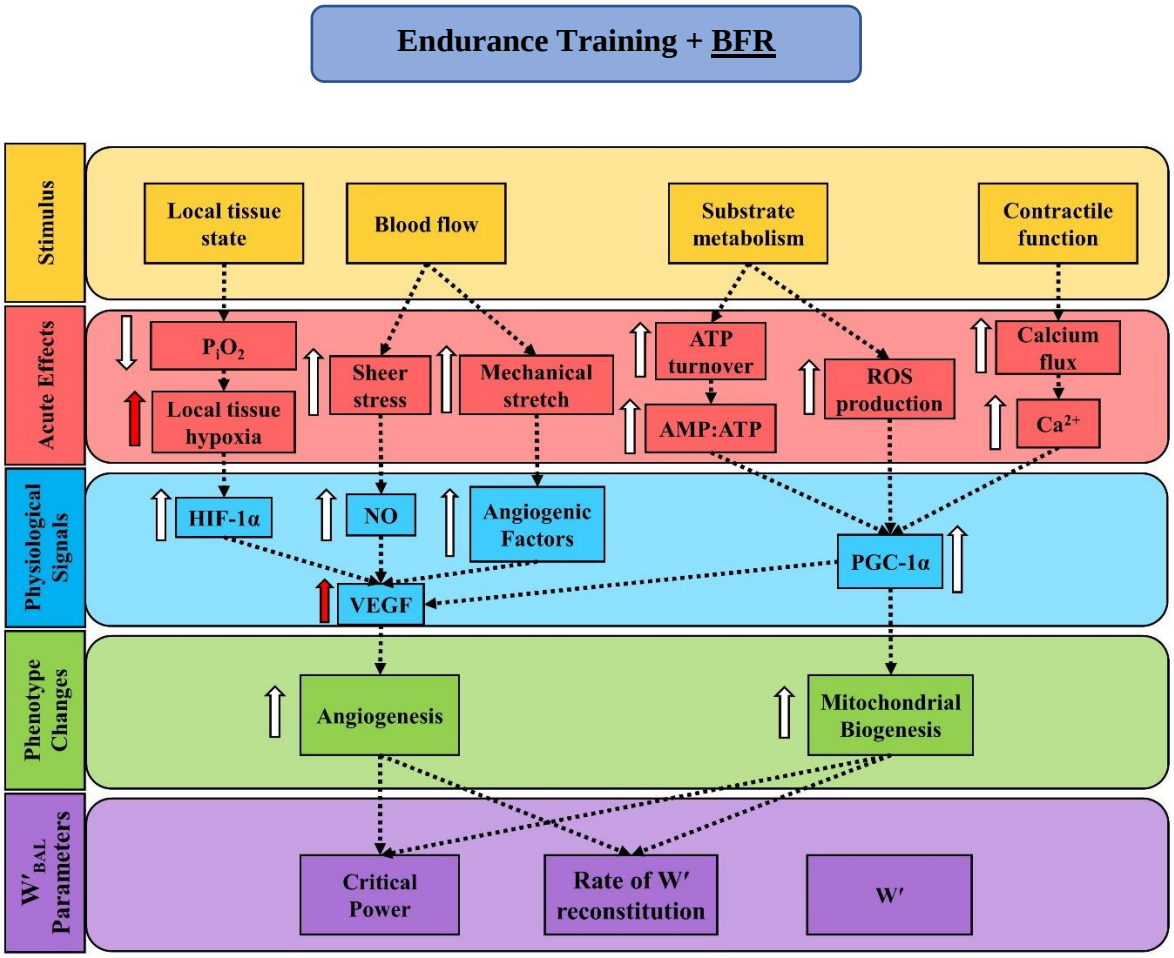


Figure 27. Hypothesised (white arrows) and measured (red arrows) signalling effects of blood flow restriction on angiogenesis and mitochondrial biogenesis.

Abbreviations and definitions: AMP, adenosine monophosphate; ATP, adenosine triphosphate; Ca^{2+}, calcium ion; HIF-1α, hypoxia-inducible factor 1-alpha; NO, nitric oxide; PGC-1α, peroxisome proliferator-activated receptor gamma coactivator 1-alpha; P_iO_2, inspired partial pressure of oxygen; ROS, reactive oxygen species; VEGF, vascular endothelial growth factor; W', work capacity above critical power; W'_{BAL}, dynamic balance of work capacity during intermittent exercise. Adapted from Mitchell (2019)

Integrating blood flow restriction into training for elite team-pursuit cyclists – a case-study

The findings from Chapters Four and Five were implemented in the training regimen of elite TP cyclists preparing for the Olympics. The aim was to use BFR to enhance their high-intensity training. The case-study provided an opportunity to explore the feasibility and practicality of integrating a BFR stimulus into the routine training of world-class TP cyclists. Notably, the elite TP cyclists found the BFR intervention feasible to integrate into their training routine. Muscle oxygenation levels were reduced to

a greater extent during BFR compared to non-elite cyclists, while interval power output was not negatively affected. These findings suggest similar acute effects for elites and non-elites. Additionally, the case-study highlighted the extensive low-intensity training volumes undertaken by these cyclists, which are proposed to have further strengthened their underlaying aerobic capabilities (Maunder *et al.* 2021), CP, and W′ reconstitution rates, identified as crucial factors in Chapter Three. However, it is important to consider that by this stage, the research findings were known to the cyclists and coaching staff, potentially influencing training strategies.

One elite TP cyclist did not tolerate the BFR application and experienced adverse symptoms. This cyclist described the symptoms during BFR application as similar to those experienced during high-intensity training. An additional case-study evaluation revealed a distinct bilateral imbalance in power output and muscle TSI levels between the cyclist's legs during high-intensity efforts above ~200 W. This asymmetry indicated an underlying vascular condition known as external iliac artery endofibrosis (EIAE). Of note elite cyclists are particularly susceptible to due to the repetitive hip flexion involved with cycling and large training volumes (Schep *et al.* 2002); however, EIAE often goes undiagnosed due to symptoms resembling those of high-intensity exertion and can be masked by biomechanical compensation (Bender *et al.* 2004).

While not recommended as a diagnostic tool, this adverse experience highlighted how the BFR stimulus may act as a provocative intervention to unmask latent vascular limitations like EIAE. Simultaneously monitoring bilateral muscle oxygenation levels via NIRS during high-intensity cycling with BFR proved effective for detecting the imbalance that prompted clinical investigation and diagnosis of EIAE in this case.

An integrated model for optimising W'_{BAL} parameters and team-pursuit performance using blood flow restriction between high-intensity intervals.

The research findings collectively suggest a framework where maximising TP performance requires optimising the parameters of the W'_{BAL} model. The addition of BFR to HIIT indicates potential strategies to enhance all three parameters:

1. BFR can improve markers of aerobic fitness, such as lactate threshold and $\dot{V}O_{2PEAK}$, potentially elevating CP. A higher CP allows for more opportunities to recover while drafting and preserves a greater portion of W′ for intense lead efforts. The proposed mechanism is that the increased shear stress and local muscle hypoxia during BFR promote the release of the angiogenic factor VEGF, improving O_2 exchange and aerobic capacities.

2. BFR may enhance metabolite clearance, delaying the onset of fatigue at high-intensities, and potentially increasing W′. Evidence for this includes improved 30-s power output, which could result from enhanced glycolytic power. A greater W′ improves tolerance of the intense TP start and lead efforts. However, once an adequate TP-specific W′ base is established, further augmenting W′ alone may provide diminishing returns.

3. BFR can heighten metabolic stresses and angiogenic signalling, potentially leading to accelerated W′ reconstitution kinetics. A faster W′ reconstitution extends intermittent exercise tolerance by replenishing the capacity for high-intensity exercise at a higher rate. The proposed mechanism is that an elevated capillary density enables improved muscle reoxygenation and metabolite clearance during recovery.

Therefore, integrating BFR during recovery phases of HIIT can amplify the training stimulus without increasing mechanical workload. The additive effects on CP, W′, and W′ reconstitution rate provide a potential approach to optimise the parameter profiles underpinning TP performance capabilities.

Limitations

While the research presented in this thesis has generated practical insights for training and pacing strategies in TP cycling, it is important to acknowledge several key limitations across the body of work. These limitations primarily pertain to modelling and measurement approaches, intervention design considerations, and trade-offs involving ecological validity.

Firstly, the modelling and measurement approaches employed in this research may have impacted the accuracy and precision of certain estimates. Although the standard errors of parameter estimates were within acceptable limits to provide confidence in the models, the 2-parameter CP modelling approach, with durations of 1-, 4-, and 10-minutes, may not provide the most precise estimation of the boundary between heavy and severe exercise intensity domains. While longer test durations could potentially improve CP determination accuracy, this may come at the cost of reduced W′ resolution around the TP event duration. However, it is important to recognise that the CP trial format and W′ reconstitution trials used in Chapter Three were previously validated by Bartram *et al*. (2018, 2021). Employing Bartram's format of CP trial durations in the present study facilitated comparisons of CP, W′, and W′ reconstitution kinetics between datasets. Furthermore, the initial high torque, low cadence start phase of the TP may have influenced CP and introduced inaccuracies in TP-specific W'_{BAL} modelling. In an effort to control for the TP start, all CP trials began from a stationary position. However, trials assessing W′ reconstitution kinetics did not replicate the high torque start, as ergometer resistance was less reliable from a stationary flywheel start.

Secondly, certain aspects of the intervention design may have introduced potential biases or limitations. The work to recovery ratio used in the BFR condition (effectively 1:1 with cuff inflation) potentially biased adaptations compared to the 1:9 ratio in the control condition. Including an additional work-matched 1:1 control would have isolated whether enhancements arose specifically from BFR or the work-recovery ratio. Additionally, using a consistent 200 mmHg cuff pressure across participants, while enhancing real-world applicability, did not account for individual differences in arterial occlusion levels influenced by factors like limb size, blood pressure, or adipose tissue thickness (Hunt *et al.* 2016).

Thirdly, in the chronic training study, not measuring CP, W′, and W′ reconstitution pre- and post-intervention precluded direct assessments of changes in these key performance determinants identified in Chapter Three. While this compromise enhanced participant recruitment, it limited a comprehensive evaluation of these factors. Furthermore, the intermittent test reflected TP demands but used a lower recovery intensity than competition, potentially underestimating improvements in W′ reconstitution relevant to the hyperbolic reconstitution region identified. Adopting fixed test intensities throughout, rather than updating based on post-intervention performance measures, may have impacted the sensitivity to detect changes in reconstitution kinetics.

Lastly, while efforts were made to enhance ecological validity, certain trade-offs were unavoidable. The elite TP case-study (Chapter Six) provided valuable insights in a real-world setting but was constrained in rigorously quantifying performance outcomes due to the proximity to the Olympic Games and the applied training environment.

Practical applications

The observational and experimental studies presented in this thesis offer several practical applications that can inform training practices and strategies for TP cycling and other sports involving intermittent high-intensity exercise.

- The findings from Chapter Three highlight the importance of CP in reducing reliance on the finite W′ and facilitating faster W′ reconstitution during TP cycling. Cyclists aiming to enhance their TP performance may benefit from tailoring their training to improve CP and accelerating W′ recovery rate. The estimated W′ reconstitution rates across different performance levels (International to Regional) provide approximate W'_{BAL} modelling guidance. These findings can inform effective pacing strategies during track cycling events by leveraging the exponential relationship between recovery intensity and W′ reconstitution rate near CP. Additionally, assessing W'_{BAL} parameters may aid talent identification and athlete selection processes.

- Chapter Four highlights the acute effects of incorporating BFR between high-intensity intervals. This intervention elicits significant physiological changes while maintaining a high-intensity exercise stimulus without increasing mechanical workload. Notably, participants tolerated the BFR intervention well, and it did not adversely affect their workout performance. This finding suggests the potential for incorporating BFR into training regimens to strategically modulate training stimuli and adaptations.

- The chronic integration of BFR into recovery phases between HIIT work bouts, as demonstrated in Chapter Five, suggests potential for enhancing adaptations to HIIT. Specifically, after eight sessions of BFR between HIIT work bouts, notable improvements were observed in intermittent high-intensity exercise performance, which is particularly relevant to TP cycling. This approach also showed indications of benefits for factors related to intermittent performance, such as 30-s mean power output, submaximal lactate threshold, $\dot{V}O_{2PEAK}$, and muscle reoxygenation. While these findings are promising, the small sample size warrants caution in generalising the results. Nonetheless, they provide initial evidence for the potential of BFR intervention not only for peaking before competition but also for use during general preparation phases of periodised training.

- The use of a consistent 200 mmHg BFR pressure, as employed in this research, allows for easy replication in field settings with minimal equipment, despite potential inter-individual differences in occlusion degree. Coaches and athletes can consider integrating BFR into the recovery phases of HIIT to strategically modulate responses and induce targeted adaptations according to their specific goals and training programs.

- The case-study of the elite track cycling squad in Chapter Six provides initial evidence for the feasible integration of BFR into endurance training to diversify training stimuli for athletes. The reported moderate to high pain ratings and reduced muscle oxygenation data may help set

expectations for athletes and coaches considering incorporating BFR. Overall, the successful integration of BFR in high-performance training settings demonstrates its viability beyond controlled research environments.

- While the findings from this thesis primarily focus on TP cycling, the implementation of BFR may also benefit other sports requiring intermittent high-intensity exercise, such as ice-skating team-pursuit, soccer, or rugby, through similar mechanisms. However, further research is needed to optimise BFR protocols for specific athletic populations and validate performance effects across different sports.

Future directions

The outcomes and results presented within this thesis highlight several key areas for future research endeavours:

- Further research on W'_{BAL} modelling for TP should consider incorporating the near-maximal power output of the initial TP lap into W' reconstitution testing. This approach may enhance the accuracy and ecological validity of W'_{BAL} modelling for the TP event. Additionally, efforts should focus on developing testing protocols that offer robust estimates while minimising the training burden on athletes.

- Further research is warranted to examine the acute effects of BFR applied between high-intensity intervals. These studies should assess additional markers of metabolic and oxidative stress, as well as evaluate the expression of mRNA proteins associated with chronic physiological adaptations that can enhance performance. Relevant markers may include indicators of muscle damage, mitochondrial biogenesis, and vascular function.

- To better understand the nuances of how BFR affects W′ reconstitution and TP performance, it is essential to examine the effects of BFR on the slow and fast components of W′ reconstitution separately. This information would provide insights into the specific mechanisms through which BFR influences the recovery process.

- Directly quantifying W′ reconstitution rates before and after BFR intervention can provide insights on whether the time constant of recovery can be modified with this technique, potentially leading to enhanced recovery strategies.

- Investigating the optimal integration of BFR into HIIT protocols is necessary to maximise adaptations. This includes exploring factors like occlusion pressure, cuff width, restriction time, and exercise intensity. Identifying the best integration within periodised training can enhance recovery capacity and targeted adaptations.

- Research should explore the potential for faster BFR application and how immediate occlusion following high-intensity cycling may impact the results. This could inform more time-efficient strategies for implementing BFR within training sessions.

- Examine the longer-term efficacy and safety of BFR-integrated training is crucial, including determining ideal progression models and implementation schedules. This would ensure that the benefits of BFR can be sustained over extended periods while mitigating potential risks.

- To optimise W′ reconstitution, it is imperative to investigate the adaptability of both the slow and fast components by exploring a range of training methods, intensities, and training volumes beyond BFR. This comprehensive approach would contribute to a more holistic understanding of recovery strategies for intermittent high-intensity sports.

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Addendum

An adverse reaction to a blood flow restriction intervention: possible case of external iliac artery endofibrosis.

Prelude

An adverse reaction experienced by an individual elite TP cyclist during BFR intervention highlighted the need for further assessment into the potential causes of intolerance.

Introduction

One elite team-pursuit (TP) cyclist exhibited an adverse response during the initial application of blood flow restriction (BFR), reporting similar sensations to those experienced at high exercise intensities during their normal training. The cyclist described a history of severe lactate accumulation sensations and impaired performance isolated to the left leg during strenuous efforts, which predated the BFR intervention. This adverse reaction and historical symptoms exhibited by the cyclist suggest an underlying exercise-induced cardiovascular disorder affecting the left leg.

The utilisation of O_2 in an exercising muscle is determined by the muscle's blood flow and O_2 extraction, as described by the Fick equation. An imbalance between O_2 supply and demand can be caused by increased muscle O_2 extraction or insufficient muscle blood flow, which can result in a reduced muscle tissue O_2 saturation index (TSI) (Grassi and Quaresima 2016). A reduced TSI leads to a lower partial pressure of O_2 in the microvasculature and diminished diffusion of O_2 from capillaries to mitochondria, impairing skeletal muscle oxidative metabolism (Poole *et al.* 2012). Impaired oxidative metabolism can increase reliance on glycolytic pathways, leading to greater metabolite production, such as lactate and hydrogen ions (Spriet 2022). An increased reliance on glycolytic energy systems could potentially explain the cyclist's symptoms of severe lactate accumulation.

While the cyclist had no preexisting diagnoses, their adverse reaction symptoms suggest possible external iliac artery endofibrosis (EIAE), a condition characterised by arterial narrowing (Getzin and Silberman 2010). Elite cyclists are particularly susceptible to EIAE due to repetitive hip flexion during cycling, which usually manifests unilaterally but can also occur bilaterally (Schep *et al.* 2002). In TP cycling position, factors associated with EIAE may be exacerbated by the pronounced hip flexion required to maintain an aerodynamic position, the high cadence resulting in more frequent hip flexion, and the maximal exertion. Symptoms of leg weakness, cramping, and fatigue with exertion are consistent with the cyclist's reaction and known EIAE presentations (Abraham *et al.* 1997). However, EIAE is likely underdiagnosed in cyclists, as symptoms may be attributed to high-intensity exercise. Diagnosis often involves provocative testing, though prevalence estimates range widely from 0.66% (Fernández-García *et al.* 2002) to 20% in competitive cyclists (Bender *et al.* 2004).

Recent research suggests that near-infrared spectroscopy (NIRS) can improve diagnostic accuracy for detecting flow limitation in the iliac artery (van Hooff *et al.* 2022). NIRS has also shown efficacy in diagnosing peripheral artery disease, with slower reoxygenation kinetics in affected versus healthy limbs (Cornelis *et al.* 2021). However, one previous study found NIRS could not differentiate athletes with external iliac artery endofibrosis from controls (Julienne *et al.* 2018). The Julienne *et al*. study had several methodological limitations that may have reduced the ability to detect EIAE, including not measuring power output differences between legs, lack of control screening for undiagnosed EIAE, and assessing athletes in recumbent versus cycling positions.

The aim of this case-study was to describe the acute adverse reaction to BFR and chronic left leg symptoms that suggest an undiagnosed case of EIAE in the cyclist. The cyclist performed a typical training session with supplementary high-intensity cycling to provoke symptoms while we monitored bilateral power output and muscle oxygenation. We hypothesised that analysing power output and oxygenation differences between the legs could reveal localised asymmetry and indicate a possible vascular condition affecting the left leg.

Methods

An elite track endurance cyclist, who had represented their nation at World Championship events, was invited for follow-up testing after experiencing an adverse response to a BFR intervention. To measure the physiological conditions during their reported symptoms, we observed the cyclist complete a typical 50-minute training ride on their personal road bicycle. The ride included a ~5-minute period of near-maximal exertion at over 400 W to provoke their symptoms, ascents (514-m vertical elevation gain), descents, and periods of active recovery.

Power output measurement

Bilateral power output (in Watts, W) was measured using independent crank-based power meters (InfoCrank, Verve Cycling, Australia). The InfoCrank power meter has a reported mean deviation (accuracy) similar to a mathematical model of treadmill cycling and variability (precision) comparable to the gold standard SRM power meter (Maier *et al.* 2017).

Muscle oxygenation measurement

Muscle tissue O_2 saturation index (TSI in %) was determined via near infrared spectroscopy (NIRS) sensors (Moxy 3, Fortiori Design LLC, Minnesota, USA). Sensors were secured with a compression bandage to the left and right vastus-lateralis muscle bellies at 40% of the distance between the greater trochanter and the lateral epicondyle of the femur.

Data collection and analysis

Power output and NIRS data were recorded at 1 Hz using a cycling computer (Edge 1030, Garmin Ltd., USA). Raw data files were downloaded to Golden Cheetah software (version 3.5, open-source) for initial processing before being imported into Excel (Microsoft Corporation, USA) for analysis. The relative power output distribution for each leg was determined by the formula:

$$Relative\ Power\ Output\ Distribution = \frac{(Left\ Leg\ Power\ Output - Right\ Leg\ Power\ Output)}{(0.5 \cdot Combined\ Leg\ Power\ Output\ )} \cdot 100 \quad \text{Equation 17}$$

The relative power output distribution quantifies the deviation of each leg's power output from the average, indicating whether the right or left leg is dominant.

Data were separated into distinct power output ranges of: <200 W, 200-250 W, 250-300 W, 300-350 W, 350-400 W, 400-450 W, 450-500 W, 500< W. In each power output range, the n, mean, and standard deviation of samples were calculated. For graphical representations, data were smoothed using 10-s moving averages. The relationship between power output and relative power output distribution between legs was assessed using Pearson correlation. Figures were constructed using GraphPad Prism (version 9.5.1, GraphPad Software, San Diego, CA, USA) to illustrate raw power output and TSI traces, as well as the relationship between power output and power output imbalance between legs.

Results

Figure 28 Panels A-D illustrate the entire cycling session for power output, bilateral power output balance, TSI, and total haemoglobin, while Panels E-H provides a higher resolution of data during a high-intensity bout. The measures of power output distribution and TSI are presented for each power output band in Table 11. At cycling intensities up to 200 W, power output distribution and TSI values were generally bilaterally symmetrical. However, above 200 W, asymmetry of power output distribution and TSI emerged, with the left leg experiencing a reduced relative power output contribution and TSI. The asymmetry of TSI and power output distribution coincided with verbal indications of the sensations experienced during the BFR application.

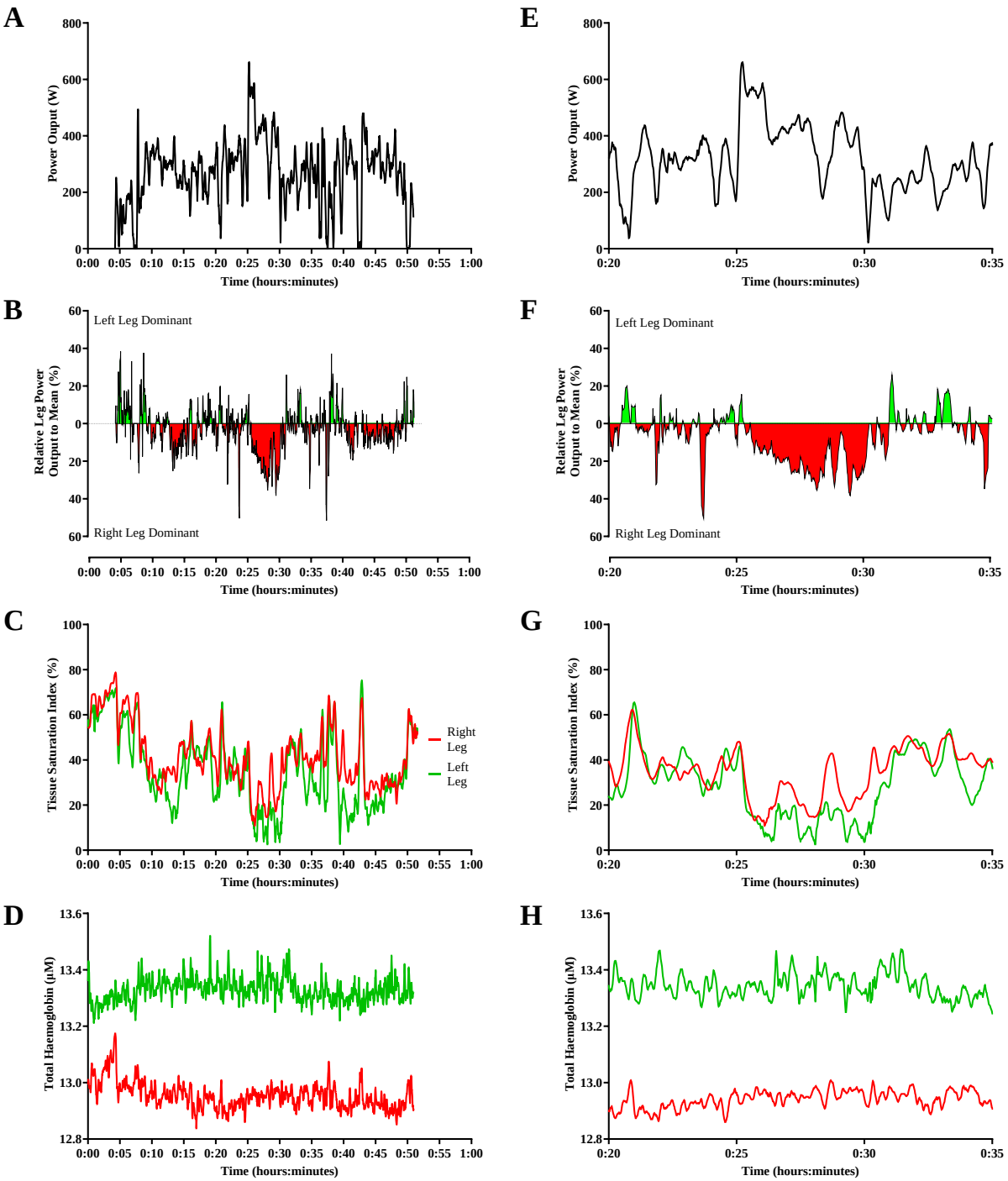

Figure 28. Physiological responses during road cycling in a participant with leg muscle oxygenation and power output imbalances.

Panels A-D shows the entire road cycling session, whereas panels E-G offers a detailed view of a prolonged high-intensity effort, specifically focusing on a high-intensity bout.

Table 11. Relative distribution of power output between legs, and tissue oxygen saturation in a cyclist at various intensities.

		Relative Power Output Distribution Between Legs	Tissue Oxygen Saturation (%)	
Intensity range (W)	Count of Data Points	Difference to Mean, and Dominant Leg	Left Leg (impacted leg)	Right Leg
<200	532	2 ± 30%, Left	43 ± 15	50 ± 12
200-250	412	2 ± 19%, Right	35 ± 14	43 ± 10
250-300	552	4 ± 16%, Right	32 ± 13	38 ± 9
300-350	492	4 ± 13%, Right	29 ± 13	35 ± 8
350-400	301	7 ± 16%, Right	26 ± 12	32 ± 8
400-450	179	13 ± 20%, Right	22 ± 14	31 ± 11
450-500	57	13 ± 16%, Right	25 ± 19	32 ± 15
500<	85	9 ± 15%, Right	26 ± 21	29 ± 18

This table illustrates the percentage difference between the power output of the dominant leg and the mean power output of both legs, alongside tissue oxygen saturation levels, across various intensity levels. The count of data points, mean percentage difference (and its direction), and oxygen saturation percentages for each leg are provided for each intensity range.

The Pearson correlation analysis provides a detailed insight into the influence of power output on the relative contribution of each leg to overall power output. As intensity increases, there is a notable shift towards the right leg for power output production, which is quantitatively supported by the significant ($p<0.001$) negative slope of -0.041 in our correlation analysis. The y-intercept is it 7.7% favouring the left leg, suggesting that at low-intensities, the left leg's power output contribution exceeds the right. However, at an intensity of 187 W (the x-intercept), both legs contribute equally. At intensities greater than 187 W, the right leg becomes increasingly dominant. Despite these significant trends, it is important to note that the R^2 value is 0.035.

Discussion

This observational case-study investigated an acute adverse reaction experienced by an elite TP cyclist during a BFR training session. During a typical road-based training session, we noted asymmetry in TSI and power output between the cyclist's legs. The left leg's contribution declined notably with increasing intensity, suggesting a potential exercise induced EIAE rather than a fixed anatomical constraint.

At cycling intensities exceeding 187 W, the left leg exhibited lower TSI and relative power output compared to the right leg. The lower TSI in the left leg indicates an imbalance between O_2 delivery and utilisation, implying inadequate O_2 supply to the left leg to meet the metabolic demands of moderate-to high-intensity cycling. While aerobic metabolism was likely impaired in the left leg at intensities greater than 187 W, the cyclist could still generate high power outputs using anaerobic glycolysis to compensate (Spriet 2022), leading to premature fatigue in the left leg. This compensation effect was evident from the left leg's dominance at lower intensities. These bilateral limb differences highlight the value of assessing both sides, as unilateral measurement could miss potential asymmetry. Previous research has also demonstrated the effectiveness of NIRS as a diagnostic tool to detect arterial flow limitations in cyclists during exercise (van Hooff *et al.* 2022).

The exertional symptoms and physiological response observed are consistent with known presentations of EIAE (Bender *et al.* 2004). The cyclist's reported symptoms during BFR, an intervention that purposefully restricts blood flow, provoked familiar symptoms. Further examination with medical personnel revealed that the cyclist had a pre-existing undiagnosed cardiovascular condition and showed symptoms of femoral artery claudication. These findings led to a referral for a definitive vascular evaluation, after which the cyclist was diagnosed with EIAE.

A limitation of the present study was that the participant knew we were evaluating potential limb asymmetries, which could have altered their relative effort between legs. However, the large imbalances measured at higher intensities likely reflect a true difference in perfusion and power-generating capacity between limbs. The Infocrank power meters used have a reported accuracy within 2% of actual power output across a range of intensities (Maier *et al.* 2017). While small variations in power measurement

accuracy may have influenced the results, the large asymmetry observed makes it unlikely to alter the overall interpretation. The Moxy NIRS sensors demonstrate excellent test-retest reliability for muscle TSI assessment when placement is consistent (Yogev *et al.* 2023). Although we obtained a single measurement, standardised sensor placement on the vastus lateralis likely provided a valid representation of limb-specific oxygenation.

Conclusion

An elite TP cyclist's adverse response to BFR training led to a bilateral evaluation during regular road cycling. This BFR protocol revealed disparities in power output and muscle O_2 saturation between the legs at high exercise intensities. These observations suggest that the adverse reaction to incorporating BFR into HIIT is potentially due to underlying vascular dysfunction, consistent with EIAE. This case highlights the value of provocative testing protocols and bilateral physiological measurements in identifying limb-specific abnormalities that may be overlooked during routine training. Despite the cyclist's partial compensation for the constraint, assessing leg symmetry and TSI at various cycling intensities revealed the full extent of the perfusion imbalance between the legs.

Appendix 1 – BFR questionnaire

General Thoughts

- What were your overall impressions of the BFR training?

Pain Levels

- Using the Borg CR10 RPE and pain scale, how would you rate the pain/discomfort during BFR?

- How did the pain or discomfort progress during each interval? Did it increase, decrease, or remain stable?

Pain Progression Across Reps

- On the Borg CR10 RPE and pain scale, how did the pain/discomfort change from the first interval to the last interval within each session?

Tolerance Over Time

- Did you notice any difference in tolerance or adaptation to the BFR between the first and second session? If so, how?

Comparison to Other Training

- How did the sensation of BFR compare to other training techniques you have experienced?

Enjoyment

- Did you find the BFR training enjoyable or not enjoyable? What contributed to this perception?

Feelings After Session

- How did your legs/body feel immediately after the BFR session?

Next Day Effects

- How did your legs/body feel the day after the BFR session? Any lingering effects?

Appendix 2 – Co-authorship forms

THE UNIVERSITY OF WAIKATO
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Co-Authorship Form

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Please indicate the chapter/section/pages of this thesis that are extracted from a co-authored work and give the title and publication details or details of submission of the co-authored work.
Chapter three
Pugh, C. F., C. M. Beaven, R. A. Ferguson, M. W. Driller, C. D. Palmer and C. D. Paton (2022). "Critical power, work capacity, and recovery characteristics of team-pursuit cyclists." International Journal of Sports Physiology and Performance 17(11): 1606-1613.

Nature of contribution by PhD candidate	Designed study, collected data, analysed data, wrote first manuscript draft, wrote and submitted final manuscript
Extent of contribution by PhD candidate (%)	80%

CO-AUTHORS

Name	Nature of Contribution
Beaven, C. M.	Contributed to study design, and manuscript drafting
Ferguson, R. A.	Contributed to study design, and manuscript drafting
Driller, M. W.	Contributed to study design, and manuscript drafting
Palmer, C. D.	Contributed to data collection, and manuscript drafting
Paton, C.D.	Contributed to study design, and manuscript drafting

Certification by Co-Authors

The undersigned hereby certify that:

- the above statement correctly reflects the nature and extent of the PhD candidate's contribution to this work, and the nature of the contribution of each of the co-authors; and

Name	Signature	Date
Beaven, C. M.		27/10/2023
Ferguson, R. A		24/10/2023
Driller, M. W		25/10/2023
Palmer, C. D		26/10/23
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July 2015

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Nature of contribution by PhD candidate	Designed study, collected data, analysed data, wrote first manuscript draft, wrote and submitted final manuscript
Extent of contribution by PhD candidate (%)	80%

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Driller, M. W.	Contributed to study design, and manuscript drafting
Beaven, C. M.	Contributed to study design, and manuscript drafting

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July 2015

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Nature of contribution by PhD candidate	Designed study, collected data, analysed data, wrote first manuscript draft, wrote final manuscript
Extent of contribution by PhD candidate (%)	80%

CO-AUTHORS

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Please indicate the chapter/section/pages of this thesis that are extracted from a co-authored work and give the title and publication details or details of submission of the co-authored work. Chapter six Pugh, C. F., C. D. Paton, R. A. Ferguson, M. W. Driller, and C. M. Beaven (2023) "Integrating blood flow restriction into training for elite team-pursuit cyclists – a case-study"

Nature of contribution by PhD candidate	Designed study, collected data, analysed data, wrote first manuscript draft, wrote final manuscript
Extent of contribution by PhD candidate (%)	80%

CO-AUTHORS

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Ferguson, R. A.		24/10/2023
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Beaven, C. M.		27/10/2023

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Appendix 3 – Ethical approval

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THE UNIVERSITY OF WAIKATO
Te Whare Wānanga o Waikato

5 August 2020

Charlie Pugh
Te Huataki Waiora, School of Health
DHECS
By email: cp115@students.waikato.ac.nz

Dear Charlie

HREC(Health)2020#57 : Key Determinants of Team Pursuit Performance in Track Cycling

Your application for HREC(Health)2020#57 was considered on 21 July 2020 by the University of Waikato Human Research Ethics Committee (Health). The Committee noted your application was initially approved by HECS Committee as HREC(HECS)2020#01 and that the study design has had minor adjustments from the initial ethics submission which are included in your new application.

We are pleased to provide formal approval for your project.

Please contact the committee by email (humanethics@waikato.ac.nz) if you wish to make changes to your project as it unfolds, quoting your application number with your future correspondence. Any minor changes or additions to the approved research activities can be handled outside the monthly application cycle.

We wish you all the best with your research.

Regards,

Emeritus Professor Roger Moltzen MNZM
Chairperson
University of Waikato Human Research Ethics Committee

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THE UNIVERSITY OF WAIKATO
Te Whare Wānanga o Waikato

10 May 2021

Charlie Pugh
Te Huataki Waiora, School of Health
DHECS
By email: cp115@students.waikato.ac.nz

Dear Charlie

HREC(Health)2021#22 : Acute effects of interval training with post-exercise blood flow restriction

Thank you for your responses to the Committee feedback.

We are now pleased to provide formal approval for your project.

Please contact the committee by email (humanethics@waikato.ac.nz) if you wish to make changes to your project as it unfolds, quoting your application number with your future correspondence. Any minor changes or additions to the approved research activities can be handled outside the monthly application cycle.

We wish you all the best with your research.

Regards,

Emeritus Professor Roger Moltzen MNZM
Chairperson
University of Waikato Human Research Ethics Committee

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THE UNIVERSITY OF WAIKATO
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3 September 2021

Charlie Pugh
Martyn Beaven
Richard Ferguson
Carl Paton
Matthew Driller

Re: HECS Ethics Approval of Application HREC(HECS)2021#40 "Effects of post-exercise blood flow restriction on aerobic and intermittent exercise performance"

Dear Charlie:

Thank you for submitting your amended application HREC(HECS)2021#40 for ethical approval.

We are pleased to provide formal approval for your project, including the following activities:

- Recruit approximately 12 well-trained adult cyclists to randomly assign to control and blood-flow restricted (BFR) groups.
- Have participants complete 4-weeks of high-intensity interval training sessions consisting of 6 repetitions at 85% of maximum 30-second power output with 5-minutes recovery between efforts per session.
- Apply pneumatic cuffs to the upper quadriceps in the BFR group for 2-minutes while the BFR and control groups lay supine on a bed during the recovery.
- Perform $\dot{V}O_2$ max and intermittent exercise tests one week before and one week after the intervention to assess the efficacy of the training.
- Total time commitment for participants will be less than 6 hours over 6 weeks.

Please contact the committee by email (hecs-ethics@waikato.ac.nz) if you wish to make changes to your project as it unfolds, quoting your application number with your future correspondence. Any minor changes or additions to the approved research activities can be handled outside the monthly application cycle.

We wish you all the best with your research.

Kind regards,

Brett Langley, PhD
Chairperson
HECS Human Ethics Committee
University of Waikato

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THE UNIVERSITY OF WAIKATO
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2 February 2021

Charlie Pugh
Martyn Beaven
Richard Ferguson
Carl Paton
Matthew Driller

Re: HECS Ethics Approval of Application HREC(HECS)2021#04 "Perceptual and physiological observations from blood-flow restricted leg cycling"

Dear Charlie:

Thank you for submitting your amended application HREC(HECS)2021#04 for ethical approval.

We are pleased to provide formal approval for your project, including the following activities:

- Recruit approximately 5 male high-performing cyclists (aged 20-30) from Cycling New Zealand's Blood-flow Restricted (BFR) Training Program.
- Provide Borg's perceived exertion (RPE) and Pain questionnaires to understand the participants perceived responses to exercise.
- Monitor heart rate via a chest-strap.
- Monitor muscle oxygen saturation (StO_2) of the quadriceps via light sensor (NIRS).
- Monitor blood pressure from a cuff on the upper arm.
- Monitor blood haemoglobin oxygen saturation using a finger-mounted light sensor (SpO_2).

Please contact the committee by email (hecs-ethics@waikato.ac.nz) if you wish to make changes to your project as it unfolds, quoting your application number with your future correspondence. Any minor changes or additions to the approved research activities can be handled outside the monthly application cycle.

We wish you all the best with your research.

Kind regards,

Brett Langley, PhD
Chairperson
HECS Human Ethics Committee
University of Waikato