SPRINT CYCLING: PHYSIOLOGICAL AND MOTIVATIONAL CONSIDERATIONS FOR PERFORMANCE AND RECOVERY

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ABSTRACT

The Match Sprint is a highly tactical track cycling discipline that requires athletes to race on multiple occasions each day and over consecutive days. The time between races varies depending on the competition schedule, but when the recovery period is brief (10 – 30 minutes) the current practice undertaken by elite riders consists of a mixture of light cycling and passive rest, although this is not evidence based. The aim of this PhD thesis was, therefore, to investigate motivational and physiological factors that could affect sprint cycling performance over repeated efforts. Performance was primarily evaluated as mean power output (MPO) during each sprint, with peak power output (PPO) being assessed as a secondary variable. In the first study (Chapter 4), fifteen strength-trained men (age: 24 ± 6 years; height: 1.81 ± 0.08 m; body mass: 83.4 ± 8.4 kg) visited the laboratory on eight occasions. During the first trial, the participants were familiarised with the performance measure, an 18 s cycling sprint, before a ramp test was performed to exhaustion for the determination of maximal oxygen uptake. The second trial was the baseline trial. In addition to performing an 18 s sprint, blood lactate concentration, tissue saturation index, and oxygen uptake were monitored during 12 minutes of passive recovery after the sprint. In the remaining trials, the recovery duration was varied (45, 90, 135, 180, 360, and 720 s) between two 18 s sprints, facilitating the mathematical modelling of performance recovery. One- and two-phase exponential functions were used to model the recovery time-course of MPO, as well as the recovery of the physiological variables. Correlation analyses were then conducted to assess the relationships between the recovery time constant for MPO and the physiological variables. The strength of these relationships ranged from trivial (r = -0.026) to moderate (ρ = -0.342), but was on all occasions not significant. Effects of sprint number (MPO: $F_{(1,14)}$ = 66.901, p < 0.001, η_p^2 =0.827; PPO: $F_{(1,14)}$ = 73.177, p < 0.001, η_p^2 = 0.839), recovery time (MPO: $F_{(5,70)}$ = 36.294, p < 0.001, η_p^2 = 0.722; PPO: $F_{(5,70)}$ = 4.975, p = 0.001, η_p^2 = 0.262), and a sprint number × recovery time interaction were found on both MPO and PPO (MPO: $F_{(2.160,30.242)}$ = 52.095, p < 0.001, η_p^2 = 0.788; PPO: $F_{(2.617,36.639)}$ = 10.553, p < 0.001, η_p^2 = 0.430), with *post hoc* tests revealing significant differences between sprints at all time-points for both variables. The main finding from the study was, therefore, that performance recovery was not complete over a recovery duration that may occur during a competition.

One limitation with the methodological design in Chapter 4 was the requirement for the participants to remain stationary on the ergometer between sprints. A strict passive recovery was used to limit the movements of the participants and to facilitate the controlled measurement of the physiological variables in recovery. A strict passive recovery would not, however, reflect a real-world scenario. Therefore, in Study 2 (Chapter 5), the performance effects of a mixture of active and passive

recovery, a protocol that is currently used by elite track cyclists, was contrasted with passive recovery. In addition, to explore the consideration that the reduction in second sprint performance that was found in Chapter 4 was as a result of a change in motivation, an alteration in second sprint duration was also included in the investigation. Sprint duration has been found to affect the effort provided by participants during sprint and repeated-sprint tasks. Twenty-four strength trained men (age: 26 ± 5 years; height: 180.3 ± 6.1 cm; body-mass: 82.3 ± 6.9 kg) participated. During each of the four experimental trials, two sprints were performed 12 minutes apart. The recovery activity between sprints was either a mixture of active and passive recovery or was just passive recovery. The first sprint was always 18 s, but the second sprint was either 9 s or 18 s. In addition to MPO and PPO, lactate concentration, ratings of sprint preparation, ratings of sprint performance, and perceptions of recovery, were measured. Post-trial and post-study questionnaires were also completed, exploring factors that may have influenced performance that day. A sprint number × recovery method interaction ($F_{(1,23)}$ = 28.791, p < 0.001, η_p^2 = 0.556) was found on PPO, with a significantly lower PPO in sprint 2 following passive recovery. A sprint number × recovery method interaction was not, however, found on MPO ($F_{(1,23)}$ = 2.513, p = 0.127, η_p^2 = 0.098). Sprint number × second sprint duration interaction effects were found on both PPO ($F_{(1,23)}$ = 9.867, p = 0.005, η_p^2 = 0.300) and MPO over the first 9 s of the sprint ($F_{(1,23)}$ = 8.922, p = 0.007, η_p^2 = 0.279), although *post hoc* tests were unable to identify the cause of either effect. Nonetheless, the existence of these sprint number × second sprint duration interaction effects, combined with responses provided to the questionnaires, provided some evidence of a change in effort depending on the duration of the task. Pre-testing data collection was then conducted (Chapter 6) to evaluate whether a greater performance loss effect size would be generated between two sprints if longer sprints (27 s compared to 18 s) were undertaken. A larger performance loss effect size could increase the probability of identifying an effect of a change in effort on repeated-sprint performance. Eight strength-trained men (25 ± 6 years; 180.4 ± 6.6 cm; 84.5 ± 8.4 kg) that had participated in Chapter 5 visited the laboratory on a single occasion. During the visit, two 27 s sprints were undertaken 12 minutes apart, with a mixture of active and passive recovery performed between sprints. The performance loss effect size was then calculated using Hedges g. Whilst the size of the effect did remain small, the effect size was found to be greater when longer sprints were undertaken (27 s sprints: Hedges g = 0.20; 18 s sprints: Hedges g = 0.13). Therefore, 27 s sprints were used in the final study.

The aim of the final study (Chapter 7) was to examine whether a simulated competition would affect repeated-sprint performance. It was proposed that the motivational effect of competition could affect performance as a result of a stress response, heightening readiness to perform. Sixteen resistance-trained men (age: 25 ± 4 years; height: 1.80 ± 0.07 m; body-mass: 83.3 ± 10.9 kg) participated. In both the control and simulated competition conditions, two 27 s sprints were

undertaken 12 minutes apart. Recordings of the R-wave to R-wave (R-R) interval duration were taken at rest, after the warm-up, and at the end of the trial for the measurement of heart rate variability (HRV), with saliva samples being taken at the same time-points for the assessment of alpha amylase (AA) activity and AA output. Five motivational components (crowd presence, financial reward, leaderboard, performance feedback, and verbal encouragement) were included in the simulated competition. Both MPO ($F_{(1,15)}$ = 12.419, p = 0.003, η_p^2 = 0.453) and PPO ($F_{(1,15)}$ = 23.760, p < 0.001, η_p^2 = 0.613) were found to be higher in the simulated competition. Alterations were also found in several HRV metrics (mean heart rate, mean R-R, the root mean square of successive differences, low frequency normalised (nu) power, high-frequency (HF) power, HFnu, and the Poincaré plot standard deviation perpendicular to the line of identity and the Poincaré plot standard deviation along the line of identity), as well as in AA activity ($F_{(1,10)}$ = 6.401, p = 0.030, η_p^2 = 0.390) and AA output ($F_{(1,10)}$ = 5.342, p = 0.043, η_p^2 = 0.348), suggesting that greater levels of physiological stress were experienced during the competition.

Overall, the findings from this PhD highlighted sprint performance and recovery considerations for athletes competing in the Match Sprint, with MPO and PPO being consistently reduced when a second sprint was performed with a recovery duration that may occur during a competition. The current practice of performing a mixture of active and passive recovery was found to aid with the recovery of PPO, although the effect on MPO was not apparent. Further investigation may, therefore, be required to guide best practice. Competition may also improve sprint cycling performance. The performance changes that were found in the simulated competition mean that competition could be used as a motivational training tool. The effect of competition on performance also generated methodological questions for researchers investigating sprint cycling performance, when the aim is for the performance to provide a better representation of a true maximal effort or in instances when researchers are seeking to generalise their findings to sports competitions.

LIST OF PUBLICATIONS AND PRESENTATIONS

Papers

- Dale, J., Muniz, D., Cimadoro, G., & Glaister, M. (2022). The short-term recovery of sprint cycling performance. *Journal of Science and Cycling, 11*(3), 33-46. https://doi.org/10.28985/1322.jsc.11

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Presentations

- Dale, J. (2019). Understanding factors that affect performance in physically and psychologically demanding tasks – short-term recovery for sprint cycling performance. Sport Health and Exercise Science Research Day, St Mary's University, London. (Oral presentation).

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List of Abbreviations

~	approximately
<	less than
>	more than
≤	less than or equal
τ	time constant tau
$t_{1/2}$	half-life
A	amplitude
AA	alpha amylase
ADP	adenosine diphosphate
ATP	adenosine triphosphate
$BLCk_1$	blood lactate clearance velocity constant
FAD	flavin adenine dinucleotide
$FADH_2$	dihydroflavine adenine dinucleotide
H^+	hydrogen ion
Hb	haemoglobin
HF	high frequency
HHb	deoxygenated haemoglobin
HRV	heart rate variability
ICC	intraclass correlation coefficient
LF	low frequency
LT1	first rise in blood lactate concentration
Mb	myoglobin
MCT	monocarboxylate transporter
MPO	mean power output
MPO_9	mean power output over 9 s
MPO_{18}	mean power output over 18 s

MRT	mean response time
NAD	nicotinamide adenine dinucleotide
NADH	reduced nicotinamide adenine dinucleotide
NIRS	near infrared spectroscopy
nu	normalised
O_2	oxygen
O_2Hb	oxygenated haemoglobin
pCa50	calcium required to achieve 50% peak tension
P_o	maximal isometric tension
PCr	phosphocreatine
Pi	inorganic phosphate
PPO	peak power output
RMSSD	root mean square of successive differences
RPE	rating of perceived exertion
ROF	rating of fatigue
SD1	poincaré plot standard deviation, perpendicular to the line of identity
SD2	poincaré plot standard deviation, along the line of identity
SDNN	standard deviation of normal-to-normal beats
SERCA	calcium ATPase pump
TBM	total body-mass
TCA	tricarboxylic acid cycle
tHb	total haemoglobin
TSI	tissue saturation index
ULF	ultra low frequency
VAS	visual analogue scale
VLF	very low frequency
V_{max}	maximal fibre shortening velocity
$\dot{V}O_2$	oxygen uptake

$\dot{V}O_{2off}$	oxygen uptake off-transition
$\dot{V}O_{2on}$	oxygen uptake on-transition
$\dot{V}O_{2max}$	maximal aerobic capacity
$\dot{V}O_{2peak}$	peak oxygen uptake
WAnT	wingate anaerobic test

Chapter 1: Introduction

1.1. INTRODUCTION

Velodrome, from the French word Vélocipède (an early bicycle) and the Greek word dromos (course), is a purpose-built venue for track cycling (Heijmans, & Mallon, 2011). Track cycling events featured in the first modern-day Olympics games held in Greece in 1896. Both sprint and endurance races were included, with distances ranging from one lap to 300 laps of the 333.3 m track (Lampros et al., 1897). In the shortest race, the one lap time trial, Paul Masson from France was the winner, completing the lap in 24 s (Lampros et al., 1897). On the same day, Masson also won the 2 km, as well as the 10 km, races (Lampros et al., 1897). At the 1900 Olympic Games in Paris, 69 competitors from seven countries entered the 1 km sprint (Mallon, 2009). The competition consisted of four rounds and in the first round 7 – 8 riders raced in each heat, with the top three riders progressing to the next round. In the quarter- and semi-finals, three riders competed in each race, but only the winner progressed (Mallon, 2009). The final then consisted of three riders, with the French rider, Albert Taillandier, winning the event (Mallon, 2009). The structure of sprint cycling events at the Olympics has continued to evolve and at the 2020 Tokyo Olympics, three sprint events featured. These were: the Match Sprint; the Team Sprint; and the Keirin.

The Match Sprint is a sprint track cycling discipline that begins with a qualifying round that is an individual time-trial, known as the Flying 200 (Parsons, 2010). For the Flying 200, each rider completes 3.5 laps of the track, with the time for the final 200 m being recorded. In the main competition, the rider with the fastest qualifying time is then matched to race against the rider with slowest time, the second fastest against the second slowest, and so forth (see Table 1.01 for the full competition format) with the winner of each race progressing to the next round (UCI, 2020). Each race will typically consist of two athletes competing against each other over three laps of the track (two laps on tracks that are greater than 333.3 m), although races can contain three or four riders, more commonly during the repechages (from the French word rescuing, a way for losing riders to re-enter the competition) or a race for places. Before each race begins, the riders will draw lots to decide which rider starts on the lower/higher side on the track. If the race is best-of-three (quarter finals onwards), in Race 2 the positions will be the reverse of those in Race 1, and if a deciding race is required (Race 3), lots will again be drawn. The rider that is on the lower side of the track must then initially lead the race, maintaining at least a walking pace until the pursuit line on the other side of the track is reached (see Figure 1.01). The riders are then permitted a maximum of two track stands of up to 30 s, where the rider balances in a stationary position. If a 30 s standstill occurs, the lead rider is instructed by the race official to continue (UCI, 2020). The sprint for the finish line can be initiated by either rider at any point over the three laps, but when the sprint has been initiated and the lead rider has entered the sprinters lane (the sprinters lane is between the black and red lines on the track – see

Figure 1.01), they must then remain there. The rider that follows may benefit from a reduction in air resistance, but will have to cover a greater distance to pass the lead rider to win the race (Track Cycling Academy, 2024). Therefore, the race distance is fixed (3 laps), but the duration of maximal effort for each sprint will vary depending on the tactics employed.

Table 1.01 Competition structure for the Match Sprint at the Tokyo Olympics (UCI, 2020).

Number of riders	Format	Event	Composition	1st	Other(s)
24	Round 1: 12 races, 2 riders in each race, winner progresses	1	Q1 – Q24	1A1	1A2
		2	Q2 – Q23	2A1	2A2
		3	Q3 – Q22	3A1	3A2
		4	Q4 – Q21	4A1	4A2
		5	Q5 – Q20	5A1	5A2
		6	Q6 – Q19	6A1	6A2
		7	Q7 – Q18	7A1	7A2
		8	Q8 – Q17	8A1	8A2
		9	Q9 – Q16	9A1	9A2
		10	Q10 – Q15	10A1	10A2
		11	Q11 – Q14	11A1	11A2
		12	Q12 – Q13	12A1	12A2
12	Repechages: 3 riders in each race, 4 riders progress	1	1A2 – 8A2 – 9A2	1B	Out, ranked according to their qualifying time
		2	2A2 – 7A2 – 10A2	2B	
		3	3A2 – 6A2 – 11A2	3B	
		4	4A2 – 5A2 – 12A2	4B	
16	Round 2: 8 races, 2 riders in each race, winner progresses	1	1A1 – 4B	1C1	1C2
		2	2A1 – 3B	2C1	2C2
		3	3A1 – 2B	3C1	3C2
		4	4A1 – 1B	4C1	4C2
		5	5A1 – 12A1	5C1	5C2
		6	6A1 – 11A1	6C1	6C2
		7	7A1 – 10A1	7C1	7C2
		8	8A1 – 9A1	8C1	8C2
8	Repechages: 2 riders in each race, 4 riders progress	1	1C2 – 8C2	1D1	Out, ranked according to their qualifying time
		2	2C3 – 7C2	2D1	
		3	3C2 – 6C2	3D1	
		4	4C2 – 5C2	4D1	
12	Round 3: 6 races, 2 riders in each race, winner progresses	1	1C1 – 4D1	1E1	1E2
		2	2C1 – 3D1	2E1	2E2
		3	3C1 – 2D1	3E1	3E2
		4	4C1 – 1D1	4E1	4E2
		5	5C1 – 8C1	5E1	5E2
		6	6C1 – 7C1	6E1	6E2
6	Repechages: 3 riders in each race, 2 riders progress	1	1E2 – 4E2 – 5E2	1F1	Out, ranked according to their qualifying time
		2	2E2 – 3E2 – 6E2	2F1	
8	Quarter-finals: best of three, 4 winners progress	1	1E1 – 2F1	1G1	Riders race for places 5-8
		2	2E1 – 1F1	2G1	
		3	3E1 – 6E1	3G1	
		4	4E1 – 5E1	4G1	
4	Semi-finals: best of three, 2 winners progress	1	1G1 – 4G1	1H1	1H2
		2	2G1 – 3G1	2H1	2H2
2	Bronze medal	1	1H2 – 2H2	Bronze	4th place
2	Final	1	1H1 – 2H1	Gold	Silver

Note: Q denotes qualifying position, A1, B1, C1, D1, E1, G1, H1 is the winner of the respective round, A2, B2, C2, D2, E2, G2, H2 is the loser of the corresponding round.

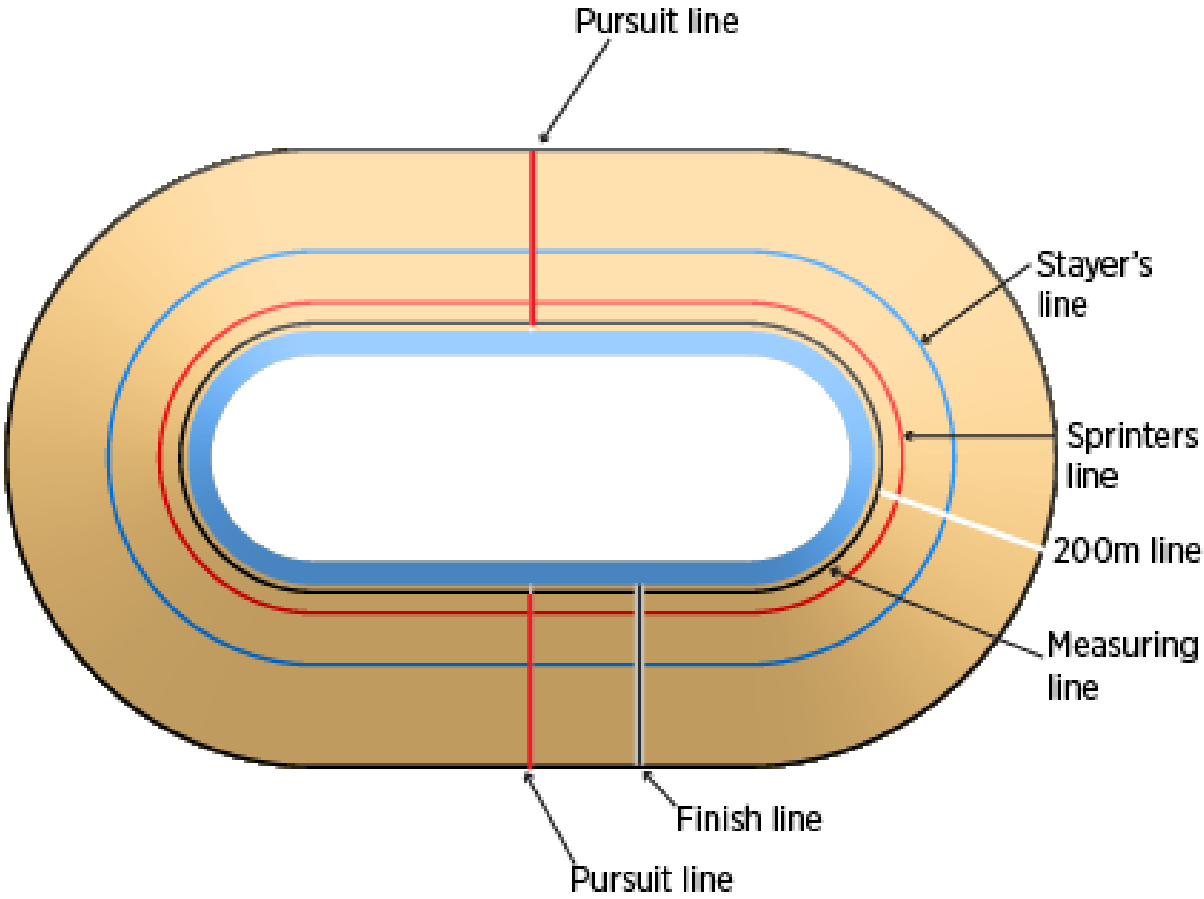

Figure 1.01 The terms given to the various lines that are painted on a cycling track. During the Match Sprint the start and finish lines are at the same point.

The recovery time between races also varies depending on the competition schedule. At the 2020 Tokyo Olympics, the Match Sprint competition took place over three days (Maia, 2021) and within each race session the riders competed between one and five times, with the average time between races being 48 ± 23 minutes. However, on 15 occasions there was less than 30 minutes between races, with the final deciding race for the gold medal being just over 15 minutes after the previous heat. At the 2016 Rio Olympics, the recovery period was on one occasion even shorter, ~ 10 minutes (Vieria, 2016). When the recovery period between races is brief, the current practice undertaken by elite track cyclists is to perform a light cycle on rollers (an easy to transport system allowing riders to cycle in a stationary position – see Figure 1.02), to have a brief period off the bike, and then to cycle lightly on rollers again (Elite Track Cyclist, personal communication, 12th September, 2019). For a sport that has been the focus of a great deal of scientific research, it is somewhat surprising that this recovery process is not evidence based (Elite Track Cyclist, personal communication, 12th September, 2019).

Figure 1.02 Track cyclists riding on rollers in the central section of the velodrome.

The aim of this research project was, therefore, to investigate sprint cycling performance, with consideration to the efforts required in the Match Sprint competition. The objective of Study 1 (Chapter 4) was to evaluate the time-course of performance recovery, as well as to assess physiological variables that could influence the recovery time-course. The aim of Study 2 (Chapter 5) was to explore the effectiveness of the recovery practice that is currently undertaken by riders between sprints (current practice vs passive rest). The effect of a change in second sprint duration was also explored, to assess whether this would alter the effort provided by the participants. Pre-testing data collection (Chapter 6) then sought to examine the effects of sprint duration on the performance loss effect size between sprints. The aim of the final study (Chapter 7) was to assess repeated-sprint performance during a simulated competition, where multiple motivational factors may be present. Alongside performance, physiological stress markers were also monitored to examine whether the participants were in a heightened state of sympathetic arousal.

Chapter 2: Literature Review

2.1. SPRINT CYCLING – PERFORMANCE AND PERFORMANCE RECOVERY

Track sprint cycling disciplines include: the Kilo (a 1 km time-trial undertaken from a stationary start); the Keirin (typically three laps behind a motorised bicycle, the derny, which will increase in speed every lap, followed by three laps of racing without the derny); the Team Sprint (a race where two opposing teams race over three laps of the track. Both teams begin from a stationary start, but are on opposite sides of the track. Each of the three riders must lead for one lap); and the Sprint, which is also known as the Match Sprint (a race typically between two riders over three laps of the track, with the aim being to cross the finish line first) (UCI, 2020). The duration of maximal effort in sprint cycling competitions has been estimated to be 15 – 60 s (Douglas et al., 2021; Ferguson et al., 2021). Therefore, within this thesis the term "sprint" will be used to reflect all maximal cycling efforts that are up to 60 s in duration. Whilst the Match Sprint will be the focus of this research thesis, data from the other sprint track cycling disciplines will also be considered.

Each Match Sprint race begins with the sound of a whistle. The rider that is on the lower side of the track must initially lead the race (UCI, 2020). The lead rider may then try to control the race tempo, whilst keeping a watchful eye on his/her opponent for any surprise attacks. In this scenario, the lead rider knows that the following rider will have to cycle a greater distance in order to overtake and win the race (Track Cycling Academy, 2024). However, air resistance is by far the greatest resistance that limits cycling performance (Faria, 1992). When one rider follows another, air resistance can be substantially reduced (Craig & Norton, 2001), meaning that a rider may choose to follow the lead rider or they may even attempt to strategically force their opponent to become the lead rider. The track itself can even affect the benefit of being the lead or following rider (Track Cycling Academy, 2024). For example, at the 2022 European Track Cycling Championships in Munich, the shorter track (200 m), which had been temporarily constructed, meant that the riders benefited greatly from being the lead rider.

"*It's still a three lap race this Match Sprint, so we're not going by distance, we're going by number of laps. So overall, it's over a lot quicker. We've (therefore) seen a lot less cat and mouse, a lot less cagey starts in this competition overall, compared to what we normally see on the longer tracks. We've even seen one rider go all-out from the start*" Joanna Rowsell (former British track cyclist and Eurosport commentator at the 2022 European Track Cycling Championships held in Munich).

At the 2020 Tokyo Olympics, a standard 250 m track was used. In the Match Sprint, the greatest time and variability in lap duration occurred during the first lap, with the least time and variability in lap duration occurring during the final lap (see Table 2.01). The effort provided for the qualifying event, the Flying 200, should, however, be more consistent. Spilt times from the

Flying 200 at the Tokyo Olympics (see Table 2.02) indicated that all but one of the riders was slower over the second 100 m, when compared to the first, which could be indicative of fatigue during the effort or it could be that the initial benefits created by moving from the high side of the track to the low side of the track were diminishing. On the first day of the competition (4th August 2020), in addition to the qualifying round the riders competed in the first and second knockout rounds, including the repechages for both rounds (see Table 2.03) (Maia, 2021). Therefore, the riders were required to perform between one and five maximal efforts over ~ 3.5-hour period. On six occasions, the recovery period between races was less than 30 minutes. On Day 2 (Thursday 5th August), the riders again raced up to five times over a three-hour period, and on seven occasions, less than 30 minutes separated the races. On the final day (Friday 6th August), the remaining four riders raced on four (Jack Carlin and Denis Dmitriev) or five (Jeffery Hoogland and Harrie Lavreysen) occasions and in the final gold medal deciding race, Jeffery Hoogland and Harrie Lavreysen had just over 15 minutes to recover from their previous heat. At the 2016 Rio Olympics, recovery periods under 30 minutes were also not uncommon. Of note, in round one, the New Zealand rider, Eddie Dawkins, lost in Heat 9 and then raced in the first of the repechages, meaning that he had ~ 10 minutes between these races (see Table 2.04) (Vieria, 2016).

Table 2.01 Race duration and lap times for Match Sprint races at the 2020 Tokyo Olympics. Data are displayed as mean ± standard deviation.

Event	Lap 1 Time (s)	Lap 2 Time (s)	Lap 3 Time (s)	Race Time
Two-rider race	45.5 ± 13.2	16.4 ± 2.0	12.5 ± 0.3	74.4 ± 14.1
Three-rider race	26.4 ± 3.2	14.6 ± 1.2	12.5 ± 0.2	53.4 ± 3.1
Four-rider race	23.2	13.4	12.4	48.9

Note: lap times were calculated for the rider that won each race.

Table 2.02 Split times (100 m and 200 m) during the Flying 200 at the 2020 Tokyo Olympics.

Rider Name	0-100 m	100-200 m
HOOGLAND Jeffrey	4.585	4.630
LAVREYSEN Harrie	4.574	4.641
CARLIN Jack	4.621	4.685
PAUL Nicholas	4.590	4.726
DMITRIEV Denis	4.670	4.661
TJON EN FA Jair	4.676	4.796
RUDYK Mateusz	4.702	4.791
KENNY Jason	4.733	4.777
WAKIMOTO Yuta	4.735	4.783
VIGIER Sebastien	4.741	4.810
XU Chao	4.753	4.831
WAMMES Nick	4.753	4.834
BOETTICHER Stefan	4.754	4.839
RAJKOWSKI Patryk	4.770	4.824
BARRETTE Hugo	4.758	4.838
QUINTERO CHAVARRO Kevin Santiago	4.761	4.865
AWANG Mohd Azizulhasni	4.747	4.879
WEBSTER Sam	4.773	4.858
LEVY Maximilian	4.768	4.878
HELAL Rayan	4.784	4.885
RICHARDSON Matthew	4.822	4.863
HART Nathan	4.800	4.896
SAHROM Muhammad Shah Firdaus	4.779	4.921
MITCHELL Ethan	4.798	4.907
YAKUSHEVSKIY Pavel	4.834	4.889
NITTA Yudai	4.819	4.909
SPIES Jean	4.866	4.921
BABEK Tomas	4.880	4.976
PONOMARYOV Sergey	4.914	5.018
BROWNE Kwesi	4.896	5.070
Mean ± Standard Deviation	4.755 ± 0.087	4.840 ± 0.102

Table 2.03 Competition schedule for the Match Sprint at the 2020 Tokyo Olympics (Maia, 2021).

Wednesday 4th August			Thursday 5th August			Friday 6th August		
Time	Race	Number of Riders	Time	Race	Number of Riders	Time	Race	Number of Riders
15.30-16.10	Qualifiers	30 riders	15.48-16.06	Round 3 (1/8) Heats 1-6	12 riders	16.10-16.16	Race 1 Semi Final	4 riders
16.35-17.11	Round 1 (1/32) Heats 1-12	24 riders	16.21-16.27	Repechages	6 riders	16.52-16.58	Race 2 Semi Final	4 riders
17.31-17.43	Repechages	12 riders	16.45-16.57	Race 1 Quarter Final	8 riders	1710-17.15	Race 3* Semi Final	4 riders
18.13-18.37	Round 2 (1/16) Heats 1-8	16 riders	1725-17.37	Race 2 Quarter Final	8 riders	18.00-18.06	Race 1 Medal Races	4 riders
18.47-18.59	Repechages	8 riders	17.21-17.25	Race 3* Quarter Final	8 riders	18.35-18.41	Race 2 Medal Races	4 riders
			18.27-18.30	5th-8th Place	4 riders	18.50-18.53	Race 3* Medal Races	4 riders

Note: * race only occurring when there was a tie.

Table 2.04 Competition schedule for the Match Sprint at the 2016 Rio Olympics.

Friday 12th August			Saturday 13th August			Sunday 14th August		
Time	Race	Number of Riders	Time	Race	Number of Riders	Time	Race	Number of Riders
16:14-16.50	Qualifiers	27 riders	10.23-10.41	Round 2 Heats 1-6	12 riders	17:04-17.08	Race 1 Final	2 riders
17.30-17.58	Round 1 Heats 1-9	18 riders	11.07-11.14	Repechages	6 riders	17.08-17.12	Race 1 Bronze/4th Place	2 riders
18.08-18.18	Repechages	9 riders	16.00-16.16	Race 1 Quarter Final	8 riders	17.42-17.46	Race 2 Final	2 riders
			16.18-16.22	9th-12th Place	4 riders	17.46-17.50	Race 2 Bronze/4th Place	2 riders
			16.34-16.50	Race 2 Quarter Final	8 riders	18.47-18.51	Race 3* Final	2 riders
			17.21-17.25	Race 3* Quarter Final	8 riders	18.47-18.51	Race 3* Bronze/4th Place	2 riders
			17.41-17.49	Race 1 Semi Final	4 riders			
			17.59-18.07	Race 2 Semi Final	4 riders			
			18.21-18.25	Race 3* Semi Final	4 riders			
			18.17-18.21	5th-8th Place	4 riders			

Note: * race only occurring when there was a tie.

2.1.1. Single Sprint Performance

When using a cycle ergometer, mechanical power output can be determined in several different ways (Douglas et al., 2021), but it is ultimately a measure of work over time (1 Watt = 1 Joule·s^{-1}). Peak power output (PPO) is the highest power output recorded during a sprint test. PPO is a major factor in sprint cycling performance (Bertucci et al., 2005). During a maximal effort cycling sprint PPO is likely to be achieved within three to five seconds (de Jong et al., 2015; Glaister et al., 2019; Wittekind et al., 2011). Power output during the remainder of the sprint will then decline, with the potential exception of some occasional fluctuations (see Figure 2.01 for an example 30 s sprint power output profile on a stationary cycle ergometer). Whilst the studies by de Jong et al. (2015), Glaister et al. (2019), and Wittekind et al. (2011) were all conducted on a stationary cycle ergometer, an example power output profile from a highly trained male sprint cyclist performing a stationary start 500 m time-trial can be seen in Figure 2.02 (Douglas et al., 2021) and a profile from a 16-year-old female sprint track cyclist racing in the Match Sprint can be seen in Figure 2.03 (Ferguson et al., 2021). The Match Sprint profile displays a sharper rise and fall in power output, but the general pattern of the response appears to be similar between all three profiles.

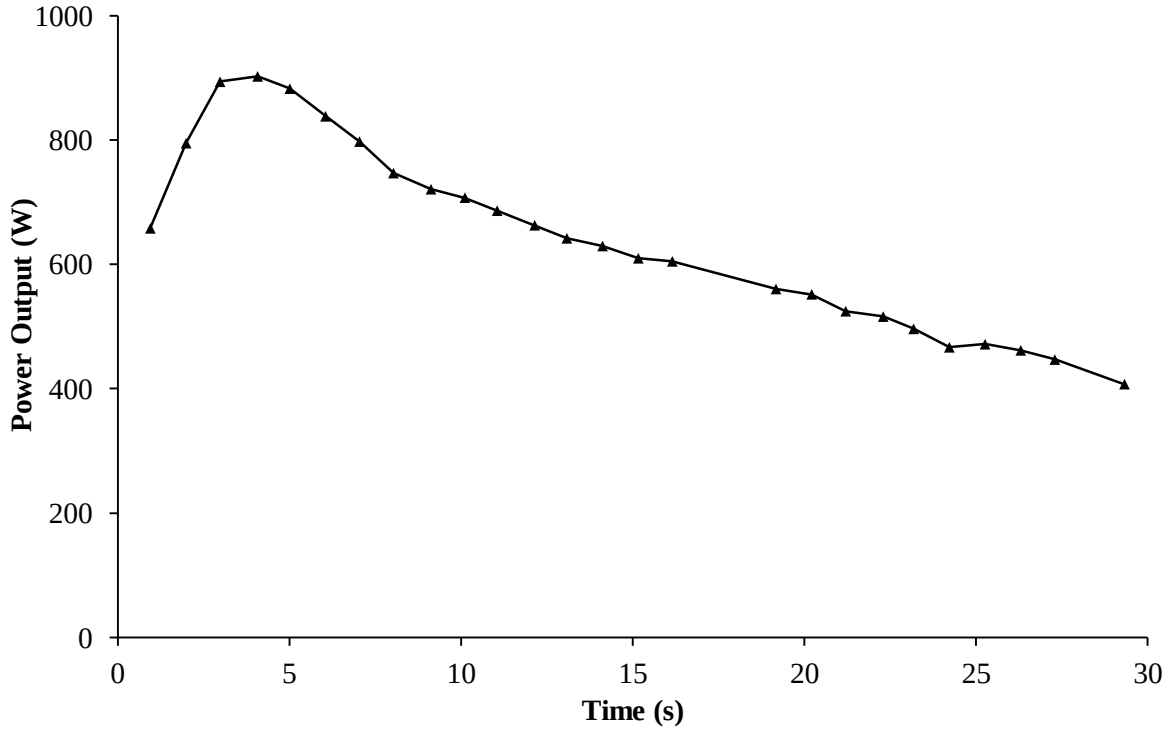

Figure 2.01 Power output during a 30 s maximal effort from a stationary start undertaken by healthy men (n = 9) on an indoor stationary cycle ergometer (Wittekind et al., 2011).

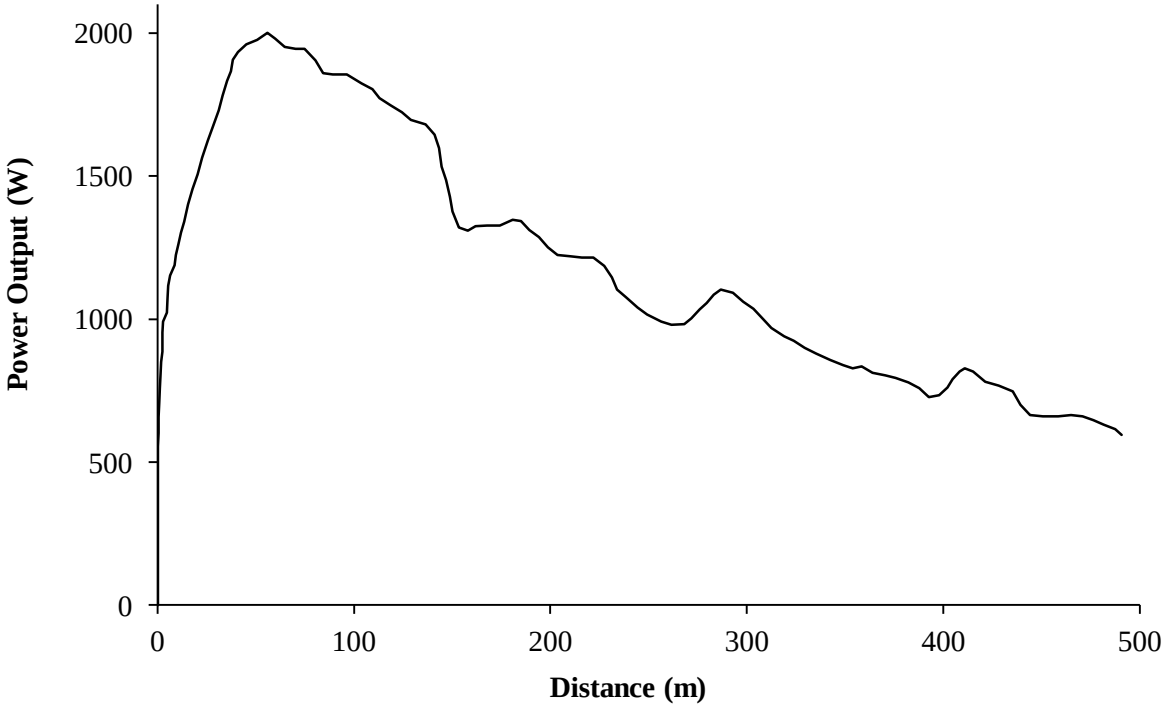

Figure 2.02 Power output during a 500 m maximal effort from a stationary start undertaken by a highly trained male sprint cyclist (Douglas et al., 2021).

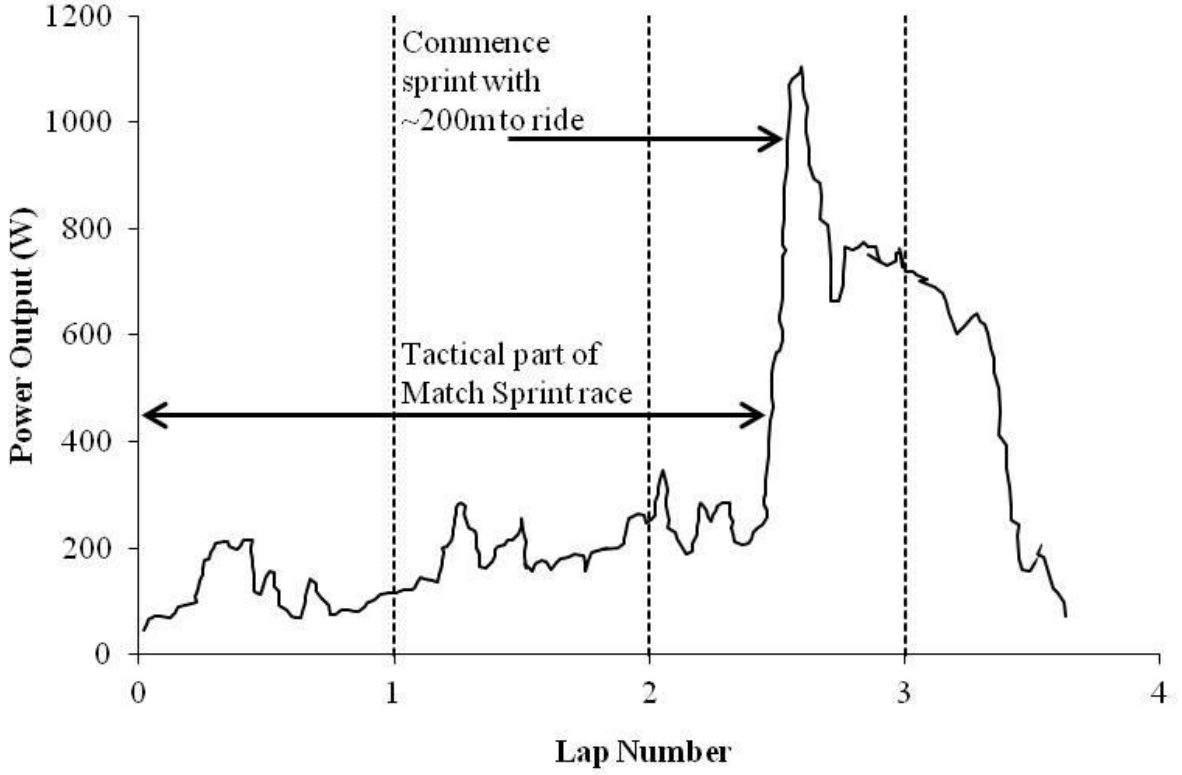

Figure 2.03 Power output during a three lap Match Sprint race undertaken by a 16 year-old female athlete (Ferguson et al., 2021).

2.1.2. Performance Recovery

The short-term recovery of sprint cycling performance has been investigated using brief recovery time-periods (1 s – 30 minutes) (Ainsworth et al., 1993; Bogdanis et al., 1995; Bogdanis et al., 1996b; Cherry et al., 1998; Esbjörnsson-Liljedahl et al., 2002; Glaister et al., 2014; Hebestreit et al., 1993; Kirkpatrick & Burrus, 2020; Zabala et al., 2008; Zabala et al., 2011). However, only the study by Glaister et al. (2014) assessed the time-course of performance recovery by modelling the kinetics of the response. Glaister et al. (2014) examined the recovery of PPO following a 30 s fatiguing protocol, by asking the participants to perform a brief 5 s maximal sprint at several recovery time-points ranging from 5 s to 160 s. Only one recovery time-point was assessed on any day and both one- and two-phase exponential functions were fit to the data. There was no clear evidence of a second order delay to the response, but a two-phase exponential function significantly improved the model fit (Glaister et al., 2014). The reported time constant (τ) and amplitude (A) for the group response (τ_0 = 16.6 s; τ_1 = 122.2 s; A_0 = 45.7%; A_1 = 54.2%) (see Figure 2.04), as well as the mean and standard deviation from the individual responses (τ_0 = 21.5 ± 13.8 s; τ_1 = 200.3 ± 130.3 s; A_0 = 53.7 ± 25.8%; A_1 = 47.1 ± 24.5%), indicated that there was an initial rapid phase to the recovery of PPO (parameters with a subscript 0), which was followed by a slower secondary phase (parameters

with a subscript 1). Considering the values that were reported for the model parameters, using 4 × τ (which provides a quick and simple way to estimate steady-state) (Jones & Poole, 2005), it could be estimated that PPO would be fully restored over ~ 9 – 15 minutes.

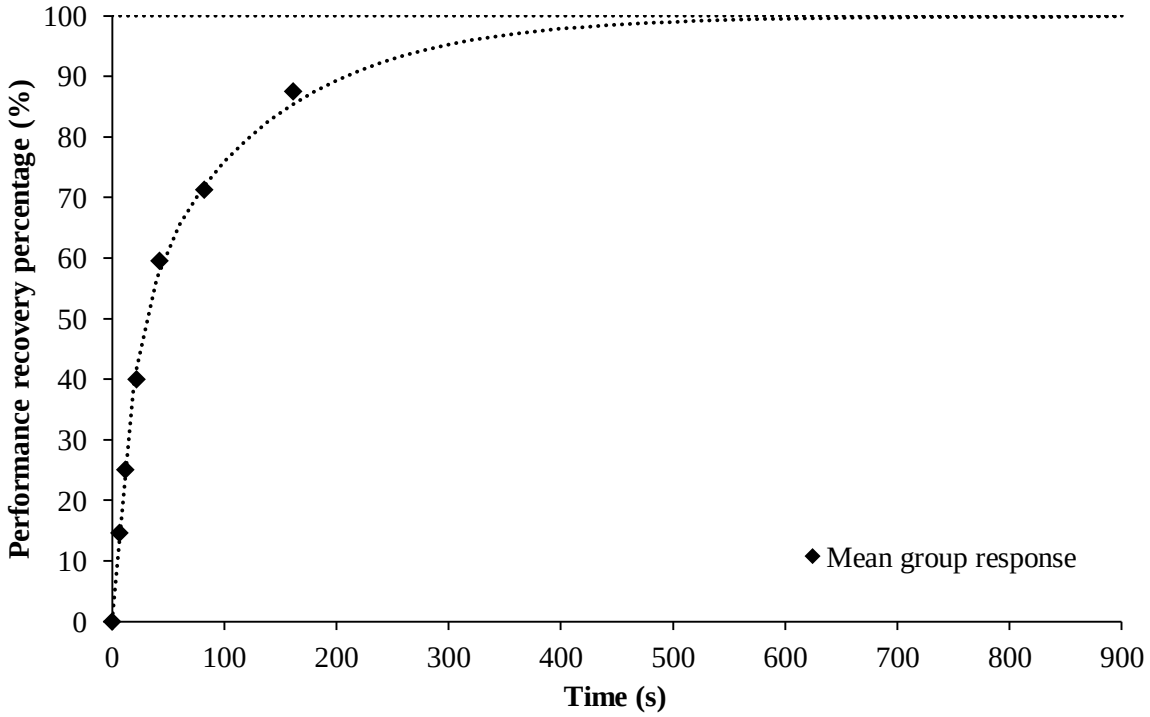

Figure 2.04 The recovery of peak power output following a 30 s sprint. Data represent the group mean response. A line of best fit (two-phase exponential) has been added (Glaister et al., 2014).

When the recovery period between Wingate anaerobic tests (WAnT) was six minutes or less, PPO has typically been found to be reduced in the second test (Bogdanis et al., 1996b; Bogdanis et al., 1995; Hebestreit et al., 1993). However, when 10 minutes or greater separated two sprints, PPO was fully restored (Hebestreit et al., 1993; Zabala et al., 2008). It should, however, be noted that Hebestreit et al. (1993) calculated PPO as the mean power output (MPO) over the first 6 s of the test. Whilst this calculation (or similar) was traditionally used for the WAnT (Driss & Vandewalle, 2013), modern day ergometers, such as the Lode Excalibur Sport (Groningen, The Netherlands), assess crank torque at a greater frequency, facilitating a more precise assessment of PPO (Driss & Vandewalle, 2013; Lanferdini et al., 2020). In the study by Hebestreit et al. (1993), PPO during the second sprint was ~ 97% of the value achieved during the first sprint. A 3% reduction in performance could be very meaningful at the elite level. The inability to detect a significant difference of this magnitude may have been the result of the small sample size (n = 8) limiting statistical power. In addition, Ferguson et al. (2021) stated that whilst PPO is a key metric for sprint cycling performance, it may not accurately reflect overall performance, especially when considering the repeated efforts that are

required during the Match Sprint competition. It is, therefore, possible that MPO or the total work (where the total work is simply the product of MPO and time) performed during the sprint may provide a better reflection of overall performance. Hebestreit et al. (1993) found that the total work performed in the second WAnT was significantly reduced after 10 minutes of recovery and Esbjörnsson-Liljedahl et al. (2002) found that MPO was significantly reduced in a third WAnT, when the tests were performed 20 minutes apart.

2.1.3. Summary

In summary, at the elite level the Match Sprint track cycling competition may occur over several days and the riders may have to compete on multiple occasions each day. The time between races varies and may be as little as 10 minutes. Power output is the performance metric that is most commonly used in sprint cycling research. During a sprint, PPO will usually occur after 3 – 5 s, with power output typically declining thereafter. MPO may, however, provide a better reflection of overall sprint performance. When the duration between sprints is similar to the shortest recovery periods that occur during a competition, performance restoration may not be complete.

2.2. SPRINT CYCLING – NEUROMUSCULAR ACTIVITY AND FATIGUE

Optimal sprint cycling performance requires the activation of multiple body segments across various joints via precise inter- and intra-muscular coordination (O'Bryan et al., 2014). Neuromuscular fatigue reflects an inability of skeletal muscles to produce force or power output (Collins et al., 2018). Whilst the activity levels of the *semimembranosus*, *biceps femoris*, *tibialis anterior*, *tensor fasciae latae*, and *gluteus maximus* will all fail to reach their maximum during a cycling sprint (Dorel et al., 2012), the reduction in power output after the initial peak, as well as the inability to repeatedly produce the same MPO and PPO, could be as a result of skeletal muscle fatigue. The cause of fatigue during cycling sprints may either relate to peripheral changes within the muscle or be because the central nervous system fails to sufficiently activate the required motor neurons (Gandevia, 2001). Collins et al. (2018) stated that the rate of performance decline during a repeated-sprint task would be greater during the early sprints (sprints 1 – 5) than the later sprints (sprints 5 – 10) and that fatigue in the early sprints was likely to be from a peripheral origin, whereas during the later sprints fatigue would consist of both central and peripheral components. That being said, one limitation when studying neuromuscular fatigue following a dynamic task, such as a cycling sprint, is that fatigue is generally evaluated during the performance of an isometric task (Collins et al., 2018). An isometric contraction lacks task specificity and there may be a delay between the

performance of the activity and the performance of the isometric contraction. Nonetheless, an understanding of the physiological processes that are required for muscular contractions to occur, as well as any factors that could inhibit optimal contractile function, may be insightful for performance and for performance recovery in the Match Sprint.

2.2.1. Skeletal Muscle Physiology

From a structural perspective (see Figure 2.05), muscles are surrounded by a layer of connective tissue that is termed the epimysium. Another layer of connective tissue, the perimysium, then surrounds bundles of muscle fibres, which are also known as fascicles. Finally, individual muscle fibres are surrounded by the endomysium, as well as a cell membrane, known as the sarcolemma (Frontera & Ochala, 2015). Cylindrical myofibrils, which contain myofilaments, are found within each muscle fibre. The two most abundant myofilaments are actin and myosin, constituting ~ 70 – 80% of the protein content of a single fibre (Frontera & Ochala, 2015). It is the orderly nature of these myofilaments that give muscle fibres their striated appearance.

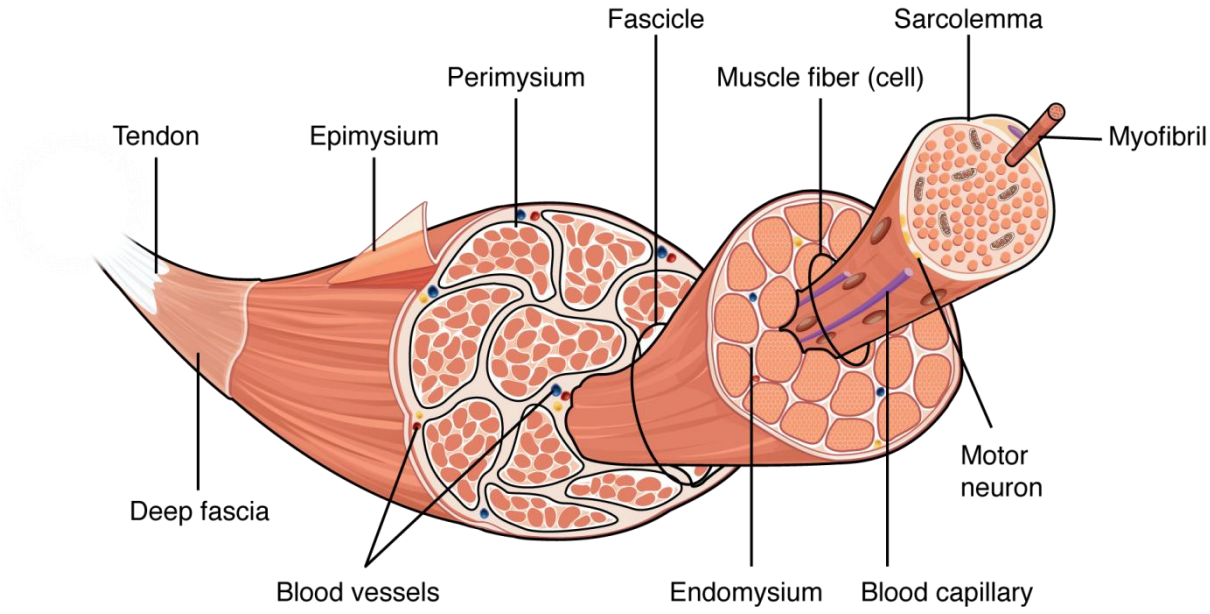

Figure 2.05 Connective tissue layers within a skeletal muscle (Biga et al., 2020).

The functional unit of a muscle fibre is the sarcomere. A Z-line or Z-disk, where the Z denotes the German word for between, ‘zwischen’, is found at both ends (see Figure 2.06). The support protein, titin, anchors the thick filament, myosin, longitudinally to the Z-disk (McCuller et al.,

2020). F-actin, or the thin filament, is a double helical structure, consisting of linked monomeric units of G-actin (McCuller et al., 2020). Tropomyosin runs along actin and is bound at every 7th actin monomer by troponin-T. Troponin also contains troponin-C and troponin-I. Troponin-I inhibits the binding of actin and myosin by blocking the actin binding sites. Therefore, for a muscular contraction to occur there must be a shift in tropomyosin to expose the actin binding sites. The binding of calcium to troponin-C causes this shift (McCuller et al., 2020).

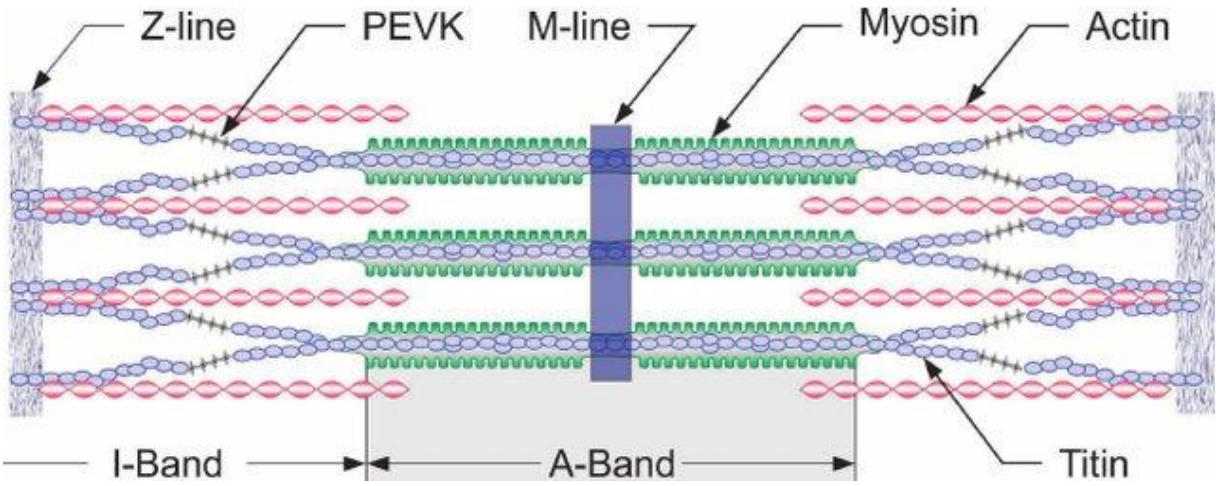

Figure 2.06 Schematic drawing of a sarcomere. Note: Z-lines are at the ends of the sarcomere and the M-line is in the middle. The A-Band and I-Band represent the dark and light portions. The proline, glutamate, valine and lysine-rich (PEVK) region of titin constitutes an entropic spring that provides passive tension to striated muscle (Herzog et al., 2014).

For the required calcium to be released into the muscle cell, an action potential is first initiated in the motor cortex of the brain (Allen et al., 2008). An upper motor neuron then transmits the excitatory signal from the cortex (Zayia & Tadi, 2023). Upper and lower motor neurons form a two-neuron circuit (Zayia & Tadi, 2023). Excitation of the lower motor neuron in the spinal cord then leads to an action potential being carried along an axon via neural synapses until the axon terminal is reached (Allen et al., 2008). The action potential stimulates the opening of calcium channels on the synaptic bulb and the influx of calcium into the bulb, causes exocytosis of the neurotransmitter, acetylcholine, from the synaptic vesicles (Sam & Bordoni, 2023). Acetylcholine is then released into the pre-synaptic cleft before binding with receptors on the post-synaptic membrane, resulting in the opening of voltage-gated channels (Greig & Jones, 2016). Positive ions are then drawn into the muscle cell producing a graded potential. If the voltage reaches the threshold potential, an action potential is generated. This action potential travels along the muscle membrane (McCuller et al., 2020). Invaginations of the sarcolemma form the T-tubule system, providing a route for the signal to travel into the interior of the muscle cell (Jayasinghe et al., 2013). The sarcoplasmic reticulum is a

complex bag-like structure found on either side of the T-tubule (Greig & Jones, 2016). Dihydropyridine receptors are located on the T-tubule and ryanodine receptors are found on the membrane of the sarcoplasmic reticulum, although there are only half as many dihydropyridine receptors as there are ryanodine receptors (see Figure 2.07). Therefore, only every other ryanodine receptor can be matched to a dihydropyridine receptor (Greig & Jones, 2016). When the T-tubule is depolarised, the dihydropyridine receptors act on the paired ryanodine receptors, allowing calcium to move out of the sarcoplasmic reticulum. Some of this calcium will then bind to the closed ryanodine receptors, opening these channels and facilitating the movement of more calcium into the muscle cell, where it can bind with troponin-C (Greig & Jones, 2016). After excitation, calcium is released from troponin-C and is actively pumped back into the sarcoplasmic reticulum (Frontera & Ochala, 2015). The calcium ATPase pump (SERCA) is one of three major proteins that contribute to the adenosine triphosphate (ATP) requirements of a muscular contraction (MacIntosh et al., 2012). The other proteins are the sodium-potassium ATPase pump, which pumps sodium out of the cell and potassium into the cell, and myosin ATPase, which is required during the cross-bridge cycle (MacIntosh et al., 2012). During the cross-bridge cycle, ATP is required to release the myosin head from actin (the transition from positions a to b in Figure 2.08) (Fitts, 2008). The hydrolysis of ATP is facilitated by myosin ATPase, although adenosine diphosphate (ADP) and an inorganic phosphate (Pi) remain bound to myosin at this point in time (Frontera & Ochala, 2015). The myosin head moves from a weakly bound state to a strongly bound state, before releasing Pi and undergoing the power stroke (transition from stages c to e in Figure 2.08) (Fitts, 2008). ADP is then released from myosin, completing the cycle and returning the coupling to the rigour state (Fitts, 2008). In the absence of ATP, the myosin head would remain strongly bound to actin. ATP is, therefore, essential for muscular contractions to occur. However, intramuscular stores are limited (Maughan & Gleeson, 2004).

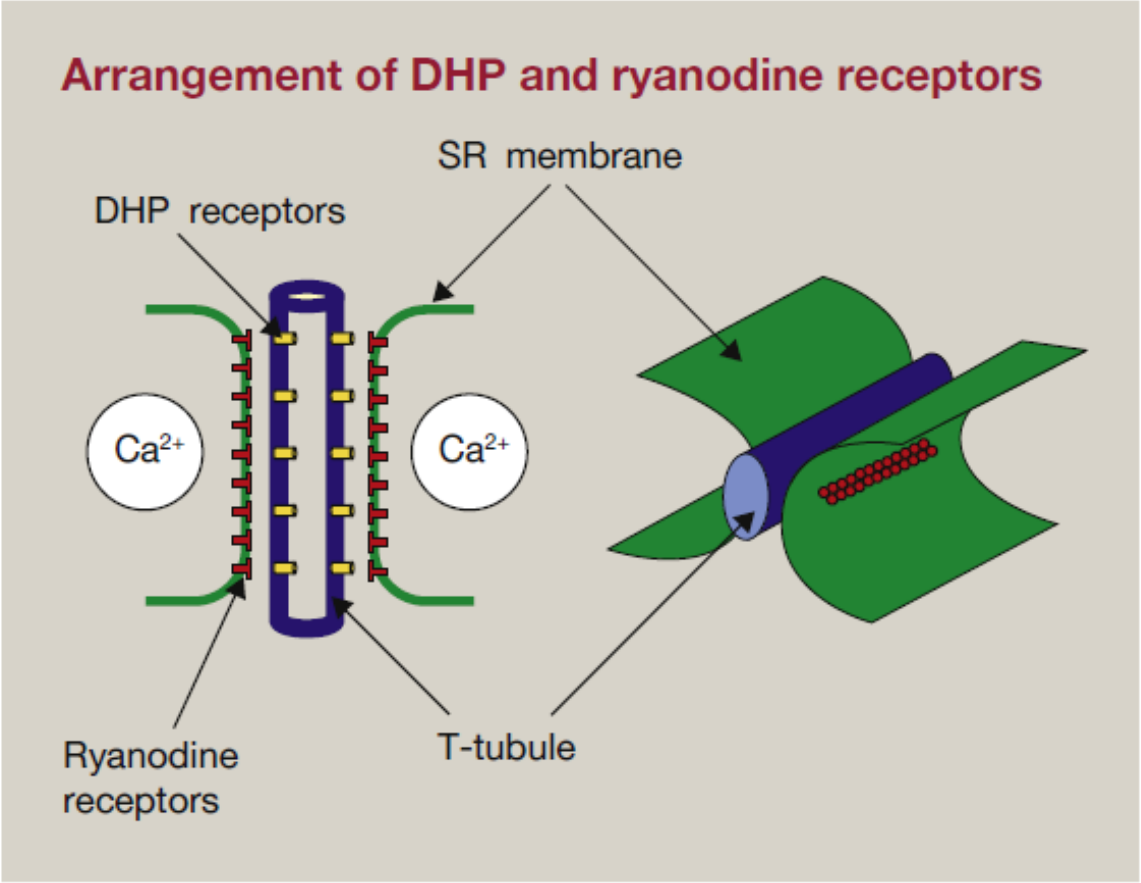

Figure 2.07 The T-tubule system, the sarcoplasmic reticulum (SR) and the dihydropyridine (DHP) and ryanodine receptors. Note: Ca^{2+} denotes calcium (Greig & Jones, 2016).

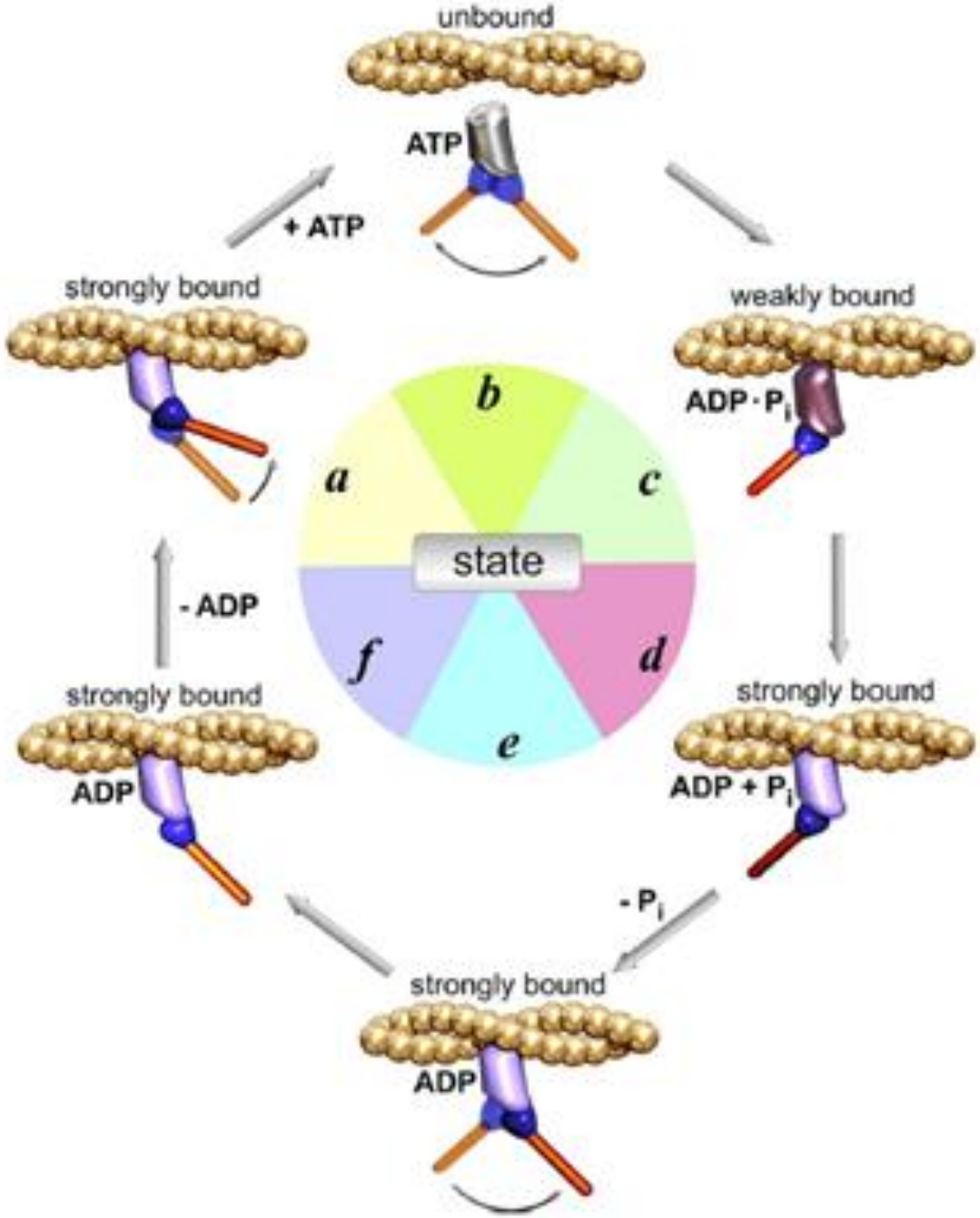

Figure 2.08 The cross-bridge cycle. Note: Letters a-f depict stages in the cycle. ATP denotes adenosine triphosphate, ADP adenosine diphosphate, and Pi inorganic phosphate (Fitts, 2008).

2.2.2. Energetics

ATP consists of adenine and ribose (adenosine), linked to three phosphates (see Figure 2.09). The bonds between the two outer phosphate groups are high-energy bonds, which can release a considerable amount of energy (McArdle et al., 2007).

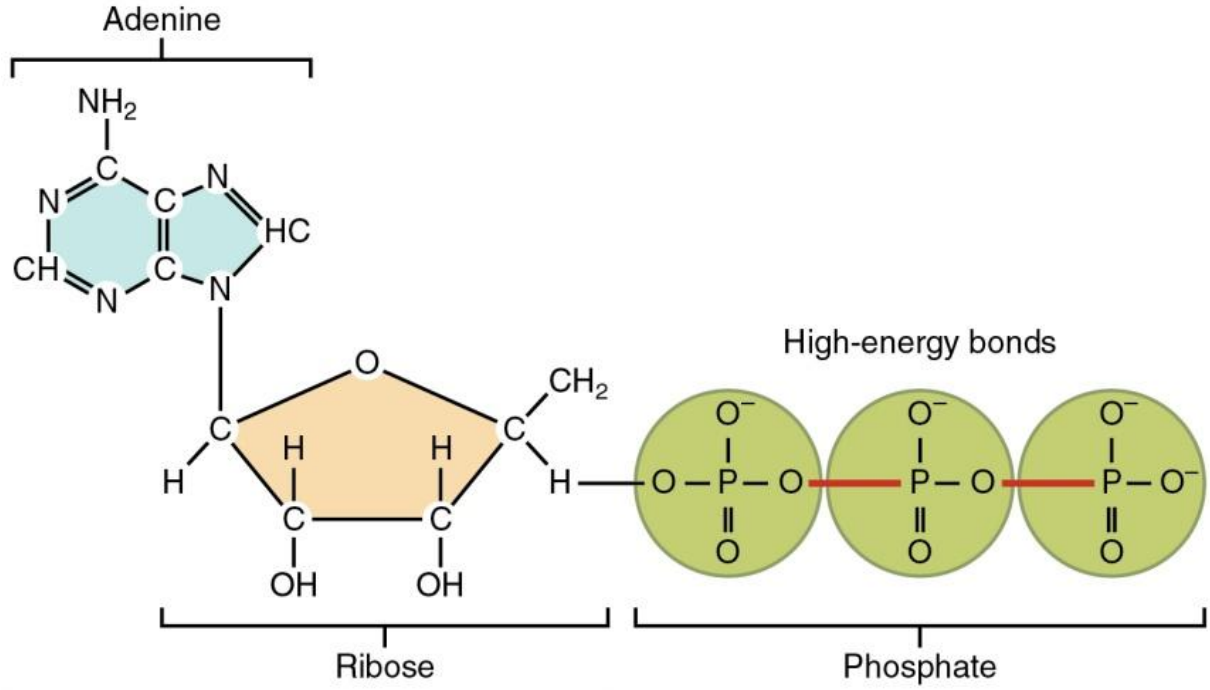

Figure 2.09 Composition of adenosine triphosphate (Rodewell et al., 2015). Note: C denotes carbon, H is hydrogen, N is nitrogen, O is oxygen, and P is phosphate.

2.2.2.1. Stored Adenosine Triphosphate

Muscle biopsies indicate that at rest, ATP concentration in the *vastus lateralis* is ~ 23.9 – 27.0 mmol·kg dm^{-1} (Bogdanis et al., 1995; Bogdanis et al., 1996b; Bogdanis et al., 1998; Casey et al., 1996; Gaitanos et al., 1993; Zhao et al., 2000), which is a relatively small store, sufficient to fuel just 1 – 2 s of maximal work (Glaister et al., 2005; Hargreaves & Spriet, 2020). Following a single cycling sprint (6 – 30 s), ATP concentration has been reported to range from 16.7 to 20.2 mmol·kg dm^{-1} (Bogdanis et al., 1995; Bogdanis et al., 1996b; Bogdanis et al., 1998; Casey et al., 1996; Gaitanos et al., 1993; Zhao et al., 2000), suggesting that ATP concentration will decrease, but that stores will not be entirely depleted during a sprint. In fact, the total degradation of ATP is prevented by innate regulatory mechanisms (Gastin, 2001). Following five minutes of recovery after a 30 s cycling sprint, Zhao et al. (2000) found that ATP concentration had increased significantly from the post-sprint value. After another five minutes of recovery, ATP concentration increased further, albeit the

concentration still remained lower than the resting level (see Table 2.05) (Zhao et al., 2000). However, in contrast to the restoration of the ATP stores reported by Zhao et al. (2000), Bogdanis et al. (1996b) found that 3.8 minutes after a 30 s cycling sprint, ATP concentration remained at the post-sprint level. Following a second sprint of either 10 s or 30 s, irrespective of the second sprint duration, no further reductions in stored ATP occurred (see Table 2.06) (Bogdanis et al., 1996b). Therefore, ATP will decrease during a sprint and the complete replenishment will likely take longer than 10 minutes.

Table 2.05 Adenosine triphosphate concentration in the *vastus lateralis* at rest, following a 30 s sprint, and after five and ten minutes of recovery (Zhao et al., 2000). Data are displayed as mean ± standard deviation.

	Rest	Post Sprint	Recovery 5 minutes	Recovery 10 minutes
ATP ($mmol \cdot kg\ dm^{-1}$)	25.9 ± 1.4	16.7 ± 1.8*	19.7 ± 2.1*†	23.4 ± 1.8*†‡

Note: * denotes a significant difference ($p < 0.05$) from rest, † a significant difference ($p < 0.05$) from post-sprint, and ‡ a significant difference ($p < 0.05$) from recovery 5 minutes. ATP denotes adenosine triphosphate and dm is dry muscle.

Table 2.06 Adenosine triphosphate concentration in the *vastus lateralis* at rest, following a 30 s sprint, after 3.8 minutes of recovery (pre sprint 2), and after a second sprint of 10 s or 30 s (Bogdanis et al., 1996b). Data are displayed as mean ± standard deviation.

	Rest	Post Sprint 1	Pre Sprint 2	Post Sprint 2 (10 s)	Post Sprint 2 (30 s)
ATP ($mmol \cdot kg\ dm^{-1}$)	27.0 ± 1.1	19.6 ± 1.3*	22.2 ± 1.4*	19.7 ± 1.8*	20.5 ± 1.7*

Note: * denotes a significant difference ($p < 0.01$) from rest. ATP denotes adenosine triphosphate and dm is dry muscle.

Supplementation of orally administered ATP has been investigated as a means of enhancing exercise performance (Freitas et al., 2019; Jordan et al., 2004). In a recent systematic review, González-Marenco et al. (2024) stated that maximal strength was enhanced with supplementation, but PPO during a cycling sprint was not improved nor were intramuscular ATP stores. It was, however, not clear how the physiological mechanisms that were proposed to explain the ergogenic properties of this supplement, namely a vasodilatory effect resulting in an enhancement of blood flow and a stimulation of calcium release from the sarcoplasmic reticulum, would improve maximal strength performance, but not PPO during a sprint. The ergogenic properties of ATP supplementation and the mechanisms of action may, therefore, require further investigation. Nonetheless, cycling sprints in the Match Sprint competition will require maximal efforts that are substantially longer than a few seconds, meaning that ATP resynthesis must occur to meet the requirements of the task. Fortunately, well-regulated pathways exist to enable its regeneration, with both the phosphagen and glycolytic systems not requiring oxygen (O_2) (Gastin, 2001).

2.2.2.2. The Phosphagen System

Three reactions occur within the phosphagen system (see Equations 2.01 – 2.03) (Baker et al., 2010). Whilst the creatine kinase (Equation 2.01) and adenylate kinase (Equation 2.02) reactions result in the production of ATP, the creatine kinase reaction has a substantially greater capacity (Baker et al., 2010). Resting phosphocreatine (PCr) concentration in the *vastus lateralis* has been reported to be 75.2 – 83.9 mmol·kg dm^{-1} (Bogdanis et al., 1995; Bogdanis et al., 1996b; Bogdanis et al., 1998; Casey et al., 1996; Gaitanos et al., 1993). The maximal rate of ATP regeneration via the phosphagen system has been estimated to be ~ 9 mmol·kg dm^{-1}·s^{-1} (Maughan & Gleeson, 2004). PCr breakdown for the formation of ATP during maximal work does not, however, occur in a linear fashion. The pattern of breakdown is, in fact, one of exponential decay (see Figure 2.10) (Glaister, 2005; Walter et al., 1997). Following 9 s of repeated rapid plantar flexion, PCr concentration was 38.6% ± 2.4% of its resting value, decreasing to 8.41% ± 0.91% after 30 s, with no further reduction when the task was performed for 60 s (Walter et al., 1997). From a sprint cycling perspective, after a 6 s sprint, Gaitanos et al. (1993) reported a muscle PCr concentration (*vastus lateralis*) of 32.9 mmol·kg dm^{-1}, a 57% reduction from the resting value and after 10 s and 20 s cycling sprints, Bogdanis et al. (1998) reported 55% and 73% decreases in PCr concentration. After a 30 s sprint, reductions of 59% (Casey et al., 1996), 67% (Zhao et al., 2000), 80% (Bogdanis et al., 1995), and 83% (Bogdanis et al., 1996b), have been reported. When two sprints were undertaken four minutes apart, Bogdanis et al. (1996b) reported that a significant restoration of PCr had occurred prior to the second sprint, albeit PCr concentration was still lower than the resting level. Following the second sprint, PCr concentration was only reduced to the same level as it had been after the first sprint, despite a lower start-point (see Table 2.07). Individuals that resynthesized PCr faster were also found to have performed better during the second sprint (Bogdanis et al., 1996b).

$$PCr + ADP + H^+ \xleftrightarrow{creatine\ kinase} ATP + Cr \qquad \text{(Equation 2.01)}$$

$$ADP + ADP \xleftrightarrow{adenylate\ kinase} ATP + AMP \qquad \text{(Equation 2.02)}$$

$$AMP + H^+ \xrightarrow{AMP\ deaminase} IMP + NH_4^+ \qquad \text{(Equation 2.03)}$$

where AMP is adenosine monophosphate, ADP is adenosine diphosphate, ATP is adenosine triphosphate, Cr is creatine, H is hydrogen, IMP is inosine monophosphate, and PCr is phosphocreatine.

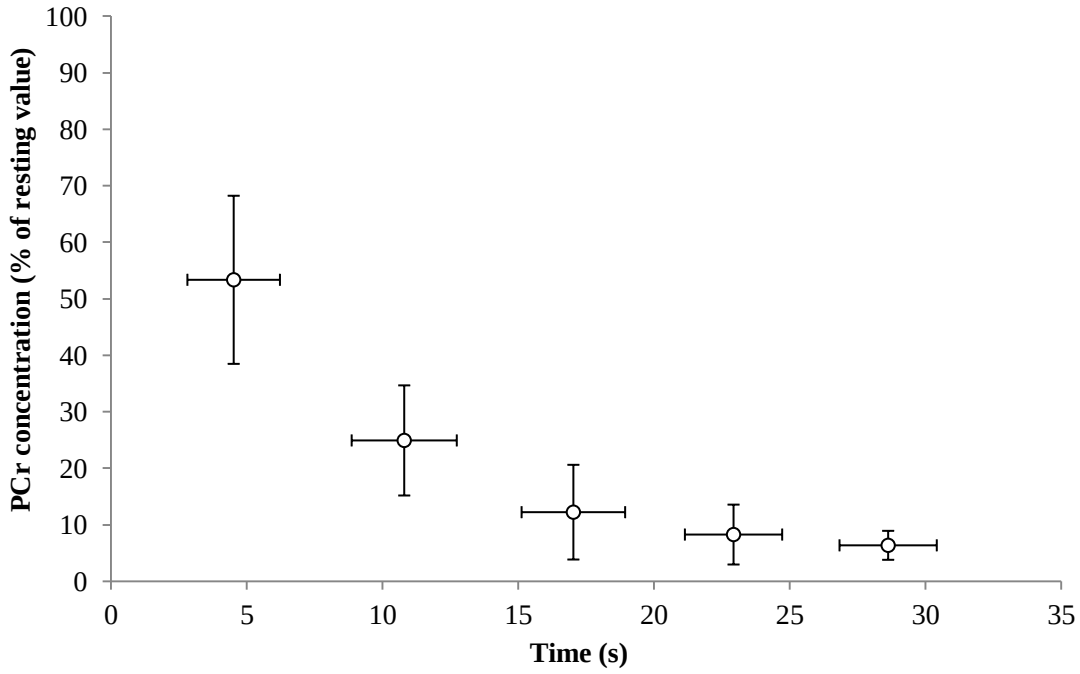

Figure 2.10 Phosphocreatine (PCr) concentration assessed at the medial *gastrocnemius* using localised nuclear magnetic resonance imaging during 30 seconds of repeated maximal plantar flexion. Open circles represent PCr concentration as a percentage of the resting values and error bars represent the standard deviation of measurement time (horizontal) and concentration (vertical) (Glaister, 2005; Walter et al., 1997).

Table 2.07 Phosphocreatine concentration in the *vastus lateralis* at rest, following a 30 s sprint, after 3.8 minutes of recovery (pre sprint 2), and after a second sprint of 10 s or 30 s (Bogdanis et al., 1996b). Data are displayed as mean ± standard deviation.

	Rest	Post Sprint 1	Pre Sprint 2	Post Sprint 2 (10 s)	Post Sprint 2 (30 s)
Phosphocreatine ($mmol \cdot kg\ dm^{-1}$)	75.2 ± 6.2	12.6 ± 1.7*	58.5 ± 3.3*†	15.3 ± 2.0*‡	8.8 ± 3.5*‡

Note: dm denotes dry muscle. * denotes a significant ($p < 0.01$) difference from rest, † a significant difference ($p < 0.01$) compared to post sprint 1, and ‡ a significant difference ($p < 0.01$) compared to pre sprint 2.

The importance of PCr for short duration high-intensity exercise means that creatine monohydrate is one of the most commonly used ergogenic aids (Hall et al., 2021). The rationale for supplementation is: 1) to enhance PCr stores in the muscle; 2) to facilitate a faster regeneration of PCr in recovery; and 3) to buffer hydrogen ion (H^+) accumulation (Hall et al., 2021; Lemon, 2002). In a recent meta-analysis, Glaister and Rhodes (2022) concluded that creatine supplementation did have a positive effect on repeated-sprint cycling performance, evidenced by an increase in MPO, although changes in PPO and fatigue index were not found. The inclusion criteria (5 – 20 sprints, sprint duration ≤ 10 s, recovery time ≤ 90 s) would not, however, reflect conditions in the Match Sprint. That being said, even the recovery period used by Bogdanis et al. (1996b) (four minutes) would be too

short to represent conditions in the Match Sprint. Following 10 minutes of recovery after a 30 s cycling sprint, Zhao et al. (2000) reported that PCr concentration was actually greater than the resting value (see Table 2.08); although the authors acknowledged that the restoration rate was faster than others have reported. The time-course of PCr resynthesis has been modelled using a one-phase exponential function (Bogdanis et al., 1995; Yoshida, 2002; Yoshida et al., 2013), with Bogdanis et al. (1995) suggesting that 13.6 minutes after a 30 s sprint, 95% of the total resynthesis would be complete, but the range of values reported (3.9 – 25 minutes) varied substantially. It has also been suggested that the restoration process may be biphasic (Harris et al., 1976), with the secondary phase being dependent on muscle pH (McMahon & Jenkins, 2002; Sahlin et al., 1979; Walter et al., 1997).

Table 2.08 Phosphocreatine concentration in the *vastus lateralis* at rest, following a 30 s sprint, and after five and ten minutes of recovery (Zhao et al., 2000). Data are displayed as mean ± standard deviation.

	Rest	Post Sprint	Recovery 5 Minutes	Recovery 10 Minutes
Phosphocreatine ($mmol \cdot kg\ dm^{-1}$)	84.3 ± 3.5	28.0 ± 1.7*	80.0 ± 1.0†	89.5 ± 2.8*†‡

Note: dm denotes dry muscle. * denotes a significant difference ($p < 0.05$) from rest, † a significant difference ($p < 0.05$) from post-sprint, and ‡ a significant difference ($p < 0.05$) from recovery 5 minutes.

The pH scale provides a measure of the acidity/alkalinity of a solution, specifically this relates to the H^+ concentration (McArdle et al., 2007). Equations 2.01 and 2.03 indicated that H^+ are, in fact, consumed, not produced, within the phosphagen system, although Baker et al. (2010) stated that the H^+ that is used in the AMP deaminase reaction is of limited importance, whereas the H^+ that is consumed during the creatine kinase reaction is responsible for the slight alkalisation that occurs at the start of exercise. A common misconception about energy systems is, however, that they respond in a sequential manner (Gastin, 2001). Activation of the glycolytic system is almost immediate after the start of exercise (Baker et al., 2010; Jones et al., 1985). The production of AMP via the adenylate kinase reaction (Equation 2.02), as well as the increasing concentration of free calcium and Pi, all stimulate enzymes within the glycolytic pathway (Baker et al., 2010).

2.2.2.3. The Glycolytic System

The capacity of the glycolytic system is much greater than the phosphagen system (Maughan & Gleeson, 2004). At rest, glycogen stores in the *vastus lateralis* were reported to be 327.5 ± 14.3 $mmol \cdot kg\ dm^{-1}$ (Bogdanis et al., 1996b). Following a 30 s cycling sprint, glycogen levels were reduced to 228.3 ± 18.2 $mmol \cdot kg\ dm^{-1}$, a level that remained unchanged throughout the four-

minute recovery period. During a second 30 s sprint, there was a further decrease in muscle glycogen, although the reduction was just 45% of the decrease that was recorded during the first sprint (see Table 2.09). The authors suggested that this may have been as a result of a pH induced inhibition of key glycolytic enzymes (Bogdanis et al., 1996b) (discussed further in section 2.2.2.7.). The glycolytic pathway consists of a series of reactions that can be divided into two phases (see Figure 2.11). The first phase has been termed the investment phase, as two ATP molecules are consumed during this phase (only one ATP molecule is used when glycogen, as opposed to glucose, is broken down). During the second phase, the payoff phase, four ATP molecules are produced. Glycolysis, therefore, results in a net gain of two/three molecules of ATP, although two molecules of reduced nicotinamide adenine dinucleotide (NADH) and two molecules of pyruvate are also produced. Whilst the aerobic pathway will be considered in detail in due course, if ATP regeneration by the aerobic pathway is inadequate, an increase in the concentration of NADH occurs in the mitochondria. In turn, both the tricarboxylic acid cycle (TCA) and the mitochondrial NADH shuttle become inhibited, leading to a build-up of NADH and a decrease in NAD^+ in the cytosol of the cell (Gladden, 2011). As NAD^+ is required for glycolysis, for the process to continue, the cytolytic enzyme, lactate dehydrogenase, facilitates the regeneration of NAD^+ (Melkonian & Schury, 2022), but results in the production of "lactic acid" (see Figure 2.12). For many years, it was suggested that this production of lactic acid was a major determinant of fatigue, a concept that has regularly been re-enforced by athletes, journalists, and commentators (Brooks, 2001; Cairns, 2006).

Table 2.09 Glycogen concentration in the *vastus lateralis* at rest, following a 30 s sprint, after 3.8 minutes of recovery (pre sprint 2), and after a second sprint of 10 s or 30 s (Bogdanis et al., 1996b). Data are displayed as mean ± standard deviation.

	Rest	Post Sprint 1	Pre Sprint 2	Post Sprint 2 (10 s)	Post Sprint 2 (30 s)
Glycogen ($mmol \cdot kg\ dm^{-1}$)	327.5 ± 20.2	228.3 ± 25.7*	240.5 ± 36.1*	223.5 ± 35.4*	183.5 ± 24.2*⁑

Units are $mmol \cdot kg$ dry $muscle^{-1}$. * denotes a significant ($p < 0.01$) difference from rest, and ⁑ a significant ($p < 0.05$) difference compared to pre sprint 2.

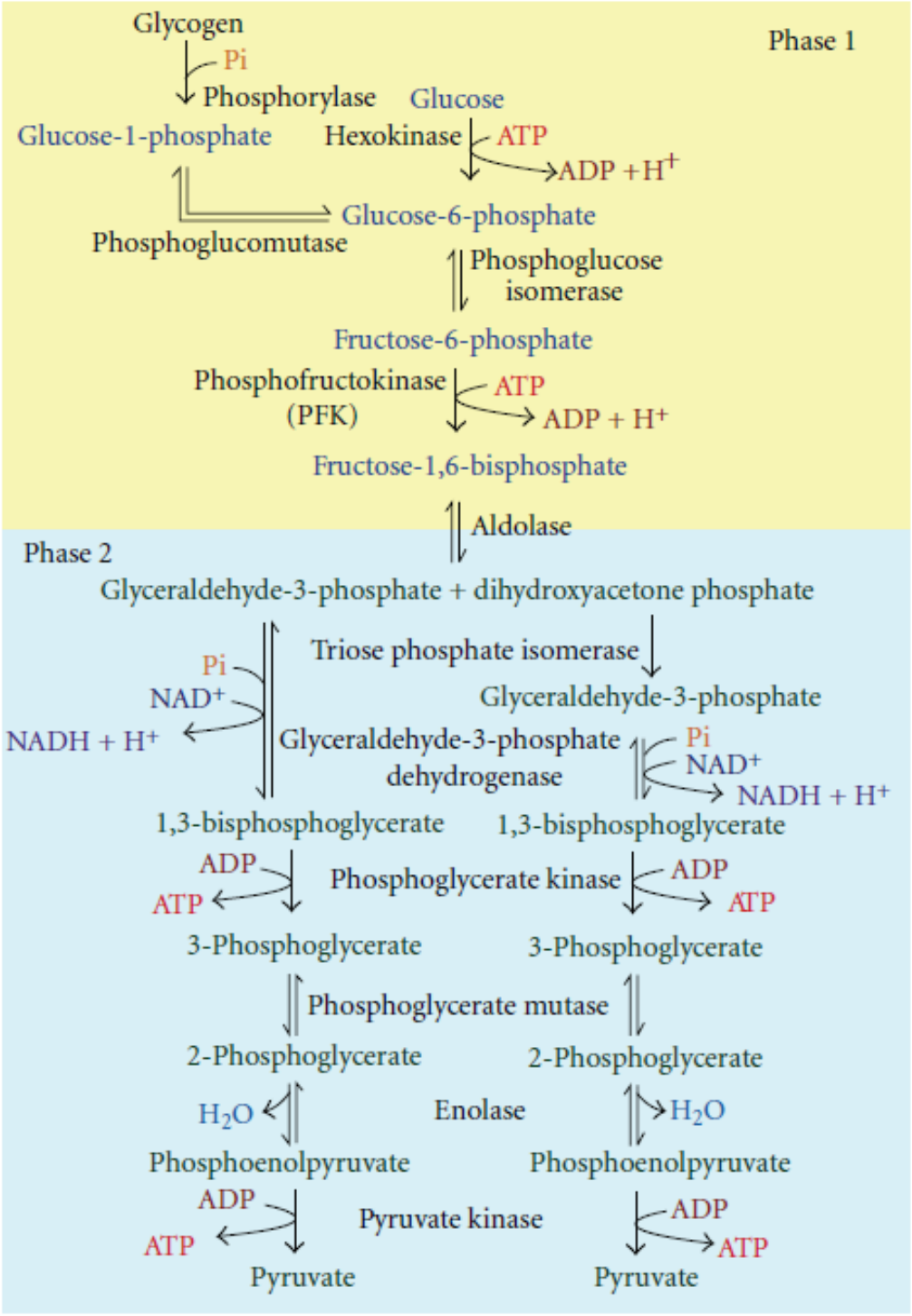

Figure 2.11 The glycolytic pathway. Note: Pi denotes inorganic phosphate, ATP adenosine triphosphate, ADP adenosine diphosphate, H^+ a proton, NAD^+ nicotinamide dinucleotide, and H_2O is water (Baker et al., 2010).

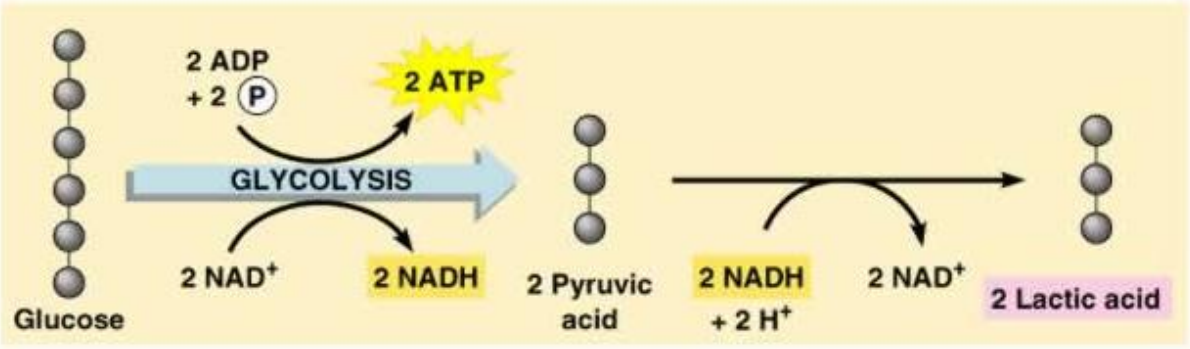

Figure 2.12 "Lactic acid" formation following the breakdown of glucose to pyruvic acid. Note: ATP denotes adenosine triphosphate, ADP adenosine diphosphate, H^+ a proton, and NAD^+ is nicotinamide dinucleotide.

2.2.2.4. Lactic Acid

In 1780, the Swedish chemist, Karl Wilhelm Scheele, first described the presence of an acid in sour milk that was termed lactic acid (Kompanje et al., 2007), although when the pH of muscle cells (~ range 6.2 – 7.0) is considered, if lactic acid is formed in the human body, it would immediately be ionized releasing an H^+ and leaving the acid salt, lactate (Robergs et al., 2004) (see Figure 2.13). Robergs et al. (2004), however, stated that this explanation of muscular acidosis is simply not supported by the biochemical reactions that occur, suggesting that a net release of two protons is simply the end result of glycolysis (see Table 2.10). Robergs et al. (2004) also suggested that if pyruvate is converted to lactate, this reaction will consume an H^+ (see Figure 2.14), meaning that rather than creating acidosis, lactate production from pyruvate will instead buffer it (Robergs et al., 2004). Unsurprisingly, the suggestions by Robergs et al. (2004) generated a great deal of debate (Gladden, 2008), although it was surprising that Robergs et al. (2004) suggested that no protons were consumed in the reaction that converts 1,3-bisphosphoglycerate and ATP to 3-phosphoglycerate and ATP. Berg et al. (2012) indicated that a proton is required for this reaction (see Figure 2.15). The proposal by Robergs et al. (2004) was described by Brooks (2018) as simply being untenable, with Brooks (2018) stating that the source of H^+ production during muscular contractions remains unknown. However, Robergs et al. (2019, 2023, 2024) have continued to present new evidence regarding the cause of muscular acidosis during exercise. That being said, irrespective of the cause, it is accepted that lactate and H^+ are produced during intense exercise and that their movement out of the muscle cell is coupled (Gladden, 2008; Juel, 1988; Juel, 1997).

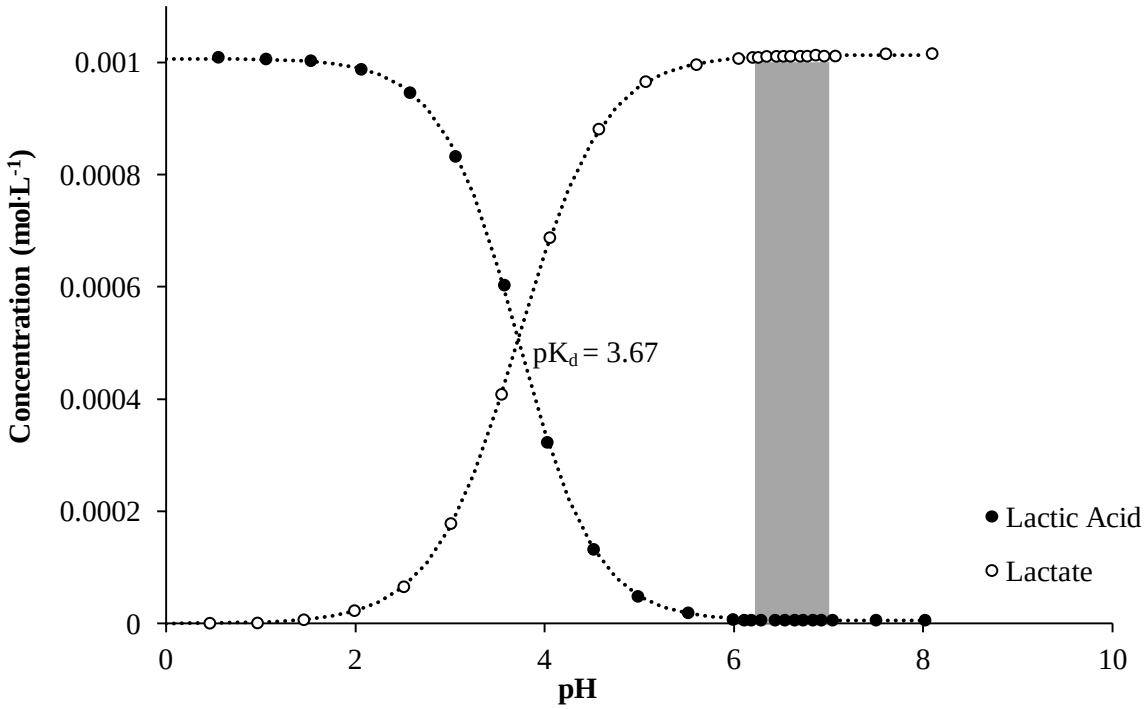

Figure 2.13 The pH dependent disassociation of lactic acid to lactate. Note: pK_d represents the disassociation constant and the shaded area represents the physiological pH range for muscle cells. The dotted line represents a sigmoidal function fitted to the data. A lactate concentration of 1 $mmol \cdot L^{-1}$ was chosen as it represents the resting conditions for skeletal muscles (Robergs et al., 2023).

Table 2.10 The source of protons during the breakdown of glucose and glycogen (Robergs et al., 2004).

Reaction	H^+ Source	
	Glu	Gly
Glucose + ATP ⟶ Glucose 6-phosphate + ADP + H^+	1	
Glucose 6-phosphate ⟶ Fructose 6-phospahte		
Fructose 6-phospahte + ATP ⟶ Fructose 1,6-bisphosphate + ADP + H^+	1	1
Fructose 1,6-bisphosphate ⟶ Dihydroxyacetone phosphate + Glyceraldehyde 3-phosphate		
Dihydroxyacetone phosphate ⟶ Glyceraldehyde 3-phosphate		
2 Glyceraldehyde 3-phosphate + 2 NAD^+ + 2 Pi ⟶ 2 1,3-bisphosphoglycerate + 2 NADH + 2 H^+	2	2
2 1,3-bisphosphoglycerate + ADP ⟶ 2 3-phoshoglycerate + 2 ATP		
2 3-phoshoglycerate ⟶ 2 2-phosphoglycerate		
2 2-phosphoglycerate ⟶ 2 phospheonolpyruvate + 2 H_20		
2 phospheonolpyruvate + 2 ADP + 2 H^+ ⟶ 2 Pyruvate + 2 ATP	-2	-2

Glu denotes glucose, Gly Glycogen, ATP adenosine triphosphate, and ADP adenosine diphosphate.

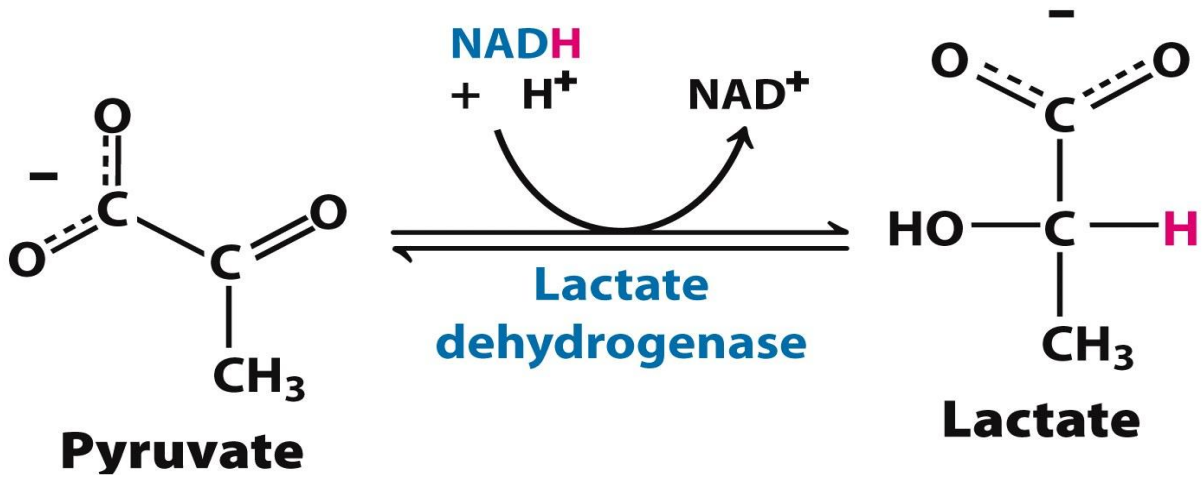

Figure 2.14 The reaction converting pyruvate to lactate. Note: C denotes carbon, O oxygen, H hydrogen, P phosphate, and ADP adenosine diphosphate (Berg et al., 2012).

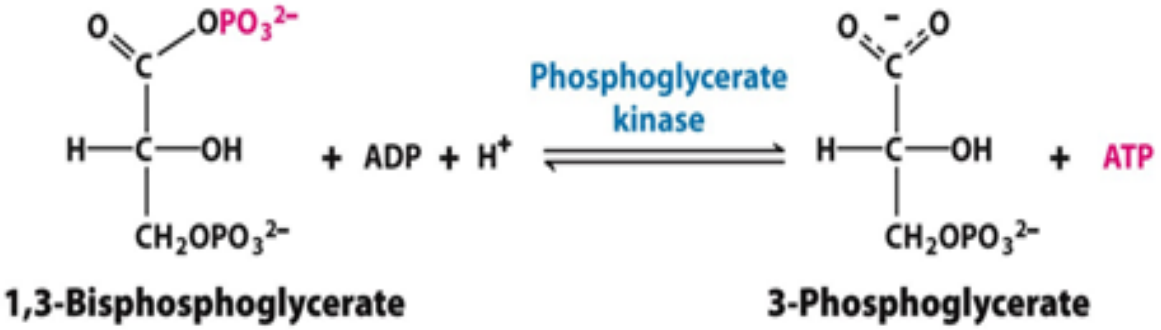

Figure 2.15 The reaction converting 1,3-biphosphoglycerate to 3-phosphoglycerate. Note: C denotes carbon, O oxygen, H hydrogen, P phosphate, ADP adenosine diphosphate, and ATP adenosine triphosphate (Berg et al., 2012).

2.2.2.5. Lactate Transport

The lactate shuttle hypothesis was first proposed by Brooks (1984). Over the last four decades, Brooks has continued to develop ideas concerning the various roles that lactate provides, as well as the means by which lactate is transported, within the human body (see Brooks, 2018). Whilst initially perceived to simply be via diffusion, it is now accepted that the movement of lactate into and out of the muscle cell will predominantly be via the monocarboxylate transporters (MCT), MCT1 and MCT4 (Gladden, 2011). MCT1 expression is greatest in slow-twitch fibres, whereas MCT4 expression is found to be higher in white glycolytic fibres (Juel & Halestrap, 1999). Driven by pH gradients, concentration gradients, and redox state, lactate and H^+ will move from the muscle into the

interstitial fluid, before entering the blood via endothelial clefts and across endothelial cells (Gladden, 2011). Within the blood itself, lactate is found in both plasma and red blood cells (Gladden, 2011). As the blood circulates through the body, lactate will then be drawn back into the interstitial fluid and into various tissues such as the liver, heart, and other muscle fibres (Goodwin et al., 2007). Whilst lactate is a major gluconeogenic precursor via the Cori cycle, the majority of lactate metabolism is directed towards oxidation (Brooks, 2018). Cardiac muscle is highly oxidative making it a major lactate consumer (Gladden, 2008). Skeletal muscles will, however, also oxidise lactate, with the oxidation rate being dependent on the metabolic rate of both the exercising and resting muscles (Gladden, 2008).

Muscle biopsies have revealed that muscle lactate concentration may rise from a resting concentration of 4.5 ± 0.4 mmol·kg dm^{-1} to 51.0 ± 4.6 mmol·kg dm^{-1} after a 10 s cycling sprint, 81.7 ± 4.7 mmol·kg dm^{-1} following a 20 s sprint (Bogdanis et al., 1998), and 119.0 ± 4.6 mmol.kg dm^{-1} after a 30 s sprint (Bogdanis et al., 1995), with corresponding pH values of 7.06 ± 0.02, 6.94 ± 0.02, 6.82 ± 0.03, and 6.72 ± 0.04 for the rest, post 10 s, post 20 s, and post 30 s time-points respectively (Bogdanis et al., 1995; Bogdanis et al., 1998). Muscle biopsies are, however, highly intrusive, meaning that there is a limit to the number of samples that can be taken during a single visit (Sahlin et al., 1978). Blood samples, on the other hand, can be taken more frequently. During recovery from intense exercise, arterial (Medbø et al., 2009; Sahlin et al., 1978), venous (Bogdanis et al., 1995; Fujitsuka et al., 1982; Medbø et al., 2009; Sahlin et al., 1976; Sahlin et al., 1978; Withers et al., 1991), and capillary (de Aguiar et al., 2015; de Aguiar et al., 2016; Gür, 2012; Hermansen & Osnes, 1972; Karlson et al., 1971; Merrels et al., 2019; Sahlin et al., 1976) blood lactate concentrations have been recorded. Merrels et al. (2019) reported that capillary lactate concentration peaked five minutes after a 60 s cycling sprint. Fujitsuka et al. (1982) found that venous lactate concentration peaked 7.65 minutes after ~ 60 s of intense running on a treadmill, and Withers et al. (1991) found that venous lactate concentration peaked ~ 7.5 minutes after 30 s, 60 s, and 90 s cycling sprints. In all three studies, lactate concentration did not return to the baseline level in the recovery periods tested, which were between 45 and 70 minutes (Fujitsuka et al., 1982; Merrels et al., 2019; Withers et al., 1991). Therefore, when considering the within session recovery periods that occur in the Match Sprint competition, if an increase in muscle lactate concentration does inhibit contractile function, removal would appear to be important for restoring sprint performance.

2.2.2.6. Effects of Lactate on Muscle Contractile Function

Skinned muscle fibre analyses facilitate the assessment of individual metabolites on muscle contractile function (Allen et al., 2008). Maximal isometric tension (P_o), maximal fibre shortening velocity (V_{max}), PPO, calcium release and reuptake by the sarcoplasmic reticulum, calcium sensitivity (pCa_{50} – the amount of calcium required to achieve 50% of peak tension), and the force calcium-concentration relationship (force-pCa relationship), have all been considered in vitro when analysing limitations in contractile function (Allen et al., 2008; Allen, 2020). Chase and Kushmerick (1988) investigated the effects of the addition of 50 $mmol \cdot L^{-1}$ of lactate on force production and shortening velocity in fast-twitch rabbit *psoas* muscles fibres at varying pH levels (6.0 – 8.0). At a pH of 7.1 the addition of 50 $mmol \cdot L^{-1}$ of lactate increased force production, whereas there was no significant difference in either force production or muscle fibre shortening velocity at the other pH values tested. However, Andrews et al. (1996) found that maximal force generation was inhibited in the presence of lactate, although not at the concentration tested by Chase and Kushmerick (1988). Andrews et al. (1996) examined maximal force and the force-pCa curves at lactate concentrations ranging from 5 $mmol \cdot L^{-1}$ to 50 $mmol \cdot L^{-1}$. In rabbit *psoas* fibres, maximal force decreased linearly up until a lactate concentration of 25 $mmol \cdot L^{-1}$. However, at a lactate concentration of 50 $mmol \cdot L^{-1}$ maximal force did not differ from the control (0 $mmol \cdot L^{-1}$). The pattern of this effect was also found to be similar in *soleus* fibres (Andrews et al., 1996), although it should be noted that the authors stated that the cause of the response was not obvious (Andrews et al., 1996). A minor reduction in maximal calcium activated force (a decrease of 2 – 3%) at a lactate concentration of 15 $mmol \cdot L^{-1}$ (Dutka & Lamb, 2000) and at 30 $mmol \cdot L^{-1}$ has subsequently been reported (Dutka & Lamb, 2000; Posterino et al., 2001), although when considering the magnitude of the reduction in force production, it has been suggested that an increase in lactate concentration is not a major contributing factor to muscle fatigue (Allen et al., 2008; Dutka & Lamb, 2000).

2.2.2.7. Effects of Acidosis on Muscle Contractile Function and on the Glycolytic Pathway

In section 2.2.2.3. it was stated that Bogdanis et al. (1996b) proposed that the reduction in energy derived from the glycolytic pathway during a repeated-sprint task was due to a decrease in pH creating an inhibitory effect on key glycolytic enzymes. A similar effect of a reduction in energy derived from the glycolytic pathway during a repeated-sprint task was reported by Gaitanos et al. (1993). However, neither Bogdanis et al. (1996b) nor Gaitanos et al. (1993) assessed the activity of any enzymes; instead, inferences were made on the basis of the changes that were found in the concentration of certain molecular compounds within the glycolytic pathway. Glaister et al. (2005) stated that whilst H^+ accumulation is known to inhibit the activity of key glycolytic enzymes, the

effect of pH on phosphofructokinase activity, which is a key glycolytic enzyme, is negligible under the physiological conditions that occur during a repeated-sprint task. That being said, the muscle biopsy data reported by Bogdanis et al. (1996b) and Gaitanos et al. (1993) clearly displays a reduction in energy derived from glycolysis. Therefore, the cause of this effect still requires further investigation, whether or not it was created by acidosis.

For many decades the standard textbook explanation of muscle fatigue during high-intensity exercise related to acidosis (Allen, 2020). Allen et al. (2020) stated that this explanation largely originated from the research by Fabiato and Fabiato (1978). In 1978, Fabiato and Fabiato (1978) found that in skinned frog *semitendinosus* muscle fibres, increases in H^+ concentration inhibited maximum tension, as well as pCa_{50} (see Figure 2.16). However, a limitation in investigations that use skinned muscle fibres is that the fibres become increasingly unstable as temperature increases (Pate et al., 1995). As reliability was greater at lower temperatures, initially research was conducted at temperatures of 15°C or lower, although these temperatures did not provide a realistic representation of the human body (Pate et al., 1995). Temperature-jump technological developments have enabled skinned fibre analyses to be conducted at temperatures (30°C) that are closer to the normal range of mammalian muscle fibres (Pate et al., 1995). Pate et al. (1995) reported that the effects of increasing H^+ concentration on V_{max} and P_o were temperature dependent (see Table 2.11), with the relative reduction in P_o being significantly dampened and V_{max} actually increasing slightly at 30°C, although the increase in V_{max} was described as being minimal and possibly related to the fitting procedures that were used. A reduction in the effect of an increase in H^+ concentration on P_o at higher temperatures (10 – 15°C vs 30°C) has subsequently been demonstrated (Knuth et al., 2006) and in a review of the literature, Allen et al. (2008) concluded that an increase in H^+ concentration was not the main cause of muscle fatigue, with the actual effects on force production being quite small. However, in stark contrast, Sundberg and Fitts (2019) stated that the multi-faceted and synergistic effects of Pi and H^+ on the contractile function of the muscle, is the major cause of fatigue during high-intensity exercise. The response and rebuttal from Fitts (2016) and Westerblad (2016) indicate that the importance that acidosis plays in fatigue during high-intensity exercise continues to be debated (means of enhancing lactate and H^+ clearance will be considered further in Section 2.4.).

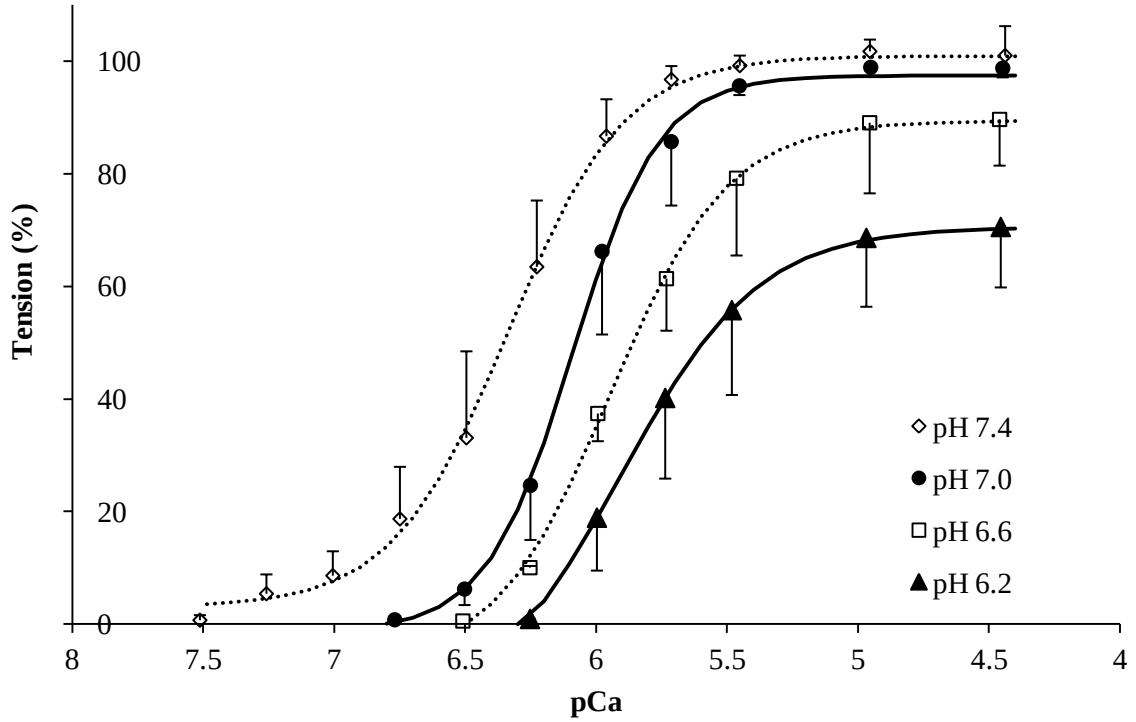

Figure 2.16 The effects of changes in pH on the force-calcium-concentration relationship (expressed on a logarithmic scale) in frog *semitendinosus* muscle fibres (Fabiato & Fabiato, 1978).

Table 2.11 The effects of temperature on isometric tension (Po) and shortening velocity (V_{max}) under control and acidic conditions. Data are presented as mean ± standard error (Pate et al., 1995).

Temperature (°C)	pH	P_o ($N \cdot mm^{-2}$)	V_{max} (muscle lengths$\cdot s^{-1}$)
30	7.0	0.28 ± 0.02	6.28 ± 0.14
30	6.2	0.23 ± 0.02*	6.68 ± 0.23
10	7.0	0.15 ± 0.01⁑	1.89 ± 0.05⁑
10	6.2	0.07 ± 0.01	1.31 0.05

* denotes a significant difference in P_o values at 30°C when pH was altered. ⁑ denotes a significant difference in P_o values at 10°C when pH was altered.

2.2.2.8. The Tricarboxylic Acid Cycle and the Electron Transport Chain

It was previously stated in section 2.2.2.3. that the glycolytic pathway resulted in the production of two/three molecules of ATP, two molecules of NADH, and two molecules of pyruvate. In the case of NADH, electrons are carried across the mitochondrial membrane by a shuttle, before ending up at the electron transport chain. One such shuttle is the glycerol 3-phosphate shuttle (see Figure 2.17) (Berg et al., 2002). In the case of the two molecules of pyruvate, the movement from the cytosol to the inner mitochondrial membrane is achieved via a pyruvate carrier (McCommis & Finck, 2015). Within the mitochondrial matrix, pyruvate is then converted to acetyl-CoA, with an additional molecule of NADH being produced at this time (see Figure 2.18) (Berg et al., 2012). Acetyl-CoA then

enters the tricarboxylic acid cycle (TCA) cycle (see Figure 2.19). For each complete cycle, three NADH, one dihydroflavine adenine dinucleotide ($FADH_2$), and one molecule of guanosine triphosphate, which is then converted to ATP, are produced. NADH and $FADH_2$ proceed to the electron transport chain, arriving at complexes I and II respectively (see Figure 2.20) (Berg et al., 2012), depositing two electrons and releasing NAD^+, flavin adenine dinucleotide (FAD), as well as H^+ (Marieb & Hoehn, 2016). As the electrons move through the electron transport chain (complexes I to IV), some of their energy is released and used to pump H^+ from the mitochondrial matrix into the intermembrane space, creating a proton gradient (Ahmad et al., 2020; Marieb, & Hoehn, 2016; Powers & Howley, 2009). At complex IV, the electrons combine with H^+ and O_2 to form water (Berg et al., 2012). The electrochemical gradient created from the pumping of H^+ into the intermembrane space, then drives the movement of H^+ back into the mitochondrial matrix via complex V (ATP synthase) (Marieb & Hoehn, 2016). ATP synthase uses this electrical energy to synthesize ATP from ADP and Pi (Marieb & Hoehn, 2016). Each NADH, therefore, results in the production of ~ 3 ATP molecules and each $FADH_2$ results in ~ 2 ATP molecules (Marieb & Hoehn, 2016). Whilst energy production via the aerobic pathway is much greater than via the anaerobic systems, the rate of ATP turnover is relatively low (see Table 2.12) (Baker et al., 2010; Sahlin et al., 1998). Therefore, the relative contribution of ATP produced via the aerobic system during a sprint may be limited.

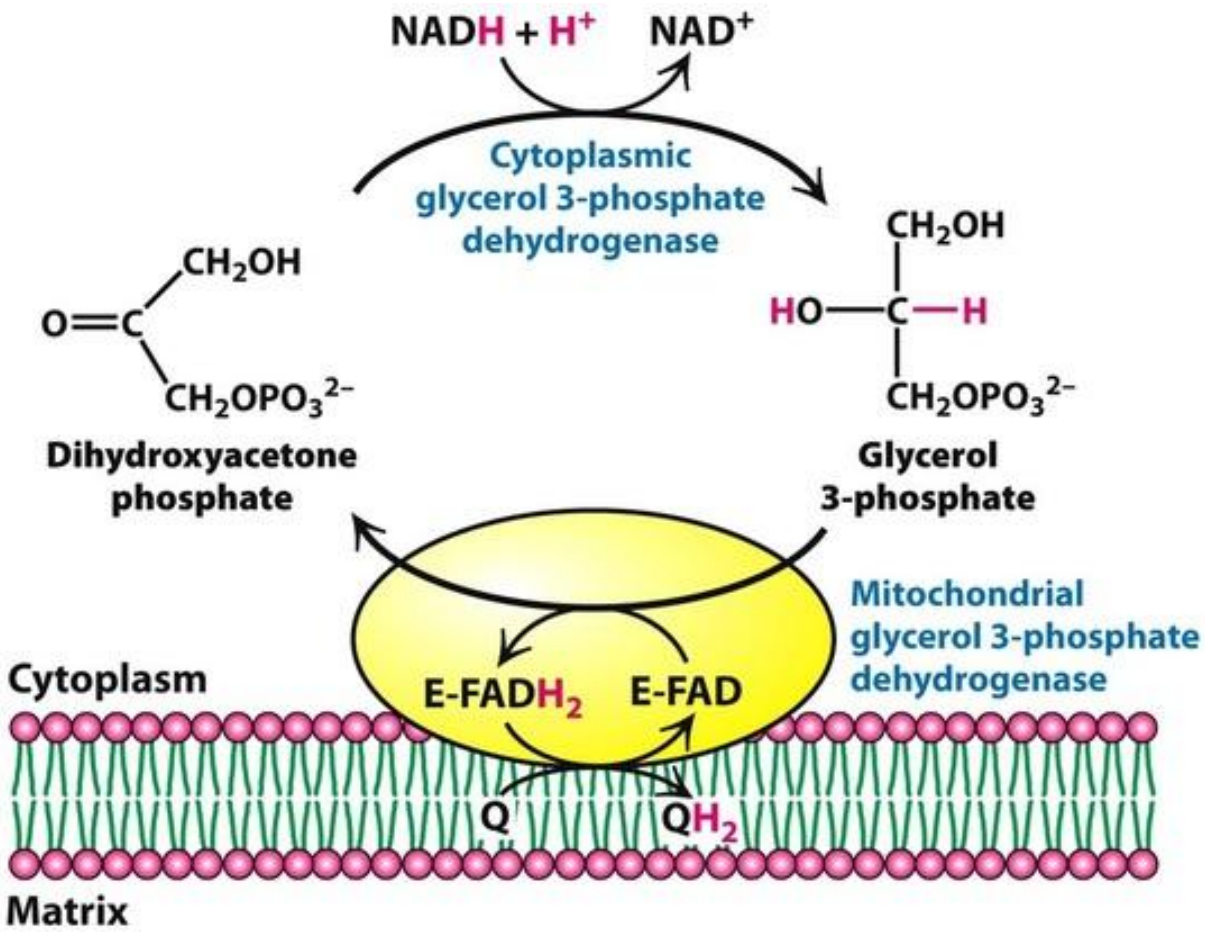

Figure 2.17 The glycerol 3-phosphate shuttle. Note: C denotes carbon, O oxygen, H hydrogen, P phosphate, FAD flavin adenine dinucleotide, and NAD nicotinamide adenine dinucleotide (Berg et al., 2012).

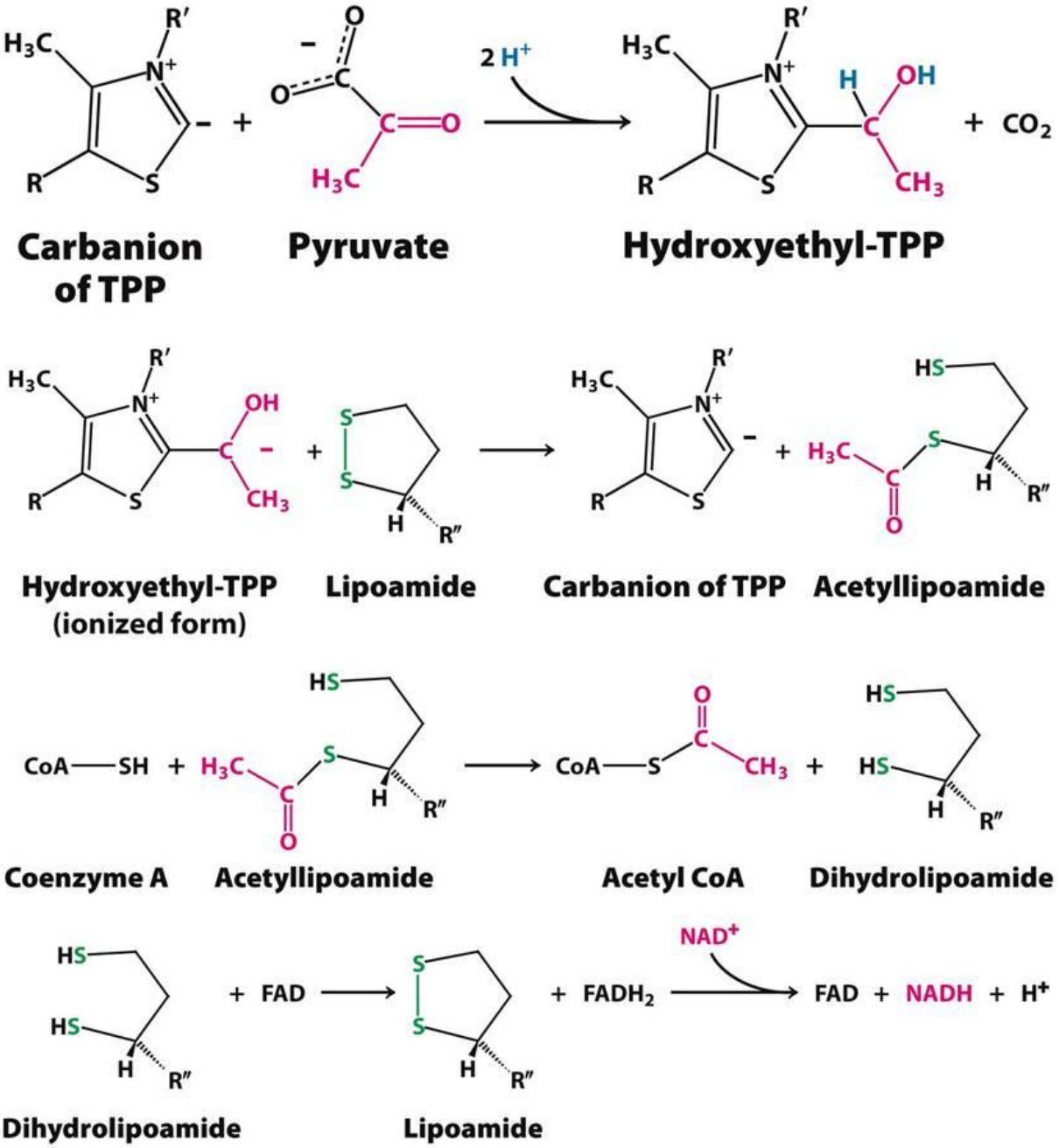

Figure 2.18 The conversion of pyruvate to acetyl-CoA. Note: C denotes carbon, O oxygen, H hydrogen, CoA coenzyme A, R represents parts of the chemical structure that are not involved in the reaction, S sulphur, N nitrogen, FAD flavin adenine dinucleotide, and NAD nicotinamide adenine dinucleotide (Berg et al., 2012).

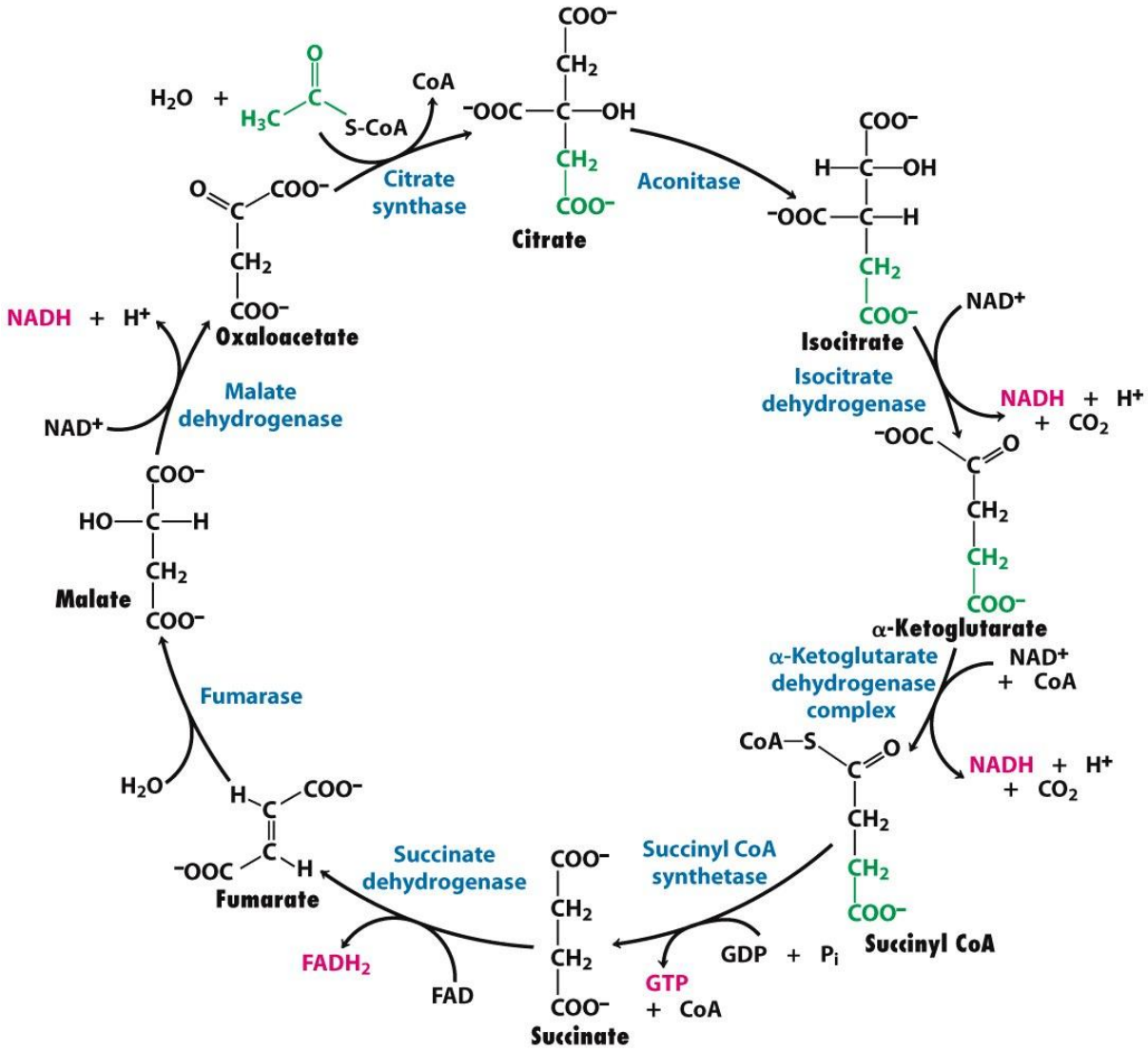

Figure 2.19 The tricarboxylic acid cycle. Note: C denotes carbon, CoA coenzyme A, FAD flavin adenine dinucleotide, GDP guanosine diphosphate, GTP guanosine triphosphate, H hydrogen, N nitrogen, NAD nicotinamide adenine dinucleotide, O oxygen, and S sulphur (Berg et al., 2012).

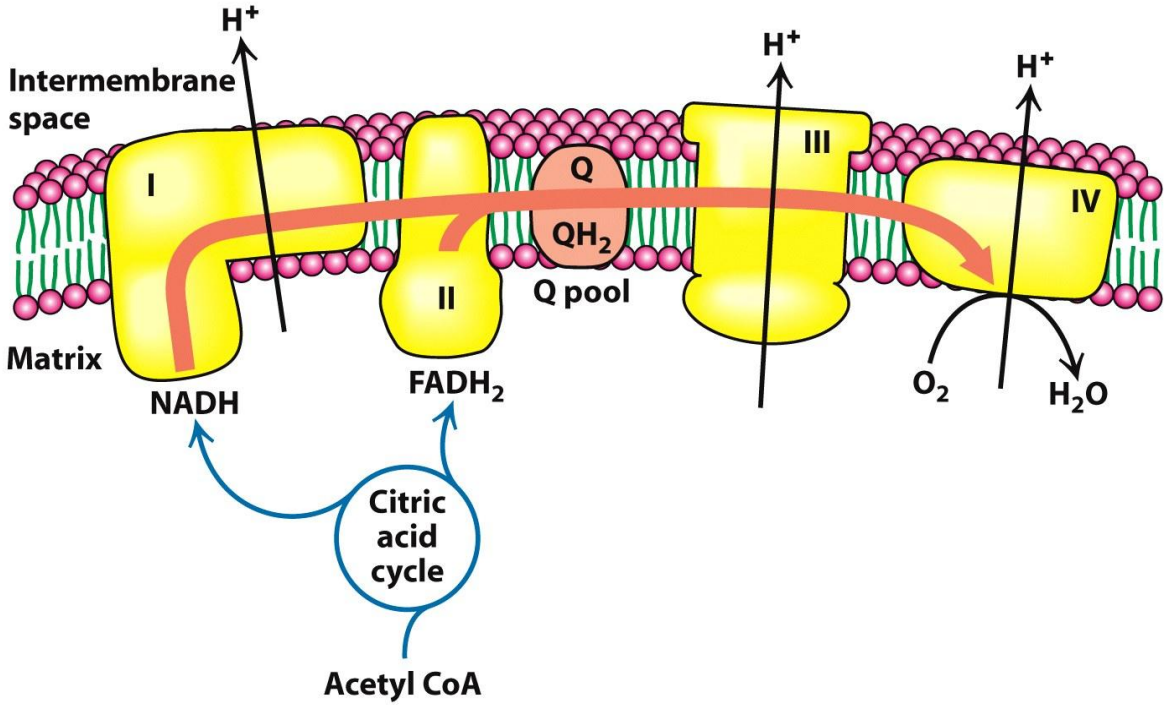

Figure 2.20 The electron transport chain. Note: FAD denotes flavin adenine dinucleotide, H hydrogen, NAD nicotinamide adenine dinucleotide, O oxygen and Q coenzyme Q (Berg et al., 2012).

Table 2.12 Maximal energy turnover rates for the phosphocreatine, glycolytic, and aerobic energy systems (Maughan, & Gleeson, 2004).

Energy System	Maximal rate of ATP resynthesis (mmol ATP·kg dm^{-1}·s^{-1})
Phosphagen System	9.0
Glycolytic System	4.5
Glycogen Oxidation	2.8
Glucose Oxidation	1.0
Fat Oxidation	1.0

Note: ATP denotes adenosine triphosphate.

2.2.2.9. Summary

In summary, for a voluntary muscular contraction to occur, a signal is initiated in the primary motor cortex of the brain. An upper motor neuron is then responsible for transmitting the signal from the cortex to a lower motor neuron in the spinal cord. The signal is then sent via an axon to the muscle. At the axon terminal, voltage-gated channels allow an influx of calcium into the synaptic bulb, causing the release of acetylcholine into the synaptic cleft. Acetylcholine then binds to receptors opening voltage-gated channels on the muscle membrane, allowing positive ions to be drawn into the muscle cell, producing a graded potential. If the threshold potential is reached, an action potential is generated, which travels along the muscle membrane. The T-tubule system provides a route for the signal to travel into the muscle cell, resulting in the release of calcium from

the sarcoplasmic reticulum. Calcium can then bind to troponin-C causing a shift in tropomyosin, allowing the myosin heads to bind with actin and a muscular contraction to occur. Following the contraction, calcium is then pumped back into the sarcoplasmic reticulum via the SERCA pump, which is one of three major proteins that contribute to the ATP requirements of a muscular contraction. Stored ATP is, however, limited, although distinct pathways enable its regeneration to occur. PCr stores are also limited and the complete rephosphorylation of creatine may not occur over the shorest recovery periods that occur duirng the Match Sprint competition. PCr resynthesis may also be a biphasic process, with the secondary phase being dependent on muscle pH. Whilst muscle glycogen stores should be sufficient to facilitate ATP regeneration for mulitple sprints, the glycolytic pathway may also be inhibited when sprints are repeated. Skinned muscle fibre analysis facilitates the investigation of the effects of individual metabolites on muscle contracile function. There is some evidence to suggest that an increase in muscle lactate concentration could inhibit force production, albeit the general opinion is that elevations in lactate concentration do not provide a major concern for performance recovery. In contrast, the role of accumulating H^+ on contractile function continues to be debated. From a practical perspective, the view of many athletes, coaches, and some sports scientists, remains that acidosis plays a major role in fatigue and its removal is essential if recovery is to be optimised. Finally, the capacity of the aerobic system is substantailly greater than the anaerobic pathways, although the rate of ATP resynthesis is much slower, which may limit the relative contribution made during a cycling sprint.

2.3. OXYGEN UPTAKE AND SPRINT PERFORMANCE

The power output that is produced during a sprint may be three-to-five times greater than the power output that elicits maximal oxygen uptake ($\dot{V}O_{2max}$) (Gastin, 2001). The duration of the sprint will, however, affect the relative contribution of ATP derived from the aerobic and anaerobic pathways (Gastin, 2001). Whilst the exact duration of each sprint in the Match Sprint competition will be considered in greater detail in section 2.7.3., Douglas et al. (2021) and Ferguson et al. (2020) suggested that maximal efforts in sprint track cycling disciplines are typically in the range of 15 – 60 s.

2.3.1. The Relative Contribution of the Aerobic and Anaerobic Energy Systems to Sprint Cycling Performance

A number of estimates have been made for the relative contribution of the aerobic and anaerobic energy systems to ATP production during cycling sprints of various durations (see Table 2.13) (Bediz et al., 1998; Beneke et al., 2002; Calbet et al., 1997; Granier et al., 1995; Medbø &

Tabata, 1989; Serresse et al., 1988; Smith & Hill, 1991; Stevens et al., 1986; Withers et al., 1991; Yang & Park, 2019). Whilst mechanical efficiency, as well as athlete type (sprinters versus middle distance runners) could affect the relative quantities (Granier et al., 1995), the energy contribution from the aerobic pathway will increase with sprint duration (Calbet et al., 1997; Medbø & Tabata, 1989; Serresse et al., 1988; Withers et al., 1991) and may also increase when sprints are repeated with an incomplete recovery period (Bogdanis et al., 1996b). When four minutes separated two WAnTs, Bogdanis et al. (1996b) found that the reduction in performance, an 18% decrease in MPO, was not as great as the decrease in energy production from the anaerobic pathways. It was, therefore, suggested that an increase in aerobic metabolism, evidenced by an increase in oxygen uptake ($\dot{V}O_2$) during the second sprint, may have compensated for some of the decrease in anaerobic energy production.

Table 2.13 Relative contribution of the aerobic and anaerobic energy systems to sprint cycling performance.

Author	Participants	Exercise mode/variations	Exercise Duration (s)	Energy system contribution	
				Aerobic (%)	Anaerobic (%)
Bediz et al. (1998)	30 sedentary men	Cycling (low resistance)	30 s	18.5	81.5
Bediz et al. (1998)	30 sedentary men	Cycling (high resistance)	30 s	19.5	80.5
Beneke et al. (2002)	11 male rugby players	Cycling	30 s	18.6	81.4
Calbet et al. (1997)	19 healthy men	Cycling	30 s	22.9	77.1
Calbet et al. (1997)	19 healthy men	Cycling	45 s	30.9	69.1
Granier et al. (1995)	7 male sprinters	Cycling (ME: 16%)	30 s	28	72
Granier et al. (1995)	7 male sprinters	Cycling (ME: 25%)	30 s	19	81
Granier et al. (1995)	7 male sprinters	Cycling (ME: Individualised %)	30 s	29	71
Granier et al. (1995)	7 male middle distance runners	Cycling (ME: 16%)	30 s	45	55
Granier et al. (1995)	7 male middle distance runners	Cycling (ME: 25%)	30 s	30	70
Granier et al. (1995)	7 male middle distance runners	Cycling (ME: Individualised %)	30 s	46	55
Medbo and Tabata (1989)	17 healthy men	Cycling	30 s (resistance designed to elicit fatigue)	40	60
Medbø and Tabata (1989)	17 healthy men	Cycling	60 s (resistance designed to elicit fatigue)	50	50
Medbø and Tabata (1989)	17 healthy men	Cycling	120-180 s (resistance designed to elicit fatigue)	65	35
Serresse et al. (1988)	25 healthy men	Cycling (ME: 16.2%)	10 s	3	97
Serresse et al. (1988)	25 healthy men	Cycling (ME: 16.2%)	30 s	28	72
Serresse et al. (1988)	25 healthy men	Cycling (ME: 16.2%)	90 s	46	54
Smith and Hill (1991)	6 healthy men	Cycling	30 s	16	84
Stevens et al. (1986)	13 junior hockey players	Cycling	30 s	44.3	55.7
Withers et al. (1991)	6 healthy men	Cycling (invasive)	30 s	28	72
Withers et al. (1991)	6 healthy men	Cycling (non-invasive)	30 s	28	72
Withers et al. (1991)	6 healthy men	Cycling (invasive)	60 s	49	51
Withers et al. (1991)	6 healthy men	Cycling (non-invasive)	60 s	49	51
Withers et al. (1991)	6 healthy men	Cycling (invasive)	90 s	64	36
Withers et al. (1991)	6 healthy men	Cycling (non-invasive)	90 s	61	39
Yang and Park (2019)	15 elite youth cyclists (mixed sex)	Cycling	10 s	13	87

Note: ME denotes mechanical efficiency.

2.3.2. Breath-by-breath Analysis of Oxygen Uptake

The consumption of O_2 by biological tissues is described by the Fick Equation (see Equation 2.04) (Jones & Poole, 2005). According to the Fick Equation, $\dot{V}O_2$ is dependent on blood flow to the tissue and the amount of O_2 that is extracted from the blood to support oxidative phosphorylation in the mitochondria (Jones & Poole, 2005). Exercise physiologists are often interested in $\dot{V}O_2$ at the muscle, although it is common to measure $\dot{V}O_2$ at the mouth and make inferences about $\dot{V}O_2$ at the exercising muscle (Jones & Poole, 2005). The measurement of $\dot{V}O_2$ at the mouth and nose is commonly conducted either by the collection, and subsequent analysis, of expired air in Douglas bags or via an automated breath-by-breath system (Jones & Poole, 2005). $\dot{V}O_{2max}$ and $\dot{V}O_{2peak}$ (peak oxygen uptake) both provide a measure of the upper limit of energy production via aerobic metabolism. Surprisingly little data has, however, been reported on the $\dot{V}O_{2max}$ of sprint cyclists, although Lee and Seo (2023) reported $\dot{V}O_{2max}$ values for Keirin cyclists, ranging from 64.6 ± 2.5 $ml \cdot kg^{-1} \cdot min^{-1}$ in their high $\dot{V}O_{2max}$ group to 49.4 ± 3.3 $ml \cdot kg^{-1} \cdot min^{-1}$ in their low $\dot{V}O_{2max}$ group. Lee and Seo (2023) also suggested that $\dot{V}O_{2max}$ was a key performance and recovery predictor in the Keirin.

$$\dot{V}O_2 = Q_T \times (CaO_2 - CvO_2) \qquad \text{(Equation 2.04)}$$

where $\dot{V}O_2$ denotes oxygen uptake, Q_T tissue blood flow, CaO_2 arterial oxygen content, and CvO_2 venous oxygen content.

The rate of change of $\dot{V}O_2$ in response to an alteration in exercise intensity represents the kinetics of the response (Jones & Poole, 2005). $\dot{V}O_2$ on-kinetics ($\dot{V}O_{2on}$) are reflective of the change in $\dot{V}O_2$ following an increase in exercise intensity, or the start of exercise, whereas the adjustment in $\dot{V}O_2$ following a decrease in exercise intensity, or the cessation of exercise, form $\dot{V}O_2$ off-kinetics ($\dot{V}O_{2off}$). Faster $\dot{V}O_{2on}$ kinetics have consistently been found in individuals with a higher $\dot{V}O_{2max}$ (Babcock et al., 1994; Berger et al., 2006; Cleuziou et al., 2005; Dale & Glaister, 2018; Dogra et al., 2013; Grey et al., 2015; Koppo et al., 2004; Norris & Petersen, 1998; Marwood et al., 2010). Faster $\dot{V}O_{2on}$ kinetics could aid sprint performance by adding to the energy pool during a sprint, potentially enhancing MPO. With regards to $\dot{V}O_{2off}$ kinetics, as PCr resynthesis occurs via the rephosphorylation of creatine by aerobically produced ATP (McMahon & Jenkins, 2002), individuals that possess faster $\dot{V}O_{2off}$ kinetics could also experience faster PCr resynthesis (Buchheit et al, 2012). The post-exercise recovery of $\dot{V}O_2$ has been found to follow a similar time-course to PCr resynthesis (Rossiter et al., 2002), at least at exercise intensities that are below $\dot{V}O_{2max}$ (Glaister et al., 2014). In general, findings regarding the relationship between $\dot{V}O_2$ parameters and repeated-sprint performance measures have, however, been mixed, ranging from trivial to very large (Aziz et al., 2000; Aziz et al.,

2007; Bishop et al., 2003, Bishop & Goodman, 2004; Buchheit, 2012; Castagna et al., 2007; de Aguiar et al., 2016; Dupont et al., 2005; Dupont et al., 2010; López-Segovia et al., 2015; Pajeja-Blanco et al., 2016; McMahon & Wenger, 1998; Wadley & Le Rossignol, 1998).

When using field tests as a surrogate measure of $\dot{V}O_{2max}$, Pareja-Blanco et al. (2016) even noted a difference in the strength of the relationship between $\dot{V}O_{2max}$ and repeated-sprint performance (mean sprint time) depending on the type of test used (multi-stage fitness test r = -0.39 versus Yo-Yo Intermittent recovery test r = -0.62). It is possible that the larger correlation found with the Yo-Yo test was due to the test providing a greater reflection of the demands of the sport (soccer), in which the participants were highly trained (Pajeja-Blanco et al., 2016). In the Match Sprint cycling competition, the duration of each sprint, the number of sprints performed in a session, and the rest period between sprints will vary, but the duration of the sprint will most likely be greater than 10 s and the shortest recovery period equal to or greater than 10 minutes. Therefore, it may not be possible to transfer the current findings from repeated-sprint studies, that have typically consisted of short duration sprints (4 – 8 s), with brief recovery periods (20 – 30 s), repeated 5 – 15 times, to guide the importance of aerobic parameters to sprint cycling performance or performance restoration. Furthermore, whilst breath-by-breath $\dot{V}O_2$ can readily be measured in most laboratories, and is often used as an indicator of muscle $\dot{V}O_2$ (Lai et al., 2006), the use of near infrared spectroscopy (NIRS) may offer a preferred means of assessing $\dot{V}O_2$ at the muscle (Ufland et al., 2013).

2.3.3. Near Infrared Spectroscopy

All electromagnetic radiation travels in waves that oscillate through space at the speed of light (Johnston & Fauber, 2016). Electric and magnetic fields lie perpendicular to each other (see Figure 2.21) (Hewitt, 2017). The electromagnetic spectrum (see Figure 2.22) incorporates the full range of electromagnetic radiation. The longest waves (radio waves) have the least energy and the lowest frequency, whereas the shortest waves (gamma waves) have the highest energy and the greatest frequency. Wavelengths at both ends of the electromagnetic spectrum are used in medical imaging: gamma rays are used in nuclear medicine, whereas radio waves are used in magnetic resonance imaging (Johnston & Fauber, 2016).

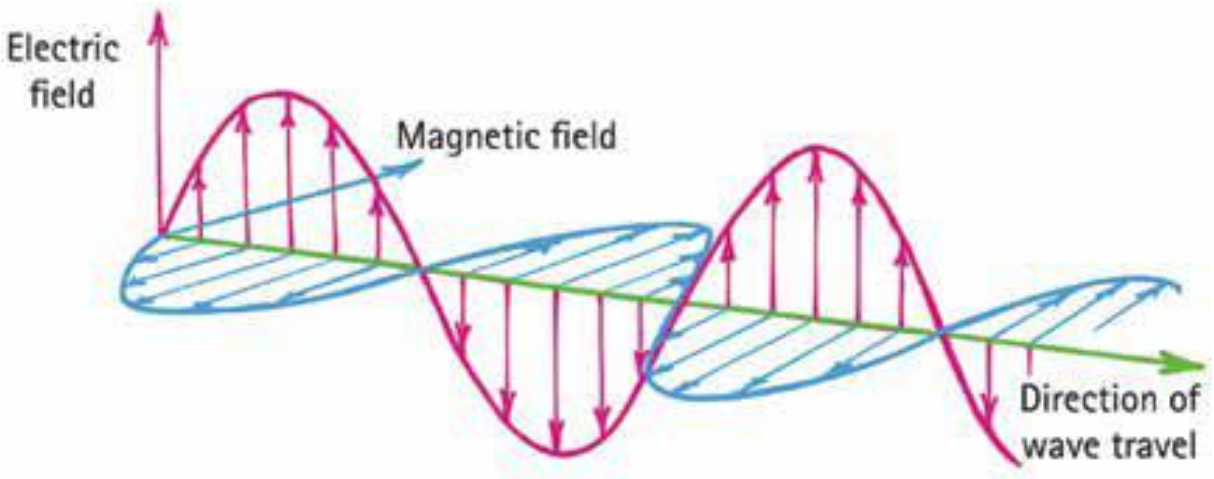

Figure 2.21 The direction of travel of an electromagnetic wave (Hewitt, 2017).

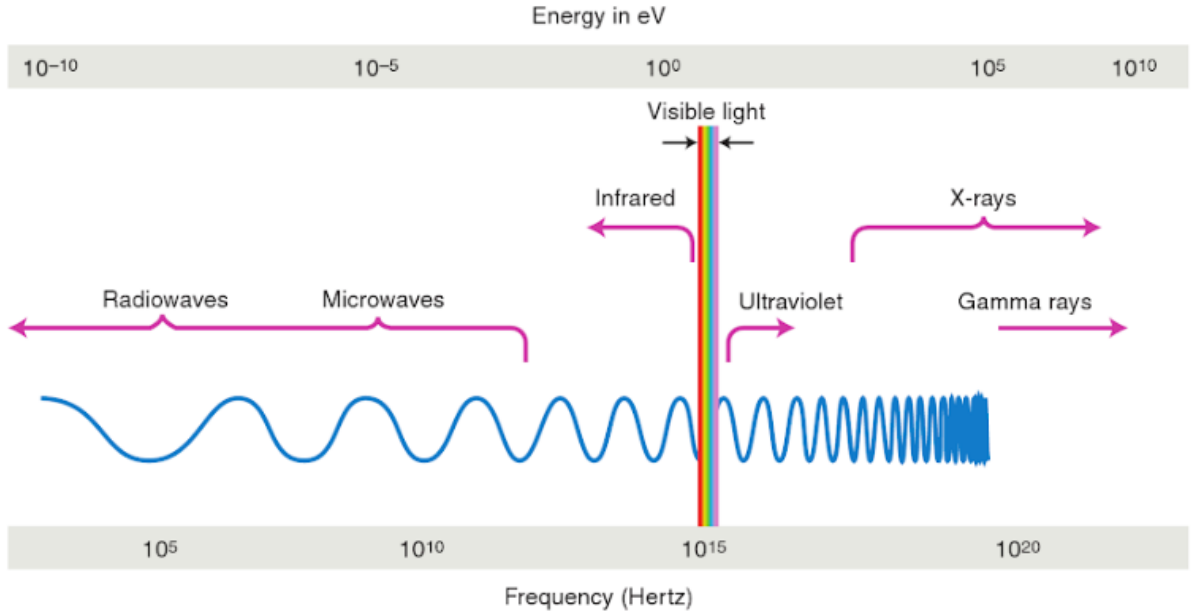

Figure 2.22 The electromagnetic spectrum (Johnston & Fauber, 2016).

Spectroscopy is the study of the interaction of electromagnetic radiation and matter (Ball, 2001). Within the infrared region of the electromagnetic spectrum, three sub-categories (near, mid, and far) exist (Bogue, 2011) (see Table 2.14). In the wavelength range 650 – 950 nm, the attenuation of the signal will be due to haemoglobin (Hb), myoglobin (Mb), skin melanin, water, adipose tissue, intra-muscular lipids, and cytochrome c oxidase (Ferrari et al., 2011). The spectral absorbance of Mb and Hb is, however, almost indistinguishable and the exact contribution that Mb makes to the signal attenuation is still debated (see Jones et al., 2016). Therefore, the combination of oxygenated Hb and Mb will be referred to as O_2Hb and, similarly, deoxygenated Hb and Mb as HHb. The sum of O_2Hb and HHb reflects total haemoglobin (tHb), which is considered to be indicative of blood flow (Smith & Billaut, 2010).

Table 2.14 Sub-categories of infrared waves (Bogue, 2011).

Category	Abbreviation	Wavelength range (nm)
Near-infrared	NIR	750 - 3000
Mid-infrared	MIR	3000 - 50000
Far-infrared	FIR	50000 - 1000000

Post-exercise muscle reoxygenation rates were first investigated using NIRS by Chance et al. (1992). Following an intense rowing protocol (~ 6 minutes of maximal effort), the time for muscle reoxygenation to reach 50% ($t_{1/2}$) of its maximum (end exercise to the peak of the overshoot) was calculated (Chance et al., 1992). In female rowers $t_{1/2}$ was 26.0 ± 4.4 s and in male rowers it was 45.3 ± 6.6 s. Whilst similar evaluation protocols have subsequently been used (Ichimura et al., 2006; Nagasawa, 2013), others have chosen to mathematically model the reoxygenation time-course with a one-phase exponential function (see Equations 2.05 & 2.06) (Bopp et al., 2011; Brizendine et al., 2013; Buchheit et al., 2012; McCully et al., 1994), although Bopp et al. (2011) questioned whether an alternative mathematical function would provide a better representation of the response. Investigating post-occlusive reactive hyperaemia in the forearm, Bopp et al. (2011) contrasted the goodness of model fit of a one-phase exponential function with two sigmoidal functions (Gompertz - see Equation 2.07 and logistic - see Equation 2.08). As hypothesized, the Gompertz function provided a superior fit to the O_2Hb data, whereas a logistic function provided a preferred fit to the HHb data (see Figure 2.23 for examples of Gompertz and exponential functions fit to the O_2Hb data). Whilst the data displayed in Figure 2.23 do support this suggestion, it is noteworthy that the data were truncated at ~ 40 s. When viewing the full pattern of the response from a representative participant (see Figure 2.24), it is questionable whether any of these models provide an accurate reflection of the response that is depicted.

$$Y = Y_{(0)} + A(1 - e^{\frac{-(t)}{\tau}}) \quad \text{(Equation 2.05)}$$

$$Y = Y_{(0)} + A(1 - e^{\frac{-(t-TD)}{\tau}}) \quad \text{(Equation 2.06)}$$

$$Y = Y_{(0)} + Ae^{-e^{\frac{(t-TD)}{\tau}}} \quad \text{(Equation 2.07)}$$

$$Y = Y_{(0)} + \frac{A}{1 + e^{-\frac{t-TD}{\tau}}} \quad \text{(Equation 2.08)}$$

where Y is the measure of interest, Y_0 represents the baseline level, A is the asymptotic amplitude, t is time, TD is the time delay (mono-exponential) or the time at the inflection point (Gompertz and logistic), and τ is the time constant.

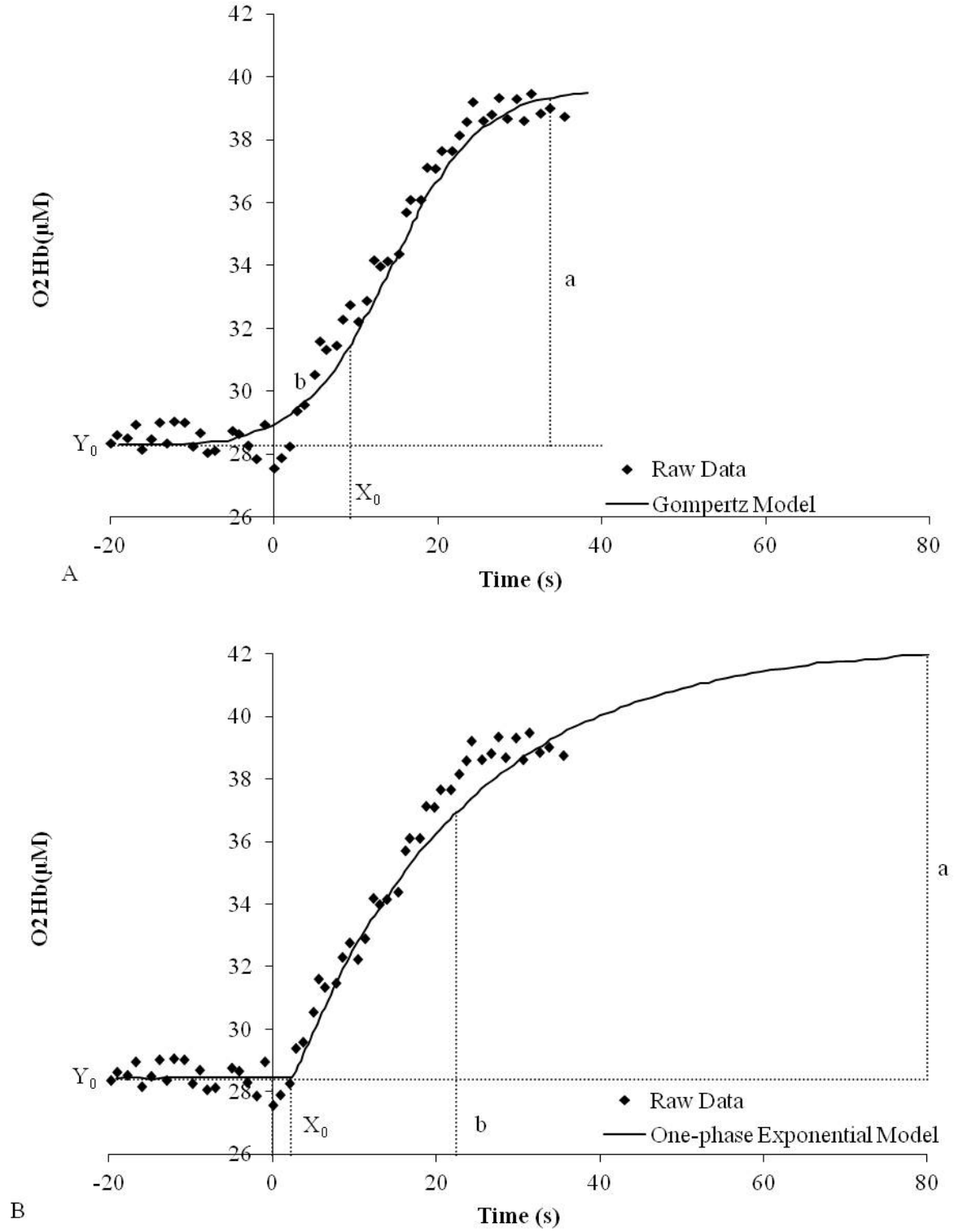

Figure 2.23 The response of oxygenated haemoglobin (O_2Hb) to post-occlusive reactive hyperaemia in the forearm. A) an example of the Gompertz function applied to the O_2Hb data; B) an example of a mono-exponential function applied to O_2Hb data (Bopp et al., 2011)

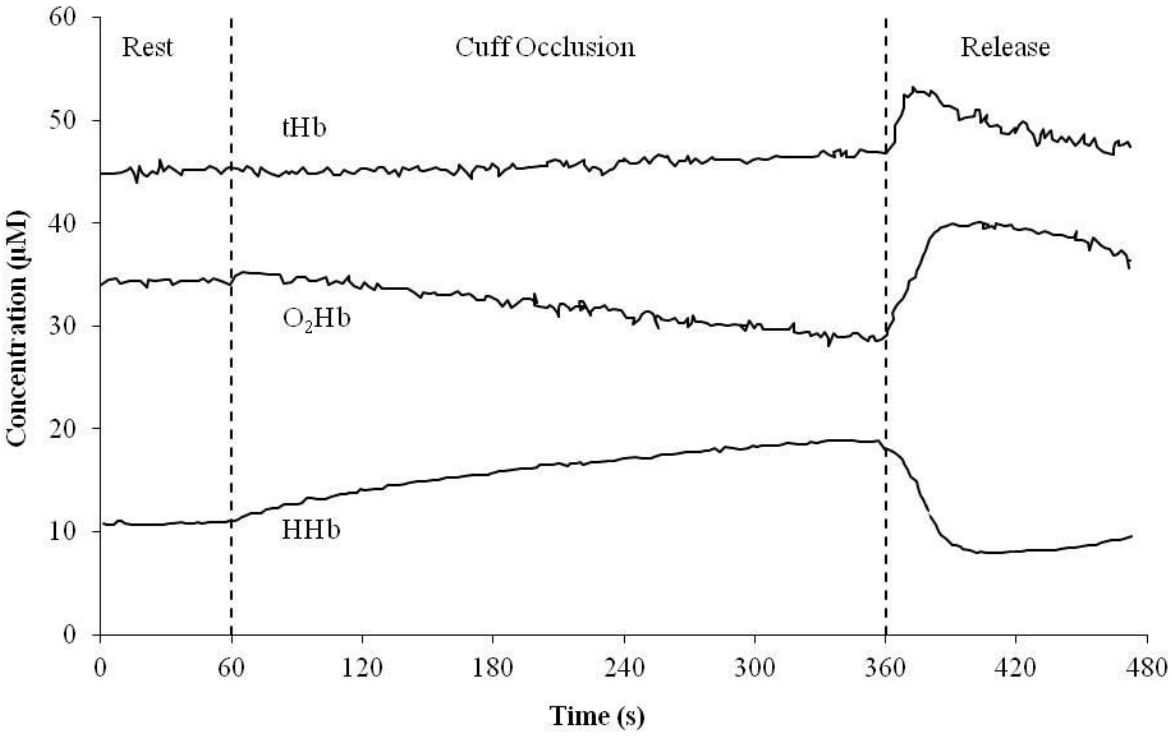

Figure 2.24 An example of the full data set for a representative participant using near infrared spectroscopy to assess the response of total haemoglobin (tHb), oxygenated haemoglobin (O_2Hb), and deoxygenated haemoglobin (HHb) to post-occlusive reactive hyperaemia in the forearm (Bopp et al., 2011).

An alternative method to calculate reoxygenation rates, employed by Buchheit and Ufland (2010), was to apply a linear model to the first 15 s of recovery data obtained during repeated-sprint exercise (the slope of the line being used as an index of reoxygenation); albeit, the rationale for using a linear function was due to the short time period between sprints, not because the authors believed that a one-phase exponential function was inappropriate. The authors also decided to focus their analysis on tissue saturation index (TSI), stating that TSI, which provides a measurement of the ratio of O_2Hb to tHb, was a better indicator of the muscle oxygenation status during exercise, when blood flow is not constant (Buchheit & Ufland, 2010). For the same reason, others (Dupont et al., 2007; Billaut & Buchheit, 2013; Kriel et al., 2016; Smith & Billaut, 2012) have chosen not to analyse O_2Hb, but to instead focus on HHb, stating that HHb is essentially independent of blood flow (Smith & Billaut, 2012). Following an eight-week endurance training programme, Buchheit and Ufland (2010) found that repeated-sprint ability improved, alongside the post-sprint recovery of TSI. However, whilst an improvement in the recovery rate of TSI with endurance training seems logical, not all findings have been in support (Chance et al., 1992; Kime et al., 2003; Nagasawa, 2013). When compared to inexperienced rowers, Chance et al. (1992) reported that muscle reoxygenation was slower in trained rowers. Likewise, Nagasawa (2013) found that when compared to sprinters or the control group, the reoxygenation rate (first 6 s of recovery TSI) following a WAnT was slower in

endurance trained individuals. Furthermore, Nagasawa (2013) found that muscle reoxygenation was delayed in participants with a higher $\dot{V}O_{2max}$. A delay in muscle reoxygenation was also reported by Buchheit et al. (2012), but Nagasawa (2013) was the first to suggest that differences in the duration of this delay may be related to training status. It may, therefore, be necessary to compute the mean response time (MRT), as well as τ, when quantifying recovery TSI using an exponential function to model the response.

2.3.4. Summary

In summary, the relative contribution of ATP production via the aerobic pathway will increase as sprint duration increases and when the recovery time between sprints is limited. Endurance-trained individuals have been found to have faster $\dot{V}O_{2on}$ kinetics than untrained individuals. Faster $\dot{V}O_{2on}$ kinetics during a sprint could aid MPO, whereas faster $\dot{V}O_{2off}$ kinetics could enhance PCr resynthesis. Findings regarding the strength of the relationship between $\dot{V}O_2$ parameters and repeated-sprint performance have been mixed, but the protocols that have been used do not accurately reflect the demands of the Match Sprint competition. NIRS can be used as a measurement tool for the non-invasive assessment of oxygenation status at the muscle itself. When using NIRS during exercise, the confounding influence of blood flow on tHb and O_2Hb means that it may be preferable to restrict the analysis to TSI and/or HHb. A one-phase exponential function can be used to model the data, with the time constant providing a measure of the rate of deoxygenation/reoxygenation. However, individuals with a high $\dot{V}O_{2max}$ may experience a delay before restoration occurs, meaning that it may be necessary to consider the MRT, as well as τ. One further consideration when measuring muscle reoxygenation using NIRS, is that the participants are often required to maintain the measurement site in a fixed position (Nagasawa, 2013), which may then not accurately reflect real world practice.

2.4. RECOVERY ACTIVITY – CURRENT PRACTICE

"If you were at a competition and you had a relative short period of time, for example 10 – 30 minutes between two heats, what would you do in that time-period?" Primary Researcher

"I would judge it on the feeling in my legs. Essentially, I would flush out any lactate on the rollers, sit down and then spin the legs for 3 – 4 mins before the next ride with no effort. If I felt the race was short and there is little to no lactic build up, I would sit down straight away and just do the 3 – 4 mins before the next race. Things are often very basic and on feel, rightly or wrongly. Strange for how much research is done on various things" (Elite Track Cyclist, personal communication, 12th September, 2019).

2.4.1. Active/Passive Recovery: Effects on Lactate Clearance

The removal of lactate in the period between races was a recovery objective given by the elite track cyclist. Whilst the role that lactate and/or acidosis play in fatigue and performance/performance recovery continues to be debated (see sections 2.2.2.6 and 2.2.2.7.), the comment provided by the athlete demonstrates the importance that is still placed on lactate removal. An active recovery would enhance blood flow, whereas a passive recovery could result in a pooling of blood in the working muscles (Bogdanis et al., 1995). Newman et al. (1937) first assessed whether performing an active recovery at increasing exercise intensities would alter the rate of blood lactate clearance. For two of the three participants, lactate clearance rates were found to increase with the intensity of the recovery exercise. However, for the third participant, a decrease in the rate of lactate removal was noted at the highest intensity tested (Newman et al., 1937). The authors, therefore, concluded that the lactate removal rate increases with exercise intensity up until a critical threshold (Newman et al., 1937). Support has subsequently been found for the notion of a critical exercise-intensity for lactate clearance (Davies et al., 1970; Hermansen & Stensvold, 1972), but the optimal intensity has been debated (Baldari et al., 2004; Baldari et al., 2005; Belcastro & Bonen, 1975; Davies et al., 1970; Devlin et al., 2014; Dodd et al., 1984; Gmada et al., 2005; Greenwood et al., 2008; Hermansen & Stensvold, 1972; McLellan & Skinner, 1982; Menzies et al., 2010).

The intensity of the recovery exercise has frequently been set as a percentage of $\dot{V}O_{2max}$ (Belcastro & Bonen, 1975; Davies et al., 1970; Dodd et al., 1984; Hermanssen & Stensvold, 1972; Stamford et al., 1981). Belcastro and Bonen (1975) suggested that 32% $\dot{V}O_{2max}$ was optimal, whereas Davies et al. (1970) suggested that an intensity of 40% $\dot{V}O_{2max}$ was ideal, and Hermanssen and Stensvold (1972) suggested that an even greater exercise intensity, 60 – 70% $\dot{V}O_{2max}$, would be preferred. When exercising at an intensity that is greater than the first rise in blood lactate concentration (LT1), further production of lactate would be expected (McLellan & Skinner, 1982).

The relative percentage of $\dot{V}O_{2max}$ where LT1 occurs is affected by training status (Jones & Carter, 2000). It would, therefore, appear preferable to investigate recovery intensities relative to LT1, as opposed to $\dot{V}O_{2max}$ (McLellan & Skinner, 1982).

An additional consideration that was raised by Stamford et al. (1981), related to the expected lactate production for the intensity of exercise that was performed, as opposed to a return to a resting baseline. In the study by Stamford et al. (1981), lactate concentration was measured on three occasions (low-intensity cycling (40% $\dot{V}O_{2max}$), high-intensity cycling (70% $\dot{V}O_{2max}$), and passive rest) for 40 minutes after 40 s of maximal cycling. Lactate recovery kinetics were modelled using an exponential function, selecting the peak lactate concentration after exercise (range 12.8 – 13.3 mmol·L^{-1}) as the start-point and the asymptotic value as either being the resting value, or the expected asymptotic value (range 0.9 – 3.5 mmol·L^{-1}) considering the intensity of exercise being performed. When the asymptotic value was set to the resting concentration, the $t_{1/2}$ of blood lactate clearance was found to be lowest (fastest recovery) in the low-intensity condition. However, when the asymptotic value was adjusted to the expected end lactate concentration, the difference between the active conditions was not significant, suggesting that the rate constant for lactate clearance may not be affected by exercise intensity. The rationale for performing an active recovery may then need to be considered. It would seem reasonable to assume that in most cases, the objective would be to optimise the return of lactate to a resting concentration, or at least close to a resting concentration, in which case exercising at an intensity that is at, or beneath, LT1, would seem sensible.

Lactate clearance rates have been evaluated at multiple recovery intensities that were just above, at, or below, LT1 (Devlin et al., 2014; Menzies et al., 2010; McLellan & Skinner, 1982). McLellan and Skinner (1982) investigated lactate clearance during six recovery conditions (-30%, -20%, -10%, 0%, and +10% of LT1, as well as passive recovery) following 10 minutes of cycling at ~ 90% $\dot{V}O_{2max}$. Both linear and quadratic functions were applied to the data and the $t_{1/2}$ of lactate recovery was calculated assuming a baseline concentration of 1.0 mmol·L^{-1}. Lactate clearance was found to be significantly faster in all active conditions than during passive recovery. Exercise at an intensity that was above LT1 was also less effective for lactate clearance than the -20%, -10%, and 0% conditions. However, the difference in the lactate clearance rate between these three intensities was not significant. The functions used to model the data (linear and quadratic) were reported to provide a good fit, although a linear function would not account for the initial rise in blood lactate concentration that would be expected following the cessation of intense exercise (Freund & Zoulomian, 1981; Fujitsuka et al., 1982; Merrells et al., 2019; Withers et al., 1991), and neither function would allow for an eventual steady-state. Beneke et al. (2010) have proposed a two-phase exponential function to model lactate recovery that would account for an elevated post-warm-up state, a post-sprint rise, and then a subsequent fall (see Equation 2.09). However, to-date, this function has

not been used to evaluate the optimal exercise intensity for lactate clearance, although others have used a one-phase exponential function to fit the data following the peak of the response (Devlin et al., 2014; Menzies et al., 2010; Stamford et al., 1981).

$$BLC(t) = \frac{A*k_1}{k_2-k_1} \times \left(e^{-k_1 t} - e^{-k_2 t}\right) + \left(BLC_{pre\ sprint} - BLC_{rest}\right) \times e^{-k_2 t} + BLC_{rest} \quad \text{(Equation 2.09)}$$

where *BLC(t)* represents the blood lactate concentration at any time-point, BLC_{rest} is the blood lactate concentration at rest, and $BLC_{pre\ sprint}$ is the concentration at the start of the sprint. *A* represents the approximate increase in lactate and k_1 is the appearance velocity constant, whereas k_2 is the disappearance velocity constant.

Following five minutes of running at 90% $\dot{V}O_{2max}$, Menzies et al. (2010) evaluated lactate clearance under six recovery conditions (passive, 40%, 60%, 80%, and 100% of LT1, as well as at a self-regulated intensity: 79 ± 5% LT1). Capillary blood samples were collected before and after the warm-up, immediately following the activity, and every four minutes after the activity until lactate concentration had returned to its baseline level. In addition to computing the first derivative and τ, the raw lactate concentration values were compared. Comparison of the raw data revealed that the higher exercise intensities (60 – 100% LT1) were more effective in clearing lactate than both the passive recovery and the lowest active recovery (40% LT1) conditions. When analysing the peak rate of clearance, as well as τ, the 60% condition was found to be less effective than the two higher intensities. Therefore, the authors concluded that an active recovery should be performed at an intensity that is at, or close to, LT1. However, Devlin et al. (2014) questioned the intensity of the fatiguing protocol that was used by Menzies et al. (2010). At the cessation of exercise, lactate concentration was reported by Menzies et al. (2010) to be 3.9 ± 0.3 $mmol \cdot L^{-1}$, a concentration that is not reflective of near maximal intensity exercise (Devlin et al., 2014). After a 30 s cycling sprint capillary blood lactate concentration has been reported to rise to values greater than 10 $mmol \cdot L^{-1}$ (Bogdanis et al., 1996a; Kirkpatrick & Burrus, 2020). Devlin et al. (2014), therefore, used a fatiguing protocol (two 30 s runs at 15 $km \cdot h^{-1}$ at a gradient of 20%) designed to generate a substantially greater end lactate concentration (11.5 ± 0.2 $mmol \cdot L^{-1}$). The subsequent recovery intensities, as well as the method used to analyse the data, were the same as those used by Menzies et al. (2010). When comparing lactate concentrations, the findings supported those of Menzies et al. (2010). Namely, all active conditions were preferable to passive recovery and the higher exercise intensities (60 – 100% LT1) were preferred to the 40% LT1 condition. However, for both τ and the first derivative, the 80% LT1 condition was found to be better than all other conditions (Devlin et al., 2014).

A final consideration concerning the use of an active recovery to enhance lactate removal, relates to whether the intensity of the recovery exercise should be constant. Stamford et al. (1981)

reported that the peak lactate concentration following 40 s of maximal cycling occurred earlier when a higher intensity of exercise was performed in the recovery period (70% $\dot{V}O_{2max}$ compared to 40% $\dot{V}O_{2max}$). Dodd et al. (1984) suggested that higher intensity exercise could facilitate a greater rate of blood flow to the muscles, resulting in an enhanced movement of lactate from the muscle to the blood, which could then be combined with sub-threshold exercise to facilitate an earlier peak, but limiting the further production of lactate thereafter. Research findings regarding the use of a combined recovery approach to enhance lactate removal are, however, limited and the outcomes have been contradictory (Dodd et al., 1984; Gmada et al., 2005). Therefore, at this time, more research would be required for a combined approach to be advocated. The benefits of performing an active recovery on subsequent performance must then also be considered.

2.4.2. Active/Passive Recovery: Effects on Performance

Findings regarding the effects of the recovery activity (active or passive) on subsequent performance have been mixed (Bogdanis et al., 1996a; Dupont et al., 2007; Kirkpatrick & Burrus, 2020; Lattier et al., 2004; Losnegard et al., 2015; McAinch et al., 2004; Mika et al., 2016; Siegler et al., 2006; Thiriet et al., 1993). From a sprint cycling perspective, following a 15 s sprint, Dupont et al. (2007) found that MPO and PPO were higher during a subsequent 30 s sprint when passive recovery was undertaken, although the duration between sprints was just 15 s. Bogdanis et al. (1996a), on the other hand, found that an active recovery resulted in a significantly greater restoration of MPO when two 30 s sprints were performed four minutes apart, whereas when 15 minutes separated two 30 s cycling sprints, Kirkpatrick and Burrus (2020) found no difference between the recovery conditions for any of the performance measures (absolute PPO, relative PPO, MPO, relative MPO, total work, and fatigue index). In the study by Kirkpatrick and Burrus (2020), the participants were, however, recreationally active women. Women may display faster recovery kinetics than men (Billaut & Smith, 2009; Laurent et al., 2010) (discussed further in section 2.7.5.). The authors also recommended that additional research should be conducted using sprinters or individuals that undertake power-based activities, as it was suggested that these individuals would be likely to produce higher power outputs, as well as being able to perform more consistently (Kirkpatrick & Burrus, 2020). No consistency measures were reported by Kirkpatrick and Burrus (2020), but Hopkins et al. (2001) stated that non-athletic women perform less reliably than non-athletic men and that athletes perform more consistently than non-athletes. The coefficient of variation for sprint cycling performance in healthy men has been reported to be 2.2% ± 1.3% for MPO and 3.3% ± 2.1% for PPO (Hebestreit et al., 1993), which is lower than the values that have typically been reported for physiological variables (6.0% – 37.3%) (Buchheit et al., 2011; Mann et al., 2014). Less variability in the performance measure would enhance statistical power (Humphreys & Drasgow, 1989).

2.4.3. Summary

In summary, an active recovery improves lactate clearance when compared to a passive recovery, with the recommended intensity of the recovery exercise being 80% LT1. The effects of performing an active recovery on sprint cycling performance are less clear, but a passive recovery may be preferable when the recovery period is very brief, whereas an active recovery may be preferred when the recovery period is slightly longer. That being said, when a 15-minute recovery period was examined, a recovery duration that may occur in the Match Sprint competition, no significant differences were found in any of the performance measures. More research was, however, recommended with strength- or power-trained individuals, who may be able to produce greater power outputs and may also be able to perform more consistently.

2.5. THE ROLE OF PACING AND EFFORT ON SPRINT CYCLING PERFORMANCE

Pacing, or pacing strategy, has been defined as the distribution of work or energy expenditure during an exercise task (Abbiss & Laursen, 2008). For sprint activities, an "all-out" pacing strategy is generally advocated (Abbiss & Laursen, 2008; Foster et al., 2004), although there is evidence to suggest that sprint and repeated-sprint performances may not actually be truly maximal from the start to the finish (Billaut et al., 2011; de Jong et al., 2015; Glaister et al., 2019; Wittekind et al., 2011). Effort has been defined as the amount of mental or physical energy given to a task (Abbiss et al., 2015), with potential motivation reflecting the maximum amount of effort that an individual is prepared to exert to satisfy a motive (Marcora, 2008). Whilst performance may not always be a direct representation of effort, if, for example, an individual exerts a 60 N force on one occasion during a hand-grip task, and then, with all else being equal, on another occasion exerts a force of 120 N, the effort provided would have increased (Brinkmann et al., 2021). Similarly, if an individual produces an MPO of 600 W during one cycling sprint and 700 W during another, effort would again appear to have increased. In sports competitions, at the elite level, incentives, such as monetary rewards or social recognition, could influence effort mobilisation (Brinkmann et al., 2021). Only a limited amount of research has, however, investigated the effects of competition on effort mobilisation during sprint cycling activities.

2.5.1. Pacing and Effort during Sprint Exercise

Participants in sprint and repeated-sprint studies may be asked to go all-out from the start and to provide a maximal effort throughout (Billaut et al., 2011; Wittekind et al., 2011). However, in-

spite of these instructions, evidence of pacing, or of sub-maximal effort, has been found to exist (Billaut et al., 2011; de Jong et al., 2015; Glaister et al., 2019; Wittekind et al., 2011). During single sprints, Wittekind et al. (2011) found that MPO was greater during both 5 s and 15 s sprints than over the same time-periods in a 45 s sprint. The PPO achieved during the 45 s sprint was also significantly lower than the PPO recorded during both the 5 s and the 15 s sprints (see Table 2.15). Likewise, Glaister et al. (2019) found that PPO was significantly lower during a 30 s sprint than during a 10 s sprint and de Jong et al. (2015) found that PPO during a 250 m time-trial (completion time: 21.4 ± 1.8 s) was lower than the PPO that was recorded during a 10 s test, where the participants were solely required to produce the highest power output possible. It would, therefore, appear that sprint duration could affect the pacing strategy employed.

Table 2.15 Peak power output, mean power output, and the time to peak power output for sprints ranging from 5 s to 45 s (Wittekind et al., 2011). Data are displayed as mean ± standard deviation.

Sprint Duration (s)	Mean Power Output over 5 s (W)	Mean Power Output over 15 s (W)	Peak Power Output (W)	Time to Peak Power Output (s)
5	866 ± 127*	-	1021 ± 121*	2.9 ± 1.1
15	865 ± 154	759 ± 92*	1004 ± 146*	3.5 ± 0.8
30	831 ± 126	744 ± 74	961 ± 131	3.3 ± 0.8
45	755 ± 122	710 ± 64	902 ± 104	3.6 ± 1.6

Note: * denotes a significant difference ($p < 0.01$) from the 45 s trial.

During a repeated-sprint task (ten 6 s sprints with 24 s of recovery between sprints), Billaut et al. (2011) investigated whether providing different information about the requirements of the task would affect performance. On one occasion the participants were correctly informed that they would perform ten sprints (control condition). On another occasion, the participants were asked to perform five sprints. However, on completion of the first five sprints, the participants were advised that they were required to perform another five sprints (deception condition), and in the final condition, the participants were asked to continue performing 6 s sprints with 24 s of recovery until instructed to stop (unknown condition). In the unknown condition, the participants were stopped after they had performed ten sprints. PPO and total work during the first sprint, as well as the total work during the first five sprints, were greater in the deception condition than in the control or unknown conditions. Therefore, the information provided about the requirements of the task affected the effort provided.

The effect of deception, via the provision of inaccurate feedback, has also been assessed on exercise performance (Ansdell et al., 2018; Ducrocq et al., 2017; Jones et al., 2015; Shei et al., 2016; Stone et al., 2012; Stone et al., 2017; Waldron et al., 2015; Wilson et al., 2012). In most studies, the participants have viewed two avatars (current and "baseline" performances) on a screen that was positioned in front of the cycle ergometer. The participants were then either asked to try to better their

previous time-trial time performance (Ansdell et al., 2018) or they were advised that the study was interested in performance reliability (Shei et al., 2016; Stone et al., 2012; Stone et al., 2017; Waldron et al., 2015; Wilson et al., 2012). In reality, the pacing avatar either provided an accurate representation of their baseline performance or the power output/speed was manipulated (positively or negatively). Ansdell et al. (2018) found that an accurately paced avatar did not alter 4 km cycling time-trial performance, but when the avatar represented a 2% increase in power output, performance was enhanced. In contrast, Jones et al. (2015) found that accurate feedback did improve performance, but deception (a 2% improvement in 16.1 km time-trial duration) provided no additional benefits. Manipulations in speed/time and power output are, however, different. During steady-state exercise, a 1% change in speed may equate to a 3% increase in power output (Flyger, 2008; Stone et al., 2017). Stone et al. (2017) found that a 2% increase in power output improved 4 km time-trial performance, but a 5% increase had no effect. An upper limit may, therefore, exist to the magnitude of deception that can be provided (Stone et al., 2017), with the deception provided by Jones et al. (2015) possibly being too large to promote an additional effect on performance.

Avatars, and manipulation of the information provided to the participants, have also been included in competition studies (Corbett et al., 2012; Williams et al., 2015; Wood et al., 2020). Both Williams et al. (2015) and Wood et al. (2020) advised their participants that the second avatar that was on the screen represented the performance of a matched participant. In reality, and similar to the previously described deception studies, the recorded avatar either represented the previous best performance by the participant (Williams et al., 2015) or their baseline performance (Wood et al., 2020). Corbett et al. (2012) applied greater creativity in their study design by placing a dividing screen between two cycle ergometers in the laboratory, advising the participants that they were not permitted to see their opponent to reduce any potential confounding effects from perceptual cues and interpersonal rivalries. One of the research team was then setup on the second ergometer. In reality, the second avatar on the screen again represented the previous best performance by the participant. The 2 km distance was completed significantly faster (competition: 184.6 ± 6.2 s; control: 188.3 ± 9.5 s) in the head-to-head competition condition. Corbett et al. (2012) also reported that the pacing profile indicated that the participants initially matched the performance of the avatar, but then went faster over the final 1 km, suggesting that they increased their effort during the second half of the task. In the study by Williams et al. (2015), power output and 16.1 km completion time were again improved under competition conditions, although on this occasion power output was improved throughout the trial. Therefore, the pacing strategy was not altered, but effort increased throughout the performance. In contrast to the improvements in performance that were found by Corbett et al. (2012) and Williams et al. (2015), during the shortest cycling time-trial distance that has been examined, a 1 km time-trial, Wood et al. (2020) found that competition did not improve performance or affect pacing. A 1 km distance does reflect a sprint track cycling discipline (the Kilo). It was, however,

notable that verbal encouragement was not included in any of the aforementioned simulated cycling competition studies. The exclusion of verbal encouragement was to help standardise the laboratory conditions and to focus the research findings on head-to-head competition, although an encouraging crowd may well be present during a sports competition, which could add to the motivational response and thus the effort provided.

2.5.2. Verbal Encouragement and Social Facilitation

Verbal encouragement has typically been found to enhance exercise performance during strength, sprint, and endurance activities (Andreacci et al., 2002; Belkhira et al., 2018; Chitwood et al., 1997; Edwards et al., 2018; Engel et al., 2019; McNair et al., 1996). When two WAnTs were performed five minutes apart, MPO, average PPO, and absolute PPO were significantly higher when verbal encouragement was provided (Edwards et al., 2018), although it was somewhat surprising that the data were not evaluated considering each sprint in the two conditions (MPO was an average over both sprints and PPO was considered as the highest value over the two sprints, as well as the average of the highest values recorded in each sprint). When two WAnTs were performed four minutes apart, Bogdanis et al. (1996b) found a large negative correlation between performance during the first sprint (MPO over 10 s and PPO) and the recovery power output (sprint 2 MPO or PPO relative to sprint 1). Therefore, if power output was enhanced by verbal encouragement during the first sprint in the study by Edwards et al. (2018), greater levels of fatigue may have been elicited, which could potentially generate a more complex relationship between sprint repetition number and verbal encouragement. It should, however, be noted that the verbal encouragement provided by Edwards et al. (2018) was well controlled. The researchers also ensured that the same female provided the encouragement and that she was present in both the control and the experimental conditions.

The presence of other people could in itself affect task performance. When other people are present during a sports performance they could be involved with the activity. For example, they could be there to watch or to judge the performance. Alternatively, they could be performing the activity themselves (Landers & McCullagh, 1976). When the other people are performing the same activity, they could be in competition or they could be there independently. For example, other athletes that are cycling round the track during a training session or other people lifting weights at the same time in the gym. It is also possible that the other people that are present are not involved with the activity. For example, in an academic setting, other people could be conducting their own research in the laboratory. The effect of the presence of the various others on sports performance may, therefore, be complex. Additional factors that could affect performance include: the competency level of the individuals performing the task (Zajonc, 1965); the complexity of the task (van Meurs et al., 2022);

and the sex of both the observer and the participant (Jung et al., 2009). Van Meurs et al. (2022) suggested that there was a potential for cognitive overload or distraction during skill-based tasks, whereas an increase in readiness to act could improve performance during effort-based tasks. With regards to the sex of the participant and the observer, Jung et al. (2009) found that men produced a higher power output during an incremental exercise stress test on a cycle ergometer when a female doctor was present, compared to when the doctor was male, whereas in women, test performance was not affected by the sex of the doctor. Heinrich et al. (2021) also recently used the rare circumstance created by COVID-19 to examine biathlon performance with and without a crowd (data compared with the previous year when a crowd was present). In men, cross-country skiing time was improved with an audience, but performance of the coordination task worsened, with the opposite effect being observed in women (Heinrich et al., 2021).

2.5.3. Monetary Rewards

Monetary rewards are one of the most frequently used incentives that are incorporated into research studies investigating effort and task performance (Brinkman et al., 2021). Shi et al. (2021) found that a $15 reward enhanced isometric hand-grip performance (force production over three minutes) within a team task. Performance improved when the reward was split evenly between the team, but was further enhanced when the reward received by each participant was related to their individual performance (Shi et al., 2021). In contrast, monetary rewards were not found to enhance performance during cycling tasks of various distances/durations (Hulleman et al., 2007; Skorski et al., 2017). In the study by Hulleman et al. (2007) the participants were only required to improve on their previous best 1.5 km time-trial performance time by just 1 s to receive a $100 (U.S. dollars) reward. Only two of the seven participants won the reward, with the completion time not differing significantly between trials. Skorski et al. (2017) also found that the offer of a reward ($7 to $350 Australian dollars for the highest average power output relative to body-mass) did not improve cycling performance during 4 km and 20 km time-trials or 6- and 30-minute duration efforts. Hulleman et al. (2007) did, however, state that the size of the reward that was offered may not have been sufficient to motivate the participants, although the importance of a financial reward to the participants was not assessed. The effectiveness of any strategy aimed at increasing the effort provided by the participants should be evaluated and not assumed (McCormick et al., 2019).

2.5.4. Summary

In summary, potential motivation reflects the maximum amount of effort that a participant is prepared to exert to satisfy a motive. Task duration, knowledge of the demands of the task, performance feedback, and competition, could all affect the amount of effort that is provided by a participant during sprint activities, a consideration that may not have received sufficient research attention. It should not, however, be assumed that all individuals will respond to the incentives or encouragement provided in the same manner. The effectiveness of any strategy aimed at increasing effort should be evaluated. That being said, if sprint performance is positively affected by these motivational factors, it is possible that physiological changes led the participants to being in a heightened state of readiness to perform.

2.6. PSYCHOPHYSIOLOGY OF SPRINT CYCLING PERFORMANCE

The stress that is experienced by an athlete during a competition could be harmful to performance (Davies & Armstrong, 1989). However, an appropriate amount of stress could also enhance exercise performance (McCormick et al., 2019). From a physiological perspective, a small region in the temporal lobe, known as the amygdala (Ressler, 2010), processes emotional stimuli and determines the response (Thau et al., 2021). If it is deemed appropriate, a signal is sent to the hypothalamus, stimulating the sympathetic nervous system, resulting in the release of catecholamines, such as epinephrine, from the adrenal glands (Thau et al., 2021). If the perceived threat continues, the hypothalamic-pituitary adreno-cortical axis is then activated, ultimately resulting in the release of cortisol from the adrenal cortex (Thau et al., 2021). Therefore, the hypothalamic-pituitary adreno-cortical axis and the sympathetic nervous system work in a coordinated fashion, although the exact nature of this relationship is debated (Kivlighan & Granger, 2006). To assess sympathetic nervous system activity, high performance liquid chromatography can be used to measure epinephrine and norepinephrine (Granger et al., 2008). The collection, extraction, and separation processes are not, however, straightforward, requiring expensive laboratory equipment and highly skilled technical personnel to conduct the analysis (Granger et al., 2008). The complexity of these measurements also means that their use in research settings is limited (Ali & Nater, 2020). Consequently, scientists have searched for other physiological markers that may provide an indirect measure of sympathetic nervous system activity (Granger et al., 2008). Heart rate responses have been proposed as one such measure (Bosch et al., 2011), but it has also been suggested that the digestive enzyme, alpha amylase (AA), could provide a surrogate marker for autonomic activity (Díaz et al., 2012). Saliva sampling could, therefore, facilitate the simultaneous assessment of the hypothalamic-pituitary adreno-cortical axis,

via the measurement of cortisol concentration, and the activity of the sympathetic nervous system, via the assessment of AA activity and output (Bosch et al., 2011).

2.6.1. Saliva Stress Markers

Three major glands produce saliva. These are: the parotid gland; the submandibular gland; and the sublingual gland (Bosch et al., 2011). Several minor glands also exist in the submucosa underlying the lips, cheeks, and palate, which provide a substantial contribution to the protein content in saliva (Humphrey & Williamson, 2001). Saliva samples can either be produced passively or via stimulation (such as chewing). Specific glands can be targeted or whole saliva can be collected. An absorbent material can be used to gather the sample (cotton swab) or the saliva can be allowed to pool in the mouth and then be moved via the tongue, or by using gravity, into a collection device (Rohleder & Nater, 2009). One factor that is often overlooked, however, is whether the saliva flow-rate needs to be considered in the analysis (Strahler et al., 2017). For the assessment of salivary cortisol, it has been suggested that accounting for flow-rate is not necessary (Strahler et al., 2017), but when considering AA, it has been suggested that it is the amount of AA that is secreted per unit of time that relates to the sympathetic response (Bosch et al., 2011), meaning that measuring the activity may be too simplistic. Vasoconstriction of the arterioles supplying the salivary glands is also a sympathetic response, which may inhibit saliva flow-rate (Chicharro et al., 1998; Gatti & Palo, 2011). Therefore, AA activity may increase if saliva flow-rate decreased, but the quantity of AA produced remained the same. Whilst it should be noted that the importance of accounting for saliva flow-rate for the analysis of AA is ongoing (Strahler et al., 2017), the most recently published recommendations did state that until the debate is resolved, saliva flow-rate should be accounted for in the analysis (Strahler et al., 2017). However, when AA has been measured as a stress marker during a sports competition, none of the studies have accounted for flow-rate (Azarbayjani et al., 2011; Caprancia et al., 2012; Caprancia et al., 2017; Chennaoui et al., 2016; Chiodo et al., 2011; Dehghan et al., 2019; De Pero et al., 2021; Kivlighan & Granger, 2006; Sinnott-O'Connor et al., 2018; Trochimiak & Hübner-Woźniak, 2014).

The stress response to competition was first investigated via the assessment of both AA and cortisol by Kivlighan and Granger (2006). Saliva samples were collected before an indoor rowing competition, as well as 20 and 40 minutes after the competition and at the same time-points on a rest day (Kivlighan & Granger, 2006). AA activity increased by 156% following the competition, with cortisol concentration increasing by 87% over the same time-period. It was, however, surprising that at the pre-competition time-point, AA levels were greater on the rest day, although this effect was only apparent in women (Kivlighan & Granger, 2006). De Pero et al. (2021) also reported an intriguing outcome at the pre-competition time-point, namely a significantly higher cortisol concentration on a training day, when compared to either a rest day or prior to a competition. The

authors suggested that this may have been the result of the training day occurring shortly after an intense training camp (De Pero et al., 2021). Vigorous physical activity has been identified as a confounding variable when measuring the stress analytes found in saliva (Strahler et al., 2017). Other considerations include: the time of day that the measurements are taken; smoking; use of medication; cleaning teeth; recent dental treatments; age; somatic diseases; as well as food and caffeine intake (Strahler et al., 2017). A full list of the current recommendations for saliva sampling is displayed in Table 2.16.

Table 2.16 Recommendations for the collection of saliva samples (Strahler et al., 2017).

Variable	Level	Recommendation
Age	Basic	• Assess age • Statistically control for age if it is associated with the outcome • Use pre-defined age restrictions
	Advanced	• Assess biological age - use a combination of questionnaires and medical examinations including hormonal assessment, e.g. puberty (Tanner stage) • Statistically control for biological age if it is associated with the outcome
Sex	Basic	• Focus on one sex • Statistically control for sex if it is associated with the outcome • Assess menstrual cycle, hormonal treatment (hormonal contraceptives, sex steroid treatment), pregnancy/breastfeeding, menopause (using self-report measures); statistically control for these factors if they are associated with the outcome
	Advanced	• Assess menstrual cycle phase using a combination of self-report and objective measures: determine sex hormones, such as progesterone and oestrogen in urine; statistically control for menstrual cycle phase if it is associated with the outcome • For acute stress reactivity studies, assess menstrual cycle phase using objective measures - exclude ovulation using ovulation test
Somatic health	Basic	• Assess acute/chronic somatic conditions and related regular medication, recent dental treatment (within last 2 weeks) using self-report measures; statistically control for these factors if they are associated with the outcome. Otherwise exclude
	Advanced	• Assess acute/chronic somatic conditions by a thorough medical screening (medical history, physical examination including anthropometric measurements, laboratory blood diagnostics); exclude these factors or statistically control for these factors if they are associated with the outcome
Medication	Basic	• Assess recent inoculation/vaccination, acute medication (using self-report measures); exclude inoculation/vaccination (depending on incubation time), acute medication (depending on active agent's half-life period or statistically control for these factors if they are associated with the outcome
	Advanced	• Assess acute medication using objective measures: determine concentrations of active agents or their metabolites in blood or urine; exclude acute medication or statistically control for these factors if they are associated with the outcome
Smoking	Basic	• Assess current smoking status (i.e., non-smokers vs smokers) (using

		self-report measures); statistically control for smoking status if it is associated with the outcome
		• Assess current smoker status (using self-report measures); exclude smokers (> 5 cigarettes/week)
	Advanced	• Assess recent smoking (using objective measures, such as determination of carbon monoxide in breath to verify smoking abstinence); exclude recent smoking
Food and Drink	Basic	• Assess food/drink consumption, chewing gum within last hour (using self-report measures: use broad categories, e.g., nothing vs. snack vs major meal, nothing vs water vs juice vs coffee vs alcohol); statistically control for these factors if they are associated with the outcome. Otherwise exclude samples
		• Instruct participants to avoid food, chewing gum, acidic/caffeinated beverages up to 1 h before saliva sampling (especially in the morning)
	Advanced	• Assess nutrition behaviour and dietary habits (using self-report measures: i.e. vegetarianism), statistically control for these factors if they are associated with the outcome
Alcohol	Basic	• Assess alcohol consumption within last 24 h (using self-report measures: specify if, when, what, and how much alcohol was consumed); statistically control for alcohol consumption if it is associated with the outcome. Exclude alcohol consumption within last 24 hours
		• Instruct participants to abstain from alcohol for at least 24 h prior to and at the time of assessment period (note: not applicable to momentary assessment studies since restrictions of everyday life routine should be avoided)
		• Assess regular heavy alcohol consumption (≥15 and ≥8 drinks per week for men and women, respectively)/alcoholism. Exclude these factors
	Advanced	• Assess recent alcohol consumption (using objective measures such as determination of ethanol (metabolites) in breath or blood to verify alcohol abstinence). Exclude recent alcohol consumption
Physical activity and fitness	Basic	• Assess physical activity levels 1 hour before sampling (use broad categories, e.g. vigorous vs moderate vs light) (using self-report measures); statistically control for physical activity if it is associated with the outcome. Exclude those saliva samples possibly confounded by vigorous physical activity
		• Instruct participants to avoid exercise or vigorous physical activity within last 24 h prior to assessment period
	Advanced	• Assess physical activity (using objective measures such as actigraphy); statistically control for physical activity if it is associated with outcome
		• Assess physical fitness (using a combination of self-report and objective measures, such as questionnaires, fitness test); statistically control for physical fitness if it is associated with the outcome
Sleep	Basic	• Assess time of awakening and daily napping within last hour (using self-report measures); statistically control for these factors if they are associated with the outcome
		• Assess night shifts (within last 4 weeks and during time of assessment) and jetlag due to recent international flights through different time zones (within last 2 weeks); exclude these factors
	Advanced	• Assess time of awakening (using objective measures, such as

	polysomnography or actigraphy); statistically control for time of awakening if it is associated with the outcome • Assess sleep habits (using self-report measures, such as sleep duration, get-up time, bedtime, frequency/length of daily naps); statistically control for these factors if they are associated with the outcome

With regards to the timing of the measurements, both AA and cortisol have distinct diurnal patterns. Cortisol concentration will typically peak within 30 minutes of waking and then gradually decrease throughout the day, whereas AA activity will be at its lowest in the morning, increasing over the day (see Figure 2.25) (Nater et al., 2007). With regards to taking measurements around a sporting competition, timings were initially based on knowledge about cortisol activity (Kivlighan & Granger, 2006). Subsequent research has, however, typically found that AA will peak immediately after a competition, with a return to baseline, or a level lower than baseline, within the subsequent 20 – 30 minutes (Caprancia et al., 2012; Caprancia et al., 2017; Chiodo et al., 2011; De Pero et al., 2021; Díaz et al., 2012). In contrast, cortisol concentration has been found to peak 30 minutes after a competition (Caprancia et al., 2012; Chiodo et al., 2011). Kivlighan and Granger (2006) may, therefore, not have recorded the peak activity of AA. An additional limitation in the study by Kivlighan and Granger (2006) was that the measurements on the control day were only time-matched. In fact, a number of studies that have investigated AA activity and cortisol concentration during a sports competition have only assessed the time course of the analytes on the day of the competition (Azarbayjani et al., 2011; Caprancia et al., 2012; Chiodo et al., 2011; Trochimiak & Hübner-Woźniak, 2014). If an exercise matched control was not included, and differences in the analytes were only found after the competition, it means that the effect of competition cannot be differentiated from the exercise undertaken. Only the studies by De Pero et al. (2021) and Díaz et al. (2012) have matched the sampling time and exercise undertaken in their control conditions.

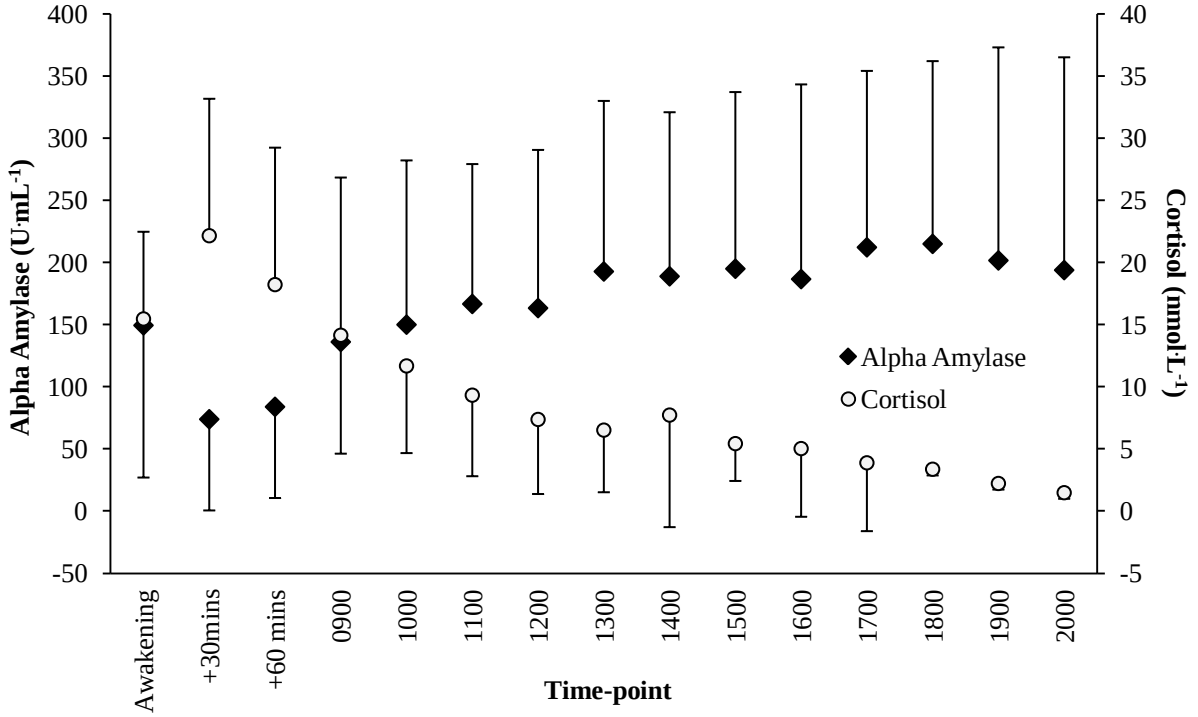

Figure 2.25 Salivary alpha amylase activity and cortisol concentration over a typical weekday for university students. Data points represent the mean and error bars the standard deviation (Nater et al., 2007).

In the study by Díaz et al. (2012), AA activity was assessed on waking, 30 and 60 minutes after waking, and then later in the day, just prior to the warm-up (16.00), and 5, 20, and 60 minutes after the activity (swimming/swimming competition). AA activity was found to be greater on the competition day at the pre-warm-up and five minutes post-competition time-points (see Figure 2.26), suggesting that an alteration in the stress response was only apparent just prior to and immediately after the competition and that by 20 minutes after the competition, measurable differences were no longer apparent. De Pero et al. (2021) assessed both AA activity and cortisol concentration before, during, and after a Team Gymnastics event. In addition to the competition day, activity levels were assessed on a time and exercise matched training day and on a time matched rest day. AA activity was found to be greater following the final event on the training day than at the pre-exercise time-point, suggesting that exercise alone increased AA activity. When compared to the training day, AA activity was greater at all time-points on the day of the competition (see Figure 2.27) (De Pero et al., 2021), meaning that competition evoked a stress response in anticipation of the activity ahead, which then added to the stress response caused by exercise. In contrast, cortisol concentration only differed between conditions at the pre-competition time-point (see Figure 2.28), leading the authors to suggest that AA activity could provide a more sensitive marker for evaluating psychophysiological stress during a competition (De Pero et al., 2021).

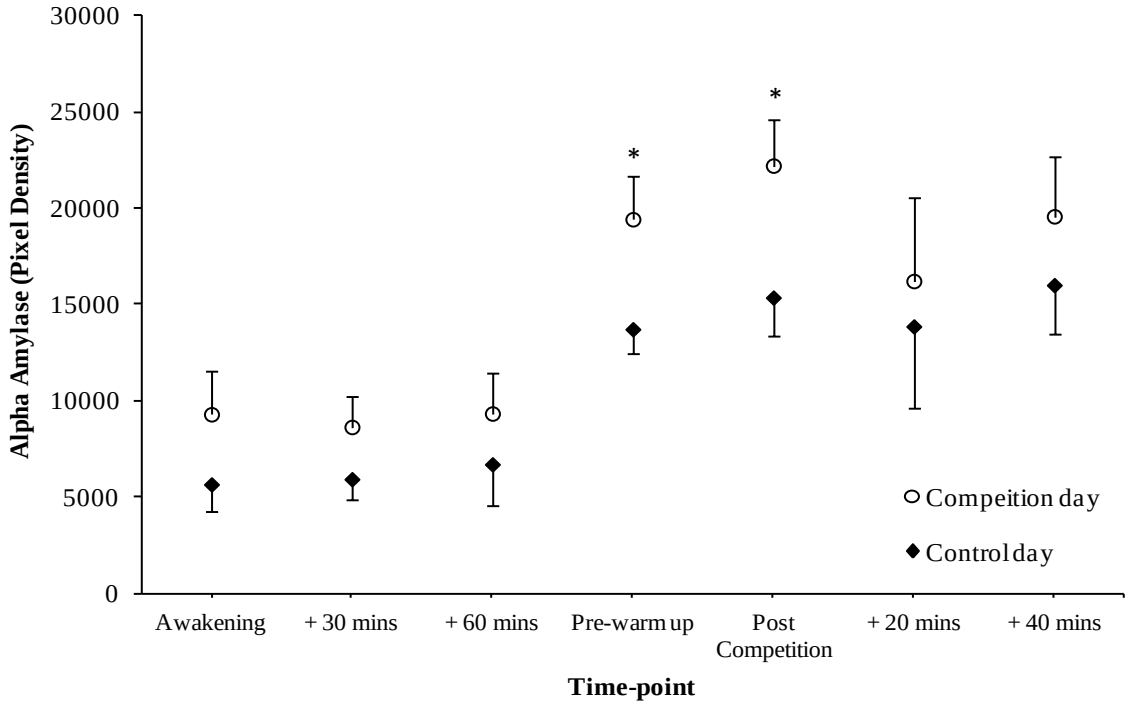

Figure 2.26 Alpha amylase activity on the day of a competition and on a time and exercise matched control day. Data points represent the mean and error bars the standard deviation. Note: * denotes a significant ($p < 0.01$) difference between conditions (Díaz et al., 2012).

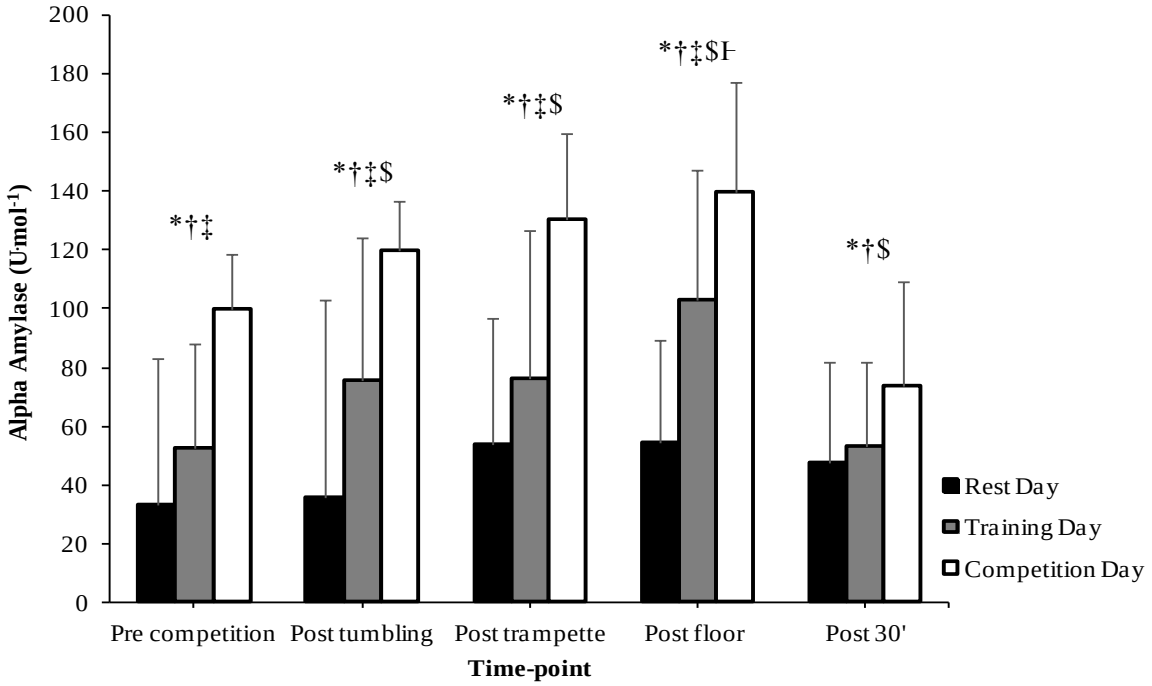

Figure 2.27 Alpha amylase activity on the day of a competition, on a time and exercise matched training day, and on a time matched rest day. Bars represent the mean and error bars the standard deviation. Note: between condition significant differences ($p < 0.05$) are denoted by: * for competition and rest days; † for competition and training days; ‡ for training and rest days. $ denotes a significant ($p < 0.05$) difference from the pre-competition time-point on the competition day and Ⱶ denotes a significant difference from the pre-competition time-point on the training day (De Pero et al., 2021).

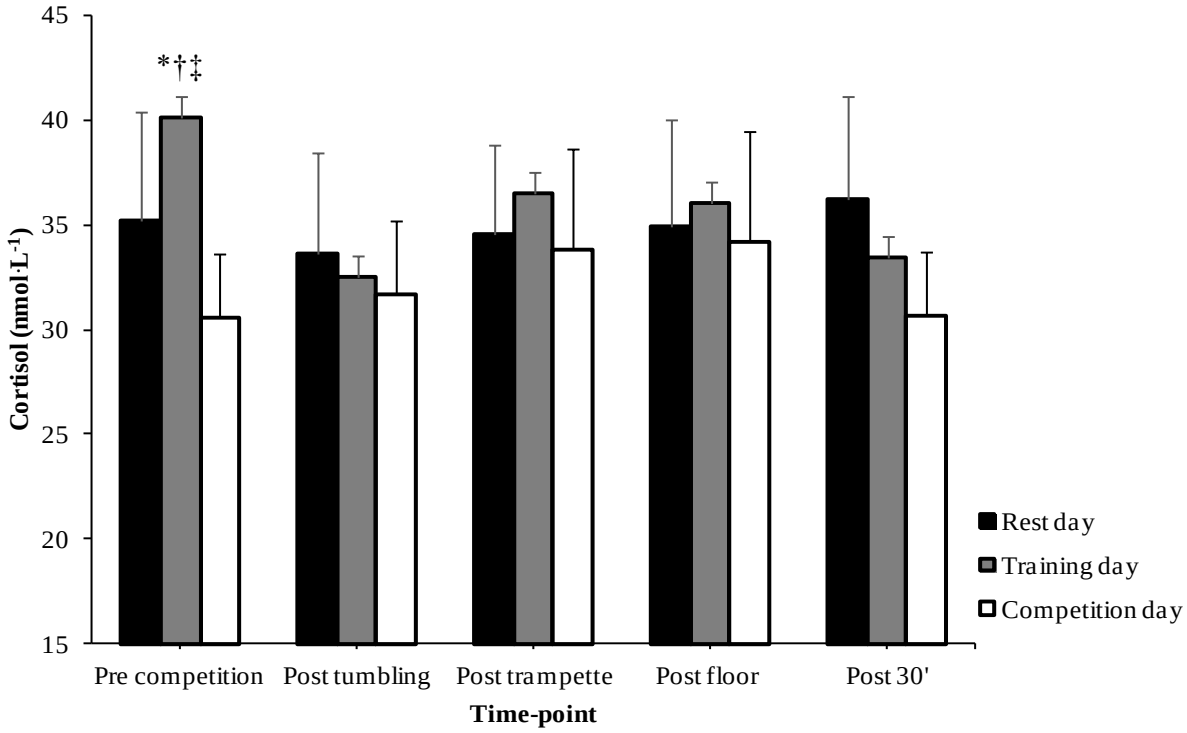

Figure 2.28 Cortisol concentration on the day of a competition, on a time and exercise matched training day, and on a time matched rest day. Bars represent the mean and error bars the standard deviation. Note: between condition significant differences ($p < 0.05$) are denoted by: * for competition and rest days; † for competition and training days; ‡ for training and rest days (De Pero et al., 2021).

2.6.2. Heart Rate Variability

The basic pattern of electrical activity captured by an electrocardiogram (ECG) is displayed in Figure 2.29 (Ashley & Niebauer, 2004). The initial P wave represents atrial depolarisation. The interval between the initiation of the P wave and the start of the QRS complex forms the PR interval (Ashley & Niebauer, 2004). Within the QRS complex, the Q wave corresponds to depolarisation of the interventricular septum, the R wave represents the depolarisation of the main mass of the ventricles, and the S wave signifies the final depolarisation of the ventricles (Ashley & Niebauer, 2004). The time interval between the QRS complex and the T wave is termed the ST segment, with the T wave representing ventricular repolarisation (Ashley & Niebauer, 2004). Heart rate variability (HRV) reflects the beat-to-beat fluctuation in heart rate (Lipponen & Taravainen, 2019). HRV is frequently used to evaluate the function of the autonomic nervous system (Lipponen & Taravainen, 2019) and is typically quantified from the ECG trace by calculating the variability in the duration of the R-R intervals (peak of one R wave to the peak of the next) (see Figure 2.30). Some heart rate monitors can also record R-R intervals and the low cost of these devices makes them easily accessible to researchers and practitioners, with Polar Electro Oy (Kempele, Finland) being one of the most

established brands (Hernández-Vicente et al., 2021). When compared to an ECG recording, both the Polar V800 (Giles et al., 2016; Hernández-Vicente, 2021) and the Polar RS800 (Tsitoglou et al., 2018) have been found to provide valid HRV recordings.

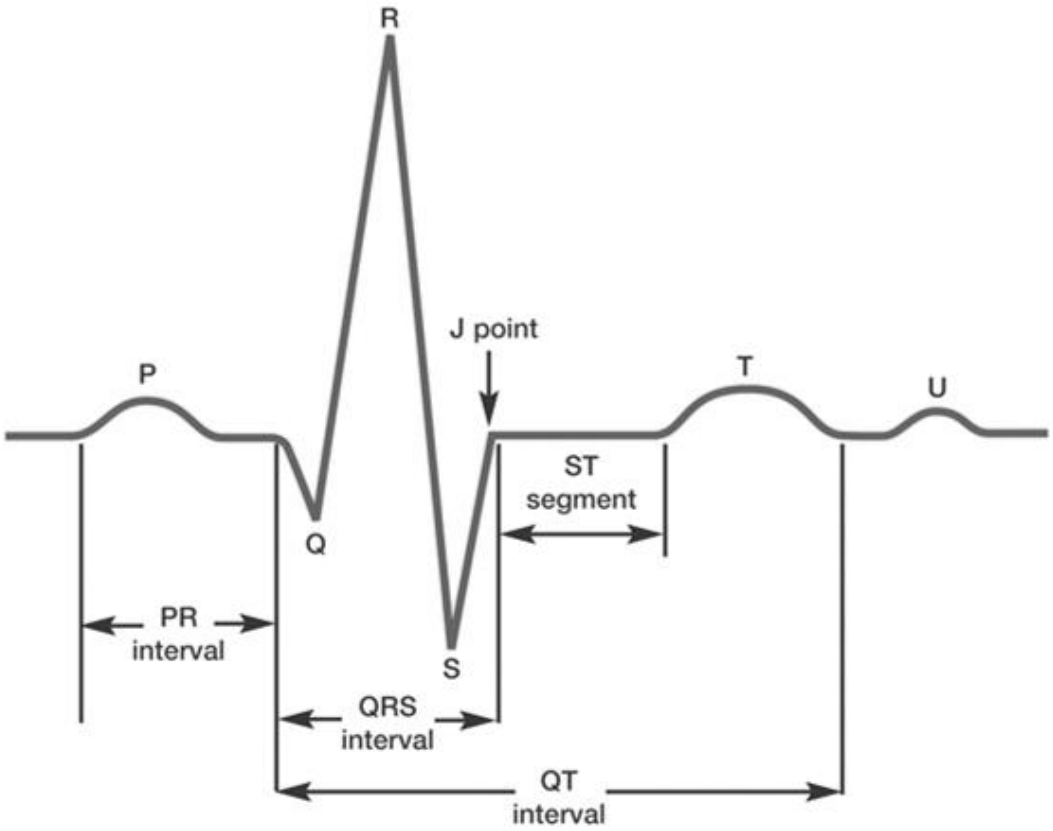

Figure 2.29 Waveforms captured using an electrocardiogram machine (Ashley & Niebauer, 2004).

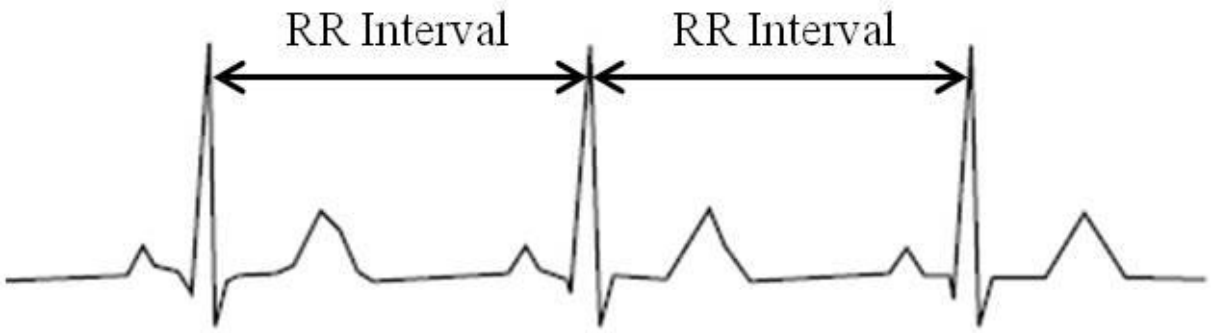

Figure 2.30 R-wave to R-wave intervals used for the calculation of heart rate variability (Aubert et al., 2003).

Body position and the duration of the recording must also be considered when measuring HRV (Shaffer & Ginsberg, 2017). Lying (D'Ascenzi et al., 2014; Edmonds et al., 2013; Goulopolou et al., 2006; Millar et al., 2009; Morales et al., 2013; Souza et al., 2019), sitting (Barak et al., 2014; Mateo et al., 2012; Murray & Raedeke, 2008), and standing (Edmonds et al., 2013) positions have all been adopted, but as body position can affect the measurement, consistency in the recording position is important (Shaffer & Ginsberg, 2017). With regards to the duration of the recording, historically, HRV was either assessed using long-term (≥ 24 hours) or short-term (5 minutes) measurements. The use of ultra-short term (< 5 minutes) measurements is also nowadays not uncommon and is appealing for both researchers and practical adherence from athletes (Shaffer & Ginsberg, 2017). Shaffer et al. (2020) have provided guidelines for the minimum recording duration that is required for a valid measurement of a number of HRV indices (see Table 2.17). In fact, in total, as many as 115 HRV metrics have been reported in the literature (Smith et al., 2013). There are also numerous software packages available to the researcher to process the data. These include: ARTiiFACT; gHRV; HRVAnalysis; KARDIA; Kubios (basic and premium); rHRV; and SinusCor.

Table 2.17 Minimum recording period for heart rate variability indices (Shaffer et al., 2020).

Minimum recording period	HRV index
10 s	HR
60 s	pNN50, NN50, RMSSD, SDNN
90 s	TINN, LF power, SD1, SD2
120 s	HRV triangular index, DFAα1
180 s	LFnu, HF power, HFnu, LF/HF, DFAα2, DET, SampEn
240 s	ShanEn

Note: HRV denotes heart rate variability, HR is heart rate, pNN50 is the percentage of successive R-R intervals that differ by more than 50 ms, NN50 is the number of successive R-R intervals that differ by more than 50 ms, RMSSD is the root mean square of successive differences, SDNN is the standard deviation of normal-to-normal beats, TINN is the baseline width of the R-R interval histogram, LF is low frequency, SD1 is the Poincaré plot standard deviation perpendicular to the line of identity, SD2 is the Poincaré plot standard deviation along the line of identity, DFA is detrended fluctuation analysis, α1 describes short-term fluctuations, α2 describes long-term fluctuations, HF is high frequency, DET is recurrence plot analysis determinism, SampEn is sample entrophy, ShanEn is Shanon entrophy.

HRV data processing must begin by first scanning the data for any irregularities, as even single distortions to the signal could significantly alter the outcome (Lippman et al., 1994). Lipponen and Tarvainen (2019) have provided a detailed description of the artefact detection algorithm that is implemented within the Kubios software package, although some inconsistencies within the text create uncertainty about the procedure that was used. Altini et al. (2016) applied a more simplistic approach, identifying any R-R intervals that differed in duration by more than 20% to the previous interval, as being an irregularity, although Plews et al. (2017) suggested that this approach may result in over-correcting data in individuals that possess a high degree of variability. Instead, Plews et al.

(2017) proposed the use of two filters. First, a 75% threshold for the interval-to-interval difference is applied. Next, any R-R intervals that are outside of a duration that is 25% less than the first quartile or 25% greater than the third quartile are flagged as erroneous (Plews et al., 2017). Following the identification of data artefacts, deletion, linear interpolation, cubic spline interpolation, and non-linear predictive interpolation, can be used to correct the data (Lippman et al., 1994). Having introduced simulated premature ventricular depolarisation to five-minute ECG recordings, Lippman et al. (1994) found that deletion and non-linear predictive interpolation were preferable to linear or cubic spline interpolation. Deletion simply requires the removal of the erroneous data, whereas non-linear predictive interpolation involves scanning artefact free R-R sequences to find the best matching sequence, that is locally similar to the artefact segment, and then replacing the erroneous data (Peltola, 2012). The processed data can then be analysed using time domain, frequency domain, and non-linear indices (see Table 2.18) (Shaffer & Ginsberg, 2017).

The advantage of time-domain indices is that they are easy to compute and interpret (Aubert et al., 2003). The standard deviation of normal-to-normal beats (SDNN) and the root mean square of successive differences (RMSSD) provide two of the most commonly reported metrics. Non-linear indices can also be used to index the unpredictability of the time series (Shaffer & Ginsberg, 2017). A Poincaré plot is produced (see Figure 2.31) by plotting the duration of every R-R interval against the previous one. An ellipse can then be fitted and measurements, such as SD1 (the standard deviation perpendicular to the line of identity) and SD2 (the standard deviation along the line of identity), derived. Both RMSSD and SD1 assess parasympathetic nervous system activity (Shaffer & Ginsberg, 2017), whereas sympathetic and parasympathetic activity will contribute to the values that are computed for SDNN and SD2. Power spectral analysis, which has been likened to light refraction by a prism into its component parts (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996), is also commonly performed in HRV analysis, using either a Fast Fourier Transform or an Autoregressive Technique (Li et al., 2019). It should, however, be noted that these processes cannot be viewed interchangeably (Pichon et al., 2006).

Frequency bands are then defined as high frequency (HF) (0.15 – 0.40 Hz), low frequency (LF) (0.04 – 0.15 Hz), very-low frequency (VLF) (0.003 – 0.04 Hz), and ultra-low frequency (ULF) (≤ 0.003 Hz) (Shaffer & Ginsberg, 2017), although during shorter measurements, the VLF band ranges from 0 – 0.04 Hz, and in humans, spectral components are typically only interpreted for the HF and LF bands (Aubert et al., 2003). The HF band reflects parasympathetic activity (Shaffer & Ginsberg, 2017). Whilst an increase in sympathetic activity may result in a shift of the LF to HF balance (Malliani et al., 1991), the LF band is not a pure index of sympathetic activity. In fact, half of the variability may be due to parasympathetic activity (Shaffer & Ginsberg, 2017). It has, therefore,

been suggested that both LF power and the LF:HF ratio may be poor indicators of the sympathetic response (Shaffer & Ginsberg, 2017). Violations to the assumption of normality are also not uncommon and could complicate the statistical procedures used, meaning that it may be necessary to transform the data, with a log transformation frequently being employed (Millar et al., 2009). The normalisation of HF and LF power may also aid with making the distribution of the data closer to normal. Normalised HF and LF data provide values as a proportion or as a percentage, which on the face of it may make them easier to understand (Burr, 2007). However, interpretation of the normalised data is, in fact, complex, as an increase in one index could result when the power of that variable increases, decreases, or remains the same (Heathers, 2014). It is, therefore, recommended that the raw data values should also be reported and considered in the interpretation of the normalised indices (Heathers, 2014)

Table 2.18 Description of heart rate variability indices (Shaffer et al., 2020).

HRV Metric	Units	Description
Time domain		
Heart rate	bpm	Number of heart-beats per minute
HRV triangular index		Integral of the density of the R-R interval divided by its height
NN	ms	Average of normal-to-normal intervals
NN50	count	Number of successive R-R intervals that differ by more than 50 ms
pNN50	%	Percentage of successive R-R intervals that differ by more than 50 ms
RMSSD	ms	Root mean square of successive R-R interval differences
SDNN	ms	Standard deviation of normal-to-normal intervals
TINN		Baseline width of the R-R interval histogram
Frequency domain		
VLF	ms^2	Absolute power of the very-low frequency band
LF	ms^2	Absolute power of the low frequency band
LFnu	nu	Relative power of the low frequency band in normal units
HF	ms^2	Absolute power of the high frequency band
HFnu	nu	Relative power of the high frequency band in normal units
LF/HF	%	Ratio of low frequency to high frequency
Total	ms^2	Sum of absolute power in the bands
Non-linear		
ApEn		Approximate entropy
D_2		An estimate of the minimum number of variables required to construct a model of system dynamics
DET	%	Recurrence plot analysis determinism
DFα1		Detrended fluctuation analysis, describes short-term fluctuations
DFα2		Detrended fluctuation analysis, describes long-term fluctuations
REC	%	Recurrence rate
SampEn		Sample entropy, measures the regularity and complexity of a time series
SD1	ms	Poincaré plot standard deviation, perpendicular to the line of identity
SD2	ms	Poincaré plot standard deviation, along the line of identity
ShanEn		Shannon entropy, measures the average information in a time series

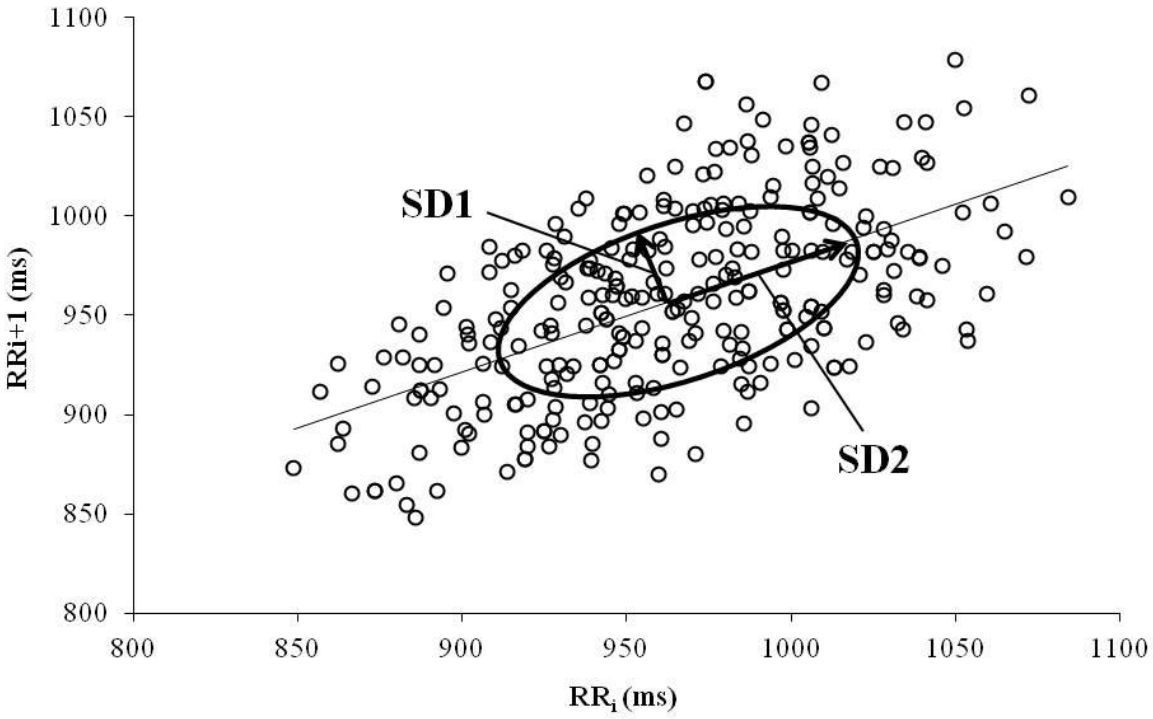

Figure 2.31 Example of a Poincaré plot, with variables SD1 and SD2 depicted (Guzik et al., 2007). Note: SD1 is the Poincaré plot standard deviation perpendicular to the line of identity, and SD2 is the Poincaré plot standard deviation along the line of identity.

In a sporting setting, HRV has been assessed following sprint and repeated-sprint cycling activities (Barak et al., 2014; Goulopolou et al., 2006; Millar et al., 2009; Niewiadauski et al., 2007), as well as during a sports competition (D'Ascenzi et al., 2014; Edmonds et al., 2013; Mateo et al., 2012; Morales et al., 2013; Murray & Raedeke, 2008; Souza et al., 2019). Following a single WAnT (Barak et al., 2014; Goulopolou et al., 2006; Millar et al., 2009) or multiple WAnTs (Millar et al., 2009; Niewiadauski et al., 2007), reductions in the time-domain indices SDNN (Millar et al., 2009; Niewiadauski et al., 2007) and RMSSD (Barak et al., 2014; Niewiadauski et al., 2007) have been reported. HF power (Niewiadauski et al., 2007), lnHF power (Goulopolou et al., 2006; Millar et al., 2009), and normalised HF power (HFnu) (Barak et al., 2014; Goulopolou et al., 2006; Millar et al., 2009) also decreased, as did LF power (Niewiadauski et al., 2007) and lnLF power (Barak et al., 2014; Goulopolou et al., 2006). Only normalised LF power (LFnu) and the LF:HF ratio increased (Goulopolou et al., 2006; Millar et al., 2009). Overall, these findings indicate an increase in physiological stress following exercise. With regards to a sports competition, greater levels of stress have also been found prior to the competition (Cervantes et al., 2009; D'Ascenzi et al., 2014; Edmonds et al., 2013; Mateo et al., 2012; Morales et al., 2013; Murray & Raedeke, 2008; Souza et al., 2019), although the findings for the specific HRV metrics have not been entirely consistent (see Table 2.19). In addition, and somewhat surprisingly, no studies have considered the HRV response during a competition, as the measurements have always been made just before the competition (Cervantes et

al., 2009; Mateo et al., 2012; Murray & Raedeke, 2008; Souza et al., 2019), on the morning of the competition (D'Ascenzi et al., 2014; Mateo et al., 2012; Morales et al., 2013), or on days before and after the competition (D'Ascenzi et al., 2014; Edmonds et al., 2013).

2.6.3. Summary

In summary, when competing, an athlete may be subject to additional stressors that are not experienced in training. These stressors could heighten the readiness of the athlete to perform and could elicit measurable changes in physiological state. The physiological stress response has two main components: a faster response, which concerns activation of the sympathetic adrenal medullary axis; and a slower response, which relates to the hypothalamic-pituitary adreno-cortical axis. Saliva sampling facilitates the assessment of both the hypothalamic-pituitary adreno-cortical axis, via the measurement of cortisol concentration, and the sympathetic adrenal medullary axis, via the measurement of AA activity. Both AA and cortisol have distinct diurnal patterns, with cortisol peaking in the morning and decreasing thereafter, whereas the opposite occurs for AA. During a sports competition, cortisol concentration may only increase after a competition, peaking 30 minutes after the competition, whereas AA activity may be elevated prior to a competition and be at its greatest immediately after the competition, returning to baseline within 30 minutes. Studies that have evaluated AA activity during a sports competition have not, however, accounted for saliva flow-rate. It has been suggested that saliva flow-rate should be considered in the analysis of AA, as it is the quantity of AA that is secreted per unit of time that provides a measure of the sympathetic response, not the activity. HRV provides an additional measure of autonomic activity, although no indices provide direct information about the sympathetic response. Nonetheless, evidence of parasympathetic withdrawal, or at least a change in autonomic activity, has been found following exercise and before a competition.

Table 2.19 Studies that have assessed the effects of competition on heart rate variability.

Author	Number of participants, gender, and training level	Design	HRV Metrics Measured	Outcome
Cervantes et al. (2009)	n = 10, masters swimmers, 4 men, 6 women	HRV assessed 30 minutes prior to a simulated competition on a training day (T1) and at the same time prior to an actual competition (T2)	Mean R-R, SDHR, NN50, ND.Tri, RMSSD, SDNN, pNN50, TINN, VLF power, LF power, HF power, LF/HF%, LFnu, HFnu, VLF%, LF%, HF%, SD1, SD2	RMSSD (T2 < T1), SD1 (T2 < T1), HF power (T2 < T1), HFnu (T2 < T1), LF/HF%, (T1 < T2)
D'Ascenzi et al. (2014)	n = 10, elite volleyball players, 10 women	HRV assessed in the morning (07.00-08.00) two days prior to a competition (T1), one day prior to a competition (T2), and on the day of a competition (T3)	Mean R-R, RMSSD, SDNN, pNN50, VLF power, LF power, HF power, Total power, VLF%, LF%, HF%, LF%/HF%, LFnu/HFnu, LFnu, HFnu, lnVLF, lnLF, lnHF, VLF%, LF%, HF%, SD1, SD2, SD1/SD2	VLF% (T1 < T2, T1 < T3), HF% (T1 < T2, T1 < T3), lnVLF (T1 < T2, T1 < T3)
Edmonds et al. (2013)	n = 9, elite rugby league, 9 youths	HRV assessed two hours prior to activity, two days before game day (T1), on game day (T2), and one (T3), two (T4), and four (T5) days post game day	Mean R-R, RMSSD, SDNN, pNN50, LF power, HF power, HFnu, SD1, SD2, α1, α2, SampEn	pNN50 (T3 < T1), HF power (T2 < T1, T3 < T1, T2 < T4, T2 < T5), HFnu (T2 < T1, T3 < T1)
Mateo et al. (2012)	n = 11, elite BMX cyclists, men. Note n = 5 for resting measures	HRV assessed in the morning (a) and just prior to the activity (b). HRV recorded on the day of a simulated competition (T1) and on days 1 (T2) and 2 (T3) of an actual competition	Mean R-R, RMSSD, SDNN, lnLF, lnHF, LF/HF, α1, SampEn	Mean R-R (T2b < T2a, T3b < T3a, T2b < T1b, T2b < T3b), RMSSD (T2b < T2a, T3b < T3a, T2b < T1b), SDNN (T2b < T2a, T3b < T3a, T2b < T1b), lnLF (T3b < T3a), lnHF (T2b < T2a, T3b < T3a, T2b < T1b), LF/HF (T2a < T2b, T3a < T3b) , α1 (T2a < T2b, T3a < T3b, T1b < T2b), SampEn (T2b < T2a, T3b < T3a, T2b < T1b)

Author	Number of participants, gender, and training level	Design	HRV Metrics Measured	Outcome
Morales et al. (2013)	n = 24, national (group a: 6 men, 8 women) and international (group b: 4 men, 6 women) judo athletes	HRV assessed in the morning prior to a simulated competition (T1) and an actual competition (T2)	HR, SDHR, Mean R-R, RMSSD, SDNN, NN50, VLF power, LF power, HF power, LF/HF, α1, α2, SD1, SD2	HR (T1a < T2a), SDHR (T2a < T1a), Mean R-R (T2a < T1a), RMSSD (T2a < T1a), SDNN (T2a < T1a), NN50 (T2a < T1a), LF/HF (T1a < T2a), α2 (T2a < T1a), SD1 (T2a < T1a), SD2 (T2a < T1a)
Murray & Raedeke, (2008)	n = 20 university students, 11 men, 9 women	Golf putting task either performed alone (T1) or in a competition condition (T2)	SDNN, LFnu, HFnu, LF/HF	LFnu (T1 < T2), HFnu (T2 < T1), LF/HF (T1 < T2)
Souza et al. (2019)	n = 54, all men, 18 canoe athletes (a), 18 street runners (b), 18 jujitsu fighters (c)	HRV assessed 30 minutes prior to training (T1) or a competition (T2)	LFnu/HFnu	LFnu/HFnu (T1abc < T2abc)

Note: < has been used to denote a significantly ($p < 0.05$) lower value in a condition. Conditions have also been coded for each study based on measurement day (e.g. T1, T2...) and group/additional time-point (e.g. T1a may represent the control condition for a particular type of athlete and T1b would be the control group for a different type of athlete). HRV denotes heart rate variability, HR is heart rate, SD is the standard deviation, NN50 is the number of successive R-R intervals that differ by more than 50 ms, IND.Tri is the triangular index, pNN50 is the percentage of successive R-R intervals that differ by more than 50 ms, RMSSD is the root mean square of successive R-R interval differences, SDNN is the standard deviation of normal-to-normal intervals, TINN is the baseline width of the R-R interval histogram, VLF is very-low frequency, LF is low frequency, HF is high frequency, nu is normalised, LF/HF is the ratio of low frequency to high frequency, α1 reflects the detrended fluctuation analysis, describing short-term fluctuations, α2 reflects the detrended fluctuation analysis, describing long-term fluctuations, SampEn is Sample entropy, SD1 is the standard deviation on a Poincaré plot perpendicular to the line of identity, and SD2 is the standard deviation on a Poincaré plot along to the line of identity.

2.7. ADDITIONAL METHODOLOGICAL CONSIDERATIONS AND SUPPLEMENTARY MEASUREMNT TOOLS

The most frequently used laboratory measure of sprint cycling performance is the 30 s WAnT (Jaafar et al., 2014). Each sprint cycling discipline (Keirin, Kilo, Match Sprint, Team Sprint) does, however, have its own technical, tactical, and physiological requirements (Douglas et al., 2021), meaning that it is possible that the WAnT may not provide an accurate reflection of the demands of each activity. In fact, no formal test has been published that specifically attempts to simulate the exertion required for Match Sprint races, especially when considering the repeated efforts that are required during a competition (Ferguson et al., 2021). The specific test protocol that is chosen could have implications for performance, as well as for performance recovery. When considering study design, a balance between ecological validity and experimental control may also be required.

2.7.1. The Warm Up

A warm-up can be either active or passive, although in preparation for a competitive sports performance, an active warm-up is most frequently undertaken (McGowan, 2015). Track cyclists typically complete their warm-up either on rollers or on the track itself (McGowan, 2015). The proposed benefits of performing a warm-up include: increasing muscle temperature; enhancing $\dot{V}O_{2on}$ kinetics; improving muscle fibre contraction velocity; and improving muscle contractile performance (McGowan, 2015). The warm-up for the WAnT typically either consists of 2 – 4 minutes of pedalling that is interspersed by two or three all-out efforts lasting 4 – 8 s or it consists of 5 – 10 minutes alternating between exercise and rest (Inbar et al., 1996). A 3 – 5 minute rest period is then allowed before the main test (Inbar et al., 1996). The traditional WAnT warm-up may, however, differ from the practice that is undertaken by track cyclists. Tomaras and MacIntosh (2011) contrasted the effects of the warm-up that was conventionally undertaken by sprint track cyclists (see Table 2.20) with a shortened 15-minute protocol (see Table 2.21). The traditional warm-up lasted a little over 50 minutes and was developed in consultation with national level cyclists and coaches. Both MPO and PPO were significantly higher during a WAnT following the shortened protocol, although when assessing the contractile function of the quadriceps using electrical stimulation of the femoral nerve, in both cases peak torque was lower than at rest. The authors, therefore, suggested that the proposed shortened warm-up may be preferable to the traditional warm-up, but that a better protocol may still exist (Tomaras & MacIntosh, 2011). The study by Tomaras and MacIntosh (2011) was also conducted more than 10 years ago and in a recent interview, the British sprint cyclist, Jason Kenny, stated that

whilst warm-up protocols may vary between riders, a 10 – 15 minute cycle with three maximal accelerations was believed to be optimal.

"*In a sprint when the flag drops it's time to go, you know there's no riding into it….so for us what we did is we basically condensed it (the warm-up) into 10 to 15 minutes initially with just three rev outs. Nice, short, really punchy and all the testing that we did showed that gets the muscles right up to temperature, it gets your head switched on, and then after that it's just a case of getting on the track and doing a couple of warm-up jumps, really getting your eye in ready to go racing*" (Jason Kenny, GB Sprint Cyclist, Eurosport, 2021)

2.7.2. Type of Start/Rider Position

For the WAnT, Inbar et al. (1996) advised that on the start command participants should pedal as fast as possible against a low resistance to overcome the inertial and frictional resistance of the flywheel (~ 3 – 4 s) before the resistive load was applied. An initial acceleration against no resistance, or a low resistance, has frequently been used during a WAnT (or similar duration sprints) (Barak et al., 2014; Goulopolou et al., 2006; Kirkpatrick & Burrus, 2020), whereas others have chosen a fixed cadence of 70 rpm at the start of the test to provide a rolling start (Bogdanis et al., 1995; Bogdanis et al., 1996a). Alternatively, a stationary start has also been used (Glaister et al., 2019; Wittekind et al., 2011). A flying start may provide greater ecological validity when considering race conditions in the Match Sprint, although a stationary start would provide greater control of the crank position for the initiation of the sprint (Cherry et al., 1998). The type of start used may affect both MPO and PPO, although the findings about the type of start that would elicit higher power outputs have been contradictory (Clark et al., 2018; MacIntosh et al., 2003; Robergs et al., 2015). Allowing the participants to rise out of the saddle during the sprint would also be typical for cyclists in the Match Sprint, and has been shown to elicit greater power outputs during a WAnT (Reiser et al., 2002), but most studies have requested that the participants remain seated throughout (Glaister et al., 2019; Kirkpatrick & Burrus, 2020; MacIntosh et al., 2003; Robergs et al., 2015; Wittekind et al., 2011), with some even utilising a harness to restrict movement (Bogdanis et al., 1995; Bogdanis et al. 1996a).

Table 2.20 Traditional warm-up protocol used by national level sprint cyclists (Tomaras & MacIntosh, 2011).

Time (minutes)	Classification	Gear Ratio	Cue
0:00-4:00	General warm-up	46:16	60% HR_{max}
4:00-8:00			65% HR_{max}
8:00-12:00			70% HR_{max}
12:00-16:00			75% HR_{max}
16:00-18:00			80% HR_{max}
18:00-20:00	Acceleration		Accelerate from 80 to 95% HR_{max}
20:00-20:06	Sprint		6 s sprint
20:06-21:30	Recovery		Cycle lightly as if preparing to stop on the track
21:30-28:00	Rest		Sit comfortably on chair
28:00-30:00	Acceleration sprint	46:16	Progressively accelerate from 0 to 35 $km \cdot h^{-1}$ over 600 m distance, followed by 6 s sprint
30:00-31:30	Recovery		Cycle lightly as if preparing to stop on the track
31:30-38:00	Rest		Sit comfortably on chair
38:00-40:00	Acceleration sprint	48:14	Progressively accelerate from 0 to 35 $km \cdot h^{-1}$ over 600 m distance, followed by 6 s sprint
40:00-41:30	Recovery		Cycle lightly as if preparing to stop on the track
41:30-48:00	Rest		Sit comfortably on chair
48:00-50:00	Acceleration sprint	48:14	Progressively accelerate from 0 to 35 $km \cdot h^{-1}$ over 600 m distance, followed by 6 s sprint
50:00-51:30	Recovery		Cycle lightly as if preparing to stop on the track

Note: HR denotes heart rate

Table 2.21 Shortened warm-up protocol (Tomaras & MacIntosh, 2011).

Time (minutes)	Classification	Gear Ratio	Cue
0:00-5:00	General warm-up	46:16	60% HR_{max}
5:00-10:00			65% HR_{max}
10:00-15:00			70% HR_{max}
15:00-15:30			Progressively accelerate to 35 $km \cdot h^{-1}$
15:30-15:36			6 s sprint
15:36-17:00	Acceleration		Cycle lightly as if preparing to stop on the track

Note: HR denotes heart rate

2.7.3. Sprint Duration

Depending on the specific discipline, track cycling sprint efforts are likely to be in the range of 15 – 60 s (Douglas et al., 2021; Ferguson et al., 2021). The race distance in the Match Sprint is typically 750 m (three laps of a 250 m track). Viewing timing data from the Kilo (1 km time-trial) at the 2018 World Track Championships, it could be estimated that if a rider went all-out from the start in a Match Sprint race it would take ~ 45 s to complete the event (see Table 2.22) (although aero-bars

are fitted to the bikes in the Kilo). A multitude of factors can, however, also influence rider tactics in the Match Sprint. Therefore, it is unlikely that a sprint cyclist will ride a Match Sprint race maximally from the start, but a rider may try to surprise their opponent and launch an early all-out effort over ~ 2 – 2.5 laps.

Table 2.22 Lap times during the Kilo (1000 m time-trial) at the 2018 World Track Cycling Championships in Apeldoorn (Netherlands) (times are cumulative).

Rider Name	Lap 1 (s)	Lap 2 (s)	Lap 3 (s)	Lap 4 (s)
Michael D'Almeida	18.692	31.799	45.612	60.518
Fabian Puestas Zapata	18.971	32.241	45.925	60.8
Eric Engler	18.458	31.605	45.378	60.462
Quentin Lafargue	18.661	31.83	45.575	60.407
Theo Bos	18.53	31.816	45.541	59.955
Matthew Glaetzer	19.011	32.081	45.701	59.745
Jeffrey Hoogland	17.358	30.129	43.985	59.459
Mean ± standard deviation	18.526 ± 0.555	31.643 ± 0.699	45.388 ± 0.641	60.192 ± 0.481

The qualifying round (The Flying 200) is an individual time-trial, which will likely require a more consistent approach. For the Flying 200, each rider completes 2.5 – 3.5 laps of the track depending on the track length (UCI, 2020). A power output profile from a Flying 200 for an elite sprint cyclist is displayed in Figure 2.32. The time recorded for the event was 10.3 s. The ride has been divided into three phases. During the first phase, power output undulates, whilst cadence and speed increase. The rider will be cycling around the high side of the track during this phase. The undulation in power output is likely to be related to the position that the rider is on the track (on the straight or going into or out of the banked bend). During the second phase, speed, power output, and cadence all increase. The rider will remain on the high side of the track during this phase. A dip in power output is then apparent at the end of the phase before a sharp rise up to ~ 2000 W, indicating the start of the third and final section. The small dip in power output may again have been related to the part of the track the rider was on or it may have been at the point the rider transitioned from being in-the-saddle to being out-the-saddle (see Figure 2.33 for an example transition), signalling the launch of the final all-out effort. The exact position on the lap where the rider commences this effort will vary between riders and may be specific to the track, but it will typically occur on the bend before the 200 m line. The rider will also now move towards the inside of the track, before crossing the 200 m start line. The estimated time from the rider coming out of the saddle to the end of the Flying 200 for the 30 entrants that competed in the Match Sprint at the 2020 Tokyo Olympics was 16.9 ± 1.1 s (see Table 2.23). The average duration of this maximal effort was not dissimilar to the shortened WAnT (18 s) undertaken by sprint track cyclists at the U.S. Olympic Training Centre (Coaching and Sports Science Division of the United States Olympic Committee, 2004).

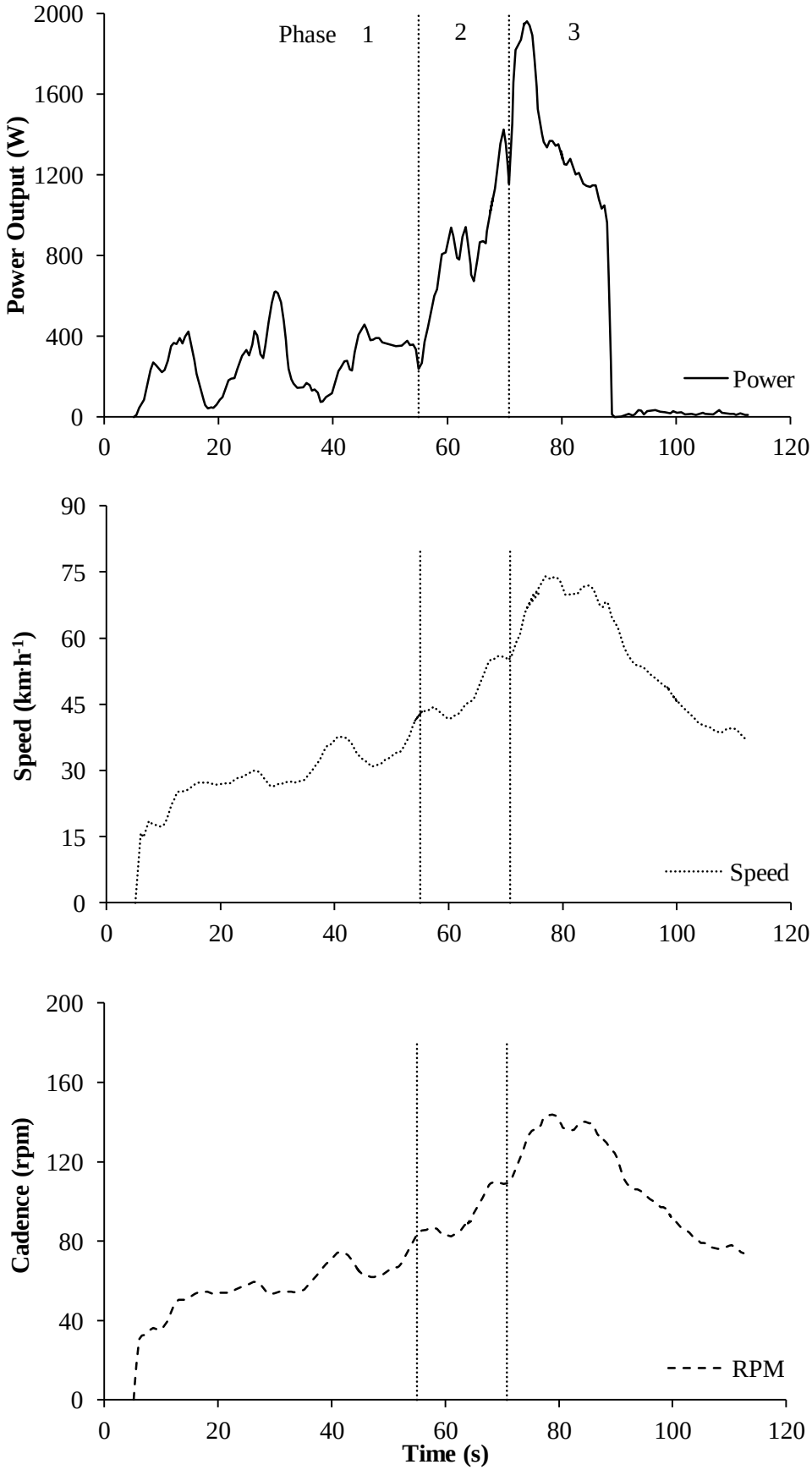

Figure 2.32 A power output profile for an elite track cyclist performing a Flying 200. RPM denotes revolutions per minute.

Table 2.23 Qualifying time and estimated maximal effort sprint times for riders competing in the Match Sprint at the Tokyo Olympics 2020.

Rider Name	Flying 200 m Time (s)	Estimated Maximal Effort Time (s)
Kwesi Browne	9.966	16.715
Patryk Rajkowski	9.594	16.372
Tomas Babek	9.856	*
Hugo Barrette	9.596	17.623
Sergey Ponomaryov	9.932	16.481
Ethan Mitchel	9.705	*
Maximilian Levy	9.646	*
Nick Wammes	9.587	17.393
Jean Spies	9.787	18.125
Rayan Helal	9.669	17.827
Jack Carlin	9.306	16.815
Matthew Richardson	9.685	18.284
Xu Chao	9.584	16.602
Wakimoto Yuta	9.518	17.413
Sebastien Vigier	9.551	*
Stefan Boetticher	9.593	16.956
Nathan Hart	9.696	18.206
Quintero Chavarro	9.626	*
Muhammed Shah Firdaus Sahrom	9.700	*
Pavel Yakushevskiiy	9.723	16.803
Nitta Yudai	9.787	17.813
Nicholas Paul	9.316	13.248
Sam Webster	9.631	*
Jair Tjon En Fa	9.472	16.743
Mohd Azizulhasni Awang	9.626	*
Mateusz Rudyk	9.493	*
Jefferey Hoegland	9.215	17.466
Harrie Lavreyson	9.215	*
Denis Dmitriev	9.331	15.468
Jason Kenny	9.510	16.632
Mean ± standard deviation	9.597 ± 0.188	16.949 ± 1.129

* denotes where it was not possible to identify when a rider transitioned from in-the-saddle to out-the-saddle to initiate the final effort.

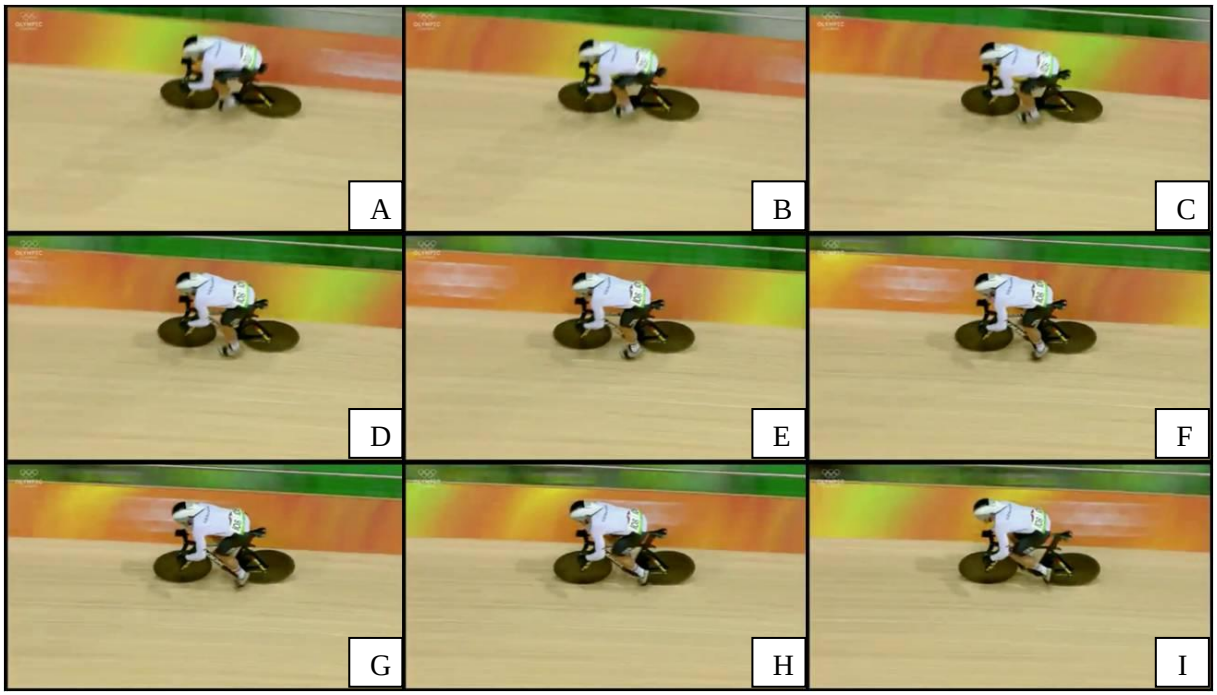

Figure 2.33 A rider transitioning from in-the-saddle to out-the-saddle during a Flying 200. In position A the rider is seated, but then begins to propel his body forward and move out the saddle (B-C) continuing in this direction with the entire saddle being visible in H and I.

2.8.4. Resistive Load

The WAnT was a modification of the W Max 30 test designed by Cumming (1973), with the difference being that the braking force in the WAnT was set relative to the total body-mass (TBM) of the participants (Ayalon et al., 1974). During pilot testing, Ayalon et al. (1974) found that a resistive load of 0.04 $kg \cdot kg^{-1}$ TBM generated the highest power output over the duration of the test (see Figure 2.34). This was supported by subsequent data on young (13 – 14 year-old) girls, whereas a fractionally higher load (0.0422 $kg \cdot kg^{-1}$ TBM) was advocated for young boys (Bar-Or, 1980, as cited by Dotan & Bar-Or, 1983). Intriguingly, Bar-Or (1987) stated that the original load advocated by the Wingate group was marginally higher still (0.045 $kg \cdot kg^{-1}$ TBM), providing what is commonly believed to be the traditional load for the WAnT. It should be noted that the Wingate Institute used a Fleisch ergometer (Fleisch-Metabo, Basel, Switzerland), whereas a substantial quantity of subsequent research used a Monark ergometer (Monark, Varberg, Sweden). The Monark ergometer uses a rope braking system, whereby a rope is wrapped around a flywheel and connected to a weighted basket, where the braking load is applied (see Figure 2.35) (Gordon et al., 2004). Each pedal revolution on a Monark ergometer results in the flywheel moving through 6 m (Gordon et al., 2004), as opposed to 10 m on a Fleisch ergometer. It has, therefore, been estimated that a mass of 0.075 $kg \cdot kg^{-1}$ TBM applied to a Monark ergometer would be the equivalent of 0.045 $kg \cdot kg^{-1}$ TBM on a Fleisch ergometer (Bar-Or & Rowland, 2004). Whilst the traditional WAnT load (0.075 $kg \cdot kg^{-1}$ TBM) is still often

employed in laboratories, others have questioned whether this resistance is optimal (Bradley & Ball, 1992; Buśko, 2005; Dotan & Bar-Or, 1983; Evans & Quinney, 1981; Lu et al., 2008; Patton et al., 1985) and a large number of different loads, and means of calculating the optimal load for sprint cycling performance, have been proposed (see Table 2.24) (Baker et al., 2001; Baker et al., 2004; Baker & Davies, 2004a; Baker & Davies, 2004b; Baker & Davies, 2006a; Baker & Davies, 2006b; Dotan & Bar-Or, 1983; Lu et al., 2008; MacIntosh et al., 2003; Patton et al., 1985; Pazin et al., 2011; Richmond et al., 2011; Rodgers & Hermiston, 2000; Tong et al., 2008; Üçok et al., 2005; Vandewalle et al., 1985; Vargas et al., 2015).

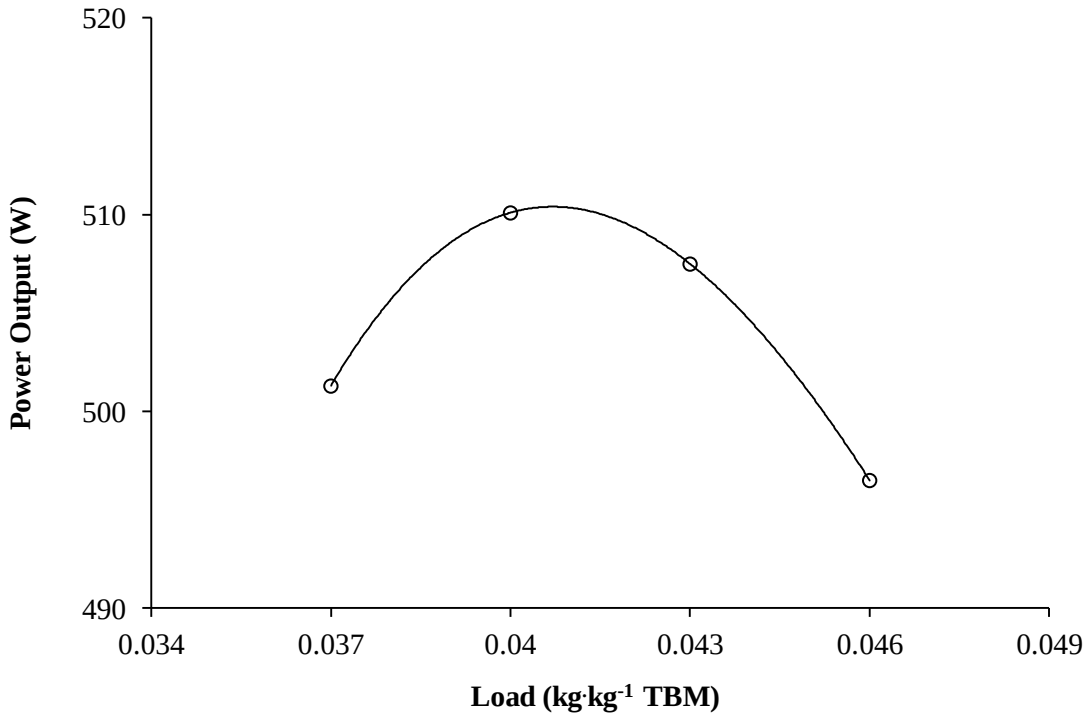

Figure 2.34 Mean power output obtained during a 30 s all-out effort on a Fleisch-Metabo ergometer (n = 5) (Ayalon et al., 1974). Note: TBM denotes total body mass.

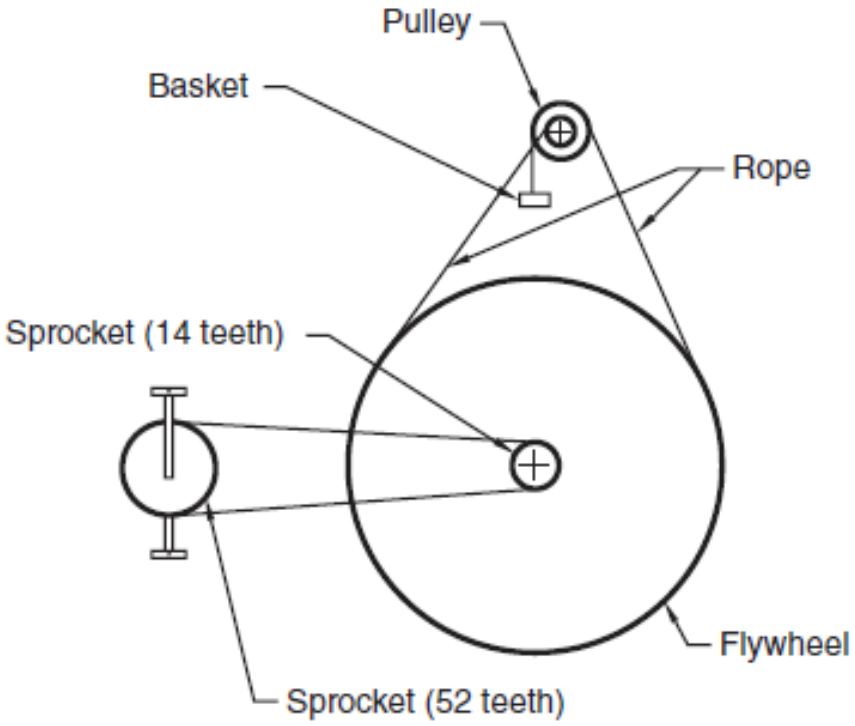

Figure 2.35 Layout of the rope-brake system for a typical Monark cycle ergometer (Gordon et al., 2004).

Table 2.24 Studies that have assessed the effects of resistive load on sprint cycling performance.

Author	Number of participants, gender, and training level	Resistive Loads	Performance Test	Additional notes	Outcome
Baker et al. (2001)	n = 8 males rugby n = 2 males sprinters	0.1 $kg \cdot kg^{-1}$ TBM, 0.1 $kg \cdot kg^{-1}$ LBM rugby 0.12 $kg \cdot kg^{-1}$ TBM, 0.12 $kg \cdot kg^{-1}$ LBM sprinters	20 s sprint		PPO ↑ in LBM MPO ↔ FI ↔
Baker et al. (2004)	n = 18 males recreationally active	FVT TBM 0.075 – 0.095 $kg \cdot kg^{-1}$ TBM, FVT LBM 0.075 – 0.095 $kg \cdot kg^{-1}$ LBM	30 s Wingate		Resistive force ↓ in LBM PPO ↑ in LBM MPO ↔ FI ↔
Baker & Davies (2004a)	n = 8 males rugby backs n = 8 males rugby forwards	FVT TBM 0.07 – 0.095 $kg \cdot kg^{-1}$ TBM, FVT LBM 0.07 – 0.095 $kg \cdot kg^{-1}$ LBM	6 s sprint FVT		Resistive force ↓ in LBM PPO ↑ in LBM
Baker & Davies (2004b)	n = 11 males soccer players	0.08 $kg \cdot kg^{-1}$ TBM, 0.08 $kg \cdot kg^{-1}$ LBM	6 s sprint		PPO ↑ in LBM
Baker & Davies (2006a)	n = 11 males karate	FVT TBM 0.07 – 0.09 $kg \cdot kg^{-1}$ TBM, FVT LBM 0.07 – 0.09$kg \cdot kg^{-1}$ LBM	8 s sprint FVT		PPO ↑ in LBM
Baker & Davies (2006b)	n = 11 males overweight and obese	0.075 $kg \cdot kg^{-1}$ TBM, 0.075 $kg \cdot kg^{-1}$ LBM	10 s sprint		PPO ↑ in LBM

Author	Number of participants, gender, and training level	Resistive Loads	Performance Test	Additional notes	Outcome
Bogdanis et al. (2007)	n = 12 preadolescent boys	FVT TBM 50% FVT TBM	10 x 6 s sprints, 24 s passive rest between sprints	Optimal load calculated using polynomial regression	PPO Sprint 1 ↔ MPO Sprint 1 ↑ FVT TBM MPO Sprint 1-10 ↑ FVT TBM FI ↔ (Sprint 1 – Sprint 10) FI ↑ 50% FVT TBM (during individual sprints)
Bogdanis et al. (2008)	n = 8 males university students	50 $g \cdot kg^{-1}$ TBM 100 $g \cdot kg^{-1}$ TBM	7 x 6 s sprints, 30 s passive rest between sprints		PPO ↑ 100 $g \cdot kg^{-1}$ TBM MPO ↑ 100 $g \cdot kg^{-1}$ TBM FI ↑ 100 $g \cdot kg^{-1}$ TBM
Dotan & Bar-Or (1983)	n = 18 females n = 17 males recreationally active	0.058 $kg \cdot kg^{-1}$ TBM, 0.067 $kg \cdot kg^{-1}$ TBM, 0.075 $kg \cdot kg^{-1}$ TBM, 0.083 $kg \cdot kg^{-1}$ TBM, 0.092 $kg \cdot kg^{-1}$ TBM	30 s Wingate	Optimal load calculated using polynomial regression	MPO optimal load women 0.084 $kg \cdot kg^{-1}$ TBM MPO optimal load men 0.086 $kg \cdot kg^{-1}$ TBM PPO continued to increase with load
Lu et al. (2008)	n = 8 males lean active n = 8 males overfat active	0.075 $kg \cdot kg^{-1}$ TBM, 0.1 $kg \cdot kg^{-1}$ TBM, 0.11 $kg \cdot kg^{-1}$ TBM, 0.125 $kg \cdot kg^{-1}$ TBM, 0.14 $kg \cdot kg^{-1}$ TBM	30 s Wingate	Optimal load calculated using polynomial regression	PPO & MPO ↑ in both groups from the initial workload Optimal load ↔ between groups Optimal load PPO lean 0.128 $kg \cdot kg^{-1}$ TBM Optimal load PPO overfat 0.117 $kg \cdot kg^{-1}$ TBM Optimal load MPO lean 0.114 $kg \cdot kg^{-1}$ TBM Optimal load MPO overfat 0.111 $kg \cdot kg^{-1}$ TBM

Author	Number of participants, gender, and training level	Resistive Loads	Performance Test	Additional notes	Outcome
MacInosh et al. (2003)	n = 4 females athletes from a variety of sports n = 9 males athletes from a variety of sports	0.085 $kg \cdot kg^{-1}$ TBM rolling start, 0.085 $kg \cdot kg^{-1}$ TBM stationary start, Individualised FVT	30 s Wingate		Optimal load ↑ compared to TBM protocol PPO ↓ in rolling start PPO ↔ between 0.085 $kg \cdot kg^{-1}$ TBM stationary start and individualised FVT MPO ↔ between 0.085 $kg \cdot kg^{-1}$ TBM stationary start and individualised FVT FI ↔ between 0.085 $kg \cdot kg^{-1}$ TBM stationary start and individualised FVT
Patton et al. (1985)	n = 19 males healthy	0.055 – 0.115 $kg \cdot kg^{-1}$ TBM	30 s Wingate		Resistance ~ 0.095 $kg \cdot kg^{-1}$ TBM optimal PPO ↑ in 0.095 $kg \cdot kg^{-1}$ TBM compared to 0.075 $kg \cdot kg^{-1}$ TBM MPO ↑ in 0.095 $kg \cdot kg^{-1}$ TBM compared to 0.075 $kg \cdot kg^{-1}$ TBM
Pazin et al. (2011)	n = 10 males strength trained n = 10 males speed trained n = 10 males active n = 10 males sedentary	FVT TBM 0.05 – 0.12 $kg \cdot kg^{-1}$ TBM	FVT 6 s sprints	Optimal load calculated using polynomial regression	Optimal load strength 0.097 $kg \cdot kg^{-1}$ TBM Optimal load speed 0.092 $kg \cdot kg^{-1}$ TBM Optimal load active 0.087 $kg \cdot kg^{-1}$ TBM Optimal load sedentary 0.08 $kg \cdot kg^{-1}$ TBM Optimal load strength > active & sedentary Optimal load speed > sedentary PPO highest in strength & lowest in sedentary PPO ↑ under intermediate loads (0.08 $kg \cdot kg^{-1}$ TBM & 0.09 $kg \cdot kg^{-1}$ TBM) than under light (0.05 $kg \cdot kg^{-1}$ TBM, 0.06 $kg \cdot kg^{-1}$ TBM) or heavy (0.11 $kg \cdot kg^{-1}$ TBM, & 0.12 $kg \cdot kg^{-1}$ TBM) loads

Author	Number of participants, gender, and training level	Resistive Loads	Performance Test	Additional notes	Outcome
Rodgers & Hermiston (2000)	n = 16 males healthy	0.075 $kg \cdot kg^{-1}$ TBM, Evans & Quinney method, 10 s max	30 s Wingate		Resistive load ↑ in Evans & Quinney PPO ↔ MPO ↓ in Evans & Quin
Tong et al. (2008)	n = 16 females lean n = 15 females overfat n = 16 males lean n = 18 females overfat	0.075 $kg \cdot kg^{-1}$ TBM, 0.086 $kg \cdot kg^{-1}$ LBM males, 0.095 $kg \cdot kg^{-1}$ LBM females	30 s Wingate		PPO ↔ lean (as expected, as equivalent load) MPO ↔ lean (as expected, as equivalent load) FI ↔ lean (as expected, as equivalent load) PPO ↓ in 0.095 $kg \cdot kg^{-1}$ LBM compared to 0.075 $kg \cdot kg^{-1}$ TBM in overfat females PPO ↔ in 0.086 $kg \cdot kg^{-1}$ LBM compared to 0.075 $kg \cdot kg^{-1}$ TBM in overfat males MPO ↓ in 0.095 $kg \cdot kg^{-1}$ LBM compared to 0.075 $kg \cdot kg^{-1}$ TBM in overfat females MPO ↓ in 0.086 $kg \cdot kg^{-1}$ LBM compared to 0.075 $kg \cdot kg^{-1}$ TBM in overfat males FI ↔ in 0.095 $kg \cdot kg^{-1}$ LBM compared to 0.075 $kg \cdot kg^{-1}$ TBM in overfat females FI ↔ in 0.086 $kg \cdot kg^{-1}$ LBM compared to 0.075 $kg \cdot kg^{-1}$ TBM in overfat males
Üçok et al. (2005)	n = 24 males untrained	0.075 $kg \cdot kg^{-1}$ TBM, 0.085 $kg \cdot kg^{-1}$ TBM, 0.095 $kg \cdot kg^{-1}$ TBM, 0.09 $kg \cdot kg^{-1}$ LBM, 0.1 $kg \cdot kg^{-1}$ LBM, 0.11 $kg \cdot kg^{-1}$ LBM	30 s Wingate		PPO ↑ in 0.1 $kg \cdot kg^{-1}$ LBM and 0.11 $kg \cdot kg^{-1}$ LBM compared to 0.075 $kg \cdot kg^{-1}$ TBM MPO ↔

Author	Number of participants, gender, and training level	Resistive Loads	Performance Test	Additional notes	Outcome
Vandewalle et al. (1985)	n = 7 females healthy n = 7 males healthy	FVT Anaerobic capacity 2.0-6.1 kg women 3.1-9.2 kg men	45 s all out effort	Optimal load calculated directly using polynomial regression and indirectly by linear regression between force and mean velocity	Optimal force either: 0.09 $kg \cdot kg^{-1}$ TBM females 0.1 $kg \cdot kg^{-1}$ TBM males
Vargas et al. (2015)	n = 10 females n = 21 males recreationally active	0.085 $kg \cdot kg^{-1}$ TBM, Individualised FVT	30 s Wingate		Females: PPO ↔ MPO ↔ FI ↔ Males: PPO ↔ MPO ↔ FI ↔

Note: ↑ denotes a significant ($p < 0.05$) increase, ↔ denotes no significant difference ($p \geq 0.05$), ↓ denotes a significant ($p < 0.05$) decrease, > denotes significantly ($p < 0.05$) greater than. FVT denotes force velocity test, TBM is total body mass, LBM is lean body mass, PPO is peak power output, MPO is mean power output, and FI is fatigue index.

It has also been suggested that the type of training that the participants undertake may need to be considered for sprint cycling load optimisation (Hale et al., 1988; Pazin et al., 2011). Pazin et al. (2011) investigated the optimal resistive load for sedentary, active, speed-trained, and strength-trained individuals. In a randomised order, participants undertook eight 6 s sprints against resistive loads ranging from 5% – 12% body-weight. A second order polynomial function was fit to the individual data sets (see Figure 2.36). From their data, the authors concluded that the optimal load was greatest for the strength trained (9.7% TBW), followed by speed trained (9.2% TBW), then active (8.7% TBW), and finally sedentary (8.0% TBW) individuals.

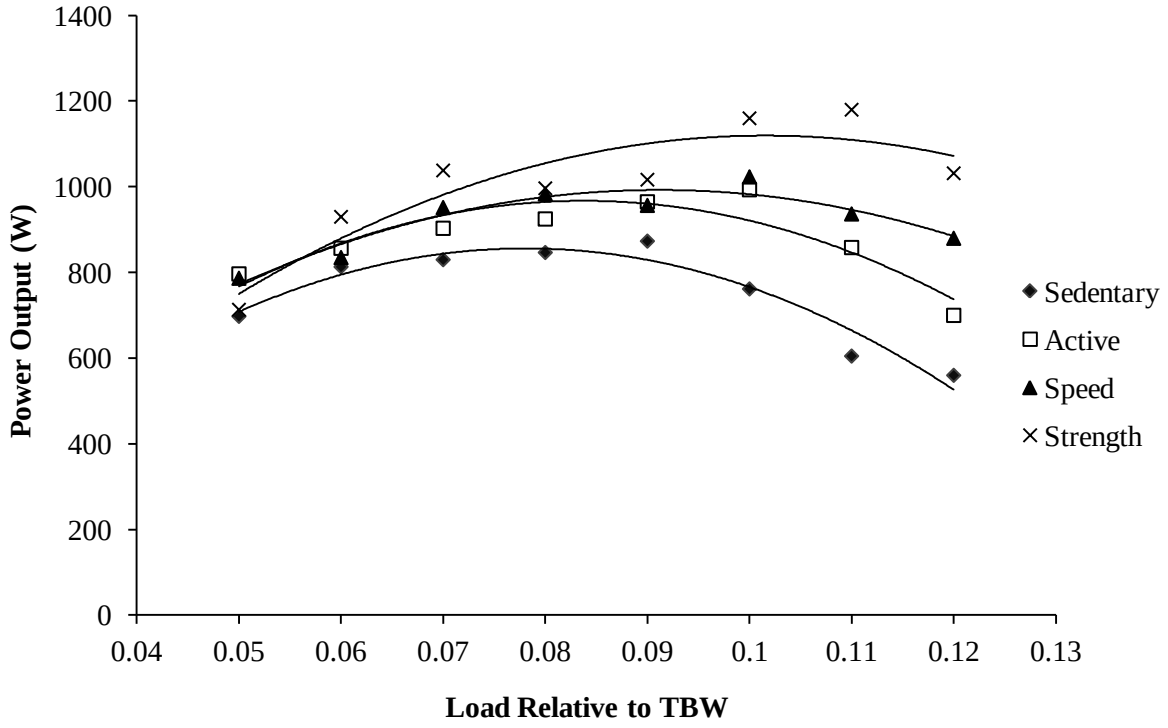

Figure 2.36 Raw data points, as well as polynomial regression curves, for power output at the loads tested for representative participants from the sedentary, active, speed-trained and strength-trained groups (Pazin et al., 2011). Note: TBW denotes total body-weight.

2.7.5. Sex

Testosterone levels are higher in men than women (Hunter et al., 2023). Testosterone produces a potent anabolic effect on muscle mass, bone mass, and Hb mass (Hunter et al., 2023). A greater muscle mass and muscle contractile velocity provide the main explanatory factors for the greater MPO and PPO that are produced by men during cycling sprints (Alper et al., 2018; Billaut et al., 2003; Billaut & Bishop, 2012; Esbjörnsson-Liljedahl et al., 1999; Esbjörnsson-Liljedahl et al., 2002; Falgairette et al., 2004; Hunter et al., 2023; Mageean et al., 2011; Perez-Gomez et al., 2008).

Muscle biopsies have also revealed that women may possess a greater percentage of Type I muscle fibres in the *vastus lateralis* (Ansdell et al., 2020; Simoneau & Bouchard, 1989), although Billaut and Bishop (2009) stated that sex differences in muscle fibre composition may not always be apparent when considering sport specialities and fitness levels. Nonetheless, a greater percentage of Type I fibres in the thigh muscles could explain the alteration in substrate utilisation (greater aerobic contribution) that has been observed in women during single cycling sprints (Hill & Smith, 1993), as well as accounting for the lower percentage decrement that has been found during repeated-sprints (Billaut & Smith, 2009; Laurent et al., 2010).

Not all studies have, however, reported a greater reduction in percentage decrement during repeated-sprint exercise in women (Alper et al., 2018; Billaut & Bishop, 2012; Esbjörnsson-Liljedahl et al., 2002). Alper et al. (2018) found that percentage decrement did not differ between men and women during five 6 s cycling sprints, but did state that the demands of the task (number of repetitions) may not have been sufficient for identifiable differences to have occurred. When twenty 5 s sprints were performed with 25 s between sprints, Billaut and Bishop (2012) found that the percentage decrement was higher in men, although when two sub-groups (7 men and 7 women) were compared, who were matched on their initial sprint performances, no significant differences existed for the total work performed or the percentage decrement (Billaut & Bishop, 2012). The authors, therefore, suggested that a gender effect on percentage decrement may simply relate to the higher power outputs that are generally produced by men, rather than a physiological difference between sexes in recovery (Billaut & Bishop, 2012). That being said, when viewing the PPO that have been reported during cycling sprints by men and women (see Table 2.25), recruiting a mixed-sex sample would increase the risk of generating bimodal data, complicating the analysis (Roberts & Russo, 1999).

Table 2.25 Peak power output (PPO) during a single sprint or the first sprint during a repeated-sprint task in men and women. Data are displayed as mean ± standard deviation.

Author	Task	Participants	PPO Sprint 1 Men (W)	PPO Sprint 1 Women (W)
Bishop et al., 2003	6 s cycling sprint	Female members of Australian Hockey Squad	-	996 ± 88
Bogdanis et al., 1995	30 s cycling sprint	Male university students	1264 ± 156	-
Bogdanis et al., 1996b	30 s cycling sprint	Male university students	1291 ± 249	-
Bogdanis et al., 1998	10 s cycling sprint	Male university students	1262 ± 172	-
Esbjörnsson-Liljedahl et al., (1999)	30 s cycling sprint	College students for sports and recreation	860 ± 98	629 ± 91
Esbjörnsson-Liljedahl et al., (2002)	30 s cycling sprint	College students for sports and recreation	837 ± 76	587 ± 68
Falgairette et al., 2004	FVT	Physical education students	1093 ± 170	648 ± 51
Kirkpatrick & Burrus, 2020	30 s cycling sprint	Recreationally active females	-	737 ± 171

Note: FVT denotes force-velocity test.

2.7.6. Perceptions

The sensations that are experienced by an individual, such as tiredness and lack of energy, could affect both performance and perceived exertion during an exercise task (Marcora et al., 2009). The rating of perceived exertion (RPE) scale is the most commonly used single item scale in exercise science (Halperin & Emmanuel, 2019). RPE has been assessed following a single sprint (Wittekind et al., 2011) and after each sprint during a repeated-sprint task (Billaut et al., 2011; Hagen & Phillips, 2018; Hureau et al., 2016; Hureau et al., 2014). RPE can be measured using either the 6 – 20 category scale (Figure 2.37A) or the category ratio 10 scale (see Figure 2.37B). Using the 6 – 20 category scale, Billaut et al. (2011) reported that RPE increased in an approximately linear fashion from the first to the last sprint (ten 6 s sprints with 24 s recovery between sprints), with a rating of ~ 13 being provided immediately after the first sprint, increasing to ~ 19 after the last sprint. It does, however, appear somewhat confusing to state that maximal effort must be provided for each sprint, yet the perceived exertion was not maximal. Abbiss et al. (2015) suggested that effort and exertion are two different constructs, although the authors acknowledged that these terms have been used interchangeably in the literature and Pageaux (2016) noted that the Oxford English dictionary defines effort as "strenuous physical or mental exertion" and exertion as "physical or mental effort", questioning whether participants would be able to clearly differentiate between these terms. The use of RPE scales to monitor perceptions of recovery following exercise has also been criticised (Glaister et al., 2012; Micklewright et al., 2017). Micklewright et al. (2017) reported that when RPE scores were tracked for 30 minutes after exhaustive exercise (ramp test), the score was always at its minimum immediately after exercise, with the authors stating that the RPE scale was never designed to be used in recovery.

a

Rating	Perceived Exertion
6	
7	Very, very light
8	
9	Very light
10	
11	Fairly light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very hard
18	
19	Very very hard
20	

b

Rating	Perceived Exertion
0	Nothing at all
0.5	Very, very weak
1	Very weak
2	Weak
3	Moderate
4	Somewhat strong
5	Strong
6	
7	Very strong
8	
9	
10	Very, very strong
•	Maximal

Figure 2.37 A) 6 – 20 category scale (Borg, 1970) and B) the category ratio 10 scale (Borg, 1982) for the assessment of ratings of perceived exertion.

The rating of fatigue (ROF) scale (see Figure 2.38) was developed by Micklewright et al. (2017). Following a series of studies, it was suggested that the ROF scale had good face- and construct-validity (Micklewright et al., 2017), although as the descriptors do not accurately reflect perceptions of recovery, the intended use must again be considered. An alternative scale that does possess face validity to assess recovery status is the Perceived Readiness Scale (see Table 2.26) (Edwards et al., 2011). Edwards et al. (2011) evaluated the effectiveness of the Perceived Readiness Scale during an interval training session (5 x 1000 m at an RPE of 17) and found that the participants were able to use the scale reliably, although if the objective was to perform maximal intensity sprint efforts, the cues provided for ratings 1 to 4 become confusing. Another means of assessing perceptions of recovery is via a visual analogue scale (VAS). Following a 30 s sprint, Glaister et al. (2012) asked the participants to indicate their recovery status by placing a mark on a 20 cm VAS at six time-points (5, 10, 20, 40, 80, 160 s), with the scale ranging from “not at all recovered” to “completely recovered” (Glaister et al., 2012). Prior to the analysis, the markings were converted to a percentage and the time-course of perceived recovery was modelled using a one-phase exponential

function (Glaister et al., 2012). The authors suggested that the participants initially underestimated their performance recovery status and at the time-point where the participants felt completely recovered, PPO was at 83.6 ± 5.2% of the criterion value (highest power output during a 5 s sprint in the first trial). The recovery time course of perceived recovery was, however, found to be similar to that of $\dot{V}O_2$ and minute ventilation (Glaister et al., 2012).

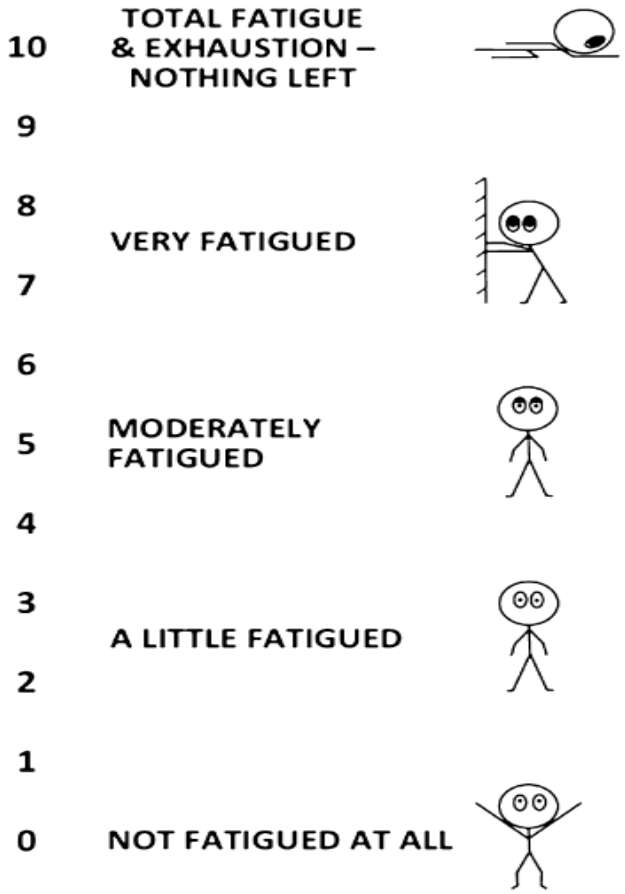

Figure 2.38 The rating of fatigue scale (Micklewright et al., 2017).

Table 2.26 Perceived readiness scale (Edwards et al., 2011).

Rating	Perceived Readiness	Cue
7	Exhausted	Unable to exercise
6	Very Tired	Unable to exercise at the required intensity
5	Tired	Not yet able to exercise at the desired intensity
4	Adequately Recovered	Able to exercise at the required intensity
3	Well Recovered	Able to exercise above the required intensity
2	Very Well Recovered	Well able to exercise above the required intensity
1	Fully Recovered	Able to exercise at maximal intensity

2.7.7. Summary

In summary, methodological considerations for sprint cycling research may require a balance between ecological validity and experimental control. A rolling start may provide greater ecological validity, but a stationary start would facilitate greater control of the crank position when the sprint is initiated. Likewise, allowing the participants to rise out of the saddle during the sprint may elicit greater power outputs, but most research has been conducted with the participants remaining seated throughout the sprint. In preparation for the sprint, a period of light cycling, interspersed with maximal accelerations is currently the method chosen by elite sprint cyclists and is often used in research settings, although this may still not be optimal. For the sprint itself, the traditional resistive load may be insufficient for strength and speed athletes to optimise performance. Whilst a 30 s test has most commonly been used, the duration may not provide an accurate representation of a Match Sprint race and as sprint duration could affect the amount of fatigue generated, it could also affect the required recovery time. A shortened, 18 s, version of the WAnT has been used by the U.S. track cycling coaching team, which was similar to the duration of maximal effort that is typically provided during the Flying 200. Regarding the sex of the participants, women have been shown to experience greater fatigue resistance during repeated-sprint tasks, albeit this may be due to the higher power outputs that are typically produced by men. Finally, the RPE scale is commonly used in exercise science, but its use during a repeated-sprint task or for assessing perceived recovery status may be questionable. Alternative scales have been developed to assess fatigue status and readiness to perform an exercise task, but the descriptors may need to be closely considered and do need to accurately reflect the constructs that they wish to measure.

2.8. SUMMARY OF THE LITERATURE REVIEW

The Match Sprint track cycling competition requires successful riders to compete on multiple occasions each day and over consecutive days. The competition begins with an individual time-trial, whereby each rider completes 3.5 laps of the track, gradually increasing speed before launching an all-out effort, typically on the bend before the final 200 m line. The duration of maximal effort will vary depending on the track, but was estimated to be ~ 17 s at the Tokyo Olympics. Head-to-head races in the main competition are then conducted over three laps of the track, with the final 200 m of the race being timed, although the objective is simply to be the first across the finish line. Maximising power output during the sprint is of great importance, but the event is also highly tactical and may not simply be won by the most powerful rider. The significant influence of air resistance and the benefits that can be gained from closely following another rider, mean that the duration of

maximal effort varies in each race depending on the tactics employed. Each sprint will, however, almost certainly require less than 45 s of maximal effort. The time between races also varies, but can be as little as 10 – 15 minutes. It is not certain whether the recovery of MPO and PPO would be complete over this time-frame.

Repeated high force muscular contractions must occur rapidly to generate the extremely high power outputs that are produced in each sprint. ATP is the energy currency in the human body and is essential for muscular contractions to occur. The quantity of stored ATP is limited, but distinct pathways exist to enable its regeneration. The relative contribution of ATP generated by each energy system will vary depending on the demands of the task, but during a cycling sprint, the majority of ATP resynthesis will occur via the anaerobic pathways. Following the sprint, individuals that display faster PCr recovery kinetics could experience an improvement in performance restoration. PCr resynthesis does, however, require aerobically generated ATP. Therefore, individuals that experience faster muscle reoxygenation could exhibit faster PCr resynthesis. Muscle reoxygenation can be measured at the site of the active muscles using NIRS or $\dot{V}O_2$ can be recorded at the mouth and nose using breath-by-breath measurement systems, with inferences then being made about the exercising muscle. PCr resynthesis may also be a biphasic process, with the primary phase relating to the oxidative formation of ATP, but the secondary phase being dictated by muscle pH. A change in muscle pH could also affect key glycolytic enzymes, limiting the amount of energy derived from the glycolytic pathway when sprints are repeated. Whilst opinions do vary substantially about the cause of acidosis during exercise, as well as the role that acidosis plays in skeletal muscle fatigue, the view of many athletes and coaches, and some sports scientists, remains that the clearance of lactate/lactic acid is essential if performance restoration is to be optimised. Finally, from an energetics perspective, the capacity of the aerobic energy system for ATP resynthesis is much greater, but the rate of regeneration is slower. The relative contribution of energy derived via the aerobic pathway during a sprint will, therefore, be less than via the anaerobic pathways, but the contribution will increase with sprint duration, as well as when sprints are repeated with an incomplete recovery period. Individuals with a higher aerobic capacity will also display faster $\dot{V}O_{2on}$ kinetics. Faster $\dot{V}O_{2on}$ kinetics could add to the energy contribution during a sprint.

From a pacing perspective, it is often stated that sprints should be undertaken all-out from the start and that the effort should be maximal throughout. There is, however, evidence to suggest that a more reserved pacing strategy may be adopted as the duration of a sprint increases. Deception studies have also provided evidence of a change in effort depending on the information provided. Potential motivation reflects the maximum amount of effort that an individual is prepared to exert to satisfy a motive. In addition to sprint duration, and the information provided about the demands of the task, competition could affect potential motivation, with the possibility that audience presence, performance comparison, financial incentives, and verbal encouragement, could all add to the

motivational response. From a physiological perspective, it is conceivable that the reason that these motivational variables could improve sprint performance is because the athlete would be in a heightened state of sympathetic arousal prior to the exercise task. The physiological response to stress has two components: a faster component, which concerns the sympathetic adrenal medullary axis; and a slower component, which relates to the hypothalamic-pituitary adreno-cortical axis. Due to the sensitivity and the speed of the response, assessing the activity of the sympathetic nervous system may be preferable when considering a sports competition, with AA providing a measurable marker. Autonomic nervous system activity can also be assessed via the measurement of HRV, but the metrics that are derived may provide a better indication of parasympathetic withdrawal, as opposed to sympathetic activation. Nonetheless, in response to stress, parasympathetic withdrawal will occur, as well as a decrease in HRV.

2.8.1. General Aim

The general aim of this PhD thesis was to investigate physiological and motivational factors that could affect sprint cycling performance and recovery, with consideration to the Match Sprint competition.

2.8.2. Specific Aims

1. To model the time-course of sprint cycling performance recovery and to examine the relationship between the performance recovery rate and various physiological factors that could potentially influence performance restoration

2. To assess the effects of the between sprint recovery activity that is currently practiced by elite sprint cyclists (a mixture of active and passive recovery) on performance restoration and to evaluate whether an alteration in second sprint duration would affect performance recovery

3. To assess the effects of a simulated competition on repeated-sprint performance and to examine whether any concurrent changes in physiological stress markers would occur

Chapter 3: General methods

3.1. PARTICIPANTS, HEALTH AND SAFETY CONSIDERATIONS, ETHICAL APPROVAL, AND CONSENT FORMS

All participants in all studies were men that were regularly undertaking strength, power, or sprint training. The sample size for each study was determined considering previous research (effect size and sample size) and resource constraints. All participants were volunteers and were recruited from both St Mary's University and local gymnasiums. No financial incentives were provided in Study 1 (Chapter 4). A £100 reward voucher was offered as a prize for the best performer (greatest MPO relative to body-mass over all trials) in Study 2 (Chapter 5) and two £100 reward vouchers were offered as prizes in Study 4 (Chapter 7). All vouchers were supplied by St Mary's University. Each participant was also provided with a personalised athlete report. Prior to any data collection for every study, ethical approval was granted by St. Mary's University Ethics Committee (Code of Approval Study 1: SMUETHICS202021009; Code of Approval Study 2: SMUETHICS202223254; Code of Approval Study 3: SMUETHICS202223254; Code of Approval Study 4: SMUETHICS202223024). The participants were also always informed about the aims, the risks, and the benefits of taking part and were required to complete a pre-activity readiness questionnaire, an informed consent form, and for Study 2 (Chapter 5) an additional COVID-19 declaration form (see Appendix). The participants were free to withdraw from any study at any time, if they so wished.

3.2. APPARATUS AND MATERIALS

3.2.1. Anthropometric Measurements

Stature, body-mass, and body-fat were determined in every study. Stature was assessed using a stadiometer (Harpenden Portable Stadiometer, Holtain Limited, Crymch, United Kingdom), with measurements being made to the nearest mm. Body-mass was measured using an electronic weighing scale (Marsden DP2400 V5 BMI indicator, Marsden Weighing Machine Group Ltd, Rotherham, United Kingdom), with measurements recorded to one decimal place in kilograms. Finally, body-fat was determined via skinfold measurements using Harpenden callipers (Baty International, Burgess Hill, United Kingdom). Two measurements were taken at each location (sub-scapular, supra iliac, biceps, and triceps), but if the repeated measurements differed by more than 10% at any location, an additional measurement was made. The formula developed by Durnin and Womersley (1974) was then used to estimate body-fat percentage.

3.2.2. Cycle Ergometer

All cycling tests were performed on an electromagnetically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands). The ergometer recorded power output at ~ 5 Hz, with MPO and PPO being calculated for each sprint. MPO was the average power output over the entire sprint and PPO was the highest value recorded. The cycle ergometer was put in Wingate mode for all sprints, and in the first three studies, the torque factor was set relative to body-mass, whereas in the final study, the resistive load was individually determined using a torque optimisation test (see Glaister et al., 2019). In each study, the participants were also asked in the first trial to indicate their preferred leg to initiate the sprints (they were given the opportunity to practice initiating sprints with both legs). Consistent with previous research, each sprint then began with the chosen leg being ~ 45° forward to the vertical axis (Billaut & Basset, 2007). A passive recovery was used between sprints in Study 1 (Chapter 4), whereas in Study 2 (Chapter 5), a mixture of active and passive recovery was contrasted with passive recovery. In Studies 3 (Chapter 6) and 4 (Chapter 7), the mixed recovery protocol from Study 2 (Chapter 5) was then adopted. The intensity of the active recovery protocol was set at 80% LT1, with the objective of maximising lactate clearance.

3.2.3. Blood Sampling

On each testing day, an enzymatic-amperometric blood lactate concentration analyser (Biosen C-Line, EFK Diagnostics, Barleben, Germany) was first calibrated with a sample containing a known concentration. Latex free gloves were worn throughout the trials and were disposed of after use. For each participant, the earlobe (the sample site) was cleaned with an alcohol swab before being punctured with a single use disposable lancet (Safe-T Pro Plus, Roche Holding, Basel, Switzerland). The first drop of blood was wiped and a 20 μL blood sample was collected in an end-to-end capillary tube (Sanguis Counting, Nümbrecht, Germany). The sample was immediately placed into a capsule that contained 1 mL of haemolysing solution, before being analysed for lactate concentration.

3.2.4. Oxygen Uptake/Muscle Oxygenation

In Study 1 (Chapter 4), a facemask and head-cap assembly were worn (Hans Rudolph, Kansas City, USA) and connected to an Oxycon Pro metabolic cart (Erich Jaeger GmbH, Hoechburg, Germany) for the breath-by-breath measurement of $\dot{V}O_2$. Prior to each trial, the flow sensor was calibrated using a multiflow 3-L syringe and the gas analyser was calibrated using gases of a known concentration (16% O_2; 5% CO_2), as well as the ambient conditions (temperature, pressure, and relative humidity) at the time of testing. A wireless NIRS transmitter (PortaMon, Artinis Medical

Systems, Elst, The Netherlands) was also positioned above the *vastus lateralis* of the non-drive sprint initiation leg and TSI, O_2Hb, HHb, and tHb were recorded. The sensor placement was set using guidelines (Hermens et al., 1999). Prior to application of the sensor, skinfold thickness was measured using callipers at the placement site. Next, the sensor location was shaved and cleaned with an alcohol swab, before a clear film (Tegaderm, 3M, St. Paul, U.S.) was placed on the thigh and strong adhesive tape used to hold the sensor in place. A black cloth was then taped over the sensor to limit external light sources affecting the reading.

3.2.5. Saliva Sampling

Saliva sampling was conducted in Study 4 (Chapter 7). For each sample, a collection aid was first attached to a 2 mL cryovial (Salimetrics, Pennsylvania, USA). Prior to collecting the first sample, the participants were asked to rinse their mouth with water. Rinsing the mouth with water was conducted to minimise sample contamination from food particles and bacterial growth (Salimetrics, 2019). The participants were also asked not to eat or drink (other than water) for at least two hours before testing and to refrain from caffeine or alcohol for 12 hours. Ten minutes after rinsing their mouth with water, the participants were then asked to swallow any saliva that was in their mouth and to sit with their head tilted forward allowing/aiding any saliva that gathered in their mouth to flow into the collection aid (passive drooling) (Salimetrics, 2021). Following the trial, the samples were weighed before being evenly divided into two vials, with one vial acting as a backup in case of any issues arising during the analysis. All vials were placed into the freezer, which was set at -80°C, until they were analysed for AA activity and output.

3.2.6. Heart Rate Monitoring

Sensors on the heart rate strap (H10, Polar Electro Oy, Kempele, Finland) were first moistened before the strap was positioned securely just beneath the sternum. The sensor was then connected via Bluetooth to a heart rate monitor (V800, Polar Electro Oy, Kempele, Finland). During the lactate profile tests (Studies 2 and 4 or Chapters 5 and 7), a heart rate measurement was recorded at the end of each stage. The recording was made to provide the participants with additional useable data within their athlete report. For the assessment of HRV in Study 4 (Chapter 7), after the heart rate sensor was attached and synced, the participants were asked to sit down on a chair and relax. Following five minutes of seated rest, R-R intervals were recorded for six minutes. Additional measurements were also recorded throughout the control and simulated competition trials.

3.2.7. Measurement Scales

In Study 2 (Chapter 5), three separate 20 cm VAS were used to assess preparedness to sprint, perceptions of recovery, and sprint performance ratings (see Appendix). The preparedness to sprint scale ranged from “not at all”, indicating that the participants did not feel at all prepared to perform their best maximal effort 18 s sprint, to “completely”, suggesting that they felt totally prepared to perform their best possible maximal effort 18 s sprint. The sprint performance scale ranged from “poor”, a very bad reflection of their best sprint performance, to “optimal”, an excellent reflection of their best sprint performance. Finally, the perceptions of recovery scale ranged from “not at all recovered”, reflective of their recovery status at the end of the first sprint, to “completely recovered”, suggesting that they believed that they could perform another maximal effort 18 s sprint, matching their previous effort. The participants were advised that there were no right or wrong responses to any of the scales and that they should place a mark that best represented their feeling at that time. In Study 4 (Chapter 7), a seven-point Likert scale was also completed during the final trial. The scale assessed the perceived impact that the five motivational factors (crowd presence, leaderboard, performance feedback, financial reward, and verbal encouragement), that were included in the simulated competition, had had on performance. The scale ranged from a “large negative effect” (-3) to a “large positive effect” (+3), with “no effect” (0) at the mid-point (see Appendix).

3.2.8. Interviews and Questionnaires

In Study 2 (Chapter 5), at the end of each trial, a post-trial questionnaire was completed on a computer (see Appendix). The questionnaire asked the participants how satisfied they were with their sprint performances that day, what they felt may have mostly influenced their performances, and how they felt that the recovery activity affected their second sprint performance. After the final trial, an additional questionnaire was completed (see Appendix). The end-of-study questionnaire asked the participants why they had chosen to participate, what factors they felt had mostly influenced their approach to the sprints, and the extent to which the offer of a financial reward had affected their approach to the sprints. The questionnaire data were subsequently organised inductively into domain themes (Meijen et al., 2022). A very brief interview was also conducted at the end of the Study 4 (Chapter 7). In the interview, the participants were asked about the reasons behind the markings that had been provided for the Likert scale ratings. The responses were recorded and later transcribed, before being considered alongside the ratings provided.

3.2.9. Statistical Procedures

For all interval data, the assumptions for the use of parametric statistical tests were first assessed. If any assumptions were violated, either data transformations were performed or non-parametric statistical procedures were used. When ordinal data were recorded, non-parametric statistical procedures were used. Data values were reported as mean ± standard deviation and the significance level for all studies was set *a priori* at $p < 0.05$. Statistical analyses were conducted using SPSS® software (SPSS for Windows Version 24 – 28, SPSS Inc, Chicago, USA). Effect sizes were also calculated and reported.

Chapter 4: The Short-term Recovery of Sprint Cycling Performance: Modelling Performance Recovery and Assessing Physiological Processes that Could Influence the Performance Recovery Time-course

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4.1. ABSTRACT

Short-term sprint cycling performance recovery was investigated with consideration to the Match Sprint. Fifteen strength-trained men (age: 24 ± 6 years; height: 1.81 ± 0.08 m; body mass: 83.4 ± 8.4 kg) were first familiarised with an 18 s sprint. During the baseline trial, blood lactate concentration, tissue saturation index (TSI), and oxygen uptake ($\dot{V}O_2$) were monitored following a single sprint. In the remaining trials, the recovery duration (45, 90, 135, 180, 360, and 720 s) between two sprints was varied. Mean power output (MPO) and peak power output (PPO) were measured for each sprint. The recovery percentage of MPO and the recovery time-course of the physiological variables were modelled using one- and two-phase exponential functions. Effects of sprint number (MPO: $F_{(1,14)} = 66.901$, $p < 0.001$, $\eta_p^2 = 0.827$; PPO: $F_{(1,14)} = 73.177$, $p < 0.001$, $\eta_p^2 = 0.839$), recovery time (MPO: $F_{(5,70)} = 36.294$, $p < 0.001$, $\eta_p^2 = 0.722$; PPO: $F_{(5,70)} = 4.975$, $p = 0.001$, $\eta_p^2 = 0.262$), and a sprint number × recovery time interaction (MPO: $F_{(2.160,30.242)} = 52.095$, $p < 0.001$, $\eta_p^2 = 0.788$; PPO: $F_{(2.617,36.639)} = 10.553$, $p < 0.001$, $\eta_p^2 = 0.430$) were found on both MPO and PPO. *Post hoc* tests revealed significant differences between sprints at all time-points for both variables. The parameters of the one-phase exponential function (A_0 = 97.4 ± 2.5%, τ_0 = 130.6 ± 95.6 s) suggested that performance recovery had stabilised within the 12-minute recovery period. However, the parameters of the two-phase function indicated that recovery was incomplete (A_0 = 87.7 ± 6.4%, A_1 = 11.9 ± 5.2%, τ_0 = 56.3 ± 33.3 s, τ_1 = 458.2 ± 283.3 s). The time constant for MPO recovery was not significantly correlated with any of the physiological variables. Therefore, the main finding from the study was that performance during a second sprint was reduced irrespective of the recovery duration. As a 12-minute recovery period may occur in the Match sprint competition, athletes may need to consider means of enhancing the short-term recovery process. There was, however, no evidence to suggest that individuals with a higher aerobic capacity, or those that experience faster muscle reoxygenation or lactate clearance, displayed a faster performance restoration-rate.

4.2. INTRODUCTION

Track cycling meetings include both sprint and endurance races. The Match Sprint is a sprint cycling discipline. At the 2020 Tokyo Olympics the Match Sprint competition occurred over three days, with a single race session on each day (Maia, 2021). During each race session the riders competed between one and five times. The average time between races was 48 ± 23 minutes, although in the final gold medal decider, Jeffrey Hoogland and Harrie Lavreysen had just over 15 minutes to recover from their previous heat. In fact, on 15 occasions riders had less than 30 minutes between races. At the 2016 Rio Olympics, the New Zealand rider, Eddie Dawkins, lost in Heat 9 in the 1/16 round and then competed in the first of the repechages. The schedule indicated that he had ~ 10 minutes between these races (Vieria, 2016). The short-term recovery of sprint cycling performance has been investigated using brief (1 s – 30 minutes) recovery time-periods (Ainsworth et al., 1993; Bogdanis et al., 1995; Bogdanis et al., 1996b; Cherry et al., 1998; Esbjörnsson-Liljedahl et al., 2002; Glaister et al., 2014; Hebestreit et al., 1993; Kirkpatrick & Burrus, 2020; Zabala et al., 2008; Zabala et al., 2011). However, only one study has assessed the kinetics of performance recovery using mathematical modelling (Glaister et al., 2014).

Unlike modelling the recovery of physiological responses, which can be monitored following a single bout of exercise, modelling the recovery of sprint performance requires each rest interval to be assessed during a separate trial (Glaister et al., 2014). In the study by Glaister et al. (2014), the participants were first required to perform a 30 s fatiguing sprint, which was followed by a short 5 s sprint after 5, 10, 20, 40, 80, or 160 s of recovery. The recovery of PPO was then modelled using one- and two-phase exponential functions. The authors reported model parameters for the two-phase exponential functions for the group mean response, as well as the mean and standard deviation of the parameters derived for the individual participants. From the information provided, it could be estimated that PPO would be fully restored after ~ 8 – 15 minutes. In support, Zabala et al. (2011) found that PPO did not differ between three WAnTs performed 15 minutes apart, and Hebestreit et al. (1993) found no difference in PPO between two WAnTs, when 10 minutes separated the tests. However, it should be noted that whilst PPO is considered to be a key metric for sprint cycling performance, it may not accurately reflect overall performance, especially when considering the repeated efforts that are required during a competition (Ferguson et al., 2021). When two WAnTs were undertaken 10 minutes apart, the total work performed during the second sprint was found to be significantly reduced (Hebestreit et al., 1993). That being said, whilst the WAnT is the most commonly used measure of sprint cycling performance (Jaafar et al., 2014), it may not provide the best reflection of the effort required for sprints in the Match Sprint competition, which could also affect the recovery time-course.

The duration of maximal effort for each sprint in the four sprint cycling disciplines will likely be between 15 s and 60 s (Douglas et al., 2021; Ferguson et al, 2021), with the Kilo requiring ~ 60 s to complete (at the elite level). The Match Sprint is, however, a highly tactical event, with the duration of maximal effort varying in each race depending on the tactics employed. When 15 s of maximal exercise is undertaken, it has been estimated that 88% of the energy used would be derived from anaerobic pathways, decreasing to 82% for a 20 s sprint, 73% for a 30 s sprint, and 55% for a 60 s sprint (Gastin, 2001). The anaerobic energy systems will, therefore, contribute heavily to sprint performance. However, as PCr resynthesis occurs via the rephosphorylation of creatine by aerobically produced ATP (McMahon & Jenkins, 2002), individuals with faster muscle reoxygenation or faster $\dot{V}O_{2off}$ kinetics could perform better during a repeated-sprint task. $\dot{V}O_{2off}$ kinetics have been found to follow a similar time-course to PCr resynthesis (Rossiter et al., 2002), at least at exercise intensities that are below $\dot{V}O_{2max}$ (Glaister et al., 2014). NIRS also facilitates the non-invasive measurement of O_2 delivery and utilisation directly at the muscle, meaning that NIRS may offer a preferred means of assessing muscle reoxygenation (Ufland et al., 2013). PCr resynthesis (McMahon & Jenkins, 2002; Walter et al., 1997), as well as the glycolytic pathway (Bogdanis et al., 1996b), could also be affected by muscle pH. The cause of muscular acidosis during exercise, and the role that lactate and H^+ production play in muscular fatigue, have been well documented and debated (Allen et al., 2008; Fitts, 2016; Robergs, 2019; Robergs et al., 2023; Robergs et al., 2024; Westerblad, 2016). The time-course of lactate recovery and performance restoration is different. Nonetheless, it is still possible that accumulating H^+ could limit sprint performance, meaning that faster lactate and H^+ clearance could be beneficial for repeated-sprint tasks.

The aim of the current study was, therefore, to model the recovery of sprint cycling performance and to assess physiological variables that may influence the recovery time-course. It was hypothesized that MPO would be reduced at all recovery time-points and that the time constant for $\dot{V}O_{2off}$ kinetics, TSI, and the lactate clearance velocity constant would correlate with the rate of performance restoration.

4.3. MATERIALS & METHODS

4.3.1. Participants

Fifteen healthy resistance-trained men over 18 years of age participated (age: 24 ± 6 years; height: 1.81 ± 0.08 m; body-mass: 83.4 ± 8.4 kg; body-fat: 14.8 ± 4.5%; $\dot{V}O_{2peak}$: 48.1 ± 6.3 $mL \cdot kg^{-1} \cdot min^{-1}$). To aid with the consistency in testing conditions, the trials were conducted at approximately the same time of day, and the participants were required to refrain from conducting any

strenuous exercise for 24 hours, ingesting caffeine for 12 hours, and consuming food for three hours prior to each trial. Before commencing the first trial, all participants were informed about the risks and benefits of taking part. The participants also provided written informed consent and were advised that they were able to withdraw from the study at any time. The study was conducted in accordance with the Declaration of Helsinki and was granted approval by St Mary's University Ethics Committee (London, United Kingdom) (see Appendix).

4.3.2. Design

4.3.2.1 Experimental Approach

All participants visited the laboratory on eight occasions. The order of the experimental conditions (Trials 3 – 7) was randomised for all recovery periods except the longest recovery duration (720 s), which was always the final trial (Trial 8). This approach was taken to facilitate the comparison of the physiological variables between the baseline trial (Trial 2) and the end of the study. The recovery periods that were used were selected considering the format employed by Glaister et al. (2014), who previously modelled the recovery time-course of PPO.

4.3.2.2. Trial 1

The aim of Trial 1 was to familiarise the participants with the cycle ergometer and the requirements of the sprint. Measurements of stature, body-mass, and body-fat were first taken and the cycle ergometer was adjusted for each participant, with the setup (seat and handlebar height, as well as fore-aft positions) being recorded to facilitate replication during the subsequent trials. The participants were asked to indicate their preferred leg to initiate the sprints, with each sprint in all trials beginning with the chosen leg being ~ 45° forward to the vertical axis. A wireless NIRS transmitter (PortaMon, Artinis Medical Systems, Elst, The Netherlands) was positioned above the *vastus lateralis* of the non-drive initiation leg for the assessment of TSI. A facemask and head-cap assembly were worn (Hans Rudolph, Kansas City, USA) for the breath-by-breath measurement of $\dot{V}O_2$ (Oxycon Pro, Erich Jaeger GmbH, Hoechburg, Germany). The participants then sat passively on the cycle ergometer for five minutes before performing a standardised warm-up (see Table 4.01). After the warm-up, and following three minutes of passive rest, the participants performed an 18 s all-out effort against a resistive load of 0.909 $Nm \cdot kg^{-1}$. The resistance was greater than the standard WAnT load, as it has been suggested that strength-trained individuals require a greater resistance to optimise performance (Pazin et al., 2011). All sprints were undertaken from a stationary start and the participants were instructed to remain seated throughout. Strong verbal encouragement was provided

during each sprint. After five minutes of passive rest, a ramp test (starting load 50 – 75 W, ramp rate 25 $W \cdot min^{-1}$) was performed to exhaustion for the determination of $\dot{V}O_{2peak}$ (highest 30 s rolling average).

Table 4.01 Warm-up and sprint protocol.

Duration (s)	Resistive Load ($Nm \cdot kg^{-1}$)	Instruction
300	0	Passive rest
120	0.187	Comfortable cadence 60-90 rpm
5 (rolling start sprint)	0.47	Spin as fast as possible
50	0.187	Comfortable cadence 60-90 rpm
5 (rest)	0*	Get into start position
5 (stationary start sprint)	0.47	Drive as hard as possible
50	0.187	Comfortable cadence 60-90 rpm
5 (rest)	0*	Get into start position
5 (stationary start sprint)	0.47	Drive as hard as possible
55	0.187	Comfortable cadence 60-90 rpm
180	0	Passive rest
18 s	0.909	Maximum Effort

Note: * prior to each sprint a resistance of 1000 W was briefly applied to stop the flywheel from moving.

4.3.2.3. Trial 2

During Trial 2 (baseline trial), the participants performed the standardised warm-up and a single 18 s sprint, which was followed by 12 minutes of passive recovery. TSI and breath-by-breath $\dot{V}O_2$ were recorded throughout the recovery period. A 20 µl capillary blood sample was also taken from the earlobe at rest, following the warm-up, 30 s after the sprint, and every minute thereafter for a further ten samples. The samples were subsequently analysed for blood lactate concentration.

4.3.2.4. Trials 3 – 8

Following the same warm-up and sprint protocol as before (Trials 1 and 2), a second 18 s sprint was performed. As the information provided could affect repeated-sprint performance (Billaut et al., 2011), the participants were not informed about the recovery duration between sprints until the first sprint had been undertaken. Between sprints the participants rested passively on the ergometer. Following the second sprint, the participants remained on the ergometer for a further five minutes before performing a self-selected cool down. During the 720 s trial, blood lactate concentration, TSI, and breath-by-breath $\dot{V}O_2$ were monitored following the first sprint. A schematic of the experimental protocol is displayed in Figure 4.01.

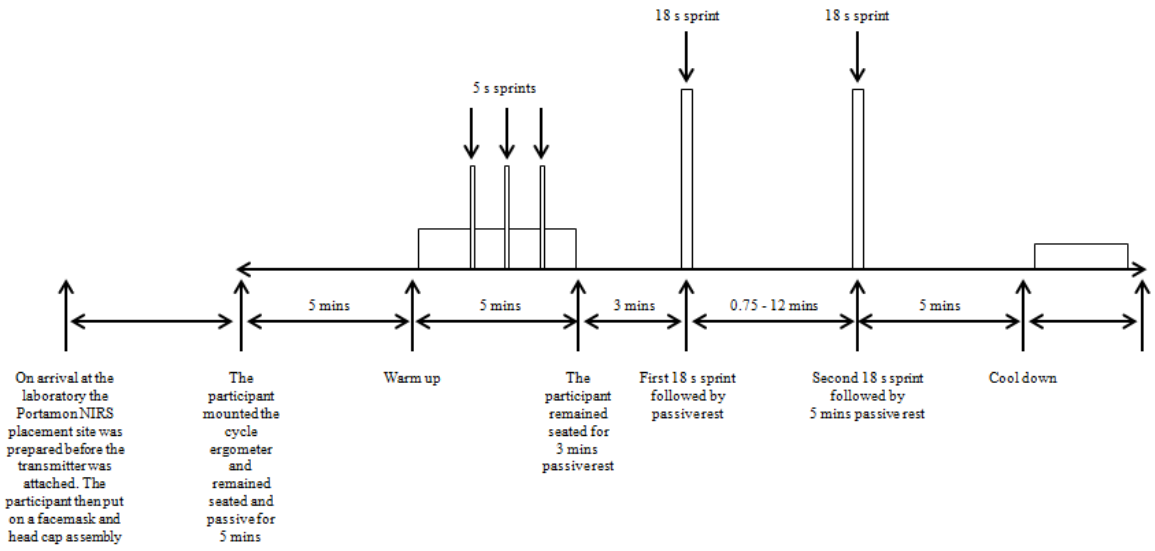

Figure 4.01 Schematic of the experimental protocol used. Note: NIRS denotes near infrared spectroscopy and mins is minutes.

4.3.3. Data Analysis

4.3.3.1. Performance

MPO was the average power output over the entire sprint and PPO was defined as the highest value recorded during the sprint. Performance recovery percentage was calculated as Sprint 2 performance (MPO for the 18 s sprint) relative to Sprint 1 (Hebestreit et al., 1993). The time-course of the recovery percentage of MPO was then modelled using both one- and two-phase exponential functions (see Equations 4.01 & 4.02), with the goodness of model fit computed by the software (Matlab R2019a, Mathworks, Natick, U.S.).

$$PR(t) = (A_0 \times \left(1 - e^{-\frac{(t-b_0)}{\tau_0}}\right) \times U_0) \qquad \text{Equation 4.01}$$

$$PR(t) = (A_0 \times \left(1 - e^{-\frac{(t-b_0)}{\tau_0}}\right) \times U_o) + (A_1 \times \left(1 - e^{-\frac{(t-TD_1)}{\tau_1}}\right) \times U_1) \qquad \text{Equation 4.02}$$

where *PR(t)* is the performance recovery percentage of mean power output at any time-point, A_0 and A_1 represent the asymptotic amplitudes for the exponential terms, b_0 allowed the function to begin from an elevated level at t = 0, TD_1 represents the time delay of the secondary phase, and τ_0 and τ_1 are the time constants. $U_0 = 0$ when $t < 0$ and $U_0 = 1$ when $t \geq 0$. $U_1 = 0$ when $t < TD_1$ and $U_1 = 1$ when $t \geq TD_1$.

4.3.3.2. Blood Lactate Concentration

A two-phase exponential function (see Equation 4.03) was used to model the blood lactate response in recovery (Beneke et al., 2010). The model also allowed the maximum post-sprint blood lactate concentration, and the time when the maximum concentration occurred, to be determined (Beneke et al., 2010).

$$BLC(t) = \frac{A*k_0}{k_1-k_0} \times \left(e^{-k_0 t} - e^{-k_1 t}\right) + \left(BLC_{pre\ sprint} - BLC_{rest}\right) \times e^{-k_1 t} + BLC_{rest} \quad \text{Equation 4.03}$$

where *BLC(t)* represents the blood lactate concentration at any time-point, BLC_{rest} is the blood lactate concentration at rest, and $BLC_{pre\ sprint}$ is the concentration measured after the warm-up. *A* represents the approximate increase in blood lactate concentration following the sprint, k_0 is the appearance velocity constant, and k_1 is the disappearance velocity constant.

4.3.3.3. Near Infrared Spectroscopy

The NIRS analysis was limited to TSI due to the confounding effect of blood flow during exercise on other variables (Dupont et al., 2007). The recovery data for TSI were then modelled using Equation 4.04 and the MRT was calculated using Equation 4.05.

$$TSI(t) = TSI_{end} + \left(A_0 \times \left(1 - e^{-\frac{(t-TD_0)}{\tau_0}}\right) \times U_0\right) \quad \text{Equation 4.04}$$

$$MRT = TD_0 + \tau_0 \quad \text{Equation 4.05}$$

where *TSI(t)* is the tissue saturation index over time, TSI_{end} is the end sprint TSI value, A_0 represents the asymptotic amplitude for the exponential term, TD_0 the time delay, and τ_0 the time constant. U_0 = 0 when $t < TD_0$ and $U_0 = 1$, when $t \geq TD_0$. MRT is the mean response time.

4.3.3.4. Oxygen Uptake

$\dot{V}O_2$ recovery data were analysed by first removing any errant breaths (values outside of four standard deviations of the local mean – the two breaths preceding and following the breath of interest) and then linearly interpolated to give second-by-second values (Dale & Glaister, 2018). The data were modelled from the peak post-sprint value using Equation 4.06. Only one time delay was included in the model as it has been suggested that both fundamental and slow components would be in operation at the completion of exercise (Özyener et al., 2001). Constraints were applied to Equation 4.06 to ensure that the eventual resting value would be physiologically viable. This was achieved by using the resting $\dot{V}O_2$ value for each participant.

$$\dot{V}O_2(t) = \dot{V}O_{2end\,peak} - (A_0 \times \left(1 - e^{-\frac{(t-TD_0)}{\tau_0}}\right) \times U_0) - (A_1 \times \left(1 - e^{-\frac{(t-TD_0)}{\tau_1}}\right) \times U_0) \quad \text{Equation 4.06}$$

where $\dot{V}O_2(t)$ is the oxygen uptake at any time-point, $\dot{V}O_{2end\,peak}$ is the highest post-sprint value, A_0 and A_1 represent the asymptotic amplitudes for the exponential terms, τ_0 and τ_1 are the time constants, and TD_0 is the time delay. $U_0 = 0$ when $t < TD_0$ and $U_0 = 1$ when $t \geq TD_0$.

4.3.4. Statistical Analysis

Statistical analyses were conducted using SPSS® software (SPSS for Windows Version 24, SPSS Inc, Chicago, USA). Values are reported as mean ± standard deviation. Statistical significance was set *a priori* at $p < 0.05$. Differences in both MPO and PPO were assessed using a two-way (sprint number × recovery time) ANOVA. If the assumption of sphericity was not satisfied, the Greenhouse-Geisser correction was applied. Where required, *post hoc* analyses were conducted using a Bonferroni correction. Correlation analyses (Pearson's r/Spearman's rho - dependent on whether the assumptions of normality were satisfied) were conducted to assess the relationship between τ for performance recovery and the recovery τ of TSI ($TSI\tau_0$) and $\dot{V}O_2$ ($\dot{V}O_{2off}\tau_0$), as well as $\dot{V}O_{2peak}$, the MRT for TSI (TSI_{MRT}), and the clearance velocity constant for blood lactate ($BLCk_1$). The magnitude of each relationship was interpreted using guidelines (Hopkins et al., 2009). To assess for a performance training effect a one-way ANOVA was performed on MPO and PPO in trial order. The coefficient of variation and the intraclass correlation coefficient (ICC), as well as their 95% confidence limits, were then calculated using recommended procedures (Schabort et al., 1999). Finally, in order to evaluate the consistency of the model parameters of the physiological variables ($BLCk_1$, TSI_{MRT}, $TSI\tau_0$, $\dot{V}O_{2off}\tau_0$) between the baseline and final trials, the coefficient of variation was computed using established methods, as outlined elsewhere (Buchheit et al., 2011).

4.4. RESULTS

4.4.1. Performance

The performance recovery percentage modelled with both one- and two-phase exponential functions, is displayed in Figure 4.02. The model parameters are displayed in Table 4.02. A two-phase exponential function improved the goodness of model fit on ten occasions. However, a one-phase exponential function was appropriate for the other five data sets. The performance recovery response from four representative participants (two where the model fit was improved with a two-phase exponential function and two where the model reverted to a one-phase exponential function), is

displayed in Figure 4.03. To ensure continuity, all correlations with the physiological variables were made using τ_0 derived from the one-phase exponential function.

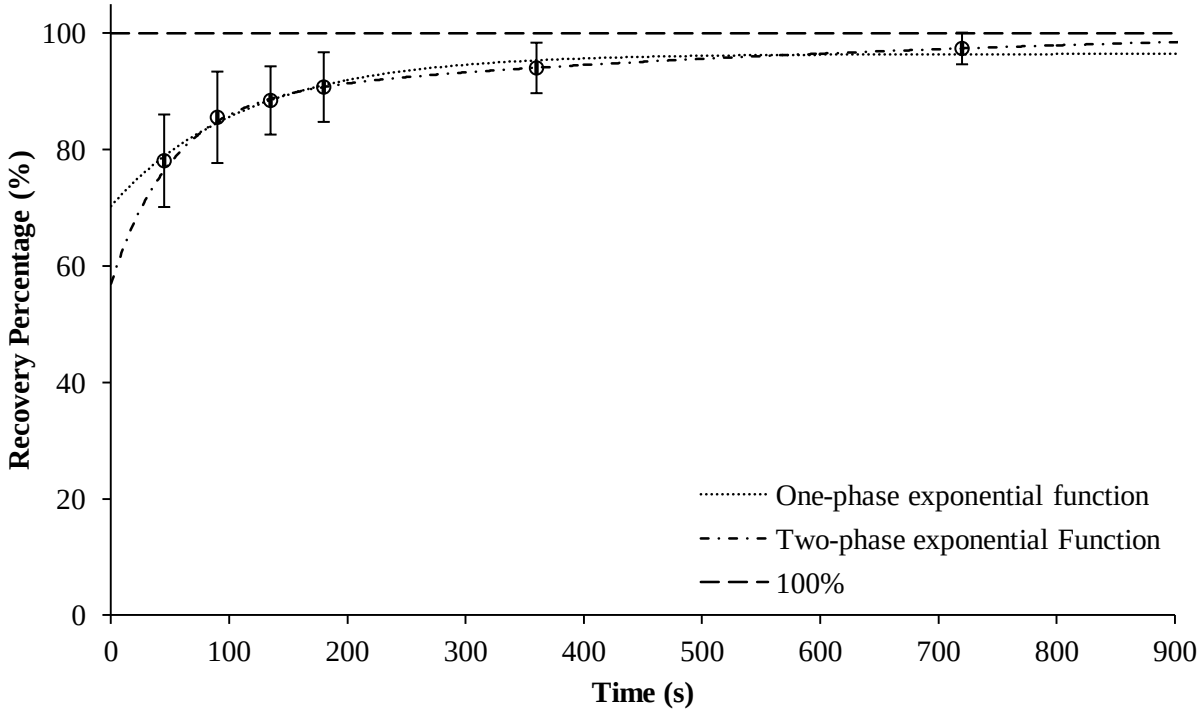

Figure 4.02 The recovery percentage of mean power output recorded at six recovery time-points fit with both one- and two-phase exponential functions. Data points display the mean and error bars the standard deviation.

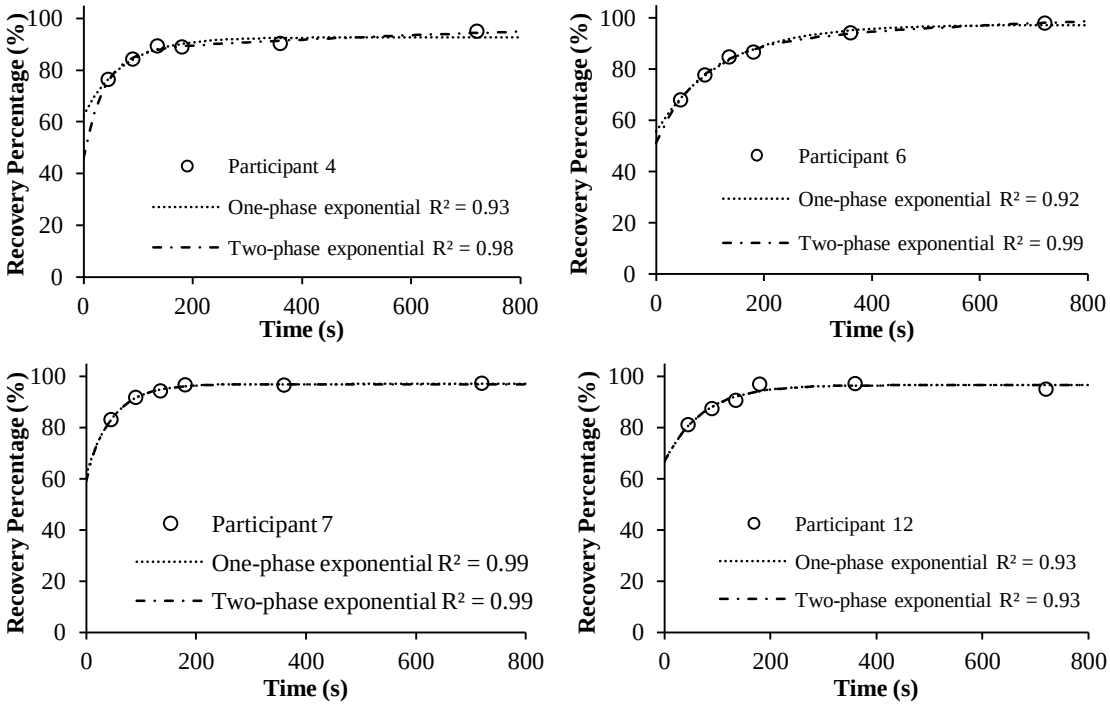

Figure 4.03 Example recovery percentage data for four representative participants. For participants 4 and 6, the model fit was improved with a double exponential function, whereas for participants 7 and 12, the model fit was not improved.

Table 4.02 Model parameters and the goodness of model fit for one- and two-phase exponential functions fit to the recovery performance data. Data are displayed as mean ± standard deviation.

	A_0 (%)	τ_0 (s)	b_0 (s)	A_1 (%)	τ_1 (s)	TD_1 (s)	R^2
One-phase exponential	97.4 ± 2.5	130.6 ± 95.6	-156.1 ± 132.5	-	-	-	0.91 ± 0.10
Two-phase exponential	87.7 ± 6.4	56.3 ± 33.3	-81.9 ± 31.6	11.9 ± 5.2	458.2 ± 283.3	73.9 ± 108.0	0.92 ± 0.10

Note: A denotes the asymptotic amplitude, τ the time constant, b is a constant that allowed the model to begin at a recovery percentage that was greater than 0%, and TD is the time delay.

An effect of sprint number (MPO: $F_{(1,14)}$ = 66.901, p < 0.001, η_p^2 =0.827; PPO: $F_{(1,14)}$ = 73.177, p < 0.001, η_p^2 = 0.839), recovery time (MPO: $F_{(5,70)}$ = 36.294, p < 0.001, η_p^2 = 0.722; PPO: $F_{(5,70)}$ = 4.975, p = 0.001, η_p^2 = 0.262), and a sprint number × recovery time interaction (MPO: $F_{(2.160,30.242)}$ = 52.095, p < 0.001, η_p^2 = 0.788; PPO: $F_{(2.617,36.639)}$ = 10.553, p < 0.001, η_p^2 = 0.430) were found on both MPO and PPO. *Post hoc* tests revealed significant (p < 0.05) differences between Sprint 1 and Sprint 2 for both MPO and PPO at all recovery time-points (see Figures 4.04 & 4.05).

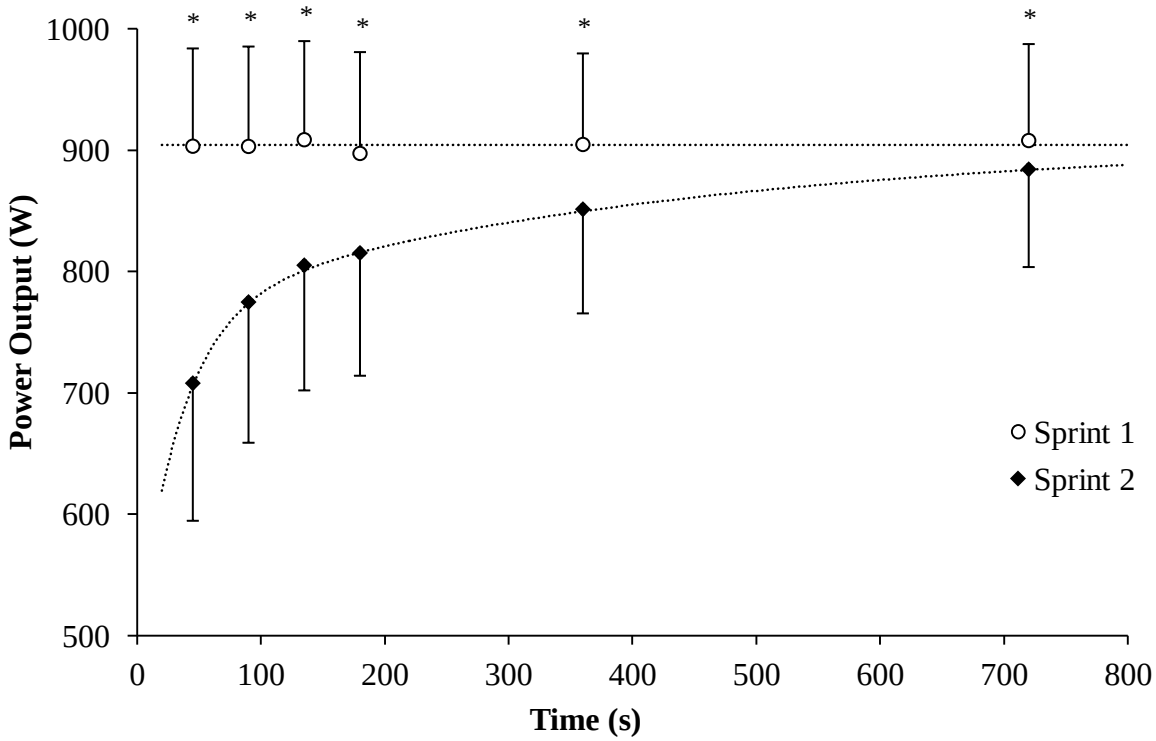

Figure 4.04 Sprint 1 and Sprint 2 mean power output at the six recovery time-points. Displayed data represent the mean and error bars the standard deviation. Lines of best fit (linear and two-phase exponential) have been added. Note: * denotes a significant difference ($p < 0.05$) in mean power output between sprints at the individual time-point.

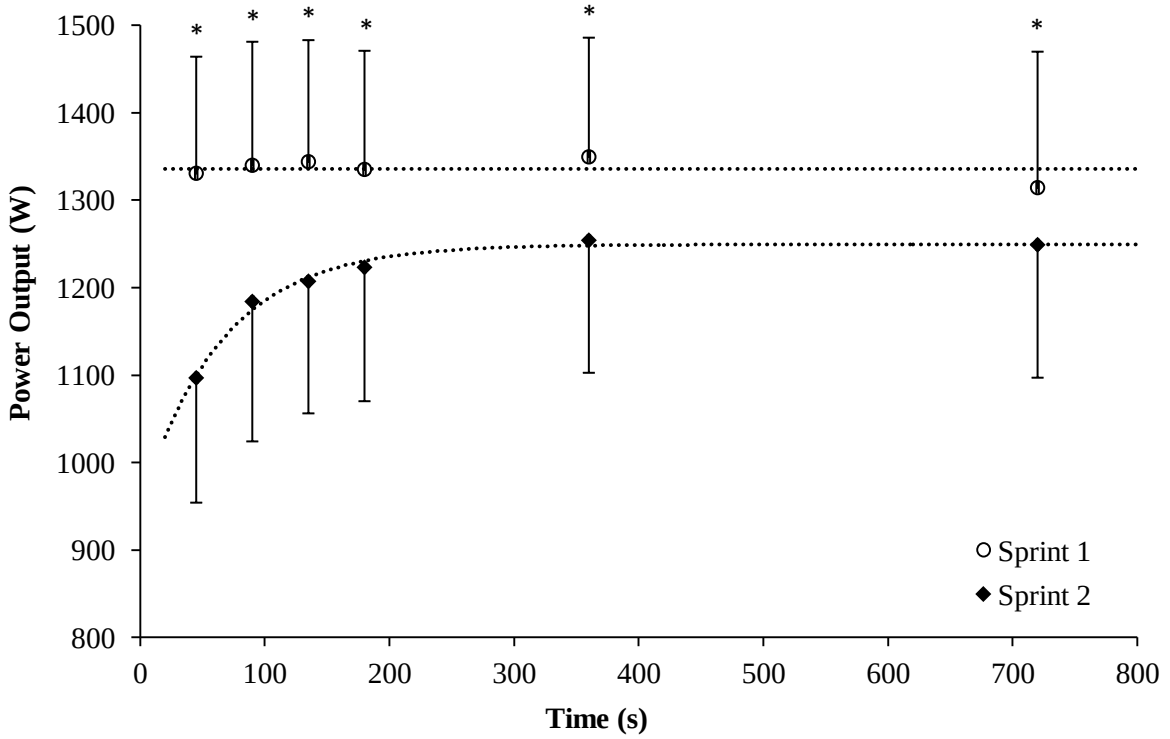

Figure 4.05 Sprint 1 and sprint 2 peak power output at the six recovery time-points. Data points represent the mean and error bars the standard deviation. Lines of best fit (linear and one-phase exponential) have been added. Note: * denotes a significant difference ($p < 0.05$) in peak power output between sprints at the individual time-point.

When the first sprint was analysed in trial order, differences in both MPO ($F_{(2.626,\ 36.768)}$ = 1.621, p = 0.205) and PPO ($F_{(3.107,\ 43.505)}$ = 0.546, p = 0.659) were not significant. The coefficient of variation for MPO was 2.1% [1.8, 2.5] and 4.6% [4.0, 5.4] for PPO, with an ICC of 0.95 [0.90, 0.98] for MPO and 0.82 [0.69, 0.92] for PPO.

4.4.2. Blood Lactate Concentration

The resting blood lactate concentration, as well as the parameters of the modelled response, can be found in Table 4.03. Blood lactate concentration was not measured for one participant due to technical issues and two data sets were not included in the analysis, as the model did not indicate any blood lactate clearance for these individuals. The coefficient of variation between trials for k_1 was 98.0% [66.7, 188.5].

Table 4.03 The model parameters for the rise and clearance of blood lactate concentration following an 18 s sprint, as well as the estimated peak concentration and the time to the peak of the response (n = 12). Data are displayed as mean ± standard deviation.

Resting ($mmol \cdot L^{-1}$)	Pre-sprint ($mmol \cdot L^{-1}$)	A ($mmol \cdot L^{-1}$)	k_0 ($minutes^{-1}$)	k_1 ($minutes^{-1}$)	R^2	Peak Lactate ($mmol \cdot L^{-1}$)	Time to Peak (minutes)
0.94 ± 0.33	3.70 ± 2.03	7.86 ± 2.3	0.74 ± 0.40	0.04 ± 0.02	0.90 ± 0.09	9.63 ± 1.94	4.42 ± 0.93

Note: A denotes the amplitude, k_0 the appearance velocity constant, and k_1 the disappearance velocity constant.

4.4.3. Near Infrared Spectroscopy

At the sensor location, the skinfold thickness was 6.3 ± 1.9 mm. Due to a large overshoot, three data sets were removed from the analysis. The parameters of the model are displayed in Table 4.04. The coefficient of variation between the baseline and final trials was 38.9% [27.1, 71.7] for τ_0 and 24.6% [17.4, 43.6] for the MRT.

Table 4.04 Model parameters and the goodness of model fit for tissue saturation index following an 18 s sprint (n = 12). Data are displayed as mean ± standard deviation.

TSI_{END}	A_0 (%)	τ_0 (s)	TD_0 (s)	MRT (s)	R^2
53 ± 10	28 ± 10	20.3 ± 8.2	11.7 ± 11.0	31.9 ± 13.9	0.88 ± 0.09

Note: TSI_{END} denotes the end of sprint tissue saturation index, A is the asymptotic amplitude, τ the time constant, TD the time delay, and MRT the mean response time.

4.4.4. Oxygen Uptake

$\dot{V}O_2$ was not recorded for one participant due to feelings of constraint when wearing a facemask. The $\dot{V}O_{2peak}$ for the remainder of the group was 48.1 ± 6.3 $ml \cdot kg^{-1} \cdot min^{-1}$. The parameters of the decay function, modelled from the peak of the response, are displayed in Table 4.05. The coefficient of variation between the baseline and 720 s trials for τ_0 was 24.3% [18.1, 38.2].

Table 4.05 Model parameters and the goodness of model fit for oxygen uptake following an 18 s sprint (n = 14). Data are displayed as mean ± standard deviation.

	End Peak ($ml \cdot min^{-1}$)	A_0 ($ml \cdot min^{-1}$)	τ_0 (s)	TD_0 (s)	A_1 ($ml \cdot min^{-1}$)	τ_1 (s)	R^2
Double-exponential	3898 ± 625	2956 ± 622	50.9 ± 13.4	13.8 ± 9.8	488 ± 201	592 ± 822	0.92 ± 0.05

Note: End peak denotes the highest post sprint value, A is the asymptotic amplitude, τ the time constant, and TD the time delay.

4.4.5. Relationship between the performance recovery-rate and the physiological variables

The strength of the relationships between the τ for performance recovery and the physiological variables ($BLCk_1$, TSI_{MRT}, $TSI\tau_0$, $\dot{V}O_{2off}\tau_0$, and $\dot{V}O_{2peak}$) ranged from negligible to moderate, but was never significant (see Figure 4.06).

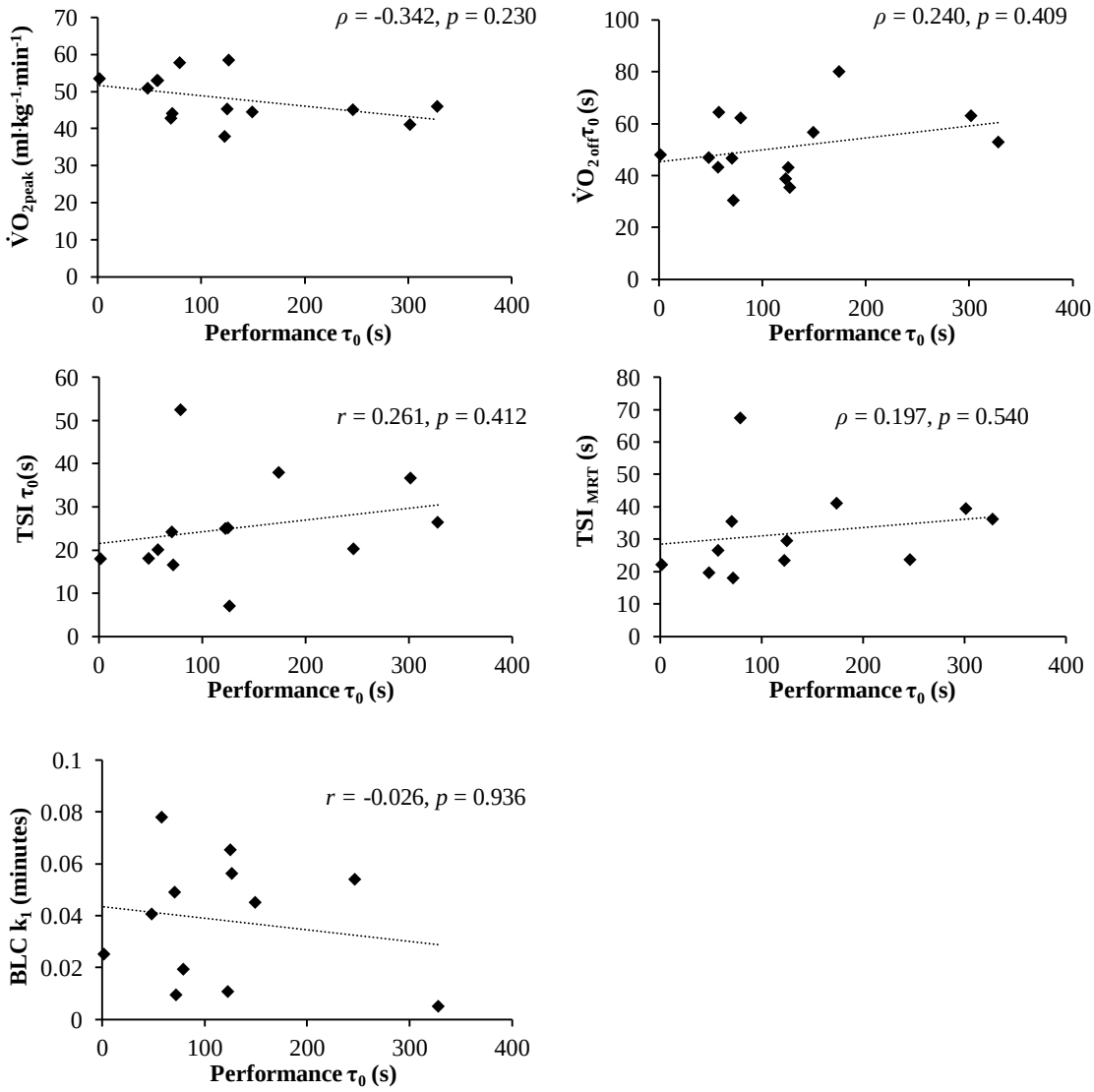

Figure 4.06 Relationship between the performance recovery-rate (the time constant derived from the one-phase exponential function) and the physiological variables ($BLCk_1$,$TSI\tau_0$, TSI_{MRT}, $\dot{V}O_{2off}\tau_0$, and $\dot{V}O_{2peak}$). For oxygen uptake variables n = 14, for tissue saturation index n = 12, and for blood lactate concentration n = 12. Note: $BLCk_1$ denotes the clearance velocity constant for blood lactate, τ is the time constant, TSI is the tissue saturation index, MRT is the mean response time, $\dot{V}O_2$ is oxygen uptake, and $\dot{V}O_{2peak}$ is peak oxygen uptake.

4.5. DISCUSSION

The aim of this study was to evaluate the short-term recovery of sprint cycling performance and to assess physiological variables that may influence the recovery time-course. In comparison to first sprint performance, both MPO and PPO were reduced during the second sprint at all recovery time-points. For the recovery percentage of MPO, a two-phase exponential function improved the goodness of model fit on 67% of occasions. TSI reached steady-state within the 12-minute recorded

period. $\dot{V}O_2$ rose briefly following the sprint, before decreasing rapidly and then more gradually, whereas blood lactate concentration peaked 4.42 minutes after the sprint and then slowly declined over the recovery period. No significant relationships were, however, found between the τ of performance restoration and the recovery of any of the physiological variables.

The parameters of the one-phase exponential function indicated that performance recovery had stabilised within the 12-minute timeframe, albeit performance restoration was not 100%, whereas the parameters of the two-phase exponential function indicated that performance restoration would continue beyond the recovery period that was tested. It has been proposed that several physiological processes, such as PCr resynthesis, recover in a biphasic fashion (Harris et al., 1976; McMahon & Jenkins, 2002; Walter et al., 1997). Therefore, the suggestion that there could be an initial fast phase to performance recovery, followed by a slower secondary phase is not unreasonable. Only one other study has modelled sprint cycling performance restoration. Whilst the authors were specifically interested in the recovery of PPO, a two-phase exponential function was found to significantly improve the goodness of model fit (Glaister et al., 2014), although it should be noted that when one of the models being compared is a more complex version of the other, it should not be possible for the more complex model to worsen the fit (Dale & Glaister, 2018). Nonetheless, in the current study, on ten occasions a two-phase exponential function improved the model fit, whereas on the other five occasions the model reverted to a one-phase exponential function. The collection of additional data points, around or greater than 12 minutes, would have been beneficial. However, comparison of the performance data revealed that both MPO and PPO were significantly reduced at all recovery time-points.

In agreement with the current findings, it has been reported that the total work completed during a WAnT was reduced when 1, 2, or 10 minutes separated the tests, although, in contrast, PPO was fully restored after 10 minutes (Hebestreit et al., 1993). Whilst the smaller sample size (n = 8) may have limited statistical power in the study by Hebestreit et al. (1993), Zabala et al. (2011) found that both MPO and PPO did not differ when three WAnTs were performed 15 minutes apart. The participants in that study were, however, elite BMX cyclists, capable of producing higher PPO than were recorded in the current study. On the one hand, it has been reported that the participants that produced the greatest power output during a WAnT displayed the slowest resynthesis of PCr (Bogdanis et al., 1996b), meaning that the recovery duration could be greater in those individuals, whereas, on the other hand, the type of training undertaken can affect physiological and performance adaptations (Mohr et al., 2007), meaning that the specific training undertaken by elite sprint cyclists could shorten the required recovery time.

The resistive load used, and the duration of the sprint, also differed between the current study and the studies by Hebestreit et al. (1993) and Zabala et al. (2011). Training background has

been found to affect load optimisation during cycling sprints, with greater loads being required for strength- and speed-trained individuals (Pazin et al., 2011). Whilst the exact duration of each sprint will vary race-to-race in the Match Sprint, an 18 s performance measure was selected, as it was estimated that this would be reflective of the maximal effort that is exerted during a Flying 200. Sprint duration has, however, been shown to affect pacing strategy, with a lower PPO being found when the duration increases (de Jong et al., 2015, Glaister et al., 2019; Wittekind et al., 2011). Therefore, the current findings can only be related to the performance measure that was selected. An additional consideration was that there may have been a training/learning effect, as the participants were not familiar with sprint cycling. Strength-trained individuals were recruited as strength training is considered to be essential for cyclists that compete in the Match Sprint (Parsons, 2010). No evidence of a training effect was found, and the coefficient of variation for both MPO and PPO was similar to values that have been reported (Hebestreit et al., 1993). A final consideration, from a performance perspective, was that during the recovery period the participants rested passively on the ergometer. This was required for the NIRS recording, but may not be reflective of the practice undertaken by sprint cyclists.

From a physiological perspective, the relationship between the τ of performance restoration and muscle reoxygenation, $\dot{V}O_{2off}$ kinetics, $\dot{V}O_{2peak}$, and $BLCk_1$, ranged from negligible to moderate, but was never significant. Research findings regarding the relationship between muscle re-oxygenation (Buchheit et al., 2012; Buchheit & Ulfand, 2012), $\dot{V}O_{2off}$ kinetics (Buchheit, 2012; Buchheit et al., 2012), $\dot{V}O_{2max}$ (Buchheit, 2012; Buchheit et al., 2012; Dupont et al., 2010; Glaister et al., 2014), and repeated-sprint performance, have been mixed, although the repeated-sprint protocols used in those studies lacked specificity when considering the Match Sprint competition. The current findings do, however, suggest that these parameters may not be of utmost importance when considering means of enhancing performance restoration. The coefficient of variation for the muscle reoxygenation parameters was similar to values that have previously been reported (Buchheit et al., 2011). The coefficient of variation for $\dot{V}O_{2off}$ kinetics was also not dissimilar to the NIRS variables, but was greater than others have found (Mann et al., 2014), although the intensity of exercise in that study was submaximal. Whilst fewer exercise transitions are typically performed for higher-intensity exercise (Koga et al., 2005), the limited number of breaths taken during a sprint, may lead to greater variability in the post-exercise peak value (the start-point), which could in turn affect the reliability of the recovery time constant. The variability between the measurements of $BLCk_1$ was, however, much larger, raising concerns about the suitability of the modelling procedure that was used. The goodness of model fit for the lactate appearance and disappearance data was comparable to the values that were computed for the other variables and the function was specifically designed to model blood lactate concentration after exercise, beginning from an elevated post warm-up state (Beneke et al., 2010). That being said, blood lactate concentration was monitored for 30 minutes after exercise in that study

(Beneke et al., 2010). Having more data points throughout a transient region may help with the consistency of a measure (Koga et al., 2005). Therefore, recording measurements for a longer recovery duration may help to improve the consistency of the lactate clearance velocity constant.

In summary, the Match Sprint requires athletes to compete on consecutive days and on multiple occasions each day, where the time between races can be as short as 10 minutes. The findings from the current study indicated that performance may be inhibited when sprints are repeated over this time frame, suggesting that riders may need to consider means of enhancing the short-term recovery process. There was, however, no evidence to suggest that individuals with a higher $\dot{V}O_{2max}$, or those that experience faster muscle reoxygenation or lactate clearance, possess a faster performance restoration-rate.

Chapter 5: The Effect of Recovery Activity and Second Sprint Duration on Repeated-sprint Performance

Dale, J., Muniz, D., Cimadoro, G., Meijen, C., & Glaister, M. (2023). Sprint cycling: current practice and motivational considerations for performance recovery. *Journal of Psychophysiology*, *37*(4), 191-203. https://doi.org/10.1027/0269-8803/a000321

5.1. ABSTRACT

The time between races varies in the track cycling competition known as the Match Sprint, but can be as little as 10 – 15 minutes. Both physiological and motivational factors may affect performance recovery. Therefore, this study investigated how the between sprint recovery activity, and an alteration in the duration of the second sprint, affected performance. Twenty-four strength trained men (age: 26 ± 5 years; height: 180.3 ± 6.1 cm; body-mass: 82.3 ± 6.9 kg) participated. During each of the four experimental trials, two sprints were performed 12 minutes apart. The first was always 18 s and the second was either 9 s or 18 s. Between sprints, passive rest or a mixture of active and passive recovery was undertaken. Mean power output (MPO) over 9 s (MPO_9), MPO over 18 s (MPO_{18}), and peak power output (PPO) were recorded. Lactate concentration, ratings of sprint preparation, ratings of sprint performance, and perceptions of recovery were also measured. Post-trial and post-study questionnaires explored factors that may have influenced performance. A sprint number × recovery method interaction ($F_{(1,23)} = 28.791$, $p < 0.001$, $\eta_p^2 = 0.556$) was found on PPO, with a significantly lower PPO in sprint 2 following passive recovery. Sprint number × second sprint duration interaction effects were also found on both PPO ($F_{(1,23)} = 9.867$, $p = 0.005$, $\eta_p^2 = 0.300$) and MPO_9 ($F_{(1,23)} = 8.922$, $p = 0.007$, $\eta_p^2 = 0.279$), although post hoc tests were unable to identify the cause of either effect. Similarly, a significant time × condition interaction was found on lactate concentration ($F_{(6.082,97.320)} = 2.982$, $p = 0.010$, $\eta_p^2 = 0.157$), with post hoc comparisons not revealing any differences between conditions at the individual time-points. The participants were typically satisfied with their sprint performances and expressed positive views about the recovery activity undertaken. The main finding was, however, that PPO was lower following passive recovery, but the effects on MPO were not apparent.

5.2. INTRODUCTION

At the 2020 Tokyo Olympics, the sprint cycling disciplines on the track were the Team Sprint, the Keirin, and the Match Sprint. The Match Sprint consists of a qualifying time-trial (the Flying 200), followed by head-to-head races, whereby two riders will compete against each other in a bid to cross the finish line first. Whilst each race takes place over three laps of the track, the actual distance that the riders maximally sprint is not fixed (Dale et al., 2022 – Chapter 4). Air resistance is a major limiting factor in cycling performance (Faria, 1992). Reductions in air resistance can be achieved by closely following another rider (Craig & Norton, 2001). Therefore, whilst the following rider will need to cover a greater distance to win the race, riders may attempt to strategically force their opponent to take the lead in this highly tactical event. The recovery time between races also varies. The average within session recovery period at the Tokyo Olympics was 48 ± 23 minutes, and in the final Gold Medal decider, the riders had just over 15 minutes to recover from their previous heat (Dale et al., 2022 – Chapter 4). At the 2016 Rio Olympics, the schedule indicated that a recovery period as short as 10 minutes may have occurred (Vieria, 2016). In laboratory-based tests, both MPO and PPO have been found to be reduced when 12 minutes separated two 18 s cycling sprints (Dale et al., 2022 – Chapter 4). Possible explanations for the loss of performance in the second sprint could be because the participants rested passively on the ergometer between sprints (Dale et al., 2022 – Chapter 4) or they could be as a result of a change in the effort provided.

Passive rest may not be reflective of the current practice that is undertaken by sprint cyclists (Dale et al., 2022 – Chapter 4), which may include a mixture of active and passive recovery (personal communication, 12th September, 2019). The rationale for performing an active recovery could be to increase blood flow to the muscle, enhancing O_2 delivery thereby aiding PCr resynthesis, as well as improving the removal of lactate and H^+ (Bogdanis et al., 1996a). The production of lactate and H^+ will occur during intense exercise and whilst the implications for muscular fatigue continue to be debated (Fitts, 2016; Westerblad, 2016), the inter- and intra-cellular movement of lactate is driven by concentration gradients, pH gradients, or by redox state (Brooks, 2018). The majority of lactate metabolism is directed towards oxidation (Brooks, 2018). Cardiac muscle is highly oxidative making it a major lactate consumer (Gladden, 2008). However, skeletal muscles will also oxidise lactate, with the oxidation rate being dependent on the metabolic rate of both the exercising and resting muscles (Gladden, 2008). Whilst not all findings have been in support (Bogdanis et al., 1996a; Dorado et al., 2004), the majority of research has indicated that performing an active recovery does improve lactate clearance (Devlin et al., 2014; Kirkpatrick & Burrus, 2020; McLellan & Skinner, 1982; Menzies et al., 2010).

From a sprint cycling performance perspective, when two 30 s WAnTs were performed 15 minutes apart, lactate clearance was enhanced when an active recovery was undertaken, but no interactions were found for any of the performance measures (absolute PPO, relative PPO, MPO, relative MPO, total work, and fatigue index) (Kirkpatrick & Burrus, 2020). The participants were, however, recreationally active women. Sprint performance recovery may be faster in women than men, albeit this may relate to the higher power outputs that are typically produced by men, rather than a difference in fatigue resistance between sexes (Billaut & Bishop, 2012). In the study by Kirkpatrick and Burrus (2020), the PPO achieved in the first sprint was on average ~ 730 W. In contrast, an average PPO of just under 1300 W was reported for recreational active men, during the first of two 30 s cycling sprints (Bogdanis et al., 1996a). When a second sprint was undertaken four minutes after the first, an active recovery resulted in a greater restoration of MPO (Bogdanis et al., 1996b). Furthermore, the improvement in performance recovery was as a result of the power generation during the first 10 s of the second sprint (Bogdanis et al., 1996a), where a disproportionate amount of the work is typically conducted (Bogdanis et al., 1996b).

PPO usually occurs a few seconds after a sprint is initiated (de Jong et al., 2015; Glaister et al., 2019; Wittekind et al., 2011), with power output declining thereafter. The optimal sprinting strategy has, therefore, been described as using an "all-out" approach (Abbiss & Laursen, 2008). There is, however, evidence to suggest that pacing does still exist during single sprints (de Jong et al., 2015; Glaister et al., 2019; Wittekind et al., 2011) and repeated-sprint activities (Billaut et al., 2011). During single sprints, a lower PPO has been found when longer sprints were undertaken (de Jong et al., 2015; Glaister et al., 2019; Wittekind et al., 2011) and during a repeated-sprint task, in comparison to the control trial, where the participants were correctly informed that they would be required to perform ten 6 s sprints with 24 s of recovery between sprints, power output was significantly higher during the first sprint, as well as the cumulated work over the first five sprints, in the deception trial, where the participants believed that they were only going to perform five sprints (Billaut et al., 2011). Therefore, the perception that the participants had about the demands of the task, affected the amount of effort that they were willing to exert. The maximum effort that an individual is prepared to provide to complete a task has been termed their potential motivation (Marcora, 2008).

The aim of the current study was, therefore, to investigate how the between sprint recovery activity that is currently undertaken by sprint cyclists (a mixture of active and passive recovery) affected performance recovery, as well as to evaluate whether a change in second sprint duration provided evidence of an alteration in pacing strategy. It was hypothesised that performing a mixture of active and passive recovery between sprints would enhance the recovery of both MPO and PPO. It was also hypothesised that performance recovery would be greater when a shorter duration second sprint was undertaken.

5.3. MATERIALS & METHODS

5.3.1. Participants

Twenty-four healthy strength-trained men (age: 26 ± 5 years; height: 180.3 ± 6.1 cm; body-mass: 82.3 ± 6.9 kg) (conducting at least two resistance training sessions per week for the last six months) volunteered to take part in this study. To aid with the consistency in testing conditions, the participants were instructed to refrain from conducting any strenuous exercise for 24 hours, ingesting alcohol and caffeine for 12 hours, and consuming food for three hours, prior to each trial. Before commencing the first trial, a pre-activity readiness questionnaire was completed, providing a screening process for any existing medical conditions and/or injuries that could have affected the ability of the participants to sprint on the cycle ergometer. Written informed consent was obtained and the participants were advised about the risks and benefits of taking part. Ethical approval was granted by St Mary's University Ethics Committee (London, United Kingdom) (see Appendix).

5.3.2. Design

5.3.2.1. *Experimental Approach*

All participants completed five trials. To reduce the possibility of an order effect, the experimental trials were performed in a randomised counterbalanced fashion. This was achieved by constructing a numerical list of every combination that the four experimental conditions could be performed. Each participant was then randomly assigned a unique number between one and twenty-four, which was matched to the corresponding trial order. For all sprints, the torque factor was set to 0.95 $Nm{\cdot}kg^{-1}$. The torque factor was greater than the standard WAnT load, as strength-trained individuals require a greater resistance to optimise sprint performance (Pazin et al., 2011). To incentivise maximal effort during each performance, a £100 gift voucher (Amazon, Seattle, USA) was rewarded to the participant that produced the highest MPO relative to body-mass over all sprints in the experimental trials.

5.3.2.2. *Trial 1*

Anthropometric measurements were first taken. The cycle ergometer was then adjusted for each participant, with the setup (seat and handlebar height, as well as fore-aft positions) being recorded to facilitate replication during the subsequent trials. Next, the participants performed a step test (starting load 40 – 100 W, step change 20 $W{\cdot}stage^{-1}$) on the cycle ergometer. Stages were three

minutes long, with 30 s of passive rest separating stages. During the passive rest period, a 20 µl capillary blood samples was taken from the earlobe. Samples were immediately analysed and the test was stopped when blood lactate concentration reached ~ 4 $mmol \cdot L^{-1}$. Software (Lactate E) for calculating blood lactate markers was used to identify LT1 via the log-log lactate threshold method, as well as to determine the corresponding power output (Newell et al., 2007). A brief rest period (~ 5 minutes) followed the step test. During the rest period, the participants were asked to view three VAS (see Appendix). One scale assessed preparedness to sprint (prior to each sprint), another perceptions of recovery (between sprints), and the final one assessed sprint performance (after each sprint). The participants were advised that there were no right or wrong responses to any of the scales and that they should place a mark that best represented their feeling at that time.

Following confirmation from the participants that they understood the requirements of each scale, they got back on the cycle ergometer, before being asked to indicate which leg they would prefer to use to initiate a stationary start sprint. Each sprint began with the chosen leg being ~ 45° forward to the vertical axis. The sprint warm-up protocol (see Table 5.01) was then explained and undertaken. The same sprint warm-up protocol was used in all the experimental trials. Three minutes after the sprint warm-up, the participants performed a 9 s familiarisation sprint and following an additional five minutes of rest (two minutes of active cycling, three minutes passively seated on the ergometer), an 18 s familiarisation sprint was performed. For all sprints, the participants were instructed to provide a maximal effort from the start, to remain seated throughout, and to keep sprinting until the resistive load was removed. Strong verbal encouragement was provided during each sprint. Positive statements were used to aid performance, e.g. “push, push, push” on sprint initiation, “you’re doing well” following the initiation, “dig-in” at approximately the half-way point, with a time remaining phrase only used towards the end of the sprint, “last 6 s, keep going” (Edwards et al., 2018). Practice measurements of the three VAS were also performed. At the end of the trial, the participants were advised about the gift voucher and the requirements to win it. Consistent with previous research, the reward was offered to encourage the participants to perform maximally (Marcora & Staiano, 2010).

Table 5.01 Sprint warm-up protocol.

Duration (s)	Resistive Load ($Nm \cdot kg^{-1}$)	Instruction
300	0	Passive rest
120	0.187	Comfortable cadence 60-90 rpm
5 (rolling start sprint)	0.47	Spin as fast as possible
50	0.187	Comfortable cadence 60-90 rpm
5 (rest)	0*	Get into start position
5 (stationary start sprint)	0.47	Drive as hard as possible
50	0.187	Comfortable cadence 60-90 rpm
5 (rest)	0*	Get into start position
5 (stationary start sprint)	0.47	Drive as hard as possible
55	0.187	Comfortable cadence 60-90 rpm
180	0	Passive rest

Note: * prior to each sprint a resistance of 1000 W was briefly applied to stop the flywheel from moving.

5.3.2.3. *Trials 2 – 5*

During each of the four experimental trials, the participants performed two cycling sprints, 12 minutes apart. Capillary blood lactate concentration was measured at rest, following the warm-up, immediately after the first sprint, as well as 3.5, 7.5, and 10.5 minutes after the first sprint. The VAS that evaluated how prepared the participants felt they were to perform an 18 s sprint was presented 30 s prior to each sprint, with the participants being asked to provide a clear mark on the scale with a pen. The scale that assessed how the participants rated their sprint performance was marked immediately after each sprint. The scale that assessed perceived recovery status was presented every 45 s for 10.5 minutes in the period between sprints. Consistent with previous research, a fresh scale was used for each measurement to prevent visual feedback from affecting the response (Glaister et al., 2012). If the participants perceived that their recovery status was complete within 10.5 minutes, the scale was no longer administered. The first sprint was always 18 s long. Following the sprint and the marking of the sprint performance scale, the participants were advised about the recovery condition (passive or active/passive mixed). During the active/passive mixed recovery trials, 30 s after the sprint, the participants cycled for 3.5 minutes at 80% of the LT1 power output (108 ± 35 W). A recovery exercise intensity that has been suggested to optimise blood lactate clearance (Devlin et al., 2014). The participants then rested passively on a chair for four minutes, before cycling again for a further 3.5 minutes at the same intensity. Following a final 30 s of passive rest, the participants performed a second sprint. The second sprint duration was either 9 s or 18 s. The participants were advised about the duration of the second sprint after the preparedness to sprint scale had been marked. In the passive recovery trials, the participants rested passively on the ergometer during the equivalent active cycling periods.

At the end of each session, a post-trial questionnaire was completed on a computer (see Appendix). The questionnaire asked how satisfied the participants were with their sprint performances

that day, what they felt may have mostly influenced their performances, and how they felt the recovery activity affected their second sprint performance. After the final trial, an additional questionnaire was completed (see Appendix). The end-of-study questionnaire asked the participants why they had chosen to participate, what factors they felt had mostly influenced their approach to the sprints, and the extent to which the offer of a financial reward had affected their approach to the sprints. A schematic of the experimental protocol is displayed in Figure 5.01.

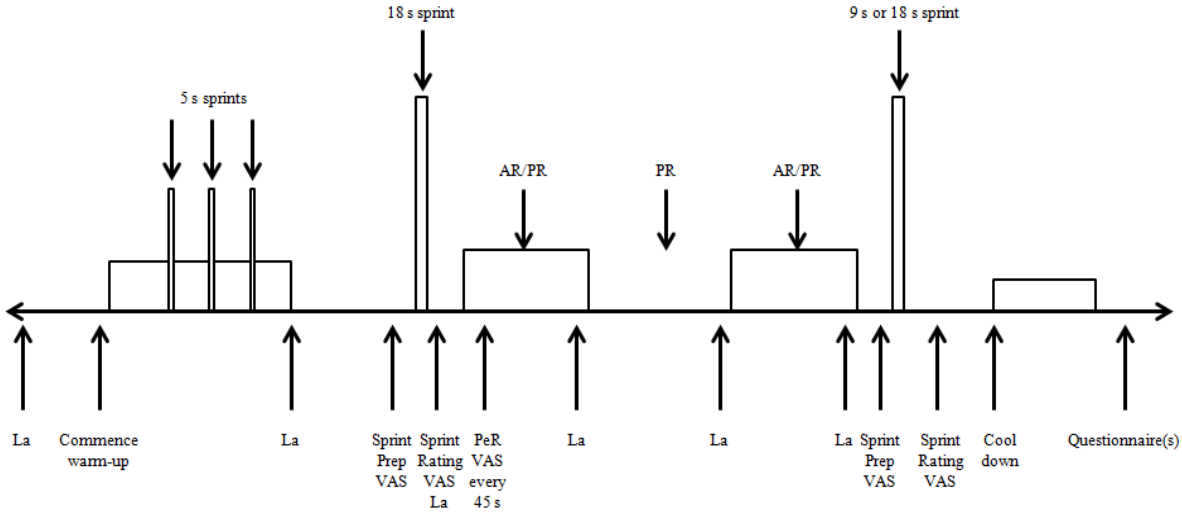

Figure 5.01 Schematic of the experimental protocol used. Note: AR denotes active recovery, La represents the time points when capillary blood samples were taken for the assessment of lactate concentration, PeR represents perceptions of recovery, PR is passive recovery, Sprint Prep VAS represents the time points when the sprint preparedness visual analogue scale was administered and Sprint Rating VAS represents the time points when the sprint rating visual analogue scale was administered.

5.3.3. Data Analysis

MPO was calculated over the first 9 s (MPO_9), and where appropriate, over the full 18 s (MPO_{18}) of the test. PPO was the highest value recorded. All VAS were 20 cm in length and markings were converted to a percentage before analysis. The time-course of perceived recovery status was modelled using a one-phase exponential growth function (see Equation 5.01). The mathematical function was selected based on previous research that has modelled physiological responses following a cycling sprint (Glaister et al., 2012). The questionnaire data were inductively organised into domain themes (Meijen et al., 2022).

$$PeR(t) = (A_0 \times \left(1 - e^{-\frac{(t)}{\tau_0}}\right) \times U_0) \quad \text{(Equation 5.01)}$$

where *PeR(t)* is the perception of recovery percentage at any time-point, A_0 represents the asymptotic amplitude for the exponential term, τ_0 is the time constants. $U_0 = 0$ when $t < 0$ and $U_0 = 1$ when $t \geq 0$.

5.3.4. Statistical Analysis

Statistical analyses were conducted using SPSS® software, version 28.0 (IBM Corporation, Armonk NY; USA). Values are reported as mean ± standard deviation. Statistical significance was set *a priori* at $p < 0.05$. PPO, MPO_9, as well as the preparedness and performance ratings were assessed using a three-way (sprint number × recovery method × second sprint duration) ANOVA. Due to violations of normality, data transformations were performed for the analysis of preparedness (taking the arcsine) and performance (cubing) ratings. Two-way ANOVAs were used to evaluate both differences in MPO_{18} (sprint number × recovery method) and lactate concentration (time × condition). Due to violations of normality, the time constant derived from the modelled response for perceptions of recovery was transformed by taking the square root. A one-way ANOVA was then used to assess for differences in τ between the four conditions. A one-way ANOVA was also used to assess for a performance training effect on PPO, MPO_9 and MPO_{18}. For the evaluation of a training effect, data from the first sprint were assessed in trial order. The coefficient of variation, the ICC, as well as 95% confidence limits, were also calculated using recommended procedures (Schabort et al., 1999). Where applicable, if the assumption of sphericity was not satisfied, the Greenhouse-Geisser correction was applied and where required, *post hoc* analyses were conducted using a Bonferroni correction.

5.4. RESULTS

5.4.1. Performance

5.4.1.1. *18 s Mean Power Output*

An effect of sprint number ($F_{(1,23)} = 39.088$, $p < 0.001$, $\eta_p^2 = 0.630$) and an effect of recovery method ($F_{(1,23)} = 10.229$, $p = 0.004$, $\eta_p^2 = 0.308$) were found on MPO_{18}, with a higher MPO_{18} being found during sprint 1 and when a mixed recovery was undertaken. The sprint number × recovery method interaction was not significant ($F_{(1,23)} = 2.513$, $p = 0.127$, $\eta_p^2 = 0.098$) (see Figure 5.02).

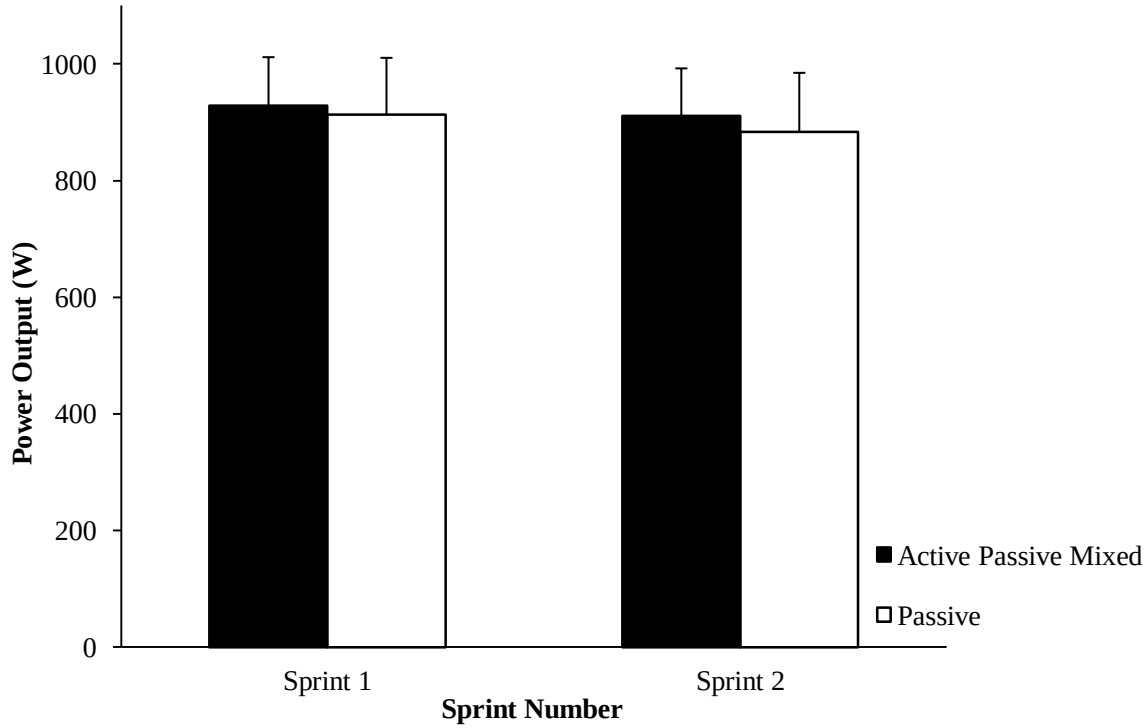

Figure 5.02 Mean power output during two 18 s sprint performed 12 minutes apart using either a mixture of active and passive recovery or just passive recovery between sprints. Bars represent the mean and error bars the standard deviation.

5.4.1.2. *9 s Mean Power Output*

An effect of sprint number ($F_{(1,23)}$ = 33.224, p < 0.001, η_p^2 = 0.591) and an effect of recovery method ($F_{(1,23)}$ = 22.883, p < 0.001, η_p^2 = 0.499) were found on MPO_9, with a higher MPO_9 being found during sprint 1 and when a mixed recovery was undertaken. A significant interaction was found for sprint number × second sprint duration ($F_{(1,23)}$ = 8.922, p = 0.007, η_p^2 = 0.279), although *post hoc* analyses did not reveal a significant difference for either sprint 1 or sprint 2 performances between the conditions (see Figure 5.03). The two-way interaction for sprint number × recovery method ($F_{(1,23)}$ = 3.343, p = 0.080, η_p^2 = 0.127) and the three-way interaction (sprint number × recovery method × second sprint duration) were not significant ($F_{(1,23)}$ = 0.326, p = 0.573, η_p^2 = 0.014).

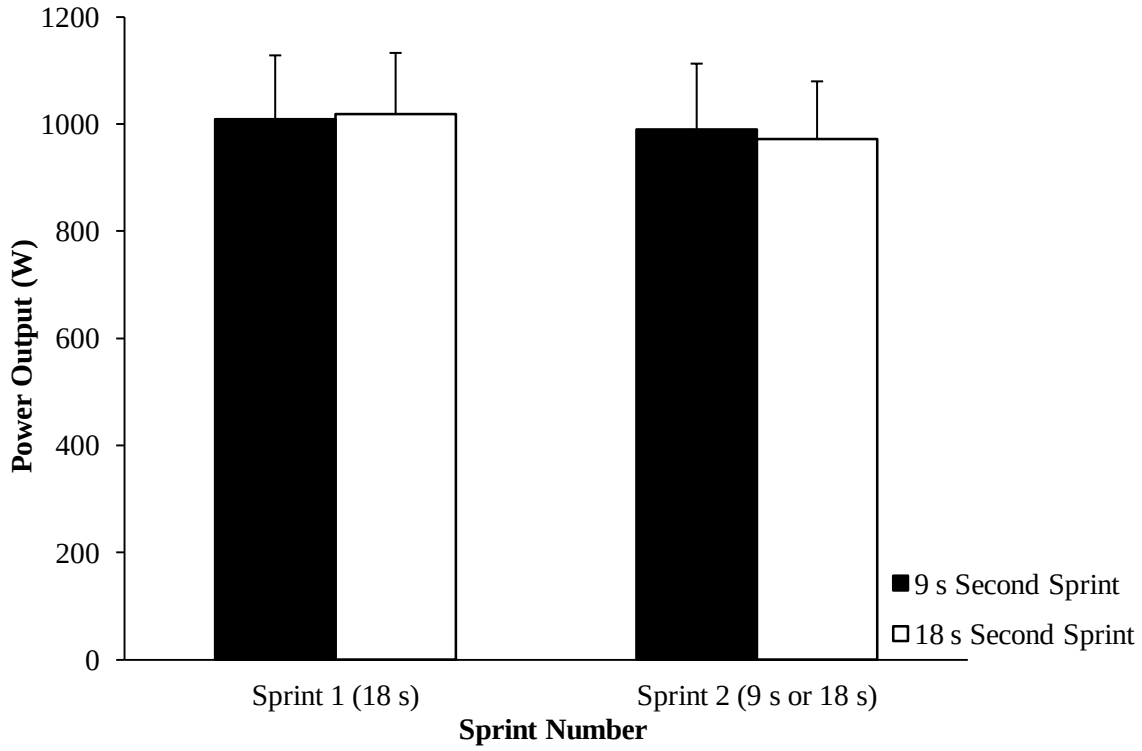

Figure 5.03 Mean power output during the first 9 s of two sprints performed 12 minutes apart. The first sprint was always an 18 s sprint. The second sprint was either 9 s or 18 s. Bars represent the mean and error bars the standard deviation.

5.4.1.3. *Peak Power Output*

An effect of sprint number ($F_{(1,23)}$ = 30.976, p < 0.001, η_p^2 = 0.574) and recovery method ($F_{(1,23)}$ = 19.660, p < 0.001, η_p^2 = 0.461) were found on PPO, with a higher PPO being found during sprint 1 and when a mixed recovery was undertaken. Two-way interactions were found for both sprint number × recovery method ($F_{(1,23)}$ = 28.791, p < 0.001, η_p^2 = 0.556) and sprint number × second sprint duration ($F_{(1,23)}$ = 9.867, p = 0.005, η_p^2 = 0.300) (see Figures 5.04 & 5.05). For the sprint number × recovery method interaction, *post hoc* analyses revealed no significant difference between recovery conditions for sprint 1 PPO, but when compared to a mixed recovery, sprint 2 PPO was significantly lower following passive recovery. For the sprint number × second sprint duration interaction, *post hoc* analyses revealed no significant differences between conditions for either sprint 1 or sprint 2. The three-way interaction (sprint number × recovery method × second sprint duration) on PPO was not significant ($F_{(1,23)}$ = 0.221, p = 0.643, η_p^2 = 0.010).

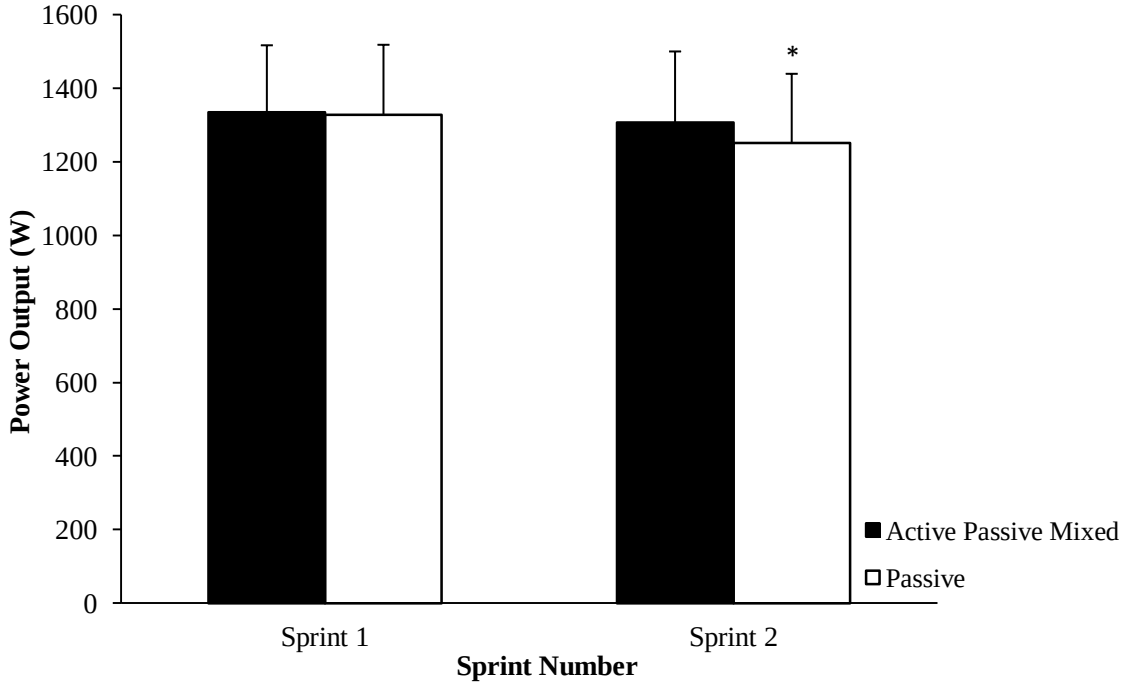

Figure 5.04 Peak power output during two sprints performed 12 minutes apart. The recovery method was either a mixture of active and passive recovery or just passive recovery. Bars represent the mean and error bars the standard deviation. Note: * denotes a significantly ($p < 0.05$) lower peak power output in sprint 2 following passive recovery when compared to a mixture of active and passive recovery.

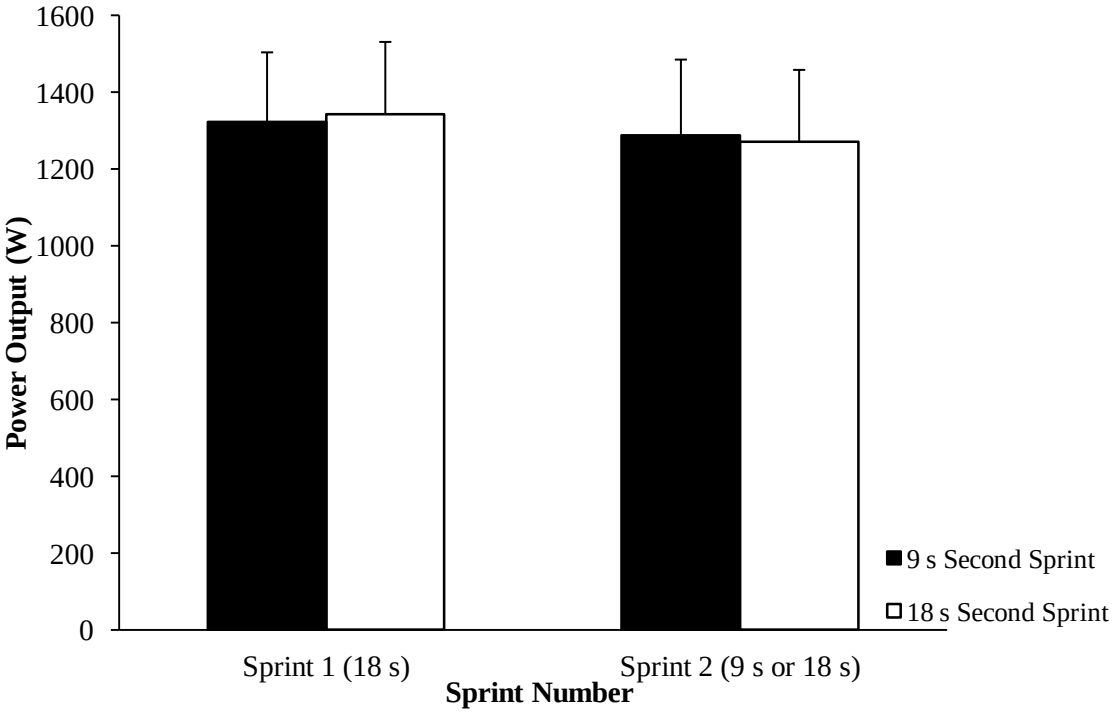

Figure 5.05 Peak power output during two sprints performed 12 minutes apart. The first sprint was always an 18 s sprint. The second sprint was either 9 s or 18 s. Bars represent the mean and error bars the standard deviation.

5.4.1.4. *Sprint Consistency*

When analysed in trial order, no significant differences were found in sprint 1 for MPO_{18} ($F_{(2.137,49.146)} = 0.598$, $p = 0.565$, $\eta_p^2 = 0.025$), MPO_9 ($F_{(3,69)} = 0.248$, $p = 0.862$, $\eta_p^2 = 0.011$), or PPO ($F_{(3,69)} = 0.059$, $p = 0.981$, $\eta_p^2 = 0.003$). The coefficient of variation and the ICC were 2.8% [2.4, 3.4] and 0.93 [0.87, 0.96] respectively for MPO_{18}, 4.1% [3.5, 4.9] and 0.89 [0.80, 0.94] respectively for MPO_9, and 4.7% [4.0, 5.6] and 0.90 [0.82, 0.95] respectively for PPO.

5.4.2. Blood Lactate Concentration

A significant effect of time ($F_{(1.680,26.872)} = 149.482$, $p < 0.001$, $\eta_p^2 = 0.903$) was found on blood lactate concentration. A significant time × condition ($F_{(6.082,97.320)} = 2.982$, $p = 0.010$, $\eta_p^2 = 0.157$) interaction was also found. However, *post hoc* tests did not reveal any significant differences between conditions at any of the six time-points (see Figure 5.06).

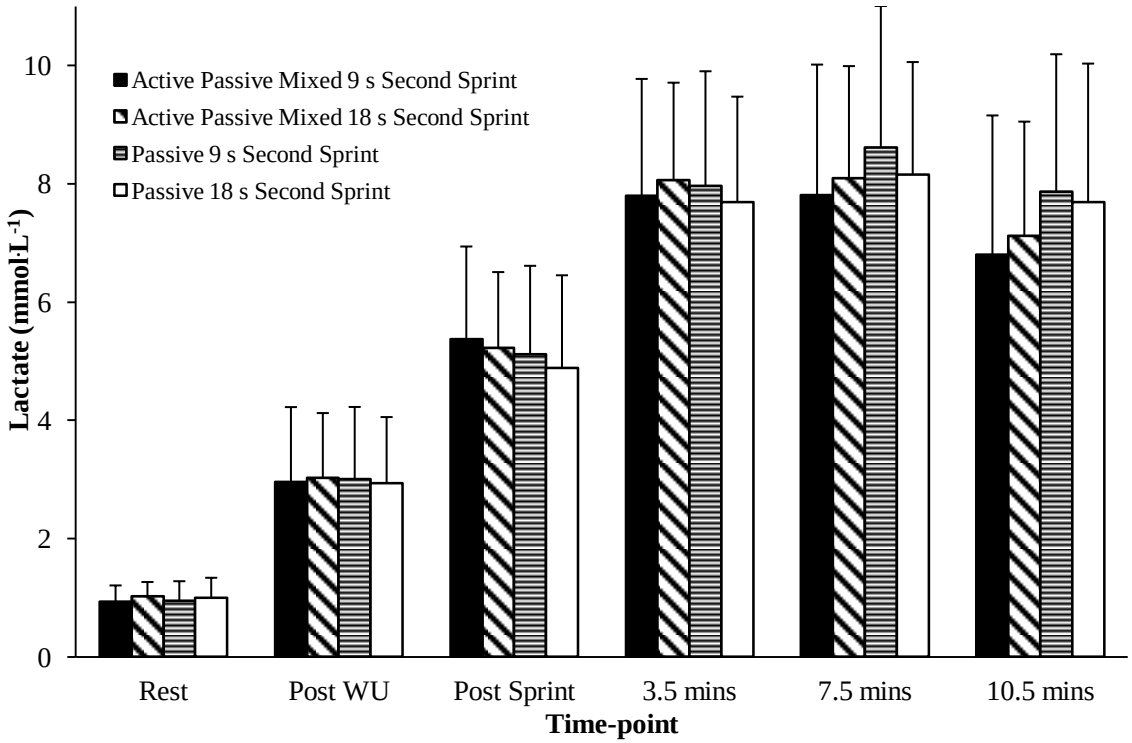

Figure 5.06 Blood lactate concentration at rest, after the warm-up (WU), immediately after the sprint, and following 3.5, 7.5, and 10.5 minutes of recovery, under four testing conditions. The recovery activity was either passive or a mixture of active and passive. The first sprint was always 18 s. The second sprint was either 9 s or 18 s. Bars represent the mean and error bars the standard deviation.

5.4.3. Preparedness to Sprint

An effect ($F_{(1,23)}$ = 6.927, p = 0.015, η_p^2 = 0.157) of sprint number was found on preparedness to sprint (see Figure 5.07), with the participants feeling better prepared for the second sprint. All other comparisons were not significant.

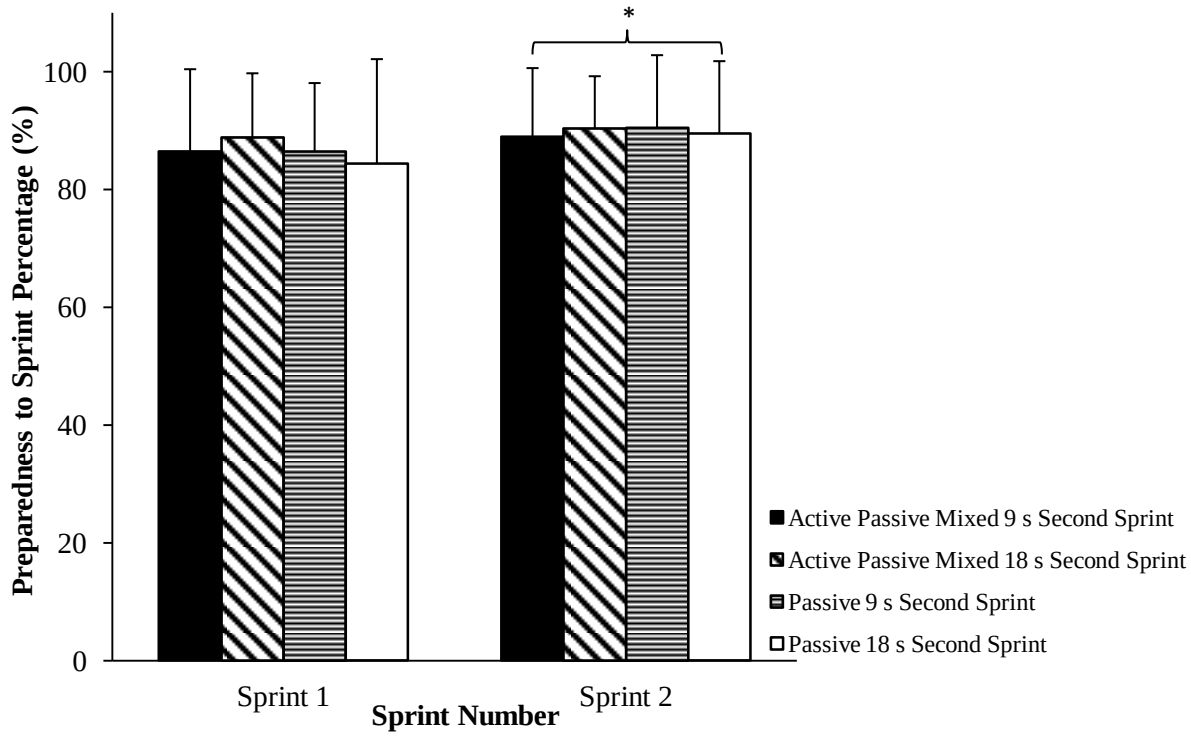

Figure 5.07 Rating of how prepared the participants were to sprint, measured just prior to two sprints performed 12 minutes apart. The recovery activity was either passive or a mixture of active and passive. The first sprint was always 18 s. The second sprint was either 9 s or 18 s. Bars represent the mean and error bars the standard deviation. Note: * denotes a significant effect (p < 0.05) of sprint number.

5.4.4. Perceptions of Recovery

Ratings of perceptions of recovery are displayed in Figure 5.08. The parameters of the modelled response are displayed in Table 5.02. The time constant did not differ significantly ($F_{(3,69)}$ = 0.689, p = 0.562, η_p^2 = 0.029) between conditions.

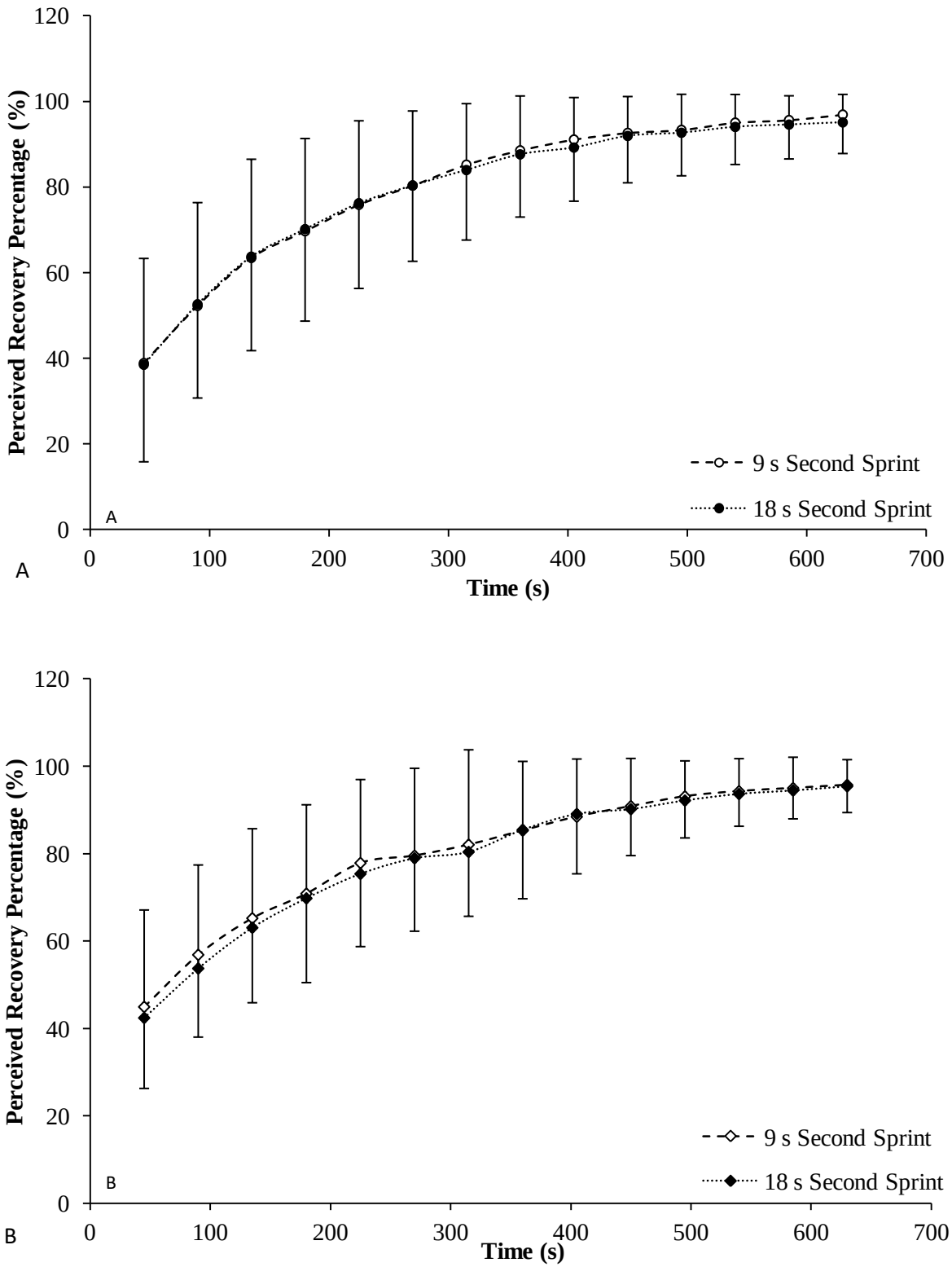

Figure 5.08 Perceptions of recovery following an 18 s sprint. The recovery activity was either passive (A) or a mixture of active and passive (B). The first sprint was always 18 s. The second sprint was either 9 s or 18 s. Points represent the mean and error bars the standard deviation.

Table 5.02 The time constant for perceptions of recovery following an 18 s sprint. The recovery activity was either passive or a mixture of active and passive. The first sprint was always 18 s. The second sprint was either 9 s or 18 s. Data are displayed as mean ± standard deviation.

Condition	Amplitude (%)	τ (s)
Active Passive Mixed 9 s Second Sprint	97.6 ± 5.3	127.1 ± 95.1
Active Passive Mixed 18 s Second Sprint	97.4 ± 5.9	158.9 ± 121.8
Passive 9 s Second Sprint	94.5 ± 7.4	140.5 ± 88.3
Passive 18 s Second Sprint	96.8 ± 5.2	140.4 ± 112.1

Note: τ denotes the time constant.

5.4.5. Sprint Performance Rating

The sprint performance rating differed significantly depending on the recovery method ($F_{(1,23)}$ = 4.300, p = 0.049, η_p^2 = 0.158) and the sprint number ($F_{(1,23)}$ = 6.313, p = 0.019, η_p^2 = 0.215), with higher ratings being found in the mixed recovery trials and following the second sprint. No other significant effects were found (see Figure 5.09).

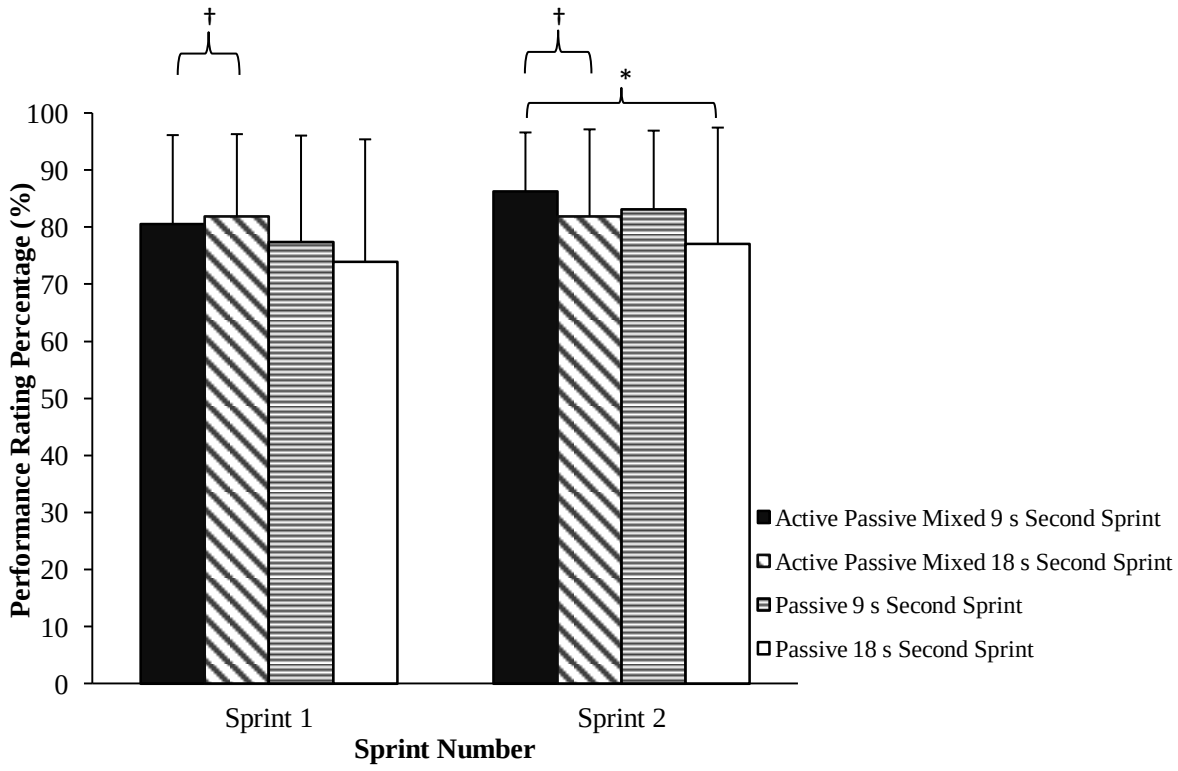

Figure 5.09 Performance rating following two sprints performed 12 minutes apart. The recovery activity was either passive or a mixture of active and passive. The first sprint was always 18 s. The second sprint was either 9 s or 18 s. Note: * denotes a significant effect (p < 0.05) of sprint number and † denotes a significant effect (p < 0.05) of recovery activity.

5.4.6. Sprint Satisfaction

The participants generally reported that they were fairly satisfied (e.g. *"I was fairly satisfied with my sprint performance. I feel that I had a better start to the sprint than previously, but struggled to hold it for the full length of time"*), satisfied (*"Good, both efforts felt strong, on the second shorter effort I felt very strong"*), or very satisfied (*"Very happy, maximal effort and kept the leg speed up as long as I could"*) with their sprint performances. However, on eight occasions, the participants stated that they were not satisfied (*"Didn't feel great. Felt better last week"*) and on one occasion not very satisfied *("Not very"*).

5.4.7. Factors that Influenced Sprint Performance

When asked about what may have mostly influenced sprint performance that day, the responses focussed on physiological factors, such as good sleep (*"I had a good night's sleep and rested most of the day"*), psychological factors, such as determination (*"My personal drive to want to perform the very best I can"*) and the information provided about the second sprint duration *("Feel the biggest influence on the 9 s sprint was being told it was 9 s just beforehand – immediately makes you feel less intimidated towards the 2nd sprint and perhaps allows you to go that extra 2 – 3% harder, knowing that there is less duration ahead")*, as well as physical factors, such as the recovery activity undertaken (*"The active recovery felt like it helped"*; *"The passive rest allowed me to fully recover after the 18 s sprint in preparation for the second sprint"*).

When specifically asked about the influence that the recovery activity may have had on second sprint performance, irrespective of the condition, the majority of participants focused on positive aspects (passive trials: "*I think it helped. I was pretty fatigued, so not doing anything seemed to be a good call"*; active/passive mixed trials: *"Moving my legs helped them to feel fresh and ready for the second sprint effort"*), although some participants stated that they would have preferred some form of active recovery to be included within the passive recovery trials *("Feel as though it may not have been the most efficient form of recovery. Ideally, would have preferred to have the legs spinning a little on the bike in-between"*), whereas when the mixed recovery protocol was undertaken, others stated that they would rather have rested passively (*"Massively influenced my perceived ability as I felt constrained by my breathing during recovery and this was compounded by my initial feeling of heavy legs before the second sprint"*).

5.4.8. Influence of a Financial Reward

When asked why they chose to participate in this study, participants mainly referenced aiding research ("*Contribute to research"*) or an interest in the project (*"Interested in the physiology of recovery and interested in finding out data on my own performance"*); no participants referred to the financial reward. In a similar fashion, no participants referred to the financial reward when asked about what provided the greatest influence on their approach to the sprints, with most of the responses referencing a personal challenge (*"I wanted to get the most out of myself for each effort, so my internal motivation to push myself influenced my sprint approach the most"*). When specifically asked the extent to which the offer of a financial reward affected their approach to the sprints, the majority of participants stated that the reward had little or no influence, although five participants suggested that the reward had some influence (*"The financial reward gave a slight extra motivation to push as hard as possible in the sprints, but not a significant difference as I wanted to push myself as hard as possible either way"*), and four participants suggested that the reward had a substantial influence (*"It helped me give everything in every attempt"*).

5.4 DISCUSSION

The aim of this study was to investigate the effects of recovery method, and an alteration in second sprint duration, on measures of sprint-cycling performance. The recovery of PPO altered depending on the between-sprint activity, with a significantly lower PPO being found in sprint 2 when a passive recovery was undertaken. Second sprint duration also affected the recovery of PPO, as well as MPO_9, although *post hoc* tests were unable to identify where those differences occurred. Blood lactate concentration differed between the conditions over time. However, *post hoc* analyses again did not reveal any significant differences between the conditions at the individual time-points. The participants felt better prepared for the second sprint than the first and rated their second sprint performance higher. Recovery method was also found to affect ratings of sprint performance, as well as MPO_9 and MPO_{18}, although interactions were not found with sprint number. Mixed views were expressed regarding the perceived benefits of the recovery activity undertaken, with the majority of participants focusing on positive elements, regardless of the condition. The time constant for perceptions of recovery did not differ between conditions and the offer of a financial reward did not appear to provide a major motivational factor for study participation or how the participants approached each sprint.

The outcome of an improvement in the restoration of PPO following a mixture of active and passive recovery, but with no significant difference in MPO over either 9 s or 18 s, differed to the finding that an active recovery did not affect PPO restoration, but improved the recovery of MPO over 6 s, 10 s, and 30 s, during a 30 s cycling sprint (Bogdanis et al., 1996a). Methodological differences included: the sample size; the duration of the sprints; the time separating sprints; the type of start to the sprint; the resistive load; the use of a harness to limit movement; and the intensity and format of the recovery activity. The sample size used in the current study was greater, which should improve statistical power. The type of start was consistent with previous sprint cycling research (Billaut & Basset, 2007) and was used to provide experimental control, although a rolling start would provide greater ecological validity when considering race conditions in the Match Sprint. The intensity and sequence of the recovery activity, as well as the time between sprints, were designed to reflect a scenario that could occur within a competition. However, when the time between two cycling sprints was very brief (15 s), a passive recovery was found to result in a significantly higher MPO and PPO than an active recovery (Dupont et al., 2007). It is, therefore, possible that the recovery time-course of MPO and PPO is affected differently by performing either an active or a passive recovery. Comparing active and passive recovery conditions at several time-points over a 12-minute period would help to clarify this suggestion.

With regards to lactate concentration, an active recovery has frequently been shown to enhance lactate clearance (Devlin et al., 2014; Kirkpatrick & Burrus, 2020; McLellan & Skinner, 1982; Menzies et al., 2010). A condition × time interaction was found for lactate concentration in the current study. The inability of *post hoc* tests to identify the location of the interaction could be because the 12-minute recovery duration was too short; especially considering that some of that recovery time was passive. The lack of an effect of an active recovery on post-exercise lactate concentration has previously been reported when the time between efforts was either four (Bogdanis et al., 1996a) or five minutes (Dorado et al., 2004). It is also possible that the rate of lactate clearance out of the muscle may have been enhanced, but that the rate of appearance in the blood increased in parallel with the rate of disappearance (Bogdanis et al., 1996a). From a participant perception perspective, in the post-trial questionnaire, some participants did indicate that the addition of active recovery was preferable to passive recovery, although others suggested the opposite. In fact, in general, the participants tended to focus on positive aspects of the recovery method used that day. Allowing the participants to freely select their recovery activity, would have added insight into individual preferences, as well as facilitating the ability to compare how individual preferences affect repeated-sprint performance.

A direct question about whether the participants perceived that the information about the duration of the second sprint affected their performance was not asked in the post-trial questionnaire. Nonetheless, when responding to what had mostly influenced their sprint performances that day, on

five occasions the participants stated that being informed that the second sprint was shorter, motivated them just prior to the sprint. A sprint duration × sprint number interaction was found on both PPO and MPO_9, although follow-up tests did not identify the cause of these effects. When asked about what had mostly influenced their approach to the sprints in the end-of-study questionnaire, the participants frequently cited an intrinsic motivational factor, namely, a personal challenge, whereas the offer of a financial reward appeared to have little or no influence on the approach taken to the sprints. Rewards have been shown to improve exercise performance during a hand-grip task (Shi et al., 2021), although cycling time-trials over shorter (1.5 km and 4 km) or longer distances (20 km) were not affected by a financial incentive (Hulleman et al., 2007; Skorski et al., 2017). The effectiveness of a particular type of motivational strategy should not, therefore, be assumed.

An additional consideration with the findings in the current study, relates to the effects that were found for sprint number and recovery method. An overall improvement in performance during the mixed recovery trials should not be interpreted to suggest that the between-sprint activity improved performance recovery. Only the existence of an interaction effect would suggest this outcome. Even though control measures were in place, it is possible that variations in first sprint performance could have contributed to the effect that was found. The overall significant reduction in sprint 2 performance for PPO, MPO_9, and MPO_{18} did, however, appear to contradict the perceptions reported by the participants. The participants rated their preparedness to sprint higher before the second sprint and then rated their second sprint performance as being better. The enhanced rating of how prepared the participants felt they were to sprint prior to the second sprint could be indicative of a lack of preparation for the first sprint. The warm-up protocol used was based on guidelines that have been provided for an 18 s sprint cycling test, but this may not have been optimal. Between-sprint measurements of perceptions of recovery could also have affected the markings taken before and after the second sprint. The time constant for perceptions of recovery did not differ between trials, with the mean values indicating that the participants perceived that they were fully recovered, or at least close to being fully recovered, within the recorded time-period. As the final measurement would have been taken close to when the second sprint scales were completed, it is possible that the recent markings could have affected the subsequent ratings.

In conclusion, when considering the shortest recovery periods that occur in the Match Sprint competition, there was evidence to suggest that the current practice undertaken by sprint cyclists could aid with the recovery of PPO, although the effects on overall performance were not apparent. Being informed that the second sprint duration was shorter provided a positive feeling for some participants and a change in performance recovery was found. However, for both PPO and MPO_9, the cause of the interaction was not established. Lactate concentration differed between conditions over time, although follow up tests did not identify any between condition differences at the individual time-points. It is possible that time off the bike may have limited lactate clearance. In contrast to the

performance findings, the participants perceived that they were better prepared to sprint prior to their second sprint and that they performed better during the second sprint. When using VAS, the possible influence of previous markings may need to be considered. Finally, a financial reward did not appear to influence the approach that the majority of participants took to the sprints. The effectiveness of motivational strategies should be evaluated, not assumed.

Chapter 6: The Effects of Sprint Duration on Repeated Sprint Performance – Pre-testing Data Collection

6.1. ABSTRACT

In Chapter 5, evidence was found for the existence of an effect of effort on sprint performance recovery. *Post hoc* tests were, however, unable to identify the cause of the interaction effects that were found. An increase in statistical power would mean that the probability of finding an effect would increase, assuming that an effect exists. The aim of this pre-data collection testing was, therefore, to evaluate whether an increase in sprint duration would change the performance loss effect size, whilst not impacting pacing strategy. Eight strength-trained men (25 ± 6 years; 180.4 ± 6.6 cm; 84.5 ± 8.4 kg) that had participated in the previous study visited the laboratory on a single occasion. During the visit, two 27 s sprints were undertaken 12 minutes apart, with a mixture of active and passive recovery performed between sprints. Peak power output (PPO) and mean power output (MPO) over 9 s and over 18 s were compared between trials for the first sprint. The performance loss effect size was then calculated using Hedges g by evaluating the reduction in MPO over the full sprints in each trial. MPO_{18}, MPO_{9}, and PPO during the first sprint did not differ between trials. However, when two 27 s sprints were undertaken 12 minutes apart, Hedges g was 0.20, compared to 0.13 when two 18 s sprints were performed. It was, therefore, suggested that an increase in sprint duration could increase the performance loss effect size, which could improve the probability of finding an effect in research that wishes to examine the impact of an effort-based intervention on repeated-sprint performance.

6.2. INTRODUCTION

In Chapters 4 and 5, sprint cycling performance was investigated with two sprints undertaken 12 minutes apart. In Chapter 4, the time-course of performance recovery was modelled and the relationship between performance recovery and several physiological processes was examined. In Chapter 5, the recovery activity either reflected current practice (a mixture of active and passive recovery) or was passive. The effect of an alteration in second sprint duration was also evaluated to assess whether the effort provided would change during the second sprint if a shorter sprint was undertaken. The first sprint in Chapter 5 was, therefore, always 18 s. However, the second sprint was either 9 s or 18 s. A sprint number × second sprint duration interaction effect was found on PPO and MPO_9, although on both occasions *post hoc* tests were unable to identify the cause of the effect. Statistical power is the probability of finding an effect, assuming that an effect exists, or the probability of not making a type 2 error (Norton & Strube, 2001). The four primary factors that affect statistical power are: the alpha level; the difference between the group means; the intra- and inter-variability in performance by the participants; and the sample size (Norton & Strube, 2001). In scientific research, the alpha level is typically set at 0.05. Whilst this is an arbitrary value and simply reflects the probability of a chance occurrence (a one in twenty chance that the null hypothesis is rejected, when it is in fact true) (Thomas et al., 2005), it is an accepted value. Increasing the sample size would increase statistical power. Research constraints, such as cost and time, do, however, need to be considered when designing a research project (Abt et al., 2020). Decreasing the error term would be beneficial, although factors such as time of day, prior intense exercise, and caffeine and alcohol consumption were controlled in Chapter 5 to minimise performance variation. Finally, increasing the size of the effect could improve statistical power.

Applying an intervention that would generate a larger effect would create the desired outcome. However, if the difference in performance between the two sprints was increased, it could also aid the possibility of finding an effect from an effort-based intervention. Reducing the recovery time between sprints or increasing the duration of the sprints may generate this outcome. The time between sprints in both Chapter 4 and Chapter 5 was selected as a recovery time that may occur in the Match Sprint competition. As the duration was a representation of the shortest recovery time that may be experienced, decreasing the time between sprints may not be the preferred solution. The duration of the sprint was also chosen considering the event, although this was based on the maximal effort that is typically provided during the Flying 200. During the main competition, the riders will race head-to-head over three laps of the track, with rider tactics dictating the exact duration of maximal effort. A longer duration sprint could generate greater levels of fatigue, which could increase the performance loss effect size, although increasing sprint duration could also generate a pacing effect

(de Jong et al., 2015; Glaister et al., 2019; Wittekind et al., 2011). The aim of this pre-testing data collection was, therefore, to evaluate the effect of sprint duration on repeated-sprint performance.

6.3. METHODS

Part A

6.3.1. Procedures

Video analysis from the 2020 Tokyo Olympics and the 2021 Track Cycling World Championship (which were held in Roubaix) was first undertaken. The primary researcher identified a series of races where the riders initiated an early sprint (see Figure 6.01 for an example of a rider launching an early sprint, taking his opponent by surprise). Both the primary researcher and a member of the supervisory team then independently viewed the footage and estimated the duration of maximal effort. Software (VideoPad Video Editor, NCH Software, Canberra, Australia) was used to provide an accurate frame-by-frame (40 frames·s^{-1}) estimation of the start and end of the sprint.

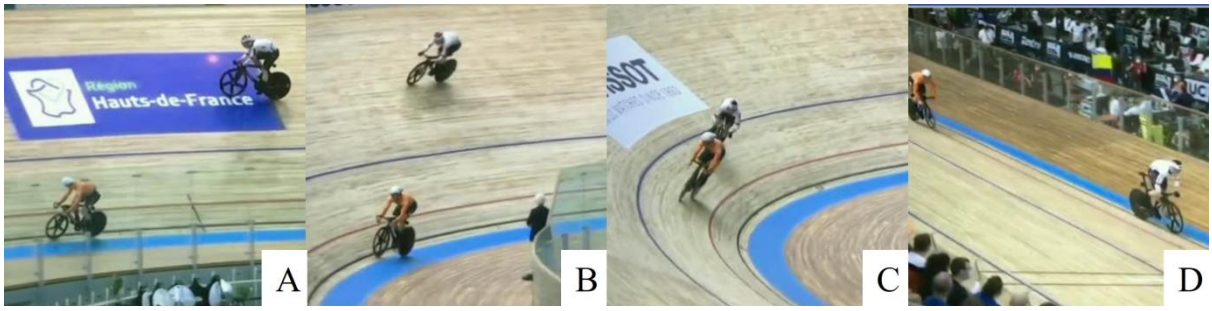

Figure 6.01 An example of a rider initiating an early sprint in an attempt to surprise his opponent. In position A, the Dutch rider (in orange) is watching the German rider (in white). In position B, the Dutch rider focuses on the track, at which point the German rider uses the banking to launch his effort (position C). In position D, the Dutch rider has reacted and must now sprint to get as close to the German rider as possible to minimise the air resistance that he must overcome.

Part B

6.3.2. Participants

Performance data were not considered in the recruitment and selection process. Instead, convenience sampling was used. Eight strength-trained men (25 ± 6 years; 180.4 ± 6.6 cm; 84.5 ± 8.4 kg) that participated in Study 2 (Chapter 5) returned to the laboratory. Informed consent was provided by the participants and ethical approval was granted by St Mary's University Ethics Committee (London, United Kingdom).

6.3.3. Procedures

The warm-up and the between-sprint recovery activity replicated the protocol that was used in the mixed active/passive recovery trials in Chapter 5. Sprints were also initiated from a stationary start using the same sprint initiation leg and the torque factor used for the sprints remained at 0.95 $Nm{\cdot}kg^{-1}$.

6.3.4. Analysis

MPO_9, MPO_{18}, and PPO were measured for the first sprint in each trial, with differences between trials being evaluated using a pared samples t-test and a statistical significance threshold of $p < 0.05$. MPO over the duration of each sprint was then calculated, with the performance loss effect size being determined for the two conditions using Hedges g (see Equations 6.01 – 6.03) (Goulet-Pelletier & Cousineau, 2018; Turner & Bernard, 2006). The size of the effect was then interpreted using guidelines (see Table 6.01).

$$d = \frac{M_1 - M_2}{SD_{pooled}} \quad \text{Equation 6.01}$$

$$SD_{pooled} = \sqrt{\frac{(n_1 - 1) \times SD_1{}^2 + (n_2 - 1) \times SD_2{}^2}{n_1 + n_2 - 2}} \quad \text{Equation 6.02}$$

$$Hedges\ g = d \times \left(1 - \frac{3}{4 \times (n_1 + n_2) - 9}\right) \quad \text{Equation 6.03}$$

where d represents Cohen's d, M_1 the mean of group or measurement 1, M_2 the mean of group or measurement 2, SD_1 and SD_2 the standard deviation of group/measurement 1 and 2 respectively.

Table 6.01 Interpretation guidelines for effect sizes calculated using Hedges g (Brydges, 2019).

Hedges g	Interpretation
0.15	Small
0.40	Medium
0.75	Large

6.4. RESULTS

Part A

6.4.1. Video Analysis

From the video analysis (Part A), it was estimated that the duration of maximal effort was in the range of 26 s – 30 s. Therefore, a 27 s sprint was selected as the sprint duration that was used in Part B.

Part B

6.4.2. Sprint 1 PPO, MPO_9, and MPO_{18}

PPO, MPO_9, and MPO_{18} for the first sprint in each trial are displayed in Table 6.02. There was not a significant difference in any of the performance metrics between trials (PPO: $t_{(7)} = 0.842$, $p = 0.428$, Hedges $g = 0.11$; MPO_9: $t_{(7)} = 1.275$, $p = 0.243$, Hedges $g = 0.13$; MPO_{18}: $t_{(7)} = 1.287$, $p = 0.239$, Hedges $g = 0.13$)

Table 6.02 Peak power output (PPO) and mean power output over 9 s (MPO_9) and over 18 s (MPO_{18}) during the first sprint performed on two occasions. The sprints were either 18 s or 27 s in duration. Data are displayed as mean ± standard deviation.

Sprint Duration	PPO (W)	MPO_9 (W)	MPO_{18} (W)
18 s sprint	1338 ± 84	1020 ± 122	911 ± 83
27 s sprint	1366 ± 238	1003 ± 128	899 ± 89

6.4.3. Sprint 1 to Sprint 2 MPO

The mean and standard deviation for the MPO for each sprint are displayed in Table 6.03. When two 18 s sprints were performed 12 minutes apart, Hedges g was 0.13 (small), whereas when two 27 s were undertaken 12 minutes apart Hedges g was 0.20 (small).

Table 6.03 Mean power output (MPO) during two sprints performed 12 minutes apart. Sprints were either 18 s or 27 s in duration. Data are displayed as mean ± standard deviation.

Sprint Duration	Sprint 1 MPO (W)	Sprint 2 MPO (W)	Percentage Change (%)
18 s sprints	911 ± 84	898 ± 92	1.4%
27 s sprints	813 ± 91	790 ± 112	2.8%

6.5. DISCUSSION

The aim of this pre-testing data collection was to evaluate the effect of sprint duration on repeated-sprint performance. MPO_{18}, MPO_9, and PPO did not differ between conditions, but the performance loss effect size was found to increase with sprint duration.

Sprint duration has previously been found to effect sprint cycling performance (de Jong et al., 2015; Glaister et al., 2019; Wittekind et al., 2011). With regards to PPO, Glaister et al. (2019) found that PPO was significantly higher during a 10 s sprint than during a 30 s sprint and de Jong et al. (2015) found that PPO was higher during a 10 s sprint, where the participants were solely required to produce the highest PPO possible, than during 250 m (21.4 ± 1.8 s), 500 m (43.7 ± 3.0 s), and 1000 m (91.9 ± 6.8 s) time-trials. Wittekind et al. (2011) provided greater depth in their analysis of the effect of sprint duration on performance. The participants completed 5, 15, 30, and 45 s sprints (Wittekind et al., 2011). Performance was evaluated via PPO and MPO for the duration of the sprint, but MPO was also evaluated over 5, 15, and 30 s (where applicable). PPO was found to be higher during the 5 s and the 15 s sprints than during the 45 s sprint (Wittekind et al., 2011). MPO over 5 s was also found to be higher in the 5 s sprint than during the 45 s sprint. Whilst MPO over 15 s was found to be higher during the 15 s sprint than during the 45 s sprint, PPO and MPO over 15 s did not differ between the 15 s sprint and the 30 s sprint. Therefore, the current findings of no change in PPO, MPO_9, and MPO_{18} between 18 s and 27 s sprints agreed with the findings by Wittekind et al. (2011), suggesting that changes in these performance metrics do not occur between 18 s and 27 s sprints.

It was also proposed that a greater performance loss effect size would occur between sprints when the sprint duration was increased from 18 s to 27 s. Hedges g was used to quantify the effect size as Cohen's d tends to overestimate the effect size when small sample sizes are used (Goulet-Pelletier & Cousineau, 2018). Hedges g was found to increase with sprint duration, although it should be noted that whilst the effect size was ~ 1.5 times larger when a longer sprint was undertaken, the size of the effect remained small. That being said, increasing the duration of the sprint from 18 s to 27 s could provide a meaningful way of increasing the performance loss effect size and thus could also aid with improving statistical power in research that aims to assess the effects of an effort-based intervention on sprint cycling performance.

Chapter 7: The Effects of a Simulated Cycling Competition on Repeated Sprint Performance and Physiological Stress Markers

7.1. ABSTRACT

The effect of simulated competition was examined on repeated-sprint cycling performance and on physiological stress markers. Sixteen resistance-trained men (age: 25 ± 4 years; height: 1.80 ± 0.07 m; body-mass: 83.3 ± 10.9 kg) participated. In the control and simulated competition conditions, two 27 s sprints were undertaken 12 minutes apart. Performance was evaluated as mean power output (MPO) during each 27 s sprint, with peak power output (PPO) also being measured. Recordings of the R-wave to R-wave interval duration were taken at rest, after the warm-up, and at the end of the trial for the measurement of heart rate variability (HRV). Saliva samples were taken at the same time-points for the assessment of alpha amylase (AA) activity and AA output. During the simulated competition, a crowd was present offering standardised verbal encouragement and the participants were competing for ranking status and financial rewards. MPO ($F_{(1,15)}$ = 12.419, p = 0.003, η_p^2 = 0.453), PPO ($F_{(1,15)}$ = 23.760, p < 0.001, η_p^2 = 0.613), AA activity ($F_{(1,10)}$ = 6.401, p = 0.030, η_p^2 = 0.390), AA output ($F_{(1,10)}$ = 5.342, p = 0.043, η_p^2 = 0.348), and normalised low frequency power ($F_{(1,14)}$ = 5.070, p = 0.041, η_p^2 = 0.266) were higher in the simulated competition, whereas high frequency power (Z = 2.229, p = 0.026, r = 0.332) and normalised high frequency power were lower ($F_{(1,14)}$ = 9.446, p = 0.008, η_p^2 = 0.386). Therefore, alongside an improvement in exercise performance, there was evidence of an increase in physiological stress in the simulated competition. Overall, the findings may be meaningful for research that requires sprint performances to provide a better representation of a true maximum, as well as being a useful training aid.

7.2. INTRODUCTION

Recommended pacing strategies for events that require efforts that are two minutes or greater, include “even” and “varied” (Abbiss & Laursen, 2008). In contrast, it has been suggested that an “all-out” approach should be adopted for sprint activities (efforts that are ≤ 60 s in duration) (Abbiss & Laursen, 2008). That being said, pacing, or at least a withholding of effort, has been found to exist during sprint and repeated-sprint activities (Billaut et al., 2011; Glaister et al., 2019; Wittekind et al., 2011), where the efforts were less than 60 s. Potential motivation reflects the maximum amount of effort than an individual is prepared to exert to satisfy a motive (Marcora, 2008). Therefore, as a withholding of effort has been found during sprint performances, it is conceivable that any factors that could increase potential motivation could also have a positive effect on sprint or repeated-sprint performances.

Simulated competition has been found to enhance cycling performance in the laboratory (Corbett et al., 2012; Williams et al., 2015); albeit, the distances examined by Corbett et al. (2012) (a 2 km time-trial) and Williams et al. (2015) (a 16.1 km time-trial) required efforts that were greater than two minutes (Wood et al., 2020). When the effect of competition was investigated over a shorter distance, a 1 km time-trial, which is a distance that represents a sprint track cycling discipline (the Kilo), Wood et al. (2020) found that pacing strategy, power output, and completion time were not affected by competition. Having said that, in all of the aforementioned competition studies (Corbett et al., 2012; Williams et al., 2015; Wood et al., 2020), an audience was not present, verbal encouragement was not provided, and rewards were not offered. The exclusion of these variables was to provide greater control of the laboratory conditions and to focus the research findings on head-to-head competition. However, in combination, these factors could enhance the motivational response, increasing the effort provided by the participants.

It is, of course, possible that competition could be detrimental to performance (Davies & Armstrong, 1989). Competition does create a considerable source of pressure for athletes (Díaz et al., 2012), although it may be the pressure of competition that generates the motivational response and the improvements in performance that have been observed. The stress that is perceived by the athlete determines the emotional response. From a physiological perspective, emotional information is processed by a small region in the temporal lobe, known as the amygdala (Thau et al., 2021). If required, a signal will be sent from the amygdala to the hypothalamus, resulting in activation of the sympathetic nervous system (Thau et al., 2021). If the stress is continued to be perceived, the hypothalamic-pituitary adreno-cortical axis will be activated (Thau et al., 2021). Whilst these stress response systems (sympathetic nervous system and the hypothalamic-pituitary adreno-cortical axis) work in a coordinated fashion (Kivlighan & Granger, 2006), the activity of the sympathetic nervous

system may provide a more sensitive measure of the stress experienced during a sports competition (De Pero et al., 2021).

The digestive enzyme, AA, provides a marker for the stress induced activity of the sympathetic nervous system (Rohleder et al., 2006). Greater levels of AA activity have been reported following high-intensity exercise (De Pero et al., 2021), as well as before (Caprancia et al., 2017; De Pero et al., 2021; Díaz et al., 2012), during (De Pero et al., 2021) and after a sports competition (Caprancia et al., 2012; Caprancia et al., 2017; Chiodo et al., 2011; De Pero et al., 2021; Díaz et al., 2012). HRV reflects the beat-to-beat fluctuation in heart rate and provides an additional measure of autonomic activity (Lipponen & Taravainen, 2019). Whilst many HRV indices can be derived from a recording, unfortunately none of them provide a specific assessment of the sympathetic response. Nonetheless, a change in autonomic activity, or evidence of parasympathetic withdrawal, has been found via changes in several HRV metrics following sprint exercise (Barak et al., 2014; Goulopolou et al., 2006; Millar et al., 2009; Niewiadauski et al., 2007) and prior to a sports competition (Cervantes et al., 2009; Mateo et al., 2012; Morgan & Mora, 2017; Murray & Raedeke, 2008; Souza et al., 2019). The aim of the current study was, therefore, to investigate the effects of simulated competition on repeated-sprint performance and on physiological stress markers. It was hypothesized that MPO, PPO, AA activity, and AA output would be higher during the simulated competition condition, whereas HRV would be lower.

7.3. MATERIALS & METHODS

7.3.1. Participants

Sixteen healthy men (age: 25 ± 4 years; height: 180.2 ± 6.5 cm; body-mass: 83.3 ± 10.9 kg; body-fat: 14.3 ± 4.4%) that were regularly (at least two sessions per week) undertaking strength, power, or sprint training, volunteered to participate. Participants from these training backgrounds were specifically recruited due to the importance of resistance training for sprint cyclists (Parsons, 2010) and because these individuals typically produce higher power outputs during cycling sprints (Pazin et al., 2011). Prior to commencing the first trial, the participants were informed about the risks and benefits of taking part and were required to complete an informed consent form, as well as a pre-activity readiness and lifestyle questionnaire (see Appendix). The study was granted approval by St Mary's University Ethics Committee (London, United Kingdom) (see Appendix).

7.3.2. Procedures

7.3.2.1. Experimental Approach

The participants were required to visit the laboratory on five occasions. During the first three trials, the participants were familiarised with sprinting on the cycle ergometer, the sprint torque factor and the active recovery intensity were determined, and baseline measurements were taken. The final two trials represented the control and simulated competition conditions. The order of the five trials was not randomised. The order of the final two trials, the control and experimental conditions, was fixed due to concerns about the potential negative effect of the motivational factors not being included in the subsequent trial, had the final two sessions been counterbalanced; an approach that was consistent with previous research (Hulleman et al., 2007). The participants were also not blinded to the aims of the study, as the intention was to assess physiological stress markers throughout the experimental and control trials, which included an assessment at the start of each trial to evaluate the anticipatory stress response. The primary performance measure was MPO during each 27 s cycling sprint. A 30 s WAnT is the most commonly performed laboratory sprint cycling test (Jaafar et al., 2014). However, the Match Sprint has its own specific requirements, whereby the duration of maximal effort is not fixed (Dale et al., 2022 – Chapter 4). Video analysis of the Match Sprint competition from the 2020 Tokyo Olympics and the 2021 Track Cycling World Championships was, therefore, undertaken to determine the duration of the performance test (Chapter 6), with the selected duration reflecting a sprint where the maximal effort was initiated early in a race.

7.3.2.2. Trial 1

The aim of Trial 1 was to determine an individualised sprint torque factor and to familiarise the participants with the sprint duration. On arrival at the laboratory, measurements of stature and body-mass were taken. The cycle ergometer was then adjusted to suit the participant, with the saddle and handlebar height, as well as the fore/aft positions being recorded, facilitating replication in the subsequent trials. A warm-up was undertaken (see Table 7.01) and five minutes after the warm-up, a series of 6 s sprints were performed every five minutes against an increasing torque factor (range 0.4 – 1.25 $Nm{\cdot}kg^{-1}$) to determine the load that produced the highest PPO (Glaister et al., 2019). All sprints were undertaken from a stationary start, with the preferred sprint initiation leg being ~ 45° forward to the vertical axis, which was consistent with previous research (Billaut & Basset, 2007). The participants were asked to provide a maximal effort from the start and to remain seated throughout. A five second countdown was provided and standardised verbal encouragement given for each sprint ("push, push, push, push….."). Ten sprints were always undertaken, although if PPO was continuing to increase on the tenth sprint, further sprints were performed using 0.05 $Nm{\cdot}kg^{-1}$

increments until PPO started to decrease (Glaister et al., 2019). On completion, and following a further five minutes rest, a 27 s familiarisation sprint was performed at the previously identified optimal torque factor (0.90 ± 0.16 $Nm \cdot kg^{-1}$). During the sprint, the participants were able to view power output in real-time on a monitor that was positioned in front of the ergometer. Standardised verbal encouragement was again provided throughout the sprint (e.g. "push, push, push, push, push" on sprint initiation, "you're doing well" following the initiation, "dig-in" at approximately the half-way point, with a time remaining phrase only used towards the end of the sprint, "last 10 s, keep going") (Edwards et al., 2018). Following the familiarisation sprint, a self-selected cool-down was performed.

Table 7.01 Warm-up protocol.

Duration (s)	Resistive Load ($Nm \cdot kg^{-1}$)	Instruction
240	0.187	Comfortable cadence 60-90 rpm
5 (rolling start sprint)	0.47	Spin as fast as possible
105	0.187	Comfortable cadence 60-90 rpm
10 (rest)	0*	Get into start position
5 (stationary start sprint)	0.47	Drive as hard as possible
110	0.187	Comfortable cadence 60-90 rpm
10 (rest)	0*	Get into start position
5 (stationary start sprint)	0.47	Drive as hard as possible
110	0.187	Comfortable cadence 60-90 rpm

Note: * prior to each sprint a resistance of 1000 W was briefly applied to stop the flywheel.

7.3.2.3. Trial 2

During Trial 2, the active recovery intensity, which would be used in the final two trials, was determined. On arrival at the laboratory, body-fat was first estimated using skinfold measurements. For the determination of the active recovery intensity, an incremental step test (starting load 40 – 180 W, step change 20 $W \cdot stage^{-1}$) was performed on the cycle ergometer. Stages were three minutes long, with 30 s passive rest taken between stages, during which time a 20 µl capillary blood sample was taken from the earlobe. The test was stopped when blood lactate concentration reached ~ 4 $mmol \cdot L^{-1}$. Software (Lactate-E) for calculating blood lactate markers was subsequently used to identify LT1 and the corresponding power output using the log-log lactate threshold method (Newell et al., 2007). Following a brief rest period (~ five minutes), a 27 s familiarisation sprint was performed; however, on this occasion, and in contrast to Trial 1, no verbal encouragement or performance feedback were provided. The removal of verbal encouragement and performance feedback was to familiarise the participants with sprinting under the conditions that would be

experienced in the baseline and control trials. After the sprint, a self-selected cool-down was performed.

7.3.2.4. Trial 3

In Trial 3, the participants were familiarised with the heart rate monitoring and saliva sampling procedures, before performing a single 27 s baseline sprint. Prior to the arrival of the participants, empty saliva collection vials were weighed. On arrival, the participants were fitted with a heart rate sensor (H10, Polar Electro Oy, Kempele, Finland) just beneath the sternum, before being asked to rinse their mouth with water and to sit down on a chair that was positioned beside the cycle ergometer. The heart rate sensor was then connected via Bluetooth to a heart rate monitor (V800, Polar Electro Oy, Kempele, Finland) and after five minutes of seated rest, R-R intervals were recorded for six minutes. Only five minutes of the recorded data were included in the analysis, which is the typical duration used to obtain valid short-term HRV measurements (Shaffer & Ginsberg, 2017). To ensure accuracy in the HRV recording, the participants remained seated throughout the measurement period and were asked to stay relaxed. Lying, seated, and standing positions have been used during HRV assessments, but a seated position may possess the greatest ecological validity when considering the movements of athletes prior to a sporting event (Mateo et al., 2012). In fact, in the Match Sprint, the riders will typically sit on chairs that are beside the track just before the race.

Following the R-R recording, guidance was provided about the saliva sampling procedure. For each saliva sample, a new collection aid was attached to a 2 mL cryovial (Salimetrics, Pennsylvania, USA). The participants were then asked to swallow any saliva that was in their mouth and to sit with their head tilted forward allowing/aiding any saliva that gathered in their mouth to flow into the collection aid (passive drooling) (Salimetrics, 2021). When collecting saliva during a sports competition in this manner, either a fixed duration (e.g. 2 minutes) (Díaz et al., 2012) or a target volume (range 1 mL – 5 mL) (Dehghan et al., 2019; Kivlighan & Granger, 2006) have been used. To ensure consistency in the timings between trials, and to make certain that the collection vials would not be overfilled, passive drooling was performed either until it was estimated that 1 mL of saliva had transferred to the vial (with the elapsed time being recorded) or if four minutes passed and it was not believed that 1 mL of saliva had been produced, the participants were asked to facilitate the movement of any remaining saliva into the vial and to stop the process. Saliva samples were then refrigerated at 2°C. Immediately after the trial, the samples were weighed. The mass of the sample was used to estimate the volume (the density of saliva was assumed to be 1 $g \cdot mL^{-1}$ (Rohleder et al., 2006)), which in combination with the duration of sample collection, facilitated the determination of saliva flow-rate ($mL \cdot min^{-1}$). The samples were then evenly divided into two vials, with one vial kept in reserve in case of any complications during the analysis. All vials were then placed into the freezer,

which was set at -80°C, until they were analysed for AA activity and output. The participants were then permitted to have a drink of water before getting onto the cycle ergometer and performing the standardised warm-up from Trial 1. Following the warm-up, the participants rested on the chair beside the ergometer for 10.5 minutes before getting back on the ergometer and performing a single 27 s baseline sprint. No encouragement or feedback were provided during the sprint and 90 s were allowed for the participants to mount the cycle ergometer and to prepare for the sprint, meaning that the total time between the warm-up and the sprint was 12 minutes. After the sprint, the participants cooled down with no resistance applied to the flywheel. When the participants had left the laboratory, a tracing of their power output profile from the sprint was made on an acetate sheet. The tracing would later (in the competition trial) be attached to the monitor that was in front of the cycle ergometer, overlaying their real-time power output. The reason for overlaying the tracing was to provide visual feedback for the participants, who would be asked to attempt to better their baseline performance.

7.3.2.5. Trial 4

Trial 4 represented the control trial. A schematic of the experimental protocol for the control and simulated competition trials is displayed in Figure 7.01. A resting R-R recording and saliva sample were first taken as previously described. Following the warm-up, another saliva sample and another R-R recording were made in the 12-minute break between the warm-up and the first of the two 27 s sprints. The saliva sample was started 30 s after the warm-up, with four minutes being allocated for sample collection. The participants then remained seated whilst an R-R recording was made for six minutes (started 4.5 minutes after the warm-up, irrespective of the duration of the saliva sample). When the recording was complete, the participants remounted the cycle ergometer and prepared themselves to perform a 27 s sprint with no encouragement or feedback. Twelve minutes of recovery were provided before a second 27 s sprint was undertaken under the same conditions. In the recovery period, no resistance was applied to the flywheel for one minute after the first 27 s sprint, but the participants were asked to continue pedalling. Following on, the participants were then required to cycle for three minutes at 80% of the power output at LT1 (89 ± 31 W), before dismounting and sitting on the chair that was next to the ergometer for four minutes. The participants then remounted the ergometer for another period of cycling (three minutes at the same intensity – 80% of LT1 power output), which was followed by 60 s of passive rest. The duration and the format of the recovery activity were similar to the protocol used by Dale et al. (2023 – Chapter 5), which provided a representation of the current practice that is undertaken by elite sprint cyclists. After the second sprint, the participants cycled with no resistance on the flywheel for two minutes before dismounting for a final saliva sample (started 30 s after the participants were asked to dismount the ergometer) and R-R

recording (started 4.5 minutes after being asked to dismount the ergometer). The participants were then encouraged to remount the cycle ergometer and to perform a self-selected cool down.

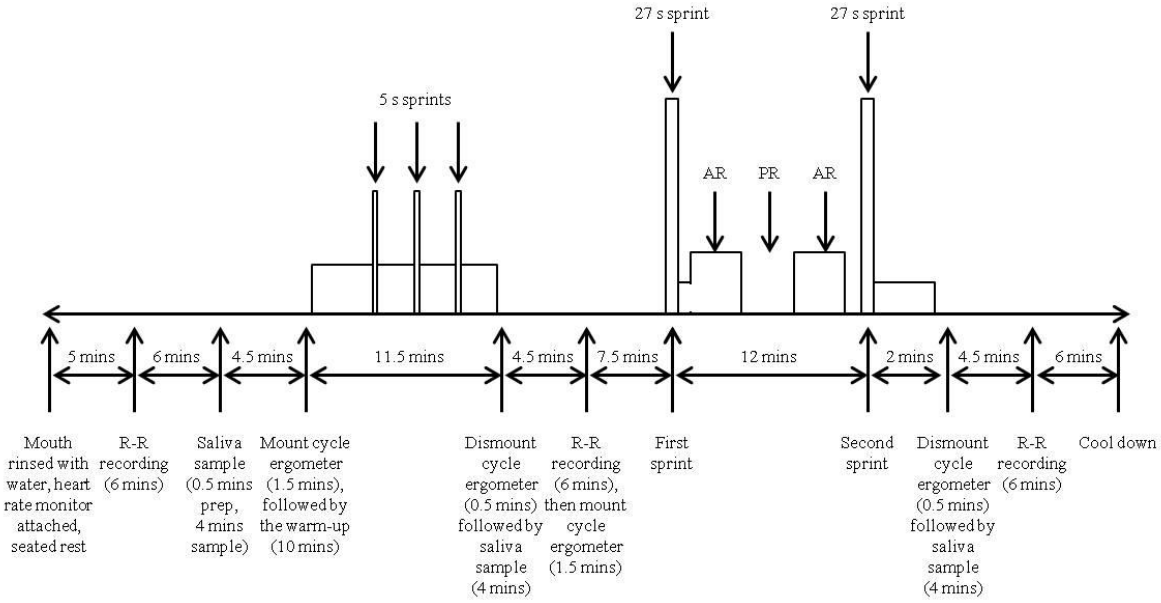

Figure 7.01 Schematic of the experimental protocol used during the control and simulated competition conditions. Note: AR denotes active recovery, PR passive recovery, R-R is the R-wave to R-wave recording, and mins is minutes.

7.3.2.6. Trial 5

The format of Trial 5 was largely the same as Trial 4: however, in this trial the participants were competing for two £100 reward vouchers (Amazon, Seattle, USA). One reward was offered for the absolute highest MPO over both sprints during the final trial. The other reward also considered MPO over both sprints in the final trial, but evaluated performance relative to the MPO that each participant had produced during their single sprint baseline performance from Trial 3. The rewards were chosen to encourage maximal performance and to ensure that all participants would have a reasonable chance of winning a reward. The specific details of the relative reward were only revealed at the start of the final trial. Real-time power output, as well as a tracing of their baseline performance could be viewed on the monitor that was positioned in front of the cycle ergometer. The participants were also advised that their performance (absolute MPO over both sprints) would be ranked and circulated to the other participants (prior consent was obtained, with each participant only being identified by their participant number). Finally, a crowd (three to five people) was present, offering the standardised verbal encouragement from Trial 1. At the end of the trial, a seven-point Likert scale (see Appendix) was completed, assessing the perceived impact that the five motivational factors (crowd presence, leaderboard, performance feedback, financial reward, and verbal encouragement)

had had on performance. The scale ranged from *a large negative effect* (-3) to *a large positive effect* (+3), with *no effect* (0) at the mid-point. The participants were also subsequently asked about the reasons for the markings provided. The responses were recorded and later transcribed. Finally, a self-selected cool down was performed.

7.3.3. Analytical Procedures

7.3.3.1. Alpha Amylase

From the salivary AA assay kit (Salimetrics, Pennsylvania, USA), the substrate was brought to room temperature before being placed in an incubator (INCU-Line IL23, VWR International Ltd, Lutterworth, United Kingdom) and warmed to 37°C. The frozen saliva samples were also thawed before being vortexed and transferred to polypropylene tubes. The tubes were then centrifuged at 1500 x g for 15 minutes. Each sample was mixed in a 1:200 ratio with the diluent as per the guidelines provided by the manufacturer (Salimetrics, 2019). From the diluent-sample mixture 8 µL was transferred to the well-plate. The substrate (320 µL/well) was then added to a strip of eight wells using a multi-channel pipette (VWR® Multi-Channel Pipette 50 – 1200 uL, Avantar, Lutterworth, United Kingdom). The well-plate was then immediately placed in a rotating incubator (PHMP-100, Grant, Cambridge, United Kingdom), which was set to rotate at 500 rpm at a temperature of 37°C. The well-plate was transferred to the plate reader (Biochrom ASYS Expert Plus, Harvard Bioscience, Holliston, USA) on two occasions. One reading was taken after 60 s and the other after 180 s. A 405 nm filter with no reference filter was selected on the plate reader. AA activity was quantified using Equation 7.01 (Salimetrics, 2019) and AA output was calculated using Equation 7.02.

$$AA\ activity\ (U{\cdot}mL^{-1}) = \Delta Abs \times 328 \qquad \text{Equation 7.01}$$

where AA denotes alpha amylase, ΔAbs denotes a change in absorption, and U denotes units

$$AA\ output\ (U{\cdot}min^{-1}) = \frac{Sample\ mass\ (g)}{Sample\ time\ (\text{min})} \times\ AA\ activity\ (U{\cdot}mL^{-1}) \qquad \text{Equation 7.02}$$

where AA denotes alpha amylase and U denotes units

7.3.3.2. Heart Rate Variability

To determine HRV, the R-R interval time series was first downloaded using software (Polar Flow, Polar Electro Oy, Kempele, Finland). Five minutes of data were taken from each six-minute recording (the first and last 30 s of each recording were excluded). An R-R interval was considered to

be erroneous if the duration differed by more than 75% from the previous interval and/or if the interval was outside of a defined acceptable range. The acceptable range was determined for each recording by calculating the duration that was 25% less than the first quartile and 25% greater than the third quartile, with all values inside this range being accepted (Plews et al., 2017). If a single R-R interval was identified as being erroneous, the number of corrected intervals that were to be inserted was calculated by dividing the duration of the erroneous interval by the average duration of the previous and subsequent intervals (Lippman et al., 1994). If multiple intervals (within ± five beats of each other) were identified as being erroneous, the total duration of the highlighted segment was calculated and divided by the average duration of the previous and subsequent intervals. Non-linear predictive interpolation was then used to correct the data. Non-linear predictive interpolation utilises R-R interval sequences that are artefact free and are locally similar to the artefact segment (Peltola, 2012). The similar R-R interval sequence then replaces the erroneous data (Peltola, 2012). The best matching comparison was achieved by selecting segments of four R-R intervals either side of the number of intervals that were to be replaced (Lippman et al., 1994). Following artefact identification and correction, the data were entered into the analytical software (HRVAnalysis, ANSLabTools, Saint-Etienne, France). Time-domain (mean heart rate, mean R-R duration, SDNN, and RMSSD), frequency-domain (LF power, HF power, LFnu, HFnu, and LF:HF ratio), and non-linear (SD1 and SD2) indices were computed. The data were detrended and resampled at 2 Hz for the frequency domain analysis (Mendonca et al., 2009) and the power spectrum was calculated using Welch's periodogram algorithm with a Hamming window of 256 points and an overlap of 50%, with the very-low, low, and high frequency bandwidths set at 0 – 0.04 Hz, 0.04 – 0.15 Hz, and 0.15 – 0.4 Hz, respectively (Pichot et al., 2016).

7.3.4. Statistical Analysis

Statistical analyses were conducted using SPSS® software, version 28.0 (IBM Corporation, Armonk NY; USA). Values are reported as mean ± standard deviation. Statistical significance was set *a priori* at $p < 0.05$. MPO and PPO were assessed using a 2 × 2 (condition × sprint number) ANOVA. A 2 × 3 (condition × time) ANOVA was used for the analysis of AA, saliva flow-rate, and the HRV variables. Due to violations of normality, data transformations were performed for AA activity, AA output, saliva flow-rate, SDNN, RMSSD, LF power, LFnu, HFnu, LF:HF, SD1, and SD2. If the assumption of sphericity was not satisfied, the Greenhouse-Geisser correction was applied. As violations of normality for HF power could not be rectified via transformation of the data, the effect of condition was analysed using a Wilcoxon signed rank test and the effect of time was assessed using a Friedman's ANOVA. A Friedman's ANOVA was also used to assess differences in the Likert scale ratings for the motivational elements. Where required, for all analyses, *post hoc* comparisons were

conducted using a Bonferroni correction. Finally, the responses to the questions regarding the Likert scale markings were grouped via the ratings provided.

7.4. RESULTS

7.4.1. Performance

There were significant effects of condition and sprint number on both MPO (condition: $F_{(1,15)}$ = 12.419, p = 0.003, η_p^2 = 0.453; sprint number: $F_{(1,15)}$ = 22.988, p < 0.001, η_p^2 = 0.605) and PPO (condition: $F_{(1,15)}$ = 23.760, p < 0.001, η_p^2 = 0.613; sprint number: $F_{(1,15)}$ = 16.314, p = 0.001, η_p^2 = 0.521), with higher power outputs being found in the first sprint and in the competition condition for both variables. The condition × sprint number interaction on MPO ($F_{(1,15)}$ = 2.755, p = 0.118, η_p^2 = 0.155) and on PPO ($F_{(1,15)}$ = 3.344, p = 0.087, η_p^2 = 0.182) was not significant (see Table 7.02).

Table 7.02 Peak and mean power output during two 27 s sprints performed 12 minutes apart, under control and simulated competition conditions. Data are displayed as mean ± standard deviation.

	Control		Competition		Effect
	Sprint 1	Sprint 2	Sprint 1	Sprint 2	
Mean Power Output (W)	727 ± 129	698 ± 126	798 ± 81	748 ± 92	a,b
Peak Power Output (W)	1207 ± 242	1147 ± 227	1332 ± 212	1217 ± 249	a,b

Note: a) denotes a significant (p < 0.05) effect of condition and b) denotes a significant effect of sprint number.

7.4.2. Alpha Amylase Activity and Saliva Flow-rate

Due to some complications with sampling (dry mouth, nausea, possible contamination to the sample), 11 complete data sets were included in the analysis of AA. There was a significant effect of condition on both AA activity ($F_{(1,10)}$ = 6.401, p = 0.030, η_p^2 = 0.390) and AA output ($F_{(1,10)}$ = 5.342, p = 0.043, η_p^2 = 0.348), with higher levels of both variables being found in the competition condition (see Table 7.03). There was also an effect of time on both variables (AA activity: $F_{(2,20)}$ = 4.776, p = 0.02, η_p^2 = 0.323; AA output: $F_{(2,20)}$ = 3.548, p = 0.048, η_p^2 = 0.262), with *post hoc* tests revealing a significant increase in AA activity post sprints when compared with rest and a significant decrease in AA output post sprints when compared with post warm-up. The interaction effect was not significant for either measure (AA activity: $F_{(2,20)}$ = 0.335, p = 0.719, η_p^2 = 0.032; AA output: $F_{(2,20)}$ = 0.302, p = 0.742, η_p^2 = 0.029) (see Table 7.03). A significant effect of time was also found on saliva flow-rate ($F_{(1.251,12.508)}$ = 16.670, p < 0.001, η_p^2 = 0.625), with *post hoc* tests revealing reductions from rest to the

post sprint time-point and from the post warm-up to the post sprint measurement. There was no effect of condition ($F_{(1,10)}$ = 0.146, p = 0.710, η_p^2 = 0.014) and no condition × time interaction ($F_{(2,20)}$ = 1.350, p = 0.282, η_p^2 = 0.119) on saliva flow-rate (see Table 7.03).

7.4.3. Heart Rate Variability

One data set was removed from the HRV analysis as the participant felt unwell after the second sprint in the competition trial, so was unable to complete the measurement. For the remaining participants, the time-domain, frequency-domain, and non-linear indices for HRV measured at rest, after the warm-up, and at the end of each trial are displayed in Table 7.04. There was a significant effect of time on all HRV metrics, whereas an effect of condition was found on mean heart rate (p < 0.001), mean R-R (p < 0.001), RMSSD (p = 0.005), LFnu (p = 0.041), HF power (p = 0.026), HFnu (p = 0.009), and SD1 (p = 0.006). Significant condition × time interaction effects were also found on mean heart rate (p = 0.002), mean R-R (p = 0.044), SDNN (p = 0.017), RMSSD (p < 0.001), LF power (p < 0.001), SD1 (p = 0.001), and SD2 (p = 0.006), with *post hoc* tests revealing a significant difference between conditions at the post warm-up time point for mean heart rate and mean R-R, and at the post sprint time-point for mean heart rate, mean R-R, SDNN, RMSSD, LF power, SD1, and SD2 (see Table 7.04).

Table 7.03 Alpha amylase activity, alpha amylase output, and saliva flow-rate assessed at rest, after the warm-up (post WU), and after two 27 s sprints, under control and simulated competition conditions (n = 11). Data are displayed as mean ± standard deviation.

	Control			Competition			Effect
	Rest	Post WU	Post Sprints	Rest	Post WU	Post Sprints	
AA activity ($U \cdot mL^{-1}$)	37.5 ± 33.7	45.0 ± 45.6	44.0 ± 32.9	42.1 ± 31.2	54.4 ± 48.7	85.7 ± 68.8	a,b,†
AA output ($U \cdot min^{-1}$)	22.2 ± 38.0	35.9 ± 59.0	19.7 ± 41.3	36.8 ± 66.0	48.3 ± 90.4	47.0 ± 105.1	a,b,‡
Saliva flow-rate ($mL \cdot min^{-1}$)	0.47 ± 0.29	0.61 ± 0.40	0.36 ± 0.44	0.64 ± 0.52	0.61 ± 0.52	0.37 ± 0.52	b,† ‡

Note: AA denotes alpha amylase, a) denotes a significant effect ($p < 0.05$) of condition and b) denotes a significant effect ($p < 0.05$) of time. † denotes a significant ($p < 0.05$) difference between the rest and post sprint time-points and ‡ denotes a significant ($p < 0.05$) difference between the post warm-up and post sprint time-points.

Table 7.04 Heart rate variability recorded at rest, after the warm-up (post WU), and after two 27 s sprints, under control and simulated competition conditions (n = 15). Data are displayed as mean ± standard deviation.

	Control			Competition			Effect
	Rest	Post WU	Post Sprints	Rest	Post WU	Post Sprints	
Time-domain							
Mean HR (bpm)	67 ± 11	79 ± 14	93 ± 19	72 ± 12	89 ± 16*	105 ± 18*	a,b,c
Mean R-R (ms)	926 ± 174	794 ± 172	678 ± 168	868 ± 168	707 ± 147*	591 ± 111*	a,b,c
SDNN (ms)	72.1 ± 27.6	54.3 ± 20.0	37.7 ± 28.4	83.6 ± 45.8	56.4 ± 32.8	26.4 ± 18.0*	b,c
RMSSD (ms)	57.2 ± 27.2	33.0 ± 21.4	20.3 ± 28.1	55.9 ± 41.8	28.0 ± 20.1	10.4 ± 12.2*	a,b,c
Frequency-domain							
LF Power (ms^2)	2265 ± 2683	956 ± 677	604 ± 982	3149 ± 4051	1178 ± 1150	401 ± 840*	b,c
LF nu (nu)	60.8 ± 16.0	71.7 ± 11.0	74.7 ± 17.2	69.6 ± 10.4	77.2 ± 13.0	79.6 ± 12.6	a,b
HF Power (ms^2)	1211 ± 981	346 ± 301	320 ± 955	1283 ± 1972	340 ± 389	77 ± 212	a,b
HFnu (nu)	37.9 ± 19.2	25.6 ± 10.5	22.2 ± 16.4	27.7 ± 9.8	19.4 ± 10.5	14.5 ± 9.1	a,b
LF:HF	2.4 ± 1.9	4.4 ± 3.8	5.2 ± 3.6	2.9 ± 1.2	5.2 ± 3.3	7.8 ± 6.1	b
Non-linear							
SD1 (ms)	41.0 ± 19.0	22.7 ± 14.9	14.4 ± 20.0	39.0 ± 26.5	20.0 ± 14.3	7.3 ± 8.7*	a,b,c
SD2 (ms)	91.9 ± 36.0	71.4 ± 25.7	50.9 ± 35.4	110.7 ± 57.4	76.9 ± 44.5	36.4 ± 24.1*	b,c

Note: HR denotes heart rate, R-R is R-wave to R-wave, SDNN the standard deviation of normal to normal beats, RMSSD is the root mean square of successive differences, LF is low frequency, HF high frequency, nu is normalised, SD1 is the Poincaré plot standard deviation perpendicular to the line of identity, and SD2 is the Poincaré plot standard deviation along the line of identity, a) denotes a significant effect of condition ($p < 0.05$), b) denotes a significant effect of time ($p < 0.05$), and c) denotes a significant condition × time interaction ($p < 0.05$). * denotes a significant difference ($p < 0.05$) between conditions at the specific time-point.

7.4.4. Motivation Ratings

The perceived influence of the five motivational elements experienced during the simulated competition trial did not differ significantly ($X^2_{(4)} = 6.897$, $p = 0.141$) (see Figure 7.02). Overall, three negative ratings were provided (3.75% of the ratings), two were for the crowd (*"The reason that I marked it as negative was because when I came into the lab and saw the people sitting there, I felt a bit nervous"*) and one was for the leaderboard (*"I just don't like it. I don't like competing"*). A no effect marking (zero rating) was made ten times (12.5% of the ratings). Three of these were for the offer of a reward (*"It's definitely not negative to a reward, but it's more of a bonus. I wasn't really thinking about that too much"),* five were for performance feedback *("I was just too busy focusing on my head down. I couldn't really see what my performance was"*), one was for the leaderboard, and one was for the crowd presence. The remaining ratings were all positive, although it was noted that it may have been difficult to differentiate between factors, such as crowd presence and verbal encouragement *("I think it might be accumulative, having people there had some accountability and then the fact that they were shouting probably drowned stuff out a little bit more, maybe? I mean towards the end it's less of an effect. I think at the start it's really explosive and you can drown out the pain quite early on, but without doing a control, I wouldn't really be able to separate"*).

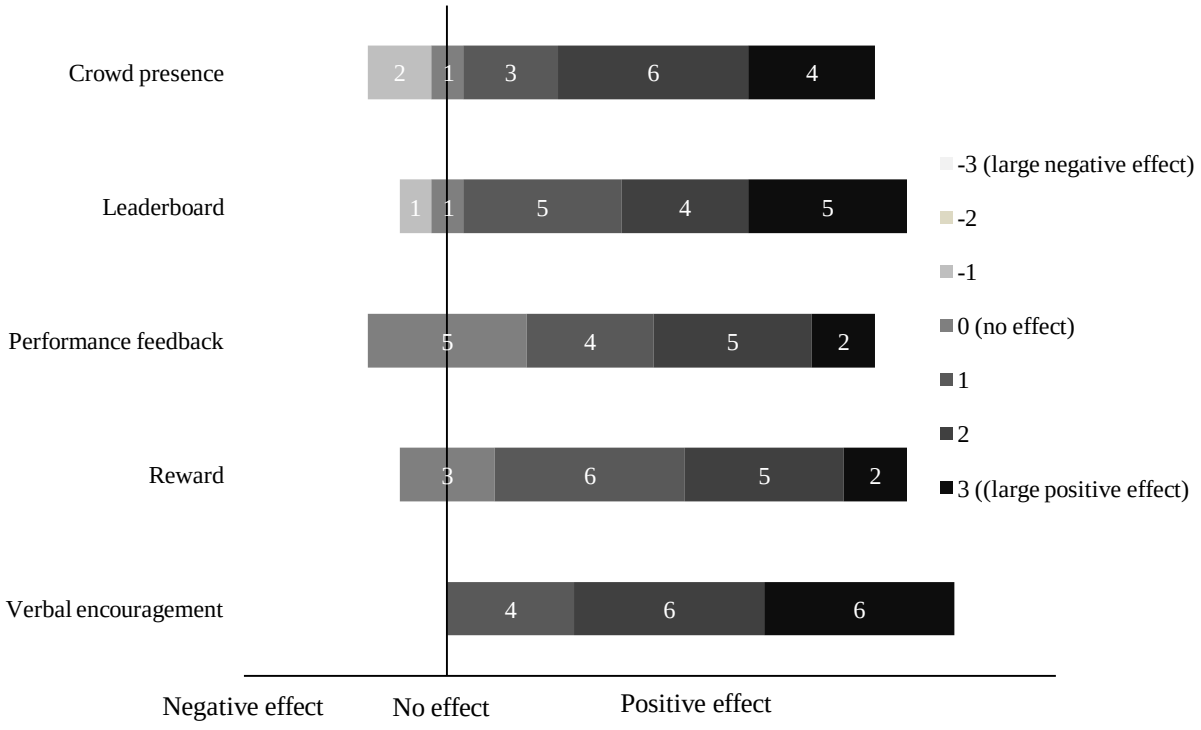

Figure 7.02 Ratings of the perceived influence of the motivational components on sprint performance. The numbers on the vertical line represents the number of ratings that were for no effect. Numbers to the left of the vertical line represent the number of ratings for each category that were for a negative effect and to the right of the line a positive effect.

7.5. DISCUSSION

The effect of simulated competition was examined on performance and on physiological stress markers during a repeated-sprint task. MPO and PPO were both greater in the simulated competition condition. There was also evidence of a change in physiological state via alterations in several HRV metrics, as well as in AA activity and AA output. There were not, however, any differences in the Likert scale ratings for the perceived influence of the five motivational elements that were included in the simulated competition.

The increase in MPO and PPO during the simulated competition condition suggests that the inclusion of the motivational elements had a positive effect on repeated-sprint performance. Improvements in exercise performance have previously been found during simulated cycling competitions (Corbett et al., 2012; Williams et al., 2015), although over the shortest time-trial distance that has been examined, a 1 km time-trial, head-to-head competition did not affect performance (Wood et al., 2020). Similarly, when a $100 reward was offered to the participants if they could improve their 1.5 km cycling time-trial performance by just 1 s, completion time and power output were not altered in the reward condition (Hulleman et al., 2007). It is, therefore, possible that head-to-head competition alone, or the offer of a financial reward in isolation, may not provide sufficient motivation to affect short to middle distance exercise performance. That being said, when verbal encouragement was provided on its own, MPO and PPO over two 30 s cycling sprints were greater when the sprints were performed with encouragement compared to when they were performed without encouragement (Edwards et al., 2018).

Of course, it cannot be established from the current findings whether verbal encouragement on its own would have produced the same effect on performance. The current study was intentionally designed to maximise the motivational stimulus during a single trial, with the perceived influence of the five motivational factors being assessed using Likert scale ratings. As no differences were identified in the Likert scale ratings, it suggests that there could have been a combined effect of the elements to generate the performance response that was found. The majority of the Likert scale ratings were, in fact, positive for all of the elements, with the only negative markings concerning the presence of an audience and the existence of a leaderboard. It is generally believed that the presence of an audience will have a positive effect on effort-based sports performance (Heinrich et al., 2021), although the effect of audience presence may be complex (van Meurs et al., 2022). For example, the skill-level of the participants (Zajonc, 1965) or the complexity of the task (van Meurs et al., 2022) could alter the response. The effect of verbal encouragement on performance may also be complex, with personality type being found to alter the outcome (Binboğa et al., 2013; Chitwood et al., 1997).

Further exploration into how the performance response that was found in the current study could be influenced by the characteristics of the individuals may, therefore, be beneficial.

Personality type was also not considered in the verbal encouragement study by Edwards et al. (2018). An additional limitation in that study, which was addressed in the current study, related to the way that the performance metrics were analysed. Before comparisons were made between the conditions, Edwards et al. (2018) averaged MPO and PPO over the two sprints that were undertaken in each condition. The highest PPO that was achieved, irrespective of sprint number, was also compared between conditions. In other words, it would not have been possible to determine whether the effect of verbal encouragement on performance differed between sprints. Large negative correlations have been found between performance during a first sprint and the recovery power output (power output in sprint 2 relative to power output in sprint 1) (Bogdanis et al., 1995; Bogdanis et al., 1996a; Bogdanis et al., 1996b). Thus, it would appear reasonable to suggest that if power output was enhanced during the first sprint, greater levels of fatigue would be elicited, which could then have a greater detrimental effect on subsequent performance. The current findings indicated that both MPO and PPO were lower in the second sprint, but that the response pattern did not differ between conditions. So, the performance findings could be interpreted either to mean that the motivational response remained the same across sprints or that the motivational response was actually greater during the second sprint, as performance was enhanced to the same relative degree as it was in the first sprint, despite the greater levels of fatigue that would have been generated in the first sprint. That being said, no physiological measurements were taken between sprints to support this suggestion, meaning that further investigation would be required to help understand and explain the outcome that was observed.

From a physiological perspective, in the current study it was proposed that the participants would be in a heightened state of arousal during the simulated competition. Changes were found between conditions for both AA activity and AA output, as well as several of the HRV metrics. De Pero et al. (2021) found that exercise on a training day increased AA activity, but that activity levels were even higher, before, during, and after exercise on the day of a competition. In the current study, both AA activity and AA output were higher in the simulated competition and as an interaction effect was not found for either variable, this suggests that there was an increase in sympathetic activity in the competition trial and that the change occurred throughout. In other words, the participants were in a heightened state of arousal from the start of the trial and then remained in this heightened state. The time course of AA activity and AA output was, however, found to differ, with AA activity being greater after exercise when compared to rest, whereas AA output was greater after the warm-up than after the sprints. It is currently recommended that saliva flow-rate should be accounted for in the analysis of AA (Strahler et al., 2017). The reason for this is because it has been suggested that it is the quantity of AA that is secreted per unit of time that relates to sympathetic activity, rather than the

activity levels that are found (Bosch et al., 2011). An additional sympathetic response is vasoconstriction of the arterioles that supply the salivary glands (Chicharro et al., 1998; Gatti & Palo, 2011). If saliva flow-rate is reduced, AA activity could increase as a result of a change in saliva flow-rate, as opposed to the quantity of AA that was generated. In the current study, saliva flow-rate was lower after the sprints when compared to both the resting and post warm-up measurements. Therefore, it is possible that performing the 27 s sprints did not increase AA production. Caution was also advised by Bosch et al. (2011) when using AA as an exclusive sympathetic marker, suggesting that this notion may be too simplistic. Nonetheless, the main finding regarding AA in the current study was that competition resulted in an increase in both AA activity and AA output, suggesting that the participants experienced greater stress in the simulated competition condition.

In contrast to the AA findings, all of the time domain and non-linear HRV indices were found to differ between conditions over time, as did the frequency domain metric LF power, with the majority of differences only occurring between conditions at the end-of-trial time-point. The pattern of this response does not provide evidence of a heightened state of arousal prior to the exercise task in the simulated competition. It is, therefore, possible that the environmental conditions did generate additional stress, but this only became apparent in these HRV metrics at the end of the trial. It is also possible that the alteration in exercise performance created the differences that were found. HRV in recovery has been found to be affected by the intensity of exercise performed (James et al., 2012; Parekh & Lee, 2005; Seiler et al., 2007), although when examining the effects of exercise intensity on HRV in recovery, the intensities being compared have been from different intensity domains. The intensity of exercise in the two trials in the current study would have been in the same intensity domain (severe). Furthermore, when contrasting higher exercise intensities (exercise at an intensity between the first and second ventilatory thresholds and exercise at an intensity greater than the second ventilatory threshold), differences in recovery HRV did not exist (Seiler et al., 2007). Therefore, it is most likely that the changes that were observed in these HRV metrics were also as a result of the environmental conditions, just not at the start of the trial. When compared to a high-stake competition, where the preparation could have been for many months or even years, a competition being simulated in a laboratory may not generate the same amount of stress in anticipation for the event. Differences in mean R-R, SDNN, and RMSSD were found in the studies by Cervantes et al. (2009) and Mateo et al. (2012) when the R-R recordings were made just before an actual sports competition and compared with the same time-point prior to a simulated competition on a training day.

In terms of interpreting the data for the individual HRV metrics, the time domain indices mean heart rate and mean R-R are effectively the same measure and whilst they are often reported in HRV studies, they do not actually provide an assessment of beat-to-beat variability. RMSSD, on the other hand, is the primary time domain HRV metric used to assess parasympathetic nervous system activity (Shaffer & Ginsberg, 2017). There is also a direct relationship between the non-linear metric

SD1 and RMSSD (Ciccone et al., 2017), meaning that the same response pattern for these variables could be expected. However, in addition to RMSSD and SD1, SDNN, LF power, and SD2 were all lower in the competition condition at the end-of-trial time-point. Sympathetic nervous system activity does contribute to the values that are computed for SDNN, LF power, and SD2, meaning that the lower values that were found for these variables in the simulated competition may, at first glance, appear surprising. However, others have reported lower SDNN (Mateo et al., 2012; Morales et al., 2012) and SD2 (Morales et al., 2012) values prior to a sports competition, displaying the complexity in the interpretation of these indices and meaning that the amount of parasympathetic withdrawal may have been be greater than the amount of sympathetic activation within the measured parameters.

In contrast to the interaction effects that were found for LF power, RMSSD, SD1, SDNN, and SD2, the normalised frequency domain indices, HFnu and LFnu, displayed a decrease in HFnu and an increase in LFnu in the competition trial, which agreed with the AA findings. HF power was also found to differ between conditions (lower in the simulated competition condition), but this may have been as a result of an inability to test for an interaction effect because of the non-parametric statistical procedures that were used. In fact, one of the benefits of normalising the data from spectral indices is that it improves their distribution, which tend to be skewed (Burr, 2007). A decrease in HFnu (Cervantes et al., 2009; Murray & Raedeke, 2008) and an increase in LFnu (Murray & Raedeke, 2008) have been reported prior to a sports competition. The HF power band is strongly associated with parasympathetic activity, but as the LF band is not a pure index of sympathetic activity (Heathers, 2014), the interpretation of the normalised spectral data is also complicated. Shaffer and Ginsberg (2017) suggested that as much as half of the LF power variability may be due to parasympathetic activity. A further challenge in comparing normalised data between studies relates to the normalisation procedure that was used. In the current study, normalisation was achieved by dividing the variable of interest by total power minus VLF power (i.e. LFnu = LF/(Total Power – VLF)). This procedure is in line with the recommendations of the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996), although others have suggested that this approach is not the most commonly employed method (Burr, 2007; Heathers, 2014). Either way, it can be stated that relative to total power minus VLF power, LF power was higher in the competition condition and HF power was lower, indicating a change in autonomic activity throughout the trial.

In summary, MPO and PPO were greater in the simulated competition condition, reflecting an enhancement in 27 s repeated-sprint performance. A finding that may be beneficial from both a training and a research perspective. There was also evidence of a change in physiological state, via alterations in a number of HRV metrics, as well as in AA activity and AA output. Aside from the limitations that have been highlighted above, overall, the findings suggested that the participants were in a heightened state of arousal during the competition trial, which could explain the changes in

performance that were observed. However, there were no differences in the perceived influence of the five motivational components, albeit further exploration into the effects of providing each motivational factor individually, as well as in combination, would now be beneficial.

Chapter 8: General Discussion, Limitations, Future Research, Practical Recommendations, and Conclusions

8.1. GENERAL DISCUSSION

The aim of this PhD thesis was to investigate repeated-sprint performance with consideration to the demands of the track cycling competition known as the Match Sprint. The main findings were: i) when sprints were repeated with a recovery period that may occur during a competition, power output was always lower during the second sprint; ii) the relationship between the performance recovery-rate and $\dot{V}O_{2max}$, the time constant for $\dot{V}O_{2off}$ kinetics, the muscle reoxygenation rate (MRT and τ), and the velocity constant for lactate clearance, ranged from negligible to moderate, but was never significant; iii) compared to when passive recovery was undertaken, PPO was higher in a second sprint when a mixture of active and passive recovery was performed between sprints, but the type of activity undertaken between sprints did not affect the recovery of MPO; iv) the existence of an interaction effect between sprint number and second sprint duration on both MPO and PPO provided evidence of a change in effort and even though a direct question about sprint duration was not asked in the post-trial questionnaire, when the duration of the second sprint was shorter, five participants reported a positive feeling towards the sprint; v) during a simulated competition both MPO and PPO were improved in a repeated-sprint task; vi) the motivational response of competition may, therefore, have been greater during the second sprint, given the greater level of performance/fatigue generated in the first sprint; vii) there was evidence of a change in physiological state during the simulated competition, via adjustments in AA activity and AA output, as well as in HRV; viii) differences in the Likert scale ratings for the perceived impact of the five motivational elements (cheering crowd, leaderboard, performance feedback, reward, and verbal encouragement) were not significant.

Overall, when evaluating short-term recovery, irrespective of the recovery method undertaken or the environmental condition, when a second sprint was performed 12 minutes after the first, a significant reduction in both MPO and PPO was found. The current practice of performing a mixture of active and passive recovery aided the recovery of PPO, but not MPO. The change in performance, as a result of the environment created in the laboratory when a competition was simulated, could provide a useful training tool, but also generated methodological considerations for research that requires maximal effort sprints to be performed.

8.1.1. Performance Recovery

MPO was the primary performance measure used in this thesis, as MPO provided a reflection of whole sprint performance. That being said, in order to achieve a high MPO, a high power output must be achieved as early as possible in the sprint. The importance of PPO for sprint cyclists

was, therefore, still acknowledged, with PPO being assessed as a secondary measure. The recovery time between sprints was varied in Chapter 4, from 45 s up to 12 minutes, but in the remaining studies it was always 12 minutes. The inclusion of multiple recovery times in Chapter 4 was to facilitate the mathematical modelling of performance recovery. The 12-minute recovery duration was chosen to reflect the shortest recovery time that may occur during the Match Sprint competition. In all studies (Chapters 4 – 7), irrespective of the recovery duration, when compared to the first sprint, MPO and PPO were reduced in the second sprint.

The short-term recovery of sprint cycling performance has previously been investigated using brief (1 s – 30 minutes) recovery time-periods (Ainsworth et al., 1993; Bogdanis et al., 1995; Bogdanis et al., 1996a; Bogdanis et al., 1996b; Cherry et al., 1998; Esbjörnsson-Liljedahl et al., 2002; Glaister et al., 2014; Hebestreit et al., 1993; Kirkpatrick & Burrus, 2020; Zabala et al., 2008; Zabala et al., 2011). When ten minutes or less has separated sprints, MPO (or total work) has been found to be reduced during the second sprint (Ainsworth et al., 1993; Bogdanis et al., 1995; Bogdanis et al., 1996a; Bogdanis et al., 1996b; Hebestreit et al., 1993). However, when either 15 minutes (Zabala et al., 2011) or 30 minutes (Zabala et al., 2008) separated sprints, differences in MPO did not exist. In Chapter 4, MPO was found to be reduced at all of the recovery time-points tested (45 s up to 12 minutes). Whilst it was possible that if a few more minutes of recovery had been provided, the complete restoration of MPO may have occurred, it was also notable that the MPO recovery percentage (sprint 2 MPO relative to sprint 1 MPO) found in Chapter 4 (97%) was not dissimilar to the findings reported by Zabala et al. (2008) (97% recovery) and Zabala et al. (2011) (98% recovery). The standard deviation reported for the sprints in the study by Zabala et al. (2011) was much greater than those found in Chapter 4 and the sample sizes were smaller (Zabala et al., 2008: n = 9, Zabala et al., 2011: n = 10). Greater variability in performance amongst participants is a key determinant of statistical power, due to the impact that this will have on the effect size, as is the sample size (Norton & Strube, 2001).

In Chapter 4, the model parameters (A_0: 97.4% ± 2.5%; τ_0: 130.6 ± 95.6 s) derived using a one-phase exponential function also indicated that the provision of a few extra minutes of recovery would not have led to the complete restoration of performance, as performance recovery had stabilised. The parameters of the two-phase exponential function (A_0: 87.7% ± 6.4%; τ_0: 56.3 ± 35.3 s; A_1: 11.9% ± 5.2%; and τ_1: 458.2 ± 283.3 s) did, however, indicate that the recovery of MPO would eventually be essentially complete, but a substantially greater recovery duration (> 30 minutes) would be required. The only other study to model sprint cycling performance recovery was by Glaister et al. (2014). The modelling procedures that were used in Chapter 4 were similar to those used Glaister et al. (2014), although their investigation was specifically interested in the recovery of PPO. Nonetheless, the authors reported that a two-phase exponential function provided a better fit to the

recovery data, although it was stated that the first-order amplitude (A_0: 45.7% ± 19.7%) was smaller than expected (Glaister et al., 2014). In Chapter 4, a two-phase exponential function improved the model fit on the majority of occasions (67%), which was in agreement with Glaister et al. (2014), but the first-order amplitude (A_0: 87.7% ± 6.4%) was much larger, suggesting that a substantial amount of recovery had occurred over the first ~ 3.75 minutes (4 × τ). Cherry et al. (1998), who also assessed the recovery time-course of PPO, had previously suggested that there was a two-phase response to sprint-cycling performance recovery. However, the authors suggested that the initial rapid phase was very short, just 6 s, and that it was related to ionic factors (Cherry et al., 1998). Mathematical modelling was not used by Cherry et al. (1998) to draw these conclusions; rather observations were made from the raw data. That being said, the suggestion that there could be an initial fast phase to performance recovery, followed by a slower secondary phase appears to be reasonable as physiological processes that are important for sprint cycling performance recovery, such as the resynthesis of PCr, occur in a biphasic fashion (Harris et al., 1976; McMahon & Jenkins, 2002; Walter et al., 1997).

With regards to PPO, PPO has typically been found to be lower when six minutes or less separated sprints (Bogdanis et al., 1995; Bogdanis et al., 1996a; Bogdanis et al., 1996b; Cherry et al., 1998; Hebestreit et al., 1993), although Ainsworth et al. (1993) did find that PPO was fully restored after just six minutes of recovery. The duration of the sprints (45 s) that were performed in the study by Ainsworth et al. (1993) was, however, longer than the duration of a WAnT or the durations used in Chapters 4 – 7. A 45 s sprint is also longer than the greatest duration of maximal effort that will likely occur during a Match Sprint race. Whilst a greater sprint duration would intuitively elicit greater levels of fatigue, which would then mean that a greater recovery time would be required for PPO to be restored (Glaister et al., 2014), pacing will exist during a 45 s sprint (Wittekind et al., 2011). Therefore, the PPO that was recorded in the sprints by Ainsworth et al. (1993) may not actually reflect a true maximal PPO. That being said, in agreement with Ainsworth et al. (1993), Hebestreit et al. (1993) found that PPO was fully restored during a 30 s WAnT after 10 minutes of recovery, which was also in contradiction to the findings in Chapters 4 – 7. The method that was used to measure PPO did, however, differ. Hebestreit et al. (1993) averaged the first 6 s of performance data to provide an assessment of PPO. A methodological approach that was appropriate at the time that the research was conducted, but the accuracy of modern-day ergometers facilitates greater precision in the assessment of the highest power output that is achieved during a sprint (Driss & Vandewalle, 2013; Lanferdini et al., 2020), with the Lode ergometer recording measurements at 5 Hz. Overall, therefore, the performance findings from this thesis suggest that athletes competing in the Match Sprint should consider means of enhancing the recovery process, when the recovery duration is brief.

8.1.2. The Relationship between Performance Recovery and Maximal Oxygen Uptake, Oxygen Uptake Off-kinetics, Muscle Reoxygenation, and Lactate Clearance

It is the belief of many sports scientists and coaches that individuals with a higher $\dot{V}O_{2max}$ will recover faster from high-intensity exercise (Aziz et al., 2000; Ulupinar et al., 2023). PCr resynthesis occurs via the rephosphorylation of creatine by aerobically produced ATP (McMahon & Jenkins, 2002). Individuals that possess faster $\dot{V}O_{2off}$ kinetics could, therefore, experience faster PCr resynthesis (Buchheit et al, 2012), resulting in an improvement in repeated-sprint performance. Individuals with a high $\dot{V}O_{2max}$ are also likely to possess faster $\dot{V}O_{2on}$ kinetics (Babcock et al., 1994; Berger et al., 2006; Cleuziou et al., 2005; Dale & Glaister, 2018; Dogra et al., 2013; Grey et al., 2015; Koppo et al., 2004; Norris & Petersen, 1998; Marwood et al., 2010). Faster $\dot{V}O_{2on}$ kinetics could aid repeated-sprint performance by adding to the energy contribution to each sprint. Finally, whilst the role of lactate/acidosis in fatigue has been well documented and debated (see section 2.2.2.6. and section 2.2.2.7.), the possibility still exists that individuals that are able to clear lactate faster could perform better during a repeated-sprint task. Therefore, in Chapter 4, the relationship between performance restoration and blood lactate clearance, muscle reoxygenation, pulmonary $\dot{V}O_{2off}$ kinetics, and $\dot{V}O_{2peak}$, was investigated. The strength of these relationships ranged from trivial (blood lactate clearance: r = -0.026) to moderate ($\dot{V}O_{2peak}$: ρ = -0.342), but was never significant.

Previous research that has investigated the relationship between lactate clearance, muscle reoxygenation, $\dot{V}O_{2max}$, $\dot{V}O_{2off}$ kinetics and repeated-sprint performance (Archiza et al., 2020; Aziz et al., 2000; Aziz et al., 2007; Bishop et al., 2003, Bishop & Goodman, 2004; Buchheit, 2012; Buchheit et al., 2011; Buchheit et al., 2012; de Aguiar et al., 2015; de Aguiar et al., 2016; Dupont et al., 2005; Dupont et al., 2010; Pajeja-Blanco et al., 2016; McMahon & Wenger, 1998; Ufland et al., 2013; Wadley & Le Rossignol, 1998) has typically used repeated-sprint protocols designed to reflect the demands of sports that require intermittent bursts of high-intensity exercise, interspersed with brief recovery periods. For example, Aziz et al. (2007) assessed the relationship between $\dot{V}O_{2max}$ and the performance of six 20 m sprints with 20 s of active recovery between sprints. The applicability of these findings to the Match Sprint is, therefore, questionable. By the same notion, it is then also difficult to make direct comparisons between the findings in Chapter 4 with previous research. The methods used to quantify both performance recovery and the recovery of the various physiological processes, has also differed. In Chapter 4, performance restoration and the recovery time course of the physiological variables were all quantified using mathematical modelling, with performance recovery being determined as MPO during the second sprint as a percentage of the MPO that was achieved during the first sprint. In other repeated-sprint research studies, the key performance outcomes usually include: the best sprint time/highest power output; the average sprint time, MPO, or total work; and some form of assessment of the ability to resist fatigue and maintain performance throughout the test. The metric that is used to assess fatigue resistance (see Glaister et al., 2008 for nine different formulas

that have been used) does provide the best comparison for the findings in Chapter 4. Findings regarding the strength of the relationship between $\dot{V}O_{2off}$ kinetics, $\dot{V}O_{2max}$, and the relevant measure of fatigue have varied considerably (see section 2.3.2.). However, in a recent meta-analysis, Ulupinar et al. (2023) reported a moderate correlation (r = -0.449) between $\dot{V}O_{2max}$ and the fatigue score. In Chapter 4, the strength of the relationship between the performance recovery rate and $\dot{V}O_{2off}$ kinetics was found to be small (ρ = 0.240) and between the performance recovery rate and $\dot{V}O_{2max}$ was found to be moderate (ρ = -0.342). Therefore, the strength of these relationships was smaller, but was not dissimilar to the findings of Ulupinar et al. (2023).

NIRS was also used in Chapter 4 to provide a local measure of muscle oxygenation at the *vastus lateralis*. Consistent with previous research, only TSI was included in the analysis (Buchheit et al., 2012). MRT was calculated as well as τ, as Nagasawa (2013) suggested that individuals with a higher $\dot{V}O_{2max}$ may experience a delay in muscle reoxygenation, meaning that MRT may provide a better reflection of the recovery time. That being said, the correlation that was found between performance recovery and both TSIτ (ρ = 0.197) and TSI_{MRT} (r = 0.261) was small and was not significant for either variable. In agreement, Buchheit et al. (2012) found that the relationship between percentage decrement during a repeated-sprint task (6 x 30 s, with 2 minutes passive recovery between sprints) and TSI was not significant, although the exact strength of the relationship was not reported. In contrast, Ufland et al. (2013) found a large (r = 0.53) correlation between muscle oxygen uptake in recovery and the adjusted mean sprint time (the adjusted mean sprint time was used as an index of fatigue), although an important distinction was made between the muscle reoxygenation rate and muscle oxygen uptake in recovery (Ufland et al., 2013). The analytical procedure that was used in Chapter 4, which was the measurement of the muscle reoxygenation rate, facilitated the assessment of the natural recovery kinetics of the NIRS data (Buchheit et al., 2011).

The modelling of lactate concentration in recovery is also not straightforward, as blood lactate concentration will continue to rise for several minutes after high-intensity exercise. Indeed, in Chapter 4, lactate concentration increased for ~ four minutes after the sprint, which was in agreement with previous research (Fujitsuka et al., 1982; Merrels et al., 2019; Withers et al., 1991). The relationship between performance recovery and the lactate clearance velocity constant was, however, trivial (r = -0.026). In contrast, Messonier et al. (1997) found a large (r = 0.67) and significant correlation between lactate disappearance and the power output that an individual could maintain during a rowing task. That being said, de Aguiar et al. (2015) reported that the correlation between repeated-sprint performance and the mean sprint time adjusted for best sprint time was small (r = 0.26). Concerns were also raised in Chapter 4 about the consistency of the blood lactate clearance velocity constant. It was, therefore, suggested that lactate concentration should in future be recorded for a longer duration in recovery to help to improve the reliability of the measurement. Overall,

therefore, there was no evidence to suggest that the physiological variables that were included in Chapter 4 should be a key objective for sprint cycling performance recovery.

8.1.3. Current Practice

In Chapter 4, to facilitate the accurate assessment of muscle oxygenation status using NIRS, the participants were required to remain stationary on the ergometer throughout the recovery period. The strict passive recovery, therefore, provided construct validity for the NIRS recording, but would have lacked ecological validity. In a real-world scenario, the riders would first need to continue cycling around the track until they came to a stop, before considering any recovery strategies. Following communication with British Cycling and conversations with an elite rider, it was established that when the time between races was brief, the practice that was undertaken by sprint cyclists was to perform a mixture of active and passive recovery. Therefore, in Chapter 5, the effect of passive recovery was contrasted with a mixed recovery protocol that was designed to reflect real-world practice. The intensity of the active recovery periods was described by the elite rider as being "a spin of the legs with no effort", with the removal of lactate being identified as a key objective. Whilst the findings in Chapter 4 did not indicate that lactate clearance was essential for performance recovery, asking the participants to simply spin the legs in recovery with no effort, would lack a degree of experimental control. Therefore, the intensity of the active recovery that was performed in the mixed recovery protocol was specifically chosen to maximise lactate clearance. The results from the study revealed that MPO was not affected by the recovery activity undertaken, but when compared to passive recovery, PPO was higher in the second sprint when a mixture of active and passive recovery was undertaken. Lactate concentration was also found to differ between conditions over time, but *post-hoc* tests were unable to identify the cause of the interaction effect.

The only other study that has previously contrasted the effects of active and passive recovery on repeated-sprint cycling performance using a sprint duration (30 s) and a recovery period (15 minutes) that could occur in the Match Sprint competition, was by Kirkpatrick and Burrus (2020). Eight performance measures (PPO, PPO relative to body mass, MPO, MPO relative to body mass, total work, fatigue index, peak RPM, mean RPM) were included in their analysis. However, not one of the variables was affected by the recovery activity undertaken. Therefore, the results of Chapter 5 regarding PPO differed to those of Kirkpatrick and Burrus (2020), but the findings with respect to MPO were in agreement. Two major differences existed between the study by Kirkpatrick and Burrus (2020) and Chapter 5. One concerned the sex of the participants and the other concerned their training background. Recreationally active women were recruited by Kirkpatrick and Burrus (2020), whereas strength-trained men participated in Chapter 5. Strength-trained men produce greater power outputs during cycling sprints than sedentary men (Pazin et al., 2011). Greater power outputs will also

typically be produced by men than women (Alper et al., 2018; Billaut et al., 2003; Billaut & Bishop, 2012; Esbjörnsson-Liljedahl et al., 1999; Esbjörnsson-Liljedahl et al., 2002; Falgairette et al., 2004; Hunter et al., 2023; Mageean et al., 2011; Perez-Gomez et al., 2008). A larger power output in the first sprint could result in a greater reduction in performance during a subsequent sprint (Billaut & Bishop, 2012; Bogdanis et al., 1995; Bogdanis et al., 1996a; Bogdanis et al., 1996b). The average PPO achieved in the first sprint in Chapter 5 was ~ 600 W higher than the average PPO that was produced in the first sprint in the study by Kirkpatrick and Burrus (2020). Greater levels of fatigue could, therefore, explain the between study difference that was found in the effect of recovery method on PPO.

Whilst no other study has assessed the benefits of performing either an active or a passive recovery on sprint cycling performance using sprint and recovery durations that may occur in the Match Sprint competition, repeated-sprint cycling performance has been evaluated using sprint durations of either 15 s or 30 s, with recovery durations ranging from 15 s to 7.5 minutes. When four minutes separated each of six 30 s WAnTs, Lopez et al. (2013) found that PPO was significantly higher in the second sprint when passive recovery was performed, but that MPO was significantly higher in sprints five and six when an active recovery was undertaken. When four WAnTs were performed with 7.5 minutes separating each sprint, Wahl et al. (2013) found no significant differences in either MPO or PPO over the sprints. Therefore, the findings by Lopez et al. (2013) and Wahl et al. (2013) differ to each other and to the findings in Chapter 5. When two cycling sprints were performed 15 s apart (a 15 s sprint followed by a 30 s sprint), Dupont et al. (2007) found that MPO and PPO were significantly lower when an active recovery was performed between sprints, but when four minutes separated two cycling sprints (both 30 s sprints), Bogdanis et al. (1996a) found that MPO was greater in the second WAnT when an active recovery was performed between sprints. The authors also stated that the difference was primarily due to the higher power outputs that were produced during the first 10 s of the sprint, even though PPO recovery did not differ between conditions (Bogdanis et al., 1996a). Therefore, the specificity of the task, as well as the duration between sprints, could affect the response to the recovery activity undertaken, meaning that the findings in Chapter 5, namely an improvement in recovery PPO, but no effect on the recovery of MPO, provided unique and relevant practical data for athletes and coaches regarding the effects of current practice on performance recovery.

The role of lactate/acidosis in performance recovery was not the primary focus of the investigation in Chapter 5. Nonetheless, the removal of lactate was stated by the elite rider, as being a main objective in the recovery period. Capillary blood lactate concentration was found to differ over time depending on the recovery activity; although, *post hoc* tests did not identify any differences at the individual recovery time-points. It was, therefore, suggested that the duration of active recovery may have been insufficient for identifiable differences to have occurred, especially as four minutes of

passive recovery was included in the mixed recovery protocol. In agreement, both Bogdanis et al. (1996a) and Dorado et al. (2004) reported no differences in lactate concentration between active and passive recovery conditions when four minutes separated two cycling sprints (Bogdanis et al., 1996a) or when four bouts of high-intensity exercise (time-to-exhaustion cycling at an intensity equating to ~ 120% $\dot{V}O_{2max}$) were undertaken with five minutes separating each bout. That being said, Baldari et al. (2005) did find a greater reduction in lactate concentration just three minutes after an active recovery was performed following six minutes of running at a velocity that would elicit ~ 90% $\dot{V}O_{2max}$ (end exercise lactate concentration ~ 8.6 $mmol \cdot L^{-1}$) and Kirkpartick and Burrus (2020) found that lactate concentration was significantly lower five minutes after a WAnT following active recovery. The intensity of the active recovery periods that were performed in Chapter 5 was specifically chosen to optimise lactate clearance. Nonetheless, it is possible that a superior recovery approach exists, both from a lactate clearance context, as well as from a performance restoration perspective.

8.1.4. Potential Motivation and Repeated Sprint Performance

Effort is the amount of mental or physical energy given to a task (Abbiss et al., 2015). The maximum amount of effort that an individual is prepared to exert to satisfy a motive is termed their potential motivation (Marcora, 2008). Sprint duration (de Jong et al., 2015; Glaister et al., 2019; Wittekind et al., 2011) and knowledge about the demands of the task (Billaut et al., 2011) have been found to affect potential motivation. Therefore, in Chapter 5, the effect of an alteration in second sprint duration was used to assess whether a change in effort could provide an explanatory factor for the reduction in second sprint performance that was found in Chapter 4, as opposed to the assumption that this was as a result of a fatigue-based physiological limitation. The interview data recorded in Chapter 5 suggested that sprint duration did positively affect the approach taken to the second sprint, at least for some participants. An interaction effect was also found between sprint number and second sprint duration on both MPO and PPO, although as *post hoc* tests were unable to identify the cause of the interactions, it appeared that further research was justified into the effects of effort on repeated-sprint performance.

In Chapter 7, the experimental intervention was a simulated competition, which was devised to maximise the motivational response. Simulated competition has been found to enhance exercise performance (Corbett et al., 2012; Williams et al., 2015; Viru et al., 2010), but from a cycling perspective, this was over distances that were 2 km (Corbett et al., 2012) or greater (16.1 km) (Williams et al., 2015). When the effect of competition was examined over a shorter distance, a 1 km time-trial, Wood et al. (2020) found that competition did not affect performance. Therefore, in Chapter 7, in order to optimise the effort response from the participants, additional motivational elements were included in the simulated competition. The approach taken was similar to the methods

that were used by Viru et al. (2010); namely, strong verbal encouragement was provided and the participants were competing for rewards that were devised to incentivise the participants to better their own previous performance, as well as the performance of the other participants. The participants were also ranked on a leaderboard and received performance feedback. The findings of a higher MPO and PPO in the simulated competition indicated that the inclusion of multiple motivational components could have created an additive effect on effort.

The perceived influence of each motivational element was also assessed using Likert scale ratings. No differences were identified in the ratings, with positive ratings generally being provided for all elements. In fact, only three negative markings (3.75% of the ratings) were made, supporting the notion that the external influences may have had an additive effect on performance. Of course, it should also be acknowledged that a bias in response styles is a possibility, with an acquiescence style referring to a tendency to use positive ratings (Pleininger & Heck, 2018). It would not, however, be possible to differentiate between a bias in the response style and an overall genuine effect. The decision to approach the problem with all of the motivational components being included in a single trial was in an attempt to maximise the motivational response. Further research into the effect of each component in isolation, and in combination, would now help to clarify the relative contribution that each element made to the changes in performance that were found.

The inclusion of the financial rewards in Chapter 7 could have been questioned following the responses provided to the questionnaire in Chapter 5. In Chapter 5, at the end of the study the participants were asked why they chose to participate, as well as whether the offer of a financial reward had affected their approach to the sprints. No participants referenced the financial reward as a reason for study participation and in response to the specific question about the reward, the majority of participants (63%) stated that the reward had little or no influence on their approach to the sprints. Previous research has also indicated that financial incentives did not affect cycling time-trial performance over shorter (1.5 km and 4 km) or longer distances (20 km) (Hulleman et al., 2007; Skorski et al., 2017). That being said, rewards have frequently been offered in research projects to incentivise performance (Brinkmann et al., 2021) and there is evidence to suggest that financial rewards could positively affect exercise performance (Shi et al., 2021). As a result, greater efforts were made in Chapter 7 to ensure that each participant would perceive that they had a chance of winning a reward.

With regards to the other four motivational elements included in Chapter 7 (crowd presence, leaderboard, performance feedback, and verbal encouragement), only the effect of verbal encouragement has directly been assessed on repeated-sprint performance. In the study by Edwards et al. (2018), two WAnTs were performed five minutes apart. PPO, average PPO, and MPO were recorded. The assessment of PPO was, however, the comparison of the highest power output over

both sprints in each condition and MPO and mean PPO were averages over both sprints in each condition. Therefore, the analytical approach did not allow for the possibility that the effect of verbal encouragement on performance could differ between sprints. It would seem reasonable to suggest that an enhanced power output during the first sprint could elicit greater levels of fatigue, which could then have a greater detrimental effect on subsequent performance. In a series of repeated-sprint studies, Bogdanis et al. (1995, 1996a, 1996b) consistently reported a strong negative correlation between the power output recorded during the first sprint and the recovery power output (power output in sprint 2 relative to power output in sprint 1). The findings in Chapter 7 did not indicate that there was an interaction effect between sprint number and condition, which could either be interpreted to suggest that the motivational response remained the same across sprints or that the motivational response was actually greater in the second sprint, as the expectation would be that greater levels of fatigue would have been generated during the first sprint. In other words, to match the same degree of performance deterioration, the motivational effect would have to be greater in the second sprint. Whether this effect would begin to change if further sprints were performed or if the participants were repeatedly exposed to the same stimuli, is not known.

8.1.5. The Effects of Simulated Competition on Physiological Stress Markers

In preparation for a competition, as well as in response to competition, an athlete may experience an increase in sympathetic arousal (Díaz et al., 2012). Athletes may, therefore, be in a heightened state of readiness to perform in a competition, which could explain the improvements in MPO and PPO that were seen in Chapter 7. There are two main components to the physiological response to stress – the autonomic nervous system and the hypothalamic-pituitary adreno-cortical axis. Whilst these stress response systems work in a coordinated fashion (Kivlighan & Granger, 2006), changes in sympathetic activity may provide a more sensitive measure of the stress experienced during a sports competition (De Pero et al., 2021). AA can be measured in saliva and provides a marker of sympathetic activity. HRV also provides an assessment of autonomic activity. Therefore, AA and HRV were measured in Chapter 7, with recordings being made at rest, after the warm-up, and at the end of the trial (during both the control and the simulated competition trials). AA activity, AA output, and LFnu power were found to be higher in the simulated competition, whereas HF power and HFnu power were lower. Changes in LF power, RMSSD, SD1, SD2, and SDNN were also found between conditions over time. However, the differences between conditions were only found at the end-of-trial time-point.

When analysing physiological data, one methodological challenge is to decide how to evaluate the variables that have been measured. For example, when analysing HRV, as many as 115 metrics have been reported in the literature (Smith et al., 2013). With regards to AA, both

AA activity and AA output can be calculated. Previous research that has assessed AA as a stress marker during a sports competition has only measured AA activity (Azarbayjani et al., 2011; Caprancia et al., 2012; Caprancia et al., 2017; Chennaoui et al., 2016; Chiodo et al., 2011; Dehghan et al., 2019; De Pero et al., 2021; Kivlighan & Granger, 2006; Sinnott-O'Connor et al., 2018; Trochimiak & Hübner-Woźniak, 2014). Bosch et al. (2011), however, criticised this approach, stating that it is the amount of AA that is secreted per unit of time that relates to the sympathetic response, not the activity of the enzyme. Therefore, if studies have only considered AA activity, a change in saliva flow-rate, which is also a sympathetic response, could have impacted the findings. That being said, in Chapter 7, both AA activity and AA output were found to be higher in the simulated competition, meaning that irrespective of the metric that was used to assess the sympathetic response, the implication was that competition elicited an increase in stress throughout the trial. It was, therefore, concluded that the AA measurements indicated that the participants were in a heightened state of arousal at their resting measurement and that they then remained in this heightened state for the remainder of the trial. The difference that was found between the pattern of the response of AA activity and AA output, related to the effect over time, suggesting that the 27 s sprints may not have led to an increase in AA production. A finding that does stimulate questions for future research that wishes to assess AA as a marker of exercise induced stress, but it does not change the conclusions that were drawn in Chapter 7 regarding the effects of a simulated competition on AA.

With regards to HRV, the metrics that have most commonly been reported in the analysis of HRV during a sports competition (LF power, LFnu, LF:HF ratio, HF power, HFnu, SD1, SD2, SDNN, and RMSSD) were selected *a priori* for analysis. A higher LFnu and a lower HF power and HFnu measurement could be interpreted to suggest that greater stress was experienced throughout the competition trial, supporting the outcome for AA activity and AA output. However, the statistical analysis of HF power was complicated by violations of normality that could not be rectified by transforming the data, meaning that the possibility of an interaction effect was not tested. One of the benefits of normalising the data from spectral indices is that it improves their distribution (Burr, 2007). However, the interpretation of the normalised HF and LF data is complicated. The HF power band is strongly associated with parasympathetic activity, but the LF band is not a pure index of sympathetic activity (Heathers, 2014). Shaffer et al. (2017) suggested that as much as half of the variability in the frequency band may be due to parasympathetic activity. When considering the raw LF power data in Chapter 7, LF power was found to be lower at the end-of-trial time-point in the simulated competition condition than it was in the control condition. Therefore, the decrease in LF power in the simulated competition suggests that parasympathetic withdrawal may have been greater than the increase in LF power as a result of sympathetic activation.

RMSSD, SD1, SD2, and SDNN were also all found to be lower at the end-of-trial time-point in the simulated competition condition. No other study that has considered HRV in a

competition environment has taken multiple HRV measurements during the competition. Instead, measurements have always been taken either in the morning (D'Ascenzi et al., 2014; Mateo et al., 2012; Morales et al., 2013), a few hours prior (Edmonds et al., 2013), or just before the competition (Cervantes et al., 2009; Mateo et al., 2012; Murray & Raedeke, 2008; Souza et al., 2019). Therefore, the finding of a change in the HRV metrics LF power, RMSSD, SD1, SD2, and SDNN differing between conditions only at the end of the trial, provided a novel finding into the effects of simulated competition on HRV. It was acknowledged that the change in exercise performance that was found in the simulated competition may have created greater physiological stress, which could have generated the changes that were observed in these HRV metrics. That being said, whilst differences in recovery HRV were found to exist when lower intensity exercise (exercise at an intensity below the first ventilatory threshold) was contrasted with higher intensity exercise (exercise at an intensity either between the first and second ventilatory thresholds or exercise at an intensity that was greater than the second ventilatory threshold), differences did not exist in recovery HRV between the two higher exercise intensities (Seiler et al., 2007). In Chapter 7, it was, therefore, suggested that it was still most likely that the changes that were found in LF power, RMSSD, SD1, SD2, and SDNN were as a result of the environmental conditions. A competition being simulated in a laboratory may not generate the same pre-event anxiety that an athlete would experience leading up to a high-stake competition, where the preparation could have been for many months or even years. Therefore, overall, the HRV results supported the notion of an increase in stress under simulated competition conditions.

8.2. Limitations

All experimental studies in this PhD thesis were conducted with strength, sprint, or power trained men. The training background of the participants was chosen due to the importance of resistance training for track cyclists and the difficulty in recruiting a sufficient sample of specifically trained track sprint cyclists. Regarding the sex of the participants, it would have been possible to use a minimum power threshold as part of the inclusion criteria, but the effects of gender on sprint performance recovery are still not fully understood. Of course, it would also have been possible to only recruit women or to examine the impact of gender on the recovery response, but if a performance reduction effect exists, it is likely to be greater in men, so it would seem reasonable to first establish the existence of an effect before further investigations were undertaken. All research was also conducted in a laboratory on a static cycle ergometer, not on a track, meaning that the transferability of the findings still needs to be established.

8.3. Further Research

Additional further research is warranted into: i) analysing methods to improve performance recovery. This could include different intensities of active recovery or the use of an ergogenic aid, although the short duration of the recovery period does present a logistical challenge; ii) modelling the time course of performance recovery with more data points (recovery times), especially with recovery times that are greater than 12 minutes; iii) contrasting the time-course of performance recovery when either an active or a passive recovery is performed; iv) evaluating the transferability of the current findings to elite level sprint cyclists; v) assessing performance recovery on the track; vi) assessing the effects of exercise performance on HRV, when intensity remains in the severe domain; vii) investigating the effect of motivational influences on repeated-sprint performance, when more sprints are performed; viii) examining whether an effect of competition would exist if the environment was repeatedly experienced; ix) examining the individual and combined effects of the motivational elements that were included in the simulated competition condition; x) investigating the effects of exercise at various intensities on AA activity and AA output; and xi) exploring the effects of gender on performance recovery, as well as analysing the recovery response when women are categorised based on their PPO.

8.4. Practical Recommendations

This PhD thesis has addressed questions about recovery for sprint cycling performance, as well as investigating the effect of potential motivation on repeated-sprint performance. Overall, the findings have provided guidance for practitioners, coaches, and sport scientists. From a performance perspective, given that brief (10 – 30 minutes) recovery periods occur frequently during the Match Sprint competition, athletes, coaches, and supporting sports scientists should consider ways of enhancing recovery to improve the chances of success. Whilst PPO is an important attribute for sprint cyclists, as the recovery of MPO was not affected by performing the current recovery protocol, further exploration was recommended to establish whether a superior recovery strategy exists. The relationship between performance restoration and the physiological variables that were measured in Chapter 4 ranged from trivial to moderate, but none were significant. Therefore, there was no evidence to suggest that improving $\dot{V}O_{2max}$, the rate of muscle reoxygenation, or enhancing lactate clearance, should be a training priority for a track sprint cyclist. In contrast, manipulation of the environmental conditions, with the simulation of a competitive environment, could positively affect effort and performance. The concurrent changes in the physiological stress markers also suggested that the stress of simulated competition heightened sympathetic arousal, which may have generated the changes in performance that were found.

From a research perspective, it was recommended that when modelling lactate clearance following intense exercise, data should be recorded for longer than 12 minutes, with 30 minutes being suggested as a recommended recording duration. With regards to stress markers and simulated competition, as the changes in the majority of HRV metrics were only found at the end of the trial, it was suggested that AA may provide a more sensitive measure of the stress that is experienced during a sports competition. However, as AA activity and AA output were found to be affected differently by exercise, it was suggested that these variables should not be viewed interchangeably. It was also suggested that when measuring HRV during a sports competition, it may be beneficial to take recordings after the event, as measurable changes in some metrics may only occur at a later time-point. Finally, the findings regarding the effect of effort on performance suggested that if researchers wish to generalise their findings to a practical sports competition, external motivational factors should be considered in the methodological design.

8.5. Conclusions

The aims and corresponding conclusions of this PhD thesis were:

1. To model the time-course of sprint cycling performance recovery and to examine the relationship between the performance recovery rate and various physiological factors that could potentially influence performance restoration

   Sprint performance recovery was modelled using both one- and two-phase exponential functions. The parameters of the one-phase exponential function suggested that performance recovery was complete within the 12-minute recovery period, albeit the amplitude of the recovery response was not 100%. When a two-phase exponential function was used to model the recovery response, the parameters of the model indicated that the complete, or close to complete, restoration of performance would occur, but after a substantially longer duration. The correlation between the performance recovery-rate and $\dot{V}O_{2peak}$, $\dot{V}O_{2off}$ kinetics, muscle reoxygenation, or the velocity constant for lactate clearance, ranged from trivial to moderate, but was never significant.

2. To assess the effects of the between sprint recovery activity that is currently practiced by elite sprint cyclists (a mixture of active and passive recovery) on performance restoration and to evaluate whether an alteration in second sprint duration would affect performance recovery

A mixture of active and passive recovery improved the recovery of PPO, but did not improve overall performance recovery, evaluated by MPO. An interaction effect was found between sprint number and second sprint duration, providing some evidence of a change in effort, although follow-up tests were unable to identify the cause of the effect.

3. To assess the effects of a simulated competition on repeated-sprint cycling performance and to examine whether any concurrent changes in physiological stress markers would occur

Both MPO and PPO were greater in the competition trial, providing evidence of a change in potential motivation caused by the atmosphere created in the laboratory when a competition was simulated. An increase in the stress response was also found in the simulated competition. A heightened state of readiness could, therefore, explain the improvements in performance that were found.

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Appendices

Appendix 1: Example of Physical Activity Readiness Questionnaire

St Mary's University Twickenham London

SCHOOL OF Sport, health and applied science

CONFIDENTIAL Medical History / Physical Activity Readiness Questionnaire (PAR-Q) FORM

This screening form must be used in conjunction with an agreed Consent Form.

Full Name: Date of Birth:

Height (cm): Weight (kg):

Have you ever suffered from any of the following medical conditions? If yes please give details:

	Yes	No	Details
Heart Disease or attack	☐	☐	_____
High or low blood pressure	☐	☐	_____
Stroke	☐	☐	_____
Cancer	☐	☐	_____
Diabetes	☐	☐	_____
Asthma	☐	☐	_____
High cholesterol	☐	☐	_____
Epilepsy	☐	☐	_____
Allergies	☐	☐	_____
Other, please give details	☐	☐	_____

Do you suffer from any blood borne diseases? If yes please give details;

Please give details of any **medication** you are currently taking or have taken regularly within the last year:

Please give details of any **musculoskeletal injuries** you have had in the **past 6 months** which have affected your capacity to exercise or caused you to take time off work or seek medical advice:

Other Important Information

During a typical week approximately how many hours would you spend exercising?

If you **smoke** please indicate how many per day:

If you drink **alcohol** please indicate how many units per week:

Are you currently taking any **supplements or medication?** Please give details:

Is there any reason not prompted above that would prevent you from participating within the relevant activity?

By signing this document I agree to inform the relevant individual(s) of any change(s) to my circumstances that would prevent me from participating in specific activities.

Signature (Participant): **Date:**

Signature (Test Coordinator*): **Date:**

**Test coordinator: The individual responsible for administering the test(s)/session and subsequent data collection*

Appendix 2: Example of Informed Consent Form

Name of Participant:

Title of the project: Modelling the recovery of sprint-cycling performance

Main investigator and contact details: Julian Dale, 130316@live.stmarys.ac.uk

Members of the research team:

1. I agree to take part in the above research. I have read the Participant Information Sheet which is attached to this form. I understand what my role will be in this research, and all my questions have been answered to my satisfaction.

2. I understand that I am free to withdraw from the research at any time, for any reason and without prejudice.

3. I have been informed that the confidentiality of the information I provide will be safeguarded.

4. I am free to ask any questions at any time before and during the study.

5. I have been provided with a copy of this form and the Participant Information Sheet.

Data Protection: I agree to the University processing personal data which I have supplied. I agree to the processing of such data for any purposes connected with the Research Project as outlined to me.

Name of participant (print)……..

Signed…….. Date……..

If you wish to withdraw from the research, please complete the form below and return to the main investigator named above.

Title of Project: _____

I WISH TO WITHDRAW FROM THIS STUDY

Name: _____

Signed: _____ Date: _____

Appendix 3: Example of COVID Declaration Form

COVID19 DECLARATION FORM FOR RESEARCH PARTICIPANTS

(To be signed in addition to the Consent Form – please read carefully)

- I have not displayed any COVID-19 symptoms or tested positive for COVID-19 in the 14 days before taking part in the research study.

- I have not knowingly been in contact with anyone displaying COVID-19 symptoms or who has tested positive for COVID-19 in the 14 days before taking part in the research study.

- I am not shielding due to underlying health conditions

- I have read, understood and signed the separate additional protocol specific Covid-19 safety measures (if appropriate)*

- I understand St Mary's has implemented Government measures to limit the spread of Coronavirus such as providing anti-bacterial hand gel throughout the University, implementing social distancing by utilising a one-way system for walking around the University, and limiting classroom participants. As such, all possible safety measures have been put in place to make the relevant research activity as safe as is reasonably possibly. I accept it is my own decision to participate and that St Mary's University cannot be held liable in the event I develop symptoms or Covid-19 infection.

I understand that if during the research, or within two weeks after the last research activity, I develop COVID-19 related symptoms (a new continuous cough, a high temperature, a loss or change to your sense of smell or taste), or come into contact with someone who has tested positive for COVID-19, I must:

- Report the developed symptoms to the lead researcher (lead researcher to insert their name and email address here)**

- Cease participation in the relevant study or studies with immediate effect

- Commence the Government Test and Trace process (https://www.gov.uk/guidance/nhs-test-and-trace-how-it-works) and complete the Test and Trace team advised isolation period if tested positive for COVID-19.

You will be able to resume participating in your St Mary's University research activity ONLY once you have received written confirmation from the Government Test and Trace management team that

you are permitted to do so, and/ or you have received a negative test result and feel well enough (evidence will be required).

* The researcher must ensure they provide participants with a copy of any protocol specific COVID-19 safety measures in advance of the testing and ask them to initial this alongside signing the consent form and CovId-19 Declaration Form.

** If the researcher receives notification from participants then they must immediately alert James Simms (james.simms@stmarys.ac.uk) and the Research Office (research@stmarys.ac.uk)

The effects of active and passive recovery on sprint-cycling performance.

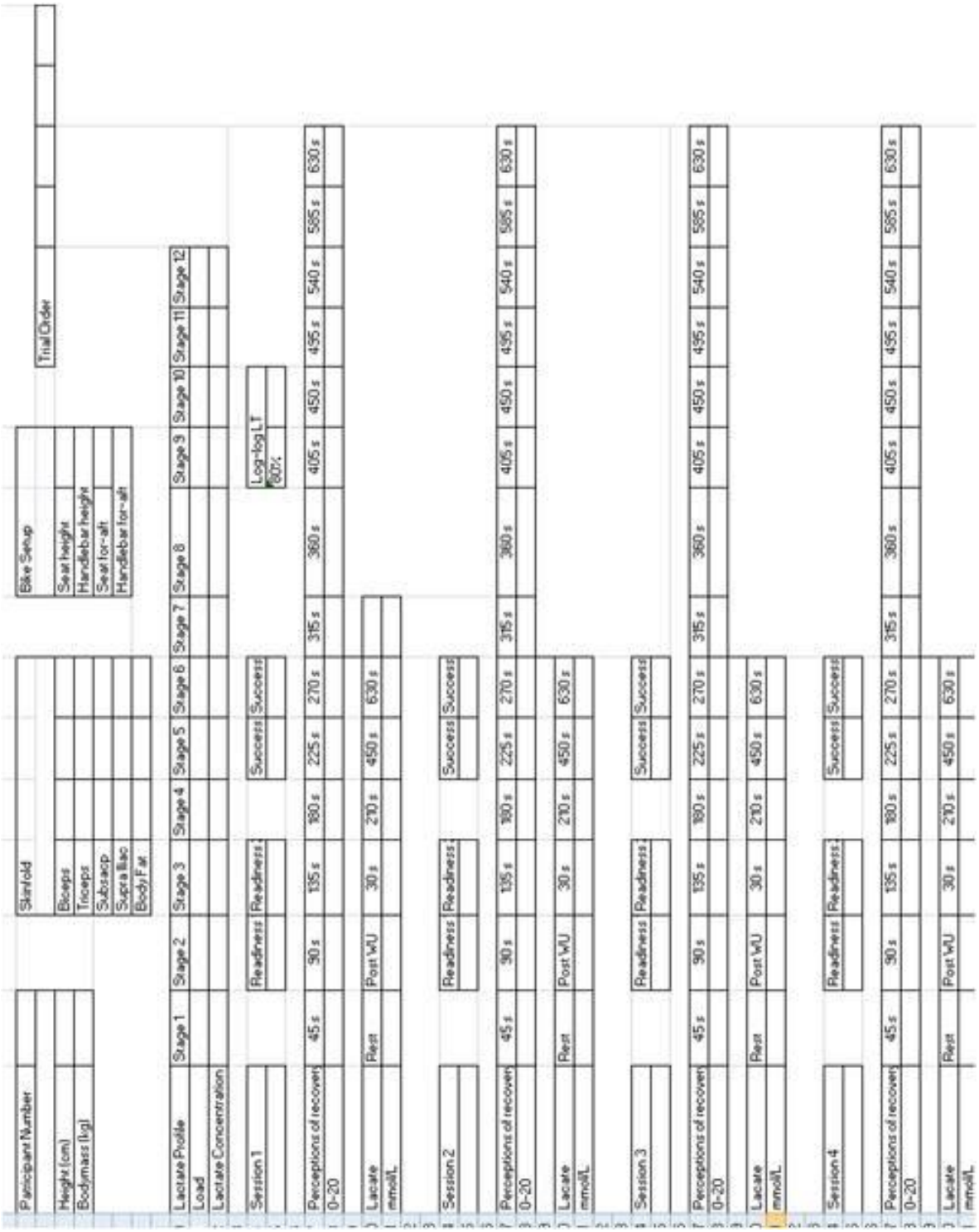
Name of participant _____

Signature of participant _____

Date _____

PARTICIPANT MUST BE GIVEN A COPY OF THIS FORM TO KEEP

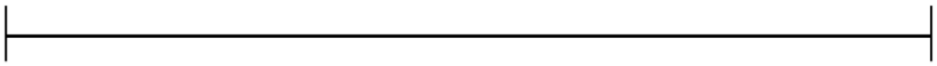
Appendix 4: Example of Data Collection Sheet



Participant Number			Skinfold								Bike Setup		Trial Order			
			Biceps								Seat height					
Height (cm)			Triceps								Handlebar height					
Bodymass (kg)			Subscap								Seat for-aft					
			Supra Iliac								Handlebar for-aft					
			Body Fat													
Lactate Profile	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5	Stage 6	Stage 7	Stage 8	Stage 9	Stage 10	Stage 11	Stage 12				
Load																
Lactate Concentration																
Session 1		Readiness	Readiness		Success	Success			Log-log LT							
									60%							
Perceptions of recovery	45 s	90 s	135 s	180 s	225 s	270 s	315 s	360 s	405 s	450 s	495 s	540 s	585 s	630 s		
0-20																
Lacate	Rest	Post WU	30 s	210 s	450 s	630 s										
mmol/L																
Session 2		Readiness	Readiness		Success	Success										
Perceptions of recovery	45 s	90 s	135 s	180 s	225 s	270 s	315 s	360 s	405 s	450 s	495 s	540 s	585 s	630 s		
0-20																
Lacate	Rest	Post WU	30 s	210 s	450 s	630 s										
mmol/L																
Session 3		Readiness	Readiness		Success	Success										
Perceptions of recovery	45 s	90 s	135 s	180 s	225 s	270 s	315 s	360 s	405 s	450 s	495 s	540 s	585 s	630 s		
0-20																
Lacate	Rest	Post WU	30 s	210 s	450 s	630 s										
mmol/L																
Session 4		Readiness	Readiness		Success	Success										
Perceptions of recovery	45 s	90 s	135 s	180 s	225 s	270 s	315 s	360 s	405 s	450 s	495 s	540 s	585 s	630 s		
0-20																
Lacate	Rest	Post WU	30 s	210 s	450 s	630 s										
mmol/L																

Appendix 5: Prepared to Sprint Visual Analogue Scale (Chapter 5)

How **prepared** do you feel to provide a maximum effort 18 s sprint?

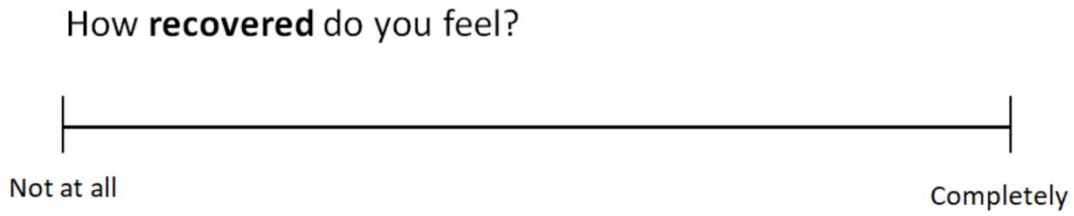


Not at all Completely

Instructions:

Please note that there are no right or wrong answers to this question. Thirty seconds prior to each sprint we would like you to indicate (via a strike) on this scale how prepared you feel to sprint. The scale ranges from '*not at all*', indicating that you do not feel prepared to perform the best maximal effort 18 s sprint, to '*completely*', suggesting that you feel prepared to perform the best maximal effort 18 s sprint possible.

Appendix 6: Perceptions of Recovery Visual Analogue Scale (Chapter 5)

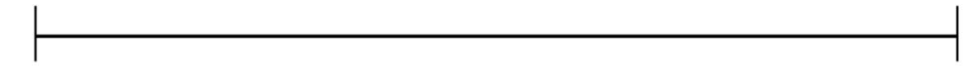


Instructions:

Please note that there are no right or wrong answers to this question. Between sprints, every 45 s, we would like you to indicate (using a strike) on this scale how recovered you feel. The scale ranges from '*not at all recovered*', this point would be reflective of your recovery status at the end of the sprint, to '*completely recovered*', suggesting that you believe that could perform another maximal effort 18 s sprint, matching your previous effort.

Appendix 7: Sprint Performance Rating Visual Analogue Scale (Chapter 5)

How would you **rate** your sprint performance?



Poor Optimal

Instructions:

Please note that there are no right or wrong answers to this question. After each sprint we would like you to indicate on this scale (via a strike) how you would rate your sprint performance. The scale ranges from '*poor*' indicating that your performance was a very bad reflection of your best sprint performance, to '*optimal*', suggesting that the sprint was a very good reflection of your best maximal sprint performance possible.

Appendix 8: Session Debrief Questionnaire (Chapter 5)

Study Title: The effects of recovery type on sprint cycling performance.

Session Debrief:

- How satisfied are you with today's sprint performances?

_____

_____

_____

- What do you feel mostly influenced your sprint performances?

_____

_____

_____

_____

- How do you feel that the recovery activity influenced your second sprint?

_____

_____

_____

Appendix 9: Study Debrief Questionnaire (Chapter 5)

St Mary's University Twickenham London

Study Title: The effects of recovery type on sprint cycling performance.

Study Debrief:

Thank you for your participation. It would be greatly appreciated if you could take a few minutes to answer a final few questions.

- Why did you choose to participate in this study?

_____

_____

_____

_____

- What do you feel mostly influenced your approach to the sprints?

_____

_____

_____

_____

- To what extent did the offer of a financial reward affect your approach to the sprints?

_____

_____

_____

_____

Appendix 10: Likert scale (Chapter 7)

Study Title: The effect of a simulated competition on repeated-sprint performance and on measures of physiological stress.

Participant Number: ____

- To what extent do you feel the following affected your sprint performance?

Crowd presence:

Large negative effect			No effect			Large positive effect
-3	-2	-1	0	1	2	3

Leaderboard:

Large negative effect			No effect			Large positive effect
-3	-2	-1	0	1	2	3

Performance feedback:

Large negative effect			No effect			Large positive effect
-3	-2	-1	0	1	2	3

Reward (two vouchers):

Large negative effect			No effect			Large positive effect
-3	-2	-1	0	1	2	3

Verbal encouragement:

Large negative effect			No effect			Large positive effect
-3	-2	-1	0	1	2	3

Appendix 11: Post-trial question (Chapter 7)

- You gave the following rating for xxx (e.g. crowd presence), why do feel that this factor had that effect?

St Mary's University Twickenham London

Appendix 12: Ethics Approval Sheet (Study 1/Chapter 4)

Name of proposer(s)	Julian Dale
Name of supervisor	Dr Mark Glaister
Programme of study	PhD
Title of project	Modelling the recovery of sprint-cycling performance

Supervisors, please complete section 1. If approved at level 1, please forward a copy of this Approval Sheet to the School Ethics Representative for their records.

SECTION 1: To be completed by supervisor.(for student research projects). Doctoral/MPhil applications must be referred to and reviewed by an Ethics Representative at Section 2 below.

☐ Approved at Level 1.

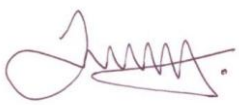
☒ Refer to Ethics Representative for consideration.

Name of Supervisor:	Mark Glaister		
Signature of Supervisor:		Date:	15/07/18

SECTION 2: To be completed by School Ethics Representative.

☒ Approved at Level 2.

☐ Level 3 consideration is required by Ethics Sub-Committee.

Signature of School Ethics Representative:		Date:	20 July 2018

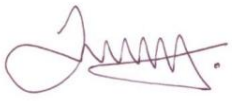
St Mary's University Twickenham London

Ethics Approval Sheet (Studies 2 & 3/Chapters 5 & 6)

Name of proposer(s)	Julian Dale
Name of supervisor	Dr Mark Glaister
Programme of study	PG Research
Title of project	The effects of active and passive recovery on sprint-cycling performance

Supervisors, please complete section 1. If approved at level 1, please forward a copy of this Approval Sheet to the Faculty Ethics Representative for their records.

SECTION 1: To be completed by supervisor.			
☐ Approved at Level 1. ☒ Refer to Faculty Ethics Representative for consideration at Level 2 or Level 3.			
Signature of Supervisor (for student research projects):		Date:	12/12/2019

SECTION 2: To be completed by Faculty Ethics Representative.			
☒ Approved at Level 2. ☐ Level 3 consideration is required by Ethics Sub-Committee.			
Signature of Faculty Ethics Representative:		Date:	16 December 2019

St Mary's University Twickenham London

Ethics Approval Sheet (Study 4/Chapter 7)

Name of proposer(s)	Julian Dale
Name of supervisor(s)	Dr Mark Glaister
Programme of study	PhD
Title of project	The effect of simulated competition on repeated-sprint performance and on measures of physiological and psychological stress.

Supervisors, please complete section 1. If approved at level 1, forward a copy of this Approval Sheet to the Faculty Ethics Representative for their records.

SECTION 1: To be completed by supervisor.(for student research projects). Doctoral/MPhil applications must be referred to and reviewed by an Ethics Representative at Section 2 below.

☐ Approved at Level 1.

☒ Refer to Ethics Representative for consideration.

Name of Supervisor:	Mark Glaister		
Signature of Supervisor:		Date:	05/09/22

SECTION 2: To be completed by Ethics Representative.

☐ Approved at Level 1

☒ Approved at Level 2

☐ Level 3 consideration is required by Ethics Sub-Committee.

Name of Faculty Ethics Representative:	Emily Martin		
Signature of Faculty Ethics Representative:		Date:	08/09/22