

Original Article

The short-term recovery of sprint cycling performance

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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, and oxygen uptake 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. Peak (PPO) and mean (MPO) power output were computed 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. Statistical significance was set *a priori* at $p < 0.05$. Effects of sprint number, recovery time, and a sprint number \times recovery time interaction were found for both PPO and MPO. *Post hoc* tests revealed significant differences between sprints at all time-points for both variables. The time constant (τ), 130.6 ± 95.6 s, of the one-phase exponential function (asymptotic amplitude [A_0] = $97.4 \pm 2.5\%$), suggested that performance recovery had stabilised within 12 minutes. However, the parameters of the two-phase function indicated that recovery was incomplete ($A_0 = 87.7 \pm 6.4\%$, $A_1 = 11.9 \pm 5.2\%$, $\tau_0 = 56.3 \pm 33.3$ s, $\tau_1 = 458.2 \pm 283.3$ s). The τ for MPO recovery was not significantly correlated with any of the physiological variables. The reduction in sprint cycling performance throughout the tested time-period could be meaningful for athletes competing in the Match Sprint.

Keywords: fatigue, recovery kinetics, repeated sprints, track cycling

1. 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 race. 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 would have had ~10 minutes between these races (Vieria, 2016). The short-term recovery of sprint cycling performance has been investigated using brief (5 s - 15 minutes) recovery time-periods (Bogdanis, Nevill, Boobis, & Lakomy, 1996; Glaister, Pattison, Dancy, & McInnes, 2014; Hebestreit, Mimura, & Bar-Or, 1993; 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). Glaister et al. (2014) modelled the recovery of peak power output (PPO) using one- and two-phase exponential functions. The subjects were first required to perform a 30 s fatiguing sprint, which was followed by a short sprint (5 s) after 5, 10, 20, 40, 80, or 160 s of recovery (Glaister et al., 2014). The two-phase exponential function significantly improved the model fit. Model parameters were reported for the group mean response, as well as the mean and standard deviation of the parameters derived for the individual participants (Glaister et al., 2014). From the information provided, it could be estimated that PPO would be fully restored after ~8 – 15 minutes.

Zabala et al. (2011) found that PPO did not differ between three Wingate Anaerobic Tests (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, Harnish, Chase, 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).

The 30 s WAnT is the most frequently used laboratory measure of sprint cycling performance (Driss & Vandewalle, 2013). In the Match Sprint, however, each head-to-head race is conducted over three laps of a 250 m track. The riders will also not simply attempt to complete the distance as fast as possible. The substantial reduction in air resistance that

can be achieved by following another rider (Craig & Norton, 2001), means that the tactics employed will dictate the duration of maximal effort. It has been estimated that during 15 s of maximal exercise, 88% of the energy would be derived from anaerobic pathways, decreasing to 82% for a 20 s sprint, and 73% for a 30 s sprint (Gastin, 2001). Muscle PCr and glycogen concentrations will decrease during a sprint (Bogdanis et al., 1996). Following the sprint, PCr resynthesis occurs via the rephosphorylation of creatine by aerobically produced adenosine triphosphate (McMahon & Jenkins, 2002). The post-exercise recovery of oxygen uptake (VO_2 off-kinetics) has been found to follow a similar time-course to PCr resynthesis (Rossiter et al., 2002), at least at exercise intensities that are below peak VO_2 ($\text{VO}_{2\text{peak}}$) (Glaister et al., 2014). Therefore, individuals with faster VO_2 off-kinetics could perform better during a repeated-sprint task. As near infrared spectroscopy (NIRS) facilitates the non-invasive measurement of oxygen delivery and utilisation directly at the muscle, NIRS may also offer a preferred means of assessing muscle reoxygenation (Ufland, Ahmaidi, & Buchheit, 2013).

An additional consideration is that PCr resynthesis (McMahon & Jenkins, 2002; Walter, Vandenborne, McCully, & Leigh, 1997), as well as the glycolytic pathway (Bogdanis et al., 1996), could be affected by muscle pH. The cause of muscular acidosis during exercise, and the role that lactate and hydrogen ion (H^+) production play in muscular fatigue, have been well documented and debated (Allen, Lamb, & Westerblad, 2008; Fitts, 2016; Robergs, 2019; Westerblad, 2016). The time-course of lactate recovery and performance restoration is different. However, it is still possible that an increase in H^+ concentration could limit sprint performance, meaning that faster lactate and H^+ clearance could be beneficial during repeated sprints.

At the elite level, the track cycling competition known as the Match Sprint may occur over several days and the riders may be required to race on multiple occasions on

each day. The recovery time between races varies depending on the competition schedule, but there is evidence to suggest that performance could be reduced during the shortest recovery periods that occur.

The aim of the current study was, therefore, to evaluate the recovery of sprint cycling performance, and to assess physiological variables that may influence the recovery time-course. It was hypothesised that: 1) the recovery of MPO would be diminished throughout, whereas the recovery of PPO would be complete at the 12-minute time-point; 2) the time constant (τ) of performance recovery would be positively associated with VO_2 off-kinetics, muscle re-oxygenation rate, and the lactate disappearance velocity constant.

2. Materials and Methods

Subjects

Fifteen male subjects, all over 18 years of age and regularly performing resistance training, volunteered to participate. The characteristics of the subjects are displayed in Table 1. To aid with the consistency in testing conditions, the subjects 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. Trials were typically conducted seven days apart at approximately the same time of day, with a minimum of 48 hours separating trials. Before commencing the first trial, the subjects were informed about the risks and benefits of participating. All subjects 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.

Table 1. Subject characteristics.

Age (years)	Height (m)	Body mass (kg)	Body fat (%)	$\text{VO}_{2\text{peak}}$ ($\text{ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$)
24 ± 6	1.81 ± 0.08	83.4 ± 8.4	14.8 ± 4.5	48.1 ± 6.3*

Note: data are displayed as mean ± standard deviation. $\text{VO}_{2\text{peak}}$ denotes peak oxygen uptake. *n = 14 for this variable as one subject was not comfortable with wearing the facemask.

Design

Familiarisation Trial (Trial 1)

At the start of Trial 1, measurements of stature and body-mass were taken. A four-site skinfold protocol was then used to estimate body-fat using a recognised formula (Durnin & Womersley, 1974). The cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands) 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 then asked to indicate their preferred leg to initiate the sprints. Consistent with previous research, each sprint in all trials began with the chosen leg being ~45° forward to the vertical axis (Billaut & Basset, 2007). A wireless NIRS transmitter (PortaMon, Artinis Medical Systems, Elst, The Netherlands) was positioned above the vastus lateralis of the non-drive initiation leg. The PortaMon device is frequently used in sports science research due to its reliability and practical applicability (McManus, Collison, & Cooper, 2018). The PortaMon is a continuous wave system, which corrects for light scattering using spatially resolved spectroscopy (McManus et al., 2018). The sensor placement was set using guidelines (Hermens et al., 1999). Prior to application, skinfold thickness was measured using callipers. The sensor location was then shaved and cleaned with an alcohol swab. 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 taped over the sensor to limit external light sources affecting the reading. A facemask and head-cap assembly

were worn (Hans Rudolph, Kansas City, USA) for the breath-by-breath measurement of VO_2 (Oxycon Pro, Erich Jaeger GmbH, Hoechburg, Germany). 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.

The subjects then sat passively on the cycle ergometer for five minutes before performing a standardised warm-up. The warm-up protocol was based on guidelines for an 18 s cycling sprint (see Table 2) (Coaching and Sports Science Division of the United States Olympic Committee, 2004). An 18 s performance measure was selected following the analysis of a power output profile of a Flying 200 undertaken by an elite sprint cyclist, as well as video analysis of Flying 200 performances at the 2012 London Olympics. The Flying 200 is the qualifying event for the Match Sprint, whereby each rider must individually complete 3.5 laps of the track, with the time for the final 200 m being recorded. The Wingate program was, therefore, selected in the Lode Ergometer Manager (LEM) software and the time set to 18 s. When using the Wingate mode in the LEM software, a constant torque is applied during the sprint. The torque factor was set at $0.909 \text{ Nm}\cdot\text{kg}^{-1}$. 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, Bozic, Bobana, Nedeljkovic, & Jaric, 2011). All sprints were undertaken from a stationary start and the subjects 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 $25 \text{ W}\cdot\text{min}^{-1}$) was performed to exhaustion for the

Table 2. Warm-up and Sprint Protocol.

Duration (s)	Resistive Load ($\text{Nm}\cdot\text{kg}^{-1}$)	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.

determination of VO_2 peak (highest 30 s rolling average).

Baseline Trial (Trial 2)

During the baseline trial the subjects performed the standardised warm-up and a single 18 s sprint, which was followed by 12 minutes of passive recovery. Tissue saturation index (TSI) and breath-by-breath VO_2 were recorded throughout the recovery period. A 20 μl capillary blood sample was 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 using an automated analyser (Biosen C-Line, EFK Diagnostics, Barleben, Germany).

Experimental Trials (Trials 3-8)

Following the same warm-up and sprint protocol as before, a second 18 s sprint was performed. The recovery duration between sprints was varied (45, 90, 135, 180, 360, 720 s). The recovery durations were selected considering previous sprint cycling research (Bogdanis et al., 1996; Hebestreit et al., 1993; Glaister et al., 2014; Zabala et al., 2011), the recovery times that occur during the Match Sprint, and following a period of pilot testing that evaluated perceptions of recovery. The subjects were not informed about the recovery duration between sprints until the first sprint had been undertaken, as the information provided to the subjects could

affect repeated-sprint performance (Billaut, Bishop, Schaerz, & Noakes, 2011). Between sprints the subjects rested passively on the ergometer and following the second sprint, the subjects 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 VO_2 were monitored following the first sprint, enabling comparisons to be made with baseline. The 720 s trial was always the final trial. The order of the other experimental trials was randomised.

Data Analysis

Performance

The sampling rate of the ergometer was 5 Hz. PPO was defined as the highest value recorded during the sprint and MPO was the average power output over the entire 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 1 & 2), with the goodness of model fit computed by the software (Matlab R2019a, Mathworks, Natick, U.S.).

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

$$PR(t) = (A_0 * \left(1 - e^{-\frac{(t-b_0)}{\tau_0}}\right) * U_0) + (A_1 * \left(1 - e^{-\frac{(t-TD_1)}{\tau_1}}\right) * U_1) \quad \text{Equation 2}$$

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 $t \geq TD_1$.

Blood Lactate Concentration

The blood lactate response in recovery was modelled using Equation 3 (Beneke, Wittekind, Mühling, Bleif, & Leithäuser, 2010). In addition to the appearance and disappearance velocity constants, the model also allows the maximum post-sprint blood lactate concentration, and the time when the maximum concentration occurs, to be determined (Beneke et al., 2010).

$$BLa(t) = \frac{A * k_0}{k_1 - k_0} * (e^{-k_0 t} - e^{-k_1 t}) + (BLa_{pre\ sprint} - BLa_{rest}) * e^{-k_1 t} + BLa_{rest} \quad \text{Equation 3}$$

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

Near Infrared Spectroscopy

The sampling rate was set at 10 Hz. Due to complications in the oxygenated haemoglobin signal during exercise, as a result of alterations in blood flow, in-line with previous research the NIRS analysis was limited to TSI (Dupont, Moalla, Matran, Bertoin, 2007). The recovery data were modelled using Equation 4. Muscle re-oxygenation may also be delayed in subjects with a higher $\text{VO}_{2\max}$ (Nagasawa, 2013). Therefore, in addition to τ , the mean response time (MRT) was calculated using Equation 5.

$$TSI(t) = TSI_{end} + (A_0 * \left(1 - e^{-\frac{(t-TD_0)}{\tau_0}}\right) * U_0) \quad \text{Equation 4}$$

$$MRT = TD_0 + \tau_0 \quad \text{Equation 5}$$

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.

Oxygen Uptake

VO_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. The data were modelled from the peak post-sprint value using Equation 6. 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, Rossiter, Ward, & Whipp, 2001). Constraints were applied to Equation 6 to ensure that the eventual resting value would be physiologically viable. This was achieved by using the resting value for each participant.

$$VO_2(t) = VO_{2end\ peak} - (A_0 * (1 - e^{-\frac{(t-TD_0)}{\tau_0}}) * U_0) - (A_1 * (1 - e^{-\frac{(t-TD_0)}{\tau_1}}) * U_0) \quad \text{Equation 6}$$

where $VO_2(t)$ is the oxygen uptake at any time-point, $VO_{2end\ peak}$ 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 $t \geq TD_0$.

Statistical Analysis

Statistical analyses were conducted using SPSS software (SPSS for Windows Version 24, SPSS Inc, Chicago, USA). Values are reported as mean \pm standard deviation. Statistical significance was set *a priori* at $p < 0.05$. Differences in both PPO and MPO were assessed using a two-way (sprint number \times 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 ρ - dependent on whether the assumptions of normality were satisfied)

were conducted to assess the relationship between the τ of performance recovery and the recovery τ of TSI ($TSI\tau_0$) and VO_2 (VO_{2offT0}), as well as VO_{2peak} , the MRT for TSI (TSI_{MRT}), and the clearance velocity constant for blood lactate ($BLak_1$). The magnitude of each relationship was interpreted using guidelines (Hopkins, Marshall, Batterham, & Hanin, 2009). To assess for a performance training effect a one-way ANOVA was performed on PPO and MPO in session order. The coefficient of variation and the intraclass correlation coefficient (ICC), as well as their 95% confidence limits, were then calculated using recommended procedures (Schabert, Hawley, Hopkins, & Blum, 1999). Finally, in order to evaluate the consistency of the model parameters of the physiological variables ($BLak_1$, TSI_{MRT} , $TSI\tau_0$, VO_{2offT0}) between the baseline and final trials, the coefficient of variation was computed using established methods, as outlined elsewhere (Buchheit, Ufland, Haydar, Laursen, & Ahmaidi, 2011).

3. Results

Performance

The performance recovery percentage, modelled with both one- and two-phase exponential functions, is displayed in Figure 1. The model parameters are displayed in Table 3. 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 subjects (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 2. To ensure continuity, all correlations with the physiological variables were made using τ_0 derived from the one-phase exponential function.

Table 3. Model parameters and the goodness of model fit for one- and two-phase exponential functions fit to the recovery percentage performance data.

	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: data are displayed as mean \pm standard deviation. 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.

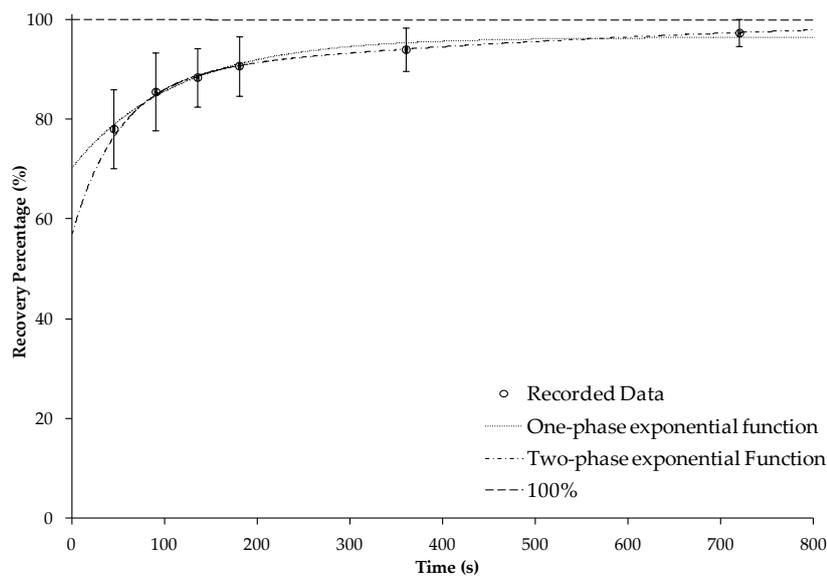


Figure 1. The recovery percentage of mean power output recorded at six recovery time-points fit with both one- and two-phase exponential functions. Data points display the mean and error bars the standard deviation.

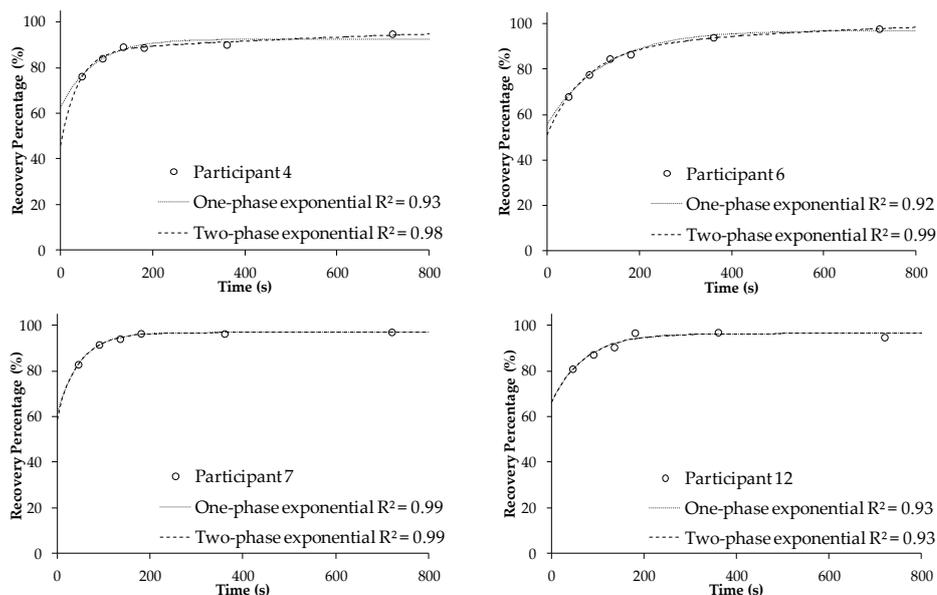


Figure 2. Example recovery percentage data for four representative participants. For participants 4 and 6, the model fit was improved with a double exponential function, whereas for participants 7 and 12, the model fit was not improved.

An effect of sprint number (PPO: $F_{(1,14)} = 73.177$, $p < 0.001$, $\eta^2_p = 0.839$; MPO: $F_{(1,14)} = 66.901$, $p < 0.001$, $\eta^2_p = 0.827$), recovery time (PPO: $F_{(5,70)} = 4.975$, $p = 0.001$, $\eta^2_p = 0.262$; MPO: $F_{(5,70)} = 36.294$, $p < 0.001$, $\eta^2_p = 0.722$), and a sprint number \times recovery time interaction (PPO: $F_{(2.617,36.639)} = 10.553$, $p < 0.001$, $\eta^2_p = 0.430$; MPO: $F_{(2.160,30.242)} = 52.095$, $p < 0.001$, $\eta^2_p = 0.788$) were found for both PPO and MPO. *Post hoc* tests revealed significant ($p < 0.05$) differences between Sprint 1 and Sprint 2 for both PPO and MPO at all recovery time-points (see Figures 3 & 4).

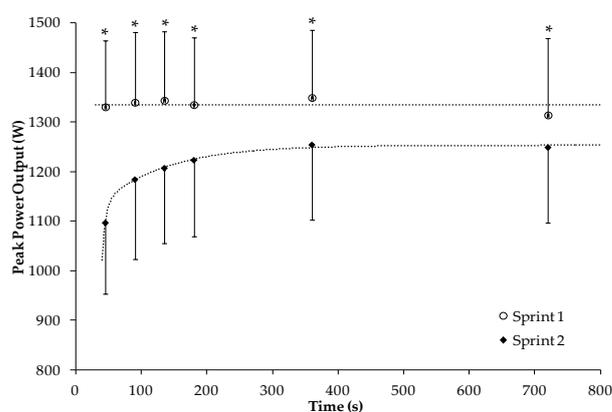


Figure 3. Sprint 1 and Sprint 2 peak power output at the six-recovery time-points. Data points represent the mean and error bars the standard deviation. Lines of best fit (linear and one-phase exponential) have been added. *denotes a significant difference ($p < 0.05$) in peak power output between sprints at the individual time-point.

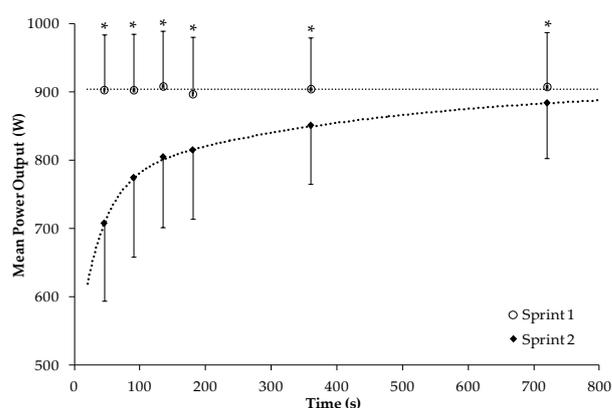


Figure 4. Sprint 1 and Sprint 2 mean power output at the six-recovery time-points. Displayed data represent the mean and error bars the standard deviation. Lines of best fit (linear and two-phase exponential) have been added. *denotes a significant difference ($p < 0.05$) in mean power output between sprints at the individual time-point.

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

Blood Lactate Concentration

The resting blood lactate concentration, as well as the parameters of the modelled response, can be found in Table 4. 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].

Near Infrared Spectroscopy

At the sensor location the skinfold measurement 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 5. 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.

Oxygen Uptake

VO_2 was not recorded for one participant due to feelings of constraint when wearing a facemask. The VO_{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 6. The coefficient of variation between the baseline and 720 s trials for τ_0 was 24.3% [18.1, 38.2].

Table 4. 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).

BL _{arest} (mmol·L ⁻¹)	BL _{apre sprint} (mmol·L ⁻¹)	A (mmol·L ⁻¹)	k ₀ (minutes ⁻¹)	k ₁ (minutes ⁻¹)	R ²	Peak Lactate (mmol·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: data are displayed as mean ± standard deviation. BL_{arest} denotes the blood lactate concentration at rest, BL_{apre sprint} is the post-warm up blood lactate concentration, A is the amplitude, k₀ the appearance velocity constant, and k₁ the disappearance velocity constant.

Table 5. Model parameters and the goodness of model fit for tissue saturation index following an 18 s sprint (n = 12).

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: data are displayed as mean ± standard deviation. 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.

Table 6. Model parameters and the goodness of model fit for oxygen uptake following an 18 s sprint (n = 14).

VO _{2end peak} (ml·min ⁻¹)	A ₀ (ml·min ⁻¹)	τ ₀ (s)	TD ₀ (s)	A ₁ (ml·min ⁻¹)	τ ₁ (s)	R ²
3898 ± 625	2956 ± 622	50.9 ± 13.4	13.8 ± 9.8	488 ± 201	592 ± 822	0.92 ± 0.05

Note: data are displayed as mean ± standard deviation. VO_{2end peak} denotes the highest post sprint value, A is the asymptotic amplitude, τ the time constant, and TD the time delay.

Relationship between performance recovery-rate and the physiological variables

The strength of the relationships between the τ of performance recovery and the physiological variables (BL_{ak1}, TSI_{MRT}, TSI_{τ0}, VO_{2offτ0}, and VO_{2peak}) ranged from negligible to moderate, but was never significant (see Figure 5).

4. 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 PPO and MPO 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, whereas VO₂ rose briefly following the sprint, before decreasing thereafter. Blood lactate concentration peaked 4.42 minutes after the

sprint, gradually declining over the recovery period tested. 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%. 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. Glaister et al. (2014) were specifically interested in the recovery of PPO

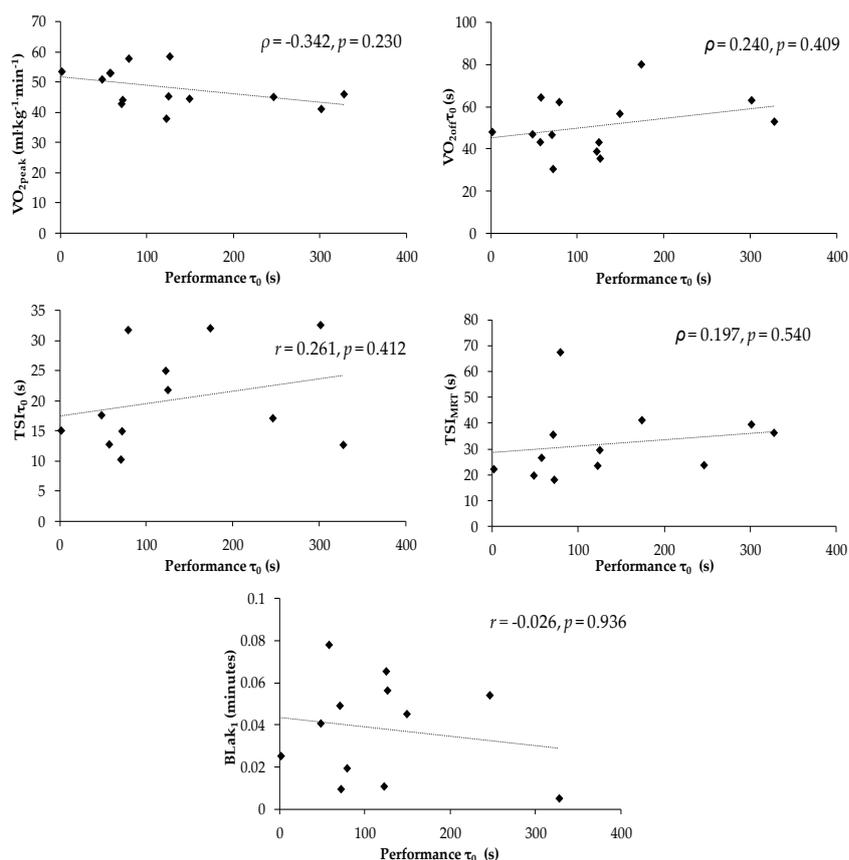


Figure 5. Relationship between performance recovery-rate (the time constant derived from the one-phase exponential function) and the physiological variables ($Blak_1$, $TSI\tau_0$, TSI_{MRT} , $VO_{2off\tau_0}$, and VO_{2peak}). For oxygen uptake variables $n = 14$, for tissue saturation index $n = 12$, and for blood lactate $n = 12$. Note: $Blak_1$ 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, VO_2 is oxygen uptake, and VO_{2peak} is peak oxygen uptake.

following a 30 s sprint. 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. Consideration was given to the recovery times that were selected in this study, although the collection of additional data points, around, or greater than 12 minutes, would have been beneficial. Comparison of the performance data did, however, reveal that both PPO and MPO were 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 had been fully restored after 10 minutes (Hebestreit et al., 1993). Whilst the smaller sample size may have limited statistical power in the study by Hebestreit et al. (1993), Zabala et al. (2011) found that both PPO and MPO did not differ when three WANts were performed 15 minutes apart. The subjects 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 subjects that produced the greatest power output during a WANt displayed the slowest resynthesis of PCr (Bogdanis et al., 1996), meaning that the recovery duration could be greater in those individuals. On the other hand, the type of training undertaken can affect physiological and performance adaptations (Mohr et al., 2007). Therefore, 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). An 18 s performance measure was selected in the current study, 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 lower PPO being found when the duration increases (de Jong et al., 2015, Wittekind, Micklewright, & Beneke, 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 subjects 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 PPO and MPO 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 subjects rested passively on the ergometer. This 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 re-oxygenation rate, VO_2 off-kinetics, $\text{VO}_{2\text{peak}}$, and blood lactate clearance, ranged from negligible to moderate, but was never significant. Research findings regarding the effects of muscle re-oxygenation (Buchheit, Abbiss, Peiffer, Laursen, 2012; Buchheit & Ufland, 2011), VO_2 off-kinetics (Buchheit, 2012; Buchheit et al., 2012; Dupont, McCall, Prieur, Millet, & Berthoin, 2010), and $\text{VO}_{2\text{max}}$ (Buchheit, 2012; Buchheit et al., 2012; Dupont et al., 2010; Glaister et al., 2014), on repeated-sprint performance, have been mixed. The coefficient of variation for the muscle reoxygenation parameters was similar to values that have previously been reported (Buchheit et al., 2011). Whilst the coefficient of variation for VO_2 off-kinetics was not dissimilar to the NIRS variables, it was greater than others have found (Mann, Webster, Lamberts, & Lambert, 2014). The

variability between the measurements of the blood lactate clearance velocity constant was, however, much larger, raising concerns about the suitability of the modelling procedure that was used. The goodness of model fit was comparable to the values that were computed for the other variables. The function was also specifically designed to model blood lactate concentration after exercise, beginning from a post warm-up elevated state (Beneke et al., 2010), although blood lactate concentrations were monitored by Beneke et al. (2010) for 30 minutes after exercise. Recording measurements for a longer recovery duration may, therefore, be necessary to improve the consistency of the clearance velocity constant.

5. Practical Applications.

The Match Sprint requires athletes to compete on consecutive days and on multiple occasions each day. The time between races can be as short as 10 minutes. The findings from the current study indicate that repeated-sprint performance may be reduced over this timeframe, 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 aerobic capacity, or those that experienced faster muscle reoxygenation, or lactate clearance, displayed a faster performance restoration-rate.

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References

1. Allen, D. G., Lamb, G. D., & Westerblad, H. (2008). Skeletal muscle fatigue: cellular mechanisms. *Physiological Reviews*, 88(1), 287-332. doi: 10.1152/physrev.00015.2007
2. Billaut, F., & Basset, F. A. (2007). Effect of different recovery patterns on

- repeated-sprint ability and neuromuscular responses. *Journal of Sports Sciences*, 25(8), 905-913. doi: 10.1080/02640410600898087
3. Billaut, F., Bishop D. J., Schaerz, S., & Noakes, T. D. (2011). Influence of knowledge of sprint number on pacing during repeated-sprint exercise. *Medicine in Science and Sports and Exercise*, 43(4), 665-672. doi: 10.1249/MSS.0b013e3181f6ee3b
 4. Beneke, R., Wittekind, A., Mühling, M., Bleif, I., & Leithäuser, R. M. (2010). Lactate response to short term exercise with elevated starting levels. *European Journal of Applied Physiology*, 110(1), 215-218. doi: 10.1007/s00421-010-1481-z
 5. Bogdanis, G. C., Nevill, M. E., Boobis, L. H., & Lakomy, H. K. A. (1996). Contribution of phosphocreatine and aerobic metabolism to energy supply during repeated sprint exercise. *Journal of Applied Physiology*, 80(3), 876-884. doi: 10.1152/jappl.1996.80.3.876
 6. Buchheit, M. (2012). Repeated-sprint performance in team sport players: associations with measures of aerobic fitness, metabolic control and locomotor function. *International Journal of Sports Medicine*, 33(3), 230-239. doi: 10.1055/s-0031-1291364
 7. Buchheit, M., Abbiss, C. R., Peiffer, J. J., & Laursen, P. B. (2012). Performance and physiological responses during a sprint interval training session: relationships with muscle oxygenation and pulmonary oxygen uptake kinetics. *European Journal of Applied Physiology*, 112(2), 767-779. doi: 10.1007/s00421-011-2021-1
 8. Buchheit, M., & Ufland, U. (2011). Effect of endurance training on performance and muscle reoxygenation rate during repeated-sprint running. *European Journal of Applied Physiology*, 111(2), 293-301. doi: 10.1007/s00421-010-1654-9
 9. Buchheit, M., Ufland, P., Haydar, B., Laursen, P. B., & Ahmaidi, S. (2011). Reproducibility and sensitivity of muscle reoxygenation and oxygen uptake recovery kinetics following running exercise in the field. *Clinical Physiology and Functional Imaging*, 31(5), 337-346. doi: 10.1111/j.1475-097X.2011.01020.x
 10. Coaching and Sports Science Division of the United States Olympic Committee. (2004). *Wingate Anaerobic Test*. Retrieved from: https://educationalathletics.weebly.com/uploads/2/9/9/0/29907311/wingate_testing.pdf
 11. Craig, N. P., & Norton, K. I. (2001). Characteristics of track cycling. *Sports Medicine*, 31(7), 457-468. doi: 10.2165/00007256-200131070-00001
 12. Dale, J., & Glaister, M. (2018). Moderate-intensity oxygen uptake kinetics: is a mono-exponential function always appropriate to model the response? *Research Quarterly for Exercise and Sport*, 89(3), 309-321. doi: 10.1080/02701367.2018.1482399
 13. de Jong, J., van der Meijden, L., Hamby S., Suckow, S., Dodge, C., de Koning, J. J., & Foster, C. (2015). Pacing strategy in short cycling time trials. *International Journal of Sports Physiology and Performance*, 10(8), 1015-22. doi: 10.1123/ijsp.2014-009
 14. Driss, T., & Vandewalle H. (2013). The measurement of maximal (anaerobic) power output on a cycle ergometer: a critical review. *BioMed Research International*, 2013, 589361. doi: 10.1155/2013/589361
 15. Dupont, G., McCall, A., Prieur, F., Millet, G., & Berthoin, S. (2010). Faster oxygen uptake kinetics during recovery is related to better repeated sprinting ability. *European Journal of Applied Physiology*, 110(3), 627-634. doi: 10.1007/s00421-010-1494-7
 16. Dupont, G., Moalla, W., Matran, R., & Berthoin, S. (2007). Effect of short recovery intensities on the performance during two Wingate tests. *Medicine and Science in Sports and Exercise*, 39(7), 1170-1176. doi: 10.1249/mss.0b013e31804c9976
 17. Durnin, J. V. G. A., & Womersley, J. (1974). Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *The British Journal of Nutrition*, 32(1), 77-97. doi: 10.1079/bjn19740060
 18. Ferguson, H. A., Harnish, C., & Chase, J. G. (2021). Using field based data to model sprint track cycling performance. *Sports Medicine Open*, 7(1), 20. doi: 10.1186/s40798-021-00310-0
 19. Fitts, R. H. (2016). The role of acidosis in fatigue: pro perspective. *Medicine and Science in Sports and Exercise*, 48(11),

- 2335-2338. doi: 10.1249/MSS.0000000000001043
20. Gastein, P. B. (2001). Energy system interaction and relative contribution during maximal exercise. *Sports Medicine*, 31(10), 725-741. doi: 10.2165/00007256-200131100-00003
 21. Glaister, M., Pattison, J. R., Dancy, B., & McInnes, G. (2014). The influence of aerobic fitness on the recovery of peak power output. *European Journal of Applied Physiology*, 114(11), 2447-2454. doi: 10.1007/s00421-014-2968-9
 22. Harris, R. C., Edwards, R. H. T., Hultman, E., Nordesjö, L.-O., Ny Lind, B., & Salin, K. (1976). The time course of phosphorylcreatine resynthesis during recovery of the quadriceps muscle in man. *Pflügers Archiv: European Journal of Applied Physiology*, 367(2), 137-142. doi: 10.1007/BF00585149
 23. Hebestreit, H., Mimura, K.-I., & Bar-Or, O. (1993). Recovery of muscle power after high-intensity short-term exercise: comparing boys and men. *Journal of Applied Physiology*, 74(6), 2875-2880. doi: 10.1152/jappl.1993.74.6.2875
 24. Hermens, H. J., Freriks, B., Merletti, R., Hägg, G. G., Stegeman, D. F., Blok, J. ... Blok, W. (1999). *SENIAM 8: European recommendations for surface electromyography*. Enschede, Netherlands: Roessingh Research and Development.
 25. Hopkins, W. G., Marshall, S. W., Batterham, A. M., & Hanin, J. (2009). Progressive statistics for studies in sports medicine and exercise science. *Medicine and Science in Sports and Exercise*, 41(1), 3-13. doi: 10.1249/MSS.0b013e31818cb278
 26. Maia, R. (2021). *Cycling Track: Results book. The Tokyo Organising Committee of the Olympic and Paralympic Games*. Retrieved from: [Olympic World Library - Results books : Tokyo 2020 / The Tokyo Organising Committee of the Olympic and Paralympic Games - Detail \(olympics.com\)](https://olympicworldlibrary.com/Results-books/Tokyo-2020/The-Tokyo-Organising-Committee-of-the-Olympic-and-Paralympic-Games-Detail-olympics.com)
 27. Mann, T. N., Webster, C., Lamberts, R. P., & Lambert, M. I. (2014). Effect of exercise intensity on post-exercise oxygen consumption and heart rate recovery. *European Journal of Applied Physiology*, 114(9), 1809-1820. doi: 10.1007/s00421-014-2907-9
 28. McMahon, S., & Jenkins, D. (2002). Factors affecting the rate of phosphocreatine resynthesis following intense exercise. *Sports Medicine*, 32(12), 761-784. doi: 10.2165/00007256-200232120-00002
 29. McManus, C. J., Collison J., & Cooper C.E. (2018). Performance comparison of the MOXY and PortaMon near-infrared spectroscopy muscle oximeters at rest and during exercise. *Journal of Biomedical Optics*, 23(1), 1-14. doi: 10.1117/1.JBO.23.1.015007
 30. Mohr, M., Krstrup, P., Nielsen, J. J., Nybo, L., Rasmussen, M. K., Juel, C., & Bangsbo, J. (2007). Effect of two different intense training regimens on skeletal muscle ion transport proteins and fatigue development. *American Journal of Physiology. Regulatory, Integrative, and Comparative Physiology*, 292(4), R1594-1602. doi: 10.1152/ajpregu.00251.2006
 31. Nagasawa, T. (2013). Slower recovery rate of muscle oxygenation after sprint exercise in long-distance runners compared with that in sprinters and healthy controls. *Journal of Strength and Conditioning Research*, 27(12), 3360-3366. doi: 10.1519/JSC.0b013e3182908fcc
 32. Özyener, F., Rossiter, H. B., Ward, S. A., & Whipp, B. J. (2001). Influence of exercise intensity on the on- and off-transient kinetics of pulmonary oxygen uptake in humans. *The Journal of Physiology*, 533(3), 891-902. doi: 10.1111/j.1469-7793.2001.t01-1-00891.x
 33. Parsons, B. (2010). Resistance training for elite-level track cyclists. *Strength and Conditioning Journal*, 32(5), 63-68. doi: 10.1519/SSC.0b013e3181e97d97
 34. Pazin, N., Bozic, P., Bobana, B., Nedeljkovic, A., & Jaric, S. (2011). Optimum loading for maximizing muscle power output: the effect of training history. *European Journal of Applied Physiology*, 111(9), 2123-2130. doi: 10.1007/s00421-011-1840-4
 35. Robergs, R. A. (2019). Invited review: quantifying proton exchange from chemical reactions - implications for the biochemistry of metabolic acidosis. *Comparative Biochemistry and Physiology. Part A Molecular & Integrated Physiology*, 235, 29-45. doi: 10.1016/j.cbpa.2019.04.024
 36. Rossiter, H. B., Ward, S. A., Kowalchuk, J. M., Howe, F. A., Griffiths, J. R., &

- Whipp, B. J. (2002). Dynamic asymmetry of phosphocreatine concentration and O₂ uptake between the on- and off-transients of moderate- and high-intensity exercise in humans. *The Journal of Physiology*, 541(3), 991-1002. doi: 10.1113/jphysiol.2001.012910
37. Schabert, E. J., Hawley, J. A., Hopkins, W. G., & Blum, H. (1999). High reliability of performance of well-trained rowers on a rowing ergometer. *Journal of Sports Sciences*, 17(8), 627-632. doi: 10.1080/026404199365650
38. Ufland, P., Ahmaidi, S., & Buchheit, M. (2013). Repeated-sprint performance, locomotor profile and muscle oxygen uptake recovery: effect of training background. *International Journal of Sports Medicine*, 34(10), 924-930. doi: 10.1055/s-0033-1333719
39. Viera, P. (2016). *Results book: Rio 2016/Organising Committee for the Olympic and Paralympic Games in Rio in 2016*. Retrieved from: <https://library.olympic.org/Default/doc/SYRACUSE/165312/results-book-rio-2016-organising-committee-for-the-olympic-and-paralympic-games-in-rio-in-2016?lg=en-GB>
40. Walter, G., Vandenborne, K., McCully, K. K., & Leigh, J. S. (1997). Noninvasive measurement of phosphocreatine recovery kinetics in single human muscles. *American Journal of Physiology*, 272(2), 525-534. doi: 10.1152/ajpcell.1997.272.2.C525
41. Westerblad, H. (2016). Acidosis is not a significant cause of skeletal muscle fatigue. *Medicine and Science in Sports and Exercise*, 48(11), 2339-2342. doi: 10.1249/MSS.0000000000001044
42. Wittekind, A. L., Micklewright, D., & Beneke, R. (2011). Teleoanticipation in all-out short-duration cycling. *British Journal of Sports Medicine*, 45(2), 114-119. doi: 10.1136/bjism.2009.061580
43. Zabala, M., Peinado, A. B., Calderón, F. J., Sampedro, J., Castillo, M. J., & Benito, P. J. (2011). Bicarbonate ingestion has no ergogenic effect on consecutive all out sprint tests in BMX elite cyclists. *European Journal of Applied Physiology*, 111(12), 3127-3134. doi: 10.1007/s00421-011-1938-8