Original Article

Maximal sprints within the warm-up does not affect pacing or performance in a 10 km cycle time trial

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ABSTRACT

This study investigated the effects of the inclusion of a post activation potentiation (PAP) specific warm-up (WU) consisting of all-out sprints on 10-kilometre cycling time trial (10 km-TT) pacing and performance. Following familiarization, thirteen well-trained male participants performed two 10 km cycle laboratory time trials following warm-ups that included either four 8 seconds (s) maximal sprints, or a matched total work performed at a constant exercise intensity. Power output (PO), heart rate (HR), and ratings of perceived exertion (RPE) were measured throughout and blood lactate (BLa) 3 minutes post exercise. There were no significant differences in total performance time, PO in any 2 km segment, RPE, or post-exercise BLa between conditions. Some significant differences ($p < .05$) were observed in heart rate and cadence between 2 km segments. Addition of four all-out sprints to the WU did not improve 10 km time trial performance or alter pacing strategy displayed. This suggests that maximal sprinting in a warming up might not be an effective strategy to improve cycling time trial endurance performance.

Keywords: Post activation potentiation; Endurance performance; PAP.

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INTRODUCTION

Research findings show that the implementation of short burst of intense exercise prior to an event can temporarily increase subsequent physical performance (Tillin & Bishop, 2009; Robins, 2005; Sale, 2002; DeRenne, 2010; Boullosa et al., 2018). This may be due to 'post activation potentiation' (PAP), which is defined by Robins (2005 p. 453) as "the phenomenon by which acute muscle force is enhanced as a result of contractile history" (Robins, 2005). Research suggests that the PAP phenomenon is caused by two mechanisms: a peripheral mechanism related to increased myosin light chain phosphorylation and a neural mechanism related to an increased recruitment of higher order motor units (Tillin & Bishop, 2009; Robins, 2005; Sale, 2002; Lorenz, 2011; Iglesias-Soler et al., 2011). A PAP-effect has been found in both strength and endurance athletes (Sale, 2002; Boullosa & Tuimil, 2009) and can be integrated into a warmup (WU) routine to increase performance when required (Tillin & Bishop, 2009; Robins, 2005; Sale, 2002; DeRenne, 2010; Boullosa et al., 2018; Lorenz, 2011).

The net effect of a PAP protocol balances between fatigue and potentiation (Tillin & Bishop, 2009; Sale, 2002; Koziris, 2009). This means that attempting to induce PAP does not always lead to increased performance but only will improve performance at a specific period when potentiation is higher than fatigue. This stresses the importance of an optimized recovery time between the PAP stimulus and the actual performance task (Sale, 2002). The length of the recovery time depends on the volume and intensity of the conditioning activity and the strength and training status of the athletes (Tillin & Bishop, 2009; Wilson et al., 2013; Chiu et al., 2013; Seitz et al., 2016; Seitz et al., 2014). Well-trained and strong individuals can evoke a stronger PAP-effect while simultaneously recover more optimally (Wilson et al., 2013; Chiu et al., 2013; Seitz et al., 2016; Seitz et al., 2014). This suggests that lesser fit individuals might need adjustments regarding conditioning activity and recovery time (Seitz et al., 2014).

The existence of PAP and its positive effect on athletic performance has been well studied in strength sports but to a lesser extent in endurance sports (Sale, 2002; DeRenne, 2010; Moir et al., 2011; Esformes & Bampouras, 2013; Silva et al., 2014; Feros et al., 2012). To date only one PAP study has been conducted in a longer endurance sport (20 km cycle time trial) setting (Silva et al., 2014). This study showed a significant ($p < .05$) 6.1% reduction in time to completion in a self-paced cycling time trial without any changes in rate of perceived exertion (RPE), blood lactate (BLa) values or pacing strategy. However, the leg press method used to induce PAP in this study, may be difficult to apply at a cycling venue. Instead, an "on the bike" warm-up is simpler to implement and reflects the warm-ups cyclists already incorporate (British Cycling, 2018). Interestingly, recently a study was conducted that investigated the effects of a three 10-second cycling sprints at 70% of peak power with 30 seconds rest in between, on 4 km self-paced cycling time trial performance (Chorley & Lamb, 2017). Although not significant ($p > .05$), this protocol led in a mean reduction in time to completion of 1.7 seconds, an increase in mean power output of 5.1 watts and an increase in mean peak pedal power force of 5.7 Newton (N). The highest increases were seen in the first 1500-meter of the race.

Based on the results of previous studies (Silva et al., 2014; Chorley & Lamb, 2017), the limited knowledge regarding the effect of PAP on endurance performance, and the practical application of cycling sprinting during a WU, new research regarding this topic is justified. The aim of this study was therefore to investigate the effects of a series of maximal all-out sprints, used in an attempt to induce a PAP effect, on self-paced 10 km time trial performance and associated physiological and perceptual responses. Based on previous studies (Silva et al., 2014; Chorley & Lamb, 2017) it was hypothesized that our PAP specific WU would improve cycling performance, especially in the earlier stage of a self-paced activity.

METHODS

Study design

The present study used a randomized design to measure the effects of repeated cycling sprints during a self-paced indoor 10-km ergometer cycling time trial. An ANOVA of repeated measurements was used to analyse the effects of the treatment protocol on power output (PO), time to completion, RPE, heart rate (HR), Pacing strategy and BLa.

Subjects

Thirteen well-trained males (age 40.4 ± 6.5 years, weight 79.2 ± 6.6 kg, height 180.5 ± 6.0 cm, BMI 24.3 ± 2.1, (critical power) CP 320.8 ± 35.9 watts) competitive cyclists with a minimum of two years of racing experience and who performed at least 4 training sessions per week were recruited from local cycling and triathlon clubs. All participants provided written informed consent before commencing any of the experimental procedures, which had received prior ethical approval at the University of Worcester (conform the declaration of Helsinki).

Procedures

Participants performed baseline testing and a familiarization trial then an experimental and a control 10 km laboratory time trials (10 km-TT) on different days, in a randomized manner and separated by at least 48 hours, on a dual-brake (air and magnetic) resisted cycle ergometer. The experiments were conducted in a climatologically controlled environment with temperatures kept between 19-20 °C. Participants were asked to prepare for the trials in the same way as for a minor competition by following their usual training and dietary routines.

Baseline tests and familiarization

To define the participants fitness levels a 3-minute all-out critical power test was performed at a Wattbike pro cycle ergometer (Clark et al., 2016). Critical power was calculated as the average wattage of the last 30-seconds of an all-out 3-minute effort (Clark et al., 2016). Height and weight were measured with a stadiometer. During the familiarization trials participants were familiarized with the standard warm up procedure, the maximal sprints and the 10 km time trial and the measurements taken.

Experimental trials

The standard WU prior to both treatment and control trial consisted of 5 minutes cycling at 100 watts and 5 minutes at 150 watts. After the standard WU the treatment group performed four-8 seconds all-out sprints at a fixed resistance (highest resistance) with 2 minutes of active rest consisting of easy spinning at an intensity of 50 watts between each sprint. After the sprints 10 minutes of rest was taken to let fatigue dissipate (Wilson et al., 2013; Kilduff et al., 2007; Seitz & Haff 2015). The 10 km-TT was self-paced and the participants could alter freely during the trial. In the control trials, the sprints were replaced by an extension of the WU with 6:30 minutes at a power output (PO) equivalent to the mean power in the sprints and active rest performed in the familiarization trial combined. This made overall energetic cost of the control WU similar to the treatment WU. Heart Rate was measured continuously throughout the experimental trials. Fingertip capillary BLa was measured 3 minutes after the end of the 10 km-TT. Ratings of Perceived Exertion were recorded every 2 km during time trials using the Borg 20 Category Scale (Borg, 1982). RPE was 'anchored' by explaining that a maximal RPE should equate to a previous experience of complete exhaustion.

Equipment

A valid and reliable (Hopker et al., 2010) dual-brake (air and magnetic) resisted cycling ergometer (Wattbike-pro, Nottingham, UK) was used for all trials. Saddle and handlebars were adjusted to the participants normal riding position and clipless pedals fitted of the participants choice. Heart rate, power output, speed, distance and cadence were continuously measured throughout the warmup, sprints and time trial. After the test, data were imported into Wattbike Expert analysis software for further analysis. A fan was places at a distance of 40 cm in front of the bike to provide participants with additional ventilation when requested throughout all trials. Heart Rate, distance, speed and cadence were measured with a Garmin Edge 500 (Garmin, Southampton, UK) heart rate analyser. The heart rate belt was placed around the participant's chest and specific gel was used to improve the connection between the electrodes and the skin. The watch was placed on the bike in a position that was optimal for the researcher to note the HR values. Fingertip capillary BLa was measured with a handheld lactate analyser (Lactate Pro 2, Arkray Group, Kyoto, Japan). Height and weight were measured with a SECA 217 stadiometer and SECA 761 scale (Seca Chino, USA).

Statistical analysis

All data were analysed using Graphpad Prism 7 software. BLa differences in between conditions, were assessed using paired samples t-tests. Differences in time to completion, power output, cadence, RPE, and HR in each 2 km segment were assessed using a repeated-measures two-way ANOVA with Sidak's multiple comparison correction and multiplicity adjusted *P* values are reported. The pacing strategy was calculated as the difference between the mean power over the entire 10 km trial and the relative power in each 2 km segment. The mean power was taken as 100% and power during each 2 km segment is presented as a percentage of the mean. All data is presented as mean ± S.D., and statistical significance was accepted at $p < .05$. Cohen's *d* effect sizes were calculated and classified as trivial < 0.2, small < 0.6, moderate < 1.2, large < 2.0, very large > 2.0 (Hopkins et al., 2009). A post hoc Pearson *R* correlation analysis was conducted to detect a possible relationship between treatment effect and sprint performance. This analysis was performed by comparing the mean power output of the four 8-second sprints with the increase or decrease in performance (power output) for the time trial of the treatment group.

RESULTS

Performance

Table 1 shows the time to completion in both conditions. No significant differences ($p < .05$) and small to trivial effect sizes were found in time to completion and power output (not shown) in any individual 2 km segment between the control and treatment group. The treatment protocol had a small effect (*d = 0.25)* positive effect on cycling performance in the first 2 km segment however this time benefit was lost in the fourth 2 km segment (*d = -0.36)*.

Time in 2 km segments

Table 1. Time in the control and treatment group 10 km-TT by 2 km segments.

	Mean SD						
	Treatment	**Control**	**Mean diff**	**95% C.I. of diff**	**p**	**Cohens d**	**Class**
0-2 km	162 ± 7.6	164 ± 8.2	-2.20	-10.65 to 6.19	.96	.25	small
2-4 km	166 ± 5.9	167 ± 6.6	-.90	-9.27 to 7.58	.99	.16	trivial
4-6 km	168 ± 6.8	168 ± 8.5	-1.00	-9.42 to 7.42	.99	.00	no effect
6-8 km	167 ± 7.3	163 ± 14.9	4.20	-4.27 to 15.58	.66	-.36	small
8-10 km	163 ± 8.3	163 ± 16.6	-.30	-8.73 to 8.12	.99	.00	no effect

Figure 1 shows the individual response to the treatment in seconds. Seven participants were faster in the treatment trial and six were slower.

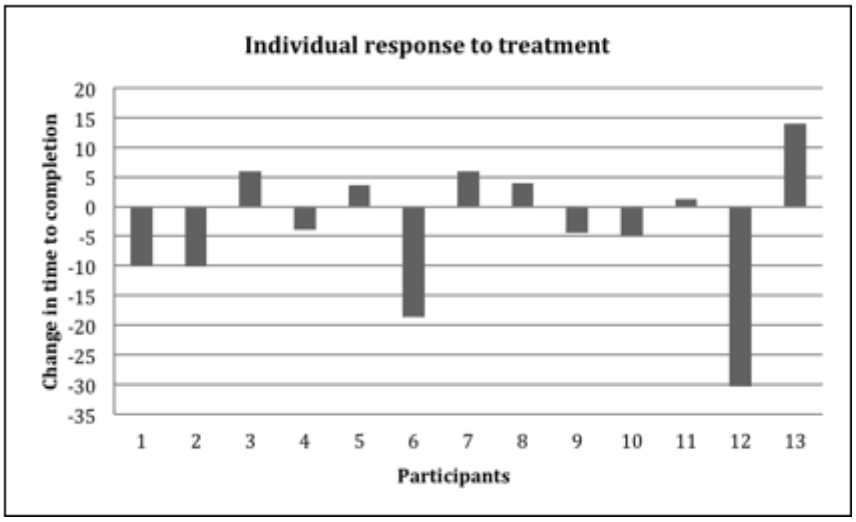

Figure 1. Time to completion for each individual participant in seconds faster or slower. Minus values are faster and plus values are slower than the control trial.

No significant correlation (p = .2573, R = .3457, R squared = .12) was found between sprint power and treatment effect (no figure shown).

Table 2 shows the cadence in both conditions. A significant* difference but small effect size in cadence between control and treatment condition was found in the first 2 km segment.

Cadence in 2 km segments

Table 2. Cadence in the control and treatment group 10 km-TT by 2 km segments and standard deviation.

	Mean SD						
Segments	**Treatment**	**Control**	**Mean diff**	**95% C.I. of diff**	**p**	**Cohens *d***	**Class**
0-2 km	103.7 ± 5.4	102.4 ± 4.7	1.31	0.2 to 2.4	*.01	0.26	small
2-4 km	101.1 ± 4.2	100.8 ± 3.6	0.39	-0.7 to 1.5	.89	0.08	trivial
4-6 km	100.1 ± 3.9	99.6 ± 3.7	0.51	-0.6 to 1.6	.71	0.13	trivial
6-8 km	99.8 ± 3.8	99.3 ± 4.1	0.49	-0.6 to 1.6	.75	0.12	trivial
8-10 km	101.3 ± 4.5	101.8 ± 4.3	-0.48	-1.6 to 0.6	.77	-0.11	trivial

** Statistically significant difference between trials.*
** Sig diff alpha < .05.*

Pacing

Table 3 and Figure 2 show the pacing strategy in both conditions. No significant difference in power output relative to trial average was found during any 2 km segment. A small positive and small negative effect size was detected in respectively the first and third 2 km segments.

Pacing in 2 km segments

Table 3. Pacing in the control and treatment group 10 km-TT by 2 km segments.

	Mean SD						
	Treatment	**Control**	**Mean diff**	**95% C.I. of diff**	**p**	**Cohens d**	**Class**
0-2 km	6.5 ± 11.7	3.7 ± 11.0	2.8	-2.5 to 8.1	.60	0.25	small
2-4 km	-2.1 ± 2.5	-2.2 ± 3.2	0.1	-5.2 to 5.4	1.00	0.04	trivial
4-6 km	-4.5 ± 3.0	-1.6 ± 9.1	-2.9	-8.2 to 2.4	.56	-0.48	small
6-8 km	-3.4 ± 5.8	-4.1 ± 9.4	0.7	-4.6 to 6.0	1.00	0.09	trivial
8-10 km	4.6 ± 6.3	5.6 ± 7.4	-1.0	-6.3 to 4.3	1.00	-0.15	trivial

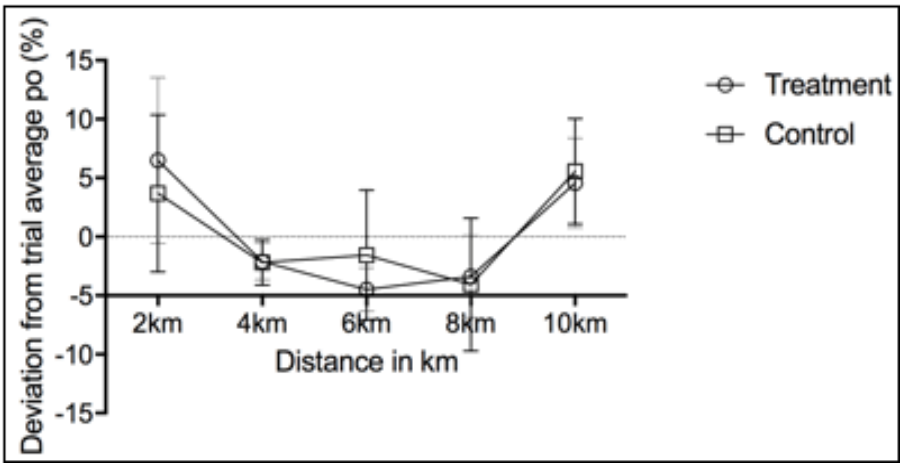

Figure 2. Pacing in the control and treatment group 10 km-TT by 2 km segments and confidence intervals.

Physiological and perceptual responses

Table 4 shows the heart rate in both conditions. A significant* difference in HR between control and treatment condition was found in the first, second and third 2 km segments accompanied with small effect sizes.

Heart rate in 2 km segments

Table 4. Heart rate in 2 km segments.

	Mean SD						
	Treatment	**Control**	**Mean diff**	**95% C.I. of diff**	**p**	**Cohens *d***	**Class**
0-2 km	147.9 ± 16.1	143.3 ± 14.8	4.54	2.4 to 6.6	*.0001	0.30	small
2-4 km	160.6 ± 13.8	157.0 ± 12.6	3.63	1.5 to 5.7	*.0001	0.27	small
4-6 km	164.4 ± 11.4	161.7 ± 11.5	2.70	0.6 to 4.8	*.0061	0.24	small
6-8 km	167.2 ± 11.2	165.6 ± 11.2	1.66	-0.4 to 3.8	.18	0.14	trivial
8-10 km	171.3 ± 11.8	170.3 ± 11.1	0.95	-1.2 to 3.0	.74	0.09	trivial

** Sig diff alpha < .05.*

Table 5 shows the rating of perceived exertion in both conditions. No significant difference in RPE was found between control and treatment groups in any 2 km segment.

RPE in 2 km segments

Table 5. Mean RPE by segments.

	Mean SD						
	Treatment	**Control**	**Mean diff**	**95% C.I. of diff**	**p**	**Cohens *d***	**Class**
2 km	15.1 ± 1.6	14.9 ± 1.5	0.15	-0.3 to 0.6	.92	0.13	trivial
4 km	16.2 ± 1.6	16.1 ± 1.4	0.08	-0.4 to 0.6	1.00	0.07	trivial
6 km	16.8 ± 1.2	16.6 ± 1.1	0.15	-0.3 to 0.6	.92	0.17	trivial
8 km	17.4 ± 1.1	17.5 ± 0.9	-0.15	-0.6 to 0.3	.92	-0.10	trivial
10 km	18.7 ± 1.1	18.8 ± 1.0	-0.15	-0.6 to 0.3	.92	-0.10	trivial

There was no difference in overall mean post-exercise BLa between treatment (12.1 ± 7.9 – 17.1 Mmol) and Control (12.3 ± 8.3 – 17.6 Mmol) trials (p = .7938, 95% C.I. = - 2.25 – 1.76 Mmol) Cohen's d = 0.08 (figure not shown).

DISCUSSION

Performance measures

The objective of this study was to investigate the effect of repetitive 8-second all-out cycling sprints on 10 km-TT cycling performance. Based on the results it can be concluded that the treatment did not lead to any significant change in performance. The treatment protocol had non-significant but positive effect on cycling performance in segment one, two, three and five. However, this improved performance was lost in segment four. The performance increases or decreases were accompanied with small to trivial or no effect sizes. The reason for the sudden decrease in segment four can't be explained. Individual data shows that seven participants were faster during the treatment trial but six were slower. Given the positive relation between muscular strength and the beneficial effects of a PAP protocol (Wilson et al., 2013; Chiu et al., 2013; Seitz et al., 2016; Seitz et al., 2014), a post hoc correlation analysis was conducted. This analysis however did not reveal a significant relationship between sprint power and treatment effect and thus fails to explain the differences in individual response to our protocol.

The results of our study differ from those of Silva et al., (2014) who found a 6.1% reduction in time to completion in a 20-kilometer cycling time trial. This might be related to differences in muscle activation in both studies. Although in our study there was no performance decrease and no differences in perceptual responses, some research suggest that the concentric contractions during the cycling sprints might negatively affected the Ca+ coupling process and potentially could have induced too much fatigue without adequate potentiation while the combination of concentric and eccentric contractions might lead to more muscle activation (Pasquet et al., 2000; Escamilla et al. 2001). Additionally, a lesser range of motion and a lack of motivation or guidance towards an optimal intensity could have led to lack of potentiation in our study (Esformes & Bampouras, 2013).

The study by Chorly and Lamb, (2017) used a WU protocol comparable to the WU protocol used in our study. However, where the protocol in our study did not lead to any differences between conditions, their protocol did lead to a (non-significant) performance increase. An important reason for this different might lay in the sprint intensity. In their study, Chorly and Lamb, (2017) used an intensity of 70% of the participants peak power output based on an all-out 6-second sprint, which was implemented three times with a 30-second rest in between. On the contrary, our protocol consisted of four maximal 8-second sprints, with two minutes active rest in between. It might be that the sprints in our protocol were too intense. Interestingly, some research

concluded that the maximal sprints could lead to excessive fatigue. In a study by McIntyre, (2007) all-out sprints led to a significant reduction in 3-kilometer time trial performance, while lower intensity sprints and a self-selected WU led to better performances. This is in line with Burnley et al., (2005) who concluded that a WU of moderate and heavy (but sub-maximal) intensities could improve cycling short time trial performance. Since less intense sprints lead to less fatigue it might be possible that submaximal sprinting would have led to a better balance between fatigue and potentiation and better performances in the 10 km time trial.

The recovery time between treatments and time trial (10 minutes) were the same in our study and the study by Silva et al., (2014). The study of Chorly and Lamb, (2017) however, used a passive rest of 5 minutes. It could be argued that perhaps the 10 minutes active rest in our study was too long and potentiation already disappeared. On the contrary, in the study by McIntyre, (2007) repeated all out sprints with a rest of only 5 minutes, negatively affected 3-kilometer time trial performance. The recovery time between the sprints and commencement of the trial utilized in our study was in line with recommendations of 8-12 minutes following inducement of PAP (Kilduff et al., 2007; Gouvea et al., 2013). Based on the principles of PAP it's plausible to assume that the lack off effect in our treatment protocol might be the result of an imbalance between potentiation and fatigue. Future studies should investigate the optimal balance between potentiation and fatigue induced by cycling sprints by measuring potentiation and fatigue directly and not only its effect on performance.

Pacing strategy

Pacing was not significantly different between the control and treatment group. Interestingly, the study by Silva et al., (2014) also showed no effect on pacing whilst they did show an improved performance. On the contrary, in a 1 km rowing PAP study by Feros et al., (2012) pacing was significantly different in the treatment group, which suggests that pacing might be affected by PAP only in shorter time trials.

Physiological measures

Both cadence (0-2 km segment) and HR (0-2 km, 2-4 km and 4-6 km segments) were significantly different between the two trials with a higher cadence and HR in the treatment group. Higher cadence may be the result of compensational mechanisms due to a loss of maximal strength caused by the sprints (Bieuzen et al., 2007) and to minimize muscular work and perception of effort (Ansley & Cangley 2009). Higher HR could be attributed to elevated post exercise oxygen consumption caused by the cycling sprints (Lavorgia et al., 2006). However, fatigue and oxygen uptake were not measured in our study. While plausible, the above-mentioned explanations can't be proven. In agreement with other studies (Silva et al., 2014; McIntyre, 2007), BLa and RPE did not significantly differ in any individual 2 km segment between control and treatment trials. Given that overall performance and pacing did not differ between trials, this finding may be expected.

Limitation of our study

In our study we did not measure fatigue and potentiation directly. Direct knowledge regarding the magnitude of fatigue and potentiation could possibly explain the lack of effect in our protocol. Interestingly, Seitz et al., (2013) shows that recovery times should be adapted to individual strength, which could have affected outcomes in both stronger and weaker participants. An individualized protocol might lead to a better balance between fatigue and potentiation and subsequent performance. Thus (for some participants) the recovery time might have been too long or too short. Such an individualized approach to a PAP specific WU design is an important element to be explored in further studies.

CONCLUSION

Our research indicates that inclusion of brief maximal all out sprinting might not provide any advantage over and above that provided by a lower intensity warm-up. Future studies should focus on the determination of the optimal intensity and rest periods of cycling warm-ups or investigate the implementation of other practical ways to potentiate cycling performance.

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