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**THE EFFECTS OF EXERCISE-INDUCED
MUSCLE DAMAGE ON PACING STRATEGY
DURING TIME-TRIAL PERFORMANCE**

by

Richard Bott

A Research Project submitted in partial fulfilment of the requirements of the University of Chester for the degree of M.Sc. Sports Sciences (Physiology Pathway)

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Abstract

Previous research has suggested that resistance training can improve endurance performance by improving muscle strength, power and speed whilst maintaining endurance capacity. However, a consequence of unaccustomed resistance training is the prolonged appearance of exercise induced muscle damage. This study examined the effects of a single bout of resistance exercise designed to elicit muscle damage on cycling performance when pacing in the first 60 seconds of a 5-minute time-trial, which was either controlled by the experimenter or self-paced by the individual. Seventeen cyclists/triathletes were randomly assigned to either a paced group (n= 8) or a self-paced group (n=9). Measurements of perceived muscle soreness, peak isokinetic strength and Creatine kinase were recorded as markers of muscle damage taken before and 48 h after muscle damage protocol. Measurements of $\dot{V}O_2$, heart rate, rate of perceived exertion and power output were recorded throughout the 5-minute time-trial, conducted before and 48 h after muscle damage. Total distance covered, peak isokinetic torque, blood lactate were reduced for both groups as well as no significant differences were seen between the groups for $\dot{V}O_2$, heart rate, RPE and power output, during time-trial, 48 h after muscle damage protocol ($P>0.05$). A trend is seen whereby participants in the paced group are able to match the performance during the first minute as that of their baseline measurement. Participants in the paced group then continue to cycle with a lower decrement in power output compared to those in the self- paced group. It could be speculated that a reduced central drive is the mechanism for a downward regulation of power output seen in the self-paced group and not

peripheral factors affecting performance as the paced group are able to maintain a similar peak power output following muscle damage protocol to baseline values.

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1. Introduction

Endurance cyclists are often required to sustain attacks, climb mountains, or sprint in the final part of a competitive road race (Tanaka & Swenson, 1998). Therefore, an increasing number of cyclists participate in resistance training alongside their traditional endurance training (Jung, 2003; Yamamoto, Lopez, Klau, Casa, Kraemer, & Maresh, 2008). Previous research has indicated that resistance training may also be beneficial to track cyclists competing in shorter endurance events lasting up to 5 minutes, where increases in muscle strength, power and speed are seen whilst maintaining endurance capacity (Levin, McGuigan & Laursen, 2009).

A consequence of resistance exercise, particularly when it is unaccustomed, is the immediate and prolonged appearance of symptoms associated with exercise-induced muscle damage (EIMD; Byrne & Eston, 2002; Twist & Eston, 2005). Such symptoms include increases in muscle soreness, swelling - as a result of increased oedema in the active muscle, elevated muscle proteins in the blood, (e.g. Creatine Kinase), impaired muscle function, and reduced neuromuscular control, that might impair exercise performance in the days after (Byrne, Twist, & Eston, 2004).

However, the influence of EIMD on sub-maximal exercise remains unclear. Elevations in oxygen consumption ($\dot{V}O_2$), minute ventilation (\dot{V}_E), respiratory exchange ratio (RER), blood lactate concentrations and heart rate have been

reported during sub-maximal running (Braun & Dutton, 2003; Chen, Nosaka, Tu, 2007; Burt, Lamb, Nicholas & Twist, 2013), probably as a consequence of changes in gait due to increased muscle soreness. However, such changes are less consistent for sub-maximal cycling, where $\dot{V}O_2$, RER and heart rate appear to remain unchanged after damaging exercise despite increases in \dot{V}_E and RPE (Davies, Rowlands, & Eston, 2009; Twist & Eston, 2009). Such inconsistencies in the physiological alterations after EIMD may be due to different damage protocols being used and studies failing to indicate whether a loss of muscle function was evident (Twist & Eston, 2009).

Twist and Eston (2005) investigated the effects of EIMD on maximal intensity intermittent exercise performance. After the muscle damage protocol, increased CK concentrations and increased perceived muscle soreness were reported at 24, 48 and 72 h. A reduced peak power output during a cycle sprint test was also noted at 24, 48, and 72 h but was lowest after 48 h. This change was consistent with peak soreness as well as the lowest rate of fatigue. The reduction in peak power observed in a maximal cycling sprint is thought to occur as a result of damage to type II muscle fibres after EIMD (Twist & Eston, 2009). As a result there is an increased recruitment of type I fibres that are less capable of producing large amounts of force than type II muscle fibres but are less susceptible to fatigue. Therefore impaired type II muscle fibre recruitment as a result of muscle-damaging exercise might have implications for competitive endurance cycling that involves repeated attacks and periods of sustained high intensity exercise.

Protocols that involve subjects exercising at submaximal work intensity to volitional exhaustion used in the assessment on endurance performance have been criticised for lacking ecological validity (Jeukendrup, Saris, Brouns & Kester, 1996). Simulated time-trials are therefore recommended as they replicate “real world” scenarios and provide a reliable measure of endurance performance (Schabert, Hawley, Hopkins, Mujika & Noakes, 1998).

Current research suggests that the cardiorespiratory responses to exercise are unchanged by exercise-induced muscle damage. However, time-trial performance is impaired because an increased sense of effort (higher RPE) resulting in individuals consciously down-regulating their exercise intensity when experiencing symptoms of muscle pain and weakness. Upon further analysis, studies have shown time-trial performance after EIMD results in lower peak and average power output and reductions in the total distance covered (Twist & Eston, 2009; Burt & Twist, 2011). However, in both Twist and Eston (2009) and Burt and Twist’s (2011) studies RPE remained unchanged during the time-trials despite reductions in \dot{V}_E , $\dot{V}O_2$, power output and blood lactate concentration. Such findings seem consistent with an altered sense of effort and a down-regulation of exercise intensity in the presence of EIMD (Marcora & Bosio, 2007; Twist and Eston, 2009). Further analysis also revealed that the largest decrement in power output occurred from the onset and during the first minute of the post EIMD time-trial.

One explanation for a downward regulation of power output during a cycling time-trial is a reduced central drive, as a result of force inhibiting neural mechanisms, which play a role in dynamic action protecting the muscle from

further damage (Burt & Twist, 2011). The mechanism for a reduction of a neural drive is thought to be due to the distension of group III and IV afferent fibres in and around the active muscle, which stimulate an increase in ventilation therefore increasing RPE, (Twist and Eston, 2009; Davies et al 2009). RPE is a recognised marker of intensity as well as identifying homeostatic disturbances during exercise (Morcora, 2009). RPE is moderated by both psychological factors (e.g. cognition, previous experience and understanding of the task) as well as situational factors (e.g. knowledge of the end point, duration and temporal changes of the task; Eston, 2012). Therefore, the central nervous system mechanism is responsible for the regulation of exercise performance during endurance exercise completed at maximal intensity (Swart, Lindsay, Lambert, Brown. &Noakes, 2012). Burt and Twist (2011) reported a downward regulation of power output which was seen from the onset of a 15 minute time-trial, which they postulated was be due to an increased anticipation of the effort required after EIMD. If it is perceived that an increase in effort is required after EIMD, it may pose a threat to the homeostatic control, which could potentially force the participant to reduce the exercise intensity from the onset (Swart et al., 2012). It has been suggested that over-predictions of exertion represent an effort to protect oneself when they are exposed to a potentially unpleasant level of exertion (Kilpatrick, Robertson, Powers, Mears, &Ferrer, 2008; Swart et al., 2012), and that participants responses to perceived exertion is higher than exertion responses taken during the onset of a time-trial. An applied example of where an increased RPE may affect training is if a track pursuit cyclist is undergoing concurrent training involving resistance training as well as high intensity cycling efforts. The quality

of the high intensity efforts may be impaired if the athlete is asked to conduct the session in a self-paced manor.

Research conducted within pain and muscle function can help bolster the argument that muscle function is centrally mediated rather than peripherally mediated.

Reports of an increased RPE for a given power output during exercise after EIMD therefore suggests that any reduction in muscle performance may be centrally rather than peripherally mediated. A tendency to over-predict pain, especially when the painful stimuli are novel, allows for protection from further danger or damage, (Kilpatrick et al., 2008; Eston, 2012). Khan, McNeil, Gandevia and Taylor, (2011) reported that when a participant is subjected to pain induced by an intramuscular injection of hypertonic saline, maximal voluntary muscular contractions are significantly reduced by 20 % but did not effect electronically evoked twitch torque. The authors suggested that the reduced voluntary torque was centrally mediated and not due to peripheral mechanisms.

When athletes engage in high-intensity exercise after muscle damaging exercise, the increased RPE as a result of increased muscle soreness may cause the individual to voluntarily down-regulate exercise intensity. Such actions are an attempt to protect the muscle from further damage and preserve muscle function within tolerable limits. What is unknown is whether external regulation in the initial stages of exercise when a participant is in a damaged state can override the typical response and alter the entire exercise

performance. Therefore the purpose of this study aims to examine the effects of a single bout of EIMD on cycling performance when pacing in the first 60 seconds of a 5 minutes time-trial which is either controlled by the experimenter (CP) or self-paced (SP) by the individual.

2. Methods

Participants

After ethical approval was approved by the Faculty of Applied Sciences Research Ethics Committee, 17 competitive trained cyclists/ triathletes (age 33.5 ± 8 years, stature 176.7 ± 6.9 cm, body mass 77.7 ± 8.2 kg, $\dot{V}O_{2\max}$ 56.2 ± 8.8 ml·kg⁻¹·min⁻¹) were invited to participate in the study. All participants recruited had not engaged in lower body resistance training within the last six months to limit the influence of prior exercise protecting the participant from muscle damage (Nosaka& Aoki, 2011). None of the participants took any form of medication during the study period. Participants were informed of the procedures and they were asked to complete a health screening questionnaire and gave written informed consent before commencement of the study. All participants were required to refrain from alcohol consumption, strenuous exercise, protein supplementation, compression clothing or ice baths 24 hours before each cycling exercise session.

Design

The study was a repeated measures, randomized control study with participants attending the laboratory on three separate occasions. During the first visit baseline measurements were recorded, comprising the assessment of maximal oxygen uptake ($\dot{V}O_{2\max}$) and lactate threshold (LT) measured from a single graded incremental cycling test to exhaustion. After a short rest, the participant then underwent a familiarization session to measurements for isokinetic strength and a 5-minute cycling time-trial (TT).

Participants returned to the laboratory between 48-96 hours later to perform baseline measurements of perceived muscle soreness (VAS), creatine kinase (CK), isokinetic muscle strength and a 5-minute TT. Immediately after participants then performed a bout of lower limb resistance exercise designed to elicit symptoms of exercise-induced muscle damage. Participants were then randomly allocated to either a paced or self-paced time-trial group. Forty- eight hours later, participants returned to the laboratory for the third and final visit where repeated measurements of VAS, CK, isokinetic strength and either a controlled-pace or self-paced 5-minute cycling TT were completed. A schematic of the study design is shown in figure 1.

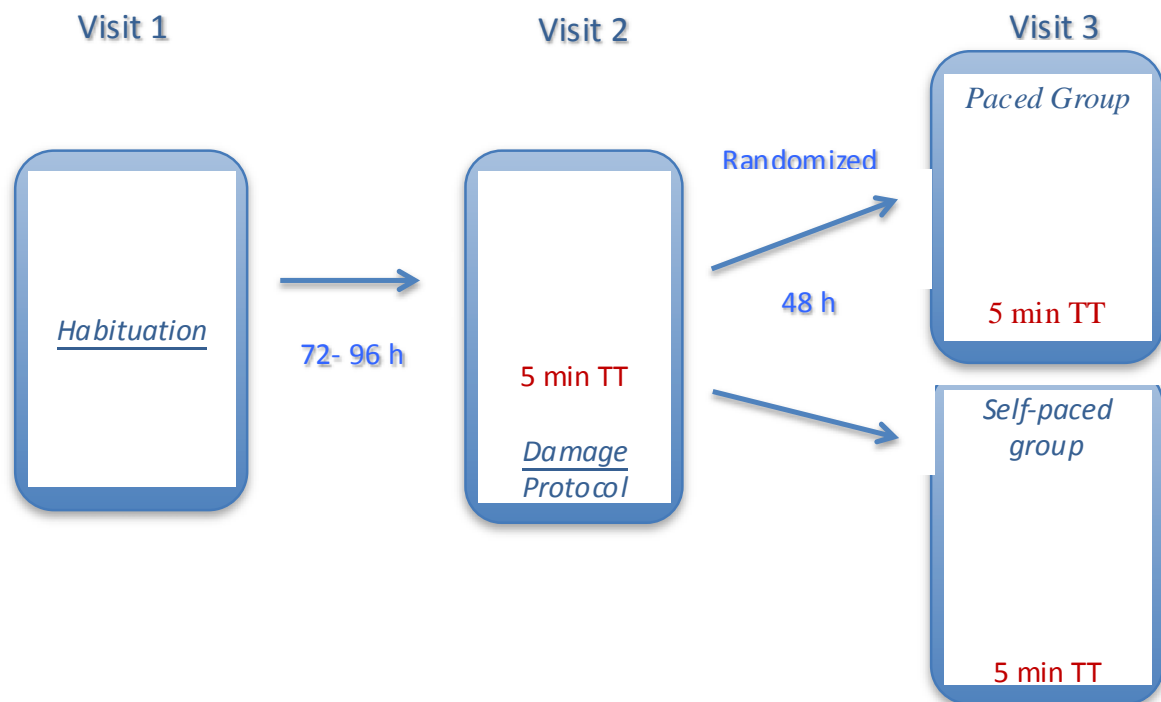


Figure 1: Schematic of study design

Assessment of lactate threshold and maximal oxygen uptake

Testing commenced at an initial workload of 150 W on an electronically braked ergometer (Lode Excalibur Sport, Lode Medical Technology, Groningen, Netherlands), with cycling intensity increasing by 30 W every 4 minutes until volitional exhaustion. Upon completion of each 4-minute stage, a fingertip capillary sample was obtained for blood lactate concentration (Arkay, Lactate Pro II, Arkay, Kyoto, Japan). Expired air and heart rate were measured continuously throughout the test using an online metabolic system (Oxycon Pro, Hoechberg, Germany). RPE was recorded at the end of the 4 minute stage. Individual LT was calculated as the workload at which the first rise in blood

lactate occurs above baseline values (reference). The $\dot{V}O_{2\max}$ was determined as the highest $\dot{V}O_2$ averaged over 30 seconds (Burt & Twist, 2011).

Isokinetic strength measurements

Participants performed a standardised warm-up of 5 minutes cycling at 100 W (Monark, 874E, Monark, Varberg, Sweden) followed by stretching of the knee extensor and flexor muscle groups. Peak isokinetic knee extensor and flexor torque was then measured on the participant's dominant limb (Biodex 3, Biodex Medical Systems, Shirley, NY, USA) at both slow (60 deg·s⁻¹) and fast (240 deg·s⁻¹) angular velocities. The dominant leg was fixed to the input arm of the dynamometer and the range of motion for each participant was manually determined by the investigator and limb mass was measured by the dynamometer to allow for gravitational correction of peak torque values. After 2 warm up trials, participants were required to perform 5 maximal efforts at each velocity with a 2 minute rest between each set. The highest torque achieved at each velocity was taken and used for analysis.

Perceived muscle soreness

Participants were asked to indicate their perceived muscle soreness for the knee extensors and flexors using a visual analogue scale (VAS). The sliding scale is numbered on the reverse, where 0 indicates no soreness on movement, and 10 indicates that the muscles are too sore to move. With hands on hips and knees shoulder width apart, participants were asked to squat to a knee joint angle of approximately 90° and then rate their perceived soreness on

the continuum. This technique has been used successfully in previous studies (e.g. Twist & Eston, 2007).

Creatine kinase (CK) measurement

Plasma CK activity was assessed from a fingertip capillary sample. A 30 μ l sample of whole fresh blood was analysed using a colorimetric assay procedure (Reflotron, Boehringer Mannheim, Germany).

Time-trial performance

Prior to muscle-damaging exercise, participants performed a 5-minute TT, covering as much distance as possible. Participants were given a 2-min warm up, during which they were required to maintain a fixed power output of 100 W. The participants then received a countdown for the start of the 5 minute TT. After the warm up, the ergometer was programmed to switch into linear mode, where the following formula was applied in accordance with the manufacturer's instructions: $W = L \times (\text{RPM})^2$ (where W = workload, L = linear factor, RPM = pedal revolutions per min). When in linear mode the ergometer acts as a mechanically braked ergometer enabling increases in work rate with increasing pedal cadence of which the alpha value was set at 0.42 (Burt & Twist, 2011). Power output, cadence (rpm), and $\dot{V}O_2$ were measured continuously throughout all time-trials and later averaged over 1) the 5-minute and 2) each 1-minute period. Peak power output was recorded and total distance covered (km) during the 5-minutes was also determined. HR was measured continuously throughout each time-trial whilst RPE was measured at random intervals within each

minute of the time-trial, in an attempt to limit any learning effect whereby the participant could use RPE as a cue as to the time left remaining altering pacing strategies. Blood lactate concentration from a fingertip capillary sample was recorded immediately and at 10 minutes after the time-trial. During time-trials, participants were not informed of the power output, or time left to complete the test, nevertheless verbal encouragement was given throughout.

After the muscle-damaging protocol, participants in the paced group cycled the first minute of the TT with the power output fixed to correspond to the power output attained during the 1st minute of their baseline time-trial. This was achieved by fixing either the flywheel resistance or the cycling cadence. The cycle ergometer was then programmed to switch into linear mode, as described above, and the remaining 4 minutes of the time-trial were self-paced by the participant. Alternatively, participants in the self-paced group performed the same time-trial procedures as before, i.e. baseline.

Muscle-damaging exercise protocol

To induce symptoms of exercise induced muscle damage, participants performed 10 sets of 10, Smith-machine squats at 80% of the individuals body mass, with 2-minute recovery between each set. Previous research has shown this technique to be successful to elicit symptoms of exercise-induced muscle damage (e.g. Davies et al., 2009).

Statistical analysis

Independent t-tests were performed on all baseline measures between the paced and self-paced groups to ensure no significant differences existed. Changes in perceived muscle soreness, isokinetic strength and CK after resistance exercise were analysed using separate paired-samples *t*-tests with Bonferroni correction to follow up any significant results.

Separate two-way (trial [2] x group [2]) ANOVA were used to assess changes in average power output, distance covered and blood lactate. To test for differences between the paced and self-paced groups, a three-way ANOVA (trial [2] x group [2] x time [2]) was used to assess changes in power output, RPE, $\dot{V}O_2$, heart rate. Assumptions of sphericity were assessed using the Mauchly test of sphericity, with any violations adjusted by use of the Greenhouse Geisser (G_G) correction. In all cases, the alpha level was set at $P < 0.05$.

3. Results

Independent *t*-tests on the physiological characteristics of participants indicated that no significant differences existed between the paced and self-paced groups ($p > 0.05$). This indicates that the recruitment and random allocation procedures were successful in forming two groups of similar performance standard.

Changes in indirect markers of exercise-induced muscle damage

Perceived muscle soreness

The results of the two way ANOVA on perceived muscle soreness after the muscle damage protocol, suggest that there was a significant main effect of time ($F_{1,30} = 17.969$, $P = 0.0001$). However there were no significant differences between the groups ($F_{1,30} = 1.38$, $P = 0.713$). Furthermore there was no interaction effect between time by group ($F_{1,30} = 0.001$, $P = 0.976$).

Peak isokinetic knee extensor torque

There was a significant difference on peak isokinetic torque, for time ($F_{1,30} = 4.983$, $P = 0.029$) after the muscle damage protocol. However no significant difference was seen between the two groups ($F_{1,30} = 0.898$, $P = 0.335$). Furthermore, there was no interaction effect between time by group ($F_{1,30} = 0.19$, $P = 0.892$).

Creatine Kinase

There was a significant difference for creatine kinase concentration, for time ($F_{1,30}=17.969$, $P=0.0001$) after the muscle damage protocol. However, no significant difference was seen between the two groups ($F_{1,30}= 0.138$, $P=0.713$). Furthermore, there was no interaction effect between time by group ($F_{1,30}=0.0001$ $P=0.976$).

Time-trial performance

A main effect for time indicated that total distanced covered during the time-trial was significantly lower at 48 h when compared to baseline or both groups ($F = 3.206$, $P < 0.05$). However, there was no significant interaction effect of time x group on peak power output ($F = 1.926$, $P > 0.05$) and post exercise blood lactate concentration ($F = 1.724$ $P > 0.05$).

Table 1. Changes in time-trial performances after muscle-damaging protocol for Paced and Self-paced groups.

	Paced Group		Self-paced Group	
	Baseline	48 h	Baseline	48 h
Distance covered (km)	2.73 ± 0.39	2.50 ± 0.45	2.75 ± 0.38	2.53 ± 0.39
Peak power output (W)	441.0 ± 55	439.3 ± 44.9	457.2 ± 109.4	415.7 ± 109.8
Average power output (W)	334.0 ± 40.1	327.3 ± 41.3	327.8 ± 40.8	308.0 ± 46.4
Blood lactate (mmol·L ⁻¹)	12.6 ± 4.0	12.3 ± 3.3	11.6 ± 2.5	9.1 ± 4.8

Values expressed as mean \pm standard deviation. No significant differences were seen between the groups ($P > 0.05$) for all measures.

Main effects

A significant difference was seen during each time point for Power output ($F_{1,15} = 2.207$, $P = 0.011$), heart rate ($F_{4,15} = 14.337$, $P = 0.0001$), RPE ($F_{4,12} = 75.856$, $P = 0.0001$) and $\dot{V}O_2$ ($F_{4,12} = 8.249$, $P = 0.002$). Likewise, there was a significant interaction between tests for power output ($F_{1,15} = 9.050$, $P = 0.009$, and trial by time for RPE ($F_{4,12} = 3.290$, $P = 0.049$). However, there were no significant effects of trial \times group on heart rate $\dot{V}O_2$ or RPE ($P > 0.05$). Likewise no significant difference was seen for trial by time by group effect was observed on power output, heart rate, RPE or $\dot{V}O_2$ ($P > 0.05$).

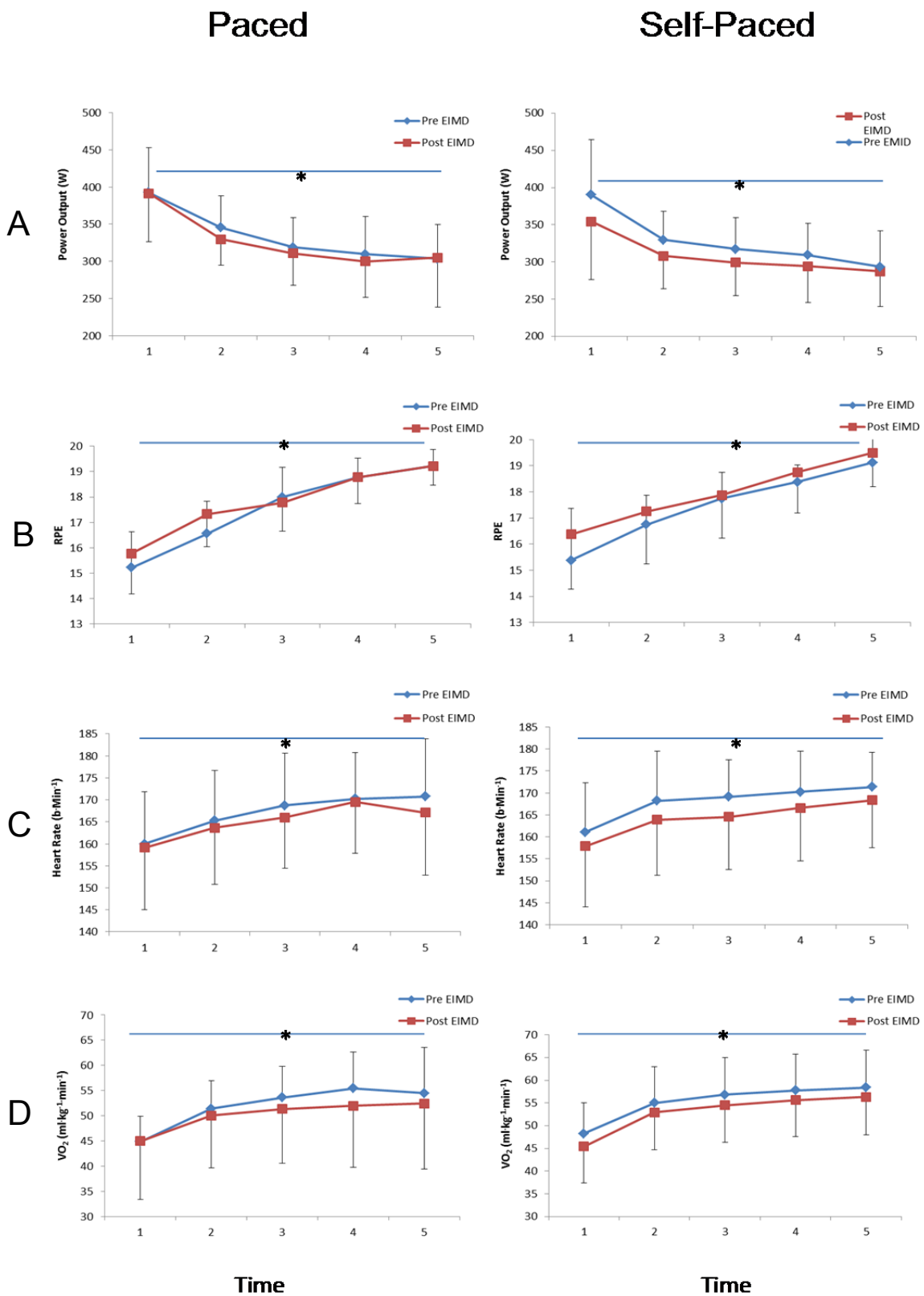


Figure 2. Changes in (A) power output, (B) RPE, (C) Heart rate, (D) $\dot{V}O_2$ at each minute interval during 5-minute time-trial for pre and 48 h post EIMD. *Significant main effect of time ($P < 0.05$)

4. Discussion

Increases were seen in perceived muscle soreness and a reduction of isokinetic peak torque at 48 h after a single bout of resistance exercise in both paced and self-paced groups. This demonstrated that the 10 x 10 squat protocol at 80 % of the participant's body weight was effective at inducing symptoms EIMD. These reported observations are consistent with those of previous studies that used the same muscle damaging protocol to elicit EIMD (Byrne & Eston, 2002; Davies et al., 2009). Plasma CK concentrations increased in both groups by 156.2 % of baseline at 48 h, indicating muscle fibre disturbance, following muscle damage protocol (Newham, Jones, & Edwards, 1983; Gleeson, Nlannin, Walsh, Field & Pritchard, 1998; Thompson, Nicholas & Williams, 1999; Byrne & Eston 2004).

No significant change was seen in $\dot{V}O_2$ during baseline and 48 h time-trials, however a trend was seen where there was an average decrease in $\dot{V}O_2$ of 3.5% and 4.2 % for paced and self-paced groups respectively throughout the 5-minute time-trial. It is postulated that this may be as a result of the groups not being able to match the intensity obtained during 48 h after performing the muscle damage protocol when compared to baseline measurements. Therefore $\dot{V}O_2$ was lower in both groups following EIMD, which, is in accordance with previous studies (Gleeson, Blannin, Zhu, Brooks & Cave, 1995; Scott, Rozenek, Russo, Crussemeyer&Lacourse, 2003;Paschalis, Koutedakis, Baltzopoulos, Mougious, Jamurtas, Theoharis, 2005; Davies et al., 2009).

Whilst not significant, the paced group showed a 5.4 % decrease in distance covered during 5-minute time-trial whereas, the self-paced group displayed a larger decrement in performance (8.1 %) following EIMD. The self-paced group exhibited a decrement of 5.9 % in average power output during the time-trial at 48 hours after EIMD whereas the paced group reported a 2 % decrement in average power output following the EIMD. There was a reduced decrement in blood lactate for the paced group (2.9 %) when compared to self-paced group (20.9 %), for the time-trial conducted 48 h after EIMD, implying that the overall intensity was lower in the self-paced group. These findings are in agreement with previous research, where a decrease in average power output after EIMD is seen due to an altered sense of effort (Byrne et al., 2004; Joseph et al., 2008; Burt & Twist 2011; St Claire Gibson & Noakes, 2004; Carson, Riek, Shahbazzpour, 2002; Proske, Weerakkody, Percival, Morgan, Gregory, Canny, 2003; Weerakkody, Percival, Morgan, Gregory, Proske, 2003), as a result of the central nervous system reducing the neural drive to peripheral muscles acting as a protective mechanism created by the subconscious part of the brain preventing any further muscle damage, St Clare Gibson & Noakes, 2004).

Results from the present study show a 9.1 % decrease in peak power output was seen for the self-paced group after EIMD. Twist and Eston (2009) also reported similar findings whereby there was an inability to generate a high peak power output after EIMD when self-paced. It has previously been suggested that EIMD may lead to the damage of type II muscle fibres (Friden, Sfikianos, Hargens & Akeson, 1983) and this may explain why some studies have shown that participants were unable to generate high peak power outputs. However, a smaller decrement was seen (0.4 %) in the paced group. This is because they

were forced to maintain a power output that was equivalent to that of baseline average power output, during the first minute of the time-trial at 48 h after EIMD. This finding may help support the argument that the reduction in power output seen after EIMD in this study was centrally governed, rather than being caused by any peripheral damage, thus altering the ability to reproduce the same power output. Consequently this may have led to an effect on altering the pacing strategy of that seen within the paced group at 48 h post EIMD.

Figure 1 illustrates that whilst the paced group were able to maintain the same power output during the first min, as that of the first minutes in the baseline time-trial, they then down regulate their power output for minutes 2-4, but are then able to increase the power output to match the same power output seen in the 5th minute as baseline. Previous studies (Twist & Eston, 2009; Burt and Twist, 2011) also noted an altered pacing strategy whereby the main alterations in pacing strategy after EIMD are seen at the outset of the time-trial, usually where the highest power output values are expected (Van IngenSchenau, de Koning& de Groot, 1992).

Figure 1 highlights that the self-paced group down regulated their power output by 9.2 ± 5.4 %, from the onset of the 5 minute time-trial, whereas the paced group maintained the same output as baseline. The self-paced group continued to down regulate their power output at each time point during the 5-minute time-trial adopting a very similar pacing strategy to that seen at baseline. This may be in response to an increased perceived muscle soreness sustained during the muscle damage protocol. Whilst not significant, a trend can be seen where the paced group were forced to maintain a similar power output/rpm during the first min of the 5-minute time-trial, which they completed in their baseline time-trial.

They were then able to complete the remaining 4 minutes of the time-trial which then was self-paced, at a smaller decrement (min 2 – 1.9 , min 3 – 3.1, min 4 – 1.7, min 5- 2.6 %) of power output at each time point compared to that of the self- paced group.

Figure 1 shows ratings of perceived exertion were higher in both groups from the onset of the 5 minute time-trial after EIMD when compared with baseline. However no significant difference was seen between the groups. Nevertheless the RPE was higher for a lower given power output and distance following EIMD in both groups. A trend can be seen, where RPE continues to be raised at each time point for the self-paced group whereas, the RPE is equal to or lower than that reported at baseline from minutes 3-5 respectively. Similar results were seen in previous studies (Twist & Eston, 2009; Burt & Twist, 2011) that found similar RPE values during time-trials before and 48 h after a single bout of EIMD. Studies that investigated EIMD on submaximal exercise also found an increase in RPE following muscle damage, (Scott et al, 2003; Davies et al, 2009). Burt & Twist (2011) postulated that this is due to the central nervous system reducing neural drive to the peripheral muscles during the time-trial after muscle damage which acts as a protective mechanism to prevent further injury. Furthermore, as a consequence of a reduction in power output, a reduction of cadence would have occurred. This is due to the time-trial being conducted using a linear mode on the ergometer, whereby the higher the cadence would mean the higher the power output. An increase in muscle soreness combined with a reduction in cadence may have resulted in an increase in the perception of effort. This is also in agreement with the studies of Deschenes, Kraemer,

McCoy, Voleck, Turner & Weinlein, 2000; and Burt & twist, (2011), who reported a higher RPE at slower pedalling cadences for the same metabolic cost.

5. Conclusion

This is the first study to evaluate the effects of muscle damage upon time-trial performance comparing paced versus self-paced groups. Whilst no significant effects were observed, a trend is seen whereby participants in the paced group are able to match the performance during the first minute to that of their baseline measurement. Participants in the paced group then continue to cycle with a lower decrement in power output compared to those in the self-paced group. It could be speculated that a reduced central drive is the mechanism for a downward regulation seen in the self-paced group not peripheral factors affecting performance as the paced group are able to maintain a similar peak power following muscle damage protocol to baseline values.

From a practical perspective, given the increase in popularity of endurance cyclists engaging in resistance exercise, coaches should be aware of the implications that exercise induced muscle damage can have upon self-paced cycling performance in the days after resistance training. If athletes are paced during periods of high intensity cycling, performance may be maintained.

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