REGULATION OF ENDURANCE PERFORMANCE: NEW FRONTIERS

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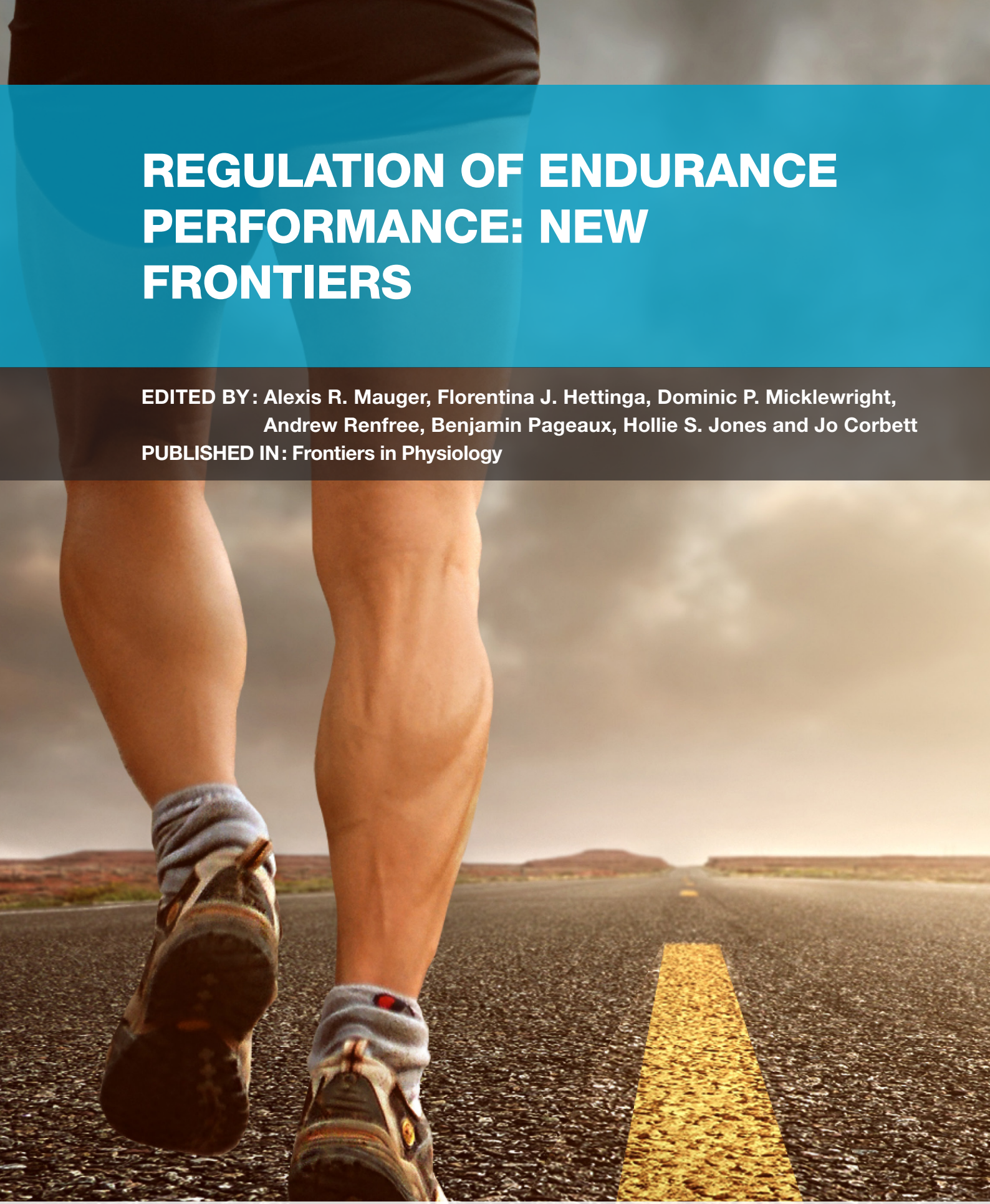

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REGULATION OF ENDURANCE PERFORMANCE: NEW FRONTIERS

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Regulation of endurance performance: new frontiers.

Successful endurance performance requires the integration of multiple physiological and psychological systems, working together to regulate exercise intensity in a way that will reduce time taken or increase work done. The systems that ultimately limit performance of the task are hotly contested, and may depend on a variety of factors including the type of task, the environment, external influences, training status of the individual and a host of psychological constructs.

These factors can be studied in isolation, or inclusively as a whole-body or integrative system. A reductionist approach has traditionally been favoured, leading to a greater understanding and emphasis on muscle and cardiovascular physiology, but the role of the brain and how this integrates multiple systems is gaining momentum. However, these differing approaches may have led to false dichotomy, and now with better understanding of both fields, there is a need to bring these perspectives together.

The divergent viewpoints of the limitations to human performance may have partly arisen because of the different exercise models studied. These can broadly be defined as open loop (where a fixed intensity is maintained until task disengagement), or closed loop (where a fixed distance is completed in the fastest time), which may involve whole-body or single-limb exercise. Closed loop exercise allows an analysis of how exercise intensity is self-regulated (i.e. pacing), and thus may better reflect the demands of competitive endurance performance. However, whilst this model can monitor changes in pacing, this is often at the expense of detecting subtle differences in the measured physiological or psychological variables of interest. Open loop exercise solves this issue, but is limited by its more restrictive exercise model. Nonetheless, much can be learnt from both experimental approaches when these constraints are recognised. Indeed, both models appear equally effective in examining changes in performance, and so the researcher should select the exercise model which can most appropriately test the study hypothesis. Given that a multitude of both internal (e.g. muscle fatigue, perception of effort, dietary intervention, pain etc.) and external (e.g. opponents, crowd presence, course topography, extrinsic reward etc.) factors likely contribute to exercise regulation and endurance performance, it may be that both models are required to gain a comprehensive understanding.

Consequently, this research topic seeks to bring together papers on endurance performance from a variety of paradigms and exercise models, with the overarching aim of comparing, examining and integrating their findings to better understand how exercise is regulated and how this may (or may not) limit performance.

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EDITORIAL
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Editorial: Regulation of Endurance Performance: New Frontiers

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Editorial on the Research Topic

Regulation of Endurance Performance: New Frontiers

INTRODUCTION

Successful endurance performance requires the integration of multiple physiological and psychological systems, working together to regulate exercise intensity in a way that will reduce time taken or increase work done. The systems that ultimately limit performance of the task are hotly contested, and may depend on a variety of factors including the type of task, the environment, external influences, training status of the individual and a host of psychological constructs. These factors can be studied in isolation, or inclusively as a whole-body or integrative system. A reductionist approach has traditionally been favored, leading to a greater understanding and emphasis on muscle and cardiovascular physiology, but the role of the brain and how this integrates multiple systems is gaining momentum. However, these differing approaches may have led to false dichotomy, and now with better understanding of both fields, there is a need to bring these perspectives together.

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Consequently, this research topic seeks to bring together papers on endurance performance from a variety of paradigms and exercise models, with the overarching aim of comparing, examining and integrating their findings to better understand how

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exercise is regulated and how this may (or may not) limit performance. To explore new frontiers, we welcomed the submission of original research, review and perspective articles on endurance performance, which specifically consider the scope and impact of their findings in the broader context of exercise regulation.

TOPIC CONTENT

This resulted in the acceptance of 24 papers (14 original research papers, 4 perspectives, 4 mini-reviews, a review, and an opinion) written by in total 84 contributing authors. Overall, the topic combines physiological with psychological viewpoints and papers explore closed-loop as well as open-loop exercise. Research papers from a predominantly physiological perspective were all directed toward a better understanding of endurance performance and its limitations, and/or directed toward optimizing endurance performance, and incorporated a wide range of methods.

FATIGUE AND RECOVERY

Fatigue and recovery were covered by several papers. VO_2 kinetics and recovery in intermittent exercise was explored by Barbosa et al. They found that endurance performance was negatively influenced by active recovery only during shorter high-intensity intermittent exercise, though probably unrelated to differences in VO_2 kinetics. Froyd et al. explored the critical fatigue threshold that has been proposed to limit endurance performance via inhibitory feedback from the group III and IV muscle afferents. They found that subjects did not terminate knee-extensor exercise at task failure because they had reached a critical threshold in peripheral fatigue and the existence of a critical peripheral fatigue threshold during intermittent isometric exercise to task failure with the knee extensors can thus be questioned. Also Neyroud et al. explored the critical fatigue threshold in their perspective article, highlighting the importance of considering interpretation of individual data and not only of group means. Muscle oxygenation, perceived fatigue and recovery were explored in speed skating by Hettinga et al. Patterns of reoxygenation and deoxygenation in the working muscles during a race are different for long-track and short-track speed skating, providing with more insights into the mechanistic physiological principles relevant for performance and recovery in elite athletes in different sports, and on how technical factors are impacting on those. Finally, Pageaux and Lepers explored mental and physical fatigue in their mini-review, and identified perception of effort as the variable altered by both prior physical exertion and mental exertion, that should be included in future studies.

THE ROLE OF THE BRAIN

Two experimental studies focused on the role of the brain in the regulation of exercise intensity. Hibbert et al. explored transcutaneous electrical nerve stimulation (TENS) effects on exercise-induced muscle pain, pacing strategy, and performance during a 5-km cycling time trial. Effects were found to be non-significant, and effectiveness of TENS could be questioned. There were indications that there was a possible effect at the start of the trials. Pires et al. explored cerebral regulation in different maximal aerobic exercise modes. Primary motor cortex activation was preserved throughout exercises, suggesting that central factors are at least partly centrally–coordinated. Angius et al. mini-reviewed the ergogenic effect of transcranial direct current stimulation on exercise performance, showing promising opportunities. However, also here it came forward that given the uncertain mechanisms and the inconsistency of outcomes of tDCS prior to exercise, the use of tDCS in exercise should be treated with some caution and future research is needed.

TRAINING PHYSIOLOGY OF ENDURANCE PERFORMANCE

Four experimental papers focused on training physiology of endurance performance. Schoenmakers et al. demonstrated that high intensity upper body interval training (HIIT) resulted in larger training effects compared to continuous training, and recommended to incorporate HIIT sessions in training regimes of recreationally active and trained handcyclists. De Araujo et al. discussed effects of HIIT they had found on hormones, metabolites, the anti-oxidant system, glycogen concentration and aerobic performance adaptations in rats into the training context of endurance runners. Guy et al. focused on effects of heat training on both endurance performance and biomarkers associated with inflammatory and immune system responses. Heat training enhanced performance and did not pose a substantial challenge to the immune system. Veldman et al. explored effects of neuromuscular electrical stimulation training on endurance performance, potentially particularly relevant for individuals with muscle weakness or patients who cannot perform voluntary contractions.

LIMITS OF HUMAN ENDURANCE PERFORMANCE: PHYSIOLOGY AND PERSONALITY TRAITS

Limits of human performance were addressed in a mini-review assessing the impact of age on physiological parameters, overviewing research on master athletes from Lepers and Stapley This paper strongly focused on physiological characteristics, where Schiphof-Godart et al. also included a psychological perspective to explore training behavior. They outline the possible influence of an athlete's passion in sport related to their exercise behavior and decision-making related to the regulation of exercise intensity. They conclude that taking into account athletes' passion could therefore be a useful tool for adequate coaching and monitoring of athlete well-being.

THE PSYCHOLOGICAL PERSPECTIVE: DECEPTION STUDIES AND IMPORTANCE OF THE ENVIRONMENT

From a psychological perspective, deception was a popular topic. Taylor and Smith demonstrated that mid-event pace deception can have a practically meaningful effect on multi-modal endurance performance, though the relative importance of different psychophysiological and emotional responses remains unclear. Williams et al. explored deceptive manipulation of competitive starting on several psychological and physiological parameters. Results demonstrated that with no detriment to performance time, but less physiological strain and more positive psychological perceptions, a pacing strategy adopting a slower start could be considered more beneficial during a stimulated 16.1 km cycling time trial. Jones et al. showed that time trial improvements were not sustained following acute provision of challenging and deceptive feedback. The presence of the pacer rather than the manipulation of performance beliefs acutely facilitated time trial performance and perceptual responses. This is in line with suggestions in the perspective of Hettinga et al., in which the science behind head-to-head competition was explored. They conclude that athlete–environment interactions are crucial factors in understanding the regulation of exercise intensity when racing against other competitors or pacers. Also Skorski et al. mention that environmental factors as important. In their mini-review, they conclude that pacing manipulations should be explored to further understand the complexity of how humans regulate pace.

When environmental factors are crucial, also the availability of feedback needs to be considered. Smits et al. examined the influence of the absence of commonly available task-related feedback on effort distribution and performance in experienced endurance athletes. They demonstrated that prior knowledge of task demands together with reliance on bodily and environmental information can be sufficient for experienced athletes to come to comparable time trial performances. In their meta-analysis, Davies et al. explored the effects of environmental feedback interventions on pacing. In line with the above studies, 26 cycling studies demonstrated environmental effects of hypoxia, thermal aspects and feedback on pacing and performance.

THE ROLE OF COGNITION IN PACING

Also, cognitive aspects in pacing were covered. Van Biesen et al. explored in their original research study if the regulation of exercise intensity during competitive track races was different between runners with and without intellectual impairment. Runners with intellectual impairment have difficulties to efficiently self-regulate their exercise intensity. Their limited cognitive resources may constrain the successful integration of appropriate pacing strategies during competitive races, and establishes the role of cognitive factors in pacing and the regulation of exercise intensity. Brick et al. provided a cognitive perspective on self-regulation and endurance performance in their perspective article. They highlighted the roles of attentional focus, cognitive control, and metacognition in self-regulated endurance performance and mental fatigue. Mental fatigue was further explored in the study of Head et al. focusing on exploring cognitive fatigue in an experimental study. The authors found a decreased Time-on-Task in bodyweight resistance training exercise tasks.

CONCLUSION

Recently, many researchers have focused on proposing frameworks to better understand the regulation of exercise intensity (Noakes, 1997; Marcora, 2008; Foster et al., 2009; Millet, 2011; Renfree et al., 2014; Smits et al., 2014; Hettinga et al.; Micklewright et al., 2017; St Clair Gibson et al., 2017; Venhorst et al., 2017). This research topic supports the notion that both internal and external variables need to be incorporated in frameworks exploring the regulation of exercise intensity. Both physiology and psychology are crucial for endurance performance, and aspects such as competitive environment, cognition and fatigue seem to be requisite to understand the regulation of exercise intensity. As yet, the way in which these factors interact in determining endurance performance is not fully understood.

The 24 papers comprising this research topic all explored the mechanisms involved in the regulation of exercise intensity. This issue was addressed within the context of a single bout of exercise, and across longer periods of time as is the case with long term changes in performance in masters athletes. As a whole, the papers have contributed to further understanding of fatigue & recovery, the role of the brain in regulatory processes, the relationship between physiological training responses and endurance performance, the limits of human performance and the influence of personality traits on endurance performance, and lastly the influence of environment, deception, and cognition on pacing. The number and range of issues considered within the broad subject of the regulation of exercise performance illustrates the complexity of the topic. Indeed, it is clear that (as stated in the introduction) a reductionist approach to understanding the regulatory process is unlikely to be sufficient. Although the papers comprising this research topic have greatly contributed to furthering our understanding of the key issues, it is still not clear how factors are "weighted" in terms of the extent to which they inform exercise regulation. Papers in this research topic alone have suggested that physiological status, psychological traits, and interactions with other competitors are all important. Researchers are encouraged to address the relative importance of these individual contributory factors in informing acute and chronic whole body behavior and performance during endurance exercise.

AUTHOR CONTRIBUTIONS

FH drafted and finalized the manuscript. ARM, AR, BP, and HJ significantly contributed to the drafts toward the final product and critically reviewed the manuscript. All authors (FH, ARM,

AR, BP, HJ, JC, and DM) provided valuable comments, thoughts and insights throughout the entire process of this topic that contributed to the final draft of this editorial, and approved the final version.

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ORIGINAL RESEARCH
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Endurance Performance during Severe-Intensity Intermittent Cycling: Effect of Exercise Duration and Recovery Type

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Slow component of oxygen uptake (VO_2SC) kinetics and maximal oxygen uptake (VO_2max) attainment seem to influence endurance performance during constant-work rate exercise (CWR) performed within the severe intensity domain. In this study, it was hypothesized that delaying the attainment of VO_2max by reducing the rates at which VO_2 increases with time (VO_2SC kinetics) would improve the endurance performance during severe-intensity intermittent exercise performed with different work:recovery duration and recovery type in active individuals. After the estimation of the parameters of the VO_2SC kinetics during CWR exercise, 18 males were divided into two groups (Passive and Active recovery) and performed at different days, two intermittent exercises to exhaustion (at 95% IVO_2max, with work: recovery ratio of 2:1) with the duration of the repetitions calculated from the onset of the exercise to the beginning of the VO_2SC (Short) or to the half duration of the VO_2SC (Long). The active recovery was performed at 50% IVO_2max. The endurance performance during intermittent exercises for the Passive (Short = 1523 ± 411; Long = 984 ± 260 s) and Active (Short = 902 ± 239; Long = 886 ± 254 s) groups was improved compared with CWR condition (Passive = 540 ± 116; Active = 489 ± 84 s). For Passive group, the endurance performance was significantly higher for Short than Long condition. However, no significant difference between Short and Long conditions was found for Active group. Additionally, the endurance performance during Short condition was higher for Passive than Active group. The VO_2SC kinetics was significantly increased for CWR (Passive = 0.16 ± 0.04; Active = 0.16 ± 0.04 L.min^{-2}) compared with Short (Passive = 0.01 ± 0.01; Active = 0.03 ± 0.04 L.min^{-2}) and Long (Passive = 0.02 ± 0.01; Active = 0.01 ± 0.01 L.min^{-2}) intermittent exercise conditions. No significant difference was found among the intermittent exercises. It can be concluded that the endurance performance is negatively influenced by active recovery only during shorter high-intensity intermittent exercise. Moreover, the improvement in endurance performance seems not be explained by differences in the VO_2SC kinetics, since its values were similar among all intermittent exercise conditions.

Keywords: aerobic, oxygen uptake, passive, active, exercise tolerance

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INTRODUCTION

The parameters of the power-time relationship, termed critical power (CP) and the curvature constant (W'), have been used to analyze the physiological responses and endurance performance during high-intensity exercise (Poole et al., 1988). CP has been considered the lower boundary of the severe-intensity domain and the W' determines the amount of external work that can be performed above CP, irrespective of the rate of its expenditure (Jones et al., 2010). By definition, all severe-intensity work rates (i.e., >CP) performed until voluntary exhaustion drive pulmonary oxygen uptake (VO_2) to a maximal value (i.e., maximal oxygen uptake—VO_2max) (Jones et al., 2010). However, during exhaustive exercise performed above the upper bound of the severe intensity domain, exercise duration would be too short to permit attainment of VO_2max Caputo and Denadai (2008). Several studies have demonstrated that endurance exercise performance within severe-intensity domain was coincident with the depletion of the W', accumulation of metabolites associated with fatigue (i.e., PCr, Pi, and H^+), and attainment of VO_2max due to VO_2 slow component (VO_2SC) development (Fukuba et al., 2003; Chidnok et al., 2013). Indeed, VO_2SC has been associated with loss in muscular efficiency (Jones et al., 2011) and has been negatively related with endurance performance (Zoladz et al., 1995; Murgatroyd et al., 2011; Barbosa et al., 2014a).

VO_2 kinetics and muscle [PCr] responses to high-intensity exercise have been reported to present both fundamental and slow component phases (Rossiter et al., 2002) being intrinsically linked. Indeed, Rossiter et al. (2002) have reported similar values of the time constant (τ) of the fundamental component ([PCr] = 38 s; VO_2 = 39 s), as well as the relative amplitude of the slow component ([PCr] = 13.9%; VO_2SC = 15.3%) of muscle [PCr] and VO_2 during high-intensity exercise. It has been proposed that progressive intramuscular depletion [PCr] during exhaustive exercise performed within severe intensity domain provides the appropriate stimulus to oxidative phosphorylation, determining the development of VO_2SC and, consequently, the attainment of VO_2max (Rossiter et al., 2002). Thus, both creatine phosphate depletion and development of the VO_2SC seem to be intimately associated with endurance performance during constant-work rate exercise (CWR) performed within the severe intensity domain.

While this scenario is well established during CWR exercise, very little information is available during intermittent exercise, which has been considered an important tool in training programs aiming to improve aerobic fitness in health and in disease (Laursen and Jenkins, 2002; Hwang et al., 2011). Indeed, intermittent exercise can improve performance comparing to CWR during high-intensity exercise (Millet et al., 2003; Chidnok et al., 2012), since the former allows resynthesis of intramuscular substrates ([PCr]) and/or clearance of fatigue-related metabolites (i.e., reconstitution of W') (Chidnok et al., 2013). However, several aspects seem to influence endurance performance during high-intensity intermittent exercises. For instance, endurance performance is progressively shorter when the work-recovery "duty-cycle" (e.g., 10:20 s, 30:60 s, 60:120 s, and 90:180 s) (Turner et al., 2006) and/or exercise intensity performed during active recovery is increased (i.e., light, moderate, heavy and severe) (Chidnok et al., 2012). These aspects influence PCr kinetics (Chidnok et al., 2013) and hypothetically, the changes of the rates at which VO_2 increases during high-intensity intermittent exercises (i.e., VO_2SC). Indeed, Chidnok et al. (2012) have demonstrated that enhanced endurance performance during severe-intensity intermittent exercise could be explained by the reconstitution of W' during recovery intervals performed at lower-intensity domains (i.e., light and moderate). At this condition, the reconstitution of W' was associated with a blunted increase in both VO_2 and integrated EMG with time, supporting the hypothesis that VO_2SC kinetics influences endurance performance during intermittent exercise. However, as discussed above, endurance performance during severe intermittent exercise is markedly modulated by both work-recovery duration and exercise intensity performed during active recovery. Thus, the possible relationship between VO_2SC and endurance performance during intermittent exercise performed with different durations (e.g., short vs. long) and recovery type (i.e., passive vs. active) remains elusive, and further studies are warranted.

However, an important issue must be considered when the possible influence of VO_2SC on endurance performance is investigated. Knowing that work-recovery duration influences endurance performance during severe intermittent exercise (Turner et al., 2006), it appears appropriate to compare exercise duration before (short condition) and after (long condition) the emergence of VO_2SC. However, many studies have verified that both the emergence and the amplitude of VO_2SC (and possibly the [PCr]) present a large intra-individual variation (Murgatroyd et al., 2011; Barbosa et al., 2014b). Thus, it would be interesting to analyze the responses of VO_2 kinetics and endurance performance during severe intermittent exercise, with both the duration of exercise and recovery periods being determined based on the individual VO_2SC kinetics response.

Thus, the current study was undertaken to compare the endurance performance and VO_2SC kinetics during high-intensity intermittent exercise performed with different work:recovery duration (short vs. long) and recovery types (passive vs. active) in active individuals. It was hypothesized that: (a) endurance performance would be improved during the exercise with passive recovery, regardless of the duration of the repetition, and; (b) endurance performance would be improved during the intermittent exercise with short duration, regardless of the recovery type. We also hypothesized that the possible interaction between exercise duration and recovery type during intermittent high intensity exercise would influence the changes to the rates at which VO_2 increases with time (VO_2SC kinetics) and consequently, endurance performance.

MATERIALS AND METHODS

Subjects

Eighteen male students (24.7 ± 4.1 years; 80.5 ± 12.5 kg; 178.1 ± 7.6 cm) that were physically active but did not participate in any regular physical exercise or sport program volunteered for

the study. All participants were healthy and free of cardiovascular, respiratory, and neuromuscular disease. All risks associated with the experimental procedures were explained prior to involvement in the study and each participant signed an informed consent form. The study was performed according to the Declaration of Helsinki and the protocol was approved by the University's Ethics Committee.

Experimental Design

The participants were instructed to report to the laboratory at the same time of the day (±2 h) on four separate occasions within a period of 2–3 week. Firstly, each volunteer performed an incremental test until exhaustion to determine the lactate threshold (LT), VO_2max and the intensity associated with VO_2max (IVO_2max). Thereafter, the volunteers were divided into two groups: passive recovery (PR) and active recovery (AR) with similar IVO_2max values. They performed the following protocols, on different days: (1) a total of two repetitions of square-wave transitions from rest to a power corresponding to 95% of the IVO_2max to determine the parameters of VO_2 kinetics. Each bout was separated by 60 min of passive rest. The VO_2 responses to the two severe exercise bouts were averaged before the analysis to reduce the breath-to-breath noise and enhance confidence in the parameters derived from the modeling process (Lamarra et al., 1987) and; (2) two intermittent exercises, with the duration of the repetitions calculated from the onset of the exercise to the beginning of the VO_2SC (Short) or to the half duration of the VO_2SC (Long). The interval between the experimental sessions was 48–72 h. The participants were instructed to arrive at the laboratory in a rested and fully hydrated state at least 3 h post-prandial. They were also asked not to perform any strenuous activity during the day before each test.

Procedures

Incremental Test

Each participant performed an incremental exercise test to obtain volitional fatigue on an electronically braked cycle ergometer (Excalibur sport, Groningen, Netherlands) to determine the participant's LT, VO_2max, and IVO_2max. The incremental protocol started at a power output of 35 W, with increasing increments of 35 W every 3 min. Previous studies have demonstrated no differences in VO_2max between incremental tests involving 1- or 3-min stage durations (Bentley and McNaughton, 2003; Roffey et al., 2007; Adami et al., 2013). The pedal cadence was kept constant (70 rpm) (Marsh and Martin, 1997). Throughout the tests, the respiratory and pulmonary gas-exchange variables were measured using a breath-by-breath gas analyzer (Quark PFTergo, Cosmed, Italy). The VO_2max was defined as the highest average 15-s VO_2 value recorded during the incremental test. IVO_2max was defined as the power output at which the VO_2max occurred. At the end of each stage, an earlobe capillary blood sample (25 μL) was collected into an eppendorf tube and analyzed for its lactate concentration ([La]) using an automated analyzer (YSI 2300 STAT, Yellow Spring, Ohio, USA). Plots of the blood [La] against the power output and VO_2 were given to two independent reviewers, who determined LT as the first sudden and sustained increase in the blood lactate level above the resting concentrations.

Constant-Workload Exercise

The participants performed two exercise transitions at 95% IVO_2max, separated by 60 min of rest. The first transition lasted 6 min and was conducted to determine the VO_2 kinetics. The second transition was conducted until voluntary exhaustion to determine the VO_2 kinetics (first 6 min) and the tlim (time to exhaustion). The protocol began with a 5 min warm-up at 50% IVO_2max and was followed by a 7 min of passive rest. Then, the participants performed 3 min of unloaded cycling at 20 W, followed by a step change in the power output to 95% IVO_2max. The pedal cadence was kept constant at 70 rpm. The second transition was terminated when the participant could not maintain a cadence of >65 rpm for >5 s despite verbal encouragement. The end-exercise VO_2 was defined as the mean VO_2 measured during the final 15 s of exercise. For the determination of [La] peak, capillary blood samples were collected 1, 3, and 5 min after the exercise, as previously described.

Intermittent Exercises

The intermittent exercises were performed at 95% IVO_2max, with the duration of the repetitions calculated from the onset of the exercise to the beginning of the VO_2SC (i.e., time delay before the onset of the development of the VO_2SC—Short) or the half duration of the VO_2SC (i.e., 50% of the difference between the Short work interval duration and the time to achieve VO_2max—Long) (**Figure 1**). The recovery was passive (PR) or active (AR) (50% IVO_2max), with duration corresponding to the half of the repetition (effort:recovery ratio of 2:1). The exercises were performed until voluntary exhaustion. The criterion of exhaustion used was the same used for the constant-workload exercise. The end-exercise VO_2 was defined as the mean VO_2 measured during the final 15 s of exercise. If the duration of the last repetition was shorter than 90 s, the highest value of the previous bout was considered, to avoid underestimating the VO_2 value.

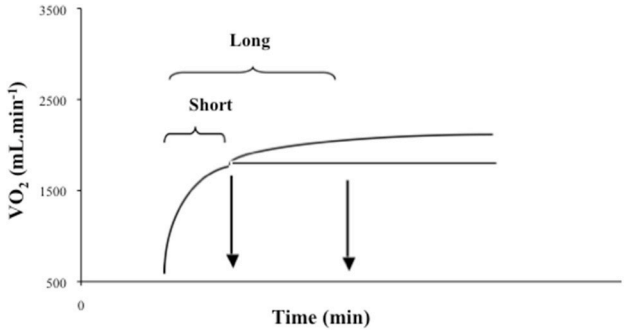

FIGURE 1 | Definition of the work intervals of the Short (beginning of the slow component) and Long (half duration of the slow component) intermittent protocols.

Modeling of VO_2 during Constant-Workload Exercise

The breath-by-breath data from each exercise were manually filtered to remove outlying breaths, which were defined as breaths ±3 SD from the adjacent five breaths. The breath-by-breath data were interpolated to give second-by-second values. For CWR, the two transitions were then time aligned to the start of the exercise and averaged to enhance the underlying response characteristics. The first 20 s of data after the onset of exercise (i.e., the phase I response) (Whipp and Rossiter, 2005) were deleted, and the biexponential model was used to analyze the VO_2 response to severe exercise, as described by the following equation:

$$VO_2(t) = VO_2baseline + Ap\,[1 - e^{-(t-TDp)/\tau p}] + As\,[1 - e^{-(t-TDs)/\tau s}] \quad (1)$$

where: $VO_2(t)$ is the absolute VO_2 at a given time t; VO_2baseline is the mean VO_2 in the baseline period; Ap, TDp, and τp are the amplitude, time delay, and time constant, respectively, describing the phase II increase in VO_2 above baseline; and As, TDs, and τs are the amplitude of, time delay before the onset of, and time constant describing the development of the VO_2SC, respectively. An iterative process was used to minimize the sum of the squared errors between the fitted function and the observed values. VO_2baseline was defined as the mean VO_2 measured over the final 60 s of exercise preceding the step transition to severe exercise. The amplitude of the VO_2SC was determined as the increase in VO_2 from TDs to the end of the modeled data (defined as As'). The end-exercise VO_2 was defined as the mean VO_2 measured over the final 15 s of exercise. The TD identified from Equation 1 was utilized to individualize the duration of the repetitions performed during short and long protocols (please see Section Intermittent exercises) and to estimate the VO_2SC kinetics [i.e., the slow component trajectory (L.min^{-2})], as described below.

In addition, a single-exponential model without time delay, with a fitting window commencing at $t = 0$ s (equivalent to the mean response time), was used to characterize the kinetics of the overall VO_2 response to exercise. The following equation describes this model:

$$VO_2(t) = VO_{2baseline} + A\,[1 - e^{-(t/\tau)}] \quad (2)$$

where: $VO_2(t)$ represents the absolute VO_2at a given time t, VO_2baseline represents the mean VO_2 measured over the final 60 s of baseline pedaling, and A and τ represent the amplitude and time constant, respectively, which describe the overall increase in VO_2 above the baseline. The VO_2 was assumed to have essentially reached its maximal value when the value of $[1–e^{-(t/\tau)}]$ from Equation 2 was 0.99 (i.e., when $t = 4.6 \times \tau$); it was assumed at this time that VO_2 was at its maximal value. Therefore, for each exercise, the time to achieve VO_2max ($TAVO_2$max) was defined as $4.6 \times \tau$. VO_2SC kinetics [i.e., the slow component trajectory (L.min^{-2})] was also estimated by calculating the slope of the VO_2 response using linear regression analysis (Chidnok et al., 2012). The data obtained before TDs (determined from Equation 1) were deleted to remove the influence of the fundamental response phase, and thereafter, VO_2 values at 60-s intervals were determined until reaching the $TAVO_2$max value and were fitted using the following equation:

$$VO_2 = ax + b \quad (3)$$

where: x represents the time, a represents the slope, and b represents the y-intercept.

Modeling of VO_2 during Intermittent Exercise

VO_2SC kinetics [i.e., the slow component trajectory (L.min^{-2})] was estimated by calculating the slope of VO_2 response using linear regression analysis (Chidnok et al., 2012). Final VO_2 values (i.e., the average VO_2 during 15 s) of each work cycle during intermittent exercise were determined up to the last completed cycle and fit using the Equation 3.

Statistical Analysis

The data are presented as means ± SD. The normality of data was checked by the Shapiro-Wilk test. A 2 × 3 two-way factorial analysis of variance (group vs. exercise condition), with repeated measures for the exercise condition factor (CWR vs. Short vs. Long) was used to analyze the VO_2, tlim, slope VO_2, [La] and HR data. When a significant interaction was found, follow-up analyses were performed using Tukey HSD test. The significance level was set at $p < 0.05$, and effect sizes were calculated using partial eta-squared (η^2). All analyses were completed using the Statistical Package for the Social Sciences (SPSS v.20.0, SPSS Inc., Chicago, IL, USA).

RESULTS

Table 1 presents the mean ± SD values of the variables obtained during the incremental test for both PR and AR groups. No significant difference was found between the groups ($p > 0.05$).

The VO_2 response profiles of a representative subject obtained during the different exercise conditions for both PR and AR groups are depicted in **Figure 2**. Based on the VO_2 kinetics parameters obtained during CWR, the repetition duration for the Short (PR = 105 ± 29 s; AR = 132 ± 39 s) and Long (PR = 252 ± 50 s; AR = 253 ± 56 s) tests were not significantly different between the groups ($p > 0.05$).

Figure 3 presents the mean ± SD values of end-exercise VO_2 measured during the different exercise conditions for both PR and AR groups. There was a significant main effect for the

TABLE 1 | Mean ± SD values of the variables obtained during the incremental test for both passive (PR) and active (AR) recovery groups.

	PR (*N* = 9)	AR (*N* = 9)
VO_2max (mL.min^{-1})	3220.4 ± 271.8	3332.4 ± 499.1
IVO_2max (W)	250.3 ± 25.5	266.9 ± 44.1
P95% (W)	235.7 ± 23.0	252.6 ± 42.7
LT (W)	106.0 ± 31.3	133.1 ± 59.0
LT (%IVO_2max)	41 ± 11	48 ± 16

VO_2max, maximal oxygen uptake; IVO_2max, intensity at VO_2max; P95%, power output relative to 95% IVO_2max; LT, lactate threshold.

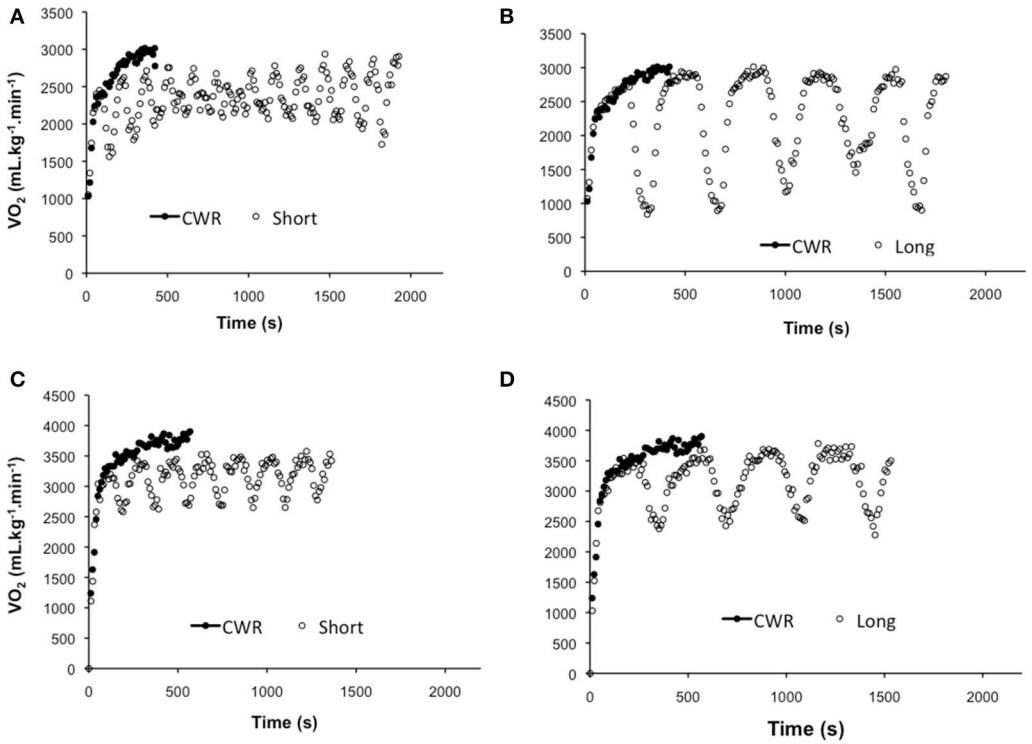

FIGURE 2 | Pulmonary oxygen uptake (VO_2) response of a representative subject to constant-work rate (CWR) exercise (closed circles) compared with intermittent exercise (open circles) performed with passive (**A**, short and **B**, long) and active (**C**, short and **D**, long) recovery.

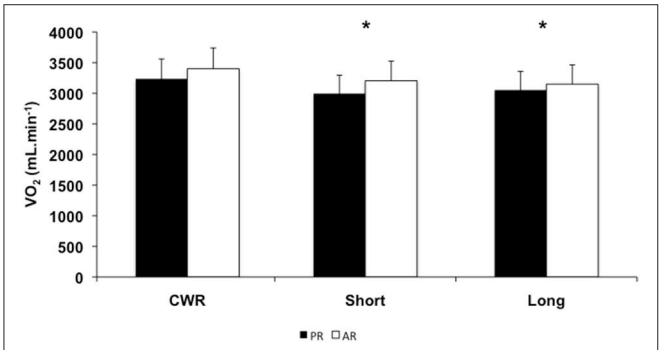

FIGURE 3 | Mean ± SD values of the end-exercise VO_2 obtained during the exercise performed in different conditions for passive (PR) (*N* = 9) and active (AR) (*N* = 9) groups. CWR—constant-work-rate exercise; *$p <$ 0.05 in relation to CWR.

exercise condition on end-exercise VO_2 values ($F = 5.47$, $p = 0.009$, $\eta^2 = 0.25$), but no effect of group ($F = 1.53$, $p = 0.23$, $\eta^2 = 0.08$) or interaction was detected ($F = 1.25$, $p = 0.29$, $\eta^2 = 0.07$). The end-exercise VO_2 values obtained during CWR (PR $= 3236.9 \pm 405.8$ mL.min^{-1}; AR $= 3488.6 \pm 415.9$ mL.min^{-1}) were higher than those attained during Short (PR $= 2995.2 \pm 337.7$ mL.min^{-1}; AR $= 3205.7 \pm 447.2$ mL.min^{-1}) and Long (PR $= 3053.3 \pm 276.1$ mL.min^{-1}; AR $= 3149.6 \pm 476.3$ mL.min^{-1}) tests ($p < 0.05$).

The mean ± SD values of tlim and VO_2 slope during CWR and intermittent exercises for the PR and AR groups are presented in **Table 2**. A group vs. exercise condition interaction ($F = 11.08$, $p = 0.000$, $\eta^2 = 0.40$) indicated longer tlim obtained during intermittent exercises (Short and Long) than CWR for both groups ($p < 0.05$). Considering the duration of the work and recovery type, tlim at Short was significantly longer than at Long only for the PR group ($p < 0.05$). Group effect (i.e., PR vs. AR) was significant only when comparing the Short intermittent protocols ($p < 0.05$), with no significant difference for Long conditions ($p > 0.05$). There was a significant main effect for the exercise condition on VO_2 slope values ($F = 95.98$, $p < 0.000$, $\eta^2 = 0.90$), but no group effect ($F = 1.86$, $p = 0.19$, $\eta^2 = 0.16$) or interaction was detected ($F = 0.02$, $p = 0.99$, $\eta^2 = 0.01$). VO_2 slope was significantly greater at CWR than Short and Long conditions ($p < 0.05$).

The mean ± SD values of [La] and HR during CWR and intermittent exercises for the PR and AR groups are presented in **Table 3**. There was a significant main effect for the exercise condition on [La] values ($F = 4.72$, $p = 0.01$, $\eta^2 = 0.22$), but no effect of group ($F = 0.05$, $p = 0.81$, $\eta^2 = 0.04$) or interaction was detected ($F = 1.76$ $p = 0.18$, $\eta^2 = 0.09$). The [La] was significantly lower at Short than CWR and Long condition ($p < 0.05$). A group *vs.* exercise condition interaction ($F = 5.00$, $p = 0.01$, $\eta^2 = 0.23$) indicated that HR was lower during Short than Long and CWR only for the PR group ($p < 0.05$).

TABLE 2 | Mean ± SD values of the time to exhaustion (tlim) and the slope of the oxygen uptake response (Slope) during the constant-work-rate (CWR) and intermittent exercise conditions (Short and Long), for passive (PR) and active (AR) recovery groups.

	PR ($N = 9$)			AR ($N = 9$)			Significance
	CWR	Short	Long	CWR	Short	Long	
tlim (s)	540	1523	984	489	902	886	$^{*}F = 11.08$
	116	411‡,†	260‡	84	239‡,**	254‡	$p = 0.000$
Slope (L.min^{-2})	0.16	0.01	0.02	0.16	0.03	0.01	$^{++}F = 5.34$
	0.04	0.01‡	0.01‡	0.04	0.04‡	0.01‡	$p = 0.01$

*Group vs. condition interaction; $^{‡}p < 0.05$ relative to the CWR condition; $^{†}p < 0.05$ relative to the Long condition; $^{**}p < 0.05$ relative to the Short condition; $^{++}$Main effect of exercise condition.

TABLE 3 | Mean ± SD values of the blood lactate concentration ([La]) and heart rate (HR) during the constant-work-rate (CWR) and intermittent exercise conditions (Short and Long), for passive (PR) and active (AR) recovery groups.

	PR			AR			Significance
	CWR	Short	Long	CWR	Short	Long	
[La] (mM)	12.4	10.3	12.1	11.2	10.9	11.8	$^{++}F = 4.72$
	2.83	3.70	3.02	2.35	2.65	2.78	$p = 0.01$
HR (bpm)	177 14	170 15†	177 11	184 7	183 6	186 5	$^{*}F = 5.00\ p = 0.01$

*Group vs. condition interaction; $^{†}p < 0.05$ relative to CWR and Long conditions; $^{++}$Main effect of exercise condition.

DISCUSSION

This, we believe, is the first study to compare the endurance performance and VO_2SC kinetics during severe-intensity intermittent exercise performed with different durations and recovery types in active individuals. The data demonstrate that endurance performance during severe-intensity intermittent exercise is negatively influenced by active recovery only during shorter (∼120 s) intermittent exercise. Interestingly, slopes describing the increases in VO_2 with time (i.e., VO_2SC) and end-exercise VO_2 were reduced during intermittent exercise (i.e., CWR vs. intermittent exercise). However, VO_2 kinetics (VO_2SC and end-exercise VO_2) were similar between work:recovery duration (short vs. long) and recovery type (passive vs. active) analyzed in the present study, therefore rejecting our original hypothesis. Thus, the relationship between VO_2 kinetics (VO_2SC and end-exercise VO_2) and endurance performance observed during CWR exercise (Jones et al., 2010; Barbosa et al., 2014a) seems to be differently regulated during severe-intensity intermittent exercise.

It has been widely reported that endurance performance during high-intensity intermittent exercise is improved when compared with CWR exercise (Demarie et al., 2000; Millet et al., 2003; Chidnok et al., 2012). However, both endurance performance and metabolic response are influenced by the characteristics of the protocol utilized during high-intensity intermittent exercise. Turner et al. (2006) analyzed the influence of duty cycle duration with the same work:recovery ratio (10:20 s, 30:60 s, 60:120 s, and 90:180 s) on pulmonary gas exchange and blood lactate dynamics during intermittent cycling exercise performed at 120% IVO_2max. At this condition, a greater metabolic response (elevated blood lactate concentration and attainment of VO_2max) and exercise intolerance (i.e., subjects could not complete 30 min of exercise) were observed only for the longer duty cycles (i.e., 60:120 s, and 90:180 s). Although our intermittent exercise protocol presents different characteristics (e.g., work:recovery = 2:1 and exercise intensity = 95% IVO_2max), it was also verified a reduced endurance performance during longer duty cycles performed with passive recovery. The intramuscular PCr concentration ([PCr]) kinetics both during and following high-intensity exercise presents a curvilinear profile and seems to be closely linked with VO_2 kinetics (Rossiter et al., 2002). For instance, under the conditions of the present study, is very likely that the amplitude of [PCr] restoration during the 240 s recovery intervals (Long protocol) was not doubled than what was presented when 120 s periods of recovery (Short protocol) were allowed. Moreover, Chidnok et al. (2013) demonstrated that [PCr] restoration become longer as the intermittent protocol continued. Thus, [PCr] is progressively lower immediately before each repetition, particularly when duty-cycle duration is lengthened. The metabolites generated by muscle contraction at this condition, such as Pi, ADP, and AMP, increase glycolytic flux and consequently, glycolytic H^+ (Adams et al., 1990; Conley et al., 1997) and lactate (Karpatkin et al., 1964) production. Low values of muscle [PCr] and pH (i.e., high values of [H^+]) and consistently high values of [Pi] and [ADP] have been associated with fatigue development during high-intensity exercise (Jones et al., 2008; Vanhatalo et al., 2010).

Another factor that can influence both endurance performance and metabolic response is the activity pattern performed during the recovery intervals between each bout (Chidnok et al., 2012). Using the CP model, Chidnok et al. (2012) demonstrated that endurance performance during intermittent exercise was enhanced only when the recovery intervals were performed below CP. Active recovery performed below CP allows a partial PCr reconstitution and/or clearance of fatigue-related metabolites (Chidnok et al., 2013), with the former being apparently more important to enhance endurance performance during high-intensity intermittent exercise. Indeed, both endurance performance (Chidnok et al., 2012) and PCr reconstitution (Chidnok et al., 2013) are higher during intermittent exercise with passive recovery than during active recovery performed bellow CP condition. Thus, a lower PCr reconstitution can explain, at least in part, the impaired endurance performance during short condition performed with active recovery, as observed in the present study.

However, a different scenario emerges from the data obtained during the Long intermittent exercise protocol. At this condition, endurance performance was not modified by the active recovery periods. Two different mechanisms, which

can occur simultaneously, could help explain this phenomenon. Firstly, the negative influence of active recovery on PCr reconstitution could be time-dependent, i.e., longer duty-cycle could allow more similar PCr reconstitution than a shorter one. The curvilinear PCr recovery profile supports this hypothesis (Harris et al., 1976). Secondly, the clearance of lactate and H^+ ions within muscles might be higher during the longer duty-cycle. A higher muscle pH can reduce, directly or indirectly (a more favorable metabolic milieu for PCr reconstitution), fatigue during high-intensity exercise. Alternatively, it is possible that [PCr] kinetics both during and following high-intensity intermittent exercise would contribute progressively less to endurance performance when the duty-cycle duration is lengthened.

The end-exercise VO_2 was not significantly different between CWR exercise and VO_2max measured during the incremental test. This is consistent with the fact that exhaustive exercise performed within the severe intensity domain (i.e., above CP) is characterized by the development of the VO_2SC, which is truncated at VO_2max. Some interventional (e.g., endurance training and priming exercise) (Jones et al., 2007; Caritá et al., 2014) and correlational studies (Barbosa et al., 2014a) have produced evidences that both VO_2 kinetics (a proxy for intramuscular PCr kinetics) (Rossiter et al., 2002) and VO_2max attainment is related to endurance performance during high-intensity exercise. Thus, it was hypothesized that VO_2SC trajectory, which reflects the interaction between VO_2SC and VO_2max attainment, could explain the endurance performance during high-intensity intermittent exercise. Indeed, it was demonstrated that VO_2SC trajectory was faster during CWR exercise than during intermittent exercise, regardless of duration and recovery type. However, similar to the results found by Chidnok et al. (2012), VO_2SC trajectory was not significantly different among intermittent exercise, and end-exercise VO_2 was lower during these conditions than at CWR exercise. Thus, substrate utilization/accumulation, VO_2 kinetics (VO_2SC trajectory and end-exercise VO_2) and endurance performance during high-intensity exercise seem to present different relationship during CWR and intermittent exercise. Priming high-intensity exercise has previously been reported to reduce the amplitude of VO_2SC and an increase in apparent W' during subsequent exercise (Caritá et al., 2014, 2015; Dekerle et al., 2015). In this context, each preceding intermittent exercise bout may have "primed" the muscle (i.e., reduces the amplitude of VO_2SC and/or raise the W') during subsequent bouts. These modifications are consistent with enhanced endurance performance, and could help to explain the apparently different metabolic regulation imposed by the interaction between intervals duration and recovery type during intermittent exercise.

Our experimental protocol (i.e., exercise intensity, work:recovery durations and recovery types) was specifically designed to investigate the hypothetical association between intermittent endurance performance and VO_2SC kinetics. Similar to previous studies (Caputo and Denadai, 2008; Barbosa et al., 2014a), both CWR and intermittent exercise were performed at 95% IVO_2max. As demonstrated in the present study, exhaustive exercise performed at this intensity is characterized by the development of the VO_2SC and VO_2max attainment. Some studies have utilized the "percentage delta" (for details please see Lansley et al., 2011) aiming to select a predetermined exercise intensity domain (i.e., heavy or severe) and/or to standardize the exercise intensity between subjects. Indeed, when compared to a more traditional method (e.g., %VO_2max), this approach allows a lower inter-subject variability of physiological responses to CWR exercise (Lansley et al., 2011). However, for the first time, the present study have normalized the wok:recovery durations based on the individual VO_2SC kinetics response. Thus, we are confident that the inter-subject variability of physiological responses during the intermittent exercise was attenuated. Finally, this study presented a possible limitation, since the effect of passive and active recovery on intermittent exercise was analyzed using 2 different groups of active individuals. Hypothetically, this experimental design could be influenced by the individual variability on both endurance performance and VO_2SC kinetics. However, PR and AR groups have presented similar data during incremental (VO_2max, IVO_2max, 95% IVO_2max and LT) and CWR exercise (endurance performance and VO_2SC kinetics). Therefore, the possibility of inter-subject variability influencing the recovery types comparisons was probably reduced. This limitation comes from the heavy testing required to be undertaken by each subject to test our research hypothesis. It is important to note that a short-term training program (6 sessions) involving high-intensity exercise (repeated all-out sprint training) have reduced the amplitude of the VO_2SC and increased tolerance to high-intensity exercise in recreationally active subjects (Bailey et al., 2009). Thus, if a repeated measures design has been utilized in our experimental approach, a confounding factor could be added to our analysis, since the volunteers would have to perform 6 bouts of severe-intensity exercise.

CONCLUSION

The present study showed that under our experimental conditions (i.e., exercise intensity, work:recovery durations and recovery type), intermittent exercise enhances endurance performance during severe-intensity exercise, independently of intervals duration and recovery type. Passive recovery is superior in relation to active recovery to enhance endurance performance only during shorter duty-cycles. Although VO_2SC trajectory is attenuated during high-intensity intermittent exercise, its alteration does not seem to explain the interaction effects of intervals duration and recovery type on endurance performance. Moreover, the end-exercise VO_2 was lower during intermittent exercise than at CWR exercise. Thus, severe-intensity intermittent exercise performed with different intervals duration and recovery type seems to modify the relationship between endurance performance and VO_2 kinetics observed during CWR exercise. Further studies using a repeated measures design are required to examine the effect of severe-intensity intermittent exercise on both endurance performance and VO_2SC in trained individuals. A threshold

in the duration of the recovery, from which PCr resynthesis and/or W' reconstitution would be less affected by active recovery could be identified. This can help to explain and confirm our main results, giving support to elaborate a more sophisticate interval training programs for different populations.

AUTHOR CONTRIBUTIONS

Study design: BD and CG. Data acquisition and analysis: LB, BD, and CG and Writing the paper: LB, BD, and CG.

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No Critical Peripheral Fatigue Threshold during Intermittent Isometric Time to Task Failure Test with the Knee Extensors

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It has been proposed that group III and IV muscle afferents provide inhibitory feedback from locomotor muscles to the central nervous system, setting an absolute threshold for the development of peripheral fatigue during exercise. The aim of this study was to test the validity of this theory. Thus, we asked whether the level of developed peripheral fatigue would differ when two consecutive exercise trials were completed to task failure. Ten trained sport students performed two exercise trials to task failure on an isometric dynamometer, allowing peripheral fatigue to be assessed 2 s after maximal voluntary contraction (MVC) post task failure. The trials, separated by 8 min, consisted of repeated sets of 10 × 5-s isometric knee extension followed by 5-s rest between contractions. In each set, the first nine contractions were performed at a target force at 60% of the pre-exercise MVC, while the 10th contraction was a MVC. MVC and evoked force responses to supramaximal electrical femoral nerve stimulation on relaxed muscles were assessed during the trials and at task failure. Stimulations at task failure consisted of single stimulus (SS), paired stimuli at 10 Hz (PS10), paired stimuli at 100 Hz (PS100), and 50 stimuli at 100 Hz (tetanus). Time to task failure for the first trial (12.84 ± 5.60 min) was longer ($P < 0.001$) than for the second (5.74 ± 1.77 min). MVC force was significantly lower at task failure for both trials compared with the pre-exercise values (both $P < 0.001$), but there were no differences in MVC at task failure in the first and second trials ($P = 1.00$). However, evoked peak force for SS, PS100, and tetanus were all reduced more at task failure in the second compared to the first trial ($P = 0.014$ for SS, $P < 0.001$ for PS100 and tetanus). These results demonstrate that subjects do not terminate exercise at task failure because they have reached a critical threshold in peripheral fatigue. The present data therefore question the existence of a critical peripheral fatigue threshold during intermittent isometric exercise to task failure with the knee extensors.

Keywords: maximal voluntary contraction, femoral nerve electrical stimulation, neuromuscular activation, neuromuscular fatigue, evoked peak force, knee extension, electromyography, rating of perceived exertion

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INTRODUCTION

Neuromuscular fatigue is often defined as a reduction in maximal voluntary contraction (MVC) force. Both (i) central fatigue, defined as a reduction in the maximal capacity of the central nervous system to maximally recruit motor units to produce force and (ii) peripheral fatigue, defined as the reduction in force originating from sites at or distal to the neuromuscular junction (Gandevia, 2001) contribute to neuromuscular fatigue. Peripheral fatigue is commonly measured as a reduction in evoked force responses to electrical or magnetic supramaximal stimulations delivered to the motor nerve to relaxed muscles (Verges et al., 2009; Millet et al., 2011).

It has been proposed that peripheral fatigue is the critical event at task failure (Amann et al., 2006; Amann and Dempsey, 2008) and that group III and IV muscle afferents provide inhibitory feedback from locomotor muscles to the central nervous system (Taylor and Gandevia, 2008), influencing the regulation of central motor drive during fatiguing exercise, and thus playing a key role in determining the moment of exhaustion (Taylor and Gandevia, 2008; Amann, 2012). It has been further proposed that a reduction in central motor drive i.e., a reduction in voluntary descending drive from the primary motor cortex usually indirectly measured via electromyography (EMG) (Amann et al., 2013), constrains the development of peripheral fatigue to a certain "critical" threshold associated with a given level of intramuscular metabolic perturbation (Amann et al., 2006). According to this model, humans may not ever exceed a critical level of peripheral fatigue, leading to the proposal of a critical peripheral fatigue threshold (Amann et al., 2006; Amann and Dempsey, 2008). As a result, when the critical peripheral fatigue threshold is approached, feedback from group III and IV muscle afferents reduces central motor drive and thus exercise intensity during self-paced exercise (Amann and Dempsey, 2008), or triggers task failure during constant load exercise (Amann et al., 2011).

In support of a critical peripheral fatigue threshold, similar levels of peripheral fatigue have been reported after constant-load endurance exercise with different degrees of arterial oxygen content (Amann et al., 2006), after intermittent isometric knee extension to task failure at different intensities (Burnley et al., 2012), after self-paced endurance exercise whether or not subjects were pre-fatigued before exercise (Amann and Dempsey, 2008), and after all-out cycling sprints whether or not subjects were pre-fatigued by electrical stimulation (Hureau et al., 2014). Support for a critical peripheral fatigue threshold is provided by studies showing greater levels of peripheral fatigue at the end of exercise following selective blockade of sensory afferents with intrathecal fentanyl injection compared to saline (Amann et al., 2009, 2011; Blain et al., 2016).

However, a critical peripheral fatigue threshold is not a universal finding, leading some authors to question the importance of peripheral fatigue in regulating exercise performance (Marcora and Staiano, 2010; Christian et al., 2014; Froyd et al., 2016; Neyroud et al., 2016). But these criticisms of this theory have been dismissed on the basis that some studies employed designs in which the interventions produced lower levels of peripheral fatigue than did the control conditions (Johnson et al., 2015). It has been argued (Broxterman et al., 2015) that to disprove the existence of a critical peripheral fatigue threshold, an experimental manipulation must cause the subjects to surpass the threshold, that is, by achieving higher levels of peripheral fatigue in the intervention condition. If inhibitory feedback from group III and IV muscle afferents constrains the extent to which peripheral fatigue develops during endurance exercise (Amann, 2011, 2012), it follows that trials of similar intensity, but different pre-fatiguing conditions will be of different durations, but should finish at similar levels of peripheral fatigue.

Therefore, the aim of this study was to test the validity of the critical peripheral fatigue threshold model during exercise until task failure. Subjects performed isometric knee extension exercise on a dynamometer, allowing assessment of peripheral fatigue at task failure. After 8 min of recovery, subjects completed a second exercise bout, also to task failure. We hypothesized that evoked peak force would be lower at task failure in the second trial compared to the first one, showing that the first exercise bout did not terminate because a critical peripheral fatigue threshold had been reached.

MATERIALS AND METHODS

Subjects

Ten sport students (five men, five women, mean ± SD age: 24 ± 4 years, body mass: 71 ± 12 kg, height: 176 ± 9 cm) participated in the study. Subjects were trained in both endurance and strength exercises and classified as performance level 3 or 4 (De Pauw et al., 2013; Decroix et al., 2016). None of the subjects had any leg injury or knee pain. Subjects were instructed to refrain from high-intensity exercise on the day prior to testing and to refrain from alcohol during the last 24 h before testing. Subjects were also instructed to eat a light meal 2–4 h before arrival to the laboratory. The study was approved by the Regional Ethics Committee in Norway (2011/1634), and the experiments were performed according to the latest (2013) revision of the Declaration of Helsinki. The subjects gave their written informed consent to participate in the study. Subjects were given a full explanation of the details and rationale of the study and were informed that they were free to withdraw at any time. The possibility that electrical stimulation might cause discomfort was fully explained as was the nature of the risks involved.

Experimental Protocol

Each subject visited the laboratory on two occasions. During the first visit, the subjects were familiarized with the procedures that would be used for assessment of neuromuscular function consisting of electrical stimulation and isometric MVC. In addition, the subjects were familiarized with the experimental trial involving intermittent isometric contractions at 60% of

Abbreviations: EMG, electromyography; MVC, maximal voluntary contraction; PS10, paired stimuli at 10 Hz; PS100, paired stimuli at 100 Hz; PS10/PS100, evoked peak force for PS10/PS100; RMS, root mean square; $RMS \cdot M^{-1}$, root mean square/M-wave peak to peak amplitude; RPE, rating of perceived exertion; SS, single stimulus; Tetanus, tetanic stimulation, 50 stimuli at 100 Hz = 0.5 s.

MVC force until task failure with knee extension on the KinCom dynamometer (Kinematic Communicator, Chattecx Corp., Chattanooga, TN). Three to five days after the familiarization visit, subjects visited the laboratory for the experimental trials.

Trials to Task Failure

Subjects performed two isometric knee extension trials with the right leg to task failure (**Figure 1A**), separated by 8 min. One-leg constant load knee extension exercise has been used to investigate the critical peripheral fatigue threshold previously (Amann et al., 2013), but with measurement of peripheral fatigue 2 min after task failure. In the present study, peripheral fatigue assessments began within 2 s following completion of the MVC (i.e., within 7 s post task failure), since we have shown that peripheral fatigue recovers substantially within 1 min after exercise cessation (Froyd et al., 2013), and it is not known if recovery of peripheral fatigue is the same after different exercise trials. During the trials, subjects performed consecutive sets of 10 × 5-s isometric contractions followed by 5-s rest between contractions (**Figure 1B**). The first nine contractions were performed at a target force at 60% of pre-exercise MVC, while the 10th contraction in each set was a MVC. Electrical stimulation to assess neuromuscular function was applied after each MVCs in each set. A target line on a 24-inch widescreen monitor, positioned in front of the subject, was used for visual feedback of the force recordings during both trials. Task failure occurred when the subject could not maintain the required force for at least 4 s for two consecutive contractions, with subjects being informed each time they failed to achieve the required force output. The experimenter made the decision when task failure had occurred. Following the second missed contraction, subjects were instructed to produce a final 5-s MVC, followed (2 s) by the electrical stimulation protocol described below.

Settings and Warm-Up

On arrival at the laboratory, subjects were secured to the dynamometer by chest and hip strapping to avoid excessive lateral and frontal plane movements. The seating was adjusted

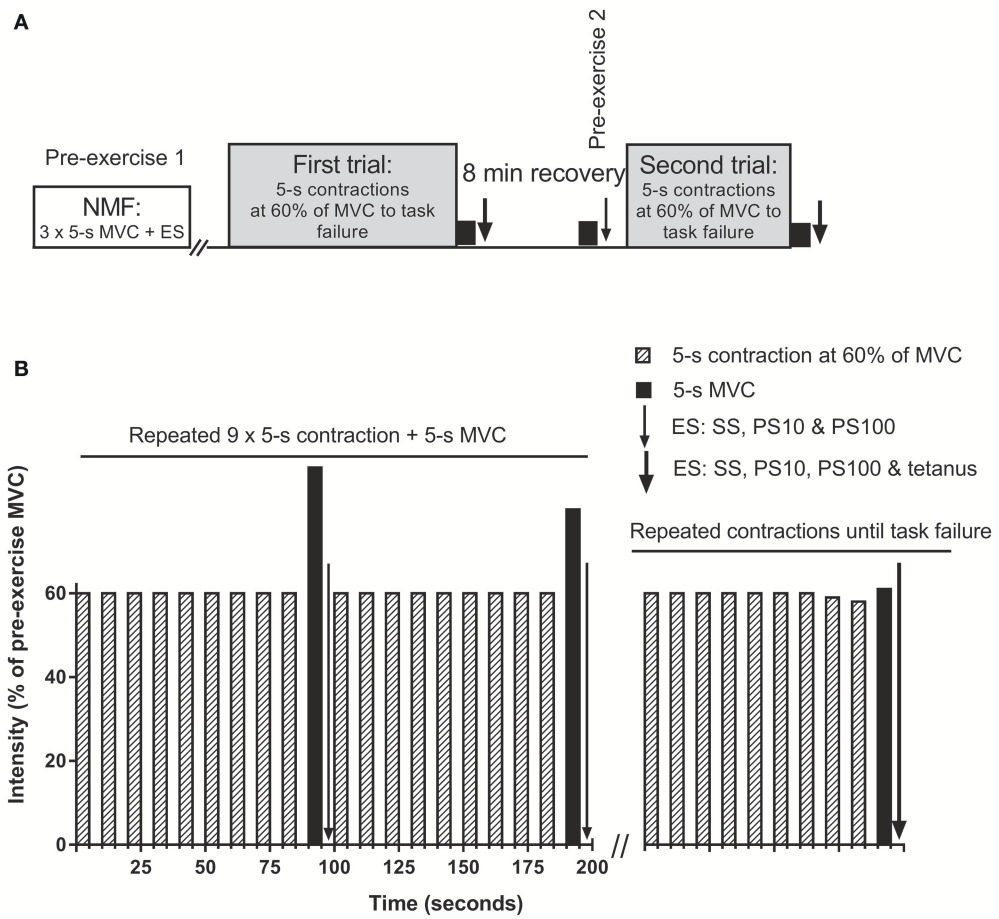

FIGURE 1 | Overview of the protocol (A) and detailed description of the trials **(B)**. **(A)** first and second trials were separated by a break of 8 min including the neuromuscular function measurements (NMF). NMF, i.e., a maximal voluntary contraction (MVC) followed within 2 s by electrical stimulation (ES), was assessed three times prior to the first trial (pre-exercise 1), after each set during the trials, at task failure, as well as 1 min before the second trial (pre-exercise 2). **(B)** trials consisted of consecutive sets of 10 × 5-s isometric contractions followed by 5-s rest between contractions. The first nine contractions were performed at a target force at 60% of pre-exercise MVC, while the 10th contraction in each set was a MVC. ES was applied in the 5-s break before the next set of contractions began. Sets of contractions were repeated until task failure. SS, single stimulus; PS10, paired stimuli at 10 Hz; PS100, paired stimuli at 100 Hz; tetanus, 50 stimuli at 100 Hz.

for each subject, with the right knee femoral epicondyle aligned with the axis of the dynamometer's rotation arm. The right lower leg was attached to the lever arm just above the lateral malleolus. The left leg was not active at any time and was secured to the dynamometer by strapping around the upper leg. The seat's backrest was reclined 10 degrees, and the dynamometer's rotation arm was kept at 90 degrees. Hip and knee angle was approximately 110 and 80 degrees, respectively. Subjects kept their hands crossed in front of their upper body and in the same position during all experiments.

Warm-up consisted of 5-s isometric contractions followed by 5-s rest. The intensity was 25% of MVC force for five contractions, 50% of MVC force for five contractions, and 75% of MVC force for two contractions. MVC force from the familiarization visit was used to determine warm-up intensity. The rest period between each set was 30 s.

Neuromuscular Function Assessment

Neuromuscular function assessment consisted of a 5-s MVC followed by a sequence of electrical stimuli. For the MVC, the subjects were instructed to produce maximal force for 5 s whilst they received strong verbal encouragement. Femoral nerve electrical stimulation on relaxed muscles consisted of single stimulus (SS), paired stimuli at 10 Hz (PS10), and paired stimuli at 100 Hz (PS100), and assessment started within 2 s after a MVC. The interval between the stimulation techniques was 1.5 s. Hence neuromuscular function assessment duration excluding MVC was approximately 3.5 s. In addition, PS100 was followed by tetanus (50 stimuli at 100 Hz = 0.5 s) once prior to the first trial and once at task failure of both trials. Thus, electrical stimulation lasted from second 2–7 after the MVC at task failure.

Pre-exercise neuromuscular function (**Figure 1A**) assessment started 2 min after the warm up. Three isometric MVCs, each lasting 5 s were performed with a 2 min break between MVCs and followed by electrical stimulation. Neuromuscular function was also assessed after each set during the trials, at task failure, and 1 min prior to the start of the second trial. Power Lab (ADInstruments Pty Ltd, Bella Vista NSW, Australia) was used to trigger the electrical stimulation.

Data Collection

Electrical Stimulation

A high voltage (maximal voltage 400 V) constant current stimulator (DS7AH, Digitimer, Hertfordshire, UK) was used to deliver square-wave stimuli of 1 ms duration. The femoral nerve was stimulated percutaneously via a 10 mm diameter self-adhesive cathode electrode (Skintact, Austria) pressed manually by the investigator onto the skin at the femoral triangle. The anode, a 130 × 80 mm self-adhesive electrode (Cefar-Compex Scandinavia AB, Sweden), was applied to the gluteal fold. The optimal stimulation intensity for one single stimulus was determined by increasing the current gradually from 10 mA until a plateau in force was reached. The current was then increased by a further 30% (current range: 35–60 mA) to ensure supramaximal stimulation. The intensity was kept constant for the same subject for all types of electrical stimulation. The subjects were instructed to relax fully whilst the electrical stimulation was applied.

EMG Recordings

EMG signals from the *vastus lateralis* and *vastus medialis* of the right leg were recorded via surface electrodes (DE-2.1 single differential surface sensors, distance between muscle site contacts = 10 mm; Delsys Inc, Boston, MA). SENIAM (Merletti and Hermens, 2000) recommendations were used for the placement of the sensors on the skin. The skin was shaved and wiped with isopropyl alcohol before the sensors were applied. The reference electrode was applied to the patella. EMG signals were sampled at 2000 Hz and amplified (gain = 1000) using Bagnoli-8 (Delsys Inc). EMG signals were transferred together with simultaneous force and electrical stimulation recordings into Power Lab (ADInstruments) and filtered using a band pass filter with a bandwidth at 15–500 Hz in Lab Chart Pro software (ADInstruments).

RPE

Perceived exertion (also known as perception of effort) defined as "the conscious sensation of how hard, heavy, and strenuous exercise is" (Pageaux, 2016), was assessed after every 8th contractions in each set for the trials using the ratings of perceived exertion (RPE) scale (Borg, 1974). Standardized instructions for the scale were given to subjects before the warm-up. Subjects were asked to rate how hard they were driving their leg during the exercise, but not to include an expression of pain in their legs.

Experimental Variables and Data Analysis

Force Data

Mean of the three successful MVCs prior to the first trial of exercise was taken as the pre-exercise MVC. Pre-exercise MVC force was used for calculation of the target force at 60% of MVC in both trials. MVC force was calculated as the highest average force sustained for 1 s. Force was also calculated for the first nine contractions of each set by averaging the force during the middle 4 s of the 5 s contractions. The force responses to electrical stimulation are reported as evoked peak force. The mean value in evoked peak force after the three MVCs was therefore used as the pre-exercise value. A reduction in evoked peak force, highlighting peripheral fatigue development, is due to factors distal to the site of stimulation, that is, at the neuromuscular junction or within the muscle. PS10/PS100 (evoked peak force for PS10/PS100) was calculated as an index of low-frequency fatigue (Verges et al., 2009).

EMG

The root mean square (RMS) of the EMG data of *vastus lateralis* and *vastus medialis* was calculated for 1 s around peak force for MVC, i.e., 500 ms before and after peak force, and for the middle 4 s of the first nine contractions of each set. M-wave peak-to-peak amplitude in response to SS was also assessed. RMS during voluntary contractions was normalized to RMS of pre-exercise MVC. In addition RMS during voluntary contractions was divided by the M-wave peak to peak amplitude of the following SS response to estimate neuromuscular activation (Millet et al., 2011). To limit the number of MVCs at task failure, voluntary

activation was not assessed to calculate the extent of central fatigue.

Statistical Analyses

After checking for the normality of data distribution using the Shaprio-Wilk's test, one-way repeated-measures ANOVAs with Bonferroni *post hoc* corrections were used to detect differences over time (pre-exercise 1, task failure first trial, pre-exercise 2, and task failure second trial; **Table 1**). Where the assumption of sphericity (Mauchy's test) was violated, the Greenhouse-Geisser Epsilon correction was applied to the degrees of freedom. A paired samples student's *t*-test was used for the following pairwise comparisons for differences at task failure between the two trials for neuromuscular function parameters expressed as a percent of baseline values (**Table 1**); the slope of within-participants RPE values between trials; and set force during the last set between the two trials. Differences in RPE values after the first set of contractions and at task failure between trials was analyzed using a one-way repeated-measures ANOVA with Bonferroni *post hoc*. The statistical significance was defined at $P < 0.05$. Effect sizes are given as Partial Eta Squared for the ANOVA and Cohen's dz for the paired *t*-tests. All analyses were performed using SPSS version 23 (SPSS, Inc., Chicago, IL), except for paired samples student's *t*-test and Cohen's dz (Microsoft Excel 2013, Microsoft Corporation, WA). The results are presented as mean ± SD.

RESULTS

Target force for each set of contractions was predetermined by the protocol, however it was slightly (∼1%) higher during the last set of contractions for the first compared with the second trial (311 ± 49 vs. 308 ± 47 N respectively, $t(9) = 2.38$, $P = 0.041$, $dz = 0.97$). As expected, time to task failure was longer ($P < 0.001$) during the first compared with the second trial (12.84 ± 5.60 vs. 5.74 ± 1.77 min, $t(9) = 5.45$, $P < 0.001$, $dz = 1.72$), indicating a 55 ± 16% reduction in time to task failure between the first and second trials.

RPE was lower at the end of the first set for the first compared with the second trial (12.0 ± 1.6 vs. 13.8 ± 1.3, $P = 0.020$; **Figure 2**). During the last set of contraction however RPE was not different between trials (18.6 ± 1.3 vs. 18.2 ± 1.5 for first and second trial respectively, $P = 0.517$). A comparison of the within-subjects RPE slopes between conditions indicated that rate of increase in RPE was higher for the second compared with the first trial (2.2 ± 0.9 vs. 1.3 ± 1.1 units/set respectively, $t(9) = 6.08$, $P < 0.001$, $dz = 1.91$).

TABLE 1 | Effects of knee extensors intermittent isometric time to task failure on knee extensors neuromuscular function.

	Pre-exercise 1	Task failure first trial	Pre-exercise 2	Task failure second trial	F	*P*-value	Effect size
MVC (N)	547 ± 123	346 ± 72 $^{+++}$	410 ± 102 $^{+++}$	336 ± 62 $^{+++,‡‡}$	(1.55, 13.97) = 56.75	< 0.001	η_p^2 =.863
Δ%		−36 ± 8		−38 ± 8		0.216	Dz = 0.42
SS (N)	152 ± 48	70 ± 17 $^{+++}$	93 ± 28 $^{+++}$	62 ± 16 $^{+++,*,‡‡}$	(1.15, 10.39) = 43.17	< 0.001	η_p^2 =.827
Δ%		−52 ± 11		−57 ± 10**		0.005	Dz = 1.15
PS10 (N)	242 ± 72	107 ± 27 $^{+++}$	133 ± 43 $^{+++}$	92 ± 28 $^{+++,**,‡‡}$	(1.19, 10.75) = 51.19	< 0.001	η_p^2 =.850
Δ%		−54 ± 11		−60 ± 10***		< 0.001	Dz = 1.65
PS100 (N)	231 ± 62	137 ± 29 $^{+++}$	166 ± 42 $^{+++}$	123 ± 30 $^{+++,***,‡‡‡}$	(1.10, 9.91) = 47.20	< 0.001	η_p^2 =.840
Δ%		−39 ± 10		−45 ± 10***		< 0.001	Dz = 2.02
PS10/PS100	1.04 ± 0.07	0.78 ± 0.08 $^{+++}$	0.79 ± 0.11 $^{+++}$	0.75 ± 0.09 $^{+++}$	(1.05, 9.46) = 32.02	< 0.001	η_p^2 =.781
Δ%		−25 ± 6		−28 ± 7		0.072	Dz = 0.65
Tetanus (N)	471 ± 141	356 ± 102 $^{++}$	NA	314 ± 101 $^{+++,***}$	(3, 27) = 62.76	< 0.001	η_p^2 =.875
Δ%		−23 ± 10		−32 ± 11***		< 0.001	Dz = 2.46
MVC RMS·M^{-1} *VL*	0.057 ±.012	0.063 ± 0.023	0.054 ± 0.014	0.070 ± 0.020	(1.54, 13.84) = 4.18	0.046	η_p^2 =.317
Δ%		11 ± 25		25 ± 28		0.098	Dz = 0.58
MVC RMS·M^{-1} *VM*	0.072 ±.015	0.079 ± 0.024	0.067 ± 0.017 $^{+}$	0.085 ± 0.023 ‡	(3, 27)= 7.29	0.001	η_p^2 =.448
Δ%		9 ± 17		18 ± 16		0.224	Dz = 0.41
PPA *VL* (mV)	3.75 ± 1.03	3.62 ± 1.01	3.25 ± 0.90 $^{+}$	3.45 ± 0.82	(3, 27) = 3.51	0.002	η_p^2 =.421
Δ%		−3 ± 10		−7 ± 13		0.249	Dz = 0.39
PPA *VM* (mV)	3.80 ± 0.61	3.55 ± 0.81	3.18 ± 0.59 $^{+}$	3.47 ± 0.92	(1.53, 13.73) = 6.44	0.015	η_p^2 =.417
Δ%		−7 ± 14		−9 ± 17		0.340	Dz = 0.32

*Values are expressed in absolute units and as a percentage change from pre-exercise 1 (Δ%). Values are expressed as means ± SD (n = 10). MVC, maximal voluntary contraction; SS, single stimulus; PS10, paired stimuli at 10 Hz; PS100, paired stimuli at 100 Hz; Tetanus, 50 stimuli at 100 Hz; RMS, root mean square; M, M-wave; VL, vastus lateralis; VM, vastus medialis; PPA, peak to peak amplitude of the M-wave; NA, not assessed. Significant difference compared with pre-exercise 1: + P < 0.05, + + + P < 0.001; significant difference between task failure of the first trial and task failure of the second trial: * P < 0.05, ** P < 0.01, *** P < 0.001; significant difference between pre-exercise 2 and task failure of the second trial: † P < 0.05, ‡‡ P < 0.01, ‡‡‡ P < 0.001.*

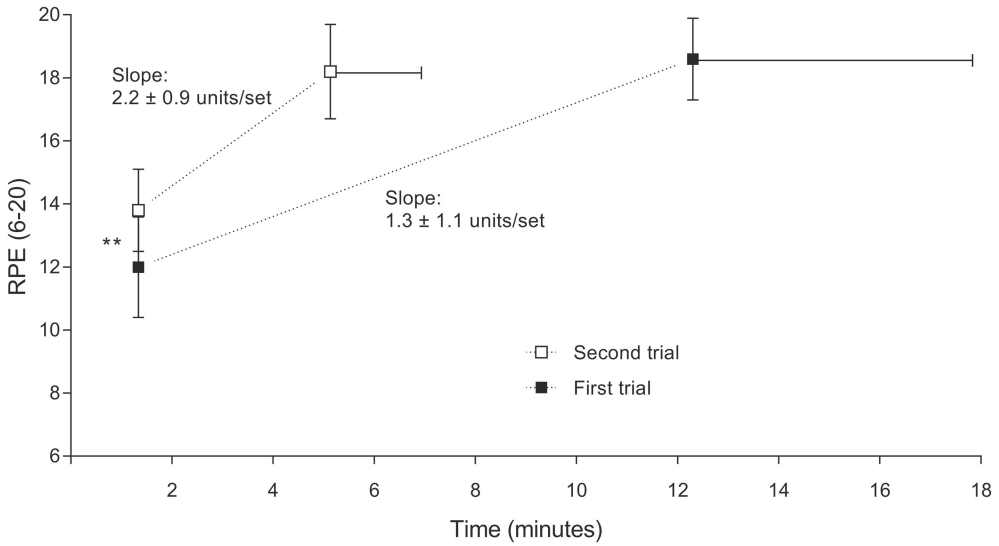

FIGURE 2 | Changes of ratings of perceived exertion (RPE) during the first and second trial, from the first to the last completed contraction set. Data are shown as mean ± SD, $n = 10$. Significant difference between first and second trial: ** $P < 0.01$. RPE during the last set was not different ($P = 0.517$) between trials.

Neuromuscular Fatigue

Neuromuscular function responses as absolute values prior to the first trial, 1 min prior to the second trial, at task failure in the first and second trials, and in addition at task failure in the first and second trials expressed in percentage of pre-exercise of the first trial, are presented in **Table 1**. MVC force was significantly lower at task failure for both trials compared with pre-exercise 1 (both $P < 0.001$), but no differences were observed between the first and the second trial at task failure ($P = 1.00$). Evoked peak force for SS, PS10, PS100, and tetanus were significantly lower at task failure for both trials compared with pre-exercise 1 (all $P < 0.001$). Importantly, these three indices of peripheral fatigue were reduced more at task failure in the second compared with the first trial ($P = 0.014$ for SS, $P = 0.002$ for PS10, and $P < 0.001$ for PS100 and for tetanus). PS10/PS100 was significantly lower at task failure for both trials compared with pre-exercise 1 (both $P < 0.001$), but no differences were found at task failure between trials ($P = 0.464$). M-wave amplitude was unchanged at task failure for both trials compared to pre-exercise 1 for the *vastus lateralis* ($P = 1.00$ and $P = 0.589$ for first and second trial respectively) and *vastus medialis* ($P = 1.00$ and $P = 0.853$ for first and second trial respectively). RMS·M^{-1} during MVC was not significantly decreased at task failure in any of the trials compared to pre exercise 1 for both *vastus lateralis* ($P = 1.00$ and $P = 0.182$ for first and second trial respectively) and *vastus medialis* ($P = 0.872$ and $P = 0.079$ for first and second trial respectively).

Individual responses to MVC, SS, PS100, and tetanus force at task failure of the first and second trial as percent change from pre-exercise prior to the first trial are presented in **Figure 3**. Significant differences between trials are presented in **Table 1**.

DISCUSSION

The present study aimed to test the validity of the critical peripheral fatigue threshold model during isometric intermittent contractions of the knee extensors continued until task failure. The most important finding of this study was that evoked peak forces (including tetanus, see below) were reduced to a greater extent at task failure after the second trial compared with the first trial, indicating that task failure in the first trial did not occur in order to ensure that a critical peripheral fatigue threshold was not exceeded during that bout of exercise.

Peripheral Fatigue at Task Failure

As expected from other studies (Amann and Dempsey, 2008; Neyroud et al., 2012; Amann et al., 2013; Hureau et al., 2014; Johnson et al., 2015), residual neuromuscular fatigue from the first trial contributed to reduced time to task failure during the second trial. However, while no differences in MVC force were found between trials at task failure, in accordance with Neyroud et al. (2012), all electrical stimulation methods including tetanus revealed higher levels of peripheral fatigue at task failure in the second trial.

Two studies have investigated the effect of pre-fatiguing concentric exercise until task failure on the critical peripheral fatigue threshold, and both studies reported differences in peripheral fatigue between trials (Amann et al., 2013; Johnson et al., 2015). In the study of Johnson et al. (2015), subjects cycled on a cycle ergometer to task failure at 85% of peak power with and without pre-fatiguing exercise of the arms. The level of peripheral fatigue was less and the exercise duration was shorter when the arm muscles were pre-fatigued by 8 × 1-min of arm-cranking at a fixed work rate. The finding of Johnson et al. (2015) are in line with another study (Amann et al., 2013), both of which indicate that pre-exercise might prevent the attainment of this so-called critical level of peripheral fatigue. In their study, Amann et al. (2013) compared fatigue induced by concentric single leg knee extension exercise with the same exercise task performed by the same leg following an exhaustive exercise bout with the other leg (Amann et al., 2013). Lower levels of peripheral fatigue were

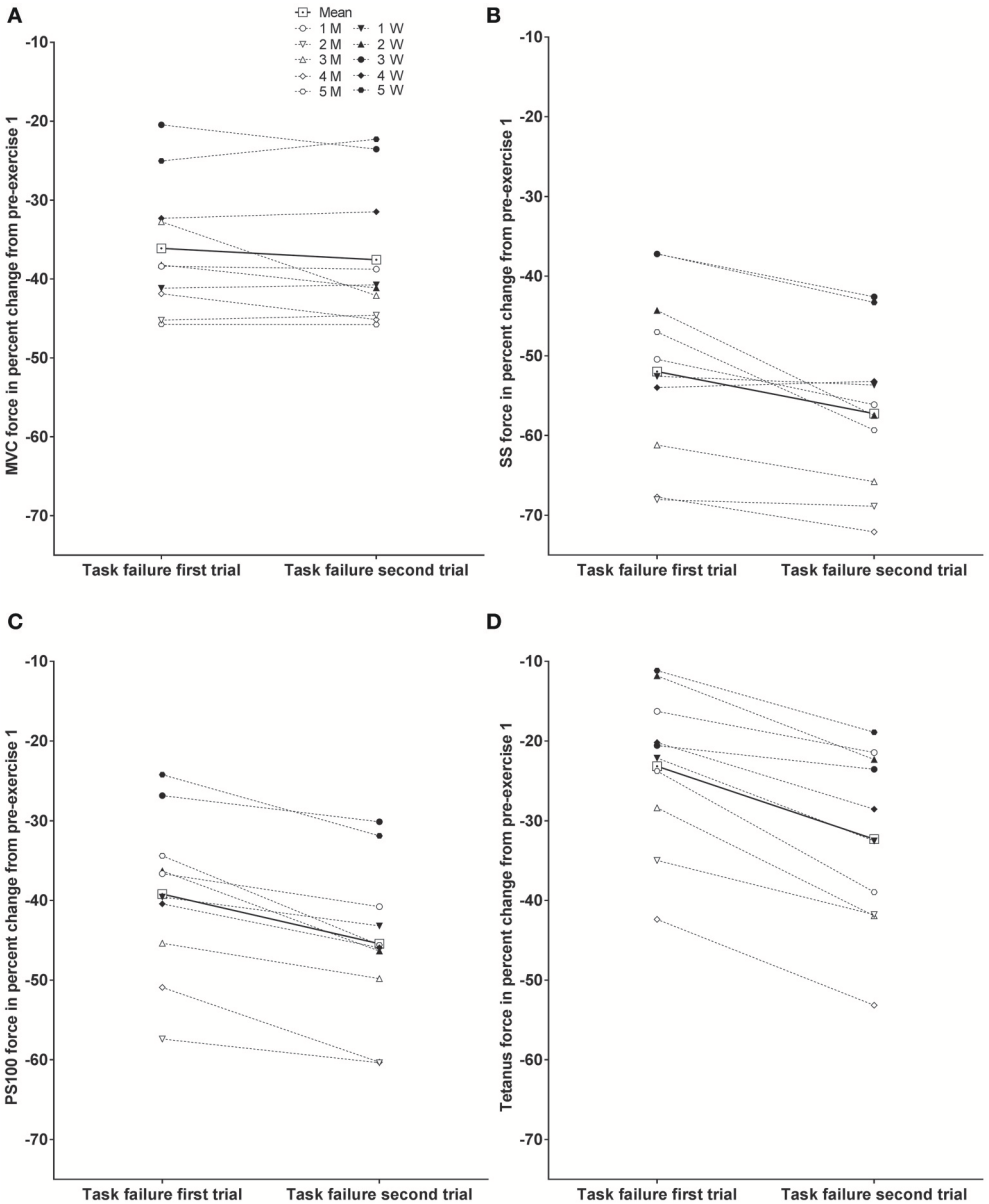

FIGURE 3 | Individual responses at task failure of the first and second trial as percent change from pre-exercise prior to the first trial; maximal voluntary contraction (MVC) force (A), evoked peak force for single stimulus (SS) **(B)**, evoked peak force for paired stimuli at 100 Hz (PS100) **(C)**, and evoked peak force for 50 stimuli at 100 Hz (tetanus) **(D)**. Male subjects (M) are indicated with open symbols, and female subjects (W) are indicated with closed symbols. Significant differences are presented in **Table 1**.

also reported after maximal intermittent dynamic leg extensions (Christian et al., 2014) or constant load cycling (Amann et al., 2007) in hypoxia versus normoxia and after longer than shorter knee extension time trials (Froyd et al., 2016).

Yet, none of these studies absolutely refutes the existence of a critical peripheral fatigue threshold since the experimental conditions differed. To our knowledge, only the present study and that of Neyroud et al. (2012) have compared the level of peripheral fatigue at task failure following a pre-fatiguing isometric trial of the same muscle group. In the latter study (Neyroud et al., 2012), the subjects performed consecutive trials of sustained isometric exercise at 20% of MVC force, interspersed with electrical muscle stimulation. MVC force at task failure was similar between trials and the level of peripheral fatigue at task failure was greater after the second trial. As in the present study, those authors concluded that task failure may not be associated with a critical threshold of peripheral fatigue. Evoked peak forces for SS and PS100 immediately at task failure were reduced equally or slightly more in the present study compared to the data of Neyroud et al. (2012).

In the present study, not only do we confirm these results for voluntary contractions only but we also show that the less frequently used stimulation method of tetanic stimulation (Place et al., 2010) also detected different evoked peak forces at task failure following the two trials. The advantage of tetanic stimulation is that the force response is less affected by potentiation than is the case with single or paired stimuli (Baudry et al., 2008). The measured reduction in evoked peak force was different for the different methods of electrical stimulation (SS > PS100 > tetanus) as previously shown for concentric knee extension exercise, highlighting the importance of the stimulation method used for the quantification of the absolute extent of peripheral fatigue (Froyd et al., 2013, 2016). Nonetheless, the finding of increased peripheral fatigue after the second bout was consistent for all methods of electrical stimulation. More recently, a critical peripheral fatigue threshold has also been questioned by considering individual vs. pooled data (Neyroud et al., 2016).

Validity of the Critical Peripheral Fatigue Threshold

Group III and IV muscle afferents provide inhibitory feedback from locomotor muscles to the central nervous system, presumably influencing the regulation of central motor drive during fatiguing exercise (Taylor and Gandevia, 2008; Amann, 2012). According to this model, humans may not ever exceed a specific level of peripheral fatigue (Amann et al., 2013). As a result, when approaching the critical peripheral fatigue threshold, group III, and IV muscle afferents should begin to inhibit muscle activation and thus cause task failure during constant load exercise (Amann et al., 2011).

Studies have reported similar levels of peripheral fatigue between constant-load cycling trials at 81–83% of peak power in normoxia vs. hypoxia (Amann et al., 2006, 2007), and between constant-load cycling trials at 83% of peak power, a 5 km cycling time-trial, and a 5 km cycling time-trial after pre-fatiguing constant-load cycling to task failure (Amann and Dempsey, 2008). In addition, further support for a critical peripheral fatigue threshold was that more peripheral fatigue was reached following selective blockade of sensory afferents with intrathecal fentanyl injection compared to saline (Amann et al., 2009, 2011; Blain et al., 2016). This suggests that group III and IV afferents might play a critical role in the prevention of dangerous levels of peripheral fatigue. However, despite reaching higher levels of peripheral fatigue, performance was not improved with inhibition of group III and IV muscle afferents with intrathecal fentanyl injection.

Interestingly, in several studies supporting the critical peripheral fatigue threshold, voluntary activation was not reduced after any of the constant load or self-paced endurance trials (Amann et al., 2007, 2011, 2013; Amann and Dempsey, 2008). It is possible that voluntary activation had recovered when measured a few minutes after end of exercise (see below). Even though there was no reduction in voluntary activation measured at task failure with the interpolated twitch technique, the authors (Amann et al., 2013) nevertheless concluded that peripheral fatigue and inhibitory feedback from group III and IV muscle afferents limited the endurance performance by restricting central motor drive to the working muscles. Since a decline in voluntary activation after exercise is indicative of a reduction in central motor drive to the muscles, available data does therefore not indicate that group III and IV muscle afferents inhibited central motor drive. However, a recent study suggests that decreased voluntary activation can be explained by inhibition of type III and IV afferents (Sidhu et al., 2017). Different levels of peripheral fatigue between trials (Neyroud et al., 2012; Amann et al., 2013; Johnson et al., 2015) without differences in voluntary activation are also incompatible with the critical peripheral fatigue threshold model of exercise regulation.

According to this model, endurance exercise performance is limited by a reduced central motor drive and hence force production at exercise termination (Amann et al., 2013). In contrast others argue that performance during endurance exercise does not terminate as a result of peripheral fatigue in the exercising muscles but is due rather to changes in the central nervous system (Marcora and Staiano, 2010; Neyroud et al., 2012, 2016; Morales-Alamo et al., 2015; Froyd et al., 2016). The basis for this conclusion was the finding that subjects were able to increase force production shortly after task failure and before there was any recovery in peripheral fatigue (Marcora and Staiano, 2010; Morales-Alamo et al., 2015). This has led other authors to propose alternative models in which RPE is not—as in the psychobiological model of endurance performance (Marcora et al., 2008; Pageaux, 2014)—or only partly—as in the flush model (Millet, 2011)—explained by the role of feedback from afferent fibers. In the present study and in others (Neyroud et al., 2012; Amann et al., 2013; Johnson et al., 2015), RPE was similar at the end of the trials despite the finding that RPE was higher in the first part of those trials when subjects were pre-fatigued by bouts of prior exercise. Those findings in addition to the differences in RPE slope between trials in the present study, also provide support for the psychobiological model of endurance performance (Marcora et al., 2008; Pageaux, 2014), the flush model (Millet, 2011), and the central governor model (Noakes, 2012).

Methodological Considerations and Limitations

While in the present study evoked peak force was assessed following isometric intermittent contractions of the knee extensors to task failure, the critical peripheral fatigue threshold model originated from studies using cycling as the exercise modality (Amann et al., 2006; Amann and Dempsey, 2008). An important limitation in cycling studies is that neuromuscular function is normally assessed several minutes after exercise cessation. In cycling studies investigating the critical peripheral fatigue threshold (Amann et al., 2006, 2009; Amann and Dempsey, 2008; Johnson et al., 2015), evoked peak force was first measured with SS 2–4 min after cycling exercise

cessation, at which time values were reduced by 32–38%. These percentage decreases in evoked peak force are very similar to those measured in the present study 7 min after the first trial (pre exercise trial 2, **Table 1**) and in our previous study, 4 and 8 min after exercise cessation (Froyd et al., 2013). It is probable that in studies in which peripheral fatigue is first assessed, at the earliest, even 2 min after exercise cessation, the absolute level of peripheral fatigue at exercise cessation is underestimated since peripheral fatigue recovers substantially within the first 1–2 min after exercise cessation (Froyd et al., 2013, 2014). Whether or not this findings contributed to the development of the critical threshold model is not known.

Subjects knew beforehand that the experiment comprised of two trials. Even though participants were strongly encouraged to exercise to their maximal capacity in both trials, it is possible that participants might have terminated the first trial in a "submaximal" state of fatigue but were motivated to exercise to higher levels of fatigue in the second trial since they knew that this was the final trial (Halperin et al., 2014). Although this could lead to biased results, this is a common feature of studies involving consecutive trials of exercise (Neyroud et al., 2012; Amann et al., 2013; Johnson et al., 2015). However, $RMS{\cdot}M^{-1}$, an index of central motor drive, during the last set and during the MVC at task failure were not different between the first and second trial, indicating that participants were not holding back at the end of the first compared to the second trial. In addition RPE values were the same at exercise termination in both trials suggesting equivalent effort.

The experimenter decided that task failure occurred when the developed force dropped below the target force. The experimenter was not blind to the aim of the study and might have allowed the subject to continue working below target force; this would have increased the development of peripheral fatigue. However, during the last set, force production was 1% lower during the second than first trial and RPE was not different between trials, indicating that differences in evoked peak force between trials could not be explained by differences in voluntary force production at target force.

CONCLUSIONS

It has been proposed that task failure during exhaustive endurance exercise is constrained by group III and IV inhibitory feedback from the exercising muscles specifically to ensure that the level of peripheral fatigue is always maintained below some critical threshold. In contrast, in this study we established that task failure occurred at different levels of evoked forces, including tetanus, during consecutive trials of similar target force. This indicates that task failure in the first trial did not occur in order to ensure that a critical peripheral fatigue threshold was not exceeded during that exercise bout.

AUTHOR CONTRIBUTIONS

The experiments were performed at Sogn og Fjordane University College, Norway. CF and TN conceptualized and designed the study. CF collected the data; CF analyzed the data, while all authors interpreted the data. CF drafted the manuscript, while all authors contributed to the manuscript and approved the final version of the manuscript.

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Are There Critical Fatigue Thresholds? Aggregated vs. Individual Data

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The mechanisms underlying task failure from fatiguing physical efforts have been the focus of many studies without reaching consensus. An attractive but debated model explains effort termination with a critical peripheral fatigue threshold. Upon reaching this threshold, feedback from sensory afferents would trigger task disengagement from open-ended tasks or a reduction of exercise intensity of closed-ended tasks. Alternatively, the extant literature also appears compatible with a more global critical threshold of loss of maximal voluntary contraction force. Indeed, maximal voluntary contraction force loss from fatiguing exercise realized at a given intensity appears rather consistent between different studies. However, when looking at individual data, the similar maximal force losses observed between different tasks performed at similar intensities might just be an "artifact" of data aggregation. It would then seem possible that such a difference observed between individual and aggregated data also applies to other models previously proposed to explain task failure from fatiguing physical efforts. We therefore suggest that one should be cautious when trying to infer models that try to explain individual behavior from aggregated data.

Keywords: task failure, endurance performance, peak twitch, maximal voluntary contraction, critical threshold, neuromuscular fatigue

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The typical answer when asking someone who just terminated a fatiguing task why stopped is "I just couldn't go on any further". If this suggests a conscious but forced decision to de-recruit the activated motor units for the task at hand, the underlying mechanisms forcing someone to terminate a fatiguing task are still not understood (Kayser, 2003). Given the great variety in physical tasks and their differing physiological constraints, it is unlikely that a single mechanism applies, just as it is likely that various types of effort will share some common pathways leading to the disengagement from the task at hand. For example, during dynamic exercise such as running or cycling, the cardiovascular strain is much greater than during an isometric contraction of a specific muscle group (Sidhu et al., 2013). For the former the sensation of effort is multimodal (e.g., breathlessness, palpitations, leg pain), while for the latter it is essentially related to the muscular effort *per se* (e.g., pain, loss of force) as the cardiorespiratory strain is limited.

The various mechanisms potentially implicated in exercise termination have been the focus of a great number of studies (for reviews see Kayser, 2003; Hunter et al., 2004; Marcora and Staiano, 2010; Enoka et al., 2011; Noakes, 2012; Amann et al., 2013; Enoka and Duchateau, 2016; Fan and Kayser, 2016; Taylor et al., 2016). In particular the role of peripheral fatigue, i.e., involving mechanisms located beyond the motor endplate, has been researched extensively. Functionally,

peripheral fatigue can be evaluated by quantifying reductions in evoked forces induced by single or paired supramaximal electrical or magnetic stimulations delivered over a motor nerve trunk before and after a fatiguing exercise. If peripheral fatigue extent has been proposed as a key determinant causing task failure by some authors (Amann and Dempsey, 2008; Amann et al., 2009, 2013; Amann, 2011; Sidhu et al., 2014; Blain et al., 2016), its role in the termination of different types of muscular effort is still hotly debated (Marcora, 2009; Marcora and Staiano, 2010; Johnson et al., 2015; Morales-Alamo et al., 2015).

A CRITICAL PERIPHERAL FATIGUE THRESHOLD AS A DETERMINANT OF TASK FAILURE?

A recently postulated mechanism shared between various types of muscular effort is a so-called "critical peripheral fatigue threshold," proposed by Amann et al. (2006, 2009, 2013) and Blain et al. (2016). According to those authors, peripheral fatigue normally does not exceed a certain individual critical threshold. If, during a given task, this threshold is reached, individuals either terminate it (open-ended tasks) or reduce the intensity (closed-ended tasks) (Amann et al., 2009). This critical peripheral fatigue threshold concept emerged from the observation of consistently reproducible peak twitch force reductions immediately following various cycling bouts to task failure (open-end) (Amann et al., 2006, 2007, 2009, 2011; Romer et al., 2007; Amann and Dempsey, 2008), as well as 5-km cycling time trials (closed-end) (Amann et al., 2006; Blain et al., 2016). Further support for such a critical peripheral fatigue threshold hypothesis was that at task failure of these open and closed-ended tasks, a greater reduction in peak twitch was reached following selective blockade of sensory afferents with intrathecal fentanyl injection compared to saline (Amann et al., 2009, 2011; Blain et al., 2016). Given the direct evidence of III-IV afferents involvement in exercise regulation from animal studies (Darques and Jammes, 1997; Dousset et al., 2004), this suggests that sensory type III-IV afferents might play a critical role in the regulation of a tightly regulated individual "permissible" extent of peripheral fatigue.

Yet, recent findings by Morales-Alamo et al. (2015) are difficult to reconcile with this notion of a critical fatigue threshold. In their hallmark study, the subjects performed 10-s long all-out isokinetic sprints before, and 10 or 60 s after an incremental maximal cycling test. Immediately at task failure of the incremental exercise, a bilateral cuff was placed around both thighs and inflated to occlude leg blood flow and hence prevent metabolite clearance. Given the (measured) low levels of phosphocreatine (PCr) and increased levels of adenosine diphosphate (ADP) at task failure, mitochondrial respiration was still high when the cuffs were inflated and the authors calculated that the little oxygen remaining was depleted within the following 3 s. Despite acidosis, anaerobic glycolysis and metabolites continued to accumulate during the ischemic recovery, as highlighted by the higher muscle lactate concentration at 60 s of occlusion compared to at 10 s. The ischemic recovery thus induced a greater metabolic disturbance and, as such, it should be expected that type III and IV afferent firing was at the very least maintained if not increased during this period (Jankowski et al., 2013; Laurin et al., 2015). As the cuff was deflated only immediately before the beginning of the sprints performed either 10 or 60 s after the incremental maximal test, a poorer sprint performance was expected after the 60 s recovery period compared to the 10 s one. Surprisingly, not only was the power developed after 10 s higher than the maximum power reached at task failure of the preceding incremental exercise test, the sprint after 60 s of ischemic recovery reached higher power than the one after 10 s. These findings suggest that despite a *milieu interne* expected to strongly stimulate type III-IV afferents, motor drive was not inhibited as much during the 10 s all-out sprints as compared to at task failure of the incremental test. Even though evoked force loss was not directly quantified in this study, nor were the experiments repeated after intrathecal fentanyl injection, the results nevertheless question a universal critical peripheral threshold hypothesis, at least for 10-s long all-out sprints.

Further evidence that questions the critical peripheral threshold comes from the different extents of evoked peak twitch reductions found at task failure from a cycling bout performed at 80% of peak power in different studies. Goodall et al. (2012) reported an average reduction of 20%, Sidhu et al. (2014) of 46%, and Amann et al. (2011) of 34%, even though all three studies used an identical exercise task. One possible explanation for the lesser peak twitch decrease found at task failure by Goodall et al. (2012) than by Amann et al. (2011) could be that task failure can occur before reaching a critical peripheral fatigue. This possibility is supported by recent results obtained by Johnson et al. (2015), who reported that the extent of evoked force loss at task failure of a cycling bout was less when realized after prior upper body exercise. It is further possible that such differences between studies reporting reductions in evoked force might be explained by differences in the characteristics of the participants involved (e.g., different training status and other inter-individual variability). On the other hand, supposing that different participant cohorts were involved in each of their studies, it is striking how the different studies conducted by Amann and colleagues consistently found a reproducible peak twitch force loss. Nevertheless, given the above mentioned discrepancies, it would seem that the critical peripheral fatigue threshold hypothesis based on "interindividual consistency of the degree of end-exercise fatigue" (Broxterman et al., 2015) needs further testing.

To foster the debate we here review a series of recent studies in which losses in MVC and evoked forces as well as changes in voluntary activation level (VAL) induced by a fatiguing effort were quantified (**Table 1**). Studies were included if they involved exercise tasks where the subjects were asked to continue for as long as possible, i.e., until task failure, and quantified MVC and evoked force losses. The tasks varied from isometric contractions with various muscle groups to dynamic exercise tasks such as cycling at a fixed power output until task failure. There clearly are discrepancies in the extent of evoked force losses following a given exercise (see **Table 1**). However, attention should be paid to the time point at which evoked forces were evaluated in

TABLE 1 | Summary of studies that quantified maximal voluntary contraction (MVC), evoked force and voluntary activation level (VAL) changes after various exercise.

Study	Muscle	Fatiguing task	Post evaluation at	TTF s	MVC loss %	Evoked force loss %	VAL loss %
Matkowski et al., 2011	KE	20% MVC to TF one leg	not specified	295	−37	−24[a]	−13
Matkowski et al., 2011	KE	20% MVC to TF two legs	not specified	245	−26		−7
Neyroud et al., 2012	KE	20% MVC to TF	at TF	246	−51	−37[a]	−7
Place et al., 2005	KE 35°	20% MVC to TF	20–30 s	974	−28	−3[a]	−19
Place et al., 2005	KE 75°	20% MVC to TF	20–30 s	398	−28	−4[a]	−14
Place et al., 2007	KE	40% MVC to TF	20–30 s	101	−16	−3[a]/−15[b]	−6
Kalmar and Cafarelli, 1999	KE	50% MVC to TF Pla	immediately after TF	66	−30	−55[b]	
Kalmar and Cafarelli, 1999	KE	50% MVC to TF Caf	immediately after TF	82.5	−30	−55[b]	
Neyroud et al., 2013	KE	50% MVC to TF	at TF	77	−34	−28[a]/−36[b]	−5
Amann et al., 2006	KE	cycling at 83% peak power to TF in Nx	2 min 30	489	−9	−24[a]/−34[b]	
Amann et al., 2006	KE	cycling at 83% peak power to TF in Hx	2 min 30	270	−11	−23[a]/−32[b]	
Amann et al., 2006	KE	cycling at 83% peak power to TF in hyperoxia	2 min 30	1162	−9	−24[a]/−32[b]	
Amann et al., 2011	KE	cycling at 80% peak power to TF	3 min	522	−10	−34[b]	−1
Amann and Dempsey, 2008	KE	cycling at 83% peak power to TF	4 min	∼600	−10	−36[b]	0
Goodall et al., 2012	KE	cycling at 80% peak power to TF in Nx	2 min 30	486	−17	−19[b]	−9
Goodall et al., 2012	KE	cycling at 80% peak power to TF in Hx	2 min 30	216	−25	−30[b]	−18
Sidhu et al., 2014	KE	cycling at 80% peak power to TF	∼40s	588	−16	−46[b]	−10
Rupp et al., 2015	KE	40% MVC to TF in Nx	immediately after TF	458	−18	−10[a]/−18[b]	−9
Rupp et al., 2015	KE	40% MVC to TF in Hx	immediately after TF	449	−16	−6[a]/−16[b]	−12
Neyroud et al., 2013	PF	50% MVC to TF	at TF	220	−30	−7[a]/−1[b]	−13
Yoon et al., 2007	EF	20% MVC to TF men	immediately after TF	636	−17	−23[b]	−10
Yoon et al., 2007	EF	20% MVC to TF women	immediately after TF	1020	−32	−33[b]	−17
Yoon et al., 2008	EF	20% MVC to TF young	immediately after TF	864	−27	−28[b]	−14
Yoon et al., 2008	EF	20% MVC to TF old	immediately after TF	1770	−38	−33[b]	−13
Neyroud et al., 2013	EF	50% MVC to TF	at TF	72	−40	−59[a]/−72[b]	−6
Yoon et al., 2007	EF	80% MVC to TF men	immediately after TF	25	−16	−37[b]	−4
Yoon et al., 2007	EF	80% MVC to TF women	immediately after TF	24	−15	−29[b]	−6
Yoon et al., 2008	EF	80% MVC to TF young	immediately after TF	24	−15	−33[b]	−4
Yoon et al., 2008	EF	80% MVC to TF old	immediately after TF	32	−9	−18[b]	−2
Fuglevand et al., 1993	FDI	20% MVC to TF	∼30 s	534	−40	−55[a]	
Fuglevand et al., 1993	FDI	35% MVC to TF	∼30 s	246	−30	−54[a]	
Fuglevand et al., 1993	FDI	65% MVC to TF	∼30 s	66	−19	−10[a]	
Neyroud et al., 2013	ADD	50% MVC to TF	at TF	114	−37	−60[a]/−63[b]	−2

TTF, time to task failure; TF, task failure; Nx, normoxia; Hx, hypoxia; KE, knee extensors; PF, plantar flexors; ADD, adductor pollicis; EF, elbow flexors and FDI, first dorsal interosseous.
[a]Indicates that peripheral fatigue extent was measured by evoking a 100-Hz paired stimulation whereas [b]means that it was measured by evoking a single stimulation.

these studies, as the delay between the moment of task failure and the subsequent evaluation affects the extent of the latter because of recovery (Neyroud et al., 2012; Froyd et al., 2013). As highlighted in **Table 1**, at task failure from a 20% MVC sustained isometric contraction of the knee extensors, reductions in evoked forces varied from 3% in Place et al. (2005) to 24% in Matkowski et al. (2011) even though a similar break (∼ 20 s) was provided between task failure and the electrically-evoked contraction. Although single and paired stimuli are classically used as indexes of peripheral fatigue, it should be mentioned that the number of stimuli delivered might affect the factors constraining evoked force production (Parmiggiani and Stein, 1981). To sum up, in light of these studies, and given MVCs performed with similar levels of voluntary activation rates, the existence of a critical peripheral fatigue threshold does not appear obvious.

COULD A MORE GLOBAL CRITICAL THRESHOLD BE INVOLVED IN TASK FAILURE?

Alternatively, the extant literature on neuromuscular fatigue appears compatible with the existence of a more global critical threshold based on MVC force loss. Indeed, in view of the different studies reported in **Table 1**, it appears that at

failure of tasks realized at a given intensity, MVC force losses were similar. A MVC force loss of ∼30–40% seems to be consistently observed following sustained isometric contractions performed with the knee extensors at 20% MVC when the MVCs were realized a few seconds after task failure (Place et al., 2005; Matkowski et al., 2011). Similar MVC force losses were found when a similar task was performed with the elbow flexors (Yoon et al., 2007, 2008). This suggests that MVC force loss might be tightly regulated to not exceed a certain threshold.

Similarly to what was observed for evoked forces, the time at which MVC force loss is assessed affects its extent (Neyroud et al., 2012; Froyd et al., 2013). Yet, if results from studies evaluating MVC force loss at a similar time delay after task failure are compared, then its extent appears to be rather consistent. When sustained isometric contractions are performed, MVC force losses also appeared consistent between the various studies for a given intensity (c.f. **Table 1**). Interestingly, a similar MVC force loss was found following a 50% MVC sustained isometric contraction performed to task failure with four different muscle groups, whereas reductions in evoked forces differed (Neyroud et al., 2013). Again, these results would support a tight regulation of MVC force loss. Accordingly, it can be hypothesized that instead of being limited by the extent of peripheral fatigue (reflected by reductions in evoked forces), exercise termination might result from a certain degree of MVC force loss being reached, the latter being exercise-intensity dependent.

However, although this new hypothesis appears seductive, close examination of individual data shows that such a critical global threshold based on MVC force loss does not hold at the individual level and rather seems a mere artifact of data aggregation. For example, considering the data obtained by Place et al. (2005) following a sustained isometric contraction at 20% MVC to task failure performed with the knee extensors at two different knee angles (35° vs. 75° of knee flexion), it appeared that averaged MVC force losses were similar between the two tasks (−28 ± 16 and −28 ± 19% following the 35° and 75° task, respectively). Yet, when considering individual values, it can be seen that MVC force loss was greater following the 35° task than the 75° one in four of the nine participants, whereas the opposite was found in the five other participants (**Figure 1A**). It thus appears that the similar averaged MVC force loss observed in this study between the two different tasks was a mere reflection of half of the participants showing one behavior and the other half behaving in the opposite manner. A similar finding is observed from the results obtained in a study comparing MVC force loss induced by a sustained isometric contraction at 50% MVC to task failure performed with four different muscle groups (Neyroud et al., 2013, **Figure 1B**).

The similar reduction in evoked forces reported between the two fatiguing tasks performed in Place et al. (2005) can also be

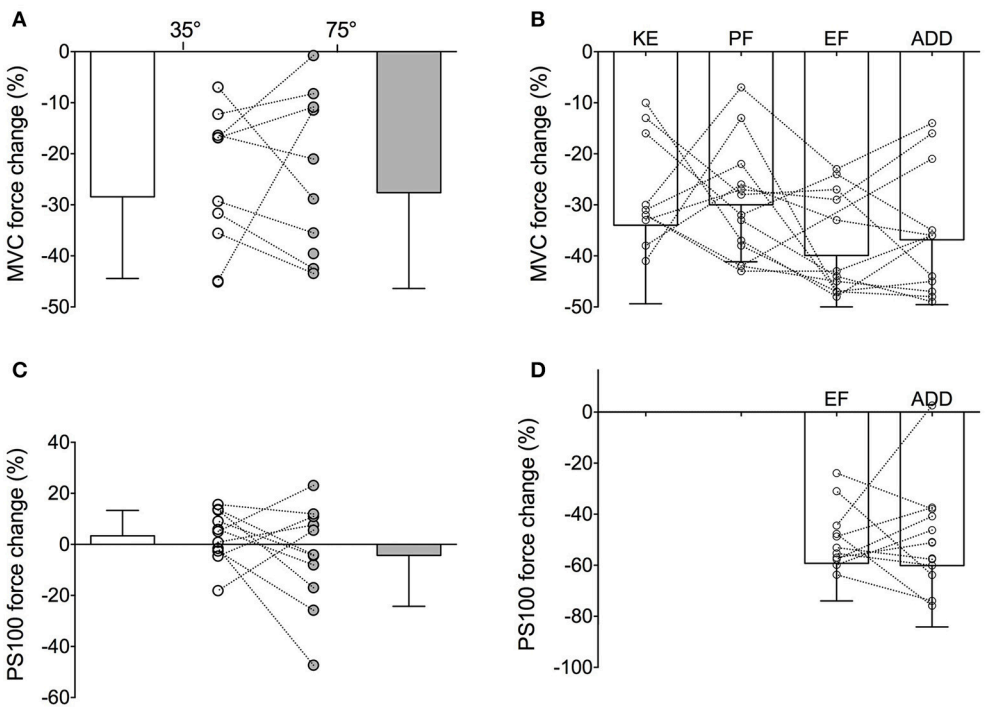

FIGURE 1 | Maximal voluntary contraction (MVC) force and 100-Hz evoked (PS100) force changes at task failure of (i) (A,C) a 20% MVC sustained isometric contraction performed with the knee extensors at knee angles of 35° (unfilled) and 75° (filled) of knee flexion and (ii) (B,D) a 50% MVC sustained isometric contraction performed with four muscle groups. For the illustration of the present purpose, PS100 force loss was only represented for the elbow flexor and *adductor pollicis* tasks in panel D as peripheral fatigue extent differed in the two other muscle groups. KE, knee extensors; PF, plantar flexors; EF, elbow flexors and ADD, *adductor pollicis*.

ascribed to cancelation of two opposite behaviors shown by the participants (some showing a greater evoked force reduction after the 35° task whereas evoked forces were reduced to a greater in extent in some others after the 75° task, **Figure 1C**). Similarly, in Neyroud et al. (2013), the evoked force loss measured at failure of the exercise involving elbow flexors and the *adductor pollicis* was similar, whereas a look at the individual values (**Figure 1D**) clearly showed that half the participants displayed greater reductions following the elbow flexor task and the other half following the *adductor pollicis* task.

As a critical threshold (global or peripheral) would be physiologically relevant only at the individual level, the above mentioned observations highlight the importance of considering interpretation of individual data and not only of group means, despite statistics. Indeed, when group means are compared and models/theories inferred from them (such as done when results of several studies are put together and new interpretations are inferred), caution should be taken to avoid drawing wrong conclusions by making ecological errors [i.e., deducing inferences about individual data from group averages (Sheppard, 2003)]. Models aiming to explain task failure and exercise performance should therefore be inferred from individual data and not averaged ones. However, for that to be possible, future studies should consider presenting both mean and individual data as proposed in **Figure 1**. Adopting such a manner of presenting results might lead to better comprehension of the mechanisms regulating exercise performance and responsible for task failure as both individual and mean data would be available to the reader.

AUTHOR CONTRIBUTIONS

All authors listed, have made substantial, direct and intellectual contribution to the work, and approved it for publication.

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Differences in Muscle Oxygenation, Perceived Fatigue and Recovery between Long-Track and Short-Track Speed Skating

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Due to the technical nature of speed skating, that is affecting physiological mechanisms such as oxygenation and blood flow, this sport provides a unique setting allowing us to uncover novel mechanistic insights of the physiological response to exercise in elite middle-distance and endurance sports. The present study aimed to examine the influence of skating mode (short-track vs. long-track) on muscle oxygenation, perceived fatigue, and recovery in elite speed skating. Muscle oxygenation of 12 talented short-track speed skaters was continuously monitored during a long-track (LT) and a short-track (ST) skating time-trial of maximal effort using near-infrared spectroscopy (NIRS) on the m. vastus lateralis for both legs. Video captures were made of each testing session for further interpretation of the muscle oxygenation. To determine recovery, perceived exertion was measured 2 and 4 h after each testing sessions. Repeated measures ANOVA's were used for statistical analysis ($p < 0.05$). After a rapid desaturation in both legs directly after the start, an asymmetry in muscle oxygenation between both legs was found during LT (tissue saturation-index (TSI%)-slope: left $= 0.053 \pm 0.032$; right $= 0.023 \pm 0.020$, $p < 0.05$) and ST speed skating (TSI%-slope: left $= 0.050 \pm 0.052$, right $= 0.001 \pm 0.053$, $p < 0.05$). Resaturation of the right leg was relatively lower in ST compared to LT. For the left leg, no difference was found between skating modes in muscle oxygenation. Respectively, two ($ST = 5.8 \pm 2.0$; $LT = 4.2 \pm 1.5$) and 4 h ($ST = 4.6 \pm 1.9$; $LT = 3.1 \pm 1.6$) after the time-trials, a higher rate of perceived exertion was found for ST. Based on our results, ST seems more physiologically demanding, and longer periods of recovery are needed after training compared to LT. Technical aspects unique to the exercise mode seem to impact on oxygenation, affecting processes related to the regulation of exercise intensity such as fatigue and recovery.

Keywords: near-infrared spectroscopy, elite athletes, training, endurance, winter sport

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INTRODUCTION

Speed skating is an intriguing sport to study, in particular because speed skaters need to adopt the biomechanically favorable crouched position that is essential for speed skating performance (Van Ingen Schenau et al., 1983; Konings et al., 2015). This crouched position combined with a relatively long gliding phase and high intramuscular forces at the same time leads to a physiological disadvantage as it is suggested to increase deoxygenation of the

working muscles (Rundell, 1996; Rundell et al., 1997; Konings et al., 2015). The presence and magnitude of deoxygenation during speed skating has been investigated using near infrared spectroscopy, demonstrating a linear relationship with knee-angle in long track speed skating (Foster et al., 1999) and an asymmetry between the legs in short track skating, particularly related to cornering (Hesford et al., 2012, 2013a,b). Most likely, the reduced blood flow to the working muscles reduces the aerobic capacity of the recruited muscle groups (Rundell, 1996), whereas not all the available oxygen can be transported toward the working muscles during speed skating. The reduced blood flow is expected to exacerbate processes related to perceived fatigue and recovery, thereby impacting on elite performance. Due to the technical nature of speed skating that is affecting physiological mechanisms such as oxygenation and blood flow, this sport provides a unique setting allowing us to uncover novel mechanistic insights into these aspects of the physiological response to exercise in elite sports.

Changes in muscle oxygenation can inform on a drop in tissue pO_2 and therefore potentially correlate with fatigue and performance. Global hypoxia decreases arterial oxygenation and can exacerbate peripheral fatigue and fatigability (Katayama et al., 2007; Romer et al., 2007). Local ischemia leading to a 7% or larger reduction in muscle oxygenation can lead to decreased muscle production in the fore-arm muscles (Murthy et al., 2001). Increased intramuscular pressure has been suggested to decrease blood flow and thereby reduce tissue oxygenation at isometric contractions >25–35% of maximal torque capacity in upper leg muscles (de Ruiter et al., 2007). Under these conditions the muscle fibers will largely depend on glycolysis as an energy source, potentially exacerbating fatigue.

Long track and short track speed skating are expected to differ in terms of asymmetry between the legs, associated with the technical nature and characteristics of the different sports. In our communication with coaches and athletes, we became aware that when imposing testing sessions of similar load in short-track (ST) and long-track (LT) skating to elite ST athletes, the athletes felt more fatigued after ST compared to LT skating sessions. As short track athletes compete on a shorter track with shorter straights and more and tighter curves during their race, differences in intra-muscular pressure, aerobic physiology and thereby experienced fatigue and recovery could be expected.

Therefore, the aim of the current study was to measure the influence of skating mode (short-track vs. long-track) on muscle oxygenation, and determine how this affects perceived fatigue and recovery in speed skating. The crouched position of speed skaters leads to a unique form of physiological stress: static occlusion of blood flow that occurs repetitively with every cycle, that might hinder recovery related to oxygenation (Rundell, 1996; Rundell et al., 1997; Foster et al., 1999). In short track speed skating, with tighter corners, longer periods of occlusion are seen for the right leg in particular (Hesford et al., 2012, 2013a,b), and we expect this will impact on recovery and perceived fatigue. It is hypothesized that the working muscles will remain more deoxygenated during short-track speed skating, associated with the relatively long periods of static contraction, and that there is thereby a larger asymmetry between the left and right leg. It is expected that this will lead to higher experienced fatigue, and longer periods of recovery after short-track speed skating compared to long-track speed skating. If a link between muscle oxygenation and fatigue is clear for the same elite athletes in a single sport (speed skating) depending only on the mode of the exercise, this suggests that measures of muscle oxygenation could inform in constructing training and recovery loads in other sports or activities involving local ischemia, even ones that do not have a specific asymmetric component.

MATERIALS AND METHODS

Participants

Twelve talented elite short-track speed skaters (11 males, 1 female) from the Dutch national U-23 short-track speed skating team (age = 19,7 ± 2,6 year, height = 179,8 ± 6,5 cm, mass = 71,0 ± 6,4 kg) participated in this study. All were also familiar with long-track speed skating, in which they engaged on a weekly basis as part of their training regime. All participants, and also their parents if they were <18 years, gave their written informed consent before participation. This study was approved by the local ethics committee (University of Essex, UK) and in accordance with the Declaration of Helsinki.

Experimental Procedures

Testing took place on a long-track (400 m) and short-track (111.12 m) speed skating oval both approved for international competition and at the same venue. Participants was asked to complete two testing conditions: a short-track time trial and a long-track time trial of comparable intensity, counter-balanced with a time interval of a week separating the two trials. All measurements took place on Tuesday mornings (08 h 30–10 h 30). The order of the testing conditions was counterbalanced between participants and separated by 1 week for each participant. Before each testing condition, participants performed their own warm-up, as they would when preparing for a normal training session.

In each testing condition participants had to complete a time-trial with flying start as fast as possible, after a warm-up designed by the coach. Warm-up and time-trial were separated by 10 min of active rest. All time trials were completed individually. In one testing condition the time-trials involved two laps on a long-track oval (800 m), in the other six laps on a short-track oval (total trial length: 666 m). All the testing sessions were set up in cooperation with the Dutch national short-track coaches. Long-track and short-track testing sessions were set up in such a way that they were of an equal expected physiological intensity (i.e., maximal) to make the different skating modes comparable, whilst interfering as little as possible with the coach and the usual training set-up to ensure an high ecological validity and practical relevance.

The muscle oxygenation of both legs was monitored continuously using NIRS during all time-trials. To quantify the physiological intensity of the time-trials heart rate was measured during the time-trials, in combination with the mean lactate values and rate of perceived exertion directly after the time-trials. Furthermore, mean duration, velocity, acceleration, and VO_2

were determined during the time-trial in order to assess if they were related to the oxygenation at local muscular level. Lastly, two relevant technical variables for speed skating, the knee- and trunk angle respectively, were determined during the time-trials, as the knee angle was found to be related with the extent of deoxygenation in speed skating (Foster et al., 1999).

To determine perceived fatigue and recovery, first of all, rates of experienced exertion were collected after each testing session, as well as after 2 h and 4 h after completion of the testing condition. Secondly, before and after each testing condition the participants performed a 6-s maximal cycle test followed by 30-s of rest. The first 6-s maximal cycle test was completed before the warm-up. The time-interval between completion of the testing condition and the second 6-s maximal cycle test was controlled for both trials, and did not exceed 5 min. In this 30-s rest period, recovery in muscle oxygenation for this single sprint was determined in order to quantify their recovery after each testing condition.

Muscle Oxygenation Measurements

A wireless spatially resolved spectroscopy (SRS) dual-wavelength oximeter (Portamon; Artinis Medical Systems, BV, The Netherlands) was used to measure absolute and relative changes in NIRS parameters. The Portamon has previously been used in speed skating in order to investigate muscle oxygenation and hemodynamics (Hesford et al., 2012, 2013a,b). The unit is self-contained and compact, measuring 83 × 52 × 20 mm and weighs with battery 84 g. The Portamon has three pairs of light-emitting diodes that emit light of wavelengths 760 and 850 nm and are positioned 30, 35, and 40 mm from the detector. With these wavelengths, changes in concentration of the chromophores hemoglobin (Hb) and myoglobin (Mb) can be detected. Although using a two-wavelength spectrometer allows a measurement of changes in the concentration of oxygenated and deoxygenated species discrimination between the oxygenated and deoxygenated forms of respectively Hb and Mb cannot be made. Therefore, the tissue oxygenation changes reported will contain contributions from both chromophores, although the myoglobin contribution is likely to be smaller (see discussions in Hesford et al., 2012, 2013a,b; Born et al., 2014) and was ignored for the purposes of this discussion.

Single distance continuous wave dual wavelength NIRS can only report on changes in chromophore concentrations. However, multi distance systems such as the Portamon device can take advantage of SRS. SRS enables biasing of the NIRS data away from superficial layers (skin/adipose) layers in favor of deeper (muscle) changes, whilst at the same time providing an absolute measure of tissue oxygen saturation TSI% (Suzuki et al., 1999). During the time-trials, NIRS was used to measure continuously changes in total muscle hemoglobin (tHb), muscle oxyhemoglobin (HbO_2), muscle deoxyhemoglobin (HHb) and muscle oxygen saturation (TSI%) in both left and right m. vastus lateralis. A baseline was determined based on 30 s averaging before participants went on-ice to start their TT. All chromophore concentration changes were presented relative to this baseline (Jones et al., 2015). Video captures of the testing sessions was used to match NIRS changes with the position and movement of the skaters.

The Portamon devices were placed on the belly of the musculus vastus lateralis on both legs, midway between the lateral femoral epicondyle and the greater trochanter of the femur. The devices were fixed into position using surgical tape, in order to ensure the optodes and detector did not move relative to the participant's skin. Precise and consistent optode placement was crucial, as quadriceps muscle oxygenation was shown to be non-uniform during exercise (Quaresima et al., 2001; Kime et al., 2005; Kennedy et al., 2006). To ensure that the NIRS measurements were not influenced by environmental light, participants wore a black bandage attached over both the Portamon devices. All participants reported that the devices in combination with the bandages did not restrict their movements in any way.

Finally, NIRS was used to examine differences in recovery between both skating modes. To measure differences in recovery after the testing conditions, participants performed a 6-s maximal cycle test before and after each testing session followed by 30-s of rest. The tests took place on a Wattbike Pro (Wattbike Ltd., UK) and participants were allowed to choose their own saddle height before their first test. During both testing conditions, saddle height was kept constant and participants were instructed to stay seated straight on the Wattbike during the 30-s of rest. Peak power output (PPO) was the highest recorded power output during each sprint. Values for ΔTSI% are reported as a change from baseline (5 s averaging before the sprint). The magnitude of the drop in ΔTSI% during the 6-s sprint was determined as the difference in TSI% between baseline and the lowest 0.5-s average during the sprint. Recovery was determined based on the half-time recovery in ΔTSI% after the 6-s sprint. Halftime recovery was defined phenomenologically as the time it took before TSI% recovered to half of the difference between the initial baseline value and the lowest TSI value.

Rate of Perceived Exertion Measurements

Participants were asked for their rates of perceived exertion (RPE) on a Borg CR10 scale directly after each testing condition (Borg, 1998). To monitor the (experienced) recovery after each testing condition, participants was also asked to report their level of fatigue on a Borg CR10 scale 2 and 4 h after each testing session, respectively. All participants were familiar with using the Borg scale, whereas the Borg scale was already used regularly after testing sessions before the start of the measurements.

Global Physiological Measurements

Respiratory gases were measured during each testing session using a wearable and wireless breath-by-breath pulmonary gas analyzer (Metamax 3B, Cortex, Germany) previously demonstrated to be valid and reliable for measuring oxygen uptake (Macfarlane and Wong, 2012). The Metamax 3B was calibrated each day a measurement took place using a Jaeger 3L syringe. For each the test an ambient air measurement was also performed. As the oxygen use and transport adaptations require some time, and thus lead to a lag in the aerobic response (Hagerman, 2000), only the last 30 s of the time-trial were

averaged, resulting in a mean oxygen uptake value (VO_2 mean). Additionally heart rate was monitored during the testing sessions by a validated portable heart rate monitor (Zephyr Bioharness, Zephyr Technology, New Zealand; Franklin and Brooks, 2012).

Velocity Measurements

Position, velocity and acceleration of the participants were monitored in all time-trials by the Local Positioning Measurement (LPM-) system (Inmotio Object Tracking BV, The Netherlands). The LPM-system is a high frequency (1000 Hz), radio-frequency based technology. Participants wore a vest containing a transponder located on the back that was connected to two antennas, one on top of each shoulder. To understand how the skaters' movements affected the differing physiological demands during one lap, positional data collected by the LPM system were synchronized with the data collected by NIRS. For the purpose of this analysis two separate elements (which are all repeated twice per lap) were defined and analyzed:

(1) Straight: the section of the lap between the two corners. The straight starts when the left blade touches the ice after the crossover of legs while exiting the corner. The straight contains in short-track speed skating one glide on each blade and ends when the left blade touches the ice after the right foot glide. During long-track speed skating the straight is characterized by several strokes containing a gliding phase, push-off, and repositioning phase (Konings et al., 2015).
(2) Corner: the phase between the end of the straight and the moment at which the skater's left blade touches the ice to begin the gliding phase of the straight the right blade touches the ice to begin the hang. This phase contains in short-track speed skating three subsections in the following order: an entry at which the skater performs respectively a series of leg crossovers, a hang in which the skater travels around the apex of the corner supported solely on the right blade, and an exit in which another series of leg crossovers while maintaining a very low position were performed (Hesford et al., 2012). In contrast, in long-track speed skating a series of leg crossovers is conducted when cornering.

Technical Measurements

The trunk- and knee angle are two crucial technical characteristics for speed skating performance (Konings et al., 2015). Participants were filmed at the straights during their testing conditions with one digital high-speed camera to assess their knee and trunk angles. The camera was placed perpendicular to the sagittal plane of the speed skater during all sessions at both speed skating tracks. To determine the trunk- (line between neck-hip and horizontal) and knee angle (between upper and lower leg) the recordings when the subject was in his gliding phase were analyzed.

Lactate Measurements

Within 1 min after completion of the second time-trial, the lactate value of the participants was measured using Lactate Pro (Arkray Inc, Japan) by using capillary blood from the finger after a prick in the finger. Lactate measurements were performed by an experienced test leader.

Data-Analysis and Statistics

All statistical analyses were performed using SPSS version 19.0. For statistical analysis the slope in ΔTSI%, ΔHbO_2, ΔHHb, and ΔtHb over time after the initial desaturation was determined for each subject. Additionally to the NIRS measurements, the positional data and video captures of the testing conditions collected using the LPM-system were used for further detailed analysis of the muscle oxygenation during the testing condition. For all variables, mean and SD were determined and used for further statistical analysis. Statistical comparisons of each measured variable between short-track and long-track skating were made by using repeated measures ANOVA. In addition, comparisons between both legs for each testing condition were performed for ΔTSI%, ΔHbO_2, ΔHHb, and ΔtHb in order to examine the occurrence of an asymmetry in blood flow to the working muscles. Finally, absolute values of TSI% of both legs in both skating modes were determined during the final three strokes of the time trial. Descriptive statistics are presented as means $\pm$ SD unless otherwise noted. The statistical significance was defined at $P < 0.05$.

RESULTS

Means $\pm$ SD of the time-trial performance for the long-track time-trials (LT) and the short-track time-trials (ST) can be found in **Table 1**. Faster completion times ($LT = 63.45 \pm 2.20$, $ST = 60.94 \pm 2.90$ s; $p = 0.005$) and a higher mean velocity ($LT = 44.36 \pm 1.97$, $ST = 38.26 \pm 1.46$ km/h; $p < 0.001$) were found for LT compared to ST. Nevertheless, no differences in heart rate ($LT = 178.8 \pm 5.8$, $ST = 179.6 \pm 6.6$ bpm; $p = 0.609$), lactate ($LT = 10.5 \pm 1.5$, $ST = 11.0 \pm 1.1$ mmol/l; $p = 0.356$) or RPE ($LT = 8.9 \pm 1.2$, $ST = 9.2 \pm 1.1$; $p = 0.555$) were found between the testing conditions, suggesting time-trials could be perceived as of a similar physiological intensity. No differences in knee angle were reported between conditions ($LT = 103.4 \pm 4.5$, $ST = 103.8 \pm 2.9^{\circ}$; $P = 0.843$). However, a lower trunk angle was found during ST ($LT = 16.6 \pm 1.1$, $ST = 15.3 \pm 0.9^{\circ}$; $p < 0.001$). In addition, deceleration of the participants was higher after ST compared to LT (see **Table 1** and **Figure 1**).

Changes in muscle oxygenation between both legs occurred in both LT as well as ST (see **Table 1** and **Figures 2–4**). Typical NIRS traces are shown for TSI (**Figure 2**) and HHb, HbO_2, and tHb (**Figures 3**, **4**). They illustrate that at the start of the race compared to baseline there is a fall in muscle oxygen saturation (TSI) caused by rise in HHb and fall in HbO_2. The fall in HbO_2 is larger than the rise in HHb, resulting in a consequent fall in tHb. As the TT progresses, muscle oxygenation (TSI) increases. This is caused entirely by an increase in HbO_2. There is no change in HHb, with the result that tHb increases along with HbO_2.

There is an asymmetry in muscle oxygenation between left and right legs as the TT progresses. **Table 1** shows the rate of change in time for the NIRS parameters. Muscle oxygenation significantly increases in the left leg compared to the right in both ST and LT. However, the difference is larger in ST than LT. These oxygenation changes are dominated by HbO_2 and not HHb. HbO_2 increases in both LT and ST with the larger differences

TABLE 1 | Means ± SD of the time-trial characteristics, the absolute TSI% values in the final three strokes of the time-trial and the slope of the recovery in muscle oxygenation throughout the time-trial for both long-track as well as short-track time-trials ($N = 12$).

	Means (*SD*)		*F*-value (sign.)
	Long-track	Short-track	
TIME-TRIAL CHARACTERISTICS			
Completion time (sec)	63.45 ± 2.90	60.94 ± 2.20	$P = 0.005$*
Velocity (km · h^{-1})	44.4 ± 2.0	38.3 ± 1.5	$P < 0.001$*
Acceleration (km · min^{-2})	−7.2 ± 2.9	−11.0 ± 2.5	$P = 0.005$*
VO_2mean (ml · kg^{-1} · min^{-1})	59.7 ± 3.6	57.8 ± 3.7	$P = 0.019$*
Lactate (mmol · l^{-1})	10.5 ± 1.5	11.0 ± 1.1	$P = 0.356$
Heart rate (min^{-1})	178.8 ± 5.8	179.6 ± 6.6	$P = 0.609$
ABSOLUTE TSI% IN THE FINAL THREE STROKES OF THE TIME-TRIAL			
Left leg	51.5 ± 5.9	50.8 ± 5.4	
Right leg	50.2 ± 4.0	46.4 ± 6.7	
Left LT vs. Left ST			$P = 0.405$
Right LT vs. Right ST			$P = 0.028$*
Left ST vs. Right ST			$P = 0.005$†
Left LT vs. Right LT			$P = 0.358$
SLOPE OF THE MUSCLE OXYGENATION RECOVERY DURING			
TIME-TRIALS ΔTSI% Slope (ΔTSI% · min^{-1})			
Left leg	5.3 ± 3.2	5.0 ± 5.2	
Right leg	2.3 ± 2.0	0.1 ± 5.3	
Left LT vs. Left ST			$P = 0.825$
Right LT vs. Right ST			$P = 0.049$*
Left ST vs. Right ST			$P = 0.001$†
Left LT vs. Right LT			$P < 0.001$†
ΔHbO_2 Slope (μM · cm · min^{-1})			
Left leg	227.2 ± 78.6	165.5 ± 35.5	
Right leg	200.1 ± 117.6	112.8 ± 61.6	
Left LT vs. Left ST			$P = 0.489$
Right LT vs. Right ST			$P = 0.004$*
Left ST vs. Right ST			$P = 0.017$†
Left LT vs. Right LT			$P = 0.507$
ΔHHb Slope (μM · cm · min^{-1})			
Left leg	5.6 ± 21.8	−4.9 ± 38.0	
Right leg	−13.1 ± 10.3	6.8 ± 45.1	
Left LT vs. Left ST			$P = 0.540$
Right LT vs. Right ST			$P = 0.151$
Left ST vs. Right ST			$P = 0.493$
Left LT vs. Right LT			$P = 0.087$
ΔtHb Slope (μM · cm · min^{-1})			
Left leg	102.8 ± 59.7	80.3 ± 26.4	
Right leg	107.0 ± 39.4	59.8 ± 15.1	
Left LT vs. Left ST			$P = 0.407$
Right LT vs. Right ST			$P = 0.008$*
Left ST vs. Right ST			$P = 0.012$†
Left LT vs. Right LT			$P = 0.842$

*Significant difference between skating mode ($p < 0.05$)
†Significant difference between both legs ($p < 0.05$).

being seen in ST. There is no significant change in HHb in either leg. An HbO_2 increase with no change in HHb is reflected in an increase in tHb. Again this increase is larger for the left leg than the right leg in ST compared to LT.

The use of spatially resolved methods enabled an absolute measurement of muscle oxygen saturation (TSI) at the end of the TT. The only difference between legs was seen for the right leg in ST. Muscle oxygenation in left ST, right ST and left ST were equivalent to one another and all higher than right ST.

The temporal changes in TSI, tHb, HbO_2, and HHb reveal significant differences in oxygenation in the course of a single lap. This can be illustrated by higher resolution analysis of a single half lap in LT (**Figure 5**) and ST (**Figure 6**). The lap was divided into segments indicated in both figures. An illustration of how these different segments relate to technique can be found in **Figure 7** (LT) and **Figure 8** (ST) During the straights in LT (**Figure 5**) the leg desaturates in the gliding phase and push-off (when the leg is on the ice), while the leg resaturates in the repositioning phase (when the leg is off the ice). In ST (**Figure 6**), the typical way of traveling (hanging) around the corner on only the right leg leads to a resaturation of the left leg and a desaturation of the right leg.

Figure 9 indicates the changes in muscle oxygenation during a 6-s sprint cycling test taken before and after the time trials. No differences were found in the drop of the ΔTSI% during the 6-s sprint between the legs, between the pre and post-test or between skating modes ($P > 0.05$; **Table 2**). The recovery halftime in ΔTSI% after the post 6-s sprint test was significantly increased after ST compared to LT. However, there were no differences in recovery rates between the legs for either skating mode (see **Table 2**). In addition, participants reported they felt more recovered 2 h ($P = 0.014$) and 4 h ($P = 0.026$) after LT compared to ST (**Table 2**).

DISCUSSION

The aim of this study was to examine the influence of speed skating mode (short-track vs. long-track) on muscle oxygenation in speed skating, and how it consequently affects processes of fatigue and recovery. In order to compare long-track and short-track speed skating, time-trials on both tracks of expected similar physiological intensity were set-up. This was done by asking the participants to perform each time-trial maximally. Although the long-track time-trials were significantly faster and longer in their duration, the lack of difference in heart rate during, and lactate value and rate of perceived exertion directly after the time-trials indicated that both skating modes were of a similar physiological intensity. A comparison of the values for heart rate, lactate, and RPE found in this study with other skating studies indicated that our subjects indeed performed maximally during the time-trials at both tracks (Rundell et al., 1997; Hesford et al., 2013b).

The TT started after a flying lap. The starting values for TSI, tHb, HHb, and HbO_2 therefore need to be compared to those seen after the first lap of our previous studies which used a standing start (Hesford et al., 2012, 2013a,b). The initial changes seen in our new short-track data are entirely consistent with our previous data (Hesford et al., 2012), i.e., an increase in HHb and a larger fall in HbO_2 resulting in a drop in TSI and tHb. The amount of hemoglobin in the optical field of view has decreased and what is seen has less oxygen bound. The

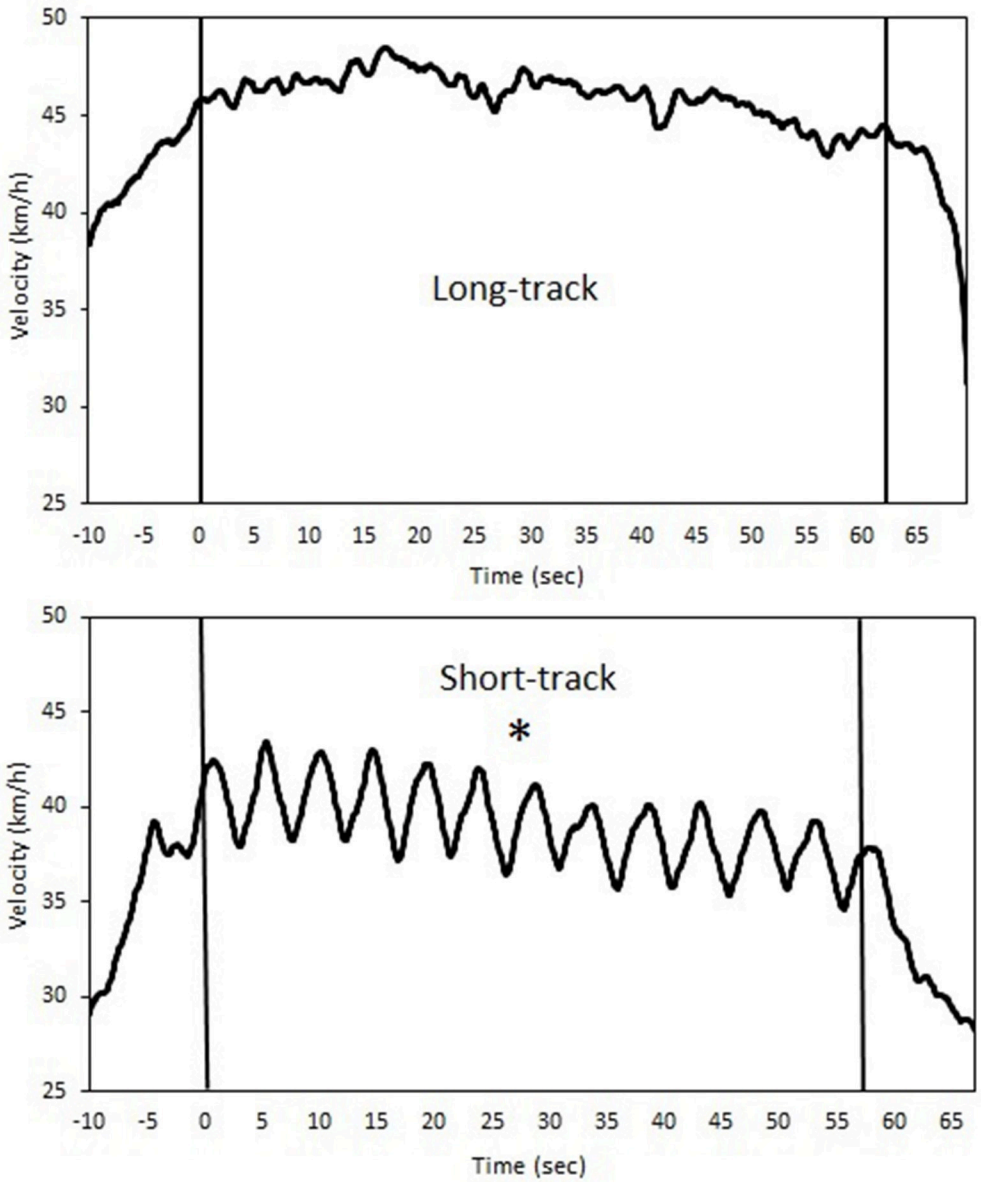

FIGURE 1 | A typical example of the velocity profiles during both long-track (upper panel) as well as short-track (lower panel) speed skating for a single time-trial (*indicates a significant difference between skating modes for velocity and acceleration, $p < 0.05$).

simplest explanation of this combination of NIRS data is that there is a decrease in blood volume flow and an increase in muscle oxygen consumption. The same trend is seen in the long-track data, indicating that here as well the combination of increased tissue oxygen demand and physical occlusion has caused a combination of vasoconstriction and increased oxygen consumption.

The two skating modes respond similarly after these initial effects. Consistent with our previous studies (Hesford et al., 2012, 2013a,b) there is an increase in muscle oxygenation with time. This is evidenced by a rise in TSI. However, this TSI increase is a result of an increase in HbO_2 alone. HHb is unaltered with the consequence that tHb increases. The amount of hemoglobin in the optical field of view has now increased and all of this increase is due to hemoglobin that has oxygen bound to it. The rate of oxygen extraction (HHb) does not change with time. The simplest explanation of this data is that the blood volume flow has increased but none of the extra arterial oxygen being delivered is consumed. In effect the blood vessels have expanded but this has not resulted in the muscles consuming more oxygen—the limitation is elsewhere. This does not of course mean that this vasodilation could not be physiologically relevant as it will also increase substrate flow to the muscle and the removal of metabolites.

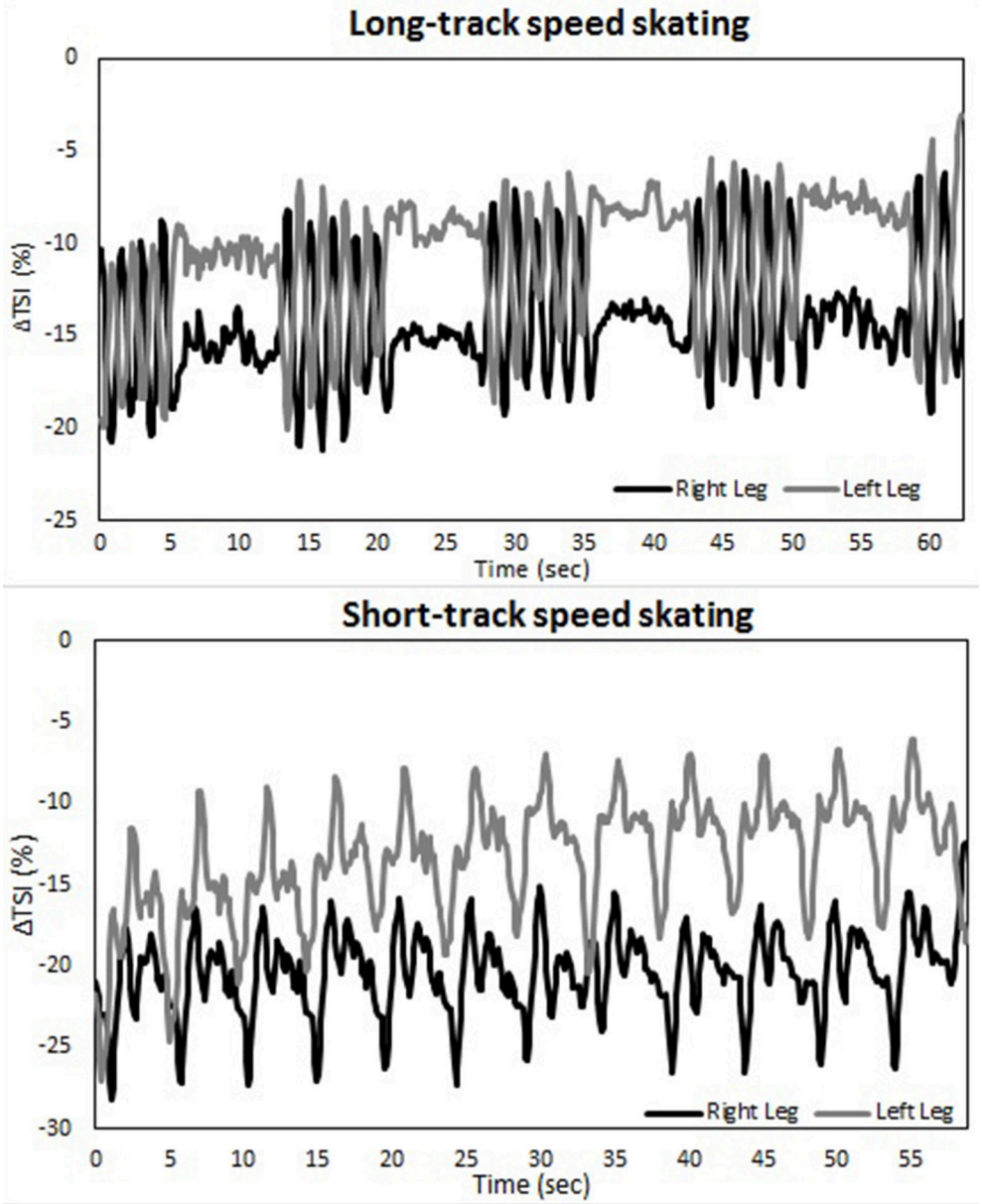

FIGURE 2 | A typical example of the Δ tissue saturation index (%) during both a long-track (upper panel) as well as a short-track (lower panel) time-trial for one subject.

Where the two skating modes do differ is in their quantitative response to these initial effects, in particular between the asymmetry of the two legs. As shown previously (Hesford et al., 2012), the rate of reoxygenation is faster in the left leg than in the right leg in short-track. However, this is not the case in long-track where no significant differences are seen. At the end if the TT the absolute muscle oxygenation is the same in the right and left legs in long-track and this value is identical to that in the left leg in short-track. However, the right leg in short-track is oxygen deprived compared to these three other states.

The reason for this asymmetry can be seen when exploring the oxygenation changes during a single lap. There are high intramuscular forces on the right leg when traveling around the corner in short-track skating, as the skater hangs on that right leg increasing deoxygenation. Consequently, the left leg that is off the ice, or at least carrying minimal weight, reoxygenates in the corners. In long-track speed skating, the NIRS measurements in combination with video captures showed a cyclic pattern of deoxygenation and reoxygenation during the straights. This pattern is consistent with the repetitive cyclic movement pattern that is executed in long track speed skating, consisting of a gliding

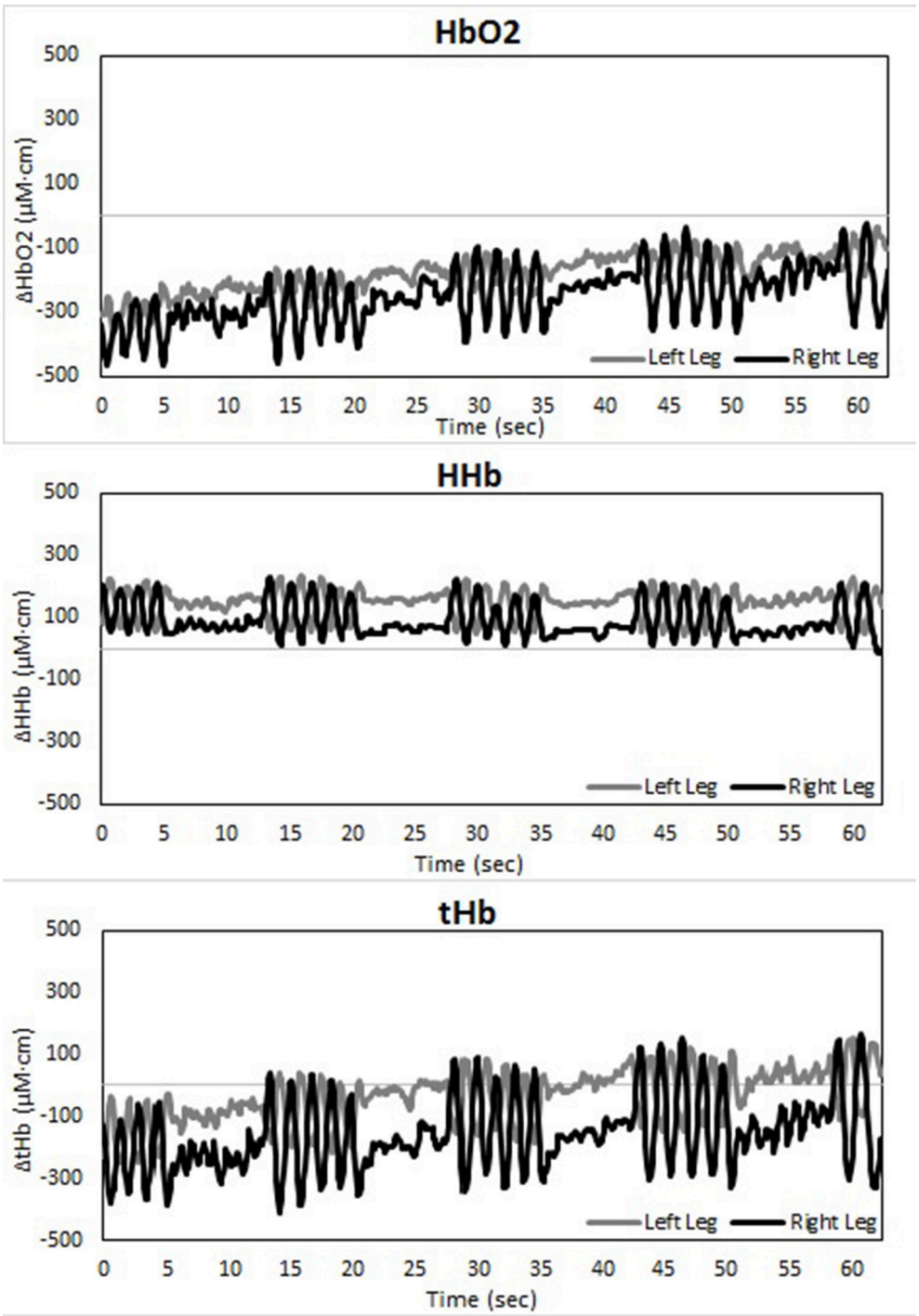

FIGURE 3 | Temporal changes in right and left vastus lateralis during the long-track time trial: HbO_2 (upper panel), HHb (middle panel), and tHb (lower panel).

phase, a push off phase and a swing phase (De Boer et al., 1987; Allinger and Van den Bogert, 1997; Konings et al., 2015). In the gliding phase and push-off phase, blood flow is occluded and deoxygenation occurs, while in the swing phase reoxygenation occurred. In the corners, a stable and consistent pattern in terms of oxygenation was visible in long-track skating: the difference between the legs is maintained and levels of oxygenation change very little. The movement pattern of leg crossovers in long-track speed skating was rhythmic, cyclic and consistent, and no long on-ice or off-ice phases are seen (**Figures 5**, **7**). Therefore, in

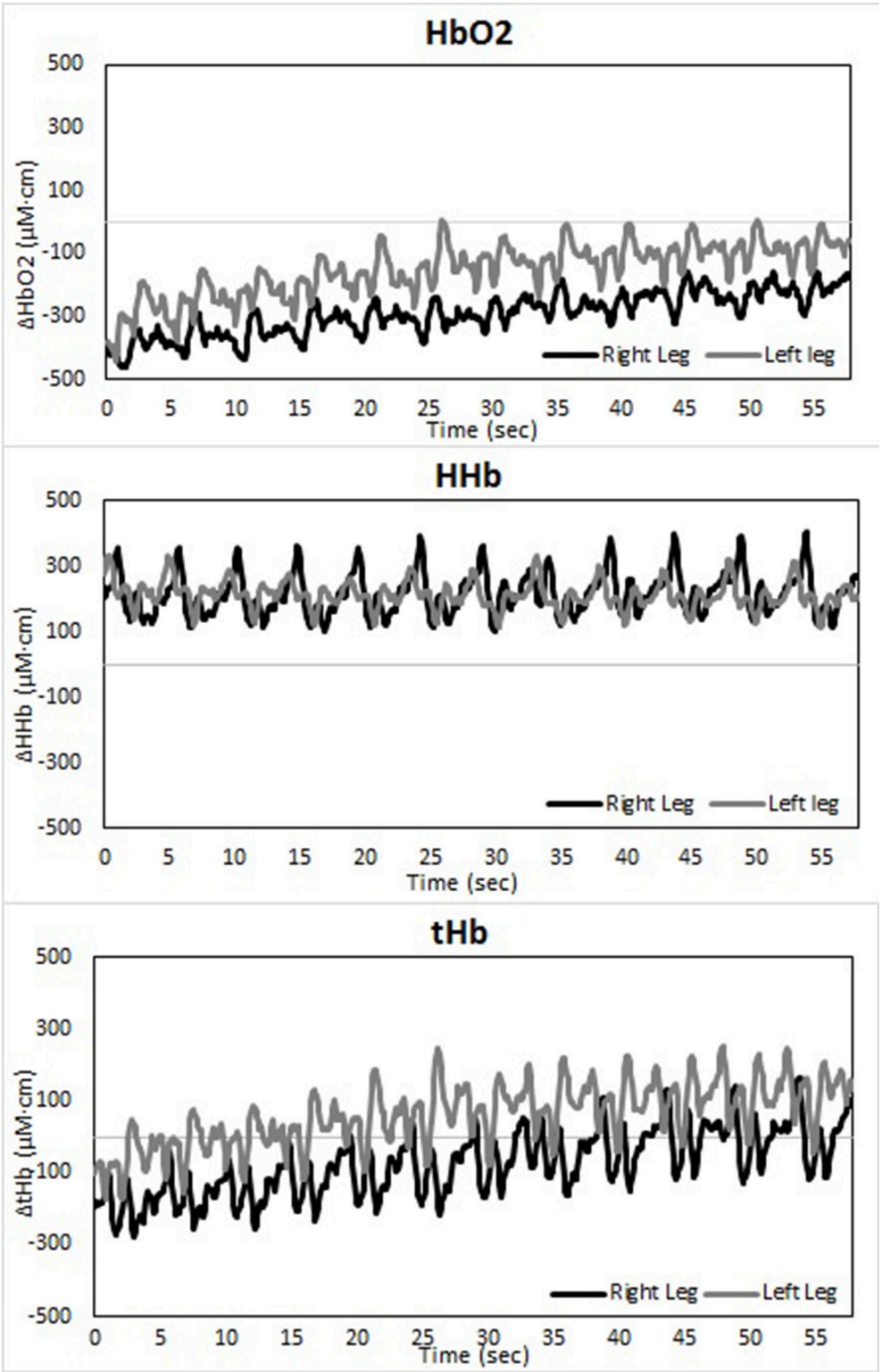

FIGURE 4 | Temporal changes in right and left vastus lateralis during the short-track time trial: HbO_2 (upper panel), HHb (middle panel), and tHb (lower panel).

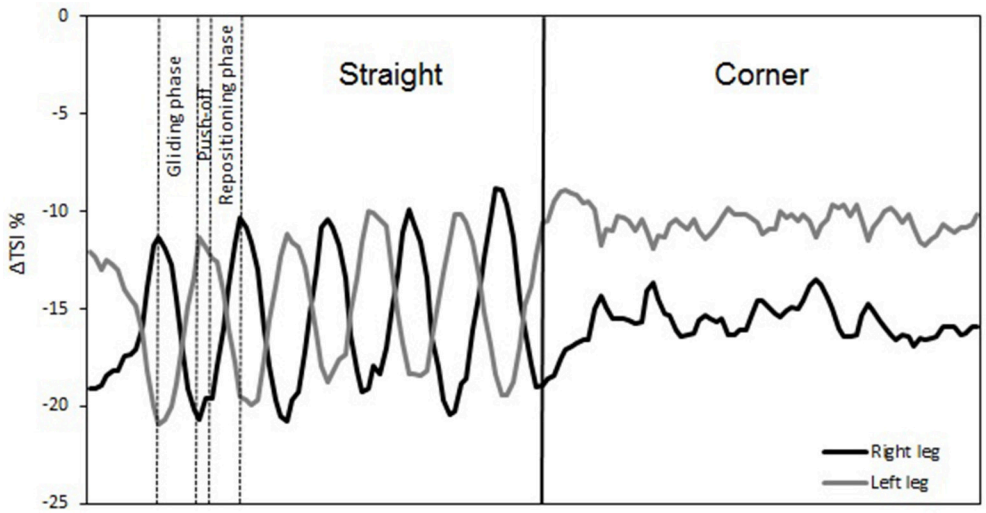

FIGURE 5 | Δ Tissue saturation index (%) in one lap during long-track speed skating. Straights in long-track speed skating are characterized by a repetitive cyclic pattern of three phases per leg: gliding phase, push-off (leg on-ice), and repositioning phase (leg off-ice). These three phases are remarked for a single stroke of the right leg. The corners are characterized by a series of leg crossovers.

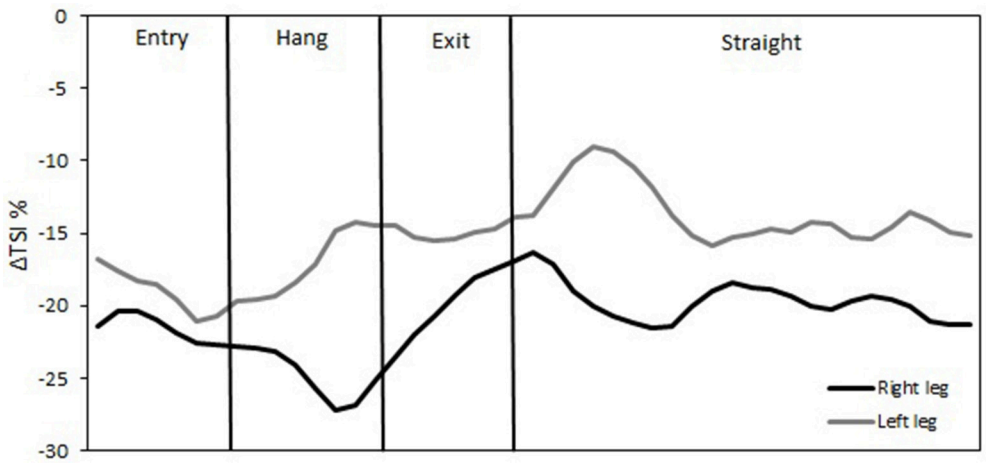

FIGURE 6 | Δ Tissue saturation index (%) in one lap during short-track speed skating. The straight in short-track speed skating contains one glide on each blade (both legs on-ice). The corner in short-track speed skating contains three subsections in the following order: an entry at which the skater performs leg crossovers, a hang in which the skater travels around the apex of the corner supported solely on the right blade, and an exit involving leg crossovers.

long-track deoxygenation largely occurred during the straight not the corners and the changes between the two legs were largely symmetrical. In contrast, in short track speed skating, a very clear asymmetry in reoxygenation occurred during the corners, with the left leg reoxygenating when off the ice and the right leg deoxygenating when on the ice throughout the corner (**Figures 6**, **8**).

Although an asymmetry in muscle oxygenation in long-track speed skating was reported before (Born et al., 2014), the current findings expand on these findings. That study reported that the difference in muscle oxygenation between both legs during the course of a 3000 m TT was only a result of a difference in the initial desaturation between both legs directly after the start (Born et al., 2014). In contrast, the present study shows that the asymmetry between both legs during long-track speed skating is also a result of a slightly enhanced reoxygenation rate in the left leg as the TT progressed (**Table 1**).

Our results agree with other findings in the literature, relating higher intramuscular pressure to hampered oxygenation and subsequent larger dependency on anaerobic energy supplies, exacerbating fatigue (de Ruiter et al., 2007; Katayama et al., 2007; Romer et al., 2007). In terms of the quantity of differences in muscle oxygenation, we found a reduction of more than 10% in TSI% over the whole test compared to baseline during both

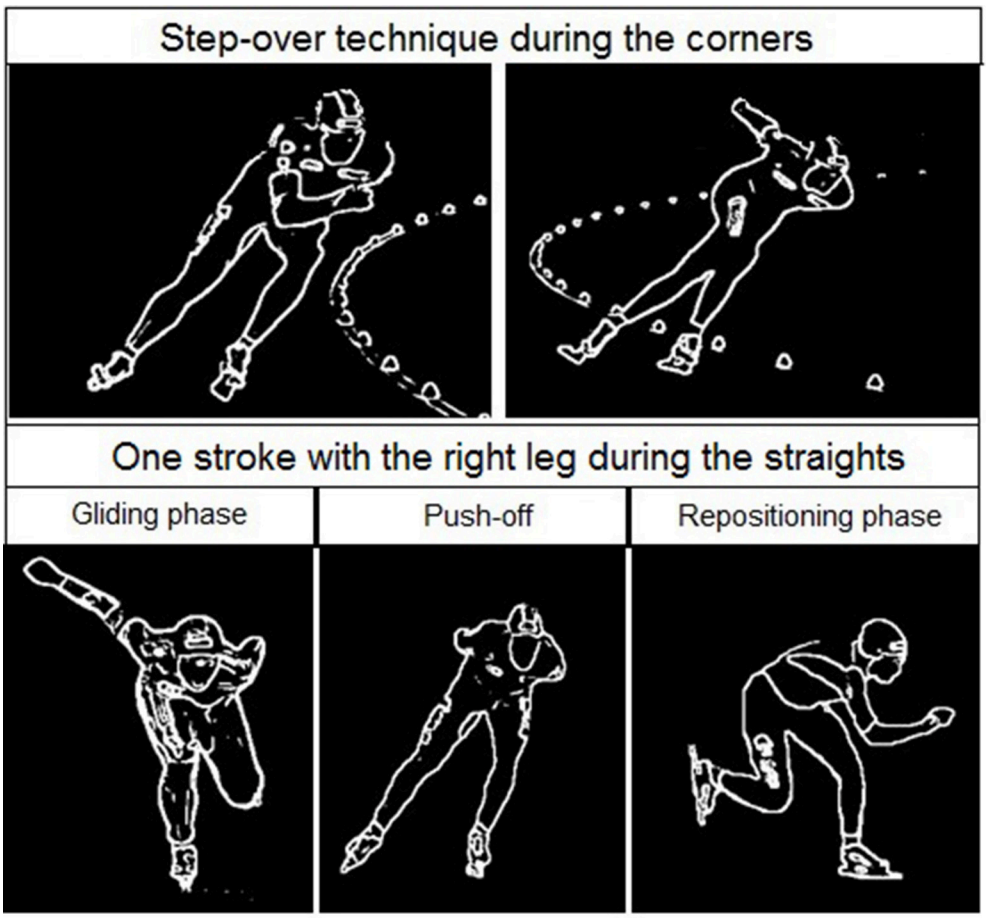

FIGURE 7 | Skating at the corner (upper panel) and the straight (lower panel) in long track speed skating. The corner typically contains a series of leg crossovers. The straight is characterized by several strokes containing a gliding phase (left panel), push-off (middle panel), and repositioning phase (right panel) as shown in the lower figure for the right leg.

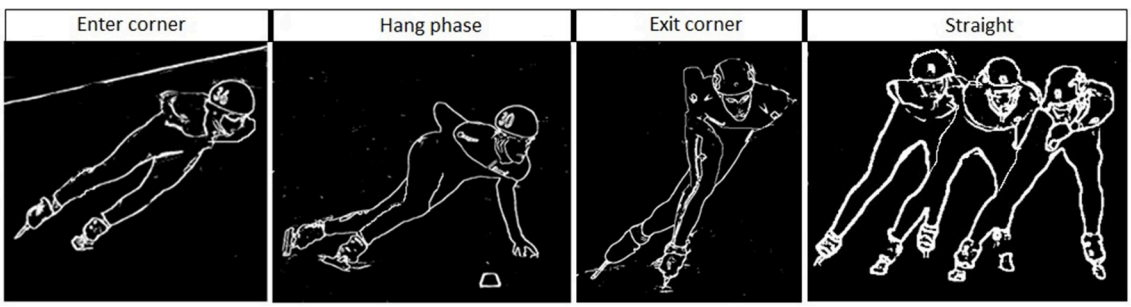

FIGURE 8 | Skating at the corner (first three panels) and the straight (right panel) in short track speed skating. The corner in short-track speed skating contains three subsections in the following order: an entry at which the skater performs leg crossovers, a hang in which the skater travels around the apex of the corner supported solely on the right blade, and an exit involving leg crossovers. The straight contains in short-track speed skating a glide on each blade and ends when the left blade touches the ice after the right foot glide.

skating modes. This is of a similar magnitude to a study in forearm muscles, where it was demonstrated that a 7% or larger reduction in muscle oxygenation was found to lead to decreased muscle force production (Murthy et al., 2001).

We hypothesized that the clearly demonstrated differences in deoxygenation and reoxygenation between sports would have an impact on perceived fatigue and recovery, in favor of long-track skating. Indeed, higher experienced fatigue after 2 and 4 h were

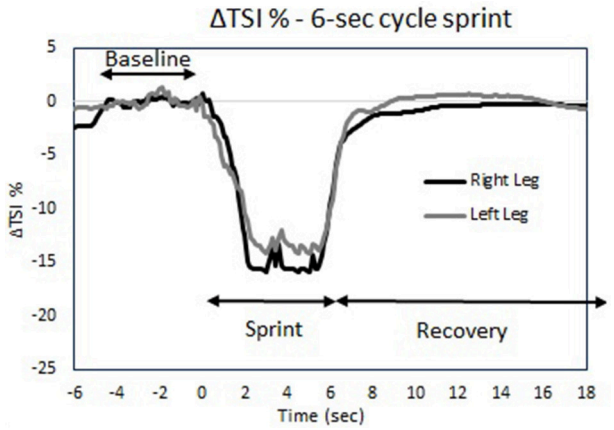

FIGURE 9 | Tissue saturation index trace (ΔTSI%) during the 6-s sprint cycle test protocol for both legs.

TABLE 2 | Recovery parameters after both testing sessions including the drop in ΔTSI% during the 6-s sprint, the halftime recovery in ΔTSI% after the 6-s sprint test, and the rate of perceived of exertion 2 and 4 h after the testing session.

		Long-track	Short-track
PPO (W)			
	Pretest	1244 ± 185	1229 ± 171
	Post-test	1135 ± 179	1096 ± 195
ΔTSI% 6-S SPRINT			
Left leg	Pretest	−10.1 ± 4.1	−10.9 ± 6.5
	Post-test	−10.2 ± 4.1	−11.1 ± 6.7
Right leg	Pretest	−10.8 ± 4.0	−11.1 ± 4.6
	Post-test	−10.5 ± 3.4	−12.0 ± 4.9
HALFTIME RECOVERY ΔTSI% (SEC)			
Left leg	Pretest	4.7 ± 3.3	4.7 ± 3.8
	Post-test[a]	5.1 ± 3.9	5.9 ± 4.8
Right leg	pretest	4.6 ± 3.7	4.5 ± 3.3
	Post-test[a]	4.8 ± 3.1	5.6 ± 4.0
RATE OF PERCEIVED EXERTION (1–10)			
	Two hours after[a]	4.2 ± 1.5	5.8 ± 2.0
	Four hours after[a]	3.1 ± 1.6	4.6 ± 1.9

[a] *Significant difference between skating mode ($p < 0.05$).*

found after the short-track time-trials compared to the long-track time-trials. This is despite the lack of a difference between lactate values, heart rate data and experienced fatigue when measured directly after the two testing sessions. Some clue to possible mechanisms came from the post-exercise 6-s maximal exercise test. Following short-track skating, there was a clear decrease in the post-exercise reoxygenation rate compared to long-track skating. Intriguingly this difference was seen in both legs, i.e., no differences were found between the right and left legs recovery rates even in short track skating. This might indicate that the differences between both legs are too small to affect processes of recovery after speed skating, however, it might also be a consequence of the relatively long time period (±5 min) between the end of training and the post-test. Nevertheless, whatever the detailed mechanism, the present study provides evidence that short-track speed skating is physiologically more demanding than long track speed skating and that this is associated with a demonstrated difference in hemodynamic patterns both during and post-race and a slower recovery from fatigue.

The application of blood flow restriction in high intensity cycling impacted on lactate accumulation and ratings of perceived exertion (Kim et al., 2015). At low exercise intensities, it was found that RPE was not affected by higher pressures in the range between 40 and 90% of arterial occlusion (Loenneke et al., 2016). A key difference in our skating exercises is that high phases of high intra-muscular pressures in the cycle are alternated with phases of unloading, when the leg is in the swing phase. Alternating pressure patterns and their impact on oxygenation and metabolism in human propulsion have not been studied very often in literature. Our work could inform on the mechanism of Kaatsu training: the application of blood flow restriction during low intensity training that has been shown to produce favorable muscle and vascular adaptations exacerbating training effect. This could be of interest for clinical populations (Loenneke et al., 2016).

In contrast to previous reports that focused on only one skating modality, the current study benefitted from using the same sample of subjects, familiar to both skating modalities, for both long-track and short-track speed skating. In agreement with previous separate studies for short-track (Rundell et al., 1997; Hesford et al., 2012, 2013b) and long-track (Foster et al., 1999), oxygenation changes in the current study are best explained by postulating a reduced blood flow to the working muscles throughout the race for both speed skating disciplines. Most likely, this reduced blood flow caused the relatively high lactate values and low oxygen uptake values in this study. This supports the assumption that a reduced blood flow reduces the aerobic capacity of the recruited muscle groups (Rundell, 1996). The current findings also indicate that the relatively low reported maximal oxygen uptake values in previous long-track speed skating studies (Smith and Roberts, 1991; De Koning et al., 1994) are a consequence of the deoxygenation of the working muscles in speed skating.

In terms of performance, traveling around the corners in short-track skating results in different velocity profiles compared to long-track speed skating. The subjects were slower during short-track speed skating when they traveled around the corner standing at one leg only. To regain their loss in velocity, the subjects accelerated when they came out of the corner and during the straight. Consequently, short-track seems to show more variability in velocity in each lap, while the long-track velocity profile seems more continuous (see **Figure 1**). Fluctuations in velocity were larger in short-track speed skating, and large fluctuations around the mean velocity are associated with higher aerodynamic energy losses (Van Ingen Schenau et al., 1983; De Koning et al., 1999; Hettinga et al., 2011, 2012). In running, a more variable interval training pattern has been shown to be associated with a slower recovery than demonstrated after continuous training (Townsend et al., 2013). In that sense, this finding also confirms that short-track speed skating is the more

demanding sports of the two, and might need a longer recovery after exercise and training.

For trainers and coaches it is important to realize that due to this element of occlusion and reduced blood flow as well as the larger fluctuations in speed, physiological load is higher than expected for their athletes during short-track speed skating compared to long-track speed skating. Moreover, training schedules based on physiological variables achieved during other non-occluded sports, such as commonly used (maximal) cycling tests, might not represent the actual physiological load of the training sessions, and trainers should beware of risks of overtraining. The working muscles of the right leg remained relatively more deoxygenated during short-track compared to long-track speed skating. This indicates that the muscles in short-track speed skating are more anaerobic than in long-track speed skating. However, as oxygen extraction does not change with time even when oxygenation recovers in long-track skating, it is not clear that this has adverse metabolic consequences at least in terms of oxygen metabolism. Nevertheless, the present study shows that a short-track training session leads to higher experienced fatigue in the hours after the training session and seems to need a longer recovery period compared to a similar long-track session.

Our findings emphasize the importance of pacing in speed skating. In particular the ability to maintain the optimal technical skating characteristics (respectively, small knee-, trunk-, and push-off angle) during the whole race, despite processes of fatigue, seemed to be crucial in elite (long-track) speed skating (Hettinga et al., 2011) and lead to different pacing behavior in speed skating compared to for example cycling (Stoter et al., 2016). Less explosive pacing strategies are chosen in speed skating, to make sure not to collapse at the end of the race. The present study showed that the crouched skating position indeed seemed to lead to a physiological disadvantage: the deoxygenation of the working muscles, which may cause an earlier onset of fatigue. As in the short-track time-trials in the present study, a relatively high decrease in velocity toward the end of the race was demonstrated, also in short-track speed skating the ability to maintain optimal technical skating characteristics at the end of the race seems crucial (Konings et al., 2016). In particular in actual competition this will be of importance, as this is performed in heats, competing against opponents (Konings et al., 2016; Noorbergen et al., 2016).

In conclusion, the current study showed that the patterns of reoxygenation and deoxygenation in the working muscles during a race are different for long-track and short-track speed skating. This seems to be a result of the high intramuscular forces on the right leg when traveling around the corner in short-track skating: athletes are usually standing on the right leg throughout the corner, that thereby remains deoxygenated for a long period where the left leg reoxygenates in the same phase when off the ice. In long-track skating, a more symmetric profile was demonstrated, in which over the straights the cyclic pattern of stance phase, push off phase and swing phase was clearly visible in terms of oxygenation. In the corners, the rhythmic leg crossovers resulted in a rather stable value for deoxygenation, that was larger for the right leg compared to the left leg. The results demonstrated that short-track speed skating seems to be the more physiologically demanding sport. This subsequently led to higher experienced fatigue directly after the exercise, as well as to longer periods of recovery needed in the hours after the short-track compared to long-track speed skating. These results can help coaches and athletes in optimizing their training protocols, and provide us with more insights into the mechanistic physiological principles relevant for elite athletes in different sports, and on how technical factors are impacting on those.

AUTHOR CONTRIBUTIONS

The measurements were conducted at InnoSportLab Thialf, with portable equipment available from the laboratory of the University of Essex. FH, MK, and CC all contributed to conception and design of the work. MK collected data. FH, MK, and CC were involved in analysis and interpretation. MK, FH, and CC wrote the paper. All authors have approved the final version of the manuscript, agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved, and all persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

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frontiers in Physiology

MINI REVIEW
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Fatigue Induced by Physical and Mental Exertion Increases Perception of Effort and Impairs Subsequent Endurance Performance

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Endurance performance involves the prolonged maintenance of constant or self-regulated power/velocity or torque/force. While the impact of numerous determinants of endurance performance has been previously reviewed, the impact of fatigue on subsequent endurance performance still needs to be documented. This review aims to present the impact of fatigue induced by physical or mental exertion on subsequent endurance performance. For the purpose of this review, endurance performance refers to performance during whole-body or single-joint endurance exercise soliciting mainly the aerobic energy system. First, the impact of physical and mental exertion on force production capacity is presented, with specific emphasize on the fact that solely physical exertion and not mental exertion induces a decrease in force production capacity of the working muscles. Then, the negative impact of fatigue induced by physical exertion and mental exertion on subsequent endurance performance is highlighted based on experimental data. Perception of effort being identified as the variable altered by both prior physical exertion and mental exertion, future studies should investigate the underlying mechanisms increasing perception of effort overtime and in presence of fatigue during endurance exercise. Perception of effort should be considered not only as marker of exercise intensity, but also as a factor limiting endurance performance. Therefore, using a psychophysiological approach to explain the regulation of endurance performance would allow a better understanding of the interaction between physiological and psychological phenomena known to impact endurance performance.

Keywords: muscle fatigue, cognitive fatigue, cycling, running, time to exhaustion, time trial, aerobic exercise, perceived exertion

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INTRODUCTION

Endurance performance involves the prolonged maintenance of constant or self-regulated power/velocity (e.g., Girard et al., 2012; Jones et al., 2016; Smits et al., 2016) or torque/force (e.g., Froyd et al., 2013; Pageaux et al., 2015a; Angius et al., 2016). Traditionally, endurance performance is measured by completion of time to exhaustion tests (i.e., open loop exercises) or time trials (i.e., closed loop exercises). While time to exhaustion tests consist in the maintenance of a fixed

power/velocity or torque/force until exhaustion (i.e., disengagement from the exercise), time trials consist in the completion of a set amount of work as quickly as possible or as much work as possible in a set time. Both time to exhaustion tests and time trials have been shown to be reliable and valid measure of endurance performance (Laursen et al., 2007; Amann et al., 2008).

Endurance performance can be investigated via the use of whole-body exercises (e.g., cycling); or single-joint exercises (e.g., one leg dynamic exercise). While whole-body exercises present the advantage of replicating real sport events in laboratory conditions, single-joint exercises provide a unique exercise model to investigate underlying mechanisms thought to impact endurance performance. As an example, single-joint exercises present the advantage of reducing the time delay between the end of the endurance exercise and the start of neuromuscular testing (Pageaux et al., 2016). Therefore, researchers, coaches and athletes can benefit of considering both exercise models as a measure of endurance performance even if whole-body and single-joint exercises are known to induce different systemic responses to the exercise (Sidhu et al., 2013).

While the impact of numerous determinants (e.g., nutrition, oxygen uptake or sleep) of endurance performance has been previously reviewed (e.g., Bassett and Howley, 2000; Joyner and Coyle, 2008; McMahon et al., 2016; Simpson et al., 2016), the impact of fatigue on subsequent endurance performance still needs to be documented. The Oxford Dictionary defines fatigue as an "*extreme tiredness resulting from mental or physical exertion or illness*" and/or "*a reduction in the efficiency of a muscle or organ after prolonged activity*." Therefore, the present review aims to present the impact of fatigue induced by physical or mental exertion on subsequent endurance performance. Firstly, the impact of physical and mental exertion on force production capacity will be presented, with specific emphasize on the fact that solely physical exertion and not mental exertion induces a decrease in force production capacity of the working muscles. Secondly, the negative impact of fatigue induced by physical exertion and mental exertion on subsequent endurance performance will be highlighted based on experimental data. Finally, as perception of effort during subsequent endurance exercise is the only variable altered by both prior physical and mental exertion, some insights on the impact of fatigue on perception of effort will be presented.

For the purpose of this review, endurance performance refers to performance during whole-body or single-joint endurance exercise soliciting mainly the aerobic energy system. Consequently, we considered only the studies that met the following criteria:

- The endurance exercise lasted at least 75 s (Gastin, 2001).
- Endurance performance was investigated as time to exhaustion tests, time trials or graded exercises.
- As this review focuses on the impact of fatigue on subsequent endurance performance, all studies included a physiological and/or psychological manipulation check attesting of the presence of fatigue prior to endurance performance measurement (see **Table 1**, "Markers of fatigue induced by the fatiguing protocol").

A comprehensive list of studies included in this review is presented in **Table 1**.

FATIGUE INDUCED BY PHYSICAL EXERTION AND MENTAL EXERTION: IMPACT ON FORCE PRODUCTION CAPACITY

When completion of physical exertion induces a reduction in force production capacity of a muscle group, fatigue is traditionally defined as muscle fatigue (Gandevia, 2001). When completion of mental exertion induces a reduction in cognitive performance and/or an increase in subjective feelings of tiredness and lack of energy, fatigue is traditionally defined as mental fatigue (Boksem and Tops, 2008).

Fatigue induced by physical exertion has been extensively studied in the literature (for review please see Enoka and Stuart, 1992; Gandevia, 2001; Enoka and Duchateau, 2008) and could be investigated by measuring the maximal force, torque or power that can be produced by a muscle or a muscle group. A reduction in maximal force, torque or power obtained during maximal voluntary contractions (MVC) is the gold standard to identify the presence of reduced force production capacity (Gandevia, 2001). This reduction in force production capacity has been shown to be caused by an inability of the central nervous system to maximally recruit the working muscles (i.e., traditionally defined as central fatigue; Gandevia, 2001) and also by changes at or distal to the neuromuscular junction, impairing contractile properties of the working muscles (i.e., traditionally defined as peripheral fatigue; Allen et al., 2008).

Fatigue induced by mental exertion is a psychobiological state caused by prolonged engagement in mentally demanding cognitive activities (Boksem and Tops, 2008). Its presence is traditionally identified by completion of questionnaires allowing the athlete/subject to report his/her feelings of fatigue, defined as tiredness and lack of energy (Boksem and Tops, 2008). This methodology has been shown to be successful in identifying presence of fatigue induced by mental exertion lasting at least 30 min (e.g., Marcora et al., 2009; Pageaux et al., 2013; Smith et al., 2016). Interestingly, as fatigue induced by mental exertion (Wang et al., 2016) and central fatigue (Taylor et al., 2000) are both phenomena occurring in brain areas upstream of the primary motor cortex, several authors have proposed an association between these two phenomena (e.g., Newsholme et al., 1992; Di Giulio et al., 2006). However, recent studies focusing on the impact of mental exertion on force production capacity demonstrated that mental exertion does not impair the ability of an athlete/subject to maximally recruit his/her working muscles (Pageaux et al., 2013, 2015b; Martin et al., 2014; Rozand et al., 2014; Duncan et al., 2015). Therefore, as only fatigue induced by physical exertion and not by mental exertion impairs force production capacity (Pageaux et al., 2015b), it seems crucial to differentiate the kind of exercise inducing fatigue.

TABLE 1 | Comprehensive list of studies investigating the impact of fatigue on subsequent endurance performance.

References	Subjects	Fatiguing protocol	Markers of fatigue induced by the fatiguing protocol	Endurance exercise	Impact of fatigue on endurance performance	RPE during the endurance performance test
FATIGUE OF A MUSCLE GROUP INVOLVED IN SUBSEQUENT WHOLE-BODY ENDURANCE EXERCISE						
Amann and Dempsey, 2008	8 competitive ♂ cyclists, PL3	2 cycling conditions: (i) to exhaustion at 83% MAP, (ii) same duration at 67% MAP	↓ in KE MVC following 83% MAP, ↓ in KE twitch in both conditions, no change in VAL	5 km cycling time trial	↑ in time to complete the time trial, with a greater ↑ following 83% MAP	leg discomfort as a confounding factor
de Morree and Marcora, 2013	10 recreationally active ♂, PL2	100 drop-jumps (20 s rest between 2 jumps)	↓ in KE MVC no change in blood lactate	15 min cycling time trial	↓ in total work completed	↑
Deley et al., 2006	9 active ♂, PL2	2 conditions lasting 20 min: i) electromyostimulation, ii) voluntary contractions. KE isometric contractions 10 s ON—10 s OFF	↓ in KE MVC, twitch and VAL; greater ↓ in KE MVC and twitch post electromyostimulation	cycling time to exhaustion at 80% VO_{2max}	↓ in time to exhaustion in both conditions, greater ↓ following electromyostimulation	not reported
Marcora et al., 2008	10 active ♂, PL2	100 drop-jumps (20 s rest between 2 jumps)	↓ in KE MVC, no change in KE muscle pain	cycling time to exhaustion at 80% MAP, ∼90 ± 7% VO_{2max}	↓ in time to exhaustion	↑
FATIGUE OF A MUSCLE GROUP INVOLVED IN SUBSEQUENT SINGLE-JOINT ENDURANCE EXERCISE						
Sherman et al., 1984	8 ♂runners, PL4	marathon	↓ in KE MVC	50 isokinetic KE concentric contractions	↓ in total work completed	not reported
Neyroud et al., 2012	14 physically active ♂, no information for PL determination	20% KE MVC time to exhaustion	↓ in KE MVC, twitch and VAL	20% KE MVC time to exhaustion	↓ in time to exhaustion	↑
FATIGUE OF A MUSCLE GROUP NON-INVOLVED IN SUBSEQUENT WHOLE-BODY ENDURANCE EXERCISE						
Johnson et al., 2014	7 moderately trained ♂, PL2	8 × 1 min interspaced by 30 s rest at 1.5–2.0 W/kg	↑ in blood lactate and ion hydrogen, no measurement of force production capacity	Incremental cycling test, time to exhaustion at 85% MAP CP and W' estimation	↓ MAP and VO_{2max} achieved during the incremental cycling test, ↓ in time to exhaustion and W', no change in CP	not reported
Johnson et al., 2015	8 moderately trained ♂, PL2	8 × 1 min interspaced by 30 s rest at 1.0–1.5 W/kg	No measurement of force production capacity of the upper limbs, ↑ in blood lactate	time to exhaustion at 85% MAP	↓ in time to exhaustion, greater ↓ in KE MVC following the control time to exhaustion test	leg discomfort as a confounding factor
FATIGUE OF A MUSCLE GROUP NON-INVOLVED IN SUBSEQUENT SINGLE-JOINT ENDURANCE EXERCISE						
Amann et al., 2013	8 recreationally active ♂, PL2	unilateral KE isotonic contractions at 85% MAP to exhaustion	↓ in KE MVC of the pre fatigued leg, ↓ in KE twitch of the pre fatigued leg, no change in VAL	controlateral KE isotonic contractions at 85% MAP to exhaustion	↓ in time to exhaustion	↑
Bangsbo et al., 1996	7 active ♂, PL2	4 × 1 min arm cranking at 137 ± 3 W	↑ in muscle lactate, no measurement of force production capacity	KE isotonic contractions at 61.4 ± 3.7 W to exhaustion	↓ in time to exhaustion	not reported
Nordsborg et al., 2003	6 active ♂, PL2	4 × 1 min arm cranking at ∼ 140 W	↑ in interstitial potassium, no measurement of force production capacity	KE isotonic contractions at 62.8 ± 3.0 W to exhaustion	↓ in time to exhaustion	not reported
Triscott et al., 2008	3 groups of 8 subjects: sedentary (PL1), resistance (PL2–3), endurance (PL2–3)	unilateral bicep curls to exhaustion (weight 5.5 kg)	↓ in EF MVC of the pre fatigued arm	controlateral bicep curls to exhaustion (weight 4.5 kg)	↓ in time to exhaustion	not reported

(Continued)

TABLE 1 | Continued

References	Subjects	Fatiguing protocol	Markers of fatigue induced by the fatiguing protocol	Endurance exercise	Impact of fatigue on endurance performance	RPE during the endurance performance test
FATIGUE INDUCED BY MENTAL EXERTION AND SUBSEQUENT WHOLE-BODY ENDURANCE EXERCISE						
MacMahon et al., 2014	18 trained ♂ and 2 trained ♀, PL2	90 min of AX-continuous performance test	↑ in heart rate during the cognitive task, ↑ in self-reported fatigue, no measurement of force production capacity	3 km running time trial	↑ in time to complete the time trial	↑ (same RPE for a lower running velocity)
Marcora et al., 2009	10 active ♂ and 6 active ♀, PL2	90 min of AX-continuous performance test	↑ in heart rate during the cognitive task, ↑ in self-reported fatigue, ↓ in cognitive performance, no measurement of force production capacity	time to exhaustion at 80% MAP	↓ in time to exhaustion	↑
Martin et al., 2016	11 professional ♂ road cyclists (PL5) and 9 recreational ♂ cyclists (PL1–2)	30 min of incongruent Stroop task	↑ in self-reported fatigue for both groups, no measurement of force production capacity	20 min cycling time trial	↓ in power output in the PL 1–2 group only no change in performance in the PL 5 group	↑ in the PL1–2 group (same RPE for lower power output) no change in the PL 5 group
Pageaux et al., 2014	10 recreationally active ♂, PL2	30 min of incongruent Stroop task	↑ in heart rate during the cognitive task, ↑ in mental demand and effort, no measurement of force production capacity	5 km running time trial	↑ in time to complete the time trial	↑
Smith et al., 2015	10 recreationally active ♂, PL2	90 min of AX-continuous performance test	↑ in heart rate during the cognitive task, ↑ in self-reported fatigue, no measurement of force production capacity	45 min self-paced intermittent running protocol replicating team sports physical demand	↓ in running velocity	↑ (same RPE for a lower running velocity)
Smith et al., 2016	12 moderately trained soccer ♂, PL2	30 min of incongruent Stroop task	↑ in self-reported fatigue, no measurement of force production capacity	Yo-Yo Intermittent Recovery Test, Level 1	↓ in running distance	↑
FATIGUE INDUCED BY MENTAL EXERTION AND SUBSEQUENT SINGLE-JOINT ENDURANCE EXERCISE						
Pageaux et al., 2013	10 active ♂, PL2	90 min of AX-continuous performance test	↑ in heart rate during the cognitive task, ↑ in self-reported fatigue, no change in KE MVC	20% KE MVC time to exhaustion	↓ in time to exhaustion	↑

All studies presented in this table are discussed within the manuscript. EF, elbow flexors; KE, knee extensors; MVC, maximal voluntary contraction; PL, performance level (De Pauw et al., 2013); RPE, ratings of perceived exertion; VAL, voluntary activation level. ↑, increase; ↓, decrease; MAP, maximal aerobic power; CP, critical power.

FATIGUE OF A MUSCLE GROUP INVOLVED IN SUBSEQUENT ENDURANCE EXERCISE IMPAIRS ENDURANCE PERFORMANCE

Whole-Body Exercise

In 2006, Deley and colleagues tested the impact of a decrease in knee extensors (KE) force production capacity, using either electromyostimulation or voluntary contractions, on the VO_2 kinetics during a cycling time to exhaustion test performed at 80% VO_{2max}. These authors demonstrated not only that the appearance of the VO_2 slow component was delayed in the electromyostimulation condition and its amplitude was lower than that obtained in the voluntary contractions condition, but also that endurance performance was reduced in both fatiguing conditions compared to the control condition. In addition, the decrease in endurance performance was greater following electromyostimulation (−25.9%) compared to voluntary contractions (−6.4%), thus in relation to the extent of KE MVC reduction (electromyostimulation: −19.9%, voluntary contractions: −11.8%). To the best of our knowledge, this study is the first to report a decrease in endurance performance induced by fatigue of a muscle group involved in subsequent endurance exercise. Similar results were observed by Amann and Dempsey (2008) with a different fatiguing protocol. In this study, a reduction in KE MVC was induced by either a cycling time to exhaustion test at 83% of maximal aerobic power (MAP) or a cycling exercise of the same duration at 67% MAP. Both fatiguing conditions induced an increase in time to complete the subsequent 5 km cycling time trial, with a greater increase following the 83% MAP condition (+6%) compared to the 67% MAP condition (+2%). This greater increase in time to complete the 5 km cycling time trial in the 83% MAP condition was associated with a greater extent of KE MVC reduction post time to exhaustion test at 83% MAP compared to cycling for the same duration at 67% MAP. However, it has to be noticed that none of the two studies aforementioned controlled for the confounding factor of prior exercise induced accumulation of metabolites, thought to influence endurance performance (Amann, 2011). For this reason, Marcora and colleagues (Marcora et al., 2008; de Morree and Marcora, 2013) performed similar experiments by looking at the effects of a fatigue protocol known to induce a significant reduction in KE force production capacity in absence of significant accumulation of muscle metabolites (Skurvydas et al., 2000, 2002). Marcora and colleagues confirmed the results of previous studies by demonstrating that even without accumulation of muscle metabolites; a reduction in KE force production capacity induces a decrease in subsequent cycling endurance performance. Therefore, when integrating the results of the studies aforementioned, it is clear that fatigue of a muscle group involved in subsequent whole-body endurance exercise decreases endurance performance.

Single-Joint Exercise

In 1984, Sherman and colleagues demonstrated that completion of a marathon decreases the amount of work performed during a work capacity test consisting in the repetition of 50 maximal leg extensions (duration of the endurance exercise ∼2 min). This impairment in endurance performance was confirmed by Neyroud et al. (2012). In this study, the duration of a continuous KE isometric contraction at 20% MVC to exhaustion was reduced by 70% when performed subsequently to an initial KE isometric contraction at 20% MVC to exhaustion. Therefore, as previously discussed for whole-body endurance exercise, fatigue of a muscle group involved in subsequent single-joint endurance exercise also decreases endurance performance.

FATIGUE OF A MUSCLE GROUP NON-INVOLVED IN SUBSEQUENT ENDURANCE EXERCISE IMPAIRS ENDURANCE PERFORMANCE

Whole-Body Exercise

To the best of our knowledge, only Johnson and colleagues investigated the impact of fatigue of a muscle group non-involved in subsequent whole-body endurance exercise on endurance performance (Johnson et al., 2014, 2015). To do so, the authors performed intermittent arm cranking to fatigue the upper limbs, and then investigated the impact of this previous exercise on subsequent cycling endurance performance. The authors demonstrated that even if the arm cranking exercise does not alter subsequent critical power estimation (Johnson et al., 2014), arm cranking impairs subsequent cycling endurance performance. This decrease in cycling endurance performance was evidenced by a decrease in MAP (−7%) and VO_{2max} (−14%) achieved during a graded exercise (Johnson et al., 2014), and a decrease in time to exhaustion at 85% MAP (∼35%; Johnson et al., 2014, 2015).

Single-Joint Exercise

When endurance performance is investigated with single-joint exercise, the impact of fatigue of a muscle group non-involved in subsequent endurance exercise on endurance performance could be investigated by either fatiguing one limb and testing the controlateral limb endurance performance, or by fatiguing the upper body (or lower body) and testing a lower body (or upper body) muscle group endurance performance. With regard to endurance performance of the controlateral limb, Amann et al. (2013) demonstrated a decrease in controlateral KE isotonic contractions time to exhaustion performed at 85% MAP (−49%) following a previous unilateral KE isotonic contractions time to exhaustion performed at same intensity. In 2008, Triscott and colleagues demonstrated a ∼20% decrease in controlateral biceps curls time to exhaustion following unilateral biceps curls to exhaustion. In both studies, neither the unilateral biceps curls time to exhaustion nor the unilateral KE isotonic contractions time to exhaustion induced a reduction in force production capacity of the controlateral limb involved in the subsequent endurance exercise. With regard to subsequent endurance performance of a lower body muscle group following prior fatiguing exercise involving the upper body, Bangsbo et al. (1996) and Nordsborg et al. (2003) demonstrated a negative impact of prior arm cranking on KE isotonic contractions time to exhaustion. By using identical fatiguing protocol (4 × 1 arm

cranking at ∼140 W), the authors observed a 26% (Bangsbo et al., 1996) and 32% (Nordsborg et al., 2003) decrease in KE isotonic contractions time to exhaustion at ∼60 W.

FATIGUE INDUCED BY MENTAL EXERTION IMPAIRS ENDURANCE PERFORMANCE

Whole-Body Exercise

Since the first study of Marcora et al. (2009) demonstrating an impairment of 15% in cycling endurance performance caused by prior mental exertion, numerous studies investigating the impact of fatigue induced by mental exertion on whole-body endurance performance have been published (MacMahon et al., 2014; Pageaux et al., 2014; Smith et al., 2015, 2016). All these studies reached a consensus on the negative impact of prior mental exertion on endurance performance; even so elite athletes present a greater resistance to fatigue induced by prior mental exertion (Martin et al., 2016). This impairment was observed during cycling (Marcora et al., 2009; Martin et al., 2016) and running exercises (MacMahon et al., 2014; Pageaux et al., 2014). Interestingly, Smith and colleagues demonstrated that fatigue induced by mental exertion also impairs prolonged intermittent (Smith et al., 2015) and graded (Smith et al., 2016) running exercises. Consequently, it seems clear that fatigue induced by mental exertion decreases endurance performance, even if prior mental exertion does not alter physiological responses to endurance exercise (Marcora et al., 2009; Pageaux et al., 2013).

Single-Joint Exercises

To the best of our knowledge, only one study investigated the impact of prior mental exertion on endurance performance. In this study, Pageaux et al. (2013) demonstrated that even if prior mental exertion does not reduce KE force production capacity, fatigue induced by mental exertion still causes a decrease in endurance performance during a continuous KE isometric contraction at 20% MVC to exhaustion. However, no study investigated the impact of fatigue induced by mental exertion on single-joint time trial.

PRIOR PHYSICAL EXERTION AND PRIOR MENTAL EXERTION INCREASE PERCEPTION OF EFFORT DURING SUBSEQUENT ENDURANCE EXERCISE

As demonstrated by the studies included in this review (see **Table 1**), endurance performance could be altered in absence (e.g., Pageaux et al., 2013) or presence (e.g., Marcora et al., 2008) of a decrease in force production capacity of the working muscles involved in the subsequent exercise. Endurance performance could also be altered with (e.g., Amann et al., 2013) or without (e.g., Marcora et al., 2009) altered physiological responses to the exercise. Therefore, these results raise a simple question: do physical exertion and mental exertion alter a common variable during subsequent endurance exercise? As shown in **Figure 1**, the answer is yes. This variable altered by both physical exertion and mental exertion is the perception of effort.

Perception of effort (also referred as perceived exertion or sense of effort), defined as "the feeling of how hard, heavy and strenuous a physical task is" (Marcora, 2010; Pageaux, 2016), is a cognitive feeling of work associated with voluntary actions (Preston and Wegner, 2009; Pageaux, 2016). This perception differs from other exercise-related sensations such as pain or discomfort (Pageaux, 2016), and can be rated via the use of psychophysiological scales such as the Borg ratings of perceived exertion scale or the category ratio (CR)10 scale (Borg, 1998). While a persistent debate exists in the literature on the neurophysiology of perceived exertion (Marcora, 2009; Pageaux, 2016), it exists strong experimental data providing evidence that perception of effort results from the neuronal process of the corollary discharge associated with the central motor command (Marcora, 2009; Pageaux, 2016; Pageaux and Gaveau, 2016). Indeed, studies using pharmacological blockade of muscle afferents demonstrated that in absence of muscle afferent feedback, perception of effort is not reduced during endurance exercise (Pageaux and Gaveau, 2016). While the increased perceived exertion in presence of fatigue of a muscle group involved in subsequent endurance exercise is associated with an increase in activity of cortical premotor and motor areas (i.e., index of central motor command) to compensate for alteration of neuromuscular properties of the working muscles (de Morree et al., 2012); the underlying mechanisms behind the increased perceived exertion induced by fatigue of a muscle group non-involved in subsequent endurance exercise and fatigue induced by mental exertion remain unclear. Although some authors proposed an increase in afferent feedback caused by fatigue of a muscle group non-involved in subsequent endurance exercise to be responsible of the increased perceived exertion (Amann et al., 2013), this hypothesis is unlikely because spinal blockade of muscle afferents does not reduce perceived exertion (Pageaux and Gaveau, 2016). An alternative hypothesis could be that prior prolonged activation of premotor and motor areas associated with the completion of the fatiguing exercise would induce intrinsic changes in the brain, inducing an alteration of the activation of premotor and motor areas in the subsequent exercise. As prolonged neural activity has been shown in animal studies to increase extracellular concentrations of adenosine (Lovatt et al., 2012), an increase in extracellular concentrations of adenosine caused by prior physical exertion could be a good candidate to explain the increased perceived exertion caused by fatigue of a muscle group non-involved in subsequent endurance exercise. This hypothesis has also been proposed to explain the increased perceived exertion caused by prior mental exertion (Pageaux et al., 2014, 2015b), and find additional support with (i) studies demonstrating a positive impact of caffeine (i.e., an antagonist of adenosine) ingestion on physical and cognitive performances (McLellan et al., 2016); and (ii) recent experimental evidence demonstrating the involvement of premotor and motor areas in cognition and decision-making process (Morsella et al., 2015; Ramkumar et al., 2016; Tomasino and Gremese, 2016). Therefore, future studies should investigate the underlying mechanisms responsible of the increased perception of effort

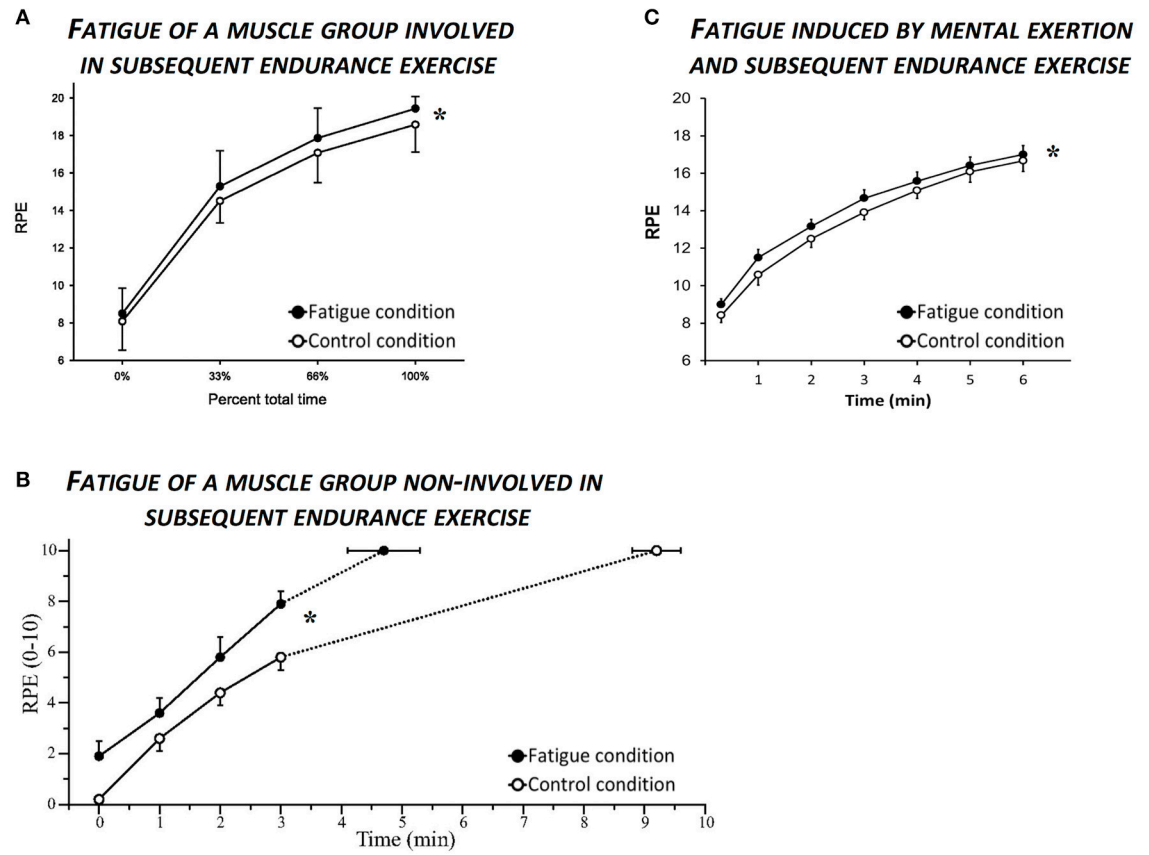

FIGURE 1 | Effects of fatigue induced by physical and mental exertion on ratings of perceived exertion (RPE) during subsequent endurance exercise. **(A)** Illustrates the increase in RPE induced by fatigue of a muscle group (knee extensors) involved in subsequent endurance exercise (constant load cycling exercise at 80% maximal aerobic power). Reprinted with permission and adapted from Marcora et al. (2008), p. R880, Figure 6A. **(B)** Illustrates the increase in RPE induced by fatigue of a muscle group (knee extensors) non-involved in subsequent endurance exercise (controlateral isotonic knee extension at 85% maximal aerobic power). Reprinted with permission and adapted from Amann et al. (2013), p. 361, Figure 4. **(C)** Illustrates the increase in RPE caused by Stroop task-induced fatigue during subsequent endurance exercise (constant load cycling exercise at 80% maximal aerobic power). Adapted from Pageaux et al. (2015b), p. 8, Figure 4A. In the three studies, RPE was higher during the fatigue condition compared to the control condition, as illustrated with the * representing a significant effect of condition ($p < 0.05$).

during exercise and caused by prior physical and mental exertion.

By integrating experimental results from different exercise modes and published by different research groups, this review provides evidence that fatigue induced by prior physical or mental exertion impairs subsequent endurance performance. While impairments in endurance performance are not associated with a common physiological alteration, perceived exertion seems to be the common variable altered by fatigue. Consequently, future studies should investigate the cause of the progressive increase in perceived exertion overtime during endurance exercise and consider perceived exertion not only as marker of exercise intensity, but also as a factor limiting endurance performance. Furthermore, as psychological interventions such as self-talk (Blanchfield et al., 2013) or subliminal images (Blanchfield et al., 2014) could be used to manipulate endurance performance; special attention should be given to models aiming to explain regulation of endurance performance with a psychophysiological approach (Marcora et al., 2008; Millet, 2011; Pageaux, 2014). Using a psychophysiological approach would allow a better understanding of the interaction between physiological and psychological phenomena known to impact endurance performance.

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Both authors have approved the final version of the manuscript and agree to be accountable for all aspects of the work.

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No Influence of Transcutaneous Electrical Nerve Stimulation on Exercise-Induced Pain and 5-Km Cycling Time-Trial Performance

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Introduction: Afferent information from exercising muscle contributes to the sensation of exercise-induced muscle pain. Transcutaneous electrical nerve stimulation (TENS) delivers low–voltage electrical currents to the skin, inhibiting nociceptive afferent information. The use of TENS in reducing perceptions of exercise-induced pain has not yet been fully explored. This study aimed to investigate the effect of TENS on exercise-induced muscle pain, pacing strategy, and performance during a 5-km cycling time trial (TT).

Methods: On three separate occasions, in a single-blind, randomized, and cross-over design, 13 recreationally active participants underwent a 30-min TENS protocol, before performing a 5-km cycling TT. TENS was applied to the quadriceps prior to exercise under the following conditions; control (CONT), placebo with sham TENS application (PLAC), and an experimental condition with TENS application (TENS). Quadriceps fatigue was assessed with magnetic femoral nerve stimulation assessing changes in potentiated quadriceps twitch force at baseline, pre and post exercise. Subjective scores of exertion, affect and pain were taken every 1-km.

Results: During TTs, application of TENS did not influence pain perceptions ($P = 0.68$, $\eta_p^2 = 0.03$). There was no significant change in mean power ($P = 0.16$, $\eta_p^2 = 0.16$) or TT duration ($P = 0.17$, $\eta_p^2 = 0.14$), although effect sizes were large for these two variables. Changes in power output were not significant but showed moderate effect sizes at 500-m ($\eta_p^2 = 0.10$) and 750-m ($\eta_p^2 = 0.10$). Muscle recruitment as inferred by electromyography data was not significant, but showed large effect sizes at 250-m ($\eta_p^2 = 0.16$), 500-m ($\eta_p^2 = 0.15$), and 750-m ($\eta_p^2 = 0.14$). This indicates a possible effect for TENS influencing performance up to 1-km.

Discussion: These findings do not support the use of TENS to improve 5-km TT performance.

Keywords: transcutaneous electrical nerve stimulation, pacing, performance, afferent feedback, time-trial

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INTRODUCTION

Pacing is the regulation of effort during exercise that aims to manage neuromuscular fatigue. It prevents excessive physiological harm and maximizes goal achievement (Edwards and Polman, 2013). To accomplish this, decisions are made based on information received from both internal, and external environments, to adjust exercise intensity (Edwards and Polman, 2013; Smits et al., 2014). Initially, pacing strategies are set in anticipation (Ulmer, 1996; Tucker, 2009), whilst regulation of intensity during exercise is influenced by afferent information from exercising muscle (Amann et al., 2009). Therefore, the ability to adjust or remove this information is of interest in pacing research, as there is potential to improve performance.

Exercise typically augments mechanical and chemical stimuli within the muscle, sensitizing, and activating nociceptive group III and IV afferent muscle fibers. These communicate information on actual or potential muscle damage to the central nervous system (O'Connor and Cook, 1999). Conscious awareness of this information forms the subjective sensation of exercise-induced pain (Loeser and Treede, 2008; Mauger, 2013). Pacing theory states that low signal (feedback) intensity will not trigger awareness (Swart et al., 2012). Yet, with an increasing stimulus intensity (i.e., intense exercise), conscious awareness is achieved, which results in appropriate decisions to change behavior (Swart et al., 2012; Edwards and Polman, 2013). Therefore, once nociceptive signals and consequently perceptions of pain become prominent, effort will be regulated to maintain discomfort at a tolerable level (Swart et al., 2012; Edwards and Polman, 2013; Mauger, 2014). This concept of a "sensory tolerance limit" (Gandevia, 2001; Hureau et al., 2016), likely occurs to prevent excessive physiological harm by limiting levels of fatigue (Amann et al., 2009). The sum of signals from a number of mechanisms contribute to this theory, including feedback from group III and IV muscle afferents but also feedback from respiratory muscles and corollary discharges (Hureau et al., 2016). Reductions to exercise performance and duration due to exercise-induced fatigue, are evidence of this concept (Amann et al., 2013). In addition, the importance of nociceptive information as part of a global tolerance limit has been demonstrated by altered nociceptive stimuli (i.e., induced or blocked), leading to changes in voluntary activation of muscle (Amann et al., 2009; Kennedy et al., 2014). Only a small number of studies, investigating analgesic interventions to augment afferent information have focused on exercise pacing and performance. Injecting fentanyl increases initial power output, but results in excessive fatigue (Amann et al., 2009), while ingestion of acetaminophen can increase mean power output during a time trial (TT) (Mauger et al., 2010) and during repeated-sprint exercise (Foster et al., 2014). An increased exercise intensity suggests nociceptive signals affect self-pacing. Yet, in acetaminophen studies, there was no change in pain perceptions. This suggests a threshold of perceived pain was adjusted and exercise intensity was increased to this tolerance limit. Therefore, there is merit for any analgesic intervention during self-paced exercise to adjust perceptions of exercise-induced pain allowing for an increased intensity (and possibly performance), before perceptions become prominent.

An alternative to ingested and injected analgesics is transcutaneous electrical nerve stimulation (TENS). Application of TENS involves low-voltage electrical currents administered to the skin for pain relief (Johnson et al., 2015). Analgesic effects are provided following the gate control theory of pain (Melzack and Wall, 1965), which controls transmission of nociceptive information. Specifically, stimulation provided by TENS targeting group II muscle afferent fibers excites inhibitory interneurons. This results in an attenuation of the ascending nociceptive stimuli from group III and IV afferent fibers (Sluka et al., 2013; Johnson et al., 2015). Based on this premise, it is possible that TENS could be used to attenuate nociceptive stimuli associated with aerobic exercise. Clinically, analgesic effects of TENS have been demonstrated for chronic musculoskeletal pain (Johnson and Martinson, 2007). In pain-free individuals, application can influence pressure pain thresholds (PPT). (Moran et al., 2011). During exercise in pain-free individuals, TENS application has improved tolerance by enhancing peripheral blood flow (Tomasi et al., 2015). Furthermore, when knee pain was induced, TENS has also reduced pain and restored quadriceps strength (Son et al., 2016). This suggests that TENS could be used in pain-free individuals to reduce pain perceptions, and possibly perceptions of exercise-induced pain. If demonstrated, a reduction of exercise-induced pain, in conjunction with possible alterations in muscle contractile properties and enhancement of blood flow, gives the potential for TENS application to be a performance enhancing strategy.

To the authors' knowledge, TENS has not been used in pain-free participants to modulate afferent feedback and reduce exercise-induced pain perceptions during self-paced exercise. Based on the potential to influence exercise-induced pain, the aim of this study was to investigate the efficacy of TENS administered before intense exercise to influence pain perceptions. It was hypothesized that TENS would influence the threshold for sensing exercise-induced pain, thereby increasing exercise intensity and performance for similar subjective pain perceptions. As TENS can influence muscle excitability and strength, a secondary objective was to assess within-exercise muscle recruitment (via electromyography) and neuromuscular fatigue. To allow the assessment of muscle recruitment, application of TENS was administered prior to exercise, in anticipation of approximately 30-min of post-stimulation analgesia, utilizing similar TENS settings displaying increased PPT within this period (Moran et al., 2011; Pantaleão et al., 2011). To allow for exercise to be within the post-stimulation time frame, a 5-km cycling TT was utilized, as it was anticipated this would be completed in ∼10-min.

METHODS

Participants

Thirteen recreationally active participants were recruited for this study (see **Table 1**). Written informed consent was provided in accordance with the Declaration of Helsinki. The inclusion of

TABLE 1 | Participant anthropometric data.

	Female $n = 4$	Male $n = 9$	Total $n = 13$
Age (years)	27.5 ± 7.4	23.3 ± 4.2	24.6 ± 5.5
Height (cm)	166.0 ± 9.1	179.5 ± 7.0	175.3 ± 9.8
Body mass (kg)	62.2 ± 9.2	77.1 ± 7.8	72.8 ± 10.6
PPO (W)	250.3 ± 39.7	321.3 ± 23.0	299.5 ± 43.7
PPO (W/kg)	4.0 ± 0.4	4.2 ± 0.5	4.2 ± 0.5
VO_{2peak} ($L.min^{-1}$)	2.6 ± 0.6	3.8 ± 0.3	3.4 ± 0.7
VO_{2peak}($ml.min.kg^{-1}$)	40.9 ± 6.3	49.4 ± 5.6	46.7 ± 6.9
Pain at PPO	7.8 ± 2.9	9.0 ± 1.1	8.6 ± 1.8

Data presented as mean ± SD. PPO, peak power output; VO_{2peak}, peak oxygen consumption; Pain at PPO, rating of perceived quadriceps pain at PPO.

both males and females was based on indications sex has no influence on level of exercise-induced pain within the time frame proposed for this study (Dannecker et al., 2012), with repeated measures trial design minimizing any potential differences. Using a medical questionnaire, all participants were screened for risk factors including suitability to the exercise, current pain, currently taking pain medication and any prior use of TENS. Participants who reported pain (chronic or acute) or prior use of TENS were excluded from the study. Participants were asked to refrain from any physical activity causing severe fatigue in the 36 h prior, as well as any caffeine intake or pain medication 2 h prior to testing sessions. Procedures were approved by Victoria University's Human Research Ethics Committee.

Experimental Procedures

Participants reported to the laboratory for seven sessions, which included four preliminary and three experimental sessions. Prior to experimental sessions, three 5-km TT sessions were conducted to familiarize participants and ensure adequate reliability of pacing and performance (Hibbert et al., unpublished data). Furthermore, during the first session participants were also familiarized with TENS, peripheral magnetic stimulation protocols and algometry (see procedures below). The final preliminary session was a peak oxygen uptake (VO_{2peak}) assessment to characterize participants' cardiorespiratory fitness. For experimental testing, on three different days separated by a minimum of 48 h, participants performed three 5-km cycling TTs in a single-blind randomized order: control (CONT), placebo with sham TENS application (PLAC), and an experimental condition with TENS application (TENS). TENS was applied for 30-min before performing a cycling TT (see TENS procedure below). Maximal voluntary contraction (MVC) force, responses to magnetic stimulation of the quadriceps and PPT were measured before TENS application (BASE), as well as pre (PRE), and immediately post (POST) exercise.

Upon reporting to the laboratory, participants were fitted with electromyography (EMG) electrodes (see below). A warm up of MVC and magnetic stimulation (see below) was conducted before BASE measurement. Participants were then fitted with TENS electrodes for 30-min of TENS application. A cycling warm-up (5-min cycling at 75 Watts) was conducted before PRE measurement of MVC. Immediately following PRE measurements, a 5-s sprint was conducted for EMG normalization purposes. Following this, the TT commenced after a verbal 3-s countdown from the researcher. To overcome flywheel inertia, participants were instructed to obtain a self-selected comfortable cadence immediately prior to beginning the TT. All exercise was conducted on a Velotron Pro cycle ergometer (RacerMate Inc., Seattle, WA, USA). Within the familiarization sessions, participants set the ergometer to their own specifications with values recorded and replicated for subsequent sessions. All TT protocols were controlled via Velotron Coaching software (Version 1.6.458, RacerMate Inc.) with all courses being flat with no wind effect. Participants were permitted to drink water *ad libitum* during trials. Participants were instructed to finish the required distance "as quickly as possible," being free to change gear and cadence throughout the TT as desired. Participants were blinded from information except for distance covered. Upon TT completion, participants were quickly assisted in moving to the MVC and magnetic stimulation apparatus, for POST assessment.

VO_2 Assessment

After TT familiarization sessions, to characterize participants, VO_{2peak} was assessed via a maximal incremental test. A ramp protocol was utilized, that equated to 30 Watts/min which commenced after a 3-min baseline period, cycling at 0 Watts (Vanhatalo et al., 2007). As participant familiarity with cycling varied, a similar test was chosen to that used for participants unfamiliar with cycling (Williams et al., 2012). Expired gas was collected and analyzed every 15-s (S-3A/I (O2) and CD-3A (CO2), AEI Technologies Inc., Pittsburgh, PA). Prior to each test, gases were calibrated with known concentrations and flow calibrations were performed using a 3-L calibration syringe. Participants were encouraged throughout the final stages and the test ceased when the participant could not maintain a cadence above 60 rpm or volitional fatigue was achieved. Peak oxygen uptake was calculated as the highest 30-s mean VO_2 and peak power defined as the highest power at test conclusion. Subjective ratings for exertion (RPE) and quadriceps pain (pain scale) were measured every minute.

Transcutaneous Electrical Nerve Stimulation (TENS)

Participants were acutely treated with TENS (N602 ProTens; Everyway Medical Instruments, New Taipei City, Taiwan) for 30-min prior to the exercise protocol. Two TENS units were used so that the area of stimulation could be increased, with one unit (two channels) dedicated to each leg. Stimulation was applied through 50 × 90 mm adhesive electrodes (Allcare; Everyway Medical Instruments, New Taipei City, Taiwan). TENS electrode placement occurred after BASE MVC measurement, with sites shaved before placement. At the conclusion of stimulation, TENS electrodes were removed. For electrode placement, participants were asked to lay in a supine position and perform a knee extension. Two electrodes were placed on the superior portion of the quadriceps, inferior to the inguinal fold over the areas of contracted muscle bulk. Two electrodes were also placed

over the inferior portion of the quadriceps, one over the vastus lateralis and one over the vastus medialis. TENS was delivered in constant mode with settings fixed at a pulse width of 200-μs, and frequency of 82.6-Hz (Chen and Johnson, 2010). The duration and settings (pulse width and frequency) of TENS was based on previous studies showing the effect of TENS on PPT (Moran et al., 2011; Pantaleão et al., 2011). In this investigation, for the assessment of muscle recruitment and practically of exercise, application of TENS was administered prior to exercise in anticipation of approximately 30-min of post-stimulation analgesia (i.e., increased PPT) as observed in previous studies (Moran et al., 2011; Pantaleão et al., 2011). Prior to study commencement, settings for the TENS units were verified using an oscilloscope (Rigol DS1054). Following calibration checks, transparent tape was placed over these controls to prevent any adjustment. For TENS application, participants were instructed to adjust intensity (via manual dials) to a level of non-painful tingling below a level that evokes involuntary muscle contraction (Moran et al., 2011; Pantaleão et al., 2011; Son et al., 2016). Throughout 30-min TENS application, participants were asked to periodically increase intensity to ensure this remained at desired level. For PLAC condition, the same electrode placement was used, but stimulation intensity was set by the researcher. To appear that stimulation was present, the TENS unit power indicator was illuminated, although the equipment did not provide stimulation. For this condition, participants were told stimulation was set to a sub-sensory level (Cheing et al., 2002). For TENS condition, participants were informed that they were receiving high TENS and low TENS for PLAC condition. In both conditions, participants were advised that they may or may not feel any stimulation and in the absence of sensation, stimulation was still active and providing analgesic effects. To account for time taken for TENS application, during CONT condition, participants laid quietly in a supine position for 30-min.

Electromyography

Electromyographic (EMG) activity of six muscles (*vastus medialis*, *vastus lateralis*, *rectus femoris*, *biceps femoris*, *medial gastrocnemius, and gluteus maximus)* was recorded from the right lower limb via Ag/AgCl bipolar rectangular electrodes with a diameter of 30 × 20 mm and an inter-electrode distance of 20 mm (Blue Sensor N-00-S, Ambu Medicotest A/S, Ølstykke, Denmark). All signals were recorded continuously at 1500 Hz via a wireless receiver (Telemyo 2400 GT, Noraxon Inc., USA). Prior to electrode placement, the limb was shaved and abraded to minimize skin impedance, and appropriate electrode placement and functionality was checked before the start of each test. When the position of quadriceps electrodes overlapped with TENS electrode placement, electrode location was marked with a waterproof felt-tip pen to ensure reliable electrode replacement within session. All electrode sites were marked for reliable placement between subsequent testing sessions. To avoid artifacts from lower-limb movements, the electrodes were well secured with rigid tape. Raw EMG signals were band-pass filtered (12–500 Hz), were full-wave rectified and Root Mean Squared using Noraxon software (MyoResearch XP version 1.08.27). Mean RMS for individual muscles was analyzed for 20-s at 250-m intervals of TT distance. Individual muscle RMS values were summed to estimate general muscle electrical activity (RMS_{sum}) (Billaut et al., 2010), and is reported as a percent of the individual maximum value obtained during a pre-exercise sprint (O'Bryan et al., 2014).

Peripheral Magnetic Stimulation

Stimulation of the femoral nerve and quadriceps muscle was conducted using a magnetic stimulator (Magstim RAPID^2; JLM Accutek Healthcare, Homebush, NSW) and a double 70-mm coil (Katayama et al., 2004; Amann et al., 2007; Billaut et al., 2013). Force responses were obtained at 1 kHz from a calibrated load cell (Extran 2kN "S" beam, model SW1, Applied Measurement, Melbourne, Australia). The load cell was connected to a non-compliant strap, which was attached around the participant's leg just superior to the malleoli of the ankle. Voluntary force and neuromuscular testing was conducted at BASE, PRE (∼3-min pre-exercise) and POST (between 40 and 60-s post-exercise). Allowing for removal of TENS electrodes and application of EMG electrodes, the delay between the end of TENS application and PRE, was approximately 10-min. Time taken from the start of PRE assessment to TT start was ∼6-min.

To determine the area of stimulation associated with the largest quadriceps twitch (Q_{tw}), the coil head was positioned high onto the thigh, between the quadriceps muscle and the femoral triangle (Katayama et al., 2004; Amann et al., 2007; Billaut et al., 2013). This position was marked and kept the same for all trials. At BASE a warm-up was conducted with brief (∼5-s) submaximal voluntary contractions increasing to a MVC separated by ∼40-s. To indicate maximal depolarization of the femoral nerve, a ramp protocol of increasing stimulus intensity (from 70 to 100%) was used to achieve a plateau in BASE Q_{tw} (Katayama et al., 2004; Amann et al., 2007; Billaut et al., 2013). A near plateau was achieved in all participants at 95–100% stimulator output. For assessment, the stimulus power was set at 100% of maximum, and single stimuli were delivered. During a 5-s MVC of the quadriceps, the femoral nerve was stimulated (superimposed single stimuli) to determine the completeness of muscle activation (Katayama et al., 2004; Amann et al., 2007; Billaut et al., 2013). Stimulation was administered when the researcher visually identified a plateau in torque (Tofari et al., 2016). Three potentiated quadriceps twitches ($Q_{tw,pot}$) were obtained 5-s after the MVC. This procedure was performed three times at BASE and PRE (60-s of rest in between) such that nine $Q_{tw,pot}$ values were obtained, with the $Q_{tw,pot}$ averaged and analyzed for peak force. The procedure was only performed once at end-exercise to reduce post-exercise assessment time and limit recovery as much as possible (Billaut et al., 2013). Surface EMG was used to assess the membrane excitability via muscle action potentials (M-waves) during potentiated twitches, with peak to peak duration and amplitude measured. With single stimuli delivered during the MVCs the quadriceps central activation ratio (CAR) was calculated as the percentage of voluntary force obtained during the superimposed contraction, that is, CAR = MVC ÷ (MVC + stimulated force) (Kent-Braun, 1999; Tofari et al., 2016). Stimulation was delivered on visual identification of torque plateau, and in some cases, it occurred before or

after the torque plateau. To account for this, a correction equation was used where torque was averaged over 100-ms before superimposed peak (Marshall et al., 2014; Tofari et al., 2016). Due to technical problems, some CAR data were not included, and these participants have been removed from analysis with total $n = 10$.

Perceptual Scores

A 6–20 scale of rated perceived exertion (RPE) (Borg, 1970), an 11-point bipolar feeling scale (FS) (Hardy and Rejeski, 1989) and a pain scale (O'Connor and Cook, 2001) were used to assess perceived effort, affect and perceived quadriceps muscle pain. Prior to commencing the study, participants were given instructions and explained all scales. During familiarization and experimental TTs, ratings were recorded at every kilometer. At the conclusion of the study, participants were asked to subjectively access the effectiveness of TENS application. Participants were asked to rate on a 1–10 scale (1; a bit, 10; a lot), the relief from pain, and impact on performance that TENS application provided. Responses were received for both TENS and PLAC conditions.

Algometer

Measures of PPT were recorded for the pressure applied to the quadriceps with an algometer (FPX algometer; Wagner Instruments) with a 1-cm^2 application surface. Recordings displayed in kilograms of force (kgf) were taken from the left leg at BASE, in the last minute of TENS application (i.e., PRE exercise) and at POST. Assessment site was the midpoint between anterior superior iliac spine and head of the patella. Recordings were taken with pressure applied to relaxed muscle at a rate of 1 $kg{\cdot}cm^{-2}{\cdot}s^{-1}$. Participants verbally reported the first point when pain (distinct from pressure or discomfort) occurred, the algometry was immediately removed and corresponding measurement recorded as PPT (Moran et al., 2011).

Statistical Analysis

Conditions are defined as control (CONT), placebo with sham TENS application (PLAC), and an experimental condition with TENS application (TENS). All data was analyzed using SPSS (version 22, SPSS Inc., Chicago, IL.), with data are reported as mean ± SD. Tests for homogeneity of variances were performed to ensure normality of the cohort for dependent variables. With normality confirmed, two-way repeated measures ANOVAs (Condition × distance) were used to analyze changes in RPE, FS and pain. One-way repeated measures ANOVAs were used to analyze changes in total duration and average power, as well as 1-km duration and mean power. Two-way repeated measures ANOVAs (Condition × time) were used to analyze changes between BASE, PRE, and POST measurements for MVC, evoked response to magnetic stimulation and PPT. Percentage change between measurements (BASE-PRE and PRE-POST) was also investigated. To investigate pacing profiles, mean power and RMS_{sum} were analyzed over 250-m using repeated measures ANOVAs (Condition × distance). Given inter-participant differences in TTs, power output is expressed as a product of an individual's mass (W/kg). When sphericity was violated, Greenhouse-Geisser correction was used to adjust degrees of freedom. Paired samples *t*-tests were used to analyze subjective ratings of the TENS. For a significant main effect, *post-hoc* comparisons were examined with a one-way repeated measures ANOVA with Sidak multiple comparisons and paired sample *t*-tests. Statistical significance levels for all tests was set at $P < 0.05$. Effect sizes for ANOVA are reported as partial eta squared (η_p^2) with a small effect at 0.01, medium effect 0.06 and a large effect > 0.14. Effect sizes for *t*-tests are reported as Cohen's *d* with a small effect at 0.2, medium 0.5, and large >0.8 (Cohen, 1988).

RESULTS

Perceptual Scores and Pain

There was a significant distance effect for RPE, FS and pain ($P < 0.01$), with all conditions having an increase in RPE and pain, whilst having a decrease in FS (**Table 2**). No significant interaction (Condition × distance) effect was found for any perceptual score, RPE ($P = 0.58$), FS ($P = 0.68$), and perceived quadriceps pain ($P = 0.68$) (**Table 2**). However, FS showed a moderate effect size and RPE had a large effect size for the interaction and trial effects. There was also no change in pressure pain thresholds attributed to TENS (see **Tables 3, 4**). Post study subjective ratings of the effectiveness of TENS were different between PLAC and TENS conditions for the level of pain relief [$t_{(12)} = 2.68$ $P = 0.02$, $d = 1.55$] and positive influence on performance [$t_{(12)} = 4.68$ $P < 0.01$, $d = 2.70$] (**Figure 1**) with large effect sizes.

Overall Performance

There was no significant difference for mean power maintained over the TT [$F_{(1.8, 21.8)} = 2.0$, $P = 0.16$, $\eta_p^2 = 0.16$], although there was a large effect size (**Figure 2A**). Completion time between conditions was not significantly different [$F_{(2, 24)} = 1.9$, $P = 0.17$, $\eta_p^2 = 0.14$] and had a large effect size (**Figure 2C**). Average power for the first kilometer was not significantly different [$F_{(1.4, 16.6)} = 1.8$, $P = 0.21$, $\eta_p^2 = 0.13$] (**Figure 2B**), as was duration [$F_{(1.8, 21.5)} = 1.7$, $P = 0.20$, $\eta_p^2 = 0.13$] (**Figure 2D**), however both had a moderate effect size. Visual inspection of data (**Figure 2D**) shows a decreased time in TENS condition. Of all participants, nine had a reduced time in the TENS condition at one kilometer compared to CONT (−3.12 ± 5.58 s to CONT, 95% CI; −6.49, 0.26) (**Figure 3D**). For total TT duration eight out of 13 improved their time in TENS compared to CONT (−3.82 ± 12.96 s to CONT, 95% CI; −11.65, 4.01) (**Figure 3C**).

Pacing

There was no interaction (Condition × distance) effect for mean power over 250-m intervals [$F_{(38, 456)} = 0.77$, $P = 0.83$, $\eta_p^2 = 0.06$]. All TTs exhibited a similar pacing strategy, however there was greater variability in mean power in the first 750-m (**Figure 4A**). To investigate the variability at the start of the TTs, one-way repeated measures ANOVAs between distance points were conducted at 250-m [$F_{(1.78, 21.36)} = 0.55$ $P = 0.56$, $\eta_p^2 = 0.04$], 500-m [$F_{(1.66, 19.91)} = 1.39$ $P = 0.27$ $\eta_p^2 = 0.10$] and 750-m [$F_{(1.69, 20.33)} = 1.31$ $P = 0.29$, $\eta_p^2 = 0.10$]. Moderate effects

TABLE 2 | Subjective ratings for exertion, affect and pain at each kilometer of TTs.

	1- km	2-km	3-km	4-km	5-km	Trial effect	Distance effect	Interaction
RPE								
CONT	11.69 ± 1.44	12.85 ± 1.77	13.69 ± 1.93	14.88 ± 2.38	16.77 ± 1.96	$P = 0.12$ $\eta_p^2 = 0.16$	$P < 0.01$ $\eta_p^2 = 0.82$	$P = 0.58$ $\eta_p^2 = 0.16$
PLAC	11.85 ± 1.68	13.00 ± 1.41	14.31 ± 1.25	15.00 ± 1.58	17.31 ± 1.75			
TENS	12.23 ± 0.73	13.62 ± 1.12	14.54 ± 1.27	15.31 ± 1.65	17.15 ± 1.52			
FS								
CONT	1.69 ± 1.88	0.54 ± 1.71	−0.23 ± 1.92	−0.54 ± 2.15	−1.31 ± 2.46	$P = 0.72$ $\eta_p^2 = 0.03$	$P < 0.01$ $\eta_p^2 = 0.69$	$P = 0.68$ $\eta_p^2 = 0.06$
PLAC	1.15 ± 1.41	0.46 ± 1.56	−0.31 ± 1.84	−0.62 ± 2.14	−0.92 ± 2.84			
TENS	1.15 ± 1.57	0.38 ± 1.66	−0.31 ± 1.84	−0.85 ± 2.30	−1.31 ± 2.66			
PAIN								
CONT	2.19 ± 1.79	2.96 ± 2.09	3.92 ± 2.29	4.62 ± 2.73	6.12 ± 2.87	$P = 0.68$ $\eta_p^2 = 0.03$	$P < 0.01$ $\eta_p^2 = 0.73$	$P = 0.68$ $\eta_p^2 = 0.05$
PLAC	1.92 ± 1.31	2.92 ± 1.71	3.58 ± 1.89	4.38 ± 2.36	5.77 ± 2.62			
TENS	2.50 ± 2.21	2.81 ± 1.44	3.54 ± 1.90	4.35 ± 2.10	6.08 ± 2.36			

Data presented as mean ± SD. RPE, Rate of perceived exertion; FS, feeling scale; Pain, perceived quadriceps pain; CONT, control. PLAC, placebo. TENS, TENS condition. η_p^2, partial eta squared. Significant distance effects are not shown.

TABLE 3 | Raw changes in PPT, MVC, CAR, and Qtw,pot.

	BASE	PRE	POST	Trial effect	Time effect	Interaction
PPT (KGF)						
CONT	5.23 ± 3.93	4.99 ± 3.57	5.35 ± 3.42	$P = 0.47$ $\eta_p^2 = 0.07$	$P = 0.10$ $\eta_p^2 = 0.19$	$P = 0.90$ $\eta_p^2 = 0.02$
PLAC	4.76 ± 3.28	4.53 ± 2.60	5.35 ± 4.30			
TENS	5.17 ± 2.32	5.12 ± 2.80	5.63 ± 3.82			
MVC (N)						
CONT	262.70 ± 78.04	254.19 ± 72.33	207.90 ± 63.19*	$P = 0.97$ $\eta_p^2 < 0.01$	$P < 0.01$ $\eta_p^2 = 0.68$	$P < 0.05$ $\eta_p^2 = 0.18$
PLAC	259.52 ± 84.58	251.71 ± 66.23	211.54 ± 66.23*			
TENS	271.16 ± 68.04	251.73 ± 74.32#	197.55 ± 61.96*			
CAR						
CONT	95.26 ± 3.16	95.18 ± 2.65	94.66 ± 4.36	$P = 0.70$ $\eta_p^2 = 0.04$	$P = 0.81$ $\eta_p^2 = 0.02$	$P = 0.44$ $\eta_p^2 = 0.10$
PLAC	94.41 ± 3.48	94.89 ± 2.25	95.46 ± 2.41			
TENS	96.72 ± 3.42	95.17 ± 2.93	95.19 ± 2.31			
QTW,POT (N)						
CONT	28.76 ± 8.75	26.93 ± 8.45	14.23 ± 7.15*	$P = 0.10$ $\eta_p^2 = 0.17$	$P < 0.01$ $\eta_p^2 = 0.80$	$P = 0.22$ $\eta_p^2 = 0.11$
PLAC	28.10 ± 7.84	27.44 ± 8.04	15.39 ± 8.44*			
TENS	30.34 ± 10.09	28.38 ± 9.82#	16.34 ± 9.87*			

*Data presented as mean ± SD. BASE, baseline; PRE, pre-exercise; POST, post-exercise; PPT, pressure pain threshold; MVC, maximal voluntary contraction; CAR, Quadriceps central activation ratio, for CAR n = 10. Qtw,pot, potentiated quadriceps twitch. CONT, control; PLAC, placebo; TENS, TENS condition; η_p^2, partial eta squared; Time point effects * difference to BASE and PRE measurement, # difference to BASE measurement.*
Post-hoc tests for M-wave amplitude time effects revealed no difference between means.

sizes were observed at 500-m and 750-m. EMG data followed a similar pattern to power output with no significant interaction (Condition × distance) effect [$F_{(40, 480)} = 1.01$, $P = 0.46$, $\eta_p^2 = 0.08$] (**Figure 4B**). Furthermore, greater variability between TTs was shown with large effects at 250-m [$F_{(1.5, 18.3)} = 2.31$, $P = 0.14$, $\eta_p^2 = 0.16$], 500-m [$F_{(1.7, 20.3)} = 2.05$, $P = 0.16$, $\eta_p^2 = 0.15$] and 750-m [$F_{(1.6, 18.8)} = 2.01$, $P = 0.17$, $\eta_p^2 = 0.14$].

Fatigue Measurements

An exercise-induced reduction in MVC was recorded PRE to POST in all conditions (see **Table 3**), with only TENS application resulting in a significant reduction between BASE and PRE measurements. This difference was not apparent when expressed as a percentage change from BASE-PRE measurements (see **Table 4**). Rather, MVC revealed a main effect of time with an exercise-induced reduction in all conditions, but no significant interaction (Condition × time). Mean Qtw,pot followed MVC values with all conditions having an exercise-induced reduction PRE-POST (see **Tables 3, 5**). Only TENS condition had a reduction in raw Qtw,pot between BASE and PRE measurements, but a non-significant percentage change (BASE-PRE) (see **Table 5**). There was no reduction in quadriceps CAR as a raw value (see **Table 3**) or as a percentage change (see **Table 4**). Percentage change responses to magnetic stimulation

TABLE 4 | Percentage changes in PPT, MVC and CAR.

	BASE—PRE Exercise	PRE Exercise—POST Exercise	Trial effect	Time effect	Interaction
PPT (KGF)					
CONT	−2.67 ± 19.07	6.65 ± 33.34	$P = 0.98$ $\eta_p^2 < 0.01$	$P = 0.08$ $\eta_p^2 = 0.25$	$P = 0.99$ $\eta_p^2 < 0.01$
PLAC	−1.71 ± 10.18	6.81 ± 22.49			
TENS	−2.56 ± 16.34	5.59 ± 22.37			
MVC (N)					
CONT	−2.51 ± 4.95	−17.46 ± 10.35*	$P = 0.04$ $\eta_p^2 = 0.24$	$P < 0.01$ $\eta_p^2 = 0.66$	$P = 0.76$ $\eta_p^2 = 0.02$
PLAC	−2.18 ± 7.05	−14.75 ± 12.89*			
TENS	−8.27 ± 8.92	−20.60 ± 13.74*			
CAR					
CONT	−0.06 ± 1.81	−0.54 ± 3.90	$P = 0.38$ $\eta_p^2 = 0.10$	$P = 0.65$ $\eta_p^2 = 0.02$	$P = 0.50$ $\eta_p^2 = 0.07$
PLAC	0.59 ± 3.13	0.65 ± 3.34			
TENS	−1.54 ± 3.20	0.07 ± 2.53			

*Data presented as mean percentage change between measurements ± SD. BASE, baseline; PRE, pre-exercise; POST, post-exercise; PPT, pressure pain threshold; MVC, maximal voluntary contraction; CAR, Quadriceps central activation ratio, for CAR n = 10. CONT, control; PLAC, placebo; TENS, TENS condition. η_p^2, partial eta squared; Time point effects * difference to BASE—PRE. Post-hoc tests for MVC trial effect revealed no significant difference.*

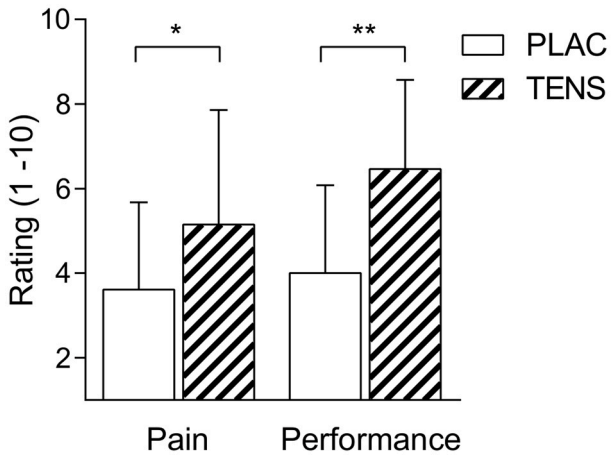

FIGURE 1 | Mean ± SD TENS belief ratings. Participants ratings of pain relief (Pain) and subsequent influence on performance (Performance). PLAC, placebo; TENS, TENS condition. Ratings are expressed on a 1–10 scale (1; a bit, 10; a lot). * indicates a difference for pain, $P < 0.05$. ** indicates a difference for performance $P < 0.01$.

are shown in **Table 5**. There was an exercise-induced reduction in all measures, except M-wave variables.

DISCUSSION

This is the first study to investigate the influence of TENS administered prior to a 5-km TT, on exercise-induced pain, with a focus on influencing exercise intensity and increasing performance. It was hypothesized, TENS application would adjust the threshold for sensing pain, allowing for increased power output for the same rating of pain. As hypothesized, TENS failed to significantly influence within-exercise subjective pain ratings, but no significant effect on pacing and performance was observed. However, a large effect size for TT duration and mean power indicate a possible difference in favor of TENS compared with PLAC. At the start of trials, moderate to large effect sizes indicate differences in power output and EMG data. This suggests a possible influence of TENS on anticipation and, consequently, the selection of an initial exercise intensity.

Pacing and Performance

The application of TENS was associated with a large effect on EMG and a moderate effect on power output at the start of the TT, suggesting that TENS application may have influenced participant's anticipation of the task (see **Figures 2**–**4**). Afferent feedback is important in setting an initial exercise intensity (Tucker, 2009). This is evident with reduced starting exercise intensities when homeostasis is threatened (Amann et al., 2007; Schlader et al., 2011). Consequently, exercise intensity is reduced in order to limit excessive levels of fatigue and maintain homeostasis. Our results seem to indicate that TENS application possibly limits afferent feedback activity prior to exercise, resulting in greater muscle recruitment (as inferred from EMG data) and power output at the start of the TT. This supports research indicating a higher intensity is chosen when afferent information is removed or modified (Amann et al., 2009). It is likely that this occurs due to the lack of feedback to inform on potential or actual muscle damage and metabolic activity. This would indicate to the brain that more work can be done without indication of serious consequences, creating a greater neural drive to exercising muscle.

TENS is shown to influence motor neuron excitability (Hopkins et al., 2002; Pietrosimone et al., 2009). In this perspective, it is interesting to note that TENS increased EMG activity, a surrogate for muscle recruitment (Ansley et al., 2004), at the start of the TT. This enhanced central neural drive was concomitant to a higher power output. Importantly, TENS did not produce any change in M-wave responses from BASE to PRE, indicating no change in resting muscle function before the cycling TT (see **Table 5**). However, another possible explanation

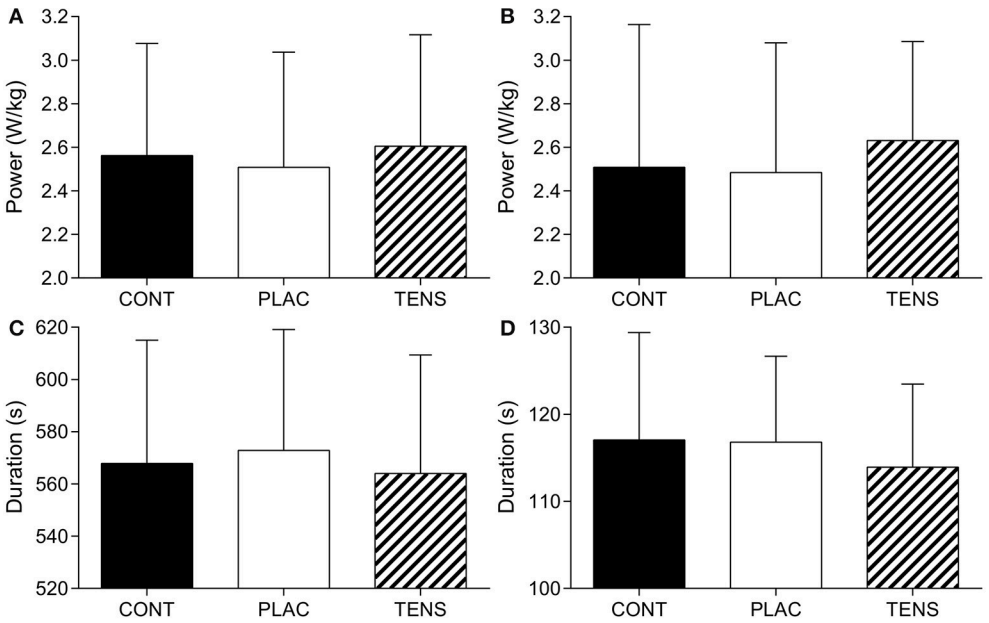

FIGURE 2 | Mean ± SD TT performance measures. TT mean power output (W/kg) **(A)**, 1-km mean power output (W/kg) **(B)**, TT duration (s) **(C)**, and 1-km duration (s) **(D)**. CONT, control; PLAC, placebo; TENS, TENS condition.

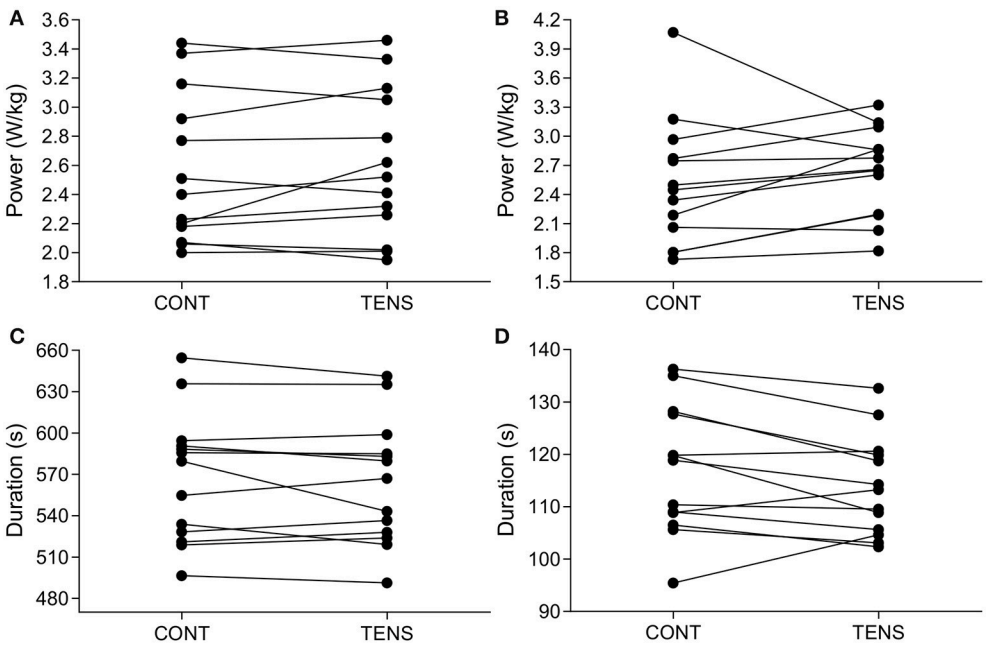

FIGURE 3 | Individual changes for TT performance measures. TT mean power output (W/kg) **(A)**, 1-km mean power output (W/kg) **(B)**, TT duration (s) **(C)**, and 1-km duration (s) **(D)**. CONT, control; PLAC, placebo; TENS, TENS condition.

for this result is that neuromuscular electrical stimulation can change voluntary muscle recruitment patterns, allowing for non-sequential activation of muscle fibers (Gregory and Bickel, 2005). Yet, stimulation protocols showing these effects are different to the method utilized in this study. It is possible this effect occurred, creating a poor recruitment of muscle after TENS stimulation which may require a greater neural drive to be produced. Alternatively, there is an association between TENS and greater local blood flow (Hallen et al., 2010). This may lead to a greater activation of type I muscle fibers which are related to cycling efficiency, thus increasing EMG activity (Coyle et al., 1992). These possible factors may explain the observed increase

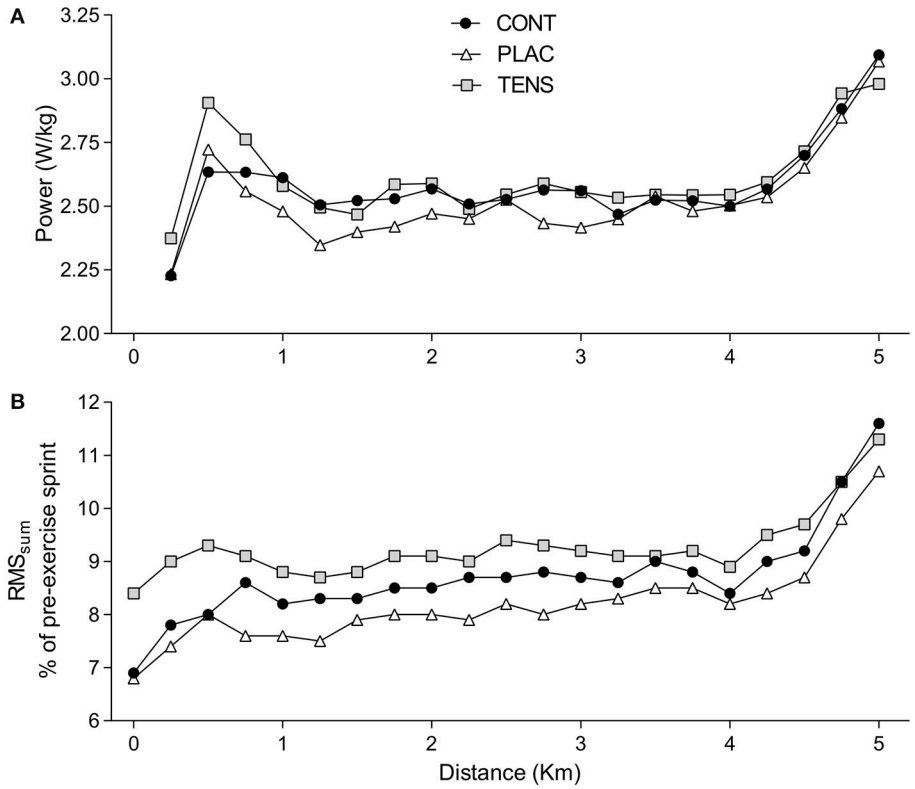

FIGURE 4 | TT pacing measures. Group mean power output **(A)** and electromyography (RMSsum) profiles **(B)**. Mean power output is averaged over 250-m intervals. RMSsum data is reported as a percentage of a pre-exercise sprint value, RMSsum was measured for 20-s at 250-m intervals. Error bars have been excluded for clarity. CONT, control; PLAC, placebo; TENS, TENS condition.

in EMG readings, allowing for higher muscle recruitment and greater power output during the early part of the TENS condition TT. However, we cannot identify the primary source from this investigation.

After the initial differences, all TTs exhibit a similar power output and EMG readings beyond one kilometer (**Figure 4**). An initial aggressive pacing strategy would likely assist performance in shorter tasks such as a 5-km TT (Abbiss and Laursen, 2008). This would create greater mechanical and chemical stimuli likely to trigger conscious awareness and influence pain perceptions (Swart et al., 2012; Edwards and Polman, 2013; Mauger, 2014). It is possible that this occurred in our study at approximately one kilometer, with increased exercise-induced stimuli diminishing the effectiveness of TENS. This may indicate that afferent information from active skeletal muscles is now unaffected and the participant uses this to pace performance. It is also likely at this stage of the intense exercise, feedback from a number of different sources, not just the active muscle, is pushing the individual close to their tolerance limit (Hureau et al., 2016). For these reasons, even with the non-significant differences in intensity at the start of the trial, it is not surprising observe similar subjective responses for pain, feeling and exertion in all conditions. Therefore, these results support the theory that exercise is regulated in part by afferent feedback to a perceived pain threshold (Mauger et al., 2010), which presumably plays a role in a global sensory tolerance limit (Hureau et al., 2016).

Practical Implications

Application of TENS did not provide any overall performance improvement for a 5-km TT. However, there was a large effect size for TT duration and moderate effect size for duration of the first kilometer (see **Figures 2**, **3**). These results provide some support for the potential of TENS to increase exercise performance. Participants completed the first kilometer of the TTs within a range of 95.44–136.28-s, and the difference between TENS and CONT conditions being -3.12 ± 5.58-s. Therefore, this research identifies that any possible benefit of TENS administered prior to exercise may be limited to events of $\leq$2 min and where exercise-induced pain is localized. However, the possible reduction in pain to increase exercise intensity poses ethical concerns for athlete safety. Administering an analgesic intervention will augment stimuli that warns of potential muscle damage, this creates the potential for a greater risk of injury through increased exercise intensity. Our study found no significant impact on exercise intensity, but also no indication of greater exercise-induced fatigue due to TENS application (i.e., PRE-POST measurement, see **Tables 3**–**5**). It may be of benefit for future investigations to confirm if effects

TABLE 5 | Percentage changes in evoked responses to magnetic stimulation.

	BASE—PRE Exercise	PRE Exercise—POST Exercise	Trial effect	Time effect	Interaction
QTW,POT (N)					
CONT	−5.09 ± 13.11	−46.75 ± 17.59*	$P = 0.48$ $\eta_p^2 = 0.06$	$P < 0.01$ $\eta_p^2 = 0.83$	$P = 0.38$ $\eta_p^2 = 0.08$
PLAC	−2.25 ± 8.02	−44.56 ± 20.52*			
TENS	−5.92 ± 9.11	−41.43 ± 26.33*			
MRFD (N.S^{-1})					
CONT	−3.91 ± 11.92	−56.73 ± 19.73*	$P = 0.48$ $\eta_p^2 = 0.06$	$P < 0.01$ $\eta_p^2 = 0.86$	$P = 0.24$ $\eta_p^2 = 0.11$
PLAC	−2.62 ± 9.31	−54.14 ± 20.78*			
TENS	−3.35 ± 16.81	−48.54 ± 23.32*			
CT (S)					
CONT	−0.61 ± 12.50	−23.78 ± 13.92*	$P = 0.44$ $\eta_p^2 = 0.07$	$P < 0.01$ $\eta_p^2 = 0.65$	$P = 0.33$ $\eta_p^2 = 0.09$
PLAC	−1.77 ± 18.50	−20.24 ± 12.60*			
TENS	4.74 ± 12.30	−23.87 ± 11.37*			
MRR (N.S^{-1})					
CONT	−6.03 ± 19.81	−59.36 ± 15.22*	$P = 0.22$ $\eta_p^2 = 0.12$	$P < 0.01$ $\eta_p^2 = 0.88$	$P = 0.78$ $\eta_p^2 = 0.01$
PLAC	−2.70 ± 23.36	−53.31 ± 22.34*			
TENS	−0.40 ± 10.04	−55.59 ± 23.30*			
$RT_{0.5}$ (N.S^{-1})					
CONT	−5.09 ± 13.25	−23.32 ± 19.02*	$P = 0.19$ $\eta_p^2 = 0.13$	$P < 0.01$ $\eta_p^2 = 0.56$	$P = 0.12$ $\eta_p^2 = 0.16$
PLAC	−3.15 ± 23.57	−21.98 ± 18.74*			
TENS	7.09 ± 17.06	−26.85 ± 15.85*			
M-WAVE AMPLITUDE (MV)					
CONT	2.63 ± 18.83	4.75 ± 13.50	$P = 0.22$ $\eta_p^2 = 0.12$	$P = 0.07$ $\eta_p^2 = 0.26$	$P = 0.45$ $\eta_p^2 = 0.06$
PLAC	−0.66 ± 7.73	3.03 ± 10.91			
TENS	1.89 ± 16.14	13.85 ± 21.50			
M-WAVE DURATION (MS)					
CONT	−4.71 ± 14.21	−7.11 ± 24.17	$P = 0.17$ $\eta_p^2 = 0.14$	$P = 0.49$ $\eta_p^2 = 0.04$	$P = 0.25$ $\eta_p^2 = 0.11$
PLAC	1.07 ± 17.25	7.96 ± 16.49			
TENS	−9.07 ± 37.90	−7.05 ± 27.86			

*Data presented as mean percentage change between measurements ± SD. BASE, baseline; PRE, pre-exercise; POST, post-exercise; Qtw,pot, potentiated quadriceps twitch; MRFD, maximal rate of force development; CT, contraction time; MRR, maximal rate of relaxation; $RT_{0.5}$, one-half relaxation time; CONT, control; PLAC, placebo; TENS, TENS condition; η_p^2, partial eta squared. Time point effects * difference to BASE—PRE.*

of TENS application occur in elite populations, to highlight any potential benefits or concerns of TENS use.

One possible future investigation could look at potential benefits of TENS use within a task. We tested the use of TENS prior a task, with a 5-km TT chosen in anticipation that exercise would be conducted in a proposed post-stimulation analgesic period. Compared to a longer task (e.g., 20-km TT), a greater exercise intensity would be observed in a 5-km TT, and therefore, a greater nociceptive stimulus is expected. Accordingly, when pain is greater, it may be harder to distinguish small changes in pain perceptions that an intervention may provide. Also theoretically, it is possible that TENS could be beneficial for longer duration tasks which are more reliant on afferent feedback for regulation (Tucker, 2009; Mauger et al., 2010), and where pain perceptions are expected to be less prominent. Furthermore, TENS is more likely to reduce pain perceptions when stimulation is active. Therefore, future investigations into the possible use of TENS to enhance exercise performance may look at utilizing TENS during trials of greater length. Ethically, it is unlikely TENS could be used within a sporting event due to doping concerns, but there may be merit in use of TENS as a within-exercise training intervention (Hughes et al., 2013).

Limitations

There are several limitations in this study. Perceptions of TENS for pain relief and influence on performance were greater than PLAC condition (see **Figure 1**). Measures were taken to minimize the influence of any placebo effect of TENS. However, participants would have clearly felt a difference in sensation between TENS and PLAC conditions. Furthermore, participants were made aware of the aims of the study, and informed they were receiving high or low TENS, but not aware of which intervention was placebo. This could have had implications on the results shown by TENS (Son et al., 2016). Placebo effects have been shown to influence exercise pain perceptions (Benedetti et al., 2007) but also the ability to produce force (Broatch et al., 2014). As participant's perceptions of TENS effectiveness on pain relief and performance were increased post study compared to PLAC condition (**Figure 1**), this could have produced changes at the start of the 5-km TT.

With physiological differences between participants, it is possible that there are responders and non-responders to this type of intervention (**Figure 3**). For example, the amount of subcutaneous fat may affect the amplitude of stimulation to afferent fibers (Hughes et al., 2013). Hence, those with lower body fat may not be able to increase the amplitude of stimulation to a level that will stimulate deeper tissue (Hughes et al., 2013). This could possibly result in different levels of stimulation between participants, leading to differing levels of afferent information, and effect on performance. This is a limitation of the study, as the final current intensity was not recorded, we cannot confirm the dose received by participants. However, application of TENS was adjusted to an individual's own sensory threshold, in effect an individual's tolerance of the stimulation. It has to be noted that applying a TENS intensity higher than what a participant can tolerate would be unethical. However, it could be speculated that those who responded to the intervention (i.e., higher initial power output) (**Figures 2**, **3**) were able to tolerate a higher current intensity during TENS application.

The potential of TENS to affect exercise-induced pain was based on previous research in pain-free individuals that influenced PPT (Moran et al., 2011), but also restored muscle strength when pain was induced (Son et al., 2016). Recent research however, has indicated a possibility for different subgroups of group III and IV muscle afferents which are sensitive to distinct metabolites. One subgroup is likely to respond to intramuscular metabolites associated with aerobic exercise, whilst another responds to noxious levels of metabolites (e.g., hypertonic saline) associated with ischemic contractions (Amann et al., 2015). These differing characteristics of muscle afferents may be a possible reason why TENS failed to significantly change exercise intensity within this study. Whilst in comparison, analgesic effects are demonstrated during exercise when pain is induced (Son et al., 2016).

Reductions in MVC and Qtw,pot were observed from BASE to PRE for TENS condition (see **Table 3**; Moran et al., 2011; Amann et al., 2015; Son et al., 2016), although percentage reductions were not significantly different (see **Tables 3, 4**). This likely resulted from the stimulation intensity being close to the threshold for muscle contraction. However, despite this apparent fatigue, it dissipated quickly as power was greatest in TENS condition early in the TT (see **Figure 4A**). Therefore, a possible limitation of prior to exercise use of TENS may be the application settings. Application duration should be limited and higher intensities avoided as this may induce peripheral fatigue that is detrimental to performance. Furthermore, PRE assessment was conducted after the cycling warm-up, which may have contributed to the reduced voluntary force, thus disguising the true influence of TENS on muscle strength properties. Settings for TENS were based on previous research indicating this would activate the gate control of pain and reduce feedback from group III and IV afferents (Moran et al., 2011). However, amplitude, stimulation duration, prior use and tolerance of opioids can compromise TENS effectiveness (Sluka et al., 2013). Participants currently taking pain medication were excluded from the study, but the prior use of opioids was not recorded. Typically, peak effects for analgesia provided by TENS will be greater when stimulation is active, or immediately after cessation (Moran et al., 2011; Vance et al., 2012). For this research, we conducted a 5-km TT, as we anticipated post-stimulation effects for approximately 30-min would be present (Moran et al., 2011). As expected, within-exercise pain perceptions were similar, but there is an absence of significant differences in exercise intensity. Therefore, by administering the intervention prior to exercise, it is possible that analgesic effectiveness of TENS for this mode of exercise is reduced.

Although the current study has several limitations, the overall multidisciplinary approach allows for assessment of a number of variables related to exercise performance. The aim of the study was to assess the efficacy of TENS on exercise performance, with a focus on exercise-induced pain. Although there were no significant differences in pain and performance, the reporting of multiple variables related to exercise intensity, fatigue but also psychological aspects represent strengths of this study.

Conclusions

In conclusion, this study found no significant effects of TENS administered prior to exercise on 5-km TT performance, although similar pain perceptions were observed. This casts doubt on the effectiveness of the application of TENS prior to exercise to modify afferent feedback and influence perceptions of exercise-induced pain. However, there are indications TENS application may influence neural drive and power output at the start of a 5-km TT. Aside from any potential changes in pain perceptions, other possible reasons for this include psychological belief in the intervention and altered muscle recruitment. Future research could investigate the effectiveness of TENS on modifying the sensation of exercise-induced muscle pain, with a focus on application during other forms of aerobic exercise.

AUTHOR CONTRIBUTIONS

All authors contributed to the study design. AH collected data, with AH, FB and RP conducted the initial data analysis. First draft of manuscript was written by AH, with all authors reviewing the manuscript and approving the final version.

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Cerebral Regulation in Different Maximal Aerobic Exercise Modes

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We investigated cerebral responses, simultaneously with peripheral and ratings of perceived exertion (RPE) responses, during different VO_{2MAX}-matched aerobic exercise modes. Nine cyclists (VO_{2MAX} of 57.5 ± 6.2 ml·kg^{-1}·min^{-1}) performed a maximal, controlled-pace incremental test (MIT) and a self-paced 4 km time trial (TT_{4km}). Measures of cerebral (COX) and muscular (MOX) oxygenation were assessed throughout the exercises by changes in oxy- (O_2Hb) and deoxy-hemoglobin (HHb) concentrations over the prefrontal cortex (PFC) and vastus lateralis (VL) muscle, respectively. Primary motor cortex (PMC) electroencephalography (EEG), VL, and rectus femoris EMG were also assessed throughout the trials, together with power output and cardiopulmonary responses. The RPE was obtained at regular intervals. Similar motor output (EMG and power output) occurred from 70% of the duration in MIT and TT_{4km}, despite the greater motor output, muscle deoxygenation (↓ MOX) and cardiopulmonary responses in TT_{4km} before that point. Regarding cerebral responses, there was a lower COX (↓ O_2Hb concentrations in PFC) at 20, 30, 40, 50 and 60%, but greater at 100% of the TT_{4km} duration when compared to MIT. The alpha wave EEG in PMC remained constant throughout the exercise modes, with greater values in TT_{4km}. The RPE was maximal at the endpoint in both exercises, but it increased slower in TT_{4km} than in MIT. Results showed that similar motor output and effort tolerance were attained at the closing stages of different VO_{2MAX}-matched aerobic exercises, although the different disturbance until that point. Regardless of different COX responses during most of the exercises duration, activation in PMC was preserved throughout the exercises, suggesting that these responses may be part of a centrally-coordinated exercise regulation.

Keywords: near-infrared spectroscopy, brain oxygenation, exercise tolerance, central fatigue, peripheral muscle fatigue

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INTRODUCTION

Studies originally suggested that cerebral oxygenation (COX) measured at the prefrontal cortex (PFC) is involved with the capacity to perform maximal aerobic exercises (Nielsen et al., 1999; Rupp and Perrey, 2008). Results obtained during controlled-pace, maximal incremental exercise test (MIT) at sea level indicated an increase in COX up to ≈80% of the peak power output (W_{PEAK}), but

a decrease from this intensity toward the exercise endpoint (Rupp and Perrey, 2008; Subudhi et al., 2008). This reduction in COX during the last 20% of the MIT, after the second ventilatory threshold (VT_2), matched a pronounced increase in muscle recruitment as measured by electromyography (EMG), suggesting that the ability to increase motor output at maximal levels was related to the deoxygenation in PFC areas (Rupp and Perrey, 2008; Perrey, 2009). In addition, this may have suggested that activation of the primary motor cortex (PMC) was not impaired, despite the COX drop in PFC areas. Therefore, at least at sea level, reductions in COX may reflect the ability of the central nervous system (CNS) to maximally increase muscle recruitment and power output at the closing stages of a MIT.

Using a different maximal exercise mode, Billaut et al. (2010) showed a similar reduction in COX at the end-spurt of a 5 km self-paced running time trial (i.e., the last 10% of the trial), which time-matched an increase in EMG. Furthermore, COX also increased during the first half of the exercise, as observed in MIT (Billaut et al., 2010). Hence, evidences provided by independent studies suggested that reductions in COX measured at PFC matched, but did not limit the increase in motor output (i.e., EMG and power output) at the closing stages of maximal controlled-pace (Rupp and Perrey, 2008) or self-paced exercise (Billaut et al., 2010). They further suggested that deoxygenation in PFC did not impair the activation in motor cortex areas during maximal motor output in these exercises.

Responses of COX may be part of a process of exercise regulation. Robertson and Marino (2016) have suggested that the PFC has a vital role for the exercise regulation and tolerance, as it integrates afferents from peripheral organs and muscles into emotional messages used to guide the decision-making in exercise. Homeostatic disturbances during exercise are integrated into stimulus motivationally significant in PFC regions, guiding the decision to continue or stop the exercise (Craig, 2002; Robertson and Marino, 2016). This regulation possibly tax the PFC areas (i.e., metabolic cost), as the interpretation of the benefit of activating motor cortex areas while tolerating unpleasant sensations triggered by homeostatic disruption is processed by PFC areas (Meeusen et al., 2016). In this sense, the capacity to perform maximal aerobic exercises could be linked to the ability of the PFC to articulate emotional messages while preserving the PMC activity, even under great metabolic disruption. In fact, independent studies using electroencephalography technique (EEG) have shown that activation in PMC can be preserved during MIT (Brümmer et al., 2011; Robertson and Marino, 2015), despite the pronounced PFC deoxygenation and homeostatic disruption that occur after the VT_2, during the last 20% of the trial (Rupp and Perrey, 2008). However, if the same response would be found in maximal self-paced exercises is yet to be verified. A recent study found a 6–12% reduction in maximal voluntary activation superimposed by transcranial magnetic stimulation (TMS) after different self-paced cycling trials, thereby suggesting that some impairment in PMC activation occurred during the exercise (Thomas et al., 2015). Therefore, an experimental setup including PFC oxygenation and PMC activation measures during maximal aerobic exercises may be insightful.

The process of exercise regulation may depend on the exercise mode under consideration, as the ability to tolerate homeostatic disruption seems to be different between controlled-pace and self-paced exercises (Lander et al., 2009; Robertson and Marino, 2016). A previous study found greater homeostatic disruption in submaximal controlled-pace exercise, as the core temperature, blood lactate concentrations and EMG were higher than in a power-matched, submaximal self-paced exercise. Accordingly, individuals perceived the controlled-pace exercise as more challenging, given the higher ratings of perceived exertion (RPE) in this exercise mode. However, it remains allusive if the different homeostatic disturbances between controlled-pace and self-paced exercise would affect the ability of the PFC to integrate these responses into emotional messages, thus impairing the PMC activation necessary to increase the motor output at maximal levels.

Therefore, the present study explored cerebral responses (PFC oxygenation and PMC activation) to maximal, controlled-pace and self-paced exercise. We further measured peripheral [muscle oxygenation (MOX)], cardiopulmonary and RPE responses to these exercises. We hypothesized that regardless of reductions in COX at the closing stages of the exercises, PMC activation would be preserved in both the exercise modes, even though the motor output was maximal. However, we did not known about differences in cerebral responses to these exercises.

MATERIALS AND METHODS

Participants and Experimental Design

Nine trained male road cyclists (32.9 ± 7.3 years old, body mass of 75.9 ± 9.0 kg, height of 175.7 ± 5.9 cm, and body fat of 10.5 ± 5.2%), experienced ($\geq$3 years) in short distance cycling time trials, volunteered to participate in this study. They were non-smokers with no neuromuscular or cardiopulmonary disorders. The procedures were explained to the participants before they were asked to sign an informed consent form. The experimental protocol conformed to the Declaration of Helsinki, being previously approved by the University of São Paulo's Ethics Committee (0023.0.342.000-10).

It is important to note that a previous study compared controlled-pace and self-paced exercises by using a submaximal power-matched exercise design (Lander et al., 2009). Instead, in the present study individuals were required to perform maximal motor output in both the exercise modes, so that we used a VO_{2MAX}-matched exercise design to accomplish this goal. In this regard, whole body exercises that elicit VO_{2MAX} (i.e., maximum oxygen uptake) values have been interpreted as maximal aerobic exercises, according to the traditional maximal aerobic exercise

Abbreviations: COX, cerebral oxygenation; DHb, hemoglobin difference; EEG, electroencephalography; EMG, electromyography; NIRS, near-infrared spectroscopy; HHb, deoxy-hemoglobin; MIT, maximal incremental exercise test; MOX, muscular oxygenation; O_2Hb, oxy-hemoglobin; $PeTCO_2$, end-tidal carbon dioxide tension; PFC, prefrontal cortex; PMC, primary motor cortex; RF, rectus femoris; RMS, root mean square; RPE, ratings of perceived exertion; THb, total hemoglobin; TT_{4km}, 4 km time trial; VE, ventilation; VL, vastus lateralis; VCO_2, carbon dioxide production; VO_2, oxygen uptake; VO_{2MAX}, maximal oxygen uptake; W_{PEAK}, peak power output.

performance paradigm (Bassett and Howley, 2000; Levine, 2008). Thus, we utilized a MIT routinely used to assess VO_{2MAX} as maximal controlled-pace exercise, because this exercise mode requires the maintenance of a target pedal cadence while the workload is increased. In contrast, a 4 km cycling time trial (TT_{4km}) was chosen as maximal self-paced aerobic exercise, as this exercise mode provides VO_2 values similar to VO_{2MAX} values obtained in MIT (Mauger et al., 2009; Williams et al., 2012). However, individuals are free to pace themselves during this exercise mode, while the distance is completed within the fastest possible time.

The cyclists were habituated to perform laboratory MIT and TT_{4km}, as they took part in previous study that used these physical exercise tests. Thus, the experimental design comprised the following sequential visits to the laboratory: (1) first visit to measure body mass, height, pectoral, abdominal, and thigh skinfold thickness (Harpenden®, West Sussex, UK), and to familiarize them with the experimental procedures (bicycle, cycle-simulator, Borg's scale, cerebral, and muscular measures); (2) second visit to perform a controlled-pace MIT; (3) third visit to perform a self-paced TT_{4km}. Important, previous studies have provided evidences for TT_{4km} reliability in trained and untrained individuals (Mauger et al., 2009; Williams et al., 2012). All tests were performed at the same time of the day, in a laboratory with temperature (21°C) and humidity (50–60%) controlled. Visits 2 and 3 were interspersed by ≈7 days, and the entire study lasted for ≈15 days. The cyclists were encouraged to maintain the training schedule (intensity and volume) throughout the study and to avoid exercises, alcohol or stimulant beverages for the 24 h before the tests.

Throughout the MIT and TT_{4km}, COX, and MOX were continuously measured through near-infrared spectroscopy (NIRS) technology, while PMC and muscle activation were continuously assessed through EEG and EMG, respectively. Furthermore, cardiopulmonary variables were continuously sampled, while RPE was obtained at regular intervals.

Exercise Protocols

After being prepared for experimental procedures, cyclists were accommodated on the bicycle; then they closed their eyes and remained in absolute rest for 2 min for baseline measurements. Immediately after the baseline assessments, a standard 7 min warm-up, consisting of a 5 min self-paced exercise and a 2 min controlled-pace exercise (cycling at 100 W with pedal cadence of 80 rpm), was performed before the MIT and TT_{4km} exercises. The 2 min controlled-pace exercise warm-up was used to normalize the exercise EMG data. The MIT was initiated immediately after the standard warm-up, while cyclists were still cycling at 100 W (80 rpm) during the controlled-pace warm-up. The power output was increased 25 $W \cdot min^{-1}$ until exhaustion, while the pedal cadence was maintained at 80 rpm. Exhaustion was determined when the pedal cadence dropped below the 80 rpm, despite strong verbal encouragement. Similarly, after the standard warm-up, cyclists immediately began the TT_{4km} while receiving verbal encouragement to complete the trial as fast as possible. Elapsed time and distance were available throughout the test, so that cyclists were free to pace themselves in the TT_{4km}.

Instruments

The controlled-pace MIT and the self-paced TT_{4km} were performed on a speed bicycle (Giant®, Thousand Oaks, CA, USA) attached to a cycle-simulator (Racer Mate®, Computrainer, Seattle, WA, EUA), and equipped with a crank (SRM®, PowerControl 7, Jülich, Köln, Germany) that provided power output (W) data at a 2 Hz frequency. The cycle-simulator was calibrated before every test, according to the manufacturer's instructions. The validity and reliability of these instruments have been reported elsewhere (Duc et al., 2007; Peveler, 2013).

Changes in COX and MOX were assessed through alterations in oxy-hemoglobin (O_2Hb) and deoxy-hemoglobin (HHb) concentrations throughout the experimental set-up, via NIRS (CW6- TechEn®, Milford, MA, USA) at a sampling rate of 25 Hz. This system monitors the tissue absorption via optical fibers optodes with light sources and detectors. The COX was monitored with optodes placed over the prefrontal lobe at the Fp1 position, according to the international 10–20 system (Subudhi et al., 2008; Billaut et al., 2010; Santos-Concejero et al., 2015). The MOX was monitored over the right vastus lateralis (VL) muscle, the optodes were positioned at ∼15 cm proximal and 5 cm lateral from the superior border of the patella (Subudhi et al., 2009); the adipose thickness was ≤5 mm at this point. Adhesive tape was used to fix the optodes into a specific plastic holder, projected with a 4.5 cm inter-optode distance to reduce artifacts during the exercise. Briefly, this inter-optode distance was chosen because it is within the range suggested to obtaining reliable NIRS measures in muscle and brain. For example, a 4.5 cm inter-optode distance has been suggested to be unaffected by changes in scalp blood flow or adipose tissue thickness (Hamaoka et al., 2007; Ekkekakis, 2009). Moreover, a modified Beer-Lambert law, based on optical densities from two continuous wavelengths of 690 and 830 nm, calculated the μmol changes in tissue O_2Hb and HHb, respectively (Perrey, 2008; Ekkekakis, 2009).

The activation in PMC was monitored throughout the experimental set-up by an EEG unit (NicoletOne V32®, Viasys Healthcare Inc., Madison, WI, USA). After measuring circumferences, frontal and sagittal planes to ensure the electrode placement according to the EEG international 10–20 system, active electrode (Ag-AgCl) was fixed with medical strips and conductive gel at the Cz position (referenced to mastoid), after exfoliation and cleaning. This position was selected to represent the cortical area responsible for motor commands to lower limbs muscles, according to the human motor homunculus (Nielsen et al., 2001; Nybo and Nielsen, 2001; Goodall et al., 2014). The electrode-skin impedance was kept <5 KΩ throughout the experimental protocol, and the electrode position was confirmed after the exercise. The surface signal was amplified (gain of 1000) and sampled at 2 kHz, using a 1–30 Hz band pass filter. In order to reduce artifacts, EEG unit's cables were fixed on the individuals' trunk, and cyclists were further oriented to maintain upper limbs as steady as possible during exercise (Thompson et al., 2008). A researcher experienced with EEG records visually inspected the

data for noise, so that data with excessive noise was excluded from the EEG analysis ($n = 2$).

The EMG of the VL and rectus femoris (RF) muscles was monitored throughout the standard warm-up and exercise (Delsys®, Chicago, IL, USA). A pair of surface EMG electrodes (impedance <5kΩ, inter-electrode distance of 2 cm) was placed over the muscle belly according to the probable muscle fiber orientation of these muscles, after cleaning, shaving and exfoliation of the skin. A surgical pen ensured the same electrode placement between the testing sessions. The EMG signal was amplified (gain of 1000) and sampled at 2 kHz.

Cardiopulmonary responses including VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$), ventilation (VE; $L \cdot min^{-1}$) and end-expiratory fraction of CO_2 ($FeCO_2$ expressed as %) were assessed using an open-system gas analyzer (Cosmed®, Quark PT, Albano Laziale, Rome, Italy), which was calibrated before each test with ambient air and gases of known composition (20.9% O_2 and 5% CO_2). The turbine flowmeter was calibrated with a 3 L syringe (Quinton Instruments®, Milwaukee, WI, USA). The cyclists wore a mask (Hans Rudolph®, Lenexa, KS, USA) connected to the gas analyzer to obtain breath-by-breath measurements of gaseous exchange throughout the experimental set-up. In addition, the heart rate (HR) was obtained throughout the experimental set-up (2 Hz frequency) by a cardio belt (Suunto®, Finland).

The cyclists were instructed to rate their perceived exertion (RPE) according to the 6–20 Borg's scale (Borg, 1982), at the end of every stage in the MIT, and at the end of every 0.5 km in the TT_{4km}. They were oriented to consider breathlessness, cardiac work, muscular strain, and body temperature when reporting the RPE (Hampson et al., 2001).

Data Analysis

In order to analyze time and exercise mode effects, experimental data were plotted as a function of paired percentages of the total exercise duration. Thus, after data processing as described below, values of power output, COX, MOX, EEG, EMG, and cardiopulmonary responses were averaged over the last 10 s of every 10% of the total exercise duration. In contrast, irregular time intervals of RPE measures implicated in non-paired percentages between MIT and TT_{4km}, thus the RPE slope was used to compare the exercise modes.

Power Output

Power output data were re-sampled to 1 Hz to remove extreme values, before averaging values at 10% intervals. The time to exhaustion and the time to complete the 4 km were further used as a performance parameter in MIT and TT_{4km}, respectively. In order to characterize the cyclists, the W_{PEAK} achieved in the MIT and TT_{4km}, calculated as the mean of the highest 30 s of the trial, was recorded.

NIRS Data

The raw data were filtered using a 0.4 Hz low pass-band filter (HomER, http://www.nmr.mgh.harvard.edu/PMI/resources/homer/home.htm), and subsequently resampled to 1 Hz. Then, exercise NIRS data were expressed relative to the last 30 s of the baseline (Δμmol), before averaging values at 10% intervals. During this baseline period participants were completely calm, with their eyes closed and without voluntary movements. The O_2Hb, HHb, and total hemoglobin concentrations ($THb = O_2Hb + HHb$) were indexes of COX and MOX (Subudhi et al., 2008; Billaut et al., 2010; Santos-Concejero et al., 2015).

EEG Data

The exercise EEG data were normalized to the signal recorded in the last 30 s of the baseline. In order to analyze the frequency domain data, power spectrum density was estimated by the Welch periodogram of detrended data, resulting in a resolution of 0.2 Hz. The area under the power spectrum curve at 10% exercise intervals was calculated for the alpha wave (α), between 8 and 13 Hz. Briefly, we have used the alpha bandwidth at Cz position to indicate PMC activation, as alpha wave levels in motor cortex areas may reflect an increased number of neurons coherently activated (Pfurtscheller and Lopes da Silva, 1999). The greater inhibited neurons-to-disinhibited neurons relationship in motor cortices suggests higher activation, reflecting the facilitation of sensory stimuli derived from PFC areas during subjective awareness (Craig, 2002; Uusberg et al., 2013). Thus, an increased alpha level during exercise may indicate a cooperative or synchronized behavior of a large number of activated neurons, therefore reflecting activation (von Stein and Sarnthein, 2000; Robertson and Marino, 2015). In addition, this frequency band has turned out to be sensitive to motor activity with less EMG activity contamination (Hilty et al., 2011).

EMG Data

The EMG signal was initially filtered with a hardware band-pass filter set at 20 and 500 Hz, thereafter the exercise EMG data were normalized as a function of the EMG recorded during the standard controlled-pace warm-up (Knutson et al., 1994; Ball and Scurr, 2013). The root mean square (RMS) calculated at every 10% of the exercise duration, provided an index of activation in VL and RF muscles.

Cardiopulmonary Data

Breath-by-breath data were filtered using moving averages, and values ≥3 SD from the local mean (the 5-breath moving average) were substituted by the local mean. Thereafter, a cubic spline interpolation provided data at every 1 s interval (DiMenna et al., 2008), before averaging the last 10 s of every 10% of the exercise duration. Important, based on the study design's rationale, we identified the VO_{2MAX} in MIT. The VO_{2MAX} was determined by the plateau criterion, an increase in VO_2 of ≤150 $ml \cdot min^{-1}$ (Taylor et al., 1955). Alternatively, VO_{2MAX} was determined when RER > 1.10, peak HR > 90% of the age-estimated HR, and RPE > 18, if a VO_2 plateau was not detected (Shephard, 1984). In addition, three evaluators identified the VT_2 visually, thus making possible comparisons with previous studies which reported cerebral responses relative to VT_2 (Rupp and Perrey, 2008; Robertson and Marino, 2015). The second increase in the VE/VO_2 relationship during MIT determined the VT_2 intensity (Meyer et al., 2005).

RPE Data

The slope derived from linear regression of the RPE-time of exercise relationship was calculated to indicate how RPE changed over the different exercise modes.

Statistics

Gaussian distribution was initially checked through Shapiro–Wilk's test. Dependent variables with regard to motor output (power output and VL and RF muscles EMG), cerebral (COX and EEG α wave), and peripheral (MOX and cardiopulmonary variables) responses were compared within and between the different exercise modes through a number of true mixed models, having time and mode of exercise as fixed factors and cyclists as random factor (Ugrinowitsch et al., 2004). When *F*-values were significant, multiple comparisons were performed through Bonferroni's test; rather than fixed factors, we were interested in the time-by-exercise mode interaction. The RPE slope during both the exercise modes was compared through a paired Student's *t*-test. All the significant p values ($p < 0.05$) attained a power $\geq$0.98. Furthermore, effect sizes (ES) for main and interaction effects were calculated (expressed as Cohen's d) and interpreted as small ($d \leq 0.20$), moderate ($0.20 < d < 0.80$) and large ($d \geq 0.80$). Results were reported as mean and standard deviation ($\pm$ SD).

RESULTS

Cyclists' Characterization

Most cyclists ($n = 8$) achieved VO_{2MAX} according to the plateau criteria, the exception was one cyclist who showed a VO_2 plateau between 150 and 200 ml·min^{-1}, so that alternative criteria were used to determine his VO_{2MAX}. They achieved a VO_{2MAX} of 57.5 $\pm$ 6.2 ml·kg^{-1}·min^{-1}, a W_{PEAK} of 368.4 $\pm$ 19.3 W, and a VT_2 of 282.2 $\pm$ 23.6 W (76.6 $\pm$ 6.4% W_{PEAK}) during MIT. Regarding the TT_{4km}, cyclists used a U-shape pacing strategy, thus reaching a W_{PEAK} of 391.9 $\pm$ 78.7 W within the first 10% of trial. The mean power output during TT_{4km} was 316.8 $\pm$ 62.0 W. The MIT was completed within 699 $\pm$ 67 s, and the TT_{4km} within 359 $\pm$ 17 s.

Motor Output

A time ($P < 0.001$) main effect was observed, so that the power output changed over time for both the exercise modes. Additionally, an exercise mode ($P < 0.01$) main effect reveled greater values in TT_{4km} than in MIT. A time-by-exercise mode interaction effect ($P < 0.01$) revealed higher power output values in TT_{4km} than MIT, from 10 to 50% of the exercise duration (**Figure 1**).

Regarding EMG responses, a time main effect ($P < 0.001$) was found in VL and RF muscles, but only VL muscle showed an exercise mode main effect ($P < 0.05$). In addition, there was a time-by-exercise mode interaction effect in VL and RF muscles ($P < 0.05$). As shown by **Figures 2A,B**, overall results were a greater muscle activation in TT_{4km}, mainly during the first half of the trial.

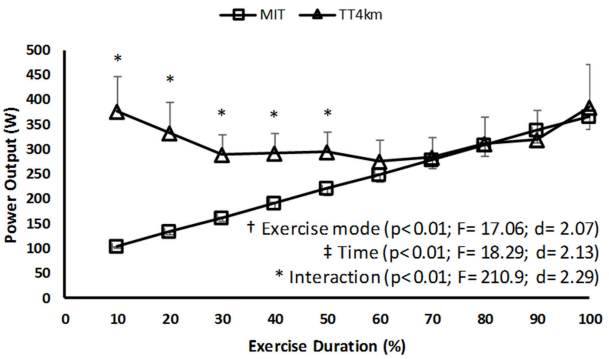

FIGURE 1 | Power output during maximal controlled-pace (MIT) and self-paced ($TT_{4\ km}$) exercise. Symbols indicate exercise mode (†) and time (‡) main effects, as well as time-by-exercise mode interaction effects (*).

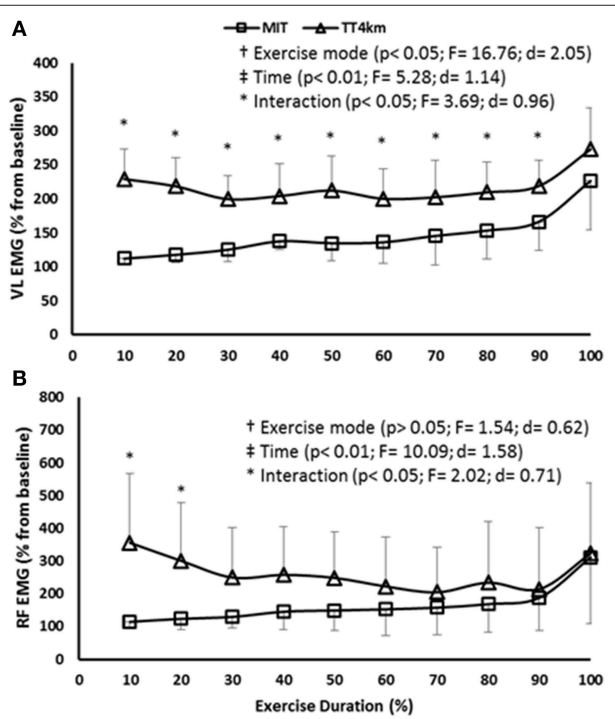

FIGURE 2 | (A,B) EMG of the vastus lateralis (VL) and rectus femoris (RF) muscle during maximal controlled-pace (MIT) and self-paced (TT_{4km}) exercise. Symbols indicate exercise mode (†) and time (‡) main effects, as well as time-by-exercise mode interaction effects (*). Panels **(A,B)** depict VL and RF EMG.

Cerebral Responses

Regarding COX responses, a time and an exercise mode main effect were observed for $\Delta[O_2Hb]$ ($P < 0.001$) and Δ[HHb] ($P < 0.001$), but only a main time effect was found in Δ[THb] ($P < 0.001$). Thus, oxygenation responses at PFC increased up to $\approx$70% of both MIT and TT_{4km}, and decrease afterwards. A time-by-exercise mode interaction effect was observed for $\Delta[O_2Hb]$ ($P < 0.01$), thus when compared to MIT, $\Delta[O_2Hb]$ was lower in

TT_{4km} than MIT at 20, 30, 40, 50, and 60%, but higher at 100% of the exercise duration (**Figures 3A–C**).

The activation in PMC was maintained in both MIT and TT_{4km}, as neither time nor time-by-exercise mode interaction effect was observed in EEG α band at the Cz position. However, an exercise mode main effect ($P < 0.01$) showed a higher activation in TT_{4km} (**Figure 4**).

Peripheral Responses

MOX responses ($\Delta[O_2Hb]$ and Δ[HHb], but not Δ[THb]) decreased up to ≈50% of the MIT and TT_{4km} (time main effect; $P < 0.001$), with lower values for this latter exercise mode (exercise mode main effect; $P < 0.001$). No time-by-exercise mode interaction effect was observed for MOX indices (**Figures 5A–C**).

Cardiopulmonary responses increased throughout the exercise modes (time main effect; $P < 0.001$), and values in the TT_{4km} were systematically greater than in MIT (exercise mode main effect; $P < 0.01$), mainly during the first half of the trials (time-by-exercise mode interaction effect; $P < 0.01$; **Figures 6A–D**).

Perceived Exertion

The different exercise modes induced different effort sensations, as there was a greater RPE slope ($P < 0.01$) in MIT than in TT_{4km} (**Figure 7**).

DISCUSSION

We hypothesized that COX reductions at the closing stages of the exercises would not impair the PMC activation, thus allowing an increasing muscle recruitment and power output (i.e., motor output) at maximal levels in both the exercise modes. The present study showed that different aerobic exercise modes induced similar, maximal motor output during the last 50% of the VO_{2MAX}-matched trials, regardless of COX reductions ($\Delta[O_2Hb]$) from 70% of both exercises. The different motor output performed before that point implicated in distinct COX responses during most of the exercises, however the PMC activation was preserved throughout the exercises. Interestingly, cyclists perceived the self-paced TT_{4km} more tolerable than MIT, even though the greater motor output and intensified physiological responses during the trial.

COX responses in both exercise modes may be partially linked to a systemic network response initiated by an increasing motor output. For example, elevations in motor output (mainly after the VT_2) can lead to elevations in cardiopulmonary responses such as hyperventilation, probably due to the greater III/IV afferent fibers-induced central command to respiratory and vascular systems (Amann et al., 2010, 2011). As a result, hyperventilation-induced hypocapnia would drive cerebral responses toward vasoconstriction and diminished blood flow, thus affecting $\Delta[O_2Hb]$ and Δ[THb] concentrations (Nielsen et al., 1999). In fact, reductions in $\Delta[O_2Hb]$ and Δ[THb] concentrations over

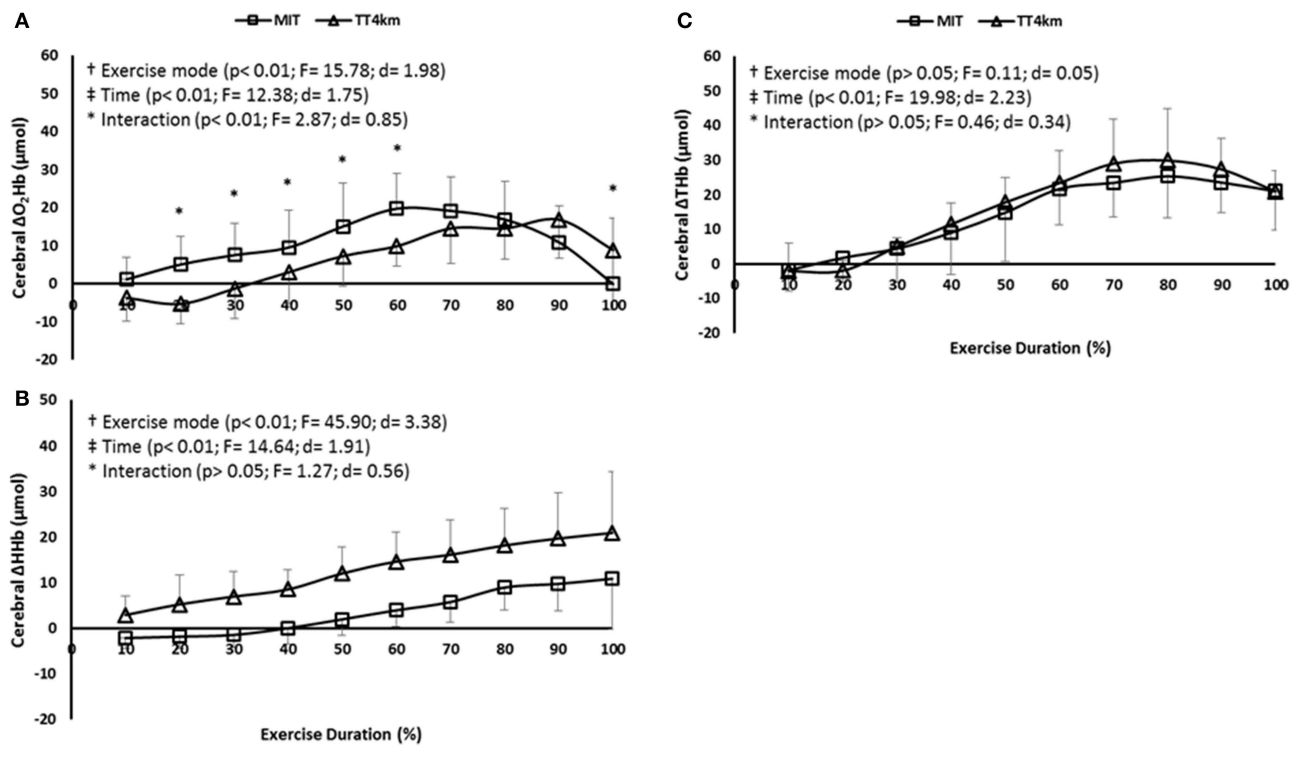

FIGURE 3 | (A–C) Cerebral oxygenation (COX) measured at the prefrontal cortex (PFC) during maximal controlled-pace (MIT) and self-paced (TT_{4km}) exercise. Symbols indicate exercise mode (†) and time (‡) main effects, as well as time-by-exercise mode interaction effects (*). Panels **(A–C)** depict $\Delta[O_2Hb]$, Δ[HHb], and Δ[THb].

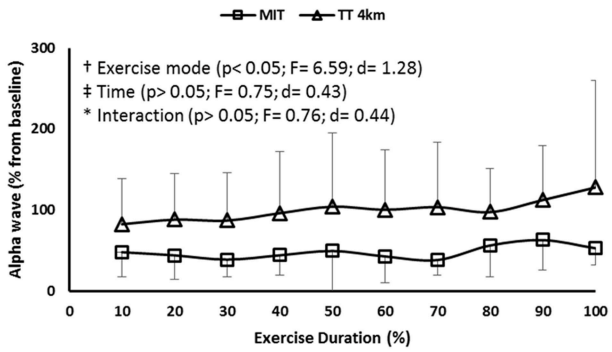

FIGURE 4 | EEG α wave measured at the Cz position during maximal controlled-pace (MIT) and self-paced ($TT_{4\ km}$) exercise. Symbols indicate exercise mode (†) and time (‡) main effects, as well as time-by-exercise mode interaction effects (*).

time (main time effects) matched an elevated motor output, hyperventilation and lowered $FeCO_2$ levels (**Figure 6C**).

The different maximal exercise modes caused different PFC deoxygenation, since TT_{4km} induced lower $\Delta[O_2Hb]$ concentrations between 20 and 60% of the total duration (but greater at the endpoint) when compared to MIT. It has been suggested that COX responses to exercise may reflect O_2 tissue extraction rather than O_2 tissue delivery (Millet et al., 2012), thus the $\Delta[O_2Hb]$ concentrations relative to Δ[THb] concentrations may indicate the local O_2 tissue extraction (González-Alonso et al., 2004). Therefore, when compared to MIT, the lower $\Delta[O_2Hb]$ relative to Δ[THb] concentrations during most of the TT_{4km} may have indicated that the maximal self-paced TT_{4km} required greater O_2 tissue extraction than MIT.

This different O_2 tissue extraction between the exercise modes may be part of a centrally-coordinated exercise regulation, suggesting a distinct participation of the PFC between MIT and TT_{4km}. The PFC projects to motor cortex areas, and may regulate motor output when integrating sensory afferent information to guide the decision-making during exercise (Ridderinkhof et al., 2004; Robertson and Marino, 2015). Thus, it is possible that the self-paced TT_{4km} has required a higher PFC activation to integrate the sensory afferents from skeletal muscles and cardiopulmonary system, thus resulting in a greater O_2 tissue extraction ($\downarrow$ $\Delta[O_2Hb]$) in this area. In fact, the greater muscle deoxygenation (MOX $\downarrow$) and cardiopulmonary response (VO_2, VE, $FeCO_2$, and HR) during most of the self-paced TT_{4km} could suggest that there was a higher III/IV muscle afferents toward interoceptive PFC areas in this exercise mode (Craig, 2002; Amann et al., 2011; Meeusen et al., 2016).

Importantly, PMC activation was maintained throughout the exercise modes, regardless of different PFC deoxygenation patterns. These results are in agreement with recent findings showing that PMC activation can be preserved throughout a MIT, even at intensities above the VT_2 (Robertson and Marino, 2015), an exercise intensity that elicits pronounced changes in COX, EMG, and cardiopulmonary responses (Rupp and Perrey, 2008). Our results provide support to the hypothesis that motor cortex activation may be preferentially preserved during strenuous exercises, in contrast to PFC areas (Subudhi et al., 2009; Robertson and Marino, 2015). In addition, results of EEG α wave further indicated greater PMC activation in TT_{4km} than in MIT, agreeing with the elevated motor output (i.e., EMG and power output) during most of this exercise mode. This could suggest a greater coherence of neural populations activation in PMC regions during TT_{4km}, perhaps reflecting a higher facilitation of sensory stimuli derived from the PFC in this self-paced exercise (Uusberg et al., 2013).

Interestingly, although the higher motor output and exaggerated PFC deoxygenation during the first half of the TT_{4km}, cyclists perceived this self-paced exercise as more tolerable than MIT, as RPE increased slower until the attainment of maximal values. Such a response may be linked to the nature of the exercise mode under consideration (Robertson and Marino, 2016). Different of controlled-pace exercises, during which individuals are forced to match a power output predetermined by the experimenter, self-paced exercises allow individuals to pace themselves according to feedback and feedforward control mechanisms (Marino et al., 2011; Noakes, 2012). In this exercise mode the CNS is allowed to regulate the recruitment/de-recruitment of motor units, so that the rate of increase in physiological disturbance would be reduced due to an alleviated muscle recruitment (Marino et al., 2011; Noakes, 2012). Actually, when comparing a power-matched, submaximal self-paced exercise with a forced-pace exercise, previous study observed a lower physiological disturbance which was associated with a less challenging perception in this former exercise mode (Lander et al., 2009).

The fact that the physiological disturbance was exacerbated in the self-paced exercise, as suggested by the lower MOX and higher cardiopulmonary responses to the TT_{4km}, does not necessarily contradicts those previous results in submaximal exercises (Lander et al., 2009), since the exercises were performed at maximal motor output levels in the present study. Perhaps the continuous muscle recruitment/de-recruitment regulation via feedback and feedforward commands during the self-paced TT_{4km} has allowed a more appropriate sensory cues integration into interoceptive PFC areas (Robertson and Marino, 2016), thus taxing the PFC with a higher metabolic cost (lower $\Delta[O_2Hb]$ relative to Δ[THb] concentrations). However, as indicated by RPE responses, the self-selection of the exercise intensity made this exercise mode more tolerable than the controlled-pace MIT, possibly as a result of a greater affective response during this exercise mode. In fact, a recent meta-analysis concluded that exercises with self-selected intensity promote greater affective response than imposed ones (Oliveira et al., 2015). Then, considering the fact that affect and RPE responses to exercise may share the same conscious mental processing (Ramalho Oliveira et al., 2015), perhaps the greater tolerance in maximal self-paced exercise could be a result of the greater affective response when self-selecting the pace of exercise.

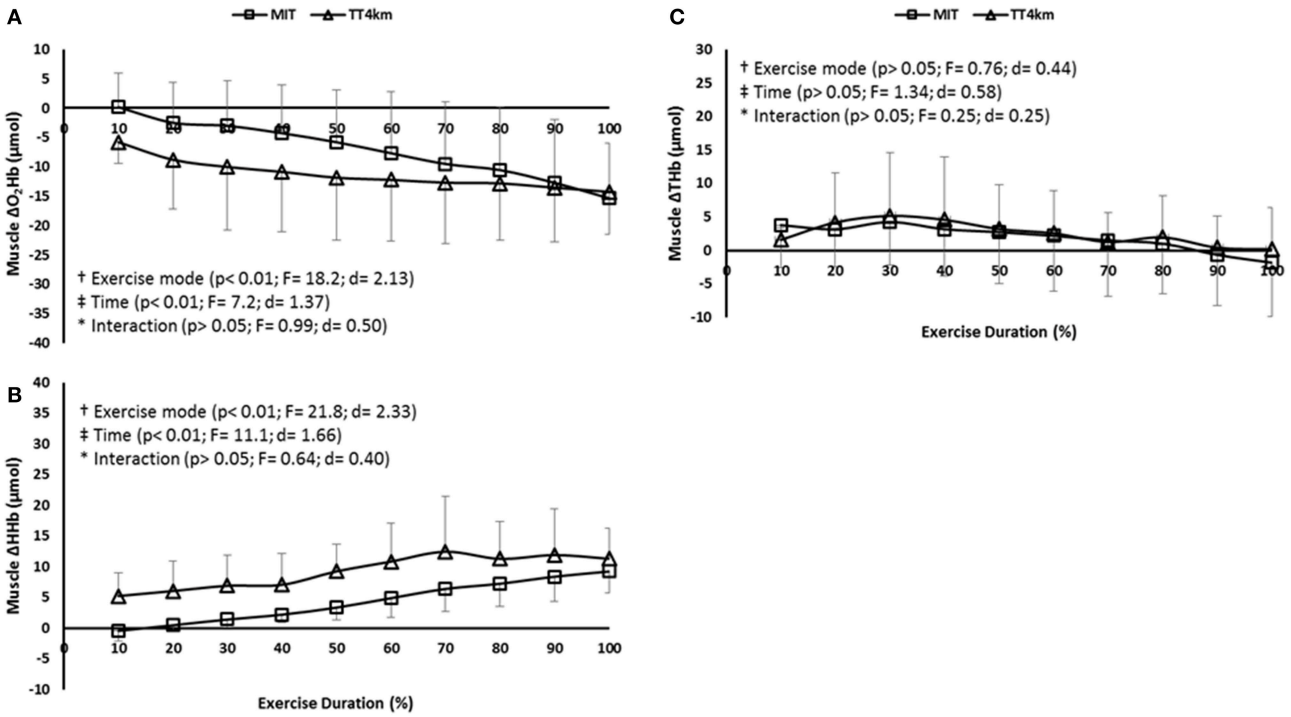

FIGURE 5 | (A–C) Muscular oxygenation (MOX) measured in vastus lateralis muscle (VL) during maximal controlled-pace (MIT) and self-paced (TT_{4km}) exercise. Symbols indicate exercise mode (†) and time (‡) main effects, as well as time-by-exercise mode interaction effects (*). Panels **(A–C)** depict Δ[O_2Hb], Δ[HHb], and Δ[THb].

Aspects of the Study's Design and Limitations

We have assumed the VO_{2MAX} as the upper limit for maximal aerobic exercise performance (Bassett and Howley, 2000; Levine, 2008), so that we used VO_{2MAX}-matched exercises while investigating cerebral responses to maximal controlled-pace and self-paced exercise. This approach ascribed rationale to the study, as we measured PFC oxygenation and PMC activation (together with peripheral variables and RPE) when individuals were required to produce maximal motor output in VO_{2MAX}-matched MIT and TT_{4km}. Then, we were able to observe how the cerebral regulation responded to these different maximal aerobic exercise modes for the entire period of exercise.

Nevertheless, although these exercises have produced similar motor output (i.e., power output and EMG) during the last 50% of the trials, they were considerably different in duration (12 vs. 6 min). We are unaware if the different exercise durations could have affected our results in some way. For example, a recent study by (Thomas et al., 2015) found a reduced rest corticospinal excitability after 20 and 40 km self-paced cycling time trial, but not after TT_{4km}. Accordingly, it was found different reductions in maximal voluntary activation after 4 km (↓ 6%), 20 km (↓ 12%), and 40 km (↓ 10%) cycling time trials. Thus, these results may have indicated that supraspinal and spinal sites (putative neuronal pathways along the corticospinal tract) responded differently between cycling trials of different durations, even though the different mean power output (i.e., motor output) between them may have further influenced the results. It is important to note that the direct comparison between studies should be made carefully, as we measured PMC activation through EEG technique during dynamic cycling, whereas that study used a maximal voluntary contraction superimposed by TMS, after the exercise cessation (Thomas et al., 2015). In this sense, rather than conflicting one with each other, these studies may indicate that cerebral regulation is complex, and differs between exercises different in nature (maximal dynamic cycling vs. maximal sustained contraction), as suggested elsewhere (Liu et al., 2005). Perhaps the combination of NIRS and EEG, together with maximal voluntary contraction superimposed by TMS, in different aerobic exercise modes of equivalent duration, such as conventional and perceptually-controlled MIT (Mauger and Sculthorpe, 2012), may respond how the different durations affected our results.

The use of EEG technique to monitor changes in cortical activation during whole body strenuous aerobic exercises has been criticized due to the inherent noise derived from upper body movement and low spatial resolution (Thompson et al., 2008; Enders and Nigg, 2016). Regarding the first aspect, we took some precaution in order to record good-quality EEG signal during exercise by using active electrodes, fixing cables and electrode, and asking individuals to maintain upper limbs as steady as possible during exercise (Thompson et al., 2008;

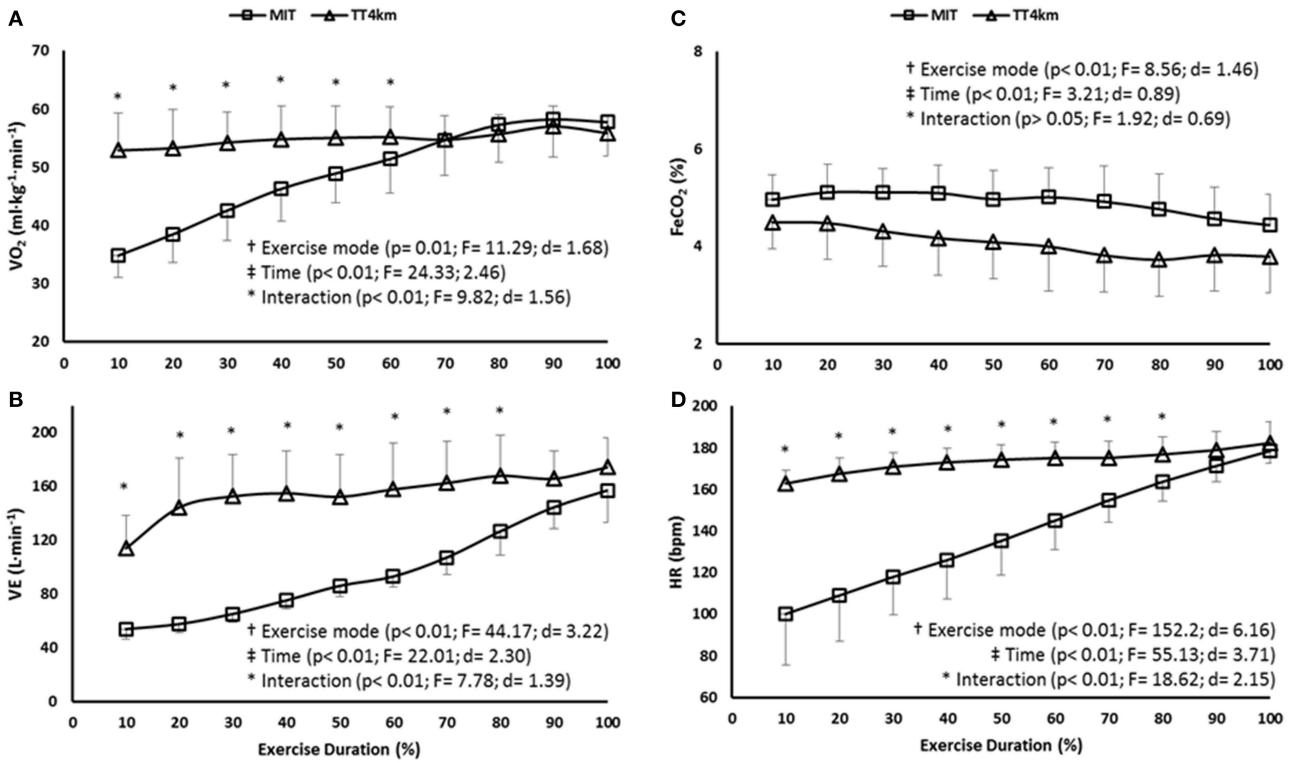

FIGURE 6 | (A–D) Cardiopulmonary responses during maximal controlled-pace (MIT) and self-paced (TT_{4km}) exercise. Symbols indicate exercise mode (†) and time (‡) main effects, and time-by-exercise mode interaction effects (*). Panels **(A–D)** depict VO_2, VE, $FeCO_2$, and HR.

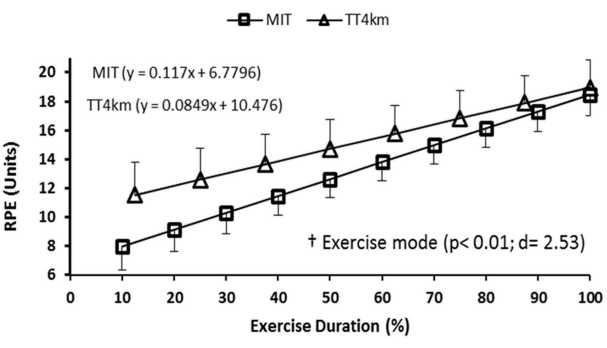

FIGURE 7 | RPE responses during maximal controlled-pace (MIT) and self-paced ($TT_{4\ km}$) exercise. Symbol indicates exercise mode main effect (†) for RPE slope.

Hilty et al., 2011). Furthermore, muscle artifacts seem to be more prevalent for frontal and posterior cortical sites than for Cz position, whereas eye movements may not increase during exercise to highly affect EEG signal, mainly the α bandwidth (Thompson et al., 2008; Hilty et al., 2011). However, we acknowledge that EEG artifacts associated with whole body strenuous exercises are a challenge to data analysis, being the reason why we actually excluded two individuals from the EEG analysis.

Regarding the low spatial resolution, it is not possible to ensure that we measured the exact cortical area responsible for the motor command of primary muscles involved in cycling (e.g., VL and RF muscles). We used the EEG international 10–20 system because we were interested in an overview of the cerebral responses during maximal cycling aerobic exercises; that is PFC oxygenation and PMC activation. Therefore, according to the Penfield's human motor Homunculus we have selected a cortical area that should satisfactorily reflect the motor command for lower limbs (hip, knee, ankle, and toes; Goodall et al., 2014), however we cannot ensure that we measured the exact area responsible for VL and RF muscles recruitment.

Although the limitations highlighted above may require some caution when interpreting cerebral responses to strenuous aerobic exercises, results of the present study may further suggest some practical implications. For example, it would be worth to know if long-term endurance training may modulate the individual's ability to tolerate PFC deoxygenation, while maintaining PMC activation. Perhaps, improvements in motor output performance after training period could be also related to a modulation of activation in these cerebral areas.

CONCLUSION

The present study showed that similar, maximal motor output was attained at the closing stages of different VO_{2MAX}-matched

aerobic exercises, regardless of different cerebral responses during most of the exercises duration. The decrease in PFC oxygenation during the last percentages of the trials impaired neither motor output (EMG and power output) nor PMC activation, suggesting that the different COX responses during exercise may be part of a centrally-coordinated regulation.

AUTHOR CONTRIBUTIONS

All of the listed authors contributed to this study, conceiving and designing the experiments (FP, TN, and CU), collecting and analyzing the data (FP, CD, RC, FP, FM, and CU), writing the manuscript (FP, FP, FM, and CU), criticizing and reviewing the manuscript (CD, RC, TN, and AG). All the listed authors approved the final version of the manuscript.

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MINI REVIEW
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The Ergogenic Effects of Transcranial Direct Current Stimulation on Exercise Performance

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The physical limits of the human performance have been the object of study for a considerable time. Most of the research has focused on the locomotor muscles, lungs, and heart. As a consequence, much of the contemporary literature has ignored the importance of the brain in the regulation of exercise performance. With the introduction and development of new non-invasive devices, the knowledge regarding the behavior of the central nervous system during exercise has advanced. A first step has been provided from studies involving neuroimaging techniques where the role of specific brain areas have been identified during isolated muscle or whole-body exercise. Furthermore, a new interesting approach has been provided by studies involving non-invasive techniques to manipulate specific brain areas. These techniques most commonly involve the use of an electrical or magnetic field crossing the brain. In this regard, there has been emerging literature demonstrating the possibility to influence exercise outcomes in healthy people following stimulation of specific brain areas. Specifically, transcranial direct current stimulation (tDCS) has been recently used prior to exercise in order to improve exercise performance under a wide range of exercise types. In this review article, we discuss the evidence provided from experimental studies involving tDCS. The aim of this review is to provide a critical analysis of the experimental studies investigating the application of tDCS prior to exercise and how it influences brain function and performance. Finally, we provide a critical opinion of the usage of tDCS for exercise enhancement. This will consequently progress the current knowledge base regarding the effect of tDCS on exercise and provides both a methodological and theoretical foundation on which future research can be based.

Keywords: tDCS, brain stimulation, exercise performance, perception of effort, cortical excitability, motor cortex

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INTRODUCTION

During sustained submaximal contraction, the excitability of spinal motoneurons and the contractile capacity of the muscle fibers are reduced (Butler et al., 2003; Allen et al., 2008), so that in order to maintain the required force or power, the input to the spinal motoneurons must increase (Taylor et al., 1996). This input (also called descending drive) is likely to originate from the corticospinal pathway, and previous experiments have demonstrated a number of factors which may moderate this (Gandevia, 2001; Enoka et al., 2011). In this regard, a failure to generate output from the motor cortex (M1) has been defined as supraspinal fatigue, and together with peripheral mechanisms, participates in muscle fatigue (Gandevia, 2001). Previous studies have suggested that

the development supraspinal fatigue is accompanied by changes in motor cortex excitability (Taylor et al., 1996).

Interventions that increase M1 excitability might increase the output from M1 (increase descending drive) thus delaying the development of supraspinal fatigue and therefore improving exercise capacity (Cogiamanian et al., 2007; Williams et al., 2013). In this regard, a neuromodulatory technique called transcranial direct current stimulation (tDCS) has been widely used to modulate the excitability of a targeted brain area through the application of a weak electrical current across the scalp. The electrical current alters the resting membrane potential of the targeted neurons, with the anodal electrode being excitatory and the cathodal being inhibitory (Nitsche et al., 2008; George and Aston-Jones, 2010). These effects can persist for up to 90 min following 9–13 min of stimulation (Nitsche and Paulus, 2001). Studies have demonstrated that acute tDCS is a safe neuromodulatory brain technique, with no or only minor side effects (Fregni et al., 2006; Poreisz et al., 2007; Palm et al., 2008; Frank et al., 2010) and is both cheap and easy to administer. Therefore, interest in tDCS' ergogenic potential has grown considerably.

Research has only recently started to investigate the effect of tDCS on physical performance and, given the prominent role of the motor and premotor brain regions in the development of supraspinal fatigue (Gandevia, 2001), most studies have attempted to target these areas. To date, there are a limited number of studies, showing inconsistent results and often with flawed methodological design. Nevertheless, the balance of evidence suggests that tDCS might have a positive effect on exercise capacity. A summary of the most significant studies on tDCS stimulation and exercise performance are shown in **Table 1**. For the purpose of this review we considered studies that adhered to the following criteria:

- Acute administration of tDCS prior to, or during, exercise in healthy participants;
- Continuous exercise lasting at least 75 s (Gastin, 2001);
- Exercise tasks involving time to exhaustion, time trial, or incremental exercise testing.

Selected studies were divided into either single joint isometric or whole body exercise. While whole-body exercise better represents real sporting competition, single-joint exercises potentially permit a better, and more controlled exploration of the physiological mechanisms associated with fatigue. This distinction is fundamental as the two exercise modalities differ in terms of metabolic, cardiorespiratory, and psychological demand, and therefore differently affect brain activity (Sidhu et al., 2013). Studies were then ordered according to publication date.

The aim of this mini-review is to provide a framework to discuss and analyse the studies involving acute administration of tDCS with the aim of improving exercise performance. A brief analysis of the physiological and psychological mechanisms and methodological limitations has been provided in order to improve the understanding of the effect of tDCS on exercise performance.

STUDIES ON SINGLE JOINT ISOMETRIC EXERCISE

The first study investigating the effect of tDCS on exercise performance was performed by Cogiamanian et al. (2007), and was comprised of two experiments. In the first, participants were divided in two groups (brain polarization and control) with both completing two elbow flexor isometric time to exhaustion (TTE) tasks. Prior to the second task, the brain polarized group received anodal or cathodal tDCS while the control group did not receive any tDCS administration. The second experiment aimed to monitor the corticospinal response following tDCS administration. No changes in MVC or EMG activity were found, but the second TTE was significantly longer following anodal tDCS, with a significant increase in corticospinal excitability observed in the second experiment. The authors were not able to provide a precise explanation for the improvement in TTE, but suggested that tDCS could act upstream of the M1 by facilitating the supraspinal drive or by protecting the M1 from inhibitory feedback arising from working muscles.

Two different studies partially replicated the study of Cogiamanian et al. (2007). Kan et al. (2013) performed a crossover study where participants performed a protocol similar to that used by Cogiamanian et al. (2007), but with a lower contraction intensity (30% MVC) and different tDCS montage (see **Table 1**). No changes in MVC, torque fluctuation, EMG, and perceived pain were found, with no improvement in TTE duration. The study of Muthalib et al. (2013) mainly aimed to monitor level of prefrontal oxygenation, and similarly to Kan et al. (2013), there was no improvement in MVC or TTE duration, along with no changes in prefrontal oxygenation following tDCS. However, Muthalib et al. (2013) monitored oxygenation in an area distant to the tDCS electrode location (M1), which might explain the lack of change in prefrontal oxygenation. Unfortunately, none of the above studies monitored the corticospinal response and therefore it is not possible to establish whether tDCS was able to increase corticospinal excitability.

A further experiment investigating the effect of tDCS on sustained isometric contraction was performed by Williams et al. (2013). In a crossover study, participants were asked to perform an isometric TTE at 20% MVC of the elbow flexors. Initially, no improvement in performance after anodal tDCS (compared to sham) was observed. Subsequently, the investigators divided participants in two sub groups: one group where TTE time was shorter than tDCS administration time ($n = 8$), and one group where TTE time was longer than tDCS administration time ($n = 10$). The first group showed a significant improvement in performance compared to the second. No significant changes in motor-evoked potentials (MEP) were found between conditions or group, but ratings of perceived exertion (RPE) were significantly reduced in the anodal tDCS condition. The subdivision of the participants according to task duration raises some doubts regarding the true efficacy of tDCS, and the experimental findings question whether tDCS is beneficial only when stimulation occurs

TABLE 1 | List of tDCS studies on exercise performance.

Articles	Sample size	Placement of electrodes	Stimulation duration	Stimulation intensity (mA)	Electrode size (cm^2)	Control condition	Muscle group investigated	Exercise protocol	Performance result
Cogiamanian et al., 2007	Study 1, $n = 9$; study 2, $n = 15$	Anodal right M1, cathodal right shoulder	10 min	1.5	35	Cathodal and control	Left elbow flexors	Isometric TTF at 35% MVC	Improvement
Muthalib et al., 2013	$n = 15$	Anodal right M1, cathodal right shoulder	10 min	2	24	Sham	Left elbow flexors at 90° flexion	Isometric TTF at 30% MVC	No improvement
Kan et al., 2013	$n = 15$	Anodal right M1, cathodal contralateral shoulder	10 min	2	24	Sham	Elbow flexors at 90° flexion	Isometric TTF at 30% MVC	No improvement
Williams et al., 2013	$n = 18$	Anodal right M1, cathodal left forehead	20 min during TTF	1.5	35	Sham	Left elbow flexors	Isometric TTF at 20% MVC	Improvement
Okano et al., 2015	$n = 10$	Anodal T3, cathodal over Fp2	20 min	2	35	Sham	Lower limbs	Cycling, from 15W +25 $Wmin^{--1}$	Improvement of ∼4%
Angius et al., 2015a	$n = 9$	Anodal right M1, cathodal Fp2	10 min	2	35	Sham and control	Lower limbs	Cycling, at 70% of peak power	No improvement
Vitor-Costa et al., 2015	$n = 11$	Active over Cz and reference over occipital protuberance	13 min	2.0	35	Sham and cathodal	Lower limbs	Cycling, at 80% peak power	Improvement
Abdelmoula et al., 2016	$n = 11$	Anodal left M1, cathodal right shoulder	10 min	1.5	35	Sham	Elbow flexors	Isometric TTF at 35% MVC	Improvement
Oki et al., 2016	$n = 13$	Anode over right M1, cathode over the left forehead	Max 20 min during TTF	1.5	35	Sham	Elbow flexors	Isometric TTF at 20% MVC	Improvement
Angius et al., 2016a	$n = 12$	Bilateral montage, active electrode over M1 and reference over the ipsilateral shoulder	10 min	2.0	35	Sham and cathodal	Lower limbs	Cycling, at 70% of peak power	Improvement
Barwood et al., 2016	Study 1, $n = 6$; study 2, $n = 8$	Anodal over T3, cathodal over the contralateral Fp2	20 min	Study 1 = 1.5 Study 2 = 2.0	35	Sham	Lower limbs	Study 1: cycling TT 20 km cycling; Study 2: cycling 25 min at 55% of peak power + TTF at 75% of peak power	No improvement
Angius et al., 2016b	$n = 9$	Extracephalic: anodal left M1 and cathodal over ipsilateral shoulder; Cephalic: anodal left M1; and cathodal over dorsolateral right prefrontal cortex	10 min	2.0	35	Sham and control	Right knee extensors	Isometric TTF at 20% MVC	Improvement with extracephalic montage

M1, Primary motor cortex; MVC, maximal voluntary contraction; TT, time trial; TTF, time to task failure.

during exercise and only to those with lower endurance capacity.

With the aim to provide a better understanding of tDCS mechanisms, Abdelmoula et al. (2016), monitored several muscles in a similar protocol to Cogiamanian et al. (2007). Similar to the findings of Cogiamanian et al. (2007), TTE duration was longer following anodal tDCS. However, this occurred in the absence of any change in neuromuscular, corticospinal or perceptual parameters. In fact, MVC, coefficient of variation of torque, EMG activity during exercise, MEP responses, and RPE did not differ between conditions. Because of the increase in TTE duration in the absence of changes in neuromuscular or corticospinal response, the authors proposed that the large tDCS electrode might have facilitated adjacent brain areas which affected the sensorimotor integration and the associated cognitive demand during the task without producing any change in the central motor command. This study however did not provide any evidence to support this suggestion.

The benefits of tDCS have been extended to older populations (Oki et al., 2016), with older adults being shown to have lower cortical excitability following tDCS than younger adults (Oliviero et al., 2006). Together with an increase in TTE duration after anodal tDCS, a slower increase in RPE was observed in agreement with previous experiments (Williams et al., 2013; Okano et al., 2015; Angius et al., 2016b). The authors (Oki et al., 2016) suggested that the increased excitability of the M1 could have reduced the neural drive necessary to perform the task, which therefore lowered RPE. An association between the magnitude of the effect of tDCS and baseline level of muscle strength was found ($r = -0.55$; $p = 0.05$). This may suggest that weaker subjects could receive more benefits compared to stronger subjects, although the authors did not further investigate this potential. Only 45% of the subjects demonstrated a positive response to tDCS, and so these findings might also in part explain the different outcomes across tDCS studies, as the efficacy of tDCS might rely on high responder participants. Future studies should therefore take into account such variables when determining the participant cohort.

Angius et al. (2016b) compared the effect of two tDCS montages (see **Table 1**) on TTE of knee extensors. TTE was significantly longer when an extracephalic montage was used without any effect on corticospinal and peripheral parameters. A reduction in RPE was found when the extracephalic montage was used, while HR and pain were unchanged. As no effect on corticospinal and peripheral parameters was found, the exact mechanisms explaining the improvement in TTE are still uncertain. However, the absence of effect on the corticospinal response could be due to the contraction intensity used (50% MVC) for the neuromuscular assessment. Indeed, the largest MEP response has been shown to occur at 50% MVC (Goodall et al., 2014), which could have masked the tDCS effect on this variable. This study suggests that an extracephalic montage is more appropriate for the improvement in exercise capacity, and could explain the null effect of tDCS shown in previous studies involving whole body exercise (Angius et al., 2015b; Barwood et al., 2016).

STUDIES ON WHOLE BODY DYNAMIC EXERCISE

The first study investigating the effect of tDCS on whole body exercise was conducted by Okano et al. (2015). In a crossover, randomized experimental design, participants performed maximal cycling exercise up to volitional exhaustion. Following anodal tDCS, maximal power output improved by ∼4%, and RPE and HR were lower compared to a sham condition (although they were not affected in the latter stages of the test). The authors suggested that anodal stimulation could have affected the activity of the insular cortex, thus reducing RPE and leading to an improvement in performance.

Angius et al. (2015a) investigated the effect of tDCS on exercise-induced muscle pain during cycling TTE and on pain perception during a cold pressor test. The authors did not find changes in TTE duration and physiological or perceptual parameters during exercise. However, following tDCS a significant reduction in perceived pain during the cold pressor test was found. The lack of effect during cycling was likely caused by the different type of pain stimulus, pain intensity perceived, or the attentional focus during each task. Furthermore, the authors suggested that the lack of effect on exercise performance could have been due to the tDCS montage used (**Table 1**), as any benefits from the anodal electrode on the M1 could have been negated by the cathodal electrode over the dorsolateral prefrontal cortex. The authors therefore suggested that a bilateral extracephalic tDCS montage would be more appropriate for whole body exercise.

An improvement in cycling TTE following tDCS was demonstrated by Vitor-Costa et al. (2015). Despite the effect on TTE, no changes in mood, physiological, or perceptual parameters were reported. It should be noted that a trend for a lower RPE following anodal tDCS was found ($p = 0.07$), suggesting that the increased M1 excitability could have made exercise feel easier for a given intensity (Williams et al., 2013; Abdelmoula et al., 2016; Angius et al., 2016a). The authors suggested that the improvement in TTE was the consequence of an increase in intracortical facilitation and M1 excitability, although this hypothesis could not be confirmed as the necessary corticospinal parameters were not monitored. In addition, the tDCS montage in this study placed one electrode over the occipital protuberance, and as a consequence the direction of current between the two electrodes could have interfered with other brain areas, thus affecting both physiological and perceptual parameters.

Angius et al. (2016a) showed an ergogenic effect of tDCS in whole-body exercise, with TTE duration increasing following anodal tDCS, paralleled a lower RPE. There were no differences observed in the cathodal and sham tDCS conditions. Following anodal tDCS, an increase in corticospinal excitability of the knee extensor muscles was also reported, leading the authors to suggest that the increased excitability of the M1 could have augmented the output to the working muscles by consequently reducing the central command required. This could have caused the lower RPE, leading participants to perceive the exercise as

easier. However, no further evidence to support this hypothesis was provided, and so speculation on such a mechanism should be treated with caution.

In two separate studies, Barwood et al. (2016) investigated the effects of tDCS on a 20 km cycling time trial and a TTE test in hot conditions. The same montage used by Okano et al. (2015) was applied with the hypothesis that tDCS would reduce the RPE for a given intensity and therefore improve cycling performance. No changes in performance in either exercise protocols were found, with no differences in RPE. Unlike Okano et al. (2015) no reduction in HR following tDCS was reported. As proposed by the authors, the discrepancy in exercise outcome compared to Okano et al. (2015) might have been caused by a non-appropriate blinding procedure, and the lack of effect in HR may have been due to the high work rate adopted. The null effects may also have been due to the negative effect of the cathodal electrode. Furthermore, hyperthermia has been well-demonstrated to induce changes in metabolic and cardiovascular demand together with an increase in central fatigue (Nybo and Nielsen, 2001), which may negate any benefits of anodal stimulation.

POSSIBLE MECHANISMS OF ACTIONS AND LIMITATIONS

Collectively, experiments to date provide interesting insights regarding the possible ergogenic effects of tDCS on exercise in healthy individuals. Despite the differences across each study regarding the experimental design, task performed, and tDCS montage, there are some experimental findings which are similar across the various experiments. Firstly, acute tDCS over the M1 does not seem to improve maximal isometric force capacity (Cogiamanian et al., 2007; Kan et al., 2013; Williams et al., 2013; Angius et al., 2015b, 2016a,b). Secondly, tasks performed at a submaximal intensity are generally improved by tDCS (Cogiamanian et al., 2007; Williams et al., 2013; Angius et al., 2015b, 2016a,b; Abdelmoula et al., 2016). Thirdly, none of the physiological or neuromuscular parameters (aside from corticospinal excitability) during exercise seem to be affected by tDCS.

Regarding the inconsistency across each study, previous research has demonstrated a range of responses following tDCS stimulation from little or no effect, to a large effect with high variability in corticospinal excitability (Horvath et al., 2015, 2016; Madhavan et al., 2016). Moreover, there is an absence of a standardized and reliable protocol to monitor the effect of tDCS on the neuromuscular response (Madhavan et al., 2016). Therefore, it is not surprising that improvements in performance were accompanied with no changes in neuromuscular function with particular interest on the corticospinal pathway. Finally, the absence of rigorous blinding procedures in a considerable number of studies (see **Table 1**) might contribute to the mixed results currently seen in the literature, and so where this is apparent the results must be interpreted with caution.

The exact mechanisms by which tDCS improves exercise performance are still unknown. It is suggested that tDCS likely facilitates the M1 by increasing its output during exercise and *possibly* reducing supraspinal fatigue (Cogiamanian et al., 2007; Williams et al., 2013). However, this hypothesis is in contrast with previous studies as the improvement in performance appears not to rely on changes in corticospinal response (Abdelmoula et al., 2016). Other authors suggest that the lower RPE following tDCS administration might explain the improvement in performance (Okano et al., 2015; Angius et al., 2016a,b). Changes in RPE have been related to the magnitude of central motor command originating from activity of motor/premotor brain areas (de Morree et al., 2012, 2014). Thus, if M1 excitability is increased following tDCS administration, it needs to receive less input to generate the amount of output required to recruit the muscle, hence, a lower RPE for a given force or power should be expected. This hypothesis is supported by previous experiments involving non-invasive brain stimulation where manipulation of premotor and motor brain areas induced variations in RPE (Goodall et al., 2013; Takarada et al., 2014; Zénon et al., 2015). However, because of the electrode size, the effects of the tDCS could possibly influence adjacent areas by influencing the sensorimotor integration during muscular contraction without affecting the motor command (Abdelmoula et al., 2016). To the best of our knowledge no studies have monitored the activity of brain areas during exercise following tDCS stimulation and therefore development of a mechanistic understanding is a clear priority.

CONCLUSION AND PERSPECTIVES

The promising outcomes of tDCS on exercise performance have recently attracted attention for its potential to be used domestically for ergogenic purposes. Unlike TMS equipment, tDCS devices are relatively small and easy to use and therefore its use by people unaware of its potential effects has been reported (Reardon, 2016). Given the uncertain mechanisms and the inconsistency of outcomes of tDCS prior to exercise, the use of tDCS prior to/during exercise should be treated with some caution. Future research should seek to identify the mechanisms underpinning the apparent ergogenic effect of tDCS, and focus should also be given the effects of long-term use. As tDCS is clearly of interest not only to the scientific, but also the public and commercial communities, researchers and publishers have a responsibility to disseminate transparent and objective studies that can further our understanding of tDCS.

Currently, the different outcomes observed in tDCS research are likely a consequence of differences between exercise type and/or tDCS set up (**Table 1**), and many of the aforementioned studies were not designed to specifically assess the mechanism by which performance was hypothesized to improve. Therefore, more studies which systematically control the tDCS variables (e.g., montage, duration, location etc.) and allow assessment of the mechanisms are required.

AUTHOR CONTRIBUTIONS

LA was involved with the conception of the content, the writing of the manuscript, the drafting process, the revisions of the

manuscript, and provided approval of the final version. He is accountable for all aspects of the work. JH was involved with the conception of the content, the drafting process, the revisions of manuscript, and provided approval of the final version. He is of the final version. He is accountable for all aspects of the work. AM was involved with the conception of the content, the writing of the manuscript, the drafting process, the revisions of the manuscript, and provided approval of the final version. He is accountable for all aspects of the work.

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High Intensity Interval Training in Handcycling: The Effects of a 7 Week Training Intervention in Able-bodied Men

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Introduction: In lower body endurance training, quantities of both moderate intensity continuous training (MICT) and high intensity interval training (HIIT) can lead to an improved physiological capacity and performance. Limited research is available regarding the endurance and muscular capacity of the upper body, and how training contributes to improvements in performance capacity is still unknown. The aim of the current study was to evaluate the effects of HIIT and MICT on the physiological capacity and handcycling performance of able-bodied men in a well-controlled laboratory setting.

Methods: Twenty four recreationally active men (22 ± 2 years; 1.84 ± 0.04 m; 79 ± 10 kg) were matched on incremental handcycling pre-test performance (peakPO) and then randomly assigned to HIIT, MICT, or a non-training control group (CON, 3 × n = 8). Participants in HIIT completed 14 interval training sessions, performing 4 × 4 min intervals at 85% heart rate reserve (%HRR), and seven continuous training sessions at 55 %HRR (every 2nd training session of the week). Participants in MICT performed 21 training sessions of 30 min at 55 %HRR. After the intervention, changes in peak oxygen uptake ($peakVO_2$) and peak power output (peakPO) were compared within and between HIIT, MICT and CON.

Results: The average external training load per training session did not differ between MICT and HIIT (p = 0.713). Improvements after HIIT in $peakVO_2$ (22.2 ± 8.1%) and peakPO (47.1 ± 20.7%) were significantly larger compared with MICT and CON (p < 0.001). Improvements after MICT in $peakVO_2$ (10.7 ± 12.9%) and peakPO (32.2 ± 8.1%) were higher compared to CON (p < 0.001). Higher improvement after HIIT occurred despite training 22% less time than MICT. No significant changes were found in CON.

Discussion: As in lower body endurance sports, HIIT proved to be very effective in improving the physiological and performance capacity of upper body exercise. Whilst physiological capacity in both training groups improved significantly compared with CON, the present study shows that $peakVO_2$ and peakPO improved more after HIIT than after MICT in able-bodied men. It is advised to include HIIT into training regimes of recreational and competitive handcyclists to improve the upper body endurance capacity.

Keywords: upper body exercise, endurance training, handcycling, physiological capacity

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INTRODUCTION

Endurance performance is regulated by, amongst other factors, the cardiovascular system, the pulmonary system, the lactate metabolism and the exercise economy of an athlete (Joyner and Coyle, 2008). The essence of training is to provide training loads that are effective in improving the performance capacity of athletes. Adaptations to endurance training are well documented for lower body exercise like running and cycling, in which doses of both moderate intensity continuous training (MICT) and high intensity interval training (HIIT) result in increases in the physiological and performance capacities of endurance athletes (Laursen, 2010; Buchheit and Laursen, 2013). Although these training modalities stimulate mitochondrial biogenesis differently (Gibala and McGee, 2008; Laursen, 2010), both training techniques result in an increased capacity to generate ATP aerobically, which ultimately can lead to an increased endurance performance.

When athletes are dependent on their upper body in endurance events—for example in handcycling or wheelchair racing—less active muscle mass is available to generate power compared to lower body exercise. It is proposed that training a smaller muscle mass may result in different physiological responses to endurance training compared to exercise regimes involving the larger lower bodies' muscle mass (Miles et al., 1989; Schneider et al., 2002). To date, limited research is available regarding upper body endurance training and the accompanying physiological adaptations. In previous studies, different MICT handcycling and armcranking protocols resulted in improved peak oxygen uptake (peakVO_2) and peak power output (peakPO) in disabled and/or spinal cord injured patients, in healthy elderly and in able-bodied men and women (Franklin, 1989; Pogliaghi et al., 2006; Valent et al., 2007; Hettinga et al., 2016). In lower body endurance training, HIIT was shown to be a time-efficient training method to induce both central and peripheral adaptations (Gibala et al., 2012), and is now considered more effective at improving the physiological and performance capacity of untrained individuals, recreationally active and trained athletes compared to MICT for a given training volume (Weston et al., 2014; Milanović et al., 2015). The effects of HIIT in upper body endurance training have been studied in patients with chronic tetraplegia (Valent et al., 2009). Although significant improvements in peakVO_2 and peakPO were reported in this study (Valent et al., 2009), the findings are of limited value in the context of sports training, due to large respiratory, biomechanical and metabolic differences between patients undergoing active rehabilitation and trained handcyclists (Lovell et al., 2012; Fischer et al., 2014; de Groot et al., 2014). In order to develop understanding and guidelines for upper body endurance training for sports and/or advanced rehabilitation practice, the effectiveness and possible role of HIIT is yet to be established and compared with other training protocols.

The aim of the current study was to evaluate the effects of HIIT, known to be effective in lower body exercise, and MICT, a protocol more common in handcycling, on the physiological capacity and handcycling performance of able-bodied men in a well-controlled laboratory setting. It was hypothesized that HIIT would improve peakVO_2 and peakPO, but not to a larger extent than a period of MICT. A secondary aim of this study was to add to the scarce reference data available concerning able-bodied participants during handcycling. Reference data are necessary to better understand the physiology of the upper body in relation to exercise using handcycling, to facilitate the interpretation of data from disabled individuals.

METHODS

Participants

Twenty four recreationally active able-bodied men, unaccustomed to upper body endurance training, volunteered to take part in the study (mean ± SD: age: 22 ± 2 year; height: 1.84 ± 0.04 m; body mass: 79 ± 10 kg). During an initial visit, study details and participation requirements were explained, and written informed consent was obtained. During visit one, participants completed three 6 min bouts of handcycling on a motor driven treadmill to familiarize with the handcycle's propulsion and steering mechanisms. Participants were instructed not to alter other training activities outside those of the study protocol. The study received approval from the local ethics committee (Center for Human Movement Sciences, Groningen) and was conducted in accordance with the Declaration of Helsinki.

Design

Before and after the 7-week training intervention, an incremental handcycling test was performed to obtain peak cardiovascular variables and to evaluate handcycling performance. Based on peakPO ascertained during the incremental pre-test, participants were matched and then randomly assigned to HIIT, MICT or a non-training control group [(CON), $3 \times n = 8$]. Participants in HIIT and MICT visited the laboratory three times per week for 7 weeks to complete a total of 21 training sessions. Participants in HIIT completed 2 interval training sessions per week, performing 4×4 min intervals at 85% heart rate reserve (%HRR), and 1 moderate intensity continuous training sessions at 55 %HRR (every 2nd training session of the week). The MICT group performed 3 continuous training sessions of 30 min per week, at an average training intensity of 55 %HRR. CON received no training, and was asked to maintain their regular activity level during the experimental period.

All training sessions and incremental tests were performed on a motor driven treadmill (Enraf Nonius, The Netherlands) in a handcycle (see **Figure 1**), which consisted of a wheelchair (RGKWheelchair Inc., England) with a mounted handcycling unit (Double Performance, The Netherlands). Prior to each training session, participants in both HIIT and MICT performed 2×4 min self-paced warm up bouts on a customized handcycle (Wolturnus, Denmark) placed on a cycletrainer (Tacx B.V., The Netherlands). Training sessions were monitored by heart rate (Polar Electro, Finland) and power output (PowerTap SL, United States). After each training session rating of perceived exertion (RPE, Borg, 1970) and local perceived discomfort (LPD) of the upper body were obtained (Bafghi et al., 2008).

FIGURE 1 | The experimental handcycle, consisting of a wheelchair with a mounted handcycling unit in front of the pulley system on the motor driven treadmill.

HIIT Training

In the first and third training session of each week, HIIT participants completed a 4 × 4 min interval training protocol. During the 4 min work intervals, average training intensity was 85 %HRR (see **Figure 2**), based on a protocol previously used by Helgerud et al. (2007) in untrained runners. Exercise intensity was achieved by adding or reducing the workload through the pulley system placed behind the treadmill (as described in Hettinga et al., 2016, see **Figure 1**), while riding at a fixed velocity of 1.67 $m \cdot s^{-1}$. Workload was adjusted after every minute in the 4-min work intervals. Between work intervals, participants received 3 min of passive rest. In the first 2 weeks of the training program, training intensity in the work intervals increased from 65 to 85 %HRR to minimize the risk of injuries and overtraining. The second training session of each week was a continuous training session of 30 min, as described in MICT Training below.

MICT Training

In MICT, participants performed three continuous training sessions per week, each for a duration of 30 min at an average exercise intensity of 55 %HRR. This intensity was achieved by either "resistance" or "velocity" training, as is common in wheelchair (van der Woude et al., 1999) and handcycle training (Hettinga et al., 2016). Three different temporal patterns were used in a fixed sequence for each subject (see **Figure 3**), to vary the training stimulus systematically over time (van der Woude et al., 1999). In resistance training, the workload was varied around a mean exercise intensity of 55 %HRR by adding or reducing workload through the pulley system every 3 min, while the velocity was kept constant at 1.67 $m \cdot s^{-1}$. During velocity training, resistance was kept constant at a workload corresponding to the workload required to handcycle at 55 %HRR, only now velocity was varied every 3 min. To assure a comfortable cadence (50–90 rpm) gearing was changed on increased riding velocities. MICT performed 11 "velocity" sessions and 10 "resistance" over the course of the study.

Incremental Exercise Test

Participants performed an incremental exercise test before and after the training intervention. Participants were asked to refrain from consuming alcohol and caffeine for at least 24-h, as well as from engaging in strenuous exercise for at least 48-h prior to testing. Both incremental pre-test and post-tests were performed at the same time of the day, to minimize circadian effects. To elicit valid maximal physiological values for upper body exercise, a test protocol was designed specific to this group after several pilot tests conducted in our laboratory. In the current protocol, gearing was fixed and treadmill velocity was a constant 1.67 $m \cdot s^{-1}$, so participants rode at 70 rpm (Sawka et al., 1983). Before the start of the incremental exercise test, a 5 min submaximal warm up was executed at 30 W. The initial power output of the test was 30 W, which increased 10 W every minute until exhaustion. PO was increased by adding weight to a pulley system at the back of the handcycle (see **Figure 1**) as introduced by van der Woude et al. (1999). When voluntary exhaustion was reached, or when rpm dropped below 70, the test was ended. Respiratory parameters were measured breath by breath, using open circuit spirometry (Oxycon Delta, Germany). The gas analyzer was calibrated prior to each test using room air, a Jaeger 3-1 syringe and a calibration gas (16.0% O_2, 5.0% CO_2). Peak power output (peakPO), peak oxygen uptake (peakVO_2), peak heart rate (peakHR), peak minute ventilation (peakVE) and the respiratory exchange ratio (RER) were calculated between the 20th and 50th s of every completed minute.

Data Analysis

All data were analyzed and calculated using SPSS 17.0 (SPSS Inc., USA), Office Excel 2010 (Microsoft Corporation, USA) and Matlab 2013 (The Mathworks, USA) and are presented as mean ± SD. Participant and training characteristics were compared using one way ANOVA. The effect of the intervention period on the physiological capacity (peakVO_2, peakVE, peakHR, and RER) and handcycling performance (peakPO) within and between groups was tested using a repeated measures ANOVA. *Post hoc* Bonferroni pairwise comparisons were used to show differences between experimental groups. The significance level of all tests was set at $p < 0.05$.

RESULTS

All 24 participants completed the study. At baseline, there were no statistical differences between the three groups with regard to age, height, body mass, and performance or cardiorespiratory variables (see **Tables 1, 2**). In HIIT, training intensity increased from 65 to 85 %HRR in the first four interval training sessions.

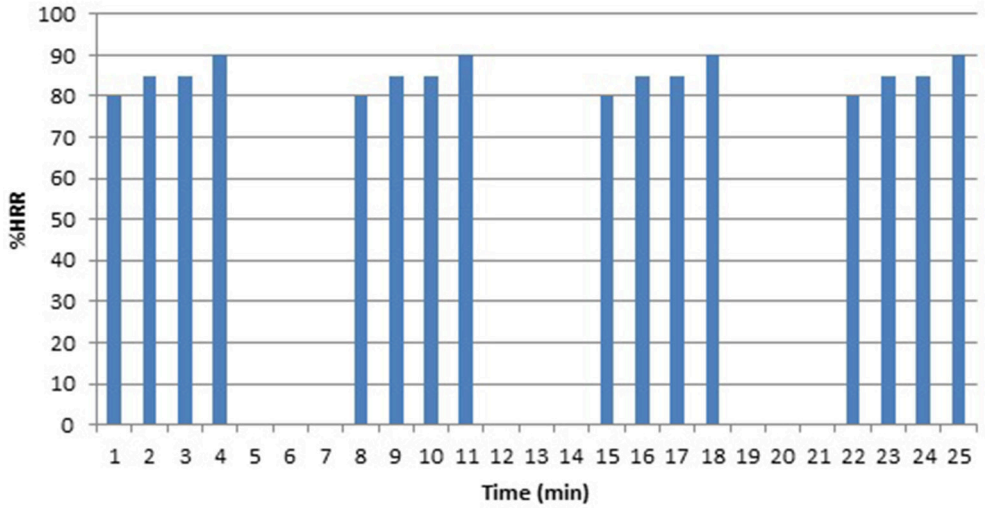

FIGURE 2 | Temporal pattern (as %HRR per minute) that was imposed in the 4 × 4 min interval training sessions in HIIT, in which the resistance was changed after every minute in the 4 min work intervals.

Thereafter, 10 interval training sessions were performed at an average intensity of 84.0 ± 1.8 %HRR. The HIIT group performed seven (3 resistance, 4 velocity) continuous training sessions at 55.5 ± 1.4 %HRR. Training intensity for the MICT group during the 21 training sessions averaged 55.3 ± 0.3% HRR. The external training load, calculated as average PO per training session∗duration of a session, did not differ between MICT and HIIT [2.32 ± 0.3 kJ vs. 2.28 ± 0.3 kJ, respectively ($p = 0.713$)]. Total training time was significantly lower in HIIT compared to MICT (434 vs. 630 min). HIIT was perceived more exhaustive than MICT ($p < 0.001$), with an average session RPE of 16 ± 1, where MICT was rated 13 ± 2. The HIIT group experienced significantly more discomfort in the upper body during the training, indicated by a LPD score of 10.7 ± 5 compared to MICT who reported on average of 5.9 ± 3 ($p < 0.001$).

Table 2 shows the peak physiological and performance capacity of both training groups and the non-training control group before (pre) and after (post) the experimental period. Repeated measures ANOVA showed there were significant increases in peakVO2, peakPO and peakVE, both within and between groups over the course of the study ($P < 0.05$). There were no main effects of time or group on body mass, RER, or peakHR.

Interaction effects showed different responses according to group. HIIT resulted in a significantly higher final peakPO than both MICT and CON ($p < 0.001$). The improvement after MICT was significantly higher than CON ($p < 0.001$). These final peakPO values represent a 47.1 ± 20.7% increase for HIIT, a 32.2 ± 8.1% increase for MICT and a 0.3 ± 5.7% increase in CON, from baseline. HIIT resulted in a significantly higher final peakVO_2 than MICT and CON ($p < 0.001$). The improvement after MICT was significantly higher than after CON ($p <0.001$). The final peakVO_2 values represent a 22.2 ± 8.1% increase for HIIT, a 10.7 ± 12.9% increase for MICT and a 4.7 ± 10.2% increase for CON. HIIT ($p = 0.002$) and MICT ($p = 0.028$) resulted in a significantly higher final peakVE than CON. These final peakVE values represent a 34.0 ± 20.6% increase for HIIT, a 25.0 ± 18.3% increase for MICT and a 1.1 ± 13.2% decrease for CON. A significant increase in RER (6.1 ± 2.4%) was found on the post-test for MICT ($p = 0.002$), which was in contrast to the decrease of 6.3 ± 6.0% after CON ($p = 0.009$).

DISCUSSION

Adaptations to endurance training are well documented for lower body exercise such as running and cycling. However, less research is available regarding upper body endurance training in general, and in handcycling specifically. This study aimed to evaluate the effects of HIIT and MICT on the physiological capacity and handcycling performance of able-bodied men. In lower body exercise, both these training modalities have been shown to improve endurance performance. The most striking outcomes of the present study were the large improvements in peakVO_2, peakPO, and peakVE after 7 weeks of HIIT. MICT also resulted in notable but smaller improvements in peakVO_2, peakPO, and peakVE. Thus, it seems that successful training protocols of lower body exercise can be used to design upper body endurance training programs in handcycling.

To date, research on handcycling is scarce and primarily focuses on the use of handcycling in the rehabilitation of spinal cord injured patients (Valent et al., 2009; Hettinga et al., 2010). With the increased interest in handcycling as a competitive sport over the past two decades, scientific interest also increased, resulting in several descriptive studies of the physiological and performance profiles of trained handcyclists (Abel et al., 2006; Lovell et al., 2012; Fischer et al., 2014). In handcycling, athletes compete in mass start road races and time trials in five different ability classes, based on their anatomic level of spinal cord injury and/or associated functional limitations (UCI, 2016). Recently, peakVO_2 and peakPO were identified as important predictors

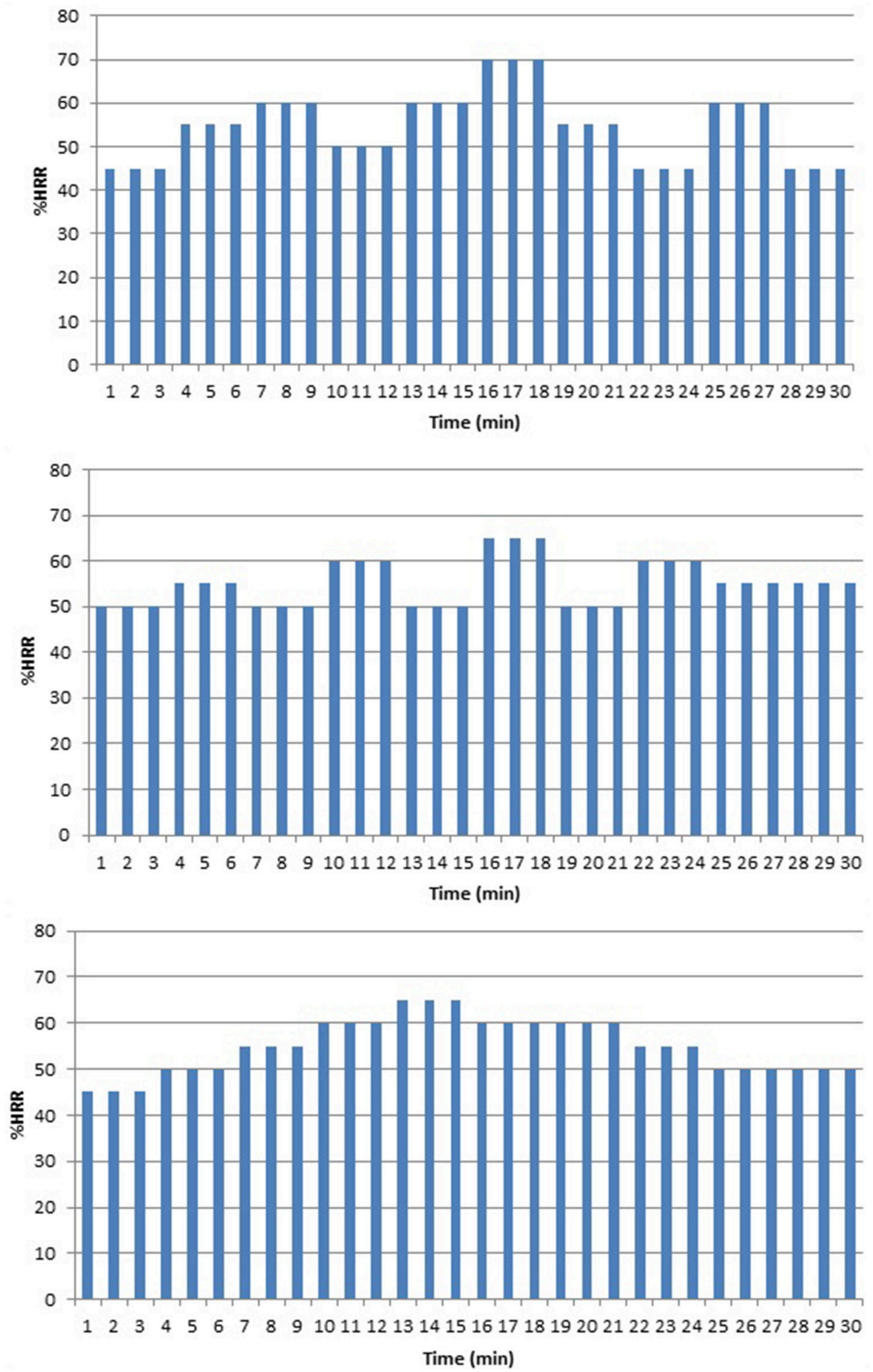

FIGURE 3 | Temporal patterns (as %HRR per minute) that were imposed in the 30 min continuous training sessions in MICT and HIIT by varying either velocity or resistance after every 3 min.

TABLE 1 | Participant characteristics for CON, MICT, and HIIT.

	CON ($n = 8$)	MICT ($n = 8$)	HIIT ($n = 8$)
Age (years)	23 ± 1.7	21 ± 2.3	23 ± 1.8
Height (m)	1.86 ± 0.02	1.83.0 ± 0.04	1.85 ± 0.04
Body mass (kg)	86.6 ± 10.1	75.1 ± 10.3	77.4 ± 7.2

Data are reported as mean ± SD.
No significant differences were found in baseline values between the training groups and the non-training control group.

of time trial (Lovell et al., 2012; de Groot et al., 2014) and race performance in trained handcyclists (Janssen et al., 2001; Fischer et al., 2015). In the present study, after HIIT and MICT there was an increase in both these variables in able-bodied men, indicating the effectiveness and importance of both these training modalities in the design of an optimal endurance training program for upper body exercise, thereby indicating the potential relevance to handcycling athletes and/or patients in an advance rehabilitation setting.

The present study provides interesting insights into the responses to upper body endurance training. The HIIT protocol resulted in significantly higher improvements in peakVO_2 and peakPO than both MICT and CON after the 7-week intervention period. This despite a 22% lower training time compared to MICT. Although the external training load was not matched *per se* prior to the training interventions, no differences were found in the average training load over the 21 training sessions across training groups. It has been suggested a longer duration of training sessions could compensate for lower intensity exercise (Overend et al., 1992; Warburton et al., 2004). However, the present study, with matched total workload and number of training sessions, does not support this claim. Instead, our results are consistent with those of Helgerud et al. (2007) who found that intensity of training cannot be compensated for by longer duration, and showed larger improvements after HIIT compared to MICT.

In the current study, we adopted an interval training protocol that resulted in an improved VO_2max(+7.2%) in a group of untrained runners after an 8 week training program (Helgerud et al., 2007). Improvements in HIIT in the current study were substantially larger compared to these findings, which can be explained by the initial inexperience of upper body endurance training in our participants. Osawa et al. (2014) reported an increase in peakPO after a 16-week period of combined leg and arm cranking HIIT. In their study, participants performed 32 training sessions including four 6 × 1 min work intervals at a workload >90% of peakPO, interspersed with 1 min active recovery (Osawa et al., 2014). Given the smaller increase of peakPO along with lower baseline values compared with the current study [96 ± 12 W (+25.0 ± 8.3%) vs. 133.2 ± 26.2 W (+47.1 ± 20.7%)], our interval protocol appears to be favorable to improve handcycling performance. The duration of work intervals is important in the programming of HIIT. Longer duration intervals may be more effective in upper body training, due to the different oxygen uptake kinetics of upper body compared to lower body exercise (Koppo et al., 2002). The relatively slow response of the "fast component" of VO_2, and the relatively late emergence of the slow VO_2 component in upper body compared to lower body exercise (Koppo et al., 2002), suggests that the interval duration of HIIT required to improve oxygen uptake must be at least 2 min in order to allow VO_2 to peak. This is in line with Midgley et al. (2006), who stated that in lower body HIIT, longer work intervals elicit maximal oxygen uptake, or at least a very high percentage of peakVO_2 and therefore provide a more effective stimulus for enhancing oxygen uptake compared to short duration intervals.

In many lower body endurance sports, around ∼80% of athletes' training sessions are performed at a relatively low intensity (Seiler, 2010). The improvements as a result of MICT therefore are of interest in the context of upper body endurance training. The present study showed that a training dose of 7 weeks, 3 × 30 min per week of handcycling at an average of 55 %HRR, resulted in improvements in incremental handcycling performance on the parameters peakVO_2 and peakPO. The increase in peakPO is in line with the findings of Hettinga et al. (2016), who reported an increase in peakPO after 7 weeks of 3 × 30 min continuous handcycling training at 65 %HRR in able-bodied women. The increase in peakVO_2 in the current study was lower than the reported increase in the study of Hettinga et al. (2016) (+18.1% vs. 10.7 ± 12.9% respectively). This difference may be explained by the higher baseline values of the male participants (33.2 ± 4.5 $ml \cdot kg^{-1} \cdot min^{-1}$) in the current study compared to female participants (28.3 ± 5.1 $ml \cdot kg^{-1} \cdot min^{-1}$) in Hettinga et al. (2016). Another explanation for the difference in improvement can be attributed to the difference in relative training intensity. In the current study, participants in MICT trained at an average power output corresponding to 55 %HRR. This training intensity was based on the findings of Knechtle et al. (2004) who reported the highest fat oxidation at 55% peakVO_2 in well-trained handcyclists. In contrast, the participants in the study by Hettinga et al. (2016) trained at an average power output corresponding to 65 %HRR. Åstrand (2003) stated that the minimum training intensity to improve peakVO_2 must be around 55–65 %HRR. The limited data now available on handcycling suggest that an exercise intensity of 65 %HRR in MICT is more favorable to improve the upper body endurance capacity.

Both HIIT and MICT improved handcycling performance and physiological capacity. Previous work from our lab (Hettinga et al., 2016) showed that training adaptations after MICT in handcycling are local and exercise specific, since no transfer effects were found in an incremental cycling test. We therefore assume that the adaptations that are responsible for the changes in peakVO_2 and peakPO after MICT in the current study are primarily local. Based on the results of the current study, it is hard to state which mechanisms are responsible for the differences in improvements after HIIT and MICT. It can be proposed that participants in HIIT became more familiar with higher workloads, as HIIT required higher work intensities. This may have contributed to an increased skeletal muscle buffering capacity, as was apparent in well trained cyclists after 6 HIIT training sessions (Weston et al., 1997). We can also speculate that the higher workloads in HIIT resulted in an increased force of the

TABLE 2 | Changes in physiological capacity and handcycling performance from pre- to post-training period for CON, MICT, and HIIT.

	CON		MICT		HIIT		
	Pre-test	Post-test	Pre-test	Post-test	Pre-test	Post-test	Interaction (group*time)
peakPO (W)	143.3 ± 13.7	143.2 ± 10.4	128.9 ± 26.9	169.0 ± 27.8*§	133.2 ± 26.2	191.3 ± 16.2*§	$F_{(2)} = 72.19$, $p < 0.001$
peakVO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	31.5 ± 3.0	32.9 ± 4.3	33.2 ± 4.5	36.5 ± 4.5*§	34.3 ± 3.8	41.9 ± 4.9*§	$F_{(2)} = 7.66$, $p = 0.003$
peakVE ($L \cdot min^{-1}$)	105.7 ± 11.0	104.1 ± 16.2	89.7 ± 20.3	109.4 ± 13.4*§	99.7 ± 20.1	130.4 ± 13.9*§	$F_{(2)} = 11.32$, $p = 0.001$
RER	1.24 ± 0.08	1.16 ± 0.06*	1.17 ± 0.05	1.24 ± 0.03*	1.19 ± 0.05	1.21 ± 0.06	$F_{(2)} = 12.57$, $p < 0.000$
peakHR (bpm)	182 ± 8	186 ± 13	180 ± 21	176 ± 18	188 ± 9	190 ± 3	$F_{(2)} = 2.25$, $p = 0.132$

Data are reported as mean ± SD.

No significant differences were found in baseline values between the training groups and the non-training control group.

**Significant different from pre-test ($p < 0.05$).*

*§Significant interaction of group*time ($p < 0.05$).*

working muscles in the handcycling motion. The m. deltoid, m. triceps and m. trapezius are the muscles that produce the main force throughout propulsion in handcycling (Arnet et al., 2012). An increase in force of these muscle groups would decrease the relative force in each propulsion at a given submaximal work intensity. This would allow an increased recruitment of the slow twitch (type 1) fibers and a reduced rate of fast twitch (type 2) fiber recruitment. This in turn may result in an improved work efficiency during the (sub)maximal workloads in the incremental handcycling test. Jacobs (2009) showed that 12 weeks of upper body strength training, without any endurance training, increased peakVO_2 in paraplegic individuals. Similarly, the use of heavy strength training has been shown to increase upper body endurance capacity in kayaking (Ualí et al., 2012) and wheelchair racing (Turbanski and Schmidtbleicher, 2010). However, at present this theory is largely speculative in relation to the current study. We did not assess changes in muscle strength after HIIT or MICT, which is a limitation and we are consequently unable to ascertain the relationship between strength, peakPO and peakVO_2. Adaptations in muscle strength after HIIT and MICT, but also after structured strength training and concurrent endurance training in relation to improvements in handcycling performance should be assessed in future research to determine optimal training regimes.

Previously, the use of an 8–12 week interval training protocol was effective in improving peakVO_2 and peakPO in the rehabilitation of spinal cord injured patients (Valent et al., 2009). Our study is one of the first to address different training modalities in upper body endurance training. The use of a homogeneous group of able-bodied men allowed us to compare responses to HIIT and MICT in a controlled setting, adding to data available required for establishing training prescriptions for upper body exercise. Although the participants in the current study were not trained handcyclists, average peakVO_2 and peakPO in this group of able–bodied men was similar to those reported for trained handcyclists (Janssen et al., 2001; de Groot et al., 2014). Based on the results of the current study, it is therefore expected that both HIIT and MICT can alter the physical capacity of recreationally active and trained handcyclists. However, as differences in physiology have been evidenced between able-bodied and disabled individuals (Bernard et al., 2000; Schilero et al., 2009), it is important to evaluate how data collected in able-bodied participants compares with people with different disabilities. The handcycle used in the current study is typically used for activities of daily living (Hettinga et al., 2010). Differences between the experimental handcycle and racing handcycles, the accompanying differences in body positioning, muscle recruitment, and movement possibilities are noteworthy (Zipfel et al., 2009). How these differences might influence adaptations to HIIT and MICT are to be assessed in future research.

CONCLUSION

The aim of the current study was to evaluate the effects of a 7 week HIIT or MICT training intervention on the physiological capacity and handcycling performance of able-bodied men in a well-controlled laboratory setting. The results indicate that HIIT improves upper body endurance capacity (peakVO_2 +22.2 ± 8.1%) and handcycling performance (peakPO +47.1 ± 20.7%) significantly more than MICT and CON. These findings suggest that HIIT sessions should be included in the training regimes of recreationally active and trained handcyclists to improve their upper body endurance performance. MICT also produced notable yet smaller improvements, by altering peakVO_2 (+10.7 ± 12.9%) and peakPO (+32.2% ± 8.1). It thus seems that both HIIT and MICT, that are known to be effective in lower body exercise can be used to design upper body endurance training programs in handcycling.

AUTHOR CONTRIBUTIONS

PS, FH, and LV contributed to conception and design of the work. PS conducted the experiment, analyzed the data and wrote the first draft. All authors (PS, FH, LV, and KR) were involved in further data analysis and drafting, and revised the manuscript critically for important intellectual content. All authors have approved the final version of the manuscript, agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved, and all persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

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Short and Long Term Effects of High-Intensity Interval Training on Hormones, Metabolites, Antioxidant System, Glycogen Concentration, and Aerobic Performance Adaptations in Rats

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The purpose of the study was to investigate the effects of short and long term High-Intensity Interval Training (HIIT) on anaerobic and aerobic performance, creatinine, uric acid, urea, creatine kinase, lactate dehydrogenase, catalase, superoxide dismutase, testosterone, corticosterone, and glycogen concentration (liver, soleus, and gastrocnemius). The Wistar rats were separated in two groups: HIIT and sedentary/control (CT). The lactate minimum (LM) was used to evaluate the aerobic and anaerobic performance (AP) (baseline, 6, and 12 weeks). The lactate peak determination consisted of two swim bouts at 13% of body weight (bw): (1) 30 s of effort; (2) 30 s of passive recovery; (3) exercise until exhaustion (AP). Tethered loads equivalent to 3.5, 4.0, 4.5, 5.0, 5.5, and 6.5% bw were performed in incremental phase. The aerobic capacity in HIIT group increased after 12 weeks (5.2 ± 0.2% bw) in relation to baseline (4.4 ± 0.2% bw), but not after 6 weeks (4.5 ± 0.3% bw). The exhaustion time in HIIT group showed higher values than CT after 6 (HIIT = 58 ± 5 s; CT = 40 ± 7 s) and 12 weeks (HIIT = 62 ± 7 s; CT = 49 ± 3 s). Glycogen (mg/100 mg) increased in gastrocnemius for HIIT group after 6 weeks (0.757 ± 0.076) and 12 weeks (1.014 ± 0.157) in comparison to baseline (0.358 ± 0.024). In soleus, the HIIT increased glycogen after 6 weeks (0.738 ± 0.057) and 12 weeks (0.709 ± 0.085) in comparison to baseline (0.417 ± 0.035). The glycogen in liver increased after HIIT 12 weeks (4.079 ± 0.319) in relation to baseline (2.400 ± 0.416). The corticosterone (ng/mL) in HIIT increased after 6 weeks (529.0 ± 30.5) and reduced after 12 weeks (153.6 ± 14.5) in comparison to baseline (370.0 ± 18.3). In conclusion, long term HIIT enhanced the aerobic capacity, but short term was not enough to cause aerobic adaptations. The anaerobic performance increased in HIIT short and long term compared with CT, without differences between HIIT short and long term. Furthermore, the glycogen super-compensation increased after

short and long term HIIT in comparison to baseline and CT group. The corticosterone increased after 6 weeks, but reduces after 12 weeks. No significant alterations were observed in urea, uric acid, testosterone, catalase, superoxide dismutase, sulfhydryl groups, and creatine kinase in HIIT group in relation to baseline and CT.

Keywords: training, anaerobic threshold, stress biomarkers, metabolism, super-compensation

INTRODUCTION

The High-intensity interval training (HIIT) is characterized by short bouts of exercise, with intensities equal or superior to anaerobic threshold, separated by periods of recovery (Billat, 2001; Gibala and Jones, 2013). HIIT is "infinitely variable," determined by intensity, recovery, series and duration (Gibala and Jones, 2013). However, little is known concerning the glycogen stores in different tissues, hormonal concentration, stress biomarkers, antioxidant systems, metabolism, and performance adaptations that may be obtained following HIIT with short and long duration.

Studies have showed that HIIT can improve the aerobic performance in a short term beyond those found by endurance training with low intensity and high volume of exercise (Rodas et al., 2000; Laursen and Jenkins, 2002; Jensen et al., 2004; Helgerud et al., 2007; Laursen, 2010; de Araujo et al., 2015; Naimo et al., 2015). Due its high intensity feature, the HIIT may be excessive, and instead of improving, shows an unaltered performance responses, physiological stress, and overtraining symptoms (Billat et al., 1999). Thus, the most HIIT protocols are commonly performed during short term (Laursen and Jenkins, 2002; Chia-Lun et al., 2016). However, are still unknown the ideal duration of HIIT to enhance aerobic performance without overtraining symptoms.

In literature review, Laursen and Jenkins (2002) showed that HIIT is effective to enhance the anaerobic threshold, maximal oxygen uptake, endurance performance, type I fibers, citrate synthase activity, and other aerobic variables in sedentary and highly trained athletes. These authors reported that HIIT duration in sedentary, highly trained cyclists and runners ranges from 2 to 8 weeks for the most interventions. A systematic review and meta-analysis reported studies lasting 2–8 weeks too (Sloth et al., 2013). Among the few studies that used the protocols above 8 weeks, the duration ranges 10–15 weeks (Hickson et al., 1977; Simoneau et al., 1985, 1987; Heydari et al., 2012) However, these authors neither analyzed a large number of biomarkers to detect the overtraining symptoms nor compared the magnitude of physiological and performance adaptations with short term.

A persistent combination of inappropriate intensities and insufficient recoveries can lead to accentuate physiological disturbance, immunosuppression and as consequence decline in performance (Halson and Jeukendrup, 2004; Hohl et al., 2009). Billat et al. (1999) showed that 4 weeks of HIIT not enhanced the performance and increased fatigue symptom, called overtraining by the authors. In this study, the fatigue symptom was based only in sympathetic activity, measured by plasma noradrenaline, making necessary other stress biomarkers and period of maladaptation to confirm overtraining symptoms in accordance to Consensus Statement (Meeusen et al., 2013).

Metabolites, enzymes, antioxidant system, immune system, anabolic, and catabolic hormones and energy stores have been used to follow the physiological responses during chronic physical stress (Halson and Jeukendrup, 2004). These analyses are essential to know workload responses related to training. However, the physiological response derived from HIIT at different durations is not yet well established. Studying HIIT in laboratory rats enables to investigate precisely the biomarkers adaptations due to better methodological control (i.e., same strain, age, and environment), extensive periods of controlled intervention and possibility to increase the number and complexity of analysis in comparison to human (Booth et al., 2010).

Thus, the present study was designed to investigate the physiological and performance adaptations after 6 weeks (short-term) and 12 weeks (long-term) of HIIT in rats. Specifically, to analyze metabolites (creatinine, uric acid, urea), muscle injury markers (creatine kinase, lactate dehydrogenase), antioxidant enzymes (catalase, superoxide dismutase), hormones (testosterone and corticosterone), glycogen concentrations (liver, soleus and gastrocnemius), and immune system (white blood cells) at baseline, after 6 and 12 weeks. It was hypothesized that HIIT short term: (1) increases aerobic and anaerobic performance similarly HIIT long term and (2) causes lower physiological stress in comparison to HIIT long term.

METHODS

Animals

All experiments involving animals were performed in accordance to the principles of laboratory animal care (NIH publication No. 86-23, revised 1985). The experimental protocol was approved by specific resolutions on Bioethics in Experiments with Animals (no. 93/08)

Fifty male *Rattus norvegicus albinus* (Wistar) rats, 60 days old, were selected for this study. Rats were maintained in collective cages (5 rats/cage). The animals received water and commercial chow (23.5% protein, 6.5% fat, 70% carbohydrate, Purina 5008, St. Louis, MO) *ad libitum* and were housed at 22 ± 2°C with an inverted 12:12-h light–dark cycle (18:00–06:00 lights on).

Adaptation to Water

The purpose of adaptation to the water was to reduce water stress without promoting physiological adaptations to physical training. The adaptation to the water environment consisted of a 5-min (31 ± 1°C) exposure daily, in tanks (80 × 80 × 80 cm) subdivided into four cylindrical compartments of 30 cm

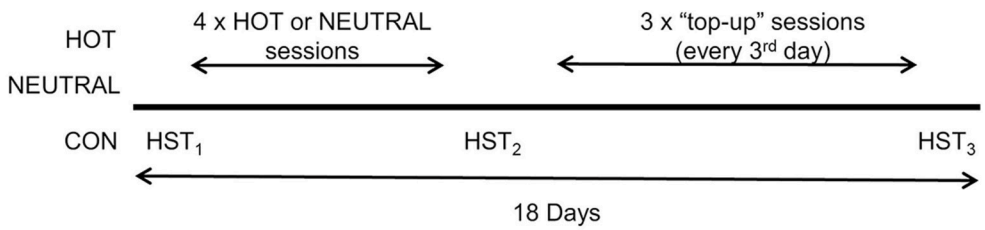

FIGURE 1 | Study timeline for Heat Training (HOT), Thermo-neutral Training (NEUTRAL), and Control (CON) groups.

Methodology

Assessment of VO_2 maxwas undertaken on a cycle ergometer (VeloTron and Velotron Coaching Software, Racermate, United States) at least 72 h before beginning the experimental trials. The intervention comprised a short-term training protocol of four training sessions on consecutive days, followed by three supplementary training sessions every 3 days. All participants completed three heat stress tests (HST_{1-3}) and seven training sessions over 18 days, with HST_1 performed as a baseline measure of heat tolerance, HST_2 completed between the end of the short-term program and before beginning the supplementary top-up training, and HST_3 completed 48 h after the final supplementary training session (**Figure 1**). Each group performed the HST in a custom-built environmental chamber at a temperature of 35°C and 70% RH. Participants in the HOT and NEUTRAL conditions completed exercise training sessions in hot and humid (35°C and 70% RH) or thermo-neutral conditions (20°C and 50% RH), respectively. Participants in the CON group did not undertake exercise training but completed the three HST's at the same intervals as HOT and NEUTRAL groups. Participants were instructed to rest and avoid moderate or high levels of physical activity on days that they were not required to attend the laboratory.

Test of Maximal Oxygen Uptake

Maximal oxygen uptake was determined by an incremental test to exhaustion on a cycle ergometer (VeloTron and Velotron Coaching Software, Racermate, United States). Briefly, the test began with participants cycling at 80–90 rpm at 120 W, with the workload increasing by 20 W every min until volitional exhaustion or when cadence was unable to be maintained above 80 rpm. Expired gases were collected via a one-way breathing system (Hans-Rudulph, United States) and analyzed by a calibrated Moxus Metabolics Measurement cart (AEI Technologies, United States). Attainment of VO_2 max was determined by the satisfaction of standard criteria (Midgley et al., 2007).

Heat Stress Test

The heat stress test was of similar design to earlier work (Garrett et al., 2009; Lorenzo et al., 2010) and comprised cycling for 3 × 10 min submaximal workloads with a 3 min rest period between workloads, followed by a 5-km self-paced TT. Following a 5 min standardized warm-up, the participants completed three 10 min workloads at 50, 60, and 70% of their peak wattage corresponding to their individualized VO_2 max. After the 70% workload was complete, a 5 min rest period was given before the start of the TT. Participants were able to view their rpm and were informed of the distance traveled every 500 m to assist with pacing. Heart rate (RS400, Polar Elektro, Finland), and core temperature (T_c) (ttec 501-3 data logger and data logger software version 10.1, Nordex Pty Ltd, Australia; MEAS 449 1RJ rectal temperature thermistor, Measurement Specialities, United States) were sampled at 5 s intervals. Fluid intake (water, *ad libitum*), rating of perceived exertion (Borg RPE 6–20, Borg, 1970) and thermal comfort (TComf) were recorded throughout the test. Nude dry body mass was recorded pre and post-exercise on a calibrated set of scales (BF-522W, Tanita, Japan) and body mass was adjusted for fluid loss and expressed as a percentage change.

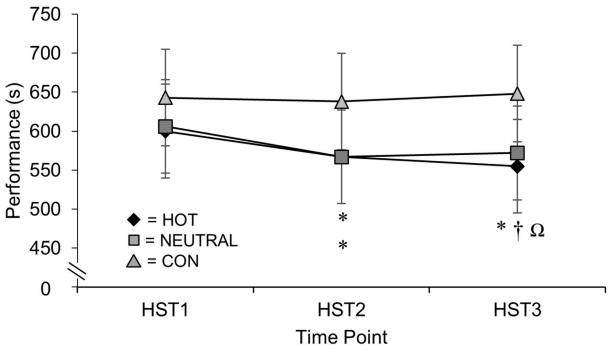

FIGURE 2 | Adjusted means ± SD of 5 km time trial performance (s) across heat stress tests (HST) 1, 2, and 3 for Heat (HOT), Thermo-neutral (NEUTRAL), and Control (CON) groups. *Faster from baseline. †Faster than HST 2. ΩHOT was faster than CON.

Blood Collection

Upon arrival at the laboratory, participants rested for 20 min before blood collection was performed. Blood was drawn in a seated position 10 min before and 10 min after each HST via a 22 g needle from a prominent superficial forearm vein located at the antecubital fossa, and drained directly into an 8.5 ml sterile serum separator Vacutainer tube containing a clot activator and gel for serum separation (Beckton and Dickson, USA). Samples were refrigerated at 4°C for 30 min to allow clotting and then centrifuged at 1000 × g at 6°C for 10 min (Rotina 420R, Hettich, Germany). Serum was removed and stored in 400 μl aliquots that were frozen immediately for

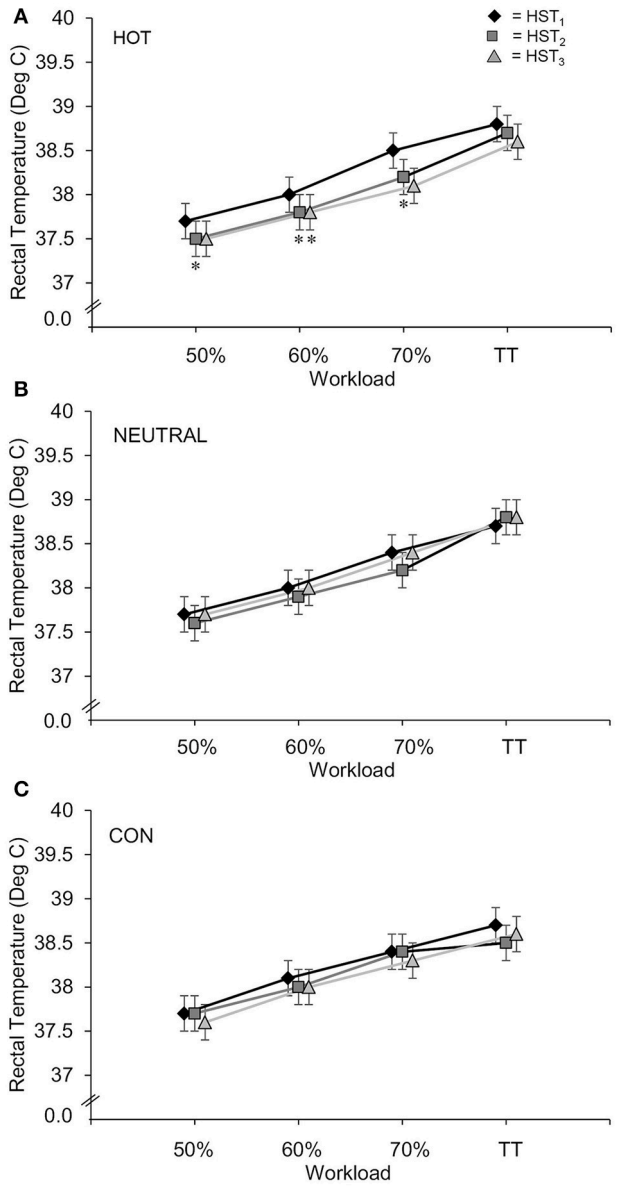

FIGURE 3 | Core temperature for Heat Training (HOT), Thermo-neutral Training (NEUTRAL), and Control (CON) groups during Heat Stress Tests (HST) 1, 2, and 3, expressed as mean ± SD. *Reduced from baseline at HST 2. †Reduced from baseline at HST 3.

a maximum of 3 months at −80°C for later analysis. Serum concentrations of IL-6 (Quantikine HS600B, R&D Systems, United States), IgM (AB137982, Abcam PLC, United Kingdom), and LPS (HIT302, Hycult, Biotechnology, Netherlands) were analyzed in duplicate by ELISA according to manufacturer's instructions.

Aerobic Interval Training

Participants in HOT and NEUTRAL undertook matched aerobic interval training on a cycle ergometer (Monark Ergomedic 828 E, Sweden) in hot and humid (35°C and 70% RH) or thermo-neutral conditions (20°C and 50% RH), respectively. The exercise-training intervention included seven training sessions comprised a standardized 3 min warm-up followed by 4 × 10 min interval at a fixed workload of 55% $\dot{V}O_2$ max. A 3 min rest period was given between each workload and water consumed *ad libitum*. A shorter duration interval-based protocol was chosen to better reflect the training status of the recreationally-trained participants; interval-based training has been shown to be beneficial for heat acclimation (Dawson et al., 1989; Kelly et al., 2016), and shorter duration training can reduce cumulative fatigue (Wingfield et al., 2016) while promoting performance (Nielsen et al., 1997). Heart rate was recorded at 5 s intervals and RPE and TComf recorded at the end of each interval. Participants self-reported symptoms of illness, inflection, soreness, or inflammation prior to the start of each training session. No symptoms of illness or infection were reported.

Statistical Analysis

Data that passed tests for homogeneity of variance were analyzed by a mixed-model analysis of variance or *t*-test (where appropriate) and significance accepted when $p \leq 0.05$. Where significant differences were indicated they were identified with the *post hoc* Tukey Test. Data is expressed as mean ± SD and change scores expressed as mean ± 90% confidence limits (CL). The baseline TT performance (s) was not normally distributed and therefore analysis of covariance was used to investigate between-group differences with participant $\dot{V}O_2$ max employed as the covariate—TT results are expressed as adjusted mean ± SD or 90% CL where appropriate. Standardized effect sizes (ES) were calculated to indicate the magnitude of change and/or difference within- and between-groups. The criteria to interpret the magnitude of ES were: <0.2 trivial, 0.2–0.6 small, 0.6–1.2 moderate, 1.2–2.0 large, and >2.0 very large (Hopkins, 2004).

Determination of biomarker concentrations and curve fit analysis was performed using GraphPad Prism Version 6.03 (GraphPad Software Inc, United States) according to the manufacturer's instructions. The manufacturer stated intra-assay precision was <10% for all assays. Statistical analyses were performed in IBM SPSS Statistics Version 22 (IBM, United States). Power analysis was conducted prior to the study and a minimum of eight participants was deemed sufficient to detect the smallest worthwhile change between means assuming the reference change in 5 km TT performance was approximately twice the magnitude of the typical error of measurement, with a Type I error of 5 and Type II error of 20%.

RESULTS

Heat Stress Test

Between Group Analyses

At HST3 a significant between-group effect for TT was evident between HOT and CON (HOT was faster by 8.2%, ±5.2%, 90% CL, $p = 0.03$). Time trial performance is presented in **Figure 2** as adjusted means from the analysis of covariance. No significant

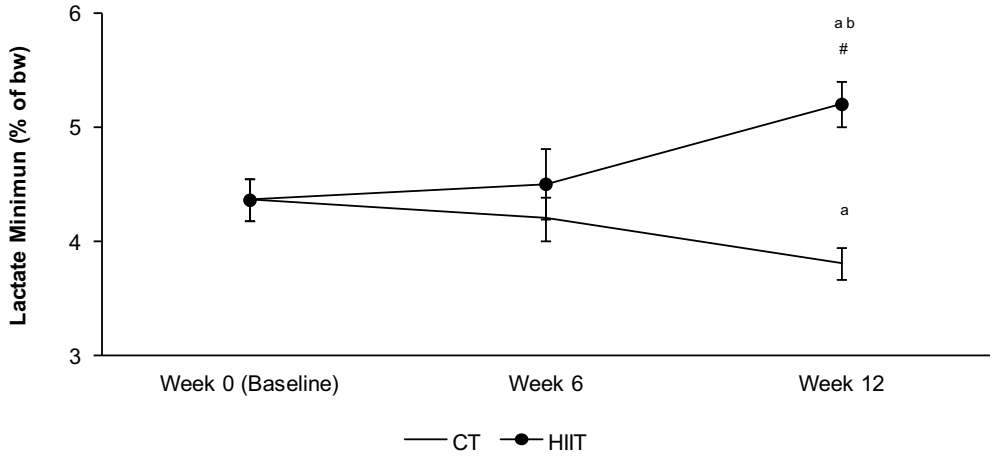

FIGURE 1 | Aerobic capacity measured by Lactate minimum test in each period of training (0 week—baseline, 6 weeks—HIIT short term and 12 weeks—HIIT long term). The intensity of lactate minimum test was determined by % of the body weight (bw). [a]Significantly different ($P < 0.05$) in relation to baseline. [b]Significantly different ($P < 0.05$) in relation to HIIT short term (6 week). [#]Significantly different ($P < 0.05$) in relation to CT group in respective period.

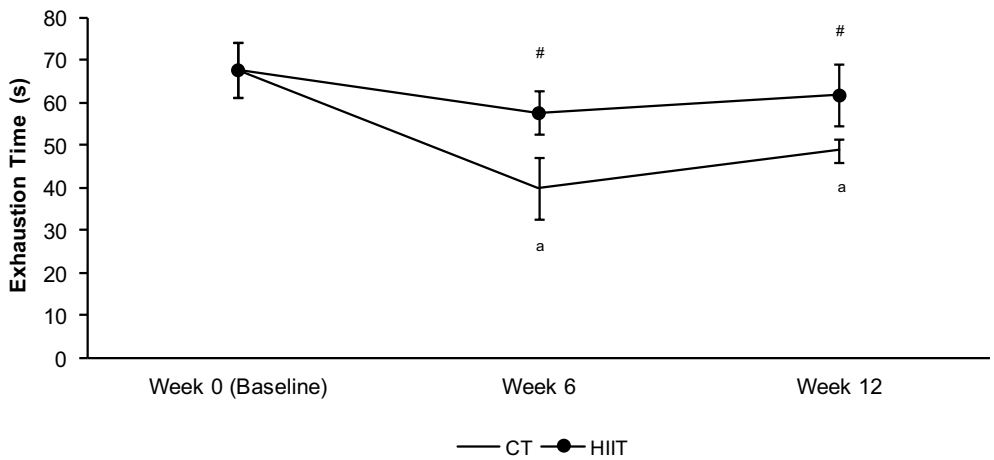

FIGURE 2 | Exhaustion time (s) at 13% of bw during the hyperlactatemia phase of the lactate minimum test. [a]Significantly different ($P < 0.05$) in relation to baseline. [#]Significantly different ($P < 0.05$) in relation to CT group in respective period.

than CT 6 weeks and baseline. HIIT long term (12 weeks) reduced the corticosterone concentration in relation to CT 12 weeks and baseline (**Table 1**).

No differences were found in testosterone, catalase and sulfhydryl groups concentration during experimental period in both HIIT groups (**Table 1**). The SOD activity after HIIT long term was higher in relation to HIIT short term group ($p = 0.05$).

No differences were found in uric acid, creatinine and urea concentrations during experimental period, but after 6 weeks the creatinine and urea had a higher variation (%) in relation to CT when compared to 12 weeks (**Figure 4**).

There are no differences in white blood cells in HIIT short term in comparison to baseline, but HIIT long term had a trend in relation to baseline ($p = 0.07$) and HIIT short term ($p = 0.08$) (**Figure 5**).

DISCUSSION

This is the first study to investigate the effects of HIIT duration on aerobic and anaerobic performances, blood biomarkers and glycogen stores. Our results contradict our hypothesis and showed a higher aerobic capacity, glycogen concentration and lower physiological stress after HIIT long term in comparison to short term.

HIIT has been an important protocol of signaling to a multitude of target cells allowing aerobic adaptations during short term further than traditional endurance training (de Araujo et al., 2013a). Some studies reported that endurance training in rats may attenuate the natural loss, but not increase the aerobic capacity in comparison to baseline (de Araujo et al., 2012, 2013b). The difficulty to develop the aerobic

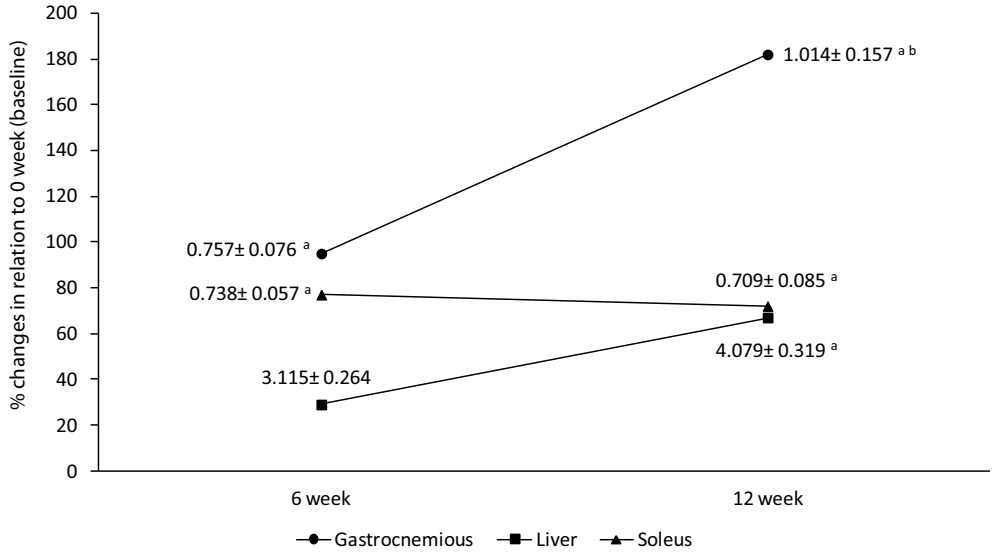

FIGURE 3 | Percentage differences in soleus, liver, and gastronemius tissues when compared to 0 week (baseline). Data labels are presented in mean ± standard error (mg/100 mg). [a]Significantly different ($P < 0.05$) in relation to baseline. [b]Significantly different ($P < 0.05$) in relation to HIIT short term.

TABLE 1 | Biochemical analyses in 0 week (baseline), 6, and 12 week.

	0 Week (baseline)	6 Week		12 Week	
	CT (*n = 10*)	CT (*n = 10*)	HIIT short term (*n = 10*)	CT (*n* = 10)	HIIT long term (*n = 10*)
HORMONES					
Corticosterone (ng/mL)	370.0 ± 18.3	335.6 ± 16.0	529.0 ± 30.5[a#]	362.0 ± 14.9	153.6 ± 14.5[ab#]
Testosterone (ng/mL)	1.8 ± 0.3	1.8 ± 0.5	1.2 ± 0.2	1.2 ± 0.3	1.2 ± 0.3
ENZYMES					
Lactate Dehydrogenase (U/L)	98.4 ± 15.1	71.7 ± 8.9	88.9 ± 15.0	93.1 ± 15.3	92.7 ± 16.6
Creatine Kinase (U/L)	159.5 ± 24.2	118.2 ± 23.9	131.1 ± 21.48	111.9 ± 16.4	89.7 ± 8.38[a]
ANTIOXIDANT					
Catalase (U/mL)	4.1 ± 1.5	5.4 ± 1.8	5. 0 ± 1.2	5.9 ± 1.3	8.1 ± 2.0
Superoxide Dismutase (U/mL)	23.2 ± 7.9	28.0 ± 9.6	20.7 ± 8.0	32.0 ± 7.1	31.1 ± 5.5[b]
Sulfhydryl Groups (μM)	1057 ± 61	1155 ± 84	819 ± 95	1150 ± 81	820 ± 58

[a]*Significantly different in relation to 0 weeks;*
[b]*Significantly different in relation to 6 weeks;*
[#]*Significantly different between groups in respective period.*

capacity has been assigned to high volume of exercise, training monotony and overtraining symptoms (Foster, 1998). Our data showed that aerobic performance after HIIT long term was higher than those observed in endurance protocols (de Araujo et al., 2012, 2013b). While the endurance protocols attenuated the natural loss of aerobic capacity, the HIIT long term increased significantly. Scariot et al. (2016) reported that aerobic training is effective to attenuate the decrease in spontaneous physical activity, but did not prevent the decline of aerobic performance. This result was attributed to small cages of confinement of laboratory rats. However, our results showed that HIIT induces increases in aerobic performance despite negative interferences of confinement during experimental period. Furthermore, the aerobic performance was accompanied with glycogen supercompensation in gastrocnemius, liver as well as reduced corticosterone and white blood cells. Thus, the effects of overtraining were not caused by HIIT long term.

Overtraining state increases the blood stress biomarkers and reduces performance (Lehmann et al., 1992; Halson and Jeukendrup, 2004). The corticosterone is a catabolic hormone and its concentration increases after overload period, as for example, after HIIT short term. The HIIT short term may be considered a transitory stress period and an important stimulus to lead a positive adaptation in the training sequence. Billat et al. (1999) reported unaltered performance and increased noradrenaline concentration after 4 weeks of HIIT. These authors speculated that HIIT may lead an overtraining state. However, an overtraining diagnosis was unaccomplished in the present study due to similar values of performance and stress biomarkers in comparison to baseline and CT. Perhaps, a HIIT short term

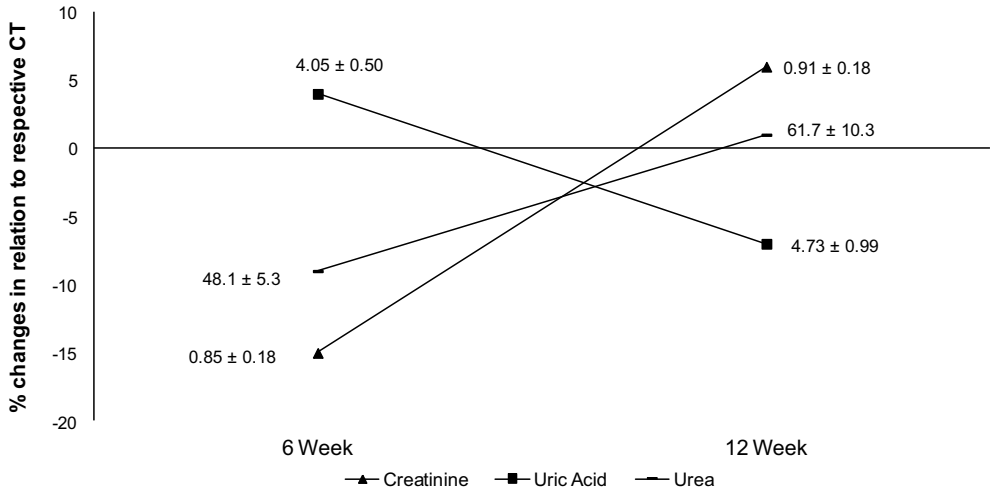

FIGURE 4 | Percentage differences in creatinine, uric acid, and urea when compared to respective CT group. Data labels are presented in mean ± standard deviation (creatinine = mg/dL; uric acid = mg/dL; urea = mg/dL).

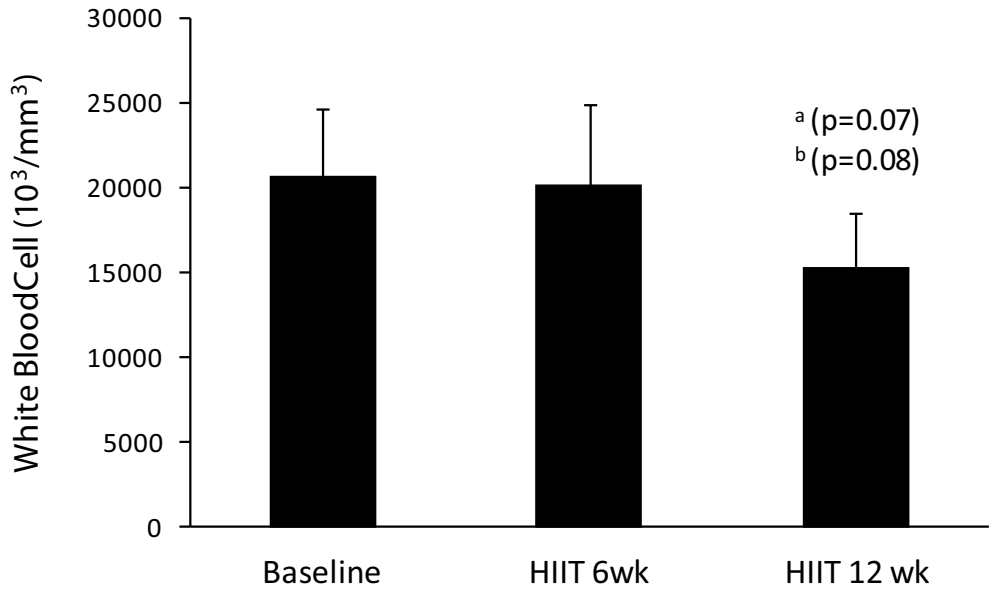

FIGURE 5 | Values of white blood cells in baseline, HIIT short term, and HIIT long term. [a]Trend to difference ($P \leq 0.07$) in relation to baseline. [b]Trend to difference ($P \leq 0.08$) in relation to 6 HIIT short term.

promotes an immediate high stress and insufficient period of positive adaptations. Thus, a long term may be more indicate to complete the organic adaptation to stress, as example, our data showed that aerobic performance enhanced after 12 weeks, but not after 6 weeks of HIIT.

Studies have shown a complex molecular interaction activated by HIIT in skeletal muscle in order to increase: angiogenesis, mitochondrial biogenesis, oxidative enzymes and other (Jensen et al., 2004; Knuttgen, 2007). Laursen (2010) showed that cellular stimulus to aerobic adaptations is predominantly dependent of AMPK-PGC1α pathway activation or CAMK- PGC1α activation, and HIIT activates more AMPK- PGC1α than CAMK- PGC1α. However, the anaerobic index not increased after HIIT short and long term. Probably, the method of anaerobic evaluation was unspecific because the animals swam continuously on the surface until exhaustion at 13% of bw, while the training sessions were performed at 50% of bw with jumps movements. This may have been a limitation of the study.

On the other hand, HIIT stimulated glycogen synthesis, but the moderate values of peak lactate may to indicate a breakdown of glycogenolysis (Vandenberghe et al., 1995; Hargreaves, 2004; Cunha et al., 2005). Possibly, with variations in overload and

series of lactate production, the exhaustion time and peak lactate would have increased. This result corroborated with Minahan et al. (2007) that not found relationship between anaerobic power and anaerobic capacity.

The glycogen synthesis in gastrocnemius increased after 6 weeks and enhanced further after 12 weeks when compared to baseline. On the other hand, our results showed that soleus muscle had a limited glycogen synthesis during HIIT and the synthesis did not exceed ∼70% of supercompensation independently of HIIT duration in relation to baseline. The feature of gastrocnemius muscle (fast-twitch fibers) was more HIIT sensible to glycogen synthesis due to higher glycogen synthase activity after exercise and capacity of glycogen repletion during the physical stress in relation to oxidative fibers (Fournier et al., 2004). These adaptations in gastrocnemius may be visualized manly after HIIT long term, indicating a higher anabolic/synthesis period in comparison to HIIT short term. Taken together, the glycogen concentration in liver after HIIT long term was higher (tendency, $p = 0.06$) than HIIT short term. The hepatic glycogen is important fuel for aerobic metabolism and aerobic performance after HIIT long term can be associated with supercompesantion in this tissue.

Creatine kinase reduced after HIIT long. Furthermore, testosterone concentration unaltered in HIIT groups in relation to CT (Halson and Jeukendrup, 2004; Brancaccio et al., 2007). The reduced physiological stress after HIIT may be observed too by urea, creatinine and uric acid concentration that did not change significantly when compared to CT group. Urea and uric acid are formed during protein catabolism and may indicate indirectly the proteolysis and stress state (Lehmann et al., 1993). The uric acid, urea, and creatinine concentration may be a supplementary analysis of the anabolic/catabolic state of training. Despite of unclear relationship of these metabolites with the training load and catabolism process, our results showed that uric acid and urea not accompanied the corticosterone variations after HIIT short and long term.

Exercise stimulates the production of reactive oxygen species (ROS) in tissues and blood due to large increases in oxygen uptake (Ji, 1993). While the ROS are formed during the physical stress, the antioxidant system improves the endogenous enzymes (Ji, 1993). Azizbeigi et al. (2013) reported that high intensity exercise strengthens the defensive system of erythrocytes against free radical damage. Our results corroborate with these authors since the sulfhydryl groups concentration indicated a reduced oxidative stress during HIIT. In association, the antioxidant enzymes unchanged significantly in relation to control, showing an insignificant disturbance of ROS.

The inflammation enhances during stress periods, as for example during high intensity periods, inappropriate recovery and overtraining state (Lehmann et al., 1992). Our data showed that white blood cells had a reduction tendency after HIIT long term in comparison to baseline and short term. This result synchronized with: (1) lower corticosterone level, (2) glycogen supercompensation and (3) higher aerobic performance after HIIT long term in relation to HIIT short term may to indicate a state of positive physiological adaptation. In literature review, Gleeson (2007) reported that several indexes of leukocyte increases in periods of intensified training and, cortisol has an immunomodulatory effect mediated by interleucin-6. Infusion of recombinant human IL-6 increases plasma cortisol (Steensberg et al., 2003; Gleeson, 2007), showing a relationship between cortisol and white blood cell. In this context, the corticosterone is a stress biomarker and its reduction associated to aerobic performance and white blood cells after HIIT long term may to indicate an anabolic period (i.e., increases in testosterone: corticorterone ratio and glycogen synthesis). Thus, the immunomodulatory effect of corticosterone reduced the white blood cell after HIIT long term. Furthermore, the reduction in white blood cell and cortiscosterone may be related with energy-rich fuel allocation, glycogen synthesis (Straub, 2014). On the other hand, the overreaching/overtraining state induces high cortisol, sympathetic activity, inflammation as well as reduction in performance.

In summary, the duration of HIIT induces different physiological adaptations and performances responses. The HIIT long term enhances the aerobic capacity and glycogen stores beyond HIIT short term without significant biomarkers stress alterations. Taken together, the corticosterone, glycogen stores, white blood cells and aerobic performance indicated a positive adaptation induced by HIIT long term. HIIT long term enables an anabolic period due to the corticosterone and white blood cells reduced in relation to HIIT short term. This anabolic state increases the glycogen synthesis and as consequence the aerobic performance, but not anaerobic performance. However, the high values of corticosterone after HIIT short term shows that 6 weeks of high intensitsy exercise induces a period of transitory stress. Furthermore, no significant interferences of HIIT duration in antioxidant system, metabolites, creatine kinase, and lactate dehydrogenase were found.

AUTHOR CONTRIBUTIONS

GD and CG participated in the elaboration of the experimental design, data collection, tabulation, discussion and writing of the manuscript. MP and IM participated in data collection, data discussion and writing of the manuscript. MD participated in the preparation of the research project and data collection.

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Acclimation Training Improves Endurance Cycling Performance in the Heat without Inducing Endotoxemia

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Purpose: While the intention of endurance athletes undertaking short term heat training protocols is to rapidly gain meaningful physical adaption prior to competition in the heat, it is currently unclear whether or not this process also presents an overt, acute challenge to the immune system. The aim of this study was therefore to examine the effects of heat training on both endurance performance and biomarkers associated with inflammatory and immune system responses.

Methods: Moderately-actively males ($n = 24$) were allocated randomly to either HOT ($n = 8$, 35°C, and 70% RH; NEUTRAL ($n = 8$, 20°C, and 45% RH); or a non-exercising control group, (CON, $n = 8$).Over the 18 day study HOT and NEUTRAL performed seven training sessions (40 min cycling at 55 of VO_2 max) and all participants completed three heat stress tests (HST) at 35°C and 70% RH. The HST protocol comprised three × sub-maximal intervals followed by a 5 km time trial on a cycle ergometer. Serum samples were collected before and after each HST and analyzed for interleukin-6, immunoglobulin M and lipopolysaccharide.

Results: Both HOT and NEUTRAL groups experienced substantial improvement to 5 km time trial performance (HOT −33 ± 20 s, $p = 0.02$, NEUTRAL −39 ± 18 s, $p = 0.01$) but only HOT were faster (−45 ± 25 s, and −12 s ± 7 s, $p = 0.01$) in HST_3 compared to baseline and HST_2. Interleukin-6 was elevated after exercise for all groups however there were no significant changes for immunoglobulin M or lipopolysaccharide.

Conclusions: Short-term heat training enhances 5 km cycling time trial performance in moderately-fit subjects by ∼6%, similar in magnitude to exercise training in neutral conditions.Three top-up training sessions yielded a further 3% improvement in performance for the HOT group. Furthermore, the heat training did not pose a substantial challenge to the immune system.

Keywords: cycling, heat acclimation, inflammation, lipopolysacharide, cytokine, endurance performance

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INTRODUCTION

Short- and medium-term heat acclimation training protocols are widely used by endurance athletes to increase both heat tolerance and subsequent competitive performances in the heat (Périard et al., 2015). Although favorable performance and physiological benefits can be realized from short term programs (≤7 days) (Garrett et al., 2011; Chalmers et al., 2014), greater benefits are likely from longer protocols (7–14 days) (Nielsen et al., 1997; Lorenzo et al., 2010; Daanen et al., 2011; Guy et al., 2015). For elite athletes, busy training, and performance schedules limit the time is available for strategies such as heat training, and addition of supplementary training sessions may sustain and/or complement the initial adaptations.

While the acute effects of short-term heat exposure on blood biomarkers associated with inflammation have been reported (Hailes et al., 2011; Gill et al., 2014), few studies have investigated the effects of longer duration heat training. The human immune system can usually deal with mild-to-moderate inflammatory responses, however, when a heat stimulus is too large, systemic inflammation can result in heat shock and potentially fatal sepsis (Bouchama et al., 2007). Athletes will generally seek a heat training stimulus that is large enough to evoke a training adaptation; however, there likely comes a point where the risk of clinical or subclinical levels of immune disturbance increases.

Exercise-induced endotoxemia is a potential risk of strenuous activity in the heat primarily attributed to translocation of lipopolysaccharide (LPS) from the gut into the circulation (Lim et al., 2009). An abundance of circulating LPS can evoke an inflammatory response, leading to heat shock, and overwhelming anti-LPS mechanisms including immunoglobulin M (IgM) (Camus et al., 1998) and cytokines operating in an anti-inflammatory role such as interleukin-6 (IL-6; Abbasi et al., 2013). Consequently, when anti-LPS mechanisms and rate of LPS clearance are inadequate to counter the heat-induced increase of LPS, endotoxemia may ensue. This outcome could potentially occur during a period of heat acclimation training if the athlete is unable to cope with the thermal loads presented. As IgM is a key antibody in neutralizing LPS (Camus et al., 1998), its concentration in circulating blood can reflect the body's response to endotoxin accumulation, and the degree of protective capacity in the event of further challenges. IgM concentration can increase substantially (∼20%) after exercise in the heat, although this elevation does not occur following 5 days of heat training (Hailes et al., 2011). Of the few studies that have investigated IL-6 as a blood biomarker during exhaustive exercise in the heat, Selkirk et al. (2008) observed a 20-fold increase in plasma concentrations following 2 h of exhaustive walking in protective clothing in very hot and humid conditions, with IL-6 inhibiting endotoxin induced increases in tumor necrosis factor alpha and cytokines. Furthermore, the neuroinflammatory response to exercise indicates that an increase in cytokine concentration such as IL-6 reaching a critical threshold, it is likely that sensations of fatigue develop to prevent traumatic injury of specific organs and other physiological systems within the body (Vargas and Marino, 2014). Therefore, athletes who undertake short or medium duration heat acclimation training programs could potentially be at increased risk of exercise-induced heat stress and immune disturbances associated with fatigue.

Recreationally-active healthy adults often participate in one-off events such as an ironman triathlon, marathon and week-long sporting events such as the Masters' Games. It appears that the threshold for the onset of exercise-induced endotoxemia is lower in untrained than trained individuals (Selkirk et al., 2008). Individuals seeking to use heat acclimation training as an additional training stimulus may choose either a short- or medium-term program, to elicit the classic thermal markers of plasma volume expansion, lower heart rate at submaximal intensities and lower end point core temperature, which collectively promote aerobic performance (Guy et al., 2015). However, addition of a heat load to training can often be very demanding, with some studies implementing challenging protocols on their participants, e.g., 90 min of cycling for 10 consecutive days (Gibson et al., 2015). It is prudent to account for both training load and accumulated inflammation from heat stress over the training period. As longer heat training sessions (>60 min) are likely fatiguing for recreationally-trained athletes, and can increase peripheral fatigue compared with shorter protocols (Wingfield et al., 2016), the addition of shorter and supplementary training sessions could yield similar benefits, but with lower overall stress.

Few studies have investigated the degree of inflammation and endotoxemia associated with short- and medium-term heat acclimation training. Therefore, the aim of this study was to investigate whether short-term heat training with the addition of supplementary sessions can improve cycling time trial (TT) performance, improve sub-maximal exercising heart rate and core temperature, and to quantify the degree of inflammation associated with heat acclimation training.

METHODS

Design

This study consisted of three groups of recreationally-active male athletes: a heat training group (HOT), a matched thermo-neutral training group (NEUTRAL), and a control (no training) group (CON), in a pre–post parallel groups design.

Participants

Twenty-four moderately trained male participants (3 ± 1 moderate-high intensity training sessions per week, duration 60 ± 15 min; mean ± SD) aged 24.5 ± 3.8 years, height 178 ± 7 cm, mass 84.6 ± 10.8 kg, body fat 17.5 ± 6.1%, and maximal oxygen uptake ($\dot{V}O_2$ max) of 45.0 ± 5.0 ml.kg.min^{-1} volunteered for the study. Prior to taking part, participants provided written informed consent in accordance with the Declaration of Helsinki and underwent a pre-screening health questionnaire including use of anti-inflammatory or immunomodulating medications (none were present). The study protocol was approved by the James Cook University Human Research Ethics Council (Approval number H5647).

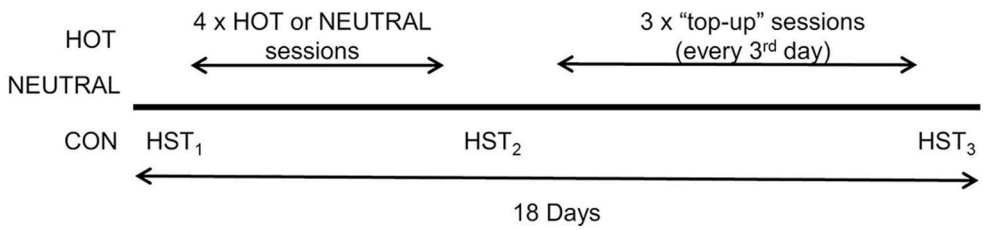

FIGURE 1 | Study timeline for Heat Training (HOT), Thermo-neutral Training (NEUTRAL), and Control (CON) groups.

Methodology

Assessment of VO_2 maxwas undertaken on a cycle ergometer (VeloTron and Velotron Coaching Software, Racermate, United States) at least 72 h before beginning the experimental trials. The intervention comprised a short-term training protocol of four training sessions on consecutive days, followed by three supplementary training sessions every 3 days. All participants completed three heat stress tests (HST_{1-3}) and seven training sessions over 18 days, with HST_1 performed as a baseline measure of heat tolerance, HST_2 completed between the end of the short-term program and before beginning the supplementary top-up training, and HST_3 completed 48 h after the final supplementary training session (**Figure 1**). Each group performed the HST in a custom-built environmental chamber at a temperature of 35°C and 70% RH. Participants in the HOT and NEUTRAL conditions completed exercise training sessions in hot and humid (35°C and 70% RH) or thermo-neutral conditions (20°C and 50% RH), respectively. Participants in the CON group did not undertake exercise training but completed the three HST's at the same intervals as HOT and NEUTRAL groups. Participants were instructed to rest and avoid moderate or high levels of physical activity on days that they were not required to attend the laboratory.

Test of Maximal Oxygen Uptake

Maximal oxygen uptake was determined by an incremental test to exhaustion on a cycle ergometer (VeloTron and Velotron Coaching Software, Racermate, United States). Briefly, the test began with participants cycling at 80–90 rpm at 120 W, with the workload increasing by 20 W every min until volitional exhaustion or when cadence was unable to be maintained above 80 rpm. Expired gases were collected via a one-way breathing system (Hans-Rudulph, United States) and analyzed by a calibrated Moxus Metabolics Measurement cart (AEI Technologies, United States). Attainment of VO_2 max was determined by the satisfaction of standard criteria (Midgley et al., 2007).

Heat Stress Test

The heat stress test was of similar design to earlier work (Garrett et al., 2009; Lorenzo et al., 2010) and comprised cycling for 3 × 10 min submaximal workloads with a 3 min rest period between workloads, followed by a 5-km self-paced TT. Following a 5 min standardized warm-up, the participants completed three 10 min workloads at 50, 60, and 70% of their peak wattage corresponding to their individualized VO_2 max. After the 70% workload was complete, a 5 min rest period was given before the start of the TT. Participants were able to view their rpm and were informed of the distance traveled every 500 m to assist with pacing. Heart rate (RS400, Polar Elektro, Finland), and core temperature (T_c) (ttec 501-3 data logger and data logger software version 10.1, Nordex Pty Ltd, Australia; MEAS 449 1RJ rectal temperature thermistor, Measurement Specialities, United States) were sampled at 5 s intervals. Fluid intake (water, *ad libitum*), rating of perceived exertion (Borg RPE 6–20, Borg, 1970) and thermal comfort (TComf) were recorded throughout the test. Nude dry body mass was recorded pre and post-exercise on a calibrated set of scales (BF-522W, Tanita, Japan) and body mass was adjusted for fluid loss and expressed as a percentage change.

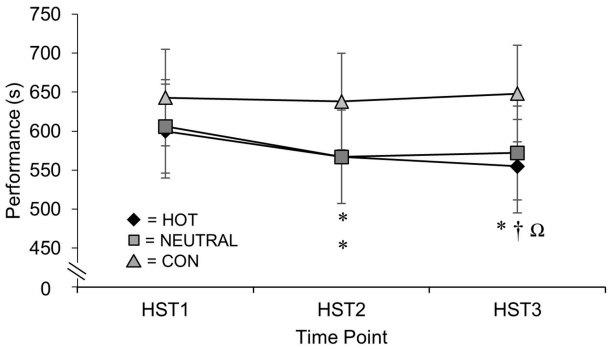

FIGURE 2 | Adjusted means ± SD of 5 km time trial performance (s) across heat stress tests (HST) 1, 2, and 3 for Heat (HOT), Thermo-neutral (NEUTRAL), and Control (CON) groups. *Faster from baseline. †Faster than HST 2. ΩHOT was faster than CON.

Blood Collection

Upon arrival at the laboratory, participants rested for 20 min before blood collection was performed. Blood was drawn in a seated position 10 min before and 10 min after each HST via a 22 g needle from a prominent superficial forearm vein located at the antecubital fossa, and drained directly into an 8.5 ml sterile serum separator Vacutainer tube containing a clot activator and gel for serum separation (Beckton and Dickson, USA). Samples were refrigerated at 4°C for 30 min to allow clotting and then centrifuged at 1000 × g at 6°C for 10 min (Rotina 420R, Hettich, Germany). Serum was removed and stored in 400 μl aliquots that were frozen immediately for

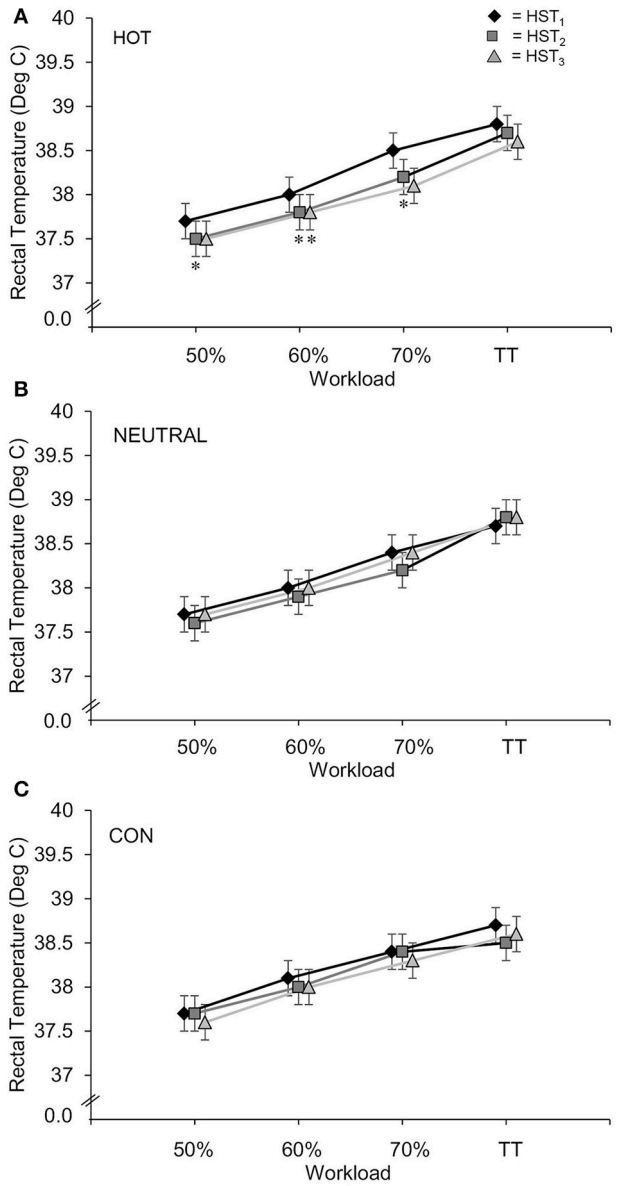

FIGURE 3 | Core temperature for Heat Training (HOT), Thermo-neutral Training (NEUTRAL), and Control (CON) groups during Heat Stress Tests (HST) 1, 2, and 3, expressed as mean ± SD. *Reduced from baseline at HST 2. †Reduced from baseline at HST 3.

a maximum of 3 months at −80°C for later analysis. Serum concentrations of IL-6 (Quantikine HS600B, R&D Systems, United States), IgM (AB137982, Abcam PLC, United Kingdom), and LPS (HIT302, Hycult, Biotechnology, Netherlands) were analyzed in duplicate by ELISA according to manufacturer's instructions.

Aerobic Interval Training

Participants in HOT and NEUTRAL undertook matched aerobic interval training on a cycle ergometer (Monark Ergomedic 828 E, Sweden) in hot and humid (35°C and 70% RH) or thermo-neutral conditions (20°C and 50% RH), respectively. The exercise-training intervention included seven training sessions comprised a standardized 3 min warm-up followed by 4 × 10 min interval at a fixed workload of 55% $\dot{V}O_2$ max. A 3 min rest period was given between each workload and water consumed *ad libitum*. A shorter duration interval-based protocol was chosen to better reflect the training status of the recreationally-trained participants; interval-based training has been shown to be beneficial for heat acclimation (Dawson et al., 1989; Kelly et al., 2016), and shorter duration training can reduce cumulative fatigue (Wingfield et al., 2016) while promoting performance (Nielsen et al., 1997). Heart rate was recorded at 5 s intervals and RPE and TComf recorded at the end of each interval. Participants self-reported symptoms of illness, inflection, soreness, or inflammation prior to the start of each training session. No symptoms of illness or infection were reported.

Statistical Analysis

Data that passed tests for homogeneity of variance were analyzed by a mixed-model analysis of variance or *t*-test (where appropriate) and significance accepted when $p \leq 0.05$. Where significant differences were indicated they were identified with the *post hoc* Tukey Test. Data is expressed as mean ± SD and change scores expressed as mean ± 90% confidence limits (CL). The baseline TT performance (s) was not normally distributed and therefore analysis of covariance was used to investigate between-group differences with participant $\dot{V}O_2$ max employed as the covariate—TT results are expressed as adjusted mean ± SD or 90% CL where appropriate. Standardized effect sizes (ES) were calculated to indicate the magnitude of change and/or difference within- and between-groups. The criteria to interpret the magnitude of ES were: <0.2 trivial, 0.2–0.6 small, 0.6–1.2 moderate, 1.2–2.0 large, and >2.0 very large (Hopkins, 2004).

Determination of biomarker concentrations and curve fit analysis was performed using GraphPad Prism Version 6.03 (GraphPad Software Inc, United States) according to the manufacturer's instructions. The manufacturer stated intra-assay precision was <10% for all assays. Statistical analyses were performed in IBM SPSS Statistics Version 22 (IBM, United States). Power analysis was conducted prior to the study and a minimum of eight participants was deemed sufficient to detect the smallest worthwhile change between means assuming the reference change in 5 km TT performance was approximately twice the magnitude of the typical error of measurement, with a Type I error of 5 and Type II error of 20%.

RESULTS

Heat Stress Test

Between Group Analyses

At HST3 a significant between-group effect for TT was evident between HOT and CON (HOT was faster by 8.2%, ±5.2%, 90% CL, $p = 0.03$). Time trial performance is presented in **Figure 2** as adjusted means from the analysis of covariance. No significant

TABLE 1 | Physiological and perceptual responses to Heat Stress Tests.

	HST_1			HST_2			HST_3		
	HOT	**NEUTRAL**	**CON**	**HOT**	**NEUTRAL**	**CON**	**HOT**	**NEUTRAL**	**CON**
$HR_{50\%}$(bpm)	139 ± 15	135 ± 12	137 ± 14	136 ± 15	133 ± 11	138 ± 13	136 ± 17	133 ± 10	133 ± 13
$HR_{60\%}$(bpm)	162 ± 15	159 ± 9	157 ± 9	155 ± 14	154 ± 9	156 ± 9	155 ± 16	154 ± 11	153 ± 11
$HR_{70\%}$(bpm)	175 ± 13	178 ± 7	170 ± 8	169 ± 13	172 ± 9	170 ± 6	168 ± 13	171 ± 9	167 ± 7
HR_{TT}(bpm)	177 ± 11	178 ± 9	169 ± 10	176 ± 9	179 ± 6	168 ± 7	179 ± 10	175 ± 10	164 ± 12
RPE_{Avg}(units)	14 ± 1	14 ± 1	15 ± 1	13 ± 2	14 ± 2	13 ± 1	13 ± 2	15 ± 3	13 ± 2
RPE_{End}(units)	17 ± 2	17 ± 2	17 ± 2	17 ± 2	18 ± 2	17 ± 3	17 ± 2	17 ± 2	16 ± 3
$TComf_{Avg}$(units)	3.0 ± 0.5	3.0 ± 0.5	3.5 ± 0.5	2.0 ± 1.0*	3.0 ± 0.5	3.0 ± 1$^{\Omega}$	2.0 ± 1.0*†	3.0 ± 0.5$^{\infty}$	3.0 ± 0.5*$^{\Omega}$
$TComf_{End}$(units)	4.0 ± 0.5	4.5 ± 0.5	4.5 ± 0.5	3.0 ± 1.0	4.5 ± 1.0$^{\infty}$	4.0 ± 1	3.0 ± 1.0*	4.0 ± 1.0	3.5 ± 1.0

*Data are expressed as mean ± SD. HOT, Heat training group; NEUTRAL, Thermo-neutral training group; CON, Control group; HR, Heart rate. Sweat loss (%) is expressed as the amount of sweat lost (kg) divided by the persons pre-exercise mass (kg) × 100. RPE_{Avg} and $TComf_{Avg}$ are the mean Rating of Perceived Exertion and Thermal Comfort rating across the entire Heat Stress Test (HST). RPE_{End}, and $TComf_{End}$ represent the values recorded at the cessation of the HST. *Significantly different from HST_1. †Significantly different from HST_2. ∞Significant difference between HOT and NEUTRAL. Ω Significant difference between HOT and CON.*

between-group effects of short-term heat training were observed for Tc (0.3 ± 0.6%, **Figure 3**), RPE, TComf, sweat loss, or HR (**Table 1**).

Within Group Analyses

Both the HOT and NEUTRAL group significantly improved TT performance in HST_2 at the end of the 7 days short-duration protocol (after four heat training sessions) compared to HST_1, with HOT 33 ± 20 s (adjusted mean ± 90% CL) faster ($p = 0.02$) and NEUTRAL 39 ± 18 s faster ($p = 0.01$) than baseline. After conclusion of the post-training top-up period, only HOT had a significant improvement in their TT performance at HST_3 compared to HST_1, completing the course 45 ± 25 s faster ($p = 0.01$) compared to their HST_1 performance. The performance of HOT in HST_3 was also significantly improved from HST_2 (12 ± 7 s, $p = 0.01$).

There was a small but significant mean reduction in exercising T_c observed in the HOT group from HST_1 to HST_2 during the 60% workload of −0.22 ± 0.14°C (mean ± 90% confidence limits, $p = 0.02$, ES = −0.53). Additionally, there was a trend for lower T_c during the 70% workload (−0.25 ± 0.21°C, $p = 0.06$, ES = −0.53) and during the TT (−0.25 ± 0.24°C, $p = 0.09$, ES = −0.45). Small-moderate significant reductions in T_c was observed in the HOT group from HST_1 to HST_3 at the 50%; −0.18 ± 0.10°C ($p = 0.016$), 60%; −0.23 ± 0.18°C ($p = 0.04$), and 70%; −0.34 ± 0.27°C ($p = 0.05$) workloads. The HOT group also experienced a small reduction in peak T_c during HST_2 compared to HST_1; −0.25 ± 0.21°C ($p = 0.057$), see **Figure 3A**. Neither the NEUTRAL nor the CON group experienced meaningful reductions in T_c in any of the HST's (**Figures 3B,C**).

The HOT group exhibited a moderate improvement in thermal comfort in HST_3 compared to HST_1 ($p \leq 0.01$). Thermal comfort was also improved in HOT during HST_2 and HST_3 compared to NEUTRAL ($p = 0.04$ and $p = 0.03$, respectively). There were no meaningful within group reductions of HR during the HST's (**Table 1**).

Inflammatory Biomarker Responses

Between-group Analyses

No significant differences between groups in any of the biomarker responses were observed either at rest or in response to any of the three HST's. Between groups there was a ∼8 ± 32% difference in post HST IL-6, ∼52 ± 111% in LPS, and ∼35 ± 36% in IgM.

Within-group Analyses

There was a large to very large (∼4 ± 2 fold) rise in serum IL-6 concentration for all groups following each HST. Serum concentrations of IgM and LPS were not substantially different following the HST for each group and there were no significant time interactions observed in any group. However, there was a trend for a small reduction in post-exercise concentrations of IgM in all participants ($n = 24$) following the first HST ($p = 0.08$, ES = 0.40). There were no within-group changes observed in serum concentration of LPS (44 ± 208%) or IgM (6 ± 61%) neither pre nor post each HST. Blood biomarker concentrations are presented in **Figure 4**.

Training Sessions

There were no within-group changes observed in exercising heart rate during each of the training sessions for the HOT or NEUTRAL groups. Although the HOT group exhibited higher HR in all training sessions compared to NEUTRAL. **Table 2** outlines the physiological and perceptual variables collected during the interval training sessions.

DISCUSSION

Short term heat training followed by supplementary top-up sessions (seven training sessions over 18 days) improved TT cycling performance, reduced exercising core temperature, and improved thermal comfort during a strenuous cycling task in the heat. In contrast, participants in the thermo-neutral (exercise) and control conditions did not experience these physiological and perceptual improvements. However,

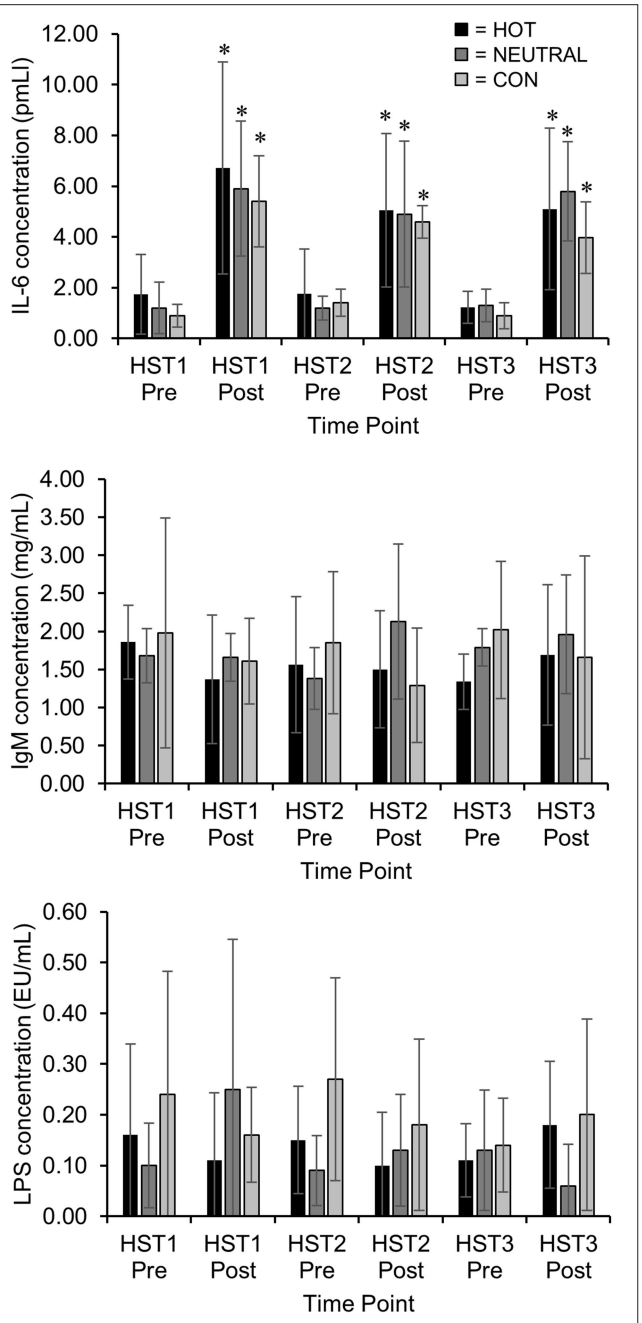

FIGURE 4 | Serum concentrations of interleukin 6 (IL-6), Immunoglobulin M (IgM), and Lipopolysaccharide pre and post Heat Stress Tests 1, 2, and 3. * Increased from pre exercise concentration.

as the thermo-neutral group also improved their 5 km TT performance after the initial short-term block of heat-training (5 training session in 7 days), it is likely a greater stimulus in terms of intensity and duration is required to elicit greater gains from heat training in shorter time periods. Although mean IL-6 concentration increased 4-fold following each HST, the exercise stimulus did not elevate other biomarkers of systemic inflammation such as IgM and LPS. As biomarker activity was largely unaffected by short-term heat training, as evidenced by IL-6 returning to basal level prior to each HST, it appears that it is possible to gain useful performance and thermoregulatory adaptations from short-duration training without compromising the immune system. Therefore, coaches and athletes can use short-term heat acclimation training coupled with supplementary heat training sessions to improve TT performance, in the confidence there is little likelihood of impairing immune system functionality.

Improvements in TT performance with short-term heat training have been reported by Lorenzo et al. (2010) in cycling and Garrett et al. (2012) in rowing. However, Garrett and colleagues did not include a control group undertaking matched training over the 5 day heat training program. It is possible that the improvement (−4 s) observed in 2000 m rowing time in that study could have been similar to that of an exercise alone control/placebo group. In our study the effects of heat training were largely similar to that of the exercise-alone group during the first week of training. However, the supplementary top-up sessions appeared to elicit further gains, indicating that while short term training offers some benefits a longer program offers additional benefits. In the study by Lorenzo and colleagues, one third of the experimental group (four out of twelve) were participants who had already completed the control condition of the experiment, consequently, the pre-exposure to exercise in the heat and heat stress test protocols. This prior exposure may have conferred a small degree of acclimation prior to taking part in the experimental portion of that study. In the present study, the inclusion of both an exercise matched (NEUTRAL) and control (CON) group allows clear interpretation of whether the heat acclimation training was responsible for the reported changes in performance and physiological adaptations. Adaptations and improvements reported previously (Lorenzo et al., 2010; Garrett et al., 2012); may relate to the increased frequency of training within a given training period. It is likely that the heat exposure resulted in ergogenic performance and thermoregulatory adaptations at the end of the 18 day period beyond that of exercise training alone.

The improved TT performance by participants in HOT was matched by those in NEUTRAL at HST_2, indicating that the stimulus for performance gain over 7-days of short-duration training in moderately-trained individuals is exercise *per se* rather than the environmental conditions under which it is performed (i.e., hot or neutral). Although, there were additional performance gains for the HOT group after the three supplementary training sessions over 10 days which increased HOT's total heat load to nine exposures (two HST's and seven training sessions, approximately 9 h). Clearly, exercise in temperate conditions results in heat production which elevates body temperature (Gleeson, 1998), and among recreationally-active participants it seems probable that this heat production is a sufficient stimulus to generate modest adaptations over 7 days. The observation of continued adaptation and performance improvement only in the HOT group after the post-training top-up period (after the full 18 days) suggests

TABLE 2 | Physiological and perceptual observations during sub-maximal aerobic interval training from training sessions one, four, and the third top up session.

	TR_1		TR_4		TU_3	
	HOT	**NEUTRAL**	**HOT**	**NEUTRAL**	**HOT**	**NEUTRAL**
HR (bpm)	161 ± 13	145 ± 9$^{\infty}$	157 ± 12	145 ± 6$^{\infty}$	154 ± 15	140 ± 13
RPE_{Avg} (units)	15 ± 1	15 ± 2	14 ± 2	15 ± 2	13 ± 3	13 ± 1†
$TComf_{Avg}$ (units)	3.0 ± 1.0	3.0 ± 1.0	3.0 ± 1.0	3.0 ± 1.0	2.0 ± 1.0	3.0 ± 1.0

Data is expressed as mean ± SD. HOT, Heat training group; NEUTRAL, Thermo-neutral training group; TR_1, Training session on day one; TU_3, Top up training session on day 15; HR, Mean heart rate across 4 × 10 min intervals. RPE_{Avg} and $TComf_{Avg}$ are the mean Rating of Perceived Exertion and Thermal Comfort rating across the training session. †Significantly different from TR_4. $^{\infty}$Significant difference between HOT and NEUTRAL.

that the generic adaptive responses experienced by NEUTRAL after 7 days had most likely run their course and plateaued. As this study recruited participants that were recreationally-active it is possible that elite endurance athletes, already well-accustomed to performing regular heat producing exercise would differentially experience greater gains compared to a matched neutral exercising group, although this remains to be determined.

Although a greater number of heat exposures (than imposed in this study) could yield more substantial physiological adaptations and performance improvements, it is also possible that this increase could trigger systemic inflammation (Lim et al., 2009). The ∼4 fold increase of IL-6 concentration in all participants after the HST may not signify heat stress *per se*, but rather the stress invoked by the exercise demand itself. IL-6 can be released into the circulation following various pathological events such as physical exercise, trauma, sepsis, and thermal injury (Natelson et al., 1996; Moldoveanu et al., 2000). There are few studies that have investigated IL-6 as a blood biomarker during exhaustive exercise in the heat, although one study reported a very large increase in IL-6 following 2 h of exhaustive walking in protective clothing at 40°C (Selkirk et al., 2008). However, a different study reported a very large increase in IL-6 following 3 h of exercise at 60–65% of VO_2 peak in typical laboratory conditions (Moldoveanu et al., 2000). Prolonged elevation of IL-6 may signify cumulative fatigue or a neuroinflammatory response (Vargas and Marino, 2014), however in the present study IL-6 returned to basal concentration prior to each HST. It appears the training load was adequate to elicit some physiological and performance benefits over the 18 day period, but not enough to elicit wider systemic or prolonged inflammation. Although IL-6 appeared to be the most sensitive blood biomarker to the exercise task, its usefulness in specifically signifying heat stress or acclimation status is limited given the non-heat specific nature of its response.

The low concentrations of LPS observed in this study indicates the participants tolerated the moderate-high heat load that was presented to them, and in doing so experienced minimal gut leakage (Pyne et al., 2014). As LPS is the primary endotoxin translocated to circulation under heat load (Yeh et al., 2013), its concentration and regulation is a primary consideration in study of responses to the heat. It appears that undertaking ∼40 min of strenuous exercise in the heat is not sufficient to evoke a systemic inflammatory response in healthy moderately active individuals. Furthermore, as IgM is a key antibody in neutralizing LPS (Camus et al., 1998), its concentration in circulating blood can reflect the body's response to endotoxin accumulation and as protection against further challenges. In this study the pre- to post-exercise change in IgM concentration in the heat was not significant, however following the first HST there was a trend ($p = 0.08$) toward reduced concentrations in all participants. It is likely that a substantial heat and/or exercise stimulus may be required for IgM concentrations to be substantially affected, although in this case it seems possible that there was some degradation of the antibody occurring. Although some between changes were observed in LPS and IgM concentrations (44 and ∼35% respectively) there was substantial uncertainty in these estimates due to high variability in the biomarker response. Only one other study has investigated the response of non-specific IgM following exercise in hot and humid conditions (Hailes et al., 2011). During that study a 20% increase of plasma IgM was reported pre- to post-exercise at day one of the heat acclimation program, this change was not present at day five, with post-exercise IgM not varying from basal levels (Hailes et al., 2011). The initial change of IgM in Hailes and colleagues' study may relate to the participants required to reach a terminal core temperature of 39.5°C, whereas in the present study core temperatures did not consistently rise to that level. Despite a substantial exercise and heat load (60 min HST), participants in the present study were able to cope with the demands of the exercise task with limited inflammation and immune disturbances.

CONCLUSIONS

Short-term heat training with the addition of supplementary top-up training sessions over 18 days enhanced time-trial performance by ∼9% in recreationally-active healthy adults, although thermo-neutral exercise training alone was a sufficient stimulus for performance gains of ∼6% over 7 days. The effects of heat training appear to become more worthwhile between 7 and 18 days. Nevertheless, training in either the heat or neutral conditions proved beneficial to performance and thermoregulatory responses compared to a control (non-exercise) condition. However, none of the experimental

groups exhibited substantial changes in LPS, IgM, or IL-6 indicating the training and heat load did not elicit an immune response. It is possible that a more intense heat training protocol may lead to greater physical and immune responses.

AUTHOR CONTRIBUTIONS

JG, DP, GD, CM, and AE contributed to the study design. JG completed data collection and conceptualization and drafting of the article. JG and KM completed Biomarker analysis. All authors performed all data analysis and conceptualizing and revising the study critically for important intellectual content, and approved the final manuscript.

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OPINION
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Effects of Neuromuscular Electrical Stimulation Training on Endurance Performance

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INTRODUCTION

Various electrical stimulation modalities are used as adjuvants to conventional training and rehabilitation programs to increase bodily function or to reduce symptoms, such as pain. One of these modalities, neuromuscular electrical stimulation (NMES), commonly refers to the transcutaneous application of electrical currents to a target muscle group with the objective to depolarize motor neurons and consequently elicit skeletal muscle contractions of substantial intensity (usually ranging from 10 to 60% of the maximal voluntary contraction). Because NMES can generate considerable muscle tension, it is frequently used as a strength training technique for healthy adults and athletes, but also as a rehabilitation tool to increase or preserve muscle function and mass in individuals with muscle weakness or patients who cannot perform voluntary contractions [e.g., patients suffering from chronic heart failure (CHF), chronic obstructive pulmonary disease (COPD), or critical illness; for reviews, see Roig and Reid, 2009; Sillen et al., 2009; Sbruzzi et al., 2010; Maddocks et al., 2011; Maffiuletti et al., 2011; Smart et al., 2013; Burke et al., 2016]. Under certain conditions, NMES training may also improve muscle oxidative capacity and result in a fast-to-slow muscle fiber type transition (Pérez et al., 2002; Gondin et al., 2011a), which could potentially enhance endurance performance. However, the relevance of such adaptations in skeletal muscle tissue for the translation to functional performance that is particularly important for sport and daily activities is not always self-evident, mainly because of the heterogeneity in study populations, NMES parameters, and outcome measures. Unfortunately, the bodies of literature that either focus on mechanistic (i.e., muscle endurance) or clinical (i.e., functional endurance) outcomes are often too disconnected. In this opinion paper, we aim to bring these bodies of literature together and discuss the impact of high- vs. low-frequency NMES training on muscle vs. functional endurance in healthy vs. clinical populations. As such, we focus on human studies that chronically applied NMES for at least 3 weeks in healthy persons and patients, and distinguish between the effectiveness of non-tetanic low-frequency NMES (that is usually administered continuously at frequencies close to 10 Hz) and tetanic high-frequency NMES (that is usually administered intermittently at frequencies close to 50 Hz) on muscle endurance and functional endurance. For clarity purposes, we refer to muscle endurance as the exercise-induced decline in voluntary or electrically-evoked force (Duchateau and Hainaut, 1988; Gondin et al., 2006) or the endurance time of a sustained single-joint contraction (Gondin et al., 2006). In contrast, we refer to functional endurance as the maximal oxygen consumption or workload (Pérez et al., 2002; Porcelli et al., 2012), the distance covered in a given time (e.g., 6-minute walk test) or the endurance time (Kim et al., 1995) for whole-body exercises such as walking and cycling. In the

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last section, we will provide some recommendations for better clinical use of NMES, and suggest potential directions for future research.

EFFECTS OF NMES TRAINING ON ENDURANCE PERFORMANCE IN HEALTHY SUBJECTS

High-frequency NMES training enhances muscle strength (for a review, see Gondin et al., 2011b), but may not affect (Duchateau and Hainaut, 1988) or even decrease muscle endurance, as demonstrated for example by a reduced ability to sustain a submaximal contraction (Gondin et al., 2006). High-frequency NMES training also appears to have a negligible influence on functional endurance, as illustrated for example by maximal oxygen consumption results (**Figure 1A**) (Pérez et al., 2002; Porcelli et al., 2012). These limited effects of high-frequency NMES training on endurance performance are somewhat surprising for different reasons. First, a single session of high-frequency NMES induces an exaggerated metabolic and cardiovascular stress when compared to torque-matched voluntary contractions (McNeil et al., 2006; Theurel et al., 2007) and high levels of muscle fatigue, mainly due to the motor unit recruitment pattern of NMES that is considerably different from voluntary contractions (for a review, see Bickel et al., 2011). Second, high-frequency NMES results in a fast-to-slow shift in fiber type distribution together with increased oxidative capacity and capillarization of the stimulated muscles (Pérez et al., 2002; Gondin et al., 2011a), i.e., adaptations that are characteristic of endurance training. However, methodological limitations pertaining to the definition and assessment of muscle or functional endurance complicate the interpretation of the data. Also, the evaluation of endurance for commonly-used muscles such as the quadriceps in normally functioning people may not be optimal as it likely suffers from ceiling effects. This suggestion is confirmed by the observation that high-frequency NMES training of abdominal muscles in healthy individuals resulted in substantial increases in abdominal strength and endurance time (Alon et al., 1987).

Data on the effects of low-frequency NMES training on muscle and functional endurance in healthy participants are relatively scarce. Three studies show improvements in functional endurance following low-frequency NMES training (e.g., improved work capacity and oxygen consumption at the anaerobic threshold), possibly mediated by adaptations in aerobic-oxidative metabolism and increased capillarization (Thériault et al., 1996; Nuhr et al., 2003; Miyamoto et al., 2016). Notwithstanding the limited data, the effects of low-frequency NMES training on functional endurance appear in general superior to those induced by high-frequency NMES training, despite the apparently similar fast-to-slow transition in fiber type distribution induced by the two NMES modalities (Thériault et al., 1996; Nuhr et al., 2003). Such divergent effectiveness of high- vs. low-frequency NMES training on endurance performance is likely caused by methodological differences between the two modalities. High-frequency NMES is usually applied intermittently and with higher current intensities in comparison with low-frequency NMES. Because of better current tolerance, low-frequency NMES sessions are generally considerably longer and can reach up to 240 min of continuous stimulation per day (e.g., Nuhr et al., 2003), vs. 20–30 min of intermittent high-frequency NMES per day (Gondin et al., 2011a). Such differences may have contributed to the greater increases in functional endurance observed after low- vs. high-frequency NMES training and strongly suggest that the long duration of low-frequency NMES sessions may be the most important parameter for increasing functional endurance.

In summary, little is known about the impact of NMES training on muscle and functional endurance in a healthy population. High-frequency NMES training has no influence or may even have a negative impact on muscle endurance while for functional endurance, low-frequency NMES training appears favorable, possibly because of the very long treatment sessions. Studies in healthy individuals that often targeted the quadriceps muscle may have suffered from ceiling effects. We speculate that NMES interventions on less-used muscle groups and with stronger study designs (large sample size, homogeneous subject characteristics, sham condition) will provide more insights into the real effectiveness of NMES training.

EFFECTS OF NMES TRAINING ON ENDURANCE PERFORMANCE IN PATIENT POPULATIONS

The suggestion that less-used muscle groups are more prone to improvement is confirmed by findings obtained in patients with pathologies affecting muscle, pulmonary, and cardiovascular function. In general, NMES training protocols are more effective in patients compared to healthy individuals, at least for functional endurance (**Figure 1A**). In various patient groups such as COPD and CHF, NMES training improved muscle strength and respiratory function, and consequently, functional endurance as reflected by increased oxygen uptake and workload, longer endurance times and farther movement distances, often indexed by the 6-minute walk test (for reviews, see Roig and Reid, 2009; Sillen et al., 2009; Sbruzzi et al., 2010; Maddocks et al., 2011; Smart et al., 2013; Burke et al., 2016). In contrast, there are few data on adaptations in muscle endurance after high- or low-frequency NMES training in patient populations. To the best of our knowledge, only three studies reported improvements in muscle endurance in different patient groups following NMES interventions (Quittan et al., 2001; Doucet and Griffin, 2013; Erickson et al., 2016). Clinical trials that applied high- and low-frequency NMES training focused mostly on functional endurance because of its clinical relevance (Vaquero et al., 1998; Bourjeily-Habr et al., 2002; Neder et al., 2002; Harris et al., 2003; Nuhr et al., 2003; Eicher et al., 2004; Banerjee et al., 2005; Deley et al., 2005; Dobsák et al., 2006; Karavidas et al., 2006; LeMaitre et al., 2006; Vivodtzev et al., 2006, 2012; Dal Corso et al., 2007).

Figure 1A, which contains data from the 14 aforementioned clinical trials, shows more favorable effects of high-frequency over low-frequency NMES training on functional endurance.

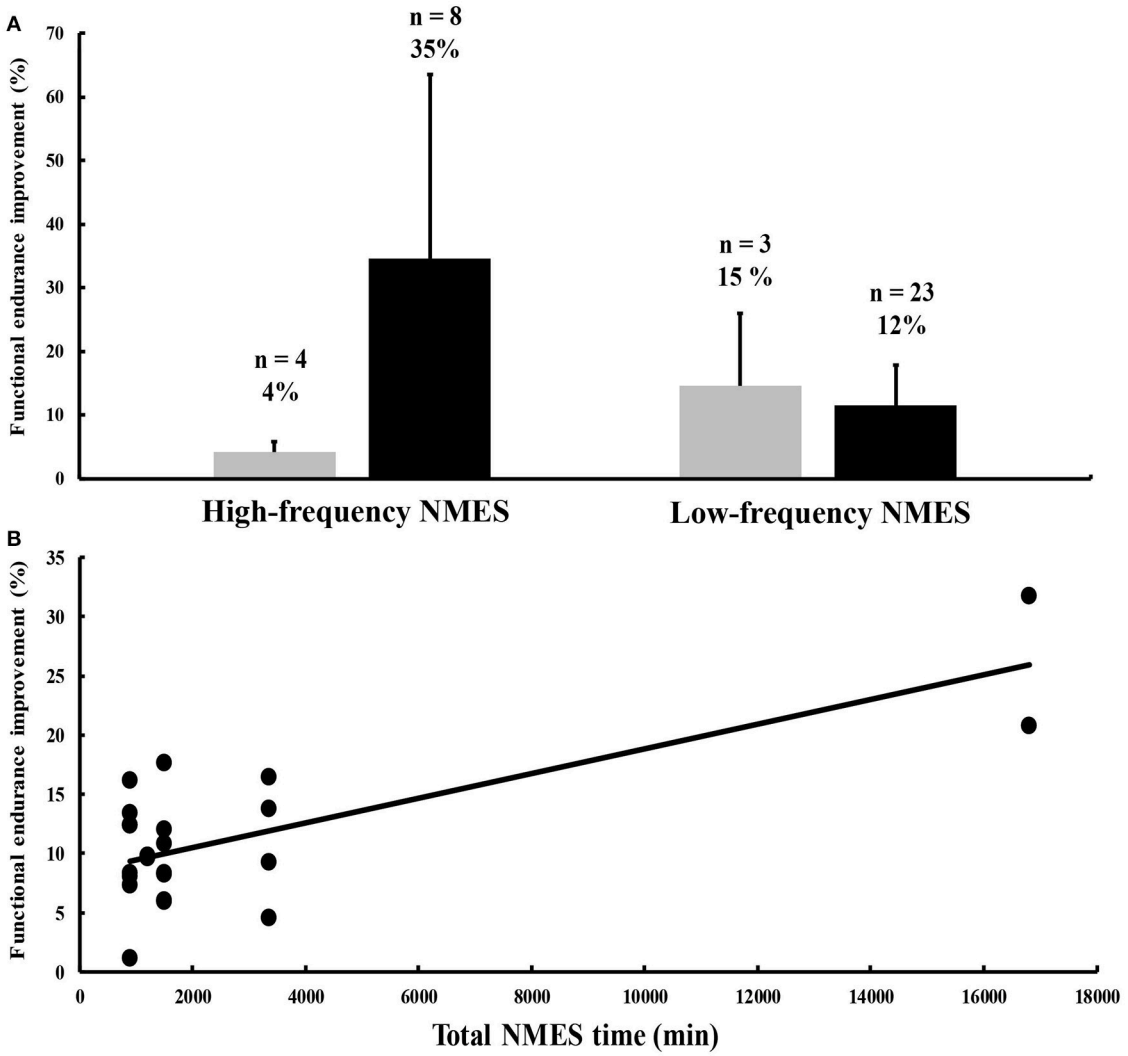

FIGURE 1 | (A) Percent improvements in functional endurance after high- and low-frequency neuromuscular electrical stimulation (NMES) interventions in healthy subjects (gray histograms) and patient populations (black histograms). The numbers above each histogram represent the number of outcome measures the mean change was computed from, and the mean percent change of these outcome measures. Most of the data, which are not stratified by condition and type of outcome measure, were extracted from Sillen et al. (2009). Vertical bars denote one standard deviation. **(B)** Relationship between the total duration of NMES treatment on the x-axis and NMES training-induced percent improvements in functional endurance in patients suffering from chronic heart failure (CHF) on the y-axis (anecdotally, the R^2 is 0.54). Data were obtained from eight different studies published between 2003 and 2006 (Harris et al., 2003; Nuhr et al., 2003; Eicher et al., 2004; Banerjee et al., 2005; Deley et al., 2005; Dobsák et al., 2006; Karavidas et al., 2006; LeMaitre et al., 2006).

This non-comprehensive analysis is confounded by the disparate definitions of functional endurance, the absence of stratification for condition, and the heterogeneity in NMES parameters. For example, for some unknown reasons, five studies in patients suffering from COPD used exclusively high-frequency NMES and showed greater increases in functional endurance (43%) compared to nine studies conducted in CHF patients that were administered almost exclusively low-frequency NMES (12%). Nevertheless, the rate of improvement may also depend on the severity of the disease. For example, patients with severe CHF improved more (26%; Nuhr et al., 2003) compared to stable patients (10%; e.g., Deley et al., 2005) following a low-frequency NMES training program. More importantly, the total duration of the NMES treatment (i.e., the training volume) from eight different studies conducted on CHF patients seems to be positively related to the magnitude of the improvement in endurance performance (**Figure 1B**).

In summary, both high- and low-frequency NMES training can increase functional endurance in patient populations, while their influence on muscle endurance is largely unknown (probably because of the poor clinical relevance of this latter variable). The impact of NMES training protocols on endurance performance appears highly dependent on the type of disease, its severity, and the total exposure to the treatment.

CONCLUSIONS

High- and low-frequency NMES training can increase functional endurance, with the magnitude of the effects being dependent on the initial condition. That is, inactive patients with advanced disease are more likely to benefit from NMES training than more active patients with stable symptoms and healthy individuals. The most likely explanation for this observation is that the quadriceps muscle, mostly targeted by NMES, is highly involved in several activities of daily living and is therefore less sensitive to improvements in active individuals. We therefore argue that NMES training is particularly useful for patients that are unable or unwilling to participate in daily activities or in regular physical exercise. Despite the heterogeneity of the studies in terms of study populations, NMES parameters, and outcome measures, we provide recommendations for clinical use and present ideas to increase treatment effectiveness. Although recent comparison studies did not show differences in the acute responses between high- and low-frequency NMES in both CHF (Sbruzzi et al., 2010) and COPD patients (Sillen et al., 2011), low-frequency NMES training seems to be particularly effective for patients with CHF, with longer treatment durations causing larger increases in functional endurance (**Figure 1B**), while for COPD patients, it is difficult to provide specific recommendations concerning high- vs. low-frequency NMES due to limited data.

Although the literature has provided us with many mechanistic and clinical insights into NMES training-induced effects on muscle and functional endurance, the trials conducted so far have some methodological limitations and need to be improved. First, there is a clear distinction between the trials in healthy participants and those in patient populations. While studies in healthy individuals focus on mechanistic parameters such as fiber type composition and capillarization and have few outcome measures that are clinically relevant, studies in patient populations merely focus on functional/clinical outcomes. While such distinctions are understandable from a patient-burden point of view, the mechanisms underlying NMES-induced adaptations may differ between diseased and healthy individuals. Therefore, randomized controlled trials are needed that compare high- and low-frequency NMES training programs for both lower and upper extremity muscle groups in patient populations vs. age- and gender-matched healthy controls. In addition, because the clinical applicability also depends on whether NMES-induced effects can still be observed after several months, follow-up measures should be included in future trials.

In conclusion, we propose here that both high- and low-frequency NMES training (and probably a combination of the two, depending on clinicians' needs) are potentially relevant to improve endurance performance, and that although their physiological effects are relatively well understood in healthy subjects, more evidence-based research is required to optimize NMES treatment protocols for various patient populations.

AUTHOR CONTRIBUTIONS

All authors listed, have made substantial, direct and intellectual contribution to the work, and approved it for publication.

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Master Athletes Are Extending the Limits of Human Endurance

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The increased participation of master athletes (i.e., >40 years old) in endurance and ultra-endurance events (>6 h duration) over the past few decades has been accompanied by an improvement in their performances at a much faster rate than their younger counterparts. Aging does however result in a decrease in overall endurance performance. Such age-related declines in performance depend upon the modes of locomotion, event duration, and gender of the participant. For example, smaller age-related declines in cycling performance than in running and swimming have been documented. The relative stability of gender differences observed across the ages suggests that the age-related declines in physiological function did not differ between males and females. Among the main physiological determinants of endurance performance, the maximal oxygen consumption (VO_{2max}) appears to be the parameter that is most altered by age. Exercise economy and the exercise intensity at which a high fraction of VO_{2max} can be sustained (i.e., lactate threshold), seem to decline to a lesser extent with advancing age. The ability to maintain a high exercise-training stimulus with advancing age is emerging as the single most important means of limiting the rate of decline in endurance performance. By constantly extending the limits of (ultra)-endurance, master athletes therefore represent an important insight into the ability of humans to maintain physical performance and physiological function with advancing age.

Keywords: aging, performance, masters, marathon, triathlon, swimming, ultra-endurance, cycling

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INCREASE IN PARTICIPATION OF MASTER ATHLETES IN (ULTRA)-ENDURANCE EVENTS

Over the past three decades, there has been a continual increase in the number of master athletes (i.e., >40 years old) in endurance and ultra-endurance (>6 h) events (Zaryski and Smith, 2005). Indeed, the percentage of male finishers older than 40 years is frequently higher than those under 40 years. At the New-York marathon, male master runners represent now more 50% of total male finishers while female master athletes represented 40% of total female finishers, respectively (Lepers and Cattagni, 2012). This finding corroborates previous observations for 100- and 161-km ultra-marathon running where master runners represent the greatest part of the finishers; up to 73% for 100-km (Hoffman et al., 2010; Knechtle et al., 2012). Similar trends have been observed for multiple discipline events such as triathlon (swimming, cycling, running). For example, master triathletes represent now more than 55% of the total

field for males and more than 45% of the total field for females at the World championship Ironman triathlon in Hawaii (Lepers et al., 2013a). The increase in participation of master athletes in endurance events inevitably has an influence on the mean age of the finishers. Generally finishers are older than 40 years in endurance and ultra-endurance events, e.g., 47 years for 100-km running (Knechtle et al., 2012), 43–44 years for 161-km running (Hoffman et al., 2010), or 43 years for ultra-cycling such as Race Across America (Shoak et al., 2013).

Different factors could explain the increase in participation of master athletes in endurance events. With the increase in life expectancy and training facilities, such as specific masters programs, the increased participation of oldest athletes (>60 years old) described may be a reflection that these athletes, who may in some cases be retired, have more available time and resources at their disposal to train and therefore to compete (Reaburn and Dascombe, 2008). However, sporting background, social category and working profession of the master athletes all remain unknown. Some may have the experience of many years of training and competition, while others only begin as they approach middle-age and beyond.

The relative increase in participation of master athletes in endurance events could also result from a decrease in participation in the younger age groups. Younger people wanting to try endurance activities may be more and more attracted to more "fun" events, such as "Obstacle course races" or "Mud runs" (http://www.outsideonline.com/1892576/american-gladiators). Indeed, an estimated 4.5 million participated in such races in the USA in 2015 alone (http://www.usatoday.com/story/sports/2015/11/02/obstacle-races-going-mainstream-more-popular-than-marathons/73743474/).

ELITE ULTRA-ENDURANCE ATHLETES GET OLDER

The current age of elite marathoners is around 30 years for both males and females (Hunter et al., 2011), but the age of peak performance in endurance events generally increases as race distance increases (Knechtle et al., 2014; Romer et al., 2014). Interestingly, data from ultra-endurance events reported that the age of the elite athletes has increased over the past few decades as have their performances. For example, at the Hawaii Ironman triathlon, since the 80's, the age of the annual top 10 finishers increased over time from 26 to 35 years for females and from 27 to 34 years for males, respectively (Gallmann et al., 2014). Anecdotally, this year (2016) the winner of the New-Zealand Ironman triathlon (i.e., Cameron Brown) was 43 years and became the oldest winner of any Ironman race. Similarly, the winner of the Austria Ironman triathlon (i.e., Marino Vanhoenacker) was 40 years old and the second placed athlete (Viktor Zyemtsev), 43 years old. Similar trends have been observed for elite ultra-marathoners who also became older over time (Hoffman and Wegelin, 2009; Eichenberger et al., 2012). During the past four decades, the age of the top 5 runners at the "Western States 161-km Endurance Run" increased from the early to the upper thirties (Hoffman and Wegelin, 2009). These data show that the upper age limit of elite ultra-endurance athletes has increased during the past decades bringing into question what the upper limit of the age of peak performance in elite ultra-endurance performance actually is.

PERFORMANCES OF MASTER ATHLETES IMPROVED AT A FASTER RATE THAN IN YOUNG ATHLETES

Age-related declines in endurance and ultra-endurance performance have been well described in the literature for swimming (Tanaka and Seals, 2003; Zamparo et al., 2012; Ferreira et al., 2016; Knechtle et al., 2016), cycling (Balmer et al., 2008; Baker and Tang, 2010; Capelli et al., 2016), running (Leyk et al., 2007; Hoffman, 2008; Hoffman et al., 2010; Knechtle et al., 2012; Lepers and Cattagni, 2012), and triathlon (Baker and Tang, 2010; Bernard et al., 2010; Lepers and Maffiuletti, 2011; Stevenson et al., 2013, Lepers et al., 2013b). The age-related decline in (ultra)-endurance performance with advancing age depends on the discipline and on gender. It ranges from 8% per decade (e.g., in 100-km male ultra-marathon runners; Knechtle et al., 2012) to 15% per decade (e.g., in Ironman female triathletes, Lepers et al., 2013a). Interestingly, the increase in participation of master athletes in ultra-endurance and endurance events over the past few decades has been accompanied by an improvement in their performance at a faster rate than young athletes. This has been observed for marathon running (Lepers and Cattagni, 2012) and also for triathlon (Lepers et al., 2013a,b; Stiefel et al., 2014). The improvement in performance of master athletes is more pronounced for the oldest age group categories (>60 years). For example, it has been shown that the best marathon male runners under 60 years did not significantly improve their performances at the New-York marathon during the 1980–2009 period, while average running time within the 70–74 years age group decreased by 7% over the same three decades (Lepers and Cattagni, 2012). Master female runners followed the same trends but the improvements occurred in all age group categories >50 years and were greater than for males. For a newest ultra-endurance sport such as Ironman triathlon that started in the early 80's (Lepers, 2008), the improvement in performance of master triathletes is much more pronounced compared to running. For example, at the Hawaii Ironman triathlon, the male 60–64 year age group triathletes improved their performance by 20% during 1986–2010 the period. The same rate of improvement was found for the best female 50–44 year age group triathletes (Lepers et al., 2013a; **Figure 1**).

The better physical condition of older athletes is likely to have increased participation, competitive spirit and performance (Maharam et al., 1999; Ransdell et al., 2009). The higher participation rates of master athletes increases the possibility of them achieving better results due to the competitive nature of the sport (Hunter and Stevens, 2013). Other reasons may explain the improved endurance performance of master athletes such as improvement of training facilities, new training methods

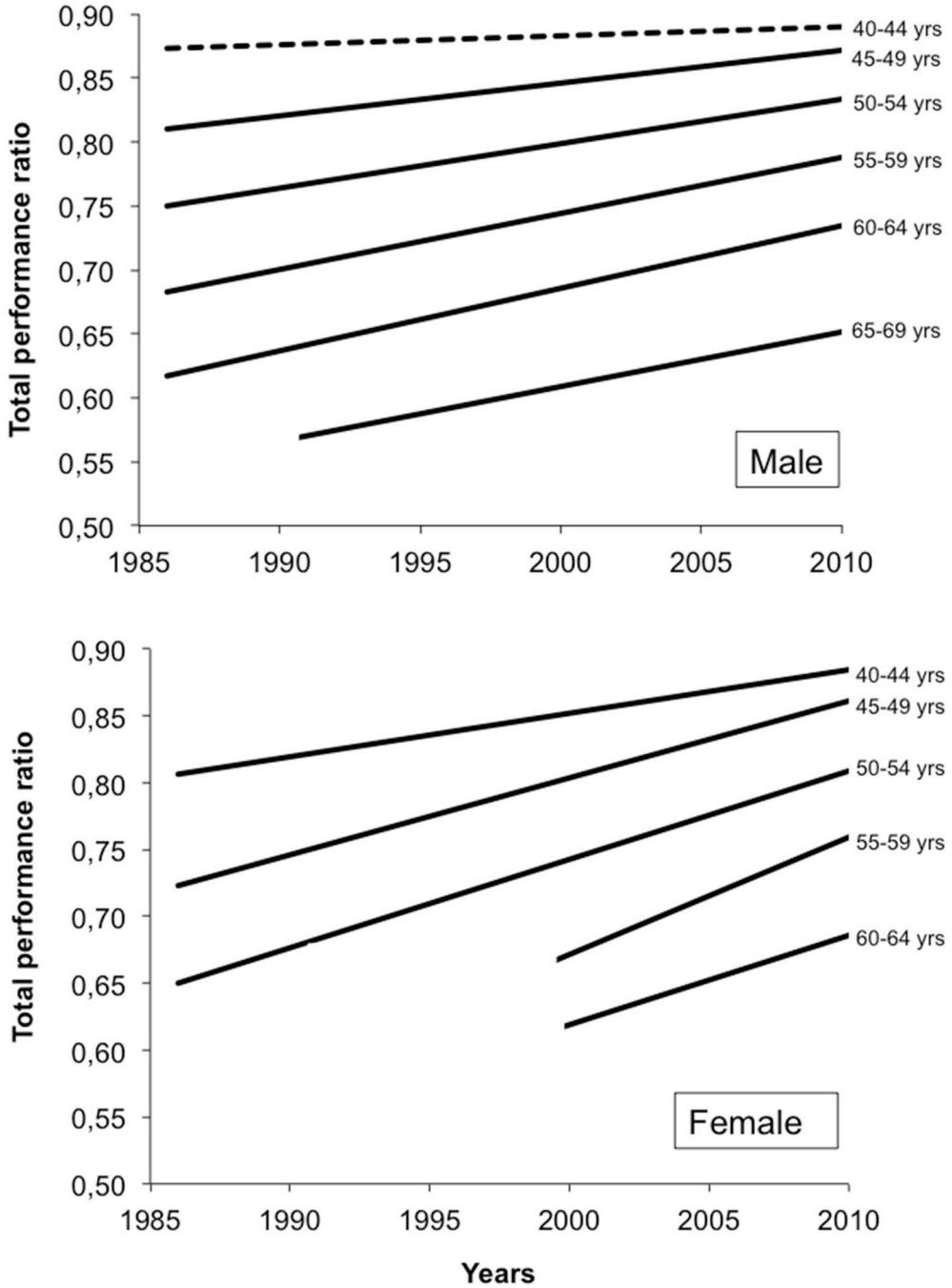

FIGURE 1 | Changes in performance ratio for total time at the Hawaii Ironman triathlon across the years for best male and female master triathletes. A performance ratio equal to 1 corresponds to the performance of the top10 elite triathletes (<40 years old). Solid lines represent conditions where the slopes of the linear regressions were significantly different ($P < 0.01$) from zero. Dashed lines indicate the slopes of the linear regressions were not significantly different from zero. Lines cross only the range of years for which data were considered.

(e.g., concurrent strength and endurance training, polarized training), master athlete coaching, nutritional strategies, and equipment (Reaburn and Dascombe, 2008; Louis et al., 2012; Piacentini et al., 2013; Brisswalter and Louis, 2014; Pugliese et al., 2015).

AGE-RELATED DECLINE OCCURS LEAST IN CYCLING

The age-related decline in endurance performance depends on the modes of locomotion. Studies on triathlon involving

running, cycling, and swimming have offered the possibility of comparing age-related declines in three disciplines for the same athlete. Interestingly, it has been observed that there was a smaller age-related decline in cycling performance than in running and swimming performances for both short- and long-distance triathlons (Bernard et al., 2010; Lepers et al., 2010, 2016; Lepers and Stapley, 2011). These findings show that age-related declines in endurance performance are specific to the mode of locomotion, although the cause for such mode-specificity is not really clear. Several hypotheses have been proposed to explain the smaller declines in cycling performance with advanced age such as difference in mechanical power between in cycling and running, a lesser reduction in lactate threshold or economy during cycling, a greater muscle fatigue during running with age or greater training volume in cycling compared to running to limit the traumatic injuries (Easthope et al., 2010; Lepers et al., 2010, 2016), but these assumptions remain speculative and require further investigations. In addition, the age-related declines in ultra-endurance performance seem also depend on exercise duration at least for cycling and running. Indeed, it has been shown that the magnitude of the declines in cycling and running performances with advancing age during short distance triathlon (3 h) are less pronounced than during long distance triathlon (10 h), while the age-related decline in swimming performance are not influenced by triathlon duration (Lepers et al., 2010).

GENDER DIFFERENCES IN PERFORMANCE HAVE INCREASED WITH AGE BUT MAY SOON ATTENUATE

Gender differences in endurance performance for elite athletes is generally close to 10% (Cheuvront et al., 2005) except for ultra-endurance swimming where females tend to reduce the gap with males (Lepers and Maffiuletti, 2011; Eichenberger et al., 2012; Knechtle et al., 2014). Men have a greater VO_{2max} than women because they have larger hearts, greater hemoglobin concentration, less body fat, and greater muscle mass per unit of body weight (Cheuvront et al., 2005). The other two primary factors that limit endurance performance at least for running including running economy and the "lactate threshold" seem not to differ between men and women (Joyner and Coyle, 2008). Gender differences in endurance performance are of biological origin and the gap between elite males and females is unlikely to narrow naturally. Several studies have shown that the gender difference in endurance performance increases with advanced age. It has been observed for running (Hunter and Stevens, 2013; Senefeld et al., 2016) and triathlon (Lepers and Maffiuletti, 2011; Lepers et al., 2013b) but is still controversial for swimming (Knechtle et al., 2016; Senefeld et al., 2016). Differences in swimming with advanced age appear to be less than in marathon running and may be related to a greater more depth in women's swimming than marathon running (Senefeld et al., 2016). The lower participation rate and less depth among women competitors in master age group categories is likely to amplify the difference between genders in endurance performance above that due to physiological differences alone (Hunter and Stevens, 2013). Environmental and social conditions and achievement motivation have played a role in limiting women's participation but it seems less and less true more recently (Deaner, 2013). For example, at the New-York marathon, differences between men and women in running times decreased for older age groups during the last three decades but since 2000 the differences have not significantly differed across age groups (Lepers and Cattagni, 2012). This relative stability of gender differences observed across the ages suggest that the age-related declines in physiological function did not differ between males and females. We suppose that in the near future, gender differences in endurance performance will be the same for all age group categories at least for the best older athletes, but this assumption will need to be verified.

AGE-RELATED CHANGES IN PHYSIOLOGICAL DETERMINANTS OF ENDURANCE EXERCISE PERFORMANCE

Previous data indicate that decrease in maximal oxygen consumption (i.e., VO_{2max}) is the predominant contributor to the decline in performance with advancing age (for details see Tanaka and Seals, 2008). Reductions in the ability to sustain a high fraction of VO_{2max}, evaluated by blood lactate threshold and submaximal exercise economy may also contribute.

VO_{2max} declines by ∼10% per decade after 30 years in healthy sedentary adults. It has been suggested that the rate of decline in VO_{2max} with age was smaller in master endurance athletes than in sedentary adults (Heath et al., 1981), however, this observation has been brought into question by more recent data. Indeed, some studies found that master endurance athletes demonstrate similar and even greater absolute rates of decline in VO_{2max} with age than healthy sedentary adults, as a result of greater baseline VO_{2max} as young adults and greater reductions in training with advancing age compared with sedentary adults (Fitzgerald et al., 1997; Eskurza et al., 2002). Trappe et al. (2003) showed that the aerobic power of octogenarian lifelong endurance athletes was approximately double that of untrained octogenarians (38 vs. 21 $ml.kg^{-1}.min^{-1}$). These remarkable aerobic capacities are the highest ever recorded in this age group. Some trained octogenarian athletes can still achieve high level of endurance performance. For example, the current marathon world record for a male octogenarian athlete was set in 2011 by a Canadian athlete (Ed Whitlock) who ran 3 h 15 min (http://www.world-masters-athletics.org/). This amazing performance corresponds to a running speed of 3.59 $m.s^{-1}$ that is only 37% lower than that of the actual absolute world record (5.72 $m.s^{-1}$, 2 h 02 min 57 s). Lepers et al. (2013c) have estimated that the VO_{2max} of Ed Whitlock was close to 50 $ml.kg^{-1}.min^{-1}$. In regards to Ed Whitlock's performance, we could expect in the future significant improvements in the performances of elderly master athletes who had high aerobic capacities when younger.

Reductions in the lactate threshold and exercise economy or locomotor efficiency in master endurance athletes may also contribute to the decrease in performance with advancing age

but to a lesser extent compared to VO_{2max}. The decrease in locomotor efficiency in master athletes remains controversial and may depend on the locomotion mode. For example, with cycling efficiency, an increase or a possible reduction (Sacchetti et al., 2010; Louis et al., 2012; Brisswalter et al., 2014) in efficiency with advanced age has been observed. The same inconsistent results have been reported concerning the energy cost of running in master athletes. For instance, Allen et al. (1985) did not observe a difference in running economy between young (25 years) and master athletes (56 years). In contrast, Sultana et al. (2012) reported a higher energy cost of running in master triathletes (≥40 years) when compared with their young counterparts. The higher energy cost of running in master athletes is associated with a lower muscle power. However, a long-lasting running exercise seems to preserve the spring-like mechanism (i.e., stiffness during running) of master athletes (Pantoja et al., 2016). The inconsistency of the results may be explained by the different age and fitness level of the tested master athletes. Interestingly, a recent study testing young (28 years) and master (60 years) triathletes showed that cycling efficiency was lower (−11%) and energy cost of running was greater (+11%) in the master compared with young triathletes (Peiffer et al., 2016); but when scaled to lean body mass, changes were more pronounced during the run (+22%). These findings suggest that at least within trained triathletes, aging can influence efficiency in both the run and cycle discipline. However, the effects of different types of training with advanced age, such as concurrent strength and endurance training (e.g., Louis et al., 2012; Piacentini et al., 2013) or polarized training (e.g., Pugliese et al., 2015) on exercise economy and more generally on endurance performance, remain to be explored.

The precise contribution of central (i.e., cardiovascular) and peripheral (i.e., oxygen extraction) factors to the reduced VO_{2max} with advancing age in master endurance athletes remains unclear (**Figure 2**). Both parameters of the Fick equation i.e., maximal cardiac output and maximal arterio-venous oxygen difference

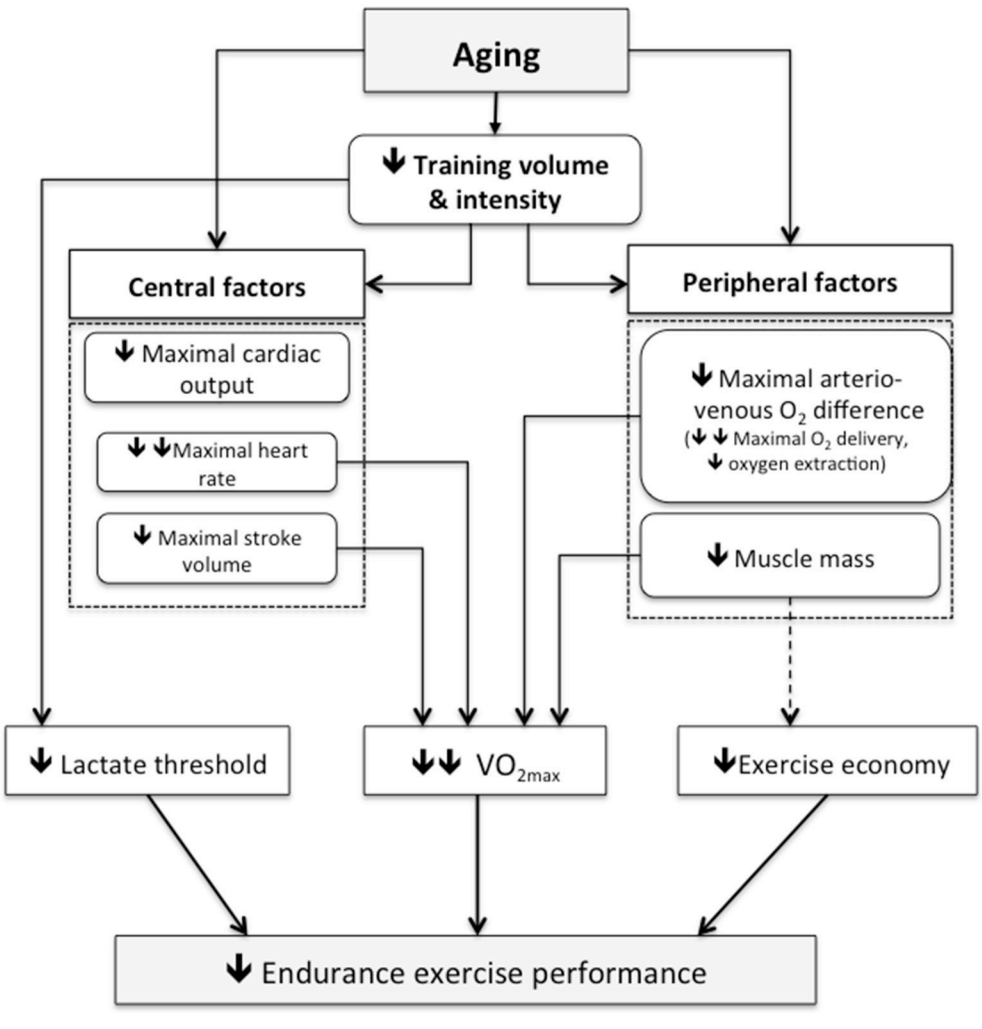

FIGURE 2 | Physiological mechanisms implicated in the age-related decline in endurance exercise performance. A double arrow expresses a main influence.

have been found to decrease in master athletes (Tanaka and Seals, 2008). The reduction in maximal cardiac output in master endurance athletes results from a reduction in both maximal heart rate and maximal stroke (Rivera et al., 1989; Ogawa et al., 1992). However, maximal heart rate seems to be the predominant mechanism mediating the reduction in maximal cardiac output with age. Interestingly, it seems that the rate of maximal heart rate decrease ($\sim$0.7 beat.min^{-1}.year^{-1}) with advancing age is similar between healthy sedentary, recreationally active and master athletes (Tanaka et al., 2001). At a peripheral level, maximal arterio-venous oxygen difference, which reflects in general the capacity of active skeletal muscles to extract and consume oxygen, has been found to decline modestly with age in master endurance athletes (Rivera et al., 1989). Moreover, it seems that maximal oxygen delivery, rather than oxygen extraction, is the major contributor to age-related decline in maximal arterio-venous oxygen difference in master endurance athletes (Tanaka and Seals, 2008). A decrease in maximal arterio-venous O_2 difference may also be secondary to an age-related decline in of muscle mass, however chronic intense endurance exercise preserves muscle mass. For example, Wroblewski et al. (2011) showed that despite an increase in total body fat percentage with age, mid-thigh muscle area, quadriceps area, and specific strength did not decline significantly with age in master athletes. Dubé et al. (2016) recently showed that lifelong exercise was associated with higher intramyocellular triglyceride and glycogen content in all muscle fibers and higher metabolic efficiency during exercise. From a neuromuscular viewpoint, Power et al. (2010) also showed master runners preserved the number of functioning motor units in the tibialis anterior well into the seventh decade of life providing evidence that chronic activity has beneficial effects not only on the muscle fibers but also on the motoneuron. This same group of research also recently showed that world champion master athletes in their 9th decade of life had a greater number of surviving motor units, better neuromuscular transmission stability and a greater amount of excitable muscle mass as compared to age-matched controls (Power et al., 2016).

The inevitable age-related decline in endurance exercise performance of endurance master athletes is closely related with reductions in exercise training volume and intensity (Tanaka et al., 1997; Eskurza et al., 2002). However, the changes in the physical (e.g., prevalence of injuries, distribution of relative times spent at the different training intensities) and behavioral (e.g., reductions in time and motivation to train) characteristics of training of the endurance master athletes with age remain poorly described in the literature.

PERSPECTIVES FOR FUTURE STUDIES

Endurance exercise performance of master athletes continues to improve each year. In a descriptive point of view, we now need a model to compare the performance master athlete of different ages (Vanderburgh, 2015). We also lack information regarding training characteristics, nutrition habits, and recovery capacities of master endurance athletes (Louis et al., 2010; Brisswalter and Louis, 2014; Borges et al., 2016; Piacentini et al., 2016). Several other questions need to be addressed in the future, such as: Is endurance competition a greater psycho-physiological stressor for master athletes compared to young athletes (Piacentini et al., 2015)? Does response to high intensity interval training differ between young and old athletes? Is fatigue by induced prolonged endurance exercise amplified with advancing age? Are the benefits from chronic exercise observed in master athletes achievable in lifelong sedentary adults who begin exercising after age 40? We also need to identify the concomitant roles genetics and training in performance of master endurance athletes. Master athletes require the continued attention of sport scientists and exercise physiologists to extend their limits of (ultra)-endurance.

AUTHOR CONTRIBUTIONS

All of the listed authors contributed to writing the manuscript (RL and PS), suggesting improvements to, and reviewing the manuscript (RL and PS). All the listed authors approved the final version of the manuscript.

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Passion and Pacing in Endurance Performance

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Endurance sports are booming, with sports passionates of varying skills and expertise battering city streets and back roads on their weekly or daily exercise rounds. The investments required for performing in endurance exercise are nevertheless considerable, and passion for their sport might explain the efforts endurance athletes are willing to make. Passion may be defined as a strong motivational force and as such might be related to the neurophysiological basis underlying the drive to exercise. A complex relationship between the brain and other systems is responsible for athletes' exercise behavior and thus performance in sports. We anticipate important consequences of athletes' short term choices, for example concerning risk taking actions, on long term outcomes, such as injuries, overtraining and burnout. We propose to consider athletes' type of passion, in combination with neurophysiological parameters, as an explanatory factor inunderstanding the apparent disparity in the regulation of exercise intensity during endurance sports. Previous research has demonstrated that athletes can be passionate toward their sport in either a harmonious or an obsessive way. Although both lead to considerable investments and therefore often to successful performances, obsessive passion may affect athlete well-being and performance on the long run, due to the corresponding inflexible exercise behavior. In this perspective we will thus examine the influence of passion in sport on athletes' short term and long term decision-making and exercise behavior, in particular related to the regulation of exercise intensity, and discuss the expected long term effects of both types of passion for sport.

Keywords: regulation of exercise intensity, exercise behavior, psychobiology, overtraining, burnout

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INTRODUCTION

Elite athletes continuously train and compete at the limits of their physiological and psychological capacities, and their training and competitive schedules are more demanding than ever (Schwellnus et al., 2016; Soligard et al., 2016). Passion for their sport might be the motivational force or "drive" providing these athletes with the required energy and determination to exercise, and might explain their willingness to endure discomfort (Curran et al., 2015) and even pain (Mauger, 2014), for the sake of sports performance (Vallerand et al., 2007). Pushing themselves to continuously sustain maximal effort over a long period of time might nevertheless entail several risks for passionate athletes, such as overtraining and injuries (Curran et al., 2015; Schwellnus et al., 2016; Soligard et al., 2016). We therefore propose that studying the nature of passion for sports might shed a light on athletes' exercise behavior (Vallerand, 2012) and subsequent risks.

In cyclic middle and long-distance exercise, athletes need to adequately estimate their physiological capacity and regulate exercise accordingly during training and races, in order to perform optimally (Foster et al., 2003; Abbiss and Laursen, 2008). As it represents a strong motivational force (Vallerand et al., 2008; Curran et al., 2015), passion might be a key factor in explaining their subsequent exercise behavior, both within a race and throughout the competitive season, derived from complex interactions between factors either promoting or reducing their drive to exercise (McCormick et al., 2015).

In this perspective, we will address athletes' exercise behavior and pacing by examining the impact of passion on the regulation of exercise intensity and exercise behavior from a psychological and a physiological angle. Adopting a psychophysiological approach to study the drive to exercise might elucidate athletes' pacing both on the short (during a single exercise bout or race) and long (a season or even an athletes' career) term and allow the examination of subsequent outcomes, such as athlete performance and well-being (McCormick et al., 2015).

PASSION IN SPORTS ACCORDING TO PSYCHOLOGY

In the field of psychology, varying definitions of motivation have been proposed, most of which consider motivation as a combination of internal and/or external stimuli, pushing people to act or to react (Vallerand, 2012). In sports, motivation is used to describe the initiation, direction, intensity, and persistence of behavior (Vallerand, 2012), in short; the "drive" to exercise. Passion is defined as a "particularly strong motivation toward a self-defining activity," and thus might be a useful construct to understand athletes' "drive" to exercise (Vallerand, 2008; Curran et al., 2015). As this drive seems imperative to sustain effort during endurance sports, notably at the elite level (Curran et al., 2015), understanding athletes' passion for sport might thus elucidate athletes' willingness to engage in demanding endurance exercise (Vallerand et al., 2003). The Dualistic Model of Passion proposed by Vallerand introduces passion as a key element in the long-term outcomes of engagement in activities (Vallerand, 2012; Bridekirk et al., 2016). By considering two distinct types of passion, namely harmonious and obsessive passion, this model explains apparent disparity in athletes' exercise behavior (Vallerand, 2012; Curran et al., 2015).

For athletes to develop a passion for their sport, they must highly value their activity, which distinguishes passion from both intrinsic and extrinsic motivation which can exist in different degrees from "not at all motivated" to "highly motivated" (Mageau et al., 2009). In accordance with Self-Determination Theory, different reasons for engaging in an activity might lead to different outcomes resulting from the two different types of passion (Ryan and Deci, 2000; Vallerand, 2012). Autonomous reasons, such as the inherent pleasure an activity brings, are known to lead to intrinsic motivation (Ryan and Deci, 2000). If the given activity becomes part of athlete's identity, a harmonious passion can result (Mageau and Vallerand, 2007). When athletes feel obliged to practice, or if the activity serves important compensatory or external functions, this will lead to a more extrinsic form of motivation, and, in time, can result in an obsessive passion (Mageau and Vallerand, 2007).

Passion and its resulting strong motivation are an essential prerequisite to attain and maintain the highest level in elite sport (Vallerand, 2012; Curran et al., 2015). A harmonious passion results from engaging repeatedly in an activity that provides athletes with positive emotional experiences, such as joy, feelings of mastery and high levels of concentration (Mageau et al., 2009; Bridekirk et al., 2016). Harmoniously passionate athletes feel free and autonomous in their choice to engage in their activity, and can maintain a healthy balance between their sport and other important life domains (Vallerand et al., 2008). Those with an obsessive passion for their sport perceive an unstoppable urge to practice, for example because their self-esteem relies on their athletic performance (Vallerand et al., 2007; Bridekirk et al., 2016).

Obsessive athletes feel they have no choice but to invest maximally in their activity: their passion has come to control their exercise behavior (Vallerand, 2012), which may lead to negative emotions (Curran et al., 2015; Bridekirk et al., 2016).

The nature of athletes' passion therefore influences decision making before and during exercise as well as throughout the competitive season (Mageau and Vallerand, 2007). Hence, understanding athletes' passion might (partially) elucidate their exercise behavior and the resulting benefits and drawbacks throughout the competitive season (Mageau and Vallerand, 2007; Vallerand, 2012).

PASSION IN SPORTS ACCORDING TO PHYSIOLOGY: A NEUROPHYSIOLOGICAL PERSPECTIVE UNDERLYING THE DRIVE TO EXERCISE

The nature of athletes' passion, and thus of their drive to exercise, might represent one of the many factors influencing exercise behavior. In past research, the regulation of this drive to sustain exercise has been examined from a neurophysiological angle (Meeusen et al., 2006; Roelands et al., 2013). It has been found that neurotransmitters, such as dopamine, noradrenalin and serotonin, can maintain or decrease arousal and motivation to continue physical effort (Meeusen et al., 2006). Modifications in the release and re-uptake of neurotransmitters can influence the regulation of athletes' power output and central fatigue and hence influence exercise performance (Roelands et al., 2013).

Dopamine for example is known to play an important role in motivation, reward, attention, addiction, control of voluntary movement and locomotion (Meeusen et al., 2006). An increase in dopamine was found to enhance motivation toward rewarding goals in prolonged exercise, resulting in improved performance (Roelands et al., 2008a). Noradrenaline on the other hand is associated with the regulation of attention, arousal, anxiety, pain, mood, and depression (Meeusen et al., 2006). Increasing its concentrations resulted in decreased time trial performance (Roelands et al., 2008b). Although no effects of serotonin level manipulations on performance could be demonstrated, high

serotonin levels appear to inhibit athletes' ability and motivation to perform an end sprint during exercise (Roelands et al., 2009).

In short, manipulations of brain dopamine or noradrenaline concentrations may delay fatigue, or on the contrary deteriorate performance in the heat (Roelands et al., 2015). Neurotransmitters thus might be considered as indispensable mediators between the drive to exercise and actual behavior, leading to adjustments in energy expenditure and effort exerted (Roelands et al., 2013).

REGULATION OF EXERCISE INTENSITY AND ENDURANCE PERFORMANCE

The goal-directed regulation of exercise intensity over an exercise bout in which athletes need to decide how and when to invest their energy has been defined as pacing (Smits et al., 2014) and its outcome can be measured as athletes' power output or velocity profile during exercise (de Koning et al., 1999; Foster et al., 2003). In order to perform optimally, athletes must choose the best pacing strategy for a given situation, based on their physiological and psychological capabilities (Baron et al., 2009). Fatigue, ambient conditions and manipulations of brain neurotransmitters clearly act on athletes' regulatory mechanisms and hence affect their exercise behavior (Roelands et al., 2013). The relationship between brain neurotransmitters and athletes' pacing behaviors seems to be mediated by the subjective interpretation of their physical state, also known as their rate of perceived exertion (RPE) (Borg, 1982; Marcora and Staiano, 2010). Interestingly, psychological factors implicated in athletes' endurance performance are thought to act through these same mechanisms. Both physiological and psychological factors act on motivation to continue or perception of effort and thus are important in the regulation of exercise intensity (Abbiss and Laursen, 2008).

In understanding exercise regulation, most recent theories share the consideration of an interaction between the physical work realized and the cognitive and psychological interpretation of the sensations this physical work causes (Abbiss et al., 2015). Millet, for example, propose the flush-model, which provides useful insight on how environmental conditions, such as sleep deprivation or nutritional strategies, may affect ultra-endurance performance (Millet, 2011). This holistic model incorporates ratings of perceived fatigue as a crucial element of the regulation of exercise intensity and provides insight in the complexity of exercise regulation (Millet, 2011).

Fatigue during exercise has been described as being composed of both a neurophysiological and a subjective component (Pereira et al., 2014; Enoka and Duchateau, 2016). Cognitive and psychological factors thus are considered to play a key role in the perception of fatigue, for example in the psychobiological model. (Marcora and Staiano, 2010; Noakes, 2012). It seemshat perception of effort or fatigue can be regarded as a source of information, used for regulating exercise intensity (Marcora and Staiano, 2010; Noakes, 2012). Maladaptive pacing strategies therefore may result from a misinterpretation of RPE or, for example, concentrating on the wrong stimuli. Due to the subjective element in the perception of effort and fatigue, athletes are at risk of either investing too much or insufficient energy during their race leading to submaximal performance (Foster et al., 1993; Baron et al., 2009). An optimal pacing strategy requires the continuous selection of the appropriate amount of effort to exert at any given moment (Baron et al., 2009), hence decision-making aspects are crucial in endurance exercise performance (Renfree et al., 2014; Smits et al., 2014; Micklewright et al., 2016). Recently, novel theoretical frameworks have been developed in order to explore decision-making and pacing.

Human-environment interactions seem crucial in understanding the regulation of exercise intensity in competitive situations. Internal factors, such as fatigue, and external factors, such as the behavior of an opponent, have indeed been shown to influence the drive to exercise, pacing and performance (Smits et al., 2014; Konings et al., 2016b). Athletes seem to adapt their behavior to the actions of their opponents, for providing athletes with an opponent or feedback has been shown to impact on their power output, in middle-distance and endurance exercise (Noorbergen et al., 2015; Konings et al., 2016a,b; Smits et al., 2016). Between competing athletes of comparable physical capacities, the drive to exercise and its resulting decision making might thus represent the difference between victory and defeat (McCormick et al., 2015).

Interestingly, the observed effects of several external manipulations (Konings et al., 2016b) have been similar to those of the previously described pharmacological (and thus internal) interventions (e.g., Meeusen et al., 2006; Roelands et al., 2013). This drive to exercise and its magnitude can thus be influenced by manipulation of various internal and external factors. One of these factors could be the nature and magnitude of an athlete's passion (Mageau and Vallerand, 2007; Vallerand, 2012), influencing pacing behavior during exercise for example by modifying the perception and prioritizing of different stimuli (Curran et al., 2015; McCormick et al., 2015; Bridekirk et al., 2016).

PASSION AND PACING

Passion for sport is a strong motivational force, encouraging athletes to engage in strenuous exercise and pushing them to realize the countless training sessions and competitions needed to reach the top. Hence it might aid in enhancing athletic performance (Vallerand, 2012; McCormick et al., 2015).

Harmonious passion indeed leads to a strong drive to exercise, compatible with enhanced concentration, feelings of mastery and positive emotions during a race (Mageau and Vallerand, 2007; Vallerand, 2012). These positive emotions have been linked to the development of optimal pacing behavior in sports, as athletes can use and manage their emotions to guide them toward optimal performance (Baron et al., 2009), and to weigh their options wisely (Vallerand et al., 2008; Curran et al., 2015). Harmonious passion also protects against negative distraction, unrealistic goals and stress during exercise (Vallerand et al., 2007; Curran et al., 2015), and helps athletes to prioritize long term benefits over short term profits (Rip et al., 2006; Vallerand, 2012; Curran

et al., 2015). As such, harmonious passion is believed crucial for attaining high levels of performance both on the short and the long term (Vallerand, 2012).

Although obsessive passion intuitively seems beneficial, it mostly leads to negative outcomes, both on the short as on the long term (Mageau and Vallerand, 2007; Curran et al., 2015). Due to its compulsive nature, this type of passion is related to negative affect, stress, anxiety and feelings of guilt during exercise (Vallerand, 2012; Curran et al., 2015; Bridekirk et al., 2016).

During a race, dealing with both negative emotions and the task at hand is mentally and emotionally exhausting, so obsessive passion decreases athletes' ability to concentrate and attain a state of "flow" (Bridekirk et al., 2016). Moreover, it hampers endurance performance by causing augmented feelings of exertion or erroneous interpretations of the athlete's capacities (Bridekirk et al., 2016). A strong obsession with their activity may thus render athletes incapable of regulating their efforts wisely (Curran et al., 2015; Bridekirk et al., 2016) and may result in risky, all-out pacing strategies and in the inability to accept defeat (Curran et al., 2015; Bridekirk et al., 2016). Obsessive athletes also might intentionally disregard the limits of their physical and mental capacities for the sake of a short term victory (Curran et al., 2015), hence disregarding rational and "safe" decisions that might prevent accidents, overtraining and overuse injuries (Rip et al., 2006).

Passion of an obsessive nature is therefore expected to prove particularly disadvantageous, considering the complex task of managing training and competition loads throughout a season (Bridekirk et al., 2016; Schwellnus et al., 2016; Soligard et al., 2016). This inadequate decision-making regarding exercise behavior might eventually lead to overuse injuries, overtraining syndrome and burnout in obsessive athletes (Vallerand, 2012; Curran et al., 2015).

FURTHER PRACTICAL IMPLICATIONS IN SPORTS: TRAINING ADHERENCE, BURNOUT, AND OVERTRAINING SYNDROME

The limiting factor for reaching and maintaining elite level in sports nowadays seems to be an athlete's capability to remain injury-free and to endure high training loads for a long period of time (Schwellnus et al., 2016). Current demands in elite sports enhance athletes' risk for insufficient physical and mental recovery (Meeusen et al., 2013). This may lead to athletes experiencing burnout or overtraining syndrome, resulting in physical and mental exhaustion and a considerably decreased drive to exercise (Curran et al., 2015). Several biological, neurochemical, and hormonal regulation mechanisms associated with the development of these symptoms have been identified (Meeusen et al., 2013). A major risk factor for injuries, overtraining and illness might be inadequate load management throughout the season (Schwellnus et al., 2016; Soligard et al., 2016), which in turn has been suggested to be affected by factors of various nature, such as fatigue, psychology, and metabolic and hormonal factors (Schwellnus et al., 2016; Soligard et al., 2016). Symptoms of athlete burnout for example include both emotional and physical exhaustion (Rip et al., 2006). Based on the above, we propose that athletes' passion and drive to exercise might lead to either adequate or inadequate pacing behavior and the subsequent effects on long term outcomes during an athlete's career. It was indeed demonstrated that measuring physical training load does not suffice in predicting or preventing long term injuries and neither do neurophysiological parameters alone (Meeusen et al., 2013; Schwellnus et al., 2016; Soligard et al., 2016).

Overtraining and burnout may be caused by an interaction of both physical and psychological factors. Indeed, modifications of neurotransmitters in the brain lead to an alteration in pacing and willingness to exercise but also result in both the physical and emotional exhaustion characteristic for athlete burnout (Roelands et al., 2013; Curran et al., 2015). In addition to these physiological parameters, psychological aspects, such as mood states, already have been identified as possible markers of overtraining and athlete burnout (Soligard et al., 2016). Indeed, perceived motivational climate as well as athletes' commitment and coping with adversity might increase their vulnerability to detrimental long term outcomes (McCormick et al., 2015; Schwellnus et al., 2016; Soligard et al., 2016). These factors interestingly all are closely related to the nature of athletes' passion for and drive to exercise (Vallerand, 2012; Curran et al., 2015; Bridekirk et al., 2016).

CONCLUSION

Elite endurance athletes must display a considerable drive and thus a great passion for their sport in order to perform at elite level (Vallerand, 2012). Neurotransmitters have been identified as indispensable mediators between this drive to exercise and athletes' actual behavior. They inform athletes about their physical limits and the importance of continuing their effort, leading to adjustments in pacing strategies. In addition, external stimuli, such as opponents, could evoke similar responses by increasing the drive to exercise, hence affecting athletic decision-making. Passion for sport may come with a price for athletes obsessive about their sport.

We propose that the nature of elite athletes' passion for sport may provide useful insight in their decision making during exercise. It might affect pacing during a single exercise bout, but also influence exercise behavior during one or several years. Taking into account athletes' passion could therefore be a useful tool for adequate coaching and monitoring of athlete well-being. Although some athletes may be admirably capable of fitting their sport and high training loads into their ever-busy lives, obsessive athletes are expected to be extremely vulnerable to risks of injuries including overuse injuries, overtraining, burnout and drop out (Vallerand, 2012; Curran et al., 2015; Soligard et al., 2016).

AUTHOR CONTRIBUTIONS

LS and FH contributed to conception and design of the work, drafted it and revised it critically for important intellectual content. Both authors have approved the final version of the manuscript, agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

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The Influence of Mid-Event Deception on Psychophysiological Status and Pacing Can Persist across Consecutive Disciplines and Enhance Self-paced Multi-modal Endurance Performance

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Purpose: To examine the effects of deceptively aggressive bike pacing on performance, pacing, and associated physiological and perceptual responses during simulated sprint-distance triathlon.

Methods: Ten non-elite, competitive male triathletes completed three simulated sprint-distance triathlons (0.75 km swim, 500 kJ bike, 5 km run), the first of which established personal best "baseline" performance (BL). During the remaining two trials athletes maintained a cycling power output 5% greater than BL, before completing the run as quickly as possible. However, participants were informed of this aggressive cycling strategy before and during only one of the two trials (HON). Prior to the alternate trial (DEC), participants were misinformed that cycling power output would equal that of BL, with on-screen feedback manipulated to reinforce this deception.

Results: Compared to BL, a significantly faster run performance was observed following DEC cycling ($p < 0.05$) but not following HON cycling (1348 ± 140 vs. 1333 ± 129 s and 1350 ± 135 s, for BL, DEC, and HON, respectively). As such, magnitude-based inferences suggest HON running was more *likely* to be slower, than faster, compared to BL, and that DEC running was *probably* faster than both BL and HON. Despite a trend for overall triathlon performance to be quicker during DEC (4339 ± 395 s) compared to HON (4356 ± 384 s), the only significant and *almost certainly* meaningful differences were between each of these trials and BL (4465 ± 420 s; $p < 0.05$). Generally, physiological and perceptual strain increased with higher cycling intensities, with little, if any, substantial difference in physiological and perceptual response during each triathlon run.

Conclusions: The present study is the first to show that mid-event pace deception can have a practically meaningful effect on multi-modal endurance performance, though the relative importance of different psychophysiological and emotional responses remains unclear. Whilst our findings support the view that some form of anticipatory "template"

may be used by athletes to interpret levels of psychophysiological and emotional strain, and regulate exercise intensity accordingly, they would also suggest that individual constructs such as RPE and affect may be more loosely tied with pacing than previously suggested.

Keywords: deception, triathlon, multisport, pacing, rating of perceived exertion, affect, rating of perceived effort, teleoanticipation

INTRODUCTION

During sprint-distance triathlon, an athlete's overall finishing time comprises a 0.75 km swim, 20 km cycle, and 5 km run, each of which is separated by only a brief period of "transition". Each discipline imposes unique residual demands on the next (Peeling and Landers, 2009) and differs in its contribution to total time (swim ∼17%, cycle ∼51%, run ∼27%; Taylor et al., 2012; Taylor and Smith, 2013). An optimum pacing strategy during triathlon therefore needs to balance the relative intensity within each discipline against the benefits or consequences of these intensities in relation to overall finishing time and/or position (Edwards and Polman, 2013). Indeed, completing the swim at the highest sustainable pace (i.e., 100% of isolated time-trial pace) has been shown to significantly impair overall short-distance triathlon performance time (∼1 min 45 s), compared to swimming at 80–85% of isolated time-trial intensity (Peeling et al., 2005). Thus, it would seem that maintaining a reserve capacity throughout the swim is essential if overall triathlon performance is to be optimized. Conversely, Suriano and Bishop (2010) have demonstrated that aggressive pacing of the cycle section (i.e., equivalent to mean power output during an isolated time-trial) significantly impairs subsequent running speed but enhances total cycle-run time over the sprint-distance format. Although this study failed to include an initial swimming leg, the findings appear to support the view that cycling at the highest sustainable intensity may be the best strategy to optimize overall performance during short-distance triathlon events.

Despite these points, it is not yet clear how expectations, beliefs and perceptions might influence the pursuit, and success, of aggressive mid-race pacing strategies during multi-modal endurance performance. Indeed, it is not unreasonable to suggest that attenuations in performance following an aggressively paced cycling section may, at least partly, be the result of triathletes having preconceived expectations of this strategy and the need to reduce their subsequent (i.e., running) exercise intensity as a result (Hausswirth et al., 1999). As such, it is thought that athletes are likely to perceive a higher than usual mid-event pace, and associated levels of psychophysiological strain, as posing an increased threat to the successful completion of an exercise task and, therefore, as having a "price to pay" at a later stage of performance (i.e., reduction in subsequent pace to restore anticipated levels of psychophysiological strain, and so reduce risk of premature exhaustion or harm) (de Koning et al., 2011; Cohen et al., 2013; Micklewright et al., 2015). However, whether altering the perceived "riskiness" of aggressive pacing during cycling can help to ameliorate impairments in subsequent running, and thus enhance overall triathlon performance, is yet to be elucidated.

It has been suggested that practically meaningful changes in triathlon running may result from deceptive pace manipulation, equivalent to the smallest worthwhile change in performance (i.e., typical within-athlete variability or coefficient of variation) (Taylor and Smith, 2014). More specifically, Taylor and Smith (2014) have demonstrated that run performance during sprint-distance triathlon may be enhanced by the imposition of a deceptively aggressive starting strategy (3% faster than baseline performance), when compared to more conservative approaches to initial pace deception (3% slower than, and equal to, baseline performance). These findings would appear to support the view that individual's typically maintain some form of reserve capacity during self-paced exercise and perform at a relative intensity somewhat below their task-specific maximum capacity, even when their intention is to optimize performance (Stone et al., 2012; St Clair Gibson et al., 2013). Furthermore, this study adds weight to the idea that an individual's expectations, beliefs and perceptions play an important role in how much reserve capacity they are willing to utilize during self-paced multi-modal exercise tasks. Given these points, it is reasonable to suggest that deceptively aggressive bike pacing may allow triathletes to maximize their performance within this discipline, help to avoid the reductions in running performance which may typically follow this strategy (i.e., Suriano and Bishop, 2010) and, in turn, optimize overall event time. However, as far as we are aware there are no studies to date which have examined the effects of deceptively aggressive bike pacing on triathlon performance.

There is a similar lack of experimental evidence regarding the relative importance of different perceptual responses to pacing and performance during multi-modal exercise (Wu et al., 2014). Indeed, the aforementioned study of Taylor and Smith (2014) reported non-significant trends for increased ratings of perceptual strain during the first 1.66 km of triathlon running when deceptively higher speeds were imposed, and vice-versa. Beyond this point (i.e., during self-paced completion of the run), a common pattern of development for many perceptual responses was seen between deceptive run conditions. These observations provide tentative evidence of the robustness that different psychophysiological and emotional responses have to manipulations of expectations and beliefs, and offer an insight into the relative importance of these perceptions in contextualizing or "framing" past, present, and future demands (and pacing) during multi-modal exercise. However, it is apparent that the findings and conclusions of Taylor and Smith (2014) may have been limited by the timing of deceptive pace

manipulation relative to the simulated triathlon overall (i.e., between 72–81% of total time), combined with the relative contribution of the run section to overall performance time in the event (i.e., ∼28% of total time). As such, the scope for deceptive manipulations of pace to make a meaningful difference to triathlon performance and distinguish the relative importance of perceptual mediators to pace regulation and reserve maintenance may therefore be greater during the earlier swim and cycle sections of the event.

With the aforementioned points in mind, and given that the cycling section typically contributes the highest proportion of overall triathlon time, this study examined the effects of deceptively aggressive bike pacing on performance, physiological and perceptual responses, and pacing during simulated sprint-distance triathlon. More specifically, it was hypothesized that completing the cycling section closer to the highest sustainable intensity (i.e., mean isolated time trial power output) would improve previous best simulated triathlon performance, irrespective of whether triathletes were made aware of this pacing strategy or not. However, it was also hypothesized that making triathletes aware of this aggressive cycling strategy would impair subsequent run and overall performance, relative to a deceptive pacing condition.

METHODS

Participants

Ten non-elite, trained male triathletes gave written, informed consent to participate in this study, with a mean (±*SD*) age, body mass, stature, and peak oxygen uptake ($\dot{V}O_{2peak}$) of 36.8 ± 8.9 years, 1.79 ± 0.08 m, 76.3 ± 7.2 kg, and 54.3 ± 5.7 $ml \cdot kg^{-1} \cdot min^{-1}$, respectively. Participants had been competing in triathlons for a minimum of 12 months and were all in their "off-season" throughout the study. The training completed by the group during the study period averaged 1.4 $h \cdot wk^{-1}$ (3.2 $km \cdot wk^{-1}$) swimming, 2.3 $h \cdot wk^{-1}$ (84.0 $km \cdot wk^{-1}$) cycling, 2.2 $h \cdot wk^{-1}$ (21.7 $km \cdot wk^{-1}$) running, in addition to 1.3 $h \cdot wk^{-1}$ of strength and conditioning. Before the completion of any data collection, all participants completed a medical history questionnaire and, having had the research procedures, requirements, benefits, and risks explained to them, they each provided written, informed consent. At this initial stage participants were told, incorrectly, that the intention of the study was to establish the reliability and validity of simulated sprint-distance triathlon performance, and associated physiological and perceptual responses. All study procedures were approved by the institutional ethics committee and, in line with internationally recognized ethical standards for deceptive sport and exercise science research (Harriss and Atkinson, 2015), all participants were fully debriefed upon completion of all trials, informed how they were deceived and why such deception was necessary, and were given the option to withdraw their data. Participants were permitted to follow their usual training regime throughout the study but were instructed to avoid training in the 24 h preceding each trial. As such, participants were asked to record and manage their training and dietary/fluid intake in order to maintain a consistent approach to the 24 h period preceding each trial.

Procedure and Apparatus

Participant's completed a total of eight testing sessions each, with the first four consisting of an "all-out" (non-drafted) swimming time-trial performed in their usual (25 m) training pool, separate incremental running and cycling tests to volitional exhaustion to establish each participant's peak physiological (i.e., $\dot{V}O_{2peak}$ and heart rate [HR_{peak}]) and performance (i.e., running speed [V_{max}] and power output [W_{max}]) characteristics, and a "race pace" familiarization of the sprint-distance triathlon simulation (750 m swim, 500 kJ bike, 5 km run) that they would be required to complete during subsequent experimental triathlon trials. Having completed all preliminary testing, each participant then performed an isolated cycling time-trial (TT) which required the completion of 500 kJ of work as quickly as possible. In light of the work by Suriano and Bishop (2010), it was reasoned that including this trial would determine each participant's highest sustainable intensity during a 500 kJ time-trial and would therefore serve as a benchmark with which to interpret cycling performance (and associated physiological or perceptual responses) during subsequent simulated triathlon trials. The remaining trials required each participant to complete three separate simulated sprint-distance triathlons (0.75 km swim, 500 kJ bike, 5 km run). These were performed at the same time of day, separated by an average of 8 days (range, 3–14 days) and completed in a maximum of 21 days. During all laboratory trials, swimming was performed in a temperature-controlled flume (Fastlane, Endless Pools, UK; water temperature ∼24.3°C), with all cycling and running completed in an adjacent environmentally controlled room (mean air temperature 21.7°C and mean relative humidity 56.5% across all trials). Electric fans were also placed ∼1 m in front of participants to provide continuous and consistent levels of additional air ventilation (∼4 $m \cdot s^{-1}$, CIMA AR-816 digital anemometer) throughout all cycling and running sections. Cycling was completed on an electromagnetically braked ergometer (SRM; Jülich, Welldorf, Germany) and running was performed on a motorized treadmill (HPCosmos, Traunstein, Germany).

The first simulated triathlon trial served to establish personal best "baseline" performance (BL). Swimming was completed at a fixed intensity equivalent to 90% of the average velocity recorded during each participant's preliminary "all-out" 750 m time-trial. As Peeling et al. (2005) have suggested that sprint-distance triathlon performance may be optimized by athletes maintaining this swimming intensity it was considered as a valid way to incorporate this discipline into short-distance triathlon simulations (Stevens et al., 2013). Having completed the swim and exited the flume participants were instructed to complete the remainder of the simulated triathlon (including transitions) as quickly as possible, as they would during competitive performance. The second and third simulated triathlon trials were completed in a randomized and counterbalanced order, with each requiring participants to maintain a prescribed power output for the entirety of the 500 kJ cycling section, before completing the run as quickly as possible. During both of these trials the (average) power output that participants were required to maintain was 5% greater than that achieved during BL performance. However, participants were only correctly

informed of this prior to and during one of these trials (HON). Before (and during) the alternate trial (DEC), participants were misinformed that they would be required to maintain a power output equal to that of their BL performance. As such, the on-screen feedback provided during this trial was manipulated so that it displayed average and real-time power output values 5% lower than they truly were, as measured by the SRM ergometer. The only other feedback provided during each cycling performance was verbal confirmation of every 5% (25 kJ) of total work completed. It was reasoned that informing participants of the HON pacing manipulation at this stage of the study (rather than during the pre-study period) would have helped to facilitate their best-possible BL performance and avoid any "holding back," in light of the greater demands that performing "as fast as possible" would likely lead to during subsequent trials (i.e., HON).

The magnitude of deception employed was selected based on the previously established coefficient of variation (CV) for power output during simulated triathlon cycling (CV = 4.8%; 95% CI = 3.4–8.4%) and associated estimates of sample size requirements (Taylor et al., 2012). As such, it was reasoned that a 5% manipulation of power output would allow for the imposition of a worthwhile performance change, whilst also being subtle enough to avoid any detection by participants across trials. Furthermore, the aggressiveness of this imposed pacing strategy (relative to TT performance) was comparable to previous non-deceptive manipulations of triathlete pacing during simulated sprint-distance cycle-run trials (Suriano and Bishop, 2010).

Throughout all running performances, the treadmill was interfaced with the computer-based NetAthlonTM software package (WebRacing Inc., Madison, WI) which was, in turn, projected onto a large monitor positioned in front of the treadmill. This provided a virtual representation of each participants progress over a flat 5 km run course in the form of an on-screen avatar (viewed from a second person perspective), in addition to numerical feedback regarding distance covered, current speed and average speed. In addition to this feedback, participants were informed prior to HON and DEC trials that they would be racing against a second on-screen avatar during the run which represented a replay of their BL performance. More specifically, participants were instructed to try their best to beat (or at least match) this on-screen "opponent." The view seen by each participant was always of the avatar representing their current performance. This meant that they were only able to see both avatars if they were performing worse than their BL trial (i.e., in a "chase" position). With this in mind, the distance separating both avatars was constantly displayed on-screen so that participants were able to keep track of their relative performance and respond to any changes in pace that were made during the BL trial. Upon completion of each run, the NetAthlonTM software stored distance, speed and time data at 1 s intervals for subsequent analysis.

The duration of first and second transition during HON and DEC trials replicated those recorded during BL performance (221 ± 31 and 93 ± 22 s, respectively) and were comparable to previous studies of simulated triathlon performance (Hausswirth et al., 2010; McGawley et al., 2012; Taylor et al., 2012; Taylor and Smith, 2014). The methods adopted to examine the respiratory responses of participants (see Section Physiological responses) meant that fluid intake was only possible during the cycling section of simulated triathlon. As such, participants were allowed to consume water *ad libitum* whenever these measures were not being recorded. More specifically, participants were instructed to drink as dictated by their levels of thirst, which is suggested as a more important factor to control during triathlon simulations than specific measures of hydration status (Noakes, 2010; Stevens et al., 2013). In any case, there were no significant differences in the volume of water consumed by each participant during simulated triathlon (or isolated TT) performances (mean volume 317 ± 177 ml across trials; $p > 0.05$).

Physiological Responses

During all laboratory trials, breath-by-breath measurements of oxygen uptake ($\dot{V}O_2$), respiratory exchange ratio (RER) and ventilation ($\dot{V}_E$) were obtained (Cortex Metalyzer, Leipzig, Germany), alongside heart rate (HR; RS_{400}, Polar Electro Kempele, Finland) and fingertip capillary blood lactate concentration ($[BLa^-]$; Lactate Pro 2, Arkray, Japan). Prior to each laboratory trial participants fitted a HR transmitter belt underneath their triathlon suit, with baseline measurements then obtained for $[BLa^-]$ and body mass. During simulated triathlon trials, measures of $[BLa^-]$ were obtained post-swim, at the end of every 100 kJ cycle section completed, and upon completion of each 1.66 km section of the run. These measures were also taken at the end of every 100 kJ during isolated TT performance. Body mass was measured immediately upon completion of each experimental trial. During isolated TT and simulated triathlon trials, the gas analysis system was fitted to participants immediately before they began cycling, by means of a leak-free face-mask and head-strap. However, to allow for fluid intake, this face-mask was removed from participants between 75–125, 175–225, 275–325, 375–425, and 475–500 kJ of the bike. During simulated triathlon trials this system (i.e., face-mask) was then reapplied at the end of second transition (i.e., once participants had mounted the treadmill) and was kept on for the duration of the run. Following each experimental trial, cardiorespiratory data was interpolated to 1 s averages using the manufacturer's software to match the frequency of this data with that of cycling power output and running speed. Mean HR values were determined for each triathlon discipline, whilst mean values for respiratory data were established for both the bike and run sections. In order to profile discipline-specific cardiorespiratory responses, data were averaged for 50–75 kJ of every 100 kJ cycle section completed and for each 1.66 km section of simulated triathlon running.

Perceptual Responses

During each experimental trial, verbal ratings of perceived exertion, effort, muscular pain, breathlessness, thermal discomfort, affect, and arousal were obtained using the same scales and instructions as outlined by previous studies of sprint-distance triathlon (Taylor and Smith, 2013, 2014). Whilst the relative order of these scales remained the same throughout the study, the first scale presented in the sequence

was randomized and counterbalanced for each participant, so as to minimize the interference between the relatively high number of separate perceptual responses. In the final 100 m of each triathlon swim, participants were prompted by an underwater visual signal to consider (and memorize) their perceptual status so that they could provide verbal responses to each scale during first transition. Perceptual responses were then obtained at the end of every 100 kJ cycle section and upon completion of each 1.66 km section of the run. These measures were also taken at the end of every 100 kJ during isolated cycling time-trials.

Statistical Analysis

All analyses were conducted using SPSS for Windows (Version 22, SPSS Inc., Chicago, USA) and Microsoft Excel (Microsoft Excel, 2007). A series of one-way repeated-measures ANOVA's were used to examine differences in swim, cycle, run and overall performance measures between BL, HON, and DEC triathlon trials, and to establish whether any performance differences existed between isolated cycling time-trials and the cycling section of each simulated triathlon. The same method of analysis was used to examine discipline-specific differences between trials in relation to the mean physiological and perceptual responses observed. In order to better consider the practical significance of results, data was also assessed by way of magnitude-based inferences (Batterham and Hopkins, 2006). Such analysis, performed using a published spreadsheet (Hopkins, 2003), provides quantitative (%) chances of "positive," "trivial," or "negative" effects between trials, based on the 90% confidence interval of the change value relative to a predetermined smallest worthwhile effect. With regards to cycling, running and overall performance data, the smallest worthwhile change values were based on those established by Taylor et al. (2012) during simulated sprint-distance triathlon performance of non-elite athletes (∼2.4, ∼0.6, and ∼1.2%, respectively). Likewise, the smallest worthwhile changes in physiological responses established by Taylor et al. (2012) were used to make magnitude-based inferences regarding these measures. However, given their lack of established CV values during triathlon, the smallest worthwhile change for each perceptual measure was set relative to 0.2 times the pooled between-subject *SD* (Hopkins, 2000).

Two-way within-subjects (trial x distance) ANOVA's were used to establish main effects of cycling condition and distance completed using mean 100 kJ section values for power output, $\dot{V}O_2$, $\dot{V}_E$, RER, [BLa^-], HR, perceived exertion, effort, muscular pain, breathlessness, affect, arousal, and thermal discomfort as dependent variables. The same analysis was used to examine data obtained during the running section of each simulated triathlon trial, using mean 1.66 km section values for speed and the same physiological and perceptual measures as dependent variables. Repeated measures ANOVA's were then used to identify changes in these variables during the course of each discipline. If the Mauchly test indicated a violation of sphericity then analysis of variance was adjusted using the Greenhouse–Geisser correction factor to reduce the likelihood of type I error. Where appropriate, Bonferroni-adjusted *post-hoc* tests were used to identify specific differences within and between trials. For all statistical procedures the level of significance was set at $p < 0.05$ and adjusted accordingly. All data are expressed as mean ± standard deviation and effect sizes for ANOVA outcomes as partial eta squared (η_p^2).

RESULTS

Performance Measures

As summarized in **Table 1**, there were no statistically significant differences in cycling time between HON, DEC, and TT, though each of these trials was significantly faster compared to BL ($p < 0.05$). As such, mean power output was significantly higher during TT, HON, and DEC, vs. BL (246 ± 34, 236 ± 33, and 236 ± 33, vs. 225 ± 32 W, respectively, $p < 0.05$). These power output values corresponded to 71, 65, 68, and 68% of W_{peak}, for TT, BL, HON, and DEC, respectively. Mean running speed during each triathlon trial corresponded 77, 77, and 78% of V_{peak}, for BL, HON, and DEC, respectively. Although these values suggest a trend for faster run performance during DEC, compared to both BL and HON, this was only statistically significant in comparison to BL ($p < 0.05$). Similarly, whilst there was a non-significant trend for overall triathlon time to be shorter during DEC than HON (by ∼17 s), the only statistically significant differences were between each of these trials and BL, which was between 2 and 3% slower overall than both DEC and HON ($p < 0.05$).

Repeated-measures ANOVA showed no main effect on power output for cycling distance, but did reveal a significant main effect for cycling condition and a significant condition × distance interaction, indicating differences across conditions in power output profiles when plotted against distance covered (**Figure 1A**). This assertion was supported by *post-hoc* analysis which highlighted a consistently higher power for each 100 kJ section during TT vs. BL. Although the pacing profiles during TT and BL developed in a similar (i.e., parallel) manner for much of the cycling bout, it was also evident that the marked increase in power output observed during the final 50 kJ of TT was absent during BL. During triathlon running, repeated-measures ANOVA revealed a significant main effect on speed for distance, but no main effect for condition and no condition × distance interaction (**Figure 1B**). As such, *post-hoc* analysis highlighted significant increases in speed for each successive 1.66 km section ($p < 0.05$) which culminated in an apparent "end-spurt" in the final 600 m of all triathlon trials.

With regard to the practical significance of performance differences, magnitude-based inferences suggest that cycling time

TABLE 1 | Mean ± *SD* overall and discipline-specific performance times during each simulated triathlon and isolated time-trial ($n = 10$).

	Swim (s)	Cycling (s)	Run (s)	Overall (s)
TT	–	2067 ± 312[b]	–	–
BL	848 ± 99	2270 ± 368[a,c,d]	1348 ± 140[c]	4465 ± 420[c,d]
DEC	848 ± 99	2158 ± 344[b]	1333 ± 129[b]	4339 ± 395[b]
HON	848 ± 99	2159 ± 343[b]	1350 ± 135	4356 ± 384[b]

NB, Significantly different from; TT, [a]$p < 0.05$; BL, [b]$p < 0.05$; DEC, [c]$p < 0.05$; HON, [d]$p < 0.05$.

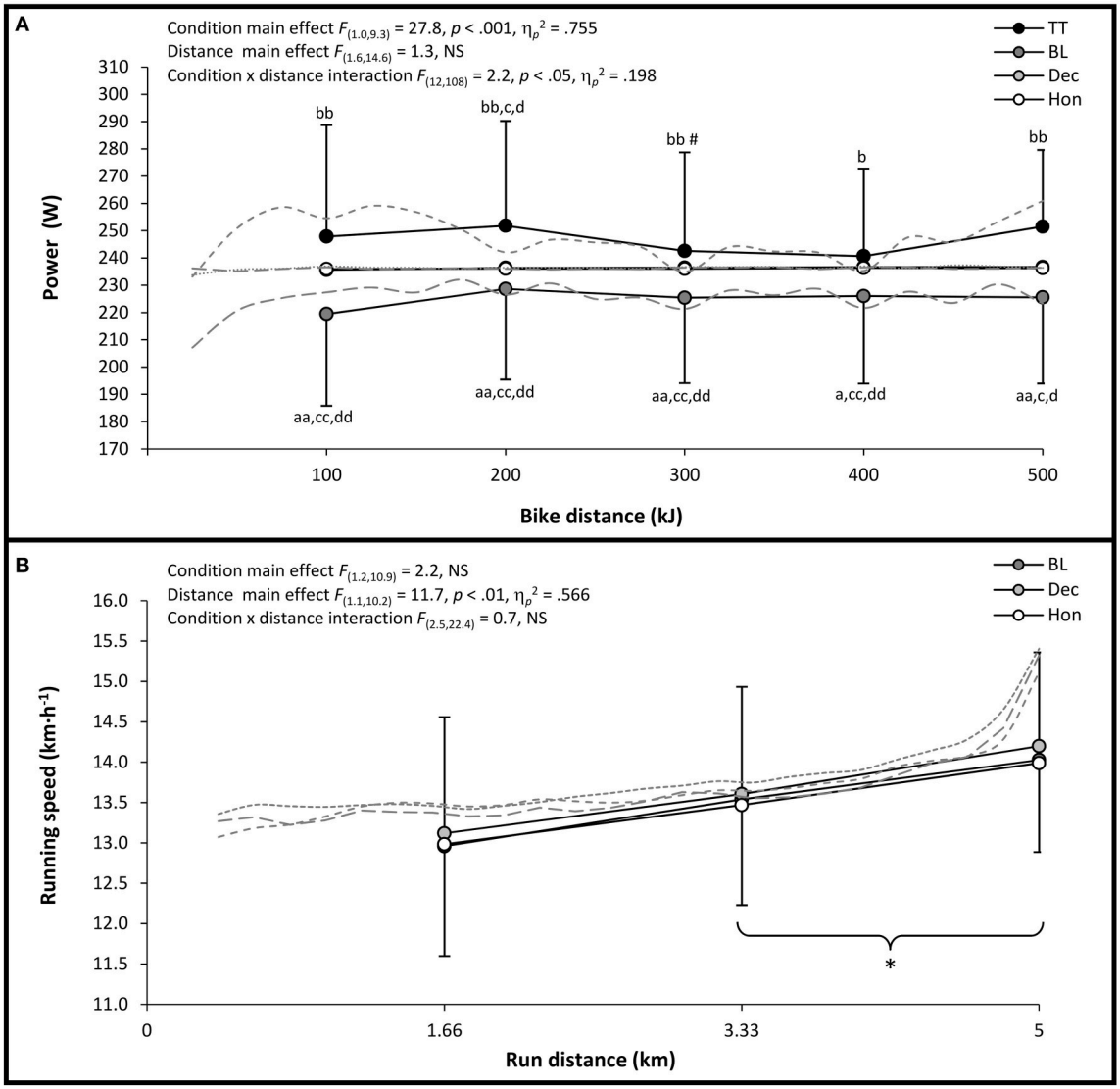

FIGURE 1 | (A) Mean ± *SD* power output for each 100 kJ (solid lines) and 25 kJ (dashed lines) completed in each cycling condition, **(B)** Mean running speed for each 1.66 km (solid lines) and 200 m (dashed lines) section completed in each triathlon trial. Significantly different from; TT, $^{a}p < 0.05$,$^{aa}p < 0.01$; BL, $^{b}p < 0.05$, $^{bb}p < 0.01$; DEC, $^{c}p < 0.05$, $^{cc}p < 0.01$; HON, $^{d}p < 0.05$, $^{dd}p < 0.01$; initial value, $^{*}p < 0.05$; previous value, $^{\#}p < 0.05$, (parentheses indicate significance in all conditions).

and power output were *almost certainly* better during TT, DEC, and HON, in comparison to BL (i.e., 100% likelihood of each being meaningfully faster than BL). Whilst DEC and HON cycling performances were *probably* worse compared to that of TT (i.e., 90% likelihood), there were *almost certainly* no performance differences between the DEC and HON cycling (i.e., 100% likelihood). Interestingly, whilst any practically important difference appeared *unclear*, it was more likely that HON running performance was meaningfully slower, than faster, vs. BL (i.e., 28:57:15% likelihood of HON being practically slower, of trivial difference, or practically faster than BL). On the other hand, DEC running performance was *probably* faster than both BL and HON (i.e., 89 and 79% likelihood, respectively). In terms of overall triathlon performance, there was *almost certainly* no difference between DEC and HON (i.e., 100% likelihood), although both were *almost certainly* faster vs. BL (i.e., 100% likelihood of each being meaningfully faster than BL).

Further to these findings, post-experimental debriefing revealed that all participants; (i) failed to identify the aggressive manipulation of cycling power output during DEC and, similarly (ii) believed that cycling intensity was highest (i.e., "most difficult") during their HON performance.

Physiological Measures

Table 2 summarizes the mean physiological responses during all triathlon and isolated cycling trials. There were no significant

TABLE 2 | Mean ± SD physiological responses during triathlon and TT trials ($n = 10$).

	$\dot{V}O_2$ (L·min^{-1})	$\dot{V}_E$ (L·min^{-1})	RER	HR (b·min^{-1})	[BLa$^-$] (mmol·L^{-1})
SWIM					
BL				115 ± 18	3.4 ± 2.0
DEC				113 ± 15	3.2 ± 1.5
HON				113 ± 16	3.2 ± 1.5
CYCLE					
TT	3.35 ± 0.40[b,c]	109.74 ± 22.38[b]	1.00 ± 0.04[b]	155 ± 11[b,c,d]	6.9 ± 3.2[b,d]
BL	3.12 ± 0.37[a]	94.43 ± 17.39[a,d]	0.94 ± 0.04[a,c]	145 ± 10[a]	3.9 ± 2.3[a,c,d]
DEC	3.15 ± 0.35[a]	99.35 ± 14.81	0.96 ± 0.04[b]	148 ± 11[a]	4.8 ± 2.2[b]
HON	3.20 ± 0.37	101.27 ± 18.08[b]	0.97 ± 0.04	149 ± 11[a]	4.8 ± 2.5[a,b]
Run					
BL	3.59 ± 0.47	115.31 ± 24.94	0.92 ± 0.04	163 ± 10	6.4 ± 2.6
DEC	3.64 ± 0.50	118.68 ± 26.54	0.93 ± 0.03	162 ± 10	6.8 ± 3.0
HON	3.56 ± 0.46	115.73 ± 25.29	0.93 ± 0.03	162 ± 9	6.0 ± 2.5

NB, Significantly different from; TT, [a]$p < 0.05$; BL, [b]$p < 0.05$; DEC, [c]$p < 0.05$; HON, [d]$p < 0.05$.

differences in mean physiological responses (i.e., HR and [BLa$^-$]) elicited by the swim section of each simulated triathlon ($p > 0.05$). Mean cycling intensity during each trial corresponded to 91, 85, 87, and 87% of HR_{peak}, and 87, 81, 83, and 82% of $\dot{V}O_{2peak}$, for TT, BL, HON, and DEC, respectively. As such, comparisons of each cycling bout revealed that physiological responses during TT were significantly higher than those recorded during BL ($p < 0.05$). Furthermore, the greater demands of HON and DEC cycling were reflected in a number of elevated physiological responses compared to BL, particularly that of [BLa$^-$].

Despite these observations, mean HR and $\dot{V}O_2$ values did not significantly differ between BL, HON, and DEC cycling ($p > 0.05$). Although no significant physiological differences were evident between HON and DEC cycling, it is noteworthy that only HON had a mean $\dot{V}O_2$ which was not significantly lower than TT ($p > 0.05$). Mean intensity during each triathlon run corresponded to 92, 91, and 92% of HR_{peak}, and 87, 86, and 88% of $\dot{V}O_{2peak}$, for BL, HON, and DEC, respectively. As summarized in **Table 2**, there were no significant differences in mean physiological responses during BL, HON, and DEC running ($p > 0.05$).

Magnitude-based inferences suggested that the likelihood of a practically meaningful elevation in all physiological responses during TT vs. the cycling section of all triathlon trials ranged from *likely* to *almost certain* (i.e., 82 to 100% likelihood of being meaningfully higher during TT). Likewise, almost all physiological responses were *possibly* to *almost certainly* higher during DEC and HON cycling compared to BL (i.e., 62 to 98% likelihood of being meaningfully higher vs. BL), with mean $\dot{V}O_2$ the only exception. As such, it was *likely* (i.e., 90% certain) that any difference in mean $\dot{V}O_2$ between DEC and BL cycling sections was trivial. Mean physiological responses during DEC and HON cycling were of trivial or unclear difference. During running, most of the practically meaningful physiological differences were seen between HON and DEC, with $\dot{V}O_2$, $\dot{V}_E$, and [BLa$^-$] values being either *likely* or *possibly* lower during HON (i.e., 58 to 81% likelihood of a meaningful difference).

Figure 2 profiles the physiological responses during simulated triathlon and isolated cycling bouts, including the outcomes of two-way (trial x distance) ANOVA's and *post-hoc* comparisons. As such, significant main effects of cycling condition were found for all physiological measures, whilst there were main effects for distance on HR, $\dot{V}O_2$, and $\dot{V}_E$ ($p < 0.05$). No significant condition × distance interactions were found for any physiological measure ($p > 0.05$). *Post-hoc* analysis revealed much of the disparity in physiological response to be between BL and TT trials conditions, with direct comparisons of HON and DEC data revealing no significant differences ($p > 0.05$). However, there was a trend for respiratory measures during HON to be higher than DEC, which was indirectly supported by the disparity in significant differences when comparing each of these trials with BL and/or TT. Significant main effects of distance on physiological responses (HR and RER) were found to be a result of significant differences in all conditions between measures taken during the first 100 kJ section and all subsequent measurement intervals. The profile of physiological response during each simulated triathlon run is detailed in **Figure 3**, which also includes results of primary and *post-hoc* statistical analysis. As suggested by **Table 2**, there were no significant main effects of prior cycling condition on any physiological measure during running, nor were any significant condition × distance interactions evident ($p > 0.05$). However, significant main effects of run distance were found for HR, $\dot{V}O_2$, and $\dot{V}_E$ ($p < 0.05$), with all trials demonstrating significant increases in HR and $\dot{V}_E$ from each 1.66 km section to the next.

Perceptual Measures

Table 3 summarizes group mean perceptual responses during the completion of TT and triathlon cycling trials. As such, no significant differences in perceptual strain were elicited by the swim section of each triathlon. Furthermore, there were no statistically significant differences between triathlon trials in mean perceptual responses during cycling or running. It was evident that TT cycling was associated with significantly higher mean RPE compared to all bouts of triathlon cycling. It is

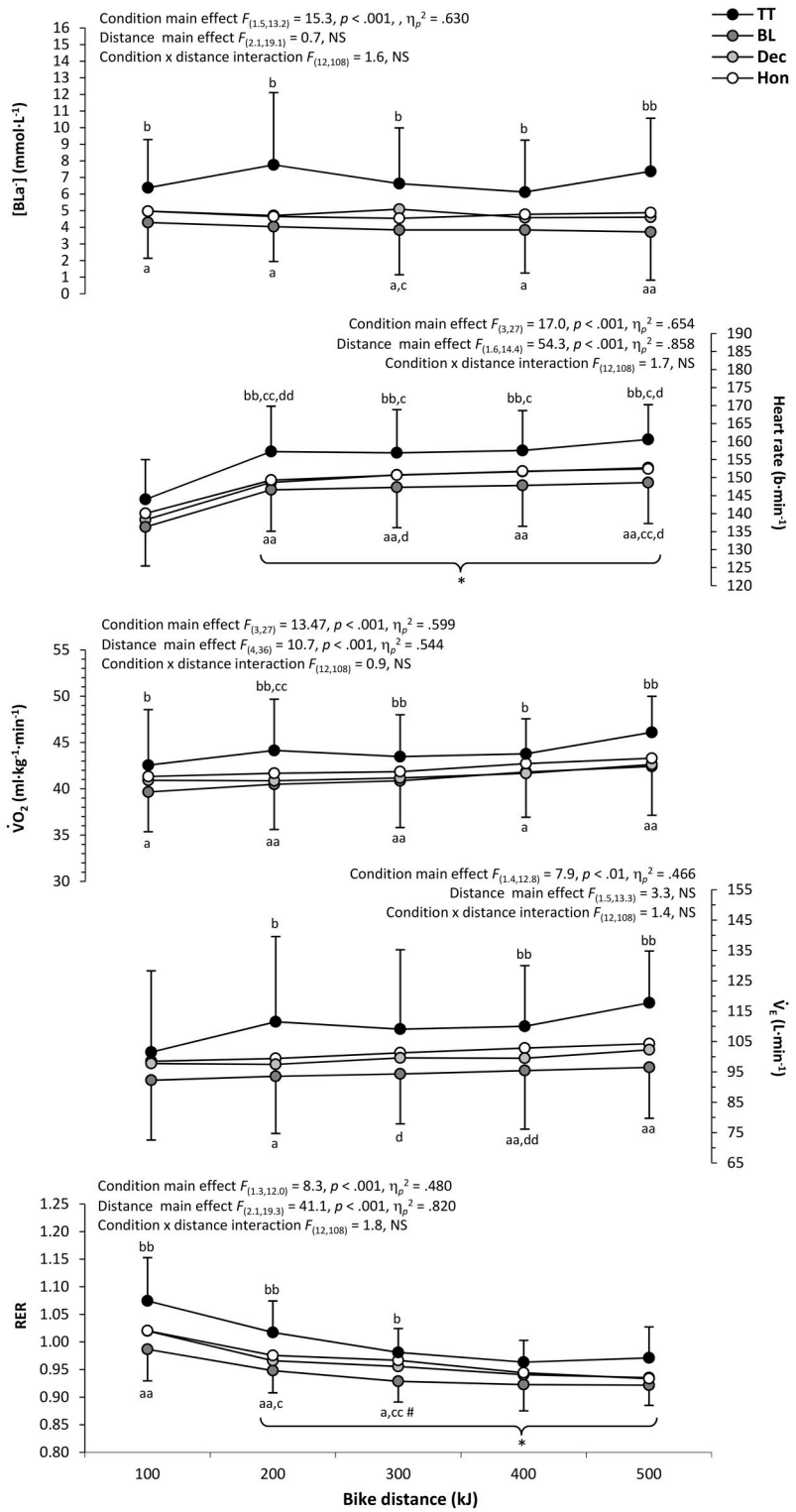

FIGURE 2 | Mean ± *SD* physiological responses for each 100 kJ cycling section. Significantly different from; TT, $^{a}p < 0.05$, $^{aa}p < 0.01$; BL, $^{b}p < 0.05$, $^{bb}p < 0.01$; DEC, $^{c}p < 0.05$, $^{cc}p < 0.01$; HON, $^{d}p < 0.05$, $^{dd}p < 0.01$; initial value, $^{*}p < 0.05$; previous value, $^{\#}p < 0.05$ (parentheses indicate significance in all conditions).

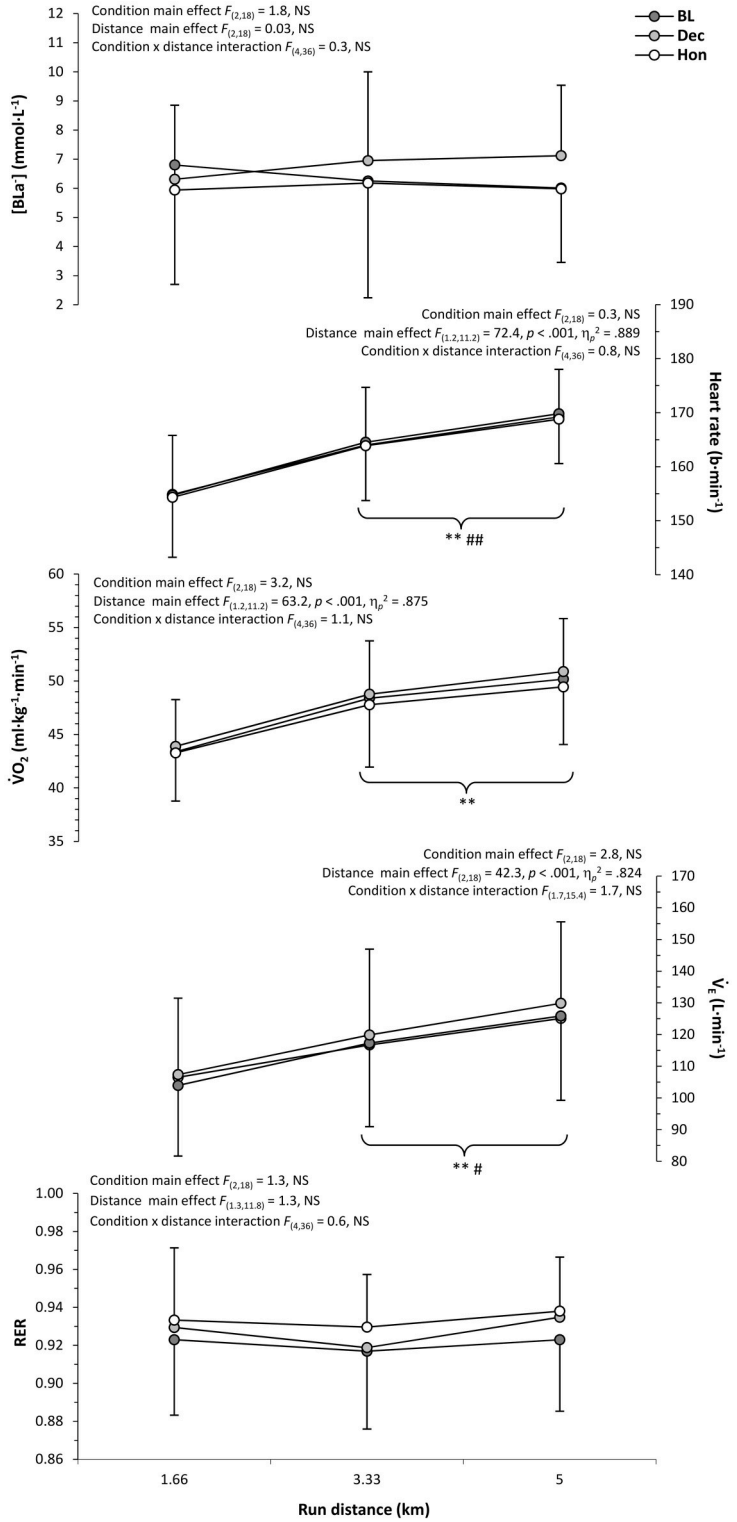

FIGURE 3 | Mean ± *SD* physiological responses for each 1.66 km run section. Significantly different from; initial value, $^{**}p < 0.01$; previous value, $^{\#}p < 0.05$, $^{\#\#}p < 0.01$ (parentheses indicate significance in all conditions).

also noteworthy that only during HON were there no other significant differences in mean perceptual response compared to those during TT.

Based on magnitude-based inferences, mean perceptual response during TT vs. the cycling section of all triathlon trials was *likely* to *almost certainly* higher for all measures (i.e., 71 to 99% likelihood), except for affect and arousal. As such, mean affect was *likely* lower during TT vs. all other bouts of cycling (i.e., 81 to 89% likelihood). In the case of arousal, there were no clearly meaningful differences evident, with the most likely outcome being a *trivial* difference between trials (i.e., 55 to 75% likelihood). Comparisons between BL, DEC, and HON cycling revealed *trivial* or *unclear* differences in almost all perceptual responses, with thermal strain being the only exception to this. As such, thermal strain was *likely* higher during DEC compared to BL (i.e., 88% certain). During running, thermal strain was again one of few perceptual responses to meaningfully differ between trials, being *likely* lower during both HON and DEC (i.e., 92 and 93% certainty, respectively), compared to BL. The only meaningful difference in perceived exertion was a *possibly* lower mean score during DEC vs. HON running (i.e., 67% certain). Further to this, differences in affect were limited to DEC being *likely* higher (i.e., more positive) than both BL and HON (i.e., 84 and 82% certainty, respectively).

Based on magnitude-based inferences, a meaningfully higher mean perceptual response during TT vs. the cycling section of all triathlon trials ranged from *likely* to *almost certain* for all measures (i.e., 71 to 99% likelihood), except for affect and arousal. As such, mean affect was *likely* lower during TT vs. all other bouts of cycling (i.e., 81 to 89% likelihood). In the case of arousal, there were no clear or meaningful differences evident, with the most likely outcome being a *trivial* difference between trials (i.e., 55 to 75% likelihood). Comparisons between BL, DEC, and HON cycling sections revealed *trivial* or *unclear* differences in almost all mean perceptual responses, with thermal strain being the only exception to this. As such, mean thermal strain was *likely* higher during DEC compared to BL (i.e., 88% certain). During running, thermal strain was again one of few perceptual responses to meaningfully differ between trials, being *likely* lower during both HON and DEC (i.e., 92 and 93% certainty, respectively), compared to BL. The only meaningful difference in perceived exertion was a *possibly* lower mean score during DEC vs. HON running (i.e., 67% certain). Further to this, differences in affect were limited to DEC being *likely* higher (i.e., more positive) than both BL and HON (i.e., 84 and 82% certainty, respectively).

Distance profiles (and associated statistical outcomes) of perceptual measures during cycling and running sections of each trial are presented in **Figures 4**, **5**, respectively. Significant distance effects were found for all perceptual measures during cycling ($p < 0.05$), whilst a significant main condition effect was evident for all perceptual responses except for affect and arousal ($p > 0.05$). A significant condition × distance interaction was only apparent for RPE and breathlessness ($p < 0.05$). During running, significant distance effects were found for all perceptual measures ($p < 0.05$), although no condition effects or condition × distance interactions were evident for any perceptual response ($p > 0.05$). Further to these findings, collated individual perceptual responses across the duration of each triathlon trial revealed strong correlations with the percentage of overall triathlon time completed ($r = 0.92$–0.97, $p < 0.05$). Repeated-measures ANOVA showed the relationship (i.e., r coefficient) between individual participants' perceptual status and percentage of overall triathlon time was largely unaffected by cycling condition, with no statistically significant main effects found ($p > 0.05$).

As a simple index of the momentary risk perception associated with pacing behavior, the so-called "Hazard Score" (de Koning et al., 2011) was individually calculated and profiled across each triathlon trial by multiplying RPE values by the proportion of overall triathlon distance remaining at that particular point in time (**Figure 6A**). Analysis via two-way repeated-measures ANOVA failed to show a significant main effect on Hazard Score for triathlon condition or a significant condition × distance interaction, although there was a significant main effect for total triathlon distance. Hazard Scores were also calculated specifically for cycling and running sections by multiplying reported RPE values by the proportion of discipline-specific distance remaining at that point. For cycling-specific Hazard Scores (**Figure 6B**), two-way repeated-measures ANOVA showed significant main effects for condition [$F_{(3.0, 27.0)} = 4.5$, $p < 0.05$, $\eta_p^2 = 0.33$] and distance [$F_{(1.5, 13.6)} = 1029.1$, $p < 0.001$, $\eta_p^2 = 0.99$], although no significant condition-by-distance interaction was seen ($p > 0.05$). *Post-hoc* analysis highlighted that between-condition differences during cycling were attributable to the Hazard Scores of TT, which were significantly higher compared to HON at 200 kJ ($p < 0.05$), and vs. both BL and DEC at 400 kJ ($p < 0.05$). The same analysis of running-specific Hazard Scores (**Figure 6C**) failed to show a significant main condition effect or significant condition x distance interaction ($p > 0.05$), although there was a significant main effect for running distance [$F_{(1.1, 10.0)} = 684.2$, $p < 0.001$, $\eta_p^2 = 0.99$).

DISCUSSION

The aim of this study was to ascertain the effects of deceptively aggressive bike pacing on performance, and associated physiological and perceptual responses, during simulated sprint-distance triathlon. With this in mind, the experimental hypothesis that cycling closer to the highest sustainable intensity (i.e., mean isolated time trial power output) would improve previous best simulated triathlon performance was accepted. This was the case irrespective of whether or not triathletes were made aware of this relatively aggressive pacing strategy. The decision to accept this hypothesis was based on the finding of significant ($p < 0.05$) and almost certainly meaningful improvements in the overall simulated triathlon times of both HON and DEC, compared to that of previous best (i.e., BL) performance. Similarly, the hypothesis that making triathletes aware of aggressive cycle pacing would impair subsequent run and overall performance, relative to that of a deceptive pacing condition, was also accepted. This decision was made in light of the significant ($p < 0.05$) and probably meaningful improvements in running time during DEC, compared to

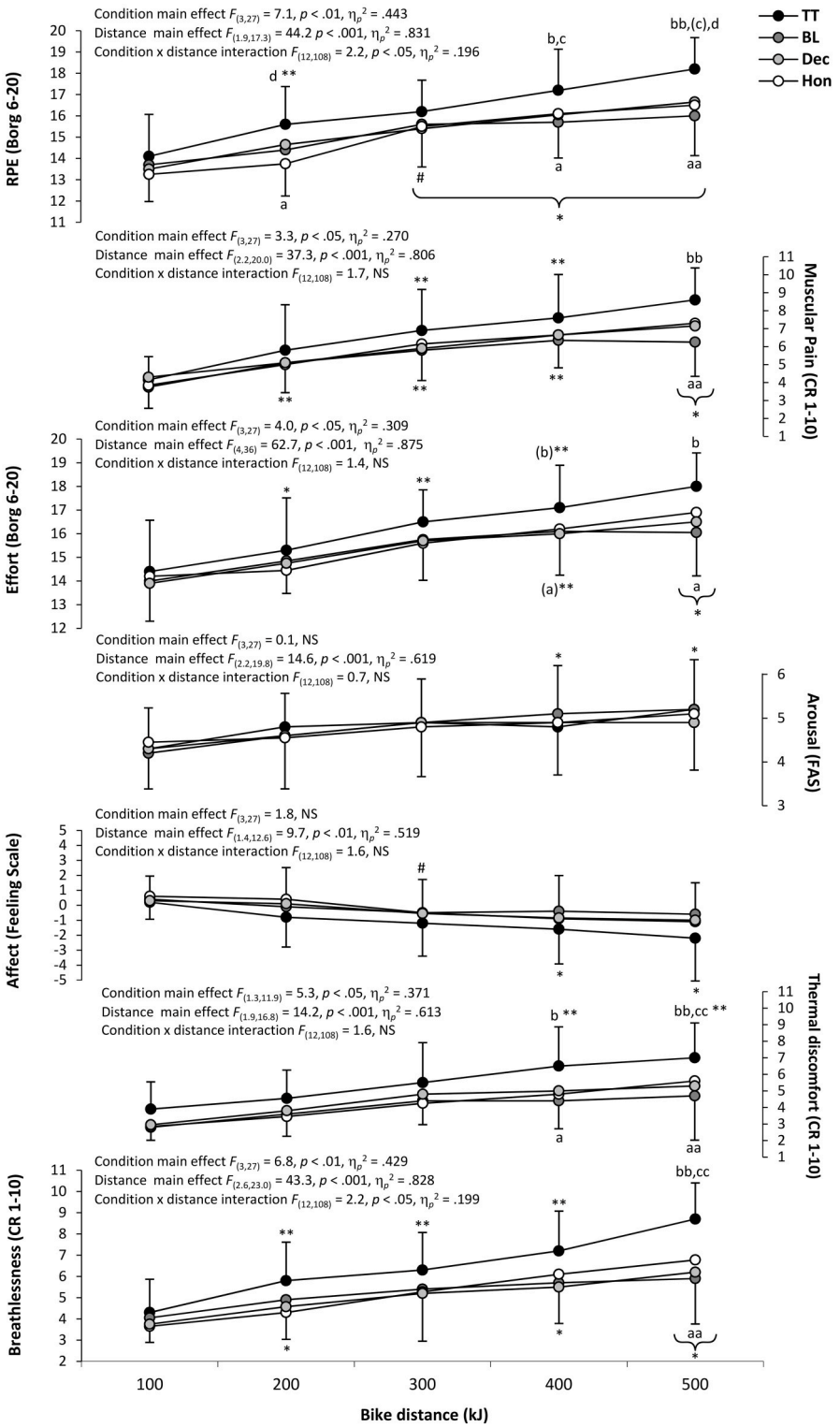

FIGURE 4 | Mean ± *SD* perceptual responses for each 100 kJ cycle section. Significantly different from; TT, $^{(a)}p = 0.051$, $^{a}p < 0.05$, $^{aa}p < 0.01$; BL, $^{(b)}p = 0.051$, $^{b}p < 0.05$, $^{bb}p < 0.01$; DEC,$^{(c)}p = 0.051$, $^{c}p < 0.05$, $^{cc}p < 0.01$; HON, $^{d}p < 0.05$, $^{dd}p < 0.01$; initial value, $^{*}p < 0.05$, $^{**}p < 0.01$; previous value, $^{\#}p < 0.05$ (parentheses indicate significance in all conditions).

TABLE 3 | Mean ± *SD* perceptual responses during BL, DEC, HON, and TT trials ($n = 10$).

	Exertion	Effort	Muscular Pain	Thermal Discomfort	Breathlessness	Arousal	Affect
SWIM							
BL	12.5 ± 2.1	12.8 ± 2.1	2.7 ± 1.2	2.3 ± 1.3	3.6 ± 1.4	4.1 ± 1.0	1.4 ± 1.8
DEC	11.8 ± 1.8	12.2 ± 1.7	2.5 ± 1.3	2.3 ± 0.8	3.0 ± 1.0	4.3 ± 1.0	1.4 ± 1.8
HON	11.9 ± 2.2	12.0 ± 2.6	2.9 ± 2.1	2.1 ± 0.9	2.5 ± 1.7	3.7 ± 1.1	2.1 ± 2.1
CYCLE							
TT	16.3 ± 1.5[b,c,d]	16.3 ± 1.7	6.6 ± 1.9	5.5 ± 1.9	6.5 ± 1.6[b,c]	4.8 ± 1.0	−1.1 ± 2.0
BL	15.1 ± 1.3[a]	15.4 ± 1.6	5.5 ± 1.4	4.0 ± 1.2	5.2 ± 1.3[a]	4.8 ± 1.0	−0.2 ± 1.8
DEC	15.3 ± 1.6[a]	15.4 ± 1.8	5.8 ± 1.9	4.4 ± 1.2	5.0 ± 1.6[a]	4.7 ± 0.9	−0.4 ± 2.1
HON	15.0 ± 1.7[a]	15.5 ± 2.0	5.8 ± 1.7	4.2 ± 1.3	5.2 ± 1.7	4.8 ± 1.1	−0.3 ± 2.2
RUN							
BL	16.9 ± 1.5	17.0 ± 1.6	7.3 ± 1.9	6.4 ± 1.5	7.8 ± 1.6	5.4 ± 0.9	−1.4 ± 2.2
DEC	16.5 ± 1.8	16.9 ± 2.3	7.1 ± 1.9	5.8 ± 2.1	7.3 ± 2.0	5.3 ± 0.9	−0.8 ± 2.6
HON	16.6 ± 1.9	16.7 ± 2.2	7.1 ± 2.0	5.8 ± 1.9	7.4 ± 2.0	5.3 ± 1.0	−1.5 ± 2.6

NB, Significantly different from; TT, [a]$p < 0.05$; BL, [b]$p < 0.05$; DEC, [c]$p < 0.05$; HON, [d]$p < 0.05$.

BL, and the apparent failure of triathletes to significantly or meaningfully improve on their BL run performance during HON ($p > 0.05$, possibly trivial/unclear difference). Furthermore, whilst the 17 s difference between HON and DEC running times did not reach statistical significance, it would appear *probable* or *likely* that this represents a meaningfully quicker run performance during DEC. Indeed, the differences in running performance between DEC and the relatively slower BL and HON trials are comparable to those observed during the deceptively manipulated triathlon running trials of Taylor and Smith (2014). As highlighted by these authors, such differences cannot be ignored given that an average of only 9 s can separate the run and overall event ranking positions for of the top 20 sprint-distance triathletes at (age-group) World Championship level (ITU, 2012).

The current study findings therefore extend those of previous deception research to offer further evidence that expectations and beliefs regarding a particular exercise task and/or intervention are likely to influence athletes' perception of internal and external stimuli, and the subsequent conscious (anticipatory) pacing decisions they make in attempting to optimize performance (Micklewright et al., 2010; Stone et al., 2012; Taylor and Smith, 2014; Williams et al., 2014, 2015; Waldron et al., 2015; Shei et al., 2016). It has been speculated that this is the case during multi-modal exercise (Hausswirth et al., 1999), with previous simulated triathlon studies finding that a relatively aggressive mid-event (i.e., cycling) pacing strategy leads to subsequent reductions in running performance (Hausswirth et al., 1999; Suriano and Bishop, 2010). However, this is the first study to offer clear experimental evidence in support of this suggestion, with the superior running performance of DEC illustrating that expectations regarding aggressive mid-event pacing can strongly influence subsequent exercise intensity regulation and performance during multi-modal exercise. As such, the profile of run pacing during DEC revealed a more aggressive starting strategy coupled with earlier initiation of an end-spurt, relative to BL and HON trials (**Figure 1B**).

It would therefore appear that deceptively aggressive bike pacing allows triathletes to maximize their sustainable intensity in this discipline, without the subsequent impairments in running performance which are typically seen when athletes are made aware of this mid-event cycling strategy. This corroborates with the suggestion that athletes perceive higher and/or earlier than anticipated levels of exercise intensity as posing a greater risk to the completion of an exercise task and, therefore, as having a "price to pay" at a later stage of performance (i.e., reduction in running pace to maintain sufficient reserve and avoid premature exhaustion or risk of harm) (Cohen et al., 2013; Micklewright et al., 2015). Task-specific expectations and beliefs therefore appear to play a key role in determining how much reserve capacity individuals are willing and able to utilize in the pursuit of optimal self-paced multi-modal exercise performance. With this in mind, there may be a common need, particularly amongst non-elite sprint-distance triathletes, to "relearn" what constitutes an optimal pacing strategy across the entire event. More specifically, if triathletes are to optimize short-distance event performance then it would appear that the holding back of any reserve capacity should be minimized during the cycle section. That is, the highest sustainable intensity should be maintained so as to replicate isolated time-trial performance as closely as possible, as suggested by Suriano and Bishop (2010). Likewise, the highest sustainable (even) pace should be established during the early stages of the triathlon run so that there is minimal available reserve with which to perform a final end-spurt. However, given that the pacing template of experienced triathletes is likely to be well-established (Baron et al., 2011) further research is needed to establish the extent to which such "re-education" of pacing is possible, how it may be facilitated by sports scientists and coaches, and ways in which such deviation from a previously-favored pacing strategy may be influenced by individual risk-perception and risk-taking traits (Micklewright et al., 2015).

As highlighted in a recent review of factors influencing pacing during triathlon (Wu et al., 2014), it is evident that the perceptual mechanisms underpinning multi-modal endurance performance

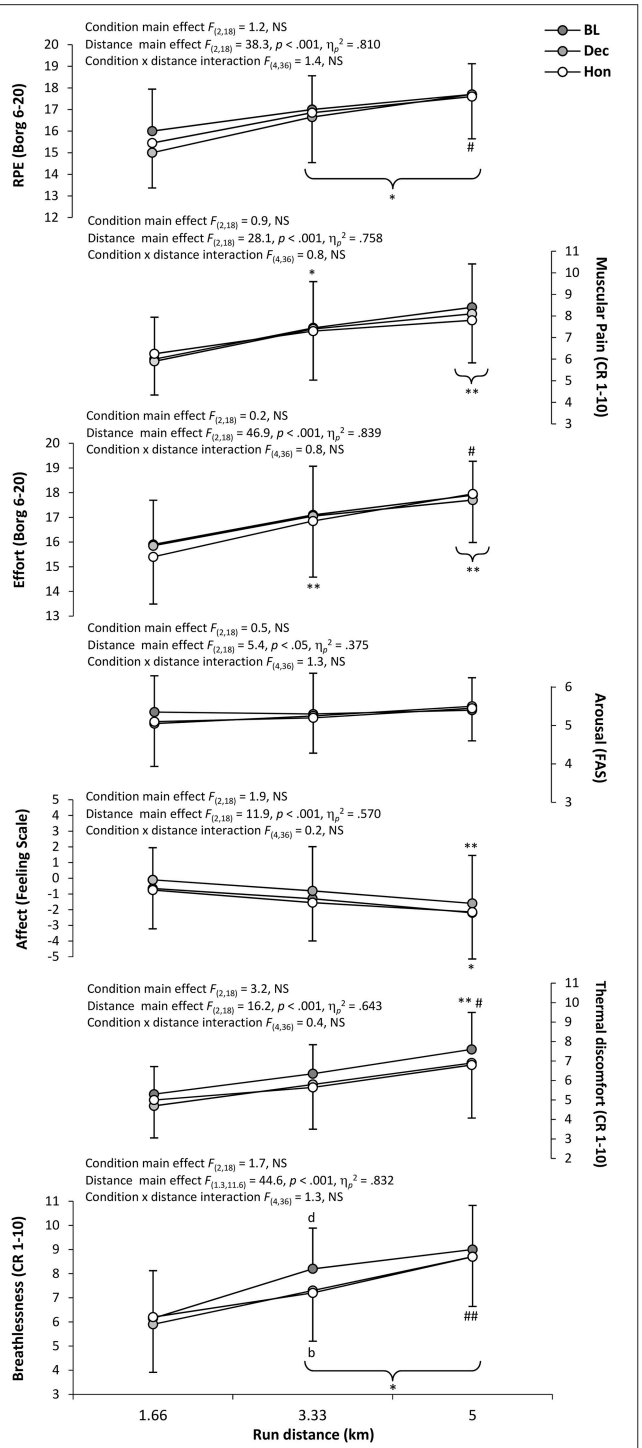

FIGURE 5 | Mean ± *SD* perceptual responses for each 1.66 km run section. Significantly different from; BL, $^{b}p < 0.05$; HON, $^{d}p < 0.05$; initial value, $^{*}p < 0.05$, $^{**}p < 0.01$; previous value, $^{\#}p < 0.05$, $^{\#\#}p < 0.01$ (parentheses indicate significance in all conditions).

have been largely neglected by research to date. Indeed, whilst a number of studies have examined the physiological responses of triathletes to manipulations of cycling intensity (Hausswirth et al., 1999, 2001; Solano et al., 2003; Suriano and Bishop, 2010), this is the first study to have considered how a number of perceptual responses may also be influenced by the relative intensity of triathlon-specific cycling and subsequent running. Furthermore, the diversity and frequency of physiological measures obtained during the current study offers a previously unavailable profile of how these responses may develop as a result of both deceptive and non-deceptive manipulations of cycle pacing during complete triathlon performance. Generally, it would appear that levels of physiological and perceptual strain increased with higher cycling intensities during the current study, with little, if any, substantial difference in physiological and perceptual response during each triathlon run. There was also a broad trend for physiological and perceptual strain to increase as a greater proportion of each discipline, and overall triathlon performance, was completed (**Figures 2**–**5**).

These observations underline the suggested "holding back" of a progressively decreasing reserve capacity over the course of "fastest possible" triathlon performance (i.e., "BL"). They would also appear to confirm that the anticipatory process of reserve maintenance is sensitive to levels of both physiological and perceptual strain during self-paced multi-modal exercise (Swart et al., 2009; Tucker, 2009). However, it is evident that any differences in physiological or perceptual response observed during each simulated triathlon trial were much more subtle than those seen for performance-related measures, particularly when comparing HON and DEC trials. The failure to establish clear links between physical and/or perceptual responses and performance is not uncommon in contemporary pacing research (Micklewright et al., 2010; Jones et al., 2014; Rhoden et al., 2014). Indeed, such findings reinforce the view that psychophysiological processes interact in a complex and multidimensional manner during the regulation of self-paced exercise performance (Renfree et al., 2012; Jones et al., 2014). As such, the methods used to examine physical and perceptual factors during future studies may need further refinement (e.g., increased frequency, consideration of the specific thoughts of participants) to be able to more clearly understand their interaction and influence during self-paced multi-modal exercise.

With this in mind, the authors are cognizant of the fact that there are potential limitations within the current study design which may have impacted the strength with which it was able address the key aims and hypotheses. Indeed, it could be argued that the counterbalancing of HON and DEC trials may have led to some participants becoming more, or less, consciously attuned to the demands of aggressive cycle pacing by the time they were exposed to DEC. Although post-experimental debriefs suggested that this was not the case, such an ordering effect could have made it less likely for those completing HON first to have been truly deceived about their pacing during their subsequent DEC performance. At the very least, the different ordering of DEC and HON trials may have the potential to influence the perceptual responses of participants and so should be considered as a limitation of the current study. Indeed, whilst participants did not report being consciously aware of any deceptive manipulation, it was evident from a number of debrief interviews that their prior experiences of either DEC

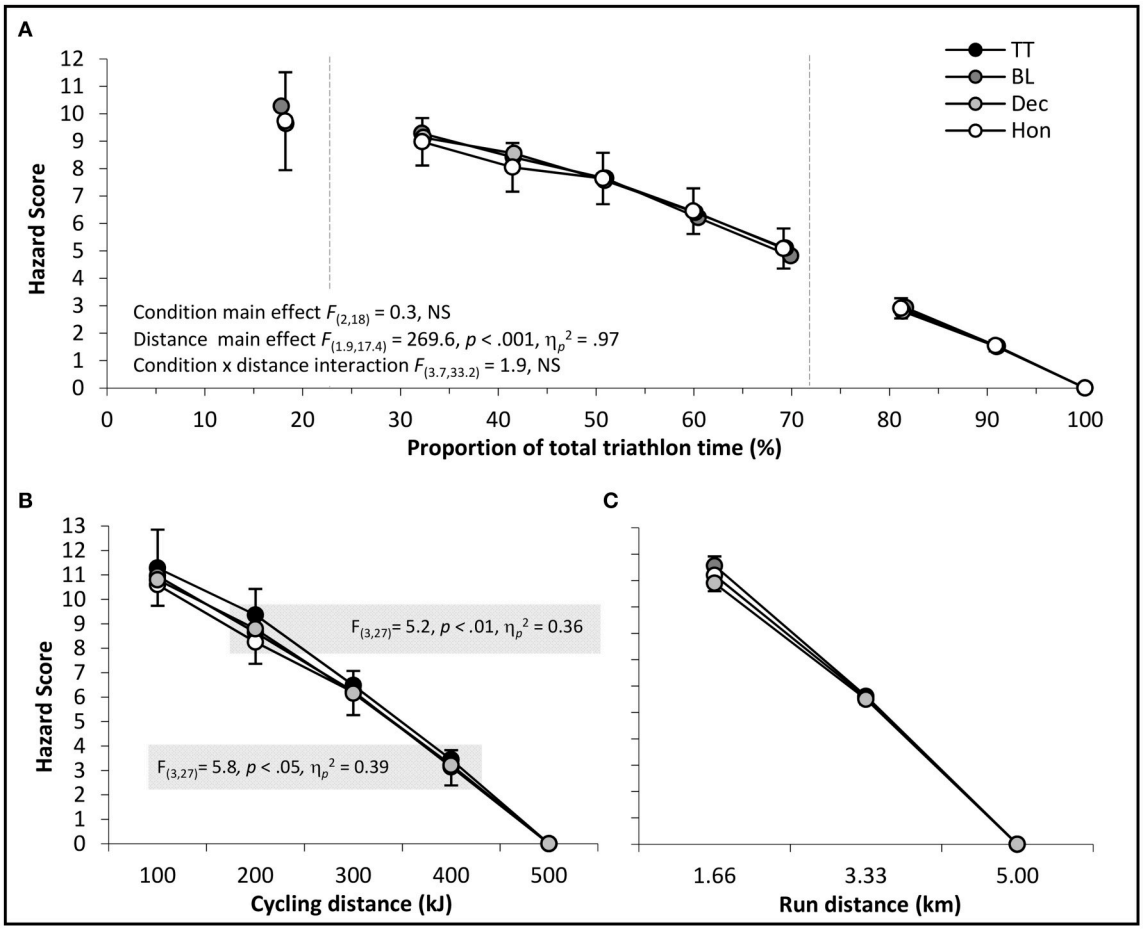

FIGURE 6 | Mean ± *SD* Hazard Scores in relation to (A) the proportion of total triathlon distance remaining, **(B)** the proportion of the bike section remaining, **(C)** the proportion of run distance remaining (dashed lines indicate transition end).

and HON somehow served to "frame" their approach to, and interpretation of, subsequent performance trials. Whilst this view corroborates with previous work focusing on the effects of prior experiences during relatively short single-mode endurance performance (e.g., Micklewright et al., 2010), it is certainly a line of study which would be of value for researchers to explore during multi-modal endurance performance. That said, the value of randomization and counterbalancing of experimental conditions within a repeated-measures study design cannot be ignored, given that not doing so may clearly be criticized for introducing confounding ordering or time-related effects (i.e., learning/familiarity, fatigue, training/fitness status, equipment). Whilst the authors are therefore confident in the robustness of the current study design, such findings must always be viewed with a degree of caution in light of the specific context of the study and the possible limitations associated with the particular approach taken.

Irrespective of these points, the current study provides valuable and novel evidence with which to address some the ongoing challenges to RPE being considered as the chief perceptual mediator of pace regulation during exercise. Indeed, based on their observations during and after aggressive mid-event pacing during single-mode (cycling) exercise, Cohen et al. (2013) concluded that RPE may be less closely tied with deviations away from template power output (i.e., reserve access) than is proposed by the "anticipatory-RPE" model of Tucker (2009). The current study would appear to lend some support to this suggestion during multi-modal exercise, given the lack of any significant difference in RPE during each simulated triathlon. Furthermore, the conversion of RPE values into a supposedly more meaningful index of pacing "riskiness" (i.e., the Hazard Score of de Koning et al., 2011) failed to distinguish between each triathlon trial of the present study, despite substantial differences in pacing and performance between cycling and running sections of each trial. On the other hand, some of the current study observations would still seem to suggest that triathletes utilize discipline-specific templates to interpret and manage levels of psychophysiological strain, and that these templates can be influenced by task-specific beliefs and expectations. Indeed, whilst they were not statistically different, if the profiles of RPE increase during each period of triathlon cycling were maintained beyond the end

of the discipline (i.e., projected forward), then an RPE value of 20 (i.e., "maximal exertion") would not have been reached until 130, 108, and 103% of the total triathlon duration for BL, DEC, and HON, respectively. Extending the findings of Taylor and Smith (2014), this would appear to further illustrate the supposed role of RPE in maintaining a reserve capacity during "fastest possible" self-paced triathlon performance (i.e., BL trial) and highlight the subtle, but practically meaningful, effects of deception on the regulation and forecasting of RPE during individual triathlon modalities, both of which are indicative of discipline-specific RPE templates. However, it is important to note that these between trial differences in projected levels of psychophysiological strain were not exclusive to RPE and were evident in the profiles of all other perceptual responses.

Given these points, it is appears likely that an array of psychophysiological factors may indeed influence pacing decisions during exercise, possibly by way of "fine-tuning" the "coarse" relationship between RPE growth and momentary power output (Cohen et al., 2013). This suggestion is not unique, with a growing number of contemporary pacing studies theorizing that perceptions other than RPE (e.g., sense of effort, perceived muscular pain, breathlessness, thermal strain, and affect) are of equal, if not greater, importance to anticipatory pace regulation and reserve capacity maintenance (Micklewright et al., 2010; Renfree et al., 2012; Stone et al., 2012; Jones et al., 2014; Pageaux, 2014; Williams et al., 2014, 2015). In particular, an individual's affective status has been suggested as a potentially more influential mediator of pace regulation than RPE (Baron et al., 2011; Jones et al., 2014; Renfree et al., 2014). On one hand, it would appear that the findings of the current study fail to support to this suggestion during multi-modal exercise, given the lack of statistically significant difference in affective response during each simulated triathlon. However, there was a *likely* meaningful trend for more positive levels of affect to be sustained throughout the quicker, more aggressive, and thus most physiologically demanding triathlon run, which followed the deceptively aggressive cycling condition. This would corroborate with the view that more negative affect is associated with reduced tolerance of physiological strain and poorer performance (Renfree et al., 2012), although it would also appear to disagree with the findings of Taylor and Smith (2014) which demonstrated more negative levels of affect throughout deceptively quicker, more aggressive, and thus more physiologically stressful, triathlon running. As such, it would seem that performance enhancement by deception may somehow be linked to an altered association between affective status and physiological strain, leading to a greater willingness to persevere with workloads that would otherwise be considered unsustainable.

However, given the difficulty in clearly distinguishing between the affective responses of each triathlon trial of the present study, it is evident that further research is required to confirm and better understand if, how, and why, someone's emotional status (i.e., levels of affect and arousal) may influence pace regulation more than "what" they are feeling (i.e., RPE, effort, thermal discomfort, breathlessness), particularly during multi-modal exercise. With this in mind, it may also be of value for researchers to examine whether the deceptive enhancement of both single and multi-modal performance reflects a change in the specific thoughts of participants, rather than an altered interpretation of common psychophysiological scales (Brick et al., 2016).

CONCLUSIONS

This study has shown that the imposition of deceptively aggressive cycle pacing, derived from previous "fastest possible" self-paced performance, enhances subsequent run and overall performance during simulated sprint-distance triathlon. It also suggests that interceptive sensations associated with fatigue and effort may be perceived differently according to an individual's expectations and beliefs regarding the past, present and future demands of pacing during multi-modal exercise. This would appear to be the case regardless of whether psychophysiological strain is established using RPE or by more distinct measures of interceptive sensations and emotions (i.e., sense of effort, perceived muscular pain, breathlessness, thermal strain, affect, and arousal). Whilst some form of anticipatory "template" may therefore be used by athletes to regulate the development of psychophysiological strain across a particular multi-modal exercise task, it would appear that the influence of afferent feedback on this process can be manipulated to modify pacing and enhance performance. Although these points echo previous conclusions (e.g., Taylor and Smith, 2014) this study demonstrates, for the first time, that the influence of manipulated task beliefs on the interaction between psychophysiological status and pacing can persist across consecutive modes of self-paced exercise, so as to optimize multi-modal performance. As such, it is hoped that the findings of the current study serve to catalyze the exploration and improved understanding of the anticipatory psychophysiological mechanisms which govern pace regulation across consecutive modes of exercise.

AUTHOR CONTRIBUTIONS

Both of the listed authors made a significant contribution to this study, including conceiving and designing the experiments (DT and MS), collecting, analyzing, and/or interpreting the data (DT and MS), conceptualizing and drafting the initial manuscript (DT), and critically reviewing/revising the manuscript (DT and MS). Further to these points, DT conducted the final approval of the version to be published (in agreement with MS) and is accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Deceptive Manipulation of Competitive Starting Strategies Influences Subsequent Pacing, Physiological Status, and Perceptual Responses during Cycling Time Trials

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Little is currently known regarding competitor influence on pacing at the start of an event and in particular the subsequent effect on the remaining distance. The purpose of the present study was to investigate the influence of starting pace on the physiological and psychological responses during cycling time trials (TT) utilizing an innovative approach allowing pace to be accurately and dynamically replicated, as well as deceptively manipulated. Ten competitive male cyclists completed five 16.1 km TT, two baseline trials performed alone (BLs), and three with a simulated, dynamic avatar of which they were to match the pace of for the initial 4 km. The avatar represented either the cyclist's fastest BL performance (NORM), 105% (FAST), or 95% (SLOW), of fastest BL performance (FBL). Physiological and psychological responses were measured every quartile of the TT. Despite manipulating a starting speed of ± 5% of fastest previous performance, there was no effect on overall 16.1 km TT performance. Manipulated starting strategies did however evoke different physiological and perceptual responses. Whole trial differences found that SLOW produced lower HR, VO_2, BLa and RPE than FBL ($p \leq 0.03$) and higher SE than FAST ($p \leq 0.03$). Additionally, FAST had greater internal attention than NORM ($p < 0.04$). Over time all psychological and physiological variables had a significant condition × quartile interaction in the initial or second quartile mediated by the prescribed starting strategies. Furthermore, RPE, affect, and internal attention remained elevated throughout FAST despite an attenuation in pace during self-selection of pace. There were no differences in performance time when manipulating a 16.1 km cycling TT starting strategy. A slow start, encouraged greater positive perceptions, and less negative physiological consequences than a faster start, and produces no impairment to performance time. It would therefore be considered an advantage in a non-drafting event, not to follow pace of fellow, superior competitors at the start of an event but perform a more negative pacing strategy, with the potential for a greater speed increase against opponents in the latter stages.

Keywords: pacing, time trials, deception, power output, perceived exertion, affect, self-efficacy

INTRODUCTION

Athletes select their starting strategy based on previous experience and task knowledge (Tucker, 2009; Tucker and Noakes, 2009; Smits et al., 2014; Williams et al., 2014b, 2015). Whilst this is the case during solo events, in the initial stages of a competitive race, athletes often do not self-select their pace, but rather adjust their speed to that performed by their opponents (Thiel et al., 2012). Although the athlete may initially envisage an overall pacing schema during an event, schemas are continuously modified in response to external factors such as opponents and tactics (Thiel et al., 2012; Thompson, 2015). Tactics represent dynamic decisions of how and when to invest energy (Smits et al., 2014), together with conscious choices to disrupt their opponent's performance (Thiel et al., 2012). Equally, decisions are made to alter work rate to ensure no harm to physiological status, or to avoid premature termination of the task (Micklewright et al., 2010; Thiel et al., 2012; Williams et al., 2014a). Often this is in response to a poor decision selecting unsustainable starting speeds and clearly supports the importance of interactive psychophysiological decision making (Swart et al., 2009).

Emotional responses play a key role in human decision making (Martino et al., 2006). The reaction to a competitor's movement is based on self-confidence and previous experience in a competitive situation (Foster et al., 1993; Wellner et al., 2010), integrated with the athletes' anxiety, motivation and excitement on the day of the event (St Clair Gibson et al., 2006). In cycling, changes in pacing strategy can significantly affect performance (van Ingen et al., 1992), specifically the exercise intensity elicited during the starting phase of an event (Mattern et al., 2001). It is not, however, well-understood which type of pacing strategy results in the best possible performance, as manipulating starting workloads has been investigated across athletic events of varying durations (Abbiss and Laursen, 2008).

Previous research has employed different distances or durations; <6 min (Aisbett et al., 2009; Bailey et al., 2011), 4–10 km (Gosztyla et al., 2006; Hausswirth et al., 2010; Taylor and Smith, 2014), >10 km (Mattern et al., 2001), and have been assessed within different modes of exercise; running (Gosztyla et al., 2006; Hausswirth et al., 2010), cycling (Mattern et al., 2001; Aisbett et al., 2009; Bailey et al., 2011; Hettinga et al., 2012), or multi-sports such as triathlon (Taylor and Smith, 2014). Furthermore, each have also used diverse magnitudes of increases and decreases in performance intensity (3–15%), and more importantly, they have employed averaged intensity manipulations. Whilst some have used average values from the initial start phases of a self-selected trial (Mattern et al., 2001; Gosztyla et al., 2006), others have included methods with limited ecological validity using whole-trial average manipulations (Aisbett et al., 2009; Hausswirth et al., 2010; Bailey et al., 2011; Hettinga et al., 2012; Taylor and Smith, 2014). The fixed pace nature of the starting strategy will produce conflicting results when compared to trials which are completely self-paced. Equally opponents do not follow a fixed pacing strategy and therefore athletes need to be reactive and responsive to dynamic changes in pace.

Previous research that has used exact, dynamic pacing profiles and investigated different pacing behaviors in both halves of a 4 km TT have found competitor influences (Konings et al., 2016). Observations of athlete behavior has demonstrated the display of intuitive behavior to follow a faster opponent in the opening stages of a race and that a deliberate decision to alter their pacing strategy to compete is evident. The presence of a competitor ostensibly alters the initial 4 km of an athlete's performance in a longer TT (16.1 km TT), whether through motivational, attentional focus (Williams et al., 2014a) or decision making influences (Williams et al., 2015). Previous investigations suggest that a reduced external focus has been observed in the initial 4 km and the final 4 km of a trial where competitors are present (Williams et al., 2014a). Whilst competitors influence attentional focus, it is also well documented that exercise intensity mediates the shift in attentional direction (Hutchinson and Tenenbaum, 2007).

Research is yet to investigate such competitor and intensity influence in a combined setting, specifically in the context of starting strategy manipulations. Knowledge of such effects could help identify the tolerable magnitude of performance increase at the start of an event and the influences on the remaining duration of an event. Furthermore, despite previous research employing starting strategy manipulations and the notion that competitors induce faster starting strategies (Tomazini et al., 2015; Williams et al., 2015), few have examined the perceptual responses of forced starting speeds, and their influence on the subsequent work-rate when able to self-select pace.

The aim of this study was to explore cyclists' responses to an opponent's pace at the start of an event, and specifically investigate the influence of such a starting strategy on the remaining task duration. This would be examined through performance and physiological effects, together with previously unexplored cognitive and perceptual responses. Additionally, the employment of visual avatars to follow as pacers, allowed an exact pacing replication of a previous starting strategy, rather than whole-trial, or starting strategy, average. In accordance with previous, similar literature it was hypothesized that the faster starting strategy would be debilitative to performance (Mattern et al., 2001) and increase negative perceptual responses (Williams et al., 2015).

MATERIALS AND METHODS

Participants

Ten competitive male cyclists with the following mean (SD) characteristics, age, 33 (7) year; body mass, 81.9 (6.2) kg; height, 180.1 (5.4) cm; peak power output, 4.8 (0.4) $W.kg^{-1}$; and $\dot{V}O_{2peak}$, 54.0 (3.2) $ml{\cdot}kg^{-1}{\cdot}min^{-1}$ participated in this study. Participants also had >2 years competitive cycling experience and current training volumes were >9 h per week. The institutional ethics committee approved the study and all participants gave informed consent before completing pre-exercise health screening.

Experimental Design

Participants visited the laboratory on six occasions performing a maximal oxygen uptake procedure and five 16.1 km TT's conducted using a repeated measures, counter balanced design. The trials were performed at the same time of day (±2 h) to

minimize circadian variation and were separated with 3–7 days to limit training adaptations. Participants were asked to maintain normal activity and sleep pattern throughout the testing period, and to replicate the same diet for the 24 h preceding each testing session. During the 24 h period prior to each trial, participants refrained from any strenuous exercise, excessive caffeine, or alcohol consumption. In the 2 h before each visit, participants consumed 500 ml of water and refrained from food consumption. Participants were informed that the study was examining the influence of visual feedback during TTs, and were fully debriefed regarding the true nature of the study upon completion of all trials (Jones et al., 2013).

Peak Oxygen Uptake

The first visit involved the maximal aerobic capacity test performed on a cycle ergometer (Excalibur Sport Lode, Groningen, Netherlands). Following a 5 min warm-up at 100 W, participants began the protocol at a prescribed resistance in accordance with accepted guidelines (British Cycling, 2003), and 20 $W.min^{-1}$ increments were applied until participants reached volitional exhaustion.. Continuous respiratory gas analysis (Oxycon Pro, Jaeger, GmbH Hoechburg, Germany) and heart rate (HR) (Polar Electro OY, Kempele, Finland) were measured throughout. HR_{peak}, VO_{2peak} and W_{peak} were calculated as the highest 30s average.

Time Trials

During the following five visits, participants performed a 16.1 km cycling TT on their own bike, mounted on a cycle ergometer (Computrainer Pro, Racermate ONE, Seattle, USA). Participants were informed that different feedback effects were being tested and instructed to complete each TT in the fastest time possible, preparing as if it were a genuine event. No verbal encouragement was given to the participants during any trial in order to prevent inconsistencies in the provision of feedback. Participants were fully debriefed as to the nature of the investigation once all trials had been completed.

The first two TTs were used to establish fastest baseline performance and to familiarize participants with the equipment procedures. Prior to each TT, participants completed a 5 min warm-up at 70% HR_{max}, determined from the maximal test, followed by 2 min rest. During the TT the ergometer was interfaced with 3D visual software and calibrated prior to each trial according to manufacturer's instructions. The visual display was projected onto a 230 cm screen positioned 130 cm away from the cyclist's front wheel, with the middle of the screen approximately eye level to the cyclists in a riding position. Whilst performing the initial 4 km during each trial the participants received visual feedback of a road as if they were performing on a flat, road-based 16.1 km TT course and their distance covered in km. Once they had reached 4 km the visual feedback of the road was removed and participants were only able to see their distance covered for the remaining 12 km.

The three final TTs were randomized and counterbalanced in order, with the initial 4 km of each performed with visual avatar, virtual road, current distance covered and distance between rider and avatar each displayed on the screen. Participants were instructed to keep pace with the avatar as closely as possible for the entire first 4 km section, after which the visual display would be removed and they should attempt to complete the remaining 12 km in the fastest time possible. One of the three experimental TTs was performed with an avatar which replicated the exact pacing strategy and speed the participant performed during their previous fastest baseline performance (NORM). A second trial displayed an avatar representing their fastest baseline pacing profile, but at a 5% greater speed (FAST), whilst the third experimental TT displayed an avatar with a speed 5% slower than each participant's fastest baseline pacing profile (SLOW). The manipulation was applied to the speed of the avatar at 34 Hz intervals in order to accurately replicate the exact FBL pacing strategy, ±5% in speed. The participants were not informed as to what the avatar's performance represented, only to follow them as closely as possible. They were reminded to increase their speed to stay with the avatar during the trial if the gap between themselves and the avatar was >10 m.

Experimental Measures

Power output, speed and elapsed time were obscured from the view of the participants throughout the TT, stored and each subsequently downloaded for analysis. Heart rate was also blinded and recorded continuously using the Polar team system sampled at 5 s frequencies and averaged as quartile data points for analysis. During each TT, breath-by-breath respiratory gasses were measured for the duration of a kilometer at every 4 km (e.g., 3.5–4.5 km), expressed in 5 s intervals and subsequently averaged for each quartile analysis. Finger-tip blood lactate (BLa) was also collected at the end of each 4 km quartile during the time trials. Participants were asked to remain in their usual cycling position whilst a capillary blood sample was procured from the finger-tip during the trial (Lactate pro Two LT-1730, Arkray, Japan).

During the initial 4 km participants were asked to rate their perceived exertion (RPE) on a 6–20 scale Borg scale (Borg, 1970), and their affect every kilometer. Affective feeling states (Hardy and Rejeski, 1989) indicating whether exercise felt pleasant or unpleasant, was measured using an 11-point Likert scale ranging from −5 to +5 with verbal anchors at all odd integers and zero (+5 = very good, +3 = good, +1 = fairly good, 0 = neutral, −1 = fairly bad, −3 = bad, –5 = very bad). For the following 12 km participants were asked to rate their RPE, affect, self-efficacy and attentional focus every 4 km. Participant's self-efficacy to continue at the current pace (SE_{pace}) was recorded on a 0–100% scale divided into 5% integer intervals. The scale was adopted as previously recommended (Bandura, 1977), with the questions constructed specific to the task due to perform. Attentional focus was recorded using a 10-point Likert scale (Tenenbaum and Connolly, 2008), with participants asked to indicate where their attention had been focused over the last kilometer in relation to external and internal thoughts. Attentional focus was also measured retrospectively, as a maintenance check, once the trial was completed. This was recorded as a percentage of attention that was focused on internal thoughts during different distances (whole-trial, 0–4, 4–8, 8–12, and 12–16.1 km).

Statistical Analysis

Normality was assessed using Shapiro-Wilk approach suitable for the sample size used. Paired *t*-tests were performed to analyse the presence of any systematic bias between the two baseline trials. Only the faster of the two baselines (FBL) was included in the inferential analysis. Six participants performed their fastest baseline in their first baseline trial and the four in their second baseline suggesting that any learning effect was not sufficient to significantly influence overall performance time. The effects of condition (FBL, NORM, FAST, SLOW) and distance quartile (0–4, 4–8, 8–12, and 12–16.1 km) on completion time, PO, speed, HR, RPE, affect, self-efficacy, attentional focus, blood lactate and VO_2 were analyzed using the Mixed procedure for repeated measures (Peugh, 2005). Various plausible covariance structures were assumed for each dependent variable and the one that minimised the Hurvich and Tsai's criterion (AICC) value was chosen as the best fitting and used for the final model. A quadratic term for distance quartile was entered into the model where appropriate and removed where no significance value was observed. *Post hoc* pairwise comparisons with Sidak-adjusted *p*-values were conducted where a significant F ratio was observed. Statistical significance was accepted as $p < 0.05$ (IBM Statistics 22.0; SPSS Inc., Chicago, IL).

RESULTS

Across all conditions there was no significant main effect for condition ($F = 0.8$, $p = 0.51$) observed for TT time (**Table 1**). There were no significant differences in time ($t_9 = 0.53$; $p = 0.6$), speed ($t_9 = -0.35$, $p = 0.7$), power output ($t_9 = -1.18$, $p = 0.3$), heart rate ($t_9 = 1.08$, $p = 0.3$), RPE ($t_9 = 0.0$, $p = 0.1$), affect ($t_9 = 0.32$, $p = 0.08$), self-efficacy ($t_9 = 1.18$, $p = 0.3$), or attention ($t_9 = -0.42$, $p \geq 0.07$) between the two familiarization TT.

Starting Strategy

There was a main effect for condition for initial 4 km time ($F = 769.5$, $p < 0.001$) with all conditions significantly faster than SLOW ($p < 0.001$) and all conditions significantly slower than FAST ($p < 0.001$). There was no significant difference between FBL and NORM (MD = −0.007, CL = −0.1, 0.9; $p = 1.0$) (**Table 2**). During the initial 4 km of the FAST and SLOW TT, participants actually rode at 3.6 ± 1.9% above and 5.0 ± 0.1% below fastest baseline speed, respectively. Two participants were unable to keep the <10 m gap during 2–4 km in the FAST trial.

Whole-Trial

Speed had a significant main effect for quartile ($F = 8.5$, $p = 0.006$) and a significant condition × quartile interaction ($F = 7.8$, $p < 0.001$), but no main effect for condition ($F = 1.5$, $p = 0.26$). The third quartile was significantly slower in speed than the second and fourth ($p \leq 0.005$). *Post hoc* analysis of the interaction effect illustrated that during the first quartile SLOW speed was significantly slower than FBL and FAST ($p \leq 0.02$). During the second quartile and third quartile SLOW was performed with a significantly faster speed than FAST ($p = 0.03$), and during the last quartile SLOW was performed at a significantly faster speed than FAST and NORM ($p \leq 0.01$) (**Figure 1A**).

TABLE 1 | Mean ± SD values for whole trial variables during each trial condition.

	FBL	NORM	FAST	SLOW
Time (mins)	26.6 ± 1.0	26.8 ± 1.2	26.5 ± 0.9	26.7 ± 1.1
Speed (km.h^{-1})	36.4 ± 1.4	36.0 ± 1.5	36.5 ± 1.2	36.2 ± 1.5
Power output (W)	259 ± 26	252 ± 28	260 ± 15	255 ± 26
Heart Rate (bpm)	161 ± 14	155 ± 14	159 ± 15	154 ± 16

TABLE 2 | Mean ± SD values for the initial quartile during each starting strategy conditions.

	FBL	NORM	FAST	SLOW
Time (mins)	6.6 ± 0.3*#	6.6 ± 0.3*#	6.4 ± 0.2*	6.9 ± 0.3#
Power output (W)	264 ± 29*	263 ± 29*	290 ± 28*	231 ± 25
Speed (km.h^{-1})	36.4 ± 1.4*	36.3 ± 1.4	37.7 ± 1.3*	34.6 ± 1.4
Bla (mmol.l^{-1})	7.3 ± 2.7*	6.4 ± 2.4*#	9.2 ± 3.2*	3.5 ± 1.1
Heart rate (bpm)	153 ± 12	150 ± 14	153 ± 13	140 ± 16
RER	1.15 ± 0.05	1.16 ± 0.06	1.19 ± 0.04	1.15 ± 0.04
$\dot{V}E$ (ml.min^{-1})	120.9 ± 27.9*#	123.4 ± 26.4*#	147.1 ± 28.8*	99.6 ± 17.8#
$\dot{V}O_2$ (ml·kg^{-1}·min^{-1})	44.2 ± 5.0*	43.7 ± 3.9*	45.9 ± 9.3	38.8 ± 4.1
Affect	0.45 ± 2.2	0.19 ± 1.8	−0.9 ± 1.7*	0.95 ± 1.6
Attention (%)	65.2 ± 31.2	27.5 ± 21.5	69.2 ± 28.1*	27.5 ± 23.7
RPE	16.6 ± 1.5*	16.0 ± 1.9	16.9 ± 1.8*	15.0 ± 1.8
SE (%)	82.5 ± 23.6#	85.5 ± 24.8#	57.5 ± 35.7	100.0 ± 0.0#

*Denotes significantly different to SLOW ($p < 0.05$); #denotes significantly ($p < 0.05$) different to FAST.

Power output had a significant main effect for quartile ($F = 6.8$, $p < 0.001$) and a significant condition × quartile interaction ($F = 14.7$, $p < 0.001$), however there was no main effect for condition ($F = 1.8$, $p = 0.2$). The third quartile was performed with significantly less power than the first and fourth ($p \leq 0.002$). *Post hoc* analysis of the interaction found that FAST, FBL and NORM, during the first quartile, had significantly greater power than SLOW ($p < 0.001$), but during the second quartile there was a significantly greater power performed during SLOW than FAST ($p < 0.02$).

Physiological Responses

There was a significant main effect for condition ($F = 5.2$, $p = 0.009$), quartile ($F = 41.9$, $p < 0.001$) and condition × quartile interaction ($F = 12.4$, $p < 0.001$) for HR. SLOW had a significantly lower HR than FBL (MD = 5.4, CL = 0.4, 10.8; $p = 0.03$) and FAST (MD = 3.6, CL = 0.7, 6.5; $p = 0.01$). There was a significantly lower HR in the first quartile than the remainder of the TT ($p < 0.001$). The interaction *post hoc* analysis illustrated during the first quartile SLOW was performed with a significantly lower HR than all other conditions ($p < 0.001$) (**Figure 1B**).

There was a significant difference in blood lactate between trials ($F = 10.8$, $p < 0.001$), with lower values produced during SLOW than FBL, NORM, FAST ($p \leq 0.002$). There was no significant main effect for quartile ($F = 1.2$, $p = 0.33$), however

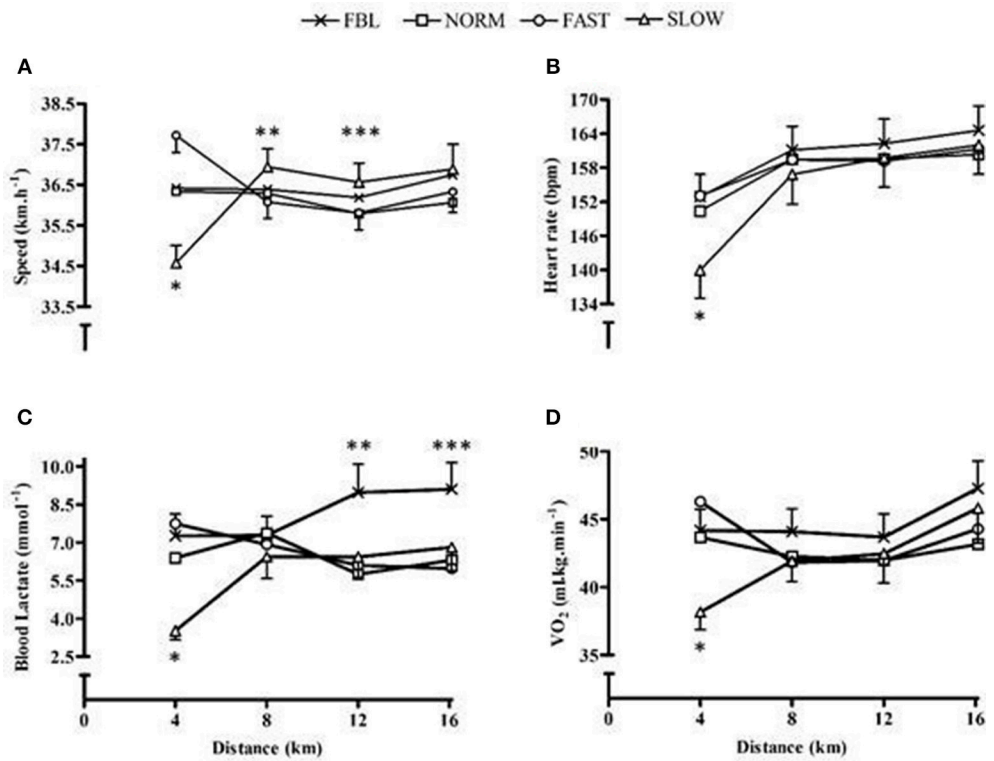

FIGURE 1 | Whole-trial mean and SEM physiological responses for each condition across distance quartiles, illustrating significant interaction effects. **(A)** Speed *denotes SLOW significantly slower than both FBL and FAST ($p \leq 0.018$), **denotes SLOW significantly faster than FAST ($p = 0.028$), ***denotes SLOW significantly faster than both NORM and FAST ($p \leq 0.01$); **(B)** Heart rate, *denotes significantly lower heart rate in SLOW than all other conditions ($p \leq 0.001$); **(C)** Blood lactate, * denotes significantly lower values during SLOW than all other conditions ($p \leq 0.02$), ** denotes significantly higher values in FBL than all other conditions ($p \leq 0.04$), ***denotes significantly higher values in FBL than NORM ($p = 0.02$); **(D)** $\dot{V}O_2$, *denotes significantly lower $\dot{V}O_2$ during SLOW than FBL and NORM ($p \leq 0.02$).

there was a significant condition × quartile interaction ($F = 3.8$, $p < 0.001$) (**Figure 1C**). A significant main effect for condition ($F = 0.01$, $p = 0.01$) and quartile ($F = 10.7$, $p < 0.001$) and a significant interaction ($F = 9.0$, $p < 0.001$) was identified for VE. FAST had a significantly greater VE than SLOW (MD = 12.9, CL = 2.8, 23.1; $p = 0.007$) and VE significantly increased over time ($p \leq 0.002$). The *post hoc* analysis for the interaction illustrated during the initial quartile FAST was significantly higher and SLOW was significantly lower than all NORM and FBL ($p \leq 0.001$). There was no significant main effect for condition for VO_2 ($F = 2.9$, p = 0.06), but a main effect for quartile ($F = 7.6$, $p = 0.001$) and a significant interaction effect ($F = 3.3$, $p = 0.008$) (**Figure 1D**). There was also a significant random intercept ($p = 0.04$). *Post hoc* analysis illustrated $\dot{V}O_2$ significantly increase over time ($p \leq 0.03$) and during the initial quartile of SLOW participant's $\dot{V}O_2$ was significantly lower than FBL ($p = 0.01$) and NORM ($p = 0.02$). RER values did not have a significant main effect for condition ($F = 1.2$, $p = 0.31$), however a significant main effect for quartile ($F = 8.2$, $p = 0.001$), a significant interaction effect ($F = 3.9$, $p = 0.004$) and a significant random slope ($p = 0.03$) identifying participants having different RER patterns. Pairwise comparisons of the interaction effect showed that during the second quartile FAST had a significantly lower RER than SLOW.

Psychological Responses

RPE had a significant main effect for condition ($F = 8.1$, $p = 0.001$), quartile ($F = 37.5$, $p < 0.001$) and interaction effect ($F = 2.5$, $p = 0.02$) (**Figure 2A**). There was a significantly greater RPE during FBL than NORM (MD = 0.6, CL = 0.04, 1.2; $p = 0.03$) and SLOW (MD = 0.9, CL = 0.08, 1.8; $p = 0.03$). During the initial quartile RPE was significantly lower in SLOW than FBL and FAST ($p \leq 0.002$). Affect was observed to have a significant main effect for quartile ($F = 11.8$, $p < 0.001$), with the final quartile having a significantly lower affect compared to the first and second quartile ($p \leq 0.005$) (**Figure 2B**). However, there was no main effect for condition ($F = 1.5$, $p = 0.24$) or an interaction effect ($F = 1.6$, $p = 0.17$).

Self-efficacy had a significant main effect for condition ($F = 10.7$, $p < 0.001$) and a significant condition × quartile interaction ($F = 3.5$, $p = 0.002$), but no main effect for quartile ($F = 1.4$, $p = 0.27$). *Post hoc* analysis found significantly lower SE during FAST than SLOW (MD = −16.9, CL = −25.9, −7.8; $p < 0.001$), and during the first quartile FAST has significantly lower SE than all conditions ($p \leq 0.001$) (**Figure 2C**). There was a main effect for condition for during-trial attentional focus ($F = 5.2$, $p = 0.005$). FAST had significantly greater internal attentional focus than NORM (MD = 16.0, CL = 1.0, 30.9; $p = 0.03$). There was a significant main effect for quartile ($F = 24.2$, $p < 0.001$) with the

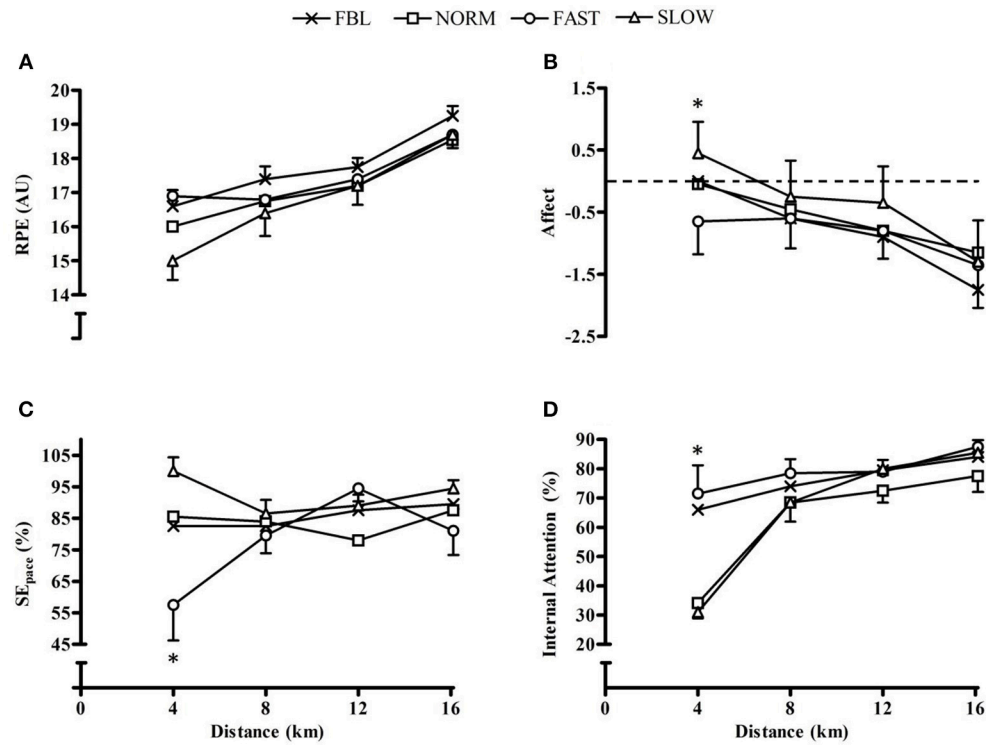

FIGURE 2 | Whole-trial mean and SEM psychological responses for each condition across distance quartiles, illustrating significant interaction effects. (A) RPE; **(B)** Affect, *denotes significantly lower affect in SLOW than both FBL and FAST ($p \leq 0.002$); **(C)** SE_{pace}, *denotes significantly lower SE_{pace} during FAST than all other conditions ($p \leq 0.001$); and **(D)** Attentional focus, *denotes significantly higher internal attention during FBL than NORM ($p = 0.003$).

first quartile having significantly lower internal attention than the other three ($p < 0.001$) and the fourth having significantly greater internal attention than all other quartiles ($p \leq 0.04$). There was also a significant interaction ($F = 2.1$, $p = 0.05$) as during the initial 4 km there was significantly greater internal attentional focus during FBL than NORM (MD = 31.8, CL = 9.6, 54.0; $p = 0.003$) (**Figure 2D**).

Post-trial attentional focus had a significant main effects for condition ($F = 4.2$, $p = 0.02$), quartile ($F = 18.3$, $p < 0.001$) and an interaction effect ($F = 7.7$, $p < 0.001$). There was significantly greater internal attentional focus during FAST than NORM (MD = 12.6, CL = 0.3, 25.1; $p = 0.04$). The first 4 km had significantly less internal attention than all other time points ($p < 0.001$). In the first 4 km FBL had greater internal attention than NORM (MD = 37.7, CL = 17.5, 57.9; $p < 0.001$) and SLOW (MD = 37.7, CL = 16.8, 58.6; $p < 0.001$). FAST had greater internal attention than NORM (MD = 41.7, CL = 21.5, 61.9; $p < 0.001$) and SLOW (MD = 41.7, CL = 21.5, 6.9; $p < 0.001$).

DISCUSSION

This study was the first to represent the influence of following a dynamic pace enforced by a fellow cyclist during the opening stages of a 16.1 km TT, and its influence on subsequent pace and previously unexplored perceptual responses. Enforcing manipulated starting speeds of ± 5% of FBL did not affect overall 16.1 km TT performance. Although performances were not significantly altered, pacing strategy decisions, physiological and psychological responses were different and dependent on the starting intensity. As prescribed by the avatar's pace in the initial 4 km performance, data confirm that different starting strategies were effectively enforced during manipulated TTs with significantly slower speeds performed in the initial 4 km during SLOW compared to FBL and FAST. Performing the accurate BL starting strategy (NORM), in comparison to FBL, resulted in no differences in performance or physiological responses, and elicited comparable RPE and affect, however, different psychological responses were observed (self-efficacy and attentional focus). Performing a slower start was associated with a lower RPE than all other starting strategies and a more negative affect compared to the faster start. Such response was observed, not only in the initial 4 km, but also for the whole trial, reflecting the relationship between RPE and intensity (St Clair Gibson et al., 2006). This importantly illustrates that the effects of starting strategy intensity on the RPE and perceptions during the remainder of a TT of this distance.

Although changes in starting strategies produced no differences in overall completion time, speed or power between trials, the subsequent pacing profiles for each trial differed depending on the relative speed maintained during the initial 4 km. During SLOW, participants produced greater speed during the remaining 12 km than all other starting condition trials. In contrast, during FAST, participants decreased their speed during the second quartile, significantly slower than the SLOW

trial. These results demonstrated that cyclists made a decision, in both conditions, to change their pace after a forced starting strategy (Renfree et al., 2014; Smits et al., 2014). Cyclists were unable to maintain the elevated speed required during FAST and therefore had to reduce the intensity during the remaining 12 km by 4.5%, in comparison to an increase in the remaining 12 km during SLOW; 2.1% and NORM trials; 2.3%. This emphasizes differences in exertional decision making during competition if athletes are to deviate away from typical, optimal pacing strategies, particularly at the start.

During FAST, the initially elevated physiological responses (HR, VE, and BLa) and RPE, were attenuated after the starting 4 km, but still produced significantly higher values overall compared to SLOW. Previous research has suggested that such responses in the initial quartile could have had a prolonged effect on the remaining duration, with participants unable to recover adequately during the trial (Mattern et al., 2001; Hettinga et al., 2012). This could explain the present study's fast start not facilitating overall performance, despite decreasing performance time during the initial 4 km. Additionally, the observed significantly greater internal attentional focus could have been induced through conscious attempts to regulate effort. Moreover, an increase in starting speed producing lower self-efficacy, could have been due to uncertainty, either linked with limited prior experience of such a starting pace, or concern over elevated physiological feedback and its potential negative effect. This suggests that whilst pace and performance declined when participants were able to self-select workload, the subsequent cognitive, perceptual and physiological responses were arbitrated by the responses to the initial enforced pace (Mattern et al., 2001). The present results also suggest that a 5% increase in self-selected intensity at the start of a 16.1 km TT is too great for the level of rider used in this particular study to sustain. This pace manipulation was unable to be performed with the average increase in pace in FAST at 3.6%. This is important to note for future deception manipulations and particularly stresses the difference in this manipulation to those using fixed power or speed. Whilst using fixed paced manipulation would avoid the variation in manipulation observed in the current study. Using fixed, less ecologically valid methods may be more exposed to athletes detecting the deception and perhaps would lead to greater negative changes in pace, greater physiological disturbance, and worse psychological feeling states in the subsequent duration of the event. Equally leaving the choice to the participant as to whether they would follow the pace of the lead opponent would also increase variations in starting intensities and limit the overall effects found on subsequent pace.

The present study enhances knowledge regarding the influences of performing an initial pace enforced by competition and the interesting psychological processes associated with different starting stratgies; but they dispute previous proposals of a debilitative conservative starting pace (Aisbett et al., 2009; Lima-Silva et al., 2010; Bailey et al., 2011; Hettinga et al., 2012). It has been thought that a conservative starting pace increases the risk of not producing an optimal completion time (Smits et al., 2014), and it has also been suggested that it can be a high-risk strategy to not follow superior competitors at the start of a race (Renfree et al., 2014). The present results demonstrate, that there is no detriment to completion time during a 16.1 km TT if a 5% slower initial speed is adopted in the initial starting phase. This study shows that an initial slower start decreases the initial accumulation of metabolites and the heightened physiological responses allowing workload to be increased during the remaining self-paced 12 km. This decision to increase pace, possibly due to lower physiological strain (HR, $\dot{V}O_2$, VE, BLa), and the more positive psychological responses (reduced RPE and internal attention, and improved affect and self-efficacy), lessens the effect of a slower start. This not only enables a similar performance time, but participants also continued to have more positive cognitive and perceptual responses during the remainder of the trial. Such findings promote the advantages of a slow start in non-drafted TTs and highlight important associations with athlete motivation, ability and likelihood of producing a greater endspurt in an event, perhaps when opponents are experiencing more fatiguing symptoms. It would be of interest to investigate such manipulation effects during shorter or longer duration events where there is less or more distance to catch-up after a slower start. Similarly, they offer training and future performance implications suggestive of athlete's adherence to high intensities whether through enjoyment or motivation if the initial workload is lowered. Of interest would be the investigation into this effect during an event which provides efficiencies from drafting a competitor, whereby a similar manipulation could explore at what intensity would the efficiencies gained from drafting not warrant the extra energy to keep with your opponent.

A further aim of this work was to expand on previous research investigating the presence of a competitors influencing attentional focus during exertion (Williams et al., 2014a). Reductions in internal attentional focus were previously shown to inhibit the rise in perceived exertion during performance in the presence of competitors. Differences seen in RPE and internal attentional focus in the initial 4 km between FBL and NORM using the same pacing profile, in the present study, support the previous investigation. Additionally, exercise intensity has been proposed as a mediator of attentional focus (Hutchinson and Tenenbaum, 2007), and this was also observed in the present results, since the presence of a faster avatar and the prescribed increase in intensity, was insufficient to draw attention externally and failed to prevent a rise in perceived exertion. Furthermore, in the presence of a slower avatar, internal attentional focus and RPE were significantly reduced compared to no avatar, or a faster avatar. This could however be because the riders were not asked to compete with the avatar, but to simply match its pace. The importance of instructions may explain differential results to previous research since, the impact of a competitive environment is likely to have additional influences (Schunk, 1995), other than a visual display that would encourage an external focus. Motivational processes have been previously explored in specific events that serve to direct attentional focus toward sources of information, from which it was observed that the motivational influence on attention mechanisms, adaptively regulates perceptual and conceptual processes (van der Linden and Eling, 2006; Williams et al., 2014b). This has been previously found in an experiment with athletes perceiving a great

performance when deceived to be performing in the presence of a non-competitive, experimental accomplice (Bath et al., 2012). In addition to the present investigation, motivation could be further explored as to whether its influence changes at different stages of a competition or task duration (i.e., start or end of the trial) or within competitive and non-competitive scenarios.

CONCLUSION

These results suggest that with no detriment to performance time, but less physiological strain and more positive psychological perceptions, a pacing strategy adopting a slower start could be considered more beneficial during a stimulated 16.1 km cycling TT. Despite beginning the TT with a conservative pace, resulting in a performance disadvantage of ∼18 s, participants were able to overcome this deficit, when they self-selected their subsequent pace. Not only were they able to produce a similar completion time, but also had more positive perceptual responses; reduced RPE and greater affect. Similarly, whilst the view is that attention and affect are dynamic in the face of task progression, the result suggest during high-effort tasks, non-preferable changes in such state may be difficult to recover. This perhaps indicates the necessity for directed cognitive interventions within-task to aid the reversal of the detrimental psychological responses accompanying a fast start.

Practically, these findings highlight the possible importance of not following a pacing strategy that is influenced by lead competitors when unable to gain efficiencies through drafting, since the detrimental impact could be prevented and still enable an equivalent performance. The ability to dictate an early pace on fellow competitors may be beneficial through the impact on cognitive and perceptual responses and their associated influence on pacing decisions. Importantly, a chosen starting strategy has residual influences on the remaining distance, with both physiological responses and perceptual valence determined by the prescribed starting strategies.

AUTHOR CONTRIBUTIONS

Each author (EW, HJ, SS, DM, CB, AM, LM) contributed to the conception or design of the work. EW, HJ and AM contributed to the analysis and EW, HJ, AM, SS, DM, CB, LM the interpretation of data for the work. Each author (EW, HJ, SS, DM, CB, AM, LM) contributed to the drafting the work and revising it critically for important intellectual content. Each author (EW, HJ, SS, DM, CB, AM, LM) contributed to the final approval of the version to be published; and Each author (EW, HJ, SS, DM, CB, AM, LM) is in agreement to be accountable for all aspects of the work.

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Improvements in Cycling Time Trial Performance Are Not Sustained Following the Acute Provision of Challenging and Deceptive Feedback

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The provision of performance-related feedback during exercise is acknowledged as an influential external cue used to inform pacing decisions. The provision of this feedback in a challenging or deceptive context allows research to explore how feedback can be used to improve performance and influence perceptual responses. However, the effects of deception on both acute and residual responses have yet to be explored, despite potential application for performance enhancement. Therefore, this study investigated the effects of challenging and deceptive feedback on perceptual responses and performance in self-paced cycling time trials (TT) and explored whether changes in performance are sustained in a subsequent TT following the disclosure of the deception. Seventeen trained male cyclists were assigned to either an accurate or deceptive feedback group and performed four 16.1 km cycling TTs; (1 and 2) ride-alone baseline TTs where a fastest baseline (FBL) performance was identified, (3) a TT against a virtual avatar representing 102% of their FBL performance (PACER), and (4) a subsequent ride-alone TT (SUB). The deception group, however, were initially informed that the avatar accurately represented their FBL, but prior to SUB were correctly informed of the nature of the avatar. Affect, self-efficacy and RPE were measured every quartile. Both groups performed PACER faster than FBL and SUB ($p < 0.05$) and experienced lower affect ($p = 0.016$), lower self-efficacy ($p = 0.011$), and higher RPE ($p < 0.001$) in PACER than FBL. No significant differences were found between FBL and SUB for any variable. The presence of the pacer rather than the manipulation of performance beliefs acutely facilitates TT performance and perceptual responses. Revealing that athletes' performance beliefs were falsely negative due to deceptive feedback provision has no effect on subsequent perceptions or performance. A single experiential exposure may not be sufficient to produce meaningful changes in the performance beliefs of trained individuals beyond the acute setting.

Keywords: feedback, previous performance, pacing, deception, cycling, endurance performance, self-efficacy

INTRODUCTION

During self-paced endurance exercise, athletes will adopt a pacing strategy in the endeavor to produce an optimal performance whilst preventing the occurrence of premature fatigue (Abbiss and Laursen, 2008; Hettinga et al., 2012). Performance-related feedback from external sources is interpreted in accordance with the current pace, internal physiological feedback and the task goals. This integration of information will then govern the continuous and dynamic process of during-task decision-making regarding pace (Renfree et al., 2014; Smits et al., 2014). When external feedback is interpreted in relation to an individual's beliefs in their ability to meet the task demands, it has the potential to elicit positive or negative perceptual experiences (Hutchinson et al., 2008) and performance outcomes (Halson and Martin, 2013). Furthermore, strong beliefs regarding an individual's abilities in their performance have been positively associated with motor performance (McKay et al., 2012), maximal force production (Ness and Patton, 1979; Kalasountas et al., 2007), running efficiency (Stoate et al., 2012), effort tolerance (Hutchinson et al., 2008), and positive affect (McAuley and Courneya, 1992; Stoate et al., 2012).

Previous experience, and the appraisal of the success/failure of this prior effort, will significantly influence an individual's beliefs in their abilities to meet the demands of a similar, future task (Bandura, 1997; Sitzmann and Yeo, 2013). In addition to efficacious beliefs, this prior experience is also considered to be a key determinant of pacing strategies in endurance performance (Micklewright et al., 2010). Therefore it is of interest to explore how prior performance beliefs can be manipulated to enhance self-efficacy and improve future performances. To date, however, few studies have manipulated these beliefs and assessed the residual effects in future exercise bouts. Deception is one method by which these beliefs can be manipulated in order to explore the effects on pacing decisions and performance in self-paced exercise (Jones et al., 2013). For example, the provision of false external feedback prior to or during an exercise bout allows self-beliefs to be surreptitiously augmented in order for behavior to be examined without the influence of unwanted expectancies.

Deceptive conditions present situations of challenge or threat, as described by the Biopsychosocial (BPS) model or, within this exertive context, the Theory of Challenge and Threat States in Athletes (TCTSA) (Blascovich et al., 2004; Jones et al., 2009). These theories propose that threat states occur when an individual perceives that the demands of the task cannot be met by available resources. The nature of deception means that either knowledge of the exercise duration/distance is surreptitiously manipulated (i.e., task demands), or individuals are unknowingly misled as to the level of their own ability (i.e., resources). An example of the latter can be seen in a recent study where deceptive feedback was used to manipulate cyclists' knowledge of their prior time trial performance (Jones et al., 2016). The presence of a visual avatar that unknowingly represented a performance 2% faster than the athletes' baseline effort created a threatening situation for the self. Although performance improved in comparison to the baseline, this improvement was of an equal amount to athletes who knowingly rode against an avatar of their exact baseline performance. This control group, however, may not have experienced a challenge state if the magnitude of the feedback provided did not encourage a motivational focus on success (Blascovich et al., 2004). Challenge states have previously elicited enhanced performances, therefore providing athletes with accurate knowledge of challenging feedback, for example a 2% faster avatar, may reveal the extent to which deception alone may influence performance.

Challenge and threat states have also been shown to influence cognitive processes as well as behavior, where an individual's motivation may be focussed on success in a challenge state and on the prevention of failure in a threat state (Blascovich et al., 2004). These positive and negative motivational states have been found to influence effort, emotions, decision-making, efficacious appraisals and physiological responses (Skinner and Brewer, 2002; Meijen et al., 2013; Vine et al., 2013) and could therefore explain previous findings of altered perceptual responses, such as RPE and affect, in deceptive conditions (Stone et al., 2012; Jones et al., 2016). In the previously mentioned study by Jones et al. (2016), the deception group experienced more negative affect and higher perceived exertion than the control group which supports these aforementioned patterns within threat states. Identifying what factors drive the nature of these perceptual and behavior changes will allow for a greater understanding of the effects of deceptive, or threatening, feedback and its potential application as a training tool.

Most prior deception research has investigated within-trial acute effects of this feedback provision, therefore the residual effects of deceptive interventions are relatively unknown. According to self-efficacy theory (Bandura, 1977), success in a previous performance is thought to most greatly strengthen efficacious perceptions and bring about behavior change (Hutchinson et al., 2008). In instances where deceptive feedback elicits an improvement in performance, unless the athlete can knowingly and accurately appraise their performance as being successful, self-efficacy and resultant behavior changes may not be sustained in future efforts. This is also reverberated by the theory of Psychological Momentum, where expectations of win/loss outcomes are determined as a function of recent successes or failure (Hubbard, 2015). Due to the very nature of deception, individuals may not be explicitly aware that they have performed beyond what they believed possible if the deception remains concealed prior to a future performance. This is supported by the absence of a performance change in a subsequent trial following the provision of deceptive feedback in Jones et al.'s (2016) study, although efficacious experiences were more positive. It is therefore of interest to explore how the disclosure of a deceptive intervention, and the conscious and accurate appraisal of the outcome of a previous performance, may influence self-efficacy and performance in a subsequent performance.

A recent study assessed the residual effects of deception by revealing the true nature of the deception to participants prior to a subsequent trial (Shei et al., 2016). Cyclists were provided with false feedback of a baseline time trial performance using a visual avatar to unknowingly represent 102% of their average baseline power output. Participants then performed a

subsequent TT following the disclosure of the deception where, again, they performed against the 102% avatar but this time with accurate knowledge. The resultant performance times were faster in the deception and subsequent TTs compared to the baseline. The authors concluded that the improved performance in the deception trial could be sustained after participants had been informed of the deception. What Shei et al. (2016) did not acknowledge however, is that the mere presence of the avatar in both TTs was likely to have had a motivational influence and could alone explain the faster performances (Williams et al., 2014, 2015; Jones et al., 2016). The lack of a control group and the use of a static avatar set as a depiction of the average baseline power output further limits the study, as indeed it also does in some other deception research (Stone et al., 2012).

The first aim of the present study was to investigate the effects of challenging vs. threatening performance feedback on perceptual responses and performance in 16.1 km self-paced cycling TTs. Secondly, we aimed to explore the residual effects of this acute feedback provision, following the correction of false beliefs incurred via deception. It was hypothesized that (1) performance would be improved with the presence of performance feedback, regardless of feedback accuracy, and (2) the deception group would maintain the performance improvement in a subsequent trial and experience more positive perceptual responses following the disclosure of the deception.

METHODS

Participants

Seventeen trained male cyclists with race experience in 16.1 km TTs volunteered for the study. Match-paired, random allocation was used to allocate participants to either an accurate (ACC; $N = 9$) or deceptive (DEC; $N = 8$) feedback group based on VO_{2peak} values and performance times attained in TT1 (**Table 1**). Participants provided prior written informed consent in accordance with the Declaration of Helsinki and the study was approved by Edge Hill University's research ethics committee. Participants were classified as "trained" according to VO_{2peak} and peak power output values (De Pauw et al., 2013).

TABLE 1 | Mean (SD) descriptive data for the ACC and DEC experimental groups.

	ACC group ($N = 9$)	DEC group ($N = 8$)
Age (yrs)	33.0 (6.0)	37.9 (6.5)
Height (cm)	180.0 (3.1)	178.5 (6.7)
Body mass (kg)	77.2 (5.9)	79.4 (5.4)
Absolute PPO (W)	371 (35)	380 (24)
Relative PPO (W/kg)	4.8 (0.5)	4.8 (0.4)
Absolute VO_{2peak} ($L \cdot min^{-1}$)	4.1 (0.4)	4.2 (0.3)
Relative VO_{2peak} ($mL \cdot kg \cdot min^{-1}$)	54.1 (5.9)	53.3 (4.4)

PPO, peak power output; VO_{2peak}, peak oxygen uptake.

Research Design

A 2×3 (group $\times$ trial) between- and within-subject experimental design was adopted and participants visited the laboratory on five separate occasions, 2–7 days apart and at the same time of day (± 2 h). All visits were completed within a 3 week period and the final trial was completed no more than 7 days after the penultimate visit. A maximal incremental test was completed on the first visit, before both groups completed four 16.1 km cycling TTs (**Figure 1**).

Maximal Incremental Test

Height and body mass were recorded on the participants' initial visit followed by a continuous incremental ramp test to maximal exertion on a cycle ergometer (Excalibur Sport, Lode, Groningen, The Netherlands) to determine VO_{2peak}. A 5 min warm-up was performed at 100 W and then initial workloads were determined using established guidelines (Wooles et al., 2003). Increments of 20 W were applied every minute until the required power output could no longer be maintained. Breath-by-breath pulmonary ventilation and gas exchange data were measured throughout the test (Oxycon Pro, Jaeger, GmbH, Hoechburg, Germany) to determine oxygen consumption, which was normalized to pre-exercise body mass data. The VO_{2peak} was defined as the highest VO_2 value recorded over a 20 s period. Heart rate (Polar Team System, Finland) was recorded continuously using a 5 s sampling rate and verbal encouragement was provided.

Experimental Trials

All participants performed four self-paced 16.1 km TT on their own bicycles, using a calibrated electromagnetically-braked cycle ergometer (CompuTrainer Pro^{TM}, RacerMate, Seattle, USA); previously shown to be a reliable measure of power output (Stone et al., 2011). A 0.6% coefficient of variation was found in our laboratory for between-trial variation in performance times ($n = 31$) and a 0.6% smallest worthwhile change in road TT performance has been previously reported (Paton and Hopkins, 2006). The first two TTs (TT1, TT2) were used for familiarization, but to prevent sub-maximal efforts being produced, participants were not informed of this. A flat, virtual course was projected onto a 230 cm screen in front of the rider by the ergometry software, which depicted the participants' speed profile as a synchronized graphical avatar. Time and power output were recorded at a rate of 34 Hz, but distance covered was the only variable displayed. After a 10 min warm-up at 70% of HR_{max}, the drafting option in the software was disabled and participants were instructed to complete each TT in the fastest time possible.

Each individual's fastest performance from the two baseline trials was classified as their "fastest baseline" (FBL) and used in all subsequent analysis. In the third TT (PACER), the software represented each participants' FBL performance profile on the screen as a pacer alongside their current performance, depicted as a dynamic and exact replication of the FBL speed profile (**Figure 2**). In addition to total distance covered, the distance between the participants' avatar and the pacer was also displayed onscreen for both groups. Participants in the ACC group were correctly informed that this pacer was 2% faster than their own FBL performance. In contrast, the pacer in the DEC group

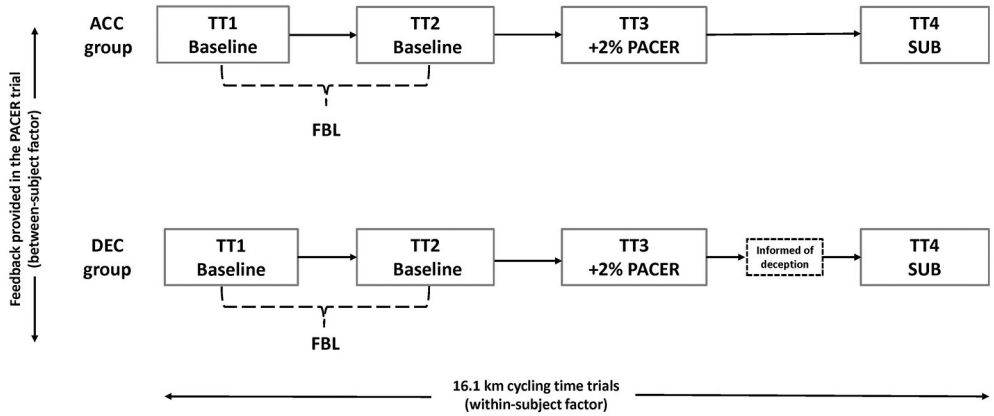

FIGURE 1 | Trial schematic of the research design.

FIGURE 2 | Representation of the visual feedback provided in the PACER TT.

also represented a performance corresponding to 2% faster than their FBL but participants were told that it was an accurate representation of their FBL performance. On the final visit, a subsequent TT (SUB) was performed, which was an exact replication of the FBL procedures with no pacer in either group and distance covered feedback only. Immediately before participants in the DEC group commenced their SUB TT, they were informed of the true nature of the pacer that they had performed with in their previous trial. Identical information was given verbally to each participant which stated that the pacer had not represented their fastest baseline TT but had in fact been set 2% faster. No other feedback relating to their performances was provided.

Perceptual Responses

Participants were fully briefed with the instructions for the use of affect, RPE and self-efficacy scales. Affect was measured using the validated 11-point Feeling Scale ranging from +5 (very good) to −5 (very bad) (Hardy and Rejeski, 1989). Participants were informed that their responses should reflect the affective or emotional components of the exercise and not the physical sensations of effort or strain. Borg's (1970) 6–20 scale was used to measure RPE and for task-specific self-efficacy, participants reported "*how confident are you to continue at your current pace for the remaining distance of the trial?*" using a percentage scale from 0% (*cannot do at all*) to 100% (*absolutely certain can do*) (Bandura, 1997; Welch et al., 2010). Verbal responses for affect, RPE and self-efficacy were recorded every 4 km during each TT.

Physiological Variables

Heart rate was measured continuously and respiratory gas analysis recorded expired air every 4 km. Fingertip capillary blood lactate (BLa; Lactate Pro 2, LT-1730, Arkray, Japan) was analyzed prior to each trial and at 4 km intervals.

Statistical Analysis

Linear mixed modeling was used to explore the effects of distance (4, 8, 12, and 16.1 km), trial (FBL, PACER, SUB) and group (ACC, DEC) on all repeated-measures dependent variables; power output, speed, affect, RPE, self-efficacy, heart rate, BLa, V_E, VO_2 and RER. Distance, trial and group were modeled as fixed effects and participant as a random effect. Distance was modeled as a continuous variable where linear or quadratic responses were evident, and otherwise modeled as a categorical variable where saturated means modeling was most appropriate. Various plausible covariance structures were assumed, with the structure that minimized the Hurvich and Tsai's criterion (AICC) value chosen for the final fitted model. Performance times were analyzed with fixed effects included for trial and group. Differences between all dependent variables in TT1 and TT2 were analyzed using paired *t*-tests. In the event of significant fixed main or interaction effects, post hoc comparisons with Sidak adjusted *P* values were used to identify significant differences between paired means. Two-tailed statistical significance was accepted as $P < 0.05$ and analyses were conducted using IBM SPSS Statistics 22 (SPSS Inc., Chicago, IL). Descriptive sample statistics are reported as mean and standard deviation (SD) and confidence intervals (CI) are reported at the 95% level. Effect sizes

are reported using Cohen's d to indicate the magnitude of the differences between means.

RESULTS

Performance Variables

Performance times for the ACC group in FBL, PACER and SUB were 26:31 (1:44), 26:15 (1:31), and 26:40 (1:30) min, respectively. For the DEC group, performance times were 26:40 (0:52), 26:22 (0:44), and 26:34 (0:54) min. Performance times between trials were significantly different ($F = 4.9$; $P = 0.015$), with pairwise comparisons indicating that PACER was performed in a significantly faster time than FBL (mean difference (MD) $= -17$ s; CI $= -0.55, -0.01$; $P = 0.042$, $d = 0.20$) and SUB (MD $= -19$ s; CI $= -0.59, -0.03$; $P = 0.027$, $d = 0.14$; **Table 2**). Performance time in SUB was not significantly different to FBL (MD $= 2$ s; CI $= -0.24, 0.30$; $P = 0.99$, $d = 0.07$). There was not a significant group x trial difference ($F = 0.7$; $P = 0.49$), therefore the differences in performance times between trials were similar in both the ACC and DEC groups.

Significant main effects for power output were found for distance ($F = 91.9$; $P < 0.001$) and trial ($F = 9.2$; $P < 0.001$). Post hoc analysis revealed that PACER was performed at a significantly higher power output than both FBL (MD $= 7$ W; CI $= 3.17, 10.70$; $P < 0.001$; $d = 0.22$) and SUB (MD $= 8$ W; CI $= 4.34, 12.03$; $P < 0.001$; $d = 0.27$). Similarly, main effects for speed were found for distance ($F = 29.9$; $P < 0.001$) and trial (Speed: $F = 7.0$; $P = 0.001$). Speed was significantly higher in PACER than both FBL (MD $= 0.4$ km·hr^{-1}; CI $= 0.16, 0.59$; $P < 0.001$; $d = 0.20$) and SUB (MD $= 0.4$ km·hr^{-1}; CI $= 0.19, 0.64$; $P < 0.001$; $d = 0.13$).No significant group × trial interactions were found for power output or speed (PO: $F = 0.4$; $P = 0.69$, Speed: $F = 0.3$; $P = 0.72$) indicating that pacing strategies in each trial were similar between the ACC and DEC groups (**Figure 3**).

Perceptual Responses

Significant main effects for affect were found for distance ($F = 16.3$; $P < 0.001$) and trial ($F = 4.5$; $P = 0.02$), with significantly lower affect in PACER than FBL (MD $= -0.69$; CI $= -1.28, -0.11$; $P = 0.016$, $d = 0.95$; **Figure 4A**). Main effects for RPE were similarly found for distance $F = 14.2$; $P < 0.001$) and trial ($F = 4.6$; $P = 0.012$). RPE in PACER was significantly higher than in FBL (MD $= 0.7$; CI $= 0.34, 1.04$; $P < 0.001$, $d = 0.36$) and SUB (MD $= 0.4$, CI $= 0.07, 0.78$; $P = 0.014$, $d = 0.23$; **Figure 4B**). For self-efficacy, significant group ($F = 4.9$; $P = 0.042$) and trial ($F = 8.9$; $P = 0.001$) main effects were found, showing that the DEC group were significantly less confident than the ACC group (MD $= -14.2\%$; CI $= -27.81$, -0.55; $P = 0.042$, $d = 2.61$). Self-efficacy was lower in PACER than FBL (MD $= -7.6\%$; CI $= -13.76, -1.48$; $P = 0.011$, $d = 0.97$) and SUB (MD $= -10.0\%$; CI $= -16.13, -3.82$; $P = 0.001$, $d = 1.22$; **Figure 4C**).

Physiological Variables

Significant main effects for heart rate were found for trial ($F = 7.5$; $P = 0.002$) and distance ($F = 57.7$; $P <$

TABLE 2 | Mean (SD) physiological responses at each distance quartile in 16.1 km time trials for the ACC and DEC groups.

	ACC group				DEC group			
	4 km	**8 km**	**12 km**	**16.1 km**	**4 km**	**8 km**	**12 km**	**16.1 km**
HEART RATE (beats·min^{-1})								
FBL	157 (14)	164 (14)	167 (14)	169 (13)	145 (8)	154 (13)	157 (14)	160 (14)
PACER	160 (9)	169 (10)	170 (11)	172 (10)	147 (9)	158 (12)	160 (13)	162 (13)
SUB	155 (14)	163 (13)	164 (12)	167 (12)	145 (8)	155 (11)	157 (12)	160 (13)
V_E (L·min^{-1})								
FBL	120.5 (28.3)	121.4 (30.7)	120.0 (31.0)	138.0 (35.4)	127.5 (33.1)	127.2 (33.8)	127.0 (33.9)	151.6 (32.1)
PACER	131.5 (30.9)	132.4 (35.7)	136.7 (38.7)	143.4 (37.4)	136.9 (35.7)	137.8 (30.6)	137.0 (29.0)	154.5 (21.4)
SUB	120.9 (22.9)	117.5 (25.8)	120.2 (31.6)	147.6 (34.0)	125.6 (25.9)	126.8 (19.4)	125.8 (19.8)	147.1 (23.4)
VO_2 (L·min^{-1})								
FBL	3.5 (0.5)	3.5 (0.6)	3.4 (0.6)	3.6 (0.6)	3.6 (0.4)	3.5 (0.5)	3.4 (0.5)	3.7 (0.5)
PACER	3.7 (0.5)	3.6 (0.6)	3.5 (0.6)	3.7 (0.6)	3.7 (0.2)	3.6 (0.4)	3.5 (0.3)	3.7 (0.2)
SUB	3.5 (0.4)	3.4 (0.5)	3.4 (0.5)	3.8 (0.5)	3.5 (0.2)	3.5 (0.3)	3.5 (0.3)	3.7 (0.3)
RER								
FBL	1.11 (0.04)	1.12 (0.04)	1.11 (0.04)	1.15 (0.08)	1.12 (0.08)	1.14 (0.08)	1.13 (0.07)	1.19 (0.09)
PACER	1.19 (0.05)	1.15 (0.04)	1.14 (0.03)	1.16 (0.04)	1.20 (0.10)	1.16 (0.10)	1.14 (0.10)	1.18 (0.10)
SUB	1.13 (0.06)	1.09 (0.07)	1.08 (0.07)	1.16 (0.10)	1.21 (0.05)	1.17 (0.04)	1.15 (0.05)	1.20 (0.06)
BLa (mmol·L^{-1})								
FBL	7.8 (3.3)	8.9 (3.0)	8.7 (2.9)	9.1 (3.3)	10.5 (3.7)	10.4 (4.4)	11.1 (1.5)	10.8 (5.1)
PACER	8.9 (2.4)	8.9 (3.3)	9.4 (3.9)	9.7 (3.5)	11.4 (4.6)	12.1 (5.1)	12.3 (5.3)	12.2 (4.1)
SUB	6.7 (2.6)	6.1 (3.3)	6.6 (4.2)	9.3 (4.6)	10.7 (4.6)	10.7 (4.9)	10.4 (4.8)	11.0 (4.2)

V_E, minute ventilation; VO_2, pulmonary oxygen uptake; RER, respiratory exchange rate; BLa, blood lactate.

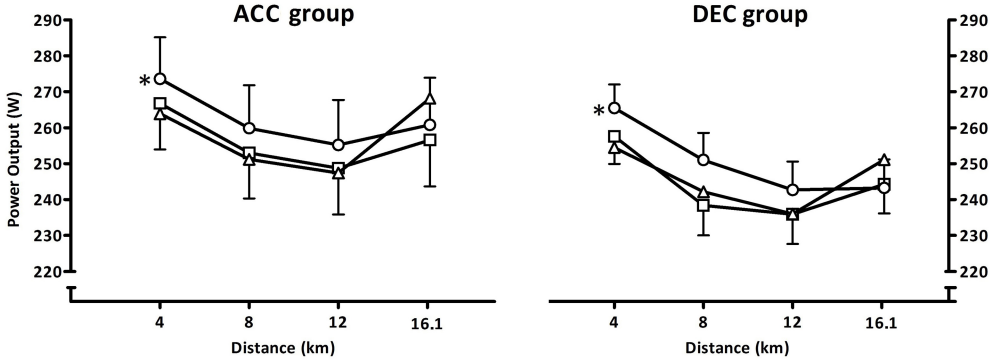

FIGURE 3 | Mean (SEM) power output at each distance quartile in 16.1 km time trials for the ACC and DEC groups. *Denotes significantly higher mean power output than FBL and SUB ($P < 0.001$).

0.001). Significantly higher values were found in PACER than FBL (MD = 3 beats·min^{-1}; CI = 0.51, 6.44; P = 0.017, $d = 0.25$) and SUB (MD = 4 beats·min^{-1}; CI = 1.42, 7.45; $P = 0.002$, $d = 0.36$). A significant trial x distance interaction was also found ($F = 2.7$; $P = 0.036$) and post hoc analysis revealed significantly higher heart rate in PACER than FBL at 8 km (MD = 5 beats·min^{-1}; CI = 0.21, 0.57; $P = 0.021$, $0 = 0.35$) and in PACER than SUB at 8 km (MD = 5 beats·min^{-1}; CI = 0.51, 8.97; $P = 0.024$, $d = 0.42$) and 12 km (MD = 5 beats·min^{-1}; CI = 1.03, 9.51; $P = 0.01$, $d = 0.44$). Significant main effects for V_E were found for distance ($F = 20.20$,$P < 0.001$) and trial ($F = 9.6$; $P < 0.001$). V_E in PACER was significantly higher than in FBL (MD = 9.6 L·min^{-1}; 95 = 1.74, 17.50; $P = 0.012$, $d = 1.13$) and SUB (MD = 13.6 L·min^{-1}; 95 = 5.37, 21.78; $P < 0.001$, $d = 0.29$). A significant main effect for distance was found for RER ($F = 56.5$, $P < 0.001$) and both trial ($F = 4.0$; $P < 0.029$) and distance ($F = 21.2$, $P < 0.001$) main effects were found for VO_2. Mean VO_2 was significantly higher in PACER than SUB (MD = 12.6 L·min^{-1}; CI = 7.77, 243.82; $P = 0.033$, $d = 1.13$) (**Table 2**). A significant main effect for BLA was found for trial ($F = 6.3$; $P = 0.005$), with higher values found in PACER than SUB (MD = 1.6 mmol·L^{-1}; CI = 0.46, 2.72; $P = 0.003$, $d = 0.23$). The difference in BLa between PACER and FBL was also approaching significance (MD = 1.1 mmol·L^{-1}; CI = −0.04, 2.19; $P = 0.062$, $d = 0.83$; **Table 2**).

TT1–TT2

Between-group analysis for TT1 and TT2 data revealed no significant differences for performance time, RPE, self-efficacy, V_E, VO_2, RER or BLa ($P > 0.083$). In the ACC group, power output and speed were significantly higher at 4 km in TT1 than TT2 (PO: MD = 9 W; CI = 1.2, 18.2; $P = 0.03$, Speed: MD = 0.5 km.h-1; CI = 0.03, 0.92; $P = 0.038$). Heart rate was significantly higher in TT1 than TT2 for both groups. In the ACC group, heart rate was higher at 4 and 8 km ($P < 0.008$), and at 4, 8 and 12 km in the DEC group ($P < 0.029$). A significant difference was found in the DEC group for affect at 16.1 km, with a higher value found in TT2 than TT1 (MD = 1.3; CI = 0.18, 2.32; $P = 0.028$). Nine participants performed TT1 faster than TT2 and eight participants performed TT2 in a faster time.

DISCUSSION

The main findings demonstrate that cycling TT performance is not influenced by the manipulation of previous performance beliefs and is instead facilitated acutely via the provision of visual feedback. Both the ACC and DEC groups equally improved performance with the presence of a pacer representative of a 2% faster performance than their FBL. Similarly, both groups experienced lower affect and self-efficacy and higher RPE in this PACER trial. The novel inclusion of a subsequent trial following the disclosure of the nature of the deception aimed to evaluate whether the manipulation of beliefs can elicit an enduring change of behavior and perceptual appraisals. However, residual effects of this feedback provision were absent in both groups as neither perceptions nor performance differed between FBL and SUB. This suggests that the facilitation of a visual avatar has only acute effects which are not sustained residually. This is evident irrespective of whether the avatar is an accurate representation of a 2% faster profile of an athlete's previous performance or whether the athlete falsely believes that this 2% faster avatar represents their previous performance. Even with corrected knowledge of the prior deception, the DEC group's perceptual responses nor performance differed in their SUB TT.

The current study supported previous findings which evidenced acute facilitative effects of visual feedback provision on performance during self-paced cycling TTs (Corbett et al., 2012; Stone et al., 2012; Williams et al., 2014, 2015; Jones et al., 2016). Both groups performed against the same magnitude of pacer (102% of FBL) but were provided with different instructions and therefore had different pre-performance beliefs. The key findings indicate that the presence of a pacer improves performance but the accuracy of the feedback provided, and thus the participants' beliefs, had no effect on the extent of this improvement. This is

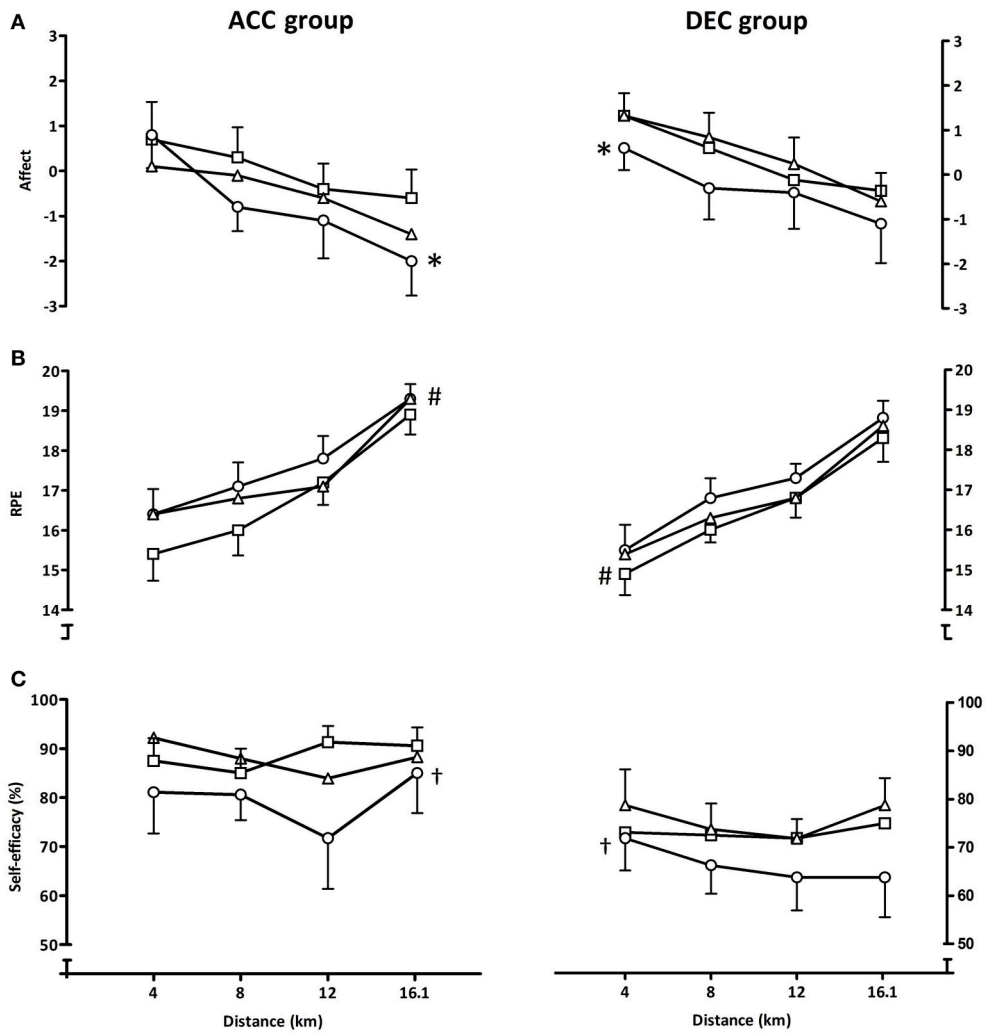

FIGURE 4 | Mean (SEM) affect (A), RPE (B), and self-efficacy (C) responses at each distance quartile in 16.1 km time trials for the ACC and DEC groups. *Denotes significantly lower mean affect than FBL ($P < 0.005$). #Denotes significantly higher mean RPE than FBL and SUB ($P < 0.005$). †Denotes significantly lower mean self-efficacy than FBL and SUB ($P < 0.005$).

in support of a recent study which also demonstrated that TT performance improvements were similar between deception and control groups despite differences in performance beliefs (Jones et al., 2016). Furthermore, whilst supporting the first hypothesis, physiological and perceptual responses did not differ between groups; RPE, heart rate, BLa, V_E and VO_2 all increased in PACER and affect and self-efficacy were lower, further indicating that beliefs did not influence other variables. The ACC group were able to focus on success without the threat of failure as they had accurate knowledge of the augmented performance feedback and, therefore, a more socially acceptable failure outcome. Contrastingly, the DEC group were exposed to threat as they were misinformed about the augmented feedback. In this case, the failure to match a performance believed to be achievable would not be considered an acceptable outcome by these trained, competitive cyclists. Interestingly, this threat state did not result in a slower performance or more negative perceptual responses, as supported by previous evidence of differences in perceptual and behavior outcomes between challenge and threat situations (Skinner and Brewer, 2002; Meijen et al., 2013; Vine et al., 2013). Consequently, activating motivational processes via the use of challenging/threatening visual feedback appears to be more effective at improving performance than conditions of no activation. However, the neurological mechanisms of these motivational processes might differ and warrant exploration in future research.

The absence of a difference between groups is in contrast to previous research in which deceptive exposures have

elicited performance improvements beyond that of a control condition (Corbett et al., 2012; Stone et al., 2012). Stone et al. (2012) highlighted the potentially confounding effect of social facilitation on the findings and acknowledged that an accurately informed group competing against a 102% pacer would reveal the extent to which competition alone may have influenced their findings. The inclusion of a 102% accurate feedback group in this study, and resultant findings that TT performance did not differ to the DEC group, therefore supports that simply the presence of challenging visual feedback is sufficient to evoke a faster TT performance, and not the beliefs associated with the feedback (Weinberg et al., 1979, 1981).

The true nature of the deception was revealed to the participants in the DEC group prior to completion of the SUB TT. This information acted to correct the false belief that they had performed worse in PACER in comparison to their FBL. Similarly, however, performance and perceptions following this disclosure did not vary in comparison to the ACC group, refuting our second hypothesis. This differs from the findings of Shei et al. (2016) who used a feedback manipulation which was also revealed to participants and observed performance improvements. Instead, our data suggest that these results were likely confounded by the presence of the pacer in the subsequent trial. In the present study, the absence of between-group differences did not demonstrate that the correction of false beliefs, intended to produce positive beliefs and stimulate psychological momentum, influenced cycling TT performance or perceptual responses. This may be explained by the explication of the deception disclosure. Participants were simply informed that the feedback provided in PACER was false but no explicit reference was made to the performance outcome, such as completion time. Therefore, it cannot be concluded that all participants reappraised their prior performance as successful and thus inducing a positive effect on self-efficacy.

In summary, the practical implications of feedback provision, either accurate and challenging or non-contingent and threatening in nature, may be subject to the success or failure of the performance during the exposure and thus is an area warranting further exploration. It should be noted that a true control condition was not included in this study, i.e., accurate feedback of a pacer representing the participants' FBL performance, therefore the reader should also consider previous findings in their evaluation of the current study (see Stone et al., 2012; Jones et al., 2016). Finally, it is possible that a single experiential exposure may not be sufficient to produce meaningful changes in the performance beliefs of trained individuals beyond the acute setting and future research should explore the manipulation of beliefs over longer periods.

CONCLUSION

The main findings from this study extend support that deception has no additional influence on 16.1 km cycling TT performance or perceptual responses than simply the presence of challenging feedback. This therefore suggests that the accuracy of visual feedback provided to athletes and the resultant performance beliefs might be superfluous. Revealing to athletes that their prior performance beliefs were falsely negative due to an exposure to deceptive feedback has no effect on subsequent perceptions or performance.

AUTHOR CONTRIBUTIONS

HJ, Conceptualizing and designing the study, data acquisition, data analysis, drafting the article, revising it critically for intellectual content, final approval of the version to be published, agreement to be accountable for all aspects of the work; EW, Conceptualizing and designing the study, data acquisition, final approval of the version to be published; DM, revising it critically for intellectual content, final approval of the version to be published, agreement to be accountable for all aspects of the work; SS, Interpretation of the data, revising it critically for intellectual content, final approval of the version to be published, agreement to be accountable for all aspects of the work; CB, Interpretation of the data, revising it critically for intellectual content, final approval of the version to be published, agreement to be accountable for all aspects of the work; AM, Data analysis, revising it critically for intellectual content, final approval of the version to be published, agreement to be accountable for all aspects of the work; LM, Interpretation of the data, revising it critically for intellectual content, final approval of the version to be published, agreement to be accountable for all aspects of the work.

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The Science of Racing against Opponents: Affordance Competition and the Regulation of Exercise Intensity in Head-to-Head Competition

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Athlete–environment interactions are crucial factors in understanding the regulation of exercise intensity in head-to-head competitions. Previously, we have proposed a framework based on the interdependence of perception and action, which allows us to explore athletic behavior in the more complex pacing situations occurring when athletes need to respond to actions of their opponents. In the present perspective we will further explore whether opponents, crucial external factors in competitive sports, could indeed be perceived as social invitations for action. Decisions regarding how to expend energy over the race are based on internal factors such as the physiological/biomechanical capacity of the athlete in relation to external factors such as those presented by opponents. For example: Is the athlete able to overtake competitors, or not? We present several experimental studies that demonstrate that athletes regulate their exercise intensity differently in head-to-head competition compared to time-trial exercises: Relational athlete-environment aspects seem to outweigh benefits of the individual optimal energy distribution. Also, the behavior of the opponents has been shown to influence pacing strategies of competing athletes, again demonstrating the importance of relational athlete–environment aspects in addition to strictly internal factors. An ecological perspective is presented in which opponents are proposed to present social affordances, and decision-making is conceptualized as a resultant of affordance-competition. This approach will provide novel insights in tactical decision-making and pacing behavior in head-to-head competitions. Future research should not only focus on the athlete's internal state, but also try to understand opponents in the context of the social affordances they provide.

Keywords: athlete–environment interactions, decision-making, pacing, endurance sport

THE REGULATION OF EXERCISE INTENSITY THROUGHOUT THE YEARS: A FOCUS ON UNDERSTANDING HOW TO PACE A TIME-TRIAL

In endurance sports, athletes are continuously required to make decisions about how and when to invest their limited energy resources over time to achieve the completion of one or multiple exercise tasks (Smits et al., 2014). This goal-directed regulation of the exercise intensity over an exercise bout has been defined as "pacing" (Abbiss and Laursen, 2008) and is widely recognized as an essential determinant for performance (Edwards and Polman, 2013). Research has focused on explaining how athletes pace their races and regulate their exercise intensity. Modeling studies have been conducted as early as in the 80's (Van Ingen Schenau, 1980, 1982; Van Ingen Schenau and De Groot, 1983; Van Ingen Schenau et al., 1983, 1990; Van Ingen Schenau and Cavanagh, 1990) demonstrating that in terms of aerodynamics and power losses, a fast start strategy was optimal for time-trials shorter than 2 min. For the longer distances, an even-paced strategy fluctuating around average velocity was advisable (Abbiss and Laursen, 2008). More recently, modeling studies explored optimal pacing in middle-distance time-trials of about 2 min' duration (Hettinga et al., 2011, 2012) while collecting experimental evidence to support their outcomes in reality. The success of a fast start strategy was confirmed. Nevertheless, the studies provided food for thought regarding how fast a fast start should be, and if there would be differences between sports regarding optimal pacing. A subsequent experimental study demonstrated that indeed, pacing differs between sports (Stoter et al., 2016). When instructed to perform optimally with a fast start strategy, cyclists started explosively with a very fast first quartile of the race. Skaters also started fast, but less explosive and spread their energy over a relatively fast first half of the race, with similar muscle fatigue levels compared to cycling at the finish line.

The above provides an example of how modeling studies supported by experimental studies can lead to new insights into pacing and the regulation of energy expenditure. And in addition, many experimental studies have been conducted, manipulating different pacing strategies since the nineties and onwards (Foster et al., 1993, 2003, 2004, 2005; De Koning et al., 2005, 2011), providing interesting insights into actual pacing outcomes. Based on these, as well as on observational studies exploring athletic behavior in competition, several theoretical frameworks have been proposed to understand the process of pacing. In 1996, the model of teleoanticipation was introduced (Ulmer, 1996) and further expanded upon in the central governor theory (Noakes, 1997, 2012) providing the first larger context on how pacing might actually be regulated. The concept of teleoanticipation suggested that the execution of a task is regulated in an anticipatory way, in which a pre-planned strategy or template (Foster et al., 2009) for how the task should be performed is already formed before the start of the race, based on information of the endpoint; a feedback control system must exist, including a programmer that takes into consideration the finishing point of the race (Ulmer, 1996; Noakes, 1997). Since then, other theoretical frameworks were introduced (Marcora, 2008a,b; Edwards and Polman, 2013). However, it has only been very recently that the importance of decision-making aspects and the external environment was emphasized in the context of pacing (Renfree et al., 2014a; Smits et al., 2014). As a result, most previous models have not addressed athlete–environment interactions, and most experimental and modeling studies focused solely on time-trial exercise: racing against the clock. Yet most competitive sport are characterized by head-to-head competitions, where all contenders start at the same time and the winner of the event is the one who passes the finish line first. In head-to-head middle-distance and endurance competition, athlete–environment interactions are crucial in understanding the regulation of exercise intensity (Smits et al., 2014). Previously, we have proposed a framework based on the interdependence of perception and action, which allows us to incorporate, understand, and explore athletic behavior in more complex tactical pacing situations occurring when athletes need to respond to actions of their opponents (Smits et al., 2014). This ecological framework toward pacing argues that the external world provides the athlete with several social invitations for action (Smits et al., 2014). In the following, we will illustrate this concept by focusing on arguably the most important external factor present in competitive sports: the opponent. We will overview studies that have been conducted on racing against opponents, and discuss how these have contributed to understanding the science behind racing against opponents. Finally, we will further elaborate on our previously proposed ecological framework to understand pacing, tactics, behavioral responses, and decision-making of athletes when racing against opponents.

DOES AN OPPONENT MAKE A DIFFERENCE AT ALL?

Indeed, the presence of an opponent has been shown to influence performance (Hulleman et al., 2007; Peveler and Green, 2010; Bath et al., 2012; Corbett et al., 2012; Stone et al., 2012; Lambrick et al., 2013; Tomazini et al., 2015; Williams et al., 2015a,b; Jones et al., 2016; Konings et al., 2016c). In general, improved performance is seen during competitive running and cycling trials compared to individual or non-competitive trials (Corbett et al., 2012; Stone et al., 2012; Tomazini et al., 2015; Williams et al., 2015a,b; Konings et al., 2016c). However, the actual presence and perception of the opponent seems to be crucial. Even the prospect of a monetary incentive ($100,-) did not improve 1500 m cycling performance when the "competitor" (i.e., best previous performance so far) was not visible during the trial (Hulleman et al., 2007).

Interestingly, the presence of a second runner did not improve 5-km running performance when the distance between the athlete and second runner was maintained at ∼10 m during the time-trial (Bath et al., 2012). The perception of approaching or getting further behind your opponent might even be a crucial variable (Meerhoff et al., 2014). However, starting 1 min behind (chasing) or in front (being chased) of an opponent did not affect

performance (Peveler and Green, 2010). Konings et al. (2016c) recently showed that also the actual behavior of the opponent affected pacing behavior (Konings et al., 2016c). That is, a faster starting opponent evoked a faster start than a slower starting opponent. The presence, as well as the behavior of an opponent affect the decision-making process and pacing behavior of an athlete when competing in a race. Previous research has made several suggestions to explain why athletes perform better when an opponent is present. For example, an increased motivation (McCormick et al., 2015) and a change in attentional focus from internal to external aspects have been mentioned (Williams et al., 2015a). Experimentally, it was demonstrated that when an opponent is present, athletes were able to handle higher levels of peripheral fatigue (Konings et al., 2016b).

Also in the field, it was demonstrated that athlete–environment interactions affected athletic decisions and pacing behavior. Observational studies with large datasets and advanced statistics explored different competitive events involving head-to-head competition such as short track speed skating, rowing, running and cycling (Jones and Whipp, 2002; Paton and Hopkins, 2006; Dwyer et al., 2013; Hanley, 2013, 2014, 2015; Moffatt et al., 2014; Renfree et al., 2014b; Edwards et al., 2016; Konings et al., 2016a; Noorbergen et al., 2016). In all these studies, tactical components, such as favorable positioning, drafting, competing for the optimal line, and minimizing fall risk, seemed to influence decisions and draw athletes away from the energetically favorable strategies as would be performed in time-trial exercise. One could even lose a gold medal despite a higher average velocity due to adverse positioning wide on the bend (Jones and Whipp, 2002), particularly in important events such as the Olympic Games and World Championships (Thiel et al., 2012; Renfree and St Clair Gibson, 2013). Only when an all-out strategy could be adopted from the beginning of the race, athletes adopted a comparable pacing strategy to time-trial sports and modeling studies (Hanon and Gajer, 2009; Noorbergen et al., 2016). Interestingly, sports with a high beneficial effect of drafting behind your opponent (e.g., short-track speed skating, cycling) are characterized by a relatively slow development of the race (Moffatt et al., 2014; Konings et al., 2016a; Noorbergen et al., 2016). A remarkable exception is the relatively fast pace adopted in the elimination discipline in track cycling (Dwyer et al., 2013). This might be explained by the unique character of the discipline in which every two laps the last ranked competitor is eliminated.

In contrast, in competitive sports with a relatively low beneficial effect of drafting such as race walking or middle-distance and marathon running, (sub-)elite runners tend to adopt a pacing strategy in the beginning of the race that they cannot sustain until the end of the race (Hanley et al., 2011; Thiel et al., 2012; Hanley, 2013, 2014; Renfree and St Clair Gibson, 2013; Deaner et al., 2015). In addition, the slowdown was higher for men compared to women during marathons (Deaner et al., 2015). In the Oxford-Cambridge Boat race, in which being in second position has negative effects associated with being in the wake of the leading boat, increasing energy costs, a fast start strategy has been employed by all teams since 1890 (Edwards et al., 2016), even though energetically, an even paced strategy would be favorable (Van Ingen Schenau et al., 1983). The team in the lead after the first quartile of the race won the race in 81% of the cases. It thus seems that the possibility of drafting, and the magnitude of associated energy-saving effects of drafting, is an important determinant for pacing behavior and tactical decision-making. It has been proposed that pacing against opponents can be seen as collective behavior (Renfree et al., 2015) in which indeed, drafting benefits are a crucial determinant for behavior. Finally, in certain situations athletes may even decide to cooperate rather than compete with their opponents (Hanley, 2015).

All these examples based on experimental and observational data have demonstrated that racing against an opponent is different from riding a time-trial: balancing tactical (dis)advantages against the energetically optimal distribution pace is required to perform optimally. This is demonstrating the need for a theoretical framework incorporating athlete–(social pacing) environment interactions.

PERCEPTION-ACTION AND ATHLETE-PACING ENVIRONMENT MUTUALITY

In the following, a framework (based on ecological psychology and first introduced in Smits et al., 2014) is outlined that allows us to incorporate, understand and explore athletic behavior in the more complex tactical pacing situations occurring when athletes need to respond to actions of their opponents.

Since the mechanization of the worldview in the Seventeenth century, mind-body and man-environment dualism have dominated the scientific disciplines (e.g., Lombardo, 1987; Reed, 1996). The separation of mind and body that ensued has had huge consequences for the way in which scientists have conceptualized cognition and how it relates to pacing and decision-making in the regulation of exercise intensity. Most important, the environment in which sport behavior takes place became conceived of as meaningless, consisting merely of matter in motion (see e.g., Neisser, 1967). This ultimately led scientists to become interested in hypothetical constructs like mental sensations, memory, and information processing (the basics of cognitive psychology). These processes have now become more valuable to researchers than the experience of the concrete and real events and behaviors that actually take place in the environment.

In response, in the late 1970's Gibson formulated what would become the foundations of ecological psychology (Gibson, 1977, 1979; Reed, 1996; Gibson and Pick, 2000). This is a theoretical perspective "... in which the psychological experiences and activities of persons and animals are placed firmly at the center of our field" (Reed, 1996, p. 6). It is an approach that has two mutualities at its core: (i) that of athlete and environment, and (ii) that of perception and action. Both these mutuality's come together in the concept of affordances: the opportunities for action that are presented to us by the environment in which we perform. Affordances are meaningful and real relations between athletes and their environments that are perceived directly. They are defined relative to the action-capabilities of an athlete, and allow for prospective control of actions. What is important

for understanding decision-making in pacing is that they are dynamic. All (sport) behavior is regulated relative to affordances (for a full account, see Fajen et al., 2009; Barsingerhorn et al., 2012).

Affordances can be characterized as invitations for action from the environment (Withagen et al., 2012). In the context of sport, the environment of an athlete consists of invitations from objects, places and events, and—important for the current perspective—other people, relevant for that athlete. Affordances become available and dissipate, and allow the athlete to make trade-offs between choosing to persist in a given behavior (e.g., remain on current pace, or not respond to an action of the opponent) or switching to a different one (speed up or slow down, or follow the actions of an opponent or even overtake him/her). In this way, the performance environment surrounds athletes with a multitude of invitations for action. It is up to the athlete to act upon certain affordances, and not on others. This leads to the notions of affordance based decision-making and the affordance-competition hypothesis (Cisek, 2007; Cisek and Kalaska, 2010).

Traditionally, decision-making in natural behavior is interpreted as a sequential process in which the selection of a particular behavior (keeping at current pace, speeding up, slowing down, overtake, etc.) occurs before the behavior is specified, or coded by the brain, and to be executed by the body. The theory of affordances proposes a radical departure from this line of reasoning, and suggests that the selection and the specification of behavior are essentially the same dynamic process (Cisek and Kalaska, 2010; Barsingerhorn et al., 2012). The affordance competition hypothesis describes how the interactions between an individual's needs, action capabilities, and the environment provide for the specification of potential affordances (and related actions). From this viewpoint, decision-making should be understood as emerging from specification of simultaneously available affordances, and the competition between them (Smits et al., 2014). Similarly, in understanding how organisms choose between affordances in situations in which different affordances can be utilized, Reed (Reed, 1993) posited that intentional patterns of organization emerge in situations in which organisms have a real choice of behavior. Following a Darwinian line of reasoning, Reed argued that intentions: "are 'species' that emerge out of competition among perceptual and action processes for utilizing affordances" (Reed, 1993, p. 65; see also Withagen et al., 2012). A further extensive overview of research that has examined the affordance-competition-hypothesis in neuropsychological and neurophysiological research can be found in Cisek and Kalaska (2010).

OPPONENTS AS SOCIAL INVITATIONS FOR ACTION

Head-to-head competition takes place in a complex performance environment. Complex, in this context, refers to the notion that the ongoing modulation of behavior during head-to-head competition is embedded in a complex system that comprises the athlete, opponents, as well as a large number of other interacting components (Araújo et al., 2006; Araújo and Davids, 2009). Following the idea of athlete-environment mutuality, the complex performance environment allows athletes to continuously modify ongoing behavior or pre-planned race strategies, on the basis of—and resulting from the competition between—the affordances that are available to them. This includes opportunities for action that are constrained by factors internal to the athlete and for instance dictated by the athlete's energy systems (fatigue (Konings et al., 2016b) and pain (Mauger, 2014; Astokorki and Mauger, 2017). It also includes opportunities for action presented by the athlete's material and technology (such as distance and speed information available from external devices Boya et al., 2014; Smits et al., 2016). But most importantly for our current argument, there are affordances presented by the social environment, such as opponents in a competition or race (Konings et al., 2016c). We propose that opponents present social affordances; social invitations for behavior. In racing against opponents, three categories of social affordances can be identified (see also Fajen et al., 2009). Firstly, there are affordances *for* opponent(s), that is, what opportunities for action are available for opponent(s)? And even, does the perceiving and acting athlete present an opponent with affordances? For example: if an athlete starts very fast, it is likely that he or she "invites" their opponent to start faster than (s)he would when riding alone (Konings et al., 2016c). Secondly, there are affordances *of* an opponent: What actions does an opponent invite the perceiving and acting athlete to do? Related to drafting opportunities for example, decisions are shaped by the opportunities opponents offer in terms of energy efficiency when staying behind them. Another example is overtaking: the decision to overtake is dependent on the actions and proximity of the opponents, an interesting aspect for further research (Al et al., 2016; de Jong et al., 2016; Hettinga et al., 2016). Finally, there are affordances for *joint action*. These are the opportunities for action available to a group or system as a whole. There are likely to be differences between pacing behaviors in situations where pacing is construed or imposed, for instance through the use of a pace-setter or rabbit in track-and field running, and those instances where pacing behavior is more "organic." The collective behavior patterns as those described to emerge in peloton formations, such as peloton phase transitions, phase symmetry, peloton divisions, and between-rider distances (Trenchard et al., 2014; Renfree et al., 2015) are related to affordances for joint action.

IMPLICATIONS, RECOMMENDATIONS, AND CONCLUSIONS

To conclude, we have seen how opponents influence performance and make a difference to the pacing strategies that are enacted. A framework is presented that provides an alternative way of understanding decision-making regarding the regulation of exercise intensity in social contexts. An approach, rooted in the affordance-competition hypothesis as described in Smits et al. (2014) is put forward. Perception-action and athlete–environment interactions are a crucial

factor in understanding the regulation of exercise intensity in head-to-head middle-distance and endurance competition. Decision-making is the actualization of affordances, and a result of the competition between simultaneously available affordances. Decisions regarding how to expend energy over the race are based on relationships between internal factors such as the physiological/biomechanical capacity of the athlete and external factors such as opponents. For example: Is the athlete able to accelerate fast enough to overtake competitors? How does fatigue impact on the decision to accelerate?

Decision-making, conceptualized as resulting from the competition between affordances, implies that not all affordances presented at a certain moment in time—however effective they may seem—are actualized. In the competition between affordances, only certain survive. For instance, a fast-starting (group of) cyclists might afford other cyclists a similar fast start. With experience and training, an athlete can learn to discover other affordances, for instance that a fast-starting (group of) cyclists requires an alternative response, because of the negative effects it can have on pacing. This resisting of certain affordances in competition might be difficult for the athlete, but it is an important skill to learn with regard to pacing. In a similar vein, not all affordances invite. As argued by Withagen et al. (2012, p. 255): "Although (experienced) affordances can have the potential to invite a certain activity, the vast majority of affordances do not. [...] a single object generally affords multiple behaviors to an individual, and not all of these affordances invite."

When in competition, opponents present a multitude of affordances that influence motivation, attentional focus (perception), the ability to tolerate fatigue and pain, positioning, drafting, falls risk and collective behavior. The presented framework explains that athletes' decision-making cannot and should not be understood in the independent understanding of these aspects. Rather, decision-making, and modification of ongoing behavior can only be properly understood in the context of the simultaneous availability of multiple affordances (related to opponents, fatigue, positioning, etc.) and the competition between them. For future research into the effect of opponents on the regulation of exercise intensity it is therefore advised to understand opponents in the context of the *social affordances* that they provide and the changes they invite in the ongoing behavior of athletes. At the same time, internal aspects are crucial, and we need to understand how and to what extent internal and external factors interact. Studies of competitive behavior in the field are crucial here, but also elegantly designed experiments manipulating both external factors (such as athlete behavior) as well as internal aspects (such as fatigue).

AUTHOR CONTRIBUTIONS

FH and GP contributed to conception and design of the work, drafted it and revised it critically for important intellectual content. All authors (FH, MK, GP) contributed to the final draft and have approved the final version of the manuscript, agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All persons designated as authors qualify for authorship, and all those who qualify for authorship are listed.

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The Manipulation of Pace within Endurance Sport

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In any athletic event, the ability to appropriately distribute energy is essential to prevent premature fatigue prior to the completion of the event. In sport science literature this is termed "*pacing.*" Within the past decade, research aiming to better understand the underlying mechanisms influencing the selection of an athlete's pacing during exercise has dramatically increased. It is suggested that pacing is a combination of anticipation, knowledge of the end-point, prior experience and sensory feedback. In order to better understand the role each of these factors have in the regulation of pace, studies have often manipulated various conditions known to influence performance such as the feedback provided to participants, the starting strategy or environmental conditions. As with all research there are several factors that should be considered in the interpretation of results from these studies. Thus, this review aims at discussing the pacing literature examining the manipulation of: (i) energy expenditure and pacing strategies, (ii) kinematics or biomechanics, (iii) exercise environment, and (iv) fatigue development.

Keywords: pacing factors, energy expenditure, fatigue, kinematic, exercise environment

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INTRODUCTION

Any athletic event inevitably has a beginning and an endpoint. In order to reach this endpoint in the fastest possible time, athletes need to appropriately distribute their energy expenditure, in a way that all available energetic resources are used but not so early so as to experience premature fatigue prior to the finish line (St. Clair Gibson et al., 2006). In sport science literature this has been termed as "*pacing*," "*pacing strategy*," "*pacing profile*" and/or "*pacing pattern*." This regulation of speed, power or energy expenditure throughout an exercise task is extremely important in the optimisation of performance. Based on current research, pacing appears to be regulated by complex relationships between the brain and other physiological systems (St. Clair Gibson and Noakes, 2004; Noakes et al., 2005; Abbiss and Laursen, 2008). Several models have been proposed to explain this phenomena including: the teleoanticipatory theory (Ulmer, 1996; St. Clair Gibson and Noakes, 2004), the central governor model (Noakes et al., 2001), the perception based model (Tucker, 2009), the pacing awareness model (Edwards and Polman, 2013) and the psychobiological model (Marcora, 2010; Pageaux, 2014).

Since our last review on athletic pacing (Abbiss and Laursen, 2008) research examining physiological, psychological and environmental factors influencing pacing during exercise has dramatically increased. Much of this research has: (i) observed the pacing profiles adopted by athletes in competition (Abbiss and Laursen, 2008), (ii) examined the effects of directly manipulating pacing on performance (Foster et al., 1993; Aisbett et al., 2009a; Skorski et al., 2014), or (iii) examined the effects of manipulating feedback (Albertus et al., 2005; Mauger et al., 2009; Faulkner et al., 2011; Castle et al., 2012), task demands or environmental conditions (Clark et al., 2007; Abbiss et al., 2010; Peiffer and Abbiss, 2011) on pacing and performance. Such research has been instrumental in improving our understanding of how humans regulate energy expenditure

and the "optimal" pacing strategies to adopt during various exercise tasks. However, as with all research there are several factors that should be considered in the interpretation of results from these studies. This manuscript will discuss these considerations in the context of pacing. Specifically this review will discuss factors important to the interpretation of pacing literature examining the manipulation of: (i) energy expenditure or pacing strategies, (ii) kinematics or biomechanics, (iii) exercise environment, and/or (iv) fatigue development.

MANIPULATION OF ENERGY EXPENDITURE OR PACING

Technological advancements have made it easier to quantify both mechanical power output and resistive forces experience during exercise and as a result it has been possible to mathematically model optimal pacing profiles by minimizing the time required to complete a given amount of work. Such modeling typically indicates that exercise performance can be optimized if athletes produce greater power output during periods of high external resistance (i.e., when accelerating, traveling into a headwind or against gravity) and lower power outputs produced during periods of lower resistance (Swain, 1997; Atkinson et al., 2007; Wells and Marwood, 2016). However, the degree to which an athlete might be able to vary exercise intensity, in order to compensate for high resistive forces, is dictated by their psychophysiological capabilities (Atkinson et al., 2007). Indeed, most theoretical models highlight cognitive and physiological characteristics as key variables that dictate the regulation of pacing during exercise (St. Gibson et al., 2006; Pageaux, 2014; Renfree et al., 2014).

Several studies aimed at better understanding optimal pacing profiles have deliberately forced participants to adopt specific pacing strategies in order to determine, if such pacing profiles improve performance above theoretically submaximal strategies (Aisbett et al., 2009a,b) and if athletes are able to adhere to the required power outputs (Thomas et al., 2013). An important consideration with regards to these studies is that forcing athletes to adopt a given workload, even if only for a short period of an event, could in itself influence overall performance. Indeed, choice or the anticipation of the opportunity for choice has been associated with increased activity of brain regions directly involved in reward processing (Fujiwara et al., 2013; Lewthwaite et al., 2015) and has therefore been considered as an important regulator e.g., for motor learning (Lewthwaite et al., 2015). Thus, forcing athletes to adopt a given pacing profile might remove self-choice, giving them less control over the situation and influence performance and motivation. It has also been shown that compared with self-paced exercise, externally controlling exercise intensity alters attentional focus, resulting in the predominance of reactive, rather than proactive cognitive control (Brick et al., 2016). Interestingly, a reduction in the ability to control exercise intensity does not only influence cognitive function but also results in greater physiological stress and reduced exercise capacity, when compared to a self-paced exercise task (Billat et al., 2006; Lander et al., 2009). For instance, Thomas et al. (2013) found that several participants were unable to complete the same amount of work during an externally controlled, even paced cycling trial, when compared with prior self-paced 20 km cycling trials. Conversely, several studies have also observed lower physiological stress during externally controlled trials (Zadow et al., 2015; Brick et al., 2016), when compared with matched work self-paced trials, possibly as a result of reduced cognitive load (Garcin et al., 2008; Lander et al., 2009). However, despite of lower physiological strain perceptual responses (i.e., RPE) were similar (Thomas et al., 2013) or even higher (Lander et al., 2009) in externally controlled trials. Thus, the ability to voluntarily fluctuate power output in accordance with sensations of fatigue during the exercise bout seems to be an important factor in the regulation of pace (Lander et al., 2009). Enforced or externally paced strategies, force the athlete to abandon their pacing pattern and minimize opportunities for self-managing fatigue (Lander et al., 2009).

The reason for inconsistencies with regards to the effects of externally controlling pace, within the literature, is not clear. However, it should also be noted that within the current pacing literature various methods have been used in order to enforce specific pacing strategies. Such methods have included ergometers set to automatically regulate intensity regardless of stroke rate or cycling cadence (Abbiss et al., 2009), or requirements of athletes to maintain a given intensity based on visual (Zadow et al., 2015) or audio feedback (Thompson et al., 2003; Skorski et al., 2014). Furthermore, some studies used simulated competitors (i.e., avatars) to examine the effects of their presence on self-selected pacing strategies (Shei et al., 2016). It should be noted that each of these methods not only differ in the mechanisms by which external pacing is controlled, but also in the feedback provided to participants. Given that the cognitive demand and attentional focus is likely to differ between each of these methods caution should be taken comparing results across studies or in the implementation of such findings into the field. It is of note that the current literature which has enforced a specific pacing condition, in order to approximate mathematical models or better understand optimal pacing strategies, has typically only performed a single trial in each pacing condition. While research has shown consistent and moderate reliability in cycling time trials with an enforced starting pace (first quarter of 5 min cycling time trials, Aisbett et al., 2015), we are unaware of any studies that have deliberately and systematically trained an athlete's pacing profile. Such research might be meaningful since it has been shown that both training (Kennedy and Bell, 2003) and heat acclimation (Racinais et al., 2015) results in the adoption of a more even pacing profile during an endurance task, which is theoretically optimal based on mathematical modeling and better approximates the profile adopted by well-trained or elite athlete (Wilberg and Pratt, 1988).

MANIPULATION OF KINEMATICS AND BIOMECHANICS

To date, research examining the effects of manipulating kinematic and/or biomechanical variables on pacing and

performance is somewhat limited. This is surprising, considering that these variables are often manipulated in sport science practice in an attempt to improve force/power development, prevent fatigue, reduce fluid resistance and ultimately improve performance (Overton, 2013). Moreover, it is plausible that athletes change movement kinematics in order to prevent premature fatigue or vice versa that the fatigue state changes an athletes movement patterns. For example, it has previously been found that during constant-pace middle-distance (∼5–6 min) cycling to task failure, cyclists increase trunk flexion as peripheral fatigue (i.e., decrease in force or power resulting from changes beyond the neuromuscular junction) develops, presumably in order to increase muscle recruitment and torque production at the hip and knee (Overton, 2013). Additionally, within this study it was also found that significant changes in the angle orientation of the trunk, hip and knee in the coronal plane throughout the pedal cycle were observed from 80 to 100% of task failure and paralleled increases in EMG amplitude (Overton, 2013). Further, vertical leg stiffness decreases with increasing fatigue which results in a decrease in stride frequency during sprint running (Morin et al., 2006). In this regard, world-class runners show a larger decease in stride frequency within the last 50 m of a 400 m running race when compared to recreational athletes (Hanon and Gajer, 2009). As velocity decreases in the last part of a 400 m run, stride length and stride frequency also decrease. The decrease in stride length already occurs between 200 and 300 m, whereas stride frequency can mostly be attributed to the last 50 m (Hanon and Gajer, 2009). This decrease in stride frequency might be due to a greater stride length earlier in the race which increases ground force production and thus fatigue development (Mero et al., 1992; Hanon and Gajer, 2009). These findings are important as they highlight that fatigue development, kinematics and biomechanics are not consistent throughout an exercise task, which influences the quantification of optimal pacing using mathematical modeling.

To date, few studies have deliberately manipulated movement kinematics in order to examine the influence on self-selected pacing strategies. However, studies have examined the influence of manipulating various biomechanical/kinematic variables on power/force production and fatigue development. Indeed, it is often found that performance is reduced when forcing cyclists to adopt a pedaling rate that differ considerably from self-selected cadences, even if such cadences are more economical or reduce neuromuscular fatigue development (Abbiss et al., 2009). It has also been shown that instructing cyclists to maintain consistent trunk and hip joint kinematics during a fatiguing bout of cycling reduces the time to task failure in some but not all athletes. These results indicate that restricting kinematic variation that normally occurs, presumably in order to overcome fatigue development, might compromise performance (Overton, 2013). The reason time to task failure is reduced when joint kinematics are held consistent is currently unclear but may be due to an athlete's inability to manage fatigue by slightly changing movement kinematics (Overton, 2013) or associated the restriction of self-choice, thereby altering motivation (Fujiwara et al., 2013; Lewthwaite et al., 2015). Regardless, such findings are important in the mathematical modeling of optimal pacing since kinematics and fluid resistance is not consistent during the exercise task.

A change in kinematics is likely to be even more crucial in water-based sports like rowing or swimming where resistive forces are higher compared to land-based sports (Smith et al., 2002; Maglischo, 2003). Swimmers usually increase speed by a combination of increasing stroke length and/or stroke rate throughout the event (Smith et al., 2002). Hence manipulating one or both factors might be crucial for swim efficacy, economy and thus performance (Aspenes and Karlsen, 2012). For instance, Swaine and Reilly (1983) found significant changes in oxygen uptake and minute ventilation when stroke rate was manipulated during high-intensity front-crawl swimming on a swim bench. A later study manipulated stroke rate during 200 m breaststroke swimming (Thompson et al., 2003). Using a programmable audible pacing device swimmers were paced at 91, 95, 100, and 107% of their average stroke rate in a previous self-paced trial, yet no statistical significance between trials was observed in performance times, physiological responses (heart rate, blood lactate, oxygen-uptake) and rating of perceived exertion (Thompson et al., 2003). However, when athletes were forced to increase their pace by 2% they swam the 200 m with a significantly higher stroke rate (Thompson et al., 2004). Thus, even though research looking into possible effects of manipulation kinematics and/or biomechanics on pacing is lacking, there is a strong rationale to assume that such enforced changes might alter fluid resistance, fatigue development and optimal pacing during exercise.

MANIPULATION OF EXERCISE ENVIRONMENT

In order to further our understanding of the mechanisms important in the regulation of exercise intensity a large number of studies have observed the self-selected pacing profiles of athletes in a range of environmental conditions but particularly hypoxic or hot conditions. While exercise capacity is reduced in hypoxia, few studies to date have observed drastic changes in pacing during exercise in hypoxia. Conversely, a recent meta-analysis demonstrated that during prolonged exercise in the heat a clear decrease in exercise intensity from commencement of the trial is typically observed (Davies et al., 2016). The authors explain this by the nature of heat interventions, as a marked change in room temperature is easier to identify by participants compared to a change in oxygen content (Davies et al., 2016). It is therefore likely that adjustments in power output or speed are made sooner in the heat trials due to earlier changes in afferent feedback (Davies et al., 2016). Conversely, maximal oxidative capacity and power output is reduced upon exposure to a hypoxic environment compared to normoxia (Davies et al., 2016). Interestingly though, the meta-analysis showed that power output in the starting section of the hypoxia trials was not significantly different to normoxia, which indicates a delay in the adjustment of the pacing pattern in hypoxia (Davies et al., 2016). Such findings are important since they highlight that physiological stress during exercise is not always constant and as

a result the optimal pace to adopt is dynamic and might differ to those predicted in current mathematical models (i.e., even pacing).

It is important to note that altering environmental conditions might influence pacing through effecting not only physiological but also psychological state/function. Indeed, the above mentioned alterations might not only influence muscle oxygen delivery and peripheral fatigue but also influence central motor drive to the exercising skeletal musculature (Davies et al., 2016). It should also be noted that several strategies of altering the exercise environment are likely to influence pacing with greatest effect on cognitive/psychological, rather than physiological function. For instance, it has been shown that inaccurate feedback (Wilson et al., 2012) or the type of feedback (distance vs. duration) (Abbiss et al., 2016) can alter self-selected pacing. The effects of such manipulations on pacing could be the result of effects on motivation, task engagement, attentional focus, and range of other factors believed to be important to pacing during exercise. While the precise mechanisms are beyond the scope of this manuscript, it should be noted that as with physiological stress, one's cognitive state is dynamic and does not necessarily increase linear with exercise duration (de Koning et al., 2011; Wilson et al., 2012). Consequently, understanding the kinetics of central, peripheral and mental fatigue development during exercise is likely to be important in the determination of optimal pacing strategies.

MANIPULATION OF FATIGUE DEVELOPMENT

It is well accepted that central (Meeusen et al., 2006; Taylor et al., 2016), peripheral (Marcora and Staiano, 2010; de Morree and Marcora, 2013; Taylor et al., 2016) and mental fatigue (Marcora et al., 2009; Pageaux et al., 2015) development reduces exercise capacity. However, the influence of such fatigue on pacing during exercise is not entirely clear. As such, recent research has examined the effects of systematically manipulating fatigue development in various regions of the body on self-selected pacing during exercise. Over decades researchers have been arguing on the different origins of fatigue, especially the difference between central and peripheral fatigue. As recently outlined a reason for this dilemma might be the inability of current terminology to accommodate the scope of conditions ascribed to fatigue (Enoka and Duchateau, 2016). Defining the different terms used in the following paragraph in regards to fatigue would extend the scope of this review and have been published elsewhere (St. Clair Gibson and Noakes, 2004; Abbiss and Laursen, 2005; Noakes et al., 2005; Meeusen et al., 2006; Enoka and Duchateau, 2016; Taylor et al., 2016).

Effects of Pre-exercise Peripheral Fatigue

Amann and Dempsey (2008) previously described the effects of locomotor muscle fatigue on overall 5-km time trial performance. In this study it was found that compared to a control condition, time to completion as mean power output during a 5 km cycling time trial was reduced following constant-workload cycling until exhaustion (Amann and Dempsey, 2008). Pre-exercise (peripheral) fatigue levels were assessed via changes in potential twitch force of the vastus lateralis, vastus medialis, and rectus femoris. Potential twitch force was significantly reduced after the constant-workload cycling indicating substantial levels of locomotor muscle fatigue (Amann and Dempsey, 2008). Time to completion was reduced by 2 to 6% and mean power output by 4 to 14% compared to the non-fatigued control condition. Given that power output rose to almost identical levels in the final 200 m of each trial, despite different levels of pre-exercise fatigue, it was speculated that during the trial subjects adopted a more conservative exercise intensity to avoid further accumulation of peripheral fatigue before the end of the trial (Amann and Dempsey, 2008). As such, the authors of this study suggested, that the development of locomotor muscle fatigue an important influence on central motor drive and consequently can compromise exercise performance (Amann and Dempsey, 2008).

Interestingly, a recently published study described the effect of a pre-exercise eccentric fatiguing protocol (100 drop jumps) on pacing in a 15-min cycling trial conducted 30 min after (de Morree and Marcora, 2013). There was no difference in pacing between conditions, even though overall performance was worse in the fatigued trial (de Morree and Marcora, 2013). Nonetheless, participants were again able to increase power output in the last 3 min of the fatigued trial resulting in a similar end-spurt as in the control condition (de Morree and Marcora, 2013). The authors suggested that finishing the race is paramount thus participants choose a lower power output for most of the trial. Near the end of the trial, when the risk of not finishing the race is negligible, most participants significantly increase power output in the end spurt (de Morree and Marcora, 2013).

To summarize, it seems that pre-exercising interventions compromising locomotor muscle performance, either metabolically (Amann and Dempsey, 2008) or mechanically (de Morree and Marcora, 2013), impair overall performance but not the pacing pattern. As previously shown pacing seems to be stable under various conditions (Stone et al., 2011; Thomas et al., 2012; Périard and Racinais, 2016). However, in several endurance competitions, athletes have to compete in different races over several days and/or weeks (e.g., stage races, heats, and finals) pushing various physiological systems to exhaustion (Abbiss and Laursen, 2005), which supposedly results in a multifaceted accumulation of fatigue.

Effects of Accumulated Fatigue

Since it is believed that our distribution of exercise intensity is regulated to prevent premature fatigue, it is likely that pacing is altered by interventions that result in either different amounts of stored energy before exercise or altered substrate use during exercise (Tucker, 2009). In this regard, a recently published study observed that fatigue induced by a 6 day training intervention impacted pacing at the beginning and end of 40 km cycling time trials (Skorski et al., 2015). The 6 day training period consisted of two cycling sessions a day. The morning sessions

consisted of either constant high-intensity endurance training or high-intensity interval training in an alternating manner. In the afternoon sessions, participants had to cycle for 3 h at a moderate-intensity. Participants adopted a parabolic-shaped pattern in both recovered conditions, whereas in the fatigued condition, the pattern was even from the beginning with a greater end-spurt in the last 4 km (slow–fast pacing (Abbiss and Laursen, 2008). The authors of this study hypothesized that training induced fatigue might have a stronger effect on pacing than muscle fatigue *per se* (Skorski et al., 2015). Indeed, whole body fatigue development is multifaceted and influenced by several metabolic, neuromuscular and psychological pathways. Within this study heart rate, blood lactate and respiratory exchange ratio during the time trial were significantly reduced after 6 days of hard training, indicating reduced sympathetic nervous system activity (Skorski et al., 2015). This might lead to disturbed glycolytic energy mobilization and cardiovascular responses (Jeukendrup et al., 1992; Halson and Jeukendrup, 2004; Faude et al., 2009) and a shift of the energy-supplying process in favor of increased fat and decreased carbohydrate use (Jeukendrup et al., 1992). However, after 2 days of recover athletes showed the same pacing pattern as before the training period. Nonetheless, such a shift in substrate utilization might be of practical relevance regarding that in several endurance competitions, athletes have to compete in different races over several days and/or weeks (e.g., stage races, heats, and finals), supposedly leading to a similar accumulation of fatigue. For example, road cyclists compete on 90–100 competition days, comprising 1-d races, 1-wk tour races, and 3-wk tour races (Abbiss and Laursen, 2005). Within each of these races, cyclists might perform different competition requirements (e.g., flat, long stages, time trials, uphill ascents), resulting in exhaustion of various physiological systems (Mujika and Padilla, 2001; Abbiss and Laursen, 2005). Depending on the competition and the stage type, heart rate values range between 51 and 89% of maximum hear rate, with power outputs between 192 and 380 W (Mujika and Padilla, 2001). This leads to high demands of aerobic and anaerobic pathways, which might result in a multifaceted accumulation of fatigue and hence a shift in substrate utilization.

Effects of Mental Fatigue

Mental fatigue is a change in psychobiological state (Marcora et al., 2009) which has subjective and objective manifestations including increased resistance against further effort (Meijman, 2000), changes in mood (Hockey, 1983) and feelings of "tiredness" (Martin et al., 2015). Mental fatigue has been shown to reduce exercise capacity during prolonged moderate and high-intensity exercise (Marcora et al., 2009; Brownsberger et al., 2013). However, Martin et al. (2015), recently found that maximal anaerobic performance was not affected by a mentally fatiguing task performed prior to a 3-min all-out cycling test. The authors speculate that this might be due to the type of the task (Martin et al., 2015). In contrast to the aforementioned studies Martin et al. (2015) analyzed the effects of mental fatigue on a short anaerobic cycling task. During short events it appears that an increase in peripheral fatigue development might be associated with an increase in central nervous recruitment to prevent a decrease in performance (Martin et al., 2015). Conversely, prolonged endurance exercise performance might also be influenced by a development of central fatigue resulting in a reduction of voluntary muscle activation (Gandevia, 2001). Indeed, Thomas et al. (2015) recently observed that the contribution of central and peripheral process to fatigue is task-dependent. Shorter, high intensity time trials result in a greater degree of peripheral fatigue, whereas central fatigue has an increased contribution to longer, lower intensity time trials (Thomas et al., 2015). Thus, short-duration maximal anaerobic tasks might be less influenced by alterations in the mental state when compared with the fatigue development during prolonged aerobic endurance exercise (Martin et al., 2015).

Mental fatigue appears to have little influence on physiological factors (e.g., heart rate, oxygen consumption) (Marcora et al., 2009; Pageaux et al., 2015) but often results in greater ratings of perceived exertion when compared to non-fatigue conditions (Marcora et al., 2009; Marcora and Staiano, 2010). As such, it is hypothesized that rating of perceived exertion plays an important role in self-selected pacing which is in accordance with the psychobiological model of endurance performance recently proposed by Marcora (2010) and Pageaux (2014). Indeed, it has been suggested that exercise intensity is regulated based on one's perceived exertion in order to ensure that "catastrophic" or "critical" disturbances to homeostasis do not occur (Noakes et al., 2001; St. Clair Gibson et al., 2006). This is supported by the relatively stable increase in perceived exertion that is typically observed during high-intensity, self-paced exercise (i.e., a time trial) (Noakes, 2008; Cohen et al., 2013). It has been further proposed that the product of the momentary perceived exertion and the fraction of distance remaining (referred to as a hazard score) might provide an indication of changes in intensity during self-paced exercise (de Koning et al., 2011). Importantly, it has also been suggested that one's perception of effort is centrally derived and largely unaffected by peripheral afferent sensory feedback; however, the role of afferent feedback in the regulation of intensity during self-paced exercise has been debated (Marcora, 2009, 2010; Amann and Secher, 2010).

Even though there is an increasing research interest in effects of mental fatigue on endurance performance and pacing it has to be noted that the majority of the published studies generally used artificial and relatively short-term cognitively demanding tasks to induce mental fatigue (e.g., 30 to 90 min Stroop-tests). These tasks might not be directly transferable into athletic or everyday environments, thus research looking into the effects of more practically relevant tasks is needed. Additionally, to the author's knowledge no research, to date, has been published examining the effects of accumulated mental fatigue on performance and/or pacing. Such research, however, is warranted regarding that anecdotal reports from coaches and athletes seem to describe mental fatigue as a phenomenon that is rather developed throughout a season than in acute scenarios.

CONCLUSIONS

Much of the research assessing the accuracy or validity of theoretical and mathematical models of pacing has examined the psychobiological response to directly manipulating the pacing of individuals, and/or examined the influence of manipulating kinematics or biomechanics, exercise environment, and/or fatigue development on self-selected pacing strategies. Given the complexity of how humans regulate pace, it is likely that the manipulation of anyone of these factors has considerable effect on a range of aspects important to task performance. As such, understanding the effects of manipulating such factors will aid in better understanding optimal pacing.

AUTHOR CONTRIBUTIONS

All authors listed, have made substantial, direct and intellectual contribution to the work, and approved it for publication.

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frontiers in Physiology

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Cycling in the Absence of Task-Related Feedback: Effects on Pacing and Performance

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Introduction: To achieve personal goals in exercise task completion, exercisers have to regulate, distribute, and manage their effort. In endurance sports, it has become very commonplace for athletes to consult task-related feedback on external devices to do so. The aim of the present study was to explore the importance of the presence of this information by examining the influence of the absence of commonly available task-related feedback on effort distribution and performance in experienced endurance athletes.

Methods: A 20-km cycling time trial was performed. Twenty Participants from a homogenous cyclist population were appointed to a group that did not receive any feedback (NoF), or a group that could consult task-related feedback (i.e., speed, heart rate, power output, cadence, elapsed time, and elapsed distance) continuously during their trial (FF).

Results: The distribution of power output (PO) differed between groups. Most evident is the spurt at the end of the trial of FF, which was not incorporated by NoF. Nevertheless, no between-group differences were found in performance time (FF: 28.86 ± 3.68 vs. NoF: 30.95 ± 2.77 min) and mean PO controlled by body mass (FF: 3.61 ± 0.60 vs. NoF: 3.43 ± 0.38 W/kg). Also, no differences in rating of perceived exertion scores were found.

Conclusion: The current study provides a first indication that prior knowledge of task demands together with reliance on bodily and environmental information can be sufficient for experienced athletes to come to comparable time trial performances. This questions the necessity of the presence of in-race instantaneous task-related feedback via external devices for maximizing performance. Moreover, it seems that different pacing strategies emerge depending on sources of information available to experienced athletes.

Keywords: energy regulation, external device, information, end spurt, race strategy, time trial

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INTRODUCTION

Athletes are continuously required to make decisions whether to persist in a given behavior or switch to a different one, balancing performance goals against threats of premature exhaustion. Such a dilemma is not limited to the sport context. Engagement in physical activity and a healthy lifestyle requires the selection of appropriate and comfortable intensities for a particular duration to stay sufficiently active. The goal-directed distribution and management of effort across the duration of an exercise bout is also known as *pacing* (Edwards and Polman, 2012). There is an ongoing debate about what influences the selection of an optimal pacing strategy (Smits et al., 2014) or why individuals select a strategy that is too intense, causing premature fatigue, or too conservative, resulting in poor performance or lack of physiological adaptations (Renfree et al., 2014). In view of improving the current understanding of the factors relevant in determining effort distribution in ongoing exercise, the current study considered the importance of commonly available task-related feedback for decision-making in pacing in endurance cyclists.

Pacing and performance can only be optimized if athletes make decisions based on the most relevant information (Renfree et al., 2014). A recent review (Smits et al., 2014) initiated a framework in which pacing is considered as a continuous decision-making process, fuelled by reciprocal interactions between processes internal to the athlete and the environment in which the athlete acts. In addition, it was suggested that the use of bodily and environmental information should not be considered in isolation for a given moment, but also in anticipation to factors such as knowledge of the likely demands of the remaining exercise bout (e.g., certainty about the endpoint and duration) and personal goals (Smits et al., 2014). Moreover, prior experience has been indicated to be important in successfully completing pacing tasks (Mauger et al., 2009; Micklewright et al., 2010; Edwards and Polman, 2013; Smits et al., 2014).

In endurance sports, it has become commonplace for athletes to consult task-related feedback (e.g., current speed, cadence, heart rate, power output, elapsed time and elapsed distance) on external devices. The contribution of such feedback has been critically examined in existing research in the area of deception and pacing strategies (Jones et al., 2013). Research with deceptive feedback-interventions during endurance trials has indicated that (a) pacing strategy selection is based on the perceived distance of a time trial rather than the actual distance (Nikolopoulos et al., 2001); (b) athletes deceived of the actual distance completed the subsequent performance trial based on perceived effort rather than on actual distance (Paterson and Marino, 2004); (c) pacing is influenced by an interaction between feedback and previous experience (Micklewright et al., 2010); and (d) time trial performance does not differ between accurate and inaccurate split-time feedback conditions (Wilson et al., 2012).

Non-deceptive feedback studies have also considered the relation between task-related feedback and pacing. No performance differences were found between groups of inexperienced participants that either did or did not receive prior knowledge of distance and distance feedback during 4-km cycling time trials. It was suggested that the inexperienced participants who did receive task-related feedback demonstrated a greater reliance on afferent feedback (e.g., from heart, lungs, skeletal muscles) than on task-related feedback, and were conservative when setting a pacing strategy (Williams et al., 2012). Other research (Foster et al., 2009) found cautiousness during early trials within unexperienced but fit participants, followed by progressively increased effort during later trials as participants became more confident that the time trial could be completed without unreasonable levels of exertion. It was stated that this cautiousness is not unlike the slower speed of completion that is typically observed in motor learning tasks adopted to reduce errors. A study in which groups of experienced participants did or did not receive prior knowledge of distance and distance feedback during 4-km cycling time trials found better initial trial performance within the group that received feedback (Mauger et al., 2009). This indicates that athletes may choose to pace themselves according to task-related feedback if their experience supports this as a successful strategy (Micklewright et al., 2010). Finally, it has been suggested that it is not the task-related feedback itself that is important, but how an athlete interprets and acts upon it (Micklewright et al., 2010). For example, athletes decided to start an end spurt when they believed that an exercise task is 90% completed (Catalano, 1973).

If pacing is considered as a buffering mechanism to enable successful completion of certain strenuous tasks, then prior experience and accurate knowledge of the task demands are crucial to success (Edwards and Polman, 2013). When we consider prior experience in pacing as familiarity with interpreting and acting upon instantaneous bodily and environmental information in anticipation to likely demands of the remaining task and personal goals, it can be hypothesized that athletes who have gained such experience actually do not need task-related feedback from external devices to successfully complete a task of which the demands are known; even though the task as such might be rather novel, such as cycling a road cycling time-trial. No endurance exercise studies have been found focussing on the necessity of the presence of in-race instantaneous task-related feedback that is nowadays commonly available via external devices (e.g., bike computer, running watch). Therefore, the aim of the present study was to examine the influence of an absence of commonly available task-related feedback on effort distribution and performance in experienced endurance athletes while riding a time trial. To do so, pacing (i.e., power-distribution) and performance during a 20-km cycling time trial of a group that did not receive any instantaneous task-related feedback (NoF) was compared with a group that could consult task-related feedback continuously during the trial (FF). Based upon the above, we expected no inferior performance in NoF compared to FF.

MATERIALS AND METHODS

Participants

A homogenous group of 20 experienced and trained [i.e., "performance level 3" (De Pauw et al., 2013)] male

cyclists/triathletes (6.4 ± 5.5 years of experience in their sports and 4.6 ± 2.4 training bouts per week), familiar with the process of pacing in their sports, was selected and completed the Physical Activity Readiness Questionnaire (Thomas et al., 1992) and provided written informed consent. The study was approved by a local Ethics Committee and conformed with the Declaration of Helsinki.

Research Design

All participants completed an incremental cycling exercise test (*ICET*) to volitional exhaustion to determine maximal cardiorespiratory values. Furthermore, each participant performed a 20-km cycling time trial as fast as possible while being randomly allocated to an experimental group that received no feedback (NoF) or a control group that was allowed full feedback (FF). Participants did not perform a familiarization trial, as we were interested in imposing a relatively novel task such as cyclists in the Grand Tours are experiencing: each time trial or stage is different, cycled under different conditions. Imposing a familiarized time trial condition in a repeated measures design—instead of a rather novel task in our current design—would compromise ecological validity of the study when interested in road cycling. In addition, we expected that the importance of feedback would be higher in a rather novel task.

All tests were performed in a laboratory with conditioned temperature and relative humidity.

Incremental Cycling Exercise Test (ICET)

The ICET was performed on a cycle ergometer (Lode Excalibur; Lode BV, Groningen) at a pedal frequency of 80 rpm. After a 10 min warming-up at a work rate of 150 W and 1 min passive rest, the test started on an exercise intensity which was equivalent to 3 W/kg*[participant's body mass, kg]. This equivalent provided comparable relative starting exercise intensities for all participants and corresponded to a power output (PO) that would elicit ∼65–70% of maximal oxygen consumption (VO_{2max}; Hawley and Noakes, 1992; Rønnestad et al., 2011). PO was increased every 2 min by 30 W until the participant reached volitional exhaustion (i.e., cadence <80 rpm). PO, heart rate (HR), Rating of Perceived Exertion [RPE; Category Ratio version ranged from 0 to 10 (Borg, 1982)], rate of oxygen consumption, and carbon dioxide production were recorded for further analysis. Respiratory gas exchange was measured breath-by-breath using open-circuit spirometry (Oxycon Delta; Enrich Jaeger, Hoechberg, Germany). Before each test, the gas analyser was calibrated using a Jaeger 3-L syringe, room air, and a standard gas mixture (5.04% CO_2). HR was recorded every 2 s (Polar Electro, Kempele, Finland).

Time Trial

Participants conducted the trial using their own bike mounted on an ergotrainer (Tacx Flow T1680, Wassenaar, The Netherlands). A power meter (CycleOps PowerTap Elite+, Madisson, USA; sample frequency: 1 Hz, accuracy: ±1.5%) was used to record PO, time and covered distance during each trial for subsequent data-analysis. Previous research has shown that this power meter provides valid and reliable PO measurements in laboratory tests (PO range: 100–450 W; Bertucci et al., 2005). Also, participants were asked to rate their perceived exertion (RPE) at least once within every 4-km block, but at irregular intervals (i.e., after 4, 6, 11, 15, 18, and 20-km of the trial completed for the participants in both groups) to avoid that it would provide the feedback-blinded participants any distance or time feedback indirectly. It should be noted that, because the Tacx does not incorporate the non-linear relation between PO and velocity, 20-km cycling on a Tacx is not fully identical to 20-km on the road outside or, for example, on a Velotron ergometer.

Full Feedback (FF) Control-Group and No Feedback (NoF) Experimental-Group

For participants allocated to FF ($n = 10$), task-related feedback was provided during the entire trial. As a result, they could continuously consult their PO, speed, HR, cadence, covered distance, and time elapsed. Participants appointed to NoF ($n = 10$) did not receive any feedback during the trial ("blinded"). They only knew they had to cycle 20-km as fast as possible and a stop-sign would be provided when they covered this distance.

Within this experimental design the performance-environment (i.e., exercising in the laboratory) and -goal (i.e., completing the trial as fast as possible) were the same for both groups. However, whereas NoF-participants were reliant on their own resources (i.e., perceived bodily exertion and prior experience with performing time trials) during their trial, participants within FF were able to evaluate their perceived bodily exertion, interim performance, and future task demands via external devices.

Preparing Data for Analysis

To examine the pacing strategy and performance of both groups over the trial, participants' PO-distribution curves were considered. In order to compare the PO-distribution between FF and NoF, the mean PO-distribution curves of both groups over the entire trial were established. To do so, first we normalized the PO-distribution curve of each participant to 1250 data points. This number of data points was based on the completion time in seconds of the fastest participant. Following this, the power data was controlled for body mass differences between participants [i.e., participants' PO throughout the trial divided by their body mass (PO, W/kg)]. In addition to considering PO-data (i.e., PO), we were also interested in how the groups relatively distributed their PO over the trial and how the groups' PO was related to the maximal PO-capacity of the participants within the groups. As a consequence, participants' PO throughout the trial was divided by their mean PO over the trial [PO_{rel}, –], as well as divided by their peak PO established during ICET [PO_{ICET}, –].

To compare overall performance between FF and NoF, calculated group-means of PO and PO_{ICET}, and of the performance time [PT] were used. Furthermore, to consider whether there were differences in PO between and within groups at different intervals within the trial, the PO- and PO_{rel}-distributions were divided into 10 equal-sized segments (from now on to be called *10%-segments* and abbreviated with *S1* till

S10, whereas S1 = 0–10%; S2 = 10–20%; etc.). Also, paired differences between neighboring 10%-segments (from now on to be called *change-segment* and abbreviated with *CS1* till *CS9*) were calculated (i.e., CS1 = S2–S1; CS2 = S3–S2; etc.) to examine whether PO-changes over subsequent 10%-segments within the groups differ between the groups. Finally, to consider whether RPE differed between groups, RPE group means were calculated for each time the participants rated their perceived exertion during the trial.

Analysis

To determine whether there were between-group differences in anthropometric characteristics, and ICET- and overall-performance measures, independent *t*-tests were conducted. Repeated measures ANOVA's were used to examine the effects of feedback condition on PO at different parts during the race (i.e., 10%-segments) and PO-changes over the race (i.e., change-segments). If a main effect for group was found, Bonferroni corrected independent *t*-tests were performed to consider within which specific segment(s) PO differed between groups. If a main effect for segment was found, Bonferroni corrected paired-samples *t*-tests were performed to consider which specific neighboring 10%-segments of PO differed from each other within groups.

Finally, to consider differences in perceived exertion between groups, independent *t*-tests on mean RPE-scores were performed. As RPE was asked at irregular intervals, no repeated measures ANOVA was applied for the RPE-scores analysis.

Effect sizes were calculated as appropriate. An effect size of 0.2 is considered as small, 0.5 as medium, and >0.8 as large (Cohen, 1992). For all tests a two tailed significance was used with an alpha of 0.05.

RESULTS

Participants

The group characteristics are provided in **Table 1**. No between-group differences were found in anthropometric characteristics and cardiorespiratory values.

Overall Performance

Figure 1 illustrates the mean PO-distribution curves over the entire trial per group (FF top left and NoF top right) and for both groups together (bottom). To visualize how PO over the trial is related to the peak PO established during ICET (PPO), a 70%*PPO-boundary per group (dotted lines) is incorporated. The mean PO-distribution curve of FF is usually above or at the 70%*PPO-boundary, whereas the curve of NoF is usually situated at or below the boundary. Nevertheless, the higher mean PO_{ICET} in FF [0.73 ± 0.06 (–)], compared to NoF [0.68 ± 0.06 (–)], was not significant, but accompanied by a large effect size (Cohen's $d = 0.85$). Also, differences in mean PT (FF: 28.86 ± 3.68 vs. NoF: 30.95 ± 2.77 min; Cohen's $d = 0.64$) and mean PO (FF: 3.61 ± 0.60 vs. NoF: 3.43 ± 0.38 W/kg; Cohen's $d = 0.37$) between groups were not significant, which indicates an absence of performance differences between groups.

TABLE 1 | Comparison of anthropometric characteristics and ICET-measures [Mean (*SD*)] of 20 male endurance athletes divided into two groups.

	FF[a]	NoF[a]	*p*-value	*d*	*r*
Age (years) at first test[b]	28.2 (7.8)	27.2 (5.4)	0.91	–	0.034
Height (cm)	186 (5)	188 (6)	0.28	0.50	–
Body mass (kg) at ICET[c]	78.7 (7.9)	76.1 (10.4)	0.54	0.28	–
HR_{max} (bpm)[d]	196 (10)	194 (7)	0.66	0.20	–
PPO (W)[e]	387 (50)	381 (33)	0.73	0.14	–
PPO (W/kg)[e]	4.95 (0.67)	5.04 (0.46)	0.72	0.16	–
VO_{2max} ($ml \cdot min^{-1}$)[f]	4220 (685)	4473 (576)	0.40	0.40	–
VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$)[b,f]	53.7 (7.1)	59.0 (7.7)	0.095	–	0.38

[a] *FF, Full Feedback control-group (n = 10); NoF, No Feedback experimental-group (n = 10);*
[b] *For the variables which violated assumptions of normal distribution, Mann-Whitney U-Tests were used;*
[c] *ICET, incremental cycling exercise test;*
[d] *HR_{max}, maximal heart rate;*
[e] *PPO, peak power output;*
[f] *VO_{2max}, maximal oxygen consumption; because of an abnormal result in the VO_{2max}-result of one of the participants in NoF, this result has been excluded. Therefore, $n_{NoF} = 9$ for VO_{2max} ($ml \cdot min^{-1}$) and VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$). No differences were found.*

Segment Performance within Groups

Figure 2 shows the 10%-segments for both PO and PO_{rel} per group. A segment main effect was found for both PO and PO_{rel} within FF [respectively $F_{(1.66)} = 5.12$; $P = 0.02$, and $F_{(1.70)} = 4.89$; $P = 0.03$]. *Post-hoc* comparisons revealed that mean PO in FF was higher in S10, compared to S9, for both PO [$t_{(9)} = -5.97$, $P < 0.001$; Cohen's $d = 0.77$] and PO_{rel} [$t_{(9)} = -6.07$; $P < 0.001$; Cohen's $d = 1.87$], whereas mean PO in S3 was lower than in S2 for PO [$t_{(9)} = 3.96$; $P = 0.003$; Cohen's $d = 0.09$] and nearly for PO_{rel} [$t_{(9)} = 3.68$; $P = 0.005$; Cohen's $d = 0.32$]. There was no significant main effect for NoF.

Segment Performance between Groups

A group by segment interaction effect was found for both PO [$F_{(1.74)} = 3.97$; $P = 0.03$] and PO_{rel} [$F_{(1.77)} = 3.95$; $P = 0.03$]. *Post-hoc* comparisons revealed that mean PO in S10 was higher in FF, compared to NoF, for PO_{rel} [$t_{(18)} = 4.94$; $P < 0.001$; Cohen's $d = 2.21$] and nearly for PO [$t_{(18)} = 3.03$; $P = 0.007$; Cohen's $d = 1.36$], whereas mean PO in S5 was higher in NoF for PO_{rel} [$t_{(18)} = -3.36$; $P = 0.003$; Cohen's $d = 1.50$].

Table 2 provides an overview of the change-segments for both PO and PO_{rel} per group. A group by segment interaction effect was found for both PO [$F_{(3.17)} = 8.14$, $P < 0.001$] and PO_{rel} [$F_{(2.93)} = 7.81$; $P < 0.001$]. *Post-hoc* comparisons revealed that the mean change in PO was higher in FF, compared to NoF, for both PO [$t_{(12.14)} = 6.08$; $P < 0.001$; Cohen's $d = 2.72$] and PO_{rel} in CS9 [$t_{(12.95)} = 6.06$; $P < 0.001$; Cohen's $d = 2.71$].

The segment analysis indicates that the PO-distribution of the groups differed from each other. Most evident is the spurt at the end of the trial of FF, which was not incorporated by NoF. In

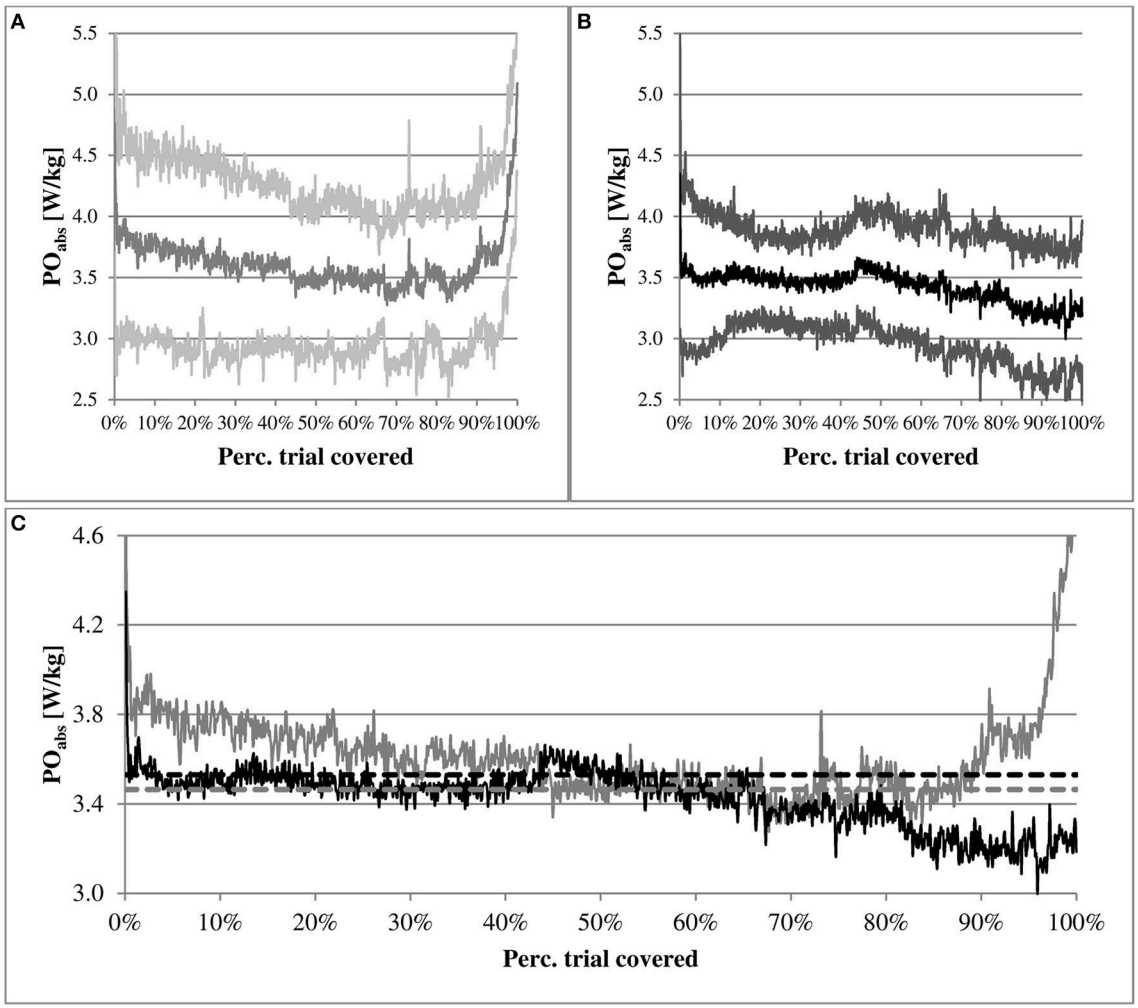

FIGURE 1 | Mean distribution curves per group of participants' power output divided by their body mass (PO). On the top left **(A)** the curve of the Full Feedback (FF) group, and on the top right **(B)** the curve of the No Feedback (NoF) group. The brighter upper and lower curves within both top graphs represent the standard deviations. On the bottom **(C)** the curves of FF (gray) and NoF (black) together. The bottom graph also includes two dotted straight lines that represent boundaries corresponding with 70% of the peak PO established during the incremental cycling exercise test of FF (gray) and NoF (black).

contrast, NoF increased their PO halfway through the trial and FF did not.

Perceived Exertion

No differences in perceived exertion scores were found (see **Figure 3**).

DISCUSSION

The main aim of the current study was to examine the effects of an absence of task-related feedback on effort distribution and performance in experienced endurance athletes. To do so, pacing and performance during a 20-km cycling time trial of a group that could not consult task-related feedback (NoF) were compared with a group for whom task-related feedback was provided during the entire trial (FF). The results show no spurt at the end of the trial of NoF, whereas FF incorporated an end spurt. Notwithstanding this and other differences in pacing strategy between groups, no difference in overall performance between groups was found. This supports our hypothesis to find no inferior performance in NoF compared to FF. This finding suggests that in middle distance exercise, experienced athletes do not need task-related feedback from external devices to successfully complete a task of which the demands are known. However, the difference in pacing behavior visible toward the end of the race indicates that task-related feedback influences certain aspects of decision-making regarding how and when to invest the available energy over the race.

The lack of performance differences between groups contrasts with the suggestion that cautiousness and a slower speed of completion—designed to reduce errors (e.g., premature exhaustion)—is typically observed in performing motor tasks someone is unfamiliar with (Foster et al., 2009). The PO of NoF was usually at or below the 70%*PPO-boundary, whereas FF

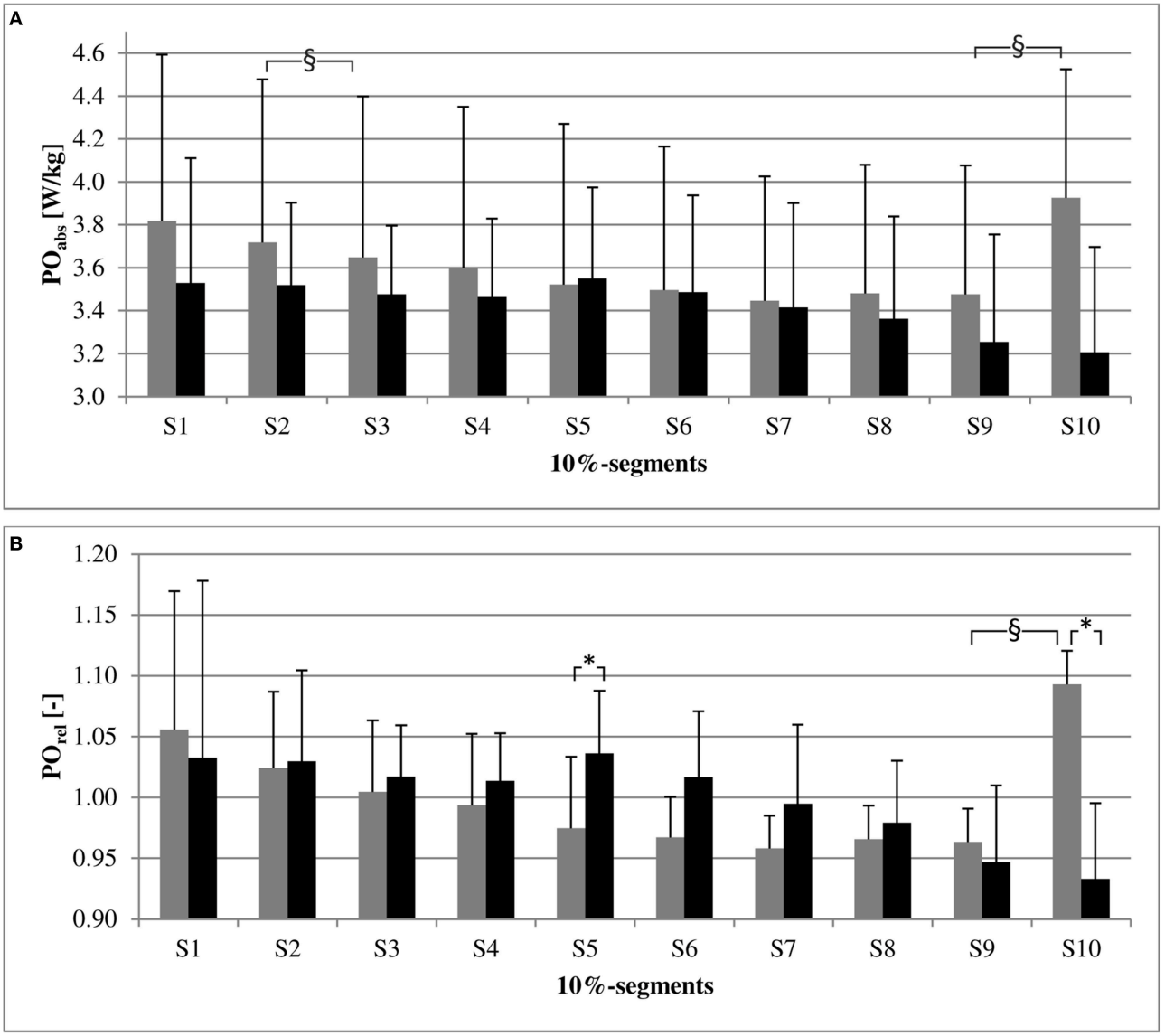

FIGURE 2 | Comparison of power output (PO) characteristics [Mean (*SD*)] of 10%-segments between and within groups ($n = 10$ per group) for PO (top graph, A) and PO_{rel} (bottom graph, B). PO, Mean of participants' power output (PO) divided by their body mass; PO_{rel}, Mean of participants' PO divided by their mean PO over the trial; Gray bars, Full Feedback group; Black bars, No Feedback group; 10%-segments, the PO- and PO_{rel}-distributions were divided into 10 equal-sized segments (S1 = 0–10%; S2 = 10–20%; etc.). Significant between group differences are marked by * and within group differences by §.

usually exercised above or at the boundary. Although this finding could suggest that NoF might have included some cautiousness within their pacing strategy, between group analyses of overall performance, PO-segments and RPE did not indicate an obvious structural conservativeness in NoF's pacing strategy compared to FF.

Performance

A study that compared the performances between groups that did or did not receive distance feedback during multiple 4-km cycling time trials found a better initial trial performance within the group that received distance feedback (Mauger et al., 2009). However, in our study feedback-blinded participants had prior knowledge of the demands (i.e., distance to be covered) of the trial. It has been argued that experience developed during previous (training) bouts reinforces interoceptive sensitivity (Baron et al., 2011). Our participants were experienced in performing exercise bouts of different intensities and duration, and in different environmental circumstances, which makes it possible that they have gained an experience-based awareness of the effort they are able to sustain for endurance trials with different demands (Foster et al., 2004; Hettinga et al., 2006). The absence of feedback-devices meant that our NoF-participants were solely reliant on their own resources (i.e., perceived bodily

TABLE 2 | Difference between neighboring 10%-segments within groups (i.e., change-segments).

	PO [W/kg][b]			PO_{rel} [–][c]		
Change-segment	**FF[a]**		**NoF[a]**	**FF**		**NoF**
CS1[e] (=S2–S1[d])	−0.100 (0.277)		−0.011 (0.283)	−0.031 (0.082)		−0.003 (0.083)
CS2 (=S3–S2)	−0.070 (0.056)		−0.043 (0.168)	−0.020 (0.017)		−0.013 (0.049)
CS3 (=S4–S3)	−0.049 (0.120)		−0.007 (0.101)	−0.011 (0.031)		−0.003 (0.032)
CS4 (=S5–S4)	−0.079 (0.147)		0.082 (0.161)	−0.019 (0.037)		0.023 (0.047)
CS5 (=S6–S5)	−0.025 (0.070)		−0.064 (0.135)	−0.008 (0.019)		−0.020 (0.040)
CS6 (=S7–S6)	−0.051 (0.149)		−0.071 (0.080)	−0.009 (0.046)		−0.022 (0.025)
CS7 (=S8–S7)	−0.034 (0.117)		−0.052 (0.111)	0.008 (0.027)		−0.016 (0.033)
CS8 (=S9–S8)	−0.004 (0.084)		−0.109 (0.126)	−0.002 (0.024)		−0.032 (0.038)
CS9 (=S10–S9)	0.449 (0.238)	[*]	−0.048 (0.101)	0.130 (0.068)	[§]	−0.014 (0.032)

[a] *FF, Full Feedback control-group (n = 10); NoF; No Feedback experimental-group (n = 10);*
[b] *PO, Mean of participants' power output (PO) divided by their body mass;*
[c] *PO_{rel}, Mean of participants' PO divided by their mean PO over the trial;*
[d] *S1–10: 10%-segments;*
[e] *CS1–9: difference between neighboring 10%-segments.*
*Found significant differences within the post-hoc Bonferroni corrected independent t-tests for PO and PO_{rel} are marked by, respectively, * and §.*

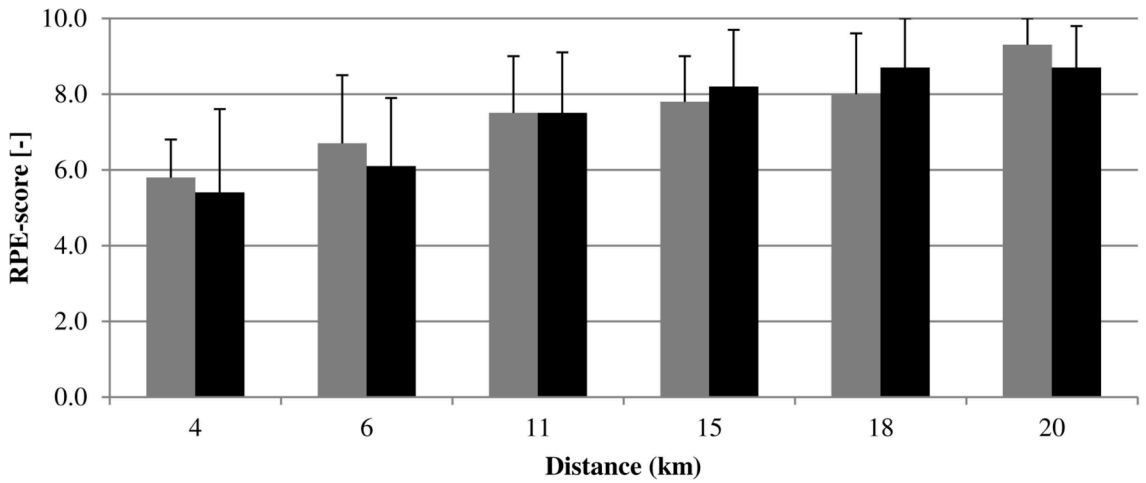

FIGURE 3 | Comparison of RPE-characteristics [Mean (*SD*)] between groups (*n* = 10 per group) for several moments during the trial. Gray bars, Full Feedback group; Black bars, No Feedback group; Distance (km), Completed distance (km) within the trial at which the RPE was asked, in which was taken into account that within each 4-km block the RPE was asked at least once. No differences were found.

exertion and prior time trial experience) and prior knowledge of the task demands while distributing their effort over the trial. With this in mind, together with the fact that no performance differences were found between groups, it can be suggested that prior knowledge of task demands together with reliance on bodily information is sufficient for experienced athletes to come to comparable time trial performances when receiving full feedback.

Effort Distribution and Perceived Exertion

The within-group analysis of power distribution indicates that FF demonstrated a fairly intensive initial phase, followed by a moderate steady middle part, and finishing with an end spurt. Such a parabolic-shaped (i.e., U- or J-shape) strategy is often observed in endurance exercise (Edwards and Polman, 2012). In contrast, NoF showed limited variability in PO within their trial. Moreover, PO- and relative PO-changes differed between groups during the end phase. No PO-change in NoF during the last 10% of the trial was demonstrated, compared to the penultimate 10%, whereas a significant PO-increase in FF during the last 10% was shown. An important implication is that different pacing strategies emerge depending on sources of information available to experienced athletes. Future studies should focus on addressing which information is of importance at what segment of the race, for example by studying gaze behavior and introducing or retracting sources of information during the race (Boya and Micklewright, 2016).

With regard to the end phase; it has been argued that athletes often utilize their remaining energetic reserves—maintained in order to avoid premature exhaustion—in a spurt when they believe they are close to the endpoint of the task (Catalano,

1973; De Koning et al., 2011). The absence of instantaneous task-related feedback made that NoF, in contrast to FF, never had explicit certainty about the remaining distance to be covered, which could have been a considerable interference with determining the moment at which they could exploit their energy reserves. This, in turn, might have prevented them from appealing to their remaining energetic reserves, even though the end phase of the trial was reached. If this were the case, the absence of explicit endpoint knowledge would induce conservativeness during the end phase and hinder maximizing performance. Such a conservative end phase should have led to finishing less exerted compared to finishing with an end spurt. However, this was not supported by our RPE data. Future studies are needed to further explore what will happen when for example introducing endpoint information in the last phase of the race, or what will be the effect of an opponent. In 4-km time trials with known end-point, athletes adapt their strategies to the behavior of their opponent (Konings et al., 2016a). Is this also the case in open-loop exercise?

Taking into account the absence of overall performance- and RPE-differences between groups, together with the limited varied PO-distribution of NoF, it could be suggested that NoF decided to pursue a pacing strategy that enabled personal goal achievement without the incorporation of an end spurt. This pre-planned pacing strategy would be in anticipation to the prior knowledge of the task demands and the knowledge that they would never have explicit certainty of reaching the point after which they could exploit their energy reserves in a spurt. This reasoning fits with recent pacing ideas that decision-making in pacing is based on instantaneous bodily and environmental information, as well as in anticipation to factors such as knowledge of the likely demands of the remaining exercise bout (e.g., certainty about the endpoint and duration) and personal goals (Smits et al., 2014); and pre-planning a pacing strategy using an appropriate situation-specific strategy may be a useful way to distribute effort and optimize performance for that event (Edwards and Polman, 2012).

Within the overall pacing strategy of NoF, characteristics can be recognized from a combination of an evenly paced (i.e., steady PO) and all-out paced (i.e., attempting to maintain a challenging PO for the duration of the bout) strategy. If this is the case, participants in NoF possibly pursued a particular relatively steady but challenging pace they expected to be sustainable for their estimated durations of the trial (possibly based on their experience-based effort-awareness (Hettinga et al., 2006) and including a certain safety margin) and provided a performance that can compete with performances in familiar circumstances as well. The aim of an all-out strategy is to maintain a challenging PO for the duration of the bout, but practical observations suggest PO will deteriorate (Edwards and Polman, 2012); as can also be observed during the end phase of the overall PO-distribution of NoF. Keeping a challenging pace, in turn, should eventually have elicited a considerable perceived exertion in NoF, which can explain why NoF's final RPE does not significantly differ from that of the end sprinting FF-group.

Pacing and Task-Related Feedback

We examined how the absence of task-related feedback affected time trial execution in experienced athletes, in which the effects of the absence of distance feedback eventually seemed to be most affecting in strategy selection. However, we do not exclude that other task-related feedback could also have been integrated into the decision-making in pacing in FF. Recent research with eye-tracking measurements (Boya et al., 2015) has demonstrated that experienced cyclists who could consult speed-, distance-, PO-, cadence-, HR-, and time-feedback mainly directed their gaze to speed and distance information during their trials. Moreover, it has been suggested that cyclists may choose to pace themselves according to speed feedback if their experience supports this as a successful strategy (Micklewright et al., 2010). Our results further elaborate on the idea that an experience-based awareness of the effort one is able to sustain for different durations of exercise seems robust in time trial exercise (Hulleman et al., 2007). The current study provides a first indication that task-related feedback on external devices, including speed feedback, seems not essential for experienced athletes to come to a comparable endurance performance. This further confirms that interpreting and acting upon bodily information is important in pacing (Smits et al., 2014) and hence recommends exercisers of all levels to pay (more) attention to developing familiarity with self-monitoring (i.e., interpret) and self-regulation (i.e., act) in improving their pacing skills. Also, our results could act as an entry point for reconsidering the way in which task-related feedback on external devices should be used during exercise tasks.

Finally, our results indicate that the consultability of distance feedback (i.e., possibility to gain precise endpoint knowledge) influences effort distribution; which was most obvious during the end phase of the trial. Exercising some cautiousness and (consequently) making situation-based (pre-planned) adjustments to the pacing strategy were proposed as possible consequences of the absence of distance feedback, but our results are not fully conclusive about this. It has already been demonstrated that fit participants with limited specific endurance sports experience were cautious during initial trials (Foster et al., 2009). During later trials, they made adjustments in their strategy and progressively increased effort as they became confident that the time trial could be completed with a particular strategy without negative consequences. Future research with multiple endurance trials should reveal whether such a learning-effect will also occur within experienced feedback-blinded athletes. Lastly, in exercisers' natural (competitive) environment, properties such as optic flow (Parry et al., 2012) as well as the presence of opponents (Konings et al., 2016b) have been shown to be of influence on performance and decision-making in pacing. Such properties were not incorporated in the present experimental set-up as yet. Future research should thus also be arranged with experimental conditions that are representative of the exercisers' natural environments (Smits et al., 2014) to explore the impact of environmental properties on exercise performance and pacing while external feedback devices are present or not.

AUTHOR CONTRIBUTIONS

FH and BS designed the study protocol, BS and RP conducted the initial data analysis. All authors contributed to interpretation and conception of the work as well as further development of the proposed ideas. BS wrote a first draft of the work. All authors

were involved in further drafting, editing, and final approval of the manuscript.

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REVIEW
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Effect of Environmental and Feedback Interventions on Pacing Profiles in Cycling: A Meta-Analysis

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In search of their optimal performance athletes will alter their pacing strategy according to intrinsic and extrinsic physiological, psychological and environmental factors. However, the effect of some of these variables on pacing and exercise performance remains somewhat unclear. Therefore, the aim of this meta-analysis was to provide an overview as to how manipulation of different extrinsic factors affects pacing strategy and exercise performance. Only self-paced exercise studies that provided control and intervention group(s), reported trial variance for power output, disclosed the type of feedback received or withheld, and where time-trial power output data could be segmented into start, middle and end sections; were included in the meta-analysis. Studies with similar themes were grouped together to determine the mean difference (MD) with 95% confidence intervals (CIs) between control and intervention trials for: *hypoxia, hyperoxia, heat-stress, pre-cooling,* and various forms of *feedback*. A total of 26 studies with cycling as the exercise modality were included in the meta-analysis. Of these, four studies manipulated oxygen availability, eleven manipulated heat-stress, four implemented pre-cooling interventions and seven studies manipulated various forms of feedback. Mean power output (MPO) was significantly reduced in the middle and end sections ($p < 0.05$), but not the start section of hypoxia and heat-stress trials compared to the control trials. In contrast, there was no significant change in trial or section MPO for hyperoxic or pre-cooling conditions compared to the control condition ($p > 0.05$). Negative feedback improved overall trial MPO and MPO in the middle section of trials ($p < 0.05$), while informed feedback improved overall trial MPO ($p < 0.05$). However, positive, neutral and no feedback had no significant effect on overall trial or section MPO ($p > 0.05$). The available data suggests exercise regulation in hypoxia and heat-stress is delayed in the start section of trials, before significant reductions in MPO occur in the middle and end of the trial. Additionally, negative feedback involving performance deception may afford an upward shift in MPO in the middle section of the trial improving overall performance. Finally, performance improvements can be retained when participants are informed of the deception.

Keywords: pacing, cycling, hypoxia, deception, hyperoxia, heat-stress, pre-cooling, feedback

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INTRODUCTION

The ability to appropriately distribute energy expenditure throughout an exercise task is critical in order to optimize athletic performance (St. Clair Gibson and Noakes, 2004; Abbiss and Laursen, 2008). In the sport science literature this is known as "pacing" or the "pacing strategy" or "pacing profile" and refers to the self-regulation of power (or velocity) during athletic competitions in which athletes are free to vary their exercise intensity (de Morree and Marcora, 2013; Skorski et al., 2015). Research aimed at understanding the underlying mechanisms influencing the selection of pace during exercise has dramatically increased within the last decade. Based on current research, pacing appears to be regulated by complex relationships between the brain and other physiological systems (St. Clair Gibson and Noakes, 2004; Abbiss and Laursen, 2008). Several models have been proposed to explain this phenomena including: the teleoanticipatory theory (Ulmer, 1996; St. Clair Gibson et al., 2006), the central governor model (Noakes et al., 2001), the perception based model (Tucker, 2009), the pacing awareness model (Edwards and Polman, 2013) and the psychobiological model (Marcora, 2010; Pageaux, 2014). Many of these models, however not all, acknowledge that afferent sensory feedback from various physiological systems is received and regulated within the brain and integrated into the pacing strategy as a person responds to ongoing internal stimuli, as well as environmental factors and other external stimuli (Noakes et al., 2001, 2005; St. Clair Gibson and Noakes, 2004). In addition, factors such as knowledge of the task duration or distance remaining (Swart et al., 2009), memory of prior experiences (Mauger et al., 2009), and motivation and mood (de Morree and Marcora, 2013) are also thought to be important factors in the regulation of exercise intensity.

In recent years, a number of studies have shown that physiological, psychological and environmental factors can affect overall performance and pacing (Tucker and Noakes, 2009). These factors include oxygen availability (Amann et al., 2006; Clark et al., 2007; Tucker et al., 2007; Périard and Racinais, 2016), heat-stress (Peiffer and Abbiss, 2011), wind velocity (Teunissen et al., 2013), hydration status (Dugas et al., 2009), carbohydrate (Abbiss et al., 2008) and caffeine ingestion (Wiles et al., 2006), pre-cooling strategies (Duffield et al., 2010), motivation (Corbett et al., 2012), fatigue (Skorski et al., 2015), deception (Stone et al., 2012; Jones et al., 2016b; Shei et al., 2016), pacing feedback (Thompson et al., 2003, 2004) and music (Atkinson et al., 2004). However, on the basis of existing studies it is still difficult to arrive at an overall conclusion as to whether these manipulations have a negative or positive effect on pacing and performance, and indeed which part of the pacing strategy changes (start, middle and end) during trials.

Recently a number of reviews have attempted to explain the influence of deception (Jones et al., 2013; Williams et al., 2014), decision making (Edwards and Polman, 2013; Renfree et al., 2014; Smits et al., 2014; McCormick et al., 2015) and neurophysiological determinants (Roelands et al., 2013) on pacing. However, to date, a meta-analysis investigating the effect of different environmental and extrinsic manipulations on the actual pacing strategy of trained participants during self-paced time-trials is still lacking. This study resolved to estimate the probability that a difference in pacing strategy, due to interventions such as environmental stressors or feedback manipulation, is practically meaningful. Therefore, the aim of this study was to conduct a meta-analysis to provide an overview as to how these types of manipulations affect pacing strategy and exercise performance.

METHODS

A computerized literature search was undertaken, using 21 different key terms ("athletes," "pacing," "strategy," "hypoxia," "hyperoxia," "heat," "precool," "feedback," "deception," environment; "profile," "self-paced," "exercise," "teleoanticipation," "central," "peripheral," "fatigue," "time-trial," "performance," "experience," and "perceived exertion") based on the PRIMSA checklist (Liberati et al., 2009; Beller et al., 2013) and the search strategy proposed by Higgins (2011). Combinations of these words were used to systematically search databases, from the following databases: PubMed, SPORTDiscus and MEDLINE (via EBSCO). The literature search began in June 2013 and concluded at 1st September 2016 and was complemented with citation tracking of key primary and review articles.

Selection Criteria

Articles were evaluated with respect to their suitability and relevance for the desired context based on the criteria described below. The selection process is also illustrated by a flow chart in **Figure 1**. Studies not fulfilling these criteria but considered important for the topic are included within the discussion.

A study was only included in the meta-analysis if it fulfilled the following requirements:

1. The existence of a control group or condition without any pacing manipulation, i.e., with the subjects acting as their own controls (randomized crossover design).
2. The performance task had to be reported in terms of at least three sections (start, middle and end) to quantify the effect of an intervention on the pacing strategy of the time-trial. Furthermore, each study had to have measured power output as a performance metric. This parameter was the most commonly reported performance metric in the literature where consistent measurement error data was also provided. Therefore, despite a number of pacing studies providing velocity or time data and meeting all other criteria, they were still excluded as they did not provide power output data. As a consequence, modes of exercise researched in the literature such as running, swimming, skating and rowing were excluded.
3. Pacing data had to be reported as mean and standard deviation (SD), and/or standard error of the mean (SEM), either in tables or figures. Three studies presented their data using SEM (Tatterson et al., 2000; Amann et al., 2006; Byrne et al., 2011), to normalize these data they were converted back to SD values by the primary author ($SD = SEM \times \sqrt{N}$).
4. Studies were only included if the trial was self-paced. If this information was not disclosed in the paper, the corresponding

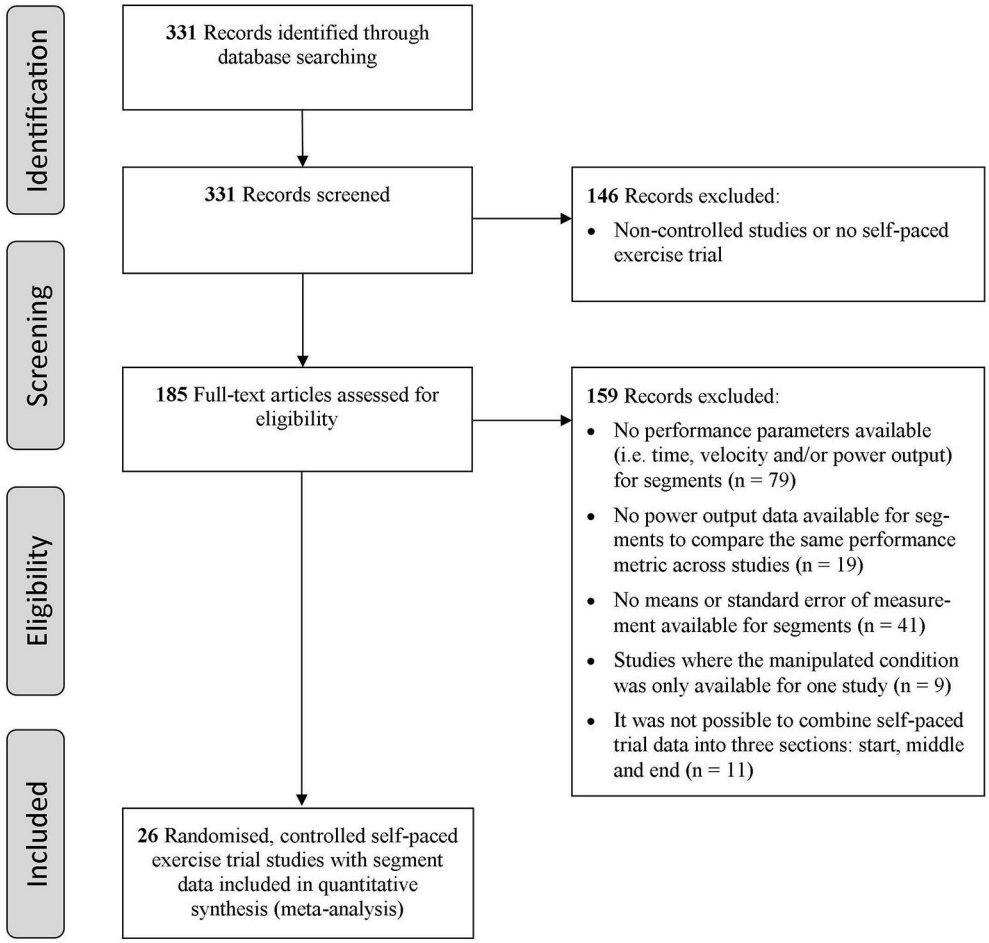

FIGURE 1 | Flow chart summary of the study selection process.

author was contacted. When pacing was influenced by an outside source (e.g., coach or researcher) the study was excluded. Studies manipulating the starting strategy or which incorporated intermittent "efforts" during a trial were also excluded.

5. Studies must have indicated whether or not participants received feedback and the type of feedback received (e.g., elapsed time, distance or power output during the trial). If this information was not disclosed in the paper, the corresponding author was contacted.
6. The study must have been published in an internationally peer-reviewed scientific journal.

Classification of the Studies

Of the initial 185 peer-reviewed studies identified, a total of 26 studies satisfying the inclusion criteria were analyzed. These studies were comprised of 44 different trial comparisons. Studies were coded for the descriptive environmental or extrinsic variables manipulated by the researchers. For example, environmental conditions (fraction of inspired oxygen levels (F_iO_2), temperature, humidity and wind velocity) and feedback received or withheld during the trials. As a result, four different themes (or groups) were identified: (1) oxygen availability (e.g., hypoxia, $F_iO_2 < 0.21$; normoxia ~0.21; iso-oxia, $F_iO_2 > 0.21$ to 0.30; hyperoxia $F_iO_2 > 0.30$), (2) heat-stress, (3) pre-cooling strategies prior to trials in hot conditions (e.g., wearing a cooling vest or cold water immersion), and (4) feedback (e.g., full or no feedback, positive, negative, neutral or informed feedback).

Feedback groups were defined as the following: *full feedback*, where all available feedback was provided and given accurately (control trial); *no feedback*, where all feedback was withheld; *neutral feedback*, where participants raced a virtual on-screen avatar that accurately represented the mean power output (MPO) of a previous performance; *positive deceptive feedback*, where participants were informed they were performing better than in reality (e.g., informed they had traveled a greater distance than they actually had or informed that the ambient and their core temperatures were lower than they actually were); *negative deceptive feedback*, where participants were informed they were performing worse than they were in reality or where performance feedback was inaccurate. For example, performance deception, where participants competed against a previous performance,

where the MPO was increased compared to the previous (baseline) trial and participants were either aware (Jones et al., 2016a,b) or unaware (Jones et al., 2016a,b; Shei et al., 2016); and finally *informed feedback*, where participants completed a final trial after being informed that their previous trial in the presence of a pacer was set at a greater exercise intensity than their baseline trial (Jones et al., 2016a; Shei et al., 2016).

Data Extraction

For all studies, power output was extracted for the control and intervention conditions for MPO of the whole trial and each section of the trial (start, middle and end). A large proportion of the analyzed studies ($n = 25$) displayed their results in figures, hence the mean and SD were measured from plots and error bars by the primary researcher using a hand T-square ruler measuring to the nearest millimeter. Each figure was enlarged to A3 size, printed and fixed to a bench. Mean values were measured from the middle of each plot and SD at the top edge of each error bar for every segment. In order to prevent a bias all measurements were repeated exactly a month later by the same person, following the same protocol. Intra-rater reliability between measurements was calculated using the statistical Software R (Vienna, Austria: R Foundation for Statistical Computing). To estimate the level of agreement between the two measures the intra-class correlation coefficient (ICC) was calculated and interpreted according to the thresholds described by (Landis and Koch, 1977). Analysis revealed an almost perfect correlation between the two measures (ICC = 0.99; 95% confidence intervals (CI): 0.99 and 0.99). We were therefore confident there was minimal researcher and measurement bias when extracting the data for the meta-analysis (Landis and Koch, 1977).

Study Quality Assessment

Although it was not a requirement for the inclusion criteria, the PEDro scale (Machado et al., 2016) was used to quantify methodological quality of included studies. Briefly, the PEDro scale assesses research against 11 criteria related to study design, from which a score can be assigned to a specific paper from 0 to 11. A score ≥ 7 is considered "high quality," a score of 5 or 6 is deemed "moderate quality," and ≤ 4 defined as "poor quality" (Machado et al., 2016).

Data Analysis

The meta-analysis was performed in statistical Software R (Vienna, Austria: R Foundation for Statistical Computing) using the package metaphor (Viechtbauer, 2010). The mean difference (MD) between control and intervention trial for each study was analyzed using a multi-level random effects model including a random effect for start, middle and end sections to account for the dependencies between results from the same studies, represented by 95% confidence internals (CIs). Separate theme analyses for each section were carried out using a random effects model. Heterogeneity was assessed using the Q statistic, described by the I^2 statistic, and publication bias using funnel plots.

For further comparison of pacing strategies we also calculated the pacing index (IP) (Le Meur et al., 2011; Wu et al., 2014) for control and experimental conditions for all studies that reported overall trial MPO. The IP reports the exercise intensity for each segment as a percentage of overall trial MPO and is derived using the following equation:

$$(\textit{Segment mean power output} / \textit{Overall trial mean power output}) * 100$$

As the majority of studies did not report individual participant data it was not possible to calculate a SD for the IP metric and subsequently include in the meta-analysis. Therefore, the IP data were analyzed using an exploratory graphical analysis of the difference between control and experimental condition IP (i.e., control IP—experimental IP for each individual segment).

RESULTS

Search Results

In total 331 articles were found, out of which 185 were identified as peer-reviewed controlled studies. These articles were evaluated according to the specified inclusion criteria (**Figure 1**) and we identified 26 studies with a total number of 351 subjects that met all inclusion criteria. Almost all studies used a randomized cross-over design, except for studies (Williams et al., 2012; Jones et al., 2016a,b; Schmit et al., 2016; Smits et al., 2016) which used a parallel group design.

Four studies manipulated oxygen availability, 15 manipulated environmental temperature or implemented a pre-cooling intervention in heat, and seven studies manipulated feedback. In all of these studies cycling was the chosen exercise mode. Specifically, investigations manipulating environmental conditions included: *hypoxia* ($n = 3$), *hyperoxia* ($n = 2$), *heat-stress* ($n = 11$) and *pre-cooling interventions* in the heat ($n = 4$). Seven investigations manipulated feedback however a number of different manipulations were undertaken including: *positive deceptive feedback* ($n = 3$), *negative deceptive feedback* (e.g., performance deception, $n = 3$), *neutral feedback* ($n = 2$), *no feedback* ($n = 3$) and *informed feedback* ($n = 2$). One study (Castle et al., 2012) analyzed the effects of heat-stress or deceiving participants of ambient and core temperature by providing positive deceptive feedback, thus data were included in two themes, heat-stress and feedback, respectively. Participants' maximal oxygen uptake ($\dot{V}O_{2max}$) from the 19 studies which reported this parameter was (mean ± SD) 61.3 ± 4.6 $mL^{-1}.kg^{-1}.min$. Three studies only provided peak power output (PPO) data (393 ± 51 W), while four studies did not disclose participants' $\dot{V}$ O_{2max} or PPO. Most studies exclusively investigated male participants, except for one, which reported having one female participant amongst a male cohort (Périard and Racinais, 2015). However, one study did not report the gender of their participants (Schmit et al., 2016). An overview of included studies is provided in **Table 1**.

Finally, when studies were assessed using the PEDro scale, all 26 studies were considered as "high quality," with a PEDro score of (mean ± SD) 8.4 ± 0.6. Due to the nature of heat intervention trials, the blinding of participants is not possible,

TABLE 1 | Overview of the analyzed studies.

Study, year	Study design	No. of participants and training status[a]	Exercise protocol	Intervention	Time point of environmental exposure or intervention manipulation	Performance measurement (total time or mean power output (MPO) in watts)	PEDro Score
OXYGEN AVAILABILITY							
Amann et al., 2006	Crossover	8 Well-trained male cyclists (63.3 ± 1.3 ml $kg^{-1}min^{-1}$)	5 km cycling time-trial	Time-trial in normoxia (FiO_2 = 0.21; CON.), hypoxia (FiO_2 = 0.15; INTER), iso-oxia (FiO_2 = 0.24–0.30; INTER) and hyperoxia (FiO_2 = 1.00; INTER)	Exposure 3 min before trial, after a warm-up in normoxia	FiO_2: 0.21 = 458 ± 7 s FiO_2: 0.15 = 483 ± 8 s FiO_2: 0.24–0.30 = 351 ± 7 s FiO_2: 1.00 = 439 ± 7 s	9
Clark et al., 2007	Crossover	10 Well-trained male cyclists and triathletes (67.7 ± 1.3 ml $kg^{-1}min^{-1}$)	5 min cycling time-trial	Time-trial in normoxia (FiO_2 = 0.21; CON) and hypoxia (FiO_2 = 0.19; 0.16; and 0.14 INTER)	Exposure before, during and after warm-up. Total of 40 min in hypoxia before trial	FiO_2: 0.21 = 367 ± 42 MPO FiO_2: 0.19 = 346 ± 41 MPO FiO_2: 0.16 = 329 ± 38 MPO FiO_2: 0.14 = 294 ± 37 MPO	10
Périard and Racinais, 2016	Crossover	12 Well-trained male cyclists	Cycling time-trial to complete 750 kJ	Time-trial in normoxia (FiO_2 = 0.21; CON) and hypoxia (FiO_2 = 0.15; INTER)	Exposure 5 min before trial, after a warm-up in normoxia	FiO_2: 0.21 = 48.2 ± 5.7 min FiO_2: 0.15 = 60.1 ± 6.5 min	9
Tucker et al., 2007	Crossover	11 Well-trained male cyclists (395 ± 33 PPO)	20 km cycling time-trial	Time-trial in normoxia (FiO_2 = 0.21; CON) and hyperoxia (FiO_2 = 0.40; INTER)	Time point unknown, including oxygen inhaled in warm-up	FiO_2: 0.21 = 28 ± 8 min FiO_2: 0.40 = 27 ± 34 min	9
HEAT-STRESS							
Abbiss et al., 2008	Crossover	10 Well-trained male cyclists (61.7 ± 5.0 ml $kg^{-1}min^{-1}$)	16.1 km cycling time-trial	Time-trial in temperate (18.1°C, 58% RH; CON) and heat (32°C, 50% RH; INTER)	Exposure during warm-up (90 min fixed workload) and at rest. Total of 116.3 min in heat before trial	18.1°C = 25.4 ± 1.6 min 32°C = 27.5 ± 1.9 min	8
Altareki et al., 2009	Crossover	9 Competitive male triathletes and cyclists (61.7 ± 8.6 ml $kg^{-1}min^{-1}$)	4 km cycling time-trial	Time-trial in cool (13°C, 40%; CON) and heat (35°C, 60% RH; INTER)	Exposure before, during and after warm-up. Total of 21 min in heat before trial	13°C = 382.8 ± 18.2 s 35°C = 390.1 ± 19.6 s	8
Castle et al., 2012	Crossover	7 Recreational male cyclists (58.8 ± 5.7 ml $kg^{-1}min^{-1}$)	30 min cycling time-trial	Time-trial in temperate (22°C, 43% RH; CON) and heat (31°C, 64% RH; INTER)	Exposure during a 7 min warm-up in heat before trial	22°C = 179.9 ± 50.9 MPO 31°C = 168.1 ± 54.1 MPO	9
Peiffer and Abbiss, 2011	Crossover	9 Trained male cyclists (60.5 ± 4.5 ml $kg^{-1}min^{-1}$)	40 km cycling time-trial	Time-trial in temperate (22°C, 40% RH; CON), cool (17°C, 40% RH; INTER), warm (27°C 40% RH; INTER), and hot (32°C, 40% RH; INTER)	Time point unknown, including temperature in warm-up	17°C = 58.8 ± 2.0 min 22°C = 59.0 ± 2.3 min 27°C = 59.1 ± 2.3 min 32°C = 60.7 ± 2.9 min	8
Périard et al., 2011	Crossover	8 Well-trained male cyclists (66.4 ± 5.3 ml $kg^{-1}min^{-1}$)	40 km cycling time-trial	Time-trial in temperate (20°C, 40%: CON) and heat (35°C, 60% RH; INTER)	Exposure 5 min before trial, after a warm-up in temperate	20°C = 59.8 ± 2.6 min 35°C = 64.3 ± 2.8 min	8
Périard and Racinais, 2015	Crossover	11 Well-trained cyclists, 10 males and 1 female (60.2 ± 6.3 ml $kg^{-1}min^{-1}$)	Cycling time-trial to 750 kJ complete	Time-trial in temperate (20°C, 40%; CON) and heat (35°C, 60% RH; INTER)	Exposure 5 min before trial, after a warm-up in temperate	20°C = 48.8 ± 12.7 min 35°C = 55.8 ± 14.4 min	8

(Continued)

TABLE 1 | Continued

Study, year	Study design	No. of participants and training status[a]	Exercise protocol	Intervention	Time point of environmental exposure or intervention manipulation	Performance measurement (total time or mean power output (MPO) in watts)	PEDro Score
Périard and Racinais, 2016	Crossover	12 Well-trained male cyclists	Cycling time-trial to 750 kJ complete	Time-trial in temperate (18°C, 40%; CON) and heat (35°C, 60% RH; INTER)	Exposure 5 min before trial, after a warm-up in temperate	18°C = 48.2 ± 5.7 min 35°C = 55.4 ± 5.0 min	8
Schmit et al., 2016	Parallel group	34 Well-trained triathletes, gender unknown. Temperate (n = 22; 63.3 ± 2.1 ml $kg^{-1}min^{-1}$), Heat (n = 12; 62.2 ± 3.6)	20 km cycling time-trial	Time-trial in temperate (21°C, 50%; CON) acute heat exposure (35°C, 50% RH at 0; INTER and 11 ± 4 days; INTER)	Exposure 15 min before trial during a warm-up, the second heat trail took place 11 ± 4 days after trial 1 with no acclimatization	*0 days* 21°C = 32.2 ± 2.0 min 35°C = 33.2 ± 1.58 min *11 ± 4 days after first trial* 21°C = 31.5 ± 1.4 min 35°C = 32.4 ± 1.2 min	7
Tatterson et al., 2000	Crossover	11 National male road cyclists (66.7 ± 13.6)	30 min cycling time-trial	Time-trial in temperate (23°C, 60% RH; CON) and heat (32°C, 60% RH; INTER)	Time point unknown, including temperature in warm-up	23°C = 345 ± 9 MPO 32°C = 323 ± 8 MPO	8
Tucker et al., 2004	Crossover	10 Recreational male cyclists (376 ± 47 PPO)	20 km cycling time-trial	Time-trial in cool (15°C, 60% RH; CON) and heat (35°C, 60% RH; INTER)	Time point unknown, including temperature in warm-up	15°C = 28.8 ± 1.8 min 35°C = 29.6 ± 1.9 min	8
VanHaitsma et al., 2016	Crossover	20 Trained male cyclists (54.8 ± 5.9)	40 km cycling time-trial	Time-trial in temperate (21°C, 20% RH; CON) and heat (35°C, 25% RH; INTER)	Time point unknown, including temperature in warm-up	21°C = 75.2 ± 6.6 min 35°C = 79.0 ± 7.2 min	8
PRE-COOLING STRATEGIES							
Barwood et al., 2012	Crossover	11 Trained male cyclists	40 km cycling time-trial	Time-trial in heat (32°C, 53% RH) with use of no spray (CON), menthol spray (INTER) and placebo cooling spray (INTER)	10 min warm-up in heat, existed chamber (5 min rest in ∼22°C), re-entered and sprayed with solution. Total of ∼23 min in heat	CON = 71.58 ± 62 min Placebo = 70.94 ± 6.1 min Menthol = 71.04 ± 5.5 min	9
Byrne et al., 2011	Crossover	7 Recreational male cyclists	30 min cycling time-trial	Time-trial in the heat (32°C, 60% RH) ingesting 900mL of hot (37°C; CON) or cold fluid (2°C; INTER) 10 min before trial	After fluid ingestion, 5 min at rest in heat, temperature of warm-up unknown	37°C fluid = 261 ± 22 MPO 2°C fluid = 275 ± 27 MPO	8
Duffield et al., 2010	Crossover	8 Moderate to well-trained male cyclists (lactate threshold 221 ± 42 W)	40 min cycling time-trial	Time-trial in the heat (33°C, 50% RH) with (INTER) and without (CON) lower body cold water immersion (CWI) at 14°C before trial	20 min of CWI, 5 min warm-up in heat. Time between CWI and warm up ∼8–10 min, ≤ 5 min from warm-up and trial start	CON = 178 ± 26 MPO 14°C CWI = 198 ± 25 MPO	8
Gonzales et al., 2014	Crossover	10 trained male cyclists (59.1 ± 7.0 ml $kg^{-1}min^{-1}$)	20 min cycling time-trial	Time-trial in the heat (30°C, 79% RH) with (INTER) and without (CON) a cooling vest worn and refrigerated headband	18 min warm-up with cooling vest and headband in heat. Rested in heat for 10 min (no vest) before trial. Total of 28 min in heat	CON = 222 ± 47 MPO Cooling vest = 239 ± 45 MPO	8
FEEDBACK MANIPULATIONS							
Albertus et al., 2005	Crossover	15 Competitive male cyclists (397 ± 58 PPO)	20 km cycling time-trial	Time-trial with correct fb (CON) and positive deception (Pos-fb; INTER)	Informed traveled 25-m further than they had every km	CON = 28.4 ± 1.6 min Pos-fb = 28.6 ± 1.5 min	8

(Continued)

TABLE 1 | Continued

Study, year	Study design	No. of participants and training status[a]	Exercise protocol	Intervention	Time point of environmental exposure or intervention manipulation	Performance measurement (total time or mean power output (MPO) in watts)	PEDro Score
Castle et al., 2012	Crossover	7 Recreational male cyclists (58.8 ± 5.7 ml $kg^{-1}min^{-1}$)	30 min cycling time-trial	Time-trial in the heat without deception (31°C, 64% RH; CON) and with deception (told the temperature was 26°C, 60% RH, reality: 32°C, 65% RH; Pos-fb; INTER)	From the onset of the trial and displayed incorrectly during trial on a computer screen	CON = 179.9 ± 50.9 MPO Pos-fb = 184.4 ± 60.4 MPO	9
Jones et al., 2016b	Parallel group	20 Trained male cyclists, CON (n = 10; 57.6 ± 6.7 ml $kg^{-1}min^{-1}$), Deception (n = 10; 58.7 ± 6.6 ml $kg^{-1}min^{-1}$)	16.1 km cycling time-trial	Time-trial with accurate ride-alone fb (CON), against a pacer representing MPO of CON (Neutr-fb; INTER), unaware of MPO of pacer 102% above baseline (Neg-fb; INTER) and a subsequent ride-alone trial (not informed of deception in previous trial and not included in meta-analysis)	From the onset of the trial	*Control Group:* CON = 27:10 ± 2:08 min Neutr-fb = 26:47 ± 1:55 min Inform-fb = 26:55 ± 1:58 min *Deception Group:* CON = 27:00 ± 1:31 min Pos-fb = 26:41 ± 1:13 min Inform-fb = 26:56 ± 1:38 min	9
Jones et al., 2016a	Parallel group	17 Trained male cyclists, CON (n = 9; 54.1 ± 5.9 ml $kg^{-1}min^{-1}$), Deception (n = 8; 53.3 ± 4.4 ml $kg^{-1}min^{-1}$)	16.1 km cycling time-trial	Time-trial with accurate ride-alone fb (CON), against a pacer representing MPO of CON (Neutr-fb; INTER), unaware or aware that MPO of pacer was increased 102% above (Neg-fb; INTER) and a subsequent ride-alone trial after being informed of the nature of a previous trial (Inform-fb; INTER)	From the onset of the trial	*Control Group:* CON = 26:31 ± 1:44 min Neutr-fb = 26:15 ± 1:31 min Inform-fb = 26:40 ± 1:30 min *Deception Group:* CON = 26:40 ± 0:52 min Pos-fb = 26:22 ± 0:44 min Inform-fb = 26:34 ± 0:54 min	9
Shei et al., 2016	Crossover	14 Competitive male cyclists (61.6 ± 0.6 ml $kg^{-1}min^{-1}$)	4 km cycling time-trial	Time-trial with correct fb ride-alone (CON), unaware of MPO of pacer 102% above baseline (Neg-fb; INTER) and a subsequent trial after deception was revealed with known pacer at 102% (Inform-fb; INTER)	From the onset of the trial	CON = 366.4 ± 3.6 MPO Neg-fb = 358.6 ± 2.7 MPO Inform-fb = 358.1 ± 2.8 MPO	8
Smits et al., 2016	Parallel group	20 Trained male cyclists and triathletes, CON (n = 10;53.7 ± 7.1 ml $kg^{-1}min^{-1}$) and No-fb (n = 10; 59.0 ± 7.7 ml $kg^{-1}min^{-1}$)	20 km cycling time-trial	Time-trial with distance only fb (CON) and no fb (No-fb; INTER)	From the onset of the trial, cyclists stopped at completed distance	CON = 28.7 ± 3.7 min No-fb = 31.0 ± 2.8 min	8
Swart et al., 2009	Crossover	12 Competitive cyclists (56.6 ± 6.6 ml $kg^{-1}min^{-1}$)	40 km cycling time-trial	Time-trial with distance only fb (CON) and no fb (No-fb; INTER)	From the onset of the trial, at final km, cyclists were then informed they had 1-km to complete	CON = 265.5 ± 36.4 MPO No-fb = 256.6 ± 36.6 MPO	8

(Continued)

TABLE 1 | Continued

Study, year	Study design	No. of participants and training status[a]	Exercise protocol	Intervention	Time point of environmental exposure or intervention manipulation	Performance measurement (total time or mean power output (MPO) in watts)	PEDro Score
Waldron et al., 2015	Crossover	9 Well-trained male cyclists (60.5 ± 3.3 ml $kg^{-1}min^{-1}$)	4 km cycling time-trial	Time-trial with correct ascending fb (CON) and increased ascending fb by 102% compared to CON (Pos-fb; INTER)	An ascending distance clock was continuously displayed and visible to participants	CON = 354 ± 39 s Pos-fb = 372 ± 36 s	9
Williams et al., 2012	Parallel group	22 Non-competitive and untrained males in cycling, CON (n = 11) and No-fb (n = 11) groups (50 ± 9 ml $kg^{-1}min^{-1}$)	4 km cycling time-trial	Time-trial with distance only fb (CON) and no fb (No-fb; INTER)	From the onset of the trial, cyclists stopped at completed distance	*Group 1* CON = 198 ± 39 MPO No-fb = 179 ± 47 MPO *Group 2* CON = 181 ± 37 MPO No-fb = 171 ± 53 MPO	9

a Data for VO_{2max} in mL/kg/min and peak power output in watts are presented in mean ± standard deviations. PPO, peak power output; FiO_2, fraction of inspired oxygen; RH, relative humidity; CON, control trial; INTER, intervention trial; fb, feedback; Pos-fb, positive deceptive feedback; Neg-fb, negative deceptive feedback; Neutr-fb, neutral feedback; No-fb, no; Inform-fb, informed of the nature of a previous trial.

however in other conditions it is possible to blind participants to the intervention condition and in eleven studies this was the case. In these studies participants were blinded to a manipulation in: oxygen availability ($n = 4$) (Amann et al., 2006; Clark et al., 2007; Tucker et al., 2007; Périard and Racinais, 2016), pre-cooling ($n = 1$) (Barwood et al., 2012) and feedback ($n = 6$) (Albertus et al., 2005; Castle et al., 2012; Waldron et al., 2015; Jones et al., 2016a,b; Shei et al., 2016). Only one study implemented a double-blind design (Clark et al., 2007). **Figure 2** illustrates the number of research trials that fulfilled each criteria of the PEDro scale.

Analysis of Studies Which Investigated the Manipulation of Hypoxia

A total of five MPO data points were extracted from three studies for the start, middle and end sections of trials and overall MPO (Amann et al., 2006; Clark et al., 2007; Périard and Racinais, 2016). Mean power output was significantly reduced for all intervention trials (Mean Difference (MD) = −49.33 watts (W), 95% confidence interval (95% CI) = −76.89 to −21.81, $p = 0.000$) compared to the control (normoxia) trial (**Figure 3**). There was a significant reduction in MPO for the middle (MD = −48.36 W, 95% CI = −86.86 to −9.95, $p = 0.014$) and end sections (MD = −54.48 W, 95% CI = −103.24 to −5.71, $p = 0.029$) of the intervention trials. Interestingly, one study reported no meaningful changes across all trial sections for low (0.19 F_iO_2), moderate (0.16 F_iO_2) and high (0.14 F_iO_2) stimulated altitude compared to sea level (Clark et al., 2007). In addition, a large variation in section MPO, indicated by the range of the 95% CIs, was observed across all three sections of the intervention trials compared to the control (normoxia) trial in this study (**Figure 3**). One study (Amann et al., 2006) demonstrated a marked reduction in the pacing index change score (**Figure 12A**) across the trial for the hypoxic trial IP (FiO_2 0.15) relative to the control (normoxic) trial condition.

Analysis of Studies Which Investigated the Manipulation of Hyperoxia

Two studies provided a total of three MPO data points for the start, middle and end sections and for overall time-trials (Amann et al., 2006; Tucker et al., 2007). There was no significant change in overall MPO for trials completed in hyperoxia (MD = 23.72 W, 95% CI = −24.64 to 72.08, $p = 0.336$). Furthermore, power output remained relatively stable through the start (MD = 13.05 W, 95% CI = −116.79 to 142.89, $p = 0.844$), middle (MD = 23.85 W, 95% CI = −51.92 to 99.61, $p = 0.537$) and end sections (MD = 26.87 W, 95% CI = −44.91 to 98.65, $p = 0.463$) of the hyperoxia trials respectively (**Figure 4**).

Analysis of Studies Which Investigated the Manipulation of Heat-Stress

Heat-stress (**Figure 5**) had a significant, negative impact on time-trial performance from a sample of 14 MPO data points per trial section MPO and for the whole trial MPO across

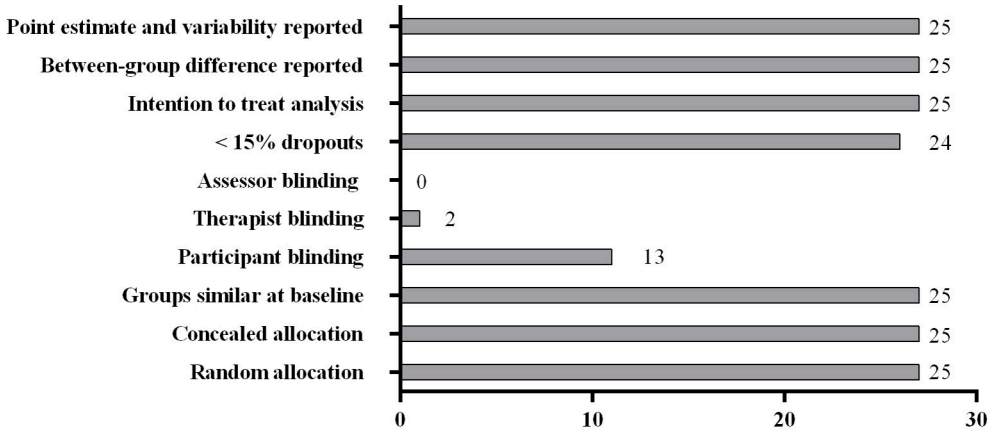

FIGURE 2 | Number of studies meeting individual PEDro [Physiotherapy Evidence Database] criteria.

11 included studies (MD = −24.79 W, 95% CI = −39.52 to −10.06, $p = 0.001$) (Tatterson et al., 2000; Tucker et al., 2004; Abbiss et al., 2008; Altareki et al., 2009; Peiffer and Abbiss, 2011; Périard et al., 2011; Castle et al., 2012; Périard and Racinais, 2015, 2016; Schmit et al., 2016; VanHaitsma et al., 2016). Trial section data demonstrated a gradual decline in MPO (and Pacing Index Change Score) as the trials progressed under hot conditions compared to the control condition (**Figure 12**). Mean power output was significantly reduced in the middle (MD = −27.54 W, 95% CI = −50.36 to −4.72, $p = 0.018$) and end sections (MD = −38.43 W, 95% CI = −67.89 to −8.97, $p = 0.011$) but not the start section (MD = −11.12 W, 95% CI = −36.64 to 14.40, $p = 0.393$).

Analysis of Studies Which Investigated the Manipulation of Pre-Cooling Strategies

Five data points per trial section MPO and for the whole trial MPO were extracted from four studies (Duffield et al., 2010; Byrne et al., 2011; Barwood et al., 2012; Gonzales et al., 2014), to analyse the effect of pre-cooling interventions in hot conditions on time-trial performance. Overall, no significant difference in trial MPO was detected in pre-cooling trials compared to control (no cooling intervention) (MD = 3.90 W, 95% CI = −34.41 to 42.22, $p = 0.842$). The meta-analysis detected no significant changes in MPO for the start (MD = 0.41 W, 95% CI = −63.64 to 64.47, $p = 0.990$), middle (MD = 2.87 W, 95% CI = −67.27 to 73.00, $p = 0.934$) and end (MD = 8.43 W, 95% CI = −56.92 to 73.77, $p = 0.801$) sections in the four studies (**Figure 6**).

Analysis of Studies Which Investigated the Manipulation of Positive Deceptive Feedback

Overall and trial section MPO was extracted from three studies that provided three data points per section (Albertus et al., 2005; Castle et al., 2012; Waldron et al., 2015). There was no significant change in trial MPO when cyclists were provided with positive deceptive feedback (MD = 2.45 W, 95% CI = −35.31 to 40.20, $p = 0.899$). Furthermore, this trend was apparent across the start (MD = −0.85 W, 95% CI = −83.20 to −81.51, $p = 0.984$), middle (MD = 4.11 W, 95% CI = −44.36 to 52.58, $p = 0.868$) and end (MD = 0.71 W, 95% CI = −87.52 to 88.93, $p = 0.987$) sections of trials. Notably, wider 95% CIs were found when cyclists received positive deceptive feedback compared to the other feedback groups, indicating a greater variability in overall performance and section MPO with this form of feedback (**Figure 7**). This was particularly evident, when cyclists were informed that their core and environmental temperature were lower (0.3° and 26°C, respectively) compared to control (Castle et al., 2012).

Analysis of Studies Which Investigated the Manipulation of Negative Deceptive Feedback

A total of four data points per trial section and overall trial MPO, were extracted from three studies that provided participants with negative deceptive feedback (Jones et al., 2016a,b; Shei et al., 2016). Generally, when participants were provided negative deceptive feedback there were small but significant improvements in power output (MD = 8.67 W, 95% CI = 3.13 to 14.21, $p = 0.002$) compared to receiving full and accurate feedback. In these studies, participants were racing against an avatar pacer they believed was programmed to mimic their previous trial MPO performance, however, in reality the pacer was programmed at a higher MPO (102%). Trial section data revealed a tendency for greater power outputs to be attained in the start section (MD = 6.59 W, 95% CI = −0.46 to 13.64, $p = 0.067$). A significant improvement in trial MPO was found in the middle section of trials (MD = 11.16 W, 95% CI = −0.46 to 13.64, $p = 0.001$). However, no significant changes were found in the end section of trials (MD = 0.25 W, 95% CI = −5.32 to 5.83, $p = 0.929$). Additionally, the width of the 95% CI were notably smaller compared to positive deceptive feedback, indicating there was less variability

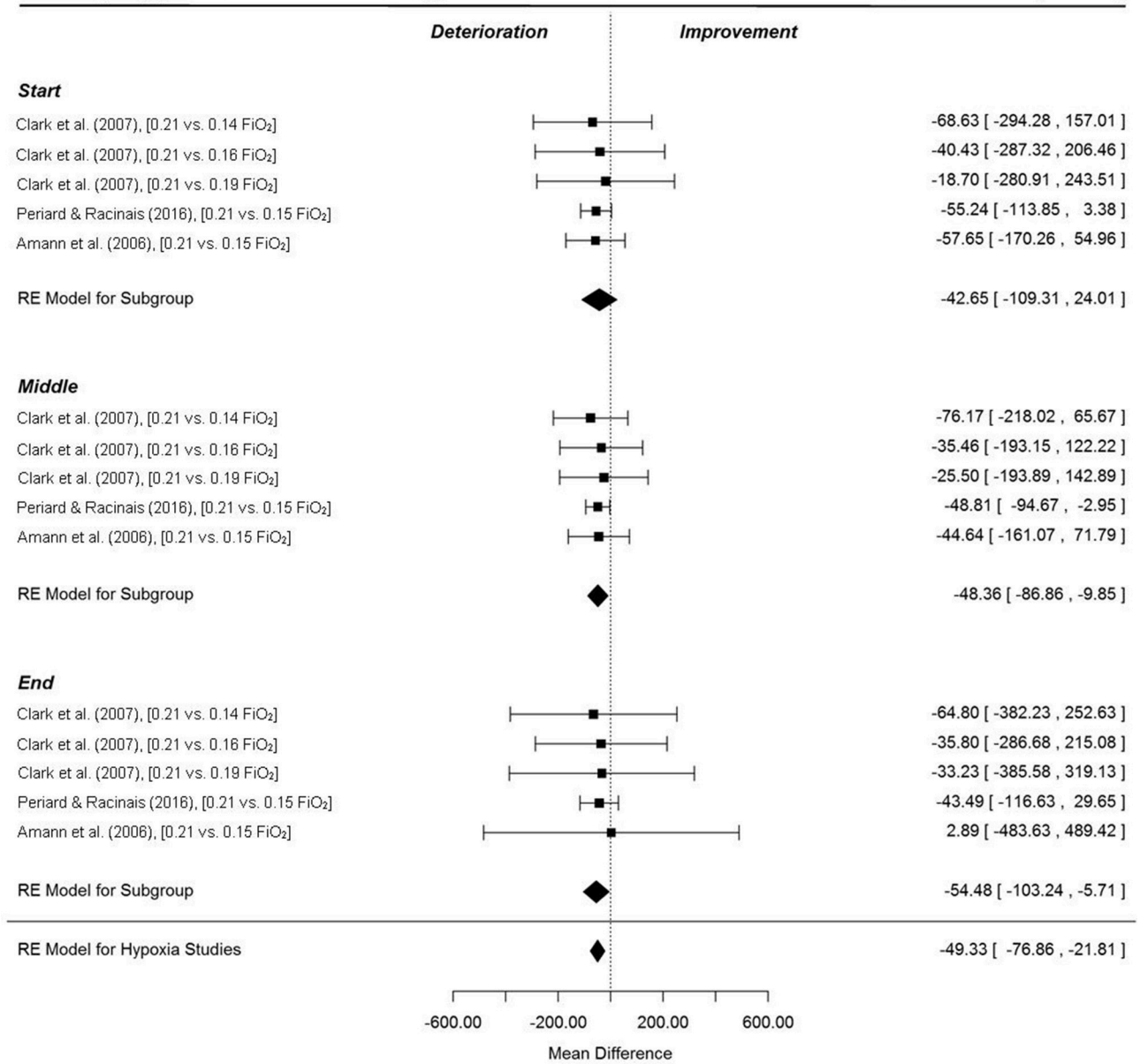

FIGURE 3 | Forest plot for hypoxia meta-analysis illustrating power output during start, middle and end sections compared to normoxia trials. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within the meta-analysis. The diamond represents the overall mean difference for each split with the width of the diamond signifying the 95% CIs.

in overall performance and individual section MPO when participants were provided with negative deceptive feedback (**Figure 8**).

Analysis of Studies Which Investigated the Manipulation of Neutral Feedback

Two data points per trial section MPO and for the whole trial MPO were extracted from two studies (Jones et al., 2016b; Waldron et al., 2015). **Figure 9** shows that neutral feedback, where feedback was accurately given or where participants raced a virtual on-screen avatar that accurately represented the MPO of a previous performance, did not statistically influence overall MPO (MD = 4.32 W, 95% CI = −5.21 to 13.86, $p = 0.374$). There were no significant improvements in MPO across the start (MD = 11.73 W, 95% CI = −11.71 to 35.16, $p = 0.327$), middle (MD = 6.37 W, 95% CI = −8.98 to 21.77, $p = 0.414$)

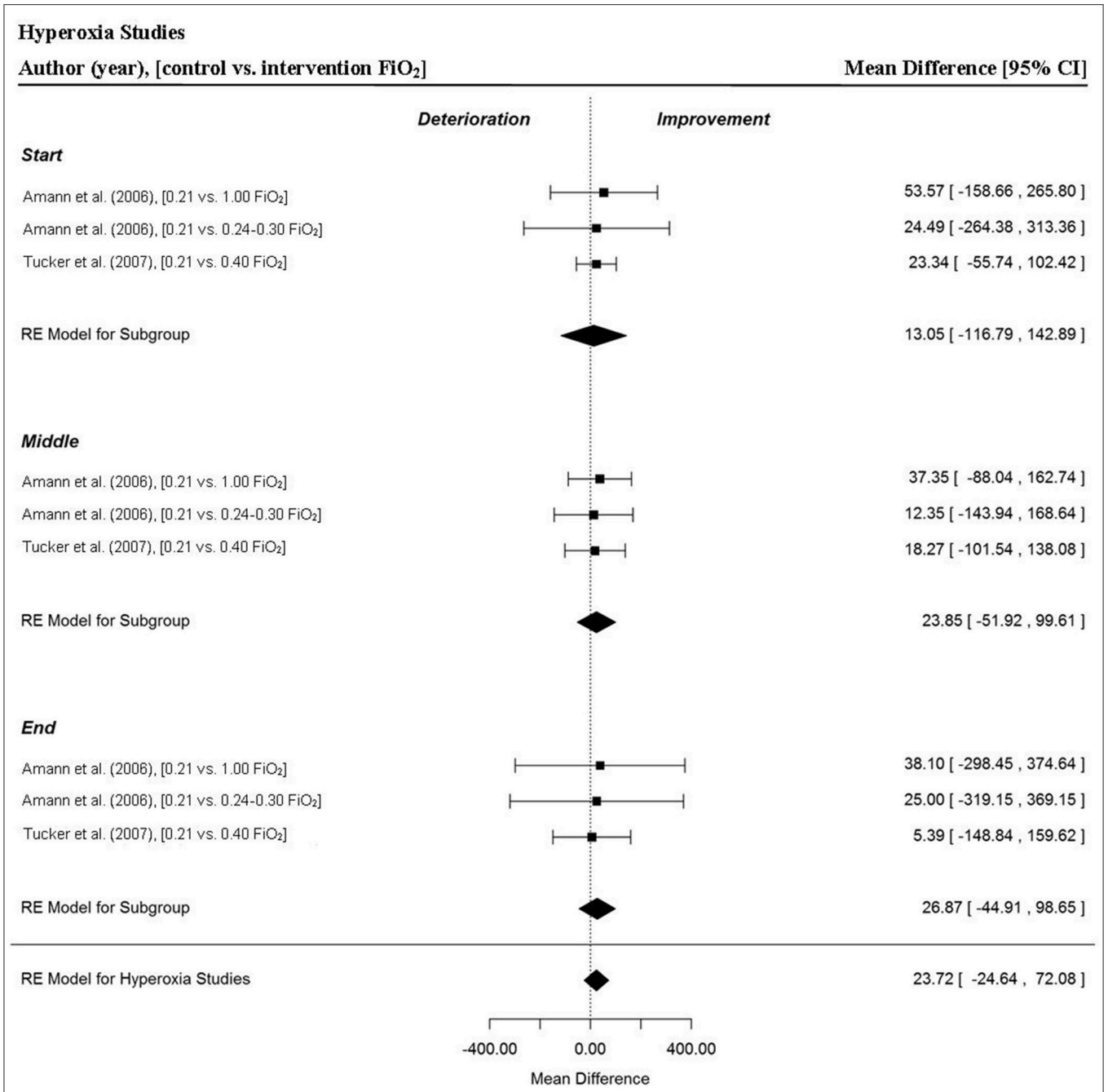

FIGURE 4 | Forest plot for hyperoxia meta-analysis illustrating power output during start, middle and end sections compared to normoxia trials. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within the meta-analysis. The diamond represents the overall mean difference for each split with the width of the diamond signifying the 95% CIs.

or end (MD = −0.17 W, 95% CI = −14.39 to 14.05, $p = 0.981$) sections.

Analysis of Studies Which Investigated the Manipulation of No Feedback

Data points were extracted from three different comparisons and three separate investigations (Swart et al., 2009; Williams et al., 2012; Smits et al., 2016). In these studies, where all visual and verbal performance feedback was withheld, no significant changes in power output were found (MD = 11.34 W, 95% CI = −12.67 to 35.34, $p = 0.355$). There were no significant changes in MPO in the start (MD = 7.23 W, 95% CI = −43.03 to 57.53, $p = 0.778$), middle (MD = 8.26 W, 95% CI = −25.31 to 41.84, $p = 0.630$) and end (MD =

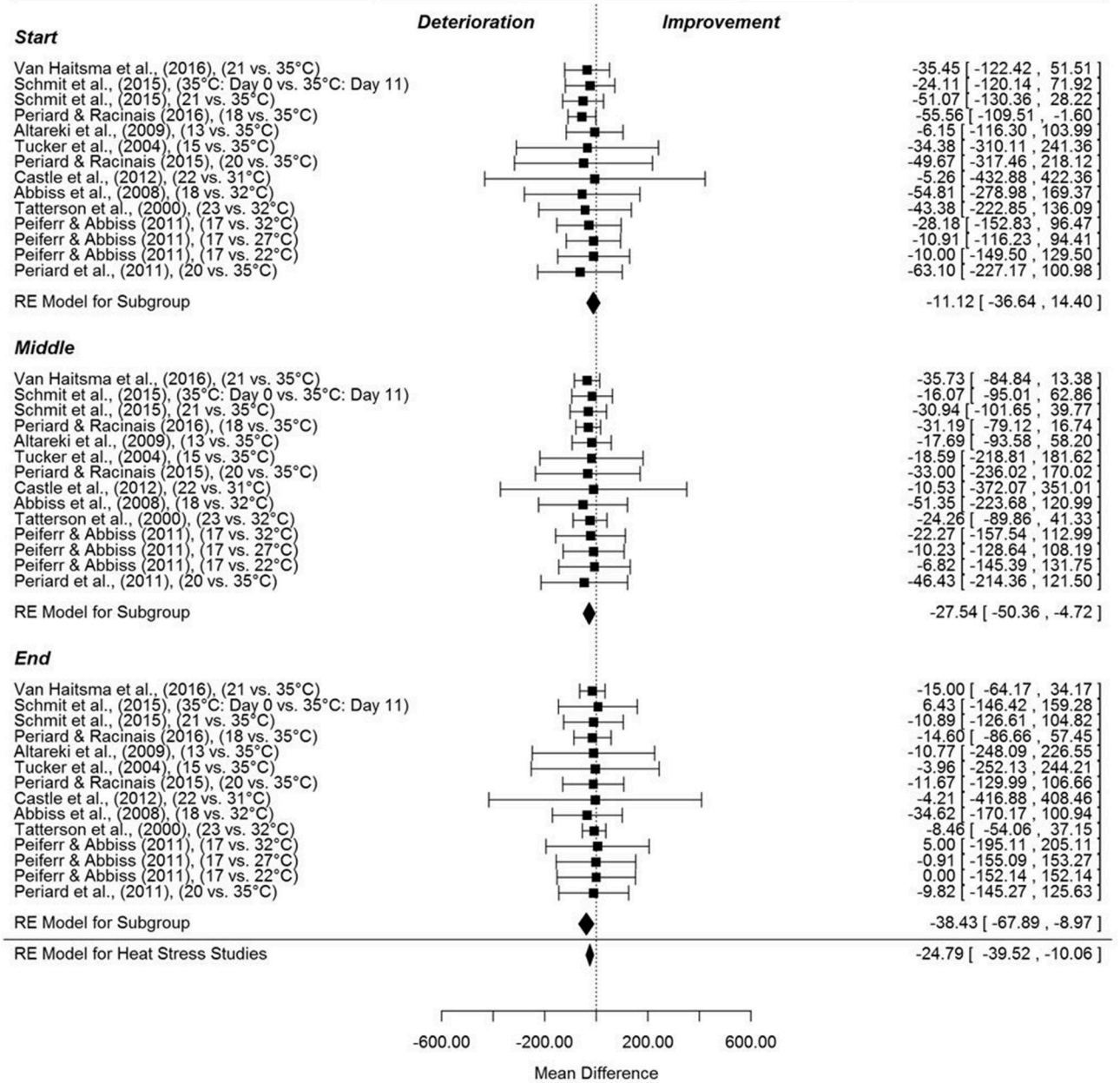

FIGURE 5 | Forest plot for heat-stress meta-analysis illustrating power output during start, middle and end sections compared to temperate trials. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within the meta-analysis. The diamond represents the overall mean difference for each split with the width of the diamond signifying the 95% CIs.

20.94 W, 95% CI = −26.05 to 67.93, $p = 0.382$) sections of trials.

Analysis of Studies Which Investigated the Manipulation of Informed Feedback

Three data points per trial section MPO and for the whole trial MPO were extracted from two studies (Jones et al., 2016a; Shei et al., 2016). Overall, a significant change in trial MPO was found when participants completed an informed trial following a negative feedback trial (MD = 10.55 W, 95% CI = 0.97 to 20.12, $p = 0.031$). In these studies, participants were either unaware (Jones et al., 2016a; Shei et al., 2016) or aware (Jones et al., 2016a) of the increased MPO of their previous trial (102%), represented by a virtual on-screen avatar. Participants completed

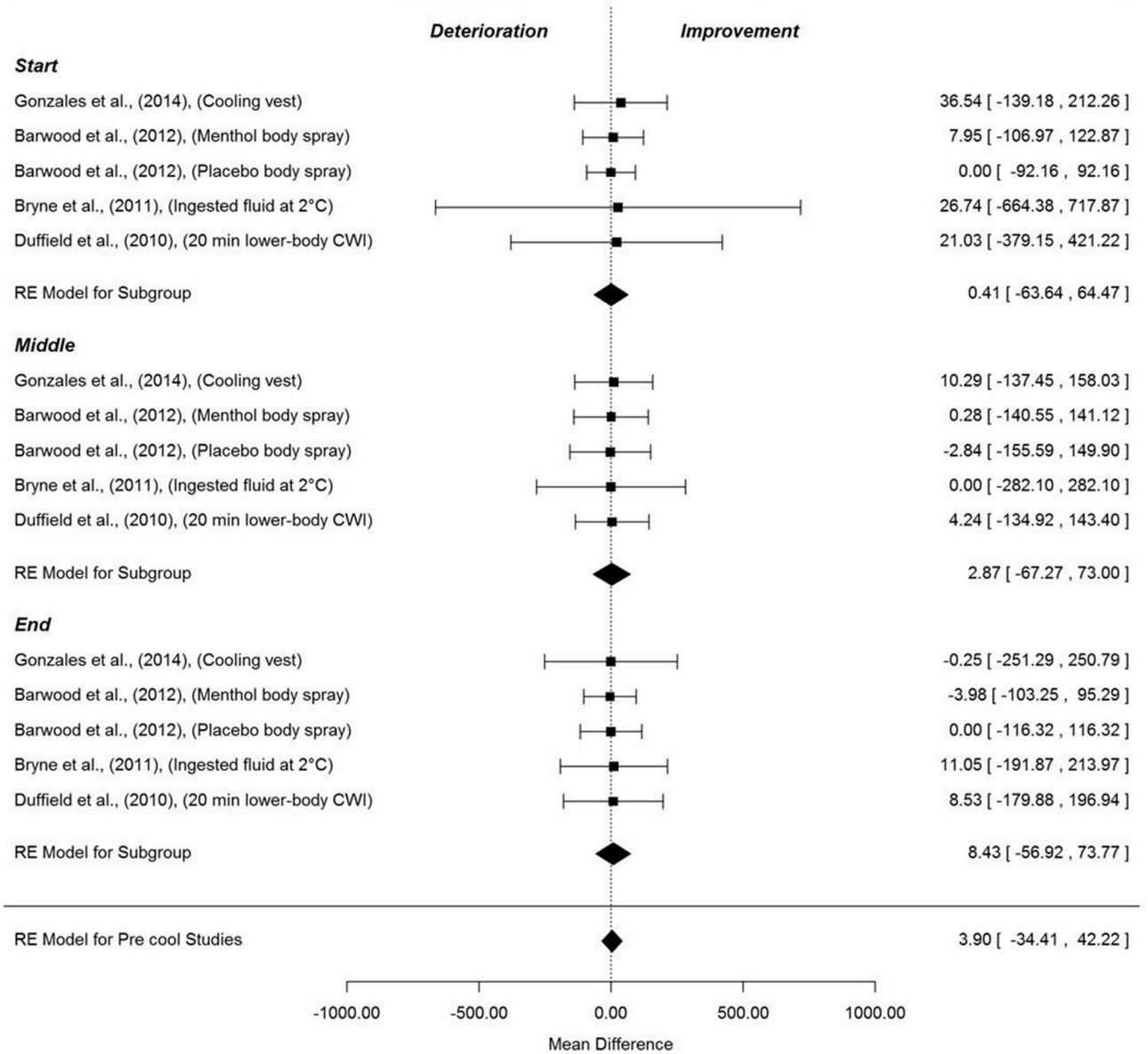

FIGURE 6 | Forest plot for pre-cooling intervention meta-analysis illustrating power output during start, middle and end sections power output for start, middle and end sections compared to no intervention. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within the meta-analysis. The diamond represents the overall mean difference for each split with the width of the diamond signifying the 95% CIs.

a subsequent (informed) trial with the presence of a pacer and known MPO increase (102%) (Shei et al., 2016) or with no pacer (Jones et al., 2016a). Trial section data revealed a tendency for a small increase in power outputs in the end section of the trial (MD = 7.00 W, 95% CI = −1.01 to 15.00, $p = 0.087$). However, MPO across the start (MD = −2.01 W, 95% CI = −15.01 to 10.99, $p = 0.762$), and middle (MD = 9.01 W, 95% CI = −8.09 to 26.12, $p = 0.302$) sections of the trial were non-significant. Unexplained variance was low at the start (19%) and end (0%) of the trial, however substantially greater heterogeneity of variance (69%) was evident in the middle section of the trial.

Analysis for Heterogeneity

Unexplained variance, indicated by the I^2 statistic, was low (0%) across all environmental and almost all feedback groups, indicating low heterogeneity for the start, middle and end

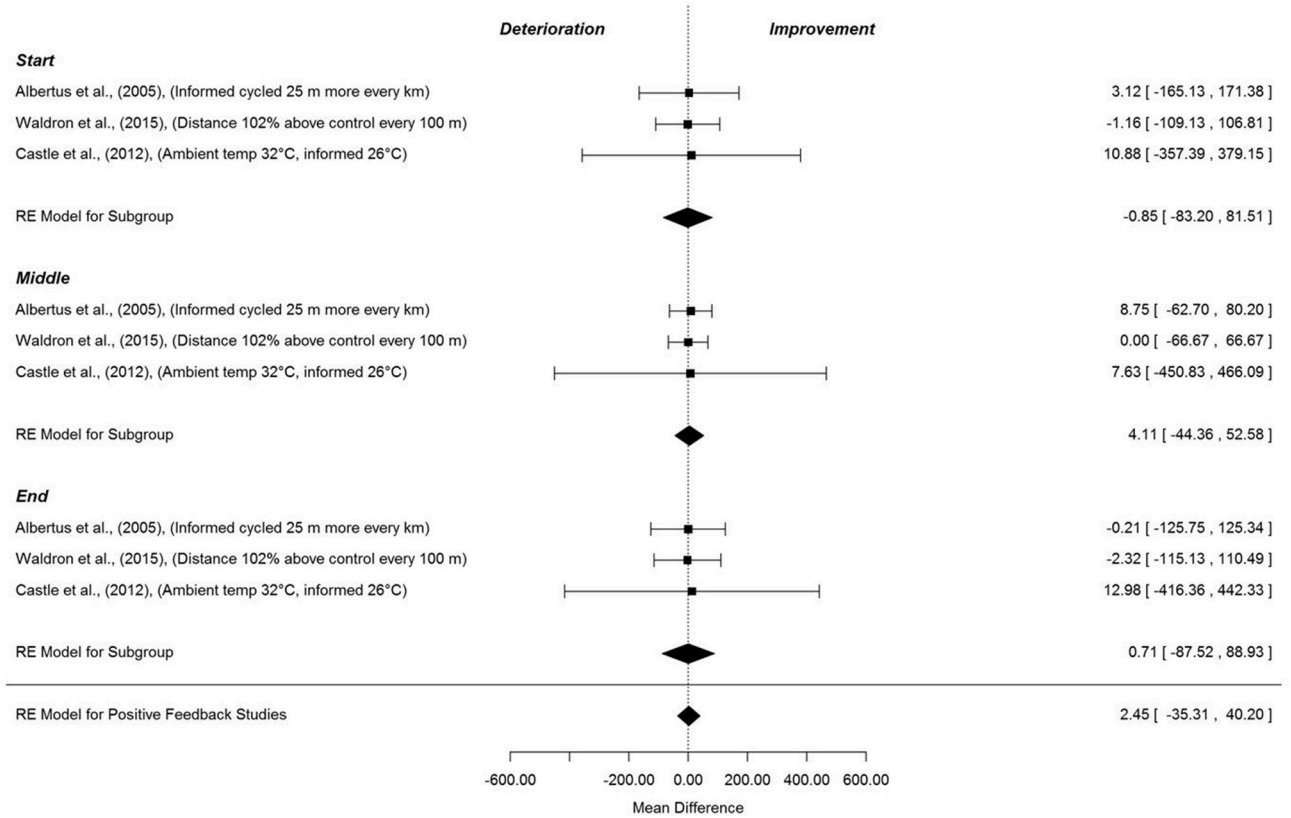

FIGURE 7 | Forest plot for positive deceptive feedback meta-analysis illustrating power output during start, middle and end sections power output for start, middle and end sections compared to full feedback. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within the meta-analysis. The diamond represents the overall mean difference for each split with the width of the diamond signifying the 95% CIs.

sections respectively. An exception was the informed feedback group where a large percentage of unexplained variance was found for the middle section of trials. However, the meta-analysis included a small sample in the defined themes, with the exception of the heat-stress group, therefore the Q-test is likely underpowered for detecting true heterogeneity. Individual group results are outlined in **Table 1**.

DISCUSSION

Previously a number of reviews have been published concerning deception (Jones et al., 2013; Williams et al., 2014), decision making (Edwards and Polman, 2013; Renfree et al., 2014; Smits et al., 2014; McCormick et al., 2015) and neurophysiological determinants (Roelands et al., 2013) of pacing. However, to our knowledge this is the first meta-analysis to have quantified how the different environmental conditions and various forms of performance feedback used in studies to date have affected the pacing strategy exhibited during self-paced time-trials. By segmenting performance into three sections; the start, middle and end, this analysis provides further insight into how participants regulate their exercise under differing experimental conditions.

Pacing and Oxygen Availability

The meta-analysis demonstrated in hypoxia trials participants' significantly reduced their MPO in comparison with their normoxia time-trials. However, the MPO for the start section of the hypoxia trials was not significantly different to the respective normoxia trials, which indicates there was a delay in the adjustment of the pacing strategy in hypoxia because it was only as the hypoxia trials progressed, that a significant reduction in MPO (in the middle and final sections) was observed in comparison with the normoxia trials. Notably in the hypoxia studies, participants began to inhale their allocated oxygen content for a period of time prior to the trials beginning (M $\pm$ SD: 16 $\pm$ 21 min) but were deceived concerning the nature of the inspired oxygen content. Therefore, despite inspiring gas mixtures with a reduced FiO_2, well before the beginning of the hypoxia trials, participants began their hypoxic trials

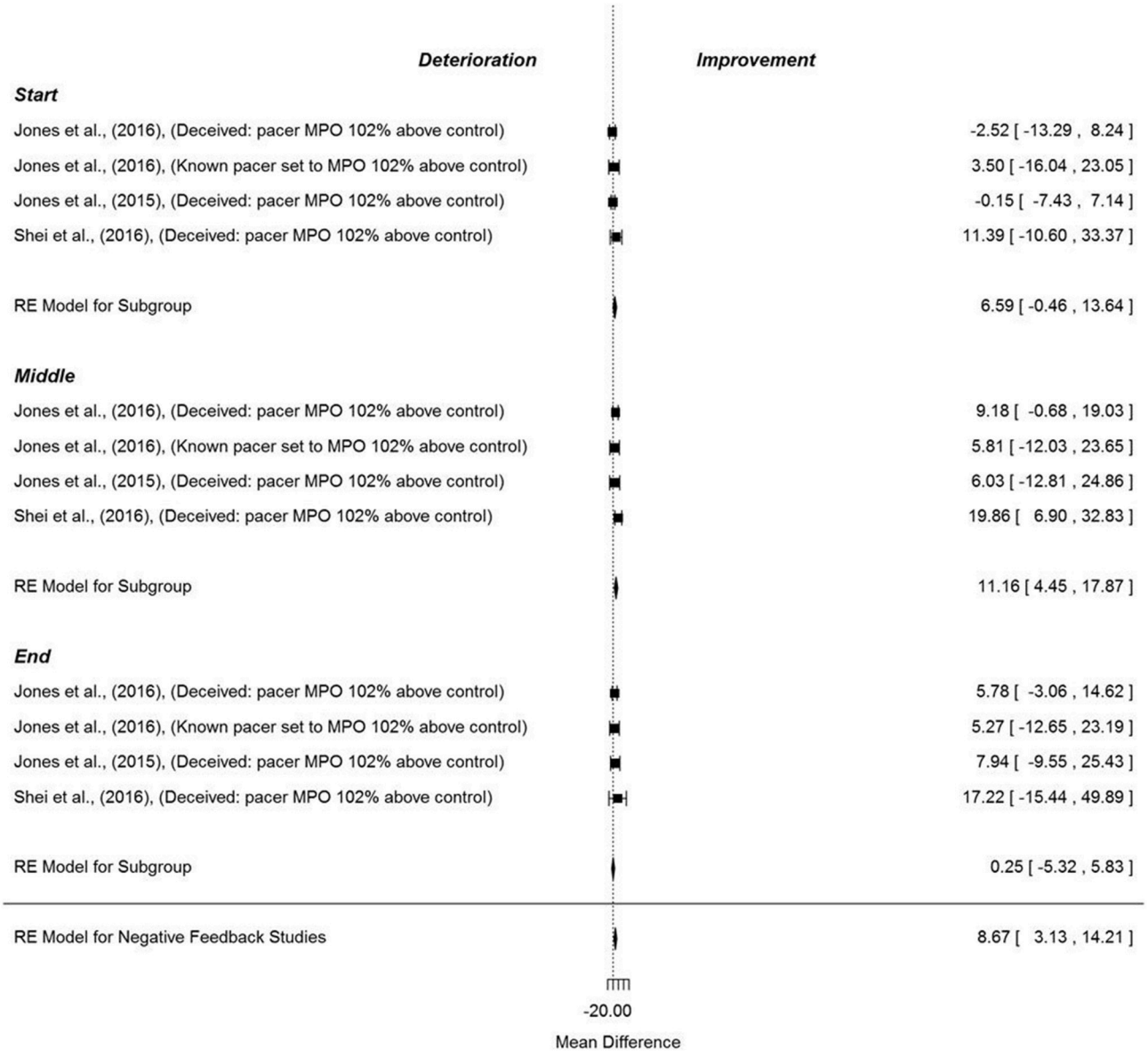

FIGURE 8 | Forest plot for negative deceptive feedback meta-analysis illustrating power output during start, middle and end sections compared to full feedback. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within the meta-analysis. The diamond represents the overall mean difference for each split with the width of the diamond signifying the 95% CIs.

with a power output close to their "normal" normoxic trial pacing strategy. This similarity in starting MPO, irrespective of inspired FiO_2, has been observed previously. It appears that when participants are blinded to the inspired oxygen content, there is a time lag between when the exercise begins and when the pacing strategy is changed compared to the normoxic condition. This suggests afferent feedback, from peripheral chemoreceptors in the aortic and carotid bodies sensing the F_iO_2 disturbance, takes a significant amount of time to be assimilated and acted upon. A time lag spanning between 30 and 60 s from the start of the exercise bout before a pacing adjustment is made has previously been reported (Peltonen et al., 2001a; Amann et al., 2006; Johnson et al., 2009; Henslin et al., 2013). Furthermore, it has been demonstrated that Phase I of the $\dot{V}O_2$ fast component is similar in hypoxic, normoxic, and hyperoxic conditions at the onset of exercise (Peltonen et al., 2001a), which may indicate

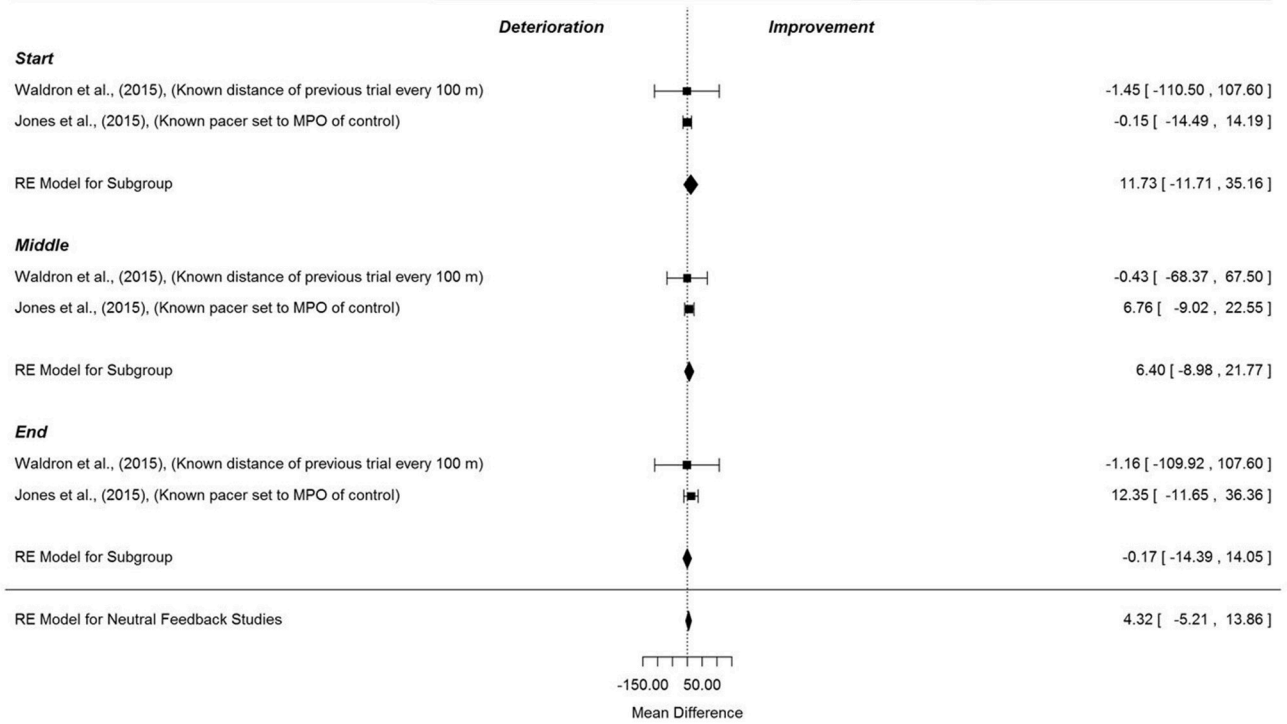

FIGURE 9 | Forest plot for neutral feedback meta-analysis illustrating power output during start, middle and end sections compared to full feedback. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within the meta-analysis. The diamond represents the overall mean difference for each split with the width of the diamond signifying the 95% CIs.

there is an aspect of physiological pre-conditioning present at the start of a known exercise challenge, which occurs irrespective of current gaseous exchange conditions. However, once the exercise begins and afferent feedback is integrated then a decision is made which leads to a reduced central motor drive after a period of 30 s or more of the hypoxic trial. We must however acknowledge that this argument is speculative and might only hold for exercise challenges where there is a requirement for a high exercise intensity to be undertaken in reduced FiO_2 conditions equivalent to low-moderate altitude, as was the case in this meta-analysis.

The reduction in MPO for the middle and end trial sections in hypoxic trials suggest participants adjusted their power output, as chemoreceptors detected the reduction in oxygen availability and peripheral capillary oxygen saturation, compared to the normoxic condition (Johnson et al., 2009). A further explanation for the reduction in MPO in the hypoxia trials is that due to a reduction in oxygen delivery the aerobic energy production was compromised, relative to the normoxic condition, and remained suppressed throughout the hypoxic trials (Peltonen et al., 2001a). This would potentially lead to a relatively greater anaerobic energy contribution in the hypoxic trials compared to the normoxic trial during middle and end sections of the trials. Subsequently, a greater proportion of the finite anaerobic capacity would be expended earlier in the hypoxia trial. This reduction in the rate and capacity for aerobic energy contribution coupled with the need for a greater utilization of the anaerobic capacity in the hypoxia trials, when integrated within brain centers, might have led to the motor cortex reducing central motor drive to the exercising skeletal musculature in order to reduce MPO and energy expenditure.

Notwithstanding the changes in the physiological milieu, there are also other factors to consider that would have potentially affected participants' MPO as they progressed through the hypoxic trials. It is plausible participants became increasingly aware of heightened sensory feedback informing the brain of the compromised metabolic rate during the hypoxic trials, and the subsequent development of fatigue and subsequently allocated time to deliberative decision-making. It has been hypothesized that incoming sensory afferent feedback, knowledge of the trial distance or duration (Swart et al., 2009), current momentary perceived effort influenced by prior experience (Mauger et al., 2009), current expectations (such as outcome and strategic goals) (Baden et al., 2004; Renfree et al., 2014) and knowledge of current physical capacities (Renfree et al., 2014) are integrated into decision making processes during self-paced exercise. At what point information resides in different brain centers and moves across sub-conscious, pre-conscious and conscious states is difficult to discriminate; however if the participant consciously

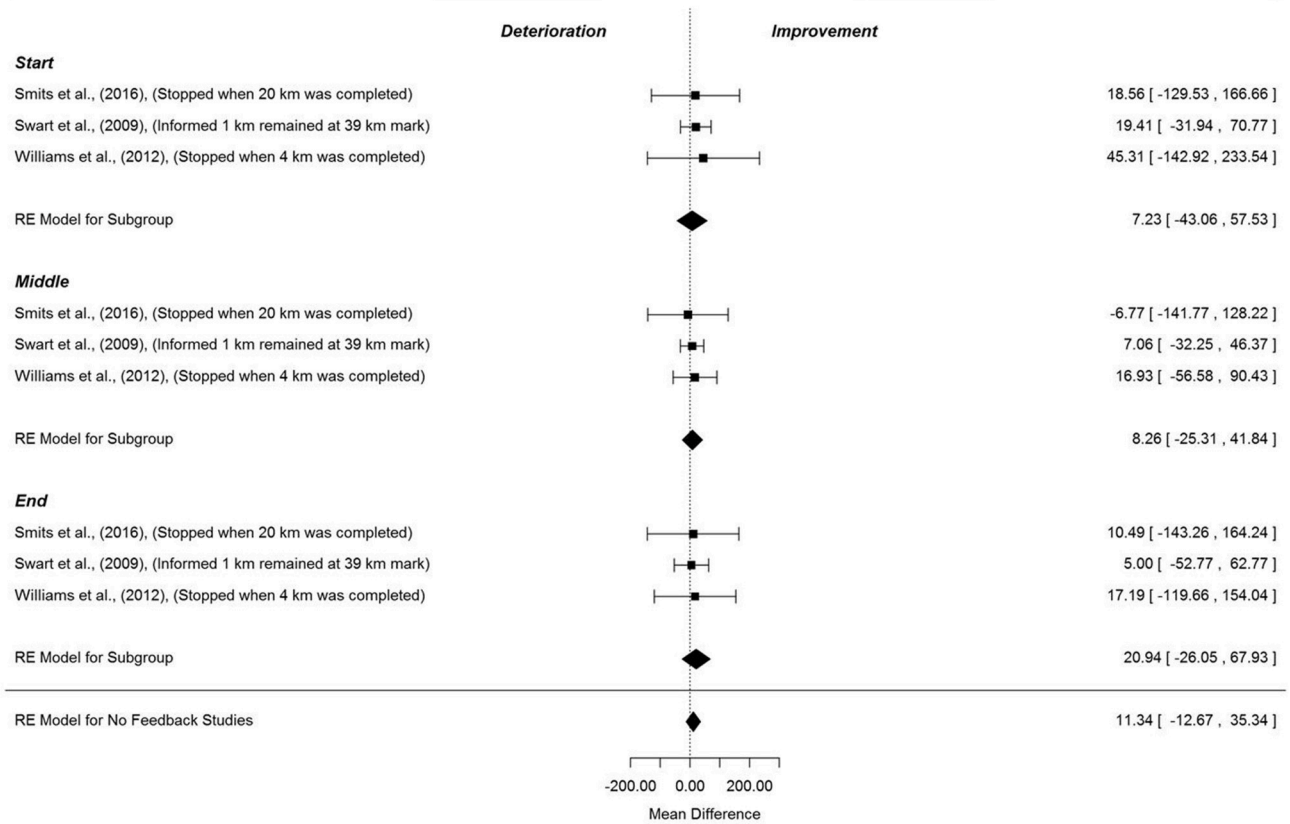

FIGURE 10 | Forest plot for no feedback meta-analysis illustrating power output during start, middle and end sections compared to full feedback. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within the meta-analysis. The diamond represents the overall mean difference for each split with the width of the diamond signifying the 95% CIs.

perceives their actual level of exertion is above that which they would normally expect at a particular point in the trial then they might decide to reduce their level of effort accordingly (de Koning et al., 2011; Micklewright et al., 2015). It would seem likely that in the hypoxic trial participants consciously regulated their exercise intensity, as postulated by the psycho-biological model, and that a decision was made to reduce MPO in order to stave off developing fatigue apparent during the middle and end trial sections (Marcora, 2010).

It is worth highlighting however that one investigation reported no significant changes in MPO across all three trial sections during hypoxic trials compared to the normoxia trial (Clark et al., 2007). In this study when well-trained cyclists' first (5 min) time-trial was either at 3200- or 2200-m simulated altitude, they demonstrated a conservative approach in the start section of subsequent trials at lower simulated altitudes (200- and 1200-m respectively). The authors attributed this observation to an order effect rather than a learning effect, as participants had completed two familiarization trials in normoxia before completing trials at the various altitudes levels. Therefore, it seems apparent when participants are blinded to high simulated altitude they may subsequently select an inappropriate starting pace in subsequent trials at lower altitudes. Finally, it is worth noting that the pacing index change score for the trials in the Amann et al. (2006) study looks markedly different compared to the other studies (**Figure 12A**). In this study, a more aggressive pace was attempted in the hypoxia trial relative to the normoxia trial which led to a marked reduction in middle and end section MPO, suggesting that the initial pace was considerably misjudged.

The meta-analysis found no significant evidence that hyperoxia improves overall trial MPO or section MPO, despite previous literature supporting the benefits of inhaling hyperoxic air during time-trials (Peltonen et al., 1995, 1997, 2001a,b; Nielsen et al., 1999; Amann et al., 2006; Tucker et al., 2007). However, this might be attributable to the small sample size and so further research is warranted to determine the efficacy of inspiring hyperoxic gas mixtures and to understand how exercise is regulated when athletes inhale oxygen enriched air. It is important to mention that participants in the included studies were also blinded to the fact that they were inpsiring oxygen enriched air (Amann et al., 2006; Tucker et al., 2007). Therefore, whether knowledge of breathing hyperoxia encourages a

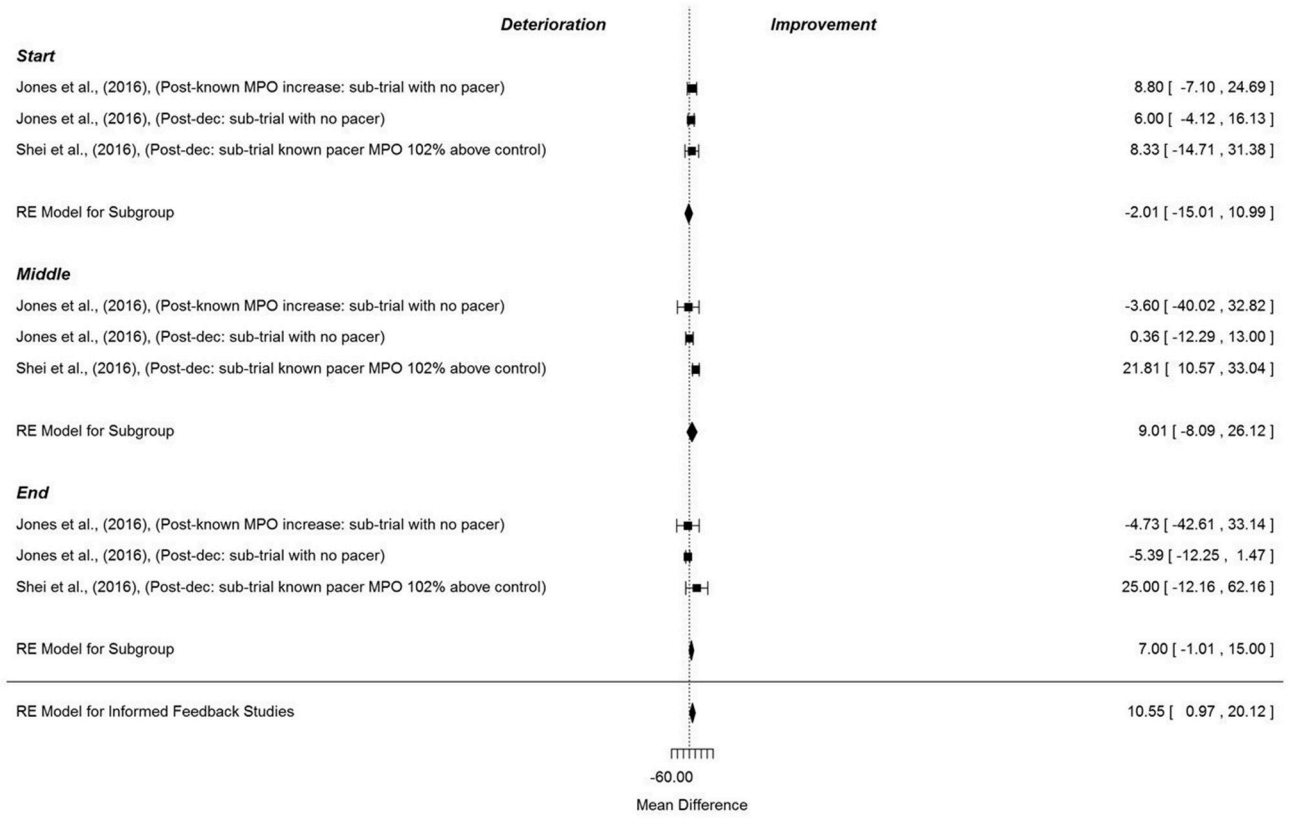

FIGURE 11 | Forest plot for informed feedback meta-analysis illustrating power output during start, middle and end sections compared to full feedback. Squares represent individual study mean difference and the lines represent 95% CIs. The size of the square is proportional to the weight of the study within the meta-analysis. The diamond represents the overall mean difference for each split with the width of the diamond signifying the 95% CIs.

conscious decision to upregulate exercise intensity or not, compared to a normoxic trial, requires further exploration.

Pacing and Heat-Stress

When participants were exposed to hot and humid conditions the meta-analysis clearly demonstrated participant's MPO was significantly reduced compared to more temperate conditions. Notably there was less variability demonstrated in the RE model for the different sub-group sections (start, middle and end) in the heat-stress studies than in the studies which manipulated oxygen content. A possible explanation might be the nature of heat interventions, as a marked change in room temperature is easily identifiable whereas a change in the oxygen content of the air being inspired is not. It is therefore likely that appropriate adjustments to power output were made sooner in the heat trials due to earlier changes in afferent feedback.

In a similar trend to the hypoxia trials, the meta-analysis found power outputs produced during the start section of trials were similar to the control (temperate) trial. This finding is supportive of previous reports where the initial exercise intensity in hot conditions follows a similar pattern to temperate conditions for the first ∼10 to 15 min of exercise, despite elevated skin temperatures, thermal sensation and perceived effort (Tatterson et al., 2000; Tucker et al., 2004; Ely et al., 2009; Périard et al., 2011). However, if participants are inexperienced at time-trialing in heat they can misjudge the start section of the trial by beginning the trial too aggressively (Schlader et al., 2011b; Castle et al., 2012). Notably, for a number of the heat-stress trials, the pacing index change score was relatively greater and lower for the start and end sections, respectively, compared to the temperate condition trial (**Figure 12B**). This indicates an inability for participants to adopt an optimal pacing strategy from the onset of the trial in the hot condition. Subsequently the pacing strategy was substantially adjusted as the trial progressed which might partly explain why a reduced performance outcome is observed compared to the temperate condition.

In some of the studies, the exposure to the heat-stress prior to the time-trial (i.e., during the standardized warm-up) was $\leq$ 15 min in duration (Castle et al., 2012; Schmit et al., 2016; VanHaitsma et al., 2016) or longer (Abbiss et al., 2008; Altareki et al., 2009). However, in the remaining studies either no mention was made as to participants being exposed to the heat-stress

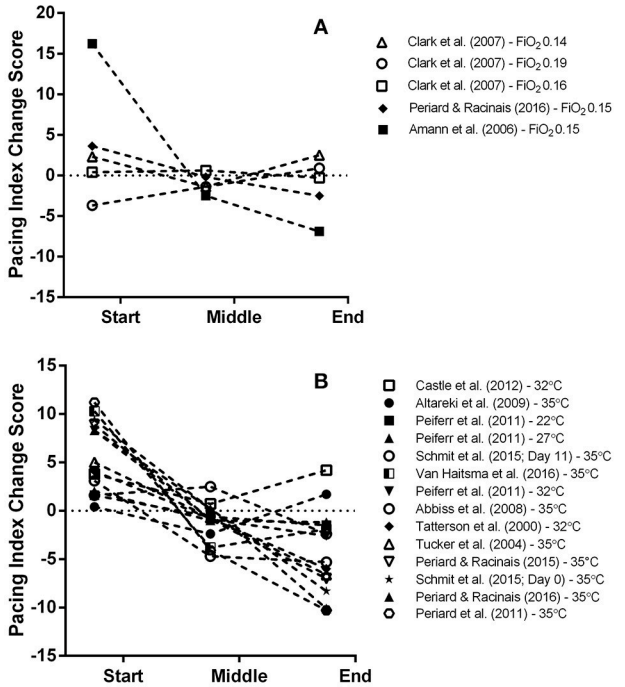

FIGURE 12 | Exploratory graphical analysis of the pacing index change score difference between control and hypoxic condition (A) and control and heat-stress condition **(B)** for each individual segment. For an explanation of how the pacing index change score was calculated please refer to section Data analysis of the Methods.

prior to the time-trials, or they did not expose their participants to heat-stress in the warm-up period (Tatterson et al., 2000; Tucker et al., 2004; Peiffer and Abbiss, 2011; Périard et al., 2011; Périard and Racinais, 2015, 2016). If we consider the role of afferent feedback, perceived effort and thermal sensation in exercise regulation during exercise in the heat, then the absence of any exposure to heat prior to the time-trial would delay the onset of the development of heightened afferent feedback from thermoreceptors and any subsequent increase in perceived effort and thermal sensation (Schmit et al., 2016). In this scenario, participants might begin with a starting pace at a higher level (similar to a temperate condition), than if they had been exposed to the heat-stress during the warm-up period, due to the role skin temperature has in mediating thermo-behavioral responses (Schlader et al., 2011a). If participants began the heat-stress trial too aggressively, due a lack of change in thermal sensation from no pre-time-trial heat exposure, then they would need to considerably adjust their exercise intensity in the middle section of the trial to avoid prematurely fatiguing. Unfortunately, due to time-trial duration varying markedly between studies in the meta-analysis, it was not possible to demonstrate if this trend was indeed apparent from a comparison of start, middle and end trial sections between those studies involving pre-time-trial heat exposure and those that did not.

Mean power output was observed to decline in the middle and end sections of hot condition trials compared to temperate trials across studies which investigated both short and long time-trial durations. An inspection of core temperature data from these studies revealed that during time-trials lasting $\leq$ 30 min and/or where participants weren't exposed to heat up to 20 min before the trial, core temperature changed in a similar fashion for trials in both temperate and hot conditions. This would suggest pacing was adjusted to avoid significant changes in core temperature (Nybo, 2008). The potential afferent driver of the adjustment in the pacing strategy is outside of the scope of this meta-analysis, however, it could be due to other factors such as significant increases in skin temperature (Jay and Kenny, 2009), perceived exertion (Crewe et al., 2008) and thermal sensation (Schlader et al., 2010).

It is well known that during exercise in hot conditions there is an increased skin blood flow, in order to dissipate heat, which compromises blood flow to the working musculature and in turn leads to a reduction in gross mechanical efficiency (Hettinga et al., 2007). This consequence appears to become more problematic in continuous exercise lasting over 30 min in duration, as the maintenance of blood pressure takes priority over blood flow to the skin and working musculature (Casa, 1999). As a result, differences between the rates of metabolic heat production and net heat loss, and the exchange of evaporative heat, will lead to increased core temperatures for longer duration time-trials in hot compared to temperate conditions (Jay and Kenny, 2009). This is due to the uncompensatable heat gain over the course of the trial, significantly impacting circulatory responses, leading to a decrease in $\dot{V}$ O_2 and subsequent reduction in exercise intensity (Périard and Racinais, 2016).

Finally, some of the reduction of the power output in hot conditions could be attributable to discrepancies across studies which made no mention, or did not account for, one or more of the following factors: (i) whether the temperature of the control trial was in accordance with laboratory recommendations for exercise in a thermoneutral environment (18 to 23°C, relative humidity <70%) (Tucker et al., 2004; Altareki et al., 2009; Peiffer and Abbiss, 2011) and ii) if the relative increase in temperature from control to intervention was considered. For instance, the temperature difference between intervention and control conditions in the study by Altareki et al. (2009) was 22°C compared to 15°C by Périard et al. (2011). It is possible that the relative temperature increase from cool ($\leq$18°C) to hot ($\geq$30°C) conditions may be influential in terms of exercise regulation and may warrant further exploration. Third, there were a number of confounding variables in terms of comparing data across studies such as, a lack of information concerning the clothing participants wore during the trials, some studies not allowing participants to consume fluids during the trial (Altareki et al., 2009; Peiffer and Abbiss, 2011) and the lack of a consistent approach to using convective cooling methods providing equivalent volumes and rates of air flow to replicate the conditions that athletes encounter during outdoor competitions (25–40 km h^{-1} when cycling) (Dugas et al., 2009). Indeed, many of the heat intervention studies in the current meta-analysis provided less than the recommended volumes and rates of air flow during indoor cycling trials (M $\pm$ SD: 19.2 $\pm$ 8.9 km h^{-1}). In addition, three studies did not mention whether convective cooling was used in heat trials (Castle et al., 2012; Schmit et al.,

2016; VanHaitsma et al., 2016). It is, of course, also possible that researchers deliberately provided sub-optimal convective air flow to induce hyperthermia in their participants. Nonetheless, the differences in available air flow to dissipate heat may partly explain the variation of power output between individual studies.

Pacing and Pre-cooling Interventions

In general, pre-cooling interventions aim to counter the effects of exercising in heat by cooling the body prior to the exercise to increase the "thermal reservoir" (Nielsen, 1994; Levels et al., 2012). In the current meta-analysis the pooled data for studies investigating pre-cooling interventions demonstrated no significant effect on overall trial or trial section MPO compared to the control trial, despite a number of the studies having reported small increases in MPO following 20 min of cold water (14°C) lower body immersion (Duffield et al., 2010), ingestion 900 mL of cold fluid (2°C) (Byrne et al., 2011) or after wearing a cooling vest (18 min during warm-up) (Gonzales et al., 2014). Some of the included studies in the meta-analysis had reported lowered skin and muscle temperatures, and perceived thermal sensation for their participants following the pre-cooling intervention prior to their time-trials. These parameters have been suggested to reduce thermoregulatory responses by delaying the redistribution of cardiac output to the periphery and sweat responses for heat dissipation (Casa, 1999), during the early stages of exercise (∼15 min) following pre-cooling strategies, although once exercise exceeds ∼10 min, any physiological and perceptual changes induced by pre-cooling appears to dissipate (Minett et al., 2011). Therefore, any change in the pacing strategy resulting from pre-cooling interventions would be likely detected in the start section of trials. In the current meta-analysis, the exercise duration of the included studies were considerably longer than 10 min (M ± SD: 40 ± 22 min), and so the start section took a number of minutes to complete however no significant changes in MPO for the start section of the trials was detected. A closer inspection of core temperature data from the original manuscripts revealed that with the exception of ingesting 900 mL of cold fluid (Byrne et al., 2011), the pre-cooling strategies of the included studies were unsuccessful in lowering body temperature before the start of the time-trial, compared to the control condition (Duffield et al., 2010; Barwood et al., 2012; Gonzales et al., 2014). This is likely attributable to the effectiveness of the intervention itself and the time period between the completion of the pre-cooling method and warm-up or between the warm-up and the start of exercise (M ± SD: 12 ± 8 min).

Given the findings of the meta-analysis, careful consideration should be given as to whether pre-cooling interventions are of benefit to athletes' performances when competing in a hot environment, especially as the logistical aspects of undertaking pre-cooling interventions can be disruptive. In addition, if the pre-cooling intervention has the potential to lower muscle temperature then it might actually be detrimental for performance in short duration (<12 min) events (Levels et al., 2012). It is worth noting however that with only four studies meeting the inclusion criteria for this meta-analysis, our findings are likely to be underpowered and further research in this area is needed to fully elucidate the effects of pre-cooling interventions, particularly in terms of exercise regulation during the start section of the trials.

Finally two studies (Duffield et al., 2010; Byrne et al., 2011) had cyclists refrain from consuming water during their respective trials. Previous reports have shown that removing the physical act of drinking may alter the participant's perception of thirst, and therefore, potentially influence their motivation and pacing to limit further fluid loss (Dugas et al., 2009; Cheung et al., 2015). This was demonstrated by Dugas et al. (2009) who restricted the fluid consumption of participants below *ad libitum* and found time-trial performance was impaired in male cyclists. It's plausible that the effectiveness of the pre-cooling methods included in the analysis, may have been confounded by the absence of fluid consumption in some studies.

Pacing and Feedback Manipulations

Overall, positive, neutral and no feedback had no significant effect on overall or individual segment MPO (**Figures 7**, **9**, and **10**, respectively). However, negative feedback, or more specifically performance deception feedback, did elicit significant improvements in performance and individual segment power output. In these studies, participants were racing against a virtual on-screen pacer they believed was programmed to mimic MPO from their previous best trial performance, however in reality the pacer was programmed with a MPO which was 2% greater (Jones et al., 2016a,b; Shei et al., 2016). Participants tended to produce an increased power output at the start of the "deception" trial and also produced a significantly greater power output during the middle section. The variability of power output was relatively low in these trials which might suggest the change in power output, caused by the deception, was sufficient to improve performance but not so great as to be intolerable. Previous work by Stone et al. (2012) found when participants were deceived of a 2% increase in the MPO they demonstrated a greater anaerobic energy contribution at 90% of a 4000-m time-trial and a concomitant significant improvement in power output. The meta-analysis did not find an improvement for the end section of trials, however this might be because trials were segmented into thirds rather than 10% bins as in the Stone et al. (2012) study. The meta-analysis also demonstrated informed feedback (**Figure 11**; where participants completed a time-trial subsequent to being informed the MPO of a pacer in their previous trial was set to a greater exercise intensity than their baseline performance) led to improved trial performance compared to an original (baseline) time-trial.

Collectively, these findings from different forms of feedback indicate that participants are preconditioned to start exercise at a similar intensity in the early stages of a trial despite the absence or manipulation of feedback. However, negative feedback, such as deceiving participants about their actual power output can achieve subtle increases in power output during the start and middle trial sections and an improved overall trial MPO. However, recent research suggests the presence of an on-screen avatar pacer in these studies, provides the additional motivation of a "competitor," and may partly explain the increases in power output observed (Stone et al., 2012; Shei et al., 2016). It is worth

noting however, that the neutral feedback condition in the meta-analysis did not demonstrate a significant improvement in trial MPO when a "competitor" pacer was present, as compared with the baseline (control) time-trial where no pacer was present.

LIMITATIONS OF THE META-ANALYSIS

A limitation of the meta-analysis was that a large number of studies were discounted because they did not report power output data. Power output was decided upon as the performance metric of choice for the analysis as measurement error data is consistently reported for this parameter in the pertinent literature. Consequently, a number of pacing studies providing velocity or time data were excluded, despite meeting all other criteria. Additionally, three studies in rowing, where power output was measured, were excluded because once studies were allocated into the defined themes the sample size in each group was not sufficient to be included in a meta-analysis (Mujika et al., 2010; Taylor et al., 2014; Murray et al., 2016). As a consequence of these constraints, modes of exercise researched in the literature such as running, swimming, skating and rowing were excluded. The exclusion of a large quantity of studies from the pacing literature subsequently impacted the sample size, particularly when studies where allocated into themes for the meta-analysis. For example, the null effect found with regard to pre-cooling interventions in hot conditions were certainly impacted by the small sample size ($n = 4$) and the diverse range of methods and experimental designs. Interpretations of exercise regulation in the included themes should therefore be viewed with caution, until additional studies suitable for a meta-analysis, are available to provide greater understanding of exercise regulation within the pacing literature. A further limitation of the analysis is that the pacing resolution was compromised, because trial power output data could only be segmented in to three sections (start, middle and end sections) rather than for example, 10% bins which would have provided greater insight into changes within the pacing strategy for the various interventions. This was due to the large variability in trial duration and distance being used in the included studies. Finally, with the exception of one study that recruited one female participant into a male cohort (Périard and Racinais, 2015), only male participants' were recruited in the included studies. Therefore, the findings have an obvious gender bias, as unfortunately, there is a dearth of studies to date investigating changes in the pacing strategies exhibited by females.

APPLICATION AND FUTURE RECOMMENDATIONS

We recommend that researchers be attentive to the criteria for developing high quality research, such as those outlined in the PEDro scale (Machado et al., 2016) to provide more robust datasets to undertake a meta-analysis. Additionally, it would be helpful if research investigating different manipulations on pacing provided a pacing index, which is a relatively simple measure to report, rather than focusing primarily on overall performance outcomes. The pacing index allows for direct comparisons between genders, age or in the instance of a meta-analysis, between different studies of similar themes (Le Meur et al., 2011; Wu et al., 2014).

The notable delay in exercise regulation during the start section of time-trials in hypoxia and heat-stress highlights the importance of taking into account the exposure time to the environmental stressor prior to and during a time-trial or a competitive race. Experimental investigators and coaches need to be mindful of the effects that exposure to different environmental stressors will have on the participant's/athlete's pacing strategy and overall performance. From a practical perspective, coaches and athletes need to consider adapting the "normal" race pacing strategy to the environmental conditions to mitigate against an error in pace judgment occurring from the start of the race. For athletes who compete globally, their race preparation needs to include practicing different pacing strategies under different race conditions to arrive at the optimal pacing strategy for each environmental condition. Coaches might consider the use of *subtle* deceptive negative feedback in training to elicit a change in pacing strategy and athlete performance. Having achieved a performance improvement there appears to be some evidence that the performance can be maintained even after the deception is revealed, however in a real-world setting this practice would be controversial, as there are ethical, integrity and moral aspects to be considered, which could be potentially harmful to the athlete and to the relationship with the coach.

Finally, there are a number of specific considerations for experimental investigators that were highlighted in conducting this meta-analysis. Firstly, when undertaking heat-stress trials in laboratory settings it is important consider: (i) the amount of change in temperature between control and intervention trials, (ii) whether the control trial represents conditions appropriate for a thermo-neutral trial, (iii) the availability of fluids for *ad libitum* drinking, (iv) the clothing worn and (v) adopting convective cooling methods that reflect outdoor conditions. Research studies manipulating the oxygen content of inspired air should be mindful that order effects may occur when trials simulating moderate altitude are followed by lower altitude trials. Studies manipulating feedback using a pacer/pacing device should consider the size of the effect this might have on the primary outcome measures exhibited by the participants relative to the size of the effect from the form of feedback being manipulated.

CONCLUSIONS

The meta-analysis demonstrated that in trials where the environmental conditions were manipulated (e.g., hypoxia, hyperoxia or heat-stress) MPO was generally not significantly different to the control (normoxic or temperate) condition for the start section. However, MPO in the middle and end sections was found to be significantly reduced when participants were exposed to different levels of hypoxia and heat-stress. The available data demonstrated hyperoxia, pre-cooling strategies and some forms of feedback (positive, neutral and no feedback) did not

significantly change trial or section MPO compared to their control condition. However, negative feedback, such as deceiving participants about their actual power output when competing against a virtual competitor, can result in small but significant increases in power output during the start and middle sections of trials and an improved trial MPO. Once informed of the deception, the meta-analysis also demonstrated participants can still produce an improved MPO in comparison to their original baseline time-trial.

AUTHOR CONTRIBUTIONS

MD, BC, MW, SS, LG, PS, KT: (1) substantial contributions to the conception and design of research, acquisition of literature, analysis and interpretation of data for the manuscript; (2) substantial contributions drafting the work or revising it critically for important intellectual content; (3) substantial contributions to the final approval of the version to be published; (4) all authors acknowledge and agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Pacing Profiles in Competitive Track Races: Regulation of Exercise Intensity Is Related to Cognitive Ability

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Pacing has been defined as the goal-directed regulation of exercise intensity over an exercise bout, in which athletes need to decide how and when to invest their energy. The purpose of this study was to explore if the regulation of exercise intensity during competitive track races is different between runners with and without intellectual impairment, which is characterized by significant limitations in intellectual functioning (IQ $\leq$ 75) and adaptive behavioral deficits, diagnosed before the age of 18. The samples included elite runners with intellectual impairment ($N = 36$) and a comparison group of world class runners without impairment ($N = 39$), of which 47 were 400 m runners (all male) and 28 were 1500 m-runners (15 male and 13 female). Pacing was analyzed by means of 100 m split times (for 400 m races) and 200 m split times (for 1500 m races). Based on the split times, the average velocity was calculated for four segments of the races. Velocity fluctuations were defined as the differences in velocity between consecutive race segments. A mixed model ANOVA revealed significant differences in pacing profiles between runners with and without intellectual impairment ($p < 0.05$). Maximal velocity of elite 400 m runners with intellectual impairment in the first race segment (7.9 ± 0.3 m/s) was well below the top-velocity reached by world level 400 m runners without intellectual impairment (8.9 ± 0.2 m/s), and their overall pace was slower ($F = 120.7$, $p < 0.05$). In addition, both groups followed a different pacing profile and inter-individual differences in pacing profiles were larger, with differences most pronounced for 1500 m races. Whereas, male 1500 m-runners without intellectual impairment reached a high velocity in the first 100 m (7.2 ± 0.1 m/s), slowly decelerated in the second race segment (−0.6 ± 0.1 m/s), and finished with an end sprint (+0.9 ± 0.1 m/s); the 1500 m runners with intellectual impairment started slower (6.1 ± 0.3 m/s), accelerated in the second segment (+0.2 ± 0.7 m/s), and then slowly decreased until the finish ($F = 6.8$, $p < 0.05$). Our findings support the hypothesis that runners with intellectual impairment have difficulties to efficiently self-regulate their exercise intensity. Their limited cognitive resources may constrain the successful integration of appropriate pacing strategies during competitive races.

Keywords: running, 400 m, 1500 m, track and field, intelligence

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INTRODUCTION

A vital component for success in running events is the pacing strategy (Abbiss and Laursen, 2008; Tucker, 2009). The optimal pacing strategy can be a learned pattern, based on extensive experience gained during training and previous competitions (Foster et al., 2009, 2014); however, many factors can affect the pacing strategies adopted during running events. An individuals' pacing strategy is dependent on performance goals (e.g., world record attempt vs. qualification during heats; Thompson, 2015), environmental conditions (e.g., temperature) (Tucker, 2009; Roelands et al., 2013) and the presence of opponents (Konings et al., 2016a,b). In competition, athletes must set and adjust their pace based on feelings such as perceived exertion (Abbiss and Laursen, 2008) or pain (Mauger, 2014). Hence, the actual pacing profile observed during competition does not always resemble the pre-planned strategy adopted by the athlete and/or the coach. Competitors need to take into account the distance remaining until finish and also the actions of their opponents (St Clair Gibson et al., 2006; De Koning et al., 2011; Swart et al., 2012). When considering an athletic event involving direct competition between two or more individual athletes, the environment becomes even more complex (Renfree et al., 2014; Konings et al., 2016a,b).

Several recent reviews have described pacing as a process of decision-making (Renfree et al., 2014; Smits et al., 2014). It was recently proposed that effective cognitive control during performance requires both proactive, goal-driven processes and reactive, stimulus-driven processes (Brick et al., 2016). Although the importance of decision-making upon effort regulation was acknowledged (De Koning et al., 2011; Renfree and St Clair Gibson, 2013), very little is understood about decision-making processes involved in pacing or the underlying psychological mechanisms. To understand how exercisers regulate their exercise capacity, and to identify the role cognition plays in optimal self-regulation, the study of pacing in athletes with intellectual impairments could be an interesting design. Although pacing is commonly accepted as an important cognitive determinant in running (St Clair Gibson et al., 2006; Tucker et al., 2006; Abbiss and Laursen, 2008; Hanon et al., 2008; Tucker, 2009; De Koning et al., 2011; Hanon and Thomas, 2011; Saraslanidis et al., 2011; Thiel et al., 2012; Reardon, 2013; Renfree et al., 2014; Smits et al., 2014) only one study has investigated pacing in individuals with intellectual impairment. Micklewright et al. (2012) demonstrated an explicit link between pacing and cognitive development by looking into pacing behavior of school children in different stages of cognitive development. The study confirmed that developing a pacing strategy is at least in part determined by cognitive mechanisms. In their study, after doing a control test for age (5–14 years), pacing differences were distinguished between groups of school children in different stages of cognitive development. In another study it was demonstrated in a large sample of elite swimmers, athletes, basketball- and table tennis players with intellectual impairment that their cognitive abilities relevant to sport in general (e.g., visual processing, reaction and decision making speed, short-term memory and fluid reasoning) were significantly reduced compared to equally well-trained athletes without impairment (Van Biesen et al., 2016b), so it can be assumed that specific cognitive abilities relevant to pacing and performance in running (i.e., decision making, anticipation) will also be influenced by having an intellectual impairment. A first study exploring this analyzed the ability of runners with an intellectual impairment to maintain a pre-planned velocity over 400 m, an essential aspect of pacing (Van Biesen et al., 2016a). It was demonstrated that runners with an intellectual impairment were not able to maintain the required sub-maximal velocity and accelerated toward the end, in contrast to athletes without impairment of similar training volume. This provided the first evidence for the impact of cognitive ability on pacing ability. The present study will now focus on exploring data of athletes in actual competitions to explore how cognitive ability impacts on pacing and performance in competition.

The purpose of the present study was to explore if the regulation of exercise intensity during competitive 400 and 1500 m track races is different when pacing profiles are compared between high level runners with and without intellectual impairment. It is hypothesized that runners with intellectual impairment will have a different, more variable pacing strategy compared to runners without intellectual impairment. If we detect an effect of having an intellectual impairment on pacing profiles during the race, this will provide evidence to support the assumption that the regulation of runners' exercise intensity over the race is, at least partly, dependent on their cognitive skill level. In addition, a difference in pacing profiles between the groups will create an evidence-based rationale for organizing separate competitions for runners with intellectual impairment in the Paralympic Games.

MATERIALS AND METHODS

Participants

Data for this study were derived from a sample of 47,400 and 281,500 m runners, of which 36 elite runners with mild intellectual impairment (28 males and 8 females) and a comparison group of 39 runners without impairment (34 males and 5 females). The runners with intellectual impairment competed at the 2014 Open European Championship Athletics, in Bergen Op Zoom, The Netherlands, organized by the International Federation for Para-Athletes with Intellectual Impairment (INAS). They competed in 400 or 1500 m races and all met the criteria for diagnosis of intellectual disability as set by the American Association on Intellectual and Developmental Disabilities: IQ $\leq$ 75, significant deficits in adapted behavior and manifested before the age of 18. More specifically, the IQ scores of the runners with intellectual impairment were 64.7 $\pm$ 8.7 (male 400 m), 59.6 $\pm$ 8.7 (male 1500 m) and 60.4 $\pm$ 7.9 (female 1500 m). None of the participants had severe intellectual impairment or a genetic syndrome (e.g., Down Syndrome). The runners with intellectual impairment (aged 24.4 $\pm$ 4.5 years) had on average 9.6 $\pm$ 4.8 years of experience and 9.4 $\pm$ 4.0 h/week training volume. The control data was obtained from the International Association of Athletics Federation's (IAAF) 12th World Championships in Berlin in 2009 (Helmar et al., 2009a,b).

For the 1500-m world record performances of men and women, split times were obtained from http://www.iaaf.org/ and http://wn.com/ respectively. Descriptive information of the participants in the control group (age, training volume, IQ scores) was not available. The study was approved by the local ethics committee (Commissie Medische Ethiek, KU Leuven).

Procedure

Pacing profiles were analyzed by means of 100 m split times (for 400 m races) and 200 m split times (for 1500 m races). The most recent World Record data were retrieved from the IAAF website (Reardon, 2013; International Association of Athletics Federations, 2016). Split times were publically available on the IAAF website for the control group, and split times were calculated for the runners with intellectual impairment on the basis of video images recorded during the race. Their races were filmed with three 25 Hz SONY Cameras for the 400-m race, and one camera for the 1500-m race. The positions of the cameras are depicted in **Figure 1**. During the 1500-m race a large cone was placed in view of the camera as a reference point for the calculation of the 100, 500, 900, and 1300-m split time. Before the start of every 400-m race, the camera captured the first athlete in starting position (lane 1 or the most inner athlete). From the moment the athletes took off, the camera was switched to the designated split time mark to capture every athlete passing by.

Data Reduction and Calculation

Based on the split times and distance, the average velocity was calculated for four segments of the race: 0–100 m, 100–200 m, 200–300 m, and 300–400 m for the 400 m races and 0–100 m, 100–500 m, 500–1000 m, and 1000–1500 m for the 1500 m races. Velocity fluctuated within the segments indicating accelerations (i.e., positive fluctuations) or decelerations (i.e., negative fluctuations).

Data Analyses

Statistics were performed using SPSS (version 19.0, SPSS Inc., Chicago Ill, USA) with level of significance set at $p < 0.05$. For the 400 m race, a mixed model ANOVA was performed to analyze the differences in running patterns over different time points during the race (within factor), between male runners with and without intellectual impairment (between factor), for heats and finals. The mixed model ANOVA was also performed to analyze the differences in running patterns over different time points (within) between runners with and without intellectual impairment (between) in the 1500 m finals. Intra-individual coefficients of variation of running speed within each race were calculated based on 100-m split times (for the 400 m races) and 200-m split times (for the 1500-m races).

RESULTS

400 m Group Differences in Race Strategy

Figure 2 shows the overall pacing strategy during the men's 400-m races. Average velocity plots per segment are shown for the heats and finals. No significant differences in velocity were found between finals and heats for runners without intellectual impairment, whereas average velocity at all time-points was higher in the final race than during heats for runners with intellectual impairment. Both groups initially performed an acceleration followed by a deceleration, however, the pacing strategy significantly differed between both groups of runners in heats and finals as shown by the significant interaction effect (**Table 1**). The runners without intellectual impairment gradually

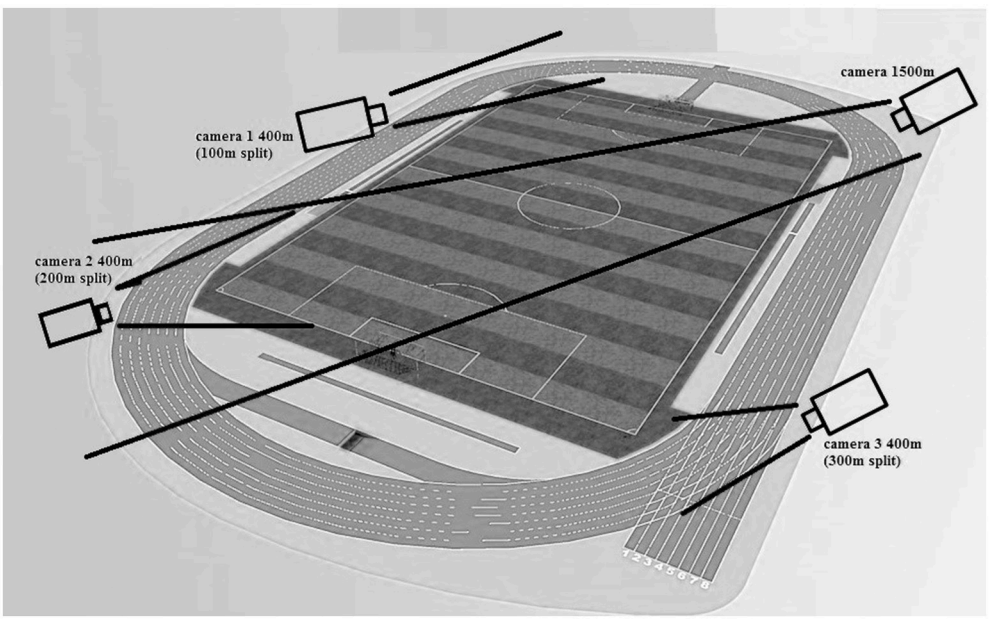

FIGURE 1 | Camera positions for split time calculations during 400 and 1500-m races.

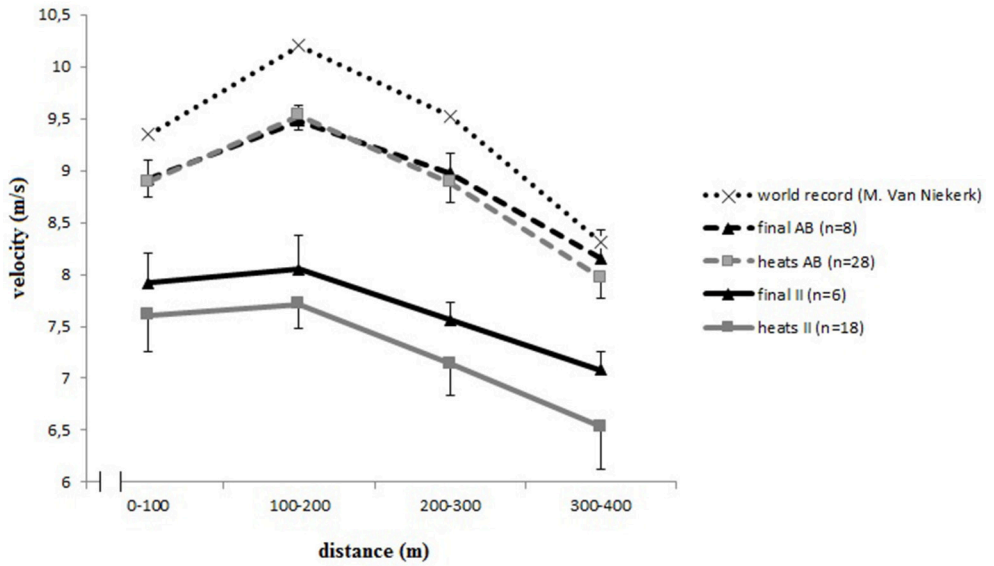

FIGURE 2 | Men's 400-m pacing profiles. INAS, International Federation for para-athletes with intellectual impairment; II, intellectual impairment; AB, able bodied.

TABLE 1 | Mixed model Anova results for velocity fluctuations in four races: 400 m male final and heats, 1500 m male and female final between runners with and without intellectual impairment.

	df	*F*	η^2	*p*
400 M FINAL MEN				
ME_w velocity	1, 14	67.23	0.95	<0.001*
ME_b impairment	1, 14	241.56	0.95	<0.001*
IE velocity × impairment	1, 14	12.50	0.79	0.001*
400 M HEATS MEN				
ME_w velocity	1, 46	333.74	0.96	<0.001*
ME_b impairment	1, 46	1265.90	0.97	<0.001*
IE velocity × impairment	1, 46	123.33	0.63	<0.001*
1500 M FINAL MEN				
ME_w velocity	1, 14	5.25	0.61	0.02*
ME_b impairment	1, 14	45.21	0.79	<0.001*
IE Velocity × impairment	1, 14	35.36	0.92	<0.001*
1500 M FINAL WOMEN				
ME_w velocity	1, 12	10.31	0.79	0.004*
ME_b impairment	1, 12	58.94	0.86	<0.001*
IE velocity × impairment	1, 12	66.79	0.96	<0.001*

*Df, degrees of freedom; *$p < 0.05$, ME_w, main effect of the within-subjects factor; ME_b, main effect of the between subjects factor; IE, interaction effect.*

decelerated halfway after a fast start. The deceleration, traveling between 9.5 and 8 m/s, concluded with a steeper decline in the latter part. For the runners with intellectual impairment, the decline occurred with a steep descent from 8 until 7 m/s. The result of the *post hoc* analyses as shown in **Table 2** indicated that fluctuations in the final race segment were significantly different between both groups of runners in the heats ($F = 7.1$, $p < 0.05$); however, not for the finals ($F = 7.1$, $p = 0.1$).

Overall, runners with intellectual impairment demonstrated a slower running speed than runners without intellectual impairment. The ANOVA showed a significant main effect of the within factor velocity in the 400 m heats and 400 m final races (**Table 1**). In the first race segment (0–100 m) of the final, runners with intellectual impairment accelerated to a velocity of 7.9 m/s, whereas runners without intellectual impairment accelerated to 8.9 m/s ($F = 120.7$, $p < 0.05$, **Table 2**). Another difference between both groups was observed in the second race segment (100–200 m). In both the final and the heats, runners with intellectual impairment accelerated (0.1 ± 0.2 m/s); however, this acceleration was less pronounced than demonstrated by the runners without intellectual impairment (0.6 ± 0.1 m/s); The latter group reached their maximal speed after 200 m ($F = 21.4$, $p < 0.05$).

400 m Individual Differences in Race Strategy

Coefficients of variance (CV) were calculated as a measure of intra-individual variance. The average CV of the male runners with intellectual impairment who ran the 400 m final, semi-finals, and/or qualifications in Bergen op Zoom was 8.1 ± 2.9% whereas the coefficient of variation during the World Championships in Berlin was 6.9 ± 1.6%.

1500 m Race Group Differences

Figures 3, **4** display the pacing strategies applied by respectively male and female runners during their 1500 m final race. The velocity fluctuations within every race segment are quantified in **Table 2**. An overall comparison of the distance by velocity plots (**Figure 3**) shows that male runners with and without intellectual impairment followed a different, almost inverse, pacing profile, confirmed by a significant interaction effect

TABLE 2 | Comparison of velocity fluctuations over four segments of the races between runners with and without intellectual impairment.

	With intellectual impairment			Without intellectual impairment			*F*	ES Cohen d
	Mean (m/s)	SD	95% CI	Mean (m/s)	SD	95% CI		
400 M FINAL (MEN, *N* = 14)								
Q1	7.9	0.3	[7.6, 8.2]	8.9	0.2	[8.7, 9.1]	120.7*	3.9
Q2	0.1	0.2	[−0.1, 0.4]	0.6	0.1	[0.5, 0.7]	21.4*	3.2
Q3	**−0.5**	**0.2**	[−0.7, −0.3]	**−0.5**	**0.2**	[−0.7, −0.3]	1.2	0
Q4	**−0.5**	**0.3**	[−0.8, −0.2]	**−0.8**	**0.2**	[−1.0, −0.7]	7.1	1.2
400 M HEATS (MEN, *N* = 46)								
Q1	7.6	0.4	[7.4, 7.8]	8.9	0.2	[8.8, 8.9]	120.7*	4.1
Q2	0.1	0.3	[−0.1, 0.3]	0.6	0.2	[0.6, 0.7]	21.4*	2.0
Q3	**−0.6**	**0.2**	[−0.7, −0.5]	**−0.6**	**0.3**	[−0.7, −0.5]	1.2	0
Q4	**−0.6**	**0.2**	[−0.7, −0.5]	**−0.9**	**0.3**	[−1.0, −0.7]	7.1*	1.2
1500 M FINAL (MEN, *N* = 14)								
Q1	6.1	0.3	[5.9, 6.3]	7.2	0.1	[7.1, 7.3]	−6.8*	5.0
Q2	0.2	0.7	[−0.3, 0.6]	**−0.6**	**0.1**	[−0.7, −0.6]	3.8*	1.6
Q3	**−0.4**	**0.3**	[−0.6, −0.2]	0.3	0.0	[0.3, 0.3]	−7.6*	3.3
Q4	**−0.1**	**0.4**	[−0.3, 0.2]	0.9	0.1	[0.9, 1.0]	−7.6*	3.4
1500 M FINAL (WOMEN, *N* = 12)								
Q1	4.9	0.1	[4.8, 5.0]	6.5	0.1	[6.3, 6.6]	−28.8*	16.0
Q2	0.3	0.2	[0.1, 0.5]	**−0.8**	**0.1**	[−1.0, −0.6]	10.6*	7.0
Q3	**−0.5**	**0.2**	[−0.6, −0.3]	0.6	0.0	[0.6, 0.7]	−15.8*	7.8
Q4	0.3	0.6	[−0.1, 0.8]	0.2	0.0	[0.1, 0.3]	0.7	0.2

*Q1, first race segment (0–100 m); Q2, second race segment (100–200 m or 100–500 m); Q3, third race segment (200–300 m or 500–1000 m); Q4, fourth race segment (300–400 m or 1000–1500 m); CI, Confidence interval; SD, standard deviation; *$p < 0.05$, negative velocity fluctuations (= deceleration) is highlighted in bold.*

(**Table 1**). After reaching a relatively high velocity in the first 100 m (6.1 m/s), male runners without intellectual impairment controlled their pace and slowly decelerated in the second segment of the race (100–500 m) to finish with an end sprint (1000–1500 m), whereas runners with intellectual impairment started slower, accelerated in the second segment, and then slowly decreased velocity until the end ($F = 6.8$, $p < 0.05$). The comparison between female 1500 m runners with and without intellectual impairment (**Figure 4**) also revealed inverse pacing profiles between both groups of runners, with runners with intellectual impairment accelerating until 500 m, followed by a deceleration until 1100 m, and a variable strategy until finish. The runners without intellectual impairment did the opposite, decelerating between 100 and 500 m, followed by accelerating until 1300 m, and then maintaining their velocity until finish. Significant differences were found (**Table 1**) between the groups in the first three segments of the race (0–1000 m). Only in the final segment (1000–1500 m) both female groups slightly accelerated.

1500 m Race Intra- and Inter-Individual Differences

In **Figures 3**, **4**, the individual race velocity profiles during the final 1500 m races are plotted. Based on visual inspection, it can be seen that the inter-individual differences were large in the group of runners with intellectual impairment compared to the runners without intellectual impairment. The inter-individual differences were also more pronounced for runners with intellectual impairment. CV was calculated to express the intra-individual differences in velocity over the race. However, during the World Championship final male 1500 m runners without intellectual impairment demonstrated a CV of 7.3 ± 0.5%, and runners with intellectual impairment had an average CV of 5.5 ± 3.1%. Female world championships finalists had a similar CV (6.5 ± 2.7% for runners with intellectual impairment compared to 5.8 ± 0.5% for runners without).

DISCUSSION

The purpose of this study was to explore the differences in pacing strategy between well-trained middle distance runners with and without intellectual impairment. Clear differences in pacing profiles were observed between runners with and without intellectual impairment. Results indicated that runners with intellectual impairment paced their race differently and with greater variance than runners without intellectual impairment. The differences were observed in 400 and 1500 m races, and for both distances, the differences were most pronounced in the first half of the race. Our findings largely support the hypothesis that having an intellectual impairment impacts on the ability of runners with intellectual impairment to effectively regulate their exercise intensity over the race, supporting the assumption that this ability is at least partly dependent on cognitive skill level. To our knowledge, this was the first study to compare pacing profiles

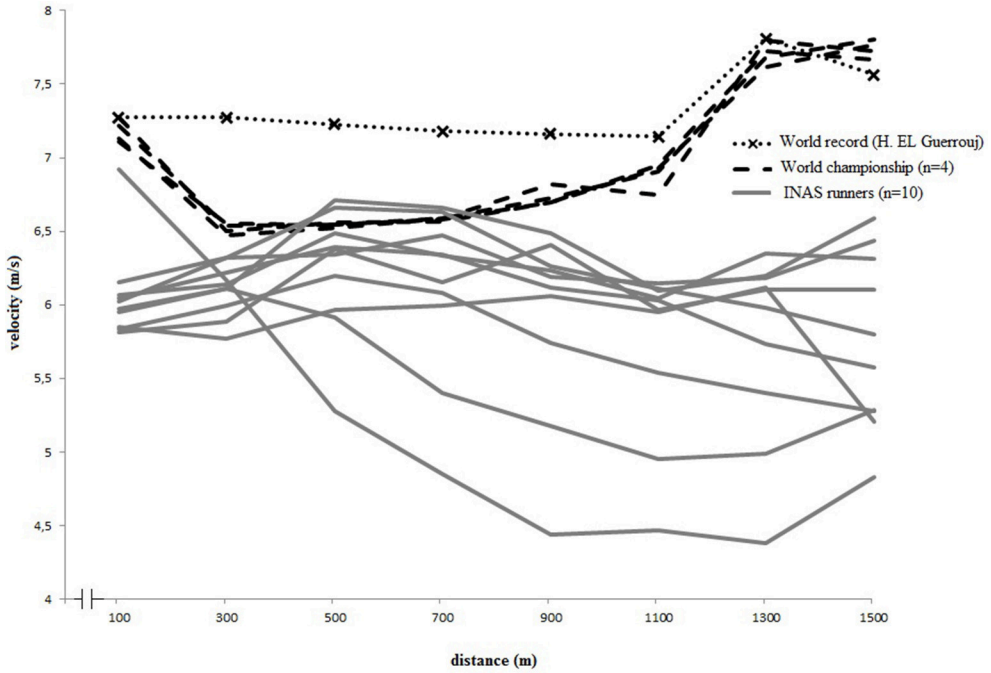

FIGURE 3 | Individual pacing strategies of elite men's 1500 m finalists (II and non-II) vs. World Record. INAS, International Federation for para-athletes with intellectual impairment.

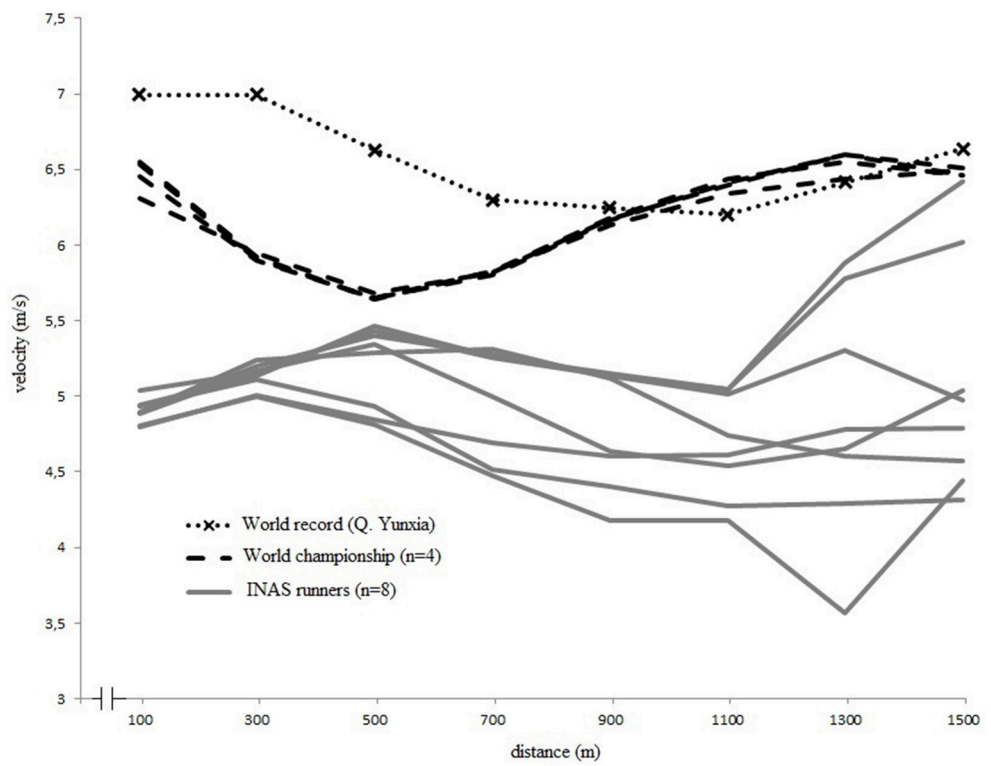

FIGURE 4 | Individual pacing strategies of elite women's 1500 m finalists (II and non-II) vs. World Record. INAS, International Federation for para-athletes with intellectual impairment.

during competitive races of well-trained high level runners with and without intellectual impairment.

Within the literature, pacing has been described as an important cognitive factor in middle-distance and endurance performance that is regulated by the brain (St Clair Gibson et al., 2006; Tucker, 2009) and has been defined as the goal-directed regulation of exercise intensity over an exercise bout, in which athletes need to decide how and when to invest their energy (Smits et al., 2014). The optimal pacing strategies for different running distances were described extensively in the literature (Tucker et al., 2006; Abbiss and Laursen, 2008; Hanon et al., 2008; Thiel et al., 2012; Reardon, 2013; Thompson, 2015). Thompson (2015) described that for the 400-m event, a positive pacing profile is the most optimal strategy; where the speed of the athlete gradually decreases during the race. Other studies also suggested a positive pacing profile as the optimal strategy during a 400-m event (Tucker et al., 2006; Abbiss and Laursen, 2008; Reardon, 2013). Runners are decelerating toward the latter segment of the 400-m race, primarily due to developing fatigue (Thompson, 2015). All world records for 400-m races have been run with a positive pacing strategy (Reardon, 2013), with the results of this study showing that runners with intellectual impairment overall also use a positive pacing strategy over the 400 m running event. Their typical profile of decline of velocity in the two different segments of the second half of the race (slow decline/fast decline) was also be observed in the world record race run by Wayde Van Niekerk in the Olympic Final in Rio 2016 (Vazel, 2016).

Regarding the 1500-m event, an optimal pacing strategy for a 1500 m race is even paced in the middle section; however, overall it is more parabolic according to literature (Hanon et al., 2008; Thompson, 2015). Thomas et al. (2013) showed that though even pacing might theoretically be optimal for endurance performance (De Koning et al., 1999), but in athlete's reality a parabolic shaped pattern might be more appropriate since the cyclists in their study were not able to finish the race when forced into an even paced pattern. In addition, it is important to note that these findings are from cyclists, as differences in optimal pacing might exist between different sports due to their specific characteristics (Stoter et al., 2016). The male world record by El Guerrouj however followed the even paced strategy, rather than the parabolic strategy, with an acceleration at the end (http://www.iaaf.org/); whereas the female world record by Yunxia followed a parabolic pacing strategy (http://wn.com/), at overall higher velocities. In our study, the runners with intellectual impairment adopted different pacing strategies compared to what is considered optimal in literature, or what is logically assumed optimal (i.e., world record performance). The male runners with intellectual impairment were not able to perform an end sprint; which is, probably because they started at very high velocities. Instead of choosing for a controlled, slower pace during the middle part of the race, we assume that the runners might have been physiologically forced to slow downmaking sure not to deplete energy stores prematurely to the races completion (St Clair Gibson et al., 2006). The female runners with intellectual impairment sustained their high start velocity over a long period during the initial segment of the race, before decelerating in the mid-section. They were then able to perform an end sprint at the end of the race; although their average speed overall was lower compared to runners without intellectual impairment.

With respect to the individual patterns of runners with intellectual impairment, high inter-individual variation during the race was observed, with different competitors within the same race applying different race strategies. Runners with intellectual impairment also showed more variance in velocity fluctuations during the race compared to the runners without intellectual impairment. The more consistent strategy applied by runners without intellectual impairment corresponded with Foster et al. (2014) who found a CV of 1.5–3.0% in 1-mile world record performances. In another study by Thiel et al. (2012) the CV during Olympic finals ranged between 3.6 and 11.4%; and, in the finals of the long distance races, the pace varied every 100 m between 1.6 and 2.7% (Thiel et al., 2012). In our study, the variation in running speed is large in runners with intellectual impairment, especially when comparing it to the world records. Using field data, the present study demonstrated that runners with intellectual impairment race with a larger intra-individual variability. Speed fluctuations result in relatively larger air frictional losses (Van Ingen Schenau et al., 1992); leading to a decrease in running economy and a subsequent decrease in performance (Foster et al., 2014). Large velocity fluctuations of competitors during the races can be related to their inability to control their own pace and to maintain a preplanned velocity, as we have demonstrated in a previous study (Van Biesen et al., 2016a). It can also be the result of athletes running a very tactical race (Reardon, 2013), athletes trying to separate themselves from the rest of the athletes when running in a pack (Foster et al., 2014), or due to specific uncommon events (e.g., the fall of one or more competitors). The inter-individual variability observed in runners with intellectual impairment corresponds with findings in many other studies (not only in running) involving participants with intellectual impairment. It was previously observed that intellectual impairment is related to larger inter-individual variation in reaction times (Carmeli et al., 2008), physical fitness (Graham and Reid, 2000; Lahtinen et al., 2007), and performance on sport-specific tasks such as table tennis technical proficiency (Van Biesen et al., 2012).

Comparing to what is known from literature and assuming that the world record pacing patterns are close to optimal, the results of this study indicated that runners with intellectual impairment adopt non-optimal pacing patterns during their races. This finding could be explained by numerous external factors which have an influence on the "optimal" distribution of work, such as other competitors (Konings et al., 2016a,b). Konings et al. (2016b) were the first to show that not only the presence, but also the behavior of an opponent might affect decisions regarding the regulation of exercise intensity in laboratory-controlled conditions. As one crucial element in the diagnosis of intellectual disability is a deficit in adaptive behavior (American Assocation on Intellectual Developmental Disabilities, 2011), the behavior of opponents during races for runners with intellectual impairment can be even more

unpredictable compared to typical high level races. Also, less accomplished runners can feel forced to stay with the leading group at a pace markedly faster than their best performance. This increases the risk of premature excessive fatigue that could result in a decisive drop out later in the race (Thompson, 2015). An example of this was observed in the 1500 m final race for male runners with intellectual impairment, in which one runner started the race at a very high velocity, but he was not able to maintain this velocity and ended up finishing last (see **Figure 3**). This behavior is in line with our preceding study, in which athletes with an intellectual impairment in general were not able to maintain a pre-set sub-maximal velocity (Van Biesen et al., 2016a), but accelerated toward the finish line. It is possible that the behavior of this runner has influenced the profiles of the other finalists, who might have adapted their own pacing to this occurrence, as has been demonstrated to occur in well-trained cyclists (Konings et al., 2016a). In sports where athletes compete in heats, in direct competition with their opponents, this is known to influence their pacing as for example has been demonstrated in 500, 1000, and 1500 m short-track skating competitions (Konings et al., 2016b; Noorbergen et al., 2016). Not much is known yet on how intellectual impaired athletes respond to their opponents, but as athlete- environment interactions are crucial in pacing (Smits et al., 2014) we expect this is an important aspect and future research is needed. Motivational factors are also known to affect optimal pacing (Mauger, 2014). It is known that the increases in motivation and prior experience will reduce the subjective experience of exercise-induced pain during the race and/or increase the willingness of the runner to endure it (Mauger, 2014). Reduced levels of intrinsic motivation are often addressed in research involving participants with intellectual impairment (Hutzler and Korsensky, 2010), however the sample of participants in this specific project involved elite athletes and they were observed during competition at the European Championships, which is a context in which we can assume they perform maximally. Perhaps a more applicable explanation could be that cognitive control and adequate focus of attention are important metacognitive skills to successful pacing (Brick et al., 2016). These metacognitive skills, and most specifically the proactive cognitive control (i.e., anticipatory, goal-oriented processing of information or planning) place a great demand on cognitive resources (Braver, 2012) and these higher order cognitive skills were previously demonstrated to be reduced in elite athletes with intellectual impairment (Van Biesen et al., 2016b), who already have, by the nature of their impairment, limited cognitive resources (Van Biesen et al., unpublished manuscript). People with intellectual impairment are also known to have deficits in a range of other complex higher-order skills that are relevant to pacing (e.g., problem-solving, logical reasoning, and language-dependent strategies such as self-talk; Aitchison et al., 2013).

An interesting finding of the present study is that differences in pacing profiles during the 400 m races were rather small between both group of runners, particularly when compared to differences in the 1500 m. An explanation may be that runners with intellectual impairment, despite their lower levels of cognitive function (i.e., lower IQ), do have the relevant skills to adequately perform a 400 m race, in which an all-out approach is required. These findings correspond with the recent findings by Van Biesen et al. (2016a) that runners with intellectual impairment seem to have difficulties to self-regulate their pace when they are asked to maintain a submaximal velocity, which is required for a 1500 m. They had the tendency to accelerate, and found it difficult to control their velocity. The overall IQ scores of 400 m runners (64.72 ± 8.71) where somewhat higher than for 1500 m runners (59.94 ± 8.12) but this difference was not statistically significant ($p = 0.09$).

Overall, velocity of the runners with intellectual impairment is significantly lower compared to the runners without intellectual impairment, even though both groups consisted of elite athletes. The race observations of the runners with intellectual impairment took place at a European Championship, whereas the split times of the runners without intellectual impairment were obtained from a World Championship. The level at a World Championship is higher than that on a European Championship; however, the large difference in velocity between the two groups is probably not caused by the effect of the cognitive impairment on pacing only. Other aspects may also contribute, for instance the smaller population (i.e., easier to become a top II-runner), reduced maximal voluntary muscle contraction (Borji et al., 2014), the lack of motivation to perform maximally (Rimmer, 1994), reduced leg strength (Fernhall and Pitetti, 2001) or chronotropic incompetence (Dipla et al., 2013). However, the most important aspect to consider is the training volume. The comparison sample in this study was selected on the basis of comparable competition level (the highest obtainable). Training volume data were not available but we can assume that it is higher than the 10 h per week reported by the runners with intellectual impairment. Overall, the level of professionalism in sport for elite athletes with intellectual impairment compared to regular elite sport is not equal. Differences exist in training quality, access to top-coaches, prize money and sponsorship among other factors (Van Biesen et al., 2012).

Some other limitations of this study should also be noted. Comparison data was available for male 400 m runners only, not for female 400 m runners, and the sample size in the 1500 m races was small. In the comparison of data, we were unable to adjust for all potential confounders that may affect pacing and velocity, such as age and training history. These limitations, however, do not alter the importance of our findings, as this study was the first to show a clear difference in pacing strategy during high level running competition between athletes with and without II, in particular in the longer distances, in which pacing and self-regulation becomes more crucial. These findings have contributed to the development of sport specific classification systems and hence created opportunities for athletes with intellectual impairment the world over to participate at the highest level of competition, i.e., The Paralympic Games (Kwon and Block, 2012).

In conclusion, elite runners with intellectual impairments run at an overall slower velocity and following a significantly different pacing pattern compared to runners without an intellectual

impairment. For the 400 m race, the initial acceleration and the final deceleration observed in World record and World Championships races (runners without intellectual impairment) are less pronounced in the finals of high level competitions for runners with intellectual impairment. During the 1500 m race, both group of runners exhibit a seemingly inverse pacing profile. Large inter and intra-individual variations and fluctuations in velocity have been observed in runners with intellectual impairment. Our findings support the assumption that runners with impaired cognitive abilities are less able to regulate their exercise intensity over the race than typical runners, even if they are equally well-trained.

AUTHOR CONTRIBUTIONS

DV: Conceptualizing and drafting the article, revising it critically for important intellectual content, final approval of the version to be published, and accountability for all aspects of the work. FH, KM, and YV: Conceptualizing and revising the study critically for important intellectual content, final approval of the version to be published, and accountability for all aspects of the work.

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Thinking and Action: A Cognitive Perspective on Self-Regulation during Endurance Performance

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Self-regulation reflects an individual's efforts to bring behavior and thinking into line with often consciously desired goals. During endurance activity, self-regulation requires an athlete to balance their speed or power output appropriately to achieve an optimal level of performance. Considering that both behavior and thinking are core elements of self-regulation, this article provides a cognitive perspective on the processes required for effective pace-regulation during endurance performance. We also integrate this viewpoint with physiological and performance outcomes during activity. As such, evidence is presented to suggest that what an athlete thinks about has an important influence on effort perceptions, physiological outcomes, and, consequently, endurance performance. This article also provides an account of how an athlete might control their cognition and focus attention during an endurance event. We propose that effective cognitive control during performance requires both proactive, goal-driven processes and reactive, stimulus-driven processes. In addition, the role of metacognition—or thinking about thinking—in pace-regulation will also be considered. Metacognition is an essential component of self-regulation and its primary functions are to monitor and control the thoughts and actions required for task completion. To illustrate these processes in action, a metacognitive framework of attentional focus and cognitive control is applied to an endurance performance setting: specifically, Bradley Wiggins' successful 2015 *Hour* record attempt in cycling. Finally, future perspectives will consider the potentially deleterious effects of the sustained cognitive effort required during prolonged and strenuous endurance tasks.

Keywords: self-regulation, pacing, endurance performance, attentional focus, cognitive control, metacognition, cycling

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INTRODUCTION: SELF-REGULATION AND ENDURANCE PERFORMANCE

Self-regulation has been described as change to bring thinking and behavior into accord with often consciously desired standards or goals (Forgas et al., 2009). Applied to athletic endeavor, endurance athletes must regulate speed or power output in an attempt to achieve an optimal level of performance (Foster et al., 1994; de Koning et al., 2011; de Morree and Marcora, 2013). Successful performance regularly depends on the selection of an appropriate pacing strategy, avoiding a slower-than-optimal pace and underperformance, or an over-exuberant pace during the initial

stages of activity and, subsequently, premature fatigue (e.g., Abbiss and Laursen, 2008; Renfree and St Clair Gibson, 2013; Hanley, 2016). Perhaps nowhere is pacing as quintessentially *self*-regulated as during an individual time-trial, where pacing strategy is minimally influenced by other athletes or competitors, for example (e.g., Williams et al., 2015; Konings et al., 2016). One notable example is the *Hour* in cycling, an event where the performer attempts to cycle as far as possible within the allotted time. Completing the *Hour* successfully requires a consideration of numerous performance factors, including physical, nutritional, biomechanical, environmental, technological, and psychological variables (Zabala and Hopker, 2015). In 2015, Bradley Wiggins established a new 'official' world *Hour* record, achieving a distance of 54.526 km. Emphasizing the regulatory balancing-act required to optimally pace the *Hour*, the "non-official" world record holder, Chris Boardman (who completed a distance of 56.375 km in 1996 prior to rule changes governing the use of technology; see Zabala and Hopker, 2015), has suggested that, "In the *Hour*, you carry any mistakes with you until the end, so pacing is everything" (Wiggins, 2015, p. 13).

Much debate surrounds the processes underpinning pace-regulation during endurance activity (Abbiss et al., 2015; Renfree et al., 2015). Important recent considerations include affective state (e.g., Renfree et al., 2012; Jones et al., 2015; Rhoden et al., 2015), decision making processes (Renfree et al., 2014; Smits et al., 2014), and risk perception (Micklewright et al., 2015). However, perceived exertion has repeatedly been suggested as a key modulator of exercise intensity (e.g., de Koning et al., 2011; Eston, 2012; Smits et al., 2014) and is central to prominent models of self-paced endurance performance such as the psychobiological model (e.g., Marcora, 2010; Pageaux, 2014), and the perception-based model (Tucker, 2009). Perceived exertion has been defined as a subjective feeling of how hard or strenuous a physical task is (Borg, 1998). Despite conceptual differences on the neurophysiologic basis of effort perception and control of pacing (i.e., conscious or non-conscious; see St Clair Gibson et al., 2006; Tucker, 2009; Marcora, 2010), there is general consensus that any factor which influences perception of effort will indirectly alter pace-regulation (e.g., Marcora, 2010; Noakes, 2012). Much evidence supports this contention during endurance performance. For example, manipulation of physiological (e.g., Tucker and Noakes, 2009), pharmacological (e.g., Doherty and Smith, 2005), and environmental (e.g., competitor presence; Corbett et al., 2012; Williams et al., 2015) variables have each been shown to impact self-paced endurance performance via a dissociation of the effort perception—exercise intensity relationship.

Recent reviews of both attentional focus (Brick et al., 2014) and psychological determinants of whole-body endurance performance (McCormick et al., 2015) have also highlighted how each of these factors impact on effort perception and pace-regulation. Given that self-regulation requires both behavior (e.g., pacing) and thinking (e.g., attention) be in-line with sought after goals (Forgas et al., 2009), an increased understanding of the cognitive processes involved is important to illuminate a discussion on the regulation of endurance performance. The aim of this article, therefore, is to present, and integrate, a cognitive perspective on pace-regulation with effort perception, physiological, and performance outcomes during endurance activity. In terms of cognitive processes, the emphasis will be on attentional strategies that have been shown to impact each of these variables. This article will also consider the role of metacognition in self-regulated endurance performance.

THINKING AND PACING: ATTENTIONAL FOCUS AND COGNITIVE CONTROL

An endurance athlete's focus of attention can have a significant effect on effort perception, pace-regulation, and physiological indices of performance (Brick et al., 2014). Focusing on self-regulatory cognitions such as technique or cadence/rhythm, for example, has been shown to optimize pacing without necessarily increasing the effort perceived during endurance running (e.g., Donohue et al., 2001), race-walking (e.g., Clingman and Hilliard, 1990), rowing (e.g., Connolly and Janelle, 2003), and swimming (e.g., Couture et al., 1999) tasks. Similarly, focusing on relaxing results in an improved movement economy (i.e., reduced oxygen cost) during endurance activity (e.g., Caird et al., 1999). Not all attentional foci are beneficial to performance, however. Focusing excessively on internal bodily sensations or automated processes may exacerbate effort perceptions and negatively impact pacing (e.g., Harte and Eifert, 1995; Stanley et al., 2007) or movement economy (e.g., Schücker et al., 2014), for example. Furthermore, though distractive strategies tend to reduce effort perceptions (e.g., focusing on one's environment; Stanley et al., 2007) this may be at the expense of a slower-than-optimum pace during self-paced endurance activity (e.g., Scott et al., 1999; Connolly and Janelle, 2003).

What these studies highlight is the interaction between endurance athletes' cognitions and subsequent effort perception, physiological, and performance outcomes. Recent evidence also suggests that the most appropriate attentional strategies during performance may depend on the demands of the situation (Brick et al., 2015). For instance, during a self-paced time-trial this may be to cope with distractions, or to overcome debilitating perceptions of effort while attempting to optimize performance. As such, adopting a context-appropriate focus of attention requires both a domain-specific knowledge of cognitive strategies (e.g., MacIntyre et al., 2014) and cognitive control, or the ability to regulate thoughts and actions in accord with behavioral goals (e.g., Robertson et al., 2015; Ličen et al., 2016). According to the dual mechanisms of control framework (Braver et al., 2007), cognitive control operates via two distinct modes: proactive control and reactive control (Braver et al., 2007; Braver, 2012). Proactive control involves anticipatory, goal-oriented processing of information so that attention (e.g., focus), perception (e.g., of effort), and action (e.g., pacing) are biased in a goal-driven manner (Miller and Cohen, 2001; Braver, 2012). In contrast, reactive, or stimulus-driven cognitive control (Miller and Cohen, 2001; Corbetta and Shulman, 2002; Braver, 2012) is more automatic and transient, and reacts to urgent events or conflict by engaging control only if required (Braver et al.,

2007). Accordingly, reactive cognitive control is implicated in default mode processing and is less demanding on cognitive resources (e.g., working memory), whereas proactive control is engaged in more effortful situations and places a greater demand on cognitive resources (Braver, 2012; Braver et al., 2007).

Brick et al. (2015, 2016) recently proposed that both context-dependent proactive and reactive cognitive control are initiated during endurance activity. In a study involving 3 km time-trial running, the findings on the attentional focus of participants during a self-controlled pace trial (i.e., focus on pacing, monitoring distance information, and "chunking," or mentally breaking the distance down to smaller segments) suggested both proactive and reactive forms of control were important to pace-regulation. However, when an equivalent pacing strategy was externally-controlled by the experimenter (akin to pace-making), the most frequently reported attentional foci (i.e., relaxing, optimizing running action) suggested reactive control was the predominant form of cognitive control. Heart rate was also 2% lower in the externally-controlled condition when compared with the self-controlled pace trial, possibly as a result of the cognitive strategies engaged (Brick et al., 2016). Applying these findings to endurance performance, we propose that effective pace-regulation requires the athlete to adopt a situationally-appropriate focus of attention and mode of cognitive control. During an event such as the *Hour*, for example, the athlete receives minimal and infrequent external feedback on pacing. Accordingly, perceptions of effort may serve a vital role in pace-regulation, particularly in the early stages of the event. During the latter stages, however, when the athlete begins to fatigue, cognitive strategies become more important to overcome an ever-increasing sense of effort and maintain a target pace. In support of this contention, Chris Boardman has suggested that pacing in the *Hour* is an equation with three inter-related questions: how long to go, how hard the athlete is trying, and whether that effort sustainable? He suggests the "unnerving" answer to the latter question is "maybe" (Wiggins, 2015, p. 13). Accordingly, to achieve a desired standard the athlete must proactively adopt a focus of attention to cope with task demands in a goal-driven manner. However, when faced with an unexpected event (e.g., getting distracted, errors in pacing strategy) the endurance athlete must also reactively adapt cognition when required to optimize performance or maintain positive affect, for example (e.g., Carver and Scheier, 1998; Rhoden et al., 2015).

To conclude so far, we have presented evidence to suggest what an athlete thinks about influences effort perceptions, physiological outcomes and, consequently, endurance performance. In turn, these effects of various cognitive strategies may explain when and why an athlete will engage a particular focus. Additionally, cognitive control, or the ability to regulate thoughts and actions (e.g., Braver et al., 2007) provides an insight into an athlete's ability to align thinking with performance tasks and goals. A final consideration, however, is how an athlete controls cognition and focuses attention during endurance performance. In the following section we apply Brick et al.'s (2015) metacognitive framework of attentional focus and cognitive control to self-regulation during endurance performance.

THINKING ABOUT THINKING: METACOGNITION AND ENDURANCE PERFORMANCE

Metacognition has been defined as an individual's knowledge and cognitions about cognitive phenomena (Flavell, 1979) or, more simply, as "thinking about thinking" (Miller et al., 1970, p. 613). Metacognition can also reflect an individual's understanding of what they know and how to use that knowledge to regulate behavior (Bransford et al., 1999; Tomporowski et al., 2015). Metacognition not only consists of conscious goals, but also the activation of strategies (i.e., thoughts, behaviors) to achieve those goals (Flavell, 1979). It is also important to note that although self-regulation and metacognition have distinct origins in psychology, metacognition is considered an essential component of effective self-regulation (Dinsmore et al., 2008; Efklides, 2008; Tarricone, 2011). Accordingly, Dinsmore et al. (2008) highlight a "conceptual core" (p. 404) binding self-regulation and metacognition that involves efforts to monitor thoughts and actions, and activity to gain control over them. As such, this section will attempt to shed further light on how endurance athletes monitor and control the thoughts and actions required for effective pace-regulation.

Brick et al. (2015) recently proposed a metacognitive framework of attentional focus and cognitive control during endurance performance. Based on the facets of metacognition (e.g., Efklides, 2008), this model comprises two distinct processes: *metacognitive skills* and *metacognitive experiences*. Metacognitive skills include *planning* prior to performance (e.g., of cognitive strategies), *monitoring* during performance (e.g., of thinking and task completion), and *reviewing and evaluating* after performance (e.g., of cognitive strategies and task performance). Metacognitive experiences, in turn, are based predominantly on monitoring processes and include both implicit and explicit *metacognitive feelings* (e.g., feeling of difficulty) and explicit *metacognitive judgments and estimates* (e.g., judging whether a cognitive strategy is effective for its intended purpose). Relevant to this perspective, Efklides (2008), for example, suggests that metacognitive experiences such as feelings of task difficulty are crucial for the self-regulation of effort.

The most relevant metacognitive skills to the present discussion are planning and monitoring processes. Metacognitive planning may incorporate proactive goal setting, establishing a desired pacing strategy, or the selection of other cognitive strategies to implement during performance (Brick et al., 2015). Metacognitive planning may be particularly important when an athlete wishes to minimalize interference from potential distractors (Miller and Cohen, 2001; Braver et al., 2007). In contrast, metacognitive monitoring predominantly involves reactive or stimulus-driven cognitive control during task performance (Corbetta and Shulman, 2002; Braver et al.,

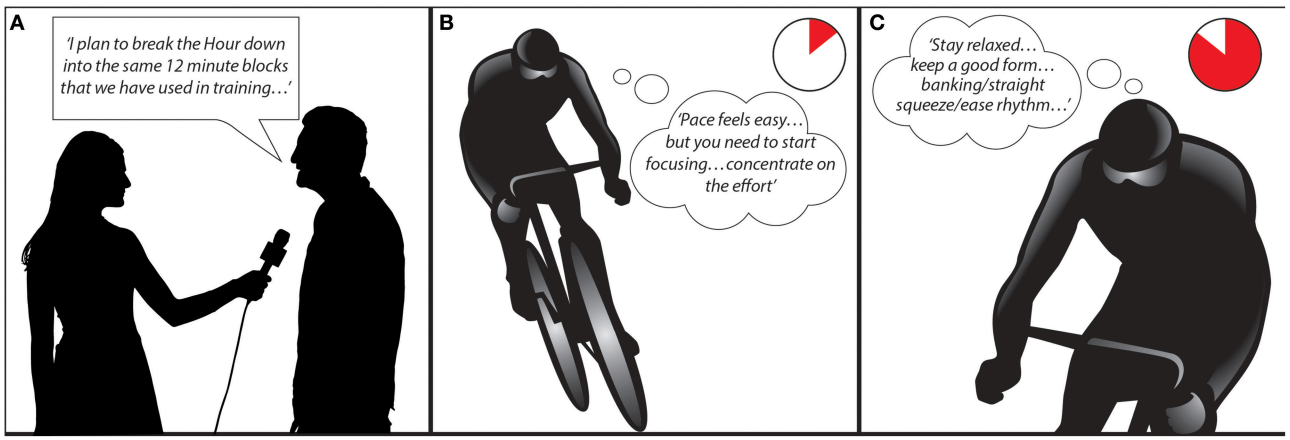

FIGURE 1 | Illustrative examples adapted from Wiggins (2015) of metacognitive planning of a cognitive strategy before performance (A), metacognitive monitoring and reactive cognitive control during performance (B), and proactive cognitive control during performance (C).

2007). Brick et al. (2015), for example, demonstrated how elite endurance runners had, through experience, developed a means of prioritizing sensory information to optimize endurance performance. Accordingly, periodic monitoring of internal sensory (e.g., perceived exertion) and/or relevant outward environmental (e.g., split times, competitors) sources of information generate implicit or explicit metacognitive feelings that form a representation of the task. Thus, while monitoring and control can occur at an implicit, non-conscious level, conscious control is engaged when metacognitive feelings (e.g., feeling of difficulty) form a representation and awareness of the task (e.g., pace is too hard) that requires an appropriate response (see Efklides, 2008). This response may be to reactively engage a cognitive strategy to cope with situational demands (e.g., focus on task-relevant stimuli) or to adopt a more appropriate pacing strategy, for example. Once initiated, the athlete may make a more explicit metacognitive judgment (e.g., this is working to maintain pace) regarding the appropriateness of their adopted focus of attention (Brick et al., 2015). Based on the outcome of this judgment, the athlete may decide to maintain their current focus, or implement an alternative, more suitable cognitive strategy.

Metacognitive skills (e.g., planning, monitoring) and experiences (e.g., feelings, judgments) may explain how endurance athletes focus attention, control cognition, and, in turn, regulate pacing. Accordingly, we propose that an athlete's efforts to monitor and control their thoughts and actions reflect the conceptual core linking metacognition and self-regulation in an endurance performance context (Dinsmore et al., 2008). To provide greater insight into these cognitive and metacognitive processes in action, the following section will integrate the theoretical constructs of attentional focus, cognitive control, and metacognitive processes with a real-world example of self-regulated pacing during endurance performance (i.e., Bradley Wiggins' successful 2015 *Hour* record attempt).

THINKING AND ACTION: COGNITIVE AND METACOGNITIVE PROCESSES DURING ENDURANCE PERFORMANCE

Many strategic considerations prior to Bradley Wiggins' 2015 *Hour* record attempt reflect metacognitive planning. His target pace (16.1 s per lap) and cadence (105 rpm) were carefully calculated to optimize his capabilities to achieve a pre-event goal distance of 55.2 km (Wiggins, 2015). One pre-planned cognitive strategy was to mentally chunk the 60 min event into blocks of 12 min, a strategy that evolved during training for the *Hour* (i.e. reflecting metacognitive planning; see **Figure 1A**). Although chunking as a strategy has not been investigated experimentally *per se*, reflective accounts (Brick et al., 2015, 2016) suggest that chunking may assist pace-related decision making by allowing the athlete set shorter-term goals within a longer duration endurance event.

It is also likely that the cognitive strategies Wiggins subsequently engaged during the *Hour* evolved from his 23 years' experience as a cyclist and domain-specific expertise as an elite time-trialist (Micklewright et al., 2010; Wiggins, 2012; MacIntyre et al., 2014). In this regard, evidence from his autobiographical account (Wiggins, 2015) suggests Wiggins employed both proactive and reactive cognitive control during the *Hour*. For example, during the initial stages when the pace felt easier (based on a metacognitive feeling of difficulty), he recounts self-instructions to start focusing, listening to his body, and to concentrate on the effort (i.e., reactive cognitive control; see **Figure 1B**). During the latter stages, however, Wiggins initiated three attentional strategies to maintain pacing and performance in a goal-driven manner (i.e., proactive cognitive control; see **Figure 1C**). These strategies were relaxation, focusing on form (technique), and synchronizing his pedaling rhythm with the track's banking and straight sections (Wiggins, 2015). Focusing on these active self-regulatory strategies has been shown to improve movement economy (e.g., relaxation; Caird et al., 1999),

and optimize pacing without elevating effort perceptions further (e.g., technique and rhythm/cadence; Clingman and Hilliard, 1990; Connolly and Janelle, 2003). It is also noteworthy that when unexpectedly high atmospheric pressure meant his goal pace and distance may not have been attainable on the day; Wiggins recalculated his target *Hour* record pace (to 16.4 s per lap), thereby maintaining goal commitment and a positive affective state (Rhoden et al., 2015; Wiggins, 2015).

This illustrative example supports the notion that efforts to monitor and control thoughts and action link self-regulation and metacognition (Dinsmore et al., 2008; Tarricone, 2011). Furthermore, it reinforces the relationships between attentional focus, and physiologic and performance outcomes during a mentally and physically strenuous task such as an individual time-trial. As such, we suggest that further elucidation of our understanding of pace-regulation during endurance tasks will only be possible with continued integration of these scientific branches of endurance research.

FUTURE PERSPECTIVES

The present article has highlighted the roles of attentional focus, cognitive control, and metacognition in self-regulated endurance performance. One issue worthy of further consideration concerns suggestions that inducing mental fatigue prior to activity may subsequently elevate effort perceptions and diminish endurance task performance (e.g., Marcora et al., 2009; MacMahon et al., 2014; Pageaux et al., 2015). Indeed, Marcora et al. (2009) suggest that both mentally and physically demanding tasks share the same neurocognitive resources. As such, mental fatigue may exert an influence on endurance performance by altering perceptions of effort independent of changes in cardiorespiratory or musculoenergetic mechanisms (Marcora et al., 2009). Despite these findings, no published study has specifically focused on the effects of mental fatigue accrued during sustained endurance performance. However, researchers have recently begun to speculate that prolonged endurance activity in itself may induce mental fatigue (Renfree et al., 2015; Brick et al., 2016) and reduce regulatory control (e.g., Rhoden et al., 2015). More so, while this perspective article has primarily considered pace-regulation in the context of individual time-trialing, competitive endurance events also require strategic decision-making during performance based on additional environmental factors, including competitor behavior, for example (e.g., Smits et al., 2014; Hanley, 2015; Konings et al., 2016). Given the importance of cognitive functioning to sustained endurance activity (e.g., Cona et al., 2015), deteriorations in performance during the latter stages of demanding endurance tasks may be in part attributable to increased mental fatigue and a reduced ability to maintain self-regulatory control. Further, investigation of these issues may provide a fruitful line of enquiry. It may be that additional performance gains are possible by reducing the cognitive demands associated with prolonged endurance activity. This may be achieved by adopting an appropriate focus of attention (e.g., relaxing), for example, or by utilizing pace-makers to reduce pace-related decision making during prolonged endurance events.

AUTHOR CONTRIBUTIONS

NB: Conceptualizing and drafting the article, revising it critically for important intellectual content, final approval of the version to be published, and accountability for all aspects of the work. TM: Conceptualizing and revising the study critically for important intellectual content, final approval of the version to be published, and accountability for all aspects of the work. MC: Conceptualizing and revising the study critically for important intellectual content, final approval of the version to be published, and accountability for all aspects of the work.

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Cognitive Fatigue Influences Time-On-Task during Bodyweight Resistance Training Exercise

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Prior investigations have shown measurable performance impairments on continuous physical performance tasks when preceded by a cognitively fatiguing task. However, the effect of cognitive fatigue on bodyweight resistance training exercise task performance is unknown. In the current investigation 18 amateur athletes completed a full body exercise task preceded by either a cognitive fatiguing or control intervention. In a randomized repeated measure design, each participant completed the same exercise task preceded by a 52 min cognitively fatiguing intervention (vigilance) or control intervention (video). Data collection sessions were separated by 1 week. Participants rated the fatigue intervention with a significantly higher workload compared to the control intervention ($p < 0.001$). Additionally, participants self-reported significantly greater energetic arousal for cognitively fatiguing task ($p = 0.02$). Cognitive fatigue did not significantly impact number of repetitions completed during the exercise task ($p = 0.77$); however, when cognitively fatigued, participants had decreased percent time-on-task (57%) relative to the no fatigue condition (60%; $p = 0.04$). RPE significantly changed over time ($p < 0.001$), but failed to show significant differences between the cognitive fatigue intervention and control intervention ($p > 0.05$). There was no statistical difference for heart rate or metabolic expenditure as a function of fatigue intervention during exercise. Cognitively fatigued athletes have decreased time-on-task in bodyweight resistance training exercise tasks.

Keywords: cognitive fatigue, bodyweight resistance training exercise, mental workload

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INTRODUCTION

Similar to athletic performance, Soldier safety, and mission success are contingent on both physical and cognitive performance. Indeed, cognitive performance (e.g., decision making, psychomotor performance, response inhibition, and vigilance) plays a key role in overall Soldier performance (Friedl et al., 2007; Wilson et al., 2013). Moreover, failures in Soldiers' cognitive ability could result in increased likelihood of human error resulting in friendly-fire incidents and collateral damage (Belenky et al., 1994; Wilson et al., 2013). Though technology may enhance Soldier performance, it may also inundate Soldiers with increased sources of information to process.

Abbreviations: ANOVA, Analysis of Variance; DSSQ, Dundee Stress State Questionnaire; EA, Energetic Arousal; NASA-TLX, National Aeronautical and Space Administration Task Load Index; RPE, Rating of Perceived Exertion; TA, Task Arousal; TRT, Task-Related Thoughts; TUT, Task-Unrelated Thoughts; VO_2, Oxygen consumption.

Unfortunately, more information for the Soldier to process may result in greater cognitive fatigue for Soldiers who have to process and relay information fluidly on the battlefield. For example, the U.S. Army is actively pursuing increasingly sophisticated sensor devices that provide situational awareness and threat detection to the Soldier on the battlefield (e.g., DARPA Squad-X project). Though this information is useful to the Soldier, it may present challenges such as mental resources depletion or bottlenecking of information processing causing cognitive fatigue (Kahneman and Tversky, 1973; Navon and Miller, 2002).

Exercise can undoubtedly elicit improvements in mental health and overall health (Biddle et al., 2000; Biddle and Mutrie, 2007; Hamer and Chida, 2008). Moreover, there is also evidence that regular exercise may act as a cognitive enhancer (Brisswalter et al., 2002; Ratey and Loehr, 2011; Chang et al., 2012) which could, for example, improve scholastic achievement (Keeley and Fox, 2009). Conversely, prior studies have provided evidence that physically demanding tasks completed prior to and during cognitive tasks result in cognitive impairment (Isaacs and Pohlman, 1991; Cian et al., 2000; Tomporowski, 2003). Moreover, when a cognitively demanding task is coupled with a physical task (dual-task), performance impairments can be observed on both the physical and cognitive components of the task (i.e., performance trade-offs; Green and Helton, 2011; Head et al., 2012). Green and Helton (2011); Green et al. (2014) suggest that these performance impairments may be the result of limited cognitive resources available to process the cognitive and physical task simultaneously. Interestingly, only recently have researchers begun to examine the effects of cognitive fatigue on subsequent performance in a physical task (Marcora et al., 2009; Pageaux et al., 2014).

Cognitive fatigue is the psychophysiological response generated by prolonged exposure to a cognitively demanding task which results in the subjective feeling of "tiredness" and "lack of energy" (Marcora et al., 2009; Pageaux et al., 2014). Cognitive fatigue has a measurable influence on physical performance and may provide a greater understanding of the causal factors affecting physical performance beyond neuromuscular fatigue (Smith et al., 2015). Marcora et al. (2009) examined the effects of participants performing a cognitively fatiguing task prior to completing a cycling task to exhaustion which resulted in participants reaching physical exhaustion sooner compared to the control condition. Pageaux et al. (2014) demonstrated that a motor response inhibition task [Stroop task; (Stroop, 1935)] impaired subsequent physical performance during a 5K treadmill time trial. Unlike Marcora and colleagues' task, participants were not pushed to exhaustion, but rather completed the treadmill task with a 6% slower completion time when preceded by the inhibition task. Cognitive fatigue has also been shown to impair physical performance in intermittent running (e.g., Yo-Yo Intermittent Recovery Test) and technical abilities such as passing and shooting in soccer (Smith et al., 2015, 2016). Furthermore, performance on continuous or prolonged intermittent exercise tasks is significantly impaired while high-intensity peak velocities were not significantly reduced (Smith et al., 2015). The amount of exercise performance impairment (2.3–17.8%; Marcora et al., 2009; Smith et al., 2015) is seemingly dependent on whether the task has a set end point (2.3–5.3%; Pageaux et al., 2014; Smith et al., 2015) or time to exhaustion/failure (17.2–17.8%; Marcora et al., 2009; Pageaux et al., 2014). However, the effects of cognitive fatigue on physical performance does not appear to be universally applicable. For example, a prior investigation found no differences in peak, critical, and estimated anaerobic work capacity on task performance as a function of cognitive fatigue in a countermovement jump task and 3 min all-out cycle test (Martin et al., 2015).

Collectively, there is mixed support for the influence of prior cognitive fatigue on subsequent physical performance. This may be partially due to the length of exercise time and also the modality (i.e., isometric vs. full body), but it is unknown if the existing paradigms can be generalized to the multitude of athletic and occupational endeavors that are high-intensity and require strength. For example, military occupations are often high-intensity but discontinuous by nature [e.g., negotiating obstacles, lifting, and climbing (Ortega et al., 1993)]. Furthermore, it is unclear whether cognitive fatigue influences perceived effort or task performance on a high intensity bodyweight resistance training exercise (strength-endurance) paradigm. Indeed, prior evidence has supported that disruptions in cognitive performance could be a function of level of intensity (e.g., $x \geq 70\%$ VO_2 max; Reilly and Smith (1986)). In other words, greater impairment may be observed by increasing the intensity of the exercise task. Thus, the present study aims to investigate the influence of cognitive fatigue on subsequent high intensity body weight resistance exercise that is self-paced and time limited. In line with previous research on continuous endurance exercise (Marcora et al., 2009; Pageaux et al., 2014; Smith et al., 2015), it is hypothesized that participants will have performance impairments (i.e., decreased repetitions completed) and modified task execution (i.e., decreased time-on-task) during the exercise task when preceded by cognitive fatigue.

MATERIALS AND METHODS

Participants

Eighteen (11 male, female 7) volunteers were recruited from local gyms located in Baltimore, Maryland, USA. Participants age ranged between 24 and 37 years ($M = 28$, $SD = 3.8$). All participants had at least 6 months experience participating in high intensity exercise routines and were free from any known illness or disease. The participants provided written informed consent in accordance with the Helsinki Accord and ethics permission was obtained from the U.S. Army Research Laboratory Institutional Research Board.

Procedure

In a counterbalanced design, all participants completed either a control (passive video watching) or a cognitively fatiguing task (vigilance) prior to performing an exercise task. The two visits took place over a 2 week period (once a week) at the same time of day for each participant in an isolated room. Thus, there were approximately 7 days between sessions. Participants were given instructions to sleep for at least 7 h

and were also instructed to avoid caffeine, nicotine, and other stimulants/depressants for at least 3 h before participating. Participants using stimulants and/or depressants were not permitted to participate in the study (Brownsberger et al., 2013). Upon arrival to the facility, participants completed an informed consent, demographics, and a Dundee Stress State Questionnaire (DSSQ; Matthews et al., 2002) pre-task stress state questionnaire. Within the informed consent, participants were shown a list of commonly used high-intensity interval training exercises tasks which included the exercise task used in the current investigation. Participants were requested to instruct the researchers if they there were not experienced in any of the exercise tasks. Volume of oxygen consumed (VO_2) was used as an indirect measure of energy expenditure. Participants were outfitted with a VO_2 measuring apparatus (COSMED K4B2, COSMED, Italy), heart rate monitor (Polar, USA) and completed a baseline VO_2 measure whereby they sat quietly in an isolated room for 5 min. Upon completion of the baseline VO_2 measure, participants completed the experimental or control intervention (i.e., cognitive fatigue or video watching, respectively). After the experimental or control intervention, the participant completed a National Aeronautical and Space Administration Task Load Index (NASA-TLX; Hart and Staveland, 1988) and DSSQ post-task stress state questionnaire. Prior to beginning the exercise task each time, participants completed a standardized dynamic warmup for 5 min which included familiarization with the exercise task with the VO_2 apparatus. After completing the warmup, participants completed the exercise task for 20 min. Participants were not permitted time keeping devices and were generally unaware of the time elapsed during the 20 min exercise task. During the exercise task, participants rated their RPE every 5 min using a RPE scale (Borg, 1998; Gearhart et al., 2001). Upon completion of the exercise task, participants completed the NASA-TLX and DSSQ post-task stress state questionnaire (see **Figure 1** for timeline of study).

Cognitive Fatigue Intervention

A traditional vigilance task was used to induce cognitive fatigue (Warm et al., 2008). The vigilance task took place in an isolated room. Participants were seated 50 cm in front of a video display terminal (53.4 × 33 cm, 60 Hz refresh rate) which was mounted at eye level. Participants' head movements were not restrained. Time-keeping devices such as watches and cell phones were surrendered at the start of the task. Participants completed a vigilance task (low Go/ high No-Go) monitoring for numeric stimuli (1–9). Participants were instructed to respond to the number 3 and withhold their response to 1–9 except the number 3. Go stimuli were presented 11% of the time. Participants were instructed to respond as fast and accurately as possible to the target number 3. Prior to the start of the task, participants completed a practice trial where they received feedback on their performance. The vigilance task was 52 min in duration and was comprised of 8 periods of watch, 6.5 min in length. The task was continuous with no rest breaks. The inclusion of the task epochs allows the researcher to examine performance impairment (i.e., accuracy and response time) as a function of time-on-task which is a behavioral indicator of cognitive fatigue (Head and Helton, 2012). Numeric digits were all the same font (Courier); however, font size varied between 48, 72, 94, 100, and 120, with height varying between 12 and 29 mm. Varied font size was used to discourage participants from using visual shape cues (Robertson et al., 1997). Each trial consisted of a single digit presented centrally on the screen for 250 ms followed immediately by a 900 ms mask. The mask consisted of a circle (29 mm in diameter) with a diagonal line in the middle spanning from one side to the other. Participants were instructed to respond with their index finger on their dominant hand using a response box.

Control Intervention

The computer screen from the vigilance task was used to present a 52 min video train documentary. The documentary was "The

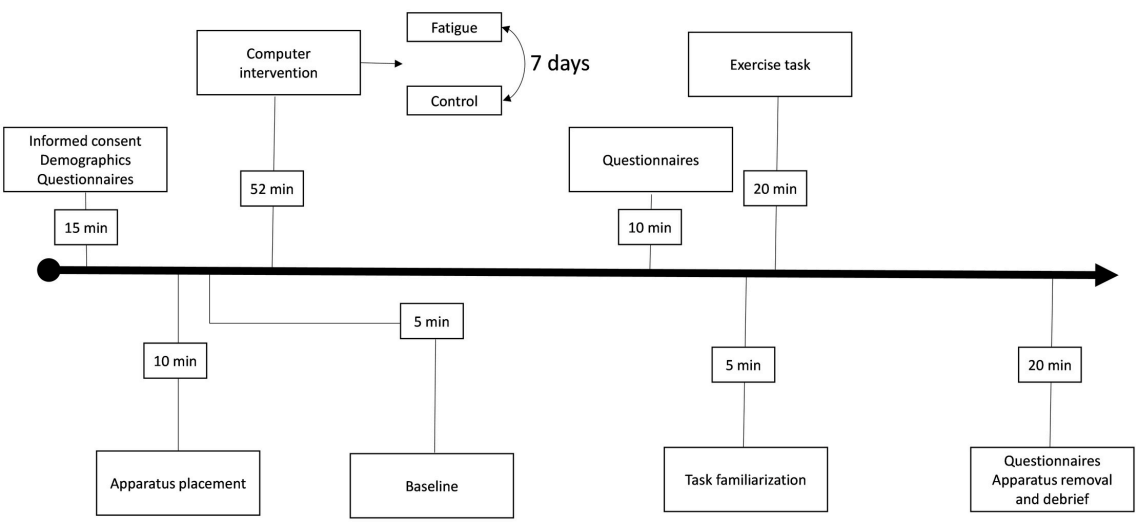

FIGURE 1 | Task timeline. Displayed is a timeline of events with corresponding durations.

American Orient Express" (Pegasus-Eagle Rock Entertainment, 2004) which consisted of footage about trains and travel. This type of stimuli has been used in similar studies due to the neutral content maintaining stable mood and heart rate (Silvestrini and Gendolla, 2007; Marcora et al., 2009; Smith et al., 2015).

Physical Exercise Task Protocol

The physical task used in the present study was a high intensity body resistance exercise routine. It was comprised of three separate exercise sets completed in succession for as many rounds as possible in 20 min. The first exercise was 5 pull-ups, followed by 10 pushups, and finally 15 unweighted squats. Participants were required to finish each set for each exercise prior to beginning a new round. A research investigator verbally counted completed repetitions during the exercise task. Upon the completion of each round (5 pull-ups, 10 pushups, 15 unweighted squats), the next round was immediately started. This specific exercise task was chosen because these bodyweight exercises are commonly configured together in a circuit workout at the local gym facilities where recruitment took place. Thus, participants were more likely to be experienced in the individual exercises and completing them consecutively.

All participants were given instructions regarding proper form for each exercise and inappropriate repetitions were not counted. For the pull ups, participants were instructed to begin by grasping a metal pullup bar overhead with both hands facing palm out with feet not touching the ground. Participants were instructed to pull themselves up until their chin was above the bar and then to lower themselves until their arms were fully extended. For the pushups, participants were instructed to start in a standard pushup position (plank) and then lower themselves until their chest made contact with the ground. Once their chest made contact with the floor, participants were instructed to extend their arms until they were back in the plank position. For the unweighted squats, participants were instructed to lower themselves to 90° of knee flexion (i.e., seated position). Once participants achieved the seated positon, they were instructed to return to a standing position. Participants were notified when they correctly completed each repetition by stating the number of correct repetitions performed. Additionally, participants were notified if they did not complete the exercise task correctly by saying "no repetition." In total, only two participants were given verbal "no repetition" warnings during the pull up portion of the exercise task. Participants were not given verbal encouragement during any task. A high-definition 60 Hz frame rate video camera (Vixia HFR42, Canon, U.S.A., Inc) was placed in a standardized location ∼15 m from the workout area to record the workout completed by the participant. The workout videos were later used for behavioral analysis.

Video Workout Protocol

A researcher, blinded to study hypothesis and subject intervention, viewed each video and quantified the number of repetitions and rounds completed, time-on-task and time off-task. Time-on-task was defined as participants actively performing the exercise task. Conversely, time off-task was defined as participants not actively performing the exercise task (e.g., transition between exercise type and resting). The researcher was given a predefined workout protocol that provided examples of how each exercise was to be performed to be counted as a repetition. To test interrater reliability, an additional blinded research assistant viewed and evaluated a subset (22 videos) of 36 videos. An intraclass correlation of 0.90 (95% confidence interval: 0.78–0.96) revealed acceptable interrater reliability (Hays and Revicki, 2005).

Psychological Scales

Consciousness and Stress State Questionnaire

An abridged version of the 90-item DSSQ (Matthews et al., 2002) was used to measure participants subjective stress state. The abridged DSSQ contains 32-items which generate a 4-factor solution: energetic arousal (EA), task arousal (TA), task-related thoughts (TRT), and task-unrelated thoughts (TUTS). These factors have been used in prior studies to determine whether participants were focused on task related or unrelated thoughts during the task and also arousal level (Head and Helton, 2012, 2013). Items from each factor are aggregated to yield a single score for each factor for pre- and post-task. The abridged DSSQ was given prior to the experimental intervention, post-intervention, and after the exercise task.

Workload Measure Scale

The NASA-TLX is a workload measure composed of 6-items (Hart and Staveland, 1988). The questionnaire contains three items that measure external demand (mental, temporal, and physical) and an additional three items (effort, performance, and frustration) that measure internal responses to the external demands. The 6-items were aggregated together for a composite global workload score. The NASA-TLX was given after each computer task and exercise task.

Rating of Perceived Exertion

The Borg rating of perceived exertion (RPE) scale is a 15-point scale ranging from 6 "No exertion" to 20 "Maximal exertion." The RPE scale is versatile and can be applied to aerobic and anaerobic exercise (Borg, 1998; Gearhart et al., 2001). During the exercise task, participants were requested to indicate their RPE at 5 min intervals by pointing to a numerical value on a standardized 15-point RPE poster board (54 × 50 cm) placed next to the exercise area. Participants were instructed to rate their effort expended on the exercise task. Each participant was provided verbal examples of "No exertion" (e.g., sitting on the couch relaxing) and "Maximal exertion" (e.g., you are giving all your effort).

Statistical Analysis

All data reported is presented as mean ± *SD* unless otherwise stated. A one-way repeated measures ANOVA was used to test whether response time and accuracy errors changed over time in the vigilance task as a manipulation check. Repeated measures *t*-tests were used to test whether the fatigue intervention vs. the control intervention effected percent time-on-task and exercise repetitions completed. Mean heart rate (bpm) and relative VO_2 ($mL \cdot kg^{-1} \cdot min^{-1}$) were calculated for each condition (baseline,

intervention, and exercise task). Heart rate and VO_2 were subjected to separate 2 (intervention type) × 3 (baseline, intervention, and exercise task) repeated measures ANOVAs to examine whether the fatigue intervention had an effect on time periods of measurement. Mean heart rate and VO_2 were calculated per 5 min period for the 20 min exercise tasks preceded by the fatigue and control intervention. Mean heart rate and VO_2 were subjected to two separate repeated measures 2 (intervention type) × 4 (time period) ANOVA to determine whether heart rate and VO_2 changed as a function of intervention type and time period. Similarly, heart rate and VO_2 was calculated per 13 min time period for the 52 min fatigue and control intervention. Mean heart rate and mean VO_2 per time period was subjected to two separate repeated measures 2 (intervention type) × 4 (time period) ANOVA as a manipulation check to determine whether heart rate and VO_2 measures changed as a function of intervention type and time period. RPE was subjected to a repeated measure 2 (fatigue type) × 4 (time block) ANOVA to determine whether the fatigue intervention vs. the control intervention affected self-reported RPE as a function of time (5 min blocks). The subjective post-intervention DSSQ sub-scales for Energetic Arousal, Task Arousal, Task-Related Thoughts, and Task Unrelated Thoughts were calculated for each individual. Due to all measures being on the same response scale (i.e., 1–5) the raw (non-standardized) scores were used as recommended (Rogosa, 1995). These scores were analyzed with 2 (fatigue type) × 3 (time period: baseline, post computer task, and exercise task) × 4 (scale: EA, TA, TRT, TUT) repeated measures ANOVA. To determine perceived workload of each task, a global workload score was calculated for each participant by averaging the 6 subscales of the NASA-TLX for each participant for each task. Global workload scores were subjected to a 2 (intervention type) × 2 (task type: computer vs. exercise task) repeated measures ANOVA. When appropriate, *post-hoc* comparisons were made with the Bonferroni multiple comparison procedure. Effect sizes for repeated measures were calculated as partial eta squared (η^2_p). Effect sizes for paired *t*-test were calculated as Cohen d_z. Significance was set at 0.05 (2-tailed). All analysis were conducted using the Statistical Package for Social Sciences, version 22 (SPSS Inc., Chicago, IL, USA).

RESULTS

Manipulation Check

The results for correct response times to targets showed impaired performance (slowing) across the vigilance task $F_{(7,\ 119)} = 27.54$, $p < 0.001$, $\eta^2_p = 0.62$. However, the result of the accuracy analysis failed to reach significance due a high accuracy rate, $F_{(1,\ 17)} = 0.43$, $p = 0.520$, $\eta^2_p = 0.07$ see **Table 1** for results of each time period. The response time result provided evidence that the vigilance task elicited cognitive fatigue. Prior investigations have examined relative elevated HR as a physiological marker for cognitive fatigue (Marcora et al., 2009). Thus, we examined change over time for heart rate while participants completed the cognitive fatigue and control intervention to verify whether the cognitive fatigue intervention was fatiguing. Additionally, we also analyzed whether metabolic expenditure changed as a function of time and task. For the 52 min control and fatigue intervention, there was a significant main effect of time on heart rate, $F_{(3,\ 51)} = 39.32$, $p < 0.001$, $\eta^2_p = 0.70$; however, there was no main effect for intervention type, $F_{(1,\ 17)} = 1.68$, $p = 0.212$, $\eta^2_p = 0.09$, or time by intervention type interaction, $F_{(3,\ 51)} = 2.17$, $p = 0.103$, $\eta^2_p = 0.11$. There was a main effect for time period for VO_2, $F_{(3,\ 51)} = 8.97$, $p < 0.001$, $\eta^2_p = 0.35$, but no main effect for intervention type, $F_{(1,\ 17)} = 0.23$, $p = 0.636$, $\eta^2_p = 0.01$, or time by intervention type interaction, $F_{(3,\ 51)} = 0.82$, $p = 0.489$, $\eta^2_p = 0.05$. Global workload, as measured by the NASA-TLX, revealed significant main effects for intervention type, $F_{(1,\ 17)} = 82.80$, $p < 0.001$, $\eta^2_p = 0.83$, and task type, $F_{(1,\ 17)} = 94.59$, $p < 0.001$, $\eta^2_p = 0.85$. Moreover, there was a significant intervention type by task type interaction, $F_{(1,\ 17)} = 70.83$, $p < 0.001$, $\eta^2_p = 0.81$. Overall, the exercise task was rated with significantly higher workload measures relative to the intervention and control task (see **Figure 2**).

Physical Performance and Task Execution

Participants completing the cognitively fatiguing intervention showed no significant difference in repetitions between the fatigue intervention (542; $SD = 119.10$) relative to the control intervention (545; $SD = 107.05$), $t_{(17)} = 0.30$, $p = 0.772$, $d_z = 0.07$. In total, 11 of the 18 participants completed less repetitions per time when completing the cognitive fatigue intervention relative to the control prior to the exercise task. Participants completing the fatigue intervention spent significantly less time-on-task (57%; $SD = 12.43$) relative to the control intervention (60%; $SD = 11.88$), see **Figure 3**, $t_{(17)} = 2.26$, $p = 0.037$, $d_z = 0.53$. For individual performance, including percent time-on-task and repetitions can be see **Figure 4**.

Physiologic Measures

There was a significant main effect of time for heart rate and VO_2 [$F_{(2,\ 34)} = 246.06$, $p < 0.001$, $\eta^2_p = 0.94$; $F_{(2,\ 34)} = 992.44$, $p < 0.001$, $\eta^2_p = 0.98$, respectively]. All other main effects and interactions failed to reach significance $p > 0.05$. *Post-hoc* *t*-test with Bonferroni corrections for time periods are presented in **Table 2**. Regardless of intervention or control, participants only had significant increases in heart rate and VO_2 when comparing the intervention and exercise task measures. For

TABLE 1 | Descriptive statistics (M; *SD*) for each time period of the cognitive task.

Period	1	2	3	4	5	6	7	8
Response time ms	358 (37.33)	392 (46.24)	407 (56.00)	416 (56.00)	417 (60.67)	426 (61.09)	428 (64.06)	439 (69.58)
Accuracy %	99.1 (2.12)	99.7 (0.89)	99.7 (0.89)	99.2 (1.87)	99.1 (1.65)	99.4 (1.19)	98.4 (2.54)	99.5 (1.06)

each 20 min exercise task preceded by the control and fatigue intervention, there was a significant main effect of time period for heart rate, $F_{(2, 34)} = 246.06$, $p < 0.001$, $\eta^2_p = 0.94$; however, there was no main effect for intervention type, $F_{(1, 17)} = 0.67$, $p = 0.425$, $\eta^2_p = 0.04$, or time by intervention type interaction, $F_{(2, 34)} = 0.81$, $p = 0.452$, $\eta^2_p = 0.05$. There was a main effect for time period for VO_2, $F_{(2, 34)} = 992.44$, $p < 0.001$, $\eta^2_p = 0.98$, but no main effect for intervention type, $F_{(1, 17)} = 0.009$, $p = 0.928$, $\eta^2_p = 0.001$, or time by intervention type interaction, $F_{(2, 34)} = 0.49$, $p = 0.933$, $\eta^2_p = 0.004$.

Psychological Measures

RPE revealed a main effect of time, $F_{(1, 51)} = 153.53$, $p < 0.001$, $\eta^2_p = 0.90$. All other main effects ($p = 0.45$, $\eta^2_p = 0.006$) and interactions ($p = 0.67$, $\eta^2_p = 0.002$) failed to reach significance (see **Figure 5**). The subjective post-intervention DSSQ analysis revealed a 3-way interaction between intervention type, time period, and scales, $F_{(6, 102)} = 2.59$, $p = 0.022$, $\eta^2_p = 0.13$. Bonferroni *post-hoc* tests revealed that for intervention there was a significant difference between fatigue and control intervention for energetic arousal and task related thoughts ($p = 0.019$). Additionally, there were differences between baseline and the fatigue intervention ($p = 0.019$) and exercise task and fatigue intervention ($p = 0.009$). See **Table 3** for a complete list of DSSQ results.

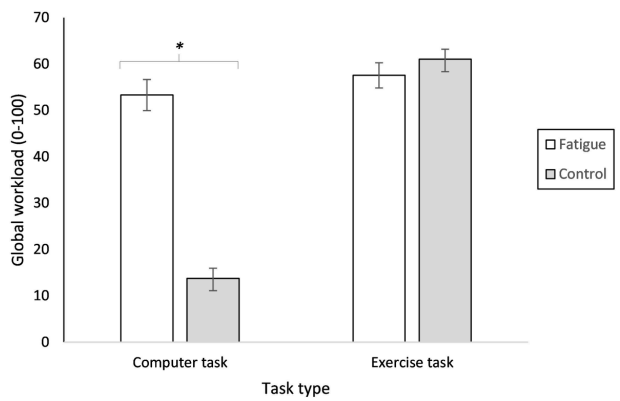

FIGURE 2 | Workload Measures. Global workload measures for computer and exercise tasks. *Significant interaction between task and intervention type ($p < 0.001$). Data are presented as mean ± SEM.

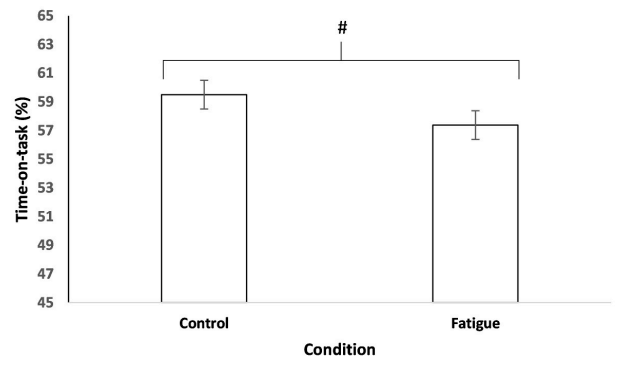

FIGURE 3 | Task execution. Percent time-on-task completed as a function of cognitive fatigue or control. #Significant main effect for condition ($p < 0.05$). Data are presented as mean ± SEM.

DISCUSSION

This study examined the influence of prior cognitive fatigue on subsequent physical performance of a high intensity full body resistance exercise task. The cognitive fatigue intervention was subjectively difficult and did produce cognitive fatigue for participants. The current findings support the hypothesis that task execution (time spent performing the task) on a full body resistance exercise task can be negatively impacted when preceded by a cognitive fatiguing task.

Vigilance Induced Cognitive Fatigue

The vigilance decrement can be characterized as an increased proportion of missed targets and slower response rate to targets over time (Mackworth, 1948; Head et al., 2012; Head and Helton, 2012, 2013). Individually and in combination, these behavioral measures reflect the difficulty and cognitively fatiguing nature of vigilance tasks (Warm and Jerison, 1984). The vigilance task utilized in the current investigation failed to show physiological differences in heart rate or VO_2.

The vigilance intervention was successful in eliciting cognitive fatigue. Participants had slowed response times to targets across the vigilance intervention which is a behavioral indicator of cognitive fatigue (Warm and Jerison, 1984). The high accuracy rate coupled with the elevated task related thoughts suggests that participants were focused on the task. Moreover, participants rated the cognitively fatiguing task as being more energetically arousing and having greater global workload relative to video control task. This elevated energetic arousal is indicative of mental resource recruitment (Warm et al., 2008; Ossowski et al., 2011). Elicited cognitive fatigue is not task specific to response inhibition or a more complex cognitive task such as the AX-CPT used in previous studies (Marcora et al., 2009; Pageaux et al., 2014). Indeed, a relatively more unadulterated measure of sustained attention such as the vigilance task used in the current study had measureable effects on physical activity.

Effects of Cognitive Fatigue on Full Body Resistance Exercise

Importantly, the percent time-on-task measure was sensitive enough to show statistical differences. Participants who were cognitively fatigued showed a 3% decrease in time-on-task relative to the no fatigue condition. Repetitions completed was possibly too coarse of a measure, lacking the sensitivity to show a performance difference between the fatigue and control conditions. Indeed, a *post-hoc* power analysis revealed that the repetitions measure ($1 - \beta = 0.06$) had far less power relative to the percent time-on-task measure ($1 - \beta = 0.58$). Importantly, the results provide evidence that physical engagement with an exercise task can be influenced by prior cognitive fatigue. In other

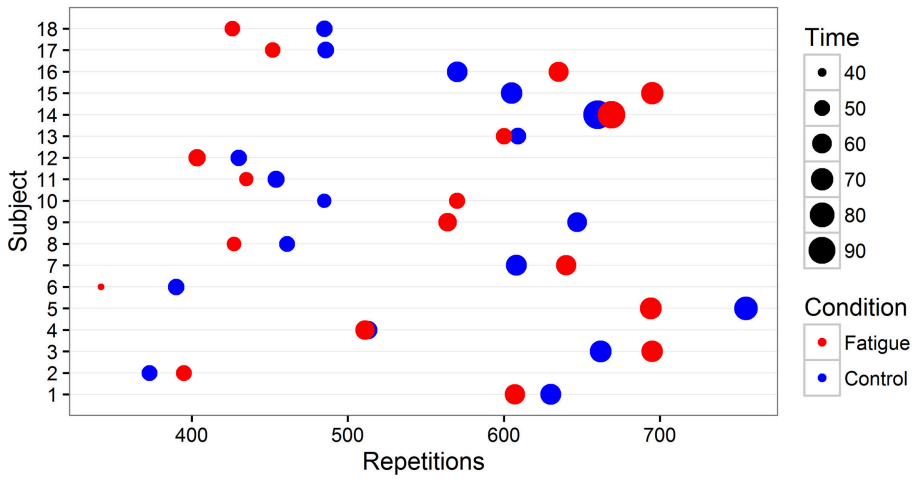

FIGURE 4 | Individual performance and task execution. Each participant ($n = 18$) is displayed on the y-axis. Percent time-on-task is visually expressed as magnitude of icons. Position of fatigue and control icons on the x-axis correspond to repetitions completed as a function of intervention.

TABLE 2 | Descriptive statistics (M; *SD*) and significance test for time period and fatigue type.

	Cognitive fatigue			Control		
	Pre-CT	**CT**	**ET**	**Pre-CT**	**CT**	**ET**
Heart rate	51.8 (30.10)	70.6 (8.90)	159.9 (19.50)$^{\$\dagger***}$	57.5 (30.95)	67.7 (9.32)	164.5 (10.60)$^{\$\dagger***}$
VO_2 ml·kg^{-1} min^{-1}	3.9 (2.54)	4.1 (4.24)$^{\$*}$	32.9 (19.93)$^{\$\dagger***}$	4.1 (.14)	4.3 (0.42)	32.7 (22.90)$^{\$\dagger***}$

CT, computer task; HR (bpm), heart rate; ET, exercise task. $^{\$}$*Main effect for time period.*
†*significantly different from CT. ***corresponds to* $p < 0.001$*; *p* < 0.01.

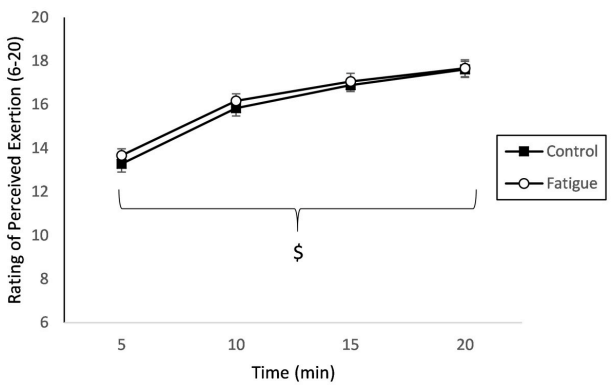

FIGURE 5 | Perceived Exertion. RPE as a function of time-on-task raw scores. $^{\$}$indicated main effect of time ($p < 0.001$). Data are presented as mean ± SEM.

words, participants might be more prone to engage in rest breaks during a physical task when previously cognitively fatigued.

Effects of Cognitive Fatigue on Physiological Measures during Full Body Resistance Exercise

The current results demonstrated that the respective baseline conditions did not significantly differ from either the cognitively fatiguing task or video control task with concern to VO_2 and heart rate. Moreover, the cognitively fatiguing and control task did not significantly differ from each other with respect to VO_2 and heart rate. As expected, there was increased metabolic expenditure and heart rate in exercise tasks relative to the baseline and computer task. As found in prior investigations, the results did not show significant differences in heart rate and metabolic expenditure as a function of cognitive fatigue (Marcora et al., 2009; Pageaux et al., 2014). Had the number of repetitions been substantially different between the interventions, an increase in both mean VO_2 and heart rate would be expected. It is likely that the measure of repetition count, VO_2 and heart rate are all too coarse to observe the 3% difference observed in time-on-task (Darter et al., 2013).

Psychological Effects of Cognitive Fatigue

The workload measure, as indexed by NASA-TLX, provided corroborating evidence that the vigilance intervention was cognitively fatiguing relative to control. Moreover, the energetic arousal measure was elevated after the vigilance task relative to the control task indicating that participants were actively engaged with the task. This is further supported by the high accuracy rate during the vigilance task. In other words, the vigilance task was cognitively demanding and participants were focused on the task. The task related thoughts measure provided evidence that participants had a significant increase in task

TABLE 3 | Descriptive statistics (M, *SD*) and significance test for the DSSQ scales and conditions.

	Mental fatigue			Control		
	Pre-CT	**Post-CT**	**Post-ET**	**Pre-CT**	**Post-CT**	**Post-ET**
EA	16.61 (1.87)	16.50 (1.95)	17.11 (2.29)	16.00 (2.46)	14.22 (2.67)	16.67 (2.84)
TA	15.67 (1.82)	16.05 (2.96)	12.17 (2.29)	15.39 (2.16)	15.11 (3.30)	12.83 (3.18)
TRT	15.78 (4.67)	22.67 (5.09)	19.06 (3.01)	13.89 (3.90)	16.17 (5.09)	19.61 (5.52)
TUT	13.00 (3.48)	15.17 (4.67)	9.72 (4.07)	12.06 (3.31)	13.28 (6.36)	10.94 (3.27)

Effect	**DF**	***F***	***P***	η^2_p
Fatigue	(1,17)	6.77	0.019	0.29
Time point	(2,34)	7.47	0.002	0.31
Scale	(3,51)	32.5	<0.001	0.66
Fatigue × time point	(2,34)	7.23	0.002	0.30
Fatigue × scale	(3,51)	3.74	0.017	0.18
Time point × scale	(6,102)	13.77	<0.001	0.45

CT, computer task; ET, exercise task; EA, Energetic Arousal; TA, Tense Arousal; TRT, Task Related Thoughts; TUT, Task Unrelated Thoughts.

related thoughts upon completing the vigilance task; however, task related thoughts decreased significantly after completion of the exercise task. This result may suggest a shifting focus from the extrinsic goal of completing the task to unrelated stimuli (Helton et al., 2011; Ossowski et al., 2011). As suggested by the lower time-on-task after cognitive fatigue, participants may be less willing to provide maximal effort when cognitively fatigued (Marcora et al., 2009; Pageaux et al., 2014; Smith et al., 2015, 2016).

Limitations and Directions for Future Research

The current investigation provides evidence that cognitive fatigue impairs time on task during a high intensity bodyweight resistance exercise task. However, some limitations are present and should be identified. Though the current investigation and prior studies on this topic have utilized different time lengths (30–90 min), cognitively fatiguing task types (e.g., response inhibition and vigilance) and stimuli (i.e., alphanumeric), it is unknown whether the difficulty of the cognitively fatiguing task has differential effects on subsequent physical performance. Indeed, dual-task paradigms (i.e., completing two tasks at the same time) have provided evidence that stimuli requiring more cognitive effort to process will generate greater performance impairments on a secondary cognitive task (Head et al., 2012; Head and Helton, 2012). This same principle may hold true when a cognitively fatiguing task is performed prior to the physical task.

Given the current and prior investigations are interested in the interaction between cognitive fatigue and physical performance, future investigations may benefit from incorporating non-invasive neurologic measures of executive function (e.g., fNIRS; Head and Helton, 2013; Byun et al., 2014). For example, fNIRS can be used to estimate the level of cognitive fatigue during the cognitively fatiguing task and the subsequent physical task. Incorporating neuro-correlate measures may permit an enhanced objective understanding of the level of cognitive fatigue experienced by the participant and more importantly how it effects their physical performance.

Lastly, given the emphasis of military application, it is unknown whether Soldiers would respond similarly as their civilian counterparts. For example, Soldiers are commonly equipped with tactical gear (e.g., improved outer tactical vest, ammunition, Army combat uniform, and weapon) that is difficult to maneuver in and is often heavy. Additionally, Soldiers likely receive more physical and mental toughness training relative to their average civilian counterpart. Thus, the cognitive fatigue intervention may need to be more extreme to illicit fatigue from Soldiers relative to civilians.

Perspective

The current findings provide evidence that cognitive fatigue impacts how subsequent physical activity is performed. Participants who are cognitively fatigued spend significantly less time-on-task (i.e., more rest breaks) than when not previously cognitively fatigued. More importantly, our findings show that the effect of cognitive fatigue is independent of specific intervention techniques (vigilance or response inhibition) and is not dependent on the exercise task being performed (running, cycling, and discontinuous exercise task). Given the evidence of impaired performance on a physical task as a function of cognitive fatigue, we advise that athletes and Soldiers do not engage in unnecessary cognitively demanding tasks prior to competitions or missions, respectively. Additionally, as suggested by Smith et al. (2015), coaches or commanders may need to assess their personnel's level of cognitive fatigue prior to physical activity.

The results of the current investigation have implications for athletes, but also for Soldiers. For example, if Soldiers engage in a cognitively fatiguing task (e.g., guard duty at a checkpoint) prior to completing a high intensity physical engagement with enemy combatants, then it may be more difficult for Soldiers to stay on task which could be detrimental due to the demanding

nature of war. Additionally, performance impairments as a result of cognitive fatigue may extend beyond gross motor skills to tasks requiring fine motor actions (Duncan et al., 2015); for example, marksmanship performance. This may have direct and dire implications for Soldiers who are cognitively fatigued and then required to engage in a firefight (e.g., friendly fire). Future studies on this topic should include fine motor tasks relevant to Soldiers (e.g., marksmanship) to determine whether the effect of cognitive fatigue on subsequent physical performance is only a gross motor phenomena.

AUTHOR CONTRIBUTIONS

JH: substantially contributed to the conception, design, acquisition and analysis of data, and interpretation of the work, was involved in drafting the work, approved the final version to be published and agree to be accountable for all aspects of the work. MT: highly contributed to the conception, design, acquisition and analysis of data, and interpretation of the work, was involved in drafting the work, approved the final version to be published and agree to be accountable for all aspects of the work. TP: highly contributed to revise the work critically for important intellectual content, approved the final version to be published and agrees to be accountable for all aspects of the work. AT: highly contributed to the conception, design, acquisition and analysis of data, and interpretation of the work, was involved in drafting the work, approved the final version to be published and agree to be accountable for all aspects of the work. ML: highly contributed to the conception, design, and interpretation of the work, was involved in drafting the work, approved the final version to be published and agrees to be accountable for all aspects of the work. WH: highly contributed to the conception, design, and interpretation of the work, was involved in drafting the work, approved the final version to be published and agrees to be accountable for all aspects of the work.

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